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Educational Differentials in U.S. Adult Mortality: Trends and Causes

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Educational Differentials in U.S. Adult Mortality: Trends and Causes

by

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Dissertation

Presented to the Faculty of the Graduate School of

The University of Texas at Austin

in Partial Fulfillment

of the Requirements

for the Degree of

Doctor of Philosophy

The University of Texas at Austin

December 2014

Dedication

To my father, from whom I inherited my curiosity, and to my mother, who taught me to quench it.

Acknowledgements

It is impossible to recount all the individuals and institutions that have supported me during my doctoral studies at the University of Texas at Austin. Yet one person, for whom I will forever be in debt, stands out above all others. Alex Weinreb has been my teacher, mentor, supervisor, colleague, friend, and confidant. He introduced me to the field of demography and accompanied me through every step of my academic training, beginning at the Hebrew University of Jerusalem nearly eight years ago and ending with this dissertation. Alex, being the true intellectual that he is, never inhibited my own intellectual wandering and was always willing to listen to and actively engage with the oddest of ideas. A handful of those ideas actually came to fruition—and that, in the words of Robert Frost, has made all the difference.

I was also lucky enough to work closely with each member of my dissertation committee, in one capacity or another, at the University of Texas at Austin. Deb Umberson first piqued my interest in the sociology of health and gave me the support I needed to grow as a scholar in that field. Deb also had a positive influence on my academic writing and—in more ways than she probably knows—on my philosophy of science. Bob Hummer taught me much of what I know about mortality in the United States, the topic of this dissertation, and his feedback was always insightful and astonishingly meticulous. But the truth of the matter is that Bob was committed to my success long before I became his student. The Talmud says "in a place where there are no people, strive to be a person." For me, Bob Hummer was that person on more than one occasion.

Mark Hayward always challenged me to think harder—often more than I was willing to admit. One out of ten times, I probably gave him a run for his money too. But this dissertation *is* better for it. I consider his graduate seminar and our countless conversations to be some of the most important experiences of my doctoral training. With Dan Powers I share a passion for statistics and all things methodological. In fact, many of the innovative methods adopted and developed in this dissertation were inspired by our hallway conversations over the years. Dan was always able to give me the lead I needed to solve a problem, and, more importantly, showed a genuine interest in how I finally got there. Last but not least, Shripad Tuljapurkar (Tulja) from Stanford University provided excellent comments, both in terms of breadth and depth, which guided me in the right direction at several crossroads.

I also wish to thank some of my fellow students at the Population Research Center, with whom I shared many ideas and benefited from their feedback and support over the years: Dustin Brown, Chi-Tsun Chiu, Joseph Lariscy, David McClendon, and many additional members, past and present, of the weekly "health lab" meetings. Thank you all.

Educational Differentials in U.S. Adult Mortality: Trends and Causes

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The University of Texas at Austin, 2014

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As life expectancy at birth in the United States approaches eighty years of age, educational differentials in adult mortality are greater than ever. One of the key sociological insights of our time is that these two processes are fundamentally interrelated. As society gains greater social capacity to control health and disease socioeconomic status (SES) becomes increasingly important for shaping healthy social environments and lifestyles, which reduce the risk of mortality. Of all SES indicators, educational attainment is perhaps the single most important predictor of mortality in the United States. Not only do low-educated Americans have shorter lifespans compared to their college-educated counterparts, on average, but they have recently suffered absolute declines in life expectancy. However, debates surrounding the extent, causes, and even validity of those trends continue. This dissertation makes several unique contributions to our understanding of lifespan inequality by educational attainment in the United States. First, using vital statistics data, it documents trends in life expectancy and lifespan variation—a unique dimension of lifespan inequality—by educational attainment for black and white Americans of both genders from 1990 to 2010. Second, it decomposes those trends by age and cause of death in order to understand the proximate causes of the educational disparity in adult mortality. Third, it evaluates the extent to which changes in the composition of education groups account for the rising education-mortality gradient.

The findings reveal that the gap in life expectancy at age 25 between the low educated (having fewer than twelve years of schooling) and the college educated has doubled among men and more than tripled among women over the study period; that life expectancy declined among low-educated white men and women (by 0.6 and 3.1 years, respectively); and that much of these trends is attributed to an increase in premature deaths from smoking-related diseases and external causes. While both sides of the selection-causation debate have merit, changes in group composition do not fully account for the increase in mortality among low-educated Americans, for whom economic circumstances have worsened. Overall, the association between educational attainment and adult mortality is pervasive, enduring, and increasing in magnitude.

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Chapter 1: Introduction

> *"...yet bread [is not] to the wise, nor yet riches to men of understanding, nor yet favour to men of skill; but time and chance happeneth to them all. For man also knoweth not his time."* (Ecclesiastes 9, 11-12)

The socioeconomic gradient in longevity is perhaps the most fundamental of social inequalities, and educational attainment is the most profound predictor of longevity of all socioeconomic indicators (Elo 2009). Since the 1960s, educational differentials in adult mortality have widened dramatically in the United States (Meara, Richards, and Cutler 2008; Montez et al. 2011; Preston and Elo 1995). College-educated, non-Hispanic white men can now expect to live about 13 more years, on average, than their low-educated counterparts (having fewer than 12 years of schooling); the same gap among women is estimated at 10.5 years; and a similar, albeit smaller, education gap in life expectancy exists among blacks and Hispanics (Olshansky et al. 2012). The highly educated not only live longer on average, but also exhibit greater mortality compression, which means that their deaths are concentrated in very old age—the most common age at death for women with 13 or more years of schooling is estimated at 90 years (Brown et al. 2012). What may have been true in the time of Ecclesiastes is certainly untrue in our time. In spite of the stochastic nature of human mortality, men and women can in fact "know their time" to an increasing degree of certainty, substituting wisdom for educational attainment, riches for income and wealth, and skill for occupation (Singh and Siahpush 2006).

The increasing educational disparity in U.S. adult mortality may have taken some by surprise, especially given the tremendous improvements in life expectancy in the

population as a whole during the twentieth century. Men's life expectancy at birth increased from 46 years in 1900 to 74 years in 2000, and women's life expectancy increased from 49 to 79 over the same period (Bell and Miller 2005). Why is it that social disparities in longevity are greater than ever at a time when life expectancy at birth in the U.S. is approaching 80? Sociological theory tells us that the simultaneous occurrence of those phenomena is less than coincidental because they are fundamentally interrelated (Freese and Lutfey 2011; Link and Phelan 1995).

Technological advances over the past 300 years brought about the mortality revolution by increasing food supply and caloric intake (Fogel and Costa 1997) as well as improving our capacity for disease control (Cutler and Miller 2005; Preston 1975). However, greater social capacity to control disease and death also meant that individuals' ability to benefit from it was increasingly shaped by knowledge, money, prestige, and social capital (Link and Phelan 2002). Individuals with higher socioeconomic status, whether measured in terms of educational attainment or income, are quicker to adopt medical information and technology (Chang and Lauderdale 2009; Christakis and Fowler 2008; Frisbie et al. 2004; Wise 2003). As a result, the education-mortality gradient is greater in preventable causes of death than in non-preventable ones (Miech et al. 2011; Phelan et al. 2004).

But while theory tells us that educational differentials in adult mortality should increase over time, the absolute declines in life expectancy among low-educated white Americans were truly unexpected. One study found that between 1990 and 2008 life expectancy at age 25 declined by 3.4 years for low-educated men and by 5.3 years for

their women counterparts (Olshansky et al. 2012). In other words, low education groups in the U.S. are not only at a disadvantage relative to high education groups, but are faring worse over time in absolute terms. Yet debates continue about the nature of those declines—and, more generally, about the nature of the association between education and mortality. While some argue that educational attainment has a causal role in shaping health and mortality outcomes (e.g., Mirowsky and Ross 2003; Montez, Hummer, and Hayward 2012), others claim that the association is largely spurious (Behrman et al. 2011).

The selection-causation problem plagues much of the social sciences and particularly research involving processes which unfold over the life course. I first became interested in the topic when working with Debra Umberson on health trajectories following widowhood. Although widowed Americans exhibit worse health outcomes than other marital status groups (Liu and Umberson 2008), they are also more select on a variety of characteristics *prior* to becoming widowed (Sasson and Umberson 2014). In much the same way, low-educated adults are select in several ways even before they complete their educational attainment. For example, intelligence and socioeconomic background predict both academic achievement (Deary et al. 2007; Neisser et al. 1996; Sirin 2005) and adult mortality (Batty et al. 2007; Deary et al. 2008; Hayward and Gorman 2004). Given the expansion of education in the U.S. since WWII, low-educated Americans have likely become more negatively select in a variety of ways, whereas the highly educated have become less positively select. This trend alone might explain the growing educational disparity in health and mortality (Goesling 2007).

My original intention was therefore to address the selection-causation problem via the changing composition of education groups in the U.S. and its effect on health disparities—an idea I developed over two graduate seminars with Mark Hayward and Robert Hummer and countless conversations with Alex Weinreb. They also convinced me to focus my efforts on mortality, which I did, and quickly discovered how many more pieces of the puzzle were missing. Studying mortality differentials the way I had planned meant greater data demands, which could only be met using vital statistics data. However, working with vital statistics data presented several difficulties. Chapter 2 is therefore devoted to addressing those difficulties and documenting trends in the mean and variation in length of life by educational attainment for black and white men and women. In the process, I develop a unique imputation method to handle missing data in the vital registry and improve on previous estimates.

However, the main goal of Chapter 2 is to expand our understanding of lifespan inequality by educational attainment beyond group differences in life expectancy. In particular, lifespan variation measures group heterogeneity (Edwards and Tuljapurkar 2005) and individual uncertainty (Edwards 2013) in length of life, and provides insights into future mortality scenarios for different subpopulations. Daniel Powers (and later Shripad Tuljapurkar) steered me to some of the methods and innovations used in that chapter. Chapter 2, inspired by Brown and colleagues (2012), is also the first study to document trends in lifespan variation by educational attainment over time.

Chapter 3 aims at understanding why life expectancy among low education groups declined since 1990 and why the education-mortality gradient has increased. The

first step toward answering these questions is identifying—in each education category—which age groups have been most vulnerable and to which causes of death. Previous studies have found that for some causes of death mortality declined across all education groups, but more so among the college educated; for other causes of death mortality declined among the college educated but increased among the low educated (Miech et al. 2011; Montez and Zajacova 2013a). However, these studies were limited to specific genders, age groups, or brief periods of time. In addition, they focused on relative differences in mortality rates, which, while important, do not disclose the burden of each cause of death (measured in years of life lost). By contrast, Chapter 3 identifies which causes of death contributed most to the rising education gap in life expectancy from 1990 to 2010 and where policy intervention can be most effective.

Chapter 4 returns to the original research question concerning the selection-causation problem. To what extent are the patterns revealed in Chapters 2 and 3—increasing mortality among low education groups and widening educational disparity in adult mortality—attributed to the changing composition of education groups over time? In other words, is the absolute decline in longevity among low-educated white Americans "real" insofar that their life circumstances have become worse in recent decades? This chapter relies on data from the Integrated Health Interview Series (IHIS) from 1997 to 2013, which mostly overlaps with the period covered in the previous chapters. The IHIS provides higher accuracy in measuring educational attainment as well as information on respondents' life circumstances prior to death, which allows me to evaluate (directly or indirectly) some of the mechanisms purported to explain the association between

educational attainment and adult mortality. This chapter joins a handful of studies addressing the compositional change in education and its effect on observed disparities in adult mortality. Building on those studies, I develop a working definition of the compositional effect and evaluate the extent to which it accounts for the trends revealed in previous chapters.

Finally, Chapter 5 reviews and integrates the findings from Chapters 2-4 and points to their theoretical, methodological, and policy implications. After all the evidence has been weighed, I reflect on the future of U.S. adult mortality and how it might unfold for different education groups in the coming decades. The chapter concludes by identifying promising directions for future research—the most important of which are perhaps the individual and social consequences of rising lifespan inequality.

Chapter 2: Trends in Life Expectancy and Lifespan Variation by Educational Attainment in the United States, 1990-2010

INTRODUCTION

Socioeconomic disparities in health and mortality are perhaps the most fundamental of social inequalities. Educational attainment is a particularly profound predictor of length of life, now surpassing both race (Harper et al. 2007; Kochanek, Arias, and Anderson 2013) and gender (Arias 2007; Rogers et al. 2010) in importance in the United States. More troubling is the fact that educational differences in life expectancy have widened dramatically since the 1980s, across all major race and gender groups, and that low educated white Americans are now seeing absolute declines in longevity (Meara, Richards, and Cutler 2008; Montez et al. 2011; Olshansky et al. 2012). As a result, highly educated Americans live longer on average and exhibit greater compression in old-age mortality, with deaths narrowly concentrated at the upper tail of the age distribution (Brown et al. 2012). Those with less than a high school degree, on the other hand, exhibit higher variability in their lifespan—the length of time an individual lives—and can expect greater uncertainty in their time of death. Educational differences in lifespan variation are similarly evident when measured across a wider age range, capturing premature as well as old-age mortality, both in the U.S. (Edwards and Tuljapurkar 2005) and in multiple European countries (van Raalte et al. 2011).

Building on prior research, this chapter extends the discussion on educational disparities in adult mortality beyond differences in what Cheung and colleagues (2005)

term "central longevity indicators"—mean, median, and modal age at death—to include disparities in lifespan variation. The motivation is twofold. First, it facilitates a more nuanced understanding of lifespan inequality and its underlying causes. Higher lifespan variation reflects both higher uncertainty in the expected time of death, from an individual standpoint, and greater heterogeneity in mortality outcomes from a population perspective (Edwards 2013; Edwards and Tuljapurkar 2005). Population-wise, increasing lifespan variation generally corresponds to a higher share of premature and potentially preventable deaths (van Raalte et al. 2011), net of change in life expectancy, and should therefore be of interest to both mortality researchers and policy makers. Second, documenting trends in both life expectancy and lifespan variation can provide insight into future mortality scenarios of both advantaged and disadvantaged groups at the subnational level. Currently, two key scenarios have been considered for the population as a whole: the compression of adult mortality (Fries 1983) and the shifting mortality regime (Bongaarts 2005; Bongaarts and Feeney 2003; Canudas-Romo 2008). Yet these scenarios have not been evaluated for different socioeconomic strata in the U.S. and particularly over time.

Adopting a multi-dimensional approach to lifespan inequality, this is the first study to reveal trends in both life expectancy *and* lifespan variation by educational attainment, over a two-decade period, for major race and gender[1] groups in the U.S. The findings replicate and revisit important facts concerning recent trends in life expectancy—that educational disparities are widening among all major gender and race groups and that white women (but not men) with fewer than 12 years of education are

increasingly and dramatically worse off in absolute terms. More surprising are the findings concerning trends in lifespan variation. Between 1990 and 2010 variation in age at death increased among high-school educated Americans despite modest gains in life expectancy; compared with the college educated in certain race-gender groups, disparities in lifespan variation have become so important that they now surpass disparities in life expectancy. These patterns defy the common narrative, based on trends in life expectancy alone, that high-school educated Americans are merely lagging behind their college educated counterparts. At the same time, this study finds that those with at least some college education have seen tremendous improvements in life expectancy coupled with steady, record low variation in age at death.

Clearly, documenting distributional differences and trends in mortality between educational attainment groups requires sample sizes which exceed most survey data, especially when sought in repeated cross-section. Mortality data from the National Vital Statistics System remain the single most comprehensive source of information on U.S. mortality, but at the same time suffer from well-known limitations concerning education reporting (Rostron, Boies, and Arias 2010). Therefore, before any empirical investigation is pursued, special attention must be given to missing and potentially misreported information on educational attainment in the vital registry. I develop a unique imputation method to handle missing data in the vital registry, taking into account all available information from observed death records and from the educational composition of the census (at-risk) population.

In summary, I argue that educational differences in lifespan variation constitute a unique dimension of inequality, and, when examined over time along trends in life expectancy, may point to diverging mortality scenarios. This chapter therefore aims to:

1. Document change in life expectancy and lifespan variation in the U.S. from 1990 to 2010 by educational attainment, specific to race and gender subgroups.
2. Examine the relative contribution of differences in means and differences in variances to overall lifespan inequality (i.e., total divergence in the distribution of age at death).
3. Evaluate which mortality scenario best describes college educated Americans, and whether low and high-school educated Americans follow in the former's footsteps or exhibit diverging trajectories in adult mortality.

EDUCATIONAL DISPARITIES IN ADULT MORTALITY

Differences in Central Longevity Indicators

One of the most robust and consistent findings in the social sciences is the association between education and mortality. Highly educated Americans experience significantly lower rates of mortality and, consequently, higher life expectancy than their less educated counterparts (Hummer and Lariscy 2011). A similar gradient is found across multiple high income countries (Baker et al. 2011). Interestingly, when measured in single years, the effect of educational attainment on the risk of mortality shows increasing returns to each additional year of education, and, for the most part, cannot be attributed to credentials alone (Montez, Hummer, and Hayward 2012).

The association between educational attainment and mortality is not only pervasive, but also becoming increasingly pronounced. The educational gradient in U.S. adult mortality appears to be widening over time (Meara, Richards, and Cutler 2008; Montez et al. 2011; Preston and Elo 1995) and across successive birth cohorts (Lauderdale 2001; Lynch 2003; Masters, Hummer, and Powers 2012). Furthermore, these patterns are consistent across all major race and gender groups and across most preventable causes of death (Miech et al. 2011).

Based on data from the National Vital Statistics System, a recent study even suggests that the least educated (non-Hispanic) white Americans have suffered absolute declines in life expectancy since 1990 (Olshansky et al. 2012). According to the study, life expectancy at age 25, e_{25}^{o}, among whites with fewer than 12 years of schooling declined from 47 to 43.6 years for men and from 54.5 to 49.2 years for women between 1990 and 2008. However, evidence from the National Health Interview Survey (NHIS), around the same period, suggests that mortality at ages 45-84 increased only among low educated women, but not men (Montez et al. 2011). With respect to widening disparities between the least (0-11) and most educated (16+), Olshansky and colleagues found that the corresponding difference in e_{25}^{o} increased from 5.1 to 13.2 years among men and from 1.9 to 10.5 years among women—seemingly phenomenal increases over a relatively short period of time. Educational disparities also grew among blacks, albeit to a lesser degree, but no absolute decline was observed among blacks with fewer than 12 years of schooling.

Significant educational differences also exist in the modal, or most common, age at death. Among non-Hispanic whites over the age of 50—of both genders—this difference was estimated at roughly five years between low (0-11) and highly (13+) educated Americans around the mid to late 1990s (Brown et al. 2012). Since the study relied on the NHIS and the Health and Retirement Study, similar estimates for other race-ethnic groups (or more refined educational categories) could not be derived due to small sample sizes.

This study relies on U.S. vital statistics to replicate and revisit results reported by Olshansky and colleagues (2012) concerning recent trends in e_{25}^{o} by educational attainment. Using a slightly modified education categorization and careful imputation of missing data, I find that both trends—widening educational disparities and absolute declines in life expectancy among the low educated—are clearly evident, albeit significantly attenuated based on more refined methodology. In addition to improving on previous estimates from the U.S. vital statistics, I extend this work by documenting corresponding trends in lifespan variation and evaluate their relative importance compared with educational disparities in life expectancy.

Disparities in Lifespan Variation

Although group differences in life expectancy constitute the primary and most commonly documented form of lifespan inequality, scholars have increasingly pushed to explore other dimensions of inequality. Most notably, efforts have been focused on various formulations of lifespan variation (Kannisto 2000; Wilmoth and Horiuchi 1999).

While there is no single, consensual measure of lifespan variation, all seem to agree that low educated groups exhibit larger variation in age at death compared to highly educated groups. Nevertheless, each measure of variation is driven by unique motivations and therefore based on different assumptions.

Edwards and Tuljapurkar (2005) convincingly argued in favor of S_{10}, the standard deviation of age at death conditional on survival to age 10, as a useful measure of lifespan inequality that is distinct from life expectancy. Their study shows that truncating infant and child mortality reveals stark differences in lifespan variation both between and within low mortality countries. In the U.S. alone, using data from the National Longitudinal Mortality Study (centered on 1981; both genders combined), S_{10} differed by more than two years between high-school graduates and those with less than a high school degree (14.6 and 16.7, respectively). Although trends in the standard deviation depend, to some extent, on the age on which S_x is conditioned (Engelman, Canudas-Romo, and Agree 2010), educational disparities in variation are nevertheless evident at older ages and in other low mortality countries. Averaging across ten European countries in the 1990s, van Raalte and colleagues (2011) found educational differences in S_{35} amounting to 1.5 years among men and 1.4 years among women. Higher lifespan variation among the low educated was largely attributed to a greater share of premature deaths due to circulatory diseases, neoplasms, and external causes.

Other scholars (e.g., Brown et al. 2012; Cheung and Robine 2007; Thatcher et al. 2010) chose to focus strictly on lifespan variation in old-age mortality, measured as the standard deviation above the modal age at death, SD(M+). This parameterization is

largely motivated by the compression of mortality toward some hypothetical limit to lifespan—or lack thereof—where deaths above the mode approximately follow a normal distribution (Kannisto 2001; Robine 2011). Unlike S_{10} and S_{35}, SD(M+) emphasizes senescent mortality by eliminating the effect of premature deaths (the left-tail skew). Nevertheless, the highly educated are advantaged regardless of how variation is measured. Among non-Hispanic white Americans of both genders, Brown and colleagues (2012) estimated the difference in SD(M+) between those with 0-11 years of schooling and those with 13+ years to be about two years (estimates vary by survey, centered around the 1990s).

Alternative measures of lifespan variation, including Theil's index, have been used to study the contribution of educational disparities to lifespan variation in the population as a whole (van Raalte et al. 2012). Although Theil's index is particularly sensitive to changes in the lower tail of the distribution (Allison 1978), unlike SD(M+), substantive conclusions are consistent with previous measures—the low educated exhibit greater heterogeneity in age at death. A detailed discussion of measures of lifespan variation and their characteristics can be found elsewhere (van Raalte and Caswell 2013; Wilmoth and Horiuchi 1999).

In this study, I use S_{25} to measure lifespan variation precisely because it captures premature as well as old-age mortality. Conditioning on survival to age 25 ensures that most educational attainment, at least at the college level, is already completed. In addition, I use the Kullback-Leibler divergence (Kullback and Leibler 1951) to evaluate differences across the entire age-at-death distribution and decompose them into

contributions from differences in mean and differences in variance. A formal definition of the latter is presented in the methods section.

WHY VARIATION MATTERS

Heterogeneity and Uncertainty

The importance of reducing educational differences in life expectancy is self-evident. But why is tackling other dimensions of lifespan inequality—and lifespan variation in particular—so important? Variation measures the spread or dispersion of observations around some central value, but at the same time also characterizes a random variable (i.e., an individual sampled from the population) and is therefore closely linked to the concepts of probability and uncertainty. In other words, measures of variation (variance, standard deviation, etc.) play both descriptive and probabilistic roles in the population sciences (Courgeau 2012).

From a population perspective, lower variation in age at death among the highly educated may reflect better access to material and nonmaterial resources (Brown et al. 2012), which in turn facilitate healthier lifestyles and greater ability to shape a healthy environment (Link and Phelan 1995). In the aggregate, greater control over health outcomes culminates in lower dispersion in age at death. By contrast, the low educated exhibit greater heterogeneity in age at death, suggesting perhaps a lower capacity to control their health over the life course. Over time, growing disparities in lifespan variation *between* educational attainment groups indicate the rising importance of education to health and mortality. At the same time, growing variation *within* education

groups may reflect the increasing importance of other social determinants in shaping health outcomes. Factors such as marital stability, parenthood, and psychological wellbeing have an especially protective effect on health among low-educated women—over and above their effect for higher education groups (Dupre and George 2011). If lifespan variation is increasing among low (and perhaps even high-school) educated Americans, then social determinants of health within educational strata warrant special attention.

Considered from an individual (or probabilistic) standpoint, higher lifespan variation also suggests a higher degree of uncertainty in the time of death. Larger values of S_{25} translate into a greater share of deaths occurring further from the expected age at death, either below or above it. All else being equal, an individual drawn from the population at random is less likely to approximate the expected age at death when the variance is larger. The same logic applies to other central longevity indicators, including the mode, as well as to alternative measures of lifespan variation.

Future survival expectations are likely to shape individuals' lifelong decision-making in multiple life domains, and these subjective assessments are in fact predictive of actual survival (Hurd and McGarry 2002). Furthermore, they are correlated with socioeconomic status (Hurd and McGarry 1995), whether because individuals are able to assess their own health status accurately or because they are aware of empirical realities surrounding them. Although demographers have long assumed the latter—the perceived declines in child mortality played a critical role in early demographic transition theory—it is not immediately clear whether and how mortality risk perceptions diffuse in the

population (Montgomery 2000). Nevertheless, if subjective survival assessments are in fact based on socioeconomic strata, then higher lifespan variation translates into higher uncertainty in the accuracy of those expectations. Additional research is required to ascertain the social and economic consequences of such uncertainty from both individual and institutional perspectives (Edwards 2013; Edwards and Tulkapurkar 2005; Wilmoth and Horiuchi 1999).

While disparities in lifespan variation reflect inequality at the population level and differential uncertainty at the individual level, long term trends in variation can also shed light on diverging (or converging) mortality scenarios by educational attainment, specific to gender and race subgroups.

Future Mortality Scenarios

Demographers have long contemplated possible scenarios for the future of mortality (Wilmoth 2000). Some have suggested that low mortality countries will continue to experience adult mortality compression (Fries 1983), particularly in old-age, accompanying gains in life expectancy or the modal age at death. However, since trends in mortality compression appear to have slowed down in recent decades, whether measured via S_{10} (Edwards and Tuljapurkar 2005) or SD(M+) (Oullette and Bourbeau 2011), others suggested an alternative scenario of shifting mortality (Bongaarts 2005; Bongaarts and Feeney 2003; Canudas-Romo 2008). According to the latter scenario, advances in central longevity indicators will continue while lifespan variation remains constant. In other words, adult mortality will continue to decline, with the age-at-death

distribution shifting to older ages while retaining the same shape (Oullette and Bourbeau 2011).

To date, both scenarios have generally been considered for low mortality countries as a whole. It remains to be seen whether country-level mortality trends are equally shared by subnational socioeconomic strata, and, specifically, by various educational attainment groups. Although mortality compression may characterize some groups, a shifting mortality scenario may better describe others. Given recent declines in life expectancy among low educated white Americans (Montez et al. 2011; Olshansky et al. 2012), it is clear that certain groups do not partake in either of those favorable scenarios. Yet several questions remain unanswered. The first relates to the quality of estimates based on U.S. vital statistics. Second, although educational disparities in life expectancy have been studied over time, less is known about trends in disparities in lifespan variation. To date, all such evidence from the U.S. has been survey-based, cross-sectional, and limited to the non-Hispanic white population. Third, temporal trends in lifespan variation can discriminate between competing mortality scenarios among the most socioeconomically advantaged Americans—and suggest new scenarios among less advantaged groups—that are otherwise not captured by trends in life expectancy alone. Most importantly, evaluating mortality scenarios for each educational attainment group on its own will answer whether low and even high-school educated Americans are simply following in the footsteps of the college educated, albeit at a slower pace, or instead are following alternate and diverging trajectories.

METHODOLOGY

Data

The basis for all subsequent analyses begins with age-specific mortality rates, with death counts in the numerator and person-years of exposure in the denominator. All-cause death counts were derived from the U.S. Multiple Cause of Death (MCD) public use files (Centers for Disease Control and Prevention 2013) in select census years—1990, 2000, and 2010—and stratified by age, gender, race, and educational attainment (see Table 2.1). The study period begins in 1990 because educational attainment was not recorded on death certificates nationwide prior to 1989 (National Center for Health Statistics 1993). In the denominator, midyear population estimates were derived from the 5% Integrated Public Use Microdata Sample (Ruggles et al. 2010) in appropriate census (and in 2010, American Community Survey) years. Although education reporting on death certificates suffers from well-known limitations—namely, that educational attainment is reported by someone other than the deceased and therefore is often heaped at 12 years from both lower and higher levels of education (Sorlie and Johnson 1996; Rostron, Boies, and Arias 2010)—it remains the single most comprehensive data source on U.S. mortality. This is especially true given the data requirements for documenting trends in disparities across the entire adult age-at-death distribution.

In both the numerator and denominator data sources, age was recoded to 5-year groups starting at 25-29 and ending with an open interval at 90+. Race was categorized as non-Hispanic white and black, excluding other race categories and persons of Hispanic origin due to small death counts or poor reporting (especially in the 1990 MCD). Since

the 2000 and 2010 censuses allowed for multiple-race categorization, whereas vital statistics continue to follow single-race categorization, counts from the former were adjusted to match the National Center for Health Statistics' bridged-race population estimates in respective years (National Center for Health Statistics 2003, 2011). Nationally, race-bridging appears to have only a minor impact on white and black population estimates, adding as much as 0.5 and 2.5 percent to single race counts, respectively, in the 2000 census (Ingram et al. 2003).

