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Explaining the Widening Education Gap in Mortality among U.S. White Women

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Abstract

Over the past half century the gap in mortality across education levels has grown in the United States, and since the mid-1980s, the growth has been especially pronounced among white women. The reasons for the growth among white women are unclear. We investigated three explanations—social-psychological factors, economic circumstances, and health behaviors—for the widening education gap in mortality from 1997 to 2006 among white women aged 45 to 84 years using data from the National Health Interview Survey Linked Mortality File (N = 46,744; 4,053 deaths). Little support was found for social-psychological factors, but economic circumstances and health behaviors jointly explained the growing education gap in mortality to statistical nonsignificance. Employment and smoking were the most important individual components. Increasing high school graduation rates, reducing smoking prevalence, and designing work-family policies that help women find and maintain desirable employment may reduce mortality inequalities among women.

Keywords

education, gradient, health disparities, mortality, women's health

The inequality in mortality risk across education levels in the United States is well documented (e.g., Hummer and Lariscy 2011), and it has increased over the past half century (Crimmins and Saito 2001; Feldman et al. 1989; Lauderdale 2001; Pappas et al. 1993; Preston and Elo 1995; Rogot, Sorlie, and Johnson 1992). Since the mid-1980s, the inequality appears to have grown more among women than men (Cutler et al. 2011; Meara, Richards, and Cutler 2008; Montez et al. 2011). Among women, this recent growth has reflected declines in mortality among the higher educated alongside increases in mortality among the lower educated (Meara et al. 2008; Montez et al. 2011). The trends, which we describe below, have been particularly unfavorable among white women (Jemal et al. 2008; Meara et al. 2008; Montez et al. 2011).

The reasons for the widening mortality gap among white women are not fully understood, and only a few studies have investigated them. Those studies largely focused on trends in specific causes of death (Meara et al. 2008; Montez and Zajacova 2013) or in health-related behaviors (Cutler et al. 2011) across education levels. They generally concluded that diverging smoking patterns played an important role. For example, two causes of death for which smoking is a major risk factor, lung cancer and chronic lower respiratory disease (CLRD), explain one quarter to one half of the growing gap in all-cause mortality since the mid-1980s among white women aged 45 to 84 years (Meara et al. 2008; Montez and Zajacova 2013). Although those studies are informative, they do not provide a complete explanation.

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By focusing almost exclusively on education-specific trends in causes of death or health behaviors, prior studies have drawn attention to "downstream," behavioral explanations (e.g., smoking and lung cancer mortality, obesity and diabetes mortality). Thus, they have not revealed why diverging behavioral patterns emerged, nor have they addressed nonbehavioral explanations such as economic policy, labor market participation, and social integration that are further "upstream" in the causal chain and that may be linked to numerous causes of death.

A complete explanation of the growing gap requires identifying the "causes of causes" (Rose 2008:128). In other words, it requires searching for broader contextual explanations that lie further upstream in the causal chain. The best insights will then be gleaned by triangulating the findings regarding the contextual explanations with findings regarding education-specific trends in causes of death. The benefits of looking for the "causes of causes" are twofold. First, the closer we get to the root causes of the growing inequality, the better chance we have of reducing it. A fundamental-cause perspective impels us to search for explanations as far upstream as possible because eliminating a downstream mechanism may have limited benefit, as other downstream mechanisms will likely take its place (Link 2008). Second, this approach may also be powerful in identifying policy levers to stem the divergence because such levers can be aimed at the contextual factors such as employment and educational policies.

In this study, we provide new evidence about the reasons for the widening gap in all-cause mortality risk across education levels among white women. We investigate a range of factors that are well-known mechanisms linking education and mortality but have not been examined in this context: social-psychological factors, economic circumstances, and health behaviors. Our findings address the growing concern among scholars and policy makers about the widening educational divide in longevity. For instance, the Healthy People initiative has sought to reduce health disparities within the U.S. population, including disparities by socioeconomic status (U.S. Department of Health and Human Services 2000). We aim to identify mechanisms that may have the greatest leverage in achieving those goals.

BACKGROUND

Prior Research

Recent studies have found that since the mid-1980s, mortality risk among white women has slightly decreased among the college educated, remained fairly stable among women with a high school credential or some college education, and increased among women with 0 to 11 years of education; the latter group was primarily responsible for the growing gap (Jemal et al. 2008; Miech et al. 2011; Montez et al. 2011; Montez and Zajacova 2013). For example, from 1986 to 1994, the age-standardized death rate of white women aged 45 to 84 years with 0 to 11 years of education (2,400 deaths per 100,000 women, or .024) was 3.4 times larger than the death rate for college-educated women (.007). By 1995 to 1998, the death rate of the low-educated group was 4.3 times larger (.026/.006), and by 2003 to 2006 4.7 times larger (.028/.006), than the rate for the high-educated group (Montez and Zajacova 2013). Life expectancy has also diverged. Meara et al. (2008) estimated life expectancy at age 25 among low-educated (0–12 years) and high-educated (≥13 years) adults in 1990 and 2000. Life expectancy increased by 1 year among high-educated white women but decreased by nearly 1 year among their low-educated peers.

