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LIVING AND DYING IN AMERICA:
AN ESSAY ON DEATHS OF DESPAIR AND THE FUTURE OF CAPITALISM

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Living and Dying in America: An Essay on Deaths of Despair and the Future of Capitalism

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ABSTRACT

This essay reviews *Deaths of Despair and the Future of Capitalism* (DEATHS), by Anne Case and Angus Deaton, a fascinating account of life and death in the United States during the late 20th and early 21st centuries. While primarily targeted towards a popular audience, the volume will be of interest to many economists and other social scientists. It postulates how public and private policies currently practiced in the United States, combined with and partly causing the declining economic and social circumstances of less educated, have led to increased mortality from drugs, suicide, and chronic liver disease. After describing the material in DEATHS in considerable detail, I suggest a variety of research questions that need to be answered to confirm or refute Case and Deaton's arguments and describe challenges to their key hypotheses. Among the latter are the ability of the postulated relationships to explain the sharply differing mortality trajectories of non-Hispanic whites, compared with other groups, and the timing of the observed mortality changes. Along the way, I raise doubts about the usefulness of the "deaths of despair" conceptualization, with its strong implications about causality.

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1. Introduction

In 2015, Anne Case and Angus Deaton published the landmark article "Rising morbidity and mortality in midlife among white non-Hispanic Americans in the 21st century" (Case and Deaton 2015). The analysis demonstrated that the mortality rates of 45-54 year old U.S. non-Hispanic whites (hereafter simply "whites", unless otherwise noted) rose from 1999-2013, an almost unprecedented increase that was not replicated for other races or in other countries. The increased mortality was concentrated among the non-college educated, with poisonings (almost all due to drugs), suicide, and chronic liver disease playing important roles.

Their 2017 follow-up paper (Case and Deaton 2017), addressed some issues with the original study (e.g. the lack of age-adjustment for key groups) and substantially expanded the analysis to consider broader age categories, sub-national estimates and cohort differences, while again focusing on the particularly adverse trends of those with a high school degree or less. That article also began to develop a set of potential explanations for the results. In it, Case and Deaton largely reject the primary importance of explanations based on changes in incomes or other economic factors and instead emphasize the role of slow-acting social forces leading to cumulative disadvantage that resulted, as one manifestation, in increased death rates for midlife less educated whites.

Deaths of Despair and the Future of Capitalism (*DEATHS*), (Case and Deaton 2020) draws heavily upon the two aforementioned papers and describes three stories of American life in the late 20th and early 21st century. The first discusses how in the United States, the benefits of economic growth have primarily been delivered to the wealthy and advantaged. The second is of the increasingly difficult economic and social circumstances facing Americans without college degrees. These take many forms including the precarious economic situation just mentioned, social isolation, and the disintegration of structures previously supporting mental and physical health. The third story is one of declining life expectancy for less educated midlife whites, with particular attention paid to rising mortality rates from drugs, suicide and chronic liver disease. The evidence provided for each of these stories is convincing to varying degrees but sometimes missing are the essential elements that tie them together and future research is needed to better understand to what extent they explain the striking mortality trends that motivated Case and Deaton's initial research on the topic.

DEATHS is written for a general audience and contains an enormous amount of useful and often fascinating information. For instance, Case and Deaton provide fairly detailed discussions of the measurement of levels and trends in mortality rates, self-reported health and a variety of morbidities. Economists familiar with this material may be able to skim it,

or efficiently gain much of the same information by reading the two research papers mentioned above. However, these concepts and much other material in the volume will be new to many. For example, despite having studied the fatal drug epidemic for several years, I found the account of the opium wars between Britain and China during the mid-19th century and the subsequent post-civil war opium epidemic in the U.S. to be absorbing and informative. The multiple failures of the American healthcare system are well summarized and the discussion of them will be particularly useful for those with limited background in this area. The chapter on the opioid epidemic is a must-read for those interested in understanding its history and sources, and who do not wish to undertake book-length explorations (e.g. Quinones, 2016; Lembke 2016; Macy 2018) of these issues. The description of America's current "Sheriff of Nottingham" redistribution of resources from the poor to the rich is noteworthy, although possibly less compelling to some readers.

Below, I provide a more detailed outline of the material presented in *DEATHS*. I then describe challenges to the hypotheses presented by Case and Deaton, as well as specific research questions that might help to confirm or refute them, and provide some concluding thoughts.

2. DEATHS: By the Chapters

2a. Things Come Apart

After an introduction, the first seven chapters of *DEATHS* convincingly argue that the something important has changed in the life experiences of middle-aged whites. Chapter 1 begins by defining life expectancy and mortality, briefly discusses mortality patterns over the lifecycle (being highest in infancy and for the elderly and lower in-between) and then explains how the leading causes of mortality changed from being dominated by infectious diseases at the beginning of the 20th century, to now being primarily due to conditions or illness such as cancer, heart disease and Alzheimer's disease. The importance of lifestyles and health behaviors are emphasized. The key take-away is illustrated in a figure showing that the mortality rates of 45-54 year old whites fell by between 0.4 and 1.5 percent yearly between 1900 and 2000, with almost continual reductions except for a short-lasting spike during the 1918 influenza epidemic and periods of stagnation in the 1920s and 1960s. The authors point out that other groups also benefited from declining mortality during the 20th century and that alternative measures of health also improved.

The second chapter, after which this section is named, demonstrates the dramatic change in this pattern for 45-54 year old whites in the 21st century. Specifically, Case and Deaton show that age-adjusted mortality rates for this group stopped declining around 1999 and modestly rose between that year and 2017. Rather than focusing on the relatively small

increase in all-cause mortality, however, they emphasize the additional deaths compared to a counterfactual two percent annual mortality reduction, representing the average decrease in annual fatalities rates for all (not just non-Hispanic) white 45-54 year olds from 1970-2000. They conclude that approximately 600,000 deaths among this group would have been avoided if mortality rates had continued to decline at this rate through 2017. Importantly, they point out that the death rates of 45-54 year olds in other wealthy countries, such as France, the United Kingdom and Sweden, continued to decrease over this period, making the U.S. an outlier. They also note that while the mortality rates of 45-54 year old whites increased in all but six states, the largest growth occurred in those such as West Virginia, Kentucky, Arkansas and Mississippi, that have relatively low average levels of education.

Chapter 3 introduces "deaths of despair" which is how Case and Deaton characterize fatalities resulting from drugs, suicide and alcohol.[1] In their empirical implementation, the last of these is proxied by chronic liver disease mortality, an important distinction because these both exclude many alcohol-related deaths and include fatalities that are unrelated to drinking.[2] For this reason, and to avoid the presumption of causality, I refer to these as drug, suicide and (chronic) liver disease mortality, frequently abbreviated as *DSL* below.

In connecting these deaths, Case and Deaton write: "the three causes of death are deeply related … All the deaths show great unhappiness with life, either momentary or prolonged. It is tempting to classify them all as suicides." (p. 39). They distinguish accidental drug poisonings from other types of accidental deaths arguing that "People who seek out such drugs [heroin and fentanyl] are not seeking death … but the high risk of death is no deterrent." (p. 39)

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[1] Interestingly, the term "deaths of despair" does not appear anywhere in their original research on this topic (Case and Deaton 2015) but is referred to 24 times in their follow-up paper (Case and Deaton 2017).

[2] The Centers for Disease Control and Prevention define a set of 54 acute and chronic causes of death that are partially or fully due to alcohol and provide a formula using these to calculate the total of fatalities and lost life years attributable to alcohol (Centers for Disease Control and Prevention 2020a). They classify the causes into those that are: 100 percent due to alcohol; directly attributable to alcohol but at less than 100 percent; and indirectly attributable, generally at much lower percentages. Chronic liver disease, includes ICD-10 codes K70, K73 and K74, which correspond, respectively, to "alcoholic liver disease", "chronic hepatitis not elsewhere classified" and "fibrosis and cirrhosis of the liver". According to the CDC, the attributions of alcohol to these three categories are 100 percent, <2 percent and <40 percent, respectively. Thus, the last two of these causes should, at most, be only partially included in the total of alcohol-related deaths. On the other hand, there are numerous other ICD-10 causes that are 100 percent attributable to alcohol – such as alcoholic psychosis, alcohol dependence syndrome, alcohol polyneuropathy, alcoholic gastritis and alcohol-induced chronic pancreatitis – and others, such as chronic pancreatitis, that while not 100 percent due to drinking, have a much higher attribution (84 percent in this case) than fibrosis and cirrhosis of the liver, or chronic hepatitis.

After highlighting the increases in DSL deaths for white 45-54 year olds since 1999, Case and Deaton also point to the simultaneous slowdown in progress reducing heart disease mortality in the U.S., in contrast to the continued declines in other wealthy countries.[3] While they are unable to identify the reasons for the unfavorable U.S. experience, they note that "drugs and alcohol associated with deaths of despair may make people more likely to die from heart disease" (p. 43) and so raise the possibility that some of these deaths might also reflect despair. With that said, they note that smoking, which remains high in some parts of the country, and increases in obesity may also be undermining progress in reducing heart disease mortality. They conclude the chapter by relating that DSL mortality rate were increasing even faster for younger whites than for those in midlife, although levels remained higher for the latter group.

