Published in final edited form as:

Am Sociol Rev. 2011 December ; 76(6): 913–934. doi:10.1177/0003122411411276.

The Enduring Association between Education and Mortality: The Role of Widening and Narrowing Disparities

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Abstract

This paper examines how educational disparities in mortality emerge, grow, decline, and disappear across causes of death in the United States and how these change contribute to the enduring association of education and mortality over time. Focusing on adults age 40–64, we first examine the extent to which disparities in all-cause mortality by education persisted from 1989–2007. We then test the “fundamental cause” prediction that mortality disparities persist, in part, by shifting to new health outcomes over time, most importantly for those causes of death that have increasing mortality rates. To test this hypothesis, we focus in depth on the period from 1999–2007, when all causes of death were coded to the same classification system. The results indicate (a) both substantial widening and narrowing of mortality disparities across causes of death, (b) almost all causes of death that had increasing mortality rates also had widening disparities by education, and (c) the total disparity by education in all-cause mortality would be about 25% smaller today were it not for newly widened or emergent disparities since 1999. These results point to the theoretical and policy importance of identifying the social forces that cause health disparities to widen over time.

Inequality is a major cause of death in the United States. In the year 2007 death rates were more than 2.5 times higher for people with low education, as indicated by less than a high school diploma, in comparison to those with high education, as indicated by at least some

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college education (Xu, Kochanek, Murphy, and Tejada-Vera 2010). Despite substantial efforts by both the federal government and private institutions to reduce disparities by socioeconomic status (SES) (Robert Wood Johnson Foundation 2006; Thomson, Mitchell, and Williams 2006), disparities have proven remarkably resilient over time. Theoretical work using the ‘fundamental cause’ perspective (Link and Phelan 1995) proposes that one way disparities have been so robust is by shifting to new outcomes; as disparities in the major health outcomes of today eventually diminish, new ones emerge or widen in the outcomes that come to predominate in the future.

This study examines the extent to which mortality disparities endure over time by emerging or widening in new health outcomes, information that is currently unknown but is a linchpin in the ‘fundamental cause’ approach. First, we examine the prediction that underlying a stable or slowly widening disparity in all-cause mortality are both substantial widening and narrowing disparities across causes of death that largely counteract each other. The alternative hypothesis is that disparities in all-cause mortality are largely stable because there is little widening or narrowing of disparities across causes of death. Second, we develop and test the hypothesis that widening mortality disparities by education should be concentrated among outcomes with increasing mortality rates. Evidence to support these hypotheses would provide empirical support for the fundamental cause perspective and point to the promise of developing a new research emphasis. Efforts to specify how social determinants *change* health disparities over time would help lay the groundwork for new interventions that not only reduce disparities but also prevent new ones from emerging.

To empirically test these hypotheses, we focus on adults aged 40–64 and use data from both U.S. Vital Statistics and the U.S. Census from 1989 to 2007. The analysis focuses on approximately 85 major causes of death (the number varies slightly by demographic group), and for each one we examine the extent to which socioeconomic disparities increased or decreased over the study period.

THEORETICAL BACKGROUND

The Trends

Analyses examining trends in mortality disparities over time often rely on education as a key measure of SES. Education typically has the strongest effect on mortality when it is compared with other SES indicators such as occupation and income, and when both direct and indirect effects are considered, it encompasses a good part of the influence of the other indicators (see Kitagawa and Hauser 1973; Mirowsky and Ross 2003; Smith 2004; Williams 1990). In terms of direct effects, education/mortality differentials persist after controlling the influence of additional SES variables such as income and employment, although the education/mortality association is attenuated somewhat (Elo and Preston 1996; Preston and Elo 1995; Rogers, Hummer, and Nam 2000). In terms of indirect effects, individuals with more education are more likely than individuals with less education to be employed, work full-time rather than part-time, obtain high-status jobs, earn more, be promoted, accrue greater wealth, avoid economic hardship, enjoy more social connections, and engage in more beneficial health behaviors (Mirowsky and Ross 2003; Ross and Wu 1995; Schnittker 2004).

Evidence for a steady, gradual increase in mortality disparities by education between the 1960s and the 1980s comes from multiple sources and studies (Duleep 1989; Feldman et al. 1989; Kitagawa and Hauser 1973; Pappas et al. 1993; Preston and Elo 1995). Stable and gradually widening mortality disparities by education have continued through the 1990s into at least the year 2001 (Ahmedin et al. 2008). Studies of healthy life expectancy (Crimmins and Saito 2001) and cohort patterns of survival (Lauderdale 2001) confirm earlier studies by demonstrating widening disparities through 1990 but do not extend their analysis to the latest decades (reviewed in Hummer and Lariscy 2011; Singh 2004; Singh and Siahpush 2002; Singh and Siahpush 2006; Steenland, Hu, and Walker 2004). In light of these past trends, we expect that in recent decades mortality disparities by education have persisted, and may have even widened somewhat.

Theoretical Background and Predictions—In this study we test ‘fundamental cause’ predictions about the process behind the persistence of disparities over time, and in doing so address two limitations in the literature. The first limitation is a heavy reliance on a few select health outcomes despite prominent calls from within the field to expand the outcomes repertoire in the disparities literature (Aneshensel 1992; Pearlin 1989). Many studies focus heavily on the general concepts of psychological distress (see studies reviewed in Thoits 1995) or self-reported overall health (Hughes and Waite 2009; Warren and Hernandez 2007). On one hand, these broad measures are valid and appropriate to evaluate predictions based on social theories that typically focus on health in general and not on specific health outcomes like obesity or cancer. On the other hand, an extension of sociological research into additional health outcomes would expand the scope and relevance of sociological research. Examining diverse and specific types of health outcomes holds substantial potential in light of the fact that so many adverse health outcomes are concentrated in the lower social strata (Miech and Hauser 2001; NCHS 1999a).

A second limitation is that the fundamental cause perspective has undergone relatively few empirical tests or demonstrations in relation to its wide influence in the field. The few existing published tests have strengthened the empirical underpinning of the approach and aided in its theoretical development and refinement (Chang and Lauderdale 2009; Link et al. 2008; Lutfey and Freese 2005; Phelan et al. 2004). However, many of the perspective’s key and unique components remain untested.

We address these limitations by closely examining a key element of the fundamental cause perspective (Link 2008; Link and Phelan 1995; Lutfey and Freese 2005): That mortality disparities by education persist because a continual stream of widening and newly emergent disparities counteracts the effects of any disparities that are diminishing. As background, Link and Phelan (1995) note that 100 years ago the top killers in the United States were tuberculosis, diarrhea, and pneumonia, and these diseases were concentrated in the lower social strata. A person living a century ago might reasonably have thought that a substantial reduction in the influence of these top killers would also lead to substantial reduction in mortality disparities by socioeconomic strata. In the subsequent 100 years, the influence of these top killers did substantially diminish, but mortality disparities did not. The old killers were replaced by new ones—specifically, cardiovascular disease, cancer, and stroke (Omran 2005; Xu et al. 2010)—that are similarly concentrated in the lower socioeconomic strata

(Avendano et al. 2006; Clark et al. 2009; Singh et al. 2003). This perspective predicts that when the United States successfully counters the top killers of today, disparities will shift to the new ones that replace them.

Embedded within this perspective are at least two empirically testable hypotheses that we consider in this study. The first is the prediction that mortality disparities widen substantially for some causes of death while narrowing for others. The “fundamental cause” perspective posits that levels of flexible resources, such as money, power, prestige, knowledge, and beneficial social connections, allow people in the higher social strata to better take advantage of ever-changing developments in health (Link and Phelan 1995). For example, people in the higher social strata have gradually stopped smoking faster than those in the lower social strata since the health dangers of smoking became known (Pampel 2005), were the first to desist from cocaine use in the late 1980s when its cultural reputation changed from glamorous to unhealthy (Miech 2008), were more likely to use statins to improve cholesterol levels once the drugs came to market (Chang and Lauderdale 2009), benefited more quickly from highly active antiretroviral therapy (HAART) to treat HIV/AIDS when the technology became available in the mid 1990s (Goldman and Lakdawalla 2005), and among adolescents have more successfully resisted the obesity epidemic in recent decades (Miech et al. 2006).

As illustrated by the examples above, the health advantage of people in the upper social strata may come from use of the latest health technologies (such as uptake of statins), a more successful use of conventional health practices long known to improve health (such as avoiding smoking), or better positioning to protect against new health threats that enter a population (such as the obesity epidemic). This health advantage persists over historical time because health developments are ever-changing. When people in the lower social strata eventually close the gap for one outcome, individuals in the upper social strata have already moved on to improvements in other outcomes. This fluidity in disparities across causes of death implies that substantial widening and narrowing of disparities should occur over time.

