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# *Mortality and Morbidity in the 21st Century*

**ABSTRACT** Building on our earlier research (Case and Deaton 2015), we find that mortality and morbidity among white non-Hispanic Americans in midlife since the turn of the century continued to climb through 2015. Additional increases in drug overdoses, suicides, and alcohol-related liver mortality—particularly among those with a high school degree or less—are responsible for an overall increase in all-cause mortality among whites. We find marked differences in mortality by race and education, with mortality among white non-Hispanics (males and females) *rising* for those without a college degree, and *falling* for those with a college degree. In contrast, mortality rates among blacks and Hispanics have continued to fall, irrespective of educational attainment. Mortality rates in comparably rich countries have continued their premillennial fall at the rates that used to characterize the United States. Contemporaneous levels of resources—particularly slowly growing, stagnant, and even declining incomes—cannot provide a comprehensive explanation for poor mortality outcomes. We propose a preliminary but plausible story in which *cumulative disadvantage* from one birth cohort to the next—in the labor market, in marriage and child outcomes, and in health—is triggered by progressively worsening labor market opportunities at the time of entry for whites with low levels of education. This account, which fits much of the data, has the profoundly negative implication that policies—even ones that successfully improve earnings

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and jobs, or redistribute income—will take many years to reverse the increase in mortality and morbidity, and that those in midlife now are likely to do worse in old age than the current elderly. This is in contrast to accounts in which resources affect health contemporaneously, so that those in midlife now can expect to do better in old age as they receive Social Security and Medicare. None of this, however, implies that there are no policy levers to be pulled. For instance, reducing the overprescription of opioids should be an obvious target for policymakers.

Around the turn the century, after decades of improvement, all-cause mortality rates among white non-Hispanic (WNH) men and women in middle age stopped falling in the United States, and began to rise (Case and Deaton 2015). Although midlife mortality continued to fall in other rich countries, and in other racial and ethnic groups in the United States, mortality rates for WNHs age 45–54 increased from 1998 through 2013. Mortality declines from the two biggest killers in middle age—cancer and heart disease—were offset by marked increases in drug overdoses, suicides, and alcohol-related liver mortality in this period. By 2014, rising mortality in midlife, led by these "deaths of despair," was large enough to offset mortality gains for children and the elderly (Kochanek, Arias, and Bastian 2016), leading to a decline in life expectancy at birth among WNHs between 2013 and 2014 (Arias 2016), and a decline in overall life expectancy at birth in the United States between 2014 and 2015 (Xu and others 2016). Mortality increases for whites in midlife were paralleled by morbidity increases, including deteriorations in self-reported physical and mental health, and rising reports of chronic pain.

Many explanations have been proposed for these increases in mortality and morbidity. Here, we examine economic, cultural and social correlates using current and historical data from the United States and Europe. This is a daunting task, whose completion will take many years; this current paper is necessarily exploratory, and is mostly concerned with the description and interpretation of the relevant data. We begin, in section I, by updating and expanding our original analysis of mortality and morbidity. Section II discusses the most obvious explanation, in which mortality is linked to resources, especially family incomes. Section III presents a preliminary but plausible account of what is happening; according to this, deaths of despair come from a long-standing process of cumulative disadvantage for those with less than a college degree. The story is rooted in the labor market, but involves many aspects of life, including marriage, child

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rearing, and religion. Although we do not see the supply of opioids as the fundamental factor, the prescription of opioids for chronic pain added fuel to the flames, making the epidemic much worse than it otherwise would have been. If our overall account is correct, the epidemic will not be easily or quickly reversed by policy; nor can those in midlife today be expected to do as well after age 65 as the current elderly. This does not mean that nothing can be done. Controlling opioids is an obvious priority, as is trying to counter the longer-term negative effects of a poor labor market on marriage and child rearing, perhaps through a better safety net for mothers with children that would make them less dependent on unstable partnerships in an increasingly difficult labor market.

PRELIMINARIES First, a few words about methods. Our earlier paper (Case and Deaton 2015) simply reported a set of facts—increases in mortality and morbidity—that were both surprising and disturbing. The causes of death underlying the mortality increases were documented, which identified the immediate causes but did little to explore underlying factors. We are still far from a smoking gun or a fully developed model, though we make a start in section III. Instead, our method here is to explore and expand the facts in a range of dimensions, by race and ethnicity, by education, by sex, by trends over time, and by comparisons between the United States and other rich countries. Descriptive work of this kind raises many new facts that often suggest a differential diagnosis, that some particular explanation cannot be universally correct because it works in one place but not another, either across the United States or between the United States and other countries. At the same time, our descriptions uncover new facts that need to be explained and reconciled.

Two measures are commonly used to document current mortality in a population: *life expectancy* and *age-specific mortality*. Although these measures are related, and are sometimes even confused—many reports on Case and Deaton (2015) incorrectly claimed that we had shown that life expectancy had fallen—they are different, and the distinction between them is important. Life expectancy at any given age is an index of mortality rates beyond that age, and is perhaps the more commonly used measure.[1] Life expectancy at age $a$ is a measure of the number of years a hypothetical person could be expected to live beyond $a$ if current age-specific mortality rates continue into the future; it is a function of mortality rates alone, and does not depend on the age structure of the population. Life expectancy,

1. For recent examples, see Chetty and others (2016), Currie and Schwandt (2016), and Arias (2016).

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without qualification, refers to life expectancy at birth (age zero), and is the number most often quoted; however, when mortality rates at different ages move in different directions, life expectancy trends can also differ by age. The calculation of life expectancy attaches to each possible age of death the probability of surviving to that age and then dying, using today's survival rates. Because early mortality rates enter all future survival probabilities, life expectancy is more sensitive to changes in mortality rates the earlier in life these occur; the often-used measure of life expectancy at birth is much more sensitive to saving a child than saving someone in midlife or old age, and changes in life expectancy can mask offsetting changes occurring in earlier or later life. In our context, where mortality rates are rising in midlife but are falling among the elderly and among children, life expectancy at birth will respond only slowly—if at all. If middle-aged mortality is regarded as an indicator of some pathology, whether economic or social—the canary in the coal mine—or as an indicator of economic success or failure (Sen 1998), life expectancy is likely to be a poor and insensitive indicator. The focus of our analysis is therefore not life expectancy but age-specific mortality, with rates defined as the number of deaths in a population of a given age per 100,000 people at risk.

In Case and Deaton (2015) we reported annual mortality results for WNH men and women (together) age 45–54 in the years between 1990 and 2013. In this paper, we present a more complete picture of midlife mortality—by sex and education group, over the full age range of midlife, using shorter age windows, over time, by cause, and by small geographic areas. We use data on mortality and morbidity from the United States and other countries that belong to the Organization for Economic Cooperation and Development, as well as data on economic and social outcomes, such as earnings, income, labor force participation, and marital status.

We are much concerned with education, and work with three educational groups: those with a high school degree or less, those with some college but no bachelor's degree, and those with a bachelor's degree or more. Among WNHs age 45–54, the share of each education group in the population has seen little change since the early 1990s, with those with no more than a high school degree making up approximately 40 percent; those with some college, 30 percent; and those with a bachelor's degree or more, 30 percent. We do *not* focus on those with *less than* a high school degree, a group that has grown markedly smaller over time, and is likely to be increasingly negatively selected on health. Whether or how education causes better health is a long-unsettled question on which we take no position, but we show health outcomes by education because they suggest likely explanations.

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For the midlife group, the unchanging educational composition since the mid-1990s rules out one explanation—that the less-educated group is doing worse because of selection, as could be the case if we had worked with high school dropouts. When we examine other age, ethnic, or racial groups, or midlife WNHs in periods before the mid-1990s, the underlying educational compositions are not constant, and selection into education must be considered as an explanation for the evidence. More generally, we note the obvious point that people with more or less education differ in many ways, so there can be no inference from our results that less educated people would have had the same health outcomes as more educated people if they had somehow been "dosed" with more years of schooling.

Our data on mortality rates come from the U.S. Centers for Disease Control and Prevention's CDC WONDER website (https://wonder.cdc.gov/wonder/help/ucd.html). Mortality by education requires special calculation, and full details of our sources and procedures are laid out in the online appendix.[2]

Early commentary on our work focused on our lack of age adjustment within the age group 45–54 (Gelman and Auerbach 2016). Indeed, the average age of WNHs age 45–54 increased by half a year between 1990 and 2015, so that part of the mortality increase we documented is attributable to this aging. Andrew Gelman and Jonathan Auerbach's (2016) age-adjusted mortality rates for WNHs in the 45–54 age group show that the increase in all-cause mortality is larger for women, a result we have confirmed on the data to 2015 (36 per 100,000 increase for women, and 9 per 100,000 increase for men between 1998 and 2015, single-year age-adjusted using 2010 as the base year, with little variation in the increases when we use different base years). In the current analysis, we work primarily with five-year age groups, and we have checked that age adjustment makes essentially no difference to our results with these groups; for example, for U.S. WNHs age 50–54, average age increased by only 0.09 year (33 days) from 1990 to 2015.

Age adjustment can be avoided by working with mortality by individual year of age, though the resulting volume of material can make presentation problematic. In the online appendix, we present selected results by single year of age, which can be compared with the results given in the main text. We discuss the separate experiences of men and women in some detail below; unless there is indication otherwise, the results apply to men and women together.

2. The online appendixes for this and all other papers in this volume may be found at the *Brookings Papers* web page, www.brookings.edu/bpea, under "Past BPEA Editions."

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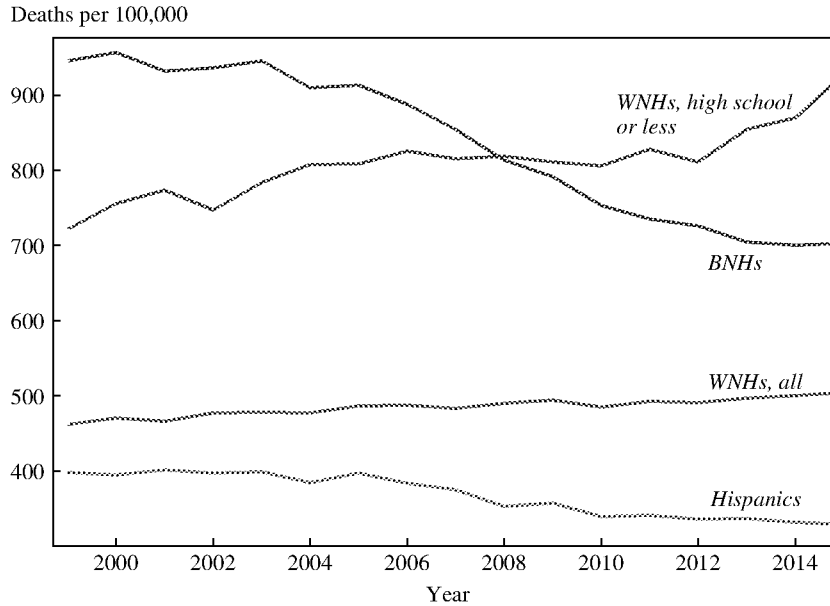
**Figure 1.** All-Cause Mortality by Race and Ethnicity for Age 50–54, 1999–2015




Sources: CDC WONDER; National Vital Statistics System; authors' calculations.

## I. Mortality and Morbidity in the United States and Other Rich Countries

We begin by dissecting changes in mortality and morbidity over space and across age, sex, race, and education. This provides a set of facts to be matched against potential explanations for the epidemic.

### *I.A. Documenting Mortality*

Increasing midlife white mortality rates, particularly for whites with no more than a high school degree, stand in contrast to mortality declines observed for other ethnic and racial groups in the United States, and those observed in other wealthy countries. Figure 1 shows mortality rates per 100,000 for men and women (combined) age 50–54 from 1999 to 2015. We show separate mortality rates for black non-Hispanics (BNHs), for Hispanics, and for all WNHs, as well as for the subset of WNHs with no more than a high school degree. The top line shows rapid mortality decline for blacks, while the bottom line shows that Hispanics continue to make progress against mortality at a rate of improvement that, as we shall see,

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**Table 1.** All-Cause Mortality for White Non-Hispanics with High School or Less and All Black Non-Hispanics by Five-Year Age Cohort, 1999 and 2015[a]

| | *1999* | | *2015* | |
|---|---|---|---|---|
| *Age* | *White non-Hispanics, high school or less* | *Blacks, all* | *White non-Hispanics, high school or less* | *Blacks, all* |
| 25–29 | 145.7 | 169.8 | 266.2 | 154.6 |
| 30–34 | 176.8 | 212.0 | 335.5 | 185.5 |
| 35–39 | 228.8 | 301.4 | 362.8 | 233.6 |
| 40–44 | 332.2 | 457.4 | 471.4 | 307.2 |
| 45–49 | 491.2 | 681.6 | 620.1 | 446.6 |
| 50–54 | 722.0 | 945.4 | 927.4 | 703.1 |
| 55–59 | 1,087.6 | 1,422.8 | 1,328.3 | 1,078.9 |
| 60–64 | 1,558.4 | 1,998.3 | 1,784.6 | 1,571.1 |

Sources: National Vital Statistics System; authors' calculations.
a. Mortality rates are expressed as deaths per 100,000 people at risk.

is similar to the rate of mortality decline in other rich countries. In contrast, WNHs are losing ground. Male WNHs are doing less badly than female WNHs, a distinction not shown here but examined in detail below, but mortality rates for both were higher in 2015 than in 1998. Although we do not have data on WNHs before 1989, we can track mortality rates for all whites age 45–54 starting in 1900; during the 20th century, these mortality rates declined from more than 1,400 per 100,000 to less than 400. After the late 1930s, mortality fell year by year, with the exception of a pause around 1960 (which likely was attributable to the rapid increase in the prevalence of smoking in the 1930s and 1940s), with rapid decline resuming in 1970, when treatments for heart disease began to improve. In this historical context of almost continuous improvement, the rise in mortality in midlife is an extraordinary and unanticipated event.

Mortality rates of BNHs age 50–54 have been and remain higher than those of WNHs age 50–54 as a whole, but have fallen rapidly, by about 25 percent from 1999 to 2015; as a result of this, and of the rise in white mortality, the black/white mortality gap in this (and other) age group(s) has been closing (National Center for Health Statistics 2016; Fuchs 2016). In this regard, the top two lines in figure 1 are of interest; the mortality rates of WNHs with a high school degree or less, which were about 30 percent *lower* than the mortality rates of blacks (irrespective of education) in 1999 (722 versus 945 per 100,000), by 2015 were 30 percent *higher* (927 versus 703 per 100,000). The same mortality crossover between BNHs and the least educated WNHs can be seen in table 1 for every five-year age group from 25–29 to 60–64; we note that for age groups younger than 45, there

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has been a decline in the fraction of WNHs with only a high school degree, so that selection may be playing some role for these younger groups.

Figure 1 presents the comparison of WNHs with a high school degree or less with *all* BNHs—including those with some college or a college degree, who carry a lower risk of mortality. Putting BNHs and WNHs with a high school degree or less head-to-head, figure 2 shows that the black/white mortality gap has closed for every five-year age cohort between the 25–29 and 50–54 age groups—due both to mortality declines for blacks, and mortality increases for whites. The racial gap in mortality among the least educated has all but disappeared. Again, we note the decline in the fraction of those with a high school degree or less in younger age cohorts; the declines are similar (20 percentage points) for WNHs and BNHs.

Figure 3 shows the comparison of the United States with selected other rich countries (Australia, Canada, France, Germany, Sweden, and the United Kingdom). This updates figure 1 in Case and Deaton (2015), using the 45–54 age band, adding 2014 and 2015, and compares unadjusted mortality in the left panel with single-year, age-adjusted mortality in the right panel. The United States and the comparison countries have been age adjusted within the age band, using 2010 as the base year and using mortality data for single years of age from the raw data. Age adjustment changes little, but somewhat smooths the rates of decline in the comparison countries. Using the age-adjusted rates, every comparison country had an average rate of decline of 2 percent a year between 1990 and 2015. Although WNHs saw that same decline until the late 1990s, it was followed by intermittent and overall mortality increases through 2015. Age-adjusted mortality rates of BNHs age 45–54 fell by 2.7 percent a year from 1999 to 2015, and those of Hispanics fell by 1.9 percent.

Online appendix figure 1 presents all-cause mortality by selected single-year ages for age 30, 40, 45, 50, 55, and 60. From age 30 through 55, U.S. WNH mortality was (at best) not falling, and for some ages increased, while rates in other rich countries fell at all ages.

Figure 4 presents mortality rate trends for midlife five-year age groups from 2000 to 2014 for U.S. WNHs, BNHs, and Hispanics, and average trends for the six comparison countries used above.[3] WNHs age 30–34 had mortality rate increases of almost 2 percent a year on average during this

3. Five of the six comparison countries reported deaths through 2013, and three of the six reported deaths through 2014. Trends for the comparison countries are estimated as the coefficient on the time trends from age-group-specific regressions of log mortality on a time trend and on a set of country indicators.

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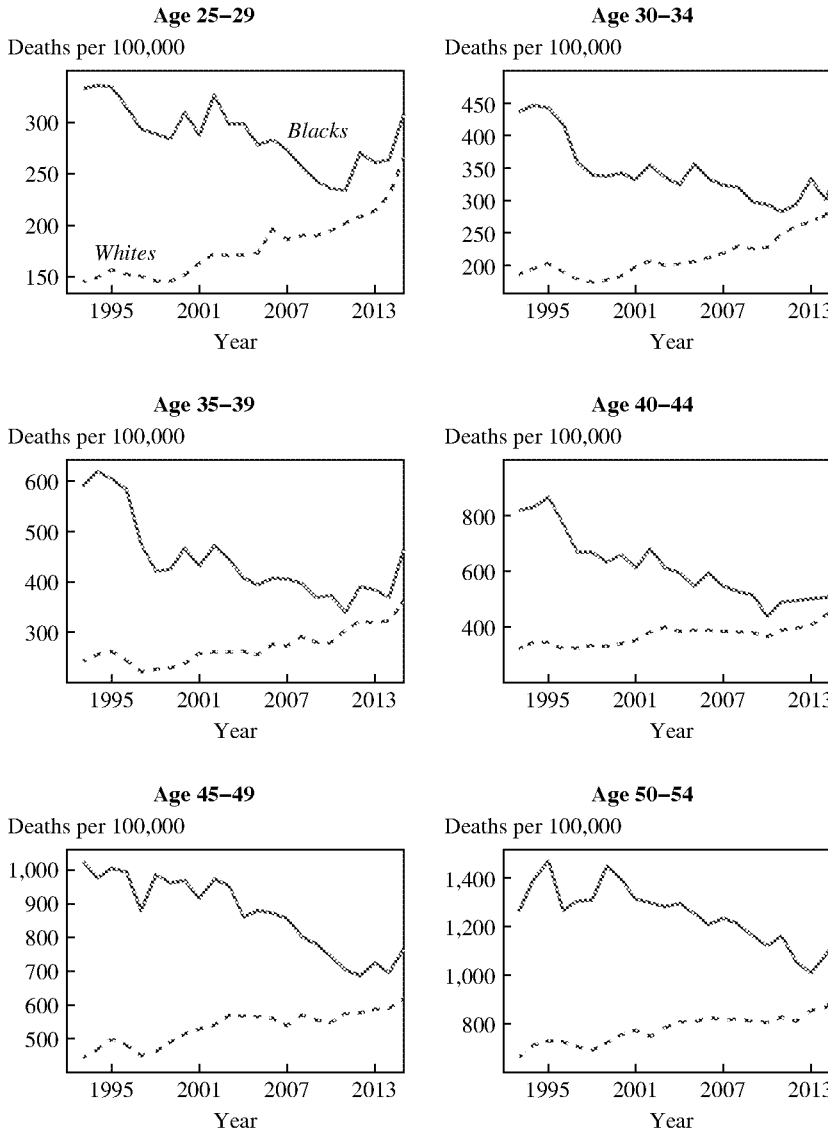
**Figure 2.** All-Cause Mortality for Black Non-Hispanics and White Non-Hispanics with a High School Degree or Less, 1993–2015




Sources: National Vital Statistics System; authors' calculations.

