

**Why has the Educational Gradient in Mortality Increased among White U.S. Women?:
Examining the Contribution of Family, Psychosocial Resources, Behaviors, and Economics.**

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ABSTRACT

The inverse association between educational attainment and adult mortality risk (“the educational gradient”) in the United States is firmly established. Since the 1980s the gradient has increased, and it did so most noticeably among white women compared with white men, black women, and black men. However, we know little about underlying causes. The few attempts to identify causes have narrowly focused on one or two “usual suspects,” such as widening disparities in smoking behavior by education, yet those few causes contributed a relatively small portion. In this study, we will use the 1986-2006 National Health Interview Survey Linked Mortality File to investigate the increase in the gradient since the mid 1980s among white women. The study moves beyond prior attempts by systematically examining a wide range of sociodemographically-informed causes such as family characteristics—marriage, fertility, and spouse’s education—that have been neglected despite their strong relevance for women’s health.

The inverse association between educational attainment and adult mortality risk (“the educational gradient”) in the United States is firmly established (Hummer and Lariscy 2011). Since at least the 1960s the gradient has increased, thereby expanding the gap in life expectancy between adults with lower versus higher levels of educational attainment (Crimmins and Saito 2001; Lauderdale 2001; Meara, Richards and Cutler 2008; Preston and Elo 1995). Interestingly, the timing and extent of the increase in the gradient has varied across demographic subgroups. For instance, some studies report that the gradient increased during the 1960s and 1970s more noticeably among white men than white women (Feldman et al. 1989; Rogot, Sorlie and Johnson 1992). Since the mid 1980s, the gradient appears to have increased most noticeably for white women compared with white men, black women, and black men (Meara et al. 2008; Montez et al. 2011).

While there is wide agreement among scholars that the gradient has indeed increased over the last half century or so, there is no consensus on the underlying causal mechanisms. The few prior attempts to identify mechanisms have focused on one or two “usual suspects,” such as widening educational disparities in smoking behavior. However, these mechanisms individually explain a relatively small portion of the increasing gradient (Cutler et al. 2010). Indeed, Montez and colleagues (2011) recently urged scholars to systematically consider these and other potentially important mechanisms such as social ties and psychosocial resources. Thus, in this study, we move beyond prior attempts to identify causes of the increasing gradient by examining a wide range of sociodemographically-informed causes, many of which have been neglected even though they are particularly salient for women’s health, such as family characteristics. Specifically, we examine four broad categories of potential mechanisms: marriage and fertility, psychosocial resources and stressors, health behaviors, and economic and material resources.

Our findings address the growing concern among scholars and policy makers about the widening educational divide in longevity. For instance, the *Healthy People* initiative (DHHS 2000) has sought to reduce and ultimately eliminate health and mortality disparities within the U.S. population, including disparities by socioeconomic status. Our findings will indicate which causal mechanisms might have the greatest leverage in reducing the disparities, *at least under current contexts*. Our findings should also stimulate further research by illustrating the benefits of focusing on specific race-gender groups and tailoring explanatory hypotheses to reflect relevant demographic trends for each group, in line with an intersectionality approach (Mullings and Schulz 2006). We focus here on non-Hispanic white women because their gradient grew most prominently since the 1980s and because these women with less than a high school diploma experienced a decline in life expectancy during this time period (Meara et al. 2008)—a disconcerting trend given the overall gains in longevity made within the United States.

Hypothesized Mechanisms

Part of the difficulty in identifying the mechanisms linking education and mortality risk (or any health outcome) is that the mechanism are multifarious at any given point in time, they may change over time, and they may vary across demographic subgroup. 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 generalization 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). Acknowledging this complexity, we attempt to mitigate it to some degree it by focusing on mechanisms that are especially relevant for the time period and birth cohorts which we examine.

