Research Report TM/03/01
December 2003

Impact assessment of the mortality effects of longer-term exposure to air pollution: exploring cause-specific mortality and susceptibility

BG Miller

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In previous work, we have developed the use of actuarial life-table methods, using PC spreadsheets, for predicting the impacts of changes in pollution levels on long-term patterns of mortality. This approach is flexible, and allows the simulation of impacts that affect only some causes of death; to date, we have focused on effects on the group of non-malignant cardio-respiratory causes as well as on all-cause mortality.

Because recent work has suggested that risks of air pollution may be greater for cardiovascular than for respiratory causes, we present and compare new predictions for cardiovascular impacts. In addition, we have for the first time modelled differences in mortality risk in different notional strata of the population, to represent differences in individual frailty or susceptibility to pollution effects.

Previous results showed that the impact of 1.8% reduction in cardio-respiratory risk was of the order of 10% smaller than that of a 1% reduction in the all-cause effect, and our results show that a purely cardiovascular 2% reduction produces an impact almost 30% smaller than the all-cause.

Similar results were obtained when the population was stratified to simulate a frailty distribution; however, when the sensitivity to the effects of pollution was allowed to differ by stratum, with the sensitivity increasing with increasing frailty, then the differences across cause groups were much reduced.

These results are interpreted as being due to the different age distributions for different causes of death, and particularly to the tendency for cardiovascular deaths to be predominantly in older persons, which limits their cumulative effects on life expectancy.

The revised spreadsheets offer great flexibility for further predictions under complex assumptions.

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1 INTRODUCTION

Quantitative assessment of the health impacts of air pollution, and of the associated costs, has attained considerable importance in recent years. This is a discipline whose principal feature is prediction into the unknown (unknowable) future, based on present knowledge of past effects. To do this well in the context of air pollution requires, amongst other things:

- knowledge or assumptions regarding the distributions and characteristics of the population for whom predictions are constructed;
- reliable estimates of the strength of relationships between air pollution and health effects;
- knowledge or assumptions regarding future changes in pollution levels that would affect the target population;
- knowledge or assumptions regarding the patterns of impacts that the changes would produce;
- a consistent methodology for calculating and summarising the predicted effects of these impacts, given all the above assumptions (and, if possible, one that can be implemented easily).

To date, reliable information about strength of relationships for long-term health effects has been available only from a limited number of US studies. Extensive analyses of mortality data from these studies, compared across cities, have pointed to a relationship with levels of particulate air pollution, both for mortality generally and particularly for causes of death classified as cardiovascular or (perhaps) respiratory (Krewski *et al*, 2000). Coefficients from these analyses have been widely discussed, and have been used in risk assessments whose aim was to quantify total predicted impacts of reductions in particle pollution.

Some results for all-cause mortality were reported as part of a DoH-funded project, with a final report by Hurley *et al* (2000). The methodology for quantitative predictions described in that report was based on actuarial life-table methods, adapted for health impact assessment at the IOM. More recent developments at the IOM, also funded by DoH, included catering for cause-specific impacts, triggered by the epidemiological observation that, in the above studies, not all causes of death related to air pollution; and the implementation of the methodology for calculation through standard spreadsheet techniques (Miller and Armstrong, 2001; Miller and Hurley, 2003).

We describe here some extensions to previous work. The initial impact assessments for cause-specific impacts looked only at the cardio-respiratory group of causes, whereas published results (Krewski *et al*, 2000) suggest that cardiovascular causes show more evidence of a relationship with pollution than do respiratory causes. We have therefore created predictions for the impact of changes in the cardiovascular hazard rates alone. In addition, we have for the first time considered and modelled differences in mortality risk of separate strata within age- and sex-specific subcohorts.

