Mortality Change Among Less Educated Americans*

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April 2020

Abstract

Changing mortality rates among less educated Americans are difficult to interpret because the least educated groups (*e.g.* dropouts) become smaller and more negatively selected over time. We derive partial identification methods that let us calculate mortality changes at constant education percentiles from 1992–2015. We find that middle-age mortality increases among non-Hispanic whites are driven almost entirely by changes in the bottom 10% of the education distribution. Drivers of mortality change differ substantially across groups. Deaths of despair explain a large share of mortality change among young non-Hispanic whites, but a small share among older whites and almost none among non-Hispanic blacks.

*We are grateful for feedback on earlier versions of this paper from Alberto Abadie, Patty Anderson, Leila Agha, John Beshears, Emily Blanchard, Raj Chetty, James Choi, Eric Edmonds, Shahe Emran, Jim Feyrer, Francisco Ferreira, Amy Finkelstein, Nate Hilger, David Laibson, JoAnna Leyenaar, Ethan Ligon, Erzo Luttmer, Ellen Meara, Nina Pavnik, Bruce Sacerdote, Na'ama Shenhav, Forhad Shilpi, Jon Skinner, Chris Snyder, Gary Solon, Bob Staiger, Doug Staiger, Michael Stepner, and Elie Tamer. Toby Lunt, Ryu Matsuura, and Taewan Roh provided excellent research assistance. All errors are our own. The authors have no competing interests. This material includes work supported by the National Science Foundation Graduate Research Fellowship under Grant No. 1122374. This manuscript includes material that previously circulated in a working paper titled "Getting Signal from Interval Data: Theory and Applications to Mortality and Intergenerational Mobility."

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1 Introduction

Mortality rates among non-Hispanic whites without college degrees have increased substantially over the last twenty years (Meara et al., 2008; Cutler and Lleras-Muney, 2010; Cutler et al., 2011; Olshansky et al., 2012; Case and Deaton, 2015; Case and Deaton, 2017). While widely publicized, this fact by itself is difficult to interpret because overall education levels have risen concurrently; the share of 50–54-year-olds with a college degree, for example, was 59% in 1992 and 42% in 2015. The average person without a college degree occupies a lower position in both the educational and the socioeconomic distribution today than in the past. It is therefore not necessarily surprising that people at a fixed low level of education are less healthy today compared with those at the same level in earlier decades. If education levels are rising, it is theoretically possible for the mortality rate to stay the same at every percentile in the education distribution, but to be higher at every education level.[1]

There are three possible interpretations of rising mortality among non-Hispanic whites without college degrees. Each has a substantially different policy implication. First, this result could be nothing more than an artifact of shifts in the education distribution, with no changes in the underlying relationship between education percentile and mortality. Second, mortality could be rising uniformly among individuals in the bottom half of the education distribution. Third, mortality could be rising substantially at the very bottom of the education distribution, with fewer changes or even improvements in the percentiles reflecting high school graduates. We will use a partial identification methodology to show that the third interpretation is the one most supported by the evidence.

This selection bias in estimates of mortality change at fixed education levels has been a major barrier to the study of disparities in death rates, not least because education is one of the only measures of socioeconomic status that is recorded in Vital Statistics data. Some researchers have argued that the bias is so large that estimates of mortality change by education level are effectively meaningless (Dowd and Hamoudi, 2014; Bound et al., 2015; Currie, 2018). Other researchers have limited analysis to population subsets where education has not substantially changed (Case and

[1] A similar phenomenon is described by the well-known college swipe, "If the worst student at this college went to (inferior college) Y, it would raise the average intelligence of both schools."

Deaton, 2015; Case and Deaton, 2017) or else argued that the selection bias does not affect estimates enough to be a concern (Meara et al., 2008).

In principle, the selection bias can be addressed by studying mortality in fixed percentile ranges of the education distribution, for example, in the bottom 10%. This would hold constant the size and relative rank of each education bin over time. While calculating mortality in fixed education percentiles has been suggested before (Bound et al., 2015), doing so is not trivial, because education levels are inherently lumpy, especially as reported in standard mortality datasets. For example, if education is bottom-coded at the 20th percentile (as in 1992, where 20% of the population are high school dropouts), the mortality rate at the 10th education percentile cannot be point estimated without strong assumptions.

In this paper, we derive new methods in partial identification to *bound* mortality at fixed education percentiles (Manski and Tamer, 2002). We show that tight and meaningful bounds on a conditional expectation function can be identified under a minimal set of structural assumptions. First, we assume that there exists a latent education rank, which is only coarsely observed in the education data; this assumption follows directly from a standard human capital model and is widespread in the literature. Second, we assume that the mortality rate is weakly declining in the latent education rank; this assumption is supported by theory and by empirical evidence. Third, we assume that the mortality–education rank function is allowed (but not required) to have kinks or discrete jumps only at major education intervals, such as high school completion. The first two assumptions are sufficient to clearly distinguish between the three scenarios above and to generate the substantive conclusions of the paper. The third allows mortality estimates to be bounded more tightly.

We then use these methods to document changes in mortality from 1992–2015 among the U.S. population aged 25–69, in constant education percentile bins. We focus in particular on two domains where researchers have noted deteriorating outcomes: (i) mortality change in the bottom half of the education distribution; and (ii) changes in deaths from poisoning, suicide and chronic liver disease, described by earlier researchers as "deaths of despair" (Case and Deaton, 2015; Case and Deaton, 2017).

We have three primary findings. First, among middle-aged non-Hispanic white (hereafter referred to as white) men and women, the group most widely discussed in the recent literature, mortality

increases are driven almost entirely by the bottom 10% of the education distribution (the part of the distribution represented by high school dropouts in 2015). From 1992 to 2015, age-adjusted mortality for whites in the least educated 10% has risen by 59–90% for women and 32–49% for men (respectively 2.2–3.1% and 1.3–1.9% per year). Mortality change in percentiles 10–45 (approximately high school completers in 2015) is considerably less dramatic, and is rising unambiguously only for white women below the age of 50 and white men under 35. The mortality increases described by Case and Deaton (2015, 2017) are thus both more severe and more focused in a narrow population subgroup than has previously been recognized.

Second, non-Hispanic blacks have experienced large improvements in mortality in all education groups *except* for the least educated 10%. Blacks in the least educated 10% have close to zero mortality change from 1992–2015; age-adjusted middle-age mortality for blacks is marginally rising for women and marginally declining for men. This has led to a substantial convergence between black and white outcomes at the bottom of the education distribution. Conditional upon being in the least educated 10% of the national distribution, white men over the age of 50 now have higher mortality than similarly-aged black men. The same holds for white women relative to black women over the age of 40.

Finally, a single proximate cause cannot expain these divergent death rates. The change in deaths from despair, which has been widely discussed in prior research and in the media, accounts for a large share of mortality increases for young whites, but a very small share of rising mortality among older whites and none of the divergent mortality rates of blacks. Further, deaths of despair have increased more uniformly across the education distribution than deaths from other causes. The least educated middle-aged white women, in particular, are now at higher risk of dying from cancer, heart diseases and respiratory diseases, among other causes, even as mortality from these causes has declined sharply for those outside of the bottom 10%. Note that earlier unadjusted estimates were particularly difficult to interpret for women, for whom education has risen considerably more than among men, creating a larger selection bias.

A long prior literature relies upon education as a marker of socioeconomic status to study mortality change, both because of its wide availability in the data, and because it is a marker of permanent

rather than transitory socioeconomic status. Olshansky et al. (2012) noted rising mortality rates among high school dropouts from 1990 to 2008, but this work was widely criticized as it did not adjust for the substantial increase in the negative selection associated with being a dropout over the sample period. Case and Deaton (2015, 2017) justified ignoring the selection bias in mortality change by focusing on population subgroups for whom education levels had not changed substantially; however, they did not look specifically at outcomes among high school dropouts exactly because of the selection bias addressed in our paper. Meara et al. (2008) and Cutler et al. (2011) similarly avoid looking at dropouts due to potential selection bias. These latter papers, along with Bound et al. (2015), propose an adjustment for selection bias that is implicitly based on stricter (and in our view, less plausible) assumptions; we relate their approach to ours in Section 3. Among these papers, only Olshansky et al. (2012) looked specifically at high school dropouts—the group among whom mortality has changed the most. Our finding of dramatically rising mortality in the bottom 10% supports the findings of Olshansky et al. (2012): the mortality increases at the bottom of the education distribution prove to be large, even after removing substantial selection bias.

Several other recent papers document the relationship between socioeconomic status and mortality. Currie and Schwandt (2016a, 2016b) study differences in mortality across counties, finding, like us, that changes in mortality inequality are highly heterogeneous across age, race and place. They show that mortality inequality across space is falling between blacks and whites and among younger individuals (especially children), but rising among older adults. They also document dramatic declines in mortality among black men. Our findings confirm this result among black men in the most educated 90%. But we find that middle-aged black men in the least educated 10% have experienced only marginal improvements, with the mortality rate falling by 11–15% over 23 years, or 0.5–0.7% per year.

Chetty et al. (2016) use deaths as reported in tax records to describe changes in mortality throughout the income distribution. While the study of mortality using tax records is an important innovation, vital statistics are likely to remain valuable as sources of information on mortality because they record cause of death in detail. Our work makes it possible to use education as a marker of socioeconomic status in the vital statistics data, which is important given that so few other predictors

of socioeconomic status are recorded. In a related approach, Goldring et al. (2016) derive a one-tailed statistical test to examine whether the mortality gradient in education is changing over time. Like us, they assume that: (i) there exists a latent education rank distribution; and (ii) mortality is monotonically declining in the latent education rank. They conclude that the education gradient is getting steeper (as do we), but their approach does not generate estimates of mortality change.

We have posted both unadjusted and constant-percentile mortality estimates for all ages and groups with the manuscript, which we hope will be useful for other researchers interested in studying U.S. mortality change. Code to calculate bounds on mortality in constant percentile groups given raw education data is also posted online.[2]

2 Data Sources

Death records from 1992–2015 were obtained from the US National Vital Statistics System of the National Center for Health Statistics (National Center for Health Statistics, 2018). Mortality rates (deaths per 100,000 people) were obtained by dividing the number of deaths in each age, race, gender and education cell by the population total from the Current Population Survey (CPS). Ages were coded in 5-year bins to mitigate bias from changing age within bins over time (Gelman and Auerbach, 2016; Case and Deaton, 2017). Education could be consistently matched across datasets in four groups: (i) less than a high school degree, (ii) high school degree/GED, (iii) some college, and (iv) a bachelor's degree or more. Annual estimates were pooled into three-year bins. Following earlier work, estimates are presented separately for men and women, and for non-Hispanic blacks and whites. Results are not shown for Hispanics, because their higher in- and out-migration over the sample period make mortality change among Hispanics more difficult to interpret (Markides and Eschbach, 2005). Mortality rates closely match those in other recent studies (Case and Deaton, 2015; Case and Deaton, 2017).

Causes of death were partitioned into the following subgroups: cancers, heart diseases, deaths of despair, injuries, and other diseases. Deaths of despair are deaths from poisoning, suicide, and alcoholic liver diseases and cirrhosis (Kochanek et al., 2016; Case and Deaton, 2017); we exclude

[2] We provide Stata and Matlab code for the bounding algorithm on Github at https://github.com/devdatalab/paper-nra-mortality/.

suicides from injuries. More detail on the distribution of deaths is reported in Table A1. Appendix B provides additional details about data construction.

3 Methods: Bounding Mortality in Constant Education Percentile Bins

3.1 Intuition and Examples

When the level of education in the population rises, individuals at each level of education mechanically occupy a lower set of ranks in the education distribution. For example, among 50–54-year-old women, dropouts were the bottom 20% in 1992 and the bottom 10% in 2015. If socioeconomic status is a driver of education, then dropouts will have lower relative socioeconomic status in 2015 than in 1992. If low socioeconomic status is also a cause of mortality through channels other than low education, then dropouts could have higher measured mortality even if mortality is unchanged at every education percentile. It is possible for measured mortality to rise at each *level* of education even if the combined mortality rate is unchanged. This statistical paradox is known as the Will Rogers Phenomenon or "stage migration" in the medical literature (Feinstein et al., 1985).

The problem can be resolved by measuring mortality at education percentiles instead of at education levels (Bound et al., 2015)—this holds the analysis sample to a group with constant size and relative rank. But this is difficult because education is observed in coarse categories that cover many percentiles. How does one calculate mortality among the least educated 10%, if the bottom 15% are bottom-coded as high school dropouts?

We resolve this issue by using recent methods from the literature on partial identification and interval-censoring. We begin by providing an intuitive explanation of how this bounding process works, before formally describing the results in the following subsection. Manski and Tamer (2002) show that a conditional expectation function (in this case, the CEF of mortality given education rank) with an interval-censored dependent variable can be *bounded* under a minimal set of structural assumptions. In this setting, the Manski and Tamer (2002) bounds are not tight enough to deliver any meaningful estimates of mortality change. We extend this method by considering three assumptions about the mortality and education distributions: (1) there exists a continuous latent education

rank distribution, which is partitioned into discrete intervals by the observed education levels; (2) expected mortality is weakly monotonically declining in the latent education rank; and (3) the conditional expectation function of mortality given education rank has no kinks or discrete jumps, except (potentially) at boundaries between major education intervals.[3]

The first assumption comes out of a standard human capital investment model where schooling costs are convex and individual educational attainment is determined by individual-specific constant cost and benefit shifters (Card, 1999). A person who is highly ranked within their bin (for instance, the highest-ranked high school dropout) is a person who would have attained a higher level of education if the cost was only marginally lower or the benefit to them only marginally higher. Consider an example where the only cost/benefit shifter is the discount rate. It is possible for two individuals (A and B) to obtain the same level of education even if A has a lower discount rate, because years of education are lumpy. However, A may be right at the margin of attaining a higher level of education and B may be right at the margin of attaining a lower level of education. If the discount rate also affects health-seeking behavior, then we would expect A to have lower mortality risk than B, even though their levels of education are the same.[4]

The second assumption—that mortality rates are weakly decreasing in education percentile—is standard in the literature on mortality-education gradients, and is supported by empirical evidence from both the United States and Europe (Pappas et al., 1993; Mackenbach et al., 2003; Meara et al., 2008; Cutler and Lleras-Muney, 2010; Goldring et al., 2016). For Assumption 3, the curvature of the mortality–education rank function is constrained to be less than twice the curvature of the mortality-income-rank function estimated in the United States (Chetty et al., 2016). Results are robust to separately loosening either Assumption 2 or Assumption 3 (Appendix E).

[3]Such kinks or jumps could occur if there are large sheepskin effects (Hungerford and Solon, 1987). For instance, we allow (but do not require) mortality to decline substantially and discretely at the marginal rank change corresponding to high school completion.

[4]Note that we are *not* making causal claims about the relationship between education rank and mortality. Rather, like the prior literature, we use education as a proxy for socioeconomic status that is readily available in mortality data. Our exercise is analogous to measuring mortality at a given income percentile, which is understood to be a meaningful measure even though the income level at that percentile may change over time. If the education *level* has a causal effect on health, then we might expect survival to improve at education ranks which reflect higher levels of education in 2015 than in 1992; our framework allows for this possibility.

An example may provide some intuition. Suppose that Assumptions 1 and 2 hold, and we know only that the mortality rate is 1000 deaths per 100,000 among the bottom 15%, and 700 deaths per 100,000 among percentiles 15–50. Let Y, the parameter of interest, be the mortality rate among the bottom 10%. Under monotonicity, Y cannot be less than 1000, as this would require a higher mortality in percentiles 10–15 than in 0–10, violating monotonicity. Monotonicity with respect to the 15–50 bin also ensures that the mortality CEF cannot be less than 700 at any percentile in the bottom 15%. If the mean value from percentiles 0–15 must be above 700, then the mean value from percentiles 0–10 cannot be over 1150, since the weighted mean of mortality in bins 0–10 and 10–15 must equal 1000.[5] We can thus bound Y in the interval [1000,1150]. Note that if Y is 1150, then this implies that the mortality function must discretely jump from 1150 to 700 right at percentile 10. Assumption 3 would rule out such a jump, further lowering the upper bound on Y.

Under Assumptions 1 and 2, we can compute bounds arithmetically following this logical process; the analytical expression for bounds in an arbitrary percentile range is given in Appendix D. To calculate bounds under a curvature constraint (Assumption 3), we use a constrained optimization solver in Matlab, described in detail in Appendix D. We calculate education percentiles in own-gender groups, following earlier work on mortality and income (Chetty et al., 2016).

Our primary results are calculated numerically using the constrained optimization routine and use all three assumptions. For a small number of population subgroups, we do not observe monotonicity in the data. In the majority of these cases, the mortality rate in the higher education group is within 5% of that in the lower education group, so the monotonicity violation is not substantive in comparison with the width of the bounds. However, for black cohorts over the age of 55 in 1992–1994, there are more substantial violations: high school graduates often have lower mortality than dropouts, and B.A. recipients often have higher mortality than individuals with some college. Such non-monotonic means do not appear for any white cohorts, nor is mortality non-monotonic in education for any groups of either race in 2013–2015. Because our constrained optimization imposes monotonic CEFs on these groups, we may *overstate* health improvements for the oldest

[5]The weights on each percentile are uniform, because the rank distribution is uniform by construction.

black age groups, because monotonicity makes mortality among dropouts look *worse* for these cohorts in 1992–1994. Given that our primary finding is divergence by education group, allowing for non-monotonic mortality among older black cohorts would only strengthen our results. Finally, we show in Appendix D that all our results are robust to optimizations that do not assume monotonicity, as well as to optimizations that do not assume that curvature is constrained.

An important characteristic of these bounds is that they are tightest for percentile intervals that are closest to those in the data. Consider a trivial example: mortality can be point estimated in the rank intervals that precisely match the data. Bins that are marginal departures from these intervals must necessarily have similar means. We select percentile bins for analysis by matching the education levels of 50–54-year-olds in 2013–2015, who are approximately the median cohort in the study, and a group focused on by the earlier literature. We thus calculate mortality for the following four education groups: (i) the bottom 10% (the share of the age 50–54 population who were high school dropouts in 2013–2015); (ii) percentiles 10 to 45 (those with high school degrees only in 2013–2015); (iii) percentiles 45 to 70 (2-year college degrees in 2013–2015); and (iv) the top 30% (Bachelor's degrees or higher in 2013–2015). Mortality estimates in education quartiles or deciles would be useful, but given the existing rank bin boundaries in the data, they cannot be bounded as tightly and are thus less informative. We therefore do not present these, but they can be readily calculated from the shared code and data. Because the data include the universe of deaths and statistical uncertainty regarding the population totals is very small, we follow the previous literature in omitting confidence intervals.

When faced with selection bias in this context, earlier researchers have used one of two methods. One set of researchers has reassigned individuals across bins at random to obtain constant percentile mortality estimates (Meara et al., 2008; Hendi, 2015; Bound et al., 2015). This is analogous to assuming that the CEF of mortality given education rank is a step function with constant mortality in each bin. In the example above, for instance, this would entail assuming that the mortality rate from percentiles 0–10 and from percentiles 10–15 is equal to 1000, the rate observed for the entire bin. This implicit assumption would also imply that mortality falls sharply between education percentiles 14.9 and 15.1. This function is unlikely to be a plausible description of reality for two reasons. First, this

function implies huge sheepskin effects in education, because it suggests that the individual who just barely completed high school (percentile 10.5 for 50–54-year-old women in 2013–2015) has considerably lower mortality than the individual who was right at the margin of completing high school but then dropped out (percentile 10.4). Second, it implies that the high school completer right at the margin of dropping out (percentile 10.5) has the same expected mortality as the high school completer right at the margin of going on to college (percentile 45). A standard human capital model would reject this function: the individuals at the margin of completing college would have had higher socioeconomic status than those at the margin of dropping out, and thus lower mortality risk. Note that this function is nevertheless contained within our bounds when we use only Assumptions 1 and 2 (shown in Appendix E), but is rejected by a curvature constraint of any magnitude. Section D.2 compares our method and results with those of Meara et al. (2008) and Bound et al. (2015) in more detail.

Other researchers have avoided the problem of selection bias by focusing on cohorts, subgroups or sample periods for whom education levels have not changed very much (e.g. Case and Deaton (2015)). While this strategy is reasonable, it is not useful for examining subgroups for whom education levels have changed substantially.

3.2 Formal Description of Bounds

In this subsection, we provide the formal framework for constructing bounds on mortality changes over time. We state our novel bounds on the CEF and relegate additional propositions and proofs to Appendix C.

Let the function $Y(x) = E(y|x)$ be defined on a known interval; without loss of generality, define this interval as $x \in [0,100]$. Assume $Y(x)$ is integrable. We want to bound $E(y|x)$ when x is known to lie in the interval $[x_k, x_{k+1}]$; there are K such intervals. Define the expected value of y in bin k as

$$r_k = \int_{x_k}^{x_{k+1}} Y(x) f_k(x) dx.$$

Note that

$$r_k = E(y|x \in [x_k, x_{k+1}])$$

. Define $r_0 = 0$ and $r_{K+1} = 100$.

Restate the following assumptions from Manski and Tamer (2002):

$$P(x \in [x_k, x_{k+1}]) = 1. \qquad \text{(Assumption I)}$$

$$E(y|x) \text{ must be weakly increasing in } x. \qquad \text{(Assumption M)}$$

$$E(y|x \text{ is interval censored}) = E(y|x). \qquad \text{(Assumption MI)}$$

Assumptions I and MI are regularity conditions about interval censoring. Assumption I yields that, if x is interval censored, it truly lies within its given bin, and assumption MI gives that the fact of interval censoring yields no additional information about x. We state assumption M in terms of being "weakly increasing" as is common in the literature; in the mortality setting, assumption M entails that survival rates are increasing in education rank, but in practice, we simply reverse the signs on the proposition below for the case where $E(y|x)$ is weakly decreasing in x.

From Manski and Tamer (2002), we have:

$$r_{k-1} \leq E(y|x) \leq r_{k+1} \qquad \text{(Manski-Tamer bounds)}$$

Suppose also that

$$x \sim U(0,100). \qquad \text{(Assumption U)}$$

This condition immediately holds in the case where we wish to condition on education ranks, which are uniform by construction.

In that case,

$$r_k = \frac{1}{x_{k+1} - x_k} \int_{x_k}^{x_{k+1}} Y(x) dx,$$

substituting the probability distribution function for the uniform distribution within bin k. Then we derive the following proposition.