Chapter 4 focuses on differences in the experiences of education subgroups, arguing that there is a clear separation in quality of life for those with and without a Bachelor's degree. College graduates earn more, and the disparity has dramatically increased since the late 1970s, they are more likely to be employed, and have lower risk of unemployment. The differences go well beyond economic circumstances. Highly educated individuals marry later, less often get divorced, live in areas with better schools and report higher life satisfaction. Case and Deaton argue that the increasing economic inequality has led to a harsher atmosphere in corporations and advantage seeking strategies among the privileged that are socially destructive.

The less educated also die earlier, in part due to less healthy behaviors such as higher smoking rates. Case and Deaton provide evidence that the mortality rates of 45-54 year old white men and women without a college-degree increased much more in absolute terms than for their more educated counterparts. Finally, they show that age-specific mortality rates were higher for later than earlier birth cohorts at virtually all ages analyzed, and that the absolute size of the cohort differences was much larger for people without than for those with a bachelor's degree.[4]

The fifth chapter focuses on the heterogeneous experiences of (non-Hispanic) blacks and whites. Case and Deaton provide evidence of faster mortality rate declines for 45-54 year old blacks than whites from 1970-2013, although starting from much higher levels and without achieving full convergence. The decrease in black mortality rates slowed in the

[3] Although, progress also largely stopped in other rich English-speaking countries (Ireland the UK, Canada and Australia) after 2010.

[4] A caveat to this last finding is that Case and Deaton show absolute but not relative changes in cohort mortality rates. This could matter since more educated groups have much lower death rates so that similar percentage increases would result in smaller absolute changes for them.

1980s, largely due to the crack and HIV epidemics, but with rapid reductions resuming in the mid-1990s, just as white progress stopped. An important source of this latter difference is that there was no increase, and in some periods actually a reduction, in black DSL mortality rates prior to 2014, when the emergence of fentanyl led to rapid increases in death rates for blacks without a college degree.

In attempting to explain these sharp racial differences, Case and Deaton argue that the slowdown in progress reducing mortality for blacks in the 1980s foreshadowed what would occur for whites 25 years later. They emphasize how inner-city African Americans were especially harmed by declines in manufacturing and transportation industries and the social disintegration that accompanied them. They note both similarities and differences between the crack cocaine epidemic of the 1980s and the current opioid epidemic stating: "A fundamental force in both cases was the lack of working-class jobs … In both epidemics, drugs that could ease psychological or physical pain were available … to populations that were hungry for the escape they seemed to offer." (p. 69). However, they caution against overstating the similarities noting that patterns of suicides differ markedly by race.

Since mortality is not the only indicator of health problems, the next two chapters focus on morbidity (ill-health) and pain. After discussing difficulties in assessing health among the living, chapter 6 provides evidence that 25-60 year old whites were more likely to self-report their health as poor or fair (rather than good, very good or excellent) in 2017 than in earlier years, with particularly large deterioration in self-reported health for those with less than a Bachelor's degree. Severe mental distress also rose for less educated 25-70 year old whites, and working-age whites without a college degree reported increasing difficulty engaging in activities such as walking, climbing stairs, shopping or socializing with friends. One possible consequence is that the share of prime-age whites without a college degree reporting that they were unable to work rose steadily after the early 1990s.

Chapter 7 discusses pain, which Case and Deaton point out is hard to measure but strongly associated with suicides. The key results are that the fraction of U.S. whites reporting pain during the previous day is: marginally higher for 40-65 year olds than in 19 comparison countries; greater for U.S. 60 year olds than at later ages; lower for those with a bachelor's degree than for those with less schooling; and that pain increased over time for less educated whites but possibly not for those with a 4-year degree.

The sources of this growth in pain are somewhat mysterious. Case and Deaton believe that increases in obesity may account for around one-quarter of the change but they are unable to explain the remainder of it and note that the shift from manufacturing to service

employment seems likely to operate in the opposite direction.[5] However, they also point out that geographic areas with relatively high levels of self-reported pain tend to also have elevated DSL mortality rates, raising the possibility of a connection between the two.

2b. DSL Mortality: Details

The next two chapters provide a more granular discussion of mortality from drugs, suicide and chronic liver disease. A key hypothesis in Chapter 8 is that all three sources of death can be thought of as a form of suicide. Thus, Case and Deaton write: "Suicides are deaths of despair. But the circumstances that can lead to suicide find less extreme forms when people turn to drugs or alcohol to seek refuge from pain, loneliness, and anxiety … grouping deaths from suicide, alcohol, and drugs captures a common underlying cause – despair – that is not easily captured when they are treated separately … [and] there was nothing unintentional about the use of intoxicating substances. Therefore, the resulting fatal drug overdoses or interactions were not true accidents." (p. 95-97) They emphasize the difficulty in correctly categorizing the cause of mortality on death certificates and that fatal overdoses are classified as accidents, unless there is clear evidence that death was the intended result.[6]

The remainder of the chapter focuses on the role of social turmoil in contributing to suicide and alcohol deaths. Case and Deaton highlight the rise in suicides for whites since the late 1990s, emphasizing that while there is no simple theory identifying who will kill themselves, growth over time in social causes such as pain, loneliness, depression, or joblessness provide one reason why suicide rates may have increased. They note that the highest suicide rates occur in sparsely populated parts of the country, where firearms are also common and reports of pain are the highest, while cautioning that these patterns could reflect confounding factors. Importantly, they emphasize that the negative association between white suicide rates and education is a relatively recent phenomenon. The chapter concludes with a discussion of alcohol, first highlighting the many health problems heavy drinking can cause and then pointing out that while the use of any alcohol is positively associated with education in the U.S., more dangerous binge and heavy alcohol consumption are more common for the less educated, particularly for more recent birth cohorts.

Chapter 9 provides a brilliant discussion of opioids, starting with the Opium war between China and Britain in the early 19th century and extending through their current key contribution to recent increases in DSL mortality. Case and Deaton describe the different

[5] A recent analysis by Cutler et al. (2020) finds that knee pain (the most common musculoskeletal complaint) declines with education, with greater pain levels among the less educated largely explained by higher rates of obesity, more physically demanding jobs, and interactions between the two.

[6] This is not quite correct. A significant fraction of overdose deaths are classified as being of undetermined intent.

types of opioids, the mechanisms through which they operate and cause addiction, and the sources of the current opioid epidemic. The latter include changes in medical practices related to the treatment of pain and the promotion of opioids for this treatment, particularly after the introduction of OxyContin in 1996. The led to growth in opioid involved overdose fatalities, often in combination with other drugs such as benzodiazepines. Prescription opioid deaths peaked around 2011, resulting from the reformulation of OxyContin to a more abuse-resistant version and other measures to reduce prescribed opioids. However, when combined with changes in illicit drug markets, this led to the substitution of prescribed opioids with cheaper but more dangerous heroin. After 2013 heroin and other street drugs were increasingly adulterated with still more powerful and hazardous fentanyl, further accelerating the rise of fatal overdoses.

Nevertheless, Case and Deaton view these supply-side factors as "misbehavior that poured fuel on the fire" to make the epidemic of despair worse than it would have otherwise being, rather than being a root cause of it. Instead, *DEATHS* primarily focuses on the demand-side of the white working class "whose already distressed lives were fertile ground for corporate greed, a dysfunctional regulatory system, and a flawed medical system" (p. 126).

2c. It's Not (Just) the Economy

In Chapter 10, Case and Deaton argue that economic conditions were directly responsible for the rise in DSL mortality. Money does buy access to healthcare and eases many financial worries, particularly in the U.S with its weak social safety net. But while noting a correlation between economic inequality and death rates, they argue that these are joint consequences of broader forces damaging to the white working class, rather than being central to their main story. They highlight the absence of any consistent pattern of poverty growth at the end of the 20th and beginning of the 21st century, when DSL mortality rates were rising, or of corresponding correlations between poverty and DSL rates across geographic regions. Importantly, trends in DSL mortality rates among white 45-54 year olds were essentially unaffected by the Great Recession of 2008 and 2009, rising at virtually the same rate during that period as before or after it. Nor was there any substantial growth in DSL deaths during the Great Recession in European countries that suffered far greater declines in economic output and implemented more severe austerity measures than the U.S.

Chapter 11 emphasizes the growing divide in the circumstances of life – both living standards and the accompanying social disintegration – between those with and without a Bachelor's degree. Case and Deaton point to rising inequality in the U.S. where the benefits of growth, which slowed dramatically at the end of the 20th century, predominantly went to those who were already most advantaged. One consequence was that white males with less than a BA degree earned less, after adjusting for inflation, than previous cohorts whereas

there were substantial earnings increases among those with college degrees. Such stagnation in long-term economic progress did not occur in other industrialized countries. Moreover, the probability of working at all declined substantially for 25-54 year old white men after 1990, and even more so subsequent to 2000 for those with without a college degree.[7] It is not just earnings or employment rates that changed for the less educated Americans, but also the nature of the work itself. Here, Case and Deaton emphasize the decline of unions, which gave workers a voice, and the increased difficulty for these individuals to work their way up to managerial positions.[8]

The arguments in chapters 10 and 11, just described, are not fully reconciled. However, a unifying theme is that economic conditions were not directly determinative but may have led to a longer-term disintegration of a way of life for less educated whites that ultimately led to rising DSL mortality rates. Indeed, in chapter 12, Case and Deaton explicitly argue that the reductions in the labor market opportunities for less educated whites resulted in declining marriage rates, increases in nonmarital childbearing and decreases in community participation over dimensions such as voting, union membership and weekly church attendance.[9] They conclude by restating their hypothesis that declining material disadvantage worked through its effects on family, community and religion, rather than directly. They suggest that changes over time, not just levels, may have been important when noting that although blacks do worse than whites on most indicators, their lives were improving in many dimensions, while those of whites were worsening, possibly explaining the racial differences in DSL mortality experiences during the 21st century.