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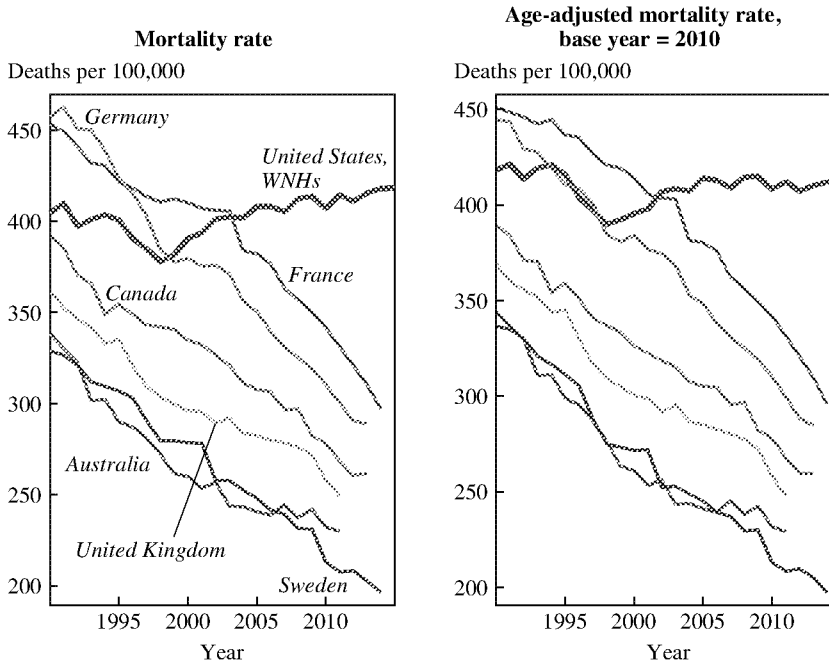
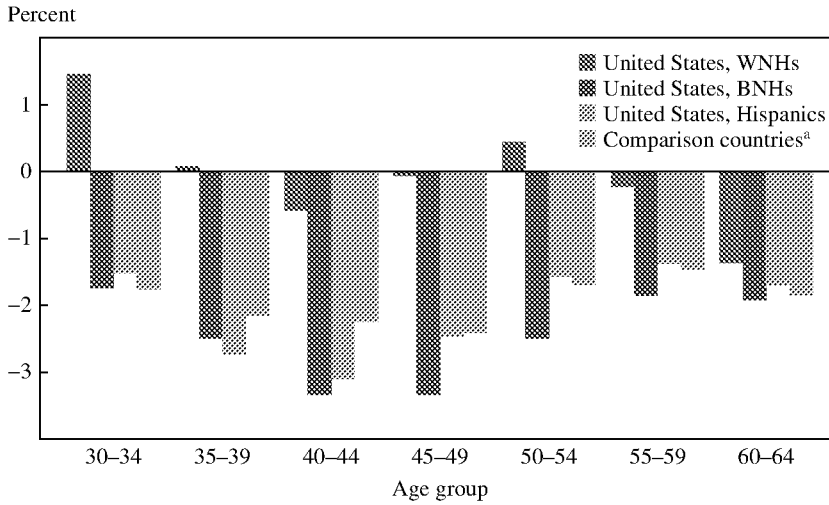
**Figure 3.** All-Cause Mortality by Country for Age 45–54, 1990–2015




Sources: National Vital Statistics System; Human Mortality Database; WHO Mortality Database; authors' calculations.

**Figure 4.** Mortality Trends by Five-Year Age Group, 2000–14




Sources: CDC WONDER; Human Mortality Database; WHO Mortality Database; authors' calculations.
a. The comparison countries are Australia, Canada, France, Germany, Sweden, and the United Kingdom.

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**Table 2.** Average Annual Percent Change in Mortality for Age 50–54 by Cause, 1999–2015

| *Country or racial or ethnic group* | *All-cause* | *Drugs, alcohol, or suicide* | *Heart disease* | *Cancer* |
|---|---|---|---|---|
| U.S. white non-Hispanics | 0.5 | 5.4 | −1.0 | −1.1 |
| U.S. black non-Hispanics | −2.3 | 0.1 | −2.7 | −2.4 |
| U.S. Hispanics | −1.5 | 1.0 | −2.5 | −1.5 |
| United Kingdom | −2.1 | 1.0 | −4.0 | −2.3 |
| Ireland | −2.6 | 3.0 | −5.1 | −2.3 |
| Canada | −1.1 | 2.5 | −3.0 | −1.8 |
| Australia | −1.0 | 2.5 | −2.8 | −1.8 |
| France | −1.3 | −1.2 | −2.9 | −1.7 |
| Germany | −1.9 | −2.3 | −3.5 | −2.1 |
| Sweden | −2.1 | 0.8 | −3.1 | −2.3 |
| Switzerland | −2.5 | −2.6 | −4.0 | −2.3 |
| Denmark | −1.8 | 0.1 | −4.7 | −2.6 |
| Netherlands | −2.3 | −0.0 | −5.5 | −1.4 |
| Spain | −2.1 | −0.3 | −3.2 | −2.0 |
| Italy | −2.1 | −2.2 | −4.7 | −2.0 |
| Japan | −2.2 | −2.1 | −1.4 | −2.8 |

Sources: National Vital Statistics System; Human Mortality Database; WHO Mortality Database; authors' calculations.

15-year period. Changes in direction for mortality rates in young adulthood or early middle age, taken alone, are less uncommon and less surprising; death rates are low at these ages, and shocks can easily lead to a change of direction (for example, HIV in the United States in the early 1990s). But the fact that the United States has pulled away from the comparison countries throughout middle age is cause for concern. Our main focus here is not on whether progress on all-cause mortality has only flatlined or has actually reversed course, although this was what attracted most public response to Case and Deaton (2015). Rather, our main point is that other wealthy countries continued to make progress while the United States did not. As we have seen, BNHs have higher mortality rates than whites, but their mortality has fallen even more rapidly than rates in Europe, while Hispanics, who have lower mortality rates than whites, have had declines in rates similar to the average in the comparison countries in all age groups.

Table 2 presents all-cause mortality trends for the 50–54 age band for U.S. WNHs, BNHs, and Hispanics, and a larger set of comparison countries—now also including Ireland, Switzerland, Denmark, the Netherlands, Spain, Italy, and Japan. The numbers in the table are the coefficients on time in (country- and cause-specific) regressions of the log of mortality

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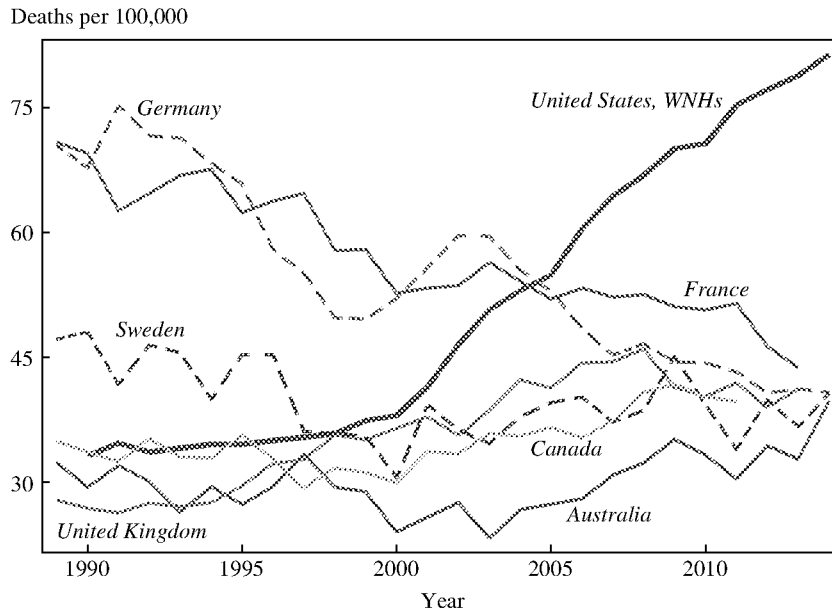
**Figure 5.** Deaths of Despair by Country for Age 50–54, 1989–2014[a]




Sources: National Vital Statistics System; Human Mortality Database; WHO Mortality Database; authors' calculations.

a. Deaths of despair refer to deaths by drugs, alcohol, or suicide.

for the cause in each column on a time trend, and the numbers can be interpreted as average annual rates of change. The mortality trend is positive for U.S. WNHs, and negative for U.S. BNHs, U.S. Hispanics, and for every other country. In this larger set of comparison countries, mortality rates for men and women age 50–54 declined by 1.9 percent a year on average between 1999 and 2014, while rates for U.S. WNHs increased by 0.5 percent a year.

That deaths of despair play a part in the mortality turnaround can be seen in figure 5, which presents mortality rates from accidental or intent-undetermined alcohol and drug poisoning, suicide, and alcoholic liver disease and cirrhosis for U.S. WNHs, and those in the comparison countries, all age 50–54. U.S. whites had much lower mortality rates from drugs, alcohol, and suicide than France, Germany, or Sweden in 1990, but while mortality rates in the comparison countries converged to about 40 deaths per 100,000 after 2000, those among U.S. WNHs doubled, to 80. The average annual rate of change from 1999 to 2015 of mortality rates from these deaths of despair are presented in column 2 of table 2. For U.S. BNHs, mortality

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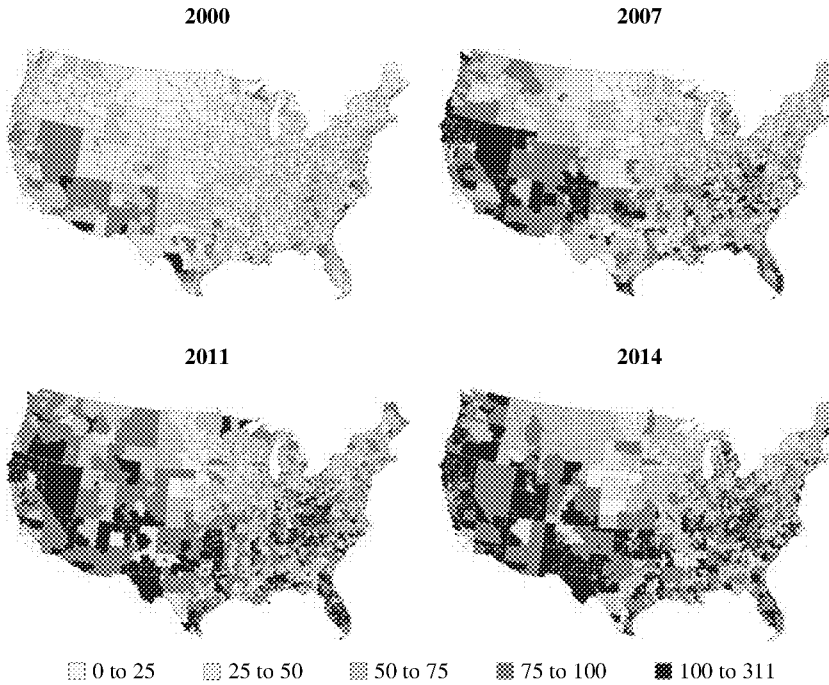
**Figure 6.** Deaths of Despair for White Non-Hispanics Age 45–54, by *Couma*, 2000–14[a]




Sources: National Vital Statistics System; authors' calculations.
a. Deaths of despair refer to deaths by drugs, alcohol, or suicide. The units are deaths per 100,000. *Coumas* are geographic units that are a blend of counties and Public Use Microdata Areas.

from these causes has been constant, at 50 deaths per 100,000 since 2000. The trends in other English-speaking countries may provide something of a warning flag; Australia, Canada, Ireland, and the United Kingdom stand alone among the comparison countries in having substantial positive trends in mortality from drugs, alcohol, and suicide during this period. However, their increases are dwarfed by the increase among U.S. whites.

The epidemic has spread from the Southwest, where it was centered in 2000, first to Appalachia, Florida, and the West Coast by the mid-2000s, and is now countrywide (figure 6). Rates have been consistently lower in the large fringe metropolitan statistical areas (MSAs), but increases have been seen at every level of residential urbanization in the United States (online appendix figure 2); it is neither an urban nor a rural epidemic, but rather both.

The units in figure 6 are small geographic areas that we refer to as *coumas*, a blend of counties and Public Use Microdata Areas (PUMAs). For

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counties that are larger than PUMAs, the *couma* is the county and is made up of PUMAs, while in parts of the country where counties are sparsely populated, one PUMA may contain many counties, and the PUMA becomes the *couma.* (Details are provided in the online appendix.) We have constructed close to 1,000 *coumas*, which cover the whole of the United States, with each containing at least 100,000 people. The geography of mortality will be explored in detail in future work; we note here that some *coumas* have relatively few deaths in the age group illustrated, so the coloring of the maps has a stochastic component that can be misleading for sparsely populated *coumas* that cover large geographic areas. That said, the spread from the Southwest matches the story told by Sam Quinones (2015), who documents the interplay between illegal drugs from Mexico and legal prescription drugs throughout the United States. Most recently, with greater attempts to control prescriptions for opioids, deaths from illegal drugs are becoming relatively more important (Hedegaard, Warner, and Miniño 2017).

We now turn to birth cohorts, beginning with the cohort born in 1935; this analysis is important for the story that we develop in section III below. (Note that, over this much longer period, the fraction of each birth cohort with a bachelor's degree or more rose. Specifically, in the birth cohorts we analyze in section III—those born between 1945 and 1980—the fraction of whites with a bachelor's degree remained constant, at 30 percent, between 1945 and 1965; increased from 30 to 40 percent for the cohorts born between 1965 and 1970; and remained stable, at 40 percent, for cohorts born between 1970 and 1980.) Figure 7 shows mortality rates for the birth cohorts of WNHs with less than a bachelor's degree at five-year intervals for birth years from 1935 to 1980, from drug overdoses (top-right panel), suicide (bottom left), alcohol-related liver deaths (bottom right), and all three together (top left). After the 1945 cohort, mortality rises with age in each birth cohort for all three causes of death; moreover, the rate at which mortality rises with age is higher in every successive birth cohort. The rise in mortality by birth cohort is not simply a level shift but also a steepening of the age-mortality profiles, at least until the youngest cohorts. Repeating the figure for all education levels pooled yields qualitatively similar results, but with the upward movement and the steepening slightly muted (online appendix figure 3); we shall return to the issue of selection into education in section III below.

As noted in Ellen Meara and Jonathan Skinner's (2015) commentary on Case and Deaton (2015), increases in mortality from deaths of despair would not have been large enough to change the direction of all-cause mortality for U.S. whites if this group had maintained its progress against other

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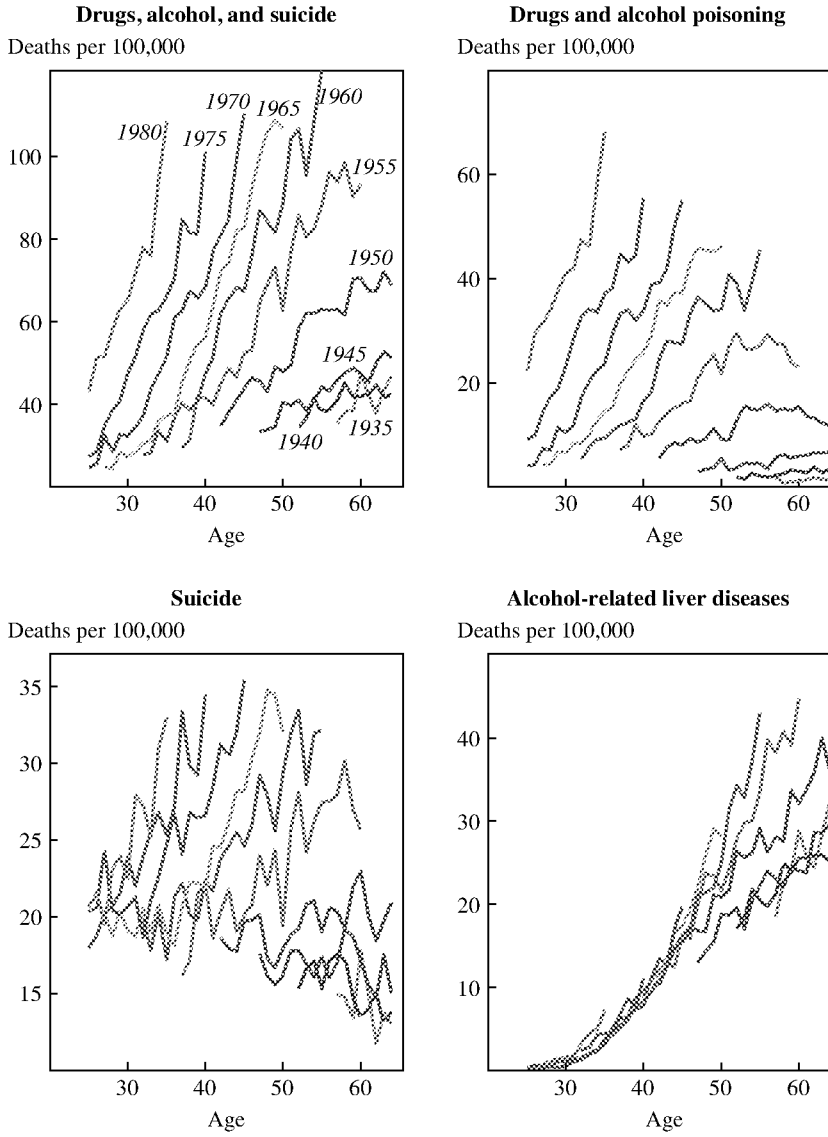
**Figure 7.** Deaths of Despair for White Non-Hispanics with Less Than a Bachelor's Degree, by Birth Cohort




Sources: National Vital Statistics System; authors' calculations.

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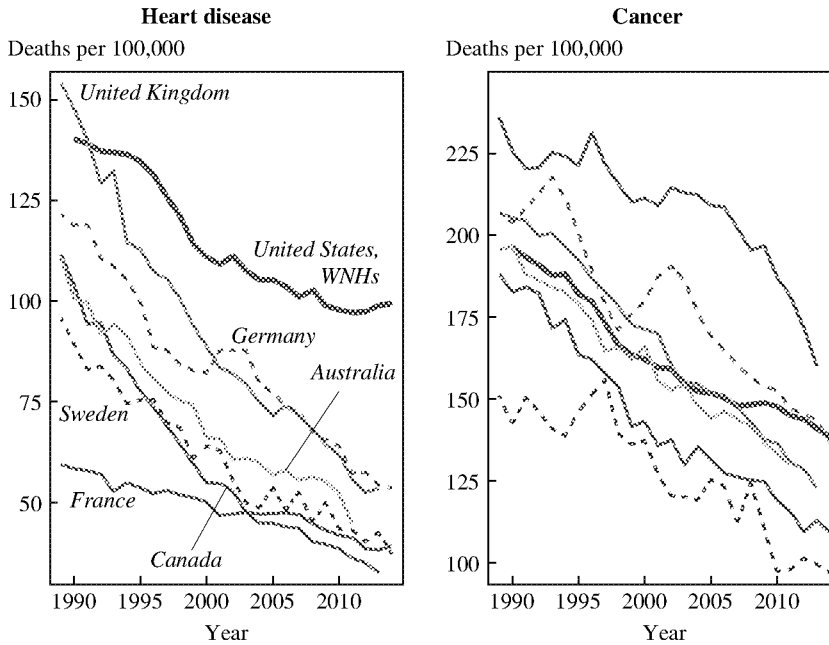
**Figure 8.** Heart Disease and Cancer Mortality by Country for Age 50–54, 1989–2014




Sources: National Vital Statistics System; Human Mortality Database; WHO Mortality Database; authors' calculations.

causes of death. For the two major causes of death in midlife—heart disease and cancer—the rate of mortality decline for age groups 45–49 and 50–54 fell from 2 percent a year on average between 1990 and 1999 to 1 percent a year between 2000 and 2014. The left panel of figure 8 presents heart disease mortality rates for U.S. WNHs and the comparison countries from 1989 to 2014. U.S. whites began the 1990s with mortality rates from heart disease that were high relative to other wealthy countries and, though rates continued to fall elsewhere, the rate of decline first slowed in the United States, and then stopped entirely between 2009 and 2015. With respect to cancer (right panel of figure 8), U.S. whites began the 1990s in the middle of the pack; again, if in less dramatic fashion, progress for U.S. whites slowed after 2000. The last two columns of table 2 show that, for both heart disease and cancer, U.S. whites age 50–54 had less than half the rate of decline observed for U.S. blacks and almost all the comparison countries for the period 1999–2014.

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The slowdown in progress on cancer can be partially explained by smoking; the decline in lung cancer mortality slowed for male WNHs age 45–49 and 50–54 from 2000 to 2014, and the mortality rate *increased* for women age 45–49 between 2000 and 2010. (See online appendix figure 4.) This puts the progress made against lung cancer by U.S. whites toward the bottom of the pack in comparison with U.S. blacks and with other wealthy countries.

Explaining the slowdown in progress in heart disease mortality is not straightforward. Many commentators have long predicted that obesity would eventually have this effect, and see little to explain (Flegal and others 2005; Olshansky and others 2005; Lloyd-Jones 2016). But the time, sex, and race patterns of obesity do not obviously match the patterns of heart disease. Although obesity rates are rising more rapidly among blacks than among whites in the United States, blacks made rapid progress against heart disease in the period 1999–2015 (see table 2 and online appendix figure 5). Beyond that, if the United States is a world leader in obesity, Britain is not far behind—25 percent of its adult population is obese, compared with 28 percent of U.S. WNHs—but Britain shows a continued decline in mortality from heart disease. Andrew Stokes and Samuel Preston (2017, p. 2) argue persuasively that deaths attributable to diabetes are understated in the United States, perhaps by a factor of four, so that the additional obesity-related deaths from diabetes are not being measured but may be incorrectly being attributed to heart disease. They note that when diabetes and cardiovascular disease are both mentioned on a death certificate, "whether or not diabetes is listed as the underlying cause is highly variable and to some extent arbitrary." If this happens in other countries, it might also explain the slowing of heart disease progress in other rich countries whose obesity rates are rising. Returning to the six comparison countries examined earlier (Australia, Canada, France, Germany, Sweden, and the United Kingdom), we find that, on average, the decline in heart disease slowed from 4.0 percent a year (1990–99) to 3.2 percent (2000–14); see figure 8. The contribution of obesity and diabetes to the mortality increases documented here clearly merits additional attention.