Proposition 1. *Let x be in bin k. Under assumptions M, I, MI (Manski and Tamer, 2002) and U, and without additional information, the following bounds on $E(y|x)$ are sharp:*

$$\begin{cases} r_{k-1} \leq E(y|x) \leq \frac{1}{x_{k+1}-x}((x_{k+1}-x_k)r_k-(x-x_k)r_{k-1}), & x<x_k^* \\ \frac{1}{x-x_k}((x_{k+1}-x_k)r_k-(x_{k+1}-x)r_{k+1}) \leq E(y|x) \leq r_{k+1}, & x \geq x_k^* \end{cases}$$

where

$$x_k^* = \frac{x_{k+1}r_{k+1}-(x_{k+1}-x_k)r_k-x_kr_{k-1}}{r_{k+1}-r_{k-1}}.$$

We provide proofs in Appendix C. The intuition behind the proof is as follows. First, find the function z which meets the bin mean and is defined as r_{k-1} up to some point j. Because z is a valid CEF, the lower bound on $E(y|x)$ is no larger than z up to j; we then show that j is precisely x_k^* from the statement. For points $x > x_k^*$, we show that the CEF which minimizes the value at point x must be a horizontal line up to x and a horizontal line at r_{k+1} for points larger than x. But there is only one such CEF, given that the CEF must also meet the bin mean, and we can solve analytically for the minimum value the CEF can attain at point x. We focus on lower bounds for brevity, but the proof for upper bounds follows a symmetric structure.

In Appendix C, we generalize this proposition to settings where Assumption U does not hold, and we also provide analytical bounds on a particular function of the CEF — the average value of the CEF, integrating from $x=a$ to $x=b$, which we define as μ_a^b. These are the analytical bounds on mortality changes on, e.g., the bottom 10%, that we employ in our results.

4 Results

We begin by demonstrating the method for a single age group (50–54), comparing estimates of mortality changes at education levels with mortality changes at constant education percentiles. We then show mortality change at constant education percentiles for all ages.

4.1 Unadjusted Mortality Changes by Education Levels, Ages 50–54

Figure 1 presents the raw data, showing total mortality (deaths / 100,000) for people aged 50–54 from 1992–1994 to 2013–2015, separately for each education level and by race and gender. The four groups of points on each graph represent individuals with (i) less than high school education; (ii) high school education; (iii) some college; and (iv) a Bachelor's degree or higher. The mean education percentile for individuals in a given education category is plotted on the X axis. In 1992–94, 19.6% of white women had less than a high school education. The average percentile rank for someone in this group is 19.6 / 2 = 9.8; mortality for this group is therefore plotted (with a black triangle in Panel A) at 9.8 on the X axis. In 2013–15, 8.5% of women had less than a high school education; their mean education percentile was 4.25 (yellow square). Intermediate points show the transition path between these years.

Among white women (Panel A), 50–54-year-old high school dropouts had mortality rates 124% higher in 2013–2015 than in 1992–94, suggesting an annualized mortality increase of 3.9% per year. Unadjusted mortality rose 33% for 50–54-year-old high-school-educated white women, rose 10% for women with some college, and fell by 34% for white women with Bachelor degrees or higher. Panels B through D present unadjusted estimates for white men, black women, and black men.

The points systematically shift to the left over time, because education for all race and gender groups rose steadily over the sample period. The decreasing average rank over time implies that unadjusted mortality changes at given education levels are biased upward by selection (Dowd and Hamoudi, 2014; Bound et al., 2015; Currie, 2018). The next section adjusts these estimates for changes in the size and relative rank of each group by studying constant percentile education groups rather than constant levels of education.

4.2 Mortality Changes in Constant Education Percentile Bins, Ages 50–54

Figure 2 presents bounds on the mortality of people aged 50–54 from 1992–1994 to 2013–2015, in four constant education percentile groups corresponding to education percentile bins in 2013–2015. The top left panel shows mortality rates for white women, with one series for each education percentile group. Mortality among the least educated 10% rose steadily from between 625 and 735 deaths per 100,000

in 1992–1994 to over 1300 in 2013–2015, an increase of 79–116%, or about 3% per year. As expected, this is a smaller increase than the unadjusted mortality change shown in Figure 1; the original point estimate for dropouts is outside the bounds for the constant rank group, and the bias in the naive estimate could be as large as 57%. However, even the lower bound on mortality change (+79%) implies a stark increase in mortality, and a change much higher than that in the next constant rank group. In percentiles 10–45, white women's mortality change is bounded between [−1%,22%]. Among the most educated 30%, white women experienced a 35–38% decline in mortality (1.4% per year).

Turning to men, we find a similar divergence of the least educated 10%. Their mortality increased between 34 and 63%, while the group from percentiles 10–45 experienced less than a 6% mortality change in either direction. White men in the top 30% saw mortality fall by 41%. As above, the naive estimates from Section 4.1 are outside of these bounds, but they are not far from the upper bound estimates.

The remaining panels of Figure 2 show estimates for 50–54-year-old black women and men respectively. Mortality rates among blacks also diverged by education group, but less so than among whites. Among 50–54-year-old black women, mortality rose by 22–24% for the bottom 10%, but declined by 19% or more among all groups in the top 90%. For black men aged 50–54, mortality declined in all education groups, by 18–30% among the bottom 10% and by 61–62% among the top 30%.

4.3 Constant Education Percentile Changes in Mortality at Other Ages

Figures 3 and 4 respectively present mortality changes for white women and men by constant education percentile for all age groups from 1992–1994 to 2013–2015, for all-cause mortality and for deaths of despair. All change estimates are divided by total mortality in 1992–1994. Total mortality changes can thus be directly interpreted as percentage changes. Changes in deaths of despair can be interpreted as the contribution of deaths of despair to the percentage change in total mortality. In other words, the numbers for deaths of despair show how all-cause mortality would have changed if death rates were changing for deaths of despair but were constant for all other causes.[6]

[6]For example, for 25–29 year old white women in the least educated 10%, the change in all-cause mortality is bounded between 65% and 100% (from [129,137] to [227,257]). The mortality rate from deaths of despair for this group rose over 400% (from [18,19] to [96,105] per 100,000), but this number is high in part because it begins at a low base. If the mortality rate for all causes other than deaths of despair was unchanged, then total mortality would have risen between 56% and 68% — these are the numbers for deaths of despair displayed on the graph.

All-cause mortality rates among white men and women aged 45–69 were similar to the trends for 50–54-year-olds discussed in Section 4.2. Deaths of despair rose substantially in percentage terms but explain only a small share of rising mortality among the least educated middle-aged whites. Among younger whites, death rates rose deeper into the education distribution, and there is less of a difference between mortality change in percentiles 0–10 and percentiles 10–45, especially among women. For these younger groups, the change in deaths from despair explains nearly all of the increase in total mortality.

Figures 5 and 6 show the same results for black women and men respectively. Mortality diverged substantially across education groups among blacks, as it did among whites, for both men and women. Mortality change in the bottom 10% fluctuated around zero. The least educated black men experienced falling mortality at most ages, while black women experienced small mortality declines at younger ages, but small mortality increases from ages 45–59. For blacks at all higher levels of education, mortality declined substantially at all ages, with particularly large gains for the most educated black men. Even though the percentage changes in deaths of despair among blacks are in some cases large, they begin from such a small base relative to total mortality that these changes have little importance for overall death rates.

Given the large mortality increase of whites in the bottom 10% and the small mortality improvements among the least educated blacks, white men over the age of 50 and white women over the age of 40 now have higher mortality than similar-age blacks, conditional on being in the least educated 10%. Before 2007, there are no age or education percentile bins where white men have higher mortality than blacks, and the same is true before 2001 for women.

Appendix Figure A2 and Table A2 further decompose these mortality changes into deaths from heart disease, cancer, injuries and other causes. It is notable that among middle-aged whites in the bottom 10%, mortality from cancer, heart disease, and other diseases all rose over the sample period.[7]

One way of summarizing these results is to aggregate mortality changes across all ages, though it masks some of the important heterogeneity. To aggregate mortality rates across ages while holding

[7]Note that in Table A2, we show the percentage changes for each cause, while Figure A2 shows the contribution of each cause to total mortality. For deaths of despair, the percentage changes are very large because they begin from a small base; but for many cohort groups, they contribute only a little bit to changes in total mortality.

constant the change in the population age distribution, we weight the age-specific mortality rates in the data with the standardized U.S. population distribution for ages 25–69.[8] Using these aggregates, Table 1 presents age-adjusted bounds on mortality change from 1992–1994 to 2013–2015 for each constant education percentile group.

The table highlights the substantial divergence of mortality rates between high and low education groups in all four gender and race groups. For whites in the least educated 10%, mortality rose substantially, by 32–49% for men and 59–90% for women. Among blacks in the least educated 10%, mortality rose marginally for women (0 to +5%) and declined for men by 11–15%. For the most educated 30% of individuals across all race and gender groups, mortality rates fell by over 35%, with the largest gains for the most educated black men.[9]

Appendix Figure A1 plots these estimates against the naive estimates of mortality change at four *levels* of education: dropouts, high school completion, some college, and B.A. or higher. As expected, naive estimates are all biased upward. Most of the naive estimates are slightly higher than the upper bound on mortality change; the bias is potentially large and in some cases, some of the naive estimates (*e.g.* white women in percentiles 45–70) have the wrong sign.

4.4 Robustness

Appendix E presents a range of robustness checks on the primary results. Appendix E.1 shows that results are robust to alternate assumptions on the bounding methodology. We show that results are similar when: (i) bin boundaries are based on education levels in 1992–1994 rather than 2013–2015; (ii) education percentiles are defined relative to members of the same race and gender, rather than just the same gender; (iii) Assumption 3 (the mortality CEF has jumps or kinks only at major education intervals) is loosened; or (iv) Assumption 2 (expected mortality is non-increasing in education) is loosened. Appendix E.2 demonstrates that our findings cannot be explained by changes in the relative distribution of black-white education ranks *within* education bins.

[8]The standardized U.S. population distribution was obtained from `https://seer.cancer.gov/stdpopulations/`.

[9]These numbers, along with mortality levels by age, education bin, race and sex for all groups are reported in the accompanying data files.

One concern with our mortality estimates may be that they are calculated by dividing the number of deaths (from NCHS data) by the population (from the CPS). As a result, if ethnic status or education is misreported in one of the two datasets, our mortality estimates could be biased. Note that for our mortality *change* estimates to be biased, the extent of misreporting would have to change in one of the two datasets over the sample period and remain the same in the other. While we cannot rule out some misreporting in one or both of these datasets, we can show that the division bias from any such misreporting is unlikely to be large enough to spuriously generate the large changes in mortality that we find among the least educated whites.

We would be most concerned if the death records increasingly overstate the number of white high school dropouts among the deceased, and/or the CPS increasingly understates the population of white high school dropouts. Either of these situations would cause our mortality change estimates to be biased upward. One way that this could happen would be if individuals who are white increasingly report Hispanic identity in the CPS, but not in the death records. A second way would be if individuals who are dropouts increasingly inflate their education when responding to the CPS (thus lowering the population count of dropouts), but their education is correctly reported on death certificates.[10] Note that if education is increasingly inflated in both death certificates and in the CPS, then our change estimates will not be biased. If there is a constant rate of differential misreporting in the CPS and in death certificates, then there will also not be any bias. For our change estimates to be biased, it needs to be the case that misreporting is changing over time in one dataset and not in the other.

We address this potential division bias in three ways. First, in Appendix E.3, we show that measurement error in ethnicity or changes in reporting patterns of Hispanic identity cannot explain our results. We simulate systematic measurement error in Hispanic identity and show that our results are sustained even with highly implausible changes in patterns of Hispanic reporting.

Second, in Appendix E.4, we bound the error that could arise from the possibility of false reporting of education or ethnicity in the CPS by examining the size of synthetic CPS dropout cohorts over

[10]It is also possible that true Hispanic identity is decreasingly reported on death certificates, or that death certificates increasingly report dropout status either among dropouts or those with high school. We view these circumstances as less likely, but the tests below address them as well.

time. If CPS respondents are increasingly overreporting their education, or if white respondents are increasingly reporting themselves as Hispanic, then the synthetic cohort of non-Hispanic white dropouts will shrink in size more than can be explained by the death rate and the rate of continuing adult education. We show that under the worst case assumptions for our hypothesis, misreporting of education in the CPS could potentially account for less than 7% of the mortality change of the least educated white women in the 1950–54 birth cohort and less than 24% in the 1960–64 birth cohort. The worst case bias for white men is even smaller. In the worst case, we would estimate mortality among 50–54-year-old white women in the least educated 10% to have risen by 45–74%, instead of 79–116%. This potential bias would thus account for only a small share of the large mortality increases that we document here, and would close less than half of the gap with individuals in the next education percentile group. As we discuss in Appendix E.4, this worst case bias scenario is very unlikely to be true; it is therefore implausible that erroneous population counts in the CPS are driving our findings.

Third, in Appendix E.5, we calculate mortality rates and other health measures using the National Health Interview Survey (NHIS). The NHIS makes it possible to measure mortality in a sample of individuals without any division bias, because survey respondents are followed up for many years and any deaths are recorded. Unfortunately, the NHIS sample is not large enough to statistically differentiate between the mortality rate among white high school dropouts and white high school completers.[11] However, our point estimates on mortality change are almost entirely within one standard error of the NHIS mortality estimates— our results are thus consistent with a survey that does not suffer from any potential division bias. Relative to our estimates from the NCHS, the NHIS mortality change estimates are slightly higher for male high school dropouts and slightly lower for female high school dropouts, with very wide confidence intervals in both cases. The NHIS broadly supports the notion of divergent outcomes between high school dropouts and high school completers; for instance, Hendi (2015) uses NHIS to calculate that life expectancy at age 25 among white female dropouts fell by 3.2 years between 1991 and 2005.

In Appendix E.5, we also examine self-reported health status in the NHIS, which is measured

[11] In some years, there are zero deaths recorded among high school dropouts for white women. Even in the five-year followups, there are only a handful of deaths in each age group.

more precisely than mortality. We find that self-reported health status declines substantially more for white female dropouts than for white women with all higher levels of education, with the difference concentrated among 40–60 year olds—the same age group that had the highest differential mortality change between dropouts and high school completers in our main analysis (Figure 3). Among men, health changes are similar between dropouts and high school graduates for young men, but among older men, dropouts experience substantially more health deterioration than high school completers, again consistent with our mortality results in Figure 4. Changes in self-reported health status are thus consistent with our finding that mortality changes among whites are driven by those in the least educated 10%. All of the NHIS results are described in more detail in Appendix E.5.

Finally, in Appendix E.6, we replicate the analysis after pooling dropouts and high school completers into a single education group. This eliminates most of the division bias and misreporting concerns because: (i) the synthetic cohort analysis above shows that the size of the less than or equal to high school (LEHS) population in the CPS cannot be biased by more than 10% either for men or for women; and (ii) the group size is much larger, so a small amount of misreporting cannot substantially shift the population size and bias the estimated mortality rate. The disadvantage of pooling these groups is that we can no longer tightly bound mortality among the bottom 10%. However, we can decisively reject the hypothesis that mortality change among the bottom 45% of whites is driven by selection alone. For cohorts under the age of 45, we continue to find that mortality rates in the bottom 45% of the education distribution have risen by more than 50% from 1992–1994 to 2013–2015. These numbers are lower than the estimates for mortality increases in the bottom 10% in the main part of the paper, because they pool the high mortality increases among the bottom 10% with the smaller mortality increases among percentiles 10–45.

To conclude, while there is undoubtedly some measurement error in education and ethnicity in both the vital statistics and the CPS data, it is very unlikely that measurement error can explain our finding of dramatically rising mortality among the least educated non-Hispanic whites. It is worth noting that other measures of socioeconomic status also have their limitations; for example, studies using income as a measure of socioeconomic status often exclude those reporting zero income, and do not

consider transfers or illicit income, which may be important at the bottom of the income distribution.

5 Conclusion

The post-war era has been characterized by improving health and survival of nearly all demographic groups in all developed countries. Rising mortality among white non-Hispanic Americans represents a major deviation from this trend, and understanding the factors behind this change is a central policy concern.

While there has been substantial interest in education as a risk factor for mortality change, the selection bias inherent in earlier estimates of mortality among the less educated has made it difficult to study. We use a partial identification approach to generate estimates of mortality change in constant education percentiles that quantify the uncertainty from changing education bin boundaries over time.

Our findings chart a middle path through the views previously expressed in the literature. We confirm that earlier estimates of mortality at constant levels of education overstated mortality increases due to selection bias. However, the mortality change due to selection bias is swamped by the actual mortality change at constant education percentiles. We show that death rates for the least educated have dramatically diverged from death rates of other groups, in virtually all middle-age race and gender groups. Non-Hispanic whites in the least educated 10% have done particularly poorly. These mortality increases have a range of causes beyond the widely discussed increases in deaths of despair.

These findings are consistent with the mortality divergence across education groups from 1981–2000 (Meara et al., 2008); we show that this divergence has continued through 2015, and cannot be explained by selection bias from rising education. Our findings also support the finding of Case and Deaton (2015; 2017) that rising middle-age mortality is concentrated among less educated whites. But our analysis focuses on more disaggregated population subgroups where education levels have changed substantially over time (high school dropouts, younger cohorts, and women)—subgroups where unadjusted estimates may be substantially biased. The large share of the rise in white mortality that has come from the bottom 10% of the education distribution, along with the increased mortality from causes other than deaths of despair, has not previously been noted.

These results provide a new perspective to recent analyses of changing mortality at different

percentiles in the income distribution (Cristia, 2009; Chetty et al., 2016). Like the poor, the least educated experience a range of socioeconomic disadvantages, such as high unemployment, low insurance coverage, poor nutrition, and exposure to harmful environmental factors. Our estimates imply that recent middle-age mortality increases among the least educated 10% are worse than those among the poorest 10%. This could be because low income is more transitory than low education or because education is a marker of early life disadvantage and reflects low socioeconomic status in the present as well as in past years.

To better understand the causes of these mortality increases, researchers have searched for factors that predict rising mortality (Cutler et al., 2011; Case and Deaton, 2017; Ruhm, 2018). Pinpointing the causes of mortality increase will require taking into account the fact that education is a key predictor of mortality change and that the proximate drivers of mortality change differ substantially across different groups.

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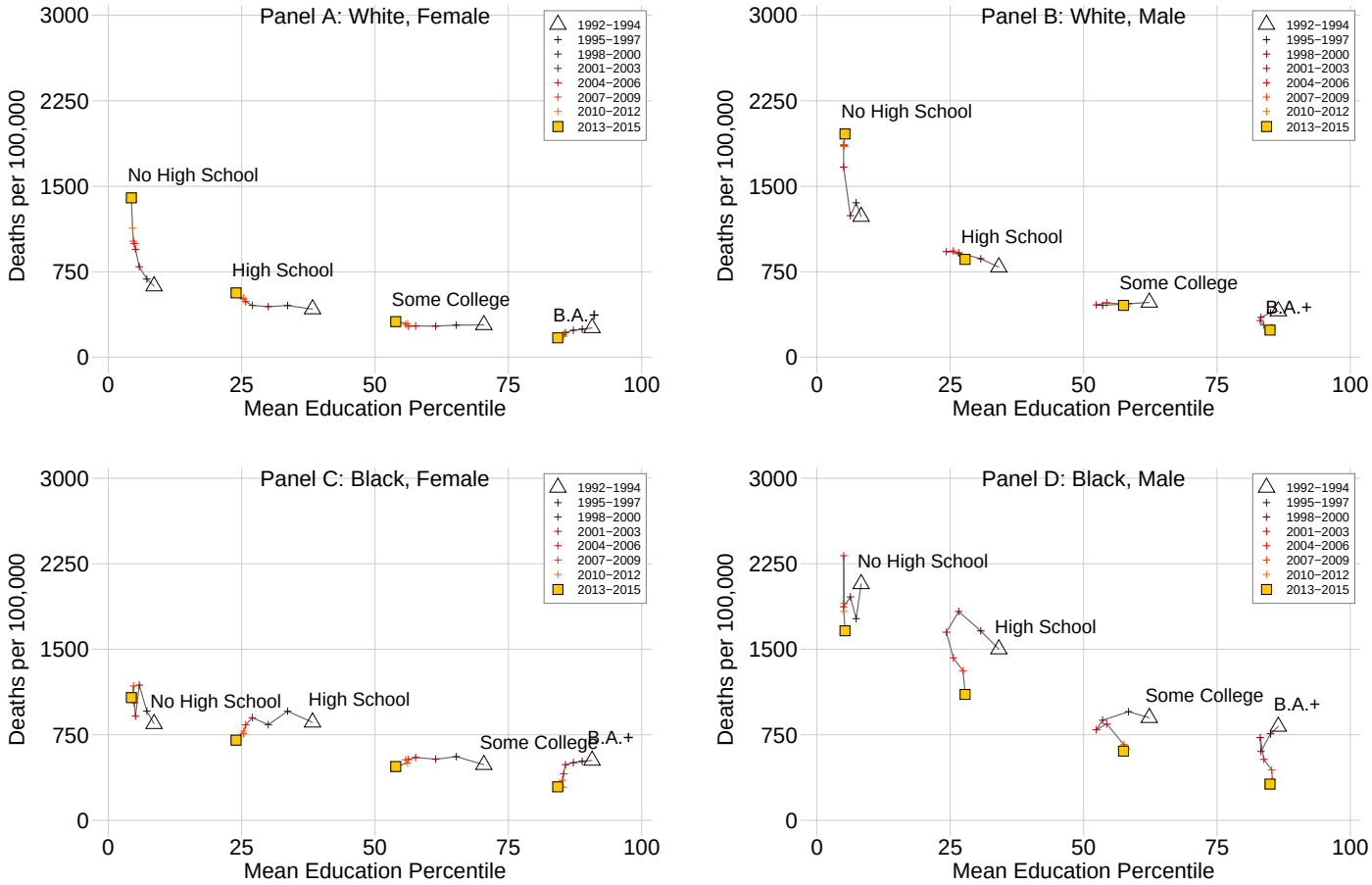
Figure 1

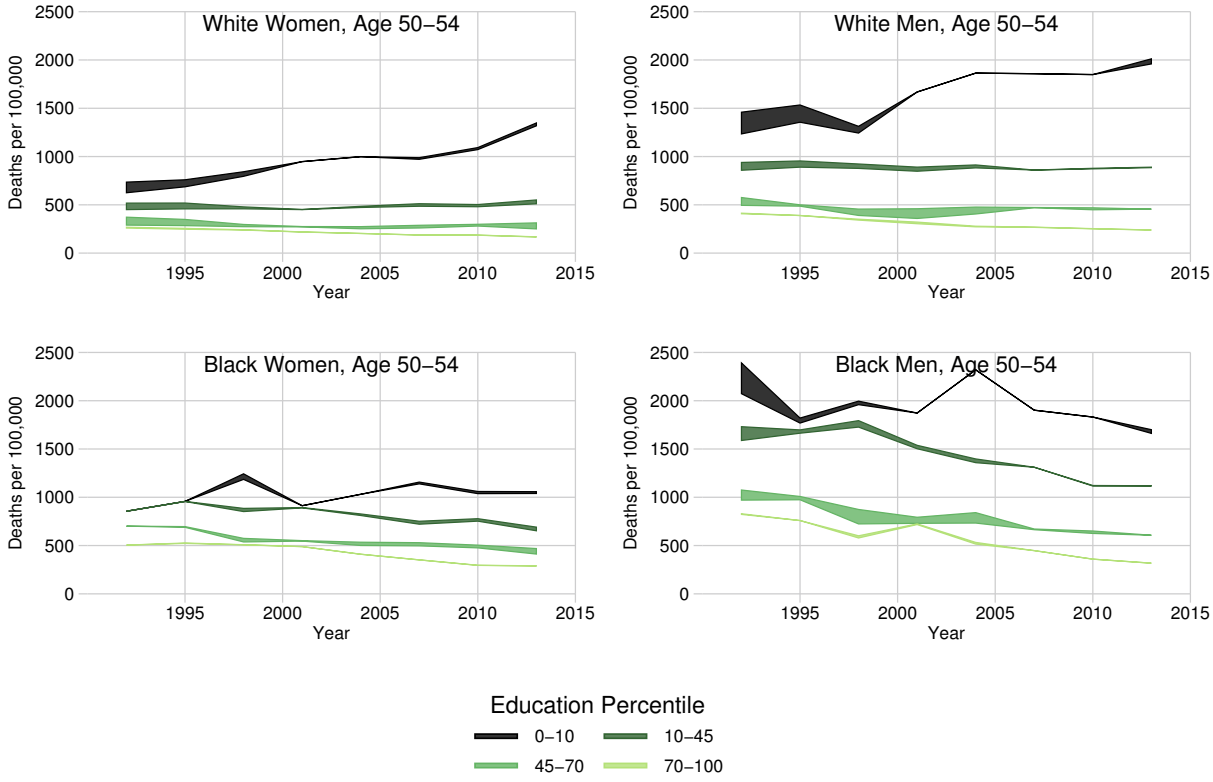
Mortality by Education Level, Age 50–54, 1992–1994 to 2013–2015



Note: "White" refers to non-Hispanic white and "Black" to non-Hispanic black. The figure shows change in mortality and average education rank for individuals aged 50–54 at different levels of education, from 1992–1994 to 2013–2015. Each point represents the average number of deaths per 100,000 people among people with one of four levels of education: No High School, High School, Some College, and a B.A. or Higher. The X coordinate of each point represents the average education percentile among people with the given level of educational completion. For example, a white woman with a high school education was at the 41st percentile of the education distribution in 1992–1994 and at the 24th percentile in 2013–2015. Education percentiles were computed using the American Communities Survey and Current Population Survey, and death records were obtained from the National Center for Health Statistics.

