Declining health and longevity in America: A comment on Case and Deaton's "Morbidity and Mortality in the 21st century"

Adriana Lleras-Muney

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Life expectancy in the US and most developed countries has been increasing for the last 150 years rather steadily. But life expectancy at birth in the US declined in 2015 for the first time since 1994. Although small declines have been observed before, Case and Deaton (2015, 2017) 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 occurring to the mortality rates of other populations, such as Europeans of the same age. Death rates from suicide, drug 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 college continues to fall.

Case and Deaton (2017) 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: mortality at a given age is higher and it rises faster with age for more recent cohorts. 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 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 the model by Lleras-Muney and Moreau (2017). This is a simple model of evolution of health and death from birth onwards. In its simplest form the behavior of mortality is 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 reproduce lifetime mortality rates and life expectancy well. I then investigate whether changes in the baseline parameters can generate patterns in mortality and morbidity similar to those documented by Case and Deaton.

Just like Case and Deaton I conclude that there are (at least) two forces

that 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 we age) could be increasing. Both of these factors would generate steepening mortality and disability age profiles. Importantly, these patterns cannot easily be explained by "temporary" conditions; in the model they can only be the result of permanent changes in the parameters beginning early in adulthood (or even earlier in life). This is again the same conclusion that Case and Deaton reach from their non-parametric analysis.

To assess the likelihood of each of these hypotheses, I use evidence from the literature to speculate on 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. While 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 by education and reflecting on possible policy implications.

1 A simple model of health and mortality estimated for the US

This section draws heavily on 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 every period health deteriorates, due to wear and tear. Importantly 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 constant over the lifetime. But individuals within the population are subject to i.i.d. shocks ε_t every period: some get higher, 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

$$\begin{aligned} H_0 &\sim N(\mu_0, \sigma_0^2) \\ H_t &= H_{t-1} - \delta t^\alpha + I + \varepsilon_t \\ \varepsilon_t &\sim N(0, \sigma_\varepsilon^2) \\ MR_t &= P(H_t < \underline{H} | H_{t-s} > \underline{H}, \forall s < t-1) \end{aligned} \tag{1}$$

with $\delta \in (0, \infty), \alpha \in (0, \infty)$, 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), then the distribution of health moves away from the threshold and causes mortality to plummet to very low levels by adolescence. Since the 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 (μ_0), two that govern the aging process (δ, α), and two that characterize the health resources provided by environment, in the form of average investments (I) and the variance of these investments or shocks (σ_ε^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). In Lleras-Muney and Moreau we investigate many of these extensions. But here I use this model because it provides a remarkly good baseline: using only five parameters it can match the basic age profile of mortality we observe in the Human Mortality Database. Therefore this simple model is a good starting point for understanding the possible factors behind the deterioration in (white) Americans' health and longevity.

2 Estimating the model for the US

I validate this model by estimating the parameters for the 1940 birth cohort, using cohort tables provided by the Social Security Administration.[2] Because cohorts born after 1940 experienced robust GDP growth, I estimate a slightly extended version of the model above, with has a sixth parameter, r. I is assumed to be increasing every period at a constant rate r, also 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.

Figure 1 shows the results of this exercise for US females. The left panel shows the (natural) 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 features of the data (for instance the exact level of mortality during reproductive ages[3]), the model matches the basic shape of mortality very well. Moreover it predicts life expectancy (up to age 65[4]) of 60.5080 for this cohort, compared to the actual life expectancy of 60.5084.[5]

