Explaining the Growing Education Gap in U.S. Adult Life Expectancy, 1990-2010

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Abstract

As life expectancy at birth in the U.S. approaches eighty years of age, educational differentials in adult mortality are greater than ever. Low-educated Americans have shorter life expectancies than their college-educated counterparts and have recently suffered absolute declines in longevity. Using vital statistics data, this study decomposes those trends by age and cause-of-death for major educational attainment groups in the U.S. from 1990 to 2010. The findings reveal an education gradient in life years lost from all major causes of death. Among low-educated whites, life expectancy declined predominantly due to rise in external and smoking-related deaths. Mortality also increased among high-school educated whites under age 55, offsetting mortality reductions in old age. Evidently, large segments of the U.S. population are diverging from the classic health transition model and instead are undergoing a series of divergence and convergence sequences resulting from changes in social conditions, health technologies, and emerging mortality risks.

INTRODUCTION

Since the 1960s, the U.S. adult mortality regime has been undergoing two fundamental changes. First, the population as a whole transitioned into the fourth stage of the epidemiological transition (Olshansky and Ault 1986), whereby chronic diseases not only substitute for infectious diseases as the leading causes of death, but are also delayed well into old age. As a result, life expectancy at birth in the U.S. is now estimated at 78.7 years (Kochanek, Arias, and Anderson 2013) for both genders combined and projected to increase well into the future (Bell and Miller 2005). Second, across the same period, scholars have documented widening socioeconomic (specifically, educational) disparities in U.S. adult mortality (Meara, Richards, and Cutler 2008; Montez et al. 2011; Olshansky et al. 2012; 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 (Sasson 2014).

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 across socioeconomic strata. Specifically, individuals with high socioeconomic status (SES) 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 between world regions (Casseli, Meslé, and Vallin 2002; McMichael et al. 2004; Vallin and Meslé 2004). However, recent findings suggest that the same divergence in mortality is occurring at the subnational level in the U.S., where low SES Americans are subject to rising mortality (Olshansky et al. 2012; Sasson 2014). 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 men and women. In other words, individuals having fewer than 12 years of schooling, who currently make up over 45 percent of white Americans aged 25 and over (Ruggles el al. 2010), have been excluded from any significant improvement in average longevity for at least a decade. Although U.S. adult life expectancy as a whole is still on the rise—owing to continued gains among blacks of all educational levels, Hispanics, and college-educated whites—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.

Using data from the vital registry from 1990 to 2010, this study aims at understanding why life expectancy has shown little progress or even declined among low and high-school educated white Americans, whereas significant gains in average longevity have been observed among highly educated groups. I focus on white Americans because blacks of all education levels have seen continued improvements in

life expectancy over the study period in spite, and perhaps because, of their ongoing disadvantage compared to whites (nevertheless, results for non-Hispanic blacks are shown in Appendix A). In keeping with the main objective, I use two decomposition methods to describe within- and between-group change in cause-specific mortality. First, in each educational attainment category, I explore which age groups have been most vulnerable to (have benefited the most from) increasing (decreasing) mortality rates, and how these patterns have contributed to change in adult life expectancy and age-at-death variability. Second, I describe the number of life years lost to each cause of death (i.e., disease burden) in each education group and how that burden 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. Finally, I discuss the theoretical and practical implications for the future of U.S. mortality.

EDUCATIONAL DIFFERENCE IN CAUSE-SPECIFIC MORTALITY

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 Hummer and Hernandez 2013; Hummer and Lariscy 2011). During the 1990s, much of the growing education 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). Using data from the National Health Interview Surveys Linked-Mortality Files (NHIS-LMF), Montez and colleagues (2011) further

showed that all-cause mortality rates increased among white women with fewer than 12 years of schooling between 1986 and 2006. A later study revealed that this trend was largely driven by rising mortality from 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 men's cause-specific mortality. Significant educational disparities were also found across major causes of premature death (cancer, heart disease, stroke, diabetes, and accidents), using vital statistics data, across 26 states and in both genders (Ma et al. 2012).

Focusing on *relative* differences, however, 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 greater among low-educated women (aged 45-84) than their college-educated counterparts, whereas the same ratio in cardiovascular diseases amounts to 5.51 (Montez and Zajacova 2013a). Yet deaths from cardiovascular diseases are much more common than those of influenza and pneumonia. Building on previous studies, an important next step is therefore to translate relative disparities into absolute number of life years lost. In other words, the cause of

death exhibiting the greatest relative risk between education groups does not necessarily account for the greatest disparity in life expectancy.

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, highly-educated Americans exhibit greater compression of mortality than their less educated counterparts (Brown et al. 2012). Sasson (2014) further revealed that S_{25}, the standard deviation of age at death over 25, 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 (Zhang and Vaupel 2009) marks the cutoff where *increasing* mortality below it and *decreasing* mortality above it both contribute to increasing age-at-death variability. As far as period mortality is concerned, increasing variation in age at death may indicate that mortality is increasing among recent birth cohorts whereas older cohorts continue to see gains in longevity (or, at the very least, mortality reductions among the latter are greater).