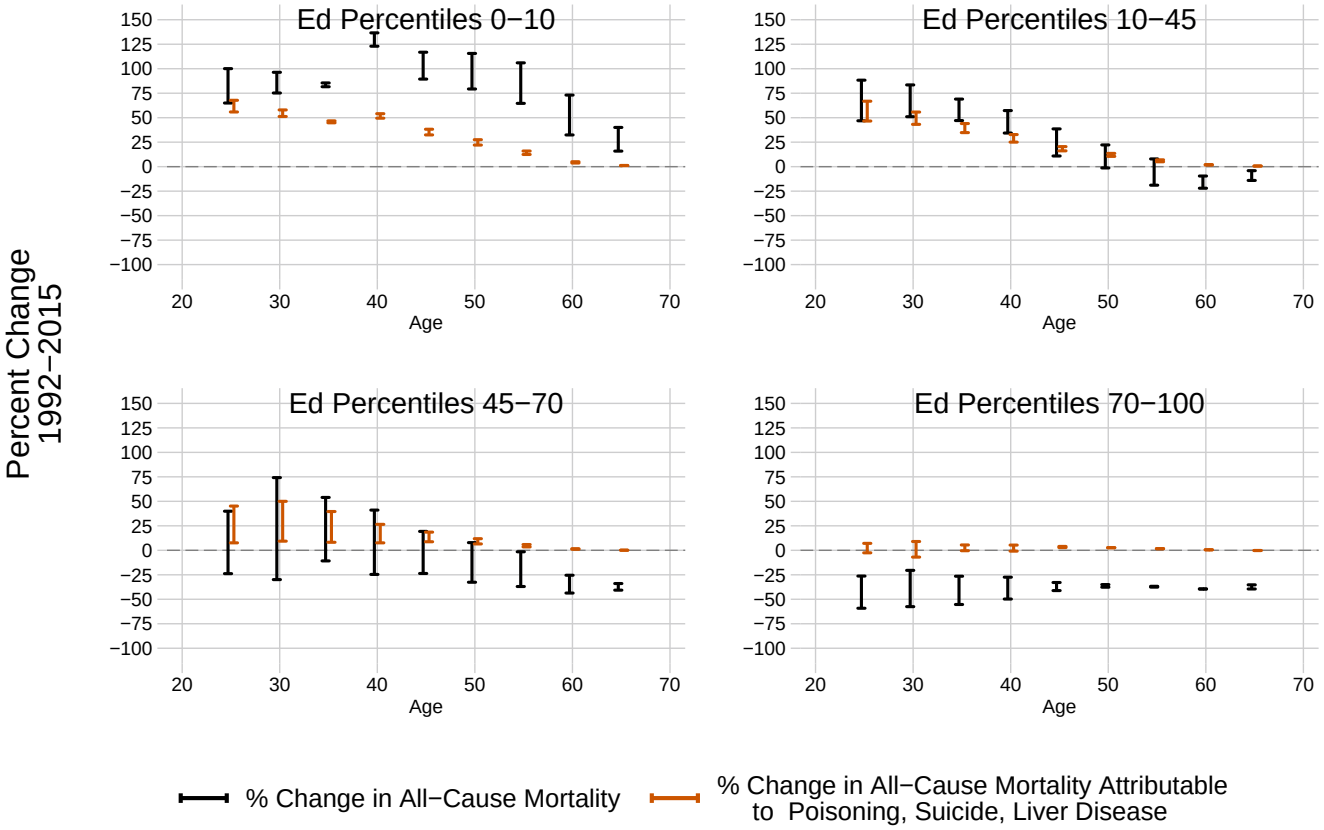
Figure 2

All-Cause Mortality Change in Constant Education Percentiles, Age 50–54, 1992–1994 to 2013–2015



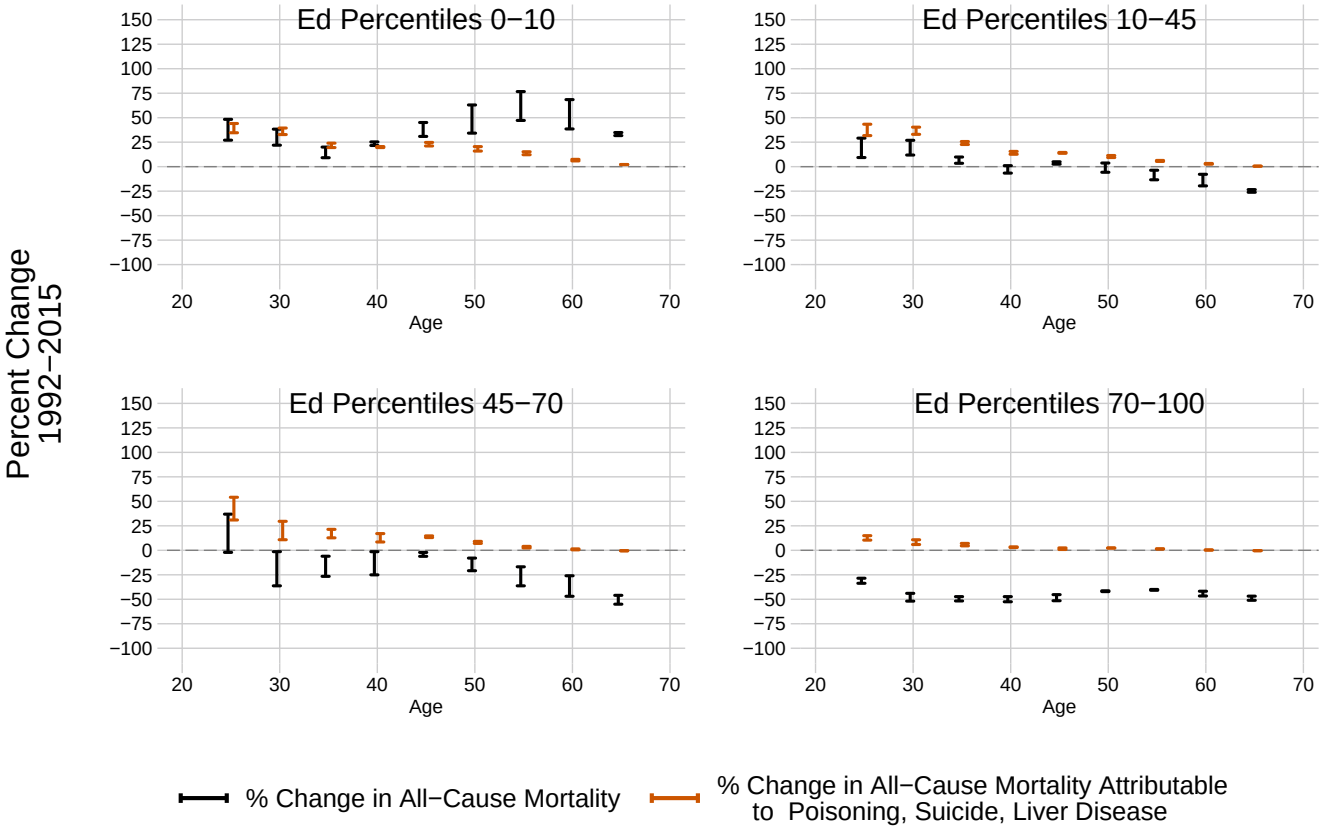
Note: "White" refers to non-Hispanic white and "Black" to non-Hispanic black. Each interval represents the bounded set containing the number of deaths per 100,000 people in a given time period, among people in the education percentiles specified in the legend. The education percentiles correspond to the percentile bins describing four levels of education in 2015: No High School, High School, Some College, and a B.A. or Higher. The lines are thicker in groups / times when the measurement of mortality is less precise because there is a greater difference between the percentile range of interest and the percentile range given in the raw education data. Bounds are computed using set identification methods described in Section 3. The sample consists of people ages 50–54. Education data were compiled from the American Communities Survey and Current Population Survey, and death records were obtained from the National Center for Health Statistics.

Figure 3
Mortality Change in Constant Education Percentiles
1992–1994 to 2013–2015, Non-Hispanic White Women, All Ages



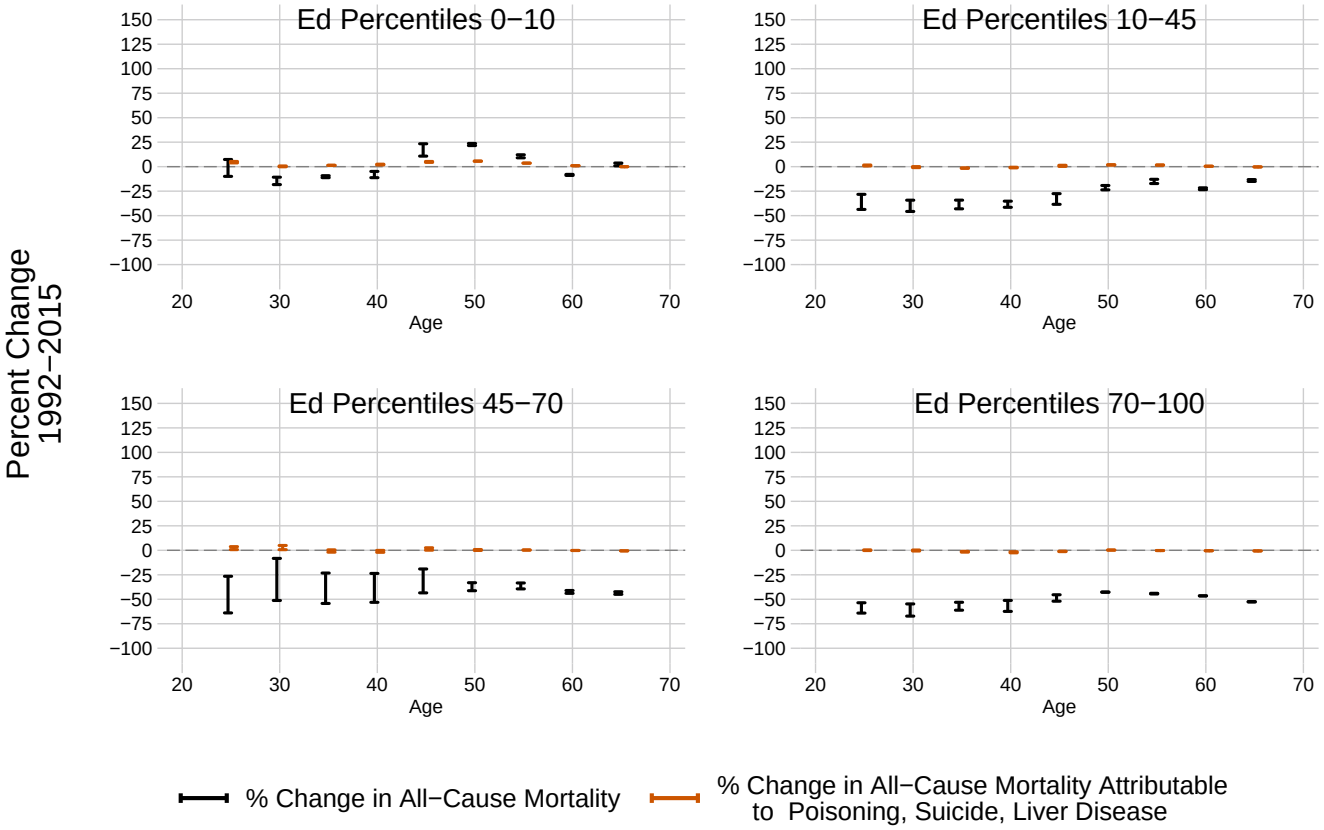
Note: "White" refers to non-Hispanic white. The graph shows changes in mortality by age, sex, race, and constant percentile education bin. The solid black lines show the bounded set containing the percentage change in the mortality rate from 1992–1994 to 2013–2015 for the given group. The orange lines show the bounded set containing the contribution of deaths of despair to the change in total mortality. In other words, they show how much total mortality would have changed if the rates of all deaths *other* than deaths of despair were unchanged. Bounds are computed using the set identification methods described in Section 3. Education data were compiled from the American Communities Survey and Current Population Survey, and death records and codes were obtained from the National Center for Health Statistics.

Figure 4
Mortality Change in Constant Education Percentiles
1992–1994 to 2013–2015, Non-Hispanic White Men, All Ages



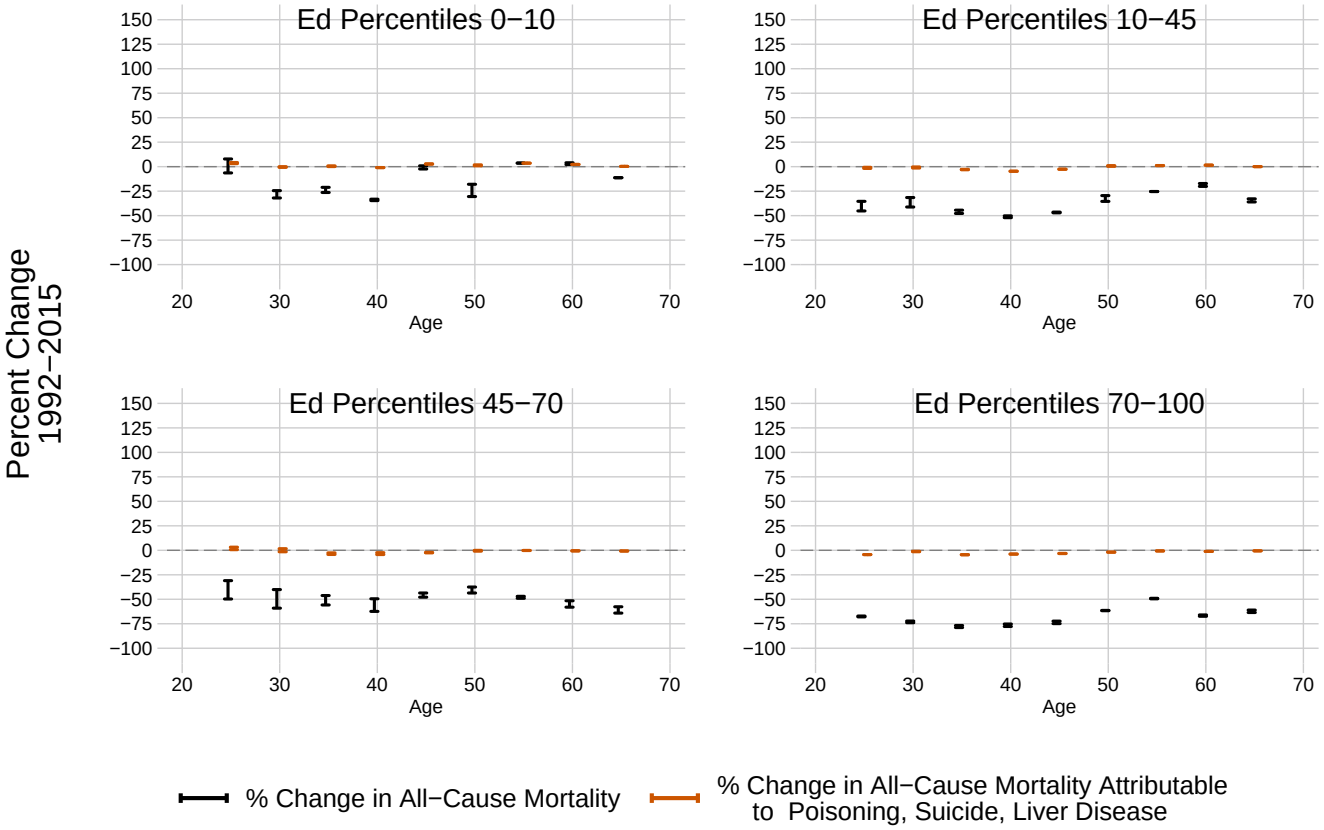
Note: "White" refers to non-Hispanic white. The graph shows changes in mortality by age, sex, race, and constant percentile education bin. The solid black lines show the bounded set containing the percentage change in the mortality rate from 1992–1994 to 2013–2015 for the given group. The orange lines show the bounded set containing the contribution of deaths of despair to the change in total mortality. In other words, they show how much total mortality would have changed if the rates of all deaths *other* than deaths of despair were unchanged. Bounds are computed using the set identification methods described in Section 3. Education data were compiled from the American Communities Survey and Current Population Survey, and death records and codes were obtained from the National Center for Health Statistics.

Figure 5
Mortality Change in Constant Education Percentiles
1992–1994 to 2013–2015, Non-Hispanic Black Women, All Ages



Note: "Black" refers to non-Hispanic black. The graph shows changes in mortality by age, sex, race, and constant percentile education bin. The solid black lines show the bounded set containing the percentage change in the mortality rate from 1992–1994 to 2013–2015 for the given group. The orange lines show the bounded set containing the contribution of deaths of despair to the change in total mortality. In other words, they show how much total mortality would have changed if the rates of all deaths *other* than deaths of despair were unchanged. Bounds are computed using the set identification methods described in Section 3. Education data were compiled from the American Communities Survey and Current Population Survey, and death records and codes were obtained from the National Center for Health Statistics.

Figure 6
Mortality Change in Constant Education Percentiles
1992–1994 to 2013–2015, Non-Hispanic Black Men, All Ages



Note: "Black" refers to non-Hispanic black. The graph shows changes in mortality by age, sex, race, and constant percentile education bin. The solid black lines show the bounded set containing the percentage change in the mortality rate from 1992–1994 to 2013–2015 for the given group. The orange lines show the bounded set containing the contribution of deaths of despair to the change in total mortality. In other words, they show how much total mortality would have changed if the rates of all deaths *other* than deaths of despair were unchanged. Bounds are computed using the set identification methods described in Section 3. Education data were compiled from the American Communities Survey and Current Population Survey, and death records and codes were obtained from the National Center for Health Statistics.

Table 1

Age-Adjusted Changes in All-Cause Mortality
by Education Percentile, 1992–2015

	0-10th	10th-45th	45th-70th	70th-100th
White Women	(+59%, +90%)	(-4%, +16%)	(-36%, -8%)	(-41%, -35%)
White Men	(+32%, +49%)	(-12%, -4%)	(-38%, -23%)	(-48%, -44%)
Black Women	(+0%, +5%)	(-27%, -22%)	(-45%, -34%)	(-51%, -48%)
Black Men	(-15%, -11%)	(-36%, -33%)	(-56%, -49%)	(-66%, -65%)

Note: "White" refers to non-Hispanic white and "black" refers to non-Hispanic black. The table shows the percent change in all-cause mortality, defined as total deaths in a year divided by population. To hold the population distribution constant, we weight the age-specific mortality rates from the data with the standardized U.S. population distribution for ages 25–69. We use age-specific mortality rates from each year, but a single set of weights for all years.

A Appendix: Additional Tables and Figures

Table A1
Distribution of Deaths by Cause, Ages 25–69 (2015)

Cause of Death	Share of Total Deaths
Cancer	30.01
Heart and other diseases of the circulatory system	21.58
Poisoning, suicide, chronic liver disease ("deaths of despair")	11.16
Diseases of the respiratory system	7.69
Accidents and injuries (primarily falls, motor vehicles, assaults)	5.72
Endocrine, nutritional and metabolic diseases	5.11
Diseases of the nervous system	3.26
Cerebrovascular diseases	3.22
Infectious and parasitic diseases	3.12
Diseases of the digestive system	2.53
Diseases of the genitourinary system	1.84
Mental and behavioural disorders	1.72
Deaths not elsewhere classified	0.99
Diseases of the blood and immune disorders	0.94
Diseases of the musculoskeletal system and connective tissue	0.54
Congenital malformations, deformations and chromosomal abnormalities	0.27
Diseases of the skin and subcutaneous tissue	0.17
Pregnancy, childbirth and the puerperium	0.06
Diseases of the eye, ear, mastoid and adnexa	0.00
Certain conditions originating in the perinatal period	0.00

Note: The table shows the distribution of causes of death for individuals aged 25–69 in 2015. Categories are defined by major headings in the ICD-10 Cause-Of-Death lists. The categories of cancer, heart disease and deaths of despair are pooled across categories following the previous literature. See Appendix B for additional details.