Following this logic, some causes of death should be more sensitive to social influences than others, a premise of the fundamental cause perspective. Causes of death for which little is known about prevention or treatment are presumably less sensitive to social influences. In support, Phelan et al. (2004) show that causes of death that are less preventable have smaller disparities by education. Those in the higher social strata cannot employ flexible resources to avoid these outcomes and therefore do not gain special advantages. Consequently, even broad, universal social changes such as improvements in the standard of living and medical care that affect the health of everyone may widen disparities in some outcomes more than others.

A second prediction that follows from the fundamental cause perspective is that widening disparities should be concentrated among causes of death with increasing mortality rates. An increasing mortality rate indicates that traditional and established health treatments and behaviors are no longer sufficient to keep a cause of death under control. Dealing with the newly emerging risks requires changes in current behaviors and/or the development of new health practices and technologies. People in the upper social strata are uniquely positioned to

make such changes, according to the fundamental cause perspective, because of their greater level of flexible resources. Link and Phelan write, “If no new diseases emerged (such as AIDS), no new risks developed (such as pollutants), no new knowledge about risks emerged (as about cigarette smoking in the 1950s and 1960s), and no new treatments were developed (such as heart transplants), the concept of fundamental social causes would not apply” (Link and Phelan 1995:87). To the extent that increasing mortality rates call for people to change their existing health beliefs and practices or adopt new ones, people in the upper social strata should more quickly make these changes and realize the benefits. Conversely, given disadvantages in resources, people in the lower social strata can adapt less quickly to new health threats.

This second prediction highlights a key (but widely unrecognized) component of the explanation for the persistence of health disparities by the fundamental cause perspective. For socioeconomic health disparities to endure, they must continually emerge and widen in the causes of death that are on the rise. These causes are the ones among the pool of potential outcomes that are destined to become the major causes of death in the future. If not present in rising causes of death, major disparities would have disappeared many decades ago when the top killers of the previous era diminished.

An important caveat is that the association between increasing mortality rates and widening disparities is not expected to be perfect. A cause of death with a high mortality rate may not display a widening disparity if little is known about how to prevent or treat the disease. Such cases preclude people in the higher social strata from more successfully using their flexible resources to avoid it (Phelan et al. 2004). But given that most causes of death today are preventable (95 out of 96 major causes of death are preventable to at least some degree, see Appendix A of Phelan et al. 2004), widening disparities should, on average, emerge among causes of death with increasing mortality rates.

Testing these two predictions of the fundamental cause perspective offers an opportunity both to advance the development of the perspective as well to inform other theories of SES and health. Evidence for the predictions would not only support the fundamental cause perspective but would also suggest new approaches to current topics of health research. For example, more information on how cause of death moderates the association of SES and health can contribute to theoretical and empirical considerations of the varied ways that macrosocial factors such as social stress (Turner, Wheaton, and Lloyd 1995) or relative deprivation (Wilkinson 1996) affect population health.

Race and Gender Differences

The posited heterogeneity in trends by cause may vary by gender and race. On one hand, the fundamental cause predictions apply to all demographic groups: All people in the upper social strata should be better able to adapt to ever-changing developments in health. Consequently, in the analyses that follow, we expect to see support for the hypotheses among men, women, whites, blacks, and Hispanics. On the other hand, the fundamental cause perspective has not yet explicitly considered potential differences across race/ethnicity and sex. Given that disadvantaged groups encounter more barriers and consequently more difficulty in converting their resources into desired goods such as health (Crimmins,

Hayward, and Seeman 2004), the influence of education on health may be attenuated among disadvantaged groups and both disparities and disparity trends may be relatively smaller than among majority groups. To preliminarily consider this possibility, the analyses stratify by demographic groups.

METHODS

The analysis uses two main data sets to calculate death rates for adults aged 40–64 by causes of death. We focus on mortality in middle adulthood and use a lower limit of age 40 to diminish the influence of educational right-censoring on our study results. Nontraditional students have become increasingly common, to the extent that more than 20% of current college students are between the ages of 25 and 40 (U.S. Census Bureau 2010). Restricting the age group to those 40 and over ensures that most adults will have completed their education. The upper limit of age 64 is standard practice in mortality studies and is a rough proxy for participation in the workforce. The population age 40–64 is an important component of the U.S. population that comprised about 30% of the population in the year 2000 (the age population to which the results are standardized, U.S. Census Bureau 2001), the age population to which the results of this study are standardized.

U.S. Vital Statistics include yearly data from all death certificates issued in the United States. In 1989—the first year in which the decedent’s educational attainment was included on the death certificate—U.S. Vital Statistics included about 2.1 million records, and by 2007 the number of deaths had increased to about 2.5 million. Education is an especially powerful and attractive dimension of SES because it (1) is available for everyone, unlike income, employment, and occupational status; (2) is generally established early in adulthood and over time is more stable than income, wealth, occupational status, or employment status; (3) precedes most other SES measures and is often required for high-status jobs, salary increases, and promotions; (4) may be more accurately reported by survey respondents and death certificate informants than the more nuanced and variable measures of income and wealth; and (5) is less likely than income and wealth to exhibit reverse causality (ill health in adulthood does not lead to loss of educational status, but it can and does lead to loss of income, employment, and occupational standing) (Elo and Preston 1996; Hummer and Lariscy 2011; Preston and Elo 1995; Rogers, Hummer, and Nam 2000). Furthermore, compared to prospective studies where income or occupation may be ascertained years before the respondent dies and may therefore be out of date, obtaining educational level from the death certificate means that education is reported at the time of death.¹

The data also contain information on the race, age, and sex of decedents, as well as underlying cause of death, and the large sample size supports detailed analysis of all these factors. For analysis of mortality by causes of death, we focus on the period from 1999 to 2007, when all deaths were coded to the same classification system: the Tenth Revision of

¹Occupation and industry are also reported on the death certificate but were available only for selected states. For example, Rosenberg et al. (1993) examined death certificate data in 1984 for 12 states, and Burnett, Maurer, and Dosemeci (1997) were able to examine death certificate data for only 24 states from 1984 through 1988. Also, data are collected for a person’s usual occupation and kind of business or industry, which may not reflect his or her last job or entire work history. Further, coding for individuals who are not currently employed may create bias, especially for those who are retired and for women who list their usual occupation as “housewife” (Burnett, Maurer, and Dosemeci 1997; Rosenberg et al. 1993).

the International Classification of Diseases, or ICD (World Health Organization 1992). In 1998 and beforehand deaths were coded to earlier versions of the ICD, which are substantially different and therefore difficult to compare. For this analysis, we use the 113 Selected Causes of Death (NCHS 1999b), which aggregate the 12,420 codes in the ICD-10 to 113 major causes. Of the causes in this classification, we excluded seven “residual” causes that had no clear interpretation (e.g., one excluded code was 111, “all other diseases”). The analysis also excludes causes of death with exceedingly low rates: specifically, causes that did not have at least three years with at least one recorded death in each of the three educational levels (“less than 12 years of education,” “12 years of education,” and “13+ years of education”). All mortality data used in this study are currently available at <http://www.nber.org/data/vital-statistics-mortality-data-multiple-cause-of-death.html>.

The second main source of data for this analysis is the U.S. Census. For calculations of mortality rates, the Census data provided information for the denominator. For the purposes of this analysis, it was necessary to derive from the Census estimates of the total population of the United States, including those incarcerated or in nursing homes. Consequently, we supplemented information on the size of the noninstitutionalized civilian population—for which the Census provides yearly information—with information on the population that was living in group quarters imputed from the decennial 1990 and 2000 censuses. For the age group under study, the group quarters population was not more than about 3% of the size of the civilian population. All analyses are age standardized to the year 2000 U.S. population, a process that controls the potential influence on mortality rates of changing proportions over time of older and younger people within the group aged 40–64.

Race is classified as Hispanic, non-Hispanic white, or non-Hispanic black. For brevity, the text refers to non-Hispanic whites and blacks as whites and blacks.