2. OBJECTIVE

The work reported here had the following objectives:

1. to assess the sensitivity of impact predictions to assumptions on cause: specifically, to run predictions similar to those already carried out, but based on effects only on cardiovascular mortality hazard rates;

2. to examine the effects on predictions of simulating variation in individual risks (susceptibility) and in the strength of effects

3 METHODS

3.1 INPUT DATA

Mortality hazards for baseline scenarios were based on published age-, sex- and cause-specific rates for England and Wales and for Scotland, for the year 1999. As before, these were summarised into six cause-groups, representing lung cancers, other cancers, cardiovascular causes, non-malignant respiratory causes, accidental causes, and other causes.

Figure 1 shows the age- and sex-specific average mortality hazard rates for cardiovascular causes and for non-malignant respiratory causes, for ages 30 and above. The last point on each curve is for ages 90+. The plots for the Scottish data are shown in grey, and show somewhat greater variation, because of the smaller population size and numbers of deaths. The graph shows clearly the higher mortality rates in men, and that the gap is much greater for the cardiovascular causes. However, this gap decreases with increasing age. It is clear that there is higher Scottish mortality from cardiovascular causes, for both sexes. For respiratory causes, it appears that the mortality in Scotland is slightly higher under the age of about 70, but more similar to England and Wales at higher ages. Rates for the other causes are not shown.