2d. American Capitalism Today

Chapters 13 through 15 document how the form of capitalism practiced in the U.S. today is failing less educated Americans. Case and Deaton characterize the American healthcare system as the "leading villain" in their story but not the only one. In the first of these chapters, they do not emphasize the overprescription of opioids, the absence of treatment, or medical mistakes, but rather the indirect harms resulting from excessively high medical costs. They document that large expenditures have not led to superior health outcomes,

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[7] The patterns of wages and employment-to-population ratios are less clear for white women due to the large increases in female labor force participation rates occurring during the second half of the 20th century.

[8] They also make arguments that some may find questionable. For instance, when discussing the change in the nature of work for the less educated, they contrast jobs in the post-World War II period stating "Now the man went to the factory, where he … found meaning in the dignity of his difficult and productive labor rather than in the size of his paycheck." (p. 164), with the less desirable work by later cohorts in services such as healthcare, cleaning and security.

[9] It is worth pointing out that some of these outcomes may have positive, as well negative, aspects and that their trends do not always differ systematically by education or race.

that Americans are not happy with their health care system, and that the major drivers the differences in U.S. medical care costs, relative to other countries, are higher prices.[10] They point to the practice of "surprise billing" – where emergency department and other medical services are outsourced to companies outside the patient's network – and to reductions in hospital competition resulting from mergers. They indicate that hospitals are now run as profit centers managed by corporate executives (rather than medical practitioners) and emphasize the various ways that health care providers of all types work together to raise prices, including through extensive and growing lobbying efforts. The resulting high costs reduce the percentage of income available for other purposes, including wages of the less educated, particularly with existing tax distortions that encourage an excessive share of total compensation in the form of health insurance.

Chapter 14 provides evidence that many markets have become less competitive and then describes three threats to low-wage workers – immigration, globalization, and automation. They argue that these are not alone the cause of rising DSL deaths; however, compared to other industrialized countries, their effects may be magnified by the weak social safety net which, in turn, reflects the American tradition of individualism, its unique history connecting race with immigration, and the historical accident by which health insurance came to be dominated by employers.

The 15th chapter documents increased concentration in many industries, resulting from growth in mergers and a declining number of startups. Profit rates have also risen, although this may partially reflect a greater role for large firms due to increasing returns to scale. Interestingly, while they believe that growth in market power has caused higher prices and lower wages in healthcare and possibly airlines and financial institutions, they *not* think this has occurred more generally. It is difficult to reconcile this conclusion with the concerns highlighted in the remainder of the chapter. These include the increased monopsony power of employers, partly due to the growth of noncompete agreements and outsourcing, with the consequential downgrading of work by large employers, and the dramatic weakening of private sector unions. They also emphasize changes in the ways firms are run, with Board of Director's frequently believing their sole obligation is to shareholders – rather than also to employees, customers and communities – and how very wealthy individuals and corporations, often through their lobbying efforts, have increased influence in implementing policies that support the already advantaged.

The last chapter provides Case and Deaton's policy recommendations. With regard to the opioid crisis, they discuss medically assisted treatment, state Medicaid expansions, and opioid prescribing regulations. They mention potential broader health system changes

[10] These issues are well known and have been extensively studied (e.g. Reid 2010; Rosenthal 2017).

related to universal health insurance coverage and health care cost controls. Minimum wage and income support policies are then covered, as are reforms to the American education system. Finally, they turn to potential modifications in policies related to corporate governance.

3. Research Questions and Challenges to Key Hypotheses

DEATHS argues that the declining economic and social circumstances of less educated whites, brought on in large part by a system of American capitalism that tilts the playing field in favor of the wealthy and advantaged, has led to increases in despair and death among this group and possibly others. The theory is audacious, provocative, and upsetting. Case and Deaton provide an array of arguments and evidence in favor of various aspects of it. However, much additional research is needed to confirm or refute the key connections, address substantial challenges to the model as articulated, and to evaluate how well its predictions conform to the data, compared to plausible alternatives. These issues are discussed next.

3a. Counterfactual Mortality Rates

Case and Deaton's foundational proposition is that the accumulation of the excesses of American capitalism during the last half century or so have led to the plight of non-college educated midlife whites. The initial discussion does not focus on the modest growth in mortality rates experienced by this group at the beginning of the 21st century but rather on the much larger difference between the change in actual death rates and a counterfactual estimate of reductions that would have occurred if prior trends had continued. Specifically, they calculate that if the mortality rates of white 45-54 year olds had declined by two percent annually, this group would have experienced 600,000 fewer deaths from 1999-2017 than actually occurred.

The assumptions underlying these counterfactual trends are fairly strong. One justification for them is that fatality rates for midlife adults have continued to fall in other industrialized countries, although not necessarily by two percent per year.[11] But it is unclear how relevant these comparisons are for the U.S. where, for example, smoking rates began to fall much earlier than in most of Europe (D. M. Cutler and Glaeser 2009; OECD 2019). This raises the possibility that the mortality reductions from tobacco-related problems, such as lung cancer and cardiovascular disease, may have been mostly complete by 1999 in the U.S. but not yet elsewhere. Other non-despair related factors could also more negatively affect the

[11] For instance, the mortality rates of 45-54 year olds in the United Kingdom declined at a slower rate from 1999-2017.

life expectancy of midlife whites in the U.S., relative to those in other wealthy countries, such as the particularly elevated U.S. obesity rates (OECD 2017).

Nor is the counterfactual two percent annual mortality rate decline for the U.S. fully convincing. It is based on 1970-2000 trends for all whites of this age, *including Hispanics*, whereas the excess deaths from 1999-2017 refer only to non-Hispanic whites.[12] This matters because Hispanics had relatively low and falling mortality rates (Ruiz, Steffen, and Smith 2013) and their share of the population increased over time, suggesting that some of the reductions for all whites (including Hispanics) partially reflect these compositional changes and likely exceeded the decrease for non-Hispanic whites alone.[13] The death rates of 45-54 year old whites also remained relatively flat from the mid-1950s through around 1970, implying that the counterfactual trend reductions would have been considerably smaller if they had based on a 1955-2000 rather than 1970-2000 trends. Finally, factors such as the previously mentioned reductions in smoking or the introduction of widespread use of hypertension medications late in the 20th century (Saklayen and Deshpande 2016), might have decreased mortality trends during that period in ways that could not be duplicated at the beginning of the 21st century.

The results are also sensitive to the starting year over which recent mortality changes are calculated. Case and Deaton, along with many other researchers (including myself in earlier work), use 1999 as the initial year because that is when the method of categorizing deaths switched from the 9th to the 10th revision of the International Classification of Diseases coding system (from ICD-9 to ICD-10 codes). This may provide a misleading indication of the longer-term developments because it happens to coincide with the beginning of particularly unfavorable mortality changes. For example, the death rates of 45-54 year old whites *fell* by 0.8 percent per year from 1990-1995 and by 1.6 percent annually from 1995-1999.[14]

Using reasonable alternative counterfactual trend assumptions dramatically changes the conclusions about the number additional midlife white lives lost. This is illustrated in Table 1, which provides information on fatality rates in 1999 as well as actual and counterfactual rates in 2018. Mortality rates per 100,000 increased by 27.9 (from 384.1 to 412.0) from 1999-2018 but the growth in death rates was more than 5-times as large, 150.4 per 100,000,

[12] Hispanic origin was unavailable prior to the 1990s, so that the longer-term historical comparisons relied on combined data for Hispanic and non-Hispanic whites.

[13] For instance, the Hispanic share of the U.S. population grew from 4.5 percent in 1970 to 12.5 percent in 2000 (U.S. Census Bureau 2012).

[14] These estimates were obtained from my analysis of death certificate data, and using bridged-race population estimates, both available from *CDC Wonder* (Centers for Disease Control and Prevention 2020b). Similar patterns were found for 25-34, 35-44 and 55-64 year old whites.

relative to Case and Deaton's two percent annual counterfactual decline. However, if the observed annual reduction in death rates from 1990-1999 were instead used as the counterfactual, the difference shrinks more than 30 percent, to 104.2 per 100,000, and utilizing observed trends from 1990-1995 reduces the original estimate by 53 percent, to 71.4 per 100,000.