Educational attainment in the MCD (numerator) data is classified in single years ranging from zero to five or more years of college. However, in 2003 educational attainment was reclassified on death certificates from single years to completed degrees. Thirty four states and the District of Columbia adopted the new classification system by 2010, with the remaining 16 states using the old classification (Murphy, Xu, and Kochanek 2013). In order to maintain consistency over time, I translated degree categories into completed years of schooling. Most importantly, the new classification collapses "12th grade, no diploma" with the 9-12 years category, leaving high-school graduates and GED holders in a separate category. Ignoring the change in classification overestimates the number of deaths among the least educated while undercounting deaths in the 12 years category. Therefore, for 2010, I reallocated deaths in the 9-12 years group to 0-11 and 12 years proportional to their relative size in the at-risk population (using the 2010 American Community Survey) by age, gender, and race. This is likely a conservative approach which accounts for departures from previously published estimates.

Next, I recoded educational attainment in the census (denominator) data to match the MCD classification of completed years of schooling (0-11; 12; 13-15; 16+). All categories below 12 years were collapsed and recoded as 0-11. Those with more than one year of college education or an Associate's degree were classified as 13-15, and those with a Bachelor's degree or higher placed in the 16+ category. Finally, all those who reported completing the 12th grade (with or without diploma), obtaining a general equivalency degree (GED), or completing "some college credit, but less than one year" were coded as 12 years. Given that those with some college education but no degree are significantly more likely to be reported as high-school graduates on death certificates (Rostron, Boies, and Arias 2010), and probably more so if they had completed less than a full year of college, I included them in the 12 years category (consistent with *completed* years reported in the MCD).[2] This classification aims to reduce non-sampling error due to education misreporting; it also departs from the categorization used by Olshansky and colleagues (2012) and explains much of the discrepancy in our estimates. In effect, it serves to inflate the denominator for the 12 years category and hence reduce mortality rates for that group at the expense of the 13-15 category.

Missing Data Imputation

The MCD suffers from a significant amount of missing data on educational attainment and, to a lesser extent, on Hispanic origin. In 1990, seven states did not report educational attainment on death certificates at all and the remaining states had an average of 10.0 percent missing information. By 2000, only three states failed to report altogether and the level of missing data among all other states declined to an average of 4.0 percent.

Information on state of occurrence is absent from the 2010 MCD public use file, but by then all states reported (some version) of educational attainment and missing information declined to 2.3 percent nationally. Similarly, three states neglected to report Hispanic origin in 1990, with reporting improving dramatically in 2000 and 2010. Since I exclude Hispanics from all subsequent analyses, the imputation of Hispanic origin serves only to allocate unclassified deaths to non-Hispanic groups. This number amounts to nearly 100,000 deaths in 1990 (most attributed to the three states and New York City failing to report), and is particularly important to include in estimating mortality rates where the numerator and denominator are unlinked.

Imputation of Hispanic origin in the MCD was based on the proportion of non-Hispanics in the census population by gender, race, age, and, where missingness was especially high, by state of occurrence.[3] Since Hispanics represent a small minority among older U.S. cohorts, imputation is unlikely to jeopardize results for the non-Hispanic majority. On the other hand, estimates *for* Hispanics would be greatly impacted by underreporting and are therefore not pursued in this study.

Although educational attainment was missing only in the numerator (death counts) and not the denominator (midyear population estimates), both sources can in fact provide useful information in imputing the former. Bayes theorem makes this relationship clear:

$$p(\text{Education}|\text{Death}) = p(\text{Death}|\text{Education}) \times p(\text{Education}) \times \frac{1}{p(\text{Death})} \qquad (2.1)$$

Such that the distribution of education in the vital registry, on the left-hand side of the equation, depends on three terms on the right-hand side: (a) the probability of mortality

conditional on educational attainment; (b) the distribution of education in the at-risk population; and (c) the distribution of overall mortality. Although Equation 2.1 describes a mathematical identity, it can be used as an imputation device if each component is estimated separately. Since $p(\text{Education})$ depends only on the at-risk population and $p(\text{Death})$ does not depend on education, both can be estimated from fully observed information by gender, race, age, and state of occurrence.[4] Clearly, the term $p(\text{Death}|\text{Education})$ also depends on missing information and instead was estimated only from states with nearly complete data—less than 10 percent missing—the convention followed by official National Center for Health Statistics publications (National Center for Health Statistics 1993). Educational attainment was then drawn randomly by gender, race, age, and state of occurrence using the estimated probabilities.[5] The final step was repeated ten times with death counts averaged across repetitions (although differences between repetitions were negligible).

This imputation approach was deemed preferable to other methods[6] because it maximizes the use of available information from both the numerator and denominator. In effect, it assumes that the relationship between education and mortality is equivalent among observed and unobserved cases, *weighted by the educational composition and overall level of mortality in each state of occurrence*—a strategy that is particularly useful in states with high proportions missing.

Methods

Once all relevant missing data were imputed, age-gender-race-education specific mortality rates were estimated and used as input for standard period life tables. In order to derive exact age-at-death distributions, 5-year log mortality rates were interpolated to single years using natural cubic splines (Berk 2008) with *n-2* knots, where *n* is the number of life-table age groups (excluding the open interval). This was to ensure minimal departure from observed data, as a log-linear (e.g., Gompertz) model might impose, yielding e^o_{25} estimates that are comparable to the original 5-year life tables. The smoothed age-at-death distributions (see Appendix A), equivalent to the d_x column in life table notation, were the basis for all subsequent calculations (e^o_{25}, S_{25}, and distributional divergence).

Measuring differences across the entire adult age-at-death distribution is less intuitive than comparing particular moments, such as life expectancy or variance. Perhaps the most common measure of distributional divergence, stemming from information theory (Shannon 1948), is the Kullback-Leibler divergence (KLD) (Kullback and Leibler 1951). It takes the form

$$KLD(p_0, p_1) = \int_{-\infty}^{\infty} p_0(x) log\left(\frac{p_0(x)}{p_1(x)}\right) dx \qquad (2.2)$$

where p_0 is the probability function of the reference group and p_1 is the probability function of the comparison group.[7] The KLD exhibits several useful properties. First, it is non-negative, with larger values indicating a higher degree of divergence. Two identical distributions will have a KLD value of zero. Second, if both distributions are normal,

then the KLD can be decomposed precisely into two additive components (Roberts and Penny 2002) such that

$$KLD(p_0, p_1) = \underbrace{\left[log\left(\frac{\sigma_1}{\sigma_0}\right) + \frac{\sigma_0^2}{2\sigma_1^2} - \frac{1}{2}\right]}_{\text{A}} + \underbrace{\left[\frac{(\mu_0 - \mu_1)^2}{2\sigma_1^2}\right]}_{\text{B}} \quad (2.3)$$

Term A sums to zero when the variances are equal, leaving contributions from the difference in means (term B) alone. Similarly, when the means are equal, term B equals zero and any remaining divergence is attributed to difference in variances (term A). Although age-at-death distributions are generally left-skewed, Edwards and Tuljapurkar (2005) suggest that Equation 2.3 remains a useful approximation.

The KLD not only provides insights into educational disparities in mortality across the entire adult age-at-death distribution, but can also tell us about the relative importance of each component—difference in means and difference in variances—to overall lifespan inequality. This is especially revealing if observed over time and can be suggestive of future mortality trends both within and between educational attainment groups. However, since the KLD does not indicate the direction of distributional divergence (e.g., which group exhibits lower or higher variance), trends in life expectancy and lifespan variation must be established beforehand.

RESULTS

Educational Differences in Life Expectancy

Life expectancy at age 25 by race, gender, and educational attainment is shown in Table 2.2. Overall, e_{25}^{o} increased by 1.5 years among non-Hispanic white women and by

3.2 years among white men between 1990 and 2010. Gains were higher among non-Hispanic blacks, amounting to 3.2 among women and 6.3 years among men.[8] Nevertheless, higher gains among blacks reflect a significantly lower starting point relative to whites of the same gender—a gap that was maintained, albeit diminished, over the twenty-year period. Consistent with the gender gap in life expectancy, women fare better than men in each race group.

Several notable patterns arise when broken down by educational attainment. First, educational disparities in life expectancy grew among all race and gender groups from 1990 to 2010. The difference in e_{25}^{o} between the most (16+) and least (0-11) educated white women grew from 2.5 to 9.3 years; a smaller increase was observed among black women, from 1.9 to 4.7 years. Educational differences in e_{25}^{o} are generally larger among men, and they too experienced widening disparities: from 6.1 to 11.9 years among whites and from 6.9 to 8.6 years among blacks. While educational disparities in mortality are still wider among men, women are slowly catching up.

Second, absolute declines in e_{25}^{o} were observed among the least educated white men and women, but not among blacks. Within two decades e_{25}^{o} declined by 3.1 years among white women and by 0.6 years among white men. At the same time, all other groups experienced gains in life expectancy. Life expectancy among the least educated black men and women increased by 5.9 and 1.9 years, respectively. Overall, in each race-gender group, the highest gains were experienced by those with 13-15 or 16+ years of education, whereas only modest gains occurred among those with 12 years of education (especially white men and women).

These patterns are generally consistent with those reported by Olshansky and colleagues (2012). However, using the current methodology, the declining trend in life expectancy is significantly attenuated for white women and disappears almost completely for white men. On the whole, the findings suggest that total gains in life expectancy are primarily driven by highly educated Americans, whereas the high-school educated lag behind and the low educated, at least among non-Hispanic whites, are increasingly worse off.

Educational Differences in Lifespan Variation

Patterns and trends in life expectancy by educational attainment are revealing, but they do not tell the whole story. Additional insights can be gleaned by exploring educational differences in lifespan variation. Trends in the standard deviation of age at death, conditional on survival to age 25, are summarized in Table 2.3. Overall, race-gender patterns mimic those of life expectancy in reverse. Higher variation is characteristic of men compared to women and blacks compared to whites. Between 1990 and 2010, S_{25} among white women plateaued around 13.2 years and increased only slightly among white men, from 14.1 to 14.5 years. The opposite was recorded among blacks—S_{25} declined from 16.4 to 15.5 years for men and from 15.7 to 14.8 years for women. This suggests that blacks, as a group, are becoming increasingly homogeneous with respect to age at death, but are nevertheless disadvantaged compared to whites of the same gender.

As with life expectancy, disaggregating trends in S_{25} by educational attainment reveals significant disparities. Across the board, those with higher levels of educational

attainment benefit from lower lifespan variation. This finding suggests that the highly educated are not only advantaged with respect to the expected age at death, but also benefit from lower dispersion or less uncertainty around that central indicator. In 2010, differences in S_{25} between the least and most educated amounted to over four years among men and about five years among women, regardless of race. In other words, although educational differences in life expectancy are greater among men, differences in variation are greater among women. Furthermore, within each race group, low educated men and women are now more similar to one another than are the highly educated.

For some groups, S_{25} has seen little to no change over time. For others, S_{25} changed dramatically over the two-decade period. The least (0-11) and most (16+) educated black men each experienced a decline of 0.8 and 1.1 years, respectively, suggesting more favorable mortality conditions (i.e., lower spread around e^{o}_{25}, which was also on the rise during this period). On the other hand, several groups experienced a significant increase in variation—almost all of which occurred among the low and high-school educated. In particular, S_{25} increased by 1.4 to 1.5 years among low educated white women and among high-school educated whites of both genders. The fact that lifespan variation increased mostly among the high-school educated is disconcerting and counters conventional wisdom based on trends in life expectancy alone (i.e., that the high-school educated merely lag behind the college educated). Only by observing differences and trends in lifespan variation do we notice diverging trajectories for the two groups. Interestingly, aside from black men who were severely disadvantaged at baseline, the college educated did not exhibit significant declines in S_{25}, which is consistent with a

shifting mortality scenario rather than further compression. At the same time, the persistence of racial differences in lifespan variation even among the highly educated suggests that further improvements are in fact clearly possible, at least among blacks.

Documenting changes in lifespan variation complements the picture painted by trends in life expectancy. However, an important question remains: what is the relative importance of each component to overall lifespan inequality? Decomposing the Kullback-Leibler divergence provides an approximate answer.

Convergence and Divergence in Age-at-death Distributions

The KLD is a unitless quantity and, hence, not as easily interpretable as e_{25}^{o} and S_{25}. Nevertheless, it exhibits two important advantages. First, the KLD allows a complete comparison between age-at-death distributions and is not limited to a single dimension of inequality, such as differences in life expectancy. Observed over time, it indicates patterns of convergence or divergence in age-at-death distributions between various subpopulations and can shed light on possible future scenarios. Second, it decomposes (approximately) into two additive terms reflecting the relative importance of differences in means and differences in variances in explaining overall lifespan inequality.

Figures 2.1 and 2.2 depict trends in KLD and its components for white and black women, respectively, by educational attainment. To facilitate comparison across different educational attainment groups, all use the same reference distribution—white women with 16+ years of education in 2010, the group with the highest life expectancy and lowest variance in age at death. As of 2010, this group represents the most favorable

lifespan conditions for women of either race. Similarly, Figures 2.3 and 2.4 show equivalent trends among white and black men, using white *men* with 16+ years of education, in 2010, as reference.

Consistent with the patterns observed in e_{25}^{o} and S_{25}, Figure 2.1 shows that low educated white women have continued to diverge from the reference distribution, representing gender-specific optimal lifespan conditions, throughout the 1990s and 2000s. During this period differences in both life expectancy and lifespan variation have contributed significantly to the overall disadvantage, and both components of inequality have been on the rise. Trends among the high-school educated are even more peculiar—whereas differences in life expectancy had declined from 1990 to 2010, differences in variation had increased (especially during the 2000s). In fact, the latter were so pronounced that they now overshadow differences in life expectancy, resulting in net divergence from the reference distribution over time. In other words, lifespan variation is not only increasing in absolute terms, but has now become more important in explaining lifespan disparities between high school and college educated white women. Clearly, the shifting mortality scenario does not characterize all educational attainment groups, let alone the low and even high-school educated. Finally, those with some college education appear to follow in the footsteps of the highly educated, which, by design, converge to the reference distribution in 2010.

Figure 2.2 presents results for black women. Similar to whites, lifespan variation contributes more significantly to total divergence among lower education groups—as much as half in the case of women with 0-11 years of education. But unlike their white

counterparts, all black women converged dramatically toward the reference distribution, primarily due to improvements in life expectancy. Lifespan variation, on the other hand, appears to have plateaued or decreased slightly among all but the high-school educated.

Figures 2.3 and 2.4 suggest that lifespan variation plays a smaller role in explaining overall disparities among men compared to women. In Figure 2.3, all but the least educated white men have seen overall improvements in lifespan, with age-at-death distributions converging to that of college-educated men in 2010. Low educated men, however, diverged from the reference distribution during the 1990s and plateaued during the 2000s. Although differences in means dominated over differences in variation in all educational attainment groups, the latter made a significant contribution to overall lifespan inequality among the low and high-school educated. Rising lifespan variation among high-school educated men—as is the rise in its relative importance—counter the notion that they merely lag behind their college educated counterparts, as one might conclude from trends in life expectancy alone. Instead, they are becoming increasingly diverse in their time of death, and, at least in this respect, more dissimilar to the college educated (who experienced continued improvements in life expectancy while lifespan variation remained steady at an all-time low).

Educational disparities in lifespan are significantly larger among black men (Figure 2.4) than in white men (notice the scale change on the vertical axis). Nevertheless, all educational attainment groups appear to have made progress toward the reference distribution over time due to gains in life expectancy, with some decline in variation as well among the least and most educated. As with white men, lifespan

variation contributes significantly to overall inequality among the low educated—but not as much as it does among women of either race group.

In summary, trends in KLD suggest that all but the least educated white men and women have seen absolute improvements in lifespan, converging in distribution to college-educated whites in their respective gender group. At the same time, a troubling pattern emerges. While high-school educated Americans in all race-gender groups have seen gains in life expectancy, most have also seen increasing heterogeneity in age at death (with the exception of black men, whose initial lifespan variation was highest and remained steady during the two-decade period). Most importantly, lifespan variation is becoming an increasingly important component of lifespan inequality among the high-school educated—often approaching and even surpassing contributions from differences in life expectancy. Focusing on mean-differences alone misses those trends and fosters a false set of suppositions about the future of mortality—that high-school educated Americans, if not the least-educated, are simply lagging behind those with college education. Educational disparities can therefore point to diverging scenarios in adult mortality.

DISCUSSION

Educational disparities in U.S. adult mortality have been on the rise for several decades (Meara, Richards, and Cutler 2008; Montez et al. 2011; Preston and Elo 1995), with the college educated faring better than any other educational attainment group. Most recently, absolute declines in life expectancy have been observed among low educated white Americans (Olshansky et al. 2012)—results which are replicated and updated in the

present study. Nevertheless, focusing on life expectancy, a central longevity indicator, overlooks other dimensions of lifespan inequality. Lifespan variation is a particularly revealing dimension of inequality reflecting uncertainty in the time of death, from an individual standpoint (Edwards 2013; Wilmoth and Horiuchi 1999), but also heterogeneity in mortality outcomes both within and between social groups (Edwards and Tuljapurkar 2005). Evidence from multiple European countries suggests that higher lifespan variation among the low educated is driven by a higher incidence of causes of premature death (van Raalte et al. 2011), rather than the same causes of death occurring earlier in life. The same pattern is likely true in the U.S., where widening educational disparities are evident in major causes of premature death (cancer, heart disease, stroke, diabetes, and accidents) in multiple reporting states and in both genders (Ma et al. 2012). To the extent that these deaths are preventable, higher lifespan variation among low educated individuals may reflect the lack of material and nonmaterial resources needed to adopt healthier lifestyles and shape healthier social environments, and in turn maximize longevity (Brown et al. 2012).

Using vital statistics data, this study is the first to document trends in both life expectancy and lifespan variation, over a two-decade period, by educational attainment for subgroups of the U.S. populations. With respect to life expectancy, the findings improve and update previous estimates, showing that educational disparities have indeed increased for white and black men women since 1990. By 2010, differences in life expectancy at age 25 between those with fewer than 12 years of schooling and those with 16 years or more amounted to 11.9 years among white men and 9.3 years among women.

Among blacks, the equivalent figures were estimated at 8.6 and 4.7 years. The present study also reevaluates recent declines in e_{25}^{o} among low educated white Americans. Although absolute declines are still evident between 1990 and 2010, they are dramatically smaller than previously reported for women (3.1 years) and disappear almost completely for men (0.6 years)—consistent with evidence from the NHIS around the same period (Montez et al. 2011). Departures from previous estimates based on vital statistics data (Olshansky et al. 2012) reflect differences in education categorization and a newly developed imputation method to handle missing data.

Trends in lifespan variation complement those in life expectancy and have several immediate implications. First, there are large educational differences in S_{25}, the standard deviation of age at death conditional on survival to age 25: they are estimated to be between 4.0 and 5.5 years in all race-gender groups. Second, although educational differences in e_{25}^{o} are greater among men, differences in S_{25} are greater among women and are equally important as the former in explaining overall lifespan inequality. Interestingly, differences in S_{25} are also larger among whites compared to blacks, regardless of gender. Third, absolute increases in S_{25}, amounting to about 1.5 years, have been observed among low *and* high-school educated whites of both genders (but not among blacks). Fourth, declines in S_{25} among college educated whites, representing the most advantaged socioeconomic strata, have been minor at best.

Taken together, the findings suggest that low educated Americans are disadvantaged not only in life expectancy, but also exhibit greater heterogeneity in age at death. Furthermore, lifespan variation is becoming increasingly important in accounting

for overall lifespan inequality between educational attainment groups—especially when comparing high-school educated Americans with their college educated counterparts. For white women, differences in S_{25} are now surpassing those in life expectancy in their relative importance. Neglecting to account for disparities in lifespan variation may therefore lead to the false conclusion that high-school educated Americans are merely lagging behind the college educated. In fact, the former are becoming increasingly diverse in their mortality outcomes despite recent gains in life expectancy. Considering total lifespan disparities, using a multi-dimensional approach to inequality, reveals diverging educational trajectories in adult mortality that are otherwise not captured by mean-differences alone.

Documenting patterns in lifespan variation is important for two reasons. First, they enrich our understanding and characterization of lifespan inequality between socioeconomic strata. From a population perspective, higher and increasing lifespan variation among low and high-school educated Americans can point to a higher prevalence of frailty (Tuljapurkar and Edwards 2011), lower capacity to optimize health over the life course (Brown et al. 2012), or the rising importance of additional social determinants of health operating *within* educational attainment groups (Dupre and George 2011).

Second, trends in lifespan variation can shed light on future mortality scenarios for different social groups. Researchers have long debated which scenario best describes the future of mortality for low mortality countries (Canudas-Romo 2008)—adult mortality compression, whereby gains in life expectancy are accompanied by decreasing

lifespan variation, or shifting mortality, whereby the age-at-death distribution is merely translated upwards while the shape of the distribution remains the same (i.e., constant variation). The findings in this study suggest that scenarios differ dramatically across socioeconomic strata when broken down by educational attainment. For example, among whites, the college-educated appear to follow a shifting mortality scenario where nearly all changes to the age-at-death distribution reflect a constant rise in life expectancy and little change in variation. At the other extreme, worsening conditions among low-educated whites are attributed primarily to absolute declines in life expectancy. Finally, high-school educated whites exhibit a very unique pattern, whereby gains in life expectancy are coupled with increasing lifespan variation. Such trends bear important implications for modeling and forecasting mortality because "subgroup differences in the variance in length of life are equivalent to subgroup differences in the age slope of mortality" (Tuljapurkar and Edwards 2011, 498) in standard modeling practices (e.g., Gompertz, logistic, and Cox proportional hazard models).

While findings in this study are illuminating, several limitations should be acknowledged. First, despite careful attention to data imputation and significant efforts to maintain consistent classification of educational attainment over time, mortality records in the National Vital Statistics System are known for their inaccuracy. Evidence from other countries suggests that census-unlinked mortality estimates tend to overestimate educational disparities (Shkolnikov et al. 2007). In the U.S., heaping educational attainment on death certificates at the high-school level is especially troublesome (Rostron, Boies, and Arias 2010). The assumptions made here have generally been

conservative yet realistic, maximizing all available information from both mortality records and census data, so as not to inflate disparities. Second, distributional differences measured via the Kullback-Leibler divergence are only an approximation based on the normality assumption. An exact decomposition into contributions from mean, variance, and residual shape differences to overall lifespan inequality is a possible extension (e.g., Handcock and Morris 1999), though not as straightforward, and can be sought in future research. Third, trends in life expectancy and lifespan variation are based on period life tables. However, growing educational disparities in mortality during the study period have been driven by cohort effects (Masters et al. 2012), which are also more consistent with the individual experience of disadvantage (and uncertainty) from a life course perspective (Dannefer 2003). Unfortunately, data requirements to estimate long term cohort mortality are overwhelming—especially by educational attainment.

In spite of those limitations, a discussion of educational disparities in mortality should not be limited to differences in life expectancy alone. Instead, a multi-dimensional approach to lifespan inequality can reveal more complex forms of convergence and divergence in age-at-death distributions over time. The most surprising result in this study is that, since 1990, high-school educated Americans have been following a mortality scenario that is distinct from all other educational attainment groups. Despite continued gains in life expectancy, they are becoming increasingly variable in age at death. It remains to be seen how increasing within-group heterogeneity corresponds to trends in age- and cause-specific mortality rates.

The source of widening educational disparities has been a topic of continued debate. Some have suggested that it reflects the causal association between education and mortality, where widening disparities are due to differential access to information and resources among the low educated (Link and Phelan 1995). Others concluded that the association between education and mortality is largely due to selection on individual and family endowments preceding educational attainment (Behrman et al. 2011). In either case, increasing lifespan variation among the low and high-school educated is part of the story—whether suggestive of increasing frailty due to selection mechanisms or indicative of increasing within-group diversity on a variety of other social factors. Future studies should therefore focus on determinants and consequences of heterogeneity within, in addition to between, educational attainment groups.

Notes:

1. Throughout the chapter I use the terms men and women (gender categories) rather than male and female (sex categories) because differences in longevity between these groups are due to a combination of biological and social factors (Bird and Rieker 2008). However, the U.S. census form and standard death certificate both inquire specifically about sex (which, in the latter case, cannot be self-reported).

2. Importantly, the 1990 census did not distinguish between those with "some college credit, but less than one year" and "1 or more years of college, no degree" as did later years. Hence, in 1990, all those with "some college education but no degree" were reallocated to "12 years" and "13-15" based on the relative proportions by gender and race in the 2000 census.

3. In the state of New York, in 1990, missing Hispanic origins on death certificates were primarily due to non-reporting by New York City and were imputed from metropolitan area, rather than state-level, statistics.

4. Since state of occurrence is not available in the 2010 MCD public use file, $p(\text{Education}|\text{Death})$ was estimated directly from observed death records assuming data are missing at random. In 1990 and 2000, in states with sparse black population, $p(\text{Death})$ was estimated using the weighted regression model

$$\log(m_{ipqr}) = \sum_i \alpha_i(\text{State}_i) + \sum_p \sum_q \sum_r \beta_{pqr}(\text{Race}_p \times \text{Gender}_q \times \text{Age}_r) + \varepsilon_{ipqr}$$

   where $\varepsilon_{ipqr} \sim N\left(0, \frac{\sigma^2}{N_{ipqr}}\right)$ and N_{ipqr} is the population size in the respective state, race, gender, and age group.

5. Since the estimated probabilities do not exactly sum to unity, they must be rescaled proportionally.

6. Other methods include imputation based solely on the at-risk population (denominator), solely on deaths records (numerator), or excluding states with incomplete data altogether.

7. Since the KLD is non-symmetric—i.e., $KLD(p_0, p_1) \neq KLD(p_1, p_0)$ in general—the reference distribution, p_0, should be consistent when comparing multiple distributions.

8. Hereafter, non-Hispanic white and non-Hispanic black are referred to as simply white and black, respectively.

Table 2.1: Death counts by race, gender, and years of schooling, United States 1990-2010

		Non-Hispanic White		Non-Hispanic Black	
Year	**Education**	**Women**	**Men**	**Women**	**Men**
1990	0-11	338,760	342,673	60,615	70,700
	12	338,761	313,921	34,606	41,168
	13-15	97,574	98,480	8,582	10,086
	16+	78,695	116,707	6,847	6,702
	Total	**853,790**	**871,781**	**110,650**	**128,656**
2000	0-11	314,706	274,279	58,963	57,741
	12	443,496	367,756	49,115	52,230
	13-15	137,133	126,957	14,872	13,589
	16+	106,417	151,847	10,459	9,131
	Total	**1,001,752**	**920,839**	**133,409**	**132,691**
2010	0-11	203,629	187,152	42,692	42,460
	12	490,761	411,653	55,776	60,979
	13-15	168,322	166,027	22,766	19,857
	16+	124,296	186,733	13,484	11,301
	Total	**987,008**	**951,565**	**134,718**	**134,597**

Table 2.2: Life expectancy at age 25 by race, gender, and years of schooling, United States 1990-2010

	White Women			White Men		
Education	**1990**	**2000**	**2010**	**1990**	**2000**	**2010**
0-11	54.0	51.5	50.9	46.0	45.2	45.4
12	55.1	55.6	55.9	48.7	50.0	50.5
13-15	55.2	56.0	56.7	49.7	52.3	52.8
16+	56.5	58.7	60.2	52.1	54.9	57.3
Total	**55.4**	**55.8**	**56.9**	**49.3**	**51.1**	**52.5**

	Black Women			Black Men		
Education	**1990**	**2000**	**2010**	**1990**	**2000**	**2010**
0-11	49.9	49.5	51.8	39.6	42.2	45.5
12	49.2	50.4	52.7	41.4	43.9	46.5
13-15	49.6	51.6	54.2	43.1	48.6	50.9
16+	51.8	54.5	56.5	46.5	50.4	54.1
Total	**50.8**	**51.6**	**54.0**	**42.2**	**45.3**	**48.5**

Note: Excluding whites and blacks of Hispanic origin.

