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Article

Growing sense of social status threat and concomitant deaths of despair among whites

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ABSTRACT

Background: A startling population health phenomenon has been unfolding since the turn of the 21st century. Whites in the United States, who customarily have the most favorable mortality profile of all racial groups, have experienced rising mortality rates, without a commensurate rise in other racial groups. The two leading hypotheses to date are that either contemporaneous economic conditions or longer-term (post-1970s) economic transformations have led to declining economic and social prospects of low-educated whites, culminating in "deaths of despair." We re-examine these hypotheses and investigate a third hypothesis: mortality increases are attributable to (false) perceptions of whites that they are losing social status.

Methods: Using administrative and survey data, we examined trends and correlations between race-, age- and, education-specific mortality and a range of economic and social indicators. We also conducted a county-level fixed effects model to determine whether changes in the Republican share of voters during presidential elections, as a marker of growing perceptions of social status threat, was associated with changes in working-age white mortality from 2000 to 2016, adjusting for demographic and economic covariates.

Findings: Rising white mortality is not restricted to the lowest education bracket and is occurring deeper into the educational distribution. Neither short-term nor long-term economic factors can themselves account for rising white mortality, because parallel trends (and more adverse levels) of these factors were being experienced by blacks, whose mortality rates are *not* rising. Instead, perceptions – misperceptions – of whites that their social status is being threatened by their declining economic circumstances seems best able to reconcile the observed population health patterns.

Conclusion: Rising white mortality in the United States is not explained by traditional social and economic population health indicators, but instead by a *perceived* decline in relative group status on the part of whites – despite no actual loss in relative group position.

1. Introduction

Recent research has put a spotlight on a major public health phenomenon that has been unfolding for nearly two decades. While all of the other high-resource countries of Europe and North America have experienced a longstanding, continual decline in mortality rates over time, it appears that there has been a rise in working-age mortality rates since 1999 for only one subgroup in the United States, non-Hispanic whites (Case & Deaton, 2015). Indeed, mortality rates rarely rise, unless a society is subject to a major disaster, such as an infectious disease epidemic, a major economic crisis, or a war.

But, the absence of a clearly identifiable phenomenon that fits a

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timeline which could produce a rise in white mortality, not to mention a shock that simultaneously addresses why other social groups in the same country did not experience a similar change in their mortality trajectories (not even blacks, whose absolute mortality rates still are far higher), has rightfully commanded scholarly and popular attention across a range of disciplines (Case & Deaton, 2015; Gelman & Auerbach, 2016; Goodwin, Kuo, Brown, Juurlink, & Raji, 2018; Schmid, 2016). Phenomena that have been largely ruled out due to their lack of fit with timeline and other aspects of mortality trends include the AIDS epidemic of the 1980s and 1990s, recessionary periods (including the Great Recession of 2008) and the impact of wars being waged abroad on U.S. veterans' health (Case & Deaton, 2017).

In this paper, we move the discussion forward. In Part 2, we reconsider how trends in white mortality have been characterized thus far. In Parts 3 and 4, we enter the debate on the two main hypotheses previously forwarded to explain these trends (Case & Deaton, 2017). Finally, in Part 5 we explore and empirically test a new hypothesis, which we suggest holds far greater explanatory power. In so doing, we not only present the most comprehensive account to date of an important population health phenomenon, but also challenge well-established approaches to understanding population health inequalities.

2. Characterizing rising white mortality

2.1. Overview of white mortality

While previous literature documented falling life expectancy among subgroups of whites in the United States (Arias, 2016; Case & Deaton, 2017; Xu, Wang, Collins, & Tang, 2007), the most visible paper on the declining health of whites is an article by Case and Deaton, published in Proceedings of the National Academy of Sciences (PNAS; Case & Deaton, 2015). This study reported that after a long-term, steady two-percent annual reduction in mortality rates in the United States and other comparably longstanding high-income, capitalist societies, there was a "… cessation and reversal …" of this trend in 1999 for U.S. non-Hispanic whites of working age, resulting in an increase in their mortality of half a percent per year, thereafter (Case & Deaton, 2015). Fig. 1 provides our replication of age-adjusted, all-cause mortality rates by race in the United States for working-age adults between ages 25 to 54,

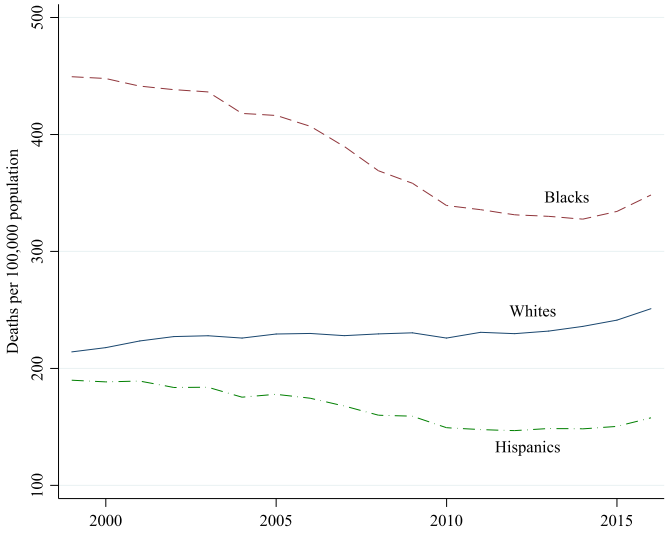


Fig. 1. All-cause age-adjusted mortality rate by race,ages25-54.
[a]Data from Multiple Cause of Death Data, CDC WONDER online database ("Centers for Disease Control and Prevention, National Center for Health Statistics. Multiple Cause of Death 1999–2016 on CDC WONDER Online Database, released December 2017. Data are from the Multiple Cause of Death Files, 1999–2016, as compiled from data provided by the 57 vital statistics jurisdictions through the Vital Statistics Cooperative Program.," 2017).

the age range of persons that appear to be most affected.

In an accompanying PNAS commentary, a separate team challenged the magnitude of the rise in mortality by demonstrating that rates were somewhat attenuated – but not eliminated – by adjusting for changes over time in the population age distribution (Gelman & Auerbach, 2016). Views have diverged on the value of age-adjustment for evaluating the rise of mortality rates, but nonetheless, the broad consensus across scientists is that the reversal of the customary decline in mortality among working-age whites, however large or small in magnitude, is a striking and unanticipated anomaly (Case & Deaton, 2016, 2017; Gelman & Auerbach, 2016).

While rising working-age white mortality is now widely acknowledged, there has been some consternation about the extent to which this phenomenon applies to whites as a whole, or whether it applies only to particular subgroups of working-age whites. Auerbach and Gelman (2017) proposed that the increase was limited to the southern and midwestern regions, but in subsequent analyses, Case and Deaton (2017) used a finer geographical disaggregation to demonstrate a more widespread pattern of increased mortality in most U.S. regions, stable trends in New England and the Pacific region, and a decline in mortality solely in the mid-Atlantic region.

Auerbach and Gelman (2017) also suggested that the mortality increase among working-age whites was limited to women. Case and Deaton (2017) found that although white women experienced a greater increase in mortality rates, these rates also increased among white men. For these reasons (and for the sake of parsimony), like Case and Deaton (2017), our starting premise is to examine as a singular phenomenon; the rise in national mortality rates of working-age white men and women.

Case and Deaton (2017) also argue that, though a rise in white mortality still can be discerned across all education groups, it is most pronounced among whites with a high school degree or less (Case & Deaton, 2015, 2017). In this subgroup, there is an increase in death rates for every five-year age group, with the exception of 60–64 year-olds – albeit the gradient of the rise differs somewhat across age groups. Thus, they define the issue as one that has emerged for whites in the lowest educational tier, and they focus their analysis on explaining why whites in this stratum have experienced a reversal in mortality fortunes (Case & Deaton, 2017).

We re-examined mortality rates across all education groups. Fig. 2 provides our calculations of all-cause mortality rates for non-Hispanic whites by five-year age groups and by education level (high school or less, some college, and college or more), and Table 1 provides average annual percentage change in mortality rate by race, education group, and age. From these two tables, we find evidence that it is perhaps too early to dismiss a pattern of rising mortality that can be observed among whites in higher education brackets.

Compared to the previous average annual two percent decline, those with a high school education or less experienced the sharpest increase in their mortality rates, which rose by 1.71 percent to 4.38 percent, depending on the age group (with higher rates of change in younger age groups). Moreover, every age group in this education category experienced an increase in mortality rates. However, whites with some college experienced a nearly equally sharp and consistent rise in mortality rates across all age groups, with rates spanning an increase of 0.50–4.92 percent. Among those in the highest education group, younger whites also experienced increased mortality rates. Those between ages 35 and 54 witnessed unchanged mortality rates, but it is unclear whether this can be viewed as a problematic function of deleterious exposures, or whether, because mortality rates were already quite low in this education-age bracket, this may be a "floor effect."

To summarize our interpretation of mortality trends, with the most conservative reading of the data, mortality rates rose for all except the most educated whites (who had stable rates), From 2000-2016, whites who fell outside the most educated group encompassed more than half (57–66 percent) of the working-age white population. Therefore, we

Fig. 2. All-cause mortality rate for non-Hispanic Whites by age-group and level.
[a]Data from CPS March ("Center for Economic and Policy Research. 2016. March CPS Uniform Extracts, Version 1.0. Washington, DC," n.d.) and Mortality Multiple Cause-of-Death Public Use Record from NVSS ("National Center for Health Statistics. Compressed Mortality File, 1999–2016 as compiled from data provided by the 57 vital statistics jurisdictions through the Vital Statistics Cooperative Program. Hyattsville, Maryland. 2017.," 2017).
[b]GA, OK, RI, and SD are excluded due to absence of education on death certificates until 2003. Post 2004 includes the four states even though education is still missing because states are unidentifiable in the public-use Vital Statistics data from 2004.

believe it is too early to dismiss rising white mortality as an issue which applies to whites more broadly, rather than solely to the most uneducated whites (Case & Deaton, 2015, 2017).

Moreover, while mortality across the entirety of the working-age group of whites has been acknowledged, most of the analytic emphasis has been on those between 50-54 years of age (Case & Deaton, 2017). The primary rationale is effectively premised on robustness. Mortality rates of younger age groups may be more volatile and influenced by factors like the AIDS epidemic (Case & Deaton, 2017). While this is true, once again, we feel that without more direct evidence, it is too early to conclude that the rise in mortality across the entirety of working age whites is not a collective phenomenon, particularly given the explanations offered by Case and Deaton (2017), and by others more recently, to which we will soon turn our attention. Still, we agree that mortality increases do not seem to be evident among those older than 54 of years of age. Therefore, we focus our analyses on whites between the ages of 25–54 years.

In sum, we concur with previous work that, since 1999, there has been a rise in working-age, white mortality rates that is generalizable across both men and women (Case & Deaton, 2017). Where we

Table 1
Average annual % change in all-cause mortality rate from 1999-2016, by race, age-group and education.

	White			Black			Hispanic		
	HS or less	Some college	BA or more	HS or less	Some college	BA or more	HS or less	Some college	BA or more
25-29	4.380	4.915	0.239	1.025	2.277	−1.974	0.671	2.594	−0.897
30-34	4.320	4.568	0.784	0.962	1.886	−1.143	0.079	3.505	0.597
35-39	3.775	3.339	−0.140	0.761	0.661	−0.996	−0.752	0.893	−2.020
40-44	2.749	2.158	−1.083	−0.589	−0.047	−2.771	−1.855	1.289	−3.719
45-49	1.724	1.267	−1.947	−1.172	−1.434	−2.382	−1.665	−0.602	−2.160
50-54	1.706	0.496	−2.240	−1.773	−0.348	−3.628	−0.710	−0.041	−0.612

[a]Data from CPS March("Center for Economic and Policy Research. 2016. March CPS Uniform Extracts, Version 1.0. Washington, DC," n.d.) and Mortality Multiple Cause-of-Death Public Use Record from NVSS("National Center for Health Statistics. Compressed Mortality File, 1999–2016 as compiled from data provided by the 57 vital statistics jurisdictions through the Vital Statistics Cooperative Program. Hyattsville, Maryland. 2017.," 2017).

principally diverge is in viewing this issue as (a) one that is isolated to whites with a high school degree and (b) one that is principally relevant for the 50–54 year age band within that educational stratum. Instead, our assessment of mortality patterns suggests a more widespread phenomenon of rising death rates among whites between 25 and 54 years of age, across all education groups. The pattern follows a gradient of sorts: the worst outcomes are concentrated in the lowest education group; the middle education group has slightly less damaging trends; and the least damaging trends are found the highest education group.

2.2. Deaths of despair and indications of a broader health phenomenon

Case and Deaton (2015, 2017) point out that at the same time during which whites have experienced a reversal in mortality trends, the causes of deaths that have increased have been those due to alcohol consumption, poisonings (which they attribute primarily to opioid use and overdose), and suicides (Case & Deaton, 2015, 2017). Because of the link of such outcomes to the experience of severe psychological strain, Case and Deaton (2015, 2017) have dubbed them "deaths of despair" (Case & Deaton, 2015).

