Behavioural Sleep Medicine Conceptualisations and Associated Treatment of Clinical Insomnia Disorder in Adults

Colin Alexander Espie BSc (Hons), MAppSci, PhD, CPsychol, CSci, FBPsS
Nuffield Department of Clinical Neurosciences,
University of Oxford

Submitted for the Degree of Doctor of Science in Medicine by Published Work to University of Glasgow, February 2013.

Summary

This thesis summarises a selection of forty-two studies [1-42], published by the author during the period 2000-2012, investigating the conceptual basis of Insomnia Disorder, and its evaluation and treatment, principally using cognitive and behavioural interventions.[a] The work reflects a range of research methodologies including experimental, psychometric, qualitative and population-based studies, and randomised controlled trials. Important theoretical contributions to the literature published in this period are also included and reference is made to major textbooks, position papers, and influential chapter contribution.

The Research Diagnostic Criteria (RDC, 2003) for Insomnia Disorder refer to persistent difficulty with initiating and/ or maintaining sleep with associated daytime consequences [11]. Despite the latter symptoms being important 'drivers' of clinical complaint, they have been relatively neglected. Therefore, proposed refinements of clinical and RDC criteria, in the Diagnostic and Statistical Manual of Mental Disorders (5th edition: DSM-5, due 2013), have been evaluated in large scale field trials. Our studies have revealed that the daytime sequelae of Insomnia Disorder comprise two statistically robust principal components; daytime performance deficits (in concentration, productivity and sleepiness) and compromised interpersonal and social functioning (low energy, mood, relationship problems) [41]. Alongside such night-time and daytime symptoms, people

[a] The thesis specifically excludes other works by the author, during the same period, on sleep disorders in special populations (e.g. learning disabilities/mental retardation, acquired brain injury, pain), on other sleep disorders (e.g. sleep apnoea) and on other disorders (e.g. epilepsy). The thesis also excludes published studies where the author contributed as a co-investigator to work initiated by international collaborators. On all included data-based papers the author is first, second or last named in the authorship list.

with insomnia exhibit reciprocally interacting behavioural, cognitive and emotional concerns about poor sleep which perpetuate the disorder and adversely affect quality of life [28]. Indeed, contemporary thinking about the aetiology and maintenance of persistent Insomnia Disorder incorporates variants of the 'psychology' of sleep [35]. The International Classification of Sleep Disorders (second revision; ICSD-2, 2005) certainly emphasises such phenotypical features, particularly in relation to Psychophysiological Insomnia, the most common form of insomnia in adults. We have reported sensitivity and specificity characteristics for this (and other) forms of insomnia, showing that cognitive behavioural features may be an important target for clinical intervention [9, 12, 21, 37]. Indeed, our Psychobiological Inhibition Model of insomnia [PIM: 6] and its presumed Attention-Intention-Effort Pathway [A-I-E: 16] have been at the forefront of understanding insomnia as a disorder of sleep preoccupation, and difficulty in down-regulating arousal at bedtime and during the night. Importantly, experimental studies using novel computer-based paradigms of selective attention bias have provided confirmatory data of the PIM/A-I-E model [13, 15, 19, 25]. Likewise, our qualitative and psychometric scale development studies have yielded reliable and valid instruments to capture cognitive/ attributional data for both descriptive and treatment outcome purposes [1, 2, 10, 14, 24, 29], such that many of our measures are recommended by international review groups [18]. Further studies have pointed to the reactivity of people with Insomnia Disorder to naturally occurring and experimental stimuli [21, 32]. A logical extension of all these findings has been to apply randomised controlled trial (RCT) methodology to investigate the efficacy and clinical effectiveness of cognitive

behavioural therapy (CBT) for insomnia. We have developed and tested a novel mode of community nurse-delivered, small group CBT across two relatively unselected primary care samples [3, 22] employing a treatment as usual (TAU) control group, to reflect real world evaluation. This manualised CBT programme ensured robust levels of treatment standardisation, and findings demonstrated statistically and clinically significant benefits to sleep pattern and daytime wellbeing that were maintained at 6 to 12 month follow up [3, 4, 5, 22]. One consequence of this work has been a demand for dissemination of CBT methods, both to clinicians and direct to the public. Collaboration with a Canadian colleague, therefore, led to the publication of a clinician's handbook in 2003 [8], which was subsequently translated into Italian and Mandarin; and publication of a 'self-help' book in 2006 [20] which is now available in French, Italian and Danish, with other translations in progress. The RCT methodology was then extended in a further trial where cancer patients with persistent insomnia were treated by cancer nurse specialists who had been trained in CBT [23]. Again, very favourable results were obtained at post-treatment and follow-up, and this work was subsequently recognised as the Best Patient Support Initiative at the UK National Oncology Awards in 2009, and received the Pfizer Prize. Recent systematic reviews by international peer groups have highlighted these three trials [3, 22, 23] in their evidence-based guidelines for clinically effective insomnia therapies [17, 33]. So Insomnia Disorder is a very common and durable disorder [39], which can be effectively treated with CBT, however, a major obstacle in practice continues to be the paucity of services available, to provide an alternative to pharmacotherapy. This is despite the evidence that a CBT approach would be preferred

by most patients [37]. Our further response to this situation has had several research strands. First, to suggest how services might be delivered in a cost-effective way using a stepped care model [26]; second, to explore abbreviated CBT by investigating its component strategies and their efficacy [7, 27, 34]; and third, to simplify the self-help proposition by publishing a very brief guide to CBT for insomnia [36] and using web and mobile technology to deliver CBT online. In relation to the latter, we recently published a placebo controlled RCT of a rich media, online CBT programme where the intervention was delivered by an animated virtual therapist. [40, 42] Results from this trial mirror the effects and effect sizes typically obtained in face-to-face therapy for sleep variables, daytime benefits and sleep-related attributions and thinking styles, thus suggesting that online CBT could (at least) provide an effective entry level treatment for a stepped care service. Finally, we have endeavoured to strengthen the dissemination of knowledge and good practice about Insomnia Disorder through overview papers in selected influential textbooks [30, 31] and through planning and co-editing the Oxford Handbook of Sleep and Sleep Disorders [38] which is a 900 page reference book that was published in 2012.

List of papers

1. Wicklow, A. and Espie, C.A. (2000) Intrusive thoughts and their relationship to actigraphic measurement of sleep: towards a cognitive model of insomnia. *Behaviour Research and Therapy* 38, 679-693
2. Espie, C.A., Inglis, S.J., Harvey, L., and Tessier, S. (2000) Insomniacs' attributions: Psychometric properties of the Dysfunctional Beliefs and Attitudes about Sleep scale and the Sleep Disturbance Questionnaire. *Journal of Psychosomatic Research* 48, 141-148
3. Espie, C.A., Inglis, S.J., Tessier, S., and Harvey, L. (2001) The clinical effectiveness of cognitive behaviour therapy for chronic insomnia: Implementation and evaluation of a *Sleep Clinic* in general medical practice. *Behaviour Research and Therapy* 39, 45-60
4. Espie, C.A., Inglis, S.J. and Harvey, L. (2001) Predicting clinically significant response to cognitive behavior therapy (CBT) for chronic insomnia in general practice: analyses of outcome data at 12 months post-treatment. *Journal of Consulting and Clinical Psychology* 69, 58-66
5. Harvey, L., Inglis, S.J. and Espie, C.A. (2002) Insomniacs' reported use of CBT components and relationship to long-term clinical outcome. *Behaviour Research and Therapy* 40, 75-83
6. Espie, C.A. (2002) Insomnia: Conceptual issues in the development, persistence and treatment of sleep disorder in adults. *Annual Review of Psychology* 53, 215-243
7. Broomfield N., and Espie, C.A. (2003) Initial insomnia and paradoxical intention: an experimental investigation of putative mechanisms using subjective and actigraphic measurement of sleep. *Behavioural and Cognitive Psychotherapy* 31, 313-324
8. Morin C.M and Espie, C.A. (2003) *Insomnia: A Clinical Guide to Assessment and Treatment*. Kluwer Academic/ Plenum Publishers, New York [ISBN 0-306-47750-5]

9. Miller, A. Espie, C.A. and Scott, J. (2004) The sleep of remitted bipolar outpatients: a controlled naturalistic study using actigraphy. *Journal of Affective Disorders* 80, 145-153
10. Harvey, K.J. and Espie, C.A. (2004) Development and preliminary validation of the Glasgow Content of Thoughts Inventory (GCTI): a new measure for the assessment of pre-sleep cognitive activity. *British Journal of Clinical Psychology* 43, 409-420
11. Edinger, J.D., Bonnet, M.H., Bootzin, R.R., Doghramji, K., Dorsey, C.M, Espie, C.A., Jamieson, A.O., McCall, W.V., Morin, C.M., Stepanski, E.J. (2004) Derivation of Research Diagnostic Criteria for Insomnia: Report of an American Academy of Sleep Medicine Work Group. *Sleep* 27, 1567-1596
12. Kohn, L. and Espie, C.A. (2005) Sensitivity and specificity of measures of the insomnia experience: A comparative study of psychophysiologic insomnia, insomnia associated with mental disorder and good sleep. *Sleep* 28, 104-112
13. Jones, B.T., Macphee, L., Broomfield, N.S., Jones, B.C. and Espie, C.A. (2005) Sleep-related attentional bias in good, moderate, and poor (primary insomnia) sleepers. *Journal of Abnormal Psychology* 114, 249-258
14. Broomfield, N.M. and Espie, C.A. (2005) Toward a reliable, valid measure of sleep effort. *Journal of Sleep Research* 14, 401-407
15. Macphee, L.M., Biello, S.M., Broomfield, N.M., MacMahon, K.M.A. & Espie C.A. (2006) Who is pre-occupied with sleep?: A comparison of attention bias in people with Psychophysiologic Insomnia, Delayed Sleep Phase Syndrome and Good Sleepers using the Induced Change Blindness paradigm. *Journal of Sleep Research* 15, 212-221
16. Espie, C.A., Broomfield, N.M., MacMahon K.M.A., Macphee, L.M. & Taylor, L.M. (2006) The attention-intention-effort pathway in the development of Psychophysiologic Insomnia: a theoretical review. *Sleep Medicine Reviews* 10, 215-245
17. Morin, C.M., Bootzin, R.R., Buysse, D.J., Edinger, J.D., Espie, C.A. & Lichstein, K.L. (2006) Psychological and behavioural treatment of insomnia. Update of the

recent evidence (1998-2004) prepared by a Task Force of the American Academy of Sleep Medicine. *Slee*p 29, 1398-1414

18. Buysse, D.J., Ancoli-Israel, S., Edinger, J.D., Lichstein, K.L. and Morin, C.M. [on behalf of co-authors including Espie, C.A.] (2006) Recommendations for a standard research assessment of insomnia. Sleep, 29, 1155-1173
19. MacMahon, K.M.A., Broomfield, N.M., Macphee, L.M. and Espie, C.A. (2006) Attention bias for sleep-related stimuli in primary insomnia and delayed sleep phase syndrome. *Sleep* 29, 1420-1427
20. Espie, C.A. (2006) *Overcoming Insomnia and Sleep Problems: A Self-Help Guide Using Cognitive Behavioral Techniques*. Constable & Robinson Ltd., London [ISBN13: 978-184529-070-2/ ISBN10: 1-84529-070-4]
21. Robertson, J.A., Broomfield, N.B. and Espie, C.A. (2007) Prospective comparison of subjective arousal during the pre-sleep period in primary sleep-onset insomnia and normal sleepers. *Journal of Sleep Research* 16, 230-238
22. Espie, C.A., MacMahon, K.M.A., Kelly, H-L., Broomfield, N.M., Douglas, N.J., Engleman, H.E., McKinstry, B., Morin, C.M. Walker, A. & Wilson, P. (2007) Randomised clinical effectiveness trial of nurse-administered small group CBT for persistent insomnia in general practice. *Sleep* 30, 574-584
23. Espie, C.A., Fleming, L., Cassidy, J., Samuel, L., Taylor, L.M., White, C.A., Douglas, N.J., Engleman, H.E., Kelly, H-L. & Paul, J. (2008) Randomized controlled clinical effectiveness trial of Cognitive Behavior Therapy versus Treatment as Usual for persistent insomnia in cancer patients. *Journal of Clinical Oncology* 26, 4651-4658
24. Waine, J., Broomfield, N.B. & Espie, C.A. (2009) Development and validation of the sleep metacognitive beliefs scale. *Journal of Behaviour Therapy & Experimental Psychiatry*, 40, 15–23
25. Woods, H., Marchetti, L.M., Biello, S.M., Espie, C.A. (2009) The clock as a focus of selective attention in those with primary insomnia: an experimental study using a modified Posner paradigm. *Behaviour Research & Therapy*, 47,231-236

26. Espie, C.A. (2009) 'Stepped care': a health technology solution for delivering Cognitive Behavioral Therapy as a first line insomnia treatment. *Sleep,* 32 (12), 1549-1558
27. Mooney, P., Espie, C.A., Broomfield, N.M. (2009) An Experimental Assessment of a Pennebaker Writing Intervention in Primary Insomnia. *Behavioral Sleep Medicine* 7, 99-105
28. Kyle, S.D, Morgan, K., Espie, C.A. (2010) Insomnia and health-related quality of life *Sleep Medicine Reviews* 14, 69–82
29. Fleming L, Gillespie S, Espie CA. (2009) The development and impact of insomnia on cancer survivors: a qualitative analysis. *Journal of Psycho Oncology* 19, 991-996
30. Espie CA, Bartlett D.J. (2009) Insomnia, in R.P. Lisak, D.D. Truong, W. Carroll & R. Bhidayasiri (eds) *International Neurology: A Clinical Approach*. Wiley-Blackwell, Oxford, pp. 554-556
31. Espie, C.A. Bartlett D.J. (2009) Clinical syndromes of adult psychiatry: Insomnia in M.G. Gelder, J.J. Lopez-Ibor and N.C. Andreasen (eds) *New Oxford Textbook of Psychiatry (Vol. 2)* (second edition) Oxford University Press, Oxford, England pp. 933-938
32. Baglioni, C., Lombardo, C., Bux, E., Hansen, S., Salveta, C., Biello, S., Violani, C., Espie, C.A. (2010) Psychophysiological reactivity to sleep-related emotional stimuli in primary insomnia *Behaviour Research and Therapy*, 48, 467-475
33. Wilson, S.J., Nutt, D.J., Alford, C., Argyropoulos, S.V., Baldwin, D.S., Bateson, A.N., Britton, T.C., Crowe, C., Dijk, D-J, Espie, C.A., Gringras, P., Hajak, G., Idzikowski, C., Krystal, A.D., Nash, J.R., Selsick, H., Sharpley, A.L. and Wade, A.G. (2010) British Association for Psychopharmacology consensus statement on evidence-based treatment of insomnia, parasomnias and circadian rhythm disorders. *Journal of Psychopharmacology,* 24 (11), 1577–1600
34. Kyle, S.D., *Mo*rgan, K., Spiegelhalder, K., & Espie, C.A. (2011) No pain, No gain: An exploratory within-subjects mixed-methods evaluation of the patient experience of sleep restriction therapy. *Sleep Medicine* 12, 735-747

35. Perlis M.L., Shaw, P., Cano, G., Espie, C.A. (2011) Models of Insomnia In MH Kryger, T Roth and WC Dement (eds) *Principles and Practice of Sleep Medicine*, 5th edition. Elsevier Saunders, NY
36. Espie, C.A. (2011) *Introduction to Coping with Insomnia & Sleep Problems*. Constable & Robinson Ltd., London [ISBN978-1-84901-620-9]
37. Espie, C.A., Barrie, L.M., Forgan, G.S. (2012) Comparative investigation of the psychophysiological and idiopathic Insomnia Disorder phenotypes: psychological characteristics, patients' perspectives and implications for clinical management. *SLEEP,* 35, 385-393
38. Morin, C.M. & Espie, C.A. (2012) The Oxford Handbook of Sleep and Sleep Disorders (Oxford Library of Psychology) Oxford University Press, USA [ISBN-10: 019537620X]
39. Green, M., Benzeval, M., Espie, C.A., Hunt, K. (2012) The longitudinal course of insomnia symptoms: inequalities by gender and occupational class among two different age cohorts followed for 20 years in the West of Scotland. *SLEEP* 35, 815-823
40. Espie, C.A., Kyle, S.D, Williams, C., Ong, J.C., Douglas, N.J., Hames, P., Brown, J.S.L. (2012) A randomized, placebo-controlled, trial of online Cognitive Behavioral Therapy for chronic Insomnia Disorder delivered via an automated media-rich web application. *SLEEP* 35, 769-781
41. Espie, C.A., Kyle, S.D, Hames, P., Cyhlarova, E., Benzeval, M. (2012) The daytime impact of DSM-5 Insomnia Disorder: comparative analysis of insomnia subtypes from the Great British Sleep Survey (n=11,129) *Journal of Clinical Psychiatry* 73, e1-e7
42. Espie, C.A., Kyle, S.D, Miller, C., Williams, C., Ong, J.C., Hames, P. (2013) Randomized, placebo-controlled, trial of online Cognitive Behavioral Therapy for persistent Insomnia Disorder: therapeutic impact upon attribution, cognition and psychopathology *Journal of Consulting & Clinical Psychology* (in press)

List of abbreviations

AASM	American Academy of Sleep Medicine
A-I-E	Attention-Intention-Effort pathway
BAP	British Association of Psychopharmacology
BD	Bipolar Disorder
BSM	Behavioural Sleep Medicine
BZ	Benzodiazepine
BZRA	Benzodiazepine Receptor Agonist
CBT	Cognitive Behavioural Therapy
CR-UK	Cancer Research UK
CSO	Chief Scientist Office, Scotland
DBAS	Dysfunctional Beliefs and Attitudes about Sleep scale
DBAS-10	Dysfunctional Beliefs and Attitudes about Sleep scale (10-item version)
DSM-IV	Diagnostic & Statistical Manual of Mental Disorders (4^{th} edition)
DSM-5	Diagnostic & Statistical Manual of Mental Disorders (5^{th} edition)
DSPS	Delayed Sleep Phase Syndrome
EEG	Electroencephalography
ERP	Event Related Potential
GBSS	Great British Sleep Survey
GCTI	Glasgow Content of Thoughts Inventory
GSES	Glasgow Sleep Effort Scale

HRQoL	Health Related Quality of Life
ICSD-2	International Classification of Sleep Disorders (2nd revision)
IAPS	International Affective Picture System
LCA	Latent Class Analysis
NCRI	National Cancer Research Institutes
OSA	Obstructive Sleep Apnoea
MBT-I	Mindfulness Based Therapy for Insomnia
MeRa	Melatonin Receptor Agonist
MRC	Medical Research Council
NHSGGC	National Health Service: Greater Glasgow & Clyde
NCRI	National Cancer Research Institutes
NIH	National Institutes of Health
PCA	Principal Components Analysis
PIM	Psychobiological Inhibition Model
PLMD	Periodic Limb Movement Disorder
PSAS	Pre-Sleep Arousal Scale
PSG	Polysomnography
RDC	Research Diagnostic Criteria
RCT	Randomised Controlled Trial
RLS	Restless Legs Syndrome
SCI	Sleep Condition Indicator
SDQ	Sleep Disturbance Questionnaire

SHPSU	MRC Social and Public Health Sciences Unit, University of Glasgow
TAU	Treatment as Usual
UGSC	University of Glasgow Sleep Centre
VHA	Veteran's Health Administration

Acknowledgements

Research is a collaborative process, and I would like to take this opportunity to thank the many people without whom my research efforts would have come to nothing. Of course, it is not easy to produce an exhaustive list; and equally rather invidious to mention some at the expense of others. So I have had to be quite general, though none the less personally grateful. If you are not named specifically; still, you know who you are!

I have to start with the participants, mostly patients with insomnia, and sometimes a host of other health problems too. You have been my inspiration, my raison d'être. I make no apology for being at heart a clinician with research interests. I thank you for allowing me in the clinic setting to see into your world. Although you came for help, and I tried to provide it, I also benefitted from the insights that you gave me. These allowed me to develop my ideas, hypotheses and research studies. Then you came again to participate in the research, giving freely of your time; and sometimes with no direct gain for yourself. Indeed, I have been humbled by your commitment to research; perhaps greater than my own. If there were times that I doubted the value of what I was doing, it was you who made me think again.

I trained in clinical psychology at a time (1978-1980) when we firmly believed in the 'scientist-practitioner' model. These two roles were essentially indivisible. Professional responsibility comprised both delivering and contributing to, evidence-based practice. Regrettably, over the years we have seen some erosion of this model, particularly its latter dimension. I am grateful to my teachers, supervisors and mentors in the discipline of clinical psychology, from those early days of my formal training. The late Dr Gerry Greene whose guidance I valued throughout my career; Dr John Taylor and Angus Scott who taught me about adult mental health; and Prof Neil Brooks and Prof Bill Lindsay who encouraged me to undertake my PhD during my first NHS post. Bill in particular was a phenomenon, and I am indebted to him.

People often ask how I first got interested in sleep. That was down to a GP in Lanarkshire who asked me in 1980 " … can you not do anything for these folk who can't sleep?" I said that I didn't know. Now I admit that there may be some dynamic about that situation which creates a conflict for me (!), but it also reflects my training ethos … I should certainly try to find out. Dr Eileen Hood and I embarked on our PhD studies in parallel, both mentored by Bill Lindsay. We were part of a cohort of Glasgow PhDs from around that time (e.g. Drs Mike Dow, David Cooke, Chris Main, Carole Allan, Jim White) who thought that research was something you got your teeth into after you qualified and got your first clinical position! These people have all gone on to make a huge contribution to science and practice. It was my privilege also to have Jim White join me in NHS Lanarkshire (1981-1984) to fight the impressive waiting lists in the towns of East Kilbride and Hamilton.

I took over a Lectureship in Clinical Psychology from Dr Bill McKinlay in 1984 (until 1988); the start of a long relationship as a member of staff with the Department of Psychological Medicine at the University of Glasgow. The clinical psychology trainees from those years were my first postgraduate students, and I'm grateful to them and to my colleagues from that time (especially Carole Allan and Gerry Greene), for the opportunities that working with the clinical programme offered. In many cases these are also friendships that have lasted till the present day.

In 1988 I got my first senior appointment in the NHS, in Ayrshire and Arran. Over the next 7 years I was privileged to work with some very creative people, and to have the opportunity to become Clinical Director of Ayrshire's Learning Disability Services. To many this move into a different patient population seemed very odd. What was I? They had thought that I 'did' adult mental health, or clinical health psychology, or epilepsy, or sleep … and now learning disability? But I have always thought that what I do is clinical psychology. There are not so many differences across populations, and the core skills are the same after all. It was actually all a patient's fault. There's that scientist practitioner again! A woman with learning disabilities and epilepsy who needed some help … and her behaviour raised more clinical (and research) questions. Anyway, thank you to her … and

back to Consulting & Clinical Psychology Services, Ayrshire. Zena Wight is by a long way the best Head of Clinical Psychology Service I have ever met. She was tremendously supportive of this phase in my career, and her wisdom then and still is greatly appreciative. Ayrshire gave me further opportunities, and I would like to acknowledge Stanley Bonthron and Dr Allan Gunning for teaching a clinician about management theory in real world practice! Thanks also to the GPs and Health Visitors of south Ayrshire for their support in conducting a successful RCT of CBT for insomnia, to the learning disability staff (and family carers) who helped us achieve a challenging home-based PSG study of sleep and epilepsy in people with complex needs, and to the research teams who worked with me on both of these projects.

Until this point I had attempted to straddle clinical practice and research, so the chance to return to the Department of Psychological Medicine at the University of Glasgow, as Professor of Clinical Psychology, and Director of the Clinical Training Programme was very attractive. Fortunately I was offered the position and in 1995 moved back to this cradle of my learning. The early years there were formative ones, and a crucial time for the profession. I am grateful to my colleagues who helped me to establish the Doctorate in Clinical Psychology (DClinPsy) and to the opportunity that Professor Brian Whiting and Dr Reg Herrington gave me to become Head of Department soon after my arrival. One of the attractions in coming back to Glasgow was the possibility that I could work closely with Prof Keith Millar and I was delighted when he moved his Behavioural Sciences Group into Psychological Medicine. He also trained in clinical psychology late in his career, as if to emphasise his commitment to that merger! Keith and I have remained firm friends over many years.

Carole Allan and I recruited a great team to support the development and expansion of clinical training in Glasgow (including Drs Liz Campbell, Anna Stallard, Liam Dorris, Neil Broomfield, Matt Wild and more recently Jason Ellis). Liz was a tremendous and wise colleague who also made a great contribution nationally. Sadly she passed away during her tenure as President of the British Psychological Society. She is greatly missed as well as fondly remembered. I spent six years as Head of Department during which time we were fortunate to get support from the university and NHS Greater Glasgow & Clyde

(NHSGGC) to establish a University Affiliated Programme in Learning Disabilities (Prof Anna Cooper, Prof Andrew Jahoda, Dr Craig Melville), a programme in neuropsychology research and training (Profs Tom McMillan and Jon Evans), and to support the Glasgow Institute for Psychosocial Interventions (Prof Kate Davidson, Andrew Gumley and Chris Williams). Tim Davison, Chief Executive of the community and primary care services in Glasgow at the time was a refreshing 'can do' NHS leader. Kate Davidson and Andrew Jahoda have been particular friends and colleagues over these many years, and I want to thank them for their support.

Research funding which began in earnest in Ayrshire through the Chief Scientist Office (CSO), continued with grants in the 2000's not only from CSO but also from The Wellcome Trust, NHSGGC Research & Development, ESRC, Cancer Research UK, National Cancer Research Institute and Breast Cancer Campaign. I am grateful to all these funding bodies for their support across many years and to their Research Officers for their advocacy and hard work on behalf of clinical research. The Dr Mortimer & Theresa Sackler Foundation, along with The Wellcome Trust and NHSGGC Research & Development were also instrumental in providing invaluable infrastructure funding support for the establishment in 2004 of the University of Glasgow Sleep Centre at the Southern General Hospital. The provision of such a facility was a watershed in my research programme and made feasible grant funding for lab-based studies e.g. from the National Institutes of Health (USA) and ESRC. I am grateful, therefore, to Professor Sir Michael Bond for all his efforts in developing productive links with the Sackler family, and to my colleagues on the Sackler Management Group (especially Dr Jonathan Cavanagh and Prof Angus Mackay). I am very indebted also to numerous people for their statistical services over the years, notably Prof Ian Ford and Dr Sarah Barry (Robertson Centre, University of Glasgow), Jim Paul (Cancer Research UK/ Beatson Oncology Centre, Glasgow) and Dr Andrew Walker (Department of Statistics, University of Glasgow and Health Economist NHSGGC).

One of the joys of my research career has been the way that research staff funded on grants, have worked alongside postgraduates completing their PhD or DClinPsy research studies. Together they helped to give UGSC its international reputation. This team, and it

has been a team, has comprised scores of people. This is where it becomes very difficult to mention some but not others. From the earliest days of UGSC, Dr Leanne Fleming has been a key person; latterly developing her own research career in sleep and cancer and becoming a good friend in the process. Drs Ken MacMahon, Simon Kyle and Maria Oto have been the backbone of our recent NIH and CSO studies, and Christine Salveta has provided a superb technical facility along with Joan Kane and Stig Hansen, and a dozen or more assistants and overnight nursing staff. At the same time, Prof Stephany Biello has provided important leadership and support from the University Department of Psychology.

Other researchers who have worked as part of the team include Drs Stephanie Inglis, Campbell Reid, Liam Dorris, Neil Broomfield, Matt Wild, Craig White, Maria Gardani, Marina Malaffo, Lauren Macphee and Heather Woods. We have also been delighted to support visiting researchers e.g. from Canada (Dr Annie Vallieres), Germany (Dr Kai Spiegelhalder), Italy (Dr Chiara Baglioni) and the Netherlands (Ilja Croijmans) and to support clinicians in training, most particularly Consultant Neurologist Dr Ian Morrison. Other clinicians too have made a notable contribution to the research including Drs Rod Duncan (neurology), Brian McKinstry and Phil Wilson (general practice), Aline Russell (clinical neurophysiology) and Breda Cullen (clinical neuropsychology). At the time of writing I am very proud to say that two postgraduates have just been awarded their PhDs (Dr Chris Harvey, Dr Megan Crawford) and a third (Chris Miller) has successfully completed the second year of his PhD as part of a Cotutelle Studentship arrangement with the University of Sydney. Louise Beattie (supervised in her PhD primarily by Stephany Biello) has also been an integral part of the work at UGSC.

I have been very fortunate over the years to have had excellent administrative support in my NHS and university positions. Anita McClelland above all deserves special mention, having worked with me not only in the NHS in Ayrshire but also as my departmental administrator in Psychological Medicine and latterly as the Business Manager of UGSC. Anita has been a tremendously loyal colleague, as well as a good friend. In recent years Louise McFadzean joined the team and has proven to be a thoughtful, diligent and enthusiatic PA. I am very grateful to Louise and also to Shona Currie who has become the

'voice' of UGSC by dealing so capably with telephone enquiries and initial screening/ recruitment to our studies.

I have always believed in the virtuous link between research, teaching, and clinical practice. With this in mind we developed the online Masters Programme in Behavioural Sleep Medicine. Dr Jason Ellis, Dr Marina Malaffo and Anita McClelland helped me to develop the concept and the proposed curriculum, and Asha Ginda and Drs Chris Harvey and Megan Crawford helped make it a reality. This has been an interesting journey! At the time of writing the MSc (MedSci) has attracted health professionals from 12 countries around the globe. Asha has been an inspiration in marketing and student support, and Chris and Megan have done a great job in advising and encouraging this interdisciplinary group of postgraduates.

Of course, research these days is also an international affair and I would like to acknowledge and thank my collaborators, near and far. In the UK in particular I would mention Prof Kevin Morgan (Loughborough University), Dr Alice Gregory (Goldsmith's College, London; external examiner to our Masters programme), Dr June Brown (Institute of Psychiatry, London), Prof Niro Siriwardena (University of Lincoln), Prof Sir Neil Douglas (University of Edinburgh), Dr Brian McKinstry (University of Edinburgh) and Dr Leslie Samuels (NHS Grampian and University of Aberdeen); plus colleagues I have not previously mentioned at the University of Glasgow – Prof Jim Cassidy (Oncology), Prof David Morrison (Public Health), and Dr Michaela Benzeval and Prof Kate Hunt (MRC-SHPSU). Further afield it has been my honour to collaborate with Prof Charles Morin and Dr Annie Vallières (Université Laval, Quebec City) on grants funded by the Canadian Institutes of Health Research, with Prof Ron Grunstein and Dr Delwyn Bartlett [University of Sydney: National Health and Medical Research Council (Australia)] and with Prof Michael Perlis (University of Philadelphia: NIH collaborative R-01 grant). Other key collaborators and senior colleagues whose support I have valued include Prof Jack Edinger (University of Colorado, Denver), Prof Allison Harvey (University of Berkeley, Ca), Prof Dick Bootzin (University of Arizona), Prof Rachel Manber (University of Stanford, Ca), Prof leoon Lack (Flinders University, Adelaide), Prof Bjørn Bjorvatn (University of Bergen), Prof Eus van Someren (University of Amsterdam) and Prof Dieter Riemann (University of

Freiburg). All of these international figures have also become firm friends, and along with a cast of others, we have enjoyed many stimulating and productive discussions and debates at congresses and work groups worldwide.

As well as contributing to the scientific literature on insomnia, I have written or edited a number of books on sleep and its disorders. I am grateful in particular to the editorial teams at Kluwer Academic/ Plenum, Constable & Robinson, and Oxford University Press for their advice support and direction, and to their marketing teams for promoting these works. My closest professional collaborator, and dearest friend in the professional sphere has been Prof Charles Morin. I first met Charles at a conference in 1988, and since then we have worked closely together on many projects including our co-written clinician's textbook (2003) and our co-edited Oxford Handbook published in 2012. I am grateful to Charles for this friendship, and for his outstanding leadership in the field.

I have enjoyed participating in the work of national and international societies. I am grateful to Prof Mary Morrell (Imperial College, London) for all the support she gave me in my role as Conference Chair when the British Sleep Society hosted the European Sleep Research Society (ESRS) Annual Meeting in Glasgow in 2008. I am grateful also to the 8 members of the ESRS Scientific Committee who served with me when I was Chair of that committee leading up to the ESRS Paris meeting this year (2012). I have been privileged to work on the editorial boards of several journals and acted as Associate and then Deputy Editor for the *Journal of Sleep Research* (Editor: Prof Derk-Jan Dijk, University of Surrey) and the American Academy of Sleep Medicine journal *SLEEP* (Editor: Prof David Dinges, University of Philadelphia). Thank you Derk-Jan and David for your support and mentorship.

In the past few years some of my research took on a new and unanticipated direction after being contacted by Peter Hames. Peter is a graduate in Experimental Psychology from the University of Oxford, but he has built a reputation as an entrepreneur in the online business sector. Together we have developed Sleepio, a 100% online CBT programme delivered by a virtual sleep expert, the Prof. Our collaboration has brought together high quality research and first class digital expertise. This has proven to be a tremendously productive working relationship as well as great fun. We share a passion

for making CBT available – one of the biggest challenges of our times. Peter has injected expertise in the execution of health behavior change into Sleepio, and his unrelenting focus on optimizing the user experience complements my clinician/ researcher perspective upon best patient care. I am particularly grateful to Peter and to the team (including Rob Mildenhall, Brandon Paluzzi, Rosie Gollancz and Sapana Agrawal) we have built at Sleepio for making these past few years amongst the best in my professional career.

In 'retirement' now from Glasgow University (2012) I am proud to be an Emeritus Professor of Clinical Psychology; but equally proud to be starting (again) in February 2013 as a Professor in the Nuffield Department of Clinical Neurosciences at the University of Oxford. The Sleep & Circadian Neuroscience Institute funded there by The Wellcome Trust and the University of Oxford promises much. I hope that I can go some way to helping Professors Russell Foster, Guy Goodwin, Daniel Freeman and many others deliver on that promise.

Finally, I want to acknowledge and thank the people who mean the most in the world to me. Without my family all this would be meaningless. My wife, Audrey is my best friend, stronghold and soul-mate. She is also a better clinical psychologist than I am! My children Craig, Carolyn and Robbie are the best that any dad could ever hope for. I am more proud of them than words can express; and our grand-daughter Mia is certainly the cutest 1-year old ever. Thank you too mum and dad for supporting me, all those years ago, in the idea that psychology was a good career choice, when others had their doubts!

Example is not the main thing in influencing others. It is the only thing.

[Attributed to Albert Schweitzer (1875–1965); lived by Alexander S. Espie (1925–1975)]

Table of Contents

1. General Introduction

1.1 What is insomnia?

Insomnia is characterized in diagnostic nosologies by persistent difficulty in initiating sleep, maintaining sleep, or waking early, or by sleep which is chronically non-restorative despite adequate sleep opportunity (cf. DSM-IV, ICSD-2). The clinical presentation is commonly that of a frustrated patient with a racing mind, trapped in a vicious circle of anxiety and poor sleep, reporting having "tried everything," and generally being unable to down-regulate arousal levels in bed. Insomnia also causes daytime impairments, including fatigue, inattention, and mood changes, and negative effects upon cognition and performance. Research diagnostic criteria (RDC) for insomnia emphasise the importance of assessing both night-time and daytime symptoms. Indeed, concerns about daytime functioning and wellbeing are common drivers for help-seeking behavior. The presence of excessive daytime sleepiness (EDS), however, is relatively unusual in insomnia. When EDS is a prominent complaint, investigations for other sleep disorders should normally be considered, including obstructive sleep apnoea syndrome (OSA), narcolepsy, periodic limb movement disorder (PLMD), and restless legs syndrome (RLS).

Table 1 Differentiation of insomnia phenotypes in ICSD-2 (adapted from Espie & Bartlett, 2009)

Classification	Sleep disorder	Essential features, complaint of insomnia plus
Insomnias	Psychophysiological insomnia	Learned sleep preventing associations, conditioned arousal, 'racing mind' phenomenon
	Paradoxical insomnia	Complaint of poor sleep disproportionate to sleep pattern and sleep duration
	Idiopathic insomnia	Insomnia typically begins in childhood or from birth
	Insomnia due to a mental disorder	Course of sleep disturbance concurrent with mental disorder
	Inadequate sleep hygiene	Daily living activities inconsistent with maintaining good-quality sleep
	Insomnia due to a medical disorder	Course of sleep disturbance concurrent with mental disorder
	Insomnia due to drug or substance	Sleep disruption caused by prescription medication, recreational drug, caffeine, alcohol or foodstuff
	Adjustment insomnia	Presence of identifiable stressor, insomnia resolves or is expected to resolve when stressor is removed

In ICSD-2, specific insomnia phenotypes are proposed (Table 1), with Psychophysiological Insomnia being the most common 'primary' form, presenting in clinical practice, with Paradoxical Insomnia and Idiopathic Insomnia much less common. The diagnosis of Inadequate Sleep Hygiene is seldom used. It should be noted, however, that contemporary thinking (cf DSM-5) reflects the use of the single term 'Insomnia Disorder' (rather than primary vs. secondary) because of mounting evidence that sleep disruption

is more often co-morbid with medical or psychiatric disorders (rather than caused by them). Indeed, insomnia is now a recognized risk factor for the evolution of, or relapse into, disorders like depression.

1.2 How common is insomnia?

Community psychiatric morbidity data show that sleep disturbance is the most common symptom of mental disease, regardless of age, sex, or ethnic group. Indeed insomnia is more common than worry, and twice as common as anxiety or depressive symptoms. Insomnia affects one-third of adults occasionally, and 9–12% on a chronic basis. It is more commonly reported in women, shift workers, and patients with medical and psychiatric disorders. Among older adults, prevalence has been estimated at 25%, although co-morbid conditions and hypnotic drugs are factors in this increased prevalence. Typically, insomnia is associated with impaired work productivity, reduced quality of life and relationship satisfaction, as well as increased ill health. The importance of insomnia to public health is illustrated by national annual costs ($92 to $107 billion USD in USA), and its cost per untreated case ($5,000 CAD in Canada).

1.3 What is the aetiology of insomnia?

Many patients report having been marginal light sleepers before developing insomnia. Sleep disturbance often arises during life change or stress, and such adjustment sleep disorder may represent a normal transient disruption of sleep. However, secondary factors, such as anxiety over sleep and faulty sleep–wake conditioning, may exacerbate and maintain the insomnia as a chronic problem when sleep itself becomes a focus for concern. People with insomnia may be hyperaroused relative to normal sleepers, for example having higher levels of cortisol and ACTH, and also find it difficult to 'down-regulate' their arousal at bedtime.

1.4 What is the course of and prognosis for insomnia?

There has been relatively little research on the natural history of insomnia. However, untreated Psychophysiological Insomnia (PI) can last for decades, and may gradually worsen over time. Indeed, there is a normal developmental trend for sleep patterns to

deteriorate, with increasing age. On the other hand, delayed sleep-phase syndrome (DSPS: a circadian rhythm sleep disorder) and insufficient sleep hygiene can be associated with developmental stage and/or lifestyle factors and may ameliorate as these are resolved. Although certain insomnias tend to persist if untreated, prognosis with effective treatment can be very good.

1.5 How can insomnia be treated?

1.5.1 Pharmacotherapy

Traditionally, insomnia has been treated pharmacologically. Barbiturates were superseded by benzodiazepine (BZ) compounds during the 1960s and 1970s. These drugs were safer in overdose, were thought to have fewer side effects, and to be less addictive. Controlled studies have demonstrated that a considerable number of BZ, of short to intermediate half-life, are effective hypnotic agents. However, from the mid-1970s potential problems became apparent, both during administration and withdrawal. Longer-acting hypnotics were prone to carry-over effects of morning lethargy, and shorter-acting drugs to 'rebound insomnia'. Furthermore, tolerance develops, leading either to increased dosing or switching to alternative medication. Although BZs used for short periods/intermittently can maintain effectiveness, these are not the treatment of choice in chronic insomnia, and are contraindicated in older adults and where insomnia may involve sleep-related breathing disorder because of their potentially depressant effects on respiration. A number of BZ compounds have been removed from the market in the United Kingdom, United States, and elsewhere. Contemporary hypnotic therapy has extended to include BZRAs (benzodiazepine receptor agonists; often referred to as the 'z' drugs), and more recently melatonin receptor agonists (MeRAs) have been introduced. Whereas the place in therapeutics of MeRAs has yet to become established, the BZRAs are often thought to offer more sustained benefit for insomnia, and to have fewer adverse effects. Nevertheless, there remains uncertainty about the effectiveness of BZRAs in chronic insomnia.

Table 2 Summary of CBT components for the treatment of chronic insomnia (adapted from Espie & Bartlett, 2009)

Components of stimulus control and sleep restriction treatment
Define individual sleep requirements
Establish parameters for bedtime period (threshold time and rising time)
Eliminate daytime napping
Differentiate rest from sleep
Schedule sleep periods with respect to needs
Establish 7 day per week compliance
Remove incompatible activity from bedroom environment
Rise from bed if wakeful (>20 min)
Avoid recovery sleep as 'compensation'
Establish stability from night to night
Adjust the sleep period as sleep efficiency improves
Components of cognitive intervention
Identify thought patterns and content that intrude
Address (mis)attributions connecting sleep and waking life
Establish rehearsal/planning time in early evening
Relaxation and imagery training
Distraction and thought blocking
Develop accurate beliefs/attributions about sleep and sleep loss
Challenge negative and invalid thoughts
Eliminate 'effort' to control sleep
Motivate to maintain behaviour and cognitive change
Utilize relapse-prevention techniques

1.5.2 Psychological Therapy

Psychological treatment for chronic insomnia, primarily in the form of cognitive behavioural therapy (CBT), has been extensively investigated in over 100 controlled studies during the past 20 years. Five meta-analyses and numerous systematic reviews have demonstrated that CBT is associated with large effect size changes (measured in standardized *z* scores) in the primary symptom measures of sleep latency (difficulty getting to sleep) and wake time after sleep-onset (difficulty remaining asleep). Around 70 per cent of patients with persistent sleep problems appear to benefit from CBT and effects are maintained to long-term follow-up. It is thought that CBT achieves these outcomes because it tackles directly the dysfunctional thoughts and maladaptive behaviours that otherwise maintain insomnia. Recent controlled studies have shown that

CBT may be effective in general practice settings with nurses delivering the intervention according to a standard protocol. Despite the superior efficacy of CBT relative to medication for insomnia, and these recent demonstrations of CBT working in real-world settings, practical problems remain in making CBT widely available. Within the CBT model, a number of strategies have strong empirical support. Behavioural procedures such as stimulus control and sleep restriction, and cognitive strategies such as paradoxical intention and thought restructuring have been extensively investigated and are outlined in Table 2.

1.5.3 Melatonin, light therapy and exercise

The pineal hormone melatonin has been the subject of highly publicized claims. However, controlled scientific research has been more limited. Several studies support its sleep-promoting effects, especially as a chronobiotic for phase advancing sleep-onset. Several MeRA products are now licensed, and more may be available soon. Bright light is a potent marker for human circadian rhythms, and has been known for some time to enable the resetting of such rhythms in advanced sleep-phase syndrome and delayed sleep-phase syndrome. The results of studies investigating the efficacy of bright light against psychological treatments for psychophysiological insomnia are somewhat preliminary, but nonetheless promising, as is the combination of CBT and light therapy for insomnia. However, a limiting factor to the value of light therapy is that continued treatment may be required to maintain therapeutic effects. Athletic people sleep well, although this may be more to do with behavioural patterning than aerobic fitness. Nevertheless, there is evidence that exercise can have positive effects upon sleep quality, particularly if taken late afternoon or early evening, and in otherwise relatively fit individuals. Morning exercise can also be an effective modality to encourage the same waking time and early morning light exposure; which help to reset sleep patterns on a daily basis.

1.6 What is Behavioural Sleep Medicine?

As defined by the American Academy of Sleep Medicine (AASM) the domain of Behavioral Sleep Medicine (BSM) comprises the behavioural dimension of normal and

abnormal sleep mechanisms and the prevention, assessment, and treatment of sleep disorders and associated behavioural and emotional problems through the application of established principles of behaviour change. From this definition it should be clear that BSM is more than CBT, and that BSM relates to all aspects of sleep disorder, and not just to insomnia.

2. Introduction to the Published Work

2.1 Work included in and excluded from the thesis

The thesis comprises a selected body of work (42 outputs), published during the period 2000-2012, investigating the conceptual basis of Insomnia Disorder, and its evaluation and treatment, principally using cognitive and behavioural interventions. On all data-based papers the author is first, second or last named in the authorship list. The thesis specifically excludes other works by the author, during the same period, on sleep disorders in special populations (e.g. learning disabilities/mental retardation, acquired brain injury, pain), on other sleep disorders (e.g. sleep apnoea) and on other disorders (e.g. epilepsy). The thesis also excludes published studies where the author contributed as a co-investigator to work that was initiated by collaborators.

2.2 Methodological approaches

The work is selected to reflect a range of research methodologies including experimental, psychometric, qualitative and population based studies, and randomised controlled trials. Important theoretical contributions to the literature published in this period are also included and reference is made to major textbooks, position papers, and influential chapter contribution.

2.3 The structure of the thesis

In Chapter 3, papers are first presented within sections pertaining to each of the following 4 major themes:

a. Pathophysiology and conceptualisation of insomnia (section 3.2)
b. Clinical and psychometric appraisal of insomnia (section 3.3)
c. Cognitive behavioural treatment of insomnia (section 3.4)
d. Dissemination of evidence-based practice (section 3.5)

At the start of each section, the inter-relationships between the various papers included in the section are summarized, to help place the work in the context. Papers are then introduced in turn along with a brief synopsis of their impact (see 2.4 below).

Chapter 4 provides a reference list relating to selected citations and Chapter 5 comprises reproduction of the full papers in published form. These papers are of course self-contained, with an abstract, a methods section which describes the approach taken, a results section which describes the data and a discussion which puts these specific findings in context at the time of publication.

2.4 Evaluation of the contribution of the published work

The approach taken was to identify papers which subsequently cited each of the individual outputs. This was achieved by conducting a citations search on the ISI Web of Knowledge website on 10 August 2012. Of course, for some papers, particularly those published recently, there had been relatively little change in the literature since the publication. However, many of the papers in the thesis have been extensively cited. In particular, examples are reported of current or recent citations to demonstrate the enduring impact of the published work.

It should be noted that background knowledge, relevant to placing each published work in context, is summarized briefly, but is not specifically referenced. This is for two reasons. First, it is not the purpose of this evaluation of the published work to present a comprehensive overview of the sleep/insomnia literature; this is available elsewhere [e.g. Morin & Espie, C.A. (2012) The Oxford Handbook of Sleep and Sleep Disorders]; and second, including such references could obscure the cited references that are provided as examples of the impact of the work.

3. The Published Work

3.1 Citation Search

Results from the Web of Knowledge search indicated an Average Citation per Item of 39.8 (range 0 – 215) when taking into account citable papers, across the first three themes. The impact of the fourth theme (on dissemination) is evidenced in other ways.

3.2 Pathophysiology and conceptualisation of insomnia

This section comprises a series of 10 papers investigating behavioural, mental, emotional and physiological aspects of insomnia, and the inter-relationships between them. Average Citation per Item for papers in this section was 32.5 (range 0 – 122).

Studies are theory-testing or theory-building in nature, with the general aim of improved understanding of aetiological and maintaining factors for insomnia disorder. There is a particular focus upon factors that might be modifiable through cognitive behavioural intervention; and the exploration of novel candidate mechanisms for targeting assessment and treatment. Here and elsewhere in the thesis, papers are largely introduced in chronological order of their publication.

3.2.1 Wicklow, A. and Espie, C.A. (2000) Intrusive thoughts and their relationship to actigraphic measurement of sleep: towards a cognitive model of insomnia. *Behaviour Research and Therapy* 38, 679-693 [Cited 78 times]

Cognitive factors seem to be particularly important in the maintenance of insomnia, and perhaps also in its aetiology. Certainly patients presenting clinically complain that falling asleep is, in large measure, a mental obstacle. CBT, therefore addresses not only the behavioural aspects of sleep scheduling (the 'what to do'), but also these cognitive and emotional challenges (how to deal with the mind). Traditionally, such data have been gathered retrospectively on a questionnaire or self-report rating scale, or prospectively on some kind of thought record or diary. However, it is important to validate the content of wakeful thinking in insomnia with 'real time' data; sometimes referred to as 'hot' cognitions. Moreover, model-building for insomnia requires us to investigate the temporal association of such thoughts and with the sleep initiation problems reported by people with insomnia.

In this study, therefore, we developed a novel approach to measure mental events. Participants kept a voice-activated audiotape-recorder at their bedside and were asked to speak aloud what was going through their minds during periods of wakefulness. These thoughts were then content-analysed, themed, and related to objective estimates of sleep, using actigraphy. The actigraph is a small device worn like a wristwatch on the non-dominant hand. It contains a micro-processor/ accelerometer unit that detects and stores movement data in 1-minute bins (epochs). Because movement is a good proxy for wakefulness, and absence of movement for sleep, proprietary software can estimate periods of sleep and wakefulness and so construct an objective profile of the main sleep parameters. Another practical advantage is that actigraphy is relatively non-instrusive.

This study has been cited 72 times and continues to be well cited, even 12 years after publication. For example, recent studies exploring pre-sleep cognitive arousal include Tang et al (2012), Wuyts et al (2012) and Suh et al (2012). Indeed, interventions are increasingly targeting such intrusive and worrisome thinking. Jansson-Fromark et al (2012) investigated whether a constructive worry (CW) intervention added to the effects of behaviour therapy (BT). A randomized, controlled design was used including a 2-week baseline, a 4-week intervention phase (sleep restriction and stimulus control [BT] or sleep restriction and stimulus control plus constructive worry [BT + CW]), and a 2-week follow-up. Compared to BT, BT + CW led to a larger decrease in insomnia severity at all three time points (*d*= 1.10). In comparison with BT, BT + CW also resulted in a larger reduction in worry at two of the time points (*d*= 0.76). The work has been influential not only in reports of adult populations but also in studies exploring mental factors across the lifespan e.g. work on repetitive negative thoughts in children and adolescents (Broeren et al, 2011; Alfano et al, 2010) and cognitive style in older adults aged 65-84 years (Willis et al, 2011).

3.2.2 Espie, C.A. (2002) Insomnia: Conceptual issues in the development, persistence and treatment of sleep disorder in adults. *Annual Review of Psychology* 53, 215-243 [Cited 122 times]

This invited paper posited the Psychobiological Inhibition Model (PIM) for the first time. The PIM is an explanatory and testable conceptualization of insomnia, contrasting

insomnia with normal (involuntary) sleep. Good sleepers seem to sleep without any difficulty. They are not good sleepers because they are (actively) good at sleeping. Rather, they seem passively to allow sleep and circadian drive to do its work. People with insomnia, on the other hand seem to have a highly activated sleep intention. The PIM explains how attentional processing in relation to sleep, and failure to sleep, dysregulates the normal sleep process – much in the same way that concern about breathing causes respiratory dysregulation or anxiety about sexual response promotes impotence.

The PIM draws on the experimental cognitive literature on selective attention to derive objective computerized tests of attentional bias, and to lay out a research agenda to evaluate the model. Our first experiment adapted the emotional Stroop task to investigate attentional bias in people with acute versus persistent insomnia secondary to cancer (Taylor et al, 2003: not included in this thesis). Our subsequent studies employed other task such as the Inducing Change Blindness [ICB: Jones et al, 2005 (3.2.3); Macphee et al, 2006 (3.2.4), Dot Probe: MacMahon et al, 2006 (3.2.6), and Modified Posner: Woods et al, 2009 (3.2.8) paradigms]. A further conceptual piece followed [Espie et al, 2006 (3.2.5)] and a large C-R01 (collaborative) grant from the National Institutes of Health (NIH) was awarded (2007-2012) to take this work forward. The data from this extensive study are being collated at the time of writing.

The influence on the field of the PIM model is evident in that the paper in *Annual Review of Psychology* has been cited 122 times. Other research groups have investigated the model directly using similar attentional paradigms (e.g. Spielberger et al, 2008) and indirectly with other methodologies such as Event Related Potentials (ERP: Bastien et al, 2008a) and the examination of sleep EEG microstructure (Bastien et al, 2008b). The paper has also been widely cited not only in 'primary' insomnia but also in studies of insomnia associated with co-morbidities, and in animal models of insomnia (Cano et al, 2008). To illustrate the influence of the paper, even 10 years after publication, studies citing the model in 2011-2012 include work on insomnia related to osteoarthritic (Von Korff et al, 2012) and other chronic pain (Tang et al, 2011, McCracken et al, 2011),

myocardial infarction (de Zambotti et al, 2011), cancer (Kim and Oh, 2011), anxiety, depression and bipolar illness (e.g. Maroti et al, 2011; Stone et al, 2012; Harvey, 2011).

3.2.3 Jones, B.T., Macphee, L., Broomfield, N.S., Jones, B.C. and Espie, C.A. (2005) Sleep-related attentional bias in good, moderate, and poor (primary insomnia) sleepers. *Journal of Abnormal Psychology* 114, 249-258 [Cited 21 times]

This study was the first in the field to use the ICB paradigm in insomnia research. The task is essentially 'spot-the-difference', where the dependent variable is the number of flickers (transitions between the original stimulus screen, a mask screen, and the changed screen) it takes for the participant to notice which object is missing from the array. The hypothesis is that people with insomnia will have a selective attentional bias towards sleep-related objects, and so will spot a change in one of those objects more quickly than good sleepers, for whom sleep objects are essentially neutral. This study confirmed that people with insomnia display selective attention towards sleep cues, and so lent support to the underlying PIM/ A-I-E model.

The paper has been well cited, for example, by a group in Germany who have engaged in a series of experimental replication and extension studies over several years (Spiegelhalder et al, 2008, 2009, 2010). The paper has been shown to be sound methodologically, and has been included in a major review of sleep misperception (Harvey and Tang, 2012) and a meta-analysis of daytime performance in insomnia (Fortier-Brochu et al, 2012). New variants of the ICB have emerged e.g. its adoption in repeated measures experiments (Moss et al, 2011) and the paper is cited in the development of structural equation approach to insomnia (Schmidt et al, 2010) and the emergence of new priming (Ree and Harvey, 2006) and clock monitoring paradigms (Tang et al, 2007).

3.2.4 Macphee, L.M., Biello, S.M., Broomfield, N.M., MacMahon, K.M.A. & Espie C.A. (2006) Who is pre-occupied with sleep?: A comparison of attention bias in people with Psychophysiologic Insomnia, Delayed Sleep Phase Syndrome and Good Sleepers using the Induced Change Blindness paradigm. *Journal of Sleep Research* 15, 212-221 [Cited 22 times]

In this second ICB study we were concerned to test our PIM/ A-I-E model of insomnia against a clinical comparator condition (DSPS). Perhaps selective attention bias to sleep

cues is an epiphenomenon of having any sleep problem or concern about your sleep, rather than something specific to insomnia? Our results indicated that people with insomnia (a psychophysiological condition) did exhibit selective attention bias, whereas those with DSPS (a chronobiological condition affecting the timing of sleep engagement) did not. These data then supported our hypothesis.

This paper, like Jones et al (3.2.3) has been cited by other research groups who have used our paradigm. Repeat citations will not be mentioned here. Rather, it is noteworthy that this paper led to our work being recognized also in the field of circadian physiology and the treatment of circadian elements of sleep disorders (e.g. Lack and Wright, 2006; Auger et al, 2011). In parallel with our work on objective evaluation of attentional bias, other groups have worked on new self-report measures that may be used in parallel with our computerized tasks [e.g. the Sleep Associated Monitoring Index (SAMI); Chan et al, 2012]. Finally, this study and our broader programme of work have been recognized as contributing to inter-disciplinary developments in cognitive science (Jensen et al, 2011).

3.2.5 Espie, C.A., Broomfield, N.M., MacMahon K.M.A., Macphee, L.M. & Taylor, L.M. (2006) The attention-intention-effort pathway in the development of Psychophysiologic Insomnia: a theoretical review. *Sleep Medicine Reviews* 10, 215-245 [Cited 57 times]

This paper was a sequel to our 2002 paper on the PIM (3.2.2), and it articulated in some detail what we had begun to refer to as the Attention-Intention-Effort pathway from acute/ transient insomnia to persistent psychophysiological insomnia. In brief, the paper describes how normal good sleep becomes upset at times of stress. There is a natural hyperarousal response at that time, which probably confers evolutionary survival value, making the person more alert/ responsive, and thereby also having symptomatic insomnia. The person's focus of attention remains selectively on the perceived source of the stress (e.g. illness, work problems, uncertainty about a specific situation or outcome). This then equates to a transient or adjustment form of insomnia. However, upon resolution of or adaptation to the stressor, sleep may not immediately recover, leading to the person (already primed to be in response mode) to become aware that they are not able to sleep. This switch in attention to the sleep process is then regarded as

challenging the 'Achilles heel' of sleep regulation (that it works well automatically but poorly when we try to sleep). A vicious cycle may then quickly evolve where increased concern about sleep leads to not only attention but to explicit intention and effort to sleep.

The paper, published in *Sleep Medicine Reviews*, has been cited 57 times; that is approximately 10 times per year since publication. Indeed, it remains influential, with 12 citations of the A-I-E model appearing between January and August 2012. One example is the study by Mitchell, Mogg & Bradley (2012) investigating relationships between insomnia, negative emotionality and attention control. They found that insomnia was not only associated with negative emotionality (combination of worry, somatic anxious arousal and rumination), but also with the interactive effect of poor attention control and negative emotionality. Thus, they concluded that poor attention control may further contribute to sleep difficulties in individuals with high negative emotionality. Hamilton et al (2012) have also leaned heavily on the A-I-E pathway in presenting their Sleep and Pain Diatheses model of fibromyalgia (FM). They propose that a wide range of biopsychosocial stressors can set the stage for FM by activating diatheses for sleep disruption and pain sensitivity. They say that sleep disruption, in those most sensitive to pain, initiates a cascade of symptoms that are codified as FM. Once this process is initiated, the symptoms of FM are perpetuated and aggravated by increased vigilance to a broad range of threat-related biopsychosocial stimuli. Thus, it is proposed that sleep is integral to the etiology of FM and also energizes a cognitive feedback loop that maintains or amplifies symptom severity over time.

3.2.6 MacMahon, K.M.A., Broomfield, N.M., Macphee, L.M. and Espie, C.A. (2006) Attention bias for sleep-related stimuli in primary insomnia and delayed sleep phase syndrome. *Sleep* 29, 1420-1427 [Cited 12 times]

Having utilized the ICB task in two studies, here we investigated attentional bias using the Dot Probe paradigm. This is another 'standard' methodology found in the experimental cognitive literature that we were the first to adapt and instrument for the insomnia field. The Dot Probe overcomes some of the methodological criticisms that have been leveled particularly at the Stroop task. The paper provided further evidence

for the specificity of selective attention mechanisms at work in the aetiology of insomnia, because we also included a DSPS clinical comparison group. Like our previous studies this paper was well received, and contributed further evidence in support of the PIM/ A-I-E model.

An indication of the influence of our work on selective attention bias, using cognitive probe tasks such as in this paper, can be found in a recent comprehensive review published in *Psychological Bulletin*. Harvey & Tang (2012) carefully evaluated no less than 13 possible explanations for what they called the puzzle of sleep mis-perception. They concluded that worry and selective attention toward sleep-related threats was a strong contender explanation for why people with insomnia might consistently overestimate night-time wakefulness and underestimate total sleep time, relative to objective measures. An example of a more experimental study is that of Ellis, Gardani & Hogh (2010). This group investigated whether asking participants about their sleep experiences, prior to testing for a perceptual bias, affects responses on a sleep-related ambiguity task. A multivariate analysis showed that the order in which participants completed the task affected the responses on the Insomnia Ambiguity Task with those given the DBAS-10 and Insomnia Severity Index first, showing greater insomnia-related interpretations than those given the Insomnia Ambiguity Task first. This effect was evident only for poor sleepers, consistent with there the notion of a priming effect.

3.2.7 Robertson, J.A., Broomfield, N.B. and Espie, C.A. (2007) Prospective comparison of subjective arousal during the pre-sleep period in primary sleep-onset insomnia and normal sleepers. *Journal of Sleep Research* 16, 230-238 [Cited 9 times]

According to Hyperarousal Theory, people with insomnia have higher arousal levels (somatic and cognitive) than normal sleepers, as a steady state condition on a 24-hour basis, in the peri-sleep-onset period and/or during sleep itself. The Stimulus Control Model suggests that the bed and bedroom environment serves as a discriminative stimulus for wakefulness, rather than for sleep, raising the possibility of insomnia representing a form of conditioned arousal. Despite the prominence of the stimulus control over the past 40 years, there has been virtually no research investigating changes

in arousal associated with the pre-sleep period. In this study, participants with insomnia were compared with a good sleeper control group across 3 time-points (3 hours prior to bed, 1 hour prior to bed, and in bed). They completed the somatic and cognitive sub-scales of the Pre-Sleep Arousal Scale (PSAS) at each time-point, so that we could investigate gradients in experienced arousal across this time-course. The study was the first to demonstrate that, whereas arousal in normal sleepers steadily declines in the evening and particularly during the 1 hour preceding going to bed; in people with insomnia, arousal fails to decrement during this latter transition, with some evidence that it may in fact increase. We suggest that this attenuation in de-arousal measured using the PSAS may reflect difficulties that people with insomnia have in down-regulating their levels of alertness, and so could be associated with greater problems initiating sleep.

The paper has been cited in a wide range of articles including longitudinal work (Fernandez-Mendoza et al, 2011; Keller & El-Sheikh, 2011), treatment trials (Cortoos et al, 2010; Harris et al, 2012) and psychometric reports (Gieselman et al, 2012) as well as being cited in influential reviews (e.g. Lack et al, 2008; Bootzin et al, 2011).

3.2.8 Woods, H., Marchetti, L.M., Biello, S.M., Espie, C.A. (2009) The clock as a focus of selective attention in those with primary insomnia: an experimental study using a modified Posner paradigm. *Behaviour Research & Therapy*, 47,231-236 [Cited 1 time]

This study describes the fourth experimental task that we have developed for investigating cognitive bias in insomnia. The modified Posner paradigm permits greater exploration of components of the attention system (shift, engage, disengage) and so helps to address one of the allocation of attention e.g do threatening stimuli (sleep cues and their emotional salience to a person with insomnia) attract attention by modulating engagement and/ or do they hold attention by modulating the disengagement component.

At the time of writing it is perhaps too early to evaluate the impact of this work as it has only been cited on one occasion.

3.2.9 Baglioni, C., Lombardo, C., Bux, E., Hansen, S., Salveta, C., Biello, S., Violani, C., Espie, C.A. (2010) Psychophysiological reactivity to sleep-related emotional stimuli in primary insomnia *Behaviour Research and Therapy*, 48, 467-475 [Cited 2 times]

As an extension of our work on selective attention bias, where the dependent variable is cognitive/performance based (i.e. a variant of reaction time or response speed), we wanted to investigate physiological reactivity to sleep salient stimuli. This work involved the adaptation of paradigms that have been used in, for example, the anxiety literature, where photographic images are presented on a computer screen and concurrent measures taken of physiological responses. The International Affective Picture System (IAPS) provides normative ratings of emotion (pleasure, arousal dominance) associated with photographs that represent a set of normative emotional stimuli for experimental investigations of emotion and attention.

We selected items from the IAPS for this study, and developed an additional set of sleep positive and sleep negative images as specific probes. The physiological parameters of particular interest to us were the corrugator and zygomatic facial muscles (groups which are activated by the frown and smile response respectively). Thus the study developed and instrumented a new paradigm for the investigation of reactivity to sleep related stimulus.

This paper has so far been cited twice, including meeting criteria for a systematic review, 'Sleep America: Managing the crisis of adult chronic insomnia and associated conditions [Kraus & Rabin (2012), *Journal of Affective Disorders*].

3.2.10 Espie, C.A., Barrie, L.M., Forgan, G.S. (2012) Comparative investigation of the psychophysiological and idiopathic Insomnia Disorder phenotypes: psychological characteristics, patients' perspectives and implications for clinical management. *SLEEP,* 35, 385-393 [Cited 1 times]

This paper is the first ever published to compare the psychophysiological and idiopathic Insomnia Disorder phenotypes in this way. Insomnia in clinical practice is a rather heterogeneous condition, so in order to understand its nature it is crucial to establish and compare different phenotypical features. Idiopathic insomnia refers to lifelong poor

sleep, or at least poor sleep since early childhood. One might expect such individuals to differ from people who were good or normal sleepers when young, but who somehow 'acquired' insomnia in their adult years. Any such differences may also have important implications for treatment, not least which CBT or other psychological [e.g. mindfulness based stress reduction (MBSR) or acceptance and commitment therapy (ACT)] strategies to employ. This study systematically, across two component experiments, investigates psychological characteristics and patient perspectives in these two insomnia groups. Published in March 2012 this paper has so far only been cited once.

3.3 Clinical and psychometric appraisal of insomnia

This section comprises 13 papers on the clinical and research evaluation of insomnia at both the symptom and syndrome level. Average Citation per Item for papers in this section was 40.0 (range 0 – 215).

Studies include psychometric scale development papers to test and validate new self-report instruments, qualitative studies of the patient experience of living with insomnia, and longitudinal evaluation of the persistence of insomnia over time.

3.3.1 Espie, C.A., Inglis, S.J., Harvey, L., and Tessier, S. (2000) Insomniacs' attributions: Psychometric properties of the Dysfunctional Beliefs and Attitudes about Sleep scale and the Sleep Disturbance Questionnaire. *Journal of Psychosomatic Research* 48, 141-148 [Cited 71 times]

People with insomnia complain that they are unable to fall asleep because of a 'racing mind'. That is they attribute insomnia to mental events, more than physical ones (like tension). Therefore, it becomes very important to have valid, reliable and sensitive measures with which to measure attributions, beliefs and attitudes about sleep. Such cognitive phenomena likely play a central role in the maintenance of insomnia.

This paper has been consistently cited since its publication in 2000. It reports further on the psychometric characteristics of two scales, the Dysfunctional Beliefs and Attitudes about Sleep scale (DBAS: Morin, 2003) and the Sleep Disturbance Questionnaire (Espie et

al, 1989) and provides a validated short form (10 items) of the former (DBAS-10), as well as confirmatory principal components analysis (PCA) of the SDQ.

Edinger and Wohlgemuth (2001) conducted a replication of our study, exploring an abbreviated version of the original DBAS (of 30 items). They showed that the DBAS-10 correlated highly with the full DBAS, had respectable internal consistency, effectively discriminated normal sleepers from insomnia sufferers, and detected cognitive changes resulting specifically from CBT intervention. They also found through PCA, three conceptually meaningful DBAS-10 subscales. Their subscale structure varied somewhat from our previous PCA findings, but overall the Edinger and Wohlgemuth report confirmed the validity and clinically utility of the DBAS-10. In a parallel study, they also demonstrated that the DBAS-10 was sensitive to treatment-related change (Edinger et al, 2001). Likewise, Smith & Trinder (2001) reported that the DBAS detected insomnia complaints in a young adult population.

Recently, Chieng-Min et al (2011) explored the association between dysfunctional sleep beliefs and vulnerability to stress-related transient sleep disturbance. One hundred thirty-two good sleepers and 307 poor sleepers were included in this study. They found that poor sleepers showed more dysfunctional beliefs than good sleepers on the DBAS-10, and that, even in good sleepers, DBAS-10 scores positively correlated with vulnerability to stress-related sleep disturbance as measured by the Ford Insomnia Response to Stress Test. Their results, therefore, suggested that dysfunctional sleep belief is not only a perpetuating factor for chronic insomnia, but that it may also serve as a risk factor for stress-related transient insomnia. The SDQ (and the DBAS) are regarded as recommended research tools for the evaluation of insomnia (Buysse et al, 2006).

3.3.2 Miller, A. Espie, C.A. and Scott, J. (2004) The sleep of remitted bipolar outpatients: a controlled naturalistic study using actigraphy. *Journal of Affective Disorders* 80, 145-153 [Cited 35 times]

It has for long been recognised that sleep and circadian function is adversely affected in psychiatric disorders. In Bipolar Disorder (BD), people have episodes of depression, but also at times episodes of mania or hypomania. In both of these phases of this disorder,

sleep may be disturbed. However, there was a question at the time we conducted this study as to whether or not sleep patterns normalised during euthymic periods; that is, when patients were clinically well and free from either depressive or manic symptoms.

We compared a group of patients with diagnosed bipolar illness during this euthymic period, with normal good sleeper control subjects, and obtained objective estimates of sleep-wake pattern using actigraphy. As previously explained, an actigraph is a device worn on the non-dominant wrist, which measures body movement sensitively in one-minute epochs. Data gathered over many consecutive days provide a very good estimate of circadian rhythm, and also of the stability (or instability) of sleep-wake function during the monitored period. The study was the first to report the finding that, even during periods when patients were at their best in terms of mental health, there was considerably more variability in their sleep-wake patterns than normal controls. This raised the possibility that lack of normalisation of sleep-wake function may be a vulnerability factor for recurrent depressive or manic episodes.

This paper has been cited 35 times. It continues to be well cited in studies exploring the euthymic state in BD (Sylvia et al, 2012), and in studies investigating risk factors (Leopold et al, 2012; Jones et al, 2006) and early intervention (Gruber et al, 2009). Harvey's group (University of California, Berkeley) have an international reputation for their work on sleep and BD. They first conducted a replication of our study in 2005 (Harvey et al, 2005), as did Jones et al (2005), and they continue to refer to the influence of our work in clinical studies (most recently, Talbot et al, 2012) and in review articles (Harvey et al, 2008). Our work has been cited also by groups interested in molecular and genetic aspects of affect regulation (e.g. Yang et al, 2009; Etain et al, 2011).

3.3.3 Harvey, K.J. and Espie, C.A. (2004) Development and preliminary validation of the Glasgow Content of Thoughts Inventory (GCTI): a new measure for the assessment of pre-sleep cognitive activity. *British Journal of Clinical Psychology* 43, 409-420 [Cited 10 times]

This paper exploited the novel methodology we had developed in a previous study [Wicklow & Espie, 2000 (3.2.1)] and reported further data on the 'live' cognitions that people with insomnia experience during periods of night-time wakefulness. The paper

goes on to report on the construction, validation, and PCA of a 25-item scale; named the Glasgow Content of thoughts Inventory (GCTI). The GCTI is both a research tool and a clinical tool, to identify the types of thinking that people engage in whilst unable to sleep. Its component structure for example identifies rehearsal and planning thoughts (where people reflect on the day past and think ahead), problem-solving thoughts (where people engage in analysis of life situations; past, present and future), sleep-related and self-monitoring thoughts (where people have heightened self-awareness of themselves, their sleep and its consequences) and thoughts that relate to their immediate environment (e.g. sounds outside). This knowledge and awareness of what people are thinking about during wakeful periods in bed then helps with the tailoring of suitable cognitive interventions.

The scale continues to be cited and used, for example, by the Stanford group (led by Manber) to explore cognitions across insomnia sub-types (Su et al, 2012) and has met criteria for systematic reviews of robust sleep self-report measures (Martoni & Biagi, 2007; Wells et al, 2009). The GCTI is one of the instruments that we included on a CD-ROM of requested scales and measures attached to our clinical handbook (Morin & Espie, 2003).

3.3.4 Edinger, J.D., Bonnet, M.H., Bootzin, R.R., Doghramji, K., Dorsey, C.M, Espie, C.A., Jamieson, A.O., McCall, W.V., Morin, C.M., Stepanski, E.J. (2004) Derivation of Research Diagnostic Criteria for Insomnia: Report of an American Academy of Sleep Medicine Work Group. *Sleep* 27, 1567-1596 [Cited 215 times]

RDC are required to standardize research in any given field. This brings numerous benefits, including increased homogeneity within and between studies and the possibilities of combining data. The process of the RDC work group, however, is also valuable in its own right. Evidence may be consensual, or it may be data-based, so the future research priorities as well as the extant research knowledge become clearer. This AASM commissioned work group, led by Edinger (then of Duke University, North Carolina) published RDC for the insomnia field.

Unsurprisingly perhaps, this 2004 paper has been cited 215 times, and has influenced individual researchers, research groups, companies and policy-makers toward greater

recognition of insomnia as a disorder worthy of attention in its own right. Although RDC do date over time, and these ones will likely be updated in the next year or two, this paper remains influential in defining insomnia for the purposes of epidemiology (e.g. Leger et al, 2011; Sivertsen et al, 2009; Greendale et al, 2010; Joffe et al, 2012), differential and co-morbid diagnosis (Budhiraja et al, 2012), psychometrics (Jansson-Fromark et al, 2012), and pharmacological (e.g. McCall et al, 2009: Joffe et al, 2010; Roth et al, 2011) and behavioural clinical trials (e.g. Manber et al, 2008).

3.3.5 *Kohn, L. and Espie, C.A. (2005) Sensitivity and specificity of measures of the insomnia experience: A comparative study of psychophysiologic insomnia, insomnia associated with mental disorder and good sleep. Sleep 28, 104-112 [Cited 21 times]*

Sensitivity refers to the capacity of a measure to identify index cases of a disorder; normally the people you are trying to select for a research or clinical purpose. Specificity refers to the usefulness of a measure to identify non-cases; that is those who do not have the disorder of interest whom you may be trying to exclude. This paper, investigating the psychometric properties of several scales commonly used in insomnia research and clinical practice, has been influential in the ongoing debate about what characteristics of sleep and sleep-related concern are specific to insomnia in its primary form, relative to insomnia in the context of co-morbid psychiatric disorder. It has been cited on a number of occasions by several, leading research groups. The group led by Kessler for example, who have pioneered the America Insomnia Survey and developed the Brief Insomnia Questionnaire continue to refer to the paper in journals ranging from *Biological Psychiatry* (Hajak et al, 2011; Roth et al, 20111) to *SLEEP* (Kessler et al, 2010). Likewise, Edinger's group (with Carney) have conducted follow-on studies exploring cut-off scores on measures of worry, rumination and psychopathology as potential predictors of insomnia disorders (e.g. Carney et al, 2010, 2011). The paper has been cited also in influential topic reviews (e.g. Manber & Chambers, 2009) and in a systematic review in *Journal of Affective Disorders* aiming to evaluate systems for classifying insomnia and reviewing theoretical models regarding the aetiology and maintaining factors of chronic insomnia (Kraus & Rabin, 2012).

3.3.6 Broomfield, N.M. and Espie, C.A. (2005) Toward a reliable, valid measure of sleep effort. *Journal of Sleep Research* 14, 401-407 [Cited 15 times]

Our A-I-E Model suggests that the end state of persistent psychophysiological insomnia is a sleep pre-occupation and sleep effort syndrome. That is, people get caught up in a self-defeating focus upon sleep and vain effort to achieve sleep. We wanted to have a simple self-report scale that would capture this notion of sleep effort. This paper describes the development and validation of the 6-item Glasgow Sleep Effort Scale (GSES). With most scales that measure psychopathology, one expects to find a significant difference between the normal and clinical populations. For example, people with an anxiety disorder will score much higher on an anxiety scale, but nonetheless the normal population will have some level of resting anxiety. Our A-I-E model, however, would suggest that normal good sleep does not involve any effort whatsoever. Therefore, not only should this scale discriminate people with insomnia from good sleepers; good sleepers would score close to zero, because for them they simply go to bed and fall asleep – nothing else. Our results confirmed these findings.

'Third wave' therapies have become of interest to insomnia researchers over the past 5 years. Unlike CBT, such approaches address the relationship that the person has with their problem. One might, therefore, regard a 'mindful' approach as the opposite of an effortful one. Some of the recent citations have explored this fresh perspective. Howell et al (2011), for example, found that mindfulness mindfulness predicted well-being both directly, and indirectly, through association with self-regulation of sleep. Likewise, Ong & Sholtes (2012) have clinically tested a mindfulness-based therapy for insomnia (MBT-I). In a 12-month follow-up study they showed improvement in sleep associated with MBT-and demonstrated that persistent sleep effort on the GSES was associated with having further episodes of insomnia during follow-up (Ong, Shapiro & Manber, 2009).

3.3.7 Buysse, D.J., Ancoli-Israel, S., Edinger, J.D., Lichstein, K.L. and Morin, C.M. [on behalf of co-authors including Espie, C.A.] (2006) Recommendations for a standard research assessment of insomnia. Sleep, 29, 1155-1173 [Cited 171 times]

This paper was the product of the Pittsburgh Consensus Conference held in 2005, when around 20 leading clinical researchers gathered for a 3 day meeting to agree upon a

standardized approach to data collection. The rationale was that international consistency in the use of clinical tools would advance the field by enabling comparison and replication of studies, and ultimately lead to the pooling of data to answer important research questions relating to the insomnia phenotypes. The fact that the paper has been cited more than 25 times per year since publication indicates that there has been a strong and positive response from the research community.

3.3.8 Waine, J., Broomfield, N.B. & Espie, C.A. (2009) Development and validation of the sleep metacognitive beliefs scale. *Journal of Behaviour Therapy & Experimental Psychiatry*, 40, 15–23 [Cited 1 time]

With the development of cognitive research and therapy, there has been growing interest in the concept of meta-cognition. In this paper, we suggested that two metacognitive belief types might operate in response to the intrusive thinking that is characteristic of insomnia: (i) beliefs concerning the meaning of the intrusions (e.g. thinking in bed prevents me getting to sleep) and (ii) plans that guide and shape the form that cognition takes (e.g. before I fall asleep, I should try and switch off my thoughts). In other branches of psychopathology, measures have been developed to evaluate meta-cognition, and in this paper, we brought this concept into the sleep field, and developed an insomnia-specific measure of meta-cognitive beliefs.

This paper has been cited by Barclay & Gregory (2009) in a study which showed that poor sleepers possess a 'perseverative iterative style' which may predispose them to catastrophize, regardless of content or affective valence; a style previously found to occur more commonly in worriers compared to others.

3.3.9 Kyle, S.D, Morgan, K., Espie, C.A. (2010) Insomnia and health-related quality of life *Sleep Medicine Reviews* 14, 69–82 [Cited 22 times]

In this paper we argue that Health-related Quality of Life (HRQoL), which has become an important construct in contemporary medicine and health care, needs to be actively measured in insomnia, to guage disorder burden and the evaluation of interventions on various aspects of functioning, in a standardized manner. We reviewed the literature on the measurement of HRQoL in insomnia populations, and the extent to which insomnia treatment improves domains of HRQoL, and concluded, from the relatively small

literature available, that insomnia impacts on diverse areas of HRQoL, and that both pharmacological and non-pharmacological interventions can produce, to varying degrees, improvements in domains spanning physical, social and emotional functioning.

The paper contained a call to increased research, and we have been gratified to see that it has been cited 22 times in the past two years. It was cited, for example, in the report on the international Consensus Sleep Diary (Carney et al, 2011) and has been referenced in epidemiological work (Zhang et al, 2012), in studies of sleep and work performance (Swanson et al, 2010) and in pharmacological treatment studies (Perez-Lloret et al, 2012). Other researchers and guideline groups developing measures for use with kidney transplant patients Burkhalter et al (2011) and functional disability (Gradinger et al, 2011) have also cited the paper.

3.3.10 Fleming L, Gillespie S, Espie CA. (2009) The development and impact of insomnia on cancer survivors: a qualitative analysis. *Journal of Psycho Oncology* 19, 991-996 [Cited 3 times]

The clinical trial that we conducted with the support of the CR-UK grant led us to recognise the important part that sleep played in the wellbeing of people who had cancer. Although, at one level, we knew that to be the case, and it was part of our motivation for conducting the trial, we were nonetheless impressed by the informal comments that many of our participants made to us. People told us that being able to sleep well again had been crucial to getting their lives back on track. It was reported to us for example that one person had said to her Professor of Oncology that "CBT for insomnia knocks your chemotherapy into a cocked hat"! In order to explore patients' personal experiences more fully, we obtained a grant from the National Cancer Research Institutes (NCRI) to conduct some qualitative studies on patient experiences. This paper summarises some of the data from that work. In turn, this has led to a 3 year grant from the Breast Cancer Campaign (ongoing at the time of writing) to study the natural history of sleep and sleep disturbance. By completion of that grant in 2013, a total of 250 women will have been followed up from diagnosis through to 1-year follow-up, and we will have novel data on the co-variation of symptoms of insomnia, fatigue, and mood.

This paper has been cited three times to date, including two studies developing protocols for internet CBT for insomnia in cancer survivors (Ritterband et al, 2012; Garland et al, 2011).

3.3.11 Kyle, S.D., Morgan, K., Spiegelhalder, K., & Espie, C.A. (2011) No pain, No gain: An exploratory within-subjects mixed-methods evaluation of the patient experience of sleep restriction therapy. *Sleep Medicine 12, 735-747* [Cited 0 times]

People with insomnia characteristically say that they have "tried everything" to solve their sleep problem. This clinical anecdote alone speaks of the resistant nature of insomnia once it gets established. Scientific data on the natural history of insomnia also bear this out, because more than 70% of people with insomnia still have insomnia one year later. This is in marked contrast, for example, to depression, which is a more naturally remitting disorder. In this context, it is encouraging that CBT is a durably effective treatment for persistent insomnia, and it seems likely that this is because it helps people achieve lasting behavioural, cognitive and attitudinal change. At least this is true for approximately two-thirds of people in clinical trials; but it also raises the question of why some people improve, whereas others do not. This paper on "No pain, no gain" investigated the possibility that home adherence to treatment guidelines (in this case Sleep Restriction Therapy) is crucial to therapeutic benefit. The paper has yet to be cited.

3.3.12 Green, M., Benzeval, M., Espie, C.A., Hunt, K. (2012) The longitudinal course of insomnia symptoms: inequalities by gender and occupational class among two different age cohorts followed for 20 years in the West of Scotland. *SLEEP* 35, 815-823 [Cited 0 times]

In the early 2000s, there was opportunity to introduce formal measurement of sleep quality into a large ongoing longitudinal study being conducted by the Medical Research Council (MRC) team at the Social and Public Health Sciences Unit (SPHSU), University of Glasgow. The Twenty-07 Study was set up in 1986 to investigate the reasons for differences in health by socio-economic circumstances, gender, area of residence, age, ethnic group and family type. Over 4,500 people were followed up for 20 years. The initial wave of data collection took place in 1987/8 when respondents were aged 15, 35 and 55 years. The final wave took place in 2007/8 when respondents were 35, 55 and 75

years old. Thus the Twenty-07 study provides unique opportunities to investigate health and life change over a 20 year span.

This paper is the first output from this study that reports data on sleep quality. Latent Class Analysis (LCA) is used to determine the underlying sleep quality sub-groups that exist in the general population and their trajectory of stability/change over time. It has not yet been cited.

3.3.13 Espie, C.A., Kyle, S.D, Hames, P., Cyhlarova, E., Benzeval, M. (2012) The daytime impact of DSM-5 Insomnia Disorder: comparative analysis of insomnia subtypes from the Great British Sleep Survey (n=11,129) *Journal of Clinical Psychiatry* 73, e1-e7 [Cited 0 times]

The workgroup for DSM-5 has recommended a paradigm shift towards the diagnosis of Insomnia Disorder, whenever diagnostic criteria are met. Thus, in this new nosology (due 2013), Insomnia Disorder may be classified alongside a concurrent mental disorder, physical disorder, or other sleep disorder. Another important feature of DSM-5 is clear specification of daytime impairments that must accompany sleep disturbance. To meet criteria for Insomnia Disorder, therefore, a patient must also complain of significant effects upon energy, mood, interpersonal functioning, concentration, productivity or ability to remain awake.

In this paper, we report on a large sample (n=11,129) of respondents to the Great British Sleep Survey (GBSS). The GBSS was designed to profile DSM-5 Insomnia Disorder, including these six daytime symptoms. The paper compares the daytime impact of Insomnia Disorder, and Insomnia Disorder associated with the various above co-morbidities, with normal sleep. At the time of writing this paper is just published.

3.4 Cognitive behavioural treatment of insomnia

This section comprises a series of 12 papers investigating non-pharmacological treatment of insomnia, using cognitive and behavioural methods. Average Citation per Item for papers in this section was 45.2 (range 1 – 167).

The majority of studies reported are primary outputs from randomized controlled trials, exploring the efficacy and clinical effectiveness of multi-component CBT in insomnia, and insomnia associated with co-morbid health problems. The studies also deal with different methods of CBT delivery (individual, group and online)

3.4.1 Espie, C.A., Inglis, S.J., Tessier, S., and Harvey, L. (2001) The clinical effectiveness of cognitive behaviour therapy for chronic insomnia: Implementation and evaluation of a *Sleep Clinic* in general medical practice. *Behaviour Research and Therapy* 39, 45-60 [Cited 93 times]

Traditionally, CBT is provided a) by a qualified clinical psychologist/psychotherapist, b) on a one-to-one basis in a clinical setting, and c) it is tailored towards the needs of the particular patient. In practice, however, this level of resource may be neither feasible nor necessary. This trial was the first in a series of studies, where we explored the clinical effectiveness of nurse-delivered, small-group CBT, delivered using a standard protocol, in the form of manualised therapy. Consistent with the clinical effectiveness tradition, the CBT intervention was compared with treatment as usual (TAU). Moreover, participants were patients attending primary care practices, and thus relatively unselected in terms of other co-morbidities.

This study, funded by the Chief Scientist Office, Scotland (Novel Health Services Initiative), has been highly cited (93 times over the past 11 years). Our approach has been associated with an interest in increasing access to CBT and a proliferation of studies which have explored new and ways of delivering insomnia treatment. Examples of citations include the short-term training of GPs in the Netherlands to improve primary care treatment approaches (Backhaus et al, 2002), the use of CBT in general practice to reduce long-term dependence on hypnotic drug use in England (Morgan et al, 2003) and to encourage medication tapering in Canada (Morin et al, 2004), the early development of internet-based treatment of insomnia (Sweden; Strom et al, 2004), the development of a comprehensive sleep education programme for dementia patients in the US (USA: McCurry et al, 2005) and group CBT for insomnia secondary to breast cancer (Canada: Savard et al, 2005). The study was also cited in numerous influential reviews in the early years after publication, including the *Lancet* (Sateia and Nowell, 2004) and *JAMA*

(Schenck et al, 2003) and NEJM (Silber, 2003). The study is still well cited in review papers, e.g. a meta-analysis by Belleville et al (2011) in *Clinical Psychology Reviews*, Bootzin et al (2011) [*Annual Review of Psychology*] and Babson et al (2010) [*Psychiatric Clinics of North America*]. In the US, dissemination of CBT to non-sleep specialists has been nationally funded under a Veteran's Health Administration (VHA) programme also cited this work (Manber et al, 2012).

Our early emphasis upon the importance of clinical effectiveness (rather than efficacy) studies has also been supported by others, both conceptually (e.g. Haynes et al, 2010; Morin, 2003) and procedurally through the development of other novel interventions, such as brief behavioural therapy for insomnia (Buysse et al, 2010).

3.4.2 Espie, C.A., Inglis, S.J. and Harvey, L. (2001) Predicting clinically significant response to cognitive behavior therapy (CBT) for chronic insomnia in general practice: analyses of outcome data at 12 months post-treatment. *Journal of Consulting and Clinical Psychology* 69, 58-66 [Cited 40 times]

This paper was a secondary analysis of the data from the above trial, the purpose of which was to determine reliable predictors of who might respond to this form of CBT for insomnia, and who might not. As such, the analyses reported in the paper extend the theme of clinical effectiveness, because the questions that arise for most clinicians are not only "does this treatment work?", but also "is this treatment likely to work for this particular patient?"

This paper was probably the first of its kind to be published in the insomnia field, and it has been well cited. Since publication, similar analyses have been conducted on trial data from other studies, including predictors of treatment response to CBT for insomnia secondary to chronic pain (Currie et al, 2002), predicting long-term outcomes following psychological treatment for hypnotic dependent insomnia (Morgan et al, 2003). Recently, the paper continues to be influential, being cited in relation to pre-therapy dispositions in treatment outcome in older adults with insomnia (Edinger et al, 2008), predictors of the effect of CBT for insomnia co-morbid with breast cancer (Tremblay, 2009), and predictors of outcomes on sleep daytime function and health-related quality of life in medical settings (Van Houdenhove et al, 2011), along with further work on

predicting adherence to sleep intervention to breast cancer chemotherapy (McChargue et al, 2012).

3.4.3 Harvey, L., Inglis, S.J. and Espie, C.A. (2002) Insomniacs' reported use of CBT components and relationship to long-term clinical outcome. *Behaviour Research and Therapy* 40, 75-83 [Cited 26 times]

This study followed up participants at one year post-therapy to investigate which parts of the CBT programme people had been using, which parts they had found helpful, and to explore the relationship between these factors and a structured evaluation of long-term clinical outcome. Interestingly, it was found that it was not always the things that people preferred that helped them most. Indeed, sleep restriction therapy, which is a particularly challenging behavioural regime, was most strongly related to good long-term outcome.

Since 2002 when this paper was published, factors associated with adherence to CBT have been more widely studied, and this paper has been cited in a good number of reports. For example, recently, adherence to CBT for insomnia among women following primary breast cancer treatment was reported by an American team (Matthews et al, 2012). Likewise, also in breast cancer patients, Trembley et al (2009) found that "at 6 month follow-up, subjectively assessed sleep improvements were best predicted by adherence to behavioural strategies." Vincent et al (2008) explored barriers to engagement in sleep restriction and stimulus control, and Arnedt et al (2007) adapted CBT to the particularly challenging scenario of sleep disturbance during alcohol recovery.

3.4.4 Broomfield N., and Espie, C.A. (2003) Initial insomnia and paradoxical intention: an experimental investigation of putative mechanisms using subjective and actigraphic measurement of sleep. *Behavioural and Cognitive Psychotherapy* 31, 313-324 [Cited 16 times]

Paradoxical intention is a psychotherapeutic technique which addresses performance anxiety problems. It has been used in the treatment of insomnia for many years (for an overview see Espie, 2010). This paper fulfilled criteria for inclusion in 2 subsequent systematic reviews (Morin et al, 2006; Smith et al, 2008) and an associated practice parameter paper on the non-pharmacological management of insomnia, published by the American Academy of Sleep Medicine (Morgenthaler et al, 2006). It was also recently

cited in a meta-analysis on the treatment of insomnia and concomitant anxiety disorder (Belleville et al, 2011). The paper has encouraged the development of a novel technique that targets the refocusing of thoughts during the evening in order to improve sleep (Gellis, 2012).

3.4.5 Morin, C.M., Bootzin, R.R., Buysse, D.J., Edinger, J.D., Espie, C.A. & Lichstein, K.L. (2006) Psychological and behavioural treatment of insomnia. Update of the recent evidence (1998-2004) prepared by a Task Force of the American Academy of Sleep Medicine. *Slee*p 29, 1398-1414 [Cited 170 times]

This review group was set up to reconsider the evidence for the efficacy and effectiveness of CBT for Insomnia based upon additional data published in the period 1999-2005. Thus the paper provides an update from an earlier review, also published under the auspices of the AASM in 1999. An expert international panel reviewed all studies that met clinical trial criteria, and the resulting review paper was used as a basis for the AASM Practice Parameter Statement on the Non-Pharmacological Treatment was published in the same issue of the journal (Morgenthaler et al, 2006).

As might be expected, an authoritative review of this type has been very highly cited, and has become a standard reference for people working in the field of clinical insomnia disorder.

3.4.6 Espie, C.A., MacMahon, K.M.A., Kelly, H-L., Broomfield, N.M., Douglas, N.J., Engleman, H.E., McKinstry, B., Morin, C.M. Walker, A. & Wilson, P. (2007) Randomised clinical effectiveness trial of nurse-administered small group CBT for persistent insomnia in general practice. *Sleep* 30, 574-584 [Cited 45 times]

This paper is the primary output, from a second major grant-funded study (Chief Scientist Office, Scotland), evaluating our nurse-delivered small-group CBT model. Again, nurses working in general practice settings, primarily health visitors, were trained to competently deliver manualised CBT during the course of 5, weekly, 50 minute sessions, working with small groups of 5 or 6 patients. As in the previous study, treatment benefits were maintained at follow-up, and the study provided further convincing evidence for the clinical effectiveness of CBT as a first-line insomnia treatment.

The influence of this paper can be evidenced by the fact that in 2011-1012 (4-5 years after publication) it is still being cited by research groups working in the USA (Epstein et al, 2012; Manber et al, 2012; Buysse et al, 2011; Bootzin et al, 2011), Canada (Morin & Benca, 2012), Norway (Bjorvatn et al, 2011), Australia (Harris et al, 2012; Fuller et al, 2011; Kippist et al, 2011), China (Lee et al, 2011) and Japan (Okajima et al, 2011).

3.4.7 Espie, C.A., Fleming, L., Cassidy, J., Samuel, L., Taylor, L.M., White, C.A., Douglas, N.J., Engleman, H.E., Kelly, H-L. & Paul, J. (2008) Randomized controlled clinical effectiveness trial of Cognitive Behavior Therapy versus Treatment as Usual for persistent insomnia in cancer patients. *Journal of Clinical Oncology* 26, 4651-4658 [Cited 50 times]

During the early 2000s, interest grew rapidly in the application of CBT for insomnia in people with co-morbid medical and psychiatric disorders. Hitherto, the dominant wisdom was that insomnia was likely a secondary consequence of other health problems. However, epidemiological data pointed to insomnia as a risk factor for the development of and relapse into depression; and a foremost concern of people with cancer as they tried to re-adjust to normal living after active treatment was completed. Indeed, our PIB/A-I-E model would predict that insomnia symptoms, 'incubated' during times of extreme stress, could become a psychophysiological disorder, self-perpetuating in its own right (Espie, 2002; Espie et al, 2006).

We were encouraged, therefore, to apply to CR-UK for a trial grant to investigate the effectiveness of CBT delivered by cancer nurses to people with persistent insomnia in the aftermath of cancer treatment. The primary output from this research was this paper published in the *Journal of Clinical Oncology*. Our work was highly regarded, and the research team was awarded the Pfizer Prize for the Best Patient Support Initiative in Cancer Care in 2009, presented at the British Oncology Association annual meeting.

Subsequent work in the cancer field has cited this paper 50 times since 2008. These citations have been widespread methodologically, in other RCTs (e.g. Ritterband et al, 2012; Morin et al, 2009; Berger et al, 2009), in meta-analyses (e.g. Kwekkeboom et al, 2010), in papers recommending developments in cancer care (Richardson et al, 2011; Luckett et al, 2010; Berger et al, 2010; Mortimer et al, 2010), in descriptive and

longitudinal studies (e.g. Savard et al, 2011; Shocat & Degan, 2010; Beck et al, 2010) and in conceptual pieces (e.g. Innominato et al, 2009: exploring the clustering of circadian disruption fatigue and anorexia in cancer patients).

3.4.8 Espie, C.A. (2009) 'Stepped care': a health technology solution for delivering Cognitive Behavioral Therapy as a first line insomnia treatment. *Sleep,* 32 (12), 1549-1558 [Cited 29 times]

There is a substantial evidence base for CBT for Insomnia, yet CBT is very seldom available. This paper analyses the challenges associated with delivering CBT to the community on a large scale, and proposes "stepped care" as a health technology that would be capable of achieving this, whilst making best use of resources. It is suggested that CBT can be disseminated simply and effectively in self-help format, for example, using booklets and the internet. This would be the least restrictive and (at least minimally) effective intervention that has an evidence base. A hierarchy above this is proposed, requiring progressively greater levels of expertise and resource, for example, manualised small-group therapy through individualised therapy, to highly specialised evaluation and treatment that might require expertise in behavioural sleep medicine.

The paper has guided thinking about how best to develop and deliver services. For example it is cited in a paper recommending standard procedures for adults in accredited sleep medicine centres in Europe (Fischer et al, 2012), published by the European Sleep Research Society. Likewise it has been cited in studies exploring the practical treatment of insomnia in general practice (Katofsky et al, 2012; Logue et al, 2012) and in clinics for people with arthritis and heart disease (Rybarczyk et al, 2011) and cancer (Savard et al, 2011). The stepped care model has also been applied to student populations (Kloss et al, 2011) and to sleep health awareness screening programmes (Fuller et al, 2011).

3.4.9 Mooney, P., Espie, C.A., Broomfield, N.M. (2009) An Experimental Assessment of a Pennebaker Writing Intervention in Primary Insomnia. *Behavioral Sleep Medicine* 7, 99-105 [Cited 4 times]

Many people with insomnia complain of rehearsing the day that has passed, and planning ahead when they lie awake in bed at night, either initially, or during a subsequent night-time awakening. Such thoughts can be experienced as intrusive and

unwelcome, yet they are not necessarily dysfunctional. Indeed, thinking things through, evaluating events and making sensible plans would be regarded as a good thing. With this in mind, in the late 1980s, we developed a cognitive control strategy, whereby people spent some time in the early part of the evening "putting the day to rest". The more recent work of Pennebaker, working with patients with anxiety disorders and chronic worry, has highlighted the importance of processing emotion that is associated with events and thoughts, and not merely dealing with the thoughts and behaviours per se. He developed what has become known as the Pennebaker Writing Task: a way of helping people to fully express their feelings. In this paper, we brought the Pennebaker Writing Task into the field of insomnia for the first time.

The paper has been cited 4 times to date, in relation to clinical populations (e.g. Jansson-Fromark et al, 2012: an RCT of a constructive worry intervention) and to college student sleep (Arigo & Smyth, 2012).

3.4.10 Wilson, S.J., Nutt, D.J., Alford, C., Argyropoulos, S.V., Baldwin, D.S., Bateson, A.N., Britton, T.C., Crowe, C., Dijk, D-J, Espie, C.A., Gringras, P., Hajak, G., Idzikowski, C., Krystal, A.D., Nash, J.R., Selsick, H., Sharpley, A.L. and Wade, A.G. (2010) British Association for Psychopharmacology consensus statement on evidence-based treatment of insomnia, parasomnias and circadian rhythm disorders. *Journal of Psychopharmacology* 24, 1577–1600 [Cited 20 times]

The British Association of Psychopharmacology (BAP) set up this review group. The approach taken was unusual in two respects. First, it was decided to review evidence for both pharmacological and non-pharmacological approaches; and second, it was decided to broaden the scope of the guideline to include not only insomnia, but also circadian disorders and parasomnias. The resultant paper is broad in its reach, and is an excellent source of information, particularly for clinicians. It is part of a BAP Guidelines series intended for that purpose. Although published only recently, this paper has already been cited 18 times, indicative of the impact of the work. Citations include practice guidelines (e.g. Fischer et al, 2012; Lingford-Hughes et al, 2012) and therapy option reviews (e.g. Buford & Nemeroff, 2012; Thorpy, 2011) and pharmacological treatment studies (e.g. Coe & Hong, 2012; Wichniak et al, 2011).

3.4.11 Espie, C.A., Kyle, S.D, Williams, C., Ong, J.C., Douglas, N.J., Hames, P., Brown, J.S.L. (2012) A randomized, placebo-controlled, trial of online Cognitive Behavioral Therapy for chronic Insomnia Disorder delivered via an automated media-rich web application. *SLEEP* 35, 769-781 [Cited 2 times]

Prior to publication of this paper, there were several smaller scale RCTs of online CBT for insomnia. Results from those studies were encouraging, but the approach had not been tested in a placebo controlled trial; nor on a sizable sample. This paper was also ground-breaking in presenting an online intervention that utilised the full range of rich media (automated interactions, animated therapist, full web and mobile support) and social networking (community) support that is now typically part of the online technology. The results of the study suggested that this form of online CBT (Sleepio; see 3.5.8) may achieve effect sizes that are similar in magnitude to face-to-face therapy, across a range of sleep and daytime function measures. The paper was selected as a rapid publication in the journal *SLEEP*, and there was an associated commentary in the same issue (Ritterband et al, 2012). The paper was only published on 1st June 2012, and so there have been no further citations at the time of writing, other than a commentary in *The Lancet* (Moore, 2012).

3.4.12 Espie, C.A., Kyle, S.D, Miller, C., Williams, C., Ong, J.C., Hames, P. (2013) Randomized, placebo-controlled, trial of online Cognitive Behavioral Therapy for persistent Insomnia Disorder: therapeutic impact upon attribution, cognition and psychopathology *Journal of Consulting & Clinical Psychology* (in press) [Cited 0 times]

This paper reports secondary outcome data from the above randomised placebo-controlled trial of online CBT for Insomnia (3.4.11). Nevertheless, these outcomes are important because thoughts, beliefs and emotions associated with sleep are very important in the aetiology and maintenance of Psychophysiological Insomnia. In comparison to the primary paper, which reports sleep and daytime function outcomes, modest placebo effects were observed on attribution, cognition and psychopathology. However, moderate to large standardised effect sizes in favour of CBT were observed for the CBT–placebo, demonstrating that CBT also has marked effects on people's psychological and emotional approach to sleep over and above the placebo response. At

the time of writing, this paper is “in press” in the leading American Psychological Association journal.

3.5 Dissemination of evidence-based practice relating to insomnia

This section comprises 8 outputs which illustrate professional contributions to the dissemination of good evidence-based practice. The majority of these are books, but exemplar chapters in influential textbooks are also provided. As such, these outputs were not subjected to a citation search. In addition to the text outputs, the section also refers to a web-based programme delivering CBT for insomnia.

3.5.1 Morin C.M and Espie, C.A. (2003) *Insomnia: A Clinical Guide to Assessment and Treatment*. Kluwer Academic/ Plenum Publishers, New York [200 pages] [ISBN 0-306-47750-5]

This clinical handbook was co-written with Charles Morin PhD (Universite Laval, Quebec City) in response to frequent requests for access to our assessment and treatment materials. Written particularly with clinical psychologists and psychological therapists in mind, the book is divided into sections covering clinical assessment and treatment, and is accompanied by a CD-ROM of commonly used clinical tools and procedures. Translations in Italian and Mandarin have also now been published.

Morin C.M and Espie, C.A. (2004) *Insonnia Guida alla valutazione e all'intervento psicologico.* McGraw-Hill Publishing Group Milan [ISBN 88-386-2798-3] [Italian translation]

Morin C.M and Espie, C.A. (2008) *Insomnia: A Clinical Guide to Assessment and Treatment*. DK Vision [ISBN 978-986-191-123-6] [Mandarin translation]

3.5.2 Espie, C.A. (2006) *Overcoming Insomnia and Sleep Problems: A Self-Help Guide Using Cognitive Behavioral Techniques*. Constable & Robinson Ltd., London [ISBN13: 978-184529-070-2/ ISBN10: 1-84529-070-4]

This is a single-authored book that provides ‘guided self-help’ directly to adults who have an insomnia problem. It is part of a highly regarded series of books written by expert authors/clinicians and published by Constable & Robinson. The aim of the series is to make effective CBT (for a wide range of disorders) more directly available to the general public. The book is sub-divided into a background section and a therapeutic section; the

latter following a session-by-session approach, to mirror clinical consultations. It will be available as a Kindle edition from November 2012. It has also now been published in French, Danish, and Italian with a Swedish translation in progress at the time of writing.

Espie, C.A. (2008) *L'insomnie et les Problemes de Sommeil*. InterEditions, Dunod, Paris [ISBN 978-2-10-050115-1] [French translation]

Espie, C.A. (2011) *At Overvinde Søvnløshed*. Forlaget Klim, Aarhus [ISBN 978-87-7955-801-4] [Danish translation]

Espie, C.A. (2013) *Superare L'insonnia*. Eclipsi SRL, Florence [Italian translation]

The book has been well received by the public. For example on www.amazon.co.uk there are 20 reviews; 13 of which give it a 5 star rating (average rating of 4.5). In the media, the book has been described as "*A sensible lucid description of the leading treatment of insomnia by an internationally renowned expert*." Allison G. Harvey PhD, University of Berkeley"

3.5.3 Espie CA, Bartlett D.J. (2009) Insomnia, in R.P. Lisak, D.D. Truong, W. Carroll & R. Bhidayasiri (eds) *International Neurology: A Clinical Approach*. Wiley-Blackwell, Oxford, pp. 554-556

This chapter illustrates contributions made to dissemination to the knowledge and training other health professionals; in this case clinical neurology.

3.5.4 Espie, C.A. Bartlett D.J. (2009) Clinical syndromes of adult psychiatry: Insomnia in M.G. Gelder, J.J. Lopez-Ibor and N.C. Andreasen (eds) *New Oxford Textbook of Psychiatry (Vol. 2)* (second edition) Oxford University Press, Oxford, England, pp. 933-938

Similar to 3.5.3, this chapter was published in the major textbook of psychiatry and illustrates contributions made to psychiatry and the broad mental health field.

3.5.5 Perlis M.L., Shaw, P., Cano, G., Espie, C.A. (2011) Models of Insomnia In MH Kryger, T Roth and WC Dement (eds) *Principles and Practice of Sleep Medicine*, 5th edition. Elsevier Saunders, NY, pp. 850-865

Principles and Practice of Sleep Medicine (now in its 5th edition) is the most influential textbook in the field, and is read by sleep specialists worldwide. This chapter (senior author) provided an overview of the most influential thinking on insomnia disorder, and featured prominently our Psychobiological Inhibition Model, incorporating the Attention-

Intention-Effort pathway. The text is linked to web-site supplementary material where CE is featured as one of the 'Pioneers in Sleep Medicine'.

3.5.6 Espie, C.A. (2011) *Introduction to Coping with Insomnia & Sleep Problems*. Constable & Robinson Ltd., London [32 pages] [ISBN 978-1-84901-620-9]

As a result of demand for the self-help guide (3.5.3), this brief version was commissioned as a booklet that primary health care practitioners, and mental health services could hand out to patients in clinical settings, providing a practicable alternative to offering prescription medication. It is also now available as a Kindle edition.

3.5.7 Morin, C.M. & Espie, C.A. (2012) The Oxford Handbook of Sleep and Sleep Disorders (Oxford Library of Psychology) Oxford University Press, USA [928 pages] [ISBN 978-0-19-537620-3]

In response to a request from the commissioning editor for the Oxford Library of Psychology, my colleague Charles Morin PhD (Université Laval, Quebec City) and I edited this substantial handbook. It is intended to provide a comprehensive, knowledge-based curriculum for the emerging field of behavioural sleep medicine. The handbook comprises over 900 pages and xx chapters, each provided by an eminent world expert incorporating current research and clinical developments in normal and abnormal sleep.

The handbook is in three sections: Section I covers the basics of normal sleep, its functions, and its relationships to emotions, cognitions, performance, psychopathology, and public health and safety issues. Section II addresses abnormal sleep, including disorders like insomnia, parasomnias, circadian rhythm disorders, and sleep apnea. An informed classification of sleep/wake disorders is presented along with a protocol for assessing sleep-wake complaints and evidence-based treatment options. Section III provides a developmental perspective on sleep and sleep problems in childhood, adolescence, and in late life, and a discussion of sleep disturbances in selected special populations.

This first edition was published in 16 March 2012 in print format. An electronic version of the handbook will be available shortly.

3.5.8 www.sleepio.com

The internet provides a pervasive milieu for healthcare delivery. Sleepio is a novel web-based cognitive behavioral therapy course delivered by an animated virtual therapist (The Prof). The program comprises a fully automated media-rich web application, driven dynamically by baseline, adherence, performance, and progress data. At the start of each session, The Prof conducts a progress review with the participant, explores the diary data submitted during the week, the participant's current sleep status and pattern, and progress achieved against goals previously set. Underlying algorithms feed the delivery of information, support, and advice in a personally tailored manner. CBT content is consistent with the literature, and covers behavioural (e.g., sleep restriction, stimulus control) and cognitive (e.g., putting the day to rest, thought re-structuring, imagery, articulatory suppression, paradoxical intention, mindfulness) strategies, as well as additional relaxation strategies (progressive muscle relaxation and autogenic training) and advice on lifestyle and bedroom factors (sleep hygiene). The intervention is based on validated manuals and has been evaluated in a randomized placebo-controlled trial.

The Sleepio programme is available directly online at www.sleepio.com or through Boots stores in the UK.

4. References

Alfano, C. A., Pina, A. A., Zerr, A. A., & Villalta, I. K. (2010). Pre-Sleep Arousal and Sleep Problems of Anxiety-Disordered Youth. *Child Psychiatry & Human Development, 41*(2). doi: 10.1007/s10578-009-0158-5

Ancoli-Israel, S. (2009). Recognition and Treatment of Sleep Disturbances in Cancer. *Journal of Clinical Oncology, 27*(35). doi: 10.1200/jco.2009.24.5993

Arigo, D., & Smyth, J. M. (2012). The benefits of expressive writing on sleep difficulty and appearance concerns for college women. *Psychology & Health, 27*(2). doi: 10.1080/08870446.2011.558196

Arnedt, J. T., Conroy, D. A., & Brower, K. J. (2007). Treatment options for sleep disturbances during alcohol recovery. *Journal of Addictive Diseases, 26*(4). doi: 10.1300/J069v26n04_06

Auger, R. R., Burgess, H. J., Dierkhising, R. A., Sharma, R. G., & Slocumb, N. L. (2011). Light Exposure Among Adolescents With Delayed Sleep Phase Disorder: A Prospective Cohort Study. *Chronobiology International, 28*(10). doi: 10.3109/07420528.2011.619906

Babson, K. A., Feldner, M. T., & Badour, C. L. (2010). Cognitive Behavioral Therapy for Sleep Disorders. *Psychiatric Clinics of North America, 33*(3). doi: 10.1016/j.psc.2010.04.011

Baglioni, C., Lombardo, C., Bux, E., Hansen, S., Salveta, C., Biello, S., . . . Espie, C. A. (2010). Psychophysiological reactivity to sleep-related emotional stimuli in primary insomnia. *Behaviour Research and Therapy, 48*(6). doi: 10.1016/j.brat.2010.01.008

Barclay, N. L., & Gregory, A. M. (2010). The presence of a perseverative iterative style in poor vs. good sleepers. *Journal of Behavior Therapy and Experimental Psychiatry, 41*(1). doi: 10.1016/j.jbtep.2009.08.003

Bastien, C. H., St-Jean, G., Morin, C. M., Turcotte, I., & Carrier, J. (2008). Chronic psychophysiological insomnia: Hyperarousal and/or inhibition deficits? An ERPs investigation. *Sleep, 31*(6) 887-898.

Bastien, C. H., St-Jean, G., Turcotte, I., Morin, C. M., Lavallee, M., & Carrier, J. (2009). Sleep spindles in chronic psychophysiological insomnia. *Journal of Psychosomatic Research, 66*(1). doi: 10.1016/j.jpsychores.2008.05.013

Beck, S. L., Berger, A. M., Barsevick, A. M., Wong, B., Stewart, K. A., & Dudley, W. N. (2010). Sleep quality after initial chemotherapy for breast cancer. *Supportive Care in Cancer, 18*(6). doi: 10.1007/s00520-009-0662-y

Belleville, G., Cousineau, H., Levrier, K., & St-Pierre-Delorme, M.-E. (2011). Meta-analytic review of the impact of cognitive-behavior therapy for insomnia on concomitant anxiety. *Clinical Psychology Review, 31*(4). doi: 10.1016/j.cpr.2011.02.004

Berger, A. M., Abernethy, A. P., Atkinson, A., Barsevick, A. M., Breitbart, W. S., Cella, D., . . . Wagner, L. I. (2010). Cancer-Related Fatigue. *Journal of the National Comprehensive Cancer Network, 8*(8).

Berger, A. M., Kuhn, B. R., Farr, L. A., Von Essen, S. G., Chamberlain, J., Lynch, J. C., & Agrawal, S. (2009). One-Year Outcomes of a Behavioral Therapy Intervention Trial on Sleep Quality and Cancer-Related Fatigue. *Journal of Clinical Oncology, 27*(35).

doi: 10.1200/jco.2008.20.8306
Bjorvatn, B., Fiske, E., & Pallesen, S. (2011). A self-help book is better than sleep hygiene advice for insomnia: A randomized controlled comparative study. *Scandinavian Journal of Psychology, 52*(6). doi: 10.1111/j.1467-9450.2011.00902.x
Bootzin, R. R., & Epstein, D. R. (2011). Understanding and Treating Insomnia. *Annual Review of Clinical Psychology, 7*. doi: 10.1146/annurev.clinpsy.3.022806.091516
Broeren, S., Muris, P., Bouwmeester, S., van der Heijden, K. B., & Abee, A. (2011). The Role of Repetitive Negative Thoughts in the Vulnerability for Emotional Problems in Non-Clinical Children. *Journal of Child and Family Studies, 20*(2). doi: 10.1007/s10826-010-9380-9
Broomfield, N. M., & Espie, C. A. (2003). Initial insomnia and paradoxical intention: an experimental investigation of putative mechanisms using subjective and actigraphiv measurement of sleep. *Behavioural and Cognitive Psychotherapy, 31*(3). doi: 10.1017/s1352465803003060
Broomfield, N. M., & Espie, C. A. (2005). Towards a valid, reliable measure of sleep effort. *Journal of Sleep Research, 14*(4). doi: 10.1111/j.1365-2869.2005.00481.x
Budhiraja, R., Parthasarathy, S., Budhiraja, P., Habib, M. P., Wendel, C., & Quan, S. F. (2012). Insomnia in Patients with COPD. *Sleep, 35*(3). doi: 10.5665/sleep.1698
Buford, U. J., & Nemeroff, C. B. (2012). Current treatment options for insomnia. *Drugs of Today, 48*(6). doi: 10.1358/dot.2012.48.6.1729395
Burkhalter, H., Sereika, S. M., Engberg, S., Wirz-Justice, A., Steiger, J., & De Geest, S. (2011). Validity of 2 sleep quality items to be used in a large cohort study of kidney transplant recipients. *Progress in Transplantation, 21*(1) 27-35.
Buysse, D. J., Ancoli-Israel, S., Edinger, J. D., Lichstein, K. L., & Morin, C. M. (2006). Recommendations for a standard research assessment of insomnia. *Sleep, 29*(9) 1155-1173.
Buysse, D. J., Germain, A., Moul, D. E., Franzen, P. L., Brar, L. K., Fletcher, M. E., . . . Monk, T. H. (2011a). Efficacy of Brief Behavioral Treatment for Chronic Insomnia in Older Adults. *Archives of Internal Medicine, 171*(10). doi: 10.1001/archinternmed.2010.535
Buysse, D. J., Germain, A., Moul, D. E., Franzen, P. L., Brar, L. K., Fletcher, M. E., . . . Monk, T. H. (2011b). Efficacy of Brief Behavioral Treatment for Chronic Insomnia in Older Adults. *Archives of Internal Medicine, 171*(2). doi: 10.1001/archinternmed.2010.535
Cano, G., Mochizuki, T., & Saper, C. B. (2008). Neural circuitry of stress-induced insomnia in rats. *Journal of Neuroscience, 28*(40). doi: 10.1523/jneurosci.1809-08.2008
Carney, C. E., Buysse, D. J., Ancoli-Israel, S., Edinger, J. D., Krystal, A. D., Lichstein, K. L., & Morin, C. M. (2012). The Consensus Sleep Diary: Standardizing Prospective Sleep Self-Monitoring. *Sleep, 35*(2). doi: 10.5665/sleep.1642
Carney, C. E., Harris, A. L., Moss, T. G., & Edinger, J. D. (2010). Distinguishing rumination from worry in clinical insomnia. *Behaviour Research and Therapy, 48*(6). doi: 10.1016/j.brat.2010.03.004
Carney, C. E., Moss, T. G., Harris, A. L., Edinger, J. D., & Krystal, A. D. (2011). Should we be anxious when assessing anxiety using the Beck Anxiety Inventory in clinical

insomnia patients? *Journal of Psychiatric Research, 45*(9). doi: 10.1016/j.jpsychires.2011.03.011

Chan, S.-F., Chen, T.-H., Liao, Y.-M., Chou, K.-R., & Tsai, P.-S. (2012). Development and preliminary validation of the Chinese version of the Sleep-Associated Monitoring Index. *International Journal of Nursing Studies, 49*(1). doi: 10.1016/j.ijnurstu.2011.07.007

Coe, H. V., & Hong, I. S. (2012). Safety of Low Doses of Quetiapine When Used for Insomnia. *Annals of Pharmacotherapy* 35. doi: 10.1345/aph.1Q697

Cortoos, A., De Valck, E., Arns, M., Breteler, M. H. M., & Cluydts, R. (2010). An Exploratory Study on the Effects of Tele-neurofeedback and Tele-biofeedback on Objective and Subjective Sleep in Patients with Primary Insomnia. *Applied Psychophysiology and Biofeedback, 35*(2). doi: 10.1007/s10484-009-9116-z

Currie, S. R., Wilson, K. G., & Curran, D. (2002). Clinical significance and predictors of treatment response to cognitive-behavior therapy for insomnia secondary to chronic pain. *Journal of Behavioral Medicine, 25*(2). doi: 10.1023/a:1014832720903

de Zambotti, M., Covassin, N., Tona, G. d. M., Sarlo, M., & Stegagno, L. (2011). Sleep onset and cardiovascular activity in primary insomnia. *Journal of Sleep Research, 20*(2). doi: 10.1111/j.1365-2869.2010.00871.x

Edinger, J. D., Bonnet, M. H., Bootzin, R. R., Doghramji, K., Dorsey, C. M., Espie, C. A., . . . Stepanski, E. J. (2004). Derivation of research diagnostic criteria for insomnia: Report of an American Academy of Sleep Medicine Work Group. *Sleep, 27*(8) 1567-1596.

Edinger, J. D., Carney, C. E., & Wohlgemuth, W. K. (2008). Pretherapy Cognitive Dispositions and Treatment Outcome in Cognitive Behavior Therapy for Insomnia. *Behavior Therapy, 39*(4). doi: 10.1016/j.beth.2007.12.001

Edinger, J. D., & Wohlgemuth, W. K. (2001). Psychometric comparisons of the standard and abbreviated DBAS-10 versions of the dysfunctional beliefs and attitudes about sleep questionnaire. *Sleep Medicine, 2*(6). doi: 10.1016/s1389-9457(01)00078-8

Edinger, J. D., Wohlgemuth, W. K., Radtke, R. A., Marsh, G. R., & Quillian, R. E. (2001). Does cognitive-behavioral insomnia therapy alter dysfunctional beliefs about sleep? *Sleep, 24*(5) 591-599.

Ellis, J., Gardani, M., & Hogh, H. (2010). Priming affects poor sleepers but not normal sleepers on an Insomnia Ambiguity Task. *Journal of Sleep Research, 19*(1). doi: 10.1111/j.1365-2869.2009.00792.x

Epstein, D. R., Sidani, S., Bootzin, R. R., & Belyea, M. J. (2012). Dismantling Multicomponent Behavioral Treatment for Insomnia in Older Adults: A Randomized Controlled Trial. *Sleep, 35*(6). doi: 10.5665/sleep.1878

Espie, C. A. (2002). Insomnia: Conceptual issues in the development, persistence, and treatment of sleep disorder in adults. *Annual Review of Psychology, 53*. doi: 10.1146/annurev.psych.53.100901.135243

Espie, C.A. (2006) *Overcoming Insomnia and Sleep Problems: A Self-Help Guide Using Cognitive Behavioral Techniques*. Constable & Robinson Ltd., London [ISBN13:

978-184529-070-2/ ISBN10: 1-84529-070-4]

Espie, C. A. (2009). "Stepped Care": A Health Technology Solution for Delivering Cognitive Behavioral Therapy as a First Line Inysomnia Treatment. *Sleep, 32*(12) 1549-1558.

Espie, C.A. (2011) *Introduction to Coping with Insomnia & Sleep Problems*. Constable & Robinson Ltd., London [ISBN978-1-84901-620-9]

Espie, C. A., Barrie, L. M., & Forgan, G. S. (2012). Comparative Investigation of the Psychophysiologic and Idiopathic Insomnia Disorder Phenotypes: Psychologic Characteristics, Patients' Perspectives, and Implications for Clinical Management. *Sleep, 35*(3). doi: 10.5665/sleep.1702

Espie CA, Bartlett D.J. (2009) Insomnia, in R.P. Lisak, D.D. Truong, W. Carroll & R. Bhidayasiri (eds) *International Neurology: A Clinical Approach*. Wiley-Blackwell, Oxford, pp. 554-556

Espie, C.A. Bartlett D.J. (2009) Clinical syndromes of adult psychiatry: Insomnia in M.G. Gelder, J.J. Lopez-Ibor and N.C. Andreasen (eds) *New Oxford Textbook of Psychiatry (Vol. 2)* (second edition) Oxford University Press, Oxford, England pp. 933-938

Espie, C. A., Broomfield, N. M., MacMahon, K. M. A., Macphee, L. M., & Taylor, L. M. (2006). The attention-intention-effort pathway in the development of psychophysiologic insomnia: A theoretical review. *Sleep Medicine Reviews, 10*(4). doi: 10.1016/j.smrv.2006.03.002

Espie, C. A., Fleming, L., Cassidy, J., Samuel, L., Taylor, L. M., White, C. A., . . . Paul, J. (2008). Randomized controlled clinical effectiveness trial of cognitive behavior therapy compared with treatment as usual for persistent insomnia in patients with cancer. *Journal of Clinical Oncology, 26*(28). doi: 10.1200/jco.2007.13.9006

Espie, C. A., Inglis, S. J., & Harvey, L. (2001). Predicting clinically significant response to cognitive behavior therapy for chronic insomnia in general medical practice: Analyses of outcome data at 12 months posttreatment. *Journal of Consulting and Clinical Psychology, 69*(1). doi: 10.1037//0022-006x.69.1.58

Espie, C. A., Inglis, S. J., Harvey, L., & Tessier, S. (2000). Insomniacs' attributions: psychometric properties of the Dysfunctional Beliefs and Attitudes about Sleep Scale and the Sleep Disturbance Questionnaire. *Journal of Psychosomatic Research, 48*(2). doi: 10.1016/s0022-3999(99)00090-2

Espie, C. A., Inglis, S. J., Tessier, S., & Harvey, L. (2001). The clinical effectiveness of cognitive behaviour therapy for chronic insomnia: implementation and evaluation of a sleep clinic in general medical practice. *Behaviour Research and Therapy, 39*(1). doi: 10.1016/s0005-7967(99)00157-6

Espie, C.A., Kyle, S.D, Hames, P., Cyhlarova, E., Benzeval, M. (2012) The daytime impact of DSM-5 Insomnia Disorder: comparative analysis of insomnia subtypes from the Great British Sleep Survey (n=11,129) *Journal of Clinical Psychiatry* 73(12) e1-e7.

Espie, C.A., Kyle, S.D, Miller, C., Williams, C., Ong, J.C., Hames, P. (2013) Randomized, placebo-controlled, trial of online Cognitive Behavioral Therapy for persistent Insomnia Disorder: therapeutic impact upon attribution, cognition and psychopathology *Journal of Consulting & Clinical Psychology* (in press)

Espie, C. A., Kyle, S. D., Williams, C., Ong, J. C., Douglas, N. J., Hames, P., & Brown, J. S. L.

(2012). A Randomized, Placebo-Controlled Trial of Online Cognitive Behavioral Therapy for Chronic Insomnia Disorder Delivered via an Automated Media-Rich Web Application. *Sleep, 35*(6). doi: 10.5665/sleep.1872

Espie, C. A., MacMahon, K. M. A., Kelly, H.-L., Broomfield, N. M., Douglas, N. J., Engleman, H. M., . . . Wilson, P. (2007). Randomized clinical effectiveness trial of nurse-administered small-group cognitive behavior therapy for persistent insomnia in general practice. *Sleep, 30*(5) 574-584.

Etain, B., Milhiet, V., Bellivier, F., & Leboyer, M. (2011). Genetics of circadian rhythms and mood spectrum disorders. *European Neuropsychopharmacology, 21*. doi: 10.1016/j.euroneuro.2011.07.007

Fernandez-Mendoza, J., Calhoun, S. L., Bixler, E. O., Karataraki, M., Liao, D., Vela-Bueno, A., . . . Vgontzas, A. N. (2011). Sleep Misperception and Chronic Insomnia in the General Population: Role of Objective Sleep Duration and Psychological Profiles. *Psychosomatic Medicine, 73*(1). doi: 10.1097/PSY.0b013e3181fe365a

Fischer, J., Dogas, Z., Bassetti, C. L., Berg, S., Grote, L., Jennum, P., . . . Pevernagie, D. (2012). Standard procedures for adults in accredited sleep medicine centres in Europe. *Journal of Sleep Research, 21*(4). doi: 10.1111/j.1365-2869.2011.00987.x

Fleming, L., Gillespie, S., & Espie, C. A. (2010). The development and impact of insomnia on cancer survivors: a qualitative analysis. *Psycho-Oncology, 19*(9). doi: 10.1002/pon.1652

Fortier-Brochu, E., Beaulieu-Bonneau, S., Ivers, H., & Morin, C. M. (2012). Insomnia and daytime cognitive performance: A meta-analysis. *Sleep Medicine Reviews, 16*(1). doi: 10.1016/j.smrv.2011.03.008

Fuller, J. M., Wong, K. K., Krass, I., Grunstein, R., & Saini, B. (2011). Sleep disorders screening, sleep health awareness, and patient follow-up by community pharmacists in Australia. *Patient Education and Counseling, 83*(3). doi: 10.1016/j.pec.2011.05.004

Garland, S. N., Carlson, L. E., Antle, M. C., Samuels, C., & Campbell, T. (2011). I-CAN SLEEP: Rationale and design of a non-inferiority RCT of Mindfulness-based Stress Reduction and Cognitive Behavioral Therapy for the treatment of Insomnia in CANcer survivors. *Contemporary Clinical Trials, 32*(5). doi: 10.1016/j.cct2011.05.014

Gellis, L. A. (2012). An Investigation of a Cognitive Refocusing Technique To Improve Sleep. *Psychotherapy, 49*(2). doi: 10.1037/a0025746

Gieselmann, A., de Jong-Meyer, R., & Pietrowsky, R. (2012). Cognitive and physical arousal in the pre-sleep phase. The German version of the Sleep-Arousal Scale (PSAS). *Zeitschrift Fur Klinische Psychologie Und Psychotherapie, 41*(2). doi: 10.1026/1616-3443/a000134

Gradinger, F., Glaessel, A., Bentley, A., & Stucki, A. (2011). Content comparison of 115 health status measures in sleep medicine using the International Classification of Functioning, Disability and Health (ICF) as a reference. *Sleep Medicine Reviews, 15*(1). doi: 10.1016/j.smrv.2010.07.001

Green, M. J., Espie, C. A., Hunt, K., & Benzeval, M. (2012). The Longitudinal Course of Insomnia Symptoms: Inequalities by Sex and Occupational Class Among Two

Different Age Cohorts Followed for 20 Years in the West of Scotland. *Sleep, 35*(6). doi: 10.5665/sleep.1882

Greendale, G. A., Wight, R. G., Huang, M.-H., Avis, N., Gold, E. B., Joffe, H., . . . Karlamangla, A. S. (2010). Menopause-associated Symptoms and Cognitive Performance: Results From the Study of Women's Health Across the Nation. *American Journal of Epidemiology, 171*(11). doi: 10.1093/aje/kwq067

Gruber, J., Harvey, A. G., Wang, P. W., Brooks, J. O., III, Thase, M. E., Sachs, G. S., & Ketter, T. A. (2009). Sleep functioning in relation to mood, function, and quality of life at entry to the Systematic Treatment Enhancement Program for Bipolar Disorder (STEP-BD). *Journal of Affective Disorders, 114*(1-3). doi: 10.1016/j.jad.2008.06.028

Hajak, G., Petukhova, M., Lakoma, M. D., Coulouvrat, C., Roth, T., Sampson, N. A., . . . Kessler, R. C. (2011). Days-Out-of-Role Associated With Insomnia and Comorbid Conditions in the America Insomnia Survey. *Biological Psychiatry, 70*(11). doi: 10.1016/j.biopsych.2011.08.010

Hamilton, N. A., Atchley, R. A., Karlson, C. W., Taylor, D., & McCurdy, D. (2012). The Role of Sleep and Attention in the Etiology and Maintenance of Fibromyalgia. *Cognitive Therapy and Research, 36*(1). doi: 10.1007/s10608-011-9390-y

Harris, J., Lack, L., Kemp, K., Wright, H., & Bootzin, R. (2012). A Randomized Controlled Trial of Intensive Sleep Retraining (ISR): A Brief Conditioning Treatment for Chronic Insomnia. *Sleep, 35*(1). doi: 10.5665/sleep.1584

Harvey, A. G. (2008). Sleep and circadian rhythms in bipolar disorder: Seeking synchrony, harmony, and regulation. *American Journal of Psychiatry, 165*(7). doi: 10.1176/appi.ajp.2008.08010098

Harvey, A. G. (2011). Sleep and Circadian Functioning: Critical Mechanisms in the Mood Disorders? In S. Nolen-Hoeksema, T. D. Cannon & T. Widiger (Eds.), *Annual Review of Clinical Psychology* (Vol. 7).

Harvey, A. G., & Tang, N. K. Y. (2012). (Mis)perception of Sleep in Insomnia: A Puzzle and a Resolution. *Psychological Bulletin, 138*(1). doi: 10.1037/a0025730

Harvey, K. J., & Espie, C. A. (2004). Development and preliminary validation of the Glasgow Content of Thoughts Inventory (GCTI): A new measure for the assessment of pre-sleep cognitive activity. *British Journal of Clinical Psychology, 43*. doi: 10.1348/0144665042388900

Harvey, L., Inglis, S. J., & Espie, C. A. (2002). Insomniacs' reported use of CBT components and relationship to long-term clinical outcome. *Behaviour Research and Therapy, 40*(1). doi: 10.1016/s0005-7967(01)00004-3

Haynes, P. L., & Bootzin, R. R. (2010). Insomnia Treatments: Moving from Efficacy to Effectiveness. *Journal of Clinical Psychology, 66*(11). doi: 10.1002/jclp.20735

Howell, A. J., Digdon, N. L., & Buro, K. (2010). Mindfulness predicts sleep-related self-regulation and well-being. *Personality and Individual Differences, 48*(4). doi: 10.1016/j.paid.2009.11.009

Innominato, P. F., Mormont, M.-C., Rich, T. A., Waterhouse, J., Levi, F. A., & Bjarnason, G. A. (2009). Circadian Disruption, Fatigue, and Anorexia Clustering in Advanced Cancer Patients: Implications for Innovative Therapeutic Approaches. *Integrative*

Cancer Therapies, 8(4). doi: 10.1177/1534735409355293

Jansson-Frojmark, M., Lind, M., & Sunnhed, R. (2012). Don't worry, be constructive: A randomized controlled feasibility study comparing behaviour therapy singly and combined with constructive worry for insomnia. *British Journal of Clinical Psychology, 51*. doi: 10.1111/j.2044-8260.2011.02018.x

Jansson-Frojmark, M., & Norell-Clarke, A. (2012). Psychometric properties of the Pre-Sleep Arousal Scale in a large community sample. *Journal of Psychosomatic Research, 72*(2). doi: 10.1016/j.jpsychores.2011.10.005

Jensen, M. S., Yao, R., Street, W. N., & Simons, D. J. (2011). Change blindness and inattentional blindness. *Wiley Interdisciplinary Reviews-Cognitive Science, 2*(5). doi: 10.1002/wcs.130

Joffe, H., Chang, Y., Dhaliwal, S., Hess, R., Thurston, R., Gold, E., . . . Bromberger, J. T. (2012). Lifetime History of Depression and Anxiety Disorders as a Predictor of Quality of Life in Midlife Women in the Absence of Current Illness Episodes. *Archives of General Psychiatry, 69*(5) 484-492.

Joffe, H., Petrillo, L., Viguera, A., Koukopoulos, A., Silver-Heilman, K., Farrell, A., . . . Cohen, L. S. (2010). Eszopiclone improves insomnia and depressive and anxious symptoms in perimenopausal and postmenopausal women with hot flashes: a randomized, double-blinded, placebo-controlled crossover trial. *American Journal of Obstetrics and Gynecology, 202*(2). doi: 10.1016/j.ajog.2009.10.868

Jones, B. T., Macphee, L. M., Broomfield, N. M., Jones, B. C., & Espie, C. A. (2005). Sleep-related attentional bias in good, moderate, and poor (primary insomnia) sleepers. *Journal of Abnormal Psychology, 114*(2). doi: 10.1037/0021-843x.114.2.249

Jones, S. H., Hare, D. J., & Evershed, K. (2005). Actigraphic assessment of circadian activity and sleep patterns in bipolar disorder. *Bipolar Disorders, 7*(2). doi: 10.1111/j.1399-5618.2005.00187.x

Jones, S. H., Tai, S., Evershed, K., Knowles, R., & Bentall, R. (2006). Early detection of bipolar disorder: a pilot familial high-risk study of parents with bipolar disorder and their adolescent children. *Bipolar Disorders, 8*(4). doi: 10.1111/j.1399-5618.2006.00329.x

Jones, S. H., & Tarrier, N. (2005). New developments in bipolar disorder. *Clinical Psychology Review, 25*(8). doi: 10.1016/j.cpr.2005.07.001

Katofsky, I., Backhaus, J., Junghanns, K., Rumpf, H.-J., Hueppe, M., von Eitzen, U., & Hohagen, F. (2012). Effectiveness of a cognitive behavioral self-help program for patients with primary insomnia in general practice - A pilot study. *Sleep Medicine, 13*(5). doi: 10.1016/j.sleep.2011.12.008

Keller, P., & El-Sheikh, M. (2011). Children's emotional security and sleep: longitudinal relations and directions of effects. *Journal of Child Psychology and Psychiatry, 52*(1). doi: 10.1111/j.1469-7610.2010.02263.x

Kessler, R. C., Coulouvrat, C., Hajak, G., Lakoma, M. D., Roth, T., Sampson, N., . . . Zammit, G. K. (2010). Reliability and Validity of the Brief Insomnia Questionnaire in the America Insomnia Survey. *Sleep, 33*(11) 1539-1549.

Kim, H. S., & Oh, E. G. (2011). An Explanatory Model for Sleep Disorders in People with Cancer. *Journal of Korean Academy of Nursing, 41*(4).

doi: 10.4040/jkan.2011.41.4.460
Kippist, C., Wong, K., Bartlett, D., & Saini, B. (2011). How do pharmacists respond to complaints of acute insomnia? A simulated patient study. *International Journal of Clinical Pharmacy, 33*(2). doi: 10.1007/s11096-011-9482-5
Kloss, J. D., Nash, C. O., Horsey, S. E., & Taylor, D. J. (2011). The Delivery of Behavioral Sleep Medicine to College Students. *Journal of Adolescent Health, 48*(6). doi: 10.1016/j.jadohealth.2010.09.023
Kohn, L., & Espie, C. A. (2005). Sensitivity and specificity of measures of the insomnia experience: A comparative study of psychophysiologic insomnia, insomnia associated with mental disorder and good sleepers. *Sleep, 28*(1) 104-112.
Kraus, S. S., & Rabin, L. A. (2012). Sleep America: Managing the crisis of adult chronic insomnia and associated conditions. *Journal of Affective Disorders, 138*(3). doi: 10.1016/j.jad.2011.05.014
Kwekkeboom, K. L., Cherwin, C. H., Lee, J. W., & Wanta, B. (2010). Mind-Body Treatments for the Pain-Fatigue-Sleep Disturbance Symptom Cluster in Persons with Cancer. *Journal of Pain and Symptom Management, 39*(1). doi: 10.1016/j.jpainsymman.2009.05.022
Kyle, S. D., Morgan, K., & Espie, C. A. (2010). Insomnia and health-related quality of life. *Sleep Medicine Reviews, 14*(1). doi: 10.1016/j.smrv.2009.07.004
Kyle, S. D., Morgan, K., Spiegelhalder, K., & Espie, C. A. (2011). No pain, no gain: An exploratory within-subjects mixed-methods evaluation of the patient experience of sleep restriction therapy (SRT) for insomnia. *Sleep Medicine, 12*(8). doi: 10.1016/j.sleep.2011.03.016
Lack, L. C., Gradisar, M., Van Someren, E. J. W., Wright, H. R., & Lushington, K. (2008). The relationship between insomnia and body temperatures. *Sleep Medicine Reviews, 12*(4). doi: 10.1016/j.smrv.2008.02.003
Lack, L. C., & Wright, H. R. (2007). Treating chronobiological components of chronic insomnia. *Sleep Medicine, 8*(6). doi: 10.1016/j.sleep.2006.10.003
Lee, H., Lim, Y., Yoo, M.-S., & Kim, Y. (2011). Effects of a Nurse-Led Cognitive-Behavior Therapy on Fatigue and Quality of Life of Patients With Breast Cancer Undergoing Radiotherapy An Exploratory Study. *Cancer Nursing, 34*(6). doi: 10.1097/NCC.0b013e31820d1734
Leger, D., Partinen, M., Hirshkowitz, M., Chokroverty, S., Hedner, J., & QUality, E. E. D. (2010). Characteristics of insomnia in a primary care setting: EQUINOX survey of 5293 insomniacs from 10 countries. *Sleep Medicine, 11*(10). doi: 10.1016/j.sleep.2010.04.019
Leopold, K., Ritter, P., Correll, C. U., Marx, C., Oezguerdal, S., Juckel, G., . . . Pfennig, A. (2012). Risk constellations prior to the development of bipolar disorders: Rationale of a new risk assessment tool. *Journal of Affective Disorders, 136*(3). doi: 10.1016/j.jad.2011.06.043
Lingford-Hughes, A. R., Welch, S., Peters, L., Nutt, D. J., Ball, D., Buntwal, N., . . . Winstock, A. (2012). BAP updated guidelines: evidence-based guidelines for the pharmacological management of substance abuse, harmful use, addiction and comorbidity: recommendations from BAP. *Journal of Psychopharmacology, 26*(7).

doi: 10.1177/0269881112444324

Logue, E. E., Bourguet, C. C., Palmieri, P. A., Scott, E. D., Matthews, B. A., Dudley, P., & Chipman, K. J. (2012). The Better Weight-Better Sleep Study: A Pilot Intervention in Primary Care. *American Journal of Health Behavior, 36*(3). doi: 10.5993/ajhb.36.3.4

Luckett, T., Butow, P. N., King, M. T., Oguchi, M., Heading, G., Hackl, N. A., . . . Price, M. A. (2010). A review and recommendations for optimal outcome measures of anxiety, depression and general distress in studies evaluating psychosocial interventions for English-speaking adults with heterogeneous cancer diagnoses. *Supportive Care in Cancer, 18*(10). doi: 10.1007/s00520-010-0932-8

MacMahon, K. M. A., Broomfield, N. M., & Espie, C. A. (2006). Attention bias for sleep-related stimuli in primary insomnia and delayed sleep phase syndrome using the dot-probe task. *Sleep, 29*(11) 1420-1427.

Manber, R., Carney, C., Edinger, J., Epstein, D., Friedman, L., Haynes, P. L., . . . Trockel, M. (2012). Dissemination of CBTI to the Non-Sleep Specialist: Protocol Development and Training Issues. *Journal of Clinical Sleep Medicine, 8*(2). doi: 10.5664/jcsm.1786

Manber, R., & Chambers, A. S. (2009). Insomnia and Depression: A Multifaceted Interplay. *Current Psychiatry Reports, 11*(6) 437-442.

Manber, R., Edinger, J. D., Gress, J. L., Pedro-Salcedo, M. G. S., Kuo, T. F., & Kalista, T. (2008). Cognitive behavioral therapy for insomnia enhances depression outcome in patients with comorbid major depressive disorder and insomnia. *Sleep, 31*(4) 489-495.

Marchetti, L. M., Biello, S. M., Broomfield, N. M., MacMahon, K. M. A., & Espie, C. A. (2006). Who is pre-occupied with sleep? A comparison of attention bias in people with psychophysiological insomnia, delayed sleep phase syndrome and good sleepers using the induced change blindness paradigm. *Journal of Sleep Research, 15*(2). doi: 10.1111/j.1365-2869.2006.00510.x

Maroti, D., Folkeson, P., Jansson-Frojmark, M., & Linton, S. J. (2011). Does Treating Insomnia With Cognitive-Behavioural Therapy Influence Comorbid Anxiety and Depression? An Exploratory Multiple Baseline Design With Four Patients. *Behaviour Change, 28*(4) 195-205.

Martoni, M., & Biagi, M. (2007). Sleep self-report measures: a literature review. *Epidemiologia E Psichiatria Sociale-an International Journal for Epidemiology and Psychiatric Sciences, 16*(4) 316-329.

Matthews, E. E., Schmiege, S. J., Cook, P. F., Berger, A. M., & Aloia, M. S. (2012). Adherence to Cognitive Behavioral Therapy for Insomnia (CBTI) Among Women Following Primary Breast Cancer Treatment: A Pilot Study. *Behavioral Sleep Medicine, 10*(3). doi: 10.1080/15402002.2012.666220

McCall, W. V., Kimball, J., Boggs, N., Lasater, B., D'Agostino, R. B., Jr., & Rosenquist, P. B. (2009). Prevalence and Prediction of Primary Sleep Disorders in a Clinical Trial of Depressed Patients with Insomnia. *Journal of Clinical Sleep Medicine, 5*(5) 454-458.

McChargue, D. E., Sankaranarayanan, J., Visovsky, C. G., Matthews, E. E., Highland, K. B.,

& Berger, A. M. (2012). Predictors of adherence to a behavioral therapy sleep intervention during breast cancer chemotherapy. *Supportive Care in Cancer, 20*(2). doi: 10.1007/s00520-010-1060-1

McCracken, L. M., Williams, J. L., & Tang, N. K. Y. (2011). Psychological Flexibility May Reduce Insomnia in Persons with Chronic Pain: A Preliminary Retrospective Study. *Pain Medicine, 12*(6). doi: 10.1111/j.1526-4637.2011.01115.x

McCurry, S. M., Gibbons, L. E., Logsdon, R. G., Vitiello, M. V., & Teri, L. (2005). Nighttime insomnia treatment and education for Alzheimer's disease: A randomized, controlled trial. *Journal of the American Geriatrics Society, 53*(5). doi: 10.1111/j.1532-5415.2005.53252.x

Millar, A., Espie, C. A., & Scott, J. (2004). The sleep of remitted bipolar outpatients: a controlled naturalistic study using actigraphy. *Journal of Affective Disorders, 80*(2-3). doi: 10.1016/s0165-0327(03)00055-7

Mitchell, L., Mogg, K., & Bradley, B. P. (2012). Relationships between insomnia, negative emotionality and attention control. *Sleep and Biological Rhythms, 10*(3). doi: 10.1111/j.1479-8425.2012.00567.x

Mooney, P., Espie, C. A., & Broomfield, N. M. (2009). An Experimental Assessment of a Pennebaker Writing Intervention in Primary Insomnia. *Behavioral Sleep Medicine, 7*(2). doi: 10.1080/15402000902762386

Morgan, K., Dixon, S., Mathers, N., Thompson, J., & Tomeny, M. (2003). Psychological treatment for insomnia in the management of long-term hypnotic drug use: a pragmatic randomised controlled trial. *British Journal of General Practice, 53*(497) 923-928.

Morgan, K., Thompson, J., Dixon, S., Tomeny, M., & Mathers, N. (2003). Predicting longer-term outcomes following psychological treatment for hypnotic-dependent chronic insomnia. *Journal of Psychosomatic Research, 54*(1). doi: 10.1016/s0022-3999(02)00569-x

Morgenthaler, T., Kramer, M., Alessi, C., Friedman, L., Boehlecke, B., Brown, T., . . . Swick, T. (2006). Practice parameters for the psychological and behavioral treatment of insomnia: An update. An American Academy of Sleep Medicine Report. *Sleep, 29*(11) 1415-1419.

Morin, C. M. (2003). Measuring outcomes in randomized clinical trials of insomnia treatments. *Sleep Medicine Reviews, 7*(3). doi: 10.1053/smrv.2002.0274

Morin, C. M., Bastien, C., Guay, B., Radouco-Thomas, M., Leblanc, J., & Vallieres, A. (2004). Randomized clinical trial of supervised tapering and cognitive behavior therapy to facilitate benzodiazepine discontinuation in older adults with chronic insomnia. *American Journal of Psychiatry, 161*(2). doi: 10.1176/appi.ajp.161.2.332

Morin, C. M., & Benca, R. (2012). Chronic insomnia. *Lancet, 379*(9821). doi: 10.1016/s0140-6736(11)60750-2

Morin, C. M., Bootzin, R. R., Buysse, D. J., Edinger, J. D., Espie, C. A., & Lichstein, K. L. (2006). Psychological and behavioral treatment of insomnia: Update of the recent evidence (1998-2004). *Sleep, 29*(11) 1398-1414.

Morin C.M and Espie, C.A. (2003) *Insomnia: A Clinical Guide to Assessment and Treatment*. Kluwer Academic/ Plenum Publishers, New York [ISBN 0-306-47750-5]

Morin, C.M. & Espie, C.A. (2012) The Oxford Handbook of Sleep and Sleep Disorders (Oxford Library of Psychology) Oxford University Press, USA [ISBN-10: 019537620X]

Morin, C. M., Vallieres, A., Guay, B., Ivers, H., Savard, J., Merette, C., . . . Baillargeon, L. (2009). Cognitive Behavioral Therapy, Singly and Combined With Medication, for Persistent Insomnia A Randomized Controlled Trial. *Jama-Journal of the American Medical Association, 301*(19) 2005-2015.

Mortimer, J. E., Barsevick, A. M., Bennett, C. L., Berger, A. M., Cleeland, C., DeVader, S. R., . . . Rugo, H. S. (2010). Studying Cancer-Related Fatigue: Report of the NCCN Scientific Research Committee. *Journal of the National Comprehensive Cancer Network, 8*(12) 1331-1339.

Moss, A. C., Albery, I. P., & Sharma, D. (2011). Development of a repeated measures affective change blindness task. *Behavior Research Methods, 43*(3). doi: 10.3758/s13428-011-0072-1

Okajima, I., Komada, Y., & Inoue, Y. (2011). A meta-analysis on the treatment effectiveness of cognitive behavioral therapy for primary insomnia. *Sleep and Biological Rhythms, 9*(1). doi: 10.1111/j.1479-8425.2010.00481.x

Ong, J., & Sholtes, D. (2010). A Mindfulness-Based Approach to the Treatment of Insomnia. *Journal of Clinical Psychology, 66*(11). doi: 10.1002/jclp.20736

Ong, J. C., Shapiro, S. L., & Manber, R. (2008). Combining mindfulness meditation with cognitive-behavior therapy for insomnia: A treatment-development study. *Behavior Therapy, 39*(2). doi: 10.1016/j.beth.2007.07.002

Perez-Lloret, S., Meza Rojas, G., Celia Menoni, M., Ruiz, G., Velasquez, C., Rodriguez, H., . . . Team, P. G. B. S. (2012). Pregabalin Beneficial Effects on Sleep Quality or Health-Related Quality of Life Are Poorly Correlated With Reduction on Pain Intensity After an 8-Week Treatment Course. *Clinical Neuropharmacology, 35*(1). doi: 10.1097/WNF.0b013e31823df2dc

Perlis M.L., Shaw, P., Cano, G., Espie, C.A. (2011) Models of Insomnia In MH Kryger, T Roth and WC Dement (eds) *Principles and Practice of Sleep Medicine*, 5th edition. Elsevier Saunders, NY

Ree, M. J., & Harvey, A. G. (2006). Interpretive biases in chronic insomnia: An investigation using a priming paradigm. *Behavior Therapy, 37*(3). doi: 10.1016/j.beth.2006.03.002

Richardson, A., Addington-Hall, J., Amir, Z., Foster, C., Stark, D., Armes, J., . . . Sharpe, M. (2011). Knowledge, ignorance and priorities for research in key areas of cancer survivorship: findings from a scoping review. *British Journal of Cancer, 105*. doi: 10.1038/bjc.2011.425

Ritterband, L. M., Bailey, E. T., Thorndike, F. P., Lord, H. R., Farrell-Carnahan, L., & Baum, L. D. (2012). Initial evaluation of an Internet intervention to improve the sleep of cancer survivors with insomnia. *Psycho-Oncology, 21*(7). doi: 10.1002/pon.1969

Robertson, J. A., Broomfield, N. M., & Espie, C. A. (2007). Prospective comparison of subjective arousal during the pre-sleep period in primary sleep-onset insomnia and normal sleepers. *Journal of Sleep Research, 16*(2). doi: 10.1111/j.1365-2869.2007.00579.x

Roth, T., Coulouvrat, C., Hajak, G., Lakoma, M. D., Sampson, N. A., Shahly, V., . . . Kessler, R. C. (2011). Prevalence and Perceived Health Associated with Insomnia Based on DSM-IV-TR; International Statistical Classification of Diseases and Related Health Problems, Tenth Revision; and Research Diagnostic Criteria/International Classification of Sleep Disorders, Second Edition Criteria: Results from the America Insomnia Survey. *Biological Psychiatry, 69*(6). doi: 10.1016/j.biopsych.2010.10.023

Rybarczyk, B., Mack, L., Harris, J. H., & Stepanski, E. (2011). Testing Two Types of Self-Help CBT-I for Insomnia in Older Adults With Arthritis or Coronary Artery Disease. *Rehabilitation Psychology, 56*(4). doi: 10.1037/a0025577

Sateia, M. J., & Nowell, P. D. (2004). Insomnia. *Lancet, 364*(9449). doi: 10.1016/s0140-6736(04)17480-1

Savard, J., Ivers, H., Villa, J., Caplette-Gingras, A., & Morin, C. M. (2011). Natural Course of Insomnia Comorbid With Cancer: An 18-Month Longitudinal Study. *Journal of Clinical Oncology, 29*(26). doi: 10.1200/jco.2010.33.2247

Savard, J., Simard, S., Ivers, H., & Morin, C. M. (2005). Randomized study on the efficacy of cognitive-bahavioural therapy for insomnia secondary to breast cancer, part I: Sleep and psychological effects. *Journal of Clinical Oncology, 23*(25). doi: 10.1200/jco.2005.09.548

Savard, J., Villa, J., Simard, S., Ivers, H., & Morin, C. M. (2011). Feasibility of a self-help treatment for insomnia comorbid with cancer. *Psycho-Oncology, 20*(9). doi: 10.1002/pon.1818

Schenck, C. H., Mahowald, M. W., & Sack, R. L. (2003). Assessment and management of insomnia. *Jama-Journal of the American Medical Association, 289*(19). doi: 10.1001/jama.289.19.2475

Schmidt, R. E., Gay, P., Ghisletta, P., & Van der Linden, M. (2010). Linking impulsivity to dysfunctional thought control and insomnia: a structural equation model. *Journal of Sleep Research, 19*(1). doi: 10.1111/j.1365-2869.2009.00741.x

Shochat, T., & Dagan, E. (2010). Sleep disturbances in asymptomatic BRCA1/2 mutation carriers: women at high risk for breast-ovarian cancer. *Journal of Sleep Research, 19*(2). doi: 10.1111/j.1365-2869.2009.00805.x

Silber, M. H. (2005). Chronic insomnia. *New England Journal of Medicine, 353*(8). doi: 10.1056/NEJMcp043762

Sivertsen, B., Krokstad, S., Overland, S., & Mykletun, A. (2009). The epidemiology of insomnia: Associations with physical and mental health. The HUNT-2 study. *Journal of Psychosomatic Research, 67*(2). doi: 10.1016/j.jpsychores.2009.05.001

Smith, M. T., Huang, M. I., & Manber, R. (2005). Cognitive behavior therapy for chronic insomnia occurring within the context of medical and psychiatric disorders. *Clinical Psychology Review, 25*(5). doi: 10.1016/j.cpr.2005.04.004

Smith, S., & Trinder, J. (2001). Detecting insomnia: comparison of four self-report measures of sleep in a young adult population. *Journal of Sleep Research, 10*(3). doi: 10.1046/j.1365-2869.2001.00262.x

Spiegelhalder, K., Espie, C., Nissen, C., & Riemann, D. (2008). Sleep-related attentional bias in patients with primary insomnia compared with sleep experts and healthy

controls. *Journal of Sleep Research, 17*(2). doi: 10.1111/j.1365-2869.2008.00641.x

Spiegelhalder, K., Espie, C., & Riemann, D. (2009). Is sleep-related attentional bias due to sleepiness or sleeplessness? *Cognition & Emotion, 23*(3). doi: 10.1080/02699930802022129

Spiegelhalder, K., Kyle, S. D., Feige, B., Prem, M., Nissen, C., Espie, C. A., & Riemann, D. (2010). The Impact of Sleep-Related Attentional Bias on Polysomnographically Measured Sleep in Primary Insomnia. *Sleep, 33*(1) 107-112.

Strom, L., Pettersson, R., & Andersson, G. (2004). Internet-based treatment for insomnia: A controlled evaluation. *Journal of Consulting and Clinical Psychology, 72*(1). doi: 10.1037/0022-006x.72.1.113

Suh, S., Ong, J. C., Steidtmann, D., Nowakowski, S., Dowdle, C., Willett, E., . . . Manber, R. (2012). Cognitions and Insomnia Subgroups. *Cognitive Therapy and Research, 36*(2). doi: 10.1007/s10608-011-9415-6

Swanson, L. M., Arnedt, J. T., Rosekind, M. R., Belenky, G., Balkin, T. J., & Drake, C. (2011). Sleep disorders and work performance: findings from the 2008 National Sleep Foundation Sleep in America poll. *Journal of Sleep Research, 20*(3). doi: 10.1111/j.1365-2869.2010.00890.x

Sylvia, L. G., Dupuy, J. M., Ostacher, M. J., Cowperthwait, C. M., Hay, A. C., Sachs, G. S., . . . Perlis, R. H. (2012). Sleep disturbance in euthymic bipolar patients. *Journal of Psychopharmacology, 26*(8). doi: 10.1177/0269881111421973

Talbot, L. S., Stone, S., Gruber, J., Hairston, I. S., Eidelman, P., & Harvey, A. G. (2012). A Test of the Bidirectional Association Between Sleep and Mood in Bipolar Disorder and Insomnia. *Journal of Abnormal Psychology, 121*(1). doi: 10.1037/a0024946

Tang, N. K. Y., Goodchild, C. E., Hester, J., & Salkovskis, P. M. (2012). Pain-related Insomnia Versus Primary Insomnia A Comparison Study of Sleep Pattern, Psychological Characteristics, and Cognitive-behavioral Processes. *Clinical Journal of Pain, 28*(5). doi: 10.1097/AJP.0b013e31823711bc

Tang, N. K. Y., Goodchild, C. E., Sanborn, A. N., Howard, J., & Salkovskis, P. M. (2012). Deciphering the Temporal Link between Pain and Sleep in a Heterogeneous Chronic Pain Patient Sample: A Multilevel Daily Process Study. *Sleep, 35*(5). doi: 10.5665/sleep.1830

Tang, N. K. Y., Schmidt, D. A., & Harvey, A. G. (2007). Sleeping with the enemy: Clock monitoring in the maintenance of insomnia. *Journal of Behavior Therapy and Experimental Psychiatry, 38*(1). doi: 10.1016/j.jbtep.2005.07.004

Taylor, L.M., Espie, C.A. & White, C.A. (2003) A comparative experimental investigation of attentional bias in people with acute versus persistent insomnia secondary to cancer. *Behavioral Sleep Medicine* 1, 200-212

Thorpy, M. (2011). Understanding and Diagnosing Shift Work Disorder. *Postgraduate Medicine, 123*(5). doi: 10.3810/pgm.2011.09.2464

Tremblay, V., Savard, J., & Ivers, H. (2009). Predictors of the Effect of Cognitive Behavioral Therapy for Chronic Insomnia Comorbid With Breast Cancer. *Journal of Consulting and Clinical Psychology, 77*(4). doi: 10.1037/a0015492

Van Houdenhove, L., Buyse, B., Gabriels, L., & Van den Bergh, O. (2011). Treating Primary Insomnia: Clinical Effectiveness and Predictors of Outcomes on Sleep, Daytime

Function and Health-Related Quality of Life. *Journal of Clinical Psychology in Medical Settings, 18*(3). doi: 10.1007/s10880-011-9250-7

Vincent, N., Lewycky, S., & Finnegan, H. (2008). Barriers to Engagement in Sleep Restriction and Stimulus Control in Chronic Insomnia. *Journal of Consulting and Clinical Psychology, 76*(5). doi: 10.1037/0022-006x.76.5.820

Von Korff, M., Vitiello, M. V., McCurry, S. M., Balderson, B. H., Moore, A. L., Baker, L. D., . . . Rybarczyk, B. D. (2012). Group interventions for co-morbid insomnia and osteoarthritis pain in primary care: The lifestyles cluster randomized trial design. *Contemporary Clinical Trials, 33*(4). doi: 10.1016/j.cct.2012.03.010

Waine, J., Broomfield, N. M., Banham, S., & Espie, C. A. (2009). Metacognitive beliefs in primary insomnia: Developing and validating the Metacognitions Questionnaire-Insomnia (MCQ-I). *Journal of Behavior Therapy and Experimental Psychiatry, 40*(1). doi: 10.1016/j.jbtep.2008.03.002

Wells, G. A., Li, T., Kirwan, J. R., Peterson, J., Aletaha, D., Boers, M., . . . Tugwell, P. S. (2009). Assessing Quality of Sleep in Patients with Rheumatoid Arthritis. *Journal of Rheumatology, 36*(9). doi: 10.3899/jrheum.090362

Wichniak, A., Wierzbicka, A., & Jernajczyk, W. (2011). Patients with insomnia and subthreshold depression show marked worsening of insomnia after discontinuation of sleep promoting medication. *Progress in Neuro-Psychopharmacology & Biological Psychiatry, 35*(7). doi: 10.1016/j.pnpbp.2011.06.010

Wicklow, A., & Espie, C. A. (2000). Intrusive thoughts and their relationship to actigraphic measurement of sleep: towards a cognitive model of insomnia. *Behaviour Research and Therapy, 38*(7). doi: 10.1016/s0005-7967(99)00136-9

Willis, T. A., Yearall, S. M., & Gregory, A. M. (2011). Self-reported Sleep Quality and Cognitive Style in Older Adults. *Cognitive Therapy and Research, 35*(1). doi: 10.1007/s10608-009-9270-x

Wilson, S. J., Nutt, D. J., Alford, C., Argyropoulos, S. V., Baldwin, D. S., Bateson, A. N., . . . Wade, A. G. (2010). British Association for Psychopharmacology consensus statement on evidence-based treatment of insomnia, parasomnias and circadian rhythm disorders. *Journal of Psychopharmacology, 24*(11). doi: 10.1177/0269881110379307

Woods, H., Marchetti, L. M., Biello, S. M., & Espie, C. A. (2009). The clock as a focus of selective attention in those with primary insomnia: An experimental study using a modified Posner paradigm. *Behaviour Research and Therapy, 47*(3). doi: 10.1016/j.brat.2008.12.009

Wuyts, J., De Valck, E., Vandekerckhove, M., Pattyn, N., Bulckaert, A., Berckmans, D., . . . Cluydts, R. (2012). The influence of pre-sleep cognitive arousal on sleep onset processes. *International Journal of Psychophysiology, 83*(1). doi: 10.1016/j.ijpsycho.2011.09.016

Yang, C.-M., Chou, C. P.-W., & Hsiao, F.-C. (2011). The Association of Dysfunctional Beliefs About Sleep With Vulnerability to Stress-Related Sleep Disturbance in Young Adults. *Behavioral Sleep Medicine, 9*(2). doi: 10.1080/15402002.2011.557990

Yang, S., Van Dongen, H. P. A., Wang, K., Berrettini, W., & Bucan, M. (2009). Assessment

of circadian function in fibroblasts of patients with bipolar disorder. *Molecular Psychiatry, 14*(2). doi: 10.1038/mp.2008.10

Zhang, J., Lam, S. P., Li, S. X., Yu, M. W. M., Li, A. M., Ma, R. C. W., . . . Wing, Y. K. (2012). Long-term outcomes and predictors of chronic insomnia: A prospective study in Hong Kong Chinese adults. *Sleep Medicine, 13*(5). doi: 10.1016/j.sleep.2011.11.015

5. Appendix of the published work

Pathophysiology and conceptualisation of insomnia

Behaviour Research and Therapy 2000*,* 38, 679-693

Annual Review of Psychology 2002*,* 53, 215-243

Journal of Abnormal Psychology 2005, 114, 249-258

Journal of Sleep Research 2006, 15, 212-221

Sleep Medicine Reviews 2006, 10, 215-245

SLEEP 2006, 29, 1420-1427

Journal of Sleep Research 2007, 16, 230-238

Behaviour Research & Therapy 2009, 47,231-236

Behaviour Research &Therapy 2010, 48, 467-475

SLEEP 2012, 35, 385-393

PERGAMON

Behaviour Research and Therapy 38 (2000) 679–693

BEHAVIOUR RESEARCH AND THERAPY

www.elsevier.com/locate/brat

Intrusive thoughts and their relationship to actigraphic measurement of sleep: towards a cognitive model of insomnia

A. Wicklow, C.A. Espie*

University of Glasgow, Department of Psychological Medicine, Gartnavel Royal Hospital, 1055 Great Western Road, Glasgow G12 0XH, UK

Abstract

Although cognitive over-arousal has been hypothesised as a causal factor in sleep-onset insomnia, relatively little is known about the specific pre-sleep intrusions which delay sleep. To investigate this relationship adequately 'live', verifiable, unobtrusive and independent monitoring of thought process and sleep pattern is essential. This study was designed with these requirements in mind. Voice-activated audiotape recordings of spontaneous thoughts, and actigraphic data from which to estimate sleep parameters, were obtained over three consecutive nights from 21 participants (63 subject nights). Content analysis of transcribed audiotapes yielded eight categories of pre-sleep intrusion. Results from correlational and regression analyses indicate that thinking about sleep and the anticipated consequences of poor sleep, along with general problem-solving are the strongest predictors of objective sleep latency. Principal Components Analysis suggests that intrusions can be subsumed under one of three factors: 'active problem-solving', 'present state monitoring' and 'environmental reactivity'. Implications for cognitive models and treatments of insomnia are discussed. © 2000 Elsevier Science Ltd. All rights reserved.

Keywords: Sleep-onset; Cognitive intrusions; Actigraphy

1. Introduction

Insomnia, defined as difficulty initiating or maintaining sleep (American Sleep Disorders

* Corresponding author.

PII: S0005-7967(99)00136-9

Association, 1997), presents in 30% of the adult and older population occasionally, and in 10–15% on a chronic basis (Foley et al., 1995; Gallup Organisation, 1991; Oyahon, Caulet & Guilleminault, 1997). Clearly, insomnia is a severe and widespread complaint. However, its origins are only partially understood.

1.1. Cognitive arousal and insomnia

Investigation of the relative influence of cognitive and physiological arousal, consistently associates the former more strongly with sleep disruption, and 'having an overactive mind' has been the attribution of poor sleep rated most highly, both by insomniacs and non-insomniacs (Broman and Hetta, 1994; Espie, Brooks & Lindsay, 1989; Evans, 1977; Lichstein & Rosenthal, 1980; Nicassio, Mendlowitz, Fussel & Petras, 1985). However, there have been conflicting conclusions from studies investigating the relationship between pre-sleep cognitive activity and different measures of sleep latency. Van Egeren, Haynes, Franzen & Hamilton (1983) found that cognitions were significantly correlated with subjective sleep latency, but not with electroencephalographic (EEG) sleep; whereas Borkovec, Grayson, O'Brien and Weerts (1979) and Kuisk, Bertelson and Walsh (1989) reported more frequent cognitive activity in objective insomniacs (i.e. sleep disturbance confirmed by EEG data) than in subjective insomniacs. Sanavio (1988) reported a very low product–moment correlation ($r = 0.09$) between pre-sleep intrusion and self-reported sleep latency, and furthermore, found no specific advantage of a tailored cognitively focused program in the treatment of sleep-onset insomnia. The possibility, therefore, remains that cognitive arousal is simply an epiphenomenon of night-time wakefulness (Freedman and Sattler, 1982; Morin, 1993).

Support for a cognitive model has also come from studies where significant sleep difficulties have been induced through the experimental manipulation of cognitive intrusions in the pre-sleep period (e.g. Gross and Borkovec, 1982; Hall et al., 1996). However, Haynes, Adams and Franzen (1981) exposed insomniacs and non-insomniacs to brief cognitive stressors and found a decrease in subjective and objective sleep-onset latency among insomniacs on stress nights. They concluded that a mental processing task which disrupts sleep-related cognitive events may, as a result, decrease sleep-onset latency, implying that the *nature* of the intrusive cognitions may be a critical factor in the final effect upon sleep. It seems possible, therefore, that failure to differentiate thought content could result in limited comparability betwen studies.

1.2. Nature of pre-sleep cognitions

Insomniacs tend to have more negative thoughts than good sleepers at bedtime (Kuisk et al., 1989; Nicassio et al., 1985; Van Egeren et al., 1983) and such thinking processes remain part of their experience even when wakened from light sleep (Borkovec, Lane & Van Oot, 1981). There is evidence that the thoughts of insomniacs may be dependent on emotional state. In a study investigating the relationship between worry and insomnia, Watts, Coyle and East (1994) found that much of the pre-sleep mental activity of 'worried insomniacs' revolved around work-related issues and general mental activity. In contrast, thoughts of 'non-worried insomniacs' tended to focus on problems they were having with the sleep process itself.

Insomniacs also feel less in control of their thinking (Watts, East & Coyle, 1995). Gendron, Blais and Morin (1998) have reported that insomniacs with co-morbid Generalised Anxiety Disorder (GAD) had higher levels of cognitive activity at bedtime than insomniacs without GAD, evaluated their thoughts as more intrusive and worrisome and attempted cognitive avoidance strategies more frequently.

Espie and Wicklow (1999) have proposed a treatment model for insomnia, comprising four pre-requisites for successful cognitive de-arousal. First, a reduction of the 'business/work' of the mind; second, a reduction of 'effort/control over sleep' itself; third, a reduction of 'affect/anxiety'; and fourth, a reduction of 'mental acuity/thought readiness'. Morin (1993) similarly has argued that dysfunctional beliefs and attitudes about sleep play a critical role in the maintenance of night wakefulness. However, the available literature concerning the *actual thought content* of insomniacs during sleep-onset is insufficiently detailed to test such models. Formal analysis of sleep-interfering cognitions has been reported in only three studies. Coyle and Watts (1991) used an extended 30-item version of the Sleep Disturbance Questionnaire (Espie et al., 1989), and reported two distinct cognitive factors. One was labelled 'sleep attitudes' and reflected anxiety about the sleep process; the other was labelled 'mental activity' reflecting non-specific cognitive activity. Six factors of night-time intrusive thoughts, i.e. mental activity and rehearsal; thoughts about sleep; family and long-term concerns; positive plans and concerns; somatic preoccupations; and work and recent concerns, were identified in a later study of young adults by Watts et al. (1994). An extensive study by Fichten et al. (1998) of the thoughts of older adults (mean age 68 years) during wakeful periods in bed yielded a three factor solution of generalized positive thinking, generalized negative thinking and thoughts related to sleep. They suggested that insomniacs may use positive thinking as a 'buffer' in an attempt to combat more negative intrusions.

1.3. Methodological issues in previous research

In many studies cognitive arousal has been measured by self-report questionnaire (e.g. Gendron et al., 1998; Sanavio, 1988) such as the Pre-Sleep Arousal Scale (PSAS; Nicassio et al., 1985), or by checklists of thought content (e.g. Watts et al., 1994). However, such measures may not adequately assess intrusions for two reasons. First, they limit responses to a *predetermined* set of items; and second, they are *retrospective* reports completed the following morning. Sleep Diary estimates of sleep (e.g. Fichten et al., 1998; Gendron et al., 1998; Sanavio, 1988) also present problems; first, due to their *limited reliability* as indices of underlying sleep pattern; and second, because of their inherently *subjective* nature. They are of course also retrospective measures. However, apart from issues of validity and reliability with these measures when considered separately, the main problem arises when *comparing* information on intrusions and information on sleep. It is simply not possible to regard these data as sufficiently independent to make reasonable judgements concerning relationships between them. Clearly, what is required is some form of 'live' and verifiable independent monitoring both of thought process and of sleep pattern.

Some workers have attempted to gather direct information on thought content and objective data on sleep. Van Egeren et al.'s (1983) subjects provided thought reports in response to a random series of tape-recorded beeps, and Kuisk et al. (1989) conducted brief interviews at

4 min intervals during the pre-sleep period. Although attempts were made in both these laboratory-based studies to be minimally intrusive, the procedures had limited ecological validity. Polysomnography (PSG) is the recognised 'gold standard' for measuring sleep, and has been employed in studies of pre-sleep cognitive activity (e.g. Broman and Hetta, 1994; Kuisk et al., 1989; Van Egeren et al., 1983). However, for this purpose PSG may introduce an experimental effect. First, PSG is physically intrusive involving electrode placement. Second, it introduces the subject to a different environment (even if home recording is possible the stimulus environment is altered). Third, and most importantly, PSG may influence reported thought content towards situational/procedural rather than internal factors. An alternative method for assessing sleep parameters, which is reliable and minimally intrusive, is the wrist actigraph. The actigraph is a small device attached to a wrist strap. Internal motion sensors record the wearer's movements and these data can be downloaded for analysis. Although the actigraph solely measures activity, it is increasingly recognised that movement is a good predictor of wakefulness and lack of movement a good predictor of sleep (American Sleep Disorders Association, 1995; Sadeh, Hauri, Kripke & Lavie, 1995). Measures derived from the wrist actigraph have been found to correlate around $r = 0.80$ to 0.95 with PSG measures (Kripke, Mullaney, Messin & Wyborney, 1978; Mullaney, Kripke & Messin, 1980; Sadeh et al., 1995).

In this study, cognitions were obtained as they arose using voice-activated audiorecording and sleep was measured using the wrist actigraph. It was considered that this methodology would be minimally intrusive, clinically and ecologically valid, being carried out in the subject's own environment, and yet being self-administered, would eliminate other extraneous influences on sleep. The purpose of the study was, firstly, to determine the nature and content of pre-sleep cognitions reported by people who complain of significant disturbance in falling asleep; and secondly, to investigate their effect (both in terms of amount and content of thinking) on subjective and objective measures of sleep.

2. Method

2.1. Subjects

Subjects were recruited through advertisement in a University staff newsletter, and notices placed in the Department. Subjects were included if they were aged between 16 and 65 years, and if they complained of clinically significant problems in falling asleep, according to International Classification of Sleep Disorders criteria, i.e. sleep latency of 30 min occurring on four out of seven nights, with or without disruption to other sleep variables (American Sleep Disorders Association, 1997), and scored in excess of 5 on the the Pittsburgh Sleep Quality Index (Buysse, Reynolds, Monk, Berman & Kupfer, 1989). Subjects were excluded if they were receiving treatment for sleep difficulties; suffering from medical conditions known to impact on sleep; suffering from a psychopathological disorder; or if they suffered intermittent awakenings without a difficulty in getting to sleep. Potential subjects completed a telephone screening interview to determine eligibility for inclusion. Twenty-one suitable participants were identified (14 female, seven male) with mean age of 36 years and average duration of sleep disturbance of

10 years (Table 1). Prior to investigation, subjects received an information leaflet on the study to which an informed consent slip was attached, and several intake questionnaires, including a Sleep History Questionnaire (from Morin, 1993) to complete and return.

2.2. Measures

Four main measures were used in this study:-

1. The subjective assessment of sleep consisted of a Sleep Diary which is a well-documented, retrospective, self report measure and is completed upon rising (Espie, 1991). This provides a summary record of parameters from the preceding night's sleep.
2. An objective measure of sleep was obtained using wrist actigraphic recording on the 'Actiwatch' (Model AW2® Cambridge Neurotechnology Ltd). The period of time the actigraph accumulates activity counts, before saving the sample and resetting the counter to zero, is referred to as the epoch length. For this study the epoch length chosen was 1 min (for sleep analysis, an interval of 1 min or less is recommended). A recording epoch is scored as 'sleep' or 'wakefulness' according to criteria determined by the program's algorithm. The actigraph also had an event marker button which, when depressed, marked the date and time.
3. The cognitive sub-scale of the Pre-Sleep Arousal Scale; a self-administered, eight item questionnaire, was completed each morning (Nicassio et al., 1985). This provided a description of the subject's level of cognitive arousal as they fell asleep. Subjects rated on a five point scale ('not at all'–'extremely') how intensely they experienced each of the symptoms as they attempted to fall asleep.
4. Subjects were provided with a voice-activated audiocassette recorder (Sony Cassette Recorder TCS-580 V) to place at their bedside on each night of investigation. This provided a 'live' measure of the content and nature of intrusive thoughts experienced as the subject attempted to fall asleep.

Table 1
Summary descriptive information on sample ($n = 21$)

		%		
Sex	Male ($n = 7$)	33.3		
	Female ($n = 14$)	66.7		
Marital status	Married ($n = 8$)	38.1		
	Single ($n = 10$)	47.6		
	Divorced ($n = 3$)	14.3		
Bed partner		47.7		
Occupational situation	Working	85.7		
	Student	14.3		
Age (years)		Mean	Range	S.D.
		36.0	19–60	10.8
Duration of sleep problem (years)		10	1–40	8.2

2.3. Procedure

The procedure for the direct monitoring of intrusive thoughts was developed after piloting the use of the voice-activated recorder on a small group of volunteers. Once set to record, subjects were instructed to leave the recorder at their bedside while they attempted to fall asleep. When having difficulty in falling asleep, they were instructed to say aloud whatever was going through their mind. No directive guidelines were given on what should be recorded in order to minimise performance anxiety and to allow subjects to express themselves freely. Subjects were also supplied with a wrist actigraph and instructed to wear it continuously for three nights. If the actigraph had to be removed, for example in wet activities, then a record was kept by depressing the actigraph's event marker button, which was also used to mark 'lights out' and 'rising time'. The Sleep Diary and Pre-Sleep Arousal Scale were completed upon rising after each night of investigation. Subjects were instructed to go to bed as normal, following their usual routine. The actigraph was removed upon completion of three nights' wear and returned to the investigators along with the completed forms and audiotape.

3. Results

3.1. Content analysis of pre-sleep cognitions

Data were available for three nights for each of the 21 subjects, i.e. 63 subject nights. The following procedure was adopted for the content analysis.

1. Tape-recorded material was transcribed.
2. Transcripts were segmented into a series of single ideas or statements which could stand alone.
3. The segmented transcripts were independently studied by the authors to generate descriptive themes.
4. Themes were then discussed and those sharing common content were grouped into categories, and then into sub-categories. The model of cognitive, somatic and environmental arousal proposed by Espie (1991) was useful here.
5. Detailed criteria for the coding of segments to categories were agreed (available from the corresponding author).
6. Segments were then categorised and an independent reliability check was conducted on a proportion of the transcripts.

This procedure generated over 1,000 thought segments. Derived thought categories are presented in Table 2 and brief definitions are provided. Reliability of coding to category was found to be satisfactory with 93% agreement between independent raters using a sample of four subjects, i.e. 12 transcripts. Agreement did not vary substantially across categories.

Since affect may also be an important factor in the pre-sleep experience, a numerical global rating of emotion was assigned to each subject night according to the dominant emotion

Table 2
Categories of thoughts derived from content analysis of audiotape-recorded material with examples for each coding category

Thought category	Sample content	*n* of thoughts	% of total
Rehearsing/planning, problem-solving	Thinking about the past day, past experiences, next day, things to do, planning things, forthcoming events, work-related and social issues, friends and family	465	43
Sleep and its consequences	Thinking about need/desire to sleep, efforts/time/expectancy to fall asleep, ease/difficulty of falling asleep quickly, importance of sleep, consequences of not sleeping, having a sleep problem, past experience of sleep	217	20
Reflection on quality of thoughts	'Thinking about thinking', mind buzzing, thoughts rushing, darting thoughts, visual imagery, random/dream-like thoughts, reference to own type of thinking, uncontrollability, unpleasantness	129	12
Arousal status	Thinking about feeling exhausted, experiencing sleepiness, preoccupation with physical tiredness	93	9
External noise	Thinking about the wind, wood creaking, traffic, clock, telephone ringing	69	6
Autonomic experiences	Thinking about heart rate, headache, tension, body movement, feeling cold, hot feet, breathless, itching, restlessness	67	6
Procedural factors	Thinking about procedure of the research itself, need to press actigraph button, thinking about what to say aloud	35	3
Rising from bed	Thinking about getting up, putting the light on	15	1

expressed on the tape, taking into account the tone and content of speech. A 10 point Likert scale ranging from −5 (extremely unpleasant) through 0 (neutral) to +5 (extremely pleasant) was used. Since this was an assessment of perceived emotion, reliability was again investigated using a sample of four subjects, i.e. 12 tape recorded nights. Agreement was deemed acceptable if numerical ratings, independently assigned, were either the same or within one integer of the same value. Concordance between raters for affective state was found to be reasonable at 80%.

Table 2 presents a profile of the nature and frequency of occurrence of each category of thought. As can be seen, by far the most commonly reported thoughts concerned rehearsing, planning and problem-solving (43% of total). Thoughts relating to the sleep process and its consequences were also quite common (20%), followed by non-specific 'thinking about thinking', and thoughts relating to arousal status (12% and 9% respectively). The remaining four categories together comprised the remaining 16%. Friedman two-way analyses of variance for related samples were used to test for systematic variations across the three nights. These showed that there was no significant difference between nights, and in particular, no 'first night effect'. Therefore, data from each night could be included in subsequent analyses.

3.2. Sleep pattern

Actigraphic and Sleep Diary data were obtained from 21 subjects for a total of 63 nights, from which the following measures were calculated: sleep onset latency (time taken to fall

Table 3
Means, ranges and standard deviations for actigraphic and sleep diary data with results of two-tailed *t*-tests for paired samples and product-moment correlations (63 subject nights)

	Actiwatch Data				Sleep Diary Data					
	Minimum	Maximum	Mean	S.D.	Minimum	Maximum	Mean	S.D.	*t*-test	Pearson
Sleep-onset latency (min)	0	157	29.6	34.49	0	240	57.69	48.55	$t = 4.801$ $p < 0.001$	$r = 0.419$ $p < 0.001$
Sleep efficiency (%)	55.1	95.4	76.59	9.38	38.3	96	71.82	12.25	$t = 2.719$ $p < 0.008$	$r = 0.194$ $p < 0.128$
Total sleep time (min)	278	539	383.17	55.42	180	540	359.71	69.19	$t = 3.010$ $p < 0.004$	$r = 0.526$ $p < 0.001$

asleep); total sleep time; and sleep efficiency (the percentage of time spent asleep relative to time in bed). Table 3 presents means and standard deviations for actigraphic and sleep diary data, and the results of two-tailed *t*-tests for paired samples. Pearson correlation coefficients were computed to evaluate the relationships between variables. Table 3 confirms that the subjects had significant sleep problems. Objectively measured sleep efficiency was 77%, well below the criterion cut-off point of 85% which defines clinical problems. Subjects typically slept around 6.5 h per night. Comparison of actigraphic and subjective measures demonstrated that, although moderately correlated (r = 0.42–0.53 for principal measures), mean scores differed significantly. Subjects overestimated sleep latency by around 28 min and underestimated time slept by a similar margin.

3.3. Relationship between sleep and cognitions

Correlational data on the relationships between derived thought categories and objective and subjective measures of sleep disturbance are presented in Table 4. It is noteworthy that the patterns of association were generally different for actigraphic and diary measures of sleep. Actigraphic sleep latency correlated modestly but significantly with three derived thought categories, including the two most frequently reported i.e. rehearsal/planning (r = 0.33) and sleep and its consequences (r = 0.37), along with autonomic functions (r = 0.29). By comparison subjective sleep latency did not correlate with any specific thought category. However, subjective sleep efficiency correlated inversely with sleep and its consequences ($r = -0.28$) and general arousal ($r = -0.37$). Both measures of sleep-onset correlated with the total number of thought segments, although for the sleep diary measure this correlation was

Table 4
Correlations between actigraphic and sleep diary measures of sleep disturbance and tape-recorded cognitions. $^{*}p < 0.05$ $^{**}p < 0.01$ $^{***}p < 0.001$

	SOL actigraph[a]	Efficiency actigraph[b]	SOL diary[a]	Efficiency diary[b]
Rehearsing/planning, problem-solving	0.326**	−0.183	0.239	0.043
Sleep and its consequences	0.372**	0.002	0.186	−0.278*
Reflection on quality of thoughts	0.034	0.220	0.194	0.089
Arousal status	0.040	−0.088	0.153	−0.370**
External noise	0.140	−0.081	0.010	−0.094
Autonomic experiences	0.289*	−0.043	0.156	−0.152
Procedural factors	−0.010	0.005	−0.029	0.115
Rising from bed	0.050	0.029	0.040	−0.045
All thoughts	0.334**	−0.095	0.261*	−0.163
PSAS cognitive subscale	0.173	−0.049	0.424***	−0.495***
Rating of emotion	−0.293*	0.019	−0.193	−0.033
F I 'active problem-solving'	0.319**	−0.160	0.222	−0.044
F II 'present state monitoring'	0.232	0.074	0.244	−0.317**
F III 'environmental concerns'	0.136	−0.066	0.079	−0.060

[a] SOL: sleep onset latency; [b]sleep efficiency: % of time in bed spent asleep.

Table 5
Results of Principal Components Analysis comprising derived factors, and factor loadings (significant values in bold)

Extraction	Eigenvalue	% of variance	Cumulative %	%Explained variance
Factor I	2.636	32.95	32.95	52.22
Factor II	1.386	17.33	50.28	27.47
Factor III	1.025	12.81	63.09	20.31
Rotated Factor Matrix		Factor I 'active problem solving'	Factor II 'present state monitoring'	Factor III 'environmental reactivity'
Rehearsing/planning, problem solving		**0.760**	0.340	0.106
Sleep and its consequences		0.111	**0.743**	0.134
Quality of thoughts		0.462	**0.545**	0.270
Arousal status		**0.608**	0.390	−0.039
External noise		0.036	0.224	**0.785**
Autonomic experiences		0.044	**0.676**	−0.128
Procedural factors		**0.821**	−0.223	0.141
Rising from bed		0.147	−0.179	**0.845**

relatively weak. Thoughts relating to external noise, procedural factors and rising from bed were unrelated to any sleep measure, perhaps due to their infrequency.

The cognitive subscale of the PSAS was not found to be associated with objectively measured sleep, whereas highly significant correlations were observed with diary measures of both sleep latency and sleep efficiency ($p < 0.001$). In contrast, unpleasant emotional state was found to correlate with objective but not subjective sleep latency.

3.4. Interrelationships between thought categories (Principal Components Analysis)

In order to develop an explanatory model, a principal components analysis was performed on the eight derived categories of thought intrusions. Varimax rotation yielded a three-factor solution, each with eigenvalue greater than 1 and explaining more than 10% of variance (Table 5). Factor I comprising problem-solving thoughts, thoughts about feeling tired and about the experimental procedure itself, explained 33% of variance and was labelled 'active problem-solving'. Factor II explained a further 17% of variance and was labelled 'present state monitoring' since it comprised reflective thoughts relating to sleep, bodily functions and general introspection. Factor III explained 13% of variance and was labelled 'environmental reactivity' since it comprised responses to or awareness of external stimulation disrupting sleep. The total explained variance of this solution, therefore, was 63%. Inspection of factor loadings in Table 5 suggests a relatively 'pure' factor structure.

Factor scores were then calculated and correlated with sleep measures. The results of this analysis are presented in the lower half of Table 4. As can be seen, 'active problem solving' was positively correlated with actigraphic sleep latency ($r = 0.32$). 'Present state monitoring' just failed to reach significant correlation with objective ($p = 0.06$) and subjective sleep latency

($p = 0.055$), but was inversely correlated with subjective sleep efficiency ($r = -0.32$). Factor III 'environmental reactivity' was unrelated to any sleep parameter.

3.5. Validation of derived factors

The cognitive sub-scale of the PSAS, although limited by its retrospective and global nature, has been widely used in previous research. Similarly, affect-laden cognitions have been reported to be more strongly associated with sleep disruption than non affect-laden thoughts (see introduction). Therefore, the PSAS sub-scale and the independent rating of emotion used in this study, were correlated with the derived factors for the purpose of preliminary validation. Correlations with the PSAS were for Factor I ($r = 0.209$; $p = 0.099$), Factor II ($r = 0.427$; $p < 0.001$) and Factor III ($r = 0.223$; $p = 0.078$) suggesting that 'present state monitoring' is more similar to the measurement properties of the PSAS than is 'active problem-solving' or 'environmental reactivity'. Correlation with emotional state was strong both for Factor II ($r = -0.420$; $p = 0.001$) and Factor III ($r = -0.419$; $p = 0.001$) suggesting that emotionality is more negative with higher levels of 'present state monitoring' and greater 'environmental reactivity'. The non-significant correlation with Factor I 'active problem-solving' ($r = -0.189$; $p = 0.138$) implies that this form of mental activity is less affect laden. It should be noted that PSAS scores and emotionality ratings were somewhat independent of one another being modestly inversely correlated ($r = -0.301$; $p = 0.016$).

3.6. Regression analyses

To further consider potential explanatory models, all measures of pre-sleep cognitive activity which correlated significantly with a sleep measure (Table 4; $p < 0.05$) were entered into a stepwise linear regression on that particular sleep variable. The results af these analyses are presented in Table 6.

An equation combining 'rehearsing and planning' thoughts with those concerning 'sleep and its consequences' explained around 16% of variance (adjusted $R^2 = 0.161$) in actigraphic sleep

Table 6
Stepwise linear regression of explanatory pre-sleep cognitive variables upon actigraphically and subjectively reported sleep disturbance

Outcome variable	F ratio	p	R^2	Adj. R^2	Explanatory variables (beta; prob)
Actigraphic data					
Sleep-onset latency	6.97	0.0019	0.188	0.161	'sleep and its consequences' (0.300; $p = 0.017$) 'rehearsing/planning problem solving' (0.235; $p = 0.048$)
Sleep efficiency	–	–	–	–	no predictors
Diary data					
Sleep-onset latency	13.36	0.0005	0.180	0.166	PSAS cognitive sub-scale (0.424; $p = < 0.001$)
Sleep efficiency	19.77	0.0001	0.244	0.232	PSAS cognitive sub-scale (−0.495; $p < 0.001$)

latency. By comparison, subjective report of sleep-onset was predicted only by scores on the cognitive sub-scale of the PSAS (Adj. $R^2 = 0.17$). Actigraphic sleep efficiency had no significant predictors, and for subjective sleep efficiency, only the cognitive sub-scale of the PSAS entered significantly (Adj. $R^2 = 0.23$).

4. Discussion

This study investigated systematically the relationship between independently gathered, objective data on mental activity and sleep pattern. There has been increasing emphasis in the insomnia literature both upon cognitive models of aetiology and maintenance, and upon cognitive mediation of treatment effects. Therefore, the results of the study are of particular relevance at this time.

The use of voice-activated audiotape recorders made it possible to gather information 'live' while subjects lay awake in bed during the pre-sleep period. Informal reports indicated that they found this procedure, and wearing the wrist actigraph, relatively non-intrusive. This is supported by content analysis of the tape-recordings where only 15 out of 1090 thought segments (3%) related to the research process itself. Also, there was no evidence of the 'first night effect', common in EEG studies of sleep, either in terms of volume or content of thought intrusion. Significant differences were observed between actigraphic and subjective assessment of sleep pattern. Indeed, diary reports overestimated objective sleep-onset latency by 100%, while still achieving modest correlation ($r = 0.42$). Such findings are entirely consistent with the literature, and underline the potential importance of considering subjective and objective data as separate factors in explanatory models. We would argue, therefore, that the data gathered in this study are both valid and reliable for their purposes of assessing cognitions (volume and content) and sleep (objective and subjective).

Categorisation of transcribed audiotape material achieved highly satisfactory levels of concordance between independent judges. Thus, the derived content analysis appears to be reasonably robust. 'Rehearsing, planning and problem-solving' thoughts formed the largest component of pre-sleep cognitive activity. Approaching half of all mental activity was of this type, comprising reflection on the day past, preparation for the next day and consideration of personal and work-related matters. It cannot be assumed, of course, that these thoughts were necessarily worrisome to the subjects, or that they caused delayed sleep. Certainly, a modest but significant correlation was observed with actigraphic sleep latency ($r = 0.33$) suggesting, at least, that the longer subjects were awake the more rehearsal and planning occurred. The Principal Components Analysis (PCA) loaded thoughts about physical tiredness and about the experimental procedure itself, along with these problem-solving thoughts into Factor I. This factor also correlated with objective sleep latency ($r = 0.32$). Interestingly, however, Factor I was the only factor which did not correlate significantly with the rating of emotional tone implying that 'active problem-solving' is a less affect-laden form of cognition. In relation to the model proposed by Espie and Wicklow (1999), it may correspond to the pre-sleep cognitive 'load' or 'business' of the mind where mental arousal primarily reflects activity rather than concern.

Thinking about 'sleep and its consequences' represented the next most frequent form of

thinking, comprising around 20% of all thought segments. Typically, subjects reported preoccupation with being unable to fall asleep and the anticipated impact which poor sleep might have on daytime performance and mood. This category again correlated with objective sleep latency ($r = 0.37$). PCA factored 'sleep and its consequences' along with 'reflection on quality of thoughts' and 'autonomic experiences' into Factor II 'present state monitoring'. This label was chosen in consideration of common factor variance which appeared to be concerned with self-awareness and self-monitoring. Although Factor II did not correlate significantly with actigraphic sleep latency, an inverse correlation was found with emotional tone, i.e. the greater the state monitoring the more unpleasant the perceived emotion. Insomniacs have long been regarded as introspective and prone to worry, and this factor seems most closely associated with this disposition, as well as the with the concept of affect-laden thinking. It is also noteworthy that concern about sleep and its consequences has previously been proposed as central to the dysfunctional beliefs and attitudes which insomniacs have about their sleep, and as an important target for change using cognitive therapy techniques (Espie, Inglis, Harvey & Tessier, 1999; Morin, 1993; Morin, Stone, Trinkle, Mercer & Remsberg, 1993). 'Autonomic experiences' was the only other type of thinking from the content analysis to be specifically correlated with objective sleep latency, and as mentioned above, this category appears associated with more general internalised focussing (Factor II).

It is particularly striking that none of the derived categories or factors of intrusive thinking correlated significantly with subjective perception of sleep latency. The Sleep Diary is the standard assessment tool in clinical practice with insomniacs and yet, in this study, it was only objective measurement which demonstrated significant association with discrete cognitions. Self-report was, however, quite strongly associated with scores on the cognitive sub-scale of the PSAS ($r = 0.42$), whereas actigraphic measurement was not ($r = 0.17$). This divergence implies that self-report and objective indices may reflect differing facets of sleep phenomenology. Inevitably, the PSAS is a global index and it might be expected to correlate better with another summary, retrospective measure such as subjective sleep efficiency. The PSAS is probably most useful in *differentiating*, i.e. high versus low arousal, insomniacs versus non-insomniacs or cognitive versus physiological hyperarousal. Although an association was found between Factor II and the PSAS ($r = 0.43$), only the thought categories of 'sleep and its consequences' and 'rehearsing, planning and problem-solving' contributed significantly to the prediction of objective sleep latency. The PSAS was the only measure to predict subjective latency and subjective sleep efficiency. There are two clear implications arising from these findings. First, diary assessment of sleep may not be sufficiently accurate for investigation of explanatory models of objective sleep pattern; and second, where cognitive models are under consideration, particular care needs to be taken to avoid 'globalising' the cognitive phenomenon itself.

Numerous reviews and meta analyses have confirmed that cognitive–behavioral treatment for insomnia is effective in treating sleep-onset insomnia (Edinger and Wohlgemuth, 1999; Morin, Culbert & Schwartz, 1994; Morin et al., 1999; Murtagh and Greenwood, 1995). However, it now seems important to understand their influence on cognitive processes per se. Presumably, if falling asleep more quickly, a treated insomniac will have fewer pre-sleep thoughts. However, the effects of specific treatments upon specific types of thoughts may tell us something both about the nature of insomnia itself and the mechanism of action of psychological treatment. Similarly, a comparative study with normal sleepers would be

valuable. The present study may provide a useful methodology for further research. It is suggested (a) that actigraphic monitoring of sleep merits further application in this field; (b) that methods of 'live' recording of intrusive thoughts should be developed further. In particular, more consideration might be given to the type and degree of emotional expression and how that can be rated from audiotape material; and (c) that the categories derived from content analysis and the factors derived from PCA in this study might be helpfully incorporated into a rating scale format for further validation.

The advantages of tailoring treatment for insomnia, although intuitively appealing, are less persuasive from empirical evidence (Edinger and Wohlgemuth, 1999; Espie et al., 1989; Sanavio, 1988). This reflects a mismatch somewhere within the matrix of *presumed* and *actual* causes/maintaining factors of insomnia, and *presumed* and *actual* mechanisms of action of interventions for insomnia. Additionally, however, is the suggestion from this study that the sources of data for the appraisal of outcome may be crucial, i.e. *objective/specific* versus *subjective/global*. Clarification of this overall matrix is the ultimate research goal which will deliver the most efficient and effective treatment.

References

American, Sleep Disorders Association (1995). Practice parameters for the use of actigraphy in the clinical assessment of sleep disorders. *Sleep*, *18*(4), 285–287.

American, Sleep Disorders Association (1997). *The International Classification of Sleep Disorders; Diagnostic and Coding Manual (Revised)*. Rochester, USA: ASDA.

Borkovec, T., Grayson, J., O'Brien, G., & Weerts, T. (1979). Relaxation treatment of pseudoinsomnia and idiopathic insomnia; an electroencephalographic evaluation. *Journal of Applied Behavior Analysis*, *12*, 37–54.

Borkovec, T. D., Lane, T. W., & VanOot, P. H. (1981). Phenomenology of sleep among insomniacs and good sleepers: Wakefulness experience when cortically asleep. *Journal of Abnormal Psychology*, *6*, 607–609.

Broman, J. E., & Hetta, J. (1994). Perceived pre-sleep arousal in patients with persistent psychophysiologic and psychiatric insomnia. *Nordic Journal of Psychiatry*, *48*(3), 203–207.

Buysse, D. J., Reynolds, C. F., Monk, T. H., Berman, S. R., & Kupfer, D. J. (1989). The Pittsburgh Sleep Quality Index: A new instrument for psychiatric practice and research. *Psychiatry Research*, *28*, 193–213.

Coyle, K., & Watts, F. N. (1991). The factorial structure of sleep dissatisfaction. *Behaviour Research and Therapy*, *29*, 513–520.

Edinger, J. D., & Wohlgemuth, W. K. (1999). The significance and management of persistent primary insomnia: The past, present and future of behavioural insomnia therapies. *Sleep Medicine Reviews*, *3*, 101–118.

Espie, C. A., Brooks, D. N., & Lindsay, W. R. (1989). An evaluation of tailored psychological treatment of insomnia. *Journal of Behaviour Therapy and Experimental Psychiatry*, *20*, 143–153.

Espie, C. A. (1991). *The psychological treatment of insomnia*. Chichester: Wiley.

Espie, C. A., & Wicklow, A. (1999). Cognitive therapy for insomnia. In C. Shapiro, & E. P. Sloan, *Sleep in Psiachiatry*. Cambridge University Press.

Espie C.A., Inglis S.J., Harvey L., Tessier S. (1999). Insomniacs' attributions: Psychometric properties of the Dysfunctional Beliefs and Attitudes about Sleep Scale and the Sleep Disturbance Questionnaire (Submitted).

Evans, F. J. (1977). Subjective characteristics of sleep efficiency. *Journal of Abnormal Psychology*, *86*, 561–564.

Fichten, C. S., Libman, E., Creti, L., Amsel, R., Tagalakis, V., & Brender, W. (1998). Thoughts during awake times in older good and poor sleepers — The Self-Statement Test: 60+. *Cognitive Therapy and Research*, *22*, 1–20.

Foley, D. J., Monjan, A. A., Brown, S. L., Simonsick, E. M., Wallace, R. B., & Blazer, D. G. (1995). Sleep complaints among elderly persons: an epidemiologic study of three communities. *Sleep*, *18*, 425–433.

Freedman, R. R., & Sattler, H. L. (1982). Physiological and psychological factors in sleep-onset insomnia. *Journal of Abnormal Psychology*, *91*, 380–389.

Gallup, Organization (1991). *Sleep in America*. Princeton, NJ: Gallup.

Gendron L., Blais F.C., Morin C.M. (1998). Cognitive activity among insomniac patients. *Sleep, 21*, 130.

Gross, R. T., & Borkovec, T. D. (1982). Effects of a Cognitive Intrusion Manipulation on the Sleep-Onset Latency of Good Sleepers. *Behaviour Therapy*, *13*, 117–124.

Hall, M., Buysse, D. J., Reynolds, C. F., Kupfer, D. J., & Baum, A. (1996). Stress related intrusive thoughts disrupt sleep onset and continuity. *Sleep Research*, *25*, 163.

Haynes, S. N., Adams, A., & Franzen, M. (1981). The effects of presleep stress on sleep-onset insomnia. *Journal of Abnormal Psychology*, *90*, 601–606.

Kripke, D. F., Mullaney, D. J., Messin, S., & Wyborney, V. G. (1978). Wrist actigraphic measures of sleep and rhythms. *Journal of Clinical Neurophysiology*, *44*, 674–676.

Kuisk, K. A., Bertelson, A. D., & Walsh, J. K. (1989). Presleep cognitive hyperarousal and affect as factors in objective and subjective insomnia. *Perceptual and Motor Skills*, *69*, 1219–1225.

Lichstein, K. L., & Rosenthal, T. L. (1980). Insomniacs' perceptions of cognitive versus somatic determinants of sleep disturbance. *Journal of Abnormal Psychology*, *89*, 105–107.

Morin, C. M. (1993). In *Insomnia: Psychological assessment and management* (pp. 195–197). New York: Guildford Press.

Morin, C. M., Stone, J., Trinkle, D., Mercer, J., & Remsberg, S. (1993). Dysfunctional beliefs and attitudes about sleep among older adults with and without insomnia complaints. *Psychology and Ageing*, *8*, 463–467.

Morin, C. M., Culbert, J. P., & Schwartz, M. S. (1994). Non-pharmacological interventions for insomnia: a meta-analysis of treatment efficacy. *American Journal of Psychiatry*, *151*, 1172–1180.

Morin C.M., Hauri P.J., Espie C.A., Spielman A., Buysse D., Bootzin R.R. 1999 Nonpharmacologic treatment of insomnia. Report prepared by a taskforce of the American Sleep Disorders Association, Standards Of Practice Committee, Sleep (in press).

Mullaney, D. J., Kripke, D. F., & Messin, S. (1980). Wrist actigraphic estimation of sleep time. *Sleep*, *3*, 83–92.

Murtagh, D. R., & Greenwood, K. M. (1995). Identifying effective psychological treatments for insomnia: a meta-analysis. *Journal of Consulting and Clinical Psychology*, *63*, 79–89.

Nicassio, P. M., Mendlowitz, D. R., Fussell, J. J., & Petras, L. (1985). The phenomenology of the pre-sleep state: the development of the pre-sleep arousal scale. *Behaviour Research and Therapy*, *23*(3), 263–271.

Oyahon, M. M., Caulet, M., & Guilleminault, C. (1997). How a general population perceives its sleep and how this relates to the complaint of insomnia. *Sleep*, *20*, 715–723.

Sadeh, A., Hauri, P. J., Kripke, D. F., & Lavie, P. (1995). The role of actigraphy in the evaluation of sleep disorders. *Sleep*, *18*, 288–302.

Sanavio, E. (1988). Pre-sleep cognitive intrusions and treatment of onset-insomnia. *Behaviour Research and Therapy*, *26*, 451–459.

Van Egeren, L., Haynes, S. N., Franzen, M., & Hamilton, J. (1983). Presleep cognitions and attributions in sleep-onset insomnia. *Journal of Behavioural Medicine*, *6*, 217–232.

Watts, F. N., Coyle, K., & East, M. P. (1994). The contribution of worry to insomnia. *British Journal of Clinical Psychology*, *33*, 211–220.

Watts, F. N., East, M. P., & Coyle, K. (1995). Insomniacs' perceived lack of control over sleep. *Psychology and Health*, *10*, 81–95.

Annu. Rev. Psychol. 2002. 53:215–43

INSOMNIA: Conceptual Issues in the Development, Persistence, and Treatment of Sleep Disorder in Adults

Colin A. Espie
Department of Psychological Medicine, University of Glasgow, Academic Centre, Gartnavel Royal Hospital, 1055 Great Western Road, Glasgow, G12 0XH, Scotland; e-mail: c.espie@clinmed.gla.ac.uk

Key Words cognitive-behavior therapy, circadian rhythm, sleep homeostasis

■ **Abstract** This paper critically reviews the evidence base for previously reported conceptual models of the development and persistence of insomnia. Although a number of perspectives have some empirical support, no one approach emerges as preeminent. Importantly, the efficacy of any particular psychological intervention cannot be taken as confirmation of presumed, underlying mechanisms. An integrated psychobiological inhibition model of insomnia is developed that accounts for the research data. The model views insomnia as arising from inhibition of de-arousal processes associated with normal sleep. It is proposed that sleep homeostatic and circadian factors are compromised by impairment of the automaticity and plasticity associated with good sleep, and that cognitive/affective processes activate the clinical complaint of insomnia. Common pathways for the action of cognitive-behavioral interventions are identified, and a research agenda is set for further conceptual and clinical study.

CONTENTS

0084-6570/02/0201-0215$14.00

AN INTRODUCTION TO INSOMNIA

The *Diagnostic and Statistical Manual of Mental Disorders* (DSM-IV) defines primary insomnia as a complaint lasting for at least 1 month of difficulty initiating and/or maintaining sleep or of nonrestorative sleep (Am. Psychiatr. Assoc. 1994). The International Classification of Sleep Disorders-Revised (ICSD-R) uses the term "psychophysiologic insomnia" for such a complaint and associated decreased functioning during wakefulness. (Am. Sleep Disorders Assoc. 1997). ICSD-R regards insomnia of 6-month duration as chronic. Both systems differentiate insomnia from circadian rhythm disorders, in which timing of the major sleep period is out of alignment with the local clock; from parasomnias, in which behavioral events occur in association with sleep (e.g., sleepwalking, night terrors); and from secondary insomnias, in which psychiatric, neurologic, or medical problems present. Disorders such as sleep apnea, with associated respiratory impairment, and disorders of excessive sleepiness (e.g., narcolepsy) are also classified separately.

Conservative estimates for chronic insomnia range from 9–12% in adulthood and up to 20% in later life, and women present about two times more than men (Bixler et al. 1979, Mellinger et al. 1985, Ford & Kamerow 1989, Gallup Organisation 1991, Foley et al. 1995, Hoch et al. 1997). Sleep disturbance is a common complaint in general practice (Shocat et al. 1999) and once established may persist over many years (Mendelson 1995). Insomnia therefore constitutes a considerable public health problem. The direct costs of assessing and treating insomnia were approximated as $14 billion in the United States and FF10 billion in France in 1995 (Walsh & Engelhardt 1999, Leger et al. 1999).

Polysomnographic assessment (PSG) comprises monitoring of the electroencephalogram (EEG) along with muscle activity, eye movement, respiration, and blood oxygen saturation levels. However, PSG is not required unless clinical presentation raises the possibility of disorders such as sleep apnea (Gillin & Byerley 1990, Douglas et al. 1992, Reite et al. 1995, Am. Sleep Disorders Assoc. 1995a). Actigraphic assessment is helpful to identify disorders of circadian function. The wrist actigraph provides data on body movement over extended periods (typically 1 minute epochs for several weeks) and is a reliable index of sleep parameters (Sadeh et al. 1995, Am. Sleep Disorders Assoc. 1995b).

In practice, structured interview is recommended (Buysse et al. 1989, Morin 1993, Spielman & Anderson 1999, Espie 2000), supplemented by a sleep diary completed upon waking, comprising information on sleep-onset latency (SOL),

wake time after sleep-onset, and total sleep time (Espie 1991). Time in bed (TIB) is calculated by subtracting rising time from bedtime, and the sleep efficiency index is computed as the ratio of total sleep time to TIB expressed as a percentage. A sleep efficiency index of 85% is the upper limit for poor sleep (Frankel et al. 1976, Coates et al. 1982), and a minimum SOL or wake time after sleep-onset of 30 minutes per night is a threshold for clinical significance (Espie et al. 1989a, 2001a). Baseline values in outcome studies, however, are typically twice this level (Morin et al. 1999b). With medication, alcohol, and ratings of sleep quality commonly included, the sleep diary quantifies sleep experience and permits study of night-to-night variability, which is important because unpredictability of sleep is a feature of insomnia (Coates et al. 1982, Roth et al. 1976).

The American Academy of Sleep Medicine (AASM) has published practice parameters for the assessment (Chesson et al. 2000) and nonpharmacological treatment of insomnia (Chesson et al. 1999). The latter were derived from systematic review (Morin et al. 1999b) following meta-analyses demonstrating the efficacy of cognitive-behavioral treatment (CBT) for adults (Morin et al. 1994, Murtagh & Greenwood 1995). Another review came to similar conclusions (Edinger & Wohlgemuth 1999). Furthermore, clinical effectiveness studies have produced comparable results (Espie et al. 2001a,b), and CBT with or without pharmacotherapy compares favorably with pharmacotherapy alone for older adults (Morin et al. 1999a). CBT, therefore, may be the treatment of first choice for chronic insomnia (Espie 1999). The role of hypnotic medication has been debated (Kripke 2000, Kramer 2000, Buysse 2000), there being little evidence of long-term efficacy for any drug. Recent textbooks also provide useful discussion: Lichstein & Morin (2000) focus on late-life insomnia, and Pressman & Orr (1997) provide an overview of insomnia in medical patients. Finally, an AASM working group on research diagnostic criteria will report in 2002 (Edinger et al. 2002). Thus, there is a sizeable, systematic, and practitioner-oriented literature on insomnia. The conceptual foundations of insomnia, however, have been more neglected, and it seems timely to review models of the development, persistence, and treatment of insomnia, to evaluate explanatory mechanisms, and to propose an evidence-based integrated model.

THE CONCEPTUAL BASIS FOR THE DEVELOPMENT AND MAINTENANCE OF INSOMNIA

A wide range of factors may play some part in insomnia. It may be helpful to consider each of these before presenting a proposed integrated model.

Normal Sleep in Human Development

Wakefulness is not pathological. On the contrary, it might be considered the preferred state because a primary function of sleep is to ensure wakeful cortical

function (Horne 1988). Prolonged wakefulness reliably induces sleep, and failure to obtain at least a core amount (sleep deprivation) leads to impaired function. Two processes interact in normal sleep (Borbely 1994). The sleep homeostat "drives" the sleep-wake schedule toward a balanced requirement because prolonged wakefulness accrues "sleep debt" (Carskadon & Dement 1981) and sleep pays off the debt; the circadian timer regulates the biological clock in approximation to the 24-hour clock (Borbely 1994, Moore-Ede et al. 1982). These processes also regulate type of sleep (Dement 1960). Thus, young children have longer sleep periods than adults and have higher proportions of rapid eye movement (REM) and "deep sleep" (non-REM stages 3 and 4). Similarly, insufficient sleep induces recovery sleep comprising proportionately more REM and deep sleep. The sleep of older adults is more fragmented and lighter (Bliwise 1993, Carskadon et al. 1982), and homeostatic drive declines with age (Buysse et al. 1993), when disturbances of the sleep-wake schedule are less well tolerated (Webb 1981, Monk et al. 1992). Thus, increasing age represents a vulnerability factor to sleep disturbance.

Quality of Sleep

Investigations of PSG and self-report generally reveal modest positive correlation. Poor sleepers overestimate sleep disturbance relative to objective criteria (see Espie 1991, pp. 17–18). However, insomniacs have reported being awake when roused from light sleep (Borkovec et al. 1981), and modified EEG criteria are associated with greater accuracy (Coates et al. 1982). There is debate over whether insomniacs are sleepy in the daytime (Seidel et al. 1984, Mendelson et al. 1984, Chambers & Keller 1993, Lichstein et al. 1994). However, sleep should not be considered only in terms of chronobiological "fitness for purpose." The quality of the sleep experience is important. Indeed, ratings of sleep quality do not necessarily correlate highly even with subjective reports of sleep pattern. Unpublished analyses from our recent clinical cohort (Espie et al. 2001b; $n = 139$) reveal modest inverse association of SOL and wake time after sleep-onset with ratings of "sleep enjoyment" ($r = -0.30$ and -0.31) and "restedness after sleep" (-0.22 and -0.19).

Predisposing, Precipitating, and Perpetuating Factors

How then might insomnia develop? A useful conceptualization comprises predisposing, precipitating, and perpetuating components (Spielman & Glovinsky 1991). Both DSM-IV and ICSD-R report familial association with light, disrupted sleep, and ICSD-R reports anxious over-concern with health as predisposing. Indeed, insomniacs appear prone to introspection and worry (e.g., Kales et al. 1984, Edinger et al. 1988, Lundh et al. 1995, Schramm et al. 1995). Research suggesting elevated autonomic and metabolic rates also implies a vulnerability factor (Bonnet & Arand 1995, 1997a). However, predisposing factors alone are unlikely to create imbalance in sleep homeostasis or circadian timing, although they might impair sleep quality and, potentially, lead to sleep state misperception (see also Bonnet &

Arand 1997b). Reduced "plasticity" (see below) might also be a predispositional factor.

Transient sleep disorder is a likely context in which to identify precipitating factors. ICSD-R defines adjustment sleep disorder associated temporally with acute stress, conflict, or environmental change, and shiftwork schedule disorder as a transient phenomenon relating to work schedules. Because the homeostat and timer regulate natural variation, it can be hypothesized that there is generally sufficient plasticity to absorb the impact of such events, and to survive more prolonged change. The severity and impact of events may need to be greater to precipitate sleep disturbance in the absence of predisposing factors. Nevertheless, studies investigating the onset of chronic insomnia have commonly found that stress or life change was a factor at the time (Healey et al. 1981, Kales & Vgontzas 1992, Morgan & Clarke 1997). Presumably, some other mechanism accounts for the persistence of a sleep problem. Why only some disturbances develop into chronic insomnia when others spontaneously remit, and why good sleep can persist during chronic stress are important questions requiring further study.

To summarize, predisposition may interact with precipitating factors to create temporary sleep disruption, but in the absence of perpetuating factors, the plasticity of the sleep-wake schedule would drive toward homeostasis and reestablish good sleep. The literature has focused primarily on presumed factors maintaining insomnia.

Mental Disorder

A common misconception is that insomnia is a symptom not meriting treatment in its own right. However, insomnia can be either a symptom (e.g., a complaint of difficulty falling asleep) or a disorder (i.e., complaint plus significant distress and functional impairment) (Harvey 2001a, Lichstein 2000). The misconception is particularly evident in the mental health field but is unsupported by the literature.

Ford & Kamerow (1989) reported on 8000 respondents, revealing that the risk of developing depression was much higher in those with preexisting insomnia. Similarly, Eaton et al. (1995) reported that having a sleep problem was the highest precursor in terms of attributable risk, identifying 47% of new cases of depression the following year. Other longitudinal studies have confirmed such findings. One report on 262 older adults suggested that frequency of depressed affect related to poor sleep, even when age, sex, and health status were accounted for (Rodin et al. 1988). Breslau et al.'s (1996) work on 1007 21–30-year-olds found that gender-adjusted relative risk for major depression in people with a history of insomnia was 4.0 (95% CI 2.2–7.0). Weissman et al.'s (1997) epidemiological survey reported an odds ratio of 5.4 (95% CI 2.6–11.1) for first-onset depression in 414 people with insomnia and no psychiatric history. Insomnia was also an independent risk factor for panic disorder and obsessive compulsive disorder. Finally, the Johns Hopkins Precursors Study found that insomnia in young men was indicative of greater risk for depression and psychiatric distress that persists for 30 years (Chang et al. 1997).

Thus, insomnia cannot be accounted for simply as presumed mental disorder. Furthermore, sleep disturbance often fails to resolve upon recovery from depression. However, the possibility that insomnia shares some psychobiological diathesis with anxiety and mood disorder is worthy of further investigation.

Faulty Conditioning

Since first proposed by Bootzin (1972), an understanding of insomnia as the product of maladaptive sleep habits has had considerable appeal. Good sleep is seen as coming under the stimulus control of the bedroom environment, which acts as a discriminative stimulus for sleep (Bootzin et al. 1991). Difficulty falling asleep may result either from failure to establish discriminative stimuli for sleep or the presence of stimuli incompatible with sleep. Poor stimulus control, therefore, might compete with sleep drive by strengthening conditioned arousal, and with circadian timing by doing so at normal bedtime. The insomniac may also nap in an armchair and so strengthen associations between sleep and nonsleeping environments. Stimulus control treatment instructions comprise lying down to sleep only when sleepy, avoiding using the bed for activities other than sleep (sexual activity excepted), getting up if unable to sleep quickly (within 15–20 minutes), repeating rising from bed as necessary throughout the night, getting up the same time every day, and avoiding napping (Bootzin 1972, Bootzin & Epstein 2000).

Only a few studies have investigated conditioning in insomnia. Haynes et al. (1982) compared student insomniacs and noninsomniacs on 12 sleep-incompatible behaviors, but found only one that differentiated the groups. Furthermore, duration of engagement in sleep-incompatible activity was unrelated to SOL. They had previously reported that the number of sleep-incompatible behaviors was not related to sleep difficulty (Haynes et al. 1974). Although over half the chronic insomniacs in Espie et al.'s (1989a) outcome study reported reading or watching TV in bed, there was no comparison group of good sleepers. Tokarz & Lawrence (1974) separated situational (reestablishing bedroom cues for sleep) from temporal components (regularizing sleep routines) and found both reduced SOL in their student sample. Zwart & Lisman (1979) conducted a study of 47 undergraduates assigned to stimulus control (all instructions), temporal control (lie down only when sleepy, rise at same time each day, do not nap), noncontingent control (a fixed number of risings within 20 minutes of retiring), countercontrol (sit up in bed and read, watch TV etc. if unable to sleep), or no treatment. They found countercontrol as effective as stimulus control, suggesting that it may ensure contingent disruption of bed and bedtime as cues for mental arousal. Davies et al. (1986) used countercontrol with older adults and found it moderately effective, although their 30% reduction in wake time after sleep-onset is less than typically reported in this population (Morin et al. 1999c).

In spite of equivocal evidence for the mechanism of effect (i.e., is it a stimulus control paradigm?), there has been little recent work on the conceptual basis of stimulus control. Harvey (2000a) reported that primary insomniacs did not differ

from good sleepers on daytime napping, variable sleep scheduling, whether they stayed in bed or got up when unable to sleep, or on engagement in sleep-incompatible activities. Nevertheless, significantly lower sleep efficiency is typical of insomniacs, and this may evidence the need for improved stimulus control. Interestingly, Bootzin has reported that stimulus control reduces sleep anticipatory anxiety as well as improving sleep (Bootzin et al. 1999). More research is required because stimulus control interventions have consistently been found to be efficacious in meta-analyses (Morin et al. 1994, 1999b; Murtagh & Greenwood 1995). Indeed, they are the only procedures recommended by AASM as comprising "standard" nonpharmacological treatment for insomnia (Chesson et al. 1999).

Poor Chronobiological Timing

In sleep phase disorders people sleep relatively normally, but during the "wrong" hours. In Delayed Sleep Phase Syndrome (DSPS), for example, in which the sleep period is phase delayed, subjects experience sleep-onset insomnia if they attempt to sleep before they are ready to sleep, and in advanced sleep phase syndrome sleep-onset is early and the phase advance results in early waking. In primary insomnia there may be an element of chronobiological dysfunction. For example, some insomniacs go to bed early and spend excessive time in bed either habitually as in the case of older adults, or as a response to having slept poorly on previous nights (Morgan 2000, Morin 1993). This contributes to poor sleep efficiency, which is affected both by TIB and total sleep time (see above), and can be improved by spending less time in bed, sleeping longer, or by a combination of the two. Similarly, napping reduces nighttime sleep drive in adults. The literature may have failed to discriminate adequately between circadian disorders and primary insomnia (Morris et al. 1990, Lack & Wright 1993). In particular, DSPS may not have been identified in studies involving younger populations. Research diagnostic criteria require clarification (Edinger et al. 2002).

It is suggested that, in the absence of competing factors, good sleep is associated with optimal, stable, and accurate circadian timing. Stimulus control instructions contain elements of temporal adjustment (see Lacks 1987, pp. 51–53; Espie 1991, pp. 51–55) and may provide *zeitgebers* (Aschoff 1951) for healthy sleep. Sleep restriction is another technique that may act both as a circadian harmonic and a reinforcer of homeostatic drive. Patients are encouraged to reduce bedtime hours (by staying up late and/or rising earlier) to approximate TIB closer to actual sleep duration (Spielman et al. 1987). Sleep restriction compresses sleep toward greater continuity, reduces wakefulness in bed, and increases sleep efficiency. Once the sleep pattern is improved, TIB may be extended, at a rate of 15 minutes per night per week, until the patient no longer gains further sleep or sleep efficiency is at risk of being reduced. Wohlegemuth & Edinger (2000) reviewed empirical findings on the efficacy of sleep restriction, and the AASM support sleep restriction as a "guideline" intervention for insomnia (Chesson et al. 1999). The synergy between stimulus control and sleep restriction is evident, and they are often presented

together. We use the term "sleep scheduling" for the combination (Espie et al. 1998, 2001b).

Recent interest in using appropriately timed bright light exposure to entrain circadian timing also focuses attention on possible chronobiological explanations of sleep disorder. Early evening bright light shifts the circadian rhythm of core body temperature and improves PSG-defined sleep in sleep-maintenance insomnia (Campbell et al. 1993), and morning light reduces SOL (Lack et al. 1995). Light exposure may also have a role in managing sleep disturbance in dementia (e.g., Satlin et al. 1992).

Physiological Hyper-Arousal

In 1967 Monroe conducted an influential study comparing 16 good sleepers with 16 poor sleepers, suggesting that poor sleepers exhibit heightened autonomic arousal (higher rectal temperature, vasoconstrictions per minute, perspiration rate, skin conductunce, body movements per hour) both prior to and during sleep. This work has been partly replicated (Stepanski et al. 1989), but other studies have failed to demonstrate arousal differentials. Higher levels of hormones indicative of adrenocortical activity have been both supported (Johns et al. 1971, Adam et al. 1986) and denied (Frankel et al. 1973). Other early work reported less rapid decline in heart rate associated with sleep in insomniacs, but others found no significant relationship between sleep-onset and heart rate or frontalis electromyography (EMG) (Haynes et al. 1974, Good 1975, Browman & Tepas 1976). Now that heart rate variability in the progression through sleep is better understood in normal subjects (e.g., Baharav et al. 1995, Bonnet & Arand 1997a), further comparative study seems warranted. Freedman & Sattler (1982) found that, prior to sleep-onset, insomniacs had higher frontalis and chin EMG than good sleepers, and the literature on relaxation and/or biofeedback treatments (e.g., Borkovec & Weerts 1976, Borkovec & Sides 1979, Freedman & Papsdorf 1976) might appear to support muscle tension as a problem in insomnia. However, there is limited evidence of tension reduction as the active mechanism, and posttreatment changes in EMG, heart rate, or respiration have proven elusive (see Espie 1991, pp. 43–45; Bootzin & Rider 1997, pp. 322–26 for review).

Interest in physiological arousal has been rekindled by evidence that insomniacs display measurable neurobiological differences from normal sleepers. Bonnet & Arand (1995) compared 10 objectively defined insomniacs with age-, sex-, and weight-matched controls and found they had significantly increased oxygen use both day and night. They suggested that increased 24-hour metabolic rate could be magnified by stress or viewed as a "higher arousal set point." In a complementary controlled study, 9 subjects with sleep state misperception (SSM) were also found to have increased metabolic rate, but less so than the primary insomniacs (Bonnet & Arand 1997b), raising the possibility that SSM is a mild version of, or precursor to, psychophysiological insomnia. Hyper-arousal has also been investigated in PSG studies. Early reports suggested insomniacs had more beta and fewer

alpha frequencies in their EEG (Freedman 1987, Freedman & Sattler 1982). Recently, Merica et al. (1998) compared spectral characteristics of 20 insomniacs and 19 controls. For all frequencies below beta, insomniacs had slower rise rates and reached lower levels, whereas beta power was increased. In REM, insomniacs showed lower levels in delta and theta bands, whereas power in faster bands was increased. These findings are consistent with slow wave deficiency in insomnia accompanied by hyper-arousal of the CNS, suggesting that insomnia may result from increased cortical activation. They noted, however, that homeostatic control of slow wave activity appeared to be intact in the patient population. A study by Loewy & Bootzin (1998) also investigated hyper-arousal and found that event-related EEG activity, as measured by auditory evoked potentials, provided evidence of information processing during sleep.

Importantly, however, not all objective poor sleepers complain of insomnia, and not all subjective insomniacs have poor sleep (Edinger et al. 2000), suggesting that physiological arousal alone is an insufficient explanation.

Cognitive Hyper-Arousal

It was first reported 25 years ago that poor sleepers complain of mental alertness more than physiological arousal (Evans 1977). Other studies have consistently associated cognitive arousal more strongly with sleep disruption, and "having an overactive mind" has been the attribution rated most highly, both by insomniacs and noninsomniacs (Lichstein & Rosenthal 1980, Nicassio et al. 1985, Broman & Hetta 1994). Espie et al. (1989a) reported that the cognitive items of the Sleep Disturbance Questionnaire (e.g., "my mind keeps turning things over," "I am unable to empty my mind") were the most highly rated; Harvey (2000b) recently replicated these findings. The Sleep Disturbance Questionnaire has been found to have modest internal consistency ($\alpha = 0.67$) (Espie et al. 2000). Although there is no gold standard measure of cognitive activity, the Pre-Sleep Arousal Scale (Nicassio et al. 1985) is widely used and has satisfactory internal consistency for its somatic and cognitive subscales ($\alpha = 0.81$ and $\alpha = 0.76$, respectively). These constructs have some degree of independence (74% unshared variance). The cognitive subscale of the Pre-Sleep Arousal Scale has also demonstrated modest ($r = 0.35$) validity compared with voice-activated recordings of presleep thoughts as a criterion measure (Wicklow & Espie 2000).

Population survey confirms that people dissatisfied with sleep report mental activity near bedtime (Ohayon et al. 1997). However, there are conflicting conclusions from studies of the relationship between cognitive activity and sleep latency. Van Egeren et al. (1983) found that audiotape-recorded cognitions were significantly correlated with subjective sleep latency, but not with polysomnographic (PSG) assessment sleep, whereas Borkovec et al. (1979) and Kuisk et al. (1989) reported more frequent cognitive activity in insomnia confirmed by PSG. Sanavio (1988) reported a low correlation ($r = 0.09$) between presleep intrusion and self-reported sleep latency, and furthermore, found no advantage of a tailored

cognitively focused program in the treatment of sleep-onset insomnia. The possibility, therefore, remains that cognitive arousal is an epiphenomenon of nighttime wakefulness (Freedman & Sattler 1982, Morin 1993).

Support for a cognitive model has also come from studies involving experimental manipulation of presleep cognitive intrusions (e.g., Gross & Borkovec 1982, Hall et al. 1996). However, Haynes et al. (1981) exposed insomniacs and noninsomniacs to brief stressors and found a decrease in subjective and objective SOL among insomniacs on stress nights. They concluded that a mental processing task that disrupts sleep-related cognitive events may decrease SOL, implying that the nature of the intrusions may be critical to the effect upon sleep. Therefore, failure to differentiate thought content could result in limited comparability between studies.

Dysfunctional Thinking

The importance of emotional arousal has been stressed, because affect-laden cognitions are more likely to interfere with sleep (Espie 1991, Morin 1993, Haynes et al. 1981, Coyle & Watts 1991). Beliefs about the negative experiences and consequences of insomnia may foster the clinical complaint. This parallels research on "worry," posited as a generic trait (Barlow 1988, Meyer et al. 1990), and studies on unwanted intrusive thoughts (Rachman & De Silva 1978, Reynolds & Salkovskis 1992). Negative and distressing cognitions are likely to contribute to the development of obsessions; however, it is not the thoughts per se that are untypical or pathological, but the meaning and concern attributed to them (Clark & Purdon 1993, 1995). The conceptual relatedness of nighttime and daytime intrusions, therefore, appears considerable.

Insomniacs have more negative thoughts than good sleepers at bedtime (Nicassio et al. 1985, Van Egeren et al. 1983, Kuisk et al. 1989), and such thinking is reported even when wakened from light sleep (Borkovec et al. 1981). The thoughts of insomniacs may be dependent on emotional state. Investigating the relationship between worry and insomnia, Watts et al. (1994) found that much of the presleep mental activity of "worried insomniacs" revolved around work and general mental activity. In contrast, thoughts of "nonworried insomniacs" focused on the sleep process itself. Insomniacs may also feel less in control of their thinking (Watts et al. 1995). Gendron et al. (1998) reported that insomniacs with comorbid generalized anxiety disorder had greater cognitive activity at bedtime than insomniacs without generalized anxiety disorder, evaluated their thoughts as more intrusive and worrisome, and attempted cognitive avoidance strategies more frequently.

Morin (1993) has argued that beliefs and attitudes play a critical role. He devised a 30-item questionnaire to identify irrational, affect-laden thoughts that intrude prior to sleep-onset (dysfunctional beliefs and attitudes about sleep scale) (Morin 1993, Morin et al. 1993). This scale comprises misconceptions about the causes of insomnia, misattributions or amplifications of the consequences of insomnia,

unrealistic sleep expectations, diminished perceptions of control, and faulty beliefs about sleep-promoting practices. It has satisfactory psychometric properties and sensitivity to change after cognitive-behavioral treatment (CBT) (Espie et al. 2000, Morin et al. 2001), and may help identify subgroups of the insomniac population (Edinger et al. 1998). Furthermore, those with subjective insomnia report more dysfunctional sleep-related cognitions than do those with objective insomnia (Edinger et al. 2000), consistent with the view that cognition/affect may influence sleep report and perhaps mediate insomniac complaint.

Formal analysis of sleep-interfering cognitions has been reported in several studies. Coyle & Watts (1991) used an extended version of the Sleep Disturbance Questionnaire (Espie et al. 1989a) and reported two distinct factors: "sleep attitudes," reflecting anxiety about the sleep process, and "mental activity," reflecting nonspecific cognitive activity. Six factors of nighttime intrusive thoughts, i.e., trivial topics, thoughts about sleep, family and long-term concerns, positive plans and concerns, somatic preoccupations, and work and recent concerns, were identified in a study of young adults (Watts et al. 1994). Extending these findings by using a good-sleeper comparison group, Harvey (2000b) reported that presleep cognitive activity of insomniacs could be distinguished by being more focused upon worry about not getting to sleep, general worries, solving problems, the time, and noises in the house, and less focused upon "nothing in particular."

A study by Fichten et al. (1998) of the thoughts of older adults during wakeful periods yielded a 3-factor solution of generalized positive thinking, generalized negative thinking, and thoughts related to sleep. They suggested that insomniacs use positive thinking as a buffer to combat negative intrusions. Also recently, Wicklow & Espie (2000) obtained voice-activated audiotape recordings of spontaneous thoughts and sleep actigraphic data from 21 poor sleepers over 3 consecutive nights. Content analysis yielded 8 categories of presleep intrusion, and a regression model indicated that thinking about sleep and the anticipated consequences of poor sleep, along with general problem-solving were the strongest predictors of objective SOL. Intrusions were subsumed under one of 3 factors: active problem-solving (e.g., rehearsing/planning events), present state monitoring (e.g., thinking about sleep/not sleeping) and environmental reactivity (e.g., attending to external noises).

Paradox and Ironic Control

This model of insomnia proposes that anxiety responses may be conditioned not only to external, situational cues but also to the individual's behavior (Ascher & Turner 1979, Espie & Lindsay 1985, Espie 1991). Fear of performance failure (insomnia) and of anticipated negative consequences of that failure is described as performance anxiety. In paradoxical treatment counterproductive attempts to fall asleep are replaced by the intention of remaining passively awake or by giving up any direct effort to fall asleep (Ascher & Turner 1980, Fogle & Dyal 1983). This rationale is supportable in that good sleepers do not use any strategies to

fall asleep. Paradoxical intention has demonstrated efficacy in controlled trials (Turner & Ascher 1979, Espie et al. 1989b) and is an intervention that reflects a "moderate degree of clinical certainty" according to the AASM (Chesson et al. 1999). Paradox continues to be used within multicomponent CBT (e.g., Espie et al. 2001b) and may be particularly useful with patients who are resistant and reactive to therapeutic suggestions (Shoham et al. 1995).

Further evidence for this type of mechanism was provided by Ansfield et al. (1996), who explored the effects of different sleep-onset instructions in good sleepers under high or low mental load. Paradoxical wakefulness was found amongst those attempting to sleep while listening to marching music. This was interpreted in terms of Wegner's (1994) theory of a self-loading system that suggests that under certain conditions the thwarted attempt to control a particular mental state can yield the opposite of what is desired. They hypothesized that failure to fall asleep on a few occasions could occur when sleep is attempted under transitory mental loads, such as at times of stress. Eventually, a person's thoughts about being unable to sleep could constitute a debilitating mental load, which when combined with the continuing frustrated desire to fall asleep, could lead to chronic insomnia.

Harvey (2001b) has explored the effects of suppressing presleep cognitive activity on sleep-onset latency. A cohort of insomniacs and good sleepers were allocated to either a suppression condition ("suppress the thought most likely to dominate your thinking as you get into bed") or nonsuppression condition ("think about anything as you get into bed, including the thought you would most likely think about as you go to sleep"). Suppress participants reported longer sleep latencies and poorer sleep quality, regardless of whether they were insomniac or not. Harvey concluded that thought suppression appeared to have the opposite effect in that it prevented sleep-onset, in a manner consistent with Wegner's theory of ironic mental control.

There are parallels between Wegner's theory and the performance anxiety model that gave rise to the adaptation of paradoxical intention from the work of Frankl (1960). Indeed, Ansfield et al. (1996) propose that their results are consistent with theories of cyclic escalation of anxiety disorders (Ascher 1981) and worry about sleep (Borkovec 1982).

AN INTEGRATED PSYCHOBIOLOGICAL MODEL OF NORMAL SLEEP

There are, thus, differing explanations of insomnia, each having some empirical support. The rest of this paper attempts to integrate this evidence into a conceptual framework. A model of the normal sleep process is presented first, because a perspective on the pathway to sleeping well is likely to inform understanding of the development, maintenance, and treatment of insomnia.

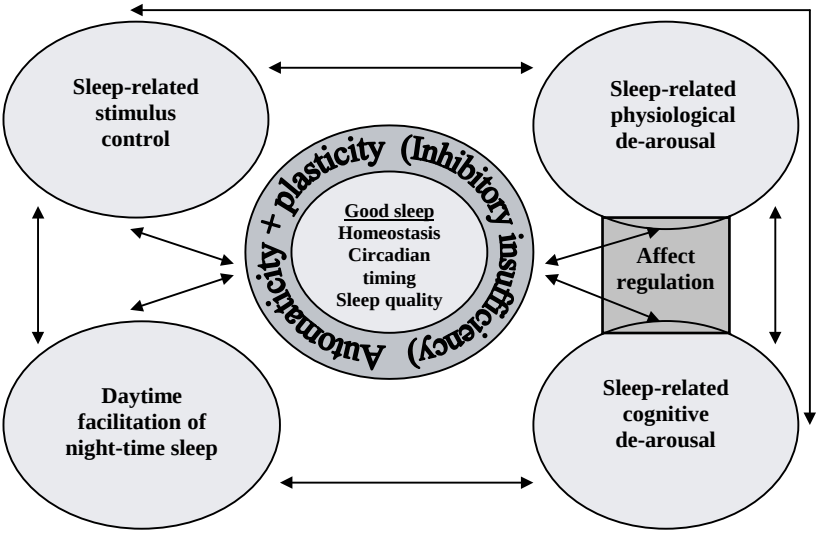


Figure 1 A psychobiological model of good sleep. Insomnia is proposed as resulting from chronic inhibition of one or more of the component processes.

Figure 1 summarizes hypothesized processes and interactions in normal sleep. The model proposes good sleep as the natural state of the human organism—its default state. That is, homeostatic and circadian processes, under normal circumstances, default to good sleep, not to insomnia. The core of the model is involuntary, harmonious interaction between homeostat and timer, which is associated with the self-perception of good quality sleep. Like all neurobehavioral systems, good sleep is assumed to have both functional plasticity and automaticity. These are presented as protective properties, "defending" the core.

Plasticity refers to the "absorb and readjust" capability of the sleep-wake system to accommodate living in the real world where situational and personal factors provoke variance and challenge normal, stable functioning. Night-to-night variability is tolerated but minimized by the good sleeper, for whom the sleep homeostat drives sleep-related behaviors effectively. In acute insomnia the norm would be recovery of sleep pattern, again reflecting the system's plasticity in function and default regression towards good sleep. Automaticity refers to the involuntary nature of the well-adjusted schedule, to the habitual, conditioned associations that are part of its stimulus control paradigm, and to the implicit expectations and assumptions the good sleeper has about sleep continuity and sleep quality. It is tempting to think of the model as the good sleeper's list of ingredients for good sleep, but that would be misleading. The good sleeper is not thought of as following a recipe to produce the perfect sleep. Rather, the good sleeper is regarded as essentially passive because internal and external cues act as automated setting conditions for sleep. Endogenous

cues to sleep such as physical and mental fatigue interact reciprocally with exogenous cues in the home environment. The good sleeper sleeps just as he walks, or talks—without thinking about it. This, presumably, is the insomniac's ambition.

The defensive properties of the good sleep paradigm, therefore, are seen as maintained by four interacting subsystems: sleep-stimulus control, physiological de-arousal, cognitive de-arousal, and daytime facilitation. In a reciprocal way, sleep homeostasis, circadian timing, and sleep quality serve to reinforce and maintain these behavioral, cognitive, and biological processes. That is, good sleep begets good psychobiological preparation for sleep, which begets good sleep.

The model predicts that the good sleeper accurately interprets physiological and mental signs of sleep readiness and, once in the bedroom, the stimulus environment there further reinforces de-arousal. Regular sleep habits for retiring and rising constitute a good predictive framework that exhibits both sensitivity and specificity. In terms of the former, a high percentage of nights that follow this pattern is associated with efficient sleep. In terms of the latter, sleep is highly specific to the bedroom and wakefulness to other environments; i.e., the good sleeper is less likely to lie awake in bed or need to sleep in the day or evening. Effective stimulus control reinforces not only circadian timing and sleep homeostasis but also contributes to cognitive de-arousal because high sleep efficiency precludes cognitive intrusion in bed. Both physiological and cognitive de-arousal are presumed to occur in parallel. Active information-processing recedes as the wake system disengages and the sleep system engages. The good sleeper is less likely to use time in bed for problem-solving or anxious thinking, nor have time to do so because sleep-onset is rapid, and nervous system adaptations proceed unhindered because the good sleeper has few inaccurate expectations or worries about sleep or wakefulness.

Crucially, the good sleeper experiences little affect associated with sleep. Affect regulation, which represents an interaction between cognitive and physiological processes, is proposed as functional when affect is essentially neutral. Dysregulation would occur with strong (negative or positive) emotions, both of which are arousing. Similarly, the good sleeper is seen as having few conscious expectations of sleep and puts no direct effort into the sleep process. Daytime attitudes and behaviors may also facilitate sleep. The good sleeper may make fewer attributions of daytime mood and performance to the preceding night's sleep. For example, when irritable or fatigued, he may be more likely to associate these with proximal events (e.g., pressure of work) than view them as contingencies of sleep pattern or sleep quality. Also, the good sleeper may be more effective at managing state/trait symptoms and/or may put less pressure on sleep to compensate for any excessive daytime routines.

Thus, the common pathway in the maintenance of sleep homeostasis, circadian timing, and sleep quality is proposed as an involuntary process of psychobiological de-arousal within which there is insufficient mental, behavioral, or physiological inhibition to impair the automaticity and plasticity of sleep. Implicit in the model is the expectation of regression towards normal sleep because of its fundamental importance both developmentally and functionally.

UNDERSTANDING INSOMNIA WITHIN THE PSYCHOBIOLOGICAL INHIBITION MODEL

At the simplest level, insomnia may be seen as a failure of automated sleep activation and maintenance. More specifically, insomnia may arise from acute inhibition of one or more of the processes normally contributing to good sleep (precipitating stage), perhaps in predisposed individuals, which may become persistent when chronic inhibition develops, preventing the natural recovery of good sleep (perpetuating stage). This conceptualization seems consistent with the lack of pathology or fundamental disorder of sleep-wake function in primary insomnia. Insomnia is proposed, therefore, as persistent loss of expression of normal sleep. The conceptualization is also consistent with evidence that chronic insomnia is amenable to cognitive-behavioral intervention (Morin et al. 1994, 1999b; Murtagh & Greenwood 1995). CBT may act to overcome inhibitory mechanisms and reestablish adaptive mental and situational "setting conditions" for the restoration of normal sleep.

Automaticity and plasticity would be weakened by inhibitory feedback from one or more of the attendant psychobiological processes (Figure 1 and Table 1). For example, poor stimulus control represented by sleep-incompatible behavior, diminished sleep-wake sensitivity and specificity, and irregular sleep habits could inhibit sleep and maintain insomnia. Similarly, affect-laden thinking would undermine cognitive de-arousal. The notion of "inhibitory sufficiency" is introduced because good sleepers do not necessarily observe all the rules of sleep preparation; e.g., good sleepers may read, drink caffeinated beverages, spend time anxiously reflecting on the day while in bed, be poor daytime copers, etc., without ever becoming insomniac. It helps to account for those individuals who are under chronic stress but who apparently still sleep well. Inhibitory sufficiency, therefore, represents the critical mass of inhibition required to outweigh the stability of an individual's default sleep pattern. Theoretically, it would need to be higher where there is strong sleep homeostasis and/or circadian timing, and less where there is predisposition to insomnia, strong precipitating factors or limited protection afforded by plasticity and automaticity.

Table 1 presents detail of the elements of the model and how they may become inhibited. Poor sleep-related stimulus control would be observed where the bedroom becomes a conditioned focus for waking activity and where the sensitivity/specificity of the sleep-wake schedule is compromised, primarily by remaining awake in bed, but also by sleeping in the daytime. Attempting to sleep longer on recovery nights and spending too long in bed could also inhibit the continuity and automaticity of sleep by reducing sleep efficiency. Stimulus control and sleep restriction treatments may be effective because they counteract the process of inhibition (stay up until sleepy, compress time in bed, etc.) and reinstate default sleep. The instruction to get up if not asleep within 15 minutes may also attenuate the inhibition associated with effort to sleep, as it can be likened to a "give up trying" instruction.

TABLE 1 Insomnia within the psychobiological inhibition model.

Factors contributing to good sleep	Insomnia: factors inhibitory to sleep homeostasis and circadian timing, and to the protection of good sleep afforded by automaticity and plasticity
Sleep stimulus control	
Sleep-compatible conditioning	Conditioned association of sleep-incompatible, waking activities (e.g., reading, watching TV, eating, talking, problem-solving) with bed and bedroom environment; keeping the light on
Sleep-wake sensitivity/specificity	Environmental latitude in sleep and wake behaviors: lying awake in bed either presleep or upon wakening, sleeping in the day, sleeping elsewhere than in bed
Regular sleep habits	Variable and/or reactive patterns: changing times for retiring and rising, extending time in bed to catch up on sleep, sleeping in at weekends, spending longer in bed than current sleep requirement—reduced sleep efficiency
Physiological de-arousal	
Sleep system engagement	Not feeling tired at bedtime, in bed too early, keeping the light on, sleep-incompatible activities, anxiety, trying too hard to sleep, tension, heart rate variability
Wake system disengagement	As above; active thinking and problem-solving, self-monitoring of internal (bodily and mental) cues, hypervigilance, poor sleep hygiene
Good sleep hygiene	Stimulants (e.g., caffeine, nicotine) in excess/near bedtime; alcohol withdrawal symptoms during the night; active exercise late evening; bedroom stuffy, hot, or cold; bed uncomfortable
Cognitive de-arousal	
Minimal cognitive drive	Rehearsing/planning/problem-solving thoughts in bed, thinking about events the previous or next day, preoccupation with sleep/sleeplessness, "stimulus hungry" mind, mind racing, unable to "switch off"
Accurate sleep-wake attribution	Dysfunctional beliefs and attitudes about sleep and consequences of not sleeping, not expecting to sleep, catastrophic thoughts, concern about next day well-being and coping
Affect regulation	
Minimal affect	Worry, anxiety, frustration, negativity or excitement, intensity in emotional tone associated with above cognitive or physiological processes
Minimal effort to sleep	Sleeplessness preoccupying: trying to control sleep/overcome insomnia, attempts to suppress thoughts/suppress affect, self-monitoring of alert/sleepiness state, performance effort to fall asleep, performance anxiety
Daytime facilitation of night sleep	
Accurate wake-sleep attribution	Attribution of impaired daytime mood, attention, performance to quality of sleep; expectation that sleep should compensate; blaming problems on insomnia; fatigue seen as pathognomic of insomnia; perception of self as insomniac
Effective coping skills	Experiencing time pressure; problems relaxing; worry, frustration, low mood; active late into evening; poor wind down

Some insomniacs report not being tired at bedtime, which undermines physiological de-arousal, and evidence of wakefulness when aroused from light sleep suggests slower engagement of the sleep system. Studies revealing elevated autonomic symptoms in poor sleepers suggest that inhibition of normal arousal is problematic. Sleep hygiene is traditionally regarded as a behavioral strategy. However, its components are primarily physiological in terms of the model of sleep inhibition. Excessive use of caffeine and strenuous exercise delay sleep onset, presumably through heightened arousal. Indeed, in one experimental study 400 mg of caffeine three times per day for one week produced increased arousal on metabolic measures and reports typical of insomniac complaint (Bonnet & Arand 1992). Similarly, environmental factors (temperature, humidity, light) could inhibit sleep physiologically. Use of alcohol may lead to dehydration or provoked awakenings because of ethanol metabolism. Observing good sleep hygiene, therefore, would remove some potential inhibitors of sleep. Similarly, relaxation-based treatments may either reciprocally inhibit autonomic activity (Wolpe 1958) and thus counteract the maintenance of physiological arousal or, perhaps more likely, facilitate mental (and physiological) de-arousal.

Problems with cognitive de-arousal appear central to insomnia. Insomniacs report intrusive thinking; some of this is reflective, but much is also worrisome. The active mind is likely to inhibit de-arousal, particularly where accompanied by negatively toned affect. The insomniac typically becomes concerned about sleeplessness and its immediate and longer-term negative consequences. This gives rise to emotional upset, ironic urgency, and performance anxiety associated with failed efforts to regain perceived loss of control over sleep. Indeed, the development of attentional bias for sleep- or insomnia-related thoughts may help to explain the maintenance of sleep disorders even after proximal sources of transient sleeplessness (e.g., stress) have passed. The fact that attempts to suppress thinking produce sleep disturbance even in normal sleepers is further evidence of insomnia arising from cognitively mediated sleep inhibition. Insomniacs often try too hard, thereby obviating passive acceptance of sleep, which is a hallmark of the good sleeper. The success of cognitive strategies for insomnia may depend, therefore, upon the extent to which they disable sleep-interfering mentation and affect. The central paradox is that deliberate efforts to do so fail to emulate the automatic nature of sleep-onset in good sleepers and may exacerbate preexisting sleep inhibition. The less direct mechanisms of behavioral interventions such as stimulus control upon cognitive processes therefore merit further attention, as do techniques using paradoxical instructions.

It is specious, of course, to set up physiological and cognitive arousal in direct competition. The psychobiological inhibition model presumes interaction between physiological, cognitive, affective, and behavioral subsystems, and indeed between daytime and nighttime variables. These relationships are represented by arrows in Figure 1. Their interaction contributes positively to the maintenance of good sleep. The corollary, however, is that interaction would also compound inhibitory effects. Indeed, Born et al. (1999) have demonstrated that normal sleepers, told that they

would be woken at 6 A.M., had significant increases in adrenocorticotropin during the hour preceding waking. This suggests that some form of cognitive priming may be linked to sleep-related biological responses.

Less attention has been paid to how daytime factors could contribute to insomnia. Good sleep may be inhibited by daytime preoccupation with impairments perceived to result from poor sleep. Just as attributional (sleep-wake) error can contribute to sleeplessness during the night, so it may form dysfunctional sleep-related schema during the day (wake-sleep). Self-perception of being insomniac may influence interpretation of everyday experiences, such as feeling fatigued or irritable, in a manner comparable to obsessional disorders in which special meaning is attributed to common thoughts and behaviors. Just as some people differ from controls only in respect of their interpretation of events, some insomniacs have normal or close-to-normal sleep, but perceive it to be abnormal. Insomniacs have been characterized as prone to worry and internalizing of anxiety. Such traits may be reinforced in the day, not only during the night. Furthermore, failure to manage daytime concerns effectively may put pressure on sleep when, in the absence of other stimulation or distraction, the insomniac spends time thinking, rehearsing, planning, and worrying. The model, therefore, includes poor coping skills and disturbed daytime affect as factors that may undermine preparation for sleep and may contribute to behavioral and cognitive sleep inhibition.

It may seem counterintuitive or even arbitrary to talk in terms of inhibition of de-arousal rather than excitation (hyper-arousal). However, the definition of insomnia as failure of expression of an essentially normal underlying sleep process is defensible. To remain awake the insomniac does not need to be hyper-aroused any more than one needs to be hyper-aroused at any other time to remain awake. Hyper-arousal is a sufficient but not necessary precondition to wakefulness. Furthermore, even if hyper-arousal were to occur initially to delay sleep it would not follow that remaining hyper-aroused would be necessary to stay awake thereafter. By comparison, if an individual is becoming sleepy (de-aroused) but subsequently becomes wakeful, it only seems necessary to infer that there has been attenuation of the de-arousal, and that explanation also seems sufficient. It is not being argued that hyper-arousal never occurs, but rather that inhibition of de-arousal does always occur. Inhibition is the lowest common denominator that can be reliably deduced. It is suggested that insomnia as a disorder of initiating or maintaining sleep is at its core a disorder of sleep engagement, not a disorder of excessive arousal.

Nevertheless, the model does account for hyper-arousal in insomnia. Hyper-arousal clearly would inhibit sleep, but it is hypothesized that there is within-group variability in insomnia (i.e., not all insomniacs are hyper-aroused). Variability between populations of insomniacs studied might explain apparently discrepant results reporting both physiological and mental measurement. Physiological hyper-arousal may be a distinct subtype of insomnia. De-arousal, however, is suggested as the reliable correlate of good quality sleep; anything else at or above a level sufficient to inhibit sleep may be associated with insomnia.

Why then has cognitive arousal been more strongly associated with insomnia than physiological arousal? It is suggested that cognition is invariably symptomatic of insomniacs' subjective nighttime experience, whereas physiological arousal is less invariably so. Accordingly, when asked to rate or attribute sleeplessness, insomniacs are likely to stress mentation and/or affect rather than physiological symptoms. There may be interpretation bias, and the possibility that cognitive activity is simply an epiphenomenon of sustained wakefulness or a by-product of the measurement process cannot yet be excluded. However, the cognitive/affective component of primary insomnia does seem to be a sine qua non in clinical practice. Like physiological arousal, cognitive arousal may represent a continuous dimension from de-arousal to hyper-arousal, exhibiting individual variability, but once again, de-arousal appears to be the correlate of good sleep. This helps to explain the common finding that insomniacs consistently appear hyper-aroused, relative to good sleepers, on measures such as the cognitive subscale of the Pre-Sleep Arousal Scale. It cannot be stated with conviction that they are hyper-aroused in any absolute sense, but it may be reasonably inferred that they are not de-arousing.

It should be noted that the model can also be applied to sleep-maintenance insomnia. This subtype is more commonly associated with mid- to late-life, when a propensity to lighter, more broken sleep develops and automaticity and plasticity may be compromised. It is suggested that in sleep-maintenance insomnia there is inhibition of the rapid, automated de-arousal response to the brief wakeful experiences typically found in older good sleepers. This results in more conscious processing of arousals, more frequent arousals, and times of extended wakefulness with difficulty returning to sleep. One way in which inhibition might occur would be through attentional bias for arousal and threat-related cues. The insomniac may selectively attend to brief arousals in the night and interpret these by means of established schema as evidence of inability to sleep. This could then set up the pernicious cycle of inaccurate attribution, affect, effort to sleep, physiological arousal and engagement of the wake system.

It is proposed, therefore, that the common pathway in insomnia is inhibition of the expression of normal sleep. It is further suggested that, whereas any of the subsystems may contribute to inhibition, it is cognitive/affective factors that serve as the "activating agent" for the phenomenology of insomnia and presentation of clinical complaint. This is evidenced by the consistent association of intrusive, affect-laden cognitions with primary insomnia and by the fact that some self-reported good sleepers sleep as poorly as insomniacs, but without experiencing concerns about sleep (Edinger et al. 2000). In the psychobiological inhibition model such cognitions are presumed to pervade nighttime and/or daytime thinking. A corollary to the suggestion of activation is that, if CBT intervention is to be effective, it must somehow close this cognitive/affective gate. This again is consistent with the proposition that stimulus control may achieve its effects by fostering cognitive de-arousal. Similarly, sleep restriction might be regarded as a paradigm involving behavioral experiments to evaluate assumptions about sleep requirements and their consequences. The circadian readjustments that form part

of both stimulus control and sleep restriction may, of course, take place without synchronous cognitive conceptual shift, but if that is the case it is suggested that insomnia complaint is likely to persist.

Relationship of the Psychobiological Inhibition Model to Other Models of Insomnia

Espie (1991, pp. 39–56) proposed a framework in which insomnia was conceptualized as mediating from activation of nervous system (central, autonomic), psychological (cognitive, emotional), or environmental (situational, temporal) arousal. These elements can all be accommodated in the current model, although the emphasis is now upon inhibition, impairing the automaticity of normal sleep. Daytime factors are also factored into the new model, and further detail of the cognitive dimension (from Espie 1992, Espie & Wicklow 2001) is presented in the cognitive de-arousal component.

Morin (1993, pp. 46–60) also presented an integrative conceptualization of insomnia. He suggested that hyper-arousal (emotional, cognitive, physiologic) is the central mediating feature of insomnia, which interacts with dysfunctional cognitions, maladaptive habits, and perceived consequences of insomnia. Although the psychobiological inhibition model makes no requirement of hyper-arousal, Morin's emphasis on cognitive factors parallels the cognitive/affective activation agent presented here. The role of dysfunctional thoughts and beliefs in the psychobiological inhibition model is consistent with Edinger et al.'s (2000) interpretation that these influence self-perception of insomnia and insomniac report. Morin also stressed the bi-directional influence of the components, such that consequences often become causes and vice versa, similar to the proposed reciprocal interaction of the elements of the psychobiological inhibition model.

Perlis et al. (1997) discussed discrepancies between PSG and subjective appraisal of sleep. They suggested that inconsistencies may be explained by the presence of high frequency EEG activity in insomnia around sleep-onset, which interferes with the development of mesograde amnesia, and results in insomniacs having blurred phenomenological distinction between sleep and wakefulness. The psychobiological inhibition model can accommodate such an association but would suggest that cortical arousal represents a failure to de-arouse, with hyper-arousal presenting only in some insomniacs. The reciprocal interaction between maintained physiological and psychological processes would inhibit both the transition to slower EEG activity and the experience of sleep-onset, but the model would suggest that it is cognitive/affective inhibition that is the more likely activating factor.

It is also noteworthy that the psychobiological inhibition model is consistent with Spielman's (1991) model of insomnia acquisition (presented above) and with Edgar's (1996) "opponent process" model. The latter proposes that circadian timing promotes wakefulness and opposes sleep drive, and that prolonged wakefulness leads to compensatory sleep responses. The psychobiological inhibition model assumes such an interaction between homeostat and timer, but also describes how

such an automated interaction may be inhibited by thoughts, emotions, and behavioral changes.

Some Implications of the Cognitively Activated Psychobiological Inhibition Model

This model raises testable hypotheses and suggests mechanisms for treatment effects. It is only possible to touch upon some of these. The central premise is that in insomnia the expression of normal sleep is chronically inhibited. Therefore, further study of sleep-related de-arousal in good sleepers is required. The model predicts phenomenology comprising attitudinal passivity towards sleep, with neutral expectations, compared with the negativity and worry of the insomniac. In terms of automaticity, it is suggested that the good sleeper is like the experienced car driver who executes a complex series of operations with minimal attentional load. In comparison, the insomniac is like the anxious learner driver: vigilant, deliberate, and errorful. The concept of automaticity in human learning has long been discussed as part of information-processing theory (Shiffrin & Schneider 1977). Its application to sleep and insomnia could be fruitful. The ability to automate is, presumably hard-wired in order to overcome problems associated with an otherwise limited-capacity information-processing system. Theories of paradox and ironic mental control are interesting because they emphasize allowing events to take their natural course, and de-emphasize the need for control and success. Effort to sleep and suppression of wakefulness, and its associated mental activity, may disengage automaticity.

Study of within-subject as well as within-group variability may lead to greater appreciation of how similarly or differently good sleepers and insomniacs respond to good and bad nights, in terms of their expectations, affect, and behavior. Furthermore, because cognitive/affective activation is seen as central, insomniacs may be differentiated not only from good sleepers, but also from noncomplaining poor sleepers. It seems important to identify this latter group for formal study and to consider degree/severity of complaint as a correlate of mental arousal/concern. The latter may be an inverse correlate of automaticity. Good sleepers and noncomplaining poor sleepers may make more benign attributions after a bad night, leaving automaticity intact. It is further suggested that sleep state misperception (SSM) may represent cognitive activation and perceived severity equivalent to that found in primary insomnia, thus differentiating SSM from good sleepers and noncomplaining poor sleepers, but not from insomniacs. Longitudinal study of SSM is required to investigate whether or not objective sleep disturbance develops over time. There is a need, however, to develop better measures of sleep quality, with sound psychometric properties, and to investigate the interrelationships between sleep quality, cognitive arousal, dysfunctional beliefs, attributional error, attentional bias, and affect, both in SSM and insomnia.

Comparison of the relapse patterns of various subgroups after bad nights or periods of poorer sleep would be interesting from another perspective. The interaction

of sleep homeostasis and circadian timing is thought to demonstrate impaired plasticity in insomniacs, who present sleep pattern variability. Their sleep-wake rhythm may be more brittle, and the repayment of sleep debt may not function as efficiently. Unfortunately, research data are usually analyzed as weekly mean values, thus concealing within-subject and within-group variability in sleep. Individual raw score variance has been reported only occasionally in insomnia, but further work using such data would be valuable. Theoretically, stability in sleep pattern would be displayed by small raw score standard deviations, and plasticity would be demonstrated by regression from higher values (e.g., at times of acute insomnia) to lower values. The time taken to accommodate and adjust from above to below some threshold value might be a useful measure of plasticity.

The "setting conditions" for insomnia are potentially wide-ranging, and further study employing experimental manipulation of situational, autonomic, mental, and affective variables would be valuable. The model would predict that only manipulations with a cognitive/affective activating component would lead to subjective sleep concern, which can vary independently of sleep disruption. The concept of inhibitory sufficiency requires investigation, and methods need to be developed to titrate experimental "doses" so that outcomes can be related to inputs. For example, if an experiment was conducted on the countercontrol procedure, it would be helpful to use standard stimuli (e.g., text to read, TV program to watch) validated for emotional valence, intrusiveness, imagery potential, etc. It should be borne in mind, of course, that disturbance of a single night is not insomnia, but only a bad night. Care must be taken, therefore, also to consider inhibitory sufficiency from the clinical perspective of generalized and enduring sleep disturbance where, presumably, retrospective experience influences response to prospective experimental factors.

The proposed model suggests that maintained arousal is the necessary and sufficient precondition for insomnia. Clearly, this hypothesis requires validation, and a between-group repeated-measures model utilizing daytime, pre-bedtime, presleep, and early-sleep measures of physiological and cognitive arousal in insomniacs and good sleepers would seem appropriate. This would also identify cases of hyper-arousal, proposed as a subpopulation of insomnia. In terms of intervention, the model suggests that cognitive behavioral strategies may act via the common pathway of disengaging mechanisms inhibitory to de-arousal, and facilitating reestablishment of automated normal sleep. Given the proposed importance of cognitive/affective de-activation, it would be particularly useful to investigate stimulus control and sleep restriction as de-activating in this context e.g., attitudinal shift, relinquishing control, precluding worry from bed. Their contribution to circadian timing and homeostatic drive, along with such de-activation, may explain the positive clinical outcomes achieved using these procedures. Specific cognitive interventions need to be explored in terms of direct versus indirect action upon sleep-related variables. Automaticity would predict that indirect (e.g., paradoxical) methods would be more efficacious than those that focus attention and intervention directly upon sleep or upon sleep-related thoughts.

CONCLUSION

Although there are a number of perspectives on the etiology and maintenance of insomnia, each of which has some empirical support, the conceptual basis lags some way behind treatment methodology and the evaluation of efficacy and effectiveness. An integrated psychobiological model has been proposed that differentiates insomnia from normal sleep in terms of inhibitory mechanisms and processes. It is hoped that the hypotheses raised here will stimulate interest in further experimental and clinical study, and that the resultant research process will improve both understanding and management of this common condition.

ACKNOWLEDGMENTS

The author was supported by research grants from Chief Scientist Office, Scottish Executive Health Department: Novel Health Research Initiative and CZH/4/2. Thanks to Dr. Charles M. Morin, Université Laval, Québec City; Dr. Daniel J. Buysse, Western Psychiatric Institute and Clinics, Pittsburgh, PA; and Dr. Allison G. Harvey, University of Oxford, UK for their helpful comments on an earlier version of this manuscript.

LITERATURE CITED

Adam K, Tomeny M, Oswald I. 1986. Physiological and psychological differences between good and poor sleepers. *J. Psychiatr. Res.* 20:301–16

Am. Psychiatr. Assoc. 1994. *Diagnostic and Statistical Manual of Mental Disorders* (*DSM–IV*). Washington, DC: APA. 4th ed.

Am. Sleep Disorders Assoc. 1995a. Practice parameters for the use of polysomnography in the evaluation of insomnia. *Sleep* 18:55–57

Am. Sleep Disorders Assoc. 1995b. Practice parameters for the use of actigraphy in the clinical assessment of sleep disorders. *Sleep* 18:285–87

Am. Sleep Disorders Assoc. 1997. *International Classification of Sleep Disorders: Diagnostic and Coding Manual*. Rochester, MA: ASDA. Revised ed.

Ansfield ME, Wegner DM, Bowser R. 1996. Ironic effects of sleep urgency. *Behav. Res. Ther.* 34:523–31

Ascher LM. 1981. Employing paradoxical intention in the treatment of agoraphobia. *Behav. Res. Ther.* 19:533–42

Ascher LM, Turner RM. 1979. Paradoxical intention and insomnia: an experimental investigation. *Behav. Res. Ther.* 17:408–11

Ascher LM, Turner RM. 1980. A comparison of two methods for the administration of paradoxical intention. *Behav. Res. Ther.* 18:121–26

Aschoff J. 1951. Die 24 stunden Periodik der Maus unter konstanten umge-bungs bedingungen. ("The 24-hour period of the mouse under constant environmental conditions.") *Naturwissenschaften* 38:506–7

Baharav A, Kotagal S, Gibbons V, Rubin BK, Pratt G, et al. 1995. Fluctuations in autonomic nervous activity during sleep displayed by power spectrum analysis of heart rate variability. *Neurology* 45:1183–87

Barlow DH. 1988. *Anxiety and its Disorders*. New York: Guilford

Bixler EO, Kales A, Soldatos CR, Kales JD,

Healy B. 1979. Prevalence of sleep disorders in the Los Angeles metropolitan area. *Am. J. Psychiatry* 136:1257–62

Bliwise DL. 1993. Sleep in normal aging and dementia. *Sleep* 16:40–81

Bonnet MH, Arand DL. 1992. Caffeine use as a model of acute and chronic insomnia. *Sleep* 15:526–36

Bonnet MH, Arand DL. 1995. 24 hour metabolic rate in insomniacs and matched normal sleepers. *Sleep* 18:581–88

Bonnet MH, Arand DL. 1997a. Heart rate variability: sleep stage, time of night, and arousal influences. *Electroencephalogr. Clin. Neurophysiol.* 102:390–96

Bonnet MH, Arand DL. 1997b. Physiological activation in patients with sleep state misperception. *Psychosom. Med.* 59:533–40

Bootzin RR. 1972. A stimulus control treatment for insomnia. *Proc. Am. Psychol. Assoc.*, pp. 395–96

Bootzin RR, Epstein DR. 2000. Stimulus control. See Lichstein & Morin 2000, pp. 167–84

Bootzin RR, Epstein D, Wood JM. 1991. Stimulus control instructions. See Hauri 1991, pp. 19–28

Bootzin RR, Lack L, Wright H. 1999. Efficacy of bright light and stimulus control instructions for sleep onset insomnia. *Sleep* 22(Suppl. 1):53–54

Bootzin RR, Rider SP. 1997. Behavioural techniques and biofeedback for insomnia. See Pressman & Orr 1997, pp. 315–28

Borbely AA. 1994. Sleep homeostasis and models of sleep regulation. In *Principles and Practice of Sleep Medicine*, ed. MH Kryger, T Roth, WC Dement, pp. 309–20. Philadelphia, PA: Saunders. 1067 pp. 2nd ed.

Borkovec TD. 1982. Insomnia. *J. Consult. Clin. Psychol.* 50:880–95

Borkovec TD, Grayson JB, O'Brien GT, Weerts TC. 1979. Relaxation treatment of pseudoinsomnia and idiopathic insomnia; an electroencephalographic evaluation. *J. Appl. Behav. Anal.* 12:37–54

Borkovec TD, Lane TW, Van Oot PA. 1981. Phenomenology of sleep among insomniacs and good sleepers: wakefulness experience when cortically asleep. *J. Abnorm. Psychol.* 90:607–9

Borkovec TD, Sides JK. 1979. Critical procedural variables related to the physiological effects of progressive relaxation: a review. *Behav. Res. Ther.* 17:119–25

Borkovec TD, Weerts TC. 1976. Effects of progressive relaxation on sleep disturbance: an electroencephalographic evaluation. *Psychosom. Med.* 38:173–80

Born J, Hansen K, Marshall L, Molle M, Fehm HL. 1999. Timing the end of nocturnal sleep. *Nature* 397:29–30

Breslau N, Roth T, Rosenthal L, Andreski P. 1996. Sleep disturbance and psychiatric disorders: a longitudinal epidemiological study of young adults. *Biol. Psychiatry* 39:411–18

Broman JE, Hetta J. 1994. Perceived pre-sleep arousal in patients with persistent psychophysiologic and psychiatric insomnia. *Nord. J. Psychiatry* 48:203–7

Browman C, Tepas D. 1976. The effects of presleep activity on all-night sleep. *Psychophysiology* 13:536–40

Buysse DJ. 2000. Rational pharmacotherapy for insomnia: time for a new paradigm. *Sleep Med. Rev.* 4:521–27

Buysse DJ, Monk TH, Reynolds CF, Mesiano D, Houck PR, et al. 1993. Patterns of sleep episodes in young and elderly adults during a 36-hour constant routine. *Sleep* 16:632–37

Buysse DJ, Reynolds CF, Monk TH, Berman SR, Kupfer DJ. 1989. The Pittsburgh Sleep Quality Index: a new instrument for psychiatric practice and research. *Psychiatry Res.* 28:193–213

Campbell SS, Dawson D, Anderson MW. 1993. Alleviation of sleep maintenance insomnia with timed exposure to bright light. *J. Am. Geriatr. Soc.* 41:829–36

Carskadon MA, Brown ED, Dement WC. 1982. Sleep fragmentation in the elderly: relationship to daytime sleep tendency. *Neurobiol. Aging* 3:321–27

Carskadon MA, Dement WC. 1981. Cumulative effects of sleep restriction on daytime sleepiness. *Psychophysiology* 18:107–13

Chambers MJ, Keller B. 1993. Alert insomniacs: Are they really sleep deprived? *Clin. Psychol. Rev.* 13:649–66

Chang PP, Ford DE, Mead LA, Patrick-Cooper L, Klag MJ. 1997. Insomnia in young men and subsequent depression. The Johns Hopkins Precursors Study. *Am. J. Epidemiol.* 146:105–14

Chesson AL, Anderson WM, Littner M, Davila D, Hartse K, et al. 1999. Practice parameters for the nonpharmacologic treatment of chronic insomnia. *Sleep* 22:1128–33

Chesson AL, Hartse K, Anderson WM, Davila D, Johnson S, et al. 2000. Practice parameters for the evaluation of chronic insomnia. *Sleep* 23:237–41

Clark DA, Purdon CL. 1993. New perspectives for a cognitive theory of obsessions. *Aust. Psychol.* 28:161–67

Clark DA, Purdon CL. 1995. The assessment of unwanted intrusive thoughts: a review and critique of the literature. *Behav. Res. Ther.* 33:967–76

Coates TJ, Killen JD, George J, Marchini E, Silverman S, et al. 1982. Estimating sleep parameters: a multitrait-multimethod analysis. *J. Consult. Clin. Psychol.* 50:345–52

Coyle K, Watts FN. 1991. The factorial structure of sleep dissatisfaction. *Behav. Res. Ther.* 29:513–20

Davies R, Lacks P, Storandt M, Bertelson AD. 1986. Countercontrol treatment of sleep-maintenance insomnia in relation to age. *Psychol. Aging* 1:233–38

Dement W. 1960. The effect of dream deprivation. *Science* 131:1705–7

Douglas NJ, Thomas S, Tan MA. 1992. The clinical value of polysomnography. *Lancet* 339:347–50

Eaton WW, Badawi MD, Melton B. 1995. Prodromes and precursors: epidemiologic data for primary prevention of disorders with slow onset. *Am. J. Psychiatry* 152:967–72

Edgar DM. 1996. Circadian control of sleep/wakefulness: implications in shiftwork and therapeutic strategies. In *Physiological Basis of Occupational Health: Stressful Environments*, ed. K Shiraki, S Sagawa, MK Yousef, pp. 253–65. Amsterdam: Academic

Edinger JD, Glenn DM, Bastian LA, Fins AI. 1998. The roles of dysfunctional cognitions and other person factors in mediating insomnia complaints. *Sleep* 21(Suppl.):144

Edinger JD, Stout AL, Hoelscher TJ. 1988. Cluster analysis of insomniacs' MMPI profiles: relation of subtypes to sleep history and treatment outcome. *Psychosom. Med.* 50:77–87

Edinger JD, Sullivan RJ, Bastian LA, Hope TV, Young M, et al. 2000. Insomnia and the eye of the beholder: Are there clinical markers of objective sleep disturbances among adults with and without insomnia complaints? *J. Consult. Clin. Psychol.* 68:586–93

Edinger JD, Wohlgemuth W. 1999. The significance and management of persistent primary insomnia: the past, present and future of behavioral insomnia therapies. *Sleep Med. Rev.* 3:101–18

Edinger JD, et al. 2002. AASM:RDC paper. In preparation

Espie CA. 1991. *The Psychological Treatment of Insomnia*. Chichester, UK: Wiley. 260 pp.

Espie CA. 1992. *Getting ready for bed: cognitive tasks and outcomes which promote good sleep*. Presented at World Congr. Cogn. Ther., Toronto

Espie CA. 1999. Cognitive behaviour therapy as the treatment of choice for primary insomnia. *Sleep Med. Rev.* 3:97–99

Espie CA. 2000. Assessment and differential diagnosis of insomnia. See Lichstein & Morin 2000, pp. 81–108

Espie CA, Brindle SJ, Tessier S, Dawson S, Hepburn T, et al. 1998. Supervised cognitive-behavior therapy for insomnia in general medical practice—preliminary results from the West of Scotland program. In *Behavior and Cognitive Therapy Today: Essays in Honour of Hans J. Eysenck*, ed. E Sanavio, pp. 67–75. Amsterdam: Elsevier. 339 pp.

Espie CA, Brooks DN, Lindsay WR. 1989a. An evaluation of tailored psychological treatment for insomnia in terms of statistical and

clinical measures of outcome. *J. Behav. Ther. Exp. Psychiatry* 20:143–53

Espie CA, Inglis SJ, Harvey L. 2001a. Predicting clinically significant response to cognitive behavior therapy (CBT) for chronic insomnia in general practice: analyses of outcome data at 12 months post-treatment. *J. Consult. Clin. Psychol.* 69:58–66

Espie CA, Inglis SJ, Harvey L, Tessier S. 2000. Insomniacs' attributions: psychometric properties of the Dysfunctional Beliefs and Attitudes about Sleep Scale and the Sleep Disturbance Questionnaire. *J. Psychosom. Res.* 48:141–48

Espie CA, Inglis SJ, Tessier S, Harvey L. 2001b. The clinical effectiveness of cognitive behaviour therapy for chronic insomnia: implementation and evaluation of a sleep clinic in general medical practice. *Behav. Res. Ther.* 39:45–60

Espie CA, Lindsay WR. 1985. Paradoxical intention in the treatment of chronic insomnia: six cases illustrating variability in therapeutic response. *Behav. Res. Ther.* 23:703–9

Espie CA, Lindsay WR, Brooks DN, Hood EH, Turvey T. 1989b. A controlled comparative investigation of psychological treatments for chronic insomnia. *Behav. Res. Ther.* 27:51–56

Espie CA, Wicklow A. 2001c. Cognitive therapy for insomnia. In *Sleep and Psychiatry*, ed. C Shapiro, EP Sloan. Cambridge: Cambridge Univ. Press. In press

Evans FJ. 1977. Subjective characteristics of sleep efficiency. *J. Abnorm. Psychol.* 86:561–64

Fichten CS, Libman E, Creit L, Amsel R, Tagalakis V, et al. 1998. Thoughts during awake times in older good and poor sleepers—the self-statement test: 60+. *Cogn. Res. Ther.* 22:1–20

Fogle DO, Dyal JA. 1983. Paradoxical giving up and the reduction of sleep performance anxiety in chronic insomniacs. *Psychoth.: Theory, Res. Practice* 20:21–30

Foley DJ, Monjan AA, Brown SL, Simonsick EM, Wallace RB, et al. 1995. Sleep complaints among elderly persons: an epidemiologic study of three communities. *Sleep* 18:425–33

Ford DE, Kamerow DB. 1989. Epidemiologic study of sleep disturbances and psychiatric disorders. *JAMA* 262:1479–84

Frankel BL, Buchbinder R, Coursey RD, Snyder F. 1973. Sleep patterns and psychological test characteristics of chronic primary insomniacs. *Sleep Res.* 2:149

Frankel BL, Buchbinder R, Coursey RD, Snyder F. 1976. Recorded and reported sleep in primary chronic insomnia. *Arch. Gen. Psychiatry* 33:615–23

Frankl VE. 1960. Paradoxical intention: a logotherapeutic technique. *Am. J. Psychoth.* 14:520–35

Freedman R. 1987. Chronic insomniacs: replication of Monroe's findings. *Psychophysiology* 24:721–22

Freedman R, Papsdorf J. 1976. Biofeedback and progressive relaxation treatment of sleep-onset insomnia: a controlled all-night investigation. *Biofeedback Self-Regul.* 1: 253–71

Freedman R, Sattler HI. 1982. Physiological and psychological factors in sleep-onset insomnia. *J. Abnorm. Psychol.* 91:380–89

Gallup Org. 1991. *Sleep in America.* Princeton, NJ: Gallup

Gendron L, Blais FC, Morin CM. 1998. Cognitive activity among insomniac patients. *Sleep* 21:130

Gillin JC, Byerley WF. 1990. The diagnosis and management of insomnia. *N. Engl. J. Med.* 322:239–48

Good R. 1975. Frontalis muscle tension and sleep latency. *Psychophysiology* 12:465–67

Gross RT, Borkovec TD. 1982. Effects of a cognitive intrusion manipulation on the sleep-onset latency of good sleepers. *Behav. Ther.* 13:117–24

Hall M, Buysse DJ, Reynolds CF, Kupfer DJ, Baum A. 1996. Stress related intrusive thoughts disrupt sleep onset and continuity. *Sleep Res.* 25:163

Harvey AG. 2000a. Sleep hygiene and sleep-onset insomnia. *J. Nerv. Mental Dis.* 188:53–55

Harvey AG. 2000b. Pre-sleep cognitive activity in insomnia: a comparison of sleep-onset insomniacs and good sleepers. *Br. J. Clin. Psychol.* 39:275–86

Harvey AG. 2001a. Insomnia: symptom or diagnosis. *Clin. Psychol. Rev.* In press

Harvey AG. 2001b. Attempted suppression of pre-sleep cognitive activity in insomnia. *Cogn. Res. Ther.* In press

Hauri P, ed. 1991. *Case Studies in Insomnia*. New York: Plenum

Haynes SN, Adams AE, Franzen M. 1981. The effects of presleep stress on sleep-onset insomnia. *J. Abnorm. Psychol.* 90:601–6

Haynes SN, Adams AE, West S, Kamens L, Safranek R. 1982. The stimulus control paradigm in sleep-onset insomnia: a multimethod assessment. *J. Psychosom. Res.* 26: 333–39

Haynes SN, Follingstad DR, McGowan WT. 1974. Insomnia: sleep patterns and anxiety levels. *J. Psychosom. Res.* 18:69–74

Healey ES, Kales A, Monroe LJ, Bixler EO, Chamberlin K, et al. 1981. Onset of insomnia: role of life-stress events. *Psychosom. Med.* 43:439–51

Hoch CC, Dew MA, Reynolds CF, Buysse DJ, Nowell PD, et al. 1997. Longitudinal changes in diary- and laboratory-based sleep measures in healthy "old old" and "young old" subjects: a three-year follow-up. *Sleep* 20:192–202

Horne J. 1988. *Why We Sleep*. Oxford: Oxford Univ. Press. 319 pp.

Johns MW, Gay MP, Masterton JP, Bruce DW. 1971. Relationship between sleep habits, adrenocortical activity and personality. *Psychosom. Med.* 3:499–508

Kales A, Bixler EO, Vela-Bueno A, Cadieux RJ, Soldatos CR, et al. 1984. Biopsychobehavioural correlates of insomnia. III. Polygraphic findings of sleep difficulty and their relationship to psychopathology. *Intern. J. Neurosci.* 23:43–56

Kales A, Vgontzas AN. 1992. Predisposition to and development and persistence of chronic insomnia: importance of psychobehavioural factors. *Arch. Intern. Med.* 152:1570–72

Kramer M. 2000. Hypnotic medication in the treatment of chronic insomnia: non nocere! Doesn't anyone care? *Sleep Med. Rev.* 4:529–41

Kripke D. 2000. Hypnotic drugs: deadly risks, doubtful benefits. *Sleep Med. Rev.* 4:5–20

Kuisk KA, Bertelson AD, Walsh JK. 1989. Presleep cognitive hyperarousal and affect as factors in objective and subjective insomnia. *Percept. Mot. Skills* 69:1219–25

Lack L, Wright H. 1993. The effect of evening bright light in delaying the circadian rhythms and lengthening the sleep of early morning awakening insomniacs. *Sleep* 16:436–43

Lack L, Wright H, Paynter D. 1995. The treatment of sleep onset insomnia with morning bright light. *Sleep Res.* 24A:338

Lacks P. 1987. *Behavioral Treatment for Persistent Insomnia*. New York: Pergamon. 164 pp.

Leger D, Levy E, Paillard M. 1999. The direct economic costs of insomnia in France. *Sleep* 22(Suppl. 2):394–401

Lichstein KL. 2000. Secondary insomnia. See Lichstein & Morin 2000, pp. 297–320

Lichstein KL, Morin CM. 2000. *Treatment of Late Life Insomnia*. Thousand Oaks, CA: Sage. 354 pp.

Lichstein KL, Rosenthal TL. 1980. Insomniacs' perceptions of cognitive versus somatic determinants of sleep disturbance. *J. Abnorm. Psychol.* 89:105–7

Lichstein KL, Wilson NM, Noe SL, Aguillard RN, Bellur SN. 1994. Daytime sleepiness in insomnia: behavioral, biological and subjective indices. *Sleep* 17:693–702

Loewy DH, Bootzin RR. 1998. Event-related potential measures of information processing in insomniacs at bedtime and during sleep. *Sleep* 21(Suppl.):98

Lundh LG, Broman JE, Hetta J. 1995. Personality traits in patients with persistent insomnia. *Pers. Individ. Differ.* 18:393–403

Mellinger GD, Balter MB, Uhlenhuth EH. 1985. Insomnia and its treatment: prevalence and correlates. *Arch. Gen. Psychiatry* 42:225–32

Mendelson WB. 1995. Long-term follow-up of chronic insomnia. *Sleep* 18:698–701

Mendelson WB, Garnett D, Linnoila M. 1984. Do insomniacs have impaired daytime functioning? *Biol. Psychiatry* 19:1261–64

Merica H, Blois R, Gaillard JM. 1998. Spectral characteristics of sleep EEG in chronic insomnia. *Eur. J. Neurosci.* 10:1826–34

Meyer TJ, Miller ML, Metzger RL, Borkovec TD. 1990. Development and validation of the Penn State Worry Questionnaire. *Behav. Res. Ther.* 28:487–95

Monk TH, Reynolds CF, Machen MA, Kupfer DJ. 1992. Daily social rhythms in the elderly and their relationship to objectively recorded sleep. *Sleep* 15:322–29

Moore-Ede MC, Sulzman FM, Fuller CF. 1982. *The Clocks that Time Us*. Cambridge, MA: Harvard Univ. Press

Morgan K. 2000. Sleep and aging. See Lichstein & Morin 2000, pp. 3–36

Morgan K, Clarke D. 1997. Risk factors for late-life insomnia in a representative general practice sample. *Br. J. Gen. Pract.* 47:166–69

Morin CM. 1993. *Insomnia: Psychological Assessment and Management*. New York: Guilford. 238 pp.

Morin CM, Blais F, Savard J. 2001. Are changes in beliefs and attitudes about sleep related to sleep improvements in the treatment of insomnia? *Behav. Res. Ther.* In press

Morin CM, Colecchi C, Stone J, Sood R, Brink D. 1999a. Behavioral and pharmacological therapies for late-life insomnia: a randomised controlled trial. *JAMA* 281:991–99

Morin CM, Culbert JP, Schwartz MS. 1994. Non-pharmacological interventions for insomnia: a meta-analysis of treatment efficacy. *Am. J. Psychiatry* 151:1172–80

Morin CM, Hauri PJ, Espie CA, Spielman A, Buysse DJ, et al. 1999b. Nonpharmacologic treatment of insomnia: an American Academy of Sleep Medicine Review. *Sleep* 22:1134–56

Morin CM, Mimeault V, Gagne A. 1999c. Non-pharmacological treatment of late-life insomnia. *J. Psychosom. Res.* 46:103–16

Morin CM, Stone J, Trinkle D, Mercer J, Remsberg S. 1993. Dysfunctional beliefs and attitudes about sleep among older adults with and without insomnia complaints. *Psychol. Aging* 8:463–67

Morris M, Lack L, Dawson D. 1990. Sleep-onset insomniacs have delayed temperature rhythms. *Sleep* 13:1–14

Murtagh DR, Greenwood KM. 1995. Identifying effective psychological treatments for insomnia: a meta-analysis. *J. Consult. Clin. Psychol.* 63:79–89

Nicassio PM, Mendlowitz DR, Fussell JJ, Petras L. 1985. The phenomenology of the pre-sleep state: the development of the pre-sleep arousal scale. *Behav. Res. Ther.* 23:263–71

Ohayon MM, Caulet M, Guilleminault C. 1997. How a general population perceives its sleep and how this relates to the complaint of insomnia. *Sleep* 20:715–23

Perlis ML, Giles DE, Mendelson WB, Bootzin RR, Wyatt JK. 1997. Psychophysiological insomnia: the behavioural model and a neurocognitive perspective. *J. Sleep Res.* 6:179–88

Pressman MR, Orr WC. 1997. *Understanding Sleep. The Evaluation and Treatment of Sleep Disorders*. Washington, DC: Am. Psychol. Assoc. 566 pp.

Rachman S, De Silva P. 1978. Abnormal and normal obsessions. *Behav. Res. Ther.* 16: 233–48

Reite M, Buysse D, Reynolds C, Mendelson W. 1995. The use of polysomnography in the evaluation of insomnia. *Sleep* 18:58–70

Reynolds M, Salkovskis PM. 1992. Comparison of positive and negative intrusive thoughts and experimental investigation of the differential effects of mood. *Behav. Res. Ther.* 30:273–81

Rodin J, McAvay G, Timko C. 1988. A longitudinal study of depressed mood and sleep disturbances in elderly adults. *J. Gerontol.* 43:45–53

Roth T, Kramer M, Lutz T. 1976. The nature of insomnia: a descriptive summary of a sleep clinic population. *Compr. Psychiatry* 17:217–20

Sadeh A, Hauri PJ, Kripke DF, Lavie P. 1995.

The role of actigraphy in the evaluation of sleep disorders. *Sleep* 18:288–302

Sanavio E. 1988. Pre-sleep cognitive intrusions and treatment of onset-insomnia. *Behav. Res. Ther.* 26:451–59

Satlin A, Volicer L, Ross V, Herz L, Campbell S. 1992. Bright light treatment of behavioral and sleep disturbances in patients with Alzheimer's disease. *Am. J. Psychiatry* 149:1028–32

Schramm E, Hohagen F, Kappler C, Grasshoff E, Berger M. 1995. Mental comorbidity of chronic insomnia in general practice attenders using DSM-III-R. *Acta Psychiatr. Scand.* 91:10–17

Seidel WF, Ball S, Cohen S, Patterson N, Yost D, et al. 1984. Daytime alertness in relation to mood, performance, and nocturnal sleep in chronic insomniacs and noncomplaining sleepers. *Sleep* 7:230–38

Shiffrin RM, Schneider W. 1977. Controlled and automatic human information processing. II. Perceptual learning, automatic attending, and a general theory. *Psychol. Rev.* 84:127–90

Shocat T, Umphress J, Israel AG, Ancoli-Israel S. 1999. Insomnia in primary care patients. *Sleep* 22(Suppl. 2):S359–65

Shoham V, Bootzin RR, Rohrbaugh M, Urry H. 1995. Paradoxical versus relaxation treatment for insomnia: the moderating role of reactance. *Sleep Res.* 24A:365

Spielman AJ, Anderson MW. 1999. The clinical interview and treatment planning as a guide to understanding the nature of insomnia: the CCNY Interview for Insomnia. In *Sleep Disorders Medicine: Basic Science, Technical Considerations and Clinical Aspects*, ed. S Chokroverty, pp. 385–426. Boston: Butterworth-Heinemann. 2nd ed.

Spielman AJ, Glovinsky PB. 1991. The varied nature of insomnia. See Hauri 1991, pp. 1–15

Spielman AJ, Saskin P, Thorpy MJ. 1987. Treatment of chronic insomnia by restriction of time in bed. *Sleep* 10:45–56

Stepanski EJ, Glinn M, Fortier J, Sicklesteel J, Zorick FJ, et al. 1989. Physiological reactivity in chronic insomnia. *Sleep Res.* 18: 306

Tokarz T, Lawrence P. 1974. *An analysis of temporal and stimulus control factors in the treatment of insomnia*. Presented at Assoc. Adv. Behav. Ther., Chicago

Turner RM, Ascher LM. 1979. A controlled comparison of progressive relaxation, stimulus control and paradoxical intention therapies for insomnia. *J. Consult. Clin. Psychol.* 47:500–8

Van Egeren L, Haynes SN, Franzen M, Hamilton J. 1983. Presleep cognitions and attributions in sleep-onset insomnia. *J. Behav. Med.* 6:217–32

Walsh JK, Engelhardt CL. 1999. The direct economic costs of insomnia in the US for 1995. *Sleep* 22(Suppl. 2):386–93

Watts FN, Coyle K, East MP. 1994. The contribution of worry to insomnia. *Br. J. Clin. Psychol.* 33:211–20

Watts FN, East MP, Coyle K. 1995. Insomniacs' perceived lack of control over sleep. *Psychol. Health* 10:81–95

Webb WB. 1981. Sleep stage responses of older and younger subjects after sleep deprivation. *Electroencephalogr. Clin. Neurophysiol.* 52:368–71

Wegner DM. 1994. Ironic processes of mental control. *Psychol. Rev.* 101:34–52

Weissman MM, Greenwald MA, Nino-Murica G, Dement WC. 1997. The morbidity of insomnia uncomplicated by psychiatric disorders. *Gen. Hosp. Psychiatry* 19:245–50

Wicklow A, Espie CA. 2000. Intrusive thoughts and their relationship to actigraphic measurement of sleep: towards a cognitive model of insomnia. *Behav. Res. Ther.* 38:679–93

Wohlegemuth WK, Edinger JD. 2000. Sleep restriction therapy. See Lichstein & Morin 2000, pp. 147–66

Wolpe J. 1958. *Psychotherapy by Reciprocal Inhibition*. Stanford, CA: Stanford Univ. Press

Zwart CA, Lisman SA. 1979. Analysis of stimulus control treatment of sleep-onset insomnia. *J. Consult. Clin. Psychol.* 47:113–19

Annual Review of Psychology
Volume 53, 2002

CONTENTS

Journal of Abnormal Psychology
2005, Vol. 114, No. 2, 249–258

0021-843X/05/$12.00 DOI: 10.1037/0021-843X.114.2.249

Sleep-Related Attentional Bias in Good, Moderate, and Poor (Primary Insomnia) Sleepers

Barry T. Jones, Lauren M. Macphee, and Niall M. Broomfield
University of Glasgow

Benedict C. Jones
University of Aberdeen

Colin A. Espie
University of Glasgow

Evidence was sought of an attentional bias toward a highly representative object of the bedroom environment in good, moderate, and poor (primary insomnia) sleepers. Using a flicker paradigm for inducing change blindness, the authors briefly presented a single scene comprising a group of bedroom environment and neutral objects to participants and then briefly replaced this scene with an identical scene containing a change made to either a bedroom environment or a neutral object. In a 3×2 entirely between-participants design, change-detection latencies revealed a sleep-related attentional bias in poor sleepers but not in good sleepers. A possible bias in moderate sleepers was also revealed. It is suggested that attentional bias has a role in the perpetuation and possibly precipitation of primary insomnia.

Keywords: attentional bias, insomnia, charge-detection, sleep disruption, circadian

Sleep disruption is not unusual. Although good sleep usually returns, either through the disappearance of the precipitating agent or through the operation of mechanisms that appear to combat sleep disruption (the sleep homeostat or the circadian timer; Borbely, 1994), sleep disruption sometimes persists. When it persists for longer than a month (and is not a side effect of other medical, neurologic, psychiatric, or psychological disorders or the result of a circadian rhythm disorder), it is called *primary insomnia* (*Diagnostic and Statistical Manual of Mental Disorders*, 4th ed. [*DSM–IV*]; American Psychiatric Association, 1994). This report explores a possible role for *attentional bias* in the perpetuation and possibly the precipitation of primary insomnia. An attentional bias toward a particular class of stimulus (e.g., sleep-related) is said to have developed when disproportionate processing resources appear to be automatically allocated to exemplars, as compared with other otherwise equivalent stimuli—producing a disproportionate impact on current cognitions. This article is based, first, on a framework that has been helpful in addressing many other clinical disorders and, second, on an extension of this framework to encompass subclinical features of sleep disorder. This extension derives from research on attentional bias in relation to understanding alcohol consumption variability along the entire alcohol use–misuse–abuse–dependence continuum, not just at the clinical pole.

First, support has been found implicating attentional bias in the perpetuation of a wide range of anxiety-related psychological disorders and concerns (see Mogg & Bradley, 1998, for a review). Support has also been found implicating attentional bias in the perpetuation of a family of substance abuse and dependence disorders: for example, with alcohol (Sharma, Albery, & Cook, 2001), heroin (Franken, Kroon, Wiers, & Jansen, 2000) and nicotine (Waters & Feyerabend, 2000). Principally, through the use of the emotional Stroop paradigm (Williams, Mathews, & MacLeod, 1996), the extent to which attention is captured by disorder-related stimuli as compared with neutral stimuli has been revealed. Generally, but not exclusively, an attentional bias has been found in those who have a clinical diagnosis but absent in those who have not—this would be more completely termed a *differential* attentional bias. Finding such a differential attentional bias has helped explain why disorders (e.g., Mogg, Mathews, Bird, & MacGregor-Morris, 1990) and abuses and dependences (e.g., Drummond, Tiffany, Glautier, & Remington, 1995) are frequently self-maintaining and why relapse so frequently occurs after initially successful treatment. In explanations such as these, attentional bias is conceived as an involuntary (i.e., unconscious, implicit) process but (important to note) gives rise to voluntary (i.e., conscious, explicit) processes. These cognitions feature as part of the disorder

Editor's Note. Colin MacLeod served as the action editor for this article.—TBB

Barry T. Jones and Lauren M. Macphee, Department of Psychology, University of Glasgow, Glasgow, Scotland; Niall S. Broomfield and Colin A. Espie, Division of Community-Based Sciences in Psychological Medicine, University of Glasgow, Glasgow, Scotland; Benedict C. Jones, School of Psychology, Aberdeen University, Scotland.

This research and preparation of the article was supported in part by Grant CZH/4/2 from the Chief Scientist Office, Scottish Executive Health Department; the Carnegie Trust for the Universities of Scotland; and Glasgow University and St Andrews University graduate scholarships.

Correspondence concerning this article should be addressed to Barry T. Jones, Department of Psychology, University of Glasgow, Glasgow G12 8 QQ, Scotland, United Kingdom or Colin A. Espie, Division of Community-Based Sciences in Psychological Medicine, University of Glasgow, Academic Centre, Gartnavel Royal Hospital, 1055 Great Western Road, Glasgow G12 0XH, Scotland, United Kingdom. E-mail: barry@psy.gla.ac.uk or c.espie@clinmed.gla.ac.uk

and can contribute to subsequent behavior decisions. Franken, Kroon, and Hendriks (2000); Franken, Kroon, Wiers, and Jansen (2000); and Ingjaldsson, Thayer, and Laberg (2003), in particular, related attentional bias and craving with risk of relapse in substance abuse and dependence.

Williams et al.'s (1996) model of emotional Stroop performance (extending Cohen, Dunbar, & McClelland's, 1990, network model of the standard Stroop effect) accounts for attentional bias at the clinical pole of disorders, such as those mentioned above, through the increased responsivity of network input units representing disorder-related concepts—an increase caused by "a history of association with [anxiety-generating] threat or loss and thereby subject to neuromodulatory control affecting the responsivity of those units" (Williams et al., 1996, p. 17). As noted earlier, attentional biases, which reflect discrete changes in the direction of attentional focus in response to "threat," have been revealed in the anxiety disorders (e.g., Keogh, Dillon, Georgiou, & Hunt, 2001). We would argue that attentional bias may also be a strong candidate model for the perpetuation of primary insomnia. Sleep is a fundamental life process much like eating or drinking. Thus, inability to sleep can be conceptualized as a significant threat as well as an anxiety generator (Espie, 2002). We believe that attentional biases favoring sleep-related stimuli will, during an acute insomnia phase, lead to persistent insomnia in two ways: by promoting sleep preoccupation and by driving heightened sleep effort. This hypothesis is consistent with the reported association between insomnia and both excessive presleep cognitive activity (Wicklow & Espie, 2000) and effortful attempts to control sleep onset (Broomfield & Espie, 2003). Together, these two processes are likely to prevent successful cognitive and somatic de-arousal, which will inhibit the recovery of normal sleep (Espie, 2002). Harvey (2002) makes parallel predictions. In her cognitive model, worries regarding inability to sleep, and the consequent daytime impact of sleep loss, are thought to promote autonomic arousal and anxiety that in turn drive selective attention toward internal–external sleep-related threat cues. Again, it is proposed that sleep disturbance is maintained by an attentional bias favoring sleep-relevant cues.

Second, although differential attentional biases have generally been sought between those who have a diagnosed disorder and those who do not, efforts to understand alcohol consumption variability show that a systematically changing differential attentional bias toward alcohol-related stimuli might be present *along* the consumption continuum (of use, misuse, abuse, dependence), not just at its clinical pole. For example, a differential attentional bias has been found with (a) "alcoholic" drinkers (using "nonalcoholic" controls; Johnsen, Laberg, Cox, Vaksdal, & Hugdahl, 1994), (b) "problem" drinkers ("nonproblem" controls; Sharma et al., 2001), (c) "heavy" social drinkers ("light" social drinking controls; Bruce & Jones, 2005), and (d) "frequent social" drinkers ("occasional social" drinking controls; Townshend & Duka, 2001). Moreover, even at the clinical pole itself, Ryan (2002) and Jones, Bruce, Livingstone, and Reed (in press) have shown that the extent of attentional bias exhibited by problem drinkers in treatment is positively related to their problem severity. Applying Williams et al.'s (1996) model to the anxiety generated within the context of the negative expectations (or expectancies) of future consumption held by those at the clinical pole (B. T. Jones, 2004; B. T. Jones, Corbin, & Fromme, 2001) helps explain this differential bias. Moreover, it has been found in social drinkers, using explicit (e.g., Lee, Greeley, & Oei, 1999; McMahon, Jones, & O'Donnell, 1994) and implicit (Gadon, Bruce, McConnochie, & Jones, 2004; Gadon & Jones, 2002; Leigh & Stacy, 1998) methodologies, that the number, range, and severity of negative expectancies increase as consumption increases along the continuum from infrequent and lighter to frequent and heavier drinker—predicting a systematically changing attentional bias along the continuum. It is important to note that the progressive increase in negative expectancies would not necessarily restrain consumption (a macrobehavior) until a threshold was exceeded (B. T. Jones, 2004; B. T. Jones & McMahon, 1998) but that while subthreshold, it would progressively impact on microbehaviors such as emotional Stroop performance. While subthreshold, consumption would be driven by positive expectancies (e.g., Goldman, Del Boca, & Darkes, 1999) and because an attentional bias would prime positive expectancies, the apparent paradox is resolved that at levels of social drinking, increases in anxiety caused by increases in negative expectancies would increase consumption.

Little comparable research (either at the clinical pole or along the sleep-problems continuum) has been carried out on sleep-related attentional bias, which is surprising because interest in the control that sleep-related objects might have over sleep behavior is long established (Bootzin, 1972). For example, within a conditioning framework, bedroom environment objects might become discriminative stimuli for sleep (e.g., Bootzin, Epstein, & Wood, 1991), but when the bedroom–sleep contingencies are broken, they might become discriminative stimuli for wakefulness. Meta-analyses have suggested that treatments for insomnia that purport to modify the stimulus control the bedroom environment has over sleep are indeed effective (Morin et al., 1999; Murtagh & Greenwood, 1995; Smith et al., 2002)—to the extent that they are recommended by the American Academy of Sleep Medicine as the standard nonpharmacological intervention (Chesson et al., 1999). Yet, evaluations of the principles of stimulus control within the sleep environment have produced inconsistent data (see Espie, 2002, for a review), which raises the possibility that considerations of stimulus control extend beyond the conditioning framework and that treatments might have other active ingredients.

