## Education Gradients in Mortality Trends by Gender and Race

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## Abstract

We examine gender and race differences in education-mortality trends among 25-64 year olds in the United States from 2001-2018. The data indicate that the relationships are heterogeneous with larger mortality reductions for less educated non-Hispanic blacks than other races and mixed results at higher levels of schooling. We also investigate the causes of death associated with changes in overall mortality rates and identify key differences across race groups and education quartiles. Drug overdoses represent the single most important contributor to increased death rates for all groups, but the sizes of these effects vary sharply. Cardiovascular disease, cancer, and HIV are the most significant sources of mortality rate reductions, with the patterns again heterogeneous across sex, race, and educational attainment. These results suggest the limitations of focusing on all-cause mortality rates when attempting to determine the sources of positive and negative health shocks affecting population subgroups. Examining specific causes of death can provide a more nuanced understanding of these trends.

JEL codes: I10; I12; I14; I24; J10

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## 1. Introduction

A long history of research identifies a positive relationship between health and education or other measures of socioeconomic status (e.g. Marmot, Shipley, and Rose, 1984; Marmot et al., 1991; Link and Phelan, 1995). Although part of this association may reflect confounding factors or reverse causation (e.g. poor health at young ages inhibiting educational attainment), careful quasi-experimental designs confirm a causal effect of education in improving health (Lleras-Muney, 2005; Clark and Royer, 2013). Many studies provide evidence of widening education gradients over time, often with differences in both the levels and trends across race groups (Kitagawa and Hauser, 1973; Meara et al., 2008; Cutler et al., 2011; Montez et al., 2011; Olshansky et al., 2012; Hendi, 2017).

In recent years, race and education disparities in mortality rates trends have received increasing attention. This focus is in part due to Case and Deaton's (2015, 2017, 2020) findings of increased mortality rates for some groups of less educated non-Hispanic whites since the start of the 21<sup>st</sup> century and because overall life expectancy in the United States fell on a year-to-year basis from 2014 to 2017 (National Center for Health Statistics, 2017). Understanding the evolution of mortality trends is important to gauge the progress of economic and social indicators. Changes in mortality that vary by race and education also have implications for the incidence of large areas of public spending, including Medicare and Social Security.

However, methodological challenges have frustrated efforts to compare changes in mortality rates over time by education and race. First, levels of schooling have generally risen which, under reasonable assumptions, results in increasing negative selection into the lowest education categories, and potentially into more educated groups as well (Dowd and Hamoudi, 2014; Bound et al., 2015). Changes in average mortality rates over time may reflect such compositional changes, at least in part. Consider the simple case used by Case and Deaton (2020) where individuals are divided into those with and without a college degree. If education is trending upwards, persons who in previous cohorts would not have graduated from college might now do so. Assuming that these marginal changes occur among individuals who, if born earlier, would have had among the lowest mortality rates for non-graduates (but higher rates than prior graduates) then, *ceteris paribus*, the negative selection alone might increase the death rates of both groups. Second, differences in the coding of education over time and across data sources complicate the construction of comparable levels of schooling. Of particular relevance here is that the recording of educational attainment on death certificates was reported as the number of grades completed before 2003 but then gradually switched to the use of categories (e.g. high school graduate, some college, college graduate). The two coding systems are not completely comparable, making the analysis of trends challenging.<sup>1</sup>

In this paper, we attempt to overcome these difficulties by constructing quartiles of educational attainment, based on single years of schooling, for population groups stratified by sex, race/ethnicity (hereafter referred to as race) and five-year age ranges, and then examining how mortality trends from 2001-2018 differ by race and education. The results indicate that this heterogeneity is important. There is little evidence of a consistent relationship between education and mortality rate changes from all causes combined, except for non-Hispanic whites (hereafter "whites) where the largest declines are experienced for the most educated. For non-Hispanic blacks (hereafter "Blacks"), the biggest reductions are for those with intermediate levels of schooling, although the differences across education levels are generally not statistically significant. Mortality trends by education vary less for Hispanics and other races. We also document that, for each race, the highest-educated quartile experienced the greatest percentage declines in mortality, although since these are often from low initial baseline levels, the absolute reductions are frequently small.<sup>2</sup>

We then examine specific causes of death to determine which are associated with increases and decreases in overall mortality rates and identify key differences across race groups and education quartiles. Non-intentional drug deaths constitute the single largest source of increased mortality across all demographic groups. There is also a marked education gradient in fatal overdoses, particularly for Blacks and whites, with the greatest growth among the least educated. While suicides and chronic liver disease have previously been suggested as important causes of the recent mortality increases among low-educated whites, we find that deaths from

<sup>&</sup>lt;sup>1</sup> There are similar difficulties with classifying education in the American Community Survey. See Rostron, et al. (2010) for a further discussion of some of these issues.

<sup>&</sup>lt;sup>2</sup> We use similar methods in a recent paper (Leive and Ruhm, 2020) focusing on educational gradients in all-cause mortality within sex, age and race groups. An important result of that study is that the hypothesis that the worst mortality trends are concentrated at the bottom of the education distribution is overly simplified and, across important dimensions, substantially incorrect. The results of the current study are not inconsistent with these findings because we focus here on race differences across all age groups, rather than on heterogeneity between 5-year age ranges.

respiratory diseases and the variety of illnesses composing our residual category have generally increased at least as much, particularly for white females. The causes of death contributing substantially to declining mortality rates are cardiovascular diseases, cancer, and HIV. However, the sizes and patterns of these changes also vary dramatically across races. For instance, loweducated Black males experience much larger reductions in these causes than corresponding whites, Hispanics, or other races. These heterogeneous patterns suggest limitations in what can be learned about the nature of the favorable or unfavorable health shocks experienced by different population groups when focusing only on all-cause mortality rates. Examining specific causes of death provides a more nuanced understanding of mortality trends.

Before turning to the empirical analysis and results, we provide a conceptual framework to consider how positive and negative health shocks might affect the mortality rates of different education and race groups.

## 2. Conceptual Framework

There are many reasons why mortality rates at a point-in-time might be negatively related to education. For example, the review by Cutler and Meara (2008) identifies at least five nonmutually exclusive, potential sources of a causal effect of education: health behaviors such as drinking, smoking and overeating; income and access to health care; labor market factors whereby highly educated individuals work in safer environments and are more likely to have comprehensive health insurance; information and cognitive skills, where those with more schooling have both better access to information and also greater ability to use it to preserve or restore health; relative position in the social hierarchy, which may directly improve health (e.g. by reducing stress) and indirectly do so by, for example, improving access to medical care and other health-enhancing inputs; social networks, from which the more highly educated may have superior access to financial, physical and emotional support.<sup>3</sup> Each of these may help to explain cross-sectional education gradients in mortality, and changes over time in them could influence its trends.

<sup>&</sup>lt;sup>3</sup> In addition, there may be confounding factors, such as genetic factors and underlying preferences like the ability to delay gratification, that cause both higher education and better health, as well as reverse causation, whereby poor health when young reduces completed education.

It is beyond the scope of this study to examine these determinants in detail. Instead, our goal is to provide descriptive evidence useful to others who are interested in conducting in-depth analyses of the trends that we identify. As a first step, we provide a conceptual framework examining how health shocks, from a wide variety of sources, potentially influence mortality trends for different socioeconomic status (SES) groups, where SES is proxied for by education in our empirical study. This framework will also often be applicable to other types of groups that have initial differences in mortality rates. For instance, it is equally informative for considering racial differences in mortality trends.

In studying changes in mortality rates across SES groups, we consider a model where the probability of death depends on investments in and depreciation or shocks to health capital (Grossman 1972). This section discusses the intuition, with further details provided in Appendix A. Death occurs when health capital falls below some minimum threshold. As a result, the probability of death depends on an individual's initial level of health capital, relative to the threshold, and health "shocks" that change it, as well as exogenous depreciation that we do not model. For a given SES group, we assume there is a distribution of initial health capital. Changes in this distribution over time determine the evolution of that group's mortality rate.

Here it is important to understand that we refer to a health "shock" as the combined net effect of any external (exogenous to the individual) influences on health capital as well as (potentially endogenous) behavioral responses to it. For example, the emergence of COVID-19 represents an external factor that possibly reduces health capital, while the extent to which it actually does so also depends on the ability and willingness of individuals to take actions to mitigate it, such as social isolation and mask-wearing. Similarly, the emergence of a new medical technology may improve health but whether or not it does so could depend on whether an individual has adequate income, health insurance coverage, and information to take advantage of it. In our framework, the net effect of either of these on health capital will be considered a negative health shock (in the first case) or a positive health shock (in the second case). Thus, our empirical analysis does not attempt to disentangle the exogenous health influences and adaptation effects just described, but rather focuses on the combined effect. We take this approach both because our data are aggregated, and because we view the net effect on health capital, and thus mortality, as relevant for policy and for understanding how groups are being differentially affected by changes over time in the determinants of health. To fix ideas, consider a negative shock that produces an equal-sized reduction in health capital for two groups that vary in their initial average levels of the capital stock. Assuming that the distribution of the baseline health capital is monotonically increasing in education, the *absolute* increase in death rates will be larger for the less educated group.<sup>4</sup> This occurs because deaths are "left-tail" events, but more so for higher SES individuals, so that the relevant portion of the distribution over which deaths are being induced is "thicker" for low SES groups. Interestingly, however, high SES individuals may experience bigger *relative* increases in mortality risk from the same-sized negative shock.

These ideas are illustrated in Figure 1, which shows the left tail of cumulative distribution functions (CDF) for health capital, assumed here to be normally distributed and with the same variance but different means for high and low SES groups. The solid lines show the CDFs without a shock and the dotted lines are moved horizontally to the left by the shock, *S*, which is assumed to be of equal size for both groups. Under these assumptions, the negative health shock increases the death rate by more for the low SES group ( $R'_l - R_l$ ) than for high SES individuals ( $R'_h - R_h$ ) due to the position of each group's tail. However, in this example, the relative increase in death rates is greater for the high SES group because their baseline mortality rate is so much lower than for the low SES group ( $R_h$  versus  $R_l$ ).

This conceptual framework implies that an equal-sized negative health shock will lead to larger absolute increases in the death rates of less-educated individuals but will not necessarily result in larger percentage increases. Importantly, it is incorrect to conclude that the less educated experience a greater negative health shock just because of their bigger absolute growth in mortality rates. On the other hand, death rates will only rise more, in absolute terms, for the higher educated group if they are hit with a more negative shock.