As we mentioned above, the handful of studies that have investigated the reasons for the widening mortality gap have focused on describing trends in causes of death. The causes identified as most important in each study vary somewhat depending on the age range examined. For example, among white women aged 45 to 84 years, lung cancer and CLRD explained one quarter to one half of the growing gap in all-cause mortality risk since the mid-1980s (Meara et al. 2008; Montez and Zajacova 2013). The mortality gap also increased for diabetes, cerebrovascular disease, and Alzheimer's disease among this age group (Montez and Zajacova 2013). Among younger women aged 25 to 64 years, deaths from accidents contributed the largest percentage to the widening gap during the mid- to late 1990s (Jemal et al. 2008). In a middle-aged sample of women aged 40 to 64 years, accidental poisoning, CLRD, and cancers of the trachea, lung, and bronchus were the main contributors between 1999 and 2007 (Miech et al. 2011).

We are aware of only one previous study that investigated some of the mechanisms that might explain the growing mortality gap. Using two U.S. population surveys, Cutler et al. (2011) examined the role of smoking and obesity in explaining the widening gap in mortality risk across education levels from the 1970s to 1990s among non-Hispanic whites aged 25 to 74 years. They found that smoking and obesity explained 10 percent to

40 percent of the increasing gap among women. However, they concluded that trends in the distribution of the behavioral risk factors were not the major explanation; instead, the mortality consequences of the behaviors became more severe.

Hypothesized Mechanisms

The association between education and mortality can strengthen for several reasons. First, the returns from education may increase such that specific mechanisms (e.g., smoking) become more strongly linked to education. This possibility is the focus of our study. In addition to the changing returns to education, new mechanisms linking education and mortality may emerge (e.g., the Internet and the "digital divide" are one possibility). Alternatively, the content of education may change in ways that make it more relevant for health. And finally, the composition of education groups may change. Specifically, as the average level of educational attainment rose steadily during the twentieth century, individuals who do not graduate from high school today may be more negatively select than in the past. Addressing compositional changes is beyond the scope of our study, but we comment on this possibility in the discussion section.

Part of the difficulty in identifying the mechanisms linking education and mortality risk (or any health outcome) is that they are multifarious at any point in time, they may change over time, and they may vary across demographic subgroups. Indeed, if we consider education as a fundamental cause (Link and Phelan 1995) of mortality disparities, then a search for mechanisms must be tempered by the recognition that "the persistence of the association over time and its generality across very different places suggests that no fixed set of intervening risk and protective factors can account for the connection" (Link et al. 2008:72). Thus, our task here is particularly challenging, and we expect to find relatively small effects of the mechanisms we examine. We examine the three main groups of mechanisms through which education has been consistently linked to health and mortality (Hummer and Lariscy 2011; Ross and Wu 1995): social-psychological factors, economic circumstances, and health behaviors.

Social-psychological factors. Higher educated adults tend to have more social-psychological resources, such as social ties (McPherson, Smith-Lovin, and Brashears 2006) and a sense of control (Mirowsky and Ross 2003), than less educated adults. For instance, higher educated adults are more likely to be married than their less educated peers, and this disparity has grown in recent decades among whites; no such growth has been observed among blacks (DiPrete and Buchmann 2006). This trend may have played a role in the widening mortality gap because marriage is the most salient social tie for many adults (Walen and Lachman 2000), and it is linked with lower mortality risk (Umberson 1992; Waite 1995). Furthermore, since the early 1970s, educational homogamy within marriage has increased, particularly at the tails of the education distribution (Schwartz and Mare 2005). The increasing homogamy may have exacerbated health disparities because better educated adults experience higher marital quality (Cherlin 1992), and the education of both spouses may influence each other's mortality (Montez et al. 2009).

Better educated individuals are less likely to experience marital, parental, and financial stress; traumatic events such as divorce, assault, and the death of a child (Lantz et al. 2005); and depression (Miech and Shanahan 2000). To the extent that the other types of mechanisms we examine have disproportionately increased exposure to these stressors and events among low educated women, they may have suffered even greater levels of psychological distress.

Economic circumstances. Education is associated with a higher likelihood of being employed, avoiding financial hardship, owning a home, and having employment-related health insurance (Ross and Wu 1995). These resources have become progressively more concentrated among higher educated groups as the U.S. labor market has bifurcated in recent decades. For example, employment rates have grown substantially more for women with higher education. From 1960 to 1990, the percentage of women 25 years of age and older employed full-time grew from just 12.0 percent to 14.7 percent among women without high school credentials, whereas it nearly doubled from 19.7 percent to 39.0 percent among women with at least 5 years of college (Spain and Bianchi 1996). The returns from education for labor participation and economic well-being have grown more among women than men (Blank and Shierholz 2006; DiPrete and Buchmann 2006).

Health behaviors. Better educated adults are more likely to exercise, not smoke, drink alcohol in moderation, and maintain a healthy body weight compared with less educated adults (Pampel, Krueger, and Denney 2010). They have

greater access to health-related information and more quickly integrate it into their lifestyles. For example, in 1954, when definitive studies linking cigarette smoking and cancer appeared in the media, 95.1 percent of college graduates claimed that they had heard the information, compared with 81.0 percent of adults with less than a high school credential (Link 2008). Although at that time, there was little difference in smoking prevalence by education, more educated adults adopted this information faster, so that by the 1990s, an educational gradient had emerged (Link 2008). Smoking trends across birth cohorts from 1908 to 1967 reveal that although smoking declined among adults with at least a high school credential, the prevalence among adults with 0 to 11 years of education remained stable among men but increased among women, especially white women (Escobedo and Peddicord 1996). In addition, between 1988-1994 and 2005-2008, educational disparities in obesity increased slightly among women but decreased among men (Ogden et al. 2010).