In an effort to more fully understand the impact that the bedroom environment might have on sleep, this article goes beyond the conditioning framework described above (which addresses one form of stimulus control) to a framework of attentional bias (which addresses another). Two previous studies have explored sleep-related attentional bias using a textual Stroop paradigm. In one, Lundh, Froding, Gyllenhammer, Broman, and Hetta (1997) found that individuals with primary insomnia showed no effect. In the other, Taylor et al. (2003) found an attentional bias in cancer patients with persistent insomnia (12–18 months) as compared with cancer patients with acute insomnia (0–3 months). In the former study, however, only one fourth sleep-related words represented bedroom objects and, in the latter, only 3/20; consequently, although informative, neither study addressed the possible influence of the bedroom environment on sleep.

Rather than use the textual Stroop paradigm, we explore sleep-related attentional bias with digitized objects using a flicker paradigm (Rensink, O'Regan, & Clark, 1997; Simons & Levin, 1997) featuring a perceptual phenomenon called *induced change blindness* (ICB; Rensink, 2002; Simons, 2000; Simons & Rensink,

2005). This research reveals that when a change is made to a visual scene (and the process of change is hidden from view), it is more difficult to detect than might be expected. Normally in this paradigm, a single feature of a visual scene is changed between successively repeated brief presentations until the change is detected. Change-detection latency is explained by a change's "grabbiness" (O'Regan & Noe, 2000), and this depends not just on the object's physical features that carry the change but also on the viewer's *history* in relation to that object. Using this technique, researchers (B. C. Jones, Jones, Blundell, & Bruce, 2002; B. T. Jones, Jones, Smith, & Copely, 2003) have found differential attentional biases between two levels of social use of alcohol and cannabis. Here, we extend their approach to explore differential attentional biases along the sleep-problems continuum.

We postulate that a systematically changing attentional bias will be found (at different points of the sleep-problems continuum) to exist toward changes made to bedroom environment objects as compared with changes made to other objects. We predict it will be greatest in poorest sleepers (having primary insomnia diagnosed), reduced in moderate sleepers (not having a clinical diagnosis but having more sleep problems than good sleepers), and absent in good sleepers (having no or few sleep problems). We propose that (a) anxiety is a mediator of sleep-related attentional bias at the clinical pole (Espie, 2002; Harvey, 2002), just as with alcohol-related attentional bias at the clinical pole of the consumption continuum, and (b) that different subclinical points of the sleep-problems continuum will generate different levels of anxiety, just as with subclinical levels of alcohol problems. Through Williams et al.'s (1996) model, we predict an increasing sleep-related attentional bias toward bedroom environment objects from good through moderate to poor sleepers. We use a highly representative set of bedroom objects and test this hypothesis with a change carried by the one most representative.

Method

Participants

Students, staff, and visitors on the campus of a large city university were asked to take part in a 15-min experiment in a nearby quiet room. Each of the opportunistically recruited 302 volunteers were tested singly. Using the procedures described below, we selected 192 for analysis (M age = 32.1 years, SD = 12.6).

Measures

Each participant completed a computer task that measured his or her visual change-detection latency (the dependent variable) to changes in photographic scenes described below. They then completed three questionnaires (used for participant exclusion and retrospective group assignment with age-matching). First, the Beck Depression Inventory (BDI; Beck & Steer. 1987) was given, followed by the Pittsburgh Sleep Quality Index (PSQI; Buysse, Reynolds, Monk, Berman, & Kupfer, 1989). The PSQI provides a global measure of sleep quality by aggregating the scores on a 0–3 subscale (3 is the negative pole) of seven different areas of sleep: subjective sleep quality, sleep latency, sleep duration, habitual sleep efficiency, sleep disturbances, use of sleep medication, and daytime dysfunction. The PSQI is a widely used, internally consistent (Cronbach's alpha = .83) screening instrument for the detection of significant sleep disturbance (using a threshold score of 6). Recent, independent study has validated this cut-off and confirmed reliability (Cronbach's alpha = .85, test–retest r = .84; Backhaus, Junghanns, Broocks, Riemann, & Hohagen, 2002). Finally, a local questionnaire was given that collected information on age, gender, and whether individuals had suffered from any psychopathology or other illness known to affect sleep or had any sleep complaint.

The global PSQI score has been shown to be effective in discriminating between nonclinical (0–5) and clinical (6–21) sleepers (e.g., Backhaus et al., 2002), and we use it for this purpose. In addition, however, we seek to explore the possible continuity of attentional bias along the sleep-problems continuum by using the PSQI's continuous measure of sleep problems to further classify nonclinical sleepers using the end regions (0–2 and 4–5) of the nonclinical PSQI range (0–5). We identify participants as "good" and "moderate" sleepers, respectively, discriminating them from "poor" sleepers and thereby creating three points on the sleep-problems continuum that can be compared. We acknowledge that our concept of moderate sleeper has, perhaps, more limitations than good sleeper, but we justify inclusion of this group for two reasons. First, epidemiological data clearly demonstrate that prevalence of insomnia varies depending on the question asked. Whereas 25–30% of the adult population report dissatisfaction with their sleep, the true prevalence of the clinical disorder is around half that rate, 10–15% (Ford & Kamerow, 1989; Gallup Organization, 1991; Oyahon, Caulet, & Guilleminault, 1997). Second, because (a) there were clear criteria for establishing a poor sleeper category, (b) the distance between the poor sleeper and good sleeper category was substantial, and (c) we wished to test the hypothesis that there would be systematically modified attentional bias across the sleep-problems continuum, we felt that the conceptual delineation of a middle category would be appropriate.

Design

The hypothesis tested was that participants with PSQI-indicated sleep problems will detect a change made to one of a collection of sleep-related objects quicker than a change made to one of a collection of neutral objects, as compared with participants with fewer PSQI-indicated sleep problems. An entirely between-participants design was adopted with the following three factors: nature of change to be detected (two levels: sleep-related and neutral), gender (two levels: male and female), and PSQI-indicated sleep quality (three levels: good, moderate, and poor). The dependent variable was change-detection latency.

Sleep problems approximately double from adulthood to later life, and women present about twice as often as men (e.g., Espie, 2002; Ford & Kamerow, 1989; Li, Wing, Ho, & Fong, 2002). Accordingly, groups were matched for age (on an individual basis rather than a group-mean basis), and gender was included as a factor in the design.

A target of approximately 300 participants was estimated to be necessary to allow for the application of the exclusion and matching procedures described below. Each participant was given the flicker ICB change-detection task followed by the BDI, the PSQI, and a local questionnaire. Once recruited into the experiment, participants were randomly assigned to one of four groups generated by crossing the factors of nature of change and gender. Group sizes of 75 or 76 were planned. For each of the four groups, participants who (a) self-reported an illness known to affect sleep (i.e., 2), (b) scored above 9 on the BDI (10–18 indicates moderate-mild depression [Beck & Steer, 1987]; no participants were found to be in this category), or (c) failed to satisfy the task requirements (i.e., to correctly identify the change to which they were exposed—9 participants) were excluded from the analyses. For analyses purposes, the remaining participants of each of the four groups were retrospectively assigned to one of the three levels of the factor of sleep quality, on the basis of their PSQI scores: good sleepers (PSQI score 0–2 inclusive), moderate sleepers (4–5 inclusive), and poor sleepers (> 5). Assignment to the newly formed 12 groups ($2 \times 2 \times 3$) was constrained by matching for age (to within 4 years) and the goal of equal group sizes (to obtain the best estimate of between-groups variation). Retrospective group assignment was blind to the dependent variable of the analyses: change-detection latency. Unmatched participants

were excluded from the analyses. Analyses were carried out on the 192 remaining participants, with a group size of 16.

Apparatus and Stimuli

An Apple iBook (MacOS 9.1) and the experiment-generation package SuperLab 1.75 (Cedrus Corporation, San Pedro, CA) were used to implement the flicker paradigm. Photographic stimuli were presented centrally and almost filled the iBook's 28-cm (diagonal) screen. The viewing distance was approximately 65 cm normal to the screen.

A different flicker pair of stimuli were used for each of the two levels of the nature of change factor (sleep-related and neutral). Each of the two pairs contained the same original stimulus (OS) shown in Figure 1. The OS was a full-color photograph (1280 × 960 pixels) taken in natural daylight of seven sleep-related objects and an equal number of neutral objects arranged in two collections on either side of the scene midline. The second stimulus of each pair was identical to the OS but for one small change: a sleep-related change was introduced for the "sleep" level of the nature of change factor (stimulus conditioned stimulus [CS-S]) and a neutral change for the "neutral" level of the same factor (stimulus CS-N). The two changed stimuli are shown in Figure 1 along with their common originating stimulus. Within a flicker paradigm, the two stimuli of a pair (OS/CS-S for the six sleep change groups and OS/CS-N for the six neutral change groups) are presented in continuous succession (each replacing the other, in register, on the iBook screen) until the change from the original to the changed stimulus (or from the changed stimulus to the original stimulus) is detected (see Figure 2). In the current experiment, following usual practice to suppress visual transients produced by the change process, a mask comprising a rectilinear matrix of Xs was presented on the screen between the OS and the changed versions (CS-S or CS-N). The OS and either the CS-S or the CS-N were presented for 250 ms, and the mask was presented for 80 ms with no other interstimulus interval and no intertrial interval (B. C. Jones et al., 2002; B. T. Jones et al., 2003).

Precautions were taken to ensure that the seven sleep-related objects included in the OS were highly representative of the sleep environment and that the object carrying the sole sleep-related change to be detected throughout the experiment was the most representative of the seven. This

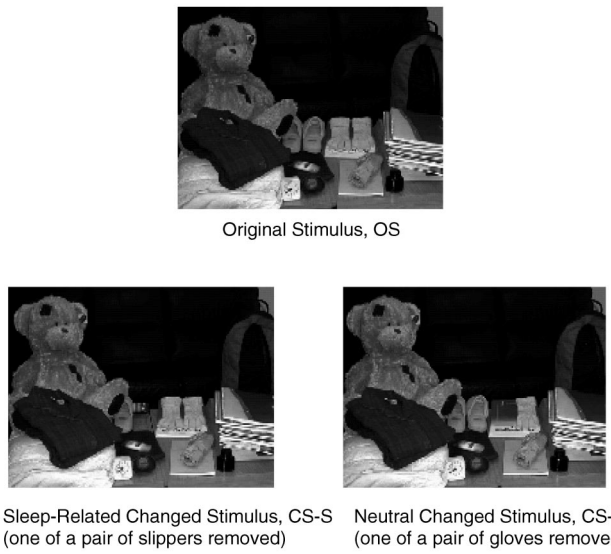


Figure 1. Gray-scale versions of the full color stimuli used: original stimulus (OS) and the two changed stimuli for each of the two levels of the nature of change factor—sleep-related change (CS-S) and neutral change (CS-N).

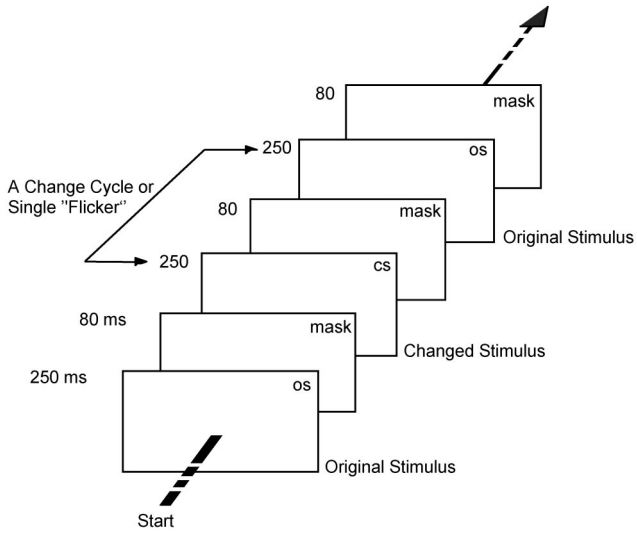


Figure 2. A flicker paradigm for inducing change blindness illustrating a change cycle or single "flicker." os = original stimulus; cs = changed stimulus.

was done in three stages. First, by asking 60 students (50% female) to list at least 5 objects that they would associate with "going to bed to sleep." Their lists were compiled to identify the 12 most listed objects. Second, examples of these objects were photographed singly, and the set of 12 objects were embedded in another set of 12 photographs of neutral objects (thought not to be bedroom and sleep-related by the experimenters and none of which had appeared in the 60 lists of sleep-related objects collected earlier). This created a 4 × 6 matrix of photographs of 12 sleep-related and 12 neutral objects. Finally, another 30 students (50% female) were asked to rate the 24 photographs of the objects on a 1–10 "sleep-relatedness" scale (1 = *highly sleep-related*, 10 = *not sleep-related at all*). The 7 objects with the highest mean sleep-relatedness score (mean range = 1.2–5.8) were selected for the sleep-related collection of objects in the OS, and the 7 with the lowest mean score (all had means of 10) were selected for the neutral collection. The sleep-related objects were teddy bear, pajamas, pillow, alarm clock, hot water bottle, hand cream, and pair of slippers. The pair of slippers had the highest sleep-related score. The neutral objects were rucksack, journal issue, files, bottle of ink, office tray, telescoped umbrella, and pair of gloves. The CS-S was made by removing one of the pair of slippers (the most representative sleep-related object) from the OS and photographing the remaining scene, and the CS-N was made by removing one of the pair of gloves from the OS. (In the absence of differential sleep-relatedness scores in this group, this object was chosen because it was physically most similar to the object carrying the sleep-related change.)

In earlier applications of the flicker paradigm to explore attentional biases in the social use of alcohol and cannabis, B. C. Jones et al. (2002) and B. T. Jones et al. (2003) used both normal and mirror-reversal versions of the original and changed stimuli as a between factor. This was done to control for possible carry-over effects from, for example, lifelong reading practices generating side preferences in visual processing. In the four studies they reported, however, B. C. Jones et al. (2002, B. T. Jones et al., 2003) found no significant main effects for or interactive effects implicating the factor of normal/mirror-reversal. For this reason, mirror-reversals of stimuli OS and CS-S/CS-N were not included in the current experiment.

Procedure

Potential participants were approached throughout the campus and asked to take part in an experiment. Care was taken during the recruitment period

to ensure that the sleep-related nature of the experiment was not apparent. Those who agreed to take part were taken to a nearby quiet room and asked to read the instructions on an iBook screen on what they should expect. Participants were then asked to press the space bar when they were ready, at which time they would see a visual scene presented on the screen for less than a second. It would keep appearing and disappearing for as long as it took them to spot a small change made to the scene between successive presentations. When they had spotted the change, participants were asked to immediately press the space bar, which would be recorded by the computer, and then to say what the change was. Participants were then asked to complete the BDI, the PSQI, and the local questionnaire. It would only have been through reading the contents of the PSQI and the local questionnaire (given after the flicker paradigm) that participants would have been explicitly exposed to the sleep-related nature of the experiment. Once the questionnaires had been completed and passed to the experimenter, the true purpose of the experiment was explained. Participants were also provided with contact information in case they wanted to be informed of the outcome of the project when it was completed.

Results

As indicated earlier, age and BDI-indicated depression associates with sleep problems. Consequently, two analyses of variance (ANOVAs) were carried out prior to the main analyses. A matching-check for age across the 12 groups was carried out using an ANOVA that was identically structured to the main analysis, that is, a three-factor between-participants ANOVA ($N = 192$, $n = 16$) but with age as the dependent variable and with the following factors and levels: nature of change (sleep-related, neutral), gender (male, female), and sleep quality (poor, moderate, good). With age as the dependent variable, there were no main, two-way, or three-way interactive effects. This indicates a good degree of equivalence in age between the 12 different groups. A second identical ANOVA was carried out with BDI scores as the dependent variable; this analysis also showed no main or interactive effects (overall BDI score: $M = 3.1$, $SD = 2.1$), indicating a corresponding equivalence.

Analysis 1: ANOVA

The number of flickers to correct change detection for each participant was entered into a three-factor between-participants ANOVA (structured as above; $N = 192$, $n = 16$). There were no significant main effects. Sleep-related change-detection latencies were no different from neutral change-detection latencies (Ms = 17.7 and 19.0, respectively; SDs = 12.3 and 11.1, respectively), $F(1, 180) = 0.51$, $p > .05$; womens' change-detection latencies were no different from mens' (Ms = 18.2 and 18.5; SDs = 7.9 and 9.4, respectively), $F(1, 180) = 0.07$, $p > .05$; and there were no differences between the change-detection latencies of poor, moderate, and good sleepers (Ms = 18.4, 16.9, and 19.8, respectively; SDs = 7.8, 7.1, and 9.7, respectively), $F(2, 180) = 1.35$, $p > .05$. Of the one 3-way and three 2-way interactions, only the Change × Sleep Quality interaction was significant, $F(2, 180) = 7.12$, $p < .01$ (see Figure 3). The interaction was interpreted using tests for simple main effects. This was done in two ways.

First, for the neutral change, no differences in change-detection latency were found between the poor, moderate, and good sleepers (Ms = 21.9, 18.4, and 16.5, respectively; SDs = 12.1, 10.9, and 6.8, respectively), $F(2, 180) = 1.69$, $p > .05$. By contrast, differences in change-detection latency were found between poor, moderate, and good sleepers for the sleep-related change (Ms = 14.5, 15.5, and 23.1, respectively; SDs = 9.4, 10.7, and 7.6, respectively), $F(2, 180) = 6.59$, $p < .01$. These differences were located using t tests. It was found that poor sleepers detected the sleep-related change significantly more quickly than good sleepers ($M = 14.5$ vs. $M = 23.1$; SDs = 8.5 vs. 7.6), $t(191) = 3.33$, $p < .01$, and moderate sleepers detected it significantly more quickly than good sleepers ($M = 15.5$ vs. $M = 23.1$, SDs = 9.1 vs. 10.1), $t(191) = 2.90$, $p < .01$. There was no difference in the change-detection latency for the sleep-related change between poor and moderate sleepers, however ($M = 14.5$ and $M = 15.5$, respectively; SDs = 9.4 and 10.7, respectively), $t(191) = 0.34$, $p > .05$.

Second, poor sleepers detected the sleep-related change significantly more quickly than the neutral change (Ms = 14.5 and 21.9, respectively; SDs = 9.4 and 12.1, respectively), $F(1, 180) = 7.11$, $p < .01$, displaying a sleep-related attentional bias. Although moderate sleepers showed the same difference direction as poor sleepers, the difference between sleep-related and neutral change detections was not significant (Ms = 15.5 and 18.4, respectively; SDs = 10.7 and 10.9, respectively), $F(1, 180) = 1.29$, $p > .05$. By contrast, good sleepers detected the change within the neutral objects significantly more quickly than within the sleep-related objects (Ms = 16.5 and 23.1, respectively; SDs = 6.8 and 7.6, respectively), $F(1, 180) = 6.21$, $p < .05$, showing a bias toward neutral rather than sleep-related objects. The effect size for poor sleepers (see Figure 4) was significant and "moderate" using Cohen's (1992) classificatory scheme for effect size comparisons (Cohen's $d = 0.60$; 95% confidence limits of 0.07 and 1.06) as compared with a "small" and nonsignificant effect size for moderate sleepers ($d = 0.21$; 95% confidence limits of −0.17 and 0.51). The effect size of the opposite bias shown by the good sleepers was significant and "large" (Cohen's $d = -0.91$; 95% confidence limits of −0.41 and −1.46).

The foregoing is consistent with poor, moderate, and good sleepers, together responding equivalently to the neutral change but responding differently to the sleep-related change. Further, no difference between the neutral and sleep-related change is found for the moderate sleepers but for the poor sleepers, there is a decrease in change-detection latency whereas for good sleepers, there is an increase.

Analysis 2: Regression

Two hierarchical regression analyses were carried out for the two different levels of the nature of change factor (sleep-related and neutral) in order to test the relationship between change-detection latency and a continuous representation of the global PSQI score (rather than a categorical representation, as was used in the ANOVA). With change-detection latency as the dependent variable, age, gender (for reasons outlined earlier), and the BDI score were first added to the regression model and then the PSQI score. Although BDI scores above 9 represent the severity of a clinical diagnosis associated with increasingly poor sleep, this does not necessarily mean that the BDI score range 0–9 can be used to represent states increasingly proximate to a clinical diagnosis (and with commensurate associations with sleep quality). Nevertheless, entering the "subclinical" BDI score into the regression model prior to the PSQI score appears to be a sensible precaution. The incremental variance explained when the PSQI score is added to

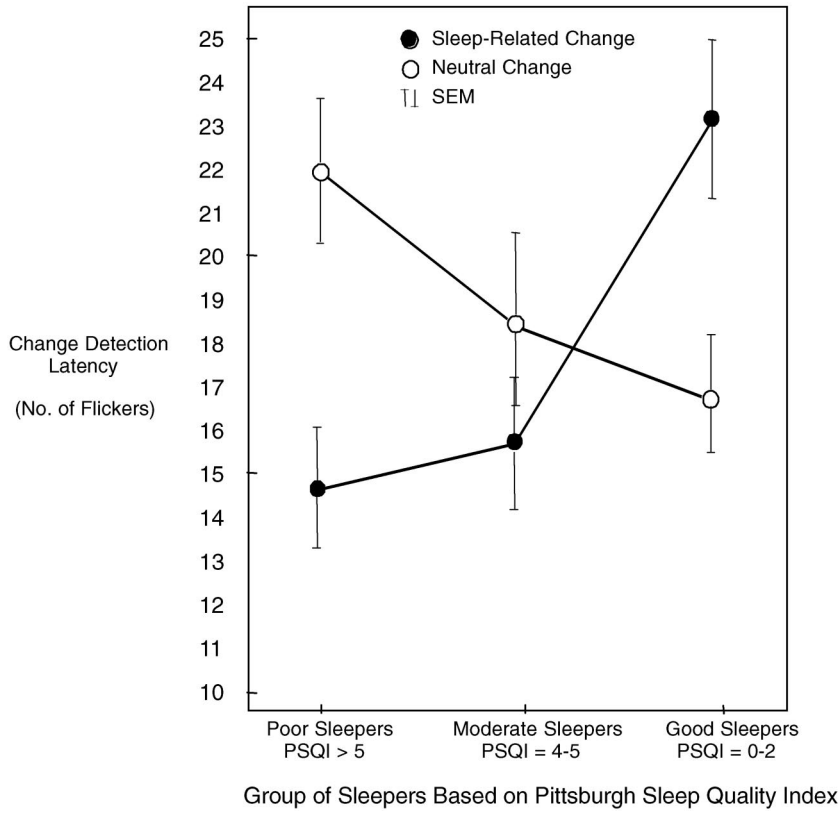


Figure 3. Mean sleep-related and neutral change-detection latencies (number of flickers to detection) in poor, moderate, and good sleepers. PSQI = Pittsburgh Sleep Quality Index; SEM = standard error of the mean.

the model already containing age, gender, and the BDI score is the critical test of the relationship.

In both models (sleep-related change and the neutral change), there was no significant variance explained in change-detection latency when age, gender, and the BDI scores were added. When the PSQI score was added to the model for the sleep-related change, however, the model deviated from the null, $F(4, 92) = 2.51$, $p < .05$. The incremental variance explained on the basis of adjusted R^2 was 10.6% ($p < .05$), and the beta for PSQI was -0.310 ($p < .05$). For the neutral change, the model did not deviate from the null when the PSQI score was added, $F(4, 92) = 2.29$, $p > .05$.

Thus, there is no relationship between PSQI-indicated sleep problems and change-detection latency for the neutral change, but for the sleep-related change, change-detection latency decreases as sleep problems increase.

Discussion

Espie's (2002) and Harvey's (2002) models of primary insomnia identify attentional bias toward sleep-related concepts as contributing to the perpetuation of primary insomnia. In both models, the heightened arousal accompanying insomnia and the belief that the night's sleep problems will negatively impact on next day's activities generate anxiety. Williams et al. (1996) postulated that anxiety raises the resting activity level of network inputs representing concepts with which the anxiety temporally occurs. The raised resting activity level is the basis of the attentional bias we predict will be found for bedroom objects in primary insomnia. This means that representations of the bedroom environment will be more likely to enter current cognitions, generate anxiety, and disrupt sleep.

Our results support this prediction, showing an attentional bias toward a representative bedroom object in sleepers who satisfy the minimal criteria for primary insomnia (our poor sleepers); change-detection latencies in poor sleepers were quicker when the change was made to the sleep-related object than to the neutral object. There was also found a marked differential attentional bias between poor sleepers and the good-sleeping controls. However, whereas the poor sleepers showed a bias toward the sleep-related object as predicted, the good sleepers showed a bias toward the neutral object. This part of our prediction, therefore, does not appear to be supported—namely, that because good sleepers would be reporting few if any sleep problems, there would be little anxiety generated within the sleep-problems context, negligible changes in the resting activity levels of the sleep-related network input units, and therefore a negligible sleep-related attentional bias. There are two explanations why the good-sleeping component of the differential attentional bias might occur.

First, it is possible that the good sleepers' choices are driven by the physical properties (physical saliencies) of the sleep-related and neutral objects themselves, rather than semantic saliencies. Particularly in complex stimuli (e.g., real-world objects collected

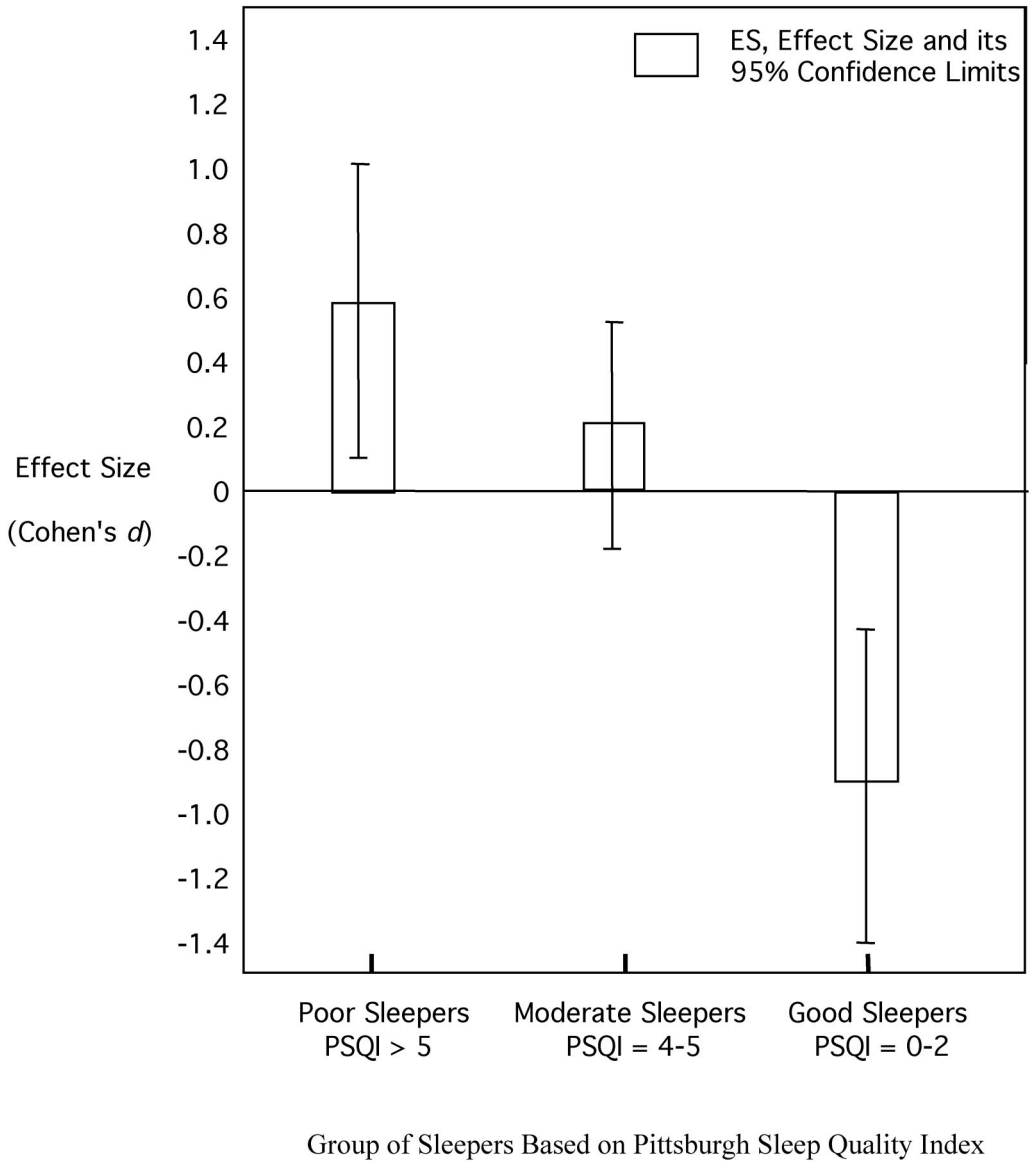


Figure 4. Effect size (Cohen's *d*) and 95% confidence limits of net attentional bias in poor, moderate, and good sleepers (net attentional bias calculated as sleep-related minus neutral change-detection latencies). PSQI = Pittsburgh Sleep Quality Index.

into a scene), values of the objects' physical properties will compete for attention resources and contribute to, or even drive, what is attended to and what reaches consciousness. Physical properties such as object size, color, brightness, complexity, and overlap as well as relative positional and configurational aspects will be important in this respect. This means that any single attentional bias measure (e.g., the comparison between the change-detection latencies of the group of good sleepers given the sleep-related change with the other group of good sleepers given the neutral changes) is ambiguous because it confounds the sleep salience manipulation (i.e., the sleep-related change vs. the neutral change) with a range of other nonsleep saliencies. If the good sleepers have comparatively problem-free sleep (a group assignment feature of the design), there will be little associated anxiety and, consequently, negligible sleep-related attentional bias. In addition, in such a case, any attentional bias would be driven by the physical saliencies of the grouped objects. We would conclude, therefore, that either the group of neutral objects in which the neutral change was embedded, or the neutral objects that carry the neutral change had more "grabby" physical properties than did the corresponding sleep-related objects; this would explain the neutral attentional bias shown by the good sleepers. When measuring differential attentional bias, however (i.e., the difference in attentional bias between poor sleepers and good sleepers), we are able to control the physical saliencies (and any other nonsleep semantic saliencies) through giving the poor and good sleeping groups the *same*

stimulus presentations. The interaction representing the differential attentional bias between poor and good sleepers is, therefore, the properly controlled measure of the effect of the PSQI-derived sleep-problem manipulation on sleep-related attentional bias. Indeed, it follows from this explanation that the apparent sleep-related attentional bias shown by the poor sleepers will be an underestimate of the real sleep-related attentional bias and will only be provided through the measure of differential attentional bias.

Second, there is an alternative explanation for the neutral bias found in good sleepers that makes no reference to physical saliencies. It is possible that our good sleepers were low in anxiety-sensitivity (Reiss, 1997), given that, using a dot probe paradigm, Keogh et al. (2001) have shown that participants with low anxiety-sensitivity under some circumstances orient away from threatening stimuli rather than toward them—in which case, our good sleepers should orient away from the set of bedroom objects if they were low in anxiety-sensitivity. If this occurred, it would assist neutral change detection but hinder sleep-related change detection and explain the bias toward the neutral set of stimuli that good sleepers show. Although there might be grounds for suggesting that good sleepers might indeed be low in trait anxiety (because this disposition might either contribute to or derive from their good-sleeping status), there is less to suggest that they might be low in anxiety-sensitivity and, as Keogh et al. noted, it is the latter rather than the former that would be an important feature of this type of explanation. Nevertheless, it remains a possibility.

However the differential attentional bias between two poles of the sleep-problems continuum is explained, it remains that the poor sleepers appear to attend to a sleep-related object more than a neutral object. If our finding with one sleep object generalizes to others, the attentional bias that it represents will feed the processes that maintain or perpetuate primary insomnia described by Espie (2002) and Harvey (2002). There is some indication that attentional bias might be an agent for not just the perpetuation of primary insomnia but also its precipitation—this possibility is raised through a consideration of the moderate sleepers' performance. On the surface, the moderate sleepers do not appear to show an attentional bias toward the sleep-related object as compared with the neutral object. If, however, the explanation of the performance of the good sleepers is indeed based on physical saliencies (as described above), then the good-sleepers performance represents a baseline condition against which the performance of the moderate sleepers can be judged, with all nonsleep saliencies controlled. When this comparison is made, good and moderate sleepers do not treat the neutral change differently (and neither do poor sleepers); by critical contrast, however, the moderate sleepers detect the sleep-related change significantly more quickly than do the good sleepers. The apparent lack of attentional bias in moderate sleepers, derived from comparing only their detection latencies to the sleep-related and neutral change, should not obscure the fact that a differential attentional bias is revealed from good to moderate sleepers when more appropriately controlled comparisons are made. If moderate sleepers do indeed exhibit a sleep-related attentional bias—generated by the anxiety accompanying *acute* sleep problems, not diagnosed as the more *chronic* primary insomnia—it could help maintain these sleep problems in much the same way as Espie (2002) and Harvey (2002) suggested that attentional bias maintains primary insomnia. If sleep problems are maintained beyond an acute phase by attentional bias, however, and if the length of time satisfies the criterion for primary insomnia, it suggests that attentional bias might not only be implicated in perpetuating primary insomnia but also in precipitating it.

Our study is the first to report a sleep-related differential attentional bias between good and poor sleepers and a possible corresponding differential bias between good and moderate sleepers. It also identifies a likely source perpetuating primary insomnia and a possible precipitating factor. We find only partial support for a continuity of sleep-related attentional bias corresponding to the sleep-problems' continuum, however, because of the failure to find a differential attentional bias between the moderate and poor sleepers.

Our findings and conclusions are limited by a number of considerations. First, the clinical pole (primary insomnia) was represented by participants with PSQI scores greater than 5. The incorporation in future research of an additional "primary insomnia" group with a higher inclusion threshold would provide an additional opportunity to compare more distant clinical and subclinical points on the sleep-problems continuum as well as points within the diagnostic category—providing a more extensive test of the hypothesis of continuity of attentional bias. Second, only a single sleep-related change and neutral change were used, and this might compromise generalizing the findings and conclusions to other bedroom and neutral objects. Care was taken, however, to ensure that all the objects used were either highly representative of the bedroom environment or not representative at all. Further, the most representative bedroom object was chosen to carry the sleep change. Finally, Bruce and Jones (2005) have recently shown with social drinkers that change detection performance in a flicker paradigm is almost entirely driven by the *set* of different objects in which the single object carrying the change (also a set member) is embedded rather than by the single object itself. If this finding extends to our sleep study (which uses similar stimulus configurations), this limitation is reduced because seven sleep objects make up the sleep set and another seven make up the neutral set. Nevertheless, our finding requires replication, and future research should extend the range of objects used. Furthermore, we do not have anxiety and anxiety-sensitivity data, and the possibility that good sleepers low in anxiety sensitivity might be orienting away from the sleep-related objects cannot be evaluated. Future research needs to pay attention to this potential explanatory source by collecting anxiety data.

Although it is some 30 years since stimulus control was proposed as offering an explanatory, and potentially modifiable, paradigm in insomnia (Bootzin, 1972), contemporary sources agree that its role in the development and amelioration of persistent insomnia remains unclear (Edinger & Wohlgemuth, 1999; Espie, 2002; Morin et al., 1999). In large part, this is because research effort has focused on treatment efficacy derived from stimulus control intervention rather than on experimental studies. We are now in the position where a psychological rather than pharmacological approach is seen as the treatment of choice for persistent primary insomnia (Espie, 1999; Smith et al., 2002) and stimulus control instructions meet American Academy of Sleep Medicine criteria for "standard treatment" (Chesson et al., 1999). The present study, which uses a novel experimental approach well suited to the systematic investigation of the stimulus control par-

adigm, illustrates one way forward for research in this area. Evidence presented of attentional bias toward the bedroom environment, and our previously reported evidence of attentional bias toward textual representations of sleep and sleeplessness (Taylor et al., 2003), raises the possibility that dysfunctional stimulus control in insomnia develops from the conditioning of nonverbal and verbal signals as threat cues that impact on selective attention through the principles of Williams et al.'s (1996) model—starting a vicious cycle. The feasibility of objectively and accurately measuring this bias, therefore, yields new opportunity for both experimental and clinical investigations of the development of primary insomnia during remission and treatment response.

References

American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: Author.

Backhaus, J., Junghanns, K., Broocks, A., Riemann, D., & Hohagen, F. (2002). Test-retest reliability of the Pittsburgh Sleep Quality Index in primary insomnia. *Journal of Psychosomatic Research, 53,* 737–740.

Beck, A. T., & Steer, R. A. (1987). *Beck Depression Inventory (BDI) manual.* San Antonio, TX: The Psychological Corporation.

Bootzin, R. R. (1972). A stimulus control treatment for insomnia. *Proceedings of the American Psychological Association,* 395–396.

Bootzin, R. R., Epstein, D., & Wood, J. M. (1991). Stimulus control instructions. In P. Hauri (Ed.), *Case studies in insomnia* (pp. 19–28). New York: Plenum Press.

Borbely, A. A. (1994). Sleep homeostasis and models of sleep regulation. In M. H. Kryger, T. Roth, & W. C. Dement (Eds.), *Principles and practice of sleep medicine* (3rd ed., pp. 309–320). Philadelphia: Saunders.

Broomfield, N. M., & Espie, C. A. (2003). Initial insomnia and paradoxical intention: An experimental investigation of putative mechanisms using subjective and actigraphic measurement of sleep. *Behavioural & Cognitive Psychotherapy, 31,* 313–324.

Bruce, G., & Jones, B. T. (2004). A pictorial Stroop paradigm reveals an alcohol attentional bias in heavier as compared with lighter social drinkers. *Journal of Psychopharmacology, 18,* 527–533.

Bruce, G., & Jones, B. T. (2005, June). Alcohol-related attentional bias in social drinkers. Paper presented at the 28th Annual Scientific Meeting of the Research Society on Alcoholism, Santa Barbara, CA.

Buysse, D. J., Reynolds, C. F., Monk, T. H., Berman, S. R., & Kupfer, D. J. (1989). The Pittsburgh Sleep Quality Index: A new instrument for psychiatric practice and research. *Psychiatric Research, 28,* 193–213.

Chesson, A. L., Anderson, W. M., Littner, M., Davilia, D., Hartse, K., Johnson, S., et al. (1999). Practice parameters for non-pharmacologic treatment of chronic insomnia. *Sleep, 22,* 1128–1133.

Cohen, J. (1992). A power primer. *Psychological Bulletin, 112,* 155–159.

Cohen, J. D., Dunbar, K., & McClelland, J. L. (1990). On the control of automatic processes: A parallel distributed processing account of the Stroop effect. *Psychological Review, 97,* 181–200.

Drummond, D. C., Tiffany, S. T., Glautier, S., & Remington, B. (1995). *Addictive behaviour: Cue exposure theory and practice.* New York: Wiley.

Edinger, J. D., & Wohlgemuth, W. (1999). The significance and management of persistent primary insomnia: The past, present and future of behavioral insomnia therapies. *Sleep Medicine Reviews, 3,* 101–118.

Espie, C. A. (1999). Cognitive behavioral therapy as the treatment of choice for primary insomnia. *Sleep Medicine Reviews, 3,* 97–99.

Espie, C. A. (2002). Insomnia: Conceptual issues in the development, persistence, and treatment of sleep disorders in adults. *Annual Review of Psychology, 53,* 215–243.

Ford, D. E., & Kamerow, D. B. (1989). Epidemiologic study of sleep disturbances and psychiatric disorders. *Journal of the American Medical Association, 262,* 1479–1484.

Franken, I. H. A., Kroon, L. Y., & Hendriks, V. M. (2000). Influence of individual differences in craving and obsessive cocaine thoughts on attentional processes in cocaine abuse patients. *Addictive Behaviors, 25,* 99–102.

Franken, I. H. A., Kroon, L. Y., Wiers, R. W., & Jansen, A. (2000). Selective cognitive processing of drug cues in heroin dependence. *Journal of Psychopharmacology, 14,* 395–400.

Gadon, L., Bruce, G., McConnochie, F., & Jones, B. T. (2004). Negative alcohol consumption outcome associations in young and mature adult social drinkers: The route to drinking restraint? *Addictive Behaviors, 29,* 1373–1387.

Gadon, L., & Jones, B. T. (2002). Implicit alcohol associations increase in number as time goes by—Though somewhat unevenly. *Alcoholism: Clinical and Experimental Research, 26,* 34A.

Gallup Organization. (1991). *Sleep in America.* Princeton, NJ: Author.

Goldman, M. S., Del Boca, F. K., & Darkes, J. (1999). Alcohol expectancy theory: The application of cognitive neuroscience. In K. E. Leonard & Howard T. Blane (Eds.), *Psychological theories of drinking and alcoholism* (2nd ed., pp. 203–246). New York: Guilford Press.

Harvey, A. G. (2002). A cognitive model of insomnia. *Behaviour Research and Therapy, 40,* 869–893.

Ingjaldsson, J. T., Thayer, J. F., & Laberg, J. C. (2003). Craving for alcohol and pre-attentive processing of alcohol stimuli. *International Journal of Psychophysiology, 49,* 29–39.

Johnsen, B. H., Laberg, J. C., Cox, W. M., Vaksdal, A., & Hugdahl, K. (1994). Alcoholic subjects' attentional bias in the processing of alcohol-related words. *Psychology of Addictive Behaviors, 8,* 111–115.

Jones, B. C., Jones, B. T., Blundell, L., & Bruce, G. (2002). Social users of alcohol and cannabis who detect substance-related changes in a change blindness paradigm report higher levels of use than those detecting substance-neutral changes. *Psychopharmacology, 165,* 93–96.

Jones, B. T. (2004). Changing alcohol expectancies: Techniques for altering motivations for drinking. In W. M. Cox & E. Klinger (Eds.), *Handbook of motivational counseling: Motivating people for change* (pp. 373–387). Chichester, England: Wiley.

Jones, B. T., Bruce, G., Livingstone, S., & Reed, E. (in press). Alcohol-related attentional bias in problem drinkers with the flicker change blindness paradigm. *Psychology of Addictive Behaviors.*

Jones, B. T., Corbin, W., & Fromme, K. (2001). A review of expectancy theory and alcohol consumption. *Addiction, 96,* 55–70.

Jones, B. T., Jones, B. C., Smith, H., & Copely, N. (2003). A flicker paradigm for inducing change blindness reveals alcohol and cannabis information processing biases in social users. *Addiction, 98,* 235–244.

Jones, B. T., & McMahon, J. (1998). Alcohol motivations as outcome expectancies. In W. M. Miller & N. Heather (Eds.), *Treating addictive behaviors* (2nd ed., pp. 75–91). New York: Plenum Press.

Keogh, E., Dillon, C., Georgiou, G., & Hunt, C. (2001). Selective attentional biases for physical threat in physical anxiety sensitivity. *Anxiety Disorders, 15,* 299–315.

Lee, N. K., Greely, J., & Oei, T. P. (1999). Relationship of positive and negative alcohol expectancies to patterns of consumption of alcohol in social drinkers. *Addictive Behaviors, 24,* 359–369.

Leigh, B. C., & Stacy, A. W. (1998). Individual differences in memory associations involving the positive and negative outcomes of alcohol use. *Psychology of Addictive Behaviors, 12,* 9–46.

Li, R. H. Y., Wing, Y. K., Ho, S. C., & Fong, S. Y. Y. (2002). Gender differences in insomnia—A study in the Hong Kong Chinese population. *Journal of Psychosomatic Research, 53,* 601–609.

Lundh, L.-G. Froding, A., Gyllenhammer, L., Broman, J.-K., & Hetta, J. (1997). Cognitive bias and memory performance in patients with persistent insomnia. *Scandanavian Journal of Behaviour Therapy, 26,* 27–35.

McMahon, J., Jones, B. T., & O'Donnell, P. (1994). Comparing positive and negative alcohol expectancies in male and female social drinkers. *Addiction Research, 1,* 349–365.

Mogg, K., & Bradley, B. P. (1998). A cognitive-motivational analysis of anxiety. *Behavioral Research Therapy, 36,* 809–848.

Mogg, K., Mathews, A., Bird, C., & MacGregor-Morris, R. (1990). Effects of stress and anxiety on the processing of threat stimuli. *Journal of Personality and Social Psychology, 59,* 1230–1237.

Morin, C. M., Hauri, P. J., Espie, C. A., Spielman, A., Buysse, D., Bootzin, R. R., et al. (1999). Nonpharmacologic treatment of insomnia: An American Academy of Sleep Medicine review. *Sleep, 22,* 1134–1156.

Murtagh, D. R., & Greenwood, K. M. (1995). Identifying effective psychological treatments for insomnia: A meta-analysis. *Journal of Consulting and Clinical Psychology, 63,* 79–89.

O'Regan, J. K., & Noe, A. (2000, June) *Experience is not something we feel but something we do: A principled way of explaining sensory phenomenology, with change blindness and other empirical consequences.* Paper presented at the Fourth Conference of the Association for the Scientific Study of Consciousness, Brussels, Belgium.

Oyahon, M. M., Caulet, M., & Guilleminault, C. (1997). How a general population perceives its sleep and how this relates to the complaint of insomnia. *Sleep, 20,* 715–723.

Reiss, S. (1997). Trait anxiety: It's not what you think it is. *Journal of Anxiety Disorders, 11,* 201–214.

Rensink, R. A. (2002). Change detection. *Annual Review of Psychology, 53,* 245–277.

Rensink, R. A., O'Regan, J. K., & Clark, J. J. (1997). To see or not to see: The need for attention to perceive changes in scenes. *Psychological Science, 8,* 368–373.

Ryan, F. (2002). Attentional bias and alcohol dependence: A controlled study using the modified Stroop paradigm. *Addictive Behaviors, 27,* 471–482.

Sharma, D., Albery, I. P., & Cook, C. (2001). Selective attentional bias to alcohol-related stimuli in problem drinkers and non-problem drinkers. *Addiction, 96,* 285–295.

Simons, D. J. (2000). Current approaches to change blindness. *Visual Cognition, 7,* 1–15.

Simons, D. J., & Levin, D. T. (1997). Change blindness. *Trends in Cognitive Sciences, 1,* 261–267.

Simons, D. J., & Rensink, R. A. (2005). Change blindness: Past, present, and future. *Trends in Cognitive Sciences, 9,* 16–20.

Smith, M. T., Perlis, M. L., Park, A., Smith, M. S., Pennington, J., Giles, D. E., & Buysse, D. J. (2002). Comparative meta-analysis of pharmacotherapy and behavior therapy for persistent insomnia. *American Journal of Psychiatry, 159,* 5–11.

Taylor, L. M., Espie, C. A., & White, C. A. (2003). Attentional bias in people with acute versus persistent insomnia secondary to cancer. *Behavioral Sleep Medicine, 1,* 200–212.

Townshend, J. M., & Duka, T. (2001). Attentional bias associated with alcohol cues: Differences between heavy and occasional social drinkers. *Psychopharmacology, 15,* 67–74.

Waters, A. J., & Feyerabend, C. (2000). Determinants and effects of attentional bias in smokers. *Psychology of Addictive Behaviors, 14,* 111–120.

Wicklow, A., & Espie, C. A. (2000). Intrusive thoughts and their relationship to an actigraphic measurement of sleep: Towards a cognitive model of insomnia. *Behaviour Research & Therapy, 38,* 679–693.

Williams, J. M. G., Mathews, A., & MacLeod, C. (1996). The emotional Stroop task and psychopathology. *Psychological Bulletin, 120,* 3–24.

Received April 8, 2003
Revision received July 16, 2004
Accepted August 4, 2004 ■

J. Sleep Res. (2006) **15,** 212–221

Who is pre-occupied with sleep? A comparison of attention bias in people with psychophysiological insomnia, delayed sleep phase syndrome and good sleepers using the induced change blindness paradigm

LAUREN M. MARCHETTI[1], STEPHANY M. BIELLO[1], NIALL M. BROOMFIELD[2], KENNETH M. A. MACMAHON[2] and COLIN A. ESPIE[2]

[1]Department of Psychology, University of Glasgow, Glasgow, UK and [2]University of Glasgow Sleep Research Laboratory, Southern General Hospital, Glasgow, UK

Accepted in revised form 16 December 2005; received 1 July 2005

SUMMARY Cognitive models of insomnia suggest that selective attention may be involved in maintaining the disorder. However, direct assessment of selective attention is limited. Using the inducing change blindness (ICB) paradigm we aimed to determine whether there is attentional preference for sleep-related stimuli in psychophysiological insomnia (PI) relative to delayed sleep phase syndrome (DSPS) and good sleepers (GS). In the ICB task, a visual scene, comprising both sleep-related and neutral stimuli, 'flickers' back and forth with one element (sleep or neutral) of the scene changing between presentations. Therefore, a 2×3 totally between-participants design was employed. The dependent variable was the number of flickers it took for the participant to identify the change. Ninety individuals (30 per group) were classified using ICSD-R criteria, self-report diaries and wrist actigraphy. As predicted, PI detected a sleep-related change significantly quicker than DSPS and GS, and significantly quicker than a sleep-neutral change. Unexpectedly, DSPS detected a sleep-related change significantly quicker than GS. No other differences were observed between the two controls. These results support the notion that there is an attention bias to sleep stimuli in PI, suggesting that selective attention tasks such as the ICB may be a useful objective index of cognitive arousal in insomnia. The results also suggest that there may be an element of sleep preoccupation associated with DSPS. Results are discussed with reference to other experiments on attentional processing in insomnia.

KEYWORDS attention bias, cognitive arousal, delayed sleep phase syndrome, inducing change blindness flicker paradigm, insomnia

INTRODUCTION

Insomnia is, in part, characterized by mental arousal (AASM, 2004, 2005). Indeed, having an overactive mind has been the attribution of poor sleep rated most highly, both by insomniacs and non-insomniacs (Bootzin *et al.*, 1991; Espie and Tweedie, 1991; Lichstein and Rosenthal, 1980; Nicassio *et al.*, 1985). Cognitions interfering with sleep typically form part of a dysfunctional belief system, and reflect a tendency towards problem-solving, worry, and heightened self-awareness both in bed and during the daytime (Harvey, 2000; Harvey and Espie, 2004; Morin *et al.*, 1993; Neitzert Semler and Harvey, in press; Watts *et al.*, 1994; Wicklow and Espie, 2000). Such studies have relied on subjective methods to index the mental component of insomnia. Self-report is, of course, appropriate because it reflects the experience of insomnia. However, there are clear advantages to objective indices, when they are available.

In this context, it is interesting that the notion of preoccupation with sleep maps neatly onto the concept of *selective*

Correspondence: Colin A. Espie, University of Glasgow Sleep Research Laboratory, Southern General Hospital, 1345 Govan Road, Glasgow G51 4TF, UK. Tel.: 0141 232 7700; fax: 0141 232 7697; e-mail: c.espie@clinmed.gla.ac.uk

attention. Psychologists interested in information processing have researched human attention extensively (Allport, 1980; Broadbent, 1958; Deutsch and Deutsch, 1963; Posner, 1980; Shiffrin and Schneider, 1977). In short, stimuli that are *salient* are likely to attract attention, because of an information processing bias or *attention bias*. Such biases can be measured objectively using computerized cognitive probe tasks where information processing speed is taken as a proxy for selective attention. These tasks use salient and neutral word or picture stimuli within an experimental test paradigm.

Support has been found implicating attention bias in the perpetuation of a wide range of anxiety-related psychological disorders (see Mogg and Bradley, 1998; Williams *et al*., 1997) and habit/dependence disorders (Jones *et al*., 2003). Generally, an attention bias has been found in those who have a clinical diagnosis, contributing to the explanation of why such disorders are frequently self-maintaining, and why relapse often occurs after emergent treatment response (Drummond *et al*., 1995; Mogg *et al*., 1990). It is thought that attention bias in anxiety is conveyed by *threat* monitoring (Matthews and MacLeod, 1994), whereas in the addictions it may be due to *craving* (Lusher *et al*., 2004).

So is it reasonable to consider attention bias a proxy for cognitive arousal in insomnia? If so, the possibility emerges for objective measurement of the mental concern associated with poor sleep. This topic has been recently reviewed (Espie *et al*., in press), however, a brief synopsis may be helpful.

Harvey's (2002) cognitive model of insomnia proposes that the anxious state generated by excessive worry, negative thoughts and autonomic arousal triggers selective attention towards and monitoring of internal and external sleep-related threat cues. Likewise, Espie's (2002) psychobiological model emphasizes the inhibitory influence of attention and intention upon normal sleep engagement. It is argued that such processes are dysregulatory because they contravene the 'automaticity' of normal unattended sleep engagement (Espie, 2002). These theories are consistent with ICSD-2 (AASM, 2005) text describing just how marked the attention to sleep is in psychophysiological insomnia (PI) – 'Concerns about sleep grow progressively over months or years as sleep gradually deteriorates until the desire to obtain a good night's sleep becomes the person's major concern' (p. I-6). This statement conveys *both* a sense of incrementing distress associated with sleeplessness (cf. threat), *and* a preoccupying longing for sleep (cf. craving) that might serve as preconditions for attention bias.

To date, three studies have applied cognitive probe tasks to insomnia. Lundh *et al*.'s (1997) study was a pioneering piece of work because it translated the emotional Stroop task into the insomnia field. The Stroop task involves target (salient) and control (neutral) words being presented at random in different ink colours. Subjects are asked to respond quickly to the presented colour by pressing the corresponding coloured button on a response box. They are instructed to ignore the actual meaning of the words. Response latencies for colour identification are automatically recorded for each stimulus. Longer response latency is thought to suggest increased attention bias because automatic processing of word meaning for salient words is likely to interfere with (slow down) colour naming relative to response time for the neutral words: the so-called interference effect.

Lundh *et al*. (1997) found that people with insomnia had prolonged response latency for sleep-related words. However, this effect was also evident in a control population of good sleepers (GS), and there was no group difference on the Stroop interference index; a result inconsistent with the attention bias hypothesis. Lundh *et al*. (1997) suggested that sleep-related words might have emotional valence irrespective of sleep problems. However, the extensive literature on the Stroop task would not predict experimental effects in normal control groups. Of course, recruited GS may have a particular interest in sleep, and this might yield a bias. Also, no measure of affective state (known to influence Stroop findings) was taken, and diagnostic criteria were not reported for the insomnia group.

Taylor *et al*. (2003) also used the Stroop task, selecting a cancer population because the primary purpose was to investigate the development of insomnia associated with stress in people who had previously been GS. None of the participants had insomnia prior to diagnosis; that is they were a 'true' secondary insomnia population rather than people whose (pre-existing) insomnia had been exacerbated. Two groups of people with cancer and insomnia, 0–3 and 12–18 months after cancer diagnosis, completed the task comprising cancer-related, sleep-related and neutral word cues. Both groups demonstrated attention bias for cancer-related words but only the persistent insomnia group demonstrated attention bias for sleep-related words. The fact that interference effects were absent at 0–3 months but evident at 12–18 months, therefore, suggested that selective attention bias towards sleep may play a role in the transition from adjustment insomnia to PI.

There has been debate over whether the Stroop task measures increased vigilance or simply reflects the impact of heightened arousal interfering with information processing when salient stimuli are presented. The dot-probe task has been posited as one solution to this problem (MacLeod *et al*., 1986). In this task, words are simultaneously presented to two areas on a computer screen. The ensuing distribution of visual attention is measured by recording detection latency for a visual probe that could appear in the spatial location of either word, immediately after the display of that word has terminated. The trials providing the data of interest are those in which one of the words is salient. By examining the impact of sets of such words on relative probe detection latencies in the two spatial areas, it is possible to determine whether visual attention has shifted towards or away from such stimuli.

MacMahon *et al*. (unpublished data) used the dot-probe task with 63 young adults across three experimental groups [PI, delayed sleep phase syndrome (DSPS) and GS]. The DSPS group was employed as a further, clinical, control sample of people who, like PI participants, had sleep-onset problems, but who would not be expected to exhibit cognitive arousal as an

explanatory mechanism for their continued wakefulness. Results supported the predictions, with those in the PI group showing a significantly greater processing bias towards sleep-related words (in comparison with neutral words) when compared with the GS and DSPS groups. Notably, the GS and DSPS groups did not differ from each other, suggesting that the underpinning mechanism maintaining DSPS is not an attention bias. This is consistent with DSPS as an endogenous circadian problem.

It is also possible to explore attention bias with digitized objects (i.e. pictures, rather than words) using a flicker paradigm featuring a perceptual phenomenon called induced change blindness (ICB: Rensink, 2002; Simons, 2000). Research reveals that when a change is made to a visual scene (and the process of change is hidden from view), it is more difficult to detect than might be expected. A single feature of a visual scene is changed between successively repeated brief presentations until the change is detected – essentially the ICB is a 'spot the difference' task. Change-detection latency, measured by the number of flickers it takes for the change to be identified, is explained by object salience. For example, in the alcohol field, problem drinkers take fewer flickers to detect an alcohol-related change and are faster to detect such changes than control subjects. Interest in the control that sleep-related objects might have over sleep behaviour is long-established. Within a conditioning framework, bedroom environment objects might become discriminative stimuli for sleep (Bootzin *et al*., 1991), but when the bedroom-sleep contingencies are broken, they might become discriminative stimulus for wakefulness. In that regard the ICB paradigm may be well suited to investigating the influence of the bedroom environment on sleep.

Jones *et al*. (2005) suggested that if attention is implicated in the development of persistent insomnia it might be expected that a systematically changing attention bias might be observed, not just at the clinical pole. In their ICB study, 192 participants (mean age 32 years) were selected for this totally between-subjects experiment. Participants completed the 15-min ICB task, after which they were assessed for sleep quality. Importantly, therefore, retrospective group assignment was blind to the dependent variable of the analyses, change detection latency. Jones *et al*.'s results revealed significant differences in change detection latency between poor, moderate and GS for the sleep-related change. Only the poor sleepers, who detected sleep-related change quicker than neutral change, demonstrated selective attention bias for sleep salient stimuli. Moderate sleepers showed a trend in the same direction. By contrast, GS detected the change with the neutral objects significantly quicker. Hierarchical regression was then applied to test the relationship between change detection latency and a continuous representation of the global PSQI score. This evidenced a systematically changing effect of sleep quality upon attention bias, independent of age, gender and depressive symptom level.

Thus, three of the four available experiments suggest that attention bias probe tasks may index and discriminate cognitive arousal in insomnia. It seems plausible then that this paradigm could be a useful research tool.

METHODS

Aims and hypotheses

The current study extends research in this field in two ways. First, this ICB experiment used novel sleep-related and sleep-neutral objects to undergo a 'change' in the sequence. Jones *et al*. (2005) measured the response to one sleep-related change and one sleep-neutral change; we wanted to rule out the possibility of an idiosyncratic effect and to improve generalizability. Second, an additional, clinical control group of DSPS was included, in keeping with the MacMahon *et al*. protocol. DSPS involves initial insomnia, similar to sleep-onset PI, but with no presumed psychological mechanism. Comparison with normal sleepers, without a clinical control, leads to problems in testing the specificity of insomnia phenomena. Therefore, the DSPS group allowed us to assess differences in selective attention between sleep disorders that are behaviourally similar in terms of sleep initiation problems.

We hypothesized that (1) people with PI would detect a sleep-related change significantly quicker than would GS or DSPS participants; (2) with a neutral change there should be slower response in PI, because their attention would be drawn to the 'sleep area' of the visual scene, and would have to shift to notice a neutral change; (3) there would be no significant difference between detection latencies to sleep-related versus sleep-neutral changes by the two control groups, GS and DSPS; and (4) there would not be differences in detection latencies between GS versus DSPS, at either level of change.

Design

A 2 (experimental condition) × 3 (group) entirely between-participants design was employed. The necessary confirmation process (making explicit the semantic nature of the stimulus carrying the change) means that a within-participant factor cannot be implemented in ICB experiments. This is because any explicit knowledge carried over by participants as a result of the confirmation process would compromise the main objective, exploring their implicit knowledge of the stimuli contained in the scene (Jones *et al*., 2003). Therefore, each group (GS, PI, DSPS) was split in half at random ($n = 15$) to represent the nature of the change introduced in the photographic image (sleep-related or sleep-neutral).

Allocation of sleep-related stimuli to the left and sleep-neutral to the right side of the visual field was chosen at random. This orientation of the stimulus presentation was kept constant in all conditions. Including mirror reversal to control for differences in scene processing, due to left–right placement, was not considered necessary because no differences have found between normal and reversed scene processing in previous ICB flicker paradigm experiments (Jones *et al*., 2003).

Participants

Data were derived from students and staff at the University of Glasgow. Appraisal of individual sleeping patterns was conducted in three phases. Phase 1 of the screening phase was based upon the information gained from an initial mass advertisement email, which, after giving a brief introduction about the Glasgow Sleep Laboratory asked five specific questions: (1) Are you someone who struggles to sleep at night? (2) Once asleep do you regularly wake during the night? (3) How many hours on average do you sleep per night? (4) Would you describe yourself as a lark or an owl? (5) Are you a good sleeper, who falls asleep as soon as your head touches the pillow and wakes up feeling refreshed in the morning? The email concluded by stating 'if you feel you would gain from being part of a sleep experiment and would like to learn a little more about your sleeping patterns, and ways to improve them, we would like to hear from you. Please send information about your sleep pattern based on the questions above'.

In total, 253 individuals responded to this email. Of these 253 respondents, 189 reported symptoms similar to PI and DSPS and were subsequently emailed for a second time (phase 2) to obtain further details of their sleep patterns. This second email asked more specific questions: 'how many minutes does it take you, on average, to fall asleep at night', 'once asleep do you wake at regular intervals throughout the night', 'How long do you predict you are awake during the night', 'On average how many hours of sleep do you get per night', 'Do you view your sleep as a problem', 'What time do you get up in the morning' , 'Do you feel refreshed?'.

From the 189 individuals emailed in phase 2, 157 responded and 97 were assessed as potentially suitable and subsequently completed the ICB procedure. The third and final phase, after the experiment, applied rigorous criteria and prospective diary and actigraphic assessment to confirm allocations; 90 of the 97 tested were included in analyses. The main advantage of the third screening phase occurring 'post experiment' was that it further limited the priming effect upon participants. Further details of recruitment and assessment are provided as part of the procedures for the investigation.

Inclusion/exclusion criteria

Participants met combined DSM-IV and ICSD-R criteria for primary insomnia of the PI type or DSPS, and those with PI were required to score >6 on the PSQI. PI exclusion criteria included active psychological or drug interventions for sleep problems, or when sleep disorder was suspected as being the result of substance misuse or physical ill health. The same exclusion criteria applied for DSPS. Good sleepers were required to score <5 on the PSQI, report themselves as being 'good' sleepers, and meet no criteria for a sleep disorder at the present time or in the past. For all three participant groups, scoring above the cut off markers for depression (BDI SF-4) resulted in exclusion from analyses. Actigraphy was used to confirm participants' subjective account of their sleeping complaint and to aid in the differentiation between PI and DSPS in those reporting sleep difficulties.

Experimental protocol and apparatus

The flicker ICB paradigm was employed. An originating stimulus (OS) was presented for 250 ms – then a mask for 80 ms – then the changed stimulus (CS) for 250 ms – and finally a mask for 80 ms (Fig. 1). This four-presentation cycle was repeated seamlessly until change-detection. Thus, the combined number of transitions between OS–CS and CS–OS (the 'flickers' to change-detection) was the dependent variable.

Sleep-related stimuli were chosen from a questionnaire distributed, randomly, to 60 individuals. This questionnaire was devised for the Jones *et al*. (2005) study. People were asked to list five or more objects that they associated with sleep and going to bed. Evaluation of the lists yielded a 'top 12' most commonly suggested items. These items were photographed and embedded in a collection of 12 neutral, individually photographed objects. A further 30 individuals were then asked to rate all 24 photographs on a 1–10 'sleep-relatedness' scale (1 = highly sleep-related, 10 = not sleep-related at all). In this study the item with the second highest rating (teddy bear, 5.6) was used, and paired with an entirely sleep-neutral item (a mug).

A different flicker pair of stimuli was used for each of the two levels of the factor; nature of change (sleep-related and neutral). Each pair contained the same OS comprising seven sleep-related objects and an equal number of neutral objects arranged in two collections on either side of the scene midline. The second stimulus (CS) of each pair was identical to the OS but for one small change: a sleep-related change (removing the teddy bear) or a neutral change (removing the mug). The two stimuli of a pair were then presented in continuous succession (each replacing the other) until the change was detected. A brief 'mask' was inserted in between the flicker pairs to suppress visual transients. The experiment was implemented using a Dell optiplex GX270 laptop and the experiment-generation package SuperLab Pro 2.02 (Cedrus Corporation, San Pedro, CA, USA). The size of the screen was 28 cm diagonal with stimuli positioned centrally; the viewing distance

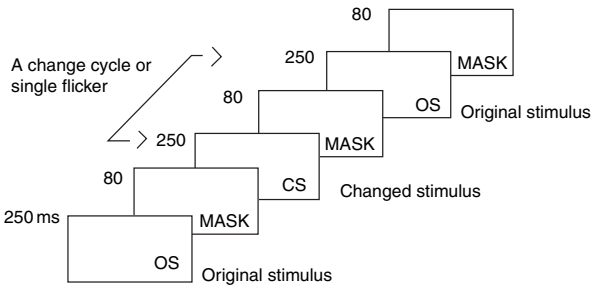


Figure 1. A flicker paradigm for inducing change blindness illustrating a change cycle or single 'flicker'. OS, original stimulus; CS, changed stimulus.

was approximately 45 cm. Stimuli were full colour photographs (1280 × 960 pixels) taken in natural daylight. Fig. 2 shows the OS, and the two changed stimuli; one with the sleep-related change (the teddy bear is taken away) and the other with the sleep-neutral change (the mug is taken away).

Procedure

As described previously, contact was first made with participants by email, inviting individuals to contact the first author with details of their sleeping patterns in answer to a brief set of questions. These questions enabled the experimenter to screen and select the responses that best resembled the traits of the various sleep groups involved in the study, invite the suitable candidates to give more specific information about sleep quality and subsequently invite them to take further part in the study. Thus, group allocation was 'largely known' to the experimenter before testing commenced.

Participants were ICB tested in an assessment room in the Department of Psychology, University of Glasgow. To ensure an equal distribution of participants across the experimental conditions (sleep and neutral), assignment to conditions alternated consecutively. Thus, if one participant completed the 'sleep condition', the following would complete the 'neutral condition'. Furthermore, an identical instruction protocol was presented to participants upon arrival, regardless of their group allocation. To begin, participants pressed any button on the response button box to display instructions which said that (when they pressed it again) they would 'see a scene on the screen that would be switched on and off repeatedly – each appearance and disappearance lasting less than a second'. They were instructed that they had to 'spot a change made to the visual scene at some point in the series of "flickers" and to indicate this detection by immediately pressing the button on the response box'. After the participants had detected and responded to the change, they were asked to 'confirm what the change was'. Only those who had correctly identified the change were retained as having completed the task (all participants who completed the ICB flicker paradigm identified the correct change in the scene). They were verbally asked if they understood the instructions and were invited to continue (without additional practice). The experiment then commenced. After completion of the flicker task each participant immediately underwent the second and detailed assessment phase. This comprised three components.

First, each participant was interviewed to evaluate his/her general sleeping patterns. The interviewer proposed questions relating to the DSM-IV and ICSD-R criteria for PI and DSPS, using our laboratory's structured interview format (Espie, 2002; (Morin and Espie, 2003).

Second, several questionnaires were administered. The Pittsburgh Sleep Quality Index (PSQI; Buysse *et al.*, 1989) provides a reliable, valid and standardized measure of sleep quality; to discriminate between 'good' and 'poor' sleepers. A PSQI global score > 5 indicates that a subject is having severe difficulty in at least two areas, or moderate difficulty in more than three areas of sleep quality. This global score conveys information about the severity of the subject's problem, and

Original stimulus, OS

Neutral changed stimulus, CS-N
(mug removed)

Sleep-related changed stimulus,
CS-S (teddy bear removed)

Figure 2. Grey scale versions of the full colour used; original stimulus (OS) and the two changed stimuli for each of the two levels of the nature of change factor – sleep-related change (CS-S) and neutral change (CS-N).

the number of problems present, through a single measure. Recent, independent study has validated this cut off and confirmed reliability (Cronbach's $\alpha = 0.85$, test–retest $r = 0.84$; Backhaus *et al*., 2002). The revised version of the Spielberger State and Trait Anxiety Inventory (STAI; Spielberger *et al*., 1983) is a reliable and valid scale. Alpha coefficients (STAI-S $\alpha = 0.93$ and STAI-T $\alpha = 0.90$) reflect strong internal consistency. Construct, concurrent, divergent and convergent validity have been demonstrated (Spielberger *et al*. 1983). Finally, the Beck Depression Inventory (BDI: (Beck and Steer, 1987) is a 21-item self-report form covering symptoms of depression. The BDI demonstrates high internal consistency, with α coefficients of 0.86 and 0.81 for psychiatric and non-psychiatric populations, respectively. The 13-item short form, adopted for this experiment, has shown similar reliabilities (Beck *et al*., 2000).

Third, participants wore an actigraph and completed a standard sleep diary (Espie and Tweedie, 1991) for seven nights following the experiment. The actigraph is a small, non-intrusive watch-like device, which records objective rest/activity periods based on wearer movements. For the use of actigraphy in insomnia patients, epoch by epoch agreement rates range from 78% to 90% (Jean-Louis *et al*., 1997; Sadeh *et al*., 1995). The inclusion of actigraphy was to help determine the sleep/wake (circadian) patterns of each participant (Sadeh *et al*., 1995) rather than to estimate sleep continuity variables. Therefore, this measure assisted differential diagnosis of PI and DSPS and confirmed group status. In particular, we used Van Someren *et al*. (1999) non-parametric circadian rhythm analysis (NPCRA) to calculate the L5 component (onset of the first 5 h of lowest movement period) which gives an estimate of circadian sleep phase onset.

Upon return, 1 week later, the participants had opportunity to discuss their sleeping patterns and were offered a copy of The Good Sleep Guide [Prepared by Colin A. Espie for guidance report (National Medical Advisory Committee, 1994) and now recommended by the British Sleep Society].

RESULTS

Descriptive and clinical data

Inspection of subjective and objective sleep quality data revealed that seven of the original participants did not meet inclusion criteria for PI, DSPS or GS; four cases due to sleeping disorders other than PI or DSPS, one case due to the patient receiving medical intervention for sleep disruption and one due to lack of sleep caused by a broken collar-bone. On five occasions, individuals believed to have PI from the general information given in response to the email advertisement, were subsequently re-allocated to the DSPS group on the basis of the more thorough interview, questionnaire measure and actigraphy data. The experimental population as a whole consisted of 45 females and 45 males with an average age of 22.8 years. Table 1 shows the demographics of the experimental population for each sleep quality group.

Table 1 also presents summary scores for the other clinical questionnaire data. There was a significant effect of group at both levels of STAI; Trait: [$F(2,87) = 30.12$, $P < 0.0001$], State: [$F(2,87) = 23.43$, $P < 0.0001$]. Scheffe *post hoc* tests revealed that PI were generally (trait) and situationally (state) more anxious than GS, $P < 0.0001$ and $P < 0.0001$, respectively. Similarly, on both STAI scales, PI scored significantly higher than DSPS $P < 0.001$ trait, $P < 0.001$ state, respectively. DSPS scored higher on the state measure of anxiety, $P < 0.001$, than GS, but were not significantly different on trait measures of anxiety, $P = 1.92$. There was no significant main effect of group for the BDI data, which revealed low mean scores in all groups [$F(2,87) = 3.04$, $P = 0.053$], although the trend in the data was for PI to score higher than either GS or DSPS.

Sleep data

Table 2 summarizes mean and standard deviation data for the PSQI, and selected sleep diary and actigraphic measures. Analyses revealed a significant effect of group at the level of PSQI [$F(2, 87) = 61.07$, $P < 0.0001$]. Scheffe *post hoc* analyses revealed that PI scored significantly higher than both GS $P < 0.0001$ and DSPS $P < 0.0001$, and DSPS scored significantly higher than GS $P < 0.01$.

On the sleep diary, total sleep time (TST) was significantly different between the three sleep quality groups [$F(2,87) = 41.22$, $P < 0.0001$], with PI participants reporting less than 5 h sleep, compared with greater than 8 h for DSPS and GS ($P < 0.0001$ and $P < 0.0001$, respectively). There was no difference in TST between GS and DSPS groups. Sleep-onset latency (SOL) was also significantly different between

Table 1 Demographic and clinical summary data

	Primary insomnia (n = *30*)		*Delayed sleep phase syndrome* (n = *30*)		*Good sleep* (n = *30*)		*Between-group analyses* (P-*value*)
	M	*SD*	*M*	*SD*	*M*	*SD*	
Age	22.5	2.81	22.7	3.56	23.2	1.69	NS
Gender (% female)	51%	–	49%	–	50%	–	NS
STAI state	37.2	7.83	32.8	8.74	24.3	5.35	< 0.0001
STAI – trait	48.1	10.52	37.9	11.40	27.3	9.15	< 0.0001
BDI – short form	4.7	3.52	2.3	3.32	2.9	2.36	0.053

STAI, State Trait Anxiety Inventory; BDI, Beck Depression Scale.

Table 2 Sleep summary data

	Primary insomnia (n = *30*)		*Delayed sleep phase syndrome* (n = *30*)		*Good sleep* (n = *30*)		*Between-group analyses* (P *value*)
	M	*SD*	*M*	*SD*	*M*	*SD*	
PSQI	9.5	2.4	4.8	3.2	2.6	1.7	< 0.0001
Diary TST (h/min)	4.83	1.83	8.33	1.81	8.27	1.46	< 0.0001
Diary SOL (min)	47.63	17.85	17.57	9.09	7.9	4.57	< 0.0001
ActigraphyL5 (24 h clock)	01 : 10	0.9	04 : 00	1.9	N/A	N/A	< 0.05

PSQI, Pittsburgh Sleep Quality Index; TST, total sleep time; SOL, sleep-onset latency; L5, onset of lowest 5 h of motor output.

groups [$F(2,87) = 88.36$, $P < 0.0001$], with PI taking significantly longer to fall asleep than DSPS ($P < 0.0001$), and GS ($P < 0.0001$), and DSPS taking significantly longer to fall asleep than GS ($P < 0.05$).

Analysis of actigraphy data using NPCRA software revealed a significant main effect of group on L5 data [$F(1,58) = 45.81$, $P < 0.0001$], with DSPS lowest peak of activity beginning significantly later than PI. These data indicate a sleep-onset phase delay of 2.9 h in DSPS relative to PI.

Change detection data

Table 3 summarizes the reaction time means (number of flickers) and standard deviations for each sleep group. To test our hypotheses, a stimulus change/sleep quality (2×3) between participants ANOVA was carried out. As predicted the stimulus change/sleep quality interaction was significant [$F(2,84) = 51.7$, $P < 0.0001$] with a significant effect of change [$F(1,84) = 70.3$, $P < 0.0001$] and quality [$F(2,84) = 3.5$, $P < 0.05$]. There was a significant main effect of change at the level of PI [$F(1, 84) = 171.5$, $P < 0.0001$] that was not present at the level of GS [$F(1,84) = 0.004$, $P = 0.95$] or DSPS [$F(1,84) = 2.2$, $P = 0.14$]. Significant main effects of quality were revealed for both the sleep change [$F(2,84) = 28.6$, $P < 0.0001$] and neutral change [$F(2,84) = 26.7$, $P < 0.0001$].

Fig. 3 illustrates the tests for simple main effects that permit the following interpretation of the stimulus change/sleep quality interaction. PI participants detected a sleep-related change significantly quicker than a sleep neutral change [$t(15) = 13.10$, $P < 0.0001$]. No such differences were observed in either the GS [$t(15) = 0.06$, $P = 0.95$], or DSPS groups [$t(15) = 1.5$, $P = 0.14$]. For the sleep-related change, responses of PI were significantly quicker than GS [$t(15) = 7.5$, $P < 0.0001$] and DSPS [$t(15) = 4.8$, $P < 0.0001$]; and responses of DSPS were significantly quicker than those of GS [$t(15) = 2.66$, $P < 0.01$]. By comparison for sleep-neutral changes, responses of GS and DSPS were significantly quicker than PI [$t(15) = 5.7$, $P < 0.0001$; $t(15) = 6.8$, $P < 0.0001$], respectively. No difference was observed between GS and DSPS.

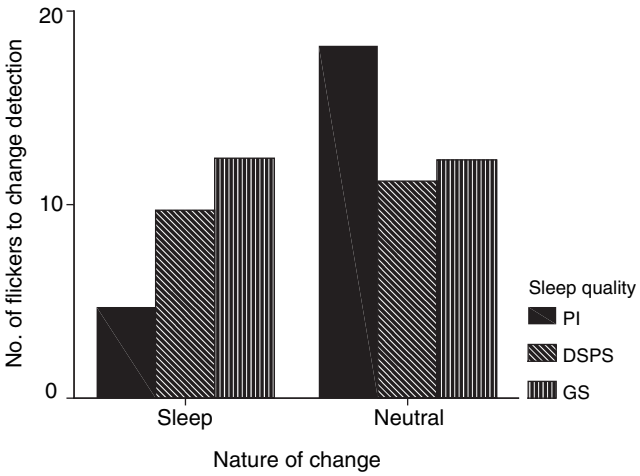


Figure 3. Mean sleep-related and sleep-neutral change detection latencies for the three sleep quality groups.

These main effects and simple effects appear consistent with our hypotheses that sleep-related stimuli would be more salient to PI, and thus detected quicker than sleep-neutral stimuli, and that PI participants would detect changes more quickly than DSPS or GS participants.

Table 3 Reaction times (RT) on the ICB change detection task. Data represent flickers to change detection

	Sleep change, mean (SD)	*Neutral change, mean (SD)*	*Collapsed mean RT*
Primary insomnia	4.7 (2.14)	18.2 (1.64)	11.6
Delayed sleep phase syndrome	9.7 (3.50)	11.2 (3.14)	10.5
Good sleep	12.4 (2.99)	12.3 (3.10)	12.4
Collapsed mean RT	8.9	13.9	

DISCUSSION

Insomnia research is constrained by the absence of reliable objective markers of the phenomenology of poor sleep. Mental arousal is a hallmark feature of insomnia. It is also a symptom of other psychopathological conditions – in which context it has been captured using techniques adapted from experimental cognitive psychology, based on the measurement of information processing speed to target stimuli. This experiment is one of a series from our laboratory in Glasgow that suggests that attention bias, measured by computerized probe tasks, may be

a useful index of cognitive arousal in insomnia. Based on our accumulating data sets, we propose that attention bias is a cognitive process, involved in the maintenance, and perhaps the development, of PI from adjustment insomnia, and detectable after the onset of the complaint.

This study applied the ICB flicker task to investigate the hypothesis that people with insomnia exhibit an attentional monitoring preference for sleep-related objects. This hypothesis was confirmed. Taken along with the results of our previous ICB study, which used different sleep-change stimuli (Jones *et al*., 2005), we suggest that this is not an idiosyncratic finding. These are the first experiments to apply a visual attention task to insomnia, and then show that people with insomnia are selectively attentive to common environmental sleep cues. Indeed, we would point out that the sleep stimuli were incidental and innocuous (this study: a teddy bear; Jones *et al*.: a slipper). They were simply objects that people associate with sleep or preparation for bed, and because people with insomnia have a heightened interest in this domain, they respond selectively to such stimuli when they are presented experimentally. Therefore, although not designed for the purpose of testing conditioned responses, our results lend weight to models of sleep-related arousal conditioning in insomnia (Bootzin, 1972; Perlis *et al*., 2001) whilst emphasizing the importance of the cognitive component.

It is also important that the objects used were not intrinsically commanding of attention, nor were they emotive or threatening. The literature on attention bias in psychological disorders is based on a threat-monitoring model, whereby words and images are presumed to grab attention because they are emotionally salient. Through conditioned association this may indeed be possible, even with everyday objects, and Harvey's (2002) model of insomnia is based largely on responses to perceived threat. However, we think it is too early to judge what may be motivating attentional processing. Studies need to be conducted that specifically manipulate the emotional valence of presented stimuli. We suggest that both threat (of sleeplessness) and craving (for sleep) are good candidate explanations that require further testing, and we have discussed this model, and potential methodologies, elsewhere (Espie *et al*., in press).

Attention bias is conceived of as an initial involuntary (unconscious, implicit) process that gives rise to voluntary (conscious, explicit) processes (Shiffrin and Schneider, 1977). In other words, pre-attentive processing guides the early, automatic capture of relevant information even when conscious access to the information is not available. A good illustration of this phenomenon was a study by Ohman and Soares (1994) that successfully elicited both psychophysiological reactivity and subjective fear in phobic participants to pictures of their feared objects when they were presented beyond their conscious awareness. It is not possible using an ICB study to address the 'level' at which processing begins, because the dependent variable is defined by awareness of the change. However, we noted that after completing the experiment many of our participants, including those with insomnia, were unaware that the experimental stimuli were particularly related to sleep or sleep disorder, yet experimental effects were found. The exploration of implicit processing and the transition to explicit processing is an important research topic (Espie *et al*., in press). Our study using the Stroop paradigm (Taylor *et al*., 2003) indicated that attention bias to sleep word cues was absent in acute/adjustment insomnia but present in persistent insomnia, and this further reinforces the importance of exploring how, and when, attention bias develops in relation to the complaint of insomnia.

In this study, we used DSPS as a control condition for our sleep-onset insomnia group. We feel it is important that our hypotheses were confirmed, not only in comparison with normal GS, but also against these clinical controls. This result replicates our finding, using the dot-probe task, of attention bias in PI relative to GS and DSPS (K.M.A. MacMahon *et al*., unpublished data). Insomnia is undoubtedly a multifactorial disorder, and one that can present heterogeneously. Separating out PI from circadian disorder in relation to difficulty initiating sleep, helps to ensure good discrimination on the independent variable. In addition, however, the contrast between PI and DSPS is that of disorders that are primarily seen as psychological and circadian, respectively. It is interesting, therefore, that in the present study we observed significant differences between GS and DSPS, with the latter exhibiting greater attention bias to the sleep stimulus change. We did not find this effect in our dot-probe experiment.

Several factors may account for the DSPS effect in this study. DSPS, particularly in younger people, may comprise two sub-populations, a socially driven DSPS and an inherent/genetic DSPS, whose responses to attentional measures may differ. The mechanisms that trigger DSPS in some individuals are often precipitated by life or social events and the effects of DSPS may lead to increased pre-sleep arousal when individuals with DSPS try to reset their clocks by attempting to sleep 'out of phase'. This could theoretically precipitate symptoms of, or the onset of PI (Lack and Bootzin, 2003). Conversely, of course, when individuals with PI are unable to fall asleep, they may inadvertently entrain their sleep to a later time, resulting in an element of DSPS. Our results may also depend on whether our DSPS participants were sleeping in phase or out of phase at the time of the experiment. In the latter case one might expect more insomnia symptoms. With this information in mind, we retrospectively assessed data from our DSPS group. Variations were evident with respect to the length of time the individuals, who were all university students, reported onset of the complaint. Some reported disorder onset in childhood and several reported a family history of DSPS, while the majority had suffered the disorder for as little as a year, and many of these reported problems occurring at the start of university. Furthermore, during the experimental period there was variation within and between DSPS subjects in terms of sleeping in and out of phase. Consequently, our results are likely explained by a combination of social, genetic and sampling frame factors, but we have insufficient numbers to separate our participants into subgroups for the purposes of

analysis. What is clear from our findings is that there may be a degree of overlap between psychological (PI) and circadian (DSPS) factors in young adult populations.

Several limitations to our methodology need to be raised. First, because this was a recruited population, our findings may not be generalizable to the clinical pole. Attention bias studies, therefore, require replication with clinical samples. Nevertheless, it should be noted that non-clinical populations do display cognitive overactivity at bedtime, excessive worry about sleep, distorted perception, and characteristic safety behaviours (cited in Harvey, 2002). Besides, processes detected at any stage of sleep disruption, particularly before the clinical extreme, may provide crucial evidence about the mechanisms involved in the maintenance/enhancement of the disorder proper. Second, this was not an intervention study. We do not know whether attention bias in insomnia reduces as a result of effective intervention, as it has been shown to do following cognitive behavioural therapy (CBT) for anxiety disorders (Matthews *et al*., 1995; Mogg *et al*., 1995). Demonstrating that established psychological treatments such as stimulus control or multi-component CBT impact attention bias in PI would add strength to the argument that such biases play a critical role. Third, our results suggest an important pathway in insomnia, but cross-sectional comparison of the type reported here cannot test aetiological factors. The work of Harvey and colleagues is leading the way in this regard by emphasizing the application of controlled experimental methods to tease out causal pathways associated with sleep associated monitoring (Neitzert-Semler and Harvey, in press; Tang *et al*., in press). One other limitation concerns the possibility that our results may be influenced by experimenter bias. Although allocation on the independent variables (sleep status and ICB condition) was known to the experimenter, our experimental methodology followed a rigorous written protocol for participant throughput and task instructions on the PC screen were identical for the ICB task. These features of experimental control make such bias an unlikely explanation for our findings.

In conclusion, the results of this study are consistent with other recent experiments in suggesting the potentially important role of selective attention bias in insomnia. A computerized cognitive probe paradigm, using tasks such as the ICB, may offer insomnia research a much-needed objective index of sleep-related mental arousal. However, at this stage we cannot say whether such attention biases are due to a generalized cognitive arousal in insomnia, to disease-specific distress or to preoccupation with disease-specific markers. Indeed, as we have acknowledged elsewhere the construct of arousal in insomnia remains poorly defined (Espie, 2002; Espie *et al*., in press). Our hope is that by developing and instrumenting measures of selective attention bias, as one facet of the cognitive process, we can help to move the field down-specific, testable lines, and away from exclusive reliance on generic self-report phenomena. Of course, we cannot yet conclude that attention bias is of primary importance in the aetiology of insomnia. It is entirely plausible that an information processing bias of this kind could arise simply because sleep is persistently disturbed, and so selective attention to sleep may be a secondary characteristic of chronic insomnia. This is one of the reasons that research purporting to explain critical pathways in the aetiology and maintenance of insomnia needs to include not only control samples of GS but also comparison groups with other forms of chronic sleep disorder.

ACKNOWLEDGEMENTS

This study received support from the Mortimer and Theresa Sackler Foundation.

REFERENCES

AASM. *Research Diagnostic Criteria for Insomnia*. AASM, Westchester, IL, 2004.

AASM. *International Classification of Sleep Disorders (ICSD-2)*, 2nd edn. AASM, Westchester, IL, 2005.

Allport, D. A. Patterns and actions: cognitive mechanisms are content specific. In: G. L. Claxton (Ed.) *Cognitive Psychology: New Directions*. Routledge & Kegan Paul, London, 1980.

Backhaus, J., Junghanns, K., Broocks, A., Riemann, D. and Hohagen, F. Test–retest reliability and validity of the Pittsburgh Sleep Quality Index in primary insomnia. *J. Psychosom. Res*., 2002, 53: 737–740.

Beck, A. T. and Steer, R. A. *Beck Depression Inventory (BDI) manual*. The Psychological Corporation, San Antonio, TX, 1987.

Beck, A. T., Steer, R. A. and Brown, G. K. *Manual for the Beck Depression Inventory – Fast Screen*. The Psychological Corporation, San Antonia, TX, 2000.

Bootzin, R. R.. Stimulus control treatment for insomnia. *Proceedings of the American Psychological Association*, 1972, 7: 395–396.

Bootzin, R. R., Epstein, D. and Wood, J. M. Stimulus control instructions. In: P. Hauri (Ed.) *Case Studies in Insomnia*. Plenum, New York, 1991.

Broadbent, D. E. *Perception and Communication*. Pergamon, Oxford, 1958.

Buysse, D. J., Reynolds, C. F., III, Monk, T. H., Berman, S. R. and Kupfer, D. J. The Pittsburgh Sleep Quality Index: a new instrument for psychiatric practice and research. *Psychiatry Res*., 1989, 28: 193–213.

Deutsch, J. A. and Deutsch, D. Attention, some theoretical considerations. *Psychol. Rev*., 1963, 70: 80–90.

Drummond, D. C., Tiffany, S. T., Glautier, S. and Remington, B. *Addictive Behaviour: Cue Exposure Theory and Practice*. Wiley, New York, 1995.

Espie, C. A. Insomnia: conceptual issues in the development, persistence, and treatment of sleep disorder in adults. *Annu. Rev. Psychol*., 2002, 53: 215–243.

Espie, C. A. and Tweedie, F. M. Sleep patterns and sleep problems amongst people with mental handicap. *J. Ment. Defic. Res*., 1991, 35, 25–36.

Espie, C. A., Broomfield, N. M., Macmahon, K. M. A., Macphee, L. M. and Taylor, L. M. The attention-intention-effort pathway in the development of psychophysiologic insomnia: an invited theoretical review. *Sleep Med. Rev*., in press.

Harvey, A. G. Pre-sleep cognitive activity: a comparison of sleep onset insomniacs and good sleepers. *Br. J. Clin. Psychol*., 2000, 39: 275–286.

Harvey, A. G. A cognitive model of insomnia. *Behav. Res. Ther*., 2002, 40: 869–893.

Harvey, K. J. and Espie, C. A. Development and preliminary validation of the Glasgow Content of Thoughts Inventory (GCTI):

a new measure for the assessment of pre-sleep cognitive activity. *Br. J. Clin. Psychol*., 2004, 43: 409–420.

Jean-Louis, G., Von Gizycki, H., Zizi, F., Spielman, A., Hauri, P. and Taub, H. The actigraph data analysis software: I. A novel approach to scoring and interpreting sleep-wake activity. *Percept. Mot. Skills*, 1997, 85: 207–216.

Jones, B. T., Jones, B. C., Smith, H. and Copley, N. A flicker paradigm for inducing change blindness reveals alcohol and cannabis information processing biases in social users. *Addiction*, 2003, 98: 235–244.

Jones, B. T., Macphee, L. M., Broomfield, N. M., Jones, B. C. and Espie, C. A. Sleep-related attentional bias in good, moderate and poor (primary insomnia) sleepers. *J. Abnorm. Psychol*., 2005, 114: 249–258.

Lack, L. and Bootzin, R. R. Circadian rhythm factors in insomnia and their treatment. In: M. L. Perlin and K. L. Lichstein (Eds) *Treating Sleep Disorders: Principles and Practice of Behavioural Sleep Medicine*. John Wiley & Sons, New Jersey, 2003.

Lichstein, K. L. and Rosenthal, T. L. Insomniacs' perceptions of cognitive versus somatic determinants of sleep disturbance. *J. Abnorm. Psychol*., 1980, 89: 105–107.

Lundh, L.-D., Froding, A., Gyllenhammer, L., Broman, J.-K. and Hetta, J. Cognitive bias and memory performance in patients with persistent insomnia. *Scand. J. Behav. Ther*., 1997, 26: 27–35.

Lusher, J., Chandler, C. and Ball, D. Alcohol dependence and the alcohol Stroop paradigm: evidence and issues. *Drug Alcohol Depend*., 2004, 75: 225–231.

MacLeod, C., Mathews, A. and Tata, P. Attentional bias in emotional disorders. *J. Abnorm. Psychol*., 1986, 95: 15–20.

Matthews, A. and MacLeod, C. Cognitive approaches to emotion and emotional disorders. *Annu. Rev. Psychol*., 1994, 45: 25–50.

Matthews, A., Mogg, K., Kentish, J. and Eysenck, M. Effects of psychological treatment on cognitive bias in generalised anxiety disorder. *Behav. Res. Ther*., 1995, 33: 293–303.

Mogg, K. and Bradley, B. P. A cognitive-motivational analysis of anxiety. *Behav. Res. Ther*., 1998, 36: 809–848.

Mogg, K., Mathews, A., Bird, C. and MacGregor-Morris, R. Effects of stress and anxiety on the processing of threat stimuli. *J. Pers. Soc. Psychol*., 1990, 59: 1230–1237.

Mogg, K., Bradley, B. P., Millar, N. and White, J. Cognitive bias in generalised anxiety disorder: a follow up study. *Behav. Res. Ther*., 1995, 33: 927–935.

Morin, C. M. and Espie, C. A. *A Clinical Guide to Assessment and Treatment*. Kluwer Academic/Plenum Publishers, New York, 2003.

Morin, C. M., Stone, J., Trinkle, D., Mercer, J. and Remsberg, S. Dysfunctional beliefs and attitudes about sleep among older adults with and without insomnia complaints. *Psychol. Ageing*, 1993, 8: 463–467.

National Medical Advisory Committee. *The Management of Anxiety and Insomnia*. Her Majesty's Stationary Office, Edinburgh, 1994.

Neitzert Semler, C. and Harvey, A. G. Daytime functioning in primary insomnia: does attentional focus contribute to real or perceived impairment? *Behav. Sleep Med.* (in press).

Nicassio, P. M., Mendlowitz, D. R., Fussell, J. J. and Petras, L. The phenomenology of the pre-sleep state: the development of the pre-sleep arousal scale. *Behav. Res. Ther*., 1985, 23: 263–271.

Ohman, A. and Soares, J. J. F. Unconscious anxiety: phobic responses to masked stimuli. *J. Abnorm. Psychol*., 1994, 103: 231–240.

Perlis, M. L., Merica, H., Smith, M. T. and Giles, D. E. Beta EEG in insomnia. *Sleep Med. Rev*., 2001, 5: 364–375.

Posner, M. I. Orienting of attention. *Q. J. Exp. Psychol*., 1980, 32: 3–25.

Rensink, R. A. Change detection. *Annu. Rev. Psychol*., 2002, 53: 245–277.

Sadeh, A., Hauri, P. J., Kripke, D. F. and Lavie, P. The role of actigraphy in the evaluation of sleep disorders. *Sleep*, 1995, 18: 288–302.

Shiffrin, R. M. and Schneider, W. Controlled and automatic human information processing II: perceptual learning, automatic attending, and a general theory. *Psychol. Rev*., 1977, 84: 127–190.

Simons, D. J. Current approaches to change blindness. *Vis. Cogn*., 2000, 7: 261–267.

Spielberger, C. D., Gorsuch, R. L., Lushene, R., Vagg, P. R. and Jacobs, G. A. *Stait-Trait Anxiety Inventory for Adults: Manual, Test and Scoring Key*. Mind Garden, Inc., Menlo Park, CA, 1983.

Tang, N. K. J., Harvey, A. G. and Schmidt, D. A. Sleeping with the enemy: clock monitoring in the maintenance of insomnia. *J. Behav. Ther. Exp. Psychiatry* (in press).

Taylor, L. M., Espie, C. A. and White, C. A. Attentional bias in people with acute versus persistent insomnia secondary to cancer. *Behav. Sleep Med*., 2003, 1: 200–212.

Van Someren, E. J. W., Swabb, D. F., Colenda, C. C., Cohen, W., McCall, W. V. and Rosenquist, B. Bright light therapy: improved sensitivity to its effects on rest-activity rhythms in Alzheimer patients by application of nonparametric methods. *Chronobiol. Int*., 1999, 16: 505–518.

Watts, F. N., Coyle, K. and East, M. P. The contribution of worry to insomnia. *Br. J. Clin. Psychol*., 1994, 33, 211–220.

Wicklow, A. and Espie, C. A. Intrusive thoughts and their relationship to actigraphic measurement of sleep: towards a cognitive model of insomnia. *Behav. Res. Ther*., 2000, 38: 679–693.

Williams, J. M. G., Watts, F. N., MacLeod, C. and Matthews, A. *Cognitive Psychology and Emotional Disorders*, 2nd edn. Wiley, Chichester, UK, 1997.

Sleep Medicine Reviews (2006) **10**, 215–245

ELSEVIER

SLEEP MEDICINE reviews

www.elsevier.com/locate/smrv

THEORETICAL REVIEW

The attention–intention–effort pathway in the development of psychophysiologic insomnia: A theoretical review

Colin A. Espie[a,*], Niall M. Broomfield[a], Kenneth M.A. MacMahon[b], Lauren M. Macphee[c], Lynne M. Taylor[d]

[a]*Sleep Research Laboratory, Section of Psychological Medicine, University of Glasgow, Southern General Hospital, Glasgow G51 4TF, Scotland, UK*
[b]*Department of Clinical Psychology, NHS Ayrshire and Arran, Scotland, UK*
[c]*Department of Psychology, University of Glasgow, Scotland, UK*
[d]*Department of Clinical Psychology, NHS Grampian, Scotland, UK*

KEYWORDS
Insomnia;
Sleep;
Psychological;
Model;
Cognitive;
Behavior;
Information-processing

Summary Psychophysiologic insomnia (PI) is the most common form of persistent primary insomnia. Its 'behavioral phenotype', comprising elements such as conditioned arousal, sleep-incompatible behavior and sleep preoccupation, has not changed markedly across several generations of diagnostic nosology. Moreover, a substantial outcome literature demonstrates that PI can be treated effectively using a range of psychological interventions. It seems evident that behavioral and cognitive factors play a part. What is less clear is exactly how PI develops and what are its crucial maintaining factors. This paper proposes an explanatory model, that we call the attention–intention–effort pathway. The argument is that sleep normalcy is a relatively automatic process. Consequently, it is vulnerable, and may be inhibited, by focused attention and by direct attempts to control its expression. Drawing upon parallels in the literature on adult psychopathology, and upon recent clinical and experimental studies on insomnia, the evidence for this pathway is considered and a research agenda is outlined. In particular, computerized tests of cognitive bias are seen as offering an objective means of appraising mental processes in insomnia. These may be applied concurrently with somatic measurements in future studies to better understand this common psycho-physiologic condition.

'Sleep (is like) a dove which has landed near one's hand and stays there as long as one does not pay any attention to it; if one attempts to grab it, it quickly flies away'

(Viktor E. Frankl (1965, p. 253): [Frankl VE. The Doctor and the soul. 2nd ed. New York: Knopf; 1965.] cited in Ansfield, Wegner and Bowser (1996) [Ansfield ME, Wegner DM, Bowser R. Ironic effects of sleep urgency. Behav Res Ther 1996;34:523–31.]

* Corresponding author. Tel.: +44 141 211 3903; fax: +44 141 357 4899.
E-mail address: c.espie@clinmed.gla.ac.uk (C.A. Espie).

1087-0792/$ - see front matter © 2006 Elsevier Ltd. All rights reserved.
doi:10.1016/j.smrv.2006.03.002

Introduction

In this paper, we propose a possible pathway for the development and maintenance of persistent psychophysiologic insomnia. Our thinking is guided by predictions from recently described insomnia models, by the relevant literature, including work undertaken in other disorders, and by recent experimental data.

Psychophysiologic insomnia

Psychophysiologic insomnia (PI) is the most common insomnia sub-type, found in 1–2% of the general population, and in 12–15% of all patients seen at sleep centers. According to clinical nosologies[3–5] and research diagnostic criteria,[6] the central pillars of PI are heightened arousal and learned sleep-preventing associations, with patients exhibiting an excessive focus upon and anxiety about sleep. A number of models has been proposed, each placing somewhat differing emphases upon these features of insomnia, nevertheless, all appear to endorse the validity of the PI phenotype[7] for a comprehensive review). Many studies evaluating psychological interventions have also been generated, and there is now substantial evidence that cognitive behavioral methods, either singly or within multi-component therapy, yield sustained reductions in insomnia complaints.[8–10]

In spite of this progress, there is much that remains to be done. Not least, there is an outstanding need to investigate the mechanisms and processes underlying the development of insomnia; and to establish what are the critical components of behavioral insomnia therapies and how they achieve their effects.[11] A timely call has been made to adopt experimental psychopathology paradigms for the purposes of testing and developing theories in relation to insomnia.[12] This approach has been fruitful in other areas of mental health research, most notably in anxiety and depressive disorders.

In recent times, we have established such an experimental program at the University of Glasgow Sleep Research Laboratory. The invitation to write this review arose from a presentation made on some of this work by the first author at the 17th Congress of the European Sleep Research Society in Prague, October, 2004. In this paper, we propose one candidate process, in still an early stage of conceptual development, that seems to merit further experimental and clinical research evaluation in PI.

The attention–intention–effort pathway

This idea has its origins in the psychobiological inhibition model of insomnia (Espie, 2002),[13] which differs from most other conceptualisations in that it takes as its starting point a perspective upon normalcy rather than pathology. The model considers what it takes to upset the course of normal good sleep, and to prevent (inhibit) its recovery. Lundh and Broman (2000)[14] similarly reflected on the importance in insomnia research of having "a sufficiently adequate understanding of how the (sleep) process typically unfolds normally" (p. 303).

It is of course known that prolonged wakefulness reliably induces sleep, and that failure to obtain at least a core amount of sleep (sleep deprivation) leads to impaired function. Within the 'two process' system (process S, sleep homeostatic drive, process C, circadian variation), the sleep homeostat drives the sleep–wake schedule toward a balanced requirement in that prolonged wakefulness accrues 'sleep debt', and the circadian timer modulates sleep propensity on approximately a 24 h cycle.[15,16] We suggest, however, that there is an implicit ancillary process that is associated with the automatic regulation of sleep–wake patterns in good sleepers. The concept of automaticity[13] refers to the largely involuntary nature of the well-adjusted sleep schedule, and to the over-learned associations that may form part of a good sleep stimulus control paradigm.[17] In other words, we see the good sleeper as essentially passive because internal and external cues act as automated setting conditions for sleep, and these are further reinforced by rapid sleep-onset. Endogenous cues to sleep, such as physical and mental fatigue, are presumed to interact reciprocally with exogenous perhaps classically conditioned cues, in the bedroom environment; so that the good sleeper approaches sleep, just as s/he walks or talks—without thinking much about it and without a consciously explicit plan.[13]

Just as homeostatic and circadian mechanisms play a central role in understanding some sleep symptoms (e.g. excessive daytime sleepiness, phase disorders), this 'third process' of automaticity may be central to PI. We hypothesise that because the sleep–wake process is essentially self-regulatory, de-arousal and sleep engagement may be particularly vulnerable if for any reason the process is switched out of its natural automated

mode. We have used the term inhibition for this switching,[13] for two reasons. First, our focus is upon factors that might be preventing the expression of normal sleep and preventing its natural recovery. Second, we do not assume that PI is associated causally with any particular sleep pathology.[a] Rather we are inclined to the view that people with PI have the potential to sleep normally if inhibitory factors can be overcome. In this context, Cognitive behavior therapy (CBT) methods may be effective in PI because they serve to overcome inhibitory mechanisms and to re-establish setting conditions for normal involuntary sleep. To the extent that any CBT method enables an individual to abandon personal agency over sleep and to return to reliance upon involuntary sleep it may be likely to achieve a good therapeutic effect.[13]

More specifically, we now propose that sleep-wake automaticity can be inhibited by selectively attending to sleep, by explicitly intending to sleep, and by introducing effort into the sleep engagement process. We call this route into PI the attention-intention-effort (A-I-E) pathway.

Selective attention

Introduction

We can go back as far as William James[18] for a definition of selective attention:

> "Everyone knows what attention is. It is the taking possession by the mind, in clear and vivid form, of one out of what seem several simultaneously possible objects or trains of thought. Focalisation, concentration of consciousness are of its essence. It implies withdrawal from some things in order to deal effectively with others."

Psychologists interested in information processing theory have researched human attention extensively. In short, we now know that stimuli that are salient to an individual are likely to attract attention. That is, there is an information processing bias toward salient stimuli. If you are engaged in the process of buying a new car of a certain model you will likely begin to notice other vehicles of that same type. It is not that more of these cars have appeared on the roads; but simply that you have developed an attention bias to something that has become a relevant stimulus.

Support has been found implicating attention bias in the perpetuation of a wide range of anxiety-related psychological disorders and concerns including panic disorder, hypochondriasis, eating disorders, obsessional disorders, generalized anxiety disorder, and PTSD[19,20] for reviews). In this field, attention bias toward potentially threatening stimuli has been of particular interest. Indeed, it has been argued that attention bias toward threat may have a causal role in anxiety disorders.[21] Evidence favouring attention bias in depression has been more equivocal,[22] with some studies demonstrating effects and some not. Some recent evidence, however, suggests that if depressed individuals do selectively attend to negative material it tends to be a more delayed, and possibly a more controlled, process than in anxiety disorders.[23]

The classic Beck cognitive model of emotional disorders assumes attention biases (as well as mnemonic and interpretive biases) are driven by negative beliefs stored in long-term memory. When activated these 'schema' guide information processing, including attention, toward stimuli congruent with them. Conviction in negative automatic thoughts is thereby increased, and hypervigilance is promoted. The anxious individual, therefore, remains preoccupied with danger and threat, and the depressed individual with failure and loss.[24] However, one of the limitations of schema theory, and the account of attention bias it offers, is that it views beliefs in a static rather than dynamic way by failing to acknowledge the top down influence of self-knowledge.[25] So-called 'metacognitive' models like the Self-Regulatory Executive Function model (S-REF)[26] account for cognitive self-regulation of attention, perception and memory, and, thus, may offer a more holistic, dynamic account. Psychological disorders are, according to S-REF, associated with a 'cognitive-attentional syndrome' that maintains attention focus on threat, promotes ruminative worry-based processing, and activates negative self-beliefs. Alternative processing routines are denied, adaptive self-knowledge is blocked and maladaptive self-knowledge is maintained. This S-REF approach is somewhat closer to our line of thinking about PI than 'classic' cognitive theory because S-REF helps to explain how intrusive, worrisome thought (and attention bias) may persist in insomnia.

It is very important to observe, however, that attention biases do not operate only in the context of threat. For example, expertise and personality have also been shown to mediate selective attention.[27,28] Likewise, in the illustration

[a] This is not to say that there is no neurobiological substrate to insomnia, but rather to suggest that such 'arousal' may be part of the expression of the A-I-E pathway itself.

introduced earlier, noticing more cars of a certain type on the roads would be unlikely to be motivated by threat. Rather, cognitive bias would probably be influenced by a more positive interest in making an eventual purchase! It is instructive, therefore, to look beyond the psychological disorders to consider how attention bias has been implicated in the perpetuation of habit/dependence disorders, including alcohol, heroin and nicotine. The stimuli that have been used in experiments on these disorders are clearly salient (related to the dependence) but if anything they tend to be reinforcers rather than threats. To take the example of alcohol, selective attention bias to behaviorally relevant word or picture stimuli has been found in alcoholics and problem drinkers, but not in social drinkers.[29,30] It seems that problem drinkers are more likely to notice alcohol-related stimuli in the environment, that this attention bias 'reminds' them of drinking, and that it may even mediate the maintenance of their addiction by producing 'craving'.[30] We will come back to ways in which both threat and incentive may be relevant to PI in conceptualization of selective attention in relation to PI.

Generally, an attention bias has been found in those who have a clinical diagnosis, contributing to the explanation of why anxiety disorders[31] and abuses/dependences[32] are frequently self-maintaining, and why relapse often occurs after an emergent treatment response. In explanations such as these, attention bias is conceived as an initial involuntary (unconscious, implicit) process that gives rise to voluntary (conscious, explicit) processes.[33] In other words, pre-attentive processing guides the early, automatic capture of relevant information even when conscious access to the information is not available. A good illustration of this was a study that successfully elicited both psychophysiological reactivity and subjective fear in phobic participants to pictures of their feared objects that were presented beyond their conscious awareness.[34]

The human attention system is clearly complex and intriguing. So what might happen to sleep if it became subject to such selective monitoring and scrutiny?

Conceptualization of selective attention in relation to PI

Attention biases reflect discrete changes in the direction of attention focus, in response to stimuli that are, in some sense, salient. Sleep is certainly salient to people with PI. Contemporary ICSD-2[4] diagnostic criteria for PI include:-

> "Excessive focus on and heightened anxiety about sleep" (Criterion C1)

The accompanying ICSD-2 text describes just how marked the preoccupation with sleep can become in PI:

> "Concerns about sleep grow progressively over months or years as sleep gradually deteriorates until the desire to obtain a good night's sleep becomes the person's major concern" (p. 1–6)

Interestingly, this statement conveys both a sense of incrementing distress associated with sleeplessness (cf. threat), and a preoccupying longing for sleep (cf. craving) that might serve as preconditions for attention bias. We have summarised in Table 1 what may be some of the key features of this incentive-threat comparison.

In 1943, Maslow published his influential paper on human motivation suggesting that a 'hierarchy of needs' act as motivators for human behavior.[35] According to Maslow's theory, basic needs are physiological; for example, hunger, thirst, sleep, etc. When these are satisfied they are replaced by safety needs reflecting the desire for protection against danger or deprivation. In this context, we can think of sleep as a primary reinforcer, reflecting basic physiological processes necessary for physical, intellectual and emotional well-being (Table 1). Thus, the A–I–E pathway in relation to sleep may parallel Maslow's commentary on hunger.

Table 1 Comparison of potential 'drivers' for sleep-related attention bias in Psychophysiologic Insomnia

Incentive	Threat
Sleep is a primary reinforcer	Inability to solve sleeplessness is threatening
Sleep is at top of the 'hierarchy of needs'	Safety needs come after primary physiological essentials
Sleep 'deprivation' produces craving	Sleep 'deprivation' produces worry
Hunger, thirst, oxygen as a model	Fear, anxiety as a model
Goal directed behavior is to obtain sleep	Goal directed behavior is to avoid being awake

> "For the man who is extremely and dangerously hungry, no other interest exists but food. He dreams food, he remembers food, he thinks about food, he emotes about food, he perceives only food and he wants only food (…) For our chronically and extremely hungry man, Utopia can be defined simply as a place where there is plenty of food. He tends to think that, if only he is guaranteed food for the rest of his life, he will be perfectly happy and will never want anything more. Life itself tends to be defined in terms of eating." (p. 374)

Similarly, we suggest that the person with PI experiences sleep disruption, sleep loss and perceived sleep inadequacy and so becomes atypically motivated by sleep, which is increasingly incentivised in proportion to the preoccupation associated with it. Just as food is more of a reinforcer when we are hungry, in PI we might expect that a much higher than normal value would be placed upon sleep. Of course, sleep would be a primary reinforcer for any individual, but presumably its reinforcement value might increase in relation to sleep requirement/deficit or perceived sleep requirement/deficit. The desire for sleep of good quality, therefore, may in this sense become a 'craving'.

However, consistent with the second level of Maslow's hierarchy, the perceived inability to sleep may also be conceptualised and experienced as a significant threat. Sleeplessness may be threatening. Bedroom arousal may develop in PI as a result of the conditioning of non-verbal (environmental) and verbal signals (e.g. thoughts about sleeplessness) as threat cues which impact on selective attention. But there is also another sense in which being unable to sleep might be experienced as a threat. Taking our principle of automaticity into account, people who sleep well do not usually know how they do so. Ask any normal sleeper what they do to sleep and they will probably appear rather bewildered. Sleep is not in this sense an enacted operant (cf.[17]), but rather it is passive and effortless. On the assumption that the person with PI started out as a normal sleeper, one can understand that to have apparently lost the capacity to sleep, not really knowing how you managed to sleep successfully before, might be rather threatening.

Harvey's model of insomnia[36] represents an adaptation of the cognitive perspective on psychological disorders, and finds common ground with the selective attention component of the A–I–E pathway. Harvey suggests that insomnia is maintained by a cascade of cognitive processes that includes selective attention and monitoring of the internal environment (e.g. alertness, bodily sensations) and external environment (e.g. clock-watching, environmental noise) that interact with negative beliefs, worry, misperception of sleep and the negative daytime sequelae of insomnia and the engagement of counterproductive 'safety behaviors'. Anxious people, for example, have been found to exhibit characteristic 'safety behaviors'.[37] These are overt and covert strategies that people develop in order to avoid feared outcomes. However, they generally prevent disconfirmation of catastrophic beliefs, and so in fact make feared outcomes more likely. For example, a social phobic fearful of spilling a drink in public, might grip the glass more tightly, thus (a) preventing unambiguous disconfirmation of the belief that spilling the drink is likely, and (b) increasing the likelihood of actually spilling. In insomnia, classic safety behaviors might include things like going to bed early or clock-watching. According to the Harvey model, increased monitoring for, or attention to sleep-related threat cues increases the chance of detecting such cues and thus establishes a mutually maintaining vicious cycle.[36]

So, it seems conceivable that selective attention to sleep-related cues might arise because of salience (unspecified), because of threat monitoring, because sleep is a reinforcer, or, of course, because of a combination of the latter reasons. Attention bias may be a signature of classically conditioned arousal in PI. In this respect, it would seem timely to re-consider the stimulus control model of insomnia in terms of classical/associative learning as well as its conceptualisation in terms of operant/instrumental learning.[17] It may also be worth considering which sleep parameters would be associated with different components of the A–I–E process. We suggest that increased sleep–onset latency (SOL) and wake time after sleep–onset (WASO), as the symptomatic representation of insomnia, could be indicative of conditioned arousal responses to intrinsic and extrinsic threat cues; whereas reduced sleep efficiency (SE) may reflect sleep craving through increased time in bed (TIB) (sleep opportunity) in the effort to increase total sleep time (TST). The contrast between good sleepers (GS) and those with insomnia is even clearer in the sub-group of GS who by choice mildly restrict their bedtime to 6–7 h/night on a fairly chronic basis. This applies to those busy people who accept the mild cost of some increased daytime tiredness for the choice of spending more time at work, with family, being entertained, etc. The attitude of these people towards sleep is not craving as the PI, nor even neutral as with the GS, but it may even be

somewhat negative and off-handed. For them, sleep may be seen as deserving relatively little attention.

Further relating attention bias to the concept of automaticity, we suggest that the good sleeper is like the experienced car driver who easily executes a complex series of operations with minimal attention load to the process. By comparison the person with PI is like the anxious learner driver—vigilant, deliberate and errorful.[13] The concept of automaticity in human learning has long been discussed as part of information-processing theory.[33] Some authors have suggested that the development of explicit, conscious processing is a relatively recent development in evolutionary terms.[38] Essentially, prior to humans gaining conscious thought, all learning would have flowed through an implicit acquisition process without the need for conscious, verbal reflection. Explicit, verbal learning may provide a means to 'short-cut' the development of some skills, such as driving; with the process of automatisation occurring as the skill is consolidated.

Paradoxically, it has been demonstrated, that proficient motor skills, may degrade if individuals are asked to verbalise their actions and turn their attention inwards towards the mechanics of their actions.[39,40] Perhaps, the most eloquent demonstration of this is paying attention to, and attempting to take conscious control of, the actions of your feet as you run downstairs. This is not to be recommended at the top of a flight of stairs. Thus, the development of sleep processes may be seen in much the same light. Setting conditions for sleep, responding to sleep cues, developing a sleep pattern are all part of infant training and should lead the development of sleep process that does not enter the realm of consciousness. However, if one encounters difficulties with sleep and attempts to 'take control' of the situation by directed attention, the disruption to the automaticity of the sleep process may parallel the difficulties encountered in consciously controlling one's legs whilst descending stairs. Respiration is another example of a similar phenomenon, although in this case there is no learned component. Respiration like sleep engagement, is normally an unconscious, passive, simple process. Yet, during a panic attack, thinking that not enough oxygen is being obtained induces hyperventilation that exacerbates the sensory dysphoria, light headedness, feelings of insufficient oxygen, which induces greater panic and a vicious cycle, at times leading to final unconsciousness.

Evidence of attention bias in PI

We have recently suggested that individuals with PI are characterised by high levels of metacognitive beliefs and plans for processing, which predispose them to appraise thoughts, experiences, and bodily states negatively.[41] This 'cognitive architecture', we hypothesise, promotes worry, rumination and attention bias in the pre-sleep period. We would predict, therefore, that metacognitive beliefs that promote negative appraisal of nocturnal intrusions (e.g. 'thinking at night keeps me awake') characterise the person with insomnia, as do associated metacognitive plans for processing, including those, which promote attention bias (e.g. to sleep, I must focus on how sleepy I feel).

Evidence of information processing bias in insomnia can be drawn from several sources, using differing methodologies. The most direct evidence comes from experimental studies specifically measuring or manipulating aspects of selective attention. We will review these studies after consideration of the less robust, but nevertheless interesting, descriptive literature comprising qualitative data, questionnaires and rating scales.

Phenomenological/descriptive studies

Many measures used in insomnia research contain items that reflect the person with insomnia's tendency to pre-occupying worry about sleep. These include the pre-sleep arousal scale (e.g. item 1 'worry about falling asleep'),[42] the dysfunctional beliefs and attitudes about sleep scale (DBAS) (e.g. item 4 'I am worried that if I go for 1 or 2 nights without sleep I may have a nervous breakdown'),[43] the sleep disturbance questionnaire (e.g. item 12 'I worry that I won't cope tomorrow if I don't sleep well')[44] the self-statement test: 60+ (e.g. item X 'if I don't get to sleep soon, I will feel very tired tomorrow')[45] and the anxiety and preoccupation about sleep questionnaire (e.g. item 1 'I worry about the amount of sleep I am going to get each night').[46] Interestingly, Watts et al. conducted a study comparing 'worried' and 'non-worried' insomniacs on pre-sleep mental activity.[47] The former group appeared preoccupied by work-related issues and general mental activity. In contrast, 'non-worried insomniacs' tended to focus on problems they were having with the sleep process itself. This direct focus on sleep is, of course, of particular relevance in relation to attention bias.

In a prospective study of pre-sleep mentation, Wicklow and Espie obtained voice-activated audiotape recordings of spontaneous thoughts, and sleep

actigraphic data from 21 poor sleepers over three consecutive nights.[48] Content analysis of over 1000 thought segments yielded eight categories of pre-sleep intrusion, and a regression model indicated that focusing on sleep and the anticipated consequences of poor sleep, along with general problem-solving were the strongest predictors of objective SOL. Thought content was subsumed under one of the three factors; 'active problem-solving' (e.g. rehearsing/planning events), 'present state monitoring' (e.g. thinking about-sleep/not sleeping, autonomic experiences, your own thinking) and 'environmental reactivity' (e.g. attending to external noises). Thirty-eight percent of thought segments represented present state monitoring.

The qualitative component of this study was partially replicated in a further investigation which also had a psychometric phase, leading to the development of the Glasgow content of thoughts inventory (GCTI).[49] The GCTI was found to have good internal consistency ($\alpha = 0.87$) and test–retest reliability (ICC = 0.88) and a score of 42 discriminated PI from GS groups with sensitivity of 100% and specificity of 83%. A principal components analysis of the GCTI found that present state monitoring emerged as an important factor accounting for 38% of explained variance.

Harvey conducted some parallel research.[50] Using a semi-structured interview, five areas of attention focus were investigated. It was found that people with insomnia relative to GS were more likely to attend to sensations of falling asleep and to worries/concerns, trying to solve problems and listening to noises. Good sleepers on the other hand were more likely to attend to 'nothing in particular' during their (relatively shorter) wake times. Neitzert-Semler and Harvey then reported two related studies. In the first of these, students meeting criteria for primary insomnia were compared with a GS control group using a semi-structured interview of sleep-related threat, negative thoughts, and safety behaviors.[51] People with insomnia reported more frequent monitoring, night and day, and they engaged in more safety behaviors. A path analysis suggested that monitoring may act as a driver for negative thinking in insomnia. In the second study reported in this paper some evidence emerged for the generalizability of these findings to a clinical insomnia sample.

This work was extended through the development of the sleep associated monitoring index (SAMI).[52] This 30-item scale of sleep-related threat monitoring shows good reliability ($\alpha = 0.87$) and positive correlation with the Pittsburgh sleep quality index (PSQI[132]). Importantly, moderate correlation ($r = 0.36$) with the Penn state worry questionnaire suggests that the SAMI score is not simply an index of generic aspects of worry. An eight component solution was obtained following principal components analysis on a large sample ($n = 400$) of university students and staff. These components included monitoring for body sensations (daytime, pre-sleep, and on waking, each loaded as separate components), clock time and the environment. This study also explored the relationship between the SAMI (monitoring) and two other constructs used in attentional theory. Both 'amplification' (the tendency to experience somatic sensation intensely) and 'self-focus' (awareness of internally generated information) correlated with the majority of the SAMI subscales, excepting calculation of time and pre-sleep clock monitoring.

Summarizing this work, it seems that questionnaire data are broadly supportive of the notion of attention bias in PI. However, two notes of caution seem appropriate.

First, mental arousal associated with sleep may not be as crucial to the conceptualisation of PI as some of the psychological theories suggest. In a recent comparison of PI, delayed sleep phase syndrome (DSPS) and GS, we found that several self-report measures of the construct of cognitive arousal were elevated in both PI and DSPS, relative to GS.[53] This raises the possibility that such arousal may be epiphenomenal to wakefulness in PI (rather than causal). DSPS is presumed to reflect an endogenous phase delay so there is no need to infer any psychological process. On the other hand, the mechanisms that trigger DSPS are often precipitated by life or social events and the effects of DSPS may lead to increased pre-sleep arousal when individuals with DSPS try to reset their clocks by attempting to sleep 'out of phase'. This could theoretically precipitate symptoms of, or the onset of PI. Conversely, when individuals with PI are unable to fall asleep, they may inadvertently entrain their sleep to a later time, resulting in an element of DSPS.[54] Of course, worry and rumination are transdiagnostic phenomena that present across a range of psychiatric disorders.[55] Also in many medical disorders, worry is an associated feature. What the research literature needs to determine is which features of cognitive arousal are directly involved in the genesis and maintenance of persistent insomnia.

Second, the studies reviewed so far were not designed specifically to test attention bias within the context of a controlled experiment. However, a number of such studies has emerged recently,

largely from Dr Allison Harvey's research group in Oxford[b] and from our own laboratory in Glasgow.

Real world experiments

Harvey's innovative work stems from her cognitive model of insomnia in which the importance of monitoring of sleep-related threat was posited (outlined above). This research team has conducted a series of controlled experiments, involving the manipulation of attention in order to test its causal role in increasing or decreasing insomnia symptoms.

Neitzert-Semler and Harvey[56] attempted to test the hypothesis that monitoring for sleep-related threat during the day would trigger a cycle of subsequent negative thinking, perceived impairment, and subjective sleepiness. Young people with insomnia were randomly assigned either to a condition involving monitoring of body sensation (to be closely aware of the feelings and sensations and to focus attention on internal reactions), to a condition involving distraction from such monitoring (focusing upon external environment and activities) or to a no instruction control group. Results largely confirmed the prediction that the monitoring group would report higher ratings for negative thoughts, safety behaviors and daytime sleepiness than the control condition. Daytime functioning, however, was not different between groups and, unfortunately, the no monitoring manipulation was less well designed and so was relatively unsuccessful. Nevertheless, this does not detract from the findings for the monitoring group.

Neitzert-Semler and Harvey[57] assigned 51 participants meeting DSM-IV criteria for insomnia to a self-focus group (viewing themselves on a TV monitor), to a monitoring group (similar to above but also focusing on thoughts and mood) or to a no instruction group. Participants were then exposed to a 60-min neuropsychological test battery. The purpose of the study was to index the effect of attentional focus on real versus perceived performance. As hypothesized, no differences were observed in the former comparison. However, the self-focus group perceived their performance as significantly worse on the majority of tasks than the no instruction group, providing confirmation of the potential role of self-focusing as a contributory factor to the perceived daytime impairments of people with primary insomnia. By contrast, the monitoring condition did not differ from the no instruction group on any subjective performance rating. The authors suggest that the self-monitoring condition, unlike the video-TV condition, in this experiment may have resulted in insufficient self-focused attention.

Tang et al.[58] considered the importance of clock monitoring in insomnia. In a first experiment, good and poor sleepers were instructed to monitor (or not) a clock as they were trying to get to sleep. Clock monitors, whether poor or good sleepers, reported a higher worry rating and had longer SOL as indexed by sleep diary and actigraphic data. A second experiment was conducted with a clinical sample, where monitoring per se was controlled by using a digit display monitoring task to isolate the specific effects of clock watching. The degree of worry and SOL overestimation demonstrated by the clock monitoring group was greater than the control condition, thus, lending further support to the idea that attentional bias, in the form of clock watching, is not conducive to sleep.

To summarise these real world experiments, it seems that insomnia is associated, at least in terms of self-report, with sleep-related self-monitoring tendencies.

One of the challenges for research in the area of attention is to separate out sleep-specific effects from heightened generic responding. For example, research on the 'orienting response', indexed physiologically by skin conductance levels, suggests that people with insomnia exhibit a general tendency to increased attentiveness. That is they are more responsive, particularly to emotional stimuli and stress, and take longer to habituate than do good sleepers.[59–61] Although, so far, psychophysiological measures have not been taken in parallel, studies employing reaction time as a dependent variable have now gone some way towards establishing responses to sleep stimuli over and above heightened orienting per se.

Computerized experimental studies

Several studies have explored selective attention bias in insomnia using measures of information processing speed (Table 2). These studies used computerized experimental protocols where both salient and neutral stimuli were presented to investigate any systematic processing differences between good sleepers and people with sleep disorder. The paradigm in such attentional tasks is that stimulus salience interferes with response time because of the 'grabbiness' (uncontrolled prolonged attention capture or focusing) of sleep-related word or picture stimuli relative to neutral stimuli. An advantage of this experimental approach is that it does not rely on self-report, but rather posits objective reaction time differentials as a proxy for cognitive arousal. Before

[b] Now at Sleep and Psychological Disorders Laboratory, University of California, Berkeley.

Table 2 Experimental studies investigating attention bias in insomnia using information processing paradigms

Authors	Participant characteristics	Paradigm	Findings
Lundh et al.[68]	40 adults between ages of 20 and 65 and gender matched across two experimental groups (primary insomnia and good sleepers)	Stroop task	Repeated measures ANOVA examined the effects for each of the 3 stimulus types [i.e. sleep, physical threat and physical control words; and colour names versus a control sequence of letters (XXX…)]. Both the insomniacs and the controls responded more slowly to the sleep words, physical threat words and colour names, than to their matched control stimuli. There was no main effect of group with regard to any stimulus type and no significant interaction between group and sleep words [$F(1, 37)=0.21$)].
Taylor et al.[69]	33 Adults (23 F/10 M; mean age 47 years) with sleep-onset insomnia subsequent to cancer diagnosis. Mean time since diagnosis for the acute insomnia group was 2.0 months and was 14.3 months for the persistent insomnia group	Stroop task	Independent samples *t*-tests indicated no significant difference between the two groups for the cancer interference index ($t=0.91$, $p=0.37$) but there was a significant difference on the sleep interference index ($t=-2.44$, $p=0.02$). Both groups demonstrated interference for cancer words relative to neutral words, but only the persistent insomnia group exhibited interference for sleep words. Groups did not differ significantly on pre-sleep cognitive or somatic arousal (PSAS) and they used similar thought control strategies (TCQ: distraction, re-appraisal, social control)
MacMahon et al.[70]	63 adults (35 F/ 28 M; mean age 25 years). Across three experimental groups (primary insomnia (PI), delayed sleep phase syndrome (DSPS), good sleeper (GS)).	Dot probe	Orthogonal contrasts of PI versus DSPS and GS indicated a significant difference ($t=-1.88$, $p=0.03$), suggesting that participants with PI showed a greater attention bias to sleep related words than those with DSPS or GSs. A further contrast between DSPS and GS did not indicate a significant difference between these groups ($t=-1.27$, $p=0.10$), thereby supporting the hypothesis that attention bias plays a fundamental role in the disorder of PI. The possibility of an underlying trend in DSPS responses needs to be further investigated
Jones et al.[72]	192 Adults (50% F; mean age 32.1 years) age and gender matched adults across three experimental groups (poor sleepers, moderate sleepers good sleepers)	Flicker ICB	Independent samples *t*-tests revealed that Poor sleepers and Moderate sleepers detected the sleep-related change significantly quicker than Good Sleepers ($t=$ 3.33 and 2.90, both $p<0.01$). There was no difference in this change detection latency between Poor and Moderate sleepers. Poor sleepers detected the sleep-related change quicker than the neutral change [$F(1180)=7.11$, $p<0.01$] displaying a sleep-related attention bias. For Moderate sleepers this difference was not significant, and Good sleepers detected the change within the neutral objects significantly quicker than within the sleep-related objects [$F(1180)=6.21$, $p<.05$] showing a bias towards neutral rather than sleep-related objects

(*continued on next page*)

Table 2 *(continued)*

Authors	Participant characteristics	Paradigm	Findings
Macphee et al.[73]	90 Adults (50% F; mean age 22.8 years) across three experimental groups [(primary insomnia (PI), delayed sleep phase syndrome (DSPS), good sleeper (GS)]	Flicker ICB	Independent samples *t*-tests revealed that, at the level of PI, sleep-related change was detected significantly quicker than a sleep neutral change, ($t=13.10$, $p<0.0001$). No such differences were observed at the level of GS ($p=0.95$), or DSPS ($p=0.14$). At the level of sleep-related change, responses of PI were significantly quicker than GS and DSPS ($t=7.50$ and 4.80, both $p<0.0001$) and responses of DSPS were quicker than those of GS, ($t=2.66$, $p<0.01$). At the level of neutral change, responses of GS and DSPS were significantly quicker than PI ($t=5.70$ and 6.80, both $p<0.0001$). No difference was observed between GS and DSPS

reviewing the literature on the application of these experiments in insomnia research it may be helpful to describe the tasks themselves.

Attention bias tasks. Three different methods have been applied to the study of insomnia.

First, the emotional Stroop task, which has been described as the hallmark measure of attention,[62] has been used to assess selective attention bias in a wide range of conditions. The Stroop task involves target (salient) and control (neutral) words being presented at random in different ink colours. Subjects are asked to respond quickly to the presented colour by pressing the corresponding coloured button on a response box. They are instructed to ignore the actual meaning of the words. Response latencies for colour identification are automatically recorded for each stimulus. Longer response latency is thought to suggest increased attention bias because automatic processing of word meaning for the salient words is likely to interfere with (slow down) colour naming relative to response time for the neutral words: the so-called interference effect.

There has been debate in the literature over whether the Stroop task actually measures increased vigilance or simply reflects the impact of heightened arousal interfering with information processing when salient stimuli are presented.[63] Thus, Stroop data alone may be insufficient to conclude that attention bias is present in PI. A second test of cognitive bias toward semantic material, the dot-probe task, has been posited as one solution to this problem.[63] In this task, words are simultaneously presented (typically for 500 ms) to two areas on a computer screen. The ensuing distribution of visual attention is measured by recording detection latency for a visual probe that could appear in the spatial location of either word, immediately after the display of that word has terminated. Thus, the task bypasses limitations of the Stroop, by using a neutral response (a keypress) to a neutral stimulus (a 'dot'). The trials providing the data of interest are those in which one of the words is salient. By examining the impact of sets of such words on the relative probe detection latencies in the two spatial areas, it is possible to determine whether visual attention has shifted toward or away from such stimuli.

There are, however, some limitations to research on attention bias that uses word stimuli. As Yiend and Mathews[64] and others have pointed out, although words can be unequivocally negative in valence it is far from clear that they constitute a severe or highly salient threat. For this reason attention bias studies investigating state and trait

anxiety sometimes use picture stimuli (e.g. weapons, corpses, dangerous animals) that are known to evoke both subjective and physiological reactions.[65] Pictures of this kind are fairly generic threatening stimuli, but ones that nonetheless elicit greater attentional responses in anxious individuals. In the context of insomnia, it is somewhat difficult to represent sleeplessness through objects in this graphic way. Besides, we are not committed at this point to an explanation for attention bias in insomnia that is motivated solely through threat monitoring.

Nevertheless, it is possible to explore attention bias with digitised objects using a flicker paradigm featuring a perceptual phenomenon called induced change blindness (ICB).[66,67] Research using this third method reveals that when a change is made to a visual scene (and the process of change is hidden from view), it is more difficult to detect than might be expected. Normally in this paradigm, a single feature of a visual scene is changed between successively repeated brief presentations until the change is detected—essentially the ICB is a spot the difference task. Change-detection latency, measured by the number of flickers it takes for the change to be identified, is explained by a change's 'grabbiness' and this depends not just on the object's physical feature that carry the change but also on the viewer's history in relation to that object. So, for example, in the alcohol field, problem drinkers take fewer flickers to detect an alcohol-related change within the visual array than a neutral change and are faster to detect such changes than control subjects.

Experiments using computerized tasks

The first study to be published was that of Lundh, Froding, Gyllenhammar et al.[68] This was a pioneering piece of work because it translated the emotional Stroop task into the insomnia field. Lundh et al. found that people with insomnia had prolonged response latency for sleep-related words (Table 2). However, this effect was also evident in a control population of good sleepers, and there was no group difference on the Stroop interference index; a result inconsistent with the attention bias hypothesis. Lundh et al. suggested that sleep-related words might have emotional valence for people that may or may not be directly related to sleep problems. However, the extensive literature on the Stroop task would not predict experimental effects in normal control groups. Of course, recruited controls who are good sleepers may have a particular interest in sleep, and this might yield a bias. Also, in this study no measure of affective state (which is known to influence Stroop findings) was taken, and diagnostic criteria were not reported for the insomnia group.

Lundh et al.'s pioneering work in Sweden, therefore, yielded somewhat equivocal findings. However, since then, our research group in Glasgow has completed four experiments using each of the three attention bias paradigms described above (Stroop, dot probe, Induced Change Blindness) that generally indicate the presence of selective attention bias in insomnia.

In our first study, we also used the Stroop paradigm, selecting a cancer population because our primary purpose was to investigate the development of insomnia associated with a stressor in people, who had previously been good sleepers.[69] None of the participants had insomnia prior to their cancer diagnosis; that is they were a 'true' secondary insomnia population rather than people whose (pre-existing) insomnia had been exacerbated. Insomnia is common in cancer populations, so this seemed to be a valid population group to study. Two groups of people with cancer and insomnia, 0–3 months and 12–18 months after cancer diagnosis, completed the computerized emotional Stroop task comprising cancer-related, sleep-related and neutral word cues. Both groups demonstrated attention bias for cancer-related words but only the persistent insomnia group demonstrated attention bias for sleep-related words (Table 2). The fact that interference effects for sleep words were absent at 0–3 months but were evident at 12–18 months, suggests that selective attention bias towards sleep may play a role in the transition from adjustment insomnia to psychophysiological insomnia.

According to ICSD-2[4] the essential feature of Adjustment Insomnia is

> "…the presence of insomnia in association with an identifiable stressor. The sleep disturbance of Adjustment Insomnia has a relatively short duration, typically a few days to a few weeks" (p. 1–3)

In Fig. 1 we have illustrated how persistent psychophysiologic insomnia may evolve from Adjustment Insomnia following the experience of a series of stressors (such as illness). It is assumed that experiencing stress is associated with both psychological (mental, behavioral, emotional, etc.) and physiological (autonomic, cortical, metabolic, etc.) responses. These are likely to inhibit normal sleep-related de-arousal, and so produce transient sleep disturbance. If cognitive and physiological arousal becomes sustained, insomnia may persist as a symptom. If not, then the insomnia symptoms would dissipate and normal sleep would return. Our

assumption, consistent with the Taylor et al. data,[69] is that attention bias (whether implicit or explicit) during Adjustment Insomnia is selective towards the perceived source of the stress. Indeed, because of that selectivity, the insomnia symptoms per se are unlikely to grab attention whilst the stressor is active.

However, continuing the description of Adjustment Insomnia from ICSD-2:

> "…the sleep disturbance resolves, or is expected to resolve, when the specific stressor resolves, or when the individual adapts to the stressor" (p. 1–3)

Consistent with this statement, we suggest that, close to the point of normal resolution of the adjustment insomnia, selective attention upon the stressor might reduce markedly. However, in circumstances where insomnia symptoms still persist, there could be an increased risk that an attention bias towards sleep-related cues might develop. That is, attention might shift from the resolving stressor to any persisting sleep disturbance at this point. Furthermore, the transition to sleep-related implicit attention bias could be prepared by the frequent prior conditioning of sleep cues with sleeplessness during preceding weeks. Thus we may have the start of the A–I–E pathway as a self-perpetuating persistent PI, even when the original stressors have resolved or diminished.

The Taylor et al. study, however, was of a cross-sectional rather than a longitudinal design and we did not have a control group of good sleepers without medical problems. Moreover, the paradigm employed presented word stimuli for the standard supraliminal 500 ms duration. Thus it is not possible to determine to what extent the bias was pre-attentive/automatic, i.e. occurred involuntarily without intention or conscious control. The results, therefore, need to be interpreted with some caution.

We have recently completed another attention bias experiment study using the dot-probe task.[70] Sixty-three young adults across three experimental groups (PI, DSPS, GS) participated (Table 2). PI and DSPS participants met ICSD criteria for their respective disorders following extensive assessment comprising clinical interviews, the use of self-report scales, and sleep diary and actigraphy monitoring. The DSPS group was employed as a further, clinical, control sample of people who, like our PI participants, had sleep-onset problems, but whom we would not expect to exhibit cognitive arousal as an explanatory mechanism for their continued wakefulness. Rather, circadian factors are presumed to explain the emergence and maintenance of DSPS. Consequently, those with

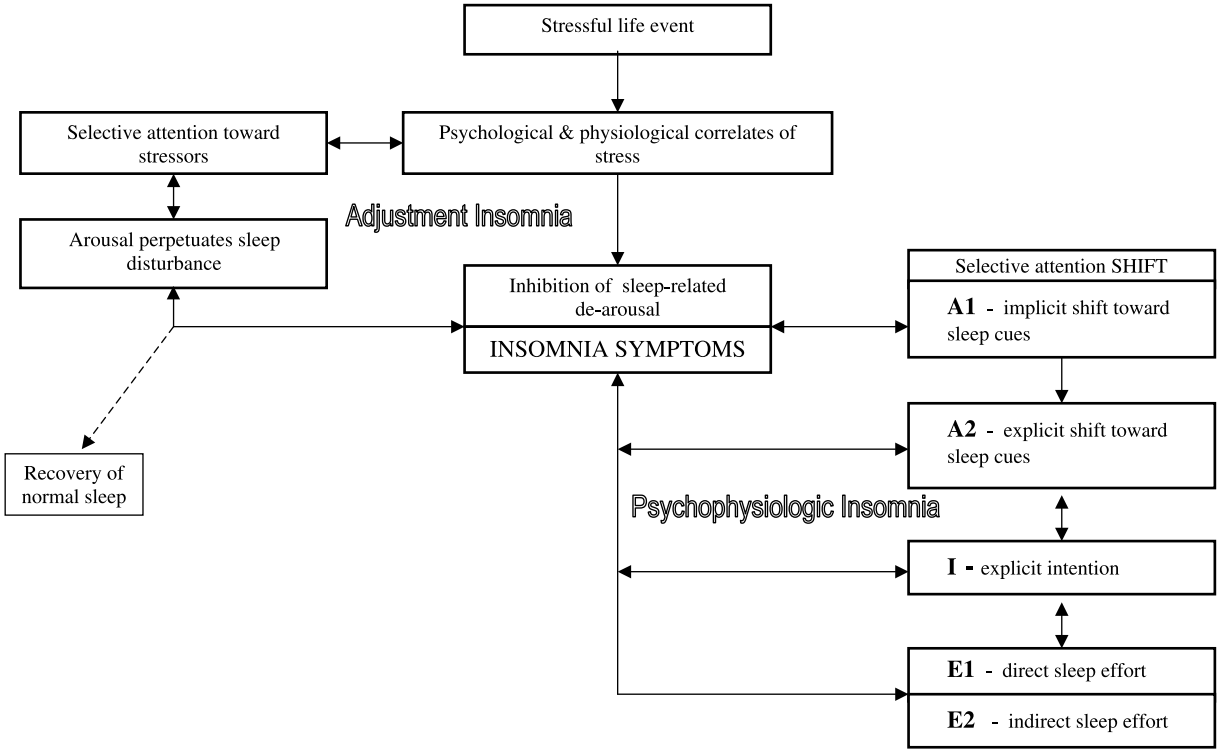


Figure 1 Proposed evolution of psychophysiologic insomnia from adjustment insomnia following the A–I–E pathway.

DSPS would not be predicted to show a cognitive processing bias to sleep-related stimuli. Results supported our predictions, with those in the PI group showing a significantly greater processing bias toward sleep-related words (in comparison to neutral words) when compared to the GS and DSPS groups (Table 2). Notably, the GS and DSPS groups did not differ from each other, suggesting that the underpinning mechanism maintaining DSPS is not an attention bias to sleep-related stimuli.

Finally, we have conducted two experiments using the ICB task. Interest in the control that sleep-related objects might have over sleep behavior is long-established. For example, within a conditioning framework, bedroom environment objects might become discriminative stimuli for sleep,[17] but when the bedroom-sleep contingencies are broken, they might become discriminative stimulus for wakefulness. In that regard it is interesting that less than one-quarter of the sleep-related words in our Stroop and dot probe studies were objects. Consequently, we felt that the ICB paradigm may be better suited to investigating the possible influence of the bedroom environment on sleep. We were also interested in the fact that, using this technique, a differential attention bias between two 'levels' of social use of alcohol and cannabis has been found.[71] In the insomnia context, we wanted to extend this approach to explore differential attention bias along the sleep problems continuum. If attention is implicated in the development of persistent insomnia we might expect to find a systematically changing attention bias, not just at the clinical pole.

In our first ICB study, 192 participants (mean age 32 years) were selected for this totally between subjects experiment[72] Table 2). Participants completed the 15-min ICB task, after which they were assessed for sleep quality and other characteristics. Importantly, therefore, retrospective group assignment was blind to the dependent variable of the analyses, change detection latency. A different flicker pair of stimuli was used for each of the two levels of the factor, nature of change (sleep-related and neutral). Each pair contained the same original stimulus comprising seven sleep-related objects and an equal number of neutral objects arranged in two collections on either side of the scene midline. The second stimulus of each pair was identical to the original stimulus but for one small change: a sleep-related change (removing one of the pair of slippers) or a neutral change (removing one of the pair of gloves). The two changed stimuli are shown in Fig. 2 along with their common originating stimulus. The two stimuli of a pair were then presented in continuous succession (each replacing the other) until the change was detected. A brief 'mask' was inserted in between the flicker pairs to suppress visual transients. We selected the sleep stimuli using a comprehensive process designed to identify objects associated with 'going to bed to sleep'. Clearly none of the objects is intrinsically threatening, and slippers emerged with the highest mean sleep-relatedness score.

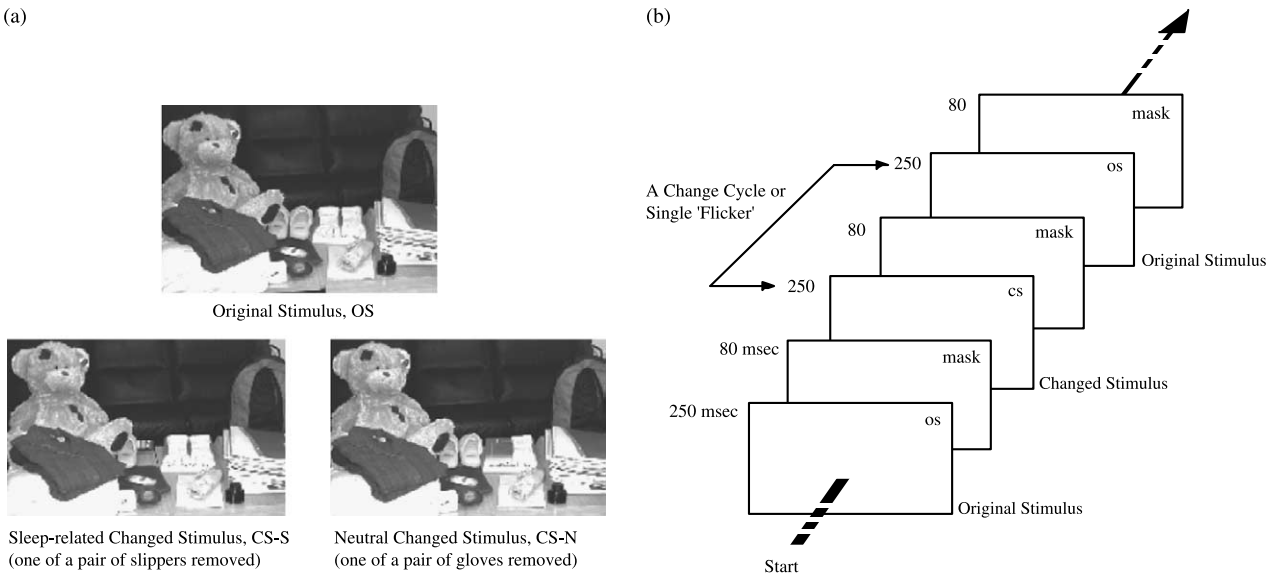


Figure 2 (a) Greyscale versions of the full color stimuli used in ICB experiment [72] Original stimulus (OS) and the two changed stimuli for each of the two levels of the factor, nature of change-sleep-related change (CS-S) and neutral change (CS-N) and (b) a Flicker paradigm for inducing change blindness illustrating a change cycle or single 'Flicker'.

Results revealed significant differences in change detection latency between poor, moderate and GS for the sleep-related change. Only the poor sleepers, who detected sleep-related change quicker than neutral change, demonstrated selective attention bias for sleep salient stimuli. Moderate sleepers showed a trend in the same direction. By contrast, GS detected the change with the neutral objects significantly quicker. Hierarchical regression was then applied to test the relationship between change detection latency and a continuous representation of the global PSQI score. This evidenced a systematically changing effect of sleep quality upon attention bias, independent of age, gender and depressive symptom level.

In simple terms, when competing for attentional resources with matched neutral stimuli, poor sleepers appeared to prioritise sleep-related stimuli. The findings for GS (of relative prioritisation of neutral stimuli) may be explained by differences in the physical saliencies of all the stimuli in the scene. That is, the neutral half of the scene may have been more salient in general, or may have included one highly salient single item, as well as relative positional and configurational aspects. Because all sleep quality groups were presented with the same complex scene, we suggest that an attentional force that is greater than existing physical saliencies is likely to have driven the responses of poor sleepers.

We have recently completed a second ICB experiment to replicate and extend the above work.[73] In this study, we improved diagnostic methods by including a clinical interview and actigraphy in the protocol, and strengthened the primary analysis by incorporating DSPS as a clinical control group. In this experiment we used different change stimuli; respectively, a teddy bear and a mug, to rule out the possibility of idiosyncratic effects to previously used stimuli. A 2 (experimental condition) by 3 (group) between-participants design was employed. Participants ($n=90$) were within one of three groups (PI, DSPS, GS) and each sleep quality group was consequently split in half at random to receive the sleep-related change, or a sleep-neutral. Group allocation was not fully known to the experimenter until the ICB experiment was completed. As predicted, the stimulus change/sleep quality interaction was significant and PI detected the sleep-related change significantly quicker than the sleep-neutral change. No such difference was observed between the sleep-related and sleep-neutral changes for either DSPS or GS participants (Table 2). Post hoc testing also revealed that, for the sleep-related change, responses of PI were significantly quicker than GS and DSPS, and that responses of DSPS were significantly quicker than those of GS. By comparison, for the neutral change, responses of GS and DSPS were significantly quicker than PI and no difference was observed between GS and DSPS.

The results of this experiment provide further evidence of attention biases to sleep-related stimuli in insomniacs. However, unlike our dot probe study[70], we also found that DSPS participants detected a sleep-related change significantly quicker than GS. We propose several possible explanations for this effect. First, DSPS, particularly in younger people, may comprise two distinct sub-groups, a socially driven DSPS and an inherent/genetic DSPS, whose responses to attentional measures may differ. Second, results may depend on whether DSPS participants were sleeping in phase or out of phase at the time of the experiment. In the latter case one might expect more insomnia symptoms. Third, and as we previously discussed[54], suggest that PI and DSPS may often share overlap symptoms. Sleep-onset PI may facilitate phase delay, and phase delay may contribute to anxiety and preoccupation about sleep initiation.

To date then, our work has shown that attention bias presents in individuals with persistent PI. The extent to which such biases are maintaining of PI, or merely epiphenomenal, remains unclear, as does whether attention bias can predispose to PI. Although some of our data suggest that attention may shift from stressor to sleep as adjustment insomnia becomes chronic, longitudinal work will be needed to confirm this. Sleep-related attention bias may be a common factor in all sleep disorders or it may help to differentiate insomnia subtypes, e.g. psychophysiologic insomnia from insomnia due to mental disorder. Again further work is needed on this.

Evoked potential studies

Two recent insomnia studies employing event-related potential (ERP) measurement of cortical arousal also merit consideration. Devoto et al.[130,131] have shown, in participants with PI, that cortical hyperarousal is not constant in poor sleepers. Rather it fluctuates depending on the quality of the previous night's sleep. Variation in P300 amplitude is thought to reflect neuroelectric activity related to cognitive processes such as attention allocation and immediate memory; larger P300 amplitudes being associated with poorer nights of sleep. These data, therefore, are important because they further evidence an association between disturbed sleep and attention processes. They suggest that P300

measurement may be of potential benefit in the further study of attention bias. Would, for example, biased attention to sleep cues in a Stroop paradigm map onto enhanced ERP activity? There is already psychophysiological evidence to this effect, on attention to emotion cues.[29]

In summary to this point, we suggest that there is a developing body of evidence that supports the proposition that sleep-related attention bias may be implicated in the development and persistence of PI. This evidence comes from both qualitative and quantitative studies. Recent experimental data from studies manipulating attentional variables, and from studies investigating information processing speed and evoked potentials, offer the most direct evidence and such data are consistent with clinical impressions of sleep preoccupation, and possibly of sleep-related conditioning, in PI.

Explicit intention

Introduction

We propose that the next stage of the inhibitory A–I–E pathway is that of developing an explicit intention to sleep. That is, an attention-for-action mechanism associates attentional focus upon sleep and sleeplessness, with intentive actions designed to deliver sleep and to eliminate wakefulness (Fig. 1). We will argue that these intentions are antithetical to the behavior of good sleepers, as they are with those seeking skilled performance in other domains,[39,40] that they further inhibit the automaticity of normal sleep (beyond the effects of attention/monitoring alone), and that, because they are usually ineffective, they lead to the engagement of sleep effort.

So far we have considered the process of attention. Now it seems helpful to consider its purpose. Why is selective attention so important? There seem to be two, related answers to this question. One is that attention has to be selective because the brain is a limited capacity processor. The other is that selectivity actually confers distinct advantages, irrespective of capacity.

At any given time, only a small proportion of information available in the environment can be selected and identified for conscious processing. Optimally, this selection should be based on the information necessary for the execution of current and planned behavior. Thus, it is adaptive to focus upon a threat, so that you can take avoidant action. Likewise, it is adaptive to focus upon an unmet need (to crave), so that you can develop a plan to meet that need. It is less urgent and less important to focus elsewhere.

Allport[74] pointed out that the majority of research in this area considered the limited information-processing capacity of the brain as the fundamental constraint underlying all operations of attention. Thus the selection function of attention arises necessarily from the notion of limited capacity. However, diverging from the idea that attention operates primarily as a mechanism for coping with central limited capacities of cognitive processing,[75,76] Allport also emphasized constraints in preparation and control of action. The idea behind this attention-for-action perspective is that integrated actions require the selection of particular aspects or attributes from the environment that are relevant to the action at hand. At the same time, any information irrelevant to the action should be ignored. Thus, attentional processes may be seen as the selection of action-relevant events or stimuli relying on particular action plans. This echoes the concluding phrase of James'[18] definition, presented earlier, that

> "(selective attention) … implies withdrawal from some things in order to deal effectively with others" (present authors' emphasis).

Likewise, Posner[77] reflects upon an early metaphor that:

"Thinking, like swinging a bat, has a "point of no return"—once committed in a particular direction, thought is ballistic in that it cannot be altered." (p. 3)

It seems then that specific aspects of the environment are overtly and covertly selected and become integrated in goal-directed action planning. This is what we mean by 'intention'. We attend so that we can intend. For the majority of human behavior this works well as an active process. Focusing and directed, purposive behavior is helpful, and it generally improves performance. However, there are certain circumstances where explicit intention is counter-productive. These arise, not least, with bodily functions that are designed to operate automatically.

A good example of this is the human sexual response. Erectile responsivity is not essentially purposive, and it can be inhibited by attention and intention, particularly when fuelled by anxiety or worry about performance failure (threat) or by desire to respond normally (incentive). There is a considerable literature demonstrating that the human sexual response is undermined by self-monitoring and self-observation, and that it is promoted by focusing away from, rather than directly upon, physiological 'performance'.[78,79]

Techniques such as 'sensate focus' have been applied successfully for 30 years or more to reduce this maladaptive self-referential monitoring of sexual response. Using this approach involves focusing upon (selectively attending to) sensory experiences (of touch) and away from overtly sexual experiences (sexual arousal). The explicit intention of responding sexually is, thereby, removed, permitting arousal to re-emerge spontaneously.

There is a long tradition of psychological theory and practice, dating back to the work of Victor Frankl[1] involving techniques such as paradox and dereflection.[80,81] These have been applied to situations where there is excessive concern about the frequency of a response occurring too often (e.g. blushing, excessive sweating) or not often enough (e.g. sexual response). This literature illustrates that attending to, and intending to produce, a desired behavioral goal can be inhibiting; and that, in turn, that by instructing patients to intend the opposite, the original desired goal may be achieved more easily.

Conceptualisation of explicit intention in relation to insomnia

To re-iterate, we propose that a dysfunctional explicit intention to sleep develops in the context of an attention-for-action mechanism that associates sleep/sleeplessness with behavior specifically designed to deliver sleep/eliminate wakefulness (Fig. 1). Thus, the automaticity of normal sleep initiation is further challenged by the emergence of a specific purpose-to sleep. Again it is instructive to think of sleep normalcy.

We suggest that normal sleepers do not exhibit a well-developed explicit intention to sleep. Rather their intentions may be more implicit than explicit. That is, it is implicit in going to bed, putting the light out, adjusting body position and so on that the purpose is to sleep. We have previously referred to these as automated setting conditions. Such preparatory behaviors certainly reflect an implicit intention—they are not indifferent to or neglectful of sleep—but they are not done with the express purpose of sleep. Indeed, exception to the general principle that good sleepers lack intentive purpose associated with bedtime behavior, may arise when their explicit intention is, in fact, to remain awake. The notion of 'abandonment of wakefulness' seems more apposite to normal good sleep.

Take the example of reading in bed. Good sleepers read in bed, so why should we discourage people with insomnia from reading in bed, as per stimulus control instructions?[17] Is reading in bed a sleep-incompatible behavior, or is it not? This may, of course, depend more upon the nature of conditioned behavioral associations, than upon the behaviors per se. However, another explanation of the essential difference between good sleepers and people with insomnia in relation to reading, is that good sleepers are more likely to have the explicit intention of remaining awake, in order to read; whereas people with insomnia are more likely to read, with the explicit intention of falling asleep. Therefore, the situation may arise that the good sleeper gets to the point of quitting reading in order to sleep, simply because they are already lapsing into micro-sleeps from time to time. Indeed, they may fight sleep off for a while before giving in (passively) to sleep. By comparison the person with insomnia is more likely to be filling wakeful time, hoping that this (or another) strategy might work to help them sleep. The contrast, therefore, is that for the good sleeper the precursor to sleep is abandoning wakefulness; whereas, for the person with PI it is trying to initiate sleep.

It also appears from the diagnostic schedule that explicitly intending to sleep is problematic for the person with PI. Although ICSD-2[4] criteria for PI are based in part on expert clinician evidence and await scientific validation, they do suggest PI patients fall asleep when they do not intend to. One of the PI criteria is:

> "Difficulty falling asleep in bed at the desired bedtime or during planned naps, but no difficulty falling asleep during other monotonous activities, when not intending to sleep." (criterion C2; p. 1–7)

The implication is that desire, planning and intention are counterproductive in PI. There is also the implication that people with PI may more readily fall asleep when they do not have the explicit intention to do so. This is consistent with the literature on paradox.

By definition, people with PI have difficulty falling asleep and remaining asleep, and by convention in insomnia research and in clinical practice we ask people to record these difficulties in a sleep diary. This raises an interesting point in relation to the measurement of sleep intention. Sleep-onset latency (SOL) is usually taken as the length of time it takes to fall asleep, after settling down with the intention of sleeping (after lights out). This fits with the insomnia model, because this is what they do. However, it would be interesting to know, from normal sleepers, on what proportion of nights they fell asleep before they ever intended to. We lack data on this, but our prediction would be that the true SOL values for some good sleeper

nights are in fact negative values, if we were to use the above definition of SOL literally.

The fact that we routinely ask patients to monitor sleep pattern on diaries raises another important point. According to our A-I-E model, to pay directed attention to the involuntary response that is sleep may impair its very automaticity. As therapists then, are we not likely to exacerbate patients sleep problems merely by asking them to complete diaries? Successful completion does require directed attention to sleep. This could be examined, experimentally, by comparing one group of PI patients on standard diary completion with a second PI group, whose sleep is monitored using a method not requiring direct participant involvement with their sleep data. We might expect however that the strongest inhibition of sleep would occur with direct monitoring of sleep during the sleep-onset period.

We would also note at this point that we believe the A-I-E pathway may be equally applicable to sleep maintenance problems. WASO is the primary measure of sleep (dis)continuity, representing the cumulative time taken to re-initiate sleep after night-time arousals. Our model would suggest that the selective attention process may be active before sleep, that it may remain active after sleep-onset and that it may re-activate during sleep, particularly during light sleep and its associated arousals. Because selective attention may be represented as a conditioned involuntary response to arousal, it does not require the person to be conscious or fully conscious. Indeed, the literature on evoked potentials suggests that the brain's capacity to discriminate the intrinsic significance or semantic content of a stimulus may persist in stage 2 and REM sleep.[82] More specifically then, in relation to WASO, it is possible that the vigilant scanning associated with selective attention becomes switched on during light sleep producing vulnerability to arousal and accentuating 'pre-existing' normal and transient nocturnal arousals. Consequently, arousals may be more likely to extend into consciousness and to frank awakenings. Whereas, it has been known for some time that even in normal sleepers there is significantly shorter awakening latency to meaningful stimuli presented during sleep,[133] it remains unclear from the research literature available at this time whether or not PI is associated with increased frequency of awakening from sleep.[134] Further research in this area is required.

However, once awake, somewhat less controversially, we propose that the person with sleep maintenance PI is then in exactly the same position as the person with sleep-onset PI. We are not the first to suggest that a central component of sleep-maintenance insomnia relates to a problem of re-initiating sleep rather than solely a disorder of intermittent wakening per se.[83] The explicit intention to get back to sleep would then apply during the night inhibiting de-arousal in the same way as at (the first) sleep-onset.

In summary, then we suggest that explicitly intending to sleep is (a) not what normal good sleepers do, (b) it is dysfunctional because it inhibits normal de-arousal, and by extension of these points (c) that normal sleep may be restored when intention to sleep is neutralized.

Evidence of explicit intention in PI

There are few studies that directly address sleep intention in PI. However, evidence from a number of sources is suggestive that explicit intention to sleep plays a part in PI.

The multiple sleep latency test (MSLT) is not routinely recommended for insomnia, partly because people with PI generally have daytime sleep latencies in the normal range or even longer.[84,85] This has been taken as support for a variety of different positions—that PI has no true daytime consequences, that people with PI are hyperaroused around the 24 h clock, and that people with insomnia find it difficult to sleep in labs or when observed or when trying to sleep. We would develop this last point by commenting that it may be important that, in the MSLT, people are explicitly trying to sleep. The typical task instruction is:

> "Please lie quietly, keep your eyes closed and try to fall asleep"
>
> (p. 1418)[86]

This could be why people with PI are unable to sleep under MSLT conditions. You might then wonder why explicitly trying to sleep would not inhibit people with other sleep disorders from sleeping during MSLT naps? Our suggestion here would be in terms of our concept of inhibitory sufficiency.[13] An explicit intention to sleep simply may not be sufficient to inhibit sleep in someone who has strong homeostatic pressure to sleep (e.g. narcolepsy) or who is otherwise sleep deprived (e.g. sleep apnea). However in PI, homeostatic pressure to sleep may be relatively weak (until sleep restriction is applied) or at best it may be highly variable; and people with PI are not objectively sleep deprived. Further experimental work varying the instructional set for the MSLT would therefore be informative. Similarly, a comparison of GS and PI

on the maintenance of wakefulness test, or simply using a non-instructional condition (e.g. 'We just want to calibrate the equipment for a few minutes with your eyes closed while you are laying there awake') could prove informative about potential underlying mechanisms.

The inhibitory properties of explicit intention in PI are also supported indirectly by a body of research on paradox and ironic control in insomnia. It has been suggested that anxiety responses may be conditioned not only to external, situational cues but also to the individual's behavior.[87-89] Fear of a performance failure (insomnia) and of anticipated negative consequences of that failure is described as performance anxiety. In the treatment known as paradoxical intention, counter-productive attempts to fall asleep are replaced by the intention of remaining passively awake or by giving up any direct intention to fall asleep.[90,91] This rationale is supported by the fact that good sleepers do not use any strategies to fall asleep. Typical instructions for paradoxical intention therapy have been summarised as follows by Morin and Espie (pp 95–97)[92]

1. When you are in bed lie in a comfortable position and put the light out.
2. In the darkened room, keep your eyes open, and try to keep them open 'just for just a little while longer'. That's your catch phrase.
3. As time goes by congratulate yourself on staying awake but relaxed.
4. Remind yourself not to try to sleep but to let sleep overtake you, as you gently try to resist it.
5. Keep this mind set going as long as you can, and if you get worried at staying awake remind yourself that that is the general idea, so you are succeeding.
6. Don't actively prevent sleep by trying to rouse yourself. Be like the good sleeper, let sleep come to you.

The emphasis, therefore, in paradoxical intention is upon natural sleep initiation and the patient is encouraged to take a passive, accepting role. Setting conditions for sleep are established (bed, comfortable, dark) but the explicit intention is to remain awake, thus obviating attempts to sleep. It is, of course, consistent with the view of wakefulness as accruing sleep debt that one of the most reliable ways to guarantee sleep is to remain awake. Paradoxical intention therapy takes advantage of this principle by prescribing wakefulness as a precursor to successful sleep. Paradoxical intention has demonstrated efficacy as a single therapy in controlled trials,[93,94] and is regarded as an intervention that reflects a 'moderate degree of clinical certainty' according to AASM practice criteria.[95,96]

Of course treatment outcome studies are not designed to test mechanisms of action, so data from such sources must be regarded as preliminary. Rather, both experimental comparison studies (GS, PI) and experimental manipulation studies[12] (in GS) are required to look at specific causal mechanisms. Four experimentally-based studies have been published that yield some evidence about the role of intention.

First, Gross and Borkovec[97] allocated good sleepers to one of three experimental conditions. All participants were instructed to 'go to sleep as quickly as possible' (p. 113) during a daytime nap opportunity. However, in one group they were told that they would have to make a speech at the end of the experiment on an unspecified subject, and in another that the speech had to be on a specific topic. The third group was a control condition with no manipulation of pre-sleep mental content. They found that the speech plus topic group had the longest mean SOL, suggesting that trying to sleep in circumstances where cognitive arousal/anxiety was increased led to greater sleep difficulty. In the Gross and Borkovec experiment it was not possible to separate out the effects of intention and cognitive task demands.

Second, Ansfield et al.[2] explored the effects of different sleep-onset instructions in good sleepers under high or low 'mental load'. This was an elegant study where two factors were systematically investigated. Good sleepers were instructed to fall asleep either "…as quickly as possible … in record time" or "… whenever you would like" (p. 526) under conditions of either "… stirring … marching band music" or "sleep-conducive … music containing restful, outdoor sounds …". Paradoxical wakefulness was found amongst those actively attempting to sleep while listening to the sleep-inhibiting music. This result was interpreted in terms of Wegner's[98] theory that the thwarted attempt to control a particular mental state can yield the opposite of what is desired. Ansfield et al. hypothesized that failure to fall asleep on a few occasions could occur when sleep is attempted under transitory mental loads, such as at times of stress. Eventually a person's thoughts about being unable to sleep could constitute a debilitating mental load which, when combined with the continuing frustrated desire to fall asleep, could lead to chronic insomnia. Interestingly, Ansfield et al. also found that explicit intention to sleep plus sleep-conducive music did not delay SOL, suggesting the importance of the interaction of

intention and mental load in insomnia. Furthermore, this group actually fell asleep more rapidly. This finding could reflect an experimental treatment effect where a relaxation response to calming music counteracted the explicit intention to sleep and delivered a sleep-promoting benefit. That is, although Ansfield et al. intended the calm music to be a neutral condition relative to the impact of the marching music, it seems plausible that it had a converse therapeutic effect.

Third, Harvey[99] has explored the effects of suppressing pre-sleep cognitive activity on SOL. People with insomnia and good sleepers were allocated either to a suppression condition ('suppress the thought most likely to dominate your thinking as you get into bed') or a non-suppression condition ('think about anything as you get into bed, including the thought you would most likely think about as you go to sleep'). Interestingly, 'suppress' participants reported longer sleep latencies and poorer sleep quality, regardless of whether they had insomnia or were good sleepers. Harvey concluded that thought suppression, whilst attempting to turn off pre-sleep intrusive thoughts, appeared to have the opposite effect in that it prevented sleep-onset, in a manner consistent with Wegner's theory of ironic mental control.[98] Wegner uses the terms 'intentional operating process' to refer to the search for mental content that will yield a desired state (e.g. drowsiness), and 'ironic monitoring process' to refer to the search for evidence of failure to achieve a desired state (e.g alertness). There are clear parallels here with Harvey's views on the negative impact of sleep-associated monitoring[36,52] and also with our concept of present state monitoring[13,48] that we now suggest forms part of the A-I-E pathway.

Before considering the final experimental study relevant to explicit intention, it is interesting that several preliminary findings show that thought control strategies may characterise people with insomnia. Harvey has examined beliefs about pre-sleep worry using the utility of pre-sleep worry questionnaire.[100] Relative to good sleepers, people with insomnia endorsed more positive belief statements about worry (e.g. 'worry in bed helps me get things sorted out in my mind'). The number of negative belief statements endorsed was not different between the groups. Harvey using the Thought Control Questionnaire for Insomnia also reported that people with insomnia used more reappraisal, worry and thought suppression strategies to control pre-sleep intrusions, relative to good sleepers.[101] Similarly, Ellis and Cropley found worry and punishment strategies to be the strategies most commonly used by people with insomnia.[102] Such attempts amongst people with insomnia to control their thoughts (so that they can clear the way for sleep), therefore, may fuel further intrusions, and so maintain sleep disturbance. We suggest that the motivation for thought control may be because, unlike normal good sleepers, people with insomnia are actively engaged with intentional sleep.

Fourth, Lundh and Hindmarsh[103] described a clinical experiment that has relevance to this thought control literature, and to what we have previously described as metacognition. Forty participants were instructed simply to monitor, but not to respond to, their thoughts and emotional state during the pre-sleep phase. This 'meta-cognitive observation' task was found to significantly reduce sleep latency. Although lacking a comparison group, the study provides a demonstration of the potential benefit to sleep of this 'mindfulness' approach, which in turn may mimic normal pre-sleep cognition.

Taking the body of available evidence as a whole then, we suggest that there is preliminary evidence to support both the presence of explicit sleep intentive processes in insomnia, and the utility of neutralizing/normalizing strategies to counteract their effects.

Sleep effort

Introduction

The third component of the proposed A-I-E pathway is what we have called sleep effort. Because, this is a developmental pathway, we conceive of A-I-E as comprising not so much discrete components as overlapping stages. Consequently, there is not a point at which intention ceases and effort commences (Fig. 1). Rather, we regard effort as a further development of intention, to the extent that the 'end state' of persistent PI (leaving aside the potential for depression to develop) could be described as a sleep effort syndrome.

Indeed, for the past century the Yerkes-Dodson Law has suggested that high physiological arousal can be disruptive to intended behaviors presumably by an excess of neural noise interfering with the sequences of choice points necessary in initiating behaviors. The decreases in signal to noise ratios at all these choice or decision points result in disruptions to skilled behaviors or voluntarily directed behaviors. Anxiety clearly increases arousal level, but so does any 'effort'. Whereas a certain amount of arousal can be a constructive behavioral

motivator, too much (or indeed too little) is likely to work against the organism. Because increased arousal in response to effort is an extremely well-learned response, we would argue why should it be different with increased effort to fall asleep?

In evidence of explicit intention in PI we illustrated explicit intention and how it interfered with sexual responsivity. An analogy for effortful preoccupation and its inhibitory consequences would be the phenomenon of stammering. It is rare for people with speech impediments of this type to have specific physiological or motor/mechanical disorder, although the 'causes' of stammering are best regarded as multi-factorial.[104] Deliberate and effortful speech production is typical, and automatic, fluent speech appears to be inhibited or compromised. Particular difficulty is evident in responsive speech to concrete questions where awareness of the correct answer and attempts to produce it lead to blocking and stammering on letters or syllables. Also, like sexual dysfunction and insomnia, there is marked frustration at being unable to accomplish what seems like a relatively simple thing—in this case to articulate words fluently. The automatic, over-learned nature of competent, fluent speech is so unremarkable that comment would seldom be made on successful everyday conversation. Consequently, difficulties with speech are of magnified concern to the stammerer. However, the 'good speaker', like the good sleeper is entirely unaware of the process, and exhibits no qualities or special competencies that are immediately obvious.

Attention to speech, intention to speak and effort to control spoken output seem to characterize the problem. This contributes to what may be one hallmark feature of effortful preoccupation, performance anxiety. Performance anxiety is found in a wide range of symptoms and disorders, particularly where psychological and physiological factors interact. Other examples include blushing, where social context triggers vasodilation but attentional and controlling responses exacerbate the symptoms, and psychogenic urinary retention where self-observation and active efforts to urinate restrict sphincter relaxation.[80] The criterion of performance is clear in each case (e.g. erection, speech, social encounter, urinating) thus making it easy for the person with the problem to identify inadequate performance or performance failure.

The impact of effortful processes has also been shown in experimental studies. For example, Wegner, Broome and Blumberg demonstrated using electrodermal measurement that trying to remember a 9-digit number during progressive muscle relaxation increased skin conductance level, whereas skin conductance decreased with relaxation alone.[105] This combination of intention to do one thing (relax) in the face of oppositional factors (mental load) parallels our concept of effort arising out of intention. 'Striving' would be a good synonym for our concept of effort.

It is also instructive that successful treatment for disorders where effort and conscious control are part of the problem does not involve reinforcement of effort as a strategy. On the contrary, disrupting the inhibition of what in our terms is the A-I-E pathway can yield good outcome. Many of these strategies are in fact paradoxical in nature. For example, in stammering, delayed auditory feedback disrupts self-observation and forced speech, and distractor or rhythmic procedures are used to enhance natural fluency rather than emphasising speech production per se.[106] At a more generic level, there is emerging evidence of the health value of the principle of acceptance (e.g.[107]). Of course, this is not a new idea because many philosophies and world religions embrace the importance of accepting certain situations instead of struggling against them. The principle has been particularly well articulated in what has become known as the mindfulness approach (e.g.[108]). where cognitive/emotional processes are observed without any ambition to change them. Although mindfulness is a much wider system of thinking and therapy than is actually required for the present purposes of understanding PI, it does lend further construct validity to intention and effort as maladaptive strategies.

Conceptualisation of sleep effort in relation to insomnia

Our suggestion is that the development of PI follows a pathway from an implicit information processing bias (pre-attentive perceptual) to an explicit processing bias (conscious mental), then to an explicit intention (responsive mental) and then to an effortful preoccupation (responsive/proactive behavioral). If selective attention is scanning mode and explicit intention is planning mode, then sleep effort is performing mode. Sleep effort is seen as comprising two, related processes—one that is direct (e.g. actively trying to sleep) and one that is indirect (e.g. increasing sleep opportunity). This conceptualisation therefore characterises the development of the behavioral response to the developing PI problem and is illustrated in Figs. 1 and 3.

A mechanical analogy may be helpful. We propose that good sleep is fully automated, in the

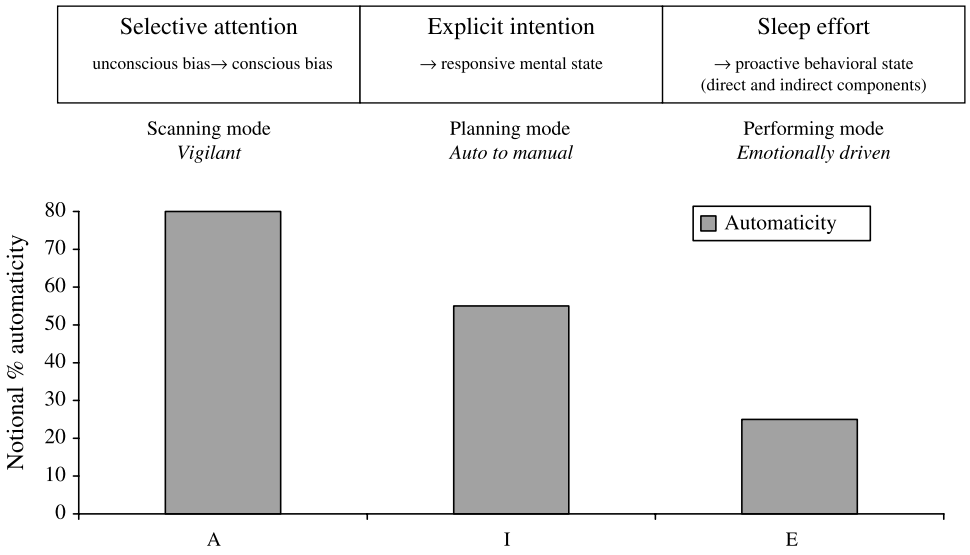


Figure 3 The development of psychophysiological insomnia following the attention–intention–effort pathway and its impact on automaticity.

context of appropriate setting conditions, but that selective attention partially impairs this automaticity. We suggest that it is then further impaired by a crucial switch towards 'manual' operation during the intentive stage. However, the lasting damage, resulting in persistent PI, may be done when a compelling need to take control and fix the problem develops. We suggest that this stage is more emotionally driven and so it will be particularly associated with evaluative considerations. This is what we mean by using the term performing mode (Fig. 3). Sleep has now become an enactment that is observed, analysed and performance reviewed. Dissatisfaction in these circumstances is likely to undermine not only sleep but also self-efficacy. Fig. 3 also illustrates notionally the cumulative effect of the A–I–E pathway upon the impairment of automaticity of the healthy sleep process.

Returning to ICSD-2[4] descriptions of PI, the following segments seem relevant to the concept of sleep effort.

> "Learned associations are marked by overconcern with the inability to sleep. A cycle develops in which the more one strives to sleep, the more agitated one becomes, and the less able one is to fall asleep" (p. 1–6)

> "... individuals with insomnia characteristically demonstrate effortful preoccupation with both the consequences of and the potential solutions for their sleep problems" (p. 1–6)

> "A sense of repeated failure to resolve sleep problems often leads to intermittent periods of resigned helplessness and help-seeking behaviors" (p. 1–7)

We suggest that the sleep effort response presents in two ways; first, as increased direct effort to get to sleep, or to get back to sleep when awake in bed; and second, as increased indirect effort to sleep by manipulating the setting conditions for normal sleep. Furthermore, 'efforts' may be both cognitive and behavioral. We will briefly describe these ideas.

Direct behavioral effort may include things like trying to force sleep to come, tossing and turning to find a sleep position, lying particularly still as if asleep, and being unwilling to 'give in' and get up when not sleeping. We have previously mentioned reading in bed with the express purpose of sleeping in this context, and people tiring themselves out by exercising vigorously is another example. Examples of direct cognitive effort include thought management, counting sheep, suppressing thoughts and distraction techniques. As will be apparent, direct efforts to sleep may also include well-intentioned strategies that have some basis in behavioral sleep medicine, e.g. the use of relaxation exercises as a sleep-inducer. Our point is that, to the extent these activities involve the direct effort to sleep, they may actually contribute towards the maintenance of PI, rather than towards its resolution. It is anecdotal, but nevertheless typical, of the person with PI to report that they have 'tried everything' to help them sleep!

Indirect sleep efforts may be particularly important in PI. This would be where maladaptive steps are taken to influence the probability of

sleep. In terms of our model, this not only upsets the default setting conditions for good sleep, but also reduces the statistical probability of sleep. At the cognitive level, people with insomnia often try to manage or contain levels of mental and emotional stimulation so that they are likely to sleep better. Sleep becomes such a priority that it is anticipated throughout the waking day. The most common behavioral responses are to increase sleep opportunity by going to bed earlier, staying in bed later, and trying to 'catch up' on lost sleep. We emphasized earlier how craving for sleep might result in increased time spent in bed. So, ironically, in an indirect behavioral effort to obtain more sleep, the net effect is to reduce sleep efficiency.

To illustrate this, consider the data presented in Table 3. This is a fictional case of a person averaging 63 min of wakefulness and with sleep efficiency of 85% based on 7 h in bed (week A). This sleep pattern is at the margin between normal sleep and insomnia, but let us suppose that the person is concerned about their sleep and its consequences, and so increases time in bed by 1 h per night for the next 7 nights. The data in week B assume that this actually results in some benefit to sleep duration (illustrated by a stable increase in sleep of 15 min per night). However, the impact on sleep efficiency reduces the average to 78% (range 66–86%). Although, this is only a modest 7% reduction from the week A value, for the homeostat to return efficiency to the 85% value, based on the new behavior of spending 8 h in bed, would require an average sleep of 408 min (480×0.85). Inspection of the raw data over the 14 nights in Table 3 show that this length of sleep was only ever obtained on 1 occasion, and that, in spite of sleeping a little more, average sleep in week B is some 36 min below that target.

Direct efforts to initiate sleep we suggest are more likely to be affect laden—e.g. lying awake trying to get to sleep. Indirect efforts such as increasing sleep opportunity may be less affect laden. Indeed, they may initially reduce anxiety because they offer the reassuring possibility of more sleep, and may actually deliver some. However, as well as undermining automaticity, they have the capacity to exacerbate insomnia symptomatology and place sleep efficiency outwith the range within which it can spontaneously recover.

Most commentators regard stimulus control/-sleep restriction as the core components of an effective CBT programs (e.g.[92]). One of the foremost reasons for this is that these interventions quickly and effectively tackle the problem of extended sleep opportunity. We cannot say at this stage by what means these procedures achieve their effects,[13] however, it should be noted that these, essentially behavioral, approaches are not inconsistent with the A-I-E pathway. Both stimulus control and sleep restriction therapies involve (a) establishing setting conditions for sleep that are largely determined by sleep needs, rather than sleep desires; (b) strengthening homeostasis and circadian timing, rather than personal agency over sleep; and (c) precluding the need for, or quickly abandoning, direct attempts to sleep, rather than trying to initiate sleep. Consistent with sleep normalcy, stimulus control and sleep restriction help to re-engage the 'two process' functions of sleep drive and timing. Consistent with the A-I-E pathway they also reinforce the implicit third process of automaticity.

Evidence of sleep effort in PI

Evidence of sleep effort in insomnia can be drawn from several sources.

In the context of paradoxical intention therapy, the outcome literature that we reviewed in the section on Explicit intention, includes three studies that provide some evidence of effortful processes and mechanisms.

Table 3 Comparison of 2 weeks of fictional sleep diary data illustrating the impact of extending sleep opportunity by 1 h

		Night 1	Night 2	Night 3	Night 4	Night 5	Night 6	Night 7	Mean
Week A	TST	380	360	390	320	350	400	300	357
	TIB	420	420	420	420	420	420	420	420
	SE	90	86	93	76	83	98	71	85
Week B	TST	395	375	405	335	365	415	315	372
	TIB	480	480	480	480	480	480	480	480
	SE	82	78	84	70	76	86	66	78

TST, total sleep time (min); TIB, time in bed (min); SE, sleep efficiency % (TST/TIB×100).

Fogle and Dyall[91] compared different approaches to delivering paradoxical instructions. They found that the instruction to 'give up trying' to sleep was just as effective in treating insomnia as the more explicitly paradoxical 'try to remain awake' instruction. In other words, ceasing to try may be the essential element in the paradoxical approach, which would be consistent with reversal of the effort component of the A-I-E pathway.

Our own early work using paradox yielded some unexpected findings. We found that it could be a very effective treatment for insomnia,[94] yet for some individuals the paradoxical directive seemed to re-focus performance anxiety and lead them to quite literally remain awake.[88] We interpreted this as an interaction between the performance focus of the individual (initially upon sleep) and the demand characteristics of the therapeutic environment, where they were being asked to stay awake (instead of to sleep), such that they tried too hard (to implement therapy). In other words, effort gets in the way. There may be parallels here with Lundh et al.'s suggestion that perfectionistic traits may be predispositional in some people with insomnia.[109]

Recently, we have completed another study on paradox.[110] This was an experimental trial that examined the effect of paradoxical intention on effort to sleep, on sleep performance anxiety, and on both objectively-estimated and subjectively-estimated SOL. Sleep effort was measured by a rating of 'When I went to bed last night, I tried really hard to get to sleep' ranging from 0 'not at all' to 6 'very much', and performance anxiety about sleep was rated using a preliminary version of the Glasgow sleep effort scale (GSES).[c] Following a seven-night baseline, 34 participants (mean age 25 years) with persistent sleep-onset insomnia (mean duration 6 years) were randomly allocated to fourteen nights of paradox, or to a control (sleep as usual) condition. The intervention period was deliberately short because we were primarily interested in the impact of paradoxical instruction upon sleep effort. Consistent with this model, participants allocated to paradox, relative to controls, showed a significant reduction in sleep effort and sleep performance anxiety. A developing trend for significantly lowered subjective (sleep diary) SOL in PI participants was also demonstrated within the brief treatment period. The important finding here was the observation that paradoxical intention appeared to operate by reducing sleep effort/anxiety. Effort change significantly correlated with SOL change when sleep anxiety was partialled out ($rp = 0.42$, $p = 0.016$). In contrast, when effort change was partialled out, sleep anxiety was not associated with SOL change ($rp = 0.08$, $p > 0.1$). This further supports the model of explicit sleep intention as inhibitory.

Some rating scales that are commonly used in insomnia research and in clinical practice contain items that suggest that an effortful approach to sleep might be implicated in PI. The dysfunctional beliefs and attitudes about sleep scale[43] is designed to measure cognitive distortions and thinking errors in insomnia. DBAS Item 7 ('When I have trouble falling asleep or getting back to sleep, I should stay in bed and try harder') forms part of a constellation of mental symptoms that Morin[111] originally related to 'faulty beliefs about sleep-promoting practices'. This item was retained (as Item 4) in a recent psychometric analysis of the DBAS that produced a shortened 10-item version comprising beliefs that demonstrably changed in response to CBT intervention.[112] A Principal Components Analysis (PCA) loaded this item on Factor III 'beliefs about the need for control over insomnia'.

Three of the 12 items in the Sleep Disturbance Questionnaire (SDQ) are also relevant to the concept of effort. In the original study using the SDQ the items 'I try too hard to get to sleep', 'I get too "worked up" at not sleeping' and 'I worry that I won't cope tomorrow if I don't sleep well' were used to select patients for paradoxical intention therapy because they were felt to reflect sleep effort and sleep performance anxiety.[44] Furthermore, Principal Components Analyses have demonstrated that these items do load together on the same construct.[44,112] However, the lack of a specific validated measure of sleep effort led us to develop a new self-report measure—the glasgow sleep effort scale (GSES).[113]

Work on the GSES began in the context of a study investigating the sensitivity and specificity of commonly-used insomnia research tools in discriminating PI, insomnia associated with mental disorder (I-MD) and GS.[114] Fifty-four adults (mean age 40 years; $n = 18$ per group) participated by completing a set of six psychometrically robust insomnia self-report instruments, along with the Beck Anxiety Scale and the Beck Depression Scale. Although the experimental groups differed on the majority of these measures, logistic regression analysis indicated that 'effortful preoccupation with sleep' (as measured by the GSES) discriminated PI from GS (with 100% sensitivity, and 94% specificity) and the GSES also discriminated I-MD from GS (100%, 100%). Furthermore, only depressive symptomatology (on the Beck Depression

[c] The final version of the GSES is described in greater detail below.

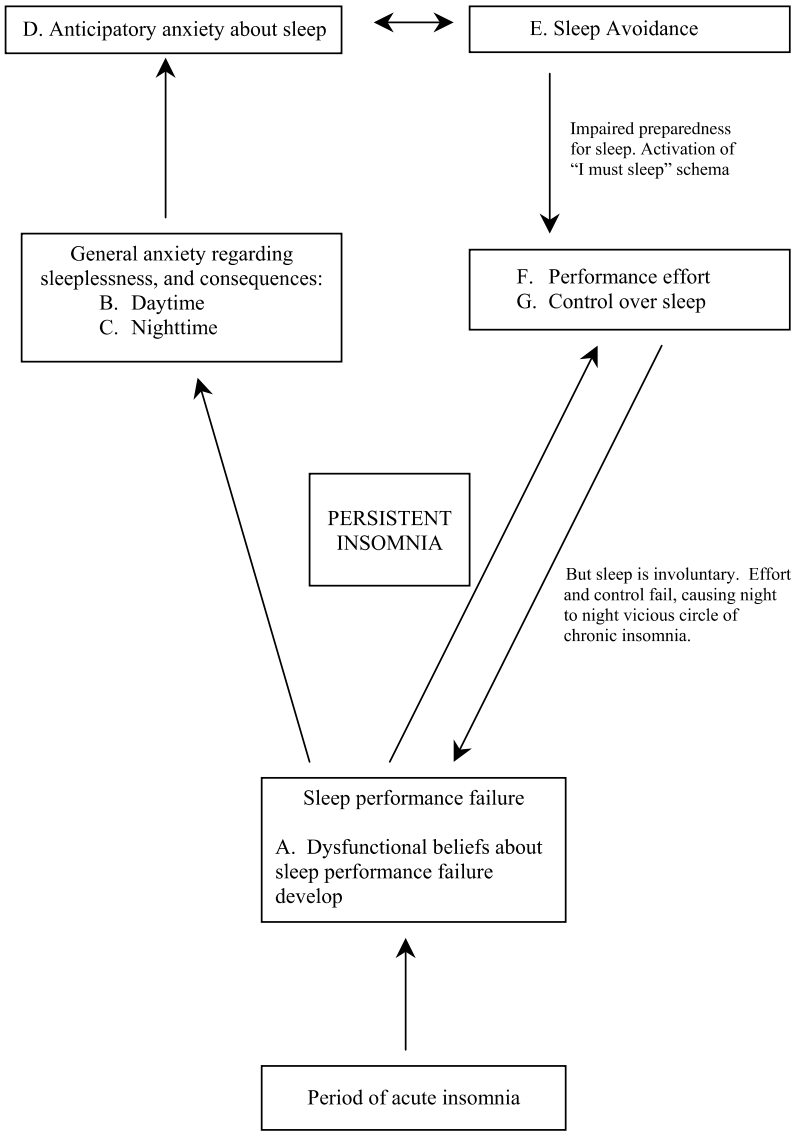


Figure 4 A preliminary working model of direct sleep effort in persistent Psychophysiologic Insomnia (reproduced from[113]).

Inventory (BDI)) discriminated I-MD from PI. No other variables entered any of the regression models.

These results not only suggest that sleep effort is an important concept in PI, but also that other mental and behavioral measurements may be less specific to PI as a disorder. Furthermore, the fact that these findings held also for insomnia associated with depression raises the interesting possibility that a continuum may exist across 'primary' and 'secondary' insomnia (at least where it is associated with depression/anxiety). This possibility is supported by other recent work on symptom reports in severe chronic insomnia which have found that 'depression-related insomnia' and PI were separable only by characteristic symptoms of depression.[115] On the other hand, Broman and Hetta[116] reported that cognitive and somatic pre-sleep arousal was not correlated with insomnia associated with affective disorder. Clearly, further work in this area is required.

We were encouraged by these results to conduct a formal validation study of the GSES.[113] Therefore, a working model was developed, integrating what we felt were the seven core subjective components of sleep effort[d] and each component was assigned a single item (Fig. 4 and Table 4). Fig. 4, thus, represents the final stage of the A-I-E pathway and illustrates what we mean by performing mode (cf. Figs. 1 and 3 and associated text). This proactive

[d] That is direct sleep effort. Indirect sleep effort (e.g. by increasing time in bed) is best measured behaviorally, on a sleep diary, rather than on a Likert Scale.

Table 4 *The Glasgow sleep effort scale*[113]

The Glasgow sleep effort scale The following seven statements relate to your night-time sleep pattern *in the past week*. Please indicate by circling one response how true each statement is for you				
1.	I put too much effort into sleeping when it should come naturally	Very much	To some extent	Not at all
2.	I feel I should be able to control my sleep	Very much	To some extent	Not at all
3.	I put off going to bed at night for fear of not being able to sleep	Very much	To some extent	Not at all
4.	I worry about not sleeping if I cannot sleep	Very much	To some extent	Not at all
	I am no good at sleeping	Very much	To some extent	Not at all
6.	I get anxious about sleeping before I go to bed	Very much	To some extent	Not at all
7.	I worry about the consequences of not sleeping	Very much	To some extent	Not at all

Relationship of Items 1–7 to components A–G in Fig. 4: 1–F; 2–G; 3–E; 4–C; 5–A; 6–D; 7–B.

state is emotionally driven in an effort to solve the problem of sleeplessness. Each core component was allocated one item in the GSES and we field-tested the scale on 89 insomnia patients and 102 good sleepers. The GSES was found to have good internal consistency ($\alpha = 77$) and discriminant validity. Mean total GSES score for PI was 7.06 (SD = 3.58) and for GS was 1.22 (SD = 1.35) [$t = 15.27$, $p < 0.0001$]. Importantly, sensitivity/specificity analysis found that a cut off score of only two correctly identified 93.3% of insomnia patients, and 87.3% of good sleepers. This result is supportive of the concept of automaticity in normal good sleep. GS simply do not endorse the items because the idea of trying to sleep is quite alien to them. There was also evidence of the construct validity of 'sleep effort' because PCA yielded a single principal component (Eigenvalue = 4.38) accounting for 62.6% of total variance, and each of the seven items loaded similarly and significantly on this factor (range 0.64–0.85).

Further work is clearly required on the GSES. Nevertheless, it has the potential to be a quick screening method to identify people with PI in the community, and used alongside other measures, to contribute to further research investigating diagnostic components of PI.

Discussion

In this paper, we have argued for an expansion of experimental cognitive research on insomnia, and have focused upon evidence relevant to the appraisal of what we believe may represent (for Psychophysiologic Insomnia at least) one critically important sleep inhibitory process: the attention–intention–effort pathway. Consistent with our starting point of understanding how PI differs from sleep normalcy,[13] we have suggested that the involuntary and automatic nature of the 'two process' sleep system is first compromised by selective attention to sleep, then imperilled by explicit intention to sleep, and finally dysregulated by a destructive combination of direct and indirect sleep effort. Therefore, PI in its end state as a 'sleep effort syndrome', may be characterised by attention bias, sleep preoccupation, and a panoply of mental and behavioral strategies designed to deliver sleep and to avoid sleeplessness, none of which would be typical in good sleepers. Of course, we must bear in mind that the true end state of insomnia may lie, not in intractable insomnia alone, but in the dysregulation of affect, because insomnia is an independent risk factor for depressive disorder.[117,118]

Evidence to support an A–I–E model is only beginning to emerge and there is much work still to do. Indeed, for now, whilst there is reasonable support for the attention component of the model, there is less evidence for the second and third components. The information processing literature on anxiety disorders emphasises attention bias toward emotionally threatening stimuli. This concept of 'sleep cue as threat' may apply to PI, although salience may be conferred on sleep cues for other reasons, not the least of which might be a 'craving' for sleep. Because, we are interested in the specificity of attention bias in insomnia, it will be important to partition and compare responses (within subjects) to threat words (e.g. tired,

wakeful, restless) and non-threat words (e.g. rested, relaxed, sleeping) using a verbal attention bias paradigm. We have also argued that attention is first 'allocated' to sleep cues as part of an unconscious process, akin to conditioning. This implies that people may be already incubating an insomnia response before they are aware that sleep and sleeplessness are grabbing their attention, as in the transition from acute/adjustment insomnia to persistent insomnia. Exploration of attention bias at the implicit or pre-attentive level is therefore required, and can be achieved using subliminal variants of attention bias probe tasks.[119]

The selective element of attention is also important to consider, because inevitably as attention is focused more in one direction, it is focused less in another direction. Posner has suggested that the attention system is not unitary, but comprises measurable cognitive components (shift, engage, disengage),[77] which are sub-served by specific, neural sub-systems[120,121] and, which are open to modulation by negative emotional stimuli.[29] In recent years, anxiety researchers have begun to apply Posner's attention model to determine whether salient threat stimuli attract attention i.e. modulate the engagement component of covert attention, and/or hold attention, i.e. modulate the disengage component.[122] Most findings for anxiety emphasise slowed disengagement from threat, i.e. a holding function.[123,124] In insomnia research, it would be useful, therefore, to determine which components of attention comprise the bias for sleep words that we have observed. Use of a modified cue-target paradigm would be appropriate for this purpose.

Similarly, we have reprised the notion of 'attention for action'.[74] That is selective attention has a purpose. In general terms, it confers evolutionary advantage by prioritising and directing activity through intention and goal-directed behavior. However, sleep may not be a response that is facilitated by such direct action; rather it may be inhibited. It remains to be demonstrated through experimental study that threat of sleeplessness and/or desire for sleep would drive explicit intention to sleep, although the idea is consistent with clinical experience and with contemporary diagnostic criteria for PI. The glasgow sleep effort scale, however, includes items that may span the A-I-E construct and reflect this 'dynamic' force of attention for action. The GSES has some encouraging sensitivity/specificity data[113,114] and may prove to be a useful measure for identifying individuals with PI. Ideally, however, sleep intention and sleep effort should not be measured solely by a self-report instrument. Some additional measure of mental/behavioral response tendency is desirable. We have illustrated how extending sleep opportunity by increasing time in bed could be a behavioral correlate of indirect sleep effort, leading to homeostatic dysregulation. In parallel, direct sleep effort (trying to force sleep to come) is likely to increase cognitive and emotional arousal.

The interrelationship between attention, intention and effort in the emergence and persistence of PI also needs to be studied. For example, researchers might consider exploring whether sleep intention and sleep effort can be reliably induced, experimentally, in good sleepers through manipulation of sleep monitoring instructions. It may also be possible to study, longitudinally, following an acute stressor event, the pattern of sleep change and associated development of attention, intention and effort, in individuals predisposed to PI. Extending the cross-sectional paradigm employed by Taylor et al.[69] in cancer patients would be useful here. Importantly, the work of Harvey and colleagues is leading the way by emphasizing the application of controlled experimental methods to tease out causal pathways in insomnia.[56–58] Computerised experimental tasks seem complementary to this work because they offer an objective index of sleep-related cognitive arousal.[69,70,72,73]

Related to these matters is the question of whether psychological treatment impacts upon the A-I-E pathway. For example, attention biases reduce following CBT therapy for anxiety disorders.[125,126] Demonstrating that established psychological treatments such as stimulus control or multi-component CBT impact attention bias in PI would add strength to the argument that such biases play a critical role. We are currently gathering data considering the impact of CBT on attention bias in individuals suffering sleep disturbance secondary to cancer, and will also have some outcome data on the GSES from other clinical trials.

We have suggested that, to the extent that any CBT method enables an individual to abandon personal agency over sleep and to return to total reliance upon involuntary sleep it may be likely to achieve a good therapeutic effect. This might result from a cognitive manipulation, aimed at changing the person's perspective upon sleep. Mindfulness approaches, which have recently been applied to insomnia treatment,[103] and other 'acceptance' based approaches, are of relevance here, as are attention-retraining methods (already used in the treatment of anxiety disorder[127]), and the more established cognitive and paradoxical

techniques that form part of standard CBT for insomnia.[92] However, we would point out that simple behavioral manipulations might be expected to achieve the end-point of involuntary sleep more quickly. Both sleep restriction and stimulus control (particularly the quarter of an hour rule) involve resisting or giving up efforts to initiate sleep and replacing them with an ultimate abandonment of wakefulness due to sleep pressure. This seems typical of normal good sleepers.

Finally, it seems important to examine the physiological substrate of the A-I-E pathway. In general, few studies have collected psychophysiological data associated with attention bias mechanisms.[122] Most employ only reaction time, despite the direct measurement potential of autonomic indices such as heart rate, skin conductance, as well as gaze direction. Additionally, examining attention allocation in PI using bedroom based (ambulatory) autonomic measurement would better explore conditioned arousal patterns in association with attention bias. Several studies have shown that differential attention responses can be activated by differing sensory and affective properties of the cue, and can be indexed using autonomic measures such as heart rate.[65] For example, deployment of attention towards sleep cues in PI may give rise to an orienting response associated with cardiac deceleration, whereas rapid deployment away might be associated with a defensive response accompanied by cardiac acceleration.[128] Importantly, this cognitive-psychophysiological framework should extend to exploration of the relationship between cognitive and cortical arousal, with data on the latter indexed from the EEG power spectrum (cf.[129]). If the attention bias paradigm provides a direct measure of cognitive arousal, and skin conductance or heart rate variability offer a parallel measure of autonomic function, then comparisons of such data with quantitative EEG parameters could prove particularly informative in exploring the underpinnings of the PI phenotype.

Practice points

1. Computerized information-processing tasks offer a novel objective method for appraising mental arousal associated with insomnia, particularly the sleep-related attention and monitoring component of arousal.
2. The A-I-E pathway offers a framework for evaluating the development of psychophysiologic insomnia as a persistent disorder following acute, stress-related sleep disturbance.
3. The Glasgow sleep effort scale is a brief measure that discriminates psychophysiologic insomnia, and operationalises this component of sleep preoccupation. It may be useful for diagnostic purposes.
4. The A-I-E pathway suggests that targeted CBT intervention to modify the attentional, the intentional or the effortful processes, which maintain insomnia may be sufficient to reduce sleep disturbance and to restore normal sleep.
5. The A-I-E model as a pathway suggests that novel interventions may be applied early to prevent the development of severe and persistent insomnia.

Research agenda

1. Prospective, longitudinal study of the A-I-E pathway is required to investigate its role in the etiology of insomnia.
2. Further studies are required to investigate the specificity of sleep-related attention bias to Psychophysiologic Insomnia, as opposed to other forms of sleep disorder.
3. Investigation of the somatic correlates of attention bias is required to understand insomnia as a psychophysiologic disorder with autonomic, cortical and cognitive components.
4. Future studies need to consider the components of attention (e.g. engagement, disengagement) that comprise selective attention bias to sleep cues in people with insomnia.
5. There is need to investigate the impact that psychological intervention has upon the A-I-E pathway. In particular, to consider whether measures of sleep-related information-processing bias and sleep effort reduce following CBT.

Acknowledgements

Supported by research grants from the Chief Scientist Office, Scottish Executive (CZH/4/Z), Cancer Research UK (C8265/A3036), The Wellcome Trust (070969/Z/03/Z) and support from the Mortimer and Theresa Sackler Foundation. The authors are grateful to Dr Allison G. Harvey (University of Berkeley, CA) for her helpful

comments and suggestions during the preparation of this paper.

References

1. Frankl VE. *The doctor and the soul*. 2nd ed. New York: Knopf; 1965.
* 2. Ansfield ME, Wegner DM, Bowser R. Ironic effects of sleep urgency. *Behav Res Ther* 1996;**34**:523–31.
3. American Sleep Disorders Association. *International classification of sleep disorders: diagnostic and coding manual (Revised)*. Rochester, MA: ASDA; 1997.
4. American Academy of Sleep Medicine. *International classification of sleep disorders: diagnostic and coding manual*. 2nd ed. Westchester, IL: AASM; 2005.
5. American Psychiatric Association. *Diagnostic and statistical manual of mental disorders*. 4th ed. Washington, DC: APA; 1994.
6. Edinger JD, Bonnet MH, Bootzin RR, Doghramji K, Dorsey CM, Espie CA, et al. Derivation of research diagnostic criteria for insomnia: report of an american academy of sleep medicine work group. *Sleep* 2004;**27**: 1567–96.
7. Perlis ML, Pigeon W, Smith MT. Etiology and pathophysiology of insomnia. In: Kryger MH, Roth T, Dement WC, editors. *The principles and practice of sleep medicine*. 4th ed. Philadelphia, PA: Saunders; 2005. p. 714–25.
8. Morin CM, Culbert JP, Schwartz MS. Non-pharmacological interventions for insomnia: a meta-analysis of treatment efficacy. *Am J Psychiatry* 1994;**151**:1172–80.
9. Murtagh DR, Greenwood KM. Identifying effective psychological treatments for insomnia: a meta-analysis. *J Consult Clin Psychol* 1995;**63**:79–89.
10. Smith MT, Perlis ML, Park A, Giles DE, Pennington JA, Buysse DJ. Behavioral treatment vs pharmacotherapy for insomnia—a comparative meta-analyses. *Am J Psychiatry* 2002;**159**:5–11.
11. Edinger JD, Wohlgemuth WK. The significance and management of primary insomnia: the past, present and future of behavioral insomnia therapies. *Sleep Med Rev* 1999;**3**: 101–18.
12. Harvey AG, Tang NKJ. Cognitive behaviour therapy for insomnia: can we rest yet? *Sleep Med Rev* 2003;**7**:237–62.
* 13. Espie CA. Insomnia: conceptual issues in the development, persistence, and treatment of sleep disorders in adults. *Annu Rev Psychol* 2002;**53**:215–43.
* 14. Lundh LG, Broman JE. Insomnia as an interaction between sleep-interfering and sleep-interpreting processes. *J Psychosom Res* 2000;**49**:299–310.
15. Borbely AA, Ackermann. Sleep homeostasis and models of sleep regulation. In: Kryger MH, Roth T, Dement WC, editors. *Principles and practice of sleep medicine*. 3rd ed. Philadelphia, PA: Saunders; 2000. p. 377–90.
16. Dijk DJ, Czeisler CA. Contribution of the circadian pacemaker and the sleep homeostat to sleep propensity, sleep structure, electroencephalographic slow waves, and sleep spindle activity in humans. *J Neurosci* 1995;**15**:3526–38.
17. Bootzin RR, Epstein D, Wood JM. Stimulus control instructions. In: Hauri P, editor. *Case studies in insomnia*. New York: Plenum Press; 1991. p. 19–28.
18. James W. *Principles of psychology*, vol. 1. New York: Holt; 1890.
19. Mogg K, Bradley BP. A cognitive—motivational analysis of anxiety. *Behav Res Ther* 1998;**36**:809–48.
20. Williams JMG, Watts FN, MacLeod C, Mathews A. *Cognitive psychology and emotional disorders*. 2nd ed. New York: Wiley; 1997.
* 21. Mathews A, MacLeod C. Cognitive approaches to emotion and emotional disorders. *Annu Rev Psychol* 1994;**45**:25–50.
22. Dalgleish T, Watts FN. Biases of attention and memory in disorders of anxiety and depression. *Clin Psychol Rev* 1990; **10**:589–604.
23. Harvey AG. Attention. In: Harvey AG, Watkins E, Mansell W, Safran R, editors. *Cognitive behavioural processes across psychological disorders: a transdiagnostic approach to assessment and treatment*. Oxford: Oxford University Press; 2004. p. 25–71.
24. Beck AT, Clark DA. An information processing model of anxiety: automatic and strategic processes. *Behav Res Ther* 1997;**35**:49–58.
25. Wells, Mathews G. *Attention and emotion: a clinical perspective*. Hillsdale, NJ: LEA; 1994.
26. Wells. *Emotional disorders and metacognition*. New York: Wiley; 2000.
27. Dalgleish T. Performance on the emotional stroop task in groups of anxious, expert and control participants. *Cognit Emotion* 1995;**9**:341–62.
28. Segerstrom S. Optimism and attentional bias for negative and positive stimuli. *Pers Soc Psychol Bull* 2001;**27**: 1334–43.
29. Stormark KM, Nordby H, Hugdahl K. Attentional shifts to emotionally charged cues: behavioural and ERP data. *Cognit Emotion* 1995;**9**:507–23.
30. Lusher J, Chandler C, Ball D. Alcohol dependence and the alcohol stroop paradigm: evidence and issues. *Drug Alcohol Dependence* 2004;**75**:225–31.
31. Mogg K, Mathews A, Bird C, MacGregor-Morris R. Effects of stress and anxiety on the processing of threat stimuli. *J Pers Soc Psychol* 1990;**59**:1230–7.
32. Drummond DC, Tiffany ST, Glautier S, Remington B. *Addictive behaviour: cue exposure theory and practice*. New York: Wiley; 1995.
33. Shiffrin RM, Schneider W. Controlled and automatic human information processing. II: perceptual learning, automatic attending, and a general theory. *Psychol Rev* 1977;**84**: 127–90.
34. Öhman A, Soares JJF. Unconscious anxiety: phobic responses to masked stimuli. *J Abnorm Psychol* 1994;**103**: 231–40.
35. Maslow. A theory of human motivation. *Psychol Rev* 1943; **50**:370–96.
* 36. Harvey AG. A cognitive model of insomnia. *Behav Res Therapy* 2002;**40**:869–93.
37. Salkovskis PM. The importance of behaviour in the maintenance of anxiety and panic: a cognitive account. *Behav Psychother* 1991;**19**:6–19.
38. Reber AS. An evolutionary context for the cognitive unconscious. *Philos Psychol* 1992;**5**:33–51.
39. Masters RSW, Polman RCJ, Hammond NV. Reinvestment: a dimension of personality implicated in skill breakdown under pressure. *Pers Indiv Differ* 1993;**14**:655–66.
40. Masters RSW, MacMahon KMA, Pall HS. Implicit motor learning in Parkinson's disease. *Rehabil Psychol* 2004;**49**: 79–82.
41. Broomfield NM, Gumley AI. and Espie CA. Candidate cognitive processes in insomnia. *J Cogn Psychother Int Q.* 2005; **19**:3–15.

* The most important references are denoted by an asterisk.

42. Nicassio PM, Mendlowitz DR, Fussell JJ, Petras L. The phenomenology of the pre-sleep state: the development of the pre-sleep arousal scale. *Behav Res Ther* 1985;**23**: 263–71.
43. Morin CM, Stone J, Trinkle D, Mercer J, Remsberg S. Dysfunctional beliefs and attitudes about sleep among older adults with and without insomnia complaints. *Psychol Ageing* 1993;**8**:463–7.
44. Espie CA, Brooks DN, Lindsay WR. An evaluation of tailored psychological treatment of insomnia. *J Behav Ther Exp Psychiatry* 1989;**20**:143–53.
45. Fichten, Libman E, Creti L, Amsel R, Tagalakis V, Brender W. Thoughts during awake times in older good and poor sleepers: the self statement test: 60+. *Cognit Ther Res* 1998;**22**:1–20.
46. Tang NKY, Harvey AG. Correcting distorted perception of sleep in insomnia: a novel behavioural experiment? *Behav Res Ther* 2004;**42**:27–39.
47. Watts FN, Coyle K, East MP. The contribution of worry to insomnia. *Br J Clin Psychol* 1994;**33**:211–20.
48. Wicklow, Espie CA. Intrusive thoughts and their relationship to actigraphic measurement of sleep: towards a cognitive model of insomnia. *Behav Res Ther* 2000;**38**:679–93.
49. Harvey KJ, Espie CA. Development and preliminary validation of the glasgow content of thoughts inventory (GCTI): a new measure for the assessment of pre-sleep cognitive activity. *Br J Clin Psychol* 2004;**43**:409–20.
50. Harvey AG. Pre-sleep cognitive activity: a comparison of sleep-onset insomniacs and good sleepers. *Br J Clin Psychol* 2000;**39**:275–86.
51. Neitzert Semler C, Harvey AG. An investigation of monitoring for sleep-related threat in primary insomnia. *Behav Res Ther* 2004;**42**:1403–20.
* 52. Neitzert Semler C, Harvey AG. Monitoring of sleep-related threat in primary insomnia: development and validation of the sleep associated monitoring inventory (SAMI). *Psychosomatic Med* 2004;**66**:242–50.
53. Minto JM, Espie CA. Is presleep cognitive over-arousal epiphenomenal in primary (psychophysiologic) sleep-onset insomnia? Under review.
54. Lack L, Bootzin RR. Circadian rhythm factors in insomnia and their treatment. In: Perlis ML, Lichstein KL, editors. *Treating sleep disorders: principles and practice of behavioral sleep medicine*. New Jersey: Wiley; 2003.
55. Harvey AG, Watkins E, Mansell W, Safran R. *Cognitive behavioural processes across psychological disorders: a transdiagnostic approach to assessment and treatment*. Oxford: Oxford University Press; 2004.
56. Neitzert Semler C, Harvey AG. An experimental investigation of daytime monitoring for sleep-related threat in primary insomnia. *Cognit Emotion*; in press.
57. Neitzert Semler C, Harvey AG. Daytime functioning in primary insomnia: does attentional focus contribute to real or perceived impairment? *Behav Sleep Med*. 2006; **4**: 85–103.
58. Tang NKJ, Harvey AG, Schmidt DA. Sleeping with the enemy: clock monitoring in the maintenance of insomnia. *J Behav Ther Exp Psychiatry*; in press.
59. Broman JE, Hetta J. Electrodermal activity in patients with persistent insomnia. *J Sleep Res* 1994;**13**:165–70.
60. Lichstein KL, Fanning J. Cognitive anxiety in insomnia: an analogue test. *Stress Med* 1990;**6**:47–51.
61. Waters WF, Adams SG, Binsk P, Varnado P. Attention, stress and negative emotion in persistent sleep-onset and sleep-maintenance insomnia. *Sleep* 1993;**16**:128–36.
62. MacLeod CM. Half a century of research on the stroop effect: an integrative review. *Psychol Bull* 1991;**120**:3–24.
63. MacLeod CM, Mathews A, Tata P. Attentional bias in emotional disorders. *J Abnorm Psychol* 1986;**95**:15–20.
64. Yiend J, Mathews A. Anxiety and attention to threatening pictures. *Q J Exp Psychol Hum Exp Psychol* 2001;**54**:665–81.
* 65. Lang PJ, Bradley MM, Cuthbert BN. Motivated attention: affect, activation and action. In: Lang PJ, Balabam M, Simons RF, editors. *Attention and orienting: sensory and motivational processes*. Hillsdale, NJ: Earlbaum; 1997.
66. Rensink RA. Change detection. *Annu Rev Psychol* 2002;**53**: 245–77.
67. Simons DJ. Current approaches to change blindness. *Vis Cogn* 2000;**7**:1–15.
68. Lundh L-G, Froding A, Gyllenhammer L, Broman J-K, Hetta J. Cognitive bias and memory performance in patients with persistent insomnia. *Scand J Behav Ther* 1997;**26**:27–35.
* 69. Taylor LM, Espie CA, White CA. A comparative experimental investigation of attentional bias in people with acute versus persistent insomnia secondary to cancer. *Behav Sleep Med* 2003;**1**:200–12.
70. MacMahon KMA, Broomfield NM, Macphee LM, Espie CA. Attention bias for sleep-related stimuli in primary insomnia and delayed sleep phase syndrome, *Sleep*; in press.
71. Jones BT, Jones BC, Smith H, Copely N. A flicker paradigm for inducing change blindness reveals alcohol and cannabis information processing biases in social users. *Addiction* 2003;**98**:235–44.
* 72. Jones BT, Macphee LM, Broomfield NM, Jones BC, Espie CA. Sleep-related attentional bias in good, moderate and poor (primary insomnia) sleepers. *J Abnorm Psychol* 2005;**114**: 249–58.
* 73. Macphee LM, Biello SM, Broomfield NM, MacMahon KMA, Espie CA. Who is pre-occupied with sleep?: a comparison of attention bias in people with psychophysiologic insomnia, delayed sleep phase syndrome and good sleepers using the induced change blindness paradigm *J Sleep Res* 2006;**15**: 1–10.
74. Allport DA. Patterns and actions: cognitive mechanisms are content specific. In: Claxton GL, editor. *Cognitive psychology: new directions*. London: Routledge & Kegan Paul; 1980. p. 26–64.
75. Broadbent. *Perception and communication*. London: Pergamon Press; 1958.
76. Deutsch J, Deutsch D. Attention: some theoretical considerations. *Psychol Rev* 1963;**70**:80–90.
77. Posner MI. Orienting of attention. *Q J Exp Psychol* 1980;**32**: 3–25.
78. Masters WH, Johnson VE. *Human sexual response*. New York: Lippincott/Williams & Wilkins; 1966.
79. Heiman JR, Meston CM. Empirically validated treatments for sexual dysfunction. In: Dobson KS, Craig KD, editors. *Empirically supported therapies: best practice in professional psychology*. New York: Sage Publications; 1998. p. 259–303.
80. Seltzer LF. *Paradoxical strategies in psychotherapy*. New York: Wiley; 1986.
81. Strong SR. Experimental studies in explicitly paradoxical interventions: results and implications. *J Behav Ther Exp Psychiatry* 1984;**15**:189–94.
82. Bastuji H, Garcia-Larrea L. Evoked potentials as a tool for the investigation of human sleep. *Sleep Med Rev* 1999;**3**: 23–45.
83. Thacher PV, Pigeon WR, Perlis ML. Do patients with sleep maintenance problems have a problem with sleep maintenance. *Behav Sleep Med*; in press.
84. Chambers MJ, Keller B. Alert insomniacs: are they really sleep deprived? *Clin Psychol Rev* 1993;**13**:649–66.

85. Stepanski EJ, Zorick F, Peters M, Roth T. Effects of sleep deprivation on alertness in chronic insomnia. *Sleep Res* 1990;**19**:297.
86. Mitler MM, Carskadon MA, Hirshkowitz M. Evaluating sleepiness. In: Kryger MH, Roth T, W.C. Dement, editors. *Principles and practice of sleep medicine*. 4th ed. Philadelphia, PA: Saunders; 2005.
87. Ascher LM, Turner RM. Paradoxical intention and insomnia: an experimental investigation. *Behav Res Ther* 1979;**17**: 408–11.
88. Espie CA, Lindsay WR. Paradoxical intention in the treatment of chronic insomnia: six case studies illustrating variability in the therapeutic response. *Behav Res Ther* 1985;**23**:703–9.
89. Espie CA. *The psychological treatment of insomnia*. Chichester: Wiley; 1991.
90. Ascher LM, Turner RM. A comparison of two methods for the administration of paradoxical intention. *Behav Res Ther* 1980;**18**:121–6.
91. Fogle DO, Dyall JA. Paradoxical giving up and the reduction of sleep performance anxiety in chronic insomniacs. *Psychother Theory Res Pract* 1983;**20**:21–30.
92. Morin CM, Espie CA. *Insomnia: a clinical guide to assessment and treatment*. New York: Kluwer/Plenum Publishers; 2003.
93. Turner RM, Ascher LM. Controlled comparison of progressive relaxation, stimulus control, and paradoxical intention therapies for insomnia. *J Consult Clin Psychol* 1979;**47**: 500–8.
94. Espie CA, Lindsay WR, Brooks DN, Hood EM, Turvey T. A controlled comparative investigation of psychological treatments of chronic sleep-onset insomnia. *Behav Res Ther* 1989;**27**:79–88.
95. Morin CM, Hauri P, Espie CA, Spielman A, Buysse DJ, Bootzin RR. Nonpharmacologic treatment of chronic insomnia: an American academy of sleep medicine review. *Sleep* 1999;**22**:1134–56.
96. Chesson AL, Anderson WM, Littner M, Davila D, Hartse K, Johnson S, et al. Practice parameters for the nonpharmacologic treatment of chronic insomnia. *Sleep* 1999;**22**: 1128–33.
97. Gross RT, Borkovec TD. Effects of a cognitive intrusion manipulation on sleep-onset latency of good sleepers. *Behav Ther* 1982;**13**:117–24.
98. Wegner DM. Ironic processes of mental control. *Psychol Rev* 1994;**101**:34–52.
99. Harvey AG. Attempted suppression of pre-sleep cognitive activity in insomnia. *Cognit Ther Res* 2001;**27**:593–602.
100. Harvey AG. Beliefs about the utility of pre-sleep worry: an investigation of individuals with insomnia and good sleepers. *Cognit Ther Res* 2003;**27**:403–14.
101. Harvey AG. I cant sleep, my mind is racing! an investigation of strategies of thought control in insomnia. *Behav Cognit Psychother* 2001;**29**:3–11.
102. Ellis J, Cropley M. An examination of thought control strategies employed by acute and chronic insomniacs. *Sleep Med* 2002;**3**:293–300.
103. Lundh L-G, Hindmarsh H. Can metacognitive observation be used in the treatment of insomnia? A pilot study of a cognitive-emotional self-observation task. *Behav Cognit Psychother* 2002;**30**:233–6.
104. Wingate M. *Foundations of stuttering*. New York: Academic Press; 2002.
105. Wegner DM, Broome A, Blumberg SJ. Ironic effects of trying to relax under stress. *Behav Res Ther* 1997;**35**:11–21.
106. Andrews G, Guitar B, Howie P. Meta-analysis of the effects of stuttering treatment. *J Speech Hear Disord* 1980;**45**: 287–307.
107. Hayes SC, Strosahl KD, Wilson KG. *Acceptance and commitment therapy: an experiential approach to behavior change*. New York: Guilford Press; 1999.
108. Segal ZV, Williams MG, Teasdale JD. *Mindfulness-based cognitive therapy for depression: a new approach to preventing relapse*. New York: Guilford Press; 2001.
109. Lundh L-G, Broman JE, Hetta J, Saboonchi F. Perfectionism and insomnia. *Scand J Behav Ther* 1994;**23**:3–18.
110. Broomfield NM, Espie CA. Initial insomnia and paradoxical intention: an experimental investigation of putative mechanisms using subjective and actigraphic measurement of sleep. *Behav Cognit Psychother* 2003; **31**:313–24.
111. Morin CM. *Insomnia: psychological assessment and management*. New York: Guilford Press; 1993.
112. Espie CA, Inglis SJ, Harvey L, Tessier S. Insomniacs' attributions: psychometric properties of the dysfunctional beliefs and attitudes about sleep scale and the sleep disturbance questionnaire. *J Psychosom Res* 2000;**48**: 141–8.
*113. Broomfield NM, Espie CA. Toward a valid, reliable measure of sleep effort. *J Sleep Res* 2005;**14**:401–7.
114. Kohn L, Espie CA. Sensitivity and specificity of measures of the insomnia experience: a comparative study of psychophysiologic insomnia, insomnia associated with mental disorder and good sleep. *Sleep* 2005;**28**:104–12.
115. Moul DE, Nofzinger EA, Pilkonis PA, Houck PR, Miewald JM, Buysse DJ. Symptom reports in severe chronic insomnia. *Sleep* 2002;**25**:553–63.
116. Broman JE, Hetta J. Perceived pre-sleep arousal in patients with persistent psychophysiologic insomnia and psychiatric insomnia. *Nord J Psychiatry* 1994;**48**:203–7.
117. Riemann, Voderholzer U. Primary insomnia: a risk factor to develop depression? *J Affect Disord* 2003; **76**:255–9.
118. Cole MG, Dendukuri N. Risk factors for depression among elderly community subjects: a systematic review and meta-analysis. *Am J Psychiatry* 2003;**160**:1147–56.
119. Bradley BP, Mogg K, Lee S. Attentional biases for negative information in induced and naturally occurring dysphoria. *Behav Res Ther* 1997;**35**:911–27.
120. Posner MI, Peterson SE. The attention system of the human brain. *Annu Rev Neurosci* 1990;**13**:25–42.
121. Posner MI, Petersen SE, Fox, Raichle ME. Localisation of cognitive operations in the human brain. *Science* 1988;**240**: 1627–31.
122. Broomfield NM, Turpin G. Covert and overt attention in trait anxiety: a cognitive psychophysiological analysis. *Biol Psychol* 2005;**68**:179–200.
123. Fox, Russo R, Bowles R, Dutton K. Do threatening stimuli draw or hold attention in sub clinical anxiety? *J Exp Psychol Gen* 2001;**130**:681–700.
124. Fox, Russo R, Dutton K. Attentional bias for threat: evidence for slowed disengagement from emotional faces. *Cognit Emotion* 2002;**16**:355–79.
125. Mathews, Mogg K, Kentish J, Eysenck M. Effects of psychological treatment on cognitive bias in generalised anxiety disorder. *Behav Res Ther* 1995;**33**:293–303.
126. Mogg K, Bradley BP, Millar N, White J. Cognitive bias in generalised anxiety disorder: a follow up study. *Behav Res Ther* 1995;**33**:927–35.
127. Wells, White J, Carter K. Attention retraining: effects on anxiety and beliefs in panic and social phobia. *Clin Psychol Psychother* 1997;**4**:226–32.

128. Cook EW, Turpin G. Differentiating orienting, startle and defense responses: the role of affect and its implications for psychopathology. In: Lang PJ, Balabam M, Simons RF, editors. *Attention and orienting: sensory and motivational processes*. Hillsdale, NJ: Erlbaum; 1997.
129. Perlis ML, Smith MT, Orff HJ, Andrews PJ, Giles DE. Beta/gamma activity in patients with primary and secondary insomnia and good sleeper controls. *Sleep* 2001;**24**:110–7.
130. Devoto A, Vialano C, Lucidi F, Lombardo C. P300 amplitude in subjects with primary insomnia is modulated by their sleep quality. *J Psychosom Res* 2003;**54**:3–10.
131. Devoto A, Mangelli S, Lucidi F, Lombardo C, Russo PM, Violani C. Quality of sleep and P300 amplitude in primary insomnia: a preliminary study. *Sleep* 2005;**28**:863–9.
132. Buysse DJ, Reynolds CF, Monk TH, Berman SR, Kupfer DJ. The pittsburgh sleep quality index: a new instrument for psychiatric practice and research. *Psychiatry Res* 1989;**28**:193–213.
133. Langford GW, Meddis R, Pearson AJD. Awakening latency from sleep for meaningful and non-meaningful stimuli. *Psychophysiology* 1974;**11**:1–6.
134. Akerstedt T, Billiard M, Bonnet M, Ficca G, Garma L, Mariotti M, et al. Awakening from sleep. *Sleep Med Rev* 2002;**6**:267–86.

Attention Bias for Sleep-Related Stimuli in Primary Insomnia and Delayed Sleep Phase Syndrome Using the Dot-Probe Task

Kenneth M A MacMahon, PhD; Niall M Broomfield, PhD; Colin A Espie, PhD

University of Glasgow Sleep Research Laboratory and Section of Psychological Medicine, Sackler Institute of Psychobiological Research, Southern General Hospital, Glasgow, Scotland, UK

Study Objectives: Cognitive models of primary insomnia (PI) suggest attention bias as a maintaining process. This study used a hallmark measure of attention bias, the dot-probe task, to determine whether attention bias to sleep-related stimuli is present in individuals with PI. Control groups of good sleepers (GS) and individuals with delayed sleep phase syndrome (DSPS), a sleep disorder with no presumed cognitive pathway and, hence, no predicted association with attention bias, were included.

Design: A between-groups (PI, DSPS, GS) design was employed. Participants completed a dot-probe task with stimuli comprising sleep-related and neutral words, balanced for length and frequency of usage. It was predicted a priori that PI would show greater attention bias to sleep stimuli compared with GS and DSPS groups. No difference between GS and DSPS was predicted.

Participants: Sixty-three individuals completed the study (PI = 21; DSPS = 22; GS = 20), with those in PI and DSPS classified by International Classification of Sleep Disorders criteria according to self-report sleep diaries and actigraphy. GS scored < 5 on the Pittsburgh Sleep Quality Index, reported being good sleepers, and met no criteria for a current or previous sleep disorder.

Interventions: N/A.

Measurements and Results: As predicted, PI showed increased vigilance for sleep-related stimuli relative to GS and DSPS. No differences between GS and those with DSPS were found. The PI group showed shorter response latencies relative to the GS and DSPS groups.

Conclusions: Results support an association between attention bias and PI. Further work must determine whether or not attention bias is a causal factor. Speeded responses in the PI group suggest heightened arousal, indicating that physiologic factors may play a related role.

Keywords: Attention bias, insomnia, delayed sleep phase syndrome, cognitive arousal, circadian

Citation: MacMahon KMA; Broomfield NM; Marchetti LM et al. Attention bias for sleep-related stimuli in primary insomnia and delayed sleep phase syndrome using the dot-probe task. *SLEEP* 2006;29(11):1420-1427.

INTRODUCTION

INSOMNIA IS A FREQUENT HEALTH COMPLAINT, WITH UP TO 33% OF THE GENERAL POPULATION REPORTING SLEEP DIFFICULTIES AT ONE TIME OR another and 9% reporting insomnia on a regular nightly basis.[1] Insomnia can occur as a secondary consequence of physical or mental illness but also as a primary disorder in itself.[2,3] Primary Insomnia (PI) is characterized as a disorder with the main complaint of disturbed or unrefreshing nocturnal sleep and associated daytime impairment, unrelated to a medical or mental disorder.

An interplay of psychological and physiologic factors have been implicated in the development and maintenance of PI, for example, higher metabolic rates, increased nocturnal melatonin secretion,[4,5] heightened cognitive arousal, and dysfunctional beliefs about sleep in those with PI.[6,7] Given that sleep disturbance may be seen as a considerable anxiety generator to an individual with PI, behavioral formulations of PI suggest that repeated episodes of sleep difficulties may lead to the poor sleeper developing conditioned arousal to the bedroom environment, thus exacerbating existing sleep difficulties. Recent cognitive models of PI[8-10] have taken this notion further and suggest that the poor sleeper is selectively vigilant for internal stimuli indicative of wakefulness (when trying to sleep) and symptoms of fatigue (during daytime activities[11]). Any indication of difficulties in either of these areas serves to heighten anxiety about sleep difficulties and their consequences,[12] thus leading to a redoubling of efforts to sleep and, paradoxically, reduced probability that sleep will occur.[13,14]

Selective vigilance, or "attention bias," has been a focus of investigation in psychopathology for several years and is well established as a maintaining mechanism in several disorders, such as social phobia or panic disorder.[15,16] However, at present, there are only 3 published studies that examine attention bias in PI, with equivocal findings that provide only limited evidence that sleep-related attention bias is present in those with PI.

Attention Bias in PI

Lundh et al,[17] using the emotional Stroop task, found that participants with PI took relatively longer to name colors in trials containing sleep-related words, in comparison to neutral words, indicating an interference effect of sleep-related stimuli. However, they also found equivalent effects in "normal sleepers," suggesting some degree of priming across the groups, or the presence of sleep difficulties in the "normal sleepers." Taylor et al[18] found a relatively heightened interference effect on the Stroop task for sleep-related words in a population with persistent insomnia (12 to 18 months), in comparison with a group with acute insomnia (up to 3 months). However, both of these groups were recovering cancer patients, and no control group of good sleepers, without medical problems, was employed.

Nonetheless, perhaps the most compelling evidence of atten-

Disclosure Statement

This was not an industry supported study. Drs. MacMahon, Broomfield, Espie have indicated no financial conflicts of interest.

Submitted for publication June 15, 2005
Accepted for publication June 12, 2006

Address correspondence to: Colin Espie, PhD, University of Glasgow Sleep Research Laboratory and Section of Psychological Medicine, Sackler Institute of Psychobiological Research, Southern General Hospital, 1345 Govan Road, Glasgow G51 4TF, Scotland, UK; Tel: 00 44 141 211 3903; Fax: 00 44 141 357 4899; E-mail: c.espie@clinmed.gla.ac.uk

tion bias in PI was provided in a recent study by Jones et al.[19] They used a novel "flicker" paradigm,[20] involving repeated brief presentations of a visual scene with a single feature (either sleep or neutral) altered on each alternate presentation. Participants defined as "poor sleepers" (> 5 on the Pittsburgh Sleep Quality Index [PSQI][21]) detected a change in a scene related to a "sleep" object (1 of a pair of slippers), significantly quicker than a non-sleep object (1 of a pair of gloves). Furthermore, a significant correlation between PSQI and detection latency for the sleep-related change was observed. There are some drawbacks with this study, most notably the use of a between-subjects design, with no assessment of whether individuals showed relatively greater bias toward the sleep-related change. Jones et al[19] also used only a single object, rather than several exemplars of a class, and classification of poor sleepers as suffering from PI was inferred purely on the basis of PSQI scores, with no clinical interview to confirm diagnosis. Although studies in the addiction literature suggest that the flicker task is a valid measure of attention bias,[22] there is little in the general psychopathology literature on its use at the present time.

Overcoming Limitations of Previous Work

The study reported in this article attempted to establish whether attention bias is associated with PI by overcoming some of the limitations of previous work in this area. Firstly, the dot-probe paradigm, a well-established "hallmark" measure of attention bias, was employed.[23] In this task, pairs of words are simultaneously presented, 1 above the other, on a computer screen, and the ensuing distribution of visual attention is measured by recording detection latency for a visual probe that appears in the spatial location of either word, immediately after the display of that word has terminated. Thus, the task bypasses limitations of the Stroop by using a neutral response (key press) to a neutral stimulus (visual probe). The trials providing the data of interest are those in which 1 of the words has emotional salience. By examining the impact of such a word on the relative probe-detection latencies in the 2 spatial areas, it is possible to determine whether visual attention has shifted toward or away from such stimuli.

In order to address the difficult question of whether attention bias is associated specifically with PI, and not simply with sleep disturbance, a control group of individuals with delayed sleep phase syndrome (DSPS) was employed. DSPS is, like PI, characterized by difficulties in initiating sleep.[24] Although the cause of this difficulty, in PI, is presumed to involve psychological factors, DSPS is believed to be the result of a circadian timing disorder. Because there is no presumed cognitive pathway to explain the emergence or maintenance of DSPS, it was predicted that those individuals who suffer from DSPS would not show attention bias to sleep-related stimuli.

A further limitation of previous studies in the sleep disorders literature has been a lack of clear differentiation between participants with PI and those who may have a mixed pattern of DSPS and PI.[25,26] Thus, objective (actigraphic) assessment of circadian sleep parameters has been used in tandem with subjective (self-report sleep log) assessment to differentiate between DSPS and PI in poor sleepers. Many sleep studies also define control participants as not "obvious" sufferers of insomnia,[17] possibly leading to the inclusion of "noncomplaining" poor sleepers. Therefore, a further control group, who actively describe themselves as "good sleepers" (GS), rather than those who report "no sleep difficulties," was used.

Aims and Predictions

Aims

This study examined whether individuals with PI show an attention bias toward sleep-related stimuli on the dot-probe task in comparison with control groups of GS and those with DSPS.

Predictions

(1) Participants with PI will show attention bias toward sleep-related stimuli in the form of reduced latency in dot-probe reaction time to sleep-related words, in comparison with neutral words.
(2) Neither DSPS nor GS will show an attention bias toward sleep-related stimuli in the form of increased latency in dot-probe reaction time to sleep-related words, in comparison with neutral words.

Ethics

Ethical approval for the study was sought from, and granted by, Greater Glasgow Primary Care Trust NHS Ethics Committee.

METHODS

Participants

An e-mail message sent round the student e-mail system requested those with "sleep problems," "night owls," or "good sleepers" to contact the first author. An initial, informal, screening process by e-mail or telephone was used to assess whether respondents were likely to meet necessary criteria. Seventy-nine individuals appeared to meet criteria and were invited to take part in the study.

Participants met Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition[2] and International Classification of Sleep Disorders-Revised[3] criteria for either PI or DSPS, and those with PI were required to score > 6 on the PSQI. Exclusion criteria included active psychological or drug interventions for sleep problems or when a sleep disorder was suspected as being the result of substance misuse. GS scored < 5 on the PSQI, reported themselves as being "good" sleepers, and met no criteria for a sleep disorder at the present time or in the past. Actigraphy was used as an aid to differentiate between PI and DSPS in those reporting sleep difficulties. All participants had normal vision or corrected to normal vision.

Final allocation of participants to groups for the purposes of analysis did not take place until all sleep-assessment data had been collated. No formally screened participants were excluded due to either psychological or drug interventions for sleep or because of substance misuse. However, 15 participants were excluded for the following reasons: failure to return all questionnaire measures (4); error score (making an upper key press for a lower target word and vice versa) on the dot-probe task above 5% threshold (1), "good sleepers" with PSQI scores > 4 (6), sleep difficulties were a combination of PI and DSPS (2), and "poor sleepers" who did not meet threshold of > 6 on the PSQI (2). One further participant (a "good sleeper") was excluded as an "outlier" because her results fell more than 2 standard deviations from her group mean.

This left a total of 63 participants who met all relevant criteria

and completed all measures and who were classified into PI (n = 21), DSPS (n = 22), or GS (n = 20) groups. A power calculation (using data from previous studies of attention bias[17,18,27]) carried out prior to the study suggested that 21 participants would be required in each of the 3 groups (PI, DSPS and GS) to detect statistically significant differences at a power of .8 with an α level set at .05.

Apparatus

The dot-probe task was run on a Dell (Bracknell, UK) Optiplex GX270 computer, with software developed by the first author on the experiment-generating package, SuperLab Pro (Version 2.02; Cedrus Corporation; San Pedro, CA). The size of the screen was 28 cm (diagonally), with stimuli in 14-point Times New Roman font in white lettering on black background, positioned centrally, on a vertical plane; the viewing distance was 45 cm. Millisecond timing was made through SuperLab Pro and an external response box (Model RB-400; Cedrus Corporation; San Pedro, CA) to avoid timing errors associated with standard keyboard or mouse input.

Actigraphy was facilitated by the Cambridge Neurotechnology (Cambridge, UK) Actiwatch system. This comprises wrist-worn actigraphs (Model AW-2), roughly the size of small wristwatches, and sleep-analysis software (Actiwatch Sleep Analysis v 1.06), capable of automatically calculating sleep parameters. There is general consensus that wrist actigraphy provides an accurate objective measure of circadian sleep and wake parameters.[28]

Materials

A stimuli list comprising 88 words was developed for this study (see Table 1). Firstly, 8 neutral, non–sleep-related practice words were divided into 4 pairs (leisure/gallant; poetry/joyful; welcome/capable; prizes/secure). Secondly, a list of 20 sleep-related words were matched to 20 neutral words. The 20 sleep-related words were originally used by Taylor et al[18] in their study of insomnia in patients recovering from cancer. Taylor et al[18] developed these words from Wicklow and Espie's[29] study of the content of pre-sleep cognitions, and, thus, they have a high degree of salience to individuals with sleep difficulties. Finally, 60 further neutral, non–sleep-related words were selected and divided into matched pairs. Although each pair of words was used on 2 occasions in the study, repeated word pairs have been used before in this paradigm with no priming effects found.[30]

Matching of words into pairs was carefully done to ensure equivalence in length and frequency of usage in the English language. The latter was achieved by consulting frequency tables constructed from the British National Corpus, a representative sample of present-day spoken and written British English[31](http://www.comp.lancs.ac.uk/ucrel/bncfreq). Analysis-of-variance (ANOVA) across the 4 sets of word pairs for frequency of occurrence in the British National Corpus showed no significant differences ($F_{3,76}$ = 0.025, p = .995).

Table 1—List of Words for Dot-Probe Task With Frequency of Occurrence per Million in the British National Corpus

Sleep Words	Frequency	Neutral Words	Frequency
Tired	41	Clubs	38
Exhausted	15	Impatient	7
Fatigue	5	Paddock	2
Dream	46	Grass	41
Bed	159	Red	149
Sheets	24	Models	53
Wakeful	0 (20)	Shuffle	3
Silence	3	Texture	9
Pillow	7	Scents	2
Naps	0 (14)	Pear	2
Dark	135	Hour	113
Alert	16	Linen	10
Snoring	0 (25)	Consist	12
Overactive	0 (22)	Quantifies	0 (11)
Arousal	4	Reforms	0 (30)
Sleepy	4	Absurd	10
Night	365	Money	374
Restless	7	Youthful	0 (5)
Tossing	2	Empower	1
Lethargy	1	Kinetics	2
Mean ± Sd	41.7 ± 87.95	Mean ± Sd	41.4 ± 87.96
Neutral Words	**Frequency**	**Neutral Words**	**Frequency**
Graft	19	Males	22
Featuring	9	Scrubbing	2
Cushion	5	Specify	13
Meals	24	Chord	6
Led	154	Lot	246
Stairs	36	Mirror	39
Invoice	5	Prodigy	1
Playful	2	Tramped	1
Margin	15	Pencil	12
Tops	10	Laps	3
Book	248	News	145
Habit	23	Steel	37
Sandals	3	Potters	1
Attendants	2	Speculates	0 (27)
Invents	0 (20)	Drafted	7
Graft	2	Disarm	1
Point	402	Water	350
Thankful	4	Entrants	5
Vaccine	4	Embrace	10
Wriggles	0 (1)	Fixation	2
Mean ± Sd	48.4 ± 103.45	Mean ± Sd	45.2 ± 93.81

Note: When frequency of occurrence is less than 1 per million, as with several of the chosen sleep words, the range of sections in the British National Corpus (out of a maximum of 100) in which the word occurs is taken as a measure of frequency of usage; this range is given in parentheses in this table.

Self-Report Measures

Demographic Measures

(1) Beck Depression Inventory-Short-Form (BDI-SF[32]) to assess for symptoms of depression.
(2) Spielberger State and Trait Anxiety Inventory (S-SAI/ S-TAI[33]) to assess levels of state and trait anxiety.
(3) National Adult Reading Test (NART[34]) to provide an estimate of reading vocabulary and ensure that participants' reading level was sufficient for the stimuli used in the dot-probe task.
(4) Caffeine Intake Questionnaire, an idiosyncratic questionnaire, to assess usual and actual recent intake of caffeine, comprising 3 questions examining caffeine intake in beverages in past 24 hours, the day so far, and usual caffeine intake. Intake was divided into units according to the caffeine content of these beverages.

Sleep Measures

(1) PSQI[21] to assess for presence and severity of sleep disturbance.
(2) Anxiety and Preoccupation about Sleep Questionnaire (APSQ[35]) to assess for specific worry about sleep difficulties.
(3) Morningness-Eveningness Questionnaire-Reduced Form (MEQ[36]) to assess preference for "morningness" or "eveningness" as an aid to classification of sleep disorder as PI or DSPS.
(4) Stanford Sleepiness Scale (SSS[37]) to quantify level of alertness during testing.

PROCEDURE

Following recruitment and screening by e-mail or telephone, potential participants were met at the University. All testing was conducted in a quiet private room in the Department of Psychology. Subjects were given a brief written summary outlining the purpose of the study, stating that it was to "understand some of the reasons why people have difficulty sleeping." No other details on the purpose or hypotheses lying behind the study were given at this stage and what would be required of them if they agreed to participate. Informed consent was then obtained. No financial remuneration nor course credits were offered for participation.

Participants were then asked to complete the dot-probe task. Each participant viewed a set of instructions, on screen, and the task was explained verbally to ensure that it was fully understood. Following this, participants completed 4 practice trials, followed by the remaining 160 experimental trials. Pairs of words were presented in a random order, with rerandomization of the order for each participant, to ensure that order effects did not confound results. Each trial consisted of the following events: a fixation cross appeared in the center of the screen for 500 milliseconds, followed by the pair of words for 500 milliseconds; the words then disappeared, and a dot-probe, consisting of an asterisk, appeared in either the upper or lower position; this remained on screen until the correct response key (upper or lower) was pressed. Half of the trials contained a sleep-related word, and there was an equal probability that the probe would replace the sleep-related or the neutral word. The relative positions (upper or lower) of each pair of words were also counterbalanced within the experiment. An intertrial interval of 1000 milliseconds followed each trial.

A brief screening interview was then conducted to assess sleep history and general psychological state. This was done after the experimental task to minimize priming for sleep difficulties on the dot-probe. The screening interview covered items in Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition and International Classification of Sleep Disorders criteria and the differential diagnosis of alternative sleep disorders, such as sleep apnoea and general mood state. Participants then completed the NART, the MEQ, and the SSS with the experimenter and the S-SAI and CIQ by themselves. All participants then completed the PSQI, APSQ, BDI-FT, and S-TAI at home and filled-out a sleep diary each night over the next week. Those who reported sleep difficulties were also asked to wear an Actiwatch day and night for this week.

On their return visit, approximately 1 week hence, participants were fully debriefed as to the purpose of the study, and their sleep data was reviewed. All participants were given a copy of the Good Sleep Guide,[38] and those who complained of either PI or DSPS were given some general advice, based on the Good Sleep Guide, in managing their sleep difficulties.

RESULTS

Participant Characteristics

Mean age across all participants was 24.4 years (SD = 7.44 years), with a total of 35 females and 28 males. In terms of group differences, ANOVA and post-hoc testing with the Tukey Honestly Significant Differences (HSD) test demonstrated that the GS group was significantly older than the DSPS group (see Table 2 for data). Given this finding, further analysis between groups used analysis-of-covariance (ANCOVA), with age as a covariate, followed by Tukey HSD test. ANCOVA and Tukey HSD across the 3 groups revealed significantly lower mean levels of state-anxiety, trait-anxiety, and depression in the GS group (28.7, 31.5, and 0.5, respectively) in comparison with those with PI (37.9, 47.7, and 3.6, respectively) or DSPS (35.6, 46.1, and 4.0, respectively). No statistical difference was present between the PI and DSPS groups on these measures. No significant differences in vocabulary level emerged between any of the groups. No difference between groups in either regular or actual recent caffeine intake was reported either (all p values > .3).

Sleep-Related Questionnaire Measures

The GS group scored significantly lower on the PSQI and APSQ than either the PI or DSPS groups. Individuals in the DSPS group were rated, by the MEQ, as significantly more "evening" type than the other 2 groups (lower scores on the MEQ reflect a greater tendency to "eveningness"). On average, participants in the PI and DSPS groups report themselves to be significantly less alert (as measured by the SSS) than their GS counterparts.

Table 2—Participant demographics within the PI, DSPS, and GS

Demographic	PI (n = 21)	DSPS (n = 22)	GS (n = 20)	$F_{2,59}$[a]/ χ^2 (2)	p Value
Age (years)	23.6 ± 6.9	21.8 ± 2.2	28.2 ± 10.1	4.50 ‡†	.02
Sex, male/ female	7/14	12 /10	9/11	1.96 ‡†	.38
BDI-FT	3.6 ± 3.1	4.0 ± 3.6	0.5 ± 0.7	5.90 ‡†	.005
S-SAI	37.9 ± 8.3	35.6 ± 7.3	28.7 ± 7.1	5.95 ‡†	.004
S-TAI	47.7 ± 11.4	46.1 ± 9.7	31.5 ± 5.7	15.54 ‡†	< .001
NART	13.5 ± 7.7	10.8 ± 3.7	11.4 ± 4.7	1.38	.26
PSQI	9.8 ± 2.4	7.4 ± 2.4	2.6 ± 1.9	46.60 ‡†	< .001
APSQ	56.9 ± 17.7	53.9 ± 15.5	20.2 ± 11.1	30.78 ‡†	< .001
MEQ	11.5 ± 3.5	8.6 ± 2.6	13.8 ± 4.4	7.55*†	.001
SSS	2.9 ± 1.2	2.8 ± 0.9	2.1 ± 0.8	5.04 ‡†	.01

Data are presented as mean ± SD unless otherwise indicated.
[a]Age $df_{2,60}$
PI refers to primary insomnia; DSPS, delayed sleep-phase syndrome; GS, good sleeper; BDI-FT; Beck Depression Inventory-Fast Track; S-SAI, Spielberg State Anxiety Inventory; S-TAI, Spielberg Trait Anxiety Inventory; NART, National Adult Reading Test; PSQI, Pittsburgh Sleep Quality Index; APSQ, Anxiety and Preoccupation about Sleep Questionnaire; R-HOMEQ, Horne and Ostberg Morningness-Eveningness Questionnaire; SSS, Stanford Sleepiness Scale
*Indicates significant ($p < .05$) difference between PI and DSPS with Tukey posthoc Test.
‡Indicates significant ($p < .05$) difference between PI and GS with Tukey posthoc Test.
†Indicates significant ($p < .05$) difference between DSPS and GS with Tukey posthoc Test.

However, SSS scores did not correlate directly with the dot-probe performance measures.

Sleep Data

Sleep Diary

ANCOVA, with age as a covariate, across groups for subjective bed time, arise time, sleep-onset latency, wake after sleep onset, and total sleep time all revealed significant ($p < .05$) differences across the groups (see Table 3 for data). Posthoc testing with the Tukey HSD test confirmed that the DSPS group reported going to bed and rising from bed significantly later than the other 2 groups. Both PI and DSPS groups reported longer sleep-onset latencies than did the GS group, although they did not differ between themselves. WASO and number of nighttime awakenings were significantly greater in the PI group in comparison with only the GS group. Total sleep time was equivalent across all 3 groups.

Actigraphy

Actigraphy aided in making the diagnosis between PI and DSPS in individual cases. Furthermore, nonparametric circadian-rhythm analyses[39] of L5 (time of onset of lowest 5 hours of activity) and M10 (time of onset of highest 10 hours of activity) confirmed that the phases of lowest and highest activity were, on average, significantly delayed (by around 2 hours) in the DSPS group, in comparison with the PI group (see Table 4 for data).

Preparation of Response-Time Data

Response times (RTs) from trials with errors were excluded, and those more than 2 SD above the mean were discarded as outliers.[15] As detailed earlier, 1 participant had an error rate above 5% and was therefore excluded from all analyses, and 1 further participant was excluded as an "outlier," with her results lying more than 2 SD from her group mean.

ANCOVA, with age as a covariate, confirmed that the groups did not differ significantly ($F_{2,59} = 1.97$, $p = .15$) on error rate (1.5% of total data) or in outliers ($F_{2,59} = 1.62$, $p = .21$), which comprised a further 3.1% of the remaining data (see Table 5 for data). However, significant differences between the groups ($F_{2,9670}$) = 151.17, $p < .001$) were evident on mean RT, with posthoc testing confirming that the PI group responded more quickly to all stimuli than the GS group who, in turn, responded more quickly to all stimuli than the DSPS group (see Table 5 for data).

Table 5 also details mean RT for each stimuli combination (position of sleep word and position of probe). In order to assess whether systematic differences were present between groups and stimuli combinations, an attention bias score was calculated, in accordance with general practice in this literature.[40] The attention bias score was calculated using the following equation: attention bias score = [(SleepUProbeL+SleepLProbeU)-(SleepUProbeU + SleepLProbeL)]/2 where Sleep = sleep-related word; U = upper; and L = lower e.g., SleepUProbeL represents the mean RT when the sleep-related word is in the upper position and the probe is in the lower position). The attention bias score summarizes the interaction between sleep-word position and probe position on RT, providing a measure of the relative speeding of RT to probes that appear in the same location as sleep-related words. Positive values reflect vigilance for sleep words relative to neutral words, whereas negative values reflect avoidance.

Mean and SD attention bias scores were +3.9 ± 9.4 for the PI group; +1.1 ± 8.3) for the DSPS group; and -2.5 ± 9.70 for the GS group.

It was predicted, a priori, that a greater degree of attention bias to sleep-related stimuli would be shown by the PI group in comparison with the DSPS and GS groups. Orthogonal contrasts, with an assumption of equal variances, on attention bias scores supported this prediction ($t_{60} = -1.88$, $p = .03$).

It was also predicted, a priori, that the DSPS and GS groups would show an equivalent degree of attention bias, ie, no significant difference would be observed. This prediction was also upheld ($t_{60} = -1.27$, $p = .10$).

Finally, the Pearson product-moment correlation coefficient was calculated between attention bias score and PSQI score to establish whether an underlying association existed across all participants. Although a positive correlation emerged, it did not reach statistical significance ($r = +.19$; $p = .13$).

Table 3—Sleep-Diary Parameters for the PI, DSPS and GS Groups

Parameters	PI (n = 21)	DSPS (n = 22)	GS (n = 20)	$F_{2,60}$	p value
Bed Time	00:38 (46.1)	02:40 (83.0)	00:24 (55.5)	24.71*†	< .001
Arise Time	09:18 (51.3)	11:10 (72.7)	08:32 (87.1)	21.48*†	< .001
SOL	40.0 ± 20.8	31.1 ± 15.3	10.2 ± 8.8	15.37*†	< .001
WASO	21.5 ± 29.0	18.7 ± 18.1	3.9 ± 5.3	5.58‡	.006
TST	442.0 ± 80.2	468.0 ± 65.8	474.1 ± 57.6	1.73	.19
Awakenings	2.2 ± 2.1	1.33 ± 1.0	1.0 ± 1.2	5.28‡	.01

Data are presented as mean ± SD except for bed time and arise time, which are presented as mean 24-h clock times with SD of minutes in parentheses
PI refers to primary insomnia; DSPS, delayed sleep-phase syndrome; GS, good sleeper; SOL, sleep-onset latency; WASO, wake time after sleep onset; TST, total sleep time; Awakenings, number of awakenings during the night
*Indicates significant ($p < .05$) difference between PI and DSPS with Tukey posthoc Test.
‡Indicates significant ($p < .05$) difference between PI and GS with Tukey posthoc Test.
†Indicates significant ($p < .05$) difference between DSPS and GS with Tukey posthoc Test.

DISCUSSION

Recent cognitive models of insomnia[8,10] predict that individuals with PI, in comparison with GS, will demonstrate heightened vigilance for sleep-related stimuli. Results from this study provide support for this prediction. Those with PI, in comparison

Table 4—Actigraphy L5 and M10 parameters for PI and DSPS

Parameters	PI (n = 21)	DSPS (n = 22)	t_{41}	p value
L5	2:26 (61.7)	4:22 (70.6)	-5.73	< .001
M10	11:40 (131.9)	13:11 (136.9)	-2.22	.03

Data are presented as mean 24-h clock times with SD of minutes in parentheses. PI refers to primary insomnia; DSPS, delayed sleep-phase syndrome.

Table 5—Overall Response Times, Error Scores, and Response Times for Word-Pairs

	PI (n = 21)	DSPS (n = 22)	GS (n = 20)	$F_{2,59}$/ $F_{2,9670}$	p value
Errors	3.5 ± 4.1	2.0 ± 1.8	2.0 ± 2.1	1.97	.15
Overall RTs	384.2 ± 71.3	410.8 ± 75.4	397.1 ± 72.4	151.2*†‡	< .001
SleepU ProbeU	384.8 ± 73.7	410.1 ± 77.3	402.5 ± 75.9		
SleepU ProbeD	388.5 ± 66.6	409.1 ± 73.5	394.0 ± 66.9		
SleepD ProbeU	386.0 ± 68.7	410.9 ± 74.8	397.0 ± 72.7		
SleepD ProbeD	380.5 ± 75.2	410.1 ± 74.8	394.2 ± 69.1		

Data are presented as mean ± SD unless otherwise indicated. All response times (RT) are shown with errors firstly removed. PI refers to primary insomnia; DSPS, delayed sleep-phase syndrome; GS, good sleeper.
*Indicates significant ($p < .05$) difference between PI and DSPS with Tukey posthoc Test.
‡Indicates significant ($p < .05$) difference between PI and GS with Tukey posthoc Test.
†Indicates significant ($p < .05$) difference between DSPS and GS with Tukey posthoc Test.

with GS, showed relatively greater vigilance, as measured by the dot-probe task, for sleep-related stimuli. Although a positive correlation between attention bias score and PSQI score (a measure of sleep quality) was demonstrated, it did not approach statistical significance. Nonetheless, results provide support for the specific prediction, from cognitive models of PI, that suggest attention bias is a component of this disorder.[8,10]

Two other findings emerged. Firstly, participants with DSPS, in comparison with GS, did not show significant evidence of attention bias, as predicted. A second, notable, finding was the significantly shorter RTs from the PI group, in comparison with GS and, in turn, with DSPS (see Table 5). There were no significant differences in overall error rates that might explain this as a speed-accuracy trade off (indeed, overall error rates were low; see Table 5). It is possible to speculate that those with DSPS are less alert (as shown by their higher SSS scores) than their GS peers, but this is unlikely to explain slowing because SSS scores per se did not predict dot-probe data. However, the speeded responses (in spite of their equally high SSS scores) of the PI group may be a reflection of increased arousal levels in this population, something that has been demonstrated in studies of physiologic aspects of PI.[4,5]

Self-report ratings in the DSPS group are also of some interest, with unexpectedly high levels of state-anxiety, trait-anxiety, and worry about sleep (see Table 2). Although differentiated, in this study, on a case-by-case basis from those with PI, there is clearly some overlap between the disorders. This raises the question of whether those with DSPS can form a "pure" enough control group against which to compare those with PI on psychological factors.[41] Indeed, some authors have suggested that circadian dysfunction may act as a precipitating mechanism in PI.[42] Interestingly, in this study, there does appear to be some degree of phase delay in the PI group, with a relatively late L5 onset of 2:26 AM (see Table 4), although it should be borne in mind that the use of a relatively young University sample suggests that social factors may be a causal factor in this late L5. Perhaps future studies would be best served by screening more carefully for DSPS but still exercising caution in the interpretation of results from this group, with the assumption that some overspill of PI may be inevitable.

There are several limitations in the current study that should be considered. Firstly, the sample was drawn from a university population, thus narrowing the age and education range of the participants (see Table 2). Furthermore, students have greater flexibility to adjust work schedules than do those in regular (9:00 AM to 5:00 PM) employment.[43] Thus, failure to sleep at night, or perform at a set time of day, may pose less of a threat to (and hence less of a focus of attention for) both the PI and DSPS groups, than would occur in a comparative sample with regular employment. Thus, potentially, this study may underestimate the degree of attention bias in both of these groups. However, participants met both Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition and International Classification of Sleep Disorders-Revised criteria for PI, and the mean PSQI scores for PI and DSPS groups were well above the mean for the GS group (9.8 and 7.4, respectively, versus 2.6). Furthermore, mean APSQ scores are comparable to those of a recent study on PI[44] and indicate a significantly greater degree of worry about sleep in the PI group and DSPS groups in comparison with the GS group (56.9 and 53.9, respectively, versus 20.1). In this case, the comparative levels of concern about sleep in the DSPS and PI groups are not reflected in the degree of attention bias, further supporting the argument that cognitive factors may play a role in PI, whereas physiologic factors may underpin DSPS.

Secondly, although this is a positive finding, discrepant findings are not uncommon in the attention-bias literature, with the role of mood state often implicated in this.[45] For example, in a study using the dot-probe task, Mogg et al[46] failed to find evidence of attention bias to exam-related stimuli in a high trait-anxious population when testing was conducted out with the exam period. However, when exams were imminent, the processing bias appeared. Again, it is feasible that a larger effect would have been seen in our study if participants were tested at a time when sleep loss was a salient threat; for example, in the evening prior to an examination or other important event the following day. A testing laboratory in the middle of the day, with no cost attached to "poor" performance on the experimental task, may fail to activate dormant schema. However, when loss of sleep is imminently threatening, such as in the early hours of the morning, or when performance on a task is particularly valued, increased vigilance for indicators of sleep readiness (at night) or negative consequences of sleep loss (in the daytime) may be observed. This leads to a further limitation of the present study. It was not possible, for both practical and theoretical reasons, to adjust time of testing for participants' circadian rhythms. Testing on the dot-probe task was conducted at the first available opportunity; hence, actigraphic assessment (which could potentially have provided information to set testing times according to individual circadian rhythms) followed the testing session. To wait until actigraphic assessment had been completed and then set specific testing times would have significantly increased the demands on participant time and would also have raised the possibility of priming to sleep-related

stimuli. Nonetheless, this is something that would be of value to consider in future studies.

One potential limitation of this study was the inclusion of sleep-related words that might not be considered "sleep-threatening," such as "pillows" or "nap." However, the words used in this study were taken directly from previous work that elicited presleep cognitions of individuals with PI.[29] Thus, it was predicted that they would be selectively processed by individuals with attention bias to sleep-related stimuli. Indeed, even if the word sets used in this study were not considered to be sufficiently "threatening," this suggests that a magnified effect could be detected if alternate word sets were used. At the time of study development, this word set, taken directly from presleep cognitions, was felt to provide the most ecologically valid option. Furthermore, the DSPS group reported sleep difficulties equal to those of the PI group (as evidenced by PSQI scores, see Table 2), yet did not show equivalent attention bias scores, suggesting that the word set used in this case appropriately discriminated by sleep disorder, rather than simply reflecting the degree of "interest" expressed by individuals in sleep problems.

Alternate grouped categories of neutral words (eg, household items or animals) have been used in some studies of attention bias to limit the possibility that bias is shown as a result of target words falling into categories, rather than actual category itself being differentially processed. The lack of categorized neutral words is a noted limitation of the present study. However, no effect of either neutral or sleep-related word lists was seen in the GS group, suggesting that the systematic bias seen in the PI group is unlikely to have been purely the result of using a category in itself. Nonetheless, future studies utilizing this paradigm should consider developing matched categories for neutral word lists.

Furthermore, although considered a relatively pure measure of attention,[16] the dot-probe task has also been described as a "relatively fragile index of anxiety related attentional biases in non-clinical studies, particularly when using word stimuli that have relatively mild threat value."[47] A possible lack of emotional salience in the word-based stimuli may minimize the vigilance effect in the present study. Future studies might consider the use of a pictorial dot-probe paradigm to maintain the degree of experimental control available from the dot probe but maximize the possibility of uncovering processing biases by increasing the saliency of stimuli.[48] Furthermore, because RT is an indirect measure of vigilance, a future study might also consider the use of psychophysiologic measures of attention, such as heart rate[49] or eye tracking,[50] in order to add credence to results.

Notwithstanding these limitations, using a "hallmark" measure of attention bias and a carefully screened sample, our results offer evidence of attention bias in young people with PI; this is consistent with cognitive models of PI.[8,10] In particular, the results are in keeping with the attentional component of a proposed "attention-intention-effort pathway" in the development of psychophysiologic insomnia.[9] Our findings also provide some support for the neurocognitive model of PI.[51] This model suggests that those with PI have greater cortical arousal, something that may be reflected in the speeded RTs recorded in the present study. Notably, Perlis et al[51] did not make recourse to attention bias as a maintaining factor in their model. However, our results would suggest that both aspects may operate, possibly in a complementary fashion, during the development of PI.

Finally, if attention bias is to be considered as a key element in the development or maintenance of PI, and not merely as an associative factor, further work to demonstrate a direct link between attention bias and PI is necessary. For example, the repeat administration of the dot-probe task at intervals when sleep difficulties are waxing and waning might demonstrate a concurrent increase and decrease in attention bias. Perhaps of even more interest would be to determine whether increasing attention to sleep-related material would lead to an increase in sleep difficulties, as has been suggested in parallel studies in the anxiety literature.[52] Furthermore, measurement of attention bias following successful and unsuccessful treatment for PI would be helpful, particularly with regard to the impact of sleep medication (effective in the short term) in comparison with the relatively enduring effects of cognitive behavioral treatment.

Further work, utilizing pictorial stimuli, additional psychophysiologic measures, and carefully screened participants, is still necessary before definitive conclusions can be drawn on any potential role of attention bias in PI. However, if further evidence emerges to support the findings of this study, it would be an important step in finding an "objective" measure of PI. At present, the cognitive arousal reported by those with PI is only measured by self-report; attention-bias tasks may offer the possibility of objective measures, sensitive to the experience of PI.

REFERENCES

1. Ancoli-Israel S, Roth T. Characteristics of insomnia in the United States: results of the 1991 National Sleep Foundation Survey. I. Sleep 1999;22:S347-53.
2. The Diagnostic and Statistical Manual-4th ed. Washington: American Psychiatric Association; 1994.
3. The International Classification of Sleep Disorders: Revised. Rochester, MN: American Sleep Disorders Association; 1997.
4. Bonnet MH, Arand DL. Insomnia, metabolic rate and sleep restoration. J Intern Med 2003;254:23-31.
5. Riemann D, Klein T, Rodenbeck A, et al. Nocturnal cortisol and melatonin secretion in primary insomnia. Psychiatry Res 2002;113:17-27.
6. Morin CM, Stone J, Trinkle D, Mercer J, Remsberg S. Dysfunctional beliefs and attitudes about sleep among older adults with and without insomnia complaints. Psychol Aging 1993;8:463-67.
7. Watts F, Coyle K, East MP. The contribution of worry to insomnia. Br J Clin Psychol 1994;33: 211 - 20.
8. Espie CA. Insomnia: Conceptual Issues in the Development, Persistence, and Treatment of Sleep Disorder in Adults. Annu Rev Psychol 2002;53:215-43.
9. Espie CA, Broomfield NM, MacMahon KMA, Macphee LM, Taylor LM. The attention-intention-effort pathway in the development of psychophysiologic insomnia: a theoretical review. Sleep Med Rev 2006:10:215-45.
10. Harvey AG. A cognitive model of insomnia. Behav Res Ther 2002;40:869-93.
11. Semler C, Harvey AG. Monitoring for sleep-related threat: a pilot study of the sleep associated monitoring index (SAMI). Psychosom Med 2004;66:242-50.
12. Harvey AG, Greenall E. Catastrophic worry in primary insomnia. J Behav Ther Exp Psychiatry 2003;34:11-23.
13. Broomfield NM, Espie CA. Toward a valid, reliable measure of sleep effort. J Sleep Res 2006:In Press.
14. Broomfield NM, Gumley AI, Espie CA. Candidate cognitive processes in psychophysiologic insomnia. J Cogn Psychother 2006:In Press.
15. Mogg K, Philippot P, Bradley BP. Selective attention to angry faces in clinical social phobia. J Abnorm Psychol 2004;113:160-5.

16. Mogg K, Bradley BP. A cognitive-motivational analysis of anxiety. Behav Res Ther 1998;36:809- 48.
17. Lundh LG, Froding A, Gyllenhammar L, Broman JE, Hetta J. Cognitive bias and memory performance in patients with persistent insomnia. Scand J Behav Therapy 1997;26:27-35.
18. Taylor LM, Espie CA, White CA. Attentional bias in people with acute versus persistent insomnia secondary to cancer. Behav Sleep Med 2003;1:200-12.
19. Jones BT, Macphee LM, Broomfield NM, Jones BC, Espie CA. Sleep-related attentional bias in good, moderate and poor (primary insomnia) sleepers. J Abnorm Psychol 2005;114:249-58.
20. Rensink R, O'Regan J, Clark J. To see or not to see: The need for attention to perceive changes in scenes. Psychol Sci 1997;8:368-73.
21. Buysse D, Reynolds C, Monk T, Berman S, Kupfer D. The Pittsburgh Sleep Quality Index: a new instrument for psychiatric practice and research. Psychiatry Res 1989;28:193-213.
22. Jones BT, Jones BC, Smith H, Copely N. A flicker paradigm for inducing change blindness reveals alcohol and cannabis information processing biases in social users. Addiction 2003;98:235-44.
23. MacLeod C, Mathews A, Tata P. Attentional bias in emotional disorders. J Abnorm Psychol 1986;95:15-20.
24. Alvarez B, Dahlitz MJ, Vignau J, Parkes JD. The delayed sleep phase syndrome: clinical and investigative findings in 14 subjects. J Neurol Neurosurg Psychiatry 1992;55:665-70.
25. Morris M, Lack L, Dawson D. Sleep-onset insomniacs have delayed temperature rhythms. Sleep 1990;13:1-14.
26. Lack L, Wright H. The effect of evening bright light in delaying the circadian rhythms and lengthening the sleep of early morning awakening insomniacs. Sleep 1993;16:436-43.
27. Lubman, D.I., Peters, L.A., Mogg, K., Bradley, B.P. & Deakin, J.F.W. (2000). Attentional bias for drug cues in opiate dependence. Psychol Med;30:169-75.
28. Tyron WW. Issues of validity in actigraphic sleep assessment. Sleep 2004; 27: 158-65.
29. Wicklow A, Espie CA. Intrusive thoughts and their relationship to actigraphic measurement of sleep: towards a cognitive model of insomnia. Behav Res Ther 2000; 38; 679-93.
30. Mathews A, Ridgeway V, Williamson D. Evidence for attention to threatening stimuli in depression. Behav Res Ther 1996; 34: 695-705.
31. Leech G, Rayson P, Wilson A. Word Frequencies in Written and Spoken English. London: Longman; 2001.
32. Beck AT, Steer RA, Brown GK. Manual for the Beck Depression Inventory-Fast Screen. San Antonio: The Psychological Corporation; 2000.
33. Spielberger CD, Gorsuch RL, Lushene R, Vagg PR, Jacobs GA. Manual for the State-Trait Anxiety Inventory. Palo Alto, CA: Consulting Psychology Press, 1983.
34. Nelson, H.E. The National Adult Reading Test: 2nd Edition. Windsor: NFER-Nelson, 1991.
35. Tang, NKY. The Anxiety and Preoccupation about Sleep Questionnaire. In Distorted Perception of Sleep in Insomnia: Phenomenology, Mechanisms and Interventions. Unpublished doctoral dissertation, 2003.
36. Adan A, Almirall H. Horne & Ostberg morningness-eveningness questionnaire: a reduced scale. Pers Ind Diff 1991; 12: 241-53.
37. Hoddes E, Zarcone V, Smythe H, Phillips R, Dement WC. Quantification of sleepiness: a new approach. Psychophysiology 1973; 10: 431-36.
38. National Medical Advisory Committee. The Management of Anxiety and Insomnia. Edinburgh: HMSO, 1994.
39. Van Someren EJW, Swaab DF, Colenda CC, Cohen W, McCall, WV, Rosenquist PB. Bright light therapy: Improved sensitivity to its effects on rest-activity rhythms in Alzheimer patients by application of nonparametric methods. Chronobiol Int 1999; 16: 505-18.
40. Bradley BP, Mogg K, Lee S. Attentional biases for negative information in induced and naturally occuring dysphoria. Behav Res Ther 1997; 35: 911- 27.
41. Minto JM, Espie CA. An investigation of pre-sleep cognitive arousal in primary insomnia, delayed sleep phase syndrome and good sleepers. Sleep 2004; 27: A263-64.
42. Lack L, Bootzin RR. Circadian Rhythm Factors in Insomnia and their Treatment. In: Perlis M, Lichstein K, eds. Treatment of Sleep Disorders: Principles and Practice of Behavioral Sleep Medicine. New York: Wiley; 2003.
43. Tankova I, Adan A, Buela-Casal G. Circadian typology and individual differences: a review. Pers Individ Differ 1994;16:671-84.
44. Tang NKY, Harvey AG. Correcting distorted perception of sleep in insomnia: a novel behavioural experiment. Behav Res Ther 2004;42:27-39.
45. Eysenck MW. Anxiety and Cognition: a Unified Theory. Hove, East Sussex: Psychology Press; 1997.
46. Mogg K, Bradley BP, Hallowell N. Attentional bias to threat: roles of trait anxiety, stressful events, and awareness. Q J Exp Psychol A 1994;47A:841-64.
47. Mogg K, Bradley BP, Dixon C, Fisher S, Twelftree H, McWilliams A. Trait anxiety, defensiveness and selective processing of threat: an investigation using two measures of attentional bias. Pers Individ Differ 2000;28:1063-77.
48. Mogg, K, Bradley BP. Some methodological issues in assessing attentional biases for threatening faces in anxiety: a replication study using a modified version of the probe detection task. Behav Res Ther 1999;37:595-604.
49. Broomfield NM, Turpin G. Covert and overt attention in trait anxiety: a cognitive psychophysiological analysis. Biol Psychol 2005;68:179-200
50. Field M, Mogg K, Bradley BP. Eye movements to smoking-related cues: effects of nicotine deprivation. Psychopharmacol 2004;173:116-23.
51. Perlis ML, Giles DF, Mendelson WB, Bootzin RR, Wyatt JK. Psychophysiological insomnia: the behavioural model and a neurocognitive perspective. J Sleep Res 1997;6:179-88.
52. Grey S, Mathews A. Effects of training on interpretation of emotional ambiguity. Q J Exper Psychol 2000;53A:1143-62.

J. Sleep Res. (2007) **16,** 230–238

Prospective comparison of subjective arousal during the pre-sleep period in primary sleep-onset insomnia and normal sleepers

JENNIFER A. ROBERTSON, NIALL M. BROOMFIELD and COLIN A. ESPIE
University of Glasgow Sleep Research Laboratory, Glasgow, UK

Accepted in revised form 13 December 2006; received 3 August 2006

SUMMARY Psychophysiological insomnia (PI) is the most common insomnia subtype, representing 12–15% of all sleep centre referrals. Diagnostic guidelines describe PI as an intrinsic sleep disorder involving both hyperarousal and learned sleep-preventing associations. Whilst evidence for the first component is reasonably compelling, evidence for learned (conditioned) sleep effects is markedly lacking. Indeed, to date no study has attempted to capture directly the conditioned arousal effect assumed to characterize the disorder. Accordingly, the present study explored variations in subjective arousal over time in 15 PI participants (sleep onset type) and 15 normal sleepers (NS). Self-report measures of cognitive arousal, somatic arousal and sleepiness were taken at three time points: 3 h before bedtime (early to mid-evening); 1 h before bedtime (late evening); and in the bedroom at lights out (bedtime) across four, 24-h cycles. Fluctuations in mean arousal and sleepiness values, and in day-to-day variation were examined using analyses of variance. Participants with PI were significantly more cognitive aroused and significantly less sleepy relative to NS, within the bedroom environment. These results support the tenet of conditioned mental arousal to the bedroom, although competing explanations cannot be ruled out. Results are discussed with reference to extant insomnia models.

KEYWORDS cognitive, conditioned arousal, psychophysiological insomnia, sleepiness, somatic

INTRODUCTION

Insomnia, the most widely reported psychiatric symptom in the UK (Singleton *et al.*, 2001) and the main reason for benzodiazepine prescribing in primary care (NICE, 2004), disrupts sleep initiation and/or maintenance and promotes daytime fatigue, decreased mood and general malaise (American Psychiatric Association, 1994). Presenting in 30% of individuals occasionally, and 10–15% chronically (Foley *et al.*, 1995; Gallup Organization, 1991; Ohayon *et al.*, 1997), insomnia may be symptomatic of several disorders. When contributing medical, sleep and psychiatric disorders are ruled out, psychophysiological insomnia (PI) is usually diagnosed [International Classification of Sleep Disorders, second edition (ICSD-2); American Academy of Sleep Medicine, 2005]. PI is the most common insomnia subtype, representing 12–15% of all referrals seen at sleep centres. Despite its prevalence, underlying mechanisms for PI remain at best only partially understood (Espie, 2002).

There is at least general acceptance as to what comprises PI. For a diagnosis to be made, patients must exhibit (i) heightened arousal and (ii) learned sleep-preventing associations, in the context of difficulty initiating and/or maintaining sleep despite adequate opportunity, with associated daytime impairment. Heightened arousal may be physiological in nature, or reflect a more cognitive hypervigilance pattern. Learned sleep-preventing associations may be in response to either internal cognitions, or external stimuli. This notion, that PI is a disorder of hyperarousal and conditioned sleep difficulty, has stood the test of time, featuring in the original (ICSD: American Sleep Disorders Association, 1990) and subsequent classification systems [ICSD-R (revised): American Sleep Disorders

Correspondence: Colin A. Espie, University of Glasgow Sleep Research Laboratory, Sackler Institute of Psychobiological Research, Southern General Hospital, 1345 Govan Road, Glasgow G51 4TF, UK. Tel.: +44 141 232 7700; fax: +44 141 3574899; e-mail: c.espie@clinmed.gla.ac.uk

Association, 1997; ICSD-2), as well as in Research Diagnostic Criteria for Insomnia (RDC-I; Edinger et al, 2005).

People with PI may be more aroused autonomically (Bonnet and Arand, 1992, 1995; Freedman and Sattler, 1982; Monroe, 1967; Stepanski *et al*., 1989), or cortically (e.g. Freedman, 1987; Freedman and Sattler, 1982; Merica *et al*., 1998) than either good sleepers (e.g. Merica & Gaillard, 1992; Lamarche and Ogilvie, 1997) or patients with insomnia secondary to major depression (Perlis et al, 2001). There are also data suggesting elevated metabolic (Bonnet and Arand, 1995) and higher adreno-cortical function (Adam *et al*., 1986; Johns *et al*., 1971) in insomnia. Furthermore, data from thought sampling (e.g. Broman and Hetta, 1994; Lichstein and Rosenthal, 1980; Nicassio *et al*., 1985; Wicklow and Espie, 2000) and experimental cognitive manipulation studies (Ansfield *et al*., 1996; Gross and Borkovec, 1982; Hall *et al*., 1996), indicate that PI is associated with elevated arousal in the form of ruminative pre-sleep cognitive activity. Essentially, this is what patients describe as a 'racing mind'.

However, it remains unclear whether such cognitive and somatic hyper- arousal are learned responses. Outcome data for Stimulus Control therapy (SCT), arguably the most effective component of behaviour therapy for PI (Murtagh and Greenwood, 1995; Morin et al 1999), only indirectly support sleep-related conditioning in PI. Although SCT is based on the premise that conditioned cues associated with successful sleep should be separated from activities incompatible with sleeping (Bootzin, 1972), there is little evidence that good sleepers and insomnia patients actually differ in their sleep (in)compatible activities (Harvey, 2000; Haynes *et al*., 1974, 1982). Moreover, the treatment effectiveness of SCT cannot be taken as a reliable demonstration of a presumptive underlying process. There is also experimental clinical evidence that the temporal components of SCT [regularizing sleep routines] are as effective as the situational (conditioning) components [re-establishing bedroom cues for sleep] in reducing latency to sleep (Tokarz and Lawrence, 1974), and that a countercontrol directive (sit up in bed and read, watch TV etc. if unable to sleep) may also be effective (Zwart and Lisman, 1979). Such data present a challenge to the conditioning model.

Even at the experiential/phenomenological level, we are unaware of any study that captured directly self-reported arousal changes associated with sleep preparation in PI relative to normal sleepers (NS). This would seem to be a logical starting point to consider whether or not PI patients de-arouse in some respect differently from NS. In the present study, therefore, process data on subjective arousal and subjective sleepiness were gathered from PI and NS participants at three time points: three hours before bedtime (early/mid-evening after dinnertime); one hour before bedtime (late evening, quiet wakefulness); and in the bedroom at lights out (bedtime). These times were specifically chosen to track changes in arousal as bedtime approached, and when entering the bedroom environment.

Relative to the NS group, we hypothesized that PI participants would report (i) greater cognitive and somatic arousal (across time points), consistent with general hyper-arousal, and (ii) elevated arousal and (iii) reduced sleepiness specifically in the bedroom, consistent with the potential situational conditioning effects associated with the bedroom environment.

METHOD

Participants

Participants (≥18 years) were recruited via email (University and National Health Service), a local newspaper advertisement and a Sleep Clinic waiting list. Potential participants were screened for eligibility using UGSRL's comprehensive sleep interview schedule, and questionnaire measures of sleep (Pittsburgh Sleep Quality Index [PSQI] – Buysse *et al*., 1989; Epworth Sleepiness Scale [ESS] – Johns, 1991), sleep beliefs (Dysfunctional Beliefs and Attitudes about Sleep Scale -10 item version [DBAS-10; - Espie *et al*., 2000) and depression [BDI-II] - Beck *et al*., 1996).

For inclusion in the PI group, participants had to have PI of sleep-onset type that was chronic (>6 months in duration) (ICSD-2: American Academy of Sleep Medicine, 2005). In addition PI participants had to score >6 on the PSQI, a recognized cut-off for identifying significant sleep disturbance (Backhaus *et al*., 2002; Buysse *et al*., 1989), and <10 on the ESS. NS participants did not meet criteria for PI, and reported satisfaction with their sleep, and being typically good sleepers, including during the past month (c.f. RDC-I criteria for NS). NS also had to score <5 on the PSQI.

Similar to previous studies (e.g. Bonnet and Arand, 1997, 1998), exclusion criteria for both groups were (a) symptomatic evidence of another sleep disorder (e.g. sleep apnoea, restless legs syndrome or periodic limb movement disorder); (b) sleep disturbance attributable to a psychiatric/medical condition, (c) significant depressive symptoms (>23 on the BDI-II), (d) being on medication (for sleep or otherwise), or (e) receiving psychological treatment (for insomnia or otherwise). Following screening, four PI participants were excluded based on BDI-II scores, one due to drug usage, and one due to significant medical problems.

A final sample of 30 participants met inclusion criteria (15 PI, 15 NS), ranging in age from 18 to 53 years old, with an average of 29.2 years. Sixteen were female and 14 were male. All participants completed the study.

Measures

Arousal and sleepiness

The following measures were selected to capture the experience of cognitive and somatic arousal, and sleepiness.

- The Pre-Sleep Arousal Scale (PSAS; Nicassio *et al*., 1985) is a brief self-administered measure in which participants rate the intensity of experienced arousal. Internal consistency for somatic (8 items) and cognitive (8 items) subscales are satisfactory ($\alpha = 0.81$ and $\alpha = 0.76$) respectively.

- The Stanford Sleepiness Scale (SSS; Hoddes *et al*., 1973) is a seven point Likert-type measure. Respondents provide a rating to match the set of descriptors that best describes their feeling of sleepiness at a given moment. The reliability of the SSS has been reported as r = 0.88 (Hoddes *et al*., 1973).
- The Neutral Cognitive Activity Scale (NCAS: Tang and Harvey, 2004) is a 7-item self-report scale validated to measure the intensity of neutral cognitive activity, i.e. making no assumption about the content of individual pre-sleep cognitive activity. The original scoring scale of 1–10 was amended to a common 5-point scale, in order to be consistent with other measures. According to our own data, the NCAS was internally consistent in this revised format (Cronbach $\alpha = 0.921$).
- The Arousal Level as Present State (ALPS) rating scale was devised by the authors to yield point in time ratings of mental arousal/alertness, physical tension, and sleepiness, utilizing a common 5-point scale (1-"not at all" to 5-"extremely").

Sleep

Participants completed a daily sleep diary (Espie, 1991) each morning upon rising after the experimental night allowing calculation of: (i) sleep onset latency (SOL), (ii) total sleep time (TST), (iii) wake time after sleep onset (WASO), and (iv) sleep efficiency (SE) ([total time slept/total time in bed] × 100), Actigraphic data were also gathered as an objective estimate of sleep, using Actiwatch AW4 units ® (Cambridge Neurotechnology Ltd.). Actigraphy is a simple, non-intrusive measure which estimates sleep parameters based on body movement (Sadeh *et al*., 1995). Participants wore the Actiwatch continuously on their non-dominant hand except during wet activities. An event marker was depressed at lights out and upon rising. Epoch length was 1 minute with sleep and wakefulness determined by the programme algorithm; (i) SOL, (ii) TST, (iii) WASO, and (iv) SE data were computed as per sleep diary data.

Design and procedure

The study was designed as a mixed models ANOVA with two levels of the between subjects factor Group (PI, NS) and three levels of the within subjects factor Time [three hours before bedtime (T-3), one hour before bedtime (T-1), at bedtime (in the bedroom) (T)]. Participants completed all measures at home and were advised to follow their normal personal schedules and routines, across a 4 night/day experimental period. The duration of the study period (four 24-hour cycles) was deemed sufficiently long to capture any patterns in the data being collected, while remaining manageable for participants to adhere to. The experimental period occurred during weekdays (Monday-Thursday), as it was postulated that participants would be more likely to have a regular/consistent bedtime than at a weekend. Potential participants were asked at screening about their regular bedtimes, and were advised to adhere to these during the study. After the study period, questionnaire measures and actigraphs were returned, and a single global compliance rating was completed. This comprised the general question 'to what extent did you comply with the instructions (relating to sleep diary completion at specified times, arousal measure completion at specified times, and actiwatch use) given to you at the start of the study?' which was rated 1 – 'not at all', 2 – 'slightly', 3 – 'moderately', 4 – 'a lot' through 5 – 'fully'. Participants were debriefed, thanked, and provided with a copy of 'The Good Sleep Guide' (National Medical Advisory Committee, 1993).

Data handling

All data were examined for normality of distribution and logarithmic transformations (log to the base of ten) were applied to skewed data. A first set of ANOVAS used mean values for each time point across the four nights of the study, with Bonferroni corrections applied to follow-up analyses (alpha level set at 0.017). A secondary analysis was conducted using standard deviation values for these means to investigate variance (day-to-day variability) in relation to evening de-arousal. BDI-II scores were entered into two analyses (ANCOVA), because of observed linear correlations (mental alertness rating on the ALPS, SSS).

RESULTS

Participants

There were eight women and seven men in each experimental group and mean ages were similar [PI = 30.7 yr. (SD 10.7); NS = 27.7 yr. (SD 7.05)]. Descriptive clinical and sleep information is presented in Table 1.

Table 1 Descriptive clinical data for the Psychophysiological Insomnia ($n = 15$[a]) and Normal Sleeper ($n = 15$) groups

	Psychophysiological Insomnia [Mean (SD): median[b]]	*Normal Sleeper [Mean (SD): median[b]]*
PSQI	10.47 (2.2)	2.93 (1.28)
ESS	4.73 (2.94)	3.21 (1.72)
DBAS-10	4.24 (1.43)	3.21 (1.72)
BDI-II	7.27 (6.66)	2.13 (3.16)
SOL (diary)	61 (39.7): 53	11 (5.86): 10
WASO (diary)	37 (42.1): 23	4 (3.91): 3
TST (diary)	348 (79.4): 366	438 (49.5): 445
SE (diary)	78 (11.7): 80	97 (1.59): 97
SOL (actigraph)	30 (23.0): 21	12 (9.95): 9
WASO (actigraph)	56 (25.8): 50	59 (26.7): 54
TST (actigraph)	354 (61.8): 352	374 (56.8): 381
SE (actigraph)	80 (6.41): 82	84 (7.58): 85

PSQI (Pittsburgh Sleep Quality Index), ESS (Epworth Sleepiness Scale), DBAS-10 (Dysfunctional Beliefs and Attitudes about Sleep Scale – 10 item version), BDI-II (Beck Depression Inventory –II), SOL (Sleep-Onset Latency: min), WASO (Wake time After Sleep-Onset; min), TST (Total Sleep Time; min), SE (Sleep Efficiency, percent).
[a]$n = 14$ for actigraphy data.
[b]for sleep data.

Clinical characteristics

Neither group demonstrated clinically important daytime sleepiness and there was no significant between group difference on the ESS. There was a non-significant trend for PI subjects to score higher than NS on the DBAS-10 ($P = 0.086$). PI participants had a higher PSQI index (10.5) than NS (2.9) ($t = 11.47$, $P = 0.0001$) and more depressive symptoms on the BDI-II ($t = 2.70$, $P = 0.012$). Nevertheless, both group means were within the 0–9 BDI-II range, indicating minimal depression.

Sleep comparisons

Subjective report

Mann-Whitney comparisons indicated that PI and NS participants differed on subjective SOL, WASO, TST, and SE (all $P < 0.001$), with PI as expected reporting poorer sleep.

Actigraphy

One participant's actigraphic data was lost due to faulty equipment, therefore, these analyses were based on 14 participants in the PI group. Again PI and NS differed significantly in the expected direction on the primary sleep variable of interest, SOL ($P = 0.008$), with a near significant effect also on WASO ($P = 0.063$). There were no differences on the other actigraphic sleep measures.

Arousal comparisons

Results for each of the four sets of dependent measures (PSAS, SSS, NCAS, ALPS) are presented with associated graphical illustrations (Figs 1–4).

Pre-sleep arousal scale

Analysis revealed a main effect of Time ($F_{(2,56)} = 53.77$, $P < 0.0001$), a main effect of Group ($F_{(1,28)} = 16.43$, $P < 0.0001$), and an interaction effect of Group X Time ($F_{(2,56)} = 10.92$, $P < 0.0001$) for the PSAS *cognitive sub-scale* (see Fig. 1a). Significant differences were found between the groups three hours before bedtime, at T-3 ($t(28) = 2.63$, $P = 0.014$), one hour before bedtime, at T-1 ($t(28) = 3.84$, $P = 0.001$), and at bedtime, T ($t(28) = 5.08$, $P < 0.0001$). On this measure, therefore, PI participants were significantly more cognitively aroused than the NS group during the evening and in the bedroom. Comparable results were obtained for a secondary analysis using individual participant standard deviation values to represent the variance around the mean. Main effects were significant for Time ($F_{(2,56)} = 66.98$, $P < 0.0001$), Group ($F_{(1,28)} = 16.43$, $P < 0.0001$) and Group X Time ($F_{(2,56)} = 13.80$, $P = 0.004$) and significant differences were observed between groups at all three time points. Inspection of Fig. 1a shows that cognitive arousal decreased in both groups over time, but that PI de-aroused less than NS group, and the rate of change during the period T-1 to T was greater in NS than in PI ($P = 0.023$). Figs 1–4 also display error bars, and it can be seen in Fig. 1a that the group variance around the group mean also reduced over time in NS relative to PI.

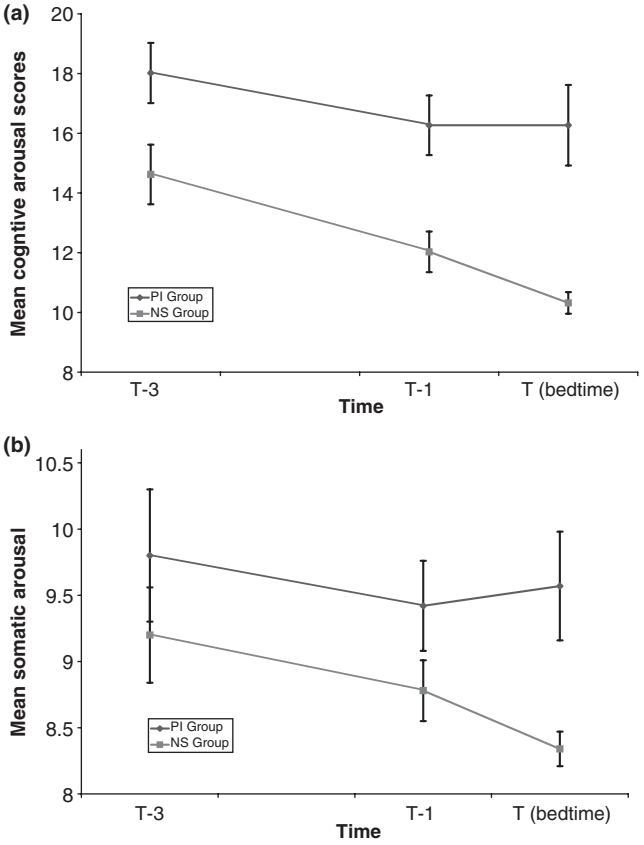


Figure 1. (a) Mean arousal scores of the PI and NS groups at each time point as measured by the cognitive subscale of the PSAS. (b) Mean arousal scores of the PI and NS groups at each time point as measured by the somatic subscale of the PSAS.

Analysis of the PSAS *somatic sub-scale* revealed a main effect of Time ($F_{(2,56)} = 5.66$, $P = 0.006$) with non-significant trends for Group ($F_{(1,28)} = 3.65$, $P = 0.066$) and for the Group X Time interaction ($F_{(2,56)} = 2.59$, $P = 0.084$). Likewise, analyses using standard deviation scores to represent individual variability yielded a significant Time main effect only ($F_{(2,56)} = 7.37$, $P = 0.011$). Mean PSAS somatic subscale scores can also be observed in Fig. 1b. There was a greater reduction in somatic arousal from 1 hour before bedtime to bedtime (T-1 to T) in NS relative to PI ($P = 0.014$).

Stanford sleepiness scale

Results for the SSS comparisons are presented in Fig. 2. ANCOVA (with BDI-II as a covariate) revealed a main effect of Time ($F_{(2,54)} = 78.78$, $P < 0.0001$), and a Group X Time interaction ($F_{(2,54)} = 11.17$, $P < 0.0001$) but no main effect of Group ($F_{(1,27)} = 0.90$, $P = 0.351$). As can be seen in Fig. 2,

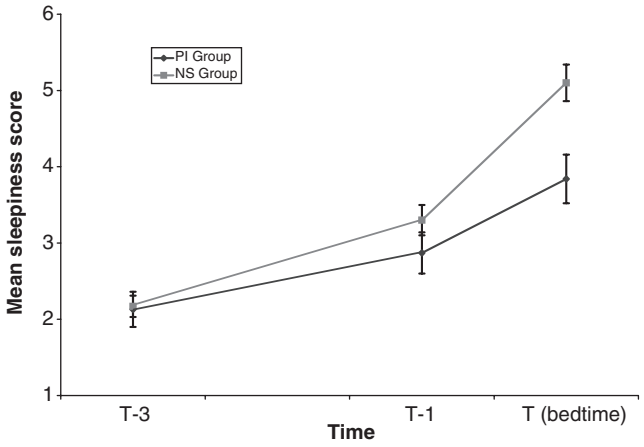


Figure 2. Mean sleepiness of rating of the PI and NS groups at each time point as measured by the SSS.

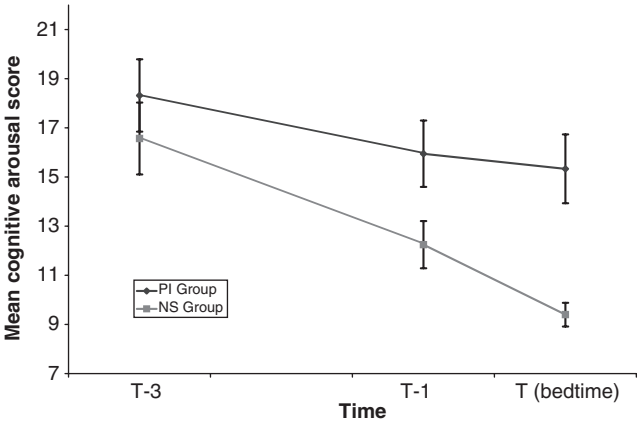


Figure 3. Mean arousal scores of the PI and NS groups at each time point as measured by the Neutral Cognitive Activity Scale.

there were no significant differences between groups three hours (t (23) = 0.12, $P = 0.903$) or one hour before bedtime (t (28) = 1.31, $P = 0.201$). However, at bedtime the PI group was significantly less sleepy than the NS group (t (28) = 3.14 $P = 0.004$). These results suggest that the progressive incrementing of sleepiness displayed by the NS participants was somehow curtailed in the PI group once they were in the bedroom, although increases in sleepiness from T-3 to T-1 ($P = 0.027$) and from T-1 to T ($P = 0.003$) were both significantly greater in NS compared with PI.

Neutral cognitive activity scale

On this measure, ANOVA revealed a main effect of Time ($F_{(2,56)} = 60.23$, $P < 0.0001$), a main effect of Group ($F_{(1,28)} = 7.29$, $P = 0.012$), and an interaction effect of Group X Time ($F_{(2,56)} = 13.32$, $P < 0.0001$) for the mean data. No significant differences were found between the groups three hours (t (28) = 0.95, $P = 0.348$) before or one hour before bedtime (t (28) = 2.44, $P = 0.021$) [when strict Bonferroni adjustments were applied]. However, at bedtime (t (22) = 4.57, $P < 0.0001$) PI participants reported significantly greater neutral cognitive activity; see Fig. 3. Analysis of night-to-night variability scores as the dependent variable also yielded main effects for Time ($F_{(2,56)} = 74.76$, $P < 0.0001$), Group ($F_{(1,28)} = 8.40$, $P = 0.014$), and an interaction effect for Group X Time ($F_{(2,56)} = 16.62$, $P < 0.0001$). The graphed pattern of results for the NCAS appears similar to the PSAS cognitive sub-scale data (c.f. Fig. 1a). Again the standard error of the mean in the NS group is much lower than that for the PI group at bedtime, and there is a significantly greater reduction in cognitive activity in the NS group from T-1 to T ($P = 0.011$).

Arousal level as present state

For the ALPS ratings of *mental alertness*, ANCOVA (with BDI-II as a covariate) was applied. Main effects of Time ($F_{(2,54)} = 27.75$, $P < 0.0001$), Group ($F_{(1,27)} = 8.53$, $P = 0.007$), and Group X Time ($F_{(2,54)} = 12.07$, $P < 0.0001$) were obtained. PI participants were significantly more alert than NS during late evening (t (22) = 2.88, $P = 0.009$) and in the bedroom (t (28) = 4.44, $P < 0.0001$). However, no between group difference was found in mental alertness three hours before bedtime (t (28) = 1.24, $P = 0.23$) (Fig. 4). Consistent with this was the finding that reduction in ALPS mental alertness was greater from T-1 to T in the NS than in the PI group ($P = 0.001$). In terms of *physical tension*, ANOVA revealed a main effect of Time ($F_{(2,56)} = 19.9$, $P < 0.0001$) but no main effect of Group ($F_{(1,28)} = 1.22$, $P = 0.278$), or Group X Time ($F_{(2,56)} = 0.88$, $P = 0.419$). There were no significant reductions in physical tension in the NS group relative to the PI group during either the T3 to T1, or the T1 to T periods. The ALPS *sleepiness* rating revealed a main effect of Time ($F_{(2,56)} = 114.40$, $P < 0.0001$), and an interaction effect of Time X Group ($F_{(2,56)} = 8.18$, $P = 0.001$) but no Group main effect ($F_{(1,28)} = 2.10$, $P = 0.159$). No significant difference was found between groups three hours (t (23) = 0.35, $P = 0.733$) or one hour before bedtime (t (22) = 0.82, $P = 0.424$). However, at bedtime, NS had higher sleepiness ratings than PI (t (24) = 3.34, $P = 0.003$) and the increase in sleepiness from T1 to T was also significantly greater in normal sleepers ($P = 0.010$). These results closely parallel our SSS data, with PI participants appearing to be less sleepy in the bedroom than NS.

Compliance with experimental instructions

Participants in both groups reported high levels of compliance. The mean global compliance rating scale score was 4.53 (SD = 0.51) out of a possible 5, with no significant differences between PI and NS. Importantly, these data indicate that participants reported completing the arousal measures (our dependent variables) at the stated times as directed by the research protocol. The scale provided space for participants to report any particular areas of non-compliance. Few completed this section at all, presumably due to high reported compliance in both samples. Amongst those who did give free text

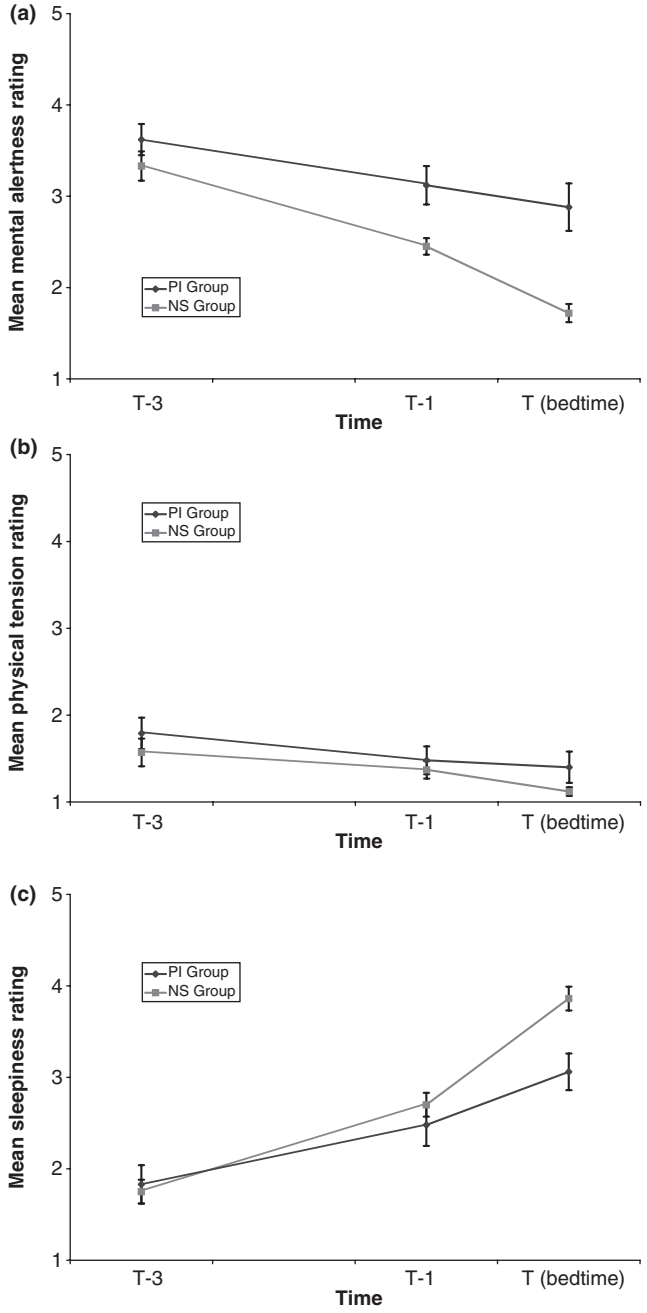


Figure 4. (a) Mean rating of the PI and NS groups at each time point as measured by the ALPS mental alertness scale. (b) Mean rating of the PI and NS groups at each time point as measured by the ALPS physical tension scale. (c). Mean rating of the PI and NS groups at each time point as measured by the ALPS sleepiness scale.

comments, several reported that they had forgotten to press the actigraph event marker button on one or more occasion.