Next consider a positive shock, such as an improvement in medical technology. This shifts the distribution of health capital to the right, with death rates declining as more persons are now above the survival threshold. An equal-sized shock translates into a larger absolute mortality rate reduction, but not necessarily a larger percentage reduction, for the low SES group than high SES group, using the converse argument to that for a negative shock described above. Greater

<sup>&</sup>lt;sup>4</sup> While we take the initial differences in baseline health capital as given, there are many reasons to expect these to be increasing with education. For instance, Ehrlich and Vin (2005) hypothesize that more educated individuals, because they have relatively high levels of average wealth, also have greater incentives to invest in life protection.

absolute mortality rate reductions for the less educated therefore do not necessarily mean they experience more beneficial health shocks, whereas smaller decreases strongly point to a less positive health shock for them. Finally, if the mortality rates of more and less educated groups move in the opposite direction, we can infer that the category with adverse (favorable) trend is more negatively (positively) shocked.

As noted above, a similar analysis can be applied when considering race-specific mortality trends. In this case, Blacks are the group with higher baseline rates, compared with the lower initial rates of non-Hispanic whites.

## 3. Data and Methods

## Construction of Death Rates by Education Quartile

We provide a brief overview of the methods of constructing group-specific education percentiles and their use in calculating corresponding mortality rates. A more detailed description of the data and imputation methods is provided in Appendix B. Our analysis spans 2001-2018 and uses three data sources as inputs for calculating death rates at the level of sex, 5-year age groups, race, education quartile, and year. First, we construct sex-age-race-education-specific death counts from the Centers for Disease Control and Prevention *Multiple Cause of Death (MCOD)* files, which provide data on the universe of deaths to U.S. residents in the specified year. Second, we utilize the National Cancer Institute's *Surveillance Epidemiology and End Results (SEER)* database to obtain population counts by sex, age and race. Third, we estimate educational attainment for each sex-age-race group using the Census Bureau's *American Community Survey (ACS)* since *SEER* does not provide information on education.

Combining these sources, we calculate educational quartiles separately by sex, 5-year age bins, gender, race and year. This approach allows the distribution of education to differ across both demographic groups and time periods. We construct death rates for age group a, race r and education quartile i, in year t as:

$$mort_{arit} = \frac{deaths_{arit}}{pop_{arit}} \tag{1}$$

where  $deaths_{arit}$  and  $pop_{arit}$  refer, respectively, to the number of deaths and population of the relevant group. Throughout, we calculate and analyze mortality rates separately for males and females, with the sex subscript excluded from equation (1) and later equations to simplify notation. We restrict analysis to ages 25 to 64 since educational attainment may not be complete for person

younger than 25 and prior mortality selection becomes increasingly important for senior citizens. For brevity, we often refer to the first through fourth education quartiles as Q1 through Q4, respectively, with Q4 indicating the highest education level and Q1 the lowest.

In some analyses, we calculate annual average mortality rates for education quartilespecific race and sex groups, across the full set of ages analyzed. These are obtained as:

$$\widehat{mort}_{rit} = \sum_{a} (w_{ari}^{2018} \times mort_{arit})$$
<sup>(2)</sup>

where  $\widehat{mort}_{rit}$  denotes the death rate for race group *r* and education quartile *i* in year *t* and  $w_{ari}^{2018}$  are weights for each 5-year age group in education quartile *i*, based on 2018 population shares, as defined by:

$$w_{ari}^{2018} = \frac{pop_{ari}^{2018}}{\sum_a pop_{ari}^{2018}}$$

for  $pop_{ari}^{2018}$  the population of age group *a*, race *r* and education quartile *i* in 2018.

Our approach constructs education quartiles that are general across all races, within age and sex groups, rather than also being race-specific. This implies that persons of different races but with the same age, gender and education will be classified in the same quartile in a given year. One result is that the lower quartiles will be disproportionately populated by race groups with below average education levels (e.g. Blacks and Hispanics) while the whites and other races will be over-represented in the higher quartiles. We return to this issue below.

There are multiple complications involved in constructing the mortality rates described by equation (1). First, education is frequently measured in discrete rather than continuous units and the data are not fully comparable across time periods or data sources. If schooling were always a continuous variable, we could easily calculate the group- and year-specific distribution of education and then divide it into percentiles. Since this is not the case, we use or construct single-year measures of education ranging from 0 to 17 years.<sup>5</sup> The *MCOD* and *ACS* sometimes record education in single year increments but frequently instead report it in categories (e.g. high school, some college, college). Appendix B describes the imputation procedures employed to construct single years of education for death and population counts, as well as methods of dealing with other

<sup>&</sup>lt;sup>5</sup> In two cases we combine groups. Persons with one or more years of college, but no bachelor's degree, are assigned a value of 14 years of education. Those with at least a year of post-graduate education are categorized as having 17 years.

complicating factors, such as cases where information on education is missing on the death certificate.

Another issue is that a single year of education may straddle quartiles. For instance, this often occurs for persons with exactly 12 years of schooling. In these cases, we proportionately assign deaths from the overlapping education cell to each quartile, based on population shares. Thus, if 12 years of education ranges from the 21<sup>st</sup> to the 40<sup>th</sup> percentile, we assign one quarter of these deaths to the bottom quartile and three-quarters to Q2. Bound et al. (2015) and Meara, Richards, and Cutler (2008) use a similar approach.

## **Regression Specifications**

A primary objective is to test whether the relationship between mortality trends and educational attainment differs by race. We do so using the regression specification:

$$mort_{arit} = \sum_{a \in A} \sum_{r \in R} \sum_{i=1}^{4} [\beta_{ari} age_a \times race_r \times Q_i] + \sum_{r \in R} [\pi_r trend \times race_r] + \sum_{r \in R} \sum_{i \neq 4} \pi_{ri} trend \times race_r \times Q_i + \epsilon_{arit}$$
(3)

where  $mort_{arit}$  is the death rate for age group *a*, race *r* and education quartile *i* in year *t*;  $Q_1, Q_2$ , and  $Q_3$  denote indicator variables for education quartiles 1, 2, and 3, with the highest quartile,  $Q_4$ , serving as the reference group;  $race_r$  is a vector of indicator variables for races: White, Black, Hispanic, and other non-Hispanics,  $age_a$  is a vector of indicators for 5-year ages from 25-64, and *trend* is a linear time trend. The regression models are estimated separately for men and women, so that each includes 160 age-race-education quartile groups. We cluster standard errors by age, race and education, and weight each cell by its population to obtain nationally-representative estimates.

#### Cause-Specific Death Rates

After examining trends in total mortality rates, we estimate models for specific causes of death. The specifications mirror equation (3), with the cause-specific mortality rates replacing the all-cause rate as dependent variables. We use detailed ICD-10 codes as reported on the death records and select causes of death to analyze using the following procedure. First, we identified the top 10 causes for each of the four ten-year age groups in the overall age range examined (25-34, 35-44, 45-54 and 55-64). We also separately categorize accidental deaths, excluding those

involving drugs, and non-intentional drug fatalities (hereafter simply referred to as drug deaths). Suicides are the final cause of death analyzed and include intentional drug fatalities. We estimate race-ethnicity education quartile trends for each of these 13 causes, as well as for a residual category.<sup>6</sup> We divide the specific sources of death into "major" and "minor" causes, where the criterion for distinguishing between them is whether the magnitude of the trend coefficient exceeds 0.8 for any group.<sup>7</sup> Using this standard, the major causes of death are cardiovascular disease, malignant neoplasms (Cancer), cerebrovascular disease (Stroke), respiratory disease, HIV, drugs, and suicide. The minor causes are diseases of the chronic liver disease, nervous system, kidneys, diabetes, non-drug accidents (Accident) and homicide. ICD-10 codes corresponding to each of these causes are detailed in Appendix Table B1.

## 4. Trends in Total Death Rates

## Descriptive Evidence

This section presents trends in annual death rates, from 2001-2018, for subgroups stratified by race and each of the four education quartiles, calculated using equation (2). Several patterns are consistent for both men (Figure 2a) and women (Figure 2b). Blacks have the highest average mortality rates for all education quartiles and in nearly all periods. The only exception is that Q1 white females have equal or marginally greater death rates in the last few years. Whites almost always have the second-highest mortality rates, with those of Hispanics and other races being much lower. For instance, quartile-specific death rates in 2018 for whites and Blacks are often double those for Hispanics and other races. The magnitude of the race differences declines with rising educational attainment but remains apparent even in the top quartile.

The quartile-specific differences in death rates may understate the racial gap between Blacks and whites. Evidence points to a variety of institutional and psychological factors that make it harder for Blacks to attain the same level of education as whites (Bertrand, Chugh, and Mullainathan 2005; Milkman, Akinola, and Chugh 2012, 2015). In light of these barriers, one might expect Blacks to be healthier than whites at given levels of educational attainment.

 $<sup>^{6}</sup>$  There is one exception. Sepsis is among the leading causes of death for 35-64 year olds but is included in the residual category because the magnitude of its trend coefficient is below 0.2 in all but one case (where it is -0.27) and it is 0.1 or less in 23 of 32 cases.

<sup>&</sup>lt;sup>7</sup> This division represents a natural breakpoint in the data and results in an equal number of major and minor causes, once the residual is included in the latter category.

Specifically, conditioning on education may introduce a form of positive health selection for Blacks relative to whites. Consistent with such selection, 28 to 33 percent of Blacks were in Q1 in 2001 and 2018, compared to just 20 percent of whites (Appendix Table C2). The positive selection is probably even more pronounced for Hispanics, who have the lowest overall amounts of education, providing a likely explanation for at least some of their lower mortality rates. However, the same is not true for other races, who are generally more educated than whites and with particularly high Q4 shares.<sup>8</sup>

We provide a detailed examination of mortality trends in the next section, but some key results are previewed in Figure 2. In particular, Q1 and Q2 whites experienced steady increases in mortality rates from 2001-2018, with more modest growth for Q3 and stable or slightly declining rates for Q4. By contrast, less-educated Blacks had some of the largest declines but starting from the highest levels. Since educational attainment rose more over time for Blacks than whites, especially at the bottom of the distribution (Appendix Table C2), these changes are not due to increasing positive selection for Blacks. Nonetheless, even in 2018, Black mortality rates continued to equal or exceed those of whites. There are few obvious trends for Hispanics or other races. Given that schooling levels are also rising over time for them, relative to whites, the changes in selection raise the possibility that the trends are marginally more favorable than they appear, but their overall rates of mortality are certainly low even without this adjustment.