Table 2.3: Adult lifespan variation (S_{25}) by race, gender, and years of schooling, United States 1990-2010

	White Women			White Men		
Education	**1990**	**2000**	**2010**	**1990**	**2000**	**2010**
0-11	15.4	16.1	16.8	16.5	16.5	16.7
12	12.9	13.3	14.4	14.1	14.6	15.5
13-15	12.5	12.4	12.9	13.6	13.4	14.0
16+	11.7	11.9	11.3	12.5	12.2	12.2
Total	**13.2**	**13.0**	**13.3**	**14.1**	**13.9**	**14.5**

	Black Women			Black Men		
Education	**1990**	**2000**	**2010**	**1990**	**2000**	**2010**
0-11	17.8	17.6	17.2	18.1	17.2	17.3
12	14.8	14.9	15.3	15.4	15.2	15.4
13-15	14.1	14.1	13.9	14.8	15.0	14.7
16+	12.9	14.0	12.5	14.4	14.3	13.3
Total	**15.7**	**15.3**	**14.8**	**16.4**	**15.6**	**15.5**

Note: Excluding whites and blacks of Hispanic origin.

Figure 2.1: Divergence and convergence in age-at-death distribution by educational attainment, non-Hispanic white women (reference: non-Hispanic white women with 16+ years of schooling in 2010)

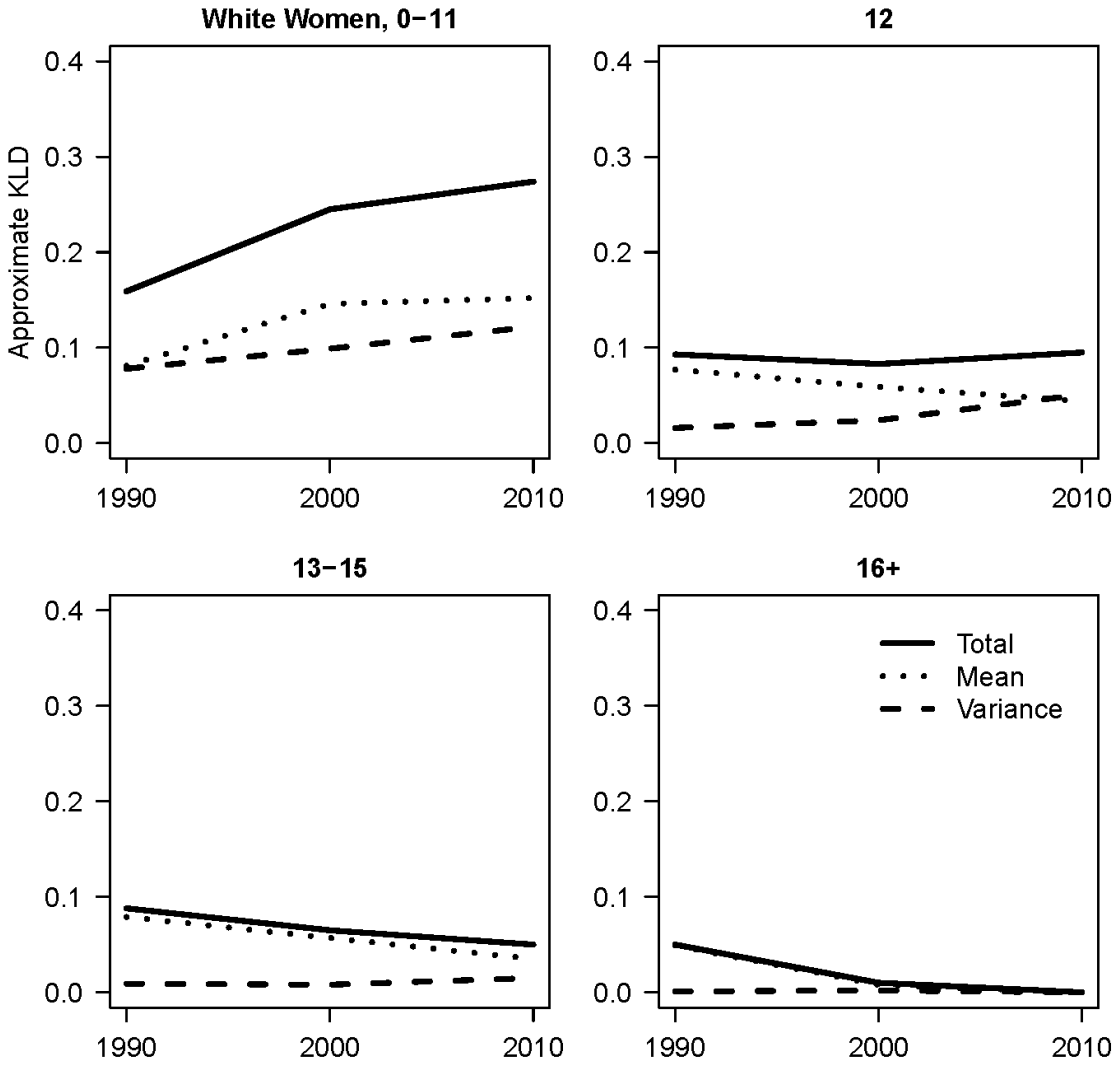


Figure 2.2: Divergence and convergence in age-at-death distribution by educational attainment, non-Hispanic black women (reference: non-Hispanic white women with 16+ years of schooling in 2010)

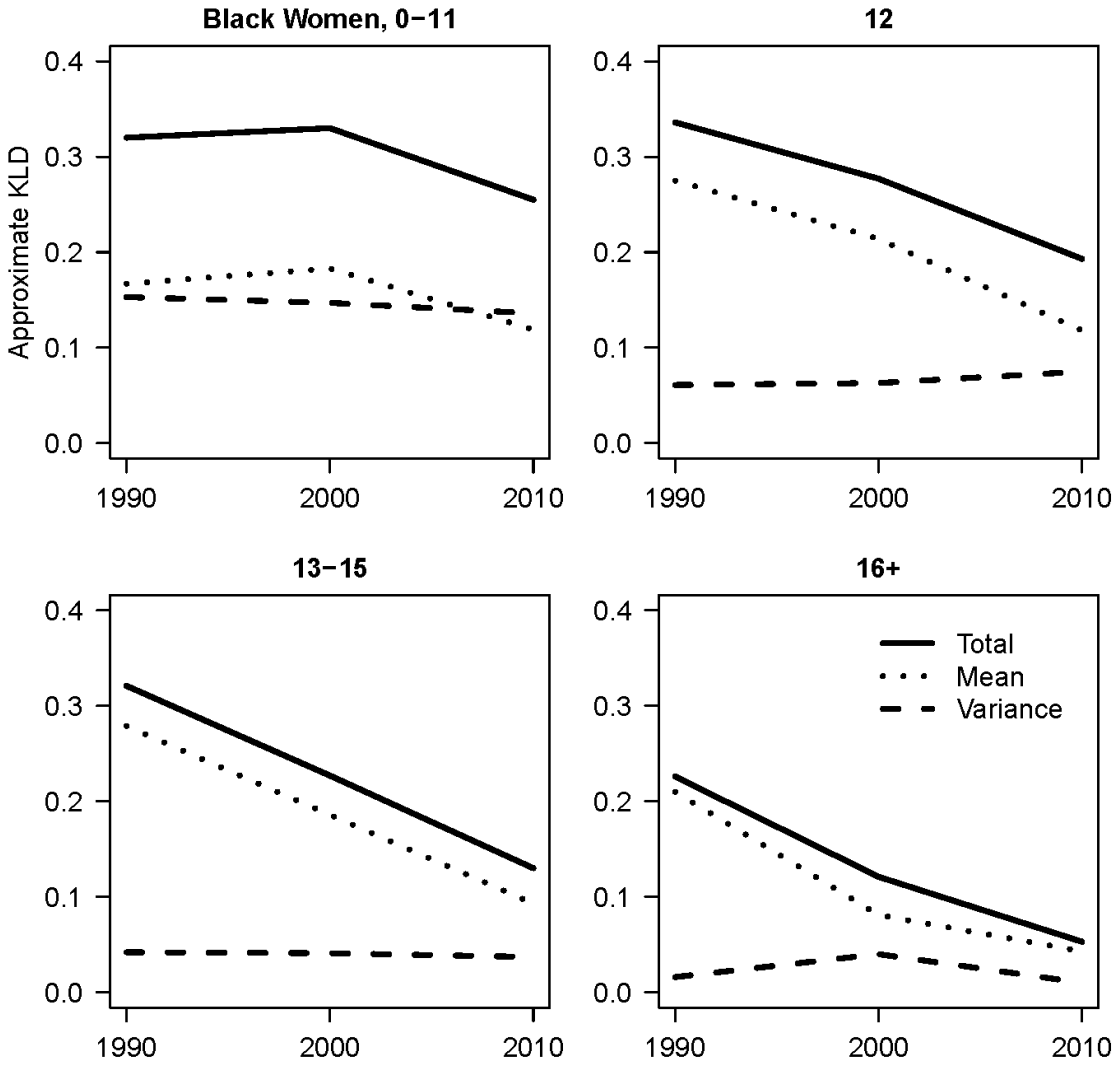


Figure 2.3: Divergence and convergence in age-at-death distribution by educational attainment, non-Hispanic white men (reference: non-Hispanic white men with 16+ years of schooling in 2010)

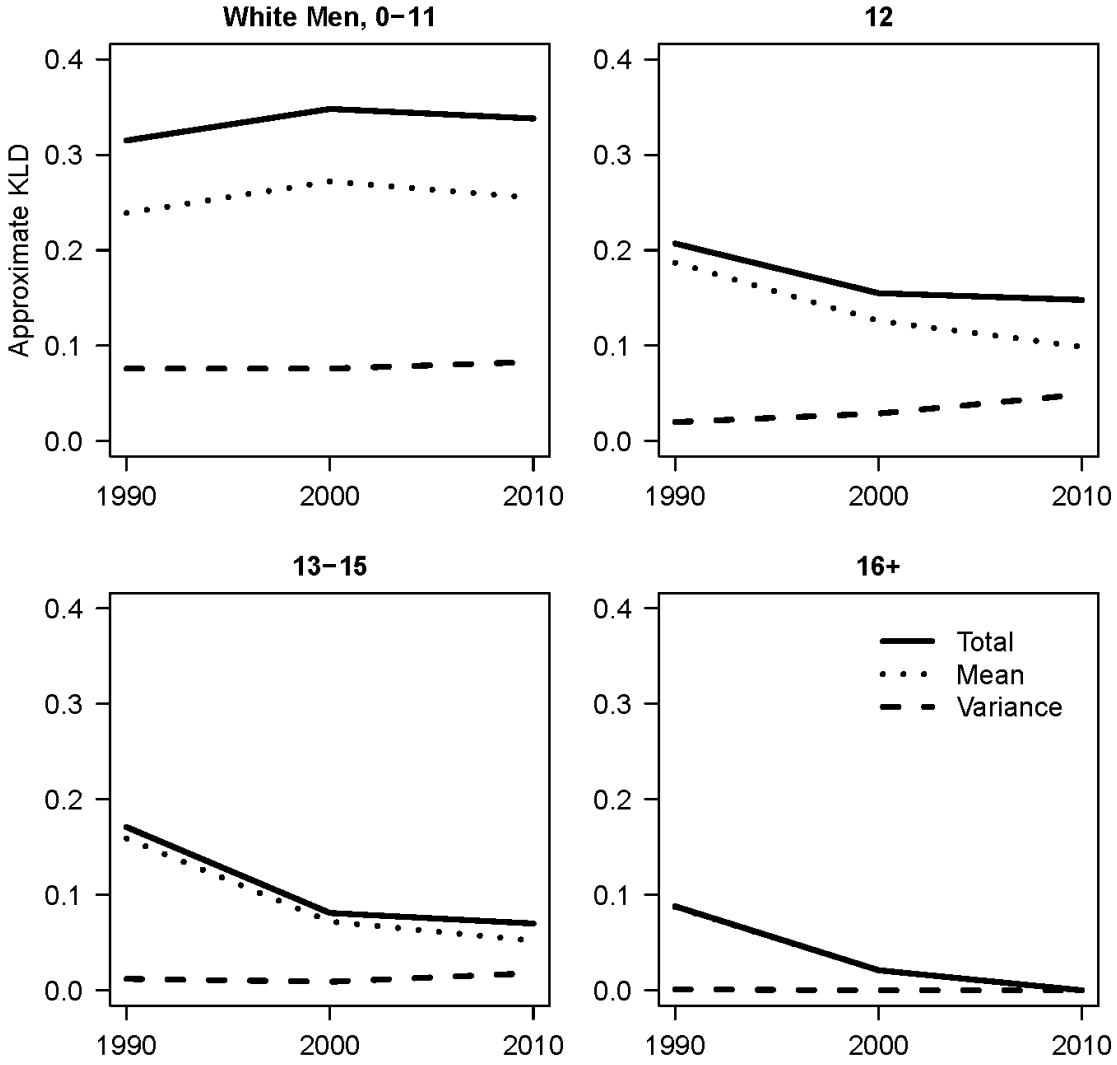
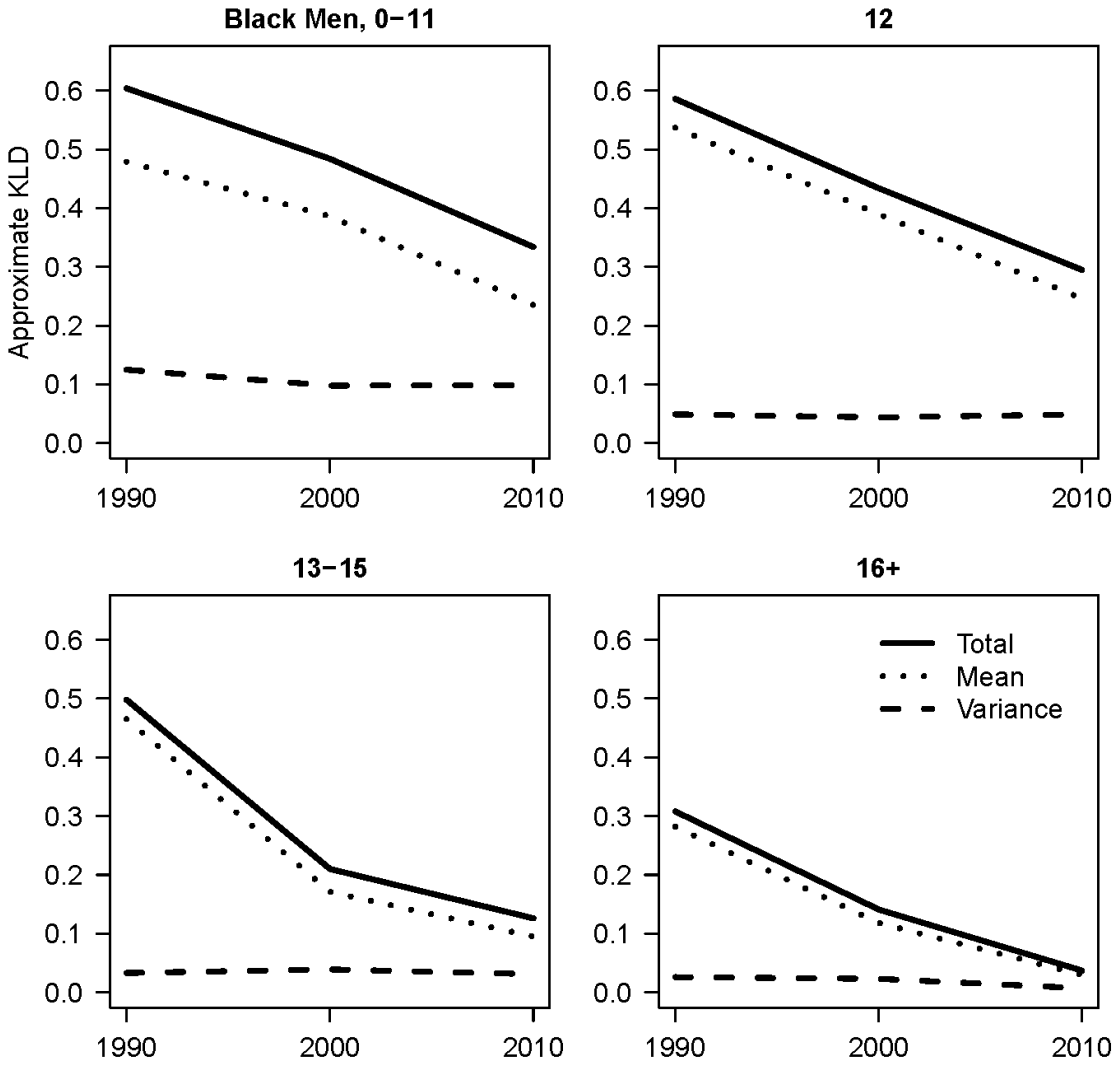


Figure 2.4: Divergence and convergence in age-at-death distribution by educational attainment, non-Hispanic black men (reference: non-Hispanic white men with 16+ years of schooling in 2010)



Note: The scale of the vertical axis differs from Figures 2.1-2.3.

Chapter 3: Decomposing the Rising Education Gap in Life Expectancy by Age and Cause of Death, United States 1990-2010

INTRODUCTION

Since the 1960s, the U.S. adult mortality regime has been undergoing two fundamental changes. First, the U.S. population as a whole has entered the fourth stage of the epidemiological transition—the age of delayed degenerative diseases (Olshansky and Ault 1986). Chronic diseases have not only substituted for infectious diseases as the leading causes of death, but are now being delayed well into old age. As a result, life expectancy is progressing toward 80 and beyond. Across this time, scholars have also documented widening educational disparities in U.S. adult mortality (Meara, Richards, and Cutler 2008; Montez et al. 2011; Preston and Elo 1995). From 1990 to 2010 alone, the gap in life expectancy at age 25 between non-Hispanic white (hereafter white) men with 0-11 and 16+ years of schooling has doubled from six to twelve years, and more than tripled among white women, increasing from 2.5 to 9.3 years over the same period (see Chapter 2). One of the key sociological insights of our time is that the two processes are fundamentally interrelated. Socioeconomic disparities in health and mortality are not increasing in spite of major advances in average longevity, but *because* of those advances (Freese and Lutfey 2011; Link 2008). The growing social capacity to control health and disease is unequally shared by groups with varying levels of socioeconomic status (SES). Specifically, high SES individuals command greater material and non-material resources,

which in turn facilitate greater access to healthy environments and lifestyles (Link and Phelan 1995; 2002; Phelan et al. 2004).

The basic tenet that all societies converge to the fourth stage of the epidemiological transition has already been challenged on a global scale, given the reversal of mortality decline in multiple countries and the recurring divergence in life expectancy (Casseli, Meslé, and Vallin 2002; McMichael et al. 2004; Vallin and Meslé 2004). But the results in Chapter 2 suggest that the same divergence in mortality is occurring in the U.S. on a subnational level, where low SES Americans are subject to rising mortality. Life expectancy has been declining since 1990 among white Americans with fewer than 12 years of schooling. During the 2000s, life expectancy also reached a plateau among whites with 12 years of schooling, for both genders. In other words, individuals with 12 or fewer years of schooling, who currently make up over 45 percent of the white American population over the age of 25 (Ruggles el al. 2010), have been excluded from any significant improvement in average longevity for at least a decade. Although U.S. life expectancy is still on the rise, owing to significant gains by blacks, Hispanics, and the college educated, the slowing down and even reversal of this trend among large segments of the population should be alarming to scholars, policy makers, and the general public.

The immediate question that follows—*what is causing these trends?*—invokes two kinds of answers. The first concerns the demographic accounting of deaths to age- and cause-specific mortality rates, and change therein, in order to identify the main culprits. Between 1986 and 2006, age-specific, all-cause mortality rates increased among

white women with fewer than 12 years of schooling (Montez et al. 2011) and particularly for lung cancer, cerebrovascular diseases, chronic lower respiratory diseases, diabetes, and Alzheimer's disease (Montez and Zajacova 2013a). During the same period, all-cause mortality among low educated men increased below age 55 and declined in older ages (Montez et al. 2011), but less is known about changes in cause-specific mortality (one exception being Miech et al. 2011). In much the same way, the modest changes in period life expectancy observed among high-school educated whites may be masking dramatic shifts in the composition and timing of underlying hazards. Uncovering trends in those hazards and how they affect life expectancy is the first step toward explaining why low and high-school educated Americans no longer benefit, on average, from advances in longevity. This is the focus of the current chapter.

The more fundamental answer to the causal question is the sociological one. It refers to the underlying social factors which govern and shape the risk of mortality over the life course for groups of different educational levels. This second type of answer to the causal question is dealt with in Chapter 4. But before the underlying social causes can be sought after, one must first understand how the composition and timing of adult mortality risks are distributed by educational attainment and how they have changed in recent decades.

Given that education is one of the primary markers of socioeconomic status, a slew of research has focused on the existence and growth of educational disparities in U.S. adult mortality (see review in Hummer and Hernandez 2013; Hummer and Lariscy 2011). During the 1990s, much of the growing gap in life expectancy was attributed to

increasing differentials in the prevalence of heart disease, cancer, and smoking-related diseases (e.g., lung cancer, chronic obstructive pulmonary diseases) and more so among women than men (Meara et al. 2008). But focusing on relative differences obscures within-group trends in cause-specific mortality. Among white women, for example, the widening gap in life expectancy conceals two disparate trends—continued reductions in mortality among the highly educated coupled with increasing mortality among the low educated (Montez and Zajacova 2013a). Furthermore, framing educational disparities in mortality using relative risks hides the absolute burden of disease inflicted on various education groups. For example, the mortality rate from influenza and pneumonia is 10.12 times higher among low educated women aged 45-84 relative to their college educated counterparts (Montez and Zajacova 2013a). An important next step, building on those previous studies, is to translate those relative disparities into absolute number of life years lost. The causes of death exhibiting the greatest disparities may not be the ones responsible for the greatest disparity in life expectancy, because even a modest increase in young-adult mortality can offset tremendous mortality reductions in old-age.

The composition and timing of mortality risks over the life course determine not only differences in life expectancy, but also differences in age-at-death variability. Indeed, Chapter 2 reveals that S_{25}, the standard deviation of age at death over 25, has increased from 1990 to 2010 by approximately 1.5 years among low and high-school educated whites of both genders (with the exception of men with fewer than 12 years of schooling). The young-old threshold age marks the cutoff where *increasing* mortality below it and *decreasing* mortality above it both contribute to increasing age-at-death

variability (Zhang and Vaupel 2009). As far as period mortality is concerned, increasing variation in age at death may indicate that recent birth cohorts are increasingly worse off in terms of their chances of survival, whereas older cohorts continue to see gains in longevity (or, at the very least, reductions in old-age mortality are greater). Using a Gompertz mortality model, Gillespie and colleagues (2014) approximate the threshold age in modern populations to be around one standard deviation below the life expectancy. The authors go on to suggest that age-at-death variability will continue to increase as old-age mortality declines, but warn that young-adult mortality, particularly among low SES groups, is another part of the equation that should be carefully monitored. A decomposition of age-specific contributions to change in life expectancy, relative to the young-old threshold age, can therefore explain why age-at-death variability is on the rise among low and high-school educated Americans.

In summary, trends in life expectancy reflect the net change in age-specific mortality rates, which in turn reflect the sum of competing risks of different causes of death over the life course. The great majority of studies on educational differences in U.S. adult mortality have relied on survey data, and while these data have proven invaluable, they can reveal only pieces of the puzzle at a time. Samples are often limited in age coverage, temporal scope, or the number of observations required to estimated age-cause-specific mortality rates. By contrast, vital statistics data practically cover the entire population and, in spite of well-known limitations concerning the quality of education reporting (Rostron, Boies, and Arias 2010; Sorlie and Johnson 1996), allow

repeated cross-sectional analysis of within- and between-group trends in age- and cause-specific mortality rates over time.

Using data from the vital registry from 1990 to 2010, this study aims at understanding why life expectancy has shown little progress or declined among low and high-school educated white Americans, while significant gains have been observed among highly educated groups. I chose to focus on white Americans because blacks of all education levels have continued to see improvements in life expectancy during the same period, in spite of their ongoing disadvantage compared to whites (nevertheless, results for non-Hispanic blacks are included in Appendix B). In keeping with the main objective, I use two decomposition methods to describe within- and between-group change in mortality risks over the life course. First, I explore which age groups have been most vulnerable to (have benefited the most from) increasing (decreasing) risk of mortality, and how they contributed to change in life expectancy and age-at-death variability. Second, I describe the disease burden (i.e., number of life years lost to each cause of death) in each education group and how it has changed over the study period. Third, I evaluate which causes of death best explain the growing educational gap in life expectancy, pointing to where policy intervention can be most effective in reducing such disparities.

METHODOLOGY

Data

The analysis uses two sources of data to estimate age-gender-education-cause-specific mortality rates. Death counts were obtained from the 1990, 2000, and 2010

Multiple Cause of Death (MCD) public use data files (Centers for Disease Control and Prevention 2013), which include information from all death certificates issued in the U.S. in a given year. Person-years of exposure were based on midyear population estimates from the 5% Integrated Public Use Microdata Sample (Ruggles et al. 2010) in respective census years. The analysis is limited to non-Hispanic white men and women (results for non-Hispanic blacks are shown in Appendix B) because Hispanic origins are more often misclassified on death certificates (Arias et al. 2010). I focus in particular on mortality between ages 25 and 85 because educational attainment at the college level is generally completed by age 25, and because age and cause of death reporting in vital registries are less reliable beyond the age of 85 (Alpérovitch et al. 2009; Hill, Preston, and Rosenwaike 2000; Tinetti et al. 2012). However, the results confirm that much of the educational gap in e_{25}^{o} is captured between ages 25 and 85.

I recoded educational attainment in both data sources into four categories based on completed years of schooling: low (0-11), high school (12), some college (13-15), and college degree or higher (16+). Education reporting on U.S. death certificates is often inaccurate or missing altogether (Rostron el al. 2010), particularly in earlier years and among older decedents (Sorlie and Johnson 1996). Furthermore, a new classification of educational attainment was introduced to death certificates in 2003, and has since then been gradually adopted by some, but not all, states (Murphy, Xu, and Kochanek 2013). Several steps were taken to ensure the consistency of educational attainment categories over time and to impute missing data. The latter is particularly important when estimating mortality rates from unlinked data (i.e., the numerator and denominator come from

different data sources) in order to avoid mortality undercount. Chapter 2 describes in detail all steps involved in data preparation and imputation.

In addition to information on the decedent's race, gender, age at death, and educational attainment, the MCD includes an underlying a cause of death code using the International Classification of Diseases (World Health Organization 2004). Deaths in the 1990 MCD file are classified according to the 9th Revision of the International Classification of Diseases (ICD), whereas those in 2000 and 2010 MCD files are classified using the 10th Revision. I grouped causes of death in nine major categories to ensure that they are consistent over time and in order to avoid cells with low death counts (see Appendix C for a complete list of codes). The categories include: infectious and parasitic diseases, neoplasms (excluding those attributed predominantly to smoking), cardiovascular diseases (CVD), cerebrovascular diseases, smoking-related diseases (SRD), respiratory diseases (excluding chronic lower respiratory diseases), diabetes mellitus, external causes, and a residual category for all other or unspecified causes. Table 3.1 summarizes the number of deaths from each of the nine categories by year, gender, and educational attainment.

Importantly, the smoking-related disease category includes causes where the smoking-attributable fraction of deaths exceeds 65 percent in men and women combined (Centers for Disease Control and Prevention 2008). These include cancers of the lip, oral cavity, pharynx, esophagus, larynx, trachea, lung, and bronchus, as well as chronic lower respiratory diseases (bronchitis, emphysema, and chronic airway obstruction). Since not all deaths in this category are necessarily due to smoking, while many deaths in other

categories *are* attributable to smoking (e.g., about 16 percent of deaths from ischemic heart disease), this category is not intended to accurately capture the total disease burden of cigarette smoking. However, by including only causes of death which are predominantly due to smoking, trends in this category most clearly and directly gauge the underlying smoking behavior of different educational attainment groups over time.

Age Decomposition of Change in Life Expectancy

Once age-gender-education-cause-specific mortality rates were estimated, I constructed multiple decrement period life tables for each gender and educational attainment group in 1990, 2000, and 2010. Using standard life table notation, the change in life expectancy between time 1 and time 2, $e_{25}^{o}(2) - e_{25}^{o}(1)$, can be decomposed into contributions from changing all-cause mortality rates in each 5-year age group in the following manner (Arriaga 1984; Preston, Heuveline, and Guillot 2001:64)

$$ {}_{n}\Delta_{x} = \frac{l_x^1}{l_{25}^1}\left(\frac{{}_{n}L_x^2}{l_x^2} - \frac{{}_{n}L_x^1}{l_x^1}\right) + \frac{T_{x+n}^2}{l_{25}^1}\left(\frac{l_x^1}{l_x^2} - \frac{l_{x+n}^1}{l_{x+n}^2}\right) \qquad (3.1) $$

That is, the contribution of the changing mortality rate between ages x and $x+n$ to the total change in life expectancy is a sum of two components. The first term on the right-hand side of Equation 1 corresponds to the change in person-years lived between ages x and $x+n$ (i.e., the direct effect). The second term reflects the change in person-years lived in *subsequent* age intervals attributed to more (or fewer) survivors in ages x to $x+n$ (i.e., the sum of indirect and interaction effects). Clearly, only the direct effect applies to the open interval

$$ {}_{\infty}\Delta_{x} = \frac{l_x^1}{l_{25}^1}\left(\frac{T_x^2}{l_x^2} - \frac{T_x^1}{l_x^1}\right) \qquad (3.2) $$

Finally, the sum of changes in each age group equals the total difference in life expectancy at age 25 between time 1 and time 2

$$\Delta e_{25}^{o} = e_{25}^{o}(2) - e_{25}^{o}(1) = \sum_{25}^{\infty} {}_{n}\Delta_{x} \quad (3.3)$$

In other words, the decomposition reveals which age groups contributed most to gains or losses in life expectancy over time. Furthermore, it can reveal scenarios where life years gained by declining mortality in some age groups are offset by losses from increasing mortality in other age groups.

