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.
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,
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.

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

![Figure 1](chart.png)

Source: National Center for Health Statistics.

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,
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,
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.

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

![Graph showing relative mortality by age cohort from 1999 to 2015.](image-url)
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
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
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.

REFERENCES FOR THE CUTLER COMMENT


**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.
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
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, $H$.

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

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

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

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

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

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, 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, $\sigma_e^2$.

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 $H = 0$ and $\sigma_e^2 = 1$. 

This content downloaded from 128.97.202.145 on Fri, 19 Oct 2018 17:38:41 UTC
All use subject to https://about.jstor.org/terms
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

---

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

![Figure 1](image)

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

a. The estimated parameters are $I = 0.0554$, $\delta = 0.0012$, $\sigma_\epsilon = 0.1515$, $\alpha = 1.3049$, $\mu_0 = 1.7424$, 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.
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 ($0.0554 \div 1.7424$) 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 $I = 0.0546$, $\delta = 0.0012$, $\sigma = 0.1534$, $\alpha = 1.3022$, $\mu = 1.6078$, 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.
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.
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.
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. 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).
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. 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).
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 (hyper-polluted) sites (Currie 2013). A careful analysis of the pollution hypothesis
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.
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 Schandt 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
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).  

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.
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
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.

REFERENCES FOR THE LLERAS-MUNEY COMMENT


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
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*

---

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. 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


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. 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.

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. 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. He

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. 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

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-

---

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. Cohabitation 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.
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.