# Regression Estimates of Trend Differences in All-Cause Mortality Rates

Figure 3 presents regression results from estimating equation (3) for death rates from all causes. The entry for Q4 is the trend "main effect" for race r,  $\hat{\pi}_r$ . Estimates for the remaining groups are calculated as the main effect plus the education quartile-specific trend coefficient. For instance, the estimated trend for Q1 and race r is calculated as  $\hat{\pi}_r + \hat{\pi}_{1r}$ . The 95 percent confidence intervals (CIs) are centered on the Q1 through Q3 total effects and indicate whether these are statistically significantly different from Q4.

<sup>&</sup>lt;sup>8</sup> These selection issues can be avoided by examining overall (not quartile-specific) mortality rates. This is done in Appendix Figure C1, which shows evidence consistent with the patterns of selection just described. For instance, mortality rates trended downwards for Blacks, while remaining essentially constant for whites, but the Black disadvantage remains sizable: Black mortality rates per 100,000 declined from 882 to 709 from 2001 to 2018 for males and from 539 to 427 for females, while barely changing (from 539 to 541 and 334 to 330) for whites.

The education gradient appears strongest for whites of both sexes, where Q1 and Q2 experienced either increased or unchanged mortality rates over time and statistically significantly worse trends than Q4. Q3 also did more poorly than Q4 but the differences are barely significant for men and insignificant for women. By contrast, there is no obvious education gradient for the other three races. None of the quartile differences are statistically significant for Blacks or Hispanics and the point estimates for Blacks suggest larger trend reductions in mortality for the two lowest quartiles. For men of other races, Q4 does better than the other three education quartiles but the differences are modest.

Results from the models just estimated may be influenced by the experiences of groups with the highest baseline mortality rates, such as those in the lower education quartiles, since equal percentage changes in death rates will have larger absolute effects for them. Figure 4 summarizes regression estimates with logs rather than levels of death rates as the dependent variables. The results indicate that Q4 has the largest percentage reductions in mortality rates, with the differences between those of the of the other three quartiles generally being statistically significant. However, these often do not translate into large absolute reductions. For example, death rates for Q4 Hispanic females are predicted to decline by 1.39% more per year than those in the Q1, but this corresponds to just a 0.45 per 100,000 larger annual reduction in the overall rate. There are no clear education gradients for the lower three quartiles, except for white females, where larger percentage declines are again observed for the more educated. Blacks and Hispanics have the most favorable log mortality trends while less-educated whites show the largest percentage increases.

## 5. Specific Causes of Death

We next examine trends in mortality rates from the specific causes described above. Figures 5 and 6 summarize the results for men and women. We display point estimates without confidence intervals or standard errors to aid interpretability, but report both the coefficients and standard errors in Appendix Tables C3 and C4.

Increases in (non-intentional) drug deaths are the single largest component contributing to rising mortality rates for all sex, race and education groups. The changes in fatal overdose rates are monotonically declining in education for Blacks and whites and, to a lesser extent, for Hispanics and other races. No other cause consistently contributes to trend increases in mortality. For whites, suicide plays a role, particularly among men, as do respiratory diseases for the two lower quartiles of both sexes. Chronic liver disease deaths contribute to higher mortality rates for whites in the bottom three quartiles, but the impact is modest. To illustrate, the estimated annual mortality rate increases per 100,000 for Q1 white males from drugs, suicide, respiratory and chronic liver diseases are 4.34, 0.92, 0.74 and 0.72, while for corresponding white females they are 2.74, 0.35, 0.97 and 0.64.

The causes of death contributing substantially to declining mortality rates are cardiovascular disease, cancer, HIV, and sometimes strokes. However, the sizes and patterns of these effects vary dramatically across races. Reductions in cancer and cardiovascular disease death rates are of roughly equal magnitude for white males but there is no education gradient and they are the only two causes of any real importance. Decreases in cancer mortality are by far the most substantial for white females, especially for the highly educated, with cardiovascular disease mortality also playing some role but only for Q3 and Q4.

The patterns for Blacks differ markedly and these dissimilarities explain the much larger overall mortality reductions they experience, particularly among the less educated. The three most important contributors to declining mortality among Black males are cardiovascular disease, cancer and HIV. These decreases are particularly large for Q1 (-4.21, -4.61 and -3.63 per 100,000) and Q2 (-5.52, -5.29 and -3.34 per 100,000). Stroke mortality also falls substantially, although by considerably less than the three causes just mentioned. Reductions in death rates from these causes have been markedly greater among the less educated than for Q4. Cardiovascular and cancer deaths also fall sharply over time for Black females, although considerably less so for Q1 (-1.83 and -0.94 per 100,000) than the other three quartiles (ranging from -3.36 to -3.80 and -2.83 to 2.94 per 100,000). Conversely, the decrease in HIV mortality is largely restricted to Q1 and Q2 (-1.64 and -1.39 per 100,000 for these two quartiles versus -0.59 and -0.36 per 100,000 for Q3 and Q4). The patterns for Hispanics and other races are generally similar to those just described for Blacks, except that the magnitudes of the mortality changes are much smaller and declines in HIV are less important.

By construction, the minor causes of death play relatively small roles (Figures 5b and 6b). We display the mortality rate changes from these sources on the same scale as the major causes to illustrate the differences in relative magnitudes between them. Most important among these are deaths from chronic liver disease for less-educated whites, but the annual changes never exceed 0.72 per 100,000 and are dwarfed by the major sources of death discussed previously. There is an

education gradient in the residual "all other causes" category for whites – the estimated annual increases are 1.39 and 1.50 per 100,000 for Q1 males and females, with much smaller changes for their Q3 and Q4 counterparts – but no corresponding education relationship for other races. Further investigation into the residual causes revealed that their contribution reflects the cumulative influence of death from many diseases that each exhibited small increases over time.

We also conducted a corresponding analysis of changes in log death rates (see Appendix Figures C2 and C3 and Tables C5 and C6). For the most part, the educational gradients are similar for trends in logs and levels. In particular: less-educated whites and Blacks experienced the largest percentage, as well as absolute, increases in drug mortality rates; there are relatively weak education gradients in cardiovascular and cancer mortality trends for white males but with somewhat stronger gradients for white and Black females; the large absolute decreases over time in black male cardiovascular and cancer mortality reflect fairly similar relative changes but starting from much higher baseline rates. Also, noteworthy are the extremely large percentage reductions in HIV mortality for virtually all groups, without much of an educational gradient for men but with suggestive evidence of one for females other than Hispanics.

## 6. Discussion

This analysis supplies descriptive evidence on mortality trends at the beginning of the 21<sup>st</sup> century by sex, education quartile and race. In context of the conceptual framework developed in Section 2, the results for all-cause mortality suggest that Blacks faced the most positive overall health shocks and non-Hispanic whites the most negative ones, as evidenced by the large absolute and percentage decreases for the former group and corresponding increases for the latter.<sup>9</sup> Within race groups, the education gradients in mortality trends vary. Among whites, the lower education quartiles experienced increases or no changes in death rates, while those of the most educated declined, implying positive shocks for the highly educated and neutral or negative ones for the lower quartiles. For Blacks, there were no clear educational gradients in absolute mortality rates but with the largest relative declines for the highlest quartile. Since equal-sized increases in health capital are predicted to decrease absolute mortality rates by more for the less educated, but possibly

<sup>&</sup>lt;sup>9</sup> There are many potential sources of these racial differences. For instance, in their analysis of health capital investments, Ehrlich and Chuma (1990) identify more rapid improvements in life expectancy, although starting from lower levels, for nonwhites than whites throughout much of the 20<sup>th</sup> century. Their theoretical model suggests that this may be due greater increases in permanent incomes for nonwhites.

with smaller percentage reductions, these results suggest larger positive shocks for those with more schooling. Given the small overall changes for Hispanics and other nonwhites, the size of any shocks appeared to be modest compared to those experienced by whites or Blacks.

These overall patterns conceal considerable heterogeneity across causes of death. For example, one reason non-Hispanic whites generally experienced the least favorable mortality trends over the first two decades of the 21<sup>st</sup> century is because they have been hardest hit by the fatal drug epidemic, as has been detailed elsewhere (e.g. Ruhm, 2019; Case and Deaton, 2020). More generally, this negative health shock increased the cause-specific fatality rates of all groups but with by far the largest growth in both absolute and percentage drug death rates among less-educated whites and Blacks. Whites were more affected than Blacks or Hispanics early in the fatal drug epidemic, because they were more widely prescribed opioids (Anderson, Green and Payne, 2009; Singhal, Tien and Hsia, 2016). Less-educated Blacks, particularly males, experienced large increases later as the main source of drug mortality shifted from prescribed to illicit opioids (such as heroin and fentanyl).

Other sources of higher mortality have been less consequential. Suicides grew over time for whites but considerably less for other races. Among the least-educated whites, respiratory disease deaths increased more than suicides for women and by similar amounts for men. The variety of illnesses composing our residual category grew at least as much as those from either suicide or chronic liver disease for less-educated whites. Notably, changes in the education gradients for all of these causes have been much less steep than the extreme differences observed for drugs. This pattern also holds for Blacks and, to a lesser extent, Hispanics and other races.

Reductions in cancer, cardiovascular and HIV deaths are the dominant causes associated with declining mortality rates. These generally reflect bigger reductions, in both absolute and percentage terms, for Blacks than whites, and higher versus lower education quartiles suggesting, in terms of our conceptual model, more positive health shocks for these groups. However, Black males represent an important exception, with the largest absolute mortality rate reductions from these causes occurring among Q1 and Q2. In combination with fairly similar percentage changes, this may indicate that Black males experienced equal or larger positive shocks than their more educated counterparts.

Prior research suggests some reasons for these decreases in mortality. For instance, the large declines in cardiovascular and cancer deaths for blacks may have occurred because of

particularly large declines in smoking for them since the mid-1960s (Garrett et al. 2011). Health insurance coverage expansions may have also reduced inequalities in access to medical care; for instance, recent evidence highlights large effects of Medicaid insurance expansions (Sommers et al. 2012, Sommers 2017, Miller et al. 2019, Borgschulte and Vogler 2020), although gaps still persist (Buchmueller et al. 2016). However, further research is needed to understand the limited progress among the least-educated quartiles (other than Black men), particularly since slowdowns in reducing cardiovascular mortality are seen as a key factor restraining improvements in life expectancy (Mehta, Abrams, and Myrskylä 2020).