Mortality rate increases varied in different parts of the country in the period 1999–2015. Of the nine census divisions, the hardest hit was East South Central (Alabama, Kentucky, Mississippi, and Tennessee), which saw mortality rates rise 1.6 percent a year on average for WNHs age 50–54, increasing from 552 to 720 deaths per 100,000 during this period. Mortality rates fell in the Mid-Atlantic division, held steady in the New England and the Pacific divisions, but grew substantially in all other divisions. A more complete picture of the change in mortality rates can be seen in figure 9,

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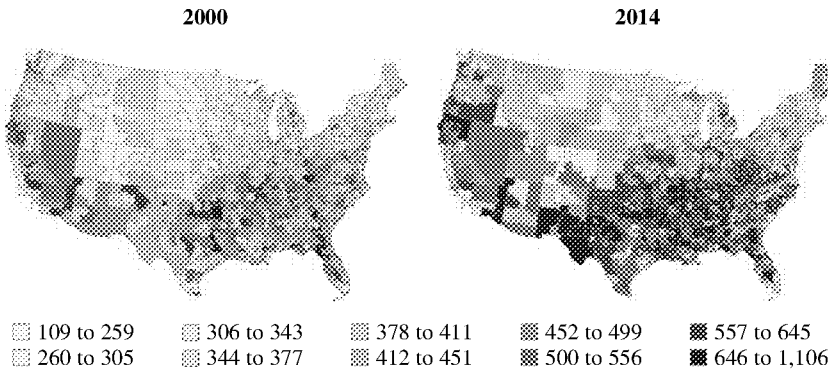
**Figure 9.** All-Cause Mortality for White Non-Hispanics Age 45–54, by *Couma,* 2000 and 2014[a]




Sources: National Vital Statistics System; authors' calculations.

a. The units are deaths per 100,000. *Coumas* are geographic units that are a blend of counties and Public Use Microdata Areas.

which maps mortality rates for WNHs, age 45–54, by the *coumas* introduced above. Figure 9 presents mortality rates by *couma* in 2000 and 2014. With the exception of the I-95 corridor, and parts of the Upper Midwest, all parts of the United States have seen mortality increases since the turn of the century; 70 percent of *coumas* saw mortality rate increases between 2000 and 2011 (the last year when the PUMAs drawn for 2000 allow a decade-long alignment of *coumas*.) Mortality rates for WNHs age 45–54 trended downward in only three states during the period 1999–2015: California, New Jersey, and New York. Although the media often report the mortality turnaround as a rural phenomenon, all-cause mortality of WNHs age 50–54 rose on average 1 percent a year in four of six residential classifications between 1999 and 2015—medium MSAs, small MSAs, micropolitan areas, and noncore (non-MSA) areas. Mortality rates were constant in large fringe MSAs during this period, and fell weakly (0.3 percent a year, on average) in the large central MSAs.

Mortality from deaths of despair and all-cause mortality are highly correlated; deaths of despair are a large and growing component of midlife all-cause mortality. But it is important to remember that changes in all-cause mortality are also driven by other causes, particularly heart disease and cancer, and that progress on those varies from state to state. Take, for example, mortality in two states that are often used to show the importance of health behaviors: Nevada and Utah. Two-thirds of Utahans are Mormon, whose adherence requires abstinence from alcohol, coffee,

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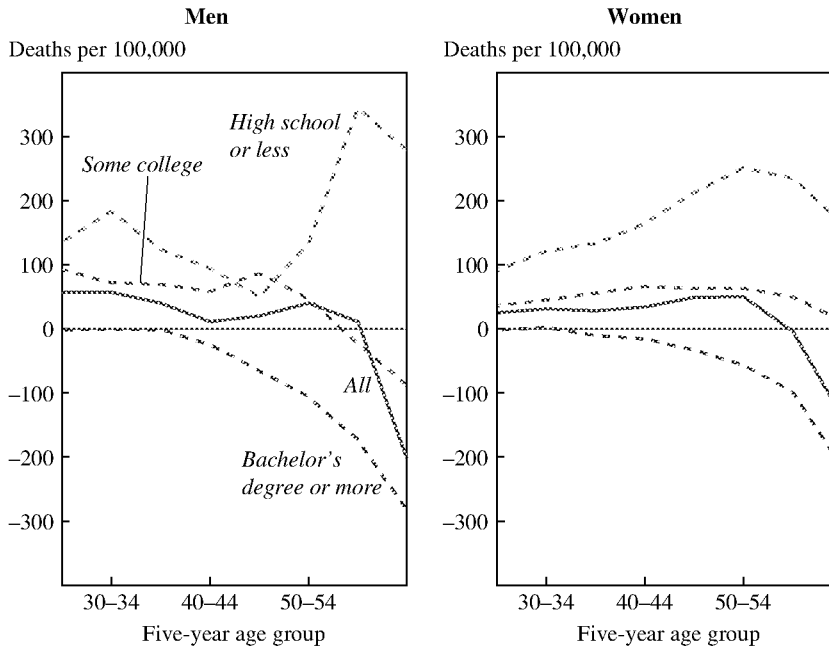
**Figure 10.** Change in Mortality for White Non-Hispanics, by Level of Education, 1998–2015




Sources: National Vital Statistics System; authors' calculations.

and tobacco. Two-thirds of Nevadans live in and around Las Vegas, also known as "Sin City." Ranking states by their *all-cause mortality* rate for WNHs age 45–54, we find that Nevada ranked 9th highest among all states in 2014; Utah ranked 31st. Heart disease mortality was twice as high in Nevada in 2014 as it was in Utah (119 per 100,000 versus 59 per 100,000). However, *both* Nevada and Utah were among the top 10 states ranked by mortality from drugs, alcohol, and suicide that year. Nevada was 4th highest, with 117 deaths per 100,000, and Utah was 10th, with 99 deaths per 100,000 WNHs age 45–54. The suicide rate doubled in Utah in this population between 1999 and 2014, and the poisoning rate increased 150 percent. Different forces—social and economic, health behavior– and health care–related—may drive changes in some causes of death, but not others, and these forces themselves are likely to change with time.

As we saw in figure 1, changes in U.S. mortality rates for WNHs differ starkly by level of education. Figure 10 shows this for men and women separately. Changes in mortality rates between 1998 (the year with the

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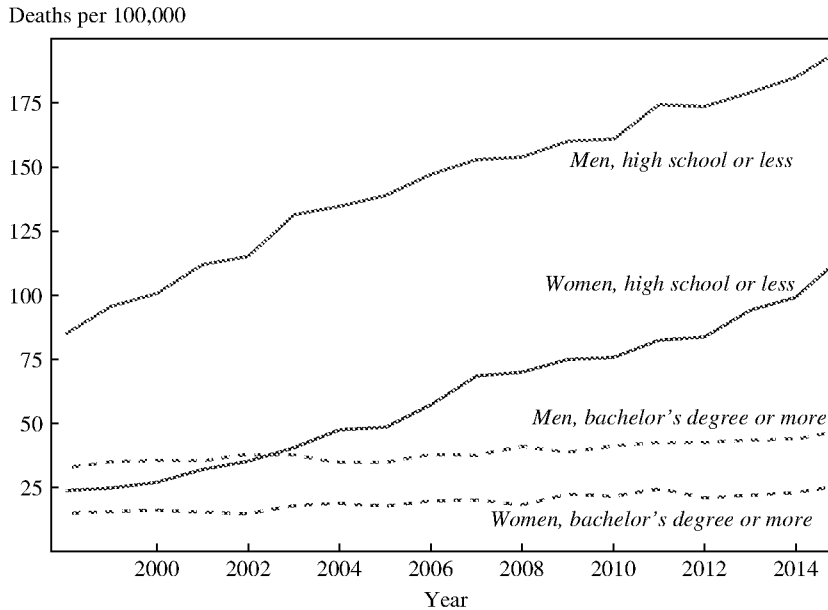
**Figure 11.** Deaths of Despair for White Non-Hispanics Age 50–54, by Level of Education, 1998–2015[a]




Sources: National Vital Statistics System; authors' calculations.
a. Deaths of despair refer to deaths by drugs, alcohol, or suicide.

lowest mortality rate for those age 45–54) and 2015 are tracked by five-year age cohort, with men in the left panel, and women in the right. From age 25–29 to age 55–59, men and women with less than a four-year college degree saw mortality rates rise between 1998 and 2015, while those with a bachelor's degree or more saw mortality rates drop, with larger decreases at higher ages. Overall, this resulted in mortality rate increases for each five-year age group, taking all education groups together, marked by the solid lines in figure 10. Although there are some differences between men and women, the patterns of changes in mortality rates are broadly similar in each education group.

The key story in figure 10 is the increase in mortality rates for both men and women without a bachelor's degree, particularly for those with no more than a high school degree. For WNHs age 50–54, figure 11 compares deaths of despair for men and women with a high school degree or less (approximately 40 percent of this population during the period 1998–2015) with those with a bachelor's degree or more (32–35 percent). For men and

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women with less education, deaths of despair are rising in parallel, pushing mortality upward. However, the *net* effect on all-cause mortality depends on what is happening to deaths from heart disease and cancer, including lung cancer, and these other causes have different patterns for men and women. We shall document these findings in more detail in future work.

During this period, the disparity in mortality grew markedly between those with and without a bachelor's degree. The mortality rate for men with less than a bachelor's degree age 50–54, for example, increased from 762 to 867 per 100,000 between 1998 and 2015, while for men with a bachelor's degree or more, mortality fell from 349 to 243. Those with less than a bachelor's degree saw progress stop in mortality from heart disease and cancer, and saw increases in chronic lower respiratory disease and deaths from drugs, alcohol, and suicide (online appendix figure 6). Moreover, increasing differences between education groups are found for each component of deaths of despair—drug overdoses, suicide, and alcohol-related liver mortality—analyzed separately (online appendix figure 7).

Our findings on the widening educational gradient in figure 10 are consistent with and extend a long-unfolding body of literature—which was recently reviewed, for example, by Robert Hummer and Elaine Hernandez (2013). Evelyn Kitagawa and Philip Hauser (1973) first identified educational gradients in mortality in the United States; and later work, particularly that of Preston and Irma Elo (1995), found that the differences widened for men between 1970 and 1980. Meara, Seth Richards, and David Cutler (2008) show a further widening from 1981 to 2000, including an absolute decline in life expectancy at age 25 for low-educated women between 1990 and 2000. They show that there was essentially no gain in adult life expectancy from 1981 to 2000 for whites with a high school degree or less, and that educational disparities widened, for both men and women, and for whites and blacks. A widely reported study by S. Jay Olshansky and others (2012) found that the life expectancy of white men and women without a high school degree *decreased* from 1990 to 2008. Given that the fraction of the population without a high school degree declined rapidly during this period—and if, as is almost certain, this fraction was increasingly negatively selected—the comparison involves two very different groups, one that was much sicker than the other when they left school (Begier, Li, and Maduro 2013). John Bound and others (2014) address the issue by looking at changes in mortality at different percentiles of the educational distribution and find no change in the survival curves for women at the bottom educational quartile between 1990 and 2010 and an improvement for men.

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Our own findings here are more negative than those in the literature. Figure 10 shows that mortality rates for those with no more than a high school degree *increased* from 1998 to 2015 for WNH men *and* women in all five-year age groups from 25–29 to 60–64. We suspect that these results differ from Meara, Richards, and Cutler (2008) because of the large differential increase in deaths from suicides, poisonings, and alcohol-related liver disease after 1999 among whites with the lowest educational attainment (see figure 11).

Mortality differentials by education among whites in the United States contrast with those in Europe. In a recent study, Johan Mackenbach and others (2016) examine mortality data from 11 European countries (or regions) over the period 1990–2010 and find that, in most cases, mortality rates fell for *all* education groups, and fell by more among the *least* educated, so that the (absolute) differences in mortality rates by education have diminished. (Disparities have increased in *relative* terms because the larger decreases among the less well educated have been less than proportional to their higher baseline mortality rates.)

### *I.B. Documenting Morbidity*

Large and growing education differentials in midlife mortality are paralleled by reported measures of midlife health and mental health. Figure 12 presents levels and changes over time (1993–2015) in the percent of WNHs at each age between 35 and 74 who report themselves to be in "excellent" or "very good" health (on a 5-point scale that includes good, fair, or poor as options). The fact that self-assessed health falls with age is a standard (and expected) result, and can be seen in all three panels, each for an education group. In the period 1999–2002, there are marked differences between the education groups in self-assessed health; 72 percent of 50-year-olds with a bachelor's degree or more report themselves in excellent or very good health, and the same is true for 59 percent of those with some college education, and for only 49 percent of those with a high school degree or less. Over the period 1999–2015, differences between education groups became more pronounced, with fewer adults in lower education categories reporting excellent health at any given age. In the years 2012–15, at age 50, the fraction of those with bachelor's degrees reporting excellent health had not changed, while that fraction fell 4 percentage points for those with some college, and 7 percentage points for those with a high school degree or less. (Beyond retirement age, which saw progress against mortality in the early 2000s, self-assessed health registers improvement as well.)

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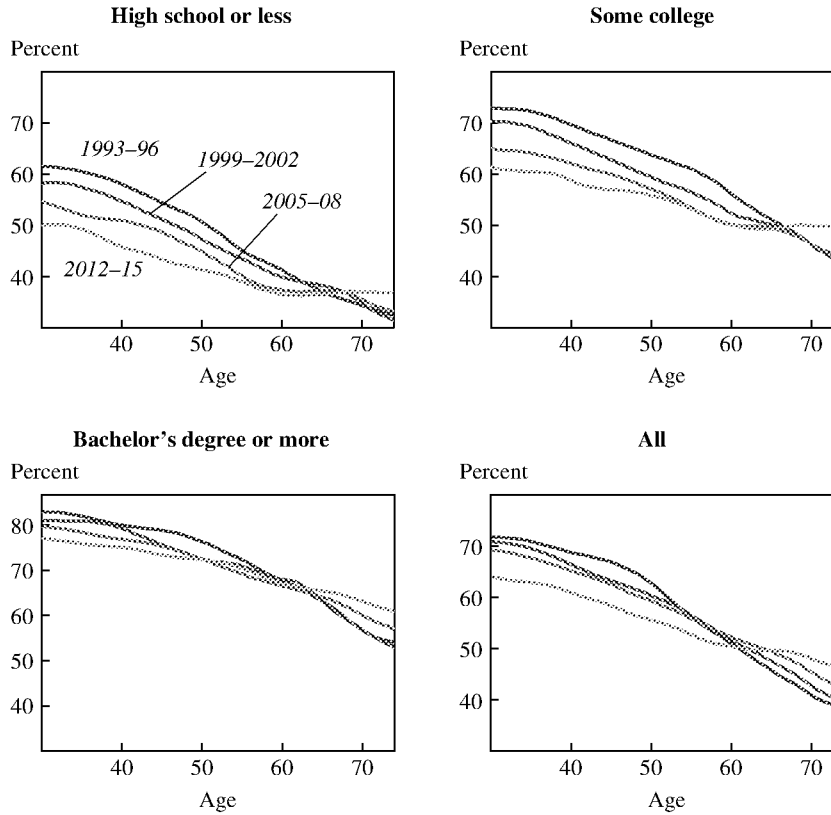
**Figure 12.** White Non-Hispanics Reporting Excellent or Very Good Health, by Survey Year




Sources: CDC Behavioral Risk Factor Surveillance System; authors' calculations.

Since the mid-1990s (when questions on pain and mental health began to be asked annually in the National Health Interview Survey), middle-aged whites' reports of chronic pain and mental distress have increased, as have their reports of difficulties with activities of daily living (Case and Deaton 2015). Figure 13 presents results for WNHs' reports of sciatic pain, for birth cohorts spaced by 10 years, separately for those with less than a four-year college degree (left panel), and those with a bachelor's degree or more (right panel). Pain is a risk factor for suicide and, as the left panel shows, for those with less than a college degree there has been a marked increase between birth cohorts in reports of sciatic pain. As was the case for mortality, the age profiles for pain steepen with each successive birth

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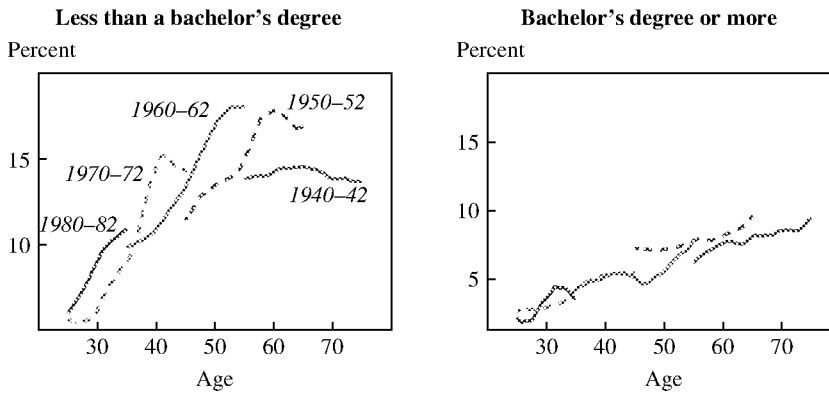
**Figure 13.** Reports of Sciatic Pain for White Non-Hispanics, by Birth Year




Sources: CDC National Health Interview Survey; authors' calculations.

cohort. For those with a bachelor's degree, successive birth cohorts overlap in their reports of pain at any given age, while for those with less education, an ever-larger share report pain in successive cohorts. Similar results obtain for other morbidities.

## II. Mortality and Incomes

Much of the commentary has linked the deteriorating health of midlife whites to what has happened to their earnings and incomes, and in particular to stagnation in median wages and in median family incomes. Because there has been real growth in per capita GDP and in mean per capita income, the poor performance for middle-class incomes can be mechanically attributed to the rising share of total income captured by the best-off Americans. This suggests an account in which stagnant incomes and deteriorating health become part of the narrative of rising income inequality; see a recent essay by Joseph Stiglitz (2015) for one provocative statement. According to this scenario, the rise in suicides, overdoses, and alcohol abuse would not have occurred if economic growth had been more equally shared. Quite apart from the question of whether, if the top had received less, the rest would have received more, we shall see that the economic story can account for part of the increases in mortality and morbidity, but only a part, and that it leaves more unexplained than it explains. Our preliminary conclusion is that, as in previous historical episodes, the changes in mortality and morbidity are only coincidentally correlated with changes in income.

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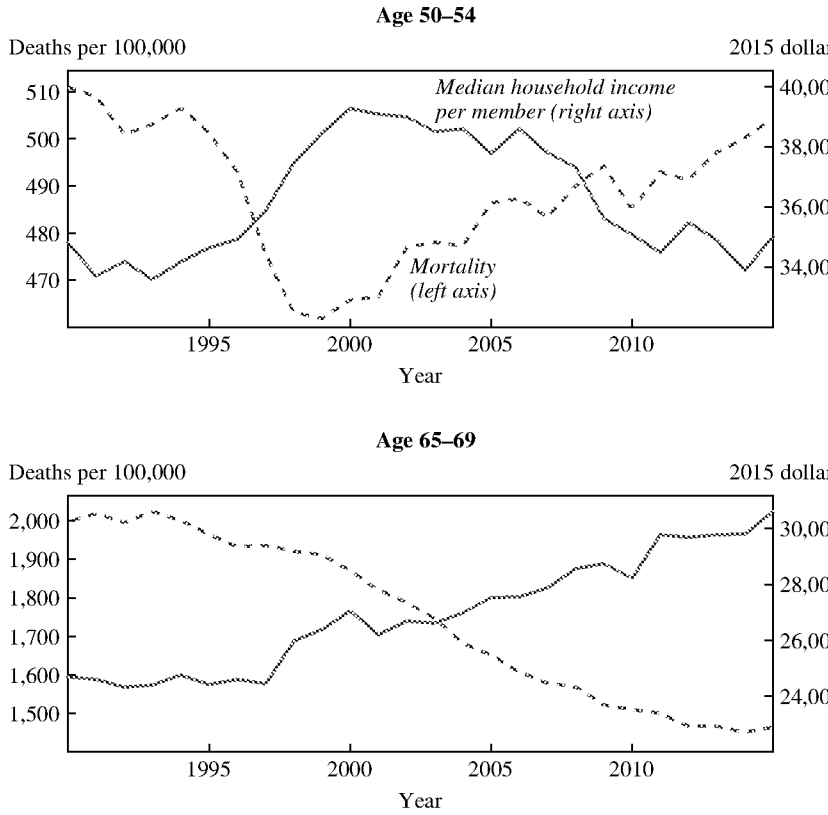
**Figure 14.** All-Cause Mortality and Median Household Income per Member for White Non-Hispanics, 1990–2015




Sources: National Vital Statistics System; Current Population Survey, March supplement; authors' calculations.