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

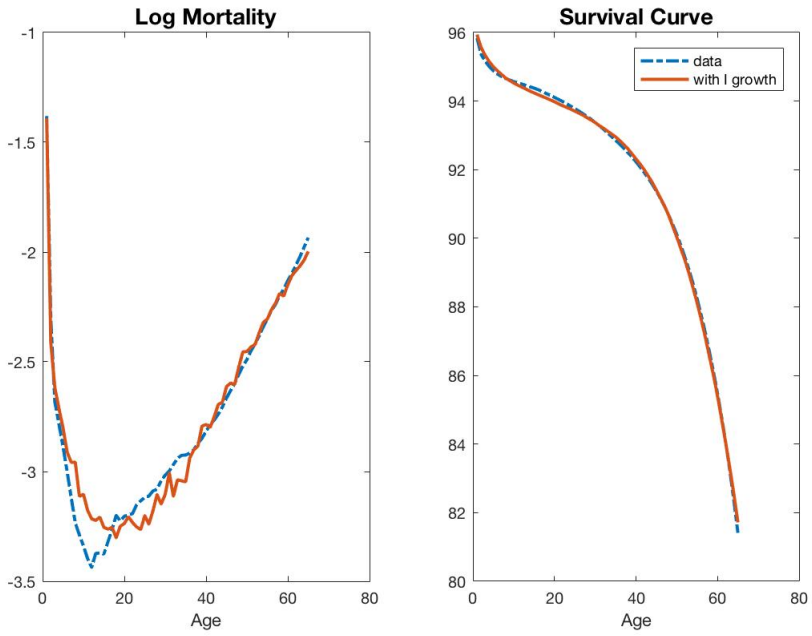
[2] Data taken from https://www.ssa.gov/oact/NOTES/as116/as116_Tbl_7_1940.html#wp1081274

[3] This feature can also be improved upon. See Lleras-Muney and Moreau (2017).

[4] The cohort tables uses projections after 2005.

[5] The estimated parameters for men are $I = 0.0546$, $\delta = 0.0012$, $\sigma_\varepsilon = 0.1534$, $\alpha = 1.3022$, $\mu_0 = 1.6078$, $r = 1.0207$. The fit is also good for men but not quite as good. 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. In Lleras-Muney and Moreau

Figure 1: Mortality and survival profiles for US females born in 1940.



The estimated parameters are $I = 0.0554$, $\delta = 0.0012$, $\sigma_\varepsilon = 0.1515$, $\alpha = 1.3049$, $\mu_0 = 1.7424$, $r = 1.0224$.

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

3 Predicting and explaining the trends in white non-hispanic mortality profiles in the US

I now use this model to investigate whether changes in any of the parameters can generate the patterns documented by Case and Deaton. I simulate the effect of changes in the key parameters of interest starting at age 20 for both mortality and disease rates. I also simulate disease rates, by assuming that individuals are sick if they are alive but their health falls below some arbitrary (health) threshold.

(2017) we estimate models that succesfully account for the hump in mortality.

3.1 Simulation results

Figures 2 and 3 show the results for mortality and morbidity. Three types of changes can rationalize Case and Deaton's findings. A decrease in the baseline level of annual health investment, a decrease in their annual rate of growth, or larger depreciation, result in steeper age profiles for both mortality and disease rates (Figure 2). Note that in all cases, the effects of changing the parameters on mortality are almost imperceptible between ages 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 (from 0 to a positive number), the variance of resources, or the death thresholds result in patterns for mortality and morbidity that differ from what we observe (Figure 3). If we allow for an exogenous increase in random accidents, 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. In Lleras-Muney and Moreau (2017), we 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 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.[6] 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.[7] Heights increased throughout, although again at a decreasing pace for those born after 1950.[8] These three measures (infant mortality, heights 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 iden-

[6] https://www.cdc.gov/mmwr/preview/mmwrhtml/mm4838a2.htm#tab1

[7] See Figure 5 of https://www.cbo.gov/publication/22010. Also see Goldin and Katz [2007a]

[8] For white men height grew by more than 4cms for birth cohorts born between 1910 and 1950, but only grew by 1 cm from 1950 to 1980 (http://www.tandfonline.com/doi/full/10.1080/03014460601116803). For women the increases are 2.1 and 1.3. Data from other sources suggest similar patterns (Bleakley et al., 2014).