Moreover, it seems that whites also are experiencing growing levels of morbidity. Case and Deaton point to a rise in chronic pain, mental distress, difficulties with activities of daily living, and self-rated health (Case & Deaton, 2015). Our own calculations, based on National Health Interview Survey Data, suggest that the risk factors for major causes of morbidity have also either risen substantially (hypertension and obesity), or slightly (smoking) in whites irrespective of educational level, though to a far greater extent, among whites with a high school degree or less (Fig. 3).

There are two main explanations as to why whites seem to be experiencing a more widespread decline in health status and morbidity, which includes but is not limited to a rise of substance use- and suicide-related mortality. The first interpretation is that because despair is more specifically connected to substance use and suicide, the increasing mortality and widespread health status decline are likely not a function of despair, but of something else that can account for worsening of these additional outcomes. The second common explanation is that the widespread decline in health status is actually consistent with, and may even reinforce, a despair-based explanation for worsening white health.

As we explain below, we favor the latter explanation. Our orientation is based on the work of Bruce McEwen and others, who have illuminated the mechanisms through which psychological stress actually may account for much of the disease and death present in more affluent countries today (McEwen, 2006; McEwen & Stellar, 1993; Sapolsky, 1990).

The mechanisms can be described as follows. The human brain has a set of neuro-endocrinological systems, which are designed to respond to the experience of psychological stress (McEwen, 2006). Allostasis is the term given to the functioning of these systems in ways that allow the brain (and body) to perceive the stress, react to the stress, and return to its pre-stress resting state (McEwen, 2006). The body is stimulated by a stress-inducing exposure and reacts in a range of ways, including elevated heart rate and blood pressure, the release of hormones such as cortisol, and other associated physiological reactions, which subsequently return to their resting levels (McEwen, 2006).

The regulation of this system – its function and its return to resting state – is predicated on an infrequent experience of stress, however big or small the stressor may be. In evolutionary terms, the system was designed to deal with situations that evoked the feeling of sudden and immediate danger (which might be experienced when encountering a large, wild animal; McEwen, 2006).

However, the reality of modern-day life is that experiences of stress are no longer infrequent (Jackson, 2014). And, even though these experiences usually do not involve sudden and immediate danger, the chronic, all-day everyday nature of low-grade stress can be more taxing over the long term to our minds and bodies than the occasional

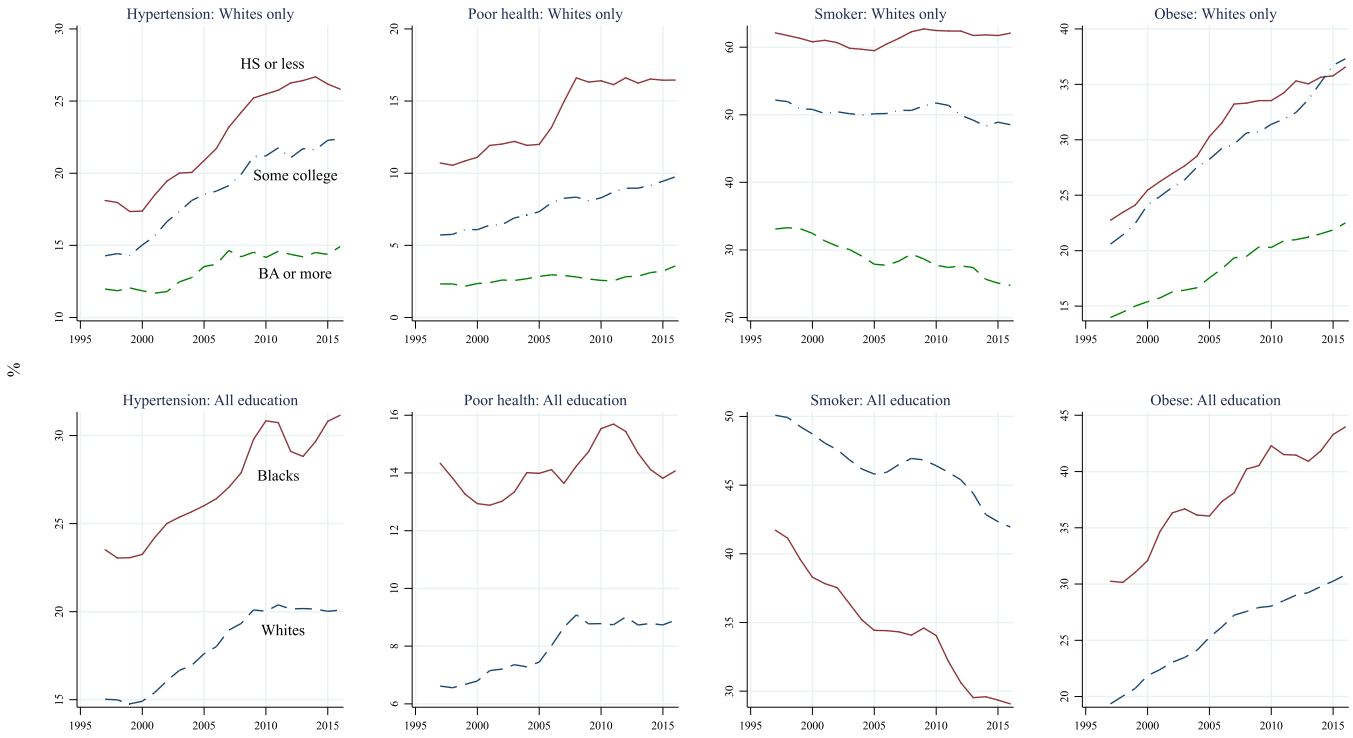


Fig. 3. Prevalence of selected risk factors by education level and race,ages25-54.
[a]Data from National Health Interview Survey (Sample Adult sample) taken from IPUMS Health Surveys (Blewett, Drew, Griffin, King, & Williams, n.d.).
[b]Weighted by sample adult weights and smoothed with 3-year averages.

confrontation with a lion or a tiger (McEwen & Stellar, 1993). This is because when stresses are chronic, the body does not have an opportunity to return to its resting state (McEwen, 2006).

This results in dysregulation of the system and leads to, for example, chronically elevated blood pressure (clinically known as hypertension), higher blood cholesterol levels, and the need for additional coping strategies such as use of comforting substances (from food to cigarettes to illicit drugs) and sedentarism, which amount to unhealthy forms of self-medication (Dallman et al., 2003; McEwen, 2006). So, on the one hand, these strategies offer a means for managing stress; but on the other, they also create risk for many of the major sources of morbidity and mortality observed today – from mental health issues to cardiovascular diseases.

In sum, our knowledge of the drivers of health supports the notion that declining white health status, including but not limited to rising mortality and morbidity, may be a reflection of despair. Moreover, the broad range of health indicators that are worsening actually strengthens, rather than detracts from the notion of psychological strain – despair – as a common underlying cause.

2.3. What might Be causing white despair: clues from patterns of black health

How to perceive, let alone search for causes of declining white health status is further complicated when we juxtapose the health status of whites with that of the health status of blacks. In absolute terms, blacks have far worse health status than whites, including higher mortality rates, higher levels of morbidity (chronic conditions), and associated risk factors, with the exceptions of smoking and alcohol consumption, which are higher among whites (see also Fig. 3; Levine et al., 2001; Ramraj et al., 2016). With tongue firmly planted in cheek, Auerbach and Gelman have observed, "… but that's not news …" (Auerbach & Gelman, 2017).

What perhaps is more newsworthy, at least insofar as understandings of population health go, is the seemingly paradoxical phenomenon of rising mortality rates in the group with the far better (lowest) mortality rate, and declining mortality rates in the group with the far worse (highest) mortality rate. *It is this phenomenon that we believe deserves inquiry*.

Moreover, as Fig. 3 suggests, the parallel rise for blacks and whites in risk factors such as hypertension and obesity, further corroborates the evidence, which suggest that the sources of rising white mortality are less related to chronic conditions, and more related to substance use and suicide.

2.4. Explanations offered by the science of population health

Though the puzzle before us is complex, it very much fits a classic question in population health literature: why are some populations healthy and others not (Evans, Barer, & Marmor, 1994)? While any number of factors may account for an individual getting sick or staying healthy, the range of factors is more restricted when one attempts to understand why some populations exhibit poorer health than others. At the population level, neither chance nor genetic endowment explains variation across groups (populations) in the vast majority of health outcomes, including mortality (Evans et al., 1994). This is because the population groups across which health status varies are not genetically distinguishable, and because the odds that consistent health differences can be explained by chance are negligible.

Why then are some populations healthier than others? Fig. 4 provides a conceptual overview. It begins with differences in the prevalence of risk factors for ill health among the populations (Evans et al., 1994; Solar & Irwin, 2006). These risk factors include the burden of allostatic load, exposure to pollution, and engagement in risky coping behaviors (related to diet, sedentarism, and substance use) that satiate in the short run, but produce ill health in the long run (Evans et al., 1994; Solar & Irwin, 2006).

But the explanation does not stop there. It cannot. The next piece of the puzzle is to understand why some populations systematically differ in the prevalence of these risk factors. The answer from decades of population health science appears to be that the root cause of differences in the prevalence of risk factors is the absence of income, wealth, and other forms of socioeconomic resources and status – either in absolute terms, or relative to others in society (Evans et al., 1994; Link & Phelan, 1995; Marmot, 2005; Solar & Irwin, 2006).

Populations with a higher prevalence of risk factors leading to disease are likely to be those that hold lower levels of resources and status (Link & Phelan, 1995). Conversely, the literature suggests that the presence of socioeconomic resources and status provides considerable buffering against the activation of these risk factors (Link & Phelan, 1995). Because lack of socioeconomic resources and status operate in this way, to trigger so many disease risk factors, and therefore so many diseases, they are often termed the "fundamental" or "root causes" of population health (Link & Phelan, 1995).

But access to socioeconomic resources and status, as fundamental as they may be, are not bestowed upon some populations and not others via a random sorting process. As the social science literature and even a rudimentary knowledge of history have taught us, resources are distributed (and redistributed) across populations through the power of markets, policies, and politics, and through the personal interactions and structural institutions that create, represent, and embody them (Darity, 2008; Ehrenreich, 2011; Hamilton, Darity Jr, Price, Sridharan, & Tippett, 2015).

For instance, the income level of a given population is conditioned on the population's labor market position, on the extent to which the population has inter-generational wealth on which members can draw in times of need and, if the population is vulnerable on both of these terms, also on the degree to which social policies in society provide resources as a matter of citizenship or residency (thus removing reliance, at least for basic needs, on markets and on family and community; Hamilton, Darity, Price, Sridharan, & Tippett, 2015).

Accordingly, higher black mortality over the course of the wide sweep of U.S. history, as well as more general black-white health inequalities, have been attributed to systematic and deeply entrenched differences in economic resources and status between blacks and whites (Darity Jr., 2008; Hamilton et al., 2015; Krieger, 1999; Laveist, 1993; Siddiqi, Jones, Bruce, & Erwin, 2016; Siddiqi, Kawachi, Keating, & Hertzman, 2013; Williams & Mohammed, 2013). Slavery, the Jim Crow era, and the present-day black-white employment and wealth inequalities they perpetuated have confined and restricted the position of blacks in American economic life since its beginning (Darity Jr., 2008; Hamilton et al., 2015).

Lack of access to stable, well-paying jobs, and lack of access to the economic advantages provided to whites through policies – like those stipulated by the New Deal and in the G.I. Bill –left blacks with a small fraction of the wealth that whites are privy to (Gordon, 2005; Hamilton et al., 2015). Today, it is estimated that blacks hold less than one tenth of the wealth of whites (Urban Institute, 2015). This has disastrous implications for many outcomes, including health. The deeply embedded black health disadvantage is therefore not at all surprising, given the prominent role of resources and status for health outcomes.

While the population health literature is rife with theoretical and empirical studies that explain population health inequalities at a moment in time, there has been far less inquiry about the factors that cause changes in population health inequalities. However, given the major lessons from the existing literature about the role of socioeconomic status for health, when puzzling population health phenomena emerge, the strongest candidate explanations seem to rest on changes in socioeconomic resources, spurred by changes in societal conditions (Link & Phelan, 1995; Marmot, 2005; Solar & Irwin, 2006).

The increase in mortality in Russia during the early 1990s was explained by the sudden loss of economic security following the rapid

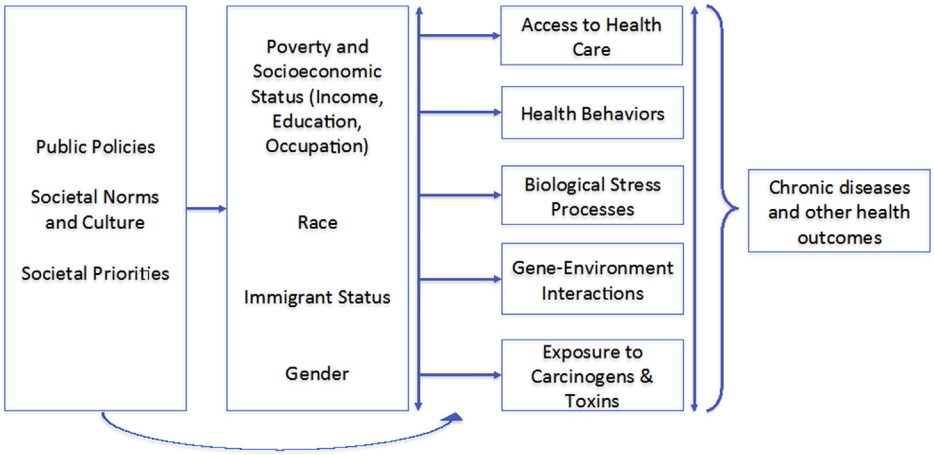


Fig. 4. WHO framework for social determinants of health
[a]Adapted from the World Health Organization framework on social determinants of health (http://www.who.int/sdhconference/resources/ConceptualframeworkforactiononSDH_eng.pdf).