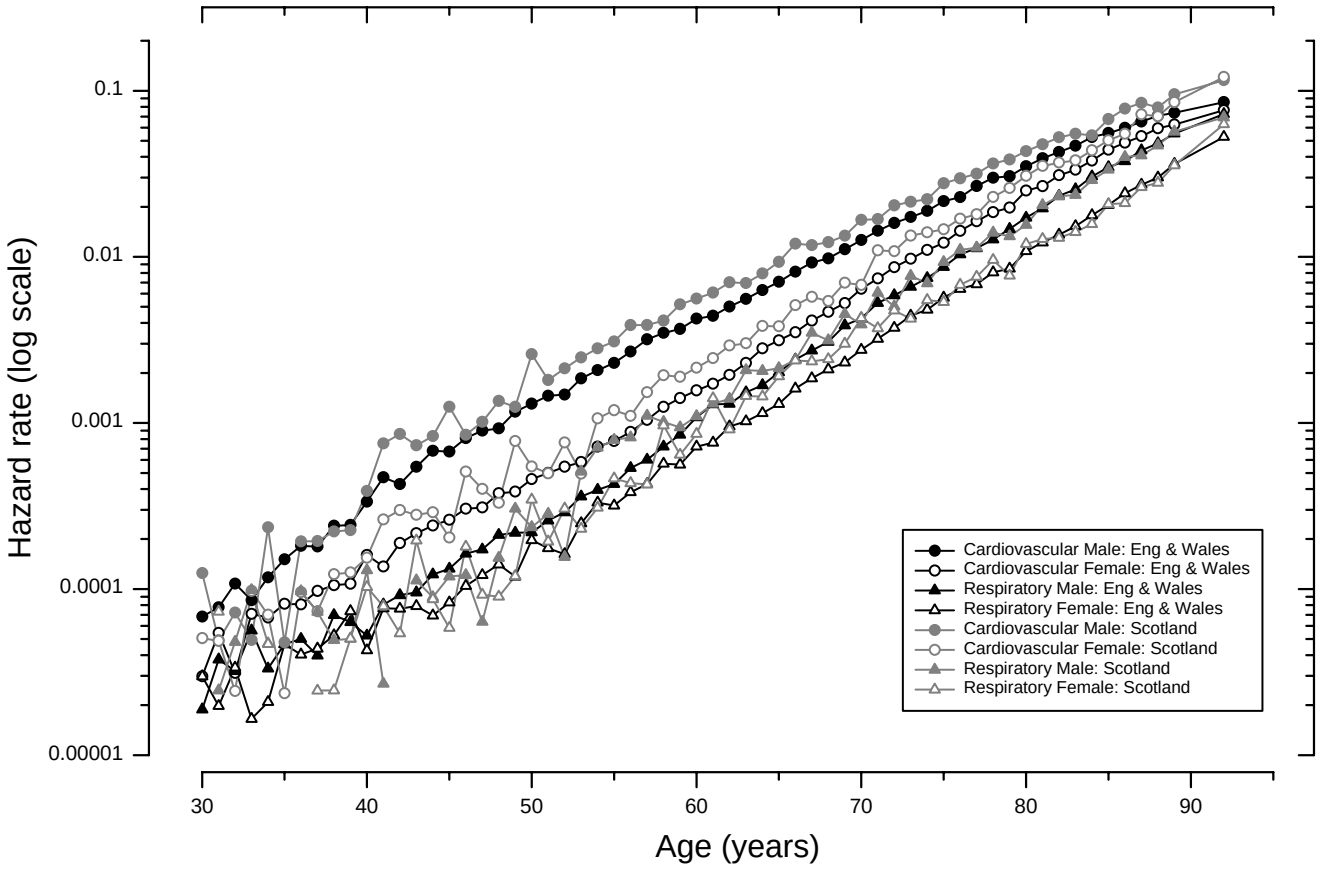

Figure 2 Age-specific mortality hazard rates (1999) by sex and country for cardiovascular and non-malignant respiratory causes for ages 30 and ove

3.2 CARDIOVASCULAR MORTALITY

We have previously described in detail how life table methods can be used to make quantitative impact assessments, by comparing the future patterns of expected life-years within chosen combinations of age cohorts. A series of developments to the methodology, which uses simple arithmetic procedures, have been implemented using the standard PC spreadsheet package MS Excel. We have previously described how differential effects on specific causes of death may be incorporated (Miller and Hurley, 2003), and have obtained quantitative estimates from assumptions of change only in the cardio-respiratory group of causes (Miller and Armstrong, 2001). The set-up for these calculations included separate baseline mortality hazard rates for the two major cause-groups of cardiovascular and non-malignant respiratory causes, and in the above work the hazards for both were subject to the same impact factor. It was therefore relatively straightforward to rerun these assessments for an impact affecting the cardiovascular rates only.

The calculations reported by Miller and Armstrong (2001) were designed to compare the effect of assuming an impact on all-cause mortality rates with that of an effect only on cardio-respiratory vascular mortality. The impact coefficients were derived from analyses of the American Cancer Society cohort study (Krewski *et al*, 2002), which had shown higher impact coefficients for cardio-vascular and cardio-respiratory causes than for all-cause mortality, suggesting strongly that these causes were more susceptible to pollution effects than the remainder of the principal cause groups. It was expected that differences in predicted impacts might arise if the age-specific pattern of rates differed by cause.

On the basis of published impact coefficients, Miller and Armstrong (2001) compared a 1% change in all-cause mortality with a 1.8% change in cardio-respiratory mortality. Details of analyses of cardiovascular mortality risks from the data of the American Cancer Society are in Table 20 of the HEI Special Report on the reanalysis, Part II (Krewski *et al*, 2000). These show that the excess risk of cardiovascular disease associated with the range of particulate exposures shown between the US cities involved was 36%, while that for all causes was 18%. Since these are in the ratio 2:1, we have compared the previous results with a 2% decrease in cardiovascular mortality rates. (Since the 36% difference across cities was associated with a range of 24.5 $\mu g.m^{-3}$ in $PM_{2.5}$ concentrations, a 2% change in cardiovascular mortality - or a 1% change in all-cause mortality - corresponds to a reduction in $PM_{2.5}$ of 1.4 $\mu g.m^{-3}$.)

3.3 VARIATION IN INDIVIDUAL RISKS

It is plausible (though very difficult to demonstrate) that any human age cohort comprises individuals with different, possible widely different, risks of mortality. In origin, those differences may be genetic, or induced by disease or accident, or by lifestyle; most are unidentified by present medical knowledge (else we would be better at predicting individual longevity). The end result, however, is that mortality hazards even for single-year age cohorts are almost certainly an average or composite over a wide but unquantified range of susceptibilities.