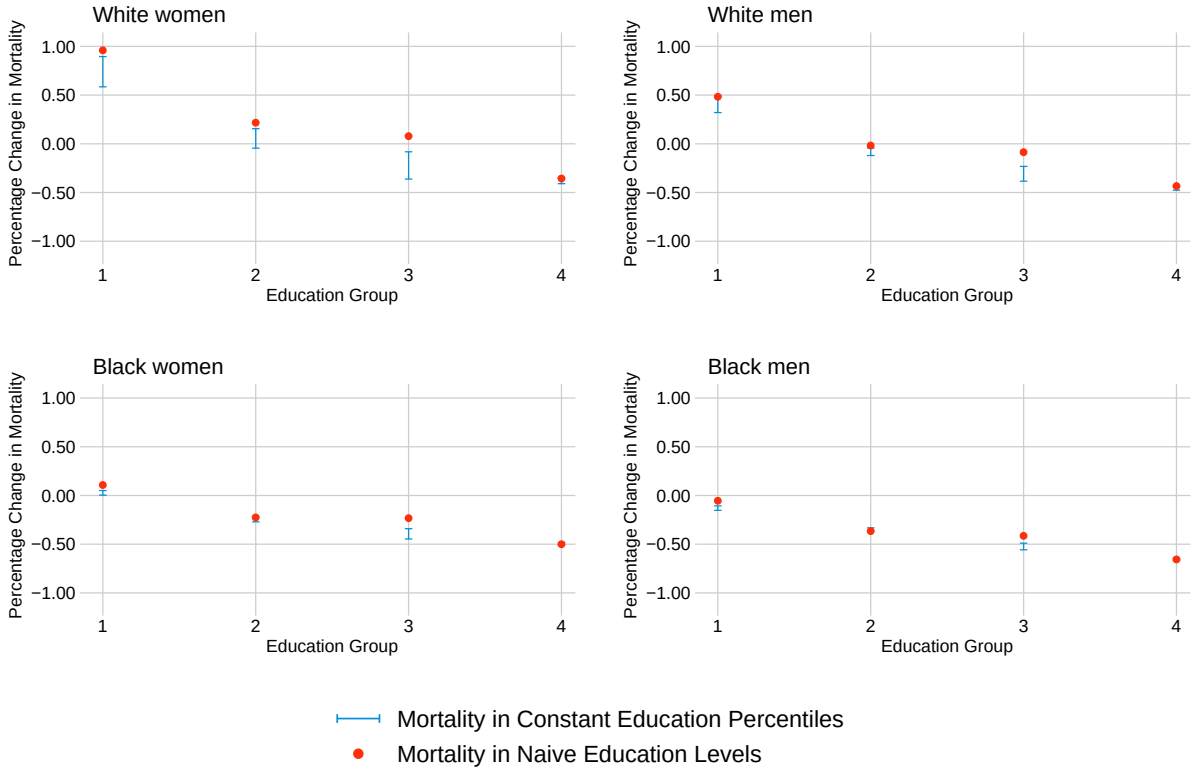
Table A2

Age-Adjusted Changes in Mortality by Education Percentile and Cause
Ages 25-69, 1992–1994 to 2013–2015

		Injuries	Cancer	Heart Disease	Despair	Other	Total
White non-Hispanic Women							
Education Percentile	0-10	(+34%, +50%)	(+22%, +33%)	(+1%, +32%)	(+386%, +431%)	(+92%, +142%)	(+59%, +90%)
	10-45	(+0%, +17%)	(-23%, -15%)	(-38%, -20%)	(+206%, +264%)	(+15%, +44%)	(-4%, +16%)
	45-70	(-31%, +7%)	(-44%, -35%)	(-58%, -33%)	(+74%, +179%)	(-22%, +18%)	(-36%, -8%)
	70-100	(-45%, -37%)	(-42%, -41%)	(-58%, -53%)	(+13%, +37%)	(-31%, -24%)	(-41%, -35%)
White non-Hispanic Men							
Education Percentile	0-10	(+10%, +25%)	(+16%, +27%)	(-7%, +6%)	(+152%, +174%)	(+54%, +83%)	(+32%, +49%)
	10-45	(-8%, +4%)	(-27%, -21%)	(-39%, -33%)	(+100%, +119%)	(-3%, +8%)	(-12%, -4%)
	45-70	(-19%, +6%)	(-47%, -38%)	(-57%, -47%)	(+59%, +100%)	(-34%, -16%)	(-38%, -23%)
	70-100	(-37%, -32%)	(-49%, -47%)	(-61%, -58%)	(+18%, +27%)	(-50%, -46%)	(-48%, -44%)
Black non-Hispanic Women							
Education Percentile	0-10	(-10%, -4%)	(-9%, -6%)	(-21%, -16%)	(+54%, +63%)	(+12%, +20%)	(+0%, +5%)
	10-45	(-42%, -36%)	(-26%, -24%)	(-42%, -37%)	(+7%, +19%)	(-19%, -11%)	(-27%, -22%)
	45-70	(-47%, -29%)	(-40%, -36%)	(-58%, -50%)	(-14%, +17%)	(-39%, -22%)	(-45%, -34%)
	70-100	(-58%, -52%)	(-44%, -43%)	(-63%, -61%)	(-31%, -21%)	(-47%, -44%)	(-51%, -48%)
Black non-Hispanic Men							
Education Percentile	0-10	(+3%, +12%)	(-23%, -20%)	(-25%, -22%)	(+20%, +27%)	(-16%, -11%)	(-15%, -11%)
	10-45	(-29%, -22%)	(-41%, -39%)	(-40%, -37%)	(-14%, -8%)	(-37%, -33%)	(-36%, -33%)
	45-70	(-47%, -30%)	(-61%, -56%)	(-58%, -53%)	(-30%, -15%)	(-56%, -50%)	(-56%, -49%)
	70-100	(-66%, -63%)	(-66%, -65%)	(-66%, -64%)	(-50%, -46%)	(-68%, -67%)	(-66%, -65%)

Note: The table shows age adjusted percentage change in mortality from 1992–1994 to 2013–2015 for individuals aged 25 to 69, by race, gender and education percentile bin. Each table entry shows the upper and lower bound on the percentage change in mortality over the sample period for the given cause and population subgroup. Ages are adjusted with a standardized U.S. population distribution, which holds constant that age distribution of the population across all years. Education percentile bins approximately describe the 2015 distribution of education across four categories: high schools dropouts (percentiles 0-10), high school graduates (10-45), some college (45-70) and B.A. or higher (70-100). "White" refers to non-Hispanic white and "Black" to non-Hispanic black.

Figure A1
Bounds on Constant Percentile Mortality Change and Naive Point Estimates
Age-Adjusted Populations, 1992–1994 to 2013–2015

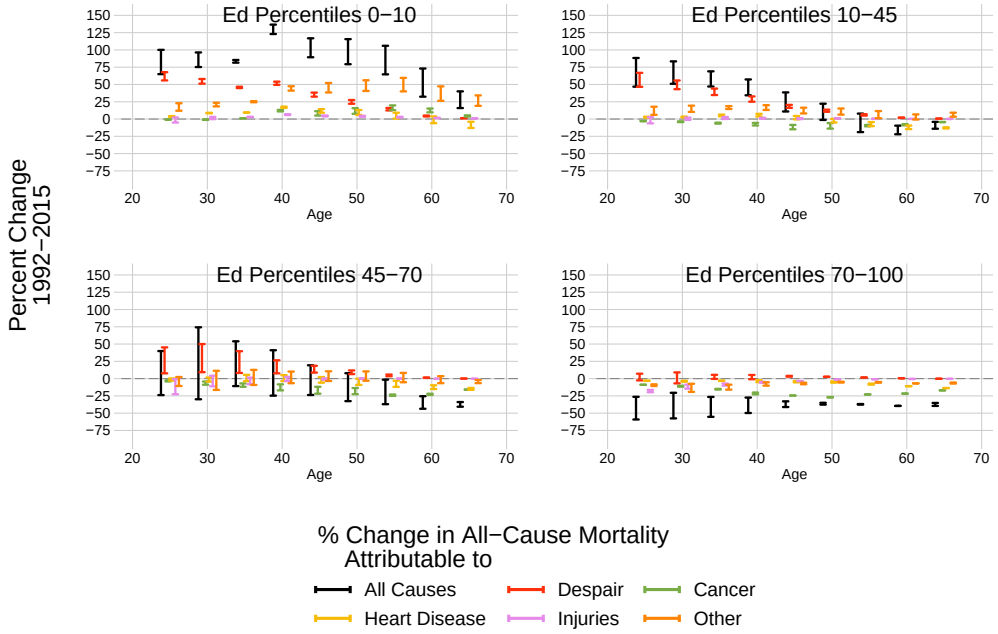


Note: "White" refers to non-Hispanic white and "Black" to non-Hispanic black. The line segments in the graph show bounds on age-adjusted mortality change from 1992–1994 to 2013–2015 for a standardized population, as in Table 1. The four education groups represent education percentiles (1) 0–10; (2) 10–45; (3) 45–70; and (4) 70–100. The points in the graph show naive estimates of mortality rates at fixed education *levels*; the four education levels for the points represent (1) high school dropouts; (2) high school completers; (3) some college; and (4) a B.A. or higher.

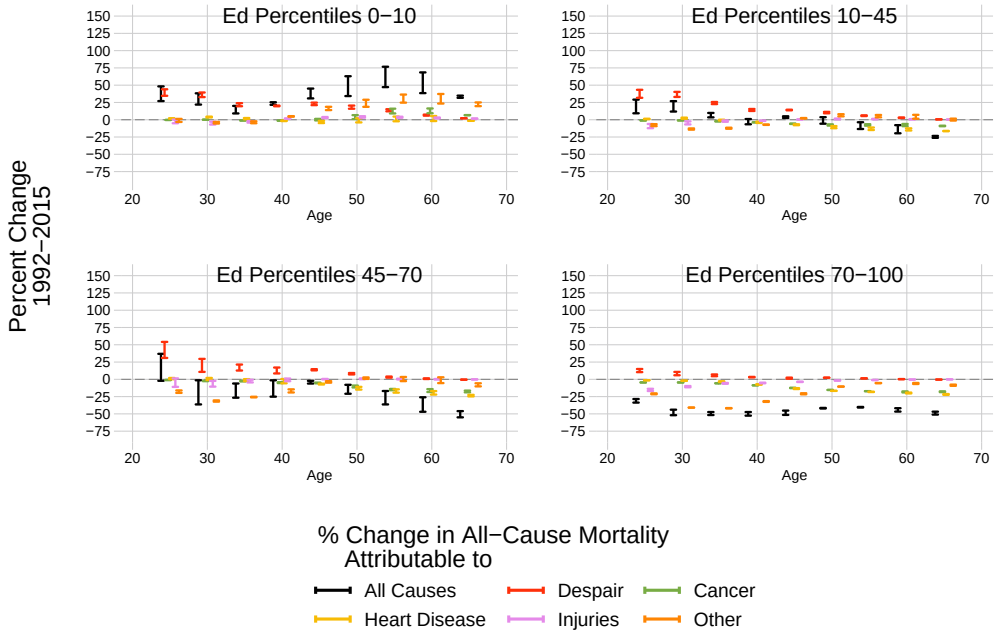
Figure A2

Change in Mortality from 1992–1994 to 2013–2015, Disaggregated Causes, All Groups

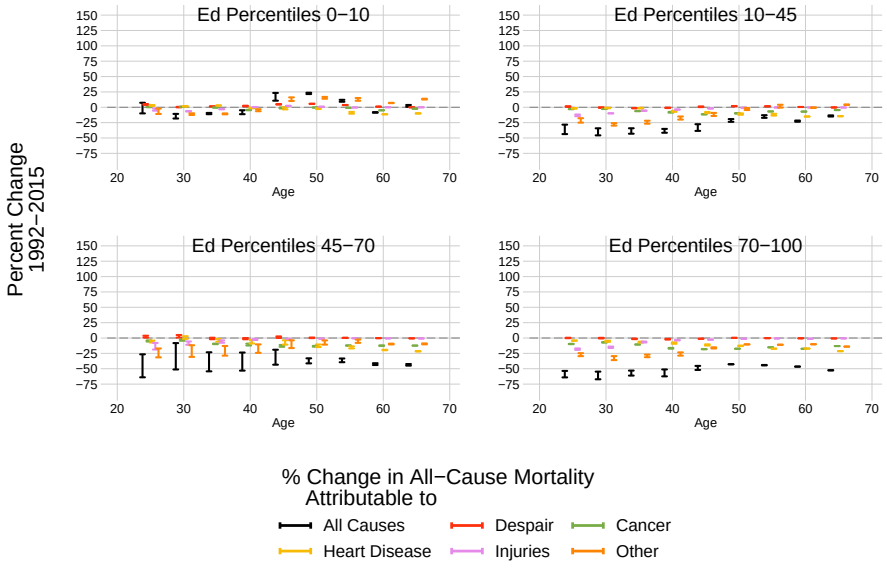
Panel A: White Women



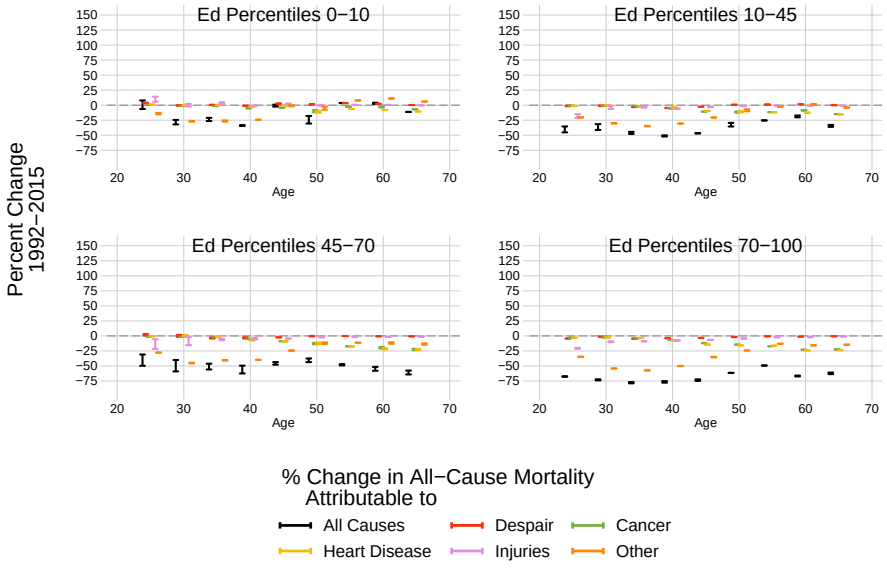
Panel B: White Men



Panel C: Black Women



Panel D: Black Men



Note: "White" refers to non-Hispanic white. The graph shows changes in mortality by age, sex, race, and constant percentile education bin. The solid black lines show the bounded set containing the percentage change in the mortality rate from 1992–1994 to 2013–2015 for the given group. The colored lines show the bounded sets containing the contribution of other causes of death (deaths of despair, cancers, heart disease, injuries and others causes) to the change in total mortality. In other words, they show how much total mortality would have changed if the rates of all deaths *other* than the specific cause were unchanged. Bounds are computed using the set identification methods described in Section 3. Education data were compiled from the American Communities Survey and Current Population Survey, and death records and codes were obtained from the National Center for Health Statistics.

B Appendix: Data Construction

This section provides additional details on data construction. The All Cause Mortality file provided by the National Center for Health Statistics reports the number of deaths, by age, race, gender, education and state.

Missing Educations. Georgia, Oklahoma, Rhode Island, and South Carolina do not consistently report education data. Because their entry and exit from the data could bias mortality trends, we drop mortality records and population totals for these states. The sample mortality rates are thus mortality rates for the remaining 46 states.

The remaining mortality records occasionally report missing education. In each age-gender-race-year category, we obtain the proportion of death records with non-missing educations belonging to each education group. We assign an education group to mortality records where education is missing, assuming that the missing distribution is the same as the non-missing distribution. For example, if 25% of mortality records with non-missing education in a given cell have a high school degree only, we assign 25% of the mortality records with missing education to have a high school degree. This practice is standard; see Case and Deaton (2015; 2017). After dropping the four states, 3.06% of white and 4.9% of black mortality records are missing education. On average, 12.6% of missing deaths are assigned to high school dropouts; even in the extreme case where *all* of these assignments were incorrect, it would erroneously assign only 0.39% of deaths to the bottom education bin, thus creating very little bias. Missing data thus cannot account for the large mortality changes described in the body of the paper.

Institutionalized Populations. The CPS does not survey institutionalized populations, *e.g.* people in prisons or hospitals, but deaths in institutions are counted in the mortality records. To obtain accurate mortality rates, we generate institutionalized populations in each year as follows:

1. We obtain counts of the institutionalized population by age, education, gender, race, and year in the U.S. Census (1990 and 2000) and American Communities Survey (2005–2015).
2. For years between Censuses (1992–1999) or between the Census and American Communities Survey (2001–2004), we impute the number of institutionalized people in each age-education-gender-race-year by generating a linear prediction of the population between nearest surveys. For example, if there were 1,000 institutionalized white women aged 50–54 with a high school degree in the 1990 Census and 1,200 in the 2000 Census, we would impute 1,100 in 1995 (where

there is no Census available).

3. We add institutionalized populations to our count of the non-institutionalized populations from the CPS. We compute mortality rates as the number of deaths divided by the total population.

The share of the institutionalized population was 1.6% in 2015. In order for errors from this imputation process to substantially bias mortality estimates, the institutionalized population would need to fluctuate wildly in the imputed years. Incarceration rates do rise substantially in the 1990s, but the change is close to linear over time, suggesting that the imputation is a good approximation.

Cause of Deaths. We partition all deaths into five groups: cancer, heart disease, deaths of despair, injuries, and other diseases. We construct these groups by using codes from the International Statistical Classification of Diseases and Related Health Problems. NCHS reports ICD-9 codes for 1992–1998 and ICD-10 codes for 1999–2015. We list below the codes pertaining to each cause of death. For consistency, we follow the data appendix and public code from Case and Deaton (2017) to define deaths from cancer, heart disease, and deaths of despair.

- Cancer. ICD-9: 140–208; ICD-10: C (all).
- Heart Disease. ICD-9: 390-429; ICD-10: I0–I9, I11, I13, I20–I51.
- Deaths of Despair. ICD-9: 571, 850–860, 950–959, 980; ICD-10: K70, K73, K74, X40–45, Y10–15, Y45, Y47, Y49, Y87.0.
- Injuries. ICD-9: 800-999 & not a death of despair; ICD-10: V, W, X, Y & not a death of despair.
- Other Diseases. All deaths not otherwise classified.

Table A1 reports the share of deaths among 25–69-year-olds in 2015, ordered by importance. We report the categories used in the paper, and then disaggregate remaining deaths according to major ICD-10 categories.

C Appendix: Proofs

C.1 Proof of Proposition 1

Part 1: Find x_k^*. First define $\mathcal{V}_k$ as the set of weakly increasing CEFs which meet the bin mean. Put otherwise, let $\mathcal{V}_k$ be the set of $v:[x_k,x_{k+1}]\to\mathbb{R}$ satisfying

$$r_k=\frac{1}{x_{k+1}-x_k}\int_{x_k}^{x_{k+1}}v(x)dx.$$

Now choose $z\in\mathcal{V}_k$ such that

$$z(x)=\begin{cases}r_{k-1}, & x_k\leq x<j\\ r_{k+1}, & j\leq x\leq x_{k+1}.\end{cases}$$

Note that z and j both exist and are unique (it suffices to show that just j exists and is unique, as then z must be also). We can solve for j by noting that z lies in $\mathcal{V}_k$, so it must meet the bin mean. Hence, by evaluating the integrals, j must satisfy:

$$\begin{aligned}r_k&=\frac{1}{x_{k+1}-x_k}\int_{x_k}^{x_{k+1}}z(x)dx\\&=\frac{1}{x_{k+1}-x_k}\left(\int_{x_k}^{j}r_{k-1}dx+\int_{j}^{x_{k+1}}r_{k+1}dx\right)\\&=\frac{1}{x_{k+1}-x_k}((j-x_k)r_{k-1}+(x_{k+1}-j)r_{k+1}).\end{aligned}$$

Note that these expressions invoke assumption U, as the integration of $z(x)$ does not require any adjustment for the density on the x axis. For a more general proof with an arbitrary distribution of x, see the following section.

With some algebraic manipulations, we obtain that $j=x_k^*$.

Part 2: Prove the bounds. In the next step, we show that x_k^* is the smallest point at which no $v\in\mathcal{V}_k$ can be r_{k-1}, which means that there must be some larger lower bound on $E(y|x)$ for $x\geq x_k^*$. In other words, we prove that

$$x_k^*=\sup\Big\{x|\text{ there exists }v\in\mathcal{V}_k\text{ such that }v(x)=r_{k-1}.\Big\}.$$

We must show that x_k^* is an upper bound and that it is the least upper bound.

First, x_k^* is an upper bound. Suppose that there exists $j' > x_k^*$ such that for some $w \in \mathcal{V}_k$, $w(j') = r_{k-1}$. Observe that by monotonicity and the bounds from Manski and Tamer (2002), $w(x) = r_{k+1}$ for $x \leq j'$; in other words, if $w(j')$ is the mean of the mean of the prior bin, it can be no lower or higher than the mean of the prior bin up to point j'. But since $j' > j$, this means that

$$\int_{x_k}^{j'} w(x)dx < \int_{x_k}^{j'} z(x)dx,$$

since $z(x) > w(x)$ for all $h \in (j,j')$. But recall that both z and w lie in $\mathcal{V}_k$ and must therefore meet the bin mean; i.e.,

$$\int_{x_k}^{x_{k+1}} w(x)dx = \int_{x_k}^{x_{k+1}} z(x)dx.$$

But then

$$\int_{j'}^{x_{k+1}} w(x)dx > \int_{j'}^{x_{k+1}} z(x)dx.$$

That is impossible by the bounds from Manski and Tamer (2002), since $w(x)$ cannot exceed r_{k+1}, which is precisely the value of $z(x)$ for $x \geq j$.

Second, j is the least upper bound. Fix $j' < j$. From the definition of z, we have shown that for some $h \in (j',j)$, $z(h) = r_{k-1}$ (and $z \in \mathcal{V}_k$). So any point j' less than j would not be a lower bound on the set — there is a point h larger than j' such that $z(h) = r_{k-1}$.

Hence, for all $x < x_k^*$, there exists a function $v \in \mathcal{V}_k$ such that $v(x) = r_{k-1}$; the lower bound on $E(y|x)$ for $x < x_k^*$ is no greater than r_{k-1}. By choosing z' with

$$z'(x) = \begin{cases} r_{k-1}, & x_k \leq x \leq j \\ r_{k+1}, & j < x \leq x_{k+1}, \end{cases}$$

it is also clear that at x_k^*, the lower bound is no larger than r_{k-1} (and this holds in the proposition itself, substituting in x_k^* into the lower bound in the second equation).

Now, fix $x' \in (x_k^*, x_{k+1}]$. Since x_k^* is the supremum, there is no function $v \in \mathcal{V}_k$ such that $v(x') = r_{k-1}$. Thus for $x' > x_k^*$, we seek a sharp lower bound larger than r_{k-1}. Write this lower bound as

$$Y_{x'}^{min} = \min\Big\{ v(x') \text{ for all } v \in \mathcal{V}_k \Big\},$$

where $Y_{x'}^{min}$ is the smallest value attained by any function $v \in \mathcal{V}_k$ at the point x'.

We find this $Y_{x'}^{min}$ by choosing the function which maximizes every point after x', by attaining the value of the subsequent bin. The function which minimizes $v(x')$ must be a horizontal line up to this point.