Using a Gompertz mortality model, Gillespie and colleagues (2014) approximate the young-old threshold age in modern populations at roughly one standard deviation below the life expectancy. They further 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, could also be on the rise. 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 increasing 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 various causes of death. 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 have (practically) full coverage of deaths in the U.S. 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.

The main objective of this study is therefore to uncover trends and patterns in underlying mortality rates across educational attainment groups, in order to explain the growing education gap in U.S. adult mortality.

METHODOLOGY

Data

Two data sources were utilized in estimating 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 was limited to non-Hispanic white men and women (results for non-Hispanic blacks are shown in Appendix A) 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 among the oldest old (Alpérovitch et al. 2009; Hill, Preston, and Rosenwaike 2000; Tinetti et al. 2012). Nonetheless, 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. These steps, including a uniquely developed method for missing data imputation, are detailed in Appendix B.

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 based on 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 deaths in the 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 nine categories include: infectious and parasitic diseases, neoplasms (excluding those predominantly attributed to smoking), cardiovascular diseases (CVD), cerebrovascular diseases, smoking-related diseases (abbreviated SRD), respiratory diseases (excluding chronic lower respiratory diseases), diabetes mellitus, external causes, and a residual category for all other or unspecified causes. Table 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)

$$ {}_n\Delta_x = \frac{l_x^1}{l_{25}^1}\left(\frac{{}_nL_x^2}{l_x^2} - \frac{{}_nL_x^1}{l_x^1}\right) + \frac{T_{x+n}^2}{l_{25}^1}\left(\frac{l_x^1}{l_x^2} - \frac{l_{x+n}^1}{l_{x+n}^2}\right) \quad (1) $$

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). Naturally, only the direct effect applies to the open interval

$$ {}_\infty\Delta_x = \frac{l_x^1}{l_{25}^1}\left(\frac{T_x^2}{l_x^2} - \frac{T_x^1}{l_x^1}\right) \quad (2) $$

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

$$\Delta e_{25}^{o} = e_{25}^{o}(2) - e_{25}^{o}(1) = \sum_{25}^{\infty} {}_{n}\Delta_{x} \quad (3)$$

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 certain 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:

$$YLL_i = e_x^{-i*} - e_x \quad (4)$$

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. This measure, however,

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

$$YLL = 60 - {}_{60}e_{25} \tag{5}$$

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 ${}_n\lambda_x$, is therefore

$${}_n\lambda_x = n - {}_nL_x \tag{6}$$

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

$${}_n\lambda_x^i = n \cdot {}_xd_{25}^i + (n \cdot l_x - {}_nL_x)\,{}_nR_x^i \tag{7}$$

where ${}_n\lambda_x^i$ 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*

$$YLL = \sum_j \sum_i {}_n\gimel_x^i \quad (8)$$

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 had the greatest toll on life years lost, how that toll had 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 in 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 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 horizontal bars represent the contribution (in years) of each 5-year age group to the total change in e_{25}^{o} over the study period. Recall that this contribution consists of life years gained (or lost) within the age interval and in all subsequent age intervals due to change in age-specific mortality rates. 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 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 e_{25}^{o} was due to rising mortality between ages 45 and 64. This suggests that middle-aged women in this education category were especially 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 opposite trends among 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 (Sasson 2014). Both components—declining mortality above the threshold and increasing mortality below it—worked to increase variation in age at death.

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 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^o_{25} also increased by 1.8 years (Sasson 2014). 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 increases in e^o_{25} 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 the life years lost, 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 that 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, ${}_{60}e_{25}$ 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 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} (see Olshansky et al. 2012; Sasson 2014), 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 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 roughly one year each over 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 adult life expectancy, temporary or total, shown earlier.

Trends among men were generally similar (but not identical) to those among women of the same educational level. Figure 4 shows that low educated men gained over one year of life due to reductions in CVD, but experienced greater combined losses from smoking-related, 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 and 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 experienced 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 declining 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 in spite of 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 (Olshansky et al. 2012; Sasson 2014). This trend was fueled by two disparate effects: absolute declines in e_{25}^{o} among the low educated and dramatic improvements among the college educated. Furthermore, high-school educated whites have seen only modest improvements in life expectancy over the two decades. Using vital statistics data, this study set out to understand how changing age- and cause-specific mortality rates have contributed to gains and losses in average longevity across educational attainment 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 white 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 declined 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 white 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 are no longer sharing these improvements in longevity and, more recently, some have been regressing. The findings in this study suggest that low-educated white 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 have taken a significant toll on the temporary life expectancy between ages 25 to 85. Deaths from diabetes, strokes, and infectious and respiratory diseases were much less prominent, but nonetheless 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 into 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.

More troubling is the fact that between 1990 and 2010 the mortality regime has become even less favorable for low and high-school educated white 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 rates 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 (Sasson 2014).

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 Surveys (e.g., Montez et al. 2011; Montez and Zajacova 2013a), but offer several important extensions. Whereas 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, the present 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 white 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 *within*-group inequality in length of life among low and high-school educated populations.