Measuring Disease Burden

In addition to age-specific contributions to change in life expectancy, it is important to understand which causes of death explain the educational gap in life expectancy, as well as absolute change in life expectancy in each educational attainment group. While Arriaga's decomposition can be extended to cause-specific contributions to *change* in life expectancy (Arriaga 1989), it does not reflect the *absolute* disease burden in the population, measured by the average number of life years lost (YLL) from each cause of death.

Perhaps the most common method of attributing life years lost to specific causes of death is based on cause-elimination life tables (Preston et al. 2001:80). This method calculates life expectancy in a hypothetical population where cause *i* has been removed entirely. The number of life years lost due to cause *i* is therefore defined as the difference between the hypothetical and the observed life expectancies:

$$YLL_i = e_x^{-i*} - e_x \quad (3.4)$$

where YLL_i is the number of years lost due to cause i, e_x is life expectancy at age x, and e_x^{-i*} is life expectancy at age x had cause i been eliminated. However, this measure suffers from several limitations. First, it refers to an (unlikely) hypothetical population where cause i is eliminated altogether.[1] Second, it assumes that competing risks of mortality are independent of each other, such that eliminating cause i will not change any of the remaining cause-specific mortality rates. Third, the measure is not additive in the sense that the sum of years lost to a set of mutually exclusive and exhaustive causes does not equal the total years lost (i.e., if all causes were eliminated then life expectancy would be infinite).

In this chapter I adopt an alternative measure of life years lost based on the cumulative incidence of each cause of death (Andersen et al. 2013):

$$YLL = 60 - {}_{60}e_{25} \tag{3.5}$$

where YLL is the number of life years lost from all causes between ages 25 and 85 and ${}_{60}e_{25}$ is the temporary life expectancy[2] in that age interval. *YLL* can be further decomposed into years lost to specific causes using standard functions from the multiple decrement life table. When the life table radix, l_{25}, equals unity, then ${}_nL_x$ represents the average number of years lived by an individual between ages x and $x+n$. The average number of years lost in that interval, denoted ${}_n\text{ל}_x$, is therefore

$${}_n\text{ל}_x = n - {}_nL_x \tag{3.6}$$

Equation 6 can then be decomposed into contributions from each cause of death (Andersen et al. 2013):

$${}_n\text{ל}_x^i = n \cdot {}_xd_{25}^i + (n \cdot l_x - {}_nL_x){}_nR_x^i \tag{3.7}$$

where ${}_n\daleth_x^i$ is the number of years lost due to cause *i* between ages *x* and *x*+*n*, l_x and ${}_nd_x$ are the standard life table functions, ${}_xd_{25}^i$ is the cumulative number of life table deaths[3] from cause *i* by age *x*, and ${}_nR_x^i = \frac{{}_nd_x^i}{{}_nd_x}$ is the fraction of deaths in the interval due to cause *i*. The first term on the right-hand side of Equation 7 can be interpreted as the number of life years lost between ages *x* and *x*+*n* due to deaths from cause *i* before age *x* (i.e., each death contributes *n* lost years); the second term equals the number of years lost due to deaths from cause *i* during the interval (i.e., each death contributes ${}_na_x$ lost years on average).

Finally, the total years lost before age *x* is the sum of years lost across all causes *i* over all intervals *j* of length *n*

$$YLL = \sum_j \sum_i {}_n\daleth_x^i \tag{3.8}$$

This alternative measure of life years lost holds several advantageous properties: (1) it is based on actual years lost within a predefined age interval, rather than on a hypothetical population where causes are completely eliminated; (2) life years lost from competing risks are additive to the total number of years lost; (3) it makes no assumption about the independence of competing risks; (4) it can easily be derived from multiple decrement life tables.

In the next section I present results from both decomposition methods. First, using Arriaga's decomposition, I show which age groups contributed most to gains or losses in total life expectancy among low, high-school, some college, and college educated white Americans from 1990 to 2010. Second, using a cause of death decomposition of temporary life expectancy, I show which causes of death had the greatest toll on life years

lost, how that toll has changed over time within each educational attainment group, and which causes explain the growing educational gradient in longevity.

RESULTS

Age Decomposition of Change in Life Expectancy

The first decomposition method concerns the contribution of change in age-specific mortality rates to the total change in life expectancy of various education groups. This is an important first step to identifying which age groups have been most vulnerable to increasing mortality rates or have benefited the most from declining mortality. Although life expectancy has been declining among low educated men and women and increasing among their college educated counterparts, these patterns may not be equally shared by all age groups (or birth cohorts, as far as period mortality is concerned). Figure 3.1 shows the age decomposition of change in life expectancy between 1990 and 2010 by educational attainment. Results in the top panel are for low (0-11 years of schooling), high school (12), some college (13-15), and college (16+) educated white women, whereas results for white men are shown in the bottom panel. The bars represent the contribution (in years) of each 5-year age group to the total change in life expectancy. Recall that this contribution consists of life years gained (or lost) within the age interval and in all subsequent intervals due to changing mortality rates within the interval. Furthermore, the sum of all age-specific contributions equals the net change in life expectancy over the two decade period. The dashed lines in Figure 3.1 mark the approximate young-old threshold age in each education category in 1990. Decreasing

mortality below the threshold age reduces age-at-death variability, whereas decreasing mortality above the threshold increases the variability.

Between 1990 and 2010, life expectancy at age 25 declined by 3.1 years among low educated women. Although mortality increased in all age groups, the bulk of the change in life expectancy was due to rising mortality between ages 45 and 64. This suggests that middle-aged women in this education category were most vulnerable to the changing mortality regime, and that targeting this group can have the greatest impact on future gains in longevity.

During the same period, life expectancy increased by less than a year among high school educated women because gains in longevity above age 55 were almost entirely offset by losses below that age. In other words, the modest increase in e_{25}^{o} masks contrasting trends for the young and the old. While middle aged and older women continue to experience reductions in mortality, the trend has reversed for younger women. Incidentally, this reversal is close to the young-old threshold age (67.2), which explains why age-at-death variability increased among high-school educated women during the study period (Chapter 2). Both components—declining mortality above the threshold and increasing mortality below it—worked to increase variation.

Trends among women with some college education resemble those of their high-school educated counterparts of the same age. However, gains in life expectancy above age 55 were greater and losses below 55 were less pronounced, which resulted in a net increase of 1.5 years in e_{25}^{o}. Finally, college educated women experienced declining

mortality across all age groups, with most gains in life expectancy attributed to ages 65 and above.

The bottom panel of Figure 3.1 shows the age decomposition results for white men. Overall, men fared better than women at each level of education, but exhibited similar age patterns. Life expectancy at age 25 declined by less than one year among low educated men, mostly due to rising mortality between ages 45 and 64. However, this trend was less pronounced than for low educated women and was offset by minor improvement in the remaining age groups. Results for high-school educated men also resembled those of women, with mortality declining significantly at older ages (over 60) and increasing only slightly at younger ages. Here, too, age-at-death variability increased as a result, but e_{25}^{o} also increased by 1.8 years (Chapter 2). Men with either some or completed college education experienced declining mortality across the board, but contributions to life expectancy were most pronounced at ages 60 and over. These resulted in net increase in e_{25}^{o} of 2.9 and 5.2 years, respectively.

Taken together, the age decomposition of change in life expectancy reveals that declining mortality at ages 60 and over, in almost all educational attainment groups, contributed most to gains in life expectancy at age 25. Furthermore, among low and high-school educated men and women mortality generally *increased* below age 60, offsetting gains, if any, at older ages. These trends can be better understood by attributing lost years of life—and change therein—to specific causes of death.

Life Years Lost by Cause of Death and Educational Attainment

The temporary life expectancy between ages 25 and 85, ${}_{60}e_{25}$, is the average number of years a person is expected to live during the 60-year interval. Although it truncates mortality above age 85, ${}_{60}e_{25}$ captures much of the educational gap in longevity. Throughout the study period, it remained about 1-2 years lower than e_{25}^{o} among men, across all educational attainment groups, and about 3 years lower than e_{25}^{o} among women. The complement of ${}_{60}e_{25}$ is the average number of life years lost from all-cause mortality (i.e., $YLL = 60 - {}_{60}e_{25}$), which can be further decomposed by cause of death.

Figure 3.2 shows the trend in total life years lost (YLL) between ages 25 and 85 by gender and educational attainment for 1990, 2000, and 2010. Mirroring the trends in e_{25}^{o} found in Chapter 2, YLL gradually increased from 9.0 years in 1990 to 11.6 years in 2010 among low educated women, plateaued around 7.4 years among high-school educated women, and declined among women with 13-15 and 16+ years of schooling (from 7.3 to 6.4 and from 6.2 to 4.2, respectively). Overall, by age 85, men had lost more years of life compared to women of the same educational level and exhibited a wider educational gap. YLL increased slightly for low educated men during the 1990s and plateaued during the 2000s at 15.8 years. In all other education groups, however, YLL declined for men: from 12.3 to 11.3 years among the high-school educated, from 11.4 to 9.2 among the "some college" category, and from 9.3 to 5.8 among the college educated. In both genders, the educational gap in YLL—the difference between the least and most

educated groups—increased over time and was greatest in 2010, reaching 7.5 years among women and 10.0 years among men.

A further decomposition of YLL reveals which causes of death underlie these trends both within and between educational attainment groups. Figure 3.3 shows the number of life years lost by cause of death and educational attainment among white women in 1990, 2000, and 2010. Among low educated women, YLL from smoking-related diseases, external causes, and residual causes (the "other" category) increased by about one year each during the study period. Together, these causes accounted for more than the total increase in YLL, but were offset by minor reductions in YLL from CVD and neoplasms (about one third of a year each). YLL also increased for diabetes and infectious and respiratory diseases, but their combined effect was less than 0.5 additional life years lost—far below the rising burden from each of the three leading causes.

Similar trends were observed among high-school educated women, but changes in YLL from different causes of death offset each other almost entirely—i.e., increases from smoking-related diseases, external, and other causes were more modest while reductions from CVD and cancers were greater than among low educated women. Surprisingly, women with some college education also experienced an increase in YLL from external and other causes, albeit to a lesser degree, and no significant change attributed to smoking-related diseases. College educated women, on the other hand, saw reductions in YLL almost uniformly across all causes—the greatest of which from CVD and neoplasms (0.7 and 0.9 years, respectively)—resulting in the overall improvement in life expectancy, temporary or total, discussed earlier.

Trends among men were generally similar (but not identical) to those among women of the same educational level. Figure 3.4 shows that low educated men gained over one year of life due to reductions in CVD, but experienced greater combined losses from smoking, external, and other causes. Losses from external and other causes also increased among men with high-school or some college education, but declined for CVD and smoking-related diseases. Like their women counterparts, college educated men saw reductions in YLL from practically all cause of death groupings.

Overall, trends in life years lost by cause of death suggest that mortality reductions from CVD and neoplasms have been a success story in all gender and education groups, but have been greatest among the college educated. By contrast, the number of life years lost from external and other causes has been rising in almost all education groups, with the exception of college educated men and women. In addition, smoking had an increasing toll on longevity among low and high-school educated women, as well as on low educated men. Changes in YLL were also observed in infectious, respiratory, and cerebrovascular diseases and in diabetes, but their toll on life years lost was significantly lower than from the leading causes of death—CVD, neoplasms, and smoking-related diseases.[4] By 2010, these three causes alone were responsible for over 50 percent of life years lost in each gender-education group. Among the low educated, however, external and other causes also had a tremendous toll on the number of years lost, both in absolute and in relative terms. This is an important observation, because even a modest increase in the number of premature deaths among

the low educated can have a significant effect on the number of life years lost, and therefore contribute to the educational gap in longevity.

The Educational Gradient in Years of Life Lost by Cause of Death

A cause-by-cause comparison of life years between education groups can also point to where educational disparities in longevity are greatest—and where health policy might have the greatest impact—which may not be immediately obvious when comparing mortality rates, or, in particular, relative differences between groups.

Two general patterns can be discerned in Figures 3.3 and 3.4. First, there is a clear educational gradient in YLL across all causes of death and in both genders, with more education generally resulting in fewer life years lost. Throughout the study period, between 60 and 80 percent of the gap in ${}_{60}e_{25}$ (or, equivalently, in total YLL) between low and college educated men and women was attributed to CVD, smoking-related diseases, and external causes. Although (non-smoking related) neoplasms constitute an important share of the absolute number of life years lost in each education groups, they explain less than 5 percent of the difference in ${}_{60}e_{25}$ between groups. Similarly, disparities in YLL attributed to diabetes and infectious, respiratory, and cerebrovascular diseases *combined* explain less than 20 percent of the gap in ${}_{60}e_{25}$ in women and less than 15 percent of the gap in men. In other words, although the educational gradient exists in nearly all causes of death, reducing mortality from CVD, smoking-related diseases, and external causes among low educated groups will have the greatest impact on closing the education gap in life expectancy.

The second clear observation is that the educational gap in YLL has increased since 1990 across all causes of death and in both genders. In causes of death where all groups saw reductions in YLL (e.g., neoplasms and CVD), the college educated saw greater reductions. But in most causes of death, YLL generally increased among low educated men and women while it declined among their college-educated counterparts. For example, in 1990, low educated men lost 1.5 additional life years from CVD relative to college educated men. This gap increased to 2.1 years by 2010 despite significant reductions in life years lost to CVD in both groups. In contrast, the gap in YLL due to smoking increased from 1.1 to 2.0 years—about half of which because of declining YLL among the college educated and the other half due to increasing YLL among the low educated. This pattern not only reflects greater health returns to higher education, but also worsening absolute conditions among low and even high-school educated groups.

DISCUSSION

From 1990 to 2010, the life expectancy gap between low and college educated white Americans has doubled for men and more than tripled for women (Chapter 2). This trend was fueled by two disparate effects—absolute declines in e_{25}^{o} among the low educated and dramatic improvements among the highly educated. Furthermore, high-school educated whites have seen only modest improvements in life expectancy over those two decades. Using vital statistics data, this chapter set out to understand how changing age- and cause-specific mortality rates have contributed to gains and losses in average longevity by different education groups. The findings uncover which education

groups have been most vulnerable to the changing mortality regime, at what ages, and from which causes of death—informing health policy and at the same time providing a glimpse into the future of U.S. adult mortality.

Four key insights are supported by the evidence: (1) unlike their college educated counterparts, low educated Americans are still subject to a mortality regime characteristic of the third stage of the epidemiological transition; (2) the educational gap in life expectancy has widened both because mortality increased among low and high-school educated Americans and because mortality decreased among the college educated; (3) rising mortality among the former was concentrated in mid-life (ages 45-64), whereas mortality reductions among the latter were concentrated in old age (65 and over); (4) life expectancy declined or plateaued among low and high-school educated Americans almost entirely due to rising mortality from smoking-related, external, and residual causes, offsetting any gains from declining mortality from CVD and neoplasms.

Since the 1960s, the U.S. has been moving toward the fourth stage of the epidemiological transition, the age of delayed degenerative diseases, where mortality from chronic diseases is shifted to old age and life expectancy at birth progresses well into the ninth decade of life (Olshansky and Ault 1986). However, large segments of the U.S. population have not experienced these improvements in longevity and, more recently, some have even been regressing. The findings in this study suggest that low educated Americans are not only subject to higher mortality rates than their college educated counterparts, but they also exhibit a cause-of-death profile characteristic of the third stage of the epidemiological transition (Omran 1971). Cardiovascular diseases

remained the leading cause of death throughout the study period, with neoplasms far behind, and the mean (life table) age at death was measured in the 70s at best. Smoking-related and external causes of death exhibited a significant toll on the number of life years lost by age 85. Deaths from diabetes, strokes, and infectious and respiratory diseases were much less prominent, but nevertheless had an observable impact on life expectancy. In contrast, both the composition and timing of mortality risks among the college educated were consistent with the fourth stage of the epidemiological transition. Deaths from infectious diseases were almost non-existent, chronic diseases were increasingly delayed to old age, and by 2010 the number of life years lost to neoplasms by age 85 matched (for men) or surpassed (for women) those lost to CVD. In other words, educational attainment in the U.S. places individuals in different health trajectories over the life course, rather than merely exacerbate (or mitigate) the risk inflicted by any given cause of death.

More troubling is the fact that, from 1990 to 2010, the mortality regime has become even less favorable for low and high-school educated Americans, and no longer seems to be a matter of "catching up" with those in the lead. Life expectancy at age 25 declined among low educated men and women primarily because mortality increased in mid-life (ages 45-64). At the same time, mortality declined across all age groups for the college educated, but because mortality had very little room for further decline at younger ages, nearly all gains in e_{25}^{o} took place over the age of 65. Trends among men and women with high school or some college education are even more peculiar. While old-age mortality declined, the subsequent gains in e_{25}^{o} were partially, if not entirely, offset by

increasing mortality under the age of 60. This pattern also explains the increasing variability in age at death found in those groups in Chapter 2.

With respect to cause-specific mortality, this study finds that the number of life years lost from all major causes of death declined for college educated white women, whereas, with the exception of CVD, non-smoking related cancers, and cerebrovascular diseases, YLL increased for low educated women. In addition, high-school educated women experienced an increasing loss of life years from smoking-related, external, and residual causes. All-cause and cause-specific YLL were generally higher among men compared to women of the same level of education, but followed the same general pattern—the main exception being that mortality from smoking-related diseases increased only for low, but not for high-school, educated men.

These results are consistent with prior research based on data from the National Health Interview Survey (e.g., Montez et al. 2011; Montez and Zajacova 2013a), but offer several important extensions. While previous studies were limited in the number of educational categories, age range, statistical power, and temporal scope, this study overcomes those difficulties by using complete data from the vital registry over a two-decade period. Furthermore, rather than compare relative risks between different education categories, this study translates cause-specific mortality rates (and change therein) into years of life lost from each cause of death.

The number of life years lost provides a direct measure of both absolute and relative burden of disease, and points to the causes of death which best explain the growing educational gap in life expectancy. The difference in temporary life expectancy

from age 25 to 85, ${}_{60}e_{25}$, between low and college educated women increased from 2.8 years in 1990 to 7.5 years in 2010. Nearly half of the *growth* in the gap is attributed to smoking-related diseases and external causes of death, where mortality increased dramatically among low educated women. The same two causes were responsible for over one third of the 4.1-year increase in the gap in ${}_{60}e_{25}$ between low and college educated men. The implication from a health policy perspective is clear: targeting premature deaths from smoking and external causes will have the greatest impact on closing the educational gap in life expectancy. Because many of these deaths occur below the young-old threshold age, preventing them will also work to reduce age-at-death variability in low and high-school educated populations. Future research is needed to further disaggregate the rise in mortality from external causes and the residual category, in order to better understand why they have become more prevalent and how to prevent them.

One limitation of this study concerns the quality of vital statistics data. Since information on educational attainment is reported by someone other than the deceased, there is a tendency for heaping at 12 years of schooling from both lower and higher levels of education (Rostron et al. 2010). Failure to report or conform to a single death certificate format also plagued multiple states over the study period (Murphy, Xu, and Kochanek 2013). Coupled with the revision of ICD codes, all of those factors presented a potential source of bias when estimating mortality trends. Several steps were taken to minimize both random and systematic sources of error, as detailed in Chapter 2, but they cannot be eliminated with certainty. It is nevertheless reassuring that results in this study

are consistent with those reported in prior research based on survey and vital statistics data, whenever they overlap in age and period, both with respect to the direction and magnitude of trends in age- and cause-specific mortality (Ma et al. 2012; Meara et al. 2008; Montez et al. 2011; Montez and Zajacova 2013a).

Perhaps the most important conclusion in this study is that low and high-school educated white Americans, who together comprise over one quarter of the U.S. adult population, are either already experiencing decline in life expectancy or on track to lose ground in the coming decades. Mortality is already on the rise among high-school educated men and women under the age of 60, and the number of life years lost by 85 is increasing due to premature deaths from smoking-related, external, and other causes. Since period mortality concerns a hypothetical population made of up of multiple birth cohorts, it is difficult to determine whether these patterns are driven by period or cohort effects. Since the 1960s, changes in mortality from heart disease and lung cancer in the U.S. have been largely driven by cohort effects (Yang 2008), which also explain the growing educational differences in mortality from those causes (Masters, Hummer, and Powers 2012). The same is likely true for other smoking-related diseases, but the case of external and other causes of death is not immediately clear. Nevertheless, the rise in young-adult mortality may be a precursor to what lies ahead for high-school educated white Americans as these cohorts enter old age.[5]

Clearly, cigarette smoking will continue to have a tremendous impact on U.S. adult mortality. Differentials in smoking behavior already explain much of the gender gap in life expectancy (Preston and Wang 2006), the Hispanic mortality advantage

(Fenelon 2013), and, according to findings in this study, the growing educational gradient in life expectancy at age 25. Smoking-related cancers and chronic lower respiratory diseases account for a significant loss of life years in all gender and education groups and especially among low and high-school educated men and women. Furthermore, the losses documented in this study necessarily underestimate the full burden of smoking on mortality from heart disease, stroke, and many other diseases. More surprising is the rise in mortality from external causes among all but the college educated, which warrants additional research.

The absolute rise in mortality among disadvantaged populations is important not only from a policy perspective, but also bears relevance for a sociological explanation. While some theories (e.g., Link and Phelan 1995) explain why high SES groups do increasingly better than low SES groups in relative terms, less is understood about why the latter have done increasingly worse in absolute terms. This chapter revealed which education groups in the U.S. have become most vulnerable over the past two decades, when during the life course they are most vulnerable, and which causes of death pose the greatest risk. The next chapter focuses on the social conditions that gave rise to those patterns and trends.

Notes:

1. Alternatively, based on the life table entropy, Keyfitz's H approximates the effect of a proportional change in the mortality rate from cause *i* on e_x (Keyfitz and Caswell 2005). However, it does not lend itself to direct cause of death decomposition where changes in mortality rates are not uniform across age groups.

2. The temporary life expectancy is similarly defined in Arriaga (1984) as

$$ {}_n e_x = \frac{T_x - T_{x+n}}{l_x} $$

   but calculated here from age 25 to age *x* rather than the average number of years lived in each *n*-year interval.

3. When the life table radix, l_{25}, equals unity, then ${}_x d_{25}^i$ is the probability of dying from cause *i* before age *x*.

4. Note the average number of life years lost to smoking-related diseases reflects the population burden as a whole, not the expected loss for an individual smoker.

5. Although mortality trends among high-school educated blacks were more favorable over the study period (see Appendix B), they remain considerably disadvantaged compared to whites of the same educational level.

Table 3.1: Midyear population estimates and number of deaths by gender, education, and cause among non-Hispanic whites

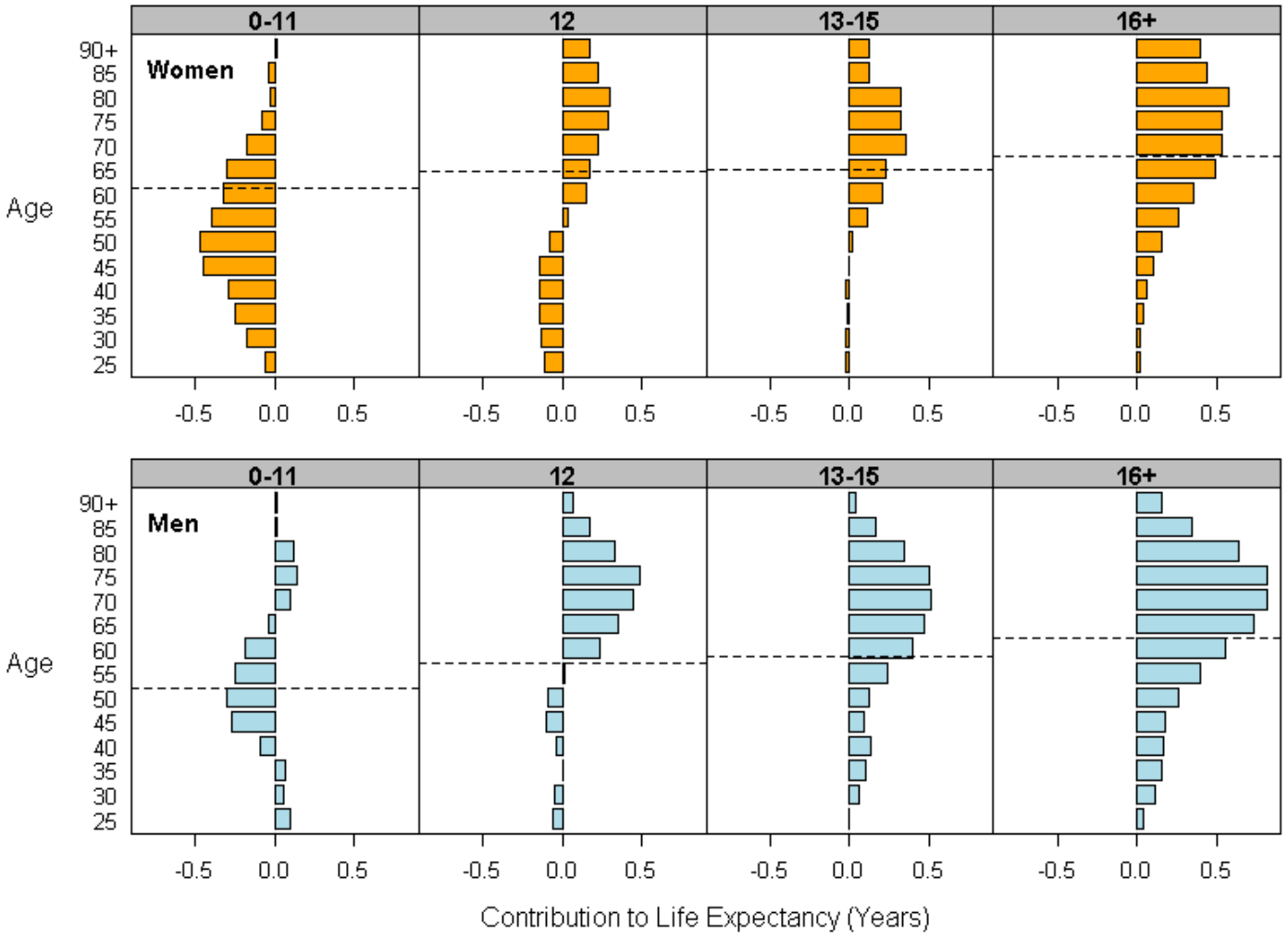
Gender	Year	Education (years)	Midyear population	Infectious/ parasitic	Neoplasms	Cardio-vascular	Respiratory	External	SRD**	Cerebro-vascular	Diabetes	Other	Total
Women	**1990**	**0-11**	12,070,975	4,782	50,393	145,973	21,718	7,958	26,499	31,846	9,133	40,456	**338,758**
		12	29,660,127	4,809	68,766	126,089	18,395	11,120	35,536	27,089	7,899	39,059	**338,762**
		13-15	11,799,518	1,255	20,754	34,171	5,412	4,170	9,587	8,461	1,950	11,814	**97,574**
		16+	12,414,257	1,146	17,701	27,371	4,524	3,361	6,697	6,847	1,385	9,663	**78,695**
	2000	**0-11**	8,281,916	5,902	39,035	116,830	17,735	7,626	34,329	28,827	9,577	54,844	**314,705**
		12	29,702,519	8,157	76,638	144,219	22,043	13,408	58,494	36,431	11,917	72,190	**443,497**
		13-15	14,961,916	2,278	26,046	41,407	6,852	5,491	17,117	11,594	3,223	23,125	**137,133**
		16+	17,494,138	1,719	23,237	30,820	5,493	4,473	11,070	9,309	1,946	18,350	**106,417**
	2010	**0-11**	5,571,690	4,994	22,964	59,864	9,687	6,951	27,484	13,252	5,393	53,040	**203,629**
		12	27,669,555	11,890	75,870	130,562	20,968	20,044	68,986	30,703	11,503	120,234	**490,760**
		13-15	17,318,233	3,854	31,207	40,028	6,826	9,725	22,769	10,238	3,551	40,125	**168,323**
		16+	22,305,450	2,609	28,311	28,288	5,018	6,573	13,619	7,570	2,125	30,183	**124,296**
Men	**1990**	**0-11**	10,347,168	5,286	50,007	136,718	19,789	16,708	53,952	20,322	6,210	33,682	**342,674**
		12	23,733,128	8,218	51,194	117,884	13,826	24,346	47,453	15,230	5,784	29,985	**313,920**
		13-15	10,361,470	4,235	17,102	35,240	4,148	8,210	13,288	4,758	1,792	9,707	**98,480**
		16+	15,252,000	5,508	22,584	42,340	5,372	7,961	13,502	5,886	1,853	11,702	**116,708**
	2000	**0-11**	7,540,518	5,041	38,290	95,180	14,343	13,861	48,120	16,734	6,977	35,732	**274,278**
		12	24,812,189	7,667	58,808	123,255	16,229	26,438	59,573	19,402	9,483	46,900	**367,755**
		13-15	13,467,696	2,742	22,388	40,897	5,343	9,745	18,825	6,838	3,376	16,803	**126,957**
		16+	18,946,760	3,083	30,758	49,345	7,071	9,526	18,590	8,944	3,572	20,957	**151,846**
	2010	**0-11**	5,605,093	4,593	25,079	53,984	9,052	12,273	34,513	8,202	5,104	34,352	**187,152**
		12	25,232,736	10,142	64,643	115,606	16,887	36,526	68,025	16,470	11,093	72,261	**411,653**

Table 3.1 cont.

