Beyond Unhealthy Behavior and Medical Care: Applying a Biological Plausible Framework Toward Understanding the Role of Long Term Economic Distress in Explaining Recent Trends in US Adult Life Expectancy

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INCOMPLETE AND VERY PRELIMINARY

Independent researchers have used a variety of data sources and methodologies to document two disturbing trends in the US in recent decades: a halt to universal gains in life expectancy (LE) across US subpopulations, and a subsequent rise in educational and income inequality in LE.(1–6). At the same time, incomes for those without a college education have stagnated at least since 1980,(7,8) and the probability that children will do better economically than their parents has been falling.(9,10), with these patterns concentrated in areas of the country hit hardest by globalization and technological change. The timing of the trends in LE suggests that the stagnating or deteriorating economic prospects of moderate-income households over the long-term in light of jobs lost to globalization and technological change may play an important role.

In this paper, we discuss and apply a conceptual framework linking long-term declining economic prospects to trends in mortality and life-expectancy. The framework emphasizes the direct and indirect impacts that chronic exposure to economic and other stressors have on the body. Geronimus, who coined the term "weathering" to refer to this phenomenon, has applied this framework toward understanding US racial and geographic inequities in healthy life expectancy(11,12). The current paper applies this framework to the study of educational inequities within the adult white and black populations. This framework provides biologically plausible mechanisms through which long-term economic trends could exert their impact on health and mortality.

Outline of the paper

We estimate models that generate plausibly causal estimates of the relationship between economic stagnation and mortality between 1990 and 2017 by leveraging geographic differences in exposure to economic distress over time to identify the role of economic conditions on mortality trends. In particular, we systematically test whether deteriorating economic prospects can explain both the stagnation in life expectancy for the less educated and the growing educational differentials in life expectancy. We find that there is a statistically and economically significant association between economic stagnation at the level of the commuting zone and a stagnation in the growth of LE among white and Black men and women in general, which is concentrated amongst the less well educated. We analyze which causes of death mediate this association finding that stress-related chronic diseases such as cardio-metabolic diseases are the major contributors.

It is plausible that behaviors such as the use of illicit drugs and smoking could mediate the relationship between economic stagnation and mortality. Alternatively access to adequate health care could play an important role. Using back of the envelope calculations, we find evidence that health behaviors and access to health care do account for a modest part of the estimated effect of long-term economic distress on the life expectancy of the less educated. However, we find a large, unexplained residual, which we interpret as suggestive of an important direct role for stress for explaining the patterns we find.

Biological Plausibility of Weathering:

A vast and growing body of evidence has established that chronic stress is associated with a broad range of poor health outcomes, including cardiovascular disease, diabetes, autoimmune disease, depression, respiratory infections, poorer wound healing and cancers.(13–17) This

evidence is based on animal models, natural and quasi-experiments in human populations, and a few human experiments that assess stressful events, physiological response, and disease outcomes in the same subjects.(18) In brief, exposure to every-day adversities shaped by structural disadvantages trigger repeated activation of physiological stress processes, activating the hypothalamic- pituitary-adrenal (HPA) axis and sympathetic nervous system (SNS).(19,20) This feedback system prepares the body for responses to stressful situations, for example by signaling for increased cortisol secretion, in order to utilize stored energy and respond to threats.(20,21) But repeated or chronic exposure to stressors inhibits the body's ability to efficiently turn off the HPA and SNS, a circumstance associated with dysregulation of glucocorticosteriods, neurotransmitters, and inflammatory cytokines.(20) Persistent activation of allostatic systems has detrimental effects on cellular systems, including pathogenic gene expression and dysregulation and acceleration of the normal cellular aging process.(22)

These pathways are thought to be especially important in the early onset of chronic stress-related diseases and cancers. A recent report provides evidence that individuals experiencing social exclusion express the over-activation of genes involved in the inflammatory response system, and the under-activation of glucocorticoid response elements critical to the anti-inflammatory response system.(23,24) Inflammation is central to cardiovascular disease and can cause DNA damage and lead to cancers. Additionally, if tumor suppressor genes are silenced via gene-specific hyper-methylation, cancerous cells tend to develop, grow, and proliferate at a rapid pace.(25–28) Growing evidence indicates that physical and social environments play a role in hypermethylation.(23,24,29) Cole(23) provides a helpful illustration of the potential pathways, depicting a dynamic flow of information from the social environment to protein formation, health, and behaviors via perceptions formed in the central nervous system, neuroendocrine

responses, and transcriptional regulation of gene expression.