DISCUSSION

As far as we are aware, this is the first study to examine systematically the process of self-reported de-arousal, during the pre-sleep period, in people with insomnia relative to normal sleepers. In particular, we compared people with PI (sleep-onset type) with normal good sleepers, across three evening time points, to test the conventional wisdom that arousal differences between groups would be, at least in part, situation dependent, consistent with a conditioned arousal model of insomnia.

Our results, first of all can be interpreted in terms of general hyperarousal theory regarding insomnia (e.g. Bonnet and Arand, 1995). For example, on the cognitive subscale of the PSAS, the PI group had elevated scores at all three time points. This higher level of mental activation, present at least 3 hours prior to bedtime and in the living areas of the house rather than in the bedroom, is suggestive of cognitive hyperarousal in PI relative to NS. Concurrent validation that this finding was robust at one hour pre-bedtime (but not at three hours) was obtained using a novel 'present state' rating of mental alertness.

Interestingly, our parallel subjective measures of somatic arousal (PSAS somatic sub-scale, ALPS physical tension rating) demonstrated only a Time main effect. That is, the groups were similar at each observation and reductions were observed over time in both NS and PI, but not at differential rates. Objectively gathered data have shown arousal differences between insomnia and good sleepers, on numerous physiological dimensions (see Introduction, this paper), so it may be that using self-report alone is insensitive to this arousal domain. Alternatively, there may be validity problems with either the PSAS or the ALPS, or both, as measures of self-reported somatic arousal. Consequently we calculated correlation co-efficients to estimate concurrent validity. These demonstrated satisfactory validity 3 hours before bedtime ($r = 0.788$), 1 hour before bedtime ($r = 0.759$) and at bedtime ($r = 0.795$). In practice, clinicians work primarily with the phenomenology of insomnia, and our data (showing that mental measures at least are sensitive) are consistent with the replicated finding that people with PI generally complain more about cognitive than somatic symptomatology (see reviews by Espie, 2002; Harvey, 2002).

However, we found stronger evidence consistent with arousal conditioning to bed-time itself and/or to the bedroom environment. Four out of seven analyses showed significant effects in support of less de-arousal at bedtime in the PI group. Furthermore, analyses of change scores consistently found that there were more sizeable reductions in cognitive activation, and greater increases in sleepiness, in normal sleepers in the period from 1 hour prior to bedtime and bedtime itself than there were in people with insomnia. There is also evidence from inspection of standard error bars of considerable homogeneity in such de-arousal responses in normal sleepers.

Although such results appear to be broadly supportive of the conditioning model of PI, we cannot discriminate from our data between bed-time and the bedroom environment. Our third and final time sample was taken, at bedtime within the bedroom. A future study may better discriminate these factors by taking an additional time sample at bedtime, but prior to participants entering the bedroom environment. Nevertheless, relative to NS, people with PI were significantly more aroused

on measures of neutral cognitive activity once in the bedroom, and they were significantly less sleepy, in the context of there being no group differences at the earlier time points. This may suggest that there is a link between going to bed and the individual's 'arousal gradient'. However, PI participants were not more alert once in the bedroom compared with NS. Rather, both groups appeared to de-arouse in the lead up to bedtime, but PI de-aroused to a markedly lesser extent. In terms of our subjective sleepiness data, a similar pattern (although in reverse) was observed. That is, both groups showed increased sleepiness over time, but the PI group less so. This is consistent with the idea that failure to de-arouse may be typical of insomnia (Espie, 2002; psychobiological inhibition model). Compared with the NS experience, it seems possible then that the process of retiring to bed failed to act as a discriminative stimulus for further de-arousal (and ensuing sleep) in the PI group (c.f. Bootzin, 1972; stimulus control model).

Further research is required with detailed definition of arousal changes over time. We only had three data points; insufficient to clarify, for example, whether linear changes in de-arousal occur over time. Liberal interpretation of the graphs might suggest that the rate of de-arousal between T-1 and T was not merely a linear extension of the de-arousal between T-2 and T-1, as might be expected if only Process S and C were operating. Rather, the apparent increase in de-arousal (and sleepiness) at bedtime may suggest that an additional mechanism such as conditioned de-arousal is indeed operating. Another advantage of using multiple time points to obtain a 'fine grain' picture would be the opportunity to examine highly specific situational effects that might be crucial to the stimulus control model (e.g. entering the bedroom, getting into bed, putting the light out, watching television, talking in bed etc.).

Of course, competing explanations unrelated to conditioning should also be considered. The finding regarding elevated neutral cognitive activity may reflect a cognitive arousal anticipation effect, or heightened sleep effort (Broomfield *et al*., 2005; Espie et al, 2006; attention-intention-effort pathway). It is possible that PI participants became preoccupied with sleep once they have decided when to go to bed, resulting in cognitive arousal. Indeed, PI patients reflect on their sleep (problem) at any time of the day or night (Harvey, 2002; cognitive model). It could be that such anticipation and monitoring simply becomes more focused proximal to bedtime. The observed group differences on sleepiness level in the bedroom could be influenced also by circadian timing. Whereas we attempted to screen out participants with explicit circadian phase delay, it is possible that circadian physiology may play some role in insomnia, just as insomnia factors may be problematic to separate from the clinical presentation of circadian rhythm sleep disorders (Lack and Bootzin, 2003).

Clearly, our findings should be taken as preliminary. Further work is required using similar prospective methodology, with concurrent psychophysiological measurement (heart rate, skin conductance) alongside self-report data. Cortical arousal (as measured by qEEG methods) may be particularly fruitful to explore because this has been posited as the primary feature of conditioned arousal in PI (Perlis *et al*., 1997: neurocognitive model) and because cortical activity might be reasonably regarded as the neurobiological substrate of cognitive activation. We also relied heavily upon self-reported sleep to define our samples and future studies might include polysomnographic screening to objectively exclude possible occult sleep disorders other than insomnia. Interview and self-report screening measures cannot reliably achieve this, and although we used actigraphy, which partially confirmed subjective sleep problems in our sample, the role of actigraphy in insomnia assessment continues to be a matter of some debate (Ancoli-Israel *et al*., 2003).

We attempted to measure compliance, but this was retrospective and likely to be subject to experimental demand characteristics. We suggest, therefore, that 'palm pilot' technology might be useful to time lock the self-report data to simultaneous assays of somatic data. Our sample, though carefully screened, was small in number. Replication of the effects we have demonstrated is also required in treatment-seeking individuals with PI. Finally, there are pros and cons to exploring arousal phenomena in natural settings (the bedroom at home) versus experimental settings (in lab). Home-based study maximizes ecological validity; whereas lab studies improve reliability of measurement. There is relatively little evidence to support the view (stated in diagnostic criteria) that PI patients sleep better away from home. Consequently, we suggest that methodological refinements of the type suggested above should be undertaken first of all under rigorous laboratory conditions, where direct measures of somatic arousal can also be achieved, and then generalized to the home environment.

In conclusion, arousal and sleepiness data from the present study are consistent with hyperarousal and conditioned arousal in insomnia. These are not mutually exclusive perspectives. It appears that difficulties with the normal process of bed-time de-arousal may characterize the experience of PI.

ACKNOWLEDGEMENTS

Supported in part by research funding from Chief Scientist Office (CZG/2/187) and by the Dr. Mortimer and Theresa Sackler Foundation.

REFERENCES

Adam, K., Tomeny, M. and Oswald, I. Physiological and psychological differences between good and poor sleepers. *Journal of Psychiatric Research*, 1986, 20: 301–16.

American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders*, 4th Ed. Washington, DC, 1994.

American Sleep Disorders Association. *The International Classification of Sleep Disorders, Revised: Diagnostic and Coding Manual*. American Sleep Disorders Association, Rochester, Minnesota, 1990.

American Sleep Disorders Association. *The International Classification of Sleep Disorders, Revised: Diagnostic and Coding Manual-Revised*. American Sleep Disorders Association, Rochester, Minnesota, 1997.

American Academy of Sleep Medicine. *International Classification of Sleep Disorders, second edition (ICSD-II): Diagnostic and Coding Manual*. American Sleep Disorders Association, Rochester, Minnesota, 2005.

Ancoli-Israel, S., Cole, R., Alessi, C., Chambers, M., Moorcroft, W. and Pollak, C. P. The role of actigraphy in the study of sleep and circadian rhythms. *Sleep*, 2003, 26: 342–392.

Ansfield, M. E., Wegner, D. M. and Bowser, R. Ironic effects of sleep urgency. *Behav. Res. and Ther*., 1996, 34: 523–531.

Backhaus, J., Junghanns, K., Broocks, A., Reimann, D. and Hohagen, F. Test-retest reliability and validity of the Pittsburgh Sleep Quality Index in primary insomnia. *J. Psychosom. Res*., 2002, 53: 737–740.

Beck, A. T., Steer, R. A. and Brown, G. K. *Beck Depression Inventory*. The Psychological Corporation, New York, 1996.

Bonnet, M. H. and Arand, D. L. Caffeine use as a model of acute and chronic insomnia. *Sleep*, 1992, 15: 526–536.

Bonnet, M. H. and Arand, D. L. 24 hour metabolic rate in insomniacs and matched normal sleepers. *Sleep*, 1995, 18: 581–588.

Bonnet, M. H. and Arand, D. L. Physiological activation in patients with sleep state misperception. *Psychosom Med*, 1997, 59: 533–540.

Bonnet, M. H. and Arand, D. L. Heart rate variability in insomniacs and matched normal sleepers. *Psychosom Med*, 1998, 60: 610–615.

Bootzin, R. R. Stimulus control treatment for insomnia. *Proceedings of the American Psychological Association*, 1972, 7: 395–396.

Broman, J. E. and Hetta, J. Perceived pre-sleep arousal in patients with persistent psychophysiologic and psychiatric insomnia. *Nordic J Psy*, 1994, 48: 203–207.

Broomfield, N. M., Gumley, A. I. and Espie, C. A. Candidate cognitive processes in psychophysiologic insomnia. *J. Cogn. Psychother*., 2005, 19: 5–17.

Buysse, D. J., Reynolds, C. F., Monk, T. H., Berman, S. R. and Kupfer, D. J. The Pittsburgh Sleep Quality Index: A new instrument for psychiatric practice and research. *Psychiatry Res*, 1989, 28: 193–213.

Edinger, J. D., Bonnet, M. H., Bootzin, R. R., Doghramji, K., Dorsey, C. M., Espie, C. A., Jamieson, A. O., McCall, W. V., Morin, C. M. and Stepanski, E. J. Derivation of research diagnostic criteria for insomnia: report of an American Academy of Sleep Medicine Work Group. *Sleep*, 2004, 27: 1567–1596.

Espie, C. A. *The Psychological treatment of insomnia*. Chichester, John Wiley, 1991.

Espie, C. A. Insomnia: Conceptual Issues in the Development, Persistence, and Treatment of Sleep Disorder in Adults. *Annual Review of Psychology*, 2002, 53: 215–243.

Espie, C. A., Inglis, S. J., Harvey, L. and Tessier, S. Insomniacs' attribution: psychometric properties of the Dysfunctional Beliefs and Attitudes about Sleep Scale and the Sleep Disturbance Questionnaire. *J. Psychosom. Res*., 2000, 48: 141–148.

Espie, C. A., Broomfield, N. M., MacMahon, K. M. A., Macphee, L. M. and Taylor, L. M. The attention-intention-effort pathway in the development of psychophysiologic insomnia: A theoretical review. *Sleep. Med. Rev*., 2006, 10: 215–245.

Foley, D. J., Monjan, A. A., Brown, S. L., Simonsick, E. M., Wallace, R. B. and Blazer, D. G. Sleep complaints among elderly persons: an epidemiologic study of three communities. *Sleep*, 1995, 18: 425–433.

Freedman, R. Chronic insomniacs: replication of Monroe's findings. *Psychophys*, 1987, 24: 721–722.

Freedman, R. R. and Sattler, H. L. Physiological and psychological factors in sleep-onset insomnia. *J. Abnorm. Psychol*., 1982, 91: 380–389.

Gallup Organization. *Sleep in America*. Princeton, NJ, 1991.

Gross, R. T. and Borkovec, T. D. Effects of a cognitive intrusion manipulation on the sleep-onset latency of good sleepers. *Behav. Ther*., 1982, 13: 112–116.

Hall, M., Buysse, D. J., Reynolds, C. F., Kupfer, D. J. and Baum, A. Stress related intrusive thoughts disrupt sleep-onset and continuity. *Sleep Res*., 1996, 25: 163.

Harvey, A. G. Sleep hygiene and sleep-onset insomnia. *J Ner Men Dis*, 2000, 188: 53–55.

Harvey, A. G. A cognitive model of insomnia. *Behav. Res. Ther*., 2002, 40: 869–893.

Haynes, S. N., Follingstad, D. R. and McGowan, W. T. Insomnia: sleep patterns and anxiety levels. *J Psychosom Res*, 1974, 18: 69–74.

Haynes, S. N., Adams, A. E., West, S., Kamens, L. and Safranek, R. The stimulus control paradigm in sleep-onset insomnia: a multimethod assessment. *J Psychosom Res*, 1982, 26: 333–339.

Hoddes, E., Zarcone, V., Smythe, H., Phillips, R. and Dement, W. C. Quantification of sleepiness: A new approach. *Psychophysiology*, 1973, 10: 431–436.

Johns, M. W. A new method for measuring daytime sleepiness: The Epworth sleepiness scale. *Sleep*, 1991, 14: 540–545.

Johns, M. W., Gay, M. P., Masterton, J. P. and Bruce, D. W. Relationship between sleep habits, adrenocortical activity and personality. *Psychosom Med*, 1971, 3: 499–508.

Lack, L. and Bootzin, R. R. Circadian Rhythm Factors in Insomnia and their Treatment. In: M. PerlisK. Lichstein (Eds) *Treatment of Sleep Disorders: Principles and Practice of Behavioral Sleep Medicine*. Wiley, New York, 2003.

Lamarche, C. H. and Ogilvie, R. D. Electrophysiological changes during the sleep onset period of psychophysiological insomniacs, psychiatric insomniacs, and normal sleepers. *Sleep*, 1997, 20: 724–733.

Lichstein, K. L. and Rosenthal, T. L. Insomniacs' perceptions of cognitive versus somatic determinants of sleep disturbance. *J. Abnorm. Psychol*., 1980, 89: 105–107.

Merica, H. and Gaillard, J. M. The EEG of the sleep onset period in insomnia: a discriminant analysis. *Physiol Behav*, 1992, 52: 199–204.

Merica, H., Blois, R. and Gaillard, J. M. Spectral characteristics of sleep EEG in chronic insomnia. *Euro J Neurosci*, 1998, 10: 1826–1834.

Morin, C. M., Hauri, P. J., Espie, C. A., Spielman, A. J., Buysse, D. J. and Bootzin, R. R. Nonpharmacologic treatment of chronic insomnia: An American Academy of Sleep Medicine Review. *Sleep*, 1999, 22: 1134–1156.

Monroe, L. J. Psychological and Physiological differences between good and poor sleepers. *J. Abnorm. Psychol*., 1967, 72: 255–264.

Murtagh, D. R. and Greenwood, K. M. Identifying Effective Psychological Treatments for Insomnia: A Meta-Analysis. *J. Consult. Clin. Psychol*., 1995, 63: 79–89.

National Institute for Clinical Excellence. *NICE issues guidance on the use of drugs for the management of insomnia*. http://www.nice.org.uk, 2004 (April): 2004/021.

National Medical Advisory Committee. The Good Sleep Guide. In: *Management of anxiety and insomnia*. HMSO, London, 1993.

Nicassio, P. M., Mendlowitz, D. R., Fussell, J. J. and Petras, L. The phenomenology of the pre-sleep state: the development of the Pre-Sleep Arousal Scale. *Behav. Res. Ther*., 1985, 23: 263–271.

Ohayon, M. M., Caulet, M. and Guilleminault, C. How a general population perceives its sleep and how this relates to the complaint of insomnia. *Sleep*, 1997, 20: 715–723.

Perlis, M. L., Smith, M. T., Andrews, P. J., Orff, H. and Giles, D. E. Beta/Gamma EEG activity in patients with primary and secondary insomnia and good sleeper controls. *Sleep*, 2001, 24: 110–117.

Perlis, M. L., Giles, D. E., Mendelson, W. B., Bootzin, R. R. and Wyatt, J. K. Psychophysiological insomnia: the behavioural model and a neurocognitive perspective. *J. Sleep. Res*., 1997, 6: 179–188.

Sadeh, A., Hauri, P. J., Kripke, D. F. and Lavie, P. The role of actigraphy in the evaluation of sleep disorders. *Sleep*, 1995, 18: 288–302.

Singleton, N., Bumpstead, R., O'Brien, M., Lee, A. and Meltzer, H. *Psychiatric morbidity among adults living in private households, 2000*. Office for National Statistics, UK, 2001.

Stepanski, E. J., Glinn, M., Fortier, J., Sicklesteel, J. and Zorick, F. J. Physiological reactivity in chronic insomnia. *Sleep Res*, 1989, 18: 306.

Tang, N. K. Y. and Harvey, A. G. Effects of cognitive arousal and physiological arousal on sleep perception. *Sleep*, 2004, 27: 69–78.

Tokarz, T. and Lawrence, P. *An analysis of temporal and stimulus control factors in the treatment of insomnia*. Paper presented at the Association for the Advancement of Behaviour Therapy, Chicago, 1974.

Wicklow, A. and Espie, C. A. Intrusive thoughts and their relationship to actigraphic measurement of sleep: towards a cognitive model of insomnia. *Behav. Res. Ther.*, 2000, 38: 679–693.

Zwart, C. A. and Lisman, S. A. Analysis of Stimulus Control Treatment of Sleep-Onset Insomnia. *J. Consult. Clin. Psychol.*, 1979, 47: 113–118.

Behaviour Research and Therapy 47 (2009) 231–236

Contents lists available at ScienceDirect

Behaviour Research and Therapy

journal homepage: www.elsevier.com/locate/brat

The clock as a focus of selective attention in those with primary insomnia: An experimental study using a modified Posner paradigm

H. Woods [a,b,*], L.M. Marchetti [a,b], S.M. Biello [a], C.A. Espie [b]

[a] Department of Psychology, University of Glasgow, Glasgow, UK
[b] Glasgow Sleep Centre, Faculty of Medicine, University of Glasgow, Glasgow, UK

ARTICLE INFO

Article history:
Received 8 August 2008
Received in revised form
4 November 2008
Accepted 16 December 2008

Keywords:
Attention bias
Insomnia
Cognitive arousal
Monitoring
Posner paradigm

ABSTRACT

Espie and colleagues [(2006). The attention–intention–effort pathway in the development of psychophysiological insomnia: a theoretical review. Sleep Medicine Reviews, 10, 215–245] propose a route into psychophysiological insomnia along the attention–intention–effort pathway which focuses on the inhibition of sleep-wake automaticity. A contributing factor to this is selective attention to sleep (alongside explicit intention to sleep and effort in the sleep engagement process). Following on from previous work on selective attention to sleep [Marchetti, L. M., Biello, S. M., Broomfield, N. M., MacMahon, K. M. A., & Espie, C. A. (2006). Who is pre-occupied with sleep?. A comparison of attention bias in people with psychphysiological insomnia, delayed sleep phase syndrome and good sleepers using the induced change blindness paradigm. *Journal of Sleep Research, 15*, 212–221; MacMahon, K., Broomfield, N., Macphee, L., & Espie, C. A. (2006). Attention bias for sleep related stimuli in primary insomnia and delayed sleep phase syndrome using the dot-probe task. *Sleep, 29*, 11] and considering the importance of monitoring both internal and external cues in the maintenance of insomnia, as highlighted in the cognitive model of insomnia [Harvey, A. G. (2002). A cognitive model of insomnia. *Behaviour Research and Therapy, 40*, 869–893], a cognitive probe task was employed to investigate further the role of the clock as a focus of selective attention in those with primary insomnia.

A 2×2 between participants design comparing reaction time of individuals with primary insomnia ($n = 22$) and normal sleepers ($n = 22$) on a modified Posner paradigm. Responses obtained from a computer task presenting times which fall within a normal sleep period were analysed.

Individuals with primary insomnia demonstrated delayed disengagement to the clock ($F(1,84) = 6.9$, $p < 0.05$) which is taken as further support for previous research demonstrating that individuals with primary insomnia exhibit an attentional bias to sleep related stimuli.

These results lend support to the attention–intention–effort model (Espie et al., 2006) and the cognitive model (Harvey, 2002) both of which recognise the importance of selective attention towards salient stimuli in the maintenance of insomnia. Possible clinical implications of attentional bias to sleep as a marker of psychopathology progression and treatment efficacy are discussed.

Introduction

Insomnia

Primary insomnia (PI) is reportedly found in 3% of the population in western industrialised countries (Ohayon, 1996, 2002). According to diagnostic criteria, heightened arousal and learned sleep preventing associations form the foundations of this disorder, with patients exhibiting excessive focus upon and anxiety about sleep (American Sleep Disorder Association, 1997 & 2005, DSM-IV; American Psychiatric Association, 1994). Numerous authors contend that PI is the result of a number of psychological factors, such as maladaptive beliefs about sleep or excessive pre-sleep intrusive thoughts (Harvey, 2002; Espie, 2002; Morin, 1993).

Espie, Broomfield, MacMahon, Macphee, & Taylor (2006) propose a route into PI along the attention–intention–effort pathway which focuses on the inhibition of sleep–wake automaticity. In their conceptual paper, the authors propose that inhibition of sleep–wake automaticity can be attributed to three processes; selectively attending to sleep, explicitly intending to sleep and introducing effort into the sleep engagement process. This model has been developed by drawing on parallels in the anxiety disorder,

* Corresponding author. Department of Psychology, University of Glasgow, Glasgow, Scotland, UK. Tel.: +44 141 330 4757.
E-mail address: heatherw@psy.gla.ac.uk (H. Woods).

0005-7967/$ – see front matter © 2008 Elsevier Ltd. All rights reserved.
doi:10.1016/j.brat.2008.12.009

alcohol and drug abuse literature as well as recent clinical and experimental studies on insomnia. The cognitive model of insomnia (Harvey, 2002) likewise highlights the importance of monitoring both internal and external cues in the maintenance of insomnia.

Selective attention to sleep

An attentional bias is said to have developed when the attention system becomes sensitive or selective to a particular theme and impacts on the individual's cognitions. Espie et al. (2006) suggest that the attentional systems of individuals with PI are particularly sensitive to sleep. Several studies have now been carried out establishing a selective attention to sleep in PI using the ICB flicker paradigm and sleep related objects (Marchetti, Biello, Broomfield, MacMahon, & Espie, 2006), using the dot probe task and sleep related words (MacMahon, Broomfield, Macphee, & Espie, 2006) and in a cancer population who have developed sleep onset difficulties (Taylor, Espie, & White, 2003).

Clock monitoring

There is widespread evidence that individuals with insomnia attribute their difficulty with sleep to excessive pre-sleep worry with PI being 10 times more likely to attribute their sleep disturbance to cognitive arousal compared to somatic arousal (Lichstein & Rosenthal, 1980). The items implicating cognition as the major cause of the sleep disturbance (e.g. 'My mind keeps turning things over') on the Sleep Disturbance Questionnaire are the most highly rated (Espie, Brooks, & Lindsay, 1989; Harvey, 2001). Therefore, we find ourselves at the juncture of having established that pre-sleep worry contributes to PI but still unable to identify a trigger for such worry.

From clinical practice it is proposed that PI selectively attend to and monitor for sleep related cues such as body sensations which are consistent or inconsistent with falling asleep and the environment for signs of not falling asleep (Harvey, 2002). One of the sleep related cues associated with the stress of not falling asleep is the bedside clock. Subjective reports highlight two types of clock monitoring in PI; how long the individual has been in bed without sleep and how many hours are left before they have to start their day (Bearpark, 1994; Harvey, 2002). Following from this, Tang, Schmidt, & Harvey (2007) investigated the association between clock monitoring, pre-sleep worry and sleep. Thirty-eight PI were instructed to either monitor a clock or a digital display unit as they were trying to get to sleep. The clock monitoring task was rated as more worry provoking and interfered with sleep more than the digital display monitoring task. The clock monitoring PI reported more pre-sleep worry and longer SOL on the experimental night compared to baseline. Hence, in the clock displaying sleep times, we have an ecologically valid stimulus which is relevant in both the perspective of the patient and clinical research but also within everyday life. Most people would be able to recall at least one occasion where sleep eluded them while being aware the time to rise was approaching.

Cognitive probe task

The aim of this study was to investigate further the role of the clock in the development and maintenance of PI, by bringing together the work discussed above with selective attention in PI and the real life experimental and clinical work carried out previously which provided valuable subjective reports of the impact of the clock and time monitoring on sleep in PI.

Although the paradigms previously used to establish an attentional bias to sleep in PI are recognised as legitimate measures of attentional bias, it was felt that using a modified Posner paradigm would enable a purer measurement of engagement and disengagement to particular cues presented to the participants.

Posner has suggested that the attention system comprises measurable cognitive components (shift, engage, disengage; Posner, 1980), which are subserved by specific, neural sub-systems (Posner & Petersen, 1990) and which are open to modulation by negative emotional stimuli (Stormark, Laberg, Bjerland, Nordby, & Hugdahl 1995). In the original Posner cue-target paradigm, participants responded to a target appearing in the same (valid) or opposite (invalid) location as a previously presented cue. Results indicated faster detection of targets on cued trails, particularly at short ($<$200 ms) cue-target intervals. This facilitation effect was taken as evidence of the time-cost of disengaging attention from the cue to the target on invalid trials (Posner and Petersen, 1990; Posner, 1988). Over recent years, researchers have begun to apply Posner's attention model to develop a paradigm which can determine whether threatening stimuli can attract attention, i.e. modulate the engagement component of covert attention, and/or hold attention, i.e. modulate the disengage component (Broomfield, Gumley, & Espie, 2005).

Methods

Aims and hypotheses

The aim of this study was two-fold. First, by presenting a pictorial representation of an ecologically valid stimulus, to obtain objective evidence of the influence upon attention of monitoring the clock when set at sleep times and therefore the suggestion of the clock and sleep time as a precursor to pre-sleep worry. Second, to explore systematically, with regard to the components of the attentional system, whether participants would take longer to categorise the target on invalid trials with PI having the longest reaction times due to delayed disengagement from this salient cue compared to normal sleepers (NS).

Design

A 2×2 between-participant design was employed for this experiment comparing NS and PI on validly and invalidly cued trials. All subjects recruited completed the computer task followed by the questionnaires detailed below. On completion of these, subjects were preliminarily assigned to either NS or PI group. Thus, it is important to note that the sleep quality for each participant was not known to the experimenter until the computer task was completed as well as any priming effect on the participants being minimised.

Participants

Participants were recruited through advertising online within the University of Glasgow, a poster campaign around the university campus and through advertising on the psychology department's undergraduate student portal for students to obtain course credits. Each individual was given an appointment time to come into the department but was given no further information on the nature of the experiment, other than that it involved a computer based task.

Sleep quality assessment

The sleep quality assessment had three components:

- an interview structured around the ICSD-2 statement of criteria for psychophysiological insomnia and the DSM-IV statement of criteria for primary insomnia;

- a self-report component where participants completed the Reduced Horne and Ostberg Morningness–Eveningness Questionnaire (MEQ-RF) and the Pittsburgh Sleep Quality Index (PSQI);
- actigraphy to confirm or discount information gained from the Horne and Ostberg questionnaire.

After completion of the computer task, those subjects meeting PI criteria were given an actiwatch to wear and sleep diary to complete for 5 days. These measures helped to confirm diagnosis of PI and to discount any circadian disorder based on data obtained from the MEQ-RF alongside the PSQI such as delayed sleep phase syndrome. Normal sleepers were not given actiwatches due to the confirmatory nature of the actigraphy within this study. Actigraphy was conducted by the Cambridge Neurotechnology 'Actiwatch' system. This comprises a wrist worn unit (model AW-4), roughly the size of a small wristwatch, and sleep analysis software (Actiwatch Sleep Analysis v 1.06), capable of automatically calculating sleep parameters. There is a general consensus that wrist actigraphy provides an accurate, objective estimate of circadian sleep and wake parameters (Tyron, 2004).

Inclusion/exclusion criteria

Participants met combined DSM-IV and ICSD-2 criteria for primary insomnia as well as scoring >6 on the PSQI. PI exclusion criteria included active psychological or drug interventions for sleep problems or when a sleep disorder other than insomnia was suspected. NS were required to score <5 on the PSQI, report no problems with their sleep and have no history of sleep problems. For both groups, scoring above 20 on the Beck Depression Inventory – Short Form (BDI-SF4) resulted in exclusion from analyses. The Spielberger State and Trait Anxiety Inventory was used to provide a comparative measurement of anxiety between NS and PI but was not used for exclusion purposes.

All participants had normal vision or corrected to normal vision. Final allocation of participants to groups for the purposes of analysis did not take place until all sleep assessment data had been collated. No formally screened participants were excluded due to either psychological or drug interventions for sleep or because of substance misuse. However, eight participants were excluded for failure to return all questionnaire measures.

A total of 44 participants were left that met all relevant criteria and completed all measures, 22 classified into PI and 22 into NS. A power calculation carried out prior to the study suggested that 21 participants would be required in each of the groups to detect statistically significant differences at a power of 0.8 with an alpha level set at 0.05.

Apparatus and stimuli

A Tiny Mediabook 380 and the experiment-generation package SuperLab 2.1 (Cedrus Corporation, San Pedro, CA) were used to implement the Posner paradigm. The size of the screen was 38 cm (diagonally). Millisecond timing was recorded through SuperLab Pro and keyboard input.

The sleep related stimuli presented was a photograph of a digital clock showing a time which is usually associated with sleeping in a normal sleep pattern, e.g. '02:00' (see Figs. 1 and 2 for diagrammatical representations of stimuli and paradigm). The stimulus set consisted of 20 digitised pictures of single stimuli. Twenty pictures were presented on the clock in the left hand box and 20 in the right hand box. Target stimuli were either two horizontal dots (..) with a diameter of 0.3 cm or two vertical dots (:) with a height of 0.3 cm. Cue and target stimuli were presented inside two boxes (6 cm high and 10.6 cm wide) and positioned 2.0 cm to the left and to the right of the central fixation point (cross shape). These boxes were continually presented on the screen. Participants were given four practice trials to ensure they were comfortable with completing the task.

Procedure

Individual trials consisted of a fixation cross, presented for 1 s, followed by the clock picture displayed for 250 ms. Targets were then presented in the same or different box (central to where one of the pictures was positioned) and remained on the screen until response. If the participant saw a target which was vertical, they were instructed to press C on the computer keyboard, if the target was horizontal, then they were instructed to press M. When the target is presented in the box on the same side as the stimuli, this is considered a valid trial. However, if the target is presented in the other box on the other side of the computer screen, this is considered an invalid trial. Latencies to detect these targets were

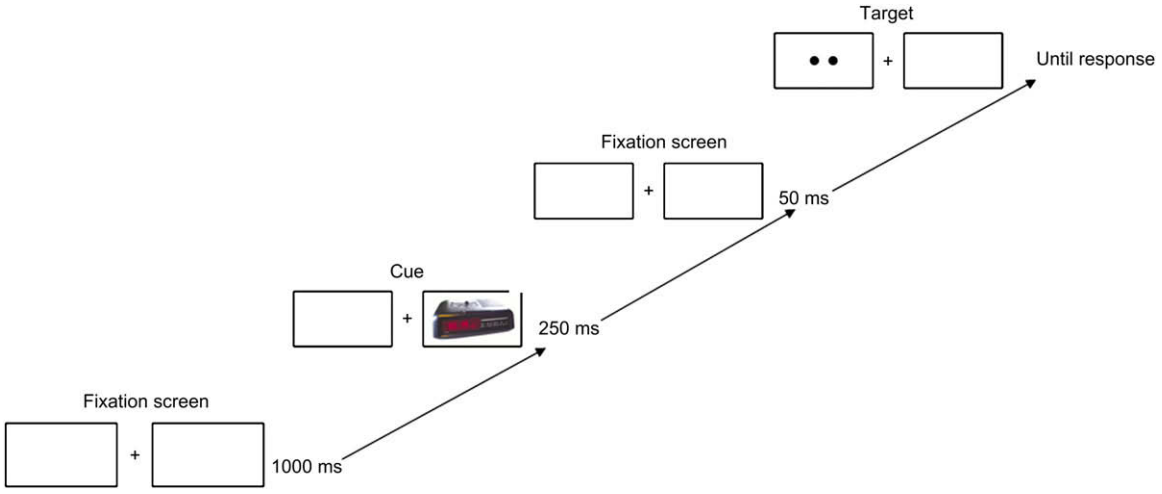


Fig. 1. Presentation sequence and times of the modified Posner paradigm. The trial is valid when the target is presented on the same side of the screen as the pictorial cue of the clock showing a sleep time. The trial is invalid when the target is presented on the opposite side of the screen as the cue (as shown above). A valid trial provides a measure of attentional engagement while an invalid trial provides a measure of attentional disengagement.

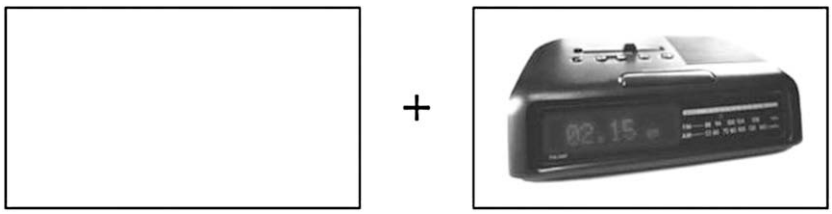


Fig. 2. Enlarged image of the clock showing a sleep time. This is an example of the stimulus used as a cue in the modified Posner paradigm.

used to index the extent to which the groups show an attentional bias. Participants were asked to complete the task as quickly as possible and to remain fixated on the central cross at all times (Figs. 1 and 2).

After completion of the task and the questionnaire pack, the true purpose of the experiment was explained. Participants who were thought to meet criteria for PI were then asked to wear an actiwatch and to complete a sleep diary for five nights following the experiment.

On return of the actiwatch and sleep diary 1 week later, each participant was given a copy of the Good Sleep Guide [prepared by Professor Colin A. Espie for the National Medical Advisory Committee (1994) and now recommended by the British Sleep Society]. Participants were also provided with contact information in case they wanted to be informed of the outcome of the project when it was completed.

Results

The experimental population was equally split between males and females with a mean age of 24 years. Table 1 shows the demographics for each sleep quality group (PI and NS) along with clinical data.

BDI and trait anxiety scores were not significantly different but PI participants had higher state anxiety ($F(1,42) = 4.92$, $p < 0.05$) which could viably reflect an increase in arousal due to presentation of a 'negative' cue.

Table 2 presents summary scores for self-reported sleep data. Analyses revealed a significant effect of group at the level of PSQI with PI having poorer sleep ($F(1,42) = 160.2$, $p < 0.0001$). Total sleep time (TST) was also significantly different ($F(1,42) = 22.34$, $p = 0.0001$) with PI reporting around 5 h of sleep compared to NS 7 h. Sleep onset latency (SOL) was also significantly different ($F(1,42 = 165.88$, $p = 0.0001$) with PI taking approximately 45 min to fall asleep compared to 9 min for NS.

Fig. 3 shows mean (±SE) reaction times on both valid and invalid trials for PI and NS. To assess whether PI was associated with an attentional bias towards sleep related times, we conducted a 2 (group: PI and NS) × 2 (cue location: valid and invalid trial) ANOVA of the RT data. This revealed a significant result for cue location ($F(1,84) = 10.53$, $p = >0.01$) as well as a significant two way interaction between group and cue location ($F(1,84) = 8.98$, $p = >0.01$). We did not obtain a significant result for group ($F(1,84) = 0.51$, $p = 0.48$).

In order to break down the interaction we then examined the data for sleep quality (NS vs. PI) and cue location (valid vs. invalid) separately. Scheffe tests were carried out to make comparisons between means for selected factors. Significant results were found between NS and PI for invalid trials ($F(1,84) = 6.9$, $p < 0.05$) and between invalid and valid trials for PI ($F(1,84) = 19.5$, $p < 0.00001$). These results suggest that PI were slower to respond on invalid trials than NS suggesting a delayed disengagement from the clock cue. There was no significant difference between valid and invalid trials for NS or between NS or PI on valid trials.

Table 1
Demographic and clinical data.

	PI ($n = 22$)		NS ($n = 22$)		Between group analyses
	M	SE	M	SE	
Age (years)	24.4	2.1	23.7	1.8	NS
Gender (% female)	50		50		NS
BDI	2.1	0.47	2.2	0.4	NS
STAI	47.9	2.48	44.6	2.6	NS
SSAI	38.2	1.8	32.2	2.0	$p < 0.05$

BDI, Beck depression inventory; STAI, Spielberger Trait Anxiety Inventory; SSAI, Spielberger State Anxiety Inventory. Significant differences were found between NS and PI on the state anxiety measure which suggests an increase in arousal due to presentation of a salient cue. NS and PI did not differ in mean age, gender split, depression or trait anxiety scores.

Discussion

The purpose of this study was two-fold. First, by presenting a pictorial representation of an ecologically valid stimulus, our aim was to obtain objective evidence of the influence upon attention of monitoring the clock, when set at sleep related time, and therefore the suggestion of the clock as a precursor to pre-sleep worry. Second, we aimed to explore systematically, with regard to the components of the attentional system, whether participants would take longer to categorise the target on invalid trials, with PI having the longest reaction times due to delayed disengagement from this salient cue compared to NS.

In line with our hypothesis we found PI showed delayed disengagement from sleep related times. With reference to Fig. 3, we can see that PI show a speeding effect on valid trials and a delayed response on invalid trials. The salience of the alarm clock cue is holding the attention of PI which enables them to process the target faster when it appears in the position vacated by the cue. This results in a delaying effect on invalid trials as it slows the movement of attention towards the target on the opposite side of the screen.

The lack of effect that the sleep related time cue had on NS reaction time should be considered. Fox, Russo, & Dutton (2002) used the Posner paradigm to compare high and low trait anxious people when presented with angry, happy or neutral faces. With the low anxious group, there was still a main effect for cue validity,

Table 2
Sleep summary data.

	PI ($n = 22$)		GS ($n = 22$)		Between group analyses
	M	SE	M	SE	
PSQI	10.8	0.5	3.0	0.4	$p < 0.0001$
Diary TST (h/min)	5.4	0.2	7.2	0.3	$p < 0.0001$
Diary SOL (min)	44.8	2.6	9.3	0.9	$p < 0.0001$

PSQI, Pittsburgh Sleep Quality Index; TST, total sleep time; SOL, sleep onset latency. Significant differences were found between NS and PI on all sleep measures. Alongside a higher score on the PSQI indicative of poorer sleep, PI took longer to fall asleep (longer SOL) as well as having less total sleep overall (lower TST).

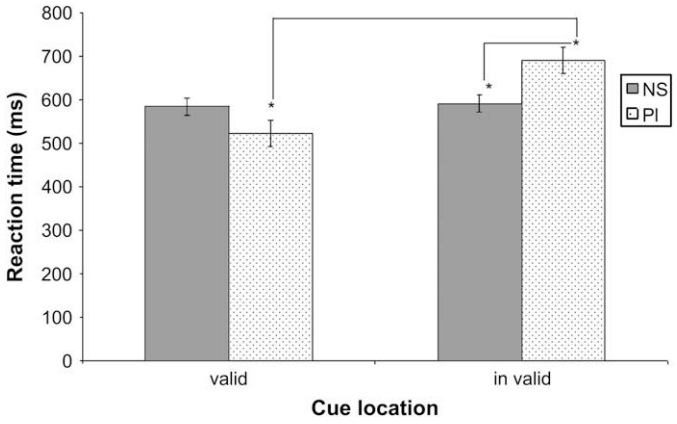


Fig. 3. Reaction time (mean ± SE) on valid and invalid trials of NS and PI. Significant differences were found between PI and NS on invalid trials and between valid and invalid trials for PI.

i.e. whether the target was validly or invalidly cued. This was as expected with longer reaction times for invalid trials compared to valid trials. This would appear to show the 'normal' time course of attention to move from one location to another. However, in the present study, we do not see any effect of cue location in the NS. In the Fox et al. study, the valence of the face presented as a cue will still be salient to an extent independent of the anxiety level of the individual. However, sleep related times may not have an impact on NS and therefore do not affect the movement of their attention in anyway. All participants were instructed to keep their eyes fixated on the cross in the centre of the computer screen. This suggests the sleep time cue was either not registering or was having very little impact on the attention of normal sleepers which is in direct contrast to PI who were very much affected by the sleep time cue. Here we present stimuli which are causing a very different reaction in individuals with a particular psychopathology compared to those without, who are displaying no effect at all.

By confirming our hypothesis that PI show a delayed disengagement from sleep related times presented on a clock, we have provided evidence for an attentional bias towards sleep related times. These results support previous work carried out using other paradigms to demonstrate an attentional bias towards sleep related stimuli (Jones, Macphee, Broomfield, Jones, & Espie, 2005; MacMahon, Broomfield, Macphee, & Espie, 2006; Marchetti et al., 2006). However, this study is novel in two ways. First, by focusing on sleep related times the present study provides objective evidence for a cognitive process which is argued to be fundamental in the maintenance of insomnia (Tang et al., 2007).

Second, we used a paradigm which directly measures attention to a familiar yet clinically relevant stimulus. By selecting a modified Posner paradigm, we obtain a measure of time taken to disengage from the clock which can therefore be attributed to attention rather than response preparation as the location of the cue is not associated with the correct response. Also, the information required to make the correct response can only be obtained from the cue itself.

This study moves insomnia research forward by objectively validating the clock as a salient sleep stimulus which impacts on attentional processing in PI and therefore impacting on sleep. By establishing an attentional bias to sleep we are able to investigate further the validity of attentional bias as a marker of disorder severity and conversely as a marker of treatment efficacy. Other areas of research such as sleep in cancer patients (Espie et al., 2008) and smoking abstinence programs (Waters, Shiffman, Bradley, & Mogg, 2003) have shown attentional bias towards the relevant salient stimuli to change in accordance with reported treatment success. Bearre, Sturt, Bruce, and Jones (2007) report a relational attentional bias in clients engaging in a heroin harm reduction service, i.e. attentional bias towards heroin increases as frequency of use increases which suggests attentional bias as a potential indicator of disorder progression.

However, there are methodological limitations to this study. First, because this was a recruited population, our ability to generalise to the clinical population may be limited. Attention bias studies, therefore, require replication with clinical samples. Nevertheless, it should be noted that non-clinical populations do display cognitive over-activity at bedtime, with clock monitoring among the most common (Harvey, 2002). Processes detected at any stage of sleep disruption, particularly before the clinical extreme, may provide crucial evidence about the mechanisms involved in the maintenance/enhancement of the disorder proper.

In conclusion, we used a cognitive probe paradigm to provide objective evidence for sleep related attentional bias in PI. This provides support for the previous research done in this field (Jones et al., 2005; MacMahon et al., 2006; Marchetti et al., 2006) and further supports the role of attention in the attention–intention–effort pathway into PI (Espie et al., 2006) by impeding the automatic passage to sleep. Also, by using the clock and sleep time, further objective evidence is provided for the role of clock monitoring which has been implicated as a trigger to the cognitive arousal widely reported by those with PI. The establishment of an attentional bias in PI provides valuable insight into the development and maintenance of insomnia, however more research is needed to understand the role of threat and craving as the underlying mechanisms for selective attention towards sleep and attentional bias as an indicator of psychopathology progression.

Acknowledgements

This research was supported by the Carnegie Trust for the Universities of Scotland and a Glasgow University graduate scholarship.

References

American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders (DSM-IV)* (4th ed.). Washington, DC: APA.

American Sleep Disorder Association. (1997). *International classification of sleep disorders (ICSD-2): Revised diagnostic and coding manual*. Rochester, MN: APA.

Bearpark, H. (1994). *Overcoming Insomnia (what is causing your sleeplessness and what you can do about it)*. Australia: Gore & Osment Publications.

Bearre, L., Sturt, P., Bruce, G., & Jones, B. T. (2007). Heroin-related attentional bias and monthly frequency of heroin use are positively associated in attenders of a harm reduction service. *Addictive Behaviors, 32*(4), 784–792.

Broomfield, N. M., Gumley, A. I., & Espie, C. A. (2005). Candidate cognitive processes in psychophysiological insomnia. *Journal of Cognitive Psychotherapy, 19*, 5–17.

Espie, C. A. (2002). Insomnia: conceptual issues in the development, persistence and treatment of sleep disorder in adults. *Annual Review of Psychology, 53*, 215–243.

Espie, C. A., Brooks, D. N., & Lindsay, W. R. (1989). An evaluation of tailored psychological treatment for insomnia in terms of statistical and clinical measures of outcome. *Journal of Behavior Therapy and Experimental Psychiatry, 20*, 143–153.

Espie, C. A., Broomfield, N. M., MacMahon, K. M. A., Macphee, L. M., & Taylor, L. M. (2006). The attention–intention–effort pathway in the development of psychophysiological insomnia: a theoretical review. *Sleep Medicine Reviews, 10*, 215–245.

Espie, C. A., Fleming, L., Cassidy, J., Samuel, L., Taylor, L. M., White, C. A., Douglas, N. J., Engleman, H. M., Kelly, H.-L., & Paul, J. (2008). Randomized controlled clinical effectiveness trial of cognitive behavior therapy compared with treatment as usual for persistent insomnia in patients with cancer. *Journal of Clinical Oncology, 26*(28), 4651–4658.

Fox, E., Russo, R., & Dutton, K. (2002). Attentional bias for threat: evidence for delayed disengagement from emotional faces. *Cognition and Emotion, 16*(3), 355–379.

Harvey, A. G. (2001). Insomnia: symptom or diagnosis? *Clinical Psychology Review, 21*(7), 1037–1059.

Harvey, A. G. (2002). A cognitive model of insomnia. *Behaviour Research and Therapy, 40*, 869–893.

Jones, B. T., Macphee, L. M., Broomfield, N. M., Jones, B. C., & Espie, C. A. (2005). Sleep related attentional bias in good, moderate and poor (primary insomnia) sleepers. *Journal of Abnormal Psychology, 114*(2), 249–258.

Lichstein, K. L., & Rosenthal, T. L. (1980). Insomniacs' perceptions of cognitive versus somatic determinants of sleep disturbance. *Journal of Abnormal Psychology, 89*, 105–107.

MacMahon, K., Broomfield, N., Macphee, L., & Espie, C. A. (2006). Attention bias for sleep related stimuli in primary insomnia and delayed sleep phase syndrome using the dot-probe task. *Sleep, 29*, 11.

Marchetti, L. M., Biello, S. M., Broomfield, N. M., MacMahon, K. M. A., & Espie, C. A. (2006). Who is pre-occupied with sleep? A comparison of attention bias in people with psychphysiological insomnia, delayed sleep phase syndrome and good sleepers using the induced change blindness paradigm. *Journal of Sleep Research, 15*, 212–221.

Morin, C. M. (1993). *Insomnia: psychological assessment and management*. New York: The Guilford Press.

Ohayon, M. (1996). Epidemiological study on insomnia in the general population. *Sleep, 19*, S7–S15.

Ohayon, M. M. (2002). Epidemiology of insomnia: what we know and what we still need to learn. *Sleep Medicine Reviews, 6*, 97–111.

Posner, M. I. (1980). Orienting of attention. *Quarterly Journal of Experimental Psychology, 32A*, 3–25.

Posner, M. I. (1988). Structures and functions of selective attention. In T. Boll, & B. Bryant (Eds.), *Master lectures in clinical neuropsychology* (pp. 173–202). Washington, DC: Am. Psych. Assoc.

Posner, M. I., & Petersen, S. E. (1990). The attention system of the human brain. *Annual Review of Neuroscience, 13*, 25–42.

Stormark, K. M., Laberg, J. C., Bjerland, T., Nordby, H., & Hugdahl, K. (1995). Autonomic cued reactivity in alcoholics: the effect of olfactory stimuli. *Addictive Behaviors, 20*, 571–584.

Tang, N. K. Y., Schmidt, D. A., & Harvey, A. G. (2007). Sleeping with the enemy: clock monitoring in the maintenance of insomnia. *Journal of Behavior Therapy and Experimental Psychiatry, 38*, 40–55.

Taylor, L., Espie, C. A., & White, C. A. (2003). Attentional bias in people with acute versus persistent insomnia secondary to cancer. *Behavioral Sleep Medicine, 1*(4), 200–212.

Tyron, W. W. (2004). Issues of validity in actigraphic sleep assessment. *Sleep, 27*, 158–165.

Waters, A. J., Shiffman, S., Bradley, B. P., & Mogg, K. (2003). Attentional shifts to smoking cues in smokers. *Addiction, 98*(10), 1409–1417.

Volume 48 Issue 6 June 2010 ISSN 0005-7967

ELSEVIER

BEHAVIOUR RESEARCH AND THERAPY

An International Multi-Disciplinary Journal

Contents

Regular Papers

Continued on outside back cover

Abstracted/indexed in: Biosis Data, Elsevier BIOBASE/Current Awareness in Biological Sciences, Curr. Cont./Soc. & Beh. Sci., EMBASE/Excerpta Medica, Ind. Med., LLBA, MEDLINE, PASCAL-CNRS Data., Psychol. Abstr., PsycSCAN, PsycINFO, PsycLIT, Res. Alert, Soc. Sci. Cit. Ind., Social Work Res. & Abstr. Also covered in the abstract and citation database SCOPUS®. Full text available on ScienceDirect®.

ISSN 0005-7967
48(6) 449–570 (2010)

Behaviour Research and Therapy 48 (2010) 467–475

Contents lists available at ScienceDirect

Behaviour Research and Therapy

journal homepage: www.elsevier.com/locate/brat

Psychophysiological reactivity to sleep-related emotional stimuli in primary insomnia

Chiara Baglioni [a,*], Caterina Lombardo [a], Emiliano Bux [a], Stig Hansen [b], Christine Salveta [c], Stephany Biello [d], Cristiano Violani [a], Colin A. Espie [c]

[a] Department of Psychology, "Sapienza" University of Rome, Via dei Marsi 78, 00185 Rome, Italy
[b] Department of Clinical Physics, South Glasgow University Hospitals Division, Southern General Hospital, 1345 Govan Road, Glasgow G51 4TF, Scotland, UK
[c] Section of Psychological Medicine and Sleep Research Laboratory, University of Glasgow, Sackler Institute of Psychobiological Research, Southern General Hospital, 1345 Govan Road, Glasgow G51 4TF, Scotland, UK
[d] Department of Psychology, University of Glasgow, 58 Hillhead Street, Glasgow G12 8QB, Scotland, UK

ARTICLE INFO

Article history:
Received 16 August 2009
Received in revised form
11 December 2009
Accepted 29 January 2010

Keywords:
Primary insomnia
Emotions
International Affective Picture System
Facial EMG
Heart rate
Cardiac vagal tone

ABSTRACT

The present study examined psychophysiological reactivity to emotional stimuli related and non-related to sleep in people with primary insomnia (PPI) and in good sleepers (GS). Twenty-one PPI and 18 GS were presented with five blocks of neutral, negative, positive, sleep-related negative and sleep-related positive pictures. During the presentation of the pictures, facial electromyography (EMG) of the corrugator and the zygomatic muscles, heart rate (HR) and cardiac vagal tone (CVT) were recorded. Subjective ratings of the stimuli were also collected. We found that only PPI exhibited greater inhibition of the corrugator activity in response to sleep-related positive stimuli compared to the other blocks of stimuli. Furthermore, PPI rated the sleep-related negative stimuli as more unpleasant and arousing and showed higher CVT in response to all stimuli as compared to GS. Results were interpreted as indicating that PPI exhibit craving for sleep-related positive stimuli, and also hyper-arousability in response to sleep-related negative stimuli, as compared to GS. Our results suggest that psychological treatment of insomnia could benefit by the inclusion of strategies dealing with emotional processes linked with sleep processes.

Introduction

Insomnia as a disorder is defined as difficulties in initiating/maintaining sleep and/or non-restorative sleep accompanied by decreased daytime functioning persisting for at least four weeks (American Academy of Sleep Medicine, AASM, 2005). Aetiological theories (Espie, 2002; Espie, Broomfield, MacMahon, MacPhee, & Taylor, 2006; Harvey, 2002; Lundh & Broman, 2000; Morin, 1993; Perlis, Giles, Mendelson, Bootzin, & Wyatt, 1997; Riemann et al., 2010) consider heightened levels of autonomic, cortical, cognitive, and emotional arousal as relevant maintaining factors of insomnia. Although widely recognized as important (e.g. Espie, 2002; Lundh & Broman, 2000; Morin, 1993; Riemann et al., 2010), investigations of the aetiological role of emotional arousal in insomnia have been classically based on indirect evidence. These have involved studies showing that neurotic temperamental traits are associated with insomnia (e.g. Espie, 1991; LeBlanc et al., 2007), studies investigating the content of intrusive thoughts experienced by people with insomnia during the pre-sleep period (e.g. Van Egeren, Hayness, Franzen, & Hamilton, 1983; Wicklow & Espie, 2000), and studies demonstrating significant comorbidity with clinical anxiety or depression (e.g. Chang, Ford, Mead, Cooper-Patrick, & Klag, 1997; Ford & Kamerow, 1989; Riemann, 2007).

Considering the dimensional approach to the study of emotions (e.g. Bradley, 2000; Lang, 2002, chap. 14), the emotional experience is described as referring to two main dimensions: the valence (positive vs negative) and the arousal (from low to high). Based on this, the majority of research considering insomnia has largely focussed on the dimension of arousal, but not on the dimension of valence. A small number of studies have investigated the relationship between positive (e.g. excited, enthusiastic) and negative (e.g. hostile, upset) affective states and poor sleep quality using self-report questionnaires (e.g. McCrae et al., 2008; Norlander, Johansson, & Bood, 2005; Scott & Judge, 2006). These studies

* Correspondence to: Chiara Baglioni, Department of Psychiatry & Psychotherapy, University of Freiburg Medical Center, Hauptstraße 5, 79104 Freiburg, Germany. Tel.: +49 761 270 6589; fax: +49 761 270 6619.
E-mail addresses: chiara.baglioni@uniklinik-freiburg.de (C. Baglioni), caterina.lombardo@uniroma1.it (C. Lombardo), emiliano.bux@alice.it (E. Bux), s.hansen@clinmed.gla.ac.uk (S. Hansen), c.salveta@clinmed.gla.ac.uk (C. Salveta), s.biello@psy.gla.ac.uk (S. Biello), Cristiano.Violani@uniroma1.it (C. Violani), c.espie@clinmed.gla.ac.uk (C.A. Espie).

0005-7967/$ – see front matter © 2010 Elsevier Ltd. All rights reserved.
doi:10.1016/j.brat.2010.01.008

showed that people with insomnia report heightened negative and diminished positive emotional states as compared to good sleepers. Three studies have examined the relationship between emotions and sleep measuring psychophysiological responses to visual stimuli validated both for the dimensions of arousal and valence. In two studies, Franzen, Buysse, Dahl, Thompson, and Siegle (2009) and Franzen, Siegle, and Buysse (2008) measured pupillary responses to high arousing positive and negative emotional pictures and low arousing neutral pictures in sleep-deprived participants as compared to control groups. Main results showed that responses to negative pictures were larger in sleep-deprived participants as compared to controls. In a functional magnetic resonance imaging (fMRI) design study of sleep deprivation, Yoo, Gujarm, Hu, Jolesz, and Walker (2007) presented pictures ranging from emotionally neutral (neutral valence, low arousal) to increasingly aversive quality (negative valence, high arousal). The sleep deprivation group displayed enhanced activity in the amygdala and reduced functional connectivity between the amygdala and the medial prefrontal cortex (MPFC). These results were interpreted as an increased neurobiological response to emotional stimuli and a reduced inhibitory influence of the MPFC on emotional reactivity after sleep deprivation. Taken together, these studies show that sleep deprivation alters emotional responses to negative stimuli, thus, sleep seems to be important for maintaining adaptive emotional processes. However, as these studies focussed on sleep deprivation, no data about emotional reactivity in insomnia are available up to date. In fact, insomnia is a condition which differs from sleep deprivation because it implies chronic sleep difficulties (more than four weeks), primarily subjective complaints, impairments in sleep quality and not necessarily in sleep quantity, and adaptation processes. Moreover, the studies aforementioned did not specifically considered the valence dimension. In fact, Yoo et al. (2007) did not use positive stimuli, and Franzen et al. (2009, 2008) used pupillary responses which are indices of more general emotional information processing. Bradley (2000) has indicated that facial electromyography (EMG) measures specifically the emotional valence. The activity of the corrugator muscle (knits the eye brow into a frown) is both potentiated by unpleasant pictures and inhibited by pleasant pictures when compared to neutral stimuli. In contrast, the activity of the zygomatic muscle (pulls the corners of the mouth back and up into a smile) increases in response to pleasant pictures, while unpleasant stimuli elicit no inhibition (e.g. Larsen, Norris, & Cacioppo, 2003). The aim of the present study was to evaluate facial EMG responses to positive and negative emotional stimuli related and not related to sleep in people with primary insomnia and good sleepers. We assumed that the presentation of symptom-relevant emotional stimuli would enhance responses in the group with insomnia due to a greater attention directed to this material and due to personal relevance.

The following predictions were considered. As compared to neutral stimuli and as compared to good sleepers, people with primary insomnia should exhibit enhanced corrugator activity in response to sleep-related negative stimuli. Additionally, they may show diminished corrugator activity and heightened zygomatic activity in response to sleep-related positive stimuli. With respect to stimuli not related to sleep, people with primary insomnia should present enhanced responses in the corrugator activity to negative stimuli. Additionally, two alternative predictions were considered with respect to positive stimuli. Diminished responses to positive stimuli in the zygomatic muscle have been reported for individuals with dysphoria compared to healthy controls (Sloan, Bradley, Dimoulas, & Lang, 2002). As insomnia is a condition highly linked with depression and with a waking complaint of negative mood changes, we may see a reduced response in the zygomatic muscle to positive stimuli in individuals with primary insomnia. Alternatively, this reduced response may be unique to depression as primary insomnia is an independent diagnostic entity from mood disorders (e.g. Perlis et al., 2006; Riemann & Voderholzer, 2003).

In addition to these hypotheses, we also expected that people with primary insomnia would respond with higher autonomic arousal to all stimuli. Thus, numerous studies have demonstrated increased levels of arousal in people with primary insomnia when compared to good sleepers (for a review see Riemann et al., 2010). Additionally, Lundh and Broman (2000) suggested that people with insomnia are characterized by an increase in the levels of arousal in response to sudden, new or emotional stimuli (*hyper-arousability*). In the current experiment, we also recorded heart rate (HR) and cardiac vagal tone (CVT) during the presentation of the visual stimuli. As an increase of the heart rate is linked to heightened arousal stimulation (Witvliet & Vrana, 1995) and higher CVT has been associated with greater reactivity to stimuli (Movius & Allen, 2005), we predicted alterations in these measures.

Finally, subjective ratings of the valence and the arousal of the pictorial stimuli were also collected. We predicted that people with primary insomnia would demonstrate an altered valence and arousal ratings of sleep-related stimuli as compared to good sleepers.

Method

Participants

Participants were 39 (31 F, 8 M) university students, including 21 individuals with primary insomnia and 18 good sleepers (Table 1). Their age ranged between 18 and 30 years (mean age ± standard deviation – sd = 22.4 y ± 2.96). Participants with primary insomnia (PPI) met the Diagnostic and Statistic Manual of Mental Disorders, Fourth Edition (DSM-IV, American Psychiatry Association, APA, 2002) criteria as measured through screening questionnaires and a clinical assessment interview. These criteria include: duration of the symptoms of at least one month, and complaints of decreased functioning at work and/or in social and personal life. Additionally, PPI met quantitative criteria of insomnia as reported by Lichstein, Durrence, Taylor, Bush, and Riedel (2003) which indicate a frequency of the symptoms of three or more nights per week. Controls participants (good sleepers – GS) met the Research Diagnostic Criteria for normal sleepers indicated by Edinger et al. (2004).

This study arises from a collaboration between the University of Glasgow Sleep Research Laboratory (UGSRL) and the Department of Psychology of the "Sapienza" University of Rome. Nineteen participants were recruited and recorded in Glasgow, 11 PPI (all F, 20.54 y ± 1.69) and 8 GS (5 F; 3 M, 23.25 y ± 3.06). Twenty participants were recruited and recorded in Rome, 10 PPI (8 F; 2 M, 23.7 y ± 2.49) and 10 GS (7 F; 3 M, 22.4 y ± 3.68).

Table 1
Study participants.

	PPI	GS	X^2/F	p
Women/men	19/2	12/6	$X^2_{(1,N=39)} = 3.37$	0.07
Age (in years)	22.8 ± 3.31	22.0 ± 2.64	$F_{(1,37)} = 0.58$	0.45
ISI	12.09 ± 3.32	1.33 ± 1.53	$F_{(1,37)} = 159.86$	<0.001
STAI-T	44.09 ± 8.53	36.11 ± 6.99	$F_{(1,37)} = 10.01$	0.03
MEQ	43.14 ± 8.71	47.72 ± 7.72	$F_{(1,37)} = 2.97$	0.09
KSS	5.00 ± 2.15	4.39 ± 1.65	$F_{(1,36)} = 0.95$	0.34

Note. Data are presented as mean ± sd. One-way ANOVAs and chi-squares were computed. Abbreviations: ISI: Insomnia Severity Index; STAI-T: State-Trait Anxiety-Trait version; MEQ: Morningness–Eveningness Questionnaire; KSS: Karolinska Sleepiness Scale; PPI: People with Primary Insomnia; GS: Good Sleepers.

The duration of insomnia was 51 ± 42 months (mean ± sd), ranging from 1 to 120 months. Five participants out of 21 reported difficulties exclusively in sleep onset, indicating difficulties in falling asleep within 30 min. All others participants reported mixed symptoms (difficulty in falling asleep within 30 min, difficulty in maintaining sleep during the night with a total time awake longer than 30 min, and wakening up in the morning at least 60 min before of the wanted time and with less than 6 h of sleep). Quality of sleep was also assessed in GS using questionnaires and the clinical assessment interview.

None of the participants reported the use of sleep medication, any relevant psychiatric conditions or other medical problems as assessed by both the questionnaires and the clinical assessment interview.

Written informed consent was obtained prior to the onset of the study. The research was approved by both the Ethics Committees of the NHS Greater Glasgow and Clyde, UK and the Department of Psychology of the "Sapienza" University of Rome, Italy.

Screening measures

1) Sleep Disorder Questionnaire (SDQ, Violani, Devoto, Lucidi, Lombardo, & Russo, 2004). The SDQ is a brief self-report categorical questionnaire which evaluates the presence of insomnia according to the DSM-IV (APA, 2002) and the quantitative (Lichstein et al., 2003) criteria. Respondents are classified into three categories: Good sleep; Clinically significant insomnia; Subthreshold insomnia. The SDQ has been validated as a brief and valid instrument, useful for the screening of insomnia (Violani et al., 2004).
2) Insomnia Severity Index (ISI, Bastién, Valliéres, & Morin, 2001). The ISI scale assesses the severity of insomnia during the previous two weeks. Respondents are classified into four categories: No clinically significant insomnia (score between 0 and 7); Subthreshold Insomnia (score between 8 and 14); Clinical insomnia – moderate severity (score between 15 and 21); Clinical Insomnia – severe (score between 22 and 28).

The order of SDQ and ISI was counterbalanced across subjects.

In the current study, participants were included in the GS group if they were classified in the category "good sleep" at the SDQ and scored below 8 on the ISI. With respect to the PPI group, participants were included if they were classified in the category "clinically significant insomnia" at the SDQ or scored above 14 on the ISI. Consequently, participants classified in the category "subthreshold insomnia" at the SDQ and scoring between 8 and 14 on the ISI were not selected. Moreover, participants classified in the category "good sleep" at the SDQ and scoring above 8 on the ISI were not selected, as those classified in the categories "subthreshold insomnia" or "clinically significant insomnia" at the SDQ and scoring below 8 on the ISI.

3) State-Trait Anxiety Inventory-Trait version (STAI-T, Spielberger, Gorsuch, & Lushenne, 1970). The STAI-T uses a 20-item scale. Respondents are asked to rate how they generally feel on a 5-point frequency scale ranging from 1 (almost never) to 4 (very much so). The STAI-T is a widely used measure of trait anxiety. The total score ranges from 20 to 80. Sesti (2000) indicates 2 cut-off scores: 40 for "moderate anxiety", 60 for "clinical anxiety"; scores below 40 are considered low anxiety. In the current study, participants were included if they scored below 60.
4) Morningness–Eveningness Questionnaire (MEQ, Horne & Ostberg, 1976). The MEQ is a 19-item questionnaire which assesses the circadian preference. The MEQ categorizes responses into five possible groups: "definitely morning types" (score between 70 and 86), "moderately morning types" (score between 59 and 69), "neither types" (score between 42 and 58), "moderately evening types" (score between 31 and 41), "definitely evening types" (score between 16 and 30). The MEQ was used in order to exclude those participants with a problem of insomnia due to advanced or delayed phase syndrome. As our sample was a student sample, advanced or delayed phase problems could be frequent due to irregular life-style times. Only participants classified as "neither types" or "moderately morning or evening types" were included in the study.
5) The Edinburgh Handedness Inventory (Oldfield, 1971). This is a self-report questionnaire which identifies the hand preference of the respondents. Participants are asked to report with which hand they do a list of actions (writing, drawing, throwing, scissors, toothbrush, knife, spoon, broom, sticking a match, opening a box). To calculate the score, the difference between the score related to the right hand and the score related to the left hand is computed. This difference is then divided for the total score and the value is multiplied for 100. Thus, the score ranges from −100 and +100, positive values indicating a tendency to right handed. In the current study, as asymmetry for specialization of brain hemispheres with respect to emotion processes have been reported (e.g. Zhou & Hu, 2006), only definitely right-handed participants (score $> +40$) were included.

All questionnaires have both the Italian and the English versions validated in samples independent from the one of the present study.

Procedure

All potential participants were approached by email or around universities sites (e.g. library, campus, lectures halls, etc.). One hundred and ten persons accepted to fill out the screening measures in Glasgow and 134 in Rome. Of those, 41 participants proved to be eligible in Glasgow, 20 GS and 21 PPI. Thirty-two participants proved to be eligible in Rome, 17 GS and 15 PPI. Both in Glasgow and in Rome, participants of the two groups were further selected to be matched on the basis of STAI-T and MEQ scores, and on the basis of hand preference. In total, 19 people were selected in Glasgow (8 GS, 11 PPI) and 21 in Rome (10 GS, 11 PPI). Based on questionnaires responses, selected participants were invited to the sleep laboratory for the experimental session, which was preceded by a clinical interview conducted by the first author (CB). One of the participants recruited within the Italian PPI cohort was excluded after the clinical interview because insomnia criteria were not satisfied.

Selected participants received detailed instructions for the experimental section and had electrodes attached as described below. A Trackit™ ambulatory Electroencephalography (EEG)/polygraphic recorder (www.llines.com) was used for acquiring data in both laboratories. The sampling rate was 256 Hz. Facial EMG over the corrugator and the zygomatic muscles on both sides of the face was recorded using 6 mm surface silver–silver chloride (Ag/AgCl) electrodes. The EMG electrodes were connected to the Trackit's bipolar channels. The "Guidelines for Human Electromyographic Research" (Fridlund & Cacioppo, 1986) were followed for the placement of the electrodes. Electrodes were attached with the GRASS adhesive paste (www.grasstechnologies.com). A single channel Lead II Electrocardiography (ECG) was recorded, using two AmbuRNeuroline 720

Single Patient Surface Electrodes (www.ambu.com), one placed just below the right clavicle, one on the left side of the torso. A third surface electrode placed over the forehead was used as ground electrode. Heart rate (HR) and cardiac vagal tone (CVT) were derived from the ECG R–R interval using the Neuroscope™ software (www.medifitgroup.com). The non-invasive index of CVT is defined as 'pulse-synchronised phase shifts in consecutive cardiac cycles' (Little, Julu, Hansen, & Reid, 1999), and is measured in arbitrary units of a linear vagal scale (LVS) (Julu, 1992). The least value in this scale is zero, equivalent to full atropinisation of human subjects (Julu, 1992).

A biocalibration was provided to check the recording: participants were asked to frown as if they were very angry and then to smile as if they were very happy. Before starting with the image viewing, participants were asked to sit on a chair in front of the computer screen at a distance of 60 cm. The size of the screen was 17 inches. The chair height was adjusted so that the centre of the computer screen was at the eye level of the participant. Images were presented in five different blocks: neutral, negative not related to sleep, negative related to sleep, positive not related to sleep, positive related to sleep. Blocks were counterbalanced within a Latin square in 5 different sequences.[1] Each sequence started with 2 neutral stimuli which were not considered for data analysis, but used for adaptation. At the end of each block the participants were asked to rate the valence and the arousal of all the pictures of the block using a subjective scale, the Self-Assessment Manikin (SAM, Bradley & Lang, 1994). Pictures were presented with Superlab, version 2.1 (www.cedrus.com).

While viewing the visual stimuli, the room was only illuminated by a table lamp shining on the wall, while during the rating the full room light was lighted. Participants were instructed to keep their head still while watching the stimuli. In order to increase the emotional impact of the stimuli, before starting with the presentation of the pictures, participants were asked to read a paragraph describing the experience of a night with insomnia taken from "*Overcoming Insomnia and Sleep Problems: A self-help guide using cognitive behavioural techniques*" (Espie, 2006, p. 62). During the task the experimenter stood behind the participant. A qualified psychologist and an advanced undergraduate psychology student conducted the experiments. Good sleepers received payment of 10 euros (or 6 English pounds) for their participation, while PPI additionally received a self-help booklet dealing with the Cognitive-Behaviour Treatment strategies of Insomnia (CBTI) developed at the Department of Psychology of the "Sapienza" University of Rome and adapted for an Italian and an English version.

Control measure for levels of sleepiness during the experimental session

The Karolinska Sleepiness Scale (KSS, Akerstedt & Gillberg, 1990). The KSS is a commonly used measure assessing state levels of sleepiness. It uses a 9-point scale ranging from 1 = "very alert" to 9 = "very sleepy, great effort to keep awake, fighting sleep". Participants are instructed to rate their sleepiness in the 10 min prior to taking the test. The KSS was administered immediately before the presentation of the pictures. A score of 7 or more indicates excessive sleepiness.

Subjective ratings of the stimuli

The Self-Assessment Manikin (SAM, Bradley & Lang, 1994). The SAM is a non-verbal pictorial assessment technique that includes manikins differing in facial expressions and aiming at measuring the dimensions of valence and arousal on two continuous 9-point scales. The scale used for assessing the valence dimension ranges from a smiling happy man to an unhappy, frowning man. The scale used for assessing the arousal dimension ranges from an excited, wide-eyed figure to a relaxed, sleepy figure.

Picture stimuli

Negative and positive stimuli not related to sleep as well as neutral stimuli were selected from the International Affective Picture System (IAPS, Lang, Bradley, & Cuthbert, 2001).[2] This is the most widely used collection of visual stimuli for inducing emotions in laboratory tests. It includes colour photographs with a known valence and arousal evaluated through the Self-Assessment Manikin. All selected stimuli had low to medium levels of arousal (<6). Twelve neutral stimuli (valence between 4 and 6; mean valence 4.97 ± 0.35, mean arousal 3.45 ± 0.62), 10 positive stimuli (valence > 6; mean valence 7.64 ± 0.60, mean arousal 4.39 ± 0.35), 10 negative stimuli (valence < 4; mean valence 2.46 ± 0.82, mean arousal 5.10 ± 0.60) were selected. No stimuli representing socially desirable situations (e.g. weddings) were chosen.

Stimuli related to sleep[3] were 10 negative (valence < 4; mean valence 3.18 ± 0.50, mean arousal 4.60 ± 0.42) and 10 positive (valence > 6; mean valence 7.47 ± 0.57, mean arousal 1.90 ± 0.21) pictures, which valence and arousal were previously assessed through the Self-Assessment Manikin. The validation of the sleep-related stimuli was tested on a different sample of 30 volunteers college students (18 F, 12 M) of the Faculty of Psychology of the "Sapienza" University of Rome, aged between 18 and 30 years. The same procedure used to validate the IAPS stimuli (Lang et al., 2001) was followed in the validation of the sleep-related stimuli. Negative sleep-related stimuli included pictures of people awake in bed at night-time. Positive sleep-related stimuli included pictures of people sleeping in bed at night-time. A statement congruent with respect to the valence of the stimulus was added to each picture in order to maximise the emotional impact of the stimuli. It was assumed that stimuli affectively congruent enhance the activation levels and facilitate responses that reach a threshold level that is necessary for identification. This assumption is consistent with the model proposed by Fazio, Sanbonmatsu, Powell, and Karders (1986), which assumes that all affective concepts with the same valence are linked in semantic memory and their combination facilitates the affective response and categorization. All sentences were derived from validated questionnaires, namely the statements for the IAPS positive pictures were taken from the State-Trait Anxiety Inventory (STAI, Spielberger et al., 1970), the statements for the IAPS negative pictures were taken from the Fear Survey Schedule (FSS, Wolpe & Lang, 1964) and the statements for the sleep-related pictures were taken from the Glasgow Content of

[1] Sequence 1: neutral – positive not related to sleep – negative not related to sleep – positive related to sleep – negative related to sleep; Sequence 2: positive not related to sleep – negative not related to sleep – positive related to sleep – negative related to sleep – neutral; Sequence 3: negative not related to sleep – positive related to sleep – negative related to sleep – neutral – positive not related to sleep; Sequence 4: positive related to sleep – neutral – positive not related to sleep – negative related to sleep – negative not related to sleep; Sequence 5: negative related to sleep – negative not related to sleep – neutral – positive not related to sleep – positive related to sleep.

[2] Numbers of the IAPS stimuli selected: *X*: 2038, 2102, 2372, 2383, 2385, 2480, 2487, 2493, 2516, 2595, 2840, 7550; *P*: 2050, 2070, 2154, 2311, 2352, 2395, 2510, 2530, 7325, 8497; *N*: 2276, 2490, 2795, 3180, 3350, 6562, 8231, 9221 9435, 9594.

[3] Sleep-related stimuli are distributed on CD-ROM and can be obtained on request from the authors.

Thoughts Inventory (GCTI, Harvey & Espie, 2004).[4] The statements for the neutral stimuli consisted in the description of the picture. All statements were formulated in the first person. Examples of statements for each valence category are: 1) positive not related to sleep stimuli: I am happy (IAPS picture number: 2070); 2) negative not related to sleep stimuli: I fear weapons (IAPS picture number: 6562); 3) positive sleep-related stimuli: I am good at falling asleep; 4) negative sleep-related stimuli: I worry about not sleeping well; 5) neutral stimuli: I am working (IAPS picture number: 7550).

Stimuli were balanced with respect to complexity, brightness, contrast of the image, and number of words in associated statements.

Results

Data preparation and analyses

Statistical analyses were conducted using the STATISTICA software (version 6, www.statsoft.com).

1) Description of the sample. Groups were compared using one-way ANOVAs (with respect to age, sleepiness, anxiety and severity of the insomnia symptoms) and chi-squares (with respect to GENDER).
2) Facial electromyography (EMG). The Spike-2 software, version 5 (www.ced.co.uk), was used for preparing the EMG data. Raw data were high-pass filtered for eliminating the DC signal. Data were then rectified, smoothed and transformed into logarithm to normalise the distribution. According to the "Guidelines for human electromyographic research" (Fridlund & Cacioppo, 1986), most psychophysiological research using EMG has focussed on some variations of EMG signal amplitude as the dependent measure. Commonly, reflexes are often quantified using peak-to-peak EMG amplitude and onset latency (phasic responses). However, the guidelines also underline that it has been suggested that maintained EMG activity represent muscular contraction more accurately than a simple amplitude. Research has used, thus, also the mean of the rectified, smoothed, and filtered EMG signal (tonic responses). In this study, we have considered both phasic and tonic responses to analyse the EMG responses to the stimuli.

   Phasic responses: Changes in the phasic responses were obtained as the difference between the peak-to-peak amplitude of the EMG recorded during the presentation of each block of pictures and the peak-to-peak amplitude of the EMG recorded during the second before the stimulus onset. Two mixed design factorial ANOVAs were computed using the GROUP as between subjects factor, the VALENCE and the SIDE OF THE FACE (left vs right) as within subjects factors, and the mean changes of activity recorded in the corrugator and the zygomatic muscles respectively as dependent variables.

   Tonic responses: Tonic EMG activity was obtained as the mean values recorded during each block of pictures (10 neutral, 10 negative not related to sleep, 10 negative related to sleep, 10 positive, 10 positive related to sleep). Two mixed design factorial ANOVAs were computed using the GROUP as between subjects factor, the VALENCE and the SIDE OF THE FACE as within subjects factors, and the mean activity recorded over the corrugator and the zygomatic muscles respectively during each block as dependent variables.
3) HR and CVT. Mean heart rate (HR) and mean cardiac vagal tone (CVT) were calculated for each stimulus block. With skewness and kurtosis < 1, the distributions of the means were considered normal. Data were analysed within two mixed design factorial ANOVAs using the GROUP as between subjects factor, the VALENCE as within subject factor and respectively the mean HR and CVT as dependent variables.
4) Subjective measures. Two mixed design factorial ANOVAs GROUP $\times$ VALENCE were computed using the GROUP (PPI vs GS) as between subjects factor, the VALENCE (neutral vs negative not related to sleep vs negative related to sleep vs positive not related to sleep vs positive related to sleep) as within subjects factor, and the subjective valence ratings (pleasant vs unpleasant) and arousal ratings (low vs high) respectively as dependent variables.
5) Cultural differences. As our sample was recruited half in Scotland and half in Italy, we consider possible cultural differences in the emotional responses. Descriptive analyses and the same mixed design factorial ANOVAs analyses aforementioned were repeated considering as between subjects factor the PLACE (Glasgow vs Rome).

When a significant principal effect or interaction was found, specific hypothesis were tested through planned comparisons.

Findings

Means $\pm$ standard deviations are reported in the text in brackets.

1) Description of the sample. A summary of the descriptive results is provided in Table 1.

The PPI group included a marginally significant higher number of females, reported higher scores on the STAI-T and had higher scores on the ISI compared to the GS group. There was no difference between the two groups due to the time of day of recording ($X^2_{(2, N=39)} = 1.48$, $p = 0.48$). Nine PPI and 8 GS were recorded in the morning (between 9:00 and 12:30), 9 PPI and 5 GS at lunch time (between 12:30 and 15:30) and 3 PPI and 5 GS in the afternoon (between 15:30 and 18:00). Two good sleepers and 4 people with insomnia reported scores higher than 7 on the KSS, however no difference between the groups was found, as shown in Table 1.

2) Facial electromyography (EMG). Results are summarized in Table 3.

No significant effects were evidenced for phasic responses of the corrugator muscle. Phasic responses of the zygomatic muscle showed a significant main effect for the factor VALENCE. Planned comparisons evidenced that both groups presented heightened activity in response to positive stimuli (related and not related to sleep) compared to negative stimuli (related and not related to sleep) (positive not related to sleep: -1.17 ± 1.77; sleep-related positive: -1.61 ± 2.03; negative not related to sleep: -2.01 ± 1.49; sleep-related negative: -1.98, $p = 0.03$; $F_{(1,21)} = 12.17$, $p = 0.002$). Additionally all participants presented increased zygomatic activity in response to positive stimuli related to sleep compared to negative stimuli related to sleep ($F_{(1,21)} = 7.60$, $p = 0.01$). Finally, all participants displayed different responses to neutral stimuli (-1.88 ± 2.03) as compared with all the emotional stimuli ($F_{(1,21)} = 5.53$, $p = 0.03$).

[4] Item numbers: *Positive not related to sleep* (from STAI): 2, 5, 10, 15, 16, 20, 21, 23, 26, 27, 30; *Negative not related to sleep* (from FSS): 5, 22, 41, 47, 48, 51, 56, 66, 61, 67; *Positive and Negative sleep-related* (from GCT): 4, 6, 8, 9, 10, 13, 15, 18, 20, 24, 26.

Table 2
Results related to facial EMG data.

SOURCE OF VARIATION	Corrugator							Zygomatic						
	df	Sum of squares	df error	Mean square	*F*	*p*	Observed Power	df	Sum of squares	df error	Mean square	*F*	*p*	Observed power
Phasic responses														
GROUP	1	5.179	17	5.179	0.520	0.48	0.11	1	34.626	21	14.876	2.327	0.14	0.142
SIDE	1	0.016	17	0.016	0.003	0.96	0.05	1	13.712	21	13.712	1.469	0.24	0.212
VALENCE	4	9.548	68	2.387	1.521	0.21	0.45	4	22.359	84	5.590	4.197	0.004	0.909
GROUP × SIDE	1	2.505	17	2.505	0.490	0.49	0.10	1	0.905	21	0.905	0.097	0.76	0.060
GROUP × VALENCE	4	5.550	68	1.387	0.884	0.48	0.27	4	4.808	84	1.202	0.903	0.76	0.275
SIDE × VALENCE	4	3.131	68	0.783	1.479	0.22	0.44	4	3.426	84	0.856	1.539	0.20	0.457
GROUP × SIDE × VALENCE	4	2.382	68	0.596	1.125	0.35	0.34	4	1.114	84	0.278	0.500	0.74	0.164
Tonic responses														
GROUP	1	1.551	36	1.551	0.117	0.73	0.06	1	51.143	36	51.143	3.758	0.06	0.471
SIDE	1	2.703	36	2.703	0.590	0.45	0.12	1	21.330	36	21.330	3.388	0.74	0.433
VALENCE	4	0.287	144	0.072	0.631	0.64	0.20	4	3.976	144	0.994	7.562	<0.001	0.996
GROUP × SIDE	1	0.014	36	0.014	0.003	0.96	0.05	1	0.116	36	0.116	0.018	0.89	0.052
GROUP × VALENCE	4	1.137	144	0.284	2.502	0.04	0.70	4	0.297	144	0.074	0.565	0.69	0.185
SIDE × VALENCE	4	0.272	144	0.068	1.027	0.40	0.32	4	0.009	144	0.002	0.048	0.10	0.059
GROUP × SIDE × VALENCE	4	0.376	144	0.094	1.422	0.23	0.43	4	0.355	144	0.089	1.828	0.13	0.545

When tonic responses of the corrugator muscle were analysed, a significant interaction GROUP × VALENCE was found. Planned comparisons showed that GS presented similar responses to all types of stimuli (neutral: 0.03 ± 1.39; positive not related to sleep: −0.09 ± 1.38; negative not related to sleep: 0.02 ± 1.36; sleep-related positive: 0.10 ± 1.37; sleep-related negative: 0.0005 ± 1.36; $F_{(1,36)} = 2.30$ $p = 0.14$). In contrast, corrugator activity in PPI decreases significantly in response to positive stimuli related to sleep (−0.239 ± 1.325), compared to the activity produced in response to all the other types of stimuli (neutral: −0.0685 ± 1.34; positive not related to sleep: −0.099 ± 1.40; negative not related to sleep: −0.0645 ± 1.37; sleep-related negative: −0.1205 ± 1.345; $F_{(1,36)} = 5.426$, $p = 0.03$). The interaction is shown in Fig. 1.

When tonic responses of the zygomatic muscle were analysed, a significant main effect of the VALENCE was found. Planned comparisons showed only that both groups displayed heightened activity in response to positive stimuli (related and not related to sleep) compared to negative stimuli (related and not related to sleep) (positive not related to sleep: 0.66 ± 1.42; sleep-related positive: 0.55 ± 1.48; negative not related to sleep: 0.41 ± 1.40; sleep-related negative: 0.37 ± 1.37; $F_{(1,36)} = 16.34$, $p < 0.001$). Additionally, a marginally significant main effect of the GROUP has to be noted, showing that PPI display enhanced reactivity to all stimuli as compared to GS.

3) HR and CVT. Results from HR and CVT responses are presented in Table 4.

No significant effects were evidenced for HR responses. When CVT was analysed, a significant main effect of the GROUP was found. People with primary insomnia (8.12 ± 2.93) displayed increased CVT in response to all stimuli compared to GS (5.027 ± 1.315).

4) Subjective measures. The results from the subjective ratings of VALENCE and AROUSAL are presented in Table 2.

Valence: Results showed a significant main effect of the VALENCE and a significant interaction GROUP × VALENCE. Planned comparisons showed that both groups rated positive stimuli (related and not related to sleep) as more pleasant as compared with negative stimuli (related and not related to sleep) (positive not related to sleep: 7.83 ± 1.35, sleep-related positive: 7.15 ± 1.48, negative stimuli: 3.13 ± 1.47, sleep-related negative: 4.44 ± 1.65; $F_{(1,37)} = 181.03$, $p < 0.001$). Moreover, both groups rated positive stimuli related to sleep as more pleasant compared to negative stimuli related to sleep ($F_{(1,37)} = 79.37$, $p < 0.001$). It has to be noted that neutral stimuli (6.42 ± 1.50) were differently rated by all participants as compared with all the emotional stimuli ($F_{(1,37)} = 12.17$, $p = 0.001$).
With respect to the interaction GROUP × VALENCE, planned comparison evidenced that GS rated negative stimuli not related to sleep (3.17 ± 1.42) as more unpleasant compared to negative stimuli related to sleep (5.11 ± 1.23; $F_{(1,37)} = 24.30$, $p < 0.001$). In contrast, PPI rated negative pictures related to sleep (3.76 ± 1.73) as unpleasant as negative pictures not related to sleep (3.10 ± 1.55; $F_{(1,37)} = 3.33$, $p = 0.08$).
Arousal: The mixed design factorial ANOVA showed a main effect of the GROUP, and a main effect of the VALENCE. Additionally, it has to be noted that a marginally significant interaction GROUP × VALENCE was detected. People with primary insomnia (4.49 ± 1.93) rated all stimuli as more arousing compared to GS (2.788 ± 1.40) regardless to the valence. Planned comparisons evidenced that both groups rated negative stimuli (related and not related to sleep) as more arousing than positive stimuli (related and not related

Table 3
HR and CVT.

SOURCE OF VARIATION	HR							CVT						
	df	Sum of squares	df error	Mean square	*F*	*p*	Observed power	df	Sum of squares	df error	Mean square	*F*	*p*	Observed power
GROUP	1	82.087	21	82.087	0.274	0.60	0.08	1	228.49	20	228.49	7.538	0.01	0.74
VALENCE	4	16.464	84	4.116	1.011	0.41	0.31	4	5.864	80	1.466	2.255	0.07	0.64
GROUP × VALENCE	4	19.265	84	4.816	1.182	0.32	0.36	4	0.608	80	0.152	0.234	0.91	0.10

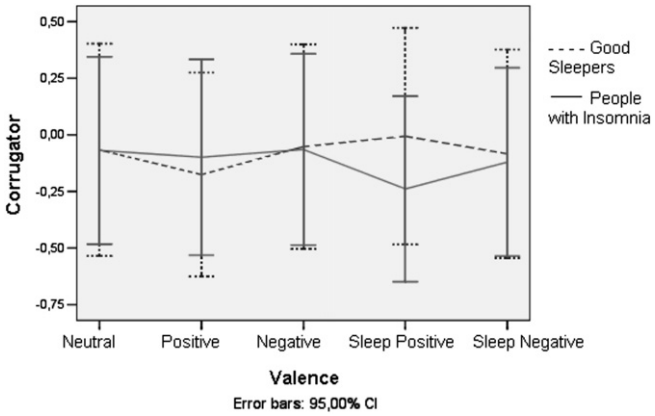


Fig. 1. Tonic responses for corrugator muscle: interaction GROUP × VALENCE.

to sleep) (negative not related to sleep: 4.949 ± 1.89; sleep-related negative: 4.462 ± 2.08, positive not related to sleep: 3.359 ± 2.12, sleep-related positive: 2.564 ± 1.54; $F_{(1,37)} = 34.09$, $p < 0.001$). Moreover, both groups rated negative stimuli related to sleep as more arousing compared to positive stimuli related to sleep ($F_{(1,37)} = 30.33$, $p < 0.001$). Finally neutral stimuli (3.10 ± 1.99) were differently rated by both PPI and GS as compared with all the emotional stimuli ($F_{(1,37)} = 10.88$, $p = 0.002$). With respect to the interaction GROUP × VALENCE, planned comparison showed that GS rated negative stimuli not related to sleep as more arousing as compared with negative stimuli related to sleep ($F_{(1,37)} = 4.04$, $p = 0.05$). They also rated as equally arousing negative and positive sleep-related stimuli ($F_{(1,37)} = 3.40$, $p = 0.08$). In contrast, PPI rated negative stimuli not related to sleep as arousing as negative stimuli related to sleep ($F_{(1,37)} = 0.01$, $p = 0.92$). Moreover, PPI rated negative stimuli related to sleep as more arousing than positive stimuli relative to sleep ($F_{(1,37)} = 37.61$, $p < 0.001$).

5) Cultural differences.

We found no statistical significant differences between the group recruited in Rome and the group recruited in Glasgow with respect to gender ($X^2_{(1,N=39)} = 0.51$, $p = 0.38$) and with respect to age (Rome: 23.05 ± 3.14; Glasgow: 21.68 ± 2.67; $F_{(1,37)} = 2.14$, $p = 0.15$).

The results from the mixed design factorial ANOVAs applied considering the PLACE (Glasgow vs Rome) as between subjects factor evidenced the following:

- Main effect of the PLACE: The sample recruited in Rome, as compared to the sample recruited in Glasgow, presented enhanced tonic EMG activity in response to all the stimuli both in the corrugator (Rome: 0.30 ± 1.33; Glasgow: −0.52 ± 0.99; $F_{(1,37)} = 5.56$, $p = 0.02$) and in the zygomatic (Rome: 1.08 ± 1.42; Glasgow: −0.09 ± 1.02; $F_{(1,37)} = 11.78$, $p = 0.001$) muscles.
- Interaction PLACE × VALENCE: A significant interaction was detected with respect to the subjective ratings of the arousal level of the stimuli ($F_{(4,48)} = 2.60$, $p = 0.04$). Planned comparisons showed that the sample recruited in Rome (4.10 ± 2.25), compared to the sample recruited in Glasgow (2.58 ± 1.71), rated the positive stimuli not related to sleep as more arousing ($F_{(1,37)} = 5.62$, $p = 0.02$).

No other statistically significant differences were observed.

Discussion

The results of our study suggest that people with primary insomnia, as compared to good sleepers, present: increased physiological craving response for positive sleep-related conditions, enhanced physiological hyper-arousabilty in response to all stimuli, and enhanced subjective hyper-arousability for negative sleep-related conditions. In fact, we found an inhibition of the tonic activity of the corrugator muscle only in people with primary insomnia in response to sleep-related positive stimuli as compared to the other blocks of stimuli. Consistently with the attention–intention–effort theory proposed by Espie et al. (2006), people with insomnia perceive sleep requirement/deficit and become increasingly motivated by sleep. The perceived inability to sleep would be experienced as a significant threat, and consequently sleep-related stimuli would enhance the arousal levels necessary for monitoring the threat. On the other hand, and not mutually exclusive with the response to the threat, people with insomnia could show specific sensitivity for good sleep which represent a desired condition for them. The desire for sleep of good quality may become a "craving" (e.g. Espie et al., 2006). Consistently with the craving response, sleep-related positive stimuli elicited also greater responses in the zygomatic muscle in people with primary insomnia compared to sleep-related negative responses, thus, suggesting that these stimuli induce a positive/craving response in people with insomnia. Additionally, the result related to the zygomatic muscle was found also in good sleepers, indicating that sleep of good quality is a pleasant experience for everybody.

Results related to our physiological measures showed no significant effects with respect to responses to negative stimuli (both sleep-related stimuli and not). It is possible that, although the IAPS was developed following the dimensional approach to emotions which do not refer to discrete emotions, certain specific content effects can influence the emotional response. As Hamm, Schupp, and Weike (2003) note, pronounced corrugator activity is related mainly to disgust-eliciting stimuli. Indeed, the IAPS stimuli generally used for inducing negative emotions associated to an increase in the activity of the corrugator muscle are rated as highly arousing and induce predominantly the emotion of disgust (e.g. photographs of amputations, tumours, skin infections, etc.). In

Table 4
Subjective responses.

SOURCE OF VARIATION	Valence ratings							Arousal ratings						
	df	Sum of squares	df error	Mean square	*F*	*p*	Observed power	df	Sum of squares	df error	Mean square	*F*	*p*	Observed power
GROUP	1	7.329	37	7.329	1.962	0.17	0.28	1	139.531	37	139.531	22.251	<0.001	1.00
VALENCE	4	593.784	148	148.446	86.256	<0.001	1.00	4	143.838	148	35.960	16.716	<0.001	1.00
GROUP × VALENCE	4	22.748	148	5.687	3.305	0.01	0.83	4	19.961	148	4.990	2.320	0.06	0.66

order to control the dimension of arousal and to match the negative stimuli from the IAPS with the negative sleep-related stimuli, these high-arousal and disgust-eliciting pictures were not used in this study. It is also possible that the presence of the experimenter during the task influenced the outcome. Although the experimenter sat behind the subject, some data suggests that observations by others can reduce facial activity (Fridlund & Cacioppo, 1986). This appears to be especially important when the emotions expressed are not desirable emotions (i.e. negative). Moreover, some studies have used relaxation techniques before the presentation of the stimuli to the participants (e.g. Fridlund & Cacioppo, 1986). These techniques could induce a lower level of activity in the muscular activity at the beginning of the trial, which could facilitate the detection of an effect. As these factors may represent confounds within this experiment, they should be taken into consideration when planning future studies.

In our study, there was no relation between primary insomnia and impairment in the emotional processing of pleasant stimuli, as was found in dysphoric individuals (Sloan, Bradley, Dimoulas, & Lang, 2002). The positive stimuli elicited greater zygomatic activity in both groups compared to unpleasant stimuli. Thus, this result suggests that decreased emotional reactivity to positive stimuli is a specific feature of depression.

Our findings show that, compared to good sleepers, people with primary insomnia respond with increased levels of arousal to all stimuli, consistently with the hyper-arousal (e.g. Riemann et al., 2010) and the hyper-arousability hypothesis (Lundh & Broman, 2000) for insomnia. This result was evidenced by enhanced cardiac vagal tone and zygomatic activity in responses to all blocks of pictures by people with primary insomnia compared to good sleepers. Although the effect for the zygomatic muscle was only marginally significant, the observed power of 0.47 is below the 0.80 level, which suggest that the result could be significant if the number of participants was increased.

In addition to these results, considering the subjective ratings of the valence and of the arousal of the stimuli, in our sample only people with primary insomnia rated sleep-negative stimuli as more arousing compared to sleep-positive stimuli. The levels of arousal of the sleep-positive stimuli, instead, were rated similarly by the two groups. This result could suggest that for people with primary insomnia it is not just the sleep-related stimulus in general (e.g. the bed) to be associated with high arousal, but only sleep-related stimuli associated with the experience of wakefulness during the night. People with primary insomnia subjectively rated negative and sleep-negative stimuli as equally unpleasant, while good sleepers rated negative stimuli as the most unpleasant compared to all other types of stimuli. This suggests that good sleepers do not view symptoms of insomnia in as negative light as those individuals with primary insomnia.

In addition to the results related to the main hypothesis of this study, our findings evidenced cultural differences both with respect to physiological and subjective measures of emotional responses. Our Italian sample responded with increased physiological activity to all stimuli and rated the positive stimuli not related to sleep as more arousing compared to the sample recruited in Glasgow. Further investigation is needed to deepen the role of different cultural and social norms in the affective experience. The cultural component of the emotional responses should be taken in consideration when investigating emotions in different contexts.

As expected, people with primary insomnia reported higher trait levels of anxiety on the STAI-T compared to good sleepers. Nonetheless, all participants were under the clinical threshold for clinical anxiety (Sesti, 2000) and reported no psychiatric condition at the clinical interview. Additionally, the two groups did not differ with respect to the level of subjective sleepiness before the recording. Furthermore, groups did not differ with respect to medications use. Thus all the differences in the emotional responses to different conditions seen in the present study could not reasonably be attributed to the presence of a psychiatric or psychological disease or to excessive subjective sleepiness.

Some points of strength and some limitations of the study have to be addressed. The use of validated sleep-related material allowed us to evaluate specific emotional responses in people with insomnia. In addition, this study is the first one to differentiate negative and positive stimuli related to sleep in insomnia research. Furthermore, we used facial EMG which has the advantage of detecting muscular activity that would not be observable with the naked eye (Ekman & Rosenberg, 2005). However it has the disadvantage of being obstructive and intrusive. Thus, participants are aware that their face movements are being measured which, of course, is different from the natural conditions in which emotions are generally expressed. Results could have been influenced by gender differences between the two groups, as the group with insomnia included more women compared to the control group. Women are generally more facially expressive and demonstrate greater stress induced changes in heart rate than men (Heponiemi, Ravaja, Elovainio, Näätänen, & Keltikangas-Järvinen, 2006).

While none of the stimuli had a high level of arousal (all values < 6), there were still differences in the arousal levels between the types of stimuli: negative stimuli not related to sleep had the highest levels of arousal compared to all other types of stimuli, and sleep-related positive stimuli had the lowest levels of arousal. The arousal dimension of the stimuli should be more closely matched in future studies. Moreover, the ratings of the sleep-related positive stimuli could have been also influenced by intrinsic aspects of the SAM. In fact, the figure which represents the low extreme of the arousal dimension is defined as a “relaxed, sleepy figure”. Indeed, both good sleepers and people with primary insomnia rated the sleep-positive stimuli with low levels of arousal.

This is the first study evaluating physiological responses to emotional stimuli considering the valence dimension of emotions in insomnia. For this reason, conclusions about the relevance of the processing of the emotional valence of symptom-related stimuli in insomnia have to be drawn carefully, and more investigations are needed. Since responses in the corrugator muscle did not show sensitivity to symptom-relevant-negative stimuli, further investigations are needed using different physiological techniques and measures (as for example, neuroimaging studies). Moreover, new studies could use dynamic stimuli (i.e. films), instead of static stimuli (i.e. pictures) to contrast the reduction of spontaneous facial activity due to intrinsic proprieties of the EMG recordings. Additionally, this study was conducted on an undergraduate sample. The findings from this sample may, therefore, not generalize to other populations, such as children, adolescents, elderly adults, patients. Indeed, the findings need to be replicated in a treatment seeking sample. Furthermore, investigations are needed to confirm the presence of a craving effect for positive sleep-related stimuli in people with insomnia. Indeed, an excessive focus on the beneficial effects of a good night of sleep would be consequently related to worries and negative thoughts about the consequences of not sleeping well. If our results will be confirmed by further research, they could suggest that psychological treatment of insomnia can benefit from the inclusion of strategies dealing with emotional processes associated to sleep disruption. Since the occurrence of isolated insomnia symptoms for a period of more than two weeks predicts increased risk of developing depression within the following three years (Riemann & Voderholzer, 2003), the understanding of the specific nature and mechanisms underlying affective regulation in primary

insomnia can help in improving the quality of treatment and in delineating preventative programs for possible consequent mood disorders.

Acknowledgments

We are in debt to Prof. Dr. Riemann (Department of Psychiatry & Psychotherapy, University of Freiburg Medical Center) for his helpful comments.

We would thanks Prof. Ron Baxendale (Faculty of Biomedical & Life Sciences of the University of Glasgow) for help and for allowing us to carry out preliminary tests of the study in his laboratory.

References

Akerstedt, T. R., & Gillberg, M. (1990). Subjective and objective sleepiness in the active individual. *International Journal of Neuroscience, 52*, 29–37.

American Academy of Sleep Medicine. (2005). *International classification of sleep disorders* (2nd ed.).. In: *Diagnostic and coding manual* Westchester, Illinois: American Academy of Sleep Medicine (AASM).

American Psychiatric Association. (2002). *Diagnostic and statistical manual of mental disorders*. text revision (DSM-IV-TR) (4th ed.). Washington, D.C.: American Psychiatric Association (APA).

Bastièn, C. H., Valliéres, A., & Morin, C. M. (2001). Validation of the Insomnia Severity Index as an outcome measure for insomnia research. *Sleep Medicine, 2*, 297–307.

Bradley, M. M. (2000). Emotion and motivation. In J. T. Cacioppo, L. G. Tassinary, & G. G. Bernston (Eds.), *Handbook of psychophysiology* (2nd ed.). Cambridge, UK: Cambridge University Press.

Bradley, M. M., & Lang, P. J. (1994). Measuring emotion: the self-assessment manikin and the semantic differential. *Journal of Behavior Therapy and Experimental Psychiatry, 25*, 49–59.

Chang, P. P., Ford, D. E., Mead, L. A., Cooper-Patrick, L., & Klag, M. J. (1997). Insomnia in youngmen and subsequent depression: the Johns Hopkins Precursors Study. *American Journal of Epidemiology, 146*, 105–114.

Edinger, J. D., Bonnet, M. H., Bootzin, R. R., Doghramji, K., Dorsey, C. M., Espie, C. A., et al. (2004). Derivation of research diagnostic criteria for insomnia: report of an American Academy of Sleep Medicine Work Group. *Sleep, 27*, 1567–1596.

Ekman, P., & Rosenberg, E. L. (2005). *What the face reveals: Basic and applied studies of spontaneous expression using the Facial Action Coding System (FACS)* (2nd ed.). New York: Oxford University Press.

Espie, C. A. (1991). *The psychological treatment of insomnia*. Chichester: John Wiley & Sons Ltd.

Espie, C. A. (2002). Insomnia: conceptual issues in the development, persistence and treatment of sleep disorders in adults. *Annual Review of Psychology, 53*, 215–243.

Espie, C. A. (2006). *Overcoming insomnia and sleep problems: A self-help guide using cognitive behavioral techniques*. London: Constable & Robinson Ltd.

Espie, C. A., Broomfield, N. M., MacMahon, K. M. A., MacPhee, L. M., & Taylor, L. M. (2006). The attention–intention–effort pathway in the development of psycho-physiologic insomnia: a theoretical review. *Sleep Medicine Reviews, 10*, 215–245.

Fazio, R. H., Sanbonmatsu, D. M., Powell, M. C., & Karders, F. R. (1986). On the automatic activation of attitudes. *Journal of Personality and Social Psychology, 50*, 229–238.

Ford, D. E., & Kamerow, D. B. (1989). Epidemiologic study of sleep disturbances and psychiatric disorders. An opportunity for prevention? *Journal of the American Medical Association, 262*, 1479–1484.

Franzen, P. L., Buysse, D. J., Dahl, R. E., Thompson, W., & Siegle, G. J. (2009). Sleep deprivation alters pupillary reactivity to emotional stimuli in healthy young adults. *Biological Psychology, 80*, 300–305.

Franzen, P. L., Siegle, G. J., & Buysse, D. J. (2008). Relationships between affect, vigilance, and sleepiness following sleep deprivation. *Journal of Sleep Research, 17*, 34–41.

Fridlund, A. J., & Cacioppo, J. T. (1986). Guidelines for human electromyographic research. *Psychophysiology, 23*, 567–589.

Hamm, A. O., Schupp, H. T., & Weike, A. I. (2003). Motivational organization of emotions: autonomic changes, cortical responses, and reflex modulation. In R. J. Davidson, K. R. Scherer, & H. H. Goldsmith (Eds.), *Handbook of affective sciences*. Oxford: Oxford University Press.

Harvey, A. G. (2002). A cognitive model of insomnia. *Behaviour Research and Therapy, 4*, 869–893.

Harvey, K. J., & Espie, C. A. (2004). Development and preliminary validation of the Glasgow Content of Thoughts Inventory (GCTI): a new measure for the assessment of pre-sleep cognitive activity. *British Journal of Clinical Psychology, 43*, 409–420.

Heponiemi, T., Ravaja, N., Elovainio, M., Näätänen, P., & Keltikangas-Järvinen, L. (2006). Experiencing positive affect and negative affect during stress: relationships to cardiac reactivity and to facial expressions. *Scandinavian Journal of Psychology, 47*, 327–337.

Horne, J. A., & Ostberg, O. (1976). A self-assessment questionnaire to determine morningness–eveningness in human circadian rhythms. *International Journal of Chronobiology, 4*, 97–110.

Julu, P. O. O. (1992). A linear scale for measuring vagal tone in man. *Journal of Autonomic Pharmacology, 12*, 109–115.

Lang, P. J. (2002). The dimensional organization of emotion. In E. E. Smith, S. Nolen-Hoeksema, B. L. Fredrickson, & G. Loftus (Eds.), *Atkinson & Hilgard's introduction to psychology* (14th ed.). Wadsworth Thompson Learning.

Lang, P. J., Bradley, M. M., & Cuthbert, B. N. (2001). *International Affective Picture System (IAPS): Affective ratings and instruction manual*. Technical Report A-6. Gainesville, Florida: University of Florida.

Larsen, J. T., Norris, C. J., & Cacioppo, J. T. (2003). Effects of positive and negative affect over electromyographic activity over zygomatic major and corrugator supercilii. *Psychophysiology, 40*, 776–785.

LeBlanc, M., Beaulieu-Bonneau, S., Mérette, C., Savard, J., Ivers, H., & Morin, C. M. (2007). Psychological and health-related quality of life factors associated with insomnia in a population based sample. *Journal of Psychosomatic Research, 63*, 157–166.

Lichstein, K. L., Durrence, H. H., Taylor, D. J., Bush, A. J., & Riedel, B. W. (2003). Quantitative criteria for insomnia. *Behaviour Research and Therapy, 41*, 427–445.

Little, C. J., Julu, P. O., Hansen, S., & Reid, S. W. (1999). Real-time measurement of cardiac vagal tone in conscious dogs. *American Journal of Physiology, 276*, 758–765.

Lundh, L. G., & Broman, J. E. (2000). Insomnia as an interaction between sleep-interfering and sleep-interpreting processes. *Journal of Psychosomatic Research, 49*, 299–310.

McCrae, C. S., McNamara, J. P., Rowe, M. A., Dzierzewski, J. M., Dirk, J., Marsiske, M., et al. (2008). Sleep and affect in older adults: using multilevel modeling to examine daily associations. *Journal of Sleep Research, 17*, 42–53.

Morin, C. M. (1993). *Insomnia, psychological assessment and management*. New York: The Guilford Press.

Movius, H. L., & Allen, J. J. (2005). Cardiac Vagal Tone, defensiveness, and motivational style. *Biological Psychology, 68*, 147–162.

Norlander, T., Johansson, Å., & BoodÅ, S. (2005). The affective personality: its relation to quality of sleep, well-being and stress. *Social Behavior & Personality, 33*, 709–722.

Oldfield, R. C. (1971). The assessment and analysis of handedness: the Edinburgh Inventory. *Neuropsychologia, 9*, 97–113.

Perlis, M. L., Smith, L. J., Lyness, J. M., Matteson, S. R., Pigeon, W. R., Jungquist, C. R., et al. (2006). Insomnia as a risk factor for onset of depression in elderly. *Behavioral Sleep Medicine, 4*, 104–113.

Perlis, M. L., Giles, D. E., Mendelson, W. B., Bootzin, R. R., & Wyatt, J. K. (1997). Psychophysiological insomnia: the behavioural model and a neurocognitive perspective. *Journal of Sleep Research, 6*, 179–188.

Riemann, D. (2007). Insomnia and comorbid psychiatric disorders. *Sleep Medicine, 8* (Suppl. 4), S15–S20.

Riemann, D., Spiegelhalder, K., Feige, B., Voderholzer, U., Berger, M., Perlis, M., et al. (2010). The hyperarousal model of insomnia: a review of the concept and its evidence. *Sleep Medicine Reviews, 14*, 19–31.

Riemann, D., & Voderholzer, U. (2003). Primary Insomnia: a risk factor to develop depression? *Journal of Affective Disorders, 76*, 255–259.

Scott, B. A., & Judge, T. A. (2006). Insomnia, emotions and job satisfaction: a multilevel study. *Journal of Management, 32*, 622–645.

Sesti, A. (2000). State-Trait Anxiety Inventory (STAI) in medication clinical traits. *Quality of Life Newsletter, 25*, 15–16.

Sloan, D. M., Bradley, M. M., Dimoulas, E., & Lang, P. J. (2002). Looking at facial expressions: dysphoria and facial EMG. *Biological Psychology, 60*, 79–90.

Spielberger, C. D., Gorsuch, R. L., & Lushenne, R. E. (1970). *STAI manual for the state-trait anxiety inventory*. Palo Alto, California: Consulting Psychologists Press.

Van Egeren, L., Hayness, S. N., Franzen, M., & Hamilton, J. (1983). Presleep cognitions and attributions in sleep onset insomnia. *Journal of Behavioral Medicine, 6*, 217–232.

Violani, C., Devoto, A., Lucidi, F., Lombardo, C., & Russo, P. M. (2004). Validity of a short insomnia questionnaire: the SDQ. *Brain Research Bulletin, 63*, 415–421.

Wicklow, A., & Espie, C. A. (2000). Intrusive thoughts and their relationship to actigraphic measurement of sleep: towards a cognitive model of insomnia. *Behaviour Research and Therapy, 38*, 679–693.

Witvliet, C. V. O., & Vrana, S. R. (1995). Psychophysiological responses as indices of affective dimensions. *Psychophysiology, 32*, 436–443.

Wolpe, J., & Lang, P. J. (1964). A fear survey schedule for use in behaviour therapy. *Behaviour Research and Therapy, 2*, 27–30.

Yoo, S. S., Gujarm, N., Hu, P., Jolesz, F. A., & Walker, M. P. (2007). The human emotional brain without sleep: a prefrontal amygdala disconnect. *Current Biology, 17*, 877–878.

Zhou, R., & Hu, S. (2006). Study of posed emotion in facial EMG asymmetry. *Perceptual and Motor Skills, 102*, 430–434.

http://dx.doi.org/10.5665/sleep.1702

Comparative Investigation of the Psychophysiologic and Idiopathic Insomnia Disorder Phenotypes: Psychologic Characteristics, Patients' Perspectives, and Implications for Clinical Management

Colin A. Espie, PhD[1,2]; Laura M. Barrie, DClinPsy[1,3]; Grant S. Forgan, DClinPsy[1,3]

[1]University of Glasgow Sleep Centre, Glasgow, Scotland; [2]Institute of Neuroscience and Psychology, [3]Section of Psychological Medicine, University of Glasgow, Glasgow, Scotland

Study Objectives: Insomnia is a common disorder, yet its proposed behavioral phenotypes are seldom differentiated. Two consecutive studies were designed to investigate psychologic characteristics and treatment preferences of people with idiopathic insomnia (IdI) relative to psychophysiologic insomnia (PI).

Design: Cross-sectional, two-group comparison studies.

Setting: Specialized sleep research center.

Participants: 40 participants (29 female, mean age 46 yr) participated in study 1. An additional cohort of 61 adults (48 female, mean age 37 yr) participated in study 2. In total, samples comprised 51 participants with PI and 50 with IdI. All participants met diagnostic criteria for their respective insomnia phenotype.

Interventions: N/A

Measurements and Results: Study 1 investigated sensitivity to arousal conditioning and sleep effort using self-report measures. Consistent with a model of conditioned arousal, participants with PI exhibited greater behavioral inhibition, i.e., sensitivity to threat and higher levels of sleep preoccupation. Study 2 investigated illness perceptions and cognitions and coping styles using self-report scales, and explored treatment acceptability based on the evaluation of 3 therapeutic scenarios. Results lend support to the hypothesis that IdI is considered somewhat more permanent than PI. Behavioral intervention was preferred to pharmacotherapy by both groups, and an acceptance treatment was considered more favorably by IdI study participants than by those with PI.

Conclusions: Many similarities between IdI and PI were observed across psychologic measures, and both groups exhibited a preference for behavioral treatment. However, their distinctive characteristics appear to suggest that an acceptance-based therapy may also be appropriate for some people with IdI.

Keywords: Insomnia, phenotype, psychology, cognitive behavior therapy, acceptance, cognition, arousal, treatment

Citation: Espie CA; Barrie LM; Forgan GS. Comparative investigation of the psychophysiologic and idiopathic insomnia disorder phenotypes: psychologic characteristics, patients' perspectives, and implications for clinical management. *SLEEP* 2012;35(3):385-393.

INTRODUCTION

Insomnia is a prevalent disorder that is challenging to manage,[1,2] partly because of its inherent heterogeneity and the limited data available on the benefits (or disadvantages) of matching treatment to clinical presentation.[3,4] The International Classification of Sleep Disorders, 2nd Edition (ICSD-2) provides diagnostic criteria for several primary insomnia subtypes, including psychophysiologic insomnia (PI) and idiopathic insomnia (IdI).[5] The goal of the current study was to compare the way that adults with each of these subtypes perceive their insomnia, and to consider any resultant implications for clinical management.

According to ICSD-2, PI develops in adulthood, may be linked to identifiable precipitating events and/or stressors, and comprises both psychologic and physiologic features such as conditioned arousal, sleep-incompatible behavior, sleep preoccupation, and excessive focus on and anxiety about sleep. In contrast, IdI is a lifelong complaint with a chronic course and few periods of sustained remission. It has been suggested that IdI may be resistant to treatment.[6] There is a limited amount of research literature on IdI, perhaps because the condition is relatively uncommon and affects fewer than 10% of those presenting with insomnia complaints.[1] Moreover, conceptualizations of insomnia development and maintenance seem to fit more closely with PI than they do with IdI.[7-11]

IdI is partly defined by the absence of precipitating and maintaining factors, and patients appear to exhibit only minor psychologic abnormalities. The use of denial and repression as coping strategies has been suggested, and there may be an association with neurodevelopmental disorders.[12,13] Higher levels of "arousability" have been reported in patients with IdI,[6] and it may be that IdI represents the extreme of an insomnia continuum, where there is less of a psychologic and more of a physiologic characteristic. Somatic hyperarousal may be present in all insomnias,[14,15] but may be a particular feature of IdI. In this regard it should be taken into consideration that conditioning of arousal also may be a factor in IdI, although this possibility has not been investigated. On the other hand, some research has found no major differences between IdI and PI on either polysomnography (PSG) recordings or psychologic measures.[16] This finding is consistent with the Diagnostic and Statistical Manual of Mental Disorders, 4th Edition (DSM-IV) Work Group conclusion that, although the PI and IdI classifica-

Submitted for publication May, 2011
Submitted in final revised form September, 2011
Accepted for publication September, 2011
Address correspondence to: Colin A. Espie, PhD, University of Glasgow Sleep Centre, Sackler Institute of Psychobiological Research and Institute of Neuroscience and Psychology, College of Medical, Veterinary and Life Sciences, University of Glasgow, G51 4TF, Scotland; Tel: +44 (0)141 232 7860; Fax: +44 (0)141 232 7697; E-mail: Colin.Espie@glasgow.ac.uk

tions offered some clinical value, there was limited empirical support to propose their distinction.[17] Perhaps because of the relative paucity of additional studies on idiopathic insomnia in the period between major editions, the DSM-5 committee appears to be taking a similar view in classifying a single insomnia disorder without subtypes being specified. (For further information see: http://www.dsm5.org/ProposedRevisions/Pages/proposedrevision.aspx?rid=65).

Clearly more research is warranted to better understand why PI and IdI appear to be (at least) clinically distinctive. The purpose of this study, therefore, was to compare how people meeting diagnostic criteria for PI and IdI conceptualize their own sleep difficulties. The study objective was to investigate similarities, and potential differences, in patients' personal perspectives, that might inform how we should approach management. Here we report two comparative studies, each having a separate clinically defined IdI sample, with its own PI comparison group. We first present the methods and results of each study, followed by an integrated discussion.

STUDY 1: COMPARATIVE INVESTIGATION OF AROUSAL CONDITIONING AND SLEEP EFFORT IN PI AND IDI

Methods

Participants

Eligible participants (18-65 yr) were recruited from the University of Glasgow Sleep Centre (UGSC) outpatient clinics and through local media advertisement, with most participants (approximately 75%) located via the media source. Rigorous clinical inclusion criteria for PI and IdI applied to all potential participants were as follows:

1. Initial telephone screening using the UGSC screening interview.
2. In-person, clinical evaluation against generic DSM-IV and specific ICSD-2 (PI and IdI) insomnia criteria, including detailed sleep history review.
3. More specifically, PI participants had to have developed persistent insomnia during adulthood, with no prior history of sleep disturbance; IdI participants had to have persistent and unremitting sleep problems since childhood.
4. Evidence of a current complaint of insomnia, verified by review of a minimum of 1 wk of sleep diary records; scores > 5 on the Pittsburgh Sleep Quality Index (PSQI)[18] and > 8 on the Insomnia Severity Index (ISI).[19]

Exclusion criteria were as follows:

1. Anyone developing insomnia between the ages of 12 and 17 yr (to reduce overlap and increase the specificity and homogeneity of the samples).
2. Any symptomatic evidence of narcolepsy, sleep apnea, restless legs syndrome/periodic limb movement disorder, circadian rhythm sleep disorder, or parasomnia using the UGSC sleep diagnostic interview.
3. Any history or present-state diagnosis of psychopathologic disorder; somatic disorder related to the onset and/or course of insomnia; evidence of substance abuse; taking medications known to influence sleep; or unstable medical condition. The Beck inventories (the Beck Depression Inventory (BDI-II)[20] and the Beck Anxiety Inventory (BAI)[21]) were used to assist evaluation of mental state and to describe and compare the final samples.

All assessment procedures were carried out by final-year, doctoral-level clinical psychology interns who were trained and supervised in behavioral sleep medicine by the first author (CAE). Consistent with current clinical assessment practice for such patients, PSG was not conducted.

Design

Cross-sectional, two-group comparison of PI and IdI.

Hypotheses

Consistent with a model of arousal conditioning, people with PI will exhibit greater sensitivity to threat (in general), and greater attention to sleep and sleep effort specifically, than will their IdI counterparts.

Measures

Behavioral Inhibition/Behavioral Activation Scale: Based on Gray's seminal work, the Behavioral Inhibition/Behavioral Activation Scales (BIS/BAS) were developed to assess a person's dispositional sensitivity to threat (behavioral inhibition: avoidance of negative outcomes) and to reward (behavioral activation: pursuit of positive outcomes).[22-24] BIS sensitivity (also known as threat sensitivity) underlies the experience of anxiety.[25,26] Consequently, we were interested in the possibility that, based on their differing historical experiences with sleep/insomnia, PI and IdI might express behavioral sensitivity in different ways.

The BIS/BAS comprises 24 items across four subscales: BIS Sensitivity (7 items: e.g., *"*criticism or scolding hurts me quite a bit"; "if I think something unpleasant is going to happen I usually get pretty "worked up*"*), and BAS Sensitivity, namely BAS Drive (4 items: e.g., *"*I go out of my way to get things I want"; "if I see a chance to get something I want I move on it right away"), BAS Fun Seeking (4 items: e.g., "I will often do things for no other reason than that they might be fun"; "I often act on the spur of the moment") and BAS Reward Responsiveness (5 items: e.g., *"*When I'm doing well at something I love to keep at it"; "it would excite me to win a contest*"*). Responses are on a 4-point scale, ranging from 1 ("very true for me") to 4 ("very false for me"), with higher scores indicating greater sensitivity. The scales have good psychometric properties in other populations. Internal consistencies in our own sample were also acceptable (Cronbach $\alpha = 0.77$, 0.76, 0.78, and 0.70, respectively).

Glasgow Sleep Effort Scale: According to ICSD-2,[5] IdI "is not associated with specific precipitating or perpetuating factors" (p. 12), whereas the related notions of sleep preoccupation and striving for sleep are regarded as "essential features" of the acquisition and maintenance of PI, i.e., *"*Learned associations are marked by overconcern with the inability to sleep. A cycle develops in which the more one strives to sleep, the more agitated one becomes, and the less able one is to fall asleep…. Concerns about sleep grow progressively over months or years as sleep gradually deteriorates until the desire to obtain good sleep becomes the person's major concern*"* (p. 6). Indeed, such selective attention to sleep is now recognized as important in several contemporary models of insomnia.[8-11] Consequently, we wanted to compare PI and IdI on this dimension.

Table 1—Demographic, mental health, and sleep characteristics of Study 1 samples; and between-group comparisons on behavioral sensitivity scales

	Psychophysiologic insomnia (n = 20)	Idiopathic insomnia (n = 20)
Demographic		
Age (yr)	49.95 (12.7)	42.15 (15.4)
Sex [n (%)]		
Female	15 (75)	14 (70)
Male	5 (25)	6 (30)
Mental health		
Beck Depression Inventory II	9.6 (6.64)	5.65 (7.24)
Beck Anxiety Inventory	12.75 (11.12)	8.2 (7.46)
Sleep		
Age of onset (yr)	34.33 (13.8)	4.7 (4.1)
Insomnia duration (yr)	16.33 (11.0)	37.15 (14.2)
Pittsburgh Sleep Quality Index	13.11 (2.95)	12.85 (3.88)
Insomnia Severity Index	17.39 (4.67)	15.25 (5.77)
Behavioral sensitivity		
Inhibition sensitivity	22.85 (2.71)	18.65 (3.77)
Activation sensitivity - drive	10.25 (2.93)	9.5 (2.52)
Activation sensitivity - fun	10.60 (2.74)	11.85 (2.83)
Activation sensitivity - reward	15.95 (2.01)	16.0 (3.03)

All data represent mean (SD), unless otherwise stated.

Table 2—Between-group comparison of Study 1 samples on items from the Glasgow Sleep Effort Scale[a]

	Psychophysiologic insomnia (n = 20)	Idiopathic insomnia (n = 20)
Glasgow sleep effort scale		
1. I put too much effort into sleeping when it should come naturally	1.10 (0.79)	0.70 (0.73)
2. I feel I should be able to control my sleep	1.15 (0.59)	0.90 (0.64)
3. I put off going to bed at night for fear of not being able to sleep	0.50 (0.61)	0.75 (0.72)
4. I worry about not sleeping if I cannot sleep	1.35 (0.59)	1.05 (0.76)
5. I am no good at sleeping	1.20 (0.69)	1.70 (0.57)
6. I get anxious about sleeping before I go to bed	0.80 (0.69)	0.30 (0.57)
7. I worry about the consequences of not sleeping	1.40 (0.68)	0.90 (0.64)

[a]All data represent mean (SD), unless otherwise stated.

The Glasgow Sleep Effort Scale (GSES) is a short (7-item) scale specifically designed to assess effortful preoccupation with sleep.[27] The GSES can be used to support a diagnosis of primary insomnia, particularly psychophysiologic insomnia,[27] and it also differentiates people with insomnia associated with mental disorder from normal sleepers.[28] A cutoff score ≥ 3 correctly identifies 93% of insomnia patients and excludes 87% of normal sleepers, representing a likelihood ratio of +7.3 (4.39-12.20) for insomnia patients relative to -0.08 (0.04-0.17) for normal sleepers. The GSES has a single factor structure with acceptable internal consistency (Cronbach $\alpha = 0.77$) and respondents score each item on a 3-point Likert scale ranging from 2 ("very much") to 0 ("not at all").[27]

Results

Sample demographics and sleep characteristics

Forty eligible participants (29 female) with a mean age of 46.1 yr (standard deviation (SD) 14.5) were recruited (Table 1). There were no differences between groups on descriptive demographic, sleep, or mental health dimensions. PSQI and ISI mean scores were indicative of clinical insomnia of moderate severity, whereas BAI and BDI scores suggest mild anxiety and minimal depressive symptoms. These results confirm that the groups were well matched

As expected, IdI and PI group allocation was supported by the IdI group reporting significantly younger mean age of insomnia onset (younger than age 5 yr compared with age 34 yr: t (38) = 9.1, P < 0.0001) and longer insomnia duration (37 yr compared with 16 yr: t (38) = 5.01, P < 0.001).

Behavioral sensitivity

Table 1 also summarizes between-group comparisons on the BIS/BAS scales. As can be seen, the PI group scored higher than the IdI group on the BIS scale (t (38) = 4.04, P < 0.001) indicating higher levels of threat sensitivity in PI. Given that historical differences are crucial to testing of the group factor (PI versus IdI), we repeated this analysis, conservatively correcting for age as a covariate, because it had been found to correlate with some of the dependent variables. The model remained significant when age was in the equation (F(2,37) = 7.98, P = 0.001). There were, however, no significant differences between PI and IdI on any of the three BAS dimensions.

Sleep effort

An omnibus, multivariate test (again with age as a covariate) was conducted across the 7 items of the GSES. This test revealed a significant between-group multivariate effect (F(7, 31) = 3.40, P = 0.008). Subsequent univariate comparisons revealed that this effect was accounted for by 4 GSES items differing between the PI and IdI groups (Table 2).

Specifically, PI endorsed "putting too much effort into sleeping" (F(2) = 4.41, P = 0.019), being "anxious about sleeping before going to bed" (F(2) = 3.87, P = 0.030) and "worrying about the consequences of not sleeping" (F(2) = 3.34, P = 0.046) more strongly than did IdI. Conversely, there was a nonsignificant trend for the IdI participants to endorse being "no good at sleeping" more strongly than PI (F(2) = 3.01, P = 0.062).

The results from Study 1, therefore, broadly confirm the hypothesis that people with PI exhibit greater levels of threat sen-

sitivity in general and a more pronounced sleep-related focus than people with IdI.

STUDY 2: COMPARATIVE INVESTIGATION OF ILLNESS PERCEPTION, COPING STYLE, AND TREATMENT ACCEPTABILITY IN PI AND IDI

Methods

Participants

Eligibility criteria and recruitment procedures were identical to those used in Study 1 with 2 exceptions. First, the conservative exclusion zone for age of insomnia onset was set slightly larger, between the ages of 10 and 18 yr. No one whose insomnia started within this age range was accepted into the study. Second, anyone with previous or current experience of psychologic treatment for insomnia was excluded, and none of the participants was on pharmacotherapy for insomnia at the time of the study. Also in this study, the Dysfunctional Beliefs and Attitudes About Sleep Scale (DBAS-16)[29] was added to give a further descriptive profile of the samples.

Design

Cross-sectional, two-group comparison of PI and IdI.

Hypotheses

People with IdI will regard their insomnia as more permanent and will be more accepting of it than will people with PI. Both groups will rate a behavioral treatment as preferable to pharmacotherapy, but the IdI group will also consider an "acceptance treatment" in preference to pharmacotherapy.

Measures

Illness Perception Questionnaire-Revised: Acknowledgment that IdI (in particular) is an unrelenting condition suggests that it may be worthwhile considering patients' perceptions from a "chronic symptom" perspective. Pain and fatigue come to mind as parallel, poorly understood psychophysiologic conditions often displaying poor treatment response,[31,32] and being associated with clinician frustration and helplessness ("compassion fatigue).[33] The Illness Perception Questionnaire-Revised[30] (IPQ-R: 21 items) has proven valuable in such disorders to investigate patient perspectives, so it was selected as a suitable measure to explore illness perceptions of IdI relative to PI. Moreover, the IPQ-R is validated to permit the word illness to be replaced, in this instance with insomnia, to make it a disease-specific scale.

The IPQ-R includes a timeline: acute/chronic subscale (6 items), which measures perceptions of permanency (high subscale score indicates permanence: e.g., *"*my insomnia is likely to be permanent rather than temporary"; "I expect to have this insomnia for the rest of my life*"*) and a timeline: cyclical subscale (4 items), which measures if an individual perceives his or her illness to be variable, with either a cyclical nature or being constant and unrelenting (high subscale score indicates variability: e.g., "I go through cycles in which my insomnia gets better and worse"; "my insomnia is very unpredictable"). The two other IPQ-R subscales are also of interest. The personal control subscale (6 items) and treatability subscale (5 items) elicit individuals' beliefs in relation to having personal control over their illness (high subscale score indicates perceived lack of control: e.g., "I have the power to influence my insomnia") or if they believe a treatment might be effective in curing their illness (high subscale score indicates low treatment expectations: e.g., "There is nothing that can help my condition*"*).

Participants responded to each item using a 5-point Likert scale ranging from 0 (strongly disagree) to 4 (strongly agree). Several previous adaptations to particular health populations have yielded good internal consistency (Cronbach α = 0.70-0.90).[30] We obtained similar values across the subscales in the current study (Cronbach α = 0.68-0.89).

Illness Cognition Questionnaire: Similar to the IPQ-R, the Illness Cognition Questionnaire (ICQ) was developed for use in chronic diseases,[34] and can be worded specifically to the "illness" in question. We included the ICQ because it evaluates two important components of mental state, in our case in relation to insomnia. One scale measures the cognitive construct of "acceptance" (6 items: e.g., "I think I can handle the problems related to my insomnia, even if the insomnia gets worse*"* and *"*I can cope effectively with my insomnia"). A high score is indicative of low acceptance. The "helplessness" scale then provides an additional measure of impact/sense of control (6 items: e.g., "my insomnia controls my life" and "my insomnia limits me in everything that is important to me*"*). A high score indicates less helplessness/more control.

Items are rated ranging from 1 (agree) to 4 (disagree) (subscale score range = 4-24) The ICQ is reported as a reliable and valid assessment of perceptions of patients with a chronic disease (Cronbach α = 0.84-0.91).[34] In our study, subscale internal reliabilities were Cronbach α = 0.81 (acceptance) and 0.77 (helplessness).

Coping Style (Brief Cope): We also wanted to compare how people with PI and IdI perceive that they typically respond when confronted with difficult or stressful events. The 28-item Brief Cope profiles 14 coping styles: active coping, planning, positive refraining, acceptance, humor, religion, using emotional support, using instrumental support, self-distraction, denial, venting, substance use, behavioral disengagement, and self-blame.[35] Responses are scored ranging from 1 ("I usually don't do this at all") to 4 ("I usually do this a lot"). Some examples of items include *"*trying to see it in a different light, to make it seem more positive" (positive reframing), "turning to work or other activities to take my mind off things*"* (self-distraction), and *"*making fun of the situation*"* (humor). The Brief Cope is said to have psychometric properties consistent with its original 60-item version (Cronbach $\alpha \leq 0.90$).[35] We did not check the internal consistency of this short-form version because each subscale comprised only 2 items.

Treatment Acceptability Scale: Finally, we included a scenario-based assessment of patients" perceptions by adapting the treatment acceptability/preferences paradigm by Morin et al.[44] to include a novel treatment descriptor on acceptance treatment. In this regard it should be noted that there is growing evidence that acceptance-based strategies may be associated with better emotional adjustment across a range chronic health conditions.[58,59] The traditional Treatment Acceptability Scale (TAS) incorporates a behavioral treatment and a pharmacologic treatment scenario, each of which participants rate for treat-

ment acceptance (2 items), willingness to comply, suitability for sleep onset, and for sleep maintenance problems, perceived effectiveness (2 items), and side effects.[36] Each dimension is rated ranging from 1 to 6, giving a possible range of 8-48 per scale. We added an Acceptance Therapy scenario to explore this additional perspective to the way adults with PI and IdI might conceptualize insomnia treatment. The treatments were described in three ways.

Behavioral treatment is a nondrug treatment method aimed at teaching individuals a set of skills to help overcome their sleep problem. It provides specific guidelines for changing poor sleep habits and for regulating sleep schedules. Education about sleep hygiene factors (e.g., bedroom environment) is also provided. Pharmacologic treatment consists of taking a prescribed pill at a specified time. The prescribed medication is a naturally-occurring hormone that is essential for sleep. The specific dosage would be based on the nature and severity of the patient's sleep problem. Acceptance treatment is a nondrug treatment method aimed at encouraging acceptance of insomnia. It is designed to develop strategies for overcoming the effect of insomnia on a patient's life (e.g., engaging in increased activity or reducing distress caused by insomnia-associated thinking).

We modeled the pharmacologic treatment on a melatonin receptor agonist (mRA) for three reasons. First, previous studies have already indicated that a conventional sleeping pill is not particularly favored by people with insomnia, with behavioral treatment receiving higher ratings[36,37]; second, we wanted to "match" the novelty aspect of acceptance therapy, as a new(er) approach; and third, how people might respond to an mRA proposition was in itself interesting. In our study, all three TAS achieved satisfactory reliability (behavioral: Cronbach $\alpha = 0.88$; pharmacologic: Cronbach $\alpha = 0.82$; acceptance: Cronbach $\alpha = 0.92$).

Results

Sample demographics and sleep characteristics

A total of 61 adults with insomnia (48 females; mean age 36.6 (15.1) yr) participated. There were no differences between PI and IdI groups on sex or age, or on educational or relationship status (Table 3). Likewise, the groups were well matched on health status (Short Form-36)[39] and alcohol consumption, and had at most minor anxiety and/or depressive symptoms. Both PI and IdI groups reported sleep disturbance, in the moderate range on the PSQI and ISI, with no significant between-group differences. Average duration of insomnia was 7 years for the PI group and 26 years for the IdI group, yet dysfunctional thinking about sleep did not differ between groups, either in terms of Dysfunctional Beliefs and Attitudes About Sleep (DBAS-16) total score (Table 3) or on its subscales. None of the participants had received previous psychologic treatment. Some in both groups reported occasional previous use of prescribed and nonprescribed medication for insomnia, although none was taking prescribed medication at the time of assessment.

Table 3—Demographic, health, and sleep characteristics of study 2 samples[a]

	Psychophysiologic insomnia (n = 31)	Idiopathic insomnia (n = 30)
Demographic		
Sex [n (%)]		
Female	24 (77.4)	24 (80)
Male	7 (22.6)	6 (20)
Age (yr)	38.52 (14.5)	34.63 (15.7)
Education [n (%)]		
Secondary completed	14 (45.2)	10 (33.3)
Tertiary completed	17 (54.8)	20 (66.7)
Relationship status [n (%)]		
With partner	13 (41.9)	14 (46.7)
Living alone	18 (58.1)	16 (53.3)
Health		
Alcohol (units/wk)	6.67 (5.85)	6.8 (6.66)
BDI II	12.39 (6.67)	10.41 (5.88)
BAI	7.74 (3.98)	8.35 (5.8)
SF-36		
Physical	76.37 (15.34)	82.73 (12.51)
Mental	63.18 (18.37)	66.37 (17.92)
Sleep		
PSQI	10.25 (3.76)	10.90 (3.38)
ISI	14.74 (5.29)	16.23 (4.7)
DBAS-16	76.28 (25.76)	74.73 (29.34)

[a]All data represent mean (SD), unless otherwise stated. BAI, Beck Anxiety Inventory; BDI, Beck Depression Inventory; DBAS-16, Dysfunctional Beliefs and Attitudes About Sleep Scale; ISI, Insomnia Severity Index; PSQI, Pittsburgh Sleep Quality Index; SF-36, Short Form-36.

Illness perception

Both groups perceived their sleep difficulties to be relatively chronic, scoring above the mid-point on the IPQ-R permanency subscale (mean item scores: Table 4). However, the IdI group perceived their insomnia to have greater permanency than did the PI group (t (59) = 2.18, P = 0.033). There were no significance differences between groups on the variability, personal control, or perceived treatability subscales. It should be noted, however, that mean scores suggest that both groups tended to view their insomnia as variable and outside of their control, and had doubts about its treatability.

Illness cognition

Both PI and IdI groups scored in the midscale range on the ICQ acceptance subscale, indicating moderate levels of insomnia acceptance/lack of acceptance (Table 4). However, there were no significant differences between participants with PI and IdI. Likewise, there were no differences between groups on the ICQ helplessness scale (both P > 0.05).

Coping strategies

A comparison of profiles of use of the 14 different strategies measured by the Brief Cope is also presented in Table 4. Interestingly, the top three endorsed strategies (interpreting mean scores from highest to lowest) for PI were acceptance, active coping, and planning. Acceptance and active coping were also the two top-rated strategies in the IdI group, but with the use of humor scoring equal second. Between-group comparison showed that patients with IdI rated using humor to cope significantly more often than those with PI (t (59) = -2.73,

Table 4—Between-group comparison of study 2 samples on illness perception, illness cognition, coping style, and treatment acceptability scales[a]

	Psychophysiologic insomnia (n = 31)	Idiopathic insomnia (n = 30)
Illness perception questionnaire		
Permanency	3.44 (0.68)	3.84 (0.69)
Variability	2.99 (0.97)	2.78 (1.11)
Personal control	3.04 (0.65)	2.83 (0.72)
Treatability	3.24 (0.53)	3.25 (0.57)
Illness cognition questionnaire		
Acceptance	14.4 (4.67)	15.7 (4.21)
Helplessness	16.5 (4.42)	17.2 (4.50)
Coping style (Brief Cope)		
Active coping	5.33 (1.45)	5.85 (1.35)
Planning	5.27 (1.41)	5.69 (1.54)
Positive reframing	4.63 (1.45)	5.38 (1.81)
Acceptance	5.67 (1.37)	5.92 (1.74)
Humor	4.33 (1.94)	5.85 (2.20)
Religion	3.67 (2.14)	2.92 (1.87)
Emotional support	4.23 (2.05)	4.69 (2.22)
Instrumental support	4.70 (2.00)	4.28 (1.65)
Self distraction	4.73 (1.48)	5.73 (1.22)
Denial	2.83 (1.39)	3.19 (1.58)
Venting	4.63 (2.61)	4.28 (1.45)
Substance use	2.70 (1.29)	2.92 (1.29)
Behavioral disengagement	3.10 (1.54)	3.23 (1.73)
Self blame	4.23 (1.98)	4.12 (1.95)
Treatment acceptability scale		
Behavioral therapy	41.3 (7.63)	42.19 (8.84)
Pharmacotherapy	32.9 (9.34)	34.17 (7.23)
Acceptance therapy	30.2 (9.74)	36.20 (8.80)

[a]All data represent mean (SD), unless otherwise stated.

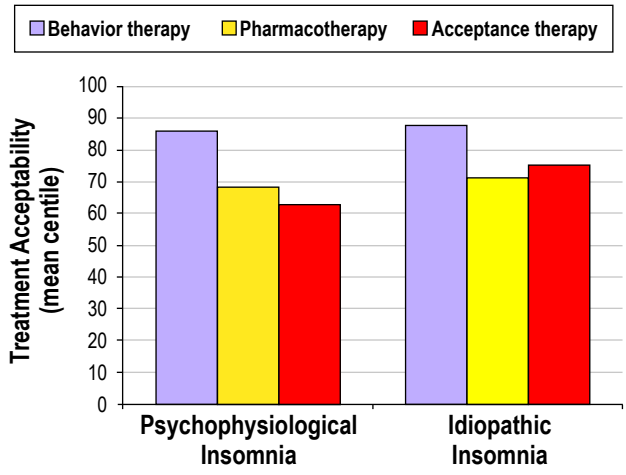


Figure 1—Between group comparison of Treatment Acceptability centile scores for Study 2 samples.

P = 0.008). The only other strategy that differentiated between group was self-distraction, which also was used more often by patients with IdI than by those with PI (t (59) = -2.72, P = 0.009]. These findings fail to achieve significance after conservative adjustment for multiple comparisons (critical value: P < 0.004).

Treatment acceptability

Table 4 also summarizes between-group comparisons across the three treatment scenarios on the TAS task. Mixed-model analysis of covariance (with age as a covariate) revealed significant effects of group (F(1) = 5.34, P = 0.025), treatment type (F(2) = 3.11, P = 0.049), and a near-significant group by treatment interaction (F(2) = 3.73, P = 0.059). There was no significant difference between PI and IdI in patient ratings of behavioral treatment or pharmacologic treatment. For the acceptance-based approach to insomnia, however, there was a significant effect, with the IdI group rating this intervention as acceptable relative to the PI group's lower ratings (t (61) = 2.40, P = 0.02). To consider the possibility that treatment acceptability covaried with duration of insomnia (independent of subtype), we correlated insomnia duration in the PI group with ratings for each treatment scenario. No significant effects were observed (behavioral (rho = -0.054); acceptance (rho = 0.026); pharmacologic (rho = -0.164); all P > 0.40).

Treatment acceptability scores for the PI and IdI groups are also presented as centiles in Figure 1. Related t-tests indicate that behavioral treatment was the highest rated both within the PI group (behavioral versus pharmacologic: t (31) = 3.30; P = 0.003; behavioral versus acceptance: t (31) = 5.03; P < 0.001] and the IdI group (behavioral versus pharmacologic: t (30) = 3.45; P = 0.002; behavioral versus acceptance: t (30) = 2.79; P = 0.01). Despite the visual impression that the PI group rated pharmacologic treatment higher than acceptance treatment, and that the IdI group rated acceptance higher than pharmacologic treatment, neither effect was not supported statistically (both P > 0.30).

The results from Study 2 lend some support to the hypothesis that IdI participants regard their insomnia as somewhat more permanent than PI. Whereas neither group regards insomnia as acceptable, an acceptance treatment was considered more favorably by IdI than by PI participants. Behavioral intervention was preferred to pharmacotherapy by both groups.

DISCUSSION

Despite some early interest in comparing and contrasting the insomnia phenotypes of IdI and PI, scientific progress has been limited. Although ICSD-2 retained the subtypes proferred in the original ICSD,[38] due to limited research data they were not included in DSM-IV and they seem unlikely to be in the DSM-5 nosology (expected publication in 2013). Nonetheless, their "clinical utility" has been recognized (compassion fatigue, DSM-IV). Therefore, we took this consensus clinical framework to conduct two consecutive studies, with the purpose of comparing and contrasting the patient perspectives and insomnia treatment preferences of people with IdI and PI. We endeavored to make clinical differentiation both valid and robust; e.g., by excluding anyone whose sleep problem developed during their teenage years, and by matching the IdI and PI groups on demographic factors, insomnia severity, and associated (but minimal) psychopathology. Thus, we recruited two relatively homogeneous insomnia subgroups

that differed primarily, and categorically, on their history of poor sleep experience.

In relation to results, the first thing to be said is that we observed many similarities between IdI and PI, across the psychologic measures that we applied. Both groups reported helplessness and that their insomnia felt out of control, and they expressed doubts about its treatability (ICQ and IPQ data). Also trying to accept their situation and to cope with it actively rather than passively seemed characteristic (Brief Cope). They also endorsed similar levels of dysfunctional thinking (DBAS-16) and both groups had a clear preference for a behavioral approach to insomnia intervention (TAS). Nevertheless, some hypothesized between-group differences were supported.

In Study 1, people with PI exhibited higher levels of behavioral inhibition or threat sensitivity (BIS), consistent with an etiologic model of vulnerability to conditioned arousal in PI. This vulnerability might find its expression during an insomnia acquisition phase, perhaps at a time of stress.[8,10,40] Likewise, analysis of sleep effort data (GSES) appears consistent with the ICSD-2 account that people with PI characteristically strive hard to sleep, and worry excessively about the consequences of not sleeping.[10,11] By way of contrast, there was a trend for people with IdI to endorse the notion that sleeping is just something that they are not any good at; perhaps not so much a learned performance failure, as a fundamental (lifelong) inability. This would be consistent with a trait hyperarousal perspective on IdI.

In Study 2, whereas both groups perceived their sleep problems to be chronic, and possibly permanent, this belief was held more strongly by people with IdI (IPQ). It is interesting, therefore, that as hypothesized, participants with IdI rated an acceptance treatment highly, an approach that seemed in comparison relatively unacceptable to people with PI (TAS). So we might speculate that there are phenotypical differences between IdI and PI in their readiness to adapt to a "living with insomnia" perspective. IdI participants were also more likely to use humor as a form of coping (Brief Cope). Humor is integral to some psychotherapeutic traditions, including Frankl's logotherapy approach,[41] from which paradoxic intention therapy for insomnia was first derived,[42] and also in what has become known as Acceptance and Commitment Therapy (ACT).[43] Perhaps humor provides some relief from the effect of symptoms on the person with insomnia, rather than directly influencing their occurrence *per se*? This would be consistent also with the comparatively higher use of self-distraction as a coping strategy in IdI.

We also replicated previous TAS experimental findings that behavioral therapy is highly rated by people with insomnia (here in both PI and IdI) and that it is generally preferred to pharmacotherapy,[36,37] in this case, to the scenario of an mRA compound, although we did not specifically mention melatonin in the study. However, our data also hint (nonsignificant trend) that people with PI might prefer pharmacotherapy to accepting that they have a chronic problem that they might need to live with (TAS).

Taken together with the cognitive-behavioral literature on insomnia and its treatment, what might our results imply for research and for clinical practice?

We know that cognitive behavioral therapy (CBT) is an effective intervention for (largely undifferentiated) persistent insomnia, with approximately two thirds of patients making a sustained response.[44-47] Therefore, there is a rationale for using CBT with both PI and IdI patients. From the data in this study, we can see that PI and IdI have common psychologic features that may form part of an insomnia-specific (e.g., dysfunctional beliefs about sleep) and a generic (e.g., sense of helplessness) focus for CBT. Moreover, both groups rated CBT highly, presumably because they thought that it would be (most) relevant to their situation.

By the same token, however, we know from the literature that one third of patients do not respond to CBT. Therefore, it seems reasonable to consider two things. First, what characterizes a nonresponder; and second, what pragmatically might be a "second line" therapy for those who fail to respond to adequately delivered CBT, as the behavioral treatment of first choice. Research has shown that it is difficult to predict, either *a priori* or *a posteriori*, who the responders and nonresponders might be[48]; although, importantly in relation to the current study, it seems likely that poor definition and subtyping in insomnia research studies could be one important factor.[49]

From our findings, we speculate that there is a rationale for considering an acceptance-based therapy as a second-line intervention for PI patients who have not responded to CBT; and potentially, as a first-line treatment for IdI. Perhaps the integration of aspects of acceptance into CBT is worth considering, although we note that the evolution of multimodal CBT over and above its components (e.g., relaxation, stimulus control, sleep restriction) has not been accompanied by greatly improved outcomes.[3] On balance, therefore, we would recommend that controlled evaluation of acceptance as a stand-alone therapy against a CBT comparator would be valuable, especially if patients can be stratified by insomnia subtype so that treatment × phenotype interactions can be reported. Such research would address two important clinical caveats: if a sleep problem cannot be eliminated, then failing to reduce its effect and associated distress would amount to poor quality care; and if a sleep problem can be solved, then living with insomnia in a contented way would be a suboptimal therapeutic solution.

To encourage more research, we note that the therapeutic value of acceptance is becoming influential across a range of other chronic medical and psychologic disorders. Contemporary "third wave" techniques such as mindfulness-based stress reduction, mindfulness-relaxation, and ACT,[50] raise the question of whether a person's relationship with their symptoms could change (rather than focusing on symptom reduction). In insomnia, this would translate into addressing concerns about lying awake in bed, rather than addressing directly the frequency or duration of lying awake in bed. There are several small or uncontrolled studies on acceptance and mindfulness approaches to insomnia in the literature,[51-57] and this work is very much welcomed. It is worth bearing in mind, however, that people may be less likely to endorse acceptance of a problem if they are participants in a study based at a center where they are actively seeking a remedy. In this study, most participants completed measures at home. Nevertheless, the study was clearly associated with our clinical research center. Therefore, there may be methodologic advantages in also evaluating acceptance with non-treatment-seeking samples and in explicitly community settings.

In conclusion, we wish to acknowledge important limitations to our studies. We used clinical interview criteria to establish a working diagnosis. Whereas this appears valid for the purpose of the study, and PSG would be unlikely to be informative about the PI/IdI discrimination, we cannot be certain that other sleep disorders were excluded. For example, it is possible that mild obstructive sleep apnea and/or periodic limb movement disorder could be present in either or both of our insomnia phenotype samples, Also, although we reviewed diary records, actigraphy may have been useful, for example, to confirm stability of poor sleep in IdI compared with night-to-night variability in PI. We also cannot completely exclude the possibility that the differences between PI and IdI reported here relate solely to the length of time that people have had problems with insomnia. Bivariate correlations of duration of insomnia with TAS scores in the PI group suggest that this was not a factor, and we included age as a covariate in all our major analyses. However, this is a research question that should be specifically addressed in the future. We would also point out that our description of acceptance treatment was necessarily brief (40 words) to match previously published behavioral treatment descriptions,[36] and possibly left the descriptor open to misinterpretation (e.g.,a coping strategy rather than a treatment). It is problematic to convey the therapeutic value (of any treatment) in so few words,and a further study should consider comparing more detailed outlines. Finally, we recognize that what people think and believe is not necessarily the best guide to what should be offered in the way of treatment, or to what will actually work for them.

ACKNOWLEDGMENTS

Infrastructure support provided by the Dr. Mortimer and Theresa Sackler Foundation. Thanks also to Shona Currie for her assistance in recruitment and to Kerry Ross for assistance in preparing this manuscript.

DISCLOSURE STATEMENT

This was not an industry supported study. The authors have indicated no financial conflicts of interest.

REFERENCES

1. Ohayon MM. Epidemiology of insomnia: what we know and what we still need to learn. Sleep Med Review 2002;6:97-111.
2. Espie CA. Stepped care: a health technology solution for delivering cognitive behavioral therapy as a first line insomnia treatment. Sleep 2009;32:1549–58.
3. Morin C, Bootzin R, Buysse D, Edinger J, Espie C, Lichstein K. Psychological and behavioral treatment of insomnia: update of the recent evidence (1998-2004). Sleep 2006;29:1398–1414.
4. Riemann D, Perlis ML. The treatments of chronic insomnia: a review of benzodiazepine receptor agonists and psychological and behavioral therapies. Sleep Med Rev 2009;13:205–14.
5. International Classification of Sleep Disorders: Diagnostic and Coding Manual: 2nd edition. Westchester, IL, American Academy of Sleep Medicine, 2005.
6. Edinger JD, Stout AL, Hoelscher T. Cluster analysis of insomniacs' MMPI profiles: relation of subtypes to sleep history and treatment outcome. Psychosom Med 1988;50:77-87.
7. Spielman AJ, Saskin P, Thorpy MJ. Treatment of chronic insomnia by restriction of time in bed. Sleep 1987;10:45–56
8. Perlis ML, Giles DF, Mendelson WB, Bootzin RR, Wyatt JK. Psychophysiological insomnia: the behavioural model and a neurocognitive perspective. J Sleep Res 1997;6:179-88.
9. Espie CA. Insomnia: conceptual issues in the development, persistence, and treatment of sleep disorder in adults. Annu Rev Psychol 2002;53:215-43.
10. Espie CA, Broomfield NM, MacMahon KMA, Macphee LM, Taylor LM. The attention-intention-effort pathway in the development of psychophysiologic insomnia: a theoretical review. Sleep Med Rev 2006;10:215-45.
11. Harvey AG. A cognitive model of insomnia. Behav Res Ther 2002;40:869-93.
12. Hauri PJ. Olmstead EM. Childhood-onset insomnia. Sleep 1980;3:59-65.
13. Hauri PJ. A cluster analysis of insomnia. Sleep 1983;6:326-38.
14. Bonnet MH, Arand DL. Hyperarousal and insomnia. Sleep Med Rev 1997;1:97-108.
15. Nofzinger EA, Buysse DJ, Germain A, Price J, Miewald J, Kupfer D. Functional neuroimaging evidence for hyperarousal in insomnia. Am J Psychiatry 2004;161: 2126-29.
16. Philip P, Guilleminault C. Adult psychophysiologic insomnia and positive history of childhood insomnia. Sleep 1996;19:3;16–22.
17. Reynolds CF, Kupfer DJ, Buysse DJ, Coble P, Yeager A. Subtyping DSM-III-R primary insomnia. a literature review by the DSM IV work group on sleep disorders. Am J Psychiatry 1991;148:432-38.
18. Buysse DJ, Reynolds CF, Monk TH, Berman SR, Kupfer DJ. The Pittsburgh sleep quality index: a new instrument for psychiatric practice and research. Psychiatry Res 1989;28:193-213.
19. Bastien CH, Vallieres A, Morin CM. Validation of the insomnia severity index as an outcome measure for insomnia research. Sleep Med 2001;2:297-307.
20. Beck AT, Steer RA, Brown GK. Beck depression inventory. New York, The Psychological Corporation, 1996.
21. Beck AT, Epstein N, Brown G, Steer R. An inventory for measuring clinical anxiety: psychometric properties. J Consult Clin Psychol 1988;56:893–7
22. Carver CS, White TL. Behavioral inhibition, behavioral activation, and affective responses to impending reward and punishment: the BIS/BAS scales. J Pers Soc Psychol 1994;67:319-333.
23. Gray JA. The neuropsychology of anxiety: an enquiry into the functions of the septo- hippocampal system. Oxford, Oxford University Press, 1982.
24. Gray JA. The neuropsychology of emotion and personality. In: Stahl SM, Iversen SD, Goodman EC, eds. Cognitive Neurochemistry. Oxford, Oxford University Press, 1987.
25. Campbell-Sills L, Liverant GI, Brown TA. Psychometric evaluation of the behavioral inhibition/behavioral activation scales in a large sample of outpatient with anxiety and mood disorders. Psychol Assess 2004;16:244-254.
26. Bijttebier P, Beck I, Claes L, Vandereycken W. Gray's reinforcement sensitivity theory as a framework for research on personality-psychopathology associations. Clin Psychol Rev 2009;29:421-30.
27. Broomfield NM, Espie CA. Towards a valid, reliable measure of sleep effort. J Sleep Res 2005;14:401–7.
28. Kohn L, Espie CA. Sensitivity and specificity of measures of the insomnia experience: a comparative study of psychophysiologic insomnia, insomnia associated with mental disorder and good sleep. Sleep 2005;28:104-12.
29. Morin CM, Vallieres A, Ivers H, Bouchard S, Bastien CH. Dysfunctional beliefs and attitudes about sleep (DBAS): validation of a briefer version (DBAS-16). Sleep 2003;26:29.
30. Moss-Morris R, Weinman J, Petrie KJ, Horne R, Cameron LD, Buick D. The revised illness perception questionnaire (IPQ-R). Psycholog Health 2002;17:1-16.
31. Van Damme S, Crombez G, Van Houdenhove B, Mariman A, Michielsen W. Well-being in patients with chronic fatigue syndrome: the role of acceptance. J Psychosom Res 2006;61:595.
32. McCracken LM, Eccleston CA. Prospective study of acceptance of pain and patient functioning with chronic pain. Pain 2005;118:164-9.
33. Figley CR. Compassion fatigue: psychotherapists' chronic lack of self care. J Clin Psychol 2002;58:1433-41.
34. Evers AWM, Kraaimaat FW, Van Lankveld W, Jongen PJH, Bijlsma JWJ. Beyond unfavorable thinking: the illness cognition questionnaire for chronic diseases. J Consult Clin Psychol 2001;69:1026–36.
35. Carver CS. You want to measure coping but your protocol's too long: consider the Brief COPE. Int J Behav Med 1997;4:92-100.
36. Morin CM, Gaulier B, Barry T, Kowatch RA. Patients' acceptance of psychological and pharmacological therapies for insomnia. Sleep 1992;15:302-5.

37. Vincent N, Lionberg C. Treatment preference and patient satisfaction in chronic insomnia. Sleep 2001;24:411-17.
38. American Sleep Disorders Association. International classification of sleep disorders. Rochester, MN, American Sleep Disorders Association, 1990.
39. Jenkinson C, Layte R, Wright L, Coulter A. The UK SF-36: an analysis and interpretation manual. Oxford, Health Services Research Unit, University of Oxford, 1996.
40. Spielman AJ, Saskin P, Thorpy MJ. Treatment of chronic insomnia by restriction of time in bed. Sleep 1987;10:45-55.
41. Frankl VE. The doctor and the soul, 2nd edition. New York, Knopf, 1965.
42. Ascher LM, Turner RM. Paradoxical intention and insomnia: an experimental investigation. Behav Res Ther 1979;17:408–11.
43. Hayes SC, Strosahl K, Wilson KG. Acceptance and commitment therapy: an experiential approach to behavior change. New York, Guildford Press, 1999.
44. Morin CM, Bootzin RR, Buysee DJ, Edinger JD, Espie CA, Lichstein KL. Psychological and behavioral treatment of insomnia: update of the recent evidence (1998-2004). Sleep 2006;29:1398-1414.
45. Riemann D, Perlis ML. The treatments of chronic insomnia: a review of benzodiazepine receptor agonists and psychological and behavioral therapies. Sleep Med Rev 2009;13:205-14.
46. NIH state-of-the-science conference statement on manifestations and management of chronic insomnia in adults. NIH Consens Sci Statements 2005;22:1-30.
47. Wilson SJ, Nutt DJ, Alford C, et al. British Association for Psychopharmacology consensus statement on evidence-based treatment of insomnia, parasomnias and circadian rhythm disorders. J Psychopharmacology 2010;24:1577–1600.
48. Espie CA, Inglis SJ, Tessier S, Harvey L. The clinical effectiveness of cognitive behaviour therapy for chronic insomnia: implementation and evaluation of a sleep clinic in general medical practice. Behav Res Ther 2001;39:45–60.
49. Edinger JD, Bonnet MH, Bootzin RR, et al. Derivation of research diagnostic criteria for insomnia: report of an American Academy of Sleep Medicine work group. Sleep 2004;27:1567-96.
50. Kabat-Zin J. Full catastrophe living: using the wisdom of your mind to face stress, pain and illness. New York, Delta, 1990.
51. Lundh L. The role of acceptance and mindfulness in the treatment of insomnia. J Cogn Psychother 2005;19: 29-39.
52. Heidenreich T, Tuin I, Pflug B, Michal M, Michalak J. Mindfulness-base cognitive therapy for persistent insomnia:a pilot study. Psychother Psychosom 2006;75:188–9.
53. Yook K, Lee SH, Ryu M, et al. Usefulness of mindfulness-based cognitive therapy for treating insomnia in patients with anxiety disorders: a pilot study. J Nerv Ment Dis 2008;196:501-3.
54. Ong JC, Shapiro SL, Manber R. Combining mindfulness meditation with cognitive-behavior therapy for insomnia: a treatment-development study. Behav Ther 2008; 39:171–82.
55. Ong JC, Shapiro SL, Manber R. Mindfulness meditation and cognitive behavioral therapy for insomnia: a naturalistic 12-month follow-up. Explore 2009; 5:30–6.
56. Britton WB, Haynes PL, Fridel KW, Bootzin RR. Polysomnographic and subjective profiles of sleep continuity before and after mindfulness-based cognitive therapy in partially remitted depression. Psychosom Med 2010;72:539-48.
57. Dalrymple KL, Fiorentino L, Politi MC, Posner D. Incorporating principles from acceptance and commitment therapy into cognitive-behavioral therapy for insomnia: a case example. J Contemp Psychother 2010; 40: 209-17.
58. Stanton AL, Revenson TA, Tennen H. Health psychology: psychological adjustment to chronic disease. Annu Rev Psychol 2007;58:565-92.
59. De Ridder D, Geenen R, Kuijer R, van Middendorp H. Psychological adjustment to chronic disease. Lancet 2008;372:246-55.

Clinical and psychometric appraisal of insomnia

Journal of Psychosomatic Research 2000, 48, 141-148

Journal of Affective Disorders 2004, 80, 145-153

British Journal of Clinical Psychology 2004, 43, 409-420

SLEEP 2004, 27, 1567-1596

SLEEP 2005, *28, 104-112*

Journal of Sleep Research 2005, 14, 401-407

SLEEP 2006, 29, 1155-1173

Journal of Behaviour Therapy & Experimental Psychiatry 2009, 40, 15–23

Sleep Medicine Reviews 2010, 14, 69–82

Journal of Psycho-oncology 2009, 19, 991-996

Sleep Medicine 2011, 12*, 735-747*

SLEEP 2012, 35, 815-823

Journal of Clinical Psychiatry 2012, 73, e1-e7

ELSEVIER

Journal of Psychosomatic Research 48 (2000) 141–148

Journal of Psychosomatic Research

Insomniacs' attributions: psychometric properties of the Dysfunctional Beliefs and Attitudes about Sleep Scale and the Sleep Disturbance Questionnaire

Colin A. Espie*, Stephanie J. Inglis, Linda Harvey, Suzan Tessier

Department of Psychological Medicine, University of Glasgow, Academic Centre, Gartnavel Royal Hospital, 1055 Great Western Road, Glasgow G12 0XH, Scotland

Abstract

Objective: Mental overactivity has been widely implicated in the development and maintenance of insomnia, making the accurate and valid measurement of cognitive variables of some importance. The purpose of this study was to investigate the psychometric properties of two existing attributional scales. **Methods:** Data are presented from 178 clinic attending insomniacs who completed the Dysfunctional Beliefs and Attitudes about Sleep Scale (DBAS) and the Sleep Disturbance Questionnaire (SDQ). Standard procedures for the psychometric evaluation of scales were adopted. **Results:** The internal consistency of the DBAS (30 items) was reasonable (Cronbach's $\alpha = 0.72$); however, a revised ten-item short form (DBAS-10) demonstrated a more robust principal component structure than the original scale (three relatively "pure" factors explained 55% of the variance). The derived subscales achieved satisfactory internal consistency, and the DBAS-10 demonstrated treatment-related measurement sensitivity. The DBAS-10, nevertheless, correlated highly ($r = 0.826$) with the DBAS. A four-factor solution for the SDQ is also presented (61% explained variance) with $\alpha = 0.67$. Internal consistency of these subscales ranged from 0.59 to 0.82. The association between the SDQ and DBAS-10 was modest ($r = 0.28$), suggesting that the scales have some independence. **Conclusions:** The scales offer potential for clinical and research work on insomnia and possible applications are discussed.

Keywords: Insomnia; Sleep; Beliefs; Scales

Introduction

Insomnia, defined as difficulty in initiating or maintaining sleep [3], is reported by up to 30% of adults and older adults, although prevalence for frequent, severe insomnia is closer to 10% to 15% [4–7]. There is evidence that such problems can persist relatively unchanged over many years [8].

A substantial body of psychological research has yielded important evidence on the genesis and maintenance of insomnia, and models and methods of cognitive behavioral treatment have emerged [9–12]. Investigation of the relative importance of cognitive and physiological arousal has found the former to be more strongly associated with sleep disruption [13–15]. Certainly, there is good evidence that attitudes, beliefs, and mental arousal are central to the complaint of insomnia [12,25].

It was first reported more than 20 years ago that poor sleepers complain of mental alertness in bed more than physiological arousal [16]. Similar conclusions were drawn in later studies with insomniacs [2,13]. However, this may be a general rule, because, in one study, having an overactive mind was the attribution of poor sleep rated most highly, by insomniacs and noninsomniacs. Both groups also rated cognitive aspects of their presleep state as being more difficult to control than somatic ones [17]. Furthermore, experimental study has found that manipulations aimed at increasing cognitive intrusion led to significantly longer latency to sleep onset [18,19]. Tape-recorded presleep cognitions [20] and presleep mental arousal in psychophysiological insomnia [15,21] have also been associated with delayed sleep latency. Indeed, one recent survey has reported that people dissatisfied with their sleep often report engaging in mental activity near bedtime [22]. Conversely, mental processing tasks that disrupt sleep-related cognitions appear to decrease sleep-onset time [23]. Thus, the na-

* Corresponding author. Tel.: 0141-211-3903; fax: 0141-357-4899.
E-mail address: c.espie@clinmed.gla.ac.uk (C.A. Espie)

0022-3999/00/$ – see front matter
PII: S0022-3999(99)00090-2

ture of the thoughts may influence the effect upon sleep. We have recently studied the relationship between thought content and actigraphically measured sleep and found that thoughts related to problem-solving, rehearsing and planning daytime events, and concern about feeling tired (but not sleeping) were the best predictors of delayed sleep onset [24].

The importance of emotional arousal also has been stressed, reflecting the finding that affect-laden (emotionally charged) cognitions are the ones most likely to interfere with the sleep process [11,12,25,26]. Thus, it may be the insomniac's beliefs about the negative experiences and consequences of insomnia that foster the development of the clinical complaint. This work on insomnia appears to parallel studies on "worry," which has been posited as a generic trait, measurable in a wide range of neurotic conditions [27,28], and studies on unwanted intrusive thoughts, which are in fact common in nonclinical populations [29,30]. Negative and distressing cognitions are the ones most likely to contribute to the development of obsessions; however, it is not the thoughts *per se* that are untypical or pathological, but the meaning and concern attributed to them [31,32]. The potential conceptual relatedness of nighttime and daytime intrusions, therefore, appears considerable.

In spite of considerable evidence of the importance of thoughts and beliefs in insomnia, there is no recognized "gold standard" measure of cognitive activity. The Pre-Sleep Arousal Scale [14] was published by Nicassio and coworkers in 1985 and demonstrated satisfactory internal consistency for both its somatic and cognitive subscales ($r = 0.81$ and $r = 0.76$, respectively). These constructs were found to have some degree of independence (74% unshared variance) and acceptable face and construct validity. In a recent study, the cognitive subscale of the PSAS demonstrated modest ($r = 0.351$), but statistically significant, validity against "live" tape-recordings of presleep thoughts as a criterion measure [24].

The Sleep Disturbance Questionnaire (SDQ) [2] was developed by Espie and coworkers in 1989 to guide the tailoring of cognitive-behavioral treatment in a major controlled outcome trial. The factor structure of the SDQ was investigated by principal components analysis, which yielded a three-factor solution, accounting for 68% of total variance. Factor 1 "mental anxiety," comprising 7 of the 12 items, accounted for the greatest proportion of explained variance. Validity and internal consistency were not investigated.

In 1993, Morin published his Dysfunctional Beliefs and Attitudes about Sleep Scale (DBAS) [1,12]. The DBAS scale is extremely useful in clinical practice as it helps to identify particular, salient, irrational, and often affect-laden thoughts that intrude prior to sleep onset. It comprises 30 analog-scaled items and the author described five subscales: misconceptions of the causes of insomnia; misattributions or amplifications of the consequences of insomnia; unrealistic sleep expectations; diminished perceptions of control; and faulty beliefs about sleep-promoting practices. However, the component structure, reliability, and validity of the DBAS scale have not been formally evaluated.

The aim of the present study was to investigate further the psychometric adequacy of two of the three aforementioned scales, namely the SDQ and the DBAS scale. Opportunity arose to undertake this work, on a large patient sample, in the context of a controlled outcome study of cognitive-behavioral therapy in general medical practice [33]. The results, therefore, derive from a methodologically sound protocol, although the insomniac sample was clinic-presenting. It was hoped that statistical appraisal of the scales would lead to more knowledgeable future application, both in clinical practice and for research purposes.

Methods

Subjects

Table 1 presents summary information on the sample, which comprised 178 adults consecutively selected into an out-patient program for treatment of chronic insomnia. The assessment protocol incorporated extensive screening for other disorders of sleep such as sleep apnea, periodic limb movements, parasomnias, and circadian disorders. Clinical interviewing and history-taking was supplemented by the use of sleep diaries [11]; actigraphic assessment of sleep pattern [39] (particularly useful to identify sleep phase disorders); and, where warranted, on the basis of interview information, polysomnographic assessment in a sleep laboratory [40]. The treatment program, which involved cognitive-behavioral therapy with the gradual withdrawal of hypnotics, has been described elsewhere [33,34]. The present cohort appears representative. The majority of patients was female (68%), and the average age of the sample was 50 years. Almost 60% had insomnia of >5-year duration and half were regular users of hypnotic drugs. Table 1 also demonstrates the severity of subjective sleep complaint (from sleep diary records). Mean sleep latency was 62 minutes and mean wake time after sleep onset (wakeful time in bed after sleep commenced) was 78 minutes. Thus, these patients represented both initial and sleep maintenance problems and appeared typical of the clinical population. Patients either presenting with, or in active treatment for, clinically significant depression or posttraumatic stress disorder were excluded. Thus, data from standard measures of emotional state revealed generally mild depressive and anxiety-based symptomatology as the norm with generally low levels of daytime sleep tendency.

Measures

Subjects completed the DBAS and SDQ at baseline entry to psychological treatment. For the purposes of

Table 1
Descriptive summary of demographic, sleep, and psychopathology data for the insomniac sample (n = 178; unless otherwise stated)

Descriptor	Values	n	Percent
Gender	F	121	68
	M	57	32
Occupational status	Working	70	39
	At home	57	32
	Retired	51	29
History of sleep problem (n = 172)	<2 years	38	22
	>2 and <5 years	34	20
	>5 and <10 years	33	19
	>10 and <20 years	33	19
	>20 years	34	20
Use of sleep medication	Not in past month	91	51
	Less than once per week	11	6
	Once or twice per week	11	6
	Three or more times per week	65	37
	Mean		SD
Age (years)	49.8		17.9
Sleep-onset latency (min)	61.5		55.1
Wake time after sleep-onset (min)	77.63		75.74
Total sleep time (hr)	5.83		1.57
Epworth Sleepiness Scale [35]	5.79		4.47
Beck Depression Inventory [36]	12.21		9.30
State–Trait Anxiety Inventory [37]			
State	36.70		13.06
Trait	43.53		12.84
Penn State Worry Questionnaire [38] (n = 172)	47.69		14.97

evaluating measurement sensitivity in this study, the DBAS was also completed at posttreatment and 3-month follow-up on smaller samples (n = 91 and 60, respectively). The DBAS comprises 30 statements reflecting beliefs and attitudes about sleep. Subjects are asked to place a mark for each statement on a 10-centimeter horizontal line with poles labeled "strongly agree" and "strongly disagree." The SDQ has 12 items, each on a 5-point scale ("never true," "seldom true," "sometimes true," "often true," "very often true"). Subjects are asked to rate items in relation to typical nights when they do not sleep well.

Results

Data on internal consistency and measurement sensitivity, and from factor-analytic studies, are reported. These will be presented for each measure in turn followed by correlational analyses of the interrelationship of the DBAS and SDQ.

DBAS

Internal consistency of the DBAS was investigated by calculation of α coefficients (Cronbach's α). These are presented in Table 2. The α for the total scale was reasonable at 0.72. Alpha if item-deleted mean was 0.71 (range 0.68 to 0.75). However, only two DBAS subscales achieved satisfactory internal consistency. Subscale 2, "misattributions or amplifications of the consequences of insomnia," had an $\alpha = 0.77$ and subscale 4, "diminished perceptions of control and predictability of sleep," a modest $\alpha = 0.41$. "Faulty beliefs about sleep-promoting practices" (subscale 5) achieved internal consistency of only 0.34 (range = 0.24 to 0.35), and "unrealistic sleep expectations" (subscale 3) had a low negative value, indicating a degree of inverse relationship among items. Subscale 1 ("misconceptions of the causes of insomnia") consisted of only two items, therefore, α was not computed. In light of these results, an exploratory principal components analysis (PCA) was conducted to consider other possible factor structures for the DBAS. However, PCA failed to achieve convergence. It seemed likely, therefore, that some amendments to the DBAS scale would be required.

Development of the DBAS-10

As the starting point for redevelopment of the scale we decided to examine the measurement sensitivity of each of the original 30 DBAS items. It was felt that items that demonstrated significant posttreatment and follow-up changes over baseline would be important to retain in a new version of the scale. Such items could be regarded as valid because they would represent a durable, treatment-related shift in attitudes and beliefs. A series of paired *t*-test comparisons was made and a conservative criterion value of $p < 0.01$ was selected for the comparisons. The results of this analysis revealed that ten items could be regarded as significantly lower at posttreatment (range of $p = 0.009$ to 0.0001; $df = 90$) and at follow-up ($p = 0.007$ to 0.0001; $df = 59$). These items were retained, therefore, for further analysis.

The ten-item scale (DBAS-10; see Table 3) was found to have internal consistency of 0.69 (Table 2). Investigation of its structure by means of PCA yielded a factorially "pure" solution, explaining 55% of total variance. Three factors had eigenvalues >1 and these accounted for 54%, 27%, and 19% of explained variance, respectively (Table 3). Factor I comprised five items from the original scale (statements 1, 2, 10, 12, 21) and was labeled "beliefs about the immediate negative consequences of insomnia." Factor II, "beliefs about the long-term negative consequences of insomnia," comprised three of the original statements (5, 8, 17), and factor III, "beliefs about the need for control over insomnia," comprised two statements (7, 22). It should be noted that statement

Table 2
Internal consistency of the SDQ, DBAS and DBAS-10 scales and their factor scales (N/A refers to factors with two or less items) (n = 178)

Scale	Coefficient α	α if item deleted (range)
DBAS total	0.717	0.684 to 0.748
DBAS subscale 1	N/A	N/A
DBAS subscale 2	0.773	0.706 to 0.783
DBAS subscale 3	−0.233	−0.856 to 0.219
DBAS subscale 4	0.411	0.302 to 0.567
DBAS subscale 5	0.343	0.240 to 0.351
DBAS-10 total	0.693	0.629 to 0.719
DBAS-10 factor I	0.733	0.650 to 0.716
DBAS-10 factor II	0.600	0.264 to 0.739
DBAS-10 factor III	N/A	N/A
SDQ total	0.674	0.614 to 0.727
SDQ factor I	0.586	0.477 to 0.698
SDQ factor II	0.818	0.714 to 0.767
SDQ factor III	N/A	N/A
SDQ factor IV	N/A	N/A

22 loads inversely on factor III, thus representing belief in the need for active control over thought processes. All factor loadings were greater than $r = 0.45$, with the majority being greater than $r = 0.68$. Internal consistency for factors I and II was calculated as 0.73 and 0.60, respectively (Table 2). Because factor III had only two items, the α was not computed.

SDQ

The internal consistency of the 12-item SDQ was calculated to be 0.67, indicating satisfactory reliability (Table 2). PCA was again used to investigate structural properties and this yielded a four-factor solution explaining 61% of total variance (Table 3). Factor I, "attributions concerning restlessness/agitation," accounted for the greatest amount of explained variance (46%). It comprised six items from the original scale (1, 3, 4, 5, 8, 9), with internal consistency of 0.59. Factor II, "attributions concerning mental overactivity," comprised three items (2, 6, 10), and had high internal reliability ($\alpha = 0.82$; Table 2). Factor III, "attributions concerning the consequences of insomnia," and factor IV, "attributions concerning lack of sleep readiness," comprised two items (11, 12) and one item (7) respectively; therefore, α-values were not computed. Loadings on factors II, III, and IV were all greater than $r = 0.77$, and on factor I were in the range $r = 0.41$ to 0.68. Inspection of Table 3 demonstrates that PCA provided a relatively "pure" solution.

Relationships between the scales

The DBAS-10 total score was found to correlate highly and significantly with the original DBAS total ($r = 0.826$). Each of the DBAS-10 factors correlated with the DBAS-10 total score (range 0.48 to 0.86; Table 4). Similarly, the SDQ factors correlated significantly with the SDQ total (0.31 to 0.88). As would be expected, the strength of relationship between factor and total scale score reflected the proportion of explained variance associated with that factor. Intercorrelation among DBAS-10 factors was modest (all $r < 0.30$). Similarly, SDQ factors appeared relatively independent with five of the six correlations being less than $r = 0.20$. SDQ factors I and II did, however, demonstrate a stronger, positive association ($r = 0.38$; $p < 0.001$).

Intercorrelation among DBAS-10 and SDQ factor scores is also presented in Table 4. A modest, but significant, relationship was found between total scores on the DBAS-10 and SDQ ($r = 0.28$), and there were few strong relationships between the measures at the factor level, suggesting that the DBAS-10 and SDQ are somewhat independent of one another. "Beliefs about the long-term negative consequences of insomnia" (DBAS-10 factor II) correlated positively, but relatively weakly ($r = 0.30$), with "attributions concerning restlessness/agitation" (SDQ factor I) and "attributions concerning consequences of insomnia" (SDQ factor III). "Beliefs about the need for control over insomnia" (DBAS-10 factor III) correlated more strongly ($r = 0.49$) with "attributions concerning mental overactivity" (SDQ factor II) and "restlessness/agitation" ($r = 0.37$; SDQ factor I). DBAS-10 factor I ("beliefs about the immediate, negative consequences of insomnia"), which accounted for the greatest proportion of variance in the DBAS-10 scale, did not correlate with any of the SDQ factors ($r = -0.13$ to 0.12).

Discussion

In spite of an increasing emphasis upon cognitive factors in understanding and treating insomnia, there are few suitable, tested measures available in this field. Those that have been introduced in the literature have not generally been subject to further rigorous scientific study, although they may have become part of common usage in clinical practice. The DBAS and the SDQ are two promising measures that have been available for the past 5 to 10 years. The results of this study indicate that each has some structural and psychometric strength.

DBAS and DBAS-10

Although the DBAS scale had good overall internal consistency, further analysis proved somewhat disappointing. Only one of the five suggested subscales demonstrated sound internal consistency and DBAS failed to achieve convergence during principal components analysis. Thus, it was not possible to confirm Morin's putative factor structure [1,12]. However, a shortened ten-item scale emerged that had many of the qualities required of a rating scale instrument.

Table 3
Results from principal components analyses of DBAS-10 and SDQ comprising derived factors, loadings (significant values in bold), labels and item content (n = 178)

Factor	Factor label	Eigenvalue	Variance (%)	Cumulative (%)	Explained variance (%)
DBAS-10 factor I	"Beliefs about the immediate negative consequences of insomnia"	2.947	29.5	29.5	53.7
DBAS-10 factor II	"Beliefs about the long-term negative consequences of insomnia"	1.487	14.9	44.3	27.2
DBAS-10 factor III	"Beliefs about the need for control over insomnia"	1.055	10.5	54.9	19.1
SDQ factor I	"Attributions concerning restlessness/ agitation"	3.434	28.6	28.6	46.8
SDQ factor II	"Attributions concerning mental overactivity"	1.568	13.1	41.7	21.4
SDQ factor III	"Attributions concerning the consequences of insomnia"	1.263	10.5	52.2	17.2
SDQ factor IV	"Attributions concerning lack of sleep readiness"	1.065	8.9	61.1	14.6

Rotated factor matrix	Item content	Factor I	Factor II	Factor III	Factor IV
DBAS-10 01 (item 01)[a]	I need 8 hours of sleep to feel refreshed and function well during the day	**0.686**	−0.113	0.145	
DBAS-10 02 (item 02)	When I don't get the proper amount of sleep on a given night, I need to catch up on the next day by napping or on the next night by sleeping longer	**0.699**	−0.077	0.087	
DBAS-10 03 (item 05)	I am concerned that chronic insomnia may have serious consequences on my physical health	0.163	**0.774**	−0.046	
DBAS-10 04 (item 07)	When I have trouble getting to sleep, I should stay in bed and try harder	0.103	0.147	**0.873**	
DBAS-10 05 (item 08)	I am worried that I may lose control over my abilities to sleep	0.125	**0.816**	−0.021	
DBAS-10 06 (item 10)	After a poor night's sleep, I know that it will interfere with my daily activities on the next day	**0.709**	0.316	0.014	
DBAS-10 07 (item 12)	When I feel irritable, depressed, or anxious during the day, it is mostly because I did not sleep well the night before	**0.617**	0.399	−0.101	
DBAS-10 08 (item 17)	When I sleep poorly on one night, I know it will disturb my sleep schedule for the whole week	−0.056	**0.477**	0.038	
DBAS-10 09 (item 21)	When I feel tired, have no energy, or just seem not to function well during the day, it is generally because I did not sleep well the night before	**0.707**	0.144	−0.298	
DBAS-10 10 (item 22)	I get overwhelmed by my thoughts at night and often feel I have no control over this racing mind	0.113	0.404	**−0.454**	
SDQ 01	I can't get into a comfortable position in bed	**0.642**	0.003	0.041	0.110
SDQ 02	My mind keeps turning things over	0.104	**0.884**	−0.084	0.067
SDQ 03	I can't get my sleep pattern into a proper routine	**0.412**	0.056	−0.032	−0.087
SDQ 04	I get too worked up at not sleeping	**0.681**	0.095	0.202	0.171
SDQ 05	I find it hard to physically let go and relax my body	**0.590**	0.202	−0.018	−0.291
SDQ 06	My thinking takes a long time to unwind	0.235	**0.804**	−0.079	0.079
SDQ 07	I don't feel tired enough at bedtime	0.016	0.084	0.049	**0.898**
SDQ 08	I try too hard to get to sleep	**0.598**	0.098	0.081	0.508
SDQ 09	My body is full of tension	**0.654**	0.358	0.104	0.240
SDQ 10	I am unable to empty my mind	0.075	**0.831**	0.332	−0.025
SDQ 11	I spend time reading/watching TV in bed when I should be sleeping	−0.076	0.019	**0.796**	0.041
SDQ 12	I worry that I won't cope tomorrow if I don't sleep well	0.251	0.036	**0.774**	0.043

[a] Item number in original DBAS.

Table 4
Intercorrelation of DBAS-10 and SDQ scales and subscales (n = 178)

	DBAS-10 factor I	DBAS-10 factor II	DBAS-10 factor III	SDQ total	SDQ factor I	SDQ factor II	SDQ factor III	SDQ factor IV
DBAS-10 total	0.858**	0.674**	0.477**	0.279**	0.264**	0.164*	0.206*	−0.021
DBAS-10 factor I		0.286**	0.191	0.034	0.051	−0.044	0.124	−0.132
DBAS-10 factor II			0.214*	0.342**	0.300**	0.149	0.273**	0.134
DBAS-10 factor III				0.402**	0.372**	0.487**	0.108	0.017
SDQ total					0.882**	0.649**	0.469**	0.309**
SDQ factor I						0.383**	0.192*	0.136
SDQ factor II							0.157	0.105
SDQ factor III								0.134

*$p < 0.01$; **$p < 0.001$.

First, items in the DBAS-10 demonstrated measurement sensitivity to cognitive-behavioral treatment. This suggests that the DBAS-10 will be useful not only as a means of descriptive quantification of "thinking errors" prior to treatment, but also as an outcome measure of treatment-related cognitive change. Second, PCA yielded a factorially "pure" solution comprising three factors that form conceptually meaningful subscales. Beliefs about the immediate (next day) negative consequences of insomnia can be discriminated from beliefs about long-term consequences (e.g., to health) and from beliefs about needing to actively control the process of getting to sleep. Third, DBAS-10 and its factor scales demonstrated satisfactory internal consistency, suggesting some stability in measurement; finally, DBAS-10 total score correlated highly ($r = 0.83$) with the original DBAS total scale score. This indicates that the overall thrust of the DBAS has been maintained in the DBAS-10 while its structural integrity has been improved.

It should be observed that the item content for factors I and II of the DBAS-10 (respectively, beliefs about the immediate, and the long-term, negative consequences of insomnia) and Morin et al.'s [12] DBAS subscale of "misattributions or amplifications of the consequences of insomnia" only partly overlaps (i.e., items 5, 12, 21). Of course, Morin [12] derived his subscales conceptually rather than statistically. It is of interest, therefore, that conceptual similarities seem to exist between the DBAS and the DBAS-10 in relation to beliefs about the consequences of insomnia, but the item content is less consistent. Likewise, both versions of the scale identify the importance of beliefs about "control." However, in the DBAS-10, item 22, concerning control over nocturnal thoughts, loads along with item 7 concerning beliefs about the need to try harder to sleep (on factor III), whereas, in the DBAS, this latter item is placed within a category of beliefs about sleep-promoting practices.

SDQ

The SDQ is a different type of measure from the DBAS-10. Whereas the latter identifies beliefs and assumptions concerning insomnia, and particularly the perceived effects of insomnia, the SDQ seeks to identify causal attributions concerning the perceived sources of the sleep problem. In this regard it should be noted that the two scales were only modestly related to one another, as evidenced by the generally nonsignificant, or otherwise low, correlation coefficients. The strongest correlation between the scales was for DBAS-10 factor III, "need for control," and SDQ factor II, "mental overactivity." It seems reasonable that insomniacs attributing sleeplessness to mental alertness would endorse beliefs concerning control. This would typify the "performance anxiety" previously reported in some insomniacs [11].

In this study, patients were not reassessed on the SDQ because it was not regarded as a likely outcome measure. Nevertheless, two principle conclusions may be drawn from the statistical analyses.

First, the SDQ also yielded a robust factor structure with relatively high factor loadings across its 12 items. Factor I appears to comprise elements of perceived physiological arousal combined with poor "stimulus control" [9,11]. Factor II represents a particularly pure factor of mental overarousal and corresponds strongly with the first factor extracted in a previous analysis of the SDQ [2]. Factor III represents attributions concerning perceived consequences of insomnia to both daytime and nighttime behavior, and factor IV, "lack of sleep readiness," preeminently relates to the principles underlying "sleep hygiene" [10]. Second, the SDQ was found to have sound internal consistency. Item deletions did not greatly affect α-values, which again indicates stability and measurement.

The original factor structure of the SDQ yielded a three-factor solution [2] compared with the four factors in the present study. Nevertheless, there are some replicated findings. The present factor II, "mental overactivity," comprises the three items (2, 6, 10) that had the highest factor loadings on a factor we originally labeled "mental anxiety." Also, factor I, "restlessness/agitation," incorporates the three items (1, 5, 9) that comprised the "physical tension" factor in the earlier report.

Application of the scales

It is suggested, therefore, that both the DBAS-10 and SDQ have a credible scientific basis and may be

useful in further studies on the cognitive basis of insomnia and its treatment. Both scales appear to have conceptually sensible factor scales that will be useful both to describe insomniacs' perceptions and attributions of sleep problems and, in the case of the DBAS-10, to evaluate changes in underlying beliefs and assumptions. The original DBAS scale will continue to be useful at a descriptive level, especially on an item-by-item basis to guide treatment.

As a measure of causal attribution, the SDQ could be compared in further studies with the Pre-Sleep Arousal Scale [14] (see Introduction). In particular, it should be noted that SDQ factors I and IV might be primarily related to physiological activation on the PSAS and factors II and III to cognitive activation. The effective targeting of patients to therapy and/or tailoring of therapy to meet presenting need continues to be a matter of both research and clinical interest and these scales may provide a valuable starting point. It is also noteworthy that the DBAS-10 and SDQ scales, being 10 and 12 items in length, respectively, can be completed quickly in the clinic (within 3 to 5 minutes) and, therefore, can be recommended to the busy clinician. Finally, both measures require further, independent study by other research groups. Reports of test–retest performance and relationship to presenting characteristics of insomnia would be particularly welcome.

Acknowledgments

This research was funded by a grant from the Chief Scientist Office, Scottish Office Department of Health, under the Novel Health Services Research Initiative, and by a grant from the Ayrshire and Arran Health Board. We particularly thank health visitors Jean McMillan, Gillian Dalziel, Evelyn Berry, Christine McArthur, Maureen McVey, and Linda MacDonald, and Barbara Gilbert (secretary), for all their hard work on this project. We are grateful also to Dr. Sandra Steele, Dr. Eilidh Renwick, and the many GPs who referred patients, and to Anne McLellan, Susan Dawson, and Tracey Hepburn, for their invaluable support.

References

[1] Morin CM, Stone J, Trinkle D, Mercer J, Remsberg S. Dysfunctional beliefs and attitudes about sleep among older adults with and without insomnia complaints. Psychol Ageing 1993;8:463– 467.
[2] Espie CA, Brooks DN, Lindsay WR. An evaluation of tailored psychological treatment of insomnia. J Behav Ther Exper Psychiatry 1989;20:143–153.
[3] American Sleep Disorders Association. The international classification of sleep disorders. Diagnostic and coding manual—revised. Rochester, Minnesota: ASDA 1997.
[4] Bixler EO, Kales A, Soldatos CR, Kales JD, Healy B. Prevalence of sleep disorders in the Los Angeles metropolitan area. Am J Psychiatry 1979;136:1257–1262.
[5] Gallup Organization. Sleep in America. Princeton, New Jersey: Gallup 1991.
[6] Foley DJ, Monjan AA, Brown SL, Simonsick EM, Wallace RB, Blazer DG. Sleep complaints among elderly persons: an epidemiologic study of three communities. Sleep 1995;18:425–433.
[7] Ford DE, Kamerow DB. Epidemiologic study of sleep disturbances and psychiatric disorders. JAMA 1989;262:1479–1484.
[8] Mendelson WB. Long-term follow-up of chronic insomnia. Sleep 1995;18:698–701.
[9] Bootzin RR, Nicassio PM. Behavioural treatments of insomnia. In: Hersen M, Eisler RE, Miller PM, eds. Progress in behaviour modification, volume 6. New York: Academic Press. pp. 1–45.
[10] Lacks P. Behavioral treatment for persistent insomnia. New York: Pergamon Press 1987.
[11] Espie CA. The psychological treatment of insomnia. Chichester, UK: John Wiley & Sons 1991.
[12] Morin CM. Insomnia: psychological assessment and management. New York: Guilford Press 1993.
[13] Lichstein KL, Rosenthal TL. Insomniacs' perceptions of cognitive versus somatic determinants of sleep disturbance. J Abnorm Psychol 1980;89:105–107.
[14] Nicassio PM, Mendlowitz DR, Fussell JJ, Petras L. The phenomenology of the pre-sleep state: the development of the Pre-Sleep Arousal Scale. Behav Res Ther 1985;23:263–271.
[15] Broman JE, Hetta J. Perceived pre-sleep arousal in patients with persistent psychophysiologic and psychiatric insomnia. Nord J Psychiatry 1994;48:203–207.
[16] Evans FJ. Subjective characteristics of sleep efficiency. J Abnorm Psychol 1977;86:561–564.
[17] Watts FN, Coyle K, East MP. The contribution of worry to insomnia. Br J Clin Psychol 1994;33:211–220.
[18] Gross RT, Borkovec TD. Effects of cognitive intrusion manipulation on sleep-onset latency of good sleepers. Behav Ther 1982; 13:112–116.
[19] Hall M, Buysse DJ, Reynolds CF, Kupfer DJ, Baum A. Stress related intrusive thoughts disrupt sleep-onset and continuity of sleep. Sleep Res 1996;25:163.
[20] Van Egeren L, Haynes SN, Franzen M, Hamilton J. Presleep cognitions and attributions in sleep-onset insomnia. J Behav Med 1983;6:217–232.
[21] Kuisk LA, Bertelson AD. Presleep cognitive hyperarousal and affect as factors in objective and subjective insomnia. Percept Motor Skills 1989;69:1219–1225.
[22] Ohayon MM, Caulet M, Guilleminault C. How a general population perceives its sleep and how this relates to the complaint of insomnia. Sleep 1997;20:715–723.
[23] Haynes SN, Adams AE, Franzen M. The effects of pre-sleep stress on sleep-onset insomnia. J Abnorm Psychol 1981;90:601–606.
[24] Wicklow A, Espie CA. Intrusive thoughts and their relationship to actigraphic measurement of sleep: towards a cognitive model of insomnia. Behav Res Ther (in press); 38.
[25] Espie CA, Wicklow A. Cognitive therapy for insomnia. In: Shapiro C, Sloan EP, eds. Sleep in psychiatry. Cambridge, UK: Cambridge University Press 2000.
[26] Coyle K, Watts FN. The factorial structure of sleep dissatisfaction. Behav Res Ther 1991;29:513–520.
[27] Barlow DH. Anxiety and its disorders. New York: Guilford Press 1988.
[28] Meyer TJ, Miller ML, Metzger RL, Borkovec TD. Development and validation of the Penn State Worry Questionnaire. Behav Res Ther 1990;28:487–495.
[29] Rachman S, De Silva P. Abnormal and normal obsessions. Behav Res Ther 1978;16:233–248.
[30] Reynolds M, Salkovskis PM. Comparison of positive and negative intrusive thoughts and experimental investigation of the differential effects of mood. Behav Res Ther 1992;30:273–281.
[31] Clark DA, Purdon CL. New perspectives for a cognitive theory of obsessions. Austral Psychologist 1993;28:161–167.
[32] Clark DA, Purdon CL. The assessment of unwanted intrusive thoughts: a review and critique of the literature. Behav Res Ther 1995;33:967–976.

[33] Espie CA, Inglis SJ, Tessier S, Harvey L. The clinical effectiveness of cognitive behaviour therapy for chronic insomnia: implementation and evaluation of a sleep clinic in general medical practice. Behav Res Ther (in press); 38.

[34] Espie CA, Brindle S, Tessier S, Dawson S, Hepburn T, McLellan A, Renwick E, Steele S, McColl J. Supervised cognitive-behaviour therapy for insomnia in general medical practice—preliminary results from the West of Scotland programme. In: Sanavio E, ed. Behavior and cognitive therapy today: essays in honour of Hans J. Eysenck. Amsterdam: Elsevier 1998.

[35] Johns MW. A new method for measuring daytime sleepiness: the Epworth Sleepiness Scale. Sleep 1991;14:540–545.

[36] Beck AT, Ward CH, Mendelson M, Mock J, Erbaugh J. An inventory for measuring depression. Arch Gen Psychiatry 1961; 4:561–571.

[37] Spielberger CD, Gorsuch RL, Lushene RE. Manual for the State-Trait Anxiety Inventory. Palo Alto, California: Consulting Psychologists Press 1970.

[38] Meyer TJ, Miller ML, Metzger RL, Borkovec TD. Development and validation of the Penn State Worry Questionnaire. Behav Res Ther 1990;28:487–495.

[39] Sadeh A, Hauri PJ, Kripke DF, Lavie P. The role of actigraphy in the evaluation of sleep disorders. Sleep 1995;18:288–302.

[40] Reite M, Buysse D, Reynolds C, Mendelson W. The use of polysomnography in the evaluation of insomnia. Sleep 1995;18:57–58.

ELSEVIER

Journal of Affective Disorders 80 (2004) 145–153

JOURNAL OF AFFECTIVE DISORDERS

www.elsevier.com/locate/jad

Research report

The sleep of remitted bipolar outpatients: a controlled naturalistic study using actigraphy

Audrey Millar[a], Colin A. Espie[a,*], Jan Scott[b]

[a] *Department of Psychological Medicine, University of Glasgow, Academic Centre, Gartnavel Royal Hospital, 1055 Great Western Rd, Glasgow G12 0XH, UK*

[b] *Division of Psychological Medicine, Institute of Psychiatry, Glasgow, UK*

Received 2 May 2002; received in revised form 14 February 2003; accepted 14 February 2003

Abstract

Background: Several sleep laboratory studies suggest sleep abnormalities in bipolar disorder. However, this is the first study to compare remitted bipolar subjects with controls on actigraphic and subjective sleep parameters in a naturalistic setting over 5 nights. *Methods:* Nineteen subjects with Bipolar I Disorder and 19 age- and gender-matched healthy controls were included. Objective sleep parameters were estimated using wrist actigraphs. Subject-rated sleep diaries and mood ratings were also completed. Sleep data were averaged for each subject across nights, and raw score standard deviations were calculated as a measure of within-subject variability. *Results:* Multivariate analyses of variance found significant group differences for both actigraphic ($F(4,33)=3.80$, $P=0.012$) and subjective measures ($F(4,31)=3.18$, $P=0.027$). Univariate analyses identified reliable differences in sleep onset latency (subjective), sleep duration (subjective), and variability of sleep duration and night wake time (actigraphic). Binary backward stepwise logistic regression demonstrated that a combination of three sleep measures correctly predicted disorder status in 84% of cases. *Limitations:* Failure to match on sociodemographic and employment status is a limitation that may provide an alternative explanation for some findings. Furthermore, in the bipolar group 18 of 19 subjects were in receipt of psychotropic medication, compared to none of the healthy control group. Also, no information was recorded about family history of mental disorders in the control group. *Conclusions:* The study suggests that the sleep of remitted bipolar outpatients measured in naturalistic settings is characteristically different from controls: bipolar subjects sleep longer, report longer onset latencies, and display greater variability across nights.

Keywords: Actigraphy; Bipolar affective disorders; Manic depression; Sleep disturbance

1. Introduction

Bipolar disorders (BP) are common, severe and persistent disorders, which affect 1.3–1.7% of the population (Kessler et al., 1996). Hyposomnia and hyperactivity are two defining features of mania, while reduced activity and hypersomnia characterise bipolar depression (Leibenluft et al., 1995). The pervasiveness of sleep disturbances in BP suggests that they are not merely symptomatic, but may be central in the aetiology of symptoms.

There is evidence that changes in sleep and activity may act as sensitive markers for relapse in BP, with

* Corresponding author.
E-mail address: c.espie@clinmed.gla.ac.uk (C.A. Espie).

0165-0327/$ - see front matter © 2003 Elsevier B.V. All rights reserved.
doi:10.1016/S0165-0327(03)00055-7

reduced sleep and increased activity being associated with the onset of a manic episode (Barbini et al., 1996; Klein et al., 1991, 1992; Leibenluft et al., 1996; Nowlin-Finch et al., 1994). There is also compelling evidence that sleep abnormalities are trait markers of mood disorder, which persist even when subjects are not in an episode. The evidence is more clear in the case of unipolar (UP) depression, where it has been shown that reduced REM latency is a stable sleep characteristic in those with a history of UP depression, even when remitted (Rush et al., 1986; Giles et al., 1990, 1993).

In one of the few studies exploring sleep characteristics of remitted BP subjects, Sitaram et al. (1982) reported an increased density and percentage of REM sleep in remitted BP patients (n=14) compared to a control group. The study also found that the patients were more sensitive to the effects of arecoline, an acetylcholine agonist that can reduce REM latency. Knowles et al. (1986) undertook electroencephalographic assessment of remitted BP patients on five consecutive nights. In many respects, the sleep of the 10 remitted patients was similar to that of 10 controls. However, the BP patients had a higher percentage of time in stage 1 sleep, and more disturbed sleep than controls.

The above data are consistent with the view that BP patients may have characteristic sleep disturbances. However, a major limitation of this research is its reliance on polysomnography (PSG). For logistic reasons polysomnographic studies are usually confined to one or two nights. In addition, the artificial nature of the sleep laboratory may disrupt natural rhythms. For example, the fact that subjects are woken at predetermined times may lead to underestimation of sleep duration in hypersomnic subjects (e.g., Wehr et al., 1985). A potential solution to these problems is offered by actigraphy, which employs a small motion-sensitive device, worn like a wristwatch, that can sample physical activity levels continuously for prolonged periods (Sadeh et al., 1995). A range of sleep-related variables is calculated from raw movement data (e.g., sleep onset latency, sleep duration, amount of wake time during the night, and 'sleep efficiency'). Although actigraphy is less precise than polysomnography, and does not discriminate sleep stages, comparative studies have reported 80–90% agreement between PSG and actigraphic recordings with respect to determination of sleep and wakefulness (Sadeh et al., 1989). The practical advantage of actigraphy is that recordings of daytime and nighttime activity can be made with minimal disruption to a subject's normal life. Actigraphic measurement has been utilised successfully in group comparisons where stability of the sleep–wake cycle is of particular interest (e.g., Gruber et al., 2000). This capacity for naturalistic measurement makes actigraphy the obvious choice to study underlying circadian abnormalities in BP subjects.

The present study compared a group of euthymic BP I subjects with age and gender matched controls on a number of actigraphically estimated, and subjectively estimated sleep variables over a 5-day period. The study also explored the relationship between sleep variables, and mood ratings to determine whether objective and subjective sleep parameters and/or daily mood ratings can be used to classify subjects as cases or controls.

2. Methods

With ethical approval, a two-group design was employed that compared subjects with a confirmed psychiatric diagnosis of BP I with healthy age- and gender-matched controls on a number of sleep parameters. Cases and controls all gave informed consent to participate in the study. No participant was involved in any other research study.

2.1. Subjects

2.1.1. BP subjects

Subjects with BP were recruited via community mental health teams, and psychiatric outpatient clinics in Glasgow and Renfrewshire. All subjects fulfilled DSM-IV criteria for BP I (American Psychiatric Association, 1995), according to case note information and recorded psychiatric diagnosis. Two researchers (AM & JS) independently confirmed each case diagnosis. Information about the current mood status of subjects was provided by the referring psychiatrist or key mental health worker (e.g., Community Psychiatric Nurse), and independently validated by AM using DSM-IV diagnostic criteria, and the subject's self-reported mood at initial interview. Only those

who were currently remitted (i.e., did not fulfil criteria for an episode) were included.

Subjects who met criteria for a major psychiatric disorder other than BP I and/or those being treated for drug or alcohol problems were excluded. Shift workers were also excluded.

2.1.2. Control subjects

Control subjects were recruited on a voluntary basis via personal and occupational links. Controls were gender- and age-matched with BP subjects. An effort was made also to recruit from diverse occupational and social class groups, and to match on socio-economic factors. Controls participated in a screening interview to exclude current and lifetime history of major psychiatric disorder, and sections of the SCID-I relevant to affective disorders were administered (Structured Clinical Interview for DSM-IV Axis-I Disorders: Clinician version; First et al., 1997). Shift workers were excluded.

2.2. Measures

Background information on psychiatric history and current prescribed medication was recorded for all participants, followed by sleep and any history of sleep problems using a Sleep History Questionnaire (based on Morin, 1993). A number of measures were recorded in both groups over a 5-day period.

Actigraphy was used to obtain objective estimates of sleep over the 5 days. Each subject wore an 'actiwatch' on their non-dominant wrist for five consecutive days and nights. The Actiwatch-R Model AW2 was used (developed by Cambridge Neurotechnology). Raw actigraphic data was then downloaded using the SLEEPWATCH* software programme, and the following parameters calculated: sleep duration, 'sleep efficiency' (the percentage of time in bed spent asleep), sleep onset latency, and wake time during the night.

Subjective measurement of sleep was obtained for the same period using sleep logs (Espie, 1991), which participants completed each morning on rising. The sleep log allowed calculation of the same sleep parameters as measured by the actigraphs.

In order to consider the relationship between sleep and mood all subjects were asked to provide a mood rating twice daily (morning and evening) for the same period, using a simple visual analogue scale (VAS) of mood rated 0–100 (on a 100-mm line), from 0—"most depressed/down I've ever felt" to 100—"most high/manic I've ever felt"; with a 'well' range defined from 35 to 65 mm (Whybrow and Guylia, 1995).

2.3. Analyses

Mean values for actigraphic and subjective parameters of sleep were calculated for each subject across the five nights. In addition, because night-to-night variability in sleep was of interest, the raw score standard deviation over the five nights was also calculated for each variable. This was shown to be a useful measure by Gruber et al. (2000). The two groups were compared on objective and subjective sleep parameters using separate multivariate analyses of variance (MANOVA) for each set of sleep measures. These analyses tested the hypothesis that there are group differences in the sets of sleep measures, while controlling for multiple comparisons. Four MANOVAs were therefore conducted: on averaged actigraphic sleep measures, on night-to-night variability of actigraphic measures, on averaged subjective sleep measures, and on night-to-night variability of subjective sleep measures (using the summary variables described above, and following the protocol of Gruber et al., 2000). In keeping with the assumptions of MANOVA the distribution of subjects responding on all parameters was assessed using the Shapiro–Wilks test of normality. In cases where the distributed data were not normal, natural log transformations were conducted prior to entry of the variable into MANOVA testing. Three actigraphic variables (averaged onset latency, standard deviation of onset latency, and standard deviation of sleep efficiency); and four subjective sleep variables (averaged onset latency, averaged night waking time, standard deviation of onset latency, and standard deviation of night waking time) were log transformed. Following each MANOVA, univariate comparisons were conducted for each sleep measure using one-way analyses of variance (ANOVA). Given that sleep abnormalities may be associated with variations in mood state, the above analyses were then repeated using mean daily mood rating as an additional covariate.

Finally, the relationship between sleep variables, mood ratings and diagnostic status was explored,

using binary backward stepwise logistic regression techniques.

3. Results

3.1. Description of the groups

Thirty-two BP subjects were identified for potential inclusion. Eleven chose not to take part and, of the 21 who consented to participate, two subjects dropped out within the first day of the study. The BP group comprised eight males and 11 females; their ages ranged from 26 to 68 years (mean=47.3, S.D.=10.61). Time since diagnosis ranged from 1 to 35 years (median 15 years) and time since last episode ranged from 1 to 96 months (median 12 months).

Eighteen BP subjects were receiving psychotropic medication, 17 of whom were in receipt of polypharmacy (more than two medications). Ten individuals were on lithium as the only mood stabiliser. Two individuals were taking other mood stabilisers, and three individuals were taking lithium in combination with another mood stabiliser. Eleven subjects were being prescribed antidepressant medication and 10 were receiving antipsychotic medication (two as depots). Two other subjects were receiving night sedation. One subject was not receiving medication.

Nineteen age- and gender-matched controls were recruited. Ages in the control group ranged from 27 to 67 years (mean 45.8, S.D. 10.93). There was no significant difference between BP subjects and controls in age (t=0.42, df 36, P=0.68). Similarly, there was no significant difference between groups in terms of marital status (χ^2=2.9, df 2, P=0.23). Despite efforts to match for occupation, the groups differed in employment status with a smaller proportion of BP subjects in part- or full-time employment (37%) than the control subjects (95%; χ^2=14.2, df 1, P<0.001).

3.2. Comparison of groups on sleep history variables

The Sleep History Questionnaire revealed consistent between group differences. All 19 BP subjects reported longstanding sleep disturbance, compared with only four of the control group (21%). There was an associated difference in how the groups rated the *stability* of their sleep pattern (from month to month, and year to year) on a four-point scale (from 1='very stable' to 4='very unstable') with the BP group reporting less stable patterns (χ^2=21.2; df 3; P<0.0001). A larger proportion of the BP group reported having at some time used sleeping pills (74 vs. 16%; χ^2=14.3; df 1; P<0.001) or alcohol to aid sleep (47 vs. 5%; χ^2=9.4; df 1; P=0.002).

In contrast to these long-term differences, the groups did not differ significantly in their reported recent use of sleeping pills (BP=16%; Controls=11%) or alcohol to aid sleep in the last 4 weeks (BP=11%; Controls=0%). Similarly, there was no difference between their ratings of sleep quality over the last 4 weeks (χ^2=1.69; df 1; P=0.64).

3.3. Comparison of groups on averaged mood measures and mood variability

On the 0–100 VAS, the BP group (mean 43.2; S.D. 10.5) rated themselves as significantly more depressed than the control group (mean 50.0; S.D. 5.1; t=−2.6; df 36; P=0.015). However, the *variability* of subjective mood across the 5 days did not differ significantly (BP mean 1.8, S.D. 0.96; Control mean 1.3, S.D. 0.74; t=1.7; df 36; P=0.10). These data suggest that, even when BP subjects do not fulfil criteria for an episode, they remain depressed compared to controls.

3.4. Comparison of groups on objective and subjective sleep measures

3.4.1. Actigraphic sleep variables

The MANOVA comparing groups on combined averaged actigraphic measures of sleep duration, onset latency, sleep efficiency, and wake time yielded a significant overall group effect (F=3.80; df 4, 33; P=0.012), suggesting that the objective sleep pattern of remitted BP patients differed from healthy controls. Results of separate univariate ANOVAs for each sleep measure, did not find any significant group differences. However, there were trends towards the BP group sleeping longer, taking longer to fall asleep, and sleeping less efficiently than controls (see Table 1).

The MANOVA comparing groups on *variability* in sleep duration, onset latency, sleep efficiency, and wake time did not show a significant difference between the two groups (F=2.45; df 4, 33; P=0.07). However, separate ANOVAs of each measure sug-

Table 1
Means and standard deviations of bipolar group and controls on actigraphic and subjective sleep parameters (averaged and variability measures), and results of univariate analyses

Variable	Bipolar group Mean (S.D.)	Control group Mean (S.D.)	df	Statistic *F*	Sig. (*P*)
Averaged actigraphic measures					
Sleep duration	434.2 (91.7)	387.5 (53.0)	37	3.69	0.063
Onset latency[a]	19.5 (22.1)	8.0 (6.9)	37	3.66	0.064
Sleep efficiency (%)	83.0 (9.2)	86.9 (3.6)	37	3.04	0.090
Night waking time	59.0 (26.0)	49.2 (17.5)	37	1.83	0.184
Standard deviations of actigraphic measures					
Sleep duration	70.0 (39.6)	44.8 (24.6)	37	5.53	0.024*
Onset latency[a]	21.4 (28.7)	8.8 (12.2)	37	3.27	0.079
Sleep efficiency (%)[a]	6.6 (6.3)	4.3 (2.3)	37	.936	0.340
Night waking time	23.6 (15.1)	15.4 (8.1)	37	4.33	0.045*
Averaged subjective measures					
Sleep duration	473.5 (112.9)	411.7 (56.1)	35	4.19	0.048*
Onset latency[a]	40.9 (45.3)	17.3 (11.0)	35	9.01	0.005**
Sleep efficiency (%)	85.7 (8.7)	89.3 (10.3)	35	1.32	0.258
Night waking time[a]	38.8 (40.8)	30.2 (51.0)	35	.362	0.552
Standard deviations of subjective measures					
Sleep duration	91.9 (63.2)	55.6 (31.8)	35	5.19	0.029*
Onset latency[a]	31.2 (54.9)	11.6 (13.5)	35	4.86	0.034*
Sleep efficiency (%)	12.5 (8.4)	6.9 (5.4)	35	6.23	0.018*
Night waking time[a]	37.2 (39.2)	20.8 (25.3)	35	1.00	0.324

[a] Indicates that the variable was transformed for the purpose of analyses. In these cases statistics (*F* values) are the result of analysis conducted on the transformed variable, but means and standard deviations reported were calculated on raw (untransformed) data.

gested statistically significant differences in variability of sleep duration and night waking time, with the BP group showing more variability than healthy controls (see Table 1). The pattern of the results of the MANOVAs and ANOVAs did not change when controlling for mood ratings.

3.4.2. Subjective sleep variables

Results of the MANOVA, which compared groups on averaged sleep duration, onset latency, sleep efficiency and wake time revealed a statistically significant multivariate effect (F=3.18, df 4, 31; P=0.03).[1] Separate univariate ANOVAs confirmed significant differences in sleep duration and onset latency; with the bipolar group sleeping longer and having a longer sleep onset latency. As shown in Table 1, these findings are broadly similar to the trends observed in the objective sleep data.

MANOVA on *variability* in subjective sleep variables did not show an overall difference between the two groups (F=1.79, df 4, 31; P=0.16). However, univariate analyses again suggested statistically significantly greater variability in sleep duration, sleep onset latency, and sleep efficiency in the BP group (Table 1). The pattern of the results of the MANOVAs and ANOVAs did not change when controlling for mood ratings.

3.5. Classification of cases and controls

To assess the ability to distinguish between BP subjects and healthy controls on the basis of sleep and mood measures, direct binary backward stepwise logistic regression analyses were applied with group membership (BP or Control) as the dependent variable, and different combinations of objective and subjective sleep parameters and mood variables as

[1] Two subjects (one from each group) had sleep logs with missing data, and were therefore excluded from the multivariate analysis of subjective sleep variables.

predictor variables. The best model extracted involved a combination of three predictor variables: one actigraphic (variability of sleep duration) with two subjective sleep variables (average sleep duration, and average onset latency). Averaged mood ratings over the 5 days made a non-significant contribution to this model.

A test of the full model with these predictors against a constant only model was significant (χ^2=23.7, df 4; P<0.0001) suggesting that this set of predictors reliably distinguished between BP (correctly assigned 15/19) and control subjects (correctly assigned 17/19), correctly classifying 84% of subjects. Table 2 shows regression coefficients, and odds ratios with 95% confidence intervals (CIs) for each predictor variable.

4. Discussion

This study suggests that the more prolonged, naturalistic measurement offered by actigraphic techniques is a useful method for studying possible sleep–wake cycle abnormalities in this disorder. It is noteworthy that the differences in sleep parameters between BP outpatients and controls identified would almost certainly have been masked by the constraints of laboratory-based methods of sleep measurement. Using actigraphy appears to be a sensitive and convenient way of identifying these abnormalities.

The study suggests that even in the absence of significant affective symptoms, the sleep of BP outpatients is abnormal relative to controls. Bipolar subjects sleep longer than healthy controls, report longer sleep onset latencies and display greater night-to-night variability in a number of objective and subjective sleep parameters. They also report less stable sleep histories, and are more likely to have been prescribed medication or used alcohol to aid their sleep. These findings provide preliminary support for the hypothesis that sleep abnormalities are trait as well as state markers of vulnerability to mood disorders.

The above findings should be interpreted cautiously given the relatively small sample size, the null findings of some of the analyses and the group differences in currently prescribed medication and subjective mood state. Although medication may partly account for the differences in sleep patterns reported, it could equally be argued that the abnormalities in sleep found in BP subjects remained in spite of adequate pharmacological prophylaxis in 18 of the 19 subjects investigated. The groups also differed significantly in their self-reported mood state with the BP group being more depressed than the control group. However, the mean mood ratings for BP subjects in this study fell within the 'normal' range on the VAS and when mood ratings were included as a covariate in the MANOVAs and ANOVAs the pattern of statistically significant differences remained constant. This suggests that the abnormalities in sleep between BP subjects and healthy controls are not simply a function of the BP subjects experiencing higher levels of inter-episode depression. This is further borne out by the results of the logistic regression analysis. The combination of objective variability in sleep duration, subjective latency in sleep onset and subjective sleep duration correctly classified 84% of the sample into cases and controls. Whilst the specificity of this combination of measures in predicting BP in comparison to other mental disorders was not tested by this study, the broad implication of this result is that a relatively simple package of measures

Table 2
Classification of cases and controls using backward stepwise logistic regression

Variables	B	Wald test	df	Sig. (P)	Odds ratio (Exp(B))	95% CI
Actigraphic variability of sleep duration	−0.04	4.4	1	0.04	0.96	0.93 to 0.99
Subjective—average sleep duration	−0.02	3.9	1	0.05	0.98	0.97 to 1.00
Subjective—average onset latency	−0.10	3.9	1	0.05	0.90	0.82 to 0.99
Constant	5.0	0.95	1	0.33		

could be used to support clinical diagnostic information with a high degree of accuracy.

The suggestion of abnormal sleep patterns, and particularly increased instability in the sleep–wake cycle in BP is consistent with the hypothesis that circadian rhythm disruption is crucial in the aetiology of the disorder. This supports the 'social zeitgebers and biological rhythms' model promoted by Ehlers et al. (1988) and further elaborated in the circadian rhythm dysregulation model described by Goodwin and Jamison (1990). At the core of these models is the notion that rhythmic disturbances produce symptoms (possibly through neurotransmitter–neuroendocrine intermediaries), which reinforce or exacerbate abnormal rhythmic processes. Furthermore, genetic factors may shorten the intrinsic period of the circadian locomotor rhythm whilst psychosocial variables may decrease an individual's capacity for entrainment of circadian rhythms. As such the methodology employed in this study, combined with repeated measures of other putative variables may be used to research these models in both BPI and BPII disorders. It may also prove helpful in reducing the misdiagnosis of BP depression (Bowden, 2001).

The findings also have clinical implications, in that they suggest that even during periods of remission, subjects with BP have difficulties in maintaining stable and adaptive sleep–wake cycles. Given the use of self-regulation of social rhythms as a core strategy in interpersonal therapy and cognitive therapy for BP (Ashman et al., 1999; Scott et al., 2001), the use of actigraphy offers an excellent additional method of self-monitoring. It could be used to establish baseline sleep and activity levels and to identify changes following 'social rhythm disrupting' or other stressful life events (Malkoff-Schartz et al., 1998). It could also be used to enhance the effectiveness of self-management techniques employed to prevent prodromal symptoms developing into a full blown relapse (Perry et al., 1999). This approach would be particularly useful in individuals who find that they have difficulty in monitoring their mental state subjectively.

4.1. Limitations

Evidence of differences in the sleep–wake cycle of remitted BP outpatients provided by this study should be regarded as preliminary, and interpreted cautiously given the relatively small sample size. It is possible that with a larger sample some of the trends towards group differences would have reached significance. Nonetheless, it should be noted that the sample size compares favourably with previous studies cited, perhaps reflecting the practical difficulties of working with this subject group.

Previous studies have utilised actigraphy with bipolar subjects (e.g., Klein et al., 1991, 1992); however, no study has specifically validated actigraphic measurement against PSG in this population. It is possible that in an anergic sample of subjects like BP patients actigraphic measures of sleep may overestimate actual sleep, although this would not explain the parallel findings in the subjective sleep data, or the greater night to night variability in BP subjects identified using actigraphy in the present study.

The groups were not matched in terms of medication. The use of medication is likely to have affected the sleep of the BP group to some extent, and many of the BP patients included in the sample had been on medication for prolonged time periods. It is possible that this in itself could have resulted in modifications to the sleep–wake cycle; however, it is unrealistic and unethical to stop the long-term treatment of BP subjects. A strength of this study is its ecological validity, and this adds weight to the implications for everyday clinical practice arising from this project.

The failure to match groups on employment status is recognised as a weakness of the study that may provide an alternative explanation for group differences in variability of the sleep–wake cycle. Difficulties of matching groups on this variable reflect the disorder status of the BP group, since many of them had discontinued work. It would be hard to control for this, since an age-matched healthy control group with the same proportions of unemployment would not necessarily provide a meaningful comparison, and would be unlikely to have the same diversity of occupational histories.

A further limitation relates to the method of measuring current mood status. No formal observer-rated scale was used to evaluate the current mood status of BP subjects; instead judgements about current mood status were based on a combination of clinical opinion (both the researcher's and the psychiatrist's/key mental

health worker's), and the subject's self-rating of mood on the VAS.

Finally, no information on family history of mood disorders was collected from either the BP or the control group. This represents a limitation, particularly with regard to the control group since it has been shown that one fifth of healthy individuals with no current or lifetime diagnosis of psychiatric disorder, but with at least one first-degree relative with mood disorder, show abnormal, depression-like EEG patterns during sleep (Lauer et al., 1995). However, even given this limitation, the study identified differences between the BP and control groups that were largely in the expected direction.

References

American Psychiatric Association, 1995. In: The Diagnostic and Statistical Manual of Mental Disorders, 4th Edition (DSM-IV). American Psychiatric Association, Washington, DC.

Ashman, S.B., Monk, T.H., Kupfer, D.J., Clark, C.H., Myers, F.S., Frank, E., Leibenluft, E., 1999. Relationship between social rhythms and mood in patients with rapid cycling bipolar disorder. Psychiatry Res. 86, 1–8.

Barbini, B., Bertelli, S., Colombo, C., Smeraldi, E., 1996. Sleep loss, a possible factor in augmenting manic episodes. Psychiatry Res. 65, 121–125.

Bowden, C., 2001. Strategies to reduce misdiagnosis of bipolar depression. Psychiatr. Serv. 52, 51–55.

Ehlers, C., Frank, E., Kupfer, D., 1988. Social zeitgebers and biological rhythms: a unified approach to understanding the etiology of depression. Arch. Gen. Psychiatry 45, 948–952.

Espie, C.A., 1991. In: The Psychological Treatment of Insomnia. Wiley, Chichester.

First, M.B., Gibbon, M., Spitzer, R.C., Williams, J.B.W., Benjamin, L.S., 1997. In: Structured Clinical Interview for DSM-IV Axis-I Disorders: (SCID-I), Clinician version. American Psychiatric Press.

Giles, D.E., Jarrett, R.B., Rush, A.J., Biggs, M.M., Roffwarg, H.P., 1993. Prospective assessment of electroencephalographic sleep in remitted major depression. Psychiatry Res. 46, 269–284.

Giles, D.E., Roffwarg, H.P., Rush, A.J., 1990. A cross-sectional study of the effects of depression on REM latency. Biol. Psychiatry 28, 697–704.

Goodwin, F., Jamison, K., 1990. In: Manic Depressive Illness. OUP, Oxford, pp. 552–556.

Gruber, R., Sadeh, A., Raviv, A., 2000. Instability of sleep patterns in children with attention-deficit/hyperactivity disorder. J. Am. Acad. Child Adolesc. Psychiatry 39 (4), 495–501.

Kessler, R.C., Nelson, C.B., McGonagle, K.A., Edlund, M.J., Frank, R.G., Leaf, P.J., 1996. The epidemiology of co-occurring addictive and mental disorders: implications for prevention and service utilisation. Am. J. Orthopsychiatry 66, 17–31.

Klein, E., Lavie, P., Meiraz, R., Sadeh, A., Lenox, R.H., 1992. Increased motor activity and recurrent manic episodes: predictors of relapse in remitted bipolar disorder patients after lithium discontinuation. Biol. Psychiatry 31, 279–284.

Klein, E., Mairaz, R., Pascal, M., Hefez, A., Lavie, P., 1991. Discontinuation of lithium treatment in remitted bipolar patients: relationship between clinical outcome and changes in sleep–wake cycles. J. Nerv. Ment. Dis. 179 (80), 499–501.

Knowles, J.B., Cairns, J., MacLean, A.W., Delva, N., Prowse, A., Waldron, J., Letemendia, F.J., 1986. The sleep of remitted bipolar depressives: comparison with sex and age matched controls. Can. J. Psychiatry 31, 295–298.

Lauer, C.J., Schreiber, W., Holsboer, F., Krieg, J.C., 1995. In quest of identifying vulnerability markers for psychiatric disorders by all-night polysomnography. Arch. Gen. Psychiatry 52, 145–153.

Leibenluft, E., Albert, P.S., Rosenthal, N.E., Wehr, T.A., 1996. Relationship between sleep and mood in patients with rapid-cycling bipolar disorder. Psychiatry Res. 63, 161–168.

Leibenluft, E., Clark, C.H., Myers, F.S., 1995. The reproducability of depressive and hypomanic symptoms across repeated episodes in patients with rapid-cycling bipolar disorder. J. Affect. Disord. 33, 83–88.

Malkoff-Schartz, S., Frank, E., Kupfer, D. et al., 1998. Stressful life events and social rhythm disruption in the onset of manic and depressive episodes of bipolar disorder. Arch. Gen. Psychiatry 55, 707–708.

Morin, C.M., 1993. In: Insomnia: Psychological Assessment and Management. Guildford Press, New York, pp. 195–197.

Nowlin-Finch, N.L., Altshuler, L.L., Szuba, M.P., Mint, J., 1994. Rapid resolution of first episodes of mania: sleep related? J. Clin. Psychiatry 55, 26–29.

Perry, A., Tarrier, N., Morriss, R., McCarthy, E., Limb, K., 1999. Randomised controlled trial of efficacy of teaching patients with bipolar disorder to identify early symptoms of relapse and obtain treatment. Br. Med. J. 318, 149–153.

Rush, A.J., Erman, M.K., Giles, D.E., Schlesser, M.A., Carpenter, G., Vasavada, N., Roffwarg, H.P., 1986. Polysomnographic findings in recently drug free and clinically remitted depressed patients. Arch. Gen. Psychiatry 43, 878–884.

Sadeh, A., Hauri, P.J., Kripke, D.F., Lavie, P., 1995. The role of actigraphy in the evaluation of sleep disorders. Sleep 18, 288–302.

Sadeh, A., Alster, J., Urbach, D., Lavie, P., 1989. Actigraphically based automatic bedtime sleep–wake scoring: validity and clinical applications. J. Ambul. Monit. 2, 209–216.

Scott, J., Garland, A., Moorhead, S., 2001. A pilot study of cognitive therapy for bipolar disorders. Psychol. Med. 31, 459–467.

Sitaram, N., Nurnberger, J.I., Gershon, E.S., Gillin, J.C., 1982. Cholinergic regulation of mood and REM sleep: potential model

and marker of vulnerability to affective disorder. Am. J. Psychiatry 139 (5), 571–576.

Wehr, T.A., Sack, D.A., Duncan, W.C., Mendelson, W.B., Rosenthal, N.E., Gillin, J.C., Goodwin, F.K., 1985. Sleep and circadian rhythms in affective patients isolated from external time cues. Psychiatry Res. 15, 327–339.

Whybrow, P.C., Guylia, L., 1995. The chronorecord: tracking bipolar patterns and treatment effects. In: Abstracts for the 2nd International Conference on New Directions in Affective Disorders, Jerusalem.

British Journal of Clinical Psychology (2004), 43, 409–420

www.bps.org.uk

Development and preliminary validation of the Glasgow Content of Thoughts Inventory (GCTI): A new measure for the assessment of pre-sleep cognitive activity

Karen J. Harvey and Colin A. Espie*

Department of Psychological Medicine, University of Glasgow, UK

Objective. To develop a self-report measure (the Glasgow Content of Thoughts Inventory [GCTI]) for the assessment of pre-sleep cognitive activity in adults with sleep-onset insomnia.

Design. A psychometric, scale development approach was used.

Method. Over three consecutive nights, 12 people with insomnia provided 'live' audio-recordings of pre-sleep thought content, which were used to generate an item pool. The results were compared to the content and categorical structure of pre-sleep cognitive activity identified by Wicklow and Espie (2000), and commonalities in thought content were used to generate a draft scale. Following further piloting, a 25-item scale was developed and administered to two groups (29 people with insomnia and 29 good sleepers), along with other self-report measures, objective (actigraphic recordings) and subjective (diary) sleep indices, and results analysed to evaluate the psychometric properties of the scale.

Results. The GCTI demonstrated evidence of construct validity, successfully discriminated between individuals with insomnia and good sleepers, and was significantly correlated with existing measures of sleep disturbance. A score of 42 yielded a sensitivity of 100% and specificity of 83%. The GCTI demonstrated good test–retest reliability (ICC = .88) and internal consistency (α = .87).

Conclusions. The GCTI appears to be a valid and reliable instrument for use with patients with sleep-onset insomnia.

Insomnia is a heterogeneous complaint reflecting reduced quality, duration or efficiency of sleep (Morin *et al.*, 1999). Primary insomnia is reported by up to 30% of

* *Correspondence should be addressed to Colin Espie, Department of Psychological Medicine, University of Glasgow, Academic Centre. Gartnavel Royal Hospital, 1055 Great Western Road, Glasgow G12 0XH Scotland, UK (e-mail: c.espie@clinmed.gla.ac.uk)*

the population, with prevalence of chronic insomnia estimated at 10–15% (Ohayon, Caulet, & Guilleminault, 1997).

In a survey of causal attributions of insomnia, Lichstein and Rosenthal (1980) found that individuals with insomnia were 10 times more likely to blame excessive cognitive rather than somatic activity for their sleep disturbance. Experimental work by Gross and Borkovec (1982) demonstrated that increased sleep-onset latency (SOL) could be induced in good sleepers by increasing cognitive arousal (telling participants that they had to give a presentation on wakening), with the effect occurring independently of physiological activity (heart rate and skin conductance). Haynes, Adams, and Franzen (1981) obtained similar results, demonstrating that estimates of SOL increased for both objective (polygraphic) and subjective (sleep diary) measures of sleep disturbance following cognitive arousal. Espie, Brooks, and Lindsay (1989) also found that 'mental overactivity' was the most common causal attribution made by clinic-presenting patients with chronic insomnia.

It has been known for some time that intrusive thinking may lead to increased SOL (Van Egeren, Haynes, Franzen, & Hamilton, 1983). However, there is a limited literature on the content of these thoughts. Watts, Coyle, and East (1994) differentiated people with insomnia into either 'worrying insomniacs', whose pre-sleep cognitions focused on various topics (e.g., trivial topics, plans, work, family and recent concerns, bodily sensations) or 'non-worrying insomniacs', whose pre-sleep cognitions focused specifically on concerns about sleep loss. Harvey (2000) analysed focus of attention, content and process characteristics in good sleepers and people with insomnia. The latter groups' attention focused on solving problems, worries, concerns, reviewing events of the day, thinking about their sleep pattern, and environmental noises. Ratings of cognitive interference, estimated duration of cognitive activity and intrusiveness (preoccupation and effect on sleep) were higher for those with insomnia than for good sleepers. Wicklow and Espie (2000) used voice-activated tape-recorders to obtain 'live' recordings of the pre-sleep thoughts of individuals with insomnia. They identified eight thought categories reflecting three factors: active problem-solving, present state-monitoring and environmental reactivity. Subjective measurement (sleep diary) and objective measurement (wrist actigraphic recordings—a device which, based on the amount of movement, can differentiate between sleep and wake periods) of SOL were also taken. Only the latter was correlated significantly with pre-sleep cognitive activity. The authors concluded that the use of 'live' recordings of pre-sleep cognitive content merited further investigation, and that their putative categorical and factorial structure of pre-sleep thinking might be used to develop a scale to assess the content of pre-sleep cognitions.

There are four existing self-report measures relevant to the pre-sleep mental state: the Pre-Sleep Arousal Scale (PSAS; Nicassio, Mendlowitz, Fussell, Petras, 1985); the Sleep Disturbance Questionnaire (SDQ; Espie *et al.*, 1989); the Dysfunctional Beliefs and Attitudes About Sleep Scale (DBAS; Morin, 1993; Morin, Stone, Trinkle, Mercer, & Remsberg 1993); and the Self-Statement Test: 60+ (SST; Fichten *et al.* 1995, 1998). The DBAS also has a 10-item short-form version (DBAS-10; Espie, Inglis, Harvey, & Tessier, 2000). Each of these was developed using appropriate scale design methods and has adequate psychometric properties. However, they are unsuitable for descriptive quantification of pre-sleep thinking because they provide more global assessment of cognitive dysfunction and arousal, and fail to reflect the specific content of pre-sleep cognitive activity such as identified by Harvey (2000) and Wicklow and Espie (2000).

The PSAS is essentially a measure to quantify arousal level and to compare somatic

with cognitive arousal state. The SDQ is an attributional measure and, like the PSAS, is insufficiently detailed to describe thought content. The DBAS scales come closer to reflecting pre-sleep thought content, but DBAS items are at the somewhat 'deeper' level of belief/attitude/schema (e.g., 'After a poor night's sleep I know that it will interfere with my daily activities the next day'). Although the SST assesses pre-sleep cognitions, it has only been validated for use with older adults and is not specific to pre-sleep mentation, but to 'nocturnal wake times'. Furthermore, items for all four measures were obtained through retrospective analysis (e.g., thought records, diaries, semi-structured interviews), which may be subject to low specificity of thought content, response limitation due to pre-defined questions, and omissions or distortions through forgetting.

The development of a scale that can reliably measure pre-sleep thoughts in terms of their nature, content and frequency of interference upon sleep would be beneficial, not only in assessment and treatment planning, but also in monitoring and evaluating treatment mechanisms and outcomes. Our aim, therefore, was to develop and evaluate the psychometric properties of a self-report measure to assess the content of pre-sleep thoughts in adults with insomnia.

Method and results

Stage 1: Derivation of an item pool

Rationale

Wicklow and Espie (2000) used 'live' audiotape recordings of the pre-sleep cognitive activity of adults with insomnia to gather data. It was felt that replication of this approach would provide the best available item pool, and permit validity analysis by comparing results with their findings.

Participants: scale development group

Participants were recruited using the university e-mail service, requesting those with 'current sleep problems interested in taking part in sleep research' to contact the researcher. Participants had to be 16 to 65-years-old; meet diagnostic criteria for significant problems falling asleep— specifically a minimum SOL of 30 min occurring at least four out of seven nights with or without disruption to other sleep variables (International Classification of Sleep Disorders—Revised [ICSD-R], 1997); and obtain a Pittsburgh Sleep Quality Index Score (PSQI; Buysse, Reynolds, Monk, Berman, & Kupfer, 1989) of 6 or above. The PSQI adequately discriminates good sleepers from poor sleepers using this cut-off (sensitivity = 89.6%, specificity = 86.5%; Buysse *et al.*, 1989). The sensitivity of a scale is the probability that an individual with the condition will be classified correctly as having the condition; specificity is the probability that a person without the condition will be classified as not having it (Fleiss, 1981).

Participants were excluded if they:

(1) were receiving psychological treatment for sleep difficulties;
(2) were currently taking, or had in the past three months taken, medication known to affect sleep;
(3) were suffering from a psychological disorder;
(4) were suffering from a chronic medical condition known to impact on sleep; or
(5) scored 20 or above on the Beck Depression Inventory (BDI; Beck, Ward,

Mendelson, Mock, & Erbaugh, 1961) to exclude individuals with insomnia with significant suspected clinical depression (*cf.* Espie *et al.*, 2000). A screening interview was arranged in which participants completed the Sleep History Questionnaire (SHQ; Morin, 1993), the Penn State Worry Questionnaire (PSWQ; Meyer, Miller, Metzger, & Borkovec, 1990), the State-Trait Anxiety Inventory (STAI; Spielberger, Gorusch, & Lushene, 1970), the PSQI and the BDI.

Of 19 respondents, seven were excluded (two failed to meet inclusion criteria, one was excluded through medication use, one reported sleep difficulties due to nightmares, two retracted from the study, and one was excluded due to missing data). Twelve people with insomnia (nine females, three males) with a mean age of 26 years (average duration of sleep disturbance = 12.0 yrs, *SD* 4.6 yrs) participated in Stage 1. These individuals were excluded from subsequent stages.

Data gathering procedure

Over three consecutive weekday nights, participants used voice-activated tape-recorders (Sony Cassette Recorder TCS–580 V) placed at their bedside to record their thoughts as they tried to sleep. Wicklow and Espie (2000) reported this procedure as non-intrusive, with no evidence of a 'first night effect'. Participants were instructed to say aloud whatever was going through their mind, whenever they wanted to, when having difficulty sleeping. To minimize performance anxiety and allow participants to speak freely, no further specific instructions were given.

Subjective and objective measures of SOL were taken to permit subsequent comparison with the insomniac group recruited to the field-testing phase (Stage 3). Participants completed a sleep diary each morning. This is a well-documented, retrospective, self-report instrument. Espie (1991) cites evidence demonstrating high test–retest reliability of diary measurement (r = .93 for poor sleepers). To provide an objective estimate of sleep, wrist actigraph recordings were obtained over three nights using the Actiwatch-R Model AW2® (Cambridge Neurotechnology Ltd). The actigraph records 'sleep' or 'wakefulness' by accumulating activity counts during a specified time interval. An epoch of 1 min was selected—the recommended interval for accurate sleep analysis (American Sleep Disorders Association [ASDA], Report 1995]. The actigraph has an event marker that participants were instructed to press when they went to bed and when they got up. The main actigraphic parameters of sleep correlate strongly with polysomnography (Kripke, Mullaney, Messin, & Wybourney, 1978). Hauri and Wisbey (1992) did report accuracy difficulties for actigraph recordings; however, error was only half that normally associated with sleep diaries.

Content analysis of pre-sleep thoughts

Data were available for at least two nights from all participants and totalled to 27 subject nights. These data were analysed by the first author according to the procedure for content analysis adopted by Wicklow and Espie (2000). First, audiotape-recorded material was transcribed; secondly, transcripts were segmented into single ideas or statements; thirdly, segments were allocated to one of eight thought categories; and fourthly, an independent reliability check on the allocation of thought segments to categories was conducted of four participants' (33%) transcripts.

Content validity

This procedure generated 423 thought segments over the 27 nights (M per night = 15.7). This is comparable to Wicklow and Espie (2000), who obtained 1,090 thought

segments over 63 nights (*M* per night = 17.3). Distribution per category was also similar, with 'rehearsal/planning/problem-solving' accounting for the majority of thought segments (32.4%, *n* = 137), followed by 'sleep and its consequences' (20.8%, *n* = 88), 'autonomic experiences' (14.9%, *n* = 63), 'reflection on quality of thoughts' (11.3%, *n* = 48), 'rising from bed' (7.3%, *n* = 31), 'arousal status' (6.9%, *n* = 29), 'external noise' (5.0%, *n* = 21) and 'procedural factors' (1.4%, *n* = 6). Concordance was satisfactory, with 95% agreement between independent raters.

Stage 2: Refinement of item pool and development of scale

Refinement of the item pool and development of the scale followed a six stage process:

(1) As described above, each thought segment was allocated to one of eight category themes. From visual inspection and discussion between the authors, each category was broken down into subcategories to reflect faithfully the range of thought content. Each specific thought segment was then allocated to a subcategory, thereby forming potential scale items (e.g., thoughts relating to 'the length of time it is taking to fall asleep' were a subcategory of 'sleep and its consequences'). This procedure, therefore, produced eight sets of items; however, only seven sets were retained because 'procedural thoughts' (e.g. 'Is that tape-recorder on?') were excluded from the analysis. These are unsuitable for inclusion in a scale, and comprised less than 2% of the total (see above).
(2) An identical allocation procedure was repeated on a random sample of data (seven participants; 16 subject nights; 345 thought segments) from the Wicklow and Espie (2000) study.
(3) Data from these two analyses were then compared for commonalities across the top five most frequently endorsed items within each category theme. This process yielded 34 potential scale items which had been frequently reported by participants from both studies, and which accounted for the majority of the total number of thoughts reported (category range = 77.3–100%).
(4) These items were reviewed by the first author and an expert clinician, and reworded as statements. A draft scale was then produced containing the 34 items ordered randomly. Instructions were added that requested respondents to indicate, using a 4-point response, how often (over the past seven nights) each thought had kept them awake. A 4-point scale (1 = 'never', 2 = 'sometimes', 3 = 'often', 4 = 'always') was selected.
(5) The draft scale was administered to a sample of people with insomnia from the scale development group (*n* = 6), and a sample of normal sleepers (*n* = 6). A semi-structured response sheet was provided for comment on what they thought the scale was measuring, their understanding of how to complete the scale, ease of completion, and views on individual items (e.g., overlap, possible omissions). Informal interviews were conducted to expand on responses.
(6) Following feedback from this process, six items were dropped because they did not discriminate, the wording of four items was amended, and five items were reorganized into two items.

These procedures resulted in the final 25-item scale, the Glasgow Content of Thoughts Inventory (GCTI), presented in the Appendix.

Stage 3: Field testing and psychometric evaluation of the GCTI

To examine the psychometric properties of the GCTI, two groups (people with insomnia and good sleepers) were recruited.

Participants: validation phase

Participants were again recruited by university e-mail. Over 150 responses from individuals reporting insomnia were received. From this pool, 59 respondents were screened using the measures described earlier (SHQ, PSQI, STAI, PSWQ, BDI) of whom 44 (75%) satisfied our inclusion criteria for insomnia (as before). Fifteen met exclusion criteria and took no further part in the study. A lower e-mail response was obtained for good sleepers (n = 45), 14 of whom were excluded due to failure to return screening measures, and two due to missing data. Good sleepers did not meet ICSD-R criteria for insomnia, and scored less than 6 on the PSQI.

The final sample comprised 29 adults with insomnia (24 females, 5 males) and 29 good sleepers (19 females, 10 males). Following checks for skewness and kurtosis, groups were compared on participant characteristics and screening results using independent sample t tests or Mann–Whitney tests as appropriate. The insomnia group was also compared to the scale development group. Due to faulty equipment, missing actiwatch data were incurred for three participants (one from the scale development group, and two people with insomnia from the validation phase). Four participants also failed to complete sleep diaries adequately (one from the scale development group, one good sleeper and two people with insomnia from the validation phase). Table 1 presents summary data for each group.

Results suggest that the insomnia group did not differ from the scale development group on age, $z = 1.33$, $p = .195$, or gender, $\chi^2(1) = 0.35$, Fisher's exact $p = .719$. There were no significant differences between the insomnia and scale development group on problem duration, $z = -.26$, $p = .810$; the PSQI, $t(39) = 1.07$, $p = .293$; the BDI, $z = -.61$, $p = .543$; the PSWQ, $t(39) = 0.45$, $p = .659$; the STAI–state anxiety, $t(39) = 0.72$, $p = .473$; the STAI–trait anxiety, $t(39) = 0.15$, $p = .988$; subjective SOL, $t(33) = 0.46$, $p = .648$; or objective SOL, $z = -.98$, $p = .339$.

Similarly, no significant differences emerged between good sleepers and people with insomnia for age, $t(56) = .473$, $p = .638$, or gender, $\chi^2(1) = 2.248$, $p = .134$. However, good sleepers scored significantly lower than people with insomnia on the PSQI, $t(46) = 12.70$, $p < .001$; the BDI, $z = -3.89$, $p < .001$; the PSWQ, $t(49) = 2.269$, $p = .028$; the STAI–state anxiety, $t(56) = 2.98$, $p = .004$; and the STAI–trait anxiety, $t(56) = 3.59$, $p = .001$. Subjective SOL was around 60 min in insomnia compared with 14 min in good sleepers, $z = -5.61$, $p < .001$. Objective SOL was estimated at around 30 min by actigraphy in the insomnia group compared with 13 min in good sleepers, $t(54) = 3.057$, $p = .003$.

Table 1 also presents comparative characteristics for Espie *et al.*'s (2000) clinic-presenting sample of 178 patients with insomnia. With the exception of age, the mean scores for both the scale development and insomnia group were close to those of Espie *et al*. It can be concluded, therefore, that the people with insomnia in this study are similar to clinic patients with respect to clinical and sleep characteristics.

Measures and procedure

To examine the psychometric properties of the GCTI, participants completed the GCTI at home along with the cognitive subscale of the PSAS (PSAS-cog; eight items, $\alpha = .76$), the DBAS–10 ($\alpha = .69$) and the SDQ (12 items yielding four factors; SDQ-F2 reflects

Table 1. Participant characteristics and scores on screening measures by group. Comparison is also made with data from a clinic sample

	Scale development group (n = 12)			Insomnia (n = 29)			Good sleepers (n = 29)			Espie *et al.* (2000) (n = 178)	
	M	*SD*	Med.	*M*	*SD*	Med.	*M*	*SD*	Med.	*M*	*SD*
Age	26.0	4.6	26.0	24.8	6.9	23.0	25.5	3.8	25.0	49.8	17.9
Problem Duration (yrs)	6.6	4.8	4.5	6.9	5.9	5.5	–	–	–	–	–
PSQI	12.1	2.5	–	11.1	2.7	–	3.5	1.7	–	–	–
BDI	10.8	6.9	9.0	9.4	7.4	9.0	2.8	2.8	2.0	12.2	9.3
PSWQ	8.8	15.1	–	46.5	14.9	–	39.0	9.8	–	47.7	15.0
STAI-S	36.	16.6	–	39.4	11.1	–	31.7	8.6	–36.7	13.1	
STAI-T	43.4	15.2	–	43.5	11.6	–	33.8	8.8	–	43.5	12.8
Subjective estimate of SOL (diary)	57.3	37.1[a]	55.0	64.7	40.8[b]	55.0	13.8	12.5[c]	10.0	61.5	55.1
Objective estimate of SOL (actigraphy)	24.8	30.0	–	32.1	26.9[b]	–	12.7	20.2	–	–	–

Notes: [a]Missing data n = 11; [b]Missing data n = 27; [c]Missing data n = 28.
Med. = median; PSQI = Pittsburgh Sleep Quality Index Score; BDI = Beck Depression Inventory; PSWQ = Penn State Worry Questionnaire; STAI-S = State Trait Anxiety Inventory – state anxiety scale; STAI-T = State-Trait Anxiety Inventory – trait anxiety scale; SOL = sleep-onset latency.

cognitive arousal; α = .82; Espie *et al.*, 2000). Participants kept sleep diaries and wrist actigraph recordings were taken for three consecutive nights (as for the scale development group).

Construct validity The PSAS-cog is known to correlate with measures of affect and sleep-onset, a finding congruent with the theory that increased mental arousal contributes to sleep disturbance (Espie, 2002). The correlation between the GCTI and PSAS-cog was computed to provide preliminary evidence of the GCTI's construct validity (n = 58). A strong positive correlation was obtained (r = .879, p < .001), confirming that frequency of cognitive intrusions is associated with pre-sleep mental arousal.

Concurrent validity The relationship between GCTI and other measures of sleep disturbance was examined for the SDQ-F2 and DBAS–10 (n = 58). The GCTI correlated significantly with both measures (SDQ-F2: r = .815, p < .001; DBAS–10: r = .732, p < .001). Similarly, relationships between the GCTI and sleep diary (n = 55) and actigraph recordings of SOL (n = 56) were investigated. Diary estimates were more strongly associated with the GCTI (r = .650) than actigraph estimates of SOL (r = .484), although both were significant (ps < .001).

Discriminant validity The ability of the GCTI to discriminate people with insomnia (n = 29) from poor sleepers (n = 29) was investigated. Those with insomnia scored higher than good sleepers (M = 58.0, SD = 10.08 vs. M =/=, 35.2 SD = 8.37); t(56) = 9.40, p < .001. Figure 1 illustrates GCTI scores for both groups. As distributional data indicate, there is limited overlap between the highest scoring good sleepers and the lowest scoring individuals with insomnia. Scores for good sleepers also appear to be more closely grouped (as indicated by the box highlighting the interquartile range) than those obtained for people with insomnia.

Sensitivity and specificity Analysis of GCTI sensitivity data suggested that a score of 42 correctly identified 100% (i.e., 29) of people with insomnia, and correctly identified 83% (i.e., 24 out of 29) of good sleepers (i.e., specificity). Using this cut-off, the total sample was split into two groups: those experiencing 'high frequency pre-sleep cognitive events' (n = 34), versus those with 'low frequency pre-sleep cognitive events' (n = 24). Differences between groups on the PSAS-cog, SDQ-F2 and DBAS-10 were analysed. Participants with high frequency pre-sleep cognitive events scored significantly higher on all three measures: PSAS-cog (M = 26.0, SD = 6.0 vs. M =/=, 13.2 SD =/, t(56) 5.1) = 8.62, p < .001; DBAS-10 (M = 53.3, SD = 15.2 vs. M =/=, 32.0 SD = 12.0), t(56) = 5.70, p < .001; and SDQ-F2 (M = 12.8, SD = 1.8) vs. M =/=, 6.7 SD = 2.2), t(56) = 11.54, p < .001. Participants with high frequency pre-sleep cognitive events also scored higher on subjective and objective measures of SOL (both ps < .001).

Test–retest reliability The GCTI was administered a second time, three weeks later. Test–retest scores were available for 26 people with insomnia (89.7%). Test–retest reliability of the GCTI using the intra-class correlation coefficient appeared highly satisfactory (ICC = .878, p < .001).

Internal consistency

Cronbach's alpha was calculated at .870 using the insomnia group (n = 29). Guttman split-half reliability was similar (.851). Item-deletion alphas give an indication of the

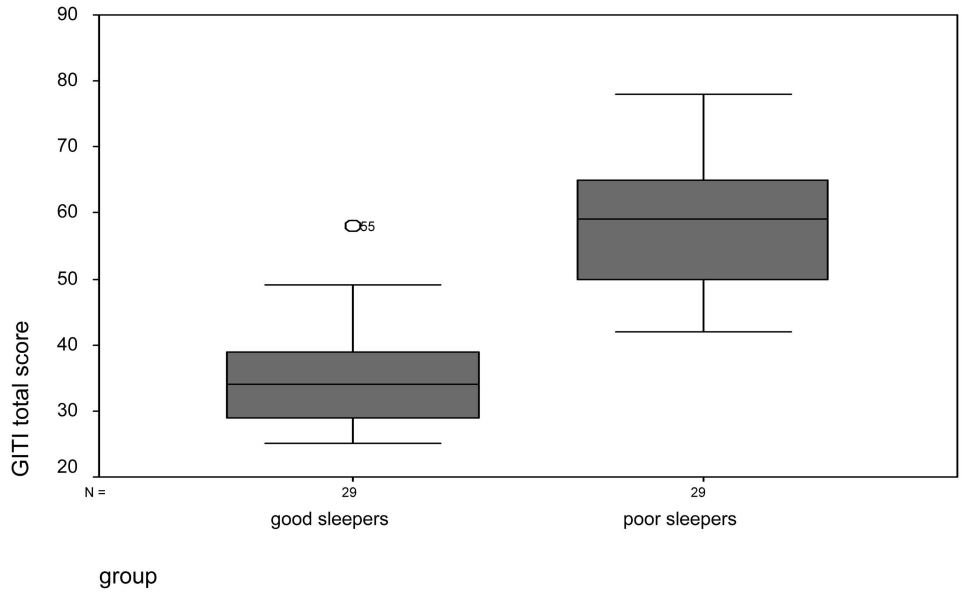


Figure 1. Box and whiskers plot demonstrating the ability of the GCTI to discriminate between good sleepers and people with insomnia.

stability of a measure when items are systematically eliminated. These remained high, with little variation between values ($M\ \alpha$ = .87, range = .855–.874). A criterion of .80 is usually considered acceptable (Nunnally & Bernstein, 1994), therefore these results appear satisfactory. The corrected item–total correlation is the correlation of a single item with the sum of all other items (Nunnally & Bernstein, 1994) and, ideally, will be modest (approximately r = .4) to ensure that a range of items is retained. The mean corrected item–total correlation was .43 (range = .12–.73).

Discussion

The purpose of this study was to develop a psychometrically robust self-report measure for the assessment of pre-sleep thought content in insomnia. We suggest that our derived measure, the Glasgow Content of Thoughts Inventory, offers considerable potential in quantifying the nature and frequency of such mentation. Items for the GCTI arose directly from the prospective experience of adults with insomnia and were validated against our previous work using similar methodology (Wicklow & Espie, 2000). The items also appear to reflect other research findings highlighting that pre-sleep cognitive events typically focus on solving problems, worries/concerns, reviewing the day, and thinking aboutsleep (Harvey, 2000; Van Egeren *et al.*, 1983; Watts *et al.*, 1994).

A test is considered to be measuring the same attribute as existing measures if intercorrelation is greater than .4 (Streiner & Norman, 1995). Field testing of the GCTI using people with insomnia and good sleepers produced preliminary evidence of the GCTI's construct validity, as it was strongly correlated with the cognitive subscale of

the PSAS, an established measure in the domain of general mental arousal. The GCTI also demonstrated acceptable association with the cognitive arousal subscale of the SDQ and the 10-item short form of the DBAS. It should be observed, however, that the GCTI probes *specific thought content* within the general domain of cognitive excitation and this is a unique aspect of this new measure.

Significant differences in GCTI total scores were obtained between people with insomnia and good sleepers, supporting the GCTI's ability to discriminate between these groups, and exploration of sensitivity and specificity revealed that a cut-off score of 42 yielded perfect sensitivity (100%) and good specificity (83%). Again this requires replication. We would suggest that differentiation of people with insomnia from non-complaining poor sleepers would also be valuable because it may be that the pre-sleep mental and emotional experience is critical to the complaint of insomnia. We hope in future, with much larger samples, to conduct principal component analyses of the GCTI that might offer quantification of the factorial structure of the content of pre-sleep thinking. This in turn would permit investigation of any specific differences in the nature of pre-sleep thought content, as well as frequency of occurrence, between people with insomnia and good sleepers.

We have reported a stronger association of pre-sleep thinking with subjective estimates of sleep than with objective (actigraphic) sleep. This is interesting because it tends to be the subjective phenomenology of the sleep experience that acts as the 'driver' of concern and help-seeking behaviour. This association of cognitive events with insomnia should not be taken to imply a causal mechanism. Nevertheless, it highlights the importance of pursuing cognitive models of insomnia where failure to de-arouse in bed and/or hyperarousal may inhibit sleep-onset (Espie, 2002).

The reliability of the GCTI, in terms of internal consistency (α = .87), item–total correlation values (M = .43) and test–retest reliability (r = .88) appear very acceptable. Although the utility of the GCTI in clinical settings was not explored, the GCTI's simple format suggests that it is easily understood by recipients, and quick to administer (less than 5 min) and to score. At this stage we see the GCTI as an aid to the clinical interview process, raising issues for discussion and perhaps identifying goals for intervention. We hope also that the GCTI will be used in both experimental and clinical psychological research on insomnia because critical pathways in the acquisition, persistence and treatment of insomnia remain poorly understood (Espie, 2002).

References

American Sleep Disorders Association (1997). *International classification of sleep disorders—revised: Diagnostic and coding manual*. Rochester, MA: Author.

American Sleep Disorders Association (ASDA) Report (1995). Practice parameters for the use of actigraphy in the clinical assessment of sleep disorders. *Sleep, 18*, 285–287.

Beck, A. T., Ward, C. H., Mendelson, M., Mock, J., & Erbaugh, J. (1961). An inventory for measuring depression. *Archives of General Psychiatry, 4*, 561–571.

Buysse, D. J., Reynolds, C. F., Monk, T. H., Berman, S. R., & Kupfer, D. J. (1989). The Pittsburgh Sleep Quality Index: A new instrument for psychiatric practice and research. *Psychiatric Research, 28*, 193–213.

Comrey, A. L. & Lee, H. B. (1992). *A first course in factor analysis* (2nd ed.). Hillsdale, NJ: Erlbaum.

Espie, C. A. (1991). *The psychological treatment of insomnia*. New York: Wiley.

Espie, C. A. (2002). Conceptual issues in the development, persistence and treatment of sleep disorder in adults. *Annual Review of Psychology 53*, 215–243.

Espie, C. A., Brooks, D. N., & Lindsay, W. R. (1989). An evaluation of tailored psychological treatment of insomnia. *Journal of Behaviour Therapy & Experimental Psychiatry, 20*, 143–153.

Espie, C. A., Inglis, S. J., Harvey, L., & Tessier, S. (2000). Insomniacs' attributions: Psychometric properties of the Dysfunctional Beliefs and Attitudes about Sleep scale and the Sleep Disturbance Questionnaire. *Journal of Psychosomatic Research, 48,* 141–148.

Fichten, C. S., Creti, L., Amsel, R., Brender, W., Weinstein, N., & Libman, E. (1995). Poor sleepers who do not complain of insomnia: Myths and realities about psychological and lifestyle characteristics of older good and poor sleepers. *Journal of Behavioural Medicine, 18*, 189–223.

Fichten, C. S., Libman, E., Creti, L., Amsel, R., Tagalakis, V., & Brender, W. (1998). Thoughts during awake times in older good and poor sleepers: The Self-Statement Test:60+. *Cognitive Therapy & Research, 22*, 1–20.

Fleiss, J. (1981). *Statistical methods for rates and proportions*. New York: Wiley

Gross, R. T., & Borkovec, T. D. (1982). Effects of cognitive intrusion manipulation on the sleep-onset latency of good sleepers. *Behaviour Therapy, 13*, 117–124.

Harvey, A. (2000). Pre-sleep cognitive activity: A comparison of sleep-onset insomniacs and good sleepers. *British Journal of Clinical Psychology, 39*, 275–286.

Hauri, P. J., & Wisbey, J. (1992). Wrist actigraphy in insomnia. *Sleep, 15*, 293–301.

Haynes, S. N., Adams, A., & Franzen, M. (1981). The effects of pre-sleep stress on sleep-onset insomnia. *Journal of Abnormal Psychology, 90*, 601–606.

Kripke, D. F., Mullaney, D. J., Messin, S., & Wybourney, V. G. (1978). Wrist actigraphic measures of sleep and rhythms. *Electroencephalogry and Clinical Neurophysiology, 44*, 674–676.

Lichstein, K. L., & Rosenthal, T. L. (1980). Insomniacs' perception of cognitive versus somatic determinance of sleep disturbance. *Journal of Abnormal Psychology, 89*, 105–107.

Meyer, T. J., Miller, M. L., Metzger, R. L., & Borkovec, T. D. (1990). Development and validation of the Penn State Worry Questionnaire. *Behaviour Research & Therapy, 28*, 487–495.

Morin, C. M. (1993). *Insomnia: Psychological assessment and management*. New York: Guilford Press.

Morin, C. M., Hauri, P. J., Espie, C. A., Spielman, A., Buysse, D. J., & Bootzin, R. (1999). Non-pharmacologic treatment of chronic insomnia: An American Academy of Sleep Medicine Review. *Sleep, 22*, 1134–1155.

Morin, C. M., Stone, J., Trinkle, D., Mercer, J., & Remsberg, S. (1993). Dysfunctional beliefs and attitude about sleep among older adults with and without insomnia complaints. *Psychology and Aging, 8*, 463–467.

Nicassio, P. M., Mendlowitz, D. R., Fussell, J. J., & Petras, L. (1985). The phenomenology of the pre-sleep state: The development of the pre-sleep arousal scale. *Behaviour Research & Therapy, 23*, 263–271.

Nunnally, J. C., & Bernstein, I. H. (1994). *Psychometric theory* (3rd ed.). New York McGraw-Hill Series in Psychology.

Ohayon, M. M., Caulet, M., & Guilleminault, C. (1997). How a general population perceives its sleep and how this relates to the complaint of insomnia. *Sleep, 20*, 715–723.

Spielberger, C. D., Gorusch, R. L., & Lushene, R. E. (1970). *Manual for the State-Trait Anxiety Inventory*. Palo Alto, CA: Consulting Psychologists Press.

Streiner, D. L., & Norman, G. R. (1995). *Health measurement scales: A practical guide to their development and use* (2nd ed.). Oxford: Oxford University Press.

Tabachnick, B. G., & Fidell, L. S. (1996). *Using multivariate statistics* (3rd ed.) New York: HarperCollins.

Van Egeren, L., Haynes, S. N., Franzen, M., & Hamilton, J. (1983). Pre-sleep cognitions and attributions in sleep-onset insomnia. *Journal of Behavioural Medicine, 6*, 217–232.

Watts, F. N., Coyle, K., & East, M. P. (1994). The contribution of worry to insomnia. *British Journal of Clinical Psychology, 33*, 211–220.

Wicklow, A., & Espie, C. A. (2000). Intrusive thoughts and their relationship to actigraphic measurement of sleep: Towards a cognitive model of insomnia. *Behaviour Research & Therapy, 38*, 679–693.

Received 16 September 2002; revised version received 11 August 2003

Appendix: The Glasgow Content of Thoughts Inventory

Here are some thoughts that people have when they can't sleep. Please indicate by placing a tick in the appropriate box how often over the past 7 nights the following thoughts have kept you awake.

		Never	Sometimes	Often	Always
1.	Things in the future				
2.	How tired/sleepy you feel				
3.	Things that happened during the day				
4.	How nervous/anxious you feel				
5.	How mentally awake you feel				
6.	Checking the time				
7.	Trivial things				
8.	How you can't stop your mind from racing				
9.	How long you've been awake				
10.	Your health				
11.	Ways you can get to sleep				
12.	Things you have to do tomorrow				
13.	How hot/cold you feel				
14.	Your work/responsibilities				
15.	How frustrated/annoyed you feel				
16.	How light/dark the room is				
17.	Noises you hear				
18.	Being awake all night				
19.	Pictures of things in your mind				
20.	The effects of not sleeping well				
21.	Your personal life				
22.	How thinking too much is the problem				
23.	Things in your past				
24.	How bad you are at sleeping				
25.	Things to do to help you sleep				

Derivation of Research Diagnostic Criteria for Insomnia: Report of an American Academy of Sleep Medicine Work Group

Jack D. Edinger[1,2], Chair; Work Group Members: Michael H. Bonnet[3]; Richard R. Bootzin[4]; Karl Doghramji[5]; Cynthia M. Dorsey[6]; Colin A. Espie[7]; Andrew O. Jamieson[8]; W. Vaughn McCall[9]; Charles M. Morin[10]; Edward J. Stepanski[11]

[1]VA & [2]Duke University Medical Centers, Durham, NC; [3]VA Medical Center, Dayton, OH; [4]University of Arizona, Tucson, AZ; [5]Thomas Jefferson University, Philadelphia, PA; [6]McLean Hospital, Belmont, MA; [7]University of Glasgow, Glasgow, UK; [8]Sleep Medicine Associates of Texas, Dallas TX; [9]Wake Forest University Health Sciences, Winston-Salem, NC; [10]Universite Laval, Quebec City, CA; [11]Rush-Presbyterian-St. Luke's Medical Center, Chicago, IL

Summary: Insomnia is a highly prevalent, often debilitating, and economically burdensome form of sleep disturbance caused by various situational, medical, emotional, environmental and behavioral factors. Although several consensually-derived nosologies have described numerous insomnia phenotypes, research concerning these phenotypes has been greatly hampered by a lack of widely accepted operational research diagnostic criteria (RDC) for their definition. The lack of RDC has, in turn, led to inconsistent research findings for most phenotypes largely due to the variable definitions used for their ascertainment. Given this problem, the American Academy of Sleep Medicine (AASM) commissioned a Work Group (WG) to review the literature and identify those insomnia phenotypes that appear most valid and tenable. In addition, this WG was asked to derive standardized RDC for these phenotypes and recommend assessment procedures for their ascertainment. This report outlines the WG's findings, the insomnia RDC derived, and research assessment procedures the WG recommends for identifying study participants who meet these RDC.

Citation: Edinger JD; Bonnet MH; Bootzin RR et al. Derivation of research diagnostic criteria for insomnia: report of an American Academy of Sleep Medicine work group. *SLEEP* 2004;27(8):1567-96.

INTRODUCTION

OVER 30 YEARS AGO, THE FIELD OF PSYCHIATRY FOUND ITSELF STRUGGLING WITH AN IMPRECISE DIAGNOSTIC SYSTEM THAT RESULTED IN UNRELIABLE DIAGNOSTIC ASSIGNMENTS ACROSS CLINICAL AND RESEARCH SETTINGS. Largely this was due to the absence of explicit operational criteria in the diagnostic manuals published to aid psychiatric research and practice. As a result, diagnostic practice was a highly subjective and unreliable process that relied as much on psychiatric clinicians' and researchers' conceptions of the diagnoses they assigned as it did on the diagnostic manuals designed to guide their decisions. Fortunately, this problem was effectively addressed by the development of Research Diagnostic Criteria (RDC)[1, 2], a set of operationally defined inclusion and exclusion criteria that standardized the definitions for a majority of the recognized psychiatric conditions. These RDC dramatically improved diagnostic reliability among clinicians and researchers and were quickly incorporated into psychiatry's diagnostic manuals.[3-6]

The field of sleep medicine currently finds itself only slightly ahead of where the field of psychiatry was 30 years ago. For some time now, sleep specialists have had at their disposal diagnostic manuals[6-9] that describe a range of sleep disorders and provide lists of diagnostic criteria for their ascertainment. However, sleep disorder diagnosis has been complicated by the existence of several distinctive nosologies that differ markedly and often produce rather discordant classification results.[6-11] Moreover, many current criteria sets for sleep diagnoses are vague or lack sufficient specificity to assure reliable diagnoses across clinical and research settings.[10-13] Recognizing this problem, work groups have convened to develop RDC-like definitions for selected sleep disorder diagnoses such as sleep apnea[14] and restless legs syndrome.[15] There is little doubt that such efforts will benefit the field greatly by standardizing clinical practice and research with such disorders. Nonetheless, for many sleep disorders, research and practice remains greatly hampered by a lack of universally accepted and precise diagnostic criteria.

Nowhere is this problem more apparent than it is in the basic and clinical research pertaining to insomnia. Although there has been general agreement that insomnia *per se* is a symptom and not necessarily an independent sleep disorder, there has been great variability in how this "symptom" has been defined in the literature. For example, some liberal definitions[16, 17] focus solely on the presence of nocturnal sleep disturbances (e.g., sleep initiation or maintenance difficulties, nonrestorative sleep), whereas other more conservative definitions require additional features such as associated daytime impairment[18, 19], sleep dissatisfaction[20], or meeting all diagnostic criteria for a sleep disorder

Disclosure Statement

Dr. Doghramji has received research support from Cephalon, Orphan, Sanofi, and GlaxoSmithKline; and has received consulting fees from Cephalon, Forest, and Sanofi. Dr. Stepanski was the site PI for contracts studying Hypnotic Compounds supported by Cephalon and Neurocrine; and has received honoraria for speaking at courses supported by the ACCP and the Atlanta School of Medicine. Dr. Jamieson is a member of the educational speakers' bureau for Sanofi-Synthelabo; and has received research support from Sanofi-Synthelabo, Sepracor, Neurocrine, Merck, Takeda America, and Cephalon. Dr. Morin has received research support from Sanofi-Synthelabo and Aventis; and is a member of the speakers' bureau for Sanofi-Synthelabo, Sepracor, and Pfizer. Dr. Edinger served as the PI for a multi-site study designed to test an investigational device for the treatment of insomnia supported by Respironics Corporation; has received honoraria for speaking engagements supported by Sepracor; and has participated in speaking engagements supported Fission Communications. Dr. Dorsey has received research support from Sanofi-Synthelabo, Takeda, Neurocrine, Pfizer, Merck, Aventis, and Cephalon. Dr. McCall has received research support from Sepracor, Takeda, and Neuronetics; has received consulting fees from King Pharmaceuticals and Sepracor; and has participated in speaking engagements supported by Wyeth and Sepracor. Dr. Bonnet has received research support from Cephalon and Pfizer. Drs. Espie and Bootzin have indicated no financial conflicts of interest.

described in one of the available nosologies.[21-25] In addition, insomnia definitions have varied as a function of inconsistent use of frequency, duration, symptom type (e.g., onset problems, maintenance difficulties) and/or severity criteria for case ascertainment.[26] As demonstrated recently by Ohayon[26], use of these varied definitions in epidemiological studies has led to drastically different conclusions regarding insomnia's general prevalence, risk factors, morbidity, and costs to society at large.

Unfortunately, such problems have not been confined to the epidemiological literature. Variable insomnia definitions have encumbered studies concerning the pathophysiology, clinical characteristics, and treatment of this form of sleep disturbance. Admittedly, the advent of consensually-derived insomnia classification schemes[4-9, 27] within the past 25 years has provided some standardization to insomnia research by providing clinical criteria sets for use in research sample characterization. However, both the degree to which researchers have adhered to criteria as well as the methods used for their ascertainment have been highly variable regardless of the specific diagnostic subtype in question. Due to this lack of standardization, synthesizing results of multiple insomnia studies is a difficult if not impossible task.

Given this problem, the American Academy of Sleep Medicine (AASM) commissioned a project designed to develop standard definitions for currently recognized insomnia disorders. This project was devoted to three Major Objectives/Specific Aims. The first aim was to conduct a critical review of the insomnia literature to determine which of the current insomnia diagnoses appear most reliable and valid regardless of the nosological system in which they are defined. The second aim was to derive standard RDC for defining each of the subset of insomnia diagnoses that seem to have the greatest empirical and consensual support. The final aim was to propose specific methods for documenting the presence/absence of the specific RDC among patients and subjects to which they are applied.

In planning this project it was recognized that the status of the available literature would set limits on what could be accomplished. For example, it was anticipated that comparisons and synthesis of results across studies would be difficult because of the historic lack of widely accepted insomnia RDC or methods for defining insomnia samples. As a result, it was expected that the conclusions and recommendations derived from this effort would likely be based, out of necessity, more on consensus than on hard evidence. Nonetheless, attempts were made to consider available evidence in addressing the objectives stated above. The remainder of this report provides a discussion of the methods used and results derived by the group assigned to address these objectives.

METHODOLOGY

Project Planning, Direction, and Execution

In August of 1999, the AASM formed an Insomnia RDC Task Force consisting of two representatives of the AASM Board of Directors as well as four additional at large members with expertise in the fields of insomnia classification and treatment. The Task Force was commissioned to develop the scope, direction and objectives/specific aims of the project and to serve as a liaison to the AASM Board. The Task Force was also charged with the naming of an Insomnia RDC Work Group (WG) to carry out all required tasks to address the three specific aims of this RDC project. In November 1999 a Chair and nine additional WG members (i.e., the coauthors of this report) were named and approved by the AASM Board to conduct the activities leading to the results reported herein.

Literature Review

During the first six months of 2000, a series of conference calls and email communications among WG members were conducted to plan the literature review strategy. Given the aforementioned project aims and the vast insomnia literature, it was decided to focus the literature review on articles that would most likely provide information about the reliability and construct validity of currently recognized insomnia diagnoses. The literature deemed relevant included: (1) articles that reported inter-rater reliability data (e.g., % agreement; kappa values) for one or more insomnia subtypes; (2) studies that compared one or more insomnia subtypes with normal sleepers or controls; (3) studies that compared two or more insomnia subtypes with each other or with other types of sleep disorders; (4) comparisons of one or more insomnia subtypes with historic normative data; (5) comparisons of treatment responsiveness of two or more insomnia subtypes undergoing the same insomnia treatment; and (6) descriptive case series studies of one or more insomnia subtypes that provide information about the defining features of the subtype(s) in question. Excluded from the literature review were treatment studies (randomized trials, case series, within-subject designs) conducted to evaluate one or more forms of insomnia therapy with a single insomnia subtype unless the trial in question provided diagnostic reliability information for the subtype being studied. Also excluded were articles written in a language other than English, single case reports, and all epidemiologic studies except those that reported diagnostic reliability data or examined differences among diagnostic insomnia subtypes. Finally, studies that focused solely on circadian rhythm disorders were excluded since the eventual development of RDC for such conditions, while potentially beneficial to the sleep medicine field, was not a specific aim/objective of this project.

The literature review was conducted by a Duke University Medical Center librarian experienced in computer-assisted library database literature searches under the supervision of the Chair of the WG. Searches were conducted within both the Medline and PSYCINFO databases for the time period between 1966 and June, 2000. Furthermore, searches were conducted for all insomnia diagnostic categories in ICSD (the categories in the 1990 [7] and the 1997[28] versions of the ICSD are identical; therefore, "ICSD" will be used throughout the text to refer to these two versions) and the 1994 version of the DSM-IV.[9] In searching each ICSD diagnosis, the terms insomnia and disorders of initiating and maintaining sleep were cross-referenced with the current name of the diagnosis and the *synonyms and key words* listed with the diagnosis in the ICSD text. In the case of the DSM-IV subtypes, the terms insomnia and disorders of initiating and maintaining sleep were cross-referenced with the current name of the diagnosis and, where indicated, a list of diagnostic subtypes. For example, in the case of Substance-induced Insomnia, such terms as alcohol use/abuse, opiate use/abuse, cocaine use/abuse, etc., were included in the search. Full documentation of the search terms used for all searches is available on request from the first author.

Data Extraction Sheets

To facilitate extraction of relevant information from each arti-

cle reviewed, the WG constructed separate reliability and validity data extraction sheets. Both forms elicited information about the citation (authors, title, journal, year, page numbers), the nature of the study sample (i.e., sample selection, types of subjects, number of subjects, female/male ratio, mean age, etc.), study setting, and study results. The Reliability Data Extraction Sheet also solicited information about the type of reliability assessment conducted and the type of reliability index reported. The Validity Data Extraction Sheet solicited information about the types of group comparisons conducted, the specific measures used, and specific findings (means, standard deviations, statistically significant group differences, etc.) reported in each article. The validity form also solicited a judgment and supporting rationale as to whether the article should be included in an evidence table constructed to assess the validity of the insomnia subtype(s) considered in the article. Finally, both forms also included a check box to indicate that no reliability or validity data were reported in the article listed on the form. Copies of the Reliability and Validity Data Extraction Sheets are shown in Appendix A.

Procedure

To facilitate the final article selection process, the literature search was conducted so that a list of citations and accompanying article abstracts would be produced for each insomnia subtype search conducted. The search results were first divided into the five subsets of articles pertaining to: (1) DSM-IV insomnia subtypes; (2) ICSD intrinsic insomnia subtypes; (3) ICSD extrinsic insomnia subtypes; (4) ICSD insomnia subtypes associated with mental disorders; and (5) ICSD insomnia subtypes associated with medical or neurological disorders. The 10-member WG was subdivided into five dyads, and each dyad was given the search results from one of the five subsets of articles. The WG dyads then reviewed their respective sets of abstracts and eliminated articles that clearly provided no information relevant to the aims of the RDC project (e.g. a randomized clinical trial with one insomnia subtype). When the potential usefulness of an article was in doubt, it was included for an initial review. In addition, dyads were encouraged to select relevant review articles for an initial review inasmuch as such articles provided additional references for consideration.

Once all dyads had completed their initial reviews and submitted their lists of the articles selected for more extensive review, AASM administrative staff obtained copies of the complete articles selected and provided members of each dyad the articles they requested. WG members then carefully reviewed the articles and completed both a Reliability and a Validity Data Extraction Sheet for each one although no such sheets were completed for the review articles. Once this review process was completed, each WG member forwarded the completed data extraction sheets to AASM administrative staff members who entered the data into a database program for subsequent analyses.

In addition to these procedures, a Ph.D. clinical psychologist experienced in sleep medicine was hired to examine each article selected by the WG (excluding review articles) for consideration and extract information about the subject selection criteria and insomnia definitions used. The specific information extracted by the contract reviewer included all self-report (e.g., historic information, presenting complaint, daytime symptoms, description of typical sleep pattern, etc.), self-monitoring data (e.g., sleep diary information), and objective measures (e.g., PSG findings) used as inclusion/exclusion criteria in each of the articles. Also, use of specific diagnostic criteria for subject selection was noted. Findings of this review process were placed into a Microsoft Excel® spreadsheet designed to systematize the database and facilitate the subsequent analyses of information acquired. A copy of the spreadsheet can be obtained from the first author upon request.

Following all data entry procedures, the data extraction sheets were reviewed, and findings supporting the reliability and validity of specific insomnia diagnoses were placed into evidence tables to help identify the most tenable insomnia diagnoses. Subsequently, a series of tabulations were conducted using Version 8.2 of the Statistical Analysis System (SAS Institute, Cary, NC) to determine how frequently various inclusion/exclusion criteria were used for insomnia and normal control sample selection in all articles reviewed. Findings from these tabulations in addition to some additional data (e.g., PSG findings) taken from the completed Validity Data Sheets were then considered in the development of RDC.

RESULTS

Articles Considered for Review and Data Extraction

Following the initial review of a large sample (N=433) of articles requested, a total of 165 articles [10, 12, 13, 20, 23, 29-188] were retained by the WG for data extraction. Table 1 provides categorical breakdowns of the total articles undergoing initial review by the WG as well as those articles selected for data extraction. The 165 articles retained included a total of 176 samples of insomnia sufferers and 83 samples of normal sleepers/controls. The 176 insomnia samples included a total of 9,808 subjects and the 83 control samples included a total of 20,818 subjects. The median size of the insomnia samples was 17.0 (1st quartile = 8; 3rd quartile = 45.5), whereas the median sample size of the normal controls was 20 (1st quartile = 10; third quartile = 42).

Diagnostic Reliability of Insomnia Subtypes

The review process uncovered a paucity of data concerning the reliability of DSM and ICSD-R insomnia subtypes. In fact, only five published studies[12, 62, 128, 138, 163] reported any reliability data, and these studies collectively included only 440 subjects. Two of these studies[12, 163] were conducted solely or in part to evaluate the diagnostic reliability of the DSM (DSM-III-R or DSM-IV) insomnia classification system. In the earlier of these studies, Schramm et al.[163] examined inter-rater reliability among clinicians using a structured interview to differentiate 3 global DSM-III-R insomnia subtypes. In the latter multi-site study, Buysse et al.[12] examined inter-rater reliability among clinicians using a standard clinical interview to identify DSM-IV insomnia subtypes. Given sample size limitations, only the reliability data for two DSM insomnia subtypes were reported in this latter study. In the third cluster analytic study, Edinger et al.[62] reported reliability data for two independent raters who reviewed archival data from a small insomnia sample (n = 31) and assigned both DSM-III-R and ICSD insomnia diagnoses. In the fourth study, Morin and colleagues[128] assessed reliability for ICSD diagnoses by comparing impressions derived from clinical interviews with diagnoses derived independently from sleep history, psychometric evaluations, and available polysomnographic findings.

Finally, Ohayon et al.[138] compared a computer-assisted structured interview with impressions of clinical interviewers to determine their agreement for detecting the presence or absence of any ICSD insomnia diagnosis.

Table 2 provides summary data taken from these five studies. These data show that the reliability indices for DSM categories derived from structured interviews in the Schramm et al.[163] study are very impressive and suggest highly acceptable reliability of these global diagnoses. In contrast, when such global diagnoses are derived from clinical interviews, as was the case in the Buysse et al.[12] study, reliabilities are less impressive. The remaining studies by Edinger et al.[62], Morin et al.[128], and Ohayon et al.[138] suggest reasonable reliability for ICSD diagnoses, but unfortunately, only the Morin et al. study provides reliability data for individual ICSD categories.

Diagnostic Validity of Insomnia Subtypes

Examination of the Validity Data Extraction Sheets showed that 113 of the 165 articles reviewed included comparisons of insomnia subtypes with each other and/or a normal control sample. However, 85 of these articles were excluded from consideration due to study design limitations that reduced the usefulness of the data reported. Many of these studies focused on samples (e.g., Parkinson's Disease, Alcohol Abusers, etc.) with obvious sleep disturbance but lacked verification that individuals in these samples actually had insomnia complaints. In other cases, insom-

Table 1–Types of Articles Considered and Retained for Data Extraction

Type of Article	# requested for initial review	%	# retained for data extraction	%
Review articles	105	24.2	0	0
Case reports	26	6.0	12	7.3
Case series – descriptive	60	13.9	31	18.8
Group comparisons	129	29.8	107	64.8
Inter-rater reliability studies	3	0.7	3	1.8
Clinical trials	37	8.5	2	1.2
Case series – treatment	29	6.7	1	0.6
Within-subject designs	13	3.0	5	3.0
Survey studies	19	4.4	2	1.2
Other	12	2.8	2	1.2
Total	**433**	**100**	**165**	**100***

*Note: Cumulative percentage actually falls just below 100% due to rounding errors.

Table 2. Studies Reporting Inter-rater Reliability Data (% agreement or Kappa values) for Insomnia Diagnoses

Study	Site(s)	Sample size & characteristics	Diagnostic System & Method Used	Insomnia Subtypes	% Agreement	Kappa
Schramm et al.[163]	Manneheim, Gottingen, & Landeck, Germany	68 sleep center patients	DSM-III-R (Structured interview)	Primary Insomnia Insomnia due to a mental disorder Insomnia due to organic factor	97 91 93	0.86 0.84 0.86
Buysse et al.[12]	Bronx (NY) Detroit (MI) Hershey (PA) Pittsburgh (PA) Rochester (NY)	216 insomnia patients referred to participating sleep centers	DSM-IV (Clinical interview)	Primary Insomnia Insomnia due to a mental disorder	 NR	0.40 0.42
Edinger et al.[62]	Durham (NC)	31 insomnia clinic patients	DSM-III-R & ICSD-90 (Chart review)	Mixed Insomnia (DSM-III-R) Mixed Insomnia (ICSD-90)	81 74	0.71 0.68
Morin et al.[128]	Richmond (VA)	20 insomnia patients	ICSD-90	Psychophysiologic Insomnia Insomnia due to a mental disorder Hypnotic-dependent insomnia Other mixed ICSD subtypes	 NR	0.54 0.64 0.70 0.84
Ohayon et al.[138]	Palo Alto (CA) Regensburg (Germany)	105 sleep center patients	ICSD-90 (Sleep EVAL interview)	Presence vs. absence of ICSD insomnia diagnosis	96.9	0.78

Table Caption: DSM-III-R = the 3rd edition of the American Psychiatric Association's Diagnostic and Statistical Manual; DSM-IV = 4th edition of the American Psychiatric Association's Diagnostic and Statistical Manual; ICSD-90 = The International Classification of Sleep Disorders, 1990 Edition; NR = Not reported

nia samples were described merely as "insomniacs" or were composed of a mixture of insomnia subtypes. Since it was not possible to ascertain much about specific insomnia subtypes from such studies, they were not used for assessing the validity of specific insomnia diagnoses. In a number of studies, results of polysomnographic studies and/or sleep logs were used both to diagnose study subjects and to compare the subtypes derived from the initial diagnostic classification process. Given the obvious confounds affecting such group comparisons, data from studies of this nature were not included in our evidence tables.

A review of the remaining 28 articles provided some evidence for the validity of the DSM[4-6] subtypes of Primary Insomnia and Insomnia due to a Mental Disorder but no group comparisons were found concerning the categories of Insomnia due to a General Medical Condition and Substance-induced Insomnia. Table 3 summarizes the findings from the subset of these studies supporting the former two insomnia diagnoses. This table demonstrates that all studies listed in the table included a Primary Insomnia sample and most included a sample of normal controls. Only three of the studies included a sample of patients with Insomnia due to Another Mental Disorder and one of these three studies subdivided this category into the ICSD Insomnia Due to Depression and Insomnia Due to an Anxiety Disorder subtypes. Most of the findings summarized in the table confirm that Primary Insomnia sufferers differ from normal controls, and the comparisons of the two types of insomnia sufferers suggest consistent differences between them. Although the data are much more limited, the evidence table also suggests differences between individuals who have Insomnia due to Another Mental Disorder and noncomplaining normal sleepers.

Twenty of the 28 articles retained included comparisons among insomnia subtypes listed in the ICSD. Table 4 lists the studies providing validity evidence for the diagnosis Sleep State Misperception (SSM) (in the forthcoming ICSD-2 [189], this diagnosis will be called Paradoxical Insomnia). This evidence table supports the validity of the SSM diagnosis inasmuch as the data listed suggest that individuals with this form of insomnia can be differentiated both from noncomplaining normal controls and from other insomnia subtypes. Several of the studies listed include comparisons of SSM patients with both normal controls and Psychophysiologic Insomnia (PSYI) sufferers. These comparisons suggest that SSM and PSYI sufferers have distinct patterns of differences from normal controls. As preordained by their definition, SSM groups overlap with normal sleepers on PSG measures of sleep time, onset latency, and wakefulness during the night whereas PSYI sufferers differ from normal sleepers on these measures. However, several of the studies suggest some sleep stage architectural differences between SSM sufferers and the other groups. Moreover, as their diagnostic name implies, SSM sufferers are distinguished from normal controls and other insomnia subtypes by an exaggerated propensity to underestimate the sleep they obtain. Finally, the table provides some limited evidence for the distinctiveness of SSM in regard to personality trait measures and indices of daytime functioning.

Since most of the studies cited in Table 4 included a PSYI group, these studies provide evidence for the distinctiveness of this diagnosis as well. Presented in Table 5 are some additional studies supporting PSYI. The studies included in this table provide comparisons of PSYI with normal controls, other insomnia subtypes, and other types of sleep disorders. The findings reported in Table 5 along with relevant data presented in Table 4 suggest that PSYI sufferers have more wakefulness during sleep than do SSM sufferers, normal controls, and groups such as narcoleptics. The two tables also suggest that PSYI subjects differ from normal sleepers and other insomnia subtypes on personality trait measures (e.g., MMPI scales). Considered collectively, these data support the notion that PSYI is a distinctive form of insomnia that differs from normal sleep, other insomnia diagnoses, and other types of sleep disorders not typically associated with insomnia complaints.

Table 6 lists studies concerning other ICSD insomnia subtypes including childhood onset (Idiopathic) Insomnia (COI), Insomnia Related to Sleep Apnea, and Insomnia Related to an Anxiety Disorder. The limited data listed in Table 6 along with the two studies listed in Table 5 suggest that COI may be distinguished from other insomnia subtypes in terms of objective and subjective sleep measures as well as the duration of complaints. Hauri's cluster analytic study[83] cited in Table 5 suggests COI sufferers may be discriminated from normal sleepers on the basis of historic data, presenting information, and sleep lab findings. However, it should be noted that the study by Philip and Guilleminault[146] listed in Table 5 showed few differences between COI and adult-onset insomnia. Considered collectively, these three reports appear to provide some minimal support for the validity of COI although the information about this condition is much more limited than it is for SSM and PSYI.

Similarly, the evidence supporting the diagnosis of Insomnia Related to Sleep Apnea (AI) is very limited. Although the WG encountered many studies that included a sleep apnea sample, in most cases it was not clear that the individuals composing such samples actually had insomnia complaints. The two studies concerning AI cited in Table 6 are the exceptions to this trend. The first study by Roehrs et al.[154] compared an AI sample with another sample of apnea patients who presented mainly with complaints of excessive daytime sleepiness (AS). The second study by Stone et al.[173] compared a sample of AI patients with another mixed insomnia group. The former study found statistically significant differences between AI and AS groups on PSG sleep and MSLT sleep latency whereas the latter study failed to find any differences on measures of daytime cognitive functioning. Thus, experimentally sound studies examining the validity of AI appear to have been so limited that it is difficult to draw conclusions about the validity of this insomnia subtype.

The one remaining study listed in Table 6 involved a comparison of normal controls with a group of individuals with Insomnia Related to an Anxiety Disorder (IAD). This study showed group differences across a range of measures including PSG parameters, ratings of sleep quality, diurnal motor speed and reaction time, and measures derived from EEG mapping studies. It should also be noted that studies [21, 152]cited in Tables 3 and 5 suggest that IAD can be discriminated from other insomnia subtypes on the basis of objective measures of sleep architecture and subjective sleep ratings and self-report measures of daytime functioning. Hence, IAD appears to be a viable diagnosis on the basis of the evidence cited.

In addition, it is noteworthy that individuals with Insomnia Related to a Depressive Disorder (ID) such as Major Depressive Disorder or Dysthymia were included as comparison samples in several studies [13, 21, 29, 86, 152, 176, 180]listed in Tables 3, 4 and 5. Considered collectively, such studies suggest differences between these and other insomnia subtypes on measures of sleep architecture, subjective sleep quality, perceptions of diurnal functioning,

Table 3. Evidence supporting validity of DSM-III-R/DSM-IV diagnoses of Primary Insomnia and Insomnia due to a Mental Disorder

Reference	Samples Compared	Measures Used Mean Age/ Or Range	Polysomnography	Subjective Sleep Estimates & Complaints	Objective/ Subjective Daytime Measures	Clinician Ratings	Other
Frankel et al, [67]	18 (5F) PRI 18 (4F) NC	44.5±16.8 yrs 45.1±16.8 yrs.	PRI > NC on SOL NC > PRI on TST & SE%	PRI overestimated SOL and underestimated TST & SE%; NC estimates not different from their PSG measures.	NR	NR	NR
Gallard[68]	16 (5F) PRI 16 (5F) NC	44±9 yrs. 44±9 yrs.	PRI > NC on SOL, TWT, TIB, NA NC > PRI on TST, SE%, SWS, and mean stage duration	NR	NR	NR	NR
Hajak et al.[79]	10 (3F) PRI 5(0F) NC	41.3± 9.5 yrs. 27.2±0.7 yrs.	PRI > NC on NA, & %TWT NC > PRI on S4% & SE%	NR	NR	NR	PRI < NC on plasma melatonin levels between 3:00 & 8:00 AM
Nowell et al. [13]	48 PRI 99 IMD	14 - 89 yrs.	NR	NR	NR	Negative conditioning & poor sleep hygiene rated more important to PRI diagnosis. The presence of psychiatric disorder rated more important to IMD	NR
Ohayon et al. [21]	73 PRI 81 ID 84 IA	15 - 96 yrs.	NR	PRI < I+D & I+A on ratings of maintenance difficulty, # insomnia sxs, insomnia duration, nightmares, & restlessness upon awakening PRI < I+D on sleep drunkenness, and breathing pauses. PRI < I+A on NA	PRI < I+D & I+A on anxious mood, concentration/attention, problems & psychic irritability. PRI < I+D on difficulty getting started, daytime sleep, depressed mood, memory problems, anxiolytic use & antidepressant use.	NR	Statistical discriminant analyses showed PRI, I+D and I+A could be discriminated from each other and from groups of depressive and anxiety disorders with insomnia symptoms
Rosa & Bonnet[155]	121 (49F) PRI 56 (21F) NC	18 - 50 yrs.	No group differences on PSG	PRI > NC on SOL, NA, & WASO.	PRI > NC on all MMPI scales except L, Mf, & MA and on STAI. PRI > NC on 5/6 POMS negative mood scales PRI > NC on 4PM MSLT PRI < NC on memory test	NR	NR
Stepanski et al.[172]	10M PRI 10M NC	37±7.5 yrs 37.5±8.8 yrs.	NR	NR	PRI > NC on MSLT SOL	NR	NR
Weissman et al.[180]	533 (371F) PRI 408 (300F) IMD 6172 (3627F) NC	51.8± 10.3 yrs 44.9±18.9 yrs. 48.0±20.0 yrs.	NR	NR	IMD > PRI > NC on health care utilization	NR	NR

Figure Caption: PRI = primary insomnia; NC = normal control; ID = insomnia due to depressive disorder; IA = insomnia due to anxiety disorder; IMD = insomnia due to a mental disorder; NR = not reported; PSG = polysomnography; SOL = sleep onset latency; NA = number of awakenings; WASO = wake time after sleep onset; TWT = total wake time; TIB = time in bed; SE% = sleep efficiency percent; SWS = slow wave sleep; S4% = stage 4 percent; MMPI = Minnesota Multiphasic Personality Inventory; STAI = State-Trait Anxiety Inventory; POMS = Profile of Mood States; MSLT = Multiple Sleep Latency Test

Table 4. Evidence supporting validity of ICSD Diagnosis of Sleep State Misperception.

Reference	Samples Compared	Measures Used: Mean Age/ Or Range	Polysomnography	Subjective Sleep Estimates & Complaints	Objective/ Subjective Daytime Measures	Other Physiologic Measure	Other Comparisons
Bonnet & Arand[44]	9 (2F) SSM 9 (2F) NC	32±8 yrs. 33±6 yrs.	Used for group identification: therefore group comparisons are confounded.	Used for group identification	NC > SSM on vigilance tests SSM > NC on MMPI scales 1,7 & 8 SSM > NC on POMS Tension, Depression, Anger & Confusion	SSM > NC on measure of 24-hour metabolic rate	NR
Borkovec et al.[47]	17 SSM 12 IP	NR - college students	SSM < IP on SWS time	IP > SSM on measure of pre-sleep body tension during initial nights in the sleep lab	NR	NR	SSM < IP on PGS sleep changes from Tx
Dorsey & Bootzin[56]	9 SSM 9 PSYI 13 NC	18 - 25 yrs.	PSYI > SSM & NC on SOL SSM > PSYI & NC on SWS%	NR	PSYI < SSM & NC on EPI introversion/extroversion scale	NR	NR
Hauri & Wisbey[86]	8 (4F) SSM 10 (8F) PSYI 13 (9F) IMD	43.9±9.4 yrs. 50±11.1 yrs 45.1±11.6 yrs.	For SSM PSG TST > actigraphy TST For PSYI & IMD PSG=actigraphy TST	For SSM PSG TST > STST For PSYI & IMD PSG TST=STST	NR	SSM < PSYI & IMD on actigraphy TST	NR
Kuisk et al.[98]	8 (4F) SSM 8 (6F) PSYI 8 (5F) NC	21 - 60 yrs.	NR	SSM < PSYI on frequency of pre-sleep/sleep onset cognitive activity	NR	NR	NR
Salin-Pascaul et al.[160]	7 (3F) SSM 7 (4F) PSYI 7 (4F) NC	36.4±5.9 yrs 35.4±6.3 yrs. 35.6±5.9 yrs.	SSM > PSYI & < NC o n SWS% SSM > NC & < PSYI on S2% SSM & PSYI > NC on S1% PSYI < SSM & NC on TST PSYI > SSM & NC on SOL, NA, WASO	SSM & PSYI < NC on TST SSM & PSYI > NC on SOL & WASO	SSM & PSYI > NC on MMPI Hy scale PSYI > NC on MMPI Hy & D scales	NR	NR
Sugerman et al.[174]	8 (6F) SSM 8 (6F) PSYI 8 (6F) NC	32.4±10.0 yrs 37.9±9.1 yrs. 32.1±9.4 yrs.	SSM > PSYI & NC on S2%, A/hr & ST/hr PSYI < SSM & NC on TST	NR	SSM > PSYI & NC in AVT errors SSM had flatter MSLT profile than PSYI & NC groups	NR	NR
Vanable et al.[176]	8 SSM 19 PSYI 11 ID 21 IOMD 24 PLMD 21 OAS	45±11.4 yrs for whole sample	NR	SSM < rest of the sample in % TST estimated PSYI > than rest of the sample in % TST estimated	NR	NR	NR

Figure Caption: SSM = sleep state misperception; PSYI = psychophysiologic insomnia, NC = normal control; ID = insomnia due to a depressive disorder; IOMD = insomnia due to a mental disorder other than depression; PLMD = periodic limb movement disorder; OSA = obstructive sleep apnea; IP = other mixed idiopathic insomnia; IMD = insomnia due to a mental disorder; PSG = polysomnography; SOL = sleep onset latency; NA = number of awakenings; WASO = wake time after sleep onset; TWT = total wake time; TIB = time in bed; SE% = sleep efficiency percent; SWS = slow wave sleep; S1% = stage 1 percent; S2% = stage 2 percent; MMPI = Minnesota Multiphasic Personality Inventory; EPI = Eysenck Personality Inventory; POMS = Profile of Mood States; MSLT = Multiple Sleep Latency Test; AVT = auditory vigilance test; NR = not reported.

Table 5. Evidence supporting validity of ICSD Diagnosis of Psychophysiologic Insomnia Excluding Relevant Studies Cited in Table 4

Reference	Samples Compared	Measures Used: Mean Age/ Or Range	Polysomnography	Subjective Sleep Estimates & Complaints	Objective/ Subjective Daytime Measures	Other Physiologic Measure	Otherv Comparisons
Aikens et al.[29]	20 PSYI 30 ID 28 PLMD 30 OAS	47.5 for all groups combined	NR	NR	PSYI, ID, & PLM > OSA on MSLT PSYI < ID & PLMD on MMPI D, Pt & Sc scales	NR	NR
Hauri[83]	89 Mixed Pts. 10 NC	Adults	NR	NR	NR	NR	PSYI & COI separated form other groups in a cluster analysis
Lee et al.[103]	24 (11F) PSYI 16 (8F) NAR	45.1±11.1 yrs. 44.4±14.6 yrs.	PSYI < NAR on SE% PSYI > NAR on SOL & REMLAT PSYI > NAR on length of 1st two REM periods.	PSYI > NAR on frequency of nightmares & recurrent dreams	NR	NR	NR
Lichstein et al.[104]	20 (11) PSYI 20 (12F) HDI	49.6±14.8 yrs. 54.8±16 yrs.	NR	NR	NR	NR	PSYI < HDI on improvement in sleep quality from insomnia treatment
Mercia & Gaillard[122]	12 (7F) PSYI 23 (12F) NC	35.9±12.3 30.0±9.5	PSYI > NC on NA & WLAT PSYI < NC on SE% & TST	NR	NR	NR	Discriminant analysis shows beta & delta differences between PSYI & NC
Philip & Guilleminault[146]	38 (20F) PSYI 27 (14F) COI	51±13 yrs. 43.9±9 yrs.	NR	PSYI < COI on frequency of nightmares PSYI < COI on SOL fear of dark during childhood	NR	NR	NR
Reynolds et al.[152]	10 (8F) PSYI 10 (5F) GAD 20 (13 F) MDD	43.8±13.6 yrs. 36.8±14.5 yrs. 37.2±15.2 yrs.	PSYI <GAD & MDD on REM density PSYI & GAD < MDD on S2%, REM% REMLAT, & Phasic REM activity PSYI FNE > GAD FNE for measures of SE% & REM activity	NR	NR	NR	NR

Figure Caption: PSYI = psychophysiologic insomnia, NC = normal control; ID = insomnia due to a depressive disorder; HDI = hypnotic-dependent insomnia; PLMD = periodic limb movement disorder; OSA = obstructive sleep apnea; COI = childhood onset (idiopathic) insomnia; NAR = narcolepsy; MDD = major depressive disorder; GAD = generalized anxiety disorder; SOL = sleep onset latency; NA = number of awakenings; WLAT = awakening latency; REM = rapid eye movement sleep; REMLAT = REM latency; SE% = sleep efficiency percent; TST = total sleep time; FNE = first night effects; S2% = stage 2 percent; MMPI = Minnesota Multiphasic Personality Inventory; MSLT = Multiple Sleep Latency Test; NR = not reported.