The analysis uses Hierarchical Linear Modeling software, version 6.08 (Raudenbush et al. 2004), to model mortality rate trajectories by cause of death. Preliminary analysis indicated that the trajectories were linear and that a quadratic term did not contribute significantly to the model. Accordingly, the analysis consisted of a two-level model with a linear estimate of rate change:

Level 1 model:

$$\begin{aligned} \text{Mortality rate}_{ti} = & \pi_{0i} + \pi_{1i}(\text{year}_{ti}) \\ & + \pi_{2i}(<\text{High School}_{ti}) \\ & + \pi_{3i}(\text{High School}_{ti}) \\ & + \pi_{4i}(<\text{High School}_{ti})(\text{year}_{ti}) \\ & + \pi_{5i}(\text{High School}_{ti})(\text{year}_{ti}) + e_{ti} \end{aligned}$$

Level 2 model:

$$\pi_{0i} = B_{00} + B_{01}(\Delta \text{in overall Mortality Rate}_i) + B_{02}(\text{Mortality Rate in 2007}_i) + r_{0i}$$

$$\pi_{1i} = B_{10} + B_{11}(\Delta \text{in overall Mortality Rate}_i) + B_{12}(\text{Mortality Rate in 2007}_i) + r_{1i}$$

$$\pi_{2i} = B_{20} + B_{21}(\Delta \text{in overall Mortality Rate}_i) + B_{22}(\text{Mortality Rate in 2007}_i) + r_{2i}$$

$$\pi_{3i} = B_{30} + B_{31}(\Delta \text{in overall Mortality Rate}_i) + B_{32}(\text{Mortality Rate in 2007}_i) + r_{3i}$$

$$\pi_{4i} = B_{40} + B_{41}(\Delta \text{in overall Mortality Rate}_i) + B_{42}(\text{Mortality Rate in 2007}_i) + r_{4i}$$

$$\pi_{5i} = B_{50} + B_{51}(\Delta \text{in overall Mortality Rate}_i) + B_{52}(\text{Mortality Rate in 2007}_i) + r_{5i}$$

where cause of death years is the Level 1 unit of analysis and cause of death is the Level 2 unit of analysis; the variable Year represents the year of survey, with Year 0 being the baseline year; and the terms e_{it} , and $r_{0i}-r_{5i}$ represent random effects with a mean of 0 and an assumed normal distribution.

In brief, the Level 1 equation predicts each cause-specific mortality rate as a function of survey year and education. The variable π_{2i} indicates the extent to which the mortality rate differed for decedents with less than 12 years of education as compared to those with 13 or more (the reference category) in the baseline year. Similarly, the variable π_{3i} indicates the extent to which the mortality rate differed for decedents with 12 years of education versus 13 or more in the baseline year. A positive value for the variable π_{4i} indicates that over the course of the study period, the mortality rate increased faster for decedents with less than 12 years of education than for those with 13+, and that the disparity between these groups therefore widened. A negative value indicates a narrowing disparity. The variable π_{5i} is similar to π_{4i} and indicates level of disparity growth (or decline) for decedents with 12 years of school versus those with 13+.

The Level 2 equation models each of the Level 1 variables as a function of (a) overall mortality rate in 2007 (e.g. some causes of death have large mortality rates and others have small ones), and (b) change in that rate over the study period. A positive value for the coefficient B_{41} would indicate that increasing overall mortality rates predict increasing disparities, and also that decreasing overall mortality rates predict decreasing disparities across decedents with low education and those with high education (the reference group). The variable B_{51} is analogous to B_{41} and compares mid vs. high education groups. The variables B_{42} and B_{52} estimate how disparity growth is affected by size of the cause of death. The inclusion of all these coefficients in the same model controls potential confounding and ensures that the model estimates size and change effects net of each other. In the Level 2 model, the main coefficients of interest are B_{41} , B_{42} , B_{51} , and B_{52} , all of which are predictors of disparity change. The other Level 2 coefficients are included in the

model to ensure that these estimates are net of the influence of size and mortality rate change on the other components in the model.

The analysis also includes predicted and simulated mortality trajectories. To estimate predicted scores, the analysis summed the predicted mortality rate for each cause of death, as calculated using the predicted intercept and coefficients from the full model for each cause of death. The simulated trajectories estimate mortality rates if no widening of disparities had occurred over the study period. To calculate these simulated rates, the analysis used the method for obtaining predicted scores as described above, but reassigned to zero any π_{4i} or π_{5i} coefficient that had a positive value.

RESULTS

Figure 1 presents the trend in all-cause mortality disparities from 1989 to 2007. In accord with past trends, the mortality disparity increased over time, as evidenced by a widening distance between the mortality rates of people with the lowest and highest education levels. In addition, the trends are monotonic, with the mortality rates highest among those with the least education and steadily declining across groups with increasing education.

In analyses not shown, we considered whether the widening was driven by a specific age group. We ran analyses parallel to those for Figure 1, but for each separate five-year age group from 40 to 65, separately for men and women. The widening was present for every single five-year age group, suggesting that the trend is a historical period effect and cannot readily be explained as a cohort effect.

Figure 2 disaggregates the trends in Figure 1 by race/ethnicity and sex. A widening mortality disparity by education is present for all demographic groups. The increase in the disparity is smallest for Hispanic men and women.

The analysis next investigated how this overall widening disparity was distributed across major causes of death as coded in the U.S. Vital Statistics. We focus on trends in disparities from 1999 to 2007 both because the widening trend continued during this time (as indicated in Figures 1 and 2) and because all causes of death were coded to the same ICD-10 classification system.

To help visualize differences by cause of death, Figure 3 presents scattergrams of the change in mortality disparities by change in total mortality rates for each demographic group. Two main findings are apparent. The first is substantial heterogeneity in disparity levels across causes of death over the study period. Widening disparities by education over the study period are indicated by dots to the right of the vertical axis and narrowing disparities by dots to the left. Both substantial widening and substantial narrowing are present among all demographic groups. For women, among whites disparities widened for 76% of the causes of death and narrowed for the other 24%, among blacks 56% of disparities widened and 44% narrowed, and among Hispanics 64% widened and 36% narrowed. For men, among whites disparities widened for 65% of the causes of death and narrowed for the other 35%, among blacks 62% of disparities widened and 38% narrowed, and among Hispanics 48% widened and 52% narrowed.

This finding indicates that the overall widening in all-cause mortality presented in Figures 1 and 2 results from a process in which the influence of widening disparities outweighs that of narrowing disparities. The widening in all-cause mortality was not the result of a uniform increase in disparities across all causes of death. Furthermore, the widening was not the result of just a few causes of death that were outliers. Instead, Figure 3 indicates that a substantial number of disparities were widening while others were narrowing, although in sum the widening influences were stronger, as Figures 1 and 2 indicate.

A second finding indicated in Figure 3 is that widening disparities were concentrated among causes of death with increasing mortality rates, and likewise, narrowing disparities were concentrated among causes of death with decreasing mortality rates. Most of the dots in the scattergrams are concentrated in the upper right quadrant—which indicates both widening disparities and increasing mortality rates—and the lower left quadrant—which indicates both narrowing disparities and decreasing mortality rates. The concentration of widening disparities among causes of death with increasing mortality rates was pronounced. Among white men, 94% of the causes of death with increasing overall mortality rates (dots in the upper half of the scattergram) had widening disparities (dots on the right hand half of the scattergram). The analogous percentage for black men is 89% and for Hispanic men it is 58%. The analogous percentage for white women is 92%, for black women 87%, and for Hispanic women 81%. More generally, the correlation between change in mortality rate and change in disparity level is high, ranging from .86 and .80 for white and Hispanic men, to around .50 for black men as well as white and black women. For Hispanic women the correlation is only .28.

Table 1 provides detailed information on the causes of death involved in the scattergrams of Figure 3. The left half of Table 1 presents the causes of death for which disparities widened and narrowed most over the study period and reveals two main findings. First, widening and narrowing disparities are driven by a wide variety of health outcomes, and not a single type. For example, among white women, the causes of death that contributed most to the widening of mortality disparities included accidental poisoning (driven almost entirely by overdoses of prescription and illegal drugs, see Paulozzi, Ballesteros, and Stevens 2006), chronic lower respiratory diseases, and malignant neoplasms. Other top-three contributors to widening disparities were HIV (among black women), breast cancer (among Hispanic women), and viral hepatitis (among Hispanic men). Causes of death that contributed most to decreases in disparities were also varied.

A second finding apparent in the left half of Table 1 is substantial variability across demographic groups in the causes of death that contribute most to changes in educational disparities in mortality over time. For example, HIV is the top contributor to widening mortality disparities among black women, but it is also the top contributor to *narrowing* mortality disparities among Hispanic women.