Aims of this Study

We examine the extent to which the three categories of mechanisms described above account for the widening education gap in all-cause mortality risk from 1997 to 2006 among non-Hispanic white women aged 45 to 84 years in the United States. We focus on white women because of their unique mortality trends across education levels during the past few decades. As described above, their mortality gap has grown markedly since the mid-1980s; moreover, life expectancy has reportedly declined among low-educated white women, in contrast to gains in life expectancy in the population overall. We also focus on white women because the reasons for the growing gap may differ by gender and race-ethnicity, given historical disparities in school quality, employment, immigration patterns, and family structure, for example. We examine the contribution of each category of mechanisms as well as individual mechanisms and relate our findings to what is currently known about the growing education gap in specific causes of death.

DATA AND METHODS

Data

Data for this study are from the public-use National Health Interview Survey (NHIS) Linked Mortality File (LMF), downloaded from the Minnesota Population Center (2012). The NHIS-LMF links adults in the 1986 through 2004 annual cross-sectional waves of the NHIS with death records in the National Death Index through December 31, 2006. The linkage is based mainly on a probabilistic matching algorithm, which correctly classifies the vital status of 98.5 percent of eligible survey records (National Center for Health Statistics 2009). In 1997, the NHIS began annually collecting data on the mechanisms of interest in this study. Thus, we use data from the 1997 to 2004 NHIS surveys and vital status information through 2006.

Educational Attainment and Time

Our analytic objective is to assess the extent to which the growing gap in mortality risk across education levels can be explained by the hypothesized mechanisms. This entails estimating an education-by-time interaction coefficient in our statistical models (described below) and quantifying how the size and statistical significance of the coefficient attenuate when the mechanisms are included in the models.

Educational attainment. We dichotomize education level (0 = 12 years or more, 1 = 0–11 years). This specification is based on prior research that has found that unfavorable mortality trends among white women with 0 to 11 years of education are the main contributor to the widening mortality gap since the mid-1980s (Montez and Zajacova 2013).[1] The percentage of non-Hispanic white women aged 45 to 84 years with 0 to 11 years of education was 15.7 percent during the first 4 survey years (1997–2000) and 12.3 percent during the latter 4 years (2001–2004). We refer to women with 0 to 11 years of education as "low educated" and other women as "high educated."

Time. Time indicates the year of exposure to the risk for death during the 1997 to 2006 follow-up. Prior studies using the NHIS-LMF have aggregated follow-up years to produce stable mortality

rates for race-gender-education subgroups (Montez et al. 2011; Montez and Zajacova 2013). In preliminary analyses, we estimated mortality rates across 1997 to 2006 using continuous and aggregated specifications of time and then determined which specification best reflected the annual rates. The aggregated term (0 = 1997–2001, 1 = 2002–2006) smoothed the annual variation and reflected the overall trend in the annual rates much better than did the continuous term. Thus, we use the aggregated (i.e., dichotomized) specification.

Mortality

The outcome is a dichotomous indicator of whether the individual died from any cause during the 1997 to 2006 follow-up. Of the 7,189 low-educated women in our sample, 1,161 (16.1 percent) died. Of the 39,555 high-educated women, 2,892 (7.3 percent) died.

Mechanisms

Social-psychological factors. We assess two components of social-psychological circumstances with strong ties to mortality: marriage and psychological distress. We include legal marital status (1 = married, 0 = unmarried) and spouse's education level. Other measures of social ties are not available in the NHIS. Spouse's education is defined as a three-level ordinal variable indicating 0 to 11 years of education (0), a high school credential or some college (1), or a bachelor's degree or higher (2) and used as a continuous predictor in the analyses. In preliminary analyses, we also examined a categorical measure of spouse's education. The findings were similar, so we chose the ordinal (continuous) measure for parsimony. Because unmarried women do not have data on spousal education, we use an internal moderator approach (Mirowsky 1999) and include marital status and the product of marital status and spouse's education.

We measure nonspecific psychological distress using the K6 Scale. The scale has strong psychometric properties and provides a standardized estimate of the prevalence and severity of mental illness in community-based populations (Kessler et al. 2002). It consists of responses to six questions about how often during the past 30 days the respondent felt so sad that nothing could cheer him or her up; nervous; restless or fidgety; hopeless; that everything was an effort; or worthless. Response categories range from 0 ("none") to 4 ("all the time"). For each respondent, we replaced missing values with the mean of their provided values and then summed the six responses for a total score between 0 and 24.

Economic circumstances. Key economic factors that shape mortality risk include employment, occupation, extreme deprivation (e.g., poverty), fluid resources (e.g., income), and accumulated resources (e.g., wealth, home ownership) (Krueger and Burgard 2011). Employment status during the previous week is dichotomized as employed (full-time or part-time) or not employed (unemployed or not in the labor force). In some analyses, we include an alternative specification that combines information about occupation among the employed. This specification comprises four dummy variables: not employed (omitted reference), white-collar (executive, administrative, and managerial or professional specialty), skilled (technical and related support, sales, administrative support, protective services, or the military), and manual (private household, service, farming, precision production, operators, transportation, or handlers and laborers). Extreme deprivation is measured by an indicator of whether the family income-to-poverty ratio was less than 1 during the previous year. In ancillary analyses, we assessed four categories of the ratio but found similar results. We measure accumulated material resources with an indicator of home ownership because the NHIS does not collect data on wealth. We also include an indicator of private health insurance coverage at interview to reflect material and nonmaterial resources garnered from past or current employment.