The purpose of this exercise is not to claim that any one of these counterfactuals are necessarily correct but rather to highlight the need for further research on this issue before arguing for any particular choice. This is important, in part, because increases in DSL mortality explain all or almost all of the actual rise in death rates during the 21st century for midlife whites, as shown below, but relatively little of the difference when compared with Case and Deaton's assumption of continued rapid fatality rate reductions. Further, as they point out, much of the stagnation in mortality rates reflects slowing progress in reducing coronary artery disease deaths (Mehta, Abrams, and Myrskylä 2020), but we poorly understand why this has occurred.

3b. Do DSL Fatalities Result from Despair?

The organizing principle of *DEATHS* is that profound changes in the United States have led to an "epidemic of deaths of despair".[15] Beginning with the book's title and throughout the discussion, it would be difficult to come away with the impression that DSL is anything other than a coherent grouping of similar causes of death that reflect the consequences of underlying distress. However, Case and Deaton actually use the term "deaths of despair" in multiple ways. For instance, early on they state: "'deaths of despair' … is a convenient label, indicating a link with unhappiness, the link with mental or behavioral health, and the lack of any infectious agent, but it is not intended to identify specific causes of despair" (pp. 40). But later in the volume they more explicitly claim: "grouping deaths from suicide, alcohol, and drugs captures a common underlying cause – despair – that is not easily captured when they are treated separately" (p. 96).

Thus, Case and Deaton believe that "deaths of despair" is both a convenient label and a deeply rooted indicator of great unhappiness? I am less convinced. Moreover, I would suggest that few can be repeatedly exposed to this term without assuming it identifies despair as the root cause of these deaths. It is precisely to avoid this connotation that I use the "drug, suicide and chronic liver disease" label throughout this essay. Whether or not despair is the ultimate cause of DSL mortality can then be treated as a testable proposition.

[15] For example, the concluding chapter discusses "what the U.S. can learn from other countries so that we might undo the epidemic of deaths of despair" (p. 259), with similar usage in multiple places elsewhere.

I am particularly concerned with Case and Deaton's claim that "it is tempting to classify them all as suicides". (p. 39) While some types of drug and alcohol use undoubtedly increase the risk of death, such general categorizations seem problematic. For instance, suicide seems a wildly inaccurate description for someone who overdoses after becoming addicted to excessively prescribed opioids following a surgical procedure; or to an individual with a genetic predisposition towards alcoholism, who is desperately trying but failing to stop drinking. And what about smokers? Using the same reasoning, tobacco-related deaths could be categorized as suicides, since the vast majority of smokers are aware of the health risks. However, few would classify smokers as suicidal, nor would it be appropriate to do so.[16]

Even suicides, which unlike deaths from drugs or chronic liver disease inarguably reflect despair, often result from transitory rather than permanent distress. An important public health approach for reducing intentional deaths is to restrict the availability of the means for self-harm (Yip et al. 2012). This saves lives because the vast majority of those surviving suicide attempts do not kill themselves later (Barber and Miller 2014). Unfortunately, suicide risk has probably risen in recent years, even holding constant overall despair, due to the widespread availability of potentially fatal drugs, like opioids (T. R. Miller et al. 2020), and expanding presence of guns (M. Miller and Hemenway 2008).

In reality, the term "deaths of despair" conceals more than it illuminates. If the different types of DSL fatalities resulted from the same causal factors, we would expect to observe fairly stable relationships between them across demographic groups, time periods and locations. But we do not. Geographic patterns of deaths from these causes vary substantially across rural and urban areas (Stein, et al., 2017), and with distinct county-level trends in deaths from drugs, suicide and alcohol use disorders (Dwyer-Lindgren et al. 2018). The age profile of rising deaths differs over time markedly for the three causes, as discussed below, and patterns of opioid fatalities vary substantially across areas of the country (Monnat et al. 2019). There are also heterogenous responses to shocks. Case and Deaton show that DSL mortality is tenuously related to most economic factors, with further evidence provided by Brown and Wehby (2019) and Ruhm (2019a). However, higher minimum wages are associated with reductions in suicides but not drug or alcohol deaths (Dow et al. 2020), while greater exposure to international trade predicts increases in fatal overdoses but not in suicide or liver disease mortality (Pierce and Schott 2020). There have also been substantial changes in the age and sex groups most affected as the dominant source of fatal overdoses shifted from prescription to illicit opioids (Ruhm 2019a).

[16] If lung cancer fatalities were included in "deaths of despair", it would at least partially undermine Case and Deaton's arguments, since mortality from this source has continued to fall in recent years (Lewis et al. 2014).

3c. Differential Increases in Drug, Suicide and Chronic Liver Disease Mortality

The contributions of drugs, suicide and liver disease fatalities to the overall rise in the mortality rates of midlife whites differ dramatically. This is illustrated in Table 2, which displays age-standardized mortality rates, per 100,000, in 1999 and 2018 for 25-54 year old whites from all causes, DSL and, separately its three components; also included for comparison purposes are death rates from respiratory diseases, which have also grown over time but presumably do not reflect despair.[17] Intentional overdose deaths are separated from other self-harm fatalities since it is unclear whether they are best categorized with drug deaths or suicides.

Two results from the table are particularly salient. First, DSL and all-cause mortality rates increased by roughly the same amount from 1999-2018 for 25-54 year old white women (32.3 versus 29.5 per 100,000), while the rise in DSL deaths rates was 74 percent larger than that for total mortality rates among corresponding men (60.8 versus 34.9 per 100,000). This implies that the growth in DSL fatalities was sufficiently large to explain the entire increase in midlife white female death rates over the period and that had DSL mortality not increased, the all-cause death rates of 25-54 year old white males would almost certainly have declined substantially.[18] Second, non-intentional drug fatalities were by far the most important contributor to these trends, accounting for 78 percent of the overall rise in DSL mortality for white men and 73 percent for women. Chronic liver disease played essentially no role for males, while mortality from this source and from non-drug suicides each explained about one-eighth of the increase in female DSL death rates.[19] Mortality rates from respiratory disease, which do not reflect despair, rose by roughly the same magnitude as those for chronic liver disease. This provides a warning about somewhat arbitrarily choosing

[17] Mortality rates are calculated using death certificate data from the *Multiple Cause of Death* files (Centers for Disease Control and Prevention 2020b) and population data from the *Surveillance Epidemiology and End Results (SEER)* program (National Cancer Institute 2020). They are age-standardized using 2018 weights for five-year population groups (25-29, 30-34, 35-39, 40-44, 45-49, and 50-54). The analysis focuses on 25-54 year olds because this is the age category of whites experiencing increases in mortality rates after 1999. Additional estimates for age subgroups within this category are provided in section 4d.

[18] Had DSL mortality rates not risen, there probably would have been some additional deaths from other causes. However, given the relatively low overall fatality rates for this age group, adjusting for these competing risks would be unlikely to change the main conclusions.

[19] The definition of chronic liver disease used here is the same as that used by Case and Deaton. If 100 percent, 40 percent and 2 percent of alcoholic liver disease, fibrosis and cirrhosis of the liver, and chronic hepatitis were instead attributed to alcohol, as the CDC does, changes in the associated death rates per 100,000 would have been somewhat larger for males (2.3 instead of 1.5) and smaller for females (3.4 instead of 4.1).

the causes of death to focus upon and then providing ex post interpretations for why they increased.

Another way of highlighting the key role of drug fatalities is to note that their growth was sufficient to explain 135 percent of the all-cause mortality rate increase for 25-54 year old white males from 1999-2018 and 82 percent of the rise for corresponding white females. The importance of rising drug deaths is fully acknowledged by Case and Deaton. However, what is less obvious is their implication that the causal mechanisms leading to these increases are similar to those for suicide and chronic liver disease.

3d. 45-54 Year Olds vs. Other Age Groups

The evidence of rising white mortality rates presented in *DEATHS* focuses heavily on 45-54 year olds. For example, nearly all of the figures and discussion in the first five chapters, where these patterns are described, exclusively supplies information for this age group. Case and Deaton justify this by stating that: "It is in midlife death rates pick up, and it is often a good place to see evolving trends in mortality." (p. 25) This reasoning makes sense if the patterns of DSL death rates for this age group are representative of those for all prime-age whites, but there is reason to doubt that they are.

To show this, Table 3 presents findings on mortality rates corresponding to those in Table 2, except with 25-54 year olds separated into 10-year age subgroups. Three results deserve attention. First, the increase in white male all-cause mortality rates was five times as large for 25-34 as for 45-54 year olds, and the rise in DSL death rates was 22 percent greater. Conversely, growth in total and DSL death rates was similar for all three age groups of females. Second, while rising drug mortality was the dominant source of the growth in DSL deaths for all age groups, this was particularly true for 24-44 year olds, where they account for approximately four-fifths of the total increase. Third, and most importantly for this discussion, the patterns for 45-54 year olds differed substantially from those of the younger two age groups, with non-drug suicides accounting for a relatively substantial (27 percent) share of the increase in DSL death rates for 45-54 year old males, while these and chronic liver disease fatalities combined for 35 percent of the rise for same aged females. Deaths from respiratory causes, which were unrelated to despair, also rose rapidly for 45-54 year old whites but much less so for their younger counterparts.