Clearly, cigarette smoking will continue to have a tremendous impact on U.S. adult mortality in the coming decades. 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 the above findings, the growing educational gradient in adult life expectancy. 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. These results mirror educational disparities in smoking behavior, which have not only persisted but also widened for whites (and blacks) since the mid-1970s (Pampel 2009).

More surprising is the rise in mortality from external causes among all but the college educated. These results, however, are consistent with those reported by Miech and colleagues (2011), showing that accidental poisoning (primarily drug overdose) was the fastest increasing cause of death among white, middle-aged men and women between 1999 and 2007, and also the cause in which the educational disparity grew most.

Limitations

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 these factors present a potential source of bias when estimating mortality trends. Several steps were taken to minimize both random and systematic sources of error (see Appendix B), 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 (e.g., Ma et al. 2012; Meara et al. 2008; Montez et al. 2011; Montez and Zajacova 2013a).

Conclusion

Numerous studies have focused on the rising education-mortality gradient, but few addressed the absolute increase in mortality among low-educated Americans. A complete explanation of this trend not only requires that high-SES individuals possess *greater* access to healthy lifestyles and environments (Link and Phelan 1995; 2002), 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). As a result, poverty increased and home ownership declined among low-educated Americans—both of which are associated with higher risk of mortality (Montez and Zajacova 2013b; Rogers, Hummer, and Everett 2013; Ross and Wu 1995).

Low-educated Americans not only face economic hardship, but are also less likely to be married than their college-educated counterparts, which places 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 the 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 contributes to health inequality because spousal education improves one's health over and above their own level of education (Brown, Hummer, and Hayward 2014).

Taken together, it is clear that the *life chances* of low-educated Americans, in the Weberian sense, have changed for the worse in recent decades—and in ways associated with greater risk of mortality. 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. Early and premature deaths from smoking and external causes are already on the rise among high-school educated men and women

under the age of 60. In other words, we are witnessing for the first time a reversal in the epidemiological (health) transition among a significant portion of the U.S. population—and not merely the "negatively select" few at the bottom of the education distribution.

The reversal of mortality decline in Eastern Europe after the fall of the Soviet Union and in sub-Saharan Africa following the AIDS epidemic has led scholars to reframe the epidemiological transition as a series of divergence and convergence sequences resulting from changes in social conditions, health technologies, and emerging mortality risks (Vallin and Mislé 2004). Taken to the subnational level, the same logic can be applied to low SES groups in the U.S., for whom mortality rates are increasing across multiple causes of death.

It is difficult to determine whether these patterns reflect period or cohort trends because these results are based on period life tables, which conjoin multiple birth cohorts into a single synthetic cohort. Since the 1960s, however, changes in mortality from heart disease and lung cancer in the U.S. have largely been 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, although the case of external and other causes of death is not immediately clear. The rise in young-adult mortality may therefore be a precursor to what lies ahead for high-school educated white Americans—and perhaps for other ethnic minorities[5]—as these cohorts enter old age. Unless social conditions improve for low-SES Americans, there is no reason to believe that they will catch up with those in the lead.

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 A), they remain considerably disadvantaged compared to whites of the same educational level.

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Table 1: Midyear population estimates and number of deaths by gender, education, and cause among non-Hispanic whites