One group of mechanisms concerns family characteristics, specifically marriage and fertility, whose trends exhibit a growing divide across education levels. First, white women with higher levels of education have become more likely to marry and stay married than their less-educated peers (DiPrete and Buchmann 2006). This trend may have contributed to the increasing gradient because getting and staying married are associated with lower mortality risk. Marriage tends to lower mortality risk by, for example, enhancing economic well-being, providing social support, and promoting healthier behaviors (Umberson 1992; Waite 1995), although marital selection may also play a role (Goldman 1993). Second, 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 might have increased the gradient if, as some research suggests, the education level of *both spouses* contributes to each other's mortality risk, thereby compounding (dis)advantages (Montez et al. 2009). A third key trend is the growing divide in fertility patterns across education levels. Rindfuss and colleagues (1996) claimed that the association between education and fertility became stronger during the 1960s-1980s because more-educated women reduced their total fertility rate and delayed childbearing to a greater extent than less-educated women. While having children may protect against breast, ovarian, and uterine cancers, a recent review noted that higher parity increases the risks of cardiovascular disease, diabetes, stroke, and death, and that it does so most notably (or exclusively in some contexts) among women living in adverse environments where the physiological strains and energy demands of reproduction are not offset by nutritional intake and reduced energy expenditures (Jasienska 2009). Thus, parity may have become more closely linked to mortality risk among low-educated women compared with higher-educated women.

Divergence in health behaviors by education level has also occurred during the last several decades. Adults with more education have greater access to health-relevant information and more quickly integrate it into their life styles. For example, in 1954 when definitive studies linking cigarette smoking and cancer appeared in the media, 95.1 percent of college graduates claimed they had heard the information compared with 81.0 percent of adults with less than a high school diploma (Link 2008). While 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 emerged (Link 2008). Furthermore, the divergence in smoking behavior between education groups since the 1960s has been more pronounced among women than men (Meara et al. 2008). Adults with more education are also more likely to exercise, drink alcohol in moderation, and are less likely to be obese (Pampel, Krueger and Denney 2010).

Divergence in economic and material resources may also have contributed to the increasing gradient. Education increases the likelihood of being employed full-time, avoiding financial hardship, owning a home, and having access to employment-related health insurance (Ross and Wu 1995). These economic and material resources have been increasing hinged to higher education as the U.S. labor market continues to bifurcate. For example, employment rates have grown substantially more for adults 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 to 14.7 percent among women with less than a high school diploma versus 19.7 to 39.4 percent among women with at least five years of college (Spain and Bianchi 1996). The divergence is further highlighted by Diprete and Buchman (2006) who compared the standard-of-living returns to education for young (25-34 years) white and black males and females with a college degree against their peers with a high school diploma across the 1964 to 2002 Current Population

Surveys and found that the standard-of-living gap grew over time among women more than men and among whites more than blacks.

The fourth group of mechanisms that we consider includes psychosocial resources and stressors. Adults with more education tend to have greater psychosocial resources such as a greater sense of control (Mirowsky and Ross 2003). They are less likely to experience marital, parental, and financial stress, and traumatic events such as divorce, assault, and death of a child, which are deleterious for health (Lantz et al. 2005). Education also reduces the likelihood of experiencing depression across the life course (Miech and Shanahan 2000). To the extent that the other three groups of mechanisms that we consider deteriorate psychosocial resources and increase exposure to negative life events, divergence in those mechanisms between education levels during our study period may also correspond with divergence in psychosocial health.

As stated earlier, the aim of the present study is to investigate why the gradient increased so prominently among non-Hispanic white women since the mid 1980s by examining the contribution of the four groups of hypothesized mechanisms. First, we ask for which specific causes of death did the educational gradient in mortality risk increase among white women during this period? Examining specific causes of death can provide additional insights about the causal mechanisms. For example, if lung cancer was the only cause of death for which the gradient increased, then this provides powerful clues about mechanisms. Examining causes of death also allows us to determine whether the strength of each mechanism varies by cause of death. Second, we ask to what extent does each of the mechanisms—marriage and fertility, psychosocial resources and stressors, health behaviors, and economic and material resources explain the increasing gradient in all-cause and cause-specific mortality risk?