### *II.A. Contemporaneous Evidence*

For middle-aged whites, there is a strong correlation between median real household income per person and mortality from 1980 and 2015; an inverse U-shaped pattern of real income, rising throughout the 1980s and 1990s and falling thereafter, matches the U-shape of mortality, which fell until 1998 and rose thereafter. After 1990, we can separate out Hispanics and look at WNHs, for whom the recent mortality experience was worse than for whites as a whole. The top panel of figure 14 shows, for households headed by WNHs age 50–54, real median household income per member from March supplements of the Current Population Survey (presented as solid lines), and (unadjusted all-cause) mortality rates for men and

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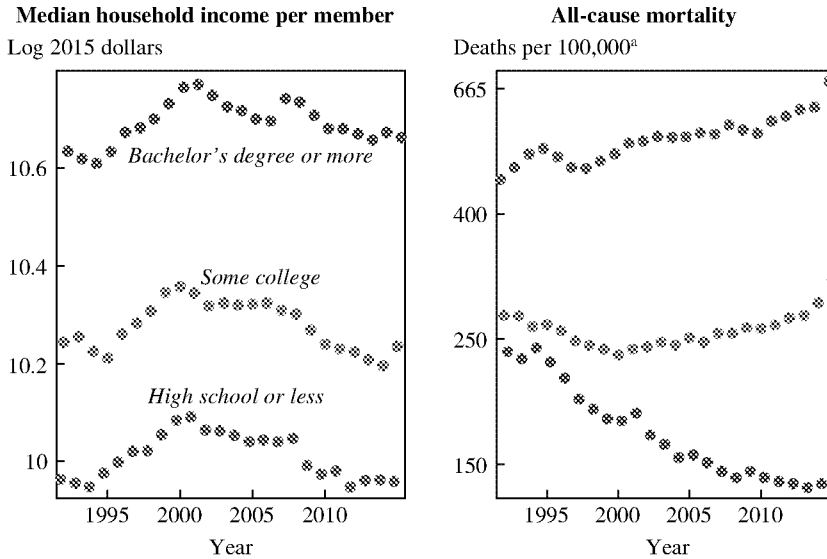
**Figure 15.** Year Margins for All-Cause Mortality and Median Household Income per Member for White Non-Hispanics Age 30–64, 1992–2015




Sources: National Vital Statistics System; Current Population Survey, March supplement; authors' calculations.
a. Note that this axis is log linear.

women age 50–54 together (dashed lines). Mortality and income match closely. The bottom panel shows mortality for the age group 65–69, and median real income per member in households headed by someone in this age band. This older group has done well since 1990 in part because, for those who qualify, initial Social Security payments are indexed to *mean* wages and are subsequently tied to the Consumer Price Index; mean wages have done better than median wages. Real incomes for those age 65–69 increased by a third between 1990 and 2015, while incomes for all middle-aged groups show an initial increase followed by subsequent decline, though the timing and magnitudes are different across age groups. Online appendix figure 8 shows that while the matching of mortality and household income is strongest for the 50–54 age group, it also appears at other ages, albeit less clearly. This looks like good evidence for the effects of income on mortality, not at an annual frequency, which the graphs clearly show is not the case, but because of the (approximate) matching of the timing of the turnarounds across age groups.

When we disaggregate by educational attainment in figure 15, there is less support for an income-based explanation for mortality. The left panel

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shows year margins for log median real income per member, for householders age 30–64, from regressions of log median real income per member on householder age effects and year effects, run separately by education group. The general widening inequality in family incomes in the United States does not show up here in any divergence between the median incomes of those with different educational qualifications, and does not match the divergence in mortality between education groups, as discussed above and seen in the right panel. The negative correlation between mortality and income could be restored by removing the divergent trends from mortality, yet there seems no principled reason to do so.

The matching of income and mortality fares poorly both for BNHs and for Hispanics. Black household incomes rose and fell in line with white household incomes for all age groups between 1990 and 2015; and indeed, after 1999, blacks with a college education experienced even more severe percentage declines in income than did whites in the same education group (figure 16). Yet black mortality rates have fallen steadily, at between 2 and 3 percent a year, for all age groups 30–34 to 60–64; see figure 4 above. The data on Hispanic household incomes are noisier; but, once again, there is no clear difference between their patterns and those for whites. However, their mortality rates have continued to decline at the previously established rate, which is the "standard" European rate of 2 percent a year, as shown in figure 4.

We do not (currently) have data on household median incomes for all the comparison countries, but Eurostat's statistics on income and living conditions provide data from 1997 for France, Germany, Ireland, Italy, the Netherlands, Spain, and the United Kingdom; and for Denmark from 2003, for Sweden from 2004, and for Switzerland from 2007. The European patterns (for all households, the data do not allow age disaggregation) are quite different from those among U.S. households, and they fall into two classes, depending on the effects of the Great Recession. In Ireland, Italy, the Netherlands, Spain, and the United Kingdom, median real family incomes rose until the recession, and were either stagnant or declining thereafter. But in Denmark, France, Germany, and Sweden, there was no slowdown in household incomes after 2007. As we have seen in figure 3 and table 2, there is no sign of differences between these two groups in the rates of mortality decline, nor of any slowing in mortality decline as income growth stopped or turned negative. If incomes work in Europe as they do in the United States, and if the income turnaround is responsible for the mortality turnaround in the United States, we would expect to see at least a slowing in the mortality decline in Europe, if only among the worst-affected countries, but there is none.

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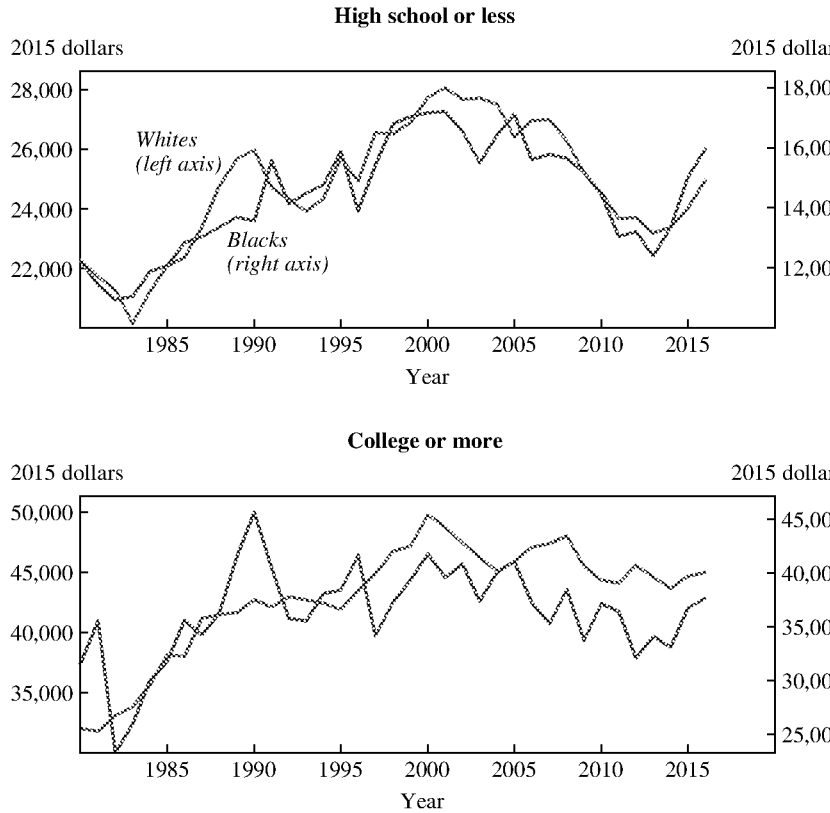
**Figure 16.** Median Household Income per Member Age 45–54, by Race, 1980–2015




Sources: Current Population Survey, March supplement; authors' calculations.

## *II.B. Discussion*

Taking all the evidence together, we find it hard to sustain the income-based explanation. For WNHs, the story can be told, especially for those age 50–54 and for the difference between this group and the elderly, but we are left with no explanation for why blacks and Hispanics are doing so well, nor for the divergence in mortality between college and high school graduates, whose mortality rates are not just diverging but actually going in opposite directions. Nor does the European experience provide support, because the mortality trends show no signs of the Great Recession in spite of its marked effects on household median incomes in some countries but not in others.

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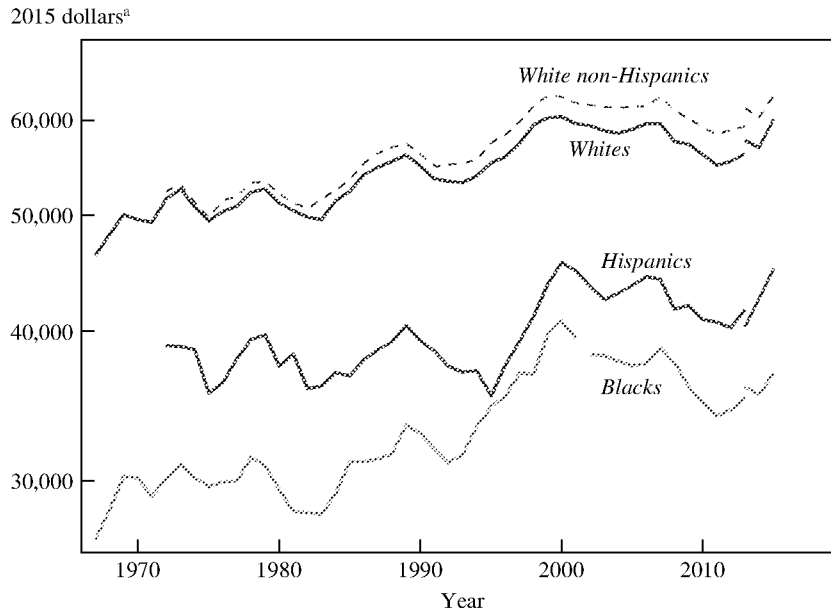
**Figure 17.** Median Household Income by Race and Ethnicity, 1967–2015




Sources: Current Population Survey, March supplement; authors' calculations.
a. Note that this axis is nonlinear.

It is possible that it is not the last 20 years that matter, but rather that the long-run stagnation in wages and incomes has bred a sense of hopelessness. But figure 17 shows that, even if we go back to the late 1960s, the ethnic and racial patterns of median family incomes are similar for whites, blacks, and Hispanics, and so can provide no basis for their sharply different mortality outcomes after 1998. Even so, in the next section, we develop an account that could implicate the long-term decline in earnings among less educated whites.

There is a body of microeconomic literature on health determinants that shows that those with higher incomes have lower mortality rates and higher life expectancy; see National Academies of Sciences, Engineering, and Medicine (2015) and Raj Chetty and others (2016) for a recent large-scale study of the United States. Income is correlated with many other relevant outcomes, particularly education, which, like race and ethnicity, is not available to Chetty and others (2016); even so, there are careful studies on smaller panels, such as that by Elo and Preston (1996), who find separately

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protective effects of income and education, even when both are allowed for together with controls for age, geography, and ethnicity. These studies attempt to control for the obviously important reverse effect of health on income by excluding those who are not in the labor force due to long-term physical or mental illness, or by not using income in the period(s) before death. Even so, there are likely also effects that are not eliminated in this way, for example, those that operate through insults in childhood that impair both adult earnings and adult health. Nevertheless, it seems likely that income is protective of health, at least to some extent, even if it is overstated in the literature that does not allow for other factors.

There is a somewhat more contested body of literature on income and mortality at business cycle frequencies. Daniel Sullivan and Till von Wachter (2009) use administrative data to document the mortality effects of unemployment among high-seniority males; and Courtney Coile, Phillip Levine, and Robin McKnight (2014) note the vulnerability to unemployment of older, preretirement workers, who are unlikely to find new jobs and may be forced into early retirement, possibly without health insurance. The mortality effects that Coile, Levine, and McKnight (2014) and Sullivan and von Wachter (2009) document are not all instantaneous but are spread over many years, and are, in any case, much smaller than the effects that would be required to justify the results in figure 14 for those age 50–54. At the aggregate level, unemployment cannot explain the mortality turnarounds in the post-2000 period; unemployment had recovered to its prerecession level by the end of the period, and was falling rapidly as mortality rose. It is of course possible that the aggregate is misleading, either because unemployment excludes discouraged workers, or because unemployment has not recovered in the places where unemployment prompted mortality; for evidence linking mortality to trade-induced unemployment, see the work of Justin Pierce and Peter Schott (2016) and David Autor, David Dorn, and Gordon Hanson (2017).

There is, however, evidence against the unemployment story from Spain in research by Enrique Regidor and others (2016), who use individual-level data for the complete population of Spain to study mortality in the years 2004–07 compared with 2008–11. In spite of the severity of the Great Recession in Spain, where unemployment rates rose from 8.2 percent in 2007 to 21.4 percent in 2011, mortality was *lower* in the later period. This was true for most causes of death, including suicide, and for people of great or little wealth, approximately measured by floor space or car ownership in 2001, as well as for age groups 10–24, 25–49, and 50–74 taken separately.

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There is a venerable body of literature arguing that good times are *bad* for health, at least in the aggregate. As early as the work of William Ogburn and Dorothy Thomas (1922), it was noted that mortality in the United States was procyclical, with the apparently paradoxical finding that mortality rates are higher during booms than slumps. The result has been frequently but not uniformly confirmed in different times and places; perhaps the best-known study in economics is by Christopher Ruhm (2000), who uses time series of states in the United States. More recently, Ruhm (2015) grapples with the same data as ours, and questions whether it remains true that recessions are good for health. A frequent finding is that traffic fatalities are procyclical, as are the effects of pollution (Cutler, Huang, and Lleras-Muney 2016). In contrast, suicides are often found to be countercyclical. Ann Stevens and others (2015) find that in the United States, many of the deaths in "good" times are among elderly women, and implicate the lower staffing levels in care facilities when labor is tight; procyclical deaths from influenza and pneumonia show up in several studies, again suggesting the importance of deaths among the elderly. To the extent that the positive macroeconomic relationship between mortality and income is driven by mortality among the elderly, it makes it easier to tell a story of income being protective among middle-aged groups, such as those on which we focus here.

Our own interpretation is that there is likely some genuine individual-level positive effect of income on health, but that it is swamped by other macro factors in the aggregate. Of the results here, particularly those shown in figure 14, we suspect that the matching relationships are largely coincidental, as has happened in other historical episodes.

The argument for coincidence is well illustrated by disaggregating the top panel of figure 14 by cause of death. As shown in section I, when we look at all-cause mortality, we need to think about deaths of despair (suicides, overdoses, and alcoholism) together with heart disease. Deaths of despair have been rising at an accelerating rate since 1990; but, for a decade, they were offset by other declining causes of mortality, including heart disease. After 1999, the deaths of despair continued to rise, and they were now much larger, while the decline in heart disease slowed and eventually stopped, so that overall mortality started to go up. Both components are smooth trends, one rising and accelerating, the other falling but decelerating. Neither one in isolation has any relation to what has been happening to income; but together, they generate a turnaround that, by chance, coincides with the inverse U in family incomes. Spurious common Us are almost as easy to explain as spurious common trends.

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In the long history of the coevolution of health and income, such coincidences are not uncommon. The Industrial Revolution and Health Revolution that began in the 18th century both owe their roots to the Enlightenment and the Scientific Revolution, but neither one drove the other; see Richard Easterlin (1999) for a persuasive account. In developing countries today, health is largely driven by public action that requires money, but the use of that money for action on health is far from automatic and depends on policy (Deaton 2013).

A more recent episode comes after 1970 in the United States, when economic growth slowed while the rate of mortality decline accelerated rapidly. Mean real per capita personal disposable income grew at 2.5 percent a year from 1950 to 1970, slowing to 2.0 percent a year from 1970 to 1990; meanwhile, for men and women age 45–54 (for all ethnicities and races), the Human Mortality Database shows that all-cause mortality fell at 0.5 percent a year from 1950 to 1970, but at 2.3 percent a year from 1970 to 1990. Although the patterns of mortality vary by sex, the acceleration in mortality decline—from slowly between 1950 and 1970 to more rapidly between 1970 and 1990—characterizes both men and women separately, and all five-year age groups from 35–39 to 55–59. But neither the slowdown in income nor the increase in inequality that accompanied it had anything to do with the acceleration in mortality decline, particularly for heart disease, which was driven by the introduction of antihypertensives after 1970, later aided by statins, and by a decline in smoking, particularly for men. These health improvements were common to all rich countries, albeit with some difference in timing, and were essentially independent of patterns of growth and inequality in different countries (Deaton and Paxson 2001, 2004; Cutler, Deaton, and Lleras-Muney 2006). Although we do not consider it explicitly here, the fact that inequality and mortality moved in opposite directions speaks against the hypothesis that relative income—your income rising more rapidly than mine, or the success of the top 1 percent—drives mortality (Deaton 2003).

If we accept these arguments, we are left with no explanation for the mortality turnaround. We suspect that more likely causes are various slowly moving social trends—such as the declining ratio of employment to population, or the decline in marriage rates—and it is to these that we turn below. We note that it is difficult to rule out explanations that depend on long-run forces, such as the fact that those age 50 in 2010, as opposed to those age 70 in 2010, were much less likely to have been better off than their parents throughout their working life (Chetty and others 2017). Even so, we need to explain why stagnant incomes have this effect on whites but not on

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blacks. Perhaps the substantial reduction in the black/white wage gap from the mid-1960s to the mid-1970s gave an enduring sense of hope to African Americans, though there has been little subsequent reason in income patterns to renew it (Bayer and Charles 2016). Many Hispanics are markedly better off than their parents or grandparents who were born abroad. Yet none of this explains why being better off than one's parents should protect against income decline, though it is not hard to see why—after a working life at lower incomes than the previous generation—falling incomes at about age 50 might be hard to deal with. (This explanation works less well for younger age cohorts, who are also bearing the brunt of this epidemic, but who are not yet old enough to know whether they will be better off than their parents during their working lives.) The historian Carol Anderson argued in an interview for *POLITICO Magazine* (Glasser and Thrush 2016) that for whites, "If you've always been privileged, equality begins to look like oppression," and contrasts the pessimism among whites with the "sense of hopefulness, that sense of what America could be, that has been driving black folk for centuries." That hopefulness is consistent with the much lower suicide rates among blacks; but beyond that, though suggestive, it is hard to confront such accounts with the data.

## III. Cumulative Disadvantage

We have seen that it is difficult to link the increasing distress in midlife to the obvious contemporaneous aggregate factors, such as income or unemployment. But some of the most convincing discussions of what has happened to working-class whites emphasize a long-term process of decline, rooted in the steady deterioration in job opportunities for people with low education; see, in particular, the work of Andrew Cherlin (2009, 2014). This process, which began for those leaving high school and entering the labor force after the early 1970s—the peak of working-class wages, and the beginning of the end of the "blue-collar aristocracy"—worsened over time, and caused, or at least was accompanied by, other changes in society that made life more difficult for less educated people, not only in their employment opportunities but also in their marriages, and in the lives of and prospects for their children. Traditional structures of social and economic support slowly weakened; no longer was it possible for a man to follow his father and grandfather into a manufacturing job, or to join the union and start on the union ladder of wages. Marriage was no longer the only socially acceptable way to form intimate partnerships, or to rear children. People moved away from the security of legacy religions or the churches of their parents

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and grandparents, toward churches that emphasized seeking an identity, or replaced membership with the search for connection or economic success (Wuthnow 1998). These changes left people with less structure when they came to choose their careers, their religion, and the nature of their family lives. When such choices succeed, they are liberating; when they fail, the individual can only hold himself or herself responsible. In the worst cases of failure, this is a Durkheim-like recipe for suicide. We can see this as a failure to meet early expectations or, more fundamentally, as a loss of the structures that give life a meaning.

As technical change and globalization reduced the quantity and quality of opportunity in the labor market for those with no more than a high school degree, a number of things happened that have been documented in an extensive literature. The real wages of those with only a high school degree declined, and the college premium increased. More people went to college—a choice that, in practical terms, was not available to those lacking the desire, capability, resources, or an understanding of the expected monetary value of a college degree. Family incomes suffered by less than the decline in wages because women participated in the labor force in greater numbers, at least up to 2000, and worked to shore up family finances; even so, there was a loss of well-being, at least for some. Chetty and others (2017) estimate that only 60 percent of the cohort born in 1960 was better off in 1990 than their parents had been at age 30. They estimate that, for those born in 1940, 90 percent were better off at 30 than their parents had been at the same age. The data do not permit an analysis, but the deterioration was likely worse for whites than blacks, and for those with no more than a high school degree. As the labor market worsens, some people switch to lower-paying jobs—service jobs instead of factory jobs—and some withdraw from the labor market. Figure 18 shows that, after the birth cohort of 1940, in each successive birth cohort, men with less than a four-year college degree were less and less likely to participate in the labor force at any given age—a phenomenon that did not occur among men with a bachelor's degree.

It is worth noting again that the fractions with and without a bachelor's degree are constant for the cohorts born between 1945 and 1965, then rise from 30 to 40 percent for cohorts born between 1965 and 1970, beyond which the fraction remains stable at 40 percent. In consequence, some of the deterioration in outcomes for the less educated cohorts born between 1965 and 1970 may be driven by a decrease in their average positive characteristics; for example, if education is selected on ability, there will be a decrease in average ability in the group without a four-year degree. Yet this cannot

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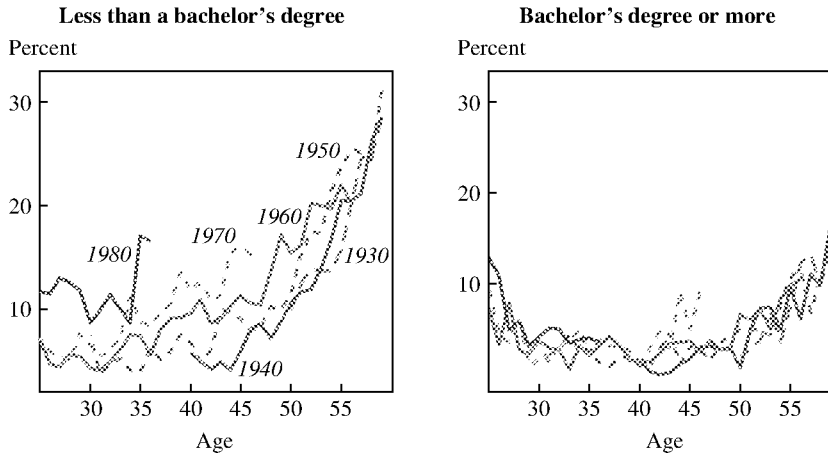
**Figure 18.** Percent of White Non-Hispanic Men Not in the Labor Force, by Birth Cohort




Sources: Current Population Survey, March supplement; authors' calculations.

be the whole story. Deterioration started for cohorts born in the 1940s and increased gradually with each birth cohort that followed. Moreover, if lower-ability people are transferred from the less to the more educated group, outcomes should also deteriorate for the latter; this is the *Will Rogers phenomenon*—that moving the most able upward from the bottom group brings down the averages in both bottom and top groups. Yet the cohort graphs show no evidence of deterioration among those with a bachelor's degree. Qualitatively, the same picture is seen when the education groups are pooled, providing an attenuated version of the left panel of figure 18 (online appendix figure 9).