Health behaviors. The most important behavioral risk factors for mortality include smoking, obesity (reflecting poor diet and physical activity), and heavy alcohol consumption as a distant third (Mokdad et al. 2004). We include smoking as a three-level variable indicating current smokers, former smokers, or individuals who never smoked (omitted reference). We include a binary measure

of body mass index to identify whether the respondent was obese (body mass index ≥ 30 kg/m^2). Alcohol consumption is a four-level variable indicating lifetime abstainers, former drinkers (consumed no alcohol in the prior year), light and moderate drinkers who consumed alcohol up to 2 days per week during the prior year (omitted reference), or heavy drinkers who consumed alcohol 3 or more days per week during the prior year.

Some women were missing data on one or more mechanisms. Fewer than 1 percent of women were missing marital status, smoking, employment, home ownership, or health insurance; 1 percent to 5 percent were missing psychological distress, occupation, alcohol consumption, spouse's education, or obesity; and 25 percent were missing poverty status. We imputed these missing values using IVEware multiple imputation software (Raghunathan, Solenberger, and Van Hoewyk 2002). Table 1 shows the resulting distribution of the mechanisms by education level among non-Hispanic white women aged 45 to 84 years in the 1997 to 2004 NHIS surveys. The table splits the eight NHIS surveys into two 4-year groups (1997–2000 and 2001–2004) to more clearly illustrate how the distributions changed. Low-educated women were disadvantaged on all mechanisms in both time periods. For example, from 1997 to 2000, they were less likely to be married (47 percent vs. 65 percent), and if they were married, they more likely to have low-educated spouses (57 percent vs. 10 percent) compared with high-educated women. The last column shows that several disadvantages grew over time. For example, the percentage of low-educated women who had never smoked decreased from 52 percent to 50 percent between the two periods, whereas the percent among high-educated women increased from 54 percent to 55 percent, which widened the education gap from 2 percent to 5 percent. Growing disparities were also pronounced for spouse's education level, employment status, private health insurance, obesity, and being a former drinker.

Methods

We first built a person-year file that aged all non-Hispanic white women aged 25 to 84 years at interview by 1 year beginning with their NHIS interview until their year of death or 2006 if they survived. The age limit helps ensure that most women had completed their education through a high school credential; it also accounts for the top-coding at 85 years. Next, we retained person-year records for women who contributed person-years during 1997 to 2006, when they were 45 to 84 years of age. This allows women to "age in" and "age out" of the sample (see Montez et al. 2011). We set the lower limit at 45 years because there are few deaths before age 45 in the NHIS-LMF (just 3 percent of deaths among white women occur before age 45; Anderson 1999) and because ages 25 to 44 contributed little to the increasing gap, at least during the 1990s (Meara et al. 2008). We set the upper limit at 84 years because mortality matches are not as reliable among women 85 and older (Ingram, Lochner, and Cox 2008) and because the noninstitutional sample excludes nursing home residents, who are predominately older white women. The final sample contained 46,744 women who contributed 293,608 person-years and 4,053 deaths.

Using the person-year file, we estimated a series of discrete-time event history models using logistic regression. All models include age (time varying from 45 to 84 years), education, time, and an education-by-time interaction. A positive interaction indicates that the mortality gap widened. We then introduce the mechanisms to assess the extent to which they attenuate the interaction and thus may "explain" the widening mortality gap.

The models were estimated with SAS version 9.3 using PROC SURVEYLOGISTIC. The models were weighted by the eligibility-adjusted sample weights and accounted for the complex sample design of the NHIS-LMF. The model estimates were then analyzed with PROC MIANALYZE to account for the multiple imputation procedure.

RESULTS

We first estimated the growth of the education gap in mortality from 1997 to 2006. Model 1 in Table 2 contains coefficients from a logistic regression model predicting the ln(odds) of death from age, time, education, and the education-by-time interaction. From 1997 to 2001, the odds of death among low-educated white women aged 45 to 84 years were 1.37 times greater ($e^{0.317} = 1.37$) than the odds among their high-educated peers. From

Table 1. Distribution of Hypothesized Mechanisms by Education Level and Survey Year among White Women Aged 45 to 84 Years, 1997 to 2004.

	1997–2000			2001–2004			
Variable	Low Education[a]	High Education[b]	Difference	Low Education[a]	High Education[b]	Difference	Difference in Differences
Social-psychological factors							
Married (%)	47.0	65.0	–18.0***	48.1	64.7	–16.6***	1.4
Spouse's education (%)							
0–11 years	57.0	9.9	47.1***	54.5	7.8	46.7***	–.4
High school or some college	40.3	57.1	–16.8***	42.3	57.1	–14.8***	2.0
College	2.7	33.0	–30.3***	3.3	35.1	–31.8***	–1.5
Psychological distress (0–24)	4.0	2.3	1.7***	4.3	2.5	1.8***	.1
Economic circumstances							
Employed part- or full-time (%)	18.4	49.8	–31.4***	18.9	51.5	–32.6***	–1.2
Income below poverty (%)	20.0	6.0	14.0***	20.6	6.1	14.5***	.5
Home ownership (%)	74.9	86.3	–11.4***	74.9	87.2	–12.3***	–.9
Private health insurance (%)	61.0	85.0	–24.0***	56.2	83.6	–27.4***	–3.4
Occupation among employed (%)							
White-collar	7.2	40.5	–33.3***	8.2	40.2	–32.0***	1.3
Skilled	25.0	41.2	–16.2***	26.2	40.7	–14.5***	1.7
Manual	67.8	18.3	49.5***	65.6	19.1	46.5***	–3.0
Health behaviors							
Smoking (%)							
Current smokers	23.2	17.4	5.8***	23.9	16.9	7.0***	1.2
Former smokers	25.0	28.6	–3.6***	26.6	28.3	–1.7***	1.9
Never smoked	51.8	53.9	–2.1***	49.5	54.9	–5.4***	–3.3
Obese (%)	26.1	20.8	5.3***	31.0	23.7	7.3***	2.0
Alcohol consumption (%)							
Lifetime abstainers	46.6	23.9	22.7***	45.2	23.2	22.0***	–.7
Former drinkers	26.6	17.8	8.8***	28.2	17.3	10.9***	2.1
Current light drinkers	23.9	47.4	–23.5***	23.4	47.7	–24.3***	–.8
Current heavy drinkers	2.8	10.9	–8.1***	3.2	11.9	–8.7***	–.6
Age at interview (years)	67.2	59.6	7.6***	66.9	59.6	7.3***	–.3
n	4,081	19,810		3,108	19,745		