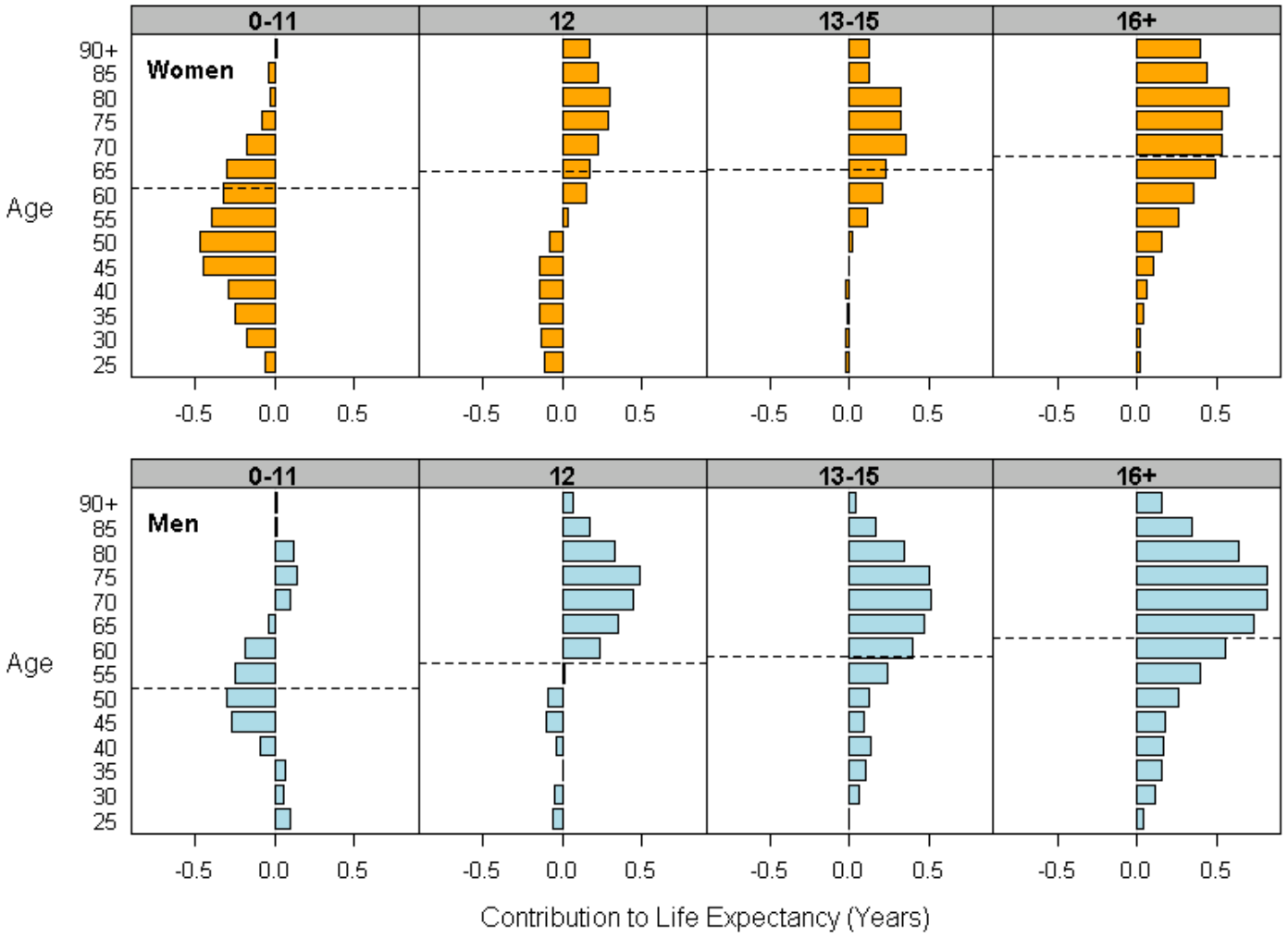
Gender	Year	Education (years)	Midyear population	Infectious/ parasitic	Neoplasms	Cardio-vascular	Respiratory	External	SRD**	Cerebro-vascular	Diabetes	Other	Total
Women	**1990**	**0-11**	12,070,975	4,782	50,393	145,973	21,718	7,958	26,499	31,846	9,133	40,456	**338,758**
		12	29,660,127	4,809	68,766	126,089	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 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 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 2: Total life years lost between ages 25 and 85 by gender and years of schooling, non-Hispanic whites 1990-2010