There is direct evidence of links between economic stress and chronic health conditions, Thus, for example, Seeman et al,(30) using data from the Multi-Ethnic Study of Atherosclerosis, found evidence that the 2008 Great Recession (GR) adversely impacted blood pressure, fasting glucose, and medication usage among participants who were most likely to be directly affected economically—working-age adults in the labor force when the GR hit and older adults relying on investment or homeownership wealth. Such findings provide evidence that acute economic shocks can result in poor stress-related health outcomes that if sustained long-term will ultimately increase risk for cardio-metabolic diseases, cancers, and other internal causes of death. Thus, while their within-cohort study of a single economic shock did not address our research question-- the long-term health sequelae of secular economic stagnation across cohorts -- it does provide evidence that the body responds to economic distress in ways that if prolonged could portend the erosion of key body systems over time and the development or progression of chronic health vulnerability.

Methods

Economic conditions

We examine both trends in age-adjusted overall mortality and Years of Life Lost (YLL) between ages 25 and 84, leveraging geographic variation in economic downturns over the past several decades to illuminate whether associations between long-term economic distress and stagnating life expectancy are plausibly causal and, if so, through which among a broad range of causes of death.

We used data from the decennial US Census and the American Community Survey to construct a location-specific measure of long-run economic distress.(31) Some studies have used geographic variation in the potential impact of the increases in trade with China to measure the negative health and mortality effects of such increases(32–34). However, the disruptive effects of trade on the US labor market are not limited to the US trade with China(35) and available evidence suggests that technological change has at least as important a negative effect on the labor market of the less than college educated as trade.(36–39) Our measure of economic distress, as elaborated below, captures shifts due both to trade and to technological change.

As a plausibly exogenous measure, we calculated the predicted (log) change in employment between 1980 and 2017 as a function of national-level changes in industrial composition, weighted by the local area distribution of employment by industry at the beginning of that period. That is,

$$\widehat{\Delta \ln E_g} = \sum_j s_{jg} \Delta ln S_j \quad (1)$$

where s_{jg} denotes the share of area g's total employment that is in industry j at baseline (1980), and $\Delta ln S_j$ is the log change in national employment share in industry j. An inspection of (1) makes it clear that the variation across local areas in this measure reflects the industrial composition of the area at baseline. Such measures, typically referred to as "Bartik instruments," have been commonly used in economics to identify plausibly exogenous measures of predicted employment growth.(40–42) Indeed, similar measures have been used in the work estimating the impact of trade on mortality.(32)

Given available data, there are several choices for the level of geographic aggregation. The smallest area identified in vital statistics data is the county, but local labor markets typically span more than single counties as employers in urban areas draw employees from not only

central urban counties where they are located but also suburban and exurban areas. States are the next largest political division, but multiple distinct labor markets exist in many states with often very different industrial bases. For example, New York City's economy is heavily dependent on the banking and financial services industry, whereas Elmira, New York, and the surrounding communities are relatively much less dependent on these sectors and more dependent on durable goods manufacturing. Thus, as a more accurate measure of local industrial composition and potential exposure to macroeconomic trends, we aggregate mortality and economic conditions to the level of the commuting zone (CZ). This commonly used grouping divides the entire country into sets of contiguous counties that, according to census "Journey to Work" data, constitute geographic clusters in which a large majority of the area's employees live and the area's residents work.(43) There were 741 such CZs in 1990.

Figure 1 displays the variation in this measure of predicted employment growth by CZ. Persons living in areas that began the period with an industrial mix well positioned to benefit from broader economic trends of the 1980s–2010s (e.g., the CZ surrounding Santa Fe, New Mexico, which had a high concentration in office machinery and computer manufacturing) were likely to experience less economic distress than people living in areas with industries that experienced severe downturns (e.g., Greenville, South Carolina, with a high concentration in textile manufacturing).