Note: Distributions are based on weighted respondent-level records.
[a]Zero to 11 years of education.
[b]Twelve or more years of education.
***$p < .001$ (age-adjusted two-tailed tests).

2002 to 2006, the odds among low-educated women were 1.66 times greater ($e^{0.317+0.192}$). The 21 percent growth in the odds ($100[e^{0.192} - 1]$) was significant at $p < .05$.

We next assessed the extent to which the widening gap can be attributed to the selected mechanisms. We did this by examining the degree to which the education-by-time interaction coefficient in Model 1 ($b = .192$, $p = .026$) attenuated when the mechanisms were statistically accounted for in Models 2 to 10. For each model, we report the ln(odds) coefficient and p value, the percentage

Table 2. Coefficients Predicting the ln(odds) of Death from Age, Time, Education, and Mechanisms among White Women Aged 45 to 84 Years, 1997 to 2006.

Variable	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6	Model 7	Model 8	Model 9	Model 10
Intercept	−11.094***	−10.526***	−11.445***	−9.251***	−9.224***	−9.743***	−12.125***	−11.983***	−10.450***	−10.678***
Age	.089***	.084***	.092***	.073***	.072***	.072***	.094***	.097***	.079***	.081***
Time[a]	−.050	−.026	−.047	.001	.002	−.008	−.045	−.056	.001	−.014
Low education[b]	.317***	.222**	.180*	.150*	.143†	.277***	.152*	.284***	.039	.244**
Low education × time	.192*	.184*	.204*	.170†	.165†	.170†	.155†	.163†	.139	.144
p value for interaction	.026	.034	.022	.052	.057	.051	.076	.062	.113	.101
Percentage explained[c]	—	4.2%	−6.2%	11.5%	14.1%	11.5%	19.3%	15.1%	27.6%	25.0%
Percentage explained[d]	—	5.1%	−5.4%	13.9%	16.6%	13.6%	23.0%	17.6%	32.7%	28.9%
Social-psychological										
Married		−.151**								
Married × spouse education		−.230***								
Psychological distress			.069***							
Economic										
Employed				−.769***		−.817***				−.784***
Income below poverty				.205**	.205**				.155*	
Home ownership				−.450***	−.447***				−.359***	
Health insurance				−.221***	−.215***				−.148**	
Occupation (not employed)										
White-collar					−1.038***				−.890***	
Skilled					−.735***				−.671***	
Manual					−.568***				−.563***	
Health behaviors										
Smoking (never)										
Current							1.072***	1.002***	.978***	.986***
Former							.575***	.506***	.543***	.495***
Obese							.126**		.075	
Alcohol (current light)										
Lifetime abstainer							.520***		.442***	
Former							.571***		.494***	
Current heavy							.054		.059	
AIC	16,072,146	16,014,289	15,934,455	15,878,928	15,872,142	15,970,985	15,774,522	15,867,515	15,635,271	15,773,523

Note: AIC = Akaike information criterion.
[a]Time is a binary variable (0 = 1997–2001, 1 = 2002–2006).
[b]Zero to 11 years of education.
[c]Percentage of the interaction coefficient in model 1 explained by the mechanisms using the ln(odds) coefficients in the table.
[d]Percentage of the interaction coefficient in model 1 explained by the mechanisms using *y*-standardized coefficients, available on request (Long and Freese 2005).
†$p < .10$. *$p < .05$. **$p < .01$. ***$p < .001$ (two-tailed tests).

attenuation of the coefficient, and the percentage attenuation of its *y*-standardized form (*y*-standardized coefficients [YSCs] are available on request). YSCs may improve the comparability of logistic regression models, whose unstandardized coefficients reflect the true effects of the predictors and the unobserved heterogeneity in the model (Long and Freese 2005; Mood 2010). The YSCs exhibited a somewhat greater degree of attenuation than did the unstandardized coefficients, but the overall findings were substantially comparable. We discuss each model using the ln(odds) coefficients to aid interpretation; we discuss the percentage attenuation across models (which is our main interest) using the YSCs because they may be more appropriate for model comparisons.