Lower wages not only brought withdrawal from the labor force, but also made men less marriageable; marriage rates declined, and there was a marked rise in cohabitation, which was much less frowned upon than had been the case a generation before. Figure 19 shows that, after the cohort of 1945, men and women with less than a bachelor's degree are less likely to have ever been married at any given age. Again, this is not occurring among those with a four-year degree. Unmarried, cohabiting partnerships are less stable than marriages. Moreover, among those who do marry, those without a college degree are also much more likely to divorce than are those with a degree. The instability of cohabiting partnerships is indeed their raison d'être, especially for the women, who preserve the option of trading up (Autor, Dorn, and Hanson 2017)—so that both men and women lose the security of the stable marriages that were the standard among their parents.

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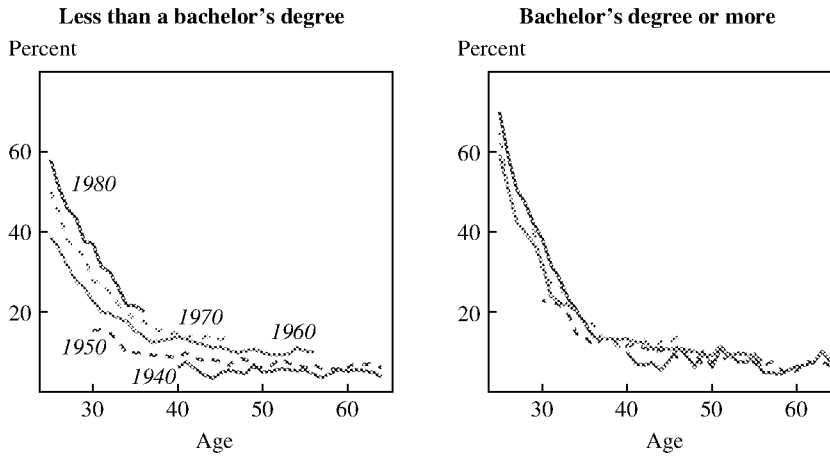
**Figure 19.** Percent of White Non-Hispanics Never Married, by Birth Cohort




Sources: Current Population Survey, March supplement; authors' calculations.

Childbearing is common in cohabiting unions, and again is less disapproved of than once was the case. But, as a result, more men lose regular contact with their children, which is bad for them, and bad for the children, many of whom live with several men during childhood. Some of a woman's partners may be unsuitable as fathers, and those who are suitable bring renewed loss to children when it is their turn to depart. It is particularly important that this behavior is more common among white women than among Hispanics or African Americans; the latter have more children out of wedlock, but have fewer cohabiting partners (Cherlin 2009). In Europe, cohabitation is also common, but is much less unstable, and not so different from marriage. Cherlin (2014) notes that it is now unusual for white American mothers without a college degree *not* to have a child outside marriage. The repeated repartnering in the United States is often driven by the need for an additional income, something that is less true in Europe, with its more extensive safety net, especially of transfer income; Britain, for example, provides unconditional child allowances that are attached to children.

Social upheaval may have taken different forms, on average, for African Americans. Black kin networks, though often looser, may be more extensive and more protective, as when grandmothers care for children. Black churches provide a traditional and continuing source of support. As has often been noted, blacks are no strangers to labor market deprivations, and may be more inured to the insults of the market.

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These accounts share much, though not all, with Charles Murray's (2012) account of decline among whites in his fictional "Fishtown." Murray argues that traditional American virtues—especially industriousness—are being lost among working-class white Americans. In this argument, the withdrawal of men from the labor force reflects this loss of industriousness; young men in particular prefer leisure—which is now more valuable because of video games (Aguiar and others 2017)—though much of the withdrawal of young men is for education (Krueger 2016). The loss of virtue is supported and financed by government payments, particularly disability payments (Eberstadt 2016). If this malaise is responsible for the mortality and morbidity epidemic, it is unclear why we do not see rising mortality rates for blacks, for Hispanics, for more educated whites, or indeed for Europeans, although this last group has universal health care and, again, a much more generous safety net. Indeed, in some European countries, disability programs are so generous and so widely claimed that average retirement ages are below the minimum legal retirement age (Gruber and Wise 2007).

According to Alan Krueger (2016), half the men who are out of the labor force are taking pain medication, and two-thirds of those take a prescription painkiller, such as an opioid. Doctors also bear responsibility for their willingness to (over)prescribe drugs (Quinones 2015; Barnett, Olenski, and Jena 2017), especially when they have little idea of how to cure addiction if and when it occurs. There are also reasonable questions about an approval system run by the U.S. Food and Drug Administration that licenses a class of drugs that has killed about 200,000 people. We should note that a central beneficiary of opioids are the pharmaceutical companies that have promoted their sales. According to Harriet Ryan, Lisa Girion, and Scott Glover (2016), Purdue Pharmaceutical had earned $31 billion from sales of OxyContin as of mid-2016.

In our account here, we emphasize the labor market, globalization, and technical change as the fundamental forces, and put less focus on any loss of virtue, though we certainly accept that the latter could be a consequence of the former. Virtue is easier to maintain when it is rewarded. Yet there is surely general agreement on the roles played by changing beliefs and attitudes, particularly the acceptance of cohabitation, and of the rearing of children in unstable cohabiting unions.

These slow-acting social forces seem to us to be plausible candidates to explain rising morbidity and mortality, particularly suicide and the other deaths of despair, which share much with suicide. As we have emphasized elsewhere (Case and Deaton 2017), purely economic accounts

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of suicide have rarely been successful in explaining the phenomenon. If they work at all, they work through their effects on family, on spiritual fulfillment, and on how people perceive meaning and satisfaction in their lives in a way that goes beyond material success. At the same time, increasing distress, and the failure of life to turn out as expected, are consistent with people compensating through other risky behaviors such as abuse of alcohol and drug use that predispose toward the outcomes we have been discussing.

### *III.A. A Framework to Interpret the Data*

A simple way of taking these stories to our data is to suppose that there is a factor that each birth cohort experiences as it enters the labor market. This might be the real wage at the time of entering; but it could be a range of other economic and social factors, including the general health of the birth cohort (Case, Fertig, and Paxson 2005); we deliberately treat this as a latent variable that we do not specify. This is related to accounts in which workers enter the labor market in a large birth cohort, or in bad times (Hershbein 2012, and the references provided therein). However, it is different, in that we emphasize the experience of all cohorts who entered the labor market after the early 1970s, and we focus on a secular deterioration of this initial condition.

We label birth cohorts by the year in which they are born—*b,* say—and assume each experiences $X^b$ as they enter the labor market, which then characterizes their labor market for the rest of their lives. Because of the factors outlined above, we might expect the effects to accumulate over time. But at this initial stage of the research, we assume that the disadvantage is constant for those in birth cohort *b* during their adult lives; we measure the factor as a *dis*advantage, which is natural for mortality, but requires reversing signs when we look at earnings. The driving variable $X$ is itself trending over time, though not necessarily linearly; our measurement will allow for any pattern. In this setup, various measures of deprivation—pain, mental distress, lack of attachment to the labor market, not marrying, suicide, addiction—will together move higher or lower as the initial condition $X^b$ goes up or down for later-born cohorts.

Figure 7, which inspired this way of thinking about the data, shows how this works for deaths of despair collectively, and for suicides, poisonings, and alcoholism separately; and figures 12, 13, 18, and 19 show the corresponding graphs for, respectively, self-reported health, pain, labor force participation, and marriage. For mortality and morbidity, we see an upward slope with age, which will be captured by a flexible age effect, with the age

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profile higher for each successive cohort, which we explain as an increase in the starting variable $X^b$. In this first analysis, we make no attempt to model the rotation of the age profiles that are apparent for some cohorts in several of these figures.

Our model for each outcome is then written as

$$y_{iab} = \alpha_i + f_i(a) + \theta_i X^b, \tag{1}$$

where $i$ indexes an outcome—suicide, pain, marriage outcomes; $b$ is the birth year; and $a$ is age. Each outcome is a function of age, shared by all birth cohorts for a given outcome, which will be estimated nonparametrically; $\theta_i$ is the parameter that links the unobservable common factor $X^b$ to each outcome $i$. The unobservable factor itself is common across outcomes. From the data underlying figures 7, 12, 13, 18, and 19, as well as for other conditions, we can estimate equation 1 by regressing each outcome on a complete set of age indicators and a complete set of year-of-birth indicators. We assume that the underlying cause of despair appeared after the 1940 birth cohort entered the market; we take this to be our first cohort, and normalize the driving variable $X$ to zero for this cohort, for all outcomes. The coefficient on the birth cohort indicator for cohort $b$ is an estimate of $\theta_i X^b$. Plotting these estimates against $b$ for each condition, we should see the latent cohort factor $X^b$, and we should see the same pattern, up to scale, for every outcome.

Figure 20 shows the results for each birth cohort born between 1940 and 1988, for WNHs age 25–64, without a bachelor's degree. The top panel presents estimates $\theta_i X^b$ for suicide, with its scale on the left; the scale for chronic joint pain, sciatic pain, mental distress, difficulty socializing, and heavy drinking is given on the right axis. (Obesity also shows a linear trend in year-of-birth effects. However, its scale is much larger, and its inclusion obscures the details of other morbidity measures.)

The bottom panel of figure 20 presents estimates for drug and alcohol poisoning, marriage (both never married, and not currently married) and, for males, not being in the labor force. We do not include alcohol-related liver diseases in this part of the analysis; the lag between behavior (heavy drinking) and mortality (cirrhosis, alcoholic liver disease) does not allow us to see the difference in the mortality consequences of heavy drinking between birth cohorts currently under the age of 50.

In the top panel of figure 20, the slopes formed by plotting $\theta_i X^b$ estimates are approximately linear for each outcome, consistent with a model in which the latent variable has increased, and increased linearly between

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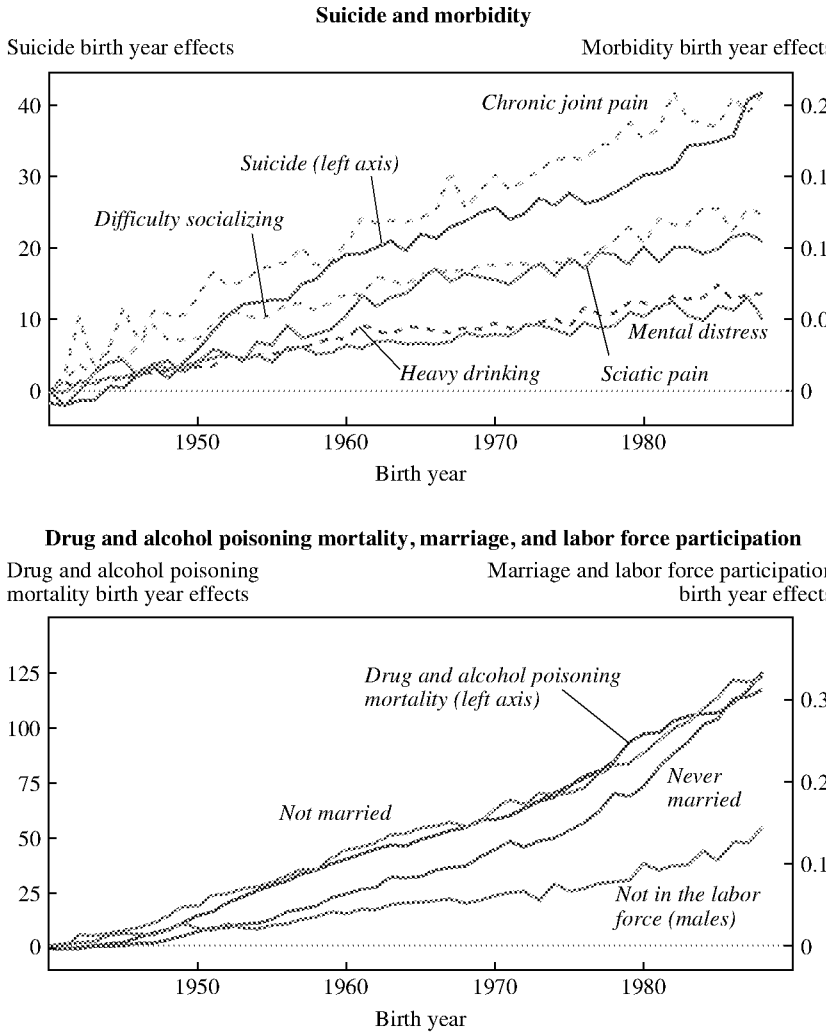
**Figure 20.** Mortality, Morbidity, Marriage, and Labor Force Participation, for Birth Years 1940–88




Sources: National Vital Statistics System; CDC National Health Interview Survey; Current Population Survey, March supplement; authors' calculations.

a. All lines except *Suicide* are measured on this axis.

b. All lines except *Drug and alcohol poisoning mortality* are measured on this axis.

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birth cohorts. For these conditions, we see that we can match the data by a common latent factor that increases linearly from one cohort to the next.

In the figure's bottom panel—for drug overdose, marriage, and labor force detachment—we see a somewhat different pattern, in which the common latent variable is "worse" than linear, with a slope that is increasing more rapidly for cohorts born after 1970 than for those born before. This is consistent with either a nonlinear effect of disadvantage on these outcomes, or the addition of a second latent factor that makes its appearance for cohorts born in about and after 1970, who would have entered the market starting in the early 1990s. As was true for suicide, pain, and isolation, each successive cohort is at higher risk of poor outcomes than the cohort it succeeded.

Note that there is nothing in our procedures that ensures that the plots in figure 20 must rise linearly, or even monotonically. That they do so is suggestive of an underlying factor at work, which may drive all these outcomes.

In a statistically inefficient but straightforward method, we can recover estimates of $X^b$ by pooling across conditions and regressing the logs of the estimated $\theta_i X^b$ coefficients on indicators for each cohort and each condition. The results confirm a nearly linear increase in $X$ across birth cohorts for suicide, heavy drinking, pain, and isolation, and a nonlinear increase for drug overdose, labor market attachment, and marriage.

One might reasonably ask what is causing what in our analysis. The use of a latent variable model allows us to avoid taking a position on this question. That said, we turn to the progressive deterioration of real wages as a possible driving variable. Figure 21 plots the (negative of) $\theta_i X^b$ coefficients from a regression of log real wages for men with less than a four-year college degree against coefficients from a regression of the percentage of men with less than a bachelor's degree who are not in the labor force.

The cohorts born between 1940 and 1988 show a decline in real wages that has become more pronounced with each successive birth cohort. This temporal decline matches the decline in attachment to the labor force. Here we also emphasize the cascading effects on marriage, health, and morbidity—and, ultimately, on deaths of despair.

Comparison figures for those with a bachelor's degree are provided in online appendix figure 10, where figures have been drawn on the same scales used in figure 20. Aside from being at risk for heavy drinking, which shows a pattern similar to those without a degree, those with a degree have seen much more limited changes in health, mental health, and marriage outcomes (with reports of pain, mental distress, and difficulty socializing between 0 and 2.5 percentage points higher in the birth cohort of 1980

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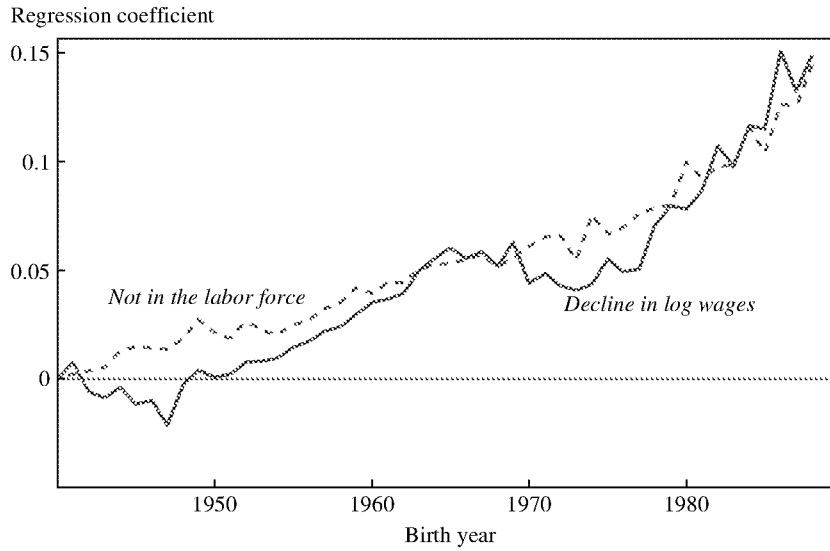
**Figure 21.** Log Wages and Labor Force Participation for White Non-Hispanic Men with Less Than a Bachelor's Degree, for Birth Years 1940–88




Sources: Current Population Survey, March supplement; authors' calculations.

relative to 1940), and flat profiles for labor force participation, suicide, and drug mortality. Controlling for age, real wages for those with a degree are on average 10 percent higher for the cohort born in 1980 relative to the cohort of 1940 (results not shown), while wages for those without a degree are 10 percent lower (figure 21).

What our data show is that the patterns of mortality and morbidity for WNHs without a college degree move together over birth cohorts, and that they move in tandem with other social dysfunctions, including the decline of marriage, social isolation, and detachment from the labor force. Figure 20 suggests that there may be two underlying factors, not one, but they are not very different, and we do not press that conclusion. Whether these factors (or factor) are "the cause" is more a matter of semantics than statistics, at least at this point. The factor could certainly represent some force that we have not identified, or we could try to make a case that the decline in real wages is the key. Behind this lie familiar stories about globalization and automation, changes in social customs that have allowed dysfunctional changes in patterns of marriage and childrearing, the decline of unions, and others. Ultimately, we see our story as about the collapse of the white

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working class after its heyday in the early 1970s, and the pathologies that accompany this decline.

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# Comments and Discussion

**COMMENT BY**

**DAVID M. CUTLER** This paper by Anne Case and Angus Deaton addresses one of the most important economic and demographic issues of our time: the rise of mortality among white non-Hispanics in the past 15 years. For some time, there has been scattered evidence that mortality rates have been increasing for certain groups of the population (Olshansky and others 2012; Meara, Richards, and Cutler 2008). Two years ago, in a widely cited paper in the *Proceedings of the National Academy of Sciences*, Case and Deaton (2015) brought the data together, documenting the systematic increase in mortality rates for white non-Hispanics since about the year 2000.

Their findings were shocking in two senses. First, mortality rates almost always decline over time. Between 1979 and 1999, for example, mortality rates for whites age 45–54 declined by 1.7 percent annually. In contrast, the mortality rates that Case and Deaton were looking at increased by 0.3 percent annually (this is for all whites, including Hispanics). Second, this pattern is dramatically different in the United States than in other rich countries. U.S. white non-Hispanics are becoming increasingly anomalous relative to their peers abroad.

A good deal of commentary was directed at these findings. Some discussions considered whether mortality rates were rising or were just flat (Gelman and Auerbach 2016). In the big picture, this is relatively immaterial—both historical trends and international comparisons lead one to expect declining mortality. Other discussions addressed whether the increase was largely confined to women, or was true for men as well (Achenbach and Keating 2016). The relative increase in mortality was greater for women than men, but both groups did poorly.



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The bigger issue, however, is about why these trends are occurring and what can be done to reverse them. What is it about the economic, social, or medical landscape that is leading to higher mortality for a very large segment of the population?

Case and Deaton address these issues in their current paper. Relative to their earlier paper, the current paper extends the analysis for an additional two years. Not surprisingly, the trends noted in the earlier paper have continued. More importantly, however, Case and Deaton make a first pass at why they believe mortality is rising.

By cause of death, the two biggest factors in the mortality reversal are the slowing down in mortality reductions from heart disease and the increase in "deaths of despair"—deaths due to drug and alcohol abuse and suicide. In their earlier paper, Case and Deaton suggested that the ready availability of opioid drugs might have exacerbated the increased mortality, especially that resulting from accidental overdoses. In their current paper, their emphasis has changed a bit. Rather than emphasizing the supply of pills, they now focus on the social and economic circumstances that lead people to take them.

Their overall suggestion is very much in the tradition of Émile Durkheim (1897): People despair when their material and social circumstances are below what they had expected. This despair leads people to act in ways that significantly harm their health. This may have a direct impact on death through suicide, or an indirect impact through heavy drinking, smoking, drug abuse, or not taking preventive medications for conditions such as heart disease. At root is economic and social breakdown.

This explanation is certainly correct. There is no way to understand the mortality pattern without considering the sources of despair, and the sources of despair must be very deep-seated indeed. Case and Deaton discuss where this despair may be coming from, and I suspect there is merit in their discussion here as well. That said, it is extremely difficult for researchers to get at all the aspects that lead individuals to be living a life that they value less than one would hope they would. Case and Deaton suggest that despair starts early in life, at the time of entering the labor force or before, as expectations about what a "middle-class life" should involve. They distinguish this from a theory that focuses only on current income, which they say cannot explain all the data because the median incomes of blacks and Hispanics have been trending in parallel to those of white non-Hispanics; yet these groups have not seen the worsening mortality rates experienced by white non-Hispanics. Again,

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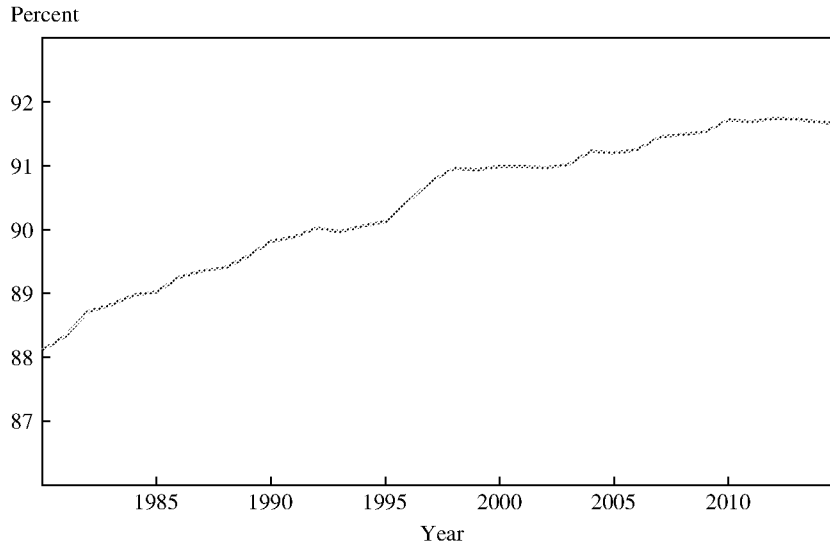
**Figure 1.** Percent of People Surviving from Age 40 to 60, 1980–2015




Source: National Center for Health Statistics.