A full examination of these age differences is well beyond the scope of this review but the results just described emphasize that the experiences of white 45-54 year olds provide an incomplete understanding of the composition, and probably the causes, of rising DSL

deaths.[20] In particular, suicide and liver disease deaths were more important for this agegroup than for younger whites and so, when focusing on 45-54 year olds, it may appear that drug deaths were part of a broader phenomenon that included suicide and chronic liver disease deaths. Conversely, the dominant role of fatal overdoses is more apparent when focusing on 25-34 or 35-44 year olds, raising the possibility of distinct causes.

In several places, Case and Deaton incorporate information for other age groups by examining outcomes separately by age and birth cohort.[21] Their earlier work (Case and Deaton 2017) hypothesizes that the observed patterns reflect the effects of cumulative disadvantage, whereby later cohorts suffered from adverse social and economic consequences for larger portions of their lives, resulting in higher mortality rates at given ages. While such an explanation is consistent with the cohort patterns, a plausible alternative is that the size of negative health shocks from sources such as dangerous opioids have grown over time for all groups, and that younger cohorts have higher death rates at specified ages because they reach them in later periods when the deleterious shocks are larger.[22]

3e. What about Nonwhites?

The extremely disparate race/ethnicity patterns of mortality changes among midlife adults presents perhaps the most significant challenge to the "deaths of despair" hypothesis, as articulated by Case and Deaton. In particular, why were the rising mortality rates observed for whites at the beginning of the 21st century largely absent for nonwhites and Hispanics, at least until the emergence of illicit fentanyl after 2013.

Case and Deaton briefly mention the well-known "Hispanic paradox", whereby Hispanics have relatively low and falling mortality rates, despite being disadvantaged along many dimensions (The Lancet 2015); however, no attempt is made to account for these patterns within the framework of despair, nor the continuing favorable mortality trends of nonwhites other than blacks.[23]

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[20] Currie and Schwandt (2016), Ruhm (2018), or Woolf and Schoomaker (2019) provide more detailed analyses of changes in age-specific mortality rates.

[21] For instance, as mentioned in the discussion of Chapter 4 above, they show that the DSL mortality rates of 25-50 year old less educated whites were higher at virtually all of these ages for more recent than for earlier birth cohorts.

[22] The difficulty in separating age, period and cohort effects is well known (Hobcraft, Menken, and Preston 1982; Aguiar and Hurst 2013; Bell and Jones 2013). Cutler (2017) provides a discussion specific to this application. Masters et al. (2018) attempt to separately estimate period and cohort effects and argue that rising drug mortality is related to period rather than cohort effects, reflecting changes in the availability and misuse of opioid-based pain killers.

[23] Instead, what they say about this is that "Hispanics are a widely heterogeneous group … [and] we do not try to tell a coherent story for them" (p. 6).

DEATHS includes a full chapter addressing differences in the mortality experiences of whites and blacks. However, the dramatic divergence in mortality trends is not satisfactorily explained. As discussed, Case and Deaton highlight the stagnation in black life expectancy gains occurring during the early 1980s and early 1990s, arguing that this was a "foreshadowing of our account of whites in the twenty-first century" (p. 67). But how so? The slowdown of progress in reducing black mortality rates during this period is barely perceptible in the data they show. Moreover, less educated whites and blacks were largely buffeted by the same social and economic forces beginning in the last quarter of the 20th century,[24] so why were blacks affected so much earlier and why did their mortality rates resume a rapid decline starting in the mid-1990s, just as white death rates began to increase?

Case and Deaton acknowledge that the twin forces driving the poor mortality performance of blacks near the end of the 20th century were the crack and HIV/AIDS epidemics. Deaths from crack cocaine emerged around 1980 and peaked in 1989, after which they substantially although not completely receded (Fryer et al. 2013).[25] HIV/AIDS mortality materialized around 1981, grew rapidly through approximately 1995, and declined considerably thereafter (Centers for Disease Control and Prevention 2001). Both crack and HIV/AIDS had far more deleterious mortality consequences for blacks than for whites (Blankenship et al. 2005; Coile and Duggan 2019). The two epidemics were also intertwined as crack cocaine use was associated with higher risk of HIV infections among poor, inner-city, and mostly black communities (Edlin et al. 1994).[26] The key point is that these two negative health shocks, which disproportionately affected blacks, were almost certainly not primarily caused by despair although as with the subsequent opioid epidemic, demand-side factors played a role in which groups were most affected by the new, more risky, form of an existing illicit drug.

An unanswered puzzle in *DEATHS* is why nonwhites were largely immune from rising DSL mortality from 1999 to 2013 (when fentanyl emerged) or why suicides remained so much less frequent for blacks than for whites, even while reported patterns of pain were fairly similar. For the first of these, the seemly anomalous racial differences can probably be explained by recognizing the deleterious consequences of opioids and other dangerous drugs often prescribed with them (e.g. benzodiazepines).

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[24] For instance, the economic returns to education during the 1980s and 1990s were comparable for blacks and whites, and declines in marriage rates were also fairly similar, although increases in non-marital births did begin to rise somewhat earlier for blacks (Barrow and Rouse 2005; Parker, Horowitz, and Mahl 2016).

[25] The emergence of crack cocaine represented a supply-driven innovation, paralleling that for fentanyl later; crack provided users with an extremely intense and addictive high that increased the profitability of sales by drug dealers (American Addiction Centers 2019).

[26] HIV/AIDS death rates continued to fall after the turn of the century, albeit by more modest amounts (Diamond 2018), which benefited blacks more than whites.

During the late 1990s and first decade of the 2000's, the widespread use of opioids was increasingly promoted as good medical practice.[27] One result was that whites, who generally receive more adequate medical care than minorities, were more often prescribed opioids (Anderson, Green, and Payne 2009; Burgess et al. 2014; Singhal, Tien, and Hsia 2016) and were more likely to die from them as a result. It is notable that the mortality rates of prime-age blacks began to increase markedly when the opioid epidemic transitioned from primarily legal to illicit drugs, particularly after 2013 when fatalities involving fentanyl exploded (Spencer et al. 2019). Viewed as a manifestation of drug problems, rather than general despair, this timing makes perfect sense. Deaths often occurred when fentanyl was mixed with illicit drugs, such as heroin or cocaine, from which nonwhites had long faced relatively high mortality risk.

None of this implies an absence of despair of among less educated white Americans today. To the contrary, suicides, while a relatively small share of the total increase in DSL mortality, reflect a combination of severe distress and availability of the means to self-harm. Graham and Pinto (2019) document lower levels of optimism for less educated whites than minorities and link this with premature mortality from DSL and closely related causes among 35-64 year olds. Blanchflower and Oswald (2019) show that life satisfaction, which reaches a trough for adults in their middle 40s and early 50s (the peak ages for suicide), has fallen modestly over time for whites since the 1970s, while trending upwards for blacks.

A potential source of these racial differences in wellbeing, hinted at by Case and Deaton, is the loss of some degree of preexisting "white privilege", resulting from reductions in discrimination that somewhat improved the position of nonwhites relative to less educated whites.[28] This possibility is certainly deserving of further research. To the extent this has occurred, it represents a partial success of the American economic and social system, and is certainly not one that should be reversed.

3f. Education Categories

In the introduction to *DEATHS*, Case and Deaton write: "A four-year degree has become *the* key marker of social status, as if there were a requirement for nongraduates to wear a circular scarlet badge bearing the letters *BA* crossed through by a diagonal red line." (p. 3) But there are multiple issues that must be addressed when determining whether the lines are this definitive, providing fertile ground for future research.

[27] In large part this reflected aggressive marketing efforts by pharmaceutical manufacturers as well as recommendations for aggressive therapeutic opioid use to treat "pain as the 5th vital sign" (Quinones 2016; Baker 2017; Presidents Commission 2017).

[28] Metzl (2019) provides a more sophisticated discussion of this point.

The first is that increases over time in schooling change the composition of education groups (Dowd and Hamoudi 2014; Bound et al. 2015; Goldring, Lange, and Richards-Shubik 2016). Consider a situation where educational attainment at a point in time is monotonically increasing in individual quality (measured by some criterion). Secular increases in schooling then imply that the marginal college graduate in a later cohort will then be less capable than B.A. degree-holders in an earlier one and that the average quality of both educational groups will have fallen over time.[29] This increasing negative selection suggests that within-group trend reductions (increases) in mortality rates are likely to be understated (overestimated), although we cannot determine whether these biases will be larger for college graduates or the less educated.

A second, technical but potentially important, concern relates to changes in the recording of educational attainment on death certificates. Before 2003, education was coded as the number of grades completed. Starting in 2003, and gradually phased in across states, categorical data (e.g. bachelor's degree) were instead provided.[30] The conventional procedure, used by Case and Deaton, is to classify decedents with 16 years or more years of education as college graduates. The issue is that some of these persons have not received a bachelor's degree, while others with 15 or fewer years of schooling have obtained one.