Models 2 and 3 examined the two components of social-psychological circumstances. Model 2 offered little support for the marriage component: the interaction attenuated slightly to .184, with a p value of .034 (5 percent attenuation of the YSC). Model 3 gave no support for the distress component. Models 4 to 6 offered some support for economic circumstances. Accounting for employment status, poverty, home ownership, and health insurance in Model 4 reduced the education-by-time interaction to .170, with a p value of .052 (14 percent attenuation of the YSC). When we disaggregated employment by occupational type in Model 5, the interaction coefficient was reduced to .165, with a p value of .057 (an almost 17 percent attenuation of the YSC). Among the economic circumstances, employment was by far the most important component. Indeed, comparing Model 6 (which contains only employment status) with Model 4 (which also includes income, home ownership, and health insurance) reveals that although the association between employment and mortality partly operated through income, home ownership, and health insurance, these three mechanisms contributed little to the widening gap net of employment. The final group of mechanisms, health behaviors, also received some support. Controlling for smoking, obesity, and alcohol use in Model 7 reduced the education-by-time interaction to .155, with a p value of .076 (23 percent attenuation of the YSC). Smoking was by far the most important behavior. Controlling only for smoking reduced the interaction term to .163, with a p value of .062 (an almost 18 percent attenuation of the YSC) in Model 8. It is noteworthy that the interaction was attenuated to a similar extent by smoking and by economic circumstances (in Model 5).

The results summarized so far indicate that diverging economic circumstances and health behaviors contributed to the growing mortality gap. Thus, we included both groups of mechanisms in Model 9. The interaction term (b = .139, p = .113) became statistically nonsignificant (33 percent attenuation of the YSC). Given that employment and smoking were the most important components, we included only these mechanisms in Model 10. They explained almost as much of the widening gap (29 percent attenuation of the YSC) as did all economic circumstances and behaviors combined, and the interaction term remained nonsignificant.[2]

Although accounting for economic circumstances and health behaviors reduced the interaction coefficient to statistical nonsignificance, their contribution to the growth in the gradient (33 percent) was relatively modest in practical terms. In addition, the interaction coefficient in Model 9 is not statistically different from the coefficient in Model 1. As we stated above, we expected to find relatively modest effects of the mechanisms that we examined given the difficulty in explaining mortality up to 10 years after a single interview and because, according to fundamental-cause theory, the contribution of any particular mechanism may vary over time. However, the results can still be used judiciously. One way to assess their validity is to contrast the mediating effects of smoking and obesity against the contribution of smoking-related and obesity-related causes of death to the widening education-mortality gap. Our finding that smoking was an important mechanism is consistent with recent research showing that lung cancer and CLRD explain a large proportion (25 percent to 50 percent) of the growth in the education-mortality gap among white women aged 45 to 84 years (Meara et al. 2008; Montez and Zajacova 2013). Thus, even a small attenuation of the interaction coefficient in our all-cause mortality models when controlling for a mechanism reported at interview (e.g., smoking) corresponds to a substantial contribution of that mechanism when assessing it using cause-of-death analyses (e.g., lung cancer). In addition, our finding (confirmed in ancillary models) that obesity contributed little to the growing gap in

Table 3. Coefficients Predicting the ln(odds) of Death from Age, Time, Education, and Health Status at Interview among White Women Aged 45 to 84 Years, 1997 to 2006.

Variable	Model 1	Model 2a	Model 2b	Model 3a	Model 3b
Intercept	−11.094***	−11.150***	−10.080***	−9.974***	−9.215***
Age	.089***	.088***	.075***	.085***	.075***
Time[a]	−.050	−.031	.002	−.001	.022
Low education[b]	.317***	.160*	.137†	.144†	.125†
Low education × time	.192*	.183*	.161†	.188*	.171†
p value for interaction	.026	.040	.070	.032	.050
Percentage explained[c]	—	4.7%	16.1%	2.1%	10.9%
Percentage explained[d]	—	5.5%	18.1%	3.7%	13.7%
Poor self-rated health		1.458***	1.358***		
No work limitations[e]				−1.142***	−1.063***
Employed			−.672***		−.548***
AIC	16,072,146	15,784,864	15,718,380	15,671,858	15,629,039

Note: AIC = Akaike information criterion.
[a]Time is a binary variable (0 = 1997–2001, 1 = 2002–2006).
[b]Zero to 11 years of education.
[c]Percentage of the interaction coefficient in Model 1 explained by the mechanisms using the ln(odds) coefficients in the table.
[d]Percentage of the interaction coefficient in Model 1 explained by the mechanisms using *y*-standardized coefficients, available on request (Long and Freese 2005).
[e]The National Health Interview Survey asked all respondents, "Does a physical, mental, or emotional problem now keep you from working at a job or business?" Respondents were classified as "unable to work," "able to work but limited in the amount or type of work," or "able to work without any limitation."
†$p < .10$. *$p < .05$. **$p < .01$. ***$p < .001$ (two-tailed tests).

all-cause mortality is consistent with research showing that diabetes-related mortality explained just 6 percent of the growing education-mortality gap among white women aged 45 to 84 years (Montez and Zajacova 2013). Smoking and obesity are also related to other causes of death; however, these comparisons provide a good first approximation of the validity of our results.