Enormous progress has been made in reducing HIV deaths and, as documented above, this has been particularly important for Blacks in the lower two education quartiles. This reflects the particularly devastating death toll of HIV on less-educated Blacks during the 1980s and early 1990s (Centers for Disease Control and Prevention, 2001), combined with the increasing use of antiretroviral drugs, starting in the late 1990s and continuing into the 21<sup>st</sup> century, that reversed many of the effects of this initial negative health shock (Diamond, 2018).

We are hesitant to draw strong implications for Hispanics and other races, given their generally small absolute, although not percentage, changes in death rates and the likelihood that the composition of these groups may have changed over time. Here, we simply note that the trends in drug, cardiovascular, cancer and HIV mortality are usually in the same direction but of more limited magnitude than for whites or Blacks, leading to modest overall death rate changes that show no clear education gradient.

A principal conclusion of this analysis is that single factor explanations for mortality trends are likely to be inadequate given the heterogeneity of experiences across population groups and that useful information can be gained by examining differences in trends by cause of death. This result is consistent with the recent study of Novosad, Rafkin, and Asher (2020) who use partial identification methods to examine trends in mortality. A corollary of this conclusion is that grouping together different causes of death may conceal important differences. For example, Case and Deaton (2015, 2017, 2020) combine deaths from drugs, suicide and chronic liver disease into a single "deaths of despair" category. Yet we find that the fatality trends differ dramatically for these causes within and across race and sex groups. As discussed, drug deaths are the dominant cause of mortality growth for the less educated. Trends in suicide and chronic liver disease are of secondary importance, with larger fatality rate increases often observed from respiratory diseases.<sup>10</sup>

Several caveats should be kept in mind when interpreting our results. First, larger annual decreases in death rates do not automatically imply better overall status. In particular, Blacks experienced the largest mortality reductions over the first two decades of the 21st century, but their death rates in 2018 almost always remained higher than those of other races. Second, our analysis does not fully control for cross-sectional compositional differences or corresponding changes occurring over time. We have noted that these might lead us to understate the mortality disadvantage of Blacks and overstate the corresponding advantage of Hispanics in any given year. Moreover, they might also affect the estimated trends. For instance, the extent to which Blacks and Hispanics are disproportionately represented in the lower education quartiles has fallen modestly over time (Appendix Table C2). This suggests a small decrease in the extent of favorable selection for them, possibly leading to slightly smaller mortality reductions (or larger increases) than if this selectivity had remained constant. Third, there could be additional changes over time in the composition of some groups, especially Hispanics and other races, due to migration into the United States from other countries (Bundiman, et al., 2020). Fourth, stated amounts of schooling may not correspond to true levels of human capital. This could reflect changes in the quality of education or of its reporting. A particular issue is that the high school graduate group includes persons with a GED or other high school equivalency exam which, at least in prior years, did not indicate the same level of skill acquisition as actual graduation from high school (Heckman, Humphries and Mader, 2011).<sup>11</sup>

Finally, the COVID-19 pandemic may have modified or reversed some of the trends documented above. In particular, the large declines in mortality rates experienced by low educated Blacks may have stalled or reversed, given data indicating that Blacks and Hispanics have experienced particularly high rates of COVID-related hospitalizations and deaths (Gross, et al.,

<sup>&</sup>lt;sup>10</sup> Consistent with the importance of heterogeneity, studies of the causal effects of economic shocks and policies show different results by cause of death. For example, Pierce and Schott (2020) find that trade shocks increase drug mortality but not deaths from suicides or alcohol-related causes. Dow et al. (2020) indicate that expansions in the EITC and the minimum wage reduce non-drug suicides, but have no effect of deaths from drugs or alcohol-related causes.

<sup>&</sup>lt;sup>11</sup> In addition to the General Educational Development (GED) credential, since 2014 high school equivalency exams include the Test Assessing Secondary Completion (TASC) and the High School Equivalency Test (HiSET). Appendix Figure C4 shows that the share of people coded as high school graduates with a GED differs little by race and that the trends have remained flat since 2008, which is the first year that GEDs were separately recorded in the ACS, indicating that this is unlikely to be an important issue for our analysis.

2020; Price-Haywood, et al., 2020). The pandemic's health effects on different populations have become an active area of research and one that will continue to be important for both academics and policymakers. We hope our methods and findings will be useful to researchers interested in estimating these and other changes in mortality by educational attainment for different demographic groups.

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Figure 1. Probability of death with and without health shock by SES



Note: Figure shows the cumulative distribution function for health capital which is assumed to have an equal variance for high and low SES groups (proxied in our empirical analysis by education) but with a higher mean value for the former group. A health shock *S* causes an equal left-ward shift of the distribution for both groups. Death occurs if health capital falls below  $H^0$ , and the fraction of the group dying is shown by the dotted lines extending to the *y*-axis. Figure shows the case where normal distributions in the "without shock" case have means of 0 and 1, equal variances of 1.0 and where the health shock shifts both distributions to the left by 0.075 standard deviations.

# Figure 2 Trends in Death Rates for 25-64 Year Olds from 2001-2018, by Education Quartile



Note: Figure shows mortality rates per 100,000 by race, education quartile, and sex from 2001 to 2018. Mortality rates are age-adjusted to 2018. Panel (a) displays trends for males and Panel (b) displays trends for females. Education quartiles are calculated separately by age, sex, and year, as described in the text.



Figure 3: Regression Estimates for Death Rate Trends of 25-64 Year Olds, by Education Quartile

Note: Figure shows regression estimates of the interactions between race, trend, and quartile from equation (3) for death rates, with males in Panel (a) and females in Panel (b). The estimate for Q4 is the interaction of trend and race. The estimates for Q1, Q2, and Q3 are calculated as the main effect plus the education quartile-specific trend coefficient. Whiskers show 95% confidence intervals clustered by age, race and education on the difference between each quartile and Q4. Regression is weighted by population of each age-race-year cell to obtain nationally-representative estimates.



Figure 4: Regression Estimates for Log Death Rate Trends of 25-64 Year Olds, by Education Quartile

Note: Figure shows regression estimates of the interactions between race, trend, and quartile from equation (3) for log death rates, with males in Panel (a) and females in Panel (b). The estimate for Q4 is the interaction of trend and race. The estimates for Q1, Q2, and Q3 are calculated as the main effect plus the education quartile-specific trend coefficient. Whiskers show 95% confidence intervals clustered by age, race and education on the difference between each quartile and Q4. Regression is weighted by population of each age-race-year cell to obtain nationally-representative estimates.



Figure 5: Regression Estimates by Causes of Death for 25-64 Year Old Males, By Education Quartile

Note: Figure shows regression estimates of the interactions between race, trend, and quartile from equation (3) for cause-specific death rates among males. Results for major causes are presented in Panel (a) and minor causes in Panel (b) Standard errors are presented in Appendix Table C3. The estimate for Q4 is the interaction of trend and race. The estimates for Q1, Q2, and Q3 are calculated as the main effect plus the education quartile-specific trend coefficient. Regression is weighted by population of each age-race-year cell to obtain nationally-representative estimates.



Figure 6: Regression Estimates by Causes of Death for 25-64 Year Old Females, By Education Quartile

Note: Figure shows regression estimates of the interactions between race, trend, and quartile from equation (3) for cause-specific death rates among females. Results for major causes are presented in Panel (a) and minor causes in Panel (b) Standard errors are presented in Appendix Table C4. The estimate for Q4 is the interaction of trend and race. The estimates for Q1, Q2, and Q3 are calculated as the main effect plus the education quartile-specific trend coefficient. Regression is weighted by population of each age-race-year cell to obtain nationally-representative estimates.

## **Appendix A Conceptual Framework**

This Appendix presents additional details on the health capital framework discussed in Section 2 of the main text. We consider a stylized model that incorporates the key ideas of standard health capital models (Grossman 1972). In particular, health capital is based on investments, which are endogenously chosen to maximize utility, depreciation, which we treat as exogenous, and health shocks. Death occurs when health capital falls below some minimum threshold for survival. On average, health capital is positively related to socioeconomic status (SES), which is proxied in our empirical analysis by education. We allow for a distribution of health capital for each group, which accounts for the realistic feature that some people with lower SES are healthier than some people with higher SES, although health rises with education on average.

As emphasized in the main text, our current usage of the term "health shock" refers to the combined effect of any external (exogenous to the individual) influences on health as well as potentially endogenous behavioral responses to it. Thus, we refer to the size of the shock as the extent to which it raises or lowers health capital, on net. The equal-sized shock studied below changes the stock of health capital of the different groups by the same amount.

Our interest is in studying what this framework predicts for changes in absolute and relative mortality rates when two different SES face groups face the same health shock. Given this objective, we are primarily concerned with how the position in the tail of each SES group's distribution changes over time, since those movements map directly to changes in mortality rates. We are not focused on structurally modeling the primitives of individual utility because our empirical methods are descriptive and measure changes in aggregate mortality rates, rather than the micro-foundations that lead to those changes. The "shocks" we refer to may capture many different phenomena that are external to the individual, such as improvements in medical technology, greater availability and prescribing of opioids, or environmental events, as well as the behavioral responses that may accompany them.

For simplicity, assume there are two groups: those with low SES and those with high SES. The stock of latent (unobserved) health capital at the end of a period for members of these two groups, denoted by  $H_l$  and  $H_h$ , is  $\mu_l + \varepsilon$  and  $\mu_h + \varepsilon$ , for  $\mu_h = \mu_l + \lambda$ , with  $\lambda > 0$  and  $\varepsilon$  a random variable normalized without loss of generalization as  $\varepsilon \sim (0,1)$ . Death occurs if health capital falls below a threshold level  $H^0$ . For group *j*, where  $j \in \{l, h\}$ , this occurs if:  $H_j \leq H^0 \quad or \quad \mu_j + \varepsilon \leq H^0.$ 

Defining  $X = H^0 - \mu_l$ , the probability of death for the low and high SES groups are:

$$R_l = F(X) \tag{A.1a}$$

and

$$R_h = F(X - \lambda) \tag{A.1b}$$

where  $F(\cdot)$  is the cumulative distribution function of the relevant distribution. High SES individuals have lower death rates than their low SES counterparts:  $R_h < R_l$ , since  $\lambda > 0$ .