While we do not have any reliable markers of individual risk (or susceptibility or frailty), nevertheless we may, for the purposes of impact assessment, make and state our assumptions about what range of differences might underlie our averages. This could be expressed as some statistical distribution; given that it is to be used as input to an arithmetic procedure, it is more straightforward to imagine a stratification of the target population. It is then necessary to specify

- what proportions of the population lie in various strata, and
- how their mortality risks differ.

In consultation with the Project Officer, it was agreed that, for the first set of investigations, it was sufficient to construct a three-way stratification, to represent broad distinctions between a small sub-group of people who are at extreme risk (because they are very susceptible or already ill), a sub-group of those at somewhat increased risk, and the remainder of the population. Table 3.1 shows how differential baseline mortality hazards can be organised by creating factors to manipulate the hazard rates for particular causes, in this case cardiovascular.

This table has the structure needed to create differential factors, but the values it contains are just one of very many possible combinations. The values used split the population notionally into the three subgroups, assumed to differ in their baseline risk of cardiovascular mortality: susceptible, increased risk, and normal, in the percentage ratios 5:10:85. For all the causes of death except cardiovascular, the factors in the table are 1, implying that the subgroups all share the same hazard rate for each of these causes. For the cardiovascular, however, we have factors implying that the rates for the three sub-groups are in the ratios 10:2:1. The factors are scaled so that their average weighted by the population percentage fractions is 1 (= 0.05×6.4516 + 0.1×1.2903 + 0.85×0.6452). Thus the groups combine to have the correct underlying baseline cardiovascular hazard rate. Both the proportions and the differential hazard factors are arbitrary, and can be varied to meet different population assumptions.

Table 3.1 Example of baseline population and hazard stratification factors

Subgroup type	Proportion %	Differential baseline hazard factor by cause					
		Lung Cancer	Other Cancer	Cardio-vascular	Respiratory	Accidents	Other
Susceptible	5	1.0000	1.0000	6.4516	1.0000	1.0000	1.0000
Increased risk	10	1.0000	1.0000	1.2903	1.0000	1.0000	1.0000
Normal	85	1.0000	1.0000	0.6452	1.0000	1.0000	1.0000
Total	100	1.0000	1.0000	1.0000	1.0000	1.0000	1.0000

Table 3.2 Example of stratified hazard impact factors; impacts on cardiovascular causes only, constant across subgroups

Subgroup type	Proportion %	Differential impact factor by cause					
		Lung Cancer	Other Cancer	Cardio-vascular	Respiratory	Accidents	Other
Susceptible	5	1.0000	1.0000	0.9800	1.0000	1.0000	1.0000
Increased risk	10	1.0000	1.0000	0.9800	1.0000	1.0000	1.0000
Normal	85	1.0000	1.0000	0.9800	1.0000	1.0000	1.0000
Total	100	1.0000	1.0000	0.9800	1.0000	1.0000	1.0000

As in previous work, it is necessary to specify the impact factors that will produce altered hazard rates for comparison with the baseline.

With the stratification organised as above, the impact factors can differ across subgroups as well as cause group. An example is shown in Table 3.2. Thus, Table 3.1 represents a spectrum of *baseline* frailty in the population whereas Table 3.2 represents the *change* to be

applied to the baseline population e.g. as a result of air pollution reductions. Table 3.2 represents a 2% reduction in cardiovascular mortality rates in all three subgroups.