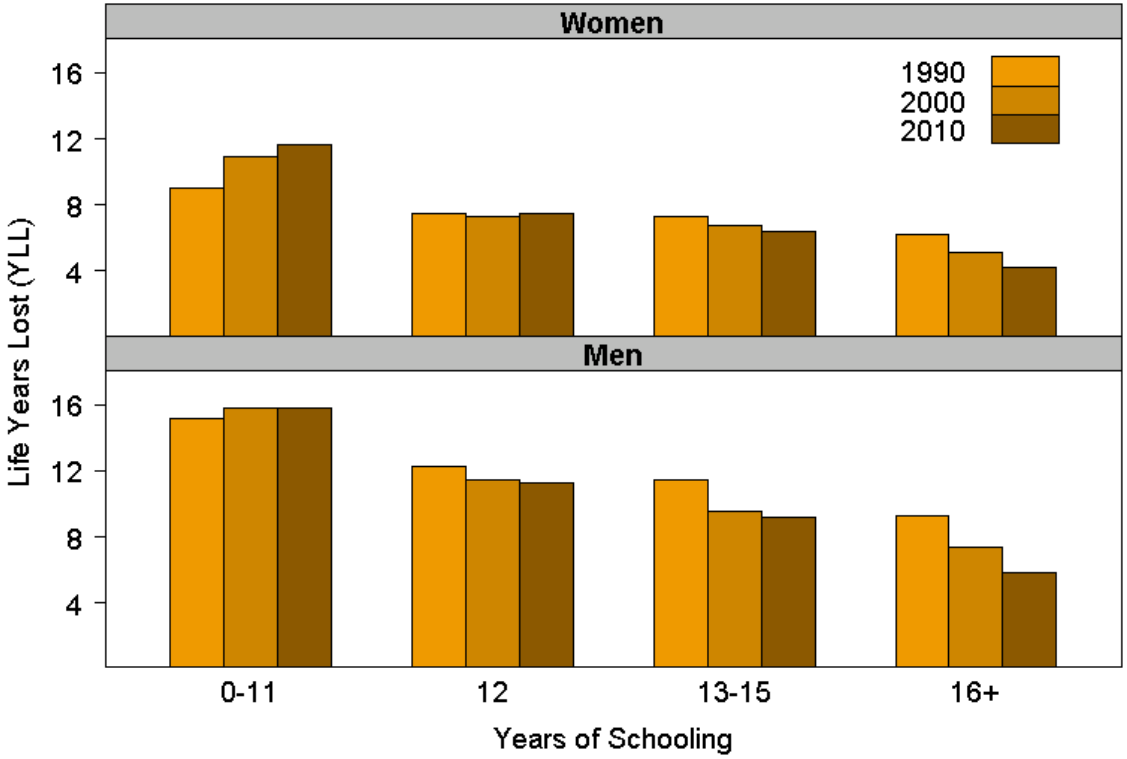
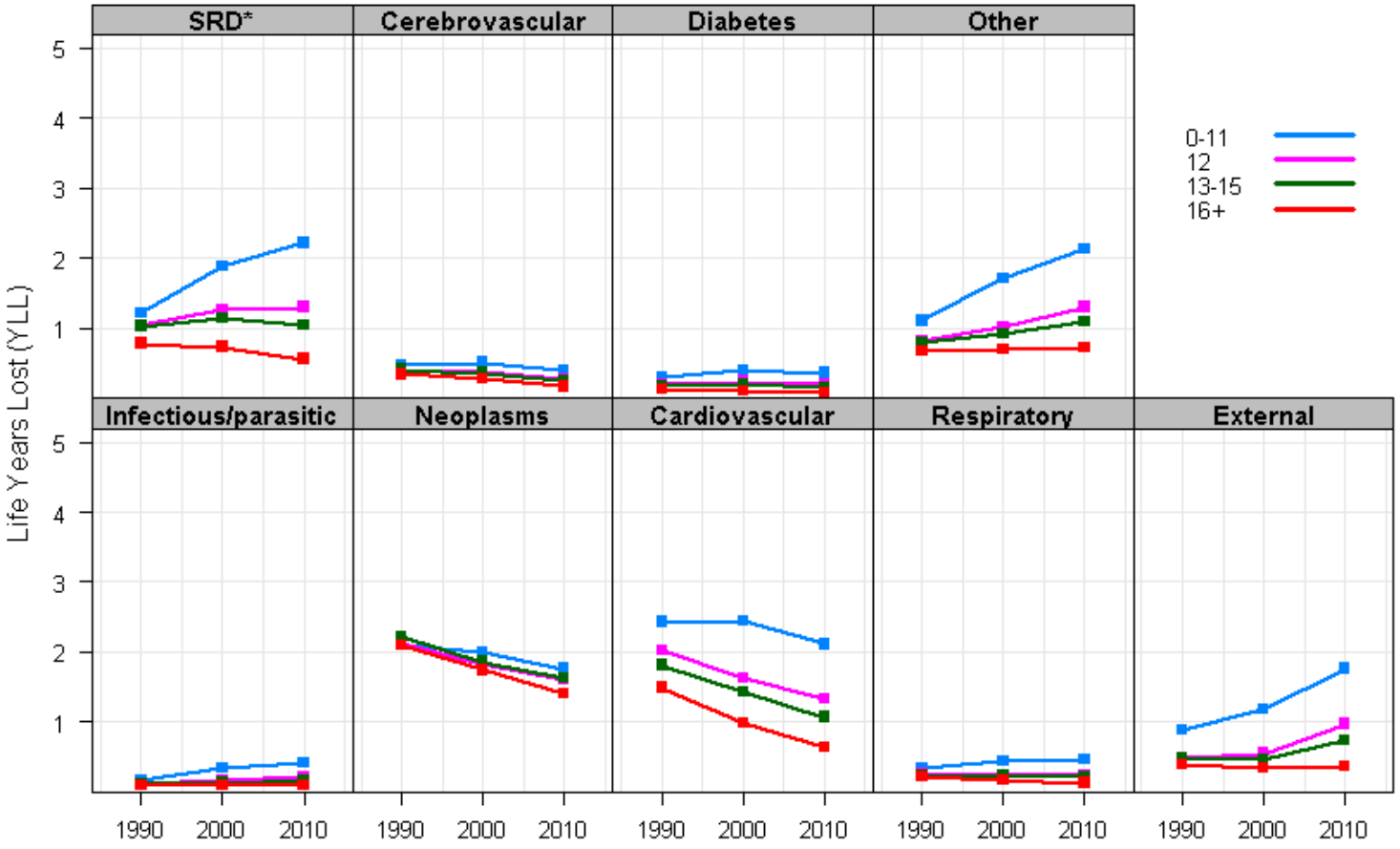
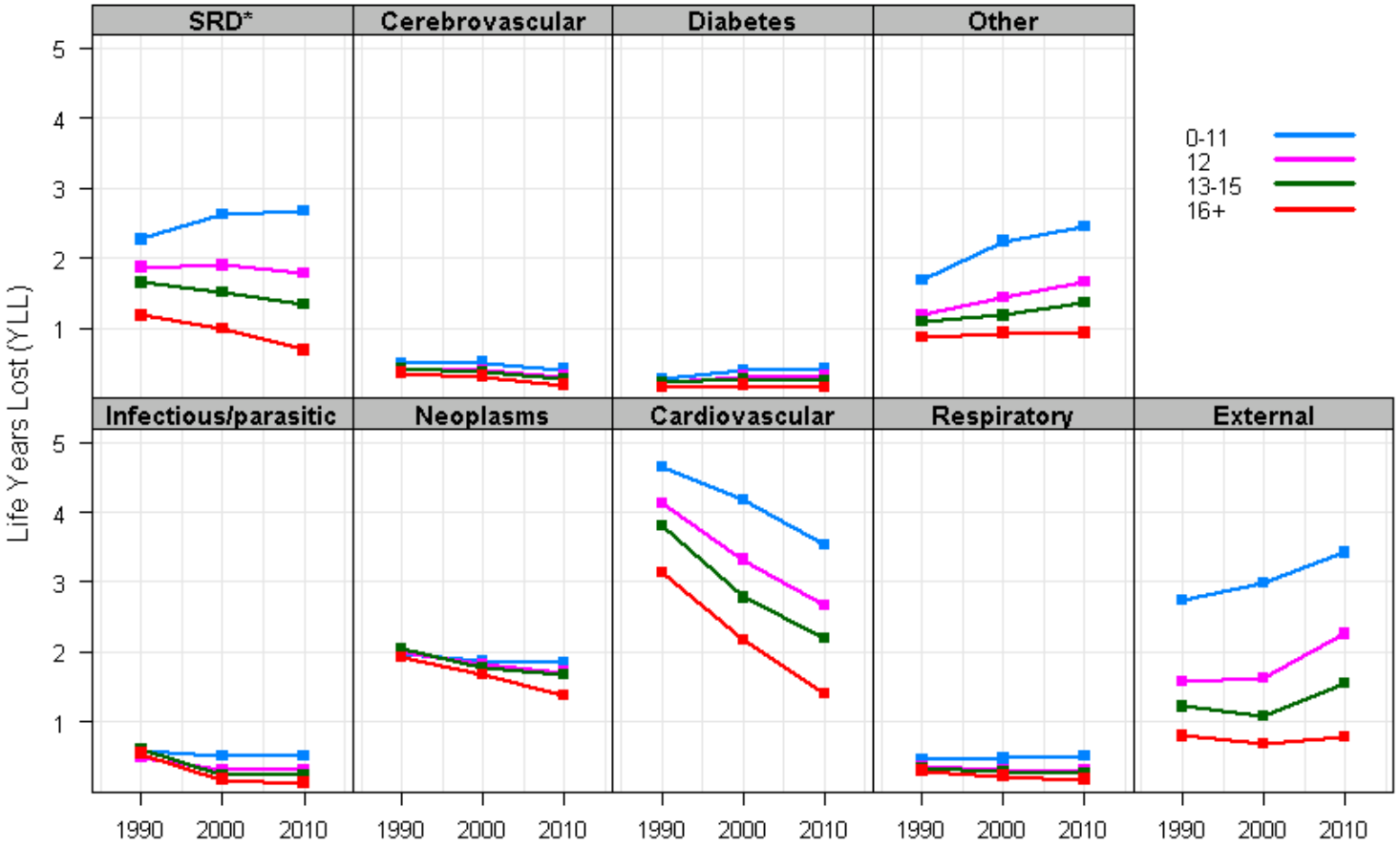