Figure 2: Three factors generate steeper age profiles in mortality and disease

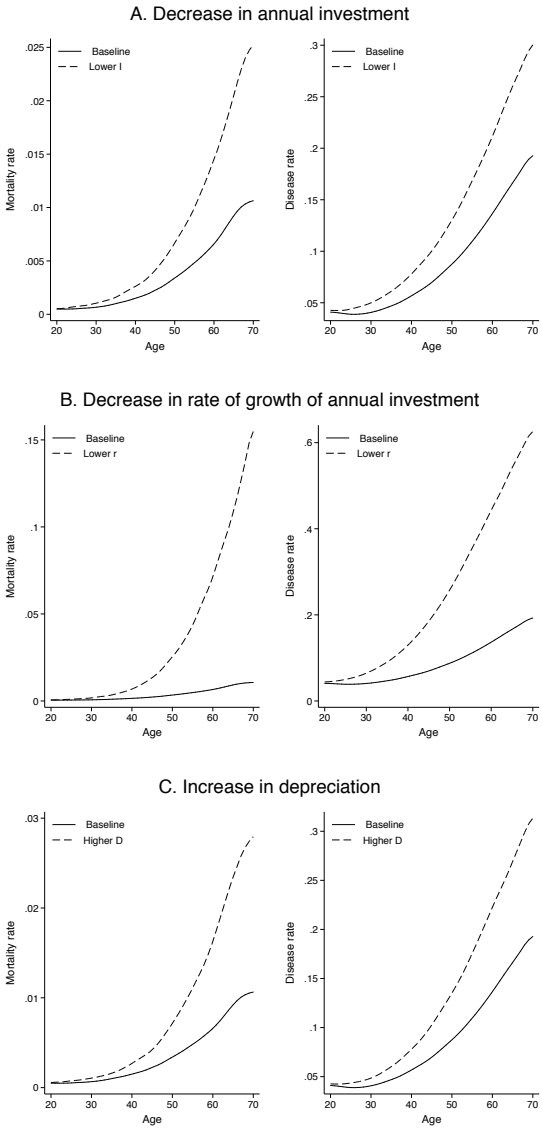
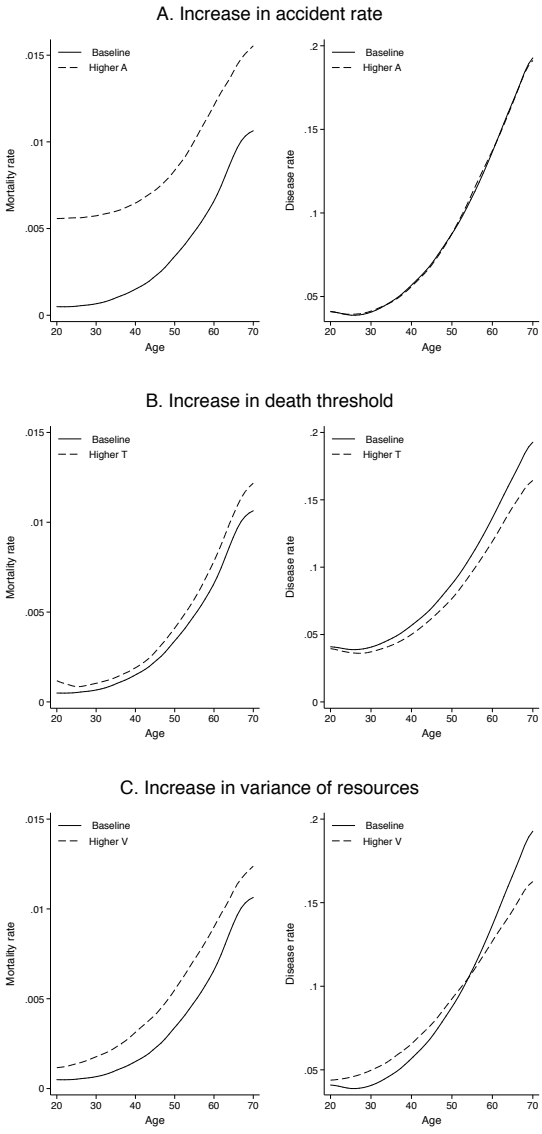


Figure 3: Other factors cannot explain changes in both mortality and morbidity age profiles



tical up to age 20, but this is not the case. Currie and Schwandt [2016] 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 suggest that initial conditions are not constant across birth cohorts. In our model this would result in the entire profile of mortality shifting downwards, 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.