Table 3.3 shows how the factors can represent differential impacts by subgroup. This example has impacts on the cardio-respiratory groups of causes in the ratios 10:2:1, scaled to produce a weighted average of a 1.8% decrease in the hazards for these causes (rather than 2% for cardiovascular causes alone). In this example, a greater reduction in hazard (11.61%) is shown for the susceptible group compared with the normal group (1.16%). This is analogous to assuming that the strata differ not only in their baseline frailty but also in their sensitivity to air pollution. If the frailest group is also the most sensitive to air pollution the gains from a reduction in air pollution will be greatest in this group.

Table 3.3 Example of stratified hazard impact factors; impacts on cardio-respiratory cause groups, differing across subgroups

Subgroup type	Proportion %	Differential impact factor by cause					
		Lung Cancer	Other Cancer	Cardio-vascular	Respiratory	Accidents	Other
Susceptible	5	1.0000	1.0000	0.8839	0.8839	1.0000	1.0000
Increased risk	10	1.0000	1.0000	0.9768	0.9768	1.0000	1.0000
Normal	85	1.0000	1.0000	0.9884	0.9884	1.0000	1.0000
Total	100	1.0000	1.0000	0.9820	0.9820	1.0000	1.0000

The spreadsheets used for previous calculations were adapted for the stratified calculations, following some tidying up of their structure, and a triplicate set of spreadsheet files was set up to perform life-table calculations from the baseline hazard rates in the manner previously described. These took as inputs a spreadsheet of the 1999 age- and cause-specific rates for England and Wales and a separate sheet containing the stratification factors, from which were created stratum-specific sub-cohort numbers and mortality rates.

A second set of triplicate spreadsheet files was set up to perform life-table calculations for scenarios including impacts on the mortality rates. These took as input the stratified baseline hazards as above, plus a further sheet containing hazard impact factors.

The final spreadsheets were set up to recombine the results from the strata, and to compare the results from the impacted scenarios with those from the baseline. The final summary sheet was made self-documenting, by including a listing of the input tables of stratification and hazard impact factors. An example of the output is shown below, as Table 3.4.

As before, separate calculations were performed for males and females. For the present work, we have assumed the same stratification and impact factors for both sexes. As before, separate estimates have been made for England and Wales and for Scotland.

Table 3.4 Example of output spreadsheet summarising impacts for males and females, and recording input table of baseline stratification factors and stratified impact factors.

	Scotland		**Males**	**Females**	**Total**			
Outputs	Start popn 2005		2,502,949	2,631,483	5,134,432			
	Years gained		191,076	178,736	369,813			
	Years per 100k popn		7634	6792	7203			
Inputs	**Population Fractions:**							
	Subpopn type	Propn %	Differential baseline hazard factor by cause					
			Lung Ca	Other Ca	Cardio	Respty	Accidents	Other
	Susceptible	5	1.0000	1.0000	6.4516	1.0000	1.0000	1.0000
	Increased risk	10	1.0000	1.0000	1.2903	1.0000	1.0000	1.0000
	Normal	85	1.0000	1.0000	0.6452	1.0000	1.0000	1.0000
	Total	100	1.0000	1.0000	1.0000	1.0000	1.0000	1.0000
	Impact Factors:							
	Subpopn type	Propn %	Differential impact factor by cause					
			Lung Ca	Other Ca	Cardio	Respty	Accidents	Other
	Susceptible	5	1.0000	1.0000	0.8711	1.0000	1.0000	1.0000
	Increased risk	10	1.0000	1.0000	0.9742	1.0000	1.0000	1.0000
	Normal	85	1.0000	1.0000	0.9871	1.0000	1.0000	1.0000
	Total	0	1.0000	1.0000	0.9800	1.0000	1.0000	1.0000

4 RESULTS

4.1 CARDIOVASCULAR EFFECTS

Table 4.1 summarises results of predictions on the effects of various impacts, for the population predicted to be alive at the start of the year 2005. We show the total size of these start populations broken down by sex and by country, and compare the life years gained by reducing the all-cause hazards for ages 30 and over by 1% from the start of 2005 onwards. We compare this with the effects of reducing cardio-respiratory hazards by 1.8%, and cardiovascular hazards only by 2%, over the same age range. As in previous work, the predicted gains from a 1% reduction in the all-cause hazards are similar in males and females, giving a total gain of 4.8 million life-years in England and Wales, and 0.46 million in Scotland. Scaled to the population sizes, the results are still similar across the sexes, and across countries.