The finding that employment was the most important dimension of economic well-being raised the possibility that it was not employment per se that was important but rather that nonemployment may indicate inability to work because of poor health. We test this alternative explanation in Table 3. First, we controlled for self-rated health at interview in Model 2a (1 = poor, 0 = fair, good, very good, or excellent). Self-rated health had only a small effect on the education-by-time interaction. Next, we added employment status in Model 2b. Net of baseline health, employment attenuated the interaction term from .183 ($p = .040$) in Model 2a to .161 ($p = .070$) in Model 2b, which is a similar degree of attenuation when employment was included in Model 6 in Table 2. We repeated the analysis using an indicator of whether the respondent reported having no "physical, mental, or emotional problem that kept them from working at a job or business" at interview. We found that any divergence in simply being able to work across education levels had little impact on the growing mortality gap (comparing Model 1 with Model 3a). Moreover, even when controlling for ability to work, employment contributed to the growing gap, as shown in Model 3b. These results suggest that the contribution of employment to diverging mortality across education levels is at least partly due to the health benefits derived from employment.[3]

A Glance at Causes of Death

We replicated the analysis for deaths from heart disease, and from lung cancer or CLRD, to assess whether the results were consistent with etiological differences in these causes of death and thus

support the validity of the mechanisms.[4] The results (available on request) support the mechanisms' validity. As expected, the growth in the gap for these causes was larger than the growth for all-cause mortality, but it was only marginally significant ($p < .10$) because of small numbers of deaths. The widening gap in heart disease mortality reflected multiple mechanisms—marriage and spousal education, economic well-being, and health behaviors—while the widening gap in lung cancer and CLRD mortality mainly reflected smoking. Smoking explained over 3 times as much of the widening gap in lung cancer and CLRD mortality (34 percent) than it did for heart disease mortality (10 percent).

DISCUSSION

In this study, we examined three explanations—social-psychological factors, economic circumstances, and health behaviors—for the widening education gap in mortality risk from 1997 to 2006 among white women aged 45 to 84 years. Social-psychological factors contributed little to the increasing gap; however, economic circumstances and health behaviors played important roles. Accounting for economic circumstances (employment, occupation, poverty, home ownership, and health insurance) and health behaviors (smoking, obesity, and alcohol consumption) together explained the growing education gap in mortality to statistical nonsignificance. In practical terms, the contribution of economic circumstances and health behaviors was relatively modest: they explained roughly one third of the growth in the gap (although they fully explained the main effect of education). Employment status and smoking were the most important components.

The role of employment is intriguing and, to our knowledge, has not been previously examined as a potential explanation of the growing education gap in mortality. Although divergence in health-related ability to work may have played some role, our results indicate that employment was, in and of itself, an important contributor. Indeed, studies have found that the better health of employed adults was mostly attributed to employment itself rather than health selection into employment (Graetz 1993). Employment provides both manifest (e.g., income) and latent benefits, such as social networks and supports and a sense of purpose; it enhances self-esteem; and it offers mental and physical activity (Creed and Macintyre 2001). Access to social networks and support through employment may have become more important in recent decades, with high divorce rates, smaller families, and geographic mobility disrupting other avenues of support.

Future studies should examine employment in detail to better understand its contribution. Like most cross-sectional analyses, this study assumes that the mechanisms measured at survey reflect a meaningful degree of exposure to the mechanism during the life course. Thus, it assumes that differences in employment rates at the time of interview reflect differences in the propensity to work during adulthood. This is the same assumption, for example, that women who report being married (or smoking) at the time of survey have histories of being married (or smoking). Nevertheless, more research is needed to elucidate the role of employment. Employment histories could be informative, particularly among older respondents who may be retired at the time of survey.

The findings have several policy implications. One goal should be to increase the high school graduation rate (i.e., receiving a diploma, not a General Educational Development certificate), which has stagnated for white women since the mid-1940s birth cohorts (Heckman and LaFontaine 2010). This goal is bolstered by research that finds a substantial drop in mortality risk associated with a high school credential, more so than any other year of education (Montez, Hummer, and Hayward 2012).

Social protection policies are also needed. For example, work-family policies should be (re)designed to allow women who want desirable jobs outside the home to secure them. Women disproportionately head single-parent households and care for children and aging parents, all of which create major obstacles to employment outside the home. The obstacles are even higher for low-educated women, who tend to be confined to low-paying jobs with little flexibility in hours, little allowance for time off to care for family members, and limited social support outside the home (Heymann 2000). Some national work-family policies do exist, but they may not help low-educated women. For example, the Family and Medical Leave

Act entitles eligible employees of certain employers up to 12 weeks of unpaid but protected leave per year to attend to certain medical and family-related needs (U.S. Department of Labor n.d.). However, the policy does not pertain to private-sector employers with fewer than 50 employees, where low-educated women are disproportionately employed. It may also be financially prohibitive for women who do not have alternative sources of income, yet women with the fewest financial resources and social ties may need a policy like the Family and Medical Leave Act the most.

Other hurdles that low-educated women disproportionately face in finding and maintaining employment should also be addressed (see, e.g., Damaske 2011; Heymann 2000; Moen and Roehling 2005). Increasing the number of good jobs for working-class adults could help low-educated women enter the labor force. Maintaining employment could be facilitated by widespread availability of affordable day care. Mandating paid parental leave, paid sick time, and flexible work schedules (where possible) could also help low-educated women maintain employment, because the jobs available to them usually lack these benefits.