Table 6. Evidence Supporting Validity of Other ICSD Diagnoses

Reference	Samples Compared	Measures Used: Mean Age/ Or Range	Polysomnography	Subjective Sleep Estimates & Complaints	Objective/ Subjective Daytime Measures	Other Physiologic Measure	Other Comparisons
IDIOPATHIC (CHILDHOOD ONSET) INSOMNIA							
Hauri & Olmstead[82]	11 (8F)COI* 11(7F) OMI	42±13 yrs. 43±12 yrs.	COI > OMI on SOL & WASO COI < OMI on TST, Phasic REM%, & mean sleep stage duration	COI > OMI on SOL	Only 1 of 42 questionnaire comparison significant. These results interpreted as a "chance" finding.	NR	COI > OMI in insomnia duration
INSOMNIA ASSOCIATED WITH SLEEP DISORDERED BREATHING							
Roehrs et al.[154]	16 (14F) AI 65 (2F) AS	39±18 yrs. 47±1.4 yrs	AI < AS on TST & S1% AI > AS on SOL, S2%, SWS%, & SE% AI < AS on RDI & several indices of O_2 desaturation	NR	AI > AS o n MSLT latency	NR	NR
Stone et al.[173]	18 (3F) AI 16 (14F) OMI	55 - 84 yrs.	Used for group identification: therefore group comparisons are confounded.	NR	AI differs from OMI at 0.05 level of significance on 2 of 12 cognitive tests, but no differences found using bonferroni corrected p = .004 (.05÷12 tests) level.	NR	NR
INSOMNIA DUE TO GENERALIZED ANXIETY DISORDER							
Saletu et al.[159]	44 (25F) IAD 34 (20F) NC	43.2±11.7 yrs. 41.1±12.3 yrs.	IAD > NC in TWT & T-WASO IAD < NC on TST & SE%	IAD < NC on subjective sleep quality ratings	IAD < NC on subjective waking quality ratings. IAD < NC on diurnal measures of fine motor and reaction time performance	IAD > NC on hypervigilance measure derived from EEG mapping	IAD > NC on sleep pressure detected by late AM EEG mapping

Figure Caption: *These age- and gender-matched samples were taken from larger samples of 20 COI & 39 OMI groups. COI = childhood onset (idiopathic) insomnia; OMI = other mixed insomnia subtypes; AI = sleep apnea with insomnia; AS = sleep apnea with excessive sleepiness; IAD = insomnia due to an anxiety disorder; NC = normal controls; SOL = sleep onset latency; WASO = wake time after onset; TWT = total wake time; TST = total sleep time; T-WASO = total wake time after sleep onset; REM = rapid eye movement sleep; S1% = stage 1 percent; S2% = stage 2 percent; SWS% = slow wave sleep percent; SE% = sleep efficiency percent; MSLT = multiple sleep latency test; O2 = oxygen; RDI = respiratory disturbance index; EEG = electroencephalogram; NR = not reported.

and personality traits. Furthermore, one of these studies[21] showed that ID could be statistically discriminated from IA and Primary Insomnia on the basis of subjective sleep ratings and self-report measures of daytime functioning. As a result, it would appear that ID warrants consideration as a separate insomnia diagnosis as well.

Finally, since two studies[29, 176] listed in the evidence tables included samples of insomnia sufferers with periodic limb movement disorder (PLMD), this diagnosis warrants consideration. Unfortunately, the data supporting the distinctiveness of this condition appears very limited. The studies listed suggest individuals with PLMD differ from some other insomnia subtypes on a limited number of personality trait measures and in regard to how accurately they estimate sleep time. Since there have been few comparisons of PLMD with other insomnia subtypes, there currently is very limited data supporting the validity of this diagnosis.

Consensual Definitions

The evidence tables help delineate the most supportable insomnia diagnoses and provide some guidance as to how these subtypes might be identified in the research subject selection process. In addition, a review of the subject selection criteria used in these studies should help identify consensual research definitions that aid in the development of RDC. However, since most studies reviewed were excluded from the evidence tables, it seemed that much information about consensual research definitions for the various subtypes considered would be ignored if only the selection criteria for studies included in the evidence tables were tabulated. Given this consideration, the WG decided to examine the subject selection criteria for all 165 articles retained for review.

Tabulations of study selection criteria showed marked variability in the types of criteria used for selection of both insomnia and normal control samples. In fact, no single criterion or criteria set was used for selection of as many as 50% of the insomnia or normal control samples described in the articles reviewed. Tabulation results showed that a total of 14 distinctive types of criteria were used for selection of at least 5% of the insomnia samples considered herein. Eight of these were also used with variable frequencies for selection of the normal control samples described in the reviewed studies. Figure 1 shows these criteria sets and the frequency with which they were employed in selecting the insomnia and normal control samples.

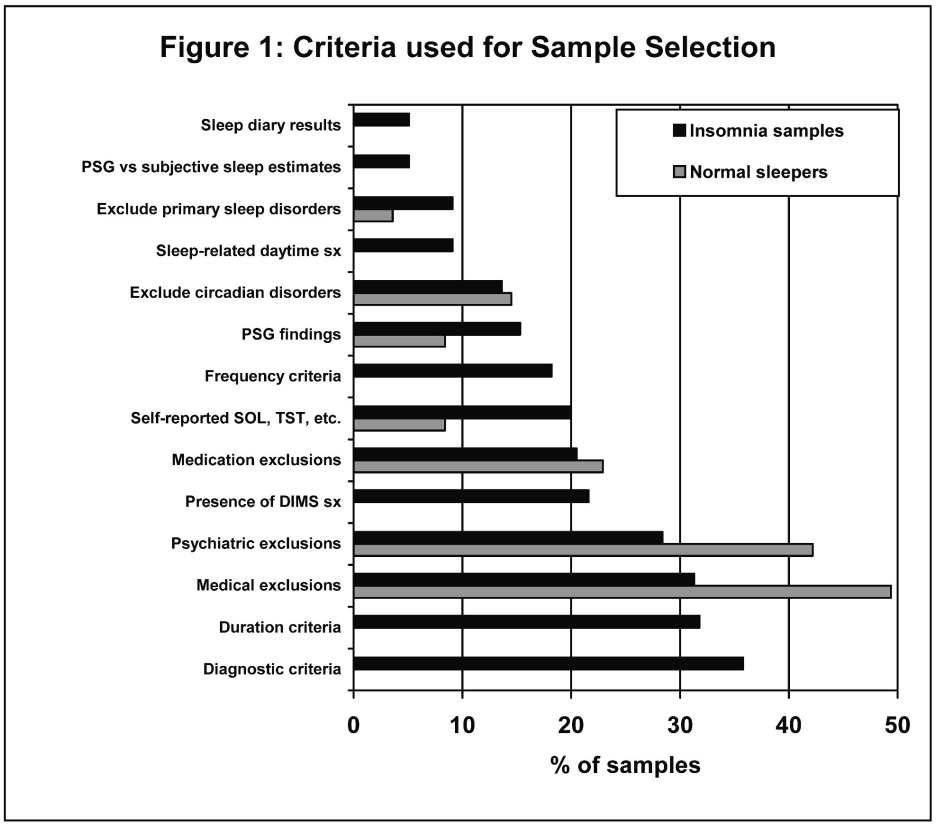


Figure Caption 1–The figure shows the 14 criteria used most frequently for subject selection. Diagnostic criteria = use of published[4-7, 27, 189] diagnostic criteria; Duration criteria = selection on the basis of a minimum insomnia duration; Medical exclusions = subjects excluded for sleep-disrupted medical conditions; Psychiatric exclusions = subjects excluded for the presence of some or any psychiatric conditions; Medication exclusions = subjects excluded for current use of psychoactive medications; Presence of DIMS complaint = subject selection on the basis of specific complaints of sleep onset difficulties, sleep maintenance difficulties, and/or nonrestorative/poor quality sleep; Self-reported SOL, TST, etc. = subjects selected who report certain predetermined mean values of certain sleep parameters such as sleep onset latency, total sleep time, etc.; Frequency criteria = subjects selected on the basis of having insomnia a predetermined minimum number of nights per week; PSG findings = certain polysomnographic findings needed for subject selection; Exclude circadian disorders = subjects who had evidence of unusual sleep/wake schedules (e.g., shift work) or circadian rhythm disorders were excluded; Sleep-related daytime symptoms = subjects were required to have daytime impairment related to sleep difficulties; Exclude primary sleep disorders = subjects who had symptoms/complaints suggestive of certain primary sleep disorders such as sleep apnea, narcolepsy, restless legs syndrome, periodic limb movements, and/or parasomnias were excluded; PSG vs. subjective sleep estimates = subjects selected on the basis of predetermined differences between polysomnographic sleep measures and their coincident subjective sleep estimates; Sleep diary results = sleep diary results were used for subject screening.

As might be expected, most of the selection criteria for the normal samples in the articles reviewed were designed to exclude individuals with evidence of any form of sleep disruption.

Typically such samples were described as individuals either without sleep complaints or without insomnia. Almost 50% of the normal control samples specifically excluded individuals with selected medical disorders that commonly disrupt sleep (e.g., chronic pain conditions). Approximately 42% of the normal samples excluded individuals with histories or symptoms of psychiatric disorders, whereas roughly 23% excluded individuals who reported ongoing use of hypnotics or other psychoactive agents. Roughly 15% of the samples excluded those with unusual sleep/wake schedules or circadian rhythm disorders. Between 8% and 9% of the samples included individuals selected on the basis of "normal values" of sleep onset latency, wake time after sleep onset (WASO) or total sleep time (TST) obtained either from self-report or a screening polysomnogram (PSG). Fewer than 4% of the samples excluded individuals with primary sleep disorders (e.g., sleep apnea, narcolepsy, restless legs syndrome, etc). About 50% of the normal samples included in the articles reviewed met at least two of these selection criteria, but over 85% of these samples were selected using three or fewer of these criteria.

About 55% of the insomnia samples were selected using at least two of the selection criteria listed in Figure 1, but slightly under 30% of these insomnia samples were selected using more than three of these criteria. When the criteria were tabulated individually, it was noted that satisfaction of published diagnostic criteria[4-7, 27] for a particular insomnia subtype was the most frequently used entry requirement in selecting insomnia samples. This requirement was used for slightly over 36% of the insomnia samples considered. For roughly 32% of the insomnia samples, minimal insomnia duration requirements were used for sample selection; the most frequently used criterion in this regard was an insomnia duration of six months or longer. Between 20% and 30% of the insomnia samples excluded individuals with sleep-disruptive medical disorders (29.7% of the samples), histories and/or current symptoms of specified psychiatric disorders (26.7% of the samples), and/or ongoing use of hypnotics or other psychoactive medications (20.9% of the samples). Subject selection for roughly 20% of the insomnia samples was based on the presence of an insomnia complaint (i.e., sleep initiation or maintenance difficulty, poor sleep quality or nonrestorative sleep) or self-reports of an average sleep onset latency (SOL), wake time after sleep onset (WASO), or total sleep time (TST) that surpassed predetermined thresholds for insomnia (e.g., SOL or WASO > 30 minutes; TST < 6 hours). Selection criteria for approximately 18% of the samples included consideration of insomnia frequency. The most commonly used frequency criterion required the occurrence of insomnia three or more nights per week. Slightly fewer than 16% of the insomnia samples were selected on the basis of meeting *a priori* thresholds (i.e., predetermined values for sleep/wake time measures, the RDI, or PLM index) during a screening polysomnogram (PSG), whereas approximately 12% of the samples excluded individuals with variable sleep/wake schedules and/or circadian rhythm disorders. Less frequently used selection criteria included the requirement of self-reported insomnia-related daytime symptoms or impairment (9.3% of the samples), exclusions for the presence of selected primary (e.g., sleep apnea, narcolepsy, etc.) sleep disorders (7% of the samples), predetermined levels of similarity or dissimilarity between subjective sleep estimates and corresponding PSG measures (5.2% of the samples), and *a priori* thresholds for mean values of SOL, WASO, and/or TST derived from screening sleep logs (5.2% of the samples).

To determine how these findings generalized to each of the insomnia subtypes listed in the evidence tables (i.e., Tables 3-6), all of the studies reviewed that contained samples of one or more of these subtypes were first selected. Specifically, studies were retained if they included individuals with DSM[4-6] Primary Insomnia and/or Insomnia due to Another Mental Disorder as well as studies that included samples with PSYI, SSM, COI, Insomnia due to PLMD, AI, IAD, and/or ID as defined by either the ASDC nosology[27] or ICSD-1.[7] Subsequently, tabulations were conducted to ascertain how frequently each of the above-mentioned study selection criteria was used in each of these types of insomnia samples.

A total of 61 articles that included a total of 81 insomnia samples were retained. The numbers of samples for each of the subtypes considered are shown in Table 7 (see table caption for list of references). Relatively few samples with ICSD diagnoses of IAD and ID were found. Since the forthcoming ICSD-2[189] and DSM texts[4-6] combine these categories into a single global insomnia diagnosis, these groups were combined with the samples assigned the DSM diagnosis of Insomnia due to Another Mental Disorder to form one larger set of samples with Insomnia Due to Mental Disorders. Results of the subsequent frequency tabulations of study selection criteria used for each of these subtypes are shown in Figures 2a-2c. Figure 2a shows tabulations for the DSM insomnia supported by the evidence tables whereas Figure 2b shows similar tabulation for the ASDC and ICSD subtypes that appear most tenable. Presented in Figure 2c are the ASDC and ICSD subtypes that received only minimal or incidental support from the evidence tables.

Figure 2a shows that the research criteria most frequently used for selecting samples of Primary Insomnia and Insomnia due to a Mental Disorder closely reflect those features emphasized in the DSM diagnostic criteria sets for these subtypes. In fact, DSM diagnostic criteria were used in subject selection for two thirds of the samples with Insomnias due to a Mental Disorder. Whereas relevant DSM criteria were used for subject selection in slightly under 40% of Primary Insomnia samples, exclusions for sleep-disruptive medical problems, active psychiatric conditions, and psychoactive medications emphasized by these criteria were used

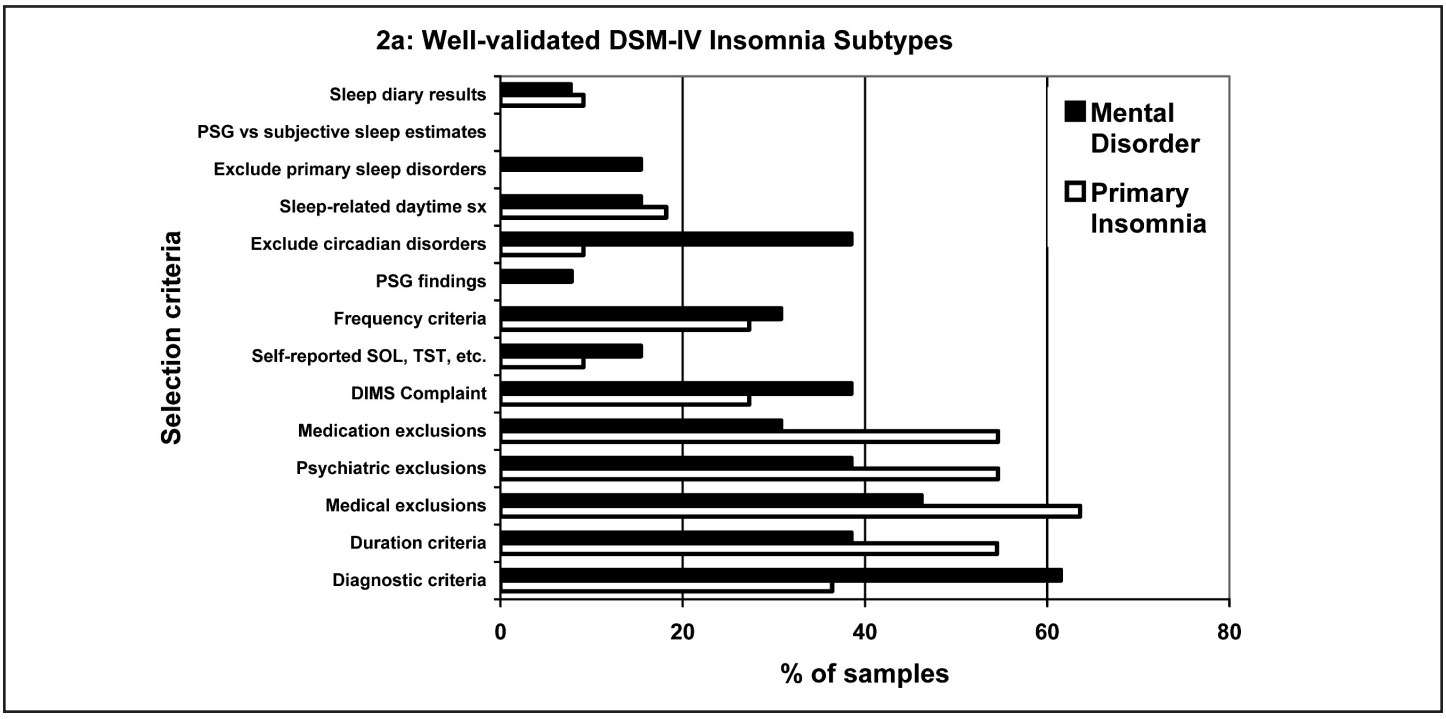
Table 7. Number of Samples of Various Insomnia Subtypes Examined for Consensual Definitions

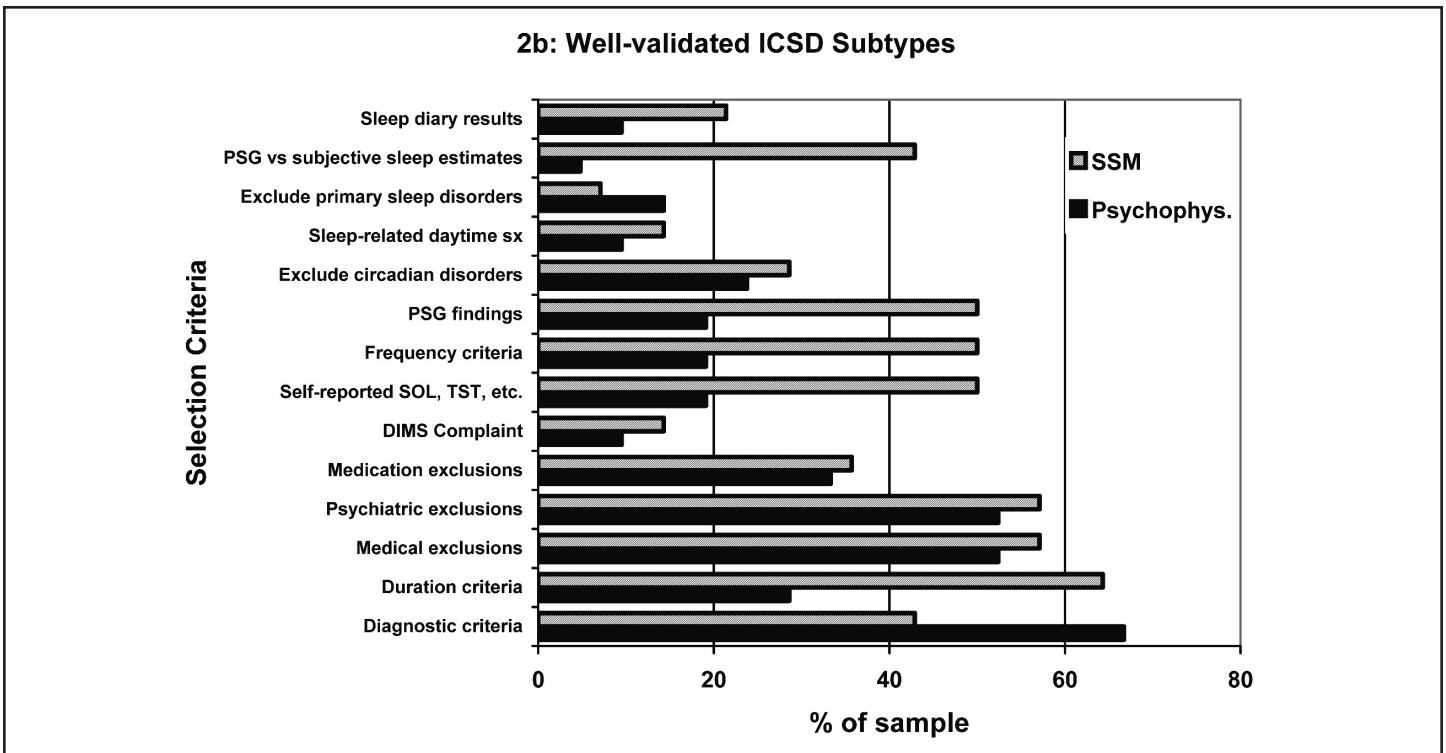
Diagnostic Category	# Samples Considered
Primary Insomnia	11
Insomnia due to Another Mental Disorder	8
Psychophysiologic Insomnia	21
Sleep State Misperception	14
Idiopathic/Childhood Onset Insomnia	3
Insomnia due to Sleep Apnea	5
Insomnia due to Periodic Limb Movement Disorder	14
Insomnia due to an Anxiety Disorder	3
Insomnia due to a Depressive Disorder	2
Total Samples	**81**

Figure Caption: Samples were derived from articles with the following reference numbers in the reference list: (13, 29, 43, 44, 46, 47, 51, 56-58, 63, 67, 68, 73, 75-77, 79, 80, 84, 86, 98, 103-105, 112, 113, 119-122, 128, 133, 135, 143, 145, 146, 148, 151-154, 156, 158-162, 166, 168, 170, 172-178, 180, 183, 188)

in selecting over 50% of these samples. The figure shows that ascertainment of an insomnia (DIMS) complaint, as well as use of duration and frequency criteria were commonly employed in subject selection of both insomnia subtypes. Exclusion of circadian rhythm disorders was mentioned in selecting one third of the insomnia samples with mental disorders but this criterion was seldom specifically mentioned in the selection of the Primary Insomnia samples. However, since this exclusion is incorporated into DSM criteria for Primary Insomnia, it can be assumed that such samples selected on the bases of these criteria excluded individuals with circadian disturbances. A similar assumption can be made about the limited consideration of daytime impairment as a selection criterion for either type of sample since the diagnostic criteria for both subtypes require evidence of such impairment. In contrast, PSG findings and subjective sleep estimate via self-report or sleep diary seem less essential since they were infrequently used as selection criteria for these subtypes.

Figure 2b shows tabulation results concerning criteria used for selection of the two most strongly supported ICSD insomnia subtypes, PSYI and SSM. These data suggest some overlap and





Figures 2a-2b–Research Selection Criteria used for Defining Insomnia Subtypes. The definitions provided in the caption for Figure 1 apply to Figures 2a, 2b, & 2c.

some notable differences in the research selection criteria used for these subtypes. Specific diagnostic criteria as well as exclusions for sleep-disruptive medical problems, active psychiatric conditions, psychoactive medications, and circadian disorders were frequently employed for the selection of both subtypes in the studies considered. Another common finding was that insomnia complaints, daytime symptoms or impairment, and exclusions for primary sleep disorders were seldom considered in sample selection for both subtypes, perhaps because diagnostic criteria that include these considerations were used with relative frequency. In contrast, the figure shows that subjective sleep impressions (i.e., self-reports, diary results), PSG findings, and comparisons of PSG and subjective sleep measures were much more common selection factors for the SSM groups than for the psychophysiologic samples. Given the nature of SSM, these latter findings admittedly are not surprising. Nonetheless, these consensual data imply the potential importance of such factors in developing RDC for this condition.

Figure 2c provides findings concerning the typical research selection/definition criteria used for the remaining ICSD diagnoses considered in the evidence tables. As was the case for the above two diagnoses, these data suggest some marked differences among these subtypes in regard to the relative importance of the selection criteria listed. PSG findings (either apneic/hypopneic events or counts of PLMs) coupled with DIMS complaints were used very frequently for selection of AI and PLMD patients, whereas specific diagnostic criteria were employed somewhat less frequently. The remaining selection criteria were used much more rarely for selecting and defining these subtypes in the studies reviewed. In contrast, insomnia complaints coupled with duration criteria were used universally to select idiopathic insomnia samples. Diagnostic criteria and exclusions for medical and sleep disorders seemed of moderate importance for sample selection/definition of this subtype. PSG data were also frequently considered in defining idiopathic samples, but mainly as a means of ruling out occult primary sleep disorders such as sleep apnea and PLMD. The remaining criteria sets were used infrequently or not at all for selecting idiopathic samples.

Derivation of Insomnia RDC

The findings derived from the literature search provide guidance in the development of a standardized definition for chronic insomnia per se as well as RDC for specific diagnostic subtypes and normal control samples. Since the literature review showed frequent use of published diagnostic criteria for insomnia subject selection, it seemed useful to begin by considering commonalities in insomnia definitions contained in the most recent of these nosologies. Both the latest DSM[6] and most recent versions of the ICSD[7, 28] systems agree that complaints of difficulty initiating sleep, difficulty maintaining sleep, and/or sleep that is nonrestorative or poor in quality constitute the sleep-related symptoms of insomnia. Furthermore, both systems require associated daytime impairment that is perceived to be the result of the nocturnal sleep symptoms. These two requirements, nocturnal sleep disturbance and associated daytime impairment, appear to repre-

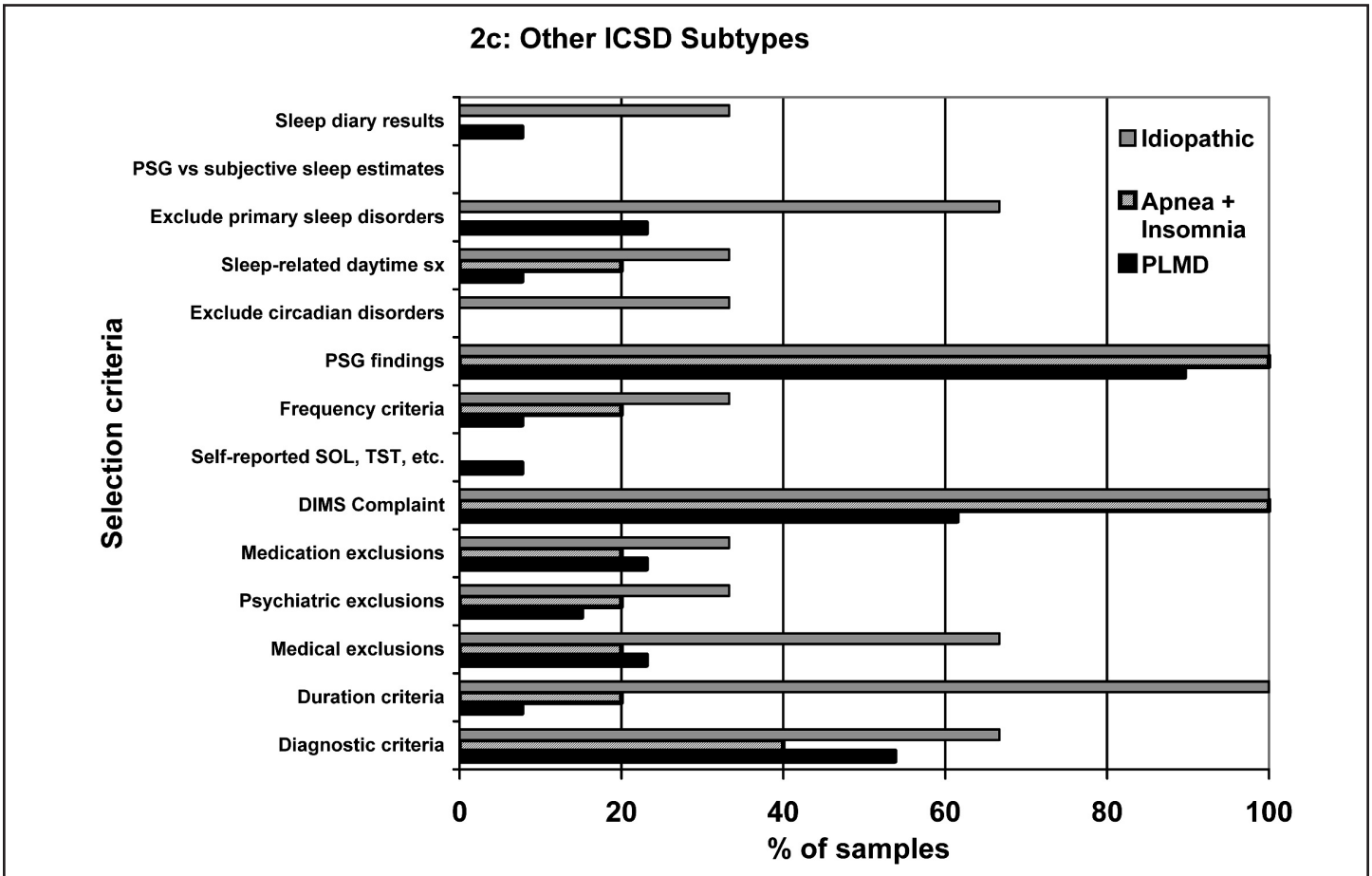


Figure 2c–Research Selection Criteria used for Defining Insomnia Subtypes. The definitions provided in the caption for Figure 1 apply to Figures 2a, 2b, & 2c.

sent universal definitional criteria that "fit" within extant insomnia nosologies and apply equally well to the various subtypes highlighted by the WG literature review.

In contrast, other frequently used definitional criteria examined do not appear of equal importance to a global insomnia definition. For example, the tabulations conducted suggest that insomnia duration may be important to the definitions of various subtypes but not for insomnia associated with PLMD or sleep apnea. Likewise, frequency criteria appear relatively important to some subtypes (e.g., SSM) but not to others. Similar statements can be made about the remaining definitional criteria examined. Hence, none of these can be applied to a universally applicable insomnia definition.

A final consideration relates to the context in which the nocturnal and diurnal insomnia symptoms occur. Although typically not stated, implicit to the term insomnia is the assumption that its associated nocturnal and diurnal symptoms arise despite a consistently adequate opportunity for sleep. That is to say, nocturnal sleep difficulties occur despite the allocation of adequate time periods and circumstances (e.g., a quiet and dark bedroom) for sleep. In this vein, the ICSD-2[189] will specifically mention this requirement in its generic insomnia definition. Given these considerations, the criteria shown are offered as universal RDC for insomnia. These criteria have been included in the ICSD-2, and the WG recommends their use for selection of adult insomnia samples by all researchers regardless of the insomnia subtype in question.

In addition to these universal criteria, additional criteria seem appropriate for the distinctive insomnia subtypes previously discussed. Results of the literature review suggest the RDC criteria sets should vary across the subtypes considered. The specific criteria sets and their justifications are provided for each subtype in the following discussion.

Research Diagnostic Criteria for Insomnia Disorder

A. The individual reports one or more of the following sleep related complaints:
   1. difficulty initiating sleep,
   2. difficulty maintaining sleep,
   3. waking up too early, or
4. sleep that is chronically nonrestorative or poor in quality.

B. The above sleep difficulty occurs despite adequate opportunity and circumstances for sleep.

C. At least one of the following forms of daytime impairment related to the nighttime sleep difficulty is reported by the individual:
   1. fatigue/malaise;
   2. attention, concentration, or memory impairment;
   3. social/vocational dysfunction or poor school performance;
   4. mood disturbance/irritability;
   5. daytime sleepiness;
   6. motivation/energy/initiative reduction;
   7. proneness for errors/accidents at work or while driving;
   8. tension headaches, and/or GI symptoms in response to sleep loss; and
   9. concerns or worries about sleep.

Primary Insomnia: This diagnosis is specific to the DSM [4-6] sleep disorders nosology. It is a fairly global diagnosis established primarily by the exclusion of other primary sleep disorders, psychiatric conditions, medical factors, and substance use/abuse as causes to the reported sleep difficulty. Given its global nature, primary insomnia subsumes several of the subtypes (e.g., PSYI, idiopathic insomnia, SSM) listed in the ICSD. These ICSD-2 subtypes are more specific and are defined by positive symptoms as well as by exclusionary criteria. As such, the utility of RDC for both primary insomnia and these more specific ICSD-2 subtypes could be questioned. However, as demonstrated by previous studies[10, 11] there is not perfect concordance between the more global primary insomnia diagnosis and the more specific ICSD-2 subtypes. For example, some patients who fail to meet criteria for any of the more specific ICSD-2 subtypes may meet criteria for primary insomnia whereas some patients who meet criteria for one of the ICSD-2 subtypes may not meet criteria for primary insomnia. In addition, there is a formidable treatment literature devoted to primary insomnia. Given these considerations, it seems reasonable to provide RDC for primary insomnia as well as for the several overlapping ICSD-2 subtypes described later.

DSM criteria for this condition include an insomnia complaint as well as a number of the exclusionary criteria highlighted by the findings shown in Figure 2a. Both the figure and the DSM texts advocate excluding medical, psychiatric, and medicinal causes of sleep disturbance. In addition, the diagnostic criteria require exclusions for other primary sleep disorders (e.g., sleep apnea, parasomnias, etc.), sleep disturbances due to unusual sleep/wake schedules or circadian rhythm disorders, and sleep difficulties arising from substance abuse. Along with these criteria, both the DSM texts and the data summarized in Figure 2a highlight the importance of duration criteria. The DSM system requires an insomnia duration of one month or longer. All six reviewed articles that used a duration criterion for primary insomnia required at least one-month duration although five of these six articles required longer durations. Nonetheless, these articles provide consensus that an insomnia duration of at least one-month is needed for this diagnosis. The following criteria incorporate these considerations in RDC for Primary Insomnia.

Research Diagnostic Criteria for Primary Insomnia

A. The individual meets the criteria for insomnia disorder.

B. The insomnia noted in A has been present for at least one month.

C. One of the following two conditions applies:
   1. There is no current or past mental or psychiatric disorder.
   2. There is a current or past mental or psychiatric disorder, but the temporal course of the insomnia shows some independence from the temporal course of the mental or psychiatric condition.

D. One of the following two conditions applies:
   1. There is no current or past sleep-disruptive medical condition.
   2. There is a current or past sleep-disruptive medical condition, but the temporal course of the insomnia shows some independence from the temporal course of the medical condition.

E. The insomnia cannot be attributed exclusively to another primary sleep disorder (e.g., sleep apnea, narcolepsy, or parasomnia) or to an unusual sleep/wake schedule or circadian rhythm disorder.

F. The insomnia cannot be attributed to a pattern of substance abuse or to use or withdrawal of psychoactive medications.

Insomnia due to a Mental Disorder: This diagnosis has been included in several editions of the DSM sleep disorders nosology. A variety of mental disorders have insomnia as a symptom, but this diagnosis is applied when insomnia arises from a mental disorder and is of sufficient severity or concern to the patient as to require separate attention in treatment. Traditionally, the ICSD system has included several subtypes of insomnias that can be subsumed within this more global DSM category. However, the ICSD-2 has adopted the DSM approach and includes the global category of Insomnia due to a Mental Disorder rather than several specific mental disorder subtypes described in the previous ICSD. As such, development of RDC for insomnia due to a mental disorder has applicability to both DSM and the current ICSD-2 classification systems.

Figure 2a suggests strong adherence to published diagnostic criteria for this insomnia subtype by previous researchers. The condition's DSM criteria include a complaint of insomnia. The criteria and Figure 2a also highlight the importance of exclusions for sleep-disruptive medical conditions and medications that disrupt sleep. In addition, the DSM criteria emphasize the relationship between the insomnia and the comorbid mental disorder and include exclusions for substance abuse and such sleep disorders as narcolepsy, breathing-related sleep disorders and parasomnias as causative factors. In regard to this latter set of exclusions, Figure 2a suggests that sleep schedule and circadian disorders may warrant exclusionary attention as well. Furthermore, the criteria emphasize the relative prominence and importance of the insomnia to distinguish this subtype from mental disorders wherein insomnia is a common albeit less central symptom.

Along with these considerations, both the DSM texts and Figure 2a suggest the importance of duration criteria for this subtype. DSM-IV criteria require a minimum insomnia duration of one month. The findings presented in Figure 2a represent five instances in which a minimum duration criterion was used, and in none of these cases was a duration of less than one month used. As such, a duration of one-month seems appropriate as a minimum criterion for this condition although longer time periods may be helpful in ascertaining the association/co-variation of insomnia and its causative mental disorder. Given these considerations the criteria shown are offered as RDC for Insomnia due to a Mental Disorder.

Psychophysiologic Insomnia: This diagnosis is specific to the ICSD system [7, 28, 189] and is one of the subtypes that may be subsumed within the more global DSM diagnosis of primary insomnia. Unlike primary insomnia, PSYI is defined by positive symptoms reflective of somatized tension and conditioned arousal as well as by several exclusionary criteria. Admittedly, there is debate[190, 191] about the utility of subdividing primary insomnia into this and other subtypes, and there is a need for much more diagnostic reliability and validity studies to settle this controversy. However, the evidence tables discussed previously provide some limited support for subdividing primary insomnia into a number of ICSD subtypes. As such, RDC for PSYI and other selected ICSD subtypes seem needed to systematize study of these subtypes so that their utility vis a vis the DSM global diagnosis of primary insomnia can be ascertained.

As suggested by Figure 2b, published diagnostic criteria frequently have been used to identify or select individuals for studies of PSYI. Central to the ICSD criteria[7, 189] for this condition are evidence of sleep-preventing associations and somatized tension/arousal that disrupt sleep. The ICSD criteria for PSYI also incorporate the above-mentioned insomnia definition and, along with the findings shown in Figure 2b, highlight the importance of exclusions for sleep-disruptive medical and psychiatric conditions as the sole cause of the insomnia. Not mentioned in the criteria for this condition are exclusions for sleep-disruptive medications, yet Figure 2b suggests such exclusions have been used with relative frequency in previous studies of this subtype. Neither the ICSD criteria nor the findings summarized in Figure 2b argue for the exclusion of co-morbid sleep disorders with the possible exception of circadian rhythm disorders. In fact, the diagnostic criteria allow for coexisting sleep disorders such as obstructive sleep apnea presumably if the observed sleep problem cannot be explained solely by this coexisting condition.

In regard to duration, Figure 2b suggests that duration criteria have been used with relative frequency in research studies concerning this subtype. In fact, duration criteria were used for the selection of 8 of the 21 psychophysiologic samples identified in the articles reviewed. Minimum insomnia duration required for these samples was variable and included three months, six months and twelve months. Despite these findings, ICSD allowed for the diagnosis of *acute* PSYI in cases where the insomnia is present for less than four weeks. Nonetheless, it seems that some period of time would be required to allow for the development of the conditioned arousal that defines this condition. Hence, as a compromise between research practice and ICSD, the WG proposes requiring a minimum insomnia duration of one-month for clear identification of established cases of this

Research Diagnostic Criteria for Insomnia due to a Mental Disorder

A. The individual meets the criteria for insomnia disorder.
B. The insomnia noted in A has been present for at least one month.
C. There is an association between the insomnia and a co-existing DSM-IV-TR-defined mental disorder as reflected by both of the following:
   1. The onset of the insomnia coincides with the onset of the associated mental disorder.
   2. The temporal course of the insomnia coincides with the temporal course of the mental disorder.
D. The insomnia is either the sole complaint or is sufficiently severe to warrant separate clinical attention.
E. One of the following two conditions applies:
   1. There is no current or past sleep-disruptive medical condition.
   2. There is a current or past sleep-disruptive medical condition, but the temporal course of the insomnia shows some independence from the temporal course of the medical condition.
F. The insomnia cannot be attributed exclusively to another primary sleep disorder (e.g., sleep apnea, narcolepsy or parasomnia) or to an unusual sleep/wake schedule or circadian rhythm disorder.
G. The insomnia cannot be attributed to a pattern of substance abuse nor to use or withdrawal of psychoactive medications.

Research Diagnostic Criteria for Psychophysiological Insomnia

A. The individual meets the criteria for insomnia disorder.
B. The insomnia noted in A has been present for at least one month.
C. The patient has evidence of conditioned sleep difficulty and/or heightened arousal in bed as indicated by one or more of the following:
   1. Excessive focus on and heightened anxiety about sleep.
   2. An inability to fall asleep in bed at the desired bedtime or during planned naps but relative ease falling asleep during other relatively monotonous activities (e.g., watching TV, reading, etc.) when not intending to sleep.
   3. Being able to sleep better away from home than at home.
   4. Mental arousal in bed characterized either by intrusive thoughts or a perceived inability to volitionally cease sleep-preventing mental activity.
   5. Heightened somatic tension in bed reflected by a perceived inability to relax the body sufficiently to allow the onset of sleep.
D. One of the following two conditions applies:
   1. There is no current or past mental disorder.
   2. There is a current or past mental disorder, but the temporal course of the insomnia shows some independence from the temporal course of the mental disorder.
E. One of the following two conditions applies:
   1. There is no current or past sleep-disruptive medical condition.
   2. There is a current or past sleep-disruptive medical condition, but the temporal course of the insomnia shows some independence from the temporal course of the medical condition.
F. The insomnia cannot be attributed solely to another primary sleep disorder (e.g., sleep apnea, narcolepsy, or parasomnia) or to an unusual sleep/wake schedule or circadian rhythm disorder.
G. The insomnia cannot be attributed to a pattern of substance abuse or to use or withdrawal of psychoactive medications.

condition. This duration criterion appears in ICSD-2 criteria for Psychophysiologic Insomnia and has been integrated into the following RDC for this condition.

Paradoxical Insomnia (*Sleep State Misperception)*: This condition represents another ICSD subtype that can be subsumed within the global DSM diagnosis of primary insomnia. It has been assigned various names including *insomnia complaint without objective findings*, *pseudo-insomnia*, *subjective insomnia*, and most recently, *sleep state misperception*. Each of these terms connotes an absence of actual sleep pathology despite the presence of an insomnia complaint. However, the origins of sleep complaints in patients with this condition remain poorly understood, although some recent studies[192, 193] suggest that excessive high frequency activity in patients' sleep EEGs may relate to their subjective sleep dissatisfaction. Accordingly, this condition has been renamed Paradoxical Insomnia in the ICSD-2 to avoid the negative connotations of its previous names. As the evidence tables provide some support for this subtype, RDC are offered to systematize future study of this condition.

As reflected by Figure 2b, the types of criteria required for this condition are qualitatively different from those required for the other subtypes considered thus far. Published diagnostic criteria[7] have been used less frequently for sample definition/selection for this subtype than for PSYI. However, duration criteria, as well as exclusions for medication, medical disorders, and psychiatric conditions have figured very prominently in sample definition. In addition, the studies reviewed show frequent use of frequency criteria, self-reports of sleep/wake measures, PSG findings, and comparisons of subjective sleep estimates with PSG data to define research participants with this condition.

In the studies reviewed, duration criteria were used for selection/definition of 9 of 14 samples, and in all of these cases, a minimum insomnia duration of six months was used. However, ICSD allows diagnosis of acute versions of this disorder in cases having insomnia complaints for less than one month. Nonetheless, the WG surmised it would be difficult to be certain about this diagnosis in the acute phase. As such, a minimum duration of one month was selected as a criterion so as to compromise between ICSD criteria and previous research practice.

Criteria related to the frequency (number of nights per week) of insomnia were used as selection criteria for 7 of the 14 samples with this condition. When frequency criteria were used, an insomnia occurring > 3 nights per week was required. However, it should be noted that the ICSD and ICSD-2 require no specific minimum frequency of sleep difficulties for diagnosis of this condition. Furthermore, frequency criteria were generally not viewed as important for the other ICSD primary insomnia subtypes (i.e., PSYI, & COI). As a consequence, frequency criteria were not included in the proposed RDC for this condition, although it is recognized that future research may show that these RDC can be improved by the addition of such criteria.

In regard to PSG criteria, both the ICSD and a review of relevant studies suggest that sleep recordings show no evidence of other sleep disorders that may account for the insomnia complaint. The original ICSD system [7]suggests "sleep latencies of less than 15 to 20 minutes and sleep durations in excess of 6fi hours" are characteristic PSG findings among patients with this condition. To corroborate this statement, PSG findings were extracted from all reviewed studies that included SSM samples. PSG values of SOL were reported for seven samples totaling 73 subjects, whereas values of TST were available for five samples totaling 48 patients. These data were used to compute weighted averages of SOL and TST by multiplying the mean values reported for each sample by the sample size, summing the results of these calculations, and then dividing the result by the total number of subjects included in the samples considered. Results of these calculations produced a weighted mean SOL of 22.8 minutes and a weighted mean TST of 415.5 minutes. These findings suggest that a minimum TST requirement of 6fi hours may be reasonable for definition of paradoxical insomnia, but a 20-minute SOL may be an overly strict requirement.

In addition to these measures, several articles used a minimum sleep efficiency value as a defining feature of this diagnosis. Sleep efficiencies of ≥ 85% or 90% were most commonly used for selection of subjects with this condition. A review of relevant articles produced mean values of sleep efficiency for four separate samples totaling 33 subjects. Computation of a weighted average produced a mean sleep efficiency value of 92.2% for these samples. Whereas these findings suggest either efficiency requirement could be employed, it should be noted that the value of 85% has commonly been regarded as normal in the insomnia literature.[194] Therefore, use of this more lenient cutoff may represent a more practical requirement for defining paradoxical insomnia.

A final consideration regarding definition of this insomnia subtype pertains to the observed mismatch between the patient's reported and recorded sleep. The studies reviewed used several different strategies to attempt to operationalize this discrepancy. One strategy required pathologically high values of SOL or WASO derived from self-report or sleep logs in subjects who show "normal" PSG measures of these parameters. Another strategy required subjects to overestimate SOL or underestimate TST by a pre-determined degree. For example, Dorsey and Bootzin[56] assigned SSM diagnoses to those who overestimated PSG determined SOL by 150% or more. However, the articles reviewed showed no consistent strategy for operationalizing this mismatch. As such, ascertaining complaints of insomnia and/or pathologic values of SOL or WASO on sleep logs in individuals who meet the above described PSG criteria may represent the most pragmatic approach for identification of paradoxical insomnia at this juncture. The RDC shown for this condition assimilate these various considerations.

Idiopathic Insomnia: This condition represents a third ICSD subtype that generally may be subsumed within the global DSM category of primary insomnia. Whereas the WG found few articles that included samples of COI, these articles provided some guidance in the development of RDC. Figure 2c shows that use of published diagnostic criteria, duration criteria, ascertainment of specific insomnia symptoms (e.g., onset, maintenance, or quality complaint), and PSG findings appear to be the most prominent selection criteria used. Included among the published criteria set is the requirement of childhood onset. The articles reviewed all included adult samples. As such, the duration criteria used in this limited number of studies appeared designed to assure a childhood onset, and one[82] of these articles specifically required an insomnia onset before age 10. Although ICSD criteria allowed the presence of comorbid sleep disorders, the few articles reviewed used PSG to exclude patients with sleep disorders such as sleep apnea and periodic limb movements.

Figure 2c shows exclusions for medications and psychiatric conditions were infrequently employed, but more attention was given to exclusions for medical conditions. ICSD criteria allow for coexisting medical and psychiatric conditions and provide no exclusionary criteria for current medication use or use/abuse of substances. However, these criteria state, " No medical or mental disease can explain the early onset of insomnia." On the basis of WG findings and published criteria the RDC shown here are offered for COI.

Research Diagnostic Criteria for Paradoxical Insomnia

A. The individual meets criteria for insomnia disorder.
B. The insomnia noted in A has been present for at least one month.
C. Nocturnal polysomnography shows a sleep time ≥ 6fi hours and a sleep efficiency ≥ 85%.
D. One or more of the following applies:
   1. The individual reports a chronic pattern of little or no sleep most nights with rare nights during which relatively normal amounts of sleep are obtained.
   2. Sleep log data during one or more weeks of monitoring show an average sleep time well below published age-adjusted normative values, often with no sleep at all indicated for several nights per week. Typically there is an absence of daytime naps following such nights.
   3. The individual shows a consistent, marked mismatch between objective findings from polysomnography and subjective sleep estimates.
E. The daytime impairment reported is consistent with that reported by other insomnia subtypes, but it is much less severe than expected given the extreme level of sleep deprivation reported. There is no report of intrusive daytime sleep episodes, disorientation, or serious mishaps due to marked loss of alertness/vigilance.
F. One of the following applies:
   1. There is no current or past mental disorder.
   2. There is a current or past mental disorder, but the temporal course of the insomnia shows some independence from the temporal course of the mental condition.
G. One of the following applies:
   1. There is no current or past sleep-disruptive medical condition.
   2. There is a current or past sleep-disruptive medical condition, but the temporal course of the insomnia shows some independence from the temporal course of the medical condition.
H. The insomnia cannot be attributed solely to another primary sleep disorder (e.g., sleep apnea, narcolepsy, or parasomnia) or to an unusual sleep/wake schedule or circadian rhythm disorder.
I. The insomnia cannot be attributed to a pattern of substance abuse or to use or withdrawal of psychoactive medications.

Insomnia Related to PLMD: This condition is specific to the ICSD system; no specific analogous diagnosis is included in the DSM system. The WG found a number of studies containing individuals whose insomnia was attributed to PLMD. It is recognized that there is an unresolved debate[120] as to whether periodic limb movements are a cause of insomnia or merely an epiphenomenon of sleep disruption or insomnia *per se*. Since further research to clarify the clinical usefulness of this diagnosis is needed to resolve this controversy, RDC for this condition would currently seem useful.

Figure 2c shows that PSG findings and a specific insomnia complaint were the two most common definitional criteria in the articles reviewed. In fact, these two selection criteria were cited more commonly for subject selection than were specific diagnos-

tic criteria. Admittedly this finding is, in part, artifactual because only articles that specifically linked a finding of PLMD and insomnia were selected for review. Nonetheless, these two components may indicate the most salient features of this condition in that Figure 2c shows relative infrequent use of the remaining selection criteria. However, it should be noted that the ICSD criteria exclude medical disorders, mental conditions and coexisting sleep disorders as explanations for presenting complaint.

Research Diagnostic Criteria for Idiopathic (Childhood Onset) Insomnia

A. The individual meets the criteria for insomnia disorder.
B. The insomnia noted in A began during childhood (i.e., before age 10) without an identifiable precipitant.
C. The insomnia has been persistent and unrelenting since its onset.
D. One of the following applies:
   1. There is no current or past mental disorder.
   2. There is a current or past mental disorder, but the temporal course of the insomnia shows some independence from the temporal course of the mental condition.
E. One of the following applies:
   1. There is no current or past sleep-disruptive medical condition.
   2. There is a current or past sleep-disruptive medical condition, but the temporal course of the insomnia shows some independence from the temporal course of the medical condition.
F. The insomnia cannot be attributed solely to another primary sleep disorder (e.g., sleep apnea, narcolepsy, or parasomnia) or to an unusual sleep/wake schedule or circadian rhythm disorder.
G. The insomnia cannot be attributed to a pattern of substance abuse or to use or withdrawal of psychoactive medications.

PLMD should be clearly distinguished from restless legs syndrome (RLS), which is itself a very common cause of insomnia. Periodic limb movements of sleep (PLMS) accompany RLS in the majority of patients, but by definition the term PLMD is only used in ICSD-2 when RLS is not present. In the past, confusion between RLS, PLMS and PLMD have led to difficulty in assessing the inclusion criteria of many research studies.

Individuals with Insomnia Related to PLMD have been identified primarily by specific PSG indices of periodic limb movement activity. Methods for quantifying this activity in the articles reviewed typically refer to the method originally suggested by Coleman.[52] The ICSD requires a rate of 5 PLMs per hour of sleep for a diagnosis of PLMD. The updated ICSD-2 requires a rate of 5 PLMs per hour in children and, in most adult cases, a rate of 15 per hour, but cautions that the PLMS rate must be interpreted in the context of a patient's sleep-related complaint. A review of the 14 articles that included PLMD samples showed specific mention of a 5 PLMs per hour cutoff in four articles, whereas no specific PLM criterion was reported in the remaining articles. Recognizing that there is not adequate data to establish any absolute cut-off for PLMS rate, and in order to assure some consistency in future PLMD studies, a minimum of 5 PLMS per hour in children and 15 PLMS per hour in adults has been included as a minimum criterion in the following RDC for insomnia related to PLMD.

Research Diagnostic Criteria for Insomnia Related to Periodic Limb Movement Disorder

A. The individual meets criteria for insomnia disorder.
B. Polysomnographic monitoring shows stereotyped limb movements that:
   1. are one half second to 5 seconds in duration,
   2. are of amplitude greater than or equal to 25% of toe dorsiflexion during calibration,
   3. occur in a sequence of four or more movements, and
   4. are separated by more than five seconds and less than 90 seconds.
C. The PLMS Index based on the scoring criteria in B exceeds five per hour in children and 15 per hour in most adult cases.
D. Other coexisting sleep disorders, including RLS, cannot account either for the insomnia noted in A or the PLM activity.

Insomnia Related to Sleep Apnea: Both the DSM and ICSD systems suggest insomnia may be attributable to sleep disordered breathing. However, the extent to which IA represents a useful clinical diagnosis has been debated. Some[105] have argued that sleep apnea is a common occult cause of insomnia complaints, whereas others[195] report that insomnia arising from sleep apnea is relatively uncommon. Since this controversy remains unresolved, RDC to allow further study the utility of this diagnosis seem warranted.

As was the case for PLMD-related insomnia, articles selected for review concerning this condition were chosen because they specified a presence of both insomnia and sleep apnea in the subjects described. Many articles included samples of apnea sufferers, but these had to be excluded because they were composed primarily of hypersomnolent individuals. As a result of our selection process, Figure 2c shows that PSG findings and insomnia symptoms were the two universally used criteria for the few samples representing this subtype considered. However, the published DSM and ICSD criteria and the findings shown in Figure 2c suggest that other selection criteria are important in defining this subtype.

Unfortunately the PSG definitional criteria for IA were not consistently defined in the studies considered. Only five articles specifically examined insomnia in the context of sleep apnea, and several of these were case reports published prior to 1980. One such paper[77] suggests the use of a minimum of "30 apneic episodes per recording night"; this index appears very crude by the recent consensus apnea definition.[14] A more recent paper[173] used a respiratory disturbance index (RDI) of $\geq$10 apneas + hypopneas per hour of sleep in selected apnea sufferers from a group of individuals with insomnia complaints, but required an RDI < 5 to identify insomnia sufferers with no apnea. It seems noteworthy that an RDI = 5 is the minimum RDI listed in the ICSD and updated ICSD-2 diagnostic criteria for sleep apnea. Given the limited articles devoted to this insomnia subtype and the lack of consensus regarding an RDI cutoff, this currently may represent the best consensual criterion to use for identifying Insomnia Related to Sleep Apnea. Hence, this RDI criterion is included in the following RDC set proposed.

Research Diagnostic Criteria for Insomnia Related to Sleep Apnea.

A. The individual meets criteria for insomnia disorder.
B. Nocturnal polysomnographic recording shows ≥ 5 respiratory events that meet current definitional criteria for apneas, hypopneas or respiratory effort-related arousals per hour of sleep.
C. Sleep-disruptive medical conditions, mental disorders, and any coexisting sleep disorders can not totally account for the insomnia noted in A.

Other insomnia subtypes: The literature provided little information about other specific insomnia subtypes. Both the DSM-IV and ICSD texts provide diagnoses for insomnias resulting from a medical disorder and insomnias related to substance use/abuse. The more recent DSM and ICSD-2 texts delineate global diagnoses for labeling these two conditions. Hence, additional RDC appear warranted for these global insomnia diagnoses.

Although WG members reviewed many articles concerning the study of sleep in various medical conditions (e.g., Parkinson's Disease, chronic pain), these studies characteristically failed to confirm the presence of insomnia complaints in the samples examined. Similarly, many studies of sleep disturbance related to substance abuse/exposure were found but, once again, these studies failed to document coincident insomnia complaints. Nonetheless, the WG consensus supports the notion that both medical conditions and use of or exposure to certain substances may give rise to insomnia. To encourage studies of the reliability, validity, course, and treatment requirements of these conditions, the criteria sets shown here are offered as provisional RDC for these disorders.

Insomnia due to Medical Condition

A. The individual meets criteria for insomnia disorder.
B. The insomnia is present for at least one month.
C. There is an association between the insomnia and a co-existing medical disorder as reflected by both of the following:
   1 The onset of the insomnia coincides with the onset of the associated medical disorder.
   2 The temporal course of the insomnia coincides with the temporal course of the medical disorder.
   (Note: Researchers should identify the specific medical disorder(s) causing the insomnia)
D. The insomnia is either the sole complaint or is sufficiently severe to warrant separate clinical attention.
E. One of the following two conditions applies:
   1. There is no current or past sleep-disruptive mental condition.
   2. There is a current or past sleep-disruptive mental condition, but the temporal course of the insomnia shows some independence from the temporal course of this mental condition.
F. The insomnia cannot be attributed exclusively to another primary sleep disorder (e.g., sleep apnea, narcolepsy, or parasomnia) or to an unusual sleep/wake schedule or circadian rhythm disorder.
G. The insomnia cannot be attributed to substance abuse or to use or withdrawal of psychoactive medications.

Insomnia due to Drug or Substance

A. The individual meets criteria for insomnia disorder.
B. The insomnia is present for at least one month.
C. One of the following applies:
   1. There is current ongoing dependence on or abuse of a drug or substance known to have sleep-disruptive properties either during periods of use/intoxication or during periods of withdrawal; or

   2. The patient has current ongoing use of or exposure to a medication, food, or toxin known to have sleep-disruptive properties in susceptible individuals.
D. There is an association between the insomnia and substance use/abuse/exposure as reflected by both of the following:
   1. The onset of the insomnia coincides with the onset of the substance use/abuse/exposure or withdrawal.
   2. The temporal course of the insomnia coincides with the exposure to, use of, or withdrawal from the substance.
   (Note: Researchers should specify the substance and whether the insomnia occurs during intoxication, chronic use, or substance withdrawal.)
E. One of the following two conditions applies:
   1. There is no current or past sleep-disruptive mental condition.
   2. There is a current or past sleep-disruptive mental condition, but the temporal course of the insomnia shows some independence from the temporal course of this mental condition.
F. One of the following two conditions applies:
   1. There is no current or past sleep-disruptive medical disorder.
   2. There is a current or past sleep-disruptive medical disorder, but the temporal course of the insomnia shows some independence from the temporal course of the comorbid medical condition.
G. The insomnia cannot be attributed exclusively to another primary sleep disorder (e.g. sleep apnea, narcolepsy, or parasomnia) or to an unusual sleep/wake schedule or circadian rhythm disorder.

Normal Sleepers: Current diagnostic manuals provide no guidance for definition of normal sleeper controls. Nonetheless, normal controls are an essential part of research concerning insomnia subtypes. The data presented in Figure 1 highlight common research practice in regard to characterization of these individuals. These data show that exclusions for sleep-disruptive medical conditions, mental disorders, and psychoactive medications were used more frequently than other criteria. Less frequently, PSG findings, self-reported sleep estimates, and exclusions for specific sleep disorders or unusual sleep wake schedules/circadian disturbances, were considered in selecting normal samples. In addition, anecdotal information from a majority of the articles suggested that a lack of any current sleep complaint represents a defining criterion for such samples. Based on these observations the RDC shown are offered as universal criteria for identifying normal sleepers for insomnia research.

Research Diagnostic Criteria for Normal Sleepers (Controls)

A. The individual has no complaints of sleep disturbance or daytime symptoms attributable to unsatisfactory sleep.
B. The individual has a routine standard sleep/wake schedule characterized by regular bedtimes and rising times.
C. There is no evidence of a sleep-disruptive medical or mental disorder.
D. There is no evidence of sleep disruption due to a substance exposure, use, abuse, or withdrawal.
E. There is no evidence of a primary sleep disorder.

Methods of Assessment

The final aim of this project was to propose specific methods for documenting the presence/absence of the RDC among patients/subjects to which they are applied. The findings summarized in Figures 1 and 2 as well as procedural information provided in the articles reviewed provide ample guidance for addressing this aim. Considering this information along with the precision required by the internal and external validity demands of the research venue, the WG offers the following methodological guidelines for insomnia RDC ascertainment.

Interview Assessment: Use of the clinical interview to ascertain insomnia symptoms and specific diagnoses was found to be a ubiquitous practice in the articles reviewed. In most instances, it appeared that findings and resulting opinions derived from a single interviewer using a standard clinic interview served as the basis of diagnostic ascertainment. Given the very modest inter-rater agreement found by Buysse et al.[12] (Table 2) for discerning global insomnia diagnoses by this method, it is clear that more reliable interview methods are desirable for the research setting. Specifically, reliable interview methods that assure consistent RDC ascertainment across clinicians and settings are needed to standardize insomnia research.

In this regard, the WG was able to identify two promising approaches. The first method, derived from those studies listed in Table 2, is that of using independent interviewers to ascertain the presence of insomnia RDC in prospective research candidates. One manner of applying this approach would be to select only those study subjects who, in the mutual opinion of independent interviewers, meet RDC for the insomnia diagnosis of interest. Alternately, a researcher could choose to demonstrate high inter-rater reliability between two or more interviewers during subject screening for the insomnia RDC in question. Use of this latter alternative would require that only a randomly selected subset of study enrollees are used to test reliability for RDC. A second method would involve the use of structured interview methodology to assure that standardized interview questions targeting essential diagnostic information are administered consistently across research candidates. Examples of this methodology are the Structured Interview for Sleep Disorders described by Schramm et al.[163] and the computer-driven Sleep Eval system described by Ohayon et al.[138] Although neither of these instruments is specifically structured to ascertain the RDC provided herein, they serve as models for the development of an instrument for reliable assessment of these criteria sets.

Polysomnography: The RDC for such subtypes as SSM, IA and PLMD require specific information from polysomnographic (PSG) recording. The WG found that PSG was often used in studies of other insomnia subtypes to rule out the presence of occult primary sleep disorders. Although PSG is not considered essential for the clinical assessment of insomnia[196], its use in establishing the insomnia RDC presented herein seems desirable. Thus, the WG recommends incorporating at least a single night of PSG into the screening process to ascertain insomnia RDC using established methods for sleep staging and event scoring.[197-200]

Sleep Diaries/Logs: Sleep diaries or logs were used sparingly in the studies reviewed for this project. In fact, only in the case of paradoxical insomnia/sleep state misperception were sleep diaries/logs used commonly, most often to assess the degree of mismatch between subjective and objective sleep measures. In such comparisons, there appeared to be no well accepted standard for confirming the diagnosis, but there seems to be some consensus that ascertaining markedly inaccurate estimates of sleep through logs or diaries is useful. The WG also recognizes the reputed[201] usefulness of sleep logs or diaries for the assessment of insomnia complaints *per se*. As such, they are considered useful in helping to establish the presence of an insomnia disorder in general. However, their usefulness for ascertaining RDC for many of the subtypes discussed will require further exploration.

Other Assessment Procedures: The WG review identified a number of additional objective and subjective measures used to assess and compare insomnia subtypes. Among the objective measures were assessments of sleep/wake motor activity (actigraphy), heart-rate variability, metabolic rate, and melatonin levels. Subjective measures were derived from both widely used and relatively obscure personality/mood questionnaires. Whereas all of these measures may provide useful information about insomnia subtypes, their utility for differentiation of insomnia subtypes has yet to be established. Thus, such additional instruments have not been included as requirements for RDC assessment.

DISCUSSION

The scientific literature includes thousands of articles concerning insomnia. Nonetheless, results of the WG's targeted literature review showed a paltry amount of research providing support of the insomnia diagnoses included in past and current sleep disorders nosologies.[4-7, 27, 29, 189] There have been astonishingly few studies designed to assess the reliability of any of these diagnostic categories, and no reliability information is available for many of the insomnia subtypes delineated in the most recent DSM[6] and ICSD[28, 189] manuals. The number of studies supporting the validity of such subtypes is, at best, only slightly more encouraging. Such findings not only make the development of research diagnostic criteria difficult, but also call into question much of the body of literature devoted to insomnia in general. Clearly, much more attention needs to be devoted to confirming the reliability and validity of the insomnia subtypes our nosologies describe if the field is to move forward.

The RDC proposed are not intended to limit the clinical assessment of insomnia patients, and we acknowledge that alternative approaches may be preferable to the RDC in non-research settings. However, the insomnia RDC should be regarded as a starting point for improving insomnia research and documenting

whether currently recognized methods of insomnia classification are optimal or need change. These RDC hopefully will discourage additional studies of poorly characterized insomnia samples that provide us little insight into the pathology and specific treatment needs of the distinctive subtypes commonly encountered in clinical venues. Furthermore, these RDC should help standardize insomnia research and facilitate the reliability and validity studies that are so sorely needed to determine if an alternate insomnia classification system may be desirable. However, the RDC should not be regarded as eternally fixed definitions. They represent initial efforts toward standardizing insomnia research, but they may undergo refinements as a function of both the degree to which they prove useful and the findings of future insomnia research.

To assist with future revisions/improvements in these preliminary RDC, it is recommended that insomnia researchers consistently report the following information in their published studies:

- The methods of recruitment (media announcements vs. clinical contacts) and types of individuals (research volunteers vs. clinical patients) enrolled in the study.
- Means, standard deviations, and ranges of common sleep measures such as total sleep time (TST), sleep onset latency (SOL), wake time after sleep onset (WASO), and sleep efficiency for each diagnostic group included in the study sample as well as any quantitative cutoffs (e.g., onset latency > 30 minutes) in such measures used for sample selection. When subjective sleep measures are reported, the source (e.g., sleep diary, questionnaire, clinical interview) of these data should be specified.
- The mean, standard deviation, and distribution of insomnia duration for each diagnostic group included in the study sample.
- The mean and distribution of insomnia frequency (number of nights per week of insomnia) for each diagnostic group in the study sample.
- Means and standard deviations of the discrepancies between subjective estimates and objective measures of TST, SOL, and WASO for samples of SSM and other diagnostic groups included in the study.
- Means and standard deviations of leg movement and respiratory disturbance indices for insomnia samples meeting RDC for insomnia related to PLMD or sleep apnea.

Researchers also are encouraged to assign applicable ICSD diagnoses (i.e., psychophysiologic, paradoxical, idiopathic insomnia subtypes) to study participants who meet RDC for primary insomnia and report frequency, duration, and quantitative sleep measures for each subtype separately. This information will help determine the utility of the ICSD subtypes over the more global primary insomnia diagnosis.

Admittedly, there are several limitations of the methodology used herein that should be considered. The literature reviewed excluded most insomnia treatment studies in which the efficacy of one or more insomnia therapies was evaluated with single insomnia subtypes. Such studies were excluded since they provided no useful empirical information (reliability/validity data) for addressing the aims of this RDC project. Nonetheless, the usefulness of the RDC proposed will need to be established for such studies if these criteria sets are to prove viable. It is also recognized that the literature review did not include relevant studies published after June 2000. Although the original time line for this project would have resulted in publication of this report at a time more proximal to that date, the complexity of the data collection and entry process delayed this final report. Since the WG considered insomnia literature published over a period exceeding 35 years, it is not likely that the most recent publications would have significantly altered the trends noted. Nevertheless, the findings reported should be considered in the context of the most recent insomnia literature. Finally, it should be noted that the proposed RDC are, in part, based on published evidence but also reflect consensus opinion of the WG panel of insomnia "experts." Whereas the limited informative literature obviated a purely evidence-based set of RDC, future refinements of these criteria sets hopefully will be made largely on empirical grounds.

REFERENCES

1. Feighner JP, Robins E, Guze SB, Woodruff RA, Jr., Winokur G, Munoz R. Diagnostic criteria for use in psychiatric research. Arch Gen Psychiatry. 1972;26(1):57-63.
2. Spitzer R, Endicott J, Robins E. Research diagnostic criteria: rationale and reliability. Arch Gen Psychiatry. 1978;35(6):773-82.
3. American Psychiatric Association. Diagnostic and statistical manual of mental disorders, 3rd edition. Washington, DC: American Psychiatric Association; 1981.
4. American Psychiatric Association. Diagnostic and statistical manual of mental disorders. 3rd edition, revised. Washington, DC: American Psychiatric Association; 1987.
5. American Psychiatric Association. Diagnostic and statistical manual of mental disorders (DSM-IV), 4th Edition Washington, DC: American Psychiatric Association; 1994.
6. American Psychiatric Association. Diagnostic and statistical manual of mental disorders, Fourth Edition, Text Revision (DSM-IV-TR®) Arlington, VA: American Psychiatric Publishing; 2000.
7. American Sleep Disorders Association International Classification of Sleep Disorders Rochester, MN: American Sleep Disorders Association; 1990.
8. World Health Organization. The ICD-10 classification of mental and behavioural disorders. Geneva: World Health Organization; 1993.
9. World Health Organization. ICD-9-CM: International classification of diseases, clinical modification, 4th Edition, 9th Revision Salt Lake City: Medicode; 1994.
10. Buysse D, Reynolds 3rd C, Hauri P, et al. Diagnostic concordance for DSM-IV sleep disorders: a report from the APA/NIMH DSM-IV field trial. American Journal of Psychiatry. 1984;151(9):1351-60.
11. Ohayon M, Roberts R. Comparability of sleep disorders diagnoses using DSM-IV and ICSD classifications with adolescents. Sleep. 2001;24(8):920-925.
12. Buysse D, Reynolds 3rd C, Kupfer D, et al. Clinical diagnoses in 216 insomnia patients using the International Classification of Sleep Disorders (ICSD), DSM-IV and ICD-10 categories: a report from the APA/NIMH DSM-IV Field Trial. Sleep. 1994;17(7):630-637.
13. Nowell PD, Buysse DJ, Reynolds CF, 3rd, et al. Clinical factors contributing to the differential diagnosis of primary insomnia and insomnia related to mental disorders. American Journal of Psychiatry. 1997;154(10):1412-6.
14. The Report of an American Academy of Sleep Medicine Task Force. Sleep-Related Breathing Disorders In Adults: Recommendations For Syndrome Definition And Measurement Techniques In Clinical Research. Sleep. 1999;22(5):667-89.
15. Allen R, Picchietti D, Hening W, et al. Restless legs syndrome: diagnostic criteria, special considerations, and epidemiology. A report from the restless legs syndrome diagnosis and epidemiology workshop at the National Institutes of Health. Sleep Medicine. 2003;4(2):101-119.
16. Quera-Salva M, Orluc A, Goldenberg F, Guilleminault C. Insomnia and use of hypnotics: study of a French population. Sleep. 1991;14(5):386-391.
17. Klink M, Quan S, Kaltenborn W, Lebowitz M. Risk factors asso-

ciated with complaints of insomnia in a general adult population. Influence of previous complaints of insomnia. Arch Intern Med. 1992;152(8):1634-1637.
18. Leger D, Guilleminault C, Dreyfus J, Delahaye C, Paillard M. Prevalence of insomnia in a survey of 12,778 adults in France. J Sleep Res. 2000;9(1):35-42.
19. Breslau N, Roth T, Rosenthal L, Andreski P. Sleep disturbance and psychiatric disorders: a longitudinal epidemiological study of young adults. Biol Psychiatry. 1996;39(6):411-8.
20. Ohayon M, Caulet M, Guilleminault C. How a general population perceives its sleep and how this relates to the complaint of insomnia. Sleep. 1997;20(9):715-23.
21. Ohayon M. Prevalence of DSM-IV diagnostic criteria of insomnia: distinguishing insomnia related to mental disorders from sleep disorders. J Psychiatr Res. 1997;31(3):333-46.
22. Ohayon MM. Prevalence, diagnosis, and treatment of chronic insomnia in the general population. Proceedings of the Satellite Symposium New Developments in the Treatment of Insomnia - Do they Really Have Any Impact on the Primary Health Care Setting?: Medical Forum International; in press.
23. Ohayon M, Caulet M, Priest R, Guilleminault C. DSM-IV and ICSD-90 insomnia symptoms and sleep dissatisfaction. Br J Psychiatry. 1997;171:382-8.
24. Ohayon M, Hong S. Prevalence of insomnia and associated factors in South Korea. J Psychosom Res. 2002;53(1):593-600.
25. Ohayon M, Partinen M. Insomnia and global sleep dissatisfaction in Finland. J Sleep Res. 2002;11(4):339-46.
26. Ohayon M. Epidemiology of insomnia: what we know and what we still need to learn. Sleep Medicine Rev. 2002;6(2):97-111.
27. Association of Sleep Disorders Centers. Diagnostic classification of sleep and arousal disorders, 1st edition. Prepared by the Sleep Disorders Classification Committee, H.P. Roffwarg, chairman. Sleep. 1979;2:1-137.
28. American Sleep Disorders Association. International classification of sleep disorders, revised: Diagnostic and coding manual. Rochester, MN: American Sleep Disorders Association; 1997.
29. Aikens J, Vanable P, Tadimeti L, Caruana-Montaldo B, Mendelson W. Differential rates of psychopathology symptoms in periodic limb movement disorders, obstructive sleep apnea, psychophysiological insomnia, and insomnia with psychiatric disorder. Sleep. 1999;22:775-80.
30. Akiskal H, Lemmi H, Dickson H, King D, Yerevanian B, Van Valkenburg C. Chronic depressions. Part 2. Sleep EEG differentiation of primary dysthymic disorders from anxious depressions. J Affect Disord. 1984;6:287-95.
31. Alling C, Balldin J, Bokstrom K, Gottfries C, Karlsson I, Langstrom G. Studies on duration of a late recovery period after chronic abuse of ethanol. A cross-sectional study of biochemical and psychiatric indicators. Acta Psychiatr Scan. 1982;66(5):384-97.
32. Almer G, Hainfellner J, Brucke T, et al. Fatal familial insomnia: a new Austrian family. Brain. 1999;122:5-16.
33. Apps M, Sheaff P, Ingram D, Kennard C, Empey D. Respiration and sleep in Parkinson's disease. J Neurol Neurosurg Psychiatry. 1985;48(12):1240-5.
34. Arriaga F, Paiva T. Clinical and EEG sleep changes in primary dysthymia and generalized anxiety: a comparison with normal controls. Neuropsychobiology. 1990-1991;24(3):109-14.
35. Arriaga F, Lara E, Matos-Pires A, Cavaglia F. Diagnostic relevance of sleep complaints in anxiety and mood disorders. European Psychiatry. 1995;10:386-90.
36. Arriaga F, Paiva T, Matos-Pires A, Cavaglia F, Lara E, L B. The sleep of non-depressed patients with panic disorder: a comparison with normal controls. Acta Psychiatr Scand. 1996;93(3):191-4.
37. Avoni P, Cortelli P, Montagna P, et al. Circadian hormonal rhythms in two new cases of fatal familial insomnia. Acta Neurol (Napoli). 1991;13(6):574-6.
38. Baekeland F, Hoy P. Reported vs recorded sleep characteristics. Arch Gen Psychiatry. 1971;24(6):548-51.
39. Becker PM, Jamieson AO, Brown WD. Dopaminergic agents in restless legs syndrome and periodic limb movements of sleep: response and complications of extended treatment in 49 cases. Sleep. 1993;16(8):713-6.
40. Birketvedt GS, Florholmen J, Sundsfjord J, et al. Behavioral and neuroendocrine characteristics of the night-eating syndrome.[see comment]. JAMA. 1999;282(7):657-63.
41. Bliwise D, Petta D, Seidel W, Dement W. Periodic leg movements during sleep in the elderly. Archives of Gerontology & Geriatrics. 1985;4(3):273-81.
42. Bliwise DL, Carskadon MA, Dement WC. Nightly variation of periodic leg movements in sleep in middle aged and elderly individuals. Archives of Gerontology & Geriatrics. 1988;7(4):273-9.
43. Bonnet MH, Arand DL. The consequences of a week of insomnia. Sleep. 1996;19(6):453-61.
44. Bonnet MH, Arand DL. Physiological activation in patients with Sleep State Misperception. Psychosomatic Medicine. 1997;59(5):533-40.
45. Bonnet MH, Arand DL. The consequences of a week of insomnia. II: Patients with insomnia. Sleep. 1998;21(4):359-68.
46. Bonnet MH, Arand DL. Activity, arousal, and the MSLT in patients with insomnia. Sleep. 2000;23(2):205-12.
47. Borkovec TD, Grayson JB, O'Brien GT, Weerts TC. Relaxation treatment of pseudoinsomnia and idiopathic insomnia: an electroencephalographic evaluation. Journal of Applied Behavior Analysis. 1979;12(1):37-54.
48. Chapman J, Cervenakova L, Petersen RB, et al. APOE in non-Alzheimer amyloidoses: transmissible spongiform encephalopathies. Neurology. 1998;51(2):548-53.
49. Chen SG, Teplow DB, Parchi P, Teller JK, Gambetti P, Autilio-Gambetti L. Truncated forms of the human prion protein in normal brain and in prion diseases. Journal of Biological Chemistry. 1995;270(32):19173-80.
50. Chen SG, Parchi P, Brown P, et al. Allelic origin of the abnormal prion protein isoform in familial prion diseases. Nature Medicine. 1997;3(9):1009-15.
51. Coates TJ, Killen JD, Silverman S, et al. Cognitive activity, sleep disturbance, and stage specific differences between recorded and reported sleep. Psychophysiology. 1983;20(3):243-50.
52. Coleman RM, Pollak CP, Weitzman ED. Periodic movements in sleep (nocturnal myoclonus): relation to sleep disorders. Annals of Neurology. 1980;8(4):416-21.
53. Cortelli P, Perani D, Parchi P, et al. Cerebral metabolism in fatal familial insomnia: relation to duration, neuropathology, and distribution of protease-resistant prion protein. Neurology. 1997;49(1):126-33.
54. Declerck AC, Verbeek I, Beecher L, Schuur J. Shifts and awakenings: valuable indicators of the quality of sleep: do these parameters react differently to hypnotics? Physiology & Behavior. 1993;54(4):815-7.
55. Dorandeu A, Wingertsmann L, Chretien F, et al. Neuronal apoptosis in fatal familial insomnia. Brain Pathology. 1998;8(3):531-7.
56. Dorsey CM, Bootzin RR. Subjective and psychophysiologic insomnia: an examination of sleep tendency and personality. Biological Psychiatry. 1997;41(2):209-16.
57. Downey R, 3rd, Bonnet MH. Training subjective insomniacs to accurately perceive sleep onset. Sleep. 1992;15(1):58-63.
58. Droste DW, Krauss JK, Hagedorn G, Kaps M. Periodic leg movements are part of the B-wave rhythm and the cyclic alternating pattern. Acta Neurologica Scandinavica. 1996;94(5):347-52.
59. Drummond SP, Gillin JC, Smith TL, DeModena A. The sleep of abstinent pure primary alcoholic patients: natural course and relationship to relapse. Alcoholism: Clinical & Experimental Research. 1998;22(8):1796-802.

60. Edinger JD, Hoelscher TJ, Webb MD, Marsh GR, Radtke RA, Erwin CW. Polysomnographic assessment of DIMS: empirical evaluation of its diagnostic value. Sleep. 1989;12(4):315-22.
61. Edinger JD, Fins AI. The distribution and clinical significance of sleep time misperceptions among insomniacs. Sleep. 1995;18(4):232-9.
62. Edinger JD, Fins AI, Goeke JM, et al. The empirical identification of insomnia subtypes: a cluster analytic approach. Sleep. 1996;19(5):398-411.
63. Ellis CM, Lemmens G, Parkes JD. Melatonin and insomnia. Journal of Sleep Research. 1996;5(1):61-5.
64. Factor SA, McAlarney T, Sanchez-Ramos JR, Weiner WJ. Sleep disorders and sleep effect in Parkinson's disease. Movement Disorders. 1990;5(4):280-5.
65. Ferini-Strambi L, Filippi M, Martinelli V, et al. Nocturnal sleep study in multiple sclerosis: correlations with clinical and brain magnetic resonance imaging findings. Journal of the Neurological Sciences. 1994;125(2):194-7.
66. Frankel BL, Patten BM, Gillin JC. Restless legs syndrome. Sleep-electroencephalographic and neurologic findings. JAMA. 1974;230(9):1302-3.
67. Frankel BL, Coursey RD, Buchbinder R, Snyder F. Recorded and reported sleep in chronic primary insomnia. Archives of General Psychiatry. 1976;33(5):615-23.
68. Gaillard JM. Chronic primary insomnia: possible physiopathological involvement of slow wave sleep deficiency. Sleep. 1978;1(2):133-47.
69. Gaillard J, et al. Temporal organization of paradoxical sleep in depressed patients with reference to brain catecholaminergic activity. Research Communications in Psychology, Psychiatry & Behavior. 1981;6(2):141-161.
70. Gaillard J-M, Iorio G, Campajola P, Kemali D. Temporal organization of sleep in schizophrenics and patients with obsessive-compulsive disorder. Advances in Biological Psychiatry Vol 15 1984, 76-83 S Karger AG, Switzerland. 1984.
71. Gallassi R, Morreale A, Montagna P, Gambetti P, Lugaresi E. "Fatal familial insomnia": neuropsychological study of a disease with thalamic degeneration. Cortex. 1992;28(2):175-87.
72. Gallassi R, Morreale A, Montagna P, et al. Fatal familial insomnia: behavioral and cognitive features. Neurology. 1996;46(4):935-9.
73. Grumet GW. Severe insomnia in psychiatric inpatients. Diseases of the Nervous System. 1968;29(4):256-8.
74. Guentchev M, Wanschitz J, Voigtlander T, Flicker H, Budka H. Selective neuronal vulnerability in human prion diseases. Fatal familial insomnia differs from other types of prion diseases. American Journal of Pathology. 1999;155(5):1453-7.
75. Guilleminault C, Eldridge F, Dement WC. Insomnia, narcolepsy, and sleep apneas. Bulletin de Physio-Pathologie Respiratoire. 1972;8(5):1127-38.
76. Guilleminault C, Eldridge FL, Dement WC. Insomnia with sleep apnea: a new syndrome. Science. 1973;181(102):856-8.
77. Guilleminault C, Eldridge FL, Phillips JR, Dement WC. Two occult causes of insomnia and their therapeutic problems. Archives of General Psychiatry. 1976;33(10):1241-5.
78. Hainfellner JA, Budka H. Disease associated prion protein may deposit in the peripheral nervous system in human transmissible spongiform encephalopathies. Acta Neuropathologica. 1999;98(5):458-60.
79. Hajak G, Rodenbeck A, Staedt J, Bandelow B, Huether G, Ruther E. Nocturnal plasma melatonin levels in patients suffering from chronic primary insomnia. Journal of Pineal Research. 1995;19(3):116-22.
80. Hajak G, Rodenbeck A, Ehrenthal HD, et al. No evidence for a physiological coupling between melatonin and glucocorticoids. Psychopharmacology. 1997;133(4):313-22.
81. Harder A, Jendroska K, Kreuz F, et al. Novel twelve-generation kindred of fatal familial insomnia from germany representing the entire spectrum of disease expression. American Journal of Medical Genetics. 1999;87(4):311-6.
82. Hauri P, Olmstead E. Childhood-onset insomnia. Sleep. 1980;3(1):59-65.
83. Hauri PJ. A cluster analysis of insomnia. Sleep. 1983;6(4):326-38.
84. Hauri P, Fisher J. Persistent psychophysiologic (learned) insomnia. Sleep. 1986;9(1):38-53.
85. Hauri PJ, Olmstead EM. Reverse first night effect in insomnia. Sleep. 1989;12(2):97-105.
86. Hauri PJ, Wisbey J. Wrist actigraphy in insomnia.[see comment]. Sleep. 1992;15(4):293-301.
87. Haynes SN, Adams AE, West S, Kamens L, Safranek R. The stimulus control paradigm in sleep-onset insomnia: a multimethod assessment. Journal of Psychosomatic Research. 1982;26(3):333-9.
88. Hertz G, Fast A, Feinsilver SH, Albertario CL, Schulman H, Fein AM. Sleep in normal late pregnancy. Sleep. 1992;15(3):246-51.
89. Hohagen F, Kappler C, Schramm E, Riemann D, Weyerer S, Berger M. Sleep onset insomnia, sleep maintaining insomnia and insomnia with early morning awakening—temporal stability of subtypes in a longitudinal study on general practice attenders. Sleep. 1994;17(6):551-4.
90. Hohagen F, Kappler C, Schramm E, et al. Prevalence of insomnia in elderly general practice attenders and the current treatment modalities. Acta Psychiatrica Scandinavica. 1994;90(2):102-8.
91. Holley JL, Nespor S, Rault R. Characterizing sleep disorders in chronic hemodialysis patients. ASAIO Transactions. 1991;37(3):M456-7.
92. Inman D, Silver S, Doghramji K. Sleep disturbances in post-traumatic stress disorders: A comparison with non-PTSD insomnia. Journal of Traumatic Stress. 1990;3:429-37.
93. Jennum P, Drewes AM, Andreasen A, Nielsen KD. Sleep and other symptoms in primary fibromyalgia and in healthy controls. Journal of Rheumatology. 1993;20(10):1756-9.
94. Johnson MD, Vnencak-Jones CL, McLean MJ. Fatal familial insomnia: clinical and pathologic heterogeneity in genetic half brothers. Neurology. 1998;51(6):1715-7.
95. Kales A, Bixler EO, Soldatos CR, Vela-Bueno A, Caldwell AB, Cadieux RJ. Biopsychobehavioral correlates of insomnia, part 1: Role of sleep apnea and nocturnal myoclonus. Psychosomatics. 1982;23(6):589-600.
96. Kalogjera-Sackellares D, Cartwright RD. Comparison of MMPI profiles in medically and psychologically based insomnias. Psychiatry Research. 1997;70(1):49-56.
97. Karlsen KH, Larsen JP, Tandberg E, Maeland JG. Influence of clinical and demographic variables on quality of life in patients with Parkinson's disease.[see comment]. Journal of Neurology, Neurosurgery & Psychiatry. 1999;66(4):431-5.
98. Kuisk LA, Bertelson AD, Walsh JK. Presleep cognitive hyperarousal and affect as factors in objective and subjective insomnia. Perceptual & Motor Skills. 1989;69(3 Pt 2):1219-25.
99. Lauerma H. Nocturnal wandering caused by restless legs and short-acting benzodiazepines. Acta Psychiatrica Scandinavica. 1991;83(6):492-3.
100. Lavie P, Hefez A, Halperin G, Enoch D. Long-term effects of traumatic war-related events on sleep. American Journal of Psychiatry. 1979;136(2):175-8.
101. Lavie P, Carmeli A, Mevorach L, Liberman N. Sleeping under the threat of the Scud: war-related environmental insomnia. Israel Journal of Medical Sciences. 1991;27(11-12):681-6.
102. Lavie P, Katz N, Pillar G, Zinger Y. Elevated awaking thresholds during sleep: characteristics of chronic war-related posttraumatic stress disorder patients. Biological Psychiatry. 1998;44(10):1060-5.
103. Lee JH, Bliwise DL, Lebret-Bories E, Guilleminault C, Dement WC. Dream-disturbed sleep in insomnia and narcolepsy. Journal of Nervous & Mental Disease. 1993;181(5):320-4.

104. Lichstein KL, Peterson BA, Riedel BW, Means MK, Epperson MT, Aguillard RN. Relaxation to assist sleep medication withdrawal. Behavior Modification. 1999;23(3):379-402.
105. Lichstein KL, Riedel BW, Lester KW, Aguillard RN. Occult sleep apnea in a recruited sample of older adults with insomnia. Journal of Consulting & Clinical Psychology. 1999;67(3):405-10.
106. Lugaresi E, Medori R, Montagna P, et al. Fatal familial insomnia and dysautonomia with selective degeneration of thalamic nuclei. New England Journal of Medicine. 1986;315(16):997-1003.
107. Lundh L-G, Lundqvist K, Broman J-E, Hetta J. Vicious cycles of sleeplessness, sleep phobia, and sleep-incompatible behaviours in patients with persistent insomnia. Scandinavian Journal of Behaviour Therapy. 1991;20(3-4):101-114.
108. Lundh L-G, Froeding A, Gyllenhammar L, Broman J-E, et al. Cognitive bias and memory performance in patients with persistent insomnia. Scandinavian Journal of Behaviour Therapy. 1997;26(1):27-35.
109. Macchi G, Rossi G, Abbamondi AL, et al. Diffuse thalamic degeneration in fatal familial insomnia. A morphometric study. Brain Research. 1997;771(1):154-8.
110. Manetto V, Medori R, Cortelli P, et al. Fatal familial insomnia: clinical and pathologic study of five new cases. Neurology. 1992;42(2):312-9.
111. Manni R, Ratti MT, Tartara A. Nocturnal eating: prevalence and features in 120 insomniac referrals. Sleep. 1997;20(9):734-8.
112. McCall WV, Edinger JD, Lininger A. Neurological findings in insomniacs with periodic limb movements.[see comment]. Journal of Neuropsychiatry & Clinical Neurosciences. 1991;3(4):429-30.
113. McCall WV, Edinger JD. Subjective total insomnia: an example of sleep state misperception. Sleep. 1992;15(1):71-3.
114. Medori R, Montagna P, Tritschler HJ, et al. Fatal familial insomnia: a second kindred with mutation of prion protein gene at codon 178. Neurology. 1992;42(3 Pt 1):669-70.
115. Medori R, Tritschler HJ, LeBlanc A, et al. Fatal familial insomnia, a prion disease with a mutation at codon 178 of the prion protein gene.[see comment]. New England Journal of Medicine. 1992;326(7):444-9.
116. Medori R, Tritschler HJ. Prion protein gene analysis in three kindreds with fatal familial insomnia (FFI): codon 178 mutation and codon 129 polymorphism. American Journal of Human Genetics. 1993;53(4):822-7.
117. Mellman TA, Uhde TW. Sleep panic attacks: new clinical findings and theoretical implications.[see comment]. American Journal of Psychiatry. 1989;146(9):1204-7.
118. Mendelson WB, Garnett D, Gillin JC, Weingartner H. The experience of insomnia and daytime and nighttime functioning. Psychiatry Research. 1984;12(3):235-50.
119. Mendelson WB. Long-term follow-up of chronic insomnia. Sleep. 1995;18(8):698-701.
120. Mendelson WB. Are periodic leg movements associated with clinical sleep disturbance?[see comment]. Sleep. 1996;19(3):219-23.
121. Mendelson WB. Effects of time of night and sleep stage on perception of sleep in subjects with sleep state misperception. Psychobiology. 1998;26(1):73-78.
122. Merica H, Gaillard JM. The EEG of the sleep onset period in insomnia: a discriminant analysis. Physiology & Behavior. 1992;52(2):199-204.
123. Mishima K, Okawa M, Hishikawa Y, Hozumi S, Hori H, Takahashi K. Morning bright light therapy for sleep and behavior disorders in elderly patients with dementia. Acta Psychiatrica Scandinavica. 1994;89(1):1-7.
124. Montagna P, Cortelli P, Avoni P, et al. Clinical features of fatal familial insomnia: phenotypic variability in relation to a polymorphism at codon 129 of the prion protein gene.[see comment]. Brain Pathology. 1998;8(3):515-20.
125. Montgomery AB, Mills J, Luce JM. Incidence of acute mountain sickness at intermediate altitude. JAMA. 1989;261(5):732-4.
126. Montplaisir J, Boucher S, Poirier G, Lavigne G, Lapierre O, Lesperance P. Clinical, polysomnographic, and genetic characteristics of restless legs syndrome: a study of 133 patients diagnosed with new standard criteria. Movement Disorders. 1997;12(1):61-5.
127. Morgan K, Lilley JM. Risk factors among incident cases of dementia in a representative British sample. International Journal of Geriatric Psychiatry. 1994;9(1):11-15.
128. Morin CM, Stone J, McDonald K. Psychological management of insomnia: a clinical replication series with 100 patients. Behavior Therapy, 1994;25:291-309.
129. Morriss RK, Wearden AJ, Battersby L. The relation of sleep difficulties to fatigue, mood and disability in chronic fatigue syndrome. Journal of Psychosomatic Research. 1997;42(6):597-605.
130. Nagayama M, Shinohara Y, Furukawa H, Kitamoto T. Fatal familial insomnia with a mutation at codon 178 of the prion protein gene: first report from Japan. Neurology. 1996;47(5):1313-6.
131. Newton JR, Shapiro C, Stewart A. Thirty years' war: a battle with insomnia. British Journal of Psychiatry. 1989;154:691-6.
132. Neylan TC, Marmar CR, Metzler TJ, et al. Sleep disturbances in the Vietnam generation: findings from a nationally representative sample of male Vietnam veterans. American Journal of Psychiatry. 1998;155(7):929-33.
133. Nozawa T, Ichikawa H, Takeuchi T. Periodic limb movements and sleep-wake disorder. Psychiatry & Clinical Neurosciences. 1998;52(2):192-4.
134. O'Keeffe ST, Noel J, Lavan JN. Restless legs syndrome in the elderly. Postgraduate Medical Journal. 1993;69(815):701-3.
135. Ohanna N, Peled R, Rubin AH, Zomer J, Lavie P. Periodic leg movements in sleep: effect of clonazepam treatment. Neurology. 1985;35(3):408-11.
136. Ohayon M. Epidemiological study on insomnia in the general population. Sleep. 1996;19(3 Suppl):S7-15.
137. Ohayon MM, Caulet M. Insomnia and psychotropic drug consumption. Progress in Neuro-Psychopharmacology & Biological Psychiatry. 1995;19(3):421-31.
138. Ohayon MM, Guilleminault C, Zulley J, Palombini L, Raab H. Validation of the sleep-EVAL system against clinical assessments of sleep disorders and polysomnographic data. Sleep. 1999;22(7):925-30.
139. Ohayon MM, Morselli PL, Guilleminault C. Prevalence of nightmares and their relationship to psychopathology and daytime functioning in insomnia subjects. Sleep. 1997;20(5):340-8.
140. Padovani A, D'Alessandro M, Parchi P, et al. Fatal familial insomnia in a new Italian kindred. Neurology. 1998;51(5):1491-4.
141. Parchi P, Castellani R, Cortelli P, et al. Regional distribution of protease-resistant prion protein in fatal familial insomnia. Annals of Neurology. 1995;38(1):21-9.
142. Parchi P, Capellari S, Chin S, et al. A subtype of sporadic prion disease mimicking fatal familial insomnia. Neurology. 1999;52(9):1757-63.
143. Parrino L, Boselli M, Buccino GP, Spaggiari MC, Di Giovanni G, Terzano MG. The cyclic alternating pattern plays a gate-control on periodic limb movements during non-rapid eye movement sleep. Journal of Clinical Neurophysiology. 1996;13(4):314-23.
144. Perani D, Cortelli P, Lucignani G, et al. [18F]FDG PET in fatal familial insomnia: the functional effects of thalamic lesions. Neurology. 1993;43(12):2565-9.
145. Perlstrom JR, Wickramasekera I. Insomnia, hypnotic ability, negative affectivity, and the high risk model of threat perception. Journal of Nervous & Mental Disease. 1998;186(7):437-40.
146. Philip P, Guilleminault C. Adult psychophysiologic insomnia and positive history of childhood insomnia. Sleep. 1996;19(3 Suppl):S16-22.
147. Plazzi G, Schutz Y, Cortelli P, et al. Motor overactivity and loss of motor circadian rhythm in fatal familial insomnia: an actigraphic

study. Sleep. 1997;20(9):739-42.
148. Poceta JS, Dalessio DJ. Identification and treatment of sleep apnea in patients with chronic headache. Headache. 1995;35(10):586-9.
149. Portaluppi F, Cortelli P, Avoni P, et al. Progressive disruption of the circadian rhythm of melatonin in fatal familial insomnia. Journal of Clinical Endocrinology & Metabolism. 1994;78(5):1075-8.
150. Reder AT, Mednick AS, Brown P, et al. Clinical and genetic studies of fatal familial insomnia. Neurology. 1995;45(6):1068-75.
151. Regestein QR, Reich P. Incapacitating childhood-onset insomnia. Comprehensive Psychiatry. 1983;24(3):244-8.
152. Reynolds CF, 3rd, Taska LS, Sewitch DE, Restifo K, Coble PA, Kupfer DJ. Persistent psychophysiologic insomnia: preliminary Research Diagnostic Criteria and EEG sleep data. American Journal of Psychiatry. 1984;141(6):804-5.
153. Riedel BW, Lichstein KL. Objective sleep measures and subjective sleep satisfaction: how do older adults with insomnia define a good night's sleep? Psychology & Aging. 1998;13(1):159-63.
154. Roehrs T, Conway W, Wittig R, Zorick F, Sicklesteel J, Roth T. Sleep-wake complaints in patients with sleep-related respiratory disturbances. American Review of Respiratory Disease. 1985;132(3):520-3.
155. Rosa RR, Bonnet MH. Reported chronic insomnia is independent of poor sleep as measured by electroencephalography.[see comment]. Psychosomatic Medicine. 2000;62(4):474-82.
156. Rosenthal L, Roehrs T, Sicklesteel J, Zorick F, Wittig R, Roth T. Periodic movements during sleep, sleep fragmentation, and sleep-wake complaints. Sleep. 1984;7(4):326-30.
157. Rossi G, Macchi G, Porro M, et al. Fatal familial insomnia: genetic, neuropathologic, and biochemical study of a patient from a new Italian kindred. Neurology. 1998;50(3):688-92.
158. Saletu B, Klosch G, Gruber G, Anderer P, Udomratn P, Frey R. First-night-effects on generalized anxiety disorder (GAD)-based insomnia: laboratory versus home sleep recordings. Sleep. 1996;19(9):691-7.
159. Saletu B, Saletu-Zyhlarz G, Anderer P, et al. Nonorganic insomnia in generalized anxiety disorder. 2. Comparative studies on sleep, awakening, daytime vigilance and anxiety under lorazepam plus diphenhydramine (Somnium) versus lorazepam alone, utilizing clinical, polysomnographic and EEG mapping methods. Neuropsychobiology. 1997;36(3):130-52.
160. Salin-Pascual RJ, Roehrs TA, Merlotti LA, Zorick F, Roth T. Long-term study of the sleep of insomnia patients with sleep state misperception and other insomnia patients. American Journal of Psychiatry. 1992;149(7):904-8.
161. Saskin P, Moldofsky H, Lue FA. Periodic movements in sleep and sleep-wake complaint. Sleep. 1985;8(4):319-24.
162. Schneider-Helmert D, Kumar A. Sleep, its subjective perception, and daytime performance in insomniacs with a pattern of alpha sleep. Biological Psychiatry. 1995;37(2):99-105.
163. Schramm E, Hohagen F, Grasshoff U, et al. Test-retest reliability and validity of the Structured Interview for Sleep Disorders According to DSM-III-R. American Journal of Psychiatry. 1993;150(6):867-72.
164. Schramm E, Hohagen F, Kappler C, Grasshoff U, Berger M. Mental comorbidity of chronic insomnia in general practice attenders using DSM-III-R. Acta Psychiatrica Scandinavica. 1995;91(1):10-7.
165. Sforza E, Montagna P, Tinuper P, et al. Sleep-wake cycle abnormalities in fatal familial insomnia. Evidence of the role of the thalamus in sleep regulation. Electroencephalography & Clinical Neurophysiology. 1995;94(6):398-405.
166. Sharpley AL, Attenburrow ME, Cowen PJ. Assessment and treatment of insomnia (including a case control study of patients with Primary Insomnia). International Journal of Psychiatry in Clinical Practice. 1997;1(2):107-117.
167. Shinba T, Murashima YL, Yamamoto K. Alcohol consumption and insomnia in a sample of Japanese alcoholics. Addiction. 1994;89(5):587-91.
168. Staedt J, Stoppe G, Kogler A, et al. Single photon emission tomography (SPET) imaging of dopamine D2 receptors in the course of dopamine replacement therapy in patients with nocturnal myoclonus syndrome (NMS). Journal of Neural Transmission - General Section. 1995;99(1-3):187-93.
169. Stein MB, Kroft CD, Walker JR. Sleep impairment in patients with social phobia. Psychiatry Research. 1993;49(3):251-6.
170. Stepanski E, Lamphere J, Badia P, Zorick F, Roth T. Sleep fragmentation and daytime sleepiness. Sleep. 1984;7(1):18-26.
171. Stepanski E, Zorick F, Roehrs T, Young D, Roth T. Daytime alertness in patients with chronic insomnia compared with asymptomatic control subjects. Sleep. 1988;11(1):54-60.
172. Stepanski E, Zorick F, Roehrs T, Roth T. Effects of sleep deprivation on daytime sleepiness in primary insomnia. Sleep. 2000;23(2):215-9.
173. Stone J, Morin CM, Hart RP, Remsberg S, Mercer J. Neuropsychological functioning in older insomniacs with or without obstructive sleep apnea. Psychology & Aging. 1994;9(2):231-6.
174. Sugerman JL, Stern JA, Walsh JK. Daytime alertness in subjective and objective insomnia: some preliminary findings. Biological Psychiatry. 1985;20(7):741-50.
175. Terzano MG, Parrino L. Evaluation of EEG cyclic alternating pattern during sleep in insomniacs and controls under placebo and acute treatment with zolpidem. Sleep. 1992;15(1):64-70.
176. Vanable PA, Aikens JE, Tadimeti L, Caruana-Montaldo B, Mendelson WB. Sleep latency and duration estimates among sleep disorder patients: variability as a function of sleep disorder diagnosis, sleep history, and psychological characteristics. Sleep. 2000;23(1):71-9.
177. Velden M, Wolk C. Baroreceptor activity and the induction of sleep. Perceptual & Motor Skills. 1996;82(1):178.
178. Vertraeten E, Cluydts R, Verbraecken J, De Roeck J. Neuropsychological functioning and determinants of morning alertness in patients with obstructive sleep apnea syndrome. J Intnernational Neuropsychology Society. 1996;2:306-14.
179. Vgontzas AN, Kales A, Bixler EO, Manfredi RL, Vela-Bueno A. Usefulness of polysomnographic studies in the differential diagnosis of insomnia. International Journal of Neuroscience. 1995;82(1-2):47-60.
180. Weissman MM, Greenwald S, Nino-Murcia G, Dement WC. The morbidity of insomnia uncomplicated by psychiatric disorders. General Hospital Psychiatry. 1997;19(4):245-50.
181. White KP, Speechley M, Harth M, Ostbye T. The London Fibromyalgia Epidemiology Study: comparing the demographic and clinical characteristics in 100 random community cases of fibromyalgia versus controls. Journal of Rheumatology. 1999;26(7):1577-85.
182. Wiegand M, Moller AA, Schreiber W, Krieg JC, Holsboer F. Alterations of nocturnal sleep in patients with HIV infection. Acta Neurologica Scandinavica. 1991;83(2):141-2.
183. Wittig RM, Zorick FJ, Blumer D, Heilbronn M, Roth T. Disturbed sleep in patients complaining of chronic pain. Journal of Nervous & Mental Disease. 1982;170(7):429-31.
184. Youngstedt SD, Kripke DF, Klauber MR, Sepulveda RS, Mason WJ. Periodic leg movements during sleep and sleep disturbances in elders. Journals of Gerontology Series A-Biological Sciences & Medical Sciences. 1998;53(5):M391-4.
185. Zerr I, Giese A, Windl O, et al. Phenotypic variability in fatal familial insomnia (D178N-129M) genotype. Neurology. 1998;51(5):1398-405.
186. Zorick FJ, Roth T, Hartse KM, Piccione PM, Stepanski EJ. Evaluation and diagnosis of persistent insomnia. American Journal of Psychiatry. 1981;138(6):769-73.
187. Zorick FJ, Kribbs N, Roehrs T, Roth T. Polysomnographic and

MMPI characteristics of patients with insomnia. Psychopharmacology. 1984;Suppl 1:2-10.
188. Zucconi M, Ferini-Strambi L, Gambini O, et al. Structured psychiatric interview and ambulatory sleep monitoring in young psychophysiological insomniacs. Journal of Clinical Psychiatry. 1996;57(8):364-70.
189. American Academy of Sleep Medicine. International Classification of Sleep Disorders: Diagnostic and Coding Manual. 2 ed Westchester, IL: American Academy of Sleep Medicine; in press.
190. Reynolds CF, 3rd, Kupfer DJ, Buysse DJ, Coble PA, Yeager A. Subtyping DSM-III-R primary insomnia: a literature review by the DSM-IV Work Group on Sleep Disorders. American Journal of Psychiatry. 1991;148(4):432-8.
191. Edinger JD, Krystal AD. Subtyping primary insomnia: is sleep state misperception a distinct clinical entity?[see comment]. Sleep Medicine Reviews. 2003;7(3):203-14.
192. Perlis ML, Smith MT, Andrews PJ, Orff H, Giles DE. Beta/Gamma EEG activity in patients with primary and secondary insomnia and good sleeper controls. Sleep. 2001;24(1):110-7.
193. Krystal AD, Edinger JD, Wohlgemuth WK, Marsh GR. NREM sleep EEG frequency spectral correlates of sleep complaints in primary insomnia subtypes. Sleep. 2002;25(6):630-40.
194. Morin CM, Edinger JD. Sleep disorders: evaluation and diagnosis. In: Turner S, M H, eds. Adult psychopathology and diagnosis. 3 ed. New York: John Wiley & Sons; 1997.
195. Bixler EO, Vgontzas AN, Lin HM, Vela-Bueno A, Kales A. Insomnia in central Pennsylvania. Journal of Psychosomatic Research. 2002;53(1):589-92.
196. Chesson A, Jr., Hartse K, Anderson WM, et al. Practice parameters for the evaluation of chronic insomnia. An American Academy of Sleep Medicine report. Standards of Practice Committee of the American Academy of Sleep Medicine. Sleep. 2000;23(2):237-41.
197. Rechtschaffen A, Kales A. A manual of standardized terminology, techniques and scoring system of sleep stages of human subjects. Los Angeles: UCLA Brain Information Service/Brain Research Institute; 1968.
198. Phillipson E, Remmers J. Indications and standards for cardiopulmonary sleep studies. American Thoracic Society. Medical Section of the American Lung Association.[see comment]. American Review of Respiratory Disease. 1989;139(2):559-68.
199. Coleman R. Periodic limb movements in sleep (nocturnal myoclonus) and restless legs syndrome. In: Guilleminault C, ed. Sleeping and waking disorders: Indications and techniques. Menlo Park, CA: Addison-Wesley; 1982:265-95.
200. American Sleep Disorders Association Task Force, (Chair) Guilleminault C. EEG arousals: scoring rules and examples. Sleep. 1992;15:173-84.
201. Sateia MJ, Doghramji K, Hauri PJ, Morin CM. Evaluation of chronic insomnia. An American Academy of Sleep Medicine review. Sleep. 2000;23(2):243-308.

APPENDIX A: RDC WORKGROUP DATA EXTRACTION SHEET

RDC WORKGROUP DATA EXTRACTION SHEET: DIAGNOSTIC RELIABILITY

Citation	**Authors:**			
	Article Title:			
	Journal:			
	Year/Volume/Page #s			
Sample Selection	**Check the method(s) used in the column to the right**			
	Randomly selected community sample (e.g., random digit dialing method)			
	Series of consecutive clinic patients			
	Prospectively selected clinic patients (not necessarily all consecutive)			
	Physician (or other health provider) Referrals			
	Archival data such as a clinic data base			
	Solicited research volunteers			
	Other/specify:			
Inclusion Criteria Used in Addition to Specified Diagnostic Criteria	**List additional criteria here:**			
Study Setting (Circle)	Sleep Disorders Center/Clinic	Mental Health Center	Other/Specify:	
	Medical Center	Private Medical Clinic		
	Nursing Home	Univ. Psychology Clinic		
Types of Subjects (Circle)	Inpatients	Long-term Care	Research Volunteers	
	Outpatients	Other/specify		
Method of Reliability Assessment	**Check the method(s) used in the column to the right**			
	Independent blinded interviewers using structured interview			
	Independent blinded interviewers without structured interview			
	Independent judges viewing video interviews of patients			
	Independent judges reviewing audio recordings of patient interviews			
	Independent judges reviewing archival data (e.g. chart reviews)			
	Other/specify: None - Reliability was not assessed in this article			
Reliability Index Used	**Check those that apply** ___% agreement between/among raters ___Reliability Coefficient (correlation between/among raters) ___Kappa values ___Other/specify: None-Reliability not assessed			

Sample Characteristics: numbers and types of diagnoses / age, gender information	Number of subjects			Age	
Diagnostic Groups: List each separately	**Women**	**Men**	**Total**	**Mean**	**S.D.**
Totals:					

Study Results: Reliability Indices	**Insomnia Diagnostic Subtype**	**Reliability Index***

* Note: It is assumed that the reliability index value shown is this column is of the type indicated on the previous page. If not, please explain here.

RDC WORKGROUP DATA EXTRACTION SHEET: DIAGNOSTIC VALIDITY

Citation	**Authors:**	
	Article Title:	
	Journal:	
	Year/Volume/Page #s	
Sample Selection	**Check the method(s) used in the column to the right**	
	Randomly selected community sample (e.g., random digit dialing method)	
	Series of consecutive clinic patients	
	Prospectively selected clinic patients (not necessarily all consecutive)	
	Physician (or other health provider) Referrals	
	Archival data such as a clinic data base	
	Solicited research volunteers	
	Other/specify:	
Inclusion Criteria Used in Addition to Specified Diagnostic Criteria	**List additional criteria here:**	
Study Setting (Circle)	Sleep Disorders Center/Clinic; Mental Health Center; Other/Specify: Medical Center; Private Medical Clinic Nursing Home; Univ. Psychology Clinic	
Types of Subjects (Circle)	Inpatients; Long-term Care; Research Volunteers Outpatients; Other/specify	
Types of Comparisons Conducted	**Check the types of comparisons in the column to the right**	
	Single Diagnostic Subtype with Normal Controls	
	Multiple Diagnostic Subtype with Normal Controls	
	Multiple Diagnostic Subtype with each other	
	Multiple Diagnostic Subtype with each other & with Normal Controls	
	Comparison of one or more subgroup with historic normative data	
	Other/specify:	
	None Conducted	
Measures Used in Making Comparisons	**Check those that apply**	
	Polysomnography R & K Measures	
	Polysomnography Spectral Measures	
	Actigraphy	
	Sleep Logs	
	Heart Rate Variability	
	MSLT Latencies	
	MMPI	
	Beck Depression Inventory	
	State Trait Anxiety Inventory	
	Gobal or Retrospective Questionaire: Specify	
	Other Psychometircs (specify):	
	Other – Specify	

Sample Characteristics: numbers and types of diagnoses / age, gender information		Number of subjects			**Age**	
	Diagnostic Groups: List each separately	**Women**	**Men**	**Total**	**Mean**	**S.D.**
	Totals:					

Results of Comparisons: Group Means, SD's and statistical differences

RDC WORKGROUP DATA EXTRACTION SHEET: DIAGNOSTIC VALIDITY *continued on next page.*

Comparative Measure	Diagnostic Groups and DSM-IV/ICSD Code #'s							
	(1)		(2)		(3)		(4)	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD
PSG/R&K								
SOL								
WASO								
TWT								
SE%								
TST								
TIB								
other								
PSG Spectral								
Actigraphy								
SOL								
WASO								
TWT								
SE%								
TST								
TIB								
other								
	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Sleep Logs								
SOL								
WASO								
TWT								
SE%								
TST								
TIB								
quality								
Heart Rate Variability								
MSLT								
MMPI								
L								
F								
K								
1								
2								
3								
4								
5								
6								
7								
8								
9								
0								
Beck Depression COG/AFF Somatic Total								
State-Trait								
Anxiety								
State								
Trait								
Other Measures								

(List)

In a few sentences, please provide a brief narrative summary of the study findings here.		
Bias to Internal Validity	**Check all that apply**	
	None - there are no biases	
	Patient/subject selection	
	Confounding factors	
	Measurement errors	
	Drop-outs	
	Non-Standardized Conditions of Measurement	
	Improper statistics	
	Insufficient power - n too small	
	Other - specify	
Bias to External Validity	Population and other issues - specify	
Should this study be included in our final diagnostic validity evidence table? Give reasons.		

Sensitivity and Specificity of Measures of the Insomnia Experience: a Comparative Study of Psychophysiologic Insomnia, Insomnia Associated with Mental Disorder and Good Sleepers

Linda Kohn, DclinPsy; Colin A. Espie, FBPsS, PhD

University of Glasgow, Scotland, UK

Study objectives: To explore proposed explanatory mechanisms in psychophysiologic insomnia by investigating the sensitivity and specificity of commonly used insomnia research tools in discriminating psychophysiologic insomnia, insomnia associated with mental disorder, and good sleepers.

Design: Cross-sectional, between-group comparison of responses from subjects with psychophysiologic insomnia, those with insomnia associated with mental disorder, and good sleepers to psychometrically robust self-report instruments.

Setting: Attendees at adult community outpatient clinics.

Participants: Fifty-four adults (36 women, 18 men; average age 40 years) across 3 groups (n = 18 per group). Participants with psychophysiologic insomnia met combined International Classification of Sleep Disorders, Revised and Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition criteria and had no history of mental disorder. Participants with insomnia associated with mental disorder satisfied the same criteria for sleep disturbance and met Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition (Structured Clinical Interview for DSM-IV axis-I Disorders) criteria for depressive disorder. The majority had comorbid anxiety disorder. Insomnia duration in the groups with psychophysiologic insomnia and insomnia associated with mental disorder was around 10 years. Good sleepers served as a control group and included self-reported good sleepers with no history of sleep problems or psychiatric disorder.

Intervention: N/A.

Measurements and Results: Analyses of variance, adjusted for multiple comparisons, indicated no between-group differences on a measure of sleep-related stimulus control, and self-reported somatic arousal was higher in subjects with insomnia associated with mental disorder than in good sleepers or those with psychophysiologic insomnia. Subjects with insomnia associated with mental disorder and psychophysiologic insomnia had poorer sleep hygiene and were characterized by heightened mental arousal. Logistic regression indicated that "effortful preoccupation with sleep" discriminated subjects with both psychophysiologic insomnia (100% sensitivity, 94% specificity) and insomnia associated with mental disorder (100%, 100%) from good sleepers and that only depressive symptomatology discriminated insomnia associated with mental disorder from psychophysiologic insomnia.

Conclusion: Psychophysiologic insomnia and insomnia associated with mental disorder may be on a continuum of insomnia severity, rather than categorically distinct. Insomnia associated with mental disorder may respond to psychological intervention. Factors specifically discriminating insomniacs from good sleepers require further investigation.

Abbreviations: PI, Psychophysiologic Insomnia; I-MD, Insomnia associated with Mental Disorder; GS, Good Sleepers; ICSD-R, International Classification of Sleep Disorders (revised); DSM-IV, Diagnostic and Statistical Manual of the Mental Disorders (4th edition); SOL, Sleep-Onset Latency; SE, Sleep Efficiency; WASO, Wake time After Sleep Onset; TST, Total Sleep Time; BDI, Beck Depression Inventory; BAI, Beck Anxiety Inventory; SCID-I, Structured Clinical Interview for DSM-IV (axis-I disorders); SBSRS-R, Sleep Behavior Self-Rating Scale (revised); PSAS, Pre-Sleep Arousal Scale; SDQ, Sleep Disturbance Questionnaire; GSES, Glasgow Sleep Effort Scale; DBAS-10, Dysfunctional Beliefs and Attitudes about Sleep scale (10 item version); SHAPS, Sleep Hygiene Awareness and Practice Scale.

Key Words: Psychophysiologic insomnia; measurement; Psychological models

Citation: Kohn L; Espie CA. Sensitivity and Specificity of measures of the insomnia experience: A comparative study of psychophysiologic insomnia, insomnia associated with mental disorder and good sleepers. *SLEEP* 2005;28(1):104-112.

INTRODUCTION

THE *DIAGNOSTIC AND STATISTICAL MANUAL OF MENTAL DISORDERS, FOURTH EDITION* (DSM-IV) DEFINES *PRIMARY INSOMNIA* AS A COMPLAINT OF DIFFICULTY initiating and/or maintaining sleep or of nonrestorative sleep lasting for at least 1 month.[1] The revised version of the *International Classification of Sleep Disorders, Revised* (ICSD-R) uses the term *psychophysiologic insomnia* and regards insomnia of 6 months duration as *chronic*.[2] Contemporary understanding of psychophysiologic insomnia refers to underlying conditioned physiologic arousal[3-5] and learned sleep-preventing associations,[6,7] with these components being regarded as mutually reinforcing (ICSD-R). However, it is also recognised that the presenting complaint usually reflects mental more than somatic preoccupation,[8-10] and so an interaction between behavioral, physiologic, and cognitive factors in the development and persistence of psychophysiologic insomnia has been proposed.[11-15]

Sleep disturbance presenting in a psychiatric condition has been regarded as "secondary" to (DSM-IV) or, more descriptively, as "associated with" mental disorder (ICSD-R). The latter can be readily observed, whereas causality would need to be carefully evidenced. To take the example of depression, there is in fact strong evidence that preexisting insomnia is a risk factor for first incidence and recurrence of a depressive episode.[16-20] These findings are rein-

Disclosure Statement
This is not an industry supported study. Drs. Espie and Kohn have indicated no financial conflicts of interest.

Submitted for publication December 2003
Accepted for publication August 2004
Address correspondence to: Professor Colin A. Espie, Section of Psychological Medicine University of Glasgow, Academic Centre, Gartnavel Royal Hospital, 1055 Great Western Road, Glasgow, G12 0XH, Scotland, UK; Tel: 44 0141 211 3903; Fax: 44 0141 357 4899; E-mail: c.espie@clinmed.gla.ac.uk

forced by recent a meta-analysis,[21] and it has been suggested that insomnia is a prodromal state of depression.[20] Thus, it appears more circumspect to refer simply to the temporal association between insomnia and mental disorder rather than to imply causality in either direction. Interestingly, however, diagnostic schedules for insomnia associated with mental disorder do not refer to any of the putative mechanisms for psychophysiologic insomnia.

There is a sizeable literature comparing people with insomnia and good sleepers across a range of cognitive, affective, and physiologic measures.[14] Surprisingly, however, there is little direct evidence of inadequate stimulus control or of conditioned arousal in bed as specific features of psychophysiologic insomnia. Furthermore, although there is some recent work of good quality on secondary insomnia,[22] to our knowledge, no study has compared insomnia associated with mental disorder with either psychophysiologic insomnia or good sleeper controls, using the descriptive self-report measures that are commonly used in insomnia research and practice. Thus, we do not have data on the sensitivity and specificity of this type of measurement. Research of this kind is important because we cannot assume discontinuity between the insomnias. Diagnostic systems, being categorical, tend to infer different underlying mechanisms and treatment responses, but evidence of such differences is unavailable.

Our aim was to investigate whether models that are applicable to psychophysiologic insomnia might also apply to sleep disturbance associated with mental disorder. More specifically, we wanted to explore self-reported behavioral, cognitive, and physiologic features to see if they evidenced continuity or discontinuity. This is a preliminary study of association rather than cause and effect. Nevertheless, because there are efficacious cognitive-behavioral treatments for persistent insomnia and effective pharmacologic treatments for short-term use,[23-26] it is possible that insomnia therapies might be better targeted if distinctive features of insomnia could be established. Similarly, cognitive-behavioral treatments might be applied to sleep disturbance associated with mental disorder if we better understood the differential diagnosis from a conceptual perspective.

METHOD

Design

The study comprised a between-subjects design with 3 independent groups: psychophysiologic insomnia (PI; n = 18), insomnia associated with mental disorder (I-MD; n = 18), and good sleepers (GS; n = 18). Between-group comparisons were made on measures of self-reported (subjective) and actigraphically estimated (objective) sleep and on self-report measures of sleep-related behavior, thinking, and arousal.

Participants

The PI group had to meet combined DSM-IV/ICSD-R criteria, with the sleep pattern being assessed by sleep history and a self-report sleep diary over 7 nights,[13] and (a) a sleep-onset latency (SOL) of more than 30 minutes, (b) a sleep efficiency (SE) of less than 85%, (c) frequent or extended nocturnal awakenings totaling more than 30 minutes of wakefulness after sleep onset (WASO), or (d) any combination of a, b and c, in each case occurring on at least 3 out of 7 nights per week for at least 1 month. Subjects were excluded from the PI group if they met criteria for any axis-I disorder or past major depressive episode (assessed by Structured Clinical Interview for DSM-IV axis-I Disorders (SCID-I)[27] or had any evidence on our locally derived screening measures of sleep-related breathing disorder or periodic limb movements in sleep. Additionally, they were excluded if they had a Beck Depression Inventory (BDI)[28] score of 20 or more,[29] or had any physical or medical problems causing insomnia (as defined in ICSD-R). Thirty consecutive potential participants, complaining of significant and persistent sleep disturbance, were identified through attendance at clinical psychology outpatient services. Of these, 7 failed to complete screening, 3 failed to attend, and 2 were excluded because they did not meet insomnia criteria (final n =18).

Participants with I-MD had to satisfy the same sleep disturbance criteria, meet DSM-IV criteria (SCID-I) for major depressive disorder, and obtain a score of ³ 20 on the BDI. These participants were initially intended to form an "insomnia associated with depression" group, but, because of the high comorbidity of depressive and anxiety disorders, a more general title has been applied. Operationally, I-MD should be taken to mean "insomnia associated with mental disorder (depressed/anxious)." We excluded other individuals who met criteria for other axis-I disorders if they had medical problems causing sleep disturbance, any clinical evidence of sleep-related breathing disorder or periodic limb movements of sleep, or if their only sleep problem was early morning wakening. This latter criterion was to increase comparability of sleep-symptom complaint between the PI and I-MD groups. The Beck Anxiety Inventory (BAI)[30] was also applied descriptively to assess severity of anxiety symptoms (all groups). Thirty-two individuals were identified through mental health services or general practice surgeries, of whom 2 failed to complete screening, 2 did not attend, 2 failed to return measures, and 8 were excluded (1 mild mental retardation, 1 medical complaint causing insomnia, 4 did not meet criteria for depressive disorder, and 2 had additional axis-I diagnoses), leaving 18 in the I-MD group.

The GS group was recruited as an opportunity sample. Eighteen out of 20 nonclinical volunteers participated (2 excluded due to temporary illness causing sleep disruption). All had (a) SOL $\leq$ 30 minutes; (b) SE > 85%; (c) WASO totaling $\leq$ 30 minutes, in each case occurring on at least 5 out of 7 nights per week as measured by the sleep diary; and (d) regarded themselves as a good sleeper.

Measures and Procedure

Following screening, participants completed a 7-night sleep diary[13] while concurrently wearing an actigraph on the nondominant wrist to provide an objective estimate of sleep. Actiwatch–Râ Model AW4 (Cambridge Neurotechnology Ltd., Cambridge, UK) devices were used with epochs of 1 minute selected for accurate sleep analysis.[31] Actigraphs had event markers, which participants pressed when they went to bed and when they got up. Objective estimates of SOL, WASO, total sleep time (TST), and SE were recovered using Sleepwatchâ software (Cambridge Neurotechnology, Ltd.), and parallel data were derived from the sleep diaries.

A set of self-report measures was selected to best reflect presumed underlying mechanisms in insomnia. Participants completed these measures at home and returned them with their sleep diaries and actigraph. The Sleep Behavior Self-Rating Scale (SBSRS)[32] assesses sleep-incompatible behaviors associated with a person's bed or bedroom and is the only published scale to quantify the stim-

ulus-control paradigm. Test-retest correlation (r = .88) and internal consistency data (K-R 20 = .70) indicate acceptable reliability, and the original authors reported good discriminant validity[32]. However, we revised the scale (SBSRS-R) to more fully address stimulus-control principles. Seven items were added, based on Bootzin's revision of his stimulus-control instructions.[7] For example, "I take naps during the day or evening" and "If I can't get to sleep within about 20 minutes I get up and move to another room until I feel sleepy again" were added, and 14 previous items that simply repeated statements, first in relation to "around sleeping time" and second to "during the day," were reduced to 7 by amending the instructions for the questionnaire. The revised SBSRS-R[a], therefore, had 14 items rated, as in the original, on a 5-point Likert scale from *never* to *very often* (α = .69 this study, n = 53).

The Pre-Sleep Arousal Scale (PSAS)[9] is a 16-item questionnaire that yields separate scale scores for physiologic arousal and cognitive arousal. Both scales have acceptable internal consistency (PSASphysiol: α =. 84 and 0.81 for good sleepers and insomniacs respectively; PSAScog: α = .67 and .76). Evidence for the construct validity of the PSAScog scale comes from studies where presleep intrusive thoughts have been directly audiotape recorded.[33] The PSAS is rated on a 5-point scale from 1 (*not at all*) to 5 (*extremely*), with items reflecting autonomic arousal on the PSASphysiol scale (eg, heart racing/pounding, tight/tense muscles) and mental arousal on the PSAScog (eg, worry about not falling asleep, being mentally alert).

A number of other measures relevant to cognitive arousal were also completed. These were the Sleep Disturbance Questionnaire (SDQ)[10], the Glasgow Sleep Effort Scale (GSES)[34] and the Dysfunctional Beliefs and Attitudes about Sleep scale–10-item version (DBAS-10).[29] The SDQ is a 12-item questionnaire to identify causal attributions concerning perceived sources of a sleep problem (α = .82). Items are rated on a 5-point scale from *never true* to *very often true* (eg, "I am unable to empty my mind"). The subscale "attributions concerning mental overactivity" was utilized in this study. The GSES was developed in response to the poor face and construct validity of an existing measure, the Sleep Anxiety Scale.[35] The GSES is a 7-item scale designed to measure elements of a general model of effortful preoccupation with sleep (ie, effort to sleep, attempts to control sleep, worry over failure to sleep). It is rated on a 3-point scale (*very much, to some extent, not at all*) and has acceptable internal consistency (α = .70) .[34] The DBAS-10 is a short-form version of an original 30-item scale measuring salient, affect-laden, irrational beliefs concerning the process of sleep.[36] The 10-item version is scored on 10-cm analogue lines from *strongly disagree* to *strongly agree* (α =. 69),[29] based on items shown to be sensitive to change after cognitive-behavioral therapy. The validity of the DBAS-10 has been recently supported.[37]

Finally, we included a measure of sleep hygiene. The Sleep Hygiene Awareness and Practice Scale (SHAPS)[38] has 19 items, and respondents are asked to state on how many nights per week they typically go to bed hungry, take caffeine within 4 hours of bedtime, exercise within 2 hours of bedtime, have their sleep disturbed by noise, etc. The original authors demonstrated that insomniacs practiced poorer sleep hygiene than good sleepers, and the SHAPS has been widely used both in clinical settings and in research.

RESULTS

Descriptive and Clinical Variables

There were proportionately more women in the PI group (PI = 4 men/14 women, I-MD = 7 men /11 women, GS = 8 men /10 women); however, this difference was not statistically significant [χ^2 (2, n = 54) = 2.11, P = .35]. Participants ranged from 21 to 64 years of age, and average age per group was late 30s or early 40s (Table 1). Both PI and I-MD groups reported insomnia of over 9 years in duration. As expected, I-MD participants had significant-

Table 1—Descriptive and Clinical Information for Each Experimental Group and Results of 1-Way Analysis of Variance with Posthoc Analyses

	Group*						
	PI	**I-MD**	**GS**	**df**	**F**	**P**	**posthoc (Scheffé)**
Age, y							
Mean	44.39	39.94	36.06	2, 51	1.63	.21	-
SD	14.91	13.37	13.23				
Duration of sleep problem, y							
Mean	9.47	9.47	-	2, 51	8.50	< .001	I-MD, PI > GS
SD	10.39	9.06					
BDI score							
Mean	8.44	28.56	1.44	2, 51	87.3	< .001	I-MD > PI > GS
SD	7.11	8.13	2.43				
BAI score							
Mean	5.61	25.56	2.00	2, 51	37.0	< .001	I-MD > PI, GS
SD	5.36	14.09	2.72				

*n = 18 for each group.
PI refers to psychophysiologic insomnia; I-MD, insomnia associated with mental disorder; GS, good sleeper; BDI, Beck Depression Inventory; BAI, Beck Anxiety Inventory.

ly higher symptomatology on the BDI (mean = 28.6), 15 of 18 were on antidepressant medication (compared with 1 in the PI and 0 in the GS groups), and 15 attended mental health services (compared with 0 in the PI or GS groups). PI participants scored lower on the BDI (mean = 8.4) but, nevertheless, significantly higher than the GS group. Recent BDI data from 139 of our clinic-presenting insomniacs suggest comparability with the present PI group (mean = 10.9, SD = 9.12).[29] An independent rater listened to a random sample of 3 audiotaped SCID-I interviews for each group (PI, I-MD, GS), and ratings for each item in each interview were compared. Cohen's k showed that, overall, the ratings made were identical (k = 1.00). Six individuals in the I-MD group had a comorbid anxiety diagnosis of generalised anxiety disorder, 6 had a diagnosis of social phobia, 7 had a diagnosis of panic disorder with agoraphobia, and 2 had a diagnosis of specific phobia. Seven participants had more than 1 comorbid diagnosis. Only 1 participant in the PI group and 2 in the I-MD group were on hypnotic medication, and the I-MD group scored considerably higher on BAI symptoms than did either the PI or GS groups (Table 1).

Sleep Variables

Several sleep variables exhibited nonnormal distributions, and the Levine statistic revealed that group variances on some measures were not equal. However, analysis of variance is relatively robust when normality assumptions are violated, and in the case of unequal variances, unless sample sizes are unequal.[39,40] Analysis of variance models, therefore, were applicable to our data. Multivariate analyses of variance were applied to compare sleep-pattern variables between groups. The 4 sleep-diary variables (SOL, WASO, TST, SE) were entered into 1 model, and this procedure was repeated for the same 4 actigraphic variables. There was a significant effect of the factor variable (Wilks' l) for both sleep diary and actigraphic variables ($F_{8,96} = 4.22$, $P < .001$ and $F_{8,96} = 2.64$, $P < .01$, respectively). Separate analyses of variance were then applied to investigate between-group differences on specific sleep variables (Table 2).

The GS group had significantly lower diary reports of SOL and WASO and significantly greater TST and higher SE than either

Table 2—Sleep-Diary And Actigraphy Estimates of Sleep for Each Experimental Group and Results of 1-Way Analysis of Variance with Posthoc Analyses

	Group*						
	PI	**I-MD**	**GS**	**df**	**F**	**P**	**posthoc (Scheffé)**
SOL diary							
Mean	41.40	52.22	11.02	2,51	9.99	< .001	I-MD, PI
SD	27.24	40.47	9.14				>GS
WASO diary							
Mean	60.75	55.64	4.72	2,51	6.99	.002	I-MD, PI
SD	49.48	70.28	5.04				>GS
TST diary							
Mean	343.7	381.9	446.2	2,51	5.66	.006	GS > PI
SD	86.35	120.7	59.43				
SE diary							
Mean	77.63	75.79	95.87	2,51	13.8	< .001	GS >
SD	11.68	18.31	3.01				PI, I-MD
SOL actigraph							
Mean	13.58	25.99	8.11	2,51	3.81	.03	I-MD>GS
SD	10.41	31.40	9.86				
WASO actigraph							
Mean	41.47	63.70	50.87	2,51	2.53	.09	-
SD	17.68	40.09	27.20				
TST actigraph							
Mean	417.8	388.4	376.8	2,51	1.70	.19	-
SD	81.25	75.56	43.46				
SE actigraph							
Mean	87.38	80.06	86.81	2,51	4.05	.02	PI >I-MD
SD	4.90	12.97	5.36				

*n = 18 for each group.
PI refers to psychophysiologic insomnia; I-MD, insomnia associated with mental disorder; GS, good sleeper; BDI, Beck Depression Inventory; BAI, Beck Anxiety Inventory; SOL, sleep-onset latency in minutes; WASO, wake time after sleep onset in minutes; TST, total sleep time in minutes; SE, sleep efficiency, %.

the PI or I-MD groups. There were no significant differences between the insomniac groups on self-reported sleep. Both the PI and I-MD groups took more than 40 minutes to fall asleep, had around 1 hour of wakefulness during the night, and had SEs around 75% (Table 2). Interestingly the PI group reported sleeping on average less (5 hours 43 minutes) than the I-MD group (6 hours 21 minutes), but this difference was not statistically significant using conservative posthoc testing.

Although the omnibus multivariate analyses of variance test also suggested significant between-group differences in actigraphic sleep, no significant between-group differences emerged for WASO or TST (Table 2). The I-MD group exhibited significantly greater SOL than the control group, and the PI group had a significantly higher SE than the I-MD group. Product-moment correlations were carried out to investigate the relationship between sleep-diary and respective actigraphic variables across groups. Only the correlation coefficient for SOL was statistically significant for the total group (n = 54: SOL: r = .62, P < 0.01; WASO: r = .04, P > 0.10; TST: r = .17, P > 0.10; SE: r = .13, P > 0.10). Within-group subjective/ actigraphic association was significant in I-MD participants for SOL (r_s = .691, P = .002: n = 18), and in GS participants for TST (r_s = .577, P = .012: n = 18).

Rating-Scale Variables Measuring the Insomnia Experience

One participant failed to complete the SBSRS-R, and 3 the DBAS-10. A series of 7, 1-way analyses of variance was applied, the results of which are presented in Table 3. Due to the multiple comparisons made, Bonferroni correction resulted in the critical value for significance of P = .007 (.05/7).

No significant differences between insomniacs (PI, I-MD) and GS were observed on the SBSRS-R, suggesting that the frequency of engagement in sleep-incompatible activities was equivalent across groups. The I-MD group, however, did exhibit poorer sleep hygiene practices (SHAPS) relative to both the PI and GS groups, and, similarly, the PI group had poorer sleep hygiene than the GS group. The GS and PI groups did not differ from one another on PSASphysiol, but both reported significantly less physiologic arousal than did the I-MD group. By comparison, however, both insomniac groups reported greater cognitive arousal (PSAScog) than the GS, although the I-MD group had higher scores than the PI group. Both insomniac groups again scored significantly higher than the GS group on the SDQ (attributions to cognitive arousal) and the DBAS-10 (dysfunctional cognitions regarding sleep), but the PI and I-MD groups did not differ from each other on either of these measures. Finally, on the

Table 3—Comparison of Experimental Groups and Results of 1-Way Analysis of Variance and Posthoc Analyses on Model-Testing Measures

	Group*						
	PI	**I-MD**	**GS**	***df***	**F**	***P***	**posthoc (Scheffé)**
SBSRS-R							
Mean	34.11	38.89	33.65	2, 50	2.44	.10	-
SD	7.63	9.07	6.54				
SHAPS							
Mean	32.89	43.39	21.11	2, 51	16.45	< .001	I-MD > PI > GS
SD	9.22	14.65	10.39				
PSASphysiol							
Mean	11.89	20.44	8.67	2, 51	22.79	< .001	I-MD > PI, GS
SD	4.30	8.28	0.91				
PSAScog							
Mean	24.94	30.83	14.00	2, 51	39.46	< .001	I-MD > PI >GS
SD	8.19	4.29	3.80				
SDQ							
Mean	12.33	13.11	7.17	2, 51	28.50	< .001	I-MD, PI >GS
SD	3.50	2.08	1.79				
GSES							
Mean	13.39	15.39	7.50	2, 51	71.79	< .001	I-MD > PI > GS
SD	3.11	1.61	0.62				
DBAS-10							
Mean	47.73	54.85	30.07	2, 48	12.25	< .001	I-MD, PI >GS
SD	15.92	21.88	13.71				

*n = 18 for each group except for Sleep Behavior Self-Rating Scale-Revised (SBSRS-R) missing 1 case, Dysfunctional Beliefs and Attitudes about Sleep Scale (10-item version) (DBAS-10) missing 3 cases.
PI refers to psychophysiologic insomnia; I-MD, insomnia associated with mental disorder; GS, good sleeper; SBSRS-R,; SHAPS, Sleep Hygiene Awareness and Practice Scale; PSASphysiol, physiologic subscale of the Pre-Sleep Arousal Scale; PSAScog, cognitive subscale of the PSAS; SDQ, Sleep Disturbance Questionnaire; GSES, Glasgow Sleep Effort Scale.

GSES, I-MD and PI participants reported higher levels of sleep effort than GS, but the I-MD group also scored significantly higher than the PI group.

The preceding analyses, therefore, indicate between-group differences on 6 of the 7 variables studied. However, possible relationships among variables need to be considered. Therefore, a correlation matrix was calculated comprising participants from the 2 insomnia groups (Table 4), which also included depression and anxiety scale scores. Inspection of Table 4 demonstrates that pairings of scores for the majority of variables, with the general exception of those involving sleep behavior (SBSRS-R) and sleep hygiene (SHAPS), were significantly intercorrelated ($r >$ .33). It is particularly noteworthy that measures of presleep arousal (PSASphysiol, PSAScog) and psychopathology (BAI and BDI) were associated ($r >= .56$). The correlation between BAI and PSASphysiol was $r = .87$.

If we are to understand the relative influence of these variables, and how they may interact, a multivariate model is required. This also helps to address the fact that our measures share method variance (self-report ratings). Thus, separate logistic-regression analyses were conducted for each of the comparisons of interest—PI/GS, I-MD/GS and PI/I-MD. These analyses should be interpreted cautiously because of the small sample sizes. Nevertheless, our aim was to identify which variable or subset of variables might best discriminate between groups and, so, to suggest lines of inquiry for future study. Variables were entered in a forward stepwise model.

For the discrimination of psychophysiologic insomnia from good sleep (PI/ GS), sleep effort (GSES) entered on the first step (B = 11.85, standard error B = 108.0), correctly identifying 100% of PI (sensitivity) and 94% of GS (specificity) ($\chi^2 = 44.67$, $df = 1$, $P < .001$). No other variable added to this discrimination. Similarly, GSES entered as the only predictor for the I-MD/GS comparison (B = 8.66, standard error B = 2055.5), correctly allocating 100% of cases to I-MD and GS groups, respectively ($\chi^2 = 44.24$, $df = 1$, $P <$ .001). For the prediction of PI versus I-MD insomniacs, only BDI score entered (B = 28.90, standard error B = 1374.4), again correctly allocating 100% of cases ($\chi^2 = 45.48$, $df = 1$, $P < .001$), suggesting that symptomatic mood was the critical between-group factor here, rather than any insomnia specific variable.

DISCUSSION

Our purpose was to explore explanatory mechanisms for psychophysiologic insomnia by investigating the sensitivity and specificity of commonly used insomnia research tools in the discrimination of PI from I-MD and from good sleep. We found no between-group differences on a measure of sleep-related stimulus control, and self-reported somatic arousal was higher in I-MD than either GS or PI. Both I-MD and PI had poorer sleep hygiene and were particularly characterized by heightened mental arousal. Our measure of "effortful preoccupation with sleep" best discriminated both PI and I-MD from GS, but only depressive symptomatology discriminated I-MD from PI. These results were drawn from a small sample in this preliminary exploratory study. Accordingly, they should be interpreted cautiously and require replication using larger samples and samples in which the nature and severity of depressive and comorbid anxiety disorders can be specifically studied.

First, our results confirm that both insomnia groups slept poorly relative to GS, according to self-report sleep-diary data. Mean SE for the PI and I-MD groups was around 77%, compared with 96% in the GS group, respectively, which are 10% below and 10% above the recognized "cut-off" for sleep disorder.[41] This pat-

Table 4—Pearson Product-Moment Correlations Between Scales for the Combined Primary Insomnia and Insomnia with Mental Disorder Groups*

Scale	1	2	3	4	5	6	7	8	9
SBSRS-R	—	.31	.13	.34†	.20	.05	.38†	.33†	.20
SHAPS		—	.45†	.20	.14	.26	.33	.45†	.50†
PSASphysiol			—	.58†	.51‡	.45†	.30	.68‡	.87‡
PSAScog				—	.59‡	.71‡	.39†	.59‡	.56‡
SDQ					—	.34†	.39†	.41†	.35†
GSES						—	.48†	.38†	.21
DBAS-10							—	.48†	.26
BDI								—	.78‡
BAI									—

*All n = 36 except Dysfunctional Beliefs and Attitudes about Sleep Scale (10-item version) (DBAS-10), with 33.
PI refers to psychophysiologic insomnia; I-MD, insomnia associated with mental disorder; GS, good sleeper; SBSRS-R, Sleep Behavior Self-Rating Scale-Revised; SHAPS, Sleep Hygiene Awareness and Practice Scale; PSASphysiol, physiologic subscale of the Pre-Sleep Arousal Scale; PSAScog, cognitive subscale of the PSAS; SDQ, Sleep Disturbance Questionnaire; GSES, Glasgow Sleep Effort Scale; BDI, Beck Depression Inventory; BAI, Beck Anxiety Inventory.
†P < .05
‡P < .01

tern was reflected also in symptom measures of "initial insomnia," "sleep-maintenance insomnia," and "insufficient sleep" (DSM-IV) because people with insomnia took significantly longer to fall asleep (40-50 minutes compared with 10 minutes), were more wakeful in the night (around 60 minutes compared with 5 minutes), and slept less than GS (around 6 hours compared with 7 hours 30 minutes). PI and I-MD groups did not differ from each other on sleep self-reports. As expected, however, the former had higher scores for depressive symptoms on the BDI (> 2 SD higher than for PI). Mean BDI score for PI participants was below the clinical range. Symptomatic anxiety on the BAI was also greater in the I-MD group. Furthermore, the majority of the I-MD group were on antidepressant pharmacotherapy and in contact with mental health services.

While these data on between-group differences suggest that our selection and allocation to experimental groups (GS, PI, I-MD) were valid, they also reveal an important confound that should be taken into account in interpreting our results. The nearly universal use of antidepressants in the I-MD group implies that this is in effect a "treated I-MD" group, albeit with a relatively high mean BDI score of 28. Future research, therefore, needs to include an "untreated" I-MD group to control for the effects of medication use. It would also be helpful to separately consider treatment responsive and nonresponsive cohorts in future studies.

In comparison with self-report, actigraphic data generally failed to confirm between-group differences in sleep, and correlation with diary measures was low in all 3 groups. SE was estimated in the range of 80% to 87% across groups, and overall association between subjective and objective data was $r = .13$ (n = 54). Most strikingly, participants in the GS group who had no history of sleep disorder; no current sleep complaint; and, by their own report, slept well during the study week provided actigraphic data suggesting that they slept only around 6 hours (self-report 7.5 hours) and that they had an average of 50 minutes of wakefulness per night (self-report 5 minutes). These results, like those from another report,[42] therefore, are strongly suggestive of validity problems with this form of measurement.

Second, the ICSD-R suggests that in PI, sufferers engage in sleep-incompatible behavior in the bedroom. However, our results evidence no differences in such behavior between GS and either PI or I-MD. It is, of course, possible that our measure of sleep-incompatible behavior (SBSRS-R) was inadequate. More plausible perhaps is the possibility that the same behaviors present, even with similar frequency, but that they have different consequences for good and poor sleepers. Thus, and for example, reading in bed may have a sleep-preventing effect only for poor sleepers. Nevertheless, our findings are consistent with early reports where insomniacs and GS exhibited similar sleep behavior[43] and where experimental intervention, using components of stimulus-control treatment, suggested that the bed becoming a "discriminative stimulus for sleep" was not the active mechanism.[44] In the only recent study, people with insomnia were found not to differ from GS on daytime napping, sleep scheduling, whether they stayed in bed or got up when unable to sleep, or on engagement in sleep-incompatible activities.[45]

Third, according to the ICSD-R, PI is also characterized by conditioned physiologic arousal. This study focused upon self-report, so no direct measurement was taken of autonomic or cortical arousal. The physiologic arousal subscale of the PSAS, nevertheless, is widely used, and, interestingly, we found that only I-MD differed from GS on this measure. This suggests that somatic arousal may be more characteristic of patient report where insomnia is associated with mental disorder. It should be noted, however, that many of our I-MD participants had symptoms of anxiety and that we found a strong positive correlation between PSASphysiol and state anxiety on the BAI ($r = .89$). The association of physiologic and anxiety symptoms in our I-MD sample could account for our results. Alternatively, there may be problems with the construct validity of this subscale of the PSAS, if it is simply a proxy for somatized anxiety. Further evidence on physiologic arousal may come from our data on the SHAPS because it has been argued that inadequate sleep hygiene delays sleep onset primarily through heightened arousal.[14] For example, caffeine produces increased arousal on metabolic measures and reports typical of insomniac complaint.[46] In the present study, sleep-hygiene practice was found to differ across the experimental groups, with I-MD having higher SHAPS scores (poorer practices) than either GS or PI, and PI having higher scores than GS.

Fourth, not all objective poor sleepers complain of insomnia, and not all subjective insomniacs have poor sleep,[47] suggesting that physiologic arousal alone may be an insufficient explanation of insomnia. We found that the I-MD group reported greater mental arousal than the PI group on the PSAScog and that both I-MD and PI groups reported more mental arousal than the GS. Insomniac thoughts during the presleep period are often worried and negative in content.[48,35] Perhaps people with insomnia associated with depression or anxiety appraise things even more negatively than do psychophysiologic those with PI? It is interesting, therefore, that the DBAS-10 results tend to confirm the PSAScog findings. Both PI and I-MD groups scored more than 2 SD higher than GS on this measure, and, although the mean score for I-MD was not significantly higher than for PI, the trend again was in that direction. It seems then that people with insomnia may be more prone to sleep-related thinking errors than are GS. This finding was further reinforced by our data from the SDQ, where both PI and I-MD groups had higher scores on a measure of causal attribution concerning mental overactivity in bed.

Moreover, with respect to mental arousal, our data from the GSES are particularly interesting. They reflect a similar distinction, in terms of severity, between I-MD and PI, with both groups exhibiting higher sleep effort that the GS group. However, our analyses suggest that an explanatory model including "effortful preoccupation with sleep" may be of special importance in understanding the cognitive arousal associated with insomnia. We observed strong intercorrelations among our variables measuring arousal, despite these being supposedly differing constructs, perhaps indicating that a common underlying insomnia construct may exist. We then found that logistic-regression models identified the GSES as the best measure to discriminate insomniacs from GS. Furthermore, this effect applied for both the PI and the I-MD groups, yielding 100% sensitivity in both cases and 94% and 100% specificity for each respectively. This result, when coupled with evidence that I-MD and PI groups were perfectly differentiated using BDI scores alone, without recourse to measures of sleep-related behavior or cognition, suggests that a *continuum* may exist across "primary" and "secondary" insomnia (at least where it is associated with depression or anxiety). This suggestion is supported by other recent work on symptom reports in patients with severe chronic insomnia, which have found that "depression-related insomnia" and PI were separable only by

characteristic symptoms of depression.[49] A caveat must be introduced, however, in relation to the fact that the BDI discriminated so well. Our patients with PI were excluded for high BDI scores, and I-MD patients were included partly on this basis. Therefore, although valid from a diagnostic standpoint, our BDI results do not reflect an independent observation in this study.

PI and I-MD, therefore, may have much in common. They may have similar mechanisms associated with their persistence and may be amenable to similar treatment, whether behavioral or pharmacologic. ICSD-R and DSM-IV categories imply that there are different pathways in these insomnia subtypes. This may not be correct. Clearly, further rigorous investigation is required to investigate "continuum versus category" hypotheses, but our results suggest that insomnia associated with depression or anxiety may simply be a more severe variant of PI. This would make sense in the context of insomnia being a risk factor for depressive illness.[21] However, an alternative explanation is that the absence of difference between the insomnia groups reflects an orthogonal relationship between insomnia and depression. There may be commonalities in insomnia just as the diagnosis of a major depressive disorder follows the same criteria regardless of whether it is "primary" or "associated with" other conditions. Therefore, both the "severity continuum" and "orthogonal construct" hypotheses appear to merit further consideration.

Further comparative work is required to replicate our findings using concomitant measures of behavior, cognition, and physiology relevant to the insomniac experience. Ideally, self-report, observational, and psychophysiologic measures should be gathered independently of one another, in order to limit the method variance associated with using any 1 form of data gathering on its own. In particular, we would like to highlight the outstanding need for better measurement, and component investigation, of the stimulus-control paradigm. The SBSRS-R may be a useful scale to describe and quantify sleep-related behavior, but it does not assess the critically important *relationship* between behavior and consequence. Inadequate stimulus control is central to diagnostic conceptualization of PI,[2] and stimulus-control treatment is the best evidenced nonpharmacologic intervention,[25] yet we still lack evidence that sleep and bedroom cues are actually associated with differential conditioning in insomnia.

Finally, if our finding is confirmed that it is primarily "effortful preoccupation with sleep" that best discriminates people with insomnia from GS, then perhaps it would be productive for researchers to spend more time studying normalcy, ie, the (perhaps relatively unremarkable) nighttime behavior, beliefs, attitudes, efforts, and physiologic responses of GS. After all, the ultimate goal of insomnia therapies should be to reinstate good sleep, not simply to achieve statistically or even clinically significant change.

REFERENCES

1. Diagnostic and Statistical Manual of the Mental Disorders, 4th ed. Washington, DC: American Psychiatric Association; 1994.
2. The International Classification of Sleep Disorders, Revised: Diagnostic and Coding Manual. Rochester, MN: American Sleep Disorders Association; 1997.
3. Monroe LJ. Psychological and physiological differences between good and poor sleepers. J Abnorm Psychol 1967;72:255-64.
4. Bonnet MH, Arand DL. 24-hour metabolic rate in insomniacs and matched normal sleepers. Sleep 1995;18:581-8.
5. Perlis ML, Merica H, Smith MT, Giles DE. Beta EEG activity and insomnia: theoretical review. Sleep Med Rev 2001;5:365-376.
6. Bootzin RR. Stimulus control treatment for insomnia. Proc Am Psychol Assoc 1972;7:395-6.
7. Bootzin RR, Epstein D, Wood JM. Stimulus control instructions. In Hauri PJ, ed. Case Studies in Insomnia. New York: Plenum Press; 1991:19-28.
8. Lichstein KL, Rosenthal TL. Insomniacs' perceptions of cognitive versus somatic determinants of sleep disturbance. J Abnorm Psychol 1980;89:105-7.
9. Nicassio PM, Mendlowitz DR, Fussell JJ, Petras L. The phenomenology of the pre-sleep state: the development of the pre-sleep arousal scale. Behav Res Therap 1985;23:263-71.
10. Espie CA, Brooks DN, Lindsay WR. An evaluation of tailored psychological treatment of insomnia. J Behav Therap Exp Psychiatry 1989;20:143-53.
11. Spielman AJ, Glovinsky PB. The varied nature of insomnia. In Hauri PJ, ed. Case Studies in Insomnia. New York: Plenum Press; 1991:1-15
12. Morin CM. Insomnia: Psychological Assessment and Management. New York: Guilford Press; 1993.
13. Espie CA. The Psychological Treatment of Insomnia. Chichester, England: J. Wiley & Sons; 1991.
14. Espie CA. Insomnia: conceptual issues in the development, persistence and treatment of sleep disorder in adults. Annu Rev Psychol 2002;53:215-43.
15. Perlis ML, Giles DE, Mendelson WB, Bootzin RR, Wyatt J K. Psychophysiological insomnia: the behavioural model and a neurocognitive perspective. J Sleep Res 1997;6:179-88.
16. Ford DE, Kamerow DB. Epidemiologic study of sleep disturbance and psychiatric disorders: an opportunity for prevention? JAMA 1989;263:1479-84.
17. Krieg JC, Lauer CJ, Hermle L, von Bardeleben U, Pollmacher T, Holsboer F. Psychometric, polysomnographic and neuroendocrine measures in subjects at high risk for psychiatric disorders: preliminary results. Neuropsychobiology 1990;23:57-67.
18. Eaton WW, Badawi M, Melton B. Prodromes and precursors: epidemiological data for primary prevention of disorders with slow onset. Am J Psychiatry 1995;152:967-72.
19. Breslau N, Roth T, Rosenthal L, Andreski P. Sleep disturbance and psychiatric disorders: a longitudinal epidemiological study of young adults. Soc Biol Psychiatry 1996;39:411-8.
20. Perlis ML, Giles DE, Buysse DJ, Tu X, Kupfer DJ. Self-reported sleep disturbance as a prodromal symptom in recurrent depression. J Affect Dis 1997;42:209-12.
21. Riemann D, Voderholzer U. Primary insomnia: a risk factor to develop depression? J Affect Dis 2003;76:255-9.
22. Lichstein KL, Wilson NM, Johnson CT. Psychological treatment of secondary insomnia. Psychol Aging 2000;15:232-40.
23. Morin CM, Culbert JP, Schwartz MS. Non-pharmacological interventions of insomnia: a meta-analysis of treatment efficacy. A J Psychiatry 1994;151:1172-80.
24. Murtagh DR, Greenwood KM. Identifying effective psychological treatments for insomnia: a meta-analysis. J Consult Clin Psychol 1995;63:79-89.
25. Morin CM, Hauri PJ, Espie CA, Spielman A, Buysse DJ, Bootzin RR. Nonpharmacologic treatment of chronic insomnia: an American Academy of Sleep Medicine Review. Sleep 1999;22:1134-56.
26. Smith MT, Perlis ML, Park A, et al. Comparative meta-analysis of pharmacotherapy and behavior therapy for persistent insomnia. Am J Psychiatry 2002;159:5-11.
27. First MB, Spitzer RL, Gibbon M, Williams JB. Structured Clinical Interview for DSM-IV Axis I Disorders (SCID-I): Clinician version. Washington DC: American Psychiatric Press; 1997.
28. Beck AT, Steer RA. Beck Depression Inventory (BDI) Manual. San Antonio: The Psychological Corporation; 1987.

29. Espie CA, Inglis SJ, Harvey L, Tessier S. Insomniacs' attributions: psychometric properties of the Dysfunctional Beliefs and Attitudes about Sleep Scale and the Sleep Disturbance Questionnaire. J Psychosom Res 2000;48:141-8.
30. Beck AT. Beck Anxiety Inventory (BAI). San Antonio: The Psychological Corporation; 1990.
31. Practice parameters for the use of actigraphy in the clinical assessment of sleep disorders. American Sleep Disorders Association. Sleep 1995:18:285-7.
32. Kazarian SS, Howe MG, Csapo KG. Development of the Sleep Behavior Self-Rating Scale. Behav Therap 1979;10:412-7.
33. Wicklow A, Espie CA. Intrusive thoughts and their relationship to actigraphic measurement of sleep: towards a cognitive model of insomnia. Behav Res Therap 2000;38:679-93.
34. Broomfield N, Espie CA. Initial insomnia and paradoxical intention: an experimental investigation of putative mechanisms using subjective and actigraphic measurement of sleep. Behav Cogn Psychotherapy 2003;31:313-24.
35. Fogle DO, Dyal JA. Paradoxical giving up and the reduction of sleep performance anxiety in chronic insomniacs. Psychotherapy Theory Res Pract 1983;20:21-30.
36. Morin CM, Stone J, Trinkle D, Mercer J, Remsberg S. Dysfunctional beliefs and attitudes about sleep among older adults with and without insomnia complaints. Psychol Aging 193;8:463-7.
37. Edinger JD, Wohlegemuth WK, Radtke RA, Marsh GR, Quillian RE. Does cognitive-behavioral insomnia therapy alter dysfunctional beliefs about sleep? Sleep 2001:24:591-9.
38. Lacks P, Rotert M. Knowledge and practice of sleep hygiene techniques in insomniacs and good sleepers. Behav Res Therap 1986;24:365-8.
39. Hinkle DE, Wiersma W, Jurs SG. Applied Statistics for the Behavioural Sciences, 3rd ed. Boston: Houghton Mifflin Company; 1994.
40. Tabachnick BG, Fidell LS. Using Multivariate Statistics, 3rd ed. New York: Harper Collins; 1996.
41. Frankel BL, Coursey RD, Buchbinder R, Snyder F. Recorded and reported sleep in primary chronic insomnia. Arch Gen Psychiatry 1976;33:615-23.
42. Pollak CP, Tryon WW, Nagaraja H, Dzwonczyk R. How accurately does wrist actigraphy identify the states of sleep and wakefulness? Sleep 2001;24:957-65.
43. Haynes SN, Adams AE, West S, Kamens L, Safranek, R. The stimulus control paradigm in sleep-onset insomnia: a multi-method assessment. J Psychosom Res 1982;26:333-9.
44. Zwart CA, Lisman SA. Analysis of stimulus control treatment of sleep-onset insomnia. J Consult Clin Psychol 1979;47:113-9.
45. Harvey AG. Sleep hygiene and sleep-onset insomnia. J Nerv Ment Dis 2000;188:53-5.
46. Bonnet MH, Arand DL. Caffeine use as a model of acute and chronic insomnia. Sleep 1992;15:526-36.
47. Edinger JD, Sullivan RJ, Bastian LA., Hope TV, Young M, et al. Insomnia and the eye of the beholder: are there clinical markers of objective sleep disturbances among adults with and without insomnia complaints? J Consult Clin Psychol 2000;68:586-93.
48. Borkovec TD, Lane TW, Van Oot PH. Phenomenology of sleep among insomniacs and good sleepers. Wakefulness experience when cortically asleep. J Abnorm Psychol 1981;90:607-9.
49. Moul DE, Nofzinger EA, Pilkonis PA., Houck PR, Miewald JM, Buysse, DJ. Symptom reports in severe chronic insomnia. Sleep 2002;25:553-63.

J. Sleep Res. (2005) **14,** 401–407

Towards a valid, reliable measure of sleep effort

NIALL M. BROOMFIELD and COLIN A. ESPIE
Department of Psychological Medicine, Gartnavel Royal Hospital, University of Glasgow, Glasgow, UK

Accepted in revised form 4 July 2005; received 6 September 2004

SUMMARY A frequent clinical observation is that patients with insomnia strive to control their sleep. However, sleep is an involuntary physiological process, which cannot be placed under full voluntary control. Therefore, direct, voluntary attempts to control sleep may actually exacerbate and perpetuate insomnia. To date, no reliable scale has been available to test this hypothesis directly. Moreover, while sleep effort is a core International Classification of Sleep Disorders – Revised criterion for psychophysiological insomnia, clinicians lack a reliable measure with which to assess the construct. In this initial scale validation study, we present psychometric data for the Glasgow Sleep Effort Scale based on a relatively small but representative sample of patients with insomnia and good sleepers. The clinical and research value of the new scale is discussed and future research directions are described.

KEYWORDS insomnia, paradoxical intention, reliability, sleep effort, validity

INTRODUCTION

Sleep is a fundamental life process, much like eating or drinking. Presumably therefore, inability to sleep is significantly threatening (Broomfield *et al.*, 2005; Harvey, 2002). This may explain the frequent clinical observation that patients with insomnia often strive to control their sleep. As sleep is an involuntary physiological process, which cannot be placed under full voluntary control, any effort to control sleep is likely to fail. Effort to sleep may, therefore, represent a key perpetuating factor in insomnia.

We conceptualize the construct of *sleep effort* as multicomponent, comprising core behavioural and cognitive elements, and describe here an initial validation study of the Glasgow Sleep Effort Scale (GSES), a new self-report scale designed to measure effort.

This initial validation study is important and timely for several reasons. First, as already indicated, the behaviour and cognition of insomnia patients often involve heightened sleep effort. This may express itself as performance anxiety about sleep, a need for control over sleep, and/or trying too hard to sleep. Interestingly, and we think this is important, good sleepers tend towards the opposite. When asked what they do to fall asleep, good sleepers usually: (i) appear bewildered and (ii) report not consciously delivering any behaviour. This supports good sleep as passive and effortless, and implicates effort as disruptive (Espie, 2002). Reliance on clinical observation to inform psychological theory testing is of course a critical tradition across psychological research studies (Salkovskis, 2002) and insomnia research is no exception (Tang and Harvey, 2003).

Secondly, consensus expert opinion has for some time recognized sleep effort as a relevant aetiological factor. The International Classification of Sleep Disorders – Revised (ICSD-R) and its predecessor (ICSD), both cite 'trying too hard to sleep' as a core factor leading to learned sleep preventing associations in psychophysiological insomnia (American Sleep Disorders Association, 1990, 1997). Yet we lack the means to address, diagnostically, the presence or otherwise of effort. Several existing scales integrate relevant single items [e.g. The Dysfunctional Beliefs and Attitudes about Sleep (DBAS) scale (Morin *et al.*, 1993); Sleep Disturbance Questionnaire (Espie *et al.*, 1989)]. This suggests acknowledgement of the aetiological relevance of sleep effort. Yet no dedicated measure is available.

Thirdly, previous work suggests sleep effort can discriminate good and poor sleepers. Kohn and Espie (in press) compared good sleepers with primary insomniacs and depressed insomniacs, and measured sleep effort using a pilot version of the GSES, which had not at that time been subject to psychometric evaluation. Effort was significantly higher among the two poor sleeping groups and regression analyses

Correspondence: Dr Niall M. Broomfield, Department of Psychological Medicine, Gartnavel Royal Hospital, University of Glasgow, 1055 Great Western Road, Glasgow G12 0XH, UK. Tel.: 0141 211 3904; fax: 0141 357 4899; e-mail: nmb2v@clinmed.gla.ac.uk

selected GSES as the best discriminatory measure between the two insomnia groups and good sleepers. A separate, initial validation study on the psychometrics of the GSES is now needed.

Fourthly, there is evidence that paradoxical intention therapy, an empirically supported insomnia treatment (Chesson *et al*., 1999; Morin *et al*., 1999) improves sleep by minimizing sleep effort. Insomnia patients allocated to 14-night paradoxical intention therapy (relax at lights out, keeping eyes open) showed, relative to controls, reduced effort *and* sleep improvement (Broomfield and Espie, 2003). This indicates that sleep effort is sensitive to change following psychological therapy and may act to maintain insomnia. Again, the measure of effort used was a pilot GSES not subject to psychometric evaluation. A valid reliable effort measure would allow identification of insomnia sufferers particularly suited to this therapeutic approach.

Fifthly, experimental data from the ironic cognitive control literature (Wegner, 1994) support effort as an insomnia-maintaining factor. Good sleepers directed to fall asleep urgently, under *high* mental load (listening to marching music) show significantly longer sleep latencies relative to good sleepers directed to sleep urgently under *low* mental load (relaxing music). Although requiring replication in insomnia patients, these data suggest effort to sleep, particularly under experimental stress conditions, drives wakefulness in good sleepers (Ansfield *et al*., 1996).

Finally, there is an outstanding need for a valid, reliable measure of sleep effort. The present initial validation study represents an important first step in this process. We report psychometric data for the GSES based on small, but representative samples of good sleepers and individuals with insomnia. As we believe effort impacts sleep initiation both at sleep onset and at times of sleep interruption *throughout* the night, the sample selected here includes initial and maintenance insomnia patients.

METHOD

Development of the pilot GSES

A pilot GSES not subject to psychometric evaluation has been used in two previous studies (Broomfield and Espie, 2003; Kohn and Espie, 2005). This pilot GSES was originally developed following discussions involving the authors of these papers. An exhaustive content analysis of relevant existing scales was completed, which resulted in a working model of sleep effort, integrating seven core components of sleep effort (Fig. 1). Each component was assigned a single item, forming the pilot GSES (Appendix 1), which we now evaluate. All items in GSES are valenced in the same way. And instructions make reference to sleep 'in the past week'. This sets up GSES as a present state rather than trait measure. Insomnia patients sleep well at certain times and on certain nights, a point reflected in formal diagnostic criteria (e.g. ICSD-R).

GSES field testing and psychometric evaluation

Participants

In order to examine GSES psychometric properties, insomnia patients and good sleepers were recruited. All participants were 18 years or older, with no upper age limit. The majority of insomnia participants were general practitioner referrals to a large treatment trial, the results of which are reported elsewhere (Espie *et al*., 2004). The remainder were obtained via university e-mail, as were the good sleepers.

All insomnia participants were screened using a standard in-house protocol (copy available on request) based on the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV; American Psychiatric Association, 1994) criteria for primary insomnia. This was completed at home, and considered sleep complaint (chronicity, number of poor nights sleep, latency, wake time after sleep onset), daytime functioning, and substance use and mood data. Sleep-disruptive medical conditions (e.g. chronic pain) and suspected other sleep disorders were also assessed. Follow-up telephone interviews clarified detail.

Inclusion criteria were a complaint of significant difficulty initiating and/or maintaining sleep, occurring 4+ nights per week for minimum 6 months, daytime impairment (concentration difficulty, fatigue) and a score >5 on the Pittsburgh Sleep Quality Index (PSQI) (Buysse *et al*., 1989). This cut-off discriminates good from poor sleepers adequately (Backhaus *et al*., 2002). Participants who reported taking 'prescribed medications to assist sleep' were included. Data on specific medication sort were not gathered. Patients with known sleep-disruptive medical conditions, suspected other sleep disorders (respiratory, muscular, neurological, circadian) such as depression, or substance abuse problems were excluded.

Good sleepers had no sleep disturbance complaint, failed to meet DSM-IV criteria for primary insomnia, agreed with the statement 'I am a good sleeper', described their sleep pattern in a typical month and this month as 'good' or 'very good', and scored <5 on the PSQI. These conservative criteria ensured a representative good sleeper cohort.

Measures and procedure

To examine psychometric properties, all participants completed the GSES, the Hospital Anxiety and Depression Scale (HADS; Zigmond and Snaith, 1983) and the DBAS scale (Morin *et al*., 1993). Scales were mailed out immediately following the telephone screening and completed at home.

The HADS-A (anxiety subscale) is a reliable, widely used measure of clinical anxiety. DBAS is a reliable, widely used measure of beliefs and attitudes regarding sleep. Inclusion of these supported concurrent validity analysis, by allowing an examination of whether GSES scores (i) merely represented elevated general anxiety and (ii) were associated with a known cognitive measure of sleep.

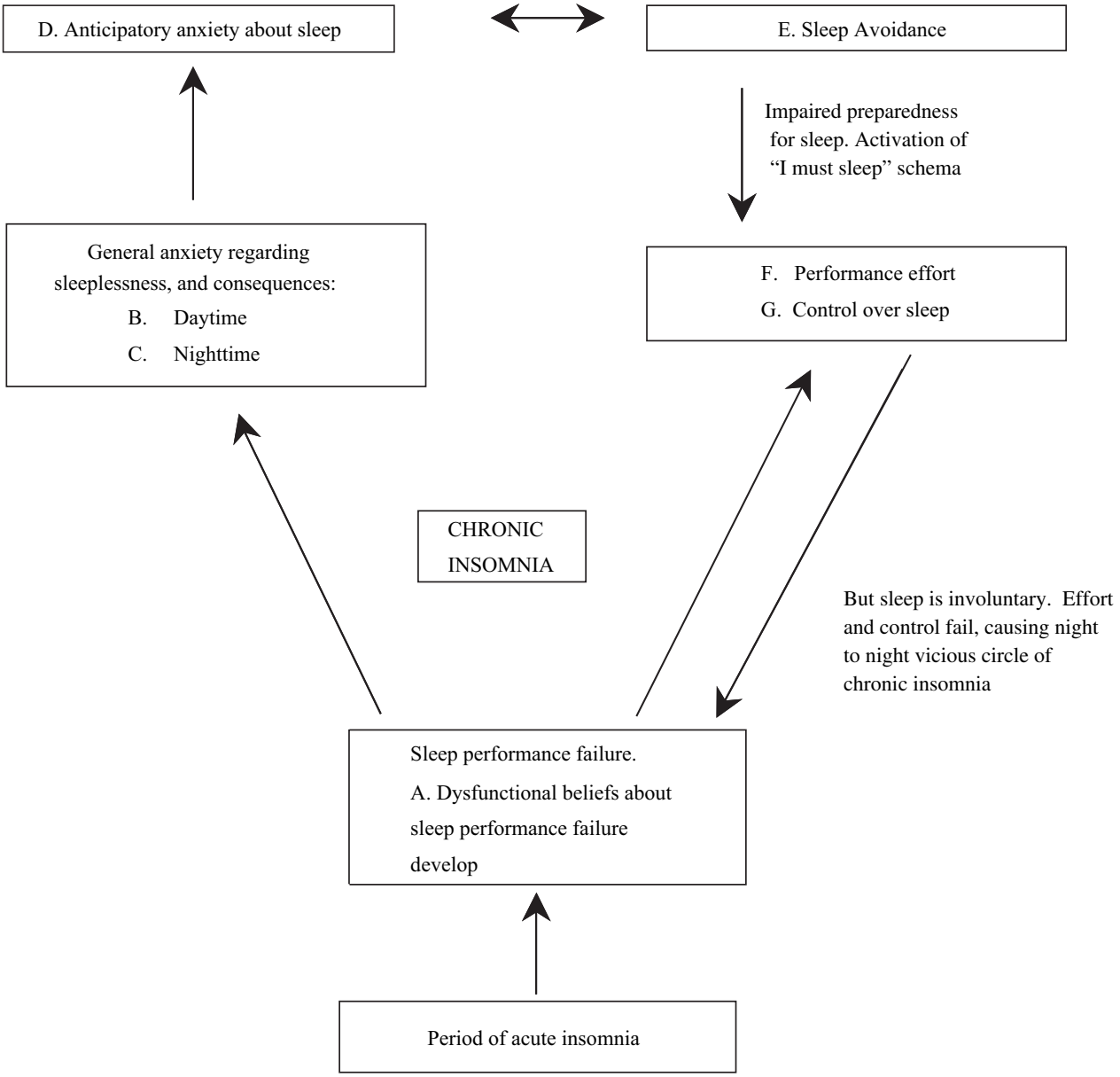


Figure 1. A preliminary working model of sleep effort.

RESULTS

Participants

A total of 89 insomnia patients and 102 good sleepers met criteria. Mean age of insomnia participants was 50.67 years, SD = 16.17 (56 were female). Mean PSQI score was 12.10, SD = 3.60 (range 5–18). Mean age of good sleepers was 34.08 years, SD = 15.98 (52 were female). Mean PSQI score was 1.96, SD = 1.30 (range 0–5).

Internal consistency

Cronbach's α was 0.77 using the insomnia patient group. Item-deletion alphas reveal the stability of a psychometric measure when individual items are systematically eliminated. For the GSES, item-deletion alphas remained high, with little variation between values (mean $\alpha = 0.75$, range 0.74–0.76). Typically, a criterion of 0.80 is deemed acceptable (Nunnaly and Bernstein, 1994). Mean corrected item–total correlation was 0.64 (range 0.49–0.73). The item-by-item correlation matrix for insomnia patients showed a range of values ($r = 0.15$–0.58) (Table 1).

Concurrent validity

Anxiety subscale scores from the HADS (Zigmond and Snaith, 1983) were available for 68 insomnia patients. For these, the association between GSES and HADS-A was non-significant ($r = 0.19$, $P > 0.1$, NS).

Table 1 Glasgow Sleep Effort Scale (GSES) item-by-item correlation matrix

GSES item	*1*	*2*	*3*	*4*	*5*	*6*	*7*
1	1.00						
2	0.44	1.00					
3	0.34	0.24	1.00				
4	0.38	0.36	0.51	1.00			
5	0.27	0.15	0.40	0.51	1.00		
6	0.37	0.29	0.52	0.56	0.42	1.00	
7	0.40	0.33	0.51	0.58	0.39	0.57	1.00

DBAS scores were available for 64 insomnia patients. For these individuals, there was also a significant moderate positive correlation between GSES and DBAS ($r = 0.50$, $P < 0.0001$).

For *all* participants, a weak positive correlation was observed between GSES and age ($r = 0.22$, $P < 0.005$). This is a small significant correlation, accounting for approximately 4% variance.

Discriminant validity

The ability of GSES to discriminate insomnia patients ($n = 89$) from good sleepers ($n = 102$) was investigated. Mean total GSES score for insomnia patients was 7.06, SD = 3.58; and 1.22, SD = 1.35 for good sleepers.

The GSES readily discriminated the groups ($t = -15.27$, d.f. = 189, $P < 0.0001$). Mean scores for individual GSES items differed significantly between groups (Table 2). Distribution examination indicated limited overlap between highest scoring good sleepers and lowest scoring insomnia patients (Fig. 2).

Given the significant association between GSES and age, to be conservative discriminant validity between groups was considered using analysis of covariance (ANCOVA), with age as covariate. GSES still readily discriminated the groups, with age partialled out [$F(1,182) = 228.52$, $P < 0.0001$].

Impact of medication

Sleep medication can affect perception of sleep. Data on whether insomnia patients used or did not use 'prescribed medication to assist sleep' were available for 70 of 89 insomnia patients (78.6%). Thirty reported non-use, 40 reported medication use. Independent *t*-test analysis indicated no significant differences between users and non-users on GSES score ($t = -0.25$, d.f. = 68, $P > 0.8$, NS).

Sensitivity and specificity

The sensitivity of a scale is the probability that an individual with the condition will be correctly classified as having the condition. Specificity is the probability that a person without the condition will be properly classified as not having the condition (Fleiss, 1981). A cut-off score of 2 correctly identified 93.3% of insomnia patients and 87.3% of good sleepers. Only seven of 89 insomniacs are not identified using this cut-off (Table 3a). Using 3 as the cutoff, 82.2% of insomnia patients and 92.2% of good sleepers were correctly identified. Only 16 of 89 insomniacs were not identified (Table 3b).

Following Bossuyt *et al*. (2003), predictive value and likelihood ratio data were also calculated (Table 4). Positive predictive and likelihood ratios were high. This suggests GSES predicts the presence of insomnia well.

Finally, using a cut-off score of 2, participants were split into two groups: 'low effort' (score of 0, 1 or 2), and 'high effort' (score 3 or more). Differences between these groups on the PSQI were then analysed. High effort participants scored higher on the PSQI (mean = 11.01, SD = 4.61; $t = -15.93$, d.f. = 189, $P < 0.0001$) relative to low effort participants (mean = 2.38, SD = 2.59).

Principal component structure of GSES

Data from 159 insomnia patients and 120 good sleepers were included in a principal component analysis (PCA) with varimax rotation. To maximize numbers, the sample used was generated from participants described here, and from two previous studies (Broomfield and Espie, 2003; Kohn and Espie, in press). One principal component was found (eigenvalue = 4.38) accounting for 62.6% of total variance. Each item loaded significantly on this factor (mean $\alpha = 0.79$, range 0.64–0.85).

Table 2 Mean Glasgow Sleep Effort Scale (GSES) item scores for insomnia (I) and good sleeper (GS) participants

	Mean item score			
	GS	*I*	*t*	*P-value*
1. I put too much effort into sleeping when it should come naturally	0.12	1.04	−10.97	0.000
2. I feel I should be able to control my sleep	0.56	1.13	− 6.27	0.000
3. I put off going to bed at night for fear of not being able to sleep	0.07	0.71	−7.34	0.000
4. I worry about not sleeping if I cannot sleep	0.25	1.17	−10.07	0.000
5. I am no good at sleeping	0.03	1.27	−16.60	0.000
6. I get anxious about sleeping before I go to bed	0.04	0.70	−7.94	0.000
7. I worry about the consequences of not sleeping	0.16	1.03	−10.26	0.000

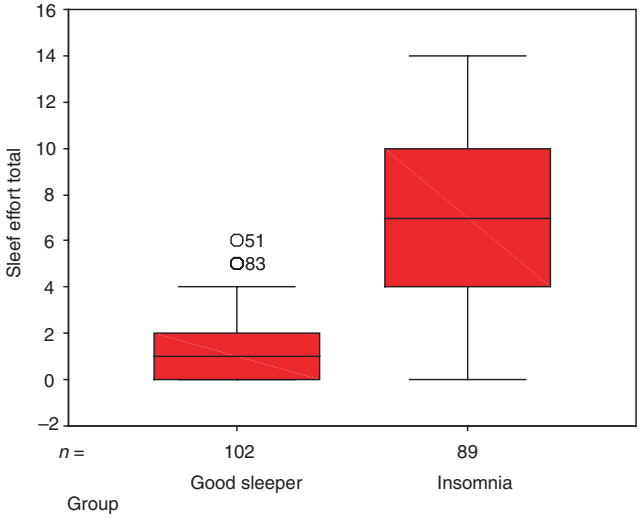


Figure 2. Box and whiskers plot demonstrating the ability of the Glasgow Sleep Effort Scale to discriminate good sleepers and insomniacs.

Table 3 Identification of insomniacs and good sleepers

	I	*GS*
(a) Using GSES cut-off of 2		
Correct	82	13
Incorrect	7	89
(b) Using GSES cut-off of 3		
Correct	73	8
Incorrect	16	94

Table 4 Diagnostic test results for the Glasgow Sleep Effort Scale score >2

Positive predictive value	0.87 (0.78–0.92)
Negative predictive value	0.94 (0.87–0.97)
Likelihood ratio for insomnia	+7.3 (4.39–12.20)
Likelihood ratio for good sleepers	−0.08 (0.04–0.17)

Range values are given within the parenthesis.

DISCUSSION

In this initial validation study, GSES psychometric evaluation was completed with 89 insomnia patients and 102 good sleepers. Field testing revealed highly acceptable data. Internal consistency and item-by-item correlation data were adequate. Item–total correlations were satisfactory, indicating no single-scale item accounts for the majority of variance. A cut-off score of 2 correctly identified 92.1% of insomnia patients and 87.3% of good sleepers, with excellent likelihood ratios at this cut-off. GSES adequately discriminated insomnia patients from good sleepers, supporting previous work (Kohn and Espie, in press). Although sleep medication can impact perception of sleep, hypnotic users did not differ from non-users on GSES. The GSES HADS-A association was non-significant, suggesting the scale is not simply measuring anxiety. Factor analysis revealed one component accounting for 62% of variance and a moderate correlation was observed with DBAS suggesting substantial unique variance (75%). This sets GSES apart from existing cognitive insomnia measures. Although requiring replication with a much larger and more carefully defined sample, these data implicate GSES as a valid, reliable measure with considerable potential for quantifying sleep effort.

Once fully validated using a larger sample, GSES should serve several purposes for the sleep medicine specialist. It will identify if effort is a relevant concern in a given insomnia case. It will also provide a useful baseline cognitive screen for dysfunctional beliefs, which may then be targeted as part of cognitive restructuring. And it will denote potential patient suitability for single-component paradoxical intention therapy. This therapy as noted improves sleep by reducing effort (Broomfield and Espie, 2003), so insomnia patients scoring high on GSES may be good responders. Research is needed on this.

Following larger scale validation, general practitioners could also use GSES to identify a core symptom of psychophysiological insomnia in sleep-disturbed individuals. The scale is short, easily administered and appears to show, at least on this initial sample reasonable sensitivity, specificity and discriminant validity. Whereas we would predict patients with other types of sleep disturbance, e.g. sleep apnoea or circadian disorders would not score highly. Research examining GSES with other sleep disorder groups is needed.

Finally, the validation of GSES will allow research into how effort perpetuates insomnia. GSES appears to provide the means to measure effort reliably, so comparison between studies is possible. Further work on putative mechanisms underpinning paradoxical intention therapy is also now possible. The evidence base for paradoxical intention therapy as a single-component therapy is established (Chesson *et al.*, 1999), but our understanding of *how* it promotes sleep change is poor. The answer will come from research using GSES to clarify the role of paradoxical intention therapy in unwinding inhibitory sleep effort.

Several limitations of this study merit consideration. First, sample size was relatively small for a validation study, and insomnia participants were obtained from a large treatment trial and from a university-based population. Recruited participants were neither asked to record sleep data to confirm diagnosis of primary insomnia nor was polysomnography (PSG) employed to rule out occult primary sleep disorders. Diagnosis was made on the basis of a sleep complaint present 4+ nights per week for 6 months, and a PSQI score >6. No inter-rater check on diagnosis was made, no data on insomnia severity and type (initial versus maintenance) were collected, and clinical interviews were not employed for screening. Future GSES research will benefit

from such detail and from additional screening methods, e.g. structured interview methodology, single-night PSG (Edinger *et al*., 2004) and inter-rater reliability data. Test–retest data should also be collected.

Furthermore, to test the validity of GSES, correlation was only made to other self-report scales (HADS, DBAS). Objective sleep assessments are needed to confirm external scale validity. This could be performed on a smaller sample. Related to this, HADS, DBAS and medication data were only available from the majority of insomnia patients and data on specific hypnotic medication type were not recorded.

Also, the insomnia and good sleeper samples differed significantly on age. Unmatched samples are not unusual in insomnia scale research however (e.g. Buysse *et al*., 1989; Fichten *et al*., 1998), probably because of epidemiology factors (Mellinger *et al*., 1985). As sleep effort is likely to play a role in insomnia maintenance *throughout* the lifespan, research with age-matched younger and with age-matched older good and poor sleepers will be desirable. Crucially, while in our sample age and GSES were significantly associated, ANCOVA demonstrated adequate GSES discriminant validity with age partialled out.

Importantly, the *majority* of insomnia patients (83%) were physician-referred. So the cohort is a clinical insomnia rather than university-based population. Notably also, good sleepers were not merely defined by absence of sleep complaint. They also described themselves as good sleepers, and reported their sleep as good or very good both typically, and in the last month. This exceeds research diagnostic guidelines (Edinger *et al*., 2004), and is a strength rarely seen in studies that use 'good' sleepers to compare.

Finally, GSES item derivation relied on an empirically *untested* model of effort. It was, however, our intention to do this. The approach of setting testable hypotheses extracted from clinical experience in order to inform theory is defendable, and has assisted our understanding and treatment of a range of psychological disorders in recent years (Salkovskis, 2002). Indeed, this should be employed more to study insomnia (Tang and Harvey, 2003). The working model we present requires empirical clarification using experimental methods, and GSES will assist with this. Carefully designed single casework is needed for tracking insomnia patients who fit the model. Related research questions include whether it is possible to induce sleep effort experimentally in good sleepers? And what is the impact of experimentally induced sleep effort on insomnia patients? This research will extend our understanding of effort in insomnia, and provide important groundwork for further study of paradoxical intention therapy and putative mechanisms.

To summarize, this validation study suggests that GSES shows good psychometric properties. It is a useful and novel measure of clinical assessment and change, which should screen out psychophysiological insomnia in sleep-disturbed individuals and aid identification of insomnia patients suited to psychological therapies.

REFERENCES

American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders*, 4th edn. APA, Washington, DC, 1994.

Ansfield, M. E., Wegner, D. M. and Bowser, R. Ironic effects of sleep urgency. *Behav. Res. Ther*., 1996, 34: 523–531.

Backhaus, J., Junghanns, K., Brooks, A., Reimann, D. and Hohagen, F. Test–retest reliability and validity of the Pittsburgh Sleep Quality Index in primary insomnia. *J. Psychosom. Res*., 2002, 53: 737–740.

Bossuyt, P. M., Reitsma, J. B., Bruns, D. E., Gatsonis, C. A., Glasziou, P., Irwig, L. M., Lijmer, J. G., Moher, D., Rennie, D., de Vet, H. C. Towards complete and accurate reporting of studies of diagnostic accuracy: the STARD initiative. *Br. Med. J*., 2003, 326: 41–44.

Broomfield, N. M. and Espie, C. A. Initial insomnia and paradoxical intention: an experimental investigation of putative mechanisms using subjective and actigraphic measurement of sleep. *Behav. Cogn. Psychother*., 2003, 31: 313–324.

Broomfield, N. M., Gumley, A. I. and Espie, C. A. Candidate cognitive processes in psychophysiologic insomnia. *J. Cogn. Psychother*., 2005, 19: 5–17.

Buysse, D. J., Reynolds, C. F., Monk, T. H., Berman, S. R. and Kupfer, D. J. The Pittsburgh Sleep Quality Index: a new instrument for psychiatric practice and research. *Psychiatry Res*., 1989, 28: 193–213.

Chesson, A. L., Anderson, W. M., Littner, M., Davila, D. and Hartse, K. Practice parameters for the non-pharmacologic treatment of chronic insomnia. *Sleep*, 1999, 22: 1128–1133.

Edinger, J., Bonnet, M. H., Bootzin, R. R., Doghramji, K., Dorsey, C. M., Espie, C. A., Jamieson, A. O., McCall, W. V., Morin, C. M. and Stepanski, E. J. *Derivation of Research Diagnostic Criteria for Insomnia*: Report of the American Academy of Sleep Medicine Work Group. 2004.

Espie, C. A., Broomfield, N. M., Kelly, H., MacMahon, K., Mckinstry, B., Douglas, N. J., Wilson, P., Englemen, H. and Walker, A. Randomised intention to treat trial of nurse-administered Cognitive Behaviour Therapy for insomnia in general practice. *Sleep*, 2004, 27: A269.

Fichten, C., Libman, E., Creti, L., Amsel, R., Tagalakis, V. and Brender, W. Thoughts during awake times in older good and poor sleepers: The self statement test: 60+. *Cogn. Ther. Res*., 1998, 22: 1–20.

Harvey, A. G. A cognitive model of insomnia. *Behav. Res. Ther*., 2002, 40: 869–893.

American Sleep Disorders Association. *International Classification of Sleep Disorders*. Rochester, MN: American Sleep Disorders Association, 1990.

American Sleep Disorders Association. *International Classification of Sleep Disorders – Revised*. Rochester, MN: American Sleep Disorders Association, 1997.

Kohn, L. and Espie, C. A. Sensitivity and specificity of the insomnia experience: a comparative study of psychophysiologic insomnia, insomnia associated with mental disorder and good sleep. *Sleep*, 2005, 28: 104–112.

Mellinger, G. D., Balter, M. B. and Uhlenhuth, E. H. Insomnia and its treatment: prevalence and correlates. *Arch. Gen. Psychol*., 1985, 42: 225–232.

Morin, C. M., Stone, J., Trinkle, D., Mercer, J. and Remsberg, S. Dysfunctional beliefs and attitudes about sleep among older adults with and without insomnia complaints. *Psychol. Aging*, 1993, 8: 463–467.

Morin, C. M., Hauri, P. J., Espie, C., Spielman, A., Buysse, D. J. and Bootzin, R. Nonpharmacologic treatment of chronic insomnia. *Sleep*, 1999, 22: 1–25.

Nunnaly, J. C. and Bernstein, I. H. *Psychometric Theory*, 3rd edn. McGraw Hill Series in Psychology, London, 1994.

Salkovskis, P. M. Empirically grounded clinical interventions: cognitive behaviour therapy progresses through a multi-dimensional approach to clinical science. *Behav. Cogn. Psychother.*, 2002, 30: 3–9.

Tang, N. K. Y. and Harvey, A. G. Cognitive behaviour therapy for primary insomnia: Can we rest yet? *Sleep Med. Rev.*, 2003, 7: 237–262.

Wegner, D. M. Ironic processes of mental control. *Psychol. Rev.*, 1994, 101: 34–52.

Zigmond, A. S. and Snaith, R. P. The hospital anxiety and depression scale. *Acta Psychiatr. Scand.*, 1983, 67: 361–370.

Recommendations for a Standard Research Assessment of Insomnia

Daniel J. Buysse, MD[1]; Sonia Ancoli-Israel, PhD[2]; Jack D. Edinger, PhD[3]; Kenneth L. Lichstein, PhD[4]; Charles M. Morin, PhD[5]

[1]Department of Psychiatry, University of Pittsburgh School of Medicine, Pittsburgh, PA; [2]Department of Psychiatry, University of California San Diego and Veterans Affairs San Diego Healthcare System, San Diego, CA; [3]Department of Psychology, Veteran's Administration Hospital and Duke University, Durham, NC; [4]Department of Psychology, University of Alabama, Tuscaloosa, AL; [5]Department of Psychology, Université Laval, Québec, QC

Study Objectives: To present expert consensus recommendations for a standard set of research assessments in insomnia, reporting standards for these assessments, and recommendations for future research.
Participants: N/A.
Interventions: N/A.
Methods and Results: An expert panel of 25 researchers reviewed the available literature on insomnia research assessments. Preliminary recommendations were reviewed and discussed at a meeting on March 10-11, 2005. These recommendations were further refined during writing of the current paper. The resulting key recommendations for standard research assessment of insomnia disorders include definitions/diagnosis of insomnia and comorbid conditions; measures of sleep and insomnia, including qualitative insomnia measures, diary, polysomnography, and actigraphy; and measures of the waking correlates and consequences of insomnia disorders, such as fatigue, sleepiness, mood, performance, and quality of life.
Conclusions: Adoption of a standard research assessment of insomnia disorders will facilitate comparisons among different studies and advance the state of knowledge. These recommendations are not intended to be static but must be periodically revised to accommodate further developments and evidence in the field.
Keywords: Insomnia, diagnosis, polysomnography, sleep diary, actigraphy, questionnaires
Citation: Buysse DJ; Ancoli-Israel S; Edinger JD et al. Recommendations for a standard research assessment of insomnia. *SLEEP* 2006;29(9):1155-1173.

INTRODUCTION

INSOMNIA IS THE MOST COMMON SLEEP COMPLAINT IN THE GENERAL POPULATION. RECENT RESEARCH HAS ADDRESSED A WIDE RANGE OF ISSUES related to this condition, including epidemiology, consequences, pathophysiology, and treatment. However, understanding the results of the research and translating these results into clinical practice have been hindered by the absence of standardized definitions, assessments, and reporting standards. For instance, a recent review of epidemiologic studies showed that the reported prevalence of insomnia in the general population can range from 2% to 48%, depending on the definition of insomnia.[1] Studies examining the clinical and physiologic characteristics of insomnia have used definitions ranging from the very broad (e.g., self-defined "good vs poor sleepers"[2]), to the very narrow (e.g., individuals with "sleep-state misperception," which can only be defined with polysomnography[3;4]).

Reviews of treatment studies demonstrate similar problems. For instance, Nowell and colleagues identified 198 studies comparing drug therapy with placebo in insomnia, but only 22 could be included in a final meta-analysis.[5] The authors needed to make several inferences regarding the diagnosis of primary insomnia because most studies failed to specify insomnia duration, clinically significant distress, or the means by which psychiatric or medical causes of insomnia were excluded. Holbrook and colleagues[6] identified 89 double-blind trials for their meta-analysis but excluded half of these, noting, "the methodologic quality of the studies was not uniform… The diversity in outcomes used and the methods of summarizing… prevented the pooling of many trials." Likewise, Smith and colleagues initially identified 194 treatment studies of primary insomnia but included only 21 in their final meta-analysis. Many primary sources were excluded because means, standard deviations (SD), and test statistics were not reported. A recent review of behavioral and psychological treatment studies for insomnia[7] also identified a wide range of diagnostic methods, specific diagnoses, and specific outcomes, with 16 of 53 eligible studies excluded from the review for reasons including the failure to document an insomnia diagnosis. In summary, consistent diagnostic, baseline, and outcome measures have not been used in published insomnia studies. Inconsistency of reporting methods and outcome data has further limited the ability to compare findings across studies.

This paper reports the findings of an expert consensus process to develop recommendations for standard assessments and reporting standards in insomnia research studies. This process included a conference held on March 10-11, 2005, in Pittsburgh, Penn, with 25 experts in insomnia research in attendance. The specific aims of this process and the accompanying conference, reflected

Disclosure Statement
This manuscript is based on a conference supported by unrestricted grants from Pfizer, Sanofi-Aventis, Sepracor, and Takeda. Dr. Buysse has served as a consultant for Actelion, Cephalon, Eli Lilly, GlaxoSmithKline, Merck, Neurocrine, Neurogen, Pfizer, Respironics, Sanofi-Aventis, Servier, Sepracor, and Takeda. Dr. Ancoli-Israel has served as a paid consultant, scientific advisory board member and/or speaker for Acadia, Cephalon, Inc., King Pharmaceuticals, Inc., Merck, Neurocrine Biosciences, Inc., Neurogen, Inc., Pfizer, Inc., Sanofi-Aventis, Sepracor, Inc., Somaxen, and Takeda Pharmaceuticals North America, Inc. Dr. Edinger has received research support from Respironics; and has participated in speaking engagements supported by Axis Healthcare, Sepracor, and Fission Communications. Dr. Lichstein has received research support from Respironics/Mini Mitter. Dr. Morin has received research support from Sanofi-Aventis and Organon; and is a consultant and/or a member of the advisory board for Sanofi-Aventis, Pfizer, Neurocrine, Takeda, Shire-Biodem, and Sepracor.

Submitted for publication December 2, 2005
Accepted for publication April 1, 2006
Address correspondence to: Dr. Daniel Buysse, 3811 O'Hara St., Pittsburgh, PA 15213; Tel: (412) 246-6413; Fax (412) 246-5300; E-mail: buyssedj@upmc.edu

in this paper, were

1. To systematically review the relative merits and disadvantages of various insomnia assessment tools in 5 key domains: insomnia diagnosis; medical, psychiatric, and sleep disorder comorbidities; quantitative sleep measurements; qualitative sleep and insomnia measurements; and daytime symptom and function measures.
2. To develop consensus recommendations regarding a standard research assessment of insomnia to be used in both assessment and treatment-outcome studies.
3. To develop consensus recommendations regarding reporting standards for each of the assessments.
4. To make recommendations regarding assessment areas and instruments that require further development in order to meet the needs of future insomnia research.

METHODS

The concept for the conference and this paper grew out of discussions at the University of Pittsburgh Mental Health Intervention Research Center (MH30915). An Organizing Committee, consisting of the authors of this paper, was charged with further developing goals and methods. The Organizing Committee invited a group of 20 additional insomnia experts to participate, based on their research and publications in the field. These individuals were chosen to provide a broad representation of different types of insomnia research, with the recognition that many other qualified investigators could also have been selected. Participants were assigned to 1 of 5 work groups corresponding to the 5 content areas being addressed: Insomnia diagnosis, comorbidities, quantitative sleep, qualitative sleep and insomnia symptoms, and daytime function and consequences. One member of the Organizing Committee served as the leader of each work group, which was responsible for reviewing the literature in its specific area to identify the range of tools and methods used in published studies. This process was aided by recent review articles[8-13] and review of primary references. The goal of this process was to develop an evidence-based qualitative review of available methods, focusing on the strengths and weaknesses of various approaches. The work groups did not conduct quantitative literature reviews, since the focus was on identifying optimal methods rather than the most commonly used methods or a description of study results. In several of the work groups, individuals were assigned more specific areas for review. The work groups conferred by telephone and e-mail to discuss their methods and preliminary findings and developed a draft summary of their findings and recommendations prior to the meeting.

At the conference, the work groups convened briefly to review their findings and discuss remaining areas of disagreement. All participants then met in plenary session, where the findings and recommendations of each work group were presented and discussed. Additional discussion focused on development of consensus regarding the recommendations for general assessment domains and specific assessments within these domains. During the course of work-group and plenary discussions, it also became clear that recommendations were needed to accommodate the different goals and resources in different types of research studies. For instance, epidemiologic studies often need to use relatively brief assessments in large numbers of individuals, whereas treatment-efficacy studies include detailed assessments in relatively smaller numbers of individuals.

The members of the Organizing Committee used the work groups' written summaries, the results of discussions, and the recommendations emanating from the conference in the development of the final paper. All conference participants had the opportunity to comment on the initial draft, which was then finalized by the Organizing Committee.

As a result of all the discussions, the 5 original domains were collapsed into the 3 broader domains presented in this paper: diagnosis of insomnia and comorbid conditions, sleep measurements, and waking correlates and consequences of insomnia. The goals and rationale for each domain are presented briefly, followed by descriptions of specific recommended measures and tools. Recommendations for reporting standards for specific assessments are also discussed within each domain.

Table 1 summarizes the recommended standard assessment of insomnia for 4 types of research studies: (1) Epidemiologic studies; (2) Mechanism and evaluation studies (i.e., studies comparing the clinical or physiologic characteristics of participants with insomnia to a reference group); (3) Efficacy studies (treatment studies focusing on relatively small, well-defined samples) and; (4) Effectiveness studies (treatment studies focusing on large community samples in routine practice settings). Although we have described the recommended assessments for these study types as though they were completely distinct, in actual practice there may be considerable overlap in study types. In such cases, it would be reasonable to include a hybrid of the recommended assessments. Each assessment method within each study type is further categorized as "Essential" or "Recommended." The descriptor "Essential" is used to indicate assessments that should be included in every study of the type noted. "Recommended" assessments are those that should be included whenever feasible. Note that the designation of a measure as "Essential" or "Recommended" varies with the particular type of research study, as indicated in Table 1. The text contains more general language than Table 1 regarding these levels of endorsement.

The recommendations in this paper are not intended to indicate that other measures should not be used nor that other measures cannot contribute useful information to the study of insomnia. Indeed, other measures may be very appropriate for specific insomnia studies, and investigators should not hesitate to use them. The goal of this paper is to present only the minimum set of assessments needed to facilitate future comparisons across studies. Toward this end, the shortest set that provides coverage in key areas was purposely selected. Thus, Table 1 is not intended to be a comprehensive summary of the available assessments within each domain.

DESCRIPTION OF ASSESSMENT DOMAINS AND RECOMMENDATIONS

1. DEFINITION/DIAGNOSIS OF INSOMNIA AND COMORBID CONDITIONS

a. Background and Rationale

Insomnia Definitions and Diagnosis

In both research studies and clinical practice, the term insomnia has been used narrowly to denote a set of sleep-specific symptoms and more broadly to refer to a heterogeneous group of sleep disorders. When defined narrowly, the term insomnia implies iso-

lated sleep-related complaints, such as a difficulty falling asleep, difficulty staying asleep, early awakening, or unrefreshing/nonrestorative sleep in an individual who has adequate circumstances and opportunity for sleep. When more broadly defined, the term insomnia connotes a sleep-wake disorder wherein sleep-specific insomnia symptoms are associated with significant waking distress or impairment and, depending on the specific insomnia disorder, other specific symptoms. To avoid confusion arising from these discrepant definitions, throughout the remainder of this article we use the term insomnia symptom to refer to the more-narrow sleep-specific insomnia definition and the term insomnia disorder or simply insomnia to denote a broader syndrome having both sleep and waking symptoms.

Even a cursory review of the relevant literature shows that a substantial proportion of studies devoted to the disorder of insomnia have included study samples selected solely on the basis of their insomnia symptoms. In some cases, researchers have recruited and enrolled samples with specific types of insomnia symptoms such as sleep-onset or sleep-maintenance difficulties. However, experience shows that use of insomnia symptoms for sample selection or characterization provides little specificity inasmuch as individuals with such sleep complaints comprise a large and very heterogeneous population. Moreover, insomnia symptoms, considered in isolation, confer limited morbidity and, thus, are of questionable clinical significance.[1] Therefore, reliance solely on insomnia symptoms for sample selection appears to be a suboptimal research practice.

Arguably, a more defensible research practice is that of defining insomnia as a disorder with impairment of sleep and waking functions. Typically, such definitions have included complaints of insomnia symptoms coupled with waking symptoms, such as functional impairments,[14;15] sleep-related distress,[14;15] or general sleep dissatisfaction.[16] Insomnia definitions that combine sleep-specific complaints with these associated daytime symptoms appear to represent clinically significant insomnia disorders that are, in fact, associated with elevated risks for morbidity.[1] However, individuals meeting such criteria are still heterogeneous with respect to actual or presumed etiology. Use of diagnostic criteria for specific insomnia-disorder subtypes from one of the currently available sleep-disorders nosologies[17-20] offers greater specificity and homogeneity in case ascertainment and research-sample selection. Although there is limited information concerning the reliability or validity of most insomnia diagnoses listed in these nosologies,[4;21;22] application of specific insomnia diagnostic criteria clearly benefits research practice, particularly when research questions pertain to specific insomnia subtypes.

Recently both research diagnostic criteria (RDC) for insomnia[4] and quantitative insomnia diagnostic criteria[23] have been proposed to improve upon previous definitions used in insomnia research. The RDC were developed by a specially commissioned work group of the American Academy of Sleep Medicine and consist of criteria sets for identification of individuals who meet general criteria for an insomnia disorder, as well as more exacting criteria sets for 9 specific insomnia diagnostic subtypes. In addition, criteria are provided for the identification of normal sleepers who may be chosen as controls for insomnia studies. These RDC present the opportunity to standardize research practice in regard to the selection or identification of research enrollees. Furthermore, these criteria have the advantage of being totally concordant with insomnia disorders listed in the second edition of the International Classification of Sleep Disorders (ICSD-2)[17] and the Clinical Modification of the International Classification of Diseases, 10th edition (ICD-10-CM). Although their reliability and validity await testing, the RDC have been developed specifically to standardize case ascertainment, and, given this purpose, their use in future insomnia studies seems preferable.

Although there is currently no consensus regarding the use of quantitative diagnostic criteria for insomnia, their use in selecting individuals who otherwise meet conditions for insomnia may ensure more homogeneous and seriously affected samples and may avoid underestimation of treatment effects due to ceiling or floor effects.[23] Based upon modal practice in published clinical trials and maximization of sensitivity and specificity, the following criteria have been suggested: sleep-onset latency (SOL) or wakefulness after sleep onset (WASO) of more than 30 minutes, frequency of at least 3 times a week, and duration of at least 6 months.[23] Applying quantitative criteria carries the potential liability of limiting the generalizability of research findings to clinical practice, where such criteria are not generally employed. Moreover, quantitative criteria do not address unrefreshing or nonrestorative sleep, one of the cardinal insomnia symptoms. The quantitative criteria described above may also reflect selection or reporting biases in the published literature. As in other domains of health science, the validity of quantitative diagnostic cutoffs is ultimately judged by how well they actually discriminate those with and without clinically significant insomnia and by their associated morbidity risk. Such data for insomnia are extremely limited, but future research studies could determine optimal quantitative sleep thresholds or other indicators of insomnia severity and their association with graded levels of morbidity. Thus, with additional supportive studies, use of quantitative criteria combined with RDC for insomnia sample selection or characterization could become optimal research practice.

Previous studies have provided various methods for screening prospective study candidates to determine if they meet criteria for insomnia or a specific insomnia subtype. Included among these methods are both brief and more lengthy questionnaires,[14;24-27] the common unstructured clinical interview,[28;29] semistructured insomnia-focused interviews,[27;30;31] and structured sleep disorders interviews,[32;33]as well as subjective and objective sleep-monitoring methods. The unstructured clinical interview that includes a thorough sleep history has been used widely for insomnia assessment and sample selection in previous studies. The lack of standardized questions and procedures for this method reduces its interrater reliability. However, when used by interviewers who are experienced with sleep disorders and insomnia diagnoses, the unstructured interview seems to be a pragmatic procedure for study-sample selection, particularly if combined with a patient-administered sleep-history questionnaire. Semistructured interviews have been developed by selected research groups to standardize research diagnostic practice. These instruments have the advantage of ensuring a more consistent and comprehensive insomnia assessment across interviewers, but data regarding the reliability and validity of these instruments is currently lacking. Furthermore, use of such instruments has, to date, been researcher or lab specific, so we currently lack a broad "experience base" upon which to judge any of these instruments. Perhaps most promising for insomnia diagnosis are the structured interviews developed by Schramm et al and Ohayon.[32;33] Both of these standardize the interviewing process and have data supporting their

reliability. Unfortunately, the interview by Schramm et al results in DSM-III-R insomnia diagnoses rather than the insomnia diagnoses listed in the current DSM-IV-TR. In contrast, the computerized Ohayon instrument known as the Sleep-EVAL interview results in DSM-IV and ICSD-90 diagnoses but is not generally available to the insomnia research community. Thus, neither of these specific instruments can be recommended for ascertaining RDC for insomnia disorder or for specific insomnia subtypes.

Evaluation of Comorbid Sleep Disorders

Other sleep disorders can also be present in individuals with symptoms of insomnia. Extended discussion of the differential diagnosis of insomnia from other sleep disorders is available elsewhere.[34] For example, even carefully screened research participants meeting RDC for insomnia may have sleep-disordered breathing on polysomnographic (PSG) evaluation.[35] Insomnia symptoms can also occur in conjunction with other sleep disorders such as sleep apnea, restless legs syndrome, periodic limb movement disorder, and circadian rhythm sleep disorders. However, the presence of other sleep disorders in insomnia samples may confound evaluation and intervention data and affect conclusions. Thus, it is prudent to include other sleep disorders in the differential diagnosis and to identify subjects with comorbid sleep disorders. The evaluation of other sleep disorders, exclusionary diagnoses, and sleep-disorder comorbidities should all be described in research reports.

Methods for evaluating other sleep disorders are similar to those for diagnosing insomnia and include clinical interviews, structured sleep interviews, and questionnaires. A number of questionnaires have been specifically developed to identify sleep apnea, focusing on common clinical characteristics such as high body mass index, increasing age, male sex, excessive daytime sleepiness, and loud snoring.[36] However, no current questionnaire specifically discriminates insomnia from sleep apnea, provides clear cutoff scores, or has strong sensitivity and specificity. These questionnaires, like clinical interviews, suffer from patients' difficulty assessing their own snoring and daytime sleepiness and from the wide distribution of apnea-related characteristics among the nonapneic population. Given these difficulties, a reasonable approach is to assess the above characteristics by interview and, if most are present, to assume a high probability of sleep apnea. Additional history from a knowledgeable sleep informant and additional data on sleepiness from measures such as the Epworth Sleepiness Scale (ESS) (e.g., a score >10-12) may also be useful. The clinical significance of periodic limb movements has been questioned,[37;38] and this condition is very difficult to assess by clinical history alone. On the other hand, restless legs syndrome, a condition frequently seen concurrent with periodic limb movements of sleep, can be assessed reliably by history and questionnaire.[39;40] A high frequency of brief awakenings (e.g., >10) during the night should arouse suspicion of periodic limb movement disorder or sleep apnea, particularly if it is accompanied by excessive daytime sleepiness. Ultimately, PSG assessment may be deemed necessary to detect apnea and PLMS for certain forms of insomnia research.

Evaluation of Comorbid Psychiatric and Medical Disorders and Medication or Substance Use. The co-occurrence of insomnia and psychiatric symptoms and disorders is common[10] and may be associated with clinically significant psychiatric disability.[41-43] The high rate of psychiatric comorbidity with insomnia is not a special case; the National Comorbidity Survey reported that every psychiatric disorder is comorbid with every other disorder.[44] To give one example, generalized anxiety disorder is comorbid with depression in 80% of cases. However, as pointed out in the recent National Institutes of Health State-of-the-Science Conference on insomnia,[45] the mechanisms and nature of the relationships between insomnia and comorbid psychiatric conditions are poorly understood and may vary between individuals and within individuals over time.[46;47] At some time points, insomnia and comorbid psychiatric conditions may be parallel and functionally independent, whereas, at other times, one may perpetuate or exacerbate the other. The RDC for Insomnia Due to a Mental Disorder require that the onset of the insomnia coincide with the onset of the mental disorders and that the temporal course of the insomnia and mental disorder coincide. In reality, the accuracy and reliability of such temporal information remains very much in doubt. For this reason, the term comorbid insomnia, supported by the National Institutes of Health State-of-the-Science Conference, may be more accurate, even if less precise. Given the complex relationships between insomnia and psychiatric disorders, it is prudent to systematically evaluate psychiatric comorbidities, to assess the nature of the relationship between the insomnia and psychiatric disorder, and to describe comorbidities in research reports. Methods for evaluating psychiatric disorders include self-report questionnaires, clinical interviews, and structured psychiatric interviews.

These issues apply equally to medical comorbidity. Evaluating medical disorders serves 3 functions: to identify comorbid medical disorders; to evaluate medical or physical characteristics associated with increased risk of other sleep disorders (e.g., sleep apnea); and to determine medical eligibility criteria, which may relate to the safety of conducting certain insomnia interventions. Common medical conditions comorbid with insomnia include cardiovascular, pulmonary, neuromuscular, endocrine, gastrointestinal, pulmonary, and neurologic disorders.[48] Cancer and sex-specific conditions (e.g., menstruation, menopause, and pregnancy) may also influence insomnia. When insomnia is comorbid with a medical condition, it is not necessarily caused by (secondary to, due to) that medical condition. As in the case of psychiatric disorders, comorbid insomnia may actually be a more accurate term. As is the case for comorbid psychiatric disorders, insomnia may be completely or partially dependent on a medical condition, may be independent, or may exacerbate a medical condition, and the nature of the relationship may change over time. The insomnia RDC for Insomnia due to a Medical Condition require that the onset of the insomnia coincide with the onset of the associated medical disorder and that the temporal course of the insomnia and medical disorder coincide. It is prudent to evaluate medical comorbidities, to assess the nature of the relationship, and to describe such comorbidities in research. General methods for evaluating medical disorders include self-report questionnaires, clinical interview, physical examination, and laboratory screening tests.[49]

A wide variety of medications and substances may cause or contribute to insomnia, daytime sleepiness, or both insomnia and daytime sleepiness.[50] These include prescription medications, over-the-counter medications, herbal and "natural" substances, and nonmedicinal substances such as alcohol and caffeine. An accurate current list of medications and substance use serves two

Table 1—Recommended Research Measures for Insomnia

Specific area	Recommended Measures	Reporting Standards	Epidemiology Studies	Mechanism/ Evaluation Studies	Treatment Efficacy Studies	Treatment Effectiveness Studies
Diagnosis						
Insomnia and other sleep disorders	Clinical history/questionnaire for sleep disorders using International Classification of Sleep Disorders 2nd ed (ICSD-2) criteria	Indication that clinical history was conducted; qualifications of interviewer; specific methods used (e.g., interview, symptom questionnaires); exclusionary diagnoses; # participants judged to have another ICSD-2 sleep disorder; insomnia age of onset, duration, frequency, specific complaints	R	E	E	R
	Clinical history/questionnaire for Research Diagnostic Criteria for Insomnia (RDC-I)	Indication whether RDC for general insomnia disorder or specific insomnia subtypes were used; description of specific assessment methods; # participants with each specific diagnosis; insomnia age of onset, duration, frequency, specific complaints	E	E	E	E
Psychiatric disorders	Structured interview (SCID I) OR Psychiatric history/ questionnaire	For SCID: specific modules used; qualifications of interviewer; exclusionary diagnoses; # of subjects with specific psychiatric diagnoses and with RDC-defined "Insomnia due to a Mental Disorder." For clinical psychiatric history or self-report questionnaires: specific methods and tools used; other information for diagnoses described above	E	E	E	E
Medical disorders	Medical history/ questionnaire	Specific methods (interview, physical examination, questionnaires); qualifications of evaluator; mean and SD of BMI for sample; specific exclusionary diagnoses; # subjects with specific comorbid medical conditions and with RDC-defined "Insomnia due to a Medical Condition."	E	E	E	E
Current medications and substances	List of medications (history, participant-generated list)	Specific methods; qualifications of evaluator; exclusionary medications/substances; list of medications used by subjects; # participants using specific medications; # of subjects using and amount of caffeine, alcohol, and tobacco (mean/SD or median/range).	E	E	E	E
Sleep and insomnia symptoms						
Global sleep and insomnia symptoms	PSQI	Global mean, SD; subscale means, SD	R	R	E	E
	Insomnia Severity Index	Total score mean, SD; % of participants in each severity range	R	R	E	E
Daily self-report of sleep	Sleep diary	Duration, format, and information assessed in the diary; thresholds or quantitative criteria for insomnia or other sleep disorder diagnoses; mean/SD for each dependent measure (bed time, final wake time, arising time; SOL, NWAK, WASO, TST, SE, SQ; timing and duration of naps or daytime sleep episodes); day-to-day variability of dependent measures	R	E	E	R
Objective Sleep	Screening polysomnography	Description of PSG protocol; AHI and PLMAI levels used to establish study eligibility or diagnoses; other PSG criteria used for study eligibility;	R	E	E	R
	PSG as dependent measure	Description of PSG protocol; total recording time (and how determined); mean/SD for AHI, PLMAI, SOL, NWAK, WASO, TST, SE.	NR	R	E[1]	NR
Rest-activity pattern	Actigraphy	Description of device and scoring software; clinical protocol; thresholds or quantitative criteria used to establish or confirm insomnia or other sleep disorder diagnoses; mean/SD for sleep onset and offset times, SOL, NWAK, WASO, TST, and SE; within-subject variability	R[2]	R	E[1]	NR
Waking correlates and consequences of insomnia						
Fatigue	Multidimensional Fatigue Inventory OR Fatigue Severity Scale	Mean/SD for total scores and subscales	R	E	E	E
Mood	Inventory of Depressive Symptomatology OR Beck Depression Inventory II	Mean/SD for total scores and total scores less sleep items	R	E	E	E
	State-Trait Anxiety Inventory	Mean/SD for Trait portion	R	E	E	E
Quality of Life and Function	SF-36	Mean/SD for total and subscale scores	R	E	E	E

E refers to a measure that is considered "essential," i.e., should be included in all studies; R, Measure is "recommended," i.e., should be included whenever feasible; NR, Not recommended for routine use; SD, standard deviation; BMI, body mass index; AHI, apnea-hypopnea index; PLMAI, the index of periodic limb movements index associated with arousals; PSG, polysomnography. Remaining abbreviations are defined in Table 2.

[1]At least 1 assessment, either polysomnography or actigraphy, is considered essential

[2]Recommended for at least a subset of participants

major functions: to identify possible causes for insomnia (e.g., excessive caffeine and alcohol use) and to determine eligibility criteria (e.g., exclusion of participants currently taking hypnotics). Therefore, research evaluations of insomnia must include a review of current medication and substance use and consideration of their positive or negative impact on sleep. When feasible, or when necessary for specific study samples, a urine drug screen is also desirable prior to the start of the study and during any treatment. Research reports should also include a description of medication screening and entry criteria. Methods for evaluating medication and substance use include clinical interviews or history and self-report checklists.

Use of Sleep Diaries, PSG, and Actigraphy for Screening. Sleep diaries, PSG, and actigraphy have been used for a variety of purposes in insomnia research studies, including screening, diagnosis, and differential diagnosis. For simplicity, since these methods may be used as both screening and dependent outcome measures, they are discussed in Section 2 below.

b. Recommendations for Measures and Reporting Standards

Diagnosis of Insomnia and Other Sleep Disorders

Recommended/essential measures: (1) Sleep history (interview, questionnaires) and (2) RDC for Insomnia. In the absence of a validated structured interview for insomnia and other sleep-disorder diagnoses, a clinical sleep history conducted by a knowledgeable clinician constitutes the basis for establishing insomnia and other sleep-disorder diagnoses. This history may be aided by locally developed questionnaires, semistructured interviews, or structured interviews (e.g., [28;30;31]). The psychometric properties of most of these instruments have not been established, but the high degree of overlap between them indicates that different labs have found the same common set of information to be useful. Information covered in the history should include the specific nocturnal and daytime symptoms of insomnia; daytime correlates and consequences of insomnia; and the duration, frequency, and severity of the symptoms. Symptoms of other sleep disorders must also be assessed, including, at a minimum, the following: obstructive sleep apnea syndrome, restless legs syndrome, periodic limb movement disorder, and circadian rhythm sleep disorders. The recently published RDC for Insomnia[4] include general criteria for an insomnia disorder, as well as more specific criteria for 9 insomnia-disorder subtypes. The RDC were designed specifically to standardize case ascertainment in insomnia research. Furthermore, these criteria are concordant with insomnia disorders listed in the ICSD-2[17] and the future ICD-10-CM. Because the RDC have only recently become available, their reliability and validity await testing. The application of RDC for insomnia may be aided by the use of checklists and/or locally developed semistructured or structured interviews.

Reporting standards. For the clinical sleep history, research reports should document that a clinical history was conducted, the type of clinician who conducted the evaluation and his or her training (e.g., psychologist, psychiatrist; graduate student, licensed, subspecialty licensed such as in sleep disorders), the specific methods used (e.g., interview, symptom questionnaires), and the number of potential or accepted research participants who were judged to have another sleep disorder according to ICSD-2 criteria. Reporting standards for RDC include a statement that the criteria were used, whether RDC for general insomnia disorder were used alone or whether RDC for the specific insomnia disorders (e.g., psychophysiological insomnia, paradoxical insomnia, etc.) were also used, a description of the specific methods used (e.g., clinical or structured interviews, phone interviews, questionnaires) provided in sufficient detail to allow replication of procedures by other investigators, and the number of participants with each specific diagnosis. Whether a clinical history or RDC are used, research reports should also provide data regarding the mean, standard deviation, and/or range for insomnia characteristics, including age of onset, duration of insomnia episode (see above), frequency of complaint, and type of symptoms (sleep onset, sleep maintenance, early morning awakening).

Diagnosis of Psychiatric Disorders

Essential measures: (1) Structured Clinical Interview for DSM-IV (SCID), Axis I[51] or (2) psychiatric history (interview, questionnaires). The SCID incorporates DSM-IV diagnostic criteria in a structured standardized interview that yields diagnoses for Axis I disorders. It is the accepted standard for evaluation of psychiatric disorders, and its use is encouraged whenever possible. Most reliability data for the SCID derive from an earlier version designed for DSM-III-R criteria.[52] SCID-I diagnoses generally have κ values (an estimate of interrater agreement) in the 0.6 range. For diagnoses relevant to insomnia, κ values in the 0.8 range are typical for major depression and generalized anxiety disorder. The relationship between diagnosed psychiatric disorders and insomnia should be carefully explored in order to distinguish Insomnia due to a Mental Disorder from other types of insomnia-psychiatric comorbidity. Whenever possible, the SCID is the preferred method for ascertaining psychiatric diagnosis. However, in some cases, the resources necessary to conduct SCID evaluations may not be feasible. In those cases, a clinical history of psychiatric disorders should be conducted, covering all major classes of psychiatric disorders such as mood disorders, anxiety disorders, psychotic disorders, eating disorders, and substance use disorders. The use of patient self-report diagnostic questionnaires, such as the Patient Health Questionnaire[53] or the Center for Epidemiological Studies—Depression Scale,[54;55] may help to establish specific diagnoses. However, symptom severity rating scales such as the Beck Depression Inventory (BDI) or the State-Trait Anxiety Inventory (STAI)(see below) are not adequate diagnostic measures.

Reporting standards. For the SCID, research reports should include which specific modules were used; who conducted the assessment and their specialty and training level; specific exclusionary diagnoses; and, if psychiatric diagnoses are not exclusionary, frequency counts for specific psychiatric diagnoses in the study sample. The proportion of subjects who have insomnia comorbid with psychiatric disorders should always be reported. In addition, the combination of RDC for Insomnia and SCID information should also permit reporting of the proportion of the sample with RDC-defined Insomnia due to a Mental Disorder. If interrater reliability of SCID diagnoses is assessed, κ values should be reported. When clinical psychiatric history or self-report questionnaires are used, research reports should indicate the specific methods and tools used in the evaluation, as well as the other information described above for the SCID.

Diagnosis of Medical Disorders

Essential measure: Medical history (interview, physical exami-

nation, questionnaires). Some form of medical history should be conducted in all insomnia research studies. A clinical history is the preferred method when feasible. The history should include a review of the individual's known medical disorders, evaluation of other common medical disorders, and a review of systems. The process may be aided by the use of published checklists, questionnaires, and semistructured interviews (e.g., the Cornell Medical Index,[56;57] Charleson Comorbidity Scale[58]) or by locally developed questionnaires. Disorders referable to all major organ systems should be assessed, including neurologic, cardiovascular, pulmonary, musculoskeletal, renal, and endocrine systems. Systematic measurement of height, weight, and vital signs should also be conducted, and body mass index should be computed. A physical examination is also recommended but is not considered essential, since there are no pathognomonic features of the physical exam in insomnia nor is there any specific finding that would rule out insomnia. Further, a physical examination may not be feasible in some research settings. However, a physical examination should be considered essential for those studies involving pharmacologic treatment, assessment that has more than minimal health risks, and vulnerable populations (e.g., medically comprised older adults). In studies in which a full history is not feasible, a checklist of common medical conditions should be completed by the participant.

Reporting standards: Research reports should include documentation of the specific methods used (interview, physical examination, questionnaires), the type of individual or individuals responsible for the evaluation (physician, psychologist, graduate student, etc.), the mean and standard deviation (SD) of the body mass index for the sample, specific exclusionary diagnoses, frequency counts of specific comorbid medical conditions, and the proportion of individuals having insomnia comorbid with medical conditions. If sufficiently detailed information is available, the proportion of subjects meeting RDC-defined criteria for Insomnia due to a Medical Condition should also be reported.

Evaluation of Medication and Substance Use

Essential measure: List of current medications (interview, participant-generated list). An accurate listing of current medication and substance use should be included in all insomnia research reports. Gathering medication information may be aided by asking the participant to bring in all current prescription and nonprescription medication bottles. Medication and substance use noted on sleep diaries may also contribute to accurate information. In addition to prescription medications, it is also important to investigate use of over-the-counter medications, herbal and other "natural" products, and supplements. Nonmedicinal substances include caffeine (including coffee, tea, soda, and pills), alcohol, tobacco, and drugs of abuse.

Reporting standards: Research reports should include documentation of a medication and substance history, specific methods used, and the person responsible for collecting this information. Exclusionary medications and substances should be indicated. In addition, the medications used by participants should be listed (grouped by major classes or categories), together with the frequencies of use within the sample. The number or frequency of participants using caffeine, alcohol, and tobacco should be noted, as well as information regarding the amount used (e.g., mean/SD or median/range).

2. ASSESSMENT OF SLEEP AND INSOMNIA SYMPTOMS

a. Background and Rationale

Several well-established methods are available for characterizing sleep and insomnia symptoms, including questionnaires, sleep diaries, PSG, and actigraphy. The assessment of sleep and insomnia symptoms can serve several different functions in research studies: to establish or rule out specific insomnia or other sleep-disorder diagnoses, to document a severity criterion for study entry, to use as a contrast measure for individuals with insomnia with a comparison group, and to measure treatment effects. The utility of a specific method may vary according to the intended purpose.

Insomnia symptoms are often heterogeneous and may in part reflect inadequate sleep duration, quality, or efficiency. A multimethod approach using multiple measurement systems (ie, subjective, behavioral, physiological) is necessary to adequately capture these different domains or components of insomnia.[13] The time frame and number of evaluations may vary according to the assessment modality and the type of research questions under investigation (e.g., epidemiology vs clinical trials). For example, a single retrospective measure of sleep disturbances may be adequate for obtaining prevalence estimates, whereas repeated daily assessments are often preferable for treatment-outcome measurement. Despite the inevitable discrepancies among subjective, behavioral, and physiological assessment modalities, they should be seen as complementary, since they provide different perspectives on the construct of insomnia.

Global insomnia symptom questionnaires capture subjective aspects of sleep experience, typically over a period of time ranging from 1 week to 1 month. Global questionnaires typically include evaluations of several different dimensions of the insomnia, including quantitative aspects of sleep (e.g., sleep-onset latency [SOL], total sleep time [TST], number of awakenings [NWAK], wake after sleep onset [WASO]), qualitative aspects of sleep (e.g., sleep quality [SQ], sleep depth), and daytime symptoms referable to insomnia (e.g., difficulty concentrating, irritability). Numerous self-report questionnaires are available to measure clinical features of insomnia symptoms and insomnia disorder (for reviews see[9;13;59]). Examples include the Pittsburgh Sleep Quality Index (PSQI),[60] the Insomnia Severity Index (ISI),[30;61] the Athens Insomnia Scale,[62] and the Insomnia Severity Questionnaire.[63] Each of these has demonstrated insomnia-control differences, changes with treatment or both differences and changes.

Sleep diaries are subjective daily reports of sleep and sleep disturbances, typically completed upon arising, to provide an estimate of the previous night's sleep. Sleep diaries typically include measures of bedtime, SOL, NWAK, WASO, terminal wakefulness (TWAK), TST, final awakening time, and sleep efficiency (SE). (See Table 2 for definitions of common sleep variables.) Diaries also commonly measure SQ or depth, medication use, and daytime behaviors, such as napping and caffeine, alcohol, and medication use. Means for each variable are usually computed based on 1 to 2 weeks of data collection.[30;64] Although several sleep diaries have been published,[30;59;65-68] there is currently no standard format.

Sleep diaries may serve several functions but are most often used as a diagnostic tool and a quantitative dependent measure to contrast insomnia patients with a comparison group or to assess the impact of interventions. As a diagnostic tool, sleep diaries pro-

Table 2—Standard Acronyms and Definitions of Sleep Measures

Sleep Measure	Abbreviation	Definition
Number of Awakenings	NWAK	Number of awakenings, excluding the final awakening before the final arising
Sleep Quality	SQ	Subjective sleep quality, typically defined by responses on an ordinal or visual analog scale
Sleep Efficiency (percentage)	SE	Percent of time in bed spent asleep. When using sleep diaries, this is calculated from other self-report variables: TST/TIB x 100
Sleep-Onset Latency	SOL	How many minutes it takes to fall asleep, starting from the moment of intention to fall asleep,
Time in Bed	TIB	Time in bed, starting from the moment of intention to fall asleep and concluding with the final arising
Total Sleep Time	TST	Actual time slept. When using sleep diaries, this is typically calculated from other self-report variables (TIB–SOL–WASO–TWAK)
Terminal Wakefulness	TWAK	Amount of awake time between the final awakening and the time of getting out of bed
Wake After Sleep Onset	WASO	Total amount of time awake during the night, excluding SOL and TWAK

vide more-detailed information about quantitative sleep-disturbance severity and variability, sleep scheduling, and sleep-wake patterns than do most retrospective reports[65] and may be less sensitive to recall biases. Diary information may be helpful for establishing a general insomnia diagnosis and for ruling out circadian rhythm disorders.[59] Comparisons of sleep diaries with PSG may also be useful for identifying objective-subjective mismatches suggestive of Paradoxical Insomnia (sleep-state misperception).[3;4] However, sleep diaries in themselves do not completely capture the diagnostic criteria for specific insomnia disorders described in DSM-IV, ICSD-2, or RDC for insomnia. For instance, sleep-diary data from 1 to 2 weeks are not equivalent to 1- to 6-month diagnostic criteria, not all diaries evaluate SQ or satisfaction, diaries typically do not address the impairment and emotional distress associated with insomnia, and diaries cannot address historical information needed to establish some insomnia diagnoses.

Considered as a quantitative dependent measure, sleep-diary estimates of sleep parameters yield a reliable and valid index of insomnia symptoms even though they do not reflect absolute values obtained from PSG.[63;64] Individuals with insomnia tend to overestimate SOL and WASO and to underestimate TST in comparison to PSG measures.[64;69] By tracking sleep over several consecutive nights (sometimes up to 3 weeks for representative parameters such as WASO),[70] sleep diaries are more likely to capture the night-to-night variability that often characterizes the sleep of chronic insomnia. As such, sleep diaries may yield a more representative sample of an individual's sleep than 1-time questionnaires or 1 or 2 nights of PSG.

One major weakness of sleep diaries is that most forms include no validity check to determine whether they were completed on the days and times indicated. This potential difficulty can be mitigated by using computer-based,[71] web-based, or telephone call-in formats (interactive voice recording system[72]), which include time stamps and accurate identification of missing data. Another potential difficulty is that derived variables may be inaccurate. For instance, calculating TST based on time in bed (TIB), SL, and WASO depends on the accuracy of each variable, and values sometimes do not "add up."

PSG can also serve a variety of functions but is primarily used for screening and quantification of sleep disturbance. Instrumentation and optimal recording methods for this procedure have been well described elsewhere in the literature.[73-77] In clinical practice, PSG is not recommended for routine assessment, differential diagnosis, or severity assessment.[29] However, PSG is useful in research studies to evaluate and quantify other sleep pathologies, such as apnea and periodic limb movements, which are often present among individuals presenting with insomnia complaints.[35;78;79] PSG may be used to exclude potential participants who exceed a certain threshold (e.g., apnea-hypopnea index ≥ 10) or simply to characterize the study sample. In the insomnia research setting, PSG has also been used to determine whether participants meet specific quantitative criteria, e.g., SOL or WASO of more than 30 minutes.[80;81]

More generally, PSG sleep measures are used as dependent measures to quantify group differences or intervention effects. In this context, it is important to distinguish between insomnia, which is a disorder defined by a set of symptoms, and sleep disturbances, which refer to quantitative measurements of sleep and wake amounts. Patients with insomnia who have PSG-identified sleep disturbances are a subset of patients meeting the diagnostic criteria for insomnia, ie, a set of self-reported symptoms.[29;82-84] For this reason, PSG cannot serve as the sole outcome measure in clinical trials studying insomnia, as defined by standard diagnostic criteria,[20] nor should PSG be considered the "gold-standard" quantitative measure of insomnia against which other measures are judged and validated. Furthermore, PSG is only feasible to conduct for 1 to 3 nights in most cases, raising the possibility that it may provide insufficient sampling to adequately capture the features of insomnia, which may not be present every night. Finally, the expense and effort involved in PSG make it best suited for assessment studies and efficacy trials and may be less appropriate for epidemiologic studies and effectiveness trials.

Several methodologic issues can affect PSG as a quantitative measure of sleep disturbance in insomnia. First is whether to use data from the first night of PSG study, which is subject to the "first-night effect." Studies vary in their use of data from the first night (eg[81;85]). These data are often used to screen out participants if they have other major sleep disorders or if they sleep better than entry criteria allow. Second, the number of PSG nights can influence the reliability and statistical properties of the derived PSG measures.[70] Once again, studies vary on the number of PSG nights, representing a balance between lower variance with more nights and practical matters, such as cost with fewer nights. Averaging 2 nights for each study time point is a common choice.[13] Third is whether to record PSG data in participants' homes or in the laboratory. Home studies do not allow supervised dosing of medications, data entry, or other testing, and there is less control over the patients' behavior and environment, which may increase data loss and variance in PSG measures.[86] During home studies, participants do not all spend the same amount of TIB. On the other hand, home studies are probably more representative of the participants' typical sleep and sleep-related behaviors, they may require less technician time and cost, and they are less influenced

by "first-night effects."[86]

A final consideration is whether to employ a fixed TIB or to allow participants to choose their TIB, again with variable reports in the literature.[12;13] Allowing participants to choose their bedtimes is more representative of "usual" sleep and is preferred when assessing interventions that seek to manipulate sleep behavior.[12;13] However, self-selected bedtimes may increase between-participant variability in PSG measures and limit the ability to identify therapeutic effects. For example, if a medication has the effect of decreasing wake time, such an effect could be offset by a participant spending more TIB. Using a fixed TIB for PSG ensures greater comparability between participants and across time points, but it may introduce therapeutic effects of its own (ie, limiting TIB), diminishes ecologic validity, and requires that PSG studies be recorded in the laboratory, in order to ensure complete adherence prospectively.

Actigraphy is the measurement of physical movement using motion sensors, typically in a small device worn on the nondominant wrist.[87] The devices reliably detect movements and are cost-effective. Actigraphy can be used to collect information for a period of days to months in the individual's home environment and during normal routines. Actigraphy data can be displayed and scored manually or by computer algorithms that give estimates of sleep-wake and circadian-rhythm parameters.[88] Specific instruments vary according to the type of motion detector and the nature of the automated algorithms that estimate sleep and wake variables based on the measured patterns of activity and inactivity. As discussed in 3 major reviews[88-90] and 2 practice parameter papers,[91;92] actigraphy provides an objective estimate of sleep and the 24-hour sleep-wake rhythm and can detect circadian rhythm disturbances. Actigraphy can also be used to identify if patients with sleep disorders are being overly active near bedtime or generally inactive throughout the day.[88-92]

Several methodologic issues must be considered in using actigraphy in insomnia research studies. Few studies have compared different sensitivity settings, different devices, and different scoring algorithms, raising questions about reliability and validity across studies. Although well validated against PSG with normal sleepers and in special populations, the sensitivity and specificity of actigraphy for identifying sleep-wake patterns in insomnia is less compelling. Actigraphy is less reliable for distinguishing between quiet wakefulness and sleep, and it tends to overscore sleep in individuals with insomnia while they are lying quietly waiting to fall asleep at the beginning of the night or after awakenings from sleep (e.g., Hauri and Wisbey).[93] For these reasons, actigraphy is most often used in conjunction with other methods, such as a diary, to ascertain the time of lights out, time to bed, time out of bed, times the device was removed, and times the patient may have been sitting still for long time periods (such as sitting in a car or at the movies).

Recent data (Lichstein et al., 2005) validating actigraphy against PSG with a large insomnia sample did demonstrate strong correspondence between these 2 measures for 4 sleep variables, NWAK, WASO, TST, and SE. Validation data for SOL were weak. Actigraphic overscoring of sleep remained a problem for individuals taking hypnotics. This study signals progress in establishing the utility of actigraphy as an independent assessment tool for insomnia. To repeat the caution above, the results of this study are specific to a particular instrument using a particular algorithm. Generalization of these results to other combinations of instruments and algorithms remains to be determined.

Actigraphy, like sleep diaries and PSG, has been used as both a screening or diagnostic tool and as a quantitative dependent measure. In the clinical setting, actigraphy is not indicated for routine diagnosis, assessment of severity, or management of insomnia,[88;91;92] but it may be a useful adjunctive tool. In the context of screening or diagnosis in research studies, actigraphy is potentially useful for screening participants to establish disturbed rest-activity patterns, examine night-to-night variability, and identify individuals with circadian rhythm disorders.[88;89] Actigraphy has also been used as a quantitative dependent measure in insomnia research studies, particularly in treatment-efficacy studies (e.g., Friedman et al[94]), and it may be used as a measure of adherence in behavioral treatment studies. The precise role of actigraphy for establishing diagnoses or measuring treatment outcomes in insomnia research has not been well established. However, it is included in this report because of its potential as an objective measure of sleep-wake patterns to supplement self-reports and to use in situations in which PSG is not feasible.

b. Recommendations for Measures and Reporting Standards

Global Sleep and Insomnia Symptoms

Recommended/essential measures: (1) PSQI and (2) ISI. The PSQI[60] is a self-rating scale designed to measure general sleep disturbances. It is composed of 4 open-ended questions and 19 self-rated items (0-3 scale) assessing SQ and disturbances over a 1-month interval (although it has also frequently been used to evaluate disturbances for the past 7 days). The PSQI evaluates 7 domains, including SOL, duration, SE, SQ, disturbances, medication, and daytime dysfunction. A summation of these 7 component scores yields a global score of SQ, ranging from 0 to 21, with a cutoff score of 5 achieving maximum sensitivity and specificity for insomnia.[95] The PSQI has excellent psychometric properties, and, although it was not specifically designed for insomnia, it has been used in numerous assessment and treatment studies of insomnia (eg,[96-98]).

The ISI[30;61] is a 7-item scale assessing the perceived severity of insomnia symptoms (initial, middle, late), the degree of satisfaction with sleep, interference with daytime functioning, noticeability of impairment, and concern caused by the sleep problem, each rated on a 0- to 4-point scale. The usual time frame for responding is the last 2 weeks. A composite score is obtained by summing up the 7 ratings (total score ranges from 0-28), with a score of 14 providing the best cutoff to optimize sensitivity and specificity.[61;99] The following scoring guidelines can be used for quantifying insomnia severity: score of 0 to 7 (no significant insomnia), 8 to 14 (subthreshold insomnia), 15 to 21 (moderate insomnia), and 22 to 28 (severe insomnia). The ISI can be used as a screening instrument or as a treatment-outcome measure. Parallel versions can also be completed by clinicians and significant others to provide collateral validation of outcome.

Reporting standards for the PSQI include specifying the mean and SD of the global score for each group and/or time point. Depending on the specific research questions, means and standard deviations may also be reported for the 7 component scores. Reporting standards for the ISI include specifying the mean and SD of the total score for each group and/or time point. In clinical trials, it is useful to report percentage of participants scoring within the different severity levels.

Daily Self-Report of Sleep

Recommended/essential measure: One- to 2-week sleep diary. A sleep diary is useful as a screening or diagnostic tool and as a quantitative dependent measure. A number of published sleep diaries are available,[30;59;65-68] but none of these can be recommended as the preferred format. The sleep diary should assess the following daily information: times of going to bed, final awakening, and arising from bed; SOL; NWAK; WASO (which typically includes wake time after sleep onset exclusive of TWAK); TST; SE; SQ; and the timing and duration of naps or daytime sleep episodes.

Reporting standards for sleep diaries include reporting the duration, format, and information assessed in the diary; thresholds or quantitative criteria used to establish or confirm an insomnia diagnosis; thresholds or criteria used to establish or confirm the diagnoses of other sleep disorder (e.g., the average or range of sleep times used to support a diagnosis of a circadian rhythm sleep disorder); mean and SD of each of the dependent measures (see above) in each participant group; and, when feasible, some measure of the day-to-day variability of these measures (e.g., average of the within-subject range, SD, or coefficient of variation for key variables).

Objective Sleep

Recommended/essential measure: Polysomnography. PSG is recommended as a screening tool and as a quantitative dependent measure. Ideally, 1 night should serve as a screening PSG in order to identify and quantify sleep-related breathing disturbances and periodic limb movements during sleep. The PSG should use validated and contemporary measures for sleep-disordered breathing (e.g., nasal pressure transducer, thermocouples, inductance plethysmography, oximetry) and limb movements (bilateral anterior tibialis electromyogram), and these should scored according to accepted criteria.[74;77;100] One or 2 additional PSG nights should be conducted to derive quantitative sleep measures. These PSG studies would include, at a minimum, 1 centrally derived electroencephalography channel, 2 electrooculogram channels, and 1 bipolar electromyogram channel. Sleep stages should be scored in epochs of 20 or 30 seconds and scored according to standard criteria.[101]

Reporting standards for PSG include a description of the PSG protocol and procedures; specification of the apnea-hypopnea index[73;77] and periodic limb movement index (with and without arousal)[74;75] used to establish study eligibility or diagnoses; specification of group mean apnea-hypopnea index and periodic limb movement index associated with arousals; specification of other PSG criteria for study eligibility (e.g., a threshold for SOL, TST, or WASO); actual sleep times, whether times were fixed by the protocol or based on participants' preferred or habitual times, and how habitual times (if any) were determined; total recording time; SOL, NWAK, WASO, TWAK, TST, and SE. If two night are collected, data from each night should be reported separately; if more than two night are recorded, data should be reported for the first night individually and the other nights as an average. Means and SDs (in original time units) should be reported for each study group but may be supplemented by median, range, or other appropriate measures.

Rest-Activity Pattern

Recommended/essential measure: Actigraphy. Actigraphy is recommended as an objective measure of sleep as indicated by rest-activity patterns, both as a screening measure and as a quantitative dependent measure. Actigraphy may be useful for studies in which PSG may not be feasible. At least 1 of these objective measures should be used whenever feasible. Actigraphy should be collected for a minimum of 3 consecutive 24-hour periods,[102] and preferably for 1 to 2 weeks, using a validated device. Raw data should be inspected, and validated scoring algorithms used. Epoch lengths up to 1 minute are usually adequate.

Reporting standards for actigraphy include description of the make and model of device used; the type and version of scoring software used; the specific clinical protocol for actigraphy; thresholds or quantitative criteria used to establish or confirm an insomnia diagnosis; thresholds or criteria used to establish or confirm the diagnoses for other sleep disorder (e.g., the average or range of sleep times used to support a diagnosis of a circadian rhythm sleep disorder); mean and SDs across the measurement interval for sleep onset and offset times, SOL, NWAK, WASO, TST, and SE. When feasible, it is also recommended that research reports include data on internight variability within subjects for key measures (e.g., average of the within-subject range, SD, or coefficient of variation). Note that SOL can only be determined if time of "lights out" is reliably recorded. Therefore, the method of identifying "lights out" should be specified, whether by actigraph event marker, diary, or other means.

3. WAKING CORRELATES AND CONSEQUENCES OF INSOMNIA

a. Conceptual Background and Rationale

The clinical disorder of insomnia includes complaints of impaired daytime functioning, as well as sleep problems. Reported daytime impairments typically include fatigue, problems with cognitive or mental abilities (e.g., attention, concentration, memory), mood disturbances or, more generally, reduced quality of life (QoL). In fact, the RDC[4] for insomnia disorder require some evidence of functional daytime impairments.

Assessment of the correlates and consequences of insomnia rely on subjective, self-rated measures, as well as on more objective and standardized neurobehavioral performance measures. Although insomnia symptoms are commonly associated with subjective reports of daytime impairments,[103] there is much more limited evidence documenting a causal relationship.[10] Indeed, just as there are significant discrepancies between subjective and objective measures of sleep-wake parameters, there are also significant discrepancies between subjective and objective deficits of neurobehavioral performance.[104;105] Whether such discrepancies are due to a faulty appraisal mechanism, poor measurement sensitivity, or other factors is largely unknown. Nonetheless, such correlates or consequences represent important targets in the assessment of insomnia because of their prevalence and salience to patients.

Insomnia-Related Behavioral, Cognitive, and Arousal Factors. Physiologic and cognitive arousal, maladaptive behaviors, and specific beliefs and attitudes may serve as predisposing or perpetuating factors in chronic insomnia.[106] Measures of these factors may therefore serve as valuable indicators of etiology or important moderating or mediating factors for treatment outcomes.

Self-report instruments that assess arousal, behaviors, and cognitions relevant to insomnia include the Dysfunctional Beliefs and Attitudes About Sleep Scale (DBAS),[107;108] the Pre-Sleep Arousal Scale,[109] the Sleep Hygiene Awareness and Practices Scale,[110] the Ford Insomnia Response to Stress Test,[111] the Arousal Predisposition Scale,[112;113] and Regestein's Hyperarousal Scale.[114;115] Arousal can also be assessed in physiologic terms with measures such as plasma or salivary cortisol[116;117] to assess hypothalamic-pituitary axis activation; heart-rate variability to assess sympathovagal tone[118]; whole-body metabolic rate[119;120]; power spectral analysis of the waking or sleep electroencephalogram[121-123]; and functional neuroimaging, e.g., [^{18}F]-fluoro-deoxyglucose positron emission tomography scans[124;125] or single photon emission computed tomography.[126]

Most measures of this sort, whether self-report or physiologic, are relatively narrow with respect to the particular construct they assess. Furthermore, the physiologic measures are often expensive or difficult to measure or require expertise not widely available. For these reasons, measures of this sort are not well suited for general use across a range of insomnia studies and are not recommended for routine use. Nevertheless, these measures are encouraged in insomnia studies that address specific research questions, e.g., arousal in insomnia patients versus controls, or changes in arousal with treatment.

Fatigue and sleepiness. Fatigue and sleepiness have been considered to be either synonymous, different states on a continuum, or fundamentally different concepts. For purposes of defining insomnia research guidelines, they will be treated as 2 distinct states. Fatigue is defined as a subjective feeling of physical and/or mental weariness or tiredness but is not necessarily associated with increased sleep propensity.[127] Fatigue is a common complaint among individuals with insomnia, compared with good sleepers (e.g., [2;103;128-132]), and may be particularly important for insomnia characterized by nonrestorative sleep. The 2 most widely used and validated scales are the Fatigue Severity Scale (FSS)[133] and the Multidimensional Fatigue Inventory (MFI).[134] Several recently described fatigue scales warrant further testing in insomnia samples. FACES[129] is a 50-item questionnaire consisting of 5 subscales (fatigue, energy, consciousness, energized, and sleepiness), which have been distinguished in insomnia patients. The Tiredness Symptoms Scale[135] is a 14-item self-report scale describing physical and mental symptoms of tiredness. This scale also differentiates fatigue versus sleepiness in control participants, patients with insomnia, patients with narcolepsy, and patients with sleep apnea.

Sleepiness represents the actual tendency or propensity to sleep and can be measured subjectively with rating scales and objectively with the Multiple Sleep Latency Test (MSLT). Subjective sleepiness has been demonstrated less commonly than fatigue in patients with insomnia.[1;103;135] A majority of studies using the MSLT have shown no significant difference between patients with insomnia and control subjects, and approximately half have actually demonstrated increased mean MSLT values (ie, reduced objective sleepiness) among those with insomnia, suggesting heightened alertness rather than increased sleepiness.[119;136-147] However, in a review of studies prior to 2000,[10] 7 of 12 studies using the Stanford Sleepiness Scale found significantly greater self-reported sleepiness in patients with insomnia as compared with controls, and other studies have shown that approximately 20% of individuals with insomnia may have elevated subjective or objective sleepiness.[103;148] This suggests that some subgroups of patients with insomnia have increased sleepiness and that self-report and objective measures may provide different results, much as they do in terms of nighttime sleep.

Mood: Depression and Anxiety. A strong relationship between insomnia symptoms and depression and anxiety symptoms has been documented in numerous epidemiologic[1] and clinical[10] studies. Insomnia symptoms are 1 of the diagnostic criteria for several mood and anxiety disorders,[20] and insomnia "secondary" to mood disorders is the most common single diagnosis among patients with chronic insomnia in the general population[149] and in clinical samples.[150] Minor mood disturbances in categories such as depression, anxiety, and irritability are commonly reported by patients with insomnia.[103] A number of studies have assessed mood disturbances among patients with insomnia compared with control participants, but findings have been inconsistent due to differing methodologies. For instance, some studies specifically excluded individuals with clinical depression before comparing insomnia and control samples, whereas others did not; the latter studies generally find evidence for increased depression and anxiety scores among the insomnia sample.[10] The choice of specific scales has also differed. The Minnesota Multiphasic Personality Inventory[151] was widely used in a number of early studies but has been less commonly used recently, whereas scales such as the Beck Depression Inventory[152] have become more widely used in recent years. The BDI presents some limitations in older adults because physical and neurovegetative symptoms may be commonly experienced as a consequence of aging itself. Therefore, the Geriatric Depression Scale[153] is widely used in studies of older depressed adults. Many rating scales of depression and anxiety symptoms include items on sleep disturbance that may contribute to elevated total "mood disturbance" scores.

Psychomotor and cognitive performance. Individuals with insomnia commonly report impaired performance on daytime tasks, particularly those requiring concentration, sustained attention, and creative thinking.[103] Improvements in subjective ratings of concentration and performance have been demonstrated with treatment.[72] However, there is currently no standard or validated format for measuring or reporting patients' self-rated concentration or psychomotor performance. Furthermore, as is the case with night-time sleep and daytime sleepiness, self-reports and objective measures often show substantial discrepancies.

Cognitive and psychomotor performance can be objectively measured and quantified. Because individuals with insomnia complain of reduced sleep amount, most insomnia studies have sought to identify deficits in cognitive performance similar to those found following partial or total sleep deprivation (for reviews, see[10;11]). These tests can be grouped into general categories, including reaction time, vigilance or attention, motor function or coordination, verbal memory, mathematical reasoning, logical reasoning, and memory. However, published reviews demonstrate worse performance among patients with insomnia in only a minority of studies (approximately 10%-25%). The low number of statistically significant findings could indicate that performance is not impaired by insomnia, that the tests have not been sensitive enough to detect subtle differences in the measured domains, or that relevant performance dimensions have not been assessed. Even considering significant differences in test results across studies, findings have not been consistent. For example, significant differences in digit span were found in 1 of 2 studies,

short-term memory in 2 of 6 studies, long-term memory in 1 of 2 studies, vigilance in 1 of 5 studies, choice reaction time in 1 of 3 studies, and continuous performance in 1 of 4 studies reviewed. Insomnia patients had significantly worse balance in both of 2 studies reporting the variable,[154;155] which may be important in light of recent findings demonstrating that insomnia is a risk factor for falls and hip fractures in the elderly.[156]

Overall, results of psychomotor and cognitive performance testing in insomnia are not consistent with the changes one would expect from sleep deprivation. One potential explanation is that objective performance impairment exists in patients with insomnia but is masked by elevated levels of arousal. An alternative explanation is that psychomotor performance impairments can be "unmasked" only with more-sensitive testing protocols. For instance, in a recent study using the constant-routine protocol, Varkevisser and Kerkhof demonstrated substantial performance decrements in insomnia participants, compared with control participants, across a number of domains,[157] even though convincing evidence of hyperarousal was not demonstrated.

Given the current state of knowledge, no specific psychomotor or cognitive-performance measure can be recommended for routine use in insomnia studies. However, the identification of performance measures sensitive to the effects of insomnia remains a high research priority. When using psychomotor and cognitive-performance measures, investigators should specify the exact measures used, the testing protocol, and data including means and SDs for each study group and each assessment time.

QoL and global function. QoL is a construct with varying definitions, most of which include dimensions such as psychological health, physical well-being, occupational fulfillment, familial/social satisfaction, and standard of living. QoL instruments can be divided into 3 types: the first and broadest in scope were developed for use in general populations; a second set have been designed to assess health-related QoL in particular, with some of these (e.g., Nottingham Health Profile, Sickness Impact Profile) including items on sleep or related domains; a third set assesses disease-specific health-related QoL. Two sleep-related QoL instruments geared toward apnea and sleepiness have been developed and validated.[158;159] The frequent coexistence of psychiatric and medical conditions with chronic insomnia provides a major challenge for the development of an insomnia-specific QoL measure.

The most frequently used QoL questionnaire in insomnia studies is the Medical Outcomes Study Short Form-36 (SF-36).[160;161] Population- and disease-specific norms are available and provide the possibility of cross-disease comparisons. Most studies in insomnia have shown differences between insomnia samples and control or normal subjects on multiple scales of the SF-36. However, there is insufficient evidence to determine whether this instrument is sensitive to treatments for insomnia. Two disease-specific QoL scales for insomnia have been published. The Quality of Life in Insomniacs Scale (QOLI)[162] has been described in two publications and awaits further validation. More recently, initial description and validation of the Hotel Dieu-16 (HD-16)[163] scale has been presented; once again, it awaits further validation.

Another means of assessing QoL is to assess job performance or success in patients with insomnia compared with controls, but few data are available on this topic. One study found that self-reported poor sleepers in the Navy received significantly fewer promotions and were less likely to be recommended for reenlistment.[164] Questionnaire studies have shown that patients with self-reported insomnia report consequences at work, including significantly more errors, significantly more accidents, and poor efficiency.[165] Healthcare utilization has also been reported to be higher among patients with insomnia than patients without insomnia.[166] To our knowledge, no psychometrically validated instruments have been applied to assess healthcare utilization, accidents, and absenteeism in insomnia.

b. Recommendations for Measures and Reporting Standards

Insomnia-related behavioral, cognitive, and arousal factors. As discussed above, the relatively narrow focus and/or expertise required renders these measures unsuitable for routine use. However, their use is encouraged in appropriate research studies. When such measures are used, it is recommended that investigators describe the actual instruments used and present data, including group means and SDs, to permit comparisons with other studies.

Fatigue

Recommended/essential measures: (1) FSS or (2) MFI. The relatively small number of insomnia studies using validated measures makes it difficult to recommend a single instrument. Given their widespread use among patients of different types and substantial validation data, the FSS[133] or the MFI[134] should be used in insomnia research studies. The FSS is a 9-item self-report scale, with each item scored on a scale of 1 (strongly disagree) to 7 (strongly agree); higher scores indicate greater fatigue. It has good internal consistency as indicated by a Cronbach α coefficient of 0.8; good test-retest reliability, as indicated by a correlation coefficient of 0.84; and sensitivity to change in clinical state. Strengths of the FSS include its simplicity and brevity. Consistent differences have been identified between patients with insomnia and control participants, indicating higher levels of fatigue among patients. Lichstein and colleagues[128] have used a cutoff score of 5.5 as an indicator of impaired daytime function among patients with insomnia, representing 1 SD below the mean for treatment-seeking insomnia patients. Weaknesses of the FSS include the small number of studies that have used the instrument; questionable validity, since every item includes the word "fatigue" without defining the term; and absence of a multidimensional structure. The MFI is a 20-item self-report instrument with 5 empirically defined subscales representing dimensions of general fatigue, physical fatigue, mental fatigue, reduced motivation, and reduced activity.[134] Individual items are rated on a 1 to 5 scale, indicating how well that statement describes the respondent, with anchors of "Yes, that is true (1)" to "No, that is not true (7)." The MFI has good internal consistency, with an average Cronbach α coefficient of 0.84. Scores for the 5 scales are obtained by adding individual items and range from a minimum score of 4 to a maximum score of 20. No global score is obtained from the MFI, but, when a single score is needed, the General Fatigue scale is recommended. Construct validity has been evaluated with known-groups comparisons and convergent validity supported by correlations with other fatigue measures. The MFI has been well validated in studies of various medical disorders, but it has been used in only a single study of insomnia.[167]

Reporting standards. For the FSS, mean and SD for the total score should be reported for each study group and each assess-

ment point. For the MFI, mean scores and SDs for the 5 subscales should be reported for each study group and each assessment point.

Sleepiness. Physiologic sleepiness has not been consistently demonstrated in patients with insomnia, although it may be present in subgroups, such as those with comorbid medical insomnia and older adults. Given the current state of the evidence, no specific measure of sleepiness is recommended for routine use in insomnia studies. However, when a specific study design calls for measurement of subjective sleepiness, the ESS[168;169] is the preferred self-report measure of sleepiness, and the MSLT[170] is the preferred physiologic measure of sleepiness. Neither measure is specifically designed to assess sleepiness in individuals with insomnia, and each has substantial limitations. The ESS has an indeterminate time frame, and answers to the specific items depend on the respondent actually being exposed to a particular situation. The MSLT, on the other hand, provides an estimate of physiologic sleep propensity on a specific day, which may be affected by recent events, sleep-wake history, and the specific instructions given. Despite their limitations, the ESS and MSLT are the most widely used measures of sleepiness and, until new metrics are developed and validated, the most appropriate measures for sleepiness. Reporting standards include description of the testing protocol for the MSLT and presentation of mean and SD values for each study group and each assessment point.

Mood (Depression and Anxiety)

Recommended/essential measures: (1) BDI-II or Inventory of Depressive Symptomatology Self-Report (IDS-SR); 2) Trait portion of the STAI (STAI-T). The strong association of insomnia with depressive and anxiety symptoms and the frequent reporting of minor mood disturbances in insomnia patients justify the routine inclusion of depression and anxiety measures in insomnia research studies. The BDI[171] is the most commonly used self-report depression measure in insomnia studies, but the BDI-II has more favorable psychometric properties. New items bring the BDI-II in compliance with DSM-IV criteria for major depression, compared with the original version of the BDI. The BDI-II is a 21-item scale; the items are each rated on a 4-point scale ranging from 0 to 3 and summed to give a single score. A total score of 0 to 13 is considered minimally depressed, 14 to 19 is consistent with mild depression, 20 to 28 with moderate depression, and 29 to 63 with severe depression. The IDS is available in both clinician-administered (IDS-C) and self-report (SR) versions. The IDS-SR is recommended for insomnia studies. The IDS-SR is a 30-item scale with each item rated on a 4-point scale ranging from 0 to 3, with higher scores indicating more severe depression. It has strong psychometric properties, including high internal consistency, significant correlation with other measures of depression severity, and sensitivity to change.[172-174] The IDS-SR covers all symptom domains in the diagnosis of DSM-IV major depressive episode and also assesses melancholic and atypical symptoms. A brief version of the IDS-SR (16 items) has also been validated.[174] As noted above, the Geriatric Depression Scale may be preferable in some studies of older adults.

The STAI[175] is the most widely reported self-report anxiety instrument in insomnia studies. The STAI measures anxiety as both a current state ("feeling right now") and an enduring trait ("generally feel"), each evaluated with 20 items scored on a scale of 0 ("not at all") to 3 ("very much so"). The trait portion of this instrument (STAI-T) is recommended for use in insomnia research studies. Scores for trait anxiety range from 20 (mild) to 80 (severe) and are obtained by adding individual item scores. Psychometric data for test-retest reliability and internal consistency have been described, and convergent validity has been measured against other anxiety rating scales.

Reporting standards for the BDI-II, IDS-SR, and STAI-T include presentation of mean scores and SDs for each study group and each assessment point. Because these instruments include sleep items that contribute to the score, it is recommended that publications report the total scores as well as scores excluding sleep items.

Psychomotor and cognitive performance. As discussed above, the lack of consistency of findings in the literature prevents a recommendation for any single psychomotor or cognitive-performance measure in insomnia. However, further investigation of such measures is strongly encouraged, and identification of specific performance deficits associated with insomnia is a high research priority. When included, measures of this sort should be described in sufficient detail to permit replication and comparisons across studies. This would include reporting of the specific measure, time of testing, test protocol, and the means and SDs for all study groups and assessment points.

Health-Related Quality of Life

Recommended/Essential Measure: SF-36.[160;176] The SF-36 includes 36 self-report items regarding daytime functioning and items are grouped into 8 dimensions, each scored from 0 to 100, with higher scores indicating better function. Population norms as well as disease-specific norms for the 8 dimensions are available and permit cross-disease comparisons. Further research using insomnia-specific QoL instruments is strongly encouraged.

Reporting Standards. Research reports should include mean scores and SDs for each study group and each assessment point on the 8 SF-36 scales.

DISCUSSION

Insomnia research has accelerated in recent years, due to new developments in epidemiology, diagnosis and classification, pathophysiology, and therapeutics. In the midst of this bourgeoning insomnia research, it is crucial for investigators to have common metrics for describing insomnia participants and research outcomes. These tools will allow investigators and clinicians to evaluate the results of specific studies as well as results across different studies. The recommendations presented here represent a first step in the direction of greater standardization of insomnia research assessments. We have recommended standard research assessments and reporting standards in 3 key areas: diagnosis of insomnia and comorbid conditions; assessment of sleep and insomnia severity; and assessment of waking correlates and consequences of insomnia.

The intention of these recommendations is to provide greater standardization across insomnia research studies. Therefore, we recommend that all future insomnia studies under development make provisions for including these measures. For studies currently being conducted and reported, we recommend that investigators report as many of the recommended measures as possible, based on the actual protocol. In many cases, investigators may

have similar measures covering the domains specified above, if not the specific measures recommended. In this case, we recommend that the reporting standards advocated above be applied to those similar measures. In particular, by providing mean and variance measures for each participant group at each measurement point, investigators can ensure that subsequent quantitative analyses will make use of the greatest possible number of studies. The recommendations in this paper are intended to be adopted voluntarily by researchers in order to improve the quality of research being conducted and reported. However, they are not intended to determine suitability or acceptability of any specific report for publication or for funding.

The recommendations in this paper are intended for use in research studies and not as guidelines for clinical care. Although clinicians may find that several of these recommendations will help to better characterize their patients and treatment outcomes, they should not be taken as substitutes for a thorough initial clinical evaluation or for clinical judgment during a course of treatment.

Even in research practice, these recommendations are intended only as a minimum set of assessments to standardize reporting. Additional study-specific assessments not only are reasonable, but are actively encouraged. Such assessments, together with the standard ones, are an important method for advancing our understanding of insomnia. For this reason, we recognize that the current recommendations will need to be modified over time as additional data become available. We may find that some of the recommended assessments do not substantially improve our understanding of insomnia and its treatment, and we may also find that other assessments prove more useful. The specific methodologies related to each assessment type are also likely to improve over time.

The recommendations in this paper are presented in categories for 4 types of insomnia research. As noted in the Introduction, we recognize that these 4 categories are not, in reality, completely distinct. Many investigators, in seeking to increase the relevance of research to clinical care, attempt to balance controls for internal consistency with the need for generalizable results. This can lead to "efficacy studies" that also have elements of "mechanism" or "effectiveness" studies. Such hybrid designs can be highly informative and can help to bridge our understanding across research domains. Thus, our recommendations for the 4 types of research studies should not be taken in overly literal terms; hybrid studies should include a reasonable mix of assessments described for each prototype.

The most controversial measure in the development of these recommendations was actigraphy. We recognize that the role of actigraphy is not well defined at present, and some of the contributors thought that it should not be presented as a recommended or essential measure. We have included it here for 2 major reasons. First, despite its limitations, actigraphy is 1 of only 2 available measures for objective sleep-wake measurements. Its ease of use makes it more feasible than PSG in many research settings, particularly large intervention studies. Second, we expect that ongoing studies will better define the validity of actigraphy in insomnia research samples. The potential utility of this method warrants its recommendation despite relatively little supporting evidence at this time.

Development of these recommendations points to several important directions for future research. Among the most pressing needs are:

- Development and testing of standardized insomnia diagnostic instruments. Although several research groups utilize and have published insomnia diagnostic interviews, these have not included fully structured formats along the lines of the SCID, and they have not been written to provide specific ICSD-2 diagnoses. Even less work has been devoted toward developing a structured interview or standardized assessment techniques for common sleep disorders that enter the differential diagnosis for insomnia. Structured interviews can be developed for sleep disorders.[32] However, an ICSD-2–based instrument will require careful assessment of sensibility to participants, training requirements, ease of use for interviewers, and interrater reliability and validity.
- Development and testing of a standardized sleep diary. Although sleep diaries are considered the gold standard for subjective assessments of sleep, there is currently no single, widely accepted format. It is a very high priority for the field to develop and gain consensus on such an instrument. Although this would appear not to be very difficult at first glance, a number of important design elements need to be considered. For instance, it will be important to consider: how to account for "dozing" time in the evening; whether and how to distinguish the time a person goes to bed from the time they actually try to sleep; whether to calculate TST and WASO from other variables, or to ask subjects directly for these values; whether to define SE from total TIB, or from intended sleep time.
- Development of neurobehavioral or cognitive measures sensitive to insomnia deficits. Identifying consistent measurable effects of insomnia on neurobehavioral function is important for a number of reasons. First, it would help to characterize the disorder more fully and to measure severity in a functional domain. Second, it would help to develop and test hypotheses regarding etiology. Third, it could have utility as an outcome measure in treatment studies. Widely used measures such as the psychomotor vigilance task (e.g., [177;178]) should be investigated more thoroughly in insomnia disorder, given their sensitivity to sleep loss, circadian phase, and individual variation (e.g., [179]). However, as noted above, such measures rely on the conceptualization of insomnia as a form of sleep loss, which may not be the case. Therefore, novel measures or measurement strategies based on different theoretical models (e.g., hyperarousal) remain a high priority.
- More-sensitive objective measures of insomnia disorder. Insomnia is currently a diagnosis based entirely on clinical history and subjective symptoms. Furthermore, an enduring puzzle of insomnia diagnosis is the frequent discrepancy between subjective and objective sleep reports as measured by traditional PSG. Although various cognitive and physiologic theories have been proposed to explain this discrepancy and the pathophysiology of insomnia in general; there is currently no widely accepted objective measurement of insomnia. Although it is by no means clear that such a measure can be identified, some potential candidates should be further assessed. These include measures such as quantitative electroencephalography (power spectral analysis), heart-rate variability during sleep, and endocrine measures (e.g., cortisol).
- Multisite studies of instrument reliability and validity. In order to maximize the utility of diagnostic and assessment in-

struments being developed, they should be subjected to testing across centers. An insomnia research network would be ideal to develop instruments, test their reliability within and across sites, and establish their validity. Unfortunately, such a network currently does not exist.

These recommendations represent a first step toward greater standardization in insomnia research. Implementation of these recommendations will undoubtedly lead to further refinements of research practice and, hopefully, toward better understanding of a health condition that affects millions of individuals worldwide.

ACKNOWLEDGMENTS

This manuscript is the final product of a conference held in Pittsburgh, Pennsylvania, on March 10-11, 2005, entitled, "Recommendations for a Standard Research Assessment of Insomnia." The conference and manuscript preparation were supported by an educational grant from the National Institute of Mental Health; by unrestricted educational grants from Pfizer, Inc; Sanofi-Aventis, Sepracor, Inc; Takeda Pharmaceuticals; and by The Mental Health Intervention Research Center at the University of Pittsburgh (MH30915). Additional support from MH24652, AG00972, and AG26177 (DJB); AG08415 and CA85264 (SAI); MH67057, HL-073259 and Department of Veterans Affairs IIR-00-091 (JDE); AG14738 and DA13574 (KLL); and MH60413 and Canadian Institutes of Health Research MT42504 (CMM). Industry sponsors did not contribute to conference planning, nor did they participate in any aspect of manuscript preparation.

Conflict of interest statements: Dr. Buysse has served as a paid consultant and scientific advisory board member for Actelion; Cephalon, Inc; Eli Lilly; GlaxoSmithKline; Merck; Neurocrine Biosciences; Neurogen, Inc; Pfizer, Inc; Respironics; Sanofi-Aventis; Sepracor, Inc; Servier; and Takeda Pharmaceuticals North America, Inc. Dr. Ancoli-Israel has served as a paid consultant, scientific advisory board member, and/or speaker for Acadia; Cephalon, Inc; King Pharmaceuticals, Inc; Merck; Neurocrine Biosciences, Inc; Neurogen, Inc; Pfizer, Inc; Sanofi-Aventis; Sepracor, Inc; Somaxen; and Takeda Pharmaceuticals North America, Inc. Dr. Edinger has received research funding from Respironics and has served as a paid consultant and/or speaker for Sepracor, Inc; Fission Communications; and Axis Health. Dr. Lichstein has served as a paid consultant for Eli Lilly; Mini Mitter/Respironics; Takeda Pharmaceuticals North America, Inc; Neurocrine Biosciences, Inc; Pfizer, Inc; and Sepracor, Inc. Dr. Morin has served as a paid consultant and scientific advisory board member for Pfizer; Neurocrine Biosciences, Inc; Sanofi-Aventis; Sepracor, Inc; Shire Biochem; and Takeda Pharmaceuticals North America, Inc.

The listed authors are reporting on behalf of the individuals who contributed to the conference and this manuscript. All participants had the opportunity to review and comment on the draft manuscript. These other contributors wer: Célyne Bastien, PhD, Québec, QC; Ruth Benca, MD, PhD, Madison, WI; Michael Bonnet, PhD, Dayton, OH; Richard Bootzin, PhD, Tucson, AZ; Christopher Drake, PhD, Detroit, MI; Andrew Krystal, MD, Durham, NC; Kevin Morgan, B.Sc., PhD, Leicestershire, UK; Michael Perlis, PhD, Rochester, NY; Dieter Riemann, MD, PhD, Freiburg, Germany; Thomas Roth, PhD, Detroit, MI; Colin Espie, PhD, Glasglow, Scotland; Leah Friedman, PhD, Stanford, CA; Göran Hajak, MD, PhD, Göttingen, Germany; Allison Harvey, PhD, Berkeley, CA; Peter Hauri, PhD, Rochester, MN; Arthur Spielman, PhD, New York, NY; Edward Stepanski, PhD, Chicago, IL; Alexandros Vgontzas, MD, Hershey, PA; James Walsh, PhD, Chesterfield, MO; William Wohlgemuth, PhD, Durham, NC.

REFERENCE

1. Ohayon MM. Epidemiology of insomnia: What we know and what we still need to learn. Sleep Med Rev. 2002;6:97-111.
2. Fichten CS, Creti L, Amsel R, Brender W, Weinstein N, Libman E. Poor sleepers who do not complain of insomnia: myths and realities about psychological and lifestyle characteristics of older good and poor sleepers. J Behav Med. 1995;18:189-223.
3. Dorsey CM, Bootzin RR. Subjective and psychophysiologic insomnia: An examination of sleep tendency and personality. Biol Psychiatry. 1997;41:209-216.
4. Edinger JD, Bonnet MH, Bootzin RR, Doghramji K, Dorsey CM, Espie CA, Jamieson AO, McCall WV, Morin CM, Stepanski EJ. Derivation of research diagnostic criteria for insomnia: report of an American Academy of Sleep Medicine Work Group. Sleep. 2004;27:1567-1596.
5. Nowell PD, Mazumdar S, Buysse DJ, Dew MA, Reynolds CF, Kupfer DJ. Benzodiazepines and zolpidem for chronic insomnia: A meta-analysis of treatment efficacy. JAMA. 1997;278:2170-2177.
6. Holbrook AM, Crowther R, Lotter A, Cheng C, King D. Meta-analysis of benzodiazepine use in the treatment of insomnia. CMAJ. 2000;162:225-233.
7. Morin CM, Bootzin RR, Buysse DJ, Edinger JD, Espie CA, and Lichstein KL. Psychological and Behavioral Treatment of Insomnia. Sleep, in press.
8. Ohayon MM. Epidemiological study on insomnia in a general population. Sleep. 1996;19:S7-S15.
9. Moul DE, Hall M, Pilkonis PA, Buysse DJ. Self-report measures of insomnia adults: rationales, choices, and needs. Sleep Med Rev. 2004;8:177-198.
10. Riedel BW, Lichstein KL. Insomnia and daytime functioning. Sleep Med Rev. 2000;4:277-298.
11. Fulda S, Schulz H. Cognitive dysfunction in sleep disorders. Sleep Med Rev. 2001;5:423-445.
12. Martin JL, Ancoli-Israel S. Assessment and diagnosis of insomnia in non-pharmacological intervention studies. Sleep Med Rev. 2002;6:379-406.
13. Morin CM. Measuring outcomes in randomized clinical trials of insomnia treatments. Sleep Med Rev. 2003;7:263-279.
14. Leger D, Guilleminault C, Dreyfus JP, Delahaye C, Paillard M. Prevalence of insomnia in a survey of 12,778 adults in France. J Sleep Res. 2000;9:35-42.
15. Breslau N, Roth T, Rosenthal L, Andreski P. Sleep disturbance and psychiatric disorders: a longitudinal epidemiological study of young adults. Biol Psychiatry. 1996;39:411-418.
16. Ohayon MM, Caulet M, Guilleminault C. How a general population perceives its sleep and how this relates to the complaint of insomnia. Sleep. 1997;20:715-723.
17. American Academy of Sleep Medicine. The International Classification of Sleep Disorders, Second Edition (ICSD-2): Diagnostic and Coding Manual. Second Edition ed. 2005.
18. World Health Organization. The ICD-9-CM: International classification of diseases, clinical modification. 4th ed. Salt Lake City: Medicode: 1994.
19. World Health Organization. The ICD-10 classification of mental and behavioural disorders. Clinical descriptions and diagnostic guidelines. Geneva: World Health Organization; 1992.
20. American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorders (DSM-IV-TR). Fourth Edition, Text Revision ed. Washington, DC: American Psychiatric Association; 2000.
21. Buysse DJ, Reynolds CF, Kupfer DJ, Thorpy MJ, Bixler E, Man-

fredi R, Kales A, Vgontzas A, Stepanski E, Roth T, Hauri P, Mesiano D. Clinical Diagnoses in 216 insomnia patients using ICSD, and proposed DSM-IV and ICD-10 categories: A report from the APA/NIMH DSM-IV field trials. Sleep. 1994;17:630-637.
22. Ohayon MM, Roberts RE. Comparability of sleep disorders diagnoses using DSM-IV and ICSD classifications with adolescents. Sleep. 2001;24:920-925.
23. Lichstein KL, Durrence HH, Taylor DJ, Bush AJ, Riedel BW. Quantitative criteria for insomnia. Behav Res Ther. 2003;41:427-445.
24. Douglass AB, Bornstein R, Nino-Murcia G, Keenan S, Miles L, Zarcone VP, Guilleminault C, Dement WC. The Sleep Disorders Questionnaire. I: Creation and multivariate structure of SDQ. Sleep. 1994;17:160-167.
25. Miles LE. The sleep questionnaire and assessment of wakefulness. In: Guilleminault C, ed. Sleeping and waking disorders: Indications and techniques. Menlo Park, CA: Addison-Wesley Pub. Co. Medical/Nursing Division; 1982:383-312.
26. Partinen M, Gislason T. Basic Nordic Sleep Questionnaire (BNSQ): a quantitated measure of subjective sleep complaints. J Sleep Res. 1995;4:150-155.
27. Lacks P. Behavioral treatment for persistent insomnia. New York, NY: Pergamon Press; 1987:63-65.
28. Edinger JD, Means MK. Overview of insomnia: Definitions, epidemiology, differential diagnosis, and assessment. In: Kryger MH, Roth T, Dement WC, eds. Principles and practice of sleep medicine, fourth edition. 4th ed. Philadelphia, PA: Saunders; 2005:702-713.
29. Chesson A, Jr., Hartse K, Anderson WM, Davila D, Johnson S, Littner M, Wise M, Rafecas J. Practice parameters for the evaluation of chronic insomnia. An American Academy of Sleep Medicine report. Standards of Practice Committee of the American Academy of Sleep Medicine. Sleep. 2000;23:237-241.
30. Morin CM. Insomnia: Psychological assessment and management. New York-London: The Guilford Press; 1993.
31. Spielman AJ, Anderson MW. The clinical interview and treatment plannins as a guide to understanding the nature of insomnia: The CCNY insomnia interview. In: Chokroverty S, ed. Sleep disorders medicine: Basic science, technical considerations, and clinical aspects. 2nd ed. Boston: Butterworth-Heinemann; 1999:385-416.
32. Schramm E, Hohagen F, Grasshoff U, Riemann D, Hajak G, Weeb HG, Berger M. Test-retest reliability and validity of a structured interview for sleep disorders according to DSM-III-R(SIS-D). Am J Psychiatry. 1993;150(6):867-872.
33. Ohayon MM, Guilleminault C, Zulley J, Palombini L, Raab H. Validation of the sleep-EVAL system against clinical assessments of sleep disorders and polysomnographic data. Sleep. 1999;22:925-930.
34. Espie CA. Assessment and differential diagnosis. In: Lichstein KL, Morin CM, eds. Treatment of late-life insomnia. Thousand Oaks, CA: Sage; 2000:81-108.
35. Lichstein KL, Riedel BW, Lester KW, Aguillard RN. Occult sleep apnea in a recruited sample of older adults with insomnia. J Consult Clin Psychol. 1999;67:405-410.
36. Maislin G, Pack AI, Kribbs NB, Smith PL, Schwartz AR, Kline LR, Schwab RJ, Dinges DF. A survey screen for prediction of apnea. Sleep. 1995;18:158-166.
37. Youngstedt SD, Kripke DF, Klauber MR, Sepulveda RS, Mason WJ. Periodic leg movements during sleep and sleep disturbances in elders. J Gerontol A Biol Sci Med Sci. 1998;53:M391-M394.
38. Vgontzas AN, Kales A, Bixler EO, Manfredi RL, Vela-Bueno A. Usefulness of polysomnographic studies in the differential diagnosis of insomnia. Int J Neurosci. 1995;82:47-60.
39. Walters AS, The International Restless Legs Syndrome Study Group. Toward a better definition of the restless legs syndrome. Mov Disord. 1995;10:634-642.
40. Wunderlich GR, Evans KR, Sills T, Pollentier S, Reess J, Allen RP, Hening W, Walters AS. An item response analysis of the international restless legs syndrome study group rating scale for restless legs syndrome. Sleep Med. 2005;6:131-139.
41. Benca RM, Obermeyer WH, Thisted RA, Gillin JC. Sleep and psychiatric disorders: A meta-analysis. Arch Gen Psychiatry. 1992;49:651-668.
42. Ford DE, Kamerow DB. Epidemiologic study of sleep disturbances and psychiatric disorders: An opportunity for prevention. JAMA. 1989;262:1479-1484.
43. Weissman MM, Greenwald S, Nino-Murcia G, Dement WC. The morbidity of insomnia uncomplicated by psychiatric disorders. Gen Hosp Psychiatry. 1997;19:245-250.
44. Kessler RC, McGonagle KA, Zhao S, Nelson CB, Hughes M, Eshleman S, Wittchen HU, Kendler KS. Lifetime and 12-month prevalence of DSM-III-R psychiatric disorders in the United States. Results from the National Comorbidity Survey. Arch Gen Psychiatry. 1994;51:8-19.
45. National Institutes of Health. NIH State-of-the-Science Panel Statement. Manifestations and managementof chronic insomnia in adults. 2005.
46. Harvey AG. Insomnia: symptom or diagnosis? Clin Psychol Rev. 2001;21:1037-1059.
47. Lichstein KL, McCrae CS, Wilson NM. Secondary insomnia: Diagnostic issues, cognitive behavioral treatment, and future directions. In: Perlis ML, Lichstein KL, eds. Treating sleep disorders: The principles and practice of behavioral sleep medicine. New York: Wiley; 2003:286-304.
48. Lee-Chiong TL, Sateia MJ, Carskadon MA. Sleep Medicine. Philadelphia, PA: Hanley & Belfus; 2002.
49. Malow BA. Approach to the patient with disordered sleep. In: Kryger MH, Roth T, Dement WC, eds. Principles and practice of sleep medicine, fourth edition. 4th ed. Philadelphia, PA: Saunders; 2005:589-593.
50. Lichstein KL. Secondary insomnia. In: Lichstein KL, Morin CM, eds. Treatment of late-life insomnia. Thousand Oaks, CA: Sage; 2000:297-319.
51. First MB, Gibbon M, Spitzer RL, Williams JBW. Structured Clinical Interview for DSM-IV Axis I Disorders SCID I: Clinician version, administration booklet. Washington, D.C.: American Psychiatric Press; 1997.
52. Segal DL, Hersen M, Van Hasselt VB. Reliability of the Structured Clinical Interview for DSM-III-R: an evaluative review. Compr Psychiatry. 1994;35:316-327.
53. Spitzer RL, Kroenke K, Williams JB. Validation and utility of a self-report version of PRIME-MD: the PHQ primary care study. Primary Care Evaluation of Mental Disorders. Patient Health Questionnaire. JAMA. 1999;282:1737-1744.
54. Beekman AT, Deeg DJ, Van Limbeek J, Braam AW, De Vries MZ, van Tilburg W. Criterion validity of the Center for Epidemiologic Studies Depression scale (CES-D): results from a community-based sample of older subjects in The Netherlands. Psychol Med. 1997;27:231-235.
55. Radloff LS. The CES-D Scale: A self-report depression scale for research in the general population. Applied Psychological Measurement. 1977;1:385-401.
56. Brodman K, Erdman A, Lorge I, Wolff H. The Cornell Medical Index: An adjunct to medical interview. JAMA. 1949;140:530-534.
57. Pendleton N, Clague JE, Horan MA, Rabbitt PM, Jones M, Coward R, Lowe C, McInnes L. Concordance of Cornell medical index self-reports to structured clinical assessment for the identification of physical health status. Arch Gerontol Geriatr. 2004;38:261-269.
58. Charlson M, Szatrowski TP, Peterson J, Gold J. Validation of a combined comorbidity index. J Clin Epidemiol. 1994;47:1245-1251.
59. Savard J, Morin C. Insomnia. In: Antony MM, Barlow DH, eds. Handbook of assessment and treatment planning for psychological disorders. New York, NY: Guilford; 2002:523-555.
60. Buysse DJ, Reynolds CF, Monk TH, Berman SR, Kupfer DJ. The Pittsburgh Sleep Quality Index (PSQI): A new instrument for psy-

chiatric research and practice. Psychiatry Res. 1989;28:193-213.
61. Bastien CH, Vallieres A, Morin CM. Validation of the insomnia severity index as an outcome measure for insomnia research. Sleep Med. 2001;2:297-307.
62. Soldatos CR, Dikeos DG, Paparrigopoulos TJ. Athens Insomnia Scale: validation of an instrument based on ICD-10 criteria. J Psychosom Res. 2000;48:555-560.
63. Spielman AJ, Saskin P, Thorpy MJ. Treatment of chronic insomnia by restriction of time in bed. Sleep. 1987;10:45-56.
64. Coates TJ, Killen JD, George J, Marchini E, Silverman S, Thoresen C. Estimating sleep parameters: a multitrait--multimethod analysis. J Consult Clin Psychol. 1982;50:345-352.
65. Sateia MJ. Epidemiology, consequences, and evaluation of insomnia. In: Lee-Chiong TL, Sateia MJ, Carskadon MA, eds. Sleep Medicine. Philadelphia, PA: Hanley and Belfus; 2002:151-160.
66. Spielman AJ, Glovinsky PB. The diagnostic interview and differential diagnosis for complaints of insomnia. In: Pressman MR, Orr WC, eds. Understanding sleep: The evaluation and treatment of sleep disorders. Washington, D.C.: American Psychological Association Press; 1997:125-160.
67. Wohlgemuth WK, Edinger JD. Sleep restriction therapy. In: Lichstein KL, Morin CM, eds. Treatment of late-life insomnia. Thousand Oaks, CA: Sage; 2000:147-184.
68. Monk TH, Reynolds CF, Kupfer DJ, Buysse DJ, Coble PA, Hayes AJ, Machen MA, Petrie SR, Ritenour AM. The Pittsburgh Sleep Diary. J Sleep Res. 1994;3:111-120.
69. Means MK, Edinger JD, Glenn DM, Fins AI. Accuracy of sleep perceptions among insomnia sufferers and normal sleepers. Sleep Med. 2003;4:285-296.
70. Wohlgemuth WK, Edinger JD, Fins AI, Sullivan RJ. How many nights are enough? The short-term stability of sleep parameters in elderly insomniacs and normal sleepers. Psychophysiology. 1999;36:233-244.
71. Edinger JD, Means MK, Stechuchak KM, Olsen MK. A pilot study of inexpensive sleep-assessment devices. Behav Sleep Med. 2004;2:41-49.
72. Krystal AD, Walsh JK, Laska E, Caron J, Amato DA, Wessel TC, Roth T. Sustained efficacy of eszopiclone over 6 months of nightly treatment: results of a randomized, double-blind, placebo-controlled study in adults with chronic insomnia. Sleep. 2003;26:793-799.
73. Phillipson EA, Remmers JE. American thoracic society consensus conference on indications and standards for cardiopulmonary sleep studies. Am Rev Respir Dis. 1989;139:559-568.
74. Coleman R. Periodic movements in sleep (nocturnal myoclonus) and restless legs syndrome. In: Guilleminault C, ed. Sleeping and waking disorders: Indications and techniques. Menlo Park, California: Addison-Wesley; 1982:265-295.
75. EEG arousals: scoring rules and examples: a preliminary report from the Sleep Disorders Atlas Task Force of the American Sleep Disorders Association. Sleep 15, 173-184. 1992.
76. Carskadon MA, Rechtschaffen A. Monitoring and staging human sleep. In: Kryger MH, Roth T, Dement WC, eds. Principles and practice of sleep medicine, fourth edition. 4th edition ed. Philadelphia, PA: Elsevier Saunders; 2005:1359-1377.
77. Hirshkowitz M, Kryger MH. Monitoring techniques for evaluating suspected sleep-disordered breathing. In: Kryger MH, Roth T, Dement WC, eds. Principles and Practice of Sleep Medicine, Fourth Edition. 4th ed. Philadelphia, PA: Elsevier Saunders; 2005:1378-1393.
78. Jacobs EA, Reynolds CF, Kupfer DJ, Lovin PA, Ehrenpreis AB. The role of polysomnography in the differential diagnosis of chronic insomnia. Am J Psychiatry. 1988;145:346-349.
79. Edinger JD, Hoelscher TJ, Webb MD, Marsh GR, Radtke RA, Erwin CW. Polysomnographic assessment of DIMS: Empirical evaluation of its diagnostic value. Sleep. 1989;12:315-322.
80. Ware JC, Walsh JK, Scharf MB, Roehrs T, Roth T, Vogel GW. Minimal rebound insomnia after treatment with 10-mg zolpidem. Clin Neuropharmacol. 1997;20:116-125.
81. Zammit GK, McNabb LJ, Caron J, Amato DA, Roth T. Efficacy and safety of eszopiclone across 6-weeks of treatment for primary insomnia. Curr Med Res Opin. 2004;20:1979-1991.
82. Chesson AL, Ferber RA, Fry JM, Grigg-Damberger M, Hartse KM, Hurwitz TD, Johnson S, Kader GA, Littner M, Rosen G, Sangal RB, Schmidt-Nowara W, Sher A. The indications for polysomnography and related procedures. Sleep. 1997;20:423-487.
83. Littner M, Hirshkowitz M, Kramer M, Kapen S, Anderson M, Bailey D, Berry RB, Davila D, Johnson S, Kushida C, Loube D, Wise M, Woodson T. Practice parameters for using polysomnography to evaluate insomnia. Sleep. 2003;26:754-757.
84. Reite M, Buysse DJ, Reynolds CF, Mendelson W. The use of polysomnography in the evaluation of insomnia. Sleep. 1995;18:58-70.
85. Scharf MB, Roth T, Vogel GW, Walsh JK. A multicenter, placebo-controlled study evaluating zolpidem in the treatment of chronic insomnia. J Clin Psychiatry. 1994;55:192-199.
86. Edinger JD, Fins AI, Sullivan RJ, Marsh GR, Dailey DS, Hope TV, Young M, Shaw E, Carlson D, Vasilas D. Sleep in the laboratory and sleep at home: comparisons of older insomniacs and normal sleepers. Sleep. 1997;20:1119-1126.
87. Ancoli-Israel S. Actigraphy. In: Kryger MH, Roth T, Dement WC, eds. Principles and practice of sleep medicine, fourth edition. 4th ed. Philadelphia, PA: Saunders; 2005:1459-1467.
88. Ancoli-Israel S, Cole R, Alessi C, Chambers M, Moorcroft W, Pollak CP. The role of actigraphy in the study of sleep and circadian rhythms. Sleep. 2003;26:342-392.
89. Sadeh A, Acebo C. The role of actigraphy in sleep medicine. Sleep Med Rev. 2002;6:113-124.
90. Sadeh A, Hauri PJ, Kripke DF, Lavie P. The role of actigraphy in the evaluation of sleep disorders. Sleep. 1995;18:288-302.
91. Practice parameters for the use of actigraphy in the clinical assessment of sleep disorders. American Sleep Disorders Association. Sleep. 1995;18:285-287.
92. Littner M, Kushida CA, Anderson WM, Bailey D, Berry RB, Davila DG, Hirshkowitz M, Kapen S, Kramer M, Loube D, Wise M, Johnson SF. Practice parameters for the role of actigraphy in the study of sleep and circadian rhythms: an update for 2002. Sleep. 2003;26:337-341.
93. Hauri PJ, Wisbey J. Wrist actigraphy in insomnia. Sleep. 1992;15:293-301.
94. Friedman L, Benson K, Noda A, Zarcone V, Wicks DA, O'Connell K, Brooks JO, Bliwise DL, Yesavage JA. An actigraphic comparison of sleep restriction and sleep hygiene treatments for insomnia in older adults. J Geriatr Psychiatry Neurol. 2000;13:17-27.
95. Backhaus J, Jughanns K, Broocks A, Riemann D, Hohagen F. Test-retest reliability and validity of the Pittsburgh Sleep Quality Index in primary insomnia. J Psychosom Res. 2002;53:737-740.
96. King AC, Oman RF, Brassington GS, Bliwise DL, Haskell WL. Moderate-intensity exercise and self-rated quality of sleep in older adults. A randomized controlled trial [see comments]. JAMA. 1997;277:32-37.
97. Nierenberg AA, Adler LA, Peselow E, Zornberg G, Rosenthal M. Trazodone for antidepressant-associated insomnia. Am J Psychiatry. 1994;151:1069-1072.
98. Nofzinger EA. Substance-induced sleep disorder. In: Widiger TA, ed. DSM-IV Sourcebook. Washington,D.C.: American Psychiatric Press, Inc.; 1994:727-735.
99. Smith S, Trinder J. Detecting insomnia: comparison of four self-report measures of sleep in a young adult population. J Sleep Res. 2001;10:229-235.
100. American Academy of Sleep Medicine Task Force. Sleep-related breathing disorders in adults: recommendations for syndrome definition and measurement techniques in clinical research. The Report of an American Academy of Sleep Medicine Task Force. Sleep. 1999;22:667-689.
101. Rechtschaffen A, Kales A. A Manual of Standardized Terminology,

Techniques and Scoring System for Sleep Stages of Human Subjects NIH Publication 204. Washington, D.C.: U.S.Government Printing Office, Department of Health Education and Welfare; 1968.
102. American Sleep Disorders Association. Practice parameters for the use of actigraphy in the clinical assessment of sleep disorders. Sleep. 1995;18:285-287.
103. Moul DE, Nofzinger EA, Pilkonis PA, Houck PR, Miewald JM, Buysse DJ. Symptom reports in severe chronic insomnia. Sleep. 2002;25:553-563.
104. Broman JE, Lundh LG, Aleman K, Hetta J. Subjective and objective performance in patients with persistent insomnia. Scand J Behav Therapy. 1992;21:115-126.
105. Vignola A, Lamoureux C, Bastien C, Morin C. Effects of chronic insomnia and use of benzodiazopines on daytime performance in older adults. Journal of Gerontology: Psychological Sciences. 2000;55B:P54-P56.
106. Perlis ML, Smith MT, Pigeon WR. Etiology and pathophysiology of insomnia. In: Kryger MH, Roth T, Dement WC, eds. Principles and Practice of Sleep Medicine. Fourth Edition ed. Philadelphia: Elsevier Saunders; 2005:714-725.
107. Morin CM, Stone J, Trinkle D, Mercer J, Remsberg S. Dysfunctional beliefs and attitudes about sleep among older adults with and without insomnia complaints. Psychol Aging. 1993;8:463-467.
108. Espie CA, Inglis SJ, Harvey L, Tessier S. Insomniacs' attributions. psychometric properties of the Dysfunctional Beliefs and Attitudes about Sleep Scale and the Sleep Disturbance Questionnaire. J Psychosom Res. 2000;48:141-148.
109. Nicassio PM, Mendlowitz DR, Fussell JJ, Petras L. The phenomenology of the pre-sleep state: the development of the pre-sleep arousal scale. Behav Res Ther. 1985;23:263-271.
110. Lacks P, Rotert M. Knowledge and practice of sleep hygiene techniques in insomniacs and good sleepers. Behaviour Research & Therapy. 1986;24:365-368.
111. Drake C, Richardson G, Roehrs T, Scofield H, Roth T. Vulnerability to stress-related sleep disturbance and hyperarousal. Sleep. 2004;27:285-291.
112. Coren S. Prediction of insomnia from arousability predisposition scores: scale development and cross-validation. Behav Res Ther. 1988;26:415-420.
113. Coren S, Mah KB. Prediction of physiological arousability: a validation of the Arousal Predisposition Scale. Behav Res Ther. 1993;31:215-219.
114. Pavlova M, Berg O, Gleason R, Walker F, Roberts S, Regestein Q. Self-reported hyperarousal traits among insomnia patients. J Psychosom Res. 2001;51:435-441.
115. Regestein QR, Dambrosia J, Hallett M, Murawski B, Paine M. Daytime alertness in patients with primary insomnia. Am J Psychiatry. 1993;150:1529-1534.
116. Vgontzas AN, Bixler EO, Lin HM, Prolo P, Mastorakos G, Vela-Bueno A, Kales A, Chrousos GP. Chronic insomnia is associated with nyctohemeral activation of the hypothalamic-pituitary-adrenal axis: clinical implications. J Clin Endocrinol Metab. 2001;86:3787-3794.
117. Rodenbeck A, Huether G, Ruther E, Hajak G. Interactions between evening and nocturnal cortisol secretion and sleep parameters in patients with severe chronic primary insomnia. Neuroscience Letters. 2002;324:159-163.
118. Bonnet MH, Arand DL. Heart rate variability in insomniacs and matched normal sleepers. Psychosom Med. 1998;60:610-615.
119. Bonnet MH, Arand DL. 24-Hour metabolic rate in insomniacs and matched normal sleepers. Sleep. 1995;18:581-588.
120. Bonnet MH, Arand DL. Physiological activation in patients with Sleep State Misperception. Psychosom Med. 1997;59:533-540.
121. Perlis ML, Smith MT, Andrews PJ, Orff H, Giles DE. Beta/Gamma EEG activity in patients with primary and secondary insomnia and good sleeper controls. Sleep. 2001;24:110-117.
122. Krystal AD, Edinger JD, Wohlgemuth WK, Marsh GR. NREM sleep EEG frequency spectral correlates of sleep complaints in primary insomnia subtypes. Sleep. 2002;25:630-640.
123. Merica H, Blois R, Gaillard JM. Spectral characteristics of sleep EEG in chronic insomnia. Eur J Neurosci. 1998;10:1826-1834.
124. Nofzinger EA, Price JC, Meltzer CC, Buysse DJ, Villemagne VL, Miewald JM, Sembrat RC, Steppe DA, Kupfer DJ. Towards a neurobiology of dysfunctional arousal in depression: the relationship between beta EEG power and regional cerebral glucose metabolism during NREM sleep. Psychiatry Res. 2000;98:71-91.
125. Nofzinger EA, Buysse DJ, Germain A, Price JC, Miewald JM, Kupfer DJ. Functional neuroimaging evidence for hyperarousal in insomnia. Am J Psychiatry. 2004;161:2126-2131.
126. Smith MT, Perlis ML, Chengazi VU, Pennington J, Soeffing J, Ryan JM, Giles DE. Neuroimaging of NREM sleep in primary insomnia: a Tc-99-HMPAO single photon emission computed tomography study. Sleep. 2002;25:325-335.
127. Vgontzas AN, Zoumakis M, Papanicolaou DA, Prolo P, Vela-Bueno A, Kales A, Chrousos GP. Chronic insomnia is associated with a shift in interlukin-6 and tumor necrosis factor secretion from nighttime to daytime. Metabolism. 2002;51:887-892.
128. Lichstein KL, Durrence HH, Riedel BW, Taylor DJ, Bush AJ. Epidemiology of Sleep: Age, Gender, and Ethnicity. New Jersey: Lawrence Erlbaum Associates, Inc.; 2004.
129. Shapiro CM, Flanigan M, Fleming JA, Morehouse R, Moscovitch A, Plamondon J, Reinish L, Devins GM. Development of an adjective checklist to measure five FACES of fatigue and sleepiness. Data from a national survey of insomniacs. J Psychosom Res. 2002;52:467-473.
130. Hauri P, Fisher J. Persistent psychophysiologic (learned) insomnia. Sleep. 1986;9:38-53.
131. Means MK, Lichstein KL, Epperson MT, Johnson CT. Relaxation therapy for insomnia: nighttime and day time effects. Behav Res Ther. 2000;38:665-678.
132. Lichstein KL, Means MK, Noe SL, Aguillard RN. Fatigue and sleep disorders. Behav Res Ther. 1997;35:733-740.
133. Krupp LB, LaRocca NG, Muir-Nash J, Steinberg AD. The fatigue severity scale. Application to patients with multiple sclerosis and systemic lupus erythematosus. Arch Neurol. 1989;46:1121-1123.
134. Smets EMA, Garssen B, Bonke B, Haes de JCJM. The Multidimensional Fatigue Inventory (MFI); Psychometric qualities of an instrument to assess fatigue. J Psychosom Res. 1995;39:315-325.
135. Schneider C, Fulda S, Schulz H. Daytime variation in performance and tiredness/sleepiness ratings in patients with insomnia, narcolepsy, sleep apnea and normal controls. J Sleep Res. 2004;13:373-383.
136. Stepanski E, Zorick F, Roehrs T, Young D, Roth T. Daytime alertness in patients with chronic insomnia compared with asymptomatic control subjects. Sleep. 1988;11:54-60.
137. Schneider-Helmert D. Twenty-four-hour sleep-wake function and personality patterns in chronic insomniacs and healthy controls. Sleep. 1987;10:452-462.
138. Edinger JD, Fins AI, Sullivan RJ, Marsh GR, Dailey DS, Hope TV, Young M, Shaw E, Carlson D, Vasilas D. Do our methods lead to insomniacs' madness?: Daytime testing after laboratory and home-based polysomnographic studies. Sleep. 1997;20:1127-1134.
139. Lichstein KL, Wilson NM, Noe SL, Aguillard RN, Bellur SN. Daytime sleepiness in insomnia: Behavioral, biological and subjective indices. Sleep. 1994;17:693-702.
140. Mendelson WB, Garnett D, Gillin JC, Weingartner H. The experience of insomnia and daytime and nighttime functioning. Psychiatry Res. 1984;12:235-250.
141. Seidel WF, Ball S, Cohen S, Patterson N, Yost D, Dement WC. Daytime alertness in relation to mood, performance, and nocturnal sleep in chronic insomniacs and noncomplaining sleepers. Sleep. 1984;7:230-238.
142. Stepanski E, Lamphere J, Badia P, Zorick F, Roth T. Sleep fragmentation and daytime sleepiness. Sleep. 1984;7(1):18-26.