I am tempted to believe this, though the evidence for any particular view about how expectations are formed and what income shocks imply is not as clear as one would like it to be.

In this comment, I pick up three parts of Case and Deaton's findings and interpretation: the age groups to which these changes are occurring; the extent to which expectations are set early in life; and changes that may be due to a greater ability to translate pain into death.

THE AGES AT WHICH MORTALITY PATTERNS CHANGE Let me start with the first issue, the age pattern for which there have been changes in mortality. Case and Deaton highlight the working-age population, roughly people from age 30 until about 60. Mortality reductions have been slowing greatly for this group. My figure 1 shows this another way, plotting the share of people surviving from age 40 to 60. In 1980, about 88 percent of people survived from age 40 to 60. By the late 1990s, the share was about 91 percent. Since then, the increase has been very modest.

However, the situation is quite different for the elderly. My figure 2 shows an international comparison of life expectancy at age 65. The United States is again a negative outlier; life expectancy in the United States has increased less rapidly than in other countries. That said, there has been a sustained increase in life expectancy for the U.S. elderly over time. Indeed,

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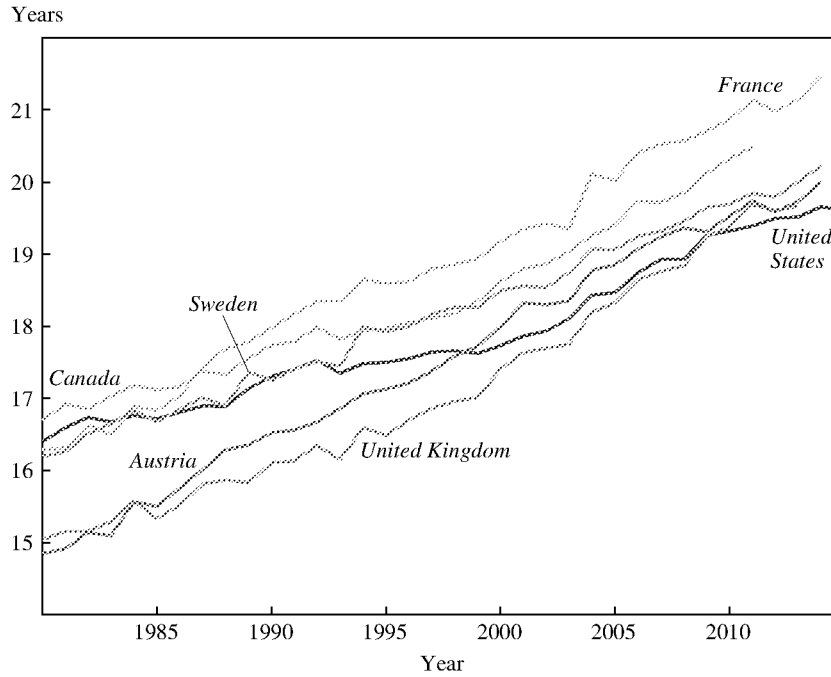
**Figure 2.** Life Expectancy at Age 65, 1980–2015




Source: Human Mortality Database.

life expectancy for the elderly U.S. population has actually increased since the late 1990s, in contrast to the nonelderly population. What is happening for the working-age population is not the same as what is happening for the elderly population.

My figure 3 shows this more directly. The figure plots the relative change in mortality for white non-Hispanics from 1999 through 2015 for each five-year age group from 40–44 to 70–74. Mortality in 1999 is normalized to 1; the line then traces out how mortality changes for each cohort relative to that base year.

For people in their prime working-age years, mortality has actually increased over time. For example, people age 40–44, 45–49, and 50–54 have all seen mortality rates rise relative to their value in 1999. This is the fact that Case and Deaton identify. Similarly, for people age 55–59, the net change in mortality has only been a small decrease.

However, as one proceeds to older ages, there is more of a sustained mortality reduction. For people age 60–64, there has been a marked,

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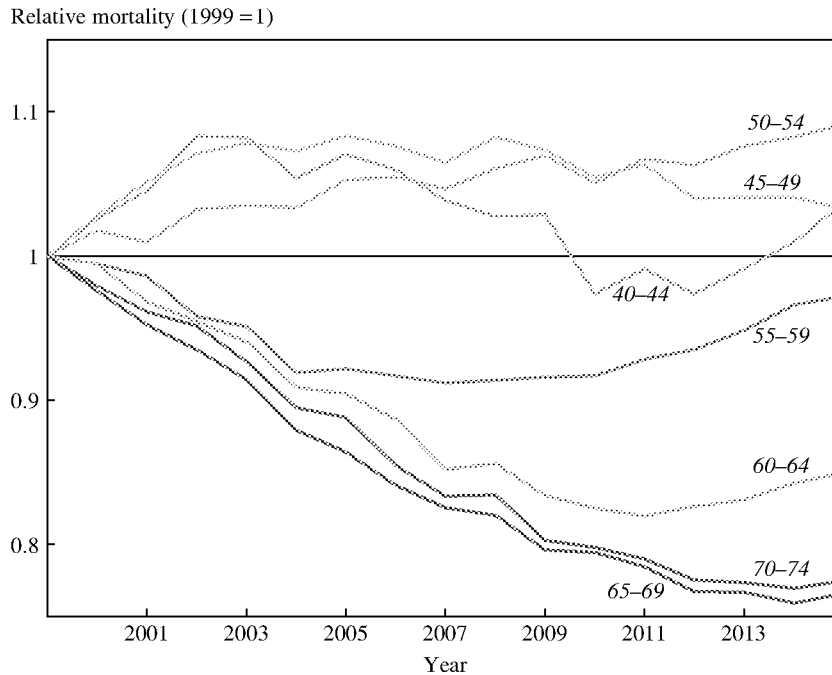
**Figure 3.** Relative Mortality for White Non-Hispanics, by Age Cohort, 1999–2015




Source: National Center for Health Statistics.

continuing mortality decline until very recently. There is a modest increase in mortality beginning in about 2011, but the overall change is still a reduction of about 15 percent. The declines are even greater for older groups. People age 65–74 have seen mortality reductions on the order of 25 percent.

The fact that the mortality pattern for the elderly differs so much from that for the working-age population suggests several possible explanations. The first is a cohort interpretation: Some cohorts are experiencing worse mortality than their predecessors were, and this mortality change will persist throughout their lifetime. I suspect this is not entirely the case, because the groups that would have experienced increased mortality from age 40 to 55 are now at an older age, and their mortality rates have not increased anywhere near the extent we saw at younger ages. To be sure, we can see a bit of a reflection of the mortality increase in the older population (witness the mortality for the group age 60–64 since 2011), but there are clearly also other factors.

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These factors may be either age or year effects. That is, the middle ages may be particularly bad for health (age effects) or we may have made greater progress against the diseases that kill people at older ages (year effects). Respiratory impairment, which largely affects people at older ages, is a possible example. As is well known, there is no way to tell age, period, and cohort effects apart.

I want to propose a hypothesis that I find intriguing, which is related to the idea of age effects. The hypothesis is that many of the economic and social changes that make midlife stressful for so many people dissipate as one nears the traditional retirement age. For example, many people are experiencing wage reductions in middle age along with the loss of guaranteed pensions and health insurance. However, retirement programs such as Social Security and Medicare help people maintain a standard of living from age 65 (or 62) on. It may be that the guarantees of Social Security and Medicare provide a level of security that allows people to enjoy a healthier life.

Testing this explanation is extremely difficult. One test, if it could be done, would be to look at life satisfaction. Examining how life satisfaction varies by age and year seems like a very good test of the despair hypothesis.

ECONOMIC OR SOCIAL DECLINE Case and Deaton discuss several measures of social and economic change, suggesting that they highlight despair. There is surely merit in these measures. But it is also worth trying to unpack the different possible sources of despair in more detail. One does not always need to understand the source of a problem to fix it; but in this case, one does.

One central question is how much of these changes is driven by the decline in stable manufacturing jobs. In many of the anecdotal accounts that one reads (Vance 2016; Alexander 2017; Goldstein 2017), it is the decline in stable, middle-class jobs that leads to many of the other social ills. Alternatively, one could tell a story of social isolation that results from changes in the quality of high school education, changes in marriage rates that stem from reduced income at young ages, changing social norms about reproduction and marriage, or any of a host of other explanations.

Relatively little work has been done on this. There is an intriguing paper by Justin Pierce and Peter Schott (2016) showing that areas that were exposed to more trade from China had greater increases in deaths from opioid overdose. However, those effects are relatively modest and could not explain the magnitude of the findings that Case and Deaton document.

A good way to test these explanations is to look at more detailed geographic data. Case and Deaton show that the increase in mortality starts in

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different periods in different areas of the country. Thus, one might be able to match up the mortality trend with area-specific economic changes.

In work with Raj Chetty and others (2016), we were able to get at this a little bit. We calculate measures of life expectancy at age 40 for different income groups in the population, divided into roughly 700 commuting zones. We have life expectancy data from 2001 through 2014. We correlated life expectancy conditional on income with a number of measures of economic and social change. For this purpose, I highlight a few results from the correlation with life expectancy for the bottom quintile of the population.

There is a strong correlation between life expectancy at age 40 and measures of adverse behaviors: smoking, drinking, and being overweight. This is what one would expect. What is more interesting, however, is that relatively few economic and demographic factors are highly correlated with life expectancy at age 40. In particular, unemployment rates in 2000 or 2010, the change in labor force participation between 1980 and 2000, and the change in manufacturing jobs during the same time period were uncorrelated with life expectancy.

To be sure, life expectancy for low-income people was particularly low in the industrial Midwest. West Virginia and eastern Kentucky lead the nation in opioid-related mortality. However, the change in life expectancy has also been very poor for some areas that are growing, such as Florida and Nevada. Economic change does not explain why these areas are doing particularly poorly in health terms. And opioid-related deaths are also very high in New England, which has low unemployment and a good jobs base. Future research using these and other data sets may allow us to understand why mortality has followed the pattern it has.

FROM DESPAIR TO DEATH The final issue I want to highlight is what happens to people who are in despair. Many anecdotal accounts of early deaths start with accounts of pain. People have various physical and mental health impairments—back pain, joint pain, depression, anxiety, and so on. Before opiates were commonly available, such pain was often not treated medically. There were some painkillers, such as Vioxx (rofecoxib), but that was withdrawn in 2004. I suspect that many people smoked or drank heavily to relieve the pain.

The crux of the revolution in the treatment of pain was the widespread availability of oxycodone, a molecule similar to morphine and heroin in its impact on the brain. Oxycodone was billed as nonaddictive, but this does not seem to be true (Van Zee 2009). People become tolerant to a dose that they are taking, and then find they need to take more to achieve the same impact. This "taking more" can consist of higher doses of prescription pain

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relievers, or illegal substances such as heroin—the street cost of which is much lower.

Heavy drinking and smoking can kill people, but it takes a long time. Addiction can kill much sooner. The net effect may thus be an increase in the extent to which despair can lead to death in the short term. Indeed, it may even be that some of the deaths caused by opioids would not have occurred without these medications. Temporary despair can lead people to take pain relievers, to which they then become addicted. The despair might have ended on its own, but the addiction becomes permanent.

It is not entirely clear what policy remedies are appropriate in this situation. But this explanation does suggest focusing a little bit more on the supply side than just on the demand side. That is, reducing access to legal and illegal opioid drugs may reduce the extent to which short-term despair leads to both temporary and permanently elevated mortality rates.

In the end, I come back to the question of remedies. So far, the market has not been able to provide a stable income and social circumstance that people value highly enough to make them want to strive for a long life. If the market cannot do so, maybe the government should do more.

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**COMMENT BY**

**ADRIANA LLERAS-MUNEY** Life expectancy in the United States and most developed countries has been increasing for the last 150 years rather steadily. But life expectancy at birth in the United States declined in 2015 for the first time since 1994. Although small declines have been observed before, Anne Case and Angus Deaton document a disturbing set of facts. Mortality rates among middle-aged, white non-Hispanics have been rising since 2000, in sharp contrast to what is happening to the mortality rates of other populations, such as Europeans of the same age. Death rates from suicide, drugs, and alcohol consumption are rising. Death rates from cardiovascular disease are no longer decreasing. Moreover, pain, disability, and other measures of physical and mental health have been worsening. These increases in mortality and poor health are concentrated among whites without college degrees—in fact, mortality among those with a college education continues to fall.

Case and Deaton show some new, remarkable patterns that suggest life expectancy will continue to fall. When plotted by birth cohort, one finds that among white non-Hispanics without a college degree, the age profile of mortality is getting steeper for each successive cohort; for more recent cohorts mortality at a given age is higher, and it rises faster with age. The same is true for measures of disability or disease; health is deteriorating faster with age for younger cohorts. Case and Deaton further hypothesize that the decline in health and longevity could be caused by worsening labor market conditions for cohorts entering the labor market in 1970 or later.

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To better understand the findings of this paper, I investigate possible underlying causes of these patterns using a model I developed with Flavien Moreau (2017). It is a simple model of evolution of health and death from birth onward. In their simplest form, mortality patterns are determined by five parameters. I estimate this model for the 1940 cohort, using cohort life tables from the Social Security Administration, and show that it can accurately reproduce lifetime mortality rates and life expectancy. I then investigate whether changes in the baseline parameters can generate patterns of mortality and morbidity similar to those documented by Case and Deaton.

Just like Case and Deaton, I conclude that at least two forces could account for their findings. First, lifetime health resources—either their level or the rate of increase—could be falling across successive cohorts. Second, the rate of health depreciation (the rate at which people age) could be increasing. Either of these factors would generate steepening mortality and disability age profiles. It is particularly important that these patterns cannot easily be explained by temporary conditions; in the model, they can only be the result of permanent changes in parameters beginning early in adulthood (or even earlier in life). This is the same conclusion that Case and Deaton reach from their nonparametric analysis.

To assess the likelihood of each of these hypotheses, I use evidence from the literature to speculate about the root causes of these changes in mortality. Stalling or falling real lifetime incomes, in combination with increasing costs of health inputs, could rationalize lower health resources. Increases in lifetime exposure to pollutants, or increases in stress (due to, for example, declining intergenerational mobility or greater inequality), could also potentially be linked to increases in the depreciation rate. Although a full evaluation of the empirical validity of these hypotheses is beyond the scope of this comment, the discussion suggests several directions for future research. I end by commenting on the results vis-à-vis education levels and reflecting on possible policy implications.

A SIMPLE MODEL OF HEALTH AND MORTALITY ESTIMATED FOR THE UNITED STATES This section draws heavily on the model of Lleras-Muney and Moreau (2017). In the baseline model, the population is born with a given level of health, $H_0$, which is normally distributed. Then, during every period, health deteriorates, due to wear and tear. This deterioration is increasing with age, rather than constant. But individuals can enhance their health stocks by devoting resources to their health. These resources, $I$, are identical for all individuals in a population and

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are constant over their lifetimes. But individuals within the population are subject to independent and identically distributed shocks, $\varepsilon_t$, every period; some get higher than average resources, and some get lower than average resources. Finally, individuals die when their health stock reaches a lower bound, $\underline{H}$.

More precisely, a cohort's health and mortality can be characterized by the following dynamic system:

$$H_0 \sim \mathcal{N}(\mu_0, \sigma_0^2)$$

$$H_t = H_{t-1} - \delta t^{\alpha} + I + \varepsilon_t$$

$$\varepsilon_t \sim \mathcal{N}(0, \sigma_\varepsilon^2)$$

$$MR_t = P(H_t < \underline{H} \mid H_{t-s} > \underline{H}, \forall s < t-1)$$

where $\delta \in (0, \infty)$, $\alpha \in (0, \infty)$, and $I \in \mathbb{R}$.

In this model, mortality falls rapidly at young ages because those with initially low levels of health die in the first periods. But if $I$ is sufficiently high (relative to the depreciation rate, $\delta$), then the distribution of health moves away from the threshold and causes mortality to plummet to very low levels by adolescence. But because the rate of depreciation increases with age, eventually health starts to fall and mortality increases. After normalization,[1] this model describes health and mortality at every age using only five parameters: one for initial conditions, $\mu_0$; two that govern the aging process, $\delta$ and $\alpha$; and two that characterize the health resources provided by the environment, in the form of average investments, $I$, and the variance of these investments or shocks, $\sigma_\varepsilon^2$.

This model is a very simplified version of reality. It does not account for accidents. It also does not allow for optimization: Here, $I$ is a constant provided by the environment, which is assumed to be stationary. Lleras-Muney and Moreau (2017) investigate many of these extensions. But here I use this model because it provides a remarkably good baseline; using only five parameters, it can match the basic age profile of mortality we observe in the Human Mortality Database for many populations. I use it to study the possible factors behind the deterioration in white Americans' health and longevity.

ESTIMATING THE MODEL FOR THE UNITED STATES I validate this model by estimating the parameters for the 1940 birth cohort, using cohort tables

1. Two parameters are not identified; we arbitrarily set $\underline{H} = 0$ and $\sigma_0^2 = 1$.

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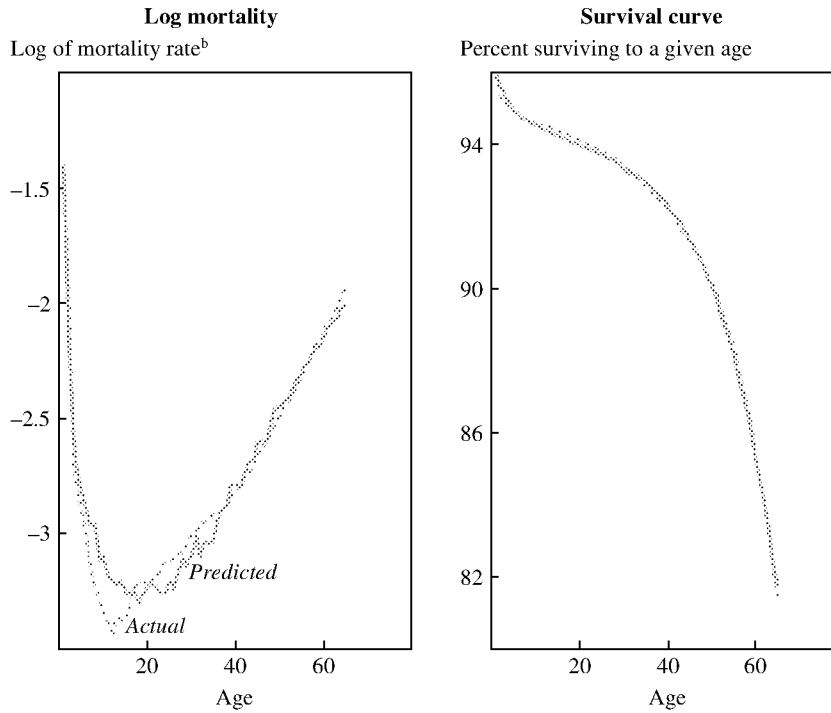
**Figure 1.** Mortality and Survival Profiles for U.S. Females Born in 1940[a]




Sources: Bell and Miller (2002); Lleras-Muney and Moreau (2017); author's calculations.
a. The estimated parameters are $I = 0.0554$, $\delta = 0.0012$, $\sigma_\varepsilon = 0.1515$, $\alpha = 1.3049$, $\mu_0 = 1.7424$, and $r = 1.0224$.
b. Mortality rates range from 0 to 1, and are approximately equal to the number of deaths at a given age divided by the number of people alive at that age. Log base 10 is used.

provided by the Social Security Administration (Bell and Miller 2002, table 7). Because cohorts born after 1940 experienced robust GDP growth, I estimate a slightly extended version of the model outlined above, which has a sixth parameter, $r$: $I$ is assumed to be increasing during every period at a constant rate, $r$, which also is to be estimated. This model cannot be solved in closed form, so estimates are obtained using the simulated method of moments by minimizing the errors in predicted survival rates at each age.

My figure 1 shows the results of this exercise for U.S. females. The left panel shows the log of the observed and the predicted mortality rate. The right panel shows the predicted and observed survival rates. Although the model does not perfectly predict some important

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features of the data (for instance, the exact level of mortality during reproductive ages), the model matches the basic shape of mortality very well. Moreover, it predicts life expectancy (up to age 65) of 60.5080 years for this cohort, compared with the actual life expectancy of 60.5084 years.[2]

The estimates show that initial health starts 1.74 standard deviations away from the "death threshold" and that the annual shock is equivalent to 0.15 standard deviation of the initial health distribution. The baseline health investment $I$ is equal to about 3 percent (0.0554 ÷ 1.7424) of the initial stock of health. Interestingly, the rate of growth of $I$ is estimated as 2.24 percent, which is remarkably close to the growth of U.S. GDP over the last century (Jones 2016).