The right half of Table 1 presents the causes of death with the greatest increases and greatest decreases in overall mortality rates during the study period. The cause of death with the greatest mortality rate increase over the study period was accidental poisoning, which ranks in the top three increasing causes of death for five of the six demographic groups. Its steep

rise is due in part to the greater availability of prescription drugs, whose nonmedical use is now the second most common form of U.S. illicit drug use (behind marijuana, see Office of Applied Studies - Substance Abuse and Mental Health Services Administration 2008) and the top cause of overdose deaths (ahead of cocaine and heroin, Paulozzi, Budnitz, and Xi 2006). In accord with the fundamental cause perspective, people with higher education have been better able to use resources to adapt to the risk factors responsible for this change, as indicated by a substantial increase in the disparity.

Table 1 shows that another cause of death that had a strong influence across all demographic groups was acute myocardial infarction. It ranked among the top three causes of death, but had a *decreasing* mortality rate. In fact, it was the cause of death with the top-ranked decrease for all groups except black men, for whom it ranked second behind malignant neoplasms of trachea, bronchus, and lung. The decrease in myocardial infarction mortality results in part from a lower incidence that has in turn resulted from population-level improvements in cholesterol levels and blood pressure, as well as decreases in smoking prevalence (Yeh et al. 2010). Table 1 shows that for all demographic groups, as the mortality rate due to acute myocardial infarction decreased, so too did its disparity by education. However, as the fundamental cause perspective predicts, these decreases were offset by almost equal or greater increases in mortality disparities in other outcomes (e.g., the increases in accidental poisoning).

While the general trend in Table 1 is for increasing causes of death to have increasing disparities and decreasing causes of death to have decreasing disparities, there are notable exceptions that warrant in-depth investigation. Among black men, the overall mortality rate due to HIV decreased, but disparities actually widened. Among white and black women, the mortality rate due to malignant neoplasms of the trachea, bronchus and lung increased, but the associated disparities narrowed. And among Hispanic women, the mortality rate due to breast cancer increased, but the disparity narrowed. These examples demonstrate that the association between changes in overall mortality rates and changes in disparity levels is strong but imperfect. To formally estimate the level of association, we turned next to hierarchical linear modeling.

Table 2 presents results from a two-level hierarchical linear model that predicts changes in disparity levels by education over the study period from 1999 to 2007. The unit of analysis for the second level is cause of death, which varies from a sample size of 79 (for Hispanic men) to 92 (for white men). The unit of analysis for the first level is the yearly mortality rate for each cause of death among decedents with low, intermediate, and high levels of education. All Level 1 coefficients are in bold, and all Level 2 coefficients—which predict the Level-one coefficients—are in regular type. The typical cause of death contributes 27 observations to the analysis pool; it contributes nine years of mortality rates (1999–2007), and for each year, it contributes three mortality rates (one for each of the three levels of education).

The main Level 1 variables in Table 2 act as expected. All coefficients for the variable “Year” are negative and statistically significant, indicating that on average mortality rates decreased over the study period among decedents with high education (the reference group).

All coefficients for the “Low education” variables are positive and statistically significant, indicating that at the start of the study period, mortality rates were higher for decedents with low (versus high) education. The coefficients for the “Mid education” variable follow the same trend. Finally, the results for the “Intercept” coefficients indicate the average mortality rate across the causes of death at the start of the study, and, as expected, are higher for men than women and blacks than whites.

The interactions of education with the “Year” variable in Table 2 are of particular interest because they directly measure whether disparities widened or narrowed over the study period. These results indicate that over time and compared to those with high education, those with low education witnessed mortality increases, so the disparity widened. The row for the B_{40} coefficients compares mortality trends across education, and all estimates are positive and statistically significant. These results indicate that the mortality rate increased faster for decedents with low education than for those with high education, so the disparity widened. This result is consistent with the graphs in Figures 1 and 2, in which the difference in the mortality rates across low and high education increased for every demographic group.

The row of results for the B_{41} coefficients in Table 2 examines a potential predictor of widening health disparities by education. Specifically, these results indicate whether a cause of death’s change in its mortality rate predicts changes in its disparity level across the highest as compared to the lowest level of education. These coefficients are positive and statistically significant for all demographic groups. The positive coefficients indicate that on average causes of death with increasing mortality rates also had increasing disparities, and causes of death with decreasing mortality rates had narrowing disparities. This pattern of results holds with a control for the size of the mortality rate for each cause of death, as indicated by the B_{42} coefficients.

The row of results for the B_{51} coefficients in Table 2 is parallel to that of the B_{41} coefficients, except that the B_{51} coefficients compare changes in disparity levels across the middle versus highest levels of education. Again, for all demographic groups, these coefficients are positive and statistically significant. The positive coefficients indicate that on average, causes of death with increasing mortality rates also had increasing disparities, and causes of death with decreasing mortality rates had narrowing disparities.

Figure 4 presents predicted and simulated mortality trends, calculated from the coefficients from the two-level hierarchical models presented in Table 2. The predicted results depicted with solid lines show the same trends in disparity change displayed in the observed data and graphed in Figure 2. Specifically, mortality disparities by education widened across demographic groups.

The simulated results depicted with dashed lines show what the group’s mortality rate would have been if no disparities had widened or emerged over the study period. To arrive at these estimates, the analysis considered the predicted rate of disparity growth for each cause of death in the analyses for each demographic group. All causes of death with a predicted widening disparity by education were reset to zero growth, so that the level of disparity at the baseline was constant throughout the study period. Causes of death with predicted

constant or declining disparities by education were left unaltered in the analysis, and the predicted mortality rates by education were then reestimated.

The simulated results show that disparity levels today would be substantially smaller if disparities had not emerged or widened, particularly the disparity between the least and most educated groups. In the absence of growing disparities, this difference would have diminished 26% for white women (from 829 to 615 deaths per 100,000), 32% for black women (from 624 to 423), and 36% for Hispanic women (from 154 to 98). Growing disparities had less influence on the difference in mortality rates across the middle education group and the highest; without growing disparities, this difference would have diminished 16% for white women (from 247 to 208 deaths per 100,000), but would remain basically unchanged for black and Hispanic women.

For men as for women, emerging and/or widening disparities had their greatest influence on the difference in mortality rates across the least and most educated groups. In the absence of growing disparities, this difference would have diminished 18% for white men (from 1,189 to 980 per 100,000), 20% for black men (from 1,326 to 1,057), and 22% for Hispanic men (from 270 to 210). And again, growing disparities had less influence on the difference in mortality rates across the middle educational group and the highest: without growing disparities, this difference would have diminished 11% for white men (from 418 to 373), but the difference would remain virtually unchanged for black and Hispanic men.

DISCUSSION

This study examines the proposition that mortality disparities by education persist over time because of their ability to continually emerge and/or widen in new outcomes. We first updated trends and documented that from 1989 to 2007 mortality disparities by education persisted and, indeed, actually widened for all demographic groups considered in this analysis. We then used the “fundamental cause” perspective to develop the predictions that (a) underlying this persistence are substantial widening and narrowing disparities across differing cause of death, and (b) widening disparities should be concentrated among causes of death with increasing mortality rates, which are the causes of death primed to predominate in the future. To test these two hypotheses we focus on the years 1999–2007, a period when there were no changes in the death classification system of the U.S. Vital Statistics.

The results provide evidence for these two predictions. Substantial widening and narrowing of disparities was most pronounced among black women, for whom disparities increased in about half the causes of death and decreased in the other half during the eight-year study period (the exact percentages are 56% and 44%, respectively). The other demographic groups also evidenced both widening and narrowing disparities for differing causes of death, with the percentage of widening disparities ranging between 48% and 76% across groups. These results indicate that waxing and waning of disparities across causes of death is substantial, continuous, and not restricted to a few outcomes. Furthermore, widening and emerging disparities play an important role in the perpetuation of mortality disparities by education; the results indicated that today mortality disparities across the least and most

educated group would be about 25% smaller if it were not for disparities that have widened or emerged since 1999.

Additional evidence for the fundamental cause perspective comes from the finding that widening disparities by education were concentrated among causes of death with increasing mortality rates. If mortality disparities by education remain strong over time by continually jumping to new outcomes, it then logically follows that they jump to outcomes that are becoming increasingly prominent and are the top killers of the future. In support of this proposition, the analysis indicates that almost all causes of death with increasing mortality rates also had increasing disparities by education. Specifically, among white decedents, 93% of the causes of death with increasing mortality rates had increasing disparities, and the analogous numbers for black and Hispanics are 88% and 70%, respectively.