Pick $\tilde{z} \in \mathcal{V}_k$ such that

$$\tilde{z}(x) = \begin{cases} \underline{Y}, & x_k \leq x' \\ r_{k+1}, & x' < x_{k+1} \leq x_{k+1} \end{cases}.$$

By integrating $\tilde{z}(x)$, we claim that $\underline{Y}$ satisfies the following:

$$\frac{1}{x_{k+1} - x_k}((x' - x_k)\underline{Y} + (x_{k+1} - x')r_{k+1}) = r_k.$$

As a result, $\underline{Y}$ from this expression exists and is unique, because we can solve the equation. Note that this integration step also requires that the distribution of x be uniform, and we generalize this argument in the following section.

By similar reasoning as above, there is no $Y' < \underline{Y}$ such that there exists $w \in \mathcal{V}_k$ with $w(x') = Y'$. Otherwise there must be some point $x > x'$ such that $w(x') > r_{k+1}$ in order that w matches the bin means and lies in $\mathcal{V}_k$; the expression for $\underline{Y}$ above maximizes every point after x', leaving no additional room to further depress $\underline{Y}$.

Formally, suppose there exists $w \in \mathcal{V}_k$ such that $w(x') = Y' < \underline{Y}$. Then $w(x') < \tilde{z}(x')$ for all $x < x'$, since w is monotonic. As a result,

$$\int_{x_k}^{x'} \tilde{z}(x)dx > \int_{x_k}^{x'} w(x)dx.$$

But recall that

$$\int_{x_k}^{x_{k+1}} w(x)dx = \int_{x_k}^{x_{k+1}} \tilde{z}(x)dx,$$

so

$$\int_{x'}^{x_{k+1}} w(x)dx > \int_{x'}^{x_{k+1}} \tilde{z}(x)dx.$$

This is impossible, since $\tilde{z}(x) = r_{k+1}$ for all $x > x'$, and by Manski and Tamer (2002), $w(x) \leq r_{k+1}$ for all $w \in \mathcal{V}_k$. Hence there is no such $w \in \mathcal{V}_k$, and therefore $\underline{Y}$ is smallest possible value at x', i.e. $\underline{Y} = Y_{x'}^{min}$.

By algebraic manipulations, the expression for $\underline{Y}=Y_x^{min}$ reduces to

$$Y_x^{min}=\frac{(x_{k+1}-x_k)r_k-(x_{k+1}-x)r_{k+1}}{x-x_k},\ x\geq x_k^*.$$

The proof for the upper bounds uses the same structure as the proof of the lower bounds.

Finally, the body of this proof gives sharpness of the bounds. For we have introduced a CEF $v\in\mathcal{V}_k$ that obtains the value of the upper and lower bound for any point $x\in[x_k,x_{k+1}]$. For any value y within the bounds, one can generate a CEF $v\in\mathcal{V}_k$ such that $v(x)=y$. □

C.2 Analytical Bounds when Uniformity Does Not Hold

Suppose we relax assumption U and merely characterize x by some known probability density function. Then we can derive the following bounds.

Proposition 2. *Let x be in bin k. Let $f_k(x)$ be the probability density function of x in bin k. Under assumptions M, I, MI (Manski and Tamer, 2002), and without additional information, the following bounds on $E(y|x)$ are sharp:*

$$\begin{cases} r_{k-1}\leq E(y|x)\leq \frac{r_k-r_{k-1}\int_{x_k}^{x} f_k(s)ds}{\int_{x}^{x_{k+1}} f_k(s)ds}, & x<x_k^* \\ \frac{r_k-r_{k+1}\int_{x}^{x_{k+1}} f_k(s)ds}{\int_{x_k}^{x} f_k(s)ds}\leq E(y|x)\leq r_{k+1}, & x\geq x_k^* \end{cases}$$

where x_k^* satisfies:

$$r_k=r_{k-1}\int_{x_k}^{x_k^*} f_k(s)ds+r_{k+1}\int_{x_k^*}^{x_{k+1}} f_k(s)ds.$$

The proof follows the same argument as in Proposition 1. With an arbitrary distribution, $\mathcal{V}_k$ now constitutes the functions $v:[x_k,x_{k+1}]\to\mathbb{R}$ which satisfy:

$$\int_{x_k}^{x_{k+1}} v(s)f_k(s)ds=r_k.$$

As before, choose $z \in \mathcal{V}_k$ such that

$$z(x) = \begin{cases} r_{k-1}, & x_k \leq x < j \\ r_{k+1}, & j \leq x \leq x_{k+1}. \end{cases}$$

Because the distribution of x is no longer uniform, j must now satisfy

$$\begin{aligned} r_k &= \int_{x_k}^{x_{k+1}} z(s) f_k(s) ds \\ &= r_{k-1} \int_{x_k}^{j} f_k(s) ds + r_{k+1} \int_{j}^{x_{k+1}} f_k(s) ds. \end{aligned}$$

This implies that $j = x_k^*$, precisely.

The rest of the arguments follow identically, except we now claim that for $x > x_k^*$, $\underline{Y} = Y_x^{min}$ satisfies the following:

$$r_k = \int_{x_k}^{x} Y_x^{min} f_k(s) ds + \int_{x}^{x_{k+1}} r_{k+1} f_k(s) ds.$$

By algebraic manipulations, we obtain:

$$Y_x^{min} = \frac{r_k - r_{k+1} \int_x^{x_{k+1}} f_k(s) ds}{\int_{x_k}^{x} f_k(s) ds}$$

and the proof of the lower bounds is complete. As before, the proof for upper bounds follows from identical logic. □

C.3 Bounds on μ_a^b

Define

$$\mu_a^b = \frac{1}{b-a} \int_a^b E(y|x) di.$$

Let Y_x^{min} and Y_x^{max} be the lower and upper bounds respectively on $E(y|x)$ given by Proposition 1. We seek to bound μ_a^b when x is observed only in discrete intervals.

Proposition 3. *Let $b \in [x_k, x_{k+1}]$ and $a \in [x_h, x_{h+1}]$ with $a < b$. Let assumptions M, I, MI (Manski and Tamer, 2002) and U hold. Then, if there is no additional information available, the following*

bounds are sharp:

$$\begin{cases} Y_b^{min} \leq \mu_a^b \leq Y_a^{max}, & h=k \\ \frac{r_h(x_k-a)+Y_b^{min}(b-x_k)}{b-a} \leq \mu_a^b \leq \frac{Y_a^{max}(x_k-a)+r_k(b-x_k)}{b-a}, & h+1=k \\ \frac{r_h(x_{h+1}-a)+\sum_{\lambda=h+1}^{k-1} r_\lambda(x_{\lambda+1}-x_\lambda)+Y_b^{min}(b-x_k)}{b-a} \leq \mu_a^b \leq \frac{Y_a^{max}(x_{h+1}-a)+\sum_{\lambda=h+1}^{k-1} r_\lambda(x_{\lambda+1}-x_\lambda)+r_k(b-x_k)}{b-a}, & h+1<k. \end{cases}$$

The order of the proof is as follows. If a and b lie in the same bin, then μ_a^b is maximized only if the CEF is minimized prior to a. As in the proof of proposition 1, that occurs when the CEF is a horizontal line at Y_x^{min} up to a, and a horizontal line Y_x^{max} at and after a. If a and b lie in separate bins, the value of the integral in bins that are contained between a and b is determined by the observed bin means. The portions of the integral that are not determined are maximized by a similar logic, since they both lie within bins. We prove the bounds for maximizing μ_a^b, but the proof is symmetric for minimizing μ_a^b.

Part 1: Prove the bounds if a and b lie in the same bin. We seek to maximize μ_a^b when $a,b \in [x_k, x_{k+1}]$. This requires finding a candidate CEF $v \in \mathcal{V}_k$ which maximizes $\int_a^b v(x)dx$. Observe that the function $v(x)$ defined as

$$v(x) = \begin{cases} Y_a^{min}, & x_k \leq x < a \\ Y_a^{max}, & a \leq x \leq x_{k+1} \end{cases}$$

has the property that $v \in \mathcal{V}_k$. For if $a \geq x_k^*$, $v = \tilde{z}$ from the second part of the proof of proposition 1. If $a < x_k^*$, the CEF in $\mathcal{V}_k$ which yields Y_a^{max} is precisely v (by a similar argument which delivers the upper bounds in proposition 1).

This CEF maximizes μ_a^b, because there is no $w \in \mathcal{V}_k$ such that

$$\frac{1}{b-a}\int_a^b w(x)dx > \frac{1}{b-a}\int_a^b v(x)dx.$$

Note that for any $w \in \mathcal{V}_k$, $\frac{1}{x_{k+1}-x_k}\int_{x_k}^{x_{k+1}} w(x)dx = \frac{1}{x_{k+1}-x_k}\int_{x_k}^{x_{k+1}} v(x)dx = r_k$. Hence in order that $\int_a^b w(x)dx > \int_a^b v(x)dx$, there are two options. The first option is that

$$\int_{x_k}^{a} w(x)dx < \int_{x_k}^{a} v(x)dx.$$

That is impossible, since there is no room to depress w given the value of v after a. If $a < x_k^*$, then

it is clear that there is no w giving a larger μ_a^b, since $r_{k-1} \leq w(x)$ for $x_{k-1} \leq x \leq a$, so w is bounded below by v. If $a \geq x_k^*$, then $v(x) = r_{k+1}$ for all $a \leq x \leq x_{k+1}$. That would leave no room to depress w further; if $\int_{x_k}^{a} w(x)dx < \int_{x_k}^{a} v(x)dx$, then $\int_{a}^{x_{k+1}} w(x)dx > \int_{a}^{x_{k+1}} v(x)dx$, which cannot be the case if $v = r_{k+1}$, by the bounds given in Manski and Tamer (2002).

The second option is that

$$\int_{b}^{x_k} w(x)dx < \int_{b}^{x_k} v(x)dx.$$

This is impossible due to monotonicity. For if $\int_{a}^{b} w(x)dx > \int_{a}^{b} v(x)dx$, then there must be some point $x' \in [a,b)$ such that $w(x') > v(x')$. By monotonicity, $w(x) > v(x)$ for all $x \in [x', x_{k+1}]$ since $v(x) = Y_a^{max}$ in that interval. As a result,

$$\int_{b}^{x_k} w(x)dx > \int_{b}^{x_k} v(x)dx,$$

since $b \in (x', x_{k+1})$. (If $b = x_{k+1}$, then only the first option would allow w to maximize the desired μ_a^b.)

Therefore, there is no such w, and v indeed maximizes the desired integral. Integrating v from a to b, we obtain that the upper bound on μ_a^b is $\frac{1}{b-a}\int_a^b Y_a^{max} dx = Y_a^{max}$. Note that there may be many functions which maximize the integral; we only needed to show that v is one of them.

To prove the lower bound, use an analogous argument.

Part 2: Prove the bounds if a and b do not lie in the same bin. We now generalize the set up and permit $a,b \in [0,100]$. Let $\mathcal{V}$ be the set of weakly increasing functions such that $\frac{1}{x_{k+1}-x_k}\int_{x_k}^{x_{k+1}} v(x)dx = r_k$ for all $k \leq K$. In other words, $\mathcal{V}$ is the set of functions which match the means of every bin. Now observe that for all $v \in \mathcal{V}$,

$$\begin{aligned}\mu_a^b &= \frac{1}{b-a}\int_a^b v(x)dx \\ &= \frac{1}{b-a}\left(\int_a^{x_{h+1}} v(x)dx + \int_{x_{h+1}}^{x_k} v(x)dx + \int_{x_k}^{b} v(x)dx\right),\end{aligned}$$

by a simple expansion of the integral.

But for all $v \in \mathcal{V}$,

$$\int_{x_{h+1}}^{x_k} v(x)dx = \sum_{\lambda=h+1}^{k-1} r_\lambda (x_{\lambda+1} - x_\lambda)$$

if $h+1<k$ and

$$\int_{x_{h+1}}^{x_k} v(x)dx = 0$$

if $h+1=k$. For in bins completely contained inside $[a,b]$, there is no room for any function in $\mathcal{V}$ to vary; they all must meet the bin means.

We proceed to prove the upper bound. We split this into two portions: we wish to maximize $\int_a^{x_{h+1}} v(x)dx$ and we also wish to maximize $\int_{x_k}^b v(x)dx$. The values of these objects are not codependent. But observe that the CEFs $v \in \mathcal{V}_k$ which yield upper bounds on these integrals are the very same functions which yield upper bounds on $\mu_a^{x_{h+1}}$ and $\mu_{x_k}^b$, since $\mu_s^t = \frac{1}{t-s}\int_s^t v(x)dx$ for any s and t. Also notice that a and x_{h+1} both lie in bin h, while b and x_k both lie in bin k, so we can make use of the first portion of this proof.

In part 1, we showed that the function $v \in \mathcal{V}$, $v:[x_h,x_{h+1}] \to \mathbb{R}$, which maximizes $\mu_a^{x_{h+1}}$ is

$$v(x) = \begin{cases} Y_a^{min}, & x_h \leq x < a \\ Y_a^{max}, & a \leq x \leq x_{h+1}. \end{cases}$$

As a result

$$\max_{v \in \mathcal{V}} \left\{ \int_a^{x_{h+1}} v(x)dx \right\} = \int_a^{x_{h+1}} Y_a^{max} dx = Y_a^{max}(x_{h+1} - a).$$

Similarly, observe that x_k and b lie in the same bin, so the function $v:[x_k,x_{k+1}] \to \mathbb{R}$, with $v \in \mathcal{V}$ which maximizes $\int_{x_k}^b v(x)dx$ must be of the form

$$v(x) = \begin{cases} Y_{x_k}^{min}, & x_k \leq x < a \\ Y_{x_k}^{max}, & b \leq x \leq x_{k+1}. \end{cases}$$

With identical logic,

$$\max_{v \in \mathcal{V}} \left\{ \int_{x_k}^b v(x)dx \right\} = \int_{x_k}^b Y_{x_k}^{max} dx = Y_{x_k}^{max}(b - x_k).$$

And by proposition 1, $x_k \leq x_k^*$ so $Y_{x_k}^{max} = r_k$. (Note that if $x_k = x_k^*$, substituting x_k^* into the second expression of proposition 1 still yields that $Y_{x_k}^{max} = r_k$.)

Now we put all these portions together. First let $h+1=k$. Then $\int_{x_{h+1}}^{x_k} v(x)dx=0$, so we maximize μ_a^b by

$$\frac{1}{b-a}(Y_a^{max}(x_{h+1}-a)+r_k(b-x_k)).$$

Similarly, if $h+1<k$ and there are entire bins completely contained in $[a,b]$, then we maximize μ_a^b by

$$\frac{1}{b-a}\left(Y_a^{max}(x_{h+1}-a)+\sum_{\lambda=h+1}^{k-1} r_\lambda(x_{\lambda+1}-x_\lambda)+r_k(b-x_k)\right).$$

The lower bound is proved analogously. Sharpness is immediate, since we have shown that the CEF which delivers the endpoints of the bounds lies in $\mathcal{V}$. As a result, there is a function delivering any intermediate value for the bounds. □

D Appendix: Methods

This section provides additional detail on the computational details for calculating bounds on mortality within arbitrary percentiles of the education distribution.

We assume that there exists a latent, continuous education rank for each individual, which is partitioned into the education levels observed in the population. This notion arises directly out of a standard human capital model such as that in Card (1999), in which the object of interest is a continuous education variable which is only observed at discrete levels, and is common or implicit in the literature on mortality and education (Meara et al., 2008; Bound et al., 2015; Goldring et al., 2016).

We wish to observe the expected value of mortality, conditional on the latent education rank being in some fixed range, such as the bottom 10%. This is challenging, because in a given year, 20% of the population may be a high school dropout, so we cannot directly observed the expected mortality of the bottom 10%.

Given the latent educational rank setup, this is an interval censoring problem. In Proposition 1 and Proposition 3, we show that in this context, expected mortality can be bounded in any arbitrary percentile rank interval, given minimal structural assumptions about the mortality-education relationship.

The bounds are tightest when the boundaries of the rank interval are close to the boundaries observed in the data. For this reason, we cannot calculate tight bounds on mortality changes for every decile; instead, we focus on four bins that approximate the rank boundaries of the four

education groups in 2015: percentiles (i) 0-10 (dropouts in 2015); (ii) 10-45 (high school graduates in 2015); (iii) 45-70 (some college in 2015); and (iv) 70-100 (B.A. or higher in 2015).

To calculate bounds, we discretize the mortality-education relationship and solve a numerical optimization problem that obtains the highest and lowest possible values of expected mortality at any given point. We assume that the mortality-education CEF is described by a 100-element vector $\hat{\gamma}_x$, which describes expected mortality at each integer education rank or percentile x. This is a nonparametric function which can take any value (between 0 and 100,000 deaths per 100,000) at each x, subject to the structural assumptions above. To bound the expectation of mortality in a particular set of education ranks, we calculate the functions $\hat{\gamma}_x$ which generate the minimum and maximum possible expectation of mortality in that set of ranks, subject to satisfying the structural assumptions, and attaining the minimum feasible mean-squared error with respect to the observed average mortality in each education bin in the data.

The solution to the problem can be understood as the outcome a two-step process. First, consider the set of all functions of expected mortality given education $\hat{\gamma}_x$ that can match the observed mean levels of mortality for each education level, and are consistent with the monotonicity and smoothness assumptions. Second, in this set, find the functions $\hat{\gamma}_x$ that respectively maximize and minimize the overall mortality in the rank bin of interest. These two functions provide the bounds on mortality for that education rank bin.

The following constrained optimization problem provides the lower bound on mortality in the bottom 10% of the latent education distribution, or $Mortality^{min}_{0-10}$:

$$Mortality_{0-10}^{min} = \min_{\hat{\gamma} \in [0,100000]^{100}} \frac{1}{10} \sum_{x=1}^{10} (\hat{\gamma}_x) \tag{D.1}$$

such that

$$\hat{\gamma}_x \text{ is weakly increasing in } x \qquad \text{(Monotonicity)}$$

$$|(\hat{\gamma}_{x+1} - \hat{\gamma}_x) - (\hat{\gamma}_x - \hat{\gamma}_{x-1})| \leq \overline{C},$$
$$\text{unless there is an education level boundary between } x-1 \text{ and } x+1 \qquad \text{(Curvature)}$$

$$\sum_{k=1}^{4} \left[\frac{\|X_k\|}{100} \left(\left(\frac{1}{\|X_k\|} \sum_{x \in X_k} \hat{\gamma}_x \right) - \overline{r}_k \right)^2 \right] = \underline{\text{MSE}}. \qquad \text{(MSE Minimization)}$$

The minimand is the average mortality in the bottom 10%. $\overline{C}$ is the highest allowable curvature across any three points that do not span an education boundary; this expression prevents sharp kinks or breaks in the mortality function. The four education levels observed in the data are indexed by k. X_k is the set of integer education ranks in bin k and $\|X_k\|$ is the width of bin k, or the number of integer ranks in that bin. $\overline{r}_k$ is the observed mortality in education bin k, and $\underline{\text{MSE}}$ is the lowest mean-squared error obtainable out of the entire set of education-mortality functions, which is typically zero. The complementary maximization problem obtains the upper bound on mortality in the bottom 10%.

D.1 Formal Description of Computational Procedure

We now provide more details on the computational procedure.

D.1.1 Conceptual Approach: Functions of the CEF

Let $f(x)$ represent the probability distribution of x. Define Γ as the set of parameterizations of the CEF that obey monotonicity and minimize mean squared error with respect to the observed interval data:

$$\Gamma = \underset{g \in G}{\operatorname{argmin}} \sum_{k=1}^{K} \left\{ \int_{x_k}^{x_{k+1}} f(x)dx \left(\left(\frac{1}{\int_{x_k}^{x_{k+1}} f(x)dx} \int_{x_k}^{x_{k+1}} s(x,g) f(x)dx \right) - \overline{r}_k \right)^2 \right\} \tag{D.2}$$

such that

$s(x,g)$ is weakly increasing in x. (Monotonicity)

Decomposing this expression, $\frac{1}{\int_{x_k}^{x_{k+1}} f(x)dx} \int_{x_k}^{x_{k+1}} s(x,g) f(x)dx$ is the mean value of $s(x,g)$ in bin k, and $\int_{x_k}^{x_{k+1}} f(x)dx$ is the width of bin k. The minimand is thus a bin-weighted MSE.[12] Recall that for the rank distribution, $x_1 = 0$ and $x_{K+1} = 100$.

The bounds on $m(\gamma)$ are therefore:

$$\begin{aligned} m^{min} &= \inf\{m(\gamma) \mid \gamma \in \Gamma\} \\ m^{max} &= \sup\{m(\gamma) \mid \gamma \in \Gamma\}. \end{aligned} \tag{D.3}$$

For example, bounds on the best linear approximation to the CEF can be defined by the following process. First, consider the set of all CEFs that satisfy monotonicity and minimize mean-squared error with respect to the observed bin means.[13] Next, compute the slope of the best linear approximation to each CEF. The largest and smallest slope constitute m^{min} and m^{max}. Note that this definition of the best linear approximator to the CEF corresponds to the *least squares set* defined by Ponomareva and Tamer (2011).

The set of CEFs that describe the upper and lower bounds in Proposition 1 are step functions with substantial discontinuities. If such functions are implausible descriptions of the data, then the researcher may wish to impose an additional constraint on the curvature of the CEF, which will generate tighter bounds. For example, examination of the mortality-income relationship (which can be estimated at each of 100 income ranks, displayed in Figure A3) suggests no such discontinuities. Alternately, in a context where continuity has a strong theoretical underpinning but monotonicity does not, a curvature

[12] While we choose to use a weighted mean squared error penalty, in principle Γ could use other penalties.

[13] In many cases, and in all of our applications, there will exist many such CEFs that exactly match the observed data and the minimum mean-squared error will be zero.

constraint can substitute for a monotonicity constraint and in many cases deliver useful bounds.

We consider a curvature restriction with the following structure:

$$s(x,\gamma) \text{ is twice-differentiable and } |s''(x,\gamma)| \leq \overline{C}. \qquad \text{(Curvature Constraint)}$$

This is analogous to imposing that the first derivative is Lipshitz.[14] Depending on the value of $\overline{C}$, this constraint may or may not bind.

The most restrictive curvature constraint, $\overline{C}=0$, is analogous to the assumption that the CEF is linear. Note that the default practice in many studies of mortality is to estimate the best linear approximation to the CEF of mortality given education (e.g., Cutler et al. (2011) and Goldring et al. (2016)). A moderate curvature constraint is therefore a *less* restrictive assumption than the approach taken in many studies. We discuss the choice of curvature restriction below.

D.1.2 Computational Approach

This section describes a method to numerically solve the constrained optimization problem suggested by Equations D.2 and D.3. We take a nonparametric approach for generality: explicitly parameterizing an unknown CEF with limited data is unsatisfying and could yield inaccurate results if the interval censoring conceals a non-linear within-bin CEF. In the context of mortality, many CEFs of interest do not appear to obey a familiar parametric form (see Figure A3).