3.2 What factors could be causing these changes?

Given these results I now discuss whether the existing literature provides any support for these possibilities.

Decline in annual health investments. 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: $I = f(x) = f(aY/p_x)$. What this suggests is that resources could be going down if either (a) lifetime incomes are falling (holding prices constant), or (b) the price of health inputs is rising (holding incomes constant). Data on lifetime income by cohort is difficult to find. A very recent paper by Guvenen et al. [2017] uses data from the Social Security Administration tracking 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 they fell. More significantly, the price of health-related goods and services has increased over time very substantially, at a much faster pace than the the cost of other goods and services, starting in the late 1970s.[9] 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 changes in the parameters have very small effects on contemporary mortality. Sustained changes have effects that are not visible immediately, but but become apparent after a substantial delay, as shown in Figure 2. Assessing whether lifetime resources fell for cohorts entering the labor market after 1970,

[9] According to the Bureau of Labor Statitics, the cost of medical goods rose many times faster than the cost of other goods. See https://www.bls.gov/opub/mlr/2014/article/one-hundred-years-of-price-change-the-consumer-price-index-and-the-american-inflation-experience.htm

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 (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 intial 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, 2004). 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. Chetty et al. [2016] 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 on this paper. 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 suggest individuals are unhappy.

Pollution (air, water and food toxins) can also result in accelerated aging. This hypothesis is supported by animal models (eg Sun et al., 2005) but is difficult to demonstrate in humans. The use of fossil fuels has increased steadily since 1900, and while 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 (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 lower SES backgrounds. For example poor individuals with low education are more likely to live close to highways and Superfund 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 cardiovascular system in the short term in humans, its long term effects seem worthy of futher investigation, particularly in light of the fact that cardiovascular mortality rates are no longer falling among adults.

Case and Deaton point to the 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 affect aging, for instance physical activity.

3.3 In Light of Education

The increase in age-adjusted mortality for white non-Hispanics as a whole is modest compared to 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 college and those without has been rising since the 1960s (e.g. Meara et al., 2008, Montez et al., 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 WWII, and for women born after 1970.[10] Nevertheless the composition of the pool could be changing despite roughly constant shares. There could be increasing selectivity in college entrance on the basis of test scores for instance.[11] 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 (eg Cutler et al., 2011). Rather the "returns to college" in terms of health appear to be on the rise.

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). Autor 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 start at a lower level and again never catch up to men. [12] The literature looking at the "college premium" has concluded 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 by education, because this requires accounting for marriage and fertility patterns by education, as well as transfers and changing prices. But the evidence does suggest that 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

[10] For working men born in 1950 years of completed education (measured at age 25-29) were 13.4 compared to 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 1980 cohort (www.cbo.gov/publication/22010).

[11] 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 gradutes 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.

[12] See Figure 3 of www.cbo.gov/publication/22010. Also see Autor.

the findings.

3.4 The importance of accounting for initial conditions

Case and Deaton contrast the experience 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 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 and trends in childhoood mortality. This makes the comparisons across groups difficult to intepret, because improvements in health conditions have delayed effects.

Black infant mortality in 1940 was much higher than 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, 2016). All else equal, these improvements early in life lower mortality throughout the lifetime, *and particularly after age 40.* In our model a population with higher initial health will have lower mortality throughout the lifetime.[13] There is also ample empirical evidence showing that early conditions have long lasting consequences on 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 et al., 2017]). Perhaps middle-aged Blacks are still reaping the health benefits of improving conditions in childhood and adolescence, and these lasting gains overshadow the detrimental effects of declining economic conditions.