We do not have direct data on the frequency of these classification problems but useful information is available from a similar modification in the reporting of education implemented in the *Current Population Survey* in 1992. Examining this change, Jaeger (1997) finds that 7.5 percent of those with 16 or more years of education had not graduated from college, while 2.1 percent of those with bachelor's degrees were coded as having fewer than 16 years of schooling. On net, this implies that the college degree group was overstated in earlier periods, when continuous rather than categorical education measures were used. While it is again not immediately obvious how this affects relative changes in fatality rates for the two education groups, available evidence suggests that the mortality rates of those classified as college educated were probably overstated in the early years, with the result that the trend reduction in their death rates was also be overestimated.[31]

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[29] Case and Deaton point out that the fraction of 45-54 year olds with a bachelor's degree remained roughly constant from 1990-2017, mitigating concerns for this age group. However, this is not true more generally. For instance, the share of white 25-29 year olds with a bachelor's degree rose from 34.0 percent in 2000 to 43.5 percent in 2018, with particularly large growth, from 35.8 to 48.4 percent, for females (Snyder, De Brey, and Dillow 2019; Table 104.20).

[30] For instance, education categories were used for 17, 53, 70, 97 and 100 percent of deaths with education reported in 2003, 2007, 2010, 2015 and 2018.

[31] Specifically, prior research indicates that the returns to education on wages are underestimated when using continuous years of schooling (Jaeger 1997; Frazis and Stewart 1999). This indicates substantial

Researchers have begun to use a variety of techniques to address these issues. The results to date are not fully consistent with Case and Deaton's conjecture of a clean demarcation for those with and without a B.A. degree. Using, a partial identification approach to bound changes in death rates from 1991-2015, Novosad, Rafkin and Asher (2020) find that mortality increases were concentrated in the bottom *decile* of the educational distribution, roughly corresponding to those who have not graduated high school. Leive and Ruhm (2020) examine mortality changes, from 2001-2017, for age-sex-race-specific education quartiles. For men, they show that the largest average mortality declines did occur for the highest education quartile, with little difference between the bottom three quartiles. Since the higher quartile roughly corresponds to college graduates, this is consistent with the categorization used by Case and Deaton. However, for women, average mortality trends improve fairly monotonically with education, which is not. Moreover, these mean differences conceal many age-sex-race groups where less educated members experienced more favorable, or no worse, mortality trends than their more educated counterparts.

3g. The Need for Hypothesis Testing

At this juncture, it is useful to restate the avenues postulated by Case and Deaton for rising DSL mortality rates during the 21st century. The first step is stagnation or deterioration of the economic circumstances facing less educated whites. These declines began by the 1970s and were probably partially due to exogenous factors, such as skill-biased technological change, but leavened by an unbridled capitalism that stripped away the protections that unions, minimum wages and the like had provided. Second, declining economic conditions led to the destruction of stabilizing social institutions, such as marriage and religious attendance, as well as other types of social capital. Finally, the obliteration of a previous way of life led to despair, ultimately culminating in rising DSL mortality of sufficient magnitude to lower life expectancy.

Substantial future research is needed to formally test whether these pathways are consistent with the evidence. Case and Deaton point to the difficulties of engaging in formal econometric testing stating: "Such techniques have their uses, but they are of little use to us here in describing the slowly evolving and large-scale disintegration that involves a historically contingent set of forces, many of which interact … Our readers will have to

negative selection into the B.A. plus group in the early years, where years of education were reported. Assuming similar patterns of health selection, this suggests relatively high baseline mortality rates for them. The exclusion of persons with 16 years of schooling but no B.A. from the college degree group in subsequent periods eliminates or reduces this negative selection and will lower measured death rates, even if there were no actual change.

decide whether our account is persuasive without the benefit of controlled trials or anything of the sort" (p. 190).

While I agree that strict causal identification strategies are not always achievable (Ruhm 2019b), this does not mitigate the importance of testing the empirical predictions and arguments to the extent possible. For this application it would be feasible to examine, albeit imperfectly, to what degree the hypothesized mechanisms plausibly account for the rise in DSL death rates. An obvious first step would be to estimate the extent to which declining economic conditions are associated with changes in DSL mortality.

Here, it is important to reiterate that Case and Deaton do *not* consider economic factors themselves to be responsible for rising death rates among less educated whites. Instead, they are hypothesized to operate indirectly, through their effects on the social and community factors that are viewed as the causal drivers. Nevertheless, the inclusion of economic controls in a regression that excludes the community and social capital variables should indicate a statistical association since they indirectly affect the dependent variables. These relationships may then be attenuated or eliminated with the addition of covariates for the social capital measures that Case and Deaton view as fundamentally causal. Alternatively, it might be possible to estimate more fully specified structural models that account for a wide variety of factors including, for example, endogenous migration between geographic areas which influences the composition of local populations and resulting death rates.

On the other hand, considerable care would be needed in interpreting direct regressions of mortality outcomes on community or social capital variables, since the latter may be influenced by many alternative factors. For instance, conservatives share many of the same concerns about the plight of less educated whites as Case and Deaton but argue that the causes are moral decay, including declines in social and family values, and the welfare state itself, rather than failures of the capitalist system (e.g. Murray 2012).[32] The models just described would not distinguish between these alternatives.

Efforts are underway to conduct at least some of the empirical testing just described. These studies generally uncover a statistical association between economic factors and DSL death rates, but the magnitudes are too small to explain more than a limited share of the mortality changes and some of the observed correlations probably reflect confounding factors (e.g. see Charles, Hurst, and Schwartz 2019; Ruhm 2019a; Venkataramani et al. 2019; Pierce and Schott 2020). Moreover, even if strong associations were obtained, there remains the vexing question of why the mortality trends have been so different for midlife whites and nonwhites.

[32] Many other noneconomic factors could potentially also influence these trends. For instance, economists have written about the importance of the pill (Goldin and Katz 2002), changes in the technology of home production (Stevenson and Wolfers 2007) and legalized abortion (Gruber, Levine, and Staiger 1999).

3h. Timing of Mortality Changes

A particular challenge for the arguments articulated by Case and Deaton is in reconciling the hypothesized causal factors with the observed timing of DSL mortality changes. To illustrate this, Figure 1 displays DSL and total mortality rates for 10-year age groups of prime-age adult whites from 1990-2018; 1990 is chosen as the starting year here since this is when Hispanic origin is first identified on death certificates. Rates are normalized to 100 in 1990, so that later entries indicate percent changes from that year.

For 45-54 year old whites, the group Case and Deaton primarily focus upon, DSL death rates remained roughly constant from 1990-1993, increased very slightly (by 0.3 percent per year) from 1993-1999, and then more rapidly (4.5 percent annually) from 1999-2017. The patterns differ somewhat at other ages. Mortality rates were essentially unchanged from 1990-1999 for 25-34 year olds and declined over the same period for 55-64 year olds, after which they rose exceptionally quickly for the younger group (6.9 percent per year from 1999-2017) and somewhat more slowly (4.4 percent annually) for the older one. DSL fatality rates grew throughout the period for 35-44 year olds, by 2.7 percent per year from 1990-1999 and 4.8 percent annually from 1999-2017. Death rates ticked downwards between 2017 and 2018 for all of these groups except 55-64 year olds.

The translation of changes in DSL fatalities to total death rates depended on what happened with other sources of death. The all-cause mortality rates of 25-34 year old whites fell substantially from 1995-1999, in large part due to reductions in homicide and HIV fatalities; the rapid growth from 2013-2017 reflected rising heroin and fentanyl deaths. By contrast, total mortality rates of 45-54 year olds dropped from 1990-1998, after which growth in DSL fatalities, particularly drug overdoses, more than offset small declines in other deaths. However, mortality rates for this group did not return to the higher 1990 levels during the analysis period. All-cause fatality rates changed little over time for 35-44 year olds, except for the heroin/fentanyl led growth after 2013, and they fell steadily for 55-64 year olds due to reductions cancer, cardiovascular and other disease sources of death.

Using somewhat different methods and a longer time period, the Social Capital Project (2019) shows that "deaths of despair" for white 45-54 year olds *declined* from the mid-1970s through early-1990s and then rose, slowly at first more rapidly after 1999.[33] Particularly interesting is their evidence that suicide rates for this group fell steadily from the 1960s through 1999, as did alcohol-related death rates from the mid-1970s through 2000. Conversely, they uncover a consistent growth in drug fatality rates since at least the late 1950s, when their analysis begins, although the increases were modest before the 1990s. Similarly, Masters, Tilstra, and Simon's (2018) examination of white 25-54 year olds

[33] The Social Capital Project uses a broader set of alcohol-related causes than just chronic liver disease.

indicates that drug deaths were flat for most groups from 1980 through the early to mid-1990s and grew rapidly starting in the late 1990s, while suicides fell from 1980 through around 2000 and then rose slowly for most groups; alcohol-related deaths also dropped through at least 2000 and with little evidence of substantial increases thereafter.[34]

In combination, these results suggest that the DSL mortality rates of midlife whites did not begin to increase substantially until at least the late 1990s, with some evidence of modestly rising drug fatality rates starting earlier, but with declines in suicide and liver disease death rates observed through the end of the 20th century.