PREDICTING AND EXPLAINING TRENDS IN U.S. WHITE NON-HISPANIC MORTALITY PROFILES I now use this model to investigate whether changes in any of the parameters can generate the patterns documented by Case and Deaton.[3] I simulate the effect of changes in the key parameters of interest starting at age 20 for both mortality and disease rates. To simulate disease rates, I assume that individuals are sick if they are alive but their health falls below some arbitrary threshold.

My figures 2 and 3 show the results of the simulation for mortality and morbidity. Three types of changes can rationalize Case and Deaton's findings: (i) a decrease in the baseline level of annual health investment, (ii) a decrease in its annual rate of growth, or (iii) greater depreciation; these three changes result in steeper age profiles for both mortality and disease rates (my figure 2). Note that in all cases, the effects of changing the parameters on mortality are almost imperceptible between age 20 and 40. These effects materialize later in life and grow with age.

Changes in other parameters cannot explain the findings. Increasing the accident rate, the variance of resources, or the death thresholds results in patterns for mortality and morbidity that differ from what we observe (my figure 3). If we allow for an exogenous increase in random accidents,

2. The estimated parameters for men are $I = 0.0546$, $\delta = 0.0012$, $\sigma_\varepsilon = 0.1534$, $\alpha = 1.3022$, $\mu_0 = 1.6078$, and $r = 1.0207$. The fit is good for men, but not quite as good as for women. This is because the 1940 male cohort has substantially higher mortality during reproductive ages that we cannot account for in the baseline model I am using here. Lleras-Muney and Moreau (2017) estimate models that successfully account for the hump in mortality.

3. I do not attempt to match the exact rate of change across cohorts here, only to provide suggestive evidence on which factors may be worthy of further investigation. Thus the estimated parameters were not chosen to match any cohort other than the 1940 cohort.

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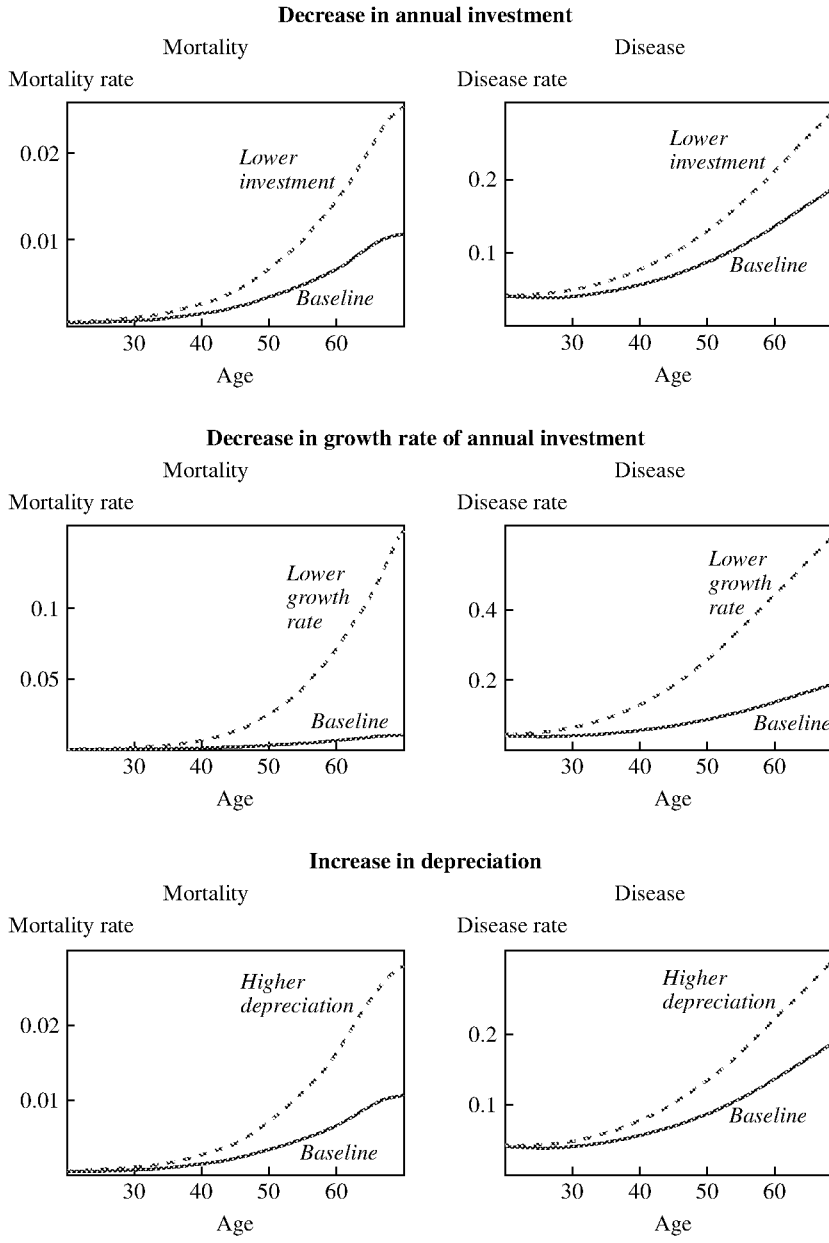
**Figure 2.** Factors That Generate Steeper Age Profiles in Mortality and Disease




Sources: Bell and Miller (2002); Lleras-Muney and Moreau (2017); author's calculations.

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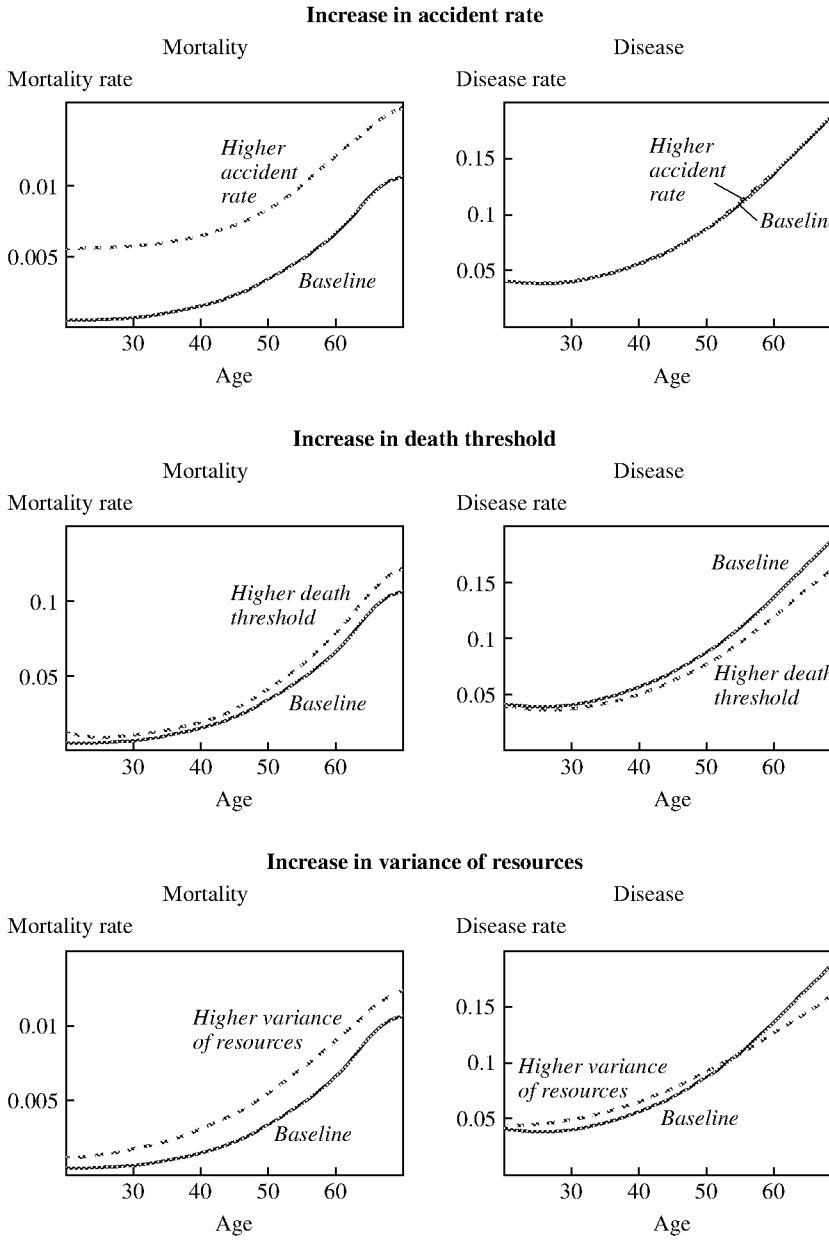
**Figure 3.** Factors That Cannot Explain Changes in Mortality and Morbidity Age Profiles




Sources: Bell and Miller (2002); Lleras-Muney and Moreau (2017); author's calculations.

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mortality increases, but its slope is unchanged. And disease rates are identical (because accidents do not kill individuals on the basis of their health levels). If we increase the threshold for dying, mortality increases at all ages, but again the age slope of mortality is unchanged. Moreover, disease rates fall, because the frailest individuals are dying. Finally, if we increase the variance of annual resources, then mortality becomes less steep and disease rates fall.

A few comments about these simulations are in order. First, I only simulate the effect of permanent changes starting at age 20 and lasting until death, rather than temporary shocks at age 20. Lleras-Muney and Moreau (2017) simulate the effects of temporary changes (lasting 10 years and then ending) at age 20—the patterns we observe in these simulations differ substantially from those shown here; after the shock ends, mortality starts reverting to its counterfactual level. We cannot generate steepening age profiles with temporary shocks.

Second, although changes in these parameters at birth would cause similar patterns, the data suggest that it is unlikely that conditions before age 20 are responsible for the declines in adult mortality we observe. Infant mortality was falling for all these cohorts (CDC 1999, table 1). Educational attainment stalled for men and grew for women born after 1950, though at a much slower pace than for cohorts born before the war (CBO 2011, figure 5; Goldin and Katz 2007a). People's height increased throughout the period, although again at a decreasing pace for those born after 1950.[4] These three measures—infant mortality, height, and education—are excellent indicators of initial conditions and early investments, and they are highly predictive of mortality in adulthood. These indicators did not decline after 1950, and thus early factors are not likely explanations for the increases in mortality.

Relatedly, the simulations assume that the entire profile of mortality is identical up to age 20, but this is not the case in reality. Janet Currie and Hannes Schwandt (2016a, p. 708) report that from 1990 to 2010, "For children and young adults below age 20, however, we found strong mortality improvements that were most pronounced in poorer counties." The fact that mortality rates before age 20 were falling for cohorts born after 1950

4. For white men, height increased by more than 4 centimeters for birth cohorts born between 1910 and 1950, but only grew by 1 centimeter for those born between 1950 and 1980 (Komlos and Lauderdale 2007). For women, the increases are 2.1 centimeters and 1.3 centimeters, respectively. Data from other sources suggest similar patterns (Bleakley, Costa, and Lleras-Muney 2014).

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suggests that initial conditions are not constant across birth cohorts. In our model, this would result in the entire profile of mortality shifting downward, and thus lower mortality in middle and old age. A proper evaluation of any explanation needs to carefully consider changes in conditions before entry into the labor market. I expand on this issue below.

*Decline in annual health investments.* The simulation results suggest that lower lifetime health resources, $I$, could generate the observed patterns. Could health resources be lower for more recent cohorts? Note that in the model, $I$ does not correspond to current income; it is expressed in health units. But health cannot be directly consumed or increased—it must be produced. Consider, then, the simplest case, where $I$ is produced using inputs $x$, which must be purchased at price $p_x$. Suppose that a constant share of one's lifetime income $a$ is spent on health at any given age and used to produce health: $I = f(x) = f(aY/p_x)$. What this suggests is that resources could be going down if either (i) lifetime incomes are falling (holding prices constant), or (ii) the price of health inputs is rising (holding incomes constant).

Data on lifetime income by cohort are difficult to find. A very recent paper by Fatih Guvenen and others (2017) uses data from the Social Security Administration that track individuals' earnings over time. It reports that lifetime income stagnated or fell for men entering the labor market in 1967 and later, and this is mostly explained by a decline in incomes upon entry into the labor market. The lifetime incomes of women did rise, though starting from a lower baseline and never reaching the level of men. It is unclear, then, what has happened to lifetime family incomes, but it is possible that they fell. More significantly, the price of health-related goods and services has increased very substantially over time, at a much faster pace than the cost of other goods and services, starting in the late 1970s.[5] Thus, in real "health" terms, incomes could be much lower for those at the bottom of the income distribution or those with less education.

Case and Deaton downplay income as an explanation. But they consider only contemporary correlations in incomes and mortality, rather than correlations in lifetime resources and adult mortality. In our model of health and mortality, one year's (temporary) changes in the parameters have very small effects on contemporary mortality. However, sustained (permanent) changes have effects that are not visible immediately, but become apparent after a substantial delay, as shown in my figure 2. Assessing whether life-

5. According to the U.S. Bureau of Labor Statistics, the cost of medical goods rose many times faster than the cost of other goods (Reed 2014).

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time health resources fell for cohorts entering the labor market after 1970, particularly for those with low education, seems worthy of further exploration. It requires a much more in-depth analysis than is provided here. It requires careful tracking of households (for example, who is married to whom and how many dependents they have), of family and governmental transfers (taxes and subsidies), and of the prices of health inputs (like exercise and medical care). Equally important, one needs a model that allows for dynamic (delayed) effects of conditions at a point in time, and that accounts for differences in initial conditions.

*Increase in depreciation (aging) rates.* What might cause higher deterioration rates or faster aging? The medical literature suggests several hypotheses. For instance, repeated exposure to stress cumulates and eventually leads to permanent changes in the functioning of the immune system (among others), a process known as "allostatic load" (Sapolsky 1994). These processes have been documented experimentally in animals. It is possible that cohorts entering the labor market in the 1970s and after would have experienced increasing levels of stress. This stress could be caused by their lower wages upon entry into the labor market. Raj Chetty and others (2017) show that cohorts born after the 1940s were less likely to do better than their parents. Perhaps these cohorts suffer stress by falling short of their expectations, as suggested by David Cutler in his comment. The changes in inequality that started in the late 1970s could also be hypothesized to lead to increased stress among these cohorts. The stress hypothesis also seems worth investigating, particularly given the "deaths of despair": alcohol and drug abuse suggests that individuals are unhappy.

Pollution (air, water, and food toxins) can also result in accelerated aging. This hypothesis is supported by animal models (Sun and others 2005), but is difficult to demonstrate in humans. The use of fossil fuels has increased steadily since 1900, and though some pollutants have been regulated since the 1970s, there are more than a thousand toxins emitted into the air and the water, and most are not regulated. For instance, PM 2.5 (that is, particulate matter with a diameter of 2.5 microns or less) has recently been linked to many diseases, but has been regulated only since 2007. Mercury, another highly toxic pollutant, has only been regulated since 2011. Thus, more recent cohorts may have accumulated substantially higher lifetime exposure to pollutants than cohorts born before the war. Moreover, exposure to pollutants is higher for those from backgrounds of lower socioeconomic status (SES). For example, poor individuals with low education are more likely to live close to highways and Superfund (hyperpolluted) sites (Currie 2013). A careful analysis of the pollution hypothesis

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needs to account for differences in lifetime exposure by race, location, and birth cohort. Because pollution has been shown to affect the human cardiovascular system in the short term, its long-term effects seem worthy of further investigation, particularly in light of the fact that cardiovascular mortality rates are no longer falling among adults.

Case and Deaton point to increases in obesity and diabetes as possible explanations, and indeed these are chronic conditions that could result in the type of effects we observe. There are many other possible factors that could also affect aging—for instance, physical activity.

IN LIGHT OF EDUCATION The increase in age-adjusted mortality for white non-Hispanics as a whole is modest compared with the increase in mortality experienced by those with less than a college degree. Several papers have documented that the gap in life expectancy between those with a college education and those without has been rising since the 1960s (Meara, Richards, and Cutler 2008; Montez and others 2011). Case and Deaton show that since 1998, mortality rates have fallen for those with college degrees, while increasing for those without.

This widening gap does not appear to be caused by a change in the composition of those with more education. Although there have been increases in the share of individuals holding a college degree, these increases have been small for cohorts born after 1950, particularly for men. The share of college graduates has been roughly constant for men born after World War II, and for women born after 1970.[6] Nevertheless, the composition of the pool could be changing despite roughly constant shares. For instance, there could be increasing selectivity in college admissions on the basis of test scores.[7] But previous papers investigating this issue have concluded that changes in the composition or behaviors of this pool do not appear sufficient to explain the growing gap in life expectancy by education (Cutler and others 2011). Rather, the "returns to college" in terms of health appear to be on the rise.

6. For working men born in 1950, years of completed education (measured at age 25–29) were 13.4 compared with 13.2 for those born in 1980. For working women, average completed years of education were 13.3 for the 1950 cohort, 13.9 for the 1970 cohort, and 14.1 for the 1980 cohort (CBO 2011).

7. Case and Deaton repeatedly note that the share of college graduates has remained unchanged, and argue this rules out changes in composition or selection as an explanation for changes in mortality. While constant shares are suggestive, they are neither necessary nor sufficient to guarantee that the pool of college graduates has remained similar over the last 50 years. For instance, college slots could be given by lottery in one year but allocated according to entrance exams in another. The same fraction of people would be accepted into college in both years, but selection (the type of individuals in college) would be vastly different.

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The labor market returns to college have also been steadily rising since the 1970s, when they reached their lowest point in the century (Goldin and Katz 2007b). David Autor (2014, p. 843) reports that "the earnings gap between college and high school graduates has more than doubled in the United States over the past three decades." For men without a college degree, median wages have declined since 1979. Women without a college degree have seen improvements in their median wages, but they started at a lower level and again have not yet caught up to men (CBO 2011, figure 3; Autor 2014). The literature looking at the "college premium" has concluded that its rise is likely due to the increase in demand for college workers, rather than changes in the composition of college workers. Again, it is difficult to estimate the changes in lifetime resources vis-à-vis education, because this requires accounting for marriage and fertility patterns in relation to education, as well as transfers and changing prices. But the evidence does suggest that the lifetime resources of the less educated may have fallen, while increasing for those with college degrees. Altogether, deteriorating wages upon entry into the labor market provide a parsimonious explanation for the findings.

THE IMPORTANCE OF ACCOUNTING FOR INITIAL CONDITIONS Case and Deaton contrast the experiences of blacks and Hispanics with that of whites in their search for explanations, and they use the comparison as another piece of evidence against the income explanation. Though blacks' changes in current income tracked that of whites, black mortality was still decreasing while white mortality was increasing, at least until 2010. But blacks, whites, and Hispanics have markedly different levels of and trends in childhood mortality. This makes the comparisons across groups difficult to interpret, because improvements in health conditions have delayed effects.

Black infant mortality in 1940 was much higher than that of whites, and it fell much more in levels (though not in percentage terms). More generally, mortality before age 20 has fallen more for blacks than for other groups (Currie and Schwandt 2016b). All else equal, these improvements early in life lower mortality throughout a person's remaining life—*particularly after age 40.* In our model, a population with higher initial health will have lower mortality throughout the lifetime (Lleras-Muney and Moreau 2017). There is also ample empirical evidence showing that early conditions have long-lasting consequences for health and mortality later in life. For instance, it is well established that conditions in utero affect mortality after age 45 (Almond and Currie 2011; Almond, Currie, and Duque 2017). Perhaps middle-aged blacks are still reaping the health benefits of improving

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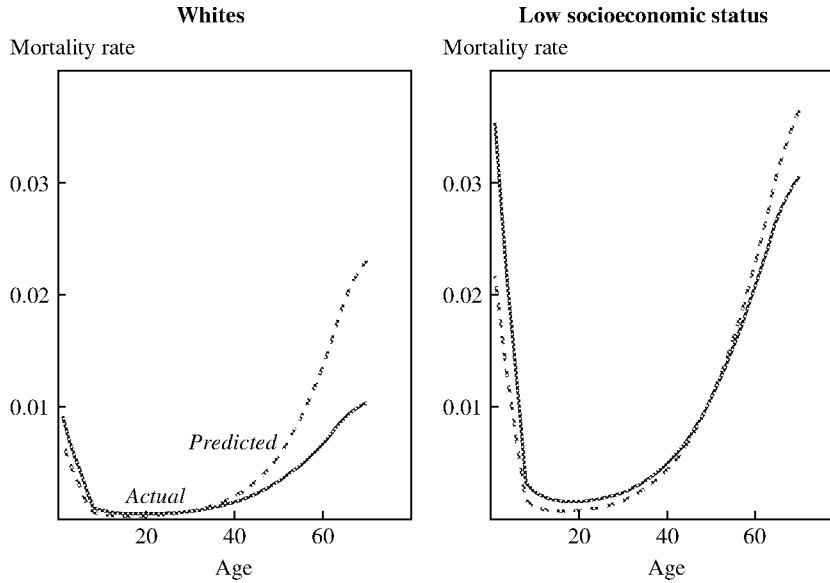
**Figure 4.** Simulated Mortality Rates Assuming Improvements in Initial Conditions and Decreases in Annual Resources at Age 20




Sources: Bell and Miller (2002); Lleras-Muney and Moreau (2017); author's calculations.

conditions in childhood and adolescence, and these long-lasting gains overshadow the detrimental effects of declining economic conditions.