We begin by considering the effects of a health shock, *S*, that has a uniformly negative effect on the health capital of all individuals. The new health capital for members of group *j* is then  $H'_j = \mu_j - S + \varepsilon$  and the risk of death of low and high SES persons becomes

$$R'_l = F(X+S) \tag{A.2a}$$

and

$$R'_{h} = F(X + S - \lambda). \tag{A.2b}$$

The probability of death has risen for both groups; however, what we are interested in are the absolute and relative changes in these risks. The absolute changes are:

$$\Delta R_l = R'_l - R_l = F(X+S) - F(X) \tag{A.3a}$$

and

$$\Delta R_h = R'_h - R_h = F(X + S - \lambda) - F(X - \lambda).$$
(A.3b)

The absolute change in the probability of death will then be higher for low SES group members if  $\Delta R_l > \Delta R_h$ , which occurs if:

$$F(X+S) - F(X+S-\lambda) > F(X) - F(X-\lambda).$$
(A.4)

This condition holds as long as the density of *F* is increasing on the interval to the left of  $H_0$  and the shock is "small" (such that  $S < \mu_l - H^0$ ). Intuitively, this occurs if deaths are "left-tail" events and the shock is not so large as to change this.

Next, consider the conditions under which the high SES group has higher *relative* increases in mortality than the low SES group, even though the absolute increase is smaller. This occurs by definition if  $\frac{\Delta R_L}{R_L} < \frac{\Delta R_H}{R_H}$ , which is equivalent to:

$$\frac{F(X+S)-F(X)}{F(X)} < \frac{F(X+S-\lambda)-F(X-\lambda)}{F(X-\lambda)}$$
(A.5)

Rearranging the inequality and canceling terms yields:

$$\frac{F(X-\lambda)}{F(X)} < \frac{F(X+S-\lambda)}{F(X+S)}.$$
(A.6)

This expression holds for S > 0 if the density of F is increasing on the interval  $(-\infty, H_0]$  which will occur for left-tail events with many common distributions (e.g. normal and T-distributions), although it need not be the case for others.

The analysis of positive health shocks is largely the reverse of that just described, with greater absolute mortality reductions anticipated for the less advantaged and with unclear predictions for relative decreases in death rates. To the extent that improvements in medical technology or other similar positive health shocks are the norm, we expect to observe larger absolute (although again not necessarily relative) mortality improvements for disadvantaged groups with relatively elevated initial death rates. Mortality rates will only trend in opposite directions if one of the SES groups experiences a positive shock while the other group experiences one that is negative.

Finally, note that this analysis will apply, with little modification, to the analysis of other types of groups that have differing baseline mortality rates. Specifically, for our application, we can compare trends for groups with high initial death rates, such as Blacks, to those with lower baseline rates, like whites and other (non-Black) race or ethnic groups.

## **Appendix B. Construction of Death Rates by Education Quartiles**

This Appendix describes our methods for constructing death rates by education quartile. We first detail the procedures used for calculating death and population counts by years of education and demographic characteristics. These counts are then used to construct death rates by group, and represent the data employed in the main regressions. Section B2 describes the specific causes of death analyzed.

## B1. Procedure to Estimate Population and Deaths by Years of Education

To estimate death counts (the numerator in mortality rate calculations), we sum all yearly deaths for the specified gender-age-race-educational attainment cell using the *MCOD* data. We drop approximately 5,400 observations with missing age out of over 45.8 million recorded deaths during this period. Prior to 2003, information on single year of education is provided on the death certificates. Beginning in 2003, approximately 16 percent of deaths measure education in one of seven categories: 8<sup>th</sup> grade or less, 9-12<sup>th</sup> grades without a diploma, high school, some college (no degree), bachelor's degree, master's degree, or a doctorate/professional degree. By 2007, just over half of records classify education using these coarser groups, rather than single years, and in 2017, nearly all deaths are recorded using the seven education categories.

For some classifications, we can reasonably assign a single year of education. Specifically, we treat high school graduation as 12 years, some college or associate's degree as 14 years, a bachelor's degree as 16 years, and a master's or doctorate/professional degree as 17 years. However, for the other education categories (e.g. "<= 8th grade" and "9-12th grade, no diploma"), this assignment cannot be done, since they include people with substantially different years of schooling. Therefore, we developed an imputation procedure for these cases. To implement the procedure, we first calculate the fraction of single year educational attainment, when these are provided, comprising each of the broader categories. For example, for grades 9 to 12 without a diploma, we calculate the percentages of deaths occurring among persons where the death certificate specifies 9, 10 and 11 years of education, respectively (and not just the broader education on a quadratic trend in years and a full set of age, sex, and race interactions, with the sample restricted to those in the specified broader education categories (e.g. 9 to 12 years of education without a diploma). To ensure a large enough sample to make these extrapolations, we use wider than five-

year age bins, specifically, combining those 25-39, 40-54 and 55-74 years of age. We restrict the time period for these regressions to be prior to 2011, since after 2010 fewer than 30 percent of death certificates record single years of education. Next we use these estimates to predict the probability of a particular number of years of schooling for persons with information only on the broad education category, conditional on the three aforementioned age categories as well as sex, race and year of death.

A potential threat to this strategy is that states adopting broad education categories might have different distributions of within-category educational attainment than those that do not. To examine whether this is a problem, we first classified states according to whether they predominantly reported continuous years of education in 2010 versus those that primarily used the broader education categories. We compared the distribution of deaths across these two classifications for those with 9, 10 and 11 years of education prior to 2003 (when all states used continuous education), conditional on having between 9 and 12 years of education without a high school degree. The distributions were nearly identical across the two types of states. We repeated this for those with 8th grade or less education, and again found very similar distributions of 0 through 8 years of education in the pre-2003 period for states using different classification methods in 2010.<sup>12</sup> These results suggest that the educational distributions in earlier years provide a useful indication of the predicted distributions in later ones.

Educational attainment is missing for roughly 5 percent of death certificates. In these cases, we assume that the education distribution within a given year, race, sex, 5-year age bin is the same for these missing certificates as when schooling is reported and include such deaths in the analysis using this allocation.

To estimate population, the denominator of death rates, we first obtain counts by age, gender, year and race group from the *SEER*. Since, the *SEER* does not include education, we computed the distribution of education shares within these cells using the *ACS* to estimate population counts by single year of education and demographic sub-group. While information on education is also

<sup>&</sup>lt;sup>12</sup> Since the data includes over 2 million deaths each year, the distributions of educational attainment between the two groups of states are statistically significant at any conventional level based on chi-squared tests. However, the magnitudes of the differences in early years between states that later code education predominantly using categories as opposed to single years are extremely small. For example, 86.59% of deaths among those with either 10 or 11 years of education had 10 years of education in states that later use categories vs. 86.79% in states that later use single years. Among those with 8 years or less of education, 51.85% have 8 years in the states later using categories vs. 50.93% in states that continue to use single years.

available from the 2000 Census, analysis indicated that these data were not fully consistent with those reported in the ACS. Since we also use the ACS for other years, we choose to exclusively use the ACS to maintain comparability over time.

Our procedure for calculating years of education is straightforward for categories up to grade 12 starting in 2008, since education is measured in single year bins in the *ACS*. Prior to 2008, grades below 8<sup>th</sup> grade were combined (nursery school to 4th grade, 5<sup>th</sup> and 6<sup>th</sup> grade, and 7<sup>th</sup> and 8<sup>th</sup> grade). We split these into each of the possible grades based on the distribution within a given race, sex and wide age bin for years 2008-2017. We record "no schooling completed", "nursery school, preschool", and "kindergarten" as 0 years of education. We assume a high school degree is equivalent to 12 years, classify 12<sup>th</sup> grade without a diploma as 11 years of schooling, and less than one year of college as 12 years. We assign "1 or more years of college credit, no degree" or an associate's degree as 14 years and assume that a college degree without additional education is equivalent to 16 years. Education beyond a college degree is coded as 17 years. Using *ACS* sample weights, we then calculate the distribution of education for each of 0 to 17 years (excluding 13 or 15 years) by 5-year age categories, gender, survey year, and sometimes race.

It is important to acknowledge the assumptions implied by proportionately assigning deaths across quartiles for years of education that span thresholds. Novosad, Ravkin and Asher (2020) note that the proportional assignment, which is also used by Meara, Richards, and Cutler (2008) and Bound et al. (2015), treats mortality rates as being flat within education categories, and only allow for discrete changes across them. By contrast, their method assumes a continuous latent education rank distribution, with mortality rates weakly declining in this rank. Assuming a step-function of mortality with proportional assignment is undesirable when education bins are wide but is less problematic when education is measured in single years of schooling, as in our analysis. Where Novosad, Ravkin and Asher (2020) consider four education bins (less than high school, high school, some college, and bachelor's degree or higher), while we split education into 16 (0, 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 14, 16 and 17 years). Given the finer granularity of our measure of education as reasonable and potentially advantageous compared to analyses that divide the sample into just four education categories.

#### B2. Classification of Causes of Death

Table B1 presents the ICD-10 codes used to classify the causes of death into the specific sources used in the regression analysis. We also have a residual "all other causes" category that includes any deaths other than for the 13 specific causes detailed below. Some drug deaths are intentional and so, in principle, could be included either in the drug fatality category or with other types of suicides. We have chosen to do the latter, so as not to understate the (relatively small) contribution of intentional deaths to mortality rate trends. Our accidental death category excludes accidental drug fatalities, which are separately analyzed given their particular importance.

А	ppendix Table B1: Causes of Death and ICD-10 Codes
Abbreviation	Description (ICD-10 Codes)
Cardiovascular	Major cardiovascular diseases (I00-I78)
Cancer	Malignant neoplasms (C00-C97)
Diabetes	Diabetes mellitus (E10-E14)
HIV	Human immunodeficiency virus (B20-B24)
Kidney	Nephritis, nephrotic syndrome, nephrosis (N00-N07,N17-N19,N25-N27)
Liver	Chronic liver disease and cirrhosis (K70, K73, K74)
Nerve	Nervous system diseases (G00-G99)
Respiratory	Respiratory diseases (J00-J98)
Stroke	Cerebrovascular diseases (I60-I69)
Drug (Non-Suicide)	Drug poisoning: accidental, undetermined intent, assault (X40-X44,X60-
	X64,X85,Y10-Y14)
Accident (Non-Drug)	Accidents, other than drugs (V01-X39, X45-X59, Y85-Y86)
Homicide	Homicide (*U01-*U02,X85-Y09,Y87.1)
Suicide	Intentional deaths, including drugs (*U03,X60-X84, Y87.0)

# **Appendix C. Supplemental Results**

Appendix Table (	C1: Average Years o	f Schooling by Educa	ation Quartile and Sex	x, 2001 and 2018				
Education	Ma	ıles	Females					
Quartile	2001	2018	2001	2018				
1	9.58	10.07	9.82	10.29				
2	12.05	12.32	12.10	12.74				
3	13.98	14.51	13.81	14.74				
4	16.40	16.47	16.17	16.53				

Note: Table shows average years of completed school by education threshold for 25-64 year olds, calculated as discussed in the text.