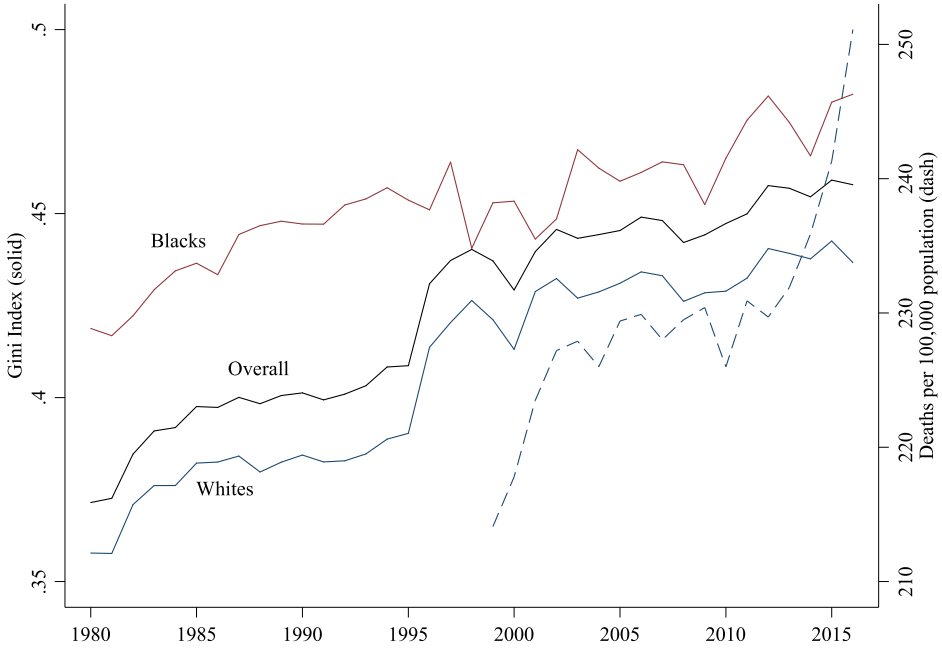


Fig. 5. House hold income gini index white mortality rate, ages25-54.
[a]Data from CPS March("Center for Economic and Policy Research. 2016. March CPS Uniform Extracts, Version 1.0. Washington, DC," n.d.) and Multiple Cause of Death Data, CDC WONDER online database("Centers for Disease Control and Prevention, National Center for Health Statistics. Multiple Cause of Death 1999–2016 on CDC WONDER Online Database, released December 2017. Data are from the Multiple Cause of Death Files, 1999–2016, as compiled from data provided by the 57 vital statistics jurisdictions through the Vital Statistics Cooperative Program.," 2017)
[b]Household survey weights are used for Gini calculation.
[c]Household income is household size adjusted with square root method.
[d]Hispanic origin is excluded for within race Gini calculation.

collapse of communism and associated rise in dependence on one's market position to procure even the basics of life (Hertzman, Siddiqi, & Bobak, 2002; Rose, 2000; Rose, Mishler, & Haerpfer, 1997). The increase in suicide rates in the United States following the Great Recession of 2008 have been attributed to the stress caused by its dramatic adverse impact on employment and earnings (Erin M. Sullivan, Joseph L. Annest, Feijun Luo, Thomas R. Simon, & Linda L. Dahlberg, 2013; Reeves et al., 2012).

Accordingly, as the post-1999 rise in white mortality has come to be observed as a population health phenomenon, the initial candidate explanations have been centered on changes in white socioeconomic circumstances. To date, there have been two major explanations of this type put forth (Case & Deaton, 2017). One focuses on contemporary changes in economic well-being. The other focuses on "slow-moving [economic and social] processes" that began decades prior (Pierson, 2003).

3. Hypothesis 1 – contemporaneous economic circumstances

3.1. Overview

The first major explanation that was forwarded to explain rising white mortality is the relatively contemporaneous change in economic circumstances, proxied by changes in median income.[38] In their analysis, Case and Deaton (2017) demonstrate a strong correlation between changes in median income and changes in mortality for middle-aged whites. However, they conclude that this is a spurious association (Case & Deaton, 2017).

Their first rationale for dismissing the role of median income is based on further parsing median income and mortality by education group (Case & Deaton, 2017). They suggest that mortality rates are increasing among whites with a high school diploma or less, and yet median income trends have displayed similar patterns of change (relative stagnation) across all education groups (Case & Deaton, 2017). More specifically, they suggest that the "… general widening of family incomes in the United States does not show up here in any divergence between the median incomes of those with different educational

qualifications …" (Case & Deaton, 2017).

As we discuss below, we share their belief that contemporary economic trends are not, in isolation, driving rising white mortality. However, we are not convinced that a lack of divergence in median income trends between education groups precludes recognition of a "… general widening of family incomes in the United States …" (Case & Deaton, 2017).

Using data from the Current Population Survey, we calculated the Gini coefficient, an overall index of the degree of income inequality, across households in the United States, with an adjustment for household size (Fig. 5). Our estimates clearly demonstrate that, indeed, by another metric that takes into account the broader income distribution, income inequality, in fact, has been growing, since 1980 both for the overall US population and intra-racially among whites and blacks, respectively. A more extensive analysis using far more sensitive measures of income echoes the finding of rising income inequality (Piketty, Saez, & Zucman, 2018). This is notably also the case for wealth inequality (Urban Institute, 2015).

Case and Deaton (2017) suggest that "… [income] inequality and mortality moved in opposite directions …" as proof that income inequality cannot be a factor in explaining rising white mortality. Because both factors are rising, it is not, on its face, implausible that rising income inequality could be associated with rising mortality. However, because both whites and blacks have experienced rising within-group income inequality (Fig. 5), but display different mortality trends, we believe that income inequality alone is an unlikely explanation for rising white mortality.

In addition, we independently analyzed trends in median income and mortality for all working-age whites and for each five-year age bracket (Fig. 6). There was an across-the-board decline or stagnation in the median incomes of working-age whites, and a rise in their mortality, though not as consistent a rise for all age categories as the 50-54-year age group. The widespread consistency of trends in median income gives particular heft to our thesis that whatever is happening to the health of whites transcends the 50-54-year age group.

The second rationale that Case and Deaton (2017) offer for dismissing the role of median income appears to us to be more defensible. They point out that, while median incomes of whites have declined or stagnated, this also has been the case for blacks (as well as for Hispanics; Case & Deaton, 2017). Moreover, as our own analyses show, in every age and education group, blacks have far lower absolute levels of median income (Fig. 7). Yet, it is only the mortality rate of whites that is rising. While contemporaneous economic factors are traditionally a front-line explanation for population health inequalities (Adler et al., 1994; Link & Phelan, 1995; Marmot, 2005), in this particular case it must be noted that blacks have had similar income declines. Therefore, the rise of white mortality without a parallel rise in black mortality is very unlikely to be explained, in isolation, by declining median income among whites.

This is also true for other pecuniary factors. Case and Deaton (2017) briefly take up the case of unemployment. They discuss the inconsistency between trends in white unemployment rates, which were falling during rising white mortality, although unemployment rates may not be the best metric of joblessness because of the phenomenon of "discouraged workers" (Benati, 2001; Case & Deaton, 2017; Estevão & Tsounta, 2011). The more pertinent issue is that, similar to median income, black unemployment trends are similar to those of whites, but whites' absolute levels of unemployment are far lower. Unemployment rates have declined dramatically for both blacks and whites since they peaked during the Great Recession, although the two to one black to white ratio remains persistent.

We also independently examined employment-population ratios (Fig. 8), to account for the discouraged worker phenomenon that is not embodied in unemployment rates (DeSilver, 2017). Overall, the employment-population ratio paints a less optimistic picture for both racial groups. Since the turn of the century, these ratios have been in decline for most age groups and across all educational strata. Of course, the black employment-population ratio is lower.

In sum, like Case and Deaton (2017), we believe that short-term economic circumstances cannot, on their own merit, explain rising white mortality. However, we endorse only one of Case and Deaton's (2017) rationales: that blacks experienced similar patterns of change in their economic conditions as whites, thus making differential mortality patterns by race difficult to reconcile.

4. Hypothesis 2 – longstanding economic circumstances

4.1. Overview

The other main hypothesis that has been put forwards is about the effects of longer-term changes in economic circumstances. Case and Deaton (2017) hypothesize that decades-long (post-1970s) changes in the American economy, marked by a declining manufacturing sector and a "… decline in the quality and quantity of opportunity in the labor market for those with no more than a high school degree …" play a "fundamental-cause role" in what they believe are the primary drivers of white mortality (Case & Deaton, 2017; Link & Phelan, 1995).

They suggest that the social effects of long-term economic decline, such as deterioration in stability of romantic relationships measured by declines in marriage rates and increases in serial partnering, may be responsible for increasing white mortality (Case & Deaton, 2017). As Cutler (2017) noted in a subsequent commentary (Available in: (available at: https://www.brookings.edu/wp-content/uploads/2017/03/6_casedeaton.pdf), n.d.), rising mortality is hypothesized to be the result of the "… breakdown of material and social circumstances …", through what Pierson (2003) might call "big, slow-moving processes" (Mahoney & Rueschemeyer, 2003). In short, the thesis is that white mortality reflects the dramatic worsening of economic and social prospects for whites with a high school education or less over the last fifty years (Case & Deaton, 2017).

However, this argument carries similar limitations as the contemporaneous economic circumstances argument we address in Part 3. The Economic trajectories for blacks are similar, and with worse absolute levels. Using data from the Current Population Survey, we calculated education levels back to 1980, when a much larger proportion of blacks than whites had only a high school degree or less (70 percent, compared to 54 percent; Fig. 9). In fact, by Case and Deaton's (2017) own speculation, the deterioration of the economic circumstances for blacks was likely worse than that of whites.

Moreover, as we have suggested, the rise in white mortality extends much further into the educational distribution and covers a broader age spectrum than the 50-54-year old age group on which this thesis largely rests. Whatever explanation is invoked must encompass a broad mortality phenomenon among whites, rather than one concentrated among one five-year age bracket of those with the least education. It also must constitute an attempted explanation for the increase in white death rates that either (a) did not occur in blacks, or, for some reason, (b) operated differently for blacks.

Case and Deaton (2017) suggest a couple of reasons. They invoke the notion of resilience, by suggesting that because blacks always have experienced "… labor market deprivations," that they may be more "… inured to the insults of the market." They also speculate that stronger norms of social support from family and community institutions (like the church) offer blacks a buffer against the stresses that economic disadvantage may create (Case & Deaton, 2017).

We examined the literature on resilience. There are three key findings we have identified, which suggest resilience may not be a strong candidate explanation at this time, though warrants future research.