Socioeconomic position

We use education to sort the population into relatively advantaged and disadvantaged groups. Education is an important driver of health, mortality, income, and wealth, and, unlike income or wealth, education is available in both census and vital statistics data. Because Americans obtain

most of their education by early adulthood, educational attainment is less likely than either income or wealth to be endogenous to adult health outcomes.

Mortality outcomes

Two types of mortality outcomes are used in these analyses: age-standardized death rates and YLL between ages 25 and 84, calculated as the difference between 60 (the maximum number of years one can live in the interval) and the expected person-years lived among those who survive to age 25 ($60 * l_{25} -_{60} L_{25}$). To calculate total and cause-specific death rates, we used a restricted version of the Multiple Cause of Death (MCD) file, including county of occurrence for each death record,(44) the public use microdata samples from the 1990 decennial census (5 percent), and the 5-year American Community Survey (ACS) for 2019, which includes respondents from the 2015-2019 surveys.(31) The beginning date for measuring mortality changes (1990) was chosen because education was first collected in the U.S. standard death certificate in 1989.(45) While this does not correspond to the period over which economic conditions are measured, we do not expect the effects of economic distress to be reflected immediately in mortality rates. The end date (2017) was selected to avoid the influence of COVID-19 on the 5-year pooled population denominator data. Because states collect vital statistics data, we divide any CZ spanning multiple states into separate state-specific areas. This split produces 886 state-CZ areas. In the appendix, we show that the effect of splitting CZs into state-specific units is minimal. To reduce noise, counts of deaths by cause, sex, age, education, and race were averaged over three years at the beginning (1989-1991) and end (2016-2018) of the period. Rates were first calculated in standard five-year age brackets used in abridged life tables.

To calculate rates by SEP, we divide the population by relative rank in educational attainment. As previous research has shown, the dramatic changes in educational attainment across birth cohorts during the 20th century may make mortality comparisons based on levels of schooling misleading.(4) Thus, we classify death certificates and census observations by whether the individual was in the bottom quartile or top three quartiles of the education distribution for their birth year, race, and sex cohort (see the appendix for details on the method for assigning quartiles).

Total and cause-specific death rates were then calculated by direct standardization using sex-specific age distributions of the combined non-Hispanic Black and white populations in 1990. Cause-specific death rates were calculated based on the underlying cause of death coded on the death certificate, grouped into 6 exhaustive and mutually exclusive categories: infectious and parasitic disease, cancers, cardio and metabolic diseases, substance use and mental health, other body systems (sometimes referred to as "internal" causes such as diseases of lung, liver, nervous system, etc.), and other causes of death (sometimes referred to as "external" causes such as accidents, homicide, etc.). The sum of changes in death rates for these categories sums to the change in the all-cause death rate. Similarly, the cause-specific effects of a change in the Bartik instrument sum to the all-cause effect within each group. Within the category of substance use and mental health, we also calculated statistics separately for two specific categories: opioid-related drug poisoning and non-drug suicide. YLL between various ages, calculated only for to all-cause mortality, were generated using standard life table methods (see the appendix for details).

The subset of state-CZ areas used in our main analyses are restricted to those with complete data and sufficient population to generate meaningful mortality rates. To that end, we

first exclude race, sex, state, and CZ cells from the analysis if there was not at least one person in each five-year age category between ages 25 and 84 in either the 5 percent public use microdata sample from the 1990 Decennial Census or the 5-year ACS file spanning 2025–2019. Because the Black population in the US is more geographically concentrated than the white population, there are a substantial number of CZs where there is a sufficient number of white residents but an insufficient number of Black residents to calculate population-based rates. We also excluded areas in seven states (Georgia, Louisiana, New York, Oklahoma, Rhode Island, South Dakota, and Washington) that did not include education on death certificates in 1990, and we excluded areas in New Hampshire because it did not include Hispanic ethnicity on death certificates in 1990. This results in samples of 707 state-CZ areas for white males and females and 311 for Black males and females. We examine the sensitivity of our results to these exclusions in the appendix.