Appendix Tuble 02. Education Quartice Distributions (Think Race Groups													
Education	Non-H	ispanic	Non-Hisp	anic Black	Hisp	oanic	Other						
Education	W	hite											
Quartile	2001	2018	2001	2018	2001	2018	2001	2018					
				Males									
1	19.84	19.66	32.95	29.81	51.82	44.71	20.89	18.90					
2	25.63	24.96	28.57	30.10	21.01	24.80	17.38	17.14					
3	26.41	26.70	24.94	25.02	16.86	18.78	23.48	24.16					
4	28.13	28.68	13.53	15.07	10.31	11.72	38.25	39.80					
				Females									
1	19.92	19.70	32.05	28.15	51.12	45.09	25.40	22.23					
2	25.86	25.09	26.34	28.93	21.01	24.59	18.28	18.40					
3	26.42	26.80	25.00	24.67	16.61	17.97	22.50	25.12					
4	27.80	28.41	16.61	18.26	11.25	12.35	33.82	34.25					

Appendix Table C2: Education Quartile Distributions Within Race Groups

Note: Table shows the distribution across education quartiles (in percentages) for the 25-64 year olds in specified race group and year. These do not total 25 percent in each case because we utilize general rather than race-specific education quartile thresholds.

	WNH					Bla	cks	Tor death	Tutes by (	Hisp	anics		Other			
	Q1	Q2	Q3	Q4	Q1	Q2	Q3	Q4	Q1	Q2	Q3	Q4	Q1	Q2	Q3	Q4
Total	5.09	2.28	0.48	-3.81	-11.43	-15.93	-8.00	-9.14	-3.46	-5.75	-1.81	-3.70	-0.26	0.48	0.06	-2.06
	(0.92)	(1.15)	(1.62)	(1.59)	(3.57)	(4.58)	(3.02)	(2.89)	(1.10)	(2.04)	(0.89)	(1.23)	(1.31)	(0.91)	(0.70)	(0.72)
Cardiovascular	-1.72	-1.76	-1.28	-1.67	-4.21	-5.52	-2.79	-3.13	-1.38	-1.72	-0.75	-1.11	-1.29	-0.58	-0.27	-0.76
	(0.75)	(0.78)	(0.78)	(0.68)	(1.65)	(2.22)	(1.45)	(1.36)	(0.64)	(0.82)	(0.38)	(0.50)	(0.80)	(0.55)	(0.23)	(0.31)
Cancer	-1.90	-2.43	-1.82	-2.10	-4.61	-5.29	-3.05	-2.51	-0.69	-1.35	-0.62	-1.07	-0.85	-0.86	-0.67	-0.77
	(0.63)	(0.97)	(0.94)	(0.95)	(1.66)	(1.99)	(1.43)	(1.14)	(0.28)	(0.62)	(0.30)	(0.50)	(0.26)	(0.44)	(0.39)	(0.32)
Drugs	4.34	3.43	1.69	0.44	2.52	1.80	0.94	0.11	0.81	0.83	0.60	0.15	1.04	1.06	0.49	0.11
-	(0.42)	(0.40)	(0.25)	(0.05)	(0.39)	(0.36)	(0.23)	(0.08)	(0.12)	(0.11)	(0.08)	(0.03)	(0.10)	(0.07)	(0.06)	(0.02)
HIV	-0.33	-0.29	-0.23	-0.16	-3.63	-3.34	-1.78	-1.28	-0.82	-1.21	-0.56	-0.46	-0.16	-0.18	-0.10	-0.05
	(0.12)	(0.09)	(0.08)	(0.04)	(0.83)	(0.73)	(0.40)	(0.28)	(0.18)	(0.31)	(0.16)	(0.11)	(0.06)	(0.07)	(0.03)	(0.02)
Suicides	0.92	0.87	0.82	0.34	0.10	0.06	0.17	0.12	0.14	0.09	0.22	0.13	0.45	0.32	0.27	0.10
	(0.05)	(0.04)	(0.06)	(0.04)	(0.06)	(0.03)	(0.03)	(0.02)	(0.06)	(0.06)	(0.03)	(0.03)	(0.08)	(0.07)	(0.05)	(0.03)
Respiratory	0.74	0.47	0.05	-0.16	-0.13	-0.35	-0.17	-0.22	0.00	-0.13	0.02	-0.11	-0.08	0.10	0.06	-0.04
	(0.29)	(0.19)	(0.13)	(0.09)	(0.13)	(0.10)	(0.15)	(0.09)	(0.02)	(0.06)	(0.03)	(0.05)	(0.13)	(0.08)	(0.04)	(0.04)
Stroke	-0.09	-0.09	-0.10	-0.15	-0.97	-1.01	-0.56	-0.45	-0.27	-0.25	-0.11	-0.12	-0.29	-0.18	-0.15	-0.20
	(0.06)	(0.05)	(0.06)	(0.06)	(0.34)	(0.38)	(0.27)	(0.19)	(0.10)	(0.10)	(0.05)	(0.07)	(0.18)	(0.12)	(0.05)	(0.09)
Liver	0.72	0.55	0.41	0.09	-0.25	-0.40	-0.20	-0.13	-0.12	-0.34	-0.01	-0.07	0.38	0.30	0.16	0.00
	(0.37)	(0.26)	(0.13)	(0.02)	(0.27)	(0.22)	(0.13)	(0.05)	(0.16)	(0.20)	(0.08)	(0.04)	(0.10)	(0.05)	(0.07)	(0.02)
Kidney	0.09	0.06	0.04	-0.02	-0.29	-0.28	-0.04	-0.14	0.01	-0.03	0.00	-0.03	0.02	0.01	0.04	-0.02
	(0.03)	(0.02)	(0.02)	(0.01)	(0.11)	(0.10)	(0.03)	(0.05)	(0.01)	(0.02)	(0.01)	(0.02)	(0.02)	(0.02)	(0.02)	(0.02)
Diabetes	0.21	0.17	0.10	-0.07	-0.14	-0.23	-0.06	-0.26	-0.19	-0.17	-0.02	-0.08	0.06	0.32	0.20	-0.05
	(0.06)	(0.04)	(0.06)	(0.03)	(0.17)	(0.16)	(0.13)	(0.13)	(0.14)	(0.10)	(0.08)	(0.05)	(0.11)	(0.09)	(0.06)	(0.05)
Nerve	0.43	0.26	0.18	0.04	0.39	0.20	0.16	0.04	0.14	0.09	0.07	0.00	0.20	0.21	0.10	0.02
	(0.12)	(0.07)	(0.08)	(0.04)	(0.13)	(0.07)	(0.07)	(0.07)	(0.05)	(0.03)	(0.04)	(0.02)	(0.03)	(0.07)	(0.03)	(0.02)
Accidents	0.16	0.01	0.03	-0.26	-0.17	-0.48	-0.13	-0.38	-0.51	-0.50	-0.21	-0.40	-0.02	-0.19	-0.21	-0.24
	(0.29)	(0.22)	(0.16)	(0.04)	(0.21)	(0.15)	(0.08)	(0.04)	(0.13)	(0.11)	(0.06)	(0.06)	(0.13)	(0.13)	(0.07)	(0.04)
Homicides	0.06	0.03	-0.04	-0.14	0.46	-0.01	-0.24	-0.42	-0.30	-0.32	-0.24	-0.25	0.02	-0.08	-0.10	-0.21
	(0.02)	(0.01)	(0.01)	(0.02)	(0.63)	(0.36)	(0.20)	(0.08)	(0.06)	(0.07)	(0.08)	(0.02)	(0.05)	(0.03)	(0.03)	(0.02)
All other causes	1.48	1.00	0.62	0.02	-0.51	-1.07	-0.25	-0.49	-0.27	-0.73	-0.21	-0.28	0.27	0.24	0.25	0.05
	(0.91)	(0.65)	(0.42)	(0.13)	(0.80)	(0.54)	(0.36)	(0.14)	(0.32)	(0.33)	(0.15)	(0.11)	(0.18)	(0.27)	(0.17)	(0.06)

Appendix Table C3: Regression estimates for death rates by cause of death, Males

Notes: Table presents regression estimates for cause of death among males. Point estimates correspond to results in Figure 5 in the text. Standard errors for each trend are reported in parentheses and clustered by age, race, and education.