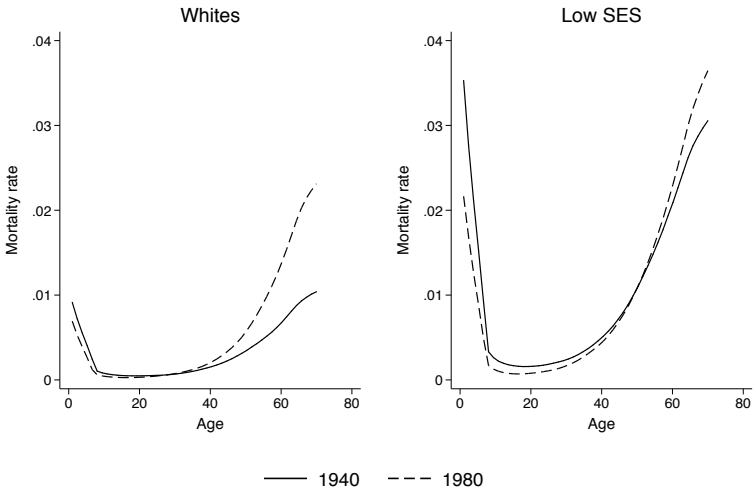
To illustrate this point I conduct another simulation. For whites I assume mean initial health in 1940 is 1.754 and I is 0.0551–these parameters match the 1940 profile of mortality, as expained in section 2. The group labeled "low SES" has lower initial health ($\mu_0 = 1$) and lower initial annual resources ($I = 0.051$) in 1940, and thus has much greater infant and child mortality. For both groups the hypothetical cohort labeled "1980", has higher initial health, and higher annual investments up to age 20, which generate lower mortality for the more recent cohort up to age 20. But at age 20 both groups see their annual health resources fall by the same proportional amount (20%).[14]

Figure 4 shows the results. For both groups mortality up to age 20 is markedly lower for the 1980 cohort, consistent with what we observe in the US. But despite the fact that both groups are hit at the same age (age 20) 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 increasing for whites. This occurs because the improvements in early conditions have delayed effects on mortality later in adulthood. For the low-SES groups these greater improvements (in levels) partly mitigate the negative shock at age 20. This suggests that it

[13] See Lleras-Muney and Moreau (2017) for proofs of these statements.

[14] These simulations are just illustrative–the parameters are not meant to match any specific mortality profile.

Figure 4: Improvements in initial conditions and decreases in I at age 20



is extremely difficult to draw conclusions about the effects of shocks without accounting for differences in conditions before the shock.

These early life improvements could explain why mortality of blacks is not falling at the same time as that of whites, despite their being hit by equally deteriorating conditions in the labor market at age 20. Interestingly Fig 1.2 in Case and Deaton's paper 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 begining to outstrip the benefits of improved childhood conditions.

4 Concluding remarks

Health and longevity appear to be in decline in the US among white non-Hispanics, particularly for those without a college education.

Case and Deaton (2017) show that current incomes and other contemporary short-term factors cannot adequately explain the patterns in the data. Instead "the data are consistent with long-run processes influencing outcomes, rather than contemporaneous shocks affecting health." The analysis in this article, 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 around the time of labor market entry. This deterioration is visible for cohorts born after 1950, and likely started occuring around age 20 (rather than at birth). It is more visible for those without college. These affected cohorts entered the labor market in the 1970s. Changes in labor market conditions starting in the 1970s resulting in lower wages, and possibly

lower lifetime real incomes for a substantial part of the population, are a likely explanation for the observed deterioration in health in middle age. But any factor affecting health to which cohorts are exposed for a long period of time starting around age 20 is a candidate explanation. Temporary changes on the other hand 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 very substantially. Policies that limit access to these drugs could save many lives, as could expansion of alternative non-lethal pain killers such as marijuana. But the data suggests 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 a deeper understanding of the underlying causes of these trends is necessary.

Mortality is declining particularly fast for those without college education. The returns to college in terms of lifetime wages and incomes, as well as in terms of longevity, is 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 by education. Another possible policy response would be to consider wage subsidies, perhaps throuh programs like the EITC, which 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

Douglas Almond and Janet Currie. Killing me softly: The fetal origins hypothesis. *The Journal of Economic Perspectives*, 25(3):153–172, 2011. URL http://www.ingentaconnect.com/content/aea/jep/2011/00000025/00000003/art00008.

Douglas Almond, Janet Currie, and Valentina Duque. Childhood Circumstances and Adult Outcomes: Act II. Technical report, National Bureau of Economic Research, 2017. URL http://www.nber.org/papers/w23017.