143. Rosa RR, Bonnet MH. Reported chronic insomnia is independent of poor sleep as measured by electroencephalography. Psychosom Med. 2000;62:474-482.
144. Sugerman JL, Stern JA, Walsh JK. Daytime alertness in subjective and objective insomnia: some preliminary findings. Biol Psychiatry. 1985;20:741-750.
145. Haynes SN, Fitzgerald SG, Shute GE, Hall M. The utility and validity of daytime naps in the assessment of sleep- onset insomnia. J Behav Med. 1985;8:237-247.
146. Pedrosi B, Roehrs T, Rosenthal L, Fortier J, Roth T. Daytime function and benzodiazepine effects in insomniacs compared to normals. Sleep Res. 1995;24:48.
147. Edinger JD, Glenn DM, Bastian LA, Marsh GR, Dailey D, Hope TV, Young M, Shaw E, Meeks G. Daytime testing after laboratory or home-based polysomnography: comparisons of middle-aged insomnia sufferers and normal sleepers. J Sleep Res. 2003;12:43-52.
148. Rosenthal LD, Meixner RM. Psychological status and levels of sleepiness-alertness among patients with insomnia. CNS Spectrums. 2003;8:114-118.
149. Ohayon MM. Prevalence of DSM-IV diagnostic criteria of insomnia: distinguishing insomnia related to mental disorders from sleep disorders. J Psychiatr Res. 1997;31:333-346.
150. Buysse DJ, Reynolds CF, Hauri PJ, Roth T, Stepanski EJ, Thorpy MJ, Bixler EO, Kales A, Manfredi RL, Vgontzas AN, Mesiano DA, Houck PR, Kupfer DJ. Diagnostic concordance for DSM-IV sleep disorders: A report from the APA/NIMH DSM-IV field trial. Am J Psychiatry. 1994;151:1351-1360.
151. Hathaway SR, McKinley JC. The Minnesota Multiphasic Personality Inventory Manual. New York: Psychological Corporation; 1967.
152. Beck AT, Steer RA, Ball R, Ranieri W. Comparison of Beck Depression Inventories -IA and -II in psychiatric outpatients. J Pers Assess. 1996;67:588-597.
153. Yesavage JA, Brink TL, Rose TL, Lum O, Huang V, Adey M, Leirer VO. Development and validation of a geriatric depression screening scale: a preliminary report. J Psychiatr Res. 1982;17:37-49.
154. Mendelson WB, Garnett D, Linnoila M. Do insomniacs have impaired daytime functioning? Biol Psychiatry. 1984;19:1261-1264.
155. Hauri PJ. Cognitive deficits in insomnia patients. Acta Neurol Belg. 1997;97:113-117.
156. Avidan AY, Fries BE, James ML, Szafara KL, Wright GT, Chervin RD. Insomnia and hypnotic use, recorded in the minimum data set, as predictors of falls and hip fractures in michigan nursing homes. J Am Geriatr Soc. 2005;53:955-962.
157. Varkevisser M, Kerkhof GA. Chronic insomnia and performance in a 24-h constant routine study. J Sleep Res. 2005;14:49-59.
158. Weaver TE, Laizner AM, Evans LK, Maislin G, Chugh DK, Lyon K, Smith PL, Schwartz AR, Redline S, Pack AI, Dinges DF. An instrument to measure functional status outcomes for disorders of excessive sleepiness. Sleep. 1997;20:835-843.
159. Flemons WW, Reimer MA. Development of a disease-specific health-related quality of life questionnaire for sleep apnea. Am J Respir Crit Care Med. 1998;158:494-503.
160. Ware JE, Snow KK, Kosinski M. SF-36 Health Survey: Manual and Interpretation Guide. Boston, MA: Health Institute, New England Medical Center; 1993.
161. Ware JC, Rose FV, McBrayer RH. The acute effects of nefazodone, trazodone and buspirone on sleep and sleep-related penile tumescence in normal subjects. Sleep. 1994;17:544-550.
162. Rombaut N, Maillard F, Kelly F, Hindmarch I. The Quality of Life of Insomniacs questionnaire (QOLI). Med Sci Res. 1990;18:845-847.
163. Leger D, Scheuermaier K, Raffray T, Metlaine A, Choudat D, Guilleminault C. HD-16: a new quality of life instrument specifically designed for insomnia. Sleep Med. 2005;6:191-198.
164. Johnson LC, Spinweber CL. Good and poor sleepers differ in Navy performance. Mil Med. 1983;148:727-731.
165. Leger D, Guilleminault C, Bader G, Levy E, Paillard M. Medical and socio-professional impact of insomnia. Sleep. 2002;25:625-629.
166. Simon GE, Von Korff M. Prevalence, burden, and treatment of insomnia in primary care. Am J Psychiatry. 1997;154:1417-1423.
167. Quesnel C, Savard J, Simard S, Ivers H, Morin CM. Efficacy of cognitive-behavioral therapy for insomnia in women treated for nonmetastatic breast cancer. J Consult Clin Psychol. 2003;71:189-200.
168. Johns MW. Reliability and factor analysis of the Epworth Sleepiness Scale. Sleep. 1992;15:376-381.
169. Johns MW. A new method for measuring daytime sleepiness: the Epworth sleepiness scale. Sleep. 1991;14:540-545.
170. Carskadon M, Dement W, Mitler M. Guidelines for the multiple sleep latency test (MSLT): A standard measure of sleepiness. Sleep. 1986;9:519-524.
171. Beck AT, Ward CH, Mendelson M, Mock J, Erbaugh J. An inventory for measuring depression. Arch Gen Psychiatry. 1961;4:561-571.
172. Rush AJ, Gullion CM, Basco MR, Jarrett RB, Trivedi MH. The Inventory of Depressive Symptomatology (IDS): psychometric properties. Psychol Med. 1996;26:477-486.
173. Trivedi MH, Rush AJ, Ibrahim HM, Carmody TJ, Biggs MM, Suppes T, Crismon ML, Shores-Wilson K, Toprac MG, Dennehy EB, Witte B, Kashner TM. The Inventory of Depressive Symptomatology, Clinician Rating (IDS-C) and Self-Report (IDS-SR), and the Quick Inventory of Depressive Symptomatology, Clinician Rating (QIDS-C) and Self-Report (QIDS-SR) in public sector patients with mood disorders: a psychometric evaluation. Psychol Med. 2004;34:73-82.
174. Rush AJ, Trivedi MH, Ibrahim HM, Carmody TJ, Arnow B, Klein DN, Markowitz JC, Ninan PT, Kornstein S, Manber R, Thase ME, Kocsis JH, Keller MB. The 16-Item Quick Inventory of Depressive Symptomatology (QIDS), clinician rating (QIDS-C), and self-report (QIDS-SR): a psychometric evaluation in patients with chronic major depression. Biol Psychiatry. 2003;54:573-583.
175. Spielberger CD, Gorsuch RL, Lushene RE. Manual for the state-trait anxiety inventory. Palo Alto, CA: Consulting Psychologists Press; 1970.
176. Ware JE, Sherbourne CD. The MOS-36-Item short form health survey (SF-36): I. Conceptual framework and item selection. Med Care. 1992;30:473-483.
177. Dinges DF, Powell JW. Microcomputer analyses of performance on a portable, simple visual RT task during sustained operations. Behavior Research Methods, Instruments, and Computers. 1985;652-655.
178. Dinges DF, Pack F, Williams K, Gillen KA, Powell JW, Ott GE, Aptowicz C, Pack AI. Cumulative sleepiness, mood disturbance, and psychomotor vigilance performance decrements during a week of sleep restricted to 4-5 hours per night. Sleep. 1997;20:267.
179. Van Dongen HP, Baynard MD, Maislin G, Dinges DF. Systematic interindividual differences in neurobehavioral impairment from sleep loss: evidence of trait-like differential vulnerability. Sleep. 2004;27:423-433.

J. Behav. Ther. & Exp. Psychiat. 40 (2009) 15–23

Contents lists available at ScienceDirect

Journal of Behavior Therapy and Experimental Psychiatry

journal homepage: www.elsevier.com/locate/jbtep

Metacognitive beliefs in primary insomnia: Developing and validating the Metacognitions Questionnaire – Insomnia (MCQ-I)

Joanne Waine [a], Niall M. Broomfield [a,*], Stephen Banham [b], Colin A. Espie [a]

[a] University of Glasgow Sleep Research Laboratory, Sackler Institute of Psychobiological Research, Southern General Hospital, Glasgow G51 4TF, Scotland, UK
[b] Department of Respiratory Medicine, Glasgow Royal Infirmary, 16 Alexandra Parade, Glasgow G31 2ER, Scotland, UK

ARTICLE INFO

Article history:
Received 25 April 2007
Received in revised form 18 February 2008
Accepted 4 March 2008

Keywords:
Metacognitive beliefs
Primary insomnia
Scale development
Sleep

ABSTRACT

Patients with Primary insomnia often experience intrusive, worrisome cognitive activity in the pre-sleep period. Metacognitive beliefs may explain this yet no valid reliable scale exists. The present study, therefore, developed the Metacognitions Questionnaire – Insomnia (MCQ-I). Following initial metacognitive insomnia profiling interviews, item refinement produced a preliminary 60-item MCQ-I. This was administered to 34 primary insomniacs and 37 normal sleepers. Psychometric data indicate primary insomniac patients score significantly higher than normal sleepers on MCQ-I. Test–retest reliability is good. Face, concurrent, construct and discriminant validity, scale sensitivity and specificity are all acceptable. Further research with larger primary insomnia and normal sleeper samples is now required.

1. Introduction

1.1. Defining primary insomnia

Primary insomnia disorder involves difficulty initiating and/or maintaining sleep despite adequate opportunity, in the absence of medical/psychiatric causes. Found in 1–2% of the general population, and 12–15% of sleep centre patients, primary insomnia is a known, independent risk factor for depression

* Corresponding author. Tel.: +44 141 232 1300; fax: +44 141 944 8867.
E-mail address: niall.broomfield@ggc.scot.nhs.uk (N.M. Broomfield).

0005-7916/$ – see front matter © 2008 Elsevier Ltd. All rights reserved.
doi:10.1016/j.jbtep.2008.03.002

(Riemann & Voderholzer, 2003). Impact on personal, professional and social functioning can be considerable, with fatigue, cognitive impairments and poor motivation commonly reported.

Whilst there is considerable evidence for efficacy of cognitive behavioural therapy in ameliorating primary insomnia (Morin, Culbert, & Schwartz, 1994; Murtagh & Greenwood, 1995; Smith et al., 2002), there remains a lack of research pinpointing how specific cognitive processes might interact, to maintain the disorder (Broomfield, Gumley, & Espie, 2005). One developing area of interest is mechanisms that fuel intrusive and worrisome cognitive activity (Harvey, 2005a).

1.2. Pre-sleep intrusive and worrisome cognitions in primary insomnia

Intrusions are spontaneously occurring, non-volitional thoughts associated with negative affect and which are difficult to control (Clark and Rhyno, 2005). Intrusions are likely to persist if appraised as threatening and receive attention focus, and at such times are experienced as worry and/or rumination (Harvey, 2005a).

There is widespread acceptance that intrusive thinking at bedtime characterises primary insomnia (e.g. Borkovec, 1982). Reports of mental events disrupting sleep are common place at clinic: "my mind keeps racing". In research studies, this is confirmed. Primary insomnia patients describe their pre-sleep thoughts as intrusive, uncontrollable and negative (Harvey, 2000; Kuisk, Bertelson, & Walsh, 1989), and *attribute* sleeping difficulties to intrusions (Broman & Hetta, 1994; Espie, Brooks, & Lindsay, 1989; Lichstein & Rosenthal, 1980; Nicassio, Mendlowitz, Fussell, & Petras, 1985). Moreover, experimentally induced cognitive intrusions delay sleep onset, in good and poor sleepers (Ansfield, Wegner, & Bowser, 1996; Gross & Borkovec, 1982, Hall, Buysse, Reynolds, Kupfer, & Baum, 1996).

Despite this evidence, theoretical explanations for *why* intrusive pre-sleep thinking characterises primary insomnia remain lacking.

1.3. The Self-Regulatory Executive Function (S-REF) model

One possible explanatory model is Wells' Self-Regulatory Executive Function (S-REF; Wells, 2000; Wells & Matthews, 1994, 1996). According to Wells, S-REF is switched on when there is a threatening discrepancy between the perceived self-state and the ideal state; for example, wakefulness when the desired goal is sleep. Critically, at the automatic processing level, physical (body state) as well as cognitive and external information is intrusive. So for the sleep-disturbed individual awake at bedtime, noise, thoughts about sleep, planning and being awake can all be intrusive (in theory) and thus lead to processing of the self as a wakeful person (cf. Wells, 2000).

S-REF may, therefore, account theoretically for the occurrence of persistent intrusive thinking in primary insomnia: wakefulness being a particularly salient intrusion to primary insomnia sufferers at bedtime, given their desired goal of sleep. Two metacognitive belief types should operate within S-REF in response to such intrusions: (i) beliefs concerning the meaning of the intrusions (e.g. thinking in bed prevents me getting to sleep) and (ii) plans that guide and shape the form that cognition takes (e.g. before I fall asleep, I should try and switch off my thoughts). We have argued elsewhere these belief types and their associated action plans e.g. thought control strategies, sleep effort, attention bias/focus should characterise insomnia (Broomfield et al., 2005). Importantly, although pre-sleep cognitive activity may not always be experienced as intrusive (e.g. Wicklow & Espie, 2000), any stimulus may do so if it clashes with a person's metacognitive beliefs, thereby triggering S-REF processing to reach a mental state more conducive to sleep e.g. a calmer mind.

1.4. Evidence for S-REF and primary insomnia

Several lines of evidence support metacognitive beliefs and associated action plans as characterising primary insomnia. First, we know primary insomnia patients engage in a range of thought control strategies at night. These include reappraisal, worry, thought suppression and punishment (Ellis & Cropley, 2002; Harvey, 2001; Ree, Harvey, Blake, Tang, & Shawe-Taylor, 2005). These strategies are likely to fuel further intrusions (Wegner, 1994) and maintain sleep disturbance. Primary insomnia patients also endorse more positive belief statements about worry (Harvey, 2003a).

Second, we know primary insomnia patients show elevated sleep effort, the anxious desire to control sleep onset (Broomfield & Espie, 2005), and selectively attend to salient internal and external threat cues. Both questionnaire based (Semler & Harvey, 2004a) and experimental work (e.g. Espie, Broomfield, MacMahon, & McPhee, 2006; Jones, Macphee, Broomfield, Jones, & Espie, 2005; Marcetti, Biello, Broomfield, MacMahon, & Espie, 2006) confirm the latter.

Finally, thought control, sleep effort and attention bias/focus have all been linked to delayed sleep onset latency (SOL) (Ansfield et al., 1996; Harvey, 2003a; Semler & Harvey, 2004b). Such cognitive 'action plans' feature in the metacognitive S-REF hierarchy (Wells, 2000), and support metacognitive belief structures as guiding intrusion appraisal.

1.5. Aim of present study

Metacognition has received very little attention in the insomnia literature. No scale exists which can directly assess presence of metacognitive beliefs. The aim of this exploratory study was, therefore, (i) to explore the presence of metacognitive beliefs in primary insomnia, (ii) to develop a metacognitive beliefs of primary insomnia questionnaire and, (iii) to gather initial data on psychometric properties of the scale, using primary insomnia and normal sleeping participants. The exploratory nature of the work meant we did not identify firm hypotheses.

2. Methods and results

Methods and results are combined, for the purposes of clarity.

2.1. Stage one: derivation of items for the questionnaire

2.1.1. Rationale

Metacognition is under-researched in primary insomnia. Therefore, initially, field interviews with individuals with primary insomnia and normal sleepers were conducted to gain direct insight into salient metacognitive beliefs held by poor sleepers, and to develop a preliminary item pool.

2.1.2. Participants

Participants for field interviews were recruited via University e-mail. Individuals with 'difficulties sleeping at night' or 'good sleepers', interested in assisting sleep research, and between 16 and 65 years old, were asked to contact the investigator and given information and consent forms.

Primary insomnia participants were not treatment seeking per se, but met strict research diagnostic criteria for primary insomnia disorder (Edinger et al., 2004): (i) difficulty initiating and/or maintaining sleep or non-restorative sleep (ii) with at least one associated daytime impairment (iii) for at least 1 month (iv) in the absence of a sleep disruptive medical/psychiatric condition, substance abuse (v) and/or other sleep disorder. Primary insomnia participants also had to complain of sleep difficulties at least three nights a week (Morin, 1993) and report a minimum SOL of $>$30 min on these nights. This follows clinical convention for diagnosing primary insomnia sleep onset type (Morin & Espie, 2003). All primary insomnia participants scored $>$6 on the Pittsburgh Sleep Quality Index (PSQI; Buysse, Reynolds, Monk, Berman, & Kupfer, 1989). Scores over 5 on PSQI reliably identify clinically significant sleep disturbance with 90% sensitivity and 87% specificity (Buysse et al., 1989). Additionally, participants were only included if they scored mild/normal on HADS depression and anxiety (Hospital Anxiety and Depression Scale; Zigmond & Snaith, 1983) and did not meet DSM-IV criteria for major depression. Any participants currently receiving any psychological treatments were also excluded.

Normal sleeping participants met RDC criteria for 'normal sleepers' (Edinger et al., 2004): (i) no complaints of sleep disturbance or daytime symptoms attributable to unsatisfactory sleep (ii) evidence of a routine standard sleep/wake schedule (iii) in the absence of a sleep disruptive medical/psychiatric condition, substance abuse (v) and/or a sleep disorder. Normal sleepers scored $<$5 on PSQI, and viewed themselves as 'good sleepers', described their sleep typically as 'good' or 'very good', and reported a mean SOL of $<$30 min. These conservative normal sleeping criteria ensured a representative cohort. HADS and DSM-IV depression criteria were per the primary insomnia group.

Participant suitability was determined using telephone screening. Of 23 respondents who returned consent forms, five primary insomniac participants were excluded (one withdrew, two failed to meet inclusion criteria, one had migraines which disrupted sleep, one had sleep paralysis and delayed sleep phase syndrome [DSPS]). No normal sleepers were excluded.

Nine primary insomnia sufferers (three males, six females; mean age = 25.3 years; mean sleep disturbance duration = 7.9 years, SD = 6.5; mean PSQI = 11.4, SD = 3.5), and nine normal sleepers (four males, five females; mean age = 28.8 years; mean PSQI score = 2.6, SD = 1.0) comprised the sample. No stage one participants took part later in the study.

2.1.3. Measures and procedures

Telephone screening followed receipt of consent. Individuals meeting inclusion criteria attended a face-to-face meeting where remaining screening measures (HADS and PSQI) were completed, and the field interview conducted. This was tape-recorded with the participant's permission and was based on a pre-existing metacognitive profiling interview (MPI; Wells, 2000), adapted for primary insomnia. In line with the original MPI, our adapted version examined recent periods of distress in the pre-sleep period in order to pinpoint salient cognitive processes. Thus, whilst the interview followed the structure and format of the Wells (2000) MPI, it was expanded to include aspects of the pre-sleep period considered intrusive to delineate relevant metacognitive beliefs/processes. Questions pertained to participants' experience of pre-sleep intrusions, the meaning and appraisals attached to intrusions (negative metacognitive beliefs about intrusions) and the metacognitive beliefs governing response (positive metacognitive beliefs about cognitive strategies). Specific coping strategies and cognitive processes employed to manage intrusions were also examined. The same areas were covered for each participant, the detail of which depended on the perceived relevance to each person. Interview format information is available from the senior author.

To further assist item identification, participants completed the MetaCognitions Questionnaire-30 (MCQ-30; Wells & Cartwright-Hatton, 2003) and the Utility of Pre-Sleep Worry Questionnaire (UPWQ; Harvey, 2003b). Finally, participants were debriefed and provided with a copy of the "Good Sleep Guide" to relay information on basic cognitive behavioural principles of good sleep (National Medical Advisory Committee, 1993).

An initial item pool was then generated through consensus discussion amongst the authors who have considerable expertise, not only on the psychological basis of insomnia, but also on models of psychopathology in general, including S-REF. The interview materials were available in literal, typed transcript format, and provided the primary source of information. That is, we identified and marked direct quotes wherever possible as potential item stems. We sought to achieve a broad representation of issues that were raised by our primary insomniac participants. Where specific parts of the interview content were similar and terminology appeared synonymous, we sought to construct items that faithfully represented what it was that participants were saying, and what we felt they were meaning by their words. In this context, interview information from normal sleepers provided important comparative data to help us recognise the differences in experience between people with primary insomnia and normal sleepers.

In addition to these rich interview data, our discussions were informed by data from other sources, so that we could effectively 'triangulate' and so confirm the validity of items. In this regard, we examined individual participant responses to the MCQ-30 and UPWQ. These questionnaires already contain generic (MCQ-30) or specific (UPWQ) items that are relevant to a metacognitive perspective on insomnia. Finally, we reviewed other existing insomnia questionnaires, from the perspective of metacognition (e.g. metacognitive beliefs arising from cognitive intrusions about sleep), and so tried to take into a further body of knowledge already available in the field (Glasgow Content of Thought Inventory GCTI, Harvey & Espie, 2004; Sleep Associated Monitoring Index SAMI, Semler & Harvey, 2004b; Glasgow Sleep Effort Scale GSES, Broomfield & Espie, 2005; Thought Control Questionnaire – Insomnia Revised TCQI-R, Ree et al., 2005; Pre-Sleep Arousal Scale, PSAS, Nicassio et al., 1985).

2.1.4. Data management

To ensure consistency with S-REF (Wells, 2000; Wells & Matthews, 1994, 1996) and Broomfield et al. (2005), derived items were then categorised by the consensus group according to metacognitive belief type (negative beliefs about danger and threat related meanings or positive beliefs about cognitive

strategies – the plan) and intrusion category (thoughts and images, being awake, physical arousal, environmental factors, or feelings) in a two by five matrix (cf. Rust & Golombok, 1999).

2.2. Stage two: refinement of the item pool and construction of the questionnaire

Duplicate items were removed from the matrix. The authors and two behavioural sleep medicine experts considered the remaining items, which were also discussed with a patient with primary insomnia meeting RDC/clinical criteria. In this way, what were felt to be the most appropriate items for each matrix cell were pinpointed. Metacognitive beliefs in response to intrusive thoughts (e.g. *Not being able to rest my mind in bed means I won't perform well the next day; Before I fall asleep, I should try and switch off my thoughts*) and wakefulness (e.g. *Being awake in bed means I have lost control of my sleep; When awake in bed, I should stay in bed and try harder to sleep*) were designated a higher number than other categories, since from interview data these were most relevant to participants. Fifty-four final items were agreed. A final discussion with authors and experts ensured clarity of statements. Lastly, resulting from further discussion and reflection upon the qualitative data, appropriate stem statements were added that appeared consistent with the way in which participants reported their insomnia problems at interview, and with the clinical experience of the authors (e.g. *Thinking in bed means* I won't get to sleep, *Before I fall asleep* I must get things sorted in my mind).

A draft scale was created by randomly ordering the 54 items. Instructions asked participants to indicate agreement on a four-point Likert scale (1 = "do not agree", 2 = "agree slightly", 3 = "agree moderately" and 4 = "agree very much") prior to falling asleep at night (cf. MCQ, MCQ-30). No reverse items were included, to avoid potential confusion of respondents.

This first draft was then sent to the $N = 9$ primary insomnia participants from field interviews. A semi-structured response sheet invited feedback and general comments. Based on responses, one item was dropped and seven items added, resulting in the 60-item preliminary Metacognitions Questionnaire – Insomnia (MCQ-I).

2.3. Stage three: MCQ-I field testing and psychometric evaluation

To investigate preliminary psychometric properties of MCQ-I, further primary insomnia and normal sleeper participants were recruited. Power calculation based on mean differences data from Cartwright-Hatton and Wells (1997) suggested that 21 participants per group were required assuming α 0.05, power at 0.8 and a large effect size.

2.3.1. Participants

The same criterion was applied to select groups. Of 67 'poor sleepers' who returned consent forms, 33 were excluded: 12 withdrew; 7 did not meet inclusion criteria for primary insomnia; 7 met DSM-IV criteria for depression, 2 had Delayed Sleep Phase Syndrome; 3 were substance abusing; 2 had chronic medical conditions. Of 51 normal sleepers who returned consent forms, 14 were excluded (12 did not meet inclusion criteria, 1 met DSM-IV criteria for depression, 1 participant returned incomplete data).

The final sample comprised 34 primary insomnia participants and 37 normal sleepers. Primary insomniacs and normal sleepers were equivalent on age and gender (both $p > 0.05$). People with primary insomnia had significantly longer SOL and sleep disturbance (both $p < 0.001$), and higher PSQI, higher HADS-A and higher HADS-D scores (all $p < 0.001$), than normal sleepers. Summary data are presented in Table 1.

2.3.2. Procedure

Following telephone screening, PSQI, HADS, MCQ-30, and MCQ-I were sent to participants for completion. Three weeks later, participants were sent the MCQ-I again, for test–retest purposes, and received the "Good Sleep Guide" (NMAC, 1993).

2.3.3. Data analysis

All continuous data were checked for normality of distribution, using the Kolmogorov–Smirnov (K–S) test. If the findings were significant, data was transformed using Log transformation. The

Table 1
Participant characteristics and screening measures (stage 3)

	Primary insomnia group ($n = 37$) Mean (SD) median	Normal sleeper group ($n = 34$) Mean (SD) median
Age	30 (9.2) 31.0	29 (9.2) 27.0
Problem duration (years)	10.2 (7.4) 7.5	0.0 (0.0) 0
PSQI	11.0 (2.7) 11.0	2.5 (1.0) 2.0
Subjective SOL (estimation in minutes from PSQI)	76.7 (45.4)[a] 65.0	9.6 (5.9)[b] 8.0
HADS (anxiety subscale)	7.6 (3.3) 8.0	3.6 (2.7) 3.0
HADS (depression scale)	3.7 (4.6) 4.0	1.1 (1.5) 1.0

[a] Primary insomnia group – missing data for subjective SOL = 2 (excluded from analysis).
[b] Normal sleeper group – missing data for subjective SOL = 1 (excluded from analysis).

distribution was then checked again. Based on the outcome, parametric or non-parametric tests were used accordingly.

One-tailed tests were employed and are reported here assuming directional hypotheses, with the exception of test–retest validity.

2.3.4. Face validity

The scale has good face validity because items were derived directly from participants who were also involved in the iterative three-stage process.

2.3.5. Concurrent validity

A significant positive correlation was found between the MCQ-I and MCQ-30 ($r = 0.69$, $p < 0.001$) corrected for attenuation. This association represents 47% of explained variance.

2.3.6. Construct validity

MCQ-I (total score) and estimated SOL (from PSQI) were positively associated ($r = 0.60$, $p < 0.001$), as were MCQ-I total score and PSQI total score ($r = 0.59$, $p < 0.001$).

2.3.7. Discriminant validity

As is evident in Fig. 1, the median value for primary insomnia participants (124.5) is higher than for normal sleepers (90.1). There is minimal overlap between lowest scoring primary insomnia participants and highest scoring normal sleepers.

MCQ-I discriminated primary insomnia (mean = 123.2, SD = 27.6) from normal sleepers (mean = 90.1, SD = 20.0) ($t = -5.838$, df = 69, $p < 0.001$), representing a medium effect size $r = 0.58$.

A linear relationship between MCQ-I and HADS-A for primary insomniacs ($r = 0.47$, $p = 0.003$) and normal sleepers ($r = 0.34$, $p = 0.02$) was observed. Therefore, ANCOVA with HADS-A partialled out was conducted. This was to establish whether differences between types of sleeper could be explained solely by anxiety levels. This indicated primary insomniacs and normal sleepers still differed significantly on MCQ-I ($F(1,68) = 10.74$, $p = 0.001$). Sleeper type explained 14% of variance.

2.3.8. Sensitivity and specificity

A cut-off of 110 on MCQ-I correctly identified 71% of primary insomniacs and 92% of normal sleepers. Using this cut-off, participants were separated into: 'high endorsement of metacognitive beliefs' (score = >111) and 'low endorsement of metacognitive beliefs' (score = <110). Mann Whitney U tests examined differences between groups on estimated SOL ($n = 68$) and PSQI score ($n = 71$). High endorsers had significantly longer SOL ($U = 142.5$, $p < 0.001$) and higher PSQI ($U = 172.5$, $p < 0.001$) than low endorsers, representing medium to large effect sizes ($r = -0.62$, -0.60, respectively).

2.3.9. Test–retest reliability

Test–retest data for 25 normal sleepers and 18 primary insomnia participants were available (mean interval 28.9 days). Intra-class correlations for all participants (ICC = 0.82, $p < 0.001$), normal sleepers (ICC = 0.63, $p < 0.001$) and primary insomniacs (ICC = 0.93, $p < 0.001$) were all significant.

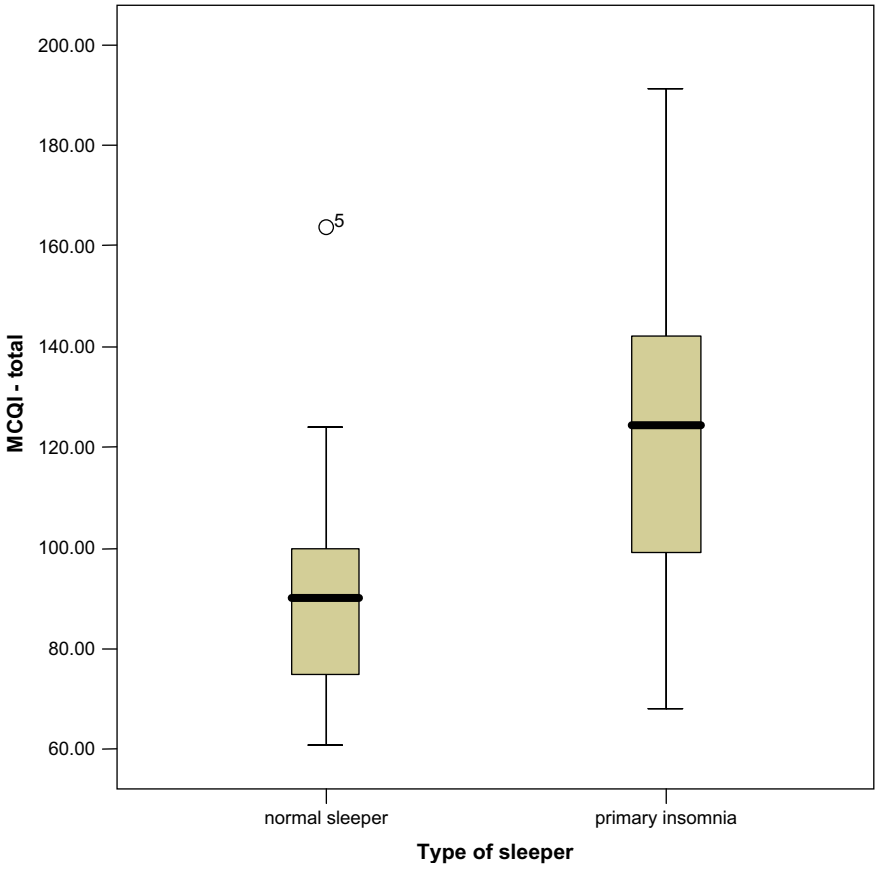


Fig. 1. Box and whiskers plot demonstrating MCQ-I's ability to discriminate between normal sleepers and primary insomnia participants.

2.3.10. Internal consistency

Internal consistency data were not calculated due to the high number of items in the preliminary version of the scale relevant to the *N* of participants ($N = 60$; cf. Cortina, 1993).

3. Discussion

The present study investigated the presence of metacognitive beliefs in primary insomnia through the refinement of a novel scale (MCQ-I), and reported initial MCQ-I psychometric data.

Using the pilot scale, significantly higher MCQ-I scores amongst primary insomniacs relative to normal sleepers were observed, an effect that remained when HADS-A was partialled out. This is the first attempt to describe and differentiate metacognition in primary insomnia, and confirms the probable relevance of S-REF to this sleep disorder. A range of metacognitive beliefs may shape pre-sleep cognition in primary insomnia (cf. Broomfield et al., 2005; Harvey, 2005a), although more work is needed. Metacognitive beliefs may reflect a stable trait of primary insomnia – a hypothesis supported by the large correlation ($r = 0.82$) between the first and second MCQ-I completion for primary insomnia participants. This was lower for normal sleepers ($r = 0.63$), suggesting items were less consistently meaningful to this group.

The current data cannot implicate metacognitive beliefs as a primary insomnia maintaining factor. Following further MCQ-I refinement, an important progression will be to test links between different S-REF components, using experimental methods. It should be possible, for instance, to induce

metacognitive beliefs/plans for processing in primary insomnia and normal sleepers, noting effect on pre-sleep intrusions and SOL. MCQ-I would provide a helpful manipulation check in this context.

MCQ-I should also prove fruitful in strengthening primary insomnia assessment. Unlike existing scales, MCQ-I can identify metacognitive beliefs in primary insomnia, of relevance for cognitive treatment approaches. Individuals high on MCQ-I may suit a package of cognitive therapy (e.g. Harvey, 2005b), or more tailored cognitive treatment emphasising attention retraining (Wells, 2000), mindfulness (Heidenreich, Tuin, Pflug, Michal, & Michalak, 2006) or metacognitive belief restructuring (Wells, 2000). Treatment approaches such as these, which directly or indirectly disengage S-REF processing, are in our view likely to be productive for certain primary insomnia patients.

The validity of the aforementioned work is strengthened given the reasonably promising preliminary psychometric properties of the MCQ-I. The MCQ-I correlated significantly with MCQ-30. Significant associations were observed between MCQ-I and both SOL and PSQI scores. MCQ-I readily discriminated primary insomnia sufferers and normal sleepers (mean scores primary insomnia = 132.2, normal sleepers = 90.1; effect size $r = 0.58$), even with anxiety controlled out. A score of 110 offered reasonable sensitivity (71%), and good specificity (92%), suggesting MCQ-I may usefully exclude normal sleepers from future research studies. Using this cut-off, high endorsers of metacognitive beliefs showed higher SOL and PSQI scores, relative to low endorsers. Lastly, test–retest reliability was highly acceptable although validation using a larger sample is now necessary.

We would acknowledge several limitations. A larger sample size would have enabled more rigorous psychometric examination, including exploration of factor structure, and computation of internal consistency data for subscales. Primary insomnia sufferers were not treatment seeking. However, mean sleep problem duration was over 10 years, and all participants met strict clinical and research diagnostic criteria (Edinger et al., 2004). Parallels can, therefore, be made with poor sleeping individuals approaching their General Practitioner for help. The inclusion of 1–2 nights of sleep data (diary and actigraphy) would have clarified more precisely the relationship between MCQ-I and SOL. And lastly, the MCQ-I refers only to the pre-sleep period; metacognitive beliefs may also relate to nighttime wakefulness after sleep onset. However, the majority of primary insomnia cognitive research to date has examined pre-sleep processes, making it a logical starting point here.

Finally, we sought to include obstructive sleep apnea patients, as a highly rigorous control group. Unfortunately, high levels of psychopathology necessitated their exclusion. Future MCQ-I research should endeavour to include a sleep specific control cohort, perhaps snorers, who would be less likely to show such elevated depression.

The present study was a preliminary scale development study. Future psychometric research of MCQ-I is required, using a substantially larger sample size, additional ancillary measures of cognitive activity, inclusion of a sleep specific control group e.g. snorers and measures of sleep. Arguably, by maximising MCQ-I sensitivity, the clinical utility of the scale will be augmented. And if findings remain as promising as ours, consideration should also be given to development of a specific metacognitive model of primary insomnia, similar to that for generalised anxiety (Wells, 1995) and obsessive–compulsive disorder (Wells & Matthews, 1994). Certainly, the MCQ-I, the first scale able to directly measure metacognitive beliefs in primary insomnia, shows promising psychometric properties.

References

Ansfield, M. E., Wegner, D. M., & Bowser, R. (1996). Ironic effects of sleep urgency. *Behaviour Research and Therapy, 34*(7), 523–531.
Borkovec, T. D. (1982). Insomnia. *Journal of Consulting and Clinical Psychology, 50*, 880–895.
Broman, J., & Hetta, J. (1994). Perceived pre-sleep arousal in patients with persistent psychophysiological and psychiatric insomnia. *Nordic Journal of Psychiatry, 38*(3), 203–207.
Broomfield, N. M., & Espie, C. A. (2005). Toward a valid, reliable measure of sleep effort. *Journal of Sleep Research, 14*, 1–7.
Broomfield, N. M., Gumley, A., & Espie, C. A. (2005). Candidate cognitive processes in psychophysiologic insomnia. *International Journal of Cognitive Psychotherapy, 19*(1), 5–18.
Buysse, D. J., Reynolds, C. F., Monk, T. H., Berman, S. R., & Kupfer, D. J. (1989). The Pittsburgh sleep quality index: a new instrument for psychiatric practice and research. *Psychiatry Research, 28*, 193–213.
Cartwright-Hatton, S., & Wells, A. (1997). Beliefs about worry and intrusions: the Meta-Cognitions Questionnaire and its correlates. *Journal of Anxiety Disorders, 11*(3), 279–296.
Clark, D. A., & Rhyno, S. (2005). Unwanted intrusive thoughts in nonclinical individuals: implications for clinical disorders. In D. Clark (Ed.), *Intrusive thoughts in clinical disorders theory, research and treatment* (pp. 1–29). New York: The Guilford Press.
Cortina, J. M. (1993). What is coefficient alpha? An examination of theory and applications. *Journal of Applied Psychology, 78*, 98–104.

Edinger, J. D., Bonnet, M. H., Bootzin, R. R., Doghramji, K., Dorsey, C. M., Espie, C. A., et al. (2004). Derivation of research diagnostic criteria for insomnia. Report of an American Academy of Sleep Medicine Work Group.
Ellis, J., & Cropley, C. (2002). An examination of thought control strategies employed by acute and chronic insomniacs. *Sleep Medicine, 3*, 393–400.
Espie, C. A., Brooks, D. N., & Lindsay, W. R. (1989). An evaluation of tailored psychological treatment of insomnia. *Journal of Behavior Therapy and Experimental Psychiatry, 20*(2), 143–153.
Espie, C. A., Broomfield, N. M., MacMahon, K. M., & McPhee, L. M. (2006). The attention–intention-effort pathway in the development of psychophysiologic insomnia: an invited theoretical review. *Sleep Medicine Reviews, 10*, 215–245.
Gross, R. T., & Borkovec, T. D. (1982). Effects of a cognitive intrusion manipulation on sleep-onset latency of good sleepers. *Behaviour Therapy, 13*, 117–124.
Hall, M., Buysse, D. J., Reynolds, C. F., Kupfer, D. J., & Baum, A. (1996). Stress related intrusive thoughts disrupt sleep onset and continuity. *Sleep Research, 25*, 163.
Harvey, A. G. (2000). Pre-sleep cognitive activity: a comparison of sleep-onset insomniacs and good sleepers. *British Journal of Clinical Psychology, 39*, 275–286.
Harvey, A. G. (2001). I can't sleep, my mind is racing! An investigation of strategies of thought control in insomnia. *Behavioural and Cognitive Psychotherapy, 29*, 3–11.
Harvey, A. G. (2003a). Beliefs about the utility of pre-sleep worry: an investigation of individuals with insomnia and good sleepers. *Cognitive Therapy and Research, 27*(4), 403–414.
Harvey, A. G. (2003b). The attempted suppression of presleep cognitive activity in insomnia. *Cognitive Therapy and Research, 27*(6), 593–602.
Harvey, A. G. (2005a). Unwanted intrusive thoughts in insomnia. In D. Clark (Ed.), *Intrusive thoughts in clinical disorders theory, research and treatment* (pp. 86–118). New York: The Guilford Press.
Harvey, A. G. (2005b). A cognitive theory and therapy for chronic insomnia. *Journal of Cognitive Psychotherapy, 19*(1), 41–60.
Harvey, K. J., & Espie, C. A. (2004). Development and preliminary validation of the Glasgow Contents of Thoughts Inventory (GCTI): a new measure for the assessment of pre-sleep cognitive activity. *British Journal of Clinical Psychology, 43*, 409–420.
Heidenreich, T., Tuin, I., Pflug, B., Michal, M., & Michalak, J. (2006). Mindfulness-based cognitive therapy for persistent insomnia: a pilot study. *Psychotherapy and Psychosomatics, 75*, 188–189.
Jones, B. T., Macphee, L., Broomfield, N. M., Jones, B. C., & Espie, C. A. (2005). Sleep-related attentional bias in good, moderate and poor (primary insomnia) sleepers. *Journal of Abnormal Psychology, 114*, 249–258.
Kuisk, L. A., Bertelson, A. D., & Walsh, J. K. (1989). Presleep cognitive hyperarousal and affect as factors in objective and subjective insomnia. *Perceptual and Motor Skills, 69*, 1219–1225.
Lichstein, K., & Rosenthal, T. (1980). Insomniacs perceptions of cognitive versus somatic determinants of sleep disturbance. *Journal of Abnormal Psychology, 89*, 105–107.
Marcetti, L. M., Biello, S. M., Broomfield, N. M., MacMahon, K. M. A., & Espie, C. A. (2006). Who is pre-occupied with sleep? A comparison of attention bias in people with psychophysiologic insomnia, delayed sleep phase syndrome and good sleepers using the induced change blindness paradigm. *Journal of Sleep Research, 15*, 212–221.
Morin, C. M. (1993). *Insomnia: Psychological assessment and management*. New York: The Guilford Press.
Morin, C. M., Culbert, J. P., & Schwartz, M. S (1994). Non-pharmacological interventions for insomnia: a meta-analysis of treatment efficacy. *American Journal of Psychiatry, 151*, 1172–1180.
Morin, C. M., & Espie, C. A. (2003). *Insomnia: A clinical guide to assessment and treatment*. New York: Kluwer Academic/Plenum Publishers.
Murtagh, D. R., & Greenwood, K. M. (1995). Identifying effective psychological treatments for insomnia: a meta-analysis. *Journal of Consulting and Clinical Psychology, 63*, 79–89.
National Medical Advisory Committee. (1993). The good sleep guide. In *Management of anxiety and insomnia*. London: HMSO.
Nicassio, P. M., Mendlowitz, D. R., Fussell, J. J., & Petras, L. (1985). The phenomenology of the pre-sleep state: the development of the pre-sleep arousal scale, *23*(3), 263–271.
Ree, M. J., Harvey, A. G., Blake, R., Tang, N., & Shawe-Taylor, M. (2005). Attempts to control unwanted thoughts in the night: development of the thought control questionnaire-insomnia revised (TCQI-R). *Behaviour Research & Therapy, 43*, 985–988.
Riemann, D., & Voderholzer, U. (2003). Primary insomnia: a risk factor to develop depression? *Journal of Affective Disorders, 76*(1), 255–259.
Rust, J., & Golombok, S. (1999). *Modern psychometrics: The science of psychological assessment*, (2nd ed.). London: Routledge.
Semler, C. N., & Harvey, A. G. (2004a). Monitoring for sleep-related threat: a pilot study of the sleep associated monitoring index (SAMI). *Psychosomatic Medicine, 66*(2), 242–250.
Semler, C. N., & Harvey, A. G. (2004b). An investigation of monitoring for sleep-related threat in primary insomnia. *Behaviour Research and Therapy, 42*(12), 1403–1420.
Smith, M. T., Perlis, M. L., Park, A., Giles, D. E., Pennington, J. A., & Buysse, D. J. (2002). Behavioural treatment versus pharmacotherapy for insomnia – a comparative meta-analyses. *American Journal of Psychiatry, 159*, 5–11.
Wegner, D. M. (1994). Ironic processes of mental control. *Psychological Review, 101*, 34–52.
Wells, A. (1995). Meta-cognition and worry: a cognitive model of generalised anxiety disorder. *Behavioural and Cognitive Psychotherapy, 23*, 301–320.
Wells, A. (2000). *Emotional disorders and metacognition innovative cognitive therapy*. Chichester: John Wiley and Sons, Ltd.
Wells, A., & Cartwright-Hatton, S. (2003). A short form of the metacognitions questionnaire: properties of the MCQ-30. *Behaviour and Research Therapy, 42*(4), 385–396.
Wells, A., & Matthews, G. (1994). *Attention and emotion: A clinical perspective*. Hove: Lawrence Erlbaum Associates.
Wells, A., & Matthews, G. (1996). Modelling cognition in emotional disorder: the S-REF model. *Behaviour and Research Therapy, 34*(11/12), 881–888.
Wicklow, A., & Espie, C. A. (2000). Intrusive thoughts and their relationship to actigraphic measurement of sleep: towards a cognitive model of insomnia. *Behaviour Research and Therapy, 28*, 679–693.
Zigmond, A. S., & Snaith, R. P. (1983). The hospital anxiety and depression scale. *Acta Psychiatrica Scandinavica, 67*(6), 361–370.

Sleep Medicine Reviews 14 (2010) 69–82

Contents lists available at ScienceDirect

Sleep Medicine Reviews

journal homepage: www.elsevier.com/locate/smrv

CLINICAL REVIEW

Insomnia and health-related quality of life

Simon D. Kyle [a,*], Kevin Morgan [b], Colin A. Espie [a]

[a] University of Glasgow Sleep Centre, Sackler Institute of Psychobiological Research, Faculty of Medicine, Southern General Hospital, Glasgow G51 4TF, Scotland
[b] Clinical Sleep Research Unit, Department of Human Sciences, Loughborough University, Leicestershire LE11 3TU, England

Keywords:
Insomnia
Health-related quality of life
Daytime functioning
Treatment
Measurement
Impairment

SUMMARY

Health-related Quality of Life (HRQoL) has become an important construct in contemporary medicine and health care, permitting assessment of disorder burden and evaluation of interventions on various aspects of functioning, in a standardized manner. Here we review literature on the measurement of HRQoL in insomnia populations, and the extent to which insomnia treatment improves domains of HRQoL. It is concluded from the relatively small literature that insomnia impacts on diverse areas of HRQoL, and that both pharmacological and non-pharmacological interventions can produce, to varying degrees, improvements in domains spanning physical, social and emotional functioning. Limitations of the current literature are identified; with particular emphasis on measurement and conceptual shortcomings. Suggestions are made in relation to improving the quality of future research, and how to further shed light on the impact of insomnia – and treatment thereof – on both HRQoL and global quality of life.

> "Sleep, like insomnia, is not just about what happens at night: it's about what happens to the day".[1(p 48)]

Introduction

Despite Insomnia being recognised as a '24-hour disorder' in both the major sleep nosologies,[2,3] historically, there has been less interest in the daytime aspects of insomnia compared with night-time symptoms and sleep parameters. Recent recommendations from leading researchers in the field[4–6] encourage further investigations into the waking consequences and correlates of insomnia. Indeed, clinical research in general, across a wide spectrum of illnesses, has moved towards a more holistic approach; looking beyond proximal symptoms, and viewing the patient within their wider psychosocial context.[7]

The purpose of this review is to: 1) give a brief overview of the known daytime consequences and morbidity associated with insomnia; 2) review work on the definition and measurement of quality of Life (QoL), or more specifically, health-related quality of Life (HRQoL), as it relates to insomnia; and finally, 3) outline a prospective research agenda, focusing on further understanding and measuring the extent to which insomnia, and it's treatment, impacts HRQoL and individual QoL.

Insomnia: daytime consequences and associated morbidity

Impairment in daytime functioning *attributed* to disturbed and/or poor quality sleep, features as one of the core diagnostic criteria for insomnia disorder.[2,3,8] Clinician reports of patient consultations,[9] and cross-sectional[10] and prospective questionnaire studies,[11,12] reveal that individuals with insomnia report consistent decrements in mood and cognitive abilities (concentration, memory, attention), coupled with elevated levels of anxiety, fatigue and physical pain/discomfort, relative to normal sleepers. Such impairments persist in those diagnosed with primary insomnia

Abbreviations: CBT, Cognitive Behavioural Therapy; CPAP, Continuous Positive Airway Pressure; DSM-IV, Diagnostic and Statistical Manual of Mental Disorders, 4th Edition; EORTC QLQ-C30, European Organization for Research and Treatment of Cancer Quality of Life Questionnaire; ERP, Event-Related Potentials; ES, Effect Size; ESS, Epworth Sleepiness Scale; FACT-G, Functional Assessment of Cancer Therapy – General; FOSQ, Functional Outcomes of Sleep Questionnaire; CGI, Clinical Global Impressions Scale; HD-16, Hotel-Dieu 16; HRQoL, Health Related Quality of Life; ICSD-2, International Classification of Sleep Disorders, Second Edition; IRQoL, Insomnia-Related Quality of Life; ISI, Insomnia Severity Index; MOS, Medical Outcomes Study; MRI, Magnetic Resonance Imaging; NAW, Number of Awakenings; NHP, Nottingham Health Profile; OISQ, Occupational Impact of Sleep Questionnaire; OSA, Obstructive Sleep Apnea; PI, Primary Insomnia; PSG, Polysomnography; PSQI, Pittsburgh Sleep Quality Index; QALY, Quality Adjusted Life Years; Q-LES-Q, Quality of Life Enjoyment and Satisfaction Questionnaire; QoL, Quality of Life; QOLI, Quality of Life of Insomniacs Questionnaire; QoLI, Quality of Life Inventory; QSQ, Quebec Sleep Questionnaire; RCT, Randomised Controlled Trial; SAQLI, The Calgary Sleep Apnea Quality of Life Index; SE, Sleep Efficiency; SF-36, Short-Form Health Survey 36; SIP, Sickness Impact Profile; SOL, Sleep Onset Latency; SWS, Slow-Wave Sleep; TAU, Treatment As Usual; TST, Total Sleep Time; WASO, Wake-time After Sleep Onset; WLQ, Work Limitations Questionnaire.

* Corresponding author. Tel.: +44 0141 232 7566.
E-mail address: s.d.kyle@clinmed.gla.ac.uk (S.D. Kyle).

1087-0792/$ – see front matter © 2009 Elsevier Ltd. All rights reserved.
doi:10.1016/j.smrv.2009.07.004

(PI), after screening and excluding co-morbid pathology.[11,13–16] Evidence for subjective (and objective) sleepiness, on the other hand, remains equivocal,[17,18] with surveys revealing approximately 25% of PIs report excessive daytime sleepiness.[19]

Although reviews of earlier studies failed to find reliable unequivocal differences in objective, neuropsychological functioning,[17,20] more recent controlled work, using sensitive measures that vary in task complexity and cognitive load, appear to be isolating and capturing specific impairments in attention and vigilance.[16,21,22] The heterogeneity in findings may be explained by both methodological (e.g., sample size and composition, assessment tools, time of testing), and theoretical factors (e.g., 'negative cognitive set', compensatory effort, and cortical arousal).[22–25] Further work is needed to tease out the contributing roles of each. Interestingly though, a recently presented meta-analysis[26] of all studies assessing neuropsychological performance in well-defined PIs, revealed significant impairments (small to moderate effect sizes) in aspects of attention, episodic and working memory, and executive functioning domains. Other groups have focused on the known relationship between sleep and consolidation of newly encoded memory traces. Specifically, PIs show an attenuation of the normal overnight sleep enhancement effect on tasks probing declarative[27] and procedural memory performance,[28] relative to normal sleeping controls.

Recent structural and functional imaging work may also shed light on the neural underpinnings of the cognitive dysfunction experienced by insomniacs. For example, Riemann et al.[29] reported decreased bilateral hippocampal volume in a small sample of PIs ($n = 8$) relative to controls, and, more recently, Altena et al.[30] found decreased gray matter volume in the left orbitofrontal cortex, in a larger sample ($n = 24$) of elderly insomniacs. Furthermore, the first published functional MRI study in PIs revealed hypoactivation of medial and inferior prefrontal regions during a verbal fluency task – a pattern which reversed/normalised post-behavioural therapy.[31] Although this imaging work is still in its infancy (and issues of causal ordering remain to be resolved), such atrophy, and functional alterations, may map onto reported daytime impairments in mood, memory, and reduced cognitive flexibility. Daytime event-related potential (ERP) data, though relatively sparse, also point to potential impairments in aspects of attention and processing speed[32,33]; though again, more work in this area using sophisticated paradigms, and large, well-defined samples is required. Recent ERP studies assessing sensory and cognitive processing pre-sleep, during sleep, and on awakening,[34,35] over multiple nights, are beginning to tease out the relationship between sleep quality and cortical arousal, which may have implications for better understanding daytime insomnia phenomenology.[36]

Large survey and population-based studies further reveal a number of increased morbidity markers in those suffering from insomnia, including: increased rates of health care utilization (physician visits, medication prescriptions) and chronic health problems,[37–39] elevated work absenteeism rates, reduced work productivity, and greater frequency of motor and non-motor accidents.[10,40,41] Such impairments may be moderated, in part, by co-occurring illness; however, workplace studies controlling for both mental and physical co-morbidities still reveal significant negative effects of insomnia on objective absenteeism, self-report work efficiency,[40] and work disability pension claims.[42] Longitudinal epidemiological studies also indicate that isolated sleep disturbance, measured at time point one, can independently predict the development of a new depressive episode 1–3 years later (for a review see [43]). Recent work also confirms insomnia as a predictor of future clinical anxiety.[44]

Crucially, perceived impact on daytime functioning serves as an important factor in driving help-seeking behaviour among individuals with insomnia, rather than simply perceived sleep loss.[45] For example, Morin et al.[46] found, in a large epidemiological study, that four out of five of the most commonly cited reasons for seeking a sleep-related consultation with a health professional, were daytime consequences of fatigue, psychological distress, physical discomfort, and reduced work productivity. Thus, once a threshold of noticeable daytime dysfunction is reached, individuals feel motivated to seek medical advice – ultimately with the hope that successful treatment will restore the particular functional impairment back to 'normal' status.[47]

Quality of Life (QoL) and Health-related Quality of Life (HRQoL)

In recent years there has been a shift towards assessing the overall impact of illness on aspects of QoL, through the measurement of HRQoL.[48] QoL and HRQoL have become well established terms in the medical and health literature – indeed, a pubmed search reveals that published work with the term 'Quality of Life' in the title or abstract has risen more than fourfold in the last ten years (1998–2008: 62,641), relative to the previous decade (1988–1998: 14,428). This has occurred mainly because of the recognition that objective changes in pathology rarely correlate with, or predict improvements in, functional capacity or patient experience, and that what the individual desires when seeking treatment is, put simply, a return to pre-illness well being. Similarly, with increasing life expectancy and medical technology advancement, the emphasis has shifted to chronic illness management (the quality of life) rather than simply the extension (quantity) of life. HRQoL has thus become a variable that can help policy makers decide on which treatments should get resources and service provision, relative to competing others (through cost-effective analyses and health technology assessment).

One effect of this increased attention to quality of life measurement has been the tendency, criticised in recent reviews and commentaries,[49–51] for researchers to include QoL and HRQoL scales in intervention and epidemiological studies without paying much attention to the concept they are purporting to measure. That is, QoL has become an 'umbrella term'[52] for a number of different concepts and definitions. The distinction between QoL and HRQoL is an important one, yet much of the health/medical literature seems to use these two terms interchangeably. QoL is widely regarded as a complex phenomenon: some argue it encompasses both objective and subjective indices of well being,[53] whereas others suggest it is a purely subjective impression of 'life satisfaction'.[50] Factors relevant to quality of life may thus range from emotional functioning and happiness, through to material well-being and education; it is therefore difficult to measure, not least with a single generic instrument. The World Health Organization[54] defines QoL as:

> "An individual's perception of their position in life, in the context of the culture and values in which they live and in relation to their goals, expectations, standards, and concerns" (WHOQoL study group).

A more contemporary definition proposed by Ruta, Camfield, & Donaldson,[55] based on seminal work by the economist-philosopher, Amartya Sen, views QoL in terms of a 'gap hypothesis':

> "Quality of Life is the gap between what a person is capable of doing and being, and what they would like to do and be; in essence it is the gap between capability and expectations" (p. 402).

Inherent in both these approaches is the importance of the individual in the assessment of QoL. The role of relativism and subjectivity in QoL assessment is perhaps best illustrated by the

'disability paradox'[56]: individuals who may have society defined functional/health impairment (e.g., cancer sufferers, amputees, the physically disabled), can report satisfactory, or in some cases, enhanced, quality of life.[50] Thus, health is just one of the many components implicitly factored into the quality of life equation. Simply put, having poorer health status does not necessarily mean that one has a lower quality of life, than say someone in impeccable health.[57]

HRQoL assessment, on the other hand, is concerned with isolating the impact of disease or illness on prominent aspects of functioning – '…the *radiating impact of pathology on the patient's wider world*'.[7(p578)] In clinical medicine the 'pathology' has both immediate, proximal symptoms (for example, in the case of insomnia, an increased sleep latency, or reduced total sleep time), and more 'downstream', distal consequences (such as reduced work performance, and social impairment). It is these latter, more psychosocial, variables that are the target of HRQoL assessment, capturing impairment relevant to patients' everyday functioning. Because health impact is easier to quantify than global QoL, most generic HRQoL instruments typically focus on similar aspects of functioning: covering physical, psychological (emotional) and social well-being.[58] Subsumed under these functioning domains are isolated symptoms, such as mood, memory, and fatigue. The various 'levels' of measurement have been likened to a pyramid[59]: the top of which can be thought of as overall subjective well-being/quality of life; the second level representing a collection of functional domains; and the third, foundation level, consisting of a number of isolated symptoms. Disease-specific HRQoL scales will tend to be tailored to aspects of impaired functioning that are most salient within a particular population, covering a mix of global functioning domains and relevant symptoms (e.g., fatigue, pain, and physical functioning in cancer patients). Table 1 provides descriptions of the scales used to assess HRQoL and QoL in insomnia populations.

Does insomnia negatively affect HRQoL?

Given the reported daytime symptoms attributed to poor sleep[2,3,8] it is reasonable to assume that individuals suffering from a chronic sleep problem may have a somewhat reduced *'downstream'* HRQoL. Indeed, about two decades ago, the first studies began to appear focusing on the relationship between insomnia and HRQoL. Rombaut et al.[60] created what they call the Quality of Life of Insomniacs (QOLI) questionnaire, a 59-item scale designed from the amalgamation of three other questionnaires (Leeds sleep evaluation questionnaire, the Jenkins sleep evaluation scale, and the Psychological well-being index) and 22 additional items, to assess functioning across five broad domains: quality of sleep, physical well-being, mood and mental state, and social and professional/work relationships. The scale therefore measures both sleep and non-sleep variables. The initial pilot study revealed good discriminant properties between untreated insomniacs and normal sleeping controls, with insomniacs showing statistical impairments on each domain. However, the scale has not been widely used since; only a few early studies have employed it,[61,62] or variants of it.[63] This is likely to be because of poor face validity (i.e., items are not grounded in the words of individuals with insomnia), the simultaneous evaluation of both sleep and daytime variables, varied response formats, and the exhaustive number of items. Nevertheless, the limited data on the QOLI do indicate sensitivity to impairment in a number of HRQoL domains, relative to normal sleepers.

The bulk of published studies focusing on HRQoL and insomnia have used a generic health status measure, the Medical Outcomes Study short-form health survey 36 (SF-36[64]). The SF-36 is a generic instrument, initially designed for assessment of health status across different disease states. Eight dimensions (36 items) assess aspects of functioning (emotional role limitations, energy and vitality, social functioning, physical functioning, physical role limitations, bodily pain, mental health) and perceived general health, yielding two component summary scores for mental and physical well being. Scores range from 0 to 100 – with lower scores indicating greater impairment in health status, or HRQoL. Short forms of the SF-36, the SF-12 and SF-8 have also been published and validated.

The first studies to use this instrument with insomnia populations consistently demonstrated lower scores on all domains, relative to normal sleepers.[39,65,66] Several large survey studies have also now reported a graded trend with insomnia severity; that is, those with more 'mild' or occasional insomnia symptoms show greater impairment on all eight domains relative to normal sleeping controls, whereas individuals (typically) satisfying criteria for insomnia disorder score significantly lower than both groups.[39,67–70,c] Such associations continue to hold after controlling for both physical[68,70] and mental health co-morbidities.[67,70] Although these studies differ in terms of what they classify as 'mild' or 'severe' insomnia, the linear pattern between HRQoL and insomnia severity consistently emerges.

More recently, Dixon et al.[71] analyzed baseline SF-36 data ($n = 209$) collected during a randomized trial of Cognitive Behavioural Therapy (CBT) to reduce hypnotic intake (cf.[72]), and compared them with UK normative values. In this study, the authors stratified domain scores by age, creating three groups (30–49, 50–69, 70–100 yrs), and compared them with their corresponding age-matched reference values. Similarly, it was found that those meeting minimum criteria for insomnia disorder (DSM-IV), in the 'young' category (ages 30–49), had significantly lower scores on all domains of the SF-36; the middle category (50–69 yrs) were impaired on all but two domains (emotional role limitation, physical role limitation); and the elderly category were significantly impaired in four out of the eight domains (pain, vitality, mental health, physical functioning), relative to reference values. The high degree of co-morbidities, particularly chronic illness and pain interference (anxiety and depression scores were in the normal to mild ranges), may mediate, to some extent, the magnitude of impaired dimensions.

In contrast to the more clinical and effectiveness based approach of Dixon et al., Walsh et al.[73] collected SF-36 data in the context of a multi-centred RCT of nightly Ezopiclone for a six-month period, on a large sample ($n = 830$, 21–64 yrs) of well-defined PIs (sleep parameter inclusion and DSM-IV criteria). Comparison of pre-treatment SF-36 scores with US normative reference values, revealed significant impairments in vitality, social functioning and the overall mental health summary component. The more selective decrements are likely to be explained by the stringent level of screening/exclusion and the nature of recruited versus referred populations.[45,74]

In a similar vein, Omvik et al.[75] recently published baseline data on daytime functioning and QoL variables from a randomized trial of CBT versus zopiclone (cf.[76]). SF-36 scores from 46 elderly individuals (>55 yrs: mean age 60.9 yrs) with DSM-IV criteria insomnia, who had undergone rigorous screening (including PSG), failed to reveal any significant evidence of impairment relative to normative values. The authors do not, however, report data for each of the eight dimensions; instead using the mental health and physical health component summary scores for comparison. This may obscure differences at the dimension level. Nonetheless, data collected on another generic instrument, the Quality of Life

[c] LeBlanc et al. used the SF-12 in their Quebec population-based study.

Table 1
Instruments used to assess HRQoL and QoL in insomnia populations.

Concept measured	Instrument	Brief description	Comments
Generic health status/HRQoL	SF-36[64]	36 Items covering 8 dimensions of functioning: physical functioning, physical role limitation, bodily pain, vitality, mental health, emotional role limitation, social functioning, health perception. Can calculate dimension scores, and two component summary scores for mental and physical well being. 12 and 8-item versions are also available.	Appears sensitive to insomnia impairment and treatment. Extensive normative data and disease norms available.
	NHP[80]	38 Items assessing impact of illness across 6 dimensions (sleep, energy, pain, physical mobility, social isolation, emotional reactions). Combined weighted values of individual items make up total dimension scores (0–100).	Simplistic dichotomous response format. Focus on extreme ill health.
	SIP[95]	136 Yes/no items grouped into 12 categories (body care and movement, ambulation, mobility, social interaction, alertness behaviour, emotional behaviour, household management, recreation and pastimes, communication, eating, work, sleep and rest). Items are completed with reference to 'today and because of health'. Global profile score, category scores, and summary physical and psychosocial scores, can be calculated.	Exhaustive number of items.
Quality of Life	QoLI[77]	Based on a model of 'life satisfaction', covering 17 domains (health and non-health) identified from the literature as being important for overall 'life satisfaction': health, selfregard, philosophy of life, standard of living, recreation, learning, creativity, social service, civic action, love relationship, friendships, relationships with children, relationships with relatives, home, neighbourhood, community.	Total score is based on domains only regarded as important and relevant by respondents.
	Q-LES-Q (short form)[101]	Contains 16 item-domains (health & non-health). 14 of these single item-domains make up the total score (physical health, mood, work, social relations, ability to function in daily life, ability to get around physically, household activities, family relationships, leisure, sexual drive, economic status, living or housing situation, vision, overall sense of well-being).	Sensitive to treatment outcome in depression and anxiety.
Disease-specific HRQoL	QOLI[60]	52 Item scale encompassing three questionnaires (Leeds Sleep evaluation questionnaire, Jenkins Sleep Evaluation Scale, Psychological well-being index) plus additional item questions, grouped into five domains: quality of sleep, quality of waking, physical well being, mood and mental state, and relationships.	Varying response formats.
	HD-16[81]	16 Item scale, covering five core domains (physical role, energy, cognitive, social, and psychological well-being). Global and domain scores can be calculated.	Items generated by insomnia patients and experts.

HD-16, hotel dieu-16; Q-LES-Q, quality of life enjoyment and satisfaction questionnaires; QOLI, quality of life of insomniacs questionnaire; QoLI, quality of life inventory; NHP, Nottingham health profile; SF-36, short-form health survey; SIP, sickness impact profile.

Inventory (QoLI[77]), in the same sample, also revealed scores within the normal range. The QoLI is based on a conceptual model of general 'life satisfaction', asking patients to rate the importance of 17 pre-determined domains deemed important for an enjoyable life, and thus covers a wide-range of items not necessarily specific to health. It is perhaps not surprising that the composite score of this measure failed to detect insomnia-relevant impairment. It is worth pointing out, however, that the key strength of this scale is that composite scores are calculated only on items that are rated as important/relevant by the individual.

Another study,[78] again focusing on older adults (58+ years), compared well-defined PIs ($n = 82$), normal sleepers ($n = 61$), and those with 'secondary' insomnia ($n = 46$) on dimensions of the SF-36. Only one difference was found for the comparison between PIs and normal sleepers; with PIs scoring lower on the vitality dimension. Those with 'secondary' insomnia were significantly impaired on seven of the eight dimensions (except role-emotional) relative to both the PI group and normal sleepers. The reduced sensitivity of the SF-36 to HRQoL impairment in elderly insomniacs, especially those with primary insomnia, seems to be a recurrent finding. It is unclear what may be mediating this trend. Perhaps elderly individuals, who have had their sleep problem for a long period of time, adapt and 'recalibrate' in a way that limits the impact of insomnia on aspects of functioning. Conversely, normative reference values for this age group may be low anyway, given the high level of co-morbidity and reduced functional abilities associated with normal ageing – subsequently obscuring potential differences.

Researchers have also used a variety of other tools to assess HRQoL. For example, Philip et al.[79] compared those with insomnia ($n = 986$), and normal sleepers ($n = 586$), on the Nottingham health profile (NHP[80]). The NHP, similar to the SF-36, measures salient areas of health functioning, comprising 38 yes/no statements under six main domains: energy level, pain, sleep, social isolation, emotional reactions, and physical abilities. Individuals with insomnia were characterized according to DSM-IV symptom criteria, and had to report difficulties at least three times per week for the last three months; daytime functioning was not assessed. Significant differences were observed on all domains, with the insomnia group evidencing significant impairment. This pattern of impairment was similarly found in a selected sub-group ($n = 442$) of the insomnia sample, screened for both organic sleep-complaints (using the Epworth Sleepiness Scale; ESS) and depressive symptoms.

Leger et al.[81] recently published the Hotel Dieu-16 (HD-16), a disease-specific measure designed to detect quality of life disturbance relevant to insomnia. Items for the HD-16 were initially generated from interviews with 20 patients and through expert consensus opinion. Factor analysis on an initial list of 43 items resulted in the final selection of 16 items, subsumed under five categories: physical role; energy, will to do things; cognitive (concentration, attention, memory); social (relationships with others); and psychological well being. Good sleepers ($n = 391$), 'mild' insomniacs ($n = 422$), and 'severe' ($n = 240$) insomniacs, were selected from SOFRES, a French polling institute. Mild insomniacs were defined as those with 'occasional' sleep difficulties; whereas those in the severe group had at least two insomnia complaints for the last month, and suffered impaired daytime functioning as a consequence. Initial screening excluded those with depressive or anxiety profiles; those with other medical co-morbidities were not identified or excluded. A linear trend was again found, similar to studies using the SF-36: mild insomniacs scored significantly lower on all dimensions, and global score, relative to good sleepers; severe insomniacs scored significantly

poorer than both groups, again on each dimension and total score. Thus, prima facie, the HD-16 does look sensitive to the functional impairments experienced by those with insomnia, and has good face validity given that items are grounded in words of sufferers. A further strength is the ability to configure a total score, which may be useful in outcome studies, as well as clinical settings. Despite being a relatively brief measure, however, dimension calculation looks to be a rather complex and arduous process (based on weighted coefficients from the factor analysis), and so this may preclude it from being used on a regular basis in clinical settings. Further studies are required to assess psychometric properties and sensitivity to change.

The HD-16 was principally devised because of the absence of a widely used insomnia-specific HRQoL instrument, as compared with, for example, the sleep apnea field, where there are three - the Functional Outcomes of Sleep Questionnaire (FOSQ[82]), the Sleep Apnea Quality of Life Index (SAQLI[83]), and the Quebec Sleep Questionnaire (QSQ[84]). The implication being: generic measures like the SF-36 may not pick up impairments relevant to those with insomnia.[85] Nevertheless, the main strength of the SF-36 is that it permits comparisons with other illnesses; which of course is vital in order to document the relative burden of insomnia. In this context, Katz and McHorney[70] found, in a cross-sectional analysis of data from the medical outcomes study (MOS), that those with mild and severe insomnia (defined in terms of frequency of symptoms over a 4-week period) scored significantly lower on all domains of the SF-36 relative to a mild hypertension reference group. More importantly, however, the magnitude and distribution of HRQoL decrements of the severe insomnia group were comparable to individuals with clinical depression and congestive heart failure (see Fig. 1), even after controlling for 16 co-morbid conditions, and various other demographics. This pervasive nature of insomnia fits well with what patients report: sleep disturbance has a knock-on effect on nearly every aspect of daily functioning,[13] whereas many other conditions are more selective in their consequences.

One final line of evidence to support the view that disturbed sleep can have a bearing on aspects of HRQoL comes from studies looking at the additive effect of poor sleep on existing conditions. In a recent study, 'poor sleep' (measured using the PSQI) was found to

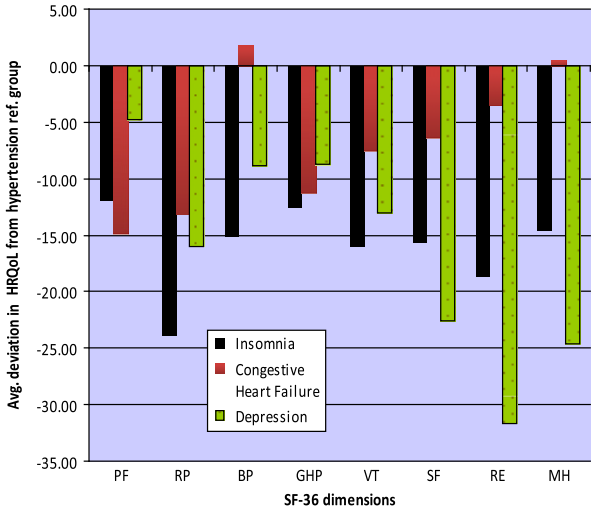


Fig. 1. Deviations in SF-36 scores for Insomnia, depression, and congestive heart failure, relative to hypertension reference group. Scores plotted from[70]. BP, bodily pain; GHP, general health perception; MH, mental health; PF, physical functioning; RE, role-emotional; RP, role-physical; SF, social functioning; VT, vitality/energy.

be a significant independent predictor of mental and physical health component scores (from the SF-36) in patients with multiple sclerosis.[86] Fortner et al.[87] again using the PSQI, categorized breast cancer patients into 'good' and 'bad' sleepers; finding that 'bad' sleepers scored lower on six (role-physical, bodily pain, vitality, social functioning, role-emotional, and mental health) of the eight SF-36 dimensions. Other studies using more concrete definitions of insomnia reveal a similar pattern. For example, Caap-Ahlgren and Delvin[88] reported that Parkinson's disease patients with insomnia had impaired HRQoL on every dimension of the SF-36, relative to those without insomnia symptoms. Rumble et al.[89] also demonstrated lower scores in lung cancer patients meeting criteria for insomnia on the 'global health/quality of life' 2-item subscale scale from the European Organization for Research and Treatment of Cancer Quality of Life Questionnaire (EORTC QLQ-C30),[90] a cancer-specific HRQoL questionnaire, relative to a control group of cancer patients without insomnia.

Overall, cross-sectional studies highlight the pervasive impact of insomnia on aspects of functioning and HRQoL, and this relationship holds, to varying degrees, after controlling for co-morbidities. Well-screened PIs appear to have more selective impairments relative to those individuals in the population based and clinical studies, which might be expected given the reported (and unreported) co-morbidities in the latter groups. There is some evidence to suggest that individuals with co-morbid insomnia experience greater/entrenched daytime functioning impairments relative to PI patients.[78] Crucially, both disease-specific and generic instruments appear to be sensitive to HRQoL impairments.

Does improving insomnia also improve aspects of HRQoL?

Because health-related functional impairments are prevalent within insomnia populations, and enshrined in the diagnostic criteria, treatment should ultimately target and alleviate such impairments. That is, improving sleep should improve functioning (this of course is based on the notion that impaired sleep is causally related to reduced HRQoL). Surprisingly, very few controlled studies (see Table 2) have included assessments of HRQoL (see[91] for a review of more general daytime parameters), and only two have specified HRQoL as the primary outcome measure. Table 2 specifies the following: all controlled studies of insomnia treatment that include an HRQoL or QoL measure; two additional uncontrolled trials of insomnia treatment, specifying HRQoL as one of the primary outcome variables of interest; and finally two large RCTs of CBT in those with insomnia and cancer (selected only as examples of how treating insomnia can modify HRQoL variables pertinent to the co-occurring illness). All papers were sourced by searching major search engines (*PubMed, ISI Web of Knowledge, ScienceDirect*), and through hand-picking citations from existing published articles.

Cross-sectional and uncontrolled studies

Leger et al.[92] conducted a cross-sectional study, selecting patients with insomnia who had been on zopiclone for at least twelve months, and compared them with a matched group of good sleepers, on a study specific QoL instrument. 'Quality of life' was assessed using questions tapping five core domains: work, relationships, safety, domestic activities, and leisure activities. The groups did not differ in terms of sleep disturbance, except that the Zopiclone patients had 'occasional' difficulties falling asleep. It was also found that both groups had comparable scores for each QoL domain. Similarly, using a cross-sectional design, Zammit et al.[65] compared recruited treated insomniacs with a matched group of untreated insomniacs, on the SF-36. They, however, found no

Table 2
Insomnia treatment studies assessing Health-Related Quality of Life as an outcome variable.

	Study	Methods/intervention	Sample size/ characteristics	Instrument used	Sleep outcome	HRQoL outcome
Uncontrolled	Hajak et al. (2002)[93]	Multi-national randomized trial of zolpidem 10 mg 5 nights/ week (remaining 2 nights were given placebo) versus nightly use, for 14 days.	789 PIs (DSM-IV) free from medication at intake.	SF-36	Both discontinuous and continuous groups had similar numbers (59 v. 65%) rating 'much improved' or 'very much improved' on the CGI-II	Improvements on all SF-36 dimensions were similar between groups. No within-subject statistical testing reported.
	Verbeek et al. (2006)[94]	Compared individual CBT with group CBT.	32 PIs (Individual CBT) versus 74 with mix of PI and co-morbid insomnia (group CBT).	Three sub-scales from the SIP (social interactions, alertness/ intellectual functioning, and recreation) and four 'sub-scales' from the RAND-36 ('general health', 'problems at work', 'social occupation', and 'feeling').	Both groups had significantly improved SOL, WASO, TST, and SE, relative to baseline	Composite ratings on both scales significantly improved at 9-month follow-up, for both group and individual CBT.
Prospective randomized controlled trials	Goldenberg et al. (1994)[62]	Multi-national RCT of zopiclone 7.5 mg versus placebo taken nightly for 2 weeks, and on demand for 6 weeks thereafter.	231 received zopiclone; 227 received placebo. Participants had to report two insomnia symptoms for inclusion, and were excluded for co-occurring illness or CNS affecting medication.	QOLI	Zopiclone group had significantly greater improvements in the sleep domain	2 month follow-up: both groups improved on the psychological wellbeing domain and overall score, but treatment group had significantly greater improvements on activity, social, and work/ profession domains.
	Walsh et al. (2000)[97]	RCT of zolpidem 10mg versus placebo (3–5 nights per week for 8 weeks).	163 PIs meeting DSM-IV criteria	SF-36	Treatment group significantly improved on patient global ratings and diary measures of SOL, TST, NAW, and sleep quality	No significant group differences at any time point (4,8 weeks).
	Morin et al. (2005)[102]	Randomized placebo controlled trial of valerian-hops and diphenhydramine.	184 'mild' insomniacs, experiencing initiation and/or maintenance problems for between 2 and 4 times per week for at least a month.	SF-36	Both treatments had a mild hypnotic effect at two-week assessment relative to placebo	Small but significant improvement on the physical component summary score for Valerian compared with placebo group at 4-week assessment point.
	Savard et al. (2005)[107]	RCT of CBT versus wait-list control.	57 females who had undergone treatment for breast cancer, and reported significant insomnia for 6 months (DSM-IV/ICSD-2 and standard quantitative criteria).	Sub-scale assessing 'health/global quality of life' taken from the EORTC QLQC30.	Significant CBT improvements on measures of SOL, WASO and SE post-treatment relative to wait-list controls.	Significantly greater improvements on global 'health/quality of life' scores for the CBT treated patients at post-treatment. Pooled data for both groups revealed maintenance of improvements at 12 months, compared to baseline.
	Scharf et al. (2005)[100]	RCT of Eszopiclone 1 mg, 2 mg or placebo nightly for 2 weeks.	231 Elderly PIs (age range 65–85) meeting DSM-IV criteria.	Q-LES-Q	Significant treatment effects for 2mg on SOL, WASO and TST relative to placebo.	2mg treatment group demonstrated improvements in five domains (physical health, mood, household activities, leisure time

					activities, and medication).
Dixon et al. (2006)[71]	RCT of group CBT versus 'no additional' treatment in a general practice clinical setting.[72]	209 Hypnotic users meeting diagnostic criteria (DSM-IV) for insomnia.	SF-36	CBT group had significantly decreased PSQI global score and lower percentage of group on hypnotics, compared with controls.	CBT group significantly improved on physical functioning, emotional role limitation, and mental health, relative to control group (6 month time adjusted means).
Espie et al. (2007)[99]	RCT of group CBT versus TAU in a clinical setting.	201 Individuals meeting DSMIV/ICSD-2 and quantitative criteria for insomnia.	SF-36	CBT group significantly improved on measures of SOL and WASO at 6 months relative to TAU group	At 6 months: significant improvements in energy/vitality and mental health dimensions (small ES) for CBT versus TAU.
Walsh et al. (2007)[73]	Multi-centred RCT of Eszopiclone 3 mg versus placebo, nightly for six months.	830 PIs	SF-36	SOL, WASO, TST significantly improved relative to placebo at six months (medium to large ES).	Vitality (small to moderate ES), social functioning, physical functioning, and bodily pain (small ES) were all significantly improved relative to placebo group at 6 months.
Espie et al. (2008)[105]	Pramgatic RCT of CBT versus TAU	150 Cancer patients undergoing active cancer treatment, and reporting significant insomnia (meeting standard quantitative criteria for at least three nights in last three months, and daytime functioning impairment).	FACT-G (functional assessment of cancer therapy). sub-scales cover physical, emotional, social and functional well-being.	CBT group significantly improved on measures of SOL, WASO and SE (large ES), relative to TAU.	At 6 months, CBT group signficantly improved on the physical and functional domains (large ES).
Soeffing et al. (2008)[98]	Randomized trial of three-component CBT (stimulus control, sleep hygiene, relaxation) versus sham biofeedback.	47 Hypnotic-dependent older adults.	SF-36	CBT significantly improved on SOL, WASO and SE (medium to large ES) relative to sham control group.	Neither group improved on any dimension of the SF-36, at 4 weeks.
Omvik et al. (2008)[75]	RCT of Zopiclone 7.5 mg versus group CBT versus pill placebo for 6 weeks.[76]	46 Elderly (mean age = 55) adults meeting criteria for PI.	SF-36 & QoLI	Post-treatment: CBT significantly improved relative to placebo and zopiclone groups on (objective) WASO and SWS; both treatment groups had improved objective SE compared with placebo (PSG). CBT group more improved on objective (WASO, SE, SWS) and subjective (WASO) parameters relative to zopiclone group at 6 months.	No evidence of improvements between or within groups, post-treatment or at 6 months follow-up (placebo group not included in analyses).

CBT, Cognitive Behavioural Therapy; DSM-IV, Diagnostic and Statistical Manual of Mental Disorders, 4th Edition; EORTC QLQ-C30, European Organization for Research and Treatment of Cancer Quality of Life Questionnaire; ES, Effect Size; FACT-G, Functional Assessment of Cancer Therapy - General; CGI-II, Clinical Global Impressions Scale; ICSD-2, International Classification of Sleep Disorders, 2nd Edition; ISI, Insomnia Severity Index; NAW, Number of Awakenings; NHP, Nottingham Health Profile; PI, Primary Insomnia; PSG, Polysomnography; PSQI, Pittsburgh Sleep Quality Index; Q-LES-Q, Quality of Life Enjoyment and Satisfaction Questionnaire; QOLI, Quality of Life of Insomniacs Questionnaire; QoLI, Quality of Life Inventory; RCT, Randomised Controlled Trial; SE, Sleep Efficiency; SF-36, Short-Form Health Survey; SIP, Sickness Impact Profile; SOL, Sleep Onset Latency; SWS, Slow-Wave Sleep; TAU, Treatment As Usual; TST, Total Sleep Time; WASO, Wake-time After Sleep Onset.

differences across dimensions between groups. The use of a non-validated questionnaire in the Leger et al. study, and the poor characterization of patients in the Zammit et al. investigation, coupled with the cross-sectional nature of both papers, means these data do not provide strong enough evidence to help elucidate the relationship between HRQoL and treatment of insomnia.

As part of a multi-national study, Hajak et al.[93] compared continuous versus non-nightly zolpidem in a large sample of insomniacs. Both groups had a similar number of 'responders', as determined by the clinical global impression improvement score, and SF-36 improvements were also similar for both groups across all dimensions (non-significant group *x* time effect). However, there was no appropriate placebo/control group, and within-group analyses were not reported on the dimensions; it is therefore hard to tell if and how treatment affected specific domains of HRQoL.

Verbeek et al.[94] reported data from both individual ($n = 32$) and group CBT ($n = 74$) on primary outcomes of both sleep and HRQoL parameters. Across treatment modalities, sleep-onset latency (SOL), wake after sleep-onset (WASO), sleep efficiency (SE), and total sleep time (TST), significantly improved and remained robust at the 9-month post-treatment assessment. To sample HRQoL domains relevant to those with insomnia, the authors selected items probing four domains ('general health', 'problems at work', 'social occupation', and 'feeling') from the RAND-36[d], a practically identical scale to the SF-36, and three subscales (social interactions, alertness/intellectual functioning, and recreation) from the 136-item sickness impact profile (SIP[95]). Both treatment groups showed comparable improvements in 'global' scores of the RAND-36 and SIP, with the SIP demonstrating the most robust effects, at 9-month follow-up. The authors, however, did not detail scores for each subscale, in either the RAND-36 or SIP, making it impossible to identify what specific HRQoL components were most sensitive to the CBT intervention. The lack of a control group also prevents ruling out non-specific effects; it is noteworthy that the intermediate phase before receiving therapy (i.e., between baseline and post-waitlist) had significant positive effects on both sleep and HRQoL measures. The authors believe sleep hygiene advice, provided at the first screening interview, may account for this, although the current literature on sleep hygiene recommendations for insomnia would perhaps argue against this interpretation. A relevant control group seems essential for an accurate interpretation of HRQoL outcomes; the placebo impact of sham CPAP, for example, has been documented in OSA patients, where 'active' and control groups can show comparable improvements across a number of functioning and HRQoL domains.[96]

Prospective controlled trials

A number of prospective controlled trials have included a validated instrument to assess QoL or HRQoL domains. Goldenberg et al.[62] conducted a multi-national RCT of zopiclone versus placebo. Two hundred and thirty one patients were randomized to receive 14 days of zopiclone (and on demand for six weeks thereafter) and 227 received placebo. All patients completed Rombaut et al.'s QOLI. At two weeks, both groups showed improvements in the psychological well-being component and overall global 'quality of life' score, but the zopiclone group demonstrated significantly greater improvements in the sleep, activity, social, and work/profession domains. Relative improvements remained for activity, sleep and social domains at the two-month follow-up.

[d] The Rand-36 is an exact replica of the SF-36 in terms of content, but has different scoring algorithms for the Bodily pain and General Health sub-scales.

Walsh et al.[97] conducted an 8 week RCT of zolpidem 10 mg versus placebo (3–5 nights per week) in 163 PIs. Although sleep parameters improved significantly post-treatment in the experimental group, there was no corresponding change on any dimension of the SF-36. Dixon et al. in their analysis of SF-36 outcome data from 209 individuals on hypnotics (with continuing insomnia) randomized to either CBT or control, found simultaneous improvements in sleep and HRQoL, relative to a control group. PSQI global scores significantly decreased, as too did the percentage of hypnotic users in the experimental group. Time-weighted mean adjustments across the 6-month follow-up period revealed significant improvements in physical functioning, emotional role limitation, and mental health, relative to the control group. On closer inspection however, these 'improvements' do appear to be largely mediated by declining functioning in the control group (e.g., up to a 16 point *decrease* for the emotional role dimension). Regardless, the data show an overall tendency for the CBT group to improve, albeit marginally, and the control group to experience further impairment. CBT improvements, in this particular population, might then be argued to act as an important buffer, preventing further (likely) decrements in HRQoL.

More recently, Soeffing et al.[98] reported data from a randomized trial of a three-component cognitive behavioural intervention (stimulus control, relaxation, and sleep hygiene) or sham biofeedback (psychological placebo), in a small sample of hypnotic-dependent older adults ($n = 47$). At post-treatment, significant effects were found for the CBT treated group in terms of subjective sleep parameters (SOL, WASO and SE; medium to large effects) but there was no corresponding improvement in SF-36 scores (mean dimension/component scores were not reported). Indeed, there was no improvement in any of the other daytime assessments probing sleepiness, fatigue, anxiety or mood. The rather early post-treatment assessment could perhaps undermine improvements, particularly using the SF-36 instrument which has a 4-week recall period.

Espie et al. [99] conducted an RCT of CBT versus treatment as usual (TAU) in general practice, using trained nurses as CBT therapists. At the 6-month follow-up, small to moderate effect sizes were found for SOL and WASO relative to controls; and a medium effect size (ES) for improvement in sleep efficiency. Sleep improvements were also accompanied by small, but significant improvements in the energy/vitality and mental health subscales of the SF-36, relative to the TAU group. Noteworthy however, these follow-up improvements are still substantially lower than normative reference values, which again is likely to be mediated by the 'real world' clinical context.

Walsh et al.[73] in their large RCT of Eszopiclone versus placebo, assessed HRQoL and daytime functioning as primary outcome measures. Changes in sleep parameters were significantly larger in the treatment group for SOL, WASO and TST (medium to large ES) relative to the placebo group at the 6-month follow-up, and 50% scored ≤ 7 on the (Insamna severity index compared with 19% in the placebo group). In terms of HRQoL, SF-36 domains of vitality, social functioning, physical functioning, and bodily pain were all significantly improved relative to the placebo group at six months (small to moderate ES for the vitality domain, and small ES for the rest of the domains). Although the authors report 'no change' in terms of sleep variables during the discontinuation period (i.e., sleep gains were maintained), they do not report data on HRQoL parameters. This is likely to be because of the short 2-week interval, coupled with the SF-36 recall period; nevertheless, it would be interesting to see if these improvements are maintained beyond 'active' treatment.

In the original study by Sivertsen et al.[76] the authors showed that PI patients treated with CBT improved in both objective and

subjective sleep parameters, relative to a zopiclone treatment group, and that these relative improvements remained robust at 6-month follow-up. In a recent follow-up report on daytime functioning measures from this trial, Omvik et al.[75] failed to find any evidence of impairment (at baseline) relative to normative values, using the SF-36 and the QoLI. Not surprisingly, then, they also failed to find a significant group interaction effect, with neither treatment improving mental or physical health component scores (a possible ceiling effect), at post-treatment follow-up. Again, however, they did not report dimension values, and so subtle component effects may be masked. One interesting finding, when groups were collapsed, was a significant association between pre-post increases in QoLI scores and increases in slow-wave sleep. This does point to a potential relationship between objective sleep and relevant quality of life variables.

Another study[100] used a generic QoL measure, the Quality of Life enjoyment and satisfaction questionnaire (Q-LES-Q[101]), comprising 16 separate dimensions (both health and non-health). Scharf and co-workers reported significant improvements in five domains (physical health, mood, household activities, leisure time activities, and medication) in a group of elderly PIs treated with Eszopiclone 2 mg, relative to a placebo group. These were accompanied by improvements in SOL, WASO and TST compared with placebo. Final assessment was however short at two-weeks, and the authors do not present baseline or post-treatment means, preventing comparison with norms or other clinical groups.

Morin et al.[102] conducted a randomized-placebo controlled trial of valerian-hops and diphenhydramine, on a group of 'mild' insomniacs ($n = 184$; experiencing difficulties in initiating and/or maintaining sleep for between 2 and 4 times per week, in the last month). Relatively mild hypnotic effects were found with both treatments, particularly at the two-week assessment. In the comparison between valerian and placebo at four weeks, the valerian group showed a small but significant relative improvement on the physical component summary of the SF-36. Dimensions scores were not presented and the mental health component summary failed to reveal any improvements. The limited and short-lived improvements in sleep may account for the marginal improvements in HRQoL, coupled with the fact that patients were not required to self-report daytime dysfunction on study entry, perhaps creating a ceiling effect, and subsequently making it harder to detect noticeable changes in functioning post-intervention.

In line with contemporary conceptualizations of insomnia as a potentially 'co-occurring' phenomenon, and not merely a secondary 'nuisance' symptom,[103,104] recent studies have examined the impact of treating insomnia within the context of other disorders on aspects of HRQoL. For example, Espie et al.[105] recently conducted a pragmatic RCT of CBT versus treatment as usual (TAU) in cancer patients ($n = 150$), who had completed active cancer treatment but also reported significant insomnia. At 6-months follow-up, the CBT group evidenced significant improvements in measures of SOL, WASO, and SE, with corresponding large effects, relative to the TAU group. The authors also included a measure of cancer-related Quality of Life, the Functional Assessment of Cancer Therapy-General (FACT-G),[106] assessing physical, emotional, social and functional domains of the cancer experience. At 6-months the CBT group demonstrated significant improvements in both the physical and functional domains (large effect sizes). The potential limitation of this scale, in the context of insomnia and HRQoL, is the inclusion of sleep items, which interestingly feature only in the subscales that showed statistical improvements. The authors also note that changes in SE and changes in statistically significant HRQoL domains were low. Results from a similar study,[107] adopting an efficacy approach, comparing CBT with a wait-list control, also demonstrated strong improvements in sleep parameters. The authors included the 'global health/quality of life' subscale from the EORTC QLQ-C30, which revealed significant and enduring improvements in treated patients from baseline to 12 month follow-up. Other sub-scales of this measure were not reported (including social, emotional, physical and cognitive functioning).

Reflections on existing insomnia-HRQoL treatment literature

From the limited treatment studies it is clear that improving sleep, in some cases, can lead to *statistical* improvements in aspects of HRQoL. However, what is far from clear is whether these improvements are *clinically meaningful:* do they really matter to the patient*?* For the most part, improvements are small and/or fall short of normative values, though this may be dependent on the particular population under investigation. Follow-up assessments have typically been short, with most occurring about 6-months post-treatment - it is possible that improvements in functioning become apparent well after sleep parameters have stabilized, particularly with CBT. The converse may also occur in those treated with hypnotics: initial improvements may not be sustained after discontinuation. Relationships between sleep parameter changes and HRQoL improvements are rarely assessed (or reported), and no study has yet documented adequate improvement in HRQoL in primary insomnia patients, using Cognitive Behavioural techniques.

Moreover, to our knowledge, no comparative controlled study of CBT versus hypnotics exists that demonstrates superior HRQoL outcomes in favour of a particular treatment modality (only one relevant RCT[75,76] has included a HRQoL measure). Comparative treatment studies (and intervention studies more generally), probing changes in HRQoL, are important because of their implications for cost-effectiveness and utility models. HRQoL scores are typically used in the calculation of Quality Adjusted life Years (QALYs) – a weighted product of life expectancy and quality of remaining life years – which can then be combined with intervention costs to produce a cost-utility ratio. This cost/QALY ratio (i.e., the difference between the costs of two interventions divided by the difference in QALYs gained) then helps establish costs per QALY for a particular insomnia intervention, relative to say non-treatment, competing insomnia treatments, and other interventions for other common medical problems.[108] The idea being that relatively inexpensive interventions (i.e., low cost per QALY) are then prioritized in terms of resource allocation, over more expensive ones. With the contemporary focus on the burden of insomnia it is clear that future intervention research must include, and pay attention to, the HRQoL concept, in order to document associated morbidity as well as utility of treatment.[109,110]

However, as a field we also need to strive for a more sophisticated understanding and conceptualization of QoL and HRQoL, as it relates to insomnia. From the studies reviewed, only a select few actually defined what they were attempting to measure (and these were typically cross-sectional or scale development/validation studies). Moreover, many refer to assessing or improving *'quality of life'* when they were actually measuring aspects of HRQoL, using a generic health status measure. It may well be true that treating insomnia does improve *'quality of life'*, but where specialised measures such as the SF-36, NHP, SIP, or even the HD-16 are used, it may be more appropriate to emphasise the measurement of health-related QoL.

The key strength of generic measures, like the SF-36, is the ability to compare scores across disease states and with normative community reference values, in a standardized manner. Such data, of course, are vital for cost-effectiveness analyses when, for example, comparing treatments, and assessing whether patients return to 'normal' status. The sacrifice, however, is the poor specificity to a particular disorder. The inclusion of non-specific domains may

dampen the sensitivity of a measure to detect change[111]; this is especially true when only summary scores are reported. Future studies should report both overall summary scores and dimension scores, particularly in light of literature questioning the independence of the mental and physical health components and their representation of individual profile scores (e.g.,[112]). Indeed, for these reasons it is recommended that intervention studies include both disease and generic HRQoL measurement – with the former typically being more sensitive to change than the latter.[113] This approach has achieved some success in the sleep apnea field[114]; and is the recommended perspective by recent insomnia expert consensus workgroups.[4]

Currently, there is no widely used insomnia-specific measure; attempts to pilot and develop new instruments, and investigate the validity/sensitivity of the recently published HD-16, should be considered an important research goal. In the absence of specific measures, researchers need to consider using validated scales that tap dimensions relevant to the insomnia experience. Qualitative work from the Pittsburgh group[115] and more recently our own group,[13] reveal the *nature* of insomnia-related functional impairments – specifically in aspects of cognition, occupational functioning, social/interpersonal relationships, and limitations in goal attainment. It is interesting that the SF-36 does not adequately assess any of these dimensions; even the social functioning scale of the SF-36 simply probes the extent and frequency of interference without detailing what that interference may be. In the case of insomnia this interference is likely to be multi-factorial – from rescheduling activities, failing to schedule or commit in the first place, cancelling at the last minute, or not enjoying social interaction when present.

Another related issue, again concerning the SF-36, is the construct validity of the energy/vitality domain. This dimension resonates best among sleep-disordered patients, particularly individuals with insomnia, capturing fatigue-related symptoms common to sleep loss/fragmentation. This however is the problem; the dimension may simply be measuring fatigue. Scale development and evaluation studies typically use the vitality sub-scale as a check for concurrent validity, revealing high correlations with instruments such as the fatigue severity scale.[116] The danger of course being, that studies (cross-sectional and treatment) may confuse assessing or improving HRQoL impairment, with modifications in levels of fatigue. This may prove problematic when, for example, collapsing scores into a physical component summary, subsequently leading the author to conclude that 'x treatment improves HRQoL'. A solution to this issue is to always document all measured dimensions, and include, and correlate, validated measures of fatigue with the vitality dimension, to see if there is a substantial overlap in variance.

A final note on generic measurement concerns the explicit focus on *'health'* and *'illness'* state terminology. The SF-36 probes the limiting impact of *'health'* on aspects of functioning; it is, after all, a health status measure. It is not clear if those with insomnia, particularly primary insomnia, actually consider poor/disturbed sleep to represent a change or variation in health state (it's not uncommon for an individual with PI to proclaim, when searching for an underlying causal factor for why they can't sleep, that they are *'healthy'* and otherwise have a good life). In comparison with other disorders, that perhaps have a more direct *health*-link, insomnia impairment may be more difficult to reliably document, and thus show improvements post-intervention, using generic health status tools. This may explain HRQoL discrepancies between clinical, pragmatic studies, and those where PIs are recruited through media adverts.

Beyond generic measurement: future directions

Many questions remain unanswered about the nature of HRQoL in insomnia. The more basic ones, such as what are the predictors/mechanisms of HRQoL impairment and subsequent improvement will become apparent as researchers increasingly investigate insomnia as a 24-h disorder. Some possible targets include: sleep parameters; daytime symptoms like fatigue, mood, and neurocognitive impairment; dysfunctional beliefs; cognitive biases; and objective markers of the stress system. Longitudinal studies on the natural evolution of insomnia, and the transition between states (i.e., good sleepers, acute insomnia, chronic insomnia), will also prove helpful in better defining causal relations between insomnia and HRQoL.

In relation to assessment and measurement, the scope of this review, a prospective research agenda should be initiated on adapting and creating new instruments that adequately probe the insomnia experience. One potential avenue, in addition to generic measurement, is to adopt a supplemental modular approach[117] to functional HRQoL assessment. That is, consider important domains that are commonly reported to be affected by insomniacs, but are not necessarily dealt with comprehensively by generic instruments. For example, occupational functioning is cited as a potential daytime consequence in the diagnostic criteria, yet little work has actually focused on this domain specifically. Encouragingly, David and Morgan[118] recently created and validated the Occupational Impact of Sleep questionnaire (OISQ), a scale that probes the impact of sleep quality on a number of work related areas. Initial data collected on the OISQ shows it has good discriminant ability to distinguish between PIs and good sleepers, and, more recently, significant correlations were found with global sleep quality (as measured by the PSQI) and sleepiness (ESS) in a Dutch sample of office workers.[119]

In this context, two recent pharmacotherapy studies are also worth mentioning.[73,120] Walsh et al.[73] assessed work limitations, using the work limitations questionnaire (WLQ[121]), in an RCT of Eszopiclone versus placebo. Baseline data showed lower scores on all domains of the instrument (time demands, physical demands, mental-interpersonal demands, output demands, and work productivity loss) relative to normative values, and somewhat comparable scores to those suffering from clinical depression. Importantly, all domains showed a relative improvement in the treatment group versus placebo, across the six-month treatment phase. Erman et al.[120] similarly assessed workplace functioning in PI patients ($n = 752$) taking part in a randomized controlled trial of Zolpidem versus placebo. The authors analyzed data from two components of the WLQ, the 'time' and 'output' sub-scales. Again, baseline values were more impaired (approximately three times greater) than normative healthy controls. Significant effects of treatment emerged after just 12 weeks, which were sustained at the 6-month follow-up, and improvements were related to global sleep changes. These data are important because they not only highlight the burden of insomnia on the workplace beyond traditional measures of absenteeism – confirming qualitative reports[122] and population based studies[41] – but also demonstrate the ability to achieve simultaneous improvements in sleep and occupational functioning. Cognitive behavioural interventions should include a similar assessment of occupational functioning in future treatment trials.

As well as the development and use of relevant instruments, researchers should also consider the merit of applying new methods and approaches to assessing insomnia-related impairment. For example, the use of ecological momentary assessment[11,12] to collect daily diary ratings of common insomnia symptoms might provide a new way to track daytime outcomes during treatment, and post-treatment, as well as establishing relationships with nightly sleep parameters. There is no reason why this method can't be adapted to assess, for example, social functioning, or subjective workplace performance. A 'significant other' perspective (qualitative and/or quantitative) would also be a useful source of information to gauge how sleep, and appropriate treatment, affects domains of HRQoL. To our knowledge no study has used this resource to probe daytime

functioning and insomnia specifically. This also raises the issue of relationship functioning and insomnia. It has been reported that individuals with insomnia have less satisfying interpersonal relationships,[10] and although it is unlikely to follow a simple cause-and-effect model (for a review see[123]), sleep quality may exert a mediating effect. It would be interesting to see if treating insomnia can also improve marital/relationship quality; certainly, improvements in marital satisfaction have been reported in sleep apnea patients after initiation of CPAP therapy.[124]

Global quality of life has been viewed pragmatically in the literature as a multi-dimensional construct, assessed using commonly reported domains deemed important. The likelihood, however, is that QoL means different things for different people – a uniquely personal perception[49] – making it difficult to accurately measure at the group level, and especially through modular assessment. One rather simple, but valid method might be to ask individuals to rate their quality of life on a likert item or visual-analogue scale. It could be argued that on making this judgment, individuals are factoring in all the domains/life areas that are important to them. This single-item format has proved sensitive when documenting, for example, how many people within a sample of insomniacs report a 'very good', 'good', or 'poor' quality of life, compared to those without sleep-complaints.[10,66] A similar approach could be tested alongside generic and modular assessment in treatment studies; single item visual-analogue scales have been shown to be valid, reliable and responsive to change in patient groups.[125] A related methodological approach would be to tailor items asking patients to report whether they think their QoL has *improved* as a result of treatment; instead of simply trying to demonstrate *reductions in functional impairments,* as generic measures typically do. The Patient Global Impression improvement scale, developed from the Clinical Global Impressions scale,[126] is used for a similar purpose, allowing patients to report the extent to which their overall illness symptoms have improved post-treatment. Such an instrument could be modified to assess perceived improvements in focused areas of functioning as well as subjective perceptions of global quality of life.

It may also be worthwhile directing efforts towards gathering basic qualitative data on the individual experience of treatment. We[13] and others[115,127] have successfully applied qualitative methods to understanding the daytime insomnia experience, revealing novel insights on the nature of functional impairments. Similar methodologies (semi-structured interviews, audio-diaries) could be applied to assessing treatment outcomes, as well as the treatment process itself. The SAQLI[83] includes a section looking at negative side effects of CPAP treatment in OSA patients; it is not yet clear what, if any, impairments in HRQoL are incurred *during* CBT for insomnia (stimulus control and sleep restriction would be likely candidates for study). While clinicians may be familiar with how sleep improvements influence patient well-being, systematic study of the patient narrative may prove helpful in understanding the treatment process (side effects, locus of improvement, mechanisms of change, adherence), and the net impact of treatment on daytime functioning and subjective quality of life (at different time-points).

The importance of measuring items that are relevant, not just to the disorder, but to the individual has been noted in the QoL and HRQoL literature,[49,57,128] and, more recently, by sleep researchers.[75,114] We are currently piloting a mixed method, patient-centred approach[129,130] to the assessment of insomnia-related quality of life (IRQoL) at the Glasgow sleep centre. By IRQoL we mean *'the impact of insomnia on aspects of life that are most salient to the individual, as reported by the individual'*. Specifically, patients are asked to generate, using their own words, the most important areas of their life that are affected by sleep disturbance. These areas are then ranked in terms of importance, and rated in relation to extent of interference over a defined time interval. Items can be assessed pre and post-treatment, within- and between-subjects. This idiographic approach permits assessment of areas that are important to the individual, which is likely to be a lot more sensitive to change given that we are enhancing the signal (relevance) and reducing the noise (non-relevance) associated with generic instruments. Moreover, it has been commented previously[4,85,114] that the major obstacle to creating an insomnia-specific QoL questionnaire is the high degree of co-morbidity. This is certainly the case when conducting pragmatic effectiveness trials, or in clinical settings, where other illnesses may mediate scores on HRQoL instruments. The strength of our individualized approach is that it emphasizes the attribution to sleep, and therefore, it should be theoretically possible to isolate the subjective impact of an intervention on sleep only, even in the context of other illnesses.

Conclusion

It is clear that insomnia does have a measurable negative impact on domains of HRQoL, and that these impairments are not simply limited to obvious domains, like vitality and energy, but also extend to other aspects of mental, social, and physical functioning. Comparisons with other illnesses, linear trends with insomnia severity, and additive effects of insomnia beyond a primary/co-occurring illness, all support and strengthen this perspective. Although research into HRQoL is in its infancy within the insomnia literature, there is already emerging data that successful treatment can improve functioning across a number of domains. Of course, to what extent group mean improvements are important to individual patients, and their daily lives, remains an unanswered question. Future research in this area should attempt to approach the issue of how insomnia treatment improves functioning and individual quality of life, by using new innovative methods and instruments. Such a task is not a trivial undertaking; the impact of insomnia on the individual[13] and society[131] is very real, and authors have suggested that the pathway between insomnia and depression, for example, may be mediated, in part, through reduced HRQoL.[132]

In order to improve the quality of future research, outcome studies should include a generic measure of HRQoL as well as other measures that are relevant to the insomnia experience, and have proven sensitivity. When reporting results, authors should specify baseline scores and their relationship with normative values, detail each profile score as well as component/global summary scores, and make some attempt at investigating the relationship between sleep improvements and HRQoL changes (i.e., correlation analysis and/or responders versus non-responders, based on ISI category change, or quantitative sleep variables).

Practice points

- HRQoL and QoL are not synonymous constructs.
- Insomnia negatively affects diverse aspects of HRQoL
- The limited treatment data tentatively suggest that successfully improving sleep, using both pharmacological and non-pharmacological interventions, can lead to significant improvements in domains of HRQoL.
- There is a great need to standardize measurement use and reporting of scores to facilitate future comparisons and meta-analyses across studies and treatment modalities.
- HRQoL measurement contributes to cost-effectiveness models and therefore plays a pivotal role in service provision and development.

Research agenda

- Include measures of HRQoL in future cross-sectional, natural evolutional, and interventional studies.
- Compare different symptom (initiating, maintenance, mixed) and diagnostic (idiopathic, psychophysiological) sub-types of insomnia on measures of HRQoL.
- Compare different treatment modalities on HRQoL primary outcomes within the same study.
- Consider assessing domains that are under-researched, yet part of the insomnia experience, such as social, occupational and relationship functioning.
- Follow-up HRQoL outcomes for longer durations and consider using new informative methods that capture the patient perspective e.g., qualitative research methodologies.
- Investigate relationships between sleep parameters (objective and subjective) and HRQoL dimensions.
- Identify non-sleep predictor variables of HRQoL impairment and change.
- Consider the effect of the insomnia treatment process on HRQoL.
- Measure the impact of treating insomnia in the context of co-morbid illness (e.g., cancer, depression, pain), on HRQoL.
- Develop and validate measures of insomnia-related quality of life that are grounded in the words of patients.

References

1. Greene G. *Insomniac*. Berkeley: University of California Press; 2008.
2. *International classification of sleep disorders: diagnostic and coding manual*. 2nd ed. Westchester, IL: American Academy of Sleep Medicine; 2005.
3. *Diagnostic and statistical manual for the assessment of mental disorders*. 4th ed. Washington, DC: American Psychiatric Association; 1994.
4. *Buysse DJ, Ancoli-Israel S, Edinger JD, Lichstein KL, Morin CM. Recommendations for a standard research assessment of insomnia. *Sleep* 2006; **29**:1155–73.
5. Morin CM. Measuring outcomes in randomized clinical trials of insomnia treatments. *Sleep Medicine Reviews* 2003;**7**:263–79.
6. *NIH state-of-the-science conference statement on manifestations and management of chronic insomnia in adults*. National Institutes of Health; 2005.
7. *Armstrong D, Lilford R, Ogden J, Wessely S. Health-related quality of life and the transformation of symptoms. *Sociol Health Illn* 2007;**29**:570–83.
8. Edinger JD, Bonnet MH, Bootzin RR, Doghramji K, Dorsey CM, Espie CA, et al. Derivation of research diagnostic criteria for insomnia: report of an American Academy of Sleep Medicine Work Group. *Sleep* 2004;**27**:1567–96.
9. Moul DE, Nofzinger EA, Pilkonis PA, Houck PR, Miewald JM, Buysse DJ. Symptom reports in severe chronic insomnia. *Sleep* 2002;**25**:553–63.
10. Roth T, Ancoli-Israel S. Daytime consequences and correlates of insomnia in the United States: results of the 1991 National Sleep Foundation survey. II. *Sleep* 1999;**22**:S354–8.
11. Buysse DJ, Thompson W, Scott J, Franzen PL, Germain A, Hall M, et al. Daytime symptoms in primary insomnia: a prospective analysis using ecological momentary assessment. *Sleep Med* 2007;**8**:198–208.
12. Levitt H, Wood A, Moul DE, Hall M, Germain A, Kupfer DJ, et al. A pilot study of subjective daytime alertness and mood in primary insomnia participants using ecological momentary assessment. *Behav Sleep Med* 2004;**2**:113–31.
13. Kyle SD, Espie CA, Morgan K. A qualitative analysis of daytime functioning and quality of life in persistent insomnia using focus groups and audio-diaries. *Sleep* 2008;**31**(Abstract Suppl.):742.
14. Lichstein KL, Means MK, Noe SL, Aguillard RN. Fatigue and sleep disorders. *Behav Res Ther* 1997;**35**:733–40.
15. Orff HJ, Drummond SPA, Nowakowski S, Perlis ML. Discrepancy between subjective symptomatology and objective neuropsychological performance in insomnia. *Sleep* 2007;**30**:1205–11.
16. Varkevisser M, Kerkhof GA. Chronic insomnia and performance in a 24-h constant routine study. *J Sleep Res* 2005;**14**:49–59.
17. Riedel BW, Lichstein KL. Insomnia and daytime functioning. *Sleep Med Rev* 2000;**4**:277–98.
18. Sanford SD, Lichstein KL, Durrence HH, Riedel BW, Taylor DJ, Bush AJ. The influence of age, gender, ethnicity, and insomnia on Epworth sleepiness scores: a normative US population. *Sleep Medicine* 2006;**7**:319–26.
19. Day R, Guido P, Helmus T, Fortier J, Roth T, Koshorek G, et al. Self-reported levels of sleepiness among subjects with insomnia. *Sleep Med.* 2001;**2**: 153–7.
20. Fulda S, Schulz H. Cognitive dysfunction in sleep disorders. *Sleep Med Rev* 2001;**5**:423–45.
21. Edinger JD, Means MK, Carney CE, Krystal AD. Psychomotor performance deficits and their relation to prior nights' sleep among individuals with primary insomnia. *Sleep* 2008;**31**:599–607.
22. Altena E, Van Der Werf YD, Strijers RLM, Van Someren EJW. Sleep loss affects vigilance: effects of chronic insomnia and sleep therapy. *J Sleep Res* 2008;**17**:335–43.
23. Harvey AG. A cognitive model of insomnia. *Behav Res Ther* 2002;**40**: 869–93.
24. Bastien CH, Fortier-Brochu E, Rioux I, LeBlanc M, Daley M, Morin CM. Cognitive performance and sleep quality in the elderly suffering from chronic insomnia: relationship between objective and subjective measures. *J Psychosom Res* 2003;**54**:39–49.
25. Winkelman JW, Buxton OM, Jensen JE, Benson KL, O'Connor SP, Wang W, et al. Reduced brain GABA in primary insomnia: preliminary data from 4T proton magnetic resonance spectroscopy (1H-MRS). *Sleep* 2008;**31**:1499–506.
26. Fortier-Brochu E, Beaulieu-Bonneau S, Ivers H, Morin CM. Insomnia and neuropsychological performance: a meta-analysis. *Sleep* 2008;**31**(Abstract Suppl.):687.
27. Backhaus J, Junghanns K, Born J, Hohaus K, Faasch F, Hohagen F. Impaired declarative memory consolidation during sleep in patients with primary insomnia: influence of sleep architecture and nocturnal cortisol release. *Biol. Psychiatry* 2006;**60**:1324–30.
28. Nissen C, Kloepfer C, Nofzinger EA, Voderholzer U, Riemann D. Impaired sleep-related memory consolidation in primary insomnia. *Sleep* 2006;**29**:681.
29. Riemann D, Voderholzer U, Spiegelhalder K, Hornyak M, Buysse DJ, Nissen C, et al. Chronic insomnia and MRI-measured hippocampal volumes: A pilot study. *Sleep* 2007;**30**:955–8.
30. Altena E, Vrenken H, Van Der Werf YD, Van Den Heuvel OA, Van Someren EJW. Structural consequences of chronic insomnia: a voxel-based morphometric study. *J Sleep Res* 2008;**17**(Suppl. 1):O19.
31. Altena E, Van Der Werf YD, Sanz-Arigita EJ, Voorn TA, Rombouts SARB, Kuijer JPA, et al. Prefrontal hypoactivation and recovery in insomnia. *Sleep* 2008;**31**:1271–6.
32. Bruder GE, Towey GP, Stewart JW, Friedman D, Tenke C, Quitkin FM. Event-related potentials in depression: influence of task, stimulus hemifield and clinical features on P3 latency. *Biol. Psychiatry* 1991;**30**:233–46.
33. Szelenberger W, Niemcewicz S. Event-related current density in primary insomnia. *Acta Neurobiologiae Experimentalis* 2001;**61**:299–308.
34. Bastien CH, St Jean G, Morin CM, Turcotte I, Carrier J. Chronic psychophysiological insomnia: hyperarousal and/or inhibition deficits? An ERPs investigation. *Sleep* 2008;**31**:887–98.
35. Devoto A, Manganelli S, Lucidi F, Lombardo C, Russo PM, Violani C. Quality of sleep and P300 amplitude in primary insomnia: a preliminary study. *Sleep* 2005;**28**:859–63.
36. Turcotte I, Bastien CH. Is quality of sleep related to the N1 and P2 ERPs in chronic psychophysiological insomnia sufferers? *Int J Psychophysiol* 2009;**72**:314–22.
37. Leger D, Guilleminault C, Bader G, Levy E, Paillard M. Medical and socio-professional impact of insomnia. *Sleep* 2002;**25**:625–9.
38. Simon GE, VonKorff M. Prevalence, burden, and treatment of insomnia in primary care. *Am J Psychiatry* 1997;**154**:1417–23.
39. Hatoum HT, Kong SX, Kania CM, Wong JM, Mendelson WB. Insomnia, health-related quality of life and healthcare resource consumption - A study of managed-care organisation enrollees. *Pharmacoeconomics* 1998;**14**:629–37.
40. Leger D, Massuel MA, Metlaine A, Sisyphe SG. Professional correlates of insomnia. *Sleep* 2006;**29**:171–8.
41. Daley M, Morin CM, LeBlanc M, Grεgoire JP, Savard J, Baillargeon L. Insomnia and its relationship to health-care utilization, work absenteeism, productivity and accidents. *Sleep Med* 2009;**10**:427–38.
42. Sivertsen B, Overland S, Neckelmann D, Glozier N, Krokstad S, Pallesen S, et al. The long-term effect of insomnia on work disability – the HUNT-2 historical cohort study. *Am J Epidemiol* 2006;**163**:1018–24.
43. Riemann D, Voderholzer U. Primary insomnia: a risk factor to develop depression? *J Affect Disord* 2003;**76**:255–9.
44. Neckelmann D, Mykletun A, Dahl AA. Chronic insomnia as a risk factor for developing anxiety and depression. *Sleep* 2007;**30**:873–80.
45. Stepanski E, Koshorek G, Zorick F, Glinn M, Roehrs T, Roth T. Characteristics of individuals who do or do not seek treatment for chronic insomnia. *Psychosomatics* 1989;**30**:421–7.
46. Morin CM, LeBlanc M, Daley M, Gregoire JP, Mθrette C. Epidemiology of insomnia: prevalence, self-help treatments, consultations, and determinants of help-seeking behaviors. *Sleep Med* 2006;**7**:123–30.

* The most important references are denoted by an asterisk.

47. Henry D, McClellen D, Rosenthal L, Dedrick D, Gosdin M. Is sleep really for sissies? Understanding the role of work in insomnia in the US. *Social Sci Med* 2008;**66**:715–26.
48. Testa MA, Simonson DC. Current concepts – assessment of quality-of-life outcomes. *N Engl J Med* 1996;**334**:835–40.
*49. Gill TM, Feinstein AR. A critical-appraisal of the quality of quality-of-life measurements. *JAMA* 1994;**272**:619–26.
50. Moons P, Budts W, De Geest S. Critique on the conceptualisation of quality of life: a review and evaluation of different conceptual approaches. *Int J Nurs Stud* 2006;**43**:891–901.
51. Dijkers M. ''What's in a name?'' The indiscriminate use of the ''Quality of life'' label, and the need to bring about clarity in conceptualizations. *International Journal of Nursing Studies* 2007;**44**:153–5.
52. Feinstein A. Clinimetric Perspectives. *Journal of Chronic Diseases* 1987;**40**:635–40.
53. Cummins R. Objective and subjective quality of life: an interactive model. *Soc Ind Res* 2000;**52**:55–72.
54. Herrman H, Metelko Z, Szabo S, Rajkumar S, Kumar S, Vanheck G, et al. Study protocol for the World-Health-Organization project to develop a quality-of-life assessment instrument (WHOQoL). *Qual Life Res* 1993;**2**:153–9.
55. Ruta D, Camfield L, Donaldson C. Sen and the art of quality of life maintenance: towards a general theory of quality of life and its causation. *J Socio Econ* 2007;**36**:397–423.
56. Albrecht GL, Devlieger PJ. The disability paradox: high quality of life against all odds. *Soc Sci Med* 1999;**48**:977–88.
57. Carr AJ, Higginson IJ. Measuring quality of life – are quality of life measures patient centred? *BMJ* 2001;**322**:1357–60.
58. Wood-Dauphinee S. Assessing quality of life in clinical research: from where have we come and where are we going? *J Clin Epidemiol* 1999;**52**:355–63.
59. Spilker B. Introduction. In: Spilker B, editor. *Quality of life assessments in clinical trials*. New York, USA: Raven Press; 1990. p. 3–9.
60. Rombaut N, Maillard F, Kelly F, Hindmarch I. The quality of life of insomniacs questionnaire (QOLI). *Med Scie Res* 1990;**18**:845–8.
61. Kelly F, Delahaye C, Gal M. Quality of life in insomnia – results of validation studies for a specifically designed questionnaire. *JAMA*; 1993:21–6.
62. Goldenberg F, Hindmarch I, Joyce CRB, Legal M, Partinen M, Pilate C. Zopiclone, sleep and health-related quality-of-life. *Human Psychopharmacology – Clinical and Experimental* 1994;**9**:245–51.
63. deSouza JCRP. Quality of life and insomnia in university psychology students. *Human Psychopharmacol – Clinical and Experimental* 1996;**11**:169–84.
64. Ware JE, Sherbourne CD. The Mos 36-item short-form health survey (Sf-36) .1. Conceptual-framework and item selection. *Med Care* 1992;**30**: 473–83.
65. Zammit GK, Weiner J, Damato N, Sillup GP, McMillan CA. Quality of life in people with insomnia. *Sleep* 1999;**22**:S379–85.
66. Hajak G, Sine SG. Epidemiology of severe insomnia and its consequences in Germany. *Eur Arch Psychiatry Clin Neurosci* 2001;**251**:49–56.
67. Leger D, Scheuermaier K, Philip P, Paillard M, Guilleminault C. SF-36: evaluation of quality of life in severe and mild insomniacs compared with good sleepers. *Psychosom Med* 2001;**63**:49–55.
68. Schubert CR, Cruickshanks KJ, Dalton DS, Klein BEK, Klein R, Nondahl DM. Prevalence of sleep problems and quality of life in an older population. *Sleep* 2002;**25**:889–93.
69. LeBlanc M, Beaulieu-Bonneau S, Mɛrette C, Savard J, Ivers H, Morin CM. Psychological and health-related quality of life factors associated with insomnia in a population-based sample. *J Psychosom Res* 2007;**63**:157–66.
*70. Katz DA, McHorney CA. The relationship between insomnia and health-related quality of life in patients with chronic illness. *J Fam Pract* 2002;**51**:229–35.
*71. Dixon S, Morgan K, Mathers N, Thompson J, Tomeny M. Impact of cognitive behavior therapy on health-related quality of life among adult hypnotic users with chronic insomnia. *Behav Sleep Med* 2006;**4**:71–84.
72. Morgan K, Dixon S, Mathers N, Thompson J, Tomeny M. Psychological treatment for insomnia in the management of long-term hypnotic drug use: a pragmatic randomised controlled trial. *British J Gen Prac* 2003;**53**:923–8.
*73. Walsh JK, Krystal AD, Amato DA, Rubens R, Caron J, Wessel TC, et al. Nightly treatment of primary insomnia with eszopiclone for six months: effect on sleep, quality of life, and work limitations. *Sleep* 2007;**30**:959–68.
74. Davidson JR, Aime A, Ivers H, Morin CM. Characteristics of individuals with insomnia who seek treatment in a clinical setting versus those who volunteer for a randomized controlled trial. *Behav Sleep Medicine* 2009;**7**:37–52.
*75. Omvik S, Sivertsen BR, Pallesen S, Bjorvatn BR, Havik OE, Nordhus IH. Daytime functioning in older patients suffering from chronic insomnia: treatment outcome in a randomized controlled trial comparing CBT with Zopiclone. *Behav Res Ther* 2008;**46**:623–41.
76. Sivertsen B, Omvik S, Pallesen S, Bjorvatn B, Havik OE, Kvale G, et al. Cognitive behavioral therapy vs zopiclone for treatment of chronic primary insomnia in older adults – a randomized controlled trial. *JAMA* 2006;**295**:2851–8.
77. Frisch MB, Cornell J, Villanueva M, Retzlaff PJ. Clinical validation of the quality of life inventory: a measure of life satisfaction for use in treatment planning and outcome assessment. *Psychol Assess* 1992;**4**:92–101.
78. Lichstein KL, Durrence HH, Riedel BW, Bayen UJ. Primary versus secondary insomnia in older adults: subjective sleep and daytime functioning. *Psych Aging* 2001;**16**:264–71.
79. Philip P, Leger D, Taillard J, Quera-Salva MA, Niedhammer I, Mosqueda JGJ, et al. Insomniac complaints interfere with quality of life but not with absenteeism: respective role of depressive and organic comorbidity. *Sleep Med* 2006;**7**:585–91.
80. Hunt SM, Mckenna SP, Mcewen J, Backett EM, Williams J, Papp E. A quantitative approach to perceived health-status – a validation-study. *J Epidemiol Community Health* 1980;**34**:281–6.
*81. Leger D, Scheuermaier K, Raffray T, Metlaine A, Choudat D, Guilleminault C. HD-16: a new quality of life instrument specifically designed for insomnia. *Sleep Med* 2005;**6**:191–8.
82. Weaver T, Laizner A, Evans L, Maislin G, Chugh D, Lyon K, et al. An instrument to measure functional status outcomes for disorders of excessive sleepiness. *Sleep* 1997;**20**:835–43.
83. Flemons WW, Reimer MA. Development of a disease-specific health-related quality of life questionnaire for sleep apnea. *Am J Respir Crit Care Med* 1998;**158**:494–503.
84. Lacasse Y, Bureau MP, Series F. A new standardised and self-administered quality of life questionnaire specific to obstructive sleep apnoea. *Thorax* 2004;**59**:494–9.
85. Moul DE, Hall M, Pilkonis PA, Buysse DJ. Self-report measures of insomnia in adults: rationales, choices, and needs. *Sleep Med Rev* 2004;**8**:177–98.
86. Merlino G, Fratticci L, Lenchig C, Valente M, Cargnelutti D, Picello M, et al. Prevalence of 'poor sleep' among patients with multiple sclerosis: an independent predictor of mental and physical status. *Sleep Med* 2009; **10**:26–34.
87. Fortner BV, Stepanski EJ, Wang SC, Kasprowicz S, Durrence HH. Sleep and quality of life in breast cancer patients. *J Pain Symptom Manage* 2002;**24**:II.
88. Caap-Ahlgren M, Dehlin O. Insomnia and depressive symptoms in patients with Parkinson's disease – relationship to health-related quality of life. An interview study of patients living at home. *Arch Gerontol Geriatr* 2001;**32**:23–33.
89. Rumble ME, Keefe FJ, Edinger JD, Porter LS, Garst JL. A pilot study investigating the utility of the cognitive-behavioral model of insomnia in early-stage lung cancer patients. *J Pain Symptom Manage* 2005;**30**:160–9.
90. Aaronson NK, Ahmedzai S, Bergman B, Bullinger M, Cull A, Duez NJ, et al. The European-organization-for-research-and-treatment-of-cancer Qlq-C30-a quality-of-life instrument for use in international clinical-trials in oncology. *J Nat Cancer Insti* 1993;**85**:365–76.
91. Krystal AD. Treating the health, quality of life, and functional impairments in insomnia. *J Clin Sleep Med* 2007;**3**:63–72.
92. Leger D, Janus C, Pellois A, Quera-Salva M, Dreyfus J. Sleep, morning alertness and quality of life in subjects treated with zopiclone and in good sleepers. Study comparing 167 patients and 381 good sleepers. *Eur Psychiatry* 1995;**10**:99S–102S.
93. Hajak G, Cluydts R, Declerck A, Estivill SE, Middleton A, Sonka K, et al. Continuous versus non-nightly use of zolpidem in chronic insomnia: results of a large-scale, double-blind, randomized, outpatient study. *Int Clin Psychopharmacol* 2002;**17**:9–17.
*94. Verbeek IH, Konings GM, Aldenkamp AP, Declerck AC, Klip EC. Cognitive behavioral treatment in clinically referred chronic insomniacs: group versus individual treatment. *Behav Sleep Med* 2006;**4**:135–51.
95. Bergner M, Bobbitt RA, Carter WB, Gilson BS. The sickness impact profile – development and final revision of a health-status measure. *Med Care* 1981;**19**:787–805.
96. Atkeson A, Basner R. Sleep and quality of life in sleep apnea. In: Verster J, Pandi-Perumal S, Streiner D, editors. *Sleep and quality of life in clinical medicine*. Towata, NJ: Humana Press; 2008. p. 79–92.
97. Walsh JK, Roth T, Randazzo A, Erman M, Jamieson A, Scharf M, et al. Eight weeks of non-nightly use of zolpidem for primary insomnia. *Sleep* 2000;**23**:1087–96.
98. Soeffing JP, Lichstein KL, Nau SD, McCrae CS, Wilson NM, Aguillard RN, et al. Psychological treatment of insomnia in hypnotic-dependant older adults. *Sleep Med* 2008;**9**:165–71.
99. Espie CA, MacMahon KMA, Kelly HL, Broomfield NM, Douglas NJ, Engleman HM, et al. Randomized clinical effectiveness trial of nurse-administered small-group cognitive behavior therapy for persistent insomnia in general practice. *Sleep* 2007;**30**:574–84.
100. Scharf M, Erman M, Rosenberg R, Seiden D, McCall V, Amato D, et al. A 2-week efficacy and safety study of eszopiclone in elderly patients with primary insomnia. *Sleep* 2005;**28**:720–7.
101. Endicott J, Nee J, Harrison W, Blumenthal R. Quality-of-life enjoyment and satisfaction questionnaire – a new measure. *Psychopharmacol Bull* 1993;**29**:321–6.
102. Morin CM, Koetter U, Bastien C, Ware J, Wooten V. Valerian-hops combination and diphenhydramine for treating insomnia: a randomized placebo-controlled clinical trial. *Sleep* 2005;**28**:747.
103. Stepanski EJ, Rybarczyk B. Emerging research on the treatment and etiology of secondary or comorbid insomnia. *Sleep Med Rev* 2006;**10**:7–18.

104. Lichstein KL. Secondary insomnia: a myth dismissed. *Sleep Med Rev* 2006;**10**:3–5.
105. Espie CA, Fleming L, Cassidy J, Samuel L, Taylor LM, White CA, et al. Randomized controlled clinical effectiveness trial of cognitive behavior therapy compared with treatment as usual for persistent insomnia in patients with cancer. *J Clin Oncol* 2008;**26**:4651–8.
106. Cella DF, Tulsky DS, Gray G, Sarafian B, Linn E, Bonomi A, et al. The functional assessment of cancer-therapy scale – development and validation of the general measure. *J Clin Oncol* 1993;**11**:570–9.
107. Savard J, Simard S, Ivers H, Morin CM. Randomized study on the efficacy of cognitive-bahavioural therapy for insomnia secondary to breast cancer, part I: sleep and psychological effects. *J Clin Oncol* 2005;**23**: 6083–96.
108. Martin SA, Aikens JE, Chervin RD. Toward cost-effectiveness analysis in the diagnosis and treatment of insomnia. *Sleep Med Rev* 2004;**8**:63–72.
109. Morgan K, Dixon S, Mathers N, Thompson J, Tomeny M. Psychological treatment for insomnia in the regulation of long-term hypnotic drug use. *Health Technol Assess* 2004;**8**:1–68.
110. Morin CM. Insomnia treatment: taking a broader perspective on efficacy and cost-effectiveness issues. *Sleep Med Rev* 2004;**8**:3–6.
111. Hill S, Harries U, Popay J. Is the short form 36 (SF-36) suitable for routine health outcomes assessment in health care for older people? Evidence from preliminary work in community based health services in England. *J Epidemiol Community Health* 1996;**50**:94–8.
112. Taft C, Karlsson J, Sullivan M. Do SF-36 summary component scores accurately summarize subscale scores? *Qual Life Res* 2001;**10**:395–404.
113. Guyatt G. Insights and limitations from health-related quality-of-life research. *J Gen Intern Med* 1997;**12**:720–1.
114. Reimer MA, Flemons WW. Quality of life in sleep disorders. *Sleep Med Rev* 2003;**7**:335–49.
115. Carey TJ, Moul DE, Pilkonis P, Germain A, Buysse DJ. Focusing on the experience of insomnia. *Behav Sleep Med* 2005;**3**:73–86.
116. Kleinman L, Zodet MW, Hakim Z, Aledort J, Barker C, Chan K, et al. Psychometric evaluation of the fatigue severity scale for use in chronic hepatitis C. *Qual Life Res* 2000;**9**:499–508.
117. Aaronson NK. Quality of life assessment in clinical-trials – methodologic issues. *Control Clin Trials* 1989;**10**:S195–208.
118. David B, Morgan K. Occupational and daytime functioning in primary insomnia: a prospective study. *J Sleep Res* 2006;**15**:157.
119. Verster JC, David B, Morgan K, Olivier B. Validation of the Dutch occupational impact of sleep questionnaire (OISQ). *Ind Health* 2008;**46**:601–6.
120. Erman M, Guiraud A, Joish VN, Lerner D. Zolpidem extended-release 12.5 mg associated with improvements in work performance in a 6-month randomized, placebo-controlled trial. *Sleep* 2008;**31**:1371–8.
121. Lerner D, Amick BC, Rogers WH, Malspeis S, Bungay K, Cynn D. The work limitations questionnaire. *Med Care* 2001;**39**:72–85.
122. Kyle SD, Espie CA, Morgan K. The impact of insomnia on occupational functioning: a 'crystallisation' of qualitative methods. *J Sleep Res* 2008;**17**(Suppl. 1):304.
123. Troxel WM, Robles TF, Hall M, Buysse DJ. Marital quality and the marital bed: examining the covariation between relationship quality and sleep. *Sleep Med Rev* 2007;**11**:389–404.
124. McFadyen TA, Espie CA, McArdle N, Douglas NJ, Engleman HM. Controlled, prospective trial of psychosocial function before and after continuous positive airway pressure therapy. *Eur Resp J* 2001;**18**:996–1002.
125. De Boer AGEM, van Lanschot JJB, Stalmeier PFM, van Sandick JW, Hulscher JBF, De Haes JCJM, et al. Is a single-item visual analogue scale as valid, reliable and responsive as multi-item scales in measuring quality of life? *Qual Life Res* 2004;**13**:311–20.
126. Guy W. *ECDEU assessment manual for psychopharmacology*. Rockville, MD: US Department of Heath, Education, and Welfare Public Health Service Alcohol, Drug Abuse, and Mental Health Administration; 1976.
127. Fox S, Buysse D, Grubb W, Ancoli-Israel S. Insomnia and daytime functional impairment: a pilot study of whether ethnographic research has a role in refining current assessment measures. *Sleep* 2007;**30**(Abstract Suppl.): 754.
128. Bilsbury CD, Richman A. A staging approach to measuring patient-centred subjective outcomes. *Acta Psychiatr Scand* 2002;**106**:5–40.
129. Ruta DA, Garratt AM, Leng M, Russell IT, Macdonald LM. A New approach to the measurement of quality-of-life - the patient-generated index. *Med Care* 1994;**32**:1109–26.
130. Kyle SD, Espie CA, Morgan K, Fleming L. The Glasgow sleep impact index: a patient generated measure for capturing sleep-related quality of life. *Sleep* 2009;**32**:0850.
131. Daley M, Morin CM, LeBlanc M, Gregoire JP, Savard J. The Economic burden of insomnia: direct and indirect costs for individuals with insomnia syndrome, insomnia symptoms, and good sleepers. *Sleep* 2009;**32**:55–64.
132. Taylor DJ. Insomnia and depression. *Sleep* 2008;**31**:447–8.

Psycho-Oncology
Psycho-Oncology (2009)
Published online in Wiley InterScience (www.interscience.wiley.com). **DOI**: 10.1002/pon.1652

The development and impact of insomnia on cancer survivors: a qualitative analysis

Leanne Fleming*, Stephanie Gillespie and Colin A. Espie
University of Glasgow Sleep Centre, Southern General Hospital, Glasgow, UK

** Correspondence to: University of Glasgow Sleep Centre, Southern General Hospital, Glasgow G51 4TF, UK. E-mail: L.Fleming@clinmed.gla.ac.uk*

Received: 7 May 2009
Revised: 18 August 2009
Accepted: 20 September 2009

Abstract

***Objectives*: To conduct the first qualitative analysis of the development and impact of insomnia on a cohort of cancer survivors.**

***Methods*: Twenty-one cancer survivors with a history of chronic insomnia contributed to four focus groups held at the University of Glasgow Sleep Research Centre. Participants' perceptions of the onset, evolution and effects of insomnia were elicited and qualitatively explored using content analysis.**

***Results*: Most participants reported insomnia onset following cancer diagnosis. Participants who had a pre-existing insomnia reported that cancer diagnosis significantly aggravated their sleep complaint. Active cancer treatment was a major contributor to poor sleep quality due to the disruption of normal daily routines. This poor sleep pattern became persistent once active treatment had ceased and participants reported becoming particularly concerned about their sleep when they were discharged into follow-up cancer care. The impact of insomnia was significant for all participants in the study and six major areas emerged as being particularly affected; mood, physical health, relationships, sleep quality, sleep-related behaviour and cognition.**

***Conclusions*: The majority of cancer survivors in this study developed disturbed sleep as a result of cancer diagnosis and their sleep disruption was exacerbated by active cancer treatment. Insomnia also had a significant impact upon quality of life and these effects persisted long beyond the cessation of active anti-cancer therapy. Early identification of insomnia symptoms in cancer care settings must be a priority to ensure that sleep disturbance is not overlooked or poorly managed.**

Keywords: cancer; oncology; insomnia; sleep; qualitative

Introduction

Insomnia is among the most distressing, debilitating and frequently reported problems experienced by cancer survivors both during and after completion of active treatment [1–3]. Prevalence estimates suggest that 30–50% of newly diagnosed or recently treated cancer patients express difficulty sleeping [4], with 19–30% meeting diagnostic criteria for chronic insomnia [3,4]. Sleep patterns commonly become disrupted around diagnosis. Recent studies on treated prostate and breast cancer groups demonstrate that almost half of patients with sleep problems report insomnia onset, which follows cancer diagnosis [5,6]. However, sleep disruptions can persist long beyond the cessation of active treatment [7,8]. One-quarter of cancer survivors experience chronic insomnia that negatively affects daytime functioning and quality of life. In a study of 982 respondents, 75% of cancer survivors had a chronic insomnia disorder that lasted six months or more [3]. This pattern of persistence is seen commonly in oncology populations with 25% of cancer survivors on long-term hypnotic medication [3]. However, this long-term pharmacotherapy is particularly undesirable to cancer patients [9–11]. Evidence demonstrates that cognitive behaviour therapy (CBT) is the treatment of choice for insomnia [12–17] and patient acceptability of this non-pharmacological intervention is high [18]. However, CBT is rarely available to cancer populations resulting in the majority resorting to self-management strategies.

Insomnia disorder contributes additional risk for experiencing persistent fatigue after cancer treatment [4]. Fatigue affects approximately 70% of patients receiving chemotherapy or radiotherapy treatments [19–21] and like insomnia is typically viewed as a temporary reaction to anti-cancer therapy. However, research has found that fatigue can persist for months following the termination of active treatment [22–24]. Results from a recent clinical effectiveness trial of CBT for insomnia in cancer patients demonstrated that CBT for insomnia

significantly reduced fatigue in these patients at post-treatment [17]. Further work is needed to clarify the directional relationship between insomnia and fatigue in this population.

Alongside fatigue, untreated insomnia also leads to an increased risk of depression [25]. Psychiatric epidemiology indicates that insomnia is an independent risk factor for the development of first-episode depressive disorder [26] and a study on depressed and non-depressed individuals demonstrated that sleep disturbance was associated with immune downregulation and decreased activity of natural killer cells [27]. These cells play an important role in anti-tumour immune responses, suggesting that insomnia may impact on cancer course and survival rates [28].

In general, insomnia in cancer patients is frequently overlooked and poorly managed [4,11] leading to it being greatly under-reported. Individuals with cancer often develop disturbed sleep following diagnosis, which commonly evolves into a chronic insomnia syndrome resulting in diminished quality of life [29,30], impaired mood [31], heightened pain and reduced energy levels [4]. Insomniacs report a higher frequency of health problems and employ greater utilisation of medical and mental health services relative to good sleepers [32,33]. Yet insomnia is a neglected problem. Despite the wealth of evidence surrounding its high prevalence and potentially negative health costs, little time and attention is devoted to cancer-related insomnia research. This paper attempts to fill gaps in the existing literature by reporting on the first qualitative study to describe the development and impact of chronic sleep disturbance in a cohort of cancer survivors with co-morbid insomnia.

Participants and methods

Participants for this study were recruited between October and December 2007 from a database established during a prior clinical effectiveness trial [17] and criteria for inclusion in this study were established during this previous trial. This included (i) participants who had been diagnosed with breast, colorectal, prostate or gynaecological cancer, (ii) participants who satisfied diagnostic criteria for chronic insomnia (mean value >30 min for complaint of delayed sleep-onset latency (SOL) and/or wake time after sleep onset (WASO), occurring $\geqslant 3$ nights per week for $\geqslant 3$ months and affecting daytime function) [34], (iii) participants were at least 1 month post cancer treatment with no further anti-cancer therapy planned (thus excluding acute insomnia and any transient effects associated with cancer treatment), (iv) participants were aged 18 years or over. The exclusion criteria were as follows: (i) short term or acute insomnia, (ii) evidence of other sleep disorders (e.g. sleep apnoea), (iii) evidence of drug misuse. All participants in the current study had received CBT for insomnia during their participation in the previous trial [17].

In all, 54 potential participants were contacted by letter asking if they would like to take part in a small focus group to discuss the ways in which insomnia affects quality of life. A patient information sheet was also enclosed providing details about the purpose and nature of the study. A total of 21 individuals responded, providing written consent, which indicated that they wished to participate and 33 failed to respond (these 33 were contacted on a second occasion by follow-up letter but when there continued to be no response, no further contact was made). Participant demographic details are given in Table 1.

Procedure

As this was an exploratory study, the use of focus groups was an appropriate approach. Alongside being an efficient means of data collection, focus groups permit participants to identify topics that are important to them and to utilise their own frames of reference for discussion. This approach permits direct access to patient experiences and perceptions. Our focus groups were held at the Glasgow Sleep Research Centre in the Southern General Hospital (a location familiar to all participants) at a convenient time. The focus groups lasted an average of 90 min and participants were randomly allocated to one of four groups. Each focus group was facilitated by L. F. (a research psychologist) and S. G. (a medical student) and consisted of only one session. Both L. F. and S. G. attended a one-day training session on 'how to run effective focus groups' in order to ensure their suitability as facilitators. The topic schedule was developed using published literature

Table 1. Participant demographics (all data is frequency except 'mean age')

Mean age (years)	**62**
Gender	
Male	7
Female	14
Civil status	
Partner	16
None	5
Occupational status	
Employed	8
Not employed	13
Cancer diagnosis	
Breast	9
Prostate	6
Bowel	5
Gynaecological	1
Mean time since cancer diagnosis (months)	34

DOI: 10.1002/pon

and our previous experience. Participants were asked to respond to the following questions:

(i) When did your sleep first become disturbed?
(ii) What do you think caused your insomnia?
(iii) What impact did insomnia have on your life?

All participants were given the opportunity to respond to each question and any discussion was encouraged and supported. At the end of the session, the facilitator provided a verbal summary of the main topics and issues highlighted during the session and asked the participants to confirm that this was an accurate account of their discussion.

Immediately following each of the sessions, the facilitators held a debriefing meeting in order to discuss and record the main themes that emerged during the focus group. The facilitators then generated summary sheets of the content of the focus groups and these were sent to each of the participants, the purpose of which was to ensure that the summary notes were an accurate reflection of the focus group discussions. These summary notes were checked, agreed, signed and returned by all participants.

Analysis

Focus groups, data collection and analysis were concurrent and data saturation was achieved. All the focus group sessions were audio-taped and were transcribed verbatim. Content analysis was used to systematically analyse the data produced from each of the focus groups and the data analysis followed established criteria for quality and rigor [35–37]. The transcripts of the focus groups were examined independently by three readers: the two facilitators and a clinical psychology trainee. The readers were from different disciplines and only one had prior knowledge of the area of research, enhancing the reliability of the findings. The readers were asked to familiarise themselves with the transcripts and record any general themes that emerged from the data. The readers were then asked to read through the transcripts again, copying units of analysis (defined as words or phrases) onto individual pieces of paper, ensuring the context was retained. This was repeated several times to ensure that no detail was overlooked. These units of analysis were subsequently allocated to each of the themes to provide confirming examples illustrative of each. This distribution was re-assessed and units of analysis moved between themes if deemed appropriate. Within each of the general themes, sub-themes were created. The sub-themes aimed to cover all information generated in each of the focus groups in greater detail. Again, the units of analysis were distributed between each of these, providing representative quotes. The readers met on several occasions to discuss their results until an agreement was reached. Inter-rater agreement was 0.72.

Results

The majority of participants ($n = 19$) reported that the onset of their sleep disturbance followed their cancer diagnosis. Only two participants stated that they had an existing problem with sleep prior to diagnosis, however, both of these remarked that their cancer diagnosis significantly aggravated their insomnia.

The 'experience of being a cancer patient' was described by five participants as the cause of sleep disturbance. Further discussion around this led to many participants describing 'suppressed stresses' as being the main contributor to poor sleep. Eleven participants blamed a lack of daily routine as the cause of their insomnia with four of these participants referring more specifically to the fact that they could not work as a result of their anti-cancer treatment. Other participants commented on the 'bad pattern' which develops as a result of lack of daily routine. Specifically, daytime naps, late retiring and rising times and lack of physical exercise were all reported to contribute to a poor sleep pattern. Cancer treatment was also described as a major cause of sleep disturbance with six people reporting that chemotherapy and/or radiotherapy led to a significant deterioration in their sleep quality. Nine participants found adjuvant hormone therapy to be the most significant contributor, with specific reference made to night sweats induced by tamoxifen in breast cancer patients and nocturia in prostate patients (Table 2).

The impact of insomnia was significant for all participants in the study. Interestingly, the majority of contributors ($n = 16$) described the effects of poor sleep as much more overwhelming than the effects of cancer treatment. From the analysis, six major categories emerged: mood, physical symptoms, relationships, sleep quality, behavioural modifications and cognitive consequences. Each of

Table 2. Participants' qualitative reports on the causes of insomnia

'I put my sleep problems down to the fact that I'd had cancer or I was on cancer drugs or something and eventually I thought, you know, I can't remember the last time I slept properly' (Participant 4: female)
'I found it was the cancer treatment rather than the cancer diagnosis, but I suppose we are all different in how it affects us psychologically' (Participant 11: female)
'It was almost three years ago I was diagnosed and I've forgotten about it (the cancer), but the sleep problem is still there' (Participant 6: female)
'I had an existing problem…but I got much worse after I was diagnosed with cancer' (Participant 14: male)
'It was either just the stress of the diagnosis catching up with me that caused my sleep problem or the diagnosis and I didn't recognise it at the time' (Participant 1: male)

DOI: 10.1002/pon

these categories contained several sub-themes (Table 3).

Mood

Changes in mood were one of the most significant aspects of sleep disruption. Participants reported that change in temperament and feeling '*different from normal*' (participant 20: male, 64 years) was common after a poor nights sleep. They lacked enthusiasm for life, were highly irritable and felt considerable frustration at their inability to control their sleep pattern. This led to feelings of helplessness and loss of confidence. A number of participants experienced high levels of guilt about feeling tired during the day. Once they had completed cancer treatment, they felt considerable pressure from family/friends to '*get back to normal*' (participant 18: female, 52 years). When they were unable to achieve certain milestones like returning to work or re-establishing their social lives due to poor sleep patterns, participants felt incredibly guilty about continuing to fulfil an 'illness role'.

Table 3. Themes and sub-themes generated from the question 'What impact did insomnia have on your life?'

Theme	Sub-themes	Frequency
Mood	Irritability	4 (19%)
	Change in temperament	7 (33%)
	Guilt about tiredness	3 (17%)
	Frustration	2 (8%)
	Helplessness	4 (19%)
	Loss of confidence	2 (8%)
	Lack of enthusiasm for life	3 (17%)
Physical symptoms	Feeling tired/fatigued	12 (58%)
	Feeling nauseous	3 (17%)
	Head thumping	4 (19%)
	Heavy eyes and body	3 (17%)
	Heightened pain	2 (8%)
	Hyper-arousal	3 (17%)
Relationships	Difficulty relating to others	2 (8%)
	Intolerant	2 (8%)
	Poor conversation skills	3 (17%)
	Restricted social interactions	10 (50%)
	Rescheduling activities/putting things off	10 (50%)
	Feeling misunderstood	3 (17%)
	Withdrawn/reclusive	4 (19%)
Sleep quality	Easily disturbed	3 (17%)
	Concern about disturbing partner	5 (25%)
	Restlessness	3 (17%)
	Frequent night-time awakenings	14 (67%)
Behavioural modifications	Daytime naps	9 (42%)
	Early retiring time	2 (8%)
	Lie-in	5 (25%)
	Sleep in different room from partner	5 (25%)
	Altered bedtime routine	3 (17%)
Cognitive consequences	Memory deficits	5 (25%)
	Lack of concentration	2 (8%)
	Cognitive intrusions about sleep	3 (17%)
	Catastrophising	9 (42%)
	Lack of control	5 (25%)
	Pressure to be 'normal'	7 (33%)
	Worry about next nights sleep	5 (25%)

Physical symptoms

Participants reported various physical symptoms, the most pronounced being feelings of fatigue and tiredness. The physical effects of sleep loss were described as similar to flu-like symptoms and included nausea, headache, heavy eyes and heightened levels of pain. Participants described engaging in a process of 'internal scrutiny', frequently monitoring for signs and symptoms of cancer returning. The physical symptoms of sleep loss resulted in some individuals' catastrophising about recurring illness.

Relationships

Sleep disturbance had a significant impact upon inter-personal relationships. Participants avoided making plans due to the erratic nature of their sleep and their inability to predict how they would feel the next day. This resulted in half of all participants restricting social interactions and postponing daily tasks. Social isolation was compounded by feelings of intolerance and difficulty relating to others.

Sleep quality

Poor sleep quality resulted in several participants sleeping in a different room from their partner in order to minimise disruption. Such an alteration to sleeping arrangements had a considerable impact on relationships and intimacy and as a result, some participants reported that relationships had broken down completely.

Behavioural modifications

In an attempt to combat the effects of disturbed sleep, participants made several modifications to their usual sleep behaviour. Most reported a cycle of trying to 'catch-up' on lost sleep by employing early retiring and late waking times, daytime naps and unusual 'ritual-like' bedtime routines. However, this level of attention and effort is widely known to disrupt sleep patterns further.

Cognitive consequences

Cognitive consequences of sleep disturbance were common and included catastrophising about the effects of poor sleep, cognitive intrusions about sleep, memory deficits and loss of concentration. These consequences were so severe for one participant that she felt unable to continue in employment and sought early retirement (Table 4).

DOI: 10.1002/pon

Table 4. Participants' qualitative reports on the impact of insomnia

'I was irritable and arguing with everybody' (Participant 14: male)
'My whole quality of life changed and I felt I couldn't do my job properly anymore' (Participant 9: female)
'I experienced constant waves of utter debilitating fatigue throughout the day' (Participant 3: female)
'I was getting into a habit of putting my head down and sleeping during the day because my chemo tired me out so much, this is why I was up at night, because I'd had my rest' (Participant 16: male)
'You can't make the effort to keep up with the nitty-gritty of conversations—they're just too much effort' (Participant 3: female)
'I've never been a morning person and but I found it was taking longer and longer to get going in the morning' (Participant 12: female)
'I find you want to keep putting things off, something physical, like cutting the grass or hedge or filling in my tax form, you know, I say, I'll do it tomorrow and I'll go to bed and get a good sleep because you know your brain is not going to function if you don't' (Participant 14: male)
'If people say to me, 'Do you fancy going to such a place on Friday?' I'll say, 'Can I phone you, it all depends on what kind of night I have on Thursday?'' (Participant 3: female)

Discussion

Insomnia commonly follows cancer diagnosis, is exacerbated by anti-cancer therapy and has significant consequences for physical and psychological well-being [3,4]. However, there is a lack of qualitative research investigating the specific effects of insomnia co-occurring with cancer. Our study directly accessed perceptions and experiences of a group of cancer survivors in an attempt to identify the development of insomnia and its impact on quality of life. Most of the participants in our study reported that they developed disturbed sleep as a result of cancer diagnosis and that this sleep disruption was exacerbated by active cancer treatment. We also established that insomnia significantly impacts upon an individuals' quality of life and that these effects are stable and persistent.

The early identification and treatment of insomnia symptoms should be incorporated as an important part of cancer symptom management. Neglecting insomnia symptoms *might* lead to the onset of a syndrome-level sleep complaint with far-reaching implications for psychological, physical and social well-being. CBT has been previously validated as the treatment of choice for insomnia within oncology populations [5,17], hence treatment options are available to prevent the development of chronic sleep complaints within cancer groups.

Participants described a shift in focus from cancer to sleep, around the time of discharge into 'follow-up cancer care'. They reported increasing anxiety over disturbed sleep as 'normal life' was expected to return at this time. When sleep continues to be disrupted, it becomes a focus, a behaviour which is known to perpetuate and exacerbate insomnia. It seems appropriate therefore, to provide sleep information/advice at this vulnerable stage in the 'cancer journey', which may help prevent the development of a chronic problem with sleep. Further research is required to evaluate the most suitable point to provide insomnia intervention during cancer treatment.

All participants voiced great concern over the absence of sleep-related information or advice during cancer treatment. This is alarming as the high percentage of cancer patients burdened with insomnia has been demonstrated by various studies [1–4]. Insomnia is poorly managed within cancer treatment and rehabilitation [4,11] as Oncology staff have neither the time nor the necessary expertise to deal with insomnia complaints. Insomnia assessments should be incorporated into cancer rehabilitation, particularly at the highly vulnerable periods of diagnosis and post-treatment/discharge.

Our sample was not representative of all individuals with cancer and sleep problems; therefore, these findings are not necessarily generalisable to the cancer population as a whole. Neither was our sample representative of all participants who met eligibility criteria as only 39% of the total number of eligible participants took part in this study. A selection bias in favour of people who feel comfortable discussing their experiences and who are highly motivated to co-operate is assumed.

The data generated in each of the focus groups were based on patients' self-reported experiences of cancer and sleep problems providing a purely subjective account. However, several studies have demonstrated that self-reports of medical status have acceptable concordance with physical or medical records, with subjective ratings reflecting physiological measures [38].

Qualitative methodologies were deemed to be the most appropriate for such an exploratory study, but we acknowledge that researcher bias and preconceptions may influence our findings. Various measures were put in place to minimise this; multiple readers studied the transcripts independently and were guided on how this was to be carried out following a systematic method [35] aiming to maximise reliability and validity [36,37].

Acknowledgements

The authors thank all participants for their valuable contribution to this study and gratefully acknowledge funding support from the National Cancer Research Institute.

Contributors: This study was funded as part of a capacity building grant award made to L. F. by the National Cancer Research Institute. L. F. conceived the idea and designed the study, conducted each of the focus groups, analysed the data and drafted the paper. S. G. assisted with the focus groups and analysed the data. C. E. critically revised the paper. L.F. is the guarantor of the paper.

Ethical approval: Ethical approval for this study was granted by South Glasgow and Clyde Research Ethics Committee on 26th March 2007.

References

1. Whelan TJ, Mohide EA, Willan AR *et al*. The supportive needs of newly diagnosed cancer patients

DOI: 10.1002/pon

attending a regional cancer centre. *Cancer* 1997;**80**: 1518–1524.
2. Munro AJ, Potter S. A quantitative approach to the distress caused by symptoms in patients treated with radical radiotherapy. *Br J Cancer* 1996;**74**:640–647.
3. Davidson JR, MacLean AW, Brundage MD, Schulze K. Sleep disturbance in cancer patients. *Soc Sci Med* 2002;**54**:1309–1321.
4. Savard J, Morin CM. Insomnia in the context of cancer: a review of a neglected problem. *J Clin Oncol* 2001;**19**:895–908.
5. Savard J, Simard S, Hervouet S, Ivers H, Lacombe L, Fradet Y. Insomnia in men treated with radical prostatectomy for prostate cancer. *Psycho-Oncology* 2005;**14**(2):147–156.
6. Savard J, Simard S, Blanchet J, Ivers I, Morin CM. Prevalence, clinical characteristics and risk factors for insomnia in the context of breast cancer. *Sleep* 2001;**24**(5):583–590.
7. Couzi RJ, Helzslsouer KJ, Fetting JH. Prevalence of menopausal symptoms among women with a history of breast cancer and attitudes towards oestrogen replacement therapy. *J Clin Oncol* 1995;**13**:2737–2744.
8. Lindley C, Vasa S, Sawyer WT *et al*. Quality of life and preferences for treatment following systemic adjuvant therapy for early-stage breast cancer. *J Clin Oncol* 1998;**16**:1380–1387.
9. Kripke D. Hypnotic drugs: deadly risks, doubtful benefits. *Sleep Med Rev* 2000;**4**:5–20.
10. Buysse DJ. Rational pharmacotherapy for insomnia: time for a new paradigm. *Sleep Med Rev* 2000;**4**:521–527.
11. Sateia MJ, Nowell PD. Insomnia. *Lancet* 2004;**364**: 1959–1973.
12. Morin CM, Hauri P, Espie CA, Spielman A, Buysse D, Bootzin RR. Nonpharmacologic treatment of chronic insomnia: An American Academy of Sleep Medicine Review. *Sleep* 1999;**22**:1134–1156.
13. Morin CM, Bootzin RR, Buysse DJ, Edinger JD, Espie CA, Lichstein KL. Psychological and behavioral treatment of insomnia: update of the recent evidence (1998–2004) prepared by a task force of the American Academy of Sleep Medicine. *Sleep*. 2006;**29**(11):1398–1414.
14. Irwin MR, Cole JC, Nicassio PM. Comparative meta-analysis of behavioral interventions for insomnia and their efficacy in middle-aged adults and in older adults 55+ years of age. *Health Psychol* 2006;**25**:3–14.
15. Smith MT, Perlis ML, Park A, Giles DE, Pennington JA, Buysse D. Behavioral treatment vs. pharmacotherapy for insomnia—a comparative meta-analyses. *Am J Psychiatry* 2002;**159**:5–11.
16. Savard J, Simard S, Ivers H, Morin CM. A randomized study on the efficacy of Cognitive-Behavioral Therapy for insomnia secondary to breast cancer: Part 1—sleep and psychological effects. *J Clin Oncol* 2005;**23**:6083–6096.
17. Espie CA, Fleming L, Cassidy J *et al*. Randomised controlled clinical effectiveness trial of cognitive behaviour therapy versus treatment as usual for persistent insomnia in cancer patients. *J Clin Oncol* 2008;**26**(28):4651–4658.
18. Davidson JR, Feldman-Stewart D, Brennenstuhl S, Ram S. How to provide insomnia interventions to people with cancer: insights from patients. *Psycho-Oncology* 2007;**16**(11):1028–1038.
19. Mock V. Evidence-based treatment for cancer-related fatigue. *J Natl Cancer Inst Monogr* 2004;**32**:112–118.
20. Stone P, Richards M, Hardy J. Fatigue in patients with cancer. *Eur J Cancer* 1998;**34**:1670–1676.
21. Portenoy RK, Itri LM. Cancer-related fatigue: guidelines for management and evaluation. *Oncologist* 1999; **4**:1–10.
22. Akechi T, Kugaya A, Okamura H *et al*. Fatigue and its associated factors in ambulatory cancer patients: a preliminary study. *J Pain Symptom Manage* 1999;**17**: 42–48.
23. Andrykowski MA, Curran SL, Lightner R. Off-treatment fatigue on breast cancer survivors: a controlled comparison. *J Behav Med* 1998;**21**:1–18.
24. Smets EMA, Visser MRM, Williams-Groot AFMN *et al*. Fatigue and radiotherapy: experience in patients 9 months following treatment. *Br J Cancer* 1998;**78**:907–912.
25. Eaton WW, Badawi M, Melton, B. Prodromes and precursors: epidemiological data for primary prevention of disorders with slow onset. *Am J Psychiatry* 1995;**152**(7):967–972.
26. Riemann D, Voderholzer U. Primary insomnia: a risk factor to develop depression. *J Affect Disord* 2003;**76**: 255–259.
27. Irwin M, Clark C, Kennedy B, Gillin JC, Ziegler M. Nocturnal catecholamines and immune function in insomniacs, depressed patients and control subjects. *Brain Behav Immun* 2003;**17**(5):365–372.
28. Kripke DF, Garfinkel, L. Mortality associated with sleep duration and insomnia. *Arch Gen Psychiatry* 2002;**59**(2):131–136.
29. Roth T, Ancoli-Israel S. Daytime consequences and correlates of insomnia in the United States: results of the 1991 National Sleep Foundation Survey II. *Sleep* 1999;**22**(Suppl. 2):S354–S358.
30. Zammit GK, Weiner J, Damato N, Sillup G, McMillan C. Quality of life in people with insomnia. *Sleep* 1999;**22**(Suppl. 2): S379–S385.
31. Pilcher JJ, Huffcutt AI. Effects of sleep deprivation on performance: a meta-analysis. *Sleep* 1996;**19**(4): 318–326.
32. Simon GE, VonKorff M. Prevalence, burden and treatment of insomnia in primary care. *Am J Psychiatry* 1997;**154**(10):1479–1484.
33. Weissman MM, Greenwald S, Nino-Murcia G, Dement WC. The morbidity of insomnia uncomplicated by psychiatric disorders. *Gen Hosp Psychiatry* 1997; **19**:245–250.
34. American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders*: *DSM-IV*. *Text Revision*. 2000.
35. Kondracki NL, Wellman NS, Amundson DR. Content analysis: review of methods and their application in nutritional education. *J Nutr Educ Behav* 2002;**34**(4): 224–230.
36. Sofaer S. Qualitative research methods. *Int J Qual Health Care* 2002;**14**(4):329–336.
37. Meyrick J. What is good qualitative research? A first step towards a comprehensive approach to judging the rigour/ quality. *J Health Psychol* 2006;**11**(5):799–808.
38. Kehoe R, Wu S, Leske MC, Chylack JL T. Comparing self-reported and physician-reported medical history. *Am J Epidemiol* 1994;**139**:813–818.

DOI: 10.1002/pon

Sleep Medicine 12 (2011) 735–747

Contents lists available at SciVerse ScienceDirect

Sleep Medicine

journal homepage: www.elsevier.com/locate/sleep

Original Article

No pain, no gain: An exploratory within-subjects mixed-methods evaluation of the patient experience of sleep restriction therapy (SRT) for insomnia

Simon D. Kyle [a,*], Kevin Morgan [b], Kai Spiegelhalder [c], Colin A. Espie [a]

[a] University of Glasgow Sleep Centre, Sackler Institute of Psychobiological Research and Institute of Neuroscience and Psychology, College of Medical, Veterinary and Life Sciences, University of Glasgow, Scotland, UK
[b] Clinical Sleep Disorders Unit, Loughborough University, England, UK
[c] Department of Psychiatry and Psychotherapy, University of Freiburg Medical Centre, Freiburg, Germany

ARTICLE INFO

Article history:
Received 1 October 2010
Received in revised form 21 December 2010
Accepted 25 March 2011

Keywords:
Insomnia
Treatment
Patient experience
Qualitative
Side-effects

ABSTRACT

Objective: To explore the patient experience of Sleep Restriction Therapy (SRT) for insomnia, with particular focus on elucidating possible side-effects, challenges to adherence and implementation and perceptions of benefit/impact.
Methods: To fully investigate the patient experience of sleep restriction therapy for insomnia we designed a within-subjects mixed-method study, employing sleep and daytime functioning questionnaires, assessments of sleep-restriction-related side-effects, prospective qualitative audio-diaries and post-treatment semi-structured interviews. University of Glasgow Sleep Centre. Eighteen patients with Primary Insomnia (mean age = 42; range 18–64). Patients took part in a 4-week brief sleep restriction intervention, involving two group sessions and two subsequent follow-up phone calls in the home environment.
Measurements and results: Sleep diaries and global measures of insomnia severity and sleep quality, as expected, demonstrated robust improvements at both post-treatment and 3-month follow-up (all large effect sizes). Daytime functioning/health-related quality of life variables similarly evidenced strong treatment effects (moderate to large effect sizes). Reported side-effects were common, with $\geqslant$50% of patients reporting impairment in 8 out of 12 listed symptoms as a consequence of initiating treatment. The four most common side-effects were 'fatigue/exhaustion' (100%), 'extreme sleepiness' (94%), 'reduced motivation/energy' (89%) and 'headache/migraine' (72%) [Mean number of symptoms per patient = 7.2 (2.4); range 3–11]. Intriguingly, both side-effect frequency and ratings of side-effect interference were associated with baseline to post-treatment improvements in sleep quality. Qualitative real-time audio-diaries during week 1 of treatment and post-treatment interviews provided rich accounts of side-effects associated with acute SRT implementation; general challenges surrounding treatment implementation and adherence/non-adherence; and modifications to sleep parameters, daytime functioning and perceptions of sleep/sleep period.
Conclusions: This work has important implications for the delivery of SRT, particularly concerning awareness of possible 'adverse events' and likely implementation/adherence challenges. Findings also pave the way for testable hypotheses concerning possible mechanisms of action involved in sleep restriction treatment.

1. Introduction

Cognitive behavioural therapy for insomnia (CBT-I) is widely accepted as an effective intervention for improving core insomnia symptoms. Indeed, CBT-I is a standard, recommended treatment [1], and is commonly regarded as the treatment modality of choice [2,3]. Remarkably, however, despite over two decades of CBT-I investigations, no single study has asked patients about their experience of CBT-I. That is, similar to the dearth of qualitative data on the subjective experience of insomnia (see [4]), there also remains a gap in the literature describing the patient perspective on treatment. Such fundamental work is long overdue, and has potentially important implications.

Firstly, although there are a number of plausible explanations as to how CBT-I exerts its therapeutic effect [5–10], there are few experimental studies that shed light on candidate mechanisms. Tracking the subjective narrative account of treatment, longitudinally, may prove fruitful in confirming, as well as discovering, factors associated with CBT-I response.

* Corresponding author. Address: University of Glasgow Sleep Centre, Sackler Institute of Psychobiological Research, Southern General Hospital, Glasgow G51 4TF, Scotland, UK. Tel.: +44 (0) 141 232 7699.
E-mail address: s.d.kyle@clinmed.gla.ac.uk (S.D. Kyle).

1389-9457/$ - see front matter © 2011 Elsevier B.V. All rights reserved.
doi:10.1016/j.sleep.2011.03.016

Secondly, it is often presumed that psychological therapies, in contrast to pharmacological interventions, are devoid of 'side-effects' [11,12]. This may be naïve, particularly in the context of the behavioural treatment of insomnia. Stimulus Control and Sleep Restriction therapies can lead to significant (acute) decrements in total sleep time, and, therefore, possibly induce daytime dysfunction over and above baseline difficulties. The presence of 'side-effects' are yet to be described, systematically, beyond anecdotal report [6,13]. Fittingly, in their recent review, Riemann and Perlis [2] asserted "*the question of adverse events has not been properly addressed up to now in research on psychological/behavioural methods, possibly taken it for granted that no such risks exist…research on adverse events for psychological treatments needs to be intensified*" (p. 213). The NIH state-of-the-science conference statement (2005) [3] came to similar conclusions: *'there is no evidence that such treatment produces adverse effects, but thus far, there has been little, if any, study of this possibility'* (p. 1056).

The lack of investigation of CBT-I side-effects can be contrasted with approaches in other areas of medicine, such as pharmaceuticals, where there tends to be rigorous recording of 'adverse events' during active therapy. Adverse event profiles are then used as cautionary notes to inform patients when initiating treatment. This is also the case for sleep apnea patients undergoing continuous positive airway pressure (CPAP) therapy, where side-effects have been associated with drop-out rates and non-adherence during clinical trials [14].

Thirdly, the benefit of prospective qualitative monitoring may be a contribution to our understanding of adherence to behavioural instructions. To date, there are only a handful of published studies investigating adherence to stimulus control and/or sleep restriction, using a variety of adherence measures – from session attendance and therapist ratings to sleep diary data. Nevertheless, this small literature indicates that: (1) consistency of bed/rising times, though not necessarily sleep reduction, may be predictive of outcome [15]; (2) questionnaire ratings of retrospective global adherence moderately relate to outcome [16]; and (3) pre-treatment sleepiness, dysthymia, lower self-efficacy, perceived barriers to treatment engagement, and CBT-related sleepiness are all associated with reduced adherence and implementation of behavioural guidelines [16–19]. Crucially, although questionnaire data indicate that behavioural aspects of CBT-I strongly relate to outcome at one year follow-up, they remain among the least liked and used components of CBT-I [20,21]. Qualitative data tracking patients' implementation of behavioural instructions could shed light on factors relevant to therapy adherence, and potentially help refine the future delivery of CBT-I.

Fourthly, post-interventional functioning/health-related quality of life (HRQoL) has been inadequately investigated in the CBT-I literature (for a review, see [22]). Indeed, given the small and mixed literature on functional assessment and outcome, some authors have questioned whether improving sleep in those with insomnia will in fact modify self-reported daytime functioning (e.g. [23,24]). Explanations for this apparent incongruence include concerns with measurement [22], and theoretical interpretations surrounding physiological arousal (e.g., [24,25]) and dysfunctional cognitive processes (e.g., [26,27]). One potential way to shed light on sleep and functioning (treatment) relationships is to simply ask participants to describe, in their own words, the impact of insomnia treatment on domains of functioning.

Qualitative methodologies are increasingly being recognised for their ability to explore topics that are poorly dealt with using conventional quantitative tools. Some recent examples in medicine and health care include: understanding adherence/non-adherence to medical regimes [28,29]; experience of illness (e.g., chronic pain [30]); the phenomenology of emotional (blunting) side-effects of antidepressants [31]; and factors that prompt/delay patients in seeking medical advice (e.g. breast cancer patients [32]). Although slow to filter into sleep medicine, perhaps given the infancy of the field, qualitative methodologies have also been applied to the treatment of sleep apnea, providing important information on inhibitory and facilitatory factors related to CPAP adherence [33].

In light of the aforementioned potential benefits of qualitative inquiry, we designed a within-subjects mixed-method study to improve understanding of the implementation, experience, and impact of a condensed sleep restriction intervention. Sleep restriction therapy (SRT) was chosen because of its strong relationship with outcome, relative ease of administration, and reliance on adherence to prescriptive guidelines. Combining common quantitative measures with qualitative techniques, it was felt, would provide a more holistic and pragmatic picture regarding patient experience and attributions, capturing strengths of both inductive and deductive research.

The treatment process was investigated using: (1) audio-diaries, to capture *in vivo* proximal reflections, and (2) post-treatment face-to-face semi-structured interviews, to provide a global experiential account. In addition to exploring the subjective experience of sleep restriction, our design permitted the evaluation of self-reported changes in daytime functioning, forming a secondary aim. Although single-component sleep restriction [34] and sleep compression [35] interventions have proven successful in terms of modifying sleep parameters, comprehensive assessment of daytime functioning impairments is lacking. Thus, we also sought to evaluate whether our brief intervention could improve daytime as well as (predicted) sleep outcome variables.

2. Methods

2.1. Participants

Individuals meeting research diagnostic criteria (RDC [36]) for primary insomnia (PI) were recruited into the study. Participants, therefore, reported difficulties with initiation and/or maintenance of sleep, lasting for at least a one-month period. Individuals were excluded if they had a co-morbid active psychiatric disorder, or medical disorder that was related to sleep disturbance (i.e. participants could have co-morbid medical ailments but they had to show temporal separation, and attributional independence, from the insomnia disorder). This was ascertained using the Glasgow Sleep Centre screening interview schedule, based on a template set out by Morin and Espie [9]. Additionally, in keeping with diagnostic criteria of insomnia as a 24-h disorder, participants had to report at least one daytime impairment attributed to disturbed sleep [36].

Participants were aged between 18 and 65, and recruited through two avenues: (1) those completing non-interventional University of Glasgow Sleep Centre (UGSC) projects; and (2) those responding to local and national adverts, seeking individuals with sleep disturbance to take part in UGSC research. Finally, those taking prescription sleep medication were accepted into the study only if they maintained a stable regime, i.e., they had not undergone a modification to treatment strategy in the four weeks prior to treatment initiation. The study was approved by the Greater Glasgow and Clyde NHS local ethics committee.

2.2. Treatment protocol

Treatment was conducted in groups of three or four and delivered by the lead researcher. Following a seven-day baseline period, treatment took place over four weeks, comprising two group

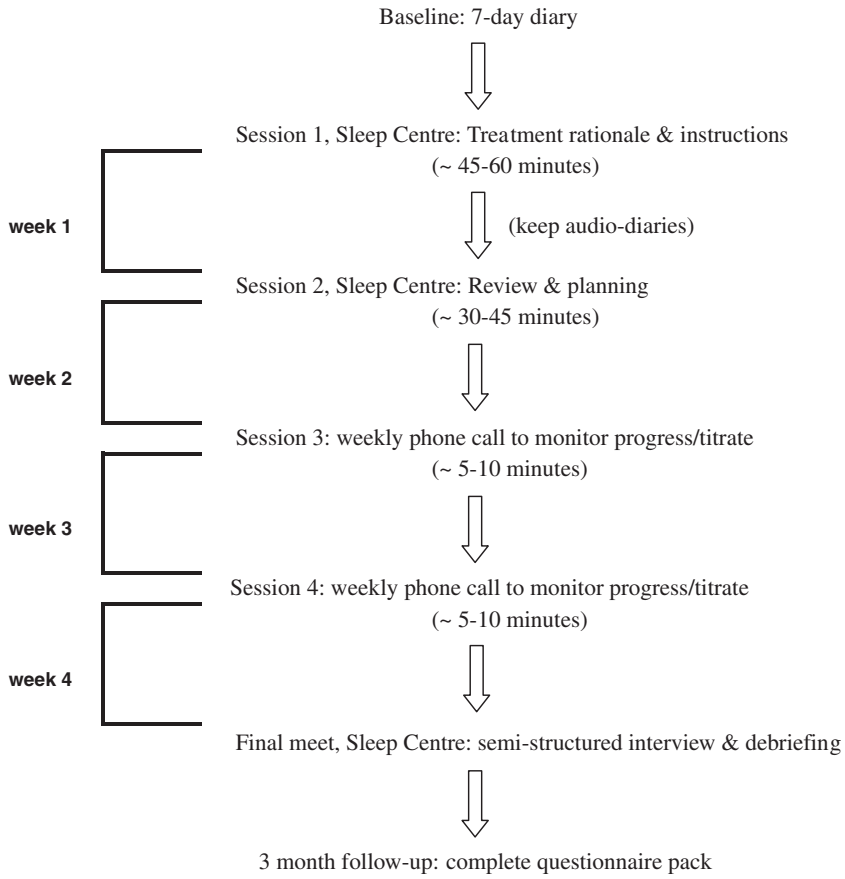


Fig. 1. Session time-line.

sessions and two individualised phone conversations (see Fig. 1 for session time-line). Sleep restriction therapy content was based on Morin and Espie [9] and the Glasgow Sleep Centre protocol for CBT [37], and delivered using PowerPoint™ presentation slides in group format. Specifically, a calculated sleep window was based on the average total sleep time for the baseline period, and was positioned according to the individual schedule of each patient (accounting, also, for circadian factors). If the patient felt that their calculated window was unachievable, then there was some negotiation to try to facilitate adaptation and adherence.

The sleep window was titrated in the following ways: if sleep efficiency for the week was $\geqslant$90%, then the sleep window was increased by 15 min; if sleep efficiency was <85%, then the sleep window was decreased by 15 min; and if values fell between 85% and 89%, then there was no change to the schedule. Downward adjustments were not made until the end of the second week. The minimum possible sleep window duration was set at 5 h.

2.3. Outcome questionnaire measures

The following measures were completed at baseline, post-treatment (four weeks), and three-month follow-up. As well as constituting an outcome variable, sleep diaries were completed each day throughout the treatment phase to guide implementation and titration.

2.3.1. Sleep

Participants completed a daily sleep diary (based on a template set out by Morin and Espie [9]), the Insomnia Severity Index (ISI [8]), the Pittsburgh Sleep Quality Index (PSQI [38]), and the Glasgow Sleep Effort Scale (GSES [39]).

2.3.2. Daytime functioning and side-effects

To assess aspects of daytime functioning and insomnia-related quality of life, participants completed the Occupational Impact of Sleep Questionnaire (OISQ [40]), the Glasgow Sleep Impact Index (GSII [41]), the Daytime Functioning and Sleep Attribution Scale (DFSAS [42]), and the Short-Form Health Survey 36 (SF-36 [43]).

The OISQ is a 24-item scale assessing the impact of sleep quality on various aspects of work-related tasks and productivity (range 0–96; greater scores reflecting more impairment). The tool discriminates between those with insomnia and good sleepers and has high internal consistency (Cronbach's alpha = .95) [40]. The GSII was developed to quantify insomnia-related impairments relevant to each individual patient. Participants write down in their own words the three most important aspects of their life impacted by poor sleep. These are subsequently ranked in importance (1–3) and rated on a 100 mm visual analogue scale (0–100; lower scores reflecting greater impairment). The GSII has demonstrated good concurrent validity, relating moderately with several SF-36 dimensions [41]. The DFSAS is a two-part scale developed to assess impairments in a range of symptoms commonly associated with insomnia disorder (part 1, range 0–36; higher scores indicating greater impairment). Part 2 of the scale asks participants to rate each item again, though this time in relation to how much poor sleep was responsible for the impairment reported in part 1 (range 0–48; higher scores reflect greater poor sleep attribution). Preliminary data indicates good discriminant and concurrent validity, and high internal consistency (Cronbach's alpha = .81 for part 1, and .89 for part 2) [42].

Participants also completed a newly-developed side-effects checklist and interference scale. This scale asks patients to check, from a list, symptoms experienced as a *consequence* of sleep

restriction therapy. Owing to the lack of an existing tool specific for CBT-I 'adverse events', items were generated spanning a mix of somatic and psychological domains, that may be perturbed by sleep deprivation and alterations to sleep timing. Checked symptoms are rated in terms of their interference with daytime functioning (0–4 scale: 'not at all' to 'very much'), following a similar format to existing published side-effects questionnaires used in pharmacological assessment (e.g. antidepressants [44]). Additional space at the end of the scale also allows patients to qualitatively report domains of impairment not listed. The scale was completed during session 2, one week after the commencement of treatment, to align with audio-diaries and to capture initial acute impact.

2.4. Qualitative methodologies

2.4.1. Audio-diaries

Handheld dictaphones (Olympus™ WS-200s) were utilised to track subjective experiences during the first week of treatment. Participants were asked to make two entries per day, according to open-ended semi-structured guidelines. In brief, on awakening (within ~30 min), participants described their experience of implementing treatment instructions for the preceding sleep period. Similarly, in the evening, approximately 2 h before going to bed, participants were instructed to reflect on the course of the day and how their previous night's sleep (with reference to their new schedule) affected their ability to function, "for better or worse". Thus, evening entries were completed sufficiently prior to the sleep-onset period to avoid creating excessive sleep preoccupation, or pre-sleep anxiety, proximal to sleep-initiation. Guidelines were purposely kept open to facilitate recording of relevant and interesting topics.

The study rationale was presented to participants as an investigation into the experience of sleep restriction. There was no priming in terms of what information we were particularly interested in; rather, it was emphasised that we wanted to know as much as possible about the experience of SRT implementation (*good* or *bad*), from the individual perspective.

2.4.2. Semi-structured interviews

Four weeks after treatment initiation, participants were interviewed by the lead researcher on their overall experience of treatment. The interview format and schedule was based on published guidelines [45] and consisted of open-ended fixed questions, with supplementary prompts – again permitting exploration of additional interesting and relevant topics. Core questions covered the following areas: treatment understanding and expectations; implementation of sleep schedule; and impact on sleep and daytime functioning.

2.5. Data preparation and analyses

2.5.1. Treatment outcome variables

Dependent variables were screened for extreme outliers and assessed for normality using histograms and boxplots. Logarithmic transformations were performed to correct skewed distributions. The trial aspect of the study was viewed as an intention-to-treat analysis; hence, the last observation carried forward (LOCF) method was used to impute missing data values. Imputation was almost exclusively confined to the three patients lost between post-treatment and follow-up assessments.

Changes in sleep diary variables (sleep-onset latency, SOL; number of awakenings, NAW; wake-time after sleep-onset, WASO; total sleep time, TST; sleep efficiency, SE; sleep quality, SQ), sleep-related questionnaires (ISI, PSQI, GSES), and daytime functioning measures (GSII, DFSAS, OISQ, SF-36) across the three time points were assessed using repeated measures ANOVA. For those variables failing to meet the sphericity assumption, degrees of freedom and corresponding probability were adjusted using the Greenhouse–Geisser correction [46]. Significant main effects were subsequently followed up using paired *t*-tests. Cohen's *d* [47] is reported to provide an indication of effect size (ES) magnitude for baseline to follow-up changes.

2.5.2. Audio-diaries and semi-structured interviews

Audio-diaries and interviews were transcribed verbatim, including pauses, laughter and false starts. Audio-diaries and interviews were analysed separately and according to the framework of thematic analysis [48]. Structured guidelines establish thematic analysis as a flexible method in its own right, unconstrained by theoretical and epistemological underpinnings.

Analysis was a recursive process involving several stages [48]. First, transcripts were read through several times to gain a sense of the whole phenomenon under investigation. On subsequent readings of individual transcripts, significant words and/or phrases were highlighted and bullet points were entered in the margins, creating preliminary coding schemes. After several further reviews of the transcripts, initial notes and codes were collated into themes. Themes were compared across individuals, looking for common recurrent themes as well as inconsistencies and contradicting cases. Themes were refined throughout the analytic process via discussions with another insomnia researcher (also a clinician) and from feedback during data presentation at lab seminars.

Given that the focus was on generating descriptions of the Sleep Restriction Therapy experience, emphasis was initially placed on identifying themes at the semantic or explicit level [48]. These were then subsequently related to, and interpreted in light of, contemporary literature on the behavioural management of insomnia, as well as additional questionnaire findings from the present study.

3. Results

3.1. Participant demographics

Participant (n = 18) demographics are presented in Table 1. The mean age of the sample was 41.9 (standard deviation: 13.2), with a range of 18–64 years. Five participants were male (13 female), and the average insomnia duration was 17.0 (14.4) years. Three participants had problems initiating sleep only, 3 had difficulties with maintaining sleep only, and the remaining 12 suffered from both initiation and maintenance difficulties. Finally, 2 participants (11%) were on prescribed sleep-promoting hypnotics at treatment intake.

3.2. Sleep outcomes

Table 2 provides an indication of main effects of time, post hoc comparisons between assessment points, and related statistical significance.

Table 1
Participant demographics.

Demographics	PI patients (n = 18)
Age (years)	41.9 (13.2)
Gender	5 male/13 female
Insomnia duration (years)	17.0 (14.4)
Insomnia sub-type	
Inititiating	3
Maintaining	3
Mixed	12
Medication	2/18 (11%)

Table 2
Treatment effects and post hoc comparisons for sleep diary and questionnaire variables.

	Baseline		Post-treatment (4 weeks)		Follow-up (3 months)		df	F	P
	M	(SD)	M	(SD)	M	(SD)			
Diary									
SOL (mins.)	41.1	30.6	19.7**	13.8	21.3**	17.3	(1.3, 21.9)	13.08	.001
TST (mins.)	319.8	75.3	323.1	48.8	367.1**,a	65.6	(2, 34)	7.03	.003
NAW	3.0	2.2	1.4***	1.2	1.7***	1.4	(1.4, 24.5)	22.50	<.001
WASO (mins.)	72.0	55.6	29.0**	35.9	26.3***	25.2	(2, 32)	17.21	<.001
SE (%)	64.2	14.4	85.3***	10.5	80.9***	13.9	(2, 34)	23.44	<.001
Sleep Quality (0–4)	1.4	0.6	2.1***	0.7	2.2**	0.9	(2, 34)	15.38	<.001
Questionnaire									
ISI	17.2	3.8	9.7***	5.2	10.1***	6.1	(1.5, 25.8)	24.45	<.001
PSQI	12.6	3.00	8.0***	2.0	7.8***	3.4	(2, 32)	27.73	<.001
GSES	9.3	3.3	5.3***	2.5	5.6***	3.5	(2, 34)	18.47	<.001

** Indicate post hoc significant changes from baseline: $p < .01$.
*** Indicate post hoc significant changes from baseline: $p < .001$.
[a] Contrast with post-treatment mean significant at $p < .05$.

3.2.1. Sleep onset latency (SOL)

There was a significant change over time in mean subjective SOL. As is evident from Table 2, mean SOL for the group decreased significantly by 21 min, from 41 min pre-treatment to 20 min at the post-treatment assessment. This improvement was sustained, and remained significant, at 3-month follow-up (ES = 0.80).

3.2.2. Total sleep time (TST)

Subjective TST remained similar from baseline (320 min) to post-treatment (323 min). At 3 months, mean TST significantly improved, relative to baseline figures, by 47 min (ES = 0.67).

3.2.3. Number of awakenings (NAW)

Number of nightly awakenings significantly decreased from baseline (3.0) to post-treatment (1.4). This effect was maintained at 3-months (M = 1.7; ES = 0.69).

3.2.4. Wake-time after sleep-onset (WASO)

A significant effect of time was again found with mean WASO values. Mean WASO of 72 min at baseline significantly reduced by 43 min to 29 min at post-treatment. These improvements were sustained and remained significant at 3 month follow-up (ES = 1.06).

3.2.5. Sleep efficiency (SE)

Mean SE significantly increased by an average of 21%, from 64% at baseline to 85% post-treatment. Three month average SE (81%) values remained highly significant relative to pre-treatment assessment, with a net improvement of 17% (ES = 1.18).

3.2.6. Sleep quality (SQ)

Small but significant improvements were recorded for subjective sleep quality, from baseline to post-treatment. These benefits were statistically maintained at 3-month follow-up (ES = 1.07).

3.2.7. Insomnia Severity Index (ISI)

Baseline mean ISI values (17.2) indicated the presence of clinical insomnia of moderate severity, according to scale cut-offs. Post-treatment reductions in ISI scores were significant (mean of 9.7 – 'sub-clinical insomnia'), and were maintained at 3 month follow-up (ES = 1.41). In terms of clinical significance, 6/18 (33%) participants scored in the 'no insomnia' range ('remitters') at post-treatment. The number of 'remitters' at follow-up increased to 8/18 (44%). Treatment 'response' rates were calculated based on recently published minimally important difference data for the ISI [49]. The number of individuals evidencing a change of at least six ISI scale points at post-treatment was 66.6% (12/18). This rate of treatment response remained the same at follow-up (66.6%).

3.2.8. Pittsburgh sleep quality index (PSQI)

Similar to the ISI, mean PSQI scores significantly decreased from baseline (13) to post-treatment (8). This improvement was also sustained at the three month assessment point (ES = 1.52).

3.2.9. Glasgow sleep effort scale (GSES)

Mean GSES score changes between baseline (9) and post-treatment (5) were highly significant. Again, this pattern remained robust at three-month follow-up (ES = 1.09).

3.3. Daytime functioning/HRQoL outcomes

Table 3 provides an indication of main effects of time, post hoc comparisons between assessment points, and related statistical significance.

3.3.1. Daytime functioning and sleep attribution scale (DFSAS)

Mean scores on part one of the DFSAS significantly reduced from baseline (18) to post-treatment (12). Comparisons between post-treatment and 3-month follow-up indicated further reductions in associated daytime impairment, which again reached statistical significance, above and beyond post-treatment effects. Effect size magnitude for baseline to follow-up change was large (ES = 1.42). Concerning part 2, the attributional component, repeated measures ANOVA indicated a main effect of time. Mean values significantly improved post-treatment, which were found to be most robust at follow-up (ES = 0.99).

3.3.2. Glasgow sleep impact index (GSII)

Both GSII ranks 1 and 2 indicated main effects of time. Post-hoc testing for rank 1 revealed significant effects at post-treatment and follow-up, with effects being most pronounced at final assessment (ES = 1.21). Rank 2 similarly demonstrated improvements across the three assessments, though effects achieved significance only at the 3-month follow-up (ES = 1.04). There was no within-subjects effect for rank 3, with mean values remaining quite stable over the assessment points.

3.3.3. Occupational impact of sleep questionnaire (OISQ)

The OISQ was completed by 16 (89%) working participants in the study. Mean OISQ values significantly improved at

Table 3
Treatment effects and post hoc comparisons for daytime functioning and HRQoL variables.

	Baseline		Post-treatment (4 weeks)		Follow-up (3 months)		df	F	P
	M	(SD)	M	(SD)	M	(SD)			
DFSAS									
Part 1	17.9	5.3	12.1**	5.8	9.9**,a	5.9	(1.4, 23.4)	11.94	.001
Part 2	24.8	10.2	18.9*	12.1	14.4**	10.7	(2, 34)	8.59	.001
GSII									
Rank 1	31.4	17.3	53.3*	24.6	57.3**	24.9	(2, 32)	8.07	.001
Rank 2	36.0	18.0	51.0	26.1	57.1**	22.3	(1.5, 24.9)	6.03	.013
Rank 3	47.4	19.6	52.0	23.1	55.5	25.0	(2, 34)	1.35	.274
OISQ									
	42.8	18.4	26.8*	13.2	26.7**	24.8	(2, 30)	4.66	.017
SF-36									
PF	91.1	10.1	91.4	9.5	91.3	8.7	(2, 34)	0.02	.985
SF	69.5	25.8	74.3	24.4	77.1	24.3	(2, 34)	1.21	.312
RP	76.8	21.8	86.4*	12.7	83.9	15.5	(2, 34)	3.78	.033
RE	73.2	21.3	77.8	16.7	82.6*	18.3	(2, 34)	3.15	.055
MH	62.8	15.0	68.3	13.5	73.3*	16.4	(2, 34)	5.05	.012
VT	39.7	12.9	46.5*	16.8	53.0**	18.8	(2, 34)	6.77	.003
BP	75.8	23.8	78.9	26.9	78.5	27.6	(2, 34)	0.13	.878
GH	63.8	16.7	66.5	18.8	69.4	14.9	(2, 32)	1.47	.244

PF, physical functioning; SF, social functioning; RP, role-physical limitations; RE, role-emotional limitations; MH, mental health; VT, energy/vitality; BP, bodily pain; GH, general health perceptions.
* Indicate post hoc significant changes from Baseline: $p < .05$.
** Indicate post hoc significant changes from Baseline: $p < .01$.
[a] Contrast with post-treatment mean sig. at $p < .05$.

Table 4
Side-effect frequency and average daytime interference rating for checked symptoms.

Symptom	% of sample reporting symptom	Daytime functioning interference rating (0–4)
Low mood	61	1.55
Fatigue/exhaustion	100	2.56
Extreme sleepiness	94	2.58
Feeling agitated	50	1.78
Bodily pain	33	1.17
Headache/migraine	72	1.31
Euphoria/intense increase in mood	39	1.29
Reduced motivation/energy	89	1.88
Changes in hunger/appetite	72	2.00
Blurred vision	22	1.00
Dizziness	28	1.40
Feeling irritable	61	2.09

post-treatment by, on average, 16 points. This effect remained robust and significant at three month follow-up (ES = 0.74).

3.3.4. Short-form health-survey 36 (SF-36)

Three dimensions of the SF-36, 'vitality/energy' (VT), 'mental health' (MH), and 'role-physical limitations' (RP) evidenced main effects of time, while 'role-emotional limitations' (RE) just narrowly missed significance at the 5% alpha level ($p = .055$). Both RE (ES = 0.48) and MH (ES = 0.67) dimensions achieved statistical significance at 3 months (relative to baseline values), whereas RP evidenced statistically significant improvements at the post-treatment assessment point only. For the Vitality dimension, both post-treatment and 3 month values (ES = 0.82) indicated significant improvements relative to baseline levels. There was no effect of time or trends for the other four dimensions.

3.4. Treatment side-effects

Impairments as a consequence of SRT initiation were common. Indeed, for eight out of the twelve listed domains, $\geqslant$50% of the sample reported sleep restriction-related impairment (see Table 4). The mean number of endorsed side-effects for each patient was 7.2 (SD = 2.4; range = 3–11). The four most commonly reported symptoms were 'fatigue/exhaustion' (100% of sample), 'extreme sleepiness' (94%), 'reduced energy/motivation' (89%) and 'headache/migraine' (72%). Subsequent ratings of the extent of 'side-effect' interference revealed that 'fatigue/exhaustion', 'extreme sleepiness', 'feeling irritable' and 'changes in hunger/appetite' interfered 'somewhat' to 'much' (mean ratings 2–3) with everyday functioning (Table 4). Six (33%) participants added, qualitatively, additional domains of impairment. These spanned the categories of: pain/discomfort, temperature regulation, word-finding difficulties, social interaction impairment, greater illness susceptibility, 'hangover'-like effects, problems with concentration and fatigue at unusual times.

We also carried out exploratory correlation analyses between number of experienced side-effects, relative daytime interference ratings for side-effects [i.e. (interference score for checked symptoms/total possible score for checked symptoms) $*$ 100], and sleep improvements (baseline to post-treatment). It was found that a higher frequency of side-effects was associated with a greater magnitude of change on the PSQI ($r = .51$, $n = 17$, $p < .05$; see Fig. 2) and, to a lesser (non-significant) degree, sleep efficiency ($r = .44$, $n = 18$, $p = .065$) and ISI ($r = .33$, $n = 18$, $p = .185$) values. Other measures of sleep continuity were weakly and non-significantly related to side-effect frequency. Greater relative side-effect daytime interference ratings were significantly related to both improvements in PSQI scores ($r = .54$, $n = 17$, $p < .05$) and reductions in sleep effort, as measured by the GSES ($r = .48$, $n = 18$, $p < .05$). Relationships between side-effects and three-month sleep outcomes were weak in magnitude and non-significant.

3.5. Qualitative results

At the outset of the study, it was our intention to recruit approximately 50% of the total sample into the qualitative component of the trial. Participants were initially selected based on random allotment, within each treatment group, but because of time constraints

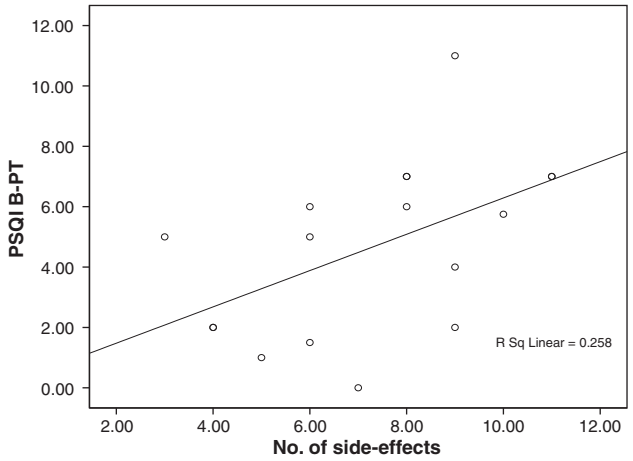


Fig. 2. Scatterplot of relationship between PSQI change scores (baseline to post-treatment) and number of experienced side-effects.

and recruitment difficulties all participants within the final few treatment groups were selected to take part. Hence, 14 (78%) participants (4 male; 10 female) completed both audio-diaries and interviews, which was slightly more than predicted from the outset, though within standard limits for qualitative research.

3.5.1. Audio-diaries

In week one of SRT, participants, in total, recorded 179 diary entries. Transcription produced ~29,100 words of data. Thorough analysis of both morning and evening entries revealed three major themes, each with respective sub-themes/categories. Selected direct quotes are presented as examples to support generated themes and pseudonyms are used to protect patient identities (see Table 5).

3.5.1.1. Daytime side-effects: 'it's made it worse'. Participants described, on the whole, feeling and functioning worse during week one of therapy relative to pre-treatment, making strong causal attributions to SRT. A reduction in sleep opportunity typically translated into less total sleep time, which had implications for the remainder of the day. Although impairment perhaps indicated that the therapy instructions were being followed and possibly beginning to work – in the sense that sleep pressure was being applied – this was clearly not without consequences. Indeed, these consequences were conveyed on awakening, proximal to the sleep-period, with common references to *'zombie'* or '*hangover*'-like states.

Table 5
Major themes/sub-themes from thematic analysis of audio-diary entries and supporting example patient quotes.

Theme	Sub-themes	Example quotes
Daytime side-effects: 'it's made it worse'	On awakening	"... I feel like I'm drunk at the moment, my head's quite swimming, and not thinking very straight at all, I find it quite hard to write in this bit of paper too" (Bill, 68–70) " In terms of sort of quality of sleep, I actually feel this morning as if I've got a hangover, and I didn't drink anything last night, I've got a headache and actually I feel quite sick" (Jennifer, 23–25)
	Throughout the course of the day	"Woke up bright and breezy, half six, Tuesday morning, raring to go, got into the car... and within twenty minutes I was absolutely exhausted, so bad that I swear I was nearly falling asleep all the way to work. It was torture, I was cross-eyed, eyes drooping, driving" (Sarah, 122–126) "Hi, it's Sunday night, about ten o clock and I'm absolutely exhausted, I've had a really bad day and never left the house the whole day, just felt so bad, I don't know if I can stay up till one o' clock. I just feel totally, at the moment, terrible" (Gillian, 58–60) "The restrictive programme has affected my ability to function, it's made it worse forme working, its been very hard at work to focus and be as sharp as you should be" (Bill, 187–188)
Adjustment to new sleep schedule	Challenges to adherence	"...this regime would be quite easy to do if you didn't have any social life, as soon as you're going out late, or drinking, or whatever, it does sort of seem to go to pot a bit. I'll certainly restart it again tonight. I think that's the difficulty come the weekend, it all goes a bit haywire, so whether that's undoing any good that it's done through the week, I possibly would think so..." (Jennifer, 196–198) "Em, just talking about the experience of implementing my new bedtime routine, I feel it's just been really negative so far, and although I can understand that it probably will work eventually, at the moment I just feel really bad. I've had about an hour and a half to two hours sleep last night, and just feel really bad today, and already just worrying about how on earth I'm gonna stay up till one o clock this morning" (Gillian, 65–71) " I'm not driving tomorrow, but frankly in order to function at work tomorrow, I'm seriously considering going to my bed probably about twelve tonight, that's about as late as I can cope (with), because I can't do another, morning I suppose like today – that was really grim, it was absolutely dire..." (Sarah, 132–135)
	Coping strategies to help facilitate adherence	"I made a point of staying with a friend to keep me awake for a while tonight... they're off work... it's made me stay awake and not be tempted to go to bed earlier than I should do" (Jim, 55–57) " I put more lights on than I normally would cos normally I would have it sort of quite dark in the lounge but I reckon more light is probably a good thing, just to keep me awake – and I think it's working" (Sarah, 39–41)
Evolving changes to the sleep experience	'Unusual' feelings of sleep pressure	"these napping situations, it's not like I'm sitting down wanting to nap but it's just that I'm tired and I'm watching the telly and I'm drifting off and that's so unlike me" (Hannah, 118–119) "this afternoon I was very dosey and sort of quite tired because I had sort of struggled a little bit with sleep last night, I got about three and a half hours all in, and this morning found it quite hard to get up. Which (is) a little bit unusual for me" (Jim, 46–48) "I got really tired about three o'clock but not the usual tired that I get – just can't be bothered doing anything – but more I could fall asleep at my desk and my eyes really gritty tired" (Lisa, 49–50)
	Changes to sleep quality: could this be working?	"I've got to admit, despite being up twice during the night, I'm having a deeper sleep than I normally get and that's been the same for the last couple of nights, although it's shorter, it's actually deeper" (Maria, 60–62) "I do feel that the quality of my sleep last night was better than before I started the programme" (Sarah, 142–143) "I'm feeling quite good today, and I had a good sleep last night, I didn't wake up at all during the night, and I went out like a light at twelve thirty which was very unusual for me, but I think it was because the whole night I was craving sleep" (Hannah, 144–145)

Sleep restriction therapy negatively impacted on numerous daytime functioning domains. On an elementary level, participants reported feeling exhausted and fatigued, experiencing difficulties with concentration and memory, as well as depressed mood. These symptoms, combined, had the net effect of impairing aspects of job performance and ability/effort to interact socially. Furthermore, 5/14 patients (36%) made reference to impaired driving abilities at least once during the course of the week. These references, in some cases, reflected difficulty maintaining wakefulness during driving, but more typically focused on compromised concentration and slowed reactions to ongoing changes in the driving environment.

3.5.1.2. Adjustment to new sleep schedule. This theme captured the practical experience of implementing set threshold and rising times. Participants reported a number of issues that made adhering (rigorously) to the programme incredibly challenging, and which frequently led to non-adherence. Such factors included: spending extended amounts of time on own, particularly at weekends; running out of activities to do during extra hours (boredom); inability to stave off sleepiness until set bed time; fear of disrupting partner by entering the bedroom after they had initiated sleep; staying out late, drinking and socialising; feeling pressure to *'perform'* when going to bed so late, and having a short period of time to obtain adequate sleep; and, finally, no indication of re-bound sleep over several nights of adherence, in parallel with accumulating daytime difficulties.

A sub-theme captured strategies that patients put in place to facilitate adjustment and promote adherence to sleep restriction instructions. These again were numerous and varied, but included the following: sleeping in a separate room from partner; partner staying up late to accompany and motivate the participant to adhere; scheduling activities or making modifications to activity levels to promote alertness, and to fend off sleep prior to set threshold time; reducing alcohol intake and late nights out; and seeking out other people's experiences of sleep restriction via the internet for additional reassurance/motivation.

3.5.1.3. Evolving changes to the sleep experience. Finally, participants reflected on how sleep quality, feelings of sleep pressure, and views on sleep, were beginning to change. These comments were made, in nearly all cases, towards the end of the first week as improvements in sleep became apparent. Changes in sleep pressure were conveyed with reference to the perceived ability to sleep longer than usual (past set rising time), and, in particular, *'unusual'* feelings of 'tiredness' and involuntary napping.

Table 6
Major themes/sub-themes from thematic analysis of post-treatment interviews and supporting example patient quotes.

Themes	Example quotes
'At the end of my tether'	"...just my mood and kinda temperament and it affected my everyday life, I thought, quite badly... and I thought things could be better if my sleep was better, and it seems to be working" (Ross, 6–7) "I've tried herbalism... yeah just trying herbs and things, potions, but as I say I think it's a whole lot of rubbish...somebody told me it would work, it worked for them, so you go and try it, it didn't work" (Bill, 41–43) "I had a wee bit of a fright about a few months ago, I didn't fall asleep at the wheel, I would say I did, but it was almost too quick... I was just really tired, and I remember the music was blasting, window was down, and I must have felt drowsy, and then for a second my head was down and then I woke up, and I touched the kerb with the car, and I think I got a fright and I just thought I have to do something about this" (Hannah, 24–27)
'This is a sleep restriction programme'	"...the sort of limited amount of sleep that you were giving us in the first night sounded pretty horrific, and it was, the first week was really tough, eh, but I think I could see the sense then, but I didn't see it immediately you know, it didn't hit me immediately that it was going to work, that sort of came in the second week' (David, 16–19) "...that week one needs to come with a health warning"(Sarah, 154) "driving was a nightmare, and I've never ever had an issue with driving before" (Bill, 84)
Adherence & adjustment	"I suppose rationally and logically, it did seem a good idea, but then I think the first week of actually doing it, I felt worse, and so you kinda, there's a temptation there to think 'och this is not working', like 'give up now'" (Lisa, 17–19) "my husband was determined I was sticking at it, you know, cos he, when I was sitting at night-time, and I was like almost falling asleep... he kept shouting at me 'get up', 'wake up, don't go to sleep', you know it was kinda like that, but I felt it very difficult to get up and do anything, I was actually too tired to" (Gillian, 135–138) "...there's been a few mornings where you're oh you're thinking, especially like Saturday and Sunday, 'there's just no way I want to get out of my bed', but then you think 'well no', especially at the weekend cos then if I lie long this morning then I don't get to sleep tonight, it's just going to start the whole thing again, that's, I suppose, the motivation, the fact that you think 'well if this makes you sleep five hours through the night, then just get up" (Lisa, 273–278)
'I actually want to go to bed now'	"I'm sleeping longer, and going into a deeper sleep, I think, when I wake during the night it's only... it's less frequent, and it's easier to get back over again... previously if I woke up during the night I'd be worried about trying to get back over again, and I'd be thinking about it, but because you're so tired by this sort of programme then you actually get back over much quicker and it seems to work" (David, 62–66) "you're so knocked that you don't have the anxiety to be anxious at that time of night, really, I'm so looking forward to going to my bed, when I get to whichever hour my times up, I say 'Yes, times up, going to bed' (laughs) so that's probably the highlight of the day" (Bill, 167–170) "I think I've altered my, I just seem to have gone onto a different plain when it comes to my attitude to sleep, another thing though I've realised is that it's possibly true that I just don't need 8 h of sleep, or even seven and a half hours sleep, or even seven, possibly I only need about six and a half hours sleep" (Louise, 78–81) "I'm not concerned, I'm not worried about not sleeping, because I know that when I go to bed I will sleep" (Sarah, 188–189)
Daytime functioning: a thermometer for success?	"I do feel like a normal sleeper, which is bizarre and great, this has been huge for... like driving, I feel like I'm calmer you know I don't get road rage, I don't... I'm not as bad as what I was, I'm taking less risks in the car, I'm not in a rush, because I'm not as anxious, I'm not on edge, I'm just a bit more chilled I think, and I think the sleep helps to centre yourself." (Hannah, 353–357) "much more sort of... quite motivated for work and stuff, eh, it definitely has had a very positive effect on my sort of daily life" (David, 87–88) "I think now I'm more likely if I've planned something for after work, to do it, I'm meant to, I've paid for most of this year west Dunbartonshire council money for a gym membership, that I ('ve) never been in, so I'm now back three times a week!" (Lisa, 185–187) "If I can build it up to six and a half I think, six and a half hours, I'm gonna feel like I can function, right, so I guess I'm thinking in my head that's where my functioning range is, six and a half hours is gonna allow me to function, eh, if I can better that then I'm hoping that's where the energy and the zest for life is going to come from" (Bill, 325–329)

This increased sleep pressure was linked to self-reported improvements in sleep, particularly reduced sleep latencies and less frequent and lengthy awakenings. It was thus towards the end of week one that patients were beginning to realise that the programme might actually be working; that the acute exhaustion and increased wake time might be a worthwhile endeavour.

3.5.2. Interviews

The same 14 participants also took part in a face-to-face interview with the lead researcher at the post-treatment assessment point. Verbatim transcription produced ~66,400 words of data. Following strict coding, a number of themes were generated, capturing impressions and experiences of sleep restriction therapy (see Table 6 for a summary of main themes and supporting quotes).

3.5.2.1. 'At the end of my tether'. Participants discussed why they had decided to take part in the sleep restriction intervention; descriptions tended to be dominated with references to daytime and quality of life impairments, as key catalysts. Related to this, it emerged that patients had tried a range of other treatments, including medication/herbal strategies, that had, for the most part, failed to effectively alleviate insomnia symptoms.

3.5.2.2. 'This is a sleep restriction programme'. Initial subjective impressions of SRT were that it was *'logical'*, and *'made sense'*; yet, for some, it did still feel *'counter-intuitive'*, relative to how they have typically tried to cope with their insomnia [i.e. extending time in bed/'catch-up' sleep: *"...telling somebody with insomnia 'stay up late' is like turkeys voting for Christmas, don't be ridiculous"* (Sarah, 432–433)]. Others, although, again, finding it logical, thought it seemed *'too simple'* to be effective *('insomnia is a chronic problem')*. This perceived ease or simplicity failed to translate into actual experience, as the first 1–2 weeks of the programme were described as incredibly difficult, and, by some, as *'hell'* and even *'torture'*. As noted in audio-diary entries, participants similarly reflected on the negative side-effects encountered in the early stages of the programme, particularly relating to extreme fatigue and sleepiness, impairing nearly all aspects of daytime functioning, including subjective driving ability. These impairments were of greater magnitude than pre-treatment functioning levels.

3.5.2.3. Adherence and adjustment. Adherence to set rising and threshold times was affected by a number of variables. These included, but were not restricted to, the following: experiencing pressure to sleep in such a condensed period, coupled with concern for next-day-functioning; external fluctuating stressors and commitments; actual felt impact of restricted sleep opportunity on daytime functioning; and boredom associated with extra hours of wakefulness. Weekend adherence was particularly difficult for participants, being adversely impacted by: socialising/alcohol consumption, the prospect of being alone and awake for such a lengthy period of time and the awareness of returning to work at the beginning of the week (catastrophizing about possible daytime consequences and coping).

Despite these obstacles, participants developed strategies to help promote adherence, such as refraining from going out/socialising late at night, not allowing self to relax in comfortable positions prior to 'threshold time' (to prevent 'dosing'/napping), keeping active and setting chores and negotiations/discussions with partner to facilitate adjustment.

Another factor that emerged was that non-adherence (mostly sleeping-in and napping) contributed to sleep-onset problems the subsequent evening, when attempting to 'restart' the programme. This 'experimental feed-back' acted as a negative reinforcer, helping to reduce non-adherence and motivate continued implementation of treatment instructions.

3.5.2.4. 'I actually want to go to bed now'. For the majority of participants significant changes to sleep tended to occur in the second or third week of treatment. These related to commonly-measured sleep parameters, such as reduced sleep latency, decreased wake-time after sleep-onset, and decreased number of awakenings. Changes to sleep, however, also extended to more 'subjectively' expressed aspects of sleep, such as quality and depth of sleep, predictability that sleep will happen, and unusual feelings of sleep pressure and *'craving'*. Interestingly, for many participants, this paradigm shift to *'looking forward'* to going to bed reduced sleep anxiety/distress and pre-occupation, both at sleep-onset and during awakenings.

Indeed, even for those few participants who felt they had not benefited from the sleep restriction programme, they did, however, describe changes to their attitude towards sleep, particularly sleep need. Having encountered side-effects during restriction, they tended to have a 'response-shift' to their own sleep duration; engendering *'this could be worse'* phenomenon, which seemed to relieve some pre-occupation/concern with sleep.

For most participants, improvements were considered to be ongoing, and their perceived-capacity to obtain more sleep, and build on gains, was a prime motivator for continuing with the programme (after formal monitoring ceased).

3.5.2.5. Daytime functioning: a thermometer for success?. Although improvements in functioning tended not to be as robust as changes to sleep, at least at the time of interview, many participants reported positive modifications to aspects of daily living, such as having more energy, being more organised, being less anxious, enhanced coping skills, adopting a more positive outlook, and even comments by significant others concerning physical appearance. These improvements were typically just evolving 1–2 weeks prior to interview, in a slight time-lag behind sleep symptom changes.

Impact on functioning was considered a thermometer by many to gauge therapy success. The continuation of a *journey*, an ongoing treatment process, was often made with reference to obtaining future improvements in aspects of functioning. Those individuals who considered SRT to have been unsuccessful in terms of improving night-time sleep, or that only reported minimal improvements (at least up until the interview), voiced that functioning was detrimentally affected during times of adherence (both acutely and throughout the four-week period). In extreme cases this meant a subsequent shift towards baseline sleep schedules, accompanied by a return to 'normal' levels of functioning; while others voiced determination to commit to the programme despite negative daytime experiences.

4. Discussion

The aim of this mixed-method study was to investigate, using both quantitative and qualitative methodologies, the patient experience of sleep restriction therapy for insomnia. We combined sleep-diaries, questionnaires, audio-diaries and semi-structured interviews in an attempt to understand the impact of SRT on sleep and functioning, and, crucially, to systematically record patient accounts of treatment implementation.

4.1. Sleep-related changes

All major sleep diary variables significantly improved from baseline to post-treatment (excluding TST), and these improvements were maintained at three months. By three months, TST

demonstrated a significant gain of 47 min, on average, relative to baseline values. Overall, sleep changes were comparable (and for some variables, larger) in terms of magnitude to those obtained in full CBT-I interventions [2] and previously published trials of sleep restriction therapy [34]. Moreover, global insomnia disorder severity (assessed using the ISI) indicated mean values for the group to be in the 'sub-clinical insomnia' range at post-treatment and follow-up, relative to baseline levels in the 'clinical moderate' range. The clinical significance of these changes was confirmed with 44% and 67% of patients being classified as 'remitters' and 'responders', respectively, at three month follow-up.

Semi-structured interviews with patients, post-treatment, supported these changes in nightly sleep parameters, specifically concerning reduced sleep-onset latencies, decreased number of awakenings (also reported as a reduced ability to remember awakenings) and decreased amounts of wake-time during the night. Interviews also revealed changes in subjective sleep quality; with participants providing rich descriptions of sleep as being '*deeper*' and '*more efficient*', as well as reporting dampened mentation at sleep-onset and during middle-of-the night awakenings. Such 'qualitative' changes and increased predictability of sleep are likely related to the harnessing of homeostatic sleep pressure and circadian re-alignment, both desired targets of sleep restriction therapy [6,7,10,50].

Importantly, participants described simultaneous modifications to how they perceived sleep. The feeling of '*craving*' sleep – as a consequence of it being denied/restricted – represented a significant shift from how participants typically viewed the sleep-onset and sleep period. In turn, this led participants to describe having reduced anxiety and worry when initiating sleep, and during middle-of-the-night awakenings. Sleep restriction may work, in part, then, by modifying – through partial sleep deprivation – the evolved threat value that sleep and associated bedtime routine have come to represent in those with primary insomnia, ultimately restoring sleep automaticity. The knock-on effect of '*craving*' sleep, in terms of reduced pre-sleep anxiety and arousal, coupled with increased nightly sleep predictability, may well relate to the robust decreases in the application of 'sleep effort' (as measured by the GSES), a concept central to recent cognitive models of psychophysiological insomnia (cf. [27,39]).

Related to this, it also emerged that some patients underwent an adjustment process with respect to their sleep need during SRT. Firstly, functioning worse in the first week than they previously had done, some patients began to feel perhaps their sleep problem was not as problematic or obstructive as once thought, which seemed to provide some level of reassurance, despite no obvious or immediate improved modifications to sleep. Secondly, experiencing improved sleep quality, yet in the face of overall shorter total sleep time, led some to question their perceptions of individual sleep need. Thus, to build on Edinger and Means' [10] description of 'pathways' implicated in CBT-I response, we also suggest, based on our participant descriptions, that SRT may have secondary or parallel effects on both 'inhibitory factors' (particularly conditioned arousal) and 'cognitive factors/dysfunctional beliefs', thought to be important in the aetiology and maintenance of insomnia. This, of course, requires additional empirical testing in the context of isolated component intervention studies.

4.2. Side-effects and daytime functioning/HRQoL

Functioning fluctuated over the course of the four-week period. Responses from our newly developed side-effects checklist indicated that at least one half of participants experienced eight of the 12 listed symptoms *as a consequence* of sleep restriction therapy during week one. Fatigue, extreme sleepiness, and reduced motivation/energy were the most commonly experienced difficulties, and, along with irritability and changes to hunger/appetite, negatively interfered with daytime functioning (to the greatest degree). Though difficult to make direct comparisons, these acute effects do appear to be substantially higher than rates reported in some pharmacotherapy studies [51]. *In vivo* diary reflections corroborated these accounts, with participants describing, at length, impairments in occupational performance, social functioning and everyday duties, citing exacerbated levels of fatigue and sleepiness and their subsequent downstream effects on cognition as responsible. Of particular prominence, more than one-third of our sample described, during diary entry recordings, concerns with driving ability; reflecting issues with maintaining wakefulness, impairments in concentration, and slowed reactions to changes in the ongoing driving environment. With numbers of this magnitude, it is clear that SRT is not an intervention with just mild and trivial effects, and it remains to be determined if such a treatment approach (at least acutely) is associated with a 'spike' in automobile accidents (as reported with hypnotic use: [52]). Clearly, the risk exists. It would be interesting to qualitatively track patients assigned to active hypnotic therapy to compare patient narratives and experiences across treatment modalities.

To the best of our knowledge, side-effects encountered during CBT-I have not been adequately described or investigated in previous literature. Perlis and colleagues [19], in their randomised trial of modafinil as an adjunctive to CBT, indicated that during week one of treatment, sleepiness, as measured by the Epworth Sleepiness Scale (ESS), approached pathological levels (ESS = ~10.5 versus a baseline of ~7), an effect that was in fact attenuated in the CBT plus modafinil group. The present study is the first to reveal, in-depth, the descriptive nature and magnitude of experienced side-effects that were attributed to the programme. Interviews suggested that for the majority of participants, side-effects subsided 1–2 weeks into treatment initiation as sleep parameters began to stabilize and improvements in sleep quality evolved.

One intriguing finding was that the number of checked side-effects positively correlated (small to moderate strength) with ISI, PSQI, and sleep efficiency change scores, from baseline to post-treatment. Side-effect daytime interference ratings were also significantly associated with PSQI and GSES change scores (again moderate strength). Although it is not entirely clear why this association exists, we can think of at least three possible explanations for this relationship. First, it could be that those who tend to monitor bodily and mental symptoms are more likely to detect and report both experienced side-effects and improvements in sleep quality. Second, and related to this, it could be that side-effects provide some validation that the treatment is actually working (in a similar manner to a drug, for example), leading to a relative coupling with acute improvements in sleep quality. This could also help explain why side-effects related weakly to 3-month outcomes, where this relationship could be likely influenced by many other extraneous variables, being sufficiently detached from the experience of acute implementation. Finally, it may be that side-effects provide an index of acute adherence to set threshold and rising times and hence mediate this relationship with the outcome; the association with decreased sleep effort gives further credence to this argument. Of note, however, this 'side-effect-outcome' account runs partially counter to the findings of Perlis et al. [19], who found that blocking/attenuating sleepiness using modafinil did not interfere with CBT-I efficacy; concluding that sleepiness may not be essential for CBT outcome. This finding, of course, does not rule out additive effects of modafinil properties beyond its stimulant action, such as possible mood enhancement effects [53], as well as increased levels of activity. Interestingly, the authors also reported a tendency for this group to be more adherent to bedtime instructions. Future studies must examine the

mechanisms of sleep restriction and predictors of response, taking into account the acute effects of implementation.

With respect to daytime functioning outcomes, significant improvements were observed on the DFSAS (parts 1&2), GSII (ranks 1&2), OISQ, and three domains of the SF-36 (role-physical, mental health and vitality/energy; role-emotional evidenced a trend). Intriguingly, changes were most pronounced on our newly developed scales (DFSAS, GSII), which may suggest enhanced sensitivity of these measures – both based on words/experiences of insomnia patients – in detecting daytime difficulties relevant to those with insomnia disorder. Overall, improvements tended to be more robust at three months than post-treatment, which may reflect the overlap between questionnaire reference period and experienced side-effects during the acute stages of treatment. This is an important consideration when assessing CBT-I gains directly following the 'active' phase. From semi-structured interviews, participants were beginning to notice improvements in domains of functioning three to four weeks into the programme, describing positive changes in energy levels, fatigue and aspects of work life and social functioning.

4.3. Adherence

Our use of qualitative methodologies provided insights into how participants adjusted and adapted to their new sleeping schedule. Audio-diary entries were particularly interesting because we were able to track experiences over time, from day 1, and gain access to moments of adherence and, importantly, non-adherence. Recent work by Vincent and colleagues [16] revealed that perceived barriers to sleep restriction and stimulus control treatment engagement (measured with a questionnaire post-CBT) predicted self-report adherence; here we were able to capture the nature of encountered barriers, in real time. Specifically, negative impact on functioning, an inability to stave off sleepiness prior to bedtime, and boredom and loneliness during extra hours were all prominent reasons for non-adhering. Such factors also affected those who were adherent, representing general challenges of sleep restriction therapy implementation.

Side-effects interacted with going to bed late, so that impaired daytime functioning not only made staying up until a late set bed time incredibly difficult, but also created pressure and anxiety for participants when attempting to initiate sleep. They were faced with both the prospect of only having *x* hours to 'obtain' sleep, and the experience of continued, and possibly enhanced, impairments the following day. Thus, for some, the normal catastrophizing about daytime consequences (cf. [54]) was in fact exacerbated during the early stages of sleep restriction, which discouraged rigorous adherence. It would seem important, therefore, to identify those who do not respond in the first few days of treatment, to prevent complete or partial disengagement from treatment instructions. Indeed, a few participants, during interviews, did indicate that if there were to be increased therapist contact during active treatment, it would be in the first week.

In this regard, it would also be a worthwhile research endeavour to extend the early work by Perlis and colleagues [19], to determine if additional stimulant therapy can attenuate daytime impairments, improve adherence, and (possibly) enhance outcome. Of course, activation therapies may not necessarily be restricted to pharmacological intervention. For example, some components of fatigue interventions used with cancer patients undergoing chemotherapy could also be applied, such as specific exercise activities [55]. Interestingly, during interviews and audio-diary entries, participants described a number of *countermeasures* they had developed, which included keeping active, engaging in discussion, setting chores, and increasing light exposure, all with the primary aim of improving alertness to promote adherence.

4.4. Implications for clinical practice and future research

There are several implications for clinical practice arising from this work. Firstly, patients need to be made fully aware of how difficult the sleep restriction programme can be, as well as the possible risks/dangers associated with the acute stages. Our participants' initial perceptions of the programme when explained – although positive and logical – were, typically, far removed from the reality of implementation. Perhaps vignettes from real participants, like those in the present study, would give the prospective CBT-I patient an insight into some of the challenges involved.

This also raises the issue of whether the *'expert patient'* (Department of Health [56]) may have a role in treatment preparation, as a way of reducing attrition and enhancing adherence. That is, in addition to vignettes, participants ('graduates') who had previously taken part in CBT-I (and benefited) could speak with prospective patients about the treatment experience and implementation. This could be in the form of a recorded video clip, for example, shown to patients during CBT-I sessions. A similar approach is currently used in some respiratory clinics to inform sleep apnea patients of the benefits of CPAP therapy, prior to initiation (e.g., [57]). Finally, supplementary motivational interventions may also have a place in the behavioural management of insomnia; preliminary data suggests motivational interviewing, for example, is effective in improving CPAP adherence [58]. Application to CBT-I is worthy of investigation.

Although, admittedly, a small sample, results indicate that a brief sleep restriction intervention can, in isolation, improve aspects of functioning and sleep. This supports other published controlled studies that have used similar brief CBT-I packages (two sessions), documenting substantial treatment gains [59,60]. Though more work is clearly required, this literature and the present findings suggest that a brief (low-resource) behavioural intervention may be delivered as a first line treatment in an attempt to widen access to evidence-based non-pharmacological insomnia treatments.

This work also has important implications for research. For example, the finding that SRT modifies sleep-related anxiety, arousal and perceptions demands greater attention in future work with a range of assessments. In particular, serial measurement of attentional bias and self-report questionnaire measures of pre-sleep mentation and applied sleep effort, over the course of sleep restriction, would be a worthwhile endeavour. Furthermore, the question need to be posed, does sleep restriction modify physiological arousal prior to sleep and also during the day? And what is the relationship, if any, with daytime functioning outcomes? It would also be informative to assess objective (cognitive) functioning throughout the intervention period, helping to establish whether acute restriction is associated with measurable impairments (as our qualitative reports suggest), and also whether therapy reverses possible baseline neurobehavioural deficits. At the Glasgow Sleep Centre we are currently conducting an in-lab assessment of SRT to investigate whether therapy induces acute objective (and subjective) daytime impairment, and to establish when these impairments resolve, carrying out assessments on a weekly basis. A related issue will be to determine, in future controlled studies, the number of patients who report side-effects during active treatment compared to an adequate control group, and if patients are prompted to seek medical attention for experienced side-effects.

4.5. Limitations

The results of the present study have to be viewed in the context of several limitations. In relation to the diary and

questionnaire data, particularly in light of the strong results, it needs to be borne in mind that this was an uncontrolled study with a small sample size, and that non-specific and other placebo-related effects cannot be ruled out without the inclusion of an adequate control group. Since the study was primarily interested in assessing the patient experience of treatment, in a valid patient-centred manner, conventional controlled trial methodology was not considered appropriate at this first stage of investigation. Such methodological rigour will be essential, going forward, to fully probe the side-effects issue (see below).

Similarly, procedural aspects of the treatment protocol may have impacted results in two important ways. Firstly, audio-diary entries may have acted as a proxy for therapist contact, enhancing motivation/support to adhere with the programme, and ultimately moderating outcome. Secondly, post-treatment interviews were conducted by the therapist (also the researcher: SK), which may have created 'demand characteristic' behaviour on the part of interviewees. On this point, however, it was made explicit to participants that we were interested in their experience, 'good or bad', and that all information was interesting and relevant.

It must also be made clear that our side-effects questionnaire differs somewhat from other measures used to assess side-effects, where the investigator will typically record adverse events and subsequently evaluate seriousness. Here, the questionnaire emphasis was on patient attributions to sleep restriction therapy in accounting for experienced impairment. This may have potentially lead to an over-reporting of side-effects by patients. Yet, at the same time, it may have also lead to the under-reporting of side-effects that were not patient-attributed to the intervention. Given that the study was designed to understand the patient experience, we do not necessarily regard these as major limitations; however, we would caution against comparing our side-effect findings with published rates from different treatment modalities. That being said, we do envisage the situation where our questionnaire could be used to prospectively compare isolated components of CBT-I (e.g. sleep restriction, stimulus control, relaxation) with, for example, 'ingredient-free' interventions (sleep hygiene, sleep education, sham biofeedback) to ascertain patient-attributed side-effects. One final point, relating to the side-effects questionnaire, concerns possible perceived synonymity between the items 'extreme sleepiness' and 'fatigue/exhaustion', leading to exaggerated rates for both. This appears unlikely in our sample given that audio-diary entries from patients reflected, clearly, complaints of both sleepiness and fatigue. Nevertheless, future questionnaire studies should attempt to define these terms on first presentation to minimise ambiguity (for a good example, see the Flinders Fatigue Scale; Gradisar et al. [61]).

Moreover, the sample was largely white, middle class and well-educated; important factors when considering aspects of SRT implementation, motivation and adherence. The generality of the findings may, therefore, be restricted. Finally, some of the questionnaire measures used in the present study were non-validated and require further testing with larger samples of individuals with insomnia. On this note, the side-effects inventory is currently undergoing reliability testing in a further treatment study at the Glasgow Sleep Centre.

5. Concluding remarks

The present study provides novel data on the implementation and experience of sleep restriction therapy for insomnia. The triangulation of quantitative and qualitative methodologies gives considerable credence to the findings. This data has several important implications concerning SRT mechanisms of action, experienced side-effects and adverse events, factors impacting adherence and, finally, perceptions of benefit. This work underlines the value of using mixed methodologies to explore poorly understood and/or under-researched phenomena.

Disclosure statement

This was not an industry supported study.

Espie has been a consultant or served on an advisory board for Sanofi-Aventis, GlaxoSmithKline, Actelion, Takeda, and Lundbeck. All other authors report no financial conflicts of interest.

Conflict of Interest

The ICMJE Uniform Disclosure Form for Potential Conflicts of Interest associated with this article can be viewed by clicking on the following link: doi:10.1016/j.sleep.2011.03.016.

References

[1] Morgenthaler T, Kramer M, Alessi C, et al. Practice parameters for the psychological and behavioral treatment of insomnia: an update. An American Academy of sleep medicine report. Sleep 2006;29:1415–9.
[2] Riemann D, Perlis ML. The treatments of chronic insomnia: a review of benzodiazepine receptor agonists and psychological and behavioral therapies. Sleep Med Rev 2009;13:205–14.
[3] National Institutes of Health (NIH) state-of-the-science conference statement on manifestations and management of chronic insomnia in adults, June 13–15, 2005. Sleep 2005;28:1049–1057.
[4] Kyle SD, Espie CA, Morgan K. '. . .Not just a minor thing, it is something major, which stops you from functioning daily': quality of life and daytime functioning in insomnia. Behav Sleep Med 2010;8:123–40.
[5] Spielman A, Caruso L, Glovinsky P. A behavioral perspective on insomnia treatment. Psychiatr Clin North Am 1987;10:541–53.
[6] Spielman AJ, Saskin P, Thorpy MJ. Treatment of chronic insomnia by restriction of time in bed. Sleep 1987;10:45–56.
[7] Pigeon WR, Perlis ML. Sleep homeostasis in primary insomnia. Sleep Med Rev 2006;10:247–54.
[8] Morin CM. Insomnia: psychological assessment and management. New York: Guilford; 1993.
[9] Morin CM, Espie CA. Insomnia: a clinical guide to assessment and treatment. Kluwer Academic/Plenum Publishers; 2003.
[10] Edinger JD, Means MK. Cognitive-behavioral therapy for primary insomnia. Clin Psychol Rev 2005;25:539–58.
[11] Nutt DJ, Sharpe M. Uncritical positive regard? issues in the efficacy and safety of psychotherapy. J Psychopharmacol 2008;22:3–6.
[12] Berk M, Parker G. The elephant on the couch: side-effects of Psychotherapy. Aust N Z J Psychiatry 2009;43:787–94.
[13] Hoelscher TJ, Edinger JD. Treatment of sleep-maintenance insomnia in older adults: sleep period reduction, sleep education, and modified stimulus control. Psychol Aging 1998;3:258–63.
[14] Haynes PL. The role of behavioural sleep medicine in the assessment and treatment of sleep disordered breathing. Clin Psychol Rev 2005;25:673–705.
[15] Riedel BW, Lichstein KL. Strategies for evaluating adherence to sleep restriction treatment for insomnia. Behav Res Ther 2001;39:201–12.
[16] Vincent N, Lewycky S, Finnegan H. Barriers to engagement in sleep restriction and stimulus control in chronic insomnia. J Consult Clin Psychol 2008;76:820–8.
[17] Vincent NK, Hameed H. Relation between adherence and outcome in the group treatment of insomnia. Behav Sleep Med 2003;1:125–39.
[18] Bouchard S, Bastien C, Morin CM. Self-efficacy and adherence to cognitive-behavioral treatment of insomnia. Behav Sleep Med 2003;1:187–99.
[19] Perlis ML, Smith MT, Orff H, Enright T, Nowakowski S, Jungquist C, et al. The effects of modafinil and cognitive behaviour therapy on sleep continuity in patients with primary insomnia. Sleep 2004;27:715–25.
[20] Harvey L, Inglis SJ, Espie CA. Insomniacs' reported use of CBT components and relationship to long-term clinical outcome. Behav Res Ther 2002;40:75–83.
[21] Vincent N, Lionberg C. Treatment preference and patient satisfaction in chronic insomnia. Sleep 2001;15:411–7.
[22] Kyle SD, Morgan K, Espie CA. Insomnia and health-related quality of life. Sleep Med Rev 2010;14:69–82.
[23] Omvik S, Sivertsen B, Pallesen S, Bjorvatn B, Havik OE, Nordhus IH. Daytime functioning in older patients suffering from chronic insomnia: treatment outcome in a randomized controlled trial comparing CBT with zopiclone. Behav Res Ther 2008;46:623–41.
[24] Horne J. Primary insomnia: a disorder of sleep, or primarily one of wakefulness? Sleep Med Rev 2010;14:3–7.
[25] Bonnet MH, Arand DL. Hyperarousal and insomnia. Sleep Med Rev 1997;1:97–108.

[26] Semler CN, Harvey AG. Daytime functioning in primary insomnia: does attentional focus contribute to real or perceived impairment? Behav Sleep Med 2006;4:85–103.
[27] Espie CA, Broomfield NM, MacMahon KM, Macphee LM, Taylor LM. The attention–intention–effort pathway in the development of psychophysiological insomnia: a theoretical review. Sleep Med Rev 2006;10:215–45.
[28] The PLoS Medicine Editors. Qualitative research: understanding patients' needs and experiences. PLoS Med 2007;4:e258.
[29] Tolmie EP, Lindsay GM, Kerr SM, Brown MR, Ford I, Gaw A. Patients' perspectives on statin therapy for treatment of hypercholesterolaemia: a qualitative study. Eur J Cardiovasc Nurs 2003;2:141–9.
[30] Osborn M, Smith JA. The personal experience of chronic benign lower back pain: an interpretative phenomenological analysis. Br J Health Psychol 1998;3:65–83.
[31] Price J, Cole V, Goodwin GM. Emotional side-effects of selective serotonin reuptake inhibitors: qualitative study. Br J Psychiatry 2009;195:211–7.
[32] Burgess C, Hunter MS, Ramirez AJ. A qualitative study of delay among women reporting symptoms of breast cancer. Br J Gen Pract 2001;51:967–71.
[33] Brostrom A, Nilsen P, Johansson P, Ulander M, Stromber A, Svanborg E, et al. Putative facilitators and barriers for adherence to CPAP treatment in patients with obstructive sleep apnea syndrome: a qualitative content analysis. Sleep Med 2010;11:126–30.
[34] Friedman L, Benson K, Noda A, et al. An actigraphic comparison of sleep restriction and sleep hygiene treatments for insomnia in older adults. J Geriatr Psychiatry Neurol 2000;13:17–27.
[35] Lichstein KL, Riedel BW, Wilson NM, Lester KW, Aguillard RN. Relaxation and sleep compression for late-life insomnia: a placebo controlled trial. J Consult Clin Psychol 2001;69:227–39.
[36] Edinger JD, Bonnet MH, Bootzin RR, et al. Derivation of research diagnostic criteria for insomnia: report of an American Academy of Sleep Medicine workgroup. Sleep 2004;27:1567–96.
[37] Espie CA, MacMahon KMA, Kelly H, et al. Randomized Clinical effectiveness trial of nurse-administered small-group cognitive behaviour therapy for persistent insomnia in General practice. Sleep 2007;30:574–84.
[38] Buysse DJ, Reynolds III CF, Monk TH, Berman SR, Kupfer DJ. The Pittsburgh Sleep Quality Index: a new instrument for psychiatric practice and research. Psychiatry Res 1989;28:193–213.
[39] Broomfield NM, Espie CA. Towards a valid, reliable measure of sleep effort. J Sleep Res 2005;14:401–7.
[40] David B, Morgan K. Occupational and daytime functioning in primary insomnia: a prospective study. J Sleep Res 2006;15(Abstract Suppl.):157.
[41] Kyle SD, Espie CA, Morgan K, Fleming L. The Glasgow sleep impact index: a patient generated measure for capturing sleep-related quality of life. Sleep 2009;32(Abstract Suppl.):0850.
[42] Kyle SD, Morgan K, Espie CA. The daytime functioning and sleep attribution scale (DFSAS): a new insomnia-specific measure to probe daytime impairment and poor sleep attributions. Sleep 2010(Suppl.):A182.
[43] Ware JE, Sherbourne CD. The MOS 36-Item Short-Form Health Survey (SF-36). 1. Conceptual-framework and item selection. Med Care 1992;30:473–83.
[44] Uher R, Farmer A, Henigsberg N, et al. Adverse reactions to antidepressants. Br J Psychiatry 2009;195:202–10.
[45] Smith JA, Osborn M. Interpretative phenomenological analysis. In: Smith JA, editor. Qualitative psychology: a practical guide to methods. London: Sage; 2003. p. 51–80.
[46] Field AP. Discovering statistics using SPSS. 3rd ed. London: Sage; 2009.
[47] Cohen J. Statistical power analysis for the behavioral sciences. 2nd ed. Hillsdale, NJ: Erlbaum; 1988.
[48] Braun V, Clarke V. Using thematic analysis in psychology. Qual Res Psychol 2006;3:77–101.
[49] Yang M, Morin CM, Shaefer K, Wallenstein GV. Interpreting score difference in the Insomnia Severity Index: using health-related outcomes to define the minimally important difference. Curr Med Res Opin 2009;25:2487–94.
[50] Krystal AD, Edinger JD. Sleep EEG predictors and correlates of the response to cognitive behavioural therapy for insomnia. Sleep 2010;33:669–77.
[51] Krystal AD. A compendium of placebo-controlled trials of the risks/benefits of pharmacological treatment for insomnia: the empirical basis for US clinical practice. Sleep Med Rev 2009;13:265–74.
[52] Gustavsen I, Bramness JG, Skurtveit S, Engeland A, Neutel I, Morland J. Road traffic accident risk related to prescriptions of the hypnotics zopiclone, zolpidem, flunitrazepam and nitrazepam. Sleep Med 2008;8:818–22.
[53] Rasetti R, Mattay VS, Stankevich B, et al. Modulatory effects of modafinil on neural circuits regulating emotion and cognition. Neuropsychopharmacol 2010;35:2101–9.
[54] Wicklow A, Espie CA. Intrusive thoughts and their relationship to actigraphic measurement of sleep: towards a cognitive model of insomnia. Behav Res Ther 2000;38:679–93.
[55] Escalante CP, Manzullo EF. Cancer-related fatigue: the approach and treatment. J Gen Intern Med 2009;24:S412–6.
[56] Department of Health. The expert patient: a new approach to chronic disease management for the 21st century. London: The Department of Health; 2001.
[57] Wiese JH, Boethel C, Phillips B, Wilson JF, Peters J, Viggiano T. CPAP compliance: video education may help! Sleep Med 2005;6:171–4.
[58] Aloia MS, Arnedt JT, Riggs RL, Hecht J, Borreli B. Clinical management of poor adherence to CPAP: motivational enhancement. Behav Sleep Med 2004;2:205–22.
[59] Edinger JD, Sampson WS. A primary care "friendly" cognitive behavioral therapy. Sleep 2003;26:177–82.
[60] Germain A, Moul DE, Franzen PL, Miewald JM, Reynolds CF, Monk TH, et al. Effects of a brief behavioural treatment for late-life insomnia: preliminary findings. J Clin Sleep Med 2007;2:407–8.
[61] Gradisar M, Lack L, Richards H, Harris J, Gallasch J, Boundy M, et al. The Flinders Fatigue Scale: preliminary psychometric properties and clinical sensitivity of a new scale for measuring daytime fatigue associated with insomnia. J Clin Sleep Med 2007;3:722–8.

http://dx.doi.org/10.5665/sleep.1882

The Longitudinal Course of Insomnia Symptoms: Inequalities by Sex and Occupational Class Among Two Different Age Cohorts Followed for 20 Years in the West of Scotland

Michael J. Green, MA[1]; Colin A. Espie, PhD[2]; Kate Hunt, PhD[1]; Michaela Benzeval, PhD[1]

[1]Medical Research Council/Chief Scientist Office: Social and Public Health Sciences Unit, Glasgow, United Kingdom; [2]University of Glasgow Sleep Centre, College of Medicine, Veterinary Medicine and Life Sciences, University of Glasgow, Glasgow, United Kingdom

Study Objectives: The natural history of insomnia symptomatology is poorly understood. Cross-sectional associations have been demonstrated among socioeconomic disadvantage, female sex, and poor sleep but it is unclear how these social factors predict patterns of insomnia symptoms over time. The aim of this article is to describe longitudinal patterns of insomnia symptoms as people age and investigate how they vary by sex and occupational class.

Design: A prospective cohort study with 20 yr of follow-up from 1987 to 1988.

Setting: West of Scotland.

Participants: One cohort approximately 36 yr of age at baseline aging to 57 yr (n = 1,444), and another aging from approximately 56 to 76 yr (n = 1,551).

Interventions: N/A.

Measurements and Results: At approximately 5-yr intervals, respondents self-reported trouble initiating and maintaining sleep. Latent class analysis identified 4 main sleep patterns: a healthy pattern with little sleeping trouble across the 20 yr; an episodic pattern, characterized by trouble maintaining sleep; a chronic pattern with trouble maintaining and initiating sleep throughout the study; and a pattern where symptoms developed during the 20-yr follow-up. Chronic patterns were more likely in the older cohort than the younger one, for women than men in the older cohort, and for those from a manual rather than a nonmanual occupational class in both cohorts. In the middle-aged cohort a developing pattern was more likely for women than men.

Conclusions*:* Chronic symptoms, characterized by both trouble maintaining and initiating sleep, are patterned by social factors.

Keywords: Insomnia, sleeplessness, sex, socioeconomic status, life course, prospective cohort

Citation: Green MJ; Espie CA; Hunt K; Benzeval M. The longitudinal course of insomnia symptoms: inequalities by sex and occupational class among two different age cohorts followed for 20 years in the west of Scotland. *SLEEP* 2012;35(6):815-823.

INTRODUCTION

Problems getting to sleep and/or remaining asleep are very common in adults,[1] and have long been regarded as the core symptoms of insomnia.[2-3] In relation to mental health, there is firm evidence that insomnia is a risk factor for the evolution of, and relapse into, depression.[4-6] Likewise, insomnia has been associated with a higher prevalence of physical health conditions such as hypertension and type 2 diabetes,[7-8] with all-cause mortality,[9] and with other adverse outcomes such as a poor quality of life and problems with work performance and personal relationships.[10,11] It seems important, therefore, from a public health perspective, to understand who sleeps well, who has trouble sleeping, and whether particular groups in the community are more or less prone to the development or resolution of insomnia symptoms during adulthood.

So far, however, the natural history of insomnia remains relatively poorly understood. The few longitudinal studies that do exist suggest that insomnia symptoms tend to become chronic and persistent,[12-14] with evidence also of remitting and recurring patterns.[15] A 3-yr follow-up of people with insomnia symptoms showed more persistent symptoms among those with more severe, clinically significant symptoms,[15] and a 4-mo study showed greater stability of symptoms among patients who reported trouble both initiating and maintaining sleep,[16] but few studies have examined symptom patterns over a period of many years.

In terms of who experiences insomnia symptoms, the prevalence tends to increase with age.[1] A meta-analysis of cross-sectional data by sex concluded that women were more likely to experience symptoms than men and that this gender difference was stronger with increasing age.[17] Other cross-sectional research has also highlighted socioeconomic status (SES) as a correlate of insomnia symptoms.[18-19] Sex effects are attenuated but not fully explained by adjustment for socioeconomic factors, as socioeconomic disadvantage is associated with both poor sleep and being female.[20-21] Longitudinally, a pattern of persistent symptoms over 3 yr has been shown to be more likely for women than men.[15] Another study showed no significantly greater incidence or persistence in insomnia symptoms over 12 mo for those in manual rather than nonmanual occupations,[13] and a separate 12-mo incidence study showed a higher likelihood of developing symptoms for women than men, and either weak or no relationships for socioeconomic factors such as income and education.[22] The lack of an effect for socioeconomic factors in these longitudinal studies contrasts with findings from cross-sectional research where people in disadvantaged socioeconomic circumstances were more likely to experience insomnia symptoms than those who were more affluent. This may be because 12 mo of follow-up is too short a time period for socioeconomic differences to emerge. It is important to un-

Submitted for publication March, 2011
Submitted in final revised form February, 2012
Accepted for publication March, 2012
Address correspondence to: Michael J Green, MA, MRC/CSO Social and Public Health Sciences Unit, 4 Lilybank Gardens, Glasgow, G12 8RZ, United Kingdom; Tel: +441413573949; Fax: +441413372389; E-mail: michael-g@sphsu.mrc.ac.uk

Table 1—Comparison of sample characteristics at baseline and final interviews

	Baseline interview (1987/1988)	Final interview (2007/2008)
1950s Cohort		
n	1,444	999
Female (%)	788 (54.6)	542 (54.3)
Manual class at baseline (%)[a]	494 (34.6)	304 (30.7)
No symptoms at Baseline (%)[a]	789 (57.8)	577 (59.2)
1930s Cohort		
n	1,551	663
Female (%)	849 (54.7)	384 (57.9)
Manual class at baseline (%)[a]	710 (45.8)	227 (34.2)
No symptoms at baseline (%)[a]	729 (49.9)	353 (54.0)

[a]These values represent percentages of valid responses. Item missingness ranged from 0-1.2% for occupational class and from 1.4-5.8% for insomnia symptoms.

derstand how these various risk factors for insomnia are associated with long-term patterns of insomnia symptoms.

The aim of this article was, first, to identify the main longitudinal patterns of insomnia symptoms across the life course, and second, to investigate whether experience of these patterns varied by sex and occupational class. Two cohorts, 20 yr apart in age, were studied over a 20-yr period, allowing comparison of those in their mid-30s to mid-50s with those in their mid-50s to mid-70s. In light of previous research, it was hypothesized that women, older respondents, and those in disadvantaged occupational classes would be especially likely to experience more persistent patterns of insomnia symptoms.

METHODS

Sample and Measures

The Twenty-07 Study[23] has followed people in 3 age cohorts – born around 1932, 1952, and 1972 – for 20 yrs. It has 2 subsamples: the regional sample, a 2-stage stratified random sample of people living in the Central Clydeside Conurbation, West of Scotland, and the localities sample of people from 2 areas of the city of Glasgow. Baseline interviews were conducted in 1987/1988 and there have been 4 follow-ups (1990/1992; 1995/1997; 2000/2004; 2007/2008), with ethics approval gained for each wave from the National Health Service and/or Glasgow University Ethics Committees. At each wave of data collection, nurses trained in survey methods conducted face-to-face individual interviews supplemented by a suite of physical measurements. Because questions on sleep were not asked at all interviews in the 1970s cohort, only the 1950s and 1930s cohorts are examined here. The 1950s cohort aged from approximately 36 to 57 yr during the study period and the 1930s cohort from 56 to 76 yr. Baseline sample sizes for these cohorts were 1,444 and 1,551, respectively (original response rates were 88.9% and 87.1%)[23] and the achieved sample has been shown to be representative of the general population of the sampled area.[24]

Insomnia symptom questions were asked at all 5 interviews over 20 yr of the study for both sleep latency (trouble getting to sleep) and sleep maintenance (waking early or during the night). Unfortunately, the specific questions asked changed during the study; nevertheless, it is possible to create broadly comparable insomnia variables over time for both dimensions (latency and maintenance). In the baseline and the first 2 follow-up interviews, respondents were asked 2 questions: "How often do you have trouble getting to sleep?" and "How often are you bothered by waking earlier than you would like to, or by waking up in the middle of the night?" Both questions had 6 response categories: *never, less than monthly, at least once a month, at least once a week, most days,* and *every day.* In waves 4 and 5, as part of the Pittsburgh Sleep Quality Index,[25] respondents were asked, "During the past month how often have you had trouble sleeping because you cannot get to sleep within 30 minutes?" and "During the past month how often have you had trouble sleeping because you wake up in the middle of the night or early morning?" Both questions had 4 categories: *not during the past month, less than once a week, once or twice a week* ,and *3 or more times a week*. To define the frequency of insomnia symptoms consistently across all 5 interviews for each symptom type – latency and maintenance – the questions have been recoded for each interview into 2 binary variables, respectively representing latency and maintenance problems, with 0 representing either no problems or a less than weekly frequency, and 1 representing sleep problems occurring at least weekly. Note, however, that the respondents were asked specifically about their sleep within the past month in the 4th and 5th interviews as opposed to being asked about their sleep generally in the earlier interviews. In addition, at each interview respondents were also shown cards with lists of common medical symptoms, including "difficulty sleeping", and asked whether they had experienced this symptom in the past 4 wk or 1 mo (yes or no). Although this question does not contain comparable information on how frequently symptoms occurred and makes no distinction between sleep latency and sleep maintenance, the question wording was consistent throughout the study, and so we took the opportunity to validate findings from the questions on sleep latency and sleep maintenance in separate sensitivity analyses using this variable, as discussed in the next sections.

Sex was coded 0 for males and 1 for females. Baseline household occupational class was used as a measure of SES. This measure is common in British health research, and represents both social standing and material resources.[26-27] Here it is coded according to the British Registrar General's 1980 classification[28] using the higher status occupation in couple households. If neither the respondent nor their partner had a current occupation then the higher class from their most recent previous occupations was used. This variable was then dichotomised with 0 for nonmanual (I through to III nonmanual) and 1 for manual (III manual through to V) classes. Twenty respondents were excluded from the analysis because of missing data on baseline occupational class.

In the most recent follow-up (2007/2008), 999 of the 1950s cohort and 663 of the 1930s cohort took part (73.2% and 65.5% of the living baseline sample). Of the 445 respondents from the 1950s cohort who were lost to follow-up by this point, 88 had died (19.8%), whereas 562 of the 888 respondents who were lost to follow-up in the 1930s cohort had died (63.3%),[29] so mortality was the most common reason for drop-out in the oldest cohort. Table 1 compares sample characteristics at the

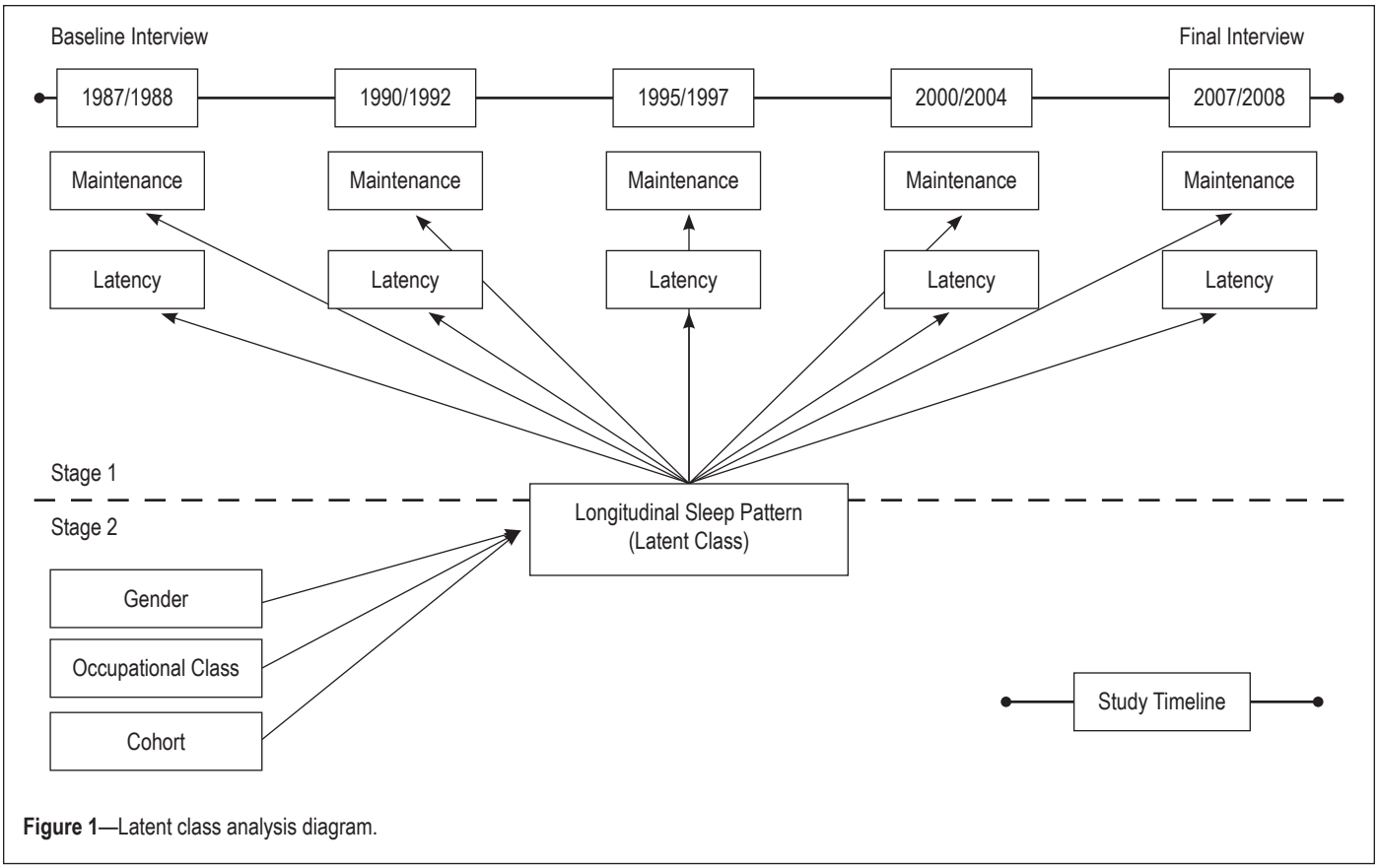


Figure 1—Latent class analysis diagram.

baseline and final interviews. Loss to follow-up was especially likely among those who reported sleep problems at baseline in both cohorts and among those who were in a manual class at baseline, especially in the older cohort. This uneven loss to follow-up is addressed using maximum likelihood estimation as discussed in the statistical analysis section.

Statistical Analysis

This article uses a repeated measures latent class analysis,[30] which identifies clusters of categorical responses (termed latent classes) across repeated measurements. It estimates the overall probability of membership in each latent class (class membership probabilities), the probability of reporting each sleep problem at each time point given latent class membership (response probabilities), and can also be used to explore associations between covariates and latent class membership. The latent class analysis is depicted diagrammatically in Figure 1. Modeling proceeded in 2 main stages relating to the 2 main aims of the study. In stage 1 the aim was to identify the most common longitudinal patterns of insomnia symptoms present within the data. Although the data contained a wide variety of different combinations of responses across the study period, the aim of the latent class analysis was to identify a smaller number of dominant groupings, representing different longitudinal patterns of insomnia symptoms. The number of latent classes was determined by estimating a series of latent class models, each with an incrementally greater number of latent classes, and then comparing these models on the basis of various model fit statistics (for details see supplementary information, part I).

The second stage of modeling investigated whether the longitudinal patterning of insomnia symptoms varied by cohort, sex, and occupational class. This was done using the optimal latent class model identified in the first stage. There are 2 main ways in which longitudinal response patterns may vary in line with covariates. First, an entirely different set of latent classes might be experienced in different groups, or second, different groups might experience similar patterns at different frequencies. It is meaningless to investigate the second type of variation unless the first type is either absent or negligible, as like cannot be compared with like.[30] Detailed analyses (see supplementary information, part II) investigating the first type of variation suggested that differences in the response probabilities for each latent class by sex, occupational class, or cohort were minor. We therefore tested for differences in the odds of class membership between sexes, occupational classes, and cohorts by adding these variables to the latent class model as predictors in a multinomial logistic regression of latent class membership. Because latent class membership was the outcome here, the odds ratios associated with each covariate represent the odds of experiencing a particular longitudinal pattern of insomnia symptoms over 20 yr, rather than the odds of symptoms at any 1 time point. Because the latent classes were based on longitudinal data that included symptoms from the baseline interviews, measured concurrently with the covariates, there is a partially cross-sectional element to the estimated associations and reverse causation is possible. Interactions between covariates in predicting latent class membership were also tested and retained if significant at the $P < 0.05$ level. Age was incorporated into the model in 2 ways: first, differences in the experience of the two cohorts, 20 years apart in age, were examined by including cohort as a proxy for age (because respondents within the same cohort were approximately the same age); second, ag-

Table 2—Co-occurrence of sleep problems over study

	Interview 1	Interview 2	Interview 3	Interview 4	Interview 5
1950s Cohort					
Mean age (standard deviation)	36.2 (0.8)	40.5 (0.9)	45.2 (1.2)	50.2 (1.3)	57.1 (0.8)
No symptoms[a]: n (%)	789 (57.8)	823 (67.4)	461 (61.4)	464 (49.8)	415 (42.6)
Latency only[a]: n (%)	109 (8.0)	61 (5.0)	39 (5.2)	39 (4.2)	48 (4.9)
Maintenance only[a]: n (%)	297 (21.7)	182 (14.9)	118 (15.7)	194 (20.8)	248 (25.4)
Both symptoms[a]: n (%)	171 (12.5)	155 (12.7)	133 (17.7)	234 (25.1)	264 (27.1)
Missing sleep data: n (%)	78 (5.4)	4 (0.3)	275 (26.8)[b]	49 (5.0)	24 (2.4)
Total n (% of baseline sample)	1,444 (100.0)	1,225 (84.8)	1,026 (71.1)	980 (67.9)	999 (69.2)
1930s Cohort					
Mean Age (standard deviation)	56.2 (0.6)	59.6 (0.8)	64.4 (1.2)	69.1 (1.0)	76.2 (0.6)
No symptoms[a]: n (%)	729 (49.9)	626 (49.5)	350 (48.5)	321 (41.4)	230 (36.7)
Latency only[a]: n (%)	133 (9.1)	97 (7.7)	52 (7.2)	40 (5.2)	41 (6.5)
Maintenance only[a]: n (%)	296 (20.3)	267 (21.1)	156 (21.6)	139 (17.9)	142 (22.6)
Both symptoms[a]: n (%)	303 (20.7)	275 (21.7)	163 (22.6)	276 (35.6)	214 (34.1)
Missing sleep data: n (%)	90 (5.8)	1 (0.1)	309 (30.0)[b]	62 (7.4)	36 (5.4)
Total n (% of baseline sample)	1,551 (100.0)	1,266 (81.6)	1,030 (66.4)	838 (54.0)	663 (42.7)

[a]To facilitate comparison across the study, percentages here are presented as proportions of those with valid sleep data. [b]Missingness is higher here because at this stage the 2 locality subsamples only received a shortened postal questionnaire that did not include the sleep questions (n = 272 for the 1950s cohort; n = 307 for the 1930s cohort).

ing effects within a cohort, i.e., from 1 interview to the next, are represented by the probabilities of symptoms at each interview as predicted by latent class membership.

All analyses were performed in Mplus version 6.1.[31] Maximum likelihood estimation was used, using all available sleep data for each respondent (n = 2,867; 1,383 from the 1950s cohort and 1,484 from the 1930s cohort), and hence these analyses are robust to sample attrition under the assumption that data are missing at random.[32] This assumes that the missingness of a variable can be predicted by the other variables in the model, i.e., if nonparticipation at later waves was related to later insomnia symptoms, but those symptoms could be predicted by cohort, sex, occupational class, and the sleep data from other waves (e.g., baseline symptoms), then the model estimates would be unbiased. For example, the model would be robust to the slightly higher rates of drop-out among those with baseline symptoms unless those who later recovered were more or less likely to be retained than those who did not recover (and then only if these differences were not explained by cohort, sex, and/or occupational class). In the third wave of interviews the 2 locality subsamples only received a postal questionnaire that did not include the questions on sleep; however, being in the locality subsamples was not related to having sleep problems at any of the other waves (chi-square test, $P > 0.05$), so there is no reason to believe that this missingness-by-design would bias the results.

As a sensitivity analysis, the latent class modeling was repeated for each type of insomnia symptom separately, to ensure no distortions had resulted from modeling the 2 symptom dimensions together. For validation purposes, given the change in wording for the 2 insomnia symptom questions between the 3rd and 4th interviews, the latent class analysis was also repeated separately for the general question on sleep difficulty. Because the general question on sleep difficulty was worded consistently over the course of the study, observation of similar latent classes for this question would suggest that the observed patterns were genuine and not an artefact of the question change (for details see supplementary information, part III).

RESULTS

Table 2 shows the prevalence of latency symptoms alone, maintenance symptoms alone, co-occurring latency and maintenance symptoms, and the proportion reporting no symptoms, at each of the 5 interviews by cohort. The co-occurrence of latency and maintenance symptoms was common, varying from 12.5% to 27.1% of respondents in the 1950s cohort as they aged from 36 to 57 yr, and 20.7% to 35.6% in the older cohort as they aged from 56 to 76 yr. Sleep maintenance problems were also frequently reported, but it was relatively rare for respondents to report latency symptoms without accompanying maintenance problems. There was a marked reduction in the prevalence of those reporting no sleep problems across the study as both cohorts aged, and a marked increase in the prevalence of sleep problems between waves 3 and 4 when the question wording changed. Comparing the cross-sectional prevalence of symptoms at the baseline interviews in the 1930s cohort with that from the final interview in the 1950s cohort, where respondents were approximately the same age, suggests a lower symptom prevalence in the older cohort (42.6% with no symptoms compared to 49.9%).

The best latent class model in terms of balancing model fit and parsimony identified 4 latent classes (see supplementary information, part I). The probabilities of problems initiating and maintaining sleep at each wave of the study for the 4 latent classes are shown in Figure 2. Error bars show 95% confidence intervals for estimates. Respondents in class 1 had a low probability of latency or maintenance problems at all of the study waves and so were labeled the *Healthy* class. The overall probability of membership in this class was 0.37. Those in class 2 had a low probability of latency problems at all time points but a relatively high probability of maintenance problems. These were labeled the *Episodic Maintenance* class (overall membership

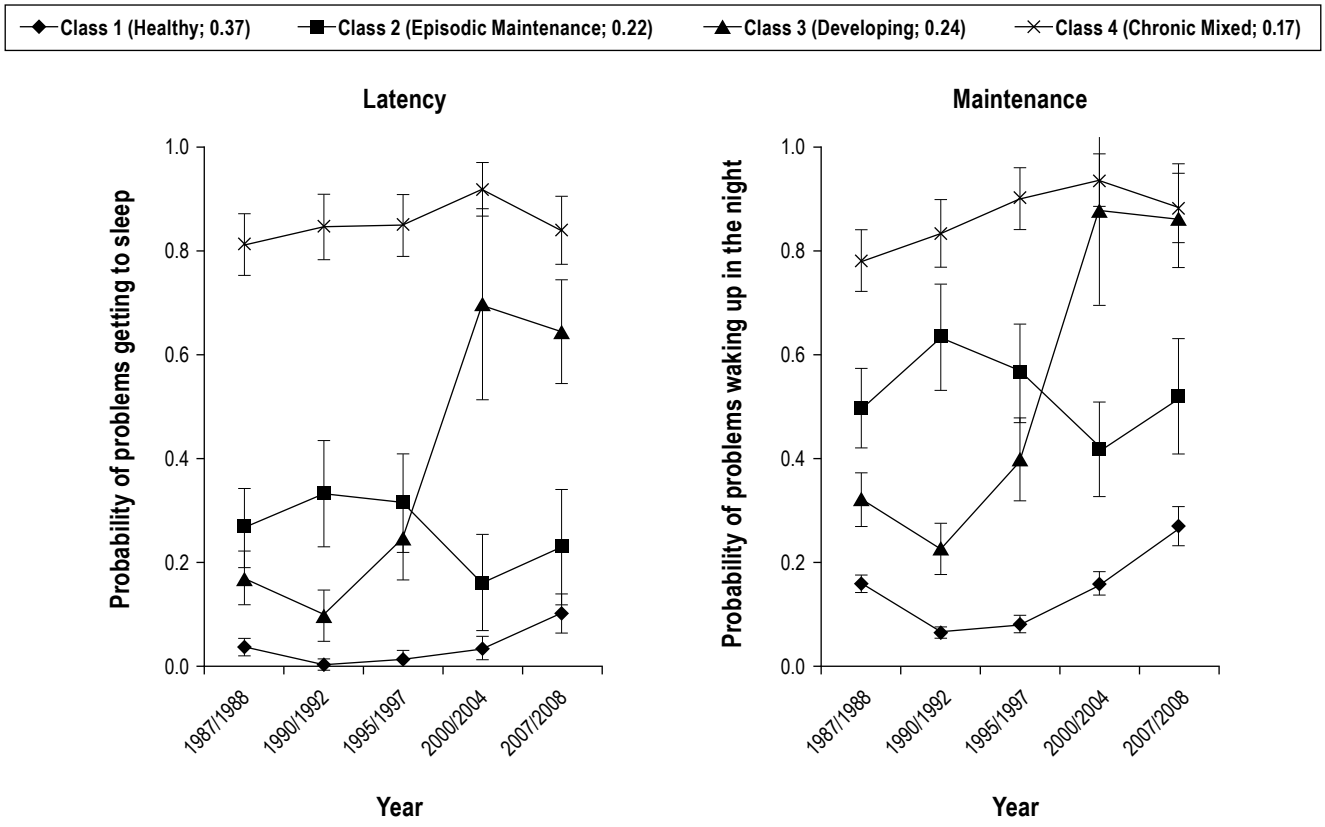


Figure 2—Probability of latency and maintenance problems at each study wave given latent class membership.

probability: 0.22). The members of class 3 had a low probability of reporting either symptom in earlier study waves, but then a high probability of both symptoms, especially maintenance problems, at waves 4 and 5. These were labeled the *Developing* class as they appeared to be developing insomnia symptoms (overall membership probability: 0.24). Finally, those in class 4 had a high probability of trouble both initiating and maintaining sleep at all time points, and were labeled the *Chronic Mixed* class (overall membership probability: 0.17). Those in the *Episodic Maintenance* class tended toward a lower probability of trouble maintaining sleep than those in the *Chronic Mixed* class; probabilities at each wave were all approximately 0.4-0.6, indicating that maintenance symptoms would be reported in approximately half of the study waves for each individual in this class. This suggests less stable or more episodic problems than in the *Chronic Mixed* class where the response probabilities for maintenance symptoms are approximately 0.8-0.9 and symptoms would be reported at most interviews. Those in the *Episodic Maintenance* class also tended to experience a higher probability of latency problems than those in the *Healthy* class, indicating that although maintenance is the primary or dominant problem in this class, latency problems also occurred occasionally and with greater frequency than where maintenance problems were not present. The absence of other potential patterns in the latent class solution, e.g., a full recovery pattern, or a pattern where latency symptoms were dominant, indicates that such patterns were relatively rare.[30]

Table 3 displays the odds ratios (OR) and 95% confidence intervals from a multinomial logistic regression of latent class

Table 3—Multinomial logistic regression of latent class membership on sex, occupational class, and cohort

	Odds ratios	95% Confidence intervals
Episodic maintenance (ref: Healthy)		
Female in 1950s cohort (ref: male)[a]	0.78	(0.43-1.41)
Female in 1930s cohort (ref: male)[a]	2.22	(1.36-3.63)
Manual (ref: Non-manual)	1.28	(0.89-1.85)
1930s cohort for males (ref: 1950s)[a]	1.45	(0.91-2.32)
1930s cohort for females (ref: 1950s)[a]	4.14	(2.24-7.66)
Developing (ref: Healthy)		
Female in 1950s cohort (ref: male)[a]	4.30	(2.65-6.98)
Female in 1930s cohort (ref: male)[a]	1.59	(1.00-2.51)
Manual (ref: Non-manual)	1.61	(1.14-2.28)
1930s cohort for males (ref: 1950s)[a]	3.55	(2.11-5.97)
1930s cohort for females (ref: 1950s)[a]	1.31	(0.85-2.02)
Chronic Mixed (ref: Healthy)		
Female in 1950s cohort (ref: male)[a]	1.20	(0.78-1.85)
Female in 1930s cohort (ref: male)[a]	3.10	(2.15-4.49)
Manual (ref: Non-manual)	3.22	(2.46-4.21)
1930s cohort for males (ref: 1950s)[a]	1.61	(1.08-2.38)
1930s cohort for females (ref: 1950s)[a]	4.16	(2.79-6.19)

[a]This odds ratio is calculated by combining the relevant main and interaction effects for sex and cohort.

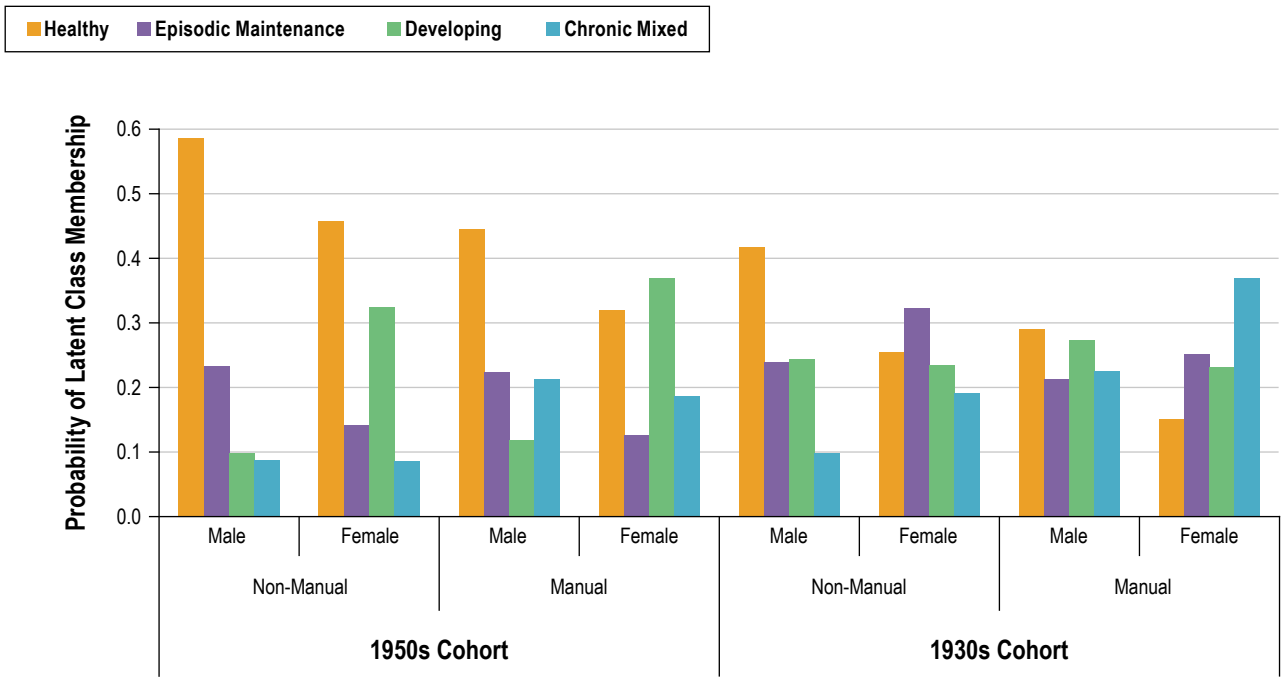


Figure 3—Latent class membership probabilities for both cohorts by sex and occupational class.

membership on sex, occupational class, and cohort (mutually adjusted). There was a significant interaction between cohort and sex ($P < 0.05$), so for ease of interpretation ORs combining the main and interaction effects have been calculated and are presented by cohort for sex, and by sex for cohort. No other interactions between sex, occupational class, and cohort were found to be significant. Women were more likely than men to be in the *Developing* class as opposed to the *Healthy* class, but the effect was larger in the 1950s than in the 1930s cohort (1950s OR 4.30, $P < 0.01$; 1930s OR 1.59, $P < 0.05$). In the 1930s cohort, but not in the 1950s cohort, women were more likely than men to be in the *Chronic Mixed* relative to the *Healthy* sleep class (OR 3.10, $P < 0.01$). Respondents from manual occupational classes in both cohorts were more likely than their nonmanual counterparts to be in the *Developing* or *Chronic Mixed* latent classes relative to the *Healthy* class, though the effect size for being in the *Chronic Mixed* class (OR 3.22, $P < 0.01$) was approximately twice that for being in the *Developing* class (OR 1.61, $P < 0.01$). Men in the 1930s cohort were more likely than men in the younger 1950s cohort to be in the *Developing* (OR 3.55, $P < 0.01$) or *Chronic Mixed* (OR 1.61, $P < 0.05$) classes as opposed to the *Healthy* sleep class. Women in the 1930s cohort were not significantly more likely than those in the 1950s cohort to be in the *Developing* rather than the *Healthy* sleep class (OR 1.31, $P = 0.22$), but there was an even stronger tendency than there was amongst men for those in the 1930s rather than the 1950s cohort to be in the *Chronic Mixed* class (OR 4.16, $P < 0.01$) rather than the *Healthy* sleep class.

Figure 3 is provided to aid interpretation and shows estimated probabilities of latent class membership for all the different combinations of these variables. Membership in the *Healthy* class was the most probable outcome for most respondents in the 1950s cohort, with the exception of women in a manual class, who were more likely to be in the *Developing* than the *Healthy* class. In the 1930s cohort, however, women were more likely to be in 1 of the symptomatic sleep classes than in the *Healthy* class, irrespective of occupational class. Women in the 1930s cohort who were in a manual class were particularly disadvantaged in that membership in the *Chronic Mixed* class was the most likely outcome for them and membership in the *Healthy* class was the least likely. Only men from nonmanual classes in the 1950s cohort had a greater than 50% chance of being in the *Healthy* sleep class.

Repeating the modeling for sleep latency and sleep maintenance problems separately revealed a 3-class solution as the best-fitting model for both types of insomnia symptom (see supplementary information, part III). In each case the 3 classes observed could be viewed as corresponding to the *Healthy*, *Developing*, and *Chronic Mixed* classes from the joint modeling. The *Episodic Maintenance* class appeared to have been subsumed into the class experiencing chronic difficulties when modeling maintenance only, and into the *Developing* class when modeling latency only. These results are therefore consistent with and support those from the joint modeling.

Given the change in question wording and concurrent increase in the prevalence of insomnia symptoms at interviews 4 and 5, it is possible that the *Developing* class represents sensitivity to question wording rather than symptom development. This issue was investigated by performing a further latent class analysis using the general question on sleep difficulty that had been worded consistently across the study, asking about symptoms within the past month at each interview. Because this question does not make the distinction between latency and maintenance problems, when modeling using these data it would be expected *a priori* not to find a class similar to the *Episodic Maintenance* class, but the other 3 classes, including

the *Developing* class, should be fairly replicable. Results from this analysis (see supplementary information, part III) suggested a 3-class model similar to those observed for sleep latency and sleep maintenance separately. As well as classes with healthy and chronic patterns there was evidence of a class with symptoms developing toward the end of the study, albeit with a smoother slope, suggesting that the *Developing* class is more reflective of symptoms developing with increasing age within each cohort than of measurement differences.

Latent class membership probabilities for the general question on difficulty sleeping were also similar to those for the questions on sleep maintenance, with a slightly lower prevalence in both of the symptomatic categories. Finally, covariate effects were all in the same direction as in the main results, though there were some differences in whether or not particular effects were significant (some small differences would be expected with the reassignment of those in the *Episodic Maintenance* class into other categories). Overall, modeling of this alternative question revealed symptom patterning similar to that observed for the main measures used, validating the findings and suggesting that instrument effects associated with the change in question wording were minor.

DISCUSSION

In this article we have presented longitudinal data on patterns of insomnia symptoms over a 20-yr period in 2 cohorts, aging from their mid-30s to mid-50s and from their mid-50s to mid-70s, respectively. Four distinct patterns of insomnia symptoms were apparent over the adult life course: a *Healthy* class with only occasional experience of sleeping trouble; an *Episodic Maintenance* class who had episodes dominated by trouble maintaining rather than initiating sleep; a *Developing* class who appeared to develop insomnia symptoms as they aged; and a *Chronic Mixed* class who had persistent trouble with both initiating and maintaining sleep. The only other study of insomnia of comparable length that we are aware of was in a cohort of young adults, age 19-20 yr at baseline, in Zûrich.[14] Our study adds information on the common trajectories of insomnia symptoms over 2 decades among older adults and shows patterning by sex, occupational class, and cohort.

Insomnia symptoms have traditionally been subtyped into problems with initiating sleep, maintaining sleep, or waking earlier than desired, though the latter may often be perceived as a maintenance problem. In a study examining all 3 symptom types over 4 mo, each symptom type in isolation had low stability, with higher stability for a combination of all 3.[16] A cross-sectional latent class analysis on data from patients with sleep disturbance also identified a class that experienced both initiation and maintenance symptoms, whereas maintenance problems predominated in other classes.[33] In this cross-sectional analysis, the class with mixed symptoms also reported more frequent symptoms of longer duration than other classes. The *Chronic Mixed* class we found is consistent with this previous research, particularly in associating chronicity with the combined experience of both initiation and maintenance problems. The *Episodic Maintenance* class may indicate a distinct vulnerability for intermittent troubled sleep. Inevitably, some of these individuals would also be captured as poor sleepers in cross-sectional studies. Differences in the social patterning of the *Chronic Mixed* and *Episodic Maintenance* classes suggest differences in etiology that could be confounded in cross-sectional research.

This study goes beyond cross-sectional associations between SES and insomnia symptoms[18,20-21,34] by showing that people in a manual occupational class were more likely than those in a nonmanual class to experience chronic insomnia symptoms. Occupational class differences may be attributable to differences in work and family characteristics,[35] in health behaviors such as smoking and drinking which are associated with poorer sleep,[1] or to socioeconomic inequalities in physical and mental health, which have both been prospectively associated with later insomnia.[13-14,22] Alternatively, people with consistently poor sleep may have experienced daytime fatigue, impairing work performance and making it difficult to obtain or retain higher status jobs. Respondents in manual classes were also more likely to be in the *Developing* class than those in nonmanual classes. Previous 12-mo incidence studies[13,22] found no SES differences, but this may have been due to the shorter follow-up period. Because occupational class preceded the development of symptoms in our analysis, this finding is broadly supportive of a causal role for occupational class, though the assumption that the *Developing* class is primarily made up of incident cases may be questionable; some may be relapsing after prolonged remission.

Respondents in the 1930s cohort were more likely than those in the 1950s cohort to experience *Chronic Mixed* or *Developing* relative to *Healthy* sleep patterns. Effects for the *Chronic Mixed* class were stronger in women, and for the *Developing* class were evident in men only. Because respondents in the same cohort were approximately the same age, these cohort differences could either be interpreted as differences in the patterning of insomnia symptoms with increasing age or as differences between birth cohorts, the former being consistent with conclusions from previous research that the prevalence of insomnia symptoms increases with age.[1] We suggest this may be underpinned by the persistence of (prior) insomnia into later years in some, plus the emergence of other incident new cases. Comparison between interviews where respondents were the same age suggested a lower prevalence of symptoms in the older cohort. Unless this is due to question wording or attrition this finding suggests that age differences may be underestimated due to an opposing cohort effect.

The common finding of poorer sleep among women[17,20-21,35] is replicated here, and is extended to show patterns of poorer sleep across the life course. Women appear more likely than men to develop insomnia symptoms in middle age (i.e. mid-30s to mid-50s), and then to retain the disadvantage as chronic symptoms in old age (i.e. mid-50s to mid-70s). This is consistent with meta-analytical findings that sex differences in insomnia increase with age.[17] Other studies have shown the effect of sex to be attenuated but not fully explained by adjustment for socioeconomic disadvantage,[20-21] and our findings concur, with distinct effects for both sex and occupational class. Biologic factors may also contribute to sex differences; the menopause transition, for example, is associated with deleterious effects on sleeping patterns.[36] This would be consistent with our observation of high risk for women developing symptoms as they move into their 50s. However, insomnia symptoms around

menopause could be attributable to other experiences common to women in midlife such as the development of acute and chronic health conditions or life stresses with associated increases in depression.[37-38]

This study has various limitations. First, it examines a fairly restricted range of insomnia symptoms; no measures of daytime consequences of sleep disturbance or dissatisfaction with sleep were used.[1] The change in question wording meant that respondents were asked about their symptoms generally in the first 3 interviews, and then specifically about symptoms in the past month for the last 2 interviews. If people respond differently based on the timeframe of the question then this could result in spurious developmental patterns, but a parallel analysis of another question on sleep that was consistently worded revealed similar findings to those presented. There may also be some unaddressed heterogeneity in the severity of symptoms reported: symptoms reported could have been anything from nightly to weekly sleeping trouble. Moreover, self- reporting biases by variables of interest may have been present; other research has shown males with insomnia to be more likely to overreport sleep disturbance than women with insomnia,[39] which could mean the sex differences observed here are underestimated. Small studies of sleep quality using objective measurement provide mixed support for these findings with poorer sleep for those in socioeconomic disadvantage,[40-41] but either no sex differences or better sleep quality for women.[39,42-43] Furthermore, the data were based on repeated snapshots rather than complete histories and so symptoms were not necessarily stable between interviews and might have been experienced prior to baseline data collection. Differential drop-out may also have introduced some bias. For example, if those who recovered from baseline symptoms were more likely to participate in later waves than those who did not recover, then levels of chronicity may have been underestimated. Finally, if occupational class has a causal relationship with insomnia, then observed effects here will have been diluted as some respondents from the manual group will have moved into nonmanual occupations over time (and thus experienced better sleep than those remaining in manual occupations) and *vice versa*.

In conclusion, 4 main patterns of insomnia symptoms over a 20-yr period have been shown among middle-aged and older adults: a healthy pattern; an episodic pattern characterized by problems maintaining sleep; a more chronic pattern with trouble both maintaining and initiating sleep; and a pattern of developing sleep symptoms. Experience of these different longitudinal patterns was stratified by social factors, namely age, sex, and occupational class.

ACKNOWLEDGMENTS

The West of Scotland Twenty-07 Study is funded by the UK Medical Research Council (MRC) (MC_US_A540_0080) and the data were originally collected by the MRC/CSO Social and Public Health Sciences Unit. The authors are grateful to all of the participants in the study, and to the survey staff and research nurses who carried it out. Michael J Green, Michaela Benzeval (MC_US_A540_0056) and Kate Hunt (MC_US_A540_0036) are also funded by the MRC. The authors are grateful to anonymous reviewers for their helpful comments on an earlier version of this manuscript.Work for this study was performed at the Medical Research Council/Chief Scientist's Office: Social and Public Health Sciences Unit in Glasgow, United Kingdom.

DISCLOSURE STATEMENT

This was not an industry supported study. Dr. Espie has received research support from Sanofi-Aventis, GSK, Actelion, Takeda, Lundbeck and Sharp. The other authors have indicated no financial conflicts of interest.

REFERENCES

1. Ohayon M. Epidemiology of insomnia: what we know and what we still need to learn. Sleep Med Rev 2002;6:97-111.
2. Diagnostic and Statistical Manual of Mental Disorders, 4th edition (DSM IV). Washington: American Psychiatric Association, 1994.
3. International Classification of Sleep Disorders: Diagnostic and Coding Manual: 2nd edition. Westchester, IL: American Academy of Sleep Medicine, 2005.
4. Riemann D, Voderholzer U. Primary insomnia: a risk factor to develop depression? J Affect Disord 2003;76:255-9.
5. Cole MG, Dendukuri N. Risk factors for depressionamong elderly community subjects: a systematic review and meta-analysis. Am J Psychiatry 2003;160:1147-56.
6. Baglioni C, Battagliese G, Feige B, et al. Insomnia as a predictor of depression: a meta-analytic evaluation of longitudinal epidemiological studies. J Affect Disord [available online: 5 Feb 2011], http://dx.doi.org/10.1016/j.jad.2011.01.011.
7. Vgontzas AN, Liao D, Bixler EO, Chrousos GP, Vela-Bueno A. Insomnia with objective short sleep duration is associated with a high risk for hypertension. Sleep 2009;32:491-7.
8. Vgontzas AN, Liao D, Pejovic S, Calhoun S, Karataraki M, Bixler EO. Insomnia with objective short sleep duration is associated with type 2 diabetes: a population-based study. Diabetes Care 2009;32:1980-5.
9. Vgontzas AN, Liao D, Pejovic S, et al. Insomnia with short sleep duration and mortality: the Penn State Cohort. Sleep 2010;33:1159-64.
10. Zammit G, Weiner J, Damato N, Sillup G, McMillan C. Quality of life in people with insomnia. Sleep 1999;22:S379-S85.
11. Doi Y, Minowa M, Tango T. Impact and correlates of poor sleep quality in Japanese white-collar employees. Sleep 2003;26:467-71.
12. Hyyppä MT, Kronholm E, Alanen E. Quality of sleep during economic recession in Finland: A longitudinal cohort study. Soc Sci Med 1997;45:731-8.
13. Morphy H, Dunn KM, Lewis M, Boardman HF, Croft PR. Epidemiology of insomnia: a longitudinal study in a UK population. Sleep 2007;30:274-80.
14. Buysse DJ, Angst J, Gamma A, Ajdacic V, Eich D, Roessler W. Prevalence, course, and comorbidity of insomnia and depression in young adults. Sleep 2008;31:473-80.
15. Morin CM, Belanger L, LeBlanc M, et al. The natural history of insomnia: a population-based 3-year longitudinal study. Arch Intern Med 2009;169:447-53.
16. Hohagen F, Käppler C, Schramm E, Riemann D, Weyerer S, Berger M. Sleep onset insomnia, sleep maintaining insomnia and insomnia with early morning awakening - temporal stability of subtypes in a longitudinal study on general practice attenders. Sleep 1994;17:551-4.
17. Zhang B, Wing Y-K. Sex Differences in insomnia: a meta-analysis. Sleep 2006;29:85-93.
18. Sekine M, Chandola T, Martikainen P, McGeoghegan D, Marmot M, Kagamimori S. Explaining social inequalities in health by sleep: the Japanese civil servants study. J Public Health 2006;28:63-70.
19. Patel N, Grandner M, Xie D, Branas C, Gooneratne N. "Sleep disparity" in the population: poor sleep quality is strongly associated with poverty and ethnicity. BMC Public Health 2010;10:475.
20. Arber S, Bote M, Meadows R. Gender and socio-economic patterning of self-reported sleep problems in Britain. Soc Sci Med 2009;68:281-9.
21. Chen Y-Y, Kawachi I, Subramanian SV, Acevedo-Garcia D, Lee Y-J. Can social factors explain sex differences in insomnia? Findings from a national survey in Taiwan. J. Epidemiol Community Health 2005;59:488-94.
22. LeBlanc M, Merette C, Savard J, Ivers H, Baillargeon L, Morin CM. Incidence and risk factors of insomnia in a population-based sample. Sleep 2009;32:1027-37.

23. Benzeval M, Der G, Ellaway A, et al. The West of Scotland Twenty-07 Study: health in the community: cohort profile. Int J Epidemiol. 2009;38:1215-23.
24. Der G. A comparison of the West of Scotland Twenty-07 Study sample and the 1991 census SARs. Glasgow: MRC Medical Sociology Unit Working Paper No. 60, 1998.
25. Buysse DJ, Reynolds CF, Monk TH, Berman SR, Kupfer DJ. The Pittsburgh sleep quality index: A new instrument for psychiatric practice and research. Psychiatry Res 1989;28:193-213.
26. Galobardes B, Shaw M, Lawlor DA, Lynch JW, Davey Smith G. Indicators of socioeconomic position (part 1). J Epidemiol Community Health 2006;60:7-12.
27. Galobardes B, Shaw M, Lawlor DA, Lynch JW, Davey Smith G. Indicators of socioeconomic position (part 2). J. Epidemiol Community Health 2006;60:95-101.
28. Office of Population Censuses and Surveys. Classification of Occupations. London: HMSO, 1980.
29. Lorimer K, Green M, Shipton D, Benzeval M. The West of Scotland Twenty-07 Study: health in the community: Wave 5 Fieldwork Report, 2007/8. Glasgow: MRC/CSO Social and Public Health Sciences Unit Working Paper 26, 2010.
30. Collins LM, Lanza ST. Latent class and latent transition analysis with applications in the social, behavioral, and health sciences. Hoboken, New Jersey: John Wiley & Sons Inc., 2010.
31. Muthén LK, Muthén BO. Mplus User's Guide. Sixth ed. Los Angeles, CA: Muthén & Muthén, 1998-2010.
32. Clarke P, Hardy R. Methods for Handling Missing Data. In: Pickles A, Maughan B, Wadsworth M, eds. Epidemiological Methods in Life Course Research. Oxford: Oxford University Press, 2007:157-80.
33. Foley KA, Sarsour K, Kalsekar A, Walsh JK. Subtypes of sleep disturbance: associations among symptoms, comorbidities, treatment, and medical costs. Behav Sleep Med 2010;8:90-104.
34. Moore PJ, Adler NE, Williams DR, Jackson JS. Socioeconomic status and health: the role of sleep. Psychosom Med 2002;64:337-44.
35. Sekine M, Chandola T, Martikainen P, Marmot M, Kagamimori S. Work and family characteristics as determinants of socioeconomic and sex inequalities in sleep: The Japanese Civil Servants Study. Sleep 2006;29:206-16.
36. Owens JF, Matthews KA. Sleep disturbance in healthy middle-aged women. Maturitas 1998;30:41-50.
37. McKinlay JB, McKinlay SM, Brambilla D. The relative contributions of endocrine changes and social circumstances to depression in mid-aged women. J Health Soc Behav 1987;28:345-63.
38. Mitchell ES, Woods NF. Symptom experiences of midlife women: observations from the Seattle midlife women's health study. Maturitas 1996;25:1-10.
39. Voderholzer U, Al-Shajlawi A, Weske G, Feige B, Riemann D. Are there gender differences in objective and subjective sleep measures? A study of insomniacs and healthy controls. Depress Anxiety 2003;17:162-72.
40. Mezick EJ, Matthews KA, Hall M, et al. Influence of race and socioeconomic status on sleep: Pittsburgh SleepSCORE Project. Psychosom Med 2008;70:410-6.
41. Friedman EM, Love GD, Rosenkranz MA, et al. Socioeconomic status predicts objective and subjective sleep quality in aging women. Psychosom Med. 2007;69:682-91.
42. Hume K, Van F, Watson A. A field study of age and gender differences in habitual adult sleep. J Sleep Res 1998;7:85-94.
43. Fukuda N, Honma H, Kohsaka M, et al. Gender difference of slow wave sleep in middle aged and elderly subjects. Psychiatry Clin Neurosci 1999;53:151-3.

SUPPLEMENTAL MATERIAL

Part I: Determining the Number of Classes

The objective at this stage was to find the latent class model that best represented the data, i.e., striking a balance between maximization of model fit and keeping the number of latent classes low (maximizing parsimony). A sequence of latent class models with ascending numbers of latent classes were computed in each cohort, and their model-fit statistics were compared to decide on the most appropriate number of classes (see Table S1). Model fit was assessed using the likelihood ratio χ^2 statistic, the Akaike Information Criterion (AIC),[1] and the Bayesian Information Criterion (BIC).[2] Lower values for the χ^2 likelihood ratio indicate improvements in model fit, and lower values for AIC and BIC suggest a more optimal balance between model fit and model parsimony. To check model identification 100 different sets of random start values were used for each latent class model. Where the best-fitting solution occurs with a high frequency (e.g., 100 times), then there can be greater confidence that the solution represents a global rather than a local maximization of model fit. We also examined entropy,[3] which indicates how distinctly respondents are being classified into latent classes by the models. Values closer to 1 are preferred as they suggest that respondents are being more definitively classified by the model. We stopped at 7 latent classes as model parsimony (measured by the BIC) and identification were clearly deteriorating with additional classes instead of improving.

In both cohorts, additional classes were at first associated with large increases in model fit (signified by large decreases in the χ^2, AIC, and BIC statistics). However, the improvement lessened with each additional class and the BIC, which offers the more stringent balance of model fit against parsimony, began to increase after a low at 4 classes. Model identification also decreased after 4 classes, suggesting that there can be less confidence in solutions that used greater numbers of classes. An increase from 4 to 5 classes, while only associated with a small rise in the BIC, also only offered a very marginal gain in terms of entropy. Taking all of these findings into consideration, the 4-class solution was seen as the best-fitting model. As this optimal 4-class model exhibited a very similar pattern of response probabilities in each cohort, it was thought sensible to combine the 2 cohorts so that statistical comparisons of latent class membership probabilities could be made between cohorts (see supplementary information, part II). As the models with more than 4 classes produced patterns of response probabilities with substantive differences between cohorts and were not well identified, models of both cohorts with more than 4 latent classes were not preferred despite increases in model fit.

Part II: Testing for Measurement Invariance Across Sex, Occupational Class, and Cohort Groups

After establishing that the 4-class model provided the best fit to the data, we then investigated covariate effects. The most

Table S1—Comparison of fit statistics and identification for models with different numbers of latent classes

No. of classes	Likelihood ratio χ^2	Degrees of freedom[a]	AIC	BIC	Identification[b]	Entropy
1950s Cohort (n = 1,383)						
1	1,822.32	993	12,368.10	12,420.42	100	1.00
2	1,054.01	998	10,925.19	11,035.07	100	0.72
3	803.26	983	10,731.08	10,898.50	96	0.67
4	723.23	974	10,655.01	10,879.98	83	0.64
5	656.97	962	10,610.82	10,893.36	10	0.68
6	606.19	950	10,585.01	10,925.09	8	0.70
7	582.27	941	10,565.52	10,963.15	3	0.72
1930s Cohort (n = 1,484)						
1	2,381.79	1,004	12,775.72	12,828.74	100	1.00
2	1,100.25	989	11,405.72	11,517.07	100	0.72
3	849.24	978	11,175.93	11,345.61	46	0.65
4	691.80	969	11,020.28	11,248.28	94	0.58
5	626.56	959	10,976.90	11,263.23	62	0.59
6	589.88	948	10,960.27	11,304.94	32	0.58
7	561.49	939	10,951.08	11,354.07	1	0.60
Both Cohorts (n = 2,867)						
2	2,307.41	2,009	26,404.51	26,541.61	100	0.85
3	1,719.48	1,993	25,871.82	26,080.45	99	0.79
4	1,496.30	1,985	25,643.75	25,923.92	99	0.73
5	1,398.77	1,975	25,552.08	25,903.78	33	0.74

[a]These values do not descend uniformly with the number of classes because between 4 and 20 cells in each latent class indicator table were deleted in the calculation of the chi-square due to extreme values. [b]Indicates how many times the best-fitting solution was found using 100 different sets of random starting values. AIC, Akaike Information Criterion; BIC, Bayesian Information Criterion.

Table S2—Comparison of model fit statistics for variant and invariant latent class models across sex, occupational class, and cohort groupings

Model (n = 2,867)	Likelihood ratio χ^2	Degrees of freedom	P value	AIC	BIC	Identification[a]	Entropy
Sex							
Variant	1,389.75	1,945	–	25,661.81	26,180.41	53	0.74
Invariant	1,470.16	1,985	–	25,663.02	25,943.18	91	0.73
Difference	+80.41	+40	< 0.01	+1.21	-237.23	+38	-0.01
Occupational Class							
Variant	1,393.25	1,945	–	25,558.11	26,076.72	73	0.73
Invariant	1,442.63	1,984	–	25,531.29	25,811.46	92	0.73
Difference	+49.38	+39	< 0.01	-26.82	-265.26	+19	0.00
Cohort							
Variant	1,415.03	1,943	–	25,648.23	26,166.84	61	0.74
Invariant	1,496.30	1,985	–	25,643.75	25,923.92	99	0.73
Difference	+81.27	+42	< 0.01	-4.48	-242.92	+38	-0.01
Cross-Classified Sex, Class, and Cohort Groupings							
Variant	2,235.43	7,792	–	33,372.18	35,464.50	1	0.87
Invariant	2,753.79	8,081	–	33,278.54	33,701.77	97	0.84
Difference	+518.36	+289	< 0.01	-93.64	-1,762.73	+96	-0.03

[a]Indicates how many times the best-fitting solution was found using 100 different sets of random starting values. AIC, Akaike Information Criterion; BIC, Bayesian Information Criterion.

parsimonious covariate model will be one in which the covariates influence class membership only, and not the probability of responses given class membership.[4] Such a condition is referred to as measurement invariance because the latent class definitions are constant across all levels of the covariates; that is, a latent class means the same thing for 1 group as it does for others. Measurement invariance indicates that different groups experience similar rather than entirely different latent class patterns.

Measurement invariance can be formally tested for by comparing a model where response probabilities for latent classes can vary across covariate groupings (the variant model) with a nested model where response probabilities for latent classes are constrained to be equal across covariate groupings (the invariant model). This is a sensitive test, however, and it may indicate that differences are statistically significant where they make little substantive difference to the interpretation of the latent classes. For this reason it is important to examine not only the outcome of this test, but also other model-fit statistics and the response probabilities for the latent classes from both models. Where measurement invariance is statistically significant, but does not alter the substantive interpretation of the classes, it can be more parsimonious to impose measurement invariance anyway, allowing for like-for-like comparisons of class membership probabilities.[4]

Table S2 displays fit statistics and chi-square difference tests for variant and invariant models across sex, occupational class, and cohort groupings, and also for a cross-classification identifying the 8 possible unique combinations of these variables. Each pair of models was tested on the whole sample (i.e., both cohorts). In each case the chi-square test showed significant group differences in response probabilities for latent classes. Inspection of the response probability estimates, however, revealed latent class patterns that were very similar and did not differ markedly in terms of interpretation (results not shown). Additionally, the invariant model was in each case better identified and had lower BIC values and the AIC was lower in most cases. Allowing response probabilities to vary across covariate groupings did not produce large improvements in the classification of respondents as indicated by the entropy statistic. Overall, it was thought that group differences were minor and imposing measurement invariance gained more in terms of parsimony than was lost in accuracy, or model fit. Particularly, this meant that like-for-like statistical comparisons could be made of latent class membership by sex, occupational class, and cohort.

Part III: Separate Modeling of Different Insomnia Symptom Questions

Table S3 displays information on model fit from models with 2, 3, and 4 classes for each of the sleep variables. In each case the 3-class model has the lowest value for the BIC, representing an optimal balance of model fit and parsimony.

Figure S1 shows the item-response probabilities for the 3-class model of each variable. As the difficulty sleeping question does not distinguish between problems with latency and maintenance, it was thought better to compare it with the 2 separate models of latency and maintenance symptoms rather than the combined model. Each of the 3 models shows classes similar to the *Developing, Chronic Mixed,* and *Healthy* classes shown in the main results. For the question on difficulty sleeping, the increase in the probability of a positive response over the study in the *Developing* pattern shows a smoother gradient than for the 2 variables where the question wording changed, but is notably still present. This suggests that the change in question wording may have accentuated the incline in symptom patterns, but that the incline is nevertheless genuine and not an artefact of question wording.

Table S3—Comparison of fit statistics and identification for models of different insomnia symptom questions

No. of classes	Likelihood ratio χ^2	Degrees of freedom	AIC	BIC	Identification[a]	Entropy
Latency (n = 2,866)						
2	161.27	20	10,376.75	10,466.16	100	0.66
3	45.82	14	10,210.03	10,359.05	82	0.56
4	13.55	8	10,186.22	10,394.85	56	0.59
Maintenance (n = 2,867)						
2	126.17	20	12,548.15	12,637.56	100	0.53
3	28.12	14	12,419.76	12,568.78	90	0.51
4	13.07	8	12,410.96	12,619.60	66	0.57
Difficulty sleeping (n = 2,924)						
2	98.43	20	12,163.62	12,253.33	100	0.59
3	62.27	14	12,100.46	12,249.98	63	0.51
4	25.07	8	12,052.32	12,261.64	59	0.49

[a]Indicates how many times the best-fitting solution was found using 100 different sets of random starting values. AIC, Akaike Information Criterion; BIC, Bayesian Information Criterion.