	13-15	15,233,235	4,006	29,693	45,122	6,529	15,551	24,563	6,743	4,517	29,304	**166,028**
	16+	22,063,609	3,957	38,765	51,322	8,352	13,381	21,156	8,546	4,370	36,884	**186,733**

Notes: Deaths counts are average of ten imputations; SRD = smoking-related diseases (bronchitis, emphysema, chronic airway obstruction and cancers of the lip, oral cavity, pharynx, esophagus, larynx, trachea, lung, and bronchus).

Figure 3.1: Age decomposition of change in life expectancy by gender and years of schooling, non-Hispanic whites 1990-2010



Note: The dashed lines mark the approximate young-old threshold age in 1990.

Figure 3.2: Total life years lost between ages 25 and 85 by gender and years of schooling, non-Hispanic whites 1990-2010

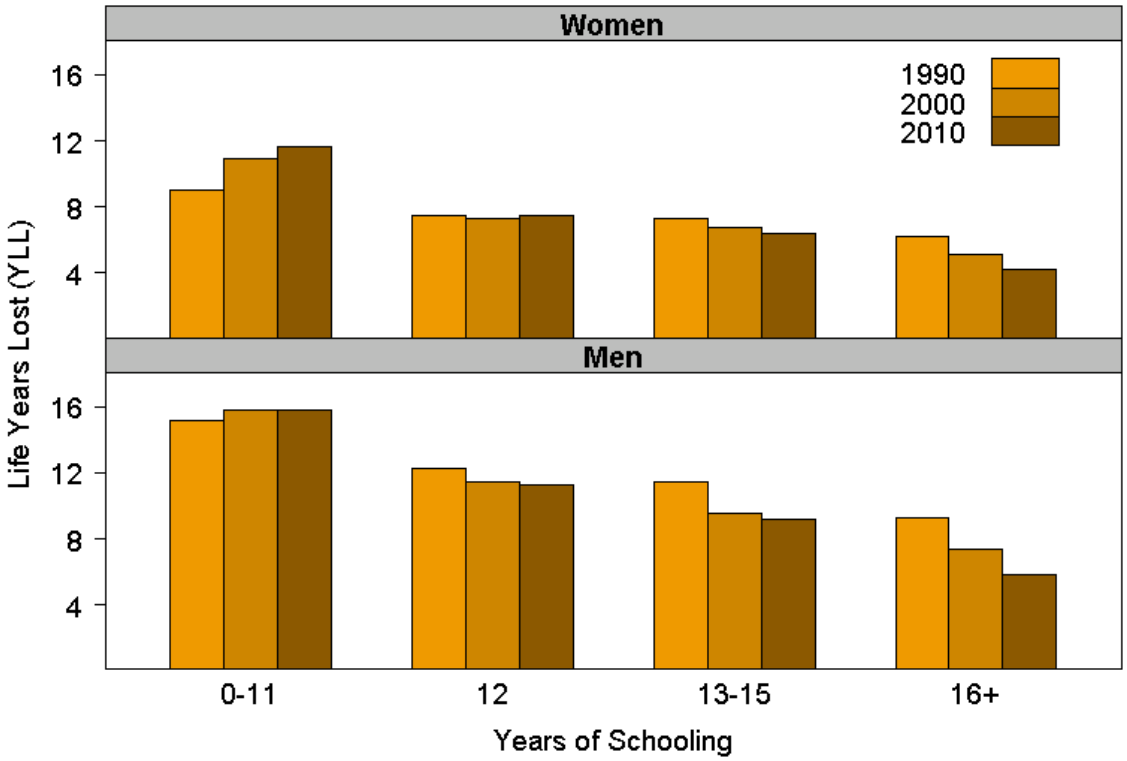
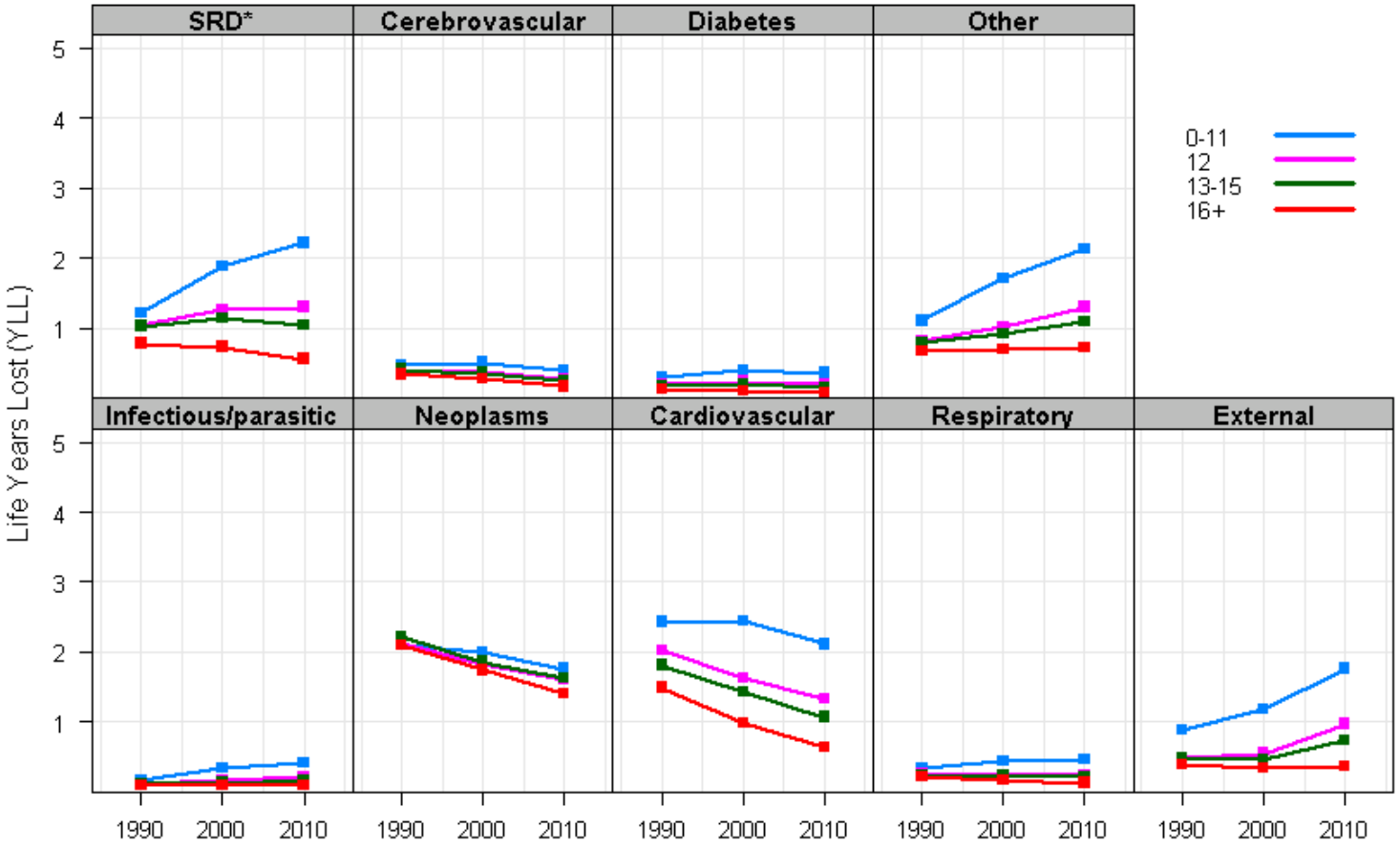
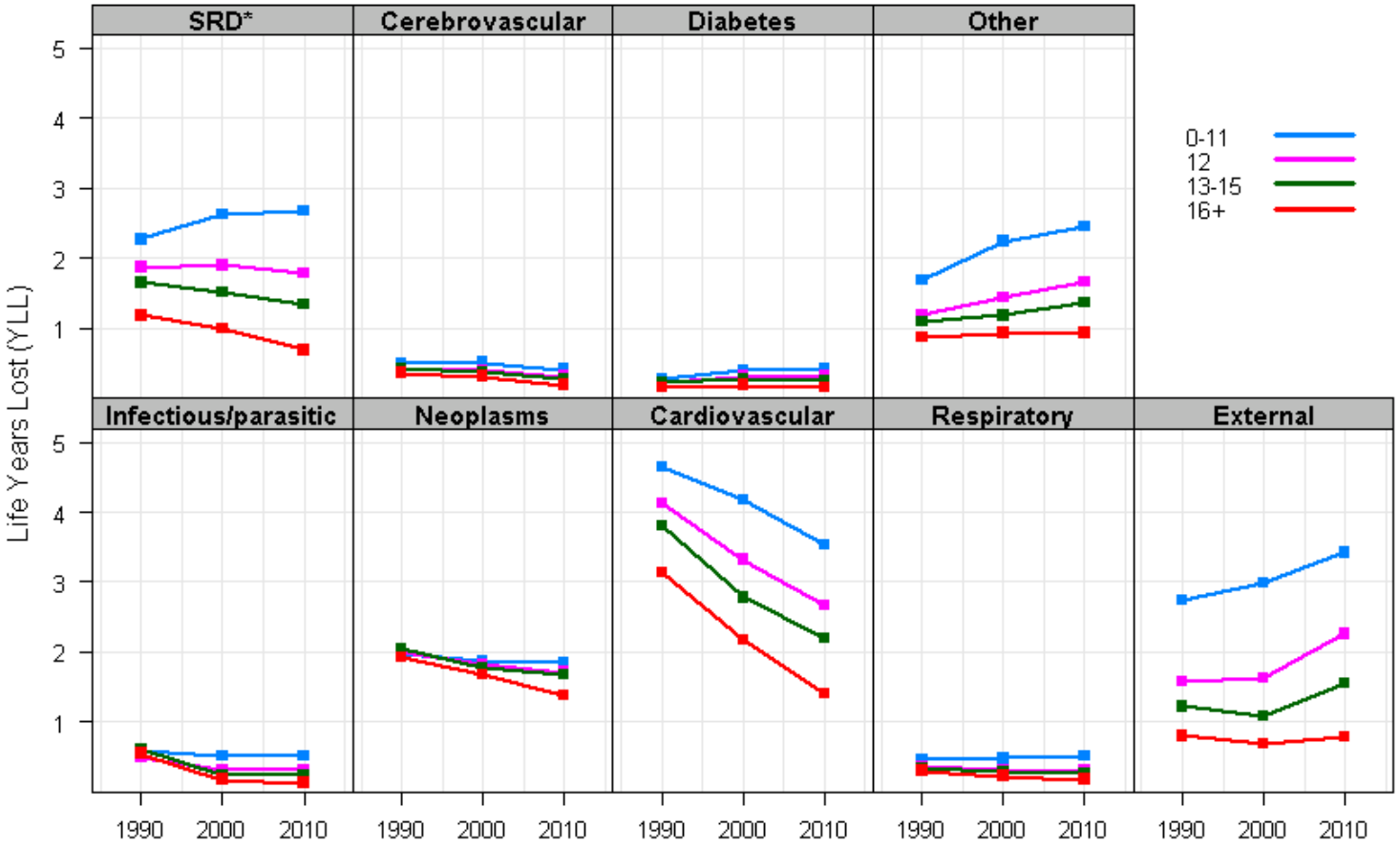


Figure 3.3: Life years lost between ages 25 and 85 by cause of death and years of schooling, non-Hispanic white women



Note: SRD = smoking-related diseases (bronchitis, emphysema, chronic airway obstruction and cancers of the lip, oral cavity, pharynx, esophagus, larynx, trachea, lung, and bronchus).

Figure 3.4: Life years lost between ages 25 and 85 by cause of death and years of schooling, non-Hispanic white men



Note: SRD = smoking-related diseases (bronchitis, emphysema, chronic airway obstruction and cancers of the lip, oral cavity, pharynx, esophagus, larynx, trachea, lung, and bronchus).

Chapter 4: Does Increasing Negative Selection Explain the Rise in Mortality among Low Educated White Americans?

INTRODUCTION

One of the most robust and consistent findings in the social sciences is the association between education and adult mortality. Not only is the association enduring (Miech et al. 2011), but it is also becoming increasingly pronounced as educational disparities in health and mortality widen (Liu and Hummer 2008; Meara, Richards, and Cutler 2008; Olshansky et al. 2012; Preston and Elo 1995). College educated Americans report better health (Zajacova, Hummer, and Rogers 2012) and benefit from lower rates (Montez et al. 2011) and higher compression of mortality (Brown et al. 2012) compared to their less educated counterparts. Furthermore, these differentials have increased dramatically in the latter half of the twentieth century. Among non-Hispanic whites, the growing educational gap in life expectancy reflects two disparate phenomena—rising mortality among those with fewer than 12 years of schooling and declining mortality among the college educated (Chapter 2; Montez and Zajacova 2013a).

Although there is general agreement that the education gradient in mortality is widening, scholars continue to debate about the nature of this trend. On one side of the debate, a significant body of literature suggests that education—and more broadly, socioeconomic status (SES)—is a primary and causal determinant of health. According to the causal perspective, educational attainment shapes individuals' access to material and nonmaterial resources, including knowledge, money, power, prestige, and social capital,

which in turn serve to minimize health risks and reduce mortality (Link and Phelan 1995). At the same time, social disadvantage accumulates with age to produce adverse health outcomes among low SES individuals (Dannefer 2003; Hatch 2005; Ross and Wu 1996). Disadvantage can consist of exposure to material hardship such as poverty, psychosocial factors such as strained relationships or lack of social support, and a variety of other life stressors.

On the other side of the debate there are those who argue that preexisting individual and family endowments determine both educational attainment and adult health outcomes (Behrman et al. 2011). According to this logic, the widening education gradient in adult mortality merely reflects the changing composition of education groups throughout the latter half of the twentieth century. Over 70 percent of the non-Hispanic white U.S. population aged 25 and over completed fewer than 12 years of schooling in 1940, compared with less than eight percent in 2010.[1] In other words, just seven decades ago those with less than high school education constituted the overwhelming majority of the U.S. adult population, whereas nowadays they represent the extreme left tail of the distribution. The expansion of education rendered low educated groups more negatively select on a variety of characteristics (e.g., family background, cognitive and non-cognitive abilities, and childhood health status) whereas the highly educated have become less positively select over time (Goesling 2007). If these characteristics are correlated with both educational attainment and with adult health outcomes, then the association between education and mortality will become more pronounced over time—even if it is not causal in nature.

The distinction between the two explanations is important for both theoretical and practical reasons. The idea that socioeconomic status shapes individuals' life chances dates back to Weber's original conception of social class (Weber 1946) and stands at the heart of contemporary sociological theories relating to health inequality (e.g., Dannefer 2003; Link and Phelan 1995). From a policy perspective, understanding education's causal role in producing health disparities can facilitate an "upstream" mode of intervention—i.e., the idea that social policy is health policy (Rose 1992)—an integral part of the Healthy People 2020 initiative to improve population health and reduce health inequality (U.S. Department of Health and Human Services 2014). Indeed, several scholars have suggested that increasing high school graduation rates can improve longevity and consequently reduce health disparities in the U.S. population (e.g., Montez, Hummer, and Hayward 2012; Montez and Zajacova 2013b). Yet the extent to which the changing composition of education groups accounts for the increasing education-mortality gradient is currently unknown.

In this chapter I provide new evidence on the selection-causation debate concerning the association between education and adult mortality. Specifically, I evaluate the extent to which the changing educational composition among non-Hispanic white Americans accounted for growing mortality differentials between 1997 and 2013. I focus on non-Hispanic white men and women (hereafter white) because they exhibit the greatest mortality differentials among all major racial and ethnic groups in the U.S. and because these growing differentials are due, at least in part, to rising mortality among the low educated (Chapter 2; Montez at al. 2011; Montez and Zajacova 2013a; Olshansky et

al. 2012). To date, only a handful of studies have examined the effect of the changing composition of education groups on health disparities in the U.S. (e.g., Goesling 2007; Cutler et al. 2011), and most did so only in passing. This is probably in large part because of obstacles posed by U.S. mortality data. Information on educational was absent from vital statistics data until 1989 (National Center for Health Statistics 1993) and is perhaps too inaccurate (Rostron, Boies, and Arias 2010) for studying fine-grained compositional changes in education. National surveys generally measure educational attainment more accurately, but record far fewer deaths and cover only limited historical periods or age range.

The chapter is divided into several sections. The first section reviews the causation-selection debate in the literature and previous evidence in support of each explanation. In the second section, I develop a working definition of the effect of changing educational composition on change in mortality risk over time (i.e., the "compositional effect"). The third section describes the primary data source, the National Health Interview Surveys from 1997 to 2013, and the analytic plan. In the fourth section, I evaluate the extent to which the compositional effect accounts for within and between group trends in the risk of mortality over the study period. The fifth and final section discusses the theoretical and policy implications of the findings.

BACKGROUND

Four groups of mechanisms purport to explain the widening education gradient in U.S. adult mortality: (I) socioeconomic factors; (II) psychosocial factors; (III) health lifestyles; and (IV) change in the composition of education groups over time. With the

exception of the fourth, which is the focus of this chapter, all other mechanisms consider the effect of education on health and mortality to be causal.

Educational attainment is perhaps the single most important predictor of health and mortality in the U.S. of all SES indicators (Elo 2009). Highly educated individuals possess both material and nonmaterial resources that allow them to benefit from medical technology, social environments, and lifestyles that are conducive to better health and lower risk of mortality (Link and Phelan 1995). In terms of material resources, education paves the way to better occupations, higher income, and greater access to health care (Cutler and Lleras-Muney 2006). Montez and Zajacova (2013b) found that economic circumstances explain as much as a third of the growing education-mortality gradient among white women aged 45-84 between 1997 and 2006. Although employment status had the largest effect, poverty status, home ownership, health insurance, and type of occupation all contributed to growing mortality differentials. Higher levels of education also confer important nonmaterial resources such as power, prestige, social capital, and access to information, which in turn lead to better health outcomes. For example, highly educated individuals were quicker to quit smoking following to the Surgeon General's report in 1964 and were more likely to influence their highly educated peers to do the same (Christakis and Fowler 2008). Similarly, high SES individuals were first to use statins in the late 1980s, which led to a reversal in the association between income and cholesterol from positive to negative (Chang and Lauderdale 2009).

The association between education and health and mortality is also hypothesized to operate via psychosocial factors. According to the human capital model of learned

effectiveness (Mirowsky and Ross 2003), schooling enhances individuals' analytical and problem solving skills, self-motivation, and sense of personal control—all of which coalesce to encourage healthy lifestyles. Indeed, highly educated individuals are less likely to smoke and drink excessively and are more likely to exercise, maintain a healthy body weight, get regular health checkups and wear seatbelts (Cutler and Lleras-Muney 2010). Importantly, the positive effects of learned effectiveness operate over and above any material resources accrued from higher levels of education. By contrast, lack of autonomy, self-esteem, or sense of mastery over one's daily work and living environments can induce chronic stress, which impairs both physical and mental health (Thoits 2010).

Although learned effectiveness can explain the existence of health disparities at a given point in time, it is less clear how the theory can explain increasing health differentials over time. First, it is unclear at which level of educational attainment these effects are concentrated (e.g., primary, secondary, or post-secondary education) and whether there are diminishing returns to education at the individual level. The effect of education on population health may in fact be underestimated in societies where the completion of secondary education is commonplace (Baker et al. 2011), implying that learned effectiveness should explain *less* of the education-health gradient with the expansion of education. Second, the theory does not explain why health and mortality outcomes worsen over time among groups of fixed education levels, as is evident among white Americans with fewer than 12 years of schooling at least since 1990.

Yet another psychosocial mechanism through which education affects health is social ties—and perhaps marriage above all other ties. Highly educated individuals are more likely to be married than their low educated counterparts and marriage fosters social support and control which benefit both physical and mental health (Hughes and Waite 2009; Liu and Umberson 2008) as well as lower mortality risk (Dupre, Beck, and Meadows 2009). Furthermore, educational assortative marriage has become increasingly commonplace in the U.S. since the 1960s (Hou and Myles 2008; Schwartz and Mare 2005) and spousal education has a positive effect on one's health over and above their own level of education (Brown, Hummer, and Hayward 2014). In other words, higher levels of education lead to more sustainable marriages with similarly educated spouses, resulting in greater health returns over the life course.

The third mechanism concerns health lifestyles, which are defined as "collective patterns of health-related behavior based on choices from options available to people according to their life chances" (Cockerham 2013:55). Although disparate health behaviors come into play as proximate causes of mortality in the previous mechanisms, health lifestyles refer to more consistent patterns of behavior that are shaped by the interplay of life choices (agency) and life chances (structure). Moving beyond methodological individualism, which reduces all mechanisms to the individual level, the health lifestyle model adopts a Bourdieusian perspective whereby lifestyles persist in stratified societies as means for class differentiation (Bourdieu 1984). Lifestyles are therefore more than the mere aggregation of individual decisions and behaviors and, in much the same way, educational attainment does more than simply increase individuals'

effective agency. For example, low SES women claim that smoking provides them with opportunities to socialize and cope with stress (Stewart et al. 2011), which may explain their high prevalence of smoking (Centers for Disease Control and Prevention 2009) despite the near-ubiquitous knowledge that cigarette smoking is hazardous to one's health across all education levels (Link 2008). Addressing health lifestyles as coherent, macro-level social structures is beyond the scope of this chapter. However, it turns out that population changes in smoking and obesity (strictly, a risk factor and not a behavior) explain less than ten percent of the growth in the education-mortality gradient from the 1970s to the 1990s (Cutler et al. 2011). Thus, although lifestyles and behaviors matter for health and mortality outcomes, they do not appear to account for much the increasing association between education and mortality.

An alternative, fourth explanation for the increasing education gradient in U.S. adult mortality—and the primary focus of this chapter—is the changing composition of education groups over time. Figure 4.1 shows the distribution of educational attainment among white Americans aged 25 and over from 1940 to 2010. In 1940, nearly 74 percent of the adult white population attained fewer than 12 years of schooling and only five percent completed 16 or more years. By 2010, less than 8 percent of the population had not finished at least 12 years of schooling and over 30 percent completed college education or higher. Over the same period, the proportion of high-school educated whites (having completed 12 years of schooling) increased from 15 to 37 percent, and the proportion of those with some college education (13-15 years) increased from nearly 6 percent in 1940, peaked at about 26 percent in 1990, and declined to 23 percent in 2010.

As far as absolute levels of education are concerned, the expansion of education since WWII and into the twenty first century had transformed the "low educated" group from an overwhelming majority to a small minority. By contrast, nearly one in every three white Americans is now college educated, compared to only one in twenty just seven decades ago.

A recent study based on the Danish Twin Registry found that the association between education and mortality is smaller among dizygotic twins relative to general population, and disappears entirely among monozygotic twins (Behrman et al. 2011). Based on those results, the authors conclude that the association is largely spurious and reflects preexisting differentials in individual endowments (e.g., genetics and cognitive ability) and family social background. For example, early life intelligence predicts educational achievements (Deary et al. 2007; Neisser et al. 1996) and adult mortality risk (Batty et al. 2007; Deary et al. 2008), although schooling itself is known to increase IQ scores (Brinch and Galloway 2012). Family social background is also known to predict both academic achievement (Sirin 2005) and adult mortality (Hayward and Gorman 2004). If the association between education and mortality is explained by a third group of factors, then the widening education-mortality gradient may be attributed to change in the composition of education groups (Goesling 2007). While the "average" American in 1940 did not graduate from high-school, an individual with the same absolute level of education would now be placed at the extreme left tail of the distribution. In other words, the low educated have become more negatively select on those unobserved factors which determine low educational attainment (in relative terms) and high risk of mortality. At the

same time, college educated Americans have become less positively select over time because they represent a greater share of population at the right tail of the distribution. Insofar as the education-mortality gradient is measured between education groups defined by absolute levels, the compositional change alone can explain why the gradient in widening.

To date, few studies have attempted to address the compositional effect empirically. Using data from the National Health Interview Surveys from 1982 to 2004, Goesling (2007) found that the rise in the educational disparity in self-rated health was partially attenuated when education groups were broken by terciles instead of absolute levels of education. Similarly, Cutler and colleagues (2011) examined the extent to which changes in the distribution of risk factors explain the growing education-mortality gradient in the U.S. from the 1970s to the 1990s. To account for compositional changes, they first predicted individuals' propensity of attending college by age, region, marital status, and income. Next, they ranked the respondents according to the predicted propensity and repeated their analysis using education groups of the same proportion (or rank) over time—arriving at the same substantive conclusions. However, both studies used broad education categories (two or three) such that compositional effects are likely to be minor (indeed, the "low educated" category comprised between 33 and 44 percent of the study population in each of those studies *after* adjusting for change in composition). Furthermore, neither of those studies evaluated the extent to which the compositional effect accounts for change in the *gross* association between education and mortality.

In this chapter I present new evidence on the selection-causation debate by addressing the compositional effect and its role in explaining: (1) increasing mortality among white Americans with fewer than 12 years of schooling (the "low educated"); (2) the widening education-mortality gradient between low- and college-educated whites. In addition, I provide a descriptive account on how the characteristics of low-educated Americans have changed throughout the study period. In the next section I develop a precise definition of the compositional effect.

DEFINING THE COMPOSITIONAL EFFECT

To say that a certain education group becomes more or less select over time presupposes a generative mechanism which determines both educational attainment and longevity, in a probabilistic sense, at the individual level. The generative mechanism, *g*, can be biological (genetics), psychological (cognitive ability), sociological (family social background), or any combination of the three. Dividing the population into discrete education groups therefore correlates to group differences in both *g* and in mortality. An arbitrary change in the proportion of discrete education groups can produce observable changes in mortality both within and between those groups even if the distribution of *g* in the population is time invariant and education has no causal effect on mortality—this is essentially the compositional effect. Provided that the generative mechanism is largely unchanged, the compositional effect can be eliminated by fixing the proportion and rank of education groups over time.

Define $R(a \leq Educ < b)_0$ as the expected mortality risk of individuals whose educational attainment level is greater than or equal to a and smaller b at baseline (time

0). Similarly, $R(a \leq Educ < b)_t$ is the expected risk for the education group defined by the same constants *a* and *b* at a later time *t*. If F_t is the cumulative distribution function of education at time *t* and Q_0 is the quantile function of education at baseline, then $R(Q_0[F_t(a)] \leq Educ < Q_0[F_t(b)])_0$ is the expected mortality risk at baseline for individuals whose educational rank is between the percentiles of *a* and *b* at time *t*. For example, suppose that individuals with 11 or fewer years of schooling comprised the bottom decile of the population at time *t*. Then $Q_0[F_t(11)]$ defines the number of years of schooling in the bottom decile of the population at time 0.