Consistent with fundamental cause perspective, causes of death with the greatest widening of disparities were associated with outcomes that had substantial change in their risk factors. The cause of death with the largest widening of disparities by education was accidental poisoning, which was top-ranked among white men and white women, and in the top three among black men and black women. The risk for accidental poisoning has increased tremendously in recent years as scripts for prescription opioids have more than quadrupled from 44 million in 1991 to 179 million in 2009 (Miech, Koester, and Dorsey Holliman 2011; Volkow 2010). As predicted by the fundamental cause perspective, the consequences of this changing risk factor fell disproportionately on people with lower education, as indicated by a widening disparity in accidental poisoning mortality. Further, this cause of death is highly preventable and receives the highest preventability score possible on the Phelan et al. (2004) rankings. Given substantial change in risk factors and high preventability, this cause of death is indeed a strong candidate for widening disparities according to the fundamental cause perspective

Another way to interpret the association between increasing mortality and widening disparities is that causes of death on the upswing are driven primarily by increased mortality rates among people in the lower socioeconomic strata. The study results suggest that people in the lower strata are more likely to suffer the consequences of new health threats that enter a society. Such threats may both be more prevalent in the lower socioeconomic strata and/or they may also have greater impact. Reasons for a greater impact include the fact that people in the lower socioeconomic strata already have poorer health (NCHS 1999a) and therefore have fewer health reserves to resist the effects of new health threats, have higher levels of unhealthy behaviors (such as smoking and lack of regular exercise, see Pampel, Krueger, and Denney 2010) that may interact with new health threats and amplify their negative effects, and, in general, are less likely to be early adopters of new technologies, health or otherwise (Rogers 2003), that may protect against new health threats.

The main findings of the study held for all demographic groups in the analysis. Heterogeneity in disparity change occurred among both men and women, as well as whites, blacks, and Hispanics. Widening mortality disparities by education were present across all demographic groups, as was a concentration of increasing disparities among causes of death with increasing mortality rates. However, differences across groups also appear. One of the

most notable was the low mortality rate of Hispanics, especially among those with lower levels of education, a finding observed in other studies and attributable in part to the healthy immigrant effect and the “salmon bias,” in which foreign-born Hispanics return to their country of origin when they fall ill (Markides and Eschbach 2010; Palloni and Arias 2004).

As hypothesized, the concentration of widening disparities among causes of death with increasing mortality rates was, in general, less pronounced among disadvantaged as compared to advantaged demographic groups. The association of increasing mortality rates and widening disparities was typically smaller for black and Hispanic decedents than it was for whites, and within racial/ethnic groups it was typically smaller for women as compared to men. These results indicate that in disadvantaged groups the increased mortality resulting from causes of death on the rise was more equally distributed across educational levels. They suggest that people with more education were relatively less successful in protecting themselves from new health threats in disadvantaged as compared to advantaged groups.

The causes of death with the largest mortality increases and the most widening of disparities varied considerably across racial/ethnic groups and sex. This finding presents an interesting challenge to the fundamental cause perspective, which at its current state of development points primarily to generic SES-related processes to explain how higher SES groups maintain a health advantage despite ever-changing health developments. The results of this study suggest that an integration of the fundamental cause perspective with literatures on racial/ethnic and sex differences would be of mutual benefit.

One theoretical implication of the study findings is that they support a potential new field of study: the social determinants that *change* health disparities over time. While current sociological work has specified important social factors that contribute to disparities, any single factor by itself is insufficient to explain why a substantial percentage of health disparities widened while, at the same time, a substantial percentage narrowed. These results suggest the presence of intermediary factors that moderate how SES-related factors influence specific health outcomes. Specification of these intermediary factors would serve as a proving ground to further detail when and how sociological processes influence specific health outcomes. Further, it would bring sociological insights to wider audience by expanding current sociological work on health disparities to a wider scope of outcomes.

In terms of policy, these results suggest that efforts to make a lasting reduction in health disparities should include policies to prevent new disparities from forming. Without such policies, current efforts to reduce existing disparities will almost certainly be offset by the growth of disparities in other outcomes. In light of the fact that about 25% of the disparity in mortality rates across most and least educated in this study resulted from recent growth of disparities, perhaps about 25% of the annual \$2.5 billion that the National Institutes of Health (NIH) allocates to the reduction of health disparities could go toward disparity prevention programs.

A key finding of this study is that it is possible to predict which mortality disparities are likely to grow in the near future, a prediction necessary for programs seeking to prevent the growth of new disparities. Specifically, the causes of death with recent increases in overall

mortality rates are most likely the ones for which disparities will widen in the near future. Programs seeking to prevent disparity growth would do well to select a cause of death or a group of causes with large, recent increases in their overall mortality rate, and then test the effectiveness of a disparity prevention policy or intervention in one city or region as compared to a control group. A successful intervention should target an upstream factor and have substantial, cumulative effects observable across numerous causes of death.

This study is limited to education as a primary indicator of SES, adults aged 40–64, and a focus on absolute rather than relative disparities. Future studies could build on our results and examine additional measures of SES, including occupation (which is available on the death certificate for selected states), income and childhood SES indicators (which are not available on the death certificate). Adult occupation and income provide an opportunity to more fully and better measure SES, which taken together with education should lead to a better SES measure and thereby lead to stronger associations with fundamental cause processes. Measures of adults' earlier SES levels when they were children, when available, provide an unusual opportunity to examine the influence of SES on fundamental cause health outcomes with little to no potential bias of reverse causation. Such additional SES measures also hold the potential to challenge the fundamental cause perspective to the extent that their association with health is uniform across diseases over time.

An additional limitation is that we focus on mortality in middle adulthood and use a lower limit of age 40 to help diminish the influence of educational right-censoring on our study results. Future analyses of different age ranges provide the opportunity for additional tests of the fundamental cause perspective. We expect that widening disparities would continue to play a substantial role in the perpetuation of health disparities, although the specific causes of death would most likely differ as a result of differences across the life course in biology and social processes. We focus on absolute disparities because they have units of analysis (in this case deaths per 100,000) that are more readily interpretable than relative disparities, which do not have units of analysis. We hope that papers such as this that focus on absolute disparities will serve as a comparison and/or counterpoint for future analyses that focus on relative disparities.

Conclusion

Mortality disparities have endured in the United States because they shift to new outcomes over time. As disparities in the major health outcomes of today eventually diminish, new ones emerge or widen in the outcomes that come to predominate in the future—a process this study shows is continual and ongoing. Identifying the upstream processes that make this shift possible offers a unique opportunity to better specify the macro-micro link between social inequality and individual health. Such knowledge will lay the groundwork for policies and interventions aimed at disparity prevention, which this study indicates will be required of any comprehensive effort to achieve long-lasting reductions in health disparities.

Acknowledgements

This paper was supported in part by R01 DA020575 and K01 DA015089, Richard Miech principal investigator on both. We thank the Eunice Kennedy Shriver NICHD-funded University of Colorado Population Center (grant R21 HD51146) for administrative and computing support; NICHD grant 1R01 053696 for research support; the National

Center for Health Statistics and the U.S. Bureau of the Census for collecting the data and making the data files available to the research public; and the reviewers, Bethany G. Everett, and Robert J. Kemp for their helpful comments and suggestions on an earlier version of this paper. We also thank the Colorado State University College of Liberal Arts Societal, Organizational, and Policy Infectious Disease Cluster for their feedback on this paper. The content of this manuscript is the sole responsibility of the authors and does not necessarily represent the official views of NIH, NICHD, the U.S. Bureau of the Census, or NCHS.