In the first place, resilience is a notion that is inferred, rather than directly observed. Resiliency is thought to be in effect when outcomes are more favorable than expected, given a prior risk profile. However

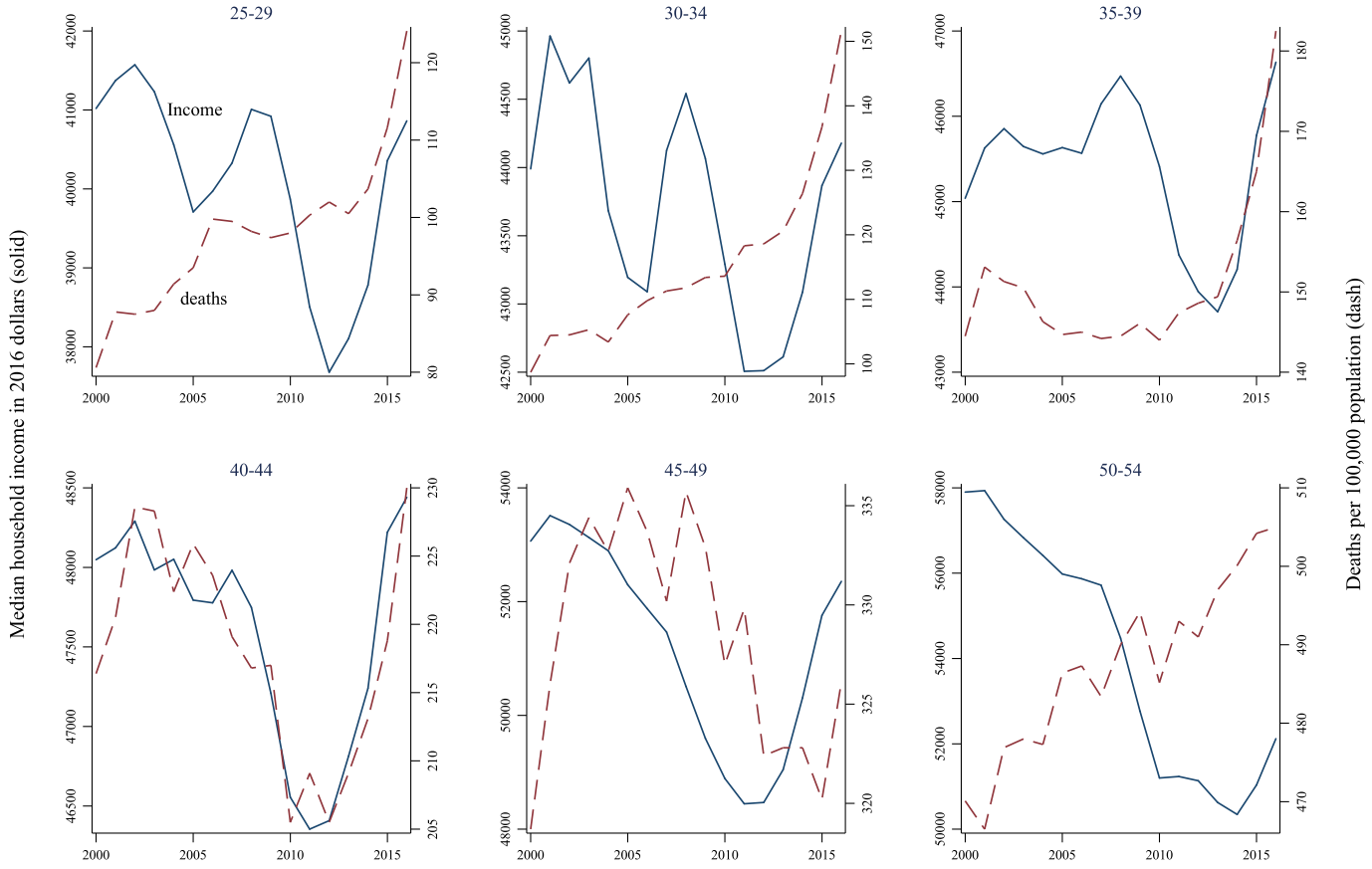


Fig. 6. Median household in come and all-cause mortality rate by age-group:Whites only.
[a]Data from CPS March("Center for Economic and Policy Research. 2016. March CPS Uniform Extracts, Version 1.0. Washington, DC," n.d.) and Multiple Cause of Death Data, CDC WONDER online database("Centers for Disease Control and Prevention, National Center for Health Statistics. Multiple Cause of Death 1999–2016 on CDC WONDER Online Database, released December 2017. Data are from the Multiple Cause of Death Files, 1999–2016, as compiled from data provided by the 57 vital statistics jurisdictions through the Vital Statistics Cooperative Program.," 2017)
[b]Household income is household size adjusted with square root method.
[c]Median income is calculated with household survey weights and is smoothed with 3-year averages.
[d]Race and age-group are identified from the household head and Hispanic origin is excluded.

there is no direct means for assessing whether more favorable outcomes are indeed the product of resilience, nor for ruling out other potential explanations for more favorable outcomes (Wright, Masten, & Narayan, 2013). To put a fine point on it, resilience is rarely more than a presumption.

Second, resilience is not produced solely by the process of inurement. Prior repeated exposure to a risk factor does not itself confer resilience against ill health. In relation to the present puzzle, blacks are unlikely to be inured, in biological and health terms, to economic stressors merely because of their longstanding exposure to economic stressors. In fact, higher allostatic load in blacks occurs precisely because of their repeated, longstanding exposure to these stressors (McEwen, 2006).

To be sure, black people have out-performed what might be expected, given the institutional disadvantages they have faced throughout history. From at least the inception of the country, managing daily life has demanded greater black intellect and fortitude to navigate a complex battlefield (Lee, Shen, & Tran, 2009). This might well be characterized as an indicator of "social resilience" (Hall & Lamont, 2013). But, social resilience imposes a biological toll (manifesting worse absolute levels of health status), rather than a biologically protective effect.

Finally, resilience is thought to occur in the presence of protective factors (McEwen, 2016; McEwen, Gray, & Nasca, 2015; Wright et al., 2013). The protective factors that Case and Deaton (2017) cite as potentially accounting for black resilience – social ties and community and institutional support – may be beneficial to blacks. However, since these ties and support have long been in place, it is difficult to reconcile why, then, blacks have higher levels of allostatic load, and worse absolute levels of health status (including mortality levels).

Another possibility is that these social ties make blacks more "resilient" to mortality and health outcomes that are mediated more strongly by acute psychological distress, which may present itself result in outcomes like substance use and suicide. At the same time, these social ties may make blacks less "resilient" to chronic stressors, which present themselves in outcomes like cardiovascular disease. However, we have little means of assessing this. It is also difficult to clearly discern which perceived stressors manifest in more acute psychological terms, rather than physiological terms, and how this differentiation might occur.

In sum, by our estimation, there is no more of a case to be made for long-term economic circumstances than there is for short-term economic circumstances. Since blacks experienced even worse long-term economic circumstances, but do not display similarly rising mortality rates, this account is difficult to endorse. It is also difficult to construct a scenario whereby resilience and buffering can, on their own, account

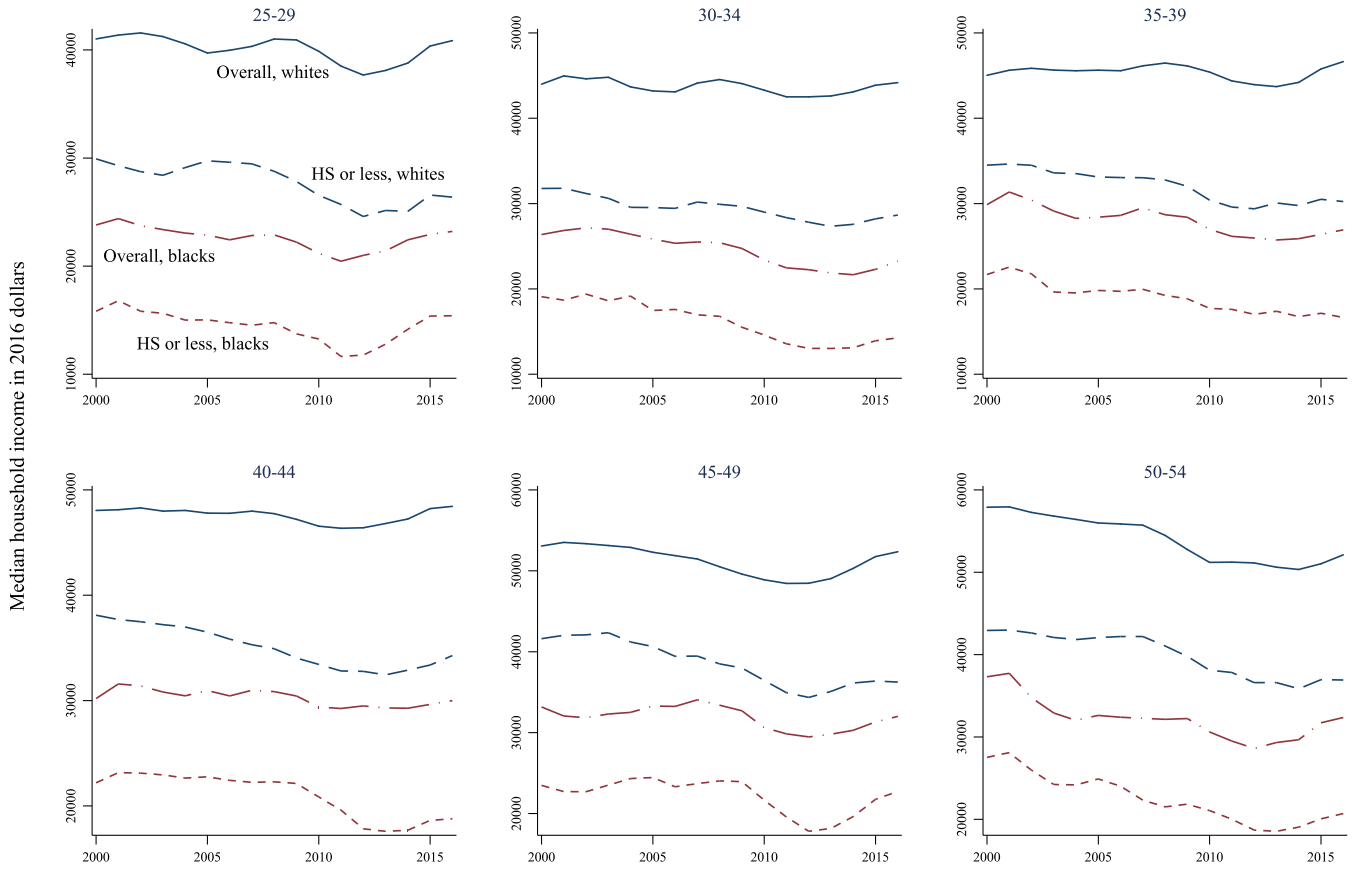


Fig. 7. Median household income by age-group and race: Overall and HS or less educated only.
[a]Data from CPS March ("Center for Economic and Policy Research. 2016. March CPS Uniform Extracts, Version 1.0. Washington, DC," n.d.).
[b]Household income is household size adjusted with square root method.
[c]Median income is calculated with household survey weights and is smoothed with 3-year averages.
[d]Race and age-group are identified from the household head and Hispanic origin is excluded.

for divergent responses to the same economic circumstances because resilience is not a product of inurement to long-term exposure to stressors, nor do protective supports seem to offer a strong candidate explanation.

5. Hypothesis 3 – status, status perceptions, and the role of status threat

5.1. Overview

At this point in the assessment, the following set of facts are clear. In the United States, black mortality rates are higher than white mortality rates, and have been so since the beginning of the nation's history (Levine et al., 2001). However, since 1999, the mortality fortunes of working-age whites have undergone a reversal, while black mortality rates (like mortality rates of all other groups and all high-income countries) continue to fall (Case & Deaton, 2017).

Traditional economic and social indicators of population health that are often found to be the main determinants of population health (Link & Phelan, 1995; Marmot, 2005; Solar & Irwin, 2006) do not explain this set of population health facts. While economic indicators, indeed, have been deteriorating – from declining median incomes, to greater precariousness in the economic (and social) lives of those with low levels of education – blacks, rather than whites, continue by far to face the most economic disadvantage. Moreover, the reversal of declining white mortality is not restricted to the most economically vulnerable groups. Indeed, the largest and most consistent rise is among whites with no college education, but even some of the most highly educated whites have experienced a rise in mortality.

A valid causal story must explain something that is occurring widely among whites and also explain why it is not occurring among blacks. We return to findings in population health regarding the centrality of socioeconomic resources and status for explaining differences in health status across population groups (Link & Phelan, 1995). If whites are not experiencing more actual socioeconomic disadvantages, and the phenomenon cuts across even socioeconomically advantaged whites, can economic differentials, which explain virtually all population health phenomena, explain rising white mortality?

We are deeply persuaded by a myriad of theoretical (Bobo, 1983; Craig, Rucker, & Richeson, 2017; Darity, Hamilton, & Stewart, 2015; Enos, 2014; Lieberson, 1968; Quillian, 1995) and empirical findings that status threat or, more accurately, perceived threat to the social status of whites, may be the main reason for rising white mortality. Thus far, this hypothesis has received only one brief mention (Case & Deaton, 2017). Perceived social status threat offers a compelling, albeit jarring, explanation for rising white mortality because: (a) it explains why white mortality rates are rising, while black death rates are not; (b) it reconciles the effect on white mortality across educational categories, even if the effect is more muted at higher levels of education; (c) it is consistent – if in an unusual way – with decades of population health theory, which has demonstrated the significance of socioeconomic status as a root cause of health outcomes; and (d) it provides a