Model Specification and Statistical Inference

As the primary test of economic effects on mortality outcomes, we estimate a series of models of the form

$$\Delta M_g = \beta_0 + \beta_1 \widehat{\Delta lnE_g} + \beta_2 Y_g + u_g \quad (2)$$

where ΔM_g is the change between 1990 and 2017 in (1) all-cause age-standardized death rate, (2) several cause-specific age-standardized death rates, or (3) all-cause YLL between 25 and 84. Given the recent evidence of worsening or stagnating mortality in middle age,(1,5) we focus the analysis of mortality rates on persons between the ages of 45 and 64. However, we consider YLL as a more comprehensive measure of adult mortality because it aggregates mortality across the adult life span and because of its close relationship to adult LE. Y_g represents per capita income at baseline, a measure of local economic resources that a community may be able to draw on to

buffer negative labor market shocks. Models are run separately by sex, race, and education group.

To the extent that poor economic conditions negatively affect health, we expect the coefficient β_1 to be negative. If, as we expect, those with less education are less able to adapt to poor or changing economic conditions, we expect these effects to be larger in magnitude than the effects on those with higher levels of education.

There are a few things that are important to note about equation (2). Both the key explanatory variable and the dependent variables represent changes over a 37 and a 27-year span, respectively. If persistent changes in local economies have mortality effects, our measure will capture these effects over that period. However, this measure will miss some economic shifts that are geographically specific. For example, although total US auto manufacturing employment stayed roughly constant between 1980 and 2017, it fell by nearly 30 percent in the CZ including Flint, Michigan, as production shifted away from the big three American automakers and out of Southeast Michigan. This measure will also miss temporary economic changes. For example, employment in mining and oil and gas extraction experienced significant growth and decline between 1980 and 2017. Thus, while our measure captures the effects of widespread and long-term trends in economic forces, it will not capture every difference in an area's economic prospects. In addition, to the extent that the US is an integrated economy, we should expect economic shocks to one area of the country to spill over to other areas. Each of these factors leads us to expect that our variable will not fully capture the effect we are interested in. Despite these limitations, we adopt this measure because it is plausibly exogenous to mortality and because it will reflect long-run rather than short-run changes in economic opportunities.

Standard regression inference assumes independence of error terms,u_g, between observations. However, in the case of health and mortality, which can be influenced by environmental conditions that span political boundaries and local economic conditions, which may be correlated because of shared economic histories, we may be concerned that observations in our data may be spatially correlated. The fear is that such correlations will tend to bias standard errors downward and thereby overstate the precision of coefficient estimates.(46) The aggregation of county-level data into CZ data is an attempt to reduce some of these economic and environmental spillovers, but some may remain. To address this issue, we correct standard error estimates, allowing correlations to be inversely related to the distance between CZ centroids.(47,48) To demonstrate the sensitivity of our results to geographic correlations, we estimate models using alternative assumptions about the distances over which such spillovers may occur (see the appendix).

The role behavioral and other risk factors

We use two approaches to assess the roles of health behaviors like illicit drug use, smoking, and drinking and other risk factors such as obesity and lack of access to medical care in explaining the connection between economic conditions and mortality. In the case of illicit drug use, death certificates can be used to directly identify deaths due to opioid and other drug poisoning. Under the important, but common assumption that patterns of drug overdose deaths reflect patterns of drug use, the coefficient β_1 from equation (2) when the measure M is drug overdose death rates compared to the same coefficient on the total death rate provides an estimate of the share of economically related mortality differences that are mediated through drug use.. To the extent that drug overdose death rates understate the impact of drug use on mortality, however, this approach

may underestimate the role of drug use as a mediator.(49) In related work, we explore the sensitivity of our results to our simplifying assumption.

For the other risk factors—smoking, heavy drinking, obesity, and lack of access to medical care—where a one-to-one match between the risk factor and a cause of death does not exist, we first estimate the relationship between economic conditions and those factors. The prevalence rates of current smoking, heavy drinking and obesity are estimated using data collected by the Behavioral Risk Factor Surveillance System (BRFSS). Each state conducts a BRFSS survey annually and each state sample is designed to be representative of the state population. While some content of the survey varies from state to state and from year to year, questions about smoking, drinking height and weight are standardized across all states. Data on smoking, drinking, height and weight was available in all states beginning in 1994, which differs slightly from the period over which we measure mortality change (1990-2017). Heavy drinking is defined as consuming more than two alcoholic drinks per day on average for men and more than 1 drink per day for women. Obesity is defined as having a body mass index (BMI = weight in kilograms divided the square of height in meters) greater than or equal to 30.0.