	WNH					Bla	cks		j	Hisp	anics		Other			
	Q1	Q2	Q3	Q4												
Total	5.41	2.10	-1.69	-3.34	-2.65	-9.03	-8.42	-9.02	-1.66	-2.49	-1.83	-2.11	-1.11	-1.04	-1.69	-1.69
	(1.57)	(0.99)	(1.45)	(1.53)	(0.72)	(2.46)	(2.68)	(3.31)	(0.74)	(1.17)	(0.80)	(0.73)	(1.48)	(1.00)	(0.80)	(0.60)
Cardiovascular	-0.34	-0.32	-0.85	-0.80	-1.83	-3.58	-3.80	-3.36	-0.91	-0.87	-0.68	-0.57	-1.01	-0.75	-0.67	-0.48
	(0.73)	(0.38)	(0.53)	(0.41)	(0.66)	(1.38)	(1.57)	(1.44)	(0.44)	(0.46)	(0.39)	(0.26)	(0.63)	(0.47)	(0.37)	(0.22)
Cancer	-1.11	-1.84	-2.14	-2.18	-0.94	-2.89	-2.83	-2.94	-0.39	-0.94	-0.73	-0.73	-0.60	-0.63	-0.53	-0.64
	(0.58)	(0.77)	(0.96)	(0.92)	(0.29)	(0.96)	(0.95)	(1.15)	(0.16)	(0.44)	(0.29)	(0.28)	(0.20)	(0.25)	(0.26)	(0.23)
Drugs	2.74	1.67	0.74	0.17	1.11	0.63	0.42	0.08	0.31	0.32	0.22	0.06	0.47	0.33	0.11	0.02
	(0.30)	(0.14)	(0.07)	(0.03)	(0.16)	(0.17)	(0.11)	(0.04)	(0.05)	(0.04)	(0.03)	(0.01)	(0.06)	(0.03)	(0.03)	(0.01)
HIV	-0.07	-0.05	-0.02	-0.01	-1.64	-1.39	-0.59	-0.36	-0.27	-0.37	-0.14	-0.07	-0.05	-0.04	-0.02	-0.01
	(0.03)	(0.02)	(0.01)	(0.00)	(0.41)	(0.30)	(0.13)	(0.05)	(0.05)	(0.08)	(0.03)	(0.02)	(0.02)	(0.01)	(0.01)	(0.00)
Suicides	0.35	0.32	0.23	0.11	0.07	0.06	0.05	0.05	0.08	0.10	0.08	0.06	0.15	0.15	0.09	0.02
	(0.03)	(0.02)	(0.02)	(0.02)	(0.01)	(0.02)	(0.02)	(0.01)	(0.02)	(0.02)	(0.01)	(0.02)	(0.04)	(0.03)	(0.02)	(0.02)
Respiratory	0.97	0.56	-0.04	-0.21	0.64	0.11	0.00	-0.27	0.04	-0.09	-0.06	-0.04	0.03	0.00	-0.05	-0.01
	(0.33)	(0.19)	(0.15)	(0.15)	(0.35)	(0.17)	(0.10)	(0.12)	(0.04)	(0.05)	(0.04)	(0.02)	(0.07)	(0.05)	(0.03)	(0.01)
Stroke	-0.10	-0.12	-0.21	-0.17	-0.53	-0.88	-0.91	-0.71	-0.26	-0.19	-0.16	-0.14	-0.37	-0.31	-0.24	-0.18
	(0.09)	(0.05)	(0.08)	(0.08)	(0.14)	(0.26)	(0.32)	(0.29)	(0.09)	(0.06)	(0.06)	(0.06)	(0.18)	(0.16)	(0.13)	(0.08)
Liver	0.64	0.50	0.29	0.08	0.14	0.00	0.01	-0.08	0.08	0.08	0.13	0.01	0.32	0.26	0.02	-0.02
	(0.16)	(0.12)	(0.09)	(0.03)	(0.09)	(0.06)	(0.04)	(0.04)	(0.05)	(0.02)	(0.04)	(0.02)	(0.08)	(0.06)	(0.03)	(0.01)
Kidney	0.03	0.02	-0.02	-0.02	-0.15	-0.30	-0.23	-0.25	-0.04	-0.05	-0.01	-0.05	-0.13	-0.05	-0.06	-0.04
	(0.02)	(0.01)	(0.02)	(0.01)	(0.08)	(0.12)	(0.11)	(0.10)	(0.02)	(0.03)	(0.01)	(0.02)	(0.08)	(0.04)	(0.02)	(0.02)
Diabetes	-0.06	-0.01	-0.10	-0.09	-0.33	-0.52	-0.52	-0.49	-0.29	-0.22	-0.19	-0.19	-0.13	-0.05	-0.09	-0.07
	(0.13)	(0.05)	(0.05)	(0.04)	(0.32)	(0.31)	(0.27)	(0.25)	(0.17)	(0.12)	(0.10)	(0.08)	(0.19)	(0.09)	(0.06)	(0.04)
Nerve	0.41	0.20	0.07	0.00	0.36	0.11	0.11	-0.05	0.09	0.04	0.05	0.00	0.11	0.09	0.03	-0.01
	(0.09)	(0.06)	(0.05)	(0.02)	(0.07)	(0.04)	(0.06)	(0.03)	(0.02)	(0.02)	(0.02)	(0.03)	(0.03)	(0.03)	(0.03)	(0.01)
Accidents	0.19	0.09	-0.06	-0.16	0.08	-0.11	-0.18	-0.24	-0.17	-0.16	-0.20	-0.17	-0.12	-0.17	-0.22	-0.26
	(0.06)	(0.05)	(0.04)	(0.01)	(0.03)	(0.04)	(0.03)	(0.04)	(0.04)	(0.05)	(0.04)	(0.04)	(0.07)	(0.07)	(0.05)	(0.03)
Homicides	0.03	0.00	-0.01	-0.04	-0.10	-0.17	-0.11	-0.11	-0.05	-0.08	-0.08	-0.11	-0.04	-0.07	-0.08	-0.06
	(0.01)	(0.01)	(0.01)	(0.01)	(0.06)	(0.04)	(0.03)	(0.02)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.02)	(0.02)	(0.01)
All other causes	1.74	1.09	0.43	-0.02	0.48	-0.11	0.17	-0.29	0.12	-0.07	-0.05	-0.16	0.27	0.21	0.02	0.02
	(0.39)	(0.28)	(0.21)	(0.04)	(0.29)	(0.23)	(0.28)	(0.07)	(0.12)	(0.07)	(0.08)	(0.10)	(0.06)	(0.11)	(0.08)	(0.04)

Appendix Table C4: Regression estimates for death rates by cause of death, Females

Notes: Table presents regression estimates for cause of death among females. Point estimates correspond to results in Figure 6 in the text. Standard errors for each trend are reported in parentheses and clustered by age, race, and education.

		W	NH	<u> </u>		Bla	icks	8		Hisp	anics		Other			
	Q1	Q2	Q3	Q4	Q1	Q2	Q3	Q4	Q1	Q2	Q3	Q4	Q1	Q2	Q3	Q4
Total	0.009	0.008	0.007	-0.010	-0.012	-0.017	-0.012	-0.025	-0.009	-0.013	-0.004	-0.022	0.004	0.003	0.001	-0.015
	(0.003)	(0.004)	(0.004)	(0.003)	(0.002)	(0.003)	(0.002)	(0.002)	(0.003)	(0.005)	(0.004)	(0.002)	(0.003)	(0.002)	(0.002)	(0.002)
Cardiovascular	-0.004	-0.006	-0.006	-0.021	-0.014	-0.017	-0.010	-0.019	-0.007	-0.009	-0.006	-0.017	-0.001	0.000	-0.003	-0.015
	(0.002)	(0.002)	(0.003)	(0.002)	(0.004)	(0.003)	(0.004)	(0.003)	(0.004)	(0.005)	(0.004)	(0.007)	(0.005)	(0.003)	(0.002)	(0.004)
Cancer	-0.012	-0.015	-0.013	-0.023	-0.027	-0.028	-0.020	-0.024	-0.004	-0.009	-0.006	-0.021	-0.008	-0.012	-0.017	-0.016
	(0.002)	(0.002)	(0.002)	(0.002)	(0.004)	(0.005)	(0.004)	(0.004)	(0.005)	(0.007)	(0.004)	(0.003)	(0.003)	(0.003)	(0.004)	(0.002)
Drugs	0.098	0.098	0.087	0.064	0.064	0.051	0.052	0.015	0.047	0.042	0.055	0.044	0.086	0.090	0.083	0.078
	(0.013)	(0.012)	(0.011)	(0.008)	(0.011)	(0.010)	(0.011)	(0.010)	(0.009)	(0.008)	(0.009)	(0.009)	(0.007)	(0.008)	(0.011)	(0.012)
HIV	-0.064	-0.069	-0.072	-0.079	-0.087	-0.092	-0.081	-0.094	-0.093	-0.116	-0.084	-0.104	-0.037	-0.056	-0.046	-0.066
	(0.021)	(0.020)	(0.020)	(0.018)	(0.014)	(0.013)	(0.013)	(0.013)	(0.013)	(0.014)	(0.014)	(0.020)	(0.019)	(0.019)	(0.017)	(0.012)
Suicides	0.023	0.024	0.031	0.021	0.008	0.004	0.014	0.018	0.010	0.007	0.020	0.021	0.024	0.019	0.022	0.014
	(0.002)	(0.002)	(0.002)	(0.001)	(0.004)	(0.002)	(0.002)	(0.004)	(0.003)	(0.004)	(0.003)	(0.004)	(0.003)	(0.004)	(0.003)	(0.004)
Respiratory	0.012	0.014	0.004	-0.017	-0.007	-0.011	-0.008	-0.025	0.011	-0.007	0.019	-0.017	0.004	0.008	0.005	0.004
	(0.002)	(0.002)	(0.003)	(0.005)	(0.002)	(0.003)	(0.003)	(0.004)	(0.008)	(0.004)	(0.013)	(0.009)	(0.006)	(0.008)	(0.010)	(0.011)
Stroke	-0.001	-0.003	-0.004	-0.027	-0.027	-0.023	-0.015	0.003	-0.012	-0.010	-0.004	0.001	0.004	-0.008	0.009	-0.038
	(0.002)	(0.002)	(0.004)	(0.005)	(0.006)	(0.003)	(0.006)	(0.022)	(0.004)	(0.006)	(0.006)	(0.014)	(0.010)	(0.004)	(0.024)	(0.007)
Liver	0.029	0.032	0.038	0.032	-0.033	-0.030	-0.010	-0.016	0.006	0.004	0.027	0.025	0.028	0.034	0.034	0.045
	(0.010)	(0.011)	(0.010)	(0.011)	(0.011)	(0.008)	(0.013)	(0.014)	(0.011)	(0.014)	(0.014)	(0.018)	(0.011)	(0.013)	(0.014)	(0.021)
Kidney	0.008	0.008	0.008	-0.012	-0.020	-0.021	0.003	-0.040	0.019	0.009	0.017	0.000	0.000	0.005	0.026	-0.005
	(0.004)	(0.003)	(0.003)	(0.011)	(0.005)	(0.005)	(0.004)	(0.010)	(0.010)	(0.008)	(0.005)	(0.026)	(0.005)	(0.008)	(0.020)	(0.015)
Diabetes	0.014	0.011	0.009	-0.013	0.000	-0.003	0.003	-0.014	0.009	0.004	0.019	0.055	0.022	0.025	0.053	0.014
	(0.004)	(0.002)	(0.003)	(0.004)	(0.004)	(0.003)	(0.003)	(0.008)	(0.007)	(0.007)	(0.013)	(0.028)	(0.008)	(0.007)	(0.017)	(0.014)
Nerve	0.023	0.019	0.012	-0.002	0.018	0.011	0.014	0.002	0.024	0.020	0.012	0.010	0.035	0.041	0.033	0.010
	(0.002)	(0.001)	(0.005)	(0.004)	(0.003)	(0.003)	(0.003)	(0.008)	(0.006)	(0.005)	(0.008)	(0.031)	(0.003)	(0.008)	(0.006)	(0.025)
Accidents	0.002	0.000	0.000	-0.019	-0.004	-0.010	-0.004	-0.022	-0.013	-0.015	-0.009	-0.032	0.000	-0.005	-0.010	-0.027
	(0.005)	(0.004)	(0.005)	(0.004)	(0.003)	(0.003)	(0.002)	(0.003)	(0.003)	(0.003)	(0.003)	(0.005)	(0.003)	(0.003)	(0.004)	(0.004)
Homicides	0.007	0.006	-0.007	-0.049	0.013	0.004	-0.006	-0.045	-0.019	-0.023	-0.031	-0.076	0.001	-0.004	-0.020	-0.083
	(0.002)	(0.002)	(0.003)	(0.005)	(0.006)	(0.005)	(0.004)	(0.008)	(0.003)	(0.003)	(0.007)	(0.006)	(0.005)	(0.004)	(0.006)	(0.007)
All other	0.010	0.009	0.007	-0.006	-0.013	-0.020	-0.010	-0.022	-0.012	-0.023	-0.012	-0.023	0.008	0.005	0.008	0.005
	(0.007)	(0.006)	(0.006)	(0.005)	(0.007)	(0.005)	(0.005)	(0.004)	(0.007)	(0.006)	(0.006)	(0.006)	(0.005)	(0.006)	(0.008)	(0.008)