It is difficult to reconcile these patterns with the narrative in *DEATHS*. The story Case and Deaton tell is one where adverse social and economic changes lead to despair and, ultimately, to increases in DSL mortality rates for less educated midlife whites. But what kind of lag structure is needed for this to make sense? The transformations emphasized all began much earlier. Real median wages of less educated workers declined from 1979-1999 and have been fairly constant since (Donovan and Bradley 2019). The fraction of unionized wage and salary workers has fallen steadily since at least 1973, with most of the reduction occurring prior to the mid-1990s (Hirsch and Macpherson 2003, updated annually).[35] Rates of divorce and permanent separations began to rise for women without college degrees starting in the early 1960's if not before (Cherlin 2010), and the proportion of children living with less educated single mothers rose substantially between 1980 and 2010.[36] Church membership has been falling since the mid-1940s, although most of this reduction has occurred during the last two decades (Jones 2019). More generally, Putnum (2000) documents declines in social capital beginning in the 1960s or 1970s, along multiple dimensions including political, civic and religious participation; workplace and informal networks; mutual trust; and altruism.

These trends indicate that most of the major social forces hypothesized to explain rising DSL mortality substantially predated the observed increases, usually by several decades. It might be possible to construct a dynamic model consistent with these results. For instance,

[34] One exception is that the drug death rates of 35-44 year old white men increased throughout the analysis period, which started in 1980. A weakness of both studies for understanding the mortality trends of non-Hispanic whites is that the Social Capital Project includes all whites (whether Hispanic or non-Hispanic) for years prior to 1999 and Masters et al. do so for all years analyzed (1980-2014). Jalal et al. (2018) present evidence of exponentially increasing drug death rates throughout the 1979-2016 period but do not separately analyze results for race/ethnicity or age subgroups.

[35] For instance, the percentage of unionized wage and salary (private sector nonagricultural) workers was 24.0, 14.9 and 10.5 (24.6, 10.4 and 6.4) percent in 1973, 1995 and 2018.

[36] Conversely, the proportion of children living with single mothers with college degrees remained essentially constant over this period.

the outcome might be a latent stock variable, ranging from despair at one extreme to optimism and high life satisfaction on the other, that is influenced by positive or negative flows of social and economic capital. A death from despair would then occur when this latent variable falls below some threshold. As the negative effects of American capitalism on less educated whites accumulated, starting in the 1970s, values of this stock variable declined and, beginning around the late 1990s, increasingly fell below the critical threshold.

However, while such an explanation is theoretically conceivable, there are reasons to doubt its validity. Even when the full effects of health shocks (i.e. the time it takes to move from the old to new equilibrium level of the latent variable) occur with a delay, the period is likely to be measured in years, not decades. Moreover, assuming an initial distribution of values for the latent capital stock variable, negative flows would be expected to fairly quickly lead to increases in deaths, even if the full effect occurs with a considerable lag. Consider the example of alcohol taxes and cirrhosis deaths. Although mortality from alcoholic cirrhosis results from a lifetime of heavy drinking, Cook and Tauchen (1982) formulate a model similar to the one just described and empirically demonstrate immediate reductions in cirrhosis deaths when alcohol taxes are raised, although it takes some years for the full effects to be manifested. Conversely, we observe little change or even falling DSL death rates through at least the mid-1990s, and even longer-lasting declines in suicides and chronic liver disease deaths, despite the decades earlier occurrence of the preconditions hypothesized to generate their rise.

Probably the most important reason for being skeptical about the Case and Deaton's conceptual framework is that there is a much simpler explanation for why DSL deaths began to rise around the mid-to-late 1990s, rather than before. We have established that drug fatalities were by far the most important component of the increase and it is widely understood that the drug environment changed sharply beginning around 1996, with the approval and aggressive marketing of OxyContin (Quinones 2016; Alpert et al. 2019). The deteriorating social and economic position of less educated Americans likely put them at higher risk when cheap potent opioids, often accompanied by other pharmaceuticals, became widely available and vigorously promoted but it seems improbable that they would have experienced anywhere near the enormous growth in fatality rates without this dramatic change in the drug environment.

Thus, rising overdose deaths probably primarily reflected a supply-driven phenomenon, with demand-side factors influencing who was most affected. Case and Deaton's view is that opioids fueled the fire of existing despair. An alternative framing is that the proliferation of opioids and other dangerous drugs killed a large number of persons who were at greatest risk for a variety of possible reasons, only one of which was despair. A key implication is

that most of these individuals would have remained alive had it not been for the supply-side forces creating the fatal drug epidemic.

Leaving drug deaths aside, it would be useful for future research to examine whether an appropriately constructed dynamic model, with a reasonable lag specification, could explain observed changes in suicide and chronic liver disease fatalities. Such a model would need to incorporate other relevant factors, such as the decline in liquor tax rates which may have been responsible for much of the (relatively small) recent increases liver disease deaths.[37] Similarly, some of the growth in suicides may be due to factors such as the wider availability of guns (M. Miller and Hemenway 2008).

However, a serious challenge to any effort portraying despair as a key determinant of these mortality rates is to explain why these death rates were *declining* over the last two decades of the 20th century, when the mechanisms hypothesized to be leading to increased misery were already well underway.

4. Concluding Thoughts

In this age of smart phone driven lack of concentration, it is increasingly difficult to take the time and energy needed to read entire books. However, making the investment to complete *DEATHS* will be well worth the effort for many. The volume is well written, interesting, crackling with insights, and flows well. It covers a great deal of ground and mostly does so seamlessly. The authors are two of the most prominent economists and their point-of-view is well worth being exposed to, whether or not you ultimately agree with their conclusions. They deserve enormous credit for taking on extremely difficult questions and approaching them from many angles. Those who have heard about "deaths of despair" but do not know what the term means will finish the volume with a much clearer understanding. Potential readers curious to learn about any of several topics – trends in mortality rates and life expectancy, the difficulties faced by the middle class and less educated, or the increasing tilt of American capitalism towards protecting the wealthy while leaving those less well-off to struggle – will find much of interest in *DEATHS*.

In short, *DEATHS* offers an attractive package for a wide variety of readers, particularly those who want to learn about the three interlocking stories presented – the current status

[37] Between 1991 and 2015, inflation-adjusted average state excise tax rates on beer, distilled spirits, and wine fell by 30, 32 and 27 percent (Naimi et al. 2018). Federal excise taxes on alcohol remained constant in nominal terms, and so declined by 42 percent in real terms, over the same period (Alcohol and Tobacco Tax and Trade Bureau 2020).

of American capitalism, the hardships experienced by less educated Americans, and the declining life expectancy of midlife whites – in an easily digestible form.

Economists, and other social scientists; however, should also read *DEATHS* with a somewhat skeptical view, and for those working in this area, with an eye towards important research questions. As discussed extensively above, the connections between the three hypotheses underlying the volume are not yet fully fleshed out and alternative explanations may be equally or more plausible.

In particular, the hypothesis that despair is the driving factor for the highlighted mortality trends is not fully convincing, particularly considering that these added fatalities largely reflect a drug crisis that has fairly well understood origins and consequences. There could also be some degree of reverse causation, whereby extreme distress is a *result* of individuals unwittingly becoming addicted to powerful prescribed or illicit opioids. A major challenge to Case and Deaton's story is to explain why it is largely white Americans who have been so adversely affected, even though the conditions for nonwhites have often been far worse and longer lasting. Indeed, the mortality rates of African-Americans have fallen substantially since the 1990s, just when death rates began rising for whites. Nor is it obvious how despair can explain the *falling* DSL mortality rates of midlife whites through at least the late 1990s, even though the negative transformations in the American economy and society described in *DEATHS* began decades earlier.

Case and Deaton provide convincing evidence of declining economic and social capital in the last quarter of the 20th century, particularly among some less educated groups. This probably exacerbated a crisis that was triggered by massive increases in the availability of opioids and other dangerous drugs starting in the late 1990s. Whatever their cause, an important aim of public health policies is to reduce the frequency with which individuals engage in risky behaviors and the negative consequences of doing so. In this regard, Currie and Schwandt (2020), among others, have pointed out that while it is undoubtedly desirable to improve underlying social and economic conditions, targeted interventions are likely to more quickly and successfully reduce drug deaths. Conversely, an almost exclusive focus on the need for broad systemic changes to a broken economic system, valuable as those might be, probably diverts attention from the key cause of a current major health crisis.

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Table 1. Actual and Counterfactual Mortality Rates, 45-54 year old Non-Hispanic Whites

	Actual/Estimated Mortality Rate	Difference vs. 1999 Mortality Rate
Actual Mortality Rate		
1999	384.1	
2018	412.0	27.9
Counterfactual 2018 Rate Basis		
2% annual reduction	261.7	150.4
1990-1999 trend continues	307.8	104.2
1990-1995 trend continues	340.8	71.4

Note: Table shows actual or counterfactual mortality rates for 45-54 year old non-Hispanic whites, and the difference between those rates and the actual 1999 rate. All mortality rates are per 100,000. The first counterfactual scenario assumes an annual 2.00 percent reduction starting in 1999. The second assumes a 1.16 percent annual decrease, which is the average yearly decline from 1990-1999. The final scenario assumes a 0.80 percent annual decline, beginning in 1995, which is the trend reduction observed from 1990-1995.