Using pooled individual-level data over all years, we first estimated linear time (t) trends ($Z_{isjt} = \delta_{0sj} + \delta_{1sj} t_{sjt} + e_{isjt}$) in these prevalence rates by state (s), for each demographic group (j) defined by sex, race, age group, and education. Depending on the case, Z is a 0/1 variable indicating the individual is a current smoker, a heavy drinker or has obesity. We then estimate the relationship between economic conditions and these behaviors for each demographic group by estimating models of the form

$$\widehat{\delta_{1sJ}} = \gamma_{0j} + \gamma_{1j}\widehat{\Delta \ln E_g} + \gamma_3 Y_g + e_{gj} \quad (3)$$

The behavioral change in each demographic group related to economic conditions is then represented by the parameter estimates $\widehat{\gamma_{1j}}$ obtained from these models. Note that in these regressions the dependent variable varies at the state, not the CZ level. This was necessitated by the geographic information available in the BRFSS data. For this part of our analysis we could have aggregated our explanatory variables up to the state level and will do so in future versions of this paper. Standard errors are estimated clustering at the state level.

To measure access to health care services, we use the share of people in each demographic group reporting in the ACS that they had health insurance in each year. We use a similar approach to predict the annual change in insurance. Health insurance information was collected beginning in 2008, so the estimated changes in coverage largely measure changes induced by the Affordable Care Act (ACA), which increased access to community rated individual insurance (marketplace) coverage and, more significantly, to Medicaid in states that elected to expand eligibility to that program. We first estimated the annual change in insurance coverage between 2008 and 2017 for each commuting zone and demographic group ($\widehat{\delta_{1gj}}$) and then estimated the relationship between economic conditions and the rate of health insurance coverage using a model similar to equation (3). The 2008-2017 period is regrettably defined over a much shorter time interval than our explanatory variable. In future versions of this paper we will use the CPS and the NHIS to explore a longer time series (19802017 for the CPS and 1997-2017 for the NHIS).

Drawing on other research which has estimated the mortality consequences of obesity, smoking, heavy drinking and health insurance coverage, we then generated rough estimates of the share of the mortality changes induced by economic change that could be attributed to individual behavioral factors and access to health care. Estimates of the direct effect of risk

factors (smoking, heavy drinking, and obesity) were produced as part of a multi-cohort meta-analysis of risk factors for mortality.(50) This study of 1.7 million individuals linked survey with administrative death data and generated direct estimates of differential risk of death and years of life lost associated with smoking and obesity. Estimates of the effect of health insurance coverage on mortality were obtained from an analysis of survey data linked to death records exploiting policy variation in the implementation of the ACA.(51)

Preliminary Results

Table 1 presents average changes in age-standardized death rates for 45- to 64-year-olds as well as changes in YLL between ages 25 and 84 across all CZs included in the analysis. The numbers reported represent average changes across the CZs we use, weighted by total population (by race and sex) at baseline, not changes in aggregate rates. Results are presented by sex, race, education, and, for the age-standardized death rates, by cause. Before stratifying by education, we find declines in all-cause mortality for all four race/sex groups, with the largest declines for Black males (-565 deaths per 100,000) and the smallest declines for white females (-51 deaths per 100,000). Across all groups, however, these improvements are concentrated among the top 75 percent of the education distribution, with Black and white women in the bottom quartile of the education distribution showing small increases in death rates.

Our estimates for YLL mirror our overall results for death rates, with the Black male and female populations exhibiting the largest overall changes and with the declines being concentrated among the better educated. Overall, Black males experienced a 3.5-year decrease in YLL between ages 25 and 84. In contrast, Black women experienced a decline of 1.6 years, compared with declines of 1.0 years and 0.2 years among White males and females, respectively. Among those in the top 75 percent of the education distribution for each group, YLL for Black

males and females decreased by 4.2 years and 2.0 years, respectively, compared with a decline of 0.9 years for Black males with low education and an increase of 0.1 years for Black females with low education. White females with low education had an increase of 1.1 YLL.