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REFERENCES

- Ahmedin, Jemal; Ward, Elizabeth; Anderson, Robert N.; Murray, Taylor; Thun, Michael J. Widening of Socioeconomic Inequalities in U.S. Death Rates, 1993–2001. *PLoS ONE*. 2008; 3:e2181. [PubMed: 18478119]
- Aneshensel, Carol S. Social Stress: Theory and Research. *Annual Review of Sociology*. 1992; 18:15–38.
- Avendano, Mauricio; Kawachi, Ichiro; Van Lenthe, Frank; Boshuizen, Hendrik C.; Mackenbach, Johan P.; Vand den Bos, GAM.; Fay, Martha E.; Berkman, Lisa F. Socioeconomic Status and Stroke Incidence in the US Elderly: The Role of Risk Factors in the EPESE Study. *Stroke*. 2006; 37:1368–1373. [PubMed: 16690902]

- Chang, Virginia W.; Lauderdale, Diane S. Fundamental Cause Theory, Technological Innovation, and Health Disparities: The Case of Cholesterol in the Era of Statins. *Journal of Health and Social Behavior*. 2009; 50:245–260. [PubMed: 19711804]
- Clark, Alexander M.; DesMeules, Marie; Luo, Wei; Duncan, Amanda S.; Wielgosz, Andy. Socioeconomic status and cardiovascular disease: risks and implications for care. *Nature Reviews Cardiology*. 2009; 6:712–722. [PubMed: 19770848]
- Crimmins EM, Saito Y. Trends in Health Life Expectancy in the United States, 1970–1990: Gender, Racial, and Educational Differences. *Journal of Social Science and Medicine*. 2001; 52:1629–1641. [PubMed: 11327137]
- Crimmins, Eileen M.; Hayward, Mark D.; Seeman, Teresa E. Race/Ethnicity, Socioeconomic Status, and Health. In: Anderson, N.; Bulatao, R.; B, C., editors. *Critical Perspectives on Race and Ethnic Differences in Health in Later Life*. Washington, DC: National Academy of Sciences; 2004. p. 310–352.
- Duleep, Harriet O. Measuring Socioeconomic Mortality Differentials Over Time. *demography*. 1989; 26:345–351. [PubMed: 2731627]
- Elo, Irma T.; Preston, Samuel H. Educational Differentials in Mortality: United States, 1979–85. *Social Science and Medicine*. 1996; 42:47–57. [PubMed: 8745107]
- Feldman, Jacob J.; Makuc, Diane M.; Kleinman, Joel C.; Cornoni-Huntley, Joan. National Trends in Educational Differentials in Mortality. *American Journal of Epidemiology*. 1989; 129:919–933. [PubMed: 2705434]
- Goldman, Dana P.; Lakdawalla, Darius N. A Theory of Health Disparities and Medical Technology. *Contributions to Economic Analysis and Policy*. 2005; 4
- Hughes, Mary Elizabeth; Waite, Linda J. Marital Biography and Health at Mid-Life. *Journal of Health and Social Behavior*. 2009; 50:344–358. [PubMed: 19711810]
- Hummer, Robert A.; Lariscy, Joseph T. Educational Attainment and Adult Mortality. In: Rogers, RG.; Crimmins, E., editors. *International Handbook of Adult Mortality*. New York: Springer Publishers; 2011.
- Kitagawa, Evelyn M.; Hauser, Philip M. *Differential Mortality in the United States: A Study in Socioeconomic Epidemiology*. Cambridge, MA: Harvard University Press; 1973.
- Lauderdale DS. Education and Survival: Birth Cohort, Period, and Age Effects. *Demography*. 2001; 38:551–62. [PubMed: 11723951]
- Link, Bruce G. Epidemiological Sociology and the Social Shape of Population Health. *Journal of Health and Social Behavior*. 2008; 49:367–384. [PubMed: 19181044]
- Link, Bruce G.; Phelan, Jo. Social Conditions as Fundamental Causes of Disease. *Journal of Health and Social Behavior*. 1995; (Extra Issue):80–94. [PubMed: 7560851]
- Link, Bruce G.; Phelan, Jo C.; Miech, Richard A.; Leckman, Emily. The Resources that Matter: Fundamental Social Causes of Health Disparities and the Challenge of Intelligence. *Journal of Health and Social Behavior*. 2008; 49:72–91. [PubMed: 18418986]
- Lutfe, Karen; Freese, Jeremy. Toward Some Fundamentals of Fundamental Causality: Socioeconomic Status and Health in the Routine Clinic Visit for Diabetes. *American Journal of Sociology*. 2005; 110:1326–1376.
- Markides, Kyriakos S.; Eschbach, Karl. Aging, Migration, and Mortality: Current Status of Research on the Hispanic Paradox. *Journals of Gerontology Series B: Psychological Sciences & Social Sciences*. 2010; 60B(Special Issue II):68–75.
- Miech, Richard A. The Formation of a Socioeconomic Health Disparity: The Case of Cocaine Use during the 1980s and 1990s. *Journal of Health and Social Behavior*. 2008; 49:352–366. [PubMed: 18771068]
- Miech, Richard A.; Hauser, Robert M. Socioeconomic Status (SES) and Health at Midlife: A Comparison of Educational Attainment with Occupation-Based Indicators. *Annals of Epidemiology*. 2001; 11:75–84. [PubMed: 11164123]
- Miech, Richard Allen; Kumanyika, Shiriki K.; Stettler, Nicolas; Link, Bruce G.; Phelan, Jo C.; Chang, Virginia W. Trends in the association of poverty with overweight among US adolescents, 1971–2004. *JAMA*. 2006; 295:2385–2393. [PubMed: 16720824]

- Miech, Richard; Koester, Steve; Holliman, Brooke Dorsey. Increasing U.S. Mortality Due to Accidental Poisoning: The Role of the Baby Boom Cohort. *Addiction*. 2011 forthcoming.
- Mirowsky, John; Ross, Catherine E. *Education, Social Status, and Health*. New York: Aldine de Gruyter; 2003.
- NCHS. *Health, United States, 1999 with Health and Aging Chartbook*. Hyattsville, MD: 1999a.
- NCHS. *Instruction Manual Part 9: ICD-10 Cause of Death Lists for Tabulating Mortality Statistics, Effective 1999*. Hyattsville, MD: National Center for Health Statistics; 1999b.
- Office of Applied Studies - Substance Abuse and Mental Health Services Administration. *Results from the 2007 National Survey on Drug Use and Health: National Findings*. Substance Abuse and Mental Health Services Administration, Office of Applied Studies; 2008. DHHS Publication No. SMA 08-4343
- Omran, Abdel R. The Epidemiological Transition: A Theory of the Epidemiology of Population Change. *Milbank Memorial Fund Quarterly*. 2005; 83:731–757.
- Palloni, Alberto; Arias, Elizabeth. Paradox Lost: Explaining the Hispanic Adult Mortality Advantage. *Demography*. 2004; 41:385–415. [PubMed: 15461007]
- Pampel, Fred C. Diffusion, Cohort Change, and Social Patterns of Smoking. *Social Science Research*. 2005; 34:117–139. [PubMed: 22485056]
- Pampel, Fred C.; Krueger, Patrick M.; Denney, Justin T. SES Disparities in Health Behaviors. *Annual Review of Sociology*. 2010; 36:349–370.
- Pappas, Gregory; Queen, Susan; Hadden, Wilbur; Fisher, Gail. The Increasing Disparity in Mortality between Socioeconomic Groups in the United States, 1960 and 1986. *The New England Journal of Medicine*. 1993; 329:103–109. [PubMed: 8510686]
- Paulozzi LJ, Ballesteros MF, Stevens JA. Recent Trends in Mortality from Unintentional Injury in the United States. *Journal of Safety Research*. 2006; 37:277–83. [PubMed: 16828115]
- Paulozzi, Leonard J.; Annett, J. Unintentional Poisoning Deaths -- United States, 1999–2004. *Morbidity and Mortality Weekly Report*. 2007; 56:93–96. [PubMed: 17287712]
- Paulozzi, Leonard J.; Budnitz, Daniel S.; Xi, Yongli. Increasing Deaths from Opioid Analgesics in the United States. *Pharmacoepidemiology and Drug Safety*. 2006; 15:618–627. [PubMed: 16862602]
- Pearlin, Leonard. The Sociological Study of Stress. *Journal of Health and Social Behavior*. 1989; 30:241–256. [PubMed: 2674272]
- Phelan, Jo C.; Link, Bruce G.; Diez-Roux, Ana V.; Kawachi, Ichiro; Levan, Bruce. "Fundamental Causes" of Social Inequalities in Mortality: A Test of the Theory. *Journal of Health and Social Behavior*. 2004; 45:265–285. [PubMed: 15595507]
- Preston, Samuel H.; Elo, Irma T. Are Educational Differentials in Adult Mortality Increasing in the United States? *Journal of Aging and Health*. 1995; 7:476–496. [PubMed: 10165966]
- Raudenbush, Stephen W.; Bryk, Anthony S.; Cheong, Yuk Fai; Congdon, Richard T. *HLM 6: Hierarchical Linear and Nonlinear Modeling*. Lincolnwood, IL: Scientific Software International, Inc.; 2004.
- Robert Wood Johnson Foundation. *Our Mission*. Robert Wood Johnson Foundation; 2006. <http://www.rwjf.org/about/mission.jsp>
- Rogers, Everett M. *Diffusion of Innovations*. New York: The Free Press; 2003.
- Rogers, Richard G.; Hummer, Robert A.; Nam, Charles B. *Living and Dying in the USA: Behavioral, Health, and Social Differentials of Adult Mortality*. New York, New York: Academic Press; 2000.
- Ross CE, Wu C-L. The Links Between Education and Health. *American Sociological Review*. 1995; 60:719–745.
- Schnittker, Jason. Education and the Changing Shape of the Income Gradient in Health. *Journal of Health and Social Behavior*. 2004; 45:286–305. [PubMed: 15595508]
- Singh, Gopal K. Area Deprivation and Widening Inequalities in U. S. Mortality, 1969–1998. *American Journal of Public Health*. 2004; 93:1137–1143. [PubMed: 12835199]
- Singh, Gopal K.; Miller, BA.; Hankey, BF.; Edwards, BK. *Area Socioeconomic Variations in U. S. Cancer Incidence, Mortality, Stage, Treatment, and Survival, 1975–1999*. Bethesda, MD: National Cancer Institute; 2003.