Table 4.1 Results of simulations, comparing predictions of impact from 1% reduction in all-cause hazards with 1.8% reduction in cardio-respiratory hazards and 2% reduction on cardiovascular hazards alone. Reductions in hazard take place from start of 2005. Impacts summarised for populations alive at start 2005.

Country	Impact	Value tabulated	Males	Females	Total
England & Wales		2005 start population (m)	26.42	26.93	53.35
	All causes	Life years gained (m)	2.387	2.396	4.784
		Life years gained per 100,000	9036	8899	8967
	Cardio-respiratory only	Life years gained (m)	2.227	2.188	4.415
		Life years gained per 100,000	8429	8123	8275
	Cardiovascular only	Life years gained (m)	1.756	1.634	3.390
		Life years gained per 100,000	6646	6067	6354
Scotland		2005 start population (m)	2.498	2.626	5.124
	All causes	Life years gained (m)	0.230	0.227	0.457
		Life years gained per 100,000	9182	8584	8875
	Cardio-respiratory only	Life years gained (m)	0.204	0.201	0.404
		Life years gained per 100,000	8153	7661	7901
	Cardiovascular only	Life years gained (m)	0.172	0.159	0.330
		Life years gained per 100,000	6869	6045	6447

The results from simulations with a 1.8% reduction in hazard rates for the cardio-respiratory group of causes show similar effects, but in all cases somewhat smaller total effects. The reduction in effect is similar across the sexes, but differs across countries; while the cause-specific impact is 7.7% lower than the all-cause for England and Wales, the difference for Scotland is 11.6%.

The impacts for a 2% reduction on cardiovascular mortality are even smaller. The total cause-specific impact was estimated at 29% lower than the all-cause effect in England and Wales, and 28% lower in Scotland.

Although the impact coefficients have been chosen to be comparable on the basis of the results from the cohort studies, it is clear that the predicted impacts differ considerably. This is precisely because the age distributions of the hazard rates for the different causes differ, and thus the impact of a proportional change will differ by cause. In particular, the cardiovascular and respiratory rates affect the older age groups. Thus, the impact of a change in a the hazards for, say, cardiovascular deaths will have a smaller impact on overall hazards at younger ages. Thus the shape of the survival curves for baseline and impacted cohorts will differ little at lower ages, and diverge significantly only at higher ages.

We have observed in earlier simulations that changes in rates in the young matter little, because the baseline rates are so low; and that changes in the old also have less impact than in the middle years of life, because the population sizes are smaller. The observations in the present simulations are consistent with this.

4.2 SIMULATING SUSCEPTIBILITY BY STRATIFICATION

Table 4.2 contains the results of predictions for a 1% reduction in all-cause hazards, along with 1.8% and 2% reductions in cardio-respiratory causes and in cardiovascular causes only respectively, for comparison with Table 4.1. The new predictions have been calculated for three separate strata within each age- and sex-specific subcohort, with numbers in the ratio 5:10:85. As detailed in Table 4.2a, these strata are assumed equal in their baseline hazards for all causes except cardiovascular, where the hazards are in the ratio 10:2:1.

It may be noticed that the predicted start populations in Table 4.2 differ slightly from those in Table 4.1. This is because for Table 4.2 the start populations are derived separately in each stratum by life-table calculations between 1999 and 2005 and then recombined. The small difference in start populations does not affect the interpretation of the observed differences.

Comparison of Table 4.2 with 4.1 shows that the predicted effects of a 1% reduction in all cause mortality were always slightly higher for the stratified simulations; but that the decreased impacts observed for the cause-specific hazard changes were even more pronounced in the stratified case. These findings applied to both countries and to both sexes.