When we calculate overall changes in cause-specific death rates, we find declines in cancer mortality for every group, though, again, magnitudes are larger for the better educated. Cardiac and metabolic (e.g., heart disease and diabetes) death rates also declined for all groups, with the differences across education groups often being modest. Consistent with other literature, we find statistically significant increases in death rates for substance use and mental health-related causes for most groups, with increases in opioid overdose deaths accounting for a large share of that total. Mortality measures that include individuals over the age of 65, such as the YLL measure we use, are less influenced by these factors.(12)

Next, leveraging economic variation among CZs, Table 2 presents estimates of the effect of economic distress on age-standardized death rates. Focusing first on the results for all education groups combined, as hypothesized, CZs where we would have predicted greater declines in employment based on their industrial composition in 1980, saw smaller mortality improvements than those where the industrial mix was more favorable. However, these effects are quite modest, and in most cases, the 95 percent confidence intervals include 0's. In contrast, for the low-education groups, we find that CZs with greater predicted employment declines showed noticeably worse mortality trends than areas with less economic distress. For example, for the low-education groups, we find that a decrease of 10 percentage points in predicted employment growth over the period led to a relative *increase* in mortality of 79.4 deaths per 100,000 among white males, 73.7 among Black males, 52.9 among white females, and 28.7 among Black females. For Black men and women, the estimated effects of the Bartik instrument

on age-standardized death rates are substantially less precise than they are for white men and women. For example, the standard errors on all-cause death rates for Black populations are roughly two to three times those for white populations. This is as we would have expected since the number of CZs that support analysis of Black mortality is less than half the number available for whites, and Black population sizes in these CZs are smaller as well.

When we look at the effect of economic conditions by cause of death, we find that the largest contributors to the overall relationship between economic distress and mortality in mid-life among the less well-educated are cancer, cardiovascular and metabolic diseases, and diseases of other internal body systems (e.g., respiratory, digestive, and nervous systems). For example, a 10-percentage point improvement in predicted employment growth was associated with 26.9 fewer cancer deaths per 100,000 for white males, 35.1 fewer for Black males, 19.7 fewer for white females, and 20.3 fewer for Black females. In contrast, we found mortality due to mental health, suicide, or substance abuse, including opioid overdose, did not contribute importantly to the association between economic decline and mortality.

We estimated similar models for YLL to all causes of death, with results presented in Table 3. For white men and women and Black men overall, a 10-percentage point drop in predicted employment growth was associated with an increase in years YLL of 0.2 years, with the estimated effects for Black women being somewhat smaller. Again, estimated effects were largest for those with low education, where a 10-percentage point improvement in economic conditions predicted reductions in YLL between 0.33 (Black females) and 0.64 (white males) years.

To aid in the interpretation of estimated coefficients on economic conditions, we also calculated simulated differences in the outcomes (change in death rates or YLL between 1990

and 2017) attributable to differences in the average economic conditions between the bottom quartile (worst economic conditions) and top quartile (best economic conditions) of CZs ranked by predicted employment growth. Figure 2 shows the difference in predicted YLL for those living in a bottom quartile CZ if the economic conditions were the same as those experienced in the top quartile CZs. That is, for each sex/race/education group we calculate $\hat{\beta}_1 * (\overline{\Delta lnE_{gQ1}} - \overline{\Delta lnE_{gQ4}})$. Without stratifying by education, the advantage of living in a CZ in the top quartile of predicted employment growth is approximately 0.6 years for white males, white females, and black females, and 0.8 years for Black males. Economic differences in longevity are considerably greater for those in the bottom quartile of the education distribution, ranging from an additional 1.34 YLL for black females to 1.91 YLL for white males in more economically distressed areas than in less distressed areas.

Because our analysis relies on geographic variation in economic conditions to identify the relationship between stress and mortality patterns, economically motivated migration represents a potential threat to the interpretation of our results if that migration is selective on health. If selective migration biases our estimates, we expect bias to be more severe in CZs that saw population declines. In the appendix we report results from excluding CZs with either large population declines (of 10%) or any population declines. The effects of such exclusions on our estimates are modest.

Obesity, health behaviors and access to medical care as mediators of economic conditions

For a risk factor to have a meaningful role as mediating economic conditions, there would need to be a discernible correlation between the risk factor and economic conditions. In table 4, we report our estimates of equation 3. These estimates are reported as the percentage point changes in each risk factor over the period 1990-2017 predicted by a 10-percentage point

increase in expected employment. Since our data on health behaviors and access to medicate care do not span the period, we use linear extrapolation to estimate these trends. In the future we will improve on these crude methods.