Continued policy efforts to reduce smoking could also stem the growing gap. However, these efforts must go beyond tobacco-control policies oriented at changing behavior (Graham et al. 2006). Qualitative research has shown that, despite knowing the health risks, socioeconomically disadvantaged women state that they smoke to relieve stress from the daily hassles of poverty, single parenting, and conflict-ridden relationships; because they are lonely and feel hopeless; because it provides a rare opportunity to socialize and feel a sense of belonging; and because they often have nothing interesting to do outside the home such as employment or affordable recreational activities (Stewart et al. 2011). These reasons also resonate with our findings that some of these distal correlates of smoking, such as employment, played an important role in the growing mortality inequality. As others have argued, a comprehensive policy framework—addressing education, poverty, and housing, for example—is needed to improve the circumstances faced by disadvantaged women (Stewart et al. 2011) because "social policy is tobacco control policy" (Graham et al. 2006:ii11).

In addition to the three categories of well-established mechanisms we examined in this study, other factors may be important. The childhood environment, for instance, has received increasing attention as indelibly shaping adult health (e.g., Duncan, Ziol-Guest, and Kalil 2010; Hayward and Gorman 2004; Kuh and Ben-Shlomo 2004) and other outcomes (e.g., Haas, Glymour, and Berkman 2011; Palloni et al. 2009). Childhood factors might have played a role in the growing mortality gap if they have polarized over time, as some research suggests (McLanahan 2004). The NHIS unfortunately does not routinely collect information about childhood conditions. Future research should assess the role of childhood contexts using other surveys that contain measures such as parental socioeconomic status and childhood health.[5]

Another potentially important factor is compositional changes. As average education levels increased over time, low-educated women may have become a more negatively select group. Although compositional changes likely occurred to some degree, empirical tests of their role in explaining diverging trends in well-being and mortality across education levels have generated little support (Blau 1998; Cutler et al. 2011; Martikainen, Blomgren, and Valkonen 2007; Meara et al. 2008). One indication that compositional changes are not the main explanation, at least for the increasing mortality among low-educated women since the mid-1980s, is that mortality among low-educated white men continued to decline (Montez et al. 2011). To glean some insights, in ancillary analyses, we controlled for the national high school graduation rate when each respondent was 17 years of age (U.S. Department of Education 1993). Controlling for the rate only slightly narrowed the widening gap (the education-by-time interaction decreased from .192 [$p = .026$] in the baseline Model to .185 [$p = .033$]), suggesting that compositional changes were not the driving force behind the growth in the mortality gap. This test should be interpreted cautiously because other contemporaneous trends that influenced mortality could confound it.

A few limitations of the study should be noted. First, data on mechanisms were collected only at the time of survey. Thus, we did not have information

on long-term exposures such as work or marital histories, which may have hindered our ability to detect their full contribution. Also, we did not have information on other potentially important mechanisms, such as parity, social networks, sense of control, and childhood environments. Our findings should not be generalized to other demographic groups, because their mortality trends and underlying mechanisms may differ. Future studies should use other data to assess trends among women aged 85 years and older, who constitute an increasing share of deaths. Although our study period is just 10 years, it reflects almost 50 years of birth cohorts (1913–1961). The diverging trends that we find likely reflect period and cohort effects (see Masters, Hummer, and Powers 2012 for recent evidence of cohort effects in the widening gradient) which we cannot partition with our data. Last, our effect sizes are fairly small, so our findings should be interpreted cautiously and validated using other data sets. In particular, the role of employment should be validated using data that contain detailed information on work histories.

CONCLUSION

The Healthy People initiative aims to improve the U.S. population's health and eliminate health disparities (U.S. Department of Health and Human Services 2000). However, disparities in the length of life among white women in the United States continued to grow during the late 1980s and through mid-2000s. This trend does not imply that such initiatives were ineffective, only that the structural forces working against them were stronger. One force appears to be the obstacles that low-educated women face in securing (desirable) employment. Our findings indicate that increasing high school graduation rates and redesigning work-family policies may improve longevity and reduce disparities among U.S. women. These solutions reflect the philosophy of Geoffrey Rose (2008), who argued that "the decisions which most affect the health of the nation are not taken in government departments of health but in those of the environment, employment, education, social security, and (especially) the Treasury" (p. 133).

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NOTES

1. In preliminary analyses, we included more detailed categories of education and similarly found that the widening gap was statistically significant only among the 0 to 11 year category, using a high school credential as the reference group.
2. In ancillary analyses, we replicated the models in Table 2 to assess whether categorizing recipients of General Educational Development certificates as low educated changed the results. We found that the higher risk for death among low-educated women was similar (b = .317 for low educated in Model 1 in Table 2 vs. b = .313 in the ancillary models), as was the education-by-time interaction coefficient (b = .192 in Model 1 in Table 2 vs. b = .208 in the ancillary models). The mechanisms attenuated the interaction coefficient in a similar pattern for both specifications.
3. In ancillary analyses, we stratified the models by age at interview to glean additional insights; however, the number of deaths in some age groups was too small to produce robust estimates. Note that although the percentage of women who were employed declined with age, many women remained employed after the traditional retirement age. For example, at age 70, 17 percent of high-educated women and 11 percent of low-educated women were employed.

4. We combined lung cancer and CLRD to increase power and because they share smoking as a major risk factor. Partly because of small numbers of deaths, other leading causes did not exhibit a statistically significant increase in the education-mortality gap.
5. Other studies have used adult height as a proxy for certain aspects of childhood, such as nutrition and pathogen exposure (e.g., Bozzoli, Deaton, and Quintana-Domeque 2009; Dowd, Zajacova, and Aiello 2009; Fogel 2004). In ancillary models, we analyzed adult height as a proxy for childhood conditions, but it did not attenuate the widening gap in mortality. However, height may be a poor proxy in high-income regions and for recent cohorts that experienced more favorable epidemiological contexts.

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