- Singh, Gopal K.; Siahpush, Mohammad. Increasing Inequalities in All-Cause and Cardiovascular Mortality among U. S. Adults Aged 25–64 Years by Area Socioeconomic Status. *International Journal of Epidemiology*. 2002; 31:600–613. [PubMed: 12055162]
- Singh, Gopal K.; Siahpush, Mohammad. Widening Socioeconomic Inequalities in U. S. Life Expectancy, 1980–2000. *International Journal of Epidemiology*. 2006; 35:969–979. [PubMed: 16684899]
- Smith, James P. Unraveling the SES-Health Connection. *Population and Development Review*. 2004; 31(supplement):108–132.
- Steenland, Kyle; Hu, Sherry; Walker, James. All-Cause and Cause-Specific Mortality by Socioeconomic Status Among Employed Persons in 27 U. S. States, 1984–1997. *American Journal of Public Health*. 2004; 94:1037–1042. [PubMed: 15249312]
- Thoits, Peggy A. Stress, Coping, and Social Support Processes: Where Are We? What Next? *Journal of Health and Social Behavior*. 1995; (Extra Issue):53–79. [PubMed: 7560850]
- Thomson, Gerald E.; Mitchell, Faith; Williams, Monique B. Examining the Health Disparities Research Plan of the National Institutes of Health: Unfinished Business. Washington DC: The National Academies Press; 2006.
- Turner, R Jay; Wheaton, Blair; Lloyd, Donald A. The Epidemiology of Social Stress. *American Sociological Review*. 1995; 60:104–125.
- U.S. Census Bureau. U.S. Census Bureau; 2001. Profiles of General Demographic Characteristics 2000. <http://www.census.gov/prod/cen2000/doc/ProfilesTD.pdf>
- U.S. Census Bureau. U.S. Census; 2010. Table A-6 Age Distribution of College Students 14 Years Old and Over, by Sex: October 1947 to 2008. <http://www.census.gov/population/www/socdemo/school.html>
- Volkow, Nora D. Congressional Caucus on Prescription Drug Abuse. Washington, DC: 2010. Report on Scientific Research on Prescription Drug Abuse.
- Warren, John Robert; Hernandez, Elaine M. Did Socioeconomic Inequalities in Morbidity and Mortality Change in the United States over the Course of the Twentieth Century? *Journal of Health and Social Behavior*. 2007; 48:335–351. [PubMed: 18198683]
- Wilkinson, Richard G. *Unhealthy Societies: The Afflictions of Inequality*. London: Routledge; 1996.
- Williams, David. Socioeconomic Differences in Health: A Review and Redirection. *Social Psychology Quarterly*. 1990; 53:81–99.
- World Health Organization. *International Statistical Classification of Diseases and Related Health Problems, Tenth Revision*. Geneva: World Health Organization; 1992.
- Xu, Jiaquan; Kochanek, Kenneth D.; Murphy, Sherry L.; Tejada-Vera, Betzaida. Deaths: Final Data for 2007. *National Vital Statistics Reports*. 2010; 58:1–73. [PubMed: 25075874]
- Yeh, Robert W.; Sidney, Stephen; Chandra, Malini; Sorel, Michael; Selby, Joseph V.; Go, Alan S. Population Trends in the Incidence and Outcomes of Acute Myocardial Infarction. *New England Journal of Medicine*. 2010; 362:2155–2165. [PubMed: 20558366]

Legend: ◆ Less than 12 years educ. ■ 12 years educ. ▲ 13-15 years educ. ● 16+ years educ. — Overall trend

Figure 1.
Trends in U.S. Mortality Levels by Education for Men and Women Aged 40–64, 1989–2007

Source: U.S. Vital Statistics and U.S. Census

Note: Age standardized to year 2000 U.S. population

Note: In 1999 classification of cause of death changed to the ICD-10 from the ICD-9. The detailed analyses that follow in this paper focus on the period 1999–2007; earlier years are presented in this graph to put the 1999–2007 trends in context.

Source: U.S. Vital Statistics and U.S. Census

Note: Age standardized to year 2000 U.S. population

Note: In 1999 classification of cause of death changed to the ICD-10 from the ICD-9. The detailed analyses in this paper focus on the period 1999–2007; earlier years are presented in this graph to put the 1999–2007 trends in context.

Legend: ◆ Less than 12 years educ. ■ 12 years educ. ▲ 13–15 years educ. ● 16+ years educ. — Overall trend

Figure 2.
Trends in U.S. Mortality Levels by Race/Ethnicity and Education for Men and Women
Aged 40–64, 1989–2007

Source: U.S. Vital Statistics and U.S. Census

Note: Age standardized to year 2000 U.S. population

Note: In 1999 classification of cause of death changed to the ICD-10 from the ICD-9. The detailed analyses in this paper focus on the period 1999–2007; earlier years are presented in this graph to put the 1999–2007 trends in context.

Figure 3.
Scatterplots of Change in U.S. Mortality Disparities by Change in Total Mortality Rates, 1999–2007.

Note: Change in total mortality rate is the observed mortality rate in 1999 subtracted from the observed mortality rate in 2007.

Note: Age standardized to year 2000 population.

Note: Change in disparity is based on two educational groups of low education (12 years of education or less) and high education (13 years of education or more). It is the absolute difference in mortality rates across these two educational groups in 1999 subtracted from the absolute difference in 2007.

Note: All correlations significant at or below the .01 level.

Source: U.S. Vital Statistics and U.S. Census
Note: Age standardized to year 2000 U.S. population

Legend:
 ◆ < than 12 years educ.
 ■ 12 years educ.
 ● 13+ year educ.
 - - - - - Projected Overall Mortality Rate, If No Widening Disparities

Figure 4.

Predicted and Simulated Mortality Trends, Based on Results in Table 2

Note: The analysis pool excludes causes of death that are residual categories, and consequently the total number of deaths in these predicted models is less than the total number of deaths in the observed data.

Source: U.S. Vital Statistics and U.S. Census

Note: Age standardized to year 2000 U.S. population

Table 1
Changes in Disparities and Changes in Overall Mortality Rate: Top Three and Bottom Three Causes of Death by Sex and Race/Ethnicity, Ages 40–64, United States 1999–2007

Top Widening and Narrowing Disparities (ICD-10 Codes)	Disparity Change	Mortality Rate Change	Top Increasing and Decreasing Causes of Death (ICD-10 Codes)	Mortality Rate Change	Disparity Change
White Women (n=90)					
Accidental poisoning (X40–X49) *	12.9	10.9	Accidental poisoning (X40–X49) *	10.9	12.9
Chronic lower respiratory diseases other than bronchitis, emphysema, or asthma (J44, J47)	7.15	0.082	Suicide by means other than firearms (U03.X60 X71.X75 X84.Y87.0)	2.39	1.85
Malignant neoplasms of trachea, bronchus, and lung (C33–C34)	5.99	–6.12	Alcoholic liver disease (K70)	1.66	3.11
Emphysema (J43)	–0.835	–1.50	Malignant neoplasms of trachea, bronchus, and lung (C33–C34)	–6.12	5.99
Chronic ischemic heart disease other than atherosclerotic (I20, I25.1–I25.9)	–3.05	–5.53	Malignant neoplasm of breast (C50)	–6.99	0.58
Acute myocardial infarction (I21–I22)	–4.77	–7.97	Acute myocardial infarction (I21–I22)	–7.97	–4.77
Black Women (n=85)					
HIV (B20–B24)	13.5	1.37	Accidental poisoning (X40–X49) *	6.45	7.92
Accidental poisoning (X40–X49) *	7.92	6.45	HIV (B20–B24)	1.37	13.5
Malignant neoplasms of trachea, bronchus, and lung (C33–C34)	5.56	–4.84	Septicemia (A40–A41)	1.06	1.69
Alcoholic liver disease (K70)	–2.49	–2.14	Diabetes mellitus (E10–E14)	–8.10	–5.32
Diabetes mellitus (E10–E14)	–5.32	–8.10	Chronic ischemic heart disease other than atherosclerotic (I20, I25.1–I25.9)	–9.80	–1.67
Acute myocardial infarction (I21–I22)	–9.77	–13.9	Acute myocardial infarction (I21–I22)	–13.9	–9.77
Hispanic Women (n=80)					
Malignant neoplasm of breast (C50)	2.98	–2.48	Accidental poisoning (X40–X49) *	1.92	–0.201
Malignant neoplasms of colon, rectum, and anus (C18–C21)	2.97	–0.275	Septicemia (A40–A41)	0.896	0.435
Renal failure (N17–N19)	2.09	0.576	Viral hepatitis (B15–B19)	0.849	0.351
Congenital malformations, deformations, and chromosomal abnormalities (Q00 Q99)	–1.06	–0.578	Chronic ischemic heart disease other than atherosclerotic (I20, I25.1–I25.9)	–3.98	0.224
HIV (B20–B24)	–2.59	–2.05	Diabetes mellitus (E10–E14)	–4.01	–0.843
Acute myocardial infarction (I21–I22)	–2.79	–5.51	Acute myocardial infarction (I21–I22)	–5.51	–2.79
White Men (n=92)					
Accidental poisoning (X40–X49) *	16.7	13.6	Accidental poisoning (X40–X49) *	13.6	16.7