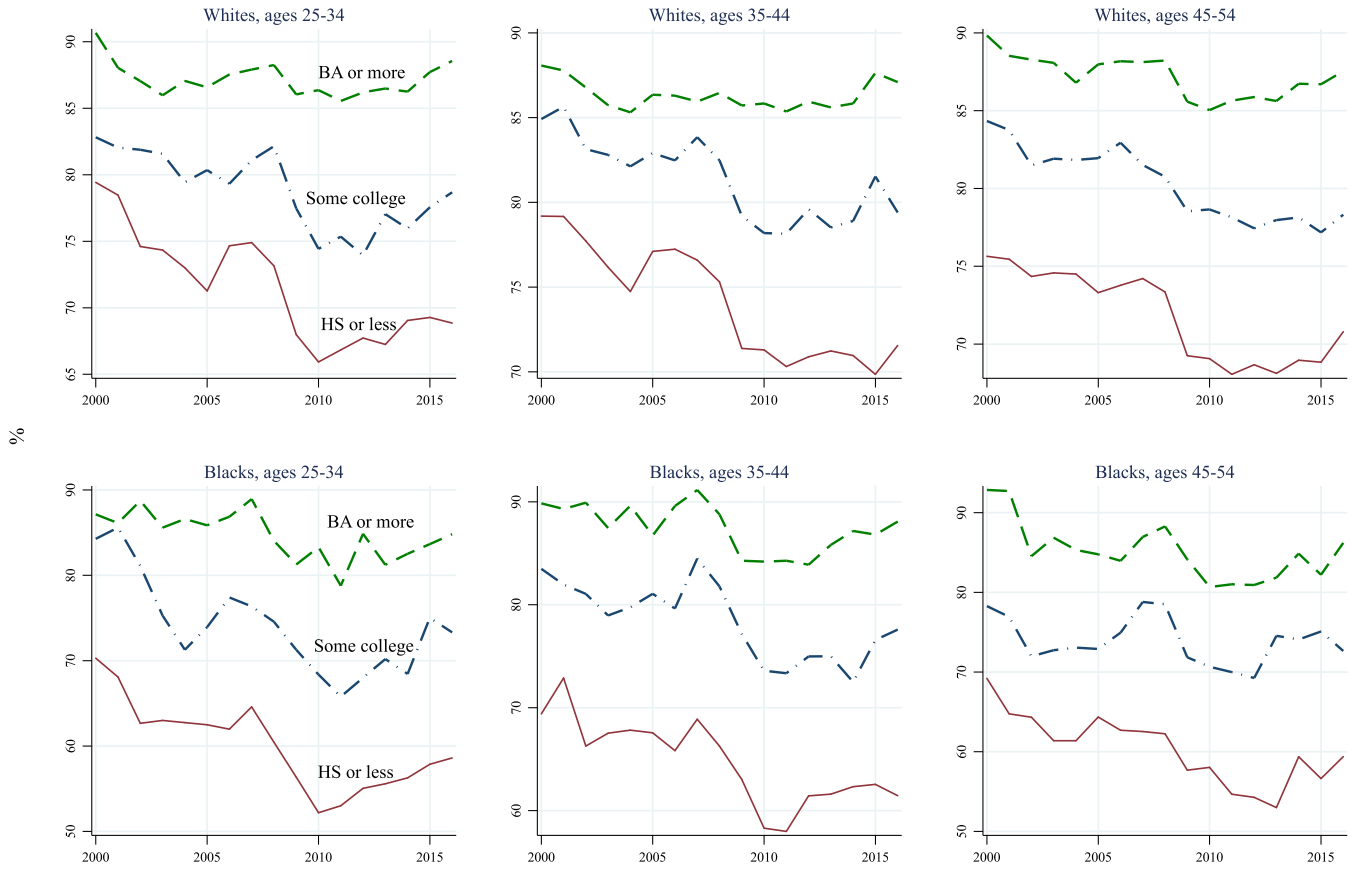


Fig. 8. Employment rate by 10-yr age-group and education level.
[a]Data from CPS March ("Center for Economic and Policy Research," 2016).
[b]Survey weights are used.

mechanism – stress and anxiety – that can induce the proximal causes of increased white mortality (substance use, suicide, cardiovascular disease, and diabetes).

5.2. Theoretical and empirical basis of social status threat

In theoretical terms, the notion of *perceived status threat* has long been established. The sociological and social psychological literatures posit that people are motivated to maintain their relative position in society and appraise this position by comparing themselves to others in society (Bobo, 1983; Enos, 2014; Lieberson, 1968). Racial threat theory suggests that, as marginalized groups increase in number, whites are more likely to worry that their status – their superior position in society – is declining (Bobo, 1983; Lieberson, 1968). Quite literally, they feel under siege.

From the economics literature comes the recently articulated notion of stratification economics (Darity et al., 2015), which brings to bear a population orientation to racial threat theory. While traditional racial threat theories are at least implicitly about individual perceptions of threat, stratification economics makes explicit that racial threat is about preserving "… the relative status of dominant *groups* …" (emphasis added; Darity et al., 2015).

Empirically, the literature is now awash with strong studies that point to a sense of status threat amongst whites. A 2017 study suggested that whites perceive blacks to have far more income and wealth than is actually the case (Badger, 2017; Kraus, Rucker, & Richeson, 2017). While the median black family in the United States earns just over half what a median white family earns (black families earn $57.30 for every $100 earned by a white family), whites perceive that blacks earn over eighty dollars or, more than eighty percent of what white families earn (Badger, 2017; Kraus et al., 2017). For wealth, the chasm between reality and white perception is even more dramatic. The median black family holds approximately five percent of the wealth of the median white family (black families have $5.04 in wealth for every $100 of the median white family's wealth), and yet whites perceive the figure to be over eighty percent (Badger, 2017; Hamilton et al., 2015; Kraus et al., 2017). These findings suggest whites perceive that blacks are economically catching up to them, even though this is not the case.

5.3. Additional descriptive analyses on indicators of racial threat

Data on white attitudes also indicate an increase in perceptions of threat to their social status. We analyzed indicators from the General Social Survey, which relate to perceptions of social status threat. We determined white trends in their levels of racial resentment (Fig. 10), happiness levels (Fig. 11), and subjective social status (Fig. 12). Unfortunately, an inadequate black sample prevented us from examining, separately, black attitudinal trajectories. Racial resentment was measured by asking respondents whether conditions make it difficult for blacks to succeed, whether blacks should be denied special favors to succeed, and whether blacks have gotten less than they deserve over the past few years. In 1986, more than fifty percent of whites did not agree with most of these statements (just over thirty percent did not agree that conditions make it difficult for blacks to succeed). By 2012, there was hardly any movement in the proportion of whites disagreeing with these statements, with the exception of the "conditions make it

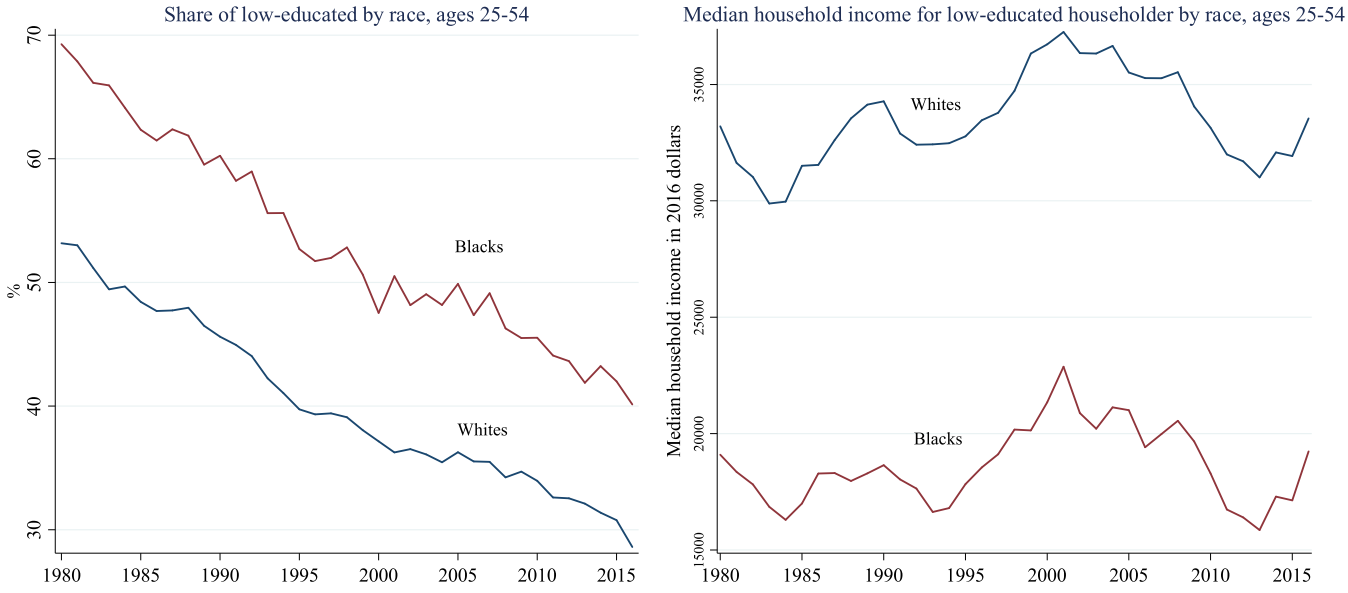


Fig. 9. Educational attainment and median for low-educated from 1980-2016.
[a] [a]Data from CPS March ("Center for Economic and Policy Research," 2016).
[b]Household income is household size adjusted with square root method.
[c]Median is calculated with household survey weights.
[d]Hispanic origin is excluded.
[e]Low-educated is defined as those who have high school diploma at most.

difficult" question, which increased to nearly a sixty percent disagreement rate.

Similar to subjective social status, whites had stable levels of happiness, which hovered on an ordinal scale above "pretty happy", but at the start of the current century, the happiness level of whites began to fall. Trends across education groups also mirrored those for subjective social status.

The General Social Survey asked respondents what they perceived their social class position to be: lower class, working class, middle class, or upper class. We created a continuous subjective social class measure by converting class position to an ordinal scale and averaging white respondents' answers for each year. From 1972 to 2004, whites collectively had a subjective social status that hovered between working-class and middle-class positions. But, thereafter, their subjective status has declined considerably, although it continues to remain predominantly between these two categories. Across education groups, trends are similar, but status decline is more pronounced among the less educated.

The 2016 election also put a spotlight on a growing sense of racial threat being experienced by whites. The widely proclaimed initial hypothesis for Trump's victory was that economically disgruntled whites saw hope in Trump's steady nod to "bringing back" jobs to blue-collar workers in predominantly white, rural areas (Monnat & Brown, 2017). For this reason, the speculation had it that if Bernie Sanders had been the democratic nominee, his pro-working-class policy platform (relative to Hilary Clinton's) may have won him the election (Chalabi, 2016; Le

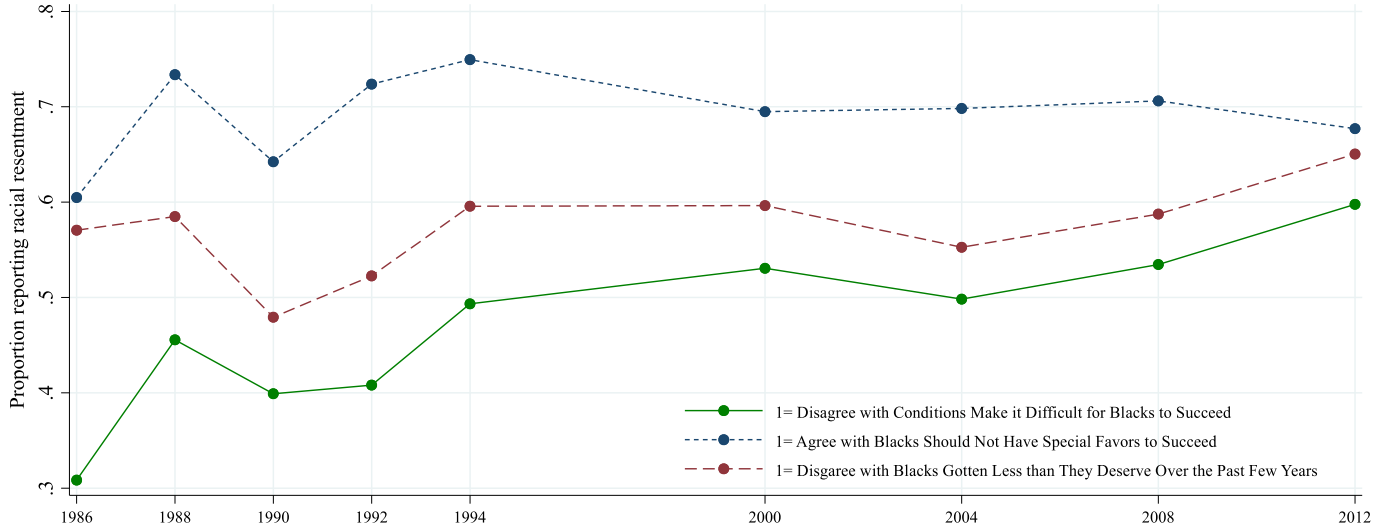


Fig. 10. Racial resenment: Non-hispanic whites, ages 25–54.
[a]Data from American National Election Studies 1986–2012 ("Center for Political Studies [producer and distributor]," 2010).