For any fixed educational attainment group, the change in the expected risk of mortality between baseline and time *t* is simply

$$\Delta = R(a \leq Educ < b)_t - R(a \leq Educ < b)_0 \quad (4.1)$$

Next, holding the proportion and rank of the education group constant over time, define the composition-adjusted change in mortality risk as

$$\Delta_{adj} = R(a \leq Educ < b)_t - R(Q_0[F_t(a)] \leq Educ < Q_0[F_t(b)])_0 \quad (4.2)$$

and the compositional effect can be found by subtracting the composition-adjusted change from the total change in expected risk

$$\Delta_c = \Delta - \Delta_{adj} = R(Q_0[F_t(a)] \leq Educ < Q_0[F_t(b)])_0 - R(a \leq Educ < b)_0 \quad (4.3)$$

These definitions can be understood more intuitively from Figure 4.2. The (hypothetical) risk of mortality is plotted against time for two groups: (a) low educated individuals (e.g., having 11 or fewer years of schooling) in blue; (b) the corresponding quantile of the low educated at time *t* in red. By construction, the lines intersect at time *t*, when the absolute and relative definitions of low education coincide. The dashed line

marks the risk of mortality at the point of intersection at time *t*. It can easily be seen that the difference between the dashed line and the blue line at time 0, which equals the total change in mortality risk, is the sum of two components: (I) the difference between the dashed line and the red line (the composition-adjusted change in mortality risk); and (II) the difference between the red line and the blue line (the compositional effect).

Similarly, the compositional effect can be determined for any other education group. The college-educated, for example, have become less *positively* select over time despite continued reductions in mortality. In this case, the composition-adjusted component and the compositional effect will likely offset each other to some degree. The next section describes the data and methodology used to empirically estimate the compositional effect among white Americans since the late 1990s.

METHODOLOGY

Data

My evaluation of the extent to which the compositional effect accounts for within and between group trends in the risk of mortality draws on nationally representative data from the Integrated Health Interview Series (IHIS) from 1997 to 2013 (Minnesota Population Center and State Health Access Data Assistance Center 2012). The IHIS is a harmonized dataset based on the National Health Interview Survey and Linked Mortality Files (National Center for Health Statistics 2013)—an annual, cross-sectional sample of the non-institutionalized U.S. population. Since 1997, one adult in each sampled household is selected at random to answer an extended health questionnaire on health

behaviors, risk factors, functional limitations, and diagnosed health conditions (all of which are self-reported). Although the NHIS is cross-sectional, a mortality status is assigned to each survey participant based on linkage with the National Death Index in a follow-up period (currently, through December 2006). The analysis is restricted to non-Hispanic white respondents aged 45 to 84 because few deaths are recorded below age 45 and age reporting is top-coded at 85. I further divided the sample into two periods: 1997-2000 and 2010-2013. The final sample consisted of N=88,357 respondents and a total of 5,181 deaths, all of which are attributed to respondents from the first study period.

The IHIS provides several advantages over alternative data sources. First, it draws a large annual sample (over 100,000 respondents in 40,000 household every year) which allows an analysis of temporal trends. Second, at least for a subset of the participants, it includes detailed information on health behaviors, risk factors, and diagnosed health conditions (collected consistently since 1997). Third, information on the highest level of educational attainment, the primary predictor variable of interest in this study, is reported in single years through 12^{th} grade and in detailed degree categories thereafter. I recoded the latter into completed years of schooling in the following manner: the categories "12th grade, no degree," "high school graduate," and "GED or equivalent" were coded as 12 years; some college education and associate's degree were coded as 14 years; a bachelor's degree was coded as 16; and all higher levels of education (master's, professional, and doctoral degrees) were coded as 18 years. In addition, I created four categories based on completed years: less than high school education (0-11), high-school

(12), some college (13-15), and college or higher (16+). Educational attainment was missing in less than one percent of the data, in which case the observation was omitted.

Analytic Plan

The main objective of this study is to estimate how much of the change in the risk of mortality is attributed to the expansion of education in the population. However, mortality follow-up is unavailable for recent survey years. In order to overcome this limitation, the analysis consists of three steps:

1. Estimate the probability of mortality over a six-year period for 1997-2000, the baseline period, for which mortality follow-up is available.
2. Predict the six-year probability of mortality for 2010-2013 based on observed health behaviors, risk factors, and multiple health indicators.
3. Use Equation 4.1 to estimate the change in the predicted risk of mortality (i.e., the probability of dying within six years) over time for each education group.
4. Use Equation 4.3 to estimate how much of the total change in the risk of mortality for low and high education groups is explained by change in group composition. Similarly, estimate how much of the education-mortality gradient between those groups is explained by compositional changes.

In Step 1, I use a logistic regression model to estimate the probability of mortality during a six-year follow-up based on all available information in the NHIS on health behaviors (e.g., smoking, alcohol consumption, physical exercise), risk factors (e.g., body mass index, hypertension), general health status (e.g., self-rated health), functional limitations

(e.g., activities of daily living), and previously diagnosed health conditions (e.g., cancer, stroke, heart attack, diabetes). Rather than choose the most important predictors a priori, the model includes a total of 113 variables (see Appendix D for a complete list), in addition to age and gender. A complex model, however, runs the risk of over-fitting the data and therefore increasing the out-of-sample prediction error. I use the lasso estimator (Tibshirani 1996; Park and Hastie 2007) to constrain model parameters by adding a shrinkage penalty, much like in ridge regression, which reduces the out-of-sample prediction error by trading bias for variance. Unlike ridge regression, the lasso also acts as a variable selection method because some regression coefficients are shrunk exactly to zero.[2]

The lasso logistic regression yielded 59 non-zero parameters from the original 113 (highlighted in Appendix D), which were then used to predict the six-year probability of death (Step 2). Educational attainment was purposely omitted from the prediction model, so that change in the predicted risk of mortality can be tracked across both relative and absolute levels of education. The prediction rests on the assumption that, conditional on health behaviors, risk factors, and other health status indicators, the six-year probability of death does not depend on educational attainment. I address this assumption in the next section.

In Step 3, the expected risk of mortality for each education group is obtained by averaging over the predicted probabilities of its members (stratified by age and gender) in each study period. Step 4 repeats the process for education groups defined by rank, rather than absolute number of completed years of schooling (the exact number of years is

randomly perturbed to ensure that no ranks are tied). Finally, I use Equations 4.1-4.3 to evaluate the total change in the probability of death and the share explained by compositional changes.

RESULTS

Change in Group Characteristics

Although the study dates back only to 1997, it nevertheless captures some of the educational expansion that has characterized American society in the past decades. Table 4.1 summarizes the change in the distribution of educational attainment among non-Hispanic whites, aged 45-84, between the two study periods. Between 1997-2000 and 2010-2013, the percentage of low educated whites (both genders combined) declined by 7.1 percentage points, from 15.2 to 8.1, and the percentage of high-school educated whites declined from 35.5 to 30.5. At the same time, the percentage of those with some college education reached nearly 30 and the percentage with college education or higher increased from 24.2 to 31.8. Overall, low and high-school educated whites have become more negatively select, comprising a smaller share of the population that is increasingly concentrated at the bottom of the education distribution. Those who attended college, on the other hand, have become less positively select as higher education became more prevalent in the population.

Throughout the rest of the section I focus primarily on results concerning the lowest education group, because Chapter 2 found that mortality has been increasing in that group since 1990. A central question in this chapter is whether the rise in mortality is

"real," in the sense that the life chances of low SES Americans have worsened in recent decades, or instead are attributed to change in the group's composition over time. Since the chapter is also concerned with the widening education-mortality gradient—defined here as the difference in the risk of mortality between the least- and most-educated groups—I show results for the latter as well. However, the change in composition of high education groups cannot explain their continued reductions in mortality because they have become *less* positively select over time. If anything, the compositional effect would offset some of those mortality reductions.

A complete investigation of the causal mechanisms discussed above is beyond the scope of this chapter (and hindered by data limitations). However, Table 4.2 shows the distribution of key factors thought to explain the association between education and mortality, and, by extension, the change in the risk of mortality over time. These include demographic characteristics (age and gender), psychosocial factors (marital status), socioeconomic factors (income below poverty threshold, employment status, and home ownership), and health behaviors and risk factors (smoking and body mass index). Column I and II of Table 4.2 summarize the group characteristics in 1997-2000, where low education is defined by absolute years of schooling (I) or the bottom 8.1 percent of the education distribution (II). Column III shows the same characteristics in 2010-2013, in which the two definitions of low education coincide. Finally, the two rightmost columns show the difference in characteristics over time using absolute and relative definitions of low education, respectively.

First, I report changes in the characteristics of those with 11 or fewer years of education. Between 1997-2000 and 2010-2013, the mean age declined slightly from 66.4 to 64.8 and the percentage of women in the group declined from 54.2 to 48.8. By 2010-2013, the group was less likely to be married (4.6% decline), the poverty rate increased from 17.9 to 26.2 percent, and home ownership declined from 78.1 to 70.8 percent. There was no significant change in employment rate. Similarly, the proportion of former smokers did not change between the two periods, but the share of current smokers increased significantly from 24.8 to 32.0 percent. Mean body mass index also increased by 1.2 units. Overall, the low educated appear to be doing worse on a variety of factors associated with high risk of mortality: they are less likely to be married, employed, or own a home and more likely to be poor, overweight, and current smokers. At the same time, the mean number of years of schooling appears to have changed only slightly from 8.7 to 8.9 years. Taken together, these patterns suggest that returns to low levels of education have changed for the worse.

Evaluating the Compositional Effect

Since the low educated have become increasingly concentrated at the bottom tail of the education distribution, there is reason to believe that some of the changes in group characteristics are due to the change in composition rather than change in returns to education. The rightmost column of Table 4.2 compares the characteristics of the bottom 8.1 percent of the education distribution between 1997-2000 and 2010-2013 (in which both absolute and relative definitions of "low education" coincide). Although many of the changes are attenuated, they have not completely disappeared. Using the more stringent

definition of low education, the percent married declined by 4.6 percent, the poverty rate increased by 4.6 percent, home ownership declined by 5.6 percent, and both body mass index and the share of current smokers increased as much, if not more, than before. Although the compositional effect does account for some changes in group characteristics, it does not account for all of them. Insofar that these factors explain the association between education and mortality, and change therein, the compositional effect should not fully account for increasing mortality among low educated whites. However, the role of the hypothesized causal mechanisms cannot be tested directly in this study.

Next, I turn to evaluate the extent to which compositional changes explain gross mortality trends within and between low (0-11) and high education (16+) groups. Figure 4.3 shows the change in the predicted probability of death over a six-year period for low and high educated white men. The total change is further decomposed into the composition-adjusted change in the predicted probability (blue) and the compositional effect (red). Between the two study periods, the predicted probability of mortality increased by 0.012 for the low educated at age 55 and by 0.016 for those aged 75. The compositional effect explains 49 and 66 percent of those increases, respectively. Among the high education group, the predicted probability of mortality declined slightly at age 55 (-0.002) and more so at age 75 (-0.007). The composition-adjusted reductions in the probability of mortality were in fact greater, but were offset by the compositional effect because the high education group became less positively select.

Figure 4.4 shows similar results for white women. First, the increases in the six-year probability of death were greater among the low educated compared to men of the

same level of education. This is consistent with findings in Chapter 2. Furthermore, the compositional effect accounted for only 10 to 21 percent of the total increase—much less than it did among men. Second, college-educated women experienced net reductions in the probability of mortality that were similar to those among college-educated men. Here too, the compositional effect offset some, but not all, of those reductions.

Finally, a difference-in-differences calculation reveals that the compositional effect explains less than 22 percent of the rising education-mortality gradient among men and less than 10 percent among women.

Consistent with prior research, the results in Figures 4.3 and 4.4 find an absolute increase in the risk of mortality among low educated whites of both genders. At the same time, mortality declined among the highly educated. Changes in the composition of education groups appear to have contributed negatively to both low and high educated groups. However, the compositional effect accounts for only 10 to 66 percent of the increase in mortality among the low educated (more so for men than for women). Mortality reductions among the highly educated were large enough to surpass the negative contribution of the compositional effect. In other words, returns to education among high education groups have increased over and above the reductions associated with change in group composition. By contrast, conditions have worsened for the low educated—particularly for women—over and above what can be explained by the compositional effect.

Evaluating Model Assumptions

The analysis is based on the predicted probability of mortality, rather than the actual risk, because mortality status is unobserved in 2010-2013. The prediction rests on the assumption that the probability of death *conditional* on observed health behaviors, risk factors, and health status is constant over time, such that group differences in mortality are explained only by changes in the distribution of those factors. This assumption is likely untrue because there may still be differentials in the conditional survival by educational attainment.[3] Indeed, the lasso model predicted 7.7 percent fewer deaths among the low education group and 35.8 percent more deaths among the high education group than observed in 1997-2000. The conditional risk of mortality is therefore underestimated among the former and overestimated among the latter. The real education-mortality gradient—and change therein—is in all likelihood greater than reported here.

DISCUSSION

The tension between causation and selection on some unobserved factors is inherent in all observational studies and has been especially visible in research on the association between educational attainment and adult mortality. There is no denying that the association is pervasive (Baker et al. 2011), enduring (Miech et al. 2011), and increasing in magnitude (Meara, Richards, and Cutler 2008; Montez et al. 2011; Olshansky et al. 2012; Preston and Elo 1995). Yet there are those who argue that the association is largely spurious because preexisting individual and family endowments—

be they biological, psychological, or sociological—determine both educational attainment and adult survival. Evidence from a Danish twin study, the "gold standard" in social science research, concluded that the effect of educational attainment on hospitalization and mortality disappears among monozygotic twins (Behrman et al. 2011). In other words, highly educated individuals are already select on a variety of factors before they complete their schooling—factors that also increase their adult survival chances. Even if selection accounts for only a portion of the association, the expansion of education in the U.S. since WWII has been so dramatic that it calls into question whether the increase in the education-mortality gradient is driven by change in the composition of education groups (Goesling 2007). Defining education groups in terms of absolute levels of education overlooks those compositional changes and potentially overestimates education's causal effect on adult mortality. Indeed, Americans with fewer than 12 years of schooling are increasingly concentrated at the bottom of the education distribution whereas college-educated Americans are becoming less positively select over time.

Separating the "compositional" and the "real" change in the risk of mortality among different education groups is important for both theoretical and practical reasons. One of the core tenets of sociology is that class circumstances determine individuals' life chances (Weber 1946), and the role of socioeconomic status and education in particular in shaping health outcomes is expected to increase as societies gain a greater capacity to control health and disease (Link and Phelan 1995; 2002). The causal argument suggests that high SES individuals are better situated to benefit from those advances, whereas the cumulative exposure to social disadvantage among low SES individuals takes a toll on

their health (Dannefer 2003). According to this framework, the increasing education-mortality gradient is attributed to change in health returns to education. The causal argument also has practical implications because, if true, it supports the creed that social policy is health policy: increasing high school graduation rates and access to college education will work to improve population health and reduce health inequality.

This chapter finds that, between 1997 and 2013, compositional changes in education account for roughly half of the increase in the six-year probability of death among white men (aged 45-84) with fewer than 12 years of schooling and less than a fifth of the increase among similarly educated white women. At the same time, college-educated men and women experienced mortality reductions over and above the negative compositional effect. Less than a quarter of the increase in the education-mortality gradient is explained by the change in composition of those education groups. These results imply that white Americans at the bottom of the education distribution are losing ground in absolute terms. This is especially true for low-educated women, who have seen greater increases in mortality than men—most of which cannot be attributed to changes in group composition.

The results further show that socioeconomic circumstances have worsened for low education groups *after* adjusting for change in composition. By 2013, the low educated were less likely to be married or own a home and more likely to be poor. The part- or full-time employment rate, which was previously shown to predict change in the risk mortality (Montez and Zajacova 2013b), did in fact improve slightly over the study period. Smoking prevalence and body mass index also increased among the low

educated, placing them at greater risk of mortality. These results are consistent with the polarization of the U.S. labor market and the increasing concentration of resources among high education groups (Autor and Dorn 2013; Autor, Katz, and Kearney 2006). It is not immediately clear, however, why health behaviors (smoking) and risk factors (body mass index) have worsened among the low educated. In part, they may have to do with increasing economic hardship (Ross and Wu 1995) or be part of a greater change in health lifestyles associated with class circumstances (Cockerham 2013).

One limitation of this study is that mortality status is unobserved for NHIS respondents in 2010-2013. The analysis therefore relies on the predicted probability of death based on observed health conditions and known risk factors. In order to overcome this limitation, I use the lasso estimator (Tibshirani 1996) to maximize the amount of relevant information for predicting mortality while minimizing the prediction error (this is done by splitting the data into training and test sets for cross-validation). However, the resulting model appears to underestimate mortality among low education groups and overestimate it among high education groups, which will act to underestimate the education-mortality gradient. In other words, results in this study are likely conservative. In addition, this method does not account for changing education differentials in survival *conditional* on the factors used to predict mortality. In spite of the limitations, this study joins the few empirical attempts made to address the compositional effect on the changing association between education and adult mortality in the U.S. and in general.

Life expectancy has been declining among low educated white Americans since 1990 (Chapter 2) and in large part because of smoking behavior (Chapter 3). At the same

time, mortality continued to decline among high education groups, which in turn led to a widening education gap in life expectancy. Claims that these patterns reflect the increasing negative selection of low education groups find minimal evidence in this chapter. Mortality is increasing in absolute terms for non-Hispanic white Americans at the bottom of the education distribution—a group subjected to increasingly worse economic circumstances. As far as proximate causes are concerned, smoking behavior is probably the main contributor to rising mortality among the low educated as well as to increasing mortality differentials relative to the college educated (see Chapter 3). Focusing on the changing distribution of educational attainment appears to be only one part of the explanation. This is because education is a changing social structure that reflects more than the average level of educational attainment in the population. An important next step is to understand how and why health returns to education have changed over time, both at the individual and at the population level.

Notes:

1. Author's calculation of the U.S. Census Integrated Public Use Microdata Sample (Ruggles et al. 2010).

2. In the context of generalized linear models, the lasso estimator is the solution to

$$\hat{\beta}(\lambda) = \arg\min_{\beta}[-log\{L(\boldsymbol{y};\beta)\} + \lambda\|\beta\|_1]$$

   where β is a vector of regression coefficients, $L(\boldsymbol{y};\beta)$ is the likelihood function, $\lambda > 0$ is the regularization (or tuning) parameter, and $\|\beta\|_1$ is the L_1 norm. The tuning parameter, which controls the amount of shrinkage, can be optimized by retaining a subset of "unseen" data for cross-validation.

3. For example, Cutler and colleagues (2011) found that population changes in smoking behavior and obesity explained less than ten percent of the growth in the education-mortality gradient between the 1970s and 1990s, and attribute the rest to changes in educational returns to those behaviors and risk factors.

Table 4.1: Change in the distribution of educational attainment among non-Hispanic white Americans, National Health Interview Surveys 1997-2013

	Period		
Education level	**1997-2000**	**2010-2013**	**Difference**
Low (0-11 years)	15.2%	8.1%	-7.1%
High school (12)	35.5%	30.5%	-5.0%
Some college (13-15)	25.1%	29.6%	4.5%
College or higher (16+)	24.2%	31.8%	7.7%
Total	100.0%	100.0%	-

Notes: Sample restricted to non-Hispanic whites aged 45-84; Adjusted for sampling weights.

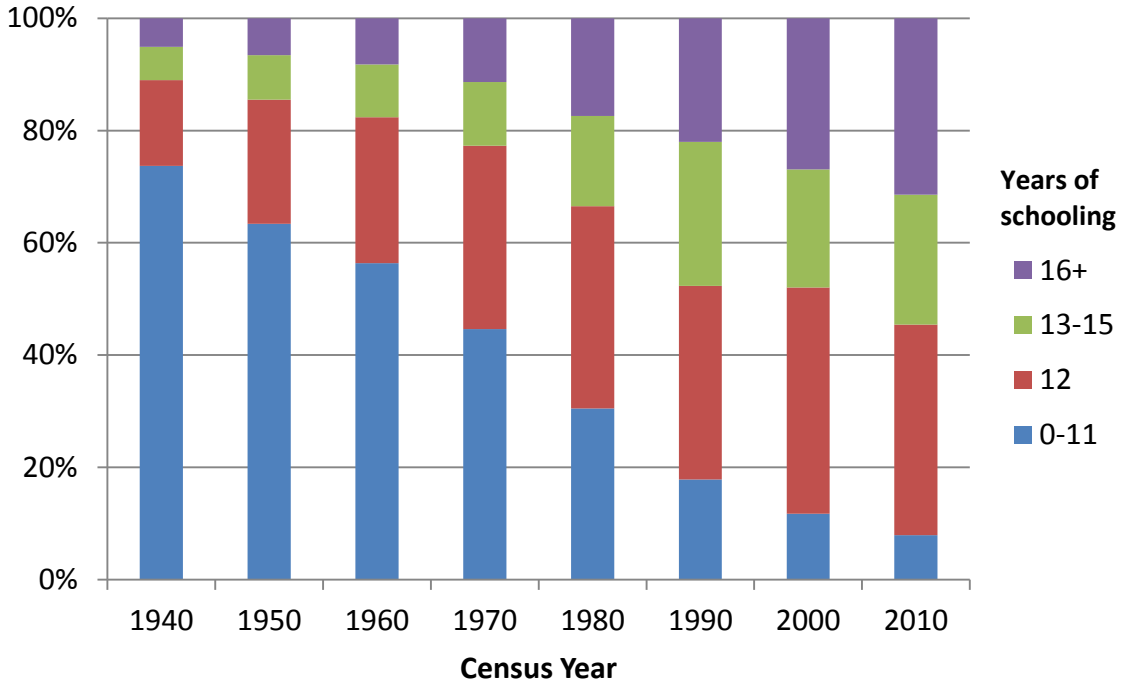
Table 4.2: Change in characteristics of low educated, non-Hispanic white Americans aged 45-84, National Health Interview Survey 1997-2013

	1997-2000		2010-2013	Difference	
	(0-11 years)	(Bottom 8.1%)	(0-11 years and bottom 8.1%)	Fixed Level	Fixed Quantile
Variable	**I**	**II**	**III**	**III-I**	**III-II**
Demographic characteristics					
Age (mean)	66.4	67.9	64.8	-1.6***	-3.1***
Women (%)	54.2	51.3	48.8	-5.4***	-2.5*
Social support					
Married (%)	58.8	56.6	52.0	-6.8***	-4.6***
Socioeconomic factors					
Income below poverty threshold (%)	17.9	21.6	26.2	8.3***	4.6**
Employed part- or full-time (%)	24.1	19.6	22.3	-1.8	2.7*
Home ownership (%)	78.1	76.4	70.8	-7.4***	-5.6***
Health behaviors and risk factors					
Current smoker (%)	24.8	23.0	32.0	7.2***	9.0***
Former smoker (%)	37.3	37.3	36.7	-0.7	-0.6
Body mass index (mean)	27.3	27.2	28.5	1.2***	1.3***
Years of schooling (mean)	8.7	7.2	8.9	0.3***	1.7***
N	7,223	3,883	3,947		

* $p < 0.05$; ** $p < 0.01$; *** $p < 0.001$ (two-tailed tests)

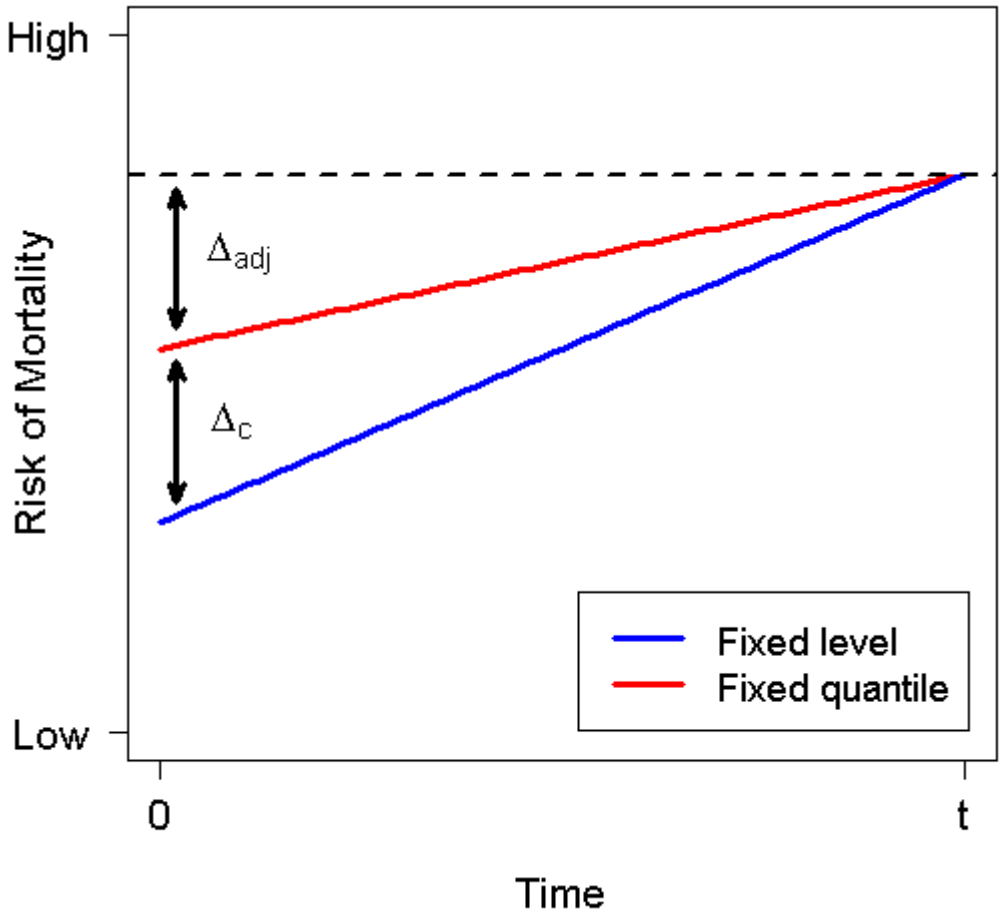
Notes: Low education = 0-11 years of schooling or lowest 8.1% of the education distribution; Adjusted for NHIS Adult Sample weights and sampling design.

Figure 4.1: Educational attainment among non-Hispanic white Americans aged 25 and over by census year, 1940-2010



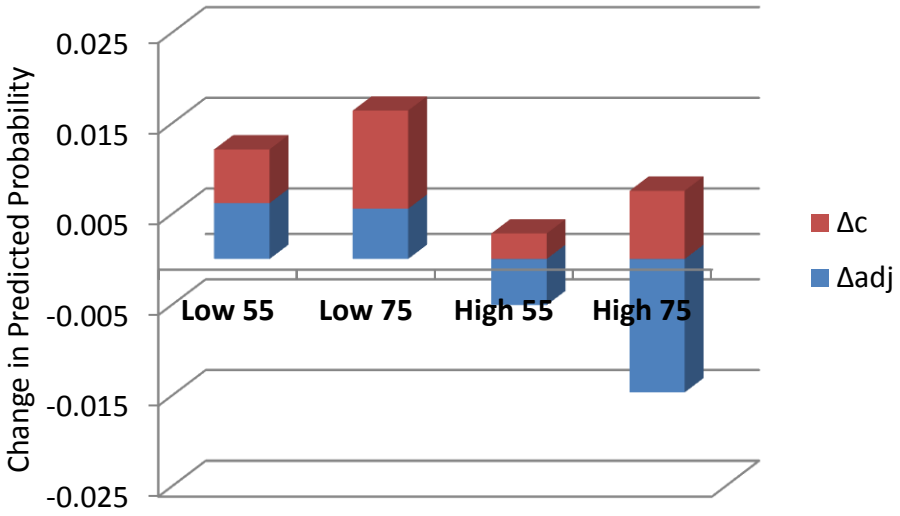
Source: Author's calculation of the U.S. Census and American Community Survey data Integrated Public Use Microdata Series (Ruggles et al. 2010).

Figure 4.2: Illustration of the compositional effect and the composition-adjusted change in mortality risk for a hypothetical low education group



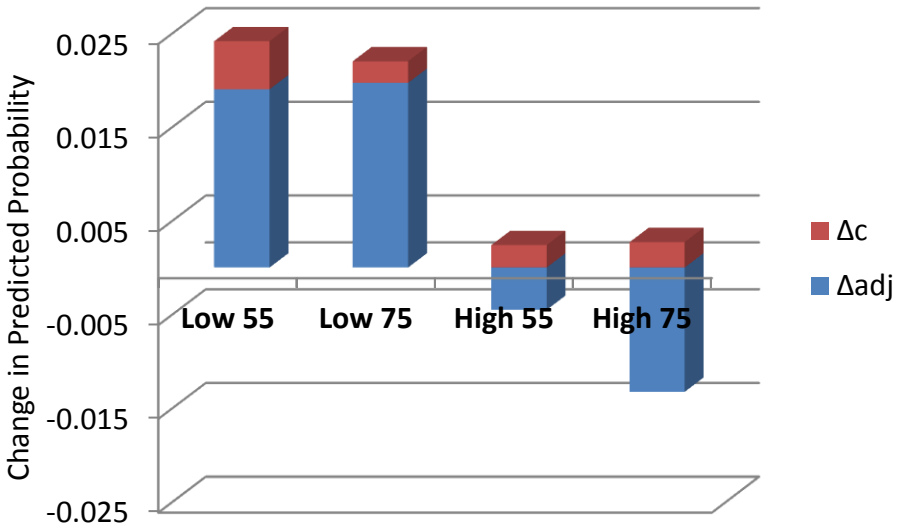
Notes: The risk of mortality between time 0 and time *t* for a fixed education level (e.g., 0-11 years of schooling) is shown in blue, whereas the change in the risk of mortality for a fixed education quantile is shown in red. The lines intersect at time *t* when the fixed quantile coincides with the proportion of the low education group. The difference between the dashed line and the red line at time 0, Δ_{adj}, is the composition-adjusted change in the risk of mortality; the difference between the red and the blue lines at time 0, Δ_c, is the change in mortality risk attributed to change in group composition (i.e., the "compositional effect").