To illustrate this point, I conduct another simulation, and report the results in my figure 4. For whites (the left panel), I assume that mean initial health in 1940 is $\mu_0 = 1.754$ and $I = 0.0551$—these parameters match the 1940 profile of mortality, as explained above. For those of low socioeconomic status (the right panel), I set initial health and initial annual resources lower, at $\mu_0 = 1$ and $I = 0.051$, respectively, thus resulting in much worse infant and child mortality. For both groups, the hypothetical 1980 cohort has better initial health, and higher annual investments up to age 20. But at age 20, both groups see their annual health resources fall by the same proportional amount (20 percent).[8]

For both groups, mortality up to age 20 is markedly lower for the 1980 cohort, consistent with what we observe in the United States. But despite

8. These simulations are only illustrative; the parameters are not meant to match any specific mortality profile.

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the fact that both groups are hit at the same age (20 years) by the same adverse shock, mortality increases at earlier ages for whites than for low-SES groups. In the time series, one would observe, for instance, that mortality at age 40 is falling for low-SES groups but is increasing for whites. This occurs because the improvements in early conditions have delayed effects on mortality and show up only later in adulthood. For the low-SES groups, these greater improvements (in levels) partly mitigate the negative shock at age 20. This illustrates that it is extremely difficult to draw conclusions about the effects of a given shock without accounting for differences in conditions before the shock.

These early life improvements could explain why the mortality of blacks is not falling at the same time as that of whites, despite their also being hit by deteriorating conditions in the labor market at age 20. Interestingly, Case and Deaton's figure 2 shows that the mortality of the black population also started to rise in 2010. So it is possible that, for blacks, adverse labor market effects are just beginning to outstrip the benefits of improved childhood conditions.

CONCLUDING REMARKS Health and longevity appear to be in decline in the United States among white non-Hispanics, particularly for those without a college education. Case and Deaton show that current incomes and other contemporary short-term factors cannot adequately explain the patterns in the data; rather, the authors point to "a long-standing process of cumulative disadvantage." The analysis I've presented in this comment, based on a cohort model of health and mortality, comes to very similar conclusions. There has been a permanent deterioration in one or more factors that affect health, starting at about the time of labor market entry. This deterioration is visible for cohorts born after 1950, and likely started occurring at about age 20 (rather than at birth). It is more visible for those without a college education. These affected cohorts entered the labor market in the 1970s. Changes in labor market conditions starting in the 1970s—which have resulted in lower wages, and possibly lower lifetime real incomes for a substantial part of the population—are a likely explanation for the observed deterioration of health in middle age. But any factor affecting health to which cohorts are exposed for a long period starting at about age 20 is a candidate explanation. Temporary changes, conversely, are unlikely to explain the findings. However, a full accounting of the patterns we observe requires a careful consideration of how the entire set of lifetime circumstances has changed for more recent cohorts.

Deaths associated with prescription drug and alcohol abuse have increased substantially. Policies that limit access to these drugs could save

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many lives, as could expansion of alternative nonlethal painkillers such as marijuana. But the data suggest that the underlying mental and physical health of a large fraction of the population is declining. Reducing access to alcohol and drugs will not reduce pain, nor reverse the underlying trend that is causing recent cohorts to be in worse health. Thus, it is necessary to gain a deeper understanding of these trends' underlying causes.

Mortality is declining particularly fast for those without a college education. The returns to college in lifetime wages and incomes, as well as longevity, are rising. If these wage and health returns are causal, then serious consideration should be paid to expanding college attendance. If education is not causing these, it would be extremely important to identify what, then, is causing the increasing gaps related to education. Another possible policy response would be to consider wage subsidies, perhaps through mechanisms like the earned income tax credit, that provide greater support for those with the lowest wages. If the trends identified by Case and Deaton continue, it is possible that future generations will be substantially worse off.

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**GENERAL DISCUSSION** Andrew Levin began by noting the incredible importance of the paper. As a resident of New Hampshire, and Vermont before that, he was acutely aware of the opioid epidemic that the authors were describing. He thought the paper could be connected to the paper in the present volume by John Fernald, Robert Hall, James Stock, and Mark Watson, and also to Laurence Ball's work on hysteresis. He explained that despair and labor market outcomes are clearly linked; despair leads to worse labor market outcomes, which then reinforces the despair. These two things tend to be difficult to disentangle when looking at long periods of time and when averaged across a number of demographic groups. One must try to distinguish structural, demographic, and cultural trends.

Levin urged the participants not to think in terms of Divisia indexes, Hodrick–Prescott filters, or other common filters, but instead to look for the canary in the coal mine. In this case, Levin argued that the canary in the coal mine is the labor force participation rate for white females age 45–54. For most of the post–World War II period, labor force participation for white females age 45–54 was rising, reaching a peak of about 77 percent in the late 1990s, and remaining there until about 2008. After 2008, it started to fall, from 77 percent to 74 percent. Some good news is that over the last couple of years, labor force participation has started to pick up again for many prime-age adults. But for white females age 45–54, it has only risen modestly, from a trough of about 74 percent up to about 74.5 percent. This suggests that the stronger labor market of the last couple of years has perhaps been helping to arrest the declining trend. As this relates to Ball's work on hysteresis, if one takes a very pessimistic view that these are all exogenous inevitable trends—as opposed to believing that monetary policy, fiscal policy, regulatory policy, and all kinds of other public and private actions can make a difference—then this really is a critical problem, he concluded.

Louise Sheiner observed that one thing the authors did not say a lot about was the "regime shift" in how pain is treated. It used to be that pain was undertreated, and most doctors would not prescribe much morphine. But suddenly this practice changed, and painkillers began to be more routinely prescribed. One interpretation of the current opioid epidemic is that deaths of despair by drug overdose may have happened anyway, absent the wide availability of opioids. But a second interpretation is that the opioid epidemic itself caused the despair. If one's child becomes addicted to opioids, one might then become depressed and start to overdrink. She wondered what prescription patterns were like in Europe, and if the practice was very different than that in the United States. She was interested

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in looking at the epidemic across different ages, and thought that surveys of life satisfaction could potentially shed more light. Surveys show that when people have kids, they tend to be less happy. She also suggested that if fewer people are employed, then perhaps one's rank in a company or the concept of being someone's employee starts to matter less.

Richard Cooper wondered about the paper's focus on educational attainment. Over many decades, the ratio of people with a high school education or less has declined sharply in the United States. If one thinks that this decline is due to the fact that more people enrolled in and finished college, and presuming that is a nonrandom decline, it may be that when comparing educational attainment across time, the groups are not actually comparable. He asked the authors to comment on this.

Deaton stated that he and Case were very careful to make sure the educational groups were the same over time. Case explained that the proportion of people with a high school degree or less from 1990 to 2015 has been roughly constant, at 40 percent. Cooper noted that some of the data on birth cohorts go back to the 1940s, and the ratio has declined sharply since then. Case responded by saying that from the birth cohort born in 1945 through the birth cohort born in 1965, the fraction of each cohort with a college degree or more has been constant at about 30 percent.

Valerie Ramey wondered if one could gain insight from other historical periods. Great Britain, for instance, did not experience a Roaring '20s like the United States, and in fact experienced economic malaise in the 1920s, followed by the Great Depression of the 1930s. She wondered how people responded back then to long periods of economic malaise, when opioids were not prevalent.

Gordon Hanson wondered if the authors could say more about the geographic dimension. Citing the work of John Bound and Harry Holzer, and more recently Rebecca Diamond and Danny Yagan, he noted that less-educated individuals tend to be unresponsive in terms of geographic mobility when faced with local labor demand shocks.[1] There is also the work by William Julius Wilson on when work disappears and what that does to localities, and J. D. Vance's continuation of that work with *Hillbilly*

1. John Bound and Harry J. Holzer, "Demand Shifts, Population Adjustments, and Labor Market Outcomes during the 1980s," *Journal of Labor Economics* 18, no. 1 (2000): 20–54; Rebecca Diamond, "The Determinants and Welfare Implications of US Workers' Diverging Location Choices by Skill: 1980–2000," *American Economic Review* 106, no. 3 (2016): 479–524; Danny Yagan, "Is the Great Recession Really Over? Longitudinal Evidence of Enduring Employment Impacts," working paper (November 2016), https://eml.berkeley.edu/~yagan/EnduringImpact.pdf.

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*Elegy.*[2] In addition, Justice Pierce and Peter Schott, as well as David Autor, David Dorn, and Hanson, have shown that trade-induced declines in manufacturing affect increases in drug- and alcohol-related deaths, particularly among young males.[3] All this evidence suggested to Hanson that there may be a feature of the local labor market that could be exploited in Case and Deaton's analysis. Additionally, there may be a way to exploit technological diffusion. If one thinks of opioids as a way of expressing one's despair, then their availability suddenly has very dramatic effects.

Emi Nakamura was struck by the thematic unity of the paper by Case and Deaton and the papers on monetary policy (in the present volume) by Marco Del Negro, Domenico Giannone, Marc Giannoni, and Andrea Tambalotti; and Michael Kiley and John Roberts. At first glance, deaths of despair and monetary policy seem as if they are about totally different things. A basic macroeconomic policy issue is how much weight to put on unemployment versus inflation and other factors. The assumption that has been maintained in labor economics and macroeconomics tends to be that not working means that one is consuming more leisure, which can be considered a good thing. One of the issues in monetary economics is that the costs of business cycles and unemployment tend to be quite low in the models that make those kinds of assumptions. There are many reasons why they may be lower than they should be, but it struck Nakamura that this is yet another one; one's work contributes in important ways to one's sense of identity, which is absent from how macroeconomists have tended to think about the cost of unemployment. The potential link between deaths of despair and labor force participation, which the authors consider near the end of their paper, may actually provide important insights for macroeconomic policy.

Robert Barro wondered about the paper's possible implications for drug policy. There is an ongoing discussion about the greater use of painkillers, and there are obvious implications for the legalization of drugs such as marijuana and cocaine, which have become popular ideas.

Carol Graham suggested that one reason mortality rates for blacks and Hispanics have not followed the increase for whites in recent years is that

2. William Julius Wilson, *When Work Disappears: The World of the New Urban Poor* (New York: Alfred A. Knopf, 1996); J. D. Vance, *Hillbilly Elegy: A Memoir of a Family and Culture in Crisis* (New York: HarperCollins, 2016).

3. Justin R. Pierce and Peter K. Schott, "Trade Liberalization and Mortality: Evidence from U.S. Counties," Working Paper no. 22849 (Cambridge Mass.: National Bureau of Economic Research, 2016); David Autor, David Dorn, and Gordon Hanson, "When Work Disappears: Manufacturing Decline and the Falling Marriage-Market Value of Men," Working Paper no. 23173 (Cambridge, Mass.: National Bureau of Economic Research, 2017).

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blacks and Hispanics tend to be more resilient to negative shocks, as shown in the psychology literature. With respect to questions about life satisfaction, Graham noted her recent work finds that poor blacks and poor Hispanics tend to have higher levels of life satisfaction than poor whites.[4] Very large gaps emerge when people are asked about five years into the future; poor blacks tend to be very optimistic but poor whites very negative.

Jason Furman noted that one advantage of the paper by Hanson, Chen Liu, and Craig McIntosh is that their data are projected well into the future, through 2050. He wondered what Case and Deaton thought their own data might look like many years from now—and, in particular, what could be said about changes in inequality of life expectancy by education or any other category for the young. Smoking trends, in particular, seem to be important. For older individuals, smoking has risen for the less educated and fallen for the more educated; but for younger individuals, smoking has fallen sharply. He wondered if these trends might make a difference in the future.

Case and Deaton do not distinguish between those with strictly less than a high school education and those with a high school education or less. Martin Feldstein noted that labor market outcomes are dramatically different for those with a high school education and those who do not finish high school. He wondered if the authors could expand on why they believed this distinction did not matter for their analysis. He also wondered about the importance of religion, and whether the authors might think about religion as something that provides a sense of community.

Justin Wolfers believed the authors mount a compelling case that there is despair among the white working class. However, he noted that many of the behaviors the authors observe are relatively uncommon. Therefore, he was not sure whether the main takeaway from the paper is that the distribution of well-being among the white working class has gotten worse, but rather that the *bottom half* of the distribution has gotten worse. He suggested that one way of thinking about despair generally is to examine the forward-looking decisions people make. One might say, "I'm going to put on my seatbelt and not eat McDonald's because I think tomorrow is going to be a good day, and it is worth sticking around for it." Regularly eating McDonald's or not wearing a seatbelt might be considered "probabilistic suicide." He wondered if the authors could say more about the broader distribution.

4. Carol Graham and Sergio Pinto, "Unhappiness in America: Desperation in White Towns, Resilience and Diversity in the Cities," in *Brookings Big Ideas for America,* edited by Michael E. O'Hanlon (Washington: Brookings Institution Press, 2017).

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Christopher Carroll proposed tying together points made by Levin and discussant Adriana Lleras-Muney, who argued that what matters as "pollution" is *prolonged* exposure to a bad labor market, one's cumulative history of exposure, and not so much one's contemporaneous circumstances. He thought it would be possible to use the available data on differing regional performance of labor markets over people's working lifetimes to construct a measure of cumulative exposure to bad economic conditions, and to see how much of the current regional variation in "deaths of despair" is explained by people's lifetime experiences as opposed to current experiences.

Steven Davis picked up on the point made by Graham about resilience, particularly the differences between whites and nonwhites within the low-educated segment—whose members, regardless of demographics, are presumably experiencing similar adverse labor market developments. He had no doubt that adverse labor market developments are important, which suggests that the groups have very different degrees of resilience in response to similar shocks. Therefore, it seems important to try to understand the sources of these differences, which seem to have changed over time. He was often struck by discussions of the psychological traumas inflicted on American troops who have been in combat abroad. Many Americans were in combat in Korea or World War II, and the related incidents of psychological trauma related to these wars seem to have increased. Though based only on casual evidence, it suggests that something about our society may have decreased resilience to bad shocks, at least in certain demographic groups.

Jonathan Pingle noted that the system of equations that identifies Case and Deaton's cohort fixed effects is very similar to the labor force participation rate model of Stephanie Aaronson and others.[5] One could interpret the cohort fixed effects as unobserved life-cycle labor force attachment, and graphing them for men reveals a clear downward decline. He suggested that the authors could jointly estimate outcomes with a model of this type; veteran status or other indicators might provide adequate natural experiments for variation in things like substance abuse and mental illness. In this context, one could start thinking about how to separately identify important social phenomena.

Deaton thanked the participants for their comments, and assured them that he and Case would do their best to incorporate them into the next version of the paper. Deaton first picked up on the issue of drugs. He

5. Stephanie Aaronson, Bruce Fallick, Andrew Figura, Jonathan Pingle, and William Wascher, "The Recent Decline in the Labor Force Participation Rate and Its Implications for Potential Labor Supply," *Brookings Papers on Economic Activity,* no. 1 (2006): 69–134.

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emphasized that the drugs he and Case were talking about in their paper are largely legal prescription drugs. He and Case were of the opinion that opioids are not the fundamental problem; he believed the world would be a much better place if doctors had never started prescribing them for moderate chronic pain—which is in the U.S. Food and Drug Administration's guidelines—because they have addicted and killed many people who would otherwise be alive. Opioids constitute a pure iatrogenic medicine that is killing people who should have never been prescribed them in the first place. Rather, he and Case think of opioids as throwing fuel on a fire that was already there. Suicides, cirrhosis, and other maladies have been around for a long time, but the prevalence of opioids has made them much more visible.

On Barro's question about drug legalization policy, Deaton noted that he and Case think that marijuana legalization is actually a good thing in this context. Pharmaceutical companies have fought hard against marijuana legalization, since it would eat into their bottom line. Though marijuana may not be very good for a person, it is much better than opioids, because marijuana will not kill you.

With respect to Wolfers's and Furman's comments on overeating and smoking, Deaton acknowledged that there are various ways of "feeding the beast." Smoking and overeating may be a part of that, and obesity is one of the variables he and Case were digging into more deeply. One thing that has been puzzling is that obesity has not yet shown up in higher death rates, though it may actually be hidden in the form of heart disease. There is also an argument that many deaths from diabetes are actually falsely diagnosed as being from heart disease. Furman's point about the upswing in smoking among the less educated may also be part of this story. Thus, Deaton stated, he and Case would certainly focus on more of this aspect.

With respect to Feldstein's question about the distinction between *less than high school* and *high school or less*, the authors purposely tried to stay away from the distinction. A famous paper by S. Jay Olshansky and colleagues attempts to draw the distinction; but coming back to Cooper's point, there is so much selection on the group over time that one really has no idea what one is looking at.[6] Those people are getting more and more negatively selected over time, and one does not know whether it is their circumstances or something more. For the 45–54 age group in particular, he

6. S. Jay Olshansky and others, "Differences in Life Expectancy Due to Race and Educational Differences Are Widening, and Many May Not Catch Up," *Health Affairs* 31, no. 8 (2012): 1803–13.

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and Case were very careful to define the education groups so that over the period of analysis, there has been very little change in the composition—which is not true for some earlier birth cohorts. Nevertheless, Deaton conceded that there are compositional effects that need to be taken into account, and that he and Case would think about them in the future.

On Nakamura's and Levin's points, Deaton noted that the first paper written in 1922 on the procyclicality of mortality by William Ogburn showed that mortality is actually higher in good times than in bad times, a result that has been regularly replicated in the literature.[7] (The reverse, however, is much less common; that is, mortality is not necessarily lower in bad times. One of the more stunning cases is Spain, where the unemployment rate after the Great Recession rose from about 5 percent to 28 percent, and every class of mortality fell like a stone.)

With respect to mortality and income, Deaton was impressed by a figure Lleras-Muney included in her presentation that showed the income growth of the top 1 percent versus the bottom 50 percent.[8] Despite the apparent flatness of growth for the bottom 50 percent, Deaton believed there was progress being made for the bottom 50 percent—less so on wages, but on incomes. "You can see on the graph if you know how to look for it," he stated. Data from the U.S. Census Bureau show quite a bit of progress.

Deaton returned to what he thought were some of the key issues—religion, marriage, children, and cumulative disadvantage. In formulating the paper, he and Case had tried to stay away from anything to do with exogeneity, instruments, natural experiments, and the like, instead opting for a more historical approach. In the top-left panel of figure 7, the age-mortality profiles are steepest for the younger cohorts (the coefficients can be different, which is why there are multiple lines rather than one). Generally speaking, the lines appear parallel, implying that deaths of despair have been happening over a long period of time and have gotten worse in parallel.

For Case and Deaton, a big factor pertains to the labor market for people who graduate from high school; in the 1970s, blue-collar aristocrats could get a job and see high returns to building skills. Most jobs were for a life-

7. William F. Ogburn and Dorothy S. Thomas, "The Influence of the Business Cycle on Certain Social Conditions," *Journal of the American Statistical Association* 18, no. 139 (1922): 324–40.

8. See the lower panel of figure 5 in Thomas Piketty, Emmanuel Saez, and Gabriel Zucman, "Distributional National Accounts: Methods and Estimates for the United States," Working Paper no. 22945 (Cambridge, Mass.: National Bureau of Economic Research, 2016).

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time; one would work in the same factory where his father and grandfather had worked. One could get married and have children, and could reasonably expect his wages to rise over time. But this kind of situation is getting scarcer and scarcer; those types of jobs hardly exist anymore, and they have been vanishing over time. These things make marriage more difficult (lifetime marriage rates are falling among this group), though cohabitating is now commonplace. Cohabitating relationships are clearly a social change, as 50 years ago such an arrangement would be socially ostracized. Today, however, the majority of white women with only a high school degree have had at least one child out of wedlock, so this has become normal behavior. The trend of increased cohabitation is also happening in Europe, Deaton explained, though one big difference is that cohabiting relationships tend to be more stable in Europe than they are in the United States.

On Feldstein's point about religion, Deaton stated that there has not actually been much decline in church attendance of the usual measure. What has changed is the *types* of religion people are practicing. Legacy religions have been replaced by "seeking" religions, which put a lot of responsibility on the individual to find his or her own way in the world. Just as one had a job in the same factory as one's father and grandfather, one would belong to the same church as one's father and grandfather. Church was a home, a place of security. But now this security is gone. Deaton joked that the Catholic Church was replaced with a 12-step group. But a 12-step group cannot give people the same degree of security.

Deaton stated that for him and Case, suicide is a very difficult thing to study. It is not well understood, and has never been well understood. One thing for sure, however, is that suicide is cumulative in nature: Families fall apart, children's lives fall apart, one's religion does not provide the same protection, and one's job no longer gives satisfaction—factors that are all likely to be associated with suicide. Throw opioids in and social disaster occurs. Though this behavior may be happening at the tail of the distribution, there are still many despairing people out there.

Deaton finished with a major policy question—a point David Cutler raised in his discussion: Is the increase in midlife mortality a cohort effect, or is it a time or age effect? It is true that the older cohorts now have access to Medicare and Social Security, and have generally done much better in terms of incomes than the younger cohorts. Deaton explained that he has spent much of his life trying to show that though income can be helpful for health, it is not the main factor. While the older cohorts are being well taken care of, they have experienced the current poor labor market conditions for a much smaller fraction of their lives than the younger cohorts.

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Those born in 1945, for example, could have been blue-collar aristocrats early in life; the majority of one's life could be pretty good, and only near the end would one have to deal with the poor labor market issues of late.

Case and Deaton admitted they did not know the answer. They were not suggesting Cutler was necessarily wrong in pinning labor market issues as the main culprit. But under this view, one would logically conclude that people are going to be OK as soon as they segue into Social Security and Medicare, and that there will not be a horrible mortality crisis coming in the next few years. If, on the other hand, it is a process of cumulative disadvantage over a very long period of time, then a real catastrophe is potentially unfolding.

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