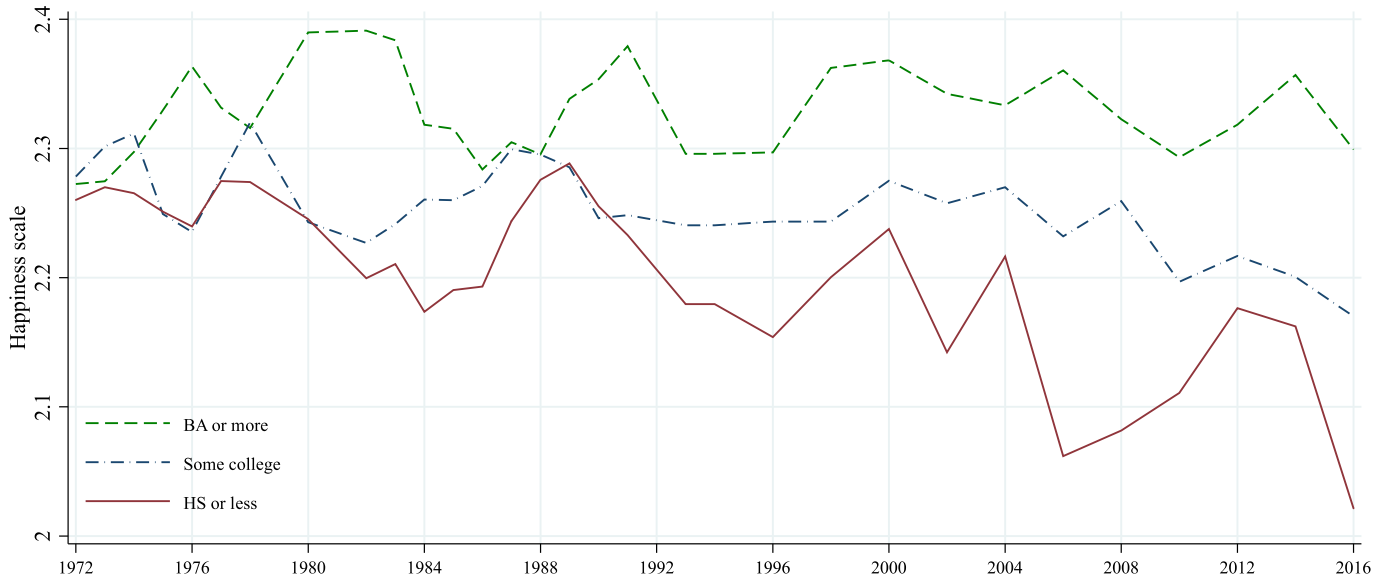


Fig. 11. Happiness by education level: Whites only, ages 25–54.
[a]Data from General Social Survey 1972–2016 (Smith, Marsden, & Hout, n.d.).
[b]Happiness is the mean levels of the ordinal happiness: not too happy = 1, pretty happy = 2, very happy = 3
[c]Hispanic origin is excluded.
[d]Data is smoothed with 3-year moving averages.

Miere, 2017).

However, recent studies have used survey data to show that racial threat to the status of whites, not economic disadvantage, was the main predictor of voting for Trump (Green, 2017; Knowles & Tropp, 2018; Major, Blodorn, & Major Blascovich, 2016; Mutz, 2018; Pettigrew, 2017). The thrust of Trump's election platform undeniably consisted of explicit racist propaganda, from the more subtle (but only marginally so) chants of “America First” and “Make America Great Again” to blatantly anti-black, anti-Hispanic, and anti-Muslim rhetoric and policy proposals that he made front and center (Green, 2017).

Survey data show that voting for Trump was associated with anxieties about growing racial diversity within the United States, and an increase in the interdependence of the United States with a broader globalizing world (Mutz, 2018). Voting for Trump also has been associated with the following attitudes: a propensity to believe that some social groups are superior to others, and that one's own social group is the most superior (Knowles & Tropp, 2018). Indeed, the confluence of these findings, combined with the demographics of Trump voters – predominantly white and spanning education, income, and gender categories (CNN Politics, 2016) – strongly supports the notion that social status threat has been weighing heavily on the minds of whites.

Of note, for the social status threat hypothesis to be a robust

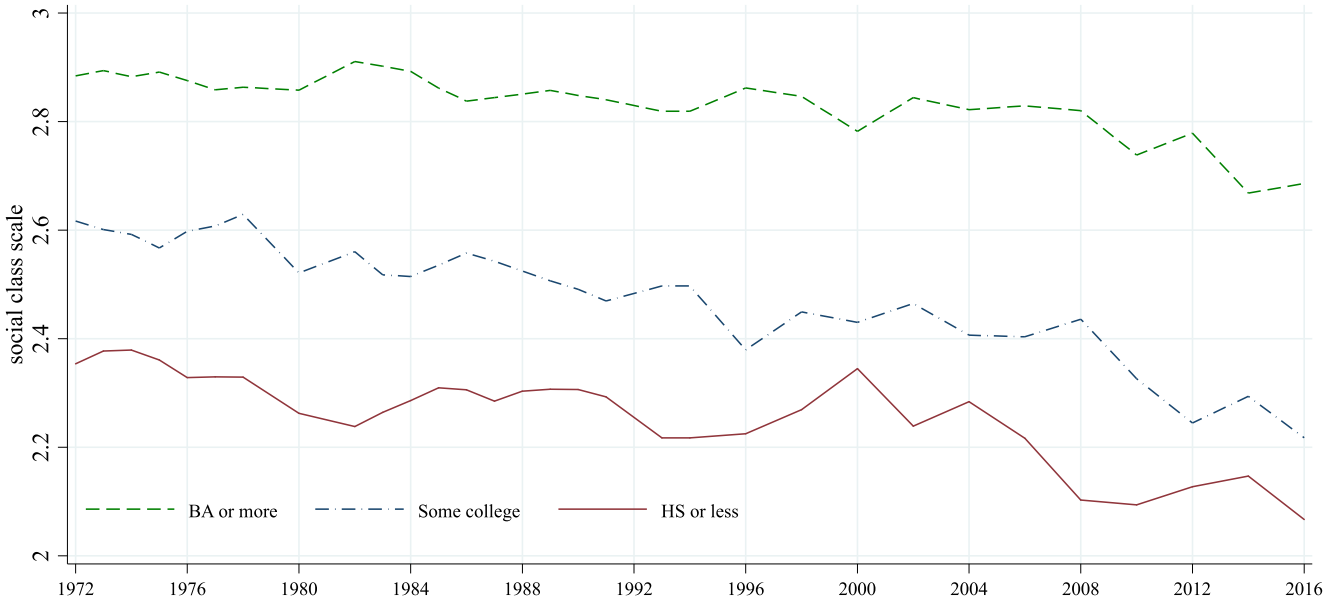


Fig. 12. Subjective social class by education level: Whites only, ages 25–54.
[a]Data from General Social Survey 1972–2016 (Smith et al., n.d.).
[b]Social class is the mean levels of the ordinal class: lower class = 1, working class = 2, middle class = 3, upper class = 4
[c]Hispanic origin is excluded.
[d]Data is smoothed with 3-year moving averages.

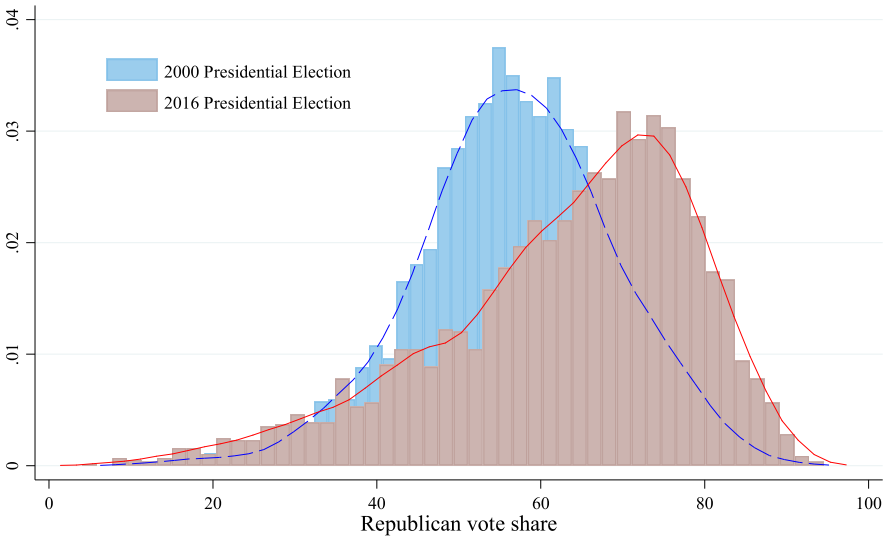


Fig. 13. County-level distributional shift in voting republican between 2000 & 2016.
[a]Data from Atlas of U.S. Presidential Elections (Leip, n.d.).

explanation for *rising* white mortality, ostensibly, perceptions of racial threat would need not only to exist, but to have increased over this time. The findings of Mutz (2018) suggest a movement from 2012 to 2016 towards perceptions of greater threat, and towards higher levels of Republican Party identification (Mutz, 2018). Moreover, our own calculations at the county-level suggest that, from 2000, at the start of the rise of white mortality, to 2016, there has been an increase in the proportion of Republican voters (Fig. 13).

Why might white status threat be increasing at this moment in time? A leading explanation from stratification economics and from sociological perspectives is that, in fact, the experience of an absolute decline in economic conditions, both short-term and long-term, may be producing a racial-threat response among whites. In other words, absolute declines in economic status of whites may produce a hyper-vigilance of sorts. This explanation would suggest that the short-term and long-term economic circumstance hypotheses forwarded by Case and Deaton (2017) are in some ways integral to the explanation of white status threat, rather than true competing explanations. However, we do not have sufficient evidence to test the veracity of this claim.

5.4. Fixed effects modeling of the racial threat-rising white mortality association

5.4.1. Premise of analyses

Recently, a smattering of studies have been exploiting the availability of population (county-level) data on the Republican share of voters in order to understand whether it is correlated with county-level opioid use, mortality, and life expectancy, with the results pointing to an association (Bilal, Knapp, & Cooper, 2018; Bor, 2017; Goldman et al., 2019; Goodwin et al., 2018; Knowles & Tropp, 2018; Major et al., 2016; Mutz, 2018). However, most of these studies have interpreted "Republican share" as a variable that represents some general economic and social malaise, much in the same spirit of Case and Deaton's (2017) thesis of a confluence of declining long-term economic and social circumstances. But no previous studies have linked the variable to its theoretical roots in social status threat.

Moreover, most previous analyses have been cross-sectional, or with shorter time horizons, and therefore have not assessed whether changes in the Republican share of voters is associated with change (rise) in white mortality. The aim of our analysis is to assess whether, at the county level, growth in the proportion of Republican Party voters over the past two decades is associated with the rise in white mortality over the same period.

Specifically, we are interested in determining whether the construct represents perceptions among whites of social status threat, as prior theory and empirical studies suggest, rather than more general economic and social malaise. Thus, our modeling strategy accounts for a large set of indicators of general malaise. Put differently, given our modeling strategy, if county-level changes in the share of Republican voters is associated with changes in county-level white mortality, then it is highly likely that this association is indicative of a link between rising white perceptions of racial threat and rising white mortality, rather than traditional economic and social population health indicators.

5.4.2. Methods

5.4.2.1. Data sources

County-level mortality. County-level all-cause mortality data for 25-64 year-old non-Hispanic whites for the years 2000–2016 was obtained from Multiple Cause of Death data available at CDC WONDER (https://wonder.cdc.gov/mcd.html). Counties with unstable mortality estimates (those with less than 20 deaths in a given year) have their mortality data suppressed and hence they were excluded. The final study sample consisted of 2241 counties for each year.

County-level republican vote share. In the United States, a move toward political support for the Republican Party has long been observed to be an outcome associated with white perceptions of racial threat (Knowles & Tropp, 2018; Major et al., 2016; Maxwell & Parent, 2012; Mutz, 2018; Walker, 2011). During the 2016 election, switching from a Democratic vote in 2012 to a Republican vote in 2016 was a better predictor of feelings of racial threat than economic anxiety – essentially "white flight" from the Democratic Party (Mutz, 2018). Furthermore, we explore the relationship between status threat and Trump preference. Trump preference is the strongest predictor for five of the nine indicators of status threat, and the second or third strongest predictor for the remaining four indicators (Appendix Table S1). Therefore, we use the change in share of Republican voters in a county as an indicator of change in perceived racial threat. Voting data for the 2000 and 2016 U.S presidential general election was obtained from the Atlas of U.S Presidential Elections.