DATA AND METHODS

Data

We use data from the 2010 release of the public-use National Health Interview Survey Linked Mortality File (NHIS-LMF). This file links adults in the 1986 through 2004 annual cross-sectional waves of the National Health Interview Survey with death records in the National Death Index through December 31, 2006. The linkage is primarily based on a probabilistic matching algorithm (Lochner et al. 2008; NCHS 2009), which correctly classifies the vital status of 98.5 percent of eligible survey records. It also incorporates vital status information from the Social Security Administration and the Centers for Medicare and Medicaid Services. Adults classified as deceased in any of the three sources are identified as deceased in the NHIS-LMF.

Our analytic sample consists of a person-year file that includes non-Hispanic white women 45 to 84 years of age during the 1986 through 2006 period. We first created a person-year file by aging all match-eligible adults 18 years of age and older by one year beginning with their interview year until their year of death, or until 2006 if they survived the follow-up period. A few adults (0.5 percent) did not report their month or year of birth. In these cases, we imputed month of birth by random assignment and year of birth by subtracting the respondents' age from their interview year. We next identified person-year records for non-Hispanic white women who: (1) were 30 to 84 years of age at the time of NHIS survey, and (2) contributed person-years records during the study period when they were 45 to 84 years of age. The first criterion helps ensure that most women had completed their education at the time of survey; it also accounts for the top-coding of ages at 85 years. The second criterion allows for women to “age-in” and “age-out” of the analytic sample. For example, a woman 35 years of age during her 1990 NHIS interview would meet the first criterion but she does not start contributing person-year records

until she turns 45 years of age in 2000. We set the lower limit at 45 years of age because there are few deaths in the NHIS-LMF for younger ages and because we want to build off of the analysis of Montez et al (2011). The final analytic sample contains <X> women who contributed <Y> person-years of exposure, and experienced <Z> deaths during the follow-up period.

Methods

The analysis first identifies specific causes of death for which the education-mortality gradient increased. The causes are classified according to the 10th International Classification of Disease (ICD-10) and grouped into 113 categories. For this analysis, the categories of interest and their 113-category codes include: heart disease (55-68), cancers excluding lung (20-43 except 27), lung cancer (27), cerebrovascular disease (70), chronic lower respiratory diseases (83-86), accidental and violent deaths (114-129), diabetes mellitus (46), influenza and pneumonia (77-78), and chronic liver diseases and cirrhosis (94-95). For each category, we build a Poisson regression model to estimate the natural logarithm of the annual mortality rate as a linear function of: (1) age as a time varying covariate ranging from 45 to 84 years, (2) education measured as four binary variables indicating less than high school, high school, some college, or a bachelor's degree or higher as the omitted reference, (3) time as an ordinal variable reflecting three seven-year time periods within the 21-year follow-up period (1986 to 1992=1, 1993 to 1999=2, 2000 to 2006= 3) (see Montez et al. 2011 for a justification), and (4) the education by time interaction. A significant and negative interaction indicates the gradient increased. The analysis then examines the extent to which each of the hypothesized mechanisms contributed to the increasing gradient in all-cause mortality and in cause-specific mortality among the causes that exhibited a steeper gradient. We estimated all models using SUDAAN, weighted the data

using the eligibility-adjusted sample weights, and corrected for the complex survey design of the NHIS-LMF.

We include three family mechanisms: marital status, spouse's education, and parity. Marital status is defined as currently married or unmarried. Spouse's education is defined as an ordinal variable indicating less than high school (0), high school (1), some college (2), or a bachelor's degree or higher (3). Because unmarried women will not have spousal education data, we include these two measures in the models as a marital status (married=1, unmarried=0) and the product of marital status and spouse's education. Equation (1) illustrates such a model predicting the risk of death (D) from marital status (MAR) and spouse's education (SPED).