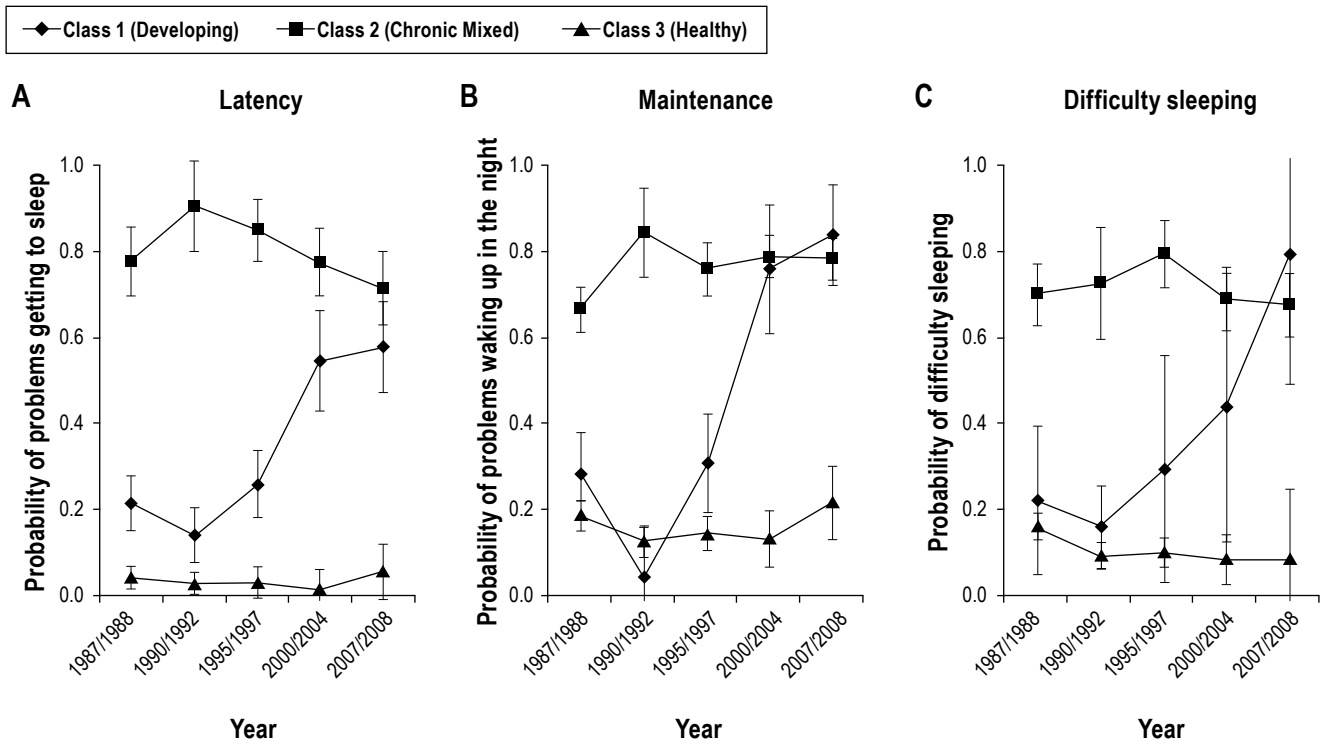


Figure S1—Response probabilities from separate 3-class models of each insomnia symptom question.

Table S4 compares the overall latent class membership probabilities and logistic regression effects of covariates in each of these 3 models. Some degree of variation would be expected here as those who would have been assigned to the *Episodic Maintenance* class in the main results will have been reclassified into other classes, affecting both membership probabilities and estimates of odds ratios. The membership probabilities for the maintenance and difficulty sleeping variables, however, did exhibit similar patterns, and covariate effects were also fairly consistent across all 3 models. Notably, the propensity for women to be in the *Developing* rather than the *Healthy* sleep class (relative to men) was consistent and significant across all models, as was the tendency for those in manual relative to nonmanual classes to be in the *Chronic Mixed* rather than the *Healthy* sleep class. Almost all of the other effects that were not significant for the question on difficulty sleeping were also not significant in either the latency-only or the maintenance-only model. These differences may therefore have more to do with only modeling a single dimension of sleep disturbance than with consistency in question wording.

Table S4—Comparison of class membership probabilities and covariate effects for different insomnia symptom questions

	Latency		Maintenance		Difficulty Sleeping	
	Value	95% Confidence intervals	Value	95% Confidence intervals	Value	95% Confidence intervals
Class membership probabilities						
Developing	0.40	–	0.24	–	0.20	–
Chronic	0.19	–	0.35	–	0.28	–
Healthy	0.41	–	0.41	–	0.52	–
Odds ratios for covariates						
Developing (ref: Healthy)						
Female (ref: Male)	3.34	(2.11-5.29)	5.13	(2.85-9.26)	5.21	(1.95-13.94)
Manual (ref: Non-Manual)	2.06	(1.49-2.85)	1.43	(0.90-2.27)	0.99	(0.40-2.43)
1930s Cohort (ref: 1950s)	1.98	(1.21-3.25)	3.24	(1.71-6.13)	1.23	(0.44-3.44)
Female*1930s Cohort	0.87	(0.42-1.83)	0.19	(0.08-0.44)	0.47	(0.13-1.64)
Chronic (ref: Healthy)						
Female (ref: Male)	0.99	(0.61-1.61)	1.61	(1.06-2.43)	1.40	(0.71-2.77)
Manual (ref: Non-Manual)	3.35	(2.50-4.50)	2.09	(1.63-2.66)	1.69	(1.33-2.15)
1930s Cohort (ref: 1950s)	1.30	(0.90-1.89)	2.04	(1.43-2.92)	1.16	(0.83-1.62)
Female*1930s Cohort	3.89	(2.16-7.02)	1.10	(0.64-1.89)	1.82	(0.93-3.57)

Expected differences aside, the data from the question on difficulty sleeping, which was consistently defined across the study, produces an overall pattern of results similar to those obtained by using the questions on maintenance and latency, which were not consistently worded. This suggests that the effect of the change in question wording was minor and that the observed results are reflective of actual population sleep patterns rather than artefacts of measurement.

REFERENCES

1. Akaike H. A new look at the statistical model identification. IEEE Trans Automat Contr 1974;19:716-23.
2. Schwarz G. Estimating the dimension of a model. Ann Stat 1978;6:461-4.
3. Ramaswamy V, Desarbo WS, Reibstein DJ, Robinson WT. An empirical pooling approach for estimating marketing mix elasticities with PIMS data. Marketing Science 1993;12:103-24.
4. Collins LM, Lanza ST. Latent class and latent transition analysis with applications in the social, behavioral, and health sciences. Hoboken, New Jersey: John Wiley & Sons Inc., 2010.

The Daytime Impact of *DSM-5* Insomnia Disorder: Comparative Analysis of Insomnia Subtypes From the Great British Sleep Survey

Colin A. Espie, PhD; Simon D. Kyle, PhD; Peter Hames, MA Oxon; Eva Cyhlarova, PhD; and Michaela Benzeval, PhD

ABSTRACT

Objective: To profile the daytime impact of the proposed *DSM-5* insomnia disorder diagnosis, with and without mental health, physical health, or other sleep disorder comorbidities; to better understand how specific daytime symptom patterns are associated with nighttime sleep in insomnia; and to compare childhood-onset and adulthood-onset insomnia disorder with respect to daytime dysfunction.

Method: Data were derived from the Great British Sleep Survey (GBSS), an open-access online population survey completed by adults who had a valid postcode and were residents of the United Kingdom. The primary variables of interest were the 6 areas that, according to the proposed *DSM-5* criteria, may be impacted in the daytime by insomnia disorder: energy, concentration, relationships, ability to stay awake, mood, and ability to get through work. These variables were compared for those with versus those without insomnia disorder and across 5 insomnia subtypes (difficulty initiating sleep, difficulty maintaining sleep, early morning awakening, a combination of these 3 core symptoms, or nonrestorative sleep). Clinically comorbid insomnia presentations (insomnia disorder with poor mental health/poor physical health/additional sleep disorder symptoms) and insomnia disorder of childhood versus adult onset were also evaluated.

Results: A total of 11,129 participants (72% female; mean age = 39 years) completed the GBSS between March 2010 and April 2011, of whom 5,083 screened as having possible insomnia disorder. Compared with those who did not have insomnia disorder, those with insomnia disorder reported greater impairment in all areas of daytime functioning (Cohen *d* range, 0.68–1.30). The greatest effects reflected negative impact on energy and mood. Participants with a combination of insomnia symptoms tended to be the most impaired (Cohen *d* range, 0.10–0.23), whereas no consistent differences emerged between the other 4 subtypes. Finally, individuals who had both insomnia disorder and poor mental health were consistently the most impaired comorbid group (Cohen *d* range, 0.15–0.65), and childhood-onset insomnia disorder had greater daytime impact than adult-onset insomnia disorder ($P < .05$ for energy; $P < .01$ for mood, concentration, and getting through work).

Conclusions: The severity of daytime impact of *DSM-5* insomnia disorder varies by insomnia type. This finding has implications for the evaluation and management of insomnia in clinical practice.

J Clin Psychiatry 2012;73(00):e000–e000

Submitted: *June 18, 2012; accepted August 16, 2012 (doi:10.4088/JCP.12m07954).*
Corresponding author: *Colin A. Espie, PhD, University of Glasgow Sleep Centre, Sackler Institute of Psychobiological Research and Institute of Neuroscience & Psychology, College of Medical, Veterinary & Life Sciences, University of Glasgow, G51 4TF, Scotland (colin.espie@glasgow.ac.uk).*

The adoption in *DSM-5* of the term *insomnia disorder* (ID) (M 00) reflects a paradigm shift, recommended by the National Institutes of Health,[1] toward coding insomnia "whenever diagnostic criteria are met, whether or not there is a coexisting psychiatric, medical, or another sleep disorder."[2] In laying aside the *DSM-IV*[3] perspective of "primary" versus "secondary" insomnia, the *DSM-5* Sleep-Wake Disorders Work Group has recognized that poor sleep may be associated with illness vulnerability.[4] For example, chronic insomnia is a risk factor for the evolution of, and relapse into, depression,[5] and cotreatment of insomnia improves depression outcomes.[6,7] Likewise, insomnia has been associated with physical disease (eg, hypertension,[8] type 2 diabetes[9]) and all-cause mortality[10] and often copresents with sleep apnea[11] where, if untreated, it may exacerbate daytime impairment, particularly excessive daytime sleepiness.[12] A further development in *DSM-5*, consistent with research diagnostic criteria and contemporary data,[13,14] is that insomnia must have a specified sequela (fatigue, daytime sleepiness, cognitive impairment, mood disturbance, impaired work function, impaired interpersonal function),[2] contrasting with the general *DSM-IV* statement of "significant distress or impairment in social, occupational, or other important areas of functioning."[3(p557)]

It seems timely to consider how these domains of daytime impairment might associate, in nature or severity, with the ID subtypes of difficulty initiating sleep, difficulty maintaining sleep, early morning awakening, and nonrestorative sleep. Although their investigation did not use *DSM-5* criteria, Léger et al,[15] reporting on primary care patients (n = 3,384), found that those with a combination of nighttime symptoms had the most severe impairments. In a controlled, in-laboratory study, Roth et al[16] found that patients with nonrestorative sleep (n = 115) reported daytime impairment similar in magnitude to those with difficulty initiating sleep (n = 56), difficulty maintaining sleep (n = 18), or a combination of symptoms (n = 37). Walsh et al[17] found that nonrestorative sleep was associated with the poorest perceived health in a well-defined sample of US health plan members (n = 6,791). The identification of *both* nighttime and daytime symptoms may be particularly important in advancing understanding of how insomnia impacts mental health.[18,19] Clinical history also requires further study. Preliminary reports suggest that childhood-onset insomnia is associated with more severe sleep complaints and a different profile of daytime dysfunction relative to adult-onset insomnia.[20–22]

Clinical Points

- *DSM-5* proposes to move away from "primary" and "secondary" insomnia, recognizing that causality is often hard to determine, that sleep and comorbid conditions interact in a bidirectional manner, and that sleep disturbance is associated with illness vulnerability.
- The new diagnostic entity of insomnia disorder highlights that significant sleep disturbance merits independent clinical attention, regardless of additional comorbidities.
- The daytime consequences of insomnia disorder are most pronounced for those with a mixed subtype (problems with both initiating and maintaining sleep), those with poor mental health, and those with insomnia of childhood onset.

In March 2010, the Great British Sleep Survey (GBSS), an online population survey based on detailed *DSM-5* criteria, was launched. The survey was conducted by Sleepio Limited (an organization dedicated to helping people sleep better through raising awareness, research, and dissemination of behavioral advice), in association with Boots UK and the Mental Health Foundation (London, England). Our major objectives were to compare and contrast both sleep disturbance and its daytime impact (1) in ID relative to a reference group with no ID, (2) in the ID subtypes (difficulty initiating sleep vs difficulty maintaining sleep vs early morning awakening vs a combination of these symptoms vs nonrestorative sleep), (3) in a comparison of childhood-onset and adult-onset ID, and (4) across clinically comorbid mental health, physical health, and other sleep disorder conditions (ID + mental health condition vs ID + physical health condition vs ID + other sleep disorder vs ID only).

METHOD

Design

We report on 11,129 respondents to the GBSS, an open-access, Web-based survey completed by adults (aged ≥ 18 years) who had a valid postcode and were residents of the United Kingdom. The strengths of this approach include accessibility, ease of use, time-stamping of data acquisition, absence of missing information, and ability to recruit a sizable sample of people meeting ID criteria. Our data are not formally sampled, because it was not our purpose to report population incidence or prevalence figures. Rather, we suggest that the sample is valid in relation to our objectives of profiling the nighttime symptoms and daytime concerns of people who meet ID criteria, with or without comorbid symptoms.

Measures

The GBSS is a brief online survey comprising personal and demographic information; appraisal of sleep pattern, sleep quality, and impact of poor sleep on daytime functioning; use of prescription and over-the-counter sleep aids; items on physical and mental health; and screening questions on sleep disorders other than insomnia. (Illustration of our methods can be viewed at http://www.sleepio.com/research, and the contemporary version of the GBSS is at http://www.greatbritishsleepsurvey.com.) The GBSS incorporated items on sleep and daytime function to permit evaluation against *DSM-5* criteria and to take account of quantitative insomnia criteria including research diagnostic criteria.[13,23] Insomnia disorder cases were defined according to the following criteria:

1. Current complaint of sleep dissatisfaction/concern (ie, scoring ≥ 2 ["somewhat"] on a 0–4 scale when asked, "Over the past month, to what extent has poor sleep troubled you in general?").
2. Complaint comprises 1 of the following:
   - Difficulty initiating sleep ≥ 31 minutes,
   - Difficulty maintaining sleep ≥ 31 minutes (individual is awake for ≥ 31 minutes during the night after initially falling asleep; could include 1 or multiple awakenings),
   - Early morning awakening (final awakening ≥ 31 minutes prior to actual rise time),
   - A combination of at least 2 of these 3 core insomnia symptoms, or
   - Nonrestorative sleep (difficulty initiating and maintaining sleep and early morning awakening ≤ 30 minutes, but all other ID criteria met).
3. Failure to endorse "very good" or "good" sleep quality (ie, required to score ≥ 2 ["average"] on a 0–4 scale when asked, "Over the past month, how would you rate your sleep quality?").
4. Complaint is associated with significant sleep-related daytime effects (ie, scoring ≥ 2 ["somewhat"] on a 0–4 scale; 0 = not at all affected, 4 = very much affected) on at least 1 of 6 domains: energy, daytime sleepiness, cognitive impairment, mood disturbance, impaired work functioning, impaired relationship functioning.
5. Sleep difficulty is reported to be affecting the person ≥ 3 nights per week.
6. Sleep difficulty has been occurring for ≥ 3 months.

The GBSS also incorporated the Sleep Disorders Screening Questionnaire, a published clinical tool for conservatively identifying possible cases of narcolepsy, obstructive sleep apnea, restless legs syndrome/periodic limb movements in sleep, circadian rhythm sleep disorder, and parasomnia[24] (see algorithm at http://www.sleepio.com/research). The GBSS inquired about health status by means of 2 items: "For my age I believe that my physical health is…" and "For my age I believe that my mental health is…," both rated on a 5-point Likert scale (0 = very good, 1 = good, 2 = average, 3 = poor, 4 = very poor). For this analysis, poor physical health and poor mental health were defined by a score ≥ 3 on the respective ratings.

Table 1. Comparison of Respondents With and Without Insomnia Disorder in Relation to Demographic and Sleep Variables

Variable	Insomnia Disorder (n = 5,083)	No Insomnia Disorder (n = 5,542)	Total Sample (N = 10,625)
Gender, % male/female	25.3/74.7*	30.8/69.2	28.2/71.8
Age, mean (SD), y	41.3 (14.8)*	37.6 (14.0)	39.4 (14.5)
Index of multiple deprivation score, mean (SD)[a]	20.1 (14.3)	20.3 (14.5)	20.2 (14.4)
Physical health score, mean (SD)[b]	1.72 (0.95)*	1.43 (0.89)	1.57 (0.93)
Mental health score, mean (SD)[b]	1.82 (1.08)*	1.29 (0.99)	1.54 (1.07)
SCI score, mean (SD)[c]	3.02 (1.25)*	6.95 (1.82)	5.07 (2.52)
Taking prescribed sleeping pills, %	12.0*	2.8	7.2
Taking over-the-counter sleep remedies, %	24.3*	9.8	16.7
Insomnia duration, % reporting			
< 12 mo	17.0		
1–5 y	38.0		
6–10 y	17.0		
≥ 11 y	28.0		

[a]A proxy for socioeconomic status, determined through residential postcode. Based on English residents only.
[b]Lower scores indicate better perceived health: 0 = very good, 4 = very poor.
[c]Scale of 0 to 10; higher values reflect better overall sleep quality.
*$P < .0001$ for comparisons between respondents with and without insomnia disorder.
Abbreviation: SCI = Sleep Condition Indicator.

Items from the GBSS were also used to calculate the Sleep Condition Indicator (SCI; 0–10 range), on which higher values reflect better overall sleep quality. The SCI has excellent sensitivity and specificity, high internal consistency reliability, and sensitivity to change following cognitive-behavioral therapy.[25] The SCI also correlates with other standard measures of sleep quality (Pittsburgh Sleep Quality Index: $r = 0.78$, n = 256; Insomnia Severity Index: $r = 0.79$, n = 256).[26]

Statistical Analysis

Potential differences associated with expressions of the independent variable (sleep status) on the primary dependent variables of interest (6 daytime domains) were evaluated using multivariate analysis of variance (MANOVA), controlling for age and gender. Significant multivariate statistics were followed up through examination of univariate *F* tests for each domain and were pursued by independent *t* tests to determine order effects in terms of severity of daytime impact. Comparisons were 2-sided, with $P < .05$ considered to indicate statistical significance. When appropriate, to control for multiple comparisons within and between subjects, a per family error rate was adopted (.05/*n* of comparisons). Relative between-group effect sizes, expressed as Cohen *d* ($M_1 - M_2 / \delta_{pooled}$),[27] were applied to estimate and to compare the magnitude of observed effects.

RESULTS

Characteristics of Participants With Versus Without Insomnia Disorder

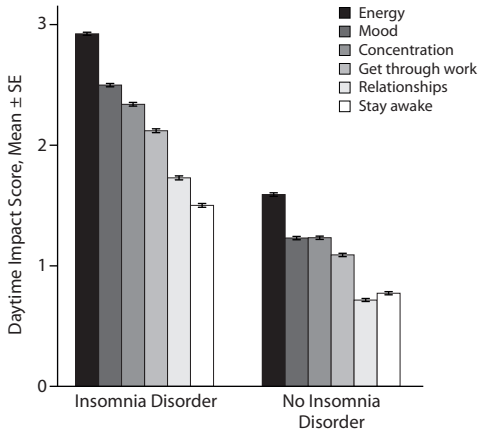
A total of 11,129 people (8,044 [72.3%] female; mean age = 39 years; range, 18–93 years) completed the survey (March 2010–April 2011). With the application of *DSM-5* criteria, 5,083 participants (45.7%) screened as having possible ID (ID group), and 5,542 did not have ID (NO-ID group) (49.8%; Table 1). A small number met insomnia criteria with duration ≤ 3 months, reflecting acute insomnia (sleep disturbance duration < 1 month; n = 131, 1.2%) or subacute insomnia (sleep disturbance duration 1–3 months; n = 373, 3.4%). These individuals were excluded, and thus the total number included in the analysis was 10,625. For those with ID, their sleep problem was typically chronic, with 83% having had insomnia for over 1 year and 45% having had it for ≥ 6 years.

A higher proportion of the ID group, relative to the NO-ID group, was female (75% vs 69%; $\chi^2 = 40.5$, $P < .0001$). The ID group was also older ($t_{10413.30} = 13.44$, $P < .0001$) and had poorer mental health ($t_{10313.16} = 26.14$, $P < .0001$) and physical health ($t_{10623} = 16.15$, $P < .0001$). We determined index of multiple deprivation (IMD) scores, a proxy for socioeconomic status, through residential postcodes (IMD analysis included English residents only). As the majority of our respondents were from England (n = 8,235), we compared IMD scores between those with insomnia (n = 3,991) and those without insomnia (n = 4,244), finding no significant difference ($t_{8233} = 0.724$, $P = .469$). Mean IMD values for our available sample (20.2, SD = 14.4) were similar to the national average (21.7, SD = 15.5).[28] Confirmation of sleep status allocations may be drawn from the finding that SCI scores for the NO-ID group were significantly higher ($t_{9862.89} = 130.46$, $P < .0001$), more than twice those of the ID group (see Table 1).

Daytime Impact of Insomnia Disorder Relative to No Insomnia Disorder

The ID and NO-ID groups were compared across the 6 *DSM-5* domains of daytime functioning. Since, by definition, the NO-ID group did not experience poor sleep on a regular basis, questions for these participants reflected the level of daytime impairment they experienced on their (occasional) nights of poor sleep. Formal analysis, with gender and age as covariates, produced an omnibus multivariate effect ($F_{6,10616} = 1035.04$, $P < .0001$) and significant univariate effects for all domains (all $P < .0001$), with the ID group reporting greater impairment (Figure 1). Relative between-group effect sizes were strongest for energy ($d = 1.30$), mood ($d = 1.24$), and concentration ($d = 1.05$) and remained large for relationships ($d = 0.96$) and getting through work ($d = 0.94$). Ability to stay awake during the day produced a smaller, though still moderate to large, between-group effect size ($d = 0.68$). This relative ordering of mean daytime impact for the ID group was paralleled by the proportions of participants who reported being "very much affected" on energy (30.7% of the ID

Figure 1. Comparison of Respondents With and Without Insomnia Disorder in Relation to Negative Impact on the 6 *DSM-5* Areas of Daytime Dysfunction[a]



[a]Higher scores reflect greater negative impact.

sample), mood (18.9%), concentration (16.9%), getting through work (14.6%), relationships (8.7%), and ability to stay awake (7.2%).

Daytime Impact Across Insomnia Disorder Subtypes

The most common subtype was a combination of the 3 core symptoms (MIXED; 61.3%), followed by difficulty maintaining sleep (DMS; 12.7%), difficulty initiating sleep (DIS; 12.4%), nonrestorative sleep (NRS; 9.5%), and early morning awakening (EMA; 4.2%) (Table 2). The MIXED group comprised the following symptom combinations: DMS + EMA (9.2% of total ID group), DIS + DMS (24.4%), DIS + EMA (6.7%), and DIS + DMS + EMA (21.0%). Subtype differences were found for gender distribution ($\chi^2 = 16.23$, $P < .01$), age ($F_{4,5078} = 87.35$, $P < .0001$), and, controlling for age and gender, self-reported mental health ($F_{4,5076} = 10.15$, $P < .0001$) and physical health ($F_{4,5076} = 7.43$, $P < .0001$). For mental health, the MIXED group was more impaired relative to the DMS group, and for physical health, the DMS group was more impaired relative to the EMA, MIXED, and NRS groups. There were also subtype differences with respect to the SCI ($F_{4,5076} = 525.9$, $P < .0001$) and prescribed sleeping pill use ($\chi^2 = 62.63$, $P < .0001$), with the MIXED group reporting the poorest overall sleep quality and being most likely to be taking prescribed sleeping pills.

In relation to the nature and magnitude of daytime impairment, formal analysis, with gender and age as covariates, produced an omnibus multivariate effect ($F_{24,17691.82} = 4.61$, $P < .0001$), and subsequent univariate effects were found for all 6 domains ($P < .05$ for staying awake and $P < .0001$ for the other 5 domains). When the Bonferroni comparison method for multiple testing was applied, those with the MIXED subtype reported greater daytime impairment relative to the other subtypes, particularly for mood, concentration, and getting through work (see Table 2). The magnitude of these differences was small (Cohen *d* range, 0.10–0.23). There were few subtype differences for problems staying awake, with the exception that the NRS group was significantly more impaired relative to the DIS group.

Daytime Impact of Insomnia Disorder Comorbid With Mental or Physical Health Problems or Additional Sleep-Related Disturbance

We investigated daytime impairment in relation to whether ID presented on its own (ID-Alone; n = 1,884, 37.1%) or with poor physical health (ID + PH; n = 166, 3.3%), poor mental health (ID + MH; n = 384, 7.6%), or another sleep disorder (ID + SLD; n = 1,691, 33.3%). Approximately 22% of participants (n = 1,138) reported ID in the presence of at least 2 (of the possible 3) comorbid presentations, reflected in the following groups: ID + PH + MH (n = 148, 2.9%), ID + MH + SLD (n = 510, 10%), ID + PH + SLD (n = 300, 5.9%). No participants reported having ID plus 3 comorbidities (ie, ID + MH + PH + SLD). For the purpose of the present analysis, we focused on daytime impairment across 4 discrete groups, ID-Alone, ID + PH, ID + MH, and ID + SLD, reflecting *DSM-5* proposals for recording possible comorbidities.

MANOVA revealed a significant main effect of comorbidity group ($F_{18,11636.63} = 18.9$, $P < .0001$), and univariate effects were found for all daytime domains ($P < .0001$; Table 3). Respondents with ID + MH were found to have greater sleep-related daytime impairment in relation to mood, concentration, and getting through work relative to every other group and reported enhanced levels of impairment for relationship functioning compared to those in the ID-Alone and ID + SLD groups. With respect to energy, the ID + MH, ID + PH, and ID + SLD groups all tended to have greater impairments relative to the ID-Alone group, and, with respect to ability to stay awake, the ID + SLD group exhibited the greatest impairment relative to the other 3 groups. The ID + MH group also had the poorest SCI score and reported greater usage of sleeping pills. Effect sizes for group differences, where the ID + MH group evidenced the greatest impairment, ranged from $d = 0.15–0.65$, with larger effects typically reflecting comparisons with the ID-Alone group.

Daytime Impact of Childhood-Onset Versus Adult-Onset Insomnia Disorder

Finally, the GBSS included the simple question, "Did you sleep well as a child?" (yes/no) to estimate idiopathic insomnia (consistent with criteria used in the *International Classification of Sleep Disorders*, second edition[29]). This allowed us to investigate the daytime impact of childhood-onset ID (n = 1,230; 24.2%) relative to adult-onset ID (n = 3,853; 75.8%, Table 4). Those who slept poorly during childhood were more likely to be younger ($t_{2193.34} = 10.97$, $P < .0001$) and female ($\chi^2 = 12.52$, $P < .0001$) and have poorer sleep quality (SCI: $t_{5081} = 5.82$, $P < .0001$). In addition, those reporting childhood onset had poorer mental health ($F_{1,5079} = 29.53$, $P < .0001$) and were more likely to be taking sleep-promoting hypnotics ($\chi^2 = 4.98$, $P < .05$) and over-the-counter remedies ($\chi^2 = 7.21$, $P < .01$). MANOVA revealed a significant main effect of group ($F_{6,5074} = 2.41$, $P < .05$). Significant univariate effects were found for 4 domains: energy ($P < .05$), mood,

Table 2. Comparison of Insomnia Disorder Subtypes in Relation to Demographic Variables and Sleep-Related Daytime Impairment

Variable	Difficulty Initiating Sleep (n = 632; 12.4%)	Difficulty Maintaining Sleep (n = 644; 12.7%)	MIXED[a] (n = 3,114; 61.3%)	Early Morning Awakening (n = 212; 4.2%)	Nonrestorative Sleep (n = 481; 9.5%)	Significant Contrasts With Bonferroni Correction
Gender, % male/female	73.4/26.6	73.8/26.2	76.4/23.6	71.2/28.2	68.6/31.4	
Age, mean (SD), y	32.3 (12.4)	46.2 (12.6)	42.4 (15.2)	40.9 (14.5)	39.7 (12.9)	
SCI score, mean (SE)[b]	3.60 (0.04)	3.38 (0.04)	2.53 (0.02)	4.4 (0.07)	4.4 (0.05)	
Physical health score, mean (SE)[c]	1.71 (0.04)	1.57 (0.04)	1.73 (0.02)	1.91 (0.07)	1.81 (0.04)	DMS > MIXED, EMA, NRS
Mental health score, mean (SE)[c]	1.75 (0.04)	1.61 (0.04)	1.88 (0.02)	1.82 (0.07)	1.77 (0.05)	MIXED > DMS
Domain of daytime functioning score, adjusted mean (SE)						
Mood	2.38 (0.04)	2.40 (0.04)	2.58 (0.02)	2.35 (0.07)	2.30 (0.05)	MIXED > DIS, DMS, EMA, NRS
Energy	2.83 (0.04)	2.85 (0.04)	2.97 (0.02)	2.90 (0.06)	2.88 (0.04)	MIXED > DIS, DMS
Relationships	1.57 (0.05)	1.72 (0.05)	1.79 (0.02)	1.63 (0.08)	1.57 (0.05)	MIXED > DIS, NRS
Staying awake	1.38 (0.05)	1.49 (0.05)	1.51 (0.02)	1.48 (0.08)	1.62 (0.05)	NRS > DIS
Concentration	2.26 (0.04)	2.29 (0.04)	2.40 (0.02)	2.15 (0.07)	2.19 (0.05)	MIXED > DIS, EMA, NRS
Getting through work	2.07 (0.05)	2.02 (0.05)	2.18 (0.02)	1.94 (0.08)	2.00 (0.05)	MIXED > DMS, EMA, NRS
Taking prescribed sleeping pills, %	8.2	9.0	14.8	5.2	6.0	

[a]Respondent had more than 1 of the core symptoms (difficulty initiating sleep, difficulty maintaining sleep, early morning awakening).
[b]Scale of 0 to 10; higher values reflect better overall sleep quality.
[c]Lower scores indicate better perceived health: 0 = very good, 4 = very poor.
Abbreviations: DIS = difficulty initiating sleep, DMS = difficulty maintaining sleep, EMA = early morning awakening, NRS = nonrestorative sleep, SCI = Sleep Condition Indicator.

Table 3. Comparison of Insomnia Disorder (ID) Presentations in Relation to Demographic Variables and Sleep-Related Daytime Impairment

Variable	ID + Mental Health Condition (n = 384)	ID + Other Sleep Disorder (n = 1,691)	ID + Physical Health Condition (n = 166)	ID Alone (n = 1,884)	Significant Contrasts With Bonferroni Correction
Gender, % male/female	23.4/76.6	29.4/70.6	18.1/81.9	22.7/77.3	
Age, mean (SD), y	37.4 (13.7)	40.6 (15.1)	41.9 (15.7)	44.2 (14.3)	
SCI score[a]					
Mean (SD)	2.75 (1.28)	3.07 (1.28)	3.05 (1.24)	3.22 (1.18)	
Mean (SE)	2.72 (0.06)	3.05 (0.03)	3.06 (1.0)	3.24 (0.03)	
Domain of daytime functioning score, adjusted mean (SE)					
Mood	2.86 (0.05)	2.46 (0.02)	2.31 (0.08)	2.30 (0.02)	ID + MH > ID + SLD, ID + PH, ID ID + SLD > ID
Energy	3.02 (0.05)	2.95 (0.02)	3.02 (0.07)	2.72 (0.02)	ID + MH, ID + PH, ID + SLD > ID
Relationships	1.92 (0.06)	1.73 (0.03)	1.75 (0.09)	1.55 (0.03)	ID + MH > ID + SLD, ID ID + SLD > ID
Staying awake	1.36 (0.06)	1.69 (0.03)	1.38 (0.09)	1.17 (0.03)	ID + SLD > ID + MH, ID + PH, ID ID + MH > ID
Concentration	2.57 (0.05)	2.34 (0.03)	2.20 (0.08)	2.12 (0.02)	ID + MH > ID + SLD, ID + PH, ID ID + SLD > ID
Getting through work	2.33 (0.06)	2.14 (0.03)	1.97 (0.09)	1.88 (0.03)	ID + MH > ID + SLD, ID + PH, ID ID + SLD > ID, ID + PH
Taking prescribed sleeping pills, %	18.0	10.6	10.8	10.9	

[a]Scale of 0 to 10; higher values reflect better overall sleep quality.
Abbreviations: MH = mental health condition, PH = physical health condition, SCI = Sleep Condition Indicator, SLD = sleep disorder.

concentration, and getting through work (all $P < .01$), with the childhood-onset group evidencing greater impairment. Between-group effects were small (range of *d* values, 0.12–0.24).

DISCUSSION

Our objective was to understand how daytime symptom patterns associate with nighttime sleep in *DSM-5* insomnia disorder.

First, our findings validate the *DSM-5* domains because the patterning of impact mirrored that reported by people in the group without ID in relation to the (infrequent) nights when the latter group does not sleep well (Figure 1). Between-group effect sizes for this comparison were large for energy, mood, concentration, relationships, and work functioning (range of *d* values, 0.94–1.30), with daytime sleepiness the least affected area, though still evidencing a moderate effect ($d = 0.68$). These results are consistent with other research suggesting that impairment of energy, mood, and cognition is characteristic of insomnia,[30–32] whereas daytime sleepiness, while elevated relative to normal sleepers, is less so.[33,34]

Second, we observed differences in symptomatology associated with the presenting subtype. The group with multiple core symptoms exhibited the greatest impairment,

Table 4. Comparison of Childhood- Versus Adulthood-Onset Insomnia in Relation to Demographic Variables and Sleep-Related Daytime Impairment

Variable	Sleep Problems in Childhood (n = 1,230; 24.2%)	No Sleep Problems in Childhood (n = 3,853; 75.8%)
Gender, % male/female	21.5/78.5**	26.5/73.5
Age, mean (SD), y	37.3 (13.9)**	42.6 (14.8)
Physical health score, mean (SE)[a]	1.76 (0.03)	1.71 (0.02)
Mental health score, mean (SE)[a]	1.96 (0.03)**	1.77 (0.02)
SCI score, mean (SE)[b]	2.82 (0.04)**	3.09 (0.02)
Taking prescribed sleeping pills, %	13.8*	11.4
Taking over-the-counter sleep remedies, %	27.2**	23.4
Domain of daytime functioning score, adjusted mean (SE)		
Mood	2.57 (0.03)**	2.48 (0.02)
Energy	2.98 (0.03)*	2.91 (0.02)
Relationships	1.78 (0.03)	1.71 (0.02)
Staying awake	1.52 (0.03)	1.50 (0.02)
Concentration	2.42 (0.03)**	2.31 (0.02)
Getting through work	2.20 (0.03)**	2.10 (0.02)

[a]Lower scores indicate better perceived health: 0 = very good, 4 = very poor.
[b]Scale of 0 to 10; higher values reflect better overall sleep quality.
*$P < .05$, **$P < .01$ for group comparisons.
Abbreviation: SCI = Sleep Condition Indicator.

most pronounced for mood. In the group with multiple symptoms, concentration was also more impaired relative to the groups with difficulty initiating sleep, early morning awakening, and nonrestorative sleep, and getting through work was more impaired relative to the groups with difficulty maintaining sleep, early morning awakening, and nonrestorative sleep. These results were statistically robust, though small in magnitude and broadly comparable to those of Léger et al.[15] Our data also indicate that nonrestorative sleep impacts functioning to a similar degree as difficulty initiating sleep, difficulty maintaining sleep, and early morning awakening, in keeping with emerging epidemiologic and experimental literature.[16,17,35]

Third, investigation of comorbid conditions revealed that in 3 domains (concentration, mood, and getting though work) the group with ID and a mental health condition reported the greatest impairment, and this group was 70% more likely to be taking sleep-promoting hypnotics. Unsurprisingly, those with ID and another sleep disorder had the greatest difficulty with maintaining wakefulness. Participants who had ID alone tended to be the least impaired, reflecting research on health-related quality of life showing that controlling for comorbidities slightly attenuates the next-day impact of poor sleep.[14] Nevertheless, robust between-group effects were evident even when we compared the group with ID alone and the group without ID (n = 1,884 vs n = 5,542, supplementary analysis: large effects for all domains [range of *d* values, 0.72–1.09] except sleepiness [$d = 0.38$]).

Finally, we found that those who had slept poorly as children (one-quarter of the ID group) had poorer sleep and mental health and greater deficits in energy, mood, concentration, and ability to get through their work. Sánchez-Ortuño et al[22] reported that such idiopathic insomnia and insomnia with mental health conditions clustered together and were associated with mood disturbance. Perhaps the link between childhood-onset insomnia and affective impairment supports an underlying, possibly genetic, vulnerability to both sleep disturbance and depression.[37]

The clinical importance of daytime concerns as an integral component of ID should not be underestimated. We found substantial daytime effects for people with all insomnia subtypes when compared with normal sleepers. It seems appropriate then to emphasize that *DSM-5* ID can be characterized by poorer sleep *and* poorer daytime well-being. It seems likely that the combination of these experiences will drive clinical complaint.[37] This interaction requires greater attention in practice, if we are to properly treat ID.[14,38] Our data suggest that this clinical attention may be particularly important for patients presenting a mixed subtype of insomnia, those with co-occurring poor mental health, and those with an early history of sleep disturbance. A therapeutic focus on how to cope with and minimize daytime symptoms could enhance "traditional" cognitive-behavioral therapy for insomnia.[39]

The strength of this study lies in the application of *DSM-5* criteria to a sizable population. We did not have a validation sample, using gold-standard clinical interviews, and our survey method is likely to have introduced error. Our screening of physical and mental health and our definition of childhood insomnia were self-reported and based on single items, so we urge caution in interpreting the results. Further work is required using face-to-face clinical evaluation and/or more comprehensive self-report methodology to improve the characterization of respondents' mental and physical health status. Our approach to data analysis intentionally reflected a categorical/diagnostic view of ID, typical of disease classification nosologies and of clinical practice. Real-world evaluations of this kind are essential to consider the *DSM-5* ID criteria. Nevertheless, we recognize that objective validation studies on nighttime and daytime symptoms are important. A recent report showed that slow-wave sleep was reduced in those with multiple core symptoms and those with difficulty maintaining sleep,[16] so it is possible that slow-wave sleep has a mediating effect on daytime performance.[40] Thus, there is a clear need for further work to characterize interactions among objectively determined sleep parameters, health status (including medication influences), and daytime functioning. Multivariate modeling would also help elucidate associations between sleep and daytime well-being and possible intermediate variables. One example would be that the impact of hypnotics on daytime functioning is well known,[41] and in our study "higher risk" groups were more likely to endorse taking prescription sleep medication. We cannot, therefore, exclude the possibility that greater levels

of daytime symptoms were in part an artifact of such other factors.

Finally, we should also mention that, when the study was conducted, we included the 6 daytime domains that were listed on the *DSM-5* Web site at that time (ie, the version dated June 2, 2010). We acknowledge that since then there have been some changes, including the addition of a domain relating to behavioral problems (eg, hyperactivity, impulsivity, aggression). This latter domain, however, may refer more to sleep problems in children and young people rather than adults.

Author affiliations: University of Glasgow Sleep Centre and Institute of Neuroscience & Psychology, University of Glasgow (Drs Espie and Kyle), and MRC Social and Public Health Sciences Unit (Dr Benzeval), Glasgow, Scotland; and Sleepio Limited (Dr Espie and Mr Hames) and Mental Health Foundation (Dr Cyhlarova), London, England.
Potential conflicts of interest: **Dr Espie** is Clinical and Scientific Director of Sleepio Ltd and developed the Web platform for the Great British Sleep Survey, but has not received income from Sleepio Ltd. **Mr Hames** is Managing Director and CEO of Sleepio Ltd and has received a salary from the company. **Drs Kyle**, **Cyhlarova**, and **Benzeval** report no potential conflict of interest.
Funding/support: No direct funding was provided for this research.

REFERENCES

1. NIH State-of-the-Science Conference Statement on Manifestations and Management of Chronic Insomnia in Adults. *NIH Consens Sci Statements*. 2005;22(2):1–30.
2. American Psychiatric Association. M 00 Insomnia Disorder. *DSM-5* Development Web site. http://www.dsm5.org/ProposedRevision/Pages/proposedrevision.aspx?rid=65#. Updated June 2, 2010.
3. American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders*, Fourth Edition. Washington, DC: American Psychiatric Association; 1994.
4. Reynolds CF 3rd, Redline S; *DSM-V* Sleep-Wake Disorders Work Group and Advisors. The *DSM-V* sleep-wake disorders nosology: an update and an invitation to the sleep community. *J Clin Sleep Med*. 2010;6(1):9–10.
5. Baglioni C, Battagliese G, Feige B, et al. Insomnia as a predictor of depression: a meta-analytic evaluation of longitudinal epidemiological studies. *J Affect Disord*. 2011;135(1–3):10–19.
6. Fava M, McCall WV, Krystal A, et al. Eszopiclone co-administered with fluoxetine in patients with insomnia coexisting with major depressive disorder. *Biol Psychiatry*. 2006;59(11):1052–1060.
7. Manber R, Edinger JD, Gress JL, et al. Cognitive behavioral therapy for insomnia enhances depression outcome in patients with comorbid major depressive disorder and insomnia. *Sleep*. 2008;31(4):489–495.
8. Vgontzas AN, Liao D, Bixler EO, et al. Insomnia with objective short sleep duration is associated with a high risk for hypertension. *Sleep*. 2009;32(4):491–497.
9. Vgontzas AN, Liao D, Pejovic S, et al. Insomnia with objective short sleep duration is associated with type 2 diabetes: a population-based study. *Diabetes Care*. 2009;32(11):1980–1985.
10. Vgontzas AN, Liao D, Pejovic S, et al. Insomnia with short sleep duration and mortality: the Penn State cohort. *Sleep*. 2010;33(9):1159–1164.
11. Benetó A, Gomez-Siurana E, Rubio-Sanchez P. Comorbidity between sleep apnea and insomnia. *Sleep Med Rev*. 2009;13(4):287–293.
12. Bjornsdottir E, Janson C, Gislason T, et al. Insomnia in untreated sleep apnea patients compared to controls. *J Sleep Res*. 2011;21(2):131–138.
13. Edinger JD, Bonnet MH, Bootzin RR, et al; American Academy of Sleep Medicine Work Group. Derivation of research diagnostic criteria for insomnia: report of an American Academy of Sleep Medicine Work Group. *Sleep*. 2004;27(8):1567–1596.
14. Kyle SD, Morgan K, Espie CA. Insomnia and health-related quality of life. *Sleep Med Rev*. 2010;14(1):69–82.
15. Léger D, Partinen M, Hirshkowitz M, et al; EQUINOX (Evaluation of daytime QUality Impairment by Nocturnal awakenings in Outpatients eXperience) Survey Investigator Group. Daytime consequences of insomnia symptoms among outpatients in primary care practice: EQUINOX International Survey. *Sleep Med*. 2010;11(10):999–1009.
16. Roth T, Zammit G, Lankford A, et al. Nonrestorative sleep as a distinct component of insomnia. *Sleep*. 2010;33(4):449–458.
17. Walsh JK, Coulouvrat C, Hajak G, et al. Nighttime insomnia symptoms and perceived health in the America Insomnia Survey (AIS). *Sleep*. 2011;34(8):997–1011.
18. Rodin J, McAvay G, Timko C. A longitudinal study of depressed mood and sleep disturbances in elderly adults. *J Gerontol*. 1988;43(2):45–53.
19. Yokoyama E, Kaneita Y, Saito Y, et al. Association between depression and insomnia subtypes: a longitudinal study on the elderly in Japan. *Sleep*. 2010;33(12):1693–1702.
20. Philip P, Guilleminault C. Adult psychophysiologic insomnia and positive history of childhood insomnia. *Sleep*. 1996;19(suppl):S16–S22.
21. Edinger JD, Stout AL, Hoelscher TJ. Cluster analysis of insomniacs' MMPI profiles: relation of subtypes to sleep history and treatment outcome. *Psychosom Med*. 1988;50(1):77–87.
22. Sánchez-Ortuño MM, Edinger JD, Wyatt JK. Daytime symptom patterns in insomnia sufferers: is there evidence for subtyping insomnia? *J Sleep Res*. 2011;20(3):425–433.
23. Lichstein KL, Durrence HH, Taylor DJ, et al. Quantitative criteria for insomnia. *Behav Res Ther*. 2003;41(4):427–445.
24. Wilson SJ, Nutt DJ, Alford C, et al. British Association for Psychopharmacology Consensus Statement on Evidence-Based Treatment of Insomnia, Parasomnias and Circadian Rhythm Disorders. *J Psychopharmacol*. 2010;24(11):1577–1601.
25. Espie CA, Kyle SD, Williams C, et al. A randomized, placebo-controlled trial of online cognitive behavioral therapy for chronic insomnia disorder delivered via an automated media-rich web application. *Sleep*. 2012;35(6):769–781.
26. Gardani M, Miller CB, Biello S, et al. The association of sleepiness and diurnal preference with salivary amylase activity [abstract]. Abstracts of the 4th International Congress of the World Association of Sleep Medicine and 5th Conference of the Canadian Sleep Society. *Sleep Med*. 2011;12(suppl 1):S1–S130.
27. Cohen J. *Statistical Power Analysis for the Behavioral Sciences*. 2nd ed. Hillsdale, NJ: Erlbaum; 1988.
28. Department for Communities and Local Government. The English Indices of Deprivation 2007. Office for National Statistics. http://www.communities.gov.uk/documents/communities/pdf/733520.pdf. Updated March 2008. Accessed August 29, 2012.
29. American Academy of Sleep Medicine. *International Classification of Sleep Disorders: Diagnostic and Coding Manual*. 2nd ed. Westchester, IL: American Academy of Sleep Medicine; 2005.
30. Fortier-Brochu E, Beaulieu-Bonneau S, Ivers H, et al. Insomnia and daytime cognitive performance: a meta-analysis. *Sleep Med Rev*. 2012;16(1):83–94.
31. Moul DE, Nofzinger EA, Pilkonis PA, et al. Symptom reports in severe chronic insomnia. *Sleep*. 2002;25(5):553–563.
32. Buysse DJ, Thompson W, Scott J, et al. Daytime symptoms in primary insomnia: a prospective analysis using ecological momentary assessment. *Sleep Med*. 2007;8(3):198–208.
33. Riedel BW, Lichstein KL. Insomnia and daytime functioning. *Sleep Med Rev*. 2000;4(3):277–298.
34. Roehrs TA, Randall S, Harris E, et al. MSLT in primary insomnia: stability and relation to nocturnal sleep. *Sleep*. 2011;34(12):1647–1652.
35. Ohayon MM. Prevalence and correlates of nonrestorative sleep complaints. *Arch Intern Med*. 2005;165(1):35–41.
36. Gregory AM, Rijsdijk FV, Lau JYF, et al. The direction of longitudinal associations between sleep problems and depression symptoms: a study of twins aged 8 and 10 years. *Sleep*. 2009;32(2):189–199.
37. Morin CM, LeBlanc M, Daley M, et al. Epidemiology of insomnia: prevalence, self-help treatments, consultations, and determinants of help-seeking behaviors. *Sleep Med*. 2006;7(2):123–130.
38. Kyle SD, Espie CA, Morgan K. "...Not just a minor thing, it is something major, which stops you from functioning daily": quality of life and daytime functioning in insomnia. *Behav Sleep Med*. 2010;8(3):123–140.
39. Harvey AG, Sharpley AL, Ree MJ, et al. An open trial of cognitive therapy for chronic insomnia. *Behav Res Ther*. 2007;45(10):2491–2501.
40. Van Someren EJ, Van Der Werf YD, Roelfsema PR, et al. Slow brain oscillations of sleep, resting state, and vigilance. *Prog Brain Res*. 2011;193:3–15.
41. Vermeeren A, Coenen AM. Effects of the use of hypnotics on cognition. *Prog Brain Res*. 2011;190:89–103.

Cognitive behavioural treatment of insomnia

Behaviour Research and Therapy 2001, 39, 45-60

Journal of Consulting & Clinical Psychology 2001, 69, 58-66

Behaviour Research and Therapy 2002, 40, 75-83

Behavioural and Cognitive Psychotherapy 2003, 31, 313-324

SLEEP 2006, 29, 1398-1414

SLEEP 2007, 30, 574-584

Journal of Clinical Oncology 2008, 26, 4651-4658

SLEEP 2009, 32 , 1549-1558

Behavioral Sleep Medicine 2009, 7, 99-105

Journal of Psychopharmacology 2010, 24, 1577–1600

SLEEP 2012, 35, 769-781

Journal of Consulting & Clinical Psychology 2012, in press

PERGAMON

Behaviour Research and Therapy 39 (2001) 45–60

BEHAVIOUR RESEARCH AND THERAPY

www.elsevier.com/locate/brat

The clinical effectiveness of cognitive behaviour therapy for chronic insomnia: implementation and evaluation of a *sleep clinic* in general medical practice

Colin A. Espie *, Stephanie J. Inglis, Susan Tessier, Linda Harvey

Department of Psychological Medicine, University of Glasgow, Glasgow, UK

Received 23 August 1999

Abstract

Chronic insomnia is a very common clinical condition which may respond well to non-pharmacological treatment. Indeed, the literature supports the efficacy of cognitive behaviour therapy (CBT). However, there has been no substantial study of clinical effectiveness. Since insomniacs typically present in general medical practice this is a crucial gap in the outcome research. This study, therefore, specifically investigated the clinical effectiveness of CBT delivered by Health Visitors (primary care nurses) trained as therapists. One hundred and thirty-nine insomniacs (mean age 51 yr) were randomised to CBT or Self-Monitoring Control (SMC) in a controlled trial. CBT comprised six group sessions (n=4 to 6 patients). After the controlled phase, SMC patients entered deferred treatment (CBT-DEF), allowing both treatment replication and long-term outcome to be investigated for a sizeable, treated sample. Repeated measures ANOVAs demonstrated superiority of CBT over SMC in substantially reducing sleep latency and wakefulness during the night. CBT-DEF replicated similar effects and maintained improvement was observed in both groups one year later. Furthermore, total sleep increased significantly during follow-up and 84% of patients initially using hypnotics remained drug-free. Results suggest that CBT administered by Health Visitors offers a clinically effective treatment for insomnia.

Keywords: Insomnia; Cognitive behaviour therapy; Clinical effectiveness

* Corresponding author. Tel.: +44-141-211-3903; fax: +44-141-357-4899.
E-mail address: c.espie@clinmed.gla.ac.uk (C.A. Espie).

0005-7967/00/$ - see front matter
PII: S0005-7967(99)00157-6

1. Introduction

Conservative estimates of the prevalence of chronic insomnia, defined as persistent difficulty in initiating or maintaining sleep (American Sleep Disorders Association, 1997), range from 9 to 12% in adulthood and up to 20% in later life, making sleep disturbance one of the most common complaints in general medical practice (Bixler, Kales, Soldatos, Kales & Healy, 1979; Foley et al., 1995; Ford & Kamerow, 1989; Gallup Organisation, 1991; Hoch et al., 1997; Mellinger, Balter & Uhlenhuth, 1985). One reason for this high prevalence is that sleep is very sensitive to physical or psychological disorder. Thus, "secondary insomnia" presents in a wide range of conditions (e.g. pain, respiratory disorder, depression), often enduring once a disordered pattern has been established, even after the medical or psychiatric problem has resolved. "Primary insomnia", of course, refers to a sleep disorder constituting the major difficulty at the time of presentation. Age-related developmental change contributes to the increased presentation rates from the middle years onwards (see Espie, 1991; Morin, 1993 for review). Statistics on the prescription of hypnotic medications (particularly benzodiazapines) also demonstrate the scale of sleep complaint within the general population (Gallup Organisation, 1991; Mellinger et al., 1985) and insomnia is now recognised to be associated with significant health-economic cost (Chilcott & Shapiro, 1996; Walsh, Engelhardt & Hartman, 1995).

Although benzodiazapines used for short periods of time, or intermittently, can maintain their effectiveness (Kales & Kales, 1984), pharmacological treatment is not recommended for primary or chronic insomnia, or in the elderly. Indeed, the evidence against the prolonged use of such drugs is overwhelming (NIMH, 1984; Russell & Lader, 1992). Over the past 20 yr considerable progress has been made in the psychological assessment and treatment of sleep problems (Bootzin & Nicassio, 1978; Espie, 1991, 1993; Lacks, 1987; Morin, 1993).

There is a sizeable literature on the effectiveness of cognitive behavioural treatment (CBT) for insomnia. Accurate sleep information, the practice of sleep hygiene and the use of specific behavioural and cognitive techniques such as relaxation therapy (see Bootzin & Nicassio, 1978), stimulus control (Bootzin, 1972; Bootzin & Rider, 1997), sleep restriction (Spielman, Saskin & Thorpy, 1983) and cognitive techniques (Espie & Lindsay 1985; Morin, 1993) has been widely endorsed. Over 50 controlled studies support the *clinical efficacy* of CBT, with meta-analyses reporting significant effect sizes for improvements in sleep latency (0.87), duration of wakenings (0.65) and sleep quality (0.94) (Morin, Culbert & Schwartz, 1994; Murtagh & Greenwood, 1995). The majority of studies has been North American, although European trials have reported similar results (Espie, Lindsay, Brooks, Hood & Turvey, 1989; Sanavio, Vidotto, Bettinardi, Rolletto & Zorzi, 1990). Around 70–80% of patients seem to benefit and CBT appears equally efficacious in older adults (Morin, Colecchi, Stone et al., 1999; Morin, Kowatch, Barry & Walton, 1993; Morin et al., in press); important because of clear contraindications for hypnotic use in this agegroup (Lader, 1992).

However, despite this evidence and the fact that CBT could be regarded as the treatment of choice for chronic insomnia (Espie, 1999; Morin et al., 1999, in press), impact upon general health care practice so far has been limited. Of course, the scale of the implementation task is such that any realistic alternative to sleeping pills must be capable of being implemented not only effectively but simply, efficiently and locally. The task is also compounded by the likelihood that few general medical practitioners will be aware of the psychological literature; the fact that few

psychologists have a special interest in sleep and in any case, may be in short supply; and the continuing tendency for the doctor-patient relationship to raise the patient's expectations of drugs. All of these factors are essentially *practical* and might be alleviated by greater dissemination of knowledge, educational opportunity for medical students and doctors, increased availability of psychological services and changing the culture surrounding 'medical' intervention.

A more fundamental factor, however, is that CBT may be time-consuming, expensive and ultimately less therapeutic to implement in general practice. There is, therefore, also an important *empirical* question around the feasibility and effectiveness of delivering CBT which has not been answered in efficacy trials, these having generally been carried out at specialised centres rather than in routine practice. Morin et al. (1994) and recently Edinger and Wohlgemuth (1999), have indicated that a major *scientific limitation* of the available evidence is the lack of any large scale appraisal of *clinical effectiveness* in ordinary clinical settings. This study, therefore, was designed specifically to investigate this research priority.

In the UK, most General Practitioners (GPs) work in group practices, often based in "health centres". The primary care teams have GPs, district nurses and health visitors as the core professionals providing community health services, with paramedical staff offering sessional input. The Health Visitor is a primary care nurse with community nursing qualifications who works across the life span, e.g. with infants during early development, with people rehabilitating from illness and with elderly people at home. In planning this study, therefore, we felt that training Health Visitors in CBT for insomnia would provide the most accessible and valid approach to managing insomnia in primary care settings, supported by a psychologist available on a consultancy basis for complex cases e.g. significant co-morbidity, or differential diagnosis. Where required, arrangements were also made for referral to a sleep laboratory. Although polysomnography (PSG) is not usually required for the diagnosis of insomnia (Reite, Buysse, Reynolds & Mendelson, 1995) it is invaluable in differential diagnosis of other suspected sleep problems.

2. Aims

The aims of the study, therefore, were to evaluate the medium to long-term (12 months post-treatment) clinical effectiveness of a primary care-based insomnia service (the *Sleep Clinic*), using Health Visitors as trained CBT therapists. In order to ensure validity and generalisability to typical clinical populations, the study required a large number of consecutively referred insomniacs whose sleep problems were both persistent and severe. The intervention was devised to test the feasibility of a cost efficient approach involving approximately 1 h of treatment time per case.

3. Methods

3.1. Subjects

Consecutive referrals of adults presenting with chronic insomnia during a 24 month period were received from GPs in Ayrshire. This part of west, central Scotland has both urban and rural communities and comprises a broad socioeconomic span. Selection, based upon International

Classification of Sleep Disorders criteria, comprised persistent difficulty initiating or maintaining sleep, occurring 4 or more nights per week, for at least 3 months (American Sleep Disorders Association, 1990). In addition, subjects had to score 5 or more on the Pittsburgh Sleep Quality Index, a cut-off point regarded as identifying significant sleep disturbance with 90% diagnostic accuracy. Furthermore, to ensure capture of the clinical population, subjects taking sleep medications were *not* excluded.

Two hundred and fifty-two subjects completed screening assessment of whom 91 failed to meet diagnostic criteria. Main exclusions were transient or subclinical insomnia (n=35), suspected Obstructive Sleep Apnoea Syndrome (OSAS; n=10), Periodic Limb Movements in Sleep (PLMS; n=6), Major Depression (n=22), Parasomnia (e.g. sleepwalking; n=8) and other diagnoses (n=10). OSAS and PLMS were subsequently confirmed by full PSG assessment in a sleep laboratory. Patients with significant depressive symptoms were referred to local services and those with parasomnias were managed on a case by case basis by the senior author, but independent of this study. Therefore, a total of 161 subjects was available of whom 139 (86%) completed the protocol to post-treatment. Twelve month follow-up data are available on 109 subjects (78% of those entering the study).

The final sample comprised 95 women and 44 men with mean age 51.4 years (S.D. 17.1; range 18-83). The majority was married and 60% were retired or ‘at home’. Over 40% reported problems of greater than 10 years duration and more than half took hypnotic sleep medication. Mean Epworth Sleepiness Scale score of 5.73 (S.D. 4.48) indicated minimal daytime sleep tendency. However, 90% rated themselves as feeling tired across the daytime. (Table 1). Participants completing the 12 month follow up assessment did not differ significantly from the complete cohort on any demographic or clinical variable.

3.2. Measures

Subjects completed the Pittsburgh Sleep Quality Index (Buysse, Reynolds, Monk, Berman & Kupfer, 1989) and two weeks of Sleep Diaries prior to interview, when a full history was taken. This was of standard format covering physical and mental health and diagnostic questions relating to the International Classification of Sleep Disorders (ICSD). The Beck Depression Inventory (BDI; Beck, Ward, Mendelson, Mock & Erbaugh, 1961), State-Trait Anxiety Inventory (STAI; Spielberger, Gorsuch & Lushene, 1970) and Penn State Worry Questionnaire (PSWQ; Meyer, Miller, Metzger & Borkovec, 1990) were administered to assess psychopathology. The Epworth Sleepiness Scale (Johns, 1991) was administered as a standard assessment of excessive daytime sleepiness; a central diagnostic symptom of OSAS and PLMS. To further improve diagnostic accuracy of suspected OSAS or PLMS, the bedpartner was interviewed where possible and actigraphic data were gathered over 5 nights using the Actiwatch® (Cambridge Neurotechnology Ltd.). The wrist actigraph is no larger than a standard wristwatch and is a simple, non-intrusive, objective measure which estimates sleep parameters based on body movement (Sadeh, Hauri, Kripke & Lavie, 1995). Motion sensors linked to a microprocessor detect and store data for downloading and analysis on PC software. Full overnight PSG assessment was conducted only to confirm diagnosis of other sleep disorders. It is widely accepted that PSG is not routinely required in the clinical evaluation of insomnia (Douglas, Thomas & Jan, 1992; Reite et al., 1995).

Sleep diaries were completed each morning upon wakening for a 2 week baseline, throughout

Table 1
Summary descriptive information on the study sample (n=139; unless otherwise stated)

Descriptor	Values	*n*	%
Gender	Female	95	68
	Male	44	32
Marital status	Married/cohabiting	86	62
	Single	18	13
	Widowed	23	17
	Divorced	12	8
Occupational status	Working	55	40
	At home	41	29
	Retired	43	31
History of sleep problem (n=131)	<2 yr	25	18
	>2 and <5 yr	27	20.5
	>5 and <10 yr	26	20
	>10 and <20 yr	26	20
	>20 yr	27	20.5
Use of sleep medication	not in past month	65	47
	<once per week	8	6
	once or twice per week	11	8
	three or more times per week	55	39
Ratings of daytime tiredness (n=105)	morning		
	never/occasionally	15	14
	often/most days	90	86
	afternoon		
	never/occasionally	11	10
	often/most days	94	90
	evening		
	never/occasionally	8	7
	often/most days	97	93

the 6 week treatment and for 1 week at follow-up. Diaries comprised information on bedtime and rising time, sleep onset latency (SOL), number of wakenings, wake time after sleep onset (WASO), duration of nighttime sleep i.e. total sleep time (TST), ratings of sleep quality and information on use of sleep medication (Espie, 1991). The Sleep Diary is the “staple” assessment tool for appraisal of insomnia and monitoring of treatment-related effects. Although self-report tends to overestimate sleep latency and wakefulness and underestimate time slept, studies consistently demonstrate significant correlation with objective measures using PSG or actigraphic data (Blood, Sack, Percy & Pen, 1997; Downey & Bonnet, 1992; Espie, 1991). Therefore, in outcome research on insomnia sleep diaries are able to reflect change over time with satisfactory accuracy. Importantly, the Sleep Diary, uniquely, captures subjective report and is the most valid instrument for studying insomnia in general practice.

3.3. Design

The study comprised an initial randomised treatment trial incorporating a treatment replication phase, followed by a longer term observational study. It was felt that this design would be particularly suited to the stated purpose of investigating clinical effectiveness in a large sample of patients to long-term follow-up.

After a minimum 2 week baseline, subjects were allocated at random either to CBT or a Self-Monitoring Control (SMC) condition, each of 6 week duration. The latter controlled for the non-specific effects of attention and of diary self-monitoring. It was hypothesised that CBT would demonstrate superiority in outcome compared with SMC and that SMC subjects would not improve. (Research has indicated consistently that chronic insomniacs do not improve when they are not actively treated, but tend to deteriorate (Mendelson, 1995; Morin et al., 1994; Murtagh & Greenwood, 1995)). SMC subjects then entered the treatment replication phase, receiving an identical treatment to the CBT group. SMC, therefore, became in effect a deferred CBT condition at this point (CBT-DEF). It was hypothesised that CBT-DEF subjects would achieve post-treatment outcomes comparable to the CBT condition. Upon completion of active treatment, both CBT and CBT-DEF groups were followed up at 12 months post-treatment. Sixty-five of the 139 subjects were randomised to SMC/ CBT-DEF and 74 to CBT, with 109 completing the follow-up assessment.

3.4. Intervention

CBT comprised a multicomponent package, based upon established principles for non-pharmacological treatment of insomnia. Subjects participated in group sessions (of 4 to 6 people) once per week, completing a six week intervention led by a Health Visitor in a local GP surgery or Health Centre. Each of the six treatment sessions was of 50 min duration; thus investment of Health Visitor time per patient averaged around 1 h. Six Health Visitors conducted the treatment sessions after extensive training from a Clinical Psychologist, a senior Health Promotion Officer and a Pharmacist. Training was conducted both by sessional teaching of the treatment components and through experiential learning by participating in ongoing groups (further details available upon request). The Health Visitors were also provided with consultancy advice from the project leader (a psychologist) and from the senior author.

Treatment comprised:

Session 1. Sleep Information (nature and function of sleep, sleep needs, disorders and effects),
Session 2. Sleep Hygiene Practices (diet, exercise and environment),
Sessions 3 and 4. Sleep Scheduling (stimulus control and sleep restriction procedures, relaxation therapy),
Sessions 5 and 6. Cognitive Therapy (thought restructuring, managing anxiety and intrusive thoughts).

The CBT intervention was didactic, following a standardised format with overhead projection of prepared material but with opportunity for discussion. Participants received written notes which collated to a procedural manual for home-based practice. Full details of these treatment procedures

are available elsewhere (Espie et al., 1998) and from the senior author. Patients who were on hypnotic medication received support in withdrawal as part of the programme and followed a graded reduction schedule negotiated to maximise individual compliance. However, the withdrawal rate was not greater than a one therapeutic dose reduction per week.

The SMC condition did not contain any specific, active treatment component. However, SMC subjects did receive a full assessment interview and completed Sleep Diaries on a night by night basis. They were told that they would start treatment in 6 weeks time and that continuing to record their sleep during the intervening period would yield valuable clinical information on their sleep pattern. If they required further help in completing the diaries they were encouraged to telephone the office for advice. SMC may be regarded, therefore, as a waiting list condition incorporating minimal contact, attention placebo and self-monitoring elements.

4. Results

4.1. Sleep pattern data

Data were analysed by means of SPSS repeated measures ANOVAs, appropriate for the mixed between-within subjects design of this study (Tabachnick & Fidell, 1996). Dependent variables were weekly mean scores for the three principal outcome measures from the Sleep Diary i.e. sleep-onset latency (SOL; min), wake time after sleep-onset (WASO; min) i.e. total time spent awake during night-time wakenings and total sleep time (TST; h). Each variable was considered in a separate ANOVA, with group as the between subjects factor comprising two levels (CBT or SMC/CBT-DEF) and time as the within subjects factor comprising four levels (baseline; post-intervention (i.e. post-CBT or post-SMC); post CBT-DEF (i.e. after the SMC group had been actively treated); 12 month follow up). Outcome data are presented visually in Figs. 1–3 for ease of comparison, with formal statistical analyses presented in Table 2.

Inspection of Fig. 1 reveals that mean SOL reduced from 61 to 28 min following CBT, com-

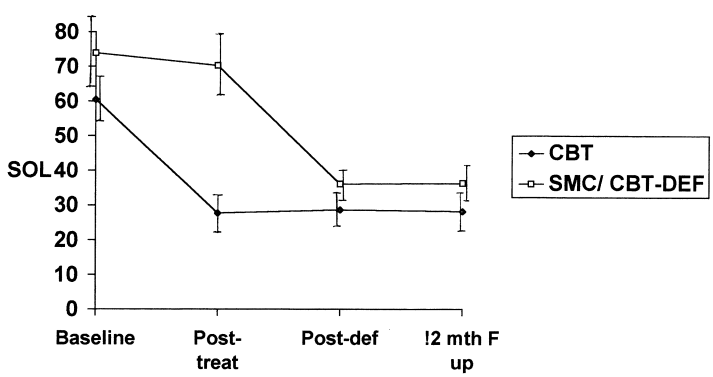


Fig. 1. Effects upon Sleep-onset latency (SOL; min) of CBT and Self-Monitoring Control (SMC) and of the latter group's deferred entry to treatment (CBT-DEF) across the experimental period.

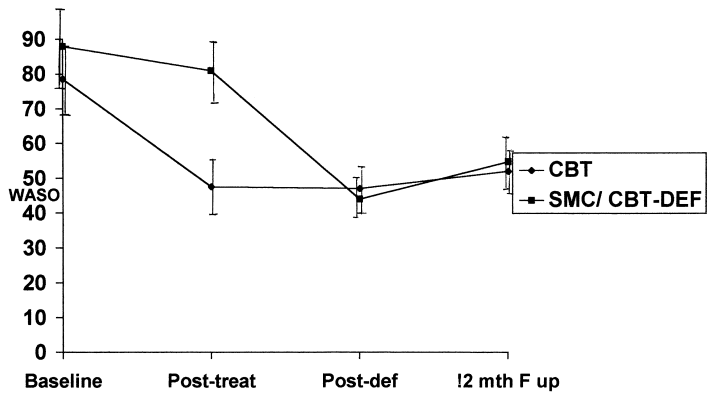


Fig. 2. Effects upon Wake time after sleep-onset (WASO; min) of CBT and Self-Monitoring Control (SMC) and of the latter group's deferred entry to treatment (CBT-DEF) across the experimental period.

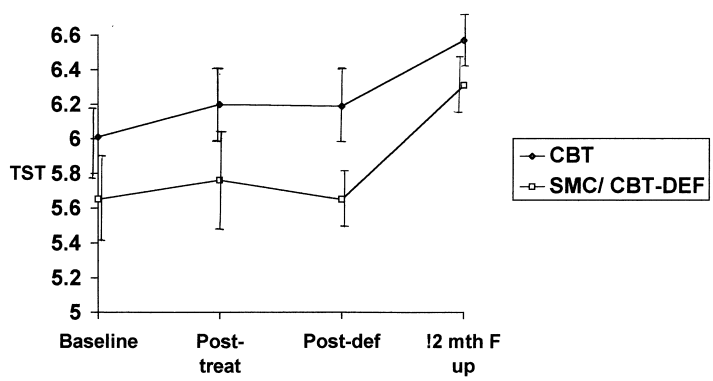


Fig. 3. Effects upon Total sleep time (TST; h) of CBT and Self-Monitoring Control (SMC) and of the latter group's deferred entry to treatment (CBT-DEF) across the experimental period.

pared with a change from 74 to 70 min associated with SMC. However, after active treatment, the latter group achieved a post-treatment SOL of 36 min. These descriptive data suggest, therefore, that CBT was an effective intervention as evidenced both by its initial superiority over SMC and by the replication of a similar outcome associated with deferred treatment. Furthermore, 12 month follow up data on 109 of the 139 participants, demonstrated stability in outcomes for both treated groups. These results are supported by formal analysis (Table 2). Highly significant main effects were observed for the effects of group, time and the group×time interaction (all $p<0.001$). Further investigation of the interaction term demonstrated that the only significant between group

Table 2
Repeated measures ANOVAs for each of the three principal outcome measures with significant main effects presented in bold typeface. Independent sample *t*-tests identify significant between group sub-effects across the four phases of the experimental design (*$p<0.05$; **$p<0.01$; ***$p<0.001$)

Variable	Effect	*F* ratio	Prob	Baseline t^{prob}	Post-treat (CBT/SMC) t^{prob}	Post-treat (CBT-DEF) t^{prob}	12 month follow-up t^{prob}
Sleep-onset latency (SOL; min)	group	11.52	**0.001**				
	time	43.55	<**0.001**				
	group×time	10.61	<**0.001**	1.54	5.29***	1.62	1.65
Wake time after sleep-onset (WASO; min)	group	1.03	0.312				
	time	20.38	<**0.001**				
	group×time	4.89	0.002	0.68	2.62**	-0.29	-0.23
Total sleep time (TST; h)	group	3.88	0.052				
	time	17.25	<**0.001**				
	group×time	0.78	0.504	-1.41	-1.76	-2.38*	-1.37

difference was at post-treatment when the CBT group had received active intervention but SMC had not yet embarked on deferred CBT (t=5.29; $p<0.001$). By comparison after the CBT-DEF phase the two conditions were no longer significantly different, but both were improved. The overall time main effect strongly supports the graphical evidence of maintained improvement to 12 months post-treatment for both groups.

Baseline data indicate that participants in both conditions also experienced significant wakefulness during the night (Fig. 2). CBT was associated with a mean reduction in WASO of around 31 min to 47 min at post-treatment compared with little change in the SMC group which remained at over 80 min. Deferred entry to treatment (CBT-DEF) did, however, reduce WASO to a mean of 44 min. As with SOL, post-treatment gains appear to be reasonably maintained (around 53 min) at long-term follow up. Evidence from repeated measures ANOVA again supports these descriptive data. The time main effect and the group×time interaction were highly significant ($p<0.001$ and p=0.002 respectively; Table 2). Investigation of sub-effects revealed that the only significant between group difference was observed after CBT treatment, prior to SMC receiving treatment (t=2.62; $p<0.01$). Fig. 2, supported by the significant time main effect, suggests that treatment gains were maintained in both groups after treatment. Graphical evidence of a slight increase in WASO for both groups at 12 months, compared to post-treatment values, proved not to be statistically significant using a related samples t-test on the combined group (t=−1.41; p=0.160; d.f.=108).

Fig. 3 presents summary data reflecting the impact of the conditions over time upon total sleep (TST). The most striking feature of these data is the relatively modest effect of intervention (whether immediate or deferred) upon TST. Participants generally continued to sleep a similar amount of time as at baseline. There appears, therefore, to be no clear superiority of CBT over SMC on this variable. At follow-up 12 months after treatment, however, a mean increase of around 0.4 to 0.55 h (25 to 35 min) per night is apparent in both groups. Formal investigation of TST revealed only a significant time main effect, reflecting this increase in total sleep during the follow up period (Table 2) and related samples t-tests confirmed that significant change occurred only between post-treatment and 12 months (t=−5.17; $p<0.001$; d.f.=108). It is noteworthy, nevertheless, that the CBT-DEF group obtained slightly less sleep after treatment than the CBT group (t=−2.38; p=0.020; Table 2). This was the only statistically significant difference between the groups across the experimental period and importantly the groups were not significantly different at baseline. The higher mean value at each phase in the CBT condition contributed to a near significant group main effect, however, the interaction term was not close to statistical significance.

4.2. Clinical importance of outcome

A summary of sleep pattern outcome, for subjects whose follow up data were available (n=109), is presented in Table 3. In overall terms, CBT was associated with a sustained, mean reduction of wakefulness from bed of around 60 min per night. Subjects typically took 30 min less time to fall asleep and had 30 min less wakefulness during the course of the night. These represented substantial proportionate changes in sleep pattern compared with baseline (50 and 36% respectively). It is noteworthy, however, that the mean increase in sleep duration was only

Table 3
Baseline to 12 month follow-up changes in sleep variables (n=109)

Variable	Mean nightly change from baseline	Percentage nightly change from baseline
Sleep-onset latency	reduction of 30 min	reduction by 50%
Wake time after sleep-onset	reduction of 30 min	reduction by 36%
Total sleep time	increase of 34 min	increase of 10%

34 min. Taken together, the results presented in Table 3 imply that subjects' sleep patterns were altered in part by gaining more sleep and in part by spending less wakeful time in bed.

Previous analyses demonstrate that immediate and deferred treatment groups responded equally to CBT. In order to consider other predictors of outcome a series of demographic and *clinically relevant*, potential explanatory variables were entered in separate regression equations for each of the three sleep pattern measures. Predictor variables included were subject sex, agegroup, marital status, occupational status, duration of sleep problem, use of hypnotic medication, medical history and several pre-treatment measures of psychopathology (BDI, STAI and PSWQ). A measure of perceived credibility of treatment was also included (Borkovec & Nau, 1972) as was a variable to define which of the therapists led the group. Pre-treatment baseline to 12 month follow up change scores were computed for the variables SOL, WASO and TST to represent clinical outcome and stepwise linear regression models were computed.

The results of regression analyses revealed that for SOL and TST no significant predictors emerged, suggesting that outcome was largely independent of any of these potential explanatory variables. For WASO, only the agegroup of the subject had any predictive effect and at a very modest level (Adj R^2=0.06; beta=-0.263; p=0.016). The oldest subjects (in the 60+ age range) achieved significantly *greater* long term reduction in WASO than the youngest subjects (aged 18 to 44 yr) (F=4.37; p=0.015; Scheffe post hoc $p<0.05$). Importantly, therefore, no contraindications for CBT treatment emerged from this analysis.

Table 4 presents baseline and post-treatment psychopathology scores. Related t-test comparisons reveal modest but significant reductions in trait anxiety and worry (both $p<0.05$). There

Table 4
Baseline and post-treatment data and paired t-test comparisons on the psychopathology of the sample (*$p<0.05$)

Variable	Baseline mean (S.D.)	Post-treatment mean (S.D.)	Paired comparison (t)	p
STAI state anxiety (n=102)	35.19 (12.16)	36.00 (12.90)	-0.74	0.460
STAI trait anxiety (n=106)	41.83 (12.74)	39.85 (11.49)	2.41	0.018*
Penn State Worry Questionnaire (n=76)	45.79 (13.99)	43.22 (12.66)	2.30	0.024*
Beck Depression Inventory (n=104)	10.94 (9.12)	10.67 (11.7)	0.28	0.781

were no changes in state anxiety or depressive symptomatology; the latter remaining in the mild range on the Beck Depression Inventory. Furthermore, of the 74 subjects originally taking hypnotic medication, 56 (76%) had stopped completely at post-treatment. Data on 60 of the original 74 subjects were available at 1 year follow-up (81%), of whom 50 (84%) no longer required hypnotics.

5. Discussion

This study set out to evaluate the clinical effectiveness of CBT for chronic insomnia using trained Health Visitors as therapists. With 139 subjects completing a controlled trial, comparing CBT with a self-monitoring control procedure (SMC), this represents the largest published outcome study on the non-pharmacological treatment of insomnia conducted in general practice. Furthermore, since the SMC group was subsequently treated with CBT, as a deferred entry to treatment procedure (CBT-DEF), a useful replication phase was incorporated in the study design. All subjects were contacted 12 months after completing treatment with 109 returning follow up measures (78%), thus permitting consideration of the medium to long-term effectiveness of the programme.

In summary of the main findings, treatment effects may be regarded as both strong and durable, with mean reduction of time awake in bed of around 60 min per night at 12 months post-treatment. Sleep-onset latency and wake time after sleep-onset each reduced by around 30 min per night, representing changes of one half and one third respectively in relation to baseline values. This equates to 7 h less in terms of sleeplessness per week for the average patient. Increases in total sleep amounted to 34 min per night; an additional 4 h sleep per week. Clearly, not all the reduction in sleeplessness was made up in sleep gain. It seems, therefore, that patients must have continued to spend less time in bed after treatment compared with baseline, but to sleep a higher proportion of the night, with less difficulty getting to sleep and remaining asleep. This would be consistent with the concept of raised ‘sleep efficiency’; the proportion of the time spent asleep to time in bed increasing as a result of treatment. Put more simply, the probability of sleeping in bed was significantly raised by CBT.

These results cannot be explained by simple attention, or passage of time, effects since SMC alone had no significant impact upon sleep, whereas active CBT was associated with marked improvement. Also, patients in the SMC condition subsequently responded equally well once CBT was introduced. Although the possibility of a placebo effect arising from structured CBT intervention cannot be entirely ruled out, since this was not specifically addressed in the present study, the results do appear similar in scale to past research, including 28 randomised placebo-controlled trials reported since 1980 (Morin et al., 1994, 1999, in press; Murtagh & Greenwood, 1995). The main difference between previous work and the present study, of course, being that the former was conducted mainly in specialised hospital settings and/or university centres with highly selected, recruited participants. The present findings from an unselected, clinically-presenting primary care population appear to provide strong validation for the CBT approach and its routine clinical effectiveness.

The durability of treatment-related change is of particular importance. In this study the great majority of participants completed follow up procedures, making the results reasonably reliable.

It also emerged that 84% of those who had originally been using hypnotic medication had successfully stopped one year later. This is a powerful indicator not only of the clinical significance of the improvements in sleep pattern which were achieved, but also of the 'mind set' of participants. It should, of course, be noted that the period from post-treatment to 12 months was not part of the controlled study reported here. All subjects had received active treatment, making it impossible to determine the longer term outcome specifically associated with CBT, as opposed to the passage of time. Nevertheless, participants in this research had lengthy histories of chronic sleep problems (Table 1) making it unlikely that many would have spontaneously improved. Furthermore, as noted earlier the evidence is that such insomnias are very persistent, if anything tending to deteriorate further over time rather than remit.

The clinical importance of the results is also evidenced by the general lack of any significant predictors of outcome arising from regression analyses of the principal sleep measures. It might be considered, for example, that participants who were younger, had less severe problems, less psychopathology or more recent and less chronic sleep problems would be more likely to benefit from CBT. However, no evidence was found to support this. Indeed, the only significant finding on any variable was that older subjects had *greater* reductions in wakefulness during the night. This is likely to be a simple reflection of lower baseline scores on this variable in younger people, in turn a reflection of sleep norms. Importantly, therefore, older adults responded equally well and being on sleep medication did not contraindicate successful outcome. Similarly, participants found the CBT intervention credible and there was no differential therapist factor effect. This suggests that the treatment package itself comprises transferable skills which can be learnt by Health Visitors, who have no prior expertise. The modest but significant reductions in trait characteristics of anxiety and worry after treatment may suggest some generalised benefit toward improving coping responses.

It seems, therefore, that Health Visitors (and potentially other nurses based in primary care settings) may offer a practicable solution to insomnia management in general practice, for several reasons. Firstly, in comparison with previous studies of CBT, using psychologists as therapists, these treatment effects are at least equivalent (cf. Morin et al., 1994, 1999, in press). Secondly, the small group approach is highly cost-efficient, enabling treatment within 1 h per case. Thirdly, Clinical Psychologists are in short supply and are more expensive; and finally, Health Visitors are integral to primary care services and have regular contact with their GP practice(s). On the basis of our experience in the West of Scotland, 10 sessions of Health Visitor time (i.e. 1 whole time equivalent, divided over say 5 staff) with 4 sessions of a Clinical Psychologist, may serve a population of 375,000. It should be reiterated that the CBT approach described here was highly structured and generally didactic. However, this does afford the possibility of 'rolling out' the model by making use of the prepared materials i.e. session by session therapist manual, patient-related notes and staff training materials. There is potential also to develop from the available materials a more self-directed intervention programme (e.g. CD-rom); the feasibility and effectiveness of which could be investigated in comparison with the small group treatment format.

There appears then to be considerable potential for the wider implementation of CBT approaches to the management of insomnia in general practice. Further research of course will be required to evaluate such implementation and this could usefully include health economic and 'quality of life' evaluation in addition to measurement of sleep parameters. The potential benefits of improved sleep both to the individual and to society are considerable and are worthy of greater

attention e.g. reduced prescribing, health gains, reduced accidents, increased productivity. Formal evaluation of treatment components would also help to identify the most active therapeutic ingredients and this could pave the way for effective early intervention.

Acknowledgements

This research was funded by a grant from the Chief Scientist Office, Scottish Office Department of Health under the Novel Health Services Research Initiative and by a grant from Ayrshire and Arran Health Board. We particularly thank the Health Visitors Jean McMillan, Gillian Dalziel, Evelyn Berry, Christine McArthur, Maureen McVey and Linda MacDonald and Barbara Gilbert (Secretary) for all their hard work. We are also grateful to Sandra Steele, Eilidh Renwick and the many other GPs who referred patients to this project and to Tracey Hepburn and Susan Dawson for their help in training the Health Visitors.

References

American Sleep Disorders Association (1990). *International classification of sleep disorders: diagnostic and coding manual.* Rochester, MA: ASDA.

American Sleep Disorders Association (1997). *Classification of sleep disorders: diagnostic and coding manual (revised).* Rochester, MA: ASDA.

Beck, A. T., Ward, C. H., Mendelson, M., Mock, J., & Erbaugh, J. (1961). An inventory for measuring depression. *Archives of General Psychiatry, 4,* 561–571.

Bixler, E. O., Kales, A., Soldatos, C. R., Kales, J. D., & Healy, B. (1979). Prevalence of sleep disorders in the Los Angeles metropolitan area. *American Journal of Psychiatry, 136,* 1257–1262.

Blood, M. L., Sack, R. L., Percy, D. C., & Pen, J. C. (1997). A comparison of sleep detection by wrist actigraphy, behavioural response and polysomnography. *Sleep, 20,* 388–395.

Bootzin, R. R., & Nicassio, P. M. (1978). Behavioural treatments of insomnia. In (pp. 1–45). M. Hersen, R. E. Eisler, & P. M. Miller, *Progress in behaviour modification, vol. 6.* New York: Academic Press.

Bootzin, R. R., & Rider, S. P. (1997). Behavioral techniques and biofeedback for insomnia. In M. R. Pressman, & W. C. Orr, *Understanding sleep: the evaluation and treatment of sleep disorders* (pp. 315–338). Washington, DC: American Psychological Association.

Borkovec, T. D., & Nau, S. D. (1972). Credibility of analogue therapy rationales. *Journal of Behaviour Therapy and Experimental Psychiatry, 3,* 257–260.

Buysse, D. J., Reynolds, C. F., Monk, T. H., Berman, S. R., & Kupfer, D. J. (1989). The Pittsburgh Sleep Quality Index: a new instrument for psychiatric practice and research. *Psychiatry Research, 28,* 193–213.

Chilcott, L. A., & Shapiro, C. M. (1996). The socioeconomic impact of insomnia: an overview. *Pharmacoeconomics, 10,* 1–14.

Douglas, N. J., Thomas, S., & Jan, M. A. (1992). The clinical value of polysomnography. *Lancet, 339,* 347–350.

Downey, R., & Bonnet, M. H. (1992). Training insomniacs to accurately perceive sleep onset. *Sleep, 15,* 58–63.

Edinger, J. D., & Wohlgemuth, W. (1999). The significance and management of persistent primary insomnia: the past, present and future of behavioral insomnia therapies. *Sleep Medicine Reviews, 3,* 101–118.

Espie, C. A. (1991). *The psychological treatment of insomnia.* Chichester, UK: J Wiley and Sons.

Espie, C. A. (1993). The practical management of insomnia: behavioural and cognitive techniques. *British Medical Journal, 306,* 509–511.

Espie, C. A. (1999). Cognitive behaviour therapy as the treatment of choice for primary insomnia. *Sleep Medicine Reviews, 3,* 97–99.

Espie, C. A., Brindle, S., Tessier, S., Dawson, S., Hepburn, T., McLellan, A., Renwick, E., Steele, S., & McColl, J. (1998). Supervised cognitive-behaviour therapy for insomnia in general medical practice — preliminary results from the West of Scotland programme. In E. Sanavio, *Behavior and cognitive therapy today: essays in honor of Hans J. Eysenck* (pp. 67–75). Kidlington, Oxford: Elsevier Science.

Espie, C. A., & Lindsay, W. R. (1985). Paradoxical intention in the treatment of chronic insomnia: six case studies illustrating variability in therapeutic response. *Behaviour Research and Therapy*, *23*, 703–709.

Espie, C. A., Lindsay, W. R., Brooks, D. N., Hood, E. M., & Turvey, T. (1989). A controlled comparative investigation of psychological treatments for chronic sleep-onset insomnia. *Behaviour Research and Therapy*, *27*, 79–88.

Foley, D. J., Monjan, A. A., Brown, S. L., Simonsick, E. M., Wallace, R. B., & Blazer, D. G. (1995). Sleep complaints among elderly persons: an epidemiologic study of three communities. *Sleep*, *18*, 425–433.

Ford, D. E., & Kamerow, D. B. (1989). Epidemiologic study of sleep disturbances and psychiatric disorders. *Journal of the American Medical Association*, *262*, 1479–1484.

Gallup Organisation (1991). *Sleep and America.* Princetown, NJ: Gallup.

Hoch, C. C., Dew, M. A., Reynolds, C. F., Buysse, D. J., Nowell, P. D., Monk, T. H., Mazumdar, S., Borland, M. D., Miewald, J., & Kupfer, D. J. (1997). Longitudinal changes in diary- and laboratory-based sleep measures in healthy "old old" and "young old" subjects: a three-year follow-up. *Sleep*, *20*, 192–202.

Johns, M. W. (1991). A new method for measuring daytime sleepiness: the Epworth Sleepiness Scale. *Sleep*, *14*, 540–545.

Kales, & Kales, J. D. (1984). *Evaluation and Treatment of Insomnia.* New York: Oxford University Press.

Lacks, P. (1987). *Behavioral Treatment for Persistent Insomnia.* New York: Psychology Practitioner Guidebooks, Pergamon Press.

Lader, M. (1992). Medical Management of Insomnia in General Practice. *The Royal Society of Medicine Services: Round Table Series* No. 28.

Mellinger, G. D., Balter, M. B., & Uhlenhuth, E. H. (1985). Insomnia and its treatment: prevalence and correlates. *Archives of General Psychiatry*, *42*, 225–232.

Mendelson, W. B. (1995). Long-term follow-up of Chronic Insomnia. *Sleep*, *18*, 698–701.

Meyer, T. J., Miller, M. L., Metzger, R. L., & Borkovec, T. D. (1990). Development and validation of the Penn State Worry Questionnaire. *Behaviour Research and Therapy*, *28*, 487–495.

Morin, C. M. (1993). *Insomnia: psychological assessment and management.* New York: Guilford Press.

Morin, C. M., Colecchi, C., & Stone, J. et al. (1999). Behavioral and pharmacological therapies for late- life insomnia: a randomised controlled trial. *Journal of the American Medical Association*, *281*, 991–999.

Morin, C. M., Culbert, J. P., & Schwartz, M. S. (1994). Non-pharmacological interventions for insomnia: a meta-analysis of treatment efficacy. *American Journal of Psychiatry*, *151*, 1172–1180.

Morin, C. M., Hauri, P. J., Espie, C. A., Spielman, A., Buysse, D. J. & Bootzin, R. R. (in press). Nonpharmacologic treatment of insomnia: report prepared by a Task Force of the American Sleep Disorders Association, Standards of Practice Committee. *Sleep.*

Morin, C. M., Kowatch, R. A., Barry, T., & Walton, E. (1993). Cognitive behavior therapy for late-life insomnia. *Journal of Consulting and Clinical Psychology*, *61*, 137–146.

Murtagh, D. R., & Greenwood, K. M. (1995). Identifying effective psychological treatments for insomnia: a meta-analysis. *Journal of Consulting and Clinical Psychology*, *63*, 79–89.

National Institute of Mental Health (1984). Consensus Conference: Drugs and insomnia — The use of medication to promote sleep. *Journal of the American Medical Association*, *251*, 2410–2414.

Russell, J., & Lader, M. (1992). *Guidelines for the prevention and treatment of benzodiazepine dependence.* London: Mental Health Foundation.

Reite, M., Buysse, D., Reynolds, C., & Mendelson, W. (1995). The use of polysomnography in the evaluation of insomnia. *Sleep*, *18*, 58.

Sadeh, A., Hauri, P. J., Kripke, D. F., & Lavie, P. (1995). The role of actigraphy in the evaluation of sleep disorders. *Sleep*, *18*, 288–302.

Sanavio, E., Vidotto, G., Bettinardi, O., Rolletto, O., & Zorzi, M. (1990). Behaviour therapy for DIMS: comparison of three treatment procedures with follow-up. *Behavioural Psychotherapy*, *18*, 151–167.

Spielberger, C. D., Gorsuch, R. L., & Lushene, R. E. (1970). *Manual for the State-Trait Anxiety Inventory*. Palo Alto, California: Consulting Psychologists Press.

Spielman, A. G., Saskin, P., & Thorpy, M. J. (1983). Sleep restriction: a new treatment of insomnia. *Sleep Research*, *12*, 286.

Tabachnick, B. G., & Fidell, L. S. (1996). *Using multivariate statistics.* New York: Harper Collins.

Walsh, J. K., Engelhardt, C. L., & Hartman, P. G. (1995). The direct economic costs of insomnia. In D. Nutt, & W. Mendelson, *Hypnotics and Anxiolytics* (pp. 369–381). London: Balliere Tindall.

Journal of Consulting and Clinical Psychology
2001, VoL 69, No. l, 58-66

X/Ol/$5.00 001: JO.J037//0022-006X.69.l.58

Predicting Clinically Significant Response to Cognitive Behavior Therapy for Chronic Insomnia in General Medical Practice: Analyses of Outcome Data at 12 Months Posttreatment

Colin A. Espie, Stephanie J. Inglis, and Linda Harvey
University of Glasgow

The clinical efficacy of cognitive behavior therapy (CBT) for chronic insomnia has been established, yet clinical effectiveness is less clear. This study presents data on 109 patients from general practice during a formal evaluation of clinical effectiveness. Two thirds achieved normative values of S30 min for sleep latency and wakefulness during the night after CBT. Furthermore, al!llost half of the sample reduced sleeplessness by 2::50%. Logistic regression revealed that initial severity did not contraindicate good outcome. Rather, greater sleep disturbance was positively associated with large symptom reduction, although lower endpoint scores were less likely. Similarly, symptoms of anxiety, depression, and thinking errors positively predicted good outcome. Hypnotic using patients responded equally well to CBT, and demographic factors were of no significant predictive value. It is concluded that CBT is clinically and durably effective for persistent insomnia in routine practice.

Insomnia is a widespread complaint affecting a sizable proportion of the general population. At least 1 in 10 adults experiences persistent difficulty getting to sleep or staying asleep (Ancoli-Israel & Roth, 1999; Ford & Kamerow, 1989; Mellinger, Balter, & Uhlenhuth, 1985), and this figure doubles to around 20% in the older adult population (Foley et al., 1995; Foley, Monjan, Simonsick, Wallace, & Blazer, 1999; Hoch et al., 1997). Not surprisingly, therefore, considerable attention has been given to the assessment and management of insomnia, culminating in guidelines being issued for clinical practice.

In relation to assessment, the American Sleep Disorders Association (ASDA) has published practice parameter statements for the use of polysomnography (ASDA, 1995b) and wrist actigraphy (ASDA, 1995a). It is accepted that the former, comprising EEG measurement of the brain's sleep stages along with recording of muscle activity, eye movement, respiration rates, and oxygen saturation levels, is not essential for the diagnosis of insomnia (Douglas, Thomas, & Tan, 1992; Reite, Buysse, Reynolds, & Mendelson, 1995). Polysomnography, however, is often crucial to appraisal and differential diagnosis of other disorders of sleep. Behavioral technologies were first reviewed by Bootzin and Engle-Friedman (1981), and the wrist actigraph is seen as the most convenient, nonintrusive device for objectively estimating sleep pattern, having been validated against polysomnography in normal participants (Hauri & Wisbey, 1992). The actigraph is a small, watch-like device that records and stores movement information in short epochs (1 min or less) by means of an accelerometer-microprocessor link. Because movement correlates highly with wakefulness and lack of movement with sleep, an algorithm enables PC-run software to accurately estimate the major sleep parameters. The monitoring of subjective sleep by means of the self-report sleep diary, however, continues to be the staple of both clinical practice and research on insomnia (Espie, 1991; Morin, 1993). The sleep diaries are completed each morning, upon rising from bed, and they represent the insomniac's perceptions not only of sleep pattern per se, but importantly also of sleep quality.

In relation to drug treatment, hypnotics may be appropriately used for transient and short-term insomnia, but their nightly use should be limited to 4 weeks or less (National Institutes of Health, 1984; Gillin & Byerley, 1990). Hypnotics are not advised for the routine or long-term treatment of persistent insomnia, because of both limited evidence of effectiveness and recognized problems on withdrawal (Kripke, 2000; Russell & Lader, 1992; Royal Society of Medicine, 1992). Contraindications for the use of hypnotics in the elderly have been particularly highlighted due to the aging of the biological clock, problems of drug accumulation, and daytime drowsiness (National Institutes of Health, 1991; Royal Society of Medicine, 1992).

In relation to nonpharmacological therapy for insomnia, it was only recently that ASDA [now the American Academy of Sleep Medicine (AASM)J commissioned an expert review group to write treatment practice parameters (Morin, Hauri, et al., 1999). Nevertheless, this position article reflects the established status of cognitive behavior therapy (CBT) as a frontline treatment for chronic

Colin A. Espie, Stephanie J. Inglis, and Linda Harvey, Department of Psychological Medicine, University of Glasgow, Glasgow, Scotland, United Kingdom.

This research was supported by grants from the Scottish Office Department of Health: Chief Scientist Office and from Ayrshire and Arran Health Board. We particularly thank the Health Visitors, who are Jean McMillan, Gillian Dalziel, Evelyn Berry, Christine McArthur, Maureen McVey, and Linda MacDonald, and Barbara Gilbert (secretary) for all their hard work. We are also grateful to Sandra Steele, Eilidh Renwick, and the many other general practitioners who referred patients to this project and to Tracey Hepburn and Susan Dawson for their help in training the Health Visitors.

Correspondence concerning this article should be addressed to Colin A. Espie, Department of Psychological Medicine, University of Glasgow, Academic Centre, Gartnavel Royal Hospital, 1055 Great Western Road, Glasgow Gl2 OXH, Scotland, United Kingdom. Electronic mail may be sent to c.espie@clinmed.gla.ac.uk.

insomnia. Typically comprising elements of stimulus control treatment, sleep restriction therapy, relaxation methods, and cognitive restructuring, either singly or in combination (see Espie, 1991; Morin, 1993), CBT has been recommended as a core component of intervention for clinical insomnia (Reite, Ruddy, & Nagel, 1997) and as the treatment of first choice for chronic insomnia (Espie, 1999). Meta-analytic data that support the efficacy of CBT have been reported elsewhere (Morin, Culbert, & Schwartz, 1994; Murtagh & Greenwood, 1995), and recent reviews and research reports have highlighted the advantages of CBT over pharmacotherapy for persistent insomnia (Edinger & Wohlgemuth, 1999), including insomnia in older adults (Morin, Colecchi, Stone, Sood, & Brink, 1999).

Unfortunately, it is not yet possible to provide satisfactory guidelines for psychological therapy that answer a key clinical question: Who benefits most from CBT for insomnia, and who benefits least? For example, Murtagh and Greenwood (1995) concluded that drug-using insomniacs may be less likely to benefit from CBT. However, work by Morin and colleagues suggests that CBT may be equally efficacious in hypnotic-using patients, and CBT may be useful to aid withdrawal from medication (Morin, Colecchi, Ling, & Sood, 1995). Espie, Inglis, Tessier, and Harvey's (2001) work supports this view. They found that hypnotic-using insomniacs responded equally well to CBT delivered in a primary care setting, and that 84% of these patients successfully withdrew from medication. If CBT were to be of limited benefit to this important clinically presenting group, then. the utility and validity of the treatment would be compromised.

The AASM article debates other potential moderators of treatment response. Whereas earlier reports suggested that younger participants were more responsive to treatment (Alperson & Biglan, 1979; Lacks & Powlishta, 1989), a series of recent studies of late-life insomnia indicate that when older adults are well screened for medical disorders and other sleep disorders (such as sleep apnea), magnitude of response is comparable to that obtained with younger patients (Davies, Lacks, Storandt, & Bertelson, 1986; Friedman, Bliwise, Yesavage, & Salom, 1991; Morin, Kowatch, Barry, & Walton, 1993; Puder, Lacks, Bertelson, & Storandt, 1983). Furthermore, there is no evidence to support the notion that older adults might find a psychological approach less acceptable. Indeed, in a recent, randomized controlled trial of behavioral and pharmacological therapies patients (mean age 65 years) consistently reported greater satisfaction with psychological therapy than drug treatment alone (Morin, Colecchi, et a!., 1999).

It might also be thought that milder sleep problems would respond readily to CBT, whereas more entrenched and severe problems would be less amenable to change. Fewer studies have directly addressed this thought, although there has been evidence for 10 to 15 years that this is not the case. Lacks, Bertelson, Gans, and Kunkel (1983) found that categorizing participants into severity groups did not differentiate in terms of significant outcome, and Espie, Lindsay, Brooks, Hood, and Turvey (1989) reported in a controlled comparative study the superiority of psychological treatments over placebo in patients referred with severe insomnia. Also, one report has suggested that patients with comorbid anxiety or depressive problems can achieve sleep improvements in keeping with those obtained by primary insomniacs (Morin, Stone, McDonald, & Jones, 1994), and Bliwise, Friedman, Nekich, and Yesavage (1995) found that improvement in sleep latency following behavioral intervention was unrelated to participants' presenting personality characteristics.

Appreciation of any indications and contraindications of good clinical outcome may be difficult to establish, however, from studies of *clinical efficacy* (which represent the majority to date). To meet the rigors of the scientific paradigm, characteristics of clinic-presenting populations (e.g., hypnotic dependence, psychopathology, poor motivation, impoverished social support) may be diluted. In simple terms, efficacy studies are weighted in favor of experimental control at the potential expense of generalization to real world settings (Clarke, 1995). For these reasons, Morin, Hauri, eta!. (1999) and Edinger and Wohlgemuth {1999) have exhorted researchers to consider clinical effectiveness studies because it is essential to establish the capacity of CBT to improve the sleep of unsolicited insomniacs in the general medical setting. In practice, of course, the distinction between efficacy and effectiveness is not absolute, but it reflects a continuum, with research varying along many dimensions such as who serves as participants and therapists, how treatment is implemented and monitored, and what the setting is in which the treatment is delivered (Hoagwood & Hibbs, 1995; Kazdin, 1998).

We have recently presented treatment outcome data on insomnia by using a methodology more consistent with the clinical effectiveness end of this continuum (see Espie eta!., 1998; Espie et al., 2001). Our participants comprised a relatively unselected sample of chronic insomniacs, consecutively presenting and treated in primary care. For example, people were accepted into the study whether or not they were currently using sleep medication, and whether or not they had comorbid anxiety or depressive symptoms. Only those who clearly had a primary disorder other than insomnia were specifically excluded. Effectiveness studies should also reflect the realities of the health system; therefore, treatment as usual may be the most appropriate control condition, and deferred entry to (active) treatment may provide the best opportunity to demonstrate clinical effects in a population who is requiring help. Using this design, our data demonstrated mean reductions of around 1 hr of sleeplessness per night, which were durable at 12 months follow-up, comparable with the extant literature. This article examines potential predictors of clinical outcome, giving particular consideration to the variables introduced in preceding paragraphs.

Method

The Sleep Clinic

The *Sleep Clinic* was developed in the West of Scotland to provide psychological treatment in a primary care setting for people presenting with severe and persistent insomnia. The assessment protocol included stringent criteria for the identification of significant sleep-onset insomnia and/or sleep-maintenance insomnia (International Classification of Sleep Disorders; ASDA, 1990), and the differential diagnosis of other sleep disorders. Consistent with recommended practice (see introductory section), overnight polysomnography assessment was conducted where disorders of sleep other than insomnia were suspected (e.g., obstructive sleep apnea), and wrist actigraphy was used to identify suspected circadian disorders (e.g., delayed sleep phase syndrome) or sleep state misperception. Sleep diaries formed the basis of outcome evaluation, and several psychopathology and other measures were applied (see below).

Participants attended six. weekly therapy sessions, each lasting *50* min. The treatment program was administered locally to small groups of 4 to 6 patients by health visitors (primary care based nurses) who had been

extensively trained in CBT for insomnia. Thus, the program was highly cost efficient, amounting to around 1 hr of therapist time per patient. The treatment format was highly structured with therapists working to a manual and patients receiving detailed notes for each session and guidance on home practice (see Espie eta!., 1998, for full details). In brief, the program comprised the following:

Session 1: Sleep information (i.e., nature and function of sleep, sleep needs, disorders and effects);

Session 2: Sleep hygiene practices (i.e., diet, exercise, and environment);

Sessions 3 and 4: Sleep scheduling (i.e., stimulus control and sleep restriction procedures, relaxation therapy); and

Sessions 5 and 6: Cognitive therapy (i.e., thought restructuring, paradoxical intention, managing anxiety and intrusive thoughts).

Participants

To investigate pretreatment variables that might predict long-term outcome after CBT for chronic insomnia, only those participants from our study cohort who had completed a 12 month posttreatment follow-up assessment were included. Therefore, of the 139 insomniacs who had completed the treatment program, 109 (78%) participated in this study. There were no significant differences between the total sample and those completing follow-up on any clinical or demographic variable (Espie et al., 2001). Importantly, there were no differences at posttreatment outcome between the two samples. Summary descriptive information on the participants is provided in Table 1, from which it is evident that these patients are typical of the clinic presenting population in having persistent problems (44% had over a 10-year duration of insomnia), with 56% taking hypnotic medication at the time of referral from their general practitioner. The sample was predominantly women with an average age of 52.1 years (*SD* = 16.2).

Measures

Apart from the general demographic and clinical information gathered, the measures used in this study can be divided into two categories; namely, outcome variables and predictor variables.

Outcome variables. Because the purpose of this study was to consider who benefits from CBT for insomnia and who does not, the outcomes of interest were the various sleep parameters, recorded on a daily basis on a self-report sleep diary (Espie, 1991). Diaries had been completed by participants throughout the experimental period of the original study (for at least 2 weeks during baseline, throughout the 6-week treatment period), and they were also completed for 1 week at 12 month follow-up. The sleep diary comprised information on sleep-onset latency (SOL: minutes), wake time after sleep onset (WASO: minutes), that is, total wakeful time during the night attributable to wakenings; number of nocturnal wakenings (nWAKE), that is, the number of discrete wakenings that the participant recalled; and a measure of sleep quality represented by enjoyment of sleep (ENJOY) rated on a 5-point Likert scale from 0 *(not at all)* through 4 *(extremely;* see Espie, 1991, pp. 80-84). The weekly mean score for each variable was calculated at 12 month posttreatment to represent sleep-pattern outcome.

Table 1
Summary Descriptive Information on the Presenting Characteristics of the 12 Month Posttreatment Study Sample (N = 109; Unless Otherwise Stated)

Descriptor	Values	*n*	%
Age	*M* = 52.1 *SD* = 16.2		
Gender	Female	78	72
	Male	31	29
Civil status	Partner	73	67
	No partner	36	33
Occupational status	Working	42	39
	Not working	67	62
History of sleep problem (*n* = 105)	<2 years	18	17
	>2 and <5 years	17	16
	>5 and < 10 years	24	23
	>lO and <20 years	21	20
	>20 years	25	24
Use of sleep medication	Taking hypnotics	59	56
	Not taking hypnotics	50	44

There has been much debate about the usefulness of appraisal of statistical significance alone, particularly in studies that are evaluating clinical populations and may be presuming clinically important effects (Jacobson & Truax, 1991; Kendall, Marrs-Garcia, Nath, & Sheldrick, 1999). Therefore, measures of change, which might represent good and poor clinical outcome for this population, were considered. High between participant variability in both baseline and outcome scores suggested it would be invalid to use standard deviation scores to establish a threshold for clinical change. It was decided, therefore, to adopt two sets of criteria for clinical significance that have been used previously in insomnia research (Espie, Brooks, & Lindsay, 1989).

First, a reduction by 50% in a particular outcome variable, relative to the baseline score for that patient, was considered clinically significant (e.g., a reduction in WASO from a mean of 80 min at baseline to 540 min at posttreatment). Dichotomous variables, therefore, were calculated for each of the four outcome measures (SOL, WASO, nWAKE, ENJOY) representing change of 549% and change of 2:50%, respectively. Second, because a minimum of 30 min of sleep disturbance, occurring at least four times per week, is one of the recognized diagnostic criteria for insomnia, it was felt that mean weekly outcome scores of SOL 530 min and WASO 530 min would provide two further approximate clinical cutoff points. Participants with this degree of sleep problem would normally be considered as subclinical, being within the range of the nonsleep-disordered population, and would not be accepted into an outcome evaluation. Thus, the 30-min criterion permits normative comparison, where clinical significance is defined as end-state functioning that falls within a normative range (Kendall et a!., 1999). Dichotomies for these two variables were created, therefore, representing achievement or nonachievement of the 30-min criterion at 12 months posttreatment. A third outcome variable for SOL and for WASO was created representing the achievement or nonachievement of both sets of criteria. It was not possible to develop comparable combined outcome criteria for the nWAKE or ENJOY variables.

Predictor variables. It was recognized that a considerable number of pretreatment factors might contribute explanatory variance within a prediction model of treatment outcome; therefore, a wide range of variables was considered. These included demographic variables [sex, age, civil status (married, cohabiting or living alone), occupation (in paid employment or not)] and aspects of the clinical history {duration of insomnia (less than 2 years; 2 to less than 5 years; 5 to less than 10 years; lO to less than 20 years; 20 years or more)], drug use (taken hypnotics in the past month or not), and daytime tiredness (tired most days vs. occasionally or not at all). The degree of presenting sleep disturbance prior to treatment was calculated by summing nightly mean scores for SOL and WASO at baseline and then creating an index of insomnia severity by subdividing patients into three groups representing one 590 min, two 91-150 min, and three 2:151 min of total sleep disturbance per night. Because the clinic routinely gathered information on psychopathology it was possible also to consider pretreatment symptoms of depression [Beck Depression Inventory (BDI); Beck & Steer, 1993], state and trait anxiety [State-Trait Anxiety Inventory (STAI); Spielberger, Gorsuch, & Lushene, 1970], and worry [Penn State Worry Questionnaire (PSWQ); Meyer, Miller, Metzger, & Borkovec, 1990), as potential predictors. Other measures available included the Sleep Disturbance Questionnaire (SDQ; Espie, Brooks, &

Lindsay, 1989; Espie, Inglis, Harvey, & Tessier, 2000) and a shortened version of the Dysfunctional Beliefs and Attitudes About Sleep scale (DBAS; Morin , Stone, Trinkle, Mercer, & Remsberg, 1993; Espie eta!., 2000). The SDQ comprises factors that correspond to participants ' attributions about the causes of their sleep problem (I, restlessness/agitation; II, mental overactivity; III, consequences of insomnia; IV, lack of sleep readiness). The DBAS comprises factors reflecting dominant attitudes and beliefs about sleep and sleeplessness (I, beliefs about the immediate negative consequences of insomnia ; II, beliefs about the negative long-term consequences of insomnia ; III, beliefs about the need for control over insomnia). Finally, Borkovec and Nau's (1972) Credibility Evaluation Questionnaire provided a measure of perceived treatment credibility, and a variable to identify which of the six therapists conducted the treatment was included to consider therapist factor.

Results

Clinical Improvement

A patient was regarded as a treatment responder if he or she achieved the relevant criterion on the variable under consideration (e.g., 50% reduction in SOL at 12 months posttreatment); otherwise the patient was coded as a nonresponder. Inspection of Table 2 reveals that 41% of patients were coded as responders in terms of SOL reduction and 50% were responders in terms of WASO reduction. Almost two thirds of patients achieved the normative comparison endpoints of 30 min (or less) for each of these variables. Thirty-five percent of patients achieved both a 50% reduction in SOL and a :530-min posttreatment value, and 40% achieved both of these outcomes for WASO. The number of wakenings from sleep (nWAKE) was reduced by half in 28%, and a similar proportion achieved a 50% increase in sleep quality as measured by their rating of sleep enjoyment (ENJOY).

Associations With Clinical Improvement

Each variable was considered first of all by examination of its relationship with each outcome measure by using the statistical test most appropriate to the type of variable (chi-square or independent *t* test, depending on whether the independent variable was categorical or continuous). The purpose of this procedure was to identify smaller subsets of potential predictors, which demonstrated some statistical effect (see Table 3). To ensure inclusion of variables with coefficients different from zero, Hosmer and Lemeshow's (1989) recommendations for logistic regression were adopted. A sensitive cutoff for statistical significance of $p < .10$ was selected for bivariate analyses, in spite of the multiple comparison testing, to identify any potentially important association that might contribute some explanatory variance in subsequent regression analyses . A number of the results identified in Table 3 as statistically significant, nevertheless, require cautious interpretation.

Notably few significant relationships were found between outcome and demographic factors. Mean age of treatment responders who achieved a WASO :530 min was 49.0 *(SD* = 15.8) compared with nonresponders who had a mean age of 56.3 *(SD* = 15.7), $t(108) = 2.32$, $p = .022$, and there was a modest effect of civil status on the variable ENJOY, indicating poorer outcome in terms of enjoyment of sleep amongst those who lived alone compared with married or cohabiting patients, ¥O. $N = 109) = 4.09, p = .043$.

As can be seen from Table 3, few sleep history variables demonstrated any relationship with outcome. In particular, duration of insomnia problem and use (or not) of a hypnotic drug were not associated with clinical outcome . There was one near-significant effect in relation to daytime tiredness, in that 47% of nonresponders on the variable nWAKE compared with only 28% of responders experienced tiredness at baseline, ¥O. $N = 109) = 3.29$, $p = .070$. Nevertheless, severity of insomnia emerged as an important factor for both of the criterion indices of SOL and WASO response. Eighty-three percent of responders achieving the SOL ;;::SO% criterion were in the most severe category before treatment (i.e., Category 3 on the index of severity), compared with 50% ofnonresponders, ¥<2.N = l3.59),p = .001; and 78% of responders on WASO ;;::SO% were in the most severe category compared with 49% of nonresponders, ¥<2. $N = 9.80$), $p = .007$. In other words, these results suggest that more severe cases do better on the 50% reduction measure. The :530 min criterion outcomes for SOL and WASO, however, seem to reflect the converse. Fifty-seven percent of responders who achieved SOL :530 min were in the most severe category at baseline compared with 78% of nonresponders, ¥(2, $N = 109) = 5.i1, p = .072$. A stronger statistical effect was observed for WASO :530 min where

Table 2
Clinically Significant Sleep Outcomes at 12 Months Posttreatment (n; %) According to Three Criteria (n = 109)

	Achievement of 50% reduction in target symptom•		Achievement of posttreatment value S:30 min		Achievement of 50% reduction and posttreatment value S:30 min"	
Variable	*n*	%	*n*	%	*n*	%
SOL	45	41.3	70	64.2	38	34.9
WASO	54	49.5	69	63.3	44	40.4
nWAKE	31	28.4				
ENJOY	29	26.6				

Note. SOL == sleep-onset latency; WASO = waketime after sleep onset; nWAKE = number of nocturnal wakenings; ENJOY = enjoyment of sleep.
• Rather than a reduction, there is an increase in relation to ENJOY.

Table 3
Investigation of Associations Between Clinical Outcomes and Pretreatment Characteristics, Prior to Entry Into Logistic Regression (n = 109)

Variable	SOL 50%	SOL 30 min	SOL 50%/ SOL 30 min	WASO 50%	WASO 30 min	WASO 50%/ WASO 30 min	nWAKE 50%	ENJOY 50%
Demographic								
Sex	.680	.834	.677	.884	.529	.836	.262	.359
Age	.189	.419	.207	.493	.022*	.257	.470	.948
Status	.586	.641	.842	.502	.966	.550	.838	.043*
Occupation	.642	.905	.859	.608	.366	.444	.932	.840
Sleep								
Duration	.842	.252	.620	.487	.610	.601	.739	.200
Drug	.725	.107	.387	.399	.582	.660	.306	.128
Tiredness	.311	.608	.559	.469	.790	.846	.070	.131
Severity	.001***	.072	.022*	.007**	.001***	.291	.368	.399
Psychopathology								
Depression	.071	.969	.034*	.035*	.150	.015*	.061	.844
State	.129	.698	.161	.044*	.043*	.018*	.265	.920
Trait	.240	.386	.071	.179	.062	.111	.170	.107
Worry	.962	.417	.530	.880	.154	.649	.424	.067
Psychological								
SDQFI	.930	.801	.991	.363	.354	.608	.759	.496
SDQ FII	.906	.715	.902	.673	.353	.833	.739	.436
SDQ Fill	.116	.675	.185	.624	.209	.371	.360	.138
SDQ FIV	.847	.944	.953	.073	.743	.053	.556	.177
DBAS FI	.578	.745	.957	.529	.227	.480	.170	.057
DBAS FII	.014*	.036*	.013*	.950	.339	.521	.837	.338
DBAS Fill	.803	.975	.960	.554	.260	.420	.688	.329
Treatment								
Credibility	.472	.607	.427	.408	.679	.808	.300	.044*
Therapist	.188	.664	.243	.213	.209	.233	.198	.140

Note. Tabulated data are *p* values from chi-square (categorical variables) or independent sample *t* tests with significant values in boldface type ($p < .10$, following Hosmer & Lemeshow, 1989). SOL = sleep-onset latency; WASO = waketime after sleep onset; nWAKE = number of nocturnal wakenings; ENJOY = enjoyment of sleep; SDQ = Sleep Disturbance Questionnaire; DBAS = Dysfunctional Beliefs and Attitudes About Sleep Scale; FI, Fll, and so on = Factor I, Factor II, and so on.
*$p < .05$. **$p < .01$. ***$p < .001$.

53% of responders were in the most severe pretreatment category compared with 86% of nonresponders, $(2, N = 109) = 12.67$, $p = .002$. Thus, the more severe cases appear to do less well on the 30-min outcome criterion. A modest but significant effect was demonstrated for the combined SOL 2:50%/:530-min criterion, $(2, N = 109) = 7.02, p = .030$. Again, there was a tendency for more severe cases to do better. No comparable effect was observed, however, for the combined WASO outcome.

Table 3 reveals that psychopathology demonstrated a number of significant relationships with the outcome variables. It is important to observe, however, that the general trend in these relationships was for higher levels of pretreatment symptomatology to be associated with better clinical outcome. For symptoms of depression, responders on the 2:50% reduction in WASO measure had a baseline mean BDI score of 12.6 (SD = 10.2) compared with the nonresponder mean of 9.1 (SD = 6.9), $t(107) = 2.14, p = .035$; and, for the combined WASO outcome, responder mean at baseline of 13.6 (SD = 10.8) compared with the nonresponder mean of 9.5 (*SD* = 7.3), $t(107) = 2.47, p = .015$. A significant effect was also observed for the combined SOL outcome [responder mean 13.7 (*SD* = 10.9) vs. nonresponder of 9.9 (*SD* = 7.8); $t(107) = 2.15, p = .035$], although, when taken separately, only the SOL :530-min criterion came close to statistical significance ($p = .071$). For nWAKE 2:50% reduction, the respective mean BDI scores were 13.4 (*SD* = 11.7) and 9.9 (SD = 7.7), $t(107) = 1.89, p = .061$. It should be borne in mind, of course, that patients with clinical depression were excluded from this study and that BDI scores were relatively low. Similarly, responders who achieved the 2:50% WASO criterion had higher baseline scores on the STAI state scale [37.4 (*SD* = 12.7) vs. 32.9 (*SD* = 8.5)], $t(107) = 2.04, p = .044$. Also, both state and trait anxiety levels were higher in responders to the :530-min WASO criterion [37.4 (*SD* = 12.7) vs. 32.9 (*SD* = 8.5)], $t(107) = 2.05, p = .043$, for state; and [44.0 (*SD* = 13.7) vs. 39.5 (SD = 9.9)], $t(107) = 1.89$, $p = .062$, for trait. Those achieving both WASO outcomes also had higher baseline state mean scores [38.9 (*SD* = 13.5) vs. 33.7 (*SD* = 9.5)]; $t(107) = 2.40, p = .018$, and those achieving both SOL outcomes had a somewhat higher trait baseline [45.5 (*SD* = 14.6) vs. 41.0 (SD = 11.3]; $t(107) = 1.83, p = .071$. Higher initial levels of worry on the PSWQ, however, were associated with poorer clinical outcome on ENJOY, the qualitative measure of sleep satisfaction [responders mean = 43.2 (*SD* = 16.3), nonresponders 49.1 (*SD* = 13.2)]; $t(99) = -1.85, p = .067$.

Other psychological factors demonstrated some association with outcome. Higher scores on DBAS Factor II (beliefs about the negative long-term consequences of insomnia) were associated with clinical improvement on both the SOL 50% reduction measure [responders mean = 163.7 (*SD* = 53.7), nonresponders

mean== 133.7 *(SD* == 67.5)]; *t(107)* == 2.49, p == .014, and the SOL :530-min criterion [155.5 (SD = 65.8) vs. 128.4 (SD == 63.0), respectively], t(l07) = 2.12, p = .036, and also on the combined SOL outcome (167.3 (SD = 54.8) vs. 135.0 (SD = 68.6)], t(l07) = 2.52, p = .013. Lower DBAS Factor I scores (beliefs about the immediate negative consequences of insomnia) were somewhat associated with greater quality of sleep on the ENJOY 50% outcome criterion [responders = 247.4 (SD = 110.8), nonresponders = 295.0 *(SD* = 111.3)], t(107) = -1.92, p = .057. Similarly, lower SDQ Factor IV scores (lack of sleep readiness) at baseline modestly discriminated clinical response in WASO using the 50% reduction criterion [responders = 2.11 (SD = 1.88), nonresponders = 2.62 *(SD* = 1.53)], t(107) = -1.81, p = .073, and the combined WASO criterion [2.07 (SD = 1.33) vs. 2.63 (SD = 1.57)], t(107) = -1.96, p = .053. ,

As can be seen from Table 3, therapist factor did not differentiate between responders and nonresponders on any clinical outcome measure, and perceived credibility of the treatment approach achieved modest significance on only one measure. Responders on the ENJOY 50% increase criterion reported higher mean treatment credibility ratings than nonresponders [25.5 *(SD* = 4.72) vs. 23.5 (SD = 4.07)], t(l01) = 2.04, p = .044.

Prediction of Clinical Improvement

The foregoing analyses represent tests of simple association or difference and, as such, do not take into account any interaction between explanatory variables in determining outcome effects. To investigate conservatively the prediction of clinically significant outcome, we selected a logistic regression model as each dependent measure was a simple dichotomous variable and the explanatory variables comprised a mix of continuous, ordinal and categorical data (Tabachnick & Fidell, 1996). The significant subset of variables (from Table 3) that had demonstrated a bivariate relationship was entered in the logistic regression model for that outcome measure to determine the combination of predictor variables which most accurately identified treatment responders and nonresponders. Results of these analyses are presented in Table 4.

Greater insomnia severity and higher scores on DBAS Factor II (beliefs about the negative long-term consequences of insomnia) together correctly predicted over half of the treatment responders and three quarters of nonresponders on the SOL 50% criterion, with severity having the stronger predictive impact. Scores for depression on the BDI, however, did not contribute significantly to the regression equation. By implication, of course, this model missed 45% of responders and 25% of nonresponders. These same two variables also emerged as accurate predictors of responding on the normative comparison variable SOL :530 min (88%), but identified fewer nonresponders (35%) and, thereby, missed 65% of nonresponders. Of the four variables considered as potential predictors only higher scores on DBAS Factor II emerged as significant for the combined SOL outcome, identifying the great majority of responders (90%) but relatively few nonresponders (21%). Three of the four variables entered contributed significantly to the prediction of WASO 50% reduction. Symptoms of depression, SDQ Factor IV (lack of sleep readiness) and severity predicted 70% and 64% of responders and nonresponders, respectively, whereas STAI state was excluded from the analysis. However, state anxiety entered along with severity as the two significant predictors of normative outcome on WASO :530 min, correctly identifying 77% of responders and almost half of nonresponders, but consequently missing 23% of responders and 50% of nonresponders. Age and trait anxiety failed to contribute additional explanatory power. Greater symptomatology of depression and lower SDQ Factor IV scores together predicted 87% of responders and 42% of nonresponders on the combined WASO criterion, with state anxiety failing to add to the equation.

Interestingly, symptoms of depression correctly predicted 100% of nonresponders on nWAKE, but identified only 13% of respond-

Table 4
Predictors of Clinically Significant Outcome for Each of the Sleep Outcome Measures (n = 109)

Outcome variable	Log likelihood	¥	*df*	Sig.	Predictors	Significance	*R*	%correct responders	%correct nonresponders
SOL 2:50% reduction	132.6	17.3	3	.0006	Severity	.010	.195	55.6	74.2
					DBAS Factor II	.061	.106		
SOL :530 min	133.7	13.1	3	.0044	DBAS Factor II	.010	.183		
					Severity	.023	.160	87.7	3S.O
SOL ;;::so%/:530 min	132.5	11.8	1	.0082	DBAS Factor II	.03S	.130	90.7	21.0
WASO ;;::SO% reduction	128.9	19.4	4	.0007	BDI	.014	.17S	70.4	64.2
					SDQ Factor IV	.032	-.139		
					Severity	.038	.137		
WASO :s:30 min	123.1	23.1	3	<.0001	Severity	.001	.274		
					STAI state	.013	.178	77.1	46.3
WASO 2:50%/:530 min	13S.1	15.9	2	.0004	BDI	.002	.231		
					SDQ Factor IV	.00S	-.196	86.6	42.2
nWAKE ;;::SO% reduction	129.9	3.4	1	.0671	BDI	.068	.010	12.S	100.0
ENJOY 2:SO% increase	95.1	11.7	3	.0085	PSWQ	.014	-.201		
					Status	.076	-.108	16.7	94.3
					Credibility	.090	.094		

Note. Results represent logistic regression analyses comprising consideration of contributing variables from Table 3 where p < .10 (following Hosmer & Lemeshow, 1989). SOL = sleep-onset latency; WASO = waketirne after sleep-onset; nWAKE = number of nocturnal wakenings; ENJOY = enjoyment of sleep; SDQ = Sleep Disturbance Questionnaire; DBAS = Dysfunctional Beliefs and Attitudes About Sleep Scale; BDI = Beck Depression Inventory; STAI = State-Trait Anxiety Inventory; PSWQ = Penn State Worry Questionnaire.

ers. Daytime tiredness did not contribute further to this effect. Finally, a combination of worry, civil status, and perceived credibility correctly predicted 94% of nonresponders in terms of ENJOY, but failed to identify 85% of responders. Worry had the greatest impact on this regression equation and DBAS Factor I (concerns about the immediate negative impact of insomnia) was excluded from the analysis.

Discussion

Our results suggest that CBT is a clinically effective treatment for persistent insomnia. Around two-thirds of patients achieved and maintained a sleep latency of s30 min 1 year after completion of therapy, and a similar proportion achieved :S30 min of wakefulness during the night. In terms of normative comparisons (Kendall et a!., 1999), these results are encouraging because they indicated substantial normalization of sleep patterns after treatment. That is, a high proportion of our patients had sleep patterns similar to normal controls after CBT. Furthermore, almost half of the participants reduced their sleep latency and night-time wakefulness by ::::::50%. Although those with greater levels of sleep disturbance at baseline were more likely to achieve this reduction, raising the possibility of regression to the mean, 35% to 40% of participants, in fact, achieved both of our outcome criteria. Changes in the number of wakenings per night of sleep and in perceived sleep quality were less pronounced, although more than one quarter achieved a 50% reduction in waking frequency and a 50% increase in sleep enjoyment.

These results are broadly comparable with the AASM article, which suggested that "between 70% and 80% of insomnia patients benefit from treatment, *50% achieving clinically meaningful outcomes* [italics added], and about one third becoming good sleepers" (Morin, Hauri, et al., 1999). The present results are also comparable with a previous study where we used the same criterion values (Espie, Brooks, & Lindsay, 1989). In the earlier study, 40% of patients achieved a final SOL of ::::;30 min, with 71% responding to stimulus control treatment (two thirds responded to the present multicomponent CBT program). Similarly, 47%-50% achieved the 50% reduction in SOL criterion compared with the present 41%, although stimulus control specifically had achieved a 64% patient response in the former study. It should be noted that the early trial excluded patients with intermittent wakenings or combined onset or maintenance insomnia.

Logistic regression analyses confirmed that initial severity of insomnia did not contraindicate good outcome and, importantly, hypnotic-using patients responded equally well to CBT. Age and other demographic factors were not of significant predictive value but symptoms of anxiety or depression and thinking errors positively predicted good outcome. Our results, therefore, cannot be simply explained by differential responsiveness across the spectra of insomnia severity or chronicity, or in terms of degree of clinical complexity.

Neither gender nor occupational status differentiated responders from nonresponders. Responders achieving 30 min or less nighttime wakefulness at 1 year follow-up were on average 6 years younger, but age did not contribute significantly to the regression equation for this variable, and it did not discriminate on any other variable. A significant difference was found in sleep quality between insomniacs who had partners and those who did not. This variable did enter the regression equation for the variable ENJOY, having a modest effect, particularly toward the prediction of nonresponding. Perhaps insomniacs without partners are less likely to experience qualitative improvement in sleep, although responding equally well in terms of sleep pattern.

Half of our sample took hypnotic medication, but drug use per se was not a contraindication for CBT. Indeed, the vast majority of hypnotic users (50 out of 59; 84%) were drug free at follow-up (Espie et a!., 2001), strongly supporting previous evidence that CBT can be useful to facilitate benzodiazepine discontinuance (Espie, Lindsay, & Brooks, 1988; Morin et al., 1995). Similarly, length of history of insomnia did not discriminate outcomes on any variable. Daytime tiredness achieved an effect on one variable, where there was a significant difference between responders and nonresponders on night wakenings, but failed to enter the regression equation for nWAKE.

Severity of insomnia emerged consistently as a predictive factor; however, the results would not support the assumption that CBT was less effective for more severe insomnias. Rather, associations between outcome and severity may simply reflect the law of initial values in that there is greater scope for large percentage of change where baseline sleeplessness is high, and to achieve the 30-min criterion where initial values are lower. Inspection of the regression data indicates that severity was the strongest element of the prediction equation for only two of the four variables on which it loaded, and the fact that severity failed to contribute significantly to the regression equations for either of the combined sleep outcomes (SOL or WASO) lends further support to the law of initial values interpretation.

There was a tendency for higher levels of anxiety or depressive symptomatology to be associated with better responding, particularly in terms of nighttime wakefulness. It should be noted, however, that patients with primary depressive illness were excluded. Nevertheless, it appears that those with relatively higher symptom levels responded to CBT by achieving greater continuity in their sleep. The fact that posttreatment BDI and STAI state anxiety scores were not significantly lower than at baseline (see Espie et a!., 2001) appears to exclude the possibility that insomnia was being treated as a secondary response to an underlying condition. Because numerous studies have found that insomniacs are somewhat neurotic (e.g., Edinger, Stout, & Hoelscher, 1988; Lundt, Broman, & Hetta, 1995), it may be more likely that such trait characteristics are part of the enduring clinical picture of people prone to insomnia.

Although only achieving significance on one variable, worry appeared to form a different relationship with outcome. Higher baseline PSWQ scores were associated with poorer sleep quality outcome, and worry emerged as the strongest of the three predictors of ENJOY in the logistic regression analysis. It seems that sleep quality improvement, but not sleep pattern improvement, may be somewhat compromised by high pretreatment levels of worry. Interestingly, however, our posttreatment data indicated that PSWQ scores did reduce modestly in association with effective CBT treatment for insomnia (Espie et al., 2001).

Other psychological factors were also investigated. The Dysfunctional Beliefs and Attitudes About Sleep scale revealed that stronger beliefs in the negative long-term consequences of insomnia (e.g., "I am concerned that chronic insomnia may have serious consequences on my physical health") were associated with good

clinical response on all three SOL outcome measures. It seems possible that greater evidence of dysfunctional thinking may be a positive indication for CBT treatment of sleep-onset insomnia. This may suggest a cognitive pathway for mediating treatment effects, and it is supported by data demonstrating significant shift in dysfunctional beliefs resulting from CBT intervention (Espie et a!., 2000). In contrast, stronger beliefs in the immediate negative consequences of insomnia (DBAS Factor I; e.g., "After a poor night's sleep I know that it will interfere with my daily activities the next day") were associated with poorer outcome in terms of sleep enjoyment. However, this factor did not contribute significantly to the overall regression equation for the variable ENJOY. Only one of the Sleep Disturbance Questionnaire factors emerged as a significant predictor (Factor IV, concerning a perception of not be tired enough to sleep). This was associated with less likelihood of achieving 50% WASO reduction or combined WASO reduction and possibly reflects an impaired homeostatic sleep drive and/or poor stimulus control.

The final areas in which predictive relationships could be explored concerned the perceived credibility of treatment and the role of the therapist. In relation to the latter, no differential effectiveness was observed across the six therapists who conducted the CBT intervention. This implies that the treatment was similarly applied, and that the therapists had acquired comparable, effective levels of skill. Treatment credibility, however, emerged as modestly important in relation to quality, but not pattern, of sleep. In particular, patients whose enjoyment of sleep failed to improve had lower initial expectations of therapy.

Our conclusions concerning predictive factors must be tempered by the fact that explained variance associated with each significant effect was in the region of 10%-25% per variable *(R* value). The results, therefore, represent fairly modest predictive models of sleep outcome. Also, the hit rate in identification of responders and nonresponders varied considerably across outcomes. For example, greater worry combined with living alone and lower perceived treatment credibility correctly identified 94% of sleep quality in nonresponders; but it contributed little explanatory power in terms of positive responding. Similarly, low depressive symptom scores identified all those who failed to achieve a 50% reduction in waking frequency, but only 1 out of 8 responders. By contrast, high levels of dysfunctional thinking along with less severe insomnia identified 88% of responders achieving sleep latency of 30 min or less, but picked up only one third of nonresponders. Correct identification rates were more equally balanced for the other variables, but it must be borne in mind that the total case identification rate only averaged around 65% for these measures.

Finally, it should be recognized that our analyses considered only pretreatment variables that may influence outcome. Many other factors may play a part in the prediction of clinical response. These include readiness to change (the predispositional stage at which the insomniac is in terms of adopting a novel approach), motivation and compliance (the extent to which parts or all of the program are implemented at home), dose response relationship (the amount of intervention required to achieve a given level of response), critical components (the unpacking of the intervention to establish critical ingredients), sequencing of intervention (the order of presentation of treatment components), psychological "set" (including psychological mindedness and self-efficacy beliefs), and relapse prevention (the assimilation and internalization of both behavioral and attitudinal responses to cope with symptom recurrence). Further investigation of these areas, of course, would be valuable.

References

Alperson, J., & Biglan, A. (1979). Self-administered treatment of sleep-onset insomnia and the importance of age. *Behavior Therapy, /0,* 347-356.

American Sleep Disorders Association. (1990). *International classification of sleep disorders, diagnostic and coding manual.* Rochester, MA: Author.

American Sleep Disorders Association. (1995a). Practice parameters for the use of actigraphy in the clinical assessment of sleep disorders. *Sleep, 18,* 285-287.

American Sleep Disorders Association. (1995b). Practice parameters for the use of polysomnography in the evaluation of insomnia. *Sleep, 18,* 55-57.

Ancoli-Israel, S., & Roth, T. (1999). Characteristics of insomnia in the United States: Results of the 1991 National Sleep Foundation Survey I. *Sleep, 22* (Suppl. 2), S347-S353.

Beck, A. T., & Steer, R. A. (1993). *The Beck Depression Inventory: Manual.* San Antonio, TX: The Psychological Corporation.

Bliwise, D. L., Friedman, L., Nekich, J. C., & Yesavage, J. A. (1995). Prediction of outcome in behaviorally based insomnia treatments. *Journal of Behaviour Therapy and Experimental Psychiatry, 26,* 17-23.

Bootzin, R. R., & Engle-Friedman, M. (1981). The assessment of insomnia. *Behavioral Assessment, 3,* 107-126.

Borkovec, T. D., & Nau, S. D. (1972). Credibility of analogue therapy rationales. *Journal of Behaviour Therapy and Experimental Psychiatry, 3,* 257-260.

Clarke, G. N. (1995). Improving the transition from basic efficacy research to effectiveness studies: Methodological issues and procedures. *Journal of Consulting and Clinical Psychology, 63,* 718-725.

Davies, R., Lacks, P., Storandt, M., & Bertelson, A. D. (1986). Counter-control treatment of sleep-maintenance insomnia in relation to age. *Psychology and Aging, 1,* 233-238.

Douglas, N. J., Thomas, S., & Tan, M. A. (1992). The clinical value of polysomnography. *Lancet, 339,* 347-350.

Edinger, J.D., Stout, A. L., & Hoelscher, T. J. (1988). Cluster analysis of insomniacs' MMPI profiles: Relation of subtypes to sleep history and treatment outcome. *Psychosomatic Medicine, 50,* 77-87.

Edinger, J.D., & Wohlgemuth, W. (1999). The significance and management of persistent primary insomnia: The past, present and future of behavioral insomnia therapies. *Sleep Medicine Reviews, 3,* 101-118.

Espie, C. A. (1991). *The psychological treatment of insomnia.* Chichester, England: Wiley.

Espie, C. A. (1999). Cognitive behaviour therapy as the treatment of choice for primary insomnia. *Sleep Medicine Reviews, 3,* 97-99.

Espie, C. A., Brindle, S., Tessier, S., Dawson, S., Hepburn, T., McLellan, A., Renwick, E., Steele, S., & McColl, J. (1998). Supervised cognitive-behaviour therapy for insomnia in general medical practice-Preliminary results from the West of Scotland programme. In E. Sanavio (Ed.), *Behavior and cognitive therapy today: Essays in honor of Hans J. Eysenck* (pp. 67-75). Kidlington, Oxford, England: Elsevier.

Espie, C. A., Brooks, D. N., & Lindsay, W. R. (1989). An evaluation of tailored psychological treatment of insomnia. *Journal of Behaviour Therapy and Experimental Psychiatry, 20,* 143-153.

Espie, C. A., Inglis, S. J., Harvey, L., & Tessier, S. (2000). Insomniacs" attributions: Psychometric properties of the Dysfunctional Beliefs and Attitudes About Sleep Scale and the Sleep Disturbance Questionnaire. *Journal of Psychosomatic Research, 48,* 141-148.

Espie, C. A., Inglis, S. J., Tessier, S., & Harvey, L. (2001). The clinical effectiveness of cognitive behaviour therapy in the treatment of chronic

insomnia: Implementation and evaluation of a sleep clinic in general medical practice. *Behaviour Research and Therapy, 39,* 45-60.

Espie, C. A., Lindsay, W. R., & Brooks, D. N. (1988). Substituting behavioural treatment for drugs in the treatment of insomnia: An exploratory study. *Journal of Behaviour Therapy and Experimental Psychiatry, 19,* 51-56.

Espie, C. A., Lindsay, W. R., Brooks, D. N., Hood, E. M., & Turvey, T. (1989). A controlled comparative investigation of psychological treatments for chronic sleep-onset insomnia. *Behaviour Research and Therapy,* 27, 79-88.

Foley, D. J., Monjan, A. A., Brown, S. L., Simonsick, E. M., Wallace, R. B., & Blazer, D. G. (1995). Sleep complaints among elderly persons: An epidemiologic study of three communities. *Sleep, 18,* 425-433.

Foley, D. J., Monjan, A. A., Simonsick, E. M., Wallace, R. B., & Blazer, D. G. (1999). Incidence and remission of insomnia among elderly adults: An epidemiologic study of 6,800 persons over three years. *Sleep, 22 (Suppl. 2),* S366-S372.

Ford, D. E., & Kamerow, D. B. (1989). Epidemiologic study of sleep disturbances and psychiatric disorders. *Journal of the American Medical Association, 262,* 1479-1484.

Friedman, L., Bliwise, D. L., Yesavage, J. A., & Salom, S. R. (1991). A preliminary study comparing sleep restriction and relaxation treatments for insomnia in older adults. *Journal of Gerontology, 46,* 1-8.

Gillin, J. C., & Byerley, W. F. (1990). The diagnosis and management of insomnia. *New England Journal of Medicine, 322,* 239-248.

Hauri, P. J. & Wisbey, J. (1992). Wrist actigraphy in insomnia. *Sleep, 15,* 293-301.

Hoagwood, K., Hibbs, E., Brent, D., & Jensen, P. (1995). Introduction to the Special Section: Efficacy and effectiveness in studies of child and adolescent psychotherapy. *Journal of Consulting and Clinical Psychology, 63,* 683-725.

Hoch, C. C., Dew, M.A., Reynolds, C. F., Buysse, D. *1.,* Nowell, P. D., Monk, T. H., Mazumdar, S., Borland, M. D., Miewald, J., & Kupfer, D. J. (1997). Longitudinal changes in diary- and laboratory-based sleep measures in healthy "old old" and "young old" subjects: A three-year follow-up. *Sleep, 20,* 192-202.

Hosmer, D. W., & Lemeshow, S. (1989). *Applied logistic regression.* New York: Wiley.

Jacobson, N. S., & Truax, P. (1991). Clinical significance: A statistical approach to defining meaningful change in psychotherapy research. *Journal of Consulting and Clinical Psychology, 59,* 12-19.

Kazdin, A. E. (1998). *Methodological issues and strategies in clinical research* (2nd ed.). Washington, DC: American Psychological Association.

Kendall, P. C., Marrs-Garcia, A., Nath, S. R., & Sheldrick, R. C. (1999). Normative comparisons for the evaluation of clinical significance. *Journal of Consulting and Clinical Psychology, 67,* 285-299.

Kripke, D. F. (2000). Chronic hypnotic use: Deadly risks, doubtful benefit. *Sleep Medicine Reviews, 4,* 5-20.

Lacks, P., Bertelson, A. D., Gans, L., & Kunkel, J. (1983). The effectiveness of three behavioral treatments for different degrees of sleep-onset insomnia. *Behavior Therapy, 14,* 593-605.

Lacks, P., & Powlishta, K. (1989). Improvement following behavioral treatment for insomnia: Clinical significance, long-term maintenance and predictors of outcome. *Behavior Therapy, 20,* 117-134.

Lundt, L-G., Broman, J-E., & Hetta, J. (1995). Personality traits in patients with persistent insomnia. *Personality and Individual Differences, 18,* 393-403.

Mellinger, G. D., Balter, M. B., & Uhlenhuth, E. H. (1985). Insomnia and its treatment: Prevalence and correlates. *Archives of General Psychiatry, 42,* 225-232.

Meyer, T. J., Miller, M. L., Metzger, R. L., & Borkovec, T. D. (1990). Development and validation of the Penn State Worry Questionnaire. *Behaviour Research and Therapy, 28,* 487-495.

Morin, C. M. (1993). *Insomnia: Psychological Assessment and Management.* New York: Guilford Press.

Morin, C. M., Colecchi, C. A., Ling, W. D., & Sood, R. K. (1995). Cognitive behavior therapy to facilitate benzodiazepine discontinuation among hypnotic dependent patients with insomnia. *Behavior Therapy, 26,* 733-745.

Morin, C. M., Colecchi, C., Stone, J., Sood, R., & Brink, D. (1999). Behavioral and pharmacological therapies for late-life insomnia: A randomized controlled trial. *Journal of the American Medical Association, 281,* 991-999.

Morin, C. M., Culbert, J. P., & Schwartz, M. S. (1994). Nonpharmacological interventions for insomnia: A meta-analysis of treatment efficacy. *American Journal of Psychiatry, 151,* 1172-1180.

Morin, C. M., Hauri, P. J., Espie, C. A., Spielman, A., Buysse, D. J., & Bootzin, R. R. (1999). Nonpharmacologic treatment of chronic insomnia: An American Academy of Sleep Medicine review. *Sleep,* 22, 1-25.

Morin, C. M., Kowatch, R. A., Barry, T., & Walton, E. (1993). Cognitive behavior therapy for late-life insomnia. *Journal of Consulting and Clinical Psychology, 61,* 137-146.

Morin, C. M., Stone, J., McDonald, K., & Jones, S. (1994). Psychological treatment of insomnia: A clinical replication series with I 00 patients. *Behavior Therapy, 25,* 159-177.

Morin, C. M., Stone, J., Trinkle, D., Mercer, J., & Remsberg, S. (1993). Dysfunctional beliefs and attitudes about sleep among older adults with and without insomnia complaints. *Psychology and Aging, 8,* 463-467.

Murtagh, D. R., & Greenwood, K. M. (1995). Identifying effective psychological treatments for insomnia: A meta-analysis. *Journal of Consulting and Clinical Psychology, 63,* 79-89.

National Institutes of Health. (1984). Consensus Conference: Drugs and insomnia-The use of medication to promote sleep. *Journal of the American Medical Association, 251,* 2410-2414.

National Institutes of Health. (1991). Consensus development conference statement: The treatment of sleep disorders of older people (March 26-28, 1990) *Sleep, 14,* 169-177.

Puder, R., Lacks, P., Bertelson, A. D., & Storandt, M. (1983). Short-term stimulus control treatment of insomnia in older adults. *Behavior Therapy, 14,* 424-429.

Reite, M., Buysse, D., Reynolds, C., & Mendelson, W. (1995). The use of polysomnography in the evaluation of insomnia. *Sleep, 18,* 58-70.

Reite, M., Ruddy, J., & Nagel, K. (1997). *Evaluation and management of sleep disorders.* Washington, DC: American Psychiatric Press.

Royal Society of Medicine. (1992). In M. Lader (Ed.), *Medical management of insomnia in general practice.* (Round Table Series No. 28). London: RSM Services.

Russell, J., & Lader, M. (1992). *Guidelines for the prevention and treatment of benzodiazepine dependence*. London: Mental Health Foundation.

Spielberger, C. D., Gorsuch, R. L., & Lushene, R. E. (1970). *Manual for the State-Trait Anxiety Inventory.* Palo Alto, CA: Consulting Psychologists Press.

Tabachnick, B. D., & Fidell, L. S. (1996). *Using multivariate statistics.* New York: Harper Collins.

Received October 21, 1999
Revision received June 29, 2000
Accepted July 6, 2000 •

PERGAMON

Behaviour Research and Therapy 40 (2002) 75–83

BEHAVIOUR RESEARCH AND THERAPY

www.elsevier.com/locate/brat

Insomniacs' reported use of CBT components and relationship to long-term clinical outcome

Linda Harvey, Stephanie J. Inglis, Colin A. Espie *

University of Glasgow, Department of Psychological Medicine, Academic Centre, Gartnavel Royal Hospital, 1055 Great Western Road, Glasgow G12 0XH, UK

Accepted 12 December 2000

Abstract

Although there is considerable evidence for the efficacy of non-pharmacological treatment of insomnia, many of the larger trials have delivered CBT in multicomponent format. This makes it impossible to identify critical ingredients responsible for improvement. Furthermore, compliance with home implementation is difficult to ascertain in psychological therapies, and even more so when trying to differentiate across a range of elements. In the present report, 90 patients who had completed 12 month follow-up after participation in a clinical effectiveness study of CBT in general medical practice, responded to a questionnaire asking them about their use of the ten components of the programme. Reports of home use were then entered as predictors of clinical response to treatment. Results indicated that reported home use of stimulus control/sleep restriction was the best predictor of clinical improvement in sleep latency and nighttime wakefulness. Cognitive restructuring also contributed significantly to reduction in wakefulness. In spite of being the most highly endorsed component (by 79% of respondents) use of relaxation did not predict improvement on any variable. Similarly, sleep hygiene was unrelated to sleep pattern change and use of imagery training was modestly predictive of poor response in terms of sleep latency. There are methodological limitations to this type of post hoc analysis, nevertheless, these results being derived from a large patient outcome series raise important issues both for research and clinical practice.

Keywords: Insomnia; Sleep; Cognitive behavioural treatment; Predictors

* Corresponding author. Tel.: +44-141-211-3903; fax: +44-141-357-4899.
E-mail address: c.espie@clinmed.gla.ac.uk (C.A. Espie).

0005-7967/01/$ - see front matter
PII: S0005-7967(01)00004-3

1. Introduction

It is difficult to gauge the extent to which patients comply with cognitive behavioural treatment (CBT). Generally, such interventions rely upon home practice and there is no objective criterion by which compliance can be measured. Beneficial effects, either within or between treatments, therefore, could be accounted for simply in terms of patterns of implementation and/or perceived credibility. On the other hand, there may be no a priori reason to suspect that acceptability varies amongst treatments. Early research on insomnia, comparing single component interventions such as stimulus control, relaxation and paradoxical intention, suggested that these treatments were equally credible, in terms of therapeutic rationale (Turner & Ascher, 1979; Espie, Lindsay, Brooks, Hood, & Turvey, 1989), although sleep hygiene education may be less so (Schoicket, Bertelson, & Lacks, 1988). There is very limited evidence on compliance. In a recent influential study, patients and significant others reported similarly high levels of compliant behaviour on a rating scale measure across CBT and pharmacological treatments, and urine screens supported their reports in terms of benzodiazepine use (Morin, Colecchi, Stone, Sood, & Brink, 1999). However, some work on relaxation therapies, using discreet counters in audiocassette machines, does suggest that people over-report home practice (Lichstein & Hoelscher, 1986). Therefore, although chronic insomniacs of all ages appear to find CBT credible and even older adults are generally accepting of a psychological approach (Morin, Gaulier, Barry, & Kowatch, 1992), we do not really know how this translates into action.

These issues are compounded by the clinical practice of multicomponent CBT delivery. Systematic comparisons of single components are more typical of efficacy studies, whereas ‘package’ treatments reflect clinical practice (Chesson et al., 1999). Recent commentaries on non-pharmacological treatments of insomnia have stressed the importance of research leading to the identification of critical ingredients in therapy (Morin et al., 1999; Edinger & Wohlgemuth, 1999). This would be valuable not only conceptually by suggesting possible mechanisms of effect, but also practically, in terms of the cost-efficient use of scarce resources and an understanding of the ‘dose–response’ relationship for psychological practice.

We have recently reported outcome data from a large clinical effectiveness study using multicomponent CBT for insomnia. Our results demonstrate significant and sustained improvements in sleep pattern and sleep quality at one year follow-up, with average reductions in sleeplessness of around 60 min per night (Espie, Inglis, Tessier, & Harvey, 2001). Up to two-thirds of our patients no longer met criteria for insomnia at one year, and 40% had reduced sleep latency and wakeful time in bed after sleep-onset by at least 50%. Over 80% of those previously on sleeping pills had stopped taking them, and patients reported finding CBT highly credible (Espie, Inglis, & Harvey, 2001). Importantly, these outcomes were obtained across the full range of clinical presentation, and were not contraindicated by factors such as severity of insomnia, associated psychopathology or use of sleep medication. Although our study was not designed to tease out treatment component effects, we do have data which permit quantification of patients’ self-reported home use of elements of the programme, and we are able to compare reported use of these components between treatment ‘responders’ and ‘non-responders’ at 1 yr follow-up.

2. Method

2.1. Design

The *Sleep Clinic* was established to conduct a controlled clinical effectiveness trial of CBT for chronic insomnia in general practice. Patients presenting with difficulty initiating or maintaining sleep, according to International Classification of Sleep Disorders Criteria (American Sleep Disorders Association, 1990) were randomly assigned either to immediate or deferred CBT, conducted in small groups, by a Health Visitor (nurse based in primary care) who had been trained in the use of a manualised CBT intervention. Treatment comprised six sessions, each of 50 min duration, comprising sleep education, sleep hygiene, stimulus control and sleep restriction, and cognitive therapy techniques. Since patients were seen in groups of 4–6 the average treatment time per patient was around 1 h. A full description of the treatment programme can be found elsewhere (Espie et al., 1998).

2.2. Subjects

A total of 139 insomniacs participated and were followed up 1 yr after completion of CBT. Summary information has been presented elsewhere (Espie, Inglis, Tessier, & Harvey, 2001; Espie, Inglis, & Harvey, 2001) but for descriptive purposes, our 12-month follow-up sample (n=109) comprised 78 females and 31 males with mean age of 52.1 yr (SD 16.2). Two-thirds of the sample had insomnia for more than 5 yr and over half were regular users of hypnotic drugs before treatment. There were no significant differences between the follow-up sample and the larger cohort who completed treatment.

2.3. Measures

These 109 subjects were mailed a questionnaire which enquired about their use of 10 components of the CBT programme. These related to: two components of ‘sleep hygiene’ i.e. lifestyle and bedroom factors, stimulus control/sleep restriction, naps, the pre-bedtime routine, progressive relaxation, cognitive control, imagery, thought-blocking and cognitive restructuring (see Table 1). The questionnaire explained that, in our further development of the *Sleep Clinic*, we were interested in discovering what they felt had been the most helpful parts of the programme and ones they used to strengthen their sleep pattern. Subjects were simply asked to respond ‘yes’ or ‘no’ to each item and to return the questionnaire by mail.

3. Results

Responses were received from 90 of the 109 patients for whom 12 month post-treatment sleep data were available (83%). This sample, which comprised 66 females and 24 males with mean age of 53.9 yr (SD 16.1), was not significantly different from the complete follow-up cohort. Stated use of the different treatment components revealed considerable variation (Table 1). Around three-quarters reported finding relaxation beneficial and almost 60% avoided napping outwith the

Table 1
Reported use (%) of CBT components at one year follow-up (n=90), and identification of potential predictors of treatment responding (responders vs non-responders on seven outcome variables) for entry to logistic regression (Chi-square: **$P<0.05$; *$P<0.15$ following Hosmer & Lemeshow, 1989)

CBT Component	% Reporting use at 12 mth post-CBT	SOL ≤30 min	SOL ≥50%	SOL both	WASO ≤30 min	WASO ≥50%	WASO both	ENJOY
Altering lifestyle (diet, caffeine, exercise)	49							*
Altering bedroom factors (mattress, temperature, lighting)	32							
Stimulus control/sleep restriction (using bed only for sleep, reducing time in bed)	41	**	*	*	**			
Not taking naps during day or evening	59				*		*	
Pre-bed routine (slowing down 90 minutes before bed, following routine)	52					*		
Relaxing (using abbreviated progressive relaxation)	74							
Cognitive control (dealing with thoughts/plans/worries early in the evening)	21					*	*	*
Imagery techniques (visualisation, imagery)	19	*	*	**				
Blocking thoughts (blocking and suppressing)	41							
Cognitive restructuring (altering beliefs about sleep)	41		*		*			

sleep period. Maintenance of a pre-bedtime routine and sleep hygiene practices associated with diet and exercise were reported by around half the participants. Stimulus control/sleep restriction, cognitive restructuring and thought blocking were identified by around 40%, with other aspects of sleep hygiene and putting the day to rest using cognitive control reported by proportionately fewer respondents (32 and 21% respectively). The least used technique was imagery at 19%. It cannot be assumed, of course, that these reports reflect behaviours initiated during CBT. For example, subjects may never have napped even prior to treatment.

Table 1 also presents comparative data for treatment responders versus non-responders. Three criteria were used to determine clinical response, for each of two sleep pattern variables—sleep-onset latency (SOL; min.) and wake time after sleep-onset (WASO; min.) (see Espie, Inglis, & Harvey, 2001). Firstly, achievement of a sleep diary value (end state functioning) of $\leq$30 min; secondly, $\geq$50% reduction in minutes of sleeplessness, attributed to that variable; and thirdly, achievement of both of these reductions. In addition, one measure of sleep quality (rating of 'sleep enjoyment' on a five point Likert scale: ENJOY) was included using the $\geq$50% reduction criterion. Thus, Table 1 shows Chi-square probabilities for use of CBT components (yes/no) associated with improvement/non-improvement on seven clinical outcomes. The use of this number of variables appears justified because of the exploratory nature of the study. Furthermore, there was, at best, modest intercorrelation amongst the seven outcome variables according to Spearman statistics (range of ρ=0.08 to 0.59). Consistent with the argument presented by Hosmer & Lemeshow (1989) and endorsed by Tabachnick & Fidell (1996) the cut-off value for detecting potential effects was set sensitively at $P<0.15$. This was in order to avoid Type II error in subsequent regression analyses. Where there is inclusion of only those values which achieve high levels of significance (such as $P<0.05$) on simple bivariate analysis there is the possibility of bias toward inter-correlated predictors at the expense of other predictors which might be prematurely excluded. In contrast we wished to ensure entry of all variables with coefficients which potentially differed from zero since the regression model itself would act conservatively at the subsequent stage.

Inspection of Table 1 suggests that stimulus control/sleep restriction may have been differentially applied between responders and non-responders. For example, the likelihood of being a responder when using this component was over four times higher for both SOL and WASO $\leq$30 min (χ^2=7.18, P=0.027; χ^2=6.59, P=0.037 respectively). Similarly, subjects using cognitive restructuring had a higher probability (times three) of responding on WASO $\leq$30 min and SOL $\geq$50% and cognitive control was modestly associated with better responding in terms of WASO $\geq$50% reduction and ENJOY. By comparison, it should be noted that imagery was associated with non-response to treatment for the SOL variables including the combined outcome SOLboth (χ^2=7.28, P=0.022). Although only 17 of the 90 patients reported using imagery (Table 1), 14 of these 17 (82%) were non-responders.

Patients' use of the 10 CBT components, of course, was not mutually exclusive. There are limitations, therefore, to focusing upon bivariate relationships, particularly since multiple comparisons were made. As described above, our primary purpose in conducting these analyses was to reduce the range of possible associations to those meeting our entry criterion ($P<0.15$) for inclusion in a multivariate model. The results of logistic regression analyses for each of these outcome variables is presented in Table 2.

Using stimulus control/sleep restriction but not imagery accounted for a significant amount of explained variance in SOL $\leq$30 min. This equation predicted 100% of responders on this outcome

Table 2
Prediction of treatment response on seven outcome variables following logistic regression analyses

	Log likelihood	Chi-square	df	sig.	Predictors	sig.	*R*	% Correct responders	% Correct non-responders
SOL ≤30 min	55.57	12.35	4	0.015	Stimulus control/sleep restriction	0.039	0.183	100.0	33.3
					Imagery	0.064	−0.145		
SOL ≥50% reduction	60.53	11.25	7	0.128	None	–	–	–	–
SOL both	60.15	9.02	4	0.061	None	–	–	–	–
WASO ≤30 min	58.72	11.53	5	0.042	Stimulus control/ sleep restriction	0.026	0.206	78.8	50.0
					Cognitive restructuring	0.130	0.065		
WASO ≥50% reduction	59.81	9.30	4	0.054	Cognitive control	0.055	0.156	76.7	52.3
					Pre-bedtime routine	0.090	0.112		
WASO both	65.30	8.36	4	0.079	None	–	–	–	–
ENJOY ≥50% increase	46.41	13.54	5	0.019	Cognitive control	0.042	0.189	28.6	91.9
					Altering lifestyle	0.056	0.167		

but only one-third of non-responders. Thus stimulus control and sleep restriction seem particularly important to reduce sleep latency to below clinical threshold levels. Similarly for WASO ≤30 min stimulus control/sleep restriction (20% of variance), combined with the lesser influence of cognitive restructuring (6%), predicted over three-quarters of responders and 50% of non-responders. No significant predictors emerged for SOL ≥50% reduction, however, a near significant equation (P=0.054) for WASO ≥50% reduction suggested that cognitive control and having a pre-bed routine were important, again predicting responding at a higher rate than non-responding. Neither of the combined outcome variables (SOLboth, WASOboth) emerged with significant predictors. In terms of sleep quality, failing to put the day to rest or address lifestyle issues appeared to predict poor response in over 90%, in terms of the variable ENJOY. It should be noted that some aspects of sleep hygiene (bedroom factors), relaxation and thought-blocking techniques did not predict clinical outcome on any variable.

4. Discussion

There are a number of reasons why a patient might use a treatment strategy. These include ease of assimilation, adaptability for and comfort in home practice, perceived relevance and perceived effectiveness. The reporting of treatment implementation of course may be subject to demand

characteristics. Subjects may wish to demonstrate appreciation. However, with a post-treatment interval of 12 months in this study demand characteristics might be expected to play a lesser role since there was no continuing contact with the therapist or the research team. Furthermore, efforts were made to enable patients freely to report which CBT elements they found useful. The fact that there was variability in terms of reported use, implies that this goal was in some measure met.

Taking the group as a whole, relaxation emerged as the most commonly endorsed strategy (74%). Relaxation clearly has high face validity, and it may be particularly memorable to patients. However, consistent with the research literature being somewhat equivocal about its efficacy for clinical populations (Morin et al., 1999), the present analysis revealed that relaxation failed to predict improvement on any of seven clinical outcomes, including a measure of sleep quality. Similarly, sleep hygiene, although commonly used as a precursor to CBT, has not emerged previously as an effective element of treatment. The results of our regression analysis support the conclusion that sleep hygiene has little effect upon clinical outcome, except perhaps those components relevant to lifestyle which may make a modest contribution to qualitative report of sleep.

The American Academy of Sleep Medicine, in its recent Practice Parameters paper, reported stimulus control as the component treatment with the highest level of empirical evidence, recommending it as a 'standard' treatment for insomnia (Chesson et al., 1999). In the present study, the stimulus control/sleep restriction component was found to be the strongest and most consistent predictor of outcome, particularly in terms of identifying treatment responders. Patients who reported using these behavioural procedures were more likely to achieve a final sleep latency of less than 30 min or to spend less than 30 min of wakefulness in bed during the night. Since patients achieving such a sleep pattern may be regarded as similar to the general population of non-complainants, because they would no longer meet diagnostic criteria, it appears that adherence to stimulus control and sleep restriction may be predictive of normative end state functioning (Kendall, Marrs-Garcia, Nath, & Sheldrick, 1999). This has important implications in terms of clinical significance of change, which is the priority in real world settings. Our practice has been to combine stimulus control instructions (after Bootzin, 1972) with sleep restriction (after Spielman, Saskin, & Thorpy, 1987) and to use the term 'sleep scheduling' with patients. Our view is that the successful re-conditioning of strong, positive bed–sleep associations using stimulus control complements improved chronobehavioural functioning (where the emphasis is upon improving sleep efficiency i.e. the ratio of actual sleep time to time in bed) by means of sleep restriction. By default, of course, sleep scheduling also reduces the probability of experiencing negative intrusive thoughts while in bed, raising another potential mode of action.

More explicit cognitive strategies also contributed to outcome according to our analysis. It is interesting that cognitive restructuring had some impact upon intermittent wakefulness (WASO $\leq$30 min) rather than initial insomnia. This is consistent with the results for the other wakefulness measure (WASO $\geq$50% reduction) where better preparation for bed, both mentally using cognitive control and behaviourally by following a pre-bed routine, was modestly predictive of response. It seems possible, therefore, that continuity of sleep is facilitated by the development and maintenance of adaptive beliefs and attitudes. Thought-blocking using articulatory suppression, however, did not add any predictive power to outcome on any variable. The apparently negative effect associated with imagery was unexpected. It seems that the non-use of imagery, perhaps in favour of stimulus control, is preferable and that imagery may have potential to exacerbate sleep-onset problems. In our CBT package subjects are encouraged to choose their own visual image, around

the theme of a pleasant sequence of events, and to integrate it within their relaxation routine. It may be that other instructional sets would prove more beneficial. Finally, we found no clear predictor of positive treatment response on our measure of sleep quality, although failure to implement lifestyle change and to put the day to rest may impair sleep enjoyment. Since qualitative data comprise an important perspective on sleep assessment and management, and perhaps upon the persistence of complaining behaviour, further work is required to ascertain how such qualitative outcomes may be improved.

It is beyond the scope of our data to offer any explanation as to the mechanism of psychological treatment effects. Besides, there are many limitations methodologically to the type of post-hoc analysis reported here. However, it is tentatively suggested that CBT which prioritises the adoption of sleep scheduling and cognitive restructuring methods may be more likely to produce clinically meaningful results, than broadly educational approaches emphasising sleep hygiene, relaxation or thought-blocking methods. This conclusion appears reasonably consistent with the outcome literature. Inevitably, further work is required to validate reports of home practice since these do not necessarily confirm actual implementation.

Acknowledgements

This research was supported by grants from the Chief Scientist Office, Scottish Office Department of Health and Ayrshire and Arran Health Board.

References

American Sleep Disorders Association (1990). *International classification of sleep disorders, diagnostic and coding manual.* Rochester, MA: American Sleep Disorders Association.

Bootzin, R. R. (1972). A stimulus control treatment for insomnia. Proceedings of the American Psychological Association, 395–396.

Chesson, A. L., Anderson, W. MCD., Littner, M., Davila, D., Hartse, K., Johnson, S., Wise, M., & Rafecas, J. (1999). Practice parameters for the nonpharmacologic treatment of chronic insomnia. An American Academy of Sleep Medicine report: Standards of Practice Committee of the AASM. *Sleep, 22*, 1128–1133.

Edinger, J. D., & Wohlgemuth, W. (1999). The significance and management of persistent primary insomnia: the past, present and future of behavioral insomnia therapies. *Sleep Medicine Reviews, 3*, 101–118.

Espie, C. A., Lindsay, W. R., Brooks, D. N., Hood, E. M., & Turvey, T. (1989). A controlled comparative investigation of psychological treatments for chronic sleep-onset insomnia. *Behaviour Research and Therapy, 27*, 79–88.

Espie, C. A., Brindle, S., Tessier, S., Dawson, S., Hepburn, T., McLellan, A., Renwick, E., Steele, S., & McColl, J. (1998). Supervised cognitive-behaviour therapy for insomnia in general medical practice—preliminary results from the West of Scotland programme. In E. Sanavio, *Behavior and cognitive therapy today: essays in honor of Hans J. Eysenck* (pp. 67–75). Oxford: Elsevier.

Espie, C. A., Inglis, S. J., Tessier, S., & Harvey, L. (2001). The clinical effectiveness of cognitive behaviour therapy in the treatment of chronic insomnia: Implementation and evaluation of a Sleep Clinic in general medical practice. *Behaviour Research and Therapy, 39*, 45–60.

Espie, C. A., Inglis, S. J., & Harvey, L. (2001). Predicting clinically significant response to cognitive behavior therapy (CBT) for chronic insomnia in general practice: analyses of outcome data at 12 months post-treatment. *Journal of Consulting and Clinical Psychology, 69*, 58–66.

Hosmer, D. W., & Lemeshow, S. (1989). *Applied logistic regression.* New York: John Wiley and Sons.

Kendall, P. C., Marrs-Garcia, A., Nath, S. R., & Sheldrick, R. C. (1999). Normative comparisons for the evaluation of clinical significance. *Journal of Consulting and Clinical Psychology*, *67*, 285–299.

Lichstein, K. L., & Hoelscher, T. J. (1986). A device for unobtrusive surveillance of home relaxation practice. *Behavior Modification*, *10*, 219–233.

Morin, C. M., Colecchi, C., Stone, J., Sood, R., & Brink, D. (1999). Behavioral and pharmacological therapies for late-life insomnia: a randomised controlled trial. *Journal of the American Medical Association*, *281*, 991–999.

Morin, C. M., Hauri, P. J., Espie, C. A., Spielman, A., Buysse, D. J., & Bootzin, R. R. (1999). Nonpharmacologic treatment of chronic insomnia: an American Academy of Sleep Medicine review. *Sleep*, *22*, 1134–1156.

Morin, C. M., Gaulier, B., Barry, T., & Kowatch, R. A. (1992). Patients' acceptance of psychological and pharmacological therapies for insomnia. *Sleep*, *15*, 302–305.

Schoicket, S. L., Bertelson, A. D., & Lacks, P. (1988). Is sleep hygiene a sufficient treatment for sleep-maintenance insomnia? *Behavior Therapy*, *19*, 183–190.

Tabachnick, B. D., & Fidell, L. S. (1996). *Using multivariate statistics.* New York: Harper Collins.

Turner, R. M., & Ascher, L. M. (1979). A controlled comparison of progressive relaxation, stimulus control and paradoxical intention therapies for insomnia. *Journal of Consulting and Clinical Psychology*, *47*, 500–508.

Spielman, A. G., Saskin, P., & Thorpy, M. J. (1987). Treatment of chronic insomnia by restriction of time in bed. *Sleep*, *10*, 45–56.

Behavioural and Cognitive Psychotherapy, 2003, 31, 313–324
Printed in the United Kingdom DOI: 10.1017/S1352465803003060

INITIAL INSOMNIA AND PARADOXICAL INTENTION: AN EXPERIMENTAL INVESTIGATION OF PUTATIVE MECHANISMS USING SUBJECTIVE AND ACTIGRAPHIC MEASUREMENT OF SLEEP

Niall M. Broomfield and Colin A. Espie

University of Glasgow, Scotland, UK

Abstract Paradoxical Intention (PI) is a cognitive treatment approach for sleep-onset insomnia. It is thought to operate by eliminating voluntary sleep effort, thereby ameliorating sleep performance anxiety, an aroused state incompatible with sleep. However, this remains untested. Moreover, few PI studies have employed objective sleep measures. The present study therefore examined the effect of PI on sleep effort, sleep anxiety and *both* objective and subjective sleep. Following a seven-night baseline, 34 sleep-onset insomniacs were randomly allocated to 14 nights of PI, or to a control (no PI) condition. Consistent with the performance anxiety model, participants allocated to PI, relative to controls, showed a significant reduction in sleep effort, and sleep performance anxiety. Sleep-onset latency (SOL) differences between PI participants and controls using an objective sleep measure were not observed, although an underlying trend for significantly lowered subjective SOL amongst PI participants was demonstrated. This may relate to actigraphic insensitivity, or more probably confirms recent suggestions that insomniacs readily overestimate sleep deficit, due to excessive anxiety about sleep. Together, results help determine putative mechanisms underlying PI, have important implications for the clinical application of PI, and emphasize the need for further PI research within an experimental cognitive framework.

Keywords: Insomnia, paradoxical intention, cognitive-behaviour therapy, performance anxiety, actigraphy.

Introduction

Evidence has converged suggesting pre-sleep cognitive activity is a key maintaining factor in sleep-onset insomnia (Espie, 2002; Harvey, 2002). Despite this, research examining stand-alone cognitive insomnia interventions remains rare. One exception to this is Paradoxical Intention (PI). Single case (Ascher, 1975; Ascher & Efran, 1978; Espie & Linday, 1985) and randomized-controlled studies (Ascher & Turner, 1979, 1980) support the utility of PI in the management of sleep-onset insomnia, and its equivalence to stimulus control and relaxation (Espie, Lindsay, Brooks, Hood, & Turvey, 1989; Ladoucer & Gros-Loius, 1986; Turner & Ascher, 1979). Indeed, PI is now regarded as a ''probably efficacious'' insomnia

Reprint requests to Niall Broomfield, Department of Psychological Medicine, Gartnavel Royal Hospital, 1055 Great Western Road, Glasgow G12 0XH, Scotland, UK. E-mail: nmb2v@clinmed.gla.ac.uk

intervention according to APA criteria (Chesson, Anderson, Littner, Davila, & Hartse, 1999; Morin et al., 1999).

Despite these outcome data, and its now routine use within multicomponent CBT (e.g. Espie, Inglis, Tessier, & Harvey, 2001), the mechanisms underlying PI remain unclear. PI is a cognitive approach, which requires poor sleepers give up voluntary effort to control sleep. The treatment achieves this by encouraging insomniacs to relax with the lights out at bedtime, keeping their eyes open. Paradoxically, the likelihood of staying awake is reduced by encouragement to do so (Espie & Lindsay, 1985).

Sleep is a behaviour, which cannot be placed under full voluntary control. One hypothesis therefore is that direct attempts to control sleep will fail, because sleep effort inhibits relaxation and sets up performance anxiety, an aroused state incompatible with sleep. Consistent with this, Ascher and Turner (1979) have argued that by eliminating voluntary sleep effort, PI minimizes sleep performance anxiety, thereby promoting rapid sleep-onset. Similarly, Espie (2002) has suggested that by diverting attention away from sleep performance, PI facilitates cognitive/affective de-arousal and promotes sleep. To date, however, these performance anxiety conceptualizations remain untested. This study, therefore, is *not* an evaluation of the efficacy of PI. Rather, it aimed to clarify whether PI institutes sleep change via sleep effort/performance anxiety reduction.

There also, at present, remains a marked lack of PI research using objective sleep measures. This is problematic. Insomniacs' self-report sleep data can be unreliable (Carskadon et al., 1976), and objective and subjective sleep measures may reflect differing response systems (Wicklow & Espie, 2000). To date, only one PI study has employed objective sleep measurement (Ott, Levine, & Ascher, 1983), and the method employed only estimated sleep-onset latency (SOL) objectively to within five minutes. A second aim of the present study was therefore to examine and compare objective (actigraphy) and subjective (self-report diary) sleep outcome following brief (14 night) PI using a reliable sleep measurement system.

A reliable and minimally intrusive objective sleep measure is the actigraph – a small wrist attachment that records the wearer's movements. There is recognition that movement is a good predictor of wakefulness, whilst lack of movement is a good predictor of sleep (American Sleep Disorders Association, 1995; Mullaney, Kripke, & Messin, 1980). Moreover, actigraphic measures correlate highly with polysomnographic (PSG) data for sleep duration and total wake time (e.g. Sadeh, Hauri, Kripke, & Lavie, 1995; Mullaney et al., 1980).

To summarize then, a study was conducted examining the performance anxiety model of PI. Sleep-onset insomniacs were randomly assigned to two weeks of PI, or a control (no PI) condition, following a one-week baseline. Voluntary sleep effort and sleep anxiety data were collected, alongside actigraphic and subjective sleep data. A One Between (Condition: Paradoxical Intention [PI], Control) and One Within factor (Time: Baseline, Week One, Week Two) design was employed. Relative to the control condition, it was predicted allocation to PI would (i) reduce sleep effort after Weeks One and Two; (ii) reduce sleep performance anxiety after Weeks One and Two; (iii) reduce objective and subjective SOL after Weeks One and Two; (iv) and raise objective and subjective sleep efficiency after Weeks One and Two. Importantly, the research was *not* designed as a treatment study, rather as an experimental examination of putative mechanisms underlying PI, as well as objective/subjective sleep outcome following the procedure.

Method

Participants

Participants were recruited using the University e-mail system and via notices placed locally. The Health Authority and University granted ethical approval. Prior to participation potential participants completed screening questionnaires assessing sleep (Pittsburgh Sleep Quality Index [PSQI] – Buysse, Reynolds, Monk, Berman, & Kupfer, 1989; Sleep History Questionnaire – Morin, 1993), anxiety (Spielberger Trait Anxiety Inventory – STAI; Spielberger, Gorsuch, & Lushene, 1970), worry (Penn State Worry Questionnaire – PSWQ; Meyer, Miller, Metzger, & Borkovec, 1990) and depression (Beck Depression Inventory – BDI; Beck, Steer, & Brown, 1996).

Respondents were included if they were between 16 and 65 years, complained of clinically significant problems falling asleep according to the revised version of the International Classification of Sleep Disorders (American Sleep Disorder Association, 1997; i.e. SOL greater than 30 min at least 4 nights per week, with or without disruption to other sleep variables), and scored in excess of 5 on the PSQI, the recognized cut-off for identifying clinically significant sleep disturbance (Buysse et al., 1989). Participants were excluded if they experienced intermittent awakenings without sleep-onset difficulties, were receiving treatment for sleeping difficulties, or were suffering any medical or psychopathological disorder impacting on sleep.

Forty-six participants were identified, all meeting criteria. Thirty-four completed the experiment (74%; mean age 25.2 years, average sleep disturbance 6.35 years). A further seven failed to attend the initial meeting, three withdrew during baseline, and two were excluded due to unreliable diary completion.

Measures

A daily sleep diary (Espie, 1991), completed upon rising for 21 days, provided measures of subjective SOL and sleep efficiency (based on time to bed, rise time, time to fall asleep, and total sleep time responses). Effort to sleep data were recorded in the diary using a 7-point scale (‘‘I tried hard to get to sleep last night’’; anchor points 0 ‘‘not at all’’, 6 ‘‘very much’’).

Two self-report scales measured sleep-related performance anxiety: the Sleep Anxiety Scale (SAS; Fogle & Dyall, 1983, see p. 26 for internal consistency data); and a specially developed scale, the Sleep Performance Anxiety Questionnaire (SPAQ). The latter comprised seven components of dysfunctional sleep monitoring (sleep effort, sleep control, sleep avoidance, bedtime worry, performance failure, anticipatory anxiety, and daytime worry). Piloting indicated the SPAQ readily distinguished good ($N = 4$; mean = 7.75, $SD = 0.96$) from poor ($N = 4$; mean = 15.50, $SD = 3.69$) sleepers. Cronbach’s alpha data for both scales are presented in the results.

Wrist actigraphic recording using the ‘‘Actiwatch’’ (Model AW2; Cambridge Neurotechnology Ltd) provided objective estimates of SOL and sleep efficiency. As noted, actigraphic measures correlate highly with polysomnographic (PSG) data for sleep duration and total wake time (Sadeh et al., 1995; Mullaney et al., 1980), and movement is a good predictor of wakefulness, whilst lack of movement is a good predictor of sleep (American Sleep Disorders Association, 1995; Mullaney et al., 1980).

Participants wore the actigraph continuously on their non-dominant hand except during wet activities. An event marker was depressed at lights out, and upon rising. Epoch length was set at 1 min. (Wicklow & Espie, 2000), with ''sleep'' or ''wakefulness'' determined by the program's algorithm.

Procedure

Following a telephone interview, participants were sent screening questionnaires (see participants section), an information sheet, and an informed consent slip. These were completed and returned by post. Participants then received a sleep diary and wrist actigraph, and were told the next seven days was a baseline measurement week.

Seven days later, participants completed the two sleep anxiety scales (Baseline), and were issued with further copies to be completed after seven days (Week One). Participants were then randomly allocated to experimental condition.

PI participants were introduced to the rationale of PI, and instructed, at lights out, to stay awake for as long as possible by keeping their eyes open. The need to resist sleep-onset gently but persistently in an environment conducive to sleep was emphasized. The use of active methods to stay awake (e.g. reading, physical movement) was discouraged. Patient expectations can influence response to PI (Epsie & Lindsay, 1985). In an attempt to control for this, half of PI participants were told to expect immediate sleep improvement (positive demand), whereas half were told to expect sleep improvement only at Week Two (counter demand). Control participants were told to continue wearing their actiwatch, and to continue completing their sleep diary.

After 14 nights, all participants returned their diaries and actigraph, and completed two final sleep anxiety scales (Week Two), and a compliance-rating sheet. They were then debriefed and thanked, and issued with the ''Good Sleep Guide'', a leaflet describing behavioural advice for home practice prepared by the second author (National Medical Advisory Committee, 1993).

Results

Participant characteristics

Participants had a mean age of 25.2 years, and mean sleep disturbance duration of 6.35 years. PI participants and controls were equitable in terms of gender ($p > .10$, NS), and did not differ on age, duration of sleep problem, trait anxiety, worry, depression or sleep quality (all $p > .10$; see Table 1 for data).

Compliance with experimental instructions

Participants reported correctly following experimental instructions (diary completion, actiwatch use) on mean = 19.2 nights. Mean compliance rating (scale 0–6) was 5.03, SD = 0.79. Non-compliance included forgetting to press the actiwatch, or to replace the actiwatch after wet activities. This was unusual.

Table 1. Demographic and questionnaire data, and sleep anxiety, sleep effort, objective and subjective sleep-onset latency (mins) and sleep efficiency (%) data at Baseline (B), Week One (W1) and Week Two (W2), for paradoxical intention (PI) and control (C) participants

	Age	Females: Males	Insomnia duration (years)	STAI-T	PSWQ	BDI	PSQI
Condition							
PI	26.00	9:8	5.93	37.35	40.58	7.94	11.18
C	24.35	10:7	6.77	37.29	40.11	9.88	10.59

		SAS			SPAQ			Sleep effort		
		B	W1	W2	B	W1	W2	B	W1	W2
PI	*M*	16.18	13.59	12.29	15.41	12.53	11.41	2.33	1.11	1.10
	SD	4.05	3.78	4.03	2.87	3.89	3.61	1.37	1.20	1.30
C	*M*	15.76	16.35	16.05	14.88	15.18	14.76	2.28	2.21	2.14
	SD	3.83	3.12	3.60	2.71	2.43	2.51	1.44	1.12	1.28

		Objective SOL			Objective efficiency			Subjective SOL			Subjective efficiency		
		B	W1	W2	B	W1	W2	B	W1	W2	B	W1	W2
PI	*M*	29.92	27.23	24.47	80.87	81.84	80.65	65.74	41.76	38.24	80.87	86.10	85.79
	SD	17.16	15.06	17.20	6.67	7.76	7.57	33.97	19.62	25.28	13.49	11.85	11.07
C	*M*	26.62	25.19	25.03	78.93	79.80	80.64	54.67	58.82	55.33	80.04	82.46	82.82
	SD	21.83	16.37	16.49	7.93	7.34	8.95	25.67	32.98	29.16	11.85	10.32	12.40

Using PI

All 17 PI participants said they lay awake with their eyes open when using PI. None used active methods (reading, television) to stay awake.

Outcome variables

All data were first examined for kurtosis and skewness and fell within acceptable limits. Data also showed homogeneous variance, following Hartley's Fmax test (Winer, 1971). Analyses then relied on a Two (Condition: Paradoxical Intention vs. Control) by Three (Time: Baseline vs. Week One vs. Week Two) ANOVA design based on weekly means. Alpha level was set at .05, two-tailed, throughout. Means and standard deviations are presented in Table 1.

Sleep effort

ANOVA revealed a main effect of Time ($F[2, 64] = 9.05$, $p = .0001$), and a Time × Condition interaction ($F[2, 64] = 6.37$, $p = .003$). In order to clarify this, Bonferroni corrected simple main effects analyses for Condition were completed across the Time variable (critical p value = .017; Keppel, 1993). As is evident in Figure 1 panel A, this indicated relative to controls, PI participants showed significantly lower sleep effort at Week One ($F[1, 33] = 7.72$, $p = .009$; critical p value = .017) and near significant lower effort at Week Two ($F[1, 33] = 5.55$, $p = .025$; critical p value = .017). No differences were observed at Baseline ($F[1, 33] = 0.01$, $p > .1$, NS).

Sleep performance anxiety

Separate ANOVAs were run for the two performance anxiety scales employed, based on participant scores at Baseline, Week One and Week Two.

Sleep Anxiety Scale (SAS). ANOVA revealed a main effect of Time ($F[2, 64] = 6.69$, $p = .002$), and an interaction effect of Time × Condition ($F[2, 64] = 9.84$, $p = .0001$). As is evident in Figure 1 panel B, simple main effects indicated PI participants displayed significantly lower sleep anxiety at Week Two ($F[1, 33] = 8.26$, $p = .007$), and a near significant trend for lower sleep anxiety at Week One ($F[1, 33] = 5.41$, $p = .026$; critical p value = .017). No differences were observed at Baseline ($F[1, 33] = 0.09$, $p > .1$, NS).

In order to examine the internal consistency of scale data, baseline item scores across participants were subjected to appraisal using Cronbach's alpha. Overall alpha coefficient was 0.86, with range of alpha values, if item deleted, of 0.83–0.87. The mean corrected item-total correlation was 0.62.

Sleep Performance Anxiety Questionnaire (SPAQ). Analysis revealed a main effect of Time ($F[2, 64] = 15.68$, $p = .0001$), and a Time × Condition interaction ($F[2, 64] = 15.48$, $p = .0001$), displayed in Figure 1 panel C. As is evident, simple main effects indicated PI participants showed significantly lower sleep anxiety at Week Two ($F[1, 33] = 9.89$, $p = .004$), and a trend for lower sleep anxiety at Week One ($F[1, 33] = 5.66$, $p = .023$). No differences were observed at Baseline ($F[1, 33] = 0.30$, $p > .1$, NS). Overall alpha coefficient

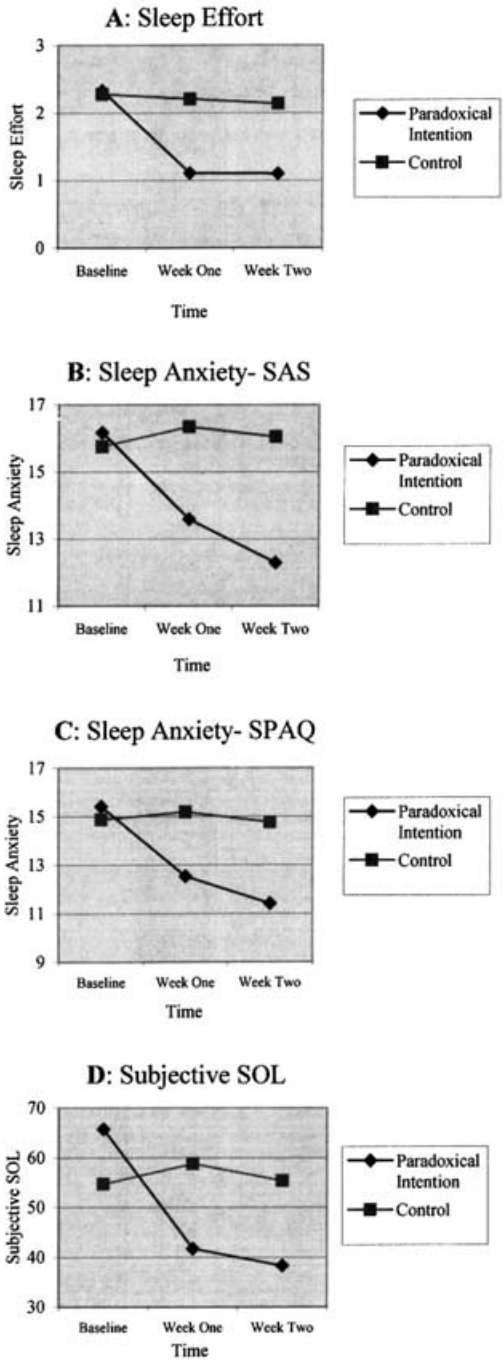


Figure 1. Mean sleep effort, sleep anxiety and subjective SOL for PI and control participants, as a function of Time

for the SPAQ was 0.70, with the range of alpha values, if item deleted, of 0.63–0.73. The mean corrected item total correlation was 0.42.

Finally, Pearson correlation coefficients were computed across participants' baseline scores on the SAS and SPAQ. This indicated correlation was ''moderate'' ($r = 0.62$, $p < .01$), representing 38.5% shared variance.

Sleep

Two participants' actigraph data were lost due to faulty equipment. Analyses were therefore based on 16 participants per condition.

Counterdemand instructions. A series of Two (Instructions: Counterdemand vs. Positive Demand) by Three (Time: Baseline vs. Week One vs. Week Two) ANOVAs for PI participants *only*, on objective and subjective SOL and sleep efficiency, revealed no significant Instruction × Time interaction effects (all $p > .1$, NS; see Table 2). Primary sleep analyses therefore compared PI participants with controls.

Objective sleep (actigraphy). ANOVA for objective SOL revealed non-significant main effects of Time ($F[2, 60] = 0.48$, $p > .1$, NS) and Condition ($F[1, 30] = 0.06$, $p > .1$, NS). The Time × Condition interaction was also non-significant ($F[2, 60] = 0.13$, $p > .1$, NS). Objective SOL remained at approximately 25–30 minutes across condition (see Table 1).

Similarly, ANOVA for objective sleep efficiency data failed to reveal any significant effects of Time ($F[2, 60] = 0.53$, $p > .1$, NS), Condition ($F[1, 30] = 0.67$, $p > .1$, NS), or Time × Condition ($F[2, 60] = 0.19$, $p > .1$, NS). Objective efficiency remained at approximately 80% (see Table 1).

Subjective sleep (diary). ANOVA revealed a main effect of Time ($F[2, 64] = 6.72$, $p = .002$), and an interaction of Time × Condition ($F[2, 64] = 9.16$, $p = .0001$), displayed in Figure 1 panel D. Simple main effects analyses could be indicative of an underlying trend for lower SOL amongst PI participants, relative to controls, at Weeks One ($F[1, 33] = 3.36$,

Table 2. Mean and standard deviation objective and subjective sleep-onset latency (mins) and sleep efficiency (%) at Baseline (B), Week One (W1) and Week Two (W2), for counterdemand (C) and positive demand (P) instructions

	Objective				Subjective			
	SOL		Efficiency		SOL		Efficiency	
Condition	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*
BC	20.73	10.52	78.31	16.96	62.03	20.97	78.30	16.96
BP	37.10	19.15	83.15	10.00	69.03	39.96	83.15	9.99
W1 C	22.41	13.71	81.82	16.04	46.66	18.41	81.82	16.04
W1 P	28.48	18.11	89.92	4.61	37.41	20.69	89.92	4.60
W2 C	19.32	10.35	82.53	14.53	39.41	28.01	82.53	14.53
W2 P	29.61	21.59	88.70	6.36	37.19	24.26	88.69	6.35

p = .076) and Two (F[1, 33] = 3.34, p = .077). However, test statistics on small samples can be unstable. No differences were observed at Baseline (F[1, 33] = 1.15, p > .1, NS).

Allocation to PI resulted in a 41.85% reduction in SOL, compared to a 1.21% increase amongst controls (see Table 1). Treatment effect size was ''moderate'' (d = 0.61; Cohen, 1988). 70.6% of PI participants reported PI helped them get to sleep quicker.

Analysis of subjective sleep efficiency data revealed a main effect of Time (F[2, 64] = 6.65, p = .024), although the Time × Condition interaction failed to reach significance (F[2, 64] = 0.73, p > .1, NS).

The relationship between objective and subjective sleep measures. As inspection of Table 1 reveals actigraphic SOL at baseline was 29.92 minutes for PI participants, and 26.62 minutes for controls. At Week Two, these scores were unchanged for both PI participants (mean = 24.47 minutes) and controls (mean = 25.03 minutes). No significant objective SOL change following PI was therefore observed.

Subjective SOL scores at baseline were much higher than objective SOL scores for PI participants (mean = 65.74 minutes) and controls (mean = 54.67 minutes). This suggests all participants *overestimated* SOL *relative to* actigraphic assessment as an objective criterion at baseline. The discrepancy between subjective (mean = 38.24 minutes) and objective (mean = 24.47 minutes) SOL scores reduced for PI participants by Week Two. Control subjects showed no discrepancy reduction (mean subjective SOL = 55.33 minutes, mean objective SOL = 25.03 minutes). This suggests allocation to PI reduced participants' tendency to overestimate subjective SOL.

Pearson correlation coefficients across all participants examined the association between objective and subjective SOL, and objective and subjective sleep efficiency (baseline – week 2). Correlations were ''low'' for SOL (r = 0.25), and ''low'' for sleep efficiency (r = 0.17).

The association between sleep effort, sleep performance anxiety and subjective sleep onset latency

To explore whether reduced sleep effort or sleep anxiety best predicted reduced *subjective* SOL, change scores for each variable (baseline – week 2) were computed and subjected to Pearson correlational analyses. SOL change was signficantly associated with sleep anxiety change (SAS: r = 0.36, p =.035; SPAQ: r = 0.43, p = .01), and with effort change (r = 0.56, p = .001). Comparing explained variance (SAS r^2 = 0.13; SPAQ r^2 = 0.18; Effort r^2 = 0.31), the association between SOL change and effort change is strongest.

Since baseline sleep anxiety was significantly associated with sleep effort (SAS; r = 0.56, p = .001), (SPAQ; r = 0.58, p = .0001), partial correlations were computed for PI participants *only* to reveal the individual effect of effort change, and anxiety change (both scales), on subjective SOL change. Effort change significantly correlated with SOL change when sleep anxiety was partialled out (SAS: r_p = 0.48, p = .029; SPAQ: r_p = 0.44, p = .045). In contrast, when effort change was partialled out, neither measure of sleep anxiety was significantly associated with SOL change (SAS: r_p = –0.2, p > .1, NS; SPAQ: r_p = –0.1, p > .1, NS). This suggests SOL reduction amongst PI participants was most strongly associated with sleep effort reduction.

Discussion

The present study has several limitations. The sample employed was non-treatment seeking, and the study timescale was short, only 21 nights. The effect of PI in the longer term is therefore unclear. The design lacked a credible but otherwise inert control treatment. So the effect of non-specific factors on the findings is unknown. Sample size was small, which underpowered the counterdemand-positive demand comparison, and the measure of sleep effort employed was not validated prior to the study.

Relative to controls, PI participants showed reduced sleep effort, reduced sleep performance anxiety and reduced SOL measured subjectively. SOL and anxiety change were significantly correlated, although SOL change amongst PI participants was most strongly associated with sleep effort change. Together, therefore, findings support a mediational performance anxiety model of PI. In other words, sleep effort and sleep anxiety are mechanisms of change in PI (cf. Ascher & Turner, 1979), and inhibitory to normal sleep function (cf. Espie, 2002).

Findings are also consistent with Wegner's model of ironic mental control (Wegner, 1994). As noted, there was a strong association between reduced sleep effort and reduced subjective SOL amongst PI participants. Thus, for insomniacs high in sleep anxiety, SOL was reduced when attempts were made to stay awake. This raises the possibility that, under high cognitive load (i.e. sleep performance anxiety), with the intentional operating system undermined, PI caused the ironic monitoring process to detect restful cognitions, leading to a perception of faster sleep onset (cf. Ansfield, Wegner, & Bowser, 1996; Wegner, 1994).

As well as supporting current theoretical conceptualizations of PI, findings may have important clinical implications. If, as the data suggest, PI institutes sleep improvement by reducing sleep effort and sleep anxiety, the approach may prove particularly beneficial to insomniacs high in these variables at assessment. There is already some evidence of a link between elevated performance anxiety and response to PI in the social anxiety literature (Ascher & Schotte, 1999). Research should now clarify more closely the association between pre-treatment sleep effort, sleep anxiety, and subsequent response to PI. Clinical assessment of these constructs is certainly feasible, and if elevated sleep effort/anxiety does predict outcome, this raises the prospect of routine screenings for stand-alone PI.

Turning to the sleep data, recognizing the lack of PI research employing objective measures, the present study obtained actigraphic *and* subjective sleep data. Contrary to hypotheses, no significant objective SOL reduction or sleep efficiency increase was observed amongst PI participants, relative to controls. The subjective sleep data did, however, indicate a marginal trend for reduced SOL amongst PI participants, and the treatment effect size was ''moderate'' (cf. Cohen, 1988). One interpretation of this may relate to actigraphic measurement error. Although actigraphic measures of sleep duration and total wake time correlate highly with Polysomnography (PSG) data (Mullaney et al., 1980; Sadeh et al., 1995), lower agreement rates for SOL have been reported (Blood, Sack, Percy, & Pen, 1997; Hauri & Wisbey, 1992). The accuracy of actigraphy in distinguishing sleep from wakefulness has also been questioned (Pollak, Tryon, Nagaraja, & Dzwonczyk, 2001; Verbeek, Klip, & Declerck, 2001). Perhaps quiet wakefulness in the pre-sleep phase was coded actigraphically as ''sleep'', lowering objective SOL scores, relative to subjective values (see Table 1), washing out any possible objective SOL effect.

Alternatively, it is possible PI shifted participants' perceptions of their sleep deficit.

Lower objective relative to subjective SOL scores were observed amongst *both* participant groups at baseline (see Table 1). Interestingly, this apparent overestimation of SOL was *only* observed amongst controls at Week 2. This raises the intriguing possibility that PI reduced participants' tendency to overestimate subjective SOL relative to objective criterion. Harvey (2002) has suggested that excessive sleep anxiety, and an attentional bias for sleep-related threat triggered by this anxiety, causes insomniacs to overestimate their sleep deficits. Perhaps by lowering sleep anxiety, PI reduced participants' tendency to overestimate time to sleep (cf. Harvey, 2002). This alteration in perception may also have involved reduced activation of metacognitive sleep beliefs (e.g. ''thinking about sleep means I'm a poor sleeper''), as the less sleep anxious PI participants experienced fewer sleep-related intrusions (Wells, 2001). Further research within an experimental cognitive framework will be needed to determine whether the present findings are indeed due to a shift in PI participants' estimations of SOL or arose due to actigraphic insensitivity.

References

American Sleep Disorder Association (1997). *The international classification of sleep disorders: Diagnostic and coding manual* (Rev.). Rochester, USA: ASDA.

American Sleep Disorders Association (1995). Practice parameters for the use of actigraphy in the clinical assessment of sleep disorders. *Sleep*, *18*, 285–287.

Ansfield, M. E., Wegner, D. M., & Bowser, R. (1996). Ironic effects of sleep urgency. *Behaviour Research and Therapy*, *34*, 523–531.

Ascher, L. M. (1975). *Paradoxical intention as a component in the behavioural treatment of sleep-onset insomnia: A case study*. Paper presented at the Association for the Advancement of Behaviour Therapy, San Francisco. December.

Ascher, L. M., & Efran, J. S. (1978). The use of paradoxical intention in a behavioural program for sleep-onset insomnia. *Journal of Consulting and Clinical Psychology*, *8*, 547–550.

Ascher, L. M., & Schotte, D. E. (1999). Paradoxical intention and recursive anxiety. *Journal of Behaviour Therapy and Experimental Psychiatry*, *30*, 71–79.

Ascher, L. M., & Turner, R. M. (1979). Paradoxical intention and insomnia: An experimental investigation. *Behaviour Research and Therapy*, *17*, 408–411.

Ascher, L. M., & Turner, R. M. (1980). A comparison of two methods for the administration of paradoxical intention. *Behaviour Research and Therapy*, *18*, 121–126.

Beck, A. T., Steer, R. A., & Brown, G. K. (1996). *Beck Depression Inventory*. New York: The Psychological Corporation.

Blood, M. L., Sack, R. L., Percy, D. C., & Pen, J. C. (1997). A comparison of sleep detection by wrist actigraphy, behavioural response and polysomnography. *Sleep*, *20*, 388–395.

Buysse, D. J., Reynolds, C. F., Monk, T. H., Berman, S. R., & Kupfer, D. J. (1989). The Pittsburgh Sleep Quality Index: A new instrument for psychiatric practice and research. *Psychiatry Research*, *28*, 193–213.

Carskadon, M. A., Dement, W. C., Mitler, M. M., Guilleminault, C., Zarcone, V. P., & Speigel, R. (1976). Self reports versus sleep laboratory findings in 122 drug-free subjects with complaints of chronic insomnia. *American Journal of Psychiatry*, *133*, 1382–1387.

Chesson, A. L., Anderson, W. M., Littner, M., Davila, D., & Hartse, K. (1999). Practice parameters for the non-pharmacologic treatment of chronic insomnia. *Sleep*, *22*, 1128–1133.

Cohen, J. (1988). *Statistical power analysis for the behavioural sciences*. Hillsdale, New Jersey: Erlbaum.

Espie, C. A. (1991). *The psychological treatment of insomnia*. Chichester: John Wiley.

Espie, C. A. (2002). Insomnia: Conceptual issues in the development, persistence, and treatment of sleep disorder in adults. *Annual Review of Psychology*, *53*, 215–243.

Espie, C.A., Inglis, S. J., Tessier, S., & Harvey, L. (2001). The clinical effectiveness of cognitive behaviour therapy for chronic insomnia: Implementation and evaluation of a sleep clinic in general medical practice. *Behaviour Research and Therapy*, *39*, 45–60.

Espie, C. A., & Lindsay, W. R. (1985). Paradoxical intention in the treatment of chronic insomnia: Six case studies illustrating variability in therapeutic response. *Behaviour Research and Therapy*, *23*, 703–709.

Espie, C. A., Lindsay, W. R., Brooks, N. D., Hood, E. H., & Turvey, T. (1989). A controlled comparative investigation of psychological treatments for chronic sleep-onset insomnia. *Behaviour Research and Therapy*, *27*, 79–88.

Fogle, D. O., & Dyall, J. A. (1983). Paradoxical giving up and the reduction of sleep performance anxiety in chronic insomniacs. *Psychotherapy: Theory, Research and Practice*, *20*, 21–30.

Harvey, A. G. (2002). A cognitive model of insomnia. *Behaviour Research and Therapy*, *40*, 869–893.

Hauri, P. J., & Wisbey, J. (1992). Wrist actigraphy in insomnia. *Sleep*, *15*, 293–301.

Keppel, G. (1993). *Design and statistical analysis*. New Jersey: Prentice Hall.

Ladouceur, R., & Gros-Luis, Y. (1986). Paradoxical intention versus stimulus control in the treatment of severe insomnia. *Journal of Behaviour Therapy and Experimental Psychiatry*, *17*, 267–269.

Meyer, T. J., Miller, M. L., Metzger, R. L., & Borkovec, T. D. (1990). Development and validation of the Penn State Worry Questionnaire. *Behaviour Research and Therapy*, *28*, 486–496.

Morin, C. M. (1993). *Insomnia: Psychological assessment and management*. New York: Guilford Press.

Morin, C. M., Hauri, P. J., Espie, C., Spielman, A., Buysse, D. J. & Bootzin, R. (1999). Nonpharmacologic treatment of chronic insomnia. *Sleep*, *22*, 1–25.

Mullaney, D. J., Kripke, D. F., & Messin, S. (1980). Wrist actigraphic estimation of sleep time. *Sleep*, *3*, 83–92.

National Medical Advisory Committee (1993). The Good Sleep Guide. In *Management of anxiety and insomnia*. London: HMSO.

Ott, B. D., Levine, B. A., & Ascher, L. M. (1983). Manipulating the explicit demand of paradoxical intention instructions. *Behavioural Psychotherapy*, *11*, 25–35.

Pollak, C. P., Tryon, W. W., Nagaraja, H., & Dzwonczyk, R. (2001). How accurately does actigraphy identify the states of sleep and wakefulness? *Sleep*, *24*, 957–965.

Sadeh, A., Hauri, P. J., Kripke, D. F., & Lavie, P. (1995). The role of actigraphy in the evaluation of sleep disorders. *Sleep*, *18*, 288–302.

Speilberger, C. D., Gorsuch, R. L., & Lushene, R. E. (1970). *Manual for the state-trait anxiety inventory*. Palo Alto, California: Consulting Psychologists Press.

Turner, R. M., & Ascher, L. M. (1979). Controlled comparison of progressive relaxation, stimulus control, and paradoxical intention therapies for insomnia. *Journal of Consulting and Clinical Psychology*, *47*, 500–508.

Verbeek, I., Klip, E. C., & Declerck, A. C. (2001). The use of actigraphy revised: The value for clinical practice in insomnia. *Perceptual and Motor Skills*, *92*, 852–856.

Wegner, D. M. (1994). Ironic processes of mental control. *Psychological Review*, *101*, 34–52.

Wicklow, A., & Espie, C. A. (2000). Intrusive thoughts and their relationship to a actigraphic measurement of sleep: Towards a cognitive model of insomnia. *Behaviour Research and Therapy*, *38*, 679–693.

Wells, A. (2001). *Emotional disorders and metacognition: Innovative cognitive therapy*. Chichester: Wiley.

Winer, B. J. (1971). *Statistical principles in experimental design* (2nd Ed.). New York: McGraw Hill.

REVIEW

Psychological And Behavioral Treatment Of Insomnia:Update Of The Recent Evidence (1998-2004)

[1]Charles M. Morin, PhD, (Chair); [2]Richard R. Bootzin, PhD; [3]Daniel J. Buysse, MD; [4]Jack D. Edinger, PhD; [5]Colin A. Espie, PhD; [6]Kenneth L. Lichstein, PhD

[1]Université Laval, Québec, Canada; [2]University of Arizona, Tucson, AZ; [3]University of Pittsburgh School of Medicine, Pittsburgh, PA; [4]VA and Duke University Medical Centers, Durham, NC; [5]University of Glasgow, Glasgow, Scotland; [6]University of Alabama, Tuscaloosa, AL

Background: Recognition that psychological and behavioral factors play an important role in insomnia has led to increased interest in therapies targeting these factors. A review paper published in 1999 summarized the evidence regarding the efficacy of psychological and behavioral treatments for persistent insomnia. The present review provides an update of the evidence published since the original paper. As with the original paper, this review was conducted by a task force commissioned by the American Academy of Sleep Medicine in order to update its practice parameters on psychological and behavioral therapies for insomnia.

Methods: A systematic review was conducted on 37 treatment studies (N = 2246 subjects/patients) published between 1998 and 2004 inclusively and identified through PsycInfo and Medline searches. Each study was systematically reviewed with a standard coding sheet and the following information was extracted: Study design, sample (number of participants, age, gender), diagnosis, type of treatments and controls, primary and secondary outcome measures, and main findings. Criteria for inclusion of a study were as follows: (a) the main sleep diagnosis was insomnia (primary or comorbid), (b) at least 1 treatment condition was psychological or behavioral in content, (c) the study design was a randomized controlled trial, a nonrandomized group design, a clinical case series or a single subject experimental design with a minimum of 10 subjects, and (d) the study included at least 1 of the following as dependent variables: sleep onset latency, number and/or duration of awakenings, total sleep time, sleep efficiency, or sleep quality.

Results: Psychological and behavioral therapies produced reliable changes in several sleep parameters of individuals with either primary insomnia or insomnia associated with medical and psychiatric disorders. Nine studies documented the benefits of insomnia treatment in older adults or for facilitating discontinuation of medication among chronic hypnotic users. Sleep improvements achieved with treatment were well sustained over time; however, with the exception of reduced psychological symptoms/distress, there was limited evidence that improved sleep led to clinically meaningful changes in other indices of morbidity (e.g., daytime fatigue). Five treatments met criteria for empirically-supported psychological treatments for insomnia: Stimulus control therapy, relaxation, paradoxical intention, sleep restriction, and cognitive-behavior therapy.

Discussion: These updated findings provide additional evidence in support of the original review's conclusions as to the efficacy and generalizability of psychological and behavioral therapies for persistent insomnia. Nonetheless, further research is needed to develop therapies that would optimize outcomes and reduce morbidity, as would studies of treatment mechanisms, mediators, and moderators of outcomes. Effectiveness studies are also needed to validate those therapies when implemented in clinical settings (primary care), by non-sleep specialists. There is also a need to disseminate more effectively the available evidence in support of psychological and behavioral interventions to health-care practitioners working on the front line.

Keywords: Insomnia, treatment, behavioral, psychological, non pharmacological

Citation: Morin CM; Bootzin RR; Buysse DJ et al. Psychological and behavioral treatment of insomnia: update of the recent evidence (1998-2004). *SLEEP* 2006;29(11):1398-1414.

1.0 INTRODUCTION

INSOMNIA IS A PREVALENT COMPLAINT BOTH IN THE GENERAL POPULATION AND IN CLINICAL PRACTICE. IT MAY PRESENT AS THE PRIMARY COMPLAINT or in association with another physical or mental-health problem. Prevalence estimates indicate that about one third of the adult population reports insomnia symptoms, 9%-12% experience additional daytime consequences, and approximately 6% meet formal criteria for an insomnia diagnosis.[1] Insomnia is more common among women, middle-aged and older adults, shift workers, and patients with medical or psychiatric disorders. Persistent insomnia can produce an important burden for the individual and for society, as evidenced by reduced quality of life, impaired daytime functioning and increased absenteeism at work, and higher health-care costs. Persistent insomnia is also associated with increased risks of depression and chronic use of hypnotics.[2-5]

The diagnosis of insomnia is based on a subjective complaint of difficulties falling or staying asleep, or nonrestorative sleep, that is associated with marked distress or significant daytime impairments.[6,7] Several indicators are useful to quantify the severity and clinical significance of insomnia. These markers may include the intensity (e.g., time to fall asleep, duration of awakenings, total sleep time), frequency, and duration of sleep difficulties. In the case of duration, a distinction is made between adjustment insomnia, a condition lasting a few days or weeks and often associated with stressful life events or changes in schedules and environment, and persistent insomnia, a condition lasting more than 1

Disclosure Statement

This was not an industry supported study. Dr. Morin has received research support from Sanofi-Aventis; has provided consulting services to Sepracor, Pfizer, Neurocrine, Takeda, and Shire Biochem; and has participated in speaking engagements supported by Takeda, Sanofi-Aventis, and Merck. Dr. Buysse is a consultant for Actelion, Cephalon, Eli Lilly, GlaxoSmithKline, Merck, Neurocrine, Pfizer, Respironics, Sanofi-Aventis, Servier, Sepracor, and Takeda. Dr. Edinger has received research support from Respironics; and has received fees for manuscript preparation from Sepracor. Dr. Lichstein is a consultant for Lilly and Takeda; has received research support from Respironics; and has participated in a speaking engagement supported by Sanofi-Aventis. Drs. Bootzin and Espie have indicated no financial conflicts of interest.

Submitted for publication April 20, 2006
Accepted for publication April 30, 2006
Address correspondence to: Charles M. Morin, Ph.D., Université Laval, École de Psychologie, Pavillon F.A.S., Québec, Québec, Canada, G1K 7P4; Email: cmorin@psy.ulaval.ca

Table 1—Psychological and Behavioral Treatments for Insomnia

Therapy	Description
Stimulus control therapy	A set of instructions designed to reassociate the bed/bedroom with sleep and to re-establish a consistent sleep-wake schedule: (1) Go to bed only when sleepy; (2) get out of bed when unable to sleep; (3) use the bed/bedroom for sleep only (no reading, watching TV, etc); (4) arise at the same time every morning; (5) no napping.
Sleep restriction therapy	A method designed to curtail time in bed to the actual amount of sleep time. For example, if a patient reports sleeping an average of 6 hours per night, out of 8 hours spend in bed, the initial recommended sleep window (from lights out to final arising time) would be restricted to 6 hours. Periodic adjustments to this sleep window are made contingent upon sleep efficiency, until an optimal sleep duration is reached.
Relaxation training	Clinical procedures aimed at reducing somatic tension (e.g., progressive muscle relaxation, autogenic training) or intrusive thoughts at bedtime (e.g., imagery training, meditation) interfering with sleep.
Cognitive therapy	Psychological methods aimed at challenging and changing misconceptions about sleep and faulty beliefs about insomnia and its perceived daytime consequences. Other cognitive procedures may include paradoxical intention or methods aimed at reducing or preventing excessive monitoring of and worrying about insomnia and its correlates/consequences.
Sleep hygiene education	General guidelines about health practices (e.g., diet, exercise, substance use) and environmental factors (e.g., light, noise, temperature) that may promote or interfere with sleep. This may also include some basic information about normal sleep and changes in sleep patterns with aging.
Cognitive-behavior therapy	A combination of any of the above behavioral (e.g., stimulus control, sleep restriction, relaxation) and cognitive procedures.

month and often several years. Insomnia complaints are typically associated with reports of daytime fatigue, problems with memory and concentration, and mood disturbances, impairments that may be the primary concerns prompting patients to seek treatment. Insomnia can be a symptom of several other conditions including medical, psychiatric, substance abuse or another sleep disorder; or, it can be a disorder in itself as in primary insomnia.[6-8]

There are several treatment options available for insomnia, including psychological/ behavioral approaches, various classes of medications, and a host of complementary and alternative therapies (e.g., herbal/dietary supplement, acupuncture). The present paper focuses on psychological and behavioral approaches to treating insomnia. These procedures have received increasing research attention in the past 2 decades, and were noted as effective therapies at a recent National Institutes of Health State-of-the-Science Conference[9]on the manifestations and management of chronic insomnia. These methods include stimulus control therapy, sleep restriction, relaxation-based interventions, paradoxical intention, cognitive therapy, and combined cognitive-behavioral therapy. A brief summary of the nature of these interventions is presented in Table 1; more extensive descriptions are available in other sources.[10,11]

2.0 PURPOSE

The objective of this paper is to provide an update of the evidence regarding the efficacy, effectiveness, durability, and generalizability of psychological and behavioral interventions for persistent insomnia. The evidence is reviewed for the treatment of both primary insomnia and insomnia associated with other medical, psychiatric, or substance abuse disorders. As with the initial review paper[12] this updated review was commissioned by the Standards of Practice Committee of the American Academy of Sleep Medicine.

3.0 METHODS

3.1 Search Methods, Keywords, and Databases

Treatment studies selected for review in this paper were identified through PsycInfo and Medline searches for research conducted from 1998 through 2004 inclusively. The following key words were used: nonpharmacologic, behavior therapy, cognitive therapy, psychotherapy, alternative medicine, stimulus control, progressive relaxation therapy or progressive muscle relaxation, paradoxical techniques or paradoxical intention, behavior modification, cognitive behavior therapy, psychological therapy, treatment, intervention, behavioral intervention, treatment, cognitive treatment, alternative treatment, therapy, biofeedback, sleep restriction, sleep deprivation, complementary therapies, mind-body and relaxation techniques, aromatherapy, biofeedback, hypnosis, imagery, or meditation, relaxation, relaxation techniques, yoga, massage. These terms were combined with sleep disorders or sleep initiation and maintenance disorders, or insomnia, or dyssomnia. The search was limited to humans, adults (18 and older), English or French language.

3.2 Selection Criteria of Treatment Studies

The initial PsycInfo and Medline searches yielded a total of 312 titles of potential interest; an additional 34 titles were identified by members of the task force through their own reading of the literature, for a total of 346 titles of potential interest. Of these, 102 abstracts were read by the task force chair for initial screening and 53 articles were selected for full review by 2 independent members of the task force. Only peer-reviewed published articles were retained at this phase. Each rater used a standard extraction sheet to summarize information about the study including experimental design, sample (number of participants, age, gender), diagnosis, type of treatments and controls, primary and secondary outcome measures, and main findings. Data extraction was completed independently and discrepancies between 2 members of a pair of raters were resolved through discussion with the chair and other members of the task force. The criteria for inclusion of a study were: (a) the main sleep diagnosis was insomnia (primary or comorbid), (b) at least 1 treatment condition was psychological or behavioral in content, (c) the study design was a randomized controlled trial, a nonrandomized group design, a clinical case series or a single subject experimental design with a minimum of 10 subjects, (d) the dependent measure included 1 or more of the following variables (as measured by daily sleep diaries, polysomnography (PSG), or actigraphy): Sleep onset latency (SOL), number of awakenings (NA), time awake after sleep onset (WASO), total sleep time (TST), sleep efficiency (SE), or sleep quality

(SQ). Studies using global measures of treatment outcome were included if those measures had been validated. Studies of circadian disorders (e.g., phase delay or phase advance syndromes) and those using other nonpharmacological therapies (e.g., light therapy, electro-sleep therapy) or complementary and alternative therapies (e.g., acupuncture) were excluded from this review.

The majority of the initial 346 titles of potential interest were excluded either because they were not treatment studies for insomnia or because the main intervention was pharmacological. Of the 53 full articles reviewed and rated, 16 were rejected. The main reasons for article rejection were: no documentation of an insomnia diagnosis, sample size smaller than 10, results were secondary analyses of other databases (e.g., predictors of treatment response) and used global and non-validated measures of outcome. A list of excluded studies with reasons for exclusion is provided in an, Appendix A. The 37 studies that met inclusion criteria are listed in Table 2. For each study, we report the study design and the evidence level (using the Sackett System, 1993), the sample size (enrolled/completed), age, gender, insomnia diagnosis (primary or secondary), types of treatment and control conditions, therapy dosage, format, setting, therapist training, treatment duration and longest follow-up, primary and secondary outcome measures, and summary of main findings. Although some studies not meeting inclusion criteria are discussed in the text they are not included in the evidence table. Based on the Sackett sytem,[13] the criteria for grading evidence level of each study were: Randomized well-designed trials with low alpha and beta error (Grade I), randomized trials with high alpha and beta error (Grade II), nonrandomized concurrently controlled studies (Grade III), nonrandomized historically controlled studies (Grade IV), case series (Grade V).

4.0 RESULTS

4.1 Descriptive Features of the Studies and Samples

Table 2 summarizes the main features of the 37 studies selected for this review paper. The number of studies meeting Sackett System standards for grading evidence levels[13] was as follows: 11 studies were graded I, 13 studies graded II, 2 studies graded III, 5 studies graded IV, and 6 studies graded V. A total of 2246 patients with insomnia were enrolled in the 37 studies and approximately 2029 of those completed treatment, for an attrition rate of less than 10% overall. With a few exceptions (studies of Veterans, substance abusers, internet-based treatment), there was a larger representation of women than men enrolled in most studies, with a typical ratio of 2:1, which is representative of insomnia prevalence estimates.[1] Nine of the 37 studies focused specifically on older adults (average age > 60 years old).

The main sleep diagnoses of patients enrolled in the studies were primary or psychophysiological insomnia (28 studies), insomnia associated with medical (4 studies) or psychiatric disorders (3 studies) or a mix of both conditions (6 studies), and hypnotic-dependent insomnia (5 studies). Eight studies examined treatment efficacy in patients with different subgroups of insomnia diagnoses, leading to a total number of studies greater than 37 studies.

The majority of reviewed studies (n = 33) relied on prospective daily sleep diaries to document treatment outcome. Participants were typically required to complete a daily diary for a minimum of a 1 or 2 week baseline period, for the duration of treatment, and for an additional 1 or 2 week period at post treatment and follow-ups. We retained 3 studies using the Pittsburgh Sleep Quality Index (PSQI) as the primary outcome.[14-16] A few studies have also included polysomnography (n = 7)[17-23]and actigraphy (n = 6)[18,19,24-27] to complement subjective reports from daily sleep diaries. Those studies are identified in Table 2 and in the appropriate subsections of the results. Primary dependent variables derived from these assessment methods were sleep onset latency (SOL), wake time after sleep onset (WASO), total sleep time (TST), sleep efficiency (SE), and sleep quality (SQ). Secondary outcomes included measures of insomnia severity (Insomnia Severity Index; ISI,[28] sleep quality (PSQI),[29] psychological symptoms (Beck Depression Inventory, BDI,[30] State-Trait Anxiety Inventory STAI[31], and fatigue (Multidimensional Fatigue Inventory, MFI[32]).

The following sections summarize the evidence regarding the efficacy of treatment for primary insomnia, the generalizability of the evidence to different forms of insomnia (primary, secondary, hypnotic-dependent), insomnia in older adults, and the clinical significance and durability of sleep improvements over time. Comparative findings of single and multifaceted therapies and of different treatment implementation models are also summarized.

4.2 Treatment of Primary Insomnia

Seventeen studies evaluated the effects of treatment for primary insomnia (see Table 2). Five of those studies were randomized clinical trials (RCT; with grade I,[17,20,21,33,34] 4 of which used CBT as the main intervention. In a comparative study of CBT (without relaxation), relaxation, and a psychological placebo (i.e., quasi-desensitization) with a sample of 75 primary insomniacs,[17] CBT produced greater improvements on the main diary and PSG-defined sleep measures (e.g., SE, WASO) relative to relaxation and control. More CBT patients (64%) achieved clinically significant outcomes compared to relaxation (12%) and placebo (8%). In an effectiveness trial[33] conducted in primary care that evaluated CBT against a wait-list control group, active treatment was found superior to the control condition on most primary and secondary outcome measures. SOL was reduced from 61 to 28 min following active treatment compared to a change from 74 to 70 min for the control condition.[a] Smaller improvements were noted on WASO. There was no significant change on TST during treatment, but an increase of about one-half hour over baseline was obtained at follow up. Of those patients using hypnotic medications at baseline, 76% were medication-free at the end of treatment and 80% at the 12-month follow up. In a comparative study[21] of CBT, medication (temazepam), and combined CBT plus medication, all 3 active treatments improved more than pill placebo on the main outcomes of WASO and SE, with a trend for the combined intervention to yield the greatest benefits. PSG data produced similar outcomes, although of smaller magnitude, but only the combined condition was significantly superior to placebo on the main outcome variables. According to PSG, more patients in the CBT (56%) and combined (68%) conditions achieved clinically significant changes (i.e., SE > 85%) relative to medication alone (47%) or placebo groups (22%). In another clinical trial of primary insomnia in older adults,[20] sleep restriction and relaxation were more effective than placebo on sleep diary variables but not on PSG measures. Sleep restriction produced the best outcome

[a]*Unless otherwise stated, reported outcomes are based on daily sleep diaries.*

Davidson et al. (2001);[42]	Non RCT; IV	14/11; 91%; 54.7; Insomnia associated with a medical condition (cancer)	Multi-component (SC, Rel, SHE)	8 wk; No FU	Sleep diaries; ISI, Hospital Anxiety and Depression Scale; QoL	Significant improvements in SOL (42 to 6 min), NA (1.7 to 1.0), WASO (42 to 11 min), SE (73 to 89%), TST (376 to 411 min), sleep quality and impairments ratings. All patients (n = 9) with baseline SOL or WASO > 30 min and SE < 85% no longer met these criteria after treatment. Significant improvements on QoL for role functioning and fatigue.
Dopke et al. (2004);[45]	NonRCT; IV	11/10; 60%; 45.6; Insomnia associated with psychological disorders	Multicomponent (SH+SC+SR+ Rel)	10 wk; No FU	Sleep diaries; ISI	No significant improvement on SOL or WASO, but significant reductions of ISI scores.
Edinger et al. (2001);[17]	RCT; I	75/70; 46.7%; 55.3; Persistent primary insomnia	CBT; Rel; Placebo (psychological)	6 wk; 6 mo	Sleep diaries; PSG; BDI, Insomnia Symptom Questionnaire, Self-efficacy Scale	CBT produced greater improvements on most sleep measures, with higher PSG and diary SE (86% and 84% respectively) and lower diary WASO (28 min) than relaxation (PSG SE: 78%, diary SE: 78%; WASO: 44 min), and placebo (PSG SE: 76%, diary SE: 76%, diary WASO: 47 min). More CBT patients (64%) achieved clinically significant outcomes compared to relaxation (12%) and placebo (8%). Greater improvements of self-efficacy in CBT and larger reductions of depressive symptoms in relaxation relative to placebo.
Edinger & Sampson (2003);[35]	RCT; II	20/19;10%;51.0; Primary insomnia	Multi-component (SC, SR, SHE); Control (SHE only)	2 wk; 3 mo	Sleep diaries; Insomnia Symptoms QuestionnaireSelf-efficacy Scale, DBAS	Significantly greater reductions in WASO (97 to 54 min) and SOL (39 to 31 min), and greater increases in SE (71 to 80%) and sleep quality in CBT patients relative to sleep hygiene education controls. These results were maintained at FU. Larger FU changes in CBT patients relative to controls on measures of restedness, insomnia symptoms, control over sleep and sleep-related cognitions.
Espie et al. (2001);[33]	RCT; I	161/138; 68%; 51.4; Persistent sleep-onset and/maintenance insomnia (probable mix of primary and secondary insomnia)	CBT (with Rel); Wait list control	6 wk; 12 mo	Sleep diaries; PSQI, BDI, STAI, Penn State Worry Questionnaire	SOL reduced from 61 to 28 min following CBT compared to 74 to 70 min for controls; results maintained at FU. Mean reduction of WASO from 78 to 47 min at posttreatment and partially maintained (53 min.) at FU. No change in TST during treatment, but increase of 30 min at FU. Of the 74 patients using medications, 56 had discontinued hypnotic usage at posttreatment and 50 were still medication free at FU.
Friedman et al. (2000);[18]	RCT; II	39/35; 69%; 64.2; Psychophysiologic (n =28), inadequate sleep hygiene (n=9), insufficient sleep (n= 2)	SR + SHE; SR (with optional nap) + SHE; SHE	4 wk; 3 mo	Sleep diaries; PSG; Actigraphy; MSLT, Stanford Sleepiness Scale	Sleep diaries: Higher SE and reduced time in bed for the two sleep restriction conditions at posttreatment. Slower but comparable improvement for the sleep hygiene only condition at FU. Actigraphy: reductions of TST at posttreatment and return towards baseline at follow-up for the two sleep restriction conditions. No group differences on any actigraphy measure. PSG (n=16) data not formally analyzed but descriptive data suggest similar pattern of results as those observed on actigraphy.
Guilleminault et al. (2002);[19]	RCT; II	130/126; 100%; 62.4; Chronic insomnia	CBT; Control (SHE); Sleep disordered breathing (SDB) treatment (Nasal CPAP or ENT nasal treatment)	6 mo; no FU	PSG; Actigraphy; Visual Analogue Scales (sleep quality, daytime fatigue); Epworth Sleepiness Scale	PSG-defined SOL reduced by 12 min in CBT groups (with or w/o sleep-disordered breathing). WASO was reduced in all groups. TST significantly increased in CBT group (without sleep-disordered breathing). No significant change in actigraphy-defined measures of SOL and WASO, but all groups improved quantity and quality of sleep at FU. No change on measure of sleepiness; fatigue ratings decreased for the sleep disordered breathing group.

Davidson et al. (2001);[42]	Non RCT; IV	14/11; 91%; 54.7; Insomnia associated with a medical condition (cancer)	Multi-component (SC, Rel, SHE)	8 wk; No FU	Sleep diaries; ISI, Hospital Anxiety and Depression Scale; QoL	Significant improvements in SOL (42 to 6 min), NA (1.7 to 1.0), WASO (42 to 11 min), SE (73 to 89%), TST (376 to 411 min), sleep quality and impairments ratings. All patients (n = 9) with baseline SOL or WASO > 30 min and SE < 85% no longer met these criteria after treatment. Significant improvements on QoL for role functioning and fatigue.
Dopke et al. (2004);[45]	NonRCT; IV	11/10; 60%; 45.6; Insomnia associated with psychological disorders	Multicomponent (SH+SC+SR+ Rel)	10 wk; No FU	Sleep diaries; ISI	No significant improvement on SOL or WASO, but significant reductions of ISI scores.
Edinger et al. (2001);[17]	RCT; I	75/70; 46.7%; 55.3; Persistent primary insomnia	CBT; Rel; Placebo (psychological)	6 wk; 6 mo	Sleep diaries; PSG; BDI, Insomnia Symptom Questionnaire, Self-efficacy Scale	CBT produced greater improvements on most sleep measures, with higher PSG and diary SE (86% and 84% respectively) and lower diary WASO (28 min) than relaxation (PSG SE: 78%, diary SE: 78%; WASO: 44 min), and placebo (PSG SE: 76%, diary SE: 76%, diary WASO: 47 min). More CBT patients (64%) achieved clinically significant outcomes compared to relaxation (12%) and placebo (8%). Greater improvements of self-efficacy in CBT and larger reductions of depressive symptoms in relaxation relative to placebo.
Edinger & Sampson (2003);[35]	RCT; II	20/19;10%;51.0; Primary insomnia	Multi-component (SC, SR, SHE); Control (SHE only)	2 wk; 3 mo	Sleep diaries; Insomnia Symptoms QuestionnaireSelf-efficacy Scale, DBAS	Significantly greater reductions in WASO (97 to 54 min) and SOL (39 to 31 min), and greater increases in SE (71 to 80%) and sleep quality in CBT patients relative to sleep hygiene education controls. These results were maintained at FU. Larger FU changes in CBT patients relative to controls on measures of restedness, insomnia symptoms, control over sleep and sleep-related cognitions.
Espie et al. (2001);[33]	RCT; I	161/138; 68%; 51.4; Persistent sleep-onset and/maintenance insomnia (probable mix of primary and secondary insomnia)	CBT (with Rel); Wait list control	6 wk; 12 mo	Sleep diaries; PSQI, BDI, STAI, Penn State Worry Questionnaire	SOL reduced from 61 to 28 min following CBT compared to 74 to 70 min for controls; results maintained at FU. Mean reduction of WASO from 78 to 47 min at posttreatment and partially maintained (53 min.) at FU. No change in TST during treatment, but increase of 30 min at FU. Of the 74 patients using medications, 56 had discontinued hypnotic usage at posttreatment and 50 were still medication free at FU.
Friedman et al. (2000);[18]	RCT; II	39/35; 69%; 64.2; Psychophysiologic (n =28), inadequate sleep hygiene (n=9), insufficient sleep (n= 2)	SR + SHE; SR (with optional nap) + SHE; SHE	4 wk; 3 mo	Sleep diaries; PSG; Actigraphy; MSLT, Stanford Sleepiness Scale	Sleep diaries: Higher SE and reduced time in bed for the two sleep restriction conditions at posttreatment. Slower but comparable improvement for the sleep hygiene only condition at FU. Actigraphy: reductions of TST at posttreatment and return towards baseline at follow-up for the two sleep restriction conditions. No group differences on any actigraphy measure. PSG (n=16) data not formally analyzed but descriptive data suggest similar pattern of results as those observed on actigraphy.
Guilleminault et al. (2002);[19]	RCT; II	130/126; 100%; 62.4; Chronic insomnia	CBT; Control (SHE); Sleep disordered breathing (SDB) treatment (Nasal CPAP or ENT nasal treatment)	6 mo; no FU	PSG; Actigraphy; Visual Analogue Scales (sleep quality, daytime fatigue); Epworth Sleepiness Scale	PSG-defined SOL reduced by 12 min in CBT groups (with or w/o sleep-disordered breathing). WASO was reduced in all groups. TST significantly increased in CBT group (without sleep-disordered breathing). No significant change in actigraphy-defined measures of SOL and WASO, but all groups improved quantity and quality of sleep at FU. No change on measure of sleepiness; fatigue ratings decreased for the sleep disordered breathing group.

Hryshko-Mullen (2000);[40]	Case replication series; V	42/42; 52%; 53.6; Primary insomnia	CBT (with Rel)	10 wk; 1 mo	Sleep diaries	CBT produced significant improvements in most sleep parameters. For patients with sleep onset insomnia, SOL was reduced from 69.4 to 32.9 min and SE increased from 59.3% to 75.6%; for those with sleep-maintenance insomnia, WASO was decreased from 65 to 41.5 min, SE increased from 62.1% to 75.9%, and TST increased from 298 to 333 min.
Jacobs et al. (2004);[34]	RCT; I	63/54; 70%; 47.1; Primary insomnia	CBT (with Rel); SHE + zolpidem; CBT + zolpidem; Pill placebo	8 wk; 12 mo	Sleep diaries; BDI, Profile of Moods State scale	SE increased by 14% and SOL decreased by 34 min in CBT group. TST increased in all 4 groups, with largest gain (78 min) in the medication only group. No differences on mood measures. Gains maintained at 12-month FU for CBT and CBT + zolpidem conditions. No information for medication only groups. More CBT patients achieved clinically significant changes on SOL and SE measures.
Lichstein et al. (1999);[50]	NonRCT; III	40/40; 58%; 52; Psychophysiologic insomnia and Hypnotic dependent insomnia	Rel with medication withdrawal; Medication withdrawal alone; Rel alone; No treatment	6 wk; 2 mo	Sleep diaries; Epworth Sleepiness Scale, BDI, STAI.	Relaxation increased SE in both medicated (from 75.7% to 79.6%) and nonmedicated insomniacs (from 67.0% to 78.8%). Worsening of all other sleep measures during medication withdrawal; relaxation did not attenuate this effect. Only medicated participants receiving relaxation reported improved sleep quality. Relaxation associated with fewer withdrawal symptoms than no relaxation. Medicated participants (with or without relaxation) reduced medication use by 78% at FU. Significant reductions of anxiety and depressive symptoms but no change in sleepiness.
Lichstein et al. (2000);[43]	RCT; I	49/44; 48%; 68.6; Insomnia secondary to medical/ psychiatric conditions	Multicomponent (SC, Rel, SHE); Wait list control	4 wk; 3 mo	Sleep diaries; STAI, Geriatric Depression Scale, Insomnia Impact Scale	No difference in outcome between insomnia secondary to medical or psychiatric disorders. Greater improvements for treated patients relative to controls with WASO reductions from 87min (baseline) to 61 min (posttreatment) to 56 min (follow-up) and SE increases from 67% (baseline) to 78% (posttreatment) to 78% (follow-up). SOL reduced and TST increased in both conditions at post-treatment. 57% of treated patients achieved clinical improvement in SE compared with 19% of control patients. None of the secondary measures were significant.
Lichstein et al. (2001);[20]	RCT; I	89/72; 74%; 68.03; Psychophysiological insomnia	Rel; SR; Placebo (psychological)	6 wk; 12 mo	Sleep diaries; PSG; Epworth Sleepiness Scale, DBAS; Insomnia Impact Scale, Fatigue Severity Scale	Sleep Restriction and Relaxation more effective than Placebo on sleep continuity variables at post and FU. WASO reduced from 67 min (baseline) to 43 (post) to 38 (FU) in Sleep Restriction, compared to 67 min (baseline), 43 (post), and 52 (FU) for Relaxation. No significant gains in daytime functioning. Sleep restriction produced best outcomes at FU. No significant change on PSG measures. All groups (including placebo) improved on the secondary variables.
Means et al. (2000);[37]	RCT; I	NS/57;68.5%; 21.2 Primary insomnia	Rel; Wait list control	3 wk; 5 wk	Sleep diaries; DBAS, Fatigue Severity and; Epworth Sleepiness Scales, Penn State Worry Questionnaire	Relaxation produced more improvement than no treatment on diary measures of WASO, SE, sleep quality, but not SOL. The magnitude of improvement was small. For example, WASO for treated insomniacs went from 22 min (baseline) to 13 min (posttreatment) and SE went from 84.8% (baseline) to 88.4% (posttreatment). No group differences for daytime measures.

Mimeault & Morin (1999);[38]	RCT; I	58/54; 59%; 50.8; Primary insomnia	Self-help CBT with telephone contact; Self-help CBT only; Wait list control	6 wk; 3 mo	Sleep diaries; PSQI, ISI, DBAS, BAI	The addition of telephone consultations to a self-help treatment slightly enhanced outcome on TWT (- 82 min vs. - 62 min) and SE (+ 15% vs. + 11%) at posttreatment, but both treated groups were comparable at follow-up and remained more improved than controls. 59% (17/29) of treated patients had SE > 80% at follow up. Improvements were also obtained on secondary measures of PSQI, ISI, DBAS and BDI. Hypnotic use decreased in all three groups.
Morgan et al. (2003);[15]	RCT; I	209/123; 67.5%; 65.5; chronic insomnia and chronic use of hypnotics	CBT (with Rel) and medication withdrawal; No treatment control	3 mo; 6 mo	PSQI (TST, SOL,SE, global score), SF-36, use of hypnotic medication, treatment costs	CBT patients showed significant improvements in PSQI scores (global, sleep latency, sleep efficiency) and reductions of hypnotic use at 3- and 6-month follow-ups. Greater percentages of the CBT group (39%) than controls (11%) achieved low hypnotic (< 50% of baseline) or no hypnotic use at 6-month follow-up. Lower SF-36 vitality scale at the three month follow-up in CBT than in control group. Greater costs for CBT initially but evidence of longer term cost offsets due to reduction of sleep medication usage.
Morin et al. (1999);[12]	RCT; I	78/72; 65%; 65.0; Primary insomnia	CBT; Med (temazepam); Combined CBT+Med; Placebo	8 wk; 24 mo	Sleep diaries; PSG; ISI	All three treatments improved significantly more than placebo, with a trend for the combined condition to yield greatest benefits. CBT reduced diary measures of WASO from 50 to 22 min, Med 55 to 29 min and combined 57 to 21 min compared with 62 to 52 min for placebo. SE increased by 17% (CBT), 11% (PCT), 21% (CBT+Med), and 4% (Placebo). PSG data showed that all three treated groups spent less time awake after sleep-onset than placebo, but only the combined approach was superior to placebo. CBT patients best sustained sleep improvements over time, whereas Medication a lone group did not, and the combined condition showed more variable long-term outcomes.
Morin et al. (2004);[22]	RCT; I	76/69; 50%; 62.5; Hypnotic-dependent insomnia	CBT; Medication taper; Combined CBT + medication taper	10 wk; 12 mo	Sleep diaries; PSG; ISI, BAI, BDI, benzodiazepine use.	All three groups reduced both the quantity (90%) and frequency (80%) of benzodiazepine use, but more participants receiving the combined intervention were drug-free at post treatment. Modest changes in sleep during initial withdrawal but participants receiving CBT, alone or combined with medication taper, reported greater sleep improvements relative to patients receiving medication taper alone. Improvements reported on secondary variables (ISI, BAI BDI) in all three groups, with gains maintained through FU.
Pallesen et al. (2003);[49]	NonRCT; III	66/55; 84%; 69.8; Primary and secondary insomnia	Rel + SHE; SC + SHE; Wait list control	4 wk; 6 mo	Sleep diaries; ISI, rating of daytime alertness, use of hypnotic medication, life satisfaction	SHE+SC reduced SOL from 87 to 56 min and WASO from 55 to 42 min. SHE+Rel reduced SOL from 71 to 51 min and WASO from 47 to 33 min. No significant group differences between treatments. Effect sizes were of medium size and greater for sleep than daytime measures. Treatment effects maintained in both treatment groups. Controls showed no improvement on any variable.

Perlis et al. (2000);[47]	Case replication series; V	85/47; 64%; 43; Mix of primary insomnia and insomnia associated with medical or psychiatric disorders	CBT	4-9 wk; NS	Sleep diaries	61% completing adequate treatment trial showed significant improvement, with averaged SOL reduction of 48 min (65%; effect size = 1.25), WASO reduction of 61 min (48%; effect size = 1.42), and TST increase of 34 min (13%; effect size = .41).
Perlis et al. (2001);[46]	Case replication series; V	89/28; 64%; 46.5; Mix of primary and secondary insomnia	CBT	4-9 wk; NS	Sleep diaries	Subjects completing a minimum adequate trial reduced their SOL (55 to 21 min: effect size = .85), WASO (83 to 28 min: effect size = 1.14), number of awakenings (effect size = .54), and increased TST (291 to 341 min: effect size = .54). Outcomes did not vary relative to medical or psychiatric morbidity.
Perlis et al. (2004);[54]	RCT; II	30/27; 70%; 41.3; Psychophysiologic insomnia	CBT + placebo drug; CBT+ modafinil 100 mg; Contact control + modafinil 100 mg	8 wk; No FU	Sleep diaries; Epworth Sleepiness Scale	Average SOL reduction of 17 min and WASO of 28 min among CBT patients. Trends suggesting lower Epworth Sleepiness Scale scores and higher adherence to prescribed bedtime during CBT among participants receiving modafinil.
Riedel et al. (1998);[51]	RCT; II	45/41; 54%; 56.6; Hypnotic-dependent insomnia, primary insomnia	SC + med withdrawal; SC alone; Med withdrawal alone; Wait list control	4-6 wk; 2 mo	Sleep diaries; Use of hypnotic medication, Epworth Sleepiness Scale, BDI, STAI	Significant improvements on most sleep parameters over time among stimulus control subjects but not in controls. Lower daytime sleepiness in treated subjects relative to controls at post-treatment and follow-up. No significant differences in outcomes between medicated an unmedicated patients. BDI and STAI showed no significant changes over time.
Rosen et al. (2000); 55	RCT; II	41/32; 66%; 47.2; Psychophysiologic insomnia	Med (estazolam) + relaxation; Med (estazolam) + imagery; Med (estazolam) + SHE	4 wk; 6 mo	Sleep diaries; BDI, Pre-Sleep Arousal Scale, Sleep Efficacy Scale, Sleep Hygiene Practice and Knowledge Scale, Taylor Manifest Anxiety Scale	All three groups showed significant increase of TST (Med + Rel = + 65 min; Med + Imagery = + 40 min; Med + SHE = + 34 min). Only the relaxation and imagery groups showed significant pre to post changes in WASO (- 17 min and - 33 min, respectively) and SE (+ 9.7%, + 7.4%). Significant improvements in all groups at FU relative to baseline sleep. Significant improvements across groups for the Pre-Sleep Arousal Scale, Sleep Efficacy Scale, and BDI (but not anxiety) from baseline to 6-month follow-up, but no group differences.
Rybarczyk et al. (2002); 27	RCT; II	51/38; 58%; 67.8; Insomnia secondary to medical illness	CBT (with Rel); Rel; Wait list control	8 wk; 4 mo	Sleep diaries, Actigraphy; PSQI, DBAS, GDS, BAI, SF-36, McGill Pain Questionnaire, Life satisfaction	CBT more effective than control at posttreatment and FU on measures of SE, WASO, PSQI, and DBAS. Relaxation produced greater change in TST than CBT at posttreatment, greater change than control in TST and PSQI at posttreatment, and greater change than control in SE, WASO, and PSQI at FU. More CBT than control patients achieved clinically significant improvement at posttreatment and FU, and a higher proportion of Relaxation than controls had clinically significant improvement at posttreatment. No group differences on actigraphy, medication use, or secondary outcomes.

Simeit et al. (2004); 16	NonRCT; IV	NS/229; 75%; 58; Insomnia secondary to cancer	CBT + Progressive muscle relaxation; CBT + Autogenic training Standard treatment	3-4 wk; 6 mo	PSQI, Cancer Quality of Life Questionnaire	Significant improvement from baseline to FU on SOL, TST, SE for all groups; no group differences. On sleep quality, daytime energy, daytime dysfunction and most other quality of life measures, improvement occurred in all groups. Sleep medication use decreased among participants receiving CBT only.
Strom et al. (2004); 39	RCT; II	109/81; 65%; 44.1; Primary insomnia	Internet CBT (with Rel); Wait list control	5 wk; 9 mo	Sleep diaries; DBAS, Medication Index	Greater improvements observed on TWT (- 55 min), TST (+ 34 min), and SE (+ 10%) in treated relatie to controls. Greater reductions of hypnotic use and improvements of DBAS scores in CBT relative to controls. Improvements noted on other sleep measures in both treated and control conditions. Attrition rate of 24%.
Verbeek et al. (1999); 48	Case replication series; V	127/86; 65%; 46; 54 psychophysiologic, 5 hypnotic dependent, 14 secondary (9 affective, 4 neurological, 1 medical)	CBT (with Rel) and medication withdrawal	6 wk; NS	Sleep diaries; Global improvement	Significant improvements on SOL (79 vs. 45 min), WASO (137 vs. 74 min), SE (56 vs. 71%). Overall treatment effect rated as "good" in 49%, "reasonable" in 37%, "no change" in 14%; no difference between hypnotic users and non-users. 37% of hypnotic users discontinued medication and 39% reduced their use. No difference between responders and non-responders on baseline measures of sleep, depression, or anxiety.
Viens et al. (2003); 23	RCT; II	23/20; 70%; 36.0; Sleep onset insomnia	Anxiety Management Training; Rel	9 wk; No FU	PSG; STAI, BDI, MMPI, Sleep onset monitor, sleep satisfaction	Self-reported SOL significantly decreased and sleep satisfaction improved significantly. SOL measured by behavioral device improved significantly for both treatments (68 and 81 min at baseline vs. 43 and 46 min. at posttreatment). No significant effects for PSG measures of sleep continuity. Secondary measures of depression and anxiety improved with treatment.
Vincent & Hameed (2003);[41]	Case replication series; V	50; 66%; 51.4; Primary insomnia	CBT (with Rel) combined with hypnotic withdrawal and stress management	7 wk; No FU	Sleep diaries;PSQI, DBAS, ISI, BDI	Significant improvements for SOL (46 vs. 26 min), TST (5.3 vs. 6.2 hrs.), SE (80 vs. 86%), but not for WASO. Significant effect also found for PSQI, ISI, and DBAS. Better treatment compliance associated with better outcomes on DBAS, ISI, and PSQI but not with SOL, TST, SE.
Waters et al. (2003);[52]	RCT; II	53/53; 70%; 44.1; Psychophysiologic insomnia	Rel; SR + SC; Med (flurazepam); SHE	4 wk; No FU	Sleep diaries	Rel treatment had greater effect on sleep onset than SR/SC and SHE, whereas SR/SC had greater effect than Rel on sleep maintenance variables (WASO, NA). Medication produced largest changes in all sleep measures.
Abbreviations	RCT: Randomized controlled trial		CBT: Cognitive-behavior therapy; Rel: Relaxation; SR: Sleep restriction; SC: Stimulus control; SHE: sleep hygiene education; Med: Medication;		PSG: Polysomnography; PSQI: Pittsurgh Sleep Quality Index; ISI: Insomnia Severity Index; DBAS: Dysfunctional Belief and Attitudes about Sleep Scale; BDI: Beck Depression Inventory; BAI: Beck Anxiety Inventory; STAI: State-Trait Anxiety Inventory	SOL: Sleep onset latency; WASO: Wake time after sleep onset; TST: Total sleep time; SE: Sleep efficiency

on sleep efficiency but there was no evidence of improvements on secondary measures of daytime functioning. In a study that focused specifically on sleep onset insomnia,[34] CBT (with relaxation) decreased SOL by a mean of 34 min with a corresponding 14% increase of SE. Sleep improvements, which had limited impact on secondary mood measures, were well maintained at follow up. Several additional studies, including both controlled and uncontrolled studies, have documented treatment efficacy for insomnia in the context of primary care settings,[14,35,36] in comparisons of different interventions[23,37] or treatment implementation models,[38,39] or as part of case series.[40,41] Some of these studies will be discussed in later sections of this paper.

4.3 Treatment of Insomnia Associated with other Medical or Psychiatric Disorders

Twelve investigations have evaluated the efficacy of psychological and behavioral treatments for insomnia associated with another medical or psychiatric disorder. These studies have focused, for example, on patients with chronic pain,[25] cancer,[16,42] alcohol dependence [26]and older adults with various medical illnesses.[27,43] Only 4 of those 12 studies were RCT (Grade I or II)[25-27,43] and the remaining were nonrandomized studies or clinical replication series.

In a study of 60 patients with insomnia associated with chronic pain,[25] CBT was significantly more effective than control on measures of SOL, WASO, and SE, but not on number of awakenings and TST. SOL was reduced from 55 min to 28 min and SE increased from 72% to 85%. Nocturnal motor activity (as measured by actigraphy) was reduced in the treated group but not in the control group; there were no group differences on pain ratings, depressive symptoms, or medication use. In a study of 51 older adults with insomnia associated with medical illness,[27] CBT and relaxation conditions were more effective than control on diary measures of WASO and SE, as well as on a measure of overall sleep quality (PSQI); the relaxation group had a greater increase in TST than CBT and controls. A higher proportion of treated patients relative to controls achieved clinically significant improvements. There were no differential group effects on actigraphy, medication use, or other secondary measures of anxiety, depression, and quality of life. In a study of 49 older adults with insomnia associated with medical and psychiatric conditions,[43]a combined intervention of stimulus control, relaxation, and education reduced WASO 25 min and increased SE 11% at post treatment. Fifty-seven percent (57%) of treated patients achieved clinically significant improvements on SE relative to 19% of control patients; there was no significant change on secondary measures of anxiety, depression, and impact of insomnia. Outcomes were similar for individuals with insomnia associated with a medical condition and those with insomnia related to a psychiatric disorder. A controlled study conducted with recovered alcoholics[26] showed modest but significant improvements of SOL (- 18 min) and SE (+10%) among insomnia-treated patients. At the 6 month follow-up, 15% of treated participants had relapses with alcohol and this proportion was not different between treated and control patients.

Several additional studies (clinical replication series or uncontrolled group studies) have also provided evidence showing that patients with medical and psychiatric disorders could also benefit from sleep/insomnia specific interventions. Two investigations with cancer patients[16,42] showed that CBT was associated with improvements of sleep and of daytime functioning (e.g., fatigue, energy). One case series study of 67 patients with psychiatric disorders and insomnia[44] reported significant improvements of sleep, mood, fatigue, and reduced use of sleep medication, while a smaller study[45] found no change on specific sleep parameters (SOL and WASO) but reported significant reductions of global insomnia severity as measured by the ISI. Additional evidence supporting the efficacy of CBT was reported in 3 clinical replications series [46-48] conducted with heterogeneous samples of patients presenting to sleep disorders clinics with a variety of primary and secondary insomnia diagnoses. Although conclusions drawn from these uncontrolled studies should be treated with caution because of the studies' high attrition rate, the evidence suggests that among those who received an adequate treatment exposure (average of 6 - 8 therapy sessions) outcomes were comparable to those patients with primary insomnia enrolled in controlled clinical trials. Furthermore, treatment response appeared comparable between patients with medical or psychiatric comorbidity and those with primary insomnia in one study. Baseline anxiety, depression, and insomnia severity did not differ among treatment responders and nonresponders.[48]

4.4 Treatment of Insomnia in Older Adults

Nearly 25% (9 studies out of 37) of reviewed studies were conducted with older adults (average age > 60 years old). This is in sharp contrast to our previous review [12] that included only a handful of studies with older subjects. Three studies focused on older adults with primary insomnia,[18,20,21] 2 on insomnia associated with medical or psychiatric illnesses,[27,43] 1 included a mix of patients with primary and comorbid insomnia,[49] 2 evaluated the impact of psychological and behavioral interventions specifically in older adults who were chronic users of hypnotic medications,[15,22] and 1 study[19] examined the moderating role of upper airway resistance syndrome in the treatment of postmenopausal insomnia. With the exception of the study conducted by Pallesen et al,[49] all these investigations were RCT.

In a study of 89 older adults with primary insomnia,[20] sleep restriction and relaxation were both more effective than a psychological placebo for reducing WASO; changes were identical (67 min to 43 min) for the 2 treatment groups at the end of the 6-week treatment phase, but sleep restriction produced the best outcome at the 1-year follow up. No significant changes were obtained on PSG measures. All 3 conditions, including placebo control, showed improvements on secondary measures of fatigue and a measure of insomnia impact. In another placebo-controlled study,[21] 76 older adults treated with CBT, medication (temazepam), or combined CBT + medication improved more than those receiving placebo on the main outcome measures of WASO and SE. PSG comparisons yielded improvements in the same direction, albeit of smaller magnitude, than those reported on sleep diaries. A greater proportion of patients treated with CBT, alone or combined with medication, achieved clinically significant improvements (i.e., SE > 85%) compared to those receiving medication alone or placebo.

In a comparison of sleep restriction, with and without an optional daytime nap, to sleep education alone,[18] both sleep restriction conditions produced greater SE increase, with reduced time spent in bed, relative to the control condition. There was no sig-

nificant group difference on actigraphy or PSG measures; TST was reduced for the sleep restriction conditions at post treatment and returned towards baseline values at the 3-month follow-up. There was a mild increase of physiological sleepiness (as measured by the MSLT) but no change on subjective sleepiness. In another investigation,[49] a combination of sleep education plus stimulus control was as effective as sleep education plus relaxation, and more effective than a wait-list control, in older adults with mixed primary and secondary insomnia; there were modest improvements of daytime measures for both active conditions. Two additional studies (reviewed in section 4.3) provided evidence that older adults with insomnia and comorbid medical disorders also benefitted from sleep-specific interventions.[20,27]

4.5 Treatment of Insomnia Among Chronic Hypnotic Users

Four investigations examined the efficacy of psychological and behavioral interventions for insomnia in the context of chronic hypnotic usage, including 2 that were conducted with older adults.[15,22] In a study of 209 chronic hypnotic users,[15] CBT (with an optional medication taper) was associated with improved PSQI scores and reductions of hypnotic use at 3- and 6-month follow ups. A greater percentage of patients treated with CBT for insomnia (39%) relative to no treatment controls (11%) achieved at least a 50% reduction of hypnotic use relative to baseline at the 6-month follow up. A cost-offset analysis revealed that while CBT added to the initial treatment cost, there was a significant cost offset at follow up resulting from a reduction of sleep medication usage. In another study comparing a supervised medication withdrawal program, alone and combined with CBT for insomnia, to CBT alone,[22] all 3 interventions produced significant reductions in both the quantity (90%) and the frequency (80%) of benzodiazepine use, and more patients in the combined approach (85%) were medication free at post-treatment than for those receiving the taper schedule alone (48%) or CBT alone (54%). There were modest changes in sleep patterns during the initial 10-week withdrawal phase, but CBT-treated patients reported greater sleep improvements relative to those receiving the medication withdrawal alone. Several improvements were also reported on secondary measures of insomnia severity (ISI), anxiety (BAI) and depressive (BDI) symptoms.

Two additional studies have examined the impact of chronic hypnotic use on outcome with middle-aged adults. One study[50] found a significant worsening of sleep parameters during medication withdrawal and the addition of relaxation therapy did not attenuate this effect. In a similar study using stimulus control as the main behavioral intervention,[51] stimulus control produced significant improvements on most sleep parameters relative to no additional treatment. There was no difference in sleep outcomes between medicated and nonmedicated patients.

4.6 Validation and Comparative Efficacy of Single and Multifaceted Therapies

Although there are several distinct psychological and behavioral therapies for insomnia, there was a clear trend/preference for investigators to combine 2 or more of these methods when treating insomnia. The most common combination involves an educational (sleep hygiene), behavioral (stimulus control, sleep restriction, relaxation), and a cognitive therapy component, usually referred to as cognitive-behavior therapy. Indeed, 21 studies have evaluated the efficacy of CBT, either with (12 studies) or without relaxation (9 studies), and 5 more studies have used a similar multi-component interventions but without cognitive therapy (see Table 2).

There has been no complete dismantling of CBT to isolate the relative efficacy of each component within the same study. However, comparisons of some components revealed that CBT was superior to relaxation alone in 1 study of primary insomnia[17] and sleep restriction was superior to relaxation at follow-up in another study with older adults.[20] In another study,[52] relaxation was more effective for sleep initiation problems relative to sleep hygiene education alone and a combination of stimulus control plus sleep restriction, whereas the latter combination had greater effects on sleep maintenance variables.

Twelve studies have isolated in a controlled trial at least 1 therapy component such as relaxation, sleep restriction, stimulus control, or paradoxical intention. All 6 studies contrasting relaxation-based interventions (either progressive muscle relaxation or similar procedures) to a control condition, have reported that this single therapy was more effective than wait-list,[27] placebo,[17,20] no treatment,[37,50] and minimal sleep hygiene education control.[52] Two studies showed that sleep restriction was superior to either placebo[20] or sleep hygiene education alone,[18] 1 study found that stimulus control was more effective than a wait-list control,[51] and 1 additional investigation reported that paradoxical intention was superior to a wait-list control for sleep onset insomnia.[24] In spite of the inclusion of a cognitive therapy component in numerous studies, no study has yet evaluated its unique contribution to outcomes.

As in the earlier review paper,[12] criteria developed by the American Psychological Association[53] for defining empirically-validated psychological treatments, were used to determine whether additional evidence was available for each psychological and behavioral interventions (See Table 3). Based on the criteria outlined in our previous review,[12] stimulus control therapy, relaxation training, and paradoxical intention met criteria for well-established psychological treatment for insomnia. With the additional evidence from the present review (indicated by studies in boldface in Table 3), sleep restriction[18,20] and CBT[17,21] would also meet criteria for well-established treatments. Furthermore, additional studies strengthened the level of evidence supporting stimulus control,[51] relaxation,[17,20,27,37] and paradoxical intention.[24]

4.7 Comparisons of Psychological/Behavioral Therapies and Medication

Five controlled studies conducted with primary/psychophysiological insomnia patients have evaluated the impact of psychological/behavioral interventions in comparison to or as an adjunct to hypnotic medications. Two studies evaluated the efficacy of CBT, singly and combined with medication,[21,34] 1 used a medication alone condition as a comparator to psychological treatments,[52] and 2 examined the incremental benefits of adding 1 treatment to the other.[54,55]

In a placebo-controlled comparison of CBT and medication (temazepam) singly and combined[21] (study described in Sections 4.2 and 4.4), all 3 active treatments were more effective than placebo on sleep continuity variables, with a trend for the combined approach to yield better outcomes. These results were corroborated with PSG measures, although the magnitudes of sleep improve-

Table 3—Key Studies Supporting Efficacy Of Psychological And Behavioral Treatments Of Insomnia

Treatment	Study reference	Evidence
Stimulus control*	Espie et al. (1989)[69]	SC > Pla & Rel
	Lacks et al. (1983)[70]	SC > Pla
	Lacks et al. (1983)[71]	SC > Pla
	Morin & Azrin (1987;1988)[72,73]	SC > IT
	Turner & Asher (1979)[74]	SC > Pla
	Riedel et al. (1998)[51]	**SC > WL**
Relaxation*	**Edinger et al. (2001)17[a]**	**Rel > Pla**
	Lichstein et al. ((2001)20[b]	**Rel > Pla**
	Lick & Heffler (1977)[75]	Rel > Pla
	Nicassio et al. (1982)[76]	Rel > No treatment
	Turner & Asher (1979)[74]	Rel > Pla
	Woolfolk & McNulty (1983)[77]	Rel > WL
	Means et al. (2000)[37]	**Rel > No treatment**
	Rybarczyk et al. (2002)[27c]	**Rel > WL**
Paradoxical	Espie et al. (1989)[69]	PI > Pla
Intention*	Turner & Ascher (1979)[74]	PI > Pla
	Broomfield et al. (2003)[24]	**PI > WL**
Sleep restriction*	Friedman et al. (1991)[78]	SR > Pla
	Friedman et al. (2000)[18]	**SR + SHE > SHE**
	Lichstein et al. (2001)[20 b]	**SR > Pla**
EMG	Nicassio et al. (1982)[76]	BF > No treatment
biofeedback†	Sanavio et al. (1990)[79]	BF > WL
	VanderPlate & Eno (1983)[80]	BF > WL
	Freedman & Papsdorf (1976)[81]	BF > Pla
CBT*	**Edinger et al. (2001)[17a]**	**CBT > Rel > Pla**
	Mimeault & Morin (1999)[38]	**CBT > WL**
	Morin et al. (1993)[82]	CBT > WL
	Morin et al. (1999)[21]	**CBT > Pla**
	Morin et al. (2004)[22]	**CBT > Medication taper**
	Perlis et al. (2004)[54]	**CBT > Contact control**
CBT (with Rel)*	**Currie et al. (2000)[25]**	**CBT > WL**
	Currie et al. (2004)[26]	**CBT > WL**
	Espie et al. (2001)[33]	**CBT > WL**
	Jacobs et al. (2004)[34]	**CBT > Pla**
	Morgan et al. (2003)[15]	**CBT > No treatment**
	Rybarczyk et al. (2002)[27c]	**CBT > WL**
Multi-	**Edinger & Sampson (2003)[35]**	**MC > SHE**
component†	**Lichstein et al. (2000)[43]**	**MC > WL**
	Waters et al. (2003)[52]	**MC > SHE**

Note: Citations in boldface are for studies published between 1998 and 2004; all other studies were published prior to this period.
BF = EMG Biofeedback; CBT = Cognitive Behavior Therapy; IT = Imagery Training; PI = Paradoxical Intention; Pla = Placebo; Rel = Relaxation; SC = Stimulus Control; SR = Sleep Restriction; SHE = Sleep Hygiene Education; WL = Wait-List Control; MC=Multicomponent.
[a,b,c]These studies provided evidence supporting more than 1 treatment.
*Well established treatments according to APA criteria for empirically supported treatments. Criteria for well-established treatments require at least 2 between-group design studies demonstrating efficacy in 1 or more of the following ways: I. superior to pill or psychological placebo or to another treatment; or equivalent to an already established treatment in a study with adequate statistical power; II. a large series of single case design experiments (n > 9) demonstrating efficacy as in I; III. the studies must be conducted with treatment manuals; IV. the characteristics of the sample must be well-described; V. the effects must have been demonstrated by at least 2 different investigators or investigatory teams.
†Probably efficacious treatments according to APA criteria for empirically supported treatments. Criteria for probably efficacious treatments are: I. 2 studies showing the treatment is more effective than a waiting-list control group, or II. 1 or more studies meeting the well-established treatment criteria I, III, and IV, but not V, or III. a small series of single case design studies (n > 3) otherwise meeting well-established treatment criteria II, III, and IV.

ments were smaller on PSG than on diary measures. Long-term follow-up data showed that subjects treated with CBT sustained their clinical gains over time, whereas those treated with medication alone did not. The combined approach showed some loss of therapeutic benefits over the follow-up periods, although there was more variability across subjects in that condition. In a similar study design with 63 young and middle-aged adults with sleep onset insomnia[34] (study described in Section 4.2), CBT was shown more effective than medication (zolpidem) and placebo on measures of SOL and SE. All 4 conditions, including placebo, increased their TST, with medication yielding the largest increase in TST (69 min), though this finding was not significantly different from the other groups. There was no significant difference between CBT alone and CBT combined with medication. Sleep changes were well maintained at the 12-month follow up for patients treated with CBT, singly or combined with medication, but no follow up data was available for those treated with medication alone. Data obtained from a Nightcap device showed sleep changes in the same direction as those from diaries, except that no improvement was obtained on any of the measures for the placebo condition.

An investigation of 41 patients treated with estazolam examined the added benefits of muscle relaxation, imagery training, and sleep hygiene education to medication.[55] There was no group difference on any outcome. Significant improvements from baseline to post treatment were obtained on WASO and SE for the relaxation (-17 min and + 9.7% respectively) and imagery training groups (- 33 min and + 7.4%). TST was increased by 34 min (education), 40 min (imagery), and 65 min (muscle relaxation) for the same period but there was no significant group difference. SOL was not changed in any of the groups. Significant changes were obtained from baseline to follow up in all 3 groups on sleep measures and on secondary measures of arousal, self-efficacy, and depressive (but not anxiety) symptoms.

Another study of 30 patients[54] examined the added benefits of modafinil when combined with CBT in the management of primary insomnia. Although there was no significant gain from modafinil in terms of sleep continuity parameters, there were trends suggesting that the addition of modafinil to CBT reduced daytime sleepiness and enhanced compliance with prescribed bedtime. Finally, data from 53 patients with primary insomnia[52] showed that while relaxation was more effective for sleep onset problems and a combination of stimulus control plus sleep restriction had more benefits for sleep maintenance variables, medication (flurazepam) produced the largest improvements on all sleep variables during the initial 2-week intervention.

4.8 Treatment Implementation Methods: Individual, Group, and Self-Help

Treatment was implemented on an individual basis in 22 studies (54%), in a group format in 11 studies (29.7%), and a few additional studies relied on self-help materials with or without additional telephone consultations. An average of 5.7 consultation visits was conducted over a mean treatment period of 6.5 weeks. 1 study directly compared the relative efficacy of CBT implemented in group or individual sessions, or through self-help written materials combined with brief telephone consultations.[56] All 3 groups produced significant improvements of sleep and secondary measures and there was no between group difference on any

measure. A similar study of insomnia in recovered alcoholics also found equivalent outcome between individual CBT and self-help CBT plus telephone consultation.[26] In contrast, 1 study[38] found that the addition of telephone consultation to self-help written material enhanced outcomes at post treatment, but those initial gains tended to disappear at follow up. An internet-based intervention produced greater improvement in several sleep parameters relative to controls,[39] but the attrition rate (24%) was higher than in studies using face-to-face consultation visits. While the majority of reviewed studies have used psychologists or psychology trainees as therapist (with treatment manual), 2 studies examined treatment efficacy as implemented by primary care physicians[36] or by nurse practitioners[33] who had also been trained before the studies; treatment benefits were generally equivalent to those obtained with therapists who had mental-health training.

4.9 Durability of Sleep Improvements

Twenty-six of the 37 reviewed studies reported follow up data of at least 1 month duration after completing treatment (mean duration = 7.7 months; range 1-36 months). The remaining 11 studies reported no follow-up data. As was the case in the initial review, a very robust finding across studies is that treatment-produced changes in sleep parameters are well maintained at short (1-3 month), intermediate (6-month), and long-term (> 12 months) follow-ups. One interesting finding from studies using sleep restriction is that total sleep time may be reduced during the initial intervention, but it is significantly increased at follow up evaluation.[20] Despite fairly robust long-term outcomes, follow-up data must be interpreted cautiously as there are relatively few studies reporting long-term (> 1 year) follow-ups (see Table 2) and, among those that do, attrition rates increase substantially over time.

5.0 CONCLUSIONS

This updated review of treatment studies conducted between 1998 and 2004 provides additional evidence that psychological and behavioral interventions represent an effective treatment option for the management of persistent insomnia. In addition to studies further documenting treatment efficacy for primary insomnia, recent studies indicate that treatment is also effective for insomnia associated with some medical conditions and, to a lesser extent, with psychiatric conditions. Treatment benefits are well sustained over time. There is still limited evidence of clinically meaningful changes beyond reductions of insomnia symptoms (i.e., improved daytime functioning, quality of life).

These findings are consistent with our previous systematic review[12] as well as with other meta-analyses of the efficacy of psychological and behavioral interventions for insomnia.[57-59] For instance, of the 17 most recent treatment studies of primary insomnia, 5 were randomized controlled trials,[17,20,21,33,34] and all 5 yielded additional evidence of significant sleep improvements with psychological and behavioral interventions. Although most of this additional evidence is based on daily sleep diaries, 3 of the 5 key studies included PSG measures and 2 of them reported outcomes that paralleled findings from diary measures. Actigraphy, on the other hand, was not very sensitive for detecting changes in sleep/wake variables in the few studies using this device.

The treatment of comorbid insomnia has received limited attention until recently, perhaps owing to the traditional notion that it would not respond to treatment unless the associated condition was treated first. The present review, as well as recent findings,[60-62] challenge this traditional notion and indicate that insomnia-specific treatment is of benefit even among those whose insomnia is associated with comorbid conditions such as cancer, pain, alcohol abuse, and some psychiatric conditions. Nonetheless, there is a need for additional prospective and randomized controlled studies of comorbid insomnia contrasting outcomes when sleep is or is not directly targeted in treatment.

The treatment of insomnia in older adults is another area previously neglected and for which there was limited evidence to guide practitioners. Nearly 25% of the studies reviewed in this paper focused on older adults. The findings from those studies indicate that older adults with primary insomnia respond to treatment as well as younger and middle-aged adults, although the presence of comorbid medical or psychiatric condition may moderate outcomes.[27,43] A recent meta-analysis [63]also confirmed that treatment effect sizes are comparable for middle-aged and older adults. There is additional evidence that psychological treatment can facilitate hypnotic discontinuation in older adults who are chronic users of hypnotics.[15,22] This is an important finding as older adults are more likely to be long-term hypnotic users which, in some cases, may perpetuate sleep disturbances. Thus, although heterogeneity in diagnosis makes it more difficult to compare studies,[64] such heterogeneity of insomnia samples also enhances generalizability of outcomes.

This review highlights an emerging trend among investigators for combining multiple treatments, which contrasts with the earlier review describing numerous studies comparing treatment efficacy among 2 or more single therapies. Indeed, 26 of the 37 reviewed studies evaluated the efficacy a multi component approaches, including 21 studies using multicomponent therapy, with or without relaxation, and 5 more combining behavioral interventions (e.g., stimulus control and sleep restriction) but without cognitive therapy. Although the reason for this shift in paradigm is not entirely clear, the use of multi-component approaches is more likely, at least on a clinical basis, to address the different facets/perpetuating factors of insomnia.[11]

Whether CBT produces outcome that is superior to single therapies remains largely unexplored. The few comparative studies available show that outcome is superior for CBT, stimulus control, and for sleep restriction relative to relaxation alone,[17,20,27,49] however, there has been no complete dismantling of CBT to isolate the relative efficacy of each component. Furthermore, although some findings[65] suggest that change in beliefs and attitudes is an important mediator of long-term outcomes, there has been no direct controlled evaluation to isolate the relative contribution of cognitive therapy.

Our previous review had identified 3 treatments as well-validated and 2 more as probably efficacious according to criteria set by the American Psychological Association.[53] This updated review provides further evidence supporting stimulus control, relaxation, paradoxical intention as well validated therapies, and new evidence to upgrade sleep restriction and CBT from probably efficacious to well-established treatments.

Although there is evidence supporting the efficacy of psychological and behavioral treatment for insomnia, there is still little information about the specificity of this treatment modality and the active therapeutic mechanisms responsible for sleep improve-

ments. With a few notable exceptions using attention-placebo conditions,[17,20] most CBT trials have used wait-list control groups, precluding the unequivocal attribution of treatment effects to any specific ingredient of psychological and behavioral treatment. The lack of a pill-placebo control equivalent in psychological outcome research makes it difficult to determine what percentage of the variance in outcomes is due to specific therapeutic ingredients (i.e., restriction of time in bed, cognitive restructuring), the measurement process (i.e., self-monitoring), or to non-specific factors (e.g., therapist attention, patients' expectations).

An important limitation noted in the previous review that is still evident in recent studies is the limited evidence documenting the clinical significance of outcomes beyond insomnia symptom reductions (i.e., reduced morbidity, improved quality of life). There is a need for broadening the scope of outcome measures[66] and for standardizing assessment methodology in insomnia research,[64,67] Furthermore, even for patients meeting criteria for what might be considered a clinically meaningful change, many such treatment responders reach a plateau and continue showing residual sleep disturbances after treatment and may remain at risk for relapse. There is a need to develop and validate more potent interventions that would increase the rate of patients reaching full remission.[9] Ongoing studies are currently examining optimal treatment dosage, treatment combination involving medication, and maintenance therapies.

A related issue is that most of the outcome evidence currently available is about improving sleep initiatiation and sleep continuity parameters, with essentially no information about the impact of treatment on more qualitative aspects of sleep, i.e., non-restorative sleep. Although this qualitative feature is part of the standard insomnia definition, no study has of yet examined the impact of psychological treatment on this variable.

Proper implementation of psychological and behavioral therapies usually requires more time than prescribing a hypnotic medication, which may represent an important barrier to using such interventions in clinical practice. Nonetheless, several studies have documented the benefits of cost-effective implementation models using nurse practitioners, group therapy, or self-help materials to complement therapist-guided intervention. Whereas such implementation models are likely to make treatment more readily available, adequate therapist training remains an important consideration in using CBT effectively to optimize outcome. Additional studies examining the relative cost-effectiveness of different insomnia interventions would be warranted[68]

In summary, this updated review provides additional evidence supporting the use of psychological and behavioral interventions for primary insomnia, for insomnia associated with medical or psychiatric conditions, and for insomnia in older adults. Additional research is still needed to develop and validate treatment algorithms that would optimize outcomes and reduce morbidity; clinical research that would examine treatment mechanisms, mediators and moderators of outcomes is also warranted; and, additional effectiveness trials are particularly needed to document outcomes in unselected patients seeking treatment in various clinical settings (e.g., primary care). Finally, an important challenge for the future will be to disseminate more efficiently the available evidence to health-care providers and translate that evidence into meaningful clinical guidelines in order to ensure a more widespread use of validated therapies.

REFERENCES

1. Ohayon M. Epidemiology of insomnia: what we know and what we still need to learn. Sleep Med Rev 2002;6:97-111
2. Breslau N, Roth T, Rosenthal L, Andreski P. Sleep disturbance and psychiatric disorders: a longitudinal epidemiological study of young adults. Biol Psychiatry 1996;39:411-18
3. Ford DE, Kamerow DB. Epidemiologic study of sleep disturbances and psychiatric disorders. An opportunity for prevention? JAMA 1989;262:1479-84
4. Simon GE, VonKorff M. Prevalence, burden, and treatment of insomnia in primary care. Am J Psychiatry 1997;154:1417-23
5. Walsh JK. Clinical and socioeconomic correlates of insomnia. J Clin Psychiatry 2004;65:13-9
6. American Academy of Sleep Medicine. International classification of sleep disorders: Diagnostic and coding manual. (2nd ed). Westchester, IL: American Academy of Sleep Medicine, 2005
7. American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorders 4th ed, Washington, DC: American Psychiatric Association, 2000
8. Edinger JD, Bonnet MH, Bootzin RR, et al. Derivation of research diagnostic criteria for insomnia: report of an American Academy of Sleep Medicine Work Group. Sleep 2004;27:1567-96
9. National Institutes of Health. National Institutes of Health State of the Science Conference Statement. Manifestations and management of chronic insomnia in adults. Sleep 2005; 28:1049-57
10. Perlis ML, Lichstein KL, eds. Treating sleep disorders: Principles and practice of behavioral sleep medicine. Hoboken, NJ: John Wiley & Sons, Inc, 2003
11. Morin CM, Espie CA. Insomnia: a clinical guide to assessment and treatment). New York: Kluwer Academic/Plenum Publishers, 2003
12. Morin CM, Hauri PJ, Espie CA, Spielman AJ, Buysse DJ, Bootzin RR. Nonpharmacologic treatment of chronic insomnia. An American Academy of Sleep Medicine review. Sleep 1999;22:1134-56
13. Sackett D. Rules of evidence and clinical recommendation. Can J Cardiol 1993;9:487-9
14. Backhaus J, Hohagen F, Voderholzer U, Riemann D. Long-term effectiveness of a short-term cognitive-behavioral group treatment for primary insomnia. Eur Arch Psychiatry Clin Neurosci 2001;251:35-41
15. Morgan K, Dixon S, Mathers N, Thompson J, Tomeny M. Psychological treatment for insomnia in the management of long-term hypnotic drug use: a pragmatic randomised controlled trial. Br J Gen Pract 2003;53:923-8
16. Simeit R, Deck R, Conta-Marx B. Sleep management training for cancer patients with insomnia. Support Care Cancer 2004;12:176-183
17. Edinger JD, Wohlgemuth WK, Radtke RA, Marsh GR, Quillian RE. Cognitive behavioral therapy for treatment of chronic primary insomnia: A randomized, controlled trial. JAMA 2001;285:1856-64
18. Friedman L, Benson K, Noda A, et al. An actigraphic comparison of sleep restriction and sleep hygiene treatments for insomnia in older adults. J Geriatr Psychiatry Neurol 2000;13:17-27
19. Guilleminault C, Palombini L, Poyares D, Chowdhuri S. Chronic insomnia, premenopausal women and sleep disordered breathing: part 2. Comparison of nondrug treatment trials in normal breathing and UARS post menopausal women complaining of chronic insomnia. J Psychosom Res 2002;53:617-23
20. Lichstein KL, Riedel BW, Wilson NM, Lester KW, Aguillard RN. Relaxation and sleep compression for late-life insomnia: a placebo controlled trial. J Consult Clin Psychol 2001;69:227-39
21. Morin CM, Colecchi C, Stone J, Sood R, Brink D. Behavioral and pharmacological therapies for late-life insomnia: a randomized clinical trial. JAMA 1999;281:991-9
22. Morin CM, Bastien CH, Guay B, Radouco-Thomas M, Leblanc J, Vallières A. Randomized clinical trial of supervised tapering and cognitive-behavior therapy to facilitate benzodiazepine discon-

tinuation in older adults with chronic insomnia. Am J Psychiatry 2004;161:332-42
23. Viens M, De Koninck J, Mercier P, St-Onge M, Lorrain D. Trait anxiety and sleep-onset insomnia: evaluation of treatment using anxiety management training. J Psychosom Res 2003;54:31-7
24. Broomfield NM, Espie CA. Initial insomnia and paradoxical intention: An experimental investigation of putative mechanisms using subjective and actigraphic measurement of sleep. Behav Cogn Psychoth 2003;31:313-24
25. Currie SR, Wilson KG, Pontefract AJ, deLaplante L. Cognitive-behavioral treatment of insomnia secondary to chronic pain. J Consult Clin Psychol 2000;68:407-16
26. Currie SR, Clark S, Hodgins DC, El-Guebaly N. Randomized controlled trial of brief cognitive-behavioural interventions for insomnia in recovering alcoholics. Addiction 2004;99:1121-32
27. Rybarczyk B, Lopez M, Benson R, Alsten C, Stepanski E. Efficacy of two behavioral treatment programs for comorbid geriatric insomnia. Psychol Aging 2002;17:288-98
28. Morin C. Insomnia: Psychological assessment and management. New York: Guilford Press, 1993
29. Buysse DJ, Reynolds CF, Monk TH, Berman SR, Kupfer DJ. The Pittsburgh Sleep Quality Index: a new instrument for psychiatric practice and research. Psychiatry Res 1989;28:193-213
30. Beck AT, Steer RA, Garbin MG. Psychometric properties of the Beck Depression Inventory: Twenty-five years of evaluation. Clin Psychol Rev 1988;8:77-100
31. Spielberger C. Manual of the State-Trait Anxiety Inventory (Form Y). Palo Alto, CA: Consulting Psychologists Press, 1983
32. Smets EM, Garssen B, Bonke B, DeHaes JC. The multidimensional fatigue inventory (MFI) psychometric qualities of an instrument to assess fatigue. J Psychosom Res 1995;39:315-25
33. Espie CA, Inglis SJ, Tessier S, Harvey L. The clinical effectiveness of cognitive behaviour therapy for chronic insomnia: implementation and evaluation of a sleep clinic in general medical practice. Behav Res Ther 2001;39:45-60
34. Jacobs GD, Pace-Schott EF, Stickgold R, Otto MW. Cognitive behavior therapy and pharmacotherapy for insomnia. Arch Intern Med 2004;164:1888-1896
35. Edinger JD, Sampson WS. A primary care "friendly" cognitive behavioral insomnia therapy. Sleep 2003;26:177-82
36. Baillargeon L, Demers M, Ladouceur R. Stimulus-control: nonpharmacologic treatment for insomnia. Can Fam Physician 1998;44:73-9
37. Means MK, Lichstein KL, Epperson MT, Johnson CT. Relaxation therapy for insomnia: nighttime and day time effects. Behav Res Ther 2000;38:665-78
38. Mimeault V, Morin CM. Self-help treatment for insomnia: bibliotherapy with and without professional guidance. J Consult Clin Psychol 1999;67:511-9
39. Ström L, Pettersson R, Andersson G. Internet-based treatment for insomnia: a controlled evaluation. J Consult Clin Psychol 2004;72:113-20
40. Hryshko-Mullen AS, Broeckl LS, Haddock CK, Peterson AL. Behavioral treatment of insomnia: the Wilford Hall Insomnia Program. Mil Med 2000;165:200-207.
41. Vincent NK, Hameed H. Relation between adherence and outcome in the group treatment of insomnia. Behav Sleep Med 2004;1:125-39
42. Davidson JR, Waisberg JL, Brundage MD, MacLean AW. Nonpharmacologic group treatment of insomnia: a preliminary study with cancer survivors. Psychooncology 2001;10:389-97
43. Lichstein KL, Wilson NM, Johnson CT. Psychological treatment of secondary insomnia. Psychol Aging 2000;15:232-40
44. Dashevsky B, Kramer M. Behavioral treatment of chronic insomnia in psychiatrically ill patients. J Clin Psychiatry 1998;59:693-9
45. Dopke CA, Lehner RK, Wells AM. Cognitive-behavioral group therapy for insomnia in individuals with serious mental illnesses: a preliminary evaluation. Psychiatr Rehabil J 2004;27:235-42
46. Perlis ML, Sharpe M, Smith MT, Greenblatt D, Giles D. Behavioral treatment of insomnia: treatment outcome and the relevance of medical and psychiatric morbidity. J Behav Med 2001;24:281-96
47. Perlis M, Aloia M, Millikan A, Boehmler J, Smith M, Greenblatt D, Giles D. Behavioral treatment of insomnia: a clinical case series study. J Behav Med 2000;23:149-61
48. Verbeek I, Schreuder K, Declerck G. Evaluation of short-term nonpharmacological treatment of insomnia in a clinical setting. J Psychosom Res 1999;47:369-83
49. Pallesen S, Nordhus IH, Kvale G, Nielsen GH, Havik OE, Johnsen BH, Skjotskift S. Behavioral treatment of insomnia in older adults: an open clinical trial comparing two interventions. Behav Res Ther 2003;41:31-48
50. Lichstein KL, Peterson BA, Riedel BN, Means MK, Epperson MT, Aguillard RN. Relaxation to assist sleep medication withdrawal. Behav Modif 1999;23:379-402
51. Riedel B, Lichstein KL, Peterson BA, Means MK, Epperson MT, Aguillarel RN. A comparison of the efficacy of stimulus control for medicated and nonmedicated insomniacs. Behav Modif 1998;22:3-28
52. Waters WF, Hurry MJ, Binks PG, et al. Behavioral and hypnotic treatments for insomnia subtypes. Behav Sleep Med 2003;1:81-101
53. Chambless DL, Hollon SD. Defining empirically supported therapies. J Consult Clin Psychol 1998;66:7-18
54. Perlis ML, Smith MT, Orff H, Enright T, Nowakowski S, Jungquist C, Plotkin K. The effects of modafinil and cognitive behavior therapy on sleep continuity in patients with primary insomnia. Sleep 2004;15:715-25
55. Rosen RC, Lewin DS, Goldberg L, Woolfolk RL. Psychophysiological insomnia: combined effects of pharmacotherapy and relaxation-based treatments. Sleep Med 2000;1:279-88
56. Bastien CH, Morin CM, Ouellet MC, Blais FC, Bouchard S. Cognitive-behavioral therapy for insomnia: comparison of individual therapy, group therapy, and telephone consultations. J Consult Clin Psychol 2004;72:653-59
57. Smith MT, Perlis ML, Park A, Smith MS, Pennington J, Giles D, Buysse D. Comparative meta-analysis of pharmacotherapy and behavior therapy for persistent insomnia. Am J Psychiatry 2002;159:5-11
58. Murtagh DR, Greenwood KM. Identifying effective psychological treatments for insomnia: a meta-analysis. J Consult Clin Psychol 1995;63:79-89
59. Morin CM, Culbert JP, Schwartz SM. Nonpharmacological interventions for insomnia: a meta-analysis of treatment efficacy. Am J Psychiatry 1994;151:1172-80
60. Rybarczyk B, Stepanski E, Fogg L, Lopez M, Barry P, Davis A. A placebo-controlled test of cognitive-behavioral therapy for comorbid insomnia in older adults. J Consult Clin Psychol 2005;73:1164-74
61. Savard J, Simard S, Ivers H, Morin CM. Randomized study on the efficacy of cognitive-behavioral therapy for insomnia secondary to breast cancer, part I: Sleep and psychological effects. J Clin Oncol 2005;23:6083-96.
62. Smith MT, Huang MI, Manber R. Cognitive behavior therapy for chronic insomnia occurring within the context of medical and psychiatric disorders. Clin Psychol Rev 2005;25:559-92
63. Irwin MR, Cole JC, Nicassio PM. Comparative meta-analysis of behavioral interventions for insomnia and their efficacy in middle-aged adults and in older adults 55+ years of age. Health Psychol 2006;25:3-14
64. Martin JL, Ancoli-Israel S. Assessment and diagnosis of insomnia in non-pharmacological intervention studies. Sleep Med Rev 2002;6:379-406
65. Morin CM, Blais F, Savard J. Are changes in beliefs and attitudes about sleep related to sleep improvements in the treatment of in-

somnia? Behav Res Ther 2002;40:741-52
66. Morin CM. Measuring outcome in randomized clinical trials of insomnia therapies. Sleep Med Rev 2003;7:263-79
67. Buysse DJ, Ancoli-Israel S, Edinger JD, Lichstein KL, Morin CM. Recommendations for a standard research assessment of insomnia. Sleep. Sleep 2006 29:1155-73.
68. Martin SA, Aikens JE, Chervin RD. Toward cost-effectiveness analysis in the diagnosis and treatment of insomnia. Sleep Med Rev 2004;8:63-72
69. Espie CA, Lindsay WR, Brooks DN, Hood EM, Turvey T. A controlled comparative investigation of psychological treatments for chronic sleep-onset insomnia. Behav Res Ther 1989;27:79-88
70. Lacks P, Bertelson AD, Sugerman J, Kunkel J. The treatment of sleep-maintenance insomnia with stimulus-control techniques. Behav Res Ther 1983;21:291-95
71. Lacks P, Bertelson AD, Gans L, Kunkel J. The effectiveness of three behavioral treatments for different degrees of sleep onset insomnia. Behav Ther 1983;14:593-605
72. Morin CM, Azrin NH. Stimulus control and imagery training in treating sleep-maintenance insomnia. J Consult Clin Psychol 1987;55:260-62
73. Morin CM, Azrin NH. Behavioral and cognitive treatments of geriatric insomnia. J Consult Clin Psychol 1988;56:748-753
74. Turner RM, Ascher LM. Controlled comparison of progressive relaxation, stimulus control, and paradoxical intention therapies for insomnia. J Consult Clin Psychol 1979;47:500-08
75. Lick JR, Heffler D. Relaxation training and attention placebo in the treatment of severe insomnia. J Consult Clin Psychol 1977;45:153-161
76. Nicassio PM, Boylan MB, McCabe TG. Progressive relaxation, EMG biofeedback and biofeedback placebo in the treatment of sleep-onset insomnia. Br J Med Psychol 1982;55:159-66
77. Woolfolk RL, McNulty TF. Relaxation treatment for insomnia: a component analysis. J Consult Clin Psychol 1983;51:495-503
78. Friedman L, Bliwise DL, Yesavage JA, Salom S. A preliminary study comparing sleep restriction and relaxation treatments for insomnia in older adults. J Gerontol 1991;46:1-8
79. Sanavio E, Vidotto G, Bettinardi O, Roletto T, Zorzi M. Behavior therapy for DIMS: Comparison of three treatment procedures with follow-up. Behav Psychother 1990;18:151-67
80. VanderPlate C, Eno EN. Electromyograph biofeedback and sleep onset insomnia: Comparison of treatment and placebo. Behav Eng 1983;8:146-53
81. Freedman R, Papsdorf JD. Biofeedback and progressive relaxation treatment of sleep-onset insomnia: a controlled, all-night investigation. Biofeedback Self Regul 1976;1:253-71
82. Morin CM, Kowatch R, Barry T, Walton E. Cognitive-behavior therapy for late-life insomnia. J Consult Clin Psychol 1993;61:137-46

Appendix A.

Studies reviewed but excluded

A. Baillargeon L, Landreville P, Verreault R, Beauchemin JP, Grégoire JP, Morin CM. Discontinuation of benzodiazepines among older insomniac adults treated through cognitive-behavioral therapy combined with gradual tapering: a randomized trial. CMAJ 2003;169:1015-20. [no sleep data]

B. Espie CA, Inglis SJ, Harvey L. Predicting clinically significant response to cognitive behavior therapy for chronic insomnia in general medical practice: analyses of outcome data at 12 months posttreatment. J Consul Clin Psychol 2001;69:58-66. [secondary analysis of another paper included in the review]

C. Greeff AP, Conradie WS. Use of progressive relaxation training for chronic alcoholics with insomnia. Psychol Rep 1998;82:407-12. [only outcome measure, sleep quality, obtained from a single 10-point rating scale]

D. Gunning M, Espie CA. Psychological treatment of reported sleep disorder in adults with intellectual disability using a multiple baseline design. J Intellect Disabil Res 2003;47:191-202. [sample size smaller than 10]

E. Hajak G, Bandelow B, Zulley J, Pittrow D. As needed pharmacotherapy combined with stimulus control treatment in chronic insomnia--Assessment of a novel intervention strategy in a primary care setting. Ann Clin Psychiatry 2002;14:1-7. [main intervention was pharmacological, with optional use of stimulus control procedures]

F. Harvey AG, Farrell C. The efficacy of a Pennebaker-like writing intervention for poor sleepers. Behav Sleep Med 2003;1:115-24. [not a clinical treatment study; no insomnia diagnosis]

G. Harvey AG, Payne S. The management of unwanted pre-sleep thoughts in insomnia: distraction with imagery versus general distraction. Behav Res Ther 2002;40:267-77. [not a clinical treatment study]

H. Krakow B, Johnston L, Melendrez D et al. An open-label trial of evidence-based cognitive behavior therapy for nightmares and insomnia in crime victims with PTSD. Am J Psychiatry 2001;158:2043-7. [primary diagnosis of post traumatic stress disorder with primary outcome measures of nightmare frequency; no insomnia diagnosis]

I. Krakow B, Melendrez D, Lee SA, Warner TD, Clark JO, Sklar D. l. Refractory insomnia and sleep-disordered breathing, a pilot study. Sleep Breath 2004;8:15-29. [main sleep diagnosis was sleep-related breathing disorder, not insomnia]

J. Lundh LG, Hindmarsh H. Can meta-cognitive observation be used in the treatment of insomnia? A pilot study of a cognitive-emotional self-observation task. Behav Cogni Psychoth 2002;30:233-36. [no insomnia diagnosis]

K. Morawetz D. Depression and insomnia: Which comes first? Australian Journal of Counseling Psychology 2001;3:19-24. [no insomnia diagnosis; did not use sleep diaries or other recognized measures to evaluate outcome]

L. Morgan K, Thompson J, Dixon S, Tomeny M, Mathers N. Predicting longer-term outcomes following psychological treatment for hypnotic-dependent chronic insomnia. J Psychosom Res 2003;54:21-9. [secondary analysis of another paper included in the review]

M. Quesnel C, Savard J, Simard S, Ivers H, Morin CM. Efficacy of cognitive-behavioral therapy for insomnia in women treated for non-metastatic breast cancer. J Consul Clin Psychol 2003;71:189-200. [final sample size smaller than 10]

N. Riedel BW, Lichstein KL. Strategies for evaluating adherence to sleep restriction treatment for insomnia. Behav Res Ther 2001;39:201-12. [secondary analysis of another paper included in the review]

O. Tang NK, Harvey AG. Correcting distorted perception of sleep in insomnia: a novel behavioural experiment? Behav Res Ther 2004;42:27-39. [not a clinical treatment study]

P. Verbeek I, Declerck G, Neven AK, Coenen A. Sleep information by telephone: Callers indicate positive effects on sleep problems. Sleep and Hypnosis 2002 ;4:47-51. [not a clinical treatment study; no diagnosis of insomnia]

RAPID PUBLICATION

Randomized Clinical Effectiveness Trial of Nurse-Administered Small-Group Cognitive Behavior Therapy for Persistent Insomnia in General Practice

Colin A. Espie, PhD[1]; Kenneth M. A. MacMahon, PhD[1]; Heidi-Louise Kelly, DClinPsy[1]; Niall M. Broomfield, PhD[1]; Neil J. Douglas, MD[2]; Heather M. Engleman, PhD[2]; Brian McKinstry, MD[3]; Charles M. Morin, PhD[4]; Andrew Walker, PhD[5]; Philip Wilson, FRCGP[6]

[1]University of Glasgow Sleep Research Laboratory, Scotland, UK; [2]Department of Sleep & Respiratory Medicine, University of Edinburgh, Scotland, UK; [3]Community Health Sciences: General Practice Section, University of Edinburgh, Scotland, UK; [4]Ecole de Psychologie, Universite Laval, Quebec City, Canada; [5]Robertson Centre for Biostatistics, University of Glasgow, Scotland, UK; [6]Section of General Practice and Primary Care, University of Glasgow, Scotland, UK

Study Objectives: Persistent insomnia, although very common in general practice, often proves problematic to manage. This study investigates the clinical effectiveness and the feasibility of applying cognitive behavior therapy (CBT) methods for insomnia in primary care.

Design: Pragmatic randomized controlled trial of CBT versus treatment as usual.

Setting: General medical practice.

Participants: Two hundred one adults (mean age, 54 years) randomly assigned to receive CBT (n = 107; 72 women) or treatment as usual (n = 94; 65 women).

Intervention: CBT comprised 5 sessions delivered in small groups by primary care nurses. Treatment as usual comprised usual care from general practitioners.

Measurements and Results: Assessments were completed at baseline, after treatment, and at 6-month follow-up visits. Sleep outcomes were appraised by sleep diary, actigraphy, and clinical endpoint. CBT was associated with improvements in self-reported sleep latency, wakefulness after sleep onset, and sleep efficiency. Improvements were partly sustained at follow-up. Effect sizes were moderate for the index variable of sleep efficiency. Participants receiving treatment as usual did not improve. Actigraphically estimated sleep improved modestly after CBT, relative to no change in treatment as usual. CBT was also associated with significant positive changes in mental health and energy/vitality. Comorbid physical and mental health difficulties did not impair sleep improvement following CBT.

Conclusion: This study suggests that trained and supervised nurses can effectively deliver CBT for insomnia in routine general medical practice. Treatment response to small-group service delivery was encouraging, although effect sizes were smaller than those obtained in efficacy studies. Further research is required to consider the possibility that CBT could become the treatment of first choice for persistent insomnia in primary healthcare.

Keywords: Insomnia, sleep, treatment, primary care, psychological intervention

Citation: Espie C; MacMahon KMA; Kelly HL et al. Randomized clinical effectiveness trial of nurse-administered small-group cognitive behavior therapy for persistent insomnia in general practice. *SLEEP* 2007;30(5):574-584

SLEEP DISTURBANCE IS THE MOST COMMON SYMPTOM OF MENTAL ILLNESS, BEING MORE COMMON THAN WORRY AND TWICE AS COMMON AS ANXIETY or depressive symptoms. Moreover, in a recent UK psychiatric morbidity study,[1] this finding held for men and women of any age or ethnic group in any region. Epidemiologic studies report the prevalence of insomnia disorder at 10% to 12%, with older adult rates at greater than 20%.[2,3] One fifth of patients consulting in primary care have insomnia.[4] Typically, difficulty initiating or maintaining sleep, or both initiating and maintaining sleep, is associated with reduced daytime alertness and productivity, poorer quality of life, impaired relationships, and increased ill health.[5-9] Two meta-analyses have reported preexisting sleep disturbance as the largest, potentially treatable, risk factor for first-episode depression and for recurrence of depression.[10,11]

Despite such findings, persistent insomnia often goes unrecognized, and care management is poorly developed.[12] Benzodiazepine hypnotics and sedative antidepressants are commonly prescribed in clinical practice, although long-term outcome data are relatively sparse,[13,14] and, although the benzodiazepine receptor agonists confer some advantages in the management of acute insomnia, there is thus far limited evidence that they are preferable for the treatment of persistent insomnia.[15] In short, the management of chronic insomnia represents a very significant gap in the clinical armamentarium.

Cognitive behavior therapy (CBT) offers 1 promising approach. Insomnia often arises from psychological factors such as conditioned arousal, maladaptive sleep habits and sleep schedules, dysfunctional thinking about sleep and its consequences, and sleep preoccupation.[16] This behavioral phenotype may be similar whether insomnia is primary or presenting in the context of psychiatric problems.[2,17] Although 3 meta-analyses have demonstrated clear benefit,[18-20] CBT efficacy trials have recruited largely among media-solicited participants, perhaps excluding patients with complex presentations. Such studies have conformed more to the traditions of clinical efficacy research, where there is an

Disclosure Statement

This is not an industry supported study. Dr. Douglas has received industry support from ResMed and is on the International Medical Advisory Board of ResMed. Dr. Morin has received research support from Sanofi-Aventis; has been a consultant to Sepracor, Pfizer, Neurocrine, Takeda, and Shire Biochem; and has participated in speaking engagements for Takeda, Sanofi-Aventis, and Merck. Dr. Walker has participated in speaking engagements for Schering-Plough, Novartis, and Boehringer-Ingelheim. Drs. Espie, MacMahon, Kelly, Broomfield, Engleman, McKinstry, and Wilson have indicated no financial conflicts of interest.

Submitted for publication August, 2006
Accepted for publication December, 2006

Address correspondence to: Professor Colin A. Espie, University of Glasgow Sleep Research Laboratory, Southern General Hospital, 1345 Govan Road, Glasgow G51 4TF, Scotland, UK; Tel: +44 (0)141-232-7700; Fax: +44 (0)141-232-7697; Email: c.espie@clinmed.gla.ac.uk

Table 1—Inclusion and Exclusion Criteria Based Primarily Upon ICSD-R/ DSM-IV

Inclusion Criteria
- Aged ≥ 18 years
- Referred by general practitioner
- Living in the community in Glasgow or Edinburgh area
- Difficulty initiating and/or maintaining sleep, comprising SOL ≥ 30 minutes and/or WASO ≥ 30 minutes, 3 or more nights per week
- Present sleep complaint for at least 6 months
- Negative complaint of insomnia impact (eg, fatigue, impaired mood)

Exclusion Criteria
- Deteriorating health or dementia
- Incapacitating pain or illness
- Untreated mental health problems
- Untreated other sleep problems

ICSD-R refers to the *International Classification of Sleep Disorders-Revised*[5]; DSM-IV, *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition*[6]; SOL sleep-onset latency; WASO, wake time after sleep-onset.

emphasis on sample homogeneity, the exclusion of comorbidities, measurement reliability, and the management of other factors that are known to influence extraneous variance. Consequently, we do not know whether it is clinically effective or feasible to translate CBT to primary care. Effectiveness studies, by way of contrast, emphasize validity and generalizability to "real-world" settings by accessing populations and following procedures that reflect more typical clinical practice. Results from a preliminary clinical effectiveness study have suggested that improvements with CBT delivered by primary care nurses may be obtained.[21] The present report is a formal intention-to-treat evaluation of this model.

METHODS

Aims and Objectives

The aim of the study was to test the effectiveness of CBT for the treatment of persistent insomnia in the "real-world" primary care setting. The major research questions were "Is CBT superior to treatment as usual (TAU) in reducing chronic sleep disturbance?", "Are observed changes in sleep pattern and sleep quality durable?", and "Are there predictors of good outcome, or contraindications to the application of CBT, for insomnia in general practice?"

Design

The study conformed to a pragmatic, randomized trial design following CONSORT guidelines. CBT was compared with TAU, this being an appropriate control for a clinical effectiveness study. Major assessments were at baseline, after treatment, and at follow-up 6 months later.

Participants

Potential participants were patients attending an appointment with their general practitioner (GP), or who were on their GP's prescribing list for a sleep medication, during the period June 2001 to July 2003. One hundred and four GPs in 19 practices in Glasgow, West Lothian, and Edinburgh identified participants. Eligibility criteria are described in Table 1. Participants had to satisfy criteria based primarily upon International Classification of Sleep Disorders -revised/Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition[5,6] and standard quantitative criteria. Consistent with this type of trial, exclusions were limited to new, untreated, or serious disorders or substance abuse problems that would make participation impractical or clinically inadvisable. Patients with physical or psychological problems were not excluded. Similarly, being on sleep (or other) medications was not an exclusion criterion.