Table 4.3 shows predictions for the same strata, but with the additional stratification of the hazard factors, as detailed in Table 4.3a. The all-cause impacts are slightly smaller than in Tables 4.1 and 4.2. However, there is now much less of a difference between the effects of an all-cause and cause-specific impacts. In England and Wales, the cardio-respiratory impact is almost the same as the all-cause, and in Scotland it is reduced by only about 3%; in both countries the impact of the stratified cardiovascular effect is much greater than in the unstratified case, showing only a 15-17% reduction from the all-cause. For ease of comparison, the results from Tables 4.1 to 4.3 are shown together in Table 4.4

The results in Table 4.2 are consistent with what we know about impacts from earlier simulations representing simpler comparisons. The stratified population we have set up has a majority with rather lower than average cardiovascular mortality rates. They will therefore

Table 4.2 Results of simulations comparing predictions of impact from 1% reduction in all-cause hazards and 1.8% and 2% reductions in cardio-respiratory hazards. Predictions made separately for strata differing in baseline cardiovascular hazard, defined in table below.

Country	Impact	Value tabulated	Males	Females	Total
England & Wales		2005 start population (m)	26.46	26.98	53.44
	All causes	Life years gained (m)	2.431	2.466	4.897
		Life years gained per 100,000	9186	9142	9164
	Cardio-respiratory only	Life years gained (m)	2.099	2.098	4.197
		Life years gained per 100,000	7934	7775	7854
	Cardiovascular only	Life years gained (m)	1.497	1.408	2.905
		Life years gained per 100,000	5659	5217	5436
Scotland		2005 start population (m)	2.502	2.631	5.134
	All causes	Life years gained (m)	0.234	0.234	0.468
		Life years gained per 100,000	9355	8877	9110
	Cardio-respiratory only	Life years gained (m)	0.190	0.192	0.383
		Life years gained per 100,000	7603	7308	7451
	Cardiovascular only	Life years gained (m)	0.147	0.138	0.285
		Life years gained per 100,000	5877	5254	5557

Table 4.2a Population and hazard stratification factors for Table 4.2

Subgroup type	Proportion %	Differential baseline hazard factor by cause					
		Lung Cancer	Other Cancer	Cardio-vascular	Respiratory	Accidents	Other
Susceptible	5	1.0000	1.0000	6.4516	1.0000	1.0000	1.0000
Increased risk	10	1.0000	1.0000	1.2903	1.0000	1.0000	1.0000
Normal	85	1.0000	1.0000	0.6452	1.0000	1.0000	1.0000
Total population		1.0000	1.0000	1.0000	1.0000	1.0000	1.0000

experience a proportionally larger part of their mortality due to other causes with lower ages at death, and an all-cause impact here has greater effects on total YOLL than at later ages. The effects of a cardio-respiratory or cardio-specific impact remain lower, for the same reasons as before, but the difference is exaggerated by the stratification. In Table 4.3, the additional stratification of the impact factors restores reverses this reduction, to the extent that the effects of a cardio- or cardio-pulmonary impact are of the same order of magnitude as the equivalent all-cause. However, all of these variations are relatively small, and in fact all are of the same order of magnitude.

Table 4.3 Results of simulations, comparing predictions of impact from reduction in hazards differing across strata. Strata as for Table 4.2, hazard impact factors as in table below.