Among the factors there is little evidence of a strong relationship between heavy drinking and economic conditions. While effects are in the expected direction, with better economic conditions predicting declines in heavy drinking, we cannot rule out the possibility that these effects are zero. Smoking, however, at least among white women, does seem to decrease more in areas with better economic conditions. For example, the share of white women ages 25-84 declined by 2.5 percentage points for every 10-percentage point improvement in employment growth. In general, these declines seem to be larger among those with less education.

The relationship between economic conditions and insurance coverage appears strongest for white men and women, where coefficients are uniformly positive. These effects are considerably larger among those with less education. Among Black men and women, while these correlations are positive for those with less education, they are not for those with more education, and the correlations are not statistically significant.

To illustrate the degree to which changes in smoking, drinking, obesity and insurance might explain the relationship between economic conditions and mortality, we first take direct estimates of the mortality effects of each of those risk factors and multiply them by the coefficients in table 4 to obtain the change in mortality predicted by the observed magnitude of behavioral changes linked to economic conditions. We can then compare these predicted values with the results reported in tables 2 and 3. For example, as shown in their Figure 6, Stringhini and colleagues(50) found that smoking reduced person years lived by 4 years among women. If, as we estimate, a 10-percentage point change in expected employment results in a 0.29

percentage point change in white women's smoking, we predict that the smoking mediated effect on YLL of that 10-percentage point economic improvement is to decrease YLL by 0.016 years (=4.0*0.0029). Since the total estimate of the economic effect was a reduction of 0.18 YLL, we conclude that the contribution of smoking behavior to that effect is on the order of 8% (0.016/0.18). While the behavioral link to economic conditions was not statistically significant, a similar calculation finds that the share of the total economic effect for white men is roughly 4%. For Black men and women, the coefficients in table 4 are larger, but not as precisely estimated. Taken at face value, however, these estimates suggest that 19% of the economic effect for Black men might be mediated through smoking, as is 11% of the total economic effect for Black women. Similar calculations of the contributions of obesity and drinking to the economic effects suggest considerably smaller shares might be explained by these pathways. We used estimates by Miller, Johnson, and Wherry(51) to similarly calculate the share of the economic effects attributable to improved insurance coverage in areas with less economic distress and found that insurance as a mediating variable could explain between 1 and 2 percent of the total impact of economic conditions on years of life lost. (Full results of these calculations are reported in the appendix)

Finally, in the case of opioid overdose there is no evidence for Blacks that opioid related deaths contribute to the patterns of death related to economic stagnation. In fact, none of the coefficients in table 2 are significant and they are all of the opposite than expected sign if economic stagnation were increasing illicit drug use and related mortality. For whites, coefficients are negative, as expected, but when there is an overall effect of economic stagnation on mortality rates, we estimate that the share mediated through opioid overdose is between 3 and 6 percent.

To sum up, our, admittedly crude estimates, suggest that neither health behaviors, nor access to medical care can explain the association we find between economic stagnation and mortality, suggesting an important direct role for the direct effects of stress.

Discussion

To be added.