Potential participants were notified of the study by their GP and through posters in clinic waiting areas and explanatory leaflets. Some GPs conducted record searches before circulating information to patients with known insomnia problems. All prospective participants were then "referred" by their GP using a simple form on which GPs marked (√) against each study criterion (Table 1). Participants had the opportunity to discuss the research with a member of the research team and to reconsult with their GP prior to consenting. All gave written informed consent. The protocol was approved by local research ethics committees. Based on previous work,[21] a sample size of 240 would have 90% power to show a 0.5 SD difference between treatments at an α level of 0.05. This was based on CBT achieving a reduction, in minutes of total wake time, at least 50% more than TAU.

After baseline assessment and anonymization, participants were randomly assigned by an administrator in an independent research group using a computer-generated random list of numbers. Allocation was strictly in order of completion of baseline data, independent of participating GP practice or location. Randomization information was kept in a locked cabinet and was inaccessible to researchers. Because of the nature of the intervention, it was not possible to blind participants or therapists to CBT and TAU allocations.

Measures

Potential participants were screened by telephone interview. Data were collected on sleep history, including diagnostic criteria, as well as medical and psychiatric history. A comprehensive face-to-face interview then obtained a more detailed history. Interview was supplemented by completion of the Pittsburgh Sleep Quality Index (PSQI),[22] the Hospital Anxiety and Depression Scale (HADS),[23] and the Epworth Sleepiness Scale (ESS).[24]

Subjective sleep pattern was assessed using a sleep diary,[25] completed for 2 weeks at each of 3 assessment points; baseline, posttreatment, and follow-up. Such diaries are the staple tool of insomnia-assessment practice[16] and offer a valid relative index of sleep disturbance, particularly when used as repeated measures.[26,27] Fourteen nights is an adequate sampling period.[28] Items "how long did it take you to fall asleep last night" (sleep-onset latency: SOL) and "how long were you awake in total last night, after you first fell asleep?" (wake time after sleep onset: WASO) assessed the central insomnia dimensions of difficulty initiating and maintaining sleep. Participants were advised to estimate WASO between initial sleep onset and rising from bed. The diary also inquired about bedtime and rising time, from which total time in bed (TIB), and then sleep efficiency percentage (SE) was

Table 2—Summary Content of the Cognitive Behavior Therapy Program

Session 1 Sleep Information
Aim: To learn about normal sleep processes and about sleep disorders
- to understand the need for sleep and its functions
- to understand sleep pattern and how it varies during the lifetime
- to understand sleep as a process with stages and phases
- to understand factors that adversely affect sleep pattern and sleep quality
- to understand the effects of sleep loss
- to understand the concept of insomnia and how it can be measured
- to understand personal sleep histories and patterns in the above context
- to begin to correct misunderstandings about sleep and sleeplessness

Session 2 Sleep Hygiene & Relaxation
Aim: To introduce practical steps toward developing a healthy sleep pattern without recourse to drugs
- to create a bedroom environment that is comfortable for sleep
- to take regular exercise that promotes fitness and enhances sleep
- to develop a stable and appropriate diet
- to reduce the undesirable effects of caffeine upon sleep
- to moderate alcohol consumption and eliminate "night caps"
- to learn relaxation skills to apply at home and in bed

Session 3 Sleep Scheduling
Aim: To reshape sleep patterns to correspond with individual sleep needs and to strengthen sleep rhythms
- to develop a good presleep routine
- to distance waking activities (eg, watching TV) from the bedroom environment
- to establish a strong bed-sleep connection
- to eliminate wakefulness from bed (rising if not asleep within around 15 minutes)
- to define restricted parameters for the individual's sleep period
- to increase sleep efficiency through scheduling sleep in relation to current total sleep
- to eliminate daytime napping
- to establish a stable night-to-night sleep pattern, rising at the same time every day
- to encourage and support people in changing their sleep routines

Session 4 Cognitive approaches
Aim: To learn ways of reducing mental alertness, repetitive thoughts, and anxiety that interfere with sleep
- to identify thought patterns that interfere with sleep
- to develop accurate beliefs and attitudes about sleep
- to prepare mentally for bed by putting the day to rest
- to learn thought distraction and imagery techniques
- to reduce efforts to control sleep and allow it to happen naturally
- to utilize these techniques to combat intrusive thoughts
- to encourage and support people in changing their mental approach
- to further adjust sleep schedules to maintain sleep efficiency

Session 5 Developing a strong & natural sleep pattern
Aim: To integrate advice from previous sessions and to maintain implementation at home
- to systematically rehearse elements of program
- to address implementation problems experienced
- to further adjust sleep schedules to maintain sleep efficiency
- to encourage and support people in maintaining their new sleep routines
- to encourage and support people in maintaining their new mental approach
- to learn relapse-prevention approaches if a sleep problem recurs

calculated (100 – [{SOL + WASO/ TIB} × 100]). Participants were trained to complete sleep diaries using established accuracy criteria.[29]

Because movement correlates with wakefulness and lack of movement with sleep,[30,31] wrist actigraphy was used to objectively estimate sleep for 14 nights before and after treatment. Actigraphs are small nonintrusive devices that record movement information by means of an accelerometer-microprocessor link. In this study, actigraphs (Cambridge Neurotechnology®, AW-4; Cambridge Neurotechnology Ltd., Cambridge, UK) were worn 24 hours per day on the nondominant wrist. An algorithm (maximum sampling frequency 32 Hz, recording all movement over 0.05 g., filters set 3-11 Hz) enabled proprietary Sleepwatch® software (Cambridge Neurotechnology Ltd) to estimate the sleep parameters SOL, WASO, and SE using 1-minute epochs. In the United States, these same hardware and software products are distributed by Minimitter Co. Inc. (Mini Mitter Co., Inc., Bend, Ore). Validity data are available on the following websites www.camntech.com and www.minimitter.com.

Several other clinical outcomes were assessed. These comprised global PSQI score, nighttime use of hypnotic medications, generic quality of life assessed using the Short Form-36,[32] and appraisal of clinical endpoints[33] (SOL and WASO ≤ 30 minutes; SE ≥ 85%) at posttreatment and follow-up.

Interventions

Cognitive Behavior Therapy

Participants assigned to CBT attended 5, weekly, 1-hour treatment sessions. These were conducted in groups of 4 to 6 participants in local general practice premises during the early afternoon or early evening. The content, aims, and objectives of each CBT session are summarized in Table 2 (further descriptions in Morin & Espie, 2003[16] or available from the first author of this paper). As can be seen in Table 2, the intervention included the common CBT components such as stimulus control, sleep restriction, and cognitive therapy strategies.

Therapists

To test a potentially generalizable model of insomnia care, we delivered CBT at "grass-roots" level, not in a specialized center or by a specialist psychologist or behavioral sleep medicine expert. Accordingly, 7 health visitors were trained to deliver CBT. In the UK, health visitors are community nurses with postqualification training and certification, who are generally based in primary care teams. They have a specific health education role and commonly encounter sleep disorders in their practice. We followed a model of "training-to-criterion" standards. That is, the health visitors had to demonstrate competence in the

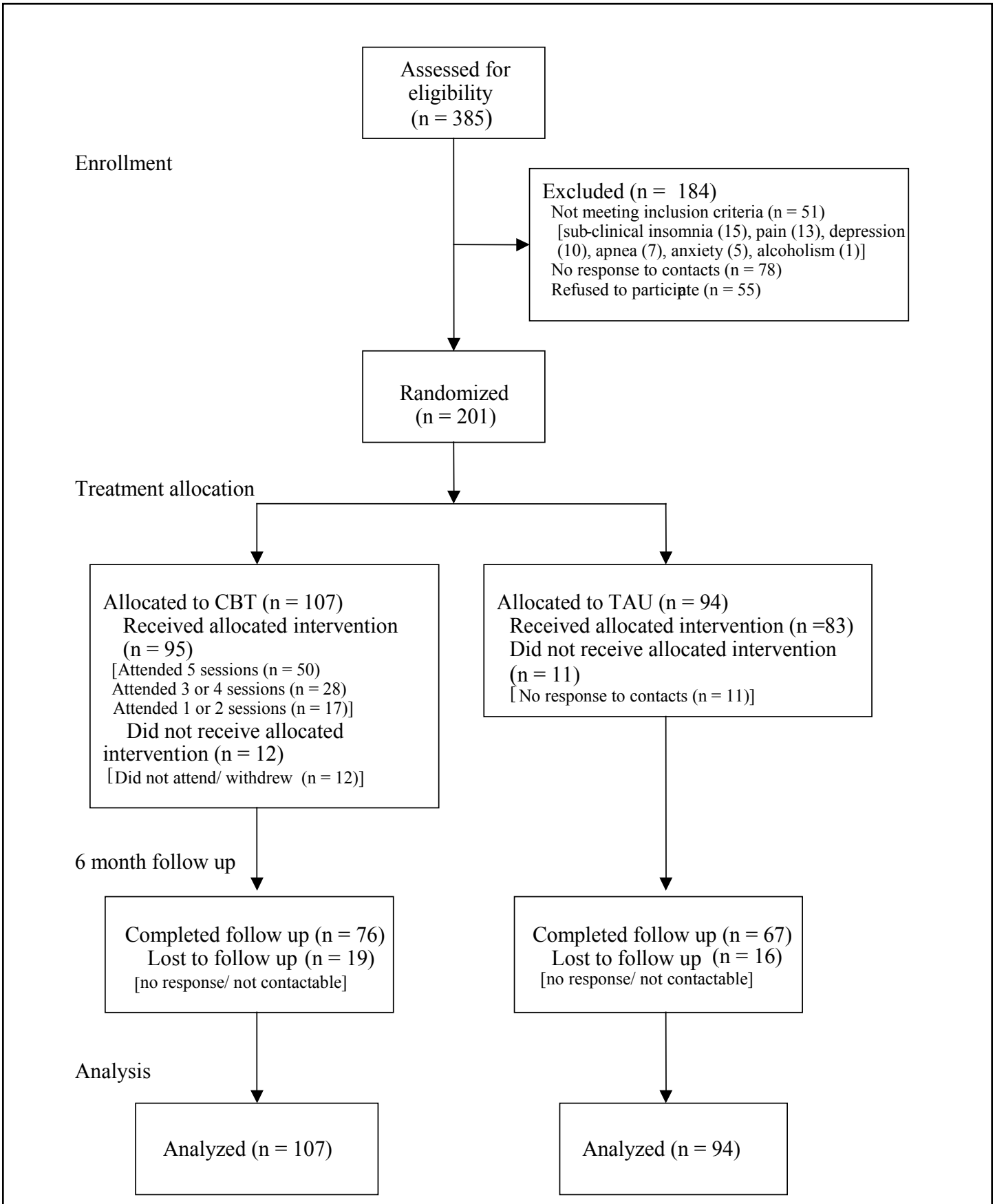


Figure 1—Participant Flowchart. CBT refers to cognitive behavior therapy; TAU treatment as usual.

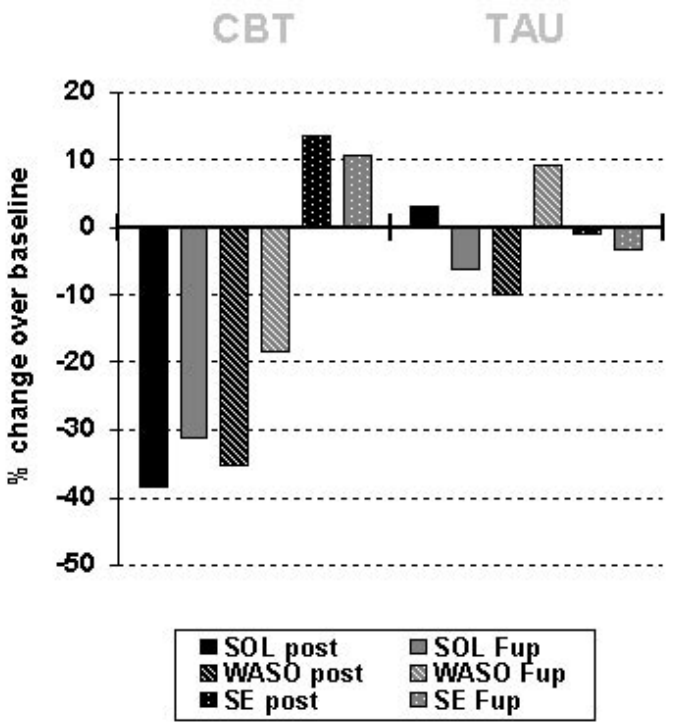


Figure 2—Sleep-diary changes at posttreatment and 6-month follow-up, expressed as percentage change from baseline values for the cognitive behavior therapy (CBT) and treatment as usual (TAU) groups. SOL refers to sleep-onset latency; WASO, wake after sleep onset; SE, sleep efficiency; post, posttreatment; Fup, follow-up

delivery of the CBT program. This was ensured by using a manualized therapy approach, participation in a short CBT course, apprenticeship learning opportunities, ongoing mentoring by an experienced clinical psychologist, and evaluation of audiotapes from randomly selected therapy sessions. Specific aspects of the training in CBT are summarized in the section on integrity/fidelity of treatment allocation.

Treatment as Usual

Effectiveness studies should replicate real clinical practices and reflect validity and generalizability.[34] Because we intended to recruit primary care patients with chronic insomnia, we expected concurrent physical and psychological symptoms, as well as concurrent treatments. The TAU comparison group thus represented normal clinical practice, in which GPs were free to offer appointments, to prescribe, and to maintain or discontinue prescriptions. What this meant in effect was that participants allocated to TAU received no additional help with their insomnia therapy, resulting from their participation in the study, but that their GPs were free to do whatever they would normally do. Indeed, in this respect, CBT was, in reality, a CBT plus TAU condition because the trial protocol explicitly permitted GPs (and other physicians and health professionals) to continue their health care provision uninterrupted with all the participants. TAU participants completed assessments as for the CBT condition but received no insomnia advice from the trial team or from our therapists. At the end of the protocol, the TAU group was provided with a booklet "The Good Sleep Guide," prepared by the first author for the National Medical Advisory Committee.[35]

Table 3—Demographic and Clinical Information on the Sample

Characteristic	CBT (n = 107)	TAU (n = 94)
Age, y[a]	54.4 ± 15.4	54.1 ± 14.4
Sex		
Women	72	65
Men	35	29
Civil status		
Partner	54	46
No partner	53	48
Working		
Yes	54	47
No	53	47
Location		
Glasgow	71	66
Edinburgh	36	28
Carstairs Deprivation Category		
1-2	37	18
3-4	23	24
5-7	47	52
Insomnia duration, y[a]	11.6 ± 9.79	10.6 ± 12.2
Insomnia presentation		
Constant	80	69
Episodic	27	23[b]
Sleep medication		
Yes	54	41
No	53	53
PSQI score[a]	12.7 ± 3.75	12.3 ± 3.55
ESS score[a]	6.05 ± 4.69	5.00 ± 4.26
Comorbid problems		
None	34	28
Physical health	11	12
Mental health	30	36
Physical and mental health	32	18
HADS-Anxiety score[a]	9.99 ± 4.10	9.63 ± 4.60
HADS-Depression score[a]	6.73 ± 3.66	7.07 ± 4.58

Data are presented as number, unless otherwise noted. CBT refers to cognitive behavior therapy; TAU: treatment as usual; PSQI: Pittsburgh Sleep Quality Index; ESS: Epworth Sleepiness Scale; HADS, Hospital Anxiety and Depression Scale.
[a]Data are presented as mean ± SD.
[b]2 missing cases.

Integrity-Fidelity of Treatment Allocation

The integrity of the treatment allocations was ensured as follows: (1) Nurse therapists attended a 12-hour, 2-day course on sleep disorders, working with groups, CBT principles, and instruction on the CBT program. (2) Therapists "sat in" on existing CBT groups and maintained an informal peer support network. (3) An experienced psychologist with training in behavioral sleep medicine acted as a mentor/consultant but did not work directly with participants. (4) The CBT program was manualized, following a recent study.[21] It comprised therapist notes, PowerPoint presentations (15 slides per session), worksheets for "break-off" times, and take-home notes with implementation guidelines. (5) Therapy sessions were audio-recorded, allowing appraisal of fidelity of CBT administration. (6) TAU participants were not seen for assessment at times when or in places where CBT assessment or intervention was operating. (7) GPs were advised of TAU allocations but were not provided with copies of CBT materials. (8)

CBT participants were asked to not make copies of materials. To strengthen this instruction, all materials were prominently marked as copyrighted.

Statistical Methods

Data analyses followed a conservative intention-to-treat model, with all allocated participants who provided baseline data included in a series of 2 (group: CBT, TAU) × 3 (time: baseline, posttreatment, follow-up) repeated-measures analyses of variance (ANOVA). Missing values were replaced using last observation carried forward, consistent with the methodology applied by Jacobs et al[57] in their intention-to-treat, placebo-controlled, trial of insomnia treatments. This approach was preferred to HLM analysis, which would have been more appropriate if the participants had been randomly assigned as groups (a "cluster" randomized trial). Participants were randomly assigned individually between the therapies. Consequently, the analysis undertaken reflects the nature of the underlying randomization (and the associated permutation test). Significant Group × Time interactions were explored posthoc by within and between sample t-tests to locate the effect. Percentage change over baseline and relative effect sizes ($d = M_1 - M_2 / \sqrt{[\{\sigma_1^2 + \sigma_2^2\}/2]}$) were computed to estimate treatment impact.[36] Consideration was paid subsequently to potential predictors of outcome using linear regression methods.

Role of the Funding Sources

Neither funding source (Chief Scientist Office, Scottish Executive; Dr. Mortimer and Theresa Sackler Foundation) participated in the study design, data analysis, or writing of this report.

RESULTS

Participant Flow

Three hundred and eighty-five adults were assessed for eligibility of whom 51 (35 women and 16 men; mean ± SD age 53.1 ± 11.2 years) were excluded, largely because of unidentified or untreated problems (Figure 1). A further 133 (87 women and 46 men; mean age 51.5 ± 17.9 years) did not complete the baseline sleep diary, leaving 201 who met criteria and were randomly assigned to treatment.

Demographic and clinical characteristics of this sample are presented in Table 3. Participants were typically middle-aged and had had insomnia for more than 10 years. Two thirds were women. Half were on sleep medication, primarily using benzodiazepine hypnotics (1 in 3 of those on hypnotic medication was on a benzodiazepine receptor agonist). Only one third of the sample had no comorbid problems. Almost 60% of the participants had some degree of mental health problem (most commonly depressive symptoms and generalized anxiety). Indeed, consistent with this finding, on the HADS, 118 (59%) scored in the clinical "caseness" range (> 10) for anxiety and 86 (43%) for depression.[23] More than one third of the sample had comorbid physical disorders, either alone (11%) or concurrent with a mental health problem (25%). Disorders of the cardiovascular (eg, high blood pressure), musculoskeletal (eg, arthritis, pain) and endocrine systems (eg, diabetes) were among the most common. Concurrent pharmacologic treatments, therefore, ranged through antidepressant, anxiolytic, β-blocker, antiinflammatory, and analgesic medications, either singly or in combination.

Participants were drawn from across the socioeconomic spectrum. The only baseline difference (Table 3 data) was a modest overrepresentation of higher socioeconomic status (DepCat group 1-2) in CBT (34.5%) relative to TAU (19%) [$\chi^2 = 6.02$, df = 2, P = 0.049]. Compared with Scottish population data,[37,38] our sample had a lower representation of midband 3 to 4 (23.5% vs 45%), a higher representation of band 1 to 2 (27.5% vs 10%), and a similar representation of the lowest band 5 to 7 (49% vs 45%).

Nineteen CBT groups were run by the therapists. Three quarters of CBT participants attended 3 or more therapy sessions. For missed sessions, participants "caught up" via discussion at the end of the subsequent attended session. The 23 participants lost from CBT/TAU during the treatment phase (Figure 1) did not respond to 2 subsequent letters or phone calls and did not differ from completers on presenting characteristics. Respectively, 80% and 81% of those receiving CBT and TAU provided data at 6-month follow-up. Thus, "drop-out" rates for CBT and TAU were similar during both intervention and follow-up. Baseline variables for noncompleters did not differ significantly from those for completers. Likewise, preliminary analyses of dependent variables revealed no differences across sites. Data therefore were pooled. No adverse events were reported anecdotally with either CBT or TAU.

Self-reported Sleep

Summary data (mean ± SD) for CBT and TAU at each assessment point are provided in Table 4. Visual inspection of these data suggests that the TAU group slept somewhat better than the CBT group at baseline. However, there were no statistically significant baseline differences in either self-reported or actigraphic sleep. Difficulty initiating sleep (SOL) declined more following CBT than TAU, the significant Group × Time interaction ($F_{2,198} = 6.64$, P = 0.002) being explained by a between-group difference at posttreatment (t = 2.74, df = 199, P = 0.004). Differences between CBT and TAU failed to maintain statistical significance at follow-up (P = 0.079). Repeated-measures ANOVA on difficulty maintaining sleep (WASO) also yielded a significant interaction term ($F_{2,198} = 7.12$, P = 0.001). However, independent samples t-tests revealed no significant differences between CBT and TAU at posttreatment (P = 0.10). SE is a measure of sleep continuity across the night. ANOVA yielded a significant Group × Time interaction ($F_{2,198} = 8.07$, P < 0.001), which was accounted for by higher posttreatment SE in the CBT group relative to TAU (t = 1.70, df = 199, P = 0.045). At follow-up, this difference was not statistically significant (P = 0.06). Total sleep time increased by 12 minutes after CBT and by 21 minutes at 6 months, compared with a 5-minute reduction for TAU. These differences, however, were not statistically significant. The interaction terms for SOL, WASO, and SE remained significant after correction for multiple comparisons on sleep-diary measures (P = 0.05/4 = 0.0125). In order to be conservative, we also repeated the above analyses entering baseline values as covariates. This did not alter any of the above findings.

Figure 2 presents percentage-change data for CBT and TAU at posttreatment and follow-up. These data illustrate marked improvement following CBT with some loss of effect at 6 months. Little change was observed with TAU. In the CBT arm, posttreat-

Table 4—Sleep Data, Before and After Intervention, and at 6-Month Follow-Up for Cognitive Behavior Therapy and Treatment as Usual Groups

SLEEP OUTCOMES	CBT	TAU
Sleep Diary		
Sleep-onset latency, min		
Baseline	60.5 ± 50.5	54.0 ± 41.1
Posttreatment	37.2 ± 42.9	55.7 ± 42.2
6-month follow-up	41.7 ± 45.5	50.7 ± 33.0
Wake after sleep onset, min		
Baseline	101.9 ± 88.2	85.0 ± 71.4
Posttreatment	66.1 ± 50.3	76.6 ± 53.1
6-month follow-up	83.0 ± 76.3	92.8 ± 63.8
Sleep efficiency, %		
Baseline	68.0 ± 19.1	73.5 ± 16.7
Posttreatment	77.1 ± 15.6	72.7 ± 16.7
6-month follow-up	75.3 ± 15.7	71.1 ± 16.7
Total sleep time, h		
Baseline	5.54 ± 1.69	5.93 ± 1.46
Posttreatment	5.74 ± 1.19	5.91 ± 1.44
6-month follow-up	5.89 ± 1.27	5.85 ± 1.21
Actigraphy		
Sleep-onset latency, min		
Baseline	23.3 ± 29.7	21.4 ± 23.3
Posttreatment	22.7 ± 22.8	20.7 ± 22.2
Wake after sleep onset, min		
Baseline	73.6 ± 37.1	56.1 ± 20.3
Posttreatment	59.0 ± 25.3	53.8 ± 23.7
Sleep efficiency, %		
Baseline	80.9 ± 9.62	84.0 ± 6.03
Posttreatment	82.7 ± 5.71	84.3 ± 4.00

CBT refers to cognitive behavior therapy; TAU, treatment as usual.

ment SOL reduction was 39% (23 minutes; d = 0.58) and was 31% (19 minutes) at follow-up (d = 0.36). For WASO, these CBT changes were 35% (36 minutes) and 19% (19 minutes), respectively (both d = 0.35). This maintained effect size at posttreatment was influenced by an increase in WASO of 10% (8 minutes) with TAU. The relative SE increase over baseline was 13% at posttreatment (d = 0.68) and was 11% at follow-up (d = 0.57) for the CBT group. These changes represented absolute increases in SE of 9.1% and 7.3% respectively. TAU was associated with a slight tendency to reduced SE

Actigraphic Estimates of Sleep

Data from 126 participants were available; 69 participants were allocated to CBT (45 women and 24 men; mean age 54.7 ± 14.6 years) and 57 to TAU (38 women and 19 men; mean age 54.7 ± 13.7 years). Demographic and clinical characteristics were similar to those of the full study sample. No effect of treatment was observed on actigraphy-derived SOL or SE (Table 4). For WASO, both Group ($F_{1,124}$ = 11.84, P = 0.001) and Time ($F_{1,124}$ = 9.55, P =0.002) main effects were significant, as was the Group × Time interaction ($F_{1,124}$ = 5.01, P = 0.027). Accordingly, a pretreatment-posttreatment change score was calculated, and an independent samples t-test conducted. This revealed a significantly greater reduction in WASO following CBT, as compared with TAU (t = 2.28, P = 0.024).

Actigraphic scores for SOL and WASO were lower and, for SE, were higher than sleep-diary estimates. Intercorrelations of weekly mean data for SOL (r = 0.340, P < 0.001), WASO (r = 0.182, P = 0.041), and SE (r = 0.275, P =0.001) were modest.

Table 5—Other Clinical Outcomes for Cognitive Behavior Therapy and Treatment as Usual Groups

OTHER CLINICAL OUTCOMES	CBT	TAU
Pittsburgh Sleep Quality Index		
Baseline	12.7 ± 3.75	12.3 ± 3.55
Posttreatment	9.84 ± 4.17	11.3 ± 3.68
6-month follow-up	8.40 ± 4.14	11.2 ± 3.24
Medication use per night		
Baseline	0.48 ± 0.92	0.61 ± 0.85
Posttreatment	0.30 ± 0.60	0.53 ± 0.68
6-month follow-up	0.26 ± 0.49	0.47 ± 0.70
SF-36		
Physical functioning		
Baseline	67.1 ± 26.5	68.5 ± 24.7
Posttreatment	71.8 ± 18.7	71.4 ± 17.9
Social functioning		
Baseline	61.9 ± 26.4	60.3 ± 28.2
Posttreatment	65.0 ± 20.0	62.4 ± 25.4
Physical role limitation		
Baseline	59.8 ± 28.9	61.7 ± 27.4
Posttreatment	62.5 ± 19.7	60.2 ± 21.2
Emotional role limitation		
Baseline	60.6 ± 23.7	62.8 ± 24.2
Posttreatment	67.0 ± 17.4	62.8 ± 21.9
Mental health		
Baseline	45.0 ± 12.9	46.4 ± 14.7
Posttreatment	50.2 ± 8.20	47.8 ± 14.2
Energy/vitality		
Baseline	38.4 ± 16.0	42.3 ± 15.1
Posttreatment	45.8 ± 12.0	43.9 ± 14.1
Pain		
Baseline	57.5 ± 22.9	59.8 ± 22.8
Posttreatment	59.1 ± 21.5	60.8 ± 19.8
General health perceptions		
Baseline	55.0 ± 22.0	56.2 ± 20.1
Posttreatment	60.6 ± 16.8	58.1 ± 18.1

Data are presented as mean ± SD; for medication use, this is mean ± SD number of tablets taken per night. CBT refers to the cognitive behavior therapy; TAU, treatment as usual; SF-36: Short Form-36.

Other Clinical Outcomes

Global sleep disturbance reduced by at least 4 PSQI points (more than 1 SD) at 6-month follow-up under CBT, compared with a 1-point change under TAU (Table 5). The Group × Time interaction effect was significant ($F_{2,198}$ = 3.83, P = 0.023), accounted for by PSQI reductions both at posttreatment (t = 1.68, df = 199, P = 0.048) and follow-up (t = 2.97, df = 199, P = 0.002) in the CBT group, compared with TAU. There was a nonsignificant reduction of hypnotic consumption in both CBT and TAU (Time main effect $F_{2,198}$ = 2.68, P = 0.074) but no significant interaction.

Following CBT, 32 participants (30%) achieved a SOL of 30 minutes or less, with 35 (33%) achieving this endpoint at 6 months. In TAU, the comparable figures were 17 (18%) and 21 (22%), respectively. These effects represent a significant improvement following CBT relative to TAU at posttreatment (χ^2 = 4.67, df =

1, P = 0.022; Fisher exact Test) but not at follow-up (χ^2 = 2.17, df = 1, P = 0.094). Fourteen participants (13%) had WASO of 30 minutes or less after CBT, with 20 (19%) obtaining this cut-off at follow-up, compared with 10 (11%) and 11 (12%), respectively, following TAU. The achievement of SE of at least 85% was also included as a conventional threshold value for normal sleep.[55,56] This criterion was achieved by 28 (26%) of CBT participants after therapy and by 21 (20%) at follow-up. For TAU, these outcomes were obtained by 16 (17%) and 13 (14%), respectively. These WASO and SE indexes of change were not significantly different upon statistical analysis.

The SF-36 was completed before and after treatment (see Table 5). Higher values indicate better perceived health. Time main effects were observed in 4 domains: physical functioning (F = 5.82, P = 0.017), mental health (F = 12.9, P = 0.001), energy/vitality (F = 15.7, P < 0.001) and general health (F = 8.89, P = 0.003) (all df 1,199). Significant Group × Time interactions, suggesting better treatment response after CBT, were obtained for 2 domains: mental health (F = 4.29, P = 0.040) and energy/vitality (F = 7.92, P < 0.005). A nonsignificant effect was observed for emotional role limitation to respond better to CBT (F = 3.43, P = 0.066).

Predictors of Outcome

It is important also to investigate if there were any factors specifically associated with better or poorer treatment response. Demographic (eg, sex, age, socioeconomic status, location of group), clinical (eg, comorbidities, psychopathology), sleep (eg, duration of insomnia, medication) and treatment-related data (eg, attendance rate, therapist, group attended) were available to inform such analyses. At the conceptual level, such independent variables are divisible into "moderator (present at baseline) and "mediator" (treatment-related) influences upon outcome. SE change from before to after treatment was selected as the dependent variable because SE is a recognized summary index of sleep disturbance and because SE demonstrated the largest posttreatment effect size. Stepwise linear regression revealed that only 2 variables contributed significantly to the prediction of SE change for the CBT group (F = 26.12, P < 0.001). Baseline SE (a moderator variable) entered on the first step (Adj. R^2 = 0.433; β = 0.611; P < 0.001) and frequency of attendance at CBT sessions (a mediating variable) contributed a small amount of additional explanatory variance (Adj. R^2 = 0.469; β = -.218; P = 0.033).

DISCUSSION

Insomnia is a problem with population prevalence and comorbidity so high that a clinical effectiveness study is required to establish if a promising intervention like CBT can be translated into a community-based treatment. CBT is normally regarded as a complex and specialized intervention; therefore, as well as effectiveness, there is the issue of feasibility. This study, therefore, investigated the impact of manualized, nurse-administered, small-group CBT on relatively unselected "real-world" participants with severe and persistent insomnia.

Our intention-to-treat data offer some support for the clinical effectiveness of CBT for insomnia. Significant reductions, totalling around 60 minutes per night, in symptom measures of SOL and WASO were observed with CBT, and SE increased by 9%. TAU did not yield comparable benefits. ANOVA models suggest that these posttreatment improvements were more convincing for sleep latency than for wakefulness during the night, and this is confirmed by effect-size data (d = 0.58 and d = 0.35, respectively). Effect size was greatest for SE (d = 0.68) indicating that, under CBT, participants were reliably sleeping through a greater proportion of their time in bed. Global sleep quality, as measured on the PSQI, also improved following CBT. Follow-up data, however, suggest some loss of therapeutic effect, particularly in WASO, although the WASO effect size was sustained, perhaps because TAU participants were somewhat more wakeful at 6 months. Effect sizes for SOL and WASO at follow-up were small to medium but remained relatively robust for SE (d = 0.57). Mean CBT reductions for SOL and WASO, in previous efficacy studies, have been about 30 minutes each,[33] similar to our findings. However, average effect sizes have been 0.88 and 0.65, respectively, considerably larger than our results. This may reflect the more severe and complex presentation of our patient group. There is also some suggestion that our study may have been underpowered to detect between-group differences at 6-month follow up, when nonsignificant probabilities for SE (P = 0.06) and SOL (P = 0.079) were obtained. Posthoc power calculations indicate that sample sizes of 235 (for SE) and 300 (for SOL) would have been required to achieve an α value of 0.05 at 80% power. We had originally planned to enrol 240 participants but achieved only 201. These data may be indicative of the considerably larger sample sizes required for effectiveness research, relative to efficacy research, because of the greater within and between-subject variability in clinical samples.

Nevertheless, and consistent with other recent data,[39] it is encouraging that factors such as the chronicity of the insomnia disorder, the absence or presence of physical and mental health comorbidities, and participant age and sex did not emerge from regression analysis as explanatory factors associated with therapeutic response to CBT. As might be expected from the law of initial values, baseline SE was the main predictor of SE improvement, explaining 43% of variance in the treated group. Thus, baseline data may moderate treatment response but, from our data, primarily in the sense that high baseline values provide greater room for improvement. Attendance rate at CBT sessions added a further 3.6% of explanatory variance and was the only treatment-related mediator of outcome. People who attended more often were likely to do better. This too is an important finding, reinforcing the importance of motivational aspects of CBT and of helping patients to conceptualize the program as a course of treatment. The interaction of insomnia severity with the likelihood of committing to achieving and sustaining change in sleep-related behavior and cognition appears worthy of further dedicated research effort.

Sleep self-report data were not mirrored by actigraphically estimated sleep, either in terms of capturing the baseline complaint of insomnia or in terms of outcome. Only WASO data demonstrated treatment-related impact, whereas, on sleep diary reports, CBT was associated with greater reduction in SOL and increase in SE. These findings parallel other recent work showing limited impact of CBT on actigraphically determined sleep.[40-42] Only 1 study, on insomnia patients with chronic pain, has demonstrated both subjective and actigraphic sleep improvement.[43] Indeed, a systematic review and practice parameters statement, published since we began this research, suggests a limited role for actigraphy applied to insomnia intervention research.[44,45] Studies have found that actigraphy consistently produces different estimates

of sleep time and number of awakenings and lower estimates of sleep latency than do sleep diaries,[46,47] and correlation between self-report and actigraphy has been generally poor.[48-50] However, such limited correlation between entirely different modes of assessment should not be unexpected. In considering the role of actigraphy in sleep assessment, Tryon[51] makes the point that the observed modest coefficients of validity of actigraphy (in relation to polysomnography) actually exceed those associated with many medical and psychological tests. Consequently, the significant impact of CBT upon actigraphically estimated WASO in the present study is interesting in part because it contrasts with the sleep-diary improvements that were observed primarily in the SOL and SE domains.

We used conservative criteria to investigate clinical endpoints. Of CBT treated. 20% to 30% achieved these endpoints (below threshold for insomnia disorder), compared with 10% to 20% of TAU participants. Compared with absolute reductions in SOL, WASO, and SE complaints, these outcomes are relatively disappointing for CBT and may reflect the initial severity of the sleep disorders in this study. That is, although CBT was associated with greater symptomatic improvement, many participants remained in the clinical range at follow-up. Certainly, if even more stringent criteria were applied (eg, reduction in symptom score of at least 0.5 SD plus 30 minutes or less of SOL or WASO), we would have obtained very few responders to CBT. Likewise, although significant PSQI-based sleep-quality improvement was achieved and sustained in the long term following CBT, the 6-month follow-up mean value of 8.4 remained considerably higher than the cut-off of 5 used for normal sleep.

Such results raise the long-standing issue in insomnia outcome research concerning the relative paucity of treated participants who endorse becoming normal sleepers after treatment. In the context of this particular study, one possible explanation for the disappointing clinical endpoints is that there may have been insufficient treatment offered through the CBT-group program, at least for a proportion of the patients. Whereas this model of care may be sufficient for some, it may not be sufficient for the majority of clinical insomnia cases. The use of more sessions, more highly skilled therapists, tailored CBT interventions, or a combination of CBT and pharmacotherapy treatments, along with proven methods to achieve and to sustain high levels of patient assimilation of treatment information and adherence to treatment protocol may all make a difference to outcomes in insomnia clinics. Although work is steadily advancing in these areas, we do not yet have the algorithms to enable us to make these judgements in an informed manner.

Nevertheless, our CBT participants demonstrated more than mere sleep-symptom change. On the SF-36, health-related quality-of-life improvement was found in domains reflecting mental health and vitality. These results suggest that CBT for insomnia may be associated with generalized benefits to everyday functioning. This is consistent with contemporary understanding of insomnia not only as a disorder that impairs the sleep experience, but also one that negatively impacts the day.[5,6] Our findings of quality-of-life change following CBT for insomnia parallel other recent data using the SF-36.[52,53]

Notwithstanding our earlier comments about sampling adequacy, our sample size (n = 201) was considerably larger than that of any previous report of insomnia treatment. Efficacy studies typically have comprised 40 to 100 participants.[33] More importantly, our participants were clinically identified, and 70% had comorbid mental or physical health problems. Only 51 of 385 potential participants were excluded, largely because of suspected untreated disorders. However, a further 133 who met criteria and consented to participate in the study, withdrew prior to random allocation to treatment. Unfortunately, we have limited data on the reasons for these withdrawals. Delays in processing individuals may have contributed to some nonresponses. Also, we suspect that many individuals who were routine users of hypnotics on prescription, who initially expressed interest when contacted by their GPs, thereafter withdrew. This may reflect the importance of patient "readiness" to adopt a CBT approach in the real-world clinical setting. Nevertheless, 50% of randomly assigned participants were on hypnotics. The personal, socioeconomic, and clinical profiles of our participants suggest that we did identify our target community population and that they had severe and chronic insomnia. Total wakefulness per night was around 2.5 hours, and baseline SE was around 70%.

We wanted to test a potentially generalizable model of care that had proven to be beneficial in a more limited previous study.[21] From our experience, it is feasible for nurses based in primary care to learn, and to deliver, a CBT program, and our results are promising for what might be regarded as a "first-line" insomnia intervention. By providing group treatment using a trained nurse, the "per patient" costs may be minimized. Crucially, however, the use of a manual ensured treatment integrity and fidelity, and, alongside training, supervision and case review, would seem a crucial component of any program "roll-out." We have summarized elsewhere how a skilled clinical psychologist or behavioral sleep medicine specialist could operate an insomnia "triage" system, allowing nurse-led group-based CBT to complement individual therapies.[54] This model, of course, requires further evaluation. Moreover, our approach would need to be tailored to the operational characteristics of other healthcare systems. Perhaps the National Health Service in Scotland is more amenable to this type of intervention in primary care because of the established health-provision role of primary care nurses (health visitors) and the day-to-day interactions between clinical psychologists and GP services. In the UK, generally, the services provided by the National Health Service are all government funded, using revenue from the taxation system. The common co-occurrence of insomnia with depressive and/ or anxiety symptoms in primary mental health care also highlights the need for investigation of CBT for insomnia as an adjunct to existing community treatment for these disorders. The intervention described here might be readily adapted for that purpose.

In interpreting our findings, several factors merit consideration. First, the dataset are limited to self-report and actigraphic estimation of sleep. Although the former in particular is appropriate to the clinical-effectiveness question, appraisal of effects upon polysomnographically defined sleep at home would be informative. Second, CBT did not specifically target hypnotic drug use. Reductions in use were observed with both CBT and TAU. This may reflect an implicit focus upon non-pharmacologic management. Nevertheless, other recent work has demonstrated that CBT can be effectively applied to hypnotic reduction as a primary outcome.[52] Third, the use of TAU as the control condition of choice for clinical effectiveness study also imposes limitations. Important among these is the fact that CBT does not control for the additional time, attention, and demand characteristics associated with provision of a therapy. Although such factors have been

controlled for in previous CBT trials, there remains the possibility that such nonspecific treatment factors played a part in the response to the CBT arm of the present study. Finally, both groups in effect had "treatment as usual," and, so, other factors may have influenced our results, even though participants were appropriately randomly assigned to a treatment group. Efficacy trials ensure better control over variation, whereas effectiveness studies give indications about potential for service implementation. Clearly, both methodologies are required to test the important possibility that CBT could become the treatment of first choice for persistent insomnia in primary care.

ACKNOWLEDGMENTS

This study was supported by the Chief Scientist Office, Scottish Executive Health Department (CZH/4/Z) and the by the Dr. Mortimer and Theresa Sackler Foundation

The authors gratefully acknowledge the administrative and practical support of Colette Fulton and Lucy McCloughan (Lothian Primary Care Network), Bridie Fitzpatrick (WestNet, Glasgow) and Brian Rae (Primary Care Division R & D, NHS Greater Glasgow). We are also grateful to Simon Dixon (University of Sheffield) for his assistance with analysis of the SF-36 data and to Lauren M. Marchetti and Carolyn J. Espie for their work on data entry. Most of all we thank the seven Health Visitors who acted as CBT therapists for the study, the General Practitioners who identified participants and, of course, the patients who participated in the trial.

TRIAL REGISTRATION

NCT00170417

REFERENCES

1. Singleton N, Bumpstead R, O'Brien M, Lee A, Meltzer H. Psychiatric morbidity among adults living in private households, 2000. London: The Office for National Statistics, HMSO; 2001.
2. Ford DE, Kamerow DB. Epidemiologic study of sleep disturbances and psychiatric disorders. JAMA 1989:262:1479-84.
3. Lichstein KL, Durrence HH, Reidel BW, Taylor DJ, Bush AJ. The Epidemiology of Sleep: Age, Gender and Ethnicity. Mahwah, NJ: Lawrence Erlbaum Associates; 2004.
4. Shocat T, Umphress J, Israel AG, Ancoli-Israel S. Insomnia in primary care patients. Sleep 1999;22: Suppl 2:S359-65.
5. International Classification of Sleep Disorders: Diagnostic and Coding Manual, revised. Rochester: American Sleep Disorders Association. 1997.
6. Diagnostic and Statistical Manual of Mental Disorders, 4th ed (DSM-IV). Washington: American Psychiatric Associatio; 1994.
7. Morgan K. Factors influencing persistent subjective insomnia in old age: a follow study of good and poor sleepers age 65-74. Age Ageing 1989;18:17-125.
8. Dew MA, Hoch CC, Buysse DJ, et al. Healthy older adults' sleep predicts all cause mortality at 4 to 19 years of follow-up. Psychosom Med 2003;65:63-73.
9. Roth T, Ancoli-Israel S. Daytime consequences of insomnia in the United States: Results of the 1991 National Sleep Foundation Survey II. Sleep 1999;22:Suppl 2, S354-63.
10. Riemann D, Voderholzer U. Primary insomnia: A risk factor to develop depression? J Affect Disord 2003;76:255-9.
11. Cole MG, Dendukuri N. Risk factors for depression among elderly community subjects: a systematic review and meta-analysis. Am J Psychiatry 2003;160:1147-56.
12. Sateia MJ, Nowell PD. Insomnia. Lancet 2004;364:1959-73.
13. Kripke D. Hypnotic drugs: deadly risks, doubtful benefits. Sleep Med Rev 2000;4:5-20.
14. Mendelson WB. A review of the evidence for the efficacy and safety of Trazodone. J Clin Psychiatry 2005;66:469-76.
15. Guidance on the use of zaleplon, zolpidem and zopiclone for the short-term management of insomnia. London: National Institute for Clinical Excellence. Technology Appraisal Guidance No.77; 2004.
16. Morin C.M, Espie CA. Insomnia: A Clinical Guide to Assessment and Treatment. New York: Kluwer Academic/ Plenum Publishers; 2003.
17. Kohn L, Espie CA. Sensitivity and specificity of measures of the insomnia experience: a comparative study of psychophysiologic insomnia, insomnia associated with mental disorder and good sleep. Sleep 2005;28:104-12.
18. Morin CM, Culbert JP, Schwartz MS. Non-pharmacological interventions for insomnia: a meta-analysis of treatment efficacy. Am J Psychiatry 1994;151:1172-80.
19. Murtagh DR, Greenwood KM. Identifying effective psychological treatments for insomnia: a meta-analysis. J Consult Clin Psychol 1995;63:79-89.
20. Smith MT, Perlis, ML, Park A, Giles DE, Pennington JA, Buysse, D. Behavioral treatment vs pharmacotherapy for insomnia—a comparative meta-analysis. Am J Psychiatry 2002;159:5-11.
21. Espie CA, Inglis SJ, Tessier S, Harvey L. The clinical effectiveness of cognitive behaviour therapy for chronic insomnia: Implementation and evaluation of a sleep clinic in general medical practice. Behav Res Therapy 2001;39:45-60.
22. Buysse DJ, Reynolds CF, Monk TH, Berman SR, Kupfer, DJ. The Pittsburgh Sleep Quality Index: a new instrument for psychiatric practice and research. Psychiatry Res 1989;28:193-213.
23. Zigmond AS, Snaith RP. The Hospital Anxiety and Depression Scale. Acta Psychiatr Scand 1983;67:361-70.
24. Johns MW. A new method for measuring daytime sleepiness: the Epworth Sleepiness Scale. Sleep 1991;14:450-545.
25. Espie CA. Assessment and differential diagnosis of insomnia in later life. In: Lichstein KL, Morin CM, eds. Treatment of Late-Life Insomnia. New York: Sage Publications; 2000:81-108.
26. Edinger JD, Fins AI, Sullivan RJ, et al. Sleep in the laboratory and sleep at home: comparisons of older insomniacs and normal sleepers Sleep 1997;20:1119-26.
27. Coates TJ, Killen KD, George J, Marchini E, Silverman S, Thoresen C. Estimating sleep parameters: a multi-trait multi-method analysis. J Consult Clin Psychol 1982;50:345-52.
28. Wohlgemuth WK, Edinger JD, Fins AI, Sullivan RJ. How many nights are enough? The short-term stability of sleep parameters in elderly insomniacs and normal sleepers. Psychophysiology 1999;36:233-44.
29. Espie CA. The Psychological Treatment of Insomnia. Chichester, England: John Wiley and Sons Ltd.; 1991.
30. American Sleep Disorders Association. Practice parameters for the use of actigraphy in the clinical assessment of sleep disorders. Sleep 1995;18:285-7.
31. Sadeh A, Hauri PJ, Kripke DF, Lavie P. The role of actigraphy in the evaluation of sleep disorders. Sleep 1995;18:288-302.
32. Jenkinson C, Layte R, Wright L, Coulter A. The UK SF-36: an analysis and interpretation manual. Oxford: Health Services Research Unit, University of Oxford; 1996
33. Morin C.M, Hauri P, Espie CA, Spielman A, Buysse D, Bootzin RR. Nonpharmacologic treatment of chronic insomnia: an American Academy of Sleep Medicine Review. Sleep 1999;22:1134-56.
34. Clarke GN. Improving the transition from basic efficacy research to effectiveness studies: methodological issues and procedures. J Consult Clin Psychol 1995;63:718-25.
35. National Medical Advisory Committee. The Management of Anxiety and Insomnia. Edinburgh: Scottish Office Home and Health Department, HMSO; 1993.

36. Cohen J. A power primer. Psychol Bulletin 1992;112:155-9.
37. Carstairs V, Morris R. Deprivation and Health in Scotland. Aberdeen: Aberdeen University Press; 1991.
38. McLoone P. Carstairs Scores for Scottish Postcode Sectors from the 1991 Census. Glasgow: Public Health Research Unit, University of Glasgow; 1994.
39. Espie CA, Inglis SJ, Harvey L. Predicting clinically significant response to cognitive behavior therapy (CBT) for chronic insomnia in general practice: analyses of outcome data at 12 months post-treatment. J Consult Clin Psychol 2001;69:58-66.
40. Rybarczyk B, Lopez M, Benson R, Alsten C, Stepanski E. Efficacy of two behavioral treatment programs for comorbid geriatric insomnia. Psychol Aging 2002;17:288-98.
41. Broomfield NM, Espie CA. Initial insomnia and paradoxical intention: an experimental investigation of putative mechanisms using subjective and actigraphic measurement of sleep. Behav Cogn Psychotherapy 2003;31:313-24.
42. Friedman L, Benson K, Noda A. An actigraphic comparison of sleep restriction and sleep hygiene treatments for insomnia in older adults. J Geriatr Psychiatry Neurol 2000:13:17-27.
43. Currie SR, Wilson KG, Pontefract AJ, deLaplante L. Cognitive-behavioral treatment of insomnia secondary to chronic pain. J Consult Clin Psychol 2000;68:407-16.
44. Ancoli-Israel S, Cole R, Alessi C, Chambers M, Moorcroft W, Pollak CP. The role of actigraphy in the study of sleep and circadian rhythms. Sleep 2003;26:342-92.
45. Littner M, Kushida CA, Anderson WM, et al. Practice parameters for the role of actigraphy in the study of sleep and circadian rhythms: an update for 2002. Sleep 2003;26:337-41.
46. Guilleminault C, Clerk A, Black J Labanowski M, Pelayo R, Claman D. Nondrug treatment trials in psychophysiologic insomnia. Ann Intern Med 1995;155:838-44.
47. Wilson KG, Watson ST, Currie SR. Daily diary and ambulatory activity monitoring of sleep in patients with insomnia associated with chronic musculoskeletal pain. Pain 1998;75:75-84.
48. Jean-Louis G, Kripke D, Ancoli-Israel S. Sleep and quality of well-being. Sleep 2000;23:1115-21.
49. Dagan Y, Zinger Y, Lavie P. Actigraphic sleep monitoring in post-traumatic stress disorder (PTSD) patients. J Psychosom Res 1997;42:577-81.
50. Wicklow A, Espie C.A. Intrusive thoughts and their relationship to actigraphic measurement of sleep: towards a cognitive model of insomnia. Behav Res Therapy 2000;38:679-93.
51. Tryon WW. Issues of validity in actigraphic sleep assessment. Sleep 2004;27:158-65.
52. Morgan K, Dixon S, Mathers M, Thompson J, Tomeny M. Psychological treatment for insomnia in the management of long-term hypnotic drug use: a pragmatic randomised controlled trial. Br J Gen Pract 2003:53:923-8.
53. Dixon S, Morgan K, Mathers M, Thompson J, Tomeny M. Impact of cognitive behaviour therapy on health related quality of life among hypnotic users with chronic insomnia. Behav Sleep Med 2007:In Press.
54. Espie CA, MacMahon KMA, Kelly H-L, et al. Manualised CBT for insomnia delivered by nurse practitioners: results from studies conducted in primary care and in oncology. Sleep Med 2005;6: Suppl 2:S29.
55. Frankel BL, Coursey RD, Buchbinder R, Snyder F. Recorded and reported sleep in primary chronic insomnia. Arch Gen Psychiatry 1976;33:615-23.
56. Coates TJ, Killen JD, George J, Marchini E, Silverman S, Thoresen CE. Discriminating good sleepers from insomniacs using all-night polysomnograms conducted at home. J Nerv Mental Dis 1982;170:224-30.
57. Jacobs GD, Pace-Schott EF, Stickgold R, Otto MW. Cognitive behavior therapy and pharmacotherapy for insomnia. Arch Intern Med 2004;164:1888-96.

VOLUME 26 · NUMBER 28 · OCTOBER 1 2008

JOURNAL OF CLINICAL ONCOLOGY

ORIGINAL REPORT

Randomized Controlled Clinical Effectiveness Trial of Cognitive Behavior Therapy Compared With Treatment As Usual for Persistent Insomnia in Patients With Cancer

Colin A. Espie, Leanne Fleming, James Cassidy, Leslie Samuel, Lynne M. Taylor, Craig A. White, Neil J. Douglas, Heather M. Engleman, Heidi-Louise Kelly, and James Paul

From the University of Glasgow Sleep Centre, Southern General Hospital; Beatson Oncology Centre, Western Infirmary, Glasgow; Anchor Unit, Aberdeen Royal Infirmary, Aberdeen; School of Health, University Campus Ayr, Beech Grove, Ayr; Edinburgh Sleep Centre, Edinburgh Royal Infirmary, Edinburgh, United Kingdom.

Submitted November 13, 2007; accepted February 11, 2008; published online ahead of print at www.jco.org on June 30, 2008.

Supported by Cancer Research UK (Grant No. C8265/A3036) and from the Dr Mortimer and Theresa Sackler Foundation.

Authors' disclosures of potential conflicts of interest and author contributions are found at the end of this article.

Corresponding author: Colin A. Espie, PhD, FBPsS, University of Glasgow Sleep Centre, Southern General Hospital, Glasgow, G51 4TF United Kingdom; e-mail: C.Espie@clinmed.gla.ac.uk.

0732-183X/08/2628-4651/$20.00

DOI: 10.1200/JCO.2007.13.9006

ABSTRACT

Purpose
Persistent insomnia is a common complaint in cancer survivors, but is seldom satisfactorily addressed. The adaptation to cancer care of a validated, cost-effective intervention may offer a practicable solution. The aim of this study was to investigate the clinical effectiveness of protocol-driven cognitive behavior therapy (CBT) for insomnia, delivered by oncology nurses.

Patients and Methods
Randomized, controlled, pragmatic, two-center trial of CBT versus treatment as usual (TAU) in 150 patients (103 females; mean age, 61 years.) who had completed active therapy for breast, prostate, colorectal, or gynecological cancer. The study conformed to CONSORT guidelines. Primary outcomes were sleep diary measures at baseline, post-treatment, and 6-month follow-up. Actigraphic sleep, health-related quality of life (QOL), psychopathology, and fatigue were secondary measures. CBT comprised five, small group sessions across consecutive weeks, after a manualized protocol. TAU represented normal clinical practice; the appropriate control for a clinical effectiveness study.

Results
CBT was associated with mean reductions in wakefulness of 55 minutes per night compared with no change in TAU. These outcomes were sustained 6 months after treatment. Standardized relative effect sizes were large for complaints of difficulty initiating sleep, waking from sleep during the night, and for sleep efficiency (percentage of time in bed spent asleep). CBT was associated with moderate to large effect sizes for five of seven QOL outcomes, including significant reduction in daytime fatigue. There was no significant interaction effect between any of these outcomes and baseline demographic, clinical, or sleep characteristics.

Conclusion
CBT for insomnia may be both clinically effective and feasible to deliver in real world practice.

J Clin Oncol 26:4651-4658. © 2008 by American Society of Clinical Oncology

INTRODUCTION

Sleep disturbance is an important, common, and distressing problem for cancer patients,[1-3] 19% to 30% of whom meet insomnia diagnostic criteria,[4,5] including negative daytime consequences.[6,7] However, insomnia is often unrecognized or poorly managed[8,21] and long-term pharmacotherapy is not desirable,[9-12] especially when fatigue is problematic,[13] yet 25% of cancer patients regularly take sleeping pills.[5]

Cognitive behavior therapy (CBT), effective for primary insomnia,[14-19] is promising because insomnia often arises during stress[5,6,20] and is perpetuated by behavioral and mental factors.[6,15,20,21]

Two randomized trials of CBT for insomnia in patients with cancer have been reported. Cannici et al[22] allocated 30 participants with a range of cancers to a three-session relaxation program or no treatment. Self-reported sleep latency reduced in the relaxation group. Recently, Savard et al[23] studied 57 women (mean age, 54 years) with insomnia caused/aggravated by breast cancer. CBT comprised 8 weekly group sessions (each 90 minutes) led by a psychologist. Sustained reductions in sleep latency and wakefulness were observed after CBT relative to control. There was no increase in total sleep but increases in sleep efficiency (proportion of time in bed spent asleep) averaged 15%.

This article reports a pragmatic, intention-to-treat evaluation of nurse-administered CBT, to evaluate potential for real world implementation of CBT across a range of cancer subtypes, and within

the clinical effectiveness tradition. Treatment as usual (TAU) was employed as the control arm.

PATIENTS AND METHODS

Aims and Objectives

Our overall aim was to test the clinical effectiveness of CBT for persistent insomnia associated with cancer in the real world. Major research questions were: "Is CBT superior to TAU in reducing chronic sleep disturbance and improving quality of life functioning?" "Are observed improvements durable?" and "Are there predictors of good outcome or contraindications to CBT as a first line treatment for insomnia in routine care?"

Participants

Potential participants (+18 years) were attending follow-up clinics at the Beatson Oncology Centre, Glasgow or Anchor Unit, Aberdeen Royal Infirmary from December 2003 to June 2005. They had to have a diagnosis of breast, prostate, bowel, or gynecological cancer, and to satisfy diagnostic criteria for chronic insomnia; mean value longer than 30 minutes for complaint of delayed sleep-onset latency (SOL) and/or wake time after sleep onset (WASO), occurring ≥ 3 nights per week for ≥ 3 months and affecting daytime function.[5,24] Participants also had to screen more than 5 on the Pittsburgh Sleep Quality Index (PSQI),[25,26] a psychometrically robust instrument that identifies clinically significant sleep disturbance. Thus acute insomnia and transient effects associated with cancer treatment/adverse effects were excluded. Treatment (radiation therapy or chemotherapy) had to be completed by ≥ 1 month with no further anticancer therapy planned (excepting adjuvant hormone therapy). Participants with acute illness, estimated prognosis fewer than 6 months, confusional problems or drug misuse, or with evidence of other sleep

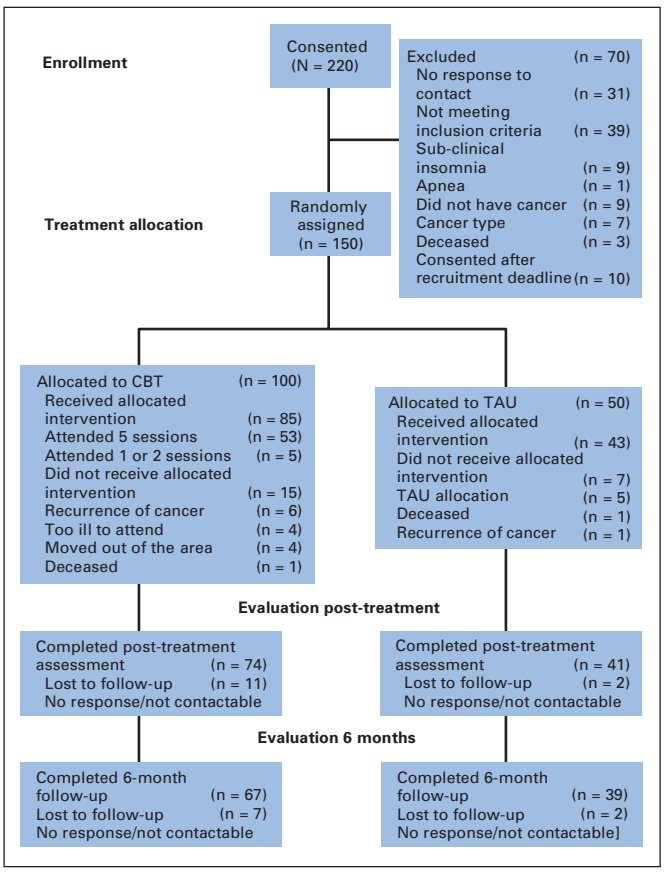


Fig 1. Participant flow chart. CBT, cognitive behavior therapy; TAU, treatment as usual.

Table 1. Demographic, Clinical, and Baseline Sleep Information on the Sample

Characteristic	CBT (n = 100)		TAU (n = 50)	
	Median	IR	Median	IR
Demographic				
Age, years	60.5	53.3-70	58	52-68
Sex				
Female	69		34	
Male	31		16	
Civil status				
Partner	72		35	
None	28		15	
Occupation				
Working	35		22	
Not	65		28	
Location				
Glasgow	54		27	
Aberdeen	46		23	
Clinical				
Cancer diagnosis				
Breast	60		27	
Prostate	22		12	
Bowel	15		9	
Gynecologic	3		2	
Time between cancer diagnosis and screening, months	23.5	10-59.5	33.5	10-83.3
No. of patients with insomnia prior to cancer diagnosis	48		17	
HADS-A	8	4.5-10	8	5-10
HADS-D	5	2-8	5	2-7
Current treatment for depression				
Yes	13		7	
No	87		43	
FSI interference subscale	3.57	2-4.96	3.43	1.87-5.43
FACT subscale				
Physical	22.6	18-25	23.0	17-25
Social	22.0	16.1-25.5	20.0	15-24.5
Emotional	19.0	16-22	19.0	15-21
Functional	18.0	13-22	18.0	15-22
Total score	80.2	68-89	80.0	66-90
Sleep				
Mean insomnia duration in months	30		27	
Standard deviation		12-60		10.8-60
Insomnia				
Constant	68		32	
Episodic	32		18	
Sleep medication				
Yes	22		7	
No*	62		36	
PSQI	13	11-16	13.5	11-15
ESS	6.5	3-9.8	8	3-10.3
Sleep onset latency*	41.0	20.3-64.8	27.4	22.4-50.0
Wake time after sleep onset*	62.0	40.7-107.5	51.0	30.5-82.0
Total sleep time*	399.0	343.3-455.9	392.0	348.0-457.9
Sleep efficiency*	80.4	69.5-85.8	82.4	74.5-88.5

NOTE. Data are categorical or median IR.
Abbreviations: IR, interquartile range; CBT, cognitive behavior therapy; TAU, treatment as usual; PSQI, Pittsburgh Sleep Quality Index; ESS, Epworth sleepiness scale; FSI, Fatigue Symptom Inventory; FACT, Functional Assessment of Cancer Therapy; HADS-A, Hospital Anxiety and Depression Scale-Anxiety; HADS-D, Hospital Anxiety and Depression Scale-Depression.
*Baseline sleep diary data: n = 84 (CBT) and n = 43 (TAU).

disorders (eg, sleep apnea; screened by reports of nightly snoring and nocturnal breathing pauses, plus Epworth sleepiness scale[27] more than 10) or of untreated psychiatric disorder, were excluded. Potential participants were notified of the study by posters/leaflets in clinic waiting areas, by mailing information to those attending upcoming clinics, or directly by staff on attendance. All participants gave written informed consent and their medical consultant agreed to their participation. The protocol was approved by local NHS research ethics committees.

Sample Characteristics

Of 220 patients who consented, 31 failed to respond to further contact (25 females; mean age, 56 years [±10.2 years]) and 39 were excluded (23 females; 61 years [±13.4 years]; Fig 1). The remaining 150 participants were randomly assigned (103 females; 61 years [±10.5 years]; age range, 38 to 86 years; Table 1).

Approaching three fourths had a partner, but less than 40% were employed. Breast cancer was the most common cancer type (58% of total), comprising 87 (84%) of 103 females recruited. There were 34 men with prostate cancer (23% of total; 72% of males). Twenty-four (16%) had colorectal cancer and gynecological cancers added a further five patients. (Recruitment of these cancer types commenced very late in the protocol, hence their under-representation.) Median interval between cancer diagnosis and presentation of insomnia complaint to the research team was longer than 2 years. However, there was a considerable range in these values because some participants were recruited during first episode illness and others after cancer recurrence.

Table 1 summarizes other information on psychopathology (Hospital Anxiety and Depression Scale [HADS]), fatigue (Fatigue Symptom Inventory [FSI]), and cancer-related quality of life (QOL; Functional Assessment of Cancer Therapy Scale–general [FACT-G]). The HADS has been validated for use for patients with cancer to screen for anxiety and depressive symptoms.[28,29] Median values for both subscales was 8, however, 20% (CBT) and 23.3% (TAU) of participants screened ≥ 11 for HADS anxiety suggesting a sizeable minority had anxiety problems.[30] Applying this criterion to the HADS depression subscale revealed a smaller proportion (CBT, 9.4%; TAU, 7%) with depressive symptoms. Twenty participants (13%) were receiving concurrent treatment for depression.

The FACT-G[31] and the FSI[32] also have good validity and internal consistency.[33,34] Fatigue was marginally higher than that reported in other centers. For example, the FSI interference score (extent to which fatigue interferes with QOL) in our total sample was higher (median, 3.57; interquartile range, 1.86 to 5.14) than in a recent validation study (mean, 1.6; standard deviation [SD], 1.8).[34] Normative comparison on the FACT-G total score revealed that QOL in our sample (median, 80; IR 66 to 90) reflected published data from several large cohorts (mean, 81; SD, 17.0).[35]

All participants had insomnia longer than 6 months with group median of 30 months, and 25% had insomnia longer than 5 years. Two-thirds reported unrelenting insomnia and 23% took hypnotic medication ≥ 1 night of the 10-night baseline. Insomnia was typically severe (median PSQI, 13),[25] higher than in a recent cancer validation study (mean, 8.15; SD, 4.70).[26] Participants were not excessively sleepy during the daytime. Symptomatic sleep complaint comprised both difficulty falling asleep (group median SOL, 35.25 minutes) and difficulty maintaining sleep (WASO, 58.5 minutes.). This reflects clinical insomnia in our patients because total wakefulness was around 90 minutes, and total sleep time (TST) around 6.5 hours. Median SE was 80.6%, below the 85% lower limit of normal sleep.

Experimental Design

The study conformed to a pragmatic, two-center, randomized trial comparing CBT with TAU. Major assessments were at baseline, post-treatment, and follow-up 6 months later. Suitable participants were allocated to either CBT or TAU by means of the centralized computer-based registration/randomization service available within the Cancer Research UK Clinical Trials Unit, Glasgow. We stratified for center, prerandomization PSQI scores, existing treatment for insomnia, and tumor type using the minimization method. A 2:1 treatment allocation, in favor of the intervention, was selected because this made efficient use of available CBT sessions and minimized the time a

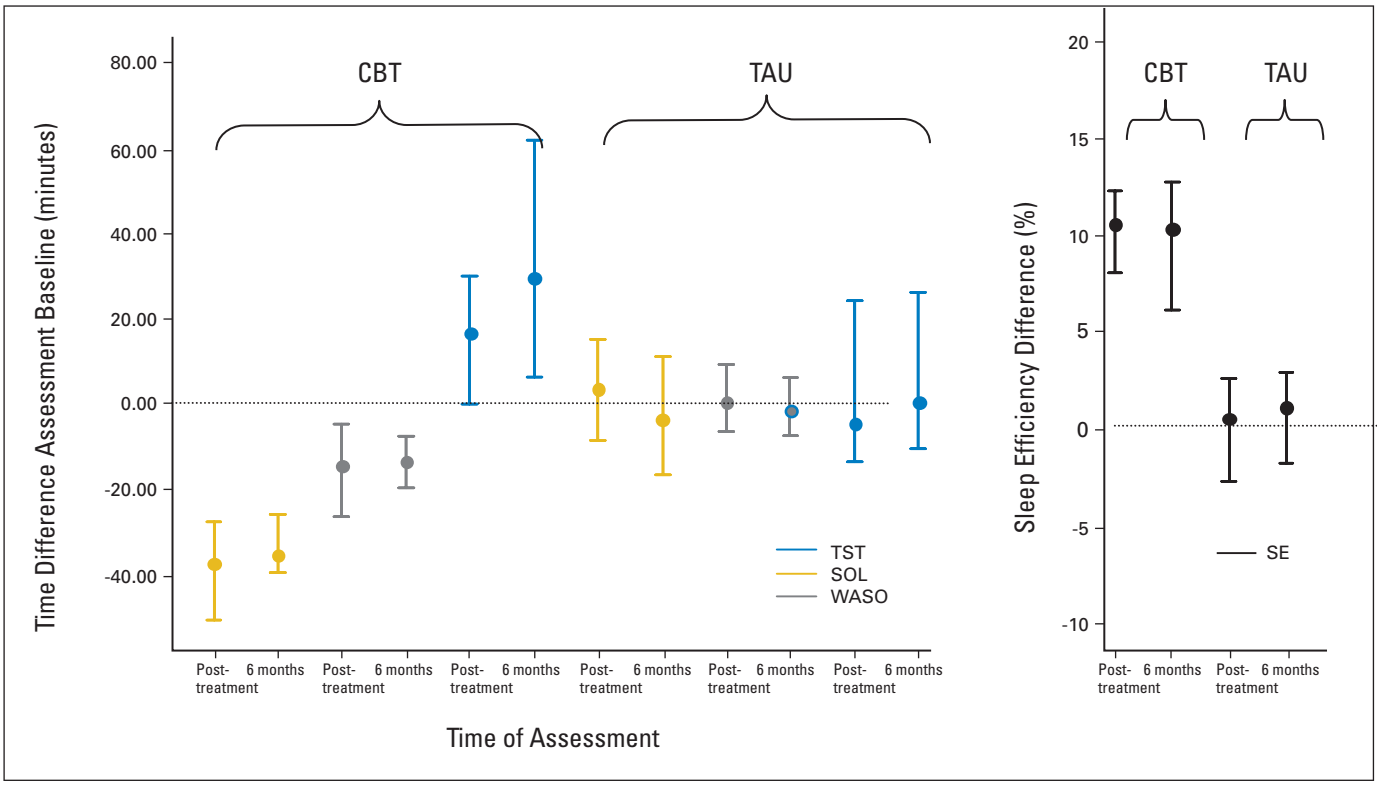


Fig 2. Changes in sleep diary measures from baseline by treatment arm. Points represent median change and the bars the range of a 95% CI for the median.

patient had to wait before starting CBT, thereby, reducing patient dropout. Due to the nature of the intervention, it was not possible to blind participants or therapists to allocation. No adverse events were reported with either CBT or TAU.

Measures

Potential participants were screened by telephone, when preliminary diagnostic information and medical/psychiatric history was collected. A comprehensive structured interview obtained a detailed history, supplemented by completion of the PSQI, Epworth sleepiness scale, HADS, FSI, and FACT-G.

Subjective sleep was assessed using a sleep diary,[12] completed for 10 days at each of three assessment points; baseline, post-treatment, and follow-up. Such diaries are the staple tool of insomnia assessment and offer a valid, relative index of sleep disturbance when used as repeated measures.[18] Items "how long did it take you to fall asleep last night" and "how long were you awake in total last night, after you first fell asleep?" assessed the central insomnia dimensions of difficulty initiating (SOL) and maintaining (WASO) sleep. The diary also enquired about bedtime and rising time, from which total time in bed, and thence SE were calculated: (SOL + WASO/time in bed) × 100. Participants were trained to complete diaries against established criteria.[36]

Actigraphy provided an objective estimate of sleep pattern over the same 10-day period. Actigraphs are nonintrusive devices that record movement through an accelerometer-microprocessor link.[37] Actigraphs (Cambridge Neurotechnology, AW-4, Cambridge, United Kingdom) were worn 24-hours per day on the nondominant wrist. An algorithm (maximum sampling frequency 32 Hz, recording all movement over 0.05g., filters set 3 to 11 Hz) enabled Sleepwatch (Cambridge Neurotechnology, Cambridge, United Kingdom) software to estimate the sleep parameters SOL/WASO/SE using 1-minute epochs.[43,44]

Data from the FSI and FACT-G taken at each assessment provided secondary outcome information to evaluate the effectiveness of CBT relative to TAU.

Interventions

CBT. Participants assigned to CBT attended five, weekly, 50-minute sessions during the early afternoon or early evening. These were conducted in groups of four to six at Maggie's Center (Glasgow) and the Clan Center (Aberdeen). These centers, run by the voluntary sector, offer an informal environment, close to but not part of the hospital itself. The content, aims, and objectives of each session are summarized in online-only Table A1 (further descriptions in Morin and Espie,[38] or from C.A.E.). The intervention included standard CBT components such as stimulus control, sleep restriction, and cognitive therapy strategies.

Therapists. We trained four experienced cancer nurses, who were released on a part-time basis from oncology nursing duties, to deliver CBT. (These were G-grade nurses in the United Kingdom National Health Service, equivalent to ward sister level of experience.) On average each nurse worked 6 hours per week on the study. We followed a model of training-to-criterion standards. That is, these nurses had to demonstrate competence in the practical delivery of the CBT program. This was ensured by participation in a short CBT course, apprenticeship learning opportunities, ongoing mentoring by an experienced clinical psychologist, and evaluation of audiotapes from randomly selected sessions.

TAU and integrity-fidelity of treatment allocation. Effectiveness studies should replicate real clinical practices and reflect validity and generalizability.[39] Because we were recruiting cancer patients with chronic insomnia, we expected concurrent physical and psychological symptoms, as well as concurrent treatments (except they had completed active anticancer treatment). The TAU comparison group thus represented normal clinical practice, where physicians were free to offer appointments, to prescribe, and to maintain/discontinue prescriptions. Effectively, TAU participants received no additional help for their insomnia. CBT was, in reality, a CBT plus TAU condition because the protocol explicitly permitted normal continuation of health care. At the end of the protocol, the TAU group was provided with *The Good Sleep Guide*.[40]

The integrity of the treatment allocations was ensured using several procedures (Table A2, online only).

Statistical Analyses

The study was designed to have 80% power to detect a standardized difference of 0.5 between the treatments (consistent with published meta-analytic data)[18-20] in the four primary sleep outcome measures (SOL, WASO, TST, SE) at post-treatment. A significance level of 0.0125 was chosen to adjust for multiple comparisons. These criteria implied recruiting 204 participants. In practice, slow recruitment meant that a total of 150 patients was randomly assigned, giving 80% power to detect a slightly larger standardized difference of 0.59.

The study was analyzed on an intention-to-treat basis. Comparison between treatments in terms of sleep and QOL variables was derived from fitting a linear mixed model using a first-order autoregressive variance structure, incorporating baseline value and other variables used for minimization as covariates. Data were transformed to approximate normality guided by the constructed variable approach before applying the model.[45] Interactions between SE and baseline parameters were examined in the context of this model; for this purpose continuous baseline data were dichotomized at the median.

P values for QOL end points were adjusted for multiple comparisons within each assessment time point using the Hochberg procedure.[46] In Figures 2 and 3 showing median change (95% CIs) in sleep and QOL parameters from baseline, confidence limits were derived from bootstrap sampling. Association between changes in QOL and

Table 2. Estimates of Standardized Effects (CBT-TAU) From the Mixed Model Applied to QOL Data

	Post-Treatment			6-Month Follow-Up		
Model	Standardized Effect	95% CI	*P*	Standardized Effect	95% CI	*P*
HADS						
Anxiety	−0.57	−0.96 to −0.18	.005*	−0.52	−0.92 to −0.12	.011*
Depression	−0.67	−1.06 to −0.28	.001†	−0.59	−0.99 to −0.19	.004*
FACT						
Physical	0.58	0.19 to 0.97	.004*	0.74	0.34 to 1.14	< .001†
Social	0.42	0.03 to 0.81	.036	0.13	−0.27 to 0.53	.529
Emotional	0.39	−0.01 to 0.78	.057	0.16	−0.25 to 0.57	.444
Functional	0.86	0.47 to 1.25	< .001†	1.17	0.77 to 1.57	< .001†
FSI						
Interference	−0.81	−1.20 to −0.42	< .001†	−.82	−1.22 to −0.42	< .001†

Abbreviations: CBT, cognitive behavior therapy; TAU, treatment as usual; QOL, quality of life; HADS, Hospital Anxiety and Depression Scale; FACT, Functional Assessment of Cancer Therapy; FSI, Fatigue Symptom Inventory.
*Significant at 5% after adjustment for multiple comparisons within each time point using the Hochberg procedure.
†Significant at 1% after adjustment for multiple comparisons within each time point using the Hochberg procedure.

changes in SE from baseline were examined using Spearman's rank correlation coefficient. Association between actigraphic and diary measures was also assessed using Spearman's correlation. Finally, the proportion of patients achieving SE higher than 85% was compared between study arms using logistic regression incorporating the baseline value and the other variables used for minimization.

RESULTS

Two thirds of CBT participants attended all therapy sessions, and 94% attended ≥ 3. For missed sessions, participants caught up at the end of the subsequent attended session. There were similar levels of attrition during the treatment phase, 18% from CBT (n = 15), and 16% from TAU (n = 7). It is possible that the dropout rate in CBT during the treatment phase was due to some people not liking the treatment. However, we have no data to confirm or refute this. Respectively, 87% and 84% of those receiving CBT and TAU provided post-treatment data, and 79% and 80% completed assessments at 6 months. Those lost to follow-up did not respond to two subsequent letters or phone calls. Baseline variables for noncompleters did not differ significantly from those for completers.

Sleep Pattern

Figure 2 presents a visual comparison, by treatment arm, of median change scores (baseline to post-treatment, baseline to follow-up) for each self-reported sleep variable. Table 2 provides estimates of standardized effects (CBT and TAU) relating to these variables.

At post-treatment, CBT was associated with median reduction in SOL of 16 minutes (95% CI, 10 to 22 minutes), and in WASO of

Table 3. Sleep Diary Results Representing Median IQR Values for the CBT and TAU Groups at Each Assessment Point, and Estimates of Standardized Effects (CBT-TAU) From the Mixed Model

Result	CBT	TAU	Standardized Effect*	95% CI	*P*
Sleep onset latency					
Baseline, median	41.0	27.4			
IQR	20.3-64.8	22.4-50.0			
Sample size	85	43			
Post-treatment, median	19.3	27.0	−0.86	−1.25 to −0.46	< .001
IQR	11.9-26.6	16.1-52.8			
Sample size	74	40			
6-month follow-up, median	19.3	22.4	−0.66	−1.06 to −0.26	.001
IQR	11.1-28.5	15.5-37.5			
Sample size	68	39			
Total sleep time					
Baseline, median	399.0	392.0			
IQR	343.3-455.9	348.0-457.9			
Sample size	85	43			
Post treatment, median	426.3	409.0	0.27	−0.12 to 0.66	.167
IQR	370.1-456.8	327.3-453.3			
Sample size	74	41			
6-month follow-up, median	438.7	413.5	0.39	−0.01 to 0.79	.054
IQR	408.6-470.6	354.0-493.0			
Sample size	68	39			
Wake time after sleep onset					
Baseline, median	62.0	51.0			
IQR	40.7-107.5	30.5-82.0			
Sample size	85	43			
Post-treatment, median	27.0	51.0	−0.97	−1.36 to −0.58	< .001
IQR	14.0-57.5	33.0-93.3			
Sample size	74	41			
6-month follow-up, median	26.1	34.0	−0.76	−1.16 to −0.36	< .001
IQR	12.6-59.4	22.5-78.0			
Sample size	68	39			
Sleep efficiency					
Baseline, median	80.4	82.4			
IQR	69.5-85.8	74.5-88.5			
Sample size	85	43			
Post-treatment, median	89.8	82.0	1.09	0.69 to 1.48	< .001
IQR	81.2-94.0	73.8-89.1			
Sample size	74	40			
6-month follow-up, median	89.8	88.5	0.88	0.48 to 1.28	< .001
IQR	82.1-94.0	72.7-91.5			
Sample size	68	39			

Abbreviations: IQR, interquartile range; CBT, cognitive behavior therapy; TAU, treatment as usual.
*For CBT-TAU.

38 minutes (95% CI, 28 to 59 minutes), the corresponding median reductions following TAU were 0 minutes (95% CI, −8.5 to 6.6) and 2 minutes (95% CI, −15 to 9). Effect sizes were moderate to large and were both highly statistically significant ($P < .001$). TST also increased by a median of 16 minutes (95% CI, −1 to 30) with CBT compared with 5 minutes (95% CI, −14 to 24) after TAU, but the difference between arms was not statistically significant. SE increased by 10% (95% CI, 9% to 12%) after CBT; the change in the TAU was 0% (95% CI, −3% to 3%). This effect size was large and highly statistically significant.

This pattern of results generally held at 6 months post-treatment. Effect sizes were somewhat reduced for WASO, SOL, and SE but remained moderate and statistically significant ($P < .001$). Changes in TST again were not statistically significant.

In summary, CBT was associated with median reduction in insomnia symptoms of almost 1 hour (SOL + WASO) compared with no change following TAU. Post-treatment and follow-up SE of 85% is commonly regarded as the lower limit of normal sleep. A higher proportion of CBT participants achieved this criterion, 51% (51 of 100) versus 34% on TAU (17 of 50; $P = .008$); at 6 months this difference was no longer significant (44%; 44/100 of patients on CBT; 48%; 24 of 50 on TAU; $P = .966$).

Tables 3 and 4 presents actigraphic results for the same sleep variables. Moderate effect sizes in favor of CBT were observed for SOL and WASO post-treatment (significant at $P < .05$). A large effect was observed on TST reflecting a reduction in sleep in the CBT arm. This reflects the impact of the sleep restriction component. At 6 months, no significant actigraphic effects were observed. It should also be noted that the SOL and WASO changes were not significant when adjusted to the conservative 1.25% level of statistical significance. Associations between actigraphic and corresponding sleep diary measures of SOL, WASO, TST and SE were modest (0.37, 0.25, 0.47 and

Table 4. Actigraph Results Representing Median Values for the CBT and TAU Groups at Each Assessment Point, and Estimates of Standardized Effects From the Mixed Model

Parameter	CBT	TAU	Standardized Effect*	95% CI	*P*
Sleep onset latency					
Baseline, median	14.8*	10.2			
IQR	6.6-23.7	4.0-22.1			
Sample size	84	42			
Post-treatment, median	7.8	9.6	−0.41	−0.80 to −0.01	.046
IQR	4.9-14.9	4.5-18.4			
Sample size	74	39			
6-month follow-up, median	9.5	11.7	−0.35	−0.76 to 0.06	.092
IQR	4.6-19.7	4.5-23.0			
Sample size	67	38			
Total sleep time					
Baseline, median	427.5	418.2			
IQR	391.7-453.7	391.6-458.8			
Sample size	84	42			
Post-treatment, median	396.7	425.5	−0.81	−1.21 to −0.42	< .001
IQR	367.3-430.7	397.3-453.2			
Sample size	74	39			
6-month follow-up	408.4	416.4	−0.19	−0.60 to 0.21	.351
IQR	371.7-443.0	381.2-443.7			
Sample size	67	38			
Wake time after sleep onset					
Baseline, median	61.6	62.4			
IQR	46.6-82.5	46.8-80.5			
Sample size	84	43			
Post-treatment, median	52.8	61.8	−0.50	−0.89 to −0.10	.014
IQR	37.7-68.0	44.1-88.1			
Sample size	74	39			
6-month follow-up, median	59.5	61.2	−0.29	−0.70 to 0.12	.164
IQR	39.5-77.0	45.6-87.9			
Sample size	67	38			
Sleep efficiency					
Baseline, median	84.2	83.5			
IQR	77.9-87.1	80.1-87.3			
Sample size	84	42			
Post-treatment, median	85.4	85.6	0.13	−0.26 to 0.52	.517
IQR	80.1-88.7	80.4-88.3			
Sample size	74	39			
6-month follow-up, median	84.6	83.3	0.33	−0.07 to 0.74	.105
IQR	79.0-88.6	78.1-87.5			
Sample size	67	38			

Abbreviations: IQR, interquartile range; CBT, cognitive behavior therapy; TAU, treatment as usual.
*For CBT-TAU.

0.31, respectively at baseline). However, these are typical of relationships observed in other contemporary studies.[43]

QOL

QOL outcomes are reported as standardized effect sizes (Table 2) with change score comparisons graphed in Figure 3. Effect sizes were moderate to large for five of seven comparisons, indicating that CBT was associated with improved QOL relative to TAU, at post-treatment and at follow-up. More specifically, CBT participants had reduced symptoms of fatigue, anxiety, and depression, and increased physical and functional QOL relative to TAU. Correlations between changes in SE from baseline to post-treatment after CBT and changes in statistically significant QOL measures were low.

Dependence of Treatment Effect on Baseline Characteristics

No statistically significant interactions were found between the CBT effect and sex ($P = .640$), age ($P = .402$), civil status ($P = .464$), occupational status ($P = .884$), Functional Assessment of Cancer Therapy (FACT)-Physical ($P = .471$), FACT-Functional ($P = .186$), or FSI interference ($P = .892$). Similarly, baseline sleep quality ($P = .883$), insomnia duration ($P = .645$), and psychological states (anxiety, $P = .471$; depression, $P = .887$) did not mediate response. There was a significant interaction with tumor type ($P = .027$) and treatment location ($P = .012$); in a model containing both these terms only treatment location retained statistical significance. Average standardized effect was higher in Glasgow (1.28; less than .0011) compared with Aberdeen (0.64; $P = .002$), although both were statistically significant and indicate benefit from CBT.

Qualitative Reports From Patients

We did not formally evaluate what patients thought of CBT. However, around 50 attended an open evening several months after completion of the trial. Table A3, online only, presents a sample of their evaluative comments.

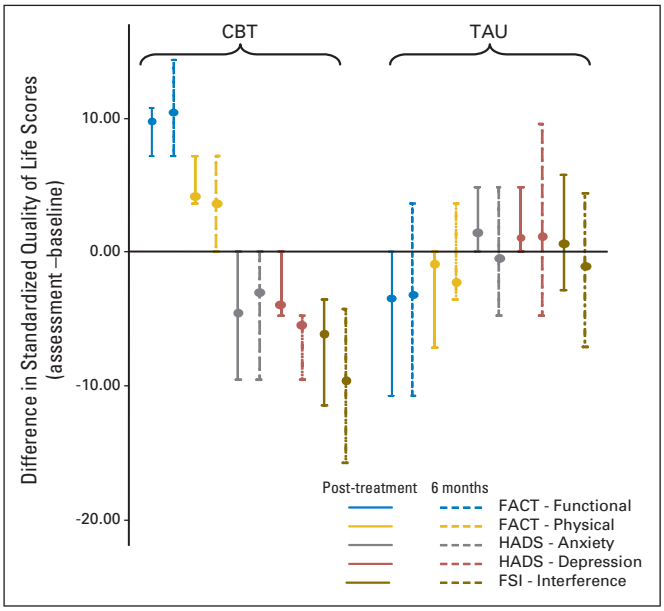


Fig 3. Changes in quality of life (QOL) measures from baseline by treatment arm. Points represent median change and the bars the range of a 95% CI for the median. QOL measures have been standardized onto a 0 to 100 scale.

DISCUSSION

Patients with cancer report that their sleep can be disturbed during stressful times associated with diagnosis/treatment, and persistently after discharge after anticancer therapy. Findings from this pragmatic trial suggest that a cognitive-behavioral approach may be clinically effective. Our results demonstrate sustained improvements in sleep with large effect sizes for subjectively estimated time taken to fall asleep and nocturnal wake time, comparable to the primary insomnia literature.[17-20] More modest effects at post-treatment were observed actigraphically. Importantly, CBT response was not attributable to any demographic or clinical subset. Moreover, we found generalized improvements in QOL, fatigue, and daytime well being.

Although psychologically based, CBT was delivered by oncology nurses, with no prior experience nor expertise in sleep medicine. This testifies to the potential of CBT itself, and to the feasibility of this treatment model. Scarce specialists might train/supervise available staff to deliver CBT as a first level intervention. Herein we used cancer nurses but we believe the important thing is that the program is delivered according to protocol, by a credible professional. We did not model cost effectiveness but extending the skills of available personnel could be economical, and permit patients with complex sleep problems to filter through for expert care. We also observed that this solution-focused approach was highly acceptable to patients and professionals. Indeed, insomnia may offer a nonstigmatizing entry point to psychological care.

Further studies are required, both in the efficacy and the effectiveness tradition. The former, explanatory approach emphasizes rigorous entry criteria and highly controlled quasiexperimental design. The latter, of which this study is a prototype, resembles real world practice, and informs care delivery. These designs are complementary and taken together will provide evidence to inform best practice for this neglected problem.

AUTHORS' DISCLOSURES OF POTENTIAL CONFLICTS OF INTEREST

The author(s) indicated no potential conflicts of interest.

AUTHOR CONTRIBUTIONS

Conception and design: Colin A. Espie, Leslie Samuel, Lynne M. Taylor, Craig A. White, Neil J. Douglas, Heather M. Engleman, Heidi-Louise Kelly, James Paul
Administrative support: Colin A. Espie, Leanne Fleming, James Cassidy, Lynne M. Taylor
Provision of study materials or patients: Colin A. Espie, James Cassidy, Leslie Samuel, James Paul
Collection and assembly of data: Colin A. Espie, Leanne Fleming, Lynne M. Taylor, James Paul
Data analysis and interpretation: Colin A. Espie, Leanne Fleming, James Paul
Manuscript writing: Colin A. Espie, Leanne Fleming, James Paul
Final approval of manuscript: Colin A. Espie, Leanne Fleming, James Cassidy, Leslie Samuel, Lynne M. Taylor, Craig A. White, Neil J. Douglas, Heather M. Engleman, Heidi-Louise Kelly, James Paul

REFERENCES

1. Kaye J, Kaye K, Madow L: Sleep patterns in patients with cancer and patients with cardiac disease. J Psych 114:107-113, 1983

2. World Health Organization: Cancer Pain Relief. Geneva, Switzerland, World Health Organization, 1986

3. Munro AJ, Potter S: A quantitative approach to the distress caused by symptoms in patients treated with radical radiotherapy. Br J Cancer 74: 640-647, 1996

4. Savard J, Simard S, Blanchet J, et al: Prevalence, clinical characteristics, and risk factors for insomnia in the context of breast cancer. Sleep 24: 5:583-590, 2001

5. Davidson JR, MacLean AW, Brundage MD, et al: Sleep disturbance in cancer patients. Social Science and Medicine 55:313-322, 2002

6. American Sleep Disorders Association: International Classification of Sleep Disorders: Diagnostic and Coding Manual (revised). Rochester, MA, American Sleep Disorders Association, 1997

7. Roth T, Ancoli-Israel S: Daytime consequences of insomnia in the United States: Results of the 1991 National Sleep Foundation survey II. Sleep 22:S354-S363, 1999 (suppl 2)

8. Sateia MJ, Nowell PD: Insomnia. Lancet 364: 1959-1973, 2004

9. Kripke D: Hypnotic drugs: Deadly risks, doubtful benefits. Sleep Medicine Reviews 4:5-20, 2000

10. Buysse DJ: Rational pharmacotherapy for insomnia: Time for a new paradigm. Sleep Medicine Reviews 4:521-527, 2000

11. Mendelson WB: A review of the evidence for the efficacy and safety of trazodone. J Clin Psychiatry 66:469-476, 2005

12. National Institute for Clinical Excellence: Guidance on the use of zaleplon, zolpidem and zopiclone for the short-term management of insomnia. Technology Appraisal Guidance 77:1-27, 2004

13. Curt GA: Fatigue in cancer. BMJ 322:1560, 2001

14. Morin CM, Hauri P, Espie CA, et al: Nonpharmacologic treatment of chronic insomnia: An American Academy of Sleep Medicine review. Sleep 22:1134-1156, 1999

15. Morin CM, Bootzin RR, Buysse DJ, et al: Psychological and behavioral treatment of insomnia: Update of the recent evidence (1998-2004): Prepared by a Task Force of the American Academy of Sleep Medicine. Sleep 29:1398-1414, 2006

16. Irwin MR, Cole JC, Nicassio PM: Comparative meta-analysis of behavioral interventions for insomnia and their efficacy in middle-aged adults and in older adults 55+ years of age. Health Psychol 25:3-14, 2006

17. Morin CM, Culbert JP, Schwartz MS: Nonpharmacological interventions for insomnia: A meta-analysis of treatment efficacy. Am J Psychiatry 151:1172-1180, 1994

18. Murtagh DR, Greenwood KM: Identifying effective psychological treatments for insomnia: A meta-analysis. J Consult Clin Psychol 63:79-89, 1995

19. Smith MT, Perlis ML, Park A, et al: Behavioral treatment vs. pharmacotherapy for insomnia: A comparative meta-analyses. Am J Psychiatry 159:5-11, 2002

20. Morin CM, Espie CA: Insomnia: A Clinical Guide to Assessment and Treatment. New York, NY, Kluwer Academic/Plenum Publishers, 2003

21. Savard J, Morin CM: Insomnia in the context of cancer: A review of a neglected problem. J Clin Oncol 19:895-908, 2001

22. Cannici J, Malcolm R, Peek LA: Treatment of insomnia in cancer patients using muscle relaxation training. J Behavior Therapy and Experimental Psychiatry 14: 3:251-256, 1983

23. Savard J, Simard S, Ivers H, et al: A randomized study on the efficacy of cognitive-behavioral therapy for insomnia secondary to breast cancer: Part 1 – sleep and psychological effects. J Clin Oncol 23:6083-6096, 2005

24. American Psychiatric Association: Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition (DSM-IV). Washington, DC, American Psychiatric Association, 1994

25. Buysse DJ, Reynolds CF, Monk TH, et al: The Pittsburgh Sleep Quality Index: A new instrument for psychiatric practice and research. Psychiatry Res 28:193-213, 1989

26. Beck SL, Schwartz AL, Towsley G, et al: Psychometric evaluation of the Pittsburgh sleep quality index in cancer patients. J Pain Symptom Manage 27:140-148, 2004

27. Johns MW: A new method for measuring daytime sleepiness: The Epworth Sleepiness Scale. Sleep 14:450-545, 1991

28. Ibbotson T, Maguire P, Selby P, et al: Screening for anxiety and depression in cancer patients: The effects of disease and treatment. Eur J Cancer 30A:37-40, 1994

29. Razavi D, Delvaux N, Farvacques C, et al: Screening for adjustment disorders and major depressive disorders in cancer in-patients. Br J Psychiatry 156:79-83, 1990

30. Zigmond AS, Snaith RP: The Hospital Anxiety and Depression Scale. Acta Psychiatrica Scandanavica 67:361-370, 1983

31. Cella DF, Tulsky DS, Gray G, et al: The Functional Assessment of Cancer Therapy scale: Development and validation of the general measure. J Clin Oncol 11:570-579, 1995

32. Hann DM, Jacobsen PB, Azzarello LM, et al: Measurement of fatigue in cancer patients: Development and validation of the Fatigue Symptom Inventory. Qual Life Res 7:301-310, 1998

33. Webster K, Odom L, Peterman A, et al: The Functional Assessment of Chronic Illness Therapy (FACIT) measurement system: Validation of version 4 of the core questionnaire. Quality of Life Research 8:604-611, 1999

34. Hann DM, Denniston MM, Baker F: Measurement of fatigue in cancer patients: Further validation of the Fatigue Symptom Inventory. Qual Life Res 9:847-854, 2000

35. Brucker PS, Yost K, Cashy J, et al: General population and cancer patient norms for the Functional Assessment of Cancer Therapy-General (FACT-G). Evaluation and the Health Professions 28:192-211, 2005

36. Davidson JR, Waisberg JL, Brundage MD, et al: Nonpharmacologic group treatment of insomnia: A preliminary study with cancer survivors. Psycho-Oncology 10:389-397, 2001

37. Chesson AL, Hartse K, Anderson WM, et al: Practice parameters for the evaluation of chronic insomnia. Sleep 23:237-241, 2000

38. Morin CM, Espie CA: Insomnia: A Clinical Guide to Assessment and Treatment. New York, NY, Kluwer Academic/Plenum Publishers, 2003

39. Clarke GN: Improving the transition from basic efficacy research to effectiveness studies: Methodological issues and procedures. J Consult Clin Psychol 63:718-725, 1995

40. National Medical Advisory Committee: The Management of Anxiety and Insomnia. Edinburgh, Scotland, Scottish Office Home & Health Department, HMSO, 1993

41. Espie CA, Inglis SJ, Tessier S, et al: The clinical effectiveness of cognitive behavior therapy for chronic insomnia: Implementation and evaluation of a sleep clinic in general medical practice. Behavior Research and Therapy 39:45-60, 2001

42. Espie CA, MacMahon KMA, Kelly HL, et al: Randomized clinical effectiveness trial of nurse-administered small group CBT for persistent insomnia in general practice. Sleep 30:574-584, 2007

43. Tryon WW: Issues of validity in actigraphic sleep assessment. Sleep 27:158-165, 2004

44. Ancoli-Israel S, Cole R, Alessi C, et al: The role of actigraphy in the study of sleep and circadian rhythms. Sleep 26:342-392, 2003

45. Atkinson AC: Regression diagnostics, transformations and constructed variables. Journal of the Royal Statistical Society, Series B-Methodological 44:1-36, 1982

46. Hochberg Y: A sharper Bonferroni procedure for multiple tests of significance. Biometrika 75:800-803, 1988

Acknowledgment

We thank the following institutions for recruitment assistance: Beatson Oncology Centre, Western Infirmary, Gartnavel Hospital, Stobhill Hospital, Aberdeen Royal Infirmary, Dr Greys Hospital, Maggie's Centre, and Clan Centre. We also thank Patricia Munro, Dorothy Molloy, Margaret Durward, Kenny Ferguson, and Alison Thomson for their support.

Appendix

The Appendix is included in the full-text version of this article, available online at www.jco.org. It is not included in the PDF version (via Adobe® Reader®).

"Stepped Care": A Health Technology Solution for Delivering Cognitive Behavioral Therapy as a First Line Insomnia Treatment

Colin A. Espie, PhD

University of Glasgow Sleep Centre, Sackler Institute of Psychobiological Research, Southern General Hospital, Scotland, UK

There is a large body of evidence that Cognitive Behavioral Therapy for insomnia (CBT) is an effective treatment for persistent insomnia. However, despite two decades of research it is still not readily available, and there are no immediate signs that this situation is about to change. This paper proposes that a service delivery model, based on "stepped care" principles, would enable this relatively scarce healthcare expertise to be applied in a cost-effective way to achieve optimal development of CBT services and best clinical care. The research evidence on methods of delivering CBT, and the associated clinical leadership roles, is reviewed. On this basis, self-administered CBT is posited as the "entry level" treatment for stepped care, with manualized, small group, CBT delivered by nurses, at the next level. Overall, a hierarchy comprising five levels of CBT stepped care is suggested. Allocation to a particular level should reflect assessed need, which in turn represents increased resource requirement in terms of time, cost and expertise. Stepped care models must also be capable of "referring" people upstream where there is an incomplete therapeutic response to a lower level intervention. Ultimately, the challenge is for CBT to be delivered competently and effectively in diversified formats on a whole population basis. That is, it needs to become "scalable". This will require a robust approach to clinical governance.

Keywords: Insomnia, psychological treatment, cognitive behavior therapy, primary care, population

Citation: Espie CA. "Stepped care": A health technology solution for delivering Cognitive Behavioral Therapy as a first line insomnia treatment. *SLEEP* 2009;32(12):1549-1558.

THERE IS COMPELLING EVIDENCE THAT COGNITIVE BEHAVIORAL THERAPY (CBT) IS A LASTINGLY EFFECTIVE TREATMENT FOR CHRONIC PRIMARY INSOMNIA, and mounting evidence that it is similarly effective for persistent insomnia associated with medical or psychiatric disorders.

The challenge for CBT is no longer to prove its credentials, but to punch its weight. For at least a decade, CBT should have been a contender as the treatment of first choice for insomnia. In reality, however, it has had very little impact on the high volume of insomnia patient care. Indeed, it has amounted to little more than a patchy cottage industry.

This is not a criticism of individual professionals, or of groups of practitioners, or of local service initiatives. People are doing their best; as indeed we have tried to do in Scotland. Rather, it is stark recognition that the challenge for CBT with respect to delivery is very much greater than so far envisaged.

The argument in this paper is that insomnia constitutes an international public health problem. As such, it needs to be addressed systemically not just clinically; that is at the level of care organisation. Prevalence and morbidity data alone demonstrate that CBT would need to be scaled up enormously if it were to address population need. This will not happen soon, or at all, unless new horizons are scanned. There needs to be serious engagement with models that are capable of supporting regional, national and international CBT service delivery.

The principal aim of this paper is to open debate on one such model, based upon the "stepped care" approach. Before doing so, a brief review of CBT is presented to reflect upon its evidence base and its suitability as a "health technology" for adaptation to stepped care.

Submitted for publication April, 2009
Submitted in final revised form July, 2009
Accepted for publication July, 2009
Address correspondence to: Colin A. Espie, PhD, University of Glasgow Sleep Centre, Sackler Institute of Psychobiological Research, Section of Psychological Medicine, Faculty of Medicine, Southern General Hospital, Glasgow G 51 4TF, Scotland, UK; E-mail: c.espie@clinmed.gla.ac.uk

The Efficacy of CBT

Nine systematic reviews or meta-analyses of CBT have been published in the past 15 years.[1-9] To take two examples, the American Academy of Sleep Medicine [AASM (formerly the American Sleep Disorders Association of Sleep)] taskforce reports (1999 and 2006)[4,8] comprised 85 clinical trials (4,194 participants), and indicated that CBT was associated with improvement in 70% of patients, that was sustained at least 6 months post-treatment. Importantly, there is growing evidence not only that sleep parameters improve, but also reports of daytime functioning. These clinical and generalized benefits reflect moderate to large standardised effect sizes (ES). Moreover, the 2006 review included 12 trials on insomnia associated with medical or psychiatric disorders, suggesting that CBT may be effective also in more complex populations.

Based on these data, AASM published practice parameters statements[10,11] using standardized appraisal criteria[12,13] endorsing the efficacy of stimulus control therapy, progressive muscular relaxation, biofeedback, paradoxical intention therapy, sleep restriction and two alternative multi-component CBT approaches. It is important then to note that CBT is a treatment modality, just as is sleep pharmacotherapy (PCT). The latter comprises a range of licensed medications, and the former a range of (seven) proven psychotherapeutic methods. Please also note that "sleep hygiene" is not one of these.

In routine practice, the overwhelming majority of insomnia patients is treated with PCT rather than CBT. In contrast to CBT, this is not evidence-based for chronic insomnia.[5,9,14] There are no data to support the long-term resolution of sleep problems following either short-term or medium-term (up to 6 months) PCT, whereas the beneficial effects of CBT are known to persist for months or years after the treatment course is completed. For example, Morin et al. (1999) compared CBT, medication (temaza-

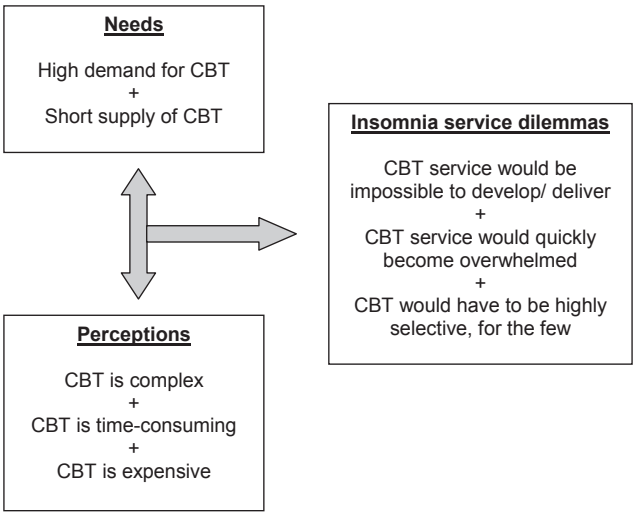


Figure 1—The CBT feasibility agenda.

pam), combined therapy, and a placebo control condition.[15] All three active treatments produced short-term improvements in sleep, but the temazapam-only condition regressed to baseline during follow-up. By comparison, both groups treated with CBT exhibited good 12-month outcome, suggesting the durable efficacy of CBT relative to PCT. Recently, Sivertsen et al. (2006) reported that CBT was associated with greater benefit than zopiclone.[16] CBT was associated with a 10% increase in polysomnographic sleep efficiency (SEFF) at post-treatment and 6-month follow-up, relative to no reliable change with zopiclone.

Based on the published evidence, the National Institutes of Health Consensus and State of the Science Statement (2005) concluded that CBT is "*as effective as prescription medications are for short-term treatment of chronic insomnia. Moreover, there are indications that the beneficial effects of CBT, in contrast to those produced by medications, may last well beyond the termination of active treatment*" (page 14).[14]

The Clinical Effectiveness of CBT

For any treatment to become a "gold standard" in routine care it is important to have effectiveness data as well as efficacy data. Effectiveness studies speak to the generalizability of beneficial effects to the population at large,[17] and although research of this type is limited for CBT (as it is for PCT), there are indications that CBT may be effective across a range of presenting populations.

For example, a comparative meta-analysis, comparing CBT outcomes in middle-aged and older adults (55 years plus), reported moderate to large ES, regardless of age, in sleep onset latency (SOL) and wake time after sleep onset (WASO).[7] Consistent with this, AASM also recommend CBT as a standard treatment for insomnia in older adults.[11] Likewise, a recent randomized controlled trial has found that CBT is clinically helpful in depressed patients with co-morbid insomnia. Manber et al (2008) compared citalopram + CBT with citalopram + "sham" CBT, and reported significant benefits not only to sleep but to depression itself, reflected in an ES difference of 0.24 in favour of citalopram + CBT on the Hamilton Rating Scale for Depression.[18]

We have recently reported on the effectiveness of CBT in post-cancer care[19]; reinforcing the earlier findings of Savard et al.[20] that CBT is effective in insomnia associated with cancer. Indeed, much of our work in Glasgow has been in the clinical effectiveness tradition, enrolling relatively unselected patients into our trials programme. We have randomized 490 patients across 3 clinical trials of CBT versus treatment as usual,[19,21,22] and have not found any consistent pattern of demographic or clinical predictors of poor response to CBT. In other words, our findings support the effectiveness of CBT, obtaining an approximate 70% treatment response regardless of severity or chronicity of presenting characteristics.[23] This is consistent with other UK data showing that insomnia in chronic hypnotic users also responds well to CBT.[24]

The Challenge for CBT

All treatment modalities evolve over time as active elements are identified and refined, as new interventions are evaluated, and as efficacy data are replicated in clinical effectiveness paradigms. CBT methods are no exception. There always will be questions on treatment outcome to answer – but this is not the main challenge. At this point, doubts about CBT do not reside in its efficacy, nor even in its effectiveness, but in its feasibility. *Can CBT really become a first line treatment for insomnia in everyday practice?*

Figure 1 illustrates some of the dilemmas to be faced in considering how to offer CBT services to the population who may benefit from it. *Can a CBT service delivery model be developed to cope with high volume of need, safely and effectively, whilst still being affordable?*

Stepped Care as a Potential Vehicle for CBT Service Delivery

An "insomnia care pathway" is required if we are to prevent nascent CBT services from being strangled at birth. Such a pathway would fit well with a stepped care design solution. We have previously suggested how a CBT for insomnia stepped care "triage" system might work,[22,64] as have others.[65] Indeed, the potential of stepped care has been discussed at professional and scientific meetings for some time. Stepped care offers a generic approach to care management,[25] and is often conceptualised as a pyramid. As can been seen in Figure 2, high patient volume is managed at the base of the pyramid using low intensity treatments, with progressively smaller volumes, and greater expertise in assessment and treatment, being concentrated towards the top step. Certainly in the UK, an important part of mental health service commissioning is focussing upon the urgent need to increase access to psychological therapies for common mental health problems, with stepped care being proferred as part of that solution.[26]

Crucial, to the successful and safe operation of stepped care, is that the level of intervention that people receive, either initially or subsequently, is not arbitrary; rather, it should reflect assessed need. Moreover, the number of steps in any stepped care model would be determined by the number of interventions or levels of intervention that are proven and available, and also by the upper limits of what within the healthcare system would be affordable.

Let us then consider criteria for selecting an "entry level" treatment within a stepped care system. At this level, represented by the base of the pyramid, the least restrictive therapy has to be identified (Figure 2). According to Bower & Gilbody (2005),[25] this should be a:

Table 1—Assumptions Underpinning the Stepped Care Model [adapted from Bower & Gilbody, 2005] [25]

1. Equivalence assumption	Minimal interventions can provide "significant health gain"
2. Efficiency assumption	Using minimal interventions reflects healthcare resource
3. Acceptability assumption	Stepped care is acceptable to patients and to professionals

- *readily accessible* form of treatment,
- *provided at the lowest cost* and
- *least personal inconvenience* to patients, and
- requiring the *lowest treatment intensity* and
- the *least specialist time*.

Bower and Gilbody go on to suggest several important principles or assumptions underlying stepped care. These are summarized in Table 1.

First, this least restrictive therapy must satisfy what is known as the equivalence assumption. That is, despite being a minimal intervention, the entry level treatment must be evidence-based to provide significant health gain. That is, a deliverable and relatively inexpensive treatment that can benefit at least a substantial proportion of patients, but without risking adverse effects. So, for example, if a simple and easily administered benign treatment had been shown to lead to remission or clinical response, in say 30% of patients, it would be equivalent in outcome for these patients to any more complex, more time-consuming, or more expensive intervention that they might otherwise (unnecessarily) receive.

Second, and extending the above argument, the efficiency assumption supports the use of the minimal effective intervention at each step as a reality check upon the true (finite) availability of healthcare resources. That is, not only would it be effective to follow a stepped care approach, it would also be prudent. By investing resources across a spread of intervention steps, it is more likely that the maximum number of patients can be treated in an optimal way, within budget. Again, using the above example, the minimal intervention might meet the needs of say 30% of the patient population. Thus, it satisfies what would otherwise be a substantial demand on resources whilst actually consuming relatively little resource, and without detriment to care outcomes. At the whole population level, this helps to deliver population health gain because it provides effective treatment quickly to many, and conserves resources (time, expertise, funds) for those patients whose effective treatment will require greater resource.

Third, a stepped care service must be acceptable both to professionals and to patients. The acceptability assumption is addressed by capacity in the system both for an initial evaluation of need and for subsequent "self-correcting movement," so that patients can shift between levels when this is deemed appropriate. Supposing an individual starts off at a given level, a decision might be made later about discharge or about stepping up to the next level, based upon their progress. This might be appraised through review of validated data collected on their condition (e.g Insomnia Severity Index[27,28] scores against threshold criteria), or through clinician judgement that outcomes are or are not (yet) satisfactory. But also, the patient would be permitted the perspective that s/he may prefer to see someone with a high level of expertise and/or for longer, even though this might not appear to be necessary from an evidence-based perspective.

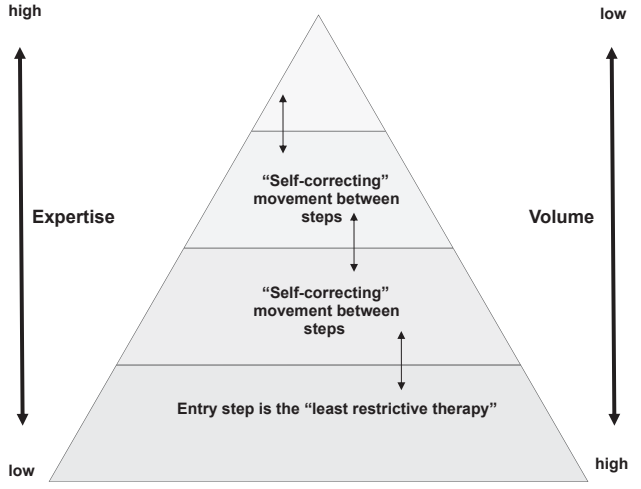


Figure 2—A generic stepped care model illustrating incremental levels (steps) of intervention complexity. The most efficient service will ensure maximal throughput by stepping patients according to need, matching interventions to needs, and making best use of available expertise.

Stepped care models have proven useful for a wide range of disorders. It is beyond the scope of this paper to go into this in detail, but, for example, a good review of stepped care for chronic illness was published recently[29] and stepped care for primary care patients with persistent depression has been encouraged for at least the past 10 years.[30] It is noteworthy in this latter respect that three meta-analyses of brief behavioral interventions for depression have been published within the past couple of years.[31-33]

Is Insomnia a Suitable Disorder for Stepped Care?

There are four main reasons for considering insomnia to be a suitable condition for stepped care.

First, insomnia is very common.[34,35] A problem that affects 20-25% of the adult population, and up to 10% on a chronic basis, requires a care pathway to be developed to manage it effectively. All practitioners and healthcare services operate on finite resources, hence the need to manage common problems both soundly and equitably.

Second, and related to the first, insomnia presents with varying levels of severity and complexity, from short-term troublesome symptom, through persistent primary disorder, to co-morbid disorder.[36] The fact that persistent insomnia is an established risk factor for mental ill-health[37,38] also suggests that judgement needs to be exercised to determine when, and at what level, a patient's insomnia should be addressed.

Third, there are pragmatic reasons to develop stepped care for the behavioral management of insomnia, not least of which is the shortage of skilled practitioners. There are relatively few people with sufficient expertise to treat the high volume of insomnia patients who might benefit from CBT. Some useful suggestions have been made about how numbers of behavioral sleep medicine specialists might be increased.[39] However, the

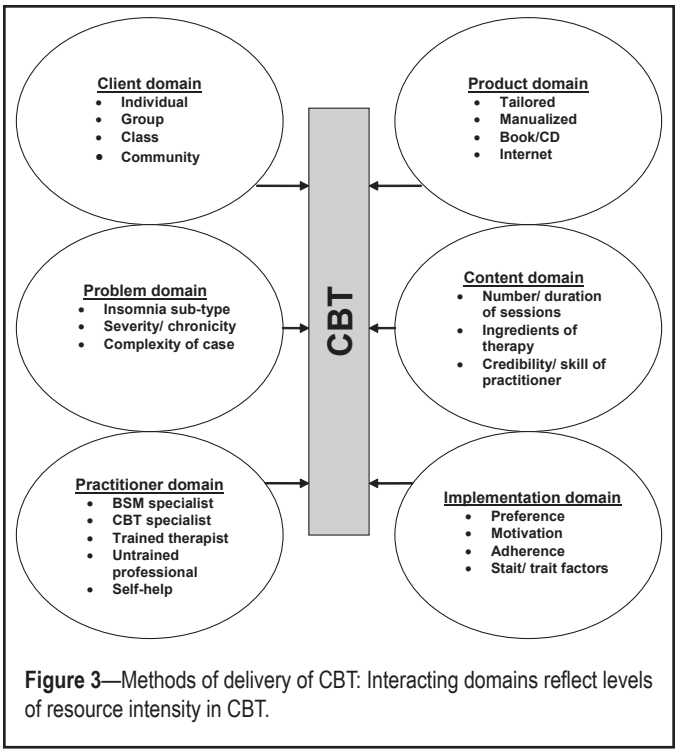


Figure 3—Methods of delivery of CBT: Interacting domains reflect levels of resource intensity in CBT.

base number remains very low, even in the US where sleep services are better established.

Fourth, insomnia is typically a chronic disorder that does not necessarily remit.[40] Certainly, there are everyday sequelae of insomnia, but these are seldom novel, acute, or inherently dangerous. Rather, the morbidities associated with persistent insomnia are longer term risks to health and well-being. Consequently, insomnia is a suitable disorder for stepped care, even at the entry level, because there may be few adverse consequences to providing a minimal intervention, even if a proportion of patients are initially non-responders. Besides, they would then be stepped up to a higher intensity treatment.

Finally, it should be noted that pharmacotherapy for insomnia also can be readily accommodated within a stepped care approach, ranging from herbal and other "medicinal" products that people purchase for themselves "over the counter" at one extreme, through primary care management, to expert prescription and review of licensed and "off label" medications. It is beyond the scope of this paper to discuss PCT in any detail, but it is important to acknowledge that the evidence-based treatment of insomnia at population level requires both CBT and PCT to be available and that, in practice, CBT and PCT may be offered to individuals in combination. Consideration of an insomnia stepped care pathway, therefore, does not intrinsically prejudice clinical practice or research study in one direction or the other.

Is CBT a Suitable Intervention for Stepped Care?

There are several compelling reasons for believing that CBT is an inherently suitable treatment modality for stepped care.

First, as psychotherapies go, CBT methods have proven to be remarkably adaptable, across a wide range of disorders.[26] CBT is no exception. Indeed, Figure 3 illustrates that there are six domains of resource intensity that might be (co-) varied to yield treatment solutions. So, for example, one-to-one therapy that is tailored specifically to a patient's complex sleep needs over numerous sessions delivered by an expert psychologist trained in behavioural sleep medicine would represent the most resource intensive approach. This may be necessary for some patients, but probably not for very many. This is a crucial point. The great majority of insomnia patients need not, should not, and could not (possibly) be treated by such highly qualified individuals.

At the other extreme, a sizeable proportion of patients might derive significant benefit from a guided self-help book, from participation in a CBT class in a community college or sports centre, or through a web-based CBT portal. There is an insufficient evidence base at present to differentiate these precisely, or indeed other resource combinations that might be constructed, but the point is that CBT is intrinsically an adaptable health technology.

Second, although a limited analogy, it may be helpful to think of stepped care as a "dosing schedule," whereby any given patient will require the necessary amount of intervention to respond (not less because it will not work, and not more because it will not add anything). "Dose" might be determined by the number of elements in the treatment, the number of sessions, the amount of personal tailoring of treatment, the qualifications and experience of the therapist, ….. and so on, as per the content of the domains in Figure 3. Consequently, entry level CBT should be the lowest dose proven to be associated with clinical improvement. Traditionally, a dose response is the relationship between the amount of exposure (dose) to a substance, and the resulting changes in body function or health (response). It has been suggested that the therapeutic session is the natural quantitative unit of psychotherapy. That is, the number of sessions is stochastically related to exposure to the active ingredients in any psychotherapy.[41] Following this rationale, Edinger et al. (2007) have shown that a brief CBT (4 session) intervention is clinically effective in primary insomnia, but less so in comorbid insomnia,[42] implying that dosing may need to reflect complexity of presentation. Whatever the input is chosen as the dosing unit, and whatever the limitations of the analogy in general, it does seem that CBT would be a suitable intervention for stepped care also because there are numerous escalating characteristics that might be applied to its delivery (cf. Figure 3).

Third, CBT is also suitable for stepped care because, in the real world, people do make choices about treatment. In one elegant study, behavioral and pharmacological treatment scenarios were offered to insomnia patients who rated these in terms of their acceptability.[43] Results showed that typical patient preference was for a behavioral treatment approach. We have recently replicated this preference for CBT in patients with psychophysiological insomnia and idiopathic insomnia. We have also demonstrated that some patients with idiopathic insomnia would be open to a (non-curative) acceptance-based approach to therapy.[44] Importantly, people make choices not only about the type of treatment that they would prefer, but they also make behavioural choices about home implementation of treatments. Such motivational processes are cognitive-behavioral in nature, again indicating the suitability of CBT for insomnia. So, whereas the number of sessions offered or attended reflects an administration (exposure) dose of CBT provided to the patient, the "absorbed" dose taken by the patient reflects their home

implementation/ adherence to the therapeutic instructions. We know, for example, that patients who achieve more consistent bedtimes and rising times have improved sleep efficiency [45] suggesting that application of advice is an important predictor of outcome. Likewise, it has been found in one study that only adherence explained variance in patients' post-treatment outcome.[46] Indeed, the importance of choice is evident in that not all patients with insomnia even seek help through traditional clinical routes.[47,48] Stepped care offers greater engagement with such issues by explicitly recognizing that acceptability is central to the way people behave (Table 1).

What would be the Entry Step for CBT that is Evidence Based?

In order to construct a stepped care model for CBT, it is of first importance to consider what would be the minimal intervention, known to provide significant health gain (Figure 2), that satisfies the assumptions summarized in Table 1.

Sleep Hygiene

In practice, the most common non-pharmacological approach is probably provision of sleep hygiene education, either verbally during consultation or by clinic leaflet. In terms of Figure 3, this represents self-help material, BUT with very limited CBT content either conceptually or therapeutically. Certainly it is unrestrictive (cf. Table 1), however, there is no evidence from trials data or from meta-analyses that sleep hygiene is an effective treatment. Indeed, sleep hygiene is not even mentioned in either the 2005 US NIH insomnia consensus statements, [14] or in the 2004 UK NICE technology appraisal on the management of insomnia.[49] Furthermore, current AASM insomnia practice parameters state that "*insufficient evidence is available for sleep hygiene education to be an option as a single therapy. Whether this therapy is effective when added to other specific approaches could not be determined from the available dat*a" (p. 1417).[11] Thus, the apparent proliferation of sleep hygiene advice as if it were a stand alone treatment, or even as an adjunct to PCT, is a practice that is independent of supporting data. Sleep hygiene does not meet the essential criteria for a minimally effective intervention offering significant health gain and so cannot be the first step within a stepped care CBT approach.

Manualized CBT

In contrast to sleep hygiene, an intervention "product" that does appear to be well evidenced is manualized CBT.[4,8,36] This is where the practitioner follows a highly structured treatment protocol based upon therapeutically indicated CBT elements (content domain in Figure 3). The manual helps to ensure treatment fidelity, and also consistency in clinical practice. Manualized CBT has been found to be effective also across a range of client groups from primary insomnia to insomnia associated with medical or psychiatric disorder. The majority of published studies has utilized individual therapy, typically provided by a psychologist, but a substantial minority has adopted a group treatment approach.[1-8]

Our own approach involves CBT practitioners delivering manualized CBT over five, weekly, small group sessions.[19,21,22] We have used nurses as therapists, for professional (e.g. familiar with protocols) and pragmatic (largest group of healthcare professionals) reasons. However, we believe that other staff (e.g. PSG staff, pharmacists) could deliver manualized CBT equally faithfully and effectively. This has yet to be formally evaluated. Taking our most recent trial as an example,[19] standardised relative ES at post-treatment and at 6-month follow up for CBT were medium to large for SOL, WASO, and SEFF, with a small ES for total sleep time. Medium ES were also found for reductions in anxiety, depressive and fatigue symptoms, and for improvements in health-related quality of life, indicating that the benefits of CBT may go beyond sleep pattern per se.

There is then substantial evidence for manualized CBT[1-8] and that it may be delivered in cost-effective ways.[19,21,22,50] Manualized CBT could be the entry level treatment in the stepped care model, but there may be a step below this.

Self-administered CBT

"Self-help" is a rapidly growing area within mental health, and consistent with this, bibliotherapy, CD-ROM, televised media and internet-delivered CBT methods have now been reported.[51] Whereas the first published paper on self-administered behavioral treatment for insomnia appeared 30 years ago,[52] it is only relatively recently that such approaches have become a focus of systematic research investigation.

A meta-analysis of 10 studies enrolling a total of 1,000 participants concludes that minimal contact (e.g., telephone support, brief appointments) or entirely self-directed (e.g., books/ booklets, audiovisual, internet) CBT yields small to medium ES.[53] Thus, there appears to be significant population benefit, but the effects are weaker than in individual or group treatment. Consistent with this conclusion are data from formal comparisons of self-administered vs. "face to face" CBT, where the latter has generally shown superior outcomes, although self-help was also beneficial.[54-56] Internet-delivered CBT is relatively novel. Three randomized trials have been reported to date[57,58,61] and results so far are encouraging, particularly given the potential of the web to reach extremely large populations

Therefore, taking all the evidence on CBT into account, it seems reasonable to conclude that self-administered CBT offers the least restrictive entry based treatment that has satisfactory outcome data, with nurse-delivered small group CBT providing the second level (Figure 4).

What Might Constitute the CBT Stepped Care Hierarchy?

The domains in Figure 3 should assist researchers to consider how resource intensity of CBT might be modelled in healthcare services and in future outcome studies. From the present literature, we know that nurses, graduate psychologists, experienced clinical psychologists, and expert clinicians have all been involved in CBT trials, and that there is an emerging range of delivery methods. Figure 4 is proposed as an exemplar of what evidence-based stepped care might look like, circa 2009. It also parallels contemporary pathways for other common mental disorders.[26,59]

Working on this occasion from top down, at the top of the hierarchy, we might have the most experienced specialists, with the relatively rare combination of CBT and Behavioral Sleep Medicine skills, working with individual complex cases, perhaps on a tertiary referral basis at a sleep centre. At the next level, clinical psychologists might take on individual CBT patients. Indeed, there may be considerable untapped potential for qualified psy-

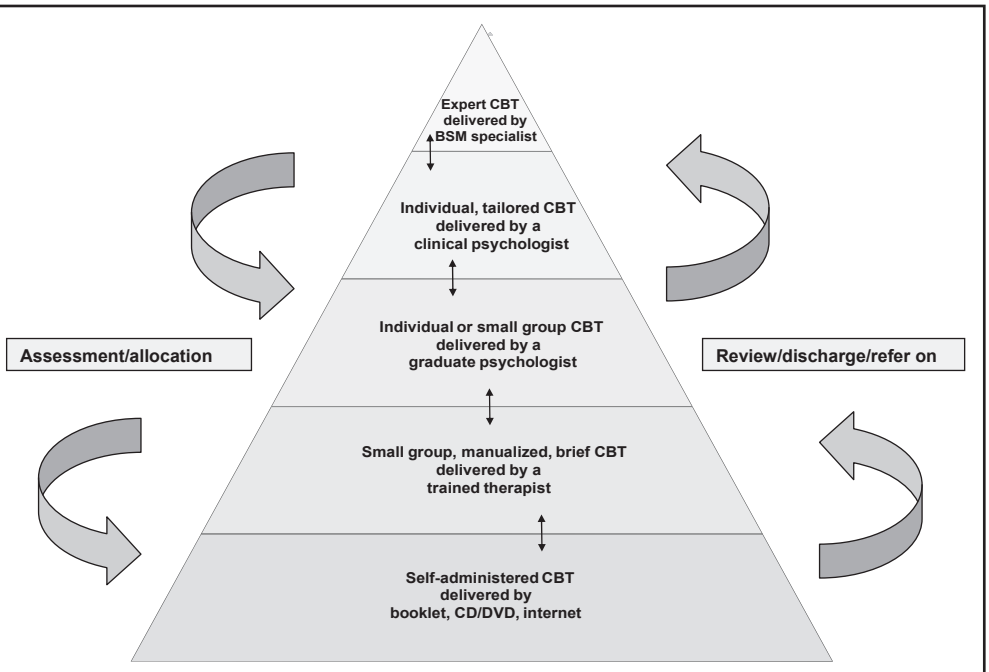


Figure 4—An evidence-based stepped care model for CBT (c. 2009) illustrating how patients might be allocated to resources in relation to assessed need, to achieve optimal service provision. Arrows represent self-correcting referral movements.

chologists who already have extensive generic training in CBT methods to extend their expertise to include insomnia disorders. Below this, a less experienced graduate student might provide individual therapy or a flexible version of group CBT where the manual could be tailored in parts to the presenting needs and problem formulation. A strictly manualized group CBT programme led by a nurse (or other professional) would then sit at the level below, and immediately above the proposed entry level of a self-administered form of CBT. The three lowest tiers might best be provided in primary care, although there is an argument also for the next again (fourth) level also being in primary care. This may depend on the nature of local services.

How would people be allocated and move between levels of the stepped care CBT system?

In the general description of stepped care, presented earlier, the equivalence assumption would suggest that people should be allocated initially to an entry level treatment, providing they are content to accept that, and that there is no specific evidence that they would be unlikely to respond and no contraindication in terms of risk. This approach places emphasis upon skilled assessment, and herein lies a potential problem — of creating a bottle-neck of (more) people waiting to have their needs evaluated, and so waiting (even longer) for treatment.

Therefore, for a stepped care system to work on a large scale it would be necessary for the healthcare system to settle upon a range of common, validated, and simple tools for initial assessment of the insomnia complaint, and of associated complaints. There is a number of such self-report scales available, as reported earlier. Indeed, consensus opinion on these is available. Likewise, a standard post-intervention assessment should be used to determine what, if any, next steps are required, coupled of course with clinical judgement.

In circumstances where a patient is subsequently "stepped up" to a more intensive intervention, particular attention would need to be paid to their expectations (which may be negative if they have already failed to respond), and to their prior knowledge (which on the other hand could be built upon). Crucially, limited or no response may indicate that the previous assessment missed something important (e.g., associated disorders), underestimated the severity of the insomnia and the "level" of CBT required to address it, misjudged the acceptability of a CBT approach, or that the primary therapeutic challenge now is that of motivation and adherence to CBT rather than CBT per se.

All of these factors indicate that iterative assessment and review is the key to the success of the stepped care system (Figure 4) and that the standardisation of these assessment processes would benefit not only consistent clinical practice but also the pooling on a large scale of research data to evaluate benefit.

The Evidence Base for Clinical Governance in CBT

All services offered to patients must be safe, effective and ethical. It is normal practice to have policies and procedures on how referrals are made, how cases are allocated, how and when to review progress, and how to end episodes of care or to commence new episodes. Likewise, it is incumbent on all professional groups to know, and to work within, the boundaries of their training and their clinical competence, and to maintain their skills for (re-)accreditation purposes. In a stepped care system it is particularly important to raise issues of clinical governance because of the relatively fluid nature of the boundaries between the steps.

At the time of writing, from an evidence-based perspective, about all we can say is that clinical leadership in CBT services can be safely and effectively provided by clinical psychology professionals. This conclusion is underpinned by the summary of points provided in Table 2, and by the available data.

In brief, CBT is undeniably a psychological theory and therapy, the published evidence for which has been delivered almost exclusively by psychology-led research.[1-8,53] In this respect, it may be important to clarify that, in Glasgow, our nurse therapists work entirely under the training, direction and supervision of the psychology team. Indeed, this actually demonstrates how a stepped care system can work, because the role of the psychologist is in assessment, triage, case management and review, and clinical service development, and not only in direct patient care. This is a fairly direct parallel with the "medical model"; clinical responsibility in our setting rests with the supervisor of the CBT programme, the clinical psychologist. Table 2 also highlights that clinical psychology professionals are trained

Table 2—Reasons for Suggesting that Clinical Leadership in CBT Should Normally Lie with Psychology

1. CBT is essentially psychological practice
2. CBT is based on psychological theory & principles
3. Psychologists have developed CBT & will continue to develop new CBT interventions
4. Psychologists are trained to use advanced CBT with complex cases
5. Psychologists are qualified to deliver CBT methods that are not yet invented
6. Psychologists have extensive mental health expertise
7. Psychologists are trained behavioural scientists
8. The published literature on CBT is almost exclusively from psychology research groups
9. Psychologists are trained as psychometricians and are trained in systemic working
10. Psychologists are developing competency frameworks for CBT methods generally

mental health professionals, with generic skills in CBT methods (and other psychosocial interventions). Importantly, therefore, they are qualified to administer psychological therapies that are not yet invented, just as physicians are (already) licensed to prescribe new medicines that come on the market. Psychologists are also the people most likely to develop, instrument and operationalize novel psychological interventions because they are doctorally qualified behavioral scientist-practitioners.

None of this is to say that other professionals do not have generic psychological competencies or specific CBT competencies (and certainly they could develop them), but the only evidence that we have currently available is that CBT is effective in contexts where it is practised by psychologists or under close psychology supervision. It is noteworthy here that there are ongoing efforts being made to develop generic competency frameworks for different levels of CBT practice, that would be open to any practitioner.[59,60]

In consequence of these matters relating to clinical governance, it seems appropriate to assert that professional leadership in behavioral aspects of sleep is essential to safe, effective, and comprehensive clinical practice in sleep medicine. It is recommended, therefore:

1. that the appointment of a suitably qualified and experienced clinical psychologist with behavioral sleep medicine credentials should become an essential criterion for the accreditation of sleep centres;
2. that the roles of this lead person should comprise a) advanced clinical practice, b) staff training and clinical supervision, and c) service development;
3. that these roles are not restricted to insomnia, but extend to other sleep disorders where a cognitive-behavioral approach to care is appropriate and is evidence-based (e.g. adherence to treatments, parasomnias)

It has to be acknowledged that there are some tricky interprofessional issues that will have to be faced here; perhaps even intra-professional ones. This has been recognised both within the sleep field,[39] and more generally.[62] It is important to understand that, quite apart from whatever extant, or future, evidence tells us about who can deliver and who can supervise CBT, there will be other sources of influence. Clinical governance arrangements, line management structures, issues relating to pay and reimbursement, may all vary nationally and locally. It is wise to accept that there will be vested professional interests and that some of these will have no genuine evidence base. Consequently, consultation, negotiation, and above all, pragmatism, will be necessary to find a workable way ahead. Further research is certainly needed to inform such debate, particularly in relation to cost-effectiveness for the purposes of manpower planning.

Consequently a further recommendation is:

4. that work should be completed urgently to consider the advantages to patient care of developing an inter-disciplinary approach to behavioral sleep medicine practice. The knowledge-base, the theoretical orientations, and the competencies of the the various professional groups who might be involved should be seen as complementary, rather than competing, bearing in mind that cost-effective use of people's time and of other health care resource is of the utmost importance

Stepped Care and the Health Care System

The stepped care model helps to ensure that the right level of effective care can be provided to the many, not just to the few, and that in so doing, relatively scarce expertise is appropriately targeted. As we have seen, this economy of resources applies not only to direct treatment but also to clinical governance of the service. All of this seems both necessary and timely for CBT.[63]

Of course, each health care system will need to consider how it could implement a stepped care model. The application of stepped care is perhaps easiest to envisage in a publicly funded health care system, like the UK National Health Service, where services are funded entirely through tax revenue (from those eligible to pay income tax) and where ability to pay has no influence over the nature or quality of care received. Nevertheless, principles such as equitability of health care provision are central to most health philosophies. Coupled with the CBT growing evidence base for remission and recovery from sleep disorder, health services, whether publically funded or insurance-based, would do well to consider the population morbidity that is normally associated with persistent insomnia. Stepped care CBT may offer a feasible service model, enabling not only insomnia patients to be treated at the right level, but having sufficient "reach" that associated population health might improve. Indeed, the potential for such "down-stream" benefits may be important, persuasive arguments for encouraging some health care organisations (such as HMOs in the US) to invest in stepped care for insomnia. The development and persistence of insomnia is not good for people, who are then more likely to go on to develop (other) costly conditions that potentially reduce the profits of the health care industry. The early and effective treatment of insomnia may improve health and save money. Moreover, there is likely to be some cost saving associated with treating at the assessed level of need within stepped care, because the majority of patients may not need to be seen beyond the primary care level of intervention.

Conclusions and Future Directions

There is compelling evidence that CBT is effective in the treatment of chronic insomnia. Moreover it appears to be popu-

lar, safe and lastingly beneficial. In short, it has an excellent product profile, but it cannot be bottled. Consequently, CBT has made little impact on the numbers of people who might need it, want it, and benefit from it.

In this paper, a stepped care model has been proposed as a health technology solution for delivering CBT as a first line insomnia treatment. The advantages of this approach are a) that it helps to ensure that patients will receive the least complex and most accessible intervention from which they are likely to benefit; b) that the greatest number of people who need treatment may receive it; c) that scarce expertise and expensive resource is available to those in greatest need; and c) that an insomnia treatment pathway can ensure seamless transition from one level of care to another, through active clinical collaboration (e.g. amongst professionals, between primary and secondary care).

At the time of writing, self-administered CBT has accumulated sufficient evidence to suggest that it can provide the least restrictive minimal intervention for the stepped care platform. Where face to face therapy is concerned, small group manualized CBT offers the potential for trained health care staff to become critical to a new CBT workforce. The clinical leadership role of psychologists is clearly best evidenced, and crucially the stepped care model would see such expertise being spread across assessment, training, supervision and service management roles, as well as direct clinical care of complex cases at an advanced level.

A hierarchy comprising five levels of CBT stepped care has been proposed. It has to be acknowledged that this represents a blend of evidence and pragmatism. In particular, the second and third levels have some similarities. These levels are differentiated by the generic nature of the healthcare workforce (in primary care) who potentially might be recruited to deliver absolutely standardized CBT for insomnia as per manual (second level); versus the graduate psychologist (third level), who are less available and more highly trained (in psychology) and so better equipped to provide basic tailoring of standard CBT, thus raising the introduction of one-on-one therapy.

In looking ahead it will be important also to consider whether a distinction needs to be made between self-help (however effective) and publically funded/ reimbursed healthcare. What an individual chooses to do privately by way of self-help and self-improvement ultimately may, or may not, breach the threshold of organized healthcare delivery through professionally staffed services. Either way, the stepped care approach appears to be sufficiently flexible as a clinical triage system and as a resource allocation model to deliver appropriate care to people at their point of need. What ultimately will drive health care professionals to participate in a stepped care approach will be the interplay between a) much greater public health recognition of the medical, psychological and socio-economic imperatives relating to insomnia; b) the potential benefits to the patient group of taking a population based approach (access and effectiveness); and c) the professional advantages of participation (role clarification and reward).

Finally, it is clear that there is a considerable research agenda. This has to focus more on "phase four," clinical effectiveness studies, capable of evaluating the insomnia health care system. This means not merely a simple allocation to group model, with analysis of comparative outcomes against "usual care," but recognition that the assessment and review process is iterative. People will move (appropriately) between levels, so we will need to know the sensitivity/ specificity of the original, and subsequent, decisions about placement within the hierarchy, in terms of treatment response and recovery. Moreover, mediator and moderator variables (e.g. patient characteristics, treatment preference, format, setting, therapist factors) that we usually try to control for, or at best try to evaluate post hoc, need to become the primary focus of research endeavour if we are to know what best works for whom. Above all, if we were to reach the dizzy heights, that the availability of CBT were no longer an issue, the single and combined use of CBT and PCT would need to be evaluated pragmatically, in real world settings, in respect of both short and long-term outcomes at each level of the stepped care model.

ACKNOWLEDGMENTS

Research supported by Chief Scientist Office, Scottish Government Health Department, The Wellcome Trust, National Health Service Research & Development, Cancer Research UK, Dr Mortimer and Theresa Sackler Foundation.

This paper was first presented as an Invited Lecture at the 22nd Annual Meeting of the Associated Professional Sleep Societies, Baltimore, June 2008.

DISCLOSURE STATEMENT

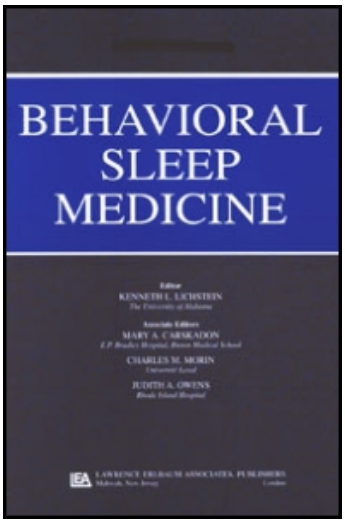
This was not an industry supported study. Dr. Espie has participated in speaking engagements for Takeda, Lundbeck, and GlaxoSmithKline and is on the advisory board of Sanofi-Aventis, GlaxoSmithKline, Lundbeck, and Actelion.

REFERENCES

1. Morin CM, Culbert JP, Schwartz SM. Nonpharmacological Interventions for Insomnia - A Metaanalysis of Treatment Efficacy. American Journal of Psychiatry 1994;151:1172-80.
2. Murtagh DRR, Greenwood KM. Identifying Effective Psychological Treatments for Insomnia - A Metaanalysis. Journal of Consulting and Clinical Psychology 1995;63:79-89.
3. Pallesen S, Nordhus IH, Kvale G. Nonpharmacological interventions for insomnia in older adults: A meta-analysis of treatment efficacy. Psychotherapy 1998;35:472-82.
4. Morin CM, Hauri PJ, Espie CA, Spielman AJ, Buysse DJ, Bootzin RR. Nonpharmacologic treatment of chronic insomnia. Sleep 1999;22:1134-56.
5. Smith MT, Perlis ML, Park A, Smith MS, Pennington J, Giles DE et al. Comparative meta-analysis of pharmacotherapy and behavior therapy for persistent insomnia. American Journal of Psychiatry 2002;159:5-11.
6. Montgomery P, Dennis J. Cognitive behavioural interventions for sleep problems in adults aged 60+. Cochrane Database Syst Rev 2003;CD003161.
7. Irwin MR, Cole JC, Nicassio PM. Comparative meta-analysis of behavioral interventions for insomnia and their efficacy in middle-aged adults and in older adults 55+years of age. Health Psychology 2006;25:3-14.
8. Morin CM, Bootzin RR, Buysse DJ, Edinger JD, Espie CA, Lichstein KL. Psychological and behavioral treatment of insomnia: Update of the recent evidence (1998-2004). Sleep 2006;29:1398-414.
9. Riemann D, Perlis ML. The treatments of chronic insomnia: A review of benzodiazepine receptor agonists and psychological and behavioral therapies. Sleep Medicine Reviews 2009;13:205-214.
10. Chesson AL, Anderson WM, Littner M, Davila D, Hartse K, Johnson S et al. Practice parameters for the nonpharmacologic treatment of chronic insomnia. Sleep 1999;22:1128-33.
11. Morgenthaler T, Kramer M, Alessi C, Friedman L, Boehlecke B, Brown T et al. Practice parameters for the psychological and behavioral treatment of insomnia: An update. An American Academy of Sleep Medicine Report. Sleep 2006;29:1415-19.
12. Sackett DL. Rules of Evidence and Clinical Recommendations. Canadian Journal of Cardiology 1993;9:487-89.

13. Eddy D. A manual for assessing health practices and designing practice policies: the explicit approach. Philadelphia: American College of Physicians; 1992.
14. NIH State-of-the-Science Conference Statement on Manifestations and Management of Chronic Insomnia in Adults. NIH Consens Sci Statements. 2005;Jun 13-15:22(2) 1-30.
15. Morin CM, Colecchi C, Stone J, Sood R, Brink D. Behavioral and pharmacological therapies for late-life insomnia - A randomized controlled trial. Jama-Journal of the American Medical Association 1999;281:991-99.
16. Sivertsen B, Omvik S, Pallesen S, Bjorvatn B, Havik OE, Kvale G et al. Cognitive behavioral therapy vs zopiclone for treatment of chronic primary insomnia in older adults - A randomized controlled trial. Journal of the American Medical Association 2006;295:2851-58.
17. Clarke GN. Improving the Transition from Basic Efficacy Research to Effectiveness Studies - Methodological Issues and Procedures. Journal of Consulting and Clinical Psychology 1995;63:718-25.
18. Manber R, Edinger JD, Gress JL, Pedro-Salcedo MGS, Kuo TF, Kalista T. Cognitive behavioral therapy for insomnia enhances depression outcome in patients with comorbid major depressive disorder and insomnia. Sleep 2008;31:489-95.
19. Espie CA, Fleming L, Cassidy J, Samuel L, Taylor LM, White CA et al. Randomized controlled clinical effectiveness trial of cognitive behavior therapy compared with treatment as usual for persistent insomnia in patients with cancer. Journal of Clinical Oncology 2008;26:4651-58.
20. Savard J, Simard S, Ivers H, Morin CM. Randomized study on the efficacy of cognitive-bahavioural therapy for insomnia secondary to breast cancer, part I: Sleep and psychological effects. Journal of Clinical Oncology 2005;23:6083-96.
21. Espie CA, Inglis SJ, Tessier S, Harvey L. The clinical effectiveness of cognitive behaviour therapy for chronic insomnia: implementation and evaluation of a sleep clinic in general medical practice. Behaviour Research and Therapy 2001;39:45-60.
22. Espie CA, MacMahon KMA, Kelly HL, Broomfield NM, Douglas NJ, Engleman HM et al. Randomized clinical effectiveness trial of nurse-administered small-group cognitive behavior therapy for persistent insomnia in general practice. Sleep 2007;30:574-84.
23. Espie CA, Inglis SJ, Harvey L. Predicting clinically significant response to cognitive behavior therapy for chronic insomnia in general medical practice: Analyses of outcome data at 12 months posttreatment. Journal of Consulting and Clinical Psychology 2001;69:58-66.
24. Morgan K, Dixon S, Mathers N, Thompson J, Tomeny M. Psychological treatment for insomnia in the management of long-term hypnotic drug use: a pragmatic randomised controlled trial. British Journal of General Practice 2003;53:923-28.
25. Bower P, Gilbody S. Stepped care in psychological therapies: access, effectiveness and efficiency - Narrative literature review. British Journal of Psychiatry 2005;186:11-17.
26. Department of Health. Commissioning a Brighter Future: Improving access to Psychological Therapies. Positive Practice Guide. Department of Health; 2007.
27. Morin CM. Insomnia: Psychological Assessment and Management. New York: The Guildford Press; 1993.
28. Bastien CH, Vallieres A, Morin CM. Validation of the Insomnia Severity Index as an outcome measure for insomnia research. Sleep Medicine 2001;2:297-307.
29. Von Korff M, Glasgow RE, Sharpe M. ABC of psychological medicine - Organising care for chronic illness. British Medical Journal 2002;325:92-94.
30. Katon W, Von Korff M, Lin E, Simon G, Walker E, Unutzer J et al. Stepped collaborative care for primary care patients with persistent symptoms of depression - A randomized trial. Archives of General Psychiatry 1999;56:1109-15.
31. Ekers D, Richards D, Gilbody S. A meta-analysis of randomized trials of behavioural treatment of depression. Psychological Medicine 2008;38:611-23.
32. Gellatly J, Bower P, Hennessy S, Richards D, Gilbody S, Lovell K. What makes self-help interventions effective in the management of depressive symptoms? Meta-analysis and meta-regression. Psychological Medicine 2007;37:1217-28.
33. Cuijpers P, van Straten A, Warmerdam L. Behavioral activation treatments of depression: A meta-analysis. Clinical Psychology Review 2007;27:318-26.
34. Ohayon MM. Epidemiology of insomnia: what we know and what we still need to learn. Sleep Medicine Reviews 2002;6:97-111.
35. Lichstein KL Durrence HH RB, Taylor D, Bush A. The Epidemiology of Sleep: Age, gender, and ethnicity. Mahwah, NJ: Lawrence Erlbaum Associates; 2004.
36. Morin CM, Espie CA. Insomnia: A clinical guide to assessment and treatment. New York: Kluwer Academic/Plenum publishers; 2003.
37. Riemann D, Voderholzer U. Primary insomnia: a risk factor to develop depression? Journal of Affective Disorders 2003;76:255-59.
38. Cole MG, Dendukuri N. Risk factors for depression among elderly community subjects: A systematic review and meta-analysis. American Journal of Psychiatry 2003;160:1147-56.
39. Perlis ML, Smith MS. How can we make CBT-I and other BSM services widely available? Journal of Clinical Sleep Medicine 2008;4:11-13.
40. Morin CM, Belanger L, LeBlanc M, Ivers H, Savard J, Espie CA et al. The natural history of insomnia: a population-based 3-year longitudinal study. Arch Intern Med 2009;169:447-53.
41. Howard KI, Kopta SM, Krause MS, Orlinsky DE. The Dose-Effect Relationship in Psychotherapy. American Psychologist 1986;41:159-64.
42. Edinger JD, Wohlgemuth WK, Radtke RA, Coffman CJ, Carney CE. Dose-response effects of cognitive-behavioral insomnia therapy: A randomized clinical trial. Sleep 2007;30:203-12.
43. Morin CM, Gaulier B, Barry T, Kowatch RA. Patients Acceptance of Psychological and Pharmacological Therapies for Insomnia. Sleep 1992;15:302-5.
44. Espie, C. A. and Barrie, L. Does 'acceptance' have a role to play in managing Idiopathic Insomnia? A group comparison of beliefs, coping style and treatment acceptance in idiopathic insomnia and psychophysiological insomnia. Sleep 2009;32:Abstract Supplement, A278.
45. Riedel BW, Lichstein KL. Strategies for evaluating adherence to sleep restriction treatment for insomnia. Behaviour Research and Therapy 2001;39:201-12.
46. Vincent NK, Hameed H. Relation between adherence and outcome in the group treatment of insomnia. Behav Sleep Med 2003;1:125-39.
47. Stinson K, Tang NKY, Harvey AG. Barriers to treatment seeking in primary insomnia in the United Kingdom: A cross-sectional perspective. Sleep 2006;29:1643-46.
48. Morin CM, LeBlanc M, Daley M, Gregoire JP, Merette C. Epidemiology of insomnia: Prevalence, self-help treatments, consultations, and determinants of help-seeking behaviors. Sleep Medicine 2006;7:123-30.
49. National Institute for Clinical Excellence (NICE). Guidance on the use of zaleplon, zolpidem and zopiclone for short-term management of insomnia. London: National Institute for Clinical Excellence (NICE) 2004;77: 27p.
50. Morgan K, Dixon S, Mathers N, Thompson J, Tomeny M. Psychological treatment for insomnia in the regulation of long-term hypnotic drug use. Health Technology Assessment 2004;8:1-68.
51. Currie S. Self-help therapies for insomnia. In: Watkins P, Clum G, eds. Handbook of self-help therapies. Routledge: Taylor & Francis Group, LLC; 2008: 215-41.
52. Alperson J, Biglan A. Self-administered treatment of sleep onset insomnia and the importance of age. Behavior Therapy 1979;10:347-56.
53. van Straten A, Cuijpers P. Self-help therapy for insomnia: A meta-analysis. Sleep Medicine Reviews 2009;13:61-71.
54. Bastien CH, Morin CM, Ouellet MC, Blais FC, Bouchard S. Cognitive-Behavioral Therapy for Insomnia: Comparison of Individual Therapy, Group Therapy, and Telephone Consultations. Journal of Consulting and Clinical Psychology 2004;72:653-59.
55. Mimeault V, Morin CM. Self-help treatment for insomnia: Bibliotherapy with and without professional guidance. Journal of Consulting and Clinical Psychology 1999;67:511-19.
56. Rybarczyk B, Lopez M, Benson R, Alsten C, Stepanski E. Efficacy of Two Behavioral Treatment Programs for Comorbid Geriatric Insomnia. Psychology and Aging 2002;17:288-98.
57. Strom L, Pettersson R, Andersson G. Internet-Based Treatment for Insomnia: A Controlled Evaluation. Journal of Consulting and Clinical Psychology 2004;72:113-20.
58. Vincent N, Lewycky S. Logging on for better sleep: RCT of the effectiveness of online treatment for insomnia. Sleep 2009;32:807-815.
59. Royal College of Psychiatrists & Royal College of General Practitioners. Psychological therapies in Psychiatry and Primary Care. Royal College of Psychiatrists, London, 2008.
60. Roth, A. D. and Pilling, S. The competencies required to deliver effective cognitive and behavioural therapy for people with depression and with

anxiety disorders. Department of Health , 2007 http://www.dh.gov.uk/en/Publicationsandstatistics/Publications/PublicationsPolicyAndGuidance/DH_078537
61. Ritterband LM, Thorndike FP, Gonder-Frederick LA, Magee JC, Bailey ET, Saylor DK, Morin CM. Efficacy of an internet-based behavioral intervention for adults with insomnia. Archives of General Psychiatry 2009;66:692-698.
62. Turpin G, Hope H, Duffy R, Fossey M, Seward J. Improving access to psychological therapies: implications for the mental health workforce. Journal of Mental Health Workforce Development 2006;12-21.
63. Lamberg L. Despite Effectiveness, Behavioral Therapy for Chronic Insomnia Still Underused. Jama-Journal of the American Medical Association 2008;300:2474-75.
64. Espie CA, MacMahon KMA, Kelly H-L, et al. Manualised CBT for insomnia delivered by nurse practitioners: results from studies conducted in primary care and in oncology. Sleep Med 2005;6: Suppl 2:S29.
65. Edinger JD, Carney C. Behavioral treatment of insomnia. In: Kushida, C, ed. Handbook of Sleep Disorders (2nd edition) Taylor & Francis Inc., New York 2008;71-90.



Behavioral Sleep Medicine
Publication details, including instructions for authors and subscription information:
http://www.informaworld.com/smpp/title~content=t775648093

An Experimental Assessment of a Pennebaker Writing Intervention in Primary Insomnia

Patricia Mooney [a]; Colin A. Espie [b]; Niall M. Broomfield [b]
[a] Elizabeth Martin Clinic, [b] Faculty of Medicine, Southern General Hospital, University of Glasgow Sleep Centre, Sackler Institute of Psychobiological Research, Glasgow, Scotland

Online Publication Date: 01 April 2009

To cite this Article Mooney, Patricia, Espie, Colin A. and Broomfield, Niall M.(2009)'An Experimental Assessment of a Pennebaker Writing Intervention in Primary Insomnia',Behavioral Sleep Medicine,7:2,99 — 105
To link to this Article: DOI: 10.1080/15402000902762386
URL: http://dx.doi.org/10.1080/15402000902762386

Behavioral Sleep Medicine, 7:99–105, 2009

ISSN: 1540-2002 print/1540-2010 online
DOI: 10.1080/15402000902762386

Routledge
Taylor & Francis Group

An Experimental Assessment of a Pennebaker Writing Intervention in Primary Insomnia

Patricia Mooney
Elizabeth Martin Clinic

Colin A. Espie and Niall M. Broomfield
University of Glasgow Sleep Centre, Sackler Institute of Psychobiological Research Faculty of Medicine, Southern General Hospital, Glasgow, Scotland

This study considers the role of pre-sleep cognitive arousal, worry, and inhibition in sleep onset difficulties. The Pennebaker writing task, which promotes emotional processing by asking people to write about their thoughts, worries, and emotions, has proven effective in several areas of health. Here, the paradigm's ability to reduce pre-sleep cognitive arousal (PSCA) and sleep onset latency (SOL) in people with insomnia was tested. Twenty-eight people with insomnia were randomized to three nights of Pennebaker writing or a control condition, following a one-night baseline. The outcomes of change over baseline at Day 4 in pre-sleep cognitive arousal and SOL were compared. Writing significantly reduced pre-sleep cognitive arousal on one out of two measures, but did not significantly reduce SOL.

The central role of cognitive arousal and worry in sleep difficulties is well-established (Espie, 2002). Converging evidence suggests the pre-sleep cognitive activity (PSCA) of people with insomnia is excessive, uncontrollable, negatively toned, and covers a broad range of topics (e.g., Harvey, 2000; Wicklow & Espie, 2000). Furthermore, there is evidence that decreasing intrusive and worrisome thoughts of people with insomnia using experimental techniques (e.g., Harvey & Payne, 2002) can improve sleep onset latency (SOL).

Harvey's (2002) model of the maintenance of primary insomnia (PI) draws on such evidence, together with studies that have shown that poor sleepers inhibit and/or internalize psychological conflicts (Edinger, Stout, & Hoelsscher, 1988; Kales, Caldwell, Soldatos, Bixler, & Kales, 1983). This model states that for poor sleepers, unwanted PSCA at night may be a consequence of inhibition of emotional concerns during the day. Such "unfinished business" not dealt with

Correspondence should be addressed to Patricia Mooney, Elizabeth Martin Clinic, 1 Burns Square, Greenock, Inverclyde, PA16 0NT, Scotland. E-mail: patricia.mooney@renver-pct.scot.nhs.uk

prior to bedtime intrudes during the pre-sleep phase, fuelling PSCA and, in turn, delaying sleep onset.

The Pennebaker writing task (e.g., Pennebaker, 1997) is purported to reverse inhibition of emotional concerns, directing individuals to express their deepest thoughts, worries, and emotions through writing. Meta-analyses (Smyth, 1998; Frisina, Borod, & Lepore, 2004) show beneficial effects on a range of physical and psychological outcomes, including sleep. In a pilot study, Harvey and Farrell (2003) randomly allocated poor sleepers to three nights of either utilizing the Pennebaker task, writing about "hobbies," or to a "no writing" control group. They found that participants who wrote about problems and worries prior to bed using the Pennebaker paradigm experienced shorter SOL compared to those who did not write.

This pilot data is encouraging but requires replication. In particular, as the authors themselves recognize, several points of methodology require examination. First, no baseline condition was employed. The sleep changes observed may, therefore, reflect inter-subject variability and/or changes unrelated to the experimental intervention. Second, it may be possible that the rationale given to the Pennebaker group informing them of the tasks benefits led to expectations of sleep improvement. Finally, no measurement was made of whether sleep effects were actually mediated by reduced PSCA. Clarifying these aspects should help us to better understand and apply the Pennebaker task to insomnia treatments. In addition, this study was carried out with a sample of poor sleepers and, as such, it is not known whether the results generalize to people who meet criteria for insomnia.

The current study was, therefore, designed to replicate and extend Harvey and Farrell's (2003) work while addressing these limitations. Thus, PSCA was measured. Furthermore, given the evidence that patient expectations can influence response to PI (Espie & Lindsay, 1985), counter-demand instructions were given to participants, which indicated that they should not expect any improvements in sleep as a result of their participation. Primary hypotheses were that, following a one-night baseline, people with insomnia randomized to the experimental condition (Pennebaker writing) would show reduced PSCA (Hypothesis 1) and SOL (Hypothesis 2) compared to participants randomized to a control condition.

METHOD

Participants

Potential participants were contacted via the e-mail systems of the University of Glasgow and National Health Service (both in the United Kingdom). Participants were aged 16 to 65 years, complaining of clinically significant difficulties falling asleep (SOL $>$ 30 min; 4+ nights per week for a minimum of 4 weeks, with or without disruption to other sleep variables; American Sleep Disorders Association, 2005), and excessive PSCA. Participants were assessed initially using a locally developed semistructured clinical interview (available from Colin A. Espie), the Insomnia Severity Index (ISI; Morin, 1993), the Pittsburgh Sleep Quality Index (PSQI; Buysse, Reynolds, Monk, Berman, & Kupfer, 1989), and the Beck Depression Inventory–II (BDI-II; Beck, Brown, & Steer, 1996). All participants scored $>$ 5 on the PQSI and $>$ 8 on the ISI, and both recognized clinical cutoff criteria (Buysse et al., 1989; Morin, 1993). Those receiving psychological or pharmacological treatment for sleep difficulties or suffering

comorbid sleep, medical, or psychiatric conditions were excluded. Twenty-eight participants met inclusion criteria (14 per group). This sample provided 80% power to successfully detect group differences at $p < .05$, based on number of participants and the standard deviations of SOL from the experimental and "sleep as usual" group of Harvey and Farrell (2003; $\alpha = 0.05$).

Design and Procedure

Participants were met and given instruction in sleep diary completion. Following blind randomization to experimental condition, participants received four envelopes (one for each study day) containing relevant group instructions and materials. As patient expectations can influence response to intervention, all participants were explicitly told *not* to expect sleep improvement during the experiment. All participants were directed to otherwise carry on with their normal daytime and sleeping practices.

Sleep and PSCA were monitored at baseline (Day and Night 1). For Days and Nights 2 to 4, experimental condition participants were instructed to "spend 20 minutes writing about any thoughts, concerns, or worries in the early evening (between 6 p.m. and 8 p.m.) outwith your bedroom environment" (cf. Harvey & Farrell, 2003). The time was chosen to minimize potential pre-bedtime arousal. Participants could write about the same or different concerns during each writing session and, to encourage honest and free expression, were not required to disclose their writings. Control participants were instructed to "complete two questionnaires about your worries between 6 p.m. and 8 p.m. outwith your bedroom environment." These questionnaires were the Penn State Worry Questionnaire (Meyer, Miller, Metzger, & Borkovec, 1990) and the Worry Domains Questionnaire (Tallis, Eysenk, & Mathews, 1992), respectively. Controls, therefore, spent a similar time focussed on worries as the writing group, but without the facilitation of emotional processing. Adherence, treatment credibility, and emotional processing data were gathered at follow up.

Measures

For both groups, a daily sleep diary completed on rising each day provided subjective SOL data. In addition, wrist actigraphy (Actiwatch Model AW2, Cambridge Neurotechnology Ltd) was used as an objective measure of SOL. The outcomes for this measure are not reported, as the use of actigraphy in insomnia (particularly with respect to SOL) remains somewhat controversial. The PSAS-C (Nicassio, Mendlowitz, Fussell, & Petras, 1985) was completed alongside the sleep diary in the morning with respect to the previous evening. Following Robertson, Broomfield, and Espie (2007), PSCA was also assessed using the following question: "How mentally alert did you feel last night while you were trying to get to sleep?" This 5-point scale ranged from 1 (*not at all*) to 5 (*extremely*). Adherence to study protocol was assessed using the following question: "To what extent did you comply with the experimental instructions given to you during the study?" This 7-point scale ranged from 0 (*not at all*) to 6 (*very much*). The number of nights each participant correctly followed the study protocol was also recorded. In the writing group, extent of worry processing was assessed using the following: "I worked through some upsetting issues while writing," "Writing helped me organize my thoughts," and "My thoughts and opinions have changed regarding the subjects I wrote about." This 4-point scale ranged from 0 (*not at all*) to 3 (*a lot*). Writing participants rated writing content overall

on a scale of −5 (*sad-anxious*) to +5 (*happy*; cf. Harvey & Farrell, 2003) and estimated time spent writing over the three days. Finally, treatment credibility and utility of writing task was examined using questions from the Therapy Evaluation Questionnaire (Borkovec & Nau, 1972).

RESULTS

Participant Characteristics

Overall mean age was 32.89 (range = 18–60; $SD = 13.96$). Ratio of females to males was 18:10. Using one-way analyses of variance (ANOVA) and chi-square (for gender), no significant group differences were found on any variable (all ps $> .10$, *ns*).

Outcome Variables

Quantitative outcome data were examined for kurtosis, skewness, and homogeneity of variance and found suitable for parametric analysis. In this respect, the primary outcome data were considered normally distributed according to the Kolmogorov–Smirnov Z tests of normal distribution: $D(27) = 0.680$, $p = .744$, *ns*; $D(27) = 1.019$, $p = .250$, *ns*; $D(28) = 0.876$, $p = .426$, *ns*, respectively. Normal Q-Q plots supported these analyses. Outcomes of change over baseline at Day 4 were employed to account for the variability in participants' scores at baseline. Writing task and control group values (PSAS–C, alertness ratings, SOL) were then compared using one-way ANOVAs, the results of which are outlined in Table 1.

The writing group did not show significantly greater reduction in PSCA relative to controls according to the PSAS-C, $F(1, 25) = 0.963$, $p = .336$, *ns*. However, the writing group showed a significantly greater decrease in PSCA according to alertness ratings relative to controls, $F(1, 25) = 6.766$, $p = .015$; effect size = 0.21. Finally, the writing group did not show significantly greater reduction in SOL relative to controls, $F(1, 26) = 1.494$, $p = .233$, *ns*.

TABLE 1
Mean Scores and Standard Deviations for Outcome Variables at Baseline, Day 4, and Change Over Baseline

	Baseline		*Day 4*		*Change Scores*	
Measure	*Writing*	*Control*	*Writing*	*Control*	*Writing*	*Control*
PSAS–C	21.92 (6.59)	19.86 (6.22)	18.31 (6.03)	18.57 (6.26)	−3.62 (6.06)	−1.29 (6.26)
Alertness	3.15 (1.14)	2.86 (0.86)	2.38 (0.87)	3.29 (0.99)	−0.77 (1.09)	0.43 (1.28)
SOL	52.86 (35.88)	84.29 (57.74)	51.07 (41.43)	64.79 (52.81)	−1.79 (21.63)	−19.50 (49.73)

Note. A negative score indicates an improvement in variables. Standard deviations are shown in parentheses. PSAS–C = Pre-Sleep Arousal Scale; SOL = sleep onset latency.

Writing

Nine of 12 writers found writing to be "at least some benefit" to work through issues, 11 indicated writing "helped them organize their thoughts to some degree," and 7 reported that "their thoughts had changed on the topic they wrote about at least to some extent" (data available from Patricia Mooney). Writers reported a mean rating of −3.58 ($SD = 1.98$) on content of writing. Thirteen of 14 complied with the recommended writing time. The same number would use the task again. Mean confidence in using the task in the future and recommending the task to a friend with insomnia were 5.00 ($SD = 1.41$) and 5.17 ($SD = 1.40$), respectively.

Adherence

On a one-way ANOVA, no significant differences were found between groups on extent of adherence, $F(1, 26) = 1.00$, $p = .327$, *ns*; number of nights adhered, $F(1, 26) = 1.00$, $p = .327$, *ns*; or how acceptable or sensible tasks appeared, $F(1, 26) = 1.00$, $p = .327$, *ns*. However, as the outcome results indicate, one participant failed to fully complete PSCA data.

DISCUSSION

The present experiment tested whether a Pennebaker writing task would reduce PSCA and SOL in people with insomnia. In support of Hypothesis 1, Pennebaker writing significantly decreased *self-rated* cognitive arousal among poor sleepers relative to a non-writing control task. The size of this effect was, however, small; and this arousal reduction effect was not found according to the PSAS-C. Contrary to Hypothesis 2, there was no significant reduction on SOL following Pennebaker writing. Despite the lack of sleep effects observed, almost all writers stated they would use this task again, and the majority would recommend it to a friend with insomnia. Subjective ratings regarding extent of emotional processing following Pennebaker writing were more modest. Compliance rating data suggested a high degree of adherence to experimental instructions, with no significant group differences, and writers also reported writing about negative or worrisome topics.

The lack of significant sleep effects is disappointing. Harvey and Farrell (2003) did observe SOL reduction among poor-sleeping students instructed in Pennebaker writing following only three nights use. However, interestingly, a second writing group instructed to write about hobbies rather than problems and concerns also responded showing similar SOL change to the Pennebaker group. A reasonable explanation may be that the rationale given to writers may have promoted expectation of sleep benefit in the form of a demand characteristic and, therefore, magnified any effect of the task. Conversely, it could be argued that the effects of the counter-demand characteristics used here may have "washed out" what could be relatively gentle effects of this task.

Although we closely followed the original protocol used by Harvey and Farrell (2003), and others in the wider Pennebaker literature (e.g., Rosenberg et al., 2002), perhaps the duration of our experiment was too short. Gillis, Lumley, Mosley-Williams, Leisen, & Roehrs (2006) examined the effects of written emotional disclosure in fibromyalgia patients and found that sleep benefits did not emerge until later on in treatment. Further examination of this paradigm over a longer period is required.

More puzzling is the observation of self-rated cognitive arousal reduction following writing in the absence of significant decreases on the PSAS-C. Three possible explanations are offered for this finding. It is possible that the measures used here may tap into different arousal components. The alertness rating employed is a measure of general cognitive arousal. However, PSAS-C correlates significantly with anxiety and depression (Nicassio et al., 1985) and may be more specifically related to negatively toned cognitive activity. Perhaps then, emotional processing purported to accompany Pennebaker writing may decrease general alertness among people with insomnia, without impacting on negatively toned cognitions. This seems unlikely, however, as there is evidence that difficulties characterized by worrisome thoughts (e.g., physical health worries; Rosenberg et al., 2002) respond best to Pennebaker writing. As no attempt was made to select participants whose PSCA was specifically negatively toned, the PSAS-C ratings obtained in the current study were not as high as expected. Perhaps a more plausible account is that the participants in the present study did not experience sufficiently high levels of negatively toned PSCA to find this task of significant benefit. Although the data were not available in the current study, comparing people with insomnia who display high and low levels of negatively toned PSCA will be an important next step in examining this paradigm. Finally, as pre-sleep worry was assessed using a retrospective judgment made in the morning on wakening, there is the possibility that the delay in reporting this may have interfered in some way with the rating made on the PSAS-C. Future research should consider using "online" assessment of worry during the pre-sleep period, for example, through the use of a voice-activated tape recorder.

Other points of relevance require brief discussion. Outcome measures were all subjectively reported (specifically, SOL). In addition, adherence was only assessed by self-report. Therefore, it is possible that participants did not fully utilize the task. Also, on looking at Table 1, there is a difference between group SOL ratings at baseline and high variability within groups. This may have lessened the scope for the writing group to improve or reduce the ability to detect effects if present. However, alternative analysis of the data taking the average for each participant across the three nights intervention phase was carried out with the same results.

In summary, as hypothesized, among people with insomnia, a Pennebaker writing task significantly reduced self-rated mental alertness according to one measure, although PSAS–C scores and SOL remained unchanged. The present study therefore failed to support previous results regarding sleep change. The brief study length, along with the counter-demand instructions, may account for this. Further research will be needed to better clarify the potential efficacy of this paradigm in people with insomnia.

REFERENCES

American Sleep Disorders Association. (2005). *The International Classification of Sleep Disorders* (2nd ed.). Rochester, MN: Author.

Beck, A. T., Brown, G., & Steer, R. A. (1996). *Beck Depression Inventory-II manual.* San Antonio, TX: Psychological Corporation.

Borkovec, T., & Nau, S. D. (1972). Credibility of analogue therapy rationales. *Journal of Behaviour Therapy and Experimental Psychiatry, 3,* 247–260.

Buysse, D. J., Reynolds, C. F., Monk, T. H., Berman, S. R., & Kupfer, D. J. (1989). The Pittsburgh Sleep Quality Index: A new instrument for psychiatric practice and research. *Psychiatry Research, 28,* 193–213.

Edinger, J. D., Stout A. L., & Hoelsscher, T. J. (1988). Cluster analysis of insomniac's MMPI profiles: Relation of subtypes to sleep history and treatment outcome. *Psychosomatic Medicine, 50,* 77–87.

Espie, C. A. (2002). Insomnia: Conceptual issues in the development, persistence and treatment of sleep disorders in adults. *Annual Review of Psychology, 53,* 215–243.

Espie, C. A., & Lindsay, W. R. (1985). Paradoxical intention in the treatment of chronic insomnia: Six cases illustrating variability in therapeutic response. *Behavioural Psychotherapy, 23,* 703–709.

Frisina, P. G., Borod, J. C., & Lepore, S. J. (2004). A meta-analysis of the effects of written emotional disclosure on the health outcomes of clinical populations. *Journal of Nervous and Mental Disease, 192,* 629–634.

Gillis, M. E., Lumley, M. A., Mosley-Williams, A., Leisen, J. C. C., & Roehrs, T. (2006). The health effects of at-home written emotional disclosure in fibromyalgia: A randomized trial. *Annals of Behavioral Medicine, 3,* 135–146.

Harvey, A. G. (2000). Pre-sleep cognitive activity in insomnia: A comparison of sleep onset insomniacs and good sleepers. *British Journal of Clinical Psychology, 38,* 401–405.

Harvey, A. G. (2002). A cognitive model of insomnia. *Behaviour Research and Therapy, 40,* 869–893.

Harvey, A. G., & Farrell, C. (2003). The efficacy of a Pennebaker-like writing intervention for poor sleepers. *Behavioral Sleep Medicine, 1,* 115–124.

Harvey, A. G., & Payne, S. (2002). The management of unwanted pre-sleep thoughts in insomnia: Distraction with imagery versus general distraction. *Behaviour Research and Therapy, 40,* 267–277.

Kales, A., Caldwell, A. B., Soldatos, C. R., Bixler, E. O., & Kales, J. D. (1983). Biopsychobehavioural correlates of insomnia II: Pattern specificity and consistency with the MMPI. *Psychosomatic Medicine, 45,* 341–356.

Meyer, T. J., Miller, M. L., Metzger, R. L., & Borkovec, T. D. (1990). Development and validation of the Penn State Worry Questionnaire. *Behaviour Research and Therapy, 28,* 486–496.

Morin, C. M. (1993). *Insomnia: Psychological assessment and management.* New York: Guilford.

Nicassio, P. M., Mendlowitz, D. R., Fussell, J. J., & Petras, L. (1985). The phenomenology of the pre-sleep state: The development of the pre-sleep arousal scale. *Behaviour Research and Therapy, 23,* 263–271.

Pennebaker, J. W. (1997). Writing about emotional experiences as a therapeutic process. *Psychological Science, 8,* 162–166.

Robertson, J., Broomfield, N. M., & Espie, C. A. (2007). Prospective comparison of subjective arousal during the pre-sleep period in primary sleep-onset insomnia and normal sleepers. *Journal of Sleep Research, 16,* 1–8.

Rosenberg, H. J., Rosenberg, S. D., Ernstoff, M. S., Wolford, G. L., Amdur, R. J., Elshamy, M. R., et al. (2002). Expressive disclosure and health outcomes in a prostate cancer population. *International Journal of Psychiatry in Medicine, 32,* 37–53.

Smyth, J. M. (1998). Written emotional expression: Effect sizes, outcome types and moderating variables. *Journal of Consulting and Clinical Psychology, 66,* 174–184.

Tallis, F., Eysenk, M., & Mathews, A. (1992). A questionnaire for the measurement of nonpathological worry. *Personality and Individual Differences, 13,* 161–168.

Wicklow, A., & Espie, C. A. (2000). Intrusive thoughts and their relationship to actigraphic measurement of sleep: Towards a cognitive model of insomnia. *Behaviour Research and Therapy, 38,* 619–694.

Journal of Psychopharmacology
http://jop.sagepub.com/

British Association for Psychopharmacology consensus statement on evidence-based treatment of insomnia, parasomnias and circadian rhythm disorders

SJ Wilson, DJ Nutt, C Alford, SV Argyropoulos, DS Baldwin, AN Bateson, TC Britton, C Crowe, D-J Dijk, CA Espie, P Gringras, G Hajak, C Idzikowski, AD Krystal, JR Nash, H Selsick, AL Sharpley and AG Wade

J Psychopharmacol 2010 24: 1577 originally published online 2 September 2010
DOI: 10.1177/0269881110379307

The online version of this article can be found at:
http://jop.sagepub.com/content/24/11/1577

Published by:
SAGE
http://www.sagepublications.com

On behalf of:
British Association for Psychopharmacology

Original Paper

Journal of Psychopharmacology
24(11) 1577–1600

DOI: 10.1177/0269881110379307
jop.sagepub.com

British Association for Psychopharmacology consensus statement on evidence-based treatment of insomnia, parasomnias and circadian rhythm disorders

SJ Wilson[1], DJ Nutt[2], C Alford[3], SV Argyropoulos[4], DS Baldwin[5], AN Bateson[6], TC Britton[7], C Crowe[8], D-J Dijk[9], CA Espie[10], P Gringras[11], G Hajak[12], C Idzikowski[13], AD Krystal[14], JR Nash[15], H Selsick[16], AL Sharpley[17] and AG Wade[18]

Abstract

Sleep disorders are common in the general population and even more so in clinical practice, yet are relatively poorly understood by doctors and other health care practitioners. These British Association for Psychopharmacology guidelines are designed to address this problem by providing an accessible up-to-date and evidence-based outline of the major issues, especially those relating to reliable diagnosis and appropriate treatment. A consensus meeting was held in London in May 2009. Those invited to attend included BAP members, representative clinicians with a strong interest in sleep disorders and recognized experts and advocates in the field, including a representative from mainland Europe and the USA. Presenters were asked to provide a review of the literature and identification of the standard of evidence in their area, with an emphasis on meta-analyses, systematic reviews and randomized controlled trials where available, plus updates on current clinical practice. Each presentation was followed by discussion, aimed to reach consensus where the evidence and/or clinical experience was considered adequate or otherwise to flag the area as a direction for future research. A draft of the proceedings was then circulated to all participants for comment. Key subsequent publications were added by the writer and speakers at draft stage. All comments were incorporated as far as possible in the final document, which represents the views of all participants although the authors take final responsibility for the document.

Keywords

Sleep, insomnia, parasomnia, circadian rhythm disorder, consensus, treatment

Introduction

Sleep disorders are common in the general population and even more so in clinical practice, yet are relatively poorly understood by doctors and other health care practitioners. These British Association for Psychopharmacology (BAP) guidelines are designed to address this problem by providing an accessible yet up-to-date and evidence-based outline of the major issues, especially those relating to reliable diagnosis and appropriate treatment. We limited ourselves to discussion of sleep problems that are not regarded as being secondary to respiratory problems (e.g. sleep apnoea – see NICE Guidance TA139), as these fall outside the remit of the BAP. We also did not consider certain neuropsychiatric disorders for which recent sets of guidelines already exist, such as narcolepsy (Billiard et al., 2006) and restless legs (Vignatelli et al., 2006) and also refer interested readers to the British Sleep Society website http://www.sleeping.org.uk. Thus the main scope of this document is to cover insomnia, circadian rhythm disorders and the more common parasomnias which are likely to present to psychiatrists or primary care physicians.

[1]Psychopharmacology Unit, University of Bristol, UK.
[2]Edmond Safra Chair in Neuropsychopharmacology, Imperial College London, UK.
[3]Applied Psychology, University of the West of England, Bristol, UK.
[4]Institute of Psychiatry, King's College, London, UK.
[5]Clinical Neuroscience Division, University of Southampton UK.
[6]Institute of Membrane and Systems Biology, Faculty of Biological Sciences, University of Leeds UK.
[7]King's College Hospital, London, UK.
[8]Mater Private Hospital, Dublin, Eire.
[9]Surrey Sleep Research Centre, University of Surrey, UK.
[10]University of Glasgow Sleep Centre, Sackler Institute of Psychological Research, University of Glasgow, UK.
[11]King's College London, and Paediatric Sleep Disorder Unit, Evelina Children's Hospital, London, UK.
[12]Sleep Disorder and Research Centre, University of Regensburg, Germany.
[13]Edinburgh Sleep Centre, SAAS, Edinburgh, UK.
[14]Duke Sleep Disorders Laboratory, Duke University School of Medicine, Durham, NC, USA.
[15]Betsi Cadwaladr University Health Board, Mold, Flintshire, UK.
[16]University College London Hospitals, UK.
[17]University Department of Psychiatry, Warneford Hospital, Oxford, UK.
[18]CPS Clinical Research Centre, Glasgow, Scotland, UK.

Corresponding author:
Sue Wilson, Psychopharmacology Unit, Dorothy Hodgkin Building, Whitson st, Bristol, BS31 1TD, UK
Email: Sue.Wilson@bristol.ac.uk

The BAP is an association of psychiatrists, psychopharmacologists and preclinical scientists who are interested in the broad field of drugs and the brain. BAP is the largest national organization of its kind worldwide, and publishes the Journal of Psychopharmacology. The Association started publishing consensus statements more than a decade ago, and the first BAP guidelines on depression were considered a landmark publication when they appeared in 1993 (Montgomery, 1993). That document, updated in 2000 and in 2008 (Anderson et al., 2000; Anderson et al., 2008), has become the standard of care in many countries as it is considered an accessible consensus to guide practising psychiatrists. Additional guidelines have covered management of bipolar disorder (Goodwin, 2003; Goodwin, 2009) drug treatments for addiction (Lingford-Hughes et al., 2004), anxiety disorders (Baldwin et al., 2005), old age psychopharmacology (Burns et al., 2006), and attention-deficit hyperactivity disorder (ADHD) (Nutt et al., 2007) all of which use a similar style and process. All guidelines are available via the BAP website (http://www.bap.org.uk).

Method

A consensus meeting was held in London on 21–22 May 2009. Those invited to attend included BAP members, representative clinicians with a strong interest in sleep disorders and recognized experts and advocates in the field, including a representative from mainland Europe and the USA. The main age groups and clinical subtypes were specifically covered by individual speakers. Presenters were asked to provide a review of the literature and identification of the standard of evidence in their area, with an emphasis on meta-analyses, systematic reviews and randomized controlled trials (RCTs) where available. Each presentation was followed by discussion, aimed to reach consensus where the evidence and/or clinical experience was considered adequate, or otherwise to flag the area as a direction for future research. A draft which pulled together the presentations and the transcript of the taped proceedings was drawn up by SJW and DJN and circulated to all speakers and other participants for comment. Key subsequent publications were added by the writer and speakers at draft stage. All comments were incorporated as far as possible in the final document, which represents the views of all participants although the authors take final responsibility for the document.

Categories of evidence for causal relationships, observational relationships and strength of recommendations are given in Table 1 and are taken from Shekelle et al., 1999. The strength of recommendation reflects not only the quality of the evidence, but also the importance of the area under study. For example, it is possible to have methodologically sound (category I) evidence about an area of practice that is clinically irrelevant, or has such a small effect that it is of little practical importance and therefore attracts a lower strength of recommendation. However, more commonly, it has been necessary to extrapolate from the available evidence leading to weaker levels of recommendation (B, C or D) based upon category I evidence statements.

The costs of the meeting were partly defrayed by unrestricted educational grants from two pharmaceutical companies (Lundbeck and GSK). Observers from these companies were invited to attend but did not participate in the summary proceedings or in drafting the guidelines. All attendees completed conflict of interest statements that are held at the BAP office according to BAP policy.

Scope of the guidelines

Our intention is to present a comprehensive statement to guide clinicians, who are managing patients in primary or secondary medical care.

Definition of insomnia

Insomnia is a common disorder whose definition is often not clearly understood. A number of international organizations with interests in sleep disorders have proposed varying definitions of insomnia that share three key elements (see Diagnostic criteria). They all agree insomnia is a condition of unsatisfactory sleep, either in terms of sleep onset, sleep maintenance or early waking. They also agree that insomnia is a disorder that impairs daytime well-being and subjective abilities and functioning, and so can be considered a '24-hour' disorder.

It is important to recognize that insomnia is a subjective disorder, and its diagnosis is through clinical observations rather than via measurements; in this sense, it is a syndrome similar to pain. The cause of insomnia may be known or not, and knowledge of causation is not necessary for a diagnosis. However, in some cases it may be possible to identify and remedy a physical cause for insomnia (see treatment section).

Insomnia often starts with a specific problem, for example a stressful life event such as the loss of a job or change to a more demanding one, or through something that changes sleep patterns such as the birth of a child or starting shift work. In some people this acute insomnia persists into a chronic state. Factors involved in the persistence of insomnia are not fully established, but include anxiety about sleep, maladaptive sleep habits and the possibility of an underlying vulnerability in sleep-regulating mechanisms. Persistence of the precipitating stressor can also contribute. Some cases of insomnia are precipitated by, or are co-morbid with, other psychiatric disorders, especially anxiety and depression, or by physical illness such as cancer or arthritis.

The nature of sleep changes with age. Older age is associated with poorer objectively measured sleep with shorter sleep time, diminished sleep efficiency, and more arousals, and these changes may be more marked in men than in women, according to a very large study of elderly people living at home in the USA (Sleep Heart Health Study, Unruh et al., 2008). In the same study the association of subjective report of poor sleep with older age was stronger in women. The higher prevalence of chronic health conditions, including sleep apnoea, in older adults did not explain changes of sleep parameters with aging and age/sex differences in these relationships.

There is some disagreement about how long insomnia should have been present for before it requires intervention (see treatment section), but there is general agreement that when insomnia causes significant personal distress or marked impairment then some form of treatment is appropriate.

Definition of insomnia: Diagnostic criteria

	A	B	C
International Classification of Sleep Disorders (ICSD) and Research Diagnostic Criteria for Insomnia (RDC) (Edinger et al., 2004)	Difficulty – initiating sleep, – maintaining sleep, – waking up too early or – sleep is chronically non-restorative or poor in quality	Occurs despite adequate opportunity and circumstances for sleep	At least one form of daytime impairment i. Fatigue or malaise ii. Attention, concentration, or memory impairment iii. Social or vocational dysfunction or poor school performance iv. Mood disturbance or irritability v. Daytime sleepiness vi. Motivation, energy, or initiative reduction vii. Proneness for errors or accidents at work or while driving viii. Tension, headaches, or gastrointestinal symptoms in response to sleep loss ix. Concerns or worries about sleep
International Classification of Diseases ICD-10 (1992)	Difficulty – falling asleep, – maintaining sleep or – non-refreshing sleep	3 times a week and for longer than 1 month	Marked personal distress or interference with personal functioning in daily living
Diagnostic and Statistical Manual of Mental Disorders DSM-IV	Predominant complaint – difficulty initiating sleep – difficulty maintaining sleep or – non-restorative sleep	For at least 1 month	Clinically significant distress or impairment in social, occupational, or other important areas of functioning

Table 1. Levels of Evidence

Categories of evidence for causal relationships and treatment	
Ia:	evidence from meta-analysis of randomized controlled trials
Ib:	evidence from at least one randomized controlled trial
IIa:	evidence from at least one controlled study without randomization
IIb:	evidence from at least one other type of quasi-experimental study
III:	evidence from non-experimental descriptive studies, such as comparative studies, correlation studies and case-control studies
IV:	evidence from expert committee reports or opinions and/or clinical experience of respected authorities

This categorization is most appropriate to questions of causal relationships. Similar taxonomies for other types of research question do not yet exist and the following is proposed.

Proposed categories of evidence for non-causal relationships	
I:	evidence from large representative population samples
II:	evidence from small, well-designed, but not necessarily representative samples
III:	evidence from non-representative surveys, case reports
IV:	evidence from expert committee reports or opinions and/or clinical experience of respected authorities

Strength of recommendation

Recommendations are graded A to D as shown below. We distinguish between the category of evidence and the strength of the associated recommendation. It is possible to have methodologically sound (category I) evidence about an area of practice that is clinically irrelevant or has such a small effect that it is of little practical importance and therefore attracts a lower strength of recommendation. More commonly, a statement of evidence only covers one part of an area in which a recommendation has to be made, or covers it in a way that conflicts with other evidence. Therefore, to produce comprehensive recommendations it is necessary to extrapolate from the available evidence. This may lead to weaker levels of recommendation (B, C or D) based upon category I evidence statements.

Strength of Recommendation	
A	directly based on category I evidence
B	directly based on category II evidence or extrapolated recommendation from category I evidence
C	directly based on category III evidence or extrapolated recommendation from category I or II evidence
D	directly based on category IV evidence or extrapolated recommendation from category I, II or III evidence

Epidemiology of insomnia

What is known about prevalence of insomnia

- Estimates of prevalence of insomnia vary according to the definition used (Ia)
- Prevalence of symptoms varies with age, with increase of nocturnal awakenings but decrease in complaints of non-restorative sleep as people get older (Ib)
- Prevalence is between 1.5 and 2 times higher in women than in men (Ia)
- Insomnia is a long-term disorder; many people have had insomnia for more than 2 years (Ib)
- Approximately half of all diagnosed insomnia is related to a psychiatric disorder (Ib)

What is not known

- What is the prevalence of distress?
- What is the significance of duration of symptoms on distress?

Studies of prevalence of insomnia in the general population indicate that one-third of adults in Western countries experience difficulty with sleep initiation or maintenance at least once a week (LeBlanc et al., 2009; Léger and Poursain, 2005; Sateia et al., 2000), and 6–15% are thought to meet criteria of insomnia in that they report sleep disturbance as well as significant daytime dysfunction (LeBlanc et al., 2009; Sivertsen et al., 2009). One-year incidence rates have been reported to be 30.7% for insomnia symptoms and 7.4% for insomnia syndrome. These rates decreased to 28.8% and 3.9% for those without a prior lifetime episode of insomnia (LeBlanc et al., 2009). There is much evidence that insomnia is a long-term disorder. In one large UK study, about three-quarters of patients reported symptoms lasting at least a year (Morphy et al., 2007) and in a population-based 3-year longitudinal study 46% of subjects who had insomnia at baseline still had it at the 3-year time point. The course of insomnia was more likely to be persistent in those with more severe insomnia at baseline, and in women and older adults (Morin et al., 2009a).

There is a higher incidence of insomnia in women, and the incidence increases in men and women as they get older. The symptom prevalence changes with age, so that people over 65 show more sleep maintenance problems but a decrease in reported daytime problems compared with younger age groups, with little change in prevalence of sleep-onset insomnia.

Diagnosis

Insomnia

Insomnia is a subjective complaint. Patients complain that sleep is inadequate, either by being too short (such as after a long period of trying to get to sleep, or due to early waking), too interrupted or not sufficiently restorative or refreshing. In many patients there is a combination of these factors. As a consequence of the disrupted sleep daytime function is impaired.

There are a number of ways in which sleep can be assessed. The most simple is by asking the patient (and family member or carer if possible) about their sleep, and a sleep diary (see Appendix). This allows the assessment of sleep difficulties over time and gauges the potential contribution of poor sleep and lifestyle habits to daytime impairment. Preliminary questions for eliminating other sleep disorders as primary diagnosis are summarized below.

Eliminating other sleep disorder as primary: preliminary questions – see Appendix for more detailed follow-up questions.

- Are you a very heavy snorer? Does your partner say that you sometimes stop breathing at night? (obstructive sleep apnoea syndrome (OSAS))
- Do your legs often twitch and can't keep still in bed? Do you wake from sleep with jerky leg movements? (restless legs syndrome (RLS), periodic limb movements in sleep (PMLS))
- Do you sometimes fall asleep in the daytime completely without warning? Do you have collapses or extreme muscle weakness triggered by emotion, for instance when you're laughing? (narcolepsy)
- Do you tend to sleep well but just at the 'wrong times'; and are these sleeping and waking times regular? (circadian rhythm sleep disorder; evidence also from sleep diary)
- Do you have unusual behaviours associated with your sleep that trouble you or that are dangerous? (parasomnias – see text)

It is important to determine if another sleep disorder (see preliminary questions above), or a physical (such as pain, heart or lung disease), neurological (such as Parkinson's disease or cerebrovascular disease) or psychiatric disorder (such as depressive illness, anxiety disorder, or substance misuse) is the primary diagnosis. In depression, however, in many cases insomnia should be regarded as a co-morbid condition, rather than as a secondary one. The majority of epidemiological evidence suggests that insomnia typically predates other psychiatric symptoms and may represent an independent risk factor for the development of depression in particular (see below).

Circadian rhythm disorder

Circadian rhythm disorders are sleep disorders where there is a mismatch between circadian rhythms and required sleep–wake cycle. Thus there can be sleeplessness when trying to sleep at a time not signalled by the internal clock, and excessive sleepiness when needing to be awake.

Some circadian disorders (jetlag and shift-work disorder) are due to an individual lifestyle, including work and travel schedules, that conflicts with the internal clock. Others are:

- delayed sleep-phase syndrome (DSPS), where there is difficulty falling asleep before 2–3 a.m. (sometimes later), and on days without work/school/college the preferred wake time is after 10 a.m., resulting in sleep-onset insomnia

and difficulty waking up in the morning on days when an early bedtime for an early start time is necessary.
- free-running sleep disorder, where there is a daily increment of sleep and wake times (getting later each day). This is often associated with insomnia of varying severity and daytime sleepiness.

Parasomnias

Parasomnias are unusual episodes or behaviours occurring during sleep which disturb the patient or others; here we addresses those that cause significant distress and therefore present for treatment. Assessment of parasomnia may be possible with a detailed history from the patient or a witness but, in general, for an adequate diagnosis referral to a specialist sleep centre for polysomnography (PSG) and video recording may be necessary. Violent or unusual night-time attacks may arise from deep non-REM sleep (night terrors and sleepwalking) or from REM sleep (severe recurrent nightmares, REM behaviour disorder), and treatments depend on which disorder is present.

Night terrors (also called sleep terrors) are recurrent episodes of abrupt awakening from deep non-REM sleep, usually in first third of the night, usually with a scream and signs of intense fear and autonomic arousal. The patient is unresponsive to comforting; they may sit up in bed and sometimes engage in automatic behaviour associated with fear and escape. There is usually no detailed recall, and if the patient wakes from a terror (not common), there is confusion and disorientation and only a vague memory of fear. Night terrors are common in children, with about 30–40% having at least one episode, and repeated episodes in about 5%. The peak age for these is at about 2–7 years, with a gradual diminution up to early adolescence (DiMario and Emery, 1987). In some cases night terrors persist into adult life; the prevalence in adults is unknown. Almost all adult patients have had night terrors or sleepwalking as a child (Crisp, 1996). There is a strong genetic component (Nguyen et al., 2008), and night terrors and sleepwalking in the same patient is fairly common.

Sleepwalking alone probably has 15–20% lifetime prevalence. The main symptom is of automatic behaviour at night with the sufferer unresponsive to surroundings and other people. The behaviour is most commonly walking around, but can include other behaviours which are highly familiar to the subject such as dressing, washing, making tea, arranging objects in the house, etc. Some cases of sleepwalking seem related to use of certain drugs, for example alcohol and hypnotics, especially zolpidem and triazolam (Pressman, 2007). It is rare for affected individuals to present for treatment, except if they have injured themselves or a partner, have put themselves into potential danger, or have excessive daytime fatigue because of nighttime disturbance. Another reason for presentation is anxiety and disruption of sleep of partner, family or housemates.

Nightmares and REM sleep behaviour disorder (RBD) are disorders arising from REM sleep, and the main difference in presentation from the non-REM episodes is that they are normally recalled by the patient, who wakes from them and is aware of the episode and can describe it. RBD is a disorder, first described in the late 1980s, with violent complex behaviour at night, which is mostly recalled by the patient. There are two sleep abnormalities; lack of atonia during REM sleep, and increased vividness and/or unpleasant content of dreams. The violent behaviour is described as 'acting out of dreams', made possible by the lack of the normal muscle paralysis in REM sleep. Its incidence is unknown (probably <1%), it occurs in older people with a steady rise after 55 years of age, and has a marked male preponderance. It may be idiopathic but much more often is associated with Parkinson's disease (it is seen in up to 50% of patients with Parkinson's disease), Lewy body dementia (~70%), and multiple system atrophy (>90%). RBD may be the first manifestation of these disorders, antedating the onset of parkinsonism, cerebellar syndrome, dysautonomia, and dementia by several years (Gagnon et al., 2006).

Figure 1 summarizes the diagnosis algorithm for sleep problems.

Recommendations

- The diagnosis of insomnia is primarily based on patient-derived and family or caregiver complaints, as determined by the clinical interview, ideally with patient diary (A).
- In some circumstances referral to a specialist sleep centre may be necessary for other investigations, for instance:
  - Differential diagnosis of circadian rhythm disorder (actigraphy) (A)
  - Other primary sleep disorder suspected including parasomnia (polysomnography) (A)
  - In the case of treatment failure (D)

Costs and consequences of insomnia

What is known about detrimental effects of insomnia

- Quality of life is impaired in insomnia (I)
- There is an increased risk of subsequent first episode depression, and of relapse into depression, in those with a pre-existing persistent insomnia (I)
- Primary insomnia is associated with poor objective sleep and impaired objectively measured daytime performance (II)
- There is an increased risk of hypertension in insomnia with objectively measured short sleep duration (II)
- Absenteeism, accidents at work and road accidents are increased in insomnia (II)

What is not known

- What are the potential confounding effects of medication and comorbid disorders in reports of increased accidents?
- To what extent do treatments rectify the health risks of insomnia?

Several large studies have demonstrated reduced quality of life, increased functional impairment and increased healthcare costs in insomnia (Chevalier et al., 1999; Léger et al., 2001; Philip et al., 2006; Simon and VonKorff, 1997; Zammit et al., 1999). Impairments in the areas of vitality, energy, emotional and mental health domains have been the most widely reported. One study shows that severe insomnia is independently associated with worsened health-related quality of life to almost the

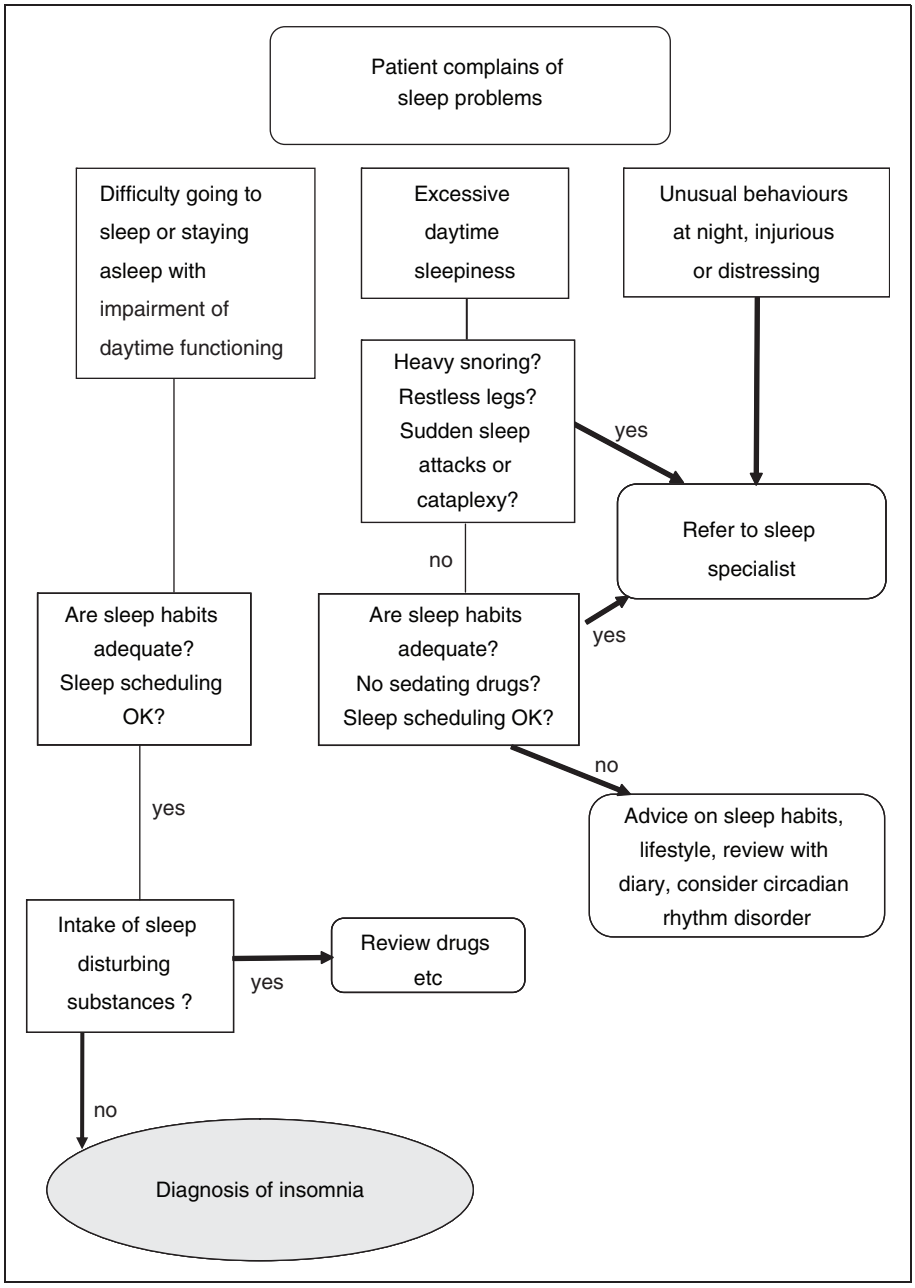


Figure 1. Diagnosis algorithm.

same extent as chronic conditions such as congestive heart failure and major depression (Katz and McHorney, 2002). Studies suggest that the resulting economic burden of insomnia is very high, with the largest proportion of all expenses (76%) attributable to insomnia-related work absences and reduced productivity (Daley et al., 2009). The incidence of road accidents is increased in individuals with insomnia (Léger and Bayon, 2010), but the potential confounding effects of medication and co-morbid medical disorders have not been studied extensively.

People with a diagnosis of insomnia have subjective complaints of poor daytime function. When compared with matched controls, they show increased **subjective** sleepiness but decreased **objective** sleepiness, due to the fact that they are usually overaroused, but feel subjectively tired. Objectively, they show poorer performance on psychomotor tasks, particularly those requiring switching of attention (e.g. frontal/executive tasks) (Edinger et al., 2008), objectively measured time awake after sleep onset (WASO) was the best predictor of impaired daytime performance. Likewise, Altena et al. (2008) have reported that people with insomnia perform more poorly on complex cognitive tasks, an effect which normalizes following cognitive behavioural therapy (CBT) intervention.

There is an increased risk of subsequent depression and anxiety disorder in primary insomnia. Insomnia has been associated with: (1) an increased risk of developing subsequent depression; (2) an increased duration of established depression; and (3)

relapse following treatment for depression (Riemann, 2009). On the other hand, sleep disturbances are widely understood as core symptoms of major depressive disorder rather than associated or co-morbid disorders (Mendlewicz, 2009). Poor sleep quality seems to correlate with high negative and low positive emotions, both in clinical and subclinical samples. Good sleep seems to be associated with high positive emotions, but not necessarily with low negative emotions (Baglioni et al., 2010).

The National Institute of Mental Health Epidemiologic Catchment Area, which interviewed 7954 adults on two occasions a year apart, first highlighted the strong association between sleep disturbance and subsequent depression. It was found that 14% of those with insomnia at the first interview had developed new major depression 1 year later (Ford and Kamerow, 1989). This increased risk of developing depression has been confirmed in other investigations: in a survey of 1200 young adults in Michigan the odds ratio of new depression was four times greater in those subjects who had insomnia 3 years earlier (Breslau et al., 1996), and of new anxiety disorder the risk was twofold greater. In a questionnaire survey of adults in the UK there was a threefold increased risk of new depression and a twofold risk of new anxiety disorder if subjects had reported one sleep problem occurring 'on most nights' a year earlier (Morphy et al., 2007). In a much longer study in Norway with two surveys 10 years apart (Neckelmann et al., 2007), the risk of having an anxiety disorder diagnosis at the second time point increased by about one and a half times if insomnia had been present at the first time point, and by about five times if insomnia was present at both time points, indicating the higher risk of long-standing insomnia. Doctors in a prospective study who had complained of insomnia during medical school in the 1950s and 1960s were twice as likely to have developed depression at follow-up in the 1990s (Chang et al., 1997).

Insomnia is associated with activation of the hypothalamic–pituitary–adrenal (HPA) axis with increased adrenocorticotrophin and cortisol in most studies (Varkevisser et al., 2005; Vgontzas et al., 1998; Vgontzas et al., 2001). When the complaint of insomnia is accompanied by short duration of sleep measured objectively, there is a three to fivefold increased overall risk of hypertension, which is comparable to that seen with other common sleep disorders, such as sleep-disordered breathing (Vgontzas et al., 2009).

Recommendation

- It is important to treat insomnia because the condition causes decreased quality of life, is associated with impaired functioning in many areas, and leads to increased risk of depression, anxiety and possibly cardiovascular disorders (A).
- Goal of treatment:
  - to less suffering and
  - improve daytime function
- Type of treatment:
  - Patient-guided
  - By particular pattern of problem, i.e. sleep onset insomnia, maintenance
  - By choice of treatments with an evidence base

Psychological treatment of insomnia

What is known about CBT for insomnia - CBTi

- CBT is an effective treatment for insomnia delivered either individually or in small group format (Ia)
- CBT has been found to be as effective as prescription medications for short-term treatment of chronic insomnia. Moreover, there are indications that the beneficial effects of CBT may last well beyond the termination of active treatment (Ia)

What is not known

- Are long-term effects of a short-term course of hypnotics better or worse than after CBT?
- Long-term effects of CBT versus optimized (e.g. intermittent) use of hypnotics in the long term

Psychological treatment of insomnia should be considered appropriate for two reasons. First, insomnia is a 'psychophysiological' disorder, in which mental and behavioural factors play predisposing, precipitating and perpetuating roles. Essential features of insomnia are heightened arousal and learned sleep-preventing associations. Arousal can reflect a general cognitive hypervigilance and many patients describe 'racing thoughts' as a problem when they are trying to sleep. A cycle develops in which the more one strives to sleep, the more agitated one becomes, and the less able one is to fall asleep. CBT for insomnia (CBTi) employs a package of interventions designed to encourage poor sleepers to think and behave like good sleepers. The therapy is manualized, and health professionals can be trained to administer it either individually or in a group setting. Therapies are multimodal, embodying techniques such as sleep restriction and stimulus control as well as cognitive restructuring. CBT then is a treatment modality, just as is sleep pharmacotherapy. The latter comprises a range of licensed medications, and the former a range of proven psychotherapeutic methods.

There have been many investigations of CBT in insomnia but it is challenging to design a randomized controlled trial as the therapy cannot be blinded, and contact with professionals is difficult to match with the comparator group. However, in 85 clinical trials involving a total of 4194 participants (including 12 trials in insomnia associated with medical/psychiatric disorders) 70% of patients who completed the course achieved sustained improvement on sleep and daytime reports, reflecting moderate-to-large effect sizes over waiting list (Irwin et al., 2006; Morin et al., 2006). Based on this and other extensive published evidence, including nine systematic reviews or meta-analyses, the National Institutes of Health Consensus and State of the Science Statement (NIH, 2005) concluded that a CBT package containing cognitive and behavioural methods is ''as effective as prescription medications are for short-term treatment of chronic insomnia. Moreover, there are indications that the beneficial effects of CBT, in contrast to those produced by medications, may last well beyond the termination of active treatment''.

In the majority of studies, CBT employs a package of the previously mentioned techniques which are designed to

encourage poor sleepers to think and behave like good sleepers. The therapy is usually performed from a manual, and health professionals can be trained to administer it either individually or in a group setting. Therapy is multimodal, embodying techniques such as sleep restriction and stimulus control as well as cognitive restructuring. Sleep restriction and stimulus control do not prolong sleep time but result in a shortening of total sleep time during the acute treatment period, because patients reduce the amount of time spent in bed by delaying bedtime or leaving the bedroom when they wake during the sleep period. This means that improvements in sleep continuity and quality parameters, rather than total sleep time, have generally been the significant outcome measures in these studies.

There have been several comparative studies of CBT versus pharmacotherapy. A recent meta-analysis (Riemann and Perlis, 2009) concludes that during the treatment period they produce comparable improvements; that psychological therapy produces significant beneficial long-term effects; and notes that studies of the long-term effects of short-term pharmacotherapy have not been reported. A recent randomized study of combined therapy (Morin et al., 2009b), in which two groups of patients underwent a 6-week CBT intervention, with one group also taking zolpidem nightly during acute treatment, found an approximately 60% response rate in both groups. After the acute phase, patients in the zolpidem group were re-randomized to extended CBT plus or minus intermittent zolpidem; combined therapy produced a higher remission rate compared with CBT alone during the 6-month extended phase and the 6-month follow-up period. In patients with persistent insomnia, the addition of medication to CBT produces added benefits during acute therapy, but long-term outcome was optimized when medication is discontinued during maintenance CBT.

Outside of the research environment, for example in clinical practice in the UK, the take-up rate for CBT is not certain – for example, in the Bristol insomnia treatment group, which is only available on a weekday during normal working hours, and involves considerable travel for many patients, only half of the patients referred from a secondary care sleep clinic for chronic insomnias agreed to attend and some dropped out before the end of the course; making treatment more accessible in terms of flexibility of times and locations an urgent goal. Provision of psychological treatments for insomnia in the UK is an issue, as there are few trained therapists and insomnia is not a priority for psychologists in the National Health Service. One approach involving 'stepped care' has been suggested (Espie, 2009) where, depending on severity, chronicity and complexity of insomnia, people could be allocated to the various levels, with self-administered CBT (e.g. a book) as the 'entry level', manualized, small-group CBT delivered by nurses as the next level, and involvement of more specialized professionals thereafter. This would enable this relatively scarce resource to be applied in a cost-effective way to achieve best clinical care.

Recommendation

- CBT-based treatment packages for chronic insomnia including sleep restriction and stimulus control are effective and therefore should be offered to patients as a first-line treatment (A).
- Increased availability of this therapy is required.

Drug treatments for insomnia

What is known about drug treatments for insomnia

- Z-drugs and short-acting benzodiazepines are efficacious for insomnia (Ia)
- Safety (adverse events and carryover effects) are fewer and less serious with decreasing half-lives (Ib)
- Prolonged release melatonin improves sleep onset latency and quality in patients over 55 (Ib)

What is not known

- Does improvement in insomnia last after treatment is stopped?
- Does treatment reduce risk of subsequent depression?

Underpinning principles – pharmacology

An overview of the way in which various drugs are thought to work, classified according to what is thought to be their primary site of action on sleep, is given in Table 2.

The sleep–wake function reflects a complex balance between arousing and sleep-inducing physiological systems. Current research suggests that arousal and wakefulness are promoted by parallel neurotransmitter systems whose cell bodies are located in brainstem or midbrain centres, with projections to the thalamus and forebrain. These activating neurotransmitters are noradrenaline, serotonin, acetylcholine, dopamine and histamine. In addition the newly discovered orexin system with cell bodies in the hypothalamus promotes wakefulness through regulating arousal 'pathways' (and inhibiting sedative ones) (Samuels and Szabadi, 2008; Saper et al., 2005). For all these arousal neurotransmitters sleep can be promoted by blocking their post-synaptic actions, leading to reduced arousal. For example, many over-the-counter (OTC) sleep-promoting agents contain antihistamines, which block the histamine H1 receptor and so decrease arousal. The relatively low efficacy of these compounds may be explained by the fact that they target only one of the parallel arousal systems. The same is true for any drug which blocks one of the other arousal systems; they produce a degree of sedation but are not generally effective hypnotics. However, some agents have specific actions on certain sleep parameters; for instance, drugs which block 5HT2 receptors (such as ritanserin or eplivanserin) can increase slow-wave sleep (Idzikowski et al., 1988; Landolt et al., 1999) whereas the alpha-1 adrenergic blocker prazosin is useful in post-traumatic stress disorder-related nightmares (Raskind et al., 2007). Trazodone is commonly used to promote sleep and has blocking actions at noradrenaline, 5HT and histamine receptors; this multiple action probably explains why it is widely used, although there are few controlled clinical trials. Other drugs such as sedating antidepressants and antipsychotics probably promote sleep in a similar fashion.

The promotion of sleep is regulated by a number of other neurotransmitters (see Table 2); primary amongst these is gamma-aminobutyric acid (GABA), the major inhibitory neurotransmitter in the brain. The majority of brain cells are inhibited by GABA, so increasing its function reduces arousal and

Table 2. Neurotransmitters and sleep in humans

Endogenous transmitter	Maintains wakefulness	Promotes sleep	Agents promoting wakefulness	Agents promoting sleep	Agents causing sedation
GABA		✓	antagonists (though not studied in humans)	agonists, positive allosteric modulators e.g. benzodiazepines	agonists, positive allosteric modulators
melatonin		✓		M1 and M2 agonists	
adenosine		✓	antagonist (caffeine)		
noradrenaline	✓		uptake blockers releasers (stimulants)		α1 antagonists
dopamine	✓		stimulants (releasers) and uptake blockers	Possibly agonists (paradoxical effect →sudden sleep attacks)	
serotonin	✓		uptake blockers	5HT2 antagonists, 5HTP	
histamine	✓		H3 antagonist	? H1 antagonists	H1 antagonists
acetylcholine	✓				Muscarinic antagonists
orexin	✓			OR1 and/or 2 antagonists	

produces sleep, and eventually anaesthesia. There are many subsets of GABA neurones distributed throughout the brain but a particular cluster in the hypothalamus (ventrolateral preoptic nucleus) can be considered to be the sleep 'switch' (Saper et al., 2005). These neurones switch off brain arousal systems at the level of the cell bodies and therefore promote sleep. GABA receptors in the cortex can also promote sedation and sleep by inhibiting the target neurones of the arousal system.

The inhibitory effects of GABA are mediated through the $GABA_A$ receptor, which is a complex of proteins with binding sites for a number of sleep-promoting drugs, in particular benzodiazepines, so-called Z-drugs and barbiturates, all of which enhance the effects of GABA's actions at the $GABA_A$ receptor. There are a number of subtypes of this receptor which are relevant for sleep, not only because of their different location in the brain but also because of the fact that some hypnotic drugs are selective for a particular subtype. The alpha-1 subtype is highly expressed in the cortex and probably mediates the sedative and hypnotic effects of many drugs that act at the benzodiazepine site; zolpidem and zaleplon target this subtype preferentially (Sanna et al., 2002). The alpha-3 subtype predominates in the reticular nucleus of the thalamus, which plays an important role in regulating sleep. This subtype is particularly targeted by eszopiclone (Jia et al., 2009). Traditional benzodiazepine hypnotics act on four subtypes – alpha 1, 2, 3 and 5 – which may explain some differences between them and the Z-drugs.

The other main sleep-promoting neurotransmitter is adenosine. Brain levels of this rise during the day and are thought to lead to sleepiness, which increases the longer the time since the last sleep. The arousing and sleep-impairing effects of caffeine (Landolt et al., 2004) are thought to be due to blockade of adenosine-A2 receptors, so attenuating this natural process (Porkka-Heiskanen et al., 2002). Caffeine is a useful translational model for insomnia as its effects in rodents are very similar to those in humans and could be used to screen potential new treatments (Paterson et al., 2007).

Melatonin is a natural hormone that is produced in the pineal gland and which has an important role in regulating circadian rhythms (Cajochen et al., 2003; Dijk and von Schantz, 2005). The circadian pacemaker in the suprachiasmatic nucleus (SCN) of the hypothalamus drives melatonin synthesis and secretion from the pineal gland. Once melatonin appears in the plasma it enters the brain and binds to melatonin receptors in the hypothalamus, forming a feedback loop. The SCN contains melatonin 1 and melatonin 2 receptors, and much research is ongoing about their role in sleep/wake regulation and circadian rhythms. Melatonin has both phase-shifting effects (changing the timing of the biological clock), and direct sleep-facilitating effects. Administering exogenous melatonin or analogues such as ramelteon (licensed in the USA) can promote sleep onset. A slow-release formulation of melatonin has been licensed on the basis of improved sleep continuity and daytime well-being in people aged over 55 years with insomnia. Melatonin production is reported to decline with age and to be lower in middle-aged and elderly patients with insomnia than in good sleepers (Attenburrow et al., 1996; Dowling et al., 2008; Haimov, 2001; Léger et al., 2004).

Underpinning principles – pharmacokinetics

The principles of the ideal hypnotic have been discussed for decades and are outlined in Figure 2. All licensed hypnotics improve one or more aspects of subjective sleep and some also improve daytime functioning (see below – but note this treatment outcome has only been seen as being important in recent years, so many drugs have not been evaluated in this parameter).

Kinetic aspects are important both in terms of how quickly the drug enters the brain and how long its effects last (see Tables 3 and 4). The faster the hypnotic enters the brain, the sooner sleep is induced. Some agents used as hypnotics have not been active in this aspect of sleep because of poor kinetic properties: for example, temazepam tablets have a poorer bioavailability and slower absorption (and thus a longer presence in the body) than the previous gel formulations. Drugs that enter the brain very quickly, though effective, may need to be taken in the bedroom or even in bed to prevent people falling asleep before they are in bed (see zolpidem

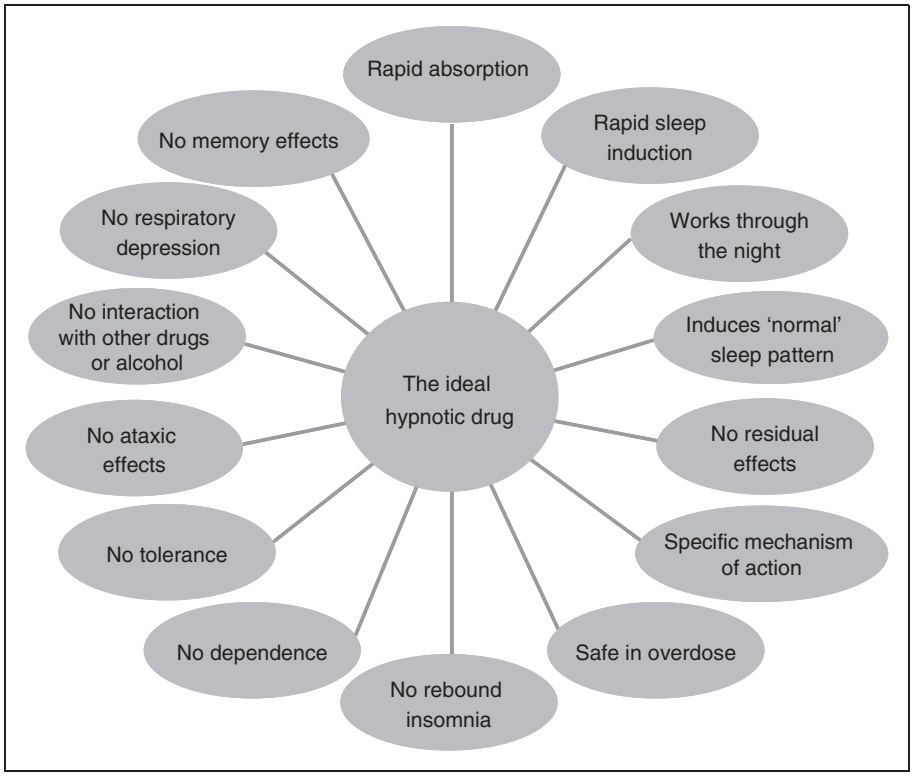


Figure 2. The ideal hypnotic drug.

Table 3. Pharmacokinetic data for benzodiazepine receptor-acting hypnotics

	Availability (%)	Plasma bound (%)	Time to T_{max} (h)	T_{half} (h)
Nitrazepam	78[a]	87[a] (85[b])	1.6[b], 1–5[l], 0.5–0.83[x]	26[a], 24[b], 20–40[l], 28–35[o], 15–38[p], 25–35[x]
Flurazepam		96[a]	0.5–1.5[l], 0.5–1[v], 0.5–1[x]	* 76[a], 40–100[l], 40–103[o], 47–100(2.3)[v], 47–100[x]
Loprazolam	90[q]	85[q]	2[q], 2[r], 0.5[x]	7–8[n], 15[o], 6–12[p], 12[q], 12[r], 4.6–11.4[x]
Lorametazepam	80[t], 70–80[u]	92[b]	2[b]	10[b], 10[o], 10–12[p], 10[t], 7.9–11.4[x]
Temazepam	91[a]	98[a], 96[b]	1.1[b], 2–3[l], 0.75–1[x]	11[a], 9.1[b], 5–20[l], 12[o], 8–15[p], 2–25[x]
Zaleplon	30[d], 30[i]	60[b]	1.4[b], 1[f], 0.9–1.5[g], 1[h], 0.8[i], 0.25–0.5[x]	1[b], 1[f], 0.9–1.1[g], 1[h], 1[i], 1[x],
Zolpidem	70[d], 70[l], 70[m]	92[b], 90[l], 92[m]	1.7–2[h], 0.75–2.6[m], 0.5[x]	2.0–2.2[h], 1.5–3.2[m], 1.5–4.5[x]
Zopiclone	70[d], 75[j], 80[l]	80[b], 45[j], 45–80[l]	1.5[c], 0.25–0.5[x]	5.6[c], 4–5[j], 5[l], 3.5–6.5[x]
Eszopiclone		52–59[e]	1.5[c], 1[e], 1–1.5[w]	3.8[c], 6[e], 6[k], 6.9–7.3[w]

Data from [a]Benet et al., 1996; [b]Nutt, 2005a, [c]Najib, 2006; [d]Drover, 2004; [e]Melton et al., 2005; [f]Patat et al., 2001; [g]Beer et al., 1994; [h]Greenblatt et al., 1998; [i]Rosen et al., 1999; [j]Gaillot et al., 1982; [k]Fernandez et al., 1993; [l]Chouinard et al., 1999; [m]Salva and Costa, 1995; [n]Clark et al., 1988; [o]Jochemsen and Breimer, 1986; [p]Ashton, 1994; [q]www.fda.gov/medwatch/safety/2006/Nov_PIs/Ativan_PI.pdf; [r]Greenblatt et al., 1976; [t]Humpel et al., 1982; [u]De Vanna et al., 2007; [v]www.fda.gov/cder/foi/label/2001/16721s74lbl.pdf; [w]Brunello et al., 2008; [x]Wagner and Wagner, 2000.
*Metabolized to desalkylflurazepam (Chouinard et al., 1999).

Table 4. Pharmacokinetic data for other hypnotics

	Availability (%)	Plasma bound (%)	Time to T_{max} (h)	T_{half} (h)
Chloral hydrate (NB $t^1/_2$ is so short, values are for the primary active metabolite trichloroethanol)	NK	35[p], 35[q]	0.76–0.98[o], 2[p], 8.2[q]	9.3–10.2[o], 9.3–10.9[p]
Triclofos sodium	NK	35[q]	8.2[q]	NK
Clomethiazole	25–42[s]	63[s]	0.92[r]	3.6–5[s]
Promethazine hydrochloride	12.3–40[t], 25[u]	NK	4.39[u]	18.6[u]
Sodium oxybate	25[y]	<1[y]	0.6–0.9[w], 0.5–2[y]	0.57–0.73[w], 0.5–1[y]
Trazodone	75[a], 60–80[b]	93[a], 89–95[b]	1–2[b], 1–2[c]	6.5[a], 6–13[b], 7–15[c]
Mirtazapine	50[d],	85[e],	0.25–2[c], 1.8[d], 2[e]	20–40[c], 16.3[d], 20–40[e]
Olanzapine	60[g]	93[g]	5[c], 6[g]	30[c], 24[f], 30[g]
Quetiapine		83[h]	1[c], 1.5[h], 2[i], 2[j]	7[c], 6[h], 5.3[i], 5.3[j]
Melatonin	15[l]	71.5[m], 80[n]	0.5–0.88[k], 0.87–1[l]	0.76–0.86[k], 1[l]

Data from [a]Benet et al., 1996; [b]DeVane, 1994; [c]Krystal, 2009; [d]Voortman and Paanakker, 2004; [e]Caraco Mirtazapine PIL, 2/08; [f]Tauscher et al., 2002; [g]zyprexa-pi LillyInfo 2009; [h]Seroquel PIL Astrazeneca; [i]Gefvert et al., 1998; [j]Davis et al., 1999; [k]Markantonis et al., 2008; [l]DeMuro et al., 2000; [m]Rizzo et al., 2002; [n]Di et al., 1998; [o]Zimmermann et al., 1998; [p]Merdink et al., 2008; [q]Sellers et al., 1978; [r]Rätz et al., 1999; [s]Jostell et al., 1978; [t]Koytchev et al., 1994; [u]Strenkoski-Nix et al., 2000; [w]Abanades et al., 2006; [y]http://www.ema.europa.eu/docs/en_GB/document_library/EPAR_-_Product_Information/human/000593/WC500057103.pdf
NK: not known.

summary of product characteristics (SPC)) (see http://www.medicines.org.uk/emc/medicine/22443/SPC/Zolpidem%20Tartrate%2010%20mg%20Tablets/NHSEvidence).

The ease of waking and the propensity to daytime carry-over ('hangover') effects are determined by the duration of action – most typically defined by the elimination half-life of the drugs (see Tables 3 and 4) and the dose taken. Drugs with half-lives of more than 6 h tend to leave sufficient residual drug in the brain to cause hangover in the morning. This was particularly the case with the first benzodiazepine hypnotics such as nitrazepam, which was associated with daytime sedation and falls (Trewin et al., 1992). The rationale for developing the Z-drugs was in part to make shorter half-life drugs with minimal carry-over effects (Nutt, 2005b). This was largely achieved, although there is some hangover seen with zopiclone (Staner et al., 2005). The very short half-life of zaleplon means that it can be taken as little as 5 h before the desired time of arising, without the risk of hangover impairment (see SPC and Walsh et al., 2000).

A very short half-life limits a drug's duration of action on sleep, and zaleplon and to some extent zolpidem are not particularly effective at maintaining sleep throughout the night. A controlled release formulation of zolpidem (CR, currently only available in the USA) prolongs its nocturnal actions and enhances sleep continuity, though only by tens of minutes (Greenblatt et al., 2006). Individual factors seem important and some people are more susceptible to carry-over than others, probably due to individual differences either in the rate of drug clearance, which can vary by as much a twofold between subjects, or sensitivity to drug actions.

Tolerance, dependence and withdrawal

Dose escalation above recommended doses in patients with insomnia alone is uncommon, and tolerance to hypnotic drug effects is not a frequent problem in clinical experience; many patients use the same dose of hypnotic for months or years and still feel it works. However, a temporary worsening of sleep, usually with increased sleep-onset latency, is reported during the withdrawal period for most agents (Hajak et al., 2009; Soldatos et al., 1999; Voshaar et al., 2004). Although there have been no head-to-head studies addressing this question, there is some lower level evidence in humans that subtype selective drugs such as eszopiclone produce less tolerance and rebound (Krystal et al., 2003; Nutt and Stahl, 2009).

Animal and human research demonstrates that brain receptor function changes in response to chronic treatment with benzodiazepine receptor agonists, and this takes time to return to pre-medication levels after cessation of medication. There is evidence from animal studies that chronic administration of benzodiazepines produces adaptive changes in the receptor which attenuate the effects of the endogenous neurotransmitter GABA, and so produce symptoms on withdrawal (Bateson, 2002). It may be possible to develop drugs with a lower propensity to such effects either through targeting specific subtypes of the benzodiazepine receptor, by changing the chemical structure to produce a different interaction at the pharmacophore, or by making partial agonists (Doble et al., 2004).

Considerations of dependence are very much contingent on what happens when treatment is stopped. A psychological dependence is seen in many patients and some are unwilling to stop treatment. If they do stop there can be relapse, where the patient's original symptoms return, or rebound of symptoms, where for one or two nights there is a worsening of sleep disturbance, with longer sleep-onset latency and increased waking during sleep; this is commonly reported by patients and has been documented in some research studies (Hajak et al., 2009; Soldatos et al., 1999). More rarely, there is a longer withdrawal syndrome. All of these can be ameliorated by resuming medication. The withdrawal syndrome is characterized by the emergence of symptoms not previously experienced, such as agitation, headache, dizziness, dysphoria, irritability, fatigue, depersonalization, hypersensitivity to noise and visual stimuli. Physical symptoms which have been described include nausea, vomiting, muscle cramps, sweating,

Table 5. Level Ia evidence of hypnotic efficacy from subjective rating of sleep or objective polysomnographic measures

	Sleep-onset latency		Total sleep time		Sleep efficiency		Wake time after sleep onset		Sleep quality
	Self-rated	PSG	Self-rated	PSG	Self-rated	PSG	Self-rated	PSG	Self-rated
Benzodiazepines	✓	✓	✓		✓	✓	✓		✓
Z-drugs	✓	✓	✓		✓	✓	✓		✓

Table 6. Effects of individual drugs (significantly different from placebo (Ib)) on sleep parameters

	Sleep-onset latency		Total sleep time		Wake time after sleep onset		Sleep quality
	Self-rated	PSG	Self-rated	PSG	Self-rated	PSG	Self-rated
temazepam	(✓)*	(✓)	✓	✓	✓	✓	✓
lormetazepam	✓	✓	✓	✓	✓	✓	✓
zopiclone	✓	✓	✓	✓	✓	✓	✓
zolpidem	✓	✓	✓	✓	✓	No	✓
zaleplon	✓	✓	No	No	No	No	✓
eszopiclone	✓	✓	✓	✓	✓	✓	✓
ramelteon	✓	✓	✓	(week 1 only)	✓	No	✓
PR melatonin	✓	✓	Not measured	No	Not measured	No	✓

PSG: polysomnography.
*Formulation changed since studies, longer absorption time with current tablet cf gel capsule previous formulation.

weakness, muscle pain or twitching and ataxia. This syndrome typically resolves within a few weeks, but in some patients it persists, and this may be related to personality traits and cognitive factors (Murphy and Tyrer, 1991).

Hypnotic drug treatment

All licensed drugs are efficacious; levels of evidence for short-term use are given in summary in Table 5 (there is as yet no systematic review or meta-analysis evidence for prolonged-release melatonin). The sleep factors which each drug improves are given in Table 6. Thus, for example, in a patient with predominantly sleep-onset insomnia, a shorter-acting drug such as zolpidem or prolonged-release melatonin might be appropriate, and for those with awakenings throughout the night a slightly longer-acting drug such as zopiclone may be preferable.

Most of the licensed drugs enhance GABA function in the brain. As well as promoting sleep these drugs are anxiolytic, anticonvulsant and myorelaxant, and can cause ataxia and memory problems when taken other than just before a period in bed. If their effect in the brain persists after waking up in the morning they are described as having 'hangover' effects, therefore differences in the pharmacokinetics of individual benzodiazepines (or Z-drugs) are of particular importance. Melatonin does not give rise to motor or memory effects. Recent clinical trials have begun to measure daytime outcomes after hypnotic medications, and beneficial effects have been reported for melatonin in over-55s, zolpidem, zopiclone, eszopiclone and lormetazepam. These measures have not been used in studies of other drugs, so their effects on daytime function are not documented.

In systematic reviews of benzodiazepines and Z-drugs, adverse events/side effects are less common and less severe for the Z-drugs zolpidem, zaleplon and eszopiclone (Buscemi et al., 2007). Controlled studies measuring cognitive and psychomotor function (such as digit–symbol substitution test, and memory) in insomnia patients have only shown next-day deleterious effects consistently after use of flurazepam (very long-acting) or very high doses of other benzodiazepines (Buscemi et al., 2005). Evidence for hypnotic effects on next-day driving in insomnia patients is limited; however, epidemiological studies show that road accidents are increased in people taking benzodiazepines or zopiclone (Barbone et al., 1998; Neutel, 1995). Studies in healthy volunteers show that residual effects of hypnotics increase with their half-life duration (Verster et al., 2006). Effects of insomnia itself on driving have not been studied, but sleep deprivation does impair driving performance (Connor et al., 2002). In a controlled study of patients with insomnia in a driving simulator there was next-day impairment after zopiclone and lormetazepam but not zolpidem, when compared with placebo (Staner et al., 2005). Transient increases in sleep-onset latency and decreases in sleep time have been reported after stopping nearly all hypnotic drugs, except with zaleplon, melatonin and ramelteon: onset and duration is related to half-life, occurring on the first or second nights after stopping with short half-life drugs, and later and more prolonged with longer-acting ones (Hartmann and Cravens, 1973; Voderholzer et al., 2001).

Recommendations

- Factors which clinicians need to take into account when prescribing are efficacy, safety, and duration of action (A).
- Other factors are previous efficacy of the drug or adverse effects, history of substance abuse or dependence (D).

Long-term hypnotic use

What is known about long-term hypnotic treatment

- Insomnia is often long-lasting and is often treated with hypnotics for long periods in clinical practice (Ib)
- These studies suggest that dependence (tolerance/withdrawal) is not inevitable with hypnotic therapy up to 1 year with eszopiclone, zolpidem, ramelteon (Ib)
- There is also evidence that dependence may be more likely with some agents or with polysomnography outcome measures as compared with self-report measures of outcome
- Intermittent dosing may further reduce the risk of tolerance and dependence (Ib)

What is not known

- How can we predict the needed treatment duration?
- How and when should treatment be discontinued?
- Should dosing for longer periods be nightly or intermittent?
- How we detect the abuse-prone individual in the clinic?
- Does hypnotic therapy affect the course of insomnia or associated conditions?

The question of long-term hypnotic treatment is one of the more controversial areas in psychopharmacology. It has long been stated that hypnotic medication should not be used long term for the treatment of insomnia. This was the consensus view of the panel of a 1983 National Institute of Health (NIH, 1983) Consensus Conference on the medication treatment of insomnia, which became a guideline for clinical practice in the USA, and later the UK Committee on Safety of Medicines and the Royal College of Psychiatrists both recommended only short-term use. While it was appreciated that benzodiazepine hypnotic agents had a favourable risk–benefit ratio and were first-line agents for insomnia management, all these reports expressed concerns about the risks of physical dependence and recommended that their use should be limited to periods of 2–3 weeks. This view was not based on data demonstrating an unfavourable transition in the risk–benefit ratio after 2–3 weeks of treatment, but appears to have been because no substantive placebo-controlled trials of hypnotics had been carried out for longer than a few weeks. Despite the recommendation for treatment with hypnotic drugs being only 2–4 weeks, many millions of patients worldwide remain on long-term treatment (Balter and Uhlenhuth, 1992; Ishigooka et al., 1999; Ohayon et al., 1999; Mellinger et al., 1985).

The reasons for long-term use are complicated and difficult to research, but are probably similar to those which affect understanding of long-term benzodiazepine treatment in anxiety disorders. We do not know the proportions of long-term users who have continuing insomnia requiring daily drug treatment, or who do not need the drug at all, or who are afraid to try discontinuing because of fear or experience of rebound insomnia. In one study where people were successful in discontinuing benzodiazepine hypnotics, a follow-up after 2 years revealed approximately 40% had resumed regular use (Belanger et al., 2005; Morin et al., 2005a), which suggests some people have enduring problems with sleep which benefit from treatment. Insomnia may have some similarities with depression, in that both represent long-term disorders in which maintenance treatment may be needed in many patients (Jindal et al., 2004). A related issue is whether early intervention at the onset of insomnia might reduce the likelihood of it persisting. There is very little evidence available on this, and it must be seen as a research priority.

Placebo-controlled trials of hypnotic treatment for durations longer than 3 weeks that can more definitely assess safety and efficacy, and determine whether dependence phenomena occur, have been undertaken only recently. Trials of nightly dosing for up to 6 months' duration suggest that tolerance and withdrawal do not generally occur with some hypnotics: eszopiclone (two studies of 6 months' duration); ramelteon (a 6-month study with outcome assessed with PSG but not self report); and temazepam (a 2-month study) (Bastien et al., 2003; Krystal et al., 2003; Mayer et al., 2009; Morin et al., 1999; Walsh et al., 2007). Other agents have not been studied for longer durations. The available evidence does not suggest there is an unfavourable risk/benefit transition at 3–4 weeks for any agent.

Open-label studies of nightly dosing for periods of up to 1 year with the agents studied (zaleplon, eszopiclone, and ramelteon) suggest that discontinuation symptoms are mild and infrequent (Ancoli-Israel et al., 2005; Richardson et al., 2009). Intermittent, non-nightly dosing is also an important consideration with respect to long-term hypnotic treatment. Many individuals do not have nightly insomnia, and treatment only on the nights when drug is needed can decrease the risks and costs of therapy and reduce psychological dependence/treatment withdrawal anxiety. There is evidence from a placebo-controlled trial for sustained efficacy and safety for 6 months of 'as needed' treatment (subjects being required to take at least three doses per week) with controlled release zolpidem 12.5 mg (Krystal et al., 2008).

In conclusion, insomnia is often long-lasting and often treated with hypnotics for long periods in clinical practice. Controlled trials of longer-term use are being undertaken and these suggest dependence (tolerance/withdrawal) is not inevitable with hypnotic therapy up to 1 year, and is not characteristic of the several agents studied. Dependence may be more likely with some agents or with PSG outcome measures as compared with self-reported measures of outcome. The longer-term safety and efficacy of many other commonly used hypnotics remain uncertain.

A number of other critical issues remain unresolved. We currently lack the means to determine who should receive longer-term treatment and to predict the required treatment duration. Lacking the means to determine the optimal duration of therapy, a rational approach is to carry out periodic trials of tapering and discontinuing medication to determine if continued therapy is indicated (Krystal, 2009). As such, the duration of treatment is decided by a series of risk/benefit decisions based on trial discontinuations. This approach provides an 'exit strategy' and thereby addresses concerns that, once started, hypnotic therapy could be unending. Concomitant CBT during tapered discontinuation may be helpful (Morin et al., 2006). Another unresolved issue is whether to implement nightly or intermittent dosing of hypnotics for a given patient. In many instances this is a practical

decision based on whether the patient can predict, when they go to bed, whether they will have sleep difficulty.

Recommendations

- Use as clinically indicated (A).
- To stop medication, try intermittent use at first if it makes sense, then try to stop at regular intervals, say every 3–6 months depending on ongoing life circumstances and with patient's consent (D).
- CBT during taper improves outcome (A).

Antidepressants

What is known about the use of antidepressants to treat insomnia

- There is limited evidence for efficacy of doxepin, trimipramine, trazodone, paroxetine in insomnia (Ib)
- Antidepressants may affect a wide range of brain receptors and have longer-lasting carry-over effects than traditional hypnotic drugs – antidepressants are associated with increased risks of road accidents especially early in treatment in depression (Ib)

What is not known

- Is the effect of antidepressants on insomnia lasting (particularly as they are often prescribed for long periods)?
- Are they more efficacious than traditional hypnotics?
- Do they improve mood or reduce the risk of emergent depression in patients?

Tricyclic and some other classes of antidepressants have long been used for insomnia, whereas the selective serotonin reuptake inhibitors (SSRI) as a class generally disrupt sleep early in a course of treatment (Mayers and Baldwin, 2005). The alerting effect of SSRIs can be offset by co-administration of sedating antidepressants such as trazodone, probably because they block 5HT2 receptors that are being overstimulated by an increase in 5HT (Kaynak et al., 2004). Other 5HT2 antagonist antidepressants such as nefazodone (now discontinued) (Hicks et al., 2002) and mirtazapine (Winokur et al., 2003) have been shown to reduce insomnia in depression, especially early in treatment.

Low doses (sub-therapeutic for depression) of sedating tricyclics, particularly amitriptyline, dosulepin and doxepin, have been used for decades to treat insomnia. This is particularly common practice in primary care in the UK, where amitriptyline 10 or 25 mg is also used for long periods in many patients with chronic illness, particularly those with pain syndromes. At this dose amitriptyline is probably acting mostly as a histamine H1 receptor antagonist, although a degree of 5HT2 and cholinergic muscarinic antagonism may also contribute. There are no controlled studies of hypnotic efficacy of low-dose amitriptyline in insomnia, and tricyclics are more likely to be lethal than licensed hypnotics in overdose (Nutt, 2005a). Controlled trials have demonstrated an effect of doxepin in insomnia at low dose (25 mg) for 4 weeks with rebound insomnia (Hajak et al., 2001), and very low 'microdose' studies using 1, 2 or 6 mg for two nights in adult (Roth et al., 2007) and elderly insomnia patients (Scharf et al., 2008) have shown sleep improvement; at this dose the antihistamine action is paramount.

Trazodone is an antagonist at 5HT1a, 5HT2 and α1 adrenergic receptors as well as a weak 5HT reuptake inhibitor, and is the second most prescribed medication for insomnia in the US. It has a perceived absence of risk, is cheap, and there are no restrictions on use duration, but 25–30% patients experience difficulty tolerating trazodone and dropout rates tend to be higher than for benzodiazepine or Z-drugs. Although there have been 18 trazodone studies measuring sleep outcomes, only two were in primary insomnia, and only one was a controlled study (Walsh et al., 1998). This study used 50 mg trazodone versus placebo, and found a significant effect on sleep maintenance parameters at week 1 but not week 2, and a high incidence of daytime somnolence. Trimipramine is a tricyclic antidepressant which blocks α-1 adrenergic, histamine H1, dopamine D2, serotonin 5HT2 and cholinergic receptors (Gross et al., 1991; Richelson, 1994). There is one controlled trial (Riemann et al., 2002) in insomnia at doses of 50–200 mg for 4 weeks which found a significant improvement in sleep efficiency as measured by PSG, paralleled by subjective improvements. Side effects were described as marginal. Paroxetine, an SSRI, was studied in patients with insomnia aged over 55 years, at a median dose of 20 mg for 6 weeks (Reynolds et al., 2006), there being a 50% response rate (placebo 38%) with subjective sleep quality and daytime well-being improved. This seemingly paradoxical action of paroxetine to improve sleep is probably related to its good efficacy in many anxiety disorders, where it seems to reduce recurrent thinking and ruminations.

Taking SSRIs, venlafaxine, mianserin or mirtazapine increases the risk of restless legs syndrome (RLS) and periodic limb movements in sleep (PMLS) (Hoque and Chesson, 2010), and SSRIs are known to induce or exacerbate sleep bruxism (Wilson and Argyropoulos, 2005).

Recommendations

- Use drugs according to a knowledge of pharmacology (A).
- Consider antidepressants when there is coexistent mood disorder but then use at therapeutic doses (A).
- Beware toxicity of tricyclic antidepressants in overdose even when low unit doses prescribed (A).

Antipsychotics

What is known about use of antipsychotics for treatment of insomnia

- Olanzapine and quetiapine improve sleep in healthy volunteers (Ib)
- Quetiapine improves sleep in primary insomnia (IIb)
- Side effects are common because of the pharmacological actions of these drugs (I)

What is not known

- How do they compare with traditional hypnotic drugs?

Atypical antipsychotics have become relatively widely used in the treatment of sleep problems with very little controlled

trial evidence, although a meta-analysis of atypical antipsychotic agents in mania indicates they all produce somnolence (Scherk et al., 2007). Research studies have been carried out in healthy volunteers. Increases in objective actual sleep time and sleep continuity and in subjective sleep quality have been reported with olanzapine (Gimenez et al., 2007; Lindberg et al., 2002; Sharpley et al., 2000), which also improves sleep continuity when added to an SSRI in depression (Sharpley et al., 2005). Quetiapine at 25 mg and 100 mg for two nights in healthy volunteers increased sleep time and efficiency and subjective sleep quality but periodic leg movements were significantly increased after 100 mg (Cohrs et al., 2004). A single small open study of quetiapine (a 25 mg dose in most patients) for 6 weeks in primary insomnia (Wiegand et al., 2008) showed improvements in total sleep time and sleep efficiency, with transient adverse effects of morning hangover and dry mouth.

Side effects of these antipsychotics are well documented and include weight gain, metabolic syndrome, extrapyramidal symptoms and risk of tardive dyskinesia. There are some case reports of abuse of quetiapine in inpatients and prisoners (reviewed in Sansone and Sansone, 2010).

Recommendation

- Side effects are common because of the pharmacological actions of these drugs and there are a few reports of abuse. Together these indicate no indication for use as first-line treatment (D).

Antihistamines

Antihistamines are sedating and are sold as over-the-counter (OTC) sleeping medications. There is limited evidence that OTC antihistamines work, although recently some modest benefits have been reported after 2 weeks' dosing with diphenhydramine in mild insomnia (Morin et al., 2005b). More profound acute effects on sleep have been reported for both promethazine and hydroxyzine in healthy volunteers (Adam and Oswald, 1986; Alford et al., 1992), but the latter is not available as an OTC hypnotic, and both have a long duration of action so are likely to cause hangover. Triprolidine is used in many other European countries and may be better as it has a shorter half-life; however, there are no placebo-controlled studies.

Antihistamines are commonly used in alleviation of insomnia in drug and alcohol withdrawal where traditional hypnotics are less suitable due to the risk of cross-dependence, although there are no controlled trials in this setting.

Recommendations

- Antihistamines have a limited role in psychiatric and primary care practice for the management of insomnia (D).
- The algorithm for the treatment of insomnia is summarized in Figure 3.

Special populations

Sleep in women: effects of menopause

Insomnia increases as women approach and pass through the menopause (Bixler et al., 2009; Kuh et al., 1997; Owens and Matthews, 1998). This is due to a variety of reasons: climacteric symptoms such as hot flushes due to hormonal changes, psychiatric disorders and a rise in the incidence of sleep-disordered breathing (Bixler et al., 2001). A recent study looking at gender differences in the clinical presentation of patients diagnosed with obstructive sleep apnoea syndrome (OSAS) showed that at the time of OSAS diagnosis, women were more likely to be treated for depression, to have insomnia and to have hypothyroidism than men with similar degree of OSAS (Shepertycky et al., 2005). In the Wisconsin cohort of individuals followed over time, there were no significant PSG sleep architectural changes associated with transition to menopause, but there was an increase in obstructive apnoeas (Young et al., 2003). In contrast, a recently published US study of normal sleepers showed that women sleep more deeply than men and that the menopause is associated with longer sleep latency and decreased slow-wave sleep. In addition, hormone therapy appeared to protect women from these unfavourable changes (Bixler et al., 2009).

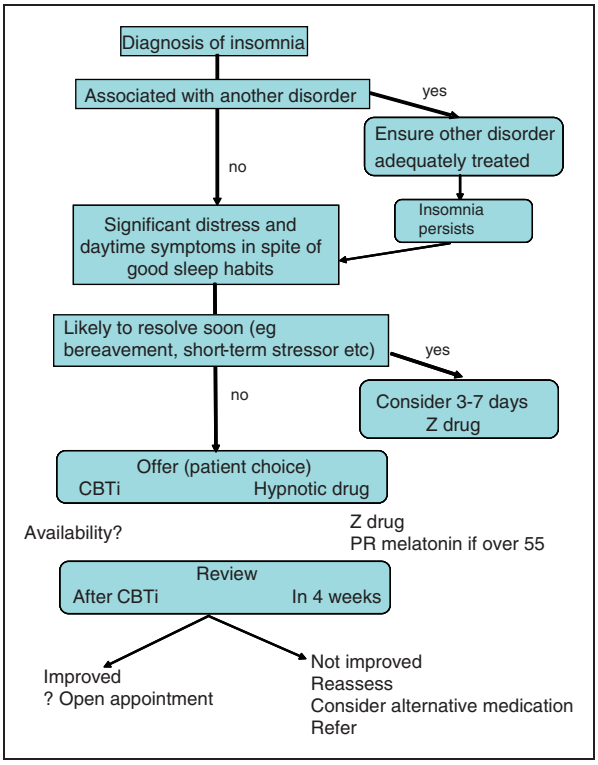


Figure 3. Treatment algorithm.

Recommendations

- Clinicians should appreciate that there is a rise in incidence of sleep-disordered breathing after the menopause and that clinical presentation, often including insomnia, in women is different than in men.
- The use of hormone therapy should involve informed individualized treatment of symptoms, looking at risks and benefits in light of recent studies.
- Follow recommendations for insomnia in other sections.

Sleep in women: effects of pregnancy

Many women complain of poor sleep during pregnancy, with the reasons varying depending on the trimester. In the first trimester, nausea, backache and urinary frequency can cause sleep disturbance. The second trimester tends to be easier but foetal movements and heartburn may be issues. By the third trimester, sleep is more disturbed with complaints again of urinary frequency and backache in addition to cramps, itch and unpleasant dreams. Most women fall asleep easily but wake more frequently (Schweiger, 1972).

If patient suffers from intractable insomnia and a pharmacological agent is required, it is helpful to note that zolpidem and diphenhydramine are in FDA class B (foetal harm possible, but unlikely; no evidence of foetal harm in animal studies); for review see Pien and Schwab, 2004. Zolpidem is preferable as it is short acting and does not have anticholinergic side effects.

RLS is common in pregnancy with a prevalence of 11–26% and is sometimes associated with anaemia (Manconi and Ferini-Strambi, 2004). Snoring and sleep-disordered breathing, especially in obese subjects can also occur and affect sleep quality (Pien and Schwab, 2004).

Recommendations

- Good sleep hygiene and lifestyle (D).
- Manage general pregnancy-associated complaints, e.g. decrease fluid intake, pillow support (D).
- The benefits of CBT in pregnancy have not been published but approach would appear sensible (B).
- Recognize RLS by careful history and investigations if necessary.
  - Dopamine agonists are contraindicated (FDA category C or greater)
  - Iron and folic acid supplementation have been shown to be effective in RLS. Supplementation is suggested even if levels are not low (D)
  - Keep caffeine low as it can exacerbate RLS (D)
  - Mild to moderate exercise in the early evening, stretching, massage (D)
- If patient suffers from intractable insomnia and a pharmacological agent is required, zolpidem is preferable as it is short acting and does not have anticholinergic side effects. Short-term use is recommended after discussion on potential risks and benefits (D).

Treatment of insomnia in the elderly

What is known about treatment of insomnia in the elderly

- Cognitive behavioural therapy is effective in insomnia in the elderly (Ia)
- Short-acting Z-drugs increase the risk of falls in elderly patients (III)
- Prolonged release melatonin given for 3 weeks improves sleep onset latency and sleep quality in patients over 55 (1b)

What is not known?

- What is the long-term efficacy and safety of melatonin?

Insomnia in elderly patients responds well to CBT (see psychological treatment section). Meta-analyses comparing CBT outcomes in middle-aged and older adults (55 years plus) have reported moderate-to-large effect sizes, regardless of age, in sleep-onset latency (SOL) and wake time after sleep onset (Irwin et al., 2006; Montgomery and Dennis, 2003).

A systematic review (Bain, 2006) and meta-analysis (Glass et al., 2005) concluded that benzodiazepine receptor agonist hypnotics had an unfavourable risk/benefit ratio in elderly patients. However, the different methods of collection and categorization of drug-related side effects in the studies included makes them difficult to interpret. Individual randomized controlled studies with short-acting Z-drugs show little evidence of adverse effects, particularly cognitive side effects in the morning. However, if a patient needs to rise within a few hours after taking a benzodiazepine agonist drug there may be undesired effects on motor control. Falls are increased after sedatives and hypnotics, neuroleptics and antipsychotics, antidepressants, benzodiazepines, non-steroidal anti-inflammatory drugs and calcium channel antagonists (Woolcott et al., 2009) and, for example, there is a 2.5-fold increase in the risk of falls in hospital after zolpidem (Rhalimi et al., 2009). However, in nursing homes the situation may be different; in a large study (Avidan et al., 2005) insomnia itself, but not hypnotic use, was associated with an increase in falls and hip fractures. Therefore the development of sleep-promoting drugs without motor side effects has been welcomed. Prolonged-release melatonin has been shown to reduce SOL and increase subjective sleep quality in two large trials in patient over 55 years (Lemoine et al., 2007; Wade et al., 2007); its effects are fairly modest but it has no known motor side effects.

Recommendations

- CBT is effective and should be offered as a first line where available (A).
- When a hypnotic is indicated in patients over 55, prolonged-release melatonin should be tried first (B).
- If a $GABA_A$ hypnotic is used then a shorter half-life will minimize unwanted hangover (A).

Treatment of sleep problems in children

What is known

- Most sleep disorders in childhood respond well to behavioural treatments (I)
- Melatonin reduces long sleep latency (following appropriate behavioural interventions) in children with sleep onset insomnia or delayed sleep phase syndrome and learning difficulties, autism and ADHD (II)
- Antihistamines may have a role in short-term symptomatic treatment (II)

What is not known

- What are the long-term effects of melatonin?

Sleep problems are commonly associated with certain genetic and neuro-developmental problems seen in childhood

including ADHD, autism, learning difficulties and epilepsy. Training and awareness of paediatric sleep disorders is poor, and accurate diagnoses and hence appropriate treatments are often delayed. Evidence from systematic review suggests that most sleep disorders in childhood respond well to behavioural treatments (Mindell et al., 2006). Appropriate sleep hygiene measures and more specific techniques of extinction, or graduated extinction, are all more effective than placebo at improving sleep and reducing the number of weekly night wakes in otherwise healthy children who regularly wake up in the night (Ramchandani et al., 2000). These interventions hold for both typically developing children and children with learning difficulties and sleep problems. These interventions may not change sleep parameters in the child, but instead improve outcomes related to impact on parents and other carers.

The sedative side effects of antihistamines may speed up behavioural programmes over short periods (France et al., 1991) but seem not to work without behavioural interventions; in a placebo-controlled double-blind trial in infants aged 6–27 months the same authors found no significant effect of 15 mg or 30 mg trimeprazine tartrate, and concluded that it is not recommended as a pharmacological treatment for infant sleep disturbance unless as an adjunct to a behavioural therapy program (France et al., 1999). Clinically, the short-term use of an H1 blocker for transient or extreme insomnia can be helpful and is frequently employed. However, tolerance can develop quickly and some children can experience dramatic and paradoxical over-arousal. Nevertheless, the TIRED RCT specifically investigated the use of diphenhydramine in infants aged 6–15 months and found it was no more effective than placebo in reducing night-time awakening (Merenstein et al., 2006).

The evidence supporting use of melatonin to reduce long sleep latency (following appropriate behavioural interventions) in populations of children with idiopathic sleep-onset insomnia (Smits et al., 2003) or DSPS and learning difficulties, autism and ADHD (van der Heijden et al., 2007) is increasingly robust. However, evidence that melatonin can significantly improve sleep fragmentation and total sleep time in this group is only weak. The majority of research in children has employed supraphysiological doses of fast-release melatonin, and although there is little evidence of short-term adverse effects, there are also only limited data on long-term potential adverse effects. Melatonin at doses between 0.5 and 12 mg is commonly used as a sedative agent in children undergoing procedures such as electroencephalography (EEG), as an alternative to sleep deprivation that does not affect the EEG morphology. A melatonin-induced sleep EEG was as useful as a sleep-deprived EEG, but children's behaviour on the day of the melatonin-induced sleep EEG recording was more acceptable to parents (Wassmer et al., 2001).

Clonidine is an antihypertensive agent with sedative side effects that may improve sleep maintenance in some children. The therapeutic window is narrow, both for adverse effects on sleep architecture and tolerability. Also tolerance to the sleep-inducing effects develops over time, leading to the need for increased doses with concomitant risk of adverse effects. Despite these concerns, it is still widely used in the UK and by as many as a third of clinicians surveyed in the USA (Schnoes et al., 2006).

Chloral hydrate and triclofos are still popular hypnotics for children but have a very long half-life and considerable potential for 'hangover' effects in children. The half-life of chloral hydrate itself is short (a few minutes), but the half-lives of its active metabolites are longer, being 8–12 h for trichloroethanol and 67 h for trichloroacetic acid. Toxicity is an important concern due to central nervous system depressant action, arrhythmogenic potential and stomach irritation.

Recommendations

- Behavioural strategies should be tried in children with disturbed sleep (A).
- Melatonin administration can be used to advance sleep onset to normal values in children with ADHD who are not on stimulant medication (A).

Treatment of insomnia in children and adults with learning disability

Epidemiological studies show a very high prevalence of sleep disturbance in people with learning disability, with findings ranging from 58–86% in children (Didden and Sigafoos, 2001) and 14–56% in adults (Brylewski and Wiggs, 1999) (this study reported a 15% prevalence of parasomnias). Positive associations have been reported between sleep disturbance and sleep breathing disorders, challenging behaviours, early childhood, severe or profound learning disability, institutional care, autism/ADHD, various genetic syndromes, physical health problems, sensory impairment, epilepsy and caffeine intake (Brylewski and Wiggs, 1999). Many different aspects contribute to aetiology, such as neurodevelopmental causes, sensory impairments, chaotic or institutionalized environments, failure of learning and psychotropic medications, including anticonvulsants.

Clinical assessment should elicit any aetiological or exacerbating factors which can be reversed. Assessment will usually take place by direct observation initially. Carers should be supported to keep a structured 24-h record of sleep pattern and behaviour. Actigraphy or EEG may be useful when a sleep disorder other than insomnia or settling difficulties is suspected. A circadian rhythm disorder should be considered in individuals with visual impairment (see below).

There is a varying degree of evidence for treatments of sleep difficulties in this heterogeneous population. The relatively small number of controlled studies in this area give support to parental/carer education and modifying environmental factors (Montgomery et al., 2004) and behavioural regimes such as chronotherapy, bedtime fading, extinction, distancing/desensitization and sleep–wake scheduling (Gunning and Espie, 2003; Wiggs and France, 2000). The use of light therapy has been described (Short and Carpenter, 1998).

There is very little evidence for effectiveness of sleep-promoting drugs apart from melatonin. A recent meta-analysis (Braam et al., 2009) shows that melatonin (1–9 mg) decreases sleep latency and number of wakes per night, and increases total sleep time in individuals with intellectual disabilities. There were few adverse events in the relatively short-term studies included, and long-term safety needs further research.

Recommendations

- Clinical assessment should describe sleep disturbance and elicit aetiological and exacerbating factors (A).
- Environmental, behavioural and educational approaches should be used first line (A).
- Melatonin is effective in improving sleep (A).
- Treatment should be planned within a capacity/best interests framework.

Treating circadian rhythm disorders

What is known

- Melatonin is effective in jet lag disorder (1a), delayed sleep phase syndrome (Ib) and free-running disorder (IIa)
- Light therapy is effective in delayed sleep-phase syndrome (III)

What is not known

- What are the best efficacy measures – subjective versus objective?
- Is there a need to distinguish between adults and adolescents in delayed sleep-phase disorder, since sleep times are somewhat delayed in normal adolescence?
- Is there a need to distinguish between sighted and blind individuals?
- Is melatonin or light therapy more effective for delayed sleep-phase disorder?

Current understanding of circadian rhythms and sleep physiology provides a strong theoretical basis for the use of melatonin in some, but not all circadian rhythm disorders (CRDs). Empirical evidence for efficacy is strong in some CRDs, but weak or absent in others. Melatonin agonists may be promising in the treatment of CRDs but there remains a need for RCTs in well-characterized CRD populations.

There is sufficient evidence to support the use of melatonin in jet lag (Herxheimer and Petrie, 2002; Sack et al., 2007b), but melatonin has to be taken near desired bedtime otherwise there may undesired daytime sleepiness. An evidence-based strategy for minimizing jet lag which includes strategic scheduling of sleep combined with melatonin is given in a recent paper by Sack (2010).

In delayed sleep-phase disorder, there is both a theoretical and an empirical basis for use of melatonin, which is effective in practice, shown in two systematic reviews (Sack et al., 2007a; MacMahon et al., 2005); however, studies in these reviews vary in the physiological and subjective outcomes measured. Direct comparison with other therapies such as timed light exposure, for which there is a little evidence of efficacy (see below), or chronotherapy, for which there are no controlled trials, has not been reported.

In free-running disorder in sighted individuals, case reports ($n = 5$) suggest a positive benefit of melatonin. The evidence in blind people is more compelling, where case reports and two small, single-blind placebo-controlled studies are positive (Sack et al., 2007a; Skene and Arendt, 2007; Skene et al., 1999).

There is no evidence of efficacy of melatonin in irregular sleep–wake rhythm, or in shift work disorder, although there have been some reports of use in shift workers with varying results (for review see Sack et al., 2007b).

Bright light therapy has been used effectively in DSPS (for review see Shirani and St Louis, 2009). Exposure to bright light of 2500 lux for 2 h in the early morning, combined with light restriction after 16:00 (dark goggles) is an effective treatment for DSPS, and a light mask offering exposure to gradually increasing light intensity through closed eyelids over the last 4 h of habitual sleep time has been shown to be effective in these patients. Despite limited evidence, the American Academy of Sleep Medicine currently considers timed phototherapy as "a rational and effective intervention for DSPT" (Sack et al., 2007a).

Recommendations

- Clinical assessment is essential in DSPS and free-running disorder (A/B).
- Melatonin may be useful in DSPS, free-running disorder and jet lag (A).
- Other approaches such as behavioural regimes and scheduled light exposure (in sighted individuals) can also be used (B/C).

Treatment of parasomnias

There is little high-level evidence for treatments in these disorders. There are no controlled trials of treatment of non-REM parasomnias in adults (see Harris and Grunstein, 2009). Priorities are to minimize possible trigger factors such as frightening films, caffeine, alcohol or meals late at night, and to make sure there is a stable and adequate sleep–wake schedule. It is important to safeguard against harm to the patient, such as by locking windows, bolting doors, or sleeping on the ground floor, and safety of the bed partner or nearby children also requires attention.

Drug treatment decisions should be based on the frequency and severity of events. Clonazepam in doses up to 3 mg per night has been reported to be effective (case series, $n = 69$) (Schenck and Mahowald, 1996). Smaller case series have reported good effects of paroxetine (Wilson et al., 1997) and imipramine (Cooper, 1987) (both effective immediately), and there is a small case series of hypnotherapy in sleepwalkers (Reid et al., 1981). A randomized controlled study of 3 weeks' treatment with 5-hydroxytryptamine in children found evidence of efficacy at 6-month follow-up (Bruni et al., 2004).

For nightmares, psychological treatments are effective and these focus on exposure – writing down dreams – or guided imagery, pleasant images, and 'changing the ending' (Burgess et al., 1998; Krakow et al., 1995). There have been a few case series showing beneficial effects of the alpha-1 adrenergic blocker prazosin in reducing nightmares related to post-traumatic stress disorder in both military and civilian settings (Raskind et al., 2007). Nightmares have been reported to be triggered or worsened by many drug treatments, including cholinesterase inhibitors, beta-blockers, SSRIs (especially paroxetine) levodopa, and following withdrawal from antidepressants.

There are no prospective or controlled studies of drug treatment of REM behaviour disorder, but case series suggest

a good effect for clonazepam 1–4 mg (Aurora et al., 2010; Boeve et al., 2004) in reducing the number of episodes and injuries during them, although it should be used with caution in patients with dementia, disorders of gait or balance, or concomitant OSAS. Smaller beneficial effects have been reported for melatonin 3–12 mg (Gagnon et al., 2006). Single case studies and small series have reported beneficial effects of clonidine (Nash et al., 2003), donepezil (Massironi et al., 2003) and sodium oxybate (Kosky et al., 2008).

Drugs which can worsen RBD or provoke its symptoms include SSRIs, venlafaxine, mirtazapine, bisoprolol, and tramadol (Gagnon et al., 2006).

Funding

This research received no specific grant from any funding agency in the public, commercial, or not-for-profit sectors. The costs of the meeting were partly defrayed by unrestricted educational grants from two pharmaceutical companies (Lundbeck and GSK).

Conflict of interest

All attendees completed conflict of interest statements that are held at the British Association for Psychopharmacology office according to BAP policy.

References

Abanades S, Farré M, Segura M, Pichini S, Barral D, Pacifici R, Pellegrini M, Fonseca F, Langohr K, De La Torre R (2006) Gamma-hydroxybutyrate (GHB) in humans: pharmacodynamics and pharmacokinetics. *Ann N Y Acad Sci* 1074: 559–576.

Adam K and Oswald I (1986) The hypnotic effects of an antihistamine: promethazine. *Br J Clin Pharmacol* 22: 715–717.

Alford CA, Rombaut N, Jones J, Foley S, Idzikowski C and Hindmarch I (1992) Acute effects of hydroxyzine on nocturnal sleep and sleep tendency the following day: a C–EEG study. *Hum Psychopharmacol* 7: 25–37.

Altena E, Van Der Werf YD, Strijers RL and Van Someren EJ (2008) Sleep loss affects vigilance: effects of chronic insomnia and sleep therapy. *J Sleep Res* 17: 335–343.

Ancoli-Israel S, Richardson GS, Mangano RM, Jenkins L, Hall P and Jones WS (2005) Long-term use of sedative hypnotics in older patients with insomnia. *Sleep Med* 6: 107–113.

Anderson IM, Ferrier IN, Baldwin RC, Cowen PJ, Howard L, Lewis G, et al. (2008) Evidence-based guidelines for treating depressive disorders with antidepressants: a revision of the 2000 British Association for Psychopharmacology guidelines. *J Psychopharmacol* 22: 343–396.

Anderson IM, Nutt DJ and Deakin JF (2000) Evidence-based guidelines for treating depressive disorders with antidepressants: a revision of the 1993 British Association for Psychopharmacology guidelines. British Association for Psychopharmacology. *J Psychopharmacol* 14: 3–20.

Ashton, CH. (1994) Guidelines for the rational use of benzodiazepines. When and what to use. *Drugs* 48: 25–40.

Attenburrow M, Dowling B and Sharpley A (1996) Case-control study of evening melatonin concentration in primary insomnia. *Br Med J* 312: 1263–1264.

Aurora RN, Zak RS, Maganti RK, Auerbach SH, Casey KR, Chowdhuri S, et al. (2010) Best practice guide for the treatment of REM sleep behavior disorder (RBD). *J Clin Sleep Med* 6: 85–95.

Avidan AY, Fries BE, James ML, Szafara KL, Wright GT and Chervin RD (2005) Insomnia and hypnotic use, recorded in the minimum data set, as predictors of falls and hip fractures in Michigan nursing homes. *J Am Geriatr Soc* 53: 955–962.

Baglioni C, Spiegelhalder K, Lombardo C and Riemann D (2010) Sleep and emotions: A focus on insomnia. *Sleep Med Rev* 14: 227–238.

Bain KT (2006) Management of chronic insomnia in elderly persons. *Am J Geriatr Pharmacother* 4: 168–192.

Baldwin DS, Anderson IM, Nutt DJ, Bandelow B, Bond A, Davidson JR, et al. (2005) Evidence-based guidelines for the pharmacological treatment of anxiety disorders: recommendations from the British Association for Psychopharmacology. *J Psychopharmacol* 19: 567–596.

Balter MB and Uhlenhuth EH (1992) New epidemiologic findings about insomnia and its treatment. *J Clin Psychiatry* 53(Suppl): 34–39.

Barbone F, McMahon AD, Davey PG, Morris AD, Reid IC, McDevitt DG, et al. (1998) Association of road-traffic accidents with benzodiazepine use. *Lancet* 352: 1331–1336.

Bastien CH, Fortier-Brochu E, Rioux I, LeBlanc M, Daley M and Morin CM (2003) Cognitive performance and sleep quality in the elderly suffering from chronic insomnia. Relationship between objective and subjective measures. *J Psychosom Res* 54: 39–49.

Bateson AN (2002) Basic pharmacologic mechanisms involved in benzodiazepine tolerance and withdrawal. *Curr Pharm Des* 8: 5–21.

Beer B, Ieni JR, Wu WH, Clody D, Amorusi P, Rose J, Mant T, Gaudreault J, Cato A and Stern W (1994) A placebo-controlled evaluation of single, escalating doses of CL 284,846, a non-benzodiazepine hypnotic. *J Clin Pharmacol* 34: 335–344.

Belanger L, Morin CM, Bastien C and Ladouceur R (2005) Self-efficacy and compliance with benzodiazepine taper in older adults with chronic insomnia. *Health Psychol* 24: 281–287.

Benet LZ, Oie S and Schwartz JB (1996) Design and optimization of dosage regimens: Pharmacokinetic data. In: Hardman JG, Limbird LE, Molinoff PB, Ruddon RW, Gilman AG (eds), *Goodman and Gilman's: The Pharmacological Basis of Therapeutics*, 9th edn. New York: McGraw-Hill, pp. 1707–1792.

Billiard M, Bassetti C, Dauvilliers Y, Dolenc-Groselj L, Lammers GJ, Mayer G, et al. (2006) EFNS guidelines on management of narcolepsy. *Eur J Neurol* 13: 1035–1048.

Bixler EO, Papaliaga MN, Vgontzas AN, Lin HM, Pejovic S, Karataraki M, et al. (2009) Women sleep objectively better than men and the sleep of young women is more resilient to external stressors: effects of age and menopause. *J Sleep Res* 18: 221–228.

Bixler EO, Vgontzas AN, Lin HM, Ten HT, Rein J, Vela-Bueno A, et al. (2001) Prevalence of sleep-disordered breathing in women: effects of gender. *Am J Respir Crit Care Med* 163: 608–613.

Boeve BF, Silber MH and Ferman TJ (2004) REM sleep behavior disorder in Parkinson's disease and dementia with Lewy bodies. *J Geriatr Psychiatry Neurol* 17: 146–157.

Braam W, Smits MG, Didden R, Korzilius H, Van GI and Curfs LM (2009) Exogenous melatonin for sleep problems in individuals with intellectual disability: a meta-analysis. *Dev Med Child Neurol* 51: 340–349.

Breslau N, Roth T, Rosenthal L and Andreski P (1996) Sleep disturbance and psychiatric disorders: a longitudinal epidemiological study of young adults. *Biol Psychiatry* 39: 411–418.

Brunello N, Bettica P, Amato D, Maier G and Nutt D (2008) Pharmacokinetics of (S)zopiclone and (S)-desmethylzopiclone

following dosing with zopiclone and eszopiclone. *European Neuropsychopharmacology* 18, S4: S581–S582.

Bruni O, Ferri R, Miano S and Verrillo E (2004) L-5-Hydroxytryptophan treatment of sleep terrors in children. *Eur J Pediatr* 163: 402–407.

Brylewski J and Wiggs L (1999) Sleep problems and daytime challenging behaviour in a community-based sample of adults with intellectual disability. *J Intellect Disabil Res* 43: 504–512.

Burgess M, Gill M and Marks I (1998) Postal self-exposure treatment of recurrent nightmares. Randomised controlled trial. *Br J Psychiatry* 172: 257–262.

Burns A, O'Brien J, Auriacombe S, Ballard C, Broich K, Bullock R, et al. (2006) Clinical practice with anti-dementia drugs: a consensus statement from British Association for Psychopharmacology. *J Psychopharmacol* 20: 732–755.

Buscemi N, Vandermeer B, Friesen C, Bialy L, Tubman M, Ospina M, et al. (2005) Manifestations and management of chronic insomnia in adults. *Evid Rep Technol Assess (Summ)* 125: 1–10.

Buscemi N, Vandermeer B, Friesen C, Bialy L, Tubman M, Ospina M, et al. (2007) The efficacy and safety of drug treatments for chronic insomnia in adults: a meta-analysis of RCTs. *J Gen Intern Med* 22: 1335–1350.

Cajochen C, Krauchi K and Wirz-Justice A (2003) Role of melatonin in the regulation of human circadian rhythms and sleep. *J Neuroendocrinol* 15: 432–437.

Chang PP, Ford DE, Mead LA, Cooper–Patrick L and Klag MJ (1997) Insomnia in young men and subsequent depression. The Johns Hopkins Precursors Study. *Am J Epidemiol* 146: 105–114.

Chevalier H, Los F, Boichut D, Bianchi M, Nutt DJ, Hajak G, et al. (1999) Evaluation of severe insomnia in the general population: results of a European multinational survey. *J Psychopharmacol* 13: S21–S24.

Chouinard G, Lefko-Singh K and Teboul E (1999) Metabolism of anxiolytics and hypnotics: benzodiazepines, buspirone, zoplicone, and zolpidem. *Cellular and Molecular Neurobiology* 19: 533–552.

Clark BG, Jue SG, Dawson GW and Ward A (1988) Loprazolam. A preliminary review of its pharmacodynamic and pharmacokinetic properties and therapeutic efficacy in insomnia. *Drugs* 31: 500–516.

Cohrs S, Rodenbeck A, Guan Z, Pohlmann K, Jordan W, Meier A, et al. (2004) Sleep-promoting properties of quetiapine in healthy subjects. *Psychopharmacology (Berl)* 174: 421–429.

Connor J, Norton R, Ameratunga S, Robinson E, Civil I, Dunn R, et al. (2002) Driver sleepiness and risk of serious injury to car occupants: population-based case control study. *Br Med J* 324: 1125.

Cooper AJ (1987) Treatment of coexistent night-terrors and somnambulism in adults with imipramine and diazepam. *J Clin Psychiatry* 48: 209–210.

Crisp AH (1996) The sleepwalking/night terrors syndrome in adults. *Postgrad Med J* 72: 599–604.

Daley M, Morin CM, LeBlanc M, Gregoire JP, Savard J and Baillargeon L (2009) Insomnia and its relationship to health-care utilization, work absenteeism, productivity and accidents. *Sleep Med* 10: 427–438.

Davis PC, Wong J and Gefvert O (1999) Analysis and pharmacokinetics of quetiapine and two metabolites in human plasma using reversed-phase HPLC with ultraviolet and electrochemical detection. *J Pharm Biomed Anal* 20: 271–282.

DeMuro RL, Nafziger AN, Blask DE, Menhinick AM and Bertino JS Jr (2000) The absolute bioavailability of oral melatonin. *J Clin Pharmacol* 40: 781–784.

DeVane CL (1994) Pharmacokinetics of the newer antidepressants: Clinical relevance. *The American Journal of Medicine* 97, S1: S13–S23.

De Vanna M, Rubiera M, Onor ML and Aguglia E (2007) Role of lormetazepam in the treatment of insomnia in the elderly. *Clin Drug Investig* 27: 325–332.

Di WL, Kadva A, Djahanbakhch O, Silman R (1998) Radioimmunoassay of bound and free melatonin in plasma. *Clin Chem* 44: 304–310.

Didden R and Sigafoos J (2001) A review of the nature and treatment of sleep disorders in individuals with developmental disabilities. *Res Dev Disabil* 22: 255–272.

Dijk DJ and von Schantz M (2005) Timing and consolidation of human sleep, wakefulness, and performance by a symphony of oscillators. *J Biol Rhythms* 20: 279–290.

DiMario FJ and Emery ES (1987) The natural history of night terrors. *Clin Pediatr (Phila)* 26: 505–511.

Doble A, Martin IL and Nutt DJ (2004) *Calming the Brain: Benzodiazepines and Related Drugs from Laboratory to Clinic*. London: Martin Dunitz Limited.

Dowling GA, Burr RL, Van Someren EJ, Hubbard EM, Luxenberg JS, Mastick J, et al. (2008) Melatonin and bright-light treatment for rest–activity disruption in institutionalized patients with Alzheimer's disease. *J Am Geriatr Soc* 56: 239–246.

Drover DR (2004) Comparative pharmacokinetics and pharmacodynamics of short-acting hypnosedatives: zaleplon, zolpidem and zopiclone. *Clin Pharmacokinet* 43: 227–238.

Edinger JD, Bonnet MH, Bootzin RR, Doghramji K, Dorsey CM, Espie CA, et al. (2004) Derivation of research diagnostic criteria for insomnia: report of an American Academy of Sleep Medicine Work Group. *Sleep* 27: 1567–1596.

Edinger JD, Means MK, Carney CE and Krystal AD (2008) Psychomotor performance deficits and their relation to prior nights' sleep among individuals with primary insomnia. *Sleep* 31: 599–607.

Espie CA (2009) "Stepped care": a health technology solution for delivering cognitive behavioral therapy as a first line insomnia treatment. *Sleep* 32: 1549–1558.

Ford DE and Kamerow DB (1989) Epidemiological study of sleep disturbances and psychiatric disorders: an opportunity for prevention? *JAMA* 262: 1479–1484.

Fernandez C, Maradeix V, Gimenez F, Thuillier A and Farinotti R (1993) Pharmacokinetics of zopiclone and its enantiomers in Caucasian young healthy volunteers. *Drug Metab Dispos* 21: 1125–1128.

France KG, Blampied NM and Wilkinson P (1991) Treatment of infant sleep disturbance by trimeprazine in combination with extinction. *J Dev Behav Pediatr* 12: 308–314.

France KG, Blampied NM and Wilkinson P (1999) A multiple-baseline, double-blind evaluation of the effects of trimeprazine tartrate on infant sleep disturbance. *Exp Clin Psychopharmacol* 7: 502–513.

Gagnon JF, Postuma RB and Montplaisir J (2006) Update on the pharmacology of REM sleep behavior disorder. *Neurology* 67: 742–747.

Gaillot J, Heusse D, Hougton GW, Marc Aurele J and Dreyfus JF (1982) Pharmacokinetics and metabolism of zopiclone. *Int Pharmacopsychiatry* 17, S2: 76–91.

Gefvert O, Bergström M, Långström B, Lundberg T, Lindström L and Yates R (1998) Time course of central nervous dopamine-D2 and 5-HT2 receptor blockade and plasma drug concentrations after discontinuation of quetiapine (Seroquel) in patients with schizophrenia. *Psychopharm* 135: 119–126.

Gimenez S, Clos S, Romero S, Grasa E, Morte A and Barbanoj MJ (2007) Effects of olanzapine, risperidone and haloperidol on

sleep after a single oral morning dose in healthy volunteers. *Psychopharmacology (Berl)* 190: 507–516.

Glass J, Lanctot KL, Herrmann N, Sproule BA and Busto UE (2005) Sedative hypnotics in older people with insomnia: meta-analysis of risks and benefits. *Br Med J* 331: 1169.

Goodwin GM (2003) Evidence-based guidelines for treating bipolar disorder: recommendations from the British Association for Psychopharmacology. *J Psychopharmacol* 17: 149–173.

Goodwin GM (2009) Evidence-based guidelines for treating bipolar disorder: revised second edition – recommendations from the British Association for Psychopharmacology. *J Psychopharmacol* 23: 346–388.

Greenblatt DJ, Schillings RT, Kyriakopoulos AA, Shader RI, Sisenwine SF, Knowles JA and Ruelius HW (1976) Clinical pharmacokinetics of lorazepam. I. Absorption and disposition of oral 14C-lorazepam. *Clin Pharmacol Ther* 20: 329–341.

Greenblatt DJ, Harmatz JS, von Moltke LL, Ehrenberg BL, Harrel L, Corbett K, Counihan M, Graf JA, Darwish M, Mertzanis P, Martin PT, Cevallos WH and Shader RI (1998) Comparative kinetics and dynamics of zaleplon, zolpidem, and placebo. *Clin Pharmacol Ther* 64: 553–561.

Greenblatt DJ, Legangneux E, Harmatz JS, Weinling E, Freeman J, Rice K, et al. (2006) Dynamics and kinetics of a modified-release formulation of zolpidem: comparison with immediate-release standard zolpidem and placebo. *J Clin Pharmacol* 46: 1469–1480.

Gross G, Xin X and Gastpar M (1991) Trimipramine: pharmacological reevaluation and comparison with clozapine. *Neuropharmacology* 30: 1159–1166.

Gunning MJ and Espie CA (2003) Psychological treatment of reported sleep disorder in adults with intellectual disability using a multiple baseline design. *J Intellect Disabil Res* 47: 191–202.

Haimov I (2001) Melatonin rhythm abnormalities and sleep disorders in the elderly. *CNS Spectr* 6: 502–506.

Hajak G, Hedner J, Eglin M, Loft H, Storustovu SI, Lutolf S, et al. (2009) A 2-week efficacy and safety study of gaboxadol and zolpidem using electronic diaries in primary insomnia outpatients. *Sleep Med* 10: 705–712.

Hajak G, Rodenbeck A, Voderholzer U, Riemann D, Cohrs S, Hohagen F, et al. (2001) Doxepin in the treatment of primary insomnia: a placebo-controlled, double-blind, polysomnographic study. *J Clin Psychiatry* 62: 453–463.

Harris M and Grunstein RR (2009) Treatments for somnambulism in adults: assessing the evidence. *Sleep Med Rev* 13: 295–297.

Hartmann E and Cravens J (1973) The effects of long-term administration of psychotropic drugs on human sleep. VI. The effects of chlordiazepoxide. *Psychopharmacologia* 33: 233–245.

Herxheimer A and Petrie KJ (2002) Melatonin for the prevention and treatment of jet lag. *Cochrane Database Syst Rev* 2: CD001520.

Hicks JA, Argyropoulos SV, Rich AS, Nash JR, Bell CJ, Edwards C, et al. (2002) Randomised controlled study of sleep after nefazodone or paroxetine treatment in out-patients with depression. *Br J Psychiatry* 180: 528–535.

Hiimpel M, Stoppelli I, Milia S and Rainer E (1982) Pharmacokinetics and biotransformation of the new benzodiazepine, lormetazepam, in man. III. Repeated administration and transfer to neonates via breast milk.

Hoque R and Chesson AL Jr (2010) Pharmacologically induced/exacerbated restless legs syndrome, periodic limb movements of sleep, and REM behavior disorder/REM sleep without atonia: literature review, qualitative scoring, and comparative analysis. *J Clin Sleep Med* 6: 79–83.

ICD-10 (1992) *The ICD-10 Classification of Mental and Behavioural Disorders – clinical descriptions and diagnostic guidelines*, 1 edn, World Health Organization.

Idzikowski C, Mills FJ, Burton S and James R (1988) 5-HT-2 antagonists ritanserin and seganserin increase human SWS. *Sleep* 7: 43.

Irwin MR, Cole JC and Nicassio PM (2006) Comparative meta-analysis of behavioral interventions for insomnia and their efficacy in middle-aged adults and in older adults 55+ years of age. *Health Psychol* 25: 3–14.

Ishigooka J, Suzuki M, Isawa S, Muraoka H, Murasaki M and Okawa M (1999) Epidemiological study on sleep habits and insomnia of new outpatients visiting general hospitals in Japan. *Psychiatry Clin Neurosci* 53: 515–522.

Jia F, Goldstein PA and Harrison NL (2009) The modulation of synaptic GABA(A) receptors in the thalamus by eszopiclone and zolpidem. *J Pharmacol Exp Ther* 328: 1000–1006.

Jindal RD, Buysse DJ and Thase ME (2004) Maintenance treatment of insomnia: what can we learn from the depression literature? *Am J Psychiatry* 161: 19–24.

Jochemsein R and Breimer DD (1986) Pharmacokinetics of temazepam compared with other benzodiazepine hypnotics – some clinical consequences. *Acta Psychiatr Scand* 74, S332: 20–31.

Jostell K-G, Agurell S, Allgén L-G, Kuylenstierna B, Lindgren J-E, Åberg G and Österlöf G (1978) Pharmacokinetics of clomethiazole in healthy adults. *Acta Pharmacologica et Toxicologica* 43: 180–189.

Katz DA and McHorney CA (2002) The relationship between insomnia and health-related quality of life in patients with chronic illness. *J Fam Pract* 51: 229–235.

Kaynak H, Kaynak D, Gozukirmizi E and Guilleminault C (2004) The effects of trazodone on sleep in patients treated with stimulant antidepressants. *Sleep Med* 5: 15–20.

Kosky C, Bonakis A, Merritt S, Higgins S, De-Lacy S and Williams A (2008) Sodium oxybate improves coexisting REM behavior disorder in narcolepsy with cataplexy. *J Sleep Res* 17: 19.

Koytchev R, Alken RG, Kirkov V, Neshev G, Vagaday M, Kunter U (1994) Absolute bioavailability of chlorpromazine, promazine and promethazine. *Arzneimittelforschung* 44: 121–125 (in German).

Krakow B, Kellner R, Pathak D and Lambert L (1995) Imagery rehearsal treatment for chronic nightmares. *Behav Res Ther* 33: 837–843.

Krystal AD (2009) A compendium of placebo-controlled trials of the risks/benefits of pharmacological treatments for insomnia: the empirical basis for U.S. clinical practice. *Sleep Med Rev* 13: 265–274.

Krystal AD, Erman M, Zammit GK, Soubrane C and Roth T (2008) Long-term efficacy and safety of zolpidem extended-release 12.5 mg, administered 3 to 7 nights per week for 24 weeks, in patients with chronic primary insomnia: a 6-month, randomized, double-blind, placebo-controlled, parallel-group, multicenter study. *Sleep* 31: 79–90.

Krystal AD, Walsh JK, Laska E, Caron J, Amato DA, Wessel TC, et al. (2003) Sustained efficacy of eszopiclone over 6 months of nightly treatment: results of a randomized, double-blind, placebo-controlled study in adults with chronic insomnia. *Sleep* 26: 793–799.

Kuh DL, Hardy R and Wadsworth M (1997) Women's health in midlife: the influence of the menopause, social factors and health in earlier life. *Br J Obstet Gynaecol* 104: 1419.

Landolt HP, Meier V, Burgess HJ, Finelli LA, Cattelin F, Achermann P, et al. (1999) Serotonin-2 receptors and human sleep: effect of a selective antagonist on EEG power spectra. *Neuropsychopharmacology* 21: 455–466.

Landolt HP, Retey JV, Tonz K, Gottselig JM, Khatami R, Buckelmuller I, et al. (2004) Caffeine attenuates waking and

sleep electroencephalographic markers of sleep homeostasis in humans. *Neuropsychopharmacology* 29: 1933–1939.

LeBlanc M, Merette C, Savard J, Ivers H, Baillargeon L and Morin CM (2009) Incidence and risk factors of insomnia in a population-based sample. *Sleep* 32: 1027–1037.

Léger D and Bayon V (2010) Societal costs of insomnia. *Sleep Med Rev* [Epub ahead of print].

Léger D and Poursain B (2005) An international survey of insomnia: under-recognition and under-treatment of a polysymptomatic condition. *Curr Med Res Opin* 21: 1785–1792.

Léger D, Laudon M and Zisapel N (2004) Nocturnal 6-sulfatoxymelatonin excretion in insomnia and its relation to the response to melatonin replacement therapy. *Am J Med* 116: 91–95.

Léger D, Scheuermaier K, Philip P, Paillard M and Guilleminault C (2001) SF-36: evaluation of quality of life in severe and mild insomniacs compared with good sleepers. *Psychosom Med* 63: 49–55.

Lemoine P, Nir T, Laudon M and Zisapel N (2007) Prolonged-release melatonin improves sleep quality and morning alertness in insomnia patients aged 55 years and older and has no withdrawal effects. *J Sleep Res* 16: 372–380.

Lindberg N, Virkkunen M, Tani P, Appelberg B, Virkkala J, Rimon R, et al. (2002) Effect of a single-dose of olanzapine on sleep in healthy females and males. *Int Clin Psychopharmacol* 17: 177–184.

Lingford-Hughes AR, Welch S and Nutt DJ (2004) Evidence-based guidelines for the pharmacological management of substance misuse, addiction and comorbidity: recommendations from the British Association for Psychopharmacology. *J Psychopharmacol* 18: 293–335.

MacMahon KM, Broomfield NM and Espie CA (2005) A systematic review of the effectiveness of oral melatonin for adults (18 to 65 years) with delayed sleep phase syndrome and adults (18 to 65 years) with primary insomnia. *Curr Psychiatry Rev* 1: 103–113.

Manconi M and Ferini-Strambi L (2004) Restless legs syndrome among pregnant women. *Sleep* 27: 350.

Massironi G, Galluzzi S and Frisoni GB (2003) Drug treatment of REM sleep behavior disorders in dementia with Lewy bodies. *Int Psychogeriatr* 15: 377–383.

Markantonis SL, Tsakalozou E, Paraskeva A, Staikou C, Fassoulaki A (2008) Melatonin pharmacokinetics in premenopausal and postmenopausal healthy female volunteers. *J Clin Pharmacol* 48: 240–245.

Mayer G, Wang-Weigand S, Roth-Schechter B, Lehmann R, Staner C and Partinen M (2009) Efficacy and safety of 6-month nightly ramelteon administration in adults with chronic primary insomnia. *Sleep* 32: 351–360.

Mayers AG and Baldwin DS (2005) Antidepressants and their effect on sleep. *Hum Psychopharmacol* 20: 533–559.

Mellinger GD, Balter MB and Uhlenhuth EH (1985) Insomnia and its treatment. Prevalence and correlates. *Arch Gen Psychiatry* 42: 225–232.

Melton ST, Wood JM and Kirkwood CK (2005) Eszopiclone for insomnia. *Ann Pharmacother* 39: 1659–1666.

Mendlewicz J (2009) Sleep disturbances: Core symptoms of major depressive disorder rather than associated or comorbid disorders. *World J Biol Psychiatry* 10: 269–275.

Merdink JL, Robison LM, Stevens DK, Hu M, Parker JC, Bull RJ (2008) Kinetics of chloral hydrate and its metabolites in male human volunteers. *Toxicology* 245: 130–140.

Merenstein D, Diener-West M, Halbower AC, Krist A and Rubin HR (2006) The trial of infant response to diphenhydramine: the TIRED study – a randomized, controlled, patient-oriented trial. *Arch Pediatr Adolesc Med* 160: 707–712.

Mindell JA, Kuhn B, Lewin DS, Meltzer LJ and Sadeh A (2006) Behavioral treatment of bedtime problems and night wakings in infants and young children. *Sleep* 29: 1263–1276.

Montgomery P and Dennis J (2003) Cognitive behavioural interventions for sleep problems in adults aged 60+. *Cochrane Database Syst Rev* 1: CD003161.

Montgomery P, Stores G and Wiggs L (2004) The relative efficacy of two brief treatments for sleep problems in young learning disabled (mentally retarded) children: a randomised controlled trial. *Arch Dis Child* 89: 125–130.

Montgomery SA (1993) Guidelines for treating depressive illness with antidepressants. *J Psychopharmacol* 7: 19–23.

Morin CM, Belanger L, Bastien C and Vallieres A (2005a) Long-term outcome after discontinuation of benzodiazepines for insomnia: a survival analysis of relapse. *Behav Res Ther* 43: 1–14.

Morin CM, Belanger L, LeBlanc M, Ivers H, Savard J, Espie CA, et al. (2009a) The natural history of insomnia: a population-based 3-year longitudinal study. *Arch Intern Med* 169: 447–453.

Morin CM, Bootzin RR, Buysse DJ, Edinger JD, Espie CA and Lichstein KL (2006) Psychological and behavioral treatment of insomnia: update of the recent evidence (1998–2004). *Sleep* 29: 1398–1414.

Morin CM, Colecchi C, Stone J, Sood R and Brink D (1999) Behavioral and pharmacological therapies for late-life insomnia: a randomized controlled trial. *JAMA* 281: 991–999.

Morin CM, Koetter U, Bastien C, Ware JC and Wooten V (2005b) Valerian-hops combination and diphenhydramine for treating insomnia: a randomized placebo-controlled clinical trial. *Sleep* 28: 1465–1471.

Morin CM, Vallieres A, Guay B, Ivers H, Savard J, Merette C, et al. (2009b) Cognitive behavioral therapy, singly and combined with medication, for persistent insomnia: a randomized controlled trial. *JAMA* 301: 2005–2015.

Morphy H, Dunn KM, Lewis M, Boardman HF and Croft PR (2007) Epidemiology of insomnia: a longitudinal study in a UK population. *Sleep* 30: 274–280.

Murphy SM and Tyrer P (1991) A double-blind comparison of the effects of gradual withdrawal of lorazepam, diazepam and bromazepam in benzodiazepine dependence. *Br J Psychiat* 158: 511–516.

Nash JR, Wilson SJ, Potokar JP and Nutt DJ (2003) Mirtazapine induces REM sleep behavior disorder (RBD) in parkinsonism. *Neurology* 61: 1161.

Najib J (2006) Eszopiclone, a nonbenzodiazepine sedative-hypnotic agent for the treatment of transient and chronic insomnia. *Clinical Therapeutics* 28: 491–516.

Neckelmann D, Mykletun A and Dahl AA (2007) Chronic insomnia as a risk factor for developing anxiety and depression. *Sleep* 30: 873–880.

Neutel CI (1995) Risk of traffic accident injury after a prescription for a benzodiazepine. *Ann Epidemiol* 5: 239–244.

Nguyen BH, Perusse D, Paquet J, Petit D, Boivin M, Tremblay RE, et al. (2008) Sleep terrors in children: a prospective study of twins. *Pediatrics* 122: e1164–e1167.

NIH (1983) Drugs and Insomnia: The Use of Medications To Promote Sleep. *NIH Consensus Statement* 4(10): 1–19.

NIH (2005) National Institutes of Health State of the Science Conference statement on Manifestations and Management of Chronic Insomnia in Adults. *Sleep* 28: 1049–1057.

Nutt DJ (2005a) Death by tricyclic: the real antidepressant scandal? *J Psychopharmacol* 19: 123–124.

Nutt DJ (2005b) NICE: The National Institute of Clinical Excellence – or Eccentricity? Reflections on the Z–drugs as hypnotics. *J Psychopharmacol* 19: 125–127.

Nutt DJ and Stahl SM (2009) Searching for perfect sleep: the continuing evolution of GABAA receptor modulators as hypnotics. *J Psychopharmacol* [Epub ahead of print].

Nutt DJ, Fone K, Asherson P, Bramble D, Hill P, Matthews K, et al. (2007) Evidence-based guidelines for management of attention-

deficit/hyperactivity disorder in adolescents in transition to adult services and in adults: recommendations from the British Association for Psychopharmacology. *J Psychopharmacol* 21: 10–41.
Ohayon MM, Caulet M, Arbus L, Billard M, Coquerel A, Guieu JD, et al. (1999) Are prescribed medications effective in the treatment of insomnia complaints? *J Psychosom Res* 47: 359–368.
Owens JF and Matthews KA (1998) Sleep disturbance in healthy middle-aged women. *Maturitas* 30: 41–50.
Patat A, Paty I and Hindmarch I (2001) Pharmacodynamic profile of zaleplon, a new non-benzodiazepine hypnotic agent. *Hum Psychopharm* 16: 369–392.
Paterson LM, Wilson SJ, Nutt DJ, Hutson PH and Ivarsson M (2007) A translational, caffeine-induced model of onset insomnia in rats and healthy volunteers. *Psychopharmacology (Berl)* 191: 943–950.
Philip P, Léger D, Taillard J, Quera-Salva MA, Niedhammer I, Mosqueda JG, et al. (2006) Insomniac complaints interfere with quality of life but not with absenteeism: Respective role of depressive and organic comorbidity. *Sleep Med* 7: 585–591.
Pien GW and Schwab RJ (2004) Sleep disorders during pregnancy. *Sleep* 27: 1405–1417.
Porkka-Heiskanen T, Alanko L, Kalinchuk A and Stenberg D (2002) Adenosine and sleep. *Sleep Med Rev* 6: 321–332.
Pressman MR (2007) Factors that predispose, prime and precipitate NREM parasomnias in adults: clinical and forensic implications. *Sleep Med Rev* 11: 5–30.
Ramchandani P, Wiggs L, Webb V and Stores G (2000) A systematic review of treatments for settling problems and night waking in young children. *Br Med J* 320: 209–213.
Raskind MA, Peskind ER, Hoff DJ, Hart KL, Holmes HA, Warren D, et al. (2007) A parallel group placebo controlled study of prazosin for trauma nightmares and sleep disturbance in combat veterans with post-traumatic stress disorder. *Biol Psychiatry* 61: 928–934.
Rätz AE, Schlienger RG, Linder L, Langewitz W, Haefeli WE (1999) Pharmacokinetics and pharmacodynamics of clomethiazole after oral and rectal administration in healthy subjects. *Clin Ther* 21: 829–840.
Reid WH, Ahmed I and Levie CA (1981) Treatment of sleepwalking: a controlled study. *Am J Psychother* 35: 27–37.
Reynolds CF III, Buysse DJ, Miller MD, Pollock BG, Hall M and Mazumdar S (2006) Paroxetine treatment of primary insomnia in older adults. *Am J Geriatr Psychiatry* 14: 803–807.
Rhalimi M, Helou R and Jaecker P (2009) Medication use and increased risk of falls in hospitalized elderly patients: a retrospective, case-control study. *Drugs Aging* 26: 847–852.
Richardson GS, Zammit G, Wang-Weigand S and Zhang J (2009) Safety and subjective sleep effects of ramelteon administration in adults and older adults with chronic primary insomnia: a 1-year, open-label study. *J Clin Psychiatry* 70: 467–476.
Richelson E (1994) The pharmacology of antidepressants at the synapse: focus on newer compounds. *J Clin Psychiatry* 55(Suppl A): 34–39.
Riemann D (2009) Does effective management of sleep disorders reduce depressive symptoms and the risk of depression? *Drugs* 69(Suppl 2): 43–64.
Riemann D and Perlis ML (2009) The treatments of chronic insomnia: a review of benzodiazepine receptor agonists and psychological and behavioral therapies. *Sleep Med Rev* 13: 205–214.
Riemann D, Voderholzer U, Cohrs S, Rodenbeck A, Hajak G, Ruther E, et al. (2002) Trimipramine in primary insomnia: results of a polysomnographic double-blind controlled study. *Pharmacopsychiatry* 35: 165–174.
Rizzo V, Porta C, Moroni M, Scoglio E and Moratti R (2002) Determination of free and total (free plus protein-bound) melatonin in plasma and cerebrospinal fluid by high-performance liquid chromatography with fluorescence detection. *J Chromatogr B Analyt Technol Biomed Life Sci* 774: 17–24.
Rosen AS, Fournie P, Darwish M, Danjou P and Troy SM (1999) Zaleplon pharmacokinetics and absolute bioavailability. *Biopharm Drug Dispos* 20: 171–175.
Roth T, Rogowski R, Hull S, Schwartz H, Koshorek G, Corser B, et al. (2007) Efficacy and safety of doxepin 1 mg, 3 mg, and 6 mg in adults with primary insomnia. *Sleep* 30: 1555–1561.
Sack RL (2010) Clinical practice. Jet lag. *N Engl J Med* 362: 440–447.
Sack RL, Auckley D, Auger RR, Carskadon MA, Wright KP Jr, Vitiello MV, et al. (2007a) Circadian rhythm sleep disorders: part II, advanced sleep phase disorder, delayed sleep phase disorder, free-running disorder, and irregular sleep–wake rhythm. An American Academy of Sleep Medicine review. *Sleep* 30: 1484–1501.
Sack RL, Auckley D, Auger RR, Carskadon MA, Wright KP Jr, Vitiello MV, et al. (2007b) Circadian rhythm sleep disorders: part I, basic principles, shift work and jet lag disorders. An American Academy of Sleep Medicine review. *Sleep* 30: 1460–1483.
Salva P and Costa J (1995) Clinical pharmacokinetics and pharmacodynamics of zolpidem. Therapeutic implications. *Clin Pharmacokinet* 29: 142–153.
Samuels ER and Szabadi E (2008) Functional neuroanatomy of the noradrenergic locus coeruleus: its roles in the regulation of arousal and autonomic function Part II: Physiological and pharmacological manipulations and pathological alterations of locus coeruleus activity in humans. *Curr Neuropharmacol* 6: 254–285.
Sanna E, Busonero F, Talani G, Carta M, Massa F, Peis M, et al. (2002) Comparison of the effects of zaleplon, zolpidem, and triazolam at various GABA(A) receptor subtypes. *Eur J Pharmacol* 451: 103–110.
Sansone RA and Sansone LA (2010) Is seroquel developing an illicit reputation for misuse/abuse? *Psychiatry (Edgmont)* 7: 13–16.
Saper CB, Scammell TE and Lu J (2005) Hypothalamic regulation of sleep and circadian rhythms. *Nature* 437: 1257–1263.
Sateia MJ, Doghramji K, Hauri PJ and Morin CM (2000) Evaluation of chronic insomnia. An American Academy of Sleep Medicine review. *Sleep* 23: 243–308.
Scharf M, Rogowski R, Hull S, Cohn M, Mayleben D, Feldman N, et al. (2008) Efficacy and safety of doxepin 1 mg, 3 mg, and 6 mg in elderly patients with primary insomnia: A randomized, double-blind, placebo-controlled crossover study. *J Clin Psychiatry* 69: 1557–1564.
Schenck C and Mahowald M (1996) Long-term, nightly benzodiazepine treatment of injurious parasomnias and other disorders of disrupted nocturnal sleep in 170 adults. *Am J Med* 100: 333–337.
Scherk H, Pajonk FG and Leucht S (2007) Second-generation antipsychotic agents in the treatment of acute mania: a systematic review and meta-analysis of randomized controlled trials. *Arch Gen Psychiatry* 64: 442–455.
Schnoes CJ, Kuhn BR, Workman EF and Ellis CR (2006) Pediatric prescribing practices for clonidine and other pharmacologic agents for children with sleep disturbance. *Clin Pediatr (Phila)* 45: 229–238.
Schweiger MS (1972) Sleep disturbance in pregnancy. A subjective survey. *Am J Obstet Gynecol* 114: 879–882.
Sellers EM, Lang-Sellers M, Koch-Weser J (1978) Comparative metabolism of chloral hydrate and triclofos. *J Clin Pharmacol* 18: 457–461.
Sharpley AL, Attenburrow ME, Hafizi S and Cowen PJ (2005) Olanzapine increases slow wave sleep and sleep continuity in SSRI-resistant depressed patients. *J Clin Psychiatry* 66: 450–454.
Sharpley AL, Vassallo CM and Cowen PJ (2000) Olanzapine increases slow-wave sleep: evidence for blockade of central 5-HT(2C) receptors in vivo. *Biol Psychiatry* 47: 468–470.