Table 2: Mortality Rates and Changes From Various Causes,
25-54 Year Old Non-Hispanic Whites

	All Causes	Drug, Suicide & Chronic Liver Disease (DSL) Deaths					Respira-tory
		All DSL	Drug, Non-Suicide	Drug Suicide	Other Suicide	Liver	
Males							
Mort. Rate: 1999	284.8	49.0	12.4	2.2	22.7	11.7	8.6
Mort. Rate: 2018	319.8	109.7	59.5	2.3	34.8	13.1	10.3
Δ: 2018 vs. 1999	34.9	60.8	47.1	0.1	12.1	1.5	1.7
% of DSL Δ			77.5%	0.1%	19.9%	2.4%	
Females							
Mort. Rate: 1999	159.4	16.2	5.0	2.4	4.7	4.2	7.6
Mort. Rate: 2018	188.9	48.5	28.6	3.0	8.7	8.3	10.4
Δ: 2018 vs. 1999	29.5	32.3	23.6	0.6	4.0	4.1	2.8
% of DSL Δ			73.0%	1.9%	12.5%	12.6%	

Note: Table shows mortality rates per 100,000 (Mort. Rate) in 1999 and 2018, as well as changes in these rates over these two years, and the percentage of the total change in mortality rates from drug, suicide and chronic liver disease (DSL) accounted for by specific cause. ICD-10 underlying cause-of-death codes for non-intentional drug deaths are: X40-44, X85, Y10-Y14. Those for intentional drug deaths, other suicides, liver disease and respiratory disease are: X60-X64; *U03, X65-X84, Y87.0; K70, K73, K74; and J00-J98). Mortality rates are age-adjusted using 2018-year population weights for five-year age groups.

Table 3: Mortality Rates and Changes in Mortality Rates from Various Causes for 25-54 Year Old Non-Hispanic Whites, by Age and Sex

	All Deaths	Drug, Suicide & Chronic Liver Disease (DSL) Deaths					Respiratory
		All	Drug, Non-Suicide	Drug Suicide	Other Suicide	Liver	
Males: 25-34							
MR Δ: 2018 vs. 1999	60.2	66.5	55.4	0.2	9.5	1.4	0.1
% of DSL Δ			83.3%	0.3%	14.2%	2.2%	
Males: 35-44							
MR Δ: 2018 vs. 1999	33.4	61.3	49.0	-0.5	11.9	0.9	0.7
% of DSL Δ			80.1%	-0.8%	19.4%	1.4%	
Males: 45-54							
MR Δ: 2018 vs. 1999	11.2	54.6	37.0	0.5	14.9	2.1	4.2
% of DSL Δ			67.9%	0.9%	27.3%	3.8%	
Females: 25-34							
MR Δ: 2018 vs. 1999	30.5	29.2	24.2	0.0	3.5	1.4	0.1
% of DSL Δ			83.0%	0.0%	12.1%	4.9%	
Females: 35-44							
MR Δ: 2018 vs. 1999	27.2	30.7	24.2	0.5	3.5	2.5	1.1
% of DSL Δ			78.9%	1.5%	11.5%	8.1%	
Females: 45-54							
MR Δ: 2018 vs. 1999	30.7	36.8	22.4	1.4	4.9	8.0	6.8
% of DSL Δ			61.0%	3.7%	13.4%	21.9%	

Note: see note on Table 2. "MR Δ: 2018 vs. 1999" indicates to the change in age-adjusted mortality rates, per 100,000, occurring between 1999 and 2018.

Figure 1: Mortality Rates for 25-64 Year Old Non-Hispanic Whites, Relative to 1990

Fig 1A: DSL Mortality Rates

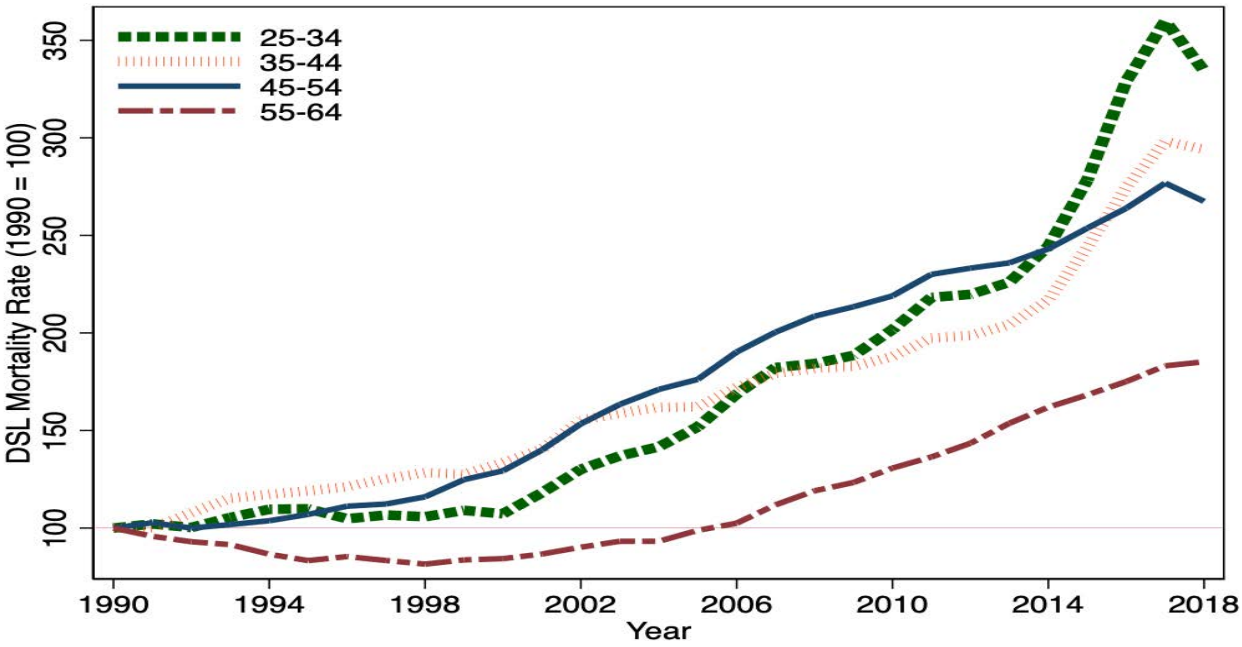
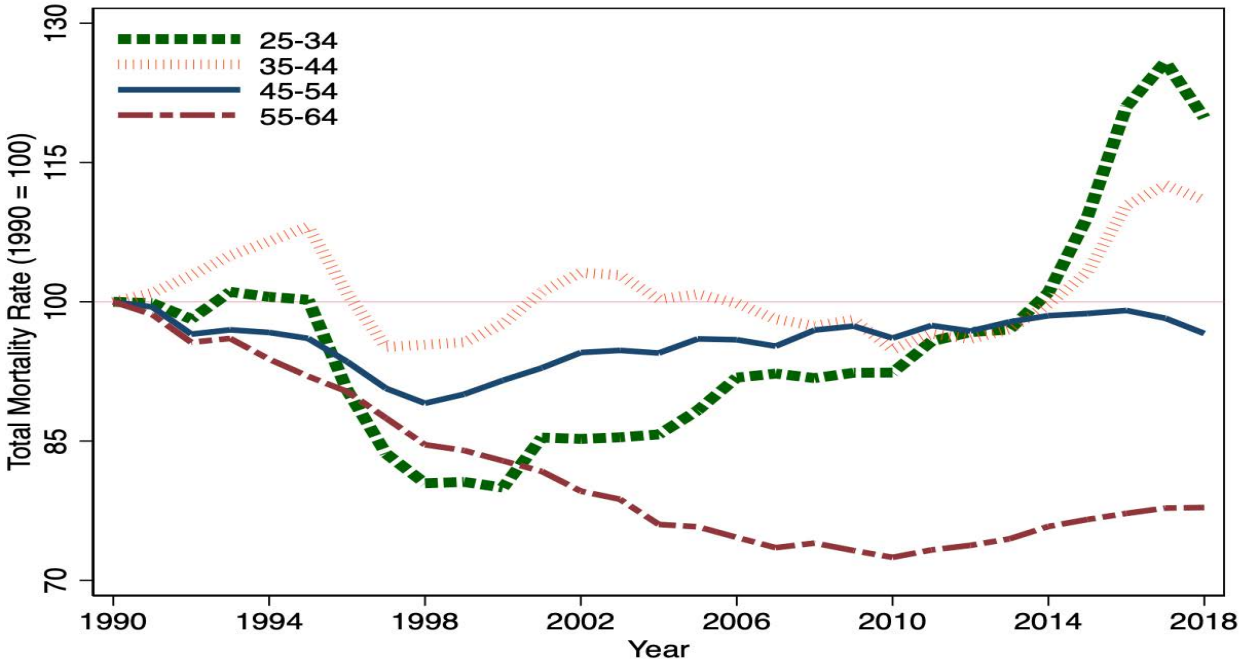


Fig. 1B: Total Mortality Rates



Note: Figure shows mortality rates as a percentage of 1999 value for the specified age group. DSL mortality rates include ICD-9 (1990-1998) underlying cause of death codes 571, 850-859, 950-959, 962, 980.0-980.5 and ICD-10 (1999-2018) codes K70, K73-K74, X40-X44, X60-X85, Y10-Y14, Y87.0, *U03.