County-level control variables. We also include a host of county-level social and economic variables in order to control for contemporaneous

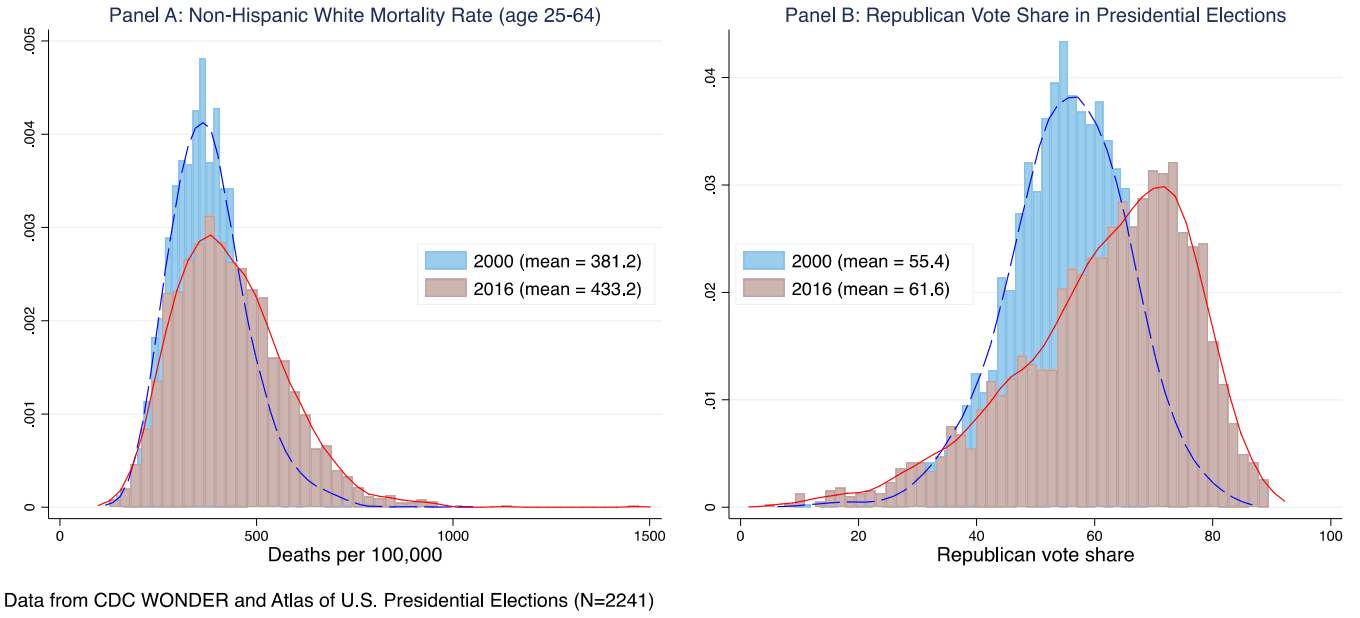


Fig. 14. County-level distributional changes between 2000 and 2016.

changes that also might influence white mortality. Data are consistently available for 2000 but not for 2016. When 2016 data are not available, we use the closest available year. Otherwise, we use the five-year average between 2012-2016. The following relevant variables come from American FactFinder (https://factfinder.census.gov): percent of population living under poverty line, percent of population with public assistance income, median household income; gini index of income inequality; median housing unit value; median gross rent; percent of population living in owner-occupied dwelling; percent of civilian employed population above 16; percent of employed in manufacturing; percent of population by marital status; and percent of population by educational attainment. Share of females and detailed race variables come from the U.S. population data available at National Cancer Institute's Surveillance Epidemiology and End Results (SEER) program (https://seer.cancer.gov/popdata/). County-level church adherence rate per 1000 population are from data collected by the Association of Statisticians of American Religious Bodies (ASARB) and distributed by the Association of Religion Data Archives (www.theARDA.com). Adherents are defined as "all members, including full members, their children, and the estimated number of other participants who are not considered members." Data on county population share in a rural area was taken from U.S. Census Bureau via American FactFinder. All monetary values are converted into 2016 inflation-adjusted constant dollars.

5.4.2.2. Model specification. We model the association between changes in Republican vote share and changes in working-age white mortality rates at the county-level using regression analysis. If we assume county-level mortality at time t is a linear function of economic and socio-demographic indicators of the county at time t, other county-specific time-constant variables, time t, and state specific time trend, our model is:

$$Y_{ist} = \beta_1 X_{ist} + \beta_2 Z'_{ist} + \vartheta_i + \mu_t + \lambda_s \mu_t + \varepsilon_{ist} \quad (1)$$

Y_{ist} is the working-age white mortality rate in county i state s at time t
X_{ist} is a Republican vote share in county i state s at time t
Z'_{ist} is a vector of economic and socio-demographic variables in county i state s at time t
ϑ_i is a county specific fixed effect
μ_t is a time t specific fixed effect
$\lambda_s \mu_t$ is a state specific linear time trend
ε_{its} is a random disturbance clustered within state

where t is [0,1] indicating 2000 and 2016 (or similar years). In order to examine the effect of change we take the first-difference of the above model (1) with an early period of 2000 and a later period of 2016 (or similar). The resultant change model is:

$$\Delta Y_{is} = \beta_1 \Delta X_{is} + \beta_2 \Delta Z'_{is} + \alpha + \alpha\lambda_s + \upsilon_{is} \quad (2)$$

where $\alpha = \mu_1 - \mu_0$ and $\upsilon_{is} = \varepsilon_{is1} - \varepsilon_{is0}$. β_1 and β_2 are the corresponding parameter and a vector of parameters to be estimated. β_1 is the parameter of interest that tells us the effect of one percentage point increase in the county's Republican vote share on the number of white deaths per 100,000 population in the county. The first-difference strategy takes care of time-constant unobserved heterogeneity between different counties (states) that might bias our estimates. However, many state specific laws and policies that might affect the mortality outcome are not always time-constant. Hence, we further control for the year and state fixed effects in our model. Moreover, we use robust standard errors, clustered at the state level to account for within-cluster correlation or heteroskedasticity. We estimate the model using the 'xtreg, fe' command in STATA-15.

We build our model from a simple regression of the main independent variable without any controls to a full model with all controls, by subsequently adding groups of controls.

$$\Delta \text{WhiteMort}_{is} = \beta_1 \Delta \text{Repshare}_{is} + \alpha + \alpha \text{ x State}_s + \text{Error}_{is} \quad (3)$$

$$\Delta \text{WhiteMort}_{is} = \beta_1 \Delta \text{Repshare}_{is} + \beta_2 \Delta \text{SocDem}_{is} + \alpha + \alpha \text{ x State}_s + \text{Error}_{is} \quad (4)$$

$$\Delta \text{WhiteMort}_{is} = \beta_1 \Delta \text{Repshare}_{is} + \beta_3 \Delta \text{Econ}_{is} + \alpha + \alpha \text{ x State}_s + \text{Error}_{is} \quad (5)$$

$$\Delta \text{WhiteMort}_{is} = \beta_1 \Delta \text{Repshare}_{is} + \beta_2 \Delta \text{SocDem}_{is} + \beta_3 \Delta \text{Econ}_{is} + \alpha + \alpha \text{ x State}_s + \text{Error}_{is} \quad (6)$$

5.4.3. Results

5.4.3.1. Descriptive characteristics(Table 2, Fig. 14, Fig. 15, Fig. 16). Fig. 14 shows the density curves between 2000 and 2016

Table 2
Summary statistics of variables pre and post.

	Pre				Post			
	mean	sd	min	max	mean	sd	min	max
2000 & 2016								
White deaths per 100,000	381.17	99.84	143.10	1023.30	433.17	139.23	125.00	1473.50
% Republican vote	55.40	10.41	8.95	84.02	61.64	14.86	4.09	89.48
% Female	50.62	1.63	35.38	55.78	50.24	1.75	34.35	56.25
% White	83.29	16.28	10.41	99.22	78.71	17.76	6.67	98.48
% Non-white	16.71	16.28	0.78	89.59	21.29	17.76	1.52	93.33
% Black	9.13	12.94	0.06	79.28	9.79	13.04	0.37	78.00
% Hispanic	5.28	9.26	0.29	88.56	8.36	11.19	0.55	91.84
% Asian or Pacific Islander	1.19	3.32	0.01	71.53	1.92	3.75	0.06	69.76
% Indian or Native	1.11	3.30	0.03	47.72	1.22	3.49	0.06	46.79
2000 & 2012–2016								
% Married	59.90	4.95	30.92	74.58	52.96	5.46	25.87	66.13
% Never married	22.82	5.41	10.89	56.13	28.14	6.19	14.52	61.06
% Widowed or divorced	17.28	2.47	7.94	26.89	18.90	3.03	8.37	29.44
% BA or more	17.16	8.29	4.90	60.50	21.58	9.61	5.15	73.67
% Some college	25.87	5.40	11.10	42.80	30.04	4.73	11.91	46.11
% HS or less	56.97	11.53	15.80	83.10	48.38	10.70	14.04	80.03
Gini Index	0.43	0.04	0.33	0.60	0.45	0.03	0.35	0.60
Median household income ($2016)	52854	13179	23334	118926	48752	12910	18972	125672
Median house value ($2016)	126643	59180	29829	707424	146409	82363	33600	871500
Median gross rent ($2016)	673.76	173.73	332.70	1699.37	747.61	199.34	386.00	1861.00
% Employed	57.65	7.31	27.80	83.60	54.40	7.75	21.90	77.90
% Employed in manufacturing	17.66	8.65	1.10	48.60	13.49	6.88	1.20	48.30
% Owner occupied housing	75.29	7.63	18.89	90.78	71.51	8.09	20.22	89.01
% Below poverty	13.41	5.76	2.10	39.70	16.49	5.84	3.70	42.50
% With public assistance	3.31	1.62	0.50	14.60	2.51	1.27	0.20	11.90
2000 & 2010								
Church adherence per 1,000	502.67	154.99	92.59	1547.31	488.49	154.52	51.32	1924.61
% Rural	51.59	28.37	0.00	100.00	49.87	28.63	0.00	100.00
Region (National share)								
Northeast (6.98)	9.33							
Midwest (33.89)	30.70							
South (45.72)	48.95							
West (13.41)	11.02							
Number of counties	2241				2241			

for the Republican vote share and the white mortality rate. The mortality rate for working-age (25–64) non-Hispanic whites has increased by 52 more deaths per 100,000 population (381 vs. 433), while the Republican vote share has grown by more than 6 percentage points (55.4 vs. 61.6). The figure suggests that the entire distribution for both variables has undergone a shift to the right over this interval.

Table 2 provides summary statistics for the sample counties for pre (2000) and post (2016 or similar) period. During this period, on average the economic and socio-demographic characteristics of the counties changed quite significantly. In terms of demographic characteristics, the non-white share increased by nearly 4.6 percentage points (16.7 vs. 21.3) with the most pronounced increase in the Hispanic population (5.3 vs. 8.4). The proportion of married people has fallen by almost 7 percentage points (59.9 vs. 53) due largely to the increased number of persons never married (22.8 vs. 28.1); and the overall population has become more educated, with the lowest educated group (those with a high-school degree or less) declining by 8.6 percentage points (57 vs. 48.4).

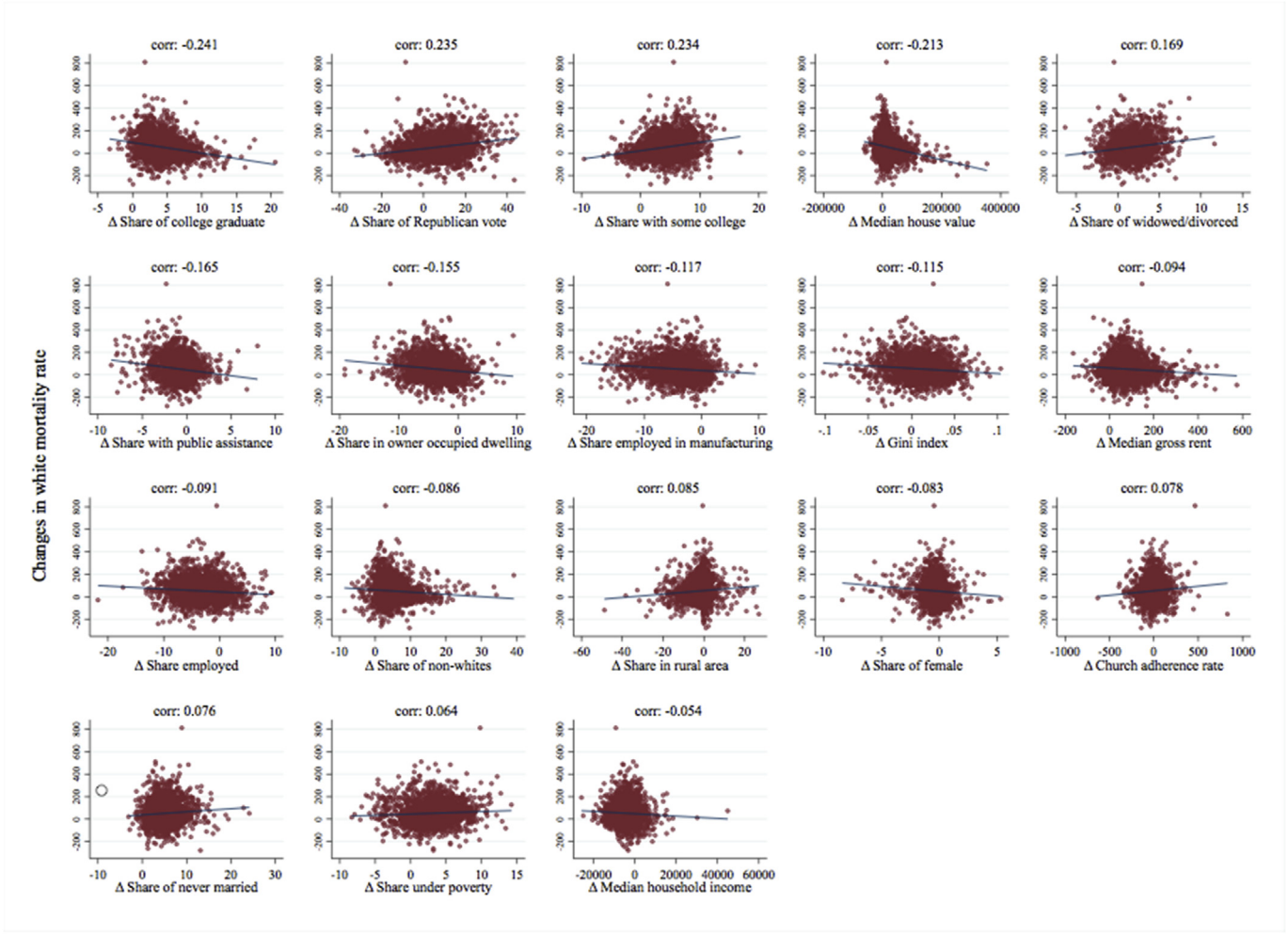
Macroeconomic conditions deteriorated between 2000 and 2016. After adjustment for inflation in 2016 dollars, the average county median household income fell by more than $4,000 ($52,854 vs. $48,752), while the average county median house value ($126,643 vs. $146,409) and the median gross rent ($674 vs. $748) both rose in real terms. Overall, fewer people had jobs (57.7 vs. 54.4), employment in manufacturing declined (17.7 vs. 13.5), and people were less likely to live in owner-occupied housing units (75.3 vs. 71.5). Moreover, the poverty rate increased (13.4 vs. 16.5) but public assistance coverage declined (3.3 vs. 2.5). Fewer people had connections to the church, as evidenced by decline in adherence rate per 1,000 residents (502.7 vs. 488.5).