Figure 3: Life years lost between ages 25 and 85 by cause of death and years of schooling, non-Hispanic white women



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

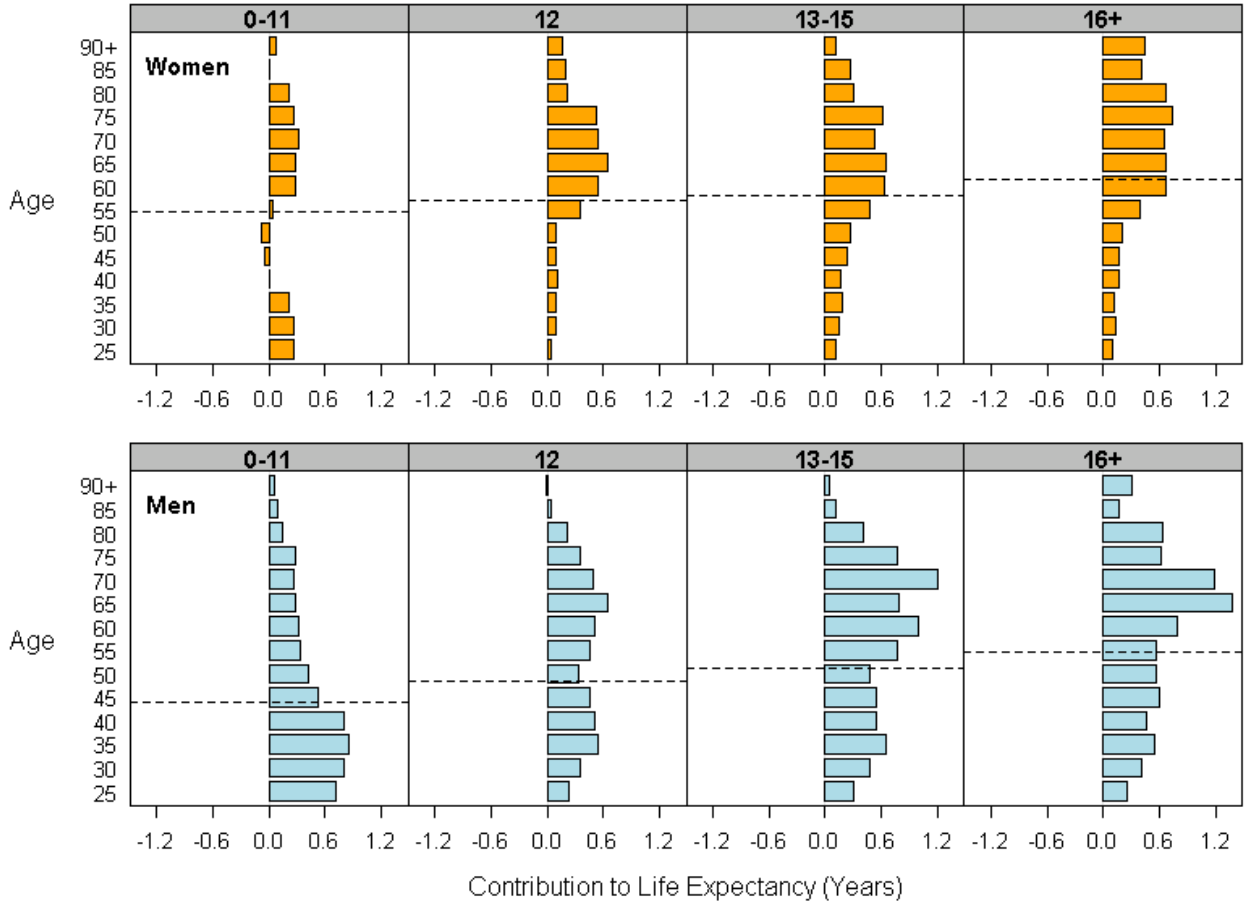
Figure 4: Life years lost between ages 25 and 85 by cause of death and years of schooling, non-Hispanic white men