D. H. Autor. Skills, Education, and the Rise of Earnings Inequality Among the 'Other 99 Percent.'. *Science*, 344:843–851. URL https://pdfs.semanticscholar.org/8749/5efeef347141de7abc77456a5037f0834740.pdf.

Hoyt Bleakley, Dora Costa, and Adriana Lleras-Muney. Health, Education, and Income in the United States, 1820–2000. In *Human Capital in History: The American Record*, pages 121–159. University of Chicago Press, 2014. URL http://www.nber.org/chapters/c12900.

Raj Chetty, Michael Stepner, Sarah Abraham, Shelby Lin, Benjamin Scuderi, Nicholas Turner, Augustin Bergeron, and David Cutler. The association between income and life expectancy in the United States, 2001-2014. *Jama*, 315(16):1750–1766, 2016. URL http://jamanetwork.com/journals/jama/fullarticle/2513561?utm$_t erm = alsomay$.

Janet Currie. Pollution and Infant Health. *Child development perspectives*, 7(4):237–242, 2013. URL http://onlinelibrary.wiley.com/doi/10.1111/cdep.12047/full.

Janet Currie and Hannes Schwandt. Inequality in mortality decreased among the young while increasing for older adults, 1990–2010. *Science*, 352(6286):708–712, 2016. URL http://science.sciencemag.org/content/352/6286/708.short.

David M. Cutler, Fabian Lange, Ellen Meara, Seth Richards-Shubik, and Christopher J. Ruhm. Rising educational gradients in mortality: the role of behavioral risk factors. *Journal of health economics*, 30(6):1174–1187, 2011. URL http://www.sciencedirect.com/science/article/pii/S0167629611001147.

Claudia Goldin and Lawrence F. Katz. Long-Run Changes in the Wage Structure: Narrowing, Widening, Polarizing. *Brookings Papers on Economic Activity*, 2007(2):135–165, 2007a. URL http://www.jstor.org/stable/27561601.

Claudia Goldin and Lawrence F. Katz. The race between education and technology: the evolution of US educational wage differentials, 1890 to 2005. Technical report, National Bureau of Economic Research, 2007b. URL http://www.nber.org/papers/w12984.

Fatih Guvenen, Greg Kaplan, Jae Song, and Justin Weidner. Lifetime Incomes in the United States over Six Decades. Technical report, National Bureau of Economic Research, 2017. URL http://www.nber.org/papers/w23371.

Charles I. Jones. The facts of economic growth. *Handbook of Macroeconomics*, 2:3–69, 2016. URL http://www.sciencedirect.com/science/article/pii/S1574004816000033.

Ellen R. Meara, Seth Richards, and David M. Cutler. The gap gets bigger: changes in mortality and life expectancy, by education, 1981–2000. *Health Affairs*, 27(2):350–360, 2008. URL http://content.healthaffairs.org/content/27/2/350.short.

Jennifer Karas Montez, Robert A. Hummer, Mark D. Hayward, Hyeyoung Woo, and Richard G. Rogers. Trends in the educational gradient of US adult mortality from 1986 through 2006 by race, gender, and age group. *Research on aging*, 33(2):145–171, 2011. URL http://journals.sagepub.com/doi/abs/10.1177/0164027510392388.

Robert M. Sapolsky. *Why zebras don't get ulcers: The acclaimed guide to stress, stress-related diseases, and coping-now revised and updated*. Macmillan, 2004. URL https://books.google.com/books?hl=enlr=id=EI88oS$_3$$fZECoi$ = $fndpg$ = $PR7dq$ = $Sapolskyots$ = $P8asGNJKJysig$ = $D252nT2yY_2VILNFuNp9m5Xau7M$.

Qinghua Sun, Aixia Wang, Ximei Jin, Alex Natanzon, Damon Duquaine, Robert D. Brook, Juan-Gilberto S. Aguinaldo, Zahi A. Fayad, Valentin Fuster, Morton Lippmann, and others. Long-term air pollution exposure and acceleration of atherosclerosis and vascular inflammation in an animal model. *Jama*, 294(23):3003–3010, 2005. URL http://jamanetwork.com/journals/jama/fullarticle/202049.