To make the problem numerically tractable, we solve the discrete problem of identifying the feasible mean value taken by $E(y|x)$ in each of N discrete partitions of x. We thus assume $E(y|x)=s(x,\gamma)$, where γ is a vector that defines the mean value of the CEF in each of the N partitions. We use $N=100$ in our analysis, corresponding to integer rank bins, but other values may be useful depending on the application. In other words, we will numerically calculate upper and lower bounds on $E(y|x\in[0,1])$, $E(y|x\in[1,2])$, ..., $E(y|x\in[99,100])$. Given continuity in the latent function, the discretized CEF will be a very close approximation of the continuous CEF; in our applications, increasing the value of N increases computation time but does not change any of our results.

[14] Let X,Y be metric spaces with metrics d_X,d_Y respectively. The function $f: X \to Y$ is *Lipschitz continuous* if there exists $K \geq 0$ such that for all $x_1,x_2 \in X$,

$$d_Y(f(x_1),f(x_2)) \leq K d_X(x_1,x_2).$$

We solve the problem through a two-step process. Define a N-valued vector $\hat{\gamma}$ as a candidate CEF. First, we calculate the minimum MSE from the constrained optimization problem given by Equation D.2. We then run a second pair of constrained optimization problems that respectively minimize and maximize the value of $m(\hat{\gamma})$, with the additional constraint that the MSE is equal to the value obtained in the first step, denoted $\underline{\text{MSE}}$. Equation D.4 shows the second stage setup to calculate the lower bound on $m(\hat{\gamma})$. Note that this particular setup is specific to the uniform rank distribution, but setups with other distributions would be similar.

$$m^{min} = \min_{\hat{\gamma} \in [0,100]^N} m(\hat{\gamma}) \tag{D.4}$$

such that

$$s(x,\hat{\gamma}) \text{ is weakly increasing in } x \qquad \text{(Monotonicity)}$$

$$|s''(x,\hat{\gamma})| \leq \overline{C}, \qquad \text{(Curvature)}$$

$$\sum_{k=1}^{K} \left[\frac{\|X_k\|}{100} \left(\left(\frac{1}{\|X_k\|} \sum_{x \in X_k} s(x,\hat{\gamma}) \right) - \overline{r}_k \right)^2 \right] = \underline{\text{MSE}} \qquad \text{(MSE Minimization)}$$

X_k is the set of discrete values of x between x_k and x_{k+1} and $\|X_k\|$ is the width of bin k. The complementary maximization problem obtains the upper bound on $m(\hat{\gamma})$.

Note that setting $m(\gamma) = \gamma_x$ (the x$^{\text{th}}$ element of γ) obtains bounds on the value of the CEF at point x. Calculating this for all ranks x from 1 to 100 generates analogous bounds to those derived in proposition 1, but satisfying the additional curvature constraint. Similarly $m(\gamma) = \frac{1}{b-a} \sum_{x=a}^{b} \gamma_x$ obtains bounds on μ_a^b.

The numerical method can easily permit the curvature constraint to vary over the CEF. For example, one might believe that there are discontinuities in the CEF at bin boundaries, due to sheepskin effects (Hungerford and Solon, 1987); high-school graduates, upon receiving a diploma, may indeed experience discretely lower mortality probability due to better labor-market outcomes. In our case, we permit the CEF to jump at bin boundaries and but impose that the CEF is curvature-constrained within each bin. In other settings, researchers might impose that the CEF has a large (but finite) curvature in one portion of its domain and be more constrained elsewhere.

D.2 Comparison with Other Approaches

The selection bias in estimates of mortality change among the less educated is widely recognized and has been examined in other studies. Meara et al. (2008) and Bound et al. (2015) adjust for this bias by randomly reassigning deaths to different education bins, so that bin sizes are comparable across time. For example, to obtain an estimate of mortality in the bottom quartile of the education distribution in 1992 when 20% of the male population are dropouts and an additional 39% are high school graduates, they would reassign $5/39 = 12.8\%$ of the high school graduate population and 12.8% of deaths among the high school graduate population to the bottom bin.

This approach is equivalent to assuming that the conditional expectation function of mortality given latent education rank takes a specific functional form—a step function that is totally flat in each education category, and has a discrete jump at each education boundary. To be concrete, this assumption states that an individual who just barely managed to complete high school (and thus has the lowest latent education rank among high school graduates) has exactly the same expected mortality risk as a high school graduate who was right at the margin of completing a two year college degree (and thus has the highest latent education rank among high school graduates). Standard human capital theory suggests that the true functional form is not flat in each category—the high school educated individuals who were at the margin of completing some higher education would have had higher socioeconomic status than those who barely made it to high school, and thus lower mortality risk.

This implicit functional form is nevertheless considered a valid functional form in our bounding exercise when we omit the smoothness constraint; the bounds in Panel D of Appendix Figure E1 contain the mortality estimates generated by the Meara, Richards and Cutler / Bound et al. (MRCB) function. But this functional form underestimates mortality among the least educated in all periods, because it constructs bins by combining dropouts with average high school graduates – even though the high school graduates with the lowest latent ranks are likely to have higher mortality than the average high school graduates. The downward bias on mortality among the least educated will be the highest when the education-mortality gradient is steep. Because this gradient has steepened over time (Goldring et al., 2016), the downward bias on mortality is higher in 2015 than in 1992, which means that mortality change among the least educated is biased downward when we use this functional form.

Note finally that these other approaches are likely to be increasingly biased when the bin boundary

shifts more over time, or when the desired outcome percentiles are very different from the bin boundaries in the raw data, but the estimates from the MRCB function will not reflect this source of error. One reason that none of these authors focus on the very bottom of the education distribution (or the percentiles approximating high school dropouts) is that the large population change in dropouts (from 20% of the population in 1992 to 10% in 2015) leads to a substantial potential for bias. With our approach, in contrast, the bounds reflect the uncertainty in the estimates and become wider in cases like these where bin boundaries have shifted substantially. Our bounds thus accurately convey the uncertainty due to misalignment between desired outcome percentiles and bin boundaries in the data.

The MRCB function generates a mortality estimate among the least educated that is close to our lower bound mortality estimate. Our results are therefore entirely consistent with Meara et al. (2008), who find that death rates among those with a high school education or less are diverging from those with any college education. We find similar effects for the period up to 2000 studied by Meara, Richards and Cutler, and show that (i) mortality by education continues to diverge from 2000–2015; and (ii) the bottom 10% of whites do particularly badly in both periods.

In contrast, Bound et al. (2015) argue that the composition adjustment effectively erases large mortality increases among non-Hispanic whites in the bottom 25%, though they continue to find average decreases in life expectancy at age 25 among non-Hispanic whites in this education group. These differences can be reconciled with our finding of substantially rising mortality rates among the least educated non-Hispanic whites. First, as noted in this section, the Bound et al. estimates are at the lower bound of mortality change because of the implicit functional form assumption. Second, we find that mortality increases are most severe among the bottom *10%*; extending the interval to the bottom 25% substantially attenuates the estimated mortality change, and makes the functional form bias larger. Third, we focus on middle-age mortality change, because of known problems of age inaccuracy among older ages (Olshansky et al., 2012); trends among individuals aged 70 and older may substantially influence life expectancy and be different from those studied here.

Finally, note that Asher et al. (2019) is a methodological paper which uses the compositional bias in mortality-by-education estimates strictly as an example to explain and motivate a statistical method. It focuses on a specific set of estimates from Case and Deaton (2015; 2017) and studies the characteristics of the compositional bias under different methodological assumptions, but does not present any of the findings reported in this paper.

Figure A3
Spline Approximations to the Empirical Mortality-Income CEF
52-Year-Old Women in 2014

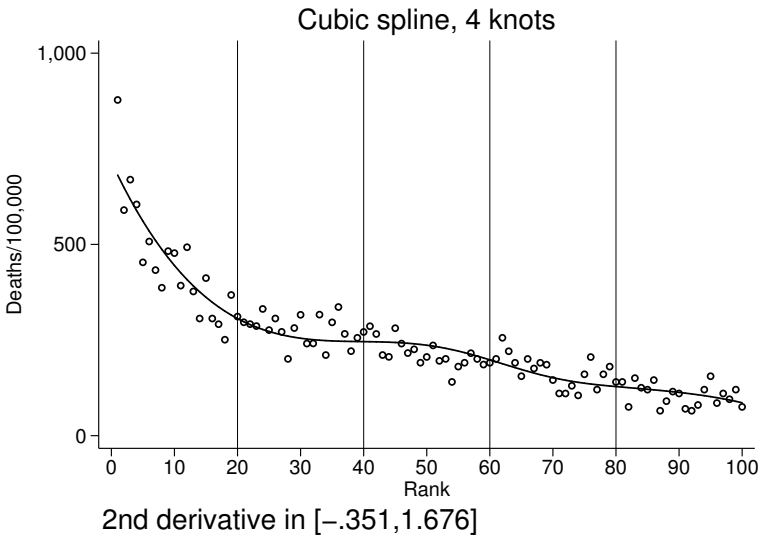


Figure A3 presents estimates of the conditional expectation function of U.S. mortality given income rank, using data from Chetty et al. (2016). The CEF is fitted using a four-knot cubic spline. The function plots the best cubic spline fit to the data series, and the circles plot the underlying data. The text under the graph shows the range of the second derivative across the support of the function.

E Appendix: Robustness Tests

This section reports changes in mortality from 1992–1994 to 2013–2015 calculated under different assumptions and parameters. We focus on the sensitivity of estimates in Figure 3 for non-Hispanic white women; results for other groups are similarly robust to the specifications here.

E.1 Robustness to Alternate Specifications and Assumptions

Percentile Bins Defined in 1992. The main figure was calculated using education percentile bins that approximated those in 2015, a year in which dropouts accounted for percentiles 0–10, high school graduates for percentiles 10–45, individuals with some college for percentiles 45–70, and individuals with a B.A. or higher accounted for the top 30%. In 1992, these four education levels respectively represented percentiles 0-19, 19-59, 59-78 and 78-100. Panel A of Figure E1 shows estimates of mortality change from 1992–2015 using the latter bin boundaries. The broad patterns of mortality change are the same. Mortality changes are slightly smaller in the bottom group, but this is what we would expect given that the bottom group is now defined as the bottom 20% rather than the bottom 10%. The overall divergence of mortality by education is unambiguous.

Ranking within Race and Gender. The body of the paper ranks individuals against members of their own gender. It thus reports, for example, changes in mortality for white women in the bottom 10% of the female education distribution. An alternate approach is to define percentiles within race and gender, and thus to examine mortality changes for white women in the bottom 10% of the white women's education distribution. This approach would be sensible if one's relative position in the own-race socioeconomic distribution was an important factor for mortality. Panel B of Figure E1 presents the main estimates, where education percentiles are defined within race and gender. The pattern of dramatically rising mortality for whites in the bottom 10% remains evident. The total increases in mortality are slightly less under this definition, because white education has increased more than black education over this period, but the change in selection bias is small.

Alternate Bounding Assumptions. The main analysis calculated bounds under the assumptions that (i) expected mortality is weakly monotonically declining in latent educational rank for all groups; and (ii) expected mortality changes sharply only at education ranks that correspond

to major changes in education levels. Note that we permitted the mortality function to experience a discrete jump or slope change of any size when crossing educational boundaries, for example, when moving from a dropout rank to a high school rank. While these assumptions are sensible and consistent with other research and data on the expectation of mortality as a function of education, we show here that loosening these assumptions weakens the precision of the main estimates but does not change any of the substantive conclusions.

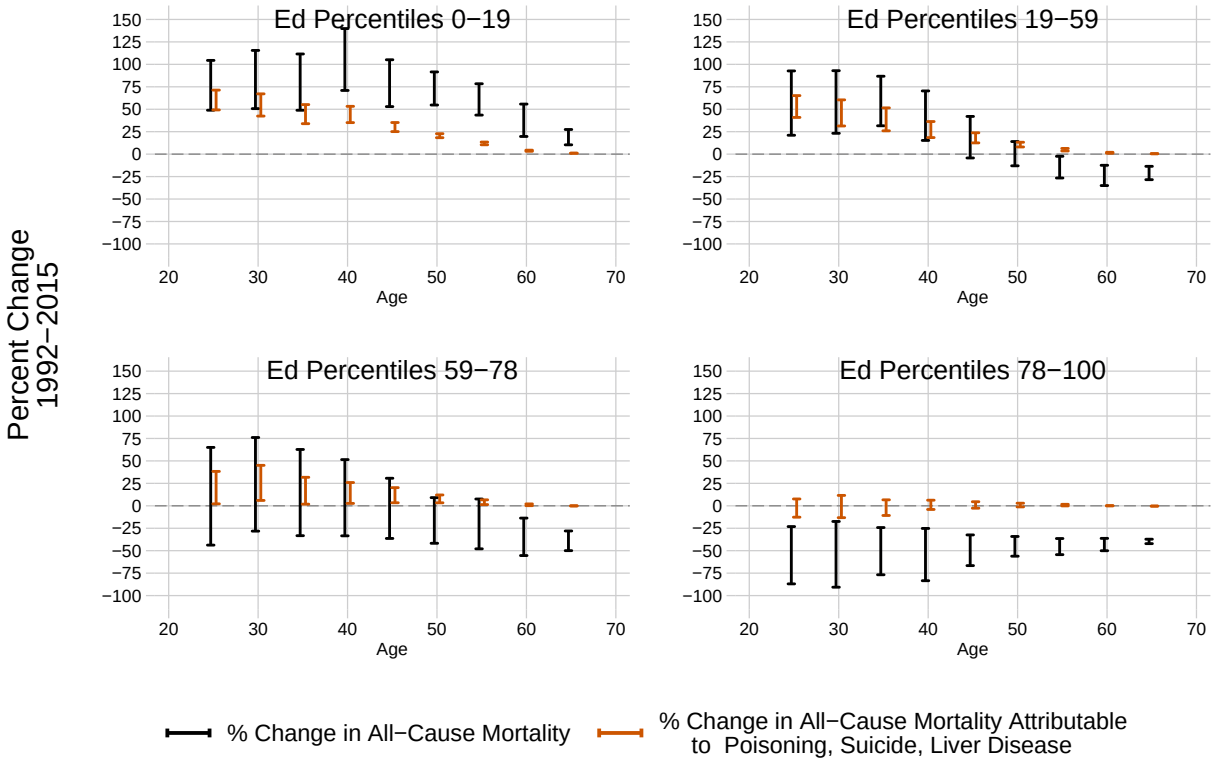
Panel C of Figure E1 presents bounds on mortality change from 1992–2015 when we abandon the monotonicity assumption, and allow mortality to be rising across some education ranks. For graph legibility, we truncate the upper bound on mortality increases when it is above +150%. Panel D of Figure E1 presents analogous bounds when we require monotonicity, but allow the mortality function to have discrete kinks or jumps at any point in the education rank distribution.

The three facts that we have highlighted in the paper remain apparent: (i) mortality gains among the less educated are not keeping up with those among the more educated, and the bottom 10% is particularly lagging; (ii) the increases in mortality among whites in the bottom 10% dwarf the changes in mortality for other groups; and (iii) "deaths of despair" explain less than half of mortality change among middle-aged whites.

Figure E1

Change in non-Hispanic White Female Mortality: Sensitivity Analysis

Panel A: 1992 Percentile Boundaries



Panel B: Within Race-Gender Percentiles

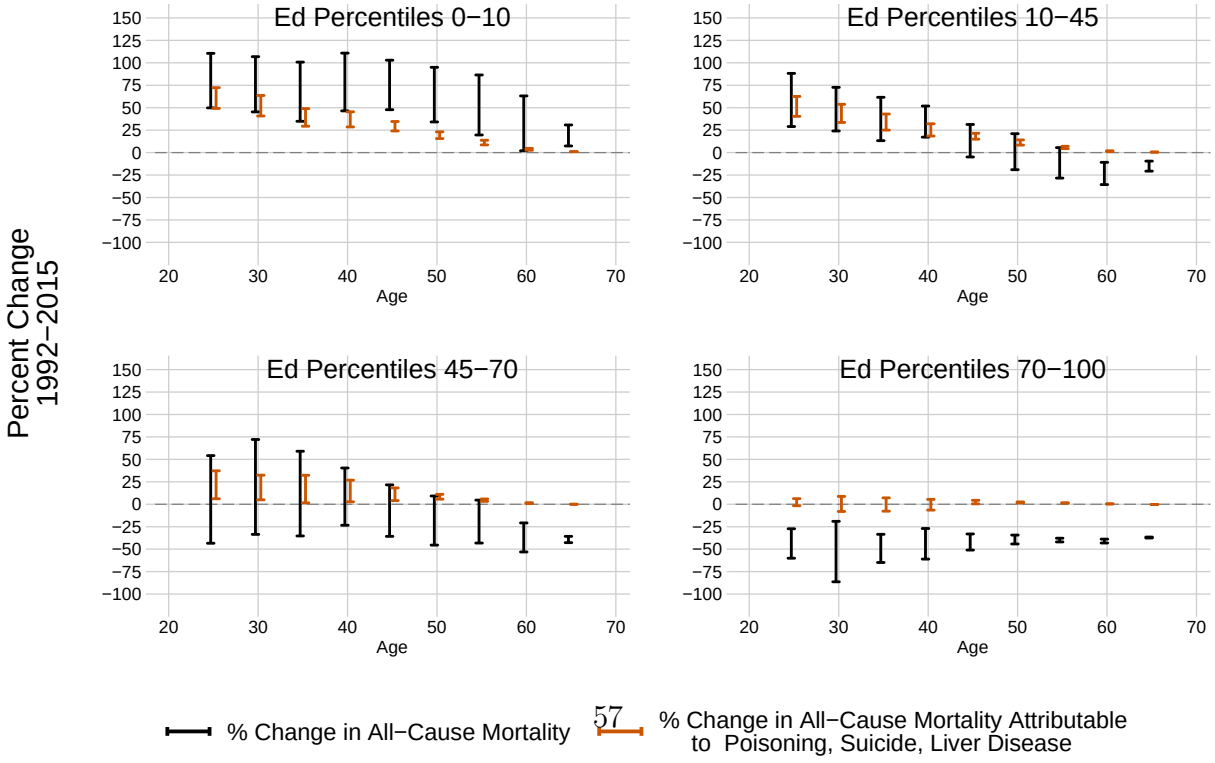
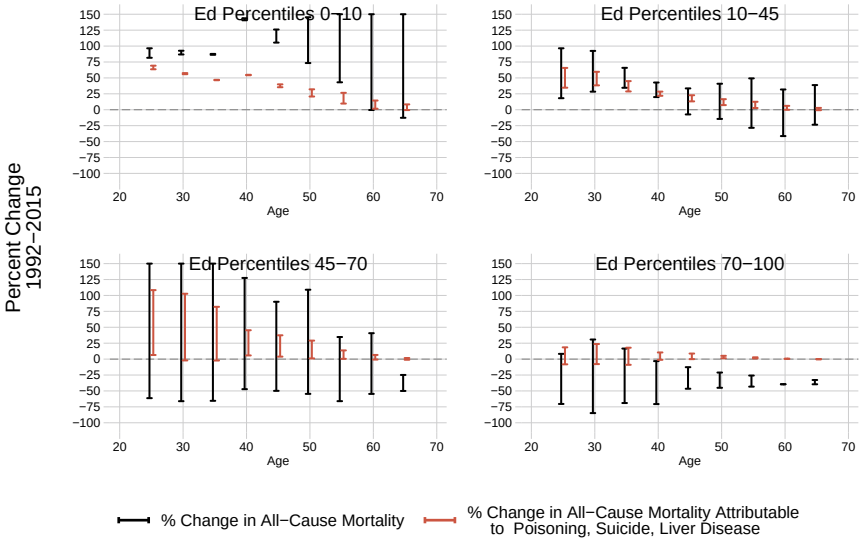


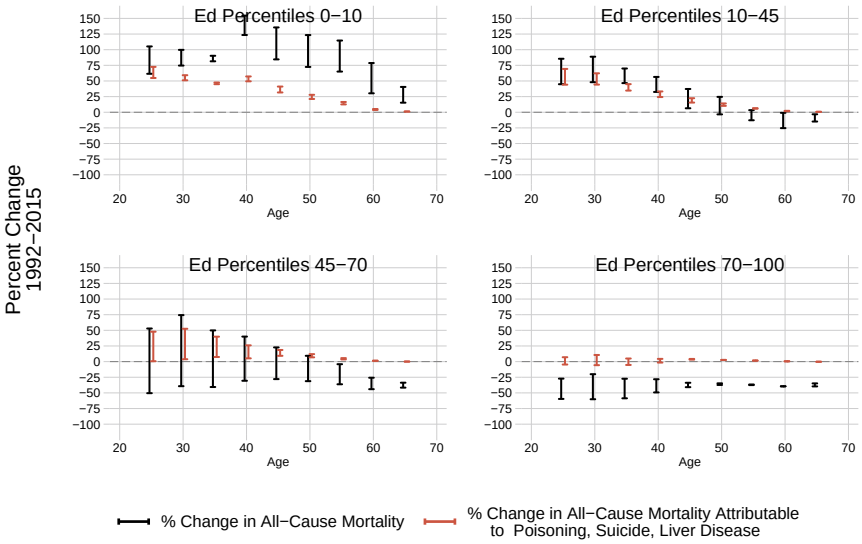
Figure E1

Change in non-Hispanic White Female Mortality: Sensitivity Analysis (Continued)

Panel C: No Monotonicity Assumption



Panel D: No Smoothness Assumption



Note: The figure shows bounds on mortality change from 1992–1994 to 2013–2015, and the contribution of deaths of despair to mortality change, for non-Hispanic whites, by age and percentile education bin, under alternate assumptions from the main body of the paper. The figure is analogous to Panel A of Figure 3, but with different bounding assumptions. Panel A defines education bins boundaries according to education levels in 1992–1994 rather than in 2013–2015. Panel B defines an individual's education percentile according to the individual's rank in the own-race and own-gender education distribution, rather than in the own-gender education distribution. Panel C estimates bounds on mortality, allowing the mortality-education CEF to be non-monotonic. Panel D estimates bounds on mortality without restricting the curvature of the mortality-education CEF. The solid black lines show the bounded set containing the percentage change in the mortality rate from 1992–1994 to 2013–2015 for the given group. The orange lines show the bounded set containing the contribution of deaths of despair to the change in total mortality. In other words, they show how much total mortality would have changed if the rates of all deaths *other* than deaths of despair were unchanged. For graphical legibility, the upper bound on mortality change in Panel C has been topcoded at +150%.

E.2 Changing Racial Composition

This section examines the hypothesis that relative socioeconomic status *within* education bins has changed for blacks relative to whites during the study period. This kind of change could bias our estimates of mortality change, because we assume that the latent education ranks of blacks and whites *within* the bottom 10% (and within other percentile groups) have not changed during the study period. For example, if whites within the bottom 10% were clustered at the top of this percentile bin in 1992 and at the bottom of this bin in 2015, then we would expect their measured mortality to rise even if the underlying mortality-education-rank CEF is unchanged. To be concrete, suppose the mean white woman, conditional on being in the bottom 10% of all women, moved from the 7th percentile to the 3rd percentile. Then comparing average mortality among white women in the bottom 10% could still be subject to selection bias, even in our constant composition estimates, because the average white woman in the bottom 10% would be more negatively selected over time.