Figure 4.3: Change in six-year probability of death between 1997-2000 and 2010-2013 by age and level of education, non-Hispanic white men



Notes: Δ_c = compositional effect; Δ_{adj} = composition-adjusted change; Low = 0-11 years of schooling; High = 16+ years of schooling; Predicted probabilities at ages 55 and 75.

Figure 4.4: Change in six-year probability of death between 1997-2000 and 2010-2013 by age and level of education, non-Hispanic white women



Notes: Δ_c = compositional effect; Δ_{adj} = composition-adjusted change; Low = 0-11 years of schooling; High = 16+ years of schooling; Predicted probabilities at ages 55 and 75.

Chapter 5: Conclusion

REVIEW OF FINDINGS

Educational differentials in U.S. adult mortality have drawn significant attention from sociologists, demographers, and public health scholars over the years (e.g., Hummer and Lariscy 2011; Meara, Richards, and Cutler 2008; Montez et al. 2011; Olshansky et al. 2012; Preston and Elo 1995). College-educated Americans live longer, on average, than their low-educated counterparts, and the education-mortality gradient has increased dramatically since the 1980s. Although several contemporary sociological theories explain why those differentials exist and why they might increase over time, the declines in life expectancy among low-educated white women and, to a lesser extent, among men, were truly unanticipated. However, debates surrounding the extent, causes, and even validity of those trends continue. The starting point of this dissertation was therefore to validate and expand our current knowledge on trends in and causes of lifespan inequality by educational attainment in the U.S. over the past two decades.

Chapter 2 relied on vital statistics data to estimate complete age-at-death distributions by educational attainment for black and white Americans of both genders and documented changes in the mean and standard deviation of those distributions over time. In the process, I developed a unique imputation method to handle missing data on educational attainment in the vital registry—a problem that is particularly severe in earlier years of the study period. The findings reveal that the gap in life expectancy at age 25, e^{o}_{25}, between the low and the college educated doubled among men and more than

tripled among women from 1990 to 2010. Among low-educated whites in particular, e_{25}^{o} declined by 0.6 and 3.1 years for men and women, respectively. The declines found in this study are significantly smaller than those reported by Olshansky and colleagues (2012) due to methodological differences in handling missing data and education categorization. Specifically, Olshansky et al. appear to underestimate e_{25}^{o} among low and high-school educated groups and overestimate e_{25}^{o} among those with some college education compared to results reported here. Nevertheless, the substantive conclusions remain: the education gap in life expectancy widened because mortality increased among low-educated white Americans and continued to decline among their college-educated counterparts. The educational disparity in mortality also grew among blacks, although no absolute declines in longevity were observed.

Chapter 2 goes beyond previous studies to document, for the first time, trends in lifespan variation by educational attainment for black and white men and women. To date, a handful of studies have shown—and only in cross-section—that high education groups benefit from lower age-at-death variation compared to low education groups. In their seminal work on the significance of education for mortality compression, Brown and colleagues (2012) found significant educational differences in SD(M+), the standard deviation of age at death above the mode, among non-Hispanic white Americans. Similar patterns were found in multiple European countries using other measures of variation (van Raalte et al. 2011, 2012). Chapter 2 builds on those studies by reporting trends in S_{25}, the standard deviation of age at death above 25, for non-Hispanic blacks and whites from 1990 to 2010. The results show that differences in S_{25} mirror those in e_{25}^{o} in

reverse—i.e., greater variation is found among low education groups relative to high education groups. Furthermore, S_{25} increased by about 1.5 years among the high-school educated in nearly all race-gender groups, with the exception of black men, and among low-educated white women. Over the same period, S_{25} showed little change among the college educated, estimated around 11.3 years for white women and 12.2 years for white men in 2010, and about one year higher among college-educated blacks in each gender group.

Importantly, low-educated blacks and whites appear to be converging in e_{25}^{o}, in each gender group, suggesting that race matters less for life expectancy at the bottom of the education distribution. The same cannot be said for the upper tail of the education distribution, where highly educated whites benefit from greater returns to education than blacks both in terms of life expectancy and in terms of lifespan variation. Also surprising is the fact that low-educated men and women in each race group are converging in terms of S_{25}, but not in e_{25}^{o}. Finally, Chapter 2 finds that educational differences in S_{25} are becoming increasingly important in explaining total lifespan inequality—the overall divergence in the distribution of age at death—by educational attainment, and in some cases even surpass differences in e_{25}^{o} (e.g., between high-school and college-educated white women). These findings underscore the importance of studying multiple dimensions of lifespan inequality in addition to differences in life expectancy.

Next, Chapter 3 uncovered the age- and cause-specific mortality patterns driving the trends found in Chapter 2, which reflect the risk profiles of different education groups and point to disparities in underlying health behaviors. Focusing on non-Hispanic whites,

the results show a clear education-gradient in the number of life years lost (YLL) from all major causes of death: infectious, cardiovascular, cerebrovascular, respiratory, and smoking-related diseases, as well as neoplasms, diabetes, external causes, and a residual category. Educational differences in YLL were greatest in cardiovascular diseases and (increasingly) in smoking-related diseases and external causes, which also accounted for much of the decline in life expectancy among low-educated men and women. Furthermore, increasing mortality in the middle ages (45-64) accounted for most of the decline in temporary life expectancy between ages 25 and 85 in those groups. Mortality also increased among high-school educated men and women under age 55, which offset gains in life expectancy due to mortality reductions in old age. This pattern explains the increase in S_{25} found in Chapter 2 because both components—increasing young-adult mortality and decreasing old age mortality—contribute to increasing variation in age at death (Gillespie, Trotter, and Tuljapurkar 2014).

Chapters 2 and 3 provided a thorough demographic account of how and why the education-mortality gradient increased in the U.S. between 1990 and 2010. Chapter 4 delved deeper into the sociological explanation underlying those patterns and trends. Specifically, it addressed the selection-causation debate surrounding the association between educational attainment and adult mortality. The causal perspective argues that increasing educational differentials in mortality reflect the rising important of education to health and adult survival (Link and Phelan 1995; Link 2008) whereas the selection argument suggests that another group of factors (e.g., cognitive ability, family background) determine both educational attainment and adult mortality risk (Behrman et

al. 2011). According to the latter perspective, change in the composition of education groups over time can explain the increasing mortality differentials even if the association between education and mortality is not causal (Goesling 2007). Chapter 4 sheds light on this debate by looking at changes in both socioeconomic circumstances and the risk of mortality of individuals at the bottom of the education distribution, regardless of their absolute level of education.

Results from the National Health Interview Survey from 1997 to 2013 reveal that compositional changes account for about one half to two thirds of the increase in the (predicted) six-year probability of mortality among low-educated white men and less than a fifth of the increase among low-educated white women. Over the same period, economic circumstances (e.g., poverty, home ownership), health behaviors (smoking), and risk factors (body mass index) worsened for those at the bottom of the education distribution whether they were measured in absolute years of schooling (0-11) or in relative position in the education distribution (the bottom 8.1%). By contrast, the predicted probability of mortality declined among college-educated whites despite the fact that they become less *positively* select over the study period. Overall, these results suggest that returns to education in terms of adult survival grew over time and that the increasing education-mortality gradient cannot be entirely accounted for by compositional changes in education groups.

IMPLICATIONS

The findings in this dissertation carry several direct implications for our understanding of social disparities in U.S. adult mortality: (I) increasing mortality among

low education groups in the U.S. cannot be explained away by changes in group composition or data quality issues, and likely stems from the worsening life circumstances of individuals with low socioeconomic status; (II) it is becoming increasingly evident that a significant segment of the U.S. population is not converging to the fourth stage of the epidemiological transition and may in fact be regressing; and (III) lifespan inequality extends beyond group differences in life expectancy. Group differences and trends in age-at-death variation in particular can shed light on future mortality scenarios for different subpopulations.

The "Life Chances" of Low Educated Americans

The association between educational attainment and adult mortality operates on multiple levels. At the individual level, schooling enhances cognitive abilities and problem-solving skills which coalesce to encourage healthy lifestyles and reduce the risk of mortality (Mirowsky and Ross 2003). At the societal level, the aggregate level of educational attainment in the population reflects the social, institutional, and technological capacity to control health and disease (Easterlin 1996). In other words, the absolute level of education matters to health and mortality outcomes on both micro and macro social scales. But the interaction between the micro and the macro also shapes individual health and mortality outcomes. Highly educated individuals relative to the rest of the population are better situated to benefit from the increasing social capacity to control health and disease. Indeed, this is Link and Phelan's (1995; 2002) key insight that social conditions are fundamental causes of disease.

Numerous studies have focused on the increasing education-mortality gradient, but few addressed the absolute increase in mortality among low and even high-school educated white Americans in certain age groups. A complete explanation of this trend not only requires that individuals with higher socioeconomic status (SES) possess *greater* access to healthy lifestyles and environments, but that conditions among low SES individuals deteriorate in absolute terms. Indeed, the U.S. labor market has become increasingly polarized since the 1970s (Autor, Katz, and Kearney 2006) and earnings inequality increased steeply (Kopczuk, Saez, and Song 2010). Low-skilled workers in particular experienced stagnation or decline in real wages (Autor and Dorn 2013), owing in part to the decline in unionization (Western and Rosenfeld 2011). These trends are consistent with the findings in Chapter 4, at least since the late 1990s, showing that poverty increased and home ownership declined among low-educated whites—factors associated with higher risk of mortality (Montez and Zajacova 2013b; Rogers, Hummer, and Everett 2013; Ross and Wu 1995). Importantly, Chapter 4 also showed that both economic circumstances and the risk of mortality worsened for the low educated net of the change in group composition (i.e., increasing negative selection).

By 2013, the low-educated were also less likely to be married, placing them at a further disadvantage because marriage improves physical and mental health (Hughes and Waite 2009; Liu and Umberson 2008) and reduces the risk of mortality (Dupre, Beck, and Meadows 2009). Even among those who were married, educational homogamy increased since the 1960s (Schwartz and Mare 2005) and contributed to rising inequality in family earnings (Hou and Myles 2008). Educational homogamy in marriage also

affects health inequality because spousal education improves one's health over and above their own level of education (Brown, Hummer, and Hayward 2014).

Overall, it is clear that the "life chances" of low-educated Americans have changed for the worse, in the Weberian sense, whether educational attainment is measured in absolute or in relative terms. Findings in this dissertation further suggest that increasing mortality among low-educated whites cannot be explained away by changes in group composition or data quality issues, and likely stems from the worsening life circumstances of individuals with low socioeconomic status.

The Partial Reversal of the Epidemiological Transition

As far as proximate causes are concerned, smoking behavior underlies much of the increase in mortality among low education groups as well as the increase in the education-mortality gradient. Findings in this dissertation show that smoking prevalence increased for non-Hispanic whites with fewer than 12 years of schooling and, about the same time, that the number of life years lost due to smoking-related diseases increased in that group (the same cannot be said for blacks, for whom the number of life years lost to smoking plateaued for women and declined for men). Clearly, educational differences in period mortality from lung cancer, emphysema, and other smoking-related diseases in the present reflect past disparities in smoking prevalence. Indeed, educational disparities in smoking behavior have not only persisted but also widened for whites and blacks (but not Hispanics) since the mid-1970s (Pampel 2009). This is in spite of the fact that educational differentials in awareness to the health consequences of smoking (specifically, lung cancer) are significantly smaller (Link 2008). A more comprehensive

sociological explanation lies perhaps in health lifestyle theory (Cockerham 2013), whereby specific health practices are the result of dispositions to act in certain ways (habitus), which themselves are the product of interaction between agency and structure.

Among non-Hispanic whites, in all but the college educated, the number of life years lost from external causes of death increased dramatically for both men and women. These results are consistent with prior research which found that accidental poisoning (primarily drug overdose) was the top increasing cause of death among white men and women aged 45-64 between 1999 and 2007, and also the cause of death in which the educational disparity grew most (Miech et al. 2011). Deaths from external causes take an especially high toll on life expectancy because they tend to occur early in life. Preventing these premature deaths, along with those attributed to smoking, will have the greatest impact on reducing the educational gap in life expectancy.

The increase in middle-age mortality for low and high-school educated whites, a subpopulation which now comprises over a quarter of the U.S. adult population, is unprecedented. It also undermines the basic tenet that societies converge to the fourth stage of the epidemiological transition (Omran 1971; Olshanksy and Ault 1986), which has already been criticized following the reversal of mortality decline in Eastern Europe after the fall of the Soviet Union and in sub-Saharan Africa following the AIDS epidemic (Casseli, Meslé, and Vallin 2002; McMichael et al. 2004). Instead, Vallin and Mislé (2004) proposed to rethink the epidemiological (or health) transition as a series of divergence and convergence sequences resulting from changes in social conditions, health technologies, and emerging mortality risks. Taken to the subnational level, the

same logic can be applied to low SES groups in the U.S. While the cardiovascular revolution since the 1970s has benefited all education groups, to varying degrees (Yang 2008; Masters, Hummer, and Powers 2012), the rise in smoking-related and external causes of death among low education groups suggests a divergence, rather than convergence, in the health transition. Unless social conditions improve for low SES Americans, there is no reason to believe that they will catch up with those in the lead.

Lifespan Variation and Future Mortality

Acknowledging that lifespan inequality amounts to more than group differences in life expectancy, sociologists and demographers have recently garnered interest in other characteristics of age-at-death distributions and particularly in lifespan variation (e.g., Cheung and Robine 2007; Edwards and Tuljapurkar 2005; Kannisto 2000; Thatcher et al. 2010; Wilmoth and Horiuchi 1999). This interest is fueled by both theoretical and practical reasons. First, lower variation in age at death among high education groups reflects a higher capacity to optimize health outcomes over the life course (Brown et al. 2012) as well as lower uncertainty surrounding the length of life (Edwards 2013). The divergence in lifespan variation by different education groups constitutes an increasingly important component of *total* lifespan inequality and warrants additional research. Second, trends in lifespan variation provide insight into probable future scenarios of U.S. adult mortality for different subpopulations. Coupled with increasing life expectancy, decreasing lifespan variation over time is consistent with mortality compression (Fries 1983) whereas constant variation suggests mortality translation (Bongaarts and Feeney 2003; Canudas-Romo 2008). An entirely different scenario is the (unexpected) decline in

life expectancy among low education groups. Third, standard mortality modeling and forecasting methods generally assume constant variation in age at death between subpopulations or over time. Observing group differences or period changes in lifespan variation can potentially invalidate results based of those methods.

Results in Chapter 2 show that lifespan variation, measured by S_{25}, is not only higher among low and high-school educated Americans, but has also grown over time (especially among whites). Chapter 3 further finds that this trend reflects increasing young-age mortality coupled with decreasing old-age mortality in those education groups. Period mortality, however, conflates the experience of multiple birth cohorts by design. Past studies have shown that trends in cause-specific adult mortality in the U.S., including some of the leading causes of death, have largely operated on a cohort basis (Yang 2008). Furthermore, cohort effects underlie much of the increasing educational disparity in U.S. adult mortality (Masters et al. 2012). Taken together, there is reason to suspect that the mortality increase among high-school educated whites under the age of 55 will carry into old age as these cohorts mature.

Among college-educated whites, on the other hand, S_{25} remained constant throughout the study period while life expectancy continued to rise. The evidence therefore supports the mortality translation scenario rather than mortality compression, suggesting no immediate limit to lifespan. However, this conclusion is only tentative and may vary depending on how lifespan variation is measured.

Patterns and trends in lifespan variation also carry several methodological implications. Standard hazard models (e.g., Gompertz, logistic, Cox proportional

hazards) assume no group differences in age-at-death variation (Tuljapurkar and Edwards 2011)—an assumption that is clearly false when comparing mortality risk across gender, race, and education groups. Possible solutions are to model group differences in the age-slope of mortality in addition to difference in the intercept, add time-varying coefficients, or introduce frailty terms (e.g., the Gamma-Gompertz). As far as mortality forecasts are concerned, the Lee-Carter method (1992), the benchmark used by both the U.S. Census Bureau and the Social Security Administration (Meseguer 2008), does allow variation in age at death to change over time (Tuljapurkar and Edwards 2011). However, it assumes that the temporal change in mortality is fixed over age groups (i.e., there is no age × time interaction), which does not appear to hold for high-school educated whites, for whom mortality declined in old age but increased below age 55. Additional research is needed to assess the sensitivity of current mortality forecasts to those assumptions.

FUTURE RESEARCH

Moving forward, I propose to extend this line of work in three main directions: (I) the role of educational expansion in the increasing education-mortality gradient; (II) the intersection of race, gender, and education in shaping lifespan inequality; and (III) the consequences of lifespan variation to individual uncertainty in length of life.

The association between education and adult mortality operates on both the micro and macro social scales, as well as their interaction. However, sociologists often treat education as an individual characteristic taken out of social and historical context. The expansion of education changes both the aggregate stock of human capital in the population *and* individual access to education. As a result, health returns to absolute and

relative levels of education change over time. Although Chapter 4 made a first attempt to address these issues, much of the educational expansion that took place in the U.S. since WWII is not captured in available mortality data. Focusing on other societies, where the expansion of education was more fully observed, can shed light on the contextual factors under which educational differentials in mortality arise. In other words, the social, institutional, and technological capacity to control health and disease is a critical component of the increasing education-mortality gradient—one that is often neglected in the literature (the exception being improvements in medical technology, e.g., Chang and Lauderdale 2009; Frisbie et al. 2004).

Israeli society makes an especially interesting case study of rising educational disparities in health and mortality during a period of educational expansion. Like many Western economies, Israel exhibits very low mortality coupled with significant SES disparities (Manor et al. 1999; 2000) which appear to be widening (Jaffe et al. 2008). However, unlike most low mortality countries, Israel underwent its educational expansion rapidly and recently. In 1983, over 50 percent of Israel's adult population had completed ten or fewer years of education; yet within three decades Israel took the second lead in post-secondary education among all OECD countries (OECD 2011). Importantly, Israeli mortality data offer several advantageous features. Each census in Israel collects detailed information ("long form" format) from 20 percent of the population, including questions on educational attainment. Records in the National Death Registry are census-linked based on unique national identity numbers, avoiding most issues concerning linkage or misreporting that are found in comparable U.S. data sources—including those used in this

dissertation. For all those reasons, Israel case makes a natural case study to extend the work in this dissertation.

Another direction for future research is the intersection of race, gender, and class in shaping health and mortality outcomes. Although this dissertation focused on educational differentials in adult mortality, it also revealed stark race and gender differences in total lifespan inequality. The race and gender gaps in life expectancy are well known, but there are also significant disparities in lifespan variation. Specifically, S_{25} is greater by 1-1.5 years among blacks compared to whites of the same gender, and lifespan variation is also greater in men compared to women, irrespective of race. But the most interesting findings lie in the intersection of race, gender, and educational attainment. Race seems to matter less for longevity at lower levels of education—this is reflected in the convergence in life expectancy *and* lifespan variation between blacks and whites in each gender. However, there is still a significant race difference in returns to education at higher levels of education. The gender-education pattern appears to be exactly the opposite. Gender differences in life expectancy (but not in lifespan variation) are greater at lower levels of education than among the college educated. Additional research can illuminate how the intersection of race, gender, and class shapes multiple dimensions of lifespan inequality.

Finally, an important topic for future research is the consequences of lifespan inequality to individuals' exposure to death and uncertainty in their own length of life. Wilkinson (1996, 57) provides an intuitive illustration of socioeconomic differences in mortality rates: assuming that SES groups are completely stratified and mortality rates are

four times higher in the low SES group, an "average" low SES individual will have known four deaths among his or her close friends for every death known by an "average" high SES individual. In other words, to the extent that social networks are stratified by SES—a valid assumption according to past studies (e.g., Christakis and Fowler 2008)—low SES individuals are more exposed to mortality among their peers. Because they are also subject to higher and increasing lifespan variation, these deaths are more spread over the life course. By contrast, deaths among high SES individuals occur predominantly in in old age. Deaths among the latter may therefore be construed as part of the "natural aging process," whereas deaths among the former are untimely and disruptive. Indeed, the timing of spousal loss during the life course affects one's mental well-being (Sasson and Umberson 2014) and may similarly apply to other deaths among friends and family members. But far less is known about the consequences of uncertainty in one's own length of life.

Higher lifespan variation among low SES individuals translates into higher uncertainty in the length of life (Edwards 2013), which potentially affects their decision-making throughout the life course. Current evidence already suggests that individuals update their subjective assessment of survival probability following parental or spousal loss (Hurd an McGarry 2002), that survival expectations vary by SES (Delavande and Rohwedder 2011) and predict actual mortality (Perozek 2008), and that individuals base their economic decisions and retirement plans on those expectations (Hurd, Smith, and Zissimopoulos 2004; Solinge and Henkens 2009). Demographers have often assumed that individuals are aware of demographic realities and change their behavior accordingly

(Montgomery 2000). For example, in pre-demographic transition societies where child mortality is prevalent, high fertility is explained in part by parents' insurance and replacement strategies (Lloyd and Ivanov 1988). Yet far less is known about coping strategies that individuals adopt, in multiple social domains, in response to increasing uncertainty in adult mortality outcomes. This topic is ripe for future research.

In conclusion, this dissertation opens many more questions than it could possibly answer. Yet it also lays the factual and theoretical groundwork needed to pursue those questions. After all the evidence is weighed, the message is clear—the association between educational attainment and adult mortality in the United States (and elsewhere) is pervasive, enduring, and increasing in magnitude.

Appendix A: Life Table Age-at-death Distributions for Low and College-educated Americans by Race and Gender, 1990-2010

Figure A1: Age-at-death distributions for non-Hispanic white women by years of schooling, United States 1990-2010

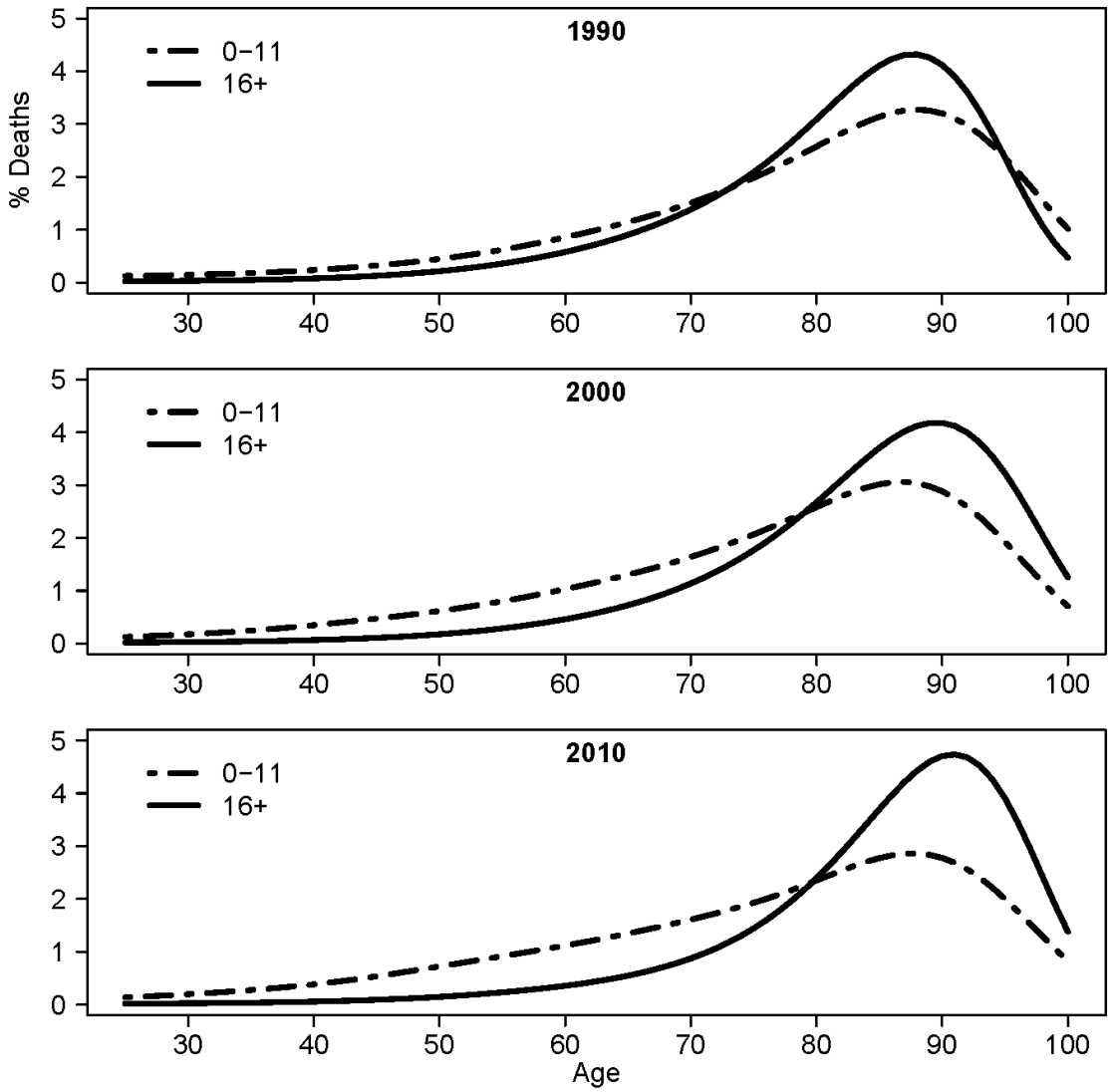


Figure A2: Age-at-death distributions for non-Hispanic black women by years of schooling, United States 1990-2010

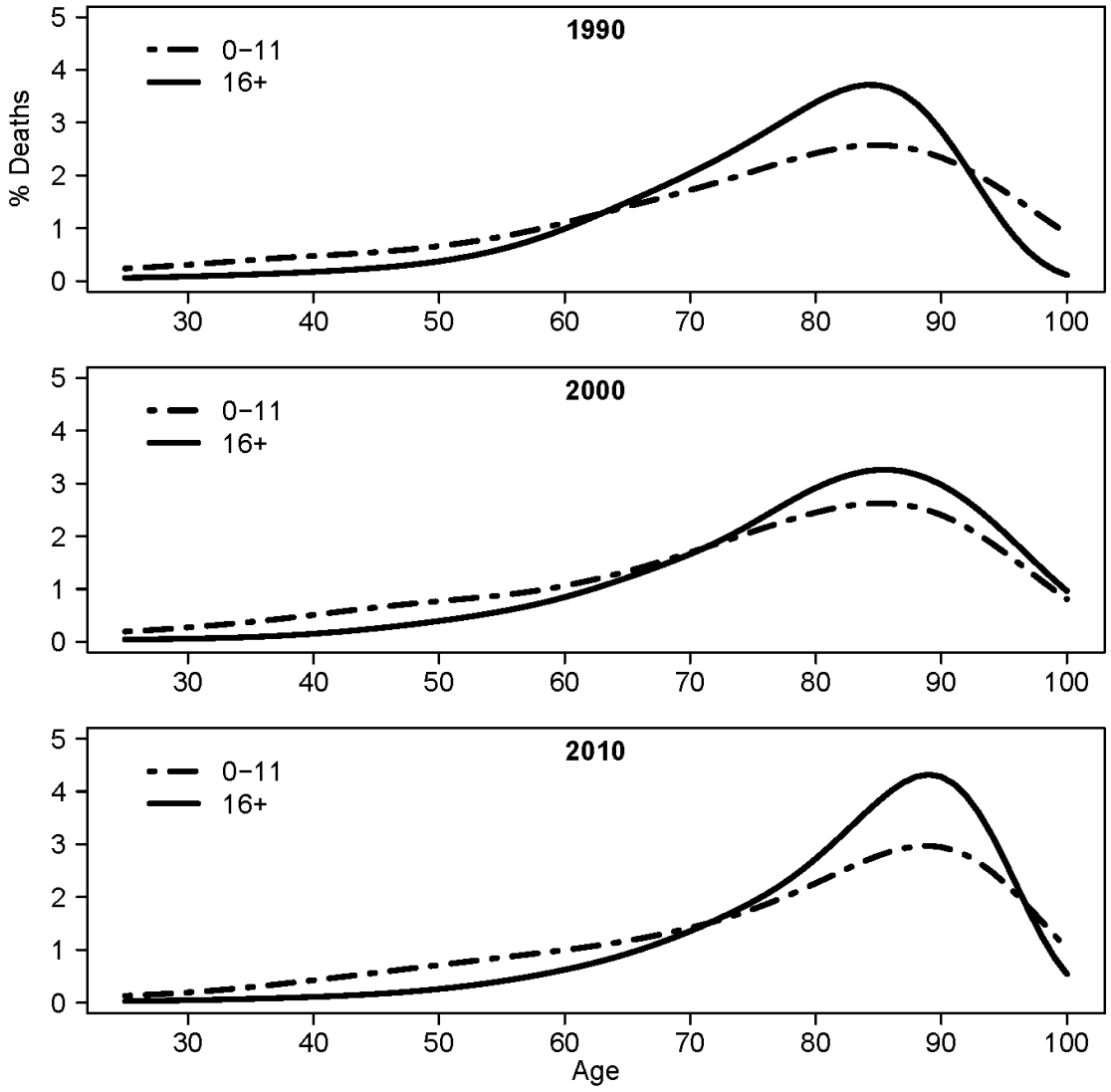


Figure A3: Age-at-death distributions for non-Hispanic white men by years of schooling, United States 1990-2010