$$D_i = b_0 + (b_1 + b_2 SPED_i) MAR_i + \varepsilon_i \quad (1)$$

The partial derivative of the risk of death with respect to marital status is $(b_1 + b_2 SPED)$, so $b_0 + b_1$ indicates the risk of death for women married to a spouse with less than a high school degree (e.g., $SPED=0$). The partial derivative with respect to spouse's education is $b_2(MAR)$ which will be zero for unmarried women (e.g., $MAR=0$). The model can be estimated using equation (2).

$$D_i = b_0 + b_1 MAR + b_2 SPED_i MAR_i + \varepsilon_i \quad (2)$$

Note that we assume no interaction between own and spouse's education, which has been found using the NHIS-LMF (Brown, forthcoming). The third family mechanism, parity, is defined as the number of live births and is included as four binary variables to allow for a potential

nonlinear association. The variables indicate 0, 1-2 (omitted reference), 3-4, and 5 or more live births.

We include two health behaviors. Smoking is measured as three binary variables indicating never smoked (omitted reference), former smoker, or current smoker. BMI, while only a proxy for health behaviors, is measured as three binary variables indicating underweight ($BMI < 18.5$), normal or overweight ($18.5 \leq BMI < 30.0$) as the omitted reference, or obese ($BMI \geq 30$). We include three indicators of economic and material resources. Employment status is included as three binary variables indicating full-time (omitted reference), part-time, or not employed. Income is the logarithm of household income. Home ownership is a binary variable and is intended to reflect longer-term economic well-being than is reflected by the income measure (the NHIS does not collect data on wealth). Finally, we include indicators of psychosocial resources. Depression is a continuous measure using the CES-D. Additional measures of psychosocial resources will be included but are not yet defined.

A small percentage of respondents (<X percent>) were missing data on one or more mechanisms. We imputed these missing values from five datasets created by IVEware multiple imputation software (Raghunathan, Solenberger and Van Hoewyk 2002).

RESULTS

Forthcoming

DISCUSSION

Forthcoming

Table 1. Sociodemographic Characteristics of White Women by Time Period

	1986-1992	1993-1999	2000-2006
Age (years)			
Education			
Less than high school			
High school			
Some college			
College			
Family Characteristics			
Married			
Spouse less than high school			
Spouse high school			
Spouse some college			
Spouse college			
Parity			
Health Behaviors			
Never smoked			
Former smoker			
Current smoker			
Underweight			
Normal or overweight			
Obese			
Economic Resources			
Income (\$)			
Employed full-time			
Employed part-time			
Not employed			
Home ownership			
Psychosocial Resources			
Depression			
Other			
Number of Deaths			
Person-Years of Exposure			

Table 2. Poisson Regression Coefficients Predicting Changes in the Annual Risk of Death By Education Level Across the 1986-2006 Period

	All Causes	Cause 1	Cause 2	Cause 3	Cause 4	Cause 5	Cause 6	Cause 7	Cause 8	Cause 9
Age										
Time										
Education (CO) ¹										
LTHS										
HS										
SC										
Education x Time (CO)										
LTHS x time										
HS x time										
SC x time										
Number of deaths										
BIC (interaction model)										
BIC (main effects model)										

¹ LTHS=less than high school; HS=high school diploma or GED; SC=some college; CO=bachelor's degree or higher

Table 3. Poisson Regression Coefficients Predicting Changes in the Annual Risk of Death By Education Level and Adjusting for Hypothesized Mechanisms for Cause of Death “Z”

	Model 1	Model 2	Model 3	Model 4	Model 5
Age	X	X	X	X	X
Time	X	X	X	X	X
Education (CO)					
LTHS	X	X	X	X	X
HS	X	X	X	X	X
SC	X	X	X	X	X
Education x Time (CO)					
LTHS x time	X	X	X	X	X
HS x time	X	X	X	X	X
SC x time	X	X	X	X	X
Family Characteristics					
Married		X	X	X	X
Spouse’s Education		X	X	X	X
Parity		X	X	X	X
Health Behaviors					
Smoking			X	X	X
BMI			X	X	X
Economic Resources					
Income				X	X
Employment Status				X	X
Home ownership				X	X
Psychosocial Resources					
Depression					X
Other					X

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