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

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

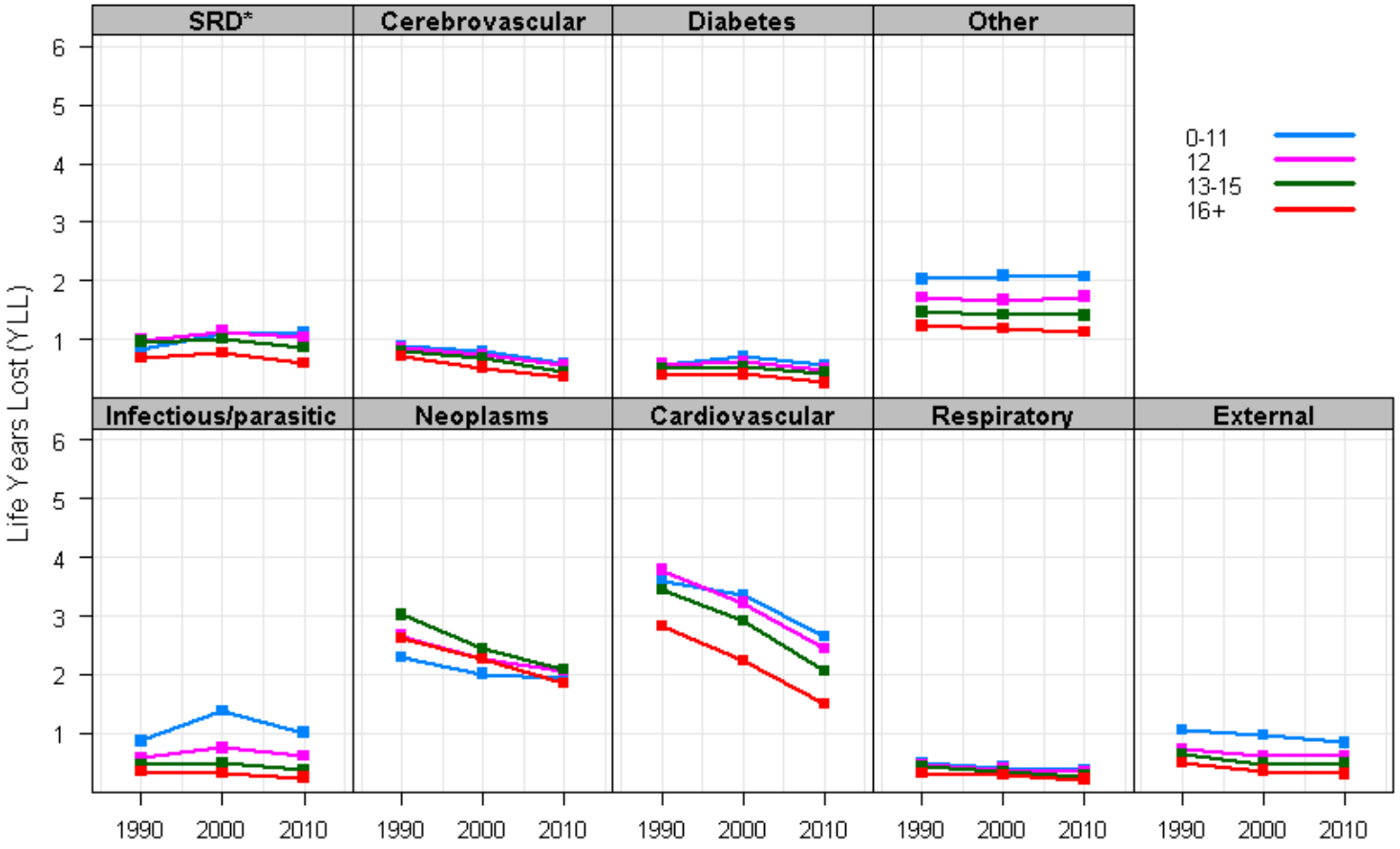
Figure A1: Age decomposition of change in life expectancy by gender and years of schooling, non-Hispanic blacks 1990-2010