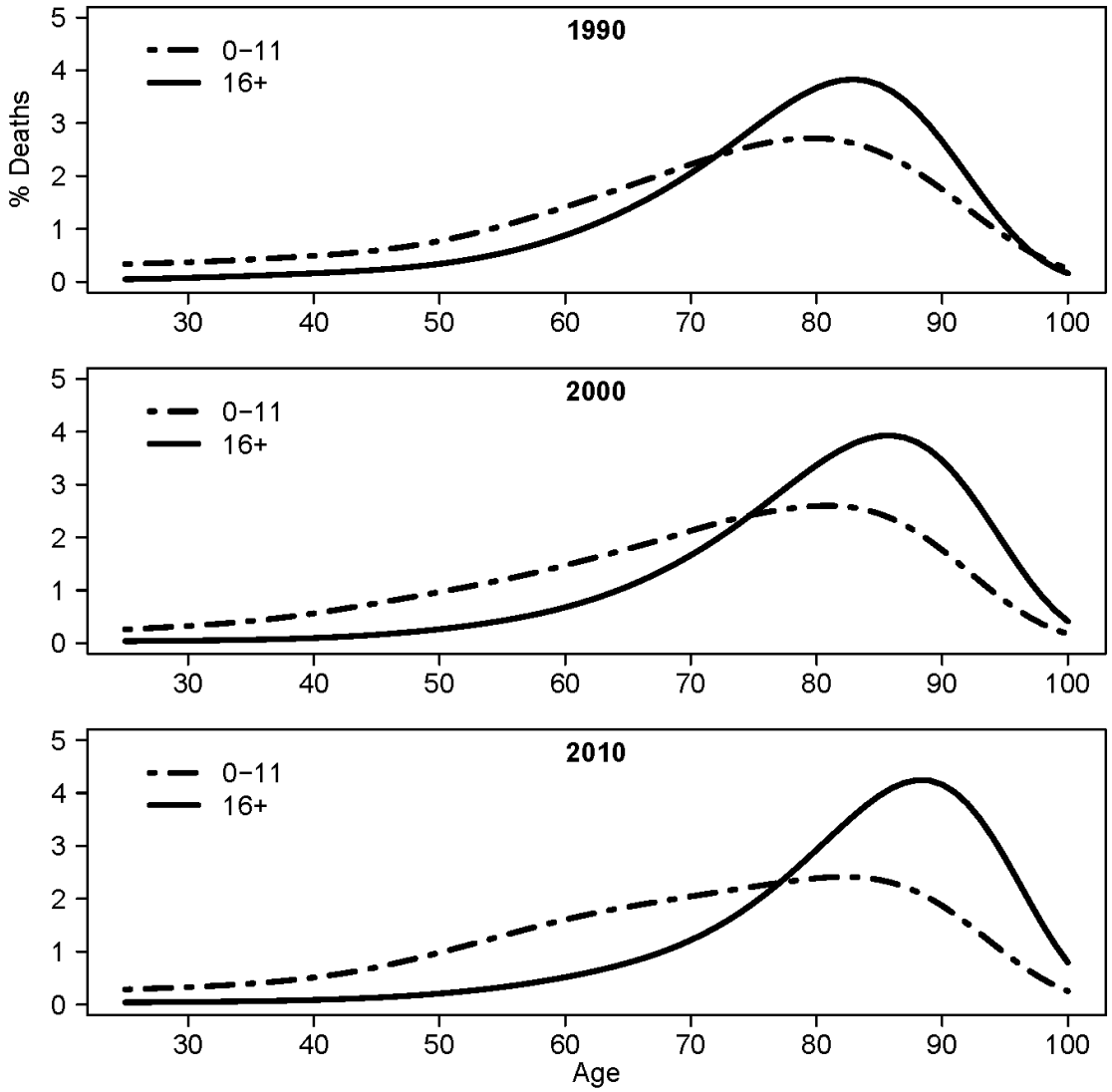
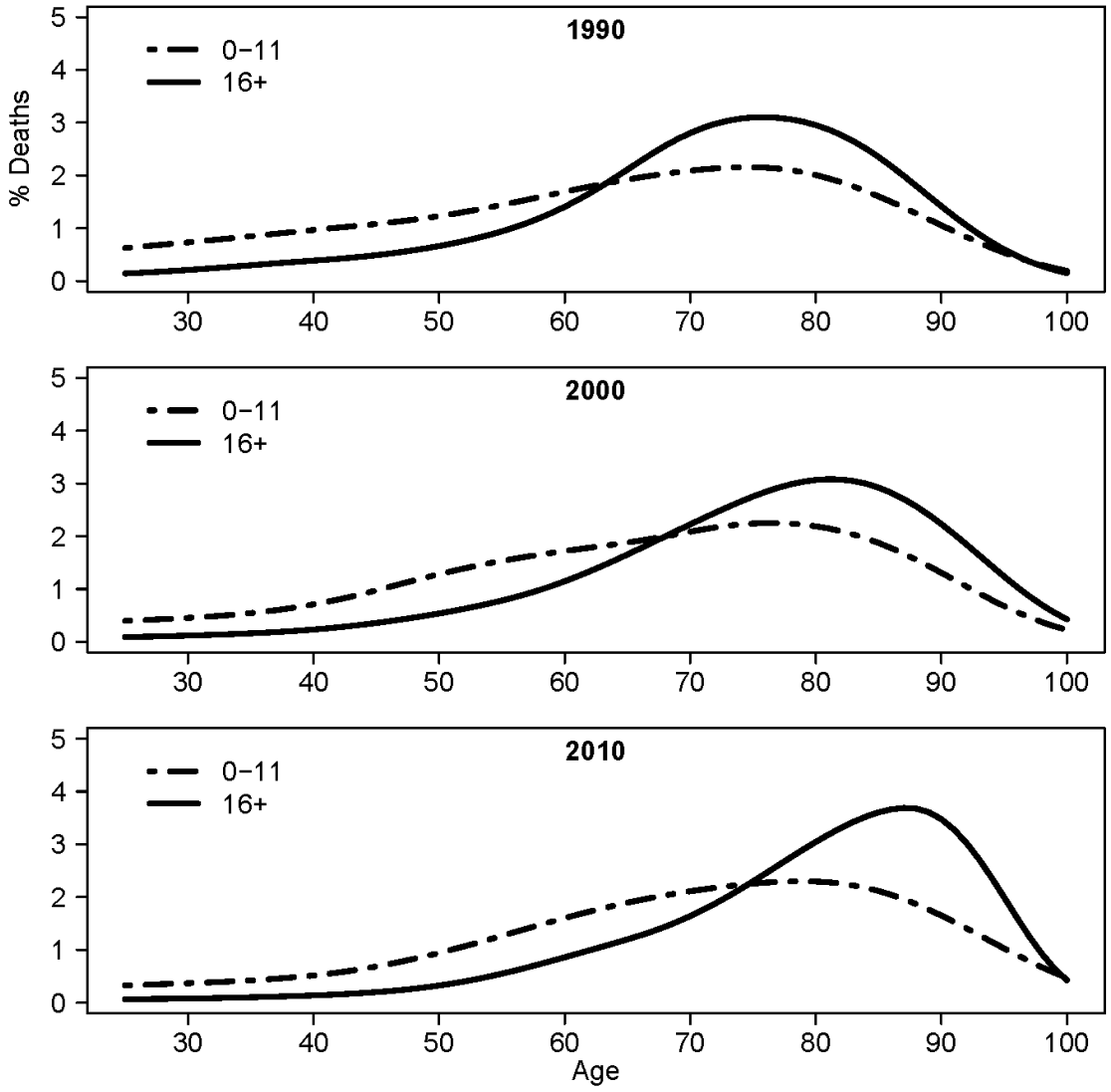


Figure A4: Age-at-death distributions for non-Hispanic black men by years of schooling, United States 1990-2010



Appendix B: Age and Cause of Death Decomposition Results for Non-Hispanic Blacks, Unites States 1990-2010

Table B1: Midyear population estimates and number of deaths by gender, education, and cause among non-Hispanic blacks

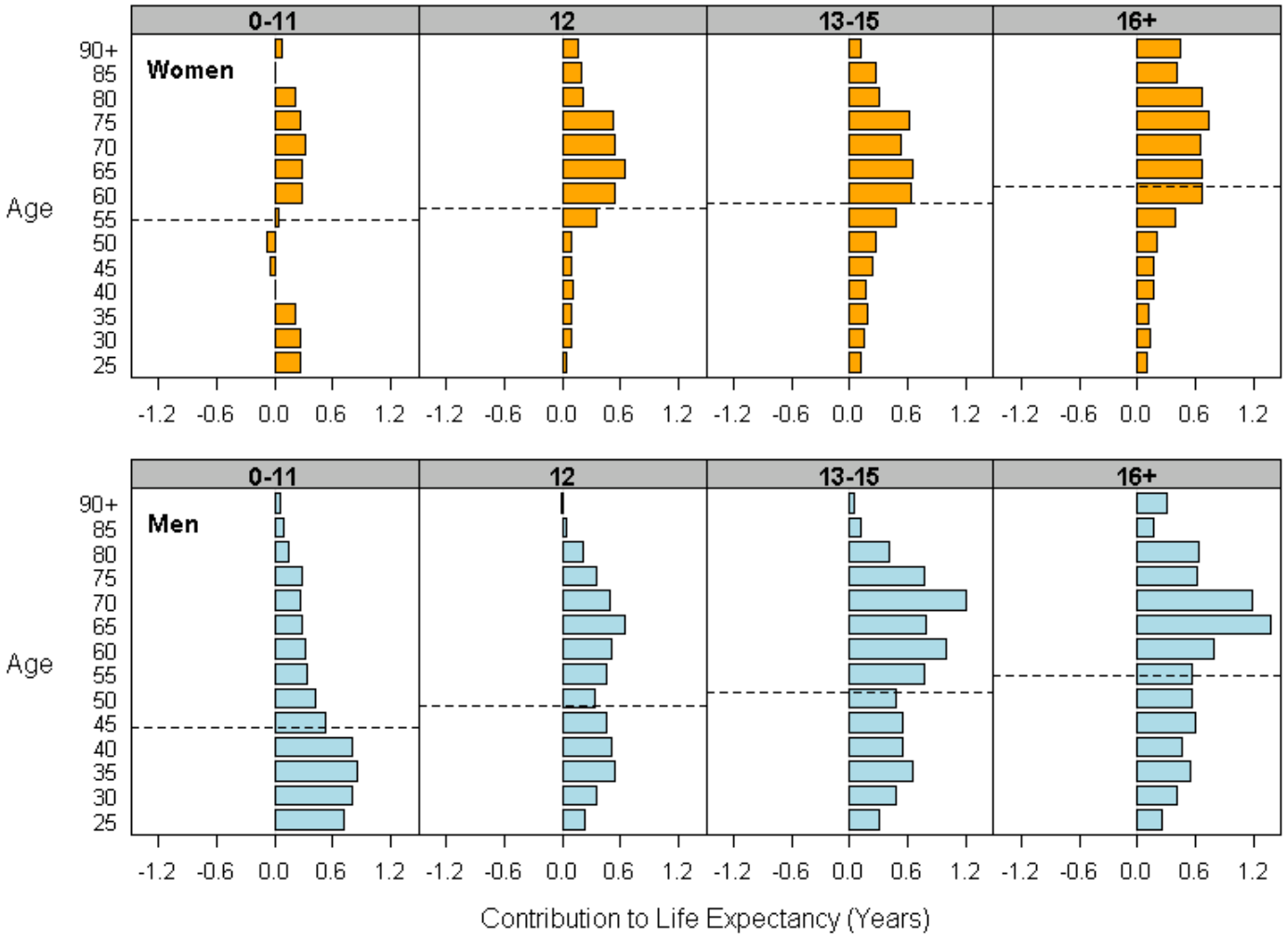
Gender	Year	Education (years)	Midyear population	Infectious/ parasitic	Neoplasms	Cardio-vascular	Respiratory	External	SRD**	Cerebro-vascular	Diabetes	Other	Total
Women	**1990**	**0-11**	2,687,518	1,878	9,628	24,323	2,814	1,707	3,278	5,824	2,876	8,287	**60,615**
		12	3,802,307	1,543	6,744	11,704	1,476	1,812	2,400	2,619	1,530	4,778	**34,606**
		13-15	1,503,842	416	1,966	2,554	370	589	555	626	327	1,180	**8,583**
		16+	1,067,580	256	1,579	2,213	293	334	378	578	266	950	**6,847**
	2000	**0-11**	2,195,391	2,586	8,190	21,489	2,591	1,388	3,974	5,467	3,372	9,906	**58,963**
		12	4,640,971	2,628	8,738	15,891	1,929	1,822	4,319	3,702	2,636	7,450	**49,115**
		13-15	2,442,581	735	3,196	4,362	564	728	1,258	1,098	711	2,219	**14,871**
		16+	1,676,881	390	2,411	3,120	415	382	814	821	477	1,630	**10,460**
	2010	**0-11**	1,822,174	1,891	5,971	12,916	1,672	1,008	3,473	3,083	2,098	10,581	**42,693**
		12	4,810,855	2,610	10,163	15,832	2,100	1,962	5,482	3,586	2,708	11,335	**55,778**
		13-15	3,595,970	968	5,143	5,895	778	1,094	2,144	1,340	1,099	4,304	**22,765**
		16+	2,532,915	476	3,250	3,419	472	520	1,083	895	511	2,859	**13,485**
Men	**1990**	**0-11**	2,248,211	2,974	11,145	23,329	3,426	5,324	9,412	4,723	1,650	8,717	**70,700**
		12	3,174,672	3,466	5,708	11,385	1,680	6,068	4,620	2,004	959	5,279	**41,169**
		13-15	1,097,340	1,236	1,328	2,680	360	1,572	950	452	227	1,281	**10,086**
		16+	808,280	834	1,110	1,843	272	697	620	337	166	822	**6,701**
	2000	**0-11**	1,974,633	3,115	8,822	17,762	2,483	3,735	8,170	3,977	1,982	7,693	**57,739**
		12	4,089,879	4,013	7,533	15,088	1,819	5,760	6,399	2,772	1,855	6,993	**52,232**
		13-15	1,840,865	1,057	2,081	3,914	430	1,671	1,431	691	507	1,806	**13,588**
		16+	1,197,480	659	1,716	2,818	306	718	888	500	370	1,156	**9,131**

Table B1 cont.

2010	**0-11**	1,799,402	1,970	6,580	11,964	1,685	2,916	5,887	2,323	1,682	7,453	**42,460**
	12	4,668,822	3,399	9,675	17,271	1,958	6,263	7,511	3,015	2,486	9,401	**60,979**
	13-15	2,617,390	1,074	3,487	5,679	575	2,063	2,109	937	899	3,034	**19,857**
	16+	1,691,059	544	2,311	3,363	377	785	1,009	584	512	1,816	**11,301**

Notes: Deaths counts are average of ten imputations; SRD = smoking-related diseases (bronchitis, emphysema, chronic airway obstruction and cancers of the lip, oral cavity, pharynx, esophagus, larynx, trachea, lung, and bronchus).

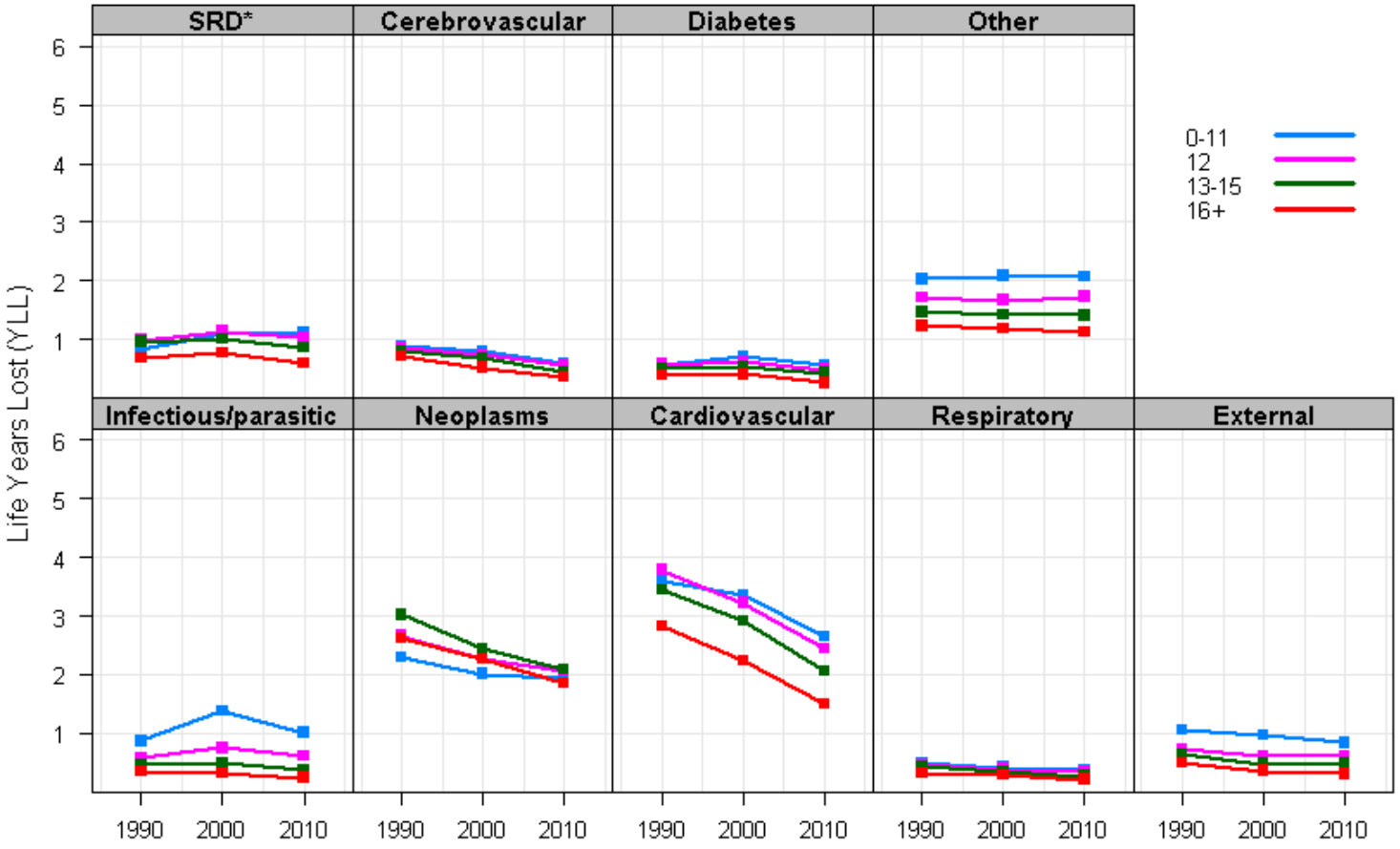
Figure B1: Age decomposition of change in life expectancy by gender and years of schooling, non-Hispanic blacks 1990-2010



Note: The dashed lines mark the approximate young-old threshold age in 1990.

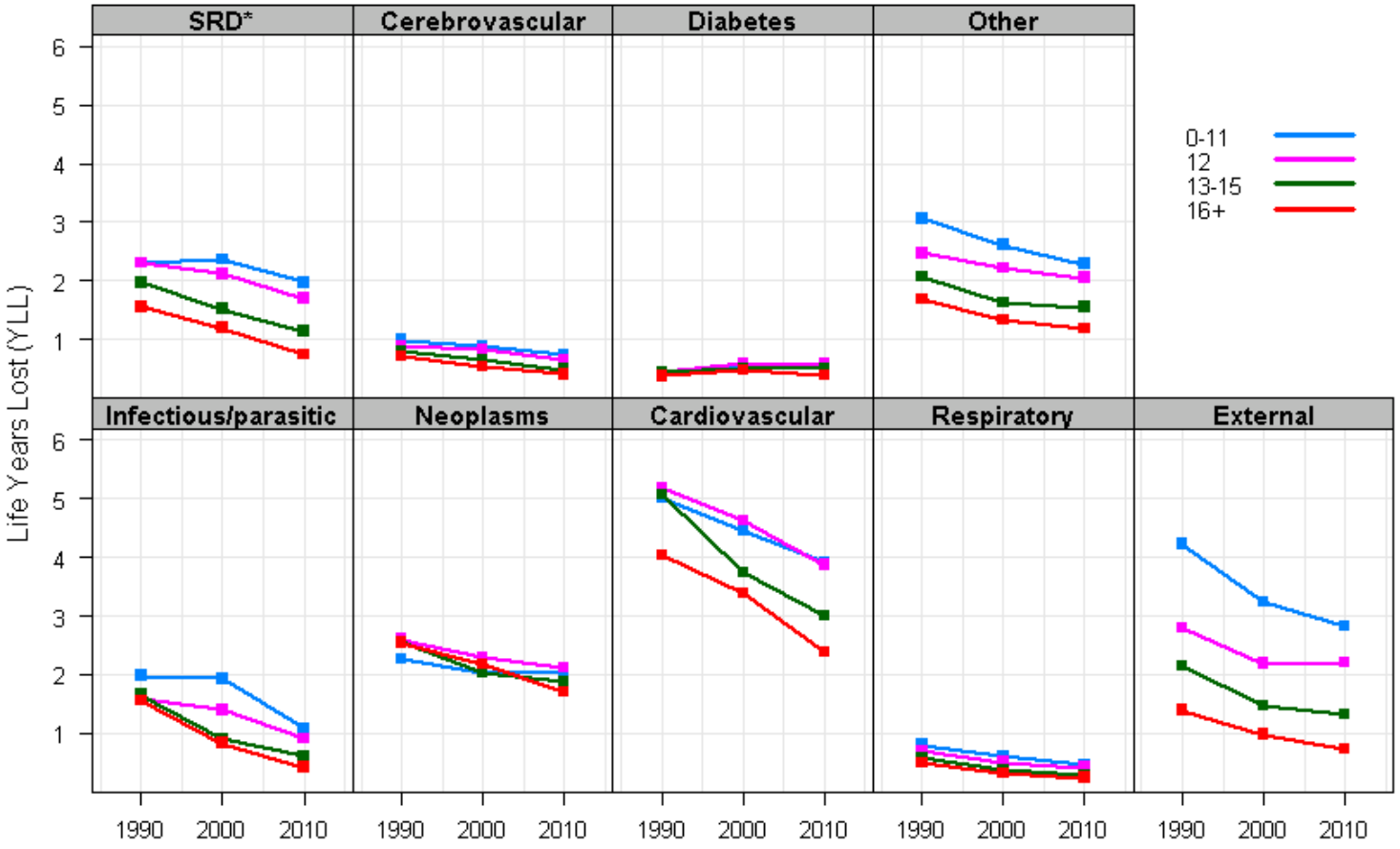
Figure B2: Total life years lost between ages 25 and 85 by gender and years of schooling, non-Hispanic blacks 1990-2010

Figure B3: Life years lost between ages 25 and 85 by cause of death and years of schooling, non-Hispanic black women



Note: SRD = smoking-related diseases (bronchitis, emphysema, chronic airway obstruction and cancers of the lip, oral cavity, pharynx, esophagus, larynx, trachea, lung, and bronchus).

Figure B4: Life years lost between ages 25 and 85 by cause of death and years of schooling, non-Hispanic black men



Note: SRD = smoking-related diseases (bronchitis, emphysema, chronic airway obstruction and cancers of the lip, oral cavity, pharynx, esophagus, larynx, trachea, lung, and bronchus).

Appendix C: ICD-codes Used for Cause of Death Grouping

Cause of death	ICD-9	ICD-10
Infectious and parasitic diseases	0-139	A00-B99
Neoplasms (excluding smoking related)	140-149, 151-160, 163-239	C16-C31, C35-D48
Cardiovascular diseases	390-429, 440-459	I00-I59, I70-I99
Cerebrovascular diseases	430-438	I60-I69
Respiratory diseases (excluding smoking-related diseases)	460-489, 493-495, 497-519	J00-J39, J45-J98
Smoking-related diseases (cancers of the lip, oral cavity, pharynx, esophagus, larynx, trachea, lung, and bronchus; bronchitis, emphysema, and chronic airway obstruction)**	150, 161-162, 490-492, 496	C00-C15, C32-C34, J40-J44
Diabetes mellitus External	250 E800-E999	E10-E14 V01-Y89
Other	All remaining codes	All remaining codes

Notes: Deaths in 1990 are classified under ICD-9 codes whereas deaths in 2000 and 2010 are classified under ICD-10; Smoking-related diseases include all causes where the smoking attributable fraction of deaths exceeds 65 percent in both genders combined (Center for Disease Control and Prevention 2008).

Appendix D: List of Variables Used in Lasso Regression Model to Predict Mortality

General health status	Ever told had ulcer
Self-rated health	Had an ulcer, past 12 months
Health status compared to 1 year ago	Had asthma attack/episode, past 12 months
Risk factors	Visited ER for asthma, past 12 months
Body mass index	Ever had bladder cancer
Ever told had hypertension	Ever had blood cancer
Ever told had hypertension on 2+ visits	Ever had bone cancer
Health behaviors	Ever had brain cancer
Cigarette smoking (current/former/never)	Ever had breast cancer
Duration of moderate activity	Ever had colon cancer
Duration of vigorous activity	Ever had esophagus cancer
Alcohol drinking status (former/current/abstainer)	Ever had gall bladder cancer
Days had 5+ drinks, past year	Ever had kidney cancer
Diagnosed conditions	Ever had more than 3 kinds of cancer
Ever told had angina pectoris	Ever had larynx cancer
Ever told had asthma	Ever had leukemia
Ever told had cancer	Ever had liver cancer
Ever told had coronary heart disease	Ever had lung cancer
Had chronic bronchitis, past 12 months	
Ever told had emphysema	
Had pain in jaw/front of ear, past 3 months	Ever had lymphoma
Had hay fever, past 12 months	Ever had mouth/tongue/lip cancer
Ever told had heart attack	Ever had other cancer
Ever told had heart condition/disease	Ever had pancreatic cancer
Ever told had diabetes	Ever had rectal cancer
Told had failing kidneys, past 12 months	Ever had melanoma
Had low back pain, past 3 months	Ever had skin cancer (non-melanoma)
Told had liver condition, past 12 months	Ever had skin cancer (unspecified kind)
Had severe headaches/migraine, past 3 months	Ever had soft tissue cancer
Had neck pain, past 3 months	Ever had stomach cancer
Told had sinusitis, past 12 months	Ever had pharynx cancer
Ever told had a stroke	Ever had thyroid cancer

Appendix D cont.

Functional limitations	Needs help with instrumental activities of daily living (IADL)
Need help with bath/shower	Difficulty carrying 10 lb. w/o special equipment
Need help in/out of bed or chairs	Difficulty walking up 10 steps w/o special equipment
Need help dressing	Difficulty grasping objects w/o special equipment
Need help eating	Difficulty reaching over head w/o special equipment
Need help using the toilet	Difficulty sitting 2 hours w/o special equipment
Need help getting around in home	Difficulty standing 2 hours w/o special equipment
Difficulty walking without equipment	Difficulty stoop/bend/kneel w/o special equipment
Needs help with activities of daily living (ADL)	Difficulty walking 1/4 mile w/o special equipment

Notes: Non-zero parameter estimates are highlighted in gray; the number of parameters exceeds the number of items on the list because categorical variables with more than two categories were dummy coded. Age and gender (not shown in table) were also included in the model.

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