As shown in Fig. 15, most variables have correlations in the expected direction. Of all covariates, change in share of Republican voters ($r = 0.24$) and college degree attainment ($r = -0.24$) were the most strongly correlated with change in white mortality, suggesting that counties that became more Republican and that did not experience much change in college attainment also had increased rates of white deaths. Median income had the weakest correlation with white mortality at a county-level.

Fig. 16 maps the changes in median household income and Republican vote share at a county-level from 2000 to 2016. As shown in the maps, there is no clear indication that the counties that experienced income decline are the same counties that have increasing white mortality rate. In fact, there is a positive correlation between the two variables for the analytic sample of 2241 counties (Appendix Fig. S1).

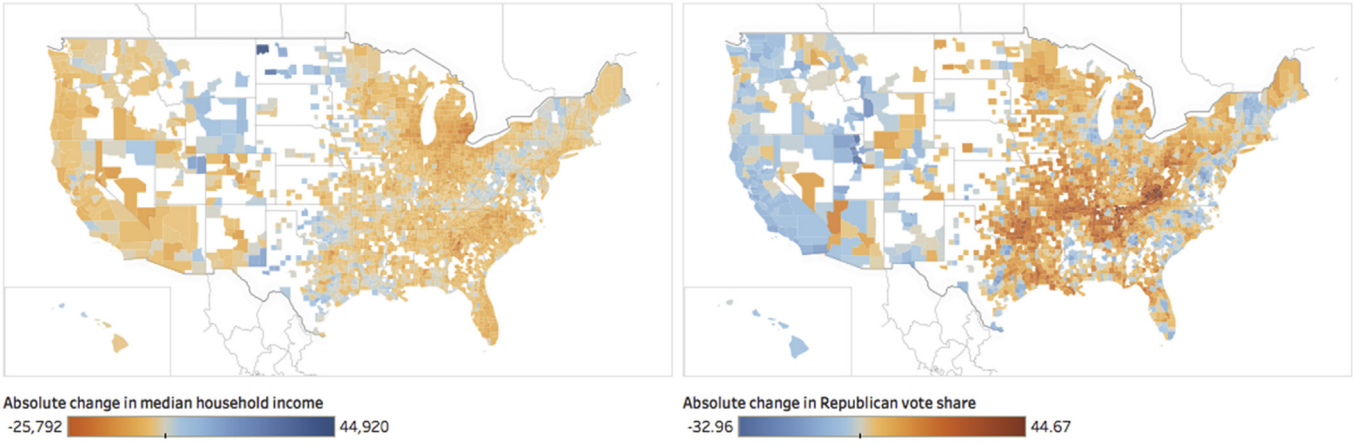
5.4.3.2. First-difference fixed effect model (Table 3). The estimation results of our regression analysis are shown in Table 3. The main independent variable, change in share of Republican voters, remains significant and positively associated with white mortality rate in all four regression models, where we successively take into account county fixed effects, time fixed effects, and state-time fixed effects, and accounted for robust standard errors clustered at the state-level.

Model 1 provided the base model, without any control variables, and indicates that a one percentage point increase in the Republican



Note: All covariates are first difference change from pre (2000) and post (~2012-2016) at a county level.

Fig. 15. Correlation between changes in white mortality rate and changes in covariates
Note: All covariates are first difference change from pre (2000) and post (~2012–2016) at a county level.



Note: Only counties that are in the analytic sample are mapped.

Fig. 16. Changes in median household income vs. Republican vote share at a county-level from 2000 to 2016
Note: Only counties that are in the analytic sample are mapped.

Table 3
Estimation results of first-difference fixed-effect model.

	Age-adjusted all-cause white mortality (ages 25–64)			
	Model 1	Model 2	Model 3	Model 4
Share of Republican vote	2.070***	2.092***	1.459***	1.492***
	(7.94)	(6.79)	(3.85)	(3.86)
Share of female		−3.950		−3.131
		(-1.40)		(-1.03)
Share of never married		2.269*		0.641
		(2.42)		(0.87)
Share of widowed/ divorced		4.740**		3.172
		(2.90)		(1.98)
Share of non-white		1.703***		1.075*
		(3.83)		(2.10)
Church adherence per 1,000		0.0600*		0.0441
		(2.49)		(1.98)
Share in rural area		1.215*		0.612
		(2.38)		(1.37)
Share of college graduate			−3.583***	−2.563*
			(-3.78)	(-2.36)
Share with some college education			0.609	0.870
			(0.62)	(0.89)
Gini index			−47.38	−29.77
			(-0.35)	(-0.23)
Median household income in 1000s			−0.275	−0.0851
			(-0.29)	(-0.098)
Median house value in 1000s			−0.374***	−0.324***
			(-4.46)	(-3.80)
Median gross rent			−0.0487	−0.0574
			(-1.36)	(-1.60)
Share employed			1.656	1.651
			(1.83)	(1.80)
Share employed in manufacturing			−0.116	−0.305
			(-0.12)	(-0.31)
Share owner occupied housing			−1.181	−1.038
			(-1.08)	(-0.95)
Share under poverty			1.659	1.496
			(1.18)	(1.08)
Share with public assistance			−4.641*	−4.183*
			(-2.46)	(-2.17)
County fixed effect	Yes	Yes	Yes	Yes
Time fixed effect	Yes	Yes	Yes	Yes
State and time fixed effect	Yes	Yes	Yes	Yes
State cluster-adjusted standard error	Yes	Yes	Yes	Yes
N	4482	4482	4482	4482

t statistics in parentheses * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$.

vote share was associated with two more deaths per 100,000 population. Model 2 adds socio-demographic controls to the base model and produces a similar result to the base model. Model 3 adds proxies for economic resources, including education. In this model, the effect of Republican share is somewhat attenuated, suggesting that controlling for economic covariates, a one percentage point increase in the Republican vote share is associated with 1.46 more deaths per 100,000 population.

Model 4 includes both economic and socio-demographic controls, and additionally controls for time-variant major economic and socio-demographic characteristics of the counties, unobserved time-constant heterogeneity across counties, and a state specific time trend. It indicates that a one percentage point increase in the Republican vote share is accompanied by an increase in white mortality rate by 1.49 deaths per 100,000 residents.

Many of the control variables are not significant, with the exceptions of: share of college graduates, median house value and the share with public assistance. A one percentage point increase in the share of college graduates was associated with nearly 2.6 fewer white deaths per 100,000 population, while a $6000 increase in median house value – a proxy for wealth – dropped the death rate by 2 deaths per 100,000 population. A one percentage point increase in public assistance coverage was associated with more than 4 fewer white deaths per 100,000 population.

We also conduct a sensitivity analyses by introducing each county control one by one into our base model, to check the effect of each variable on the parameter of the main independent variable (Appendix Table S2). Then, we start with a full model and removed each county control one by one to check if the exclusion of certain variable distorts our results (Appendix Table S3). Our estimation result remained significant with a magnitude for the Republican vote share parameter ranging between 1.4 and 2.4 for all of these regressions.

5.4.4. Limitations

There are two major limitations to our work. The first is that it is difficult to completely isolate the influence of Republican vote share, because of the interconnectedness of this variable with other socio-economic conditions. In the absence of an instrumental variable, or of a natural experiment, our study provides a conservative estimate of the effect of the Republican vote share by controlling for a host of economic and social factors (Basu, Meghani, & Siddiqi, 2017).

The second is the simultaneity in the modeling of our exposure and outcome variables; while we examined change, our model does not lag the exposure variable, in order to better assess whether the association we found is one of “cause” and “effect.” Because we lacked data on Republican vote share during non-presidential elections, we were unable to construct a lagged model.

6. Discussion

This paper extends an emergent literature on the identification and explanation of unusual white mortality trends over the past nearly two decades. This work has been principally driven by two descriptive studies from Anne Case and Angus Deaton, who have argued that, while mortality rates are falling for all other groups in the United States and for the overall populations of other comparable high-income countries, mortality rates are rising for white Americans (Case & Deaton, 2015, 2017).

Moreover, this work has argued that the rise in white mortality is largely confined to subgroups of whites. It is concentrated among whites with a high school education or less. It is found mainly in 50–54 year-olds. It is more accentuated in women than in men. It is more pronounced in some regions of the United States than others.

Case and Deaton (2017) and commentators on their work present persuasive evidence that the rise in white mortality coincides with a rise in deaths due to opioid use, alcohol use, suicide, and to a lesser extent, cardiovascular disease and diabetes, all which they consider to be the proximal causes of the phenomenon. Most significantly, they suggest that long-term (post 1970s) declines in the economic and social prospects of whites with the lowest levels of education account for the observed white mortality trends.

We depart from Case and Deaton in two important ways. First, we suggest that the phenomenon is not restricted to whites with the lowest educational attainment, nor to those in a small subset of the working-age white population. While we agree that whites with only a high school education were hardest hit, we suggest that the slightly smaller rises, and maybe even the stagnation of mortality rates in higher education brackets, is part of a collective phenomenon of worsening white mortality rates. These observations have led us to characterize the unusual post-1999 white mortality trends in more sweeping terms.

Second, we believe for two reasons that neither short-term nor longer-term economic hardship can, on its own, explain white mortality trends. First, we see the phenomenon as cutting across economic groups. Second, this phenomenon has only occurred among whites, but the economic trajectories of whites and blacks have been parallel, and

blacks have always been more economically disadvantaged in both the long- and short-term. Rather, we hypothesize that the anxiety of whites is coming from a perception – a misperception – that their dominant status in society is being threatened, which is manifesting in multiple forms of psychological and physiological stress. While stratification economics suggests that this misperception may actually be quite functional for preserving relative group status, it may have health consequences. Indeed, the empirical test we provide of our hypothesis suggests this to be the case.

To be sure, this is a startling finding. The social status threat mechanism clearly has emerged as a way to explain the election of a presidential candidate who espoused highly racist views (Green, 2017), but we are now suggesting that this mechanism also explains the highly unusual phenomenon of worsening white mortality – and worsening white health more generally. Moreover, we are suggesting that the perception of racial threat among whites is occurring in the absence of substantive evidence of a decline in their relative social status, since both whites and blacks are experiencing parallel economic declines (Badger, 2017).

In the context of the broader population health literature, our findings are all the more stunning. Traditional explanations for population health inequalities have been rooted in economic disadvantage – whether absolute or relative (Adler et al., 1994; Marmot, 2005). Groups with more social and economic disadvantage are less healthy (Link & Phelan, 1995).

For perhaps the first time, we are suggesting that a major population health phenomenon – a widespread one – cannot be explained by actual social or economic status disadvantage but instead is driven by perceived threat to status.

However, our sense from the population health literature is that sustained economic and social disadvantage still determines absolute levels of health inequalities across social groups. Throughout history, blacks have faced many more barriers to earnings and accumulating wealth than whites. These barriers also translate to more sources of economic and social stress and strain. Blacks are also subject to discrimination in the housing market (Roscigno, Karafin, & Tester, 2009), the criminal justice system (Alexander & West, 2018), the health care system (Care, Policy, & Medicine, 2009), and in nearly every facet of life (Bonilla-Silva, 2009). The combination of a tangible lack of resources and the stresses of everyday life imposed by discrimination (Ramraj et al., 2016; Siddiqi et al., 2016; Williams & Mohammed, 2013) make it quite easy to understand why blacks have far higher mortality rates than whites (Levine et al., 2001).

Our findings suggest that, while absolute levels of disadvantage are better at predicting overall, absolute levels of population health inequalities, short-term changes may be driven by a broader range of phenomena. In the present case, short term rising white mortality seems to be driven principally by anxiety among whites about losing social status, even in the absence of evidence that they are, in fact, ceding status to blacks.

Our paper makes a contribution to understanding a newly identified population health phenomenon. It also adds an interesting twist to the broader literature on population health, which until now, has not incorporated status threat as a major determinant of population health or health inequalities.

Conflicts of interest

None.

Financial disclosures

None.

Ethics statement

Our paper uses publicly available, secondary, de-identified, ecological data, and as such is exempt from ethics review.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.ssmph.2019.100449.

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