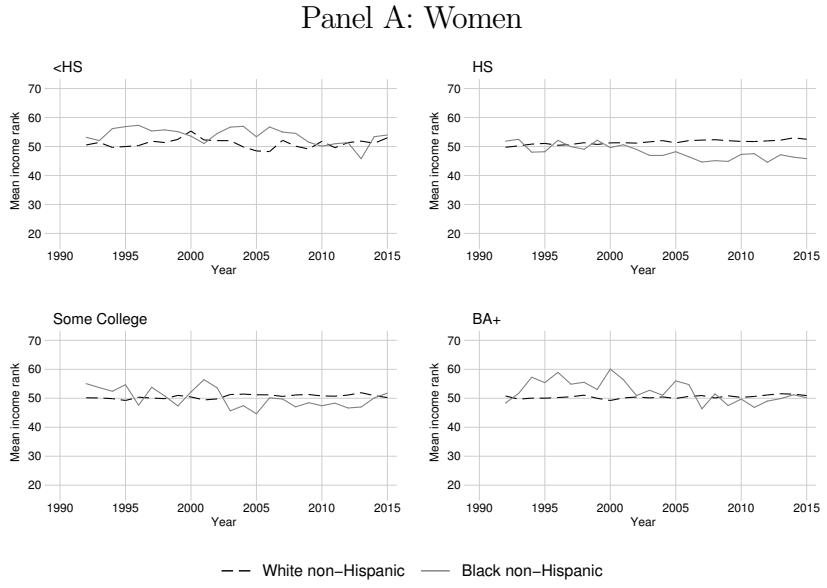
Note that Panel B of Figure E1 already rules this out as a primary explanation for rising white mortality, by showing that mortality is rising for the bottom 10% of whites, not just for whites in the bottom 10% of the national education distribution. Nevertheless, in this section we explore the possibility that the relative status of whites in the bottom 10% of the entire educational distribution has shifted relative to blacks.

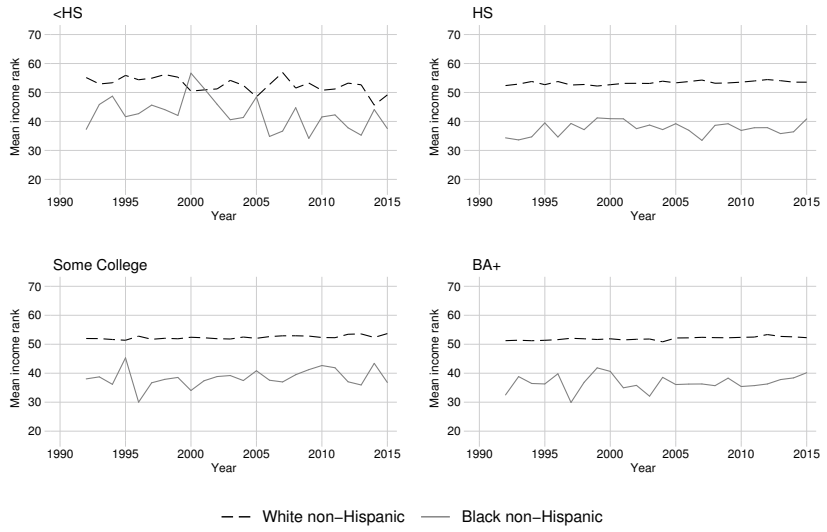
Because education data is interval censored (i.e. we only know that someone is a high school dropout, but we do not observe how close they were to completing high school), we cannot measure the education percentile more precisely. However, we can examine whether the socioeconomic status of white dropouts has changed relative to black dropouts on other measures. We focus on income as measured in the Current Population Survey.

First, we use the granular information on incomes in the CPS to rank all people by income within each gender, age and education bin in each year. We then compute the mean income rank for whites and blacks within each of the four education groups used in the body of the paper. Figure E2 plots the results of this exercise for women and men aged 50–54. The figures show that mean income ranks for blacks and whites, conditional on education level, have remained stable over time. Among women, black and white dropouts have approximately equal income rank throughout the sample period. Among high school graduates, the relative status of whites is increasing relative to that of

blacks, which would bias us *against* finding increases in mortality. Among men, whites have higher average income ranks within every education bin, but their relative advantage is stable over time. Changing relative latent education rank *within* observed education bins therefore cannot explain any of the rise in mortality of white dropouts.

Figure E2
Average Income Ranks for 50–54 year old whites and blacks





Note: The figure shows the average income rank within age, gender and education bins for non-Hispanic white and non-Hispanic black men and women at different education levels. The data source is the Current Population Survey.

E.3 Measurement Error in Race/Ethnicity

A concern that has arisen with the estimation of mortality change in the United States among white and black groups is that reporting patterns for Hispanic identity may have changed over recent decades. The Hispanic population of the U.S. has higher in- and out-migration, making mortality estimates for this group more difficult to measure (Markides and Eschbach, 2005). Hispanics also have considerably lower mortality rates than other groups (Palloni and Arias, 2004; Markides and Eschbach, 2005). If patterns of Hispanic reporting change over time, and especially if they change differentially across the Current Population Survey and the Vital Statistics databases, then estimates of mortality change for non-Hispanics could be biased.

In this section, we consider and rule out two alternative hypotheses for the measured rise in non-Hispanic white mortality. First, survey questions change subtly over time; for example, the Census permitted people to check multiple race boxes starting in 2000 (Currie, 2018). We show that there are no discontinuities in the combined population records for non-Hispanic white or black populations in our sample, suggesting multiple race reporting is not substantially biasing mortality estimates.

Second, populations' attitudes about their own racial/ethnic identity may change over time. The same person might be more likely to report herself as a given race/ethnicity in 2015 than in 1992. Because Hispanics have lower mortality rates, if white Hispanics are more likely to report Hispanic identity over time, this phenomenon would bias the non-Hispanic mortality trend upward. We conduct a bounding exercise that shows that even if an implausibly large number of whites changed their identity to Hispanic, there would still be large mortality gains among less educated non-Hispanic whites.

Finally, average misalignment between Hispanic identification on death records and in census counts would not bias mortality estimates unless the error rate changed between 1992 and 2015. Further, researchers have examined potential misreporting of Hispanic identity on death certificates, and have found that reporting of identity on death certificates is in fact reliable and consistent with census data, with error rates consistently falling below 10%, which is too low to explain the patterns described in this paper (Rosenberg et al., 1999; Arias et al., 2008; Arias et al., 2010; Ruiz et al., 2013).

Changes in survey questions. The option to check more than one race box in the 2000 Census has the potential to change the share of the population that reports as any given race (Currie, 2018). The CPS question on race changed in 2003, while the question in the ACS (which we use only to

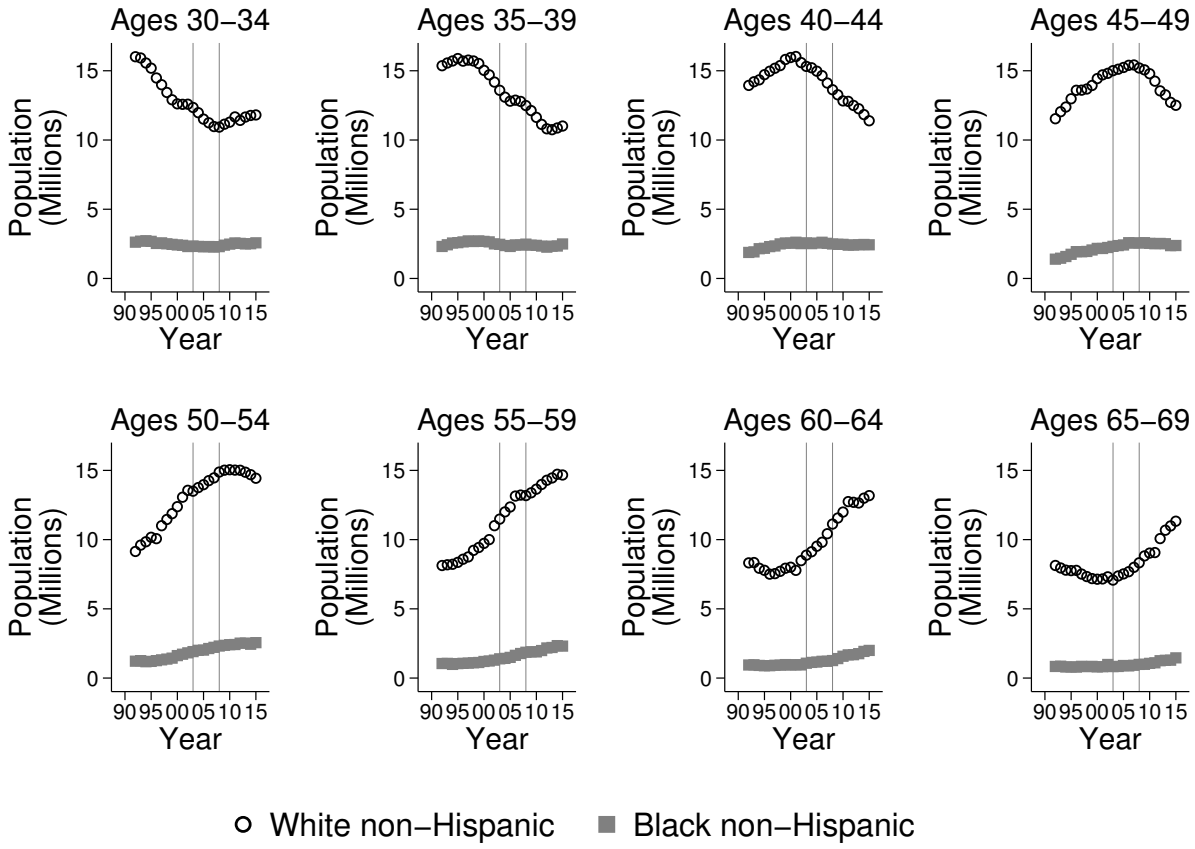
calculate the institutionalized population) changed in 2008. Figure E3 plots the total population in our dataset by age and race. There is clearly no large discontinuous change in the population of any group in either of these years, suggesting that the option to check multiple races in these national surveys cannot explain the secular trend in rising mortality among the least educated white non-Hispanics. Note also that only 5% of white 20-year-olds in the 2010 Census report multiple races, and fewer than 2% of white 50-year-olds (Currie, 2018). This is thus unlikely to be a major concern.

Differences between the Census/CPS and NCHS reporting. Because Hispanics have lower mortality than non-Hispanic whites, Hispanics who report their identity as white will lower the mortality rate among non-Hispanic whites. Measured non-Hispanic white mortality would rise over time if there is an increasing propensity for individuals to report Hispanic ethnicity over time.

Note also that even if there is misreporting of ethnic identity on death certificates on average, it would not affect our findings on mortality *changes* unless the frequency of misreporting changed substantially during the sample period. Specifically, to bias upward mortality changes among non-Hispanic whites, it would have to be the case that Hispanic identity was reported correctly on death certificates in 1992, but substantially underreported in 2015, and that there are no changes in accuracy of reporting in the CPS.

To test the extent to which changes in reporting of Hispanic identity could influence mortality estimates among non-Hispanic white mortality, we simulated misreporting in the data, focusing on non-Hispanic white women aged 50-54. Specifically, we assumed that X% of white Hispanics in 2015 would have reported themselves as non-Hispanic white in 1992. We therefore reassigned X% of white Hispanic deaths to be counted as white non-Hispanic deaths in 2015, and then recalculated bounds on mortality change from 1992–2015. Panels A and B of Figure E4 plot the results of this exercise for non-Hispanic white women and men respectively. Even in the extreme case where 20% of Hispanic women in 2015 would have reported themselves as non-Hispanic white in 1992, we would still detect an increase in mortality of over 400 deaths per 100,000 among the bottom 10%, which would put the lower bound on mortality increase at 54%.

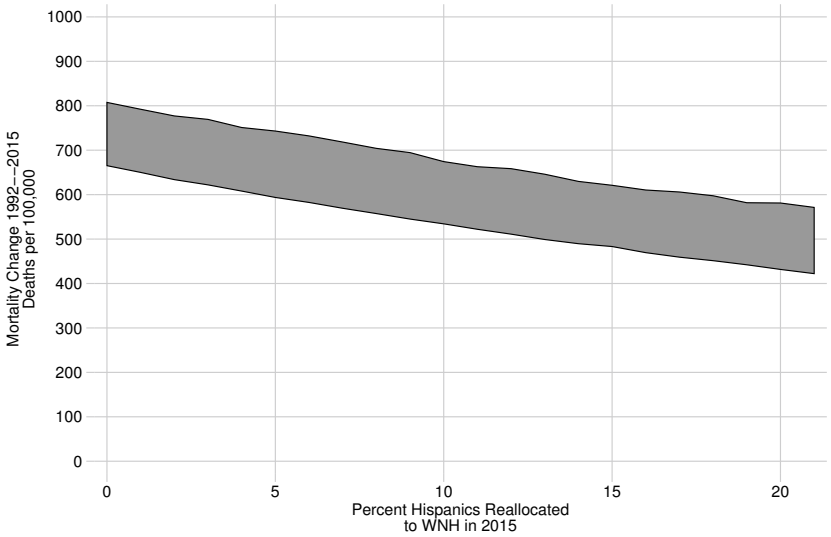
Figure E3
Population Counts by Group



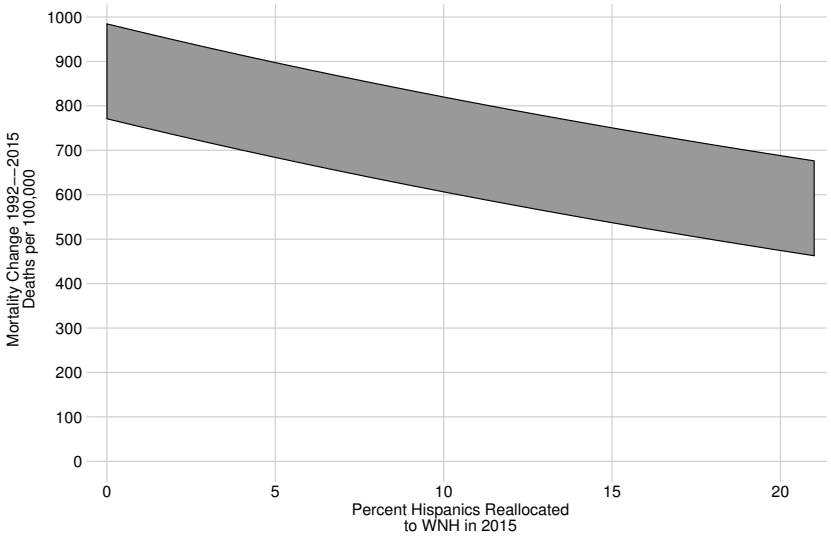
Note: The Figure shows the population in each 5-year age bin of black and white non-Hispanics according the CPS / ACS over the study sample period.

Figure E4
Mortality Changes with Simulated Measurement Error

Panel A: Non-Hispanic White Women Ages 50–54



Panel B: Non-Hispanic White Men Ages 50–54



Note: The figure displays the sensitivity of mortality estimates to measurement error in ethnicity. The figure shows how primary mortality change estimates change if we recode white Hispanic deaths in 2015 to white non-Hispanic deaths, leaving reporting in 1992 and population totals unchanged. The X axis shows the percentage of Hispanic deaths recoded as non-Hispanic deaths under each scenario. The Y axis shows bounds on mortality change from 1992–1994 to 2013–2015 among non-Hispanic white men and women aged 50-54, under each different recoding. Bounds are otherwise calculated as in the body of the paper.

E.4 Analysis of Measurement Error Using Synthetic CPS Cohorts

In this section, we construct synthetic cohorts in the CPS to assess whether misreporting of low education or ethnicity could explain the large mortality increases among the least educated non-Hispanic whites that we find in the body of the paper. If CPS respondents increasingly inflate their level of education (*i.e.* report that they have completed high school when in fact they did not), then the denominator of the mortality rate (the estimated number of high school dropouts) would be increasingly biased downward over time, causing us to overestimate mortality change.

We will put an upper bound on this source of bias by studying the size of a synthetic cohort of dropouts in the CPS. The size of a cohort of white high school dropouts can change over time for five reasons: (i) deaths; (ii) migration; (iii) continuing education; (iv) false reports of continuing education; or (v) some report that they are Hispanic, even though they would not have reported this in the past. In this section, we aim to calculate an upper limit on the share of individuals who are exiting the sample for reporting reasons; this is is also a bound on the combined bias caused by misreporting of education and ethnicity status in the mortality rate.

We measure the death rate in every period, and we assume that net migration of non-Hispanic middle-aged whites is small enough to ignore. We also estimate the number of individuals passing the GED, which is the primary form of continuing education for individuals who did not complete high school. We obtained the number of GED passers from 1992 to 2013 from the GED Testing Service.[15] The number of passers is disaggregated by 5- or 10-year age group; the GED Testing Service also reports the share of passers who are female and the share who are white. The number of passers is not further disaggregated either into single-year age bins or bins describing age * female or age * race. We therefore assume that the number of passers is distributed uniformly across ages within age bins, and that the female and white shares are the same at all ages. We expect that these assumptions will bias downward our estimates of white female passers at higher ages, as women may be more likely than men to delay continuing education due to pregnancy.

Given our estimate of the number of GED passers and our estimate of the mortality rate, we can predict how the size of a synthetic CPS cohort of high school dropouts will evolve over time. Any discrepancy between our predicted cohort size and the actual cohort size will be driven by migration

[15]For a sample report, see the 2009 GED Testing Program Statistical Report, which we downloaded from https://files.eric.ed.gov/fulltext/ED512301.pdf.

(which we expect is small), continuing education in a form other than the GED, false reporting of continuing education, or change in reporting of ethnicity. The discrepancy is therefore an upper bound on the mismeasurement of mortality due to individuals exiting the sample due to misleading reporting of education or ethnicity.

Figure E5 presents the results. The top left panel shows the analysis for non-Hispanic white female dropouts. The gray squares show the CPS population of white female dropouts in the 1950–54 birth cohort, approximately the middle 5-year birth cohort in our study. Their population falls over time due to the five factors above. The gray line is a linear trend fit to the gray points to eliminate year-on-year noise. The dashed blue line shows how the size of this cohort would have evolved over time from mortality alone, beginning at the CPS trend line in 1992, based on our estimates from the NCHS. The red line shows how this cohort would have evolved when we count deaths and GED completions. In 2015, the gap between the linearized CPS cohort size and the predicted cohort size is 6.2%. Assuming that migration in this cohort is small, this gap is an upper bound on the error in our population count that arises from false reporting of high school completion or changing in reporting of Hispanic ethnicity.

The remaining panels of the figure show the same result for white men, and for white women and men in the 1960–64 birth cohorts. Continuing education explains more of the change in cohort size for the younger 1960–64 birth cohort because individuals are more likely to complete GEDs at younger ages. The potential biases for men are smaller than for women. The potential bias is highest for women in the 1960–64 birth cohort, with a discrepancy of 23.8% between the CPS population and our predicted population. One factor which we believe could explain this discrepancy is the possibility that women are more likely to take the GED later in life than men because of early pregnancies. Our GED passing numbers did not report age profiles for men and women separately, so we had to assume that men and women are equally likely to take the exam in their thirties and forties. If men are more likely to take the exam in their teens and twenties and women are more likely to take it later in life, then our predicted measures would be even closer to the true series for both men and for women.

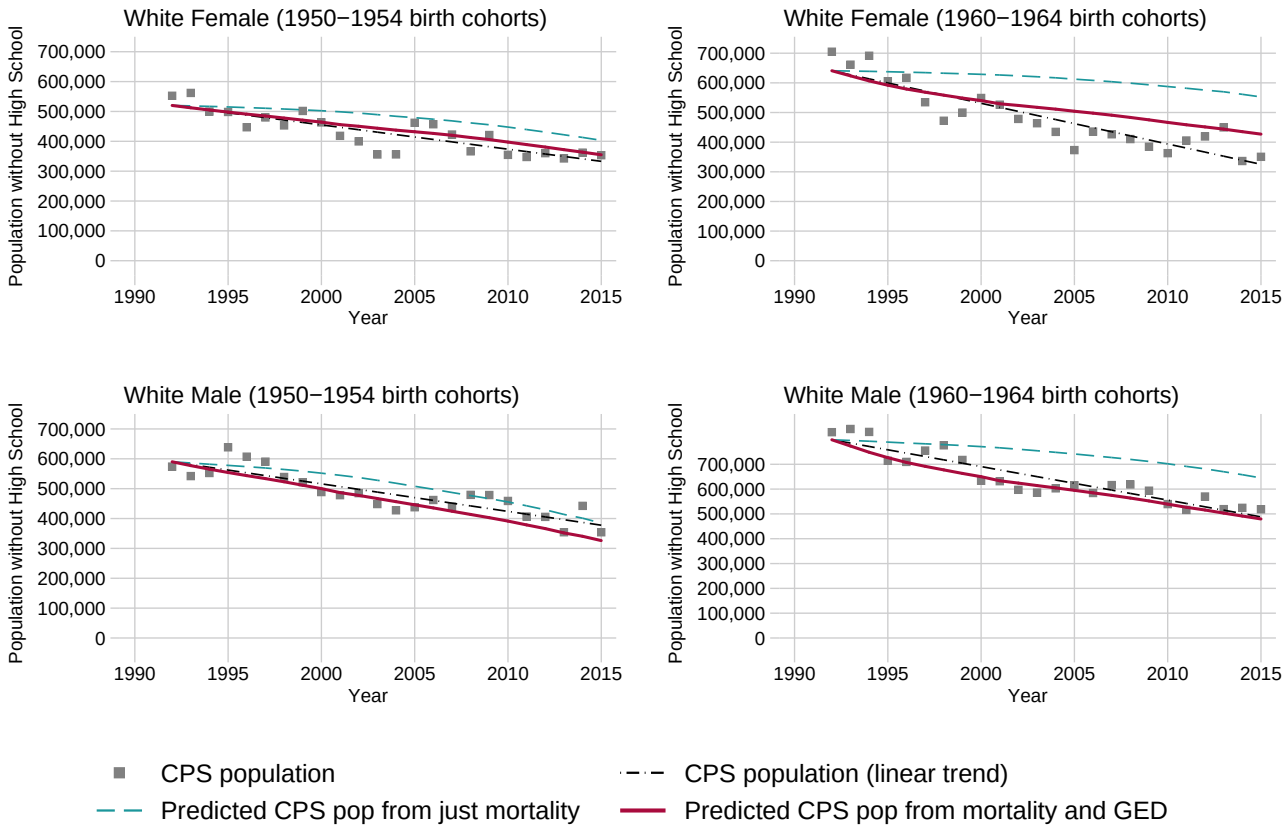
Nevertheless, for the sake of argument, we can consider how our mortality change measures would change if our mortality rates in 2015 are biased upward by the worst case estimate of 23.8%. In the paper, we report that 50–54-year-old women (corresponding to the 1960–64 birth cohort in 2015) in the least educated 10% experienced mortality increases of 79–116%. If we underestimated the population of white female dropouts in this birth cohort by 23.8% in 2015, the corrected mortality change

would be an increase of 45–74%. While this number is a lot smaller, it is still considerably higher than the mortality change estimate in the next education percentile group— among similarly-aged women in the 10th to 45th percentiles, we estimate mortality increases between 0 and 24%.