Shekelle PG, Woolf SH, Eccles M and Grimshaw J (1999) Developing clinical guidelines. *West J Med* 170: 348–351.

Shepertycky MR, Banno K and Kryger MH (2005) Differences between men and women in the clinical presentation of patients diagnosed with obstructive sleep apnea syndrome. *Sleep* 28: 309–314.

Shirani A and St Louis EK (2009) Illuminating rationale and uses for light therapy. *J Clin Sleep Med* 5: 155–163.

Short CA and Carpenter PK (1998) The treatment of sleep disorders in people with learning disabilities using light therapy. *Int J Psychiatry Clin Pract* 1: 143–145.

Simon GE and VonKorff M (1997) Prevalence, burden, and treatment of insomnia in primary care. *Am J Psychiatry* 154: 1417–1423.

Sivertsen B, Krokstad S, Mykletun A and Overland S (2009) Insomnia symptoms and use of health care services and medications: the HUNT-2 study. *Behav Sleep Med* 7: 210–222.

Skene DJ and Arendt J (2007) Circadian rhythm sleep disorders in the blind and their treatment with melatonin. *Sleep Med* 8: 651–655.

Skene DJ, Lockley SW and Arendt J (1999) Melatonin in circadian sleep disorders in the blind. *Biol Signals Recept* 8: 90–95.

Smits MG, van Stel HF, van der HK, Meijer AM, Coenen AM and Kerkhof GA (2003) Melatonin improves health status and sleep in children with idiopathic chronic sleep-onset insomnia: a randomized placebo-controlled trial. *J Am Acad Child Adolesc Psychiatry* 42: 1286–1293.

Soldatos CR, Dikeos DG and Whitehead A (1999) Tolerance and rebound insomnia with rapidly eliminated hypnotics: a meta-analysis of sleep laboratory studies. *Int Clin Psychopharmacol* 14: 287–303.

Staner L, Ertle S, Boeijinga P, Rinaudo G, Arnal MA, Muzet A, et al. (2005) Next-day residual effects of hypnotics in DSM-IV primary insomnia: a driving simulator study with simultaneous electroencephalogram monitoring. *Psychopharmacology (Berl)* 181: 790–798.

Strenkoski-Nix LC, Ermer J, DeCleene S, Cevallos W, Mayer PR (2000) Pharmacokinetics of promethazine hydrochloride after administration of rectal suppositories and oral syrup to healthy subjects American *Journal of Health-System Pharmacy* 57: 1499–1505.

Tauscher J, Jones C, Remington G, Zipursky RB, Kapur S (2002) Significant dissociation of brain and plasma kinetics with antipsychotics. *Mol Psych* 7: 317–321.

Trewin VF, Lawrence CJ and Veitch GB (1992) An investigation of the association of benzodiazepines and other hypnotics with the incidence of falls in the elderly. *J Clin Pharm Ther* 17: 129–133.

Unruh ML, Redline S, An MW, Buysse DJ, Nieto FJ, Yeh JL, et al. (2008) Subjective and objective sleep quality and aging in the sleep heart health study. *J Am Geriatr Soc* 56: 1218–1227.

Van der Heijden KB, Smits MG, Van Someren EJ, Ridderinkhof KR and Gunning WB (2007) Effect of melatonin on sleep, behavior, and cognition in ADHD and chronic sleep-onset insomnia. *J Am Acad Child Adolesc Psychiatry* 46: 233–241.

Varkevisser M, Van Dongen HP and Kerkhof GA (2005) Physiologic indexes in chronic insomnia during a constant routine: evidence for general hyperarousal? *Sleep* 28: 1588–1596.

Verster J, Veldhuijzen DS, Patat A, Olivier B and Volkerts ER (2006) Hypnotics and driving safety: Meta-analyses of randomised controlled trials applying the on-the-road driving test. *Curr Drug Saf* 1: 63–71.

Vgontzas AN, Bixler EO, Lin HM, Prolo P, Mastorakos G, Vela-Bueno A, et al. (2001) Chronic insomnia is associated with nyctohemeral activation of the hypothalamic–pituitary–adrenal axis: clinical implications. *J Clin Endocrinol Metab* 86: 3787–3794.

Vgontzas AN, Liao D, Bixler EO, Chrousos GP and Vela-Bueno A (2009) Insomnia with objective short sleep duration is associated with a high risk for hypertension. *Sleep* 32: 491–497.

Vgontzas AN, Tsigos C, Bixler EO, Stratakis CA, Zachman K, Kales A, et al. (1998) Chronic insomnia and activity of the stress system: a preliminary study. *J Psychosom Res* 45: 21–31.

Vignatelli L, Billiard M, Clarenbach P, Garcia-Borreguero D, Kaynak D, Liesiene V, et al. (2006) EFNS guidelines on management of restless legs syndrome and periodic limb movement disorder in sleep. *Eur J Neurol* 13: 1049–1065.

Voderholzer U, Riemann D, Hornyak M, Backhaus J, Feige B, Berger M, et al. (2001) A double-blind, randomized and placebo-controlled study on the polysomnographic withdrawal effects of zopiclone, zolpidem and triazolam in healthy subjects. *Eur Arch Psychiatry Clin Neurosci* 251: 117–123.

Voortman G, Paanakker JE (2004) Bioavailability of mirtazapine from Remeron® tablets after single and multiple oral dosing. *Hum Psychpharm* 10: S83.

Voshaar RC, Van Balkom AJ and Zitman FG (2004) Zolpidem is not superior to temazepam with respect to rebound insomnia: a controlled study. *Eur Neuropsychopharmacol* 14: 301–306.

Wade AG, Ford I, Crawford G, McMahon AD, Nir T, Laudon M, et al. (2007) Efficacy of prolonged release melatonin in insomnia patients aged 55–80 years: quality of sleep and next-day alertness outcomes. *Curr Med Res Opin* 23: 2597–2605.

Wagner J and Wagner ML (2000) Non-benzodiazepines for the treatment of Insomnia. *Sleep Medicine Reviews* 4: 551–581.

Walsh J, Fry JM, Erwin CW, Scharf M, Roth T and Vogel G (1998) Efficacy and safety of 14-day administration of zaleplon 5mg and 10mg for the treatment of primary insomnia. *Clin Drug Investig* 16: 354.

Walsh JK, Krystal AD, Amato DA, Rubens R, Caron J, Wessel TC, et al. (2007) Nightly treatment of primary insomnia with eszopiclone for six months: effect on sleep, quality of life, and work limitations. *Sleep* 30: 959–968.

Walsh JK, Pollak CP, Scharf MB, Schweitzer PK and Vogel GW (2000) Lack of residual sedation following middle-of-the-night zaleplon administration in sleep maintenance insomnia. *Clin Neuropharmacol* 23: 17–21.

Wassmer E, Carter PF, Quinn E, McLean N, Welsh G, Seri S, et al. (2001) Melatonin is useful for recording sleep EEGs: a prospective audit of outcome. *Dev Med Child Neurol* 43: 735–738.

Wiegand MH, Landry F, Bruckner T, Pohl C, Vesely Z and Jahn T (2008) Quetiapine in primary insomnia: a pilot study. *Psychopharmacology (Berl)* 196: 337–338.

Wiggs L and France K (2000) Behavioural treatments for sleep problems in children and adolescents with physical illness, psychological problems or intellectual disabilities. *Sleep Med Rev* 4: 299–314.

Wilson S and Argyropoulos S (2005) Antidepressants and sleep: a qualitative review of the literature. *Drugs* 65: 927–947.

Wilson SJ, Lillywhite AR, Potokar JP, Bell CJ and Nutt DJ (1997) Adult night terrors and paroxetine. *The Lancet* 350: 185.

Winokur A, DeMartinis NA III, McNally DP, Gary EM, Cormier JL and Gary KA (2003) Comparative effects of mirtazapine and fluoxetine on sleep physiology measures in patients with major depression and insomnia. *J Clin Psychiatry* 64: 1224–1229.

Woolcott JC, Richardson KJ, Wiens MO, Patel B, Marin J, Khan KM, et al. (2009) Meta-analysis of the impact of 9 medication classes on falls in elderly persons. *Arch Intern Med* 169: 1952–1960.

Young T, Rabago D, Zgierska A, Austin D and Laurel F (2003) Objective and subjective sleep quality in premenopausal, perimenopausal, and postmenopausal women in the Wisconsin Sleep Cohort Study. *Sleep* 26: 667–672.

Zammit GK, Weiner J, Damato N, Sillup GP and McMillan CA (1999) Quality of life in people with insomnia. *Sleep* 22(Suppl 2): S379–S385.

Zimmermann T, Wehling M, Schulz HU (1998) The relative bioavailability and pharmacokinetics of chloral hydrate and its metabolites. *Arzneimittelforschung* 48: 5–12 (in German).

Appendix

Suggested algorithm to screen for sleep disorder other than insomnia.
Ask the lead question, and then proceed with supplementary only if answer is 'yes'.

1. Narcolepsy

a. Do you sometimes fall asleep in the daytime completely without warning?

b. Is it literally impossible to resist 'sleep attacks' during the day?

c. Do you have collapses or extreme muscle weakness triggered by extreme emotion?

d. Do you have visual hallucinations, either just as you fall asleep or when you wake in the morning?

e. Are you paralysed and unable to move when you wake up from your sleep?

[Possible narcolepsy: 1a = "TRUE" AND (1b OR 1c OR 1d OR 1e = "TRUE")]

2. Sleep breathing disorder

a. Are you a very heavy snorer?

b. Does your partner say that you sometimes stop breathing?

c. Do you often wake up gasping for a breath?

d. Are you often excessively sleepy during the day or fall asleep without wanting to?

[Possible sleep breathing disorder: 2a = "TRUE" AND (2b OR 2c OR 2d = "TRUE")]

3. PLMS/ RLS

a. Do your legs often twitch or jerk or can't keep still in bed?

b. Is it very difficult to get to sleep because of repeated muscle jerks?

c. Do you frequently wake from sleep with sudden jerky movements or with a compulsion to move your legs?

d. Do you simply have to get out of bed and pace around to get rid of these feelings?

[Possible PLMS/ RLS: 3a = "TRUE" AND (3b OR 3c OR 3d = "TRUE")]

4. Circadian Rhythm Sleep Disorder

a. Do you tend to sleep well but just at the "wrong times"?

b. Can you sleep well enough, but only if you stay up very late?

c. Are you in a very sound sleep at normal waking time and could sleep on for hours more?

d. Can you sleep well enough, but only if you go to bed very early?

e. Do you wake very early, bright and alert and no longer sleepy?

[Possible CRSD: 4a = "TRUE" AND EITHER (4b AND 4c = "TRUE") OR (4d AND 4e = "TRUE")]

5. Parasomnia

a. Do you have unusual behaviours associated with your sleep that trouble you or that are dangerous?

b. Do you sleepwalk frequently and run the risk of injuring yourself or others?

c. Do you have frequent night terrors when you are extremely distressed but not properly awake?

d. Do you act out your dreams and risk injuring yourself or others?

e. Do you have terrible recurring nightmares?

[Possible parasomnia: 5a = "TRUE" AND EITHER (5b OR 5c OR 5d OR 5e = "TRUE")]

Reproduced with permission of Prof Colin A Espie, Glasgow

http://dx.doi.org/10.5665/sleep.1872

A Randomized, Placebo-Controlled Trial of Online Cognitive Behavioral Therapy for Chronic Insomnia Disorder Delivered via an Automated Media-Rich Web Application

Colin A. Espie, PhD[1,2,6]; Simon D. Kyle, PhD[1,2]; Chris Williams, MD[3]; Jason C. Ong, PhD[4]; Neil J. Douglas, MD, DSc[5]; Peter Hames, MA Oxon[6]; June S.L. Brown, PhD[7]

[1]University of Glasgow Sleep Centre, Glasgow, Scotland, UK; [2]Institute of Neuroscience & Psychology, University of Glasgow, Glasgow, Scotland, UK; [3]Institute of Health & Wellbeing, University of Glasgow, Glasgow, Scotland, UK; [4]Rush University Medical Center, Chicago, IL; [5]Department of Sleep Medicine, Edinburgh Royal Infirmary, Edinburgh, UK; [6]Sleepio Ltd., London, UK; [7]Institute of Psychiatry, London, UK

Study Objectives: The internet provides a pervasive milieu for healthcare delivery. The purpose of this study was to determine the effectiveness of a novel web-based cognitive behavioral therapy (CBT) course delivered by an automated virtual therapist, when compared with a credible placebo; an approach required because web products may be intrinsically engaging, and vulnerable to placebo response.

Design: Randomized, placebo-controlled trial comprising 3 arms: CBT, imagery relief therapy (IRT: placebo), treatment as usual (TAU).

Setting: Online community of participants in the UK.

Participants: One hundred sixty-four adults (120 F: [mean age 49y (18-78y)] meeting proposed DSM-5 criteria for Insomnia Disorder, randomly assigned to CBT (n = 55; 40 F), IRT placebo (n = 55; 42 F) or TAU (n = 54; 38 F).

Interventions: CBT and IRT each comprised 6 online sessions delivered by an animated personal therapist, with automated web and email support. Participants also had access to a video library/back catalogue of session content and Wikipedia style articles. Online CBT users had access to a moderated social network/community of users. TAU comprised no restrictions on usual care and access to an online sleep diary.

Measurements and Results: Major assessments at baseline, post-treatment, and at follow-up 8-weeks post-treatment; outcomes appraised by online sleep diaries and clinical status. On the primary endpoint of sleep efficiency (SE; total time asleep expressed as a percentage of the total time spent in bed), online CBT was associated with sustained improvement at post-treatment (+20%) relative to both TAU (+6%; *d* = 0.95) and IRT (+6%: *d* = 1.06), and at 8 weeks (+20%) relative to IRT (+7%: *d* = 1.00) and TAU (+9%: *d* = 0.69) These findings were mirrored across a range of sleep diary measures. Clinical benefits of CBT were evidenced by modest superiority over placebo on daytime outcomes (*d* = 0.23-0.37) and by substantial improved sleep-wake functioning on the Sleep Condition Indicator (range of *d* = 0.77-1.20). Three-quarters of CBT participants (76% [CBT] vs. 29% [IRT] and 18% [TAU]) completed treatment with SE > 80%, more than half (55% [CBT] vs. 17% [IRT] and 8% [TAU]) with SE > 85%, and over one-third (38% [CBT] vs. 6% [IRT] and 0% [TAU]) with SE > 90%; these improvements were largely maintained during follow-up.

Conclusion: CBT delivered using a media-rich web application with automated support and a community forum is effective in improving the sleep and associated daytime functioning of adults with insomnia disorder.

Keywords: Insomnia, sleep, treatment, psychological intervention, internet, web, online, rich media, application, app, animated, virtual, automated

Clinical Trial Registration: ISRCTN – 44615689.

Keywords: Sleep, psychological treatment, online, internet, virtual

Citation: Espie CA; Kyle SD; Williams C; Ong JC; Douglas NJ; Hames P; Brown JSL. A randomized, placebo-controlled trial of online cognitive behavioral therapy for chronic insomnia disorder delivered via an automated media-rich web application. *SLEEP* 2012;35(6):769-781.

INTRODUCTION

Sleep disturbance is the most common symptom of mental illness in the UK.[1] Worldwide, epidemiologic studies report the prevalence of a clinical insomnia disorder at 10% to 12%,[2,3] and longitudinal investigation has shown that, once established, insomnia disorder tends to persist.[4] Typically, insomnia is associated with increased fatigue, impaired work productivity, reduced quality of life and relationship satisfaction, as well as increased ill health.[5-9] Chronic insomnia may be a risk factor for the development of mental and physical health problems[10-13] and is possibly associated with all-cause mortality.[14-16] The importance of insomnia to public health is illustrated by national annual costs ($92 to $107 billion USD in USA),[17] and its cost per untreated case ($5,000 CAD in Canada).[18] Despite this, persistent insomnia often goes unrecognized, and care management is poorly developed.[19,20]

Benzodiazepine hypnotics and sedative antidepressants are commonly prescribed, although long-term outcome data are relatively sparse.[21,22] Whereas the benzodiazepine receptor agonists confer some advantages, there is limited evidence that they are preferable for chronic insomnia.[19,23] On the other hand, there is compelling evidence that cognitive behavioral therapy (CBT) is a lastingly effective treatment,[19,24-27] a good conceptual fit for psychological factors that commonly underlie insomnia,[5,28] and an approach that many patients prefer over a pharmacological one.[29,30]

There is support for CBT being made widely available[19,24-27,31,32]; however, the outstanding challenge is its inherent lack of scalability to meet population need.[33] This is not untypical of the broader population interest in solutions that

A commentary on this article appears in this issue on page 737.

Submitted for publication, January 2012
Submitted in final revised form March, 2012
Accepted for publication March, 2012

Address correspondence to: Colin A. Espie, University of Glasgow Sleep Centre, Sackler Institute of Psychobiological Research and Institute of Neuroscience & Psychology, College of Medical, Veterinary & Life Sciences, University of Glasgow, G51 4TF, Scotland; Tel: +44 (0)141 232 7860; Fax: +44 (0)141 232 7697; E-mail: Colin.Espie@glasgow.ac.uk

shifts the focus from the professional to the person and from the clinic to home implementation.[34] Traditionally, CBT is delivered face-to-face by a specialist psychological therapist, and so is dependent on a rare and expensive resource.[33,35] It seems inconceivable that any face-to-face therapy could replace, for example, the 12 million prescriptions for hypnotics that are written each year in England and Scotland for a combined population base of 47 million adults.[36,37] Moreover, "stepped care" models argue that expert professionals should consult on more complex cases rather than deal with routine referrals.[33,38] Although, there is evidence that nurses, trained to follow a CBT manual, can deliver effective treatment to small groups of patients[39-41] and that large community group interventions may also be feasible,[42] this approach is unlikely to offer a realistic alternative to prescribing because it too relies on regular contact, and with professionals whose duties are already many and varied.[33]

A review of 9 controlled studies, where written materials were distributed directly to people with insomnia, sometimes along with other media or telephone support, suggested some benefit, although effect sizes were generally small.[43] Six investigations of internet-based CBT offer encouraging results,[44-49] suggestive of the potential far-reaching benefits of this health technology. In perhaps the best-designed study, albeit on a small total sample (n = 45), a 16% improvement in sleep efficiency (SE: proportion of time in bed spent asleep) relative to baseline was observed following CBT (an absolute increase of 12% from pre- to post-treatment), compared with 3% (2% in absolute terms) in a wait-list group.[46] These data were mirrored by significant reductions in insomnia severity. Uncontrolled data also suggested gains were maintained. In the 2 largest studies,[47,49] significant effects of CBT over a wait-list condition were also encouraging, although limited to improvements in sleep quality and reductions in fatigue, rather than sleep parameters per se,[47] or showing small-to-moderate effects, with 49% dropout in the electronic CBT group.[49]

The literature on internet CBT for insomnia remains small and lacks a definitive study. In particular, a placebo-controlled trial is required if we are to be sure that reported improvements are not simply the result of a novel mode of treatment, participant enthusiasm, expectations, or experimental demand characteristics.[50] More than this, however, the intervention platforms evaluated so far, may not fully reflect the levels of sophistication that might be expected by contemporary internet users, for example, full web and mobile interactivity and the use of social networking.[51] Indeed, offering CBT within a supportive self-help environment may be crucial in helping people apply what they are learning.[52]

The objectives of this study were to address these scientific and technical imperatives by addressing the following questions: Is CBT for chronic insomnia disorder—delivered via an automated, media-rich web application—superior to a credible placebo intervention, as well as to a treatment as usual condition, in improving nighttime sleep and associated daytime functioning? Are these effects durable and clinically important?

METHODS

Participants from the UK community (18+ years), who had completed the online Great British Sleep Survey (GBSS), and who met proposed DSM-5 criteria for persistent Insomnia Disorder were invited to take part.[53,54] The GBSS utilized pre-established algorithms to screen for: (a) a current complaint of poor sleep (difficulty initiating and/or maintaining sleep, early morning wakening, or non-restorative sleep); with (b) significant daytime effects in ≥ 1 of 6 domains (fatigue, daytime sleepiness, cognitive impairment [e.g., concentration problems], mood disturbance, impaired occupational or academic functioning [e.g., poor productivity], impaired interpersonal/social functioning); and (c) affecting them ≥ 3 nights per week for ≥ 3 months.[55] People who reported being in "poor" or "very poor" physical or mental health, or who exceeded a threshold for other sleep disorders on our screening algorithm (published in Wilson et al.[24]) were excluded. Items from the AUDIT[56] and CAGE[57] were applied to identify heavy alcohol use, and cutoff scores on the Depression Anxiety Stress Scales provided supplementary data on mental health status.[58] The use of sleeping pills or other sleep aids was permitted. Usual care that participants had been receiving via their medical advisers, including medical prescriptions and any counselling or psychotherapy, continued in all arms. The website www.sleepio.com/research hosts illustrative material of the study evaluation and intervention procedures.

The GBSS was launched online in February 2010 by Sleepio Ltd (a company dedicated to helping people sleep better, through raising awareness, research, and the dissemination of behavioral treatment advice), in association with Boots UK (an international pharmacy-led health and beauty group) and the Mental Health Foundation (a leading UK mental health research, policy and service improvement charity). Growth was "organic in nature," driven for example by links to Boots WebMD (www.webmd.boots.com), Mental Health Foundation campaigning, and newspaper media exposure.

A total of 10,071 adults completed the GBSS from April 2010 to 25 February 2011, of whom 6,609 provided email addresses. In total, 1,342 of this latter group (20.3%) met preliminary screening criteria and were invited by email to consider taking part; of these, 276 (20.1%) replied and consented to further screening. The majority (n = 228, 82.6%) then completed further standardized assessments. Finally, to confirm current eligibility, participants completed prospectively a 7-day online sleep diary, during which they had to have a mean (baseline) sleep efficiency (SE) ≤ 79% to reflect a sleep problem of clinical severity on our primary outcome variable. Sixty-four participants were excluded during these final stages, and the remaining sample of 164 eligible, consenting participants was randomized (see participant flowchart: Figure 1).

In order to ensure real-world evaluation of the online intervention, participant enrolment was confined to email contact, and all eligibility and baseline data were automatically obtained without face-to-face verification. Ineligible participants were provided with a report comprising tailored sleep hygiene advice, and all participants were advised to contact their doctor if they had concerns about any aspect of their health. The website also hosted a list of telephone contact numbers for mental health helplines. Technical support to participants was provided by email contact or via the online community forum, where there was a dedicated discussion thread for identifying and resolving problems.

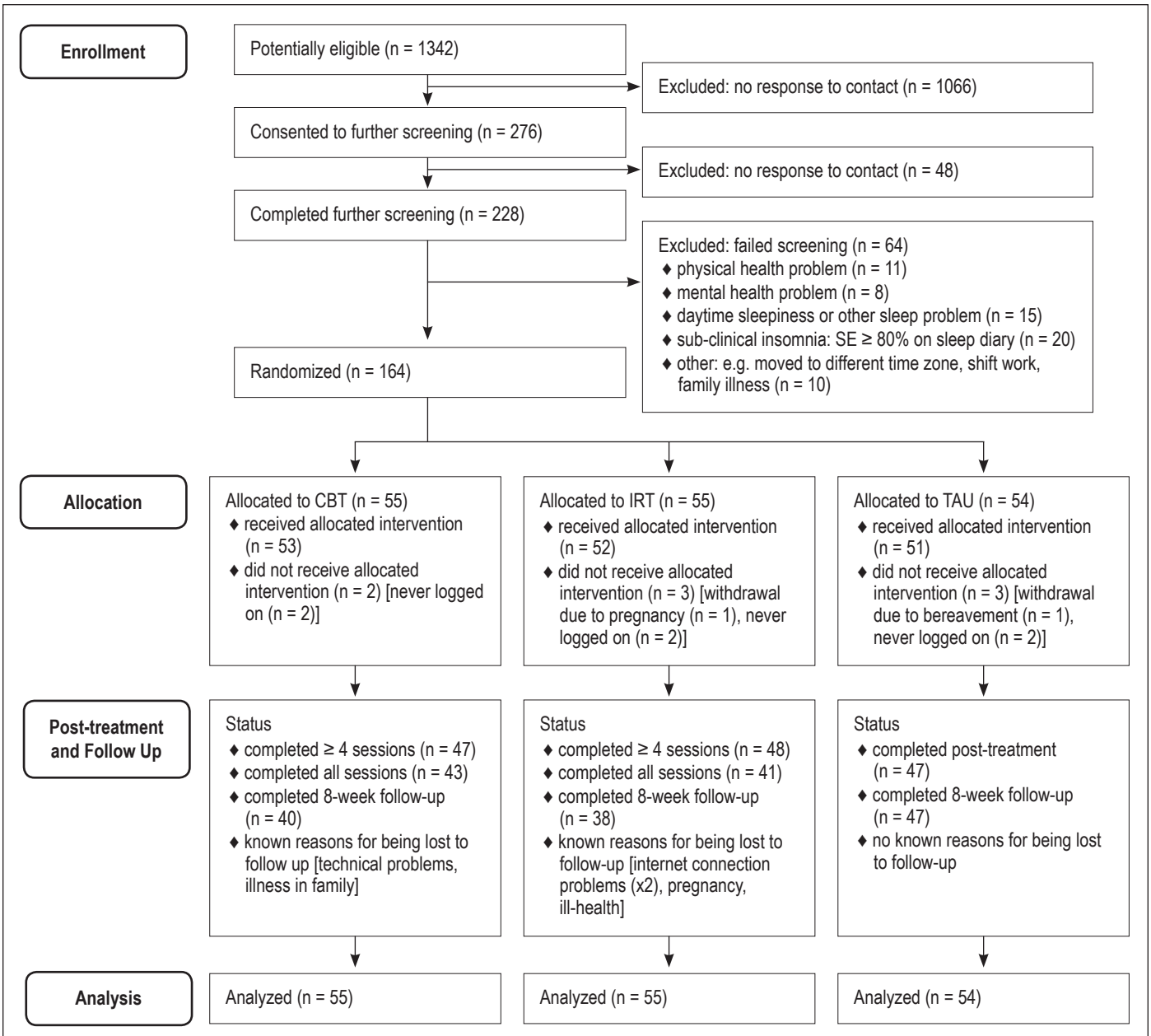


Figure 1—Flow of Participants in the Trial.

Study Design

The study was a pragmatic, parallel-group, randomized controlled trial comprising 3 treatment arms: (1) online CBT; (2) online imagery relief therapy (IRT: placebo); (3) treatment as usual (TAU), with blind assignment to group determined by a computer-generated random allocation schedule, operated by a remote independent technical operator. The trial followed CONSORT 2010 guidelines.[59] Consistent with the inclusion/exclusion criteria, the study design in effect was CBT+TAU v. IRT+TAU v. TAU alone. Major assessments were at baseline, post-treatment, and follow-up 8 weeks later. Participants randomized to the IRT placebo or TAU arm were offered the online CBT package upon completion of the study. All assessment, treatment, and data-gathering procedures were conducted online, and all queries/enquiries were managed electronically. These procedures ensured that the trial was genuinely an evaluation of a completely online CBT approach. The study protocol was approved by the University of Glasgow, Faculty of Medicine Research Ethics Committee and all participants provided informed consent online (see www.sleepio.com/research).

Assessment Measures

Participants accessed an online daily sleep diary throughout the study, to be completed each morning upon rising. They could set automated SMS (mobile text message) and/or email prompts as reminders. Such diaries are the staple tool of insomnia assessment.[60-62] Participants completed items by selecting from a drop-down menu of possible values. "How long did it take you to fall asleep last night" and "how long were you awake in total last night due to wakenings after you first fell asleep" (each variable offered as 0 min, 5 min, 10 min, 15 min, 30 min, and so on, thereafter in 15-min increments) assessed the central insomnia dimensions of difficulty initiating sleep (sleep onset latency [SOL], min) and difficulty

maintaining sleep (wake time after sleep onset [WASO], min). The diary also enquired about bedtime and rising time, from which total time in bed (TIB), and thence sleep efficiency (SE, %) were calculated; [1 – (SOL + WASO / TIB)] × 100. Total Sleep Time (TST, h) was also estimated from diary data [TIB – (SOL + WASO)]. A sensitive rating of "overall sleep quality" was obtained by dragging a slider along a dimensional analogue scale (0–100) between the poles of "very unsatisfactory" and "very satisfactory."

Diary data yielded the dependent variables of SOL, WASO, SE, TST, and sleep quality, averaged across 7 nights for each of the 3 major assessment points: baseline, post treatment, and follow-up (see www.sleepio.com/research). The primary study endpoint was change in SE from pre- to post-treatment, and from pre-treatment to follow-up for 2 reasons. First, sleep efficiency provides an overall index of insomnia by capturing both difficulties getting to sleep and staying asleep, and so was relevant for all participants of whatever insomnia subtype; second, endpoints relating to achievement of sleep efficiencies of 80%, 85%, and 90% reflect clinically important improvement and not merely statistical change.[63]

Importantly, because Insomnia Disorder must incorporate defined consequences, 6 domains of daytime function recommended by DSM-5 (energy, relationships, mood, concentration, productivity, sleepiness) were rated on a 5-point scale at each assessment phase (0 = not at all affected through to 4 = very much affected). Principal components analysis of data from our UK sample (n = 11,129) suggests that these items load ($r \geq 0.64$) on 2 factors: "daytime performance," comprising concentration, productivity, and sleepiness ratings (64.9% of the variance); and "social functioning," comprising ratings on mood, relationships, and energy (12.0% of the variance).[55] Accordingly, we used these 2 factor scores to evaluate daytime impact of treatment.

The Sleep Condition Indicator (SCI) is a new patient-reported outcome measure, specifically based upon DSM-5 Insomnia Disorder criteria.[53,54] It is brief (8-item) and has shown preliminary reliability in a field study of 11,129 participants ($\alpha = 0.894$, range of α-if-items systematically deleted = 0.877–0.898).[55] The SCI generates scores in the range 0 to 10, with higher values reflecting a person's sleep being in "better condition." SCI ≤ 5.9 identifies 95.4% of people with insomnia disorder, whereas a score ≥ 6.0 correctly identifies 76.8% of individuals without insomnia disorder.[55] The SCI also correlates with the Pittsburgh Sleep Quality Index[64] ($r = -0.78$, $n = 256$) and the Insomnia Severity Index[65] ($r = -0.79$, $n = 256$).[66] The SCI provided a secondary, clinically focused outcome measure for the study.

Finally, we selected the Depression Anxiety Stress Scale (DASS: 21 items)[58] because it takes a dimensional view of symptoms, including stress, which often co-present with insomnia. Internal consistency for the DASS is satisfactory ($\alpha = 0.82$-0.93). Items from the Sleep Disturbance Questionnaire (SDQ)[67,68] and the Glasgow Content of Thoughts Inventory (GCTI)[69] were also completed to inform CBT treatment algorithms.

The integrity of all data was assured by the online acquisition system and supporting software application. Clear guidance was provided including pop-up "tool tip" explanations for many items to ensure that they were correctly understood. All data entries were time stamped for all participants for the duration of the online course.

Treatment Groups

CBT

Participants received 6 weekly sessions delivered by an animated "virtual therapist" (The Prof). The program comprised a fully automated media-rich web application, driven dynamically by baseline, adherence, performance, and progress data. At the start of each session, The Prof conducted a progress review with the participant, explored the diary data submitted during the week, the participant's current sleep status and pattern, and progress achieved against goals previously set. Underlying algorithms fed the delivery of information, support, and advice in a personally tailored manner. CBT content was consistent with the literature,[60] and covered behavioral (e.g., sleep restriction, stimulus control) and cognitive (e.g., putting the day to rest, thought re-structuring, imagery, articulatory suppression, paradoxical intention, mindfulness) strategies, as well as additional relaxation strategies (progressive muscle relaxation and autogenic training) and advice on lifestyle and bedroom factors (sleep hygiene). The intervention was based upon a previously validated manual.[39-41] The following illustrations may be helpful. In sleep restriction, The Prof proposes a new "window" for sleep, calculated from available sleep diary data, and engages with the participant to help them select the timing (onset/offset) of this window from a set of personalized options. An example of a cognitive technique, is where another animated character (with insomnia) presents to the Prof their concerns, dysfunctional beliefs, and associated emotions. The Prof then asks the participant to choose some solutions from a menu of options and delivers this as advice to the character, who is seen to revise his thinking. The Prof then reveals to the participant that the scenario was based upon his/her own sleep-related attributions and thoughts (from baseline SDQ and GCTI data). In this way the participant is helped to learn how to restructure dysfunctional thinking. Table 1 summarizes the content and features of the intervention, permitting comparison of CBT with IRT and TAU conditions (with further illustration available at http://www.sleepio.com/research).

IRT

Imagery relief therapy was also delivered by The Prof, using the same application platform, and design and execution principles as for CBT, but with no known active therapeutic ingredient. IRT was based on a well-established and credible non-pharmacological placebo intervention[50] used in several clinical trials.[70-72] The term imagery relief therapy was selected to enhance credibility of an active and novel therapy. For example, if participants were to enter this as an internet search term, they would come upon material which would appear to be valid for a psychological problem such as sleep. However, IRT contained no active components of imagery training, or of systematic desensitization. Likewise, it did not include detailed relaxation instruction or behavioral advice about what to do during the sleep period. The participant was trained to visualize neutral objects (e.g., a key) or shapes (e.g., a yellow square) in conjunction with thinking about sequential aspects of their eve-

Table 1—Summary of treatment conditions

	CBT	IRT	TAU
Treatment content	Sleep information/education, sleep hygiene, relaxation, stimulus control, sleep restriction, cognitive techniques (restructuring, paradox, mindfulness, imagery, putting day to rest, thought stopping)	Sleep information/education, hierarchy development, imagery training, scheduled pseudo-desensitization, breathing control	N/A [Advised that most effective treatment would be offered upon completion of trial]
Duration	6 sessions over minimum of 6 weeks	6 sessions over minimum of 6 weeks	[N/A] Diary keeping only
Delivery context	Fully online Delivered by animated therapist (The Prof) No face to face contact	Fully online Delivered by animated therapist (The Prof) No face to face contact	Fully online diary recording only No face to face contact
Additional treatment features	Appointment system, interactive sessions, dynamic feedback against personal goals, progress review at start of each session, automatic calculation of sleep data over time, personal case file, end of session quiz, 24/7 access	Appointment system, Interactive sessions, dynamic feedback against personal goals, progress review at start of each session, automatic calculation of sleep data over time, personal case file, 24/7 access	N/A
Support/ motivational system	Praise/reinforcement contingent on progress, online Wikipedia of sleep educational topics Social community of users, moderated by experts Support/prompts/reminders by email and mobile SMS 'Graduation ceremony' on course completion	Praise/reinforcement contingent on progress, online Wikipedia of sleep educational topics Support/prompts/reminders by email and mobile SMS 'Graduation ceremony' on course completion	Email support only

ning routine (e.g., setting the table for dinner), and was asked to practice these pairings for 20 min/day early in the evening. The rationale for this "quasi-desensitization" framework was that successful sleep was associated with good preparation, and that neutralizing unhelpful associations with evening routines would recondition them towards automatic sleep engagement and sleep maintenance. IRT participants also received e-mail reminders from The Prof and had access to Wikipedia-style articles on sleep, its functions, and its disorders.

Protocol standardization

The integrity and fidelity of treatment allocations was assured by the online procedures which delivered the interventions. In addition, in session 1 of both CBT and IRT conditions, participants were invited to commit (or not) to the course following an explanation of the therapeutic rationale (all did so). CBT and IRT participants did not have contact with each other, nor did they have access to alternative treatment materials. Both CBT and IRT were scripted and automated, so support and length of treatment was similar. All web-based interactions were electronically stored to provide time-stamped data on participant activity (e.g., diary entries, session activities, engagement with the community, adherence to tasks).

TAU alone

In real world practice, insomnia patients often have some concurrent physical and psychological symptoms, as well as concurrent treatments. Therefore, to reflect validity, and to permit greater generalizability of findings, the protocol explicitly permitted continuation of treatment as usual health care for all participants. Physicians were free to offer appointments, to prescribe, and to maintain/discontinue prescriptions. Aside from this, TAU alone participants comprised, effectively, a wait-list group who completed measures but received no additional help for their insomnia. The only contact received by the TAU arm was reminders to complete evaluations. After the trial was completed TAU and IRT participants were offered access to the CBT intervention.

Data Management and Analysis

The study was designed to have 80% power to detect a medium effect size (Cohen[73]; consistent with published meta-analytic data[27]), based upon a 3-group ANOVA model with fixed effects, main effects, and interactions, on our primary outcome measure of SE. These criteria implied recruiting a total sample of 159 participants. All comparisons were planned and tests were 2-sided, with $P < 0.05$ considered to indicate statistical significance. Where appropriate, to control for multiple comparisons, a per family error rate was adopted to maintain the nominal error rate (0.05/n of comparisons). Analyses were performed using PASW Statistics 18 (SPSS Inc., Chicago, IL).[74] Linear mixed effects models were used, to avoid imputation of missing data (estimated at 16.1% of those commencing the trial at post-treatment and 19.9% at follow-up), predicting mean values at each assessment point (baseline, post-treatment, 8-week follow-up) and to test our hypotheses with respect to between group differences. In each model, time and treatment group were included as fixed effects, with time and group × time interaction terms. For variables exhibiting between-group differences at baseline, the baseline value was entered as a covariate. Clinical response to treatment was evaluated on an intention to

treat basis in relation to proportions of participants achieving the clinical endpoints of 80%, 85%, and 90% for SE at post-treatment and follow-up.

RESULTS

Participant Characteristics

Information on the allocated sample of 164 (120 F) adults (mean age 49 y [18–78y]) is provided in Table 2. Approximately two-thirds were employed either full-time or part-time. Post-code data provided a proxy for socioeconomic status by deriving an index of multiple deprivation (IMD). Mean IMD was 16.7 (SD 11.8); somewhat less deprived (by < 0.5 SD) than national norms (21.7 [SD 15.5]).[75] Participants were in at least average health (as per selection criteria), although around 30% and 10%, respectively, took medication for a physical or mental health problem. One in 5 participants sometimes used prescribed sleeping pills, and 40% made some use of over-the-counter (OTC) sleep aids. Twenty-nine people provided additional free text on the strategies they used to manage their insomnia. Much of this was amplification on their medication or OTCs; however, 9 described using relaxation, meditation, or yoga, and 4 used devices (e.g., ear plugs, a heat pad).

All participants had DSM-5 Insomnia Disorder, the great majority being of difficulty maintaining sleep or of mixed subtypes. Two-thirds had had insomnia > 6 years, and almost 50% for > 11 years. Participants randomized to CBT (n = 55), IRT (n = 55), and TAU (n = 54) were similar in all demographic and clinical respects. Eight of the 164 participants did not start their treatment, so the final sample receiving the allocated intervention was 156 (CBT = 53, IRT = 52, TAU = 51). There were no significant differences on any variable between this sample and the allocated sample of 164.

Treatment Attrition and Integrity

Of those receiving their allocated intervention, 43 CBT participants (82%) completed all their online therapy sessions, and 47 (88%) completed ≥ 4 sessions. This compared to 41 (79%) and 48 (92%), respectively, in the IRT group. Thus, there were similar modest levels of attrition during the treatment phase. Just under 80% (n = 125) completed follow-up assessment, comprising similar proportions of CBT and IRT, but a higher proportion of TAU (CBT [n = 40, 75%]; IRT [n = 38, 73%]; TAU [n = 47, 92%]). This was perhaps due to the latter group's anticipation of receiving active intervention immediately thereafter, as per provision of ethical approval. Reasons for withdrawal during treatment are summarized (where known) in Figure 1. There were no significant differences between treatment completers (defined as completing ≥ 4 sessions) and those who dropped out, on any variable. No harm-related or serious adverse events were reported.

Participants in the CBT group took an average of 50 days to complete the course compared with 48 days in IRT, with TAU participants typically taking one further week (58 days). Participants were generally adherent in completion of diaries. The study generated 11,278 daily diary records, of which only 239 (2.2%) had to be estimated by participants, upon prompting by The Prof, at the weekly progress review point.

Baseline Characteristics

Baseline data indicated current insomnia in the severe clinical range, with an average total wake time (SOL + WASO) of 136.5 min (SD 72.5) and mean self-reported SE of 61.3% (SD 16.2) for the sample as a whole (Table 3). Average estimated TST was 5.09 h (SD 1.47). On the SCI, the overall mean score of 2.98 (SD 1.04) was > 4 SD below the mean for good sleepers, based on our UK sample.[55] Consistent with diagnostic criteria, substantial impact was observed on daytime performance and social functioning. In descending rank order of mean (SD) impact at baseline, insomnia had a negative effect on energy (2.71 [0.80]), mood (2.50 [0.93]), concentration (2.40 [0.92]), productivity (2.12 [0.92]), relationships (1.72 [1.06]), and staying awake (1.28 [1.00]). There was modest symptomatology on the DASS, consistent with selection criteria, with stress scores significantly higher than depressive (7.80 [3.70] vs 5.05 [3.01], $t_{163} = 11.1$, $P < 0.001$) or anxiety (2.70 [2.20], $t_{163} = 21.0$, $P < 0.001$) scores, and depressive scores higher than anxiety scores ($t_{163} = 11.5$, $P < 0.001$).

One-way ANOVA revealed differences in pre-treatment scores for SOL, SE, and TST (all $P < 0.01$), in each case accounted for by the TAU group having more symptomatic scores (see Table 3). Consequently, baseline values were introduced conservatively as covariates in subsequent hypothesis testing on these variables.

Impact of Treatment on Self-Reported Sleep

Summary sleep diary data comprising pre-treatment, post-treatment and follow-up actual mean (SE) values for each group are presented in Table 3. Change scores (with 95% CI) and within group effect sizes [ES: $(M_1\text{-}M_2/\delta_{pooled})$] are also provided. ES were regarded as large ($d = 0.8$), moderate ($d = 0.5$), or small ($d = 0.3$), consistent with recognized definitions.[73] In Table 4, relative ES, representing changes over baseline observed at post-treatment and follow-up, are provided for each comparison (CBT-TAU, IRT-TAU, CBT-IRT). For all variables, significant effects in favor of CBT were observed, and these remained significant when taking account of baseline values.

CBT was associated with an absolute post-therapy increase of 19.5% (95%CI, 15.3 to 23.7) in SE (a 30.8% increase over baseline), compared with a 5.7% (95%CI, 2.79 to 8.52) gain following IRT, and 6.4% (95%CI, 2.88 to 9.86) in TAU (Table 3). A near 20% level of improvement was sustained in the CBT group at follow-up (95%CI, 15.7 to 23.6), compared with 7% (95%CI, 4.53 to 10.1) and 9% (95%CI, 4.89 to 13.7) gains in IRT and TAU. The mixed effects model confirmed a main effect for time ($F_{2,151} = 92.54$, $P < 0.0001$) and a significant treatment × time interaction ($F_{4,304} = 15.97$, $P < 0.0001$), with between-group comparisons favoring CBT at post-treatment relative to both TAU ($d = 0.95$) and IRT ($d = 1.06$), each representing large ES (Table 4). At follow-up, CBT again yielded superior outcome relative to IRT ($d = 1.00$) and TAU ($d = 0.69$).

Substantial reductions in SOL and WASO were observed in the CBT group, of around 26 min and 48 min, respectively, at both post-treatment and follow-up (see Table 3 for detailed data). By comparison, a more modest (20 min) but sustained reduction in WASO (only) was observed following IRT. TAU participants reduced their SOL by around 10 min. Mixed model analysis supported the superiority of CBT relative to TAU and

Table 2—Demographic and clinical characteristics of participants (n = 164)

Characteristic	CBT (n = 55)	IRT (n = 55)	TAU (n = 54)	All (n = 164)
Age, mean (SD), y	50.7 (13.8)	47.3 (13.0)	49.1 (13.7)	49.0 (13.5)
Gender, No. (%)				
Female	40 (72.7)	42 (76.4)	38 (70.4)	120 (73.2)
Male	15 (27.3)	13 (23.6)	16 (29.6)	44 (26.8)
Occupation, No. (%)				
Employed. Full-time	20 (36.4)	25 (45.5)	20 (37.0)	65 (39.6)
Employed, part-time	17 (30.9)	12 (21.8)	11 (20.4)	40 (24.4)
Retired	13 (23.6)	8 (14.5)	16 (29.6)	37 (22.6)
Student	3 (5.45)	3 (5.45)	2 (3.70)	8 (4.88)
Not currently employed	2 (3.64)	7 (12.7)	5 (9.26)	14 (25.5)
Index of multiple deprivation, mean (SD)[a]	16.7 (11.3)	18.4 (13.8)	15.2 (10.4)	16.7 (11.8)
Civil status, No. (%)[b]				
No Partner	20 (36.4)	20 (36.4)	missing	missing
Partner	35 (63.6)	35 (63.6)	missing	missing
Physical health, No. (%)				
0 Very good	15 (27.3)	13 (23.6)	7 (13.0)	35 (21.3)
1 Good	25 (45.4)	26 (47.3)	37 (68.5)	88 (53.7)
2 Average	15 (27.3)	16 (29.1)	10 (18.5)	41 (25.0)
Mental health, No. (%)				
Very good	16 (29.1)	11 (21.0)	13 (24.1)	40 (24.4)
Good	25 (45.4)	23 (41.8)	28 (51.8)	76 (46.3)
Average	14 (25.5)	21 (38.2)	13 (24.1)	48 (29.3)
Prescriptions for physical health, No. (%)				
Yes	18 (32.7)	14 (25.5)	18 (33.3)	50 (30.5)
No	37 (67.3)	41 (74.5)	36 (66.7)	114 (69.5)
Prescriptions for mental health, No. (%)				
Yes	4 (7.3)	7 (87.3)	6 (11.1)	17 (10.4)
No	51 (92.7)	48 (12.7)	48 (88.9)	147 (89.6)
Prescribed sleeping pills, No. (%)				
Yes	10 (18.2)	8 (14.5)	15 (27.8)	33 (20.1)
No	45 (81.8)	47 (85.5)	39 (72.2)	131 (79.9)
Over the counter sleep aids, No. (%)				
Yes	24 (43.6)	20 (36.4)	19 (35.2)	63 (38.4)
No	31 (56.4)	35 (63.6)	34 (64.8)	101 (61.6)
Duration of insomnia, No. (%), y				
< 2	9 (16.4)	7 (12.7)	6 (11.1)	22 (13.5)
2-5	13 (23.6)	16 (29.1)	5 (9.2)	34 (20.7)
6-10	10 (18.2)	13 (23.6)	9 (16.7)	32 (19.5)
≥ 11	23 (41.8)	19 (34.6)	34 (63.0)	76 (46.3)
Type of insomnia. No. (%)				
Difficulty Initiating Sleep	1 (1.8)	3 (5.4)	1 (1.9)	5 (3.0)
Difficulty Maintaining Sleep	26 (47.3)	22 (40.0)	22 (40.7)	70 (42.7)
Mixed (Initiating/Maintaining)	22 (40.0)	24 (43.6)	26 (48.1)	72 (43.9)
Early Morning Awakening	4 (7.3)	3 (5.5)	3 (5.6)	10 (6.1)
Non-Restorative Sleep	2 (3.6)	3 (5.5)	2 (3.7)	7 (4.3)

[a]These data available only for postcodes in England (n = 137). [b]These data were not collected, in error, from the TAU group.

IRT across time points for both SOL ($F_{4,304}$ = 4.55, P < 0.001) and WASO ($F_{4,306}$ = 8.53, P < 0.0001). At both time points for WASO, CBT exhibited a large ES relative to TAU (d = −0.77), and a moderate to large ES relative to IRT (d = −0.41). For SOL, there was a large effect in favor of CBT relative to IRT (d = −0.86) and a modest ES relative to TAU (d = −0.45). At follow-up, IRT was associated with lower WASO than TAU (d = −0.41). To further quantify these medium term improvements in sleep continuity, TWT reduced by some 75 min (95%CI, −56.8 to −92.7 min) following CBT, exhibiting large

Table 3—Treatment outcomes for sleep and daytime measures. Baseline, post-treatment, and follow-up data (actual mean [SE]) are presented for each group along with change scores (95% CI) and within group effect sizes (Cohen's *d*)

Treatment Group	Baseline Mean (SE)	Post-treatment Mean (SE)	Change from Baseline to Post-Treatment (95% CI)	*d*	8-wk Follow-up Mean (SE)	Change from Baseline to Follow-up (95% CI)	*d*
Sleep Efficiency, %							
CBT	63.2 (2.10)	82.7 (1.74)	19.5 (15.3 to 23.7)	1.28	82.8 (1.10)	19.6 (15.7 to 23.6)	1.37
IRT	65.1 (1.28)	70.8 (1.70)	5.70 (2.79 to 8.52)	0.55	72.4 (1.63)	7.29 (4.53 to 10.1)	0.73
TAU	55.6 (2.90)	62.0 (2.51)	6.37 (2.88 to 9.86)	0.51	64.9 (2.51)	9.30 (4.89 to 13.7)	0.59
Sleep Onset Latency, min							
CBT	47.9 (5.52)	21.5 (3.12)	-26.2 (-16.0 to -36.4)	-0.71	21.3 (2.12)	-26.6 (-17.5 to -35.7)	-0.80
IRT	48.0 (4.04)	48.0 (4.85)	-0.08 (6.37 to -6.22)	0.00	45.5 (3.96)	-2.50 (3.42 to -8.42)	-0.12
TAU	75.5 (10.1)	65.0 (8.37)	-10.5 (-1.10 to -19.8)	-0.31	62.8 (6.86)	-12.7 (0.53 to -25.9)	-0.27
Wake Time After Sleep Onset, min							
CBT	76.9 (6.49)	28.5 (4.34)	-48.4 (-35.5 to – 61.3)	-1.03	28.8 (3.22)	-48.1 (-35.4 to -60.9)	-1.04
IRT	75.0 (5.10)	54.8 (5.44)	-20.2 (-11.7 to – 28.7)	-0.66	53.1 (5.18)	-21.9 (-13.6 to -30.1)	-1.01
TAU	87.1 (7.95)	79.5 (7.40)	-7.56 (1.06 to -16.2)	-0.25	90.6 (4.15)	3.54 (26.9 to -19.8)	0.04
Total Wake Time, min							
CBT	124.8 (8.86)	50.0 (6.26)	-74.8 (-56.8 to -92.7)	-1.15	50.1 (3.70)	-74.7 (-57.7 to -91.8)	-1.21
IRT	122.9 (5.71)	102.8 (7.29)	-20.1 (-9.61 to 30.6)	-0.53	98.5 (7.03)	-24.4 (-14.0 to -37.8)	-0.65
TAU	162.5 (13.44)	144.5 (11.31)	-18.0 (-3.16 to 32.8)	-0.34	153.4 (12.6)	-9.15 (-17.7 to -36.0)	-0.09
Total Sleep Time, h							
CBT	5.11 (0.18)	5.76 (0.18)	0.65 (0.36 to 0.94)	0.63	6.30 (0.14)	1.19 (0.89 to 1.50)	1.08
IRT	5.51 (0.14)	5.87 (0.18)	0.36 (0.02 to 0.69)	0.30	5.98 (0.15)	0.47 (0.24 to 0.71)	0.56
TAU	4.65 (0.26)	5.30 (0.24)	0.65 (0.31 to 0.99)	0.54	5.44 (0.24)	0.79 (0.39 to 1.19)	0.55
Sleep quality, 0–100 rating							
CBT	43.2 (2.00)	56.3 (2.62)	13.1 (8.38 to 17.8)	0.77	57.6 (2.25)	14.4 (10.6 to 18.3)	1.04
IRT	45.0 (1.57)	52.0 (1.75)	7.08 (2.87 to 11.3)	0.47	53.9 (1.66)	8.96 (5.46 to 12.5)	0.71
TAU	41.4 (1.19)	44.0 (2.44)	2.57 (-0.92 to 6.05)	0.21	46.0 (2.51)	4.57 (0.55 to 8.59)	0.32
Sleep Condition Indicator							
CBT	3.06 (0.14)	6.30 (0.33)	3.24 (2.64 to 3.83)	1.50	6.59 (0.33)	3.53 (2.91 to 4.13)	1.60
IRT	3.00 (0.13)	4.48 (0.24)	1.48 (1.07 to 1.89)	1.00	4.95 (0.26)	1.95 (1.38 to 2.44)	1.00
TAU	2.79 (0.16)	3.78 (0.26)	0.99 (0.57 to 1.42)	0.65	4.12 (0.28)	1.33 (0.77 to 1.78)	0.71
Impact on problems with daytime performance (concentration, productivity, staying awake: 3 × 0-4 ratings)							
CBT	6.01 (0.30)	3.60 (0.39)	-2.42 (-3.10 to -1.73)	-0.98	2.54 (0.42)	-3.47 (-3.98 to -2.22)	-1.29
IRT	5.87 (0.32)	4.23 (0.41)	-1.63 (-2.31 to -0.95)	-0.67	3.41 (0.38)	-2.46 (-3.20 to -1.86)	-1.19
TAU	5.59 (0.34)	4.92 (0.38)	-0.67 (-1.34 to -0.10)	-0.28	4.58 (0.40)	-1.01 (-2.23 to -3.19)	-0.45
Impact on problems with daytime social functioning (mood, relationships, energy: 3 × 0-4 ratings)							
CBT	6.74 (0.28)	3.70 (0.41)	-3.04 (-3.82 to -2.25)	-1.06	2.79 (0.43)	-3.95 (-2.73 to -5.00)	-1.37
IRT	7.21 (0.31)	5.15 (0.38)	-2.06 (-2.71 to -1.40)	-0.88	4.15 (0.37)	-3.06 (-2.10 to -3.90)	-1.10
TAU	7.02 (0.29)	5.20 (0.35)	-1.82 (-2.57 to -1.08)	-0.68	5.35 (0.39)	-1.67 (-0.99 to -3.20)	-0.74

Table 4—Relative effect sizes (Cohen's d) for each treatment group comparison (CBT-TAU, IRT-TAU, CBT-IRT) at post-treatment and follow-up for sleep and daytime variables

	Relative effect size (*d*) Pre-treatment to post-treatment			Relative effect size (*d*) Pre-treatment to 8-wk Follow-up		
Variable	CBT-TAU	IRT-TAU	CBT-IRT	CBT-TAU	IRT-TAU	CBT-IRT
Sleep Efficiency, %	0.95	-0.06	1.06	0.69	0.15	1.00
Sleep Onset Latency, min	-0.45	0.30	-0.86	0.34	-0.27	0.86
Wake Time After Sleep Onset, min	-1.03	-0.41	-0.71	-0.77	-0.41	-0.67
Total Wake Time, min	-0.96	0.05	-1.03	-0.81	-0.21	-0.98
Total Sleep Time, h	0.00	-0.24	0.26	0.32	-0.26	0.73
Sleep quality, 0–100 rating	0.71	0.33	0.37	0.70	0.32	0.41
Sleep Condition Indicator	1.20	0.33	0.95	1.11	0.34	0.77
Impact on social functioning, 0-4 rating	-0.44	-0.10	-0.37	-0.78	-0.53	-0.24
Impact on daytime performance, 0-4 rating	-0.72	-0.40	-0.32	-0.85	-0.72	-0.23

ES compared with either IRT (d = −0.98: 24 min; 95%CI, −9.61 to −30.6) or TAU (d = −0.81: 9 min; 95%CI , −3.16 to −32.8) ($F_{4,306}$ = 9.56, P < 0.001).

Mixed models analysis also revealed significant interaction effects on TST ($F_{4,304}$ = 2.81, P = 0.026). At post-treatment, TST was increased by approximately 40 min in both CBT and TAU compared with 20 min in IRT. However, by follow-up, TST had increased by 70 min in the CBT group compared with 28 min (d = 0.73) and 47 min (d = 0.32) in IRT and TAU, respectively. Self-reported sleep quality also increased to a greater extent in CBT than in either IRT or TAU ($F_{4,306}$ = 4.06, P = 0.003), with the latter comparison representing a moderate-large effect both at post-treatment and follow-up (d = 0.70). Applying a Bonferroni correction to maintain the 0.05 error rate across all sleep diary variables (adjusted P < 0.01) would result in the TST main effects (only) failing to attain statistical significance. It should be noted that, as for the primary outcome of SE, time main effects were observed for SOL, WASO, TST, and sleep quality (all P < 0.001) in addition to the interaction terms reported above.

Impact of Treatment on Daytime Functioning

Comparative data on daytime outcomes are presented in Table 3; inspection of which indicate that there was a main effect of time for daytime performance ($F_{2,151}$ = 63.47, P < 0.0001) and for social functioning: ($F_{2,151}$ = 91.98, P < 0.0001). Visual impression suggests that both the CBT and the IRT groups improved relative to TAU, and this was confirmed by interaction effects for daytime performance ($F_{4,316}$ = 5.73, P < 0.001) and social functioning ($F_{4,316}$ = 3.78, P = 0.005). In relation to daytime performance a moderate-large effect in favor of CBT had developed by post-treatment for the CBT-TAU (d = −0.72) comparison (Table 4), and this was consolidated by follow-up (d = −0.85). However, a moderate-large effect was also evident for IRT relative to TAU by this point (d = −0.40 to −0.72). The ES for the CBT-IRT contrast, therefore, is important and reveals a small additional ES benefit favoring CBT (d = −0.23 to −0.32). A similar pattern of results was obtained with the social functioning data (Table 4). DASS total score data also suggest some generalized impact of CBT on participants' (mild) symptoms of psychopathology, with small effects for the CBT-IRT comparison observed at post-treatment (d = −0.33) and follow-up (d = −0.28).

Clinical Effects of Treatment

The SCI exhibited > 2-fold sustained improvement following CBT, represented by large CBT-TAU effects at post-treatment (d = 1.20) and follow-up (d = 1.11: Table 4). The CBT-IRT comparison also yielded large ES at both time points (d = 0.65 and d = 0.77), and placebo demonstrated a small effect (d = 0.34) relative to TAU ($F_{4,316}$ = 12.22, P < 0.0001). In terms of clinically relevant change associated with CBT, mean SCI score at post-treatment and follow-up was higher than our suggested threshold score of 6.0 used to identify normal sleepers on this measure.[55]

To be eligible for the study, all participants had to have initial SE < 80%. Therefore, it was of interest to determine the proportion within each group who exceeded this value (SE ≥ 80%) following intervention. These data are presented in Figure 2, along with comparisons on the more stringent clinical cutoff scores

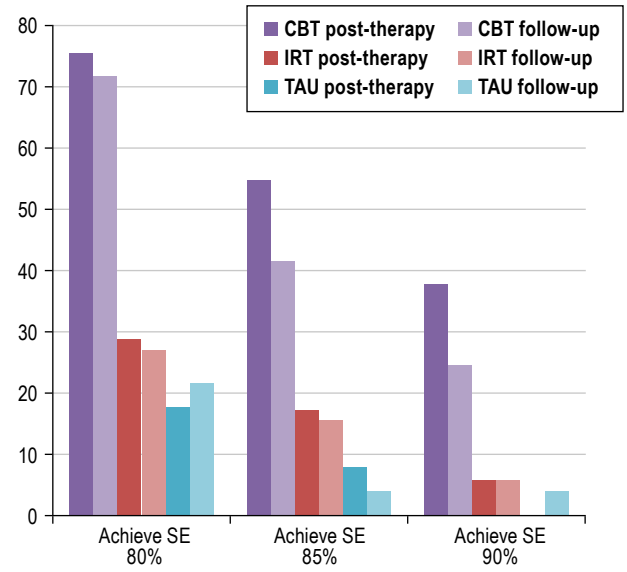


Figure 2—Percentage of patients within each treatment arm achieving sleep efficiency (SE) clinical end-points.

of SE ≥ 85% and SE ≥ 90%. Three-quarters of participants allocated to CBT completed the course with SE ≥ 80%, compared with less than one-third of those in IRT, and one in 5 of the TAU group (χ^2(2) = 33.0, P < 0.001). Likewise, 55% of CBT participants achieved a SE of 85% (χ^2(2) = 23.8, P < 0.001), and approaching 40% achieved SE ≥ 90% (χ^2(2) = 13.4, P = 0.001). These advantages of CBT over placebo and the passage of time alone, all represent large ES (w = 0.86, w = 0.72, and w = 0.51 for the 80%, 85%, and 90% criteria, respectively), where w is the square root of the standardized χ^2 statistic (ES conventions: w = 0.10 [small], 0.30 [medium], 0.50 [large]).[73] At follow-up, large effects were maintained for the 80% (w = 0.81) and 85% criteria (w = 0.62), with a medium effect observed for the 90% criterion (w = 0.38).

Finally, we wish to report that 38 IRT participants (79% of IRT completers) and 39 TAU participants (83% of those who provided post-treatment data) created CBT user accounts subsequent to completion of the trial period.

DISCUSSION

We compared CBT for insomnia, delivered via an automated media-rich web application, with a similarly delivered, credible placebo (IRT), and a waitlist TAU control group.

On our primary endpoint of SE, large pre- to post-treatment effect sizes were observed for CBT relative to IRT and TAU. These effects remained robust at follow-up. SOL was reduced by 56% (compared to 5% and 17% in IRT and TAU), and WASO was reduced by 63% (compared to 29% in IRT and a 4% increase in TAU). Global sleep-wake function, assessed by the Sleep Condition Indicator, similarly favored CBT, and clinical significance of findings was confirmed by the proportion of patients achieving SE values > 85% (post-treatment: 55% [CBT] v. 17.3% [IRT] v. 7.8% [TAU]; follow-up: 42% [CBT] v. 15% [IRT] v. 3.9% [TAU]), as well as improvements in daytime functioning. Moreover, the mean sleep parameter scores for CBT (Table 3) were within normative values (i.e.,

SOL < 30 min, WASO < 30 min, TST > 6 h,[76] and SCI > 6)[76] at follow-up.

Such outcomes appear comparable in magnitude to therapist-delivered CBT[27] and greater than the majority of online CBT studies.[44,45,47,49] Our findings are most similar to those of Ritterband et al.,[46,48] suggesting that there may be benefits associated with the design and delivery of online CBT, such as engaging animations and graphics and reminder prompts. Importantly, we also had comparatively low attrition rates (12% to 20%), in contrast with some other studies, where dropout rates have been as high as 33% pre-to post treatment,[47] and up to 49% pre-treatment to follow-up.[49]

Thus, CBT delivered using advanced web-based tools, and tested within a placebo-controlled design, had a positive and durable impact. It should be noted that IRT placebo relative to TAU, did also show some positive effects, particularly in reducing WASO, and in achieving SE endpoints for around 15% of participants. Such findings help to confirm that there was a placebo effect for at least a proportion of participants, on some outcomes. It should also be borne in mind that usual care continued in all groups, consistent with a real-world trial. Although we cannot be certain of the effects of such uncontrolled factors, it seems unlikely that usual care would systematically differ across our groups.

In contrast to substantial improvements in quantitative estimates of sleep, we observed more modest improvements on ratings of sleep quality. In the CBT group, mean scores increased by around 15 points (on a 100-point scale). Although statistically greater than IRT or TAU, the degree of absolute change and the final endpoint seem low. We cannot readily explain this, given (a) the global improvement observed on the SCI and the generalized benefits to daytime function observed with CBT, and (b) the literature that suggests that sleep quality can be more amenable than sleep pattern to improvement with CBT.[26,27] One possibility is that our dimensional, bipolar measure of sleep ("very unsatisfactory" to "very satisfactory") was not sufficiently sensitive. People tended to use mainly the central area of the scale, and we did not provide definition of intermediate points. Also the sleep quality rating was the first item on our diary, when it is more usual to be near the end.[77]

We did not investigate the association between treatment and use of medication. Indeed, our website specifically advised people not to adjust medication without consulting their doctor. Twenty percent reported taking hypnotics at baseline (Table 2) and slightly fewer (16%) reported using them at post-treatment, but this appeared unrelated to group allocation. Further research is required to consider how, if at all, online CBT may be used as an alternative to prescription medication.

We would suggest that our design was particularly rigorous, providing the first placebo-controlled evidence that online CBT for insomnia can be clinically effective. We have demonstrated that CBT effects are not merely associated with user engagement on an attractive programme, or with the demand characteristics and expectations of benefit associated with receiving treatment. IRT was considered a credible treatment by participants, reflected in low levels of attrition, faithful recording of sleep diary information, and good session completion rates. Furthermore, we included a comprehensive assessment of daytime outcomes, based on proposed revised DSM-5 criteria and research recommendations.[78] We would also suggest that there were methodological advantages associated with our technology. For example, treatment fidelity was likely enhanced because The Prof's interactions and recommendations, though highly tailored, were all pre-programmed. Indeed, standardization of protocols in online CBT may offer quality assurance that is superior to training therapists to consistently follow a manual. Technology offers greatly improved precision of measuring adherence (e.g., time stamps of page views, entries, and interactions).

In addition to demonstrating reliable interaction effects, our data also reveal main effects of time (independent of group allocation). In this respect it should be noted that all our participants started the trial in February 2011, completing in May/June 2011. One explanation of this finding may be an underlying seasonal improvement in sleep (or a reduced concern about insomnia) from late winter through to early summer. This requires further systematic study because, in most RCTs, participants are recruited sequentially, often over months or years. Thus any seasonal effect is likely to be randomly represented in the data. Of course, the time main effect may simply relate to spontaneous improvement over the study period. We cannot differentiate these possibilities at this stage. The feasibility of simultaneously commencing entire cohorts online affords potential advantages, and disadvantages. In relation to the former, online recruitment and in-parallel processing of many participants may permit highly efficient use of research resources. In terms of disadvantages, gathering a cohort for the purposes of research may not reflect the real world, where patients want to start their treatment whenever they feel ready to do so. A large scale, open trial of online CBT, with participants enrolling at the time of signing up to the site, would be welcome to address this point.

This study has a number of important limitations. Subjects were recruited by online survey and may represent a cohort unusually interested in addressing sleep problems. They certainly all had access to, and competencies in, using the internet, thus restricting the sample targeted from that of the wider population with insomnia. We also did not, for example, include polysomnography in our design, and therefore, we are unable to rule out occult sleep disorder pathology. Further work evaluating CBT with respect to objective sleep outcomes would be valuable. We also acknowledge that our selection of SE as the primary endpoint could have unduly favored CBT because the sleep restriction component of CBT can lead to improved SE, in the absence of other evidence. Whereas our outcome data across other sleep variables do corroborate significant sleep pattern gains, we would advocate using SE as part of a group of sleep endpoints in the future.

In setting the inclusion criterion of ≤ 79% for baseline SE on the sleep diary, we attempted to ensure that our participants had a current, prospectively monitored problem with sleep upon entry to the trial. In so doing, however, we recognize that we excluded those with better sleep efficiency, who may nonetheless have benefitted from CBT. In this regard, and also in our exclusion of those who reported being in either "poor" or "very poor" physical or mental health, our study departed from a real-world evaluation and limiting the generalizability of our findings. Future investigations with patients with active comorbidities,

who represent the majority of patients in clinical practice, are required. Finally, we acknowledge that our follow-up period, though experimentally controlled, was relatively short. Most face-to-face CBT-I studies have demonstrated maintenance of gains between 6 and 12 months post-treatment, with some showing durability up to 2 years.[27] Future work should assess the stability of gains over longer periods.

In keeping with a real world framework for online CBT, we wanted to have minimal contact with participants. Communication, therefore, was by email and questionnaire completion, with no face-to-face contact during the trial. We feel that demonstrating robust effects in the absence of formal contact strengthens the ecological validity of the study as well as the applicability of the approach. Contacts between the intervention system and the participants (e.g., text reminders from The Prof) and among the participants (the social community) on the other hand were integral to the program. Of course, the community here was necessarily limited, to the 53 CBT trial participants. Nevertheless, 37 (70%) posted comments on the site, indicating that this element of the program was valued, and it should be borne in mind that social networks generally increase in perceived value as they expand in scale. We do, however, recognize that it will be important to assess the specific impact of personal tailoring and community support in further online intervention studies. Consistent with the stepped care approach,[33] such work should bear in mind that personal preference is likely to play a role in motivation and adherence, such that some people may prefer to have personal support (regardless of whether or not they actually would "need" it). Indeed, there is recent evidence that brief behavioral intervention, involving only two in-person contacts, can be very effective.[79] There would be value in comparing efficacy, preference, and satisfaction between such minimal contact models and online CBT.

In conclusion, CBT delivered using an online media-rich web application with automated support and a community forum appears effective in improving the sleep and associated daytime functioning of adults with insomnia disorder. Further work is required to evaluate the objective changes associated with treatment delivered in this way. Treatment trials of insomnia associated with complex clinical presentations and associated with physical and/or mental health problems are also needed to establish any necessary pre-screening requirements for access to online, compared with, in-person CBT.

ACKNOWLEDGMENTS

No payments were made to any of the participants in this study. We wish to acknowledge and thank them for their interest in and commitment to this research, and to thank Boots WebMD and the Mental Health Foundation for their support in recruitment.

DISCLOSURE STATEMENT

This was not an industry supported study. The software and web development for this study was supported by Sleepio Limited. Dr. Espie is Clinical and Scientific Director of Sleepio Limited and a shareholder, but has not received any income from the company. He has also participated in speaking engagements and has served as a consultant for Boots Pharmaceuticals. He has also received free use of actigraphs by Philips Respironics. Dr. Hames is Managing Director and CEO of Sleepio Limited and has received a salary from the company. Dr. Williams has written workbook and online modules addressing sleep problems and is the Director of Five Areas Limited a company that provides access and training to CBT resources. The other authors have indicated no financial conflicts of interest.

REFERENCES

1. Singleton N, Bumpstead R, O'Brien M, et al. Psychiatric morbidity among adults living in private households, 2000. London: The Office for National Statistics, HMSO; 2001.
2. Ohayon MM. Epidemiology of Insomnia: what we know and what we still need to learn. Sleep Med Rev 2002;6:97-111.
3. Lichstein KL, Durrence HH, Reidel BW, et al. The epidemiology of sleep: age, gender and ethnicity. Mahwah, NJ: Lawrence Erlbaum Associates; 2004.
4. Morin CM, Belanger L, LeBlanc M, et al. The natural history of insomnia: A population-based 3-year longitudinal study. Arch Intern Med 2009;169:447-53.
5. American Academy of Sleep Medicine. International classification of sleep disorders: diagnostic and coding manual: 2nd ed. Westchester, IL: American Academy of Sleep Medicine; 2005.
6. Diagnostic and Statistical Manual of Mental Disorders, 4th ed (DSM-IV). Washington: American Psychiatric Association; 1994.
7. Morgan K. Factors influencing persistent subjective insomnia in old age: a follow study of good and poor sleepers age 65-74. Age Ageing 1989;18:17-125.
8. Roth T, Ancoli-Israel S. Daytime consequences of insomnia in the United States: Results of the 1991 National Sleep Foundation Survey II. Sleep 1999;22:Suppl 2, S354-63.
9. Kyle SD, Morgan K, Espie C. Insomnia and health-related quality of life. Sleep Med Rev 2010;14; 69-82.
10. Cole MG, Dendukuri N. Risk factors for depression among elderly community subjects: a systematic review and meta-analysis. Am J Psychiatry 2003;160:1147-56.
11. Baglioni C, Battagliese G, Feige B, et al. Insomnia as a predictor of depression: A meta-analytic evaluation of longitudinal epidemiological studies. J Affect Disord 2011;135:10-19.
12. Vgontzas AN, Liao D, Bixler EO, Chrousos GP, Vela-Bueno A. Insomnia with objective short sleep duration is associated with a high risk for hypertension Sleep 2009;32:491-7.
13. Vgontzas AN, Liao D, Pejovic S, Calhoun S, Karataraki M, Bixler EO. Insomnia with objective short sleep duration is associated with type 2 diabetes: a population-based Study. Diabetes Care 2009;32:1980-5.
14. Dew MA, Hoch CC, Buysse DJ, et al. Healthy older adults' sleep predicts all cause mortality at 4 to 19 years of follow-up. Psychosom Med 2003;65:63-73.
15. Vgontzas AN, Liao D, Pejovic S, et al. Insomnia with short sleep duration and mortality: the Penn State Cohort. Sleep 2010;33:1159-64.
16. Cappuccio FP, D'Elia L, Strazzullo P, Miller MA. Sleep duration and all-cause mortality: a systematic review and meta-analysis of prospective studies. Sleep 2010; 33:585-92.
17. Rosekind MR, Gregory KB. Insomnia risks and costs: health, safety, and quality of life. Am J Manag Care 2010;16:617-26.
18. Daley M, Morin CM, LeBlanc M, Grégoire JP, Savard J. The economic burden of insomnia: direct and indirect costs for individuals with insomnia syndrome, insomnia symptoms, and good sleepers. Sleep 2009;32:55-64.
19. NIH State-of-the-Science Conference Statement on Manifestations and Management of Chronic Insomnia in Adults. NIH Consens Sci Statements 2005;22:1-30.
20. Sateia MJ, Nowell PD. Insomnia. Lancet 2004;364:1959-73.
21. Kripke D. Hypnotic drugs: deadly risks, doubtful benefits. Sleep Med Rev 2000;4:5-20.
22. Mendelson WB. A review of the evidence for the efficacy and safety of Trazodone. J Clin Psychiatry 2005;66:469-76.
23. Guidance on the use of zaleplon, zolpidem and zopiclone for the short-term management of insomnia. London: National Institute for Clinical Excellence. Technology Appraisal Guidance No.77; 2004.
24. Wilson, SJ, Nutt DJ, Alford C, et al. British Association for Psychopharmacology consensus statement on evidence-based treatment of insomnia, parasomnias and circadian rhythm disorders. J Psychopharmacol 2010;24:1577-600.

25. Irwin MR, Cole JC, Nicassio PM. Comparative meta-analysis of behavioral interventions for insomnia and their efficacy in middle-aged adults and in older adults 55+years of age. Health Psychol 2006;25:3-14.
26. Morin CM, Bootzin RR, Buysse DJ, Edinger JD, Espie CA, Lichstein KL. Psychological and behavioral treatment of insomnia: Update of the recent evidence (1998-2004). Sleep 2006;29:1398-414.
27. Riemann D, Perlis ML. The treatments of chronic insomnia: A review of benzodiazepine receptor agonists and psychological and behavioral therapies. Sleep Med Rev 2009;13:205-214.
28. Espie CA. Insomnia: conceptual issues in the development, persistence, and treatment of sleep disorder in adults. Annu Rev Psychol 2002;53;215-43.
29. Morin CM, Gaulier B, Barry T, Kowatch RA. Patients' acceptance of psychological and pharmacological therapies in insomnia. Sleep 1992;15:302-5.
30. Espie CA, Barrie LM, Forgan GS. Comparative investigation of the psychophysiological and idiopathic insomnia disorder phenotypes: psychological characteristics patients' perspectives and implications for clinical management. Sleep 2012;35:385-393.
31. Dyas JV, Apekey TA, Tilling M, Orner R, Middleton H, Siriwardena AN. Patients' and clinicians' experiences of consultation in primary care for sleep problems and insomnia: a focus group. Br J Gen Pract 2010;60:329-33.
32. Falloon K, Arroll B, Elley CR, Fernando A. The assessment and management of insomnia in primary care. BMJ 2011;342:d2899.
33. Espie, C.A. 'Stepped care': a health technology solution for delivering Cognitive Behavioral Therapy as a first line insomnia treatment. Sleep 2009;32:1549-58.
34. Bennett-Levy J, Richards D, Farrand P, et al. Oxford guide to low intensity CBT Interventions (Oxford Guides in Cognitive Behavioural Therapy). Oxford, UK: Oxford University Press; 2010.
35. Perlis ML, Smith MT. How can we make CBT-I and other BSM services widely available? J Clin Sleep Med 2008;15:11-3.
36. National Institute of Clinical Excellence. NICE implementation uptake report: insomnia - newer hypnotic drugs. 2006. (http://www.nice.org.uk/media/640/85/TA77NICEImplUptake.pdf)
37. NHS Scotland, Information Services Division. Prescribing & Medicines: Medicines used in Mental Health 2011. (http://www.isdscotland.org/Health-Topics/Prescribing-and-Medicines/Publications/2011-09-27/2011-09-27-PrescribingMentalHealth-Report.pdf?26615542174)
38. Bower P, Gilbody S. Stepped care in psychological therapies: access, effectiveness and efficiency. Br J Psychiatry 2005;186:11-17.
39. Espie CA , MacMahon KM , Kelly H, et al. Randomized clinical effectiveness trial of nurse-administered small group cognitive behaviour therapy for persistent insomnia in general practice. Sleep 2007;30:574-84.
40. Espie CA, Inglis SJ, Harvey L. Predicting clinically significant response to cognitive behavior therapy for chronic insomnia in general medical practice: Analysis of outcome data at 12 months post treatment. J Consult Clin Psychol 2001;69:58-66.
41. Espie CA, Fleming L, Cassidy J, et al. Randomized controlled clinical effectiveness trial of cognitive behaviour therapy compared with treatment as usual for persistent insomnia in patients with cancer. J Clin Oncol 2008;26:4651-8.
42. Swift N, Stewart R, Andiappan M, Smith A, Espie CA, Brown JSL. The effectiveness of community day-long CBT-I workshops for participants with insomnia symptoms: a randomised controlled trial. J Sleep Res 2011; http://dx.doi.org/10.1111/j.1365-2869.2011.00940.x.
43. van Straten A, Cuijpers P. Self-therapy for insomnia: a meta-analysis. Sleep Med Rev 2009;13:61-71.
44. Strom L, Pettersson R, Andersson G. Internet-based treatment for insomnia: a controlled evaluation. J Consult Clin Psychol 2004;72:113-20.
45. Suzuki E, Tsuchiya M, Hirokawa K, Taniguchi T, Mitsuahashi T, Kawakami N. Evaluation of an Internet-based self-help program for better quality of sleep among Japanese workers: a randomized controlled trial. J Occup Health 2008;50:387-99.
46. Ritterband LM, Thorndike FP, Gonder-Frederick GA, et al. Efficacy of an Internet-based behavioral intervention for adults with insomnia. Arch Gen Psychiatry 2009;66:692-8.
47. Vincent N, Lewycky S. Logging on for better sleep: RCT of the effectiveness of online treatment for insomnia. Sleep 2009;32:807-15
48. Ritterband LM, Bailey ET, Thorndike FP, Lord HR, Farrell-Carnahan L, Baum LD. Initial evaluation of an internet intervention to improve the sleep of cancer survivors with insomnia. Psychooncology 2011 http://dx.doi.org/10.1002/pon.1969. [Epub ahead of print]
49. Lancee J, van den Bout J, van Straten A, Spoormaker VI. Internet-delivered or mailed self-help treatment for insomnia? A randomized waiting-list controlled trial. Behav Res Ther 2011;50;22-9.
50. Steinmark SW, Borkovec TD. Active and placebo treatment effects on moderate insomnia under counterdemand and positive demand instructions. J Abnorm Psychol 1974;83:157-63.
51. TIME Specials (2011). The 50 Best Websites of 2011. (http://www.time.com/time/specials/packages/0,28757,2087815,00.html)
52. Gellatly J, Bower P, Hennessy S, Richards D, Gilbody S, Lovell K. What makes self-help interventions effective in the management of depressive symptoms? Meta-analysis and meta-regression. Psychol Med 2007;37:1217-28.
53. Reynolds CF, Redline S. The DSM-V sleep-wake disorders nosology: an update and an invitation to the sleep community. J Clin Sleep Med 2010;15;9-10.
54. American Psychiatric Association (2010) DSM-5 development. Available from: http://www.dsm5.org/.
55. Espie CA, Kyle SD, Hames P. The daytime impact of DSM-V insomnia disorder: comparative analysis of insomnia subtype from the Great British Sleep Survey (n = 11,129). Sleep Biol Rhythms 2011;9:S241.
56. Saunders JB, Aasland OG, Babor TF, de la Fuente JR, Grant M. Development of the Alcohol Use Disorders Identification Test (AUDIT): WHO collaborative project on early detection of persons with harmful alcohol consumption. II. Addiction 1993;88:791-804.
57. Mayfield D, Mcleod G, Hall P. The CAGE questionnaire: validation of a new alcoholism screening instrument. Am J Psychiatry 1974;131:1121-3.
58. Henry JD, Crawford JR. The short-form version of the Depression Anxiety Stress Scales (DASS-21): Construct validity and normative data in a large non-clinical sample, Br J Clin Psychol 2005;44:227-39.
59. Schulz KF, Altman DG, Moher D, for the CONSORT Group. CONSORT 2010 Statement: updated guidelines for reporting parallel group randomised trials. Ann Int Med 2010;152.
60. Morin CM, Espie CA. Insomnia: a clinical guide to assessment and treatment. New York: Kluwer Academic/Plenum Publishers; 2003.
61. Edinger JD, Fins AI, Sullivan RJ, et al. Sleep in the laboratory and sleep at home: comparisons of older insomniacs and normal sleepers Sleep 1997;20:1119-26.
62. Coates TJ, Killen KD, George J, Marchini E, Silverman S, Thoresen C. Estimating sleep parameters: a multi-trait multi-method analysis. J Consult Clin Psychol 1982;50:345-52.
63. Morin CM. Measuring outcomes in randomized clinical trials of insomnia treatments. Sleep Med Rev 2003;7;263-79.
64. Buysse DJ, Reynolds CF III, Monk TH, Berman SR, Kupfer DJ. The Pittsburgh Sleep Quality Index: a new instrument for psychiatric practice and research. Psychiatry Res 1989;28:193-213.
65. Morin CM. Insomnia: Psychological assessment and management. New York: Guilford, 1993.
66. Gardani M, Miller CB, Biello S, Ellis J, Von Schantz M, Archer S. The association of sleepiness and diurnal preference with salivary amylase activity. Abstracts of 4th International Congress of WASM & 5th Conference of CSS / Sleep Med 12, Suppl. 1 (2011) S1-130.
67. Espie CA, Brooks DN, Lindsay WR. An evaluation of tailored psychological treatment for insomnia in terms of statistical and clinical measures of outcome. J Behav Ther Exp Psych 1989;20:143-53.
68. Espie CA, Inglis SJ, Harvey L, Tessier S. Insomniacs' attributions: psychometric properties of the dysfunctional beliefs and attitudes about sleep scale and the sleep disturbance questionnaire. J Psychsom Res 2000;48:141-8.
69. Harvey KJ, Espie CA. Development and preliminary validation of the Glasgow Content of Thoughts Inventory (GCTI): a new measure for the assessment of pre-sleep cognitive activity. Br J Clin Psychol 2004;43:409-20.
70. Espie CA, Lindsay WR, Brooks N, Hood EM, Turvey T. A controlled comparative investigation of psychological treatments for chronic sleep-onset insomnia. Behav Res Ther 1989;27:79-88.
71. Edinger JD, Wohlgemuth WK, Radtke RA, Marsh GR, Quillian RE. Cognitive behavioural therapy for treatment of chronic primary insomnia: a randomized controlled trial. JAMA 2001;285:1856-64.

72. Manber R, Edinger JD, Gress JL, San Pedro-Salcedo MG, Kuo TF, Kalista T. Cognitive behavioral therapy for insomnia enhances depression outcome in patients with comborbid major depressive disorder and insomnia. Sleep 2008;31:489-95.
73. Cohen J. Statistical power analysis for the behavioral sciences. 2nd ed. Hillsdale, NJ: Erlbaum, 1988.
74. Norusis MJ, Inc. SPSS. PASW Statistics 18 Advanced Statistical Procedures Companion. Chicago: SPSS Inc., 2011.
75. Department for Communities and Local Government. The English Indices of Deprivation 2007. Office for National Statistics. (http://www.communities.gov.uk/documents/communities/pdf/733520.pdf
76. Lichstein KL, Durrence HH, Taylor DJ, Bush AJ, Riedel BW. Quantitative criteria for insomnia. Behav Res Ther 2003;41:427-45.
77. Carney CE, Buysse DJ, Ancoli-Israel S, et al. The consensus sleep diary: standardizing prospective sleep self-monitoring. Sleep 2012;35:287-302.
78. Buysse DJ, Ancoli-Israel S, Edinger JD, Lichstein KL, Morin CM. Recommendations for a standard research assessment of insomnia. Sleep 2006;29:1155-73.
79. Buysse DJ, Germain A, Moul DE, et al. Efficacy of brief behavioural treatment for chronic insomnia in older adults. Arch Intern Med 2011;171:887-95.

CONSORT 2010 checklist of information to include when reporting a randomized trial*

Section/Topic	Item No	Checklist item	Reported on page No
Title and abstract			
	1a	Identification as a randomized trial in the title	769
	1b	Structured summary of trial design, methods, results, and conclusions (for specific guidance see CONSORT for abstracts)	769
Introduction			
Background and objectives	2a	Scientific background and explanation of rationale	769-70
	2b	Specific objectives or hypotheses	770
Methods			
Trial design	3a	Description of trial design (such as parallel, factorial) including allocation ratio	771
	3b	Important changes to methods after trial commencement (such as eligibility criteria), with reasons	n/a
Participants	4a	Eligibility criteria for participants	770
	4b	Settings and locations where the data were collected	770
Interventions	5	The interventions for each group with sufficient details to allow replication, including how and when they were actually administered	772-3; Table 1
Outcomes	6a	Completely defined pre-specified primary and secondary outcome measures, including how and when they were assessed	771-2
	6b	Any changes to trial outcomes after the trial commenced, with reasons	n/a
Sample size	7a	How sample size was determined	773
	7b	When applicable, explanation of any interim analyses and stopping guidelines	n/a
Randomization			
Sequence generation	8a	Method used to generate the random allocation sequence	771
	8b	Type of Randomization; details of any restriction (such as blocking and block size)	771
Allocation concealment mechanism	9	Mechanism used to implement the random allocation sequence (such as sequentially numbered containers), describing any steps taken to conceal the sequence until interventions were assigned	771, 773
Implementation	10	Who generated the random allocation sequence, who enrolled participants, and who assigned participants to interventions	771
Blinding	11a	If done, who was blinded after assignment to interventions (for example, participants, care providers, those assessing outcomes) and how	n/a
	11b	If relevant, description of the similarity of interventions	772-3
Statistical methods	12a	Statistical methods used to compare groups for primary and secondary outcomes	773-4
	12b	Methods for additional analyses, such as subgroup analyses and adjusted analyses	773-4

*We strongly recommend reading this statement in conjunction with the CONSORT 2010 Explanation and Elaboration for important clarifications on all the items. If relevant, we also recommend reading CONSORT extensions for cluster randomized trials, non-inferiority and equivalence trials, non-pharmacological treatments, herbal interventions, and pragmatic trials. Additional extensions are forthcoming: for those and for up to date references relevant to this checklist, see www.consort-statement.org.

CONSORT 2010 checklist continues on the following page

CONSORT 2010 checklist of information to include when reporting a randomized trial*

Section/Topic	Item No	Checklist item	Reported on page No
Results			
Participant flow (a diagram is strongly recommended)	13a	For each group, the numbers of participants who were randomly assigned, received intended treatment, and were analysed for the primary outcome	774; Figure 1
	13b	For each group, losses and exclusions after Randomization, together with reasons	774; Figure 1
Recruitment	14a	Dates defining the periods of recruitment and follow-up	774, 778
	14b	Why the trial ended or was stopped	n/a
Baseline data	15	A table showing baseline demographic and clinical characteristics for each group	Table 2
Numbers analysed	16	For each group, number of participants (denominator) included in each analysis and whether the analysis was by original assigned groups	Figure 1
Outcomes and estimation	17a	For each primary and secondary outcome, results for each group, and the estimated effect size and its precision (such as 95% confidence interval)	774-7; Table 3, 4
	17b	For binary outcomes, presentation of both absolute and relative effect sizes is recommended	777
Ancillary analyses	18	Results of any other analyses performed, including subgroup analyses and adjusted analyses, distinguishing pre-specified from exploratory	777
Harms	19	All important harms or unintended effects in each group (for specific guidance see CONSORT for harms)	774
Discussion			
Limitations	20	Trial limitations, addressing sources of potential bias, imprecision, and, if relevant, multiplicity of analyses	778-9
Generalisability	21	Generalisability (external validity, applicability) of the trial findings	777-8
Interpretation	22	Interpretation consistent with results, balancing benefits and harms, and considering other relevant evidence	777-9
Other information			
Registration	23	Registration number and name of trial registry	769
Protocol	24	Where the full trial protocol can be accessed, if available	n/a
Funding	25	Sources of funding and other support (such as supply of drugs), role of funders	779

*We strongly recommend reading this statement in conjunction with the CONSORT 2010 Explanation and Elaboration for important clarifications on all the items. If relevant, we also recommend reading CONSORT extensions for cluster randomized trials, non-inferiority and equivalence trials, non-pharmacological treatments, herbal interventions, and pragmatic trials. Additional extensions are forthcoming: for those and for up to date references relevant to this checklist, see www.consort-statement.org.

Randomized, placebo-controlled, trial of online Cognitive Behavioral Therapy for persistent Insomnia Disorder: therapeutic impact upon attribution, cognition and psychopathology

Abstract

Objectives: Insomnia patients complain that mental events keep them awake. This study investigates how online cognitive behavioral therapy (CBT) impacts such psychological processes and outcomes.

Method: Randomized, placebo-controlled trial of 164 adults (120 F: [mean age 49y (18–78y)] meeting DSM-5 criteria for Insomnia Disorder, assigned to CBT (n=55; 40F), Imagery Relief Therapy (IRT placebo; n=55; 42F) or Treatment as Usual (TAU; n=54; 38F). CBT/IRT comprised 6 online sessions delivered by an animated therapist, with automated web/email support and access to 'Wikipedia' articles. CBT had access to a moderated community of users. TAU comprised 'usual care'. Participants completed the Sleep Disturbance Questionnaire (SDQ), Glasgow Content of Thoughts Inventory (GCTI), and Depression Anxiety and Stress Scales (DASS) at baseline, post-treatment and 8 weeks follow-up.

Results: The sample was characterised by "mental arousal in bed" (SDQ); 'sleep and sleeplessness' and 'rehearsal and planning' thoughts (GCTI) being most common. Treatment effects were observed for all SDQ domains ('unable to relax', 'mental arousal', 'lack of routine', 'trying too hard'); relative effect sizes (CBT v. IRT) were most pronounced for 'trying too hard' (d=.76 at post-treatment, d=.51 at 8 weeks follow-up). CBT was superior to IRT on the GCTI: 'rehearsal and planning' (d=.62 and d=.26) and 'sleep and sleeplessness' (d=.74, d=.56). Small effects were observed for depression and stress symptoms. Placebo effects (IRT v. TAU) were small to moderate. Changes in attribution and thought-content were associated with improvements in global sleep quality.

Conclusion: Online CBT modifies sleep-related attributions, night-time thought content and psychopathology over and above a placebo intervention.

Keywords: Insomnia, sleep, treatment, psychological intervention, placebo, internet

Introduction

Insomnia is a prevalent and troublesome disorder where people find it difficult to get to sleep and/or remain asleep, resulting in daytime problems with fatigue, mood, concentration, relationships, work productivity and/or sleepiness. According to the proposed criteria for DSM-5, an Insomnia Disorder should be coded when such problems occur 3 or more nights per week for at least 3 months, and "*whether or not there is a co-existing psychiatric, medical, or another sleep disorder*" (www.dsm-5.org: June-02-2010; Reynolds & Redline, 2010). This is in keeping with mounting evidence that insomnia is a characteristically persistent condition (Morin et al., 2009; Green et al., 2012) that is associated with illness vulnerability, particularly the evolution of and relapse into depression (Cole & Dendukuri, 2003; Baglioni et al., 2011). In its most common form, 'Psychophysiological Insomnia' comprises hyperarousal in bed, maladaptive sleep behavior, mental arousal in the form of a racing mind, and the evolution of a vicious cycle where the harder one strives to sleep, the more elusive sleep becomes (International Classification of Sleep Disorders: American Academy of Sleep Medicine, 2005). There is a long tradition of psychological research on insomnia (e.g. Bootzin & Nicassio, 1978; Borkovec, 1982; Hauri & Fisher, 1986; Morin, 1993; Espie, 2002; Harvey, 2002; Perlis et al., 1997), suggesting that Cognitive Behavioral Therapy (CBT) is a good conceptual "fit" for insomnia. Indeed, outcome studies suggest that CBT offers the best long-term treatment benefit (Morin et al., 2006; Irwin, Cole & Nicassio, 2006; Riemann & Perlis, 2009; Wilson et al., 2010).

Although CBT is a credible and effective therapy for insomnia, it is seldom available in practice (Espie, 2009). This is a concern, not least because of the demonstrated high population prevalence rate of clinical Insomnia Disorder of 10-12%, with older adult rates >20% (Ohayon, 2002; Lichstein et al., 2004), and the paucity of skilled therapists to offer an alternative to pharmacological treatment (Espie, 2009). Nevertheless, in keeping with developments more broadly in psychological therapies

(e.g. Bower & Gilbody, 2005; Bennett-Levy et al., 2010), a considerable amount of work has been undertaken on manualised CBT for insomnia, in guided self-help, and more recently in online CBT (Espie et al., 2001, 2008; Swift et al., 2011; van Straten & Cuijpers, 2009; Vincent & Lewycky, 2009; Ritterband et al., 2009).

The first randomized placebo controlled trial of online CBT, using a rich media web-based programme, was recently conducted, demonstrating significant improvements in sleep pattern and daytime functioning (Espie et al., 2012). In this secondary analysis, emphasis is placed on the psychological processes and outcomes associated with CBT. Consistent with the formulation of insomnia as a psychophysiological condition, it is important to reflect not only upon the impact of CBT on sleep, but also its impact on a range of secondary outcomes that are likely maintaining factors, such as attribution, cognition and psychopathological status.

The objectives of this report, therefore, were to address the following questions: Is online CBT for persistent insomnia disorder superior to a credible placebo intervention as well as to a treatment as usual condition, in modifying sleep-related attributions, night-time thinking processes and daytime symptoms of depression, anxiety and stress? Are these effects durable and clinically important?

Methods

The methodology for this trial has previously been published (Espie et al., 2012) and the website www.sleepio.com/research hosts illustrative material of the study evaluation and intervention procedures. In brief, however, participants from the UK community (18+yr), who had completed the online Great British Sleep Survey (GBSS: Espie et al., under review) and who met proposed DSM-5 criteria for persistent Insomnia Disorder were invited to take part. The underlying logic of the GBSS ensured that individuals: had a) a current complaint of poor sleep (difficulty initiating and/or maintaining sleep, early morning wakening, or non-restorative sleep); with b) significant daytime effects; c)

affecting them 3 or more nights per week for ≥3 months. People with possible significant mental or physical health problems, suspected disorders of sleep other than insomnia or heavy alcohol use, were excluded conservatively, using further online screening. The use of sleeping pills or other sleep aids was permitted. Usual care that participants had been receiving from their medical advisers continued in all arms.

A total of 10,071 adults completed the GBSS during the period April 2010 to February 2011; of whom, 6,609 provided email addresses. In total, 1,342 of this latter group (20.3%) met screening criteria and were invited by email to consider taking part. Of these, 276 (20.1%) replied and consented to further screening. The majority of this group (n=228, 82.6%) then completed these further standardized assessments. Finally, to confirm current eligibility, participants completed prospectively a 7 day online sleep diary to confirm a sleep problem of clinical severity. Sixty-four participants were excluded during these final stages, and the remaining sample of 164 eligible, consenting participants was randomized into the study (Figure 1).

[Insert Figure 1]

Study design

The study was a pragmatic, parallel group randomized controlled trial comprising three treatment arms; (1) online CBT, (2) online Imagery Relief Therapy (IRT: placebo), (3) treatment as usual (TAU), with blind assignment to group determined by a computer-generated random allocation schedule, operated by a remote independent technical operator. The trial followed CONSORT 2010 guidelines (Schulz et al., 2010). Consistent with the inclusion/ exclusion criteria above, the study design in effect was CBT+TAU v. IRT+TAU v. TAU alone. Major assessments were at baseline, post-treatment, and follow-up 8 weeks later. Participants randomized to the IRT placebo or TAU arm were offered the online CBT package upon completion of the study. All assessment, treatment and data-gathering procedures were conducted online, and all queries/ enquiries were managed electronically, without any face-to-face contact. The study protocol was

approved by the University of Glasgow, Faculty of Medicine Research Ethics Committee and all participants provided informed consent online (see www.sleepio.com/research).

Assessment measures

Three self-report measures were completed at each assessment point (baseline, post-treatment, follow-up) to evaluate different aspects of sleep-related psychological wellbeing. These were secondary measures which complemented the sleep diary, completed daily, from which our primary endpoint of sleep efficiency was derived (see 'sleep diary' below). They were included to provide a profile of participants' attributional, cognitive, and psychopathological characteristics which may be important in a) developing a better understanding of insomnia as a psychophysiological condition, suited to cognitive and behavioural intervention; and in b) investigating treatment processes and outcomes associated with CBT.

Sleep Disturbance Questionnaire (SDQ)

The SDQ has been used for more than 20 years to profile the attributional beliefs of people with insomnia, to aid tailoring of CBT to individual circumstances, and to evaluate treatment-related change (e.g. Espie et al., 1989; Espie et al., 2000; Harvey et al., 2005; Smith & Trinder, 2001). The scale comprises 12 items; three relating to each of four dimensions: attribution to being 'unable to relax', to 'mental arousal', to there being a 'lack of routine' associated with sleep, and to 'trying too hard' to sleep. Studies have shown that this is a relatively 'pure' factor structure, explaining 61% of variance (Espie et al, 2000). The respondent is asked to rate each item on a 5-point scale (0 'never true', 1 'seldom true', 2 'sometimes true', 3 'often true', 4 'very often true') in relation to typical nights when s/he does not sleep well. Thus subscale scores reflect the strength of attribution towards a particular explanation of insomnia [e.g. '*My body is full of tension*' (unable to relax), '*I am unable to empty my mind*' (mental arousal), '*I can't get my sleep pattern into a proper routine*' (lack of routine), '*I get too "worked up" at not sleeping*' (trying too hard)]. Data from the present study demonstrate that the SDQ has

satisfactory reliability (α = 0.82, range of α-if-item-deleted = 0.78-0.84). There was moderate inter-correlation between the subscale scores (average r = .40).

Glasgow Content of Thoughts Inventory (GCTI)

The GCTI was originally developed using an iterative process. First, a large number of spontaneous wakeful cognitions of people with insomnia was gathered using voice-activated audio-recorders; and these were subjected to content analysis to identify major underlying themes (Wicklow & Espie, 2000). Second, a psychometric scale development study was conducted, on a separate cohort of patients. This yielded a 25-item self-report inventory of exemplar common thoughts, related to each of three themes: 'rehearsal and planning' thoughts', thoughts relating to 'sleep and sleeplessness' and thoughts reflecting 'heightened awareness' (Harvey & Espie, 2004). Items are rated on a 4-point scale (0 'never', 1 'sometimes', 2 'often', 3 'always') in response to the lead question: 'how often over the past 7 nights have the following thoughts kept you awake?' Such information is useful to tailor the cognitive component of CBT (cf. Morin & Espie, 2003). In the present study, the GCTI was condensed, to nine items: three per theme [i.e. '*what happened today and what I've got on tomorrow*', '*things that have happened in the past and how they worked out'*, '*what the future might hold and what I should be doing for things to work out well*', (rehearsal and planning); '*how long I've been lying awake'*, '*how I'm going to cope tomorrow if I don't sleep well tonight*', '*how out of control my sleep is and I don't know what to do about it*' (sleep and sleeplessness); '*noises I can hear in the house or from outside*', '*my body feeling hot or cold; or my heart beat pounding in my head*', '*trivial things of no importance that go through my mind*'(heightened awareness)]. This short-form GCTI appears to perform similarly to the original (Harvey & Espie, 2004: α = 0.87). Internal consistency in the present study was good (α = 0.79, range of α-if-item-deleted = 0.75-0.80) and average inter-correlation of the derived themes was moderate (r = .38).

Depression, Anxiety, Stress, Scale (DASS)

Finally, we selected the DASS (Henry & Crawford, 2005: 21-item) because it takes a dimensional view of psychopathological symptoms, and is commonly used in samples of participants who do not have a formal affective or anxiety disorder (as in this study). For example, the DASS has been used in previous insomnia treatment studies (Harris, Lack, Wright, Gradisar, & Brooks, 2007) and as a depression and anxiety screening tool in other medical conditions (Mitchell, Burns, & Dorstyn, 2008). Moreover, it has a subscale on stress, which often co-presents with insomnia. Each item is rated on a 4-point scale (0 '*did not apply to me at all*', 1 '*applied to me to some degree, or some of the time*', 2 '*applied to me to a considerable degree, or a good part of the time*', 3 '*applied to me very much, or most of the time*') in relation to the past week. It has three subscales, each composed of 7 items: depression (DASSdep: e.g. '*I couldn't seem to experience anything positive at all*'), anxiety (DASSanx: e.g. '*I was worried about situations in which I might panic or make a fool of myself*') and stress (DASSstress: e.g. '*I was intolerant of anything that kept me from getting on with what I was doing*') which have satisfactory internal reliability (Henry & Crawford, 2005: α = 0.88, α = 0.82, α = 0.90 respectively). In the present study these values were somewhat lower but still satisfactory for DASSdep (α = 0.78, range of α-if-item-deleted = 0.74-0.79) and DASSstress (α = 0.80, range of α-if-item-deleted = 0.76-0.81). DASSanx was less reliable, perhaps due to the limited range of scores we obtained on this subscale (α = 0.62, range of α-if-item-deleted = 0.48-0.69)

Sleep diary

Participants also completed an online diary each morning upon rising (www.sleepio.com/research). The diary-derived variable of interest here is sleep efficiency (SE, %) calculated by the formula {[1-(SOL+WASO/TIB)] x100} where SOL refers to 'sleep onset latency' (time taken to fall sleep), WASO refers to 'wake time after sleep-onset' (total time awake resulting from awakenings in the night), and TIB refers to 'time in bed' (total time from retiring to rising). Thus, SE reflects the proportion of time

spent in bed that the person is asleep, and the achievement of SE of 80%, 85% and 90% reflect clinically important improvement endpoints and not merely statistical change (c.f. Morin, 2003).

Sleep Condition Indicator

The Sleep Condition Indicator (SCI) is a patient-reported outcome measure, based upon DSM-5 Insomnia Disorder criteria. It is brief (8-item), and has shown excellent reliability in a large field study of 11,129 participants (α = 0.89, range of α-if-items systematically deleted = 0.87-0.90; Espie et al., under review). The SCI generates scores in the range 0 to 10, with higher values reflecting a person's sleep being in 'better condition'. The SCI correlates strongly with other standard index measures, of sleep quality [Pittsburgh Sleep Quality Index (Buysse et al., 1989): r = .78, n = 256] and insomnia severity [Insomnia Severity Index (Morin, 1993): r = .79, n = 256] (Gardani et al., 2011).

Treatment groups

CBT

Participants received 6 weekly sessions delivered by an animated 'virtual therapist' (The Prof). The programme comprised a fully automated media-rich web application, driven dynamically by baseline, adherence, performance and progress data, and including an online Wikipedia style sleep educational site, a social community of fellow users moderated by experts, and support, prompts and reminders sent by email and mobile SMS. At the start of each session, The Prof conducted a progress review with the participant, explored the diary data submitted during the week, the participant's current sleep status and pattern, and progress achieved against goals previously set. Underlying algorithms fed the delivery of information, support and advice in a personally-tailored manner. CBT content was consistent with the literature (e.g. Morin & Espie, 2003), and covered behavioral (e.g. sleep restriction, stimulus control) and cognitive (e.g. putting the day to rest, thought re-structuring, imagery, articulatory suppression, paradoxical intention, mindfulness) strategies, as well as additional relaxation strategies (progressive

muscle relaxation and autogenic training) and advice on lifestyle and bedroom factors (sleep hygiene). The following illustrations may be helpful. In sleep restriction, The Prof proposes a new 'window' for sleep, calculated from available sleep diary data, and engages with the participant to help them select the timing (onset/ offset) of this window from a set of personalised options. An example, of a cognitive technique, is where another animated character (with insomnia) presents to the Prof their concerns, dysfunctional beliefs and associated emotions. The Prof then asks the participant to choose some solutions from a menu of options, and delivers this as advice to the character, who is seen to revise his thinking. The Prof then reveals to the participant that the scenario was based upon his/ her own sleep-related attributions and thoughts (from baseline SDQ and GCTI data). In this way the participant is helped to learn how to restructure dysfunctional thinking. The intervention was based upon a previously validated manual, found to be effective in three randomized clinical trials (Espie et al., 2001, 2007, 2008). Illustrative examples of the CBT and IRT interventions are provided at www.sleepio.com/research.

IRT

This was also delivered by 'The Prof' using the same application platform, and design and execution principles as for CBT i.e. interactive sessions, dynamic feedback against personal goals, progress review at start of each session, automatic calculation of sleep data over time, personal case file, 24/7 access, Wikipedia of sleep; but with no known active therapeutic ingredient. IRT was based on a well-established and credible, non-pharmacological placebo intervention (Steinmark & Borkovec, 1974) which has been used successfully in several trials (Espie et al., 1989; Edinger at al., 2001; Manber et al., 2008). The term *Imagery Relief Therapy* was used in order to enhance credibility of an active and novel therapy. For example, if participants were to enter this as an internet search term, they would come upon material which would appear to be valid for a psychological problem such as sleep. However, IRT contained no active components of

imagery training, or of systematic desensitization. Likewise, it did not include detailed relaxation instruction or any behavioral advice. Rather, the participant was trained to visualise neutral objects (e.g. a key) or shapes (e.g. a yellow square) in conjunction with thinking about sequential aspects of their evening routine (e.g. setting the table for dinner), and was asked to practise these pairings for 20 minutes per day early in the evening. The rationale given for this 'quasi-desensitization' framework, was that successful sleep was associated with good preparation, and that neutralizing unhelpful associations with evening routines would recondition them towards automatic sleep engagement and sleep maintenance.

Protocol standardization

The integrity and fidelity of treatment allocations was assured by the online procedures which delivered the interventions. In addition, in session 1 of both CBT and IRT conditions, participants were invited to commit (or not) to the course following an explanation of the therapeutic rationale (all did so). Participants in different groups did not have contact with each other, nor did they have access to alternative treatment materials. Both CBT and IRT were scripted and automated so support and length of treatment was identical within each group. All web-based interactions were electronically stored to provide time-stamped data on participant activity (e.g. diary entries, session activities, engagement with the community, adherence to tasks).

TAU

In real world practice, insomnia patients often have concurrent physical and psychological symptoms, as well as concurrent treatments. Therefore, to reflect validity, and to permit greater generalizability, the protocol explicitly permitted continuation of usual health care for <u>all</u> participants. Physicians were free to offer appointments, to prescribe, and to maintain/discontinue prescriptions. Aside from this, TAU alone participants comprised, effectively, a wait-list group who completed measures but received no additional help for their insomnia. After the trial was completed TAU and IRT

participants were offered access to the CBT online intervention. The only contact received by the TAU alone arm was reminders to complete evaluation at baseline and the two follow-up points.

Data management and analysis

The study was designed to have 80% power to detect a medium effect size (Cohen, 1988) consistent with published meta-analytic data (for a review see Riemann & Perlis, 2009), based upon a 3-group ANOVA model with fixed effects, main effects and interactions, on the primary outcome measure of SE (cf. Espie et al., 2012). These criteria implied recruiting a total sample of 159 participants. All comparisons were planned and tests were two-sided, with $p<.05$ considered to indicate statistical significance. Where appropriate, to control for multiple comparisons, a per family error rate was adopted to maintain the nominal error rate (.05/ *n* of comparisons).

Analyses were performed using PASW Statistics 18 (Norusis, 2011; SPSS Inc., Chicago, Ill). Treatment effects were assessed using Linear mixed effects models, which permitted appropriate handling of missing data (cf. Olsen, Stechuchak, Edinger, Ulmer, & Woolson, 2012), predicting mean values at each assessment point (baseline, post-treatment, 8-week follow-up). In each model, time and treatment group were included as fixed effects, with time and group x time interaction terms. For variables exhibiting between group differences at baseline, the baseline value was entered as a covariate.

[Insert Table 1]

Results

Participant characteristics

Descriptive, demographic and clinical information on the allocated sample of 164 (120 F) adults [mean age 49y (18–78y)] is provided in Table 1. Approximately two-thirds were employed either full-time or part-time. Post-code data were used as a proxy for socio-economic status by deriving an Index of Multiple Deprivation (IMD). Mean IMD was 16.7 (SD = 11.8) indicating that the sample was somewhat less deprived (by <0.5 SD) than

national norms 21.7 (SD = 15.5: Office for National Statistics, 2007). Participants were generally healthy, endorsing stable average or better health (as per selection criteria), although 51 (31%) and 17 (10%), respectively, took prescription medication for a physical or mental health problem. A total of 33 participants (20%) sometimes used prescribed sleeping pills, and 63 (38%) made at least some use of over-the-counter sleep aids.

All participants had Insomnia Disorder, the great majority being of difficulty maintaining sleep or of mixed insomnia sub-types. Baseline sleep characteristics indicated current insomnia in the severe clinical range, with average total wake time per night (SOL+WASO) of ~2¼ hours and mean sleep efficiency of 61% (Table 1). Average estimated total sleep time was a little over 5 hours. Consistent with insomnia diagnostic criteria, substantial impact was observed also on daytime performance and social functioning. Two-thirds had insomnia for over 6 years, and almost 50% for over 11 years. Participants randomized to CBT (n = 55), IRT (n = 55) and TAU (n = 54) were similar in all demographic and clinical respects, with no significant differences on any baseline descriptor. Eight of the 164 participants did not start their treatment so the final sample receiving the allocated intervention was 156 [CBT = 53, IRT = 52, TAU = 51]. There were no significant differences on any variable between this sample and the allocated sample of 164.

Treatment attrition and integrity

Of those receiving their allocated intervention, 43 (82%) CBT participants completed all their online therapy sessions and 47 (88%) completed ≥4sessions. This compared to 41 (79%) and 48 (92%) respectively in the IRT group. Thus, there were similar, modest, levels of attrition during the treatment phase. Just under 80% of participants (n = 125) completed the follow-up assessment at 8 weeks. Reasons for withdrawal during treatment are summarized (where known) in Figure 1. There were no significant differences between treatment completers (defined as completing ≥ 4

sessions) and those who dropped out, on any demographic, clinical or sleep variable. Participants in the CBT group took an average of 50 days to complete the course compared with 48 in IRT, with TAU participants typically taking one more week to complete (58 days). No harm-related or serious adverse events were reported by participants.

Baseline characteristics

Of the four SDQ domains, the highest baseline value was for 'mental arousal' (M = 9.15, SD = 2.75) relative to 'trying too hard' (M = 6.74, SD = 2.75: $t(163) = 11.00$, $p < .001$), 'unable to relax' (M = 6.14, SD = 2.38: t(163) = 14.81, p < .001) and 'lack of routine' (M = 5.89, SD = 2.34: $t(163) = 14.30$, $p < .001$). Reports of 'trying to sleep' were also greater than lack of routine' ($t(163) = 4.10$, $p < .001$) and being 'unable to relax' ($t(163) = 2.93$, $p = .004$). SDQ domains inter-correlated very modestly ($r = .24$-$.49$), representing 6-24% of shared variance (R^2). At the item level, '*my mind keeps turning things over*' (mental arousal domain) was the most strongly endorsed single item on the SDQ with 77.3% rating this as a problem 'often' or 'very often'. For the other subscales, the highest scoring individual items were '*I can't get my sleep pattern into a proper routine*' (61.8%: 'lack of routine' subscale), '*I worry that I won't cope tomorrow if I don't sleep well*' (50.0%: 'trying to sleep') and '*I find it hard to physically "let go" and relax my body*' (44.6%: 'unable to relax').

On the GCTI, participants exhibited higher baseline scores for 'sleep and sleeplessness' (M = 7.89, SD = 2.63) and 'rehearsal and planning' thoughts (M = 7.62, SD = 2.65) compared to 'heightened awareness' thoughts (M = 5.24, SD = 2.31) [$t(163) = 12.83$, $p < .001$ and $t(163) = 10.74$ respectively, both $p < .001$]. There was no significant difference between rehearsal and planning and sleep thoughts, and the GCTI factors again moderately correlated ($r = .35$-$.43$, approximate $R^2 = 15\%$). Thinking about '*how long I've been lying awake'* was the item that was most commonly rated 'often' or 'very often' (by 64.6% of participants: loading on the 'sleep thoughts' subscale). Thinking

about '*what happened today and what I've got on tomorrow*' received the strongest endorsement of the items on the 'rehearsal and planning' subscale (58.2%) and concern about '*trivial things of no importance that go through my mind*' (42.7%) was the most prominent of the 'awareness thoughts'.

Consistent with our selection criteria, there was only modest baseline symptomatology on the DASS, with stress scores being significantly higher than depressive [M = 7.80, SD = 3.70 vs. M = 5.05, SD = 3.01; $t(163) = 11.1$, $p < .001$] or anxiety [M = 2.70, SD =2.20, $t(163) = 21.0$, $p < .001$] scores, and depressive scores being higher than anxiety scores [$t(163) = 11.5$, $p < .001$]. Compared with the SDQ and GCTI, the DASS subscales were more strongly inter-correlated (DASSdep/ DASSanx, $r = .53$; DASSdep/ DASSstress, $r = .55$; DASSanx/ DASSstress, $r = .57$: approximate $R^2 = 30\%$).

One-way ANOVA between treatment group comparison revealed differences in pre-treatment scores for the SDQ variables 'unable to relax' [$F(2,161) = 4.34$, $p = .015$], 'mental arousal' [$F(2,161) = 3.84$, $p = .024$] and for 'heightened awareness' [$F(2,161) = 3.73$, $p = .026$] on the GCTI, with a marginally non-significant effect for DASSanx ($p = .052$). Consequently, baseline values were introduced conservatively as covariates in subsequent hypothesis testing on these variables. The dependent variables exhibited no significant association with age, gender, socioeconomic status or any other sample demographic. Similarly, no relationship with insomnia duration or sleep pattern characteristic was observed. All ten SDQ, GCTI and DASS variables were somewhat associated with overall sleep condition on the SCI (range of $r = -.21$ to $-.35$, $R^2 \leq 12\%$).

[Insert Table 2]

Impact of treatment on sleep-related attributions

Summary data comprising pre-treatment, post-treatment and follow-up mean (SE) values for each group are presented in Table 2. Change scores (with 95% CI) and within group effect sizes (ES) are also provided. ES were regarded as large ($d = 0.8$),

moderate (d = 0.5) or small (d = 0.3), consistent with recognized definitions (Cohen, 1988). In Table 3, relative ES, representing changes over baseline observed at post-treatment and follow-up, are provided for each treatment comparison (CBT-TAU, IRT-TAU, CBT-IRT).

For all four sleep attribution variables, significant effects (summarized below) were observed, and these remained highly significant when taking account of baseline values. The mixed effects model confirmed a significant main effect for time [range of $F(2,263)$ = 13.00-49.9, all $p < .001$] and for the treatment x time interaction for all the SDQ variables. These latter represent the hypothesis-testing analyses so will be taken in turn.

For 'unable to relax', between group comparison favored CBT at post-treatment [$F(4,263) = 3.12$, $p = .016$] relative to both TAU (d = -0.72) and IRT (d = -0.56) (see Table 3). These effects were maintained at follow-up, but with smaller standardized ES. For 'lack of routine', CBT was again superior [$F(4,266) = 4.30$, $p = .002$] to TAU and IRT (moderate to large ES) at post-treatment, and at follow up). 'Mental arousal' [$F(4,270) = 4.65$, $p = .001$] and 'trying too hard' [$F(4,275) = 8.45$, $p < .001$] also exhibited significant interaction terms. For 'mental arousal', CBT was associated with stronger effects than TAU at post-treatment (d = -0.90) and follow-up (d = -0.54). The CBT-IRT comparison was also significant at each measurement point (d = -0.64 and d = -0.19). 'Trying too hard' reduced significantly following CBT compared with TAU (d = -1.15) and IRT (d = -0.76), and similar magnitude of effects were maintained 8 weeks post-treatment (Table 3). These latter variables also exhibited a small placebo response (IRT-TAU) at follow-up ['mental arousal' (d = -0.40) and 'trying too hard' (d = -0.35)].

Applying a Bonferonni correction to maintain the .05 error rate across the SDQ variables (adjusted $p = .0125$) would result in the treatment x time interaction for one outcome, being 'unable to relax', failing to attain statistical significance.

[Insert Table 3 here]

Impact of treatment on night-time thinking

Comparative data on the GCTI at baseline, post-treatment and follow-up are presented in Table 2. Main effects of time were observed for all variables [range of $F(2,264) = 14.33\text{-}54.20$, all $p < .001$]. The group x time interaction was strongest for thoughts about 'sleep and sleeplessness' [$F(4,266) = 10.92$, $p < .001$], associated with improvement in CBT relative to TAU after treatment and at 8 weeks (both $d \geq 1.00$); and by CBT relative to IRT at these measurement points (d = -0.74 and -0.56 respectively: Table 3) Superior outcomes were also demonstrated for CBT on 'rehearsal and planning' [$F(4,264) = 3.15$, $p = .015$] and 'heightened awareness' [$F(4,263) = 2.41$, $p = .049$]. Moderate effects for 'rehearsal and planning' thoughts relative to both groups were maintained at follow-up particularly for the CBT-TAU comparison (Table 3). A similar pattern was observed for 'heightened awareness' with ES in the small to moderate range, however, it should be noted that the interaction term for this variable was no longer significant following conservative correction for multiple GCTI comparisons (.05/3; $p = .017$). Response to IRT, though inferior to CBT, was moderate for thoughts about 'sleep and sleeplessness' at post-treatment (d = -0.42) and follow-up (d = -0.60) relative to TAU. Small effects in favor of IRT relative to TAU were also obtained for the two other GCTI variables at follow-up.

Impact of treatment on symptoms of psychopathology

Main effects of time were obtained for DASSdep [$F(2,322) = 16.71$, $p < .001$] and DASSstress [$F(2,337) = 26.75$, $p < .001$] with interaction effects also significant on these two variables (respectively: [$F(4,322) = 5.91$, $p < .001$] and [$F(4,337) = 3.90$, $p = .004$]). Relative ES at post-treatment were generally small and favoring CBT over TAU and IRT (Table 3). At follow-up, ES had strengthened for the CBT-TAU and the IRT-TAU comparisons, with small additional benefits of CBT over IRT on DASSdep (d = -0.29) and DASSstress (d = -0.26).

Association of change in attribution, cognition and psychopathology with sleep improvement

Sleep diary data from the trial are reported elsewhere (Espie et al., 2012) with summary data presented in Table 1. It was of interest here to consider how change on the psychological variables reported in this paper associated with the more direct benefits of CBT to insomnia symptoms per se. This was investigated in relation to the trial's primary end-point of sleep efficiency, and with respect to the Sleep Condition Indicator score, our overall index of sleep health, taking into account both night-time and day-time aspects of insomnia disorder. Accordingly, the SDQ, GCTI and DASS change scores (baseline to post-treatment, baseline to follow-up: presented in Table 1) were correlated with change scores for the SCI and for SE.

The results of this analysis support a stronger change score relationship with our global (SCI) measure than with sleep efficiency per se. Post-treatment improvement on the SCI was moderately associated with reduction in the SDQ subscales 'trying too hard' (r = -.49), 'unable to relax' (r = -.44), 'mental arousal' (r = -.42) and to a lesser extent 'lack of routine' (r = -.28) [all p <.001]. Comparable results were obtained at follow-up except that the association between sleep improvement and 'lack of routine' had strengthened (r = -.45). By contrast, smaller inverse correlations were observed with SE (range of r = -.15 to -.21; p = .08 to .015), the strongest of these being with 'lack of routine' at follow-up. Likewise, on the GCTI, there was an association between reduced night-time thoughts about 'sleep and sleeplessness' and SCI improvement (r = -.47 at post-treatment, r = -.51 at follow-up) and weaker change relationships with 'heightened awareness' (r = -.31) and 'rehearsal and planning' (r = -.28) [all p < .001]. Again, changes in SE were less closely mirrored by cognitive change; the strongest association being with reduced thoughts about 'sleep and sleeplessness' at post-treatment (r = -.23, p = .008). Reduction in DASS depressive and stress symptoms were also associated

with SCI score improvement (*r* = -.39 and -.41 respectively) at follow-up, but there were no significant associations with sleep efficiency.

Discussion

Consistent with the formulation of insomnia as a psychophysiological disorder, our participants reported particular problems with mental arousal in bed. The characteristically "racing mind" phenomenon was borne out by over 75% attributing poor sleep to being unable to empty their mind. The next strongest attribution was trying too hard to sleep. Concerns about having a poor sleep pattern were also endorsed by almost two-thirds and a significant minority had trouble relaxing (45%). These attributions are not, of course, mutually exclusive, and we observed a stable though modest inter-correlation across SDQ item scores. Data from the GCTI complemented these findings with thoughts about sleep and sleeplessness (e.g. how long I've been lying awake: 65%) and rehearsing the day past and planning ahead (58%) being very common experiences. Some participants also reported a hyper-awareness at night whereby they were conscious of their environment or internal state.

The complaint of insomnia, therefore, may be as much to do with such phenomena as it is to do with concern about sleep pattern problems per se. Indeed, the experiences seem likely to go hand-in-hand, because the individual is both lying awake (measurable in minutes) and thinking or worrying (measurable by such self-report scales). Importantly, CBT may directly impact upon each of these. Sleep restriction, for example, may increase homeostatic pressure for sleep by extending wakefulness (Pigeon & Perlis, 2006; Spielman, Saskin, & Thorpy, 1987), helping to over-ride pre-sleep arousal through the rapid induction and consolidation of sleep (Kyle et al., 2011).

Cognitive strategies on the other hand may reduce the pressure for mental arousal (e.g. putting the day to rest) or obviate its effects (e.g. cognitive restructuring, mindfulness) [Espie & Kyle, 2009]. This approach is also consistent with the perspective

that people with psychophysiological insomnia may benefit from interventions that combat hyperarousal and/ or facilitate the down-regulation of arousal, whether expressed autonomically (Bonnet & Arand, 2010), cortically (Perlis, Giles, Mendelson, Bootzin, & Wyatt, 1997) or mentally (Espie, 2002).

Trial methodologies do not, of course, permit firm conclusions to be drawn about *mechanisms* of treatment effect. Nevertheless our outcome data on the SDQ, GCTI and DASS are at least consistent with such mediation. Moreover, the fact that we had a quasi-desensitization placebo arm may offer greater confidence in the interpretation of self-reported change on what are essentially psychological variables. We will start therefore by considering the placebo response, which was evident for most of the SDQ and GCTI subscales. The relative ES for the IRT-TAU comparison were typically small in magnitude (mean d = -0.33). Nevertheless, these results do indicate that non-specific factors were associated with some improvement. Likewise, therefore, these could have been a factor in other online CBT studies, without a placebo arm, where significant improvements were reported on psychological measures (Ritterband et al., 2009; Strom et al., 2004; Lancee et al., 2011; Vincent & Lewycky, 2009). Interestingly, there were no comparable placebo effects on self-report measures of sleep (Espie et al., 2012). There are several possible explanations for our findings here. Perhaps any intervention that is perceived to address the self-management of sleep may be likely to induce at least some psychological change. Alternatively, participants knew that the trial was evaluating psychological intervention, and so they may have been predisposed first to take part; and second to respond in these domains. A third possibility, and also consistent with our rationale for conducting a placebo controlled trial, is that the experience of our online medium would be intrinsically engaging and would produce some effects in its own right. A final possibility is that our placebo may have been neutral in relation to sleep-related effects, but somehow active in relation to non-specific factors.

Turning to the CBT intervention, it is important to recognise that CBT was

associated with significant improvement over and above the effects of the placebo condition. Significant effects on the CBT-IRT comparison were consistently observed, although relatively small in magnitude (average *d* = -0.32), with the overall effect relative to TAU being moderate to large (*d* = -0.65). From this, we can conclude that CBT impacted upon attribution and cognition beyond the effects of placebo alone. Strengthening the importance of these findings, we also show that changes in attribution, cognition and psychopathology were moderately and significantly related to improvements in global sleep quality (using the SCI), though tended to be weakly related to improvements in sleep efficiency. It is perhaps not surprising that such changes were more aligned with this overall appraisal of sleep, given that the SCI comprehensively captures core features of the insomnia experience (quantitative and qualitative aspects of sleep, as well as daytime functioning). Though not possible to determine cause and effect, it might be expected that improvements in both sleep (quality and quantity) and daytime functioning will help modify the nature of sleep-related cognitions and attributions. Of course, the converse direction is also plausible and the present design cannot permit teasing out of competing explanations. Interestingly, Edinger et al. (2001), in a placebo-controlled trial of face-to-face CBT-I, also found that reductions in dysfunctional beliefs and attitudes about sleep were more strongly related to improvements in global insomnia severity (r= .60) than sleep efficiency values (r= -.25).

We excluded any potential participants who rated their mental health as “poor” or “very poor”. Consequently, our data on psychopathology are limited. Nevertheless, the DASS is a dimensional symptom measure, and permits some insights. We found that depressive and stress symptoms were more problematic at baseline than anxiety symptoms. Unsurprisingly, therefore, we did not observe much in the way of change in the latter. On the other hand, both depressive and stress symptom levels reduced following both CBT and IRT. At the eight week follow-up point, these effects were moderate to large for CBT, and moderate for IRT, with the additional effects of the CBT-

IRT comparison significant but small in magnitude (for DASS dep *d* = -0.29; for DASS stress *d* = -0.26). Future studies should recruit co-morbid clinical samples, with greater degrees of psychopathology, to determine the extent to which online CBT has generalisable effects beyond improvement of sleep parameters.

This work is not without limitation. Our sleep-related outcome measures, though validated with insomnia patients, have not been used in online treatment outcome studies; therefore, comparison with existing intervention trials is not possible. In general, insomnia treatment studies have tended to focus on night-time sleep parameters, frequently neglecting daytime functioning and treatment process measures. Future, adequately controlled, online (and face-to-face) CBT-I studies should incorporate similar measures to the present study, in addition to assessments of other relevant constructs (e.g. pre-sleep cognitive and somatic arousal [Nicassio et al., 1989], sleep effort [Broomfield & Espie, 2005], dysfunctional beliefs and attitudes about sleep [Morin et al., 2007]).

Another limitation is our relatively short-term follow-up. Face-to-face CBT-I studies often include follow-ups between 6 and 12 months and it will be important to determine the durability of online CBT-related treatment effects in future studies. Related to this, inspection of effect sizes at follow-up reveals a general weakening of treatment effects (CBT v. IRT, relative effects) and the emergence of small (within-subject) effects for the TAU group. Such patterning of data may imply a small seasonal effect on sleep (cf. Espie et al., 2012), with improvements being observed over the months when the trial was conducted [i.e. late winter (Feb) through to spring (May)].

In conclusion, this work indicates that online CBT for insomnia disorder modifies sleep-related attributions, night-time thought content and psychopathology, over and above a placebo intervention, and that these changes are associated with improvements in global sleep quality. The trial design applied herein raises the bar for what should be considered the “gold standard” in determining the true benefits of online CBT-I

interventions.

Acknowledgements
No payments were made to any of the participants in this study. We wish to acknowledge and thank them for their interest in and commitment to this research.

References

American Psychiatric Association (2010) DSM-5 development. Available from: http://www.dsm5.org/.

American Academy of Sleep Medicine. (2005). *The international classification of sleep disorders: Diagnostic and coding manual* (2nd ed.). Westchester, IL: Author.

Baglioni, C., Battagliese, G., Feige, B., Spiegelhalder, K., Nissen, C., Voderholzer, U., et al. (2011). Insomnia as a predictor of depression: A meta-analytic evaluation of longitudinal epidemiological studies. *Journal of Affective Disorders*, 135, 10-19.

Bennett-Levy, J., Richards, D., Farrand, P., Christensen, H., Griffiths, K., Kavanagh, D. et al. (2010). *Oxford Guide to Low Intensity CBT Interventions* (Oxford Guides in Cognitive Behavioural Therapy). Oxford, UK: Oxford University Press.

Bonnet, M. H., & Arand, D. L. (2010). Hyperarousal and insomnia: state of the science. *Sleep Medicine Reviews*, 14, 9-15.

Bootzin, R.R., & Nicassio, P.M. (1978). *Progress of behaviour modification*. New York, NY: Academic Press.

Borkovec, T. D. (1982). Insomnia. *Journal of Consulting and Clinical Psvchology*, 50, 880-895.

Bower, P., & Gilbody, S. (2005). Stepped care in psychological therapies: access, effectiveness and efficiency. *British Journal of Psychiatry*, 186, 11-17.

Broomfield, N.M., & Espie, C.A. (2005). Towards a valid, reliable measure of sleep effort. *Journal of Sleep Research*, 14, 401-407.

Buysse, D.J., Ancoli-Israel, S., Edinger, J.D., Lichstein, K.L. & Morin, C.M. (2006).Recommendations for a Standard Research Assessment of Insomnia. *Sleep,* 29, 1155-73.

Buysse, D.J., Reynolds III, C.F., Monk, T.H., Berman, S.R. & Kupfer, D.J. (1989). The Pittsburgh Sleep Quality Index: a new instrument for psychiatric practice and research. *Psychiatry Research*, 28, 193–213.

Cohen, J. Statistical power analysis for the behavioral sciences (2nd ed.). Hillsdale, NJ: Erlbaum, 1988.

Cole, M.G., & Dendukuri, N. (2003). Risk factors for depression among elderly community subjects: a systematic review and meta-analysis. *American Journal of Psychiatry*, 160, 1147-56.

Edinger, J.D., Wohlgemuth, W.K., Radtke, R.A., Marsh, G.R., & Quillian, R.E. (2001). Cognitive behavioural therapy for treatment of chronic primary insomnia: A randomized controlled trial. *Journal of the American Medical Association*, 285, 1856-1864.

Edinger, J.D., Bonnet, M.H., Bootzin, R.R., Doghramji, K., Dorsey, C.M., Espie, C.A., et al. (2004). Derivation of research diagnostic criteria for insomnia: report of an American Academy of Sleep Medicine workgroup. *Sleep*, 27, 1567-1596.

Espie, C.A., MacMahon, K.M.A, Kelly, H., Broomfield, N.M., Douglas, N.J. Engleman, H.M., McKinstry, B., et al. (2007). Randomized Clinical effectiveness trial of nurse-administered small-group cognitive behaviour therapy for persistent insomnia in General practice. *Sleep*, 30, 574-584.

Espie, C.A., Fleming, L., Cassidy, J., Samuel, L., Taylor, L.M., White, C.A., et al. (2008). Randomized controlled clinical effectiveness trial of cognitive behaviour therapy compared with treatment as usual for persistent insomnia in patients with cancer. *Journal of Clinical Oncology*, 26, 4651-58.

Espie, C.A., Inglis, S.J, & Harvey, L. (2001). Predicting clinically significant response to cognitive behavior therapy for chronic insomnia in general medical practice: Analysis of outcome data at 12 months post treatment. *Journal of Consulting and Clinical Psychology*, 69, 58-66.

Espie, C.A., Inglis, S.J., Harvey, L,, Tessier, S. (2000). Insomniacs' attributions: Psychometric properties of the Dysfunctional Beliefs and Attitudes about Sleep scale and the Sleep Disturbance Questionnaire. *Journal of Psychosomatic Research*, 48, 141-148.

Espie, C.A., Kyle, S.D., Chylarova, E., Hames P., Benzeval, M. (under review) The daytime impact of DSM-V insomnia disorder: comparative analysis of insomnia subtype from the Great British Sleep Survey (n=11,129).

Espie, C.A., Lindsay, W.R., Brooks, N., Hood, E.M. & Turvey, T. (1989). A controlled comparative investigation of psychological treatments for chronic sleep-onset insomnia. *Behaviour Research and Therapy*, 27, 79-88.

Espie, C.A. (2002). Insomnia: conceptual issues in the development, persistence, and treatment of sleep disorder in adults. *Annual Review of Psychology*, 53, 215-43.

Espie, C.A. (2009). 'Stepped care': a health technology solution for delivering Cognitive Behavioral Therapy as a first line insomnia treatment. *Sleep*, 32, 1549-1558.

Espie, C.A., Brooks, D.N., & Lindsay, W.R. An evaluation of tailored psychological treatment for insomnia in terms of statistical and clinical measures of outcome. *Journal of Behavior Therapy and Experimental Psychiatry*, 20, 143-153.

Espie, C.A., & Kyle, S.D. (2009). Primary insomnia: an overview of practical management using cognitive behavioural techniques. *Sleep Medicine Clinics*, 4, 559-569.

Gardani, M., Miller, C.B., Biello, S., Ellis, J., Von Schantz, M., & Archer S. (2011). The association of sleepiness and diurnal preference with salivary amylase activity. Abstracts of 4th International Congress of WASM & 5th Conference of CSS / Sleep Medicine, Suppl. 1, S1–S130.

Green, M.J., Espie, C.A., Hunt, K., & Benzeval, M. (2012). The longitudinal course of insomnia symptoms: inequalities by sex and occupational class among two different age cohorts followed for 20 years in the west of Scotland. *Sleep*, 35, 815-823.

Harris, J., Lack, L., Wright, H., Gradisar, M., & Brooks, A. (2007). Intensive Sleep Retraining treatment for chronic primary insomnia: a preliminary investigation. *Journal of Sleep Research*, 16, 276-284.

Harvey, K.J., & Espie, C.A. (2004). Development and preliminary validation of the Glasgow Content of Thoughts Inventory (GCTI): a new measure for the assessment of pre-sleep cognitive activity. *British Journal of Clinical Psychology*, 43, 409-420.

Harvey, A.G. (2002). A cognitive model of insomnia. *Behaviour Research and Therapy*, 40, 869-893.

Harvey, A.G., Schmidt, A., Scarna, A., Semler, C.N., & Goodwin, G.M. (2005). Sleep-related functioning in euthymic patients with bipolar disorder, patients with insomnia, and subjects without sleep problems. *American Journal of Psychiatry*, 162, 50-57.

Hauri, P., & Fisher, J. (1989). Persistent psychophysiological (learned) insomnia. *Sleep*, 9, 38-53.

Henry, J.D., & Crawford, J.R. (2005). The short-form version of the Depression Anxiety Stress Scales (DASS-21): Construct validity and normative data in a large non-clinical sample. *British Journal of Clinical Psychology*, 44, 227-239.

Irwin, M.R., Cole, J.C., & Nicassio, P.M. (2006). Comparative meta-analysis of behavioral interventions for insomnia and their efficacy in middle-aged adults and in older adults 55+years of age. *Health Psychology*, 25, 3-14.

Kyle, S.D., Morgan, K., & Espie, C. (2010). Insomnia and health-related quality of life. *Sleep Medicine Reviews*, 14, 69-82.

Kyle, S.D., Morgan, K., Spiegelhalder, K., & Espie, C.A. (2011). No pain, no gain: an exploratory within-subjects mixed-methods evaluation of the patient experience of sleep restriction therapy (SRT) for insomnia. *Sleep Medicine*, 12, 735-47.

Lancee, J., van den Bout, J., van Straten, A., & Spoormaker, V.I. (2012). Internet-delivered or mailed self-help treatment for insomnia? A randomized waiting-list controlled trial. *Behaviour Research and Therapy*, 50, 22-29.

Lichstein, K.L., Durrence, H.H., Reidel, B.W., Taylor, D.J., & Bush, A.J. (2004). *The Epidemiology of Sleep: Age, Gender and Ethnicity*. Mahwah, NJ: Lawrence Erlbaum Associates.

Lichstein, K.L., Durrence, H.H., Taylor, D.J., Bush, A.J., & Riedel, B.W. (2003). Quantitative criteria for insomnia. *Behaviour Research and Therapy*, 41, 427-445.

Manber, R., Edinger, J.D., Gress, J.L., San Pedro-Salcedo, M.G., Kuo, T.F., & Kalista, T. (2008). Cognitive behavioral therapy for insomnia enhances depression outcome in patients with comborbid major depressive disorder and insomnia. *Sleep*, 31, 489-495.

Mitchell, M. C., Burns, N. R., & Dorstyn, D. S. (2008) Screening for depression and anxiety in spinal cord injury with DASS-21. *Spinal Cord*, 46, 547-551.

Morin, C.M., Belanger, L., LeBlanc, M., Ivers, H., Savard, J., Espie, C.A., et al. (2009). The natural history of insomnia: A population-based 3-year longitudinal study. *Archives of internal Medicine*, 169, 447-453.

Morin, C.M., Bootzin, R.R., Buysse, D.J., Edinger, J.D., Espie, C.A., & Lichstein, K.L. (2006). Psychological and behavioral treatment of insomnia: Update of the recent evidence (1998-2004). *Sleep*, 29, 1398-414.

Morin, C.M., Vallieres, A. & Ivers, H. (2007). Dysfunctional beliefs and attitudes about sleep (DBAS): validation of a brief version (DBAS-16). *Sleep*, 30, 1547-54.

Morin, C.M., & Espie, C.A. (2003). *Insomnia: A clinical guide to assessment and treatment*. New York: Kluwer Academic/Plenum Publishers.

Morin, C.M. (1993). *Insomnia: Psychological assessment and management*. New York: Guilford.

Morin, C.M. (2003). Measuring outcomes in randomized clinical trials of insomnia treatments. *Sleep Medicine Reviews*, 7, 263-279.

Nicassio, P.M., Mendlowitz, D.R., Fussell, J.J., & Petras, L. (1989). The phenomenology of the pre-sleep state: the development of the pre-sleep arousal scale. *Behaviour Research and Therapy*, 23, 263-271.

National Institutes of Health (NIH) state-of-the-science conference statement on manifestations and management of chronic insomnia in adults, June 13-15, 2005. *Sleep*, 28, 1049-1057.

Norusis MJ, Inc. SPSS. *PASW Statistics 18 Advanced Statistical Procedures Companion*. Chicago: SPSS Inc., 2011.

Ohayon, M.M. (2002). Epidemiology of insomnia: what we know and what we still need to learn. *Sleep Medicine Reviews*, 6, 97-111.

Olsen, M.K., Stechuchak, K.M., Edinger, J.D., Ulmer, C.S., & Woolson, R.F. (2012). Move over LOCF: principled methods for handling missing data in sleep disorder trials. *Sleep Medicine*, 13, 123-132.

Perlis, M. L., Giles, D. E., Mendelson, W. B., Bootzin, R. R., & Wyatt, J. K. (1997). Psychophysiological insomnia: the behavioural model and a neurocognitive perspective. *Journal of Sleep Research*, 6, 179-188.

Pigeon, W.R., & Perlis, M.L. (2006). Sleep homeostasis in primary insomnia. *Sleep Medicine Reviews*, 10, 247-254.

Reynolds, C.F., & Redline, S. (2010). The DSM-V sleep-wake disorders nosology: An update and an invitation to the sleep community. *Journal of Clinical Sleep Medicine*, 15, 9-10.

Riemann, D., & Perlis, M.L. (2009). The treatments of chronic insomnia: A review of benzodiazepine receptor agonists and psychological and behavioral therapies. *Sleep Medicine Reviews*, 13, 205-214.

Ritterband, L.M., Bailey, E.T., Thorndike, F.P., Lord, H.R., Farrell-Carnahan, L., & Baum, L.D. (2012). Initial evaluation of an internet intervention to improve the sleep of cancer survivors with insomnia. *Psychooncology*, 21, 695-705.

Ritterband, L.M., Thorndike, F.P., Gonder-Frederick, G.A., Magee, J.C., Bailey, E.T., Saylor, D.K., et al. (2009). Efficacy of an Internet-Based Behavioral Intervention for Adults With Insomnia. *Archives of General Psychiatry*, 66, 692-698.

Schulz, K.F., Altman, D.G., Moher, D., for the CONSORT Group. (2010). CONSORT 2010 Statement: updated guidelines for reporting parallel group randomised trials. *Annals of Internal Medicine*, 152.

Spielman, A.J., Saskin, P. & Thorpy, M.J. (1987). Treatment of chronic insomnia by restriction of time in bed. *Sleep*, 10, 45-56.

Steinmark, S.W., & Borkovec, T.D. (1974). Active and placebo treatment effects on moderate insomnia under counterdemand and positive demand instructions. *Journal of Abnormal Psychology*, 83, 157-63.

Smith, S. & Trinder, J. (20001). Detecting insomnia: comparison of four self-report measures of sleep in a young adult population. *Journal of Sleep Research*, 10, 229-235.

Strom, L., Pettersson, R., & Andersson, G. (2004). Internet-based treatment for insomnia: a controlled evaluation. *Journal of Consulting and Clinical Psychology*, 72, 113–120.

Swift, N., Stewart, R., Andiappan, M., Smith, A., Espie, C.A., & Brown, J.S.L. (2012). The effectiveness of community day-long CBT-I workshops for participants with

insomnia symptoms: a randomised controlled trial. *Journal of Sleep Research*, 3, 270-280.

van Straten A, Cuijpers P. (2009). Self-therapy for insomnia: A meta-analysis. *Sleep Medicine Reviews*, 13, 61-71.

Vincent, N.,& Lewycky, S. (2009). Logging on for Better Sleep: RCT of the Effectiveness of Online Treatment for Insomnia. *Sleep*, 32, 807-815.

Wicklow, A., & Espie, C.A. (2000). Intrusive thoughts and their relationship to actigraphic measurement of sleep: towards a cognitive model of insomnia. *Behaviour Research and Therapy*, 38, 679-693.

Wilson, S.J., Nutt, D.J., Alford, C., Argyropoulos, SV, Baldwin, D.S., Bateson, A.N., et al. (2010). British Association for Psychopharmacology consensus statement on evidence-based treatment of insomnia, parasomnias and circadian rhythm disorders. *Journal of Psychopharmacology*, 24, 1577–1600.

Figure 1. Flow of Participants in the Trial

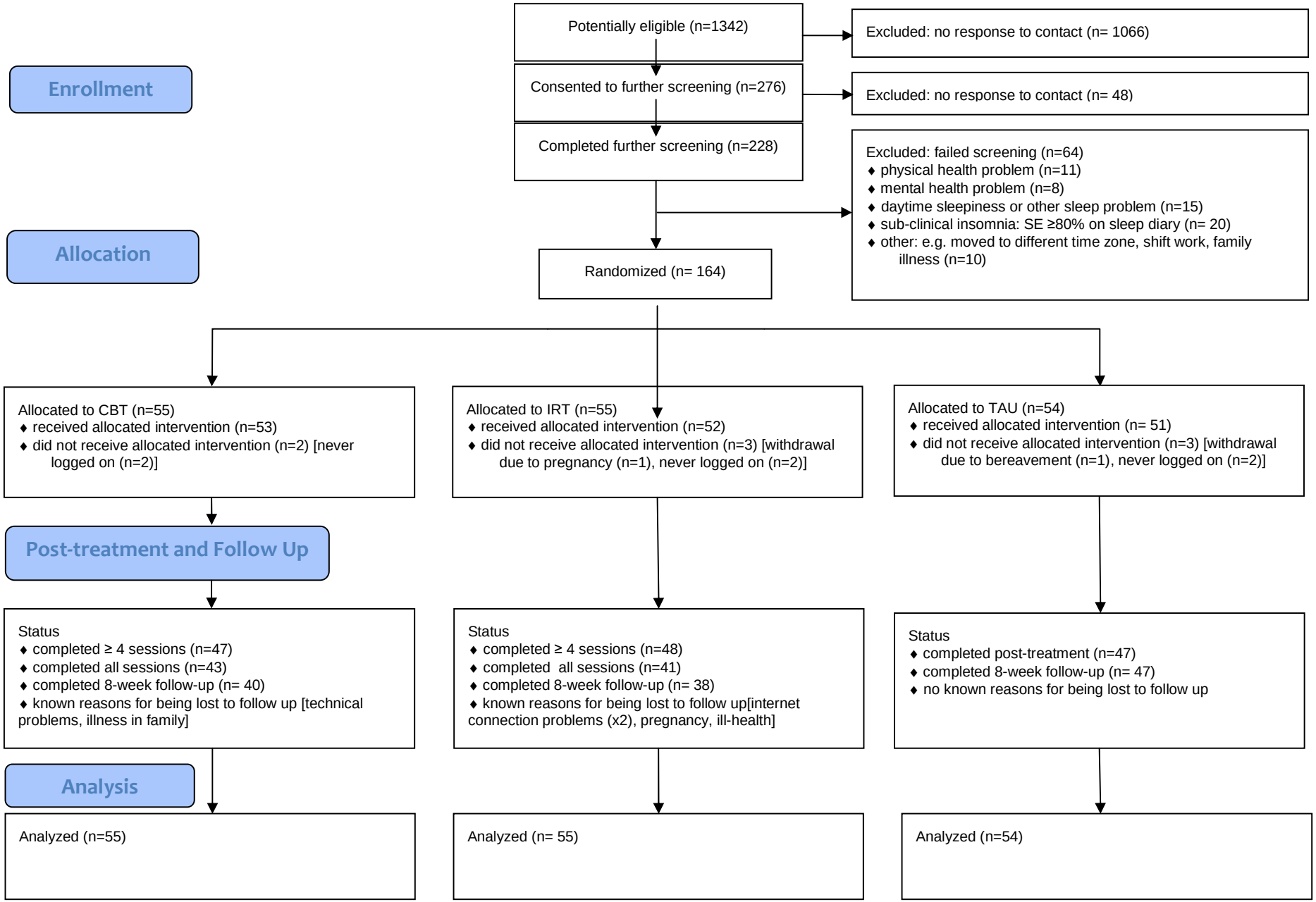


Table 1: Demographic and clinical characteristics of participants (n=164)

Characteristic	CBT (n = 55)	IRT (n = 55)	TAU (n = 54)	All (n = 164)
Age, mean (SD), y	50.7 (13.8)	47.3 (13.0)	49.1 (13.7)	49.0 (13.5)
Gender , No. (%)				
Female	40 (72.7)	42 (76.4)	38 (70.4)	120 (73.2)
Male	15 (27.3)	13 (23.6)	16 (29.6)	44 (26.8)
Occupation, No. (%)				
Employed. Full-time	20 (36.4)	25 (45.5)	20 (37.0)	65 (39.6)
Employed, part-time	17 (30.9)	12 (21.8)	11 (20.4)	40 (24.4)
Retired	13 (23.6)	8 (14.5)	16 (29.6)	37 (22.6)
Student	3 (5.45)	3 (5.45)	2 (3.70)	8 (4.88)
Not currently employed	2 (3.64)	7 (12.7)	5 (9.26)	14 (25.5)
Index of multiple deprivation, mean (SD)[a]	16.7 (11.3)	18.4 (13.8)	15.2 (10.4)	16.7 (11.8)
Civil status, No. (%)[b]				
No Partner	20 (36.4)	20 (36.4)	missing	missing
Partner	35 (63.6)	35 (63.6)	missing	missing
Physical health, No. (%)				
0 Very good	15 (27.3)	13 (23.6)	7 (13.0)	35 (21.3)
1 Good	25 (45.4)	26 (47.3)	37 (68.5)	88 (53.7)
2 Average	15 (27.3)	16 (29.1)	10 (18.5)	41 (25.0)
Mental health, No. (%)				
Very good	16 (29.1)	11 (21.0)	13 (24.1)	40 (24.4)
Good	25 (45.4)	23 (41.8)	28 (51.8)	76 (46.3)
Average	14 (25.5)	21 (38.2)	13 (24.1)	48 (29.3)
Total wake time, mean (SE), min	124.8 (8.9)	122.9 (5.7)	162.5 (13.4)	136.5 (5.7)
Hours of sleep, mean (SE), hr	5.1 (0.2)	5.5 (0.2)	4.7 (0.3)	5.1 (0.1)
Sleep efficiency, mean (SE), %	63.2 (2.1)	65.1 (1.3)	55.6 (2.9)	61.3 (1.3)
Sleep Condition Indicator, mean (SE)	3.06 (.14)	3.00 (.13)	2.79 (.16)	2.98 (.08)
Duration of insomnia, No. (%), y				
<2	9 (16.4)	7 (12.7)	6 (11.1)	22 (13.5)
2-5	13 (23.6)	16 (29.1)	5 (9.2)	34 (20.7)
6-10	10 (18.2)	13 (23.6)	9 (16.7)	32 (19.5)
≥11	23 (41.8)	19 (34.6)	34 (63.0)	76 (46.3)
Type of insomnia. No. (%)				
Difficulty Initiating Sleep	1 (1.8)	3 (5.4)	1 (1.9)	5 (3.0)
Difficulty Maintaining Sleep	26 (47.3)	22 (40.0)	22 (40.7)	70 (42.7)
Mixed (Initiating/ Maintaining)	22 (40.0)	24 (43.6)	26 (48.1)	72 (43.9)
Early Morning Awakening	4 (7.3)	3 (5.5)	3 (5.6)	10 (6.1)
Non-Restorative Sleep	2 (3.6)	3 (5.5)	2 (3.7)	7 (4.3)

[a] These data available only for postcodes in England (n=137)
[b]These data were not collected, in error, from the TAU group

Table 2: Treatment outcomes for measures of attribution, thought content and psychopathology. Baseline, post-treatment and follow-up date [mean (SE)] are presented for each group along with change scores (95% CI) and within group effect sizes (Cohen's *d*). [SDQ: Sleep Disturbance Questionnaire, GCTI: Glasgow Content of Thoughts Inventory, DASS: Depression, Anxiety, Stress Scale]

Variable	Treatment Group	Baseline Mean (SE)	Post-treatment Mean (SE)	Change from Baseline to Post-Treatment (95% CI)	*d*	8-wk Follow-up Mean (SE)	Change from Baseline to Follow-up (95% CI)	*d*
SDQ - Unable to relax	CBT	5.45 (0.31)	3.60 (0.38)	-1.85 (-2.57 to -1.01)	*-0.73*	4.03 (0.41)	-1.42 (-2.45 to -0.60)	*-0.49*
	IRT	6.22 (0.31)	5.90 (0.40)	-0.32 (-1.17 to 0.27)	*-0.14*	5.64 (0.35)	-0.58 (-1.61 to 0.70)	*-0.22*
	TAU	6.76 (0.33)	6.62 (0.36)	-0.14 (-1.22 to -0.44)	*-0.09*	6.21 (0.41)	-0.55 (-1.31 to -0.50)	*-0.26*
SDQ - Mental arousal	CBT	8.38 (0.42)	6.05 (0.52)	-2.33 (-3.30 to -1.63)	*-0.86*	6.15 (0.53)	-2.23 (-3.44 to -1.36)	*-0.69*
	IRT	9.29 (0.35)	8.60 (0.44)	-0.69 (-1.61 to 0.16)	*-0.25*	7.72 (0.39)	-1.57 (-2.77 to -1.07)	*-0.60*
	TAU	9.80 (0.32)	9.38 (0.35)	-0.42 (-0.93 to -0.17)	*-0.32*	8.87 (0.43)	-0.93 (-1.49 to -0.50)	*-0.55*
SDQ - Lack of routine	CBT	5.65 (0.32)	3.74 (0.40)	-1.91 (-2.69 to -1.31)	*-0.85*	3.65 (0.38)	-2.00 (-2.71 to -1.34)	*-0.93*
	IRT	6.11 (0.34)	5.28 (0.34)	-0.83 (-1.51 to -0.18)	*-0.40*	4.74 (0.34)	-1.37 (-1.85 to -0.62)	*-0.72*

	TAU	5.91 (0.29)	5.49 (0.32)	-0.42 (-1.03 to 0.06)	*-0.22*	5.23 (0.36)	-0.68 (-1.28 to -0.81)	*-0.33*
SDQ – Trying too hard	CBT	6.25 (0.35)	3.67 (0.42)	-2.68 (-3.47 to -2.11)	*-1.22*	3.53 (0.45)	-2.72 (-3.66 to -2.09)	*-1.11*
	IRT	7.24 (0.33)	6.40 (0.43)	-0.84 (-1.82 to -0.27)	*-0.34*	5.90 (0.40)	-1.34 (-2.44 to -0.74)	*-0.51*
	TAU	6.74 (0.35)	6.11 (0.37)	-0.63 (-1.07 to -0.78)	*-0.37*	5.96 (0.39)	-0.78 (-1.32 to -0.24)	*-0.43*
GCTI – Rehearsal and planning	CBT	7.00 (0.39)	5.60 (0.44)	-1.40 (-2.39 to -0.86)	*-0.56*	5.43 (0.52)	-1.57 (-2.78 to -0.82)	*-0.51*
	IRT	7.78 (0.36)	7.35 (0.40)	-0.43 (-0.86 to 0.51)	*-0.20*	6.56 (0.42)	-1.22 (-1.96 to -0.87)	*-0.42*
	TAU	8.09 (0.32)	7.94 (0.37)	-0.15 (-0.84 to 0.08)	*-0.10*	7.89 (0.43)	-0.20 (-0.92 to 0.28)	*-0.10*
GCTI - Sleep and sleeplessness	CBT	7.53 (0.36)	4.65 (0.48)	-2.88 (-3.94 to -2.34)	*-1.10*	4.05 (0.49)	-3.48 (-4.62 to -2.68)	*-1.14*
	IRT	8.25 (0.33)	6.98 (0.41)	-1.27 (-2.07 to -0.58)	*-0.55*	6.23 (0.38)	-2.02 (-2.89 to -1.32)	*-0.83*
	TAU	7.89 (0.35)	7.47 (0.40)	-0.42 (-0.95 to 0.01)	*-0.26*	7.32 (0.42)	-0.57 (1.35 to 0.11)	*-0.23*

GCTI – Heightened awareness	CBT	4.69 (0.29)	3.42 (0.34)	-1.27 (-1.80 to -0.48)	*-0.59*	3.33 (0.34)	-1.36 (-2.02 to -0.57)	*-0.60*
	IRT	5.16 (0.29)	4.78 (0.37)	-0.38 (-1.06 to 0.21)	*-0.19*	4.00 (0.30)	-1.16 (-1.83 to -0.53)	*-0.57*
	TAU	5.87 (0.33)	5.81 (0.23)	-0.06 (-0.86 to 0.30)	*-0.03*	5.64 (0.22)	-0.23 (-1.01 to 0.21)	*-0.11*

DASS - Depression	CBT	4.98 (0.40)	3.38 (0.42)	-1.60 (-0.79 to -2.41)	*-0.54*	2.30 (0.35)	-2.68 (-1.08 to -3.55)	*-0.85*
	IRT	4.81 (0.41)	4.04 (0.43)	-0.77 (-0.12 to -1.42)	*-0.33*	3.81 (0.46)	-1.00 (-0.21 to -1.79)	*-0.35*
	TAU	5.53 (0.46)	4.53 (0.45)	-1.00 (-0.14 to -1.86)	*-0.33*	5.47 (0.64)	-0.06 (0.93 to -1.05)	*-0.02*
DASS - Anxiety	CBT	2.32 (0.27)	1.74 (0.31)	-0.58 (-0.19 to -0.98)	*-0.40*	1.34 (0.21)	-0.98 (-0.50 to -1.46)	*-0.56*
	IRT	2.63 (0.30)	2.48 (0.29)	-0.15 (0.36 to -0.67)	*-0.08*	2.04 (0.26)	-0.59 (-0.13 to -1.06)	*-0.36*
	TAU	3.29 (0.35)	2.98 (0.36)	-0.31 (0.32 to -0.95)	*-0.14*	2.92 (0.38)	-0.37 (0.22 to -0.96)	*-0.18*
DASS - Stress	CBT	7.25 (0.53)	5.04 (0.54)	-2.21 (-1.31 to -3.10)	*-0.68*	4.36 (0.47)	-2.89 (-1.97 to -3.80)	*-0.87*
	IRT	8.31 (0.51)	7.13 (0.48)	-1.17 (-0.45 to -1.89)	*-0.46*	6.85 (0.48)	-1.46 (-0.58 to -2.34)	*-0.46*
	TAU	8.08 (0.51)	7.27 (0.50)	-0.81 (0.12 to -1.73)	*-0.25*	7.45 (0.56)	-0.63 (0.27 to -1.52)	*-0.20*

Table 3: Relative effect sizes (Cohen's *d*) for each treatment group comparison (CBT-TAU, IRT-TAU, CBT-IRT) at post-treatment and follow-up for the SDQ (Sleep Disturbance Questionnaire), GCT (Glasgow Content of Thoughts Inventory) and DASS (Depression, Anxiety, Stress Scale)

	Relative effect size (*d*) Pre-treatment to post-treatment			**Relative effect size (*d*) Pre-treatment to 8-wk Follow-up**		
Variable	**CBT-TAU**	**IRT-TAU**	**CBT-IRT**	**CBT-TAU**	**IRT-TAU**	**CBT-IRT**
SDQ						
Unable to relax	*-0.72*	*-0.09*	*-0.56*	*-0.33*	*-0.04*	*-0.28*
Mental arousal	*-0.90*	*-0.07*	*-0.64*	*-0.54*	*-0.40*	*-0.19*
Lack of routine	*-0.73*	*-0.17*	*-0.53*	*-0.64*	*-0.27*	*-0.39*
Trying too hard	*-1.15*	*-0.23*	*-0.76*	*-0.96*	*-0.35*	*-0.51*
GCTI						
Rehearsal and planning	*-0.60*	*0.10*	*-0.62*	*-0.57*	*-0.28*	*-0.26*
Sleep and sleeplessness	*-1.23*	*-0.42*	*-0.74*	*-1.09*	*-0.60*	*-0.56*
Heightened awareness	*-0.42*	*-0.07*	*-0.34*	*-0.42*	*-0.34*	*-0.05*
DASS						
Depression	*-0.20*	*0.08*	*-0.31*	*-0.78*	*-0.56*	*-0.29*
Anxiety	*-0.14*	*0.08*	*-0.26*	*-0.31*	*-0.22*	*-0.12*
Stress	*-0.43*	*-0.13*	*-0.36*	*-0.69*	*-0.44*	*-0.26*

Dissemination of evidence-based practice relating to insomnia

Insomnia: A Clinical Guide to Assessment and Treatment. Kluwer Academic/ Plenum Publishers, New York, 2003

Overcoming Insomnia and Sleep Problems: A Self-Help Guide Using Cognitive Behavioral Techniques. Constable & Robinson Ltd., London, 2006

International Neurology: A Clinical Approach. Wiley-Blackwell, Oxford, pp. 554-556, 2009

New Oxford Textbook of Psychiatry (Vol. 2) (second edition) Oxford University Press, Oxford, England pp. 933-938, 2009

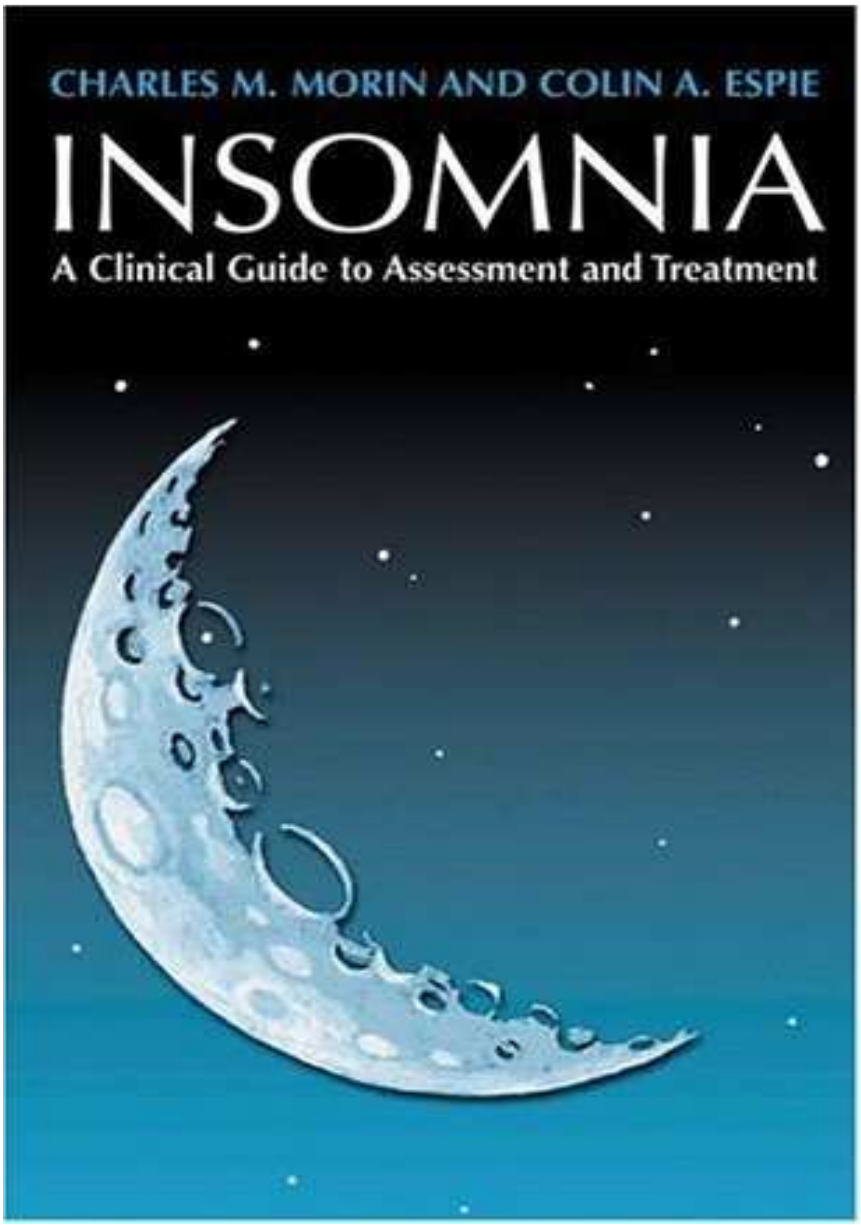
Principles and Practice of Sleep Medicine, 5th edition. Elsevier Saunders, NY, 2011

Introduction to Coping with Insomnia & Sleep Problems. Constable & Robinson Ltd., London, 2011

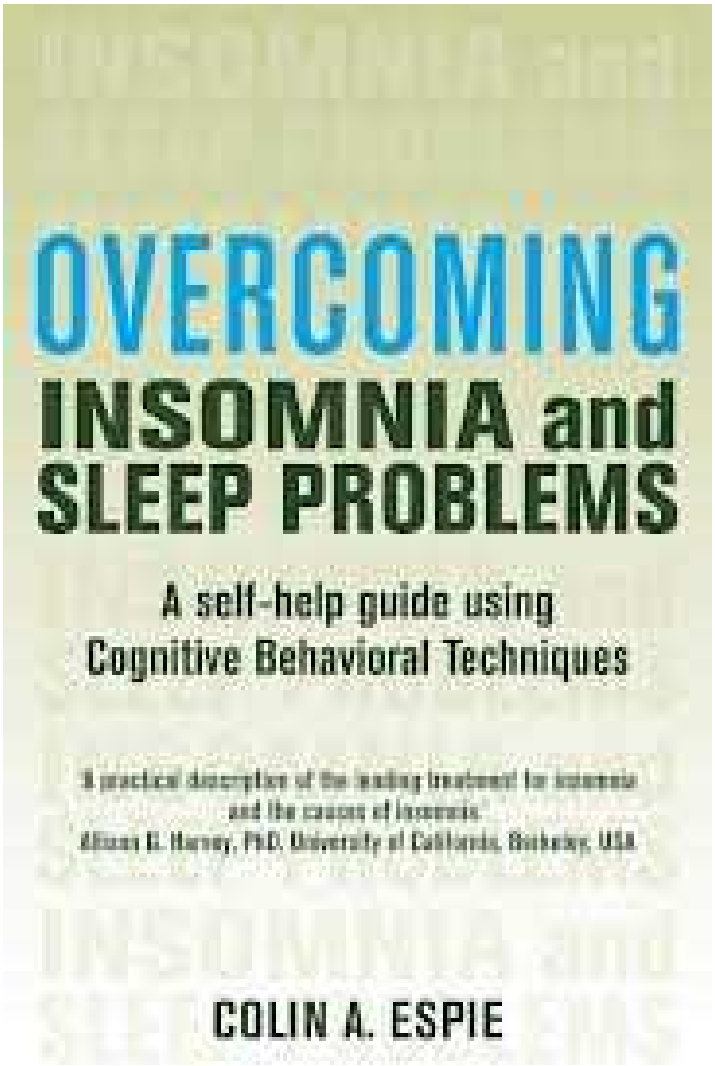
The Oxford Handbook of Sleep and Sleep Disorders (Oxford Library of Psychology) Oxford University Press, USA [928 pages], 2012

www.sleepio.com

Morin C.M and Espie, C.A. (2003) ***Insomnia: A Clinical Guide to Assessment and Treatment*****. Kluwer Academic/ Plenum Publishers, New York [ISBN 0-306-47750-5]**



Espie, C.A. (2006) ***Overcoming Insomnia and Sleep Problems: A Self-Help Guide Using Cognitive Behavioral Techniques*****. Constable & Robinson Ltd., London [ISBN13: 978-184529-070-2/ ISBN10: 1-84529-070-4]**



Chapter 142
Insomnia

Colin A. Espie[1] *and Delwyn J. Bartlett*[2,3]
1University of Glasgow Sleep Centre, Scotland, UK
[2]Woolcock Institute of Medical Research, University of Sydney, Sydney, Australia
[3]Royal Prince Alfred Hospital, Sydney, Australia

Introduction

Insomnia is frequently observed in a number of medical, neurological, and psychiatric disorders, representing a considerable public health concern. Insomnia is the repeated difficulty in initiating sleep (greater than 30 minutes), maintaining sleep (greater than 30 minutes), or waking early, which is chronically non-restorative despite adequate sleep opportunity. Within the neurological field, insomnia may present as a hypersomnia such as narcolepsy and/or as a sleep-related movement disorder including restless legs syndrome (RLS) and period limb movement (PLM) (Table 142.1).

Epidemiology

Insomnia affects one-third of adults occasionally, and 9–12% on a chronic basis. It is more commonly reported in women, shift workers, and patients with medical and psychiatric disorders. Among older adults, prevalence has been estimated at 25%, although co-morbid conditions and hypnotic drugs are factors in this increased prevalence.

Pathophysiology

Sleep disruption is often unreported until insomnia is well established. It is unclear whether the physiological changes associated with insomnia precede onset or are a consequence. High-frequency electroencephalogram (EEG) activity is exaggerated in individuals with insomnia. These findings suggest a central nervous system arousal, supporting previous research that found increased cortisol and adrenocorticotrophic hormones. This could also reflect an adaptation to poor quality sleep, as objective performance is not necessarily impaired.

Clinical features

Subjectively, sleep is non-restorative and daytime functioning is impaired. Individuals are overwhelmingly concerned about sleep onset, returning to sleep, and the unpredictability of sleep. Severity is judged by frequency (three or more times per week), with a minimum duration of 1 month. The clinical presentation is commonly one of a frustrated patient trapped in a vicious circle of anxiety and poor sleep, reporting having "tried everything," and generally unable to "down-regulate" arousal levels at bedtime.

Insomnia also causes daytime impairments, including fatigue, inattention, and mood changes, with anxiety and irritability. Less frequently, cognitive and performance abilities may be affected. The presence of excessive daytime sleepiness (EDS) is unusual in insomnia. When EDS is a prominent complaint, investigations for other sleep disorders should be considered, including obstructive sleep apnea syndrome (OSA), narcolepsy, periodic limb movement disorder (PLM), and restless legs syndrome (RLS). Additionally, head injury or depression may be causes of EDS.

Insomnias due to a drug or substance can include hypnotic dependent sleep disorder – commonly associated with benzodiazepine (BZ) drugs, where withdrawal exacerbates the primary problem, reinforcing hypnotic dependency. Psychiatric conditions, particularly affective disorders, have associated sleep symptomatology. When the diagnostic criteria for DSM-IV Axis I or Axis II disorders are fulfilled, a primary diagnosis of psychophysiological insomnia cannot be made. Sleep disturbances often precede depression, being an independent risk factor for a first episode or recurrence of depression. Insomnia due to medical conditions arises from an identified medical cause (orthopedic, neurologic, pulmonary, cardiac, etc.) and may vary with the condition. The natural history of insomnia is not clear. It is known that sleep quality is reduced with increasing age. Circadian rhythm

International Neurology: A Clinical Approach. Edited by Robert P. Lisak, Daniel D. Truong, William Carroll, and Roongroj Bhidayasiri. © 2009 Blackwell Publishing, ISBN: 978-1-4051-5738-4.

Table 142.1 Diagnosis and differentiation of the insomnias – International Classification of Sleep Disorders (ICSD-2).

Classification	Sleep disorder	Essential features Complaint of insomnia plus…
Insomnias	Psychophysiological insomnia	Learned sleep-preventing associations, conditioned arousal, "racing mind" phenomenon
	Paradoxical insomnia	Complaint of poor sleep disproportionate to sleep pattern and sleep duration
	Idiopathic insomnia	Insomnia typically begins in childhood or from birth
	Insomnia due to a mental disorder	Course of sleep disturbance concurrent with mental disorder
	Inadequate sleep hygiene	Daily living activities inconsistent with maintaining good-quality sleep
	Insomnia due to a medical disorder	Course of sleep disturbance concurrent with mental disorder
	Insomnia due to drug or substance	Sleep disruption caused by prescription medication, recreational drug, caffeine, alcohol or foodstuff
	Adjustment insomnia	Presence of identifiable stressor; insomnia resolves or is expected to resolve when stressor is removed

disorders, shift work, parasomnias, and inadequate sleep hygiene can all be triggers for insomnia.

Investigations

A thorough history incorporating questions regarding mood, lifestyle, restlessness, limb movements, and breathing is important. Sleep diary monitoring is a useful form of assessment in addition to questionnaires on beliefs and moods. Wrist actigraphy estimates sleep-wakefulness based upon body movement for up to 10 consecutive 24-hour periods and can identify paradoxical insomnia, along with circadian anomalies. Polysomnography (PSG) is undertaken only when another sleep disorder is suspected.

Treatment and management

Drug therapy

BZ compounds superseded barbiturates and, although effective short term, were found to cause potential problems with administration tolerance and withdrawal. Contemporary hypnotic therapy includes BzRAs ("z" drugs) and, more recently, melatonin receptor agonists (MeRAs), which have yet to become established. BzRAs offer fewer adverse effects; however, long-term effectiveness is less clear. Increasingly (off-label) sedative antidepressants are being used.

Melatonin, the pineal hormone, triggers sleep onset by lowering core body temperature and is a useful chronobiotic for reducing sleep latency in delayed sleep phase syndrome (DSPS).

Psychological and behavioral therapy

Psychological treatment with cognitive behavioral therapy (CBT) has demonstrated large-effect size changes in primary outcomes and is maintained at long-term follow-up. CBT is also effective in general practice and can be adapted for other settings.

Management strategies

Educating the patient about sleep is an important aspect of treating insomnia. Understanding what sleep is, how sleep changes with age, good sleep hygiene practices (reducing caffeine and alcohol, etc.), and some facts about sleep loss are starting points for self-management.

Bright light is a potent marker for human circadian rhythm, resetting sleep-times in advanced sleep phase syndrome (ASPS) and DSPS. Sleep initiation insomnia is improved with morning light and avoidance of evening light.

Exercise can positively influence sleep quality, particularly in the late afternoon or early evening. Morning exercise with light exposure suppresses melatonin, enhancing circadian rhythm and setting a constant waking time. Sleeping in a safe environment includes minimizing disruption from external factors (heating, noise, violence, others) and internal factors relating to previous experiences.

Stimulus control

Stimulus control is a reconditioning treatment forcing discrimination between daytime and sleeping environments. For the poor sleeper, the bedroom triggers associations with being awake and aroused. Treatment involves removing all stimuli that are potentially sleep-incompatible (reading and watching television) and excluding sleep from living areas. The individual is instructed to get up if not asleep within 15–20 minutes or when wakeful during the night.

Sleep restriction therapy

Sleep restriction relates to the ratio of time asleep with time in bed, and involves recording average nightly sleep duration. The aim is to slowly reduce time in bed to match recorded sleep duration, increasing sleep efficiency and confidence.

Cognitive control

Intrusive thoughts need to be addressed before bedtime. Setting aside 15–20 minutes before bedtime to rehearse the day and to plan for tomorrow allows the day to be put to rest. Thought-stopping attempts to interrupt the flow of thoughts via "blocking" techniques, such as repeating the word "the" every 3 seconds, occupy the short-term memory store (used in processing information), potentially allowing sleep to happen. Cognitive restructuring challenges faulty beliefs that help maintain both wakefulness and helplessness.

Relaxation methods include progressive relaxation, imagery training, biofeedback, meditation, hypnosis, and autogenic training, with little evidence to indicate superiority of any one approach. At the cognitive level, these techniques may act by distraction.

Paradoxical intention

Attempting to remain wakeful rather than "trying" to fall asleep (decatastrophizing technique) strengthens the sleep drive and reduces performance effort.

Treatment of insomnia should include assessment for known extrinsic causes of certain sleep disorders including alcohol, stimulants, and proprietary drugs, which interfere with sleep. Individuals need to be encouraged to seek advice early rather to than self-administer treatment. Avoiding the use of hypnotic agents would substantially reduce the number of iatrogenic cases of chronic insomnia.

Further reading

ICSD. *The International Classification of Sleep Disorders Revised: Diagnostic and Coding Manual*. American Sleep Disorders Association; 2005.

Morin C, Espie C. *Insomnia: A Clinical Guide to Assessment and Treatment*. New York: Kluwer Academic/Plenum; 2003.

NIH. State-of-the-Science Conference Statement on Manifestations and Management of Chronic Insomnia in Adults. State-of-the-Science Conference; 2005.

Perlis M, Lichstein K. *Treating Sleep Disorders: Principles and Practice of Behavioral Sleep Medicine*. Chichester: Wiley; 2003.

Smith M, Perlis M, Park A, *et al*. Comparative meta-analysis of pharmacotherapy and behavior therapy for persistent insomnia. *Am J Psychiatr* 2002; 159: 5–11.

4.14.2

Insomnias

Colin A. Espie and Delwyn J. Bartlett

Introduction

Most people's experiences of poor sleep are memorable, because sleeplessness and its daytime consequences are unpleasant. There are those, however, for whom insomnia is the norm. Persistent and severe sleep disturbance affects at least one in 10 adults and one in five older adults, thus representing a considerable public health concern. Sleep disruption is central to a number of medical and psychiatric disorders, and insomnia is usually treated by general practitioners. Therefore differential diagnosis is important, and respiratory physicians, neurologists, psychiatrists, and clinical psychologists need to be involved. The purpose of this chapter is to summarize current understanding of the insomnias, their appraisal, and treatment. Particular emphasis will be placed upon evidence-based practical management.

Clinical features

Insomnia often remains unreported, and finally presents when a poor sleep pattern is well established. Alcohol has long been a first-line self-administered sleep aid, and recent years have seen an increasing use of 'over-the-counter' preparations and 'self-help' strategies. The clinical presentation is commonly of a frustrated patient, trapped in a vicious circle of anxiety and poor sleep, who reports having 'tried everything'.[1,2]

There may be concern about the pattern of sleep. This is the most quantifiable aspect of self-report relating to, for example, length of time taken to fall asleep, frequency and duration of wakenings, or total amount of sleep. A poorly established sleep pattern can lead to unpredictability of what sleep will be like on any given night. Patients often report poor quality of sleep, and subjective perceived quality can be a critically important outcome variable. Typical reports relate to light sleep and sleep felt to be unrestorative. Although it may be unclear how such complaints relate to EEG sleep architecture, the clinician should not overlook qualitative report as it may reflect underlying pathophysiology. Concerns are normally expressed also about the daytime effects of poor sleep. These can be cognitive effects, such as fatigue, sleepiness, inattention, and some impairments in performance, or emotional effects, such as irritability and anxiety.[2]

Classification

The *International Classification of Sleep Disorders* (second edition: **ICSD-2**)[3] was published in 2005 and provides the most comprehensive account of sleep disorders, both for descriptive purposes and for differential diagnosis (see Chapter 4.14.1). ICSD-2 describes insomnias as disorders of initiating and maintaining sleep. Patients may have either sleep-onset problems or wakenings from sleep, or both of these. Table 4.14.2.1 summarizes the principal classifications that relate to the insomnias, along with some other sleep disorders where patients commonly present with insomnia symptoms. As can be seen, concomitant symptomatology, potential aetiological factors, and sleeplessness require careful assessment in order to reach a valid diagnosis.

Diagnosis and differential diagnosis

Severity of insomnia is judged along dimensions of frequency, intensity, and duration, as well as impact on daytime functioning and quality of life. Generally, the criteria for severe and chronic insomnia are a minimum duration of 6 months with problems presenting three or more nights per week. Restlessness, irritability, anxiety, daytime fatigue, and tiredness commonly accompany such presentations.[2] Mild and moderate insomnia may be diagnosed where problems are less intrusive.

Most patients presenting with insomnia have psychophysiological difficulty initiating and/or maintaining sleep. Usually marked functional effects and somatized tension associated with sleep are evident. The patient reports extreme tiredness while being unable to sleep satisfactorily and appears preoccupied with sleep and its consequences. This contrasts, for example, with the circadian disorders where, in delayed sleep-phase type, the patient may not feel sleepy until late in the normal sleep period, and in advanced sleep-phase type, may waken early and be unable to return to sleep. Taking a history, incorporating screening questions on restlessness, limb movements, and breathing can help to diagnose obstructive sleep apnoea syndrome, periodic limb movement disorder, and restless legs syndrome, although full polysomnographic evaluation may also be required.[4] However, polysomnography is not essential for the diagnosis of insomnia, for which sleep diary monitoring (see Chapter 4.14.1) is usually the most useful form of assessment.[2] Wrist actigraphy is an inexpensive objective evaluation, which

Table 4.14.2.1 The classification and differential diagnosis of the insomnias within ICSD-2

Classification	Sleep disorder	Essential features, complaint of insomnia plus
Insomnias	Psychophysiological insomnia	Learned sleep preventing associations, conditioned arousal, 'racing mind' phenomenon
	Paradoxical insomnia	Complaint of poor sleep disproportionate to sleep pattern and sleep duration
	Idiopathic insomnia	Insomnia typically begins in childhood or from birth
	Insomnia due to a mental disorder	Course of sleep disturbance concurrent with mental disorder
	Inadequate sleep hygiene	Daily living activities inconsistent with maintaining good-quality sleep
	Insomnia due to a medical disorder	Course of sleep disturbance concurrent with mental disorder
	Insomnia due to drug or substance	Sleep disruption caused by prescription medication, recreational drug, caffeine, alcohol or foodstuff
	Adjustment insomnia	Presence of identifiable stressor, insomnia resolves or is expected to resolve when stressor is removed
Sleep-related breathing disorders	Obstructive sleep apnoea syndrome	Excessive sleepiness, obstructed breathing in sleep, associated symptoms include snoring and a dry mouth
	Periodic limb movement disorder	Episodes of repetitive, highly stereotyped limb movements occurring in sleep
	Restless legs syndrome	Strong, nearly irresistible urge to move legs relieved by walking
Circadian rhythm sleep disorders	Delayed sleep phase type	Phase delay of major sleep episode, initial insomnia, excessive sleepiness in morning
	Advanced sleep-phase type	Phase advance of major sleep episode, inability to stay awake in evening, early wakening

estimates sleep/wakefulness based upon body movement.[5] Continuous recordings can be made over 5 to 10 consecutive 24-h periods. It is useful in identifying paradoxical insomnia, and charted data can be inspected for circadian anomalies.

Other causes of insomnia are reported in Table 4.14.2.1 and should not be overlooked. In particular, insomnias due to a drug or substance can include hypnotic-dependent sleep disorder, associated most commonly with benzodiazepine (BZ) drugs where withdrawal leads to exacerbation of the primary problem.[6] This can be mistaken for a severe underlying insomnia and hence reinforce hypnotic dependency. Likewise, a wide range of psychiatric conditions, particularly affective disorders, has associated sleep symptomatology (see Chapter 4.14.1). A primary diagnosis of psychophysiological insomnia cannot be made where diagnostic criteria for DSM-IV Axis I or Axis II disorders are fulfilled. However, it is very important to note that sleep disturbance often precedes depression. The bulk of the psychiatric epidemiological data indicate that insomnia is an independent risk factor for first episode depressive illness, and for recurrence of depression, in adults of all ages.[7,8] Insomnia should not be assumed to be simply a symptom of underlying depression, even when depression is present. Unless the illness courses clearly co-vary it is best to make a diagnosis of co-morbid insomnia. Similar caveats apply to insomnia associated with medical disorders, both in terms of identifying a primary illness, and concluding that insomnia has *the* status of an associated/ co-morbid disorder (see Chapter 4.14.1).

Epidemiology

Insomnia affects one-third of adults occasionally, and 9 to 12 per cent on a chronic basis. It is more common in women, in shift workers, and in patients with medical and psychiatric disorders. Prevalence amongst older adults has been estimated at up to 25 per cent and sleepiness and hypnotic drugs are risk factors for injury and fracture.[9] The decline in prescription of anxiolytics has been greater than the rate of decline for hypnotics [taking BZ and benzodiazepine receptor agonists (BzRAs) together]. Furthermore, there is increasing use of (off-label) sedative antidepressants primarily to treat insomnia.

Aetiology

Many patients report having been marginal light sleepers before developing insomnia.[4] Sleep disturbance often arises during life change or stress, and such adjustment sleep disorder may represent a normal transient disruption of sleep. However, secondary factors, such as anxiety over sleep and faulty sleep–wake conditioning, may exacerbate and maintain the insomnia as a chronic problem when sleep itself becomes a focus for concern. People with insomnia may be hyperaroused relative to normal sleepers, for example having higher levels of cortisol and ACTH, and also find it difficult to 'down-regulate' their arousal at bedtime.[2,10,11]

Course and prognosis

There has been little research on the natural course of insomnia. However, untreated psychophysiological insomnia can last for decades, and may gradually worsen over time. Indeed, there is a developmental trend for sleep pattern to deteriorate, with increasing age. On the other hand, delayed sleep-phase syndrome and insufficient sleep hygiene can be associated with lifestyle problems and may ameliorate as these are resolved. Although certain insomnias *tend* to persist if untreated, prognosis with effective treatment can be very good.

Treatment

A review of the evidence

(a) Drug therapy

Traditionally, insomnia has been treated pharmacologically. Barbiturates were superseded by BZ compounds during the 1960s and 1970s. These drugs were safer in overdose, were thought to have fewer side effects, and to be less *addictive*. Controlled studies have demonstrated that a considerable number of BZ, of short to

intermediate half-life, are effective hypnotic agents. However, from the mid-1970s potential problems became apparent, both during administration and withdrawal. Longer-acting hypnotics were prone to carry-over effects of morning lethargy, and shorter-acting drugs to 'rebound insomnia'.[6] Furthermore, tolerance develops, leading either to increased dosing or switching to alternative medication. Although BZs used for short periods/intermittently can maintain effectiveness, these are not the treatment of choice in chronic insomnia,[12] and are contraindicated in older adults and where insomnia may involve sleep-related breathing disorder because of their potentially depressant effects on respiration. A number of BZ compounds have been removed from the market in the United Kingdom, United States, and elsewhere.

Contemporary hypnotic therapy has extended to include BzRAs (often referred to as the 'z' drugs), and more recently melatonin receptor agonists (MeRAs) have been introduced. Whereas the place in therapeutics of MeRAs has yet to become established, the BzRAs are often thought to offer more sustained benefit for insomnia, and to have fewer adverse effects. Nevertheless, there remains uncertainty about the effectiveness of BzRAs in chronic insomnia.[13]

(b) Psychological therapy

Psychological treatment for chronic insomnia, primarily in the form of cognitive–behavioural therapy (CBT), has been extensively investigated in over 100 controlled studies during the past 20 years. Five meta-analyses and numerous systematic reviews have demonstrated that CBT is associated with large effect size changes (measured in standardized *z* scores) in the primary symptom measures of sleep latency (difficulty getting to sleep) and wake time after sleep-onset (difficulty remaining asleep).[14,15] Around 70 per cent of patients with persistent sleep problems appear to benefit from CBT and effects are maintained to long-term follow-up. It is thought that CBT achieves these outcomes because it tackles directly the dysfunctional thoughts and maladaptive behaviours that otherwise maintain insomnia. Recent controlled studies have shown that CBT may be effective in general practice settings with nurses delivering the intervention according to a standard protocol.[16,17] Despite the superior efficacy of CBT relative to medication for insomnia, and these recent demonstrations of CBT working in real-world settings, practical problems remain in making CBT widely available.

Within the CBT model, a number of strategies have strong empirical support. Behavioural procedures such as stimulus control and sleep restriction, and cognitive strategies such as paradoxical intention and thought restructuring have been extensively investigated[2,14,15] and are outlined briefly below.

(c) Melatonin, light therapy, and exercise

The pineal hormone melatonin has been the subject of highly publicized claims. However, scientific research has been limited. Several controlled studies support its sleep-promoting effects, but the use of melatonin continues to be controversial. At best it may be useful as a chronobiotic for reducing sleep latency.[18] Several MeRA products are currently under formal evaluation, so more data may be available soon.

Bright light is a potent marker for human circadian rhythms, and has been known for some time to enable the resetting of such rhythms in advanced sleep-phase syndrome and delayed sleep-phase syndrome.[19] The results of studies investigating the efficacy of bright light against psychological treatments for psychophysiological insomnia are awaited. A limiting factor to the value of light therapy is that continued treatment may be required to maintain therapeutic effects.

Athletic people sleep well, although this may be more to do with behavioural patterning than aerobic fitness. Nevertheless, there is evidence that exercise can have positive effects upon sleep quality, particularly if taken late afternoon or early evening, and in otherwise relatively fit individuals.[20] Morning exercise can also be an effective modality to encourage the same waking time and early morning light exposure; which help to reset sleep patterns on a daily basis

Advice about management

(a) General perspective

Non-pharmacological treatment using CBT procedures should be preferred over pharmacological treatment, in cases of severe persistent insomnia. Hypnotic agents should be recommended mainly for short term or occasional use, although longer-term trial data are now becoming available. The practitioner should be aware of morning-after effects, and potential problems of withdrawal and dependency, not only with BZs but also possibly with BzRAs. Psychological intervention may also facilitate reduction or discontinuation of medication in hypnotic-dependent person with insomnias.[21] There is limited support for the use of melatonin or exercise as treatments of choice, although light therapy seems effective for circadian disorders.

(b) Using cognitive–behavioural therapies

Brief descriptions of effective management strategies are presented in Tables 4.14.2.2 and 4.14.2.3. The following text provides explanation of underlying psychological models and further information on implementation.

(i) Sleep education and sleep hygiene

The simple provision of information ameliorates the sense of being out of control. Inaccurate attributions are challenged and misunderstandings corrected by understanding what sleep is, how

Table 4.14.2.2 Summary description of sleep hygiene and education components for the treatme nt of chronic insomnia

Components of sleep education
The need for sleep and its functions
Sleep patterns across the lifespan
Sleep as a process with stages/phases
Factors adversely affecting steep
The effects of sleep loss
The concept of insomnia
Measuring sleep and sleep problems
Components of sleep hygiene treatment
Bedroom comfortable for sleep
Regular exercise, timing, and fitness
Stable and appropriate diet
Undesirable effects of caffeine and other stimulants
Moderation of alcohol consumption
Other common 'self-help' strategies

Table 4.14.2.3 Summary description of cognitive–behavioural components for the treatment of chronic insomnia

Components of stimulus control and sleep restriction treatment
Define individual sleep requirements
Establish parameters for bedtime period (threshold time and rising time)
Eliminate daytime napping
Differentiate rest from sleep
Schedule sleep periods with respect to needs
Establish 7 day per week compliance
Remove incompatible activity from bedroom environment
Rise from bed if wakeful (>20 min)
Avoid recovery sleep as 'compensation'
Establish stability from night to night
Adjust the sleep period as sleep efficiency improves
Components of cognitive intervention
Identify thought patterns and content that intrude
Address (mis)attributions connecting sleep and waking life
Establish rehearsal/planning time in early evening
Relaxation and imagery training
Distraction and thought blocking
Develop accurate beliefs/attributions about sleep and sleep loss
Challenge negative and invalid thoughts
Eliminate 'effort' to control sleep
Motivate to maintain behaviour and cognitive change
Utilize relapse-prevention techniques

common insomnia can be, how sleep changes with age, good sleep hygiene practices, and some facts about sleep loss. Similarly, sleep hygiene provides patients with a starting point for self-management. These techniques are best construed as introductory but they will not of themselves treat insomnia effectively.

(ii) Stimulus control treatment

Stimulus control increases the bedroom's cueing potential for sleep. For good sleepers, the pre-bedtime period and the stimulus environment trigger positive associations of sleepiness and sleep. For the poor sleeper, however, the bedroom triggers associations with restlessness and lengthy night-time wakening via a stimulus-response relationship, thereby continuing to promote wakefulness and arousal. The model is similar to phobic conditions where a conditioned stimulus precipitates an anxiety response.

Treatment involves removing from the bedroom all stimuli which are potentially sleep-incompatible. Reading and watching television, for example, are confined to living rooms. Sleeping is excluded from living areas and from daytime, and wakefulness is excluded from the bedroom. The individual is instructed to get up if not asleep within 15–20 min or if wakeful during the night. Conceptually, stimulus control is a reconditioning treatment which forces discrimination between daytime and sleeping environments.

(iii) Sleep restriction therapy

Sleep restriction restricts sleep to the length of time which the person is likely to sleep. This may be equivalent to promoting 'core sleep' at the expense of 'optional sleep'. Sleep restriction primarily aims to improve sleep efficiency. Since sleep efficiency is the ratio of time asleep to time in bed, it can be improved either by increasing the numerator (time spent asleep) or by reducing the denominator (time spent in bed). People with insomnia generally seek the former, but this may not be necessary, either biologically or psychologically. Sleep restriction first involves recording in a sleep diary and calculating average nightly sleep duration. The aim, then, is to obtain this average each night. This is achieved by setting rising time as an 'anchor' each day and delaying going to bed until a 'threshold time' which permits this designated amount of sleep. Thus, the sleep period is compressed and sleep efficiency is likely to increase. The permitted 'sleep window' can then be titrated week-by-week in 15 increments in response to sleep efficiency improvements.

(iv) Cognitive control

This technique aims to deal with thought material in advance of bedtime and to reduce intrusive bedtime thinking. The person with insomnia is asked to set aside 15 to 20 min in the early evening to rehearse the day and to plan ahead for tomorrow; thus putting the day to rest. It is a technique for dealing with unfinished business and may be most effective for rehearsal, planning, and self-evaluative thoughts which are important to the individual and which, if not dealt with, may intrude during the sleep-onset period.

(v) Thought suppression

Thought-stopping and articulatory suppression attempt to interrupt the flow of thoughts. No attempt is made to deal with thought material per se but rather to attenuate thinking. With articulatory suppression the patient is instructed to repeat, subvocally, the word 'the' every 3 s. This procedure is derived from the experimental psychology literature. Articulatory suppression is thought to occupy the short-term memory store used in the processing of information. The type of material most likely to respond is repetitive but non-affect-laden thoughts, not powerful enough to demand attention. Additionally, this technique may be useful during the night to enable rapid return to sleep.

(vi) Imagery and relaxation

There is a wide range of relaxation methods including progressive relaxation, imagery training, biofeedback, meditation, hypnosis, and autogenic training, but little evidence to indicate superiority of any one approach. Furthermore, there is little evidence to support either the presumption that people with insomnia are hyper-aroused in physiological terms, or that relaxation has its effect through autonomic change. At the cognitive level, these techniques may act through distraction and the promotion of mastery. During relaxation, the mind focuses upon alternative themes such as visualized images or physiological responses. In meditation the focus is upon a 'mantra' and in self-hypnosis upon positive self-statements. Relaxation may be effective for thought processes that are anxiety-based, confused, and which flit from topic-to-topic.

(vii) Cognitive restructuring

Cognitive restructuring challenges faulty beliefs which maintain wakefulness and the helplessness which many people with insomnia report. It appears to work through appraisal by testing the validity of assumptions against evidence and real-life experience. As an evaluative technique, it may be effective with beliefs that are irrational but compelling. If such thoughts, for example 'I am going to be incapable at work tomorrow', are not challenged, they will create high levels of preoccupation and anxiety and sleep is

unlikely to occur. With cognitive restructuring, the person with insomnia learns alternative responses to replace inaccurate thinking.

(viii) Paradoxical intention

Finally, the technique of paradoxical intention is useful in situations where performance anxiety has developed, that is, where the effort to produce a response inhibits that response itself. The paradoxical instruction is to allow sleep to occur naturally through passively attempting to remain quietly wakeful rather than attempting to fall asleep. Paradox may be regarded as a decatastrophizing technique since it appears to act upon the ultimate anxious thought (of remaining awake indefinitely) initially by focusing on and enhancing this thought (a habituation model) and then subjecting it to appraisal through rationalization and experience. By intending to remain awake, and failing to do so, the strength of the sleep drive is re-established, and performance effort is reduced.

Possibilities for prevention

There is insufficient knowledge of the natural course of transient sleep disorders. Mention has been made of adjustment sleep disorder and of the association of life events and stressors with the onset of insomnia. Systematic research is required to establish the 'setting conditions' for the secondary maintenance of insomnia beyond an initial normative reaction to events. Perhaps there is an interaction with a predisposing tendency to light sleep, or with introspection and worry. The instinct to increase opportunity to sleep (spend longer in bed to catch up) when insomnia symptoms develop should probably be resisted. If anything it may be better to advise patients to limit sleep opportunity so that their pattern knits together again more quickly.

The establishment and maintenance of a regular 'tight' routine, both pre-bedtime and in terms of sleep schedule, seem to be important preventive factors. Such chronobehavioural functioning can be at risk of disruption by, for example, jet lag, shift work, weekend patterns differing from weekday, adolescent lifestyle, and retirement. Adherence to, and/or reinstatement of, an adaptive pattern seems crucial.

It is important not to underestimate the importance of attitudes and beliefs in the presentation of insomnia. Exaggerated or emotionally and mentally arousing thoughts should be dealt with promptly. Sleep loss can be distressing, but patients should be reminded that nature seeks to restore equilibrium. What they need to do is to provide the conditions under which sleep can occur rather than attempt directly to control the sleep process. Expectations are important also, since it is the breach of these which generally give rise to anxiety and dysfunctional beliefs about sleep requirements. More often than not sleep-related expectations are unrealistic and require reappraisal, even more so in older adults.

Finally, prevention should be extended to the known extrinsic causes of certain sleep disorders. Where alcohol, stimulants, or proprietary drugs interfere with sleep and the recovery of the normal sleep process, attention should be paid to these factors. Better still, patients should be encouraged to seek advice early rather than go down the path of self-administered treatment. Avoiding the use of hypnotic agents, both in general practice and during acute admissions to hospital, would substantially reduce the number of iatrogenic cases of chronic insomnia.

Further information

Espie, C.A. (2006). *Overcoming insomnia and sleep problems: a self-help guide using cognitive behavioral techniques*. Constable & Robinson Ltd, London [ISBN13: 978-184529-070-2/ ISBN10: 1-84529-070-4].

Perlis, M.L. and Lichstein, K.L. (2003). *Treating sleep disorders: principles and practice of behavioral sleep medicine*. John Wiley & Sons, Inc. Hoboken, NJ [ISBN0-47-44343-3].

Morin, C.M., Bootzin, R.R., Buysse, D.J., Edinger, J.D., Espie, C.A., and Lichstein, K.L. (2006). Psychological and behavioural treatment of insomnia. Update of the recent evidence (1998–2004) prepared by a Task Force of the American Academy of Sleep Medicine. *Sleep*, **29**, 1398–414.

Morin, C.M. and Espie, C.A. (2003). *Insomnia: a clinical guide to assessment and treatment*. Kluwer Academic/ Plenum Publishers, New York [ISBN 0-306-47750-5].

AQ: This ref is similar to ref no: 15.

AQ: This ref is similar to ref no: 2

References

1. Espie, C.A. (1991). *The psychological treatment of insomnia*. Wiley, Chichester.
2. Morin, C.M. and Espie, C.A. (2003). *Insomnia: a clinical guide to assessment and treatment*. Kluwer Academic/Plenum Publishers, New York.
3. American Academy of Sleep Medicine. (2005). *International classification of sleep disorders: diagnostic and coding manual* (2nd edn). AASM, Westchester, IL.
4. Reite, M., Buysse, D., Reynolds, C., and Mendelson, W. (1995). The use of polysomnography in the evaluation of insomnia. *Sleep*, **18**, 58–70.
5. Ancoli-Israel, S., Cole, R., Alessi, C., Chambers, M., Moorcroft, W., and Pollak, C.P. (2003). The role of actigraphy in the study of sleep and circadian rhythms. *Sleep*, **26**, 342–92.
6. Kripke, D. (1989). Hypnotic drugs: deadly risks, doubtful benefits. *Sleep Medicine Reviews*, 2000, **4**, 5–20.
7. Cole, M.G. and Dendukuri, N. (2003). Risk factors for depression among elderly community subjects: a systematic review and meta-analysis. *The American Journal of Psychiatry*, **160**, 1147–56.
8. Riemann, D. and Voderholzer, U. (2003). Primary insomnia: a risk factor to develop depression? *Journal of Affective Disorders*, **76**, 255–9.
9. Lichstein, K.L., Durrence, H.H., Reidel, B.W., Taylor, D.J., and Bush, A.J. (2004). *The epidemiology of sleep: age, gender and ethnicity*. Lawrence Erlbaum Associates, Mahwah, NJ.
10. Espie, C.A. (2002). Insomnia: conceptual issues in the development, persistence and treatment of sleep disorder in adults. *Annual Review of Psychology*, **53**, 215–43.
11. Perlis, M.L., Pigeon, W., and Smith, M.T. (2005). Etiology and pathophysiology of insomnia. In *The principles and practice of sleep medicine* (4th edn) (eds. M.H. Kryger, T. Roth, and W.C. Dement), pp. 714–25. W.B. Saunders, Philadelphia.
12. NIH. (2005). *State-of-the-science conference statement on manifestations and management of chronic insomnia in adults*. National Institutes of Health, Vol. 22, Number 2.
13. NICE. (2004). *Guidance on the use of zaleplon, zolpidem and zopiclone for the short-term management of insomnia*. Technology Appraisal Guidance No.77. National Institute for Clinical Excellence, London.
14. Smith, M.T., Perlis, M.L., Park, A., Giles, D.E., Pennington, J.A., and Buysse, D. (2002). Behavioral treatment vs pharmacotherapy for insomnia—a comparative meta-analysis. *The American Journal of Psychiatry*, **159**, 5–11.
15. Morin, C.M., Bootzin, R.R., Buysse, D.J., Edinger, J.D., Espie, C.A., and Lichstein, K.L. (2006). Psychological and behavioural treatment of insomnia. Update of the recent evidence (1998–2004) prepared by a

AQ: Please check whether the year of publication should be 1989 or 2000?

AQ: Please provide place of publication.

Task Force of the American Academy of Sleep Medicine. *Sleep*, **29**, 1398–414.
16. Espie, C.A., Inglis, S.J., Tessier, S., and Harvey, L. (2001). The clinical effectiveness of cognitive behaviour therapy for chronic insomnia: implementation and evaluation of a Sleep Clinic in general medical practice. *Behaviour Research and Therapy*, **39**, 45–60.
17. Espie, C.A., MacMahon, K.M.A., and Kelly, H.L. (2007). Randomised clinical effectiveness trial of nurse-administered small group CBT for persistent insomnia in general practice. *Sleep*, **30**, 574–84.
18. Mendelson, W.B. (1997). A critical evaluation of the hypnotic efficacy of melatonin. *Sleep*, **20**, 916–19.
19. Czeisler, C.A., Johnson, M.P., Duffy, J.F., Brown, E.N., Ronda, J.M., and Kronauer, R.E. (1990). Exposure to bright light and darkness to treat physiologic maladaptation to night work. *The New England Journal of Medicine*, **322**, 1253–8.
20. Singh, N.A., Clements, K.M., and Fiatarone, M.A. (1997). A randomized controlled trial of the effect of exercise on sleep. *Sleep*, **20**, 95–101.
21. Morgan, K., Dixon, S., Mathers, M., Thompson, J., and Tomeny, M. (2003). Psychological treatment for insomnia in the management of long-term hypnotic drug use: a pragmatic randomised controlled trial. *The British Journal of General Practice*, **53**, 923–8.

Models of Insomnia

Michael Perlis, Paul Shaw, Georgina Cano, and Colin Espie

Chapter 78

Up until the late 1990s there were only two models regarding the etiology and pathophysiology of insomnia. The relative lack of theoretical perspectives was due to at least three factors. First, the widespread conceptualization of insomnia as owing directly to hyperarousal may have made it appear that further explanation was not necessary. Second, the long-time characterization of insomnia as a symptom carried with it the clear implication that insomnia was not itself worth modeling as a disorder or disease state. Third, for those inclined toward theory, the acceptance of the behavioral models (i.e., the 3P behavioral model and the stimulus control model[1,2]), and the treatments that were derived from them, might have had the untoward effect of discouraging the development of alternative or elaborative models.

Since the 1990s there has been a proliferation of theoretical perspectives on the etiology and pathophysiology of insomnia that includes ten human models* and three animal models. In this chapter, six models (Box 78-1) are described and critiqued: the classic 3P behavioral model,[1] the stimulus control model,[2] and four models that are arguably the most influential of the modern perspectives†: the neurocognitive model,[3] the psychobiological inhibition model,[4] the *Drosophila* model,[5,6] and the rodent model.[7]

THE DEFINITION OF INSOMNIA

Currently, insomnia is conceptualized in terms of chronicity, type, and subtype. *Chronicity* refers to whether the insomnia is acute or chronic. *Type* refers to the forms of insomnia that have been identified as distinct nosologic entities including (for adults) idiopathic insomnia, psychophysiologic insomnia, paradoxical insomnia, insomnia due to inadequate sleep hygiene, and insomnia comorbid with medical or psychiatric illness. *Subtype* refers to the insomnia phenotype (initial, middle, late, or mixed insomnia). The formal definition of these entities, and discussion about their orthogonality and clinical utility, may be found elsewhere in this volume. What is relevant for the present chapter is that these diagnostic distinctions exist and thus must be taken into account by the various models; that is, each model must indicate which type of insomnia (and subtype, if pertinent) is being modeled.

THE STIMULUS CONTROL MODEL

Basic Description

Stimulus control, as originally described by Bootzin,[2] is based on the behavioral principle that one stimulus may elicit a variety of responses, depending on the conditioning history. A simple conditioning history, wherein a stimulus is always paired with a single behavior, yields a high probability that the stimulus will yield only one response. A complex conditioning history, wherein a stimulus is paired with a variety of behaviors, yields a low probability that the stimulus will yield only one response. In persons with insomnia, the normal cues associated with sleep (e.g., bed, bedroom, bedtime, etc.) are often paired with activities other than sleep. For instance, in an effort to cope with insomnia, the patient might spend a large amount of time in the bed and bedroom awake and engaging in activities other than sleep. The coping behavior appears to the patient to be both reasonable (e.g., staying in bed at least permits the patients to rest) and reasonably successful (engaging in alternative activities in the bedroom sometimes appears to result in cessation of the insomnia). These practices, however, set the stage for stimulus dyscontrol, the lowered probability that sleep-related stimuli will elicit the desired response of sleepiness and sleep. Figure 78-1 provides as schematic representation of stimulus control and stimulus dyscontrol.

Strengths and Weaknesses

The treatment that is derived from stimulus control theory is one of the most widely used behavioral treatments, and its efficacy has been well established.[8-12] The success of the therapy, however, is not sufficient evidence to say that stimulus dyscontrol is the factor, or one of the factors, responsible for predisposition to, the precipitation of, or the perpetuation of insomnia.* This is the case because the therapy includes active components that are not based solely on learning or behavioral theory. For instance, the treatment specifies that the patient should spend awake time somewhere other than the bed and that the sleep schedule should be fixed. These two interventions also influence the homeostatic and circadian regulation of sleep. Thus, the efficacy of stimulus control therapy does not necessarily provide evidence for the stimulus control model. In fact, one investigation found that the reverse of stimulus control instructions also improved sleep continuity.[13]

Another limitation of the stimulus control perspective is that it focuses solely on instrumental conditioning. That is, there are activities that can be engaged in that reduce or enhance the probability of the occurrence of sleep. The original model does not explicitly delineate how classical conditioning might also be an operational factor. That is, the regular pairing of the physiology of wake with sleep-related stimuli might lead to a scenario where sleep-related stimuli become conditioned stimuli for wakefulness. This latter possibility, although not part of the classical stimulus control perspective, is clearly consistent with it.

*A complete listing of theories and models, along with citations, is contained in Appendix 1 on the website.

†Although it is difficult to assess which models are the most influential, one approach would be based on a citation index metric. Using this index, it does indeed appear that the four contemporary models described in this chapter are the most influential.

*The conceptual time frame for causality in terms of "predisposition, precipitation, and perpetuation" was first articulated as part of the 3P model. It is used in this context to illustrate the complexity of modeling what "cause" insomnia.

Box 78-1 Potential Implications for Treatment of Insomnia

Stimulus Control Model
One unexplored implication for treatment is that physically altering the sleep environment may be helpful (e.g., paint the room a different color)

Spielman Model
The 3P model suggests that insomnia is perpetuated by sleep extension and thus should be managed with treatment protocols that restrict time in bed (i.e., compress the sleep period).

One implication for treatment is that sleep compression need not occur in a radical fashion, but could be accomplished over days or weeks.[19]

Neurocognitive Model
The neurocognitive model suggests that patients with insomnia suffer from an attenuation of the normal mesograde amnesia of sleep.

One unexplored implication for treatment is that potentiation of the normal mesograde amnesia of sleep via the use of more traditional hypnotics (e.g., benzodiazapines with effects on long-term memory) might serve to augment clinical gains, if not in general, then at least in patients with substantial sleep state misperception.

Psychobiological Inhibition Model
According to the psychobiological inhibition model, chronic insomnia is less a hyperarousal disorder and more a disorder characterized by the failure to inhibit wakefulness.

One implication for treatment is that persistent wakefulness may be the result of hypersecretion of orexin, and thus orexin antagonism might have a place in the management of insomnia.

***Drosophila* Model**
The *Drosophila* model suggests that there may be a strong genetic component to insomnia that may be related to reduced sleep ability.

One implication of the model is that it, like the 3P model, suggests that sleep opportunity should be a major focus for treatment.

Cano-Saper Model
The Cano-Saper model suggests that insomnia represents a hybrid state, one that is, from a neurobiological perspective, part wake and part sleep.

One implication for treatment, which has not yet been tested empirically, is that corticotropin releasing hormone antagonist represent an alternative way of alleviating disturbed sleep continuity.

Implications for Current and Future Research and Therapeutics

Given the efficacy of stimulus control therapy, as it is classically rendered, it would be useful to determine how much treatment outcome from cognitive behavior therapy (CBT) owes to the manipulation of this factor. One way to assess the relative importance of stimulus control would be as part of a dismantling study. To date no such study has been conducted as a single, large-scale, randomized trial.* Alternatively, experimental studies could be used to determine which, if any, specific stimuli are most associated with sleep continuity disturbance and whether alteration of these stimuli produces enhanced clinical gains.

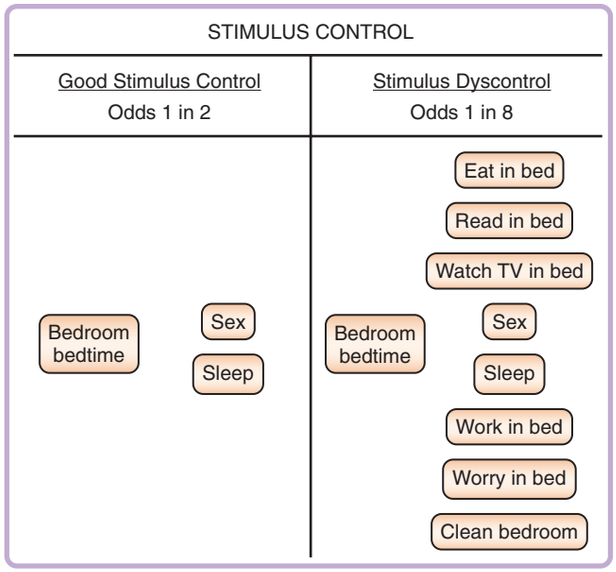


Figure 78-1 The instrumental conditioning perspective on stimulus control. *Left,* Good stimulus control: The bedroom is tightly coupled with sleep and sex where, given the orthogonality and equal probability of events, the probability of association of bedroom to sleep is 1 in 2. *Right,* Stimulus dyscontrol: The bedroom is no longer a strong associate of sleep and sex where, given the orthogonality and equal probability of events, the probability of association of bedroom to sleep is 1 in 8. The treatment implication of stimulus dyscontrol is the voluntary elimination of the nonsleep associations except for sex, which should result in instrumental conditioning.

THE 3P MODEL

The 3P behavioral model,[1] also known as the Spielman model, the three-factor model, or the behavioral model is the first fully articulated model of insomnia to gain widespread acceptance. The model delineates how insomnia occurs acutely and how acute insomnia becomes chronic and self-perpetuating. The model is based on the interaction of three factors. The first two factors (the predisposing and precipitating factors) represent a stress-diathesis conceptualization of how insomnia comes to be expressed. The third factor (the perpetuating factor) represents how behavioral considerations modulate chronicity. A schematic representation of this model is presented in Figure 78-2.

Basic Description

Predisposing factors extend across the entire biopsychosocial spectrum. Biological factors are likely to include increased basal metabolic rate, hyperreactivity, and or fundamental alterations to the neurotransmitter systems associated with sleep and wakefulness.* Psychological factors include

*The possibility of altered neurotransmission in insomnia (e.g., reduced GABAnergic tone) was recently explored by Winkelman and colleagues. See SLEEP 31(11)2008:1499-1506.

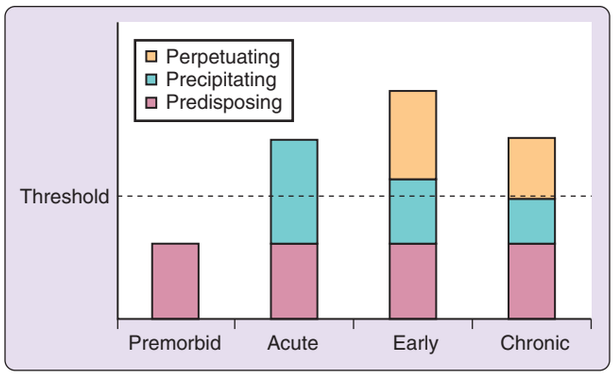


Figure 78-2 The classic 1987 rendition of the 3P model. There is a more recent representation of the model in Chapter 144. The reader is encouraged to compare the two versions of the model. The differences (e.g., allowing the predisposing factors to be represent as variable with time), while subtle, are theoretically important.

worry or the tendency to be excessively ruminative. Social factors, although rarely a focus at the theoretical level, include such things as the bed partner keeping an incompatible sleep schedule or social pressures to sleep according to a nonpreferred sleep schedule (e.g., child rearing).

Precipitating factors, as the name implies, are acute occurrences that trigger disturbance of sleep disturbance. The primary triggers are thought to be related to life stress events (including medical and psychiatric illness).

Perpetuating factors refer to the actions the insomniac person adopts that are intended to compensate for, or cope with, sleeplessness. Research and treatment have focused on three kinds of perpetuating factors: the practice of nonsleep activities in the bedroom, the tendency to stay in bed while awake, and the tendency to spend excessive amounts of time in bed. Stimulus control speaks to the first two of these considerations (as reviewed earlier).

The classic version of the 3P model focuses primarily on the last of these considerations. Excessive time in bed (or sleep extension) refers to the tendency of patients with insomnia to go to bed earlier or to get out of bed later or to engage in napping. The patient enacts such changes (compensatory activities) to increase the opportunity to get more sleep; these changes are likely highly self-reinforcing (in the short term) because they allow lost sleep to be "recovered" and the daytime effects of lost sleep to be ameliorated. The tendency toward sleep extension is, in the long term, problematic. Sleep extension leads to a mismatch between sleep opportunity and sleep ability.[1,14] The greater the mismatch, the more likely the person will spend prolonged periods wake during the given sleep period, and that this will occur regardless of what predisposed the individual to the insomnia and precipitated it.

Strengths and Weaknesses

The greatest strengths of the 3P model is that the therapy based on the theory (sleep restriction) is conceptually appealing to sleep medicine clinicians and scientists, the model is highly face valid for patients (especially when it is delivered as part of therapy), and the therapy itself (which is also compatible with, and a logical clinical application of, the two-process model of normal sleep[15]) *appears to be* very efficacious. The equivocation regarding efficacy represents one of the models weaknesses.

There have been very few studies evaluating sleep restriction therapy as a monotherapy,[8,9] and no studies evaluating the relative efficacy of sleep restriction therapy (using dismantling designs) as component of CBT. It is therefore difficult to assess the extent to which treatment efficacy supports the 3P model itself. Further, even if there were large-scale studies showing that sleep-restriction therapy produces large effects, the validation of the model would still require empirical studies (see later).

The model (while compatible with the two-process model of sleep–wake regulation) does not explicitly take into account the influences of the circadian system and sleep–wake homeostasis. Further, the model does not provide a detailed account of how one transitions from good sleep to acute insomnia (i.e., how does the precipitating factor precipitate disturbance of sleep continuity?).

In the original model it is implied that the predisposition to insomnia varies across patients but is a trait factor (static over time) within the individual patient. Presumably the postulated between-subject variability means that some patients are not prone to insomnia, some are marginally at risk, and still others are at high risk. Although it stands to reason that the vulnerability for insomnia exists on a continuum (i.e., is normally distributed), it is also plausible that everyone is at risk for acute insomnia and that this is so to the extent that insomnia represents an adaptive response to stress (i.e., real or perceived threat prevents the inhibition of wakefulness; this idea is addressed by the Cano-Saper rodent model and the psychobiological inhibition model). The postulate of within-subject variability (risk being static over time) also may be open to question. Some predispositions may be indeed be hardwired (addressed by the *Drosophila* model) but it also stands to reason that some predispositions vary over the lifespan (e.g., new sleep environments or partners, pregnancy or childrearing, altered hormonal status, effects of aging. The newer rendition of the 3P model (reviewed in Chapter 144) reconciles this issue by explicitly allowing predisposing factors to vary with time).[16]

As with stimulus control, the 3P model focuses on instrumental conditioning. It does not explicitly take into account the role of classical conditioning in chronic insomnia, i.e., the likely possibility that the regular co-occurrence of wakefulness with sleep-related stimuli might lead to a second-order, and perhaps more virulent, perpetuating factor: conditioned wakefulness or conditioned arousal.

The 3P model does provide a conceptual framework for understanding types or subtypes of insomnia. For example, it addresses why some subjects have psychophysiologic insomnia as opposed to paradoxical insomnia and why, in either case, the insomnia is expressed as one phenotype as opposed to another (initial versus middle versus late insomnia).

Implications for Current and Future Research and Therapeutics

Most of the tenets of the 3P model are untested and await empirical demonstrations. Several avenues for research are possible. Family studies or medical anthropology studies could be used to evaluate the predisposition toward

insomnia. Stress-induction studies in good sleepers, like those, for example, conducted by Hall and colleagues,[17,18] could be use to produce acute insomnia and to evaluate how a variety of biopsychosocial factors mediate the magnitude of the stress response. Longitudinal studies could be used to confirm whether the putative perpetuating factor of sleep extension does indeed mediate the transition from acute to chronic insomnia.

As for therapeutics, the 3P model has served as the conceptual basis for one treatment modality in particular: sleep restriction. This therapy, while believed by many to be the single most potent component of CBT, was developed to target one particular factor (of the three) and only as it is expressed in one particular form (i.e., sleep extension). This may explain the overall value of multicomponent CBT in that the other treatment components, it can be argued, address other perpetuating factors (e.g., stimulus control addresses engaging in nonsleep activities in the bedroom, cognitive therapy addresses the problem of catastrophic or dysfunctional thinking about insomnia, sleep hygiene addresses the misuse of counter fatigue measures). Thus, the question at hand is: In what ways might the 3P model lend itself to identifying alternative treatment targets with standard or alternative methods?[19] One possibility is to develop therapies or adapt existing therapies to target predisposing factors. Such treatments could be used to increase treatment response, decrease the risk for reoccurrence (as an adjuvant to traditional CBT), or prevent first episodes of insomnia.

In the case of treatment response, depotentiation of predisposing factors might serve to augment outcomes to the extent that they are more, as opposed to less, operational. Treatment response may be boosted if the patient is hypermetabolic by nature by providing relaxation training, if the patient is anxious by nature by providing anxiolytic treatments (medical or psychotherapeutic), or if the patient is (for social reasons) sleeping in a nonpreferred sleep phase by providing some form of chronotherapy (e.g., progressive shifts in sleep scheduling, bright light treatment, or adjuvant treatment with melatonin).

In the case of preventing relapse, one could address the factors discussed earlier or could develop interventions to prevent perpetuating factors from becoming operational during recurrence (new episodes of acute insomnia). In this instance the tendency toward sleep extension could be considered a predisposing factor. This being the case, a brief behavioral intervention could be designed that specifically targets sleep extension as a means for coping with acute insomnia. Alternatively (or in addition), rational approaches to fatigue management could be developed, such as giving instructions on how to compensate for short-term sleeplessness in a way that allows normal sleep homeostasis. In the case of prophylaxis, it might well be possible to prevent many cases of chronic insomnia by replacing sleep hygiene with an empirically validated set of rules.

THE NEUROCOGNITIVE MODEL

Basic Description

The neurocognitive model[3] is based on, and is an extension of, the 3P behavioral model as described by Spielman and colleagues.[1] The central tenets of the model include:

- a pluralistic perspective of hyperarousal (cortical, cognitive and somatic arousal);
- the specification that cortical arousal (as opposed to cognitive or somatic arousal) is central to the etiology and pathophysiology of insomnia;
- the proposition that cortical arousal, in the context of chronic insomnia, occurs as a result of classical conditioning and permits cognitive processes that do not occur with normal sleep;
- the proposition that sleep initiation and maintenance problems do not occur because of hyperarousal per se but because of increased sensory and information processing at sleep onset and during non–rapid eye movement (NREM) sleep;
- the suggestion that sleep state misperception derives from increased sensory and information processing at during NREM sleep or the attenuation of the normal mesograde amnesia of sleep.

As with the "3P" behavioral model of insomnia, it is posited that acute insomnia occurs in association with predisposing and precipitating factors and that chronic insomnia occurs in association with perpetuating factors.[1] The primary perpetuating factor is a form of instrumental conditioning that occurs with sleep extension. The neurocognitive model posits that classical conditioning can also serve as perpetuating factor for chronic insomnia and stipulates that hyperarousal needs to be construed and assessed in terms of its component domains: cognitive, somatic, and cortical arousal. With these considerations in mind, it is suggested that repeated pairing of sleep-related stimuli with insomnia-related wakefulness (arousal) ultimately causes sleep-related stimuli to elicit (or maintain) higher than usual levels of cortical arousal at around sleep onset or during the sleep period. This form of arousal is not thought to be paralleled by somatic arousal (which is posited to be more characteristic of acute insomnia) and is thought to precede, and act as the biological substrate for and precipitant of, cognitive arousal in the context of chronic insomnia.

Conditioned cortical arousal is, in turn, hypothesized to contribute to disturbance of sleep continuity or to sleep state misperception via enhanced sensory processing, enhanced information processing, and long-term memory formation. Enhanced sensory processing (detection of endogenous or exogenous stimuli and, potentially, the emission of startle or orienting responses) around sleep onset and during NREM sleep is thought to directly interfere with sleep initiation or maintenance. Enhanced information processing (detection of, and discrimination between, stimuli and the formation of a short term memory of the stimulating events) during NREM sleep is thought to blur the phenomenologic distinction between sleep and wakefulness and thus contributes to sleep state misperception. Enhanced long-term memory (detection of, and discrimination between, stimuli and recollection of the stimulating event hours after its occurrence) around sleep onset and during NREM sleep is thought to interfere with the subjective experience of sleep initiation and duration and thus contributes to the discrepancies between subjectively and objectively assessed sleep continuity.

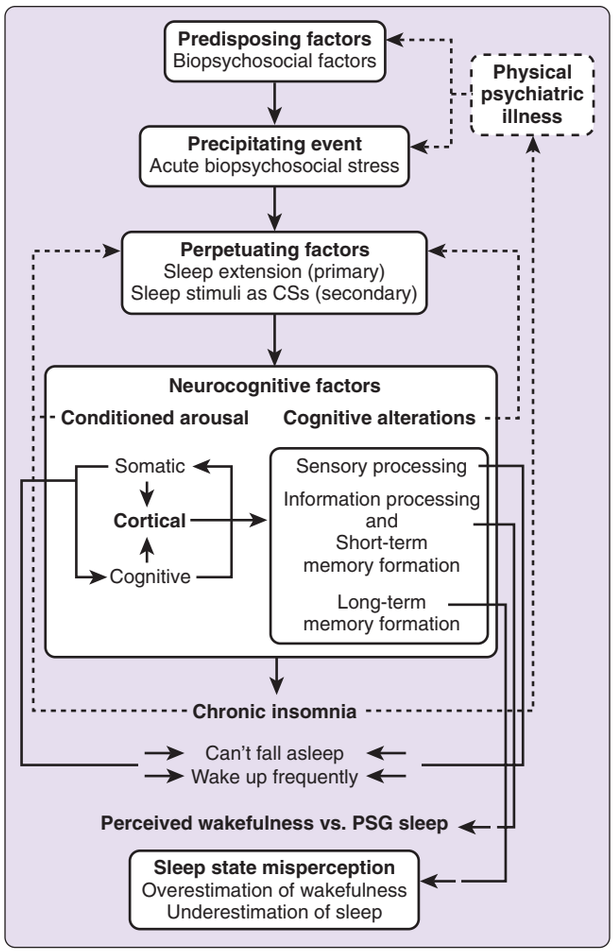


Figure 78-3 The neurocognitive model shown here differs from prior versions in several ways: *Dotted lines* are provided to highlight feedback loops; *solid lines* represent feedforward loops. The examples provided for perpetuating factors have been changed. The primary factor is designated as sleep extension (previously denoted as increased time in bed and staying awake in bed). The secondary factor is designated as sleep stimuli as conditioned stimuli. This is meant to represent when sleep stimuli become conditioned stimuli for wakefulness (arousal). CSs, ▪▪▪; PSG, polysomnographic.

Conditioned cortical arousal is hypothesized to be self-reinforcing, and for essentially two reasons. First, because sleep-related stimuli (X) act as conditioned stimuli for cortical arousal (Y), the pairing is self-reinforcing. That is, if X elicits Y, and the occurrence of Y reinforces the association of X and Y, then pairing is self-reinforcing. Second, because cortical arousal permits processes associated with wakefulness, it is likely that the elicited arousal will, on each occasion, be amplified because of ongoing sensory processing, enhanced information processing, and long-term memory formation. Taken together, these considerations virtually guarantee that the insomnia will, in the absence of its original precipitants, continue unabated and will not be subject to extinction, as usually occurs with classical conditioning. See Figure 78-3 for a schematic representation of the model.

Strengths and Weaknesses

Strengths

In general, the major strengths of the neurocognitive model are that it allows a pluralistic perspective on the concept of arousal; it does not require that hyperarousal be so intense as to directly interfere with sleep initiation and maintenance but instead posits that arousal only be sufficiently intense as to permit processes that are characteristic of wakefulness and can perpetuate wakefulness (stimulus detection, startle, orienting, stimulus identification, intention or action, and long-term recall); it delineates a mechanism beyond that of instrumental conditioning (i.e., classical conditioning as a perpetuating factor); it specifies how chronic insomnia takes on a life of its own (i.e., is self-reinforcing), and its hypotheses are falsifiable. Two lines of research (indirect and direct) provide support for the model.

The indirect evidence derives from observations about the effects of sleep on long-term memory in good sleepers and perceived wakefulness during sleep recorded on a polysomnograph (PSG) in patients with insomnia. With respect to the former, there is good evidence that normal sleepers cannot recall information from periods immediately prior to sleep,[20-23] during sleep,[24-28] or during brief arousals from sleep.[29,30] Thus, normal sleep is indeed characterized by a dense amnesia for events occurring at around sleep onset and during sleep.

With respect to the latter, there is substantial evidence that when awakened from PSG-defined sleep, patients with insomnia (as opposed to good sleepers) tend to perceive themselves to be awake rather than asleep.[31-38] This tendency, better known as *sleep state misperception*, is consistent with the neurocognitive model's perspective regarding sensory and information processing during sleep. That is, if one cue for "knowing" that one is asleep is the lack of awareness for events occurring during sleep, and if it is the case that patients with insomnia exhibit increased levels of sensory and information processing during sleep, then it would be expected that the greater level of awareness for events occurring during PSG-defined sleep serves to blur the phenomenologic distinction between sleep and wakefulness so that patients with insomnia would have difficulty indentifying PSG sleep as sleep. In this instance, what remains open to question is whether sleep state misperception can be correlated with objective measures of cortical arousal—such as by quantitative electroencephalography (qEEG), analyses of cyclic alternating pattern (CAP), or brain metabolic functional imaging—or with objective measures of increased sensory and information processing during sleep (i.e., via evoked-response potentials [ERPs]).

The direct evidence pertains to whether patients with insomnia exhibit increased cortical or central nervous system (CNS) arousal as measured by qEEG and positron emission tomography (PET), increased sensory or information processing as measured by ERPs, an attenuation in the normal mesograde amnesia of sleep, or association between sleep state misperception and objective measures of cortical arousal or ERP abnormalities. Patients with primary insomnia have been found to exhibit higher levels of cortical arousal (in terms of increased NREM high-frequency EEG) as compared to good sleepers[39-43] or patients with insomnia comorbid with major depression.[43a]

Cortical arousal (as well as increased activity involving subcortical areas and circuits) has also been observed in patients with insomnia using PET techniques.[44,45] Altered sensory and information processing have been observed with ERPs.[46-47] Correlational analyses provide evidence that that beta and gamma activity is negatively associated with the perception of sleep quality[17,48] and is positively associated with the degree of subjective-objective discrepancy.[43] There is some evidence that patients with sleep state misperception disorder (paradoxical insomnia) have been found to exhibit more beta and gamma EEG activity than good sleepers or patients with primary insomnia.[49] One study shows that patients with insomnia are better able to recognize word stimuli played during sleep-onset intervals and during early NREM sleep. This latter finding provides support for the hypothesis that there is an attenuation in the normal mesograde amnesia that accompanies sleep in patients with chronic insomnia.

Weaknesses

The primary limitations of the neurocognitive model are its failure to adequately account for the transition from good sleep to acute insomnia (like the 3P behavioral model, it primarily describes chronic insomnia), the importance of circadian and homeostatic influences on sleep, and the possibility that cortical arousal constitutes a permissive factor for worry, rumination, and monitoring behavior. The original model does not clearly address whether conditioned cortical arousal represents hyperarousal or the newer concept of the failure to inhibit wakefulness. With respect to this last point, the summary endeavors to clarify this issue by suggesting that chronic insomnia (versus acute insomnia) is perpetuated by a form of conditioned arousal that is more akin to alert wakefulness than to hyperarousal (physiologic and neurobiological states that occur with flight-or-fight–type stress responses). Finally, whereas the neurocognitive model does provide a conceptual framework for two types of insomnia (psychophysiologic and paradoxical insomnia) and how insomnia becomes self perpetuating (via classical conditioning), the model does not explicitly address how it is relevant for the other insomnia types or subtypes.

Implications for Current and Future Research and Therapeutics

There is evidence supporting the viability of the neurocognitive model, but many of the model's central tenets require further empirical validation. Apart from the research required to support the behavioral base of the model, further work is needed showing that cognitive processes (sensory and information processing and long-term memory) are reliably altered during the sleep period in patients with chronic insomnia and that altered cognitive processing has clear neurobiological concomitants (e.g., altered metabolic activity in specific brain regions) and functional consequences (sleep continuity disturbance and sleep state misperception).

Novel experimental paradigms need to be developed to test the model's core hypotheses. For example, if classical conditioning is an operative factor, experimental paradigms could also be used to evaluate whether sleep-related stimuli may be conditioned to elicit wakefulness. Experiments of this type will most likely need to be conducted in animals because they run the risk of experimental effects persisting beyond the conduct of the experiment itself. If mesograde amnesia is a primary determinant of perceived sleep quantity and quality, it should also be possible to assess the relative importance of this factor using compounds that promote amnesia (with or without sedative properties) in combination with the manipulation of situation cues. Experiments of this type will need to be conducted in humans given the centrality of self-reported sleep continuity.

The neurocognitive model may provide some insight into the potential mechanisms of action of existing therapeutics and also some guidance regarding potential targets for new treatments. In the case of existing therapeutics, pharmacotherapy might effective to the extent that the various compounds block sensory and information processing or promote amnesia within the sleep period. This idea, first espoused by Mendelson,[50-55] seems probable given the effects of benzodiazepines and benzodiazepine receptor agonists on arousal thresholds and memory formation. Sleep restriction therapy might also work via these mechanisms to the extent that this treatment modality serves to deepen sleep (augment the endogenous form of sleep-related mesograde amnesia).

Potential avenues for new medical treatments include the assessment of compounds that have greater-than-normal amnestic potential for their efficacy as hypnotics, provided that such effects can be limited to the desired sleep period and the use of diurnal stimulant therapy to promote wake extension and thereby their potential to diminish nocturnal cortical arousal via increased sleep pressure. Potential avenues for behavioral treatment include inpatient protocols that use more-intensive forms of sleep restriction therapy to promote counterconditioning, such as what is now being done with intensive sleep retraining therapy.[56]

THE PSYCHOBIOLOGICAL INHIBITION MODEL

Basic Description

The psychobiological inhibition model[15] states that stressful life events precipitate both physiologic and psychological arousal, and the consequences of this are the occurrence of selective attending to the life stressor and the occurrence of insomnia symptoms. In the case of acute insomnia, it is thought that physiologic or psychological arousal is sufficient to interfere with the normal homeostatic and circadian regulation of sleep (i.e., is sufficient to prevent the normal inhibition of wakefulness). The acute insomnia might resolve or be perpetuated based on the extent to which the stress state resolves and the patient does not attend to the acute insomnia. The shift of attention from the life stressor, implicitly or explicitly, to the insomnia symptoms is posited to be the critical event that transitions acute insomnia to a form of sleep disturbance that is self-perpetuating. A schematic representation of the model is presented in Figure 78-4.

This model substantially distinguishes itself from earlier perspectives in three fundamental and related ways. First, the point of departure for the model is the

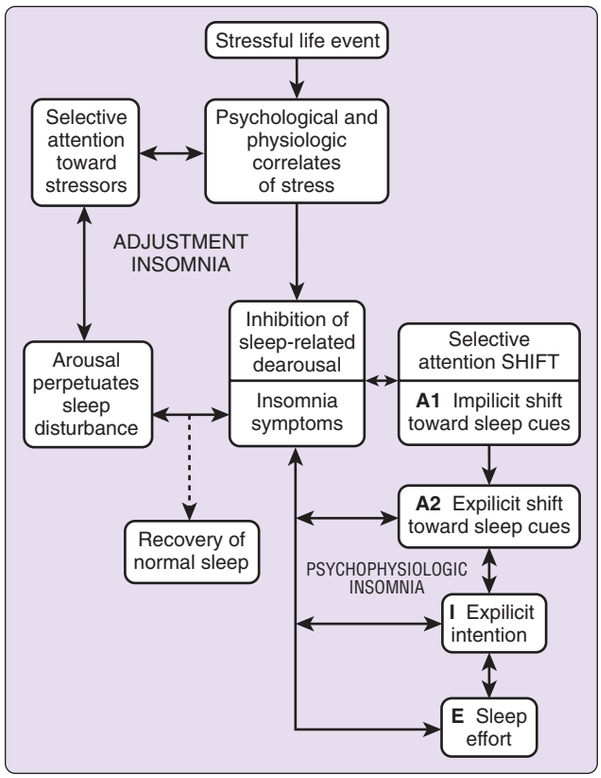


Figure 78-4 Proposed evolution of psychophysiologic insomnia from adjustment insomnia following the attention-intention-effort (A-I-E) pathway.

psychobiological framework for normal sleep that is inherent in the stimulus control perspective[4] and formally delineated in the two-process model of sleep–wake regulation.[57] Second, it is proposed (and was the first etiologic model to do so) that persistent sleep continuity disturbance occurs in relation to a failure to inhibit wakefulness (as opposed to conditioned hyperarousal). Third, the model is focused on how cognitive factors (as opposed to behavioral or physiologic factors) serve to perpetuate insomnia.

Psychobiological Framework for Normal Sleep

Espie[4,58] suggests that for normal sleepers, homeostatic and circadian processes default to good sleep, not to insomnia, and that like other neurobehavioral systems, this is ensured by plasticity and automaticity. *Plasticity* refers to the ability of the sleep system to adjust to, and/or accommodate, situational factors that disrupt normal sleep-wake functioning (e.g., circumstances that require that sleep be temporarily foreshortened or extended). In transient or acute insomnia, the norm would be the recovery of good sleep, reflecting the system's plasticity in function. *Automaticity* refers to the involuntary nature of sleep initiation and sleep maintenance. That is, that sleep is initiated and maintained automatically by the well-established conditioned associations between sleep-related stimuli and sleep and by the two-process system that governs the timing and duration of sleep and wake. Thus, under normal circumstances, sleep occurs passively (without attention, intention, or effort).

Insomnia as the Failure to Inhibit Wakefulness

The majority of insomnia models conceptualize insomnia as a disorder of hyperarousal. That is, the inability to initiate or maintain sleep derives from the occurrence of a level of arousal that is simply incompatible with sleep, where such arousal occurs acutely in relation to stress and chronically in relation to behavioral factors[58] or classical conditioning. Espie, however, has proposed an important alternative point of view, suggesting that insomnia occurs in association with a failure to inhibit wakefulness. That is, the psychobiological inhibition model suggests that in the early stages of chronic insomnia, problems with sleep initiation or sleep maintenance can occur because of fundamental alteration in the functioning of the neurobiological mechanisms that normally inhibit wakefulness and permit sleep to occur. Such an alteration is likely to be systemic; it occurs with real or perceived threat and is part of the larger flight-or-fight response. This alteration, which should dissipate along with the resolution of the acute stressor, may be potentiated by cognitive processes.

Cognitive Factors Trigger the Failure to Inhibit Wakefulness

The failure to inhibit wakefulness (in the context of chronic insomnia) is hypothesized to result from three related cognitive phenomena collectively referred to as the attention-intention-effort (A-I-E) pathway.[4] Each of the three phenomena are thought to act in concert, and in a hierarchical fashion, to transition acute stress-induced insomnia into a form of insomnia that is self-perpetuating. This is thought to occur as follows. First, when the person is unable to sleep, his or her attention is drawn to an otherwise automatic process. The very process of attending, in turn, prevents perceptual and behavioral disengagement. Second, because a primary function of attention is to promote action in response to perceived need or threat, an intentional (purposive) process is initiated that acts to further inhibit the normal downregulation of arousal. Third, when the person is unable to sleep, active effort is expended trying to fall asleep, and this effort, like enhanced attention and intention, serves only to further prevent the inhibition of wakefulness.

In sum, the psychobiological inhibition model provides a generic common pathway to chronic insomnia. Insomnia occurs in a persistent fashion when there is a sufficient level of attention, intention, or effort to outweigh good stimulus control or the intrinsic drives of the two-process system.

Strengths and Weaknesses

Strengths

There is substantial support, in general, for the concept that attention bias or selective attention plays a role in mental illness.[59] It has been found to be operational for a wide range of psychiatric disorders including panic disorder, hypochondriasis, eating disorders, obsessional disorders, generalized anxiety disorder, and posttraumatic stress disorder.[60-64] In fact, the data within these domains are sufficiently compelling that it has been argued that attention bias may have a causal role in most, if not all, anxiety disorders.[63] The concept is that the anxious person is preoccupied with danger and threat and thus

selectively attends to threat-related stimuli as he or she perceives that the danger is imminent but also potentially avoidable.

Attention bias has also been implicated in habit and dependence disorders. In this case, however, attention is not focused on threat-related stimuli per se but instead on the object of the addiction.[64] For example, in alcoholism, patients are thought to selectively attend to alcohol-related cues and that this form of attention bias can moderate or mediate addiction by producing craving. In the case of insomnia, attention bias is likely to operate in a manner akin to anxiety and dependence disorders (attention to threat or object of craving), and this might account for the nosologic requirement that insomnia (psychophysiologic insomnia) include "excessive focus on, and heightened anxiety about, sleep".[64, p. 7]

Apart from the general perspective that attention bias is relevant for mental disorders, there is also a significant amount of experimental evidence supporting the psychobiological inhibition model, and especially with respect to sleep-related attention bias and sleep-related effort. To date eight studies have been conducted whose findings reliably indicate:

- sleep-related mental preoccupation may be associated with the transition from acute to persistent insomnia in cancer patients[65];
- subjects with psychophysiologic insomnia exhibit heightened levels of attention bias as compared to good sleepers and subjects with delayed sleep phase syndrome[66,67];
- attention bias to sleep-related stimuli detected in patients with psychophysiologic insomnia may be driven by threat[57,67-69];
- there are positive linear relationships between sleep-related attentional bias and self-reported sleep quality and sleepiness;
- subjects with psychophysiologic insomnia exhibit "effortful preoccupation with sleep."[70]

Another strength of the psychobiological inhibition model is that it allows an objective means of indexing cognitive processes in insomnia. In practice, insomnia patients complain primarily of mental events interfering with sleep, including intrusive thoughts, racing thoughts, increased worry, and the inability to disengage attending to environmental noise or bodily sensations. Although such mental events appear central to the experience of insomnia, their assessment has relied primarily on self-report measures. Thus, a major strength of the psychobiological inhibition model is it concepts may be operationally defined and tested with objective measures like the computerized emotional Stroop task, the induced-change blindness task, and the dot probe task.

Finally, and perhaps most important, a major strength of the psychobiological inhibition model is the extent to which one of its central tenets (inhibition of wakefulness) is supported by both animal and human data. In the case of the former, Cano-Saper's rodent model[7] serves to highlight that there is indeed a neurobiological substrate for the concept of the failure to inhibit wakefulness and it appears to be dysregulated in rodents exposed to the cage-exchange paradigm. In humans, studies using evoked response potential methodology[46,47] suggest that patients with insomnia exhibit a diminished capacity to inhibit exterioception.

Limitations

Much of psychobiological inhibition model and the A-I-E pathway that remains to be validated (particularly the intention and effort components). Moreover, studies conducted in Glasgow now need to be replicated and extended by other research groups. An important consideration for subsequent studies will be the extent to which the psychobiological inhibition model and the A-I-E pathway apply across the range of primary insomnia types (e.g., psychophysiologic insomnia versus idiopathic and paradoxical insomnias) and the insomnia subtypes (initial, middle and late insomnias). Finally, and perhaps most difficult, is the need to create conceptualizations and measures that allow a clear distinction between, and an assessment of, the relative importance of the two primary concepts now thought to undergird the incidence and severity of insomnia: arousal or hyperarousal and the failure to inhibit wakefulness (or function typical of wakefulness).

Implications for Current and Future Research and Therapeutics

As previously suggested, the psychobiological inhibition model offers a generic common pathway to insomnia. Consequently, the model can accommodate a common pathway explanation for the effectiveness of many existing elements of cognitive behavioral therapy for insomnia (CBT-I). The model's potential explanatory power, however, is not limited to elements of CBT-I but also likely extends to potential mechanisms of action for existing medical therapeutics.

With respect to CBT-I, any behavioral or cognitive intervention that augments the inhibition of wakefulness should permit the reinstatement of normal sleep. Sleep restriction might exert its therapeutic effects via the reinstatement of sleep automaticity. That is, sleep restriction serves to increase homeostatic pressure for sleep to a point where sleep will occur inevitably and without attention, intention, or effort. Stimulus control may strengthen adaptive and automatic dearousal associations of bed and sleep and thereby diminish the conditioning effects that inhibit downregulation. Finally, relaxation, distraction, and imagery methods might reduce worry about sleep, and paradoxical intention methods might entirely refocus the A-I-E pathway away from sleep preoccupation.

With respect to the medical management of insomnia, the psychobiological inhibition model suggests that the mechanisms for existing therapies reside in their capacity to promote relaxation, inhibit exterioception, and derail sleep-related attention, intention, and effort. Clearly these are features of traditional sedatives (e.g., barbiturates, benzodiazepines, and benzodiazepine receptor agonists) but also might apply to the off-label use of antipsychotics (e.g., quetiapine, olanzapine).

Finally, the model also offers a perspective that might allow the development of new approaches. From the CBT or psychotherapeutic point of view, the psychobiological inhibition model clearly carries with it the suggestion that sensory gating training or (alternatively) mindfulness therapies may be successfully used to treat insomnia. From the

pharmacologic point of view, the psychobiological inhibition model clearly carries with it the suggestion that it may be productive to antagonize wake-promoting or wake-consolidating systems and that one such approach would be via orexin antagonism.

THE *DROSOPHILA* MODEL

Basic Description

The conceptual basis for the *Drosophila* model (as an analogue of human insomnia) is that insomnia occurs, in part, in relation to predisposing factors. This fundamental tenet of the behavioral model suggests that chronic insomnia may have a genetic component and that a portion of the variance in the incidence of insomnia[71,72] should be related to factors that are heritable, for example, the strength and plasticity of the sleep system, the trigger threshold for and intensity of the flight-or-fight response, or the strength and plasticity of sleep homeostasis and circadian processes. Consistent with this point of view is that insomnia tends to run in families and that persons with a family history of insomnia are more anxious and prone to stress-related sleep disturbances.[73,74] Thus, if insomnia results, in part, from predisposing trait characteristics, the identification of the underlying mechanisms should be feasible using genetic strategies.

Given the complexity and number of traits observed for insomnia, it seems unlikely that single gene mutations will result in an animal model that adequately captures the human condition. An alternative approach is to identify natural variants in a population that simultaneously exhibit several behavioral characteristics of insomnia. The phenotypic variation in these individuals is likely to be the result of minor changes in many genes and as a consequence is more likely to reflect the diversity of the human disorder.[75] The natural polygenic variation can be amplified over successive generations using laboratory selection and can be identified using whole-genome arrays.[76] This is the approach that undergirds the *Drosophila* model.

Evaluation of a normative dataset of wild-type *Canton-S* (*Cs*) *Drosophila* indicated that they display a sufficient range of sleep times and activity levels to make them suitable for laboratory selection (Figure 78-5A, *green bars*). *Drosophila* that demonstrated reduced sleep time in combination with increased sleep latency, reduced sleep bout duration, and elevated levels of waking activity (*insomnia-like*, referred to as *ins-l flies*) were selected and bred over successive generations. As seen in Figure 78-5B, total sleep time was progressively reduced during selection and stabilized after 60 generations. At generation 65, more than 50% of *ins-l* flies obtained less than 60 minutes of sleep in a day, and the distribution of sleep times was shifted dramatically to the left (see Fig. 78-5A, *pink bars*). Not surprisingly, the decrease in sleep time (or increase in total wake time) in selected *Drosophila* came primarily at the expense of nighttime sleep (see Fig. 78-5C). As with human insomnia, *ins-l* flies showed increased latency from lights off to the first sleep bout of the night (see Fig. 78-5D), suggesting that they have difficulty initiating sleep.[77] The *ins-l* flies also exhibited difficulties maintaining sleep as evidenced by an inability to consolidate sleep into long bouts (average sleep bout duration; see Fig. 78-5E).[7] The maximum episode of consolidated sleep that can be generated by an *ins-l* fly is only 36 ± 9 minutes versus 257 ± 22 minutes in *Cs* flies. In addition to disrupted sleep, the *ins-l* flies exhibit increased locomotor activity during waking (1.86 ± 0.03 crossings) compared to *Cs* flies (1.42 ± 0.05 crossings).

To assess the extent to which the sleep patterns of the selectively bred *Drosophila* represent a reasonable analogue of human insomnia (chronic insomnia), the sleep of the *ins-l Drosophila* was evaluated for chronicity (i.e., stability of the abnormal sleep pattern over the life span) and wake state of the *ins-l Drosophila* was evaluated for evidence that the altered form of sleep was associated with daytime consequences (i.e., fatigue, sleepiness, impaired concentration or memory) or health outcomes (increased mortality). With respect to chronicity, it was found that the sleep profile remained stable in *ins-l Drosophila* over time. Total sleep for three representative *ins-l* flies and one *Cs* fly are shown in Figure 78-5F. These three *ins-l* flies obtained a total of 358 ± 128 minutes of sleep during their first 20 days of life (and this appears stable with time) versus 17,567 ± 655 minutes of sleep over the same time period in the *Cs* flies (and this appears to trend downward with aging). Thus, the observed stability of the sleep profile of the *ins-l* flies may be viewed as chronic (i.e., the sleep disturbance does not spontaneously remit with time).

With respect to daytime consequences, the two types of *Drosophila* were evaluated for sleepiness, learning impairment, and motor or coordination difficulties. *Sleepiness* was assessed using a biomarker for sleepiness (amylase).[6] As seen in Figure 78-5G, *ins-l* flies show elevated levels of amylase relative to *Cs* flies, suggesting that they experience sleepiness (or increased sleep drive) during their primary wake period. Learning was assessed using aversive phototaxic suppression (APS).[78] In this task, flies learn to avoid a light that is paired with an aversive stimulus (quinine or humidity). In this paradigm it was shown that APS is sensitive to both sleep loss and sleep fragmentation.[79] For example, as seen in Figure 78-5H, learning is significantly impaired in the shortest sleeping *ins-l*$_{short}$ flies compared to *Cs* controls. To determine whether the selection protocol generated poor-learning *Drosophila* as a phenotype independent from the observed sleep deficit, learning in long-sleeping *ins-l* flies was also evaluated. Longer sleeping siblings maintained their ability to learn, indicating that the selection procedure did not inadvertently, and independent of sleep, contribute to poor learning. Motor and coordination difficulties were assessed by quantifying the number of spontaneous falls in young age-matched *Cs* and *ins-l* flies walking for 30 minutes in an obstacle-free environment. *Cs* flies rarely fall under these conditions. In contrast, *ins-l* flies often lose their balance (see Fig 78-5I).

Finally, health consequences were assessed in terms of mortality via a measure of lifespan duration. This approach derives from the epidemiologic studies in humans that suggest that sleep duration and insomnia are associated with an increased risk of all-cause mortality and reduced lifespan.[80] Accordingly, one would predict that if the selected lines have insomnia or are getting less sleep than they need, they would have a shortened lifespan. As seen in Figure 78-5J, this is indeed the case. It should be noted that the reduced lifespan observed in the *ins-l* flies is not a result of decreased fitness.

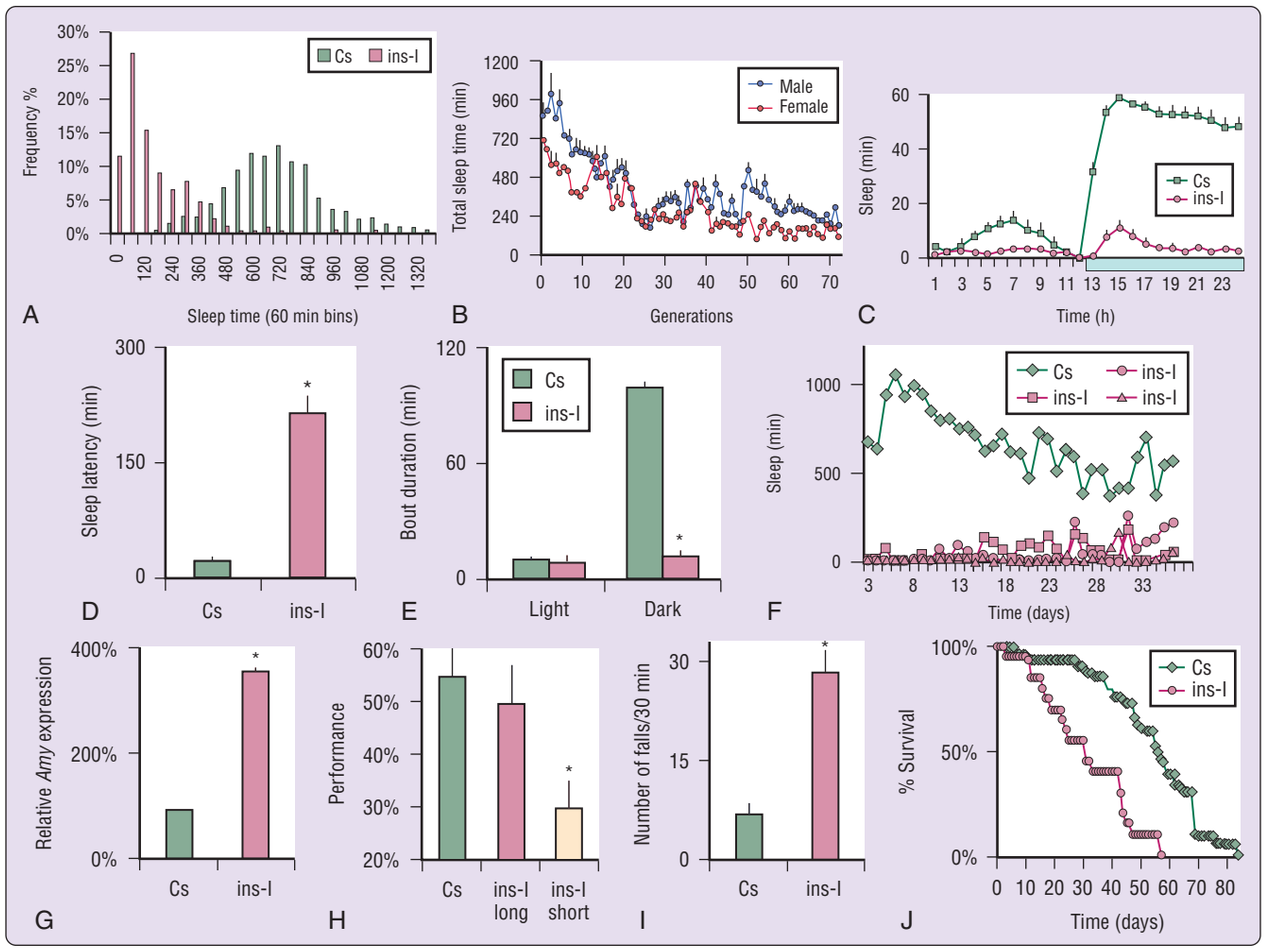


Figure 78-5 **A**, Frequency distribution of total sleep time in 60 min bins in wild type *Cs* flies ($n = 1000$) and in *ins-l* flies at generation 65 ($n = 364$). **B**, Total sleep time in males ($n = 40$) and females ($n = 40$) for successive generations of *ins-l* flies. **C,** Daily total sleep time is shown for 37 days in one *Cs* and three *ins-l* flies. **D**, Sleep in min/hr for 24 hr in *Cs* flies ($n = 32$) and *ins-l* flies ($n = 32$). The *gray rectangle* represents lights off. **E**, Sleep latency is increased in *ins-l* flies ($n = 28$) versus *Cs* flies ($n = 33$). **F,** Average sleep bout duration is reduced during the dark period in *ins-l* flies ($n = 32$) versus *Cs* flies ($n = 32$). **G,** Amylase mRNA levels are elevated in *ins-l* flies (% of Cs expression) at ZT0-1. **H,** Learning in *Cs* flies, longer-sleeping *ins-l* flies (average daily sleep time, 347 ± 55 min), and short-sleeping *ins-l* flies (average daily sleep time, 26 ± 7min) ($n = 10$ for each group) **I,** Number of falls during 30 minutes in *Cs* flies ($n = 20$) and *ins-l* flies ($n = 18$). **J,** Representative survival curve of aging *ins-l* compared to *Cs* flies (30 flies/group). *$P < .05$; error bars represent standard error of the mean.

In sum, the selection procedure was effective in producing animals with reduced total sleep time, increased sleep latency, and shortened sleeping bout duration. These sleep effects were found to be persistent and were associated with a variety of sequelae including diurnal sleepiness, impaired learning, motor or coordination difficulties, and increased mortality. Taken together, these findings suggest that the *Drosophila* model is a reasonable analogue of human insomnia (chronic insomnia).

Strengths and Weaknesses

A major strength of the *Drosophila* model is its approach: a naturally occurring set of sleep parameters (parameters that are commonly found in human insomnia) were operationally defined for use in the fly and amplified over successive generations using laboratory selection. Not only is the selection procedure an ideal one for a genetic study of insomnia, but also the use of multiple parameters ensures that the analogue condition more closely resembles the human expression of the disorder. Another strength of the model is the effort to demonstrate that the aggregate phenotype also exhibited daytime deficits with respect to sleepiness, learning impairment, coordination difficulties, and reduced lifespan.

One weakness of the model is the inability to establish (as with any animal model) the subjective complaint of insomnia. Another weakness is a level of insomnia severity that is not analogous to that seen in humans: Total sleep times are a fraction of the total sleep time seen in non–*ins-l* flies. This might suggest to some that the selection process produced a new class of sleep mutant as opposed to an idiopathic form of insomnia. The demonstration of sleepiness the *ins-l* flies are controversial. The consensus view (based on the use of the multiple sleep latentcy test [MSLT]) is that patients with chronic insomnia do not exhibit pathologic sleepiness. Finally, the method of

assessment of sleepiness is also somewhat controversial to the extent that amylase is also used as a biomarker for stress.[81-84]

Implications for Current and Future Research and Therapeutics

There are a variety of possible directions for future research. Given the complexity of insomnia, it is likely that independent selections would potentially yield alternative outcomes. That is, the genes identified in the *ins-1* flies might only represent one potential pathway to insomnia. Thus, a greater understanding of insomnia may be advanced with additional selected lines. Further, the use of molecular-genetic and genomic strategies such as Affymetrix arrays, suppressor screens, and genetic mapping may be useful for the identification of the genes that are associated with the various aggregate phenotypes. Given this latter strategy, it is important to acknowledge that gene profiling in *Drosophila* obtained by laboratory selection is likely to reveal two classes of genes: those that are causative for a given behavior and those that are a consequence of the behavioral change.[85] Most studies have focused on identifying genes that are causative for a given behavior.[76,86,87] However, given that extended waking results in substantial physiologic impairment,[88,89] including death,[90,91] the latter set of genes may also be particularly important in the context of insomnia.

THE RODENT MODEL OF ACUTE INSOMNIA

Basic Description

A rat model of acute stress–induced insomnia has been developed using a species-specific psychological stressor, cage exchange. The aim of this model was to identify the brain circuitry activated in rats experiencing stress-induced insomnia to better understand the neurobiological basis of acute insomnia.

In the cage-exchange paradigm, stress is induced by manipulating the social context rather than by applying a continual physical stressor (e.g., tone, shock).[7,44] This is accomplished by transferring a male rat from his home cage, at the peak of the sleep period, to a soiled cage previously occupied by another male rat. Because rats are very territorial, exposure to the olfactory and visual cues of a competitor, even in its absence, induces a stress fight-or-flight response including activation of the autonomic nervous system and the hypothalamic-pituitary-adrenal axis and sustained wakefulness. Several hours later, when the physiologic indicators of acute stress are attenuated, this manipulation induces a late period of disturbed sleep.

The brain circuitry activated during this late period of disturbed sleep was assessed by examining the expression of Fos, a transcription factor widely used as a marker of neuronal activity. Increased activation was observed in the cerebral cortex, limbic system, some arousal groups (locus coeruleus and tuberomamillary nucleus), and part of the autonomic system. Surprisingly, there was also simultaneous activation of the sleep-promoting areas: the ventrolateral preoptic area (VLPO) and the median preoptic nucleus. This coactivation results in a unique pattern of brain activity that differs from those observed during wake or normal sleep, because the sleep circuitry appears to be like that in a sleeping rat, whereas the arousal system and the cortex show a level of activation similar to those of wakefulness. The high level of cortical activation (as measured by Fos) was also associated with high-frequency EEG activity (distinctive of wakefulness) during NREM sleep. The co-occurrence of high-frequency EEG activity and traditional sleep frequencies also appears to represent a novel intermediate state that differs from both normal EEG sleep and wakefulness.

Subsequent experiments revealed that inactivation of discrete limbic or arousal regions, via cell-specific lesions or pharmacologic inhibition, allowed the recovery of specific sleep parameters and changed the pattern of brain activity in the cage-exchange paradigm.[7] This suggests that stress-induced insomnia requires the occurrence of a cascade of neuronal events along with the normal propensity for sleep. This cascade likely includes sensory inputs (olfactory and visual cues of a competitor) that activate limbic areas, which in turn activate part of the arousal system that subsequently activates the cerebral cortex. This latter event (cortical activation) may be measured as high-frequency EEG activity during NREM sleep, and it is eliminated after inactivating parts of the arousal system, which supports the proposed pattern of brain activation represented in Figure 78-6.

The proposition that this particular stress paradigm can induce a *novel intermediate state* needs to be considered within the context of normal sleep–wake control,[92,93] as the

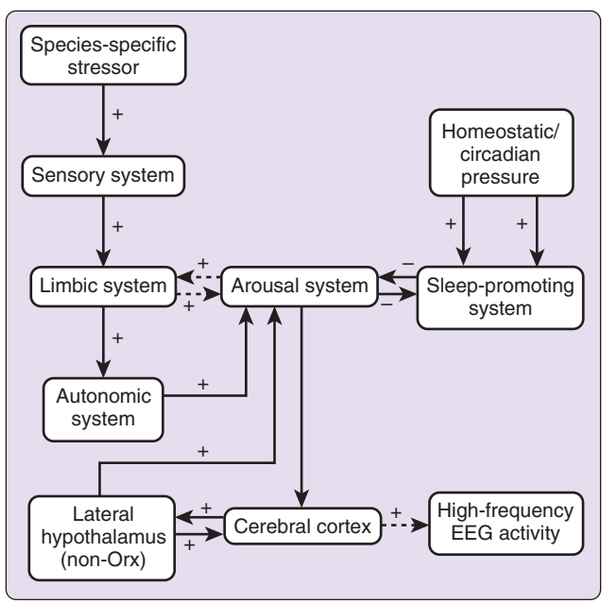


Figure 78-6 Putative circuitry involved in stress-induced insomnia: The olfactory signals are conveyed to the limbic system, which in turn activates the arousal and autonomic systems, as well as nonorexin neurons in the lateral hypothalamus. The cerebral cortex becomes highly activated by inputs from the arousal system and the lateral hypothalamus, which generates the high-frequency activity observed during NREM sleep. The reciprocal inhibition between the sleep and arousal systems would ordinarily prevent co-activation, but the homeostatic and circadian pressure keep the sleep system activated, whereas stress activates the arousal system, resulting in a unique pattern of brain activity.

existence of such a state represents an aberration of what is considered normal sleep and wake. As proposed by Saper and colleagues,[93] in normal animals there is a reciprocal inhibitory innervation between the main sleep-promoting neuronal group (VLPO), whose neurons are active during sleep, and the neuronal groups that compose the arousal system—the histaminergic tuberomammillary nucleus, the serotoninergic dorsal raphe, and the noradrenergic locus coeruleus—whose neurons are active during wakefulness. The authors have proposed that this reciprocal inhibition provides a control system that is analogous to an electrical flip-flop switch. In this case, when one side is strongly activated, it inhibits and deactivates the other side, which decreases the inhibitory input to itself (disinhibition) and reinforces its own activity. In the absence of other factors, this configuration renders a bistable circuit (stable in one or the other state), with rapid and complete transitions between states and no occurrence of intermediate states (co-activation). The circuit is switched from one state to the other due to the strong inputs generated by the gradual buildup of the circadian and homeostatic pressures. As summarized by Saper:

> *When this pressure to change becomes great enough, the same feedback properties that allow the flip-flop circuit to resist change will suddenly give way and rapidly produce a reversal of firing patterns. The flip-flop switch therefore changes behavioral state infrequently but rapidly, in contrast to the homeostatic and circadian inputs, which change continuously and slowly.*[93, p. 729]

In this context, the simultaneous activation of the VLPO and the arousal system in the cage-exchange paradigm is surprising. A possible explanation is that during stress-induced insomnia, the VLPO is fully activated because of both the homeostatic and circadian drives, but it is unable to turn off the arousal system because this is being excited intensely by inputs from the cortical and limbic systems. At the same time, the arousal system cannot turn off the VLPO because it is highly active owing to the stronger homeostatic pressure caused by the fact that the stressed rats are partially sleep deprived. This results in the simultaneous activation of opposing systems that normally are not activated in tandem, and the bistable circuit becomes inherently unstable: The switch is forced into an intermediate position. This scenario is represented in Figure 78-7.

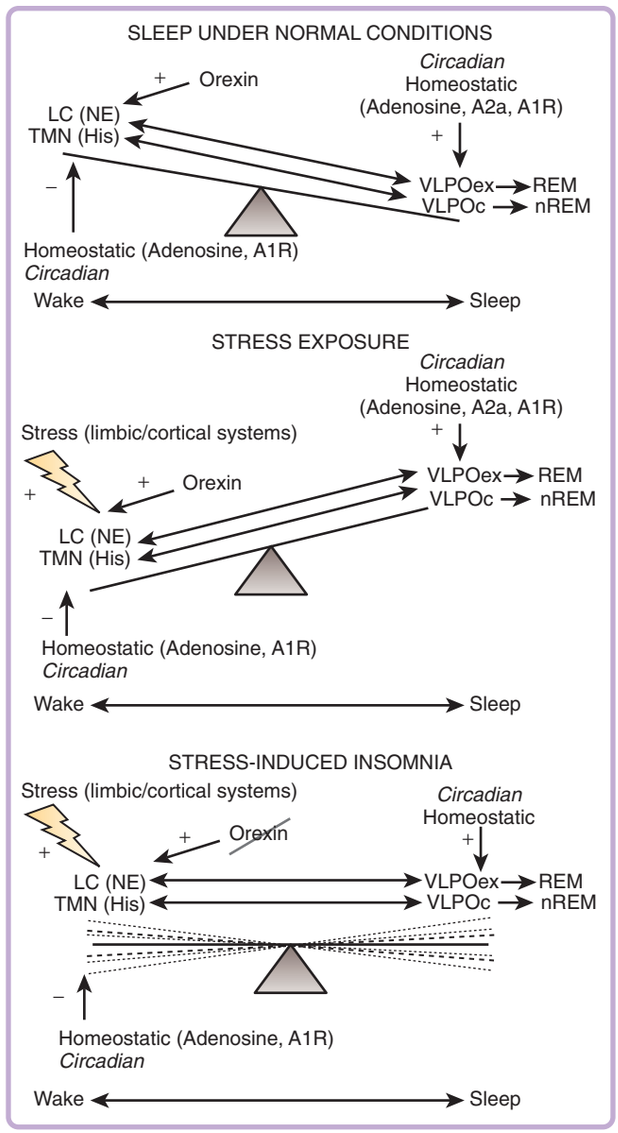


Figure 78-7 During normal sleep, the circadian and homeostatic drives enhance the activity of the sleep-promoting areas and simultaneously inhibit the arousal system, favoring the sleep state (the homeostatic effect is mediated in part by adenosine acting on A_1 and A_{2a} receptors). Stress activates part of the arousal system via cortical and limbic inputs, and this activation opposes the direction of the circadian and homeostatic drives. In stress-induced insomnia, the cortical, limbic, and arousal activation persists, but the homeostatic pressure is stronger than usual because the rats are partially sleep deprived; the circadian drive still favors the sleep state. Because these two forces are opposing and strong, the sleep–wake switch is forced into an unstable position, allowing the emergence of an intermediate state in which both sleep and wake circuitries are activated simultaneously, but each state is unable to sufficiently inhibit the other to prevent it from firing. His, histamine; LC, locus coeruleus; NE, norepinephrine; TMN, tuberomammillary nucleusVLPOc, ventrolateral preoptic nucleus core; VLPOex, ventrolateral preoptic nucleus extended.

Strengths and Weaknesses

The rat model's cage exchange paradigm has several strengths. One is the conceptualization of insomnia as part of, or precipitated by, the flight-or-fight response. It uses a psychosocial stressor (perceived territorial threat) to induce sleep continuity disturbance, and it successfully produces a form of acute insomnia that includes both initial and late subtypes. It identifies specific neuronal effects within regions implicated in the regulation of sleep and wakefulness and produces quantitative EEG findings that are consistent with those found in human insomnia. Its overall findings are consistent with the conceptualization of insomnia as a disorder of hyperarousal, and it neuronal findings suggest that acute insomnia is a hybrid state resulting from the co-activation of systems that normally function in bistable fashion.

The characterization of acute insomnia as part of (or as a consequence of) the flight-or-fight response is particularly useful. This suggests that insomnia may be, as a transitory phenomenon, an adaptive response to perceived threat[94] and is consistent with Richardson's[95] proposal that insomnia reflects the overactivity of systems extrinsic to the sleep–wake circuitry that can temporally override normal sleep–wake control to facilitate a more imperative function, the stress response. The suggestion that acute insomnia exists as a hybrid state represents an important refinement of the concept of hyperarousal and is consistent with the transition probability model described by Merica and colleagues [96,97] and with Espie's concept that insomnia occurs in association with a failure to inhibit wakefulness.[4,58]

The rat model of acute insomnia does have some limitations. As with the *Drosophila* model, it is unable to establish the subjective complaint of insomnia, and as an analogue of acute insomnia, it might not be relevant for assessing chronic insomnia, which many would argue is the more clinically relevant condition. Although there is no question that modeling chronic insomnia (e.g., using a conditioning paradigm) would be useful, the acute model might nevertheless serve as a guide for what to expect in chronic insomnia. For example, the model clearly identifies brain regions of interest and clearly delineates one kind of pathology that may be characteristic of both acute and chronic insomnia, namely, the co-activation of both sides of the flip-flop switch. Another limitation of the model may be its reliance on the Fos measure. Not all neuronal groups express Fos in association with action potential activity. Thus, this might limit the resolution of the neurobiological effects of the cage-exchange paradigm to regions that express Fos.

Implications for Current and Future Research and Therapeutics

Observations from the rat model might help to identify putative targets for pharmacologic manipulation that can guide the development of new therapies. One essential finding is that the sleep-promoting neuronal groups are fully active in the rat model, and the problem seems to be the anomalous residual activation of the arousal and limbic systems at a time they should be completely off. This suggests that shutting down the residual activity of these systems might be a better approach to treat stress-induced insomnia (and perhaps chronic insomnia) rather than potentiation of the sleep system. Further, identifying the phenotype of these neurobiological abnormalities may be helpful in the search for more-specific pharmacologic treatments, which may, in turn, yield fewer unwanted side effects.

CONCLUSION

The neurocognitive model, psychobiological inhibition model, and the Cano-Saper rodent model share at least two central tenets: Stress (threat or perceived threat) is a major precipitant of acute insomnia, and chronic insomnia involves a hybrid state where there are simultaneously higher than normal levels of CNS activation and a failure to inhibit processes normally associated with wakefulness.

The neurocognitive model and the psychobiological inhibition model differ with respect to the role of cognitive processes as they occur in chronic insomnia. The psychobiological inhibition model allows mental activity and cognitive processes to assume a central role in perpetuating insomnia: The person is awake because he or she is worrying or is attending to not sleeping. The neurocognitive model takes into account cognitive processes but does not ascribe a primary role to such phenomena: One is worrying or is attending to not sleeping because he or she is awake. Thus, cognitive phenomena serve as the flame for the psychobiological inhibition model and wind to the flame for the neurocognitive model.

The Cano-Saper model differs from the human models, at the conceptual level, because of its mechanistic emphasis on the sleep switch (as opposed to functional or environmental factors) and dysregulation of the sleep switch as it occurs acutely with homeostatic and circadian dysregulation. This difference is, however, not as profound as one might think. The question is what happens over time? Is it possible that rodents can develop chronic insomnia, and if so does this occur in a fashion that is analogous to, or relevant for, the human condition ? In the absence of data, it stands to reason that the conditioning factors that appear to be operative with human insomnia are also likely to be operational in the rodent. If true, an animal model of chronic insomnia is possible and may be used to explore the effects of conditioned arousal or conditioned wakefulness on brain function, physiology, and anatomy.

In the end, the differences in emphasis among the neurocognitive model, the psychobiological inhibition model, and the Cano-Saper model might not be a matter of which is correct but rather at what point the various models are more or less relevant. The assessment of such a proposition awaits empirical assessment, such as a large-scale natural history study focused on the factors that mediate the transitions from good sleep to acute insomnia and from acute insomnia to chronic insomnia. It stands to reason that stress response, attention bias, conditioning, and altered neurobiology all play a role across the trajectory from acute to chronic insomnia.

Finally, there is the *Drosophila* model. At first glance it appears that this model highlights an entirely different component or phase of the disease process (genetic predisposition) and as such is potentially more relevant for idiopathic insomnia and has little overlap with models that are almost entirely focused on the precipitation and perpetuation of insomnia in the context of psychophysiologic or paradoxical insomnia. This, however, might not be the case.

The Shaw *Drosophila* model might also be relevant for issues pertaining to the precipitation and perpetuation of insomnia. This may be true because the selective breeding paradigm might have resulted in a fundamental alteration of the strength and plasticity of the sleep system, the trigger threshold for and intensity of the flight-or-fight response, or the robustness of sleep or circadian processes. It is also possible that the selective breeding paradigm (particularly the short sleep aspect) might have itself directly predisposed the animals to insomnia. That is,

Shaw and colleagues bred short sleepers without altering the environment (duration of the light–dark cycles) in such a way as to be compatible with short sleep. Thus, as with humans, it is possible that the insomnia was expressed as a result of a mismatch between sleep ability and sleep opportunity.

In humans with psychophysiologic (and potentially paradoxical) insomnia, this mismatch is posited to result from sleep extension (activities enacted to recover lost sleep). In the *Drosophila* model the mismatch might occur because the animal cannot escape the environmental imperative for sleep. As with the above speculation, this concept also awaits an empirical evaluation; in this case one where the light–dark cycles may be altered to match sleep–wake propensity or where an opportunity is provided for the fly to manipulate its environment so as to allow light and dark exposure on demand. Under such conditions, if negative consequences are attenuated, this would suggest that the *Drosophila* model is also relevant for the primary insomnias.

Acknowledgement

The authors thank Dan Buysse for his feedback on this chapter. His insights and challenging perspective served to make this work immeasurably better.

❖ Clinical Pearls

While many consider theory and experimental models of clinical entities to be largely academic enterprises, this is far from the case for the models summarized in the present chapter. Each model clearly suggests one or more targets for treatment, which might or might not be currently addressed by current therapeutics.

REFERENCES

1. Spielman A, Caruso L, Glovinsky P. A behavioral perspective on insomnia treatment. Psychiatr Clin North Am 1987;10:541-553.
2. Bootzin RR. Stimulus control treatment for insomnia. Proceedings, 80th Annual Convention, Amer Psycholog Assoc 1972;395-396.
3. Perlis ML, Giles DE, Mendelson WB, et al. Psychophysiological insomnia: the behavioural model and a neurocognitive perspective. J Sleep Res 1997;6:179-188.
4. Espie CA, Broomfield NM, MacMahon KMA, et al. The attention-intention-effort pathway in the development of psychophysiologic insomnia: an invited theoretical review. Sleep Med Rev 2006;10:215-245.
5. Seugnet L, Israel S, Toledo R, Shaw PJ. Evaluating a *Drosophila* model of insomnia. Sleep 2004;27:A859.
6. Seugnet L, Boero J, Gottschalk L, et al. Identification of a biomarker for sleep drive in flies and humans. Proc Natl Acad Sci U S A 2006; 103:19913-19918.
7. Cano G, Mochizuki T, Saper CB. Neural circuitry of stress-induced insomnia in rats. J Neurosci 2008;28:10167-10184.
8. Morin CM, Culbert JP, Schwartz SM. Nonpharmacological interventions for insomnia: a meta-analysis of treatment efficacy. Am J Psychiatry 1994;151:1172-1180.
9. Murtagh DR, Greenwood KM. Identifying effective psychological treatments for insomnia: a meta-analysis. J Consult Clin Psychol 1995;63:79-89.
10. Smith MT, Perlis ML, Park A, et al. Behavioral treatment vs pharmacotherapy for insomnia—a comparative meta-analyses. Am J Psychiatry 2002;59:5-11.
11. Morgenthaler T, Kramer M, Alessi C, et al. Practice parameters for the psychological and behavioral treatment of insomnia: an update. an American Academy of Sleep Medicine report. Sleep 2006;29:1415-1419.
12. Morin CM, Bootzin RR, Buysse DJ, et al. Psychological and behavioral treatment of insomnia: update of the recent evidence (1998-2009). Sleep 2006;29(11):1398-1419.
13. Davies R, Lacks P, Storandt M, Bertelson AD. Countercontrol treatment of sleep-maintenance insomnia in relation to age. Psychol Aging 1986;1:233-238.
14. Spielman AJ, Saskin P, Thorpy MJ. Treatment of chronic insomnia by restriction of time in bed. Sleep 1987;10:45-56.
15. Borbely AA. A two process model of sleep regulation. Hum Neurobiol 1982;1:195-204.
16. Spielman AJ, Yang C, Glovinsky PB. Assessment techniques for insomnia. In: Kryger MH, Roth TG, Dement WC, editors. Principles and practice of sleep medicine. Philadelphia: WB Saunders; 2000. p. 1239-1250.
17. Hall M, Buysse DJ, Nowell PD, et al. Symptoms of stress and depression as correlates of sleep in primary insomnia. Psychosom Med 2000;62:227-230.
18. Martica MH. The impact of stress and intrusive thoughts on objective and subjective measures of sleep. University of Pittsburgh, 1995, 135 page AAT 9601255. Section B: the Sciences & Engineering 56-Univ.
19. Riedel BW, Lichstein KL, Dwyer WO. Sleep compression and sleep education for older insomniacs: self-help versus therapist guidance. Psychol Aging 1995;10(1):54-63.
20. Anthony J, Wyatt J, Bootzin R, et al. Retrograde and anterograde amnesia at sleep onset: a conceptual replication. Sleep Res 1994; 23:112.
21. Wyatt J, Bootzin R, Anthony J, Bazant S. Sleep onset is associated with retrograde and anterograde amnesia. Sleep 1994;17:502-511.
22. Guilleminault C, Dement W. Amnesia and disorders of excessive daytime sleepiness. In: Drucker-Colin R, McGaugh J, editors. Neurobiology of sleep and memory. New York: Academic Press; 1977. p. 439-456.
23. Portnoff G, Baekeland F, Goodenough DR, et al. Retention of verbal materials perceived immediately prior to onset of non-REM sleep. Percept Mot Skills 1966;22:751-758.
24. Wood J, Bootzin R, Kihlstrom J, Schachter D. Implicit and explicit memory for verbal information presented during sleep. Psycholo Sci 1992;3:236-239.
25. Bootzin R, Fleming G, Perlis M, et al. Short and long term memory for stimuli presented during sleep. Sleep Res 1991;20:258.
26. Koukkou M, Lehmann D. EEG and memory storage experiments with humans. Electroencephalogr Clin Neurophysiol 1968;25:455-462.
27. Lehmann D, Koukkou M. Computer analysis of EEG wakefulness-sleep patterns during learning of novel and familiar sentences. Electroencephalogr Clin Neurophysiol 1974;37:73-84.
28. Lasaga J, Lasaga A. Sleep learning and progressive blurring of perception during sleep. Percept Mot Skills 1973;37:51-62.
29. Goodenough D, Sapan J, Cohen H. Some experiments concerning the effects of sleep on memory. Psychophysiology 1971;8:749-762.
30. Bonnet M. Memory for events occurring during arousal from sleep. Psychophysiology 1983;20:81-87.
31. Edinger JD, Fins A. The distribution and clinical significance of sleep time misperceptions among insomniacs. Sleep 1995;18:232-239.
32. Knab B, Engel RR. Perception of waking and sleeping: possible implications for the evaluation of insomnia. Sleep 1988;11:265-272.
33. Coates T, Killen J, Silberman S, et al. Cognitive activity, sleep disturbance, and stage specific differences between recorded and reported sleep. Psychophysiology 1983;20:243.
34. Lutz T, Roth T, Kramer M, Tietz E. The relationship between objective and subjective evaluations of sleep in insomniacs. Sleep Res 1977;6:178.
35. Frankel BL, Coursey R, Buchbinder R, Snyder F. Recorded and reported sleep in primary chronic insomnia. Arch Gen Psuchiatry 1976;33:615-623.
36. Carskadon MA, Dement W, Mitler M, et al. Self-report versus sleep laboratory findings in 122 drug-free subjects with complaints of chronic insomnia. Am J Psychiatry 1976;12:1382-1388.
37. Bixler E, Kales A, Leo I, Slye E. A comparison of subjective estimates and objective sleep laboratory findings in insomnia patients. Sleep Res 1973;2:143.

38. Rechtschaffen A, Monroe L. Laboratory studies of insomnia. In: Kales A, editor. Sleep: physiology and pathology. Philadelphia: JB Lippincott; 1969. p. 158-169.
39. Freedman R. EEG power in sleep onset insomnia. Electroencephalogr Clin Neurophysiol 1986;63:408-413.
40. Merica H, Gaillard JM. The EEG of the sleep onset period in insomnia: a discriminant analysis. Physiol Behav 1992;52:199-204.
41. Merica H, Blois R, Gaillard JM. Spectral characteristics of sleep EEG in chronic insomnia. Eur J Neurosci 1998;10:1826-1834.
42. Lamarche CH, Ogilvie RD. Electrophysiological changes during the sleep onset period of psychophysiological insomniacs, psychiatric insomniacs, and normal sleepers. Sleep 1997;20:724-733.
43. Perlis ML, Smith MT, Orff HJ, et al. Beta/Gamma EEG activity in patients with primary and secondary insomnia and good sleeper controls. Sleep 2001;24:110-117.
43a. Fernardez-Mendoza J, Vela-Bueno A, Vgontzas AN, Ramos-Platôn MJ, et al. Cognitive-emotional hyperarousal as a premorbid characteristic of individuals vulnerable to insumnia. Psychosom Med 2010;72(4):397-403.
44. Nofzinger EA, Buysse DJ, Germain A, et al. Functional neuroimaging evidence for hyperarousal in insomnia. Am J Psychiatry 2004;161:2126-2129.
45. Nofzinger EA, Buysse DJ, Germain A, et al. A comparison of regional cerebral metabolism across waking and NREM sleep between primary insomnia and major depression. Sleep 2005;28:A232-A233.
46. Bastien CH, St Jean G, Morin CM, et al. Chronic psychophysiological insomnia: hyperarousal and/or inhibition deficits? An ERPs investigation. Sleep 2008;31:887-898.
47. Yang CM, Lo HS. ERP evidence of enhanced excitatory and reduced inhibitory processes of auditory stimuli during sleep in patients with primary insomnia. Sleep 2007;30:585-592.
48. Nofzinger EA, Price JC, Meltzer CC, et al. Towards a neurobiology of dysfunctional arousal in depression: the relationship between beta EEG power and regional cerebral glucose metabolism during NREM sleep. Psychiatry Res 2000;98:71-91.
49. Krystal AD, Edinger JD, Wohlgemuth WK, Marsh GR. NREM sleep EEG frequency spectral correlates of sleep complaints in primary insomnia subtypes. Sleep 2002;25:630-640.
50. Mendelson W, Martin J, Stephens H, et al. Effects of flurazapam on sleep, arousal threshold, and perception of being asleep. Psychopharmacology (Berl) 1988;95:258-262.
51. Mendelson W. Do studies of sedative/hypnotics suggest the nature of chronic insomnia. In: Monplasir J, Godbout R, editors. Sleep and biological rhythms. New York: Oxford University Press; 1990. p. 209-218.
52. Mendelson W, Maczaj M. Effects of triazolam on the perception of sleep and wakefulness in insomniacs. Ann Clin Psychiatry 1990;2:211-216.
53. Mendelson W. Pharmacologic alteration of the perception of being awake. Sleep 1993;16:641-646.
54. Mendelson W. Hypnotics and the perception of being asleep. ASDA News 1994;1:33.
55. Mendelson WB. Effects of time of night and sleep stage on perception of sleep in subjects with sleep state misperception. Psychobiology 1998;26:73-78.
56. Harris J, Lack L, Wright H, et al. Intensive sleep retraining treatment for chronic primary insomnia: a preliminary investigation. J Sleep Res 2007;16:276-284.
57. Marchetti LM, Biello SM, Broomfield NM, et al. Who is pre-occupied with sleep? A comparison of attention bias in people with psychophysiological insomnia, delayed sleep phase syndrome and good sleepers using the induced change blindness paradigm. J Sleep Res 2006;15:212-221.
58. Espie CA. Insomnia: conceptual issues in the development, persistence, and treatment of sleep disorder in adults. Annu Rev Psychol 2002;53:215-243.
59. Shiffrin RM, Schneider W. Controlled and automatic human information-processing 2. Perceptual learning, automatic attending, and a general theory. Psychol Rev 1977;84:127-190.
60. Mogg K, Bradley BP. A cognitive-motivational analysis of anxiety. Behav Res Ther 1998;36:809-848.
61. Williams JMG, Watts FN, MacLeod CM, Mathews A. Cognitive psychology and emotional disorders. 2nd ed. New York: John Wiley; 1997.
62. Mathews A, MacLeod C. Cognitive approaches to emotion and emotional disorders. Annu Rev Psychol 1994;45:25-50.
63. Dalgleish T, Watts FN. Biases of attention and memory in disorders of anxiety and depression. Clin Psychol Rev 1990;10:589-604.
64. Lusher J, Chandler C, Ball D. Alcohol dependence and the alcohol Stroop paradigm: evidence and issues. Drug Alcohol Depen 2004;75:225-231.
65. Taylor LM, Espie CA, White CA. Attentional bias in people with acute versus persistent insomnia secondary to cancer. Behav Sleep Med 2003;1:200-212.
66. Jones BT, Macphee LM, Broomfield NM, et al. Sleep-related attentional bias in good, moderate, and poor (primary insomnia) sleepers. J Abnorm Psychol 2005;114:249-258.
67. MacMahon KMA, Broomfield NM, et al. Attention bias for sleep-related stimuli in primary insomnia and delayed sleep phase syndrome using the dot-prob task. Sleep 2006;29(11):1420-1427.
68. Woods H, Marchetti LM, Biello SM, Espie CA. The clock as a focus of selective attention in those with primary insomnia: an experimental study using a modified Posner paradigm. Behav Res Ther 2009;47:231-236.
69. Spiegelhalder K, Espie C, Riemann D. Is sleep-related attentional bias due to sleepiness or sleeplessness? Cognition & Emotion 2009;23:541-550.
70. Broomfield NM, Espie CA. Toward a valid, reliable measure of sleep effort. J Sleep Res 2005;14(4):401-407.
71. Heath AC, Kendler KS, Eaves LJ, Martin NG. Evidence for genetic influences on sleep disturbance and sleep pattern in twins. Sleep 1990;13:318-335.
72. Watson NF, Goldberg J, Arguelles L, Buchwald D. Genetic and environmental influences on insomnia, daytime sleepiness, and obesity in twins. Sleep 2006;29:645-649.
73. Drake CL, Scofield H, Roth T. Vulnerability to insomnia: the role of familial aggregation. Sleep Med 2008;9:297-302.
74. Beaulieu-Bonneau S, LeBlanc M, Merette C, et al. Family history of insomnia in a population-based sample. Sleep 2007;30:1739-1745.
75. Greenspan RJ. The flexible genome. Nat Rev Genet 2001;2:383-387.
76. Dierick HA, Greenspan RJ. Molecular analysis of flies selected for aggressive behavior. Nat Genet 2006;38:1023-1031.
77. Andretic R, Shaw PJ. Essentials of sleep recordings in *Drosophila*: moving beyond sleep time. Methods Enzymol 2005;393:759-772.
78. Le Bourg E, Buecher C. Learned suppression of photopositive tendencies in *Drosophila melanogaster*. Anim Learn Behav 2002;30:330-341.
79. Seugnet L, Suzuki Y, Vine L, et al. D1 receptor activation in the mushroom bodies rescues sleep-loss-induced learning impairments in *Drosophila*. Curr Biol 2008;18:1110-1117.
80. Tamakoshi A, Ohno Y. Self-reported sleep duration as a predictor of all-cause mortality: results from the JACC study, Japan. Sleep 2004;27:51-54.
81. Granger DA, Kivlighan KT, el Sheikh M, et al. Salivary alpha-amylase in biobehavioral research: recent developments and applications. Ann N Y Acad Sci 2007;1098:122-144.
82. Nater UM, La Marca R, Florin L, et al. Stress-induced changes in human salivary alpha-amylase activity—associations with adrenergic activity. Psychoneuroendocrinology 2006;31:49-58.
83. Dantzer R, Kalin N. Salivary biomarkers of stress: cortisol and alpha-amylase. Psychoneuroendocrinology 2009;34:1.
84. DeCaro JA. Methodological considerations in the use of salivary alpha-amylase as a stress marker in field research. Am J Hum Biol 2008;20:617-619.
85. Robinson GE, Grozinger CM, Whitfield CW. Sociogenomics: social life in molecular terms. Nat Rev Genet 2005;6:257-270.
86. Edwards AC, Rollmann SM, Morgan TJ, Mackay TF. Quantitative genomics of aggressive behavior in *Drosophila melanogaster*. PLoS Genet 2006;2:e154.
87. Sorensen JG, Nielsen MM, Loeschcke V. Gene expression profile analysis of *Drosophila melanogaster* selected for resistance to environmental stressors. J Evol Biol 2007;20:1624-1636.
88. Spiegel K, Leproult R, Van Cauter E. Impact of sleep debt on metabolic and endocrine function. Lancet 1999;354:1435-1439.
89. Van Dongen HP, Maislin G, Mullington JM, Dinges DF. The cumulative cost of additional wakefulness: dose-response effects on neurobehavioral functions and sleep physiology from chronic sleep restriction and total sleep deprivation. Sleep 2003;26:117-126.
90. Rechtschaffen A, Bergmann BM, Everson CA, et al. Sleep deprivation in the rat: X. Integration and discussion of the findings. Sleep 1989;12:68-87.

91. Shaw PJ, Tononi G, Greenspan RJ, Robinson DF. Stress response genes protect against lethal effects of sleep deprivation in *Drosophila*. Nature 2002;417:287-291.
92. McGinty D, Szymusiak R. The sleep–wake switch: a neuronal alarm clock. Nat Med 2000;6:510-511.
93. Saper CB, Chou TC, Scammell TE. The sleep switch: hypothalamic control of sleep and wakefulness. Trends Neurosci 2001;24: 726-731.
94. Perlis M, Gehrman P, Pigeon WR, Findley J, et al. Neurobiologic mechanisms in chronic insomnia. Sleep Medicine Clinics 2009;4(4): 549-558.
95. Richardson GS. Human physiological models of insomnia. Sleep Medicine 2007;8:S9-S14.
96. Merica H, Fortune RD. A neuronal transition probability model for the evolution of power in the sigma and delta frequency bands of sleep EEG. Physiol Behav 1997;62:585-589.
97. Perlis ML, Merica H, Smith MT, Giles DE. Beta EEG activity and insomnia. Sleep Med Rev 2001;5:365-376.

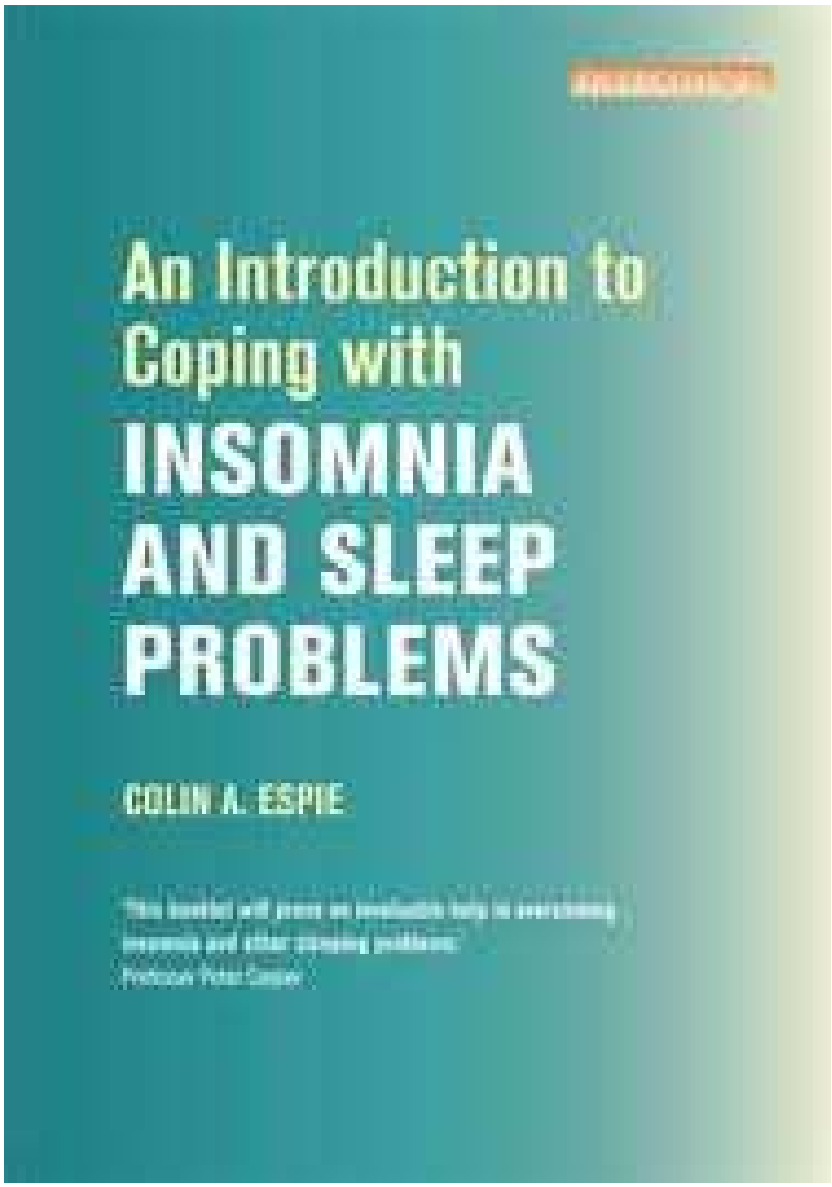
AUTHOR QUERY FORM

Dear Author

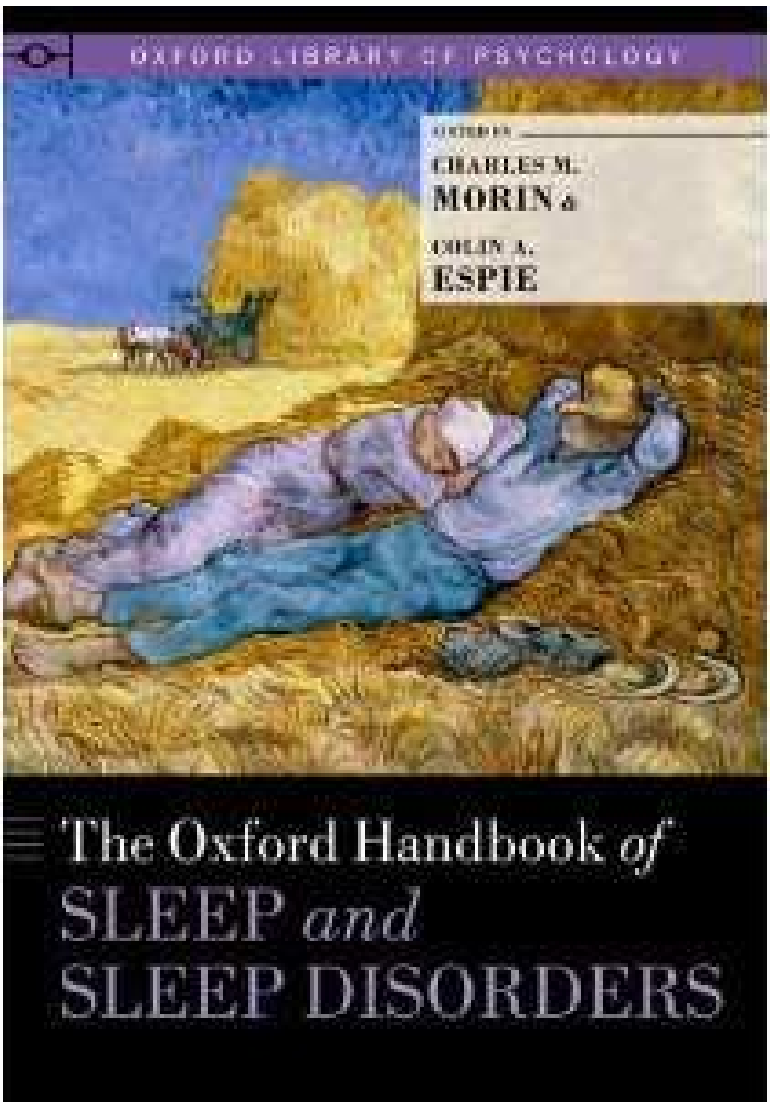
During the preparation of your manuscript for publication, the questions listed below have arisen. Please attend to these matters and return this form with your proof.
Many thanks for your assistance.

Query References	Query	Remarks
1	Au: please define CS.	

Espie, C.A. (2011) ***Introduction to Coping with Insomnia & Sleep Problems*****. Constable & Robinson Ltd., London [32 pages] [ISBN978-1-84901-620-9]**



Morin, C.M. & Espie, C.A. (2012) The Oxford Handbook of Sleep and Sleep Disorders (Oxford Library of Psychology) Oxford University Press, USA [ISBN-10: 019537620X]



www.sleepio.com