Appendix Table C5: Regression estimates for log death rates by cause of death, Males

Notes: Table presents regression estimates for cause of death among males. Point estimates correspond to results in Figure 5 in the text. Standard errors for each trend are reported in parentheses and clustered by age, race, and education.

		W	NH	<u></u>		Bla	icks			Hisp	anics		Other			
	Q1	Q2	Q3	Q4												
Total	0.019	0.012	0.000	-0.016	-0.007	-0.018	-0.018	-0.027	-0.004	-0.007	-0.007	-0.018	0.004	0.002	-0.011	-0.019
	(0.005)	(0.004)	(0.003)	(0.002)	(0.002)	(0.002)	(0.002)	(0.003)	(0.004)	(0.004)	(0.002)	(0.003)	(0.005)	(0.004)	(0.001)	(0.002)
Cardiovascular	0.007	0.001	-0.010	-0.025	-0.010	-0.022	-0.024	-0.035	-0.012	-0.013	-0.010	-0.022	-0.003	-0.004	-0.020	-0.032
	(0.004)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.003)	(0.004)	(0.004)	(0.004)	(0.004)	(0.006)	(0.007)	(0.005)	(0.003)	(0.002)
Cancer	-0.007	-0.013	-0.016	-0.023	-0.012	-0.022	-0.021	-0.024	-0.002	-0.009	-0.009	-0.010	-0.006	-0.005	-0.007	-0.015
	(0.001)	(0.001)	(0.002)	(0.001)	(0.003)	(0.002)	(0.003)	(0.005)	(0.003)	(0.002)	(0.001)	(0.004)	(0.002)	(0.002)	(0.001)	(0.002)
Drugs	0.102	0.097	0.072	0.045	0.077	0.058	0.060	0.033	0.064	0.066	0.063	0.056	0.091	0.070	0.042	0.026
	(0.007)	(0.008)	(0.008)	(0.006)	(0.016)	(0.015)	(0.013)	(0.013)	(0.012)	(0.013)	(0.011)	(0.014)	(0.010)	(0.010)	(0.009)	(0.011)
HIV	-0.036	-0.053	-0.066	-0.088	-0.070	-0.097	-0.090	-0.130	-0.107	-0.141	-0.114	-0.119	-0.030	-0.047	-0.038	-0.095
	(0.020)	(0.023)	(0.020)	(0.019)	(0.019)	(0.017)	(0.018)	(0.022)	(0.014)	(0.015)	(0.015)	(0.030)	(0.018)	(0.016)	(0.035)	(0.017)
Suicides	0.044	0.044	0.037	0.024	0.035	0.026	0.030	0.031	0.047	0.042	0.042	0.037	0.037	0.035	0.031	0.012
	(0.001)	(0.002)	(0.002)	(0.003)	(0.005)	(0.008)	(0.012)	(0.005)	(0.008)	(0.005)	(0.005)	(0.009)	(0.006)	(0.007)	(0.007)	(0.006)
Respiratory	0.023	0.018	-0.001	-0.022	0.009	-0.005	-0.007	-0.017	0.006	-0.004	0.002	-0.021	0.015	0.006	-0.010	-0.005
	(0.004)	(0.003)	(0.005)	(0.004)	(0.005)	(0.004)	(0.005)	(0.007)	(0.005)	(0.003)	(0.005)	(0.007)	(0.008)	(0.005)	(0.006)	(0.005)
Stroke	-0.003	-0.012	-0.023	-0.032	-0.022	-0.036	-0.039	-0.045	-0.019	-0.022	-0.012	-0.025	-0.012	-0.017	-0.022	-0.031
	(0.002)	(0.002)	(0.003)	(0.002)	(0.003)	(0.003)	(0.003)	(0.006)	(0.006)	(0.002)	(0.007)	(0.010)	(0.010)	(0.011)	(0.007)	(0.008)
Liver	0.053	0.054	0.052	0.042	0.013	0.005	0.011	0.006	0.023	0.031	0.042	0.041	0.039	0.041	0.018	0.009
	(0.009)	(0.010)	(0.014)	(0.016)	(0.007)	(0.007)	(0.010)	(0.014)	(0.013)	(0.010)	(0.010)	(0.015)	(0.013)	(0.010)	(0.012)	(0.015)
Kidney	0.006	0.000	-0.013	-0.025	-0.007	-0.025	-0.019	-0.049	-0.001	-0.022	-0.008	-0.048	-0.011	0.008	-0.028	-0.009
	(0.005)	(0.006)	(0.009)	(0.006)	(0.002)	(0.004)	(0.007)	(0.005)	(0.004)	(0.009)	(0.010)	(0.014)	(0.009)	(0.016)	(0.009)	(0.013)
Diabetes	0.010	0.005	-0.012	-0.036	0.010	-0.004	-0.013	-0.020	0.001	-0.011	-0.012	-0.041	0.022	0.016	-0.009	-0.023
	(0.007)	(0.004)	(0.003)	(0.008)	(0.008)	(0.006)	(0.005)	(0.008)	(0.011)	(0.006)	(0.005)	(0.011)	(0.015)	(0.008)	(0.011)	(0.008)
Nerve	0.029	0.017	0.002	-0.008	0.026	0.007	0.002	-0.015	0.023	0.010	0.002	0.018	0.030	0.035	0.007	-0.005
	(0.002)	(0.002)	(0.003)	(0.006)	(0.002)	(0.003)	(0.003)	(0.005)	(0.004)	(0.002)	(0.007)	(0.020)	(0.009)	(0.009)	(0.007)	(0.013)
Accidents	0.009	0.005	-0.008	-0.024	0.004	-0.007	-0.014	-0.029	-0.017	-0.015	-0.024	-0.033	-0.007	-0.013	-0.029	-0.058
	(0.003)	(0.003)	(0.004)	(0.004)	(0.002)	(0.003)	(0.002)	(0.003)	(0.004)	(0.004)	(0.004)	(0.006)	(0.004)	(0.005)	(0.006)	(0.007)
Homicides	0.009	0.002	-0.006	-0.028	-0.004	-0.020	-0.019	-0.025	-0.015	-0.027	-0.037	-0.061	-0.013	-0.029	-0.041	-0.039
	(0.002)	(0.003)	(0.005)	(0.008)	(0.006)	(0.005)	(0.006)	(0.009)	(0.005)	(0.002)	(0.005)	(0.015)	(0.005)	(0.004)	(0.011)	(0.013)
All other	0.029	0.024	0.008	-0.005	0.006	-0.006	-0.003	-0.015	0.002	-0.003	-0.006	-0.018	0.014	0.010	-0.002	0.002
	(0.002)	(0.003)	(0.004)	(0.003)	(0.003)	(0.004)	(0.005)	(0.005)	(0.005)	(0.004)	(0.003)	(0.010)	(0.003)	(0.005)	(0.007)	(0.010)

Appendix Table C6: Regression estimates for log death rates by cause of death, Females

Notes: Table presents regression estimates for cause of death among females. Point estimates correspond to results in Figure 5 in the text. Standard errors for each trend are reported in parentheses and clustered by age, race, and education.



Appendix Figure C1: Trends in Total Mortality Rates: 25-64 Year Olds by Race

Note: Figure shows race specific annual mortality rates measured across all education quartiles. Mortality rates are agadjusted to 2018. Panel (a) presents results for males and Panel (b) presents results for females.



Figure C2: Log Death Rates: Regression Estimates by Major Causes of Death for 25-64 Year Olds, By Education Quartile

Note: Figure shows regression estimates of the interactions between race, trend, and quartile from equation (3) for major causes of death in logs. Results for males are presented in Panel (a) and females in Panel (b). Standard errors are presented in Appendix Table C4. The estimate for Q4 is the interaction of trend and race. The estimates for Q1, Q2, and Q3 are calculated as the main effect plus the education quartile-specific trend coefficient. Regression is weighted by population of each age-race-year cell to obtain nationally-representative estimates.



Figure C3: Log Death Rates: Regression Estimates by Minor Causes of Death for 25-64 Year Olds, By Education Quartile

Note: Figure shows regression estimates of the interactions between race, trend, and quartile from equation (3) for minor causes of death in logs. Results for males are presented in Panel (a) and females in Panel (b). Standard errors are presented in Appendix Table C4. The estimate for Q4 is the interaction of trend and race. The estimates for Q1, Q2, and Q3 are calculated as the main effect plus the education quartile-specific trend coefficient. Regression is weighted by population of each age-race-year cell to obtain nationally-representative estimates.



Appendix Figure C4: Share of ACS Respondents with 12-years of Education who have a GED, 2008-2018

Note: Figure plots the percentage of ACS respondents who report 12 years of education who hold a GED by race between 2008 and 2018. GED attainment was not separately recorded in the ACS prior to 2008. Ages are restricted to 25 to 64 to match the analysis sample. Means are computed using person-level survey weights.