Country	Impact	Value tabulated	Males	Females	Total
England & Wales	All causes	2005 start population (m)	26.46	26.98	53.44
		Life years gained (m)	2.296	2.298	4.595
		Life years gained per 100,000	8678	8519	8598
	Cardio-respiratory only	Life years gained (m)	2.350	2.268	4.618
		Life years gained per 100,000	8882	8407	8642
	Cardiovascular only	Life years gained (m)	2.000	1.830	3.829
		Life years gained per 100,000	7551	6785	7164
Scotland	All causes	2005 start population (m)	2.502	2.631	5.134
		Life years gained (m)	0.220	0.219	0.439
		Life years gained per 100,000	8779	8336	8552
	Cardio-respiratory only	Life years gained (m)	0.214	0.212	0.426
		Life years gained per 100,000	8561	8051	8299
	Cardiovascular only	Life years gained (m)	0.191	0.178	0.369
		Life years gained per 100,000	7634	6792	7203

Table 4.3a Stratified impact factors for Table 4.3

Subgroup type	Proportion %	Differential impact factor by cause					
		Lung Cancer	Other Cancer	Cardio-vascular	Respiratory	Accidents	Other
All causes							
Susceptible	5	0.9355	0.9355	0.9355	0.9355	0.9355	0.9355
Increased risk	10	0.9871	0.9871	0.9871	0.9871	0.9871	0.9871
Normal	85	0.9935	0.9935	0.9935	0.9935	0.9935	0.9935
Total population		0.9900	0.9900	0.9900	0.9900	0.9900	0.9900
Cardio-respiratory only							
Susceptible	5	1.0000	1.0000	0.8839	0.8839	1.0000	1.0000
Increased risk	10	1.0000	1.0000	0.9768	0.9768	1.0000	1.0000
Normal	85	1.0000	1.0000	0.9884	0.9884	1.0000	1.0000
Total population		1.0000	1.0000	0.9820	0.9820	1.0000	1.0000
Cardiovascular only							
Susceptible	5	1.0000	1.0000	0.8711	1.0000	1.0000	1.0000
Increased risk	10	1.0000	1.0000	0.9742	1.0000	1.0000	1.0000
Normal	85	1.0000	1.0000	0.9871	1.0000	1.0000	1.0000
Total population		1.0000	1.0000	0.9800	1.0000	1.0000	1.0000

Table 4.4 Summary of life years gained (m) across total population of England and Wales, estimated from different sets of assumptions and different impacts on mortality.

Data from Tables 4.1 to 4.3

Assumptions	Mortality cause affected		
	All Causes	**Cardio-respiratory**	**Cardio-vascular**
No stratification (Table 4.1)	4.784	4.415	3.39
Stratified population (Table 4.2)	4.897	4.197	2.905
Sensitivity variable by strata (Table 4.3)	4.595	4.618	3.829

5 ACKNOWLEDGMENTS

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Our first major research programme began in the 1950s, on respiratory health problems in the coal mining industry. Major themes were quantification of airborne dust concentrations in different jobs, characterisation of types and constituents of the dusts, measurement of health effects, relationships between exposure and disease, and proposals for prevention. This research became an international benchmark for epidemiological studies of occupational health, and was the primary influence on dust standards in mines in the UK, US and other countries.

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Our current work spans many other industries including asbestos, MMMF, pesticides, chemicals, energy, telecoms, metals, textiles, construction, agriculture as well as the environment. While diseases of the respiratory tract remain a major interest, our scope now extends to many other health outcomes such as mortality, cardiovascular effects, cancer, back pain, upper-limb disorders, hearing loss, skin diseases, thermal stress and psychological stress. Related work includes the development and application of measurement and control systems, mathematical models and survey methods.

Who we work for

Our work in these areas is conducted for a wide range of organisations in the UK, the EU, and the US, including Government departments, international agencies, industry associations, local authorities, charitable organisations, and industrial and commercial companies. The IOM is a World Heath Organisation (WHO) collaborating centre and is an approved institute of the Universities of Edinburgh and Aberdeen, enjoying collaborative research links with NIOSH, IARC, and many other institutes throughout the world.

Publication

We believe that our research findings should be publicly available and subject to the scrutiny of the international scientific community. We publish our findings in the peer reviewed scientific literature and through our own series of Research Reports.

Contact

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