Therefore, even if we make worst case assumptions about bias due to misreporting of education, the overall findings of our paper are upheld. We also believe these worst case assumptions are implausible for three reasons. First, the calculation above uses the discrepancy for the 1960–64 birth cohort, which is among the largest in our sample. If we used the population discrepancy from the 1950–54 birth cohort, the bias would be considerably smaller. Second, as noted above, women may be disproportionately likely to complete the GED at higher ages. Last, there are other mechanisms for individuals to obtain continuing education after dropping out of high school, which would explain some of the remaining bias.

Finally, Figure E6 shows similar graphs documenting the change in the size of synthetic CPS cohorts of white high school completers. For these groups, we can only predict population change due to mortality, because we could not obtain population counts of the number of whites obtaining any sort of 2- or 4-year degree by year and age. Nevertheless, even without counting continuing education, the potential discrepancies are very small, and it is plausible that continuing education can explain it entirely. We therefore find it doubtful that false reporting of education in the CPS is driving the main results of substantial mortality increases among the least educated whites.

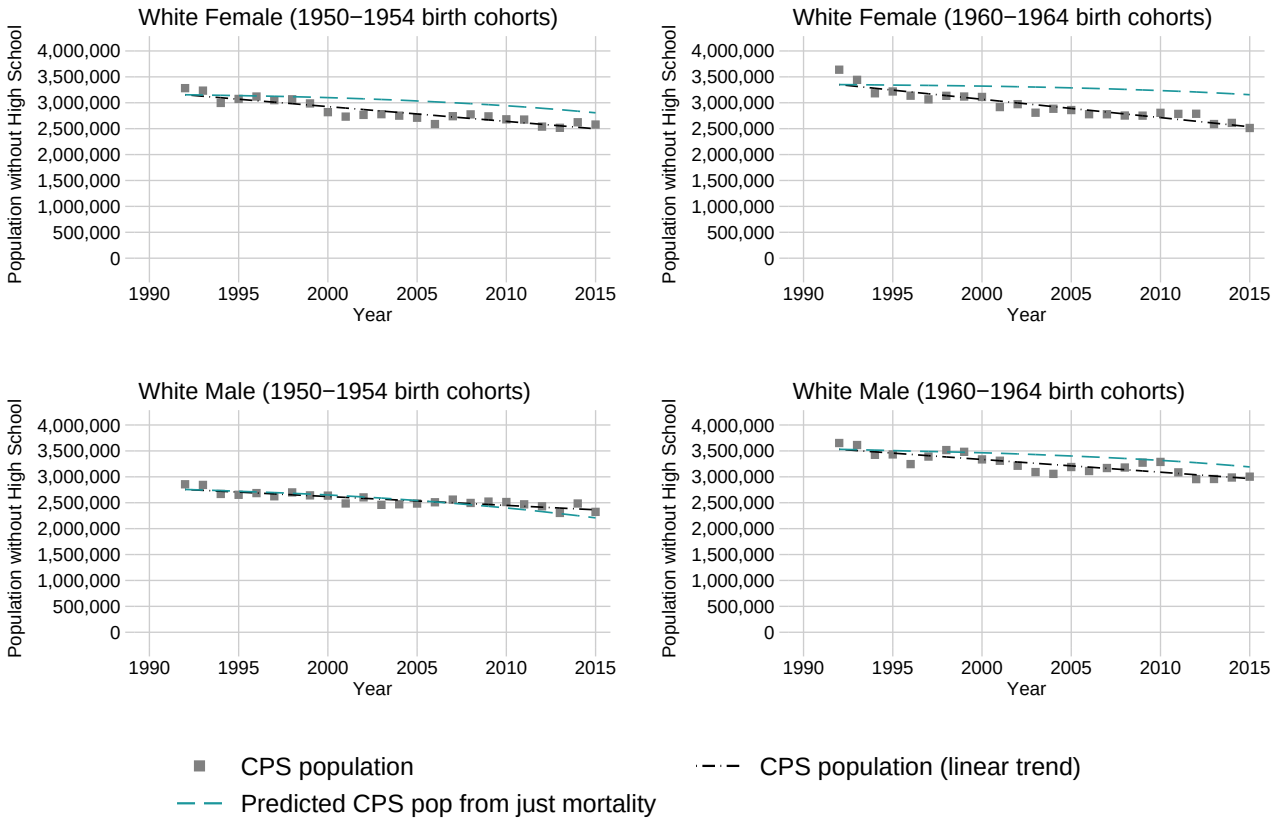
Figure E5
Bounding Measurement Error in CPS Dropout Counts



The figure displays the population in selected cohorts of CPS dropouts. These are compared with the predicted population based on measured mortality and GED completion. The gray points show the counts of members of each gender/education group in each round of the CPS. The dot-dash black line is a linear trend fit to the gray points to eliminate year-on-year noise. The dashed blue lines begins at the CPS trend line in 1992, and shows how the CPS population would have evolved from mortality alone. The solid red line shows how the CPS population would have evolved from mortality and GED completion only.

Figure E6

Bounding Measurement Error in CPS High School Completer Counts



The figure displays the population in selected cohorts of CPS high school completers. These are compared with the predicted population based on measured mortality. The gray points show the counts of members of each gender/education group in each round of the CPS. The dot-dash black line is a linear trend fit to the gray points to eliminate year-on-year noise. The dashed blue lines begins at the CPS trend line in 1992, and shows how the CPS population would have evolved from mortality alone.

E.5 Comparison with National Health Interview Survey

In this section, we use an alternate data source to validate the findings in the body of the paper. We use the National Health Interview Survey (NHIS), which is an annual repeated cross-section survey of about 35,000 households and 87,000 individuals. Mortality rates can be calculated directly from the NHIS, because NHIS records are intermittently linked to death certificate records from the National Death Index. We can therefore estimate a population subgroup mortality rate as the share of individuals who are deceased in a given followup period.

Because mortality rates and education/ethnicity are all measured for the same individuals, mortality estimates from the NHIS suffer from considerably less bias from misreporting of education. For instance, if some individuals without a high school education report that they have a high school education, both their deaths and their population will be counted among the high school group. This may create a small bias if mortality is correlated with misreporting, but it will be considerably less bias than if their deaths are counted in the dropout group and their population is counted in the high school completion group. This said, NHIS-based measures of mortality slightly underestimate aggregate mortality relative to vital statistics data, especially for older white women (Ingram et al., 2008).

We obtained public NHIS data with death record linkages from the web site of the National Bureau of Economic Research, which provided data for the years 1997 to 2011. We used data from 1997 to 2009 so that we could calculate mortality rates with at least two years of followup.[16] We aggregated results across all ages using the standardized U.S. population distribution, as in Section 4.3. Because our aim in this section is to validate the raw mortality estimates from the NCHS, we present raw mortality change for education levels, rather than bounding the mortality change in constant education percentiles.

Figure E7 below compares estimates of annualized mortality change in the NHIS vs. our estimates from the NCHS for the four key groups in our study: non-Hispanic white female dropouts and high school completers, and non-Hispanic white male dropouts and high school completers. Mortality change in the NHIS is calculated from 1997 to the last period in which the n-year mortality rate can be calculated. The red points show the NHIS mortality estimates with 95% confidence intervals. Even with the 6-year followup period, the NHIS sample is too small to precisely estimate mortality

[16]The sample of deaths in the year following the NHIS survey is extremely small. In some of our subgroups, there were zero deaths reported in the shortest followup periods.

change over the sample period. Most point estimates are very close to our mortality change estimates from NCHS data (indicated by the dashed green line), but in many cases the NHIS confidence interval includes both zero and our measure. The point estimates from NHIS suggest that white female high school completers experienced *greater* mortality increase than white female high school dropouts (contrary to our findings), but the confidence intervals show that this statement cannot be made with any precision, and the opposite conclusion is also consistent with the NHIS data. In almost all cases, our NCHS measure of mortality change is within one standard error of the NHIS estimate. Compared with our results, NHIS calculates slightly higher mortality increases for white male dropouts and slightly lower changes for white women. Our point estimates for the number of deaths in NCHS and NHIS are also very similar.

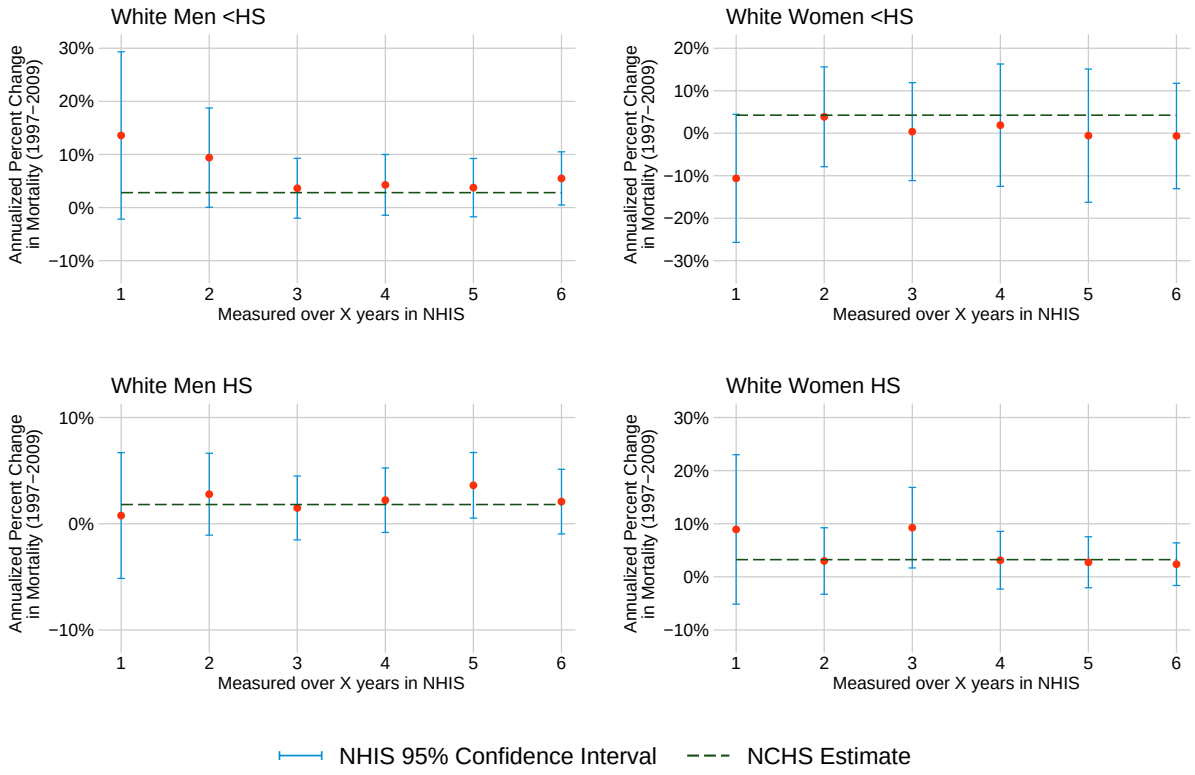
NHIS estimates of mortality are imprecise because the number of middle-aged white dropouts in the sample who die is very small. We can obtain more precise estimates of health status, which is reported by all respondents. NHIS respondents are asked to report their perception of their own health on a five point scale, where one reflects very good health, and five reflects very bad health. In Figure E8, we show the *annualized* change in self-reported health status from 1997 to 2009 at each age and education level. We use a lowess smoother to estimate the annualized change in self-reported health status at each age and education level.

The left panel shows the result for non-Hispanic white women. At all ages, dropouts have experienced worse health declines than members of any other group. Among 40–60-year-old women, self-reported health status has gotten 0.025 points worse *per year*, or 0.3 points worse on a 5 point scale from 1997 to 2009. The age pattern of the health decline closely matches our mortality results in Figure 3, with the greatest divergence between dropouts and high school graduates occurring between ages 40 and 60. We observe less change in the education-health gradient among men, but dropouts suffer the worst deterioration in health between ages 50–70, corresponding exactly to the ages where we document the greatest differential increases in mortality among the bottom 10% in Figure 4.

In conclusion, measures of mortality and self-reported health in the NHIS are broadly consistent with our main findings of increased mortality among middle-aged white men and women in the least educated 10%. These NHIS measures are less precise than the vital statistics mortality data, but they do not suffer from any division bias that could be caused by changes in how individuals respond to questions about their education or ethnicity over time.

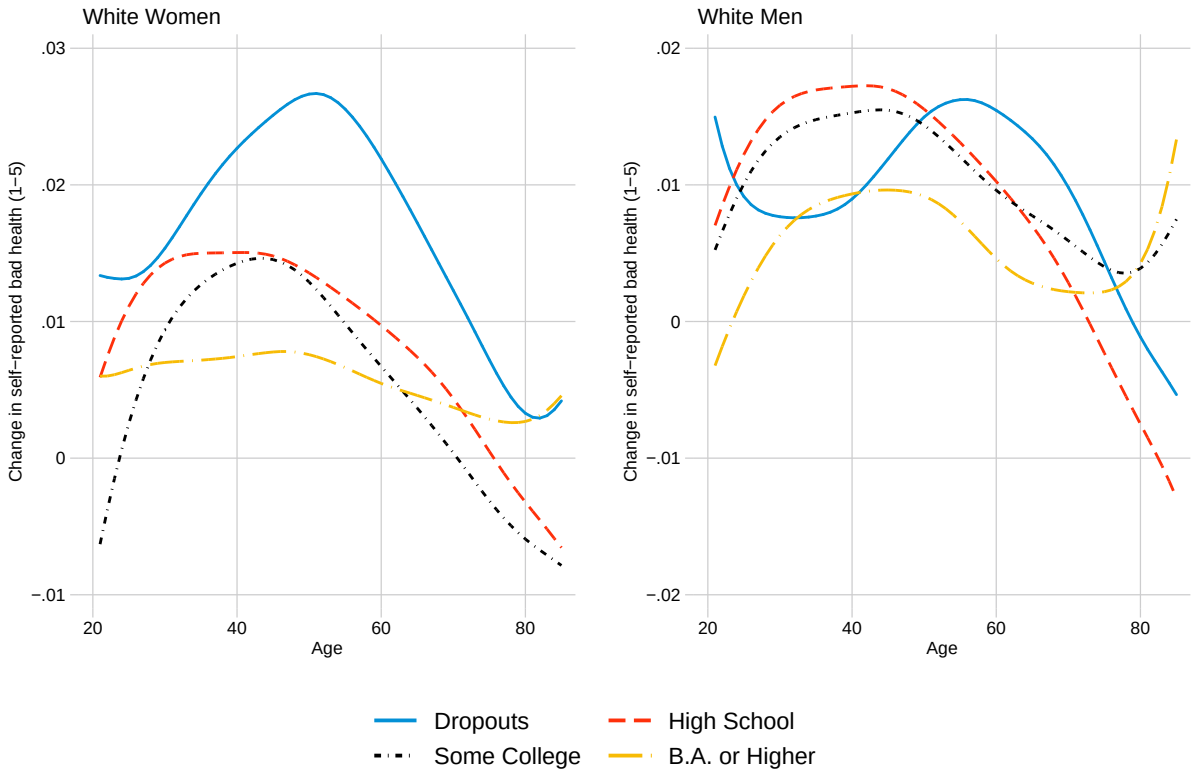
Figure E7

Annualized Mortality Change Estimates from NHIS and from NCHS (1997-2009)



The figure compares estimates of annualized mortality change in the National Health Interview Survey (NHIS) with Vital Statistics data from the NCHS, used in the body of the paper. The annualized change in the mortality rate from 1997–2009 according to NHCS (Vital Statistics data) is indicated by the dashed green line. The six different points for each panel reflect annualized mortality changes in the NHIS based on measurement of 1-year mortality, 2-year mortality, 3-year mortality, and so on. 1-year and 2-year mortality change are measured in NHIS from 1997–2009. 3-year mortality change is reported from 1997–2008, because we do not have deaths after 2011 in our data. 4-year mortality change is reported from 1997–2007, and so on. These different estimates are nevertheless broadly comparable because the mortality change estimates are annualized. Standard errors are calculated for the NHIS using NHIS sample weights.

Figure E8
Change in Self-Reported Health Status (NHIS, 1997-2009)



The figure shows a lowess fit to the annualized change in self-reported health from 1997 to 2009 at each age, according the NHIS. Self-reported health status is on a five point scale, where one represents the best health and five represents the worst. We calculate annualized change for each age cohort / education group by regressing self-reported health status on a year variable. We then plot the year coefficients for each age/education group using a lowess smoother to minimize noise across single-year age cohorts. The series therefore shows the predicted change in self-reported health for an individual in a given age, gender and education group.

E.6 Measurement Error in Education

This subsection explores the possibility that measurement error in education biases the main finding of rising mortality among the least educated whites. High school graduation rates on death certificates are thought to be inflated on average, though higher education levels appear to be reported accurately (Sorlie and Johnson, 1996). There is no evidence that this bias has changed during the sample period, but if overreporting of high school completion on death certificates (but not in the CPS) were to decline over time, it would cause mortality change among high school dropouts to biased upward.

For measurement error to explain the rising mortality among dropouts described in this paper, it would need to be the case: (i) that the measurement error in education changes substantially during the sample period; (ii) that measurement error declines considerably for deaths *other* than deaths of despair, but changes considerably less for deaths of despair; and (iii) that measurement error has declined dramatically for non-Hispanic whites, but has changed only minimally for blacks. Misreporting rates would have to differ substantially across age groups as well—for example, we find that dropouts account for nearly all mortality gain among 50–54-year-olds white women, but that rising mortality is equally distributed among dropouts and high school graduates among 25–29-year-old white women. Note also that other researchers have noted rising health disparities between dropouts and high school graduates, in datasets where misreporting of education is unlikely to be a concern (Montez et al., 2011).

We show here that even if one treats the distinction between dropouts and high school graduates as unreliable, the central finding of rising mortality among the least educated remains clear. To show this, we combine dropouts and high school graduates into a single education group, and we estimate mortality change for three constant education percentile bins: (i) percentiles 0-45 (corresponding approximately to dropouts *and* high school graduates in 2015; (ii) percentiles 45-70; and (iii) percentiles 70-100. Figure E9 shows estimates of mortality change from 1992–2015 for these three groups. We continue to find a dramatic rise in mortality among whites at the bottom of education distribution, though we have defined the bottom more broadly here. When we combine high school dropouts with high school completers, we find that mortality among the bottom 45% of the education distribution rises over 50% for white women below 40, rises 25-50% for women 45-54, and is flat at older ages. Mortality among white men in the bottom 45% is rising for age groups below 55.

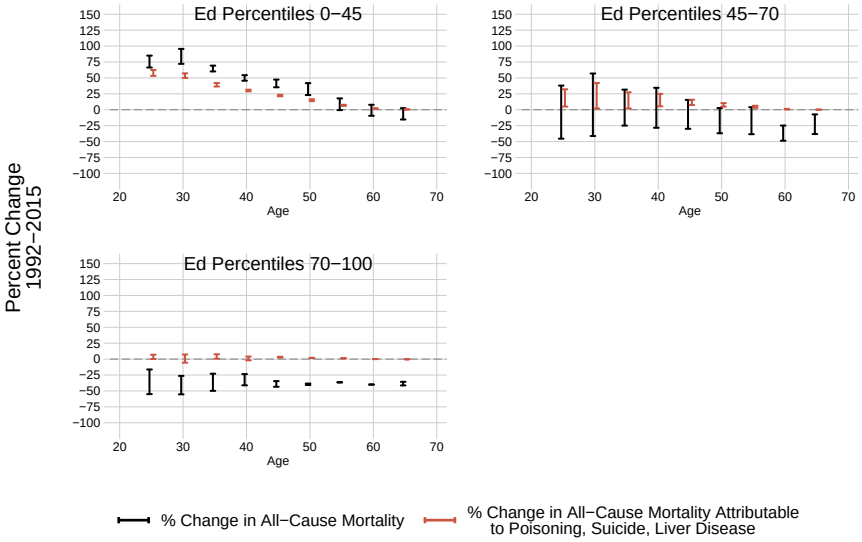
We therefore find significant increases in mortality disparities by education even when we pool high

school graduates and high school dropouts. However, by ignoring the distinction between dropouts and high school graduates, we miss the important difference in causes of death noted in Figure 3—mortality increases outside of the very bottom of the distribution are driven primarily by deaths of despair, but mortality increases in the bottom 10% are driven by a multitude of causes. Absent evidence that measurement error in death records has changed substantially during the sample period, rising mortality among the least educated 10% of the population should be taken seriously.

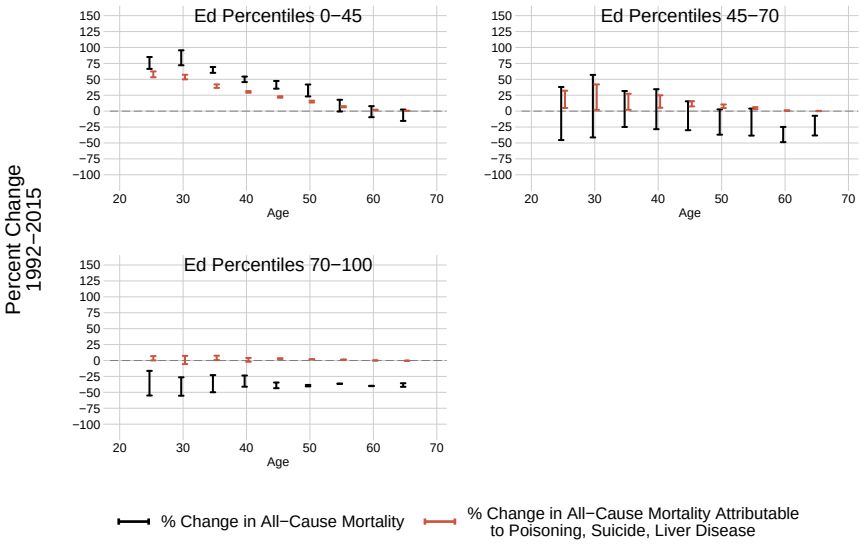
Figure E9

Change in non-Hispanic White Mortality: 3 Education Groups

Panel A: Non-Hispanic White Men



Panel B: Non-Hispanic White Women



Note: The graph shows changes in mortality by age, sex, race, and constant percentile education bin. The figure is analogous to Figures 3 and 3, but with the bottom two education categories (percentiles 0–10 and 10–45) pooled into a single category covering percentiles 0–45. The solid black lines show the bounded set containing the percentage change in the mortality rate from 1992–1994 to 2013–2015 for the given group. The orange lines show the bounded set containing the contribution of deaths of despair to the change in total mortality. In other words, they show how much total mortality would have changed if the rates of all deaths *other* than deaths of despair were unchanged. Bounds are computed using the set identification methods in Asher et al. (2019), described in Section 3. Education data were compiled from the American Communities Survey and Current Population Survey, and death records and codes were obtained from the National Center for Health Statistics.