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

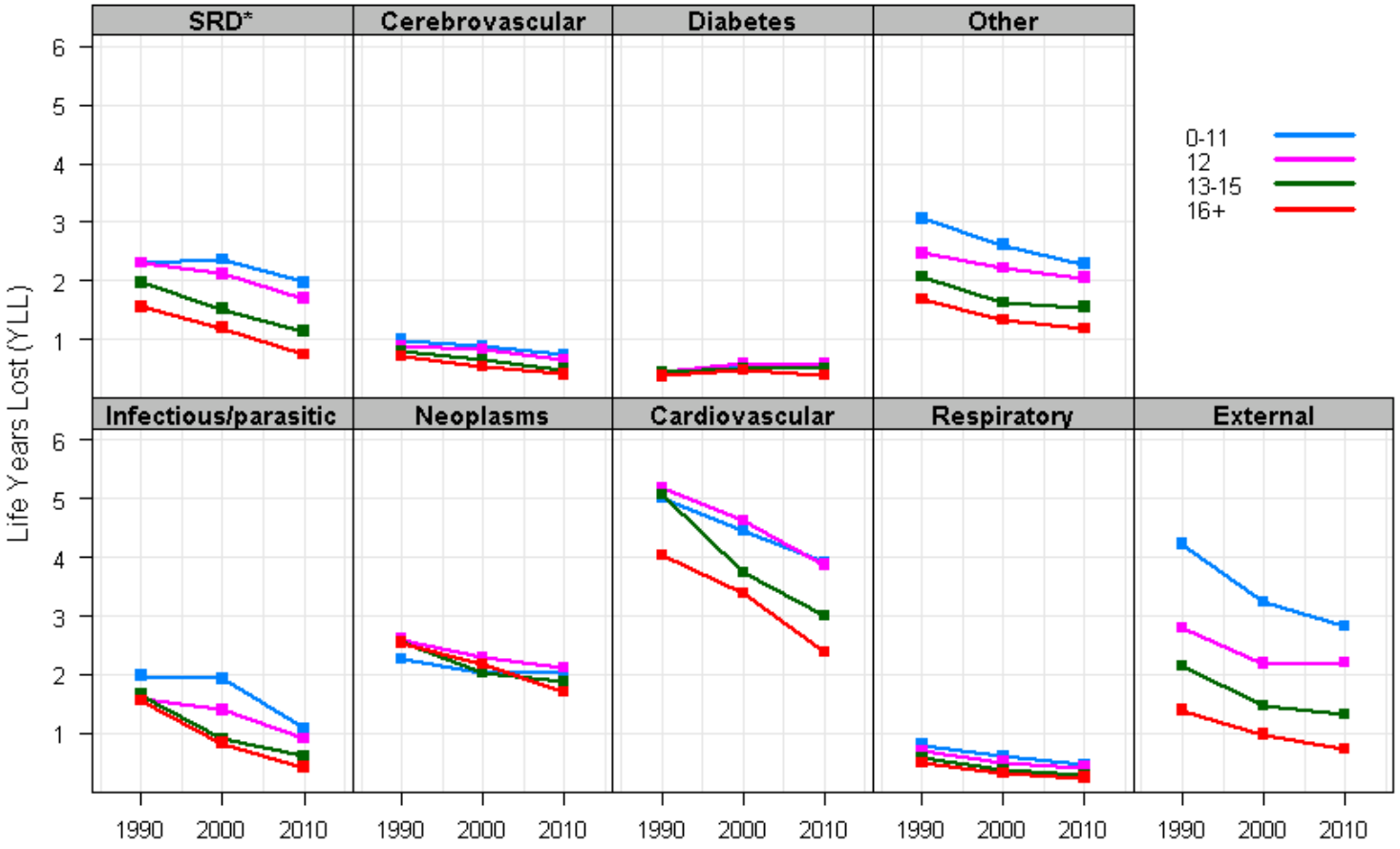
Figure A2: Total life years lost between ages 25 and 85 by gender and years of schooling, non-Hispanic blacks 1990-2010

Figure A3: 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 A4: 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 B: Procedure for Data Imputation and Recode

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).[1] 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

[1] 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.

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.[2] 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:

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

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

[2] 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.

conditional on educational attainment; (b) the distribution of education in the at-risk population; and (c) the distribution of overall mortality. Although Equation B.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.[3] 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.[4] 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 methods6 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.

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[3] 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

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

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.

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

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).