Top Widening and Narrowing Disparities (ICD-10 Codes)	Disparity Change	Mortality Rate Change	Top Increasing and Decreasing Causes of Death (ICD-10 Codes)	Mortality Rate Change	Disparity Change
Chronic lower respiratory diseases other than bronchitis, emphysema, and asthma (J44,J47)	6.40	-0.484	Suicide by means other than firearms (U03.X60 X71.X75 X84.Y87.0)	4.62	4.30
Viral hepatitis (B15–B19)	5.28	2.89	Hypertensive heart disease (I11)	3.70	3.38
Malignant neoplasms of trachea, bronchus, and lung (C33–C34)	-5.22	-16.49	Chronic ischemic heart disease other than atherosclerotic (I20, I25.1–I25.9)	-13.9	-6.41
Chronic ischemic heart disease other than atherosclerotic (I20, I25.1–I25.9)	-6.41	-13.9	Malignant neoplasms of trachea, bronchus, and lung (C33–C34)	-16.49	-5.22
Acute myocardial infarction (I21–I22)	-13.5	-23.8	Acute myocardial infarction (I21–I22)	-23.8	-13.5
Black Men (n=86)					
Malignant neoplasms of liver and intrahepatic bile ducts (C22)	11.0	5.32	Hypertensive heart disease (I11)	6.41	7.73
Hypertensive heart disease (I11)	7.73	6.41	Malignant neoplasms of liver and intrahepatic bile ducts (C22)	5.32	11.0
Accidental poisoning (X40–X49) *	7.60	5.20	Accidental poisoning (X40–X49) *	5.20	7.60
Malignant neoplasms of trachea, bronchus, and lung (C33–C34)	-5.32	-26.9	HIV (B20–B24)	-19.86	6.76
Acute myocardial infarction (I21–I22)	-7.33	-25.6	Acute myocardial infarction (I21–I22)	-25.6	-7.33
Alcoholic liver disease (K70)	-7.53	-6.42	Malignant neoplasms of trachea, bronchus, and lung (C33–C34)	-26.9	-5.32
Hispanic Men (n=79)					
Viral hepatitis (B15–B19)	2.89	2.66	Viral hepatitis (B15–B19)	2.66	2.89
Malignant neoplasms of liver and intrahepatic bile ducts (C22)	1.29	2.08	Hypertensive heart disease (I11)	2.29	.0248
Water, air and space, and other transport accidents and their sequelae (V90 V99,Y85)	1.24	0.173	Malignant neoplasms of liver and intrahepatic bile ducts (C22)	2.08	1.29
Motor vehicle accidents (**)	-5.98	-3.27	HIV (B20–B24)	-9.54	-6.00
HIV (B20–B24)	-6.00	-9.54	Chronic ischemic heart disease other than atherosclerotic (I20, I25.1–I25.9)	-11.1	-6.08
Chronic ischemic heart disease other than atherosclerotic (I20, I25.1–I25.9)	-6.08	-11.1	Acute myocardial infarction (I21–I22)	-11.64	-3.66

* Deaths due to accidental poisoning consist primarily of lethal drug overdoses (see Paulozzi and Annett 2007).

** ICD10 codes for motor vehicle accidents are V02–V04,V09.0,V09.2,V12–V14,V19.0–V19.2, V19.4–V19.6,V20–V79,V80.3–V80.5,V81.0–V81.1,V82.0–V82.1,V83–V86, V87.0–V87.8,V88.0–V88.8,V89.0, V89.2

Table 2
Two-Level Hierarchical Linear Model Predicting U.S. Mortality Rates Across of Death from 1999 to 2007

Coeff. Variable	Women				Men			
	White n=90 [†]	Black n=85 [†]	Hispanic n=80 [†]	White n=92 [†]	Black n=86 [†]	Hispanic n=79 [†]		
<i>B</i> ₁₀ Year	-0.0277*** (0.00442)	-0.0579*** (0.00915)	-0.0329*** (0.00691)	-0.0491*** (0.00391)	-0.104*** (0.0101)	-0.0689*** (0.00827)		
<i>B</i> ₂₀ Low education	6.99*** (0.842)	5.22*** (0.608)	1.25*** (0.199)	11.0*** (0.655)	12.4*** (0.632)	2.65*** (0.219)		
<i>B</i> ₃₀ Mid education	2.44*** (0.195)	4.05*** (0.272)	1.76*** (0.123)	4.56*** (0.155)	7.39*** (0.178)	3.94*** (0.202)		
<i>B</i> ₄₀ (Year)*(Low educ)	0.277*** (0.0341)	0.264*** (0.0375)	0.0842*** (0.00973)	0.236*** (0.0332)	0.38*** (0.0370)	0.0958*** (0.0166)		
<i>B</i> ₄₁ mortality rate over study period ^a	0.263*** (0.0312)	0.230*** (0.0462)	0.0502* (0.0209)	0.201*** (0.0222)	0.117*** (0.0235)	0.0223*** (0.00850)		
<i>B</i> ₄₂ Cause-specific mortality rate 2006 ^a	0.109*** (0.0142)	0.0791*** (0.0137)	0.0429*** (0.00844)	0.0566*** (0.00847)	0.0644*** (0.00635)	0.0193*** (0.00595)		
<i>B</i> ₅₀ (Year)*(Mid educ)	0.0376*** (0.00495)	-0.109*** (0.0108)	-0.0725*** (0.0129)	-0.00225 (0.00549)	-0.251*** (0.0167)	-0.205*** (0.0195)		
<i>B</i> ₅₁ mortality rate over study period ^a	0.0892*** (0.00630)	0.100*** (0.0105)	0.0705** (0.0241)	0.0970*** (0.00373)	0.0833*** (0.00845)	0.0891** (0.0292)		
<i>B</i> ₅₂ Cause-specific mortality rate 2006 ^a	0.0190*** (0.00210)	-0.00538* (0.00222)	-0.00920 (0.00499)	0.00677*** (0.00141)	-0.00773** (0.00272)	-0.0275*** (0.00522)		
<i>B</i> ₀₀ Intercept	2.21*** (0.139)	4.21*** (0.198)	1.79*** (0.104)	3.57*** (0.105)	5.93*** (0.152)	3.36*** (0.0993)		

[†] Sample size refers to Level 2, and indicates number of causes of death in the analysis. The typical cause of death contributes 27 observations to the Level 1 analysis pool; each year it contributes a mortality rate for each of the three education groups (low, mid, and high) over a period of nine years (from 1999–2007).

^a Variable grand-mean centered

note: As noted in the full equation in the methods section, the variables “mortality rate over study period” and “cause-specific mortality rate in 2007” are included as predictors of all Level 2 coefficients (including the intercept). This table does not show the full results, and presents coefficients for these variables only as they predict disparity change (i.e. *B*₄₁, *B*₄₂, *B*₅₁, and *B*₅₂). A full listing of all coefficients is available from the authors upon request.

note: The reference group is decedents with high education, defined as those with at least some college education.

note: Analysis pool excludes causes of death that are residual categories, such as “all other diseases.”