Figure 1. Bartik values, by commuting zone

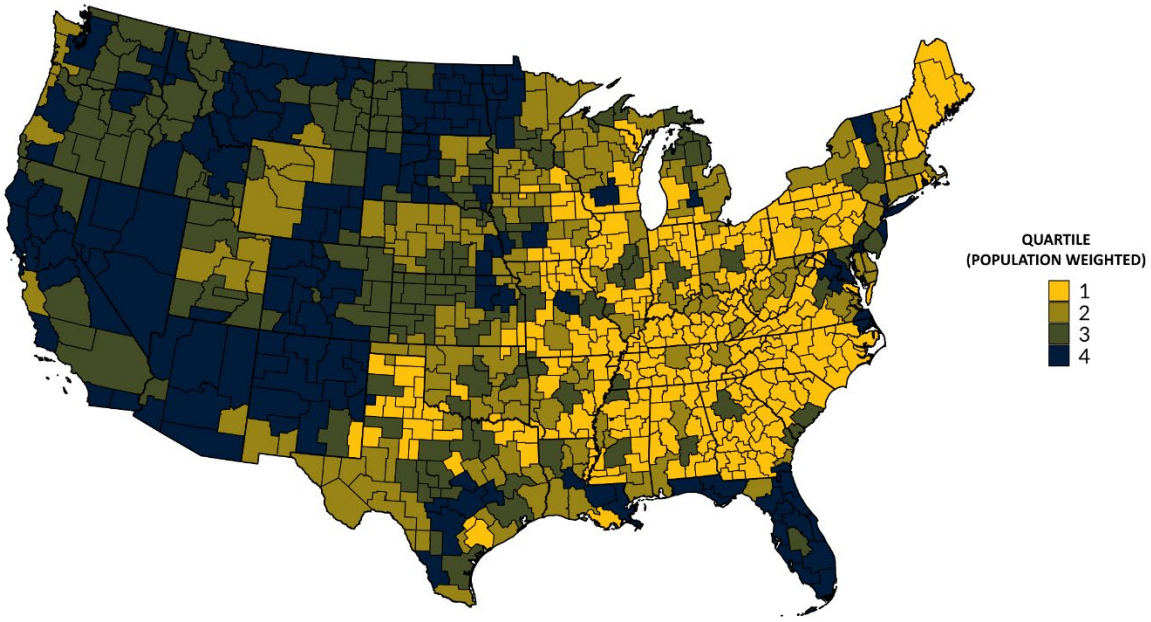
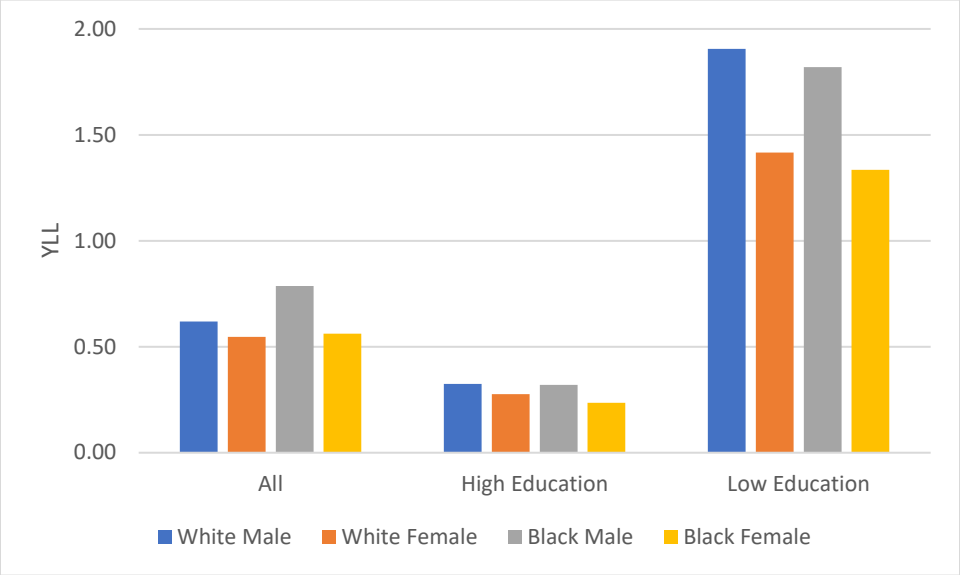


Figure 2. Difference in predicted YLL from age 25 to 84 between highest quartile of economic distress and lowest quartile



Notes: Authors' calculations are based on data from the United States decennial census, the American Community Survey (ACS),(31) and National Vital Statistics System.(44) Death counts for 1990 and 2017 are based on vital statistics data averaged over 1989–1991 and 2016–2018, respectively. Population measures for these periods were based on the 1990 Decennial Census and the 5-year ACS file covering 2015–2019. Calculations weighted by commuting zone population by race, sex, age, and education in 1990.

Table 1. Average change in mortality measures between 1990 and 2017

	WHITE MALE			BLACK MALE			WHITE FEMALE			BLACK FEMALE		
	Total	High Ed	Low Ed	Total	High Ed	Low Ed	Total	High Ed	Low Ed	Total	High Ed	Low Ed
Age-standardized death rates per 100,000, age 45-64												
All Causes	-158.8	-232.9	-73.1	-564.8	-714.5	-131.0	-51.3	-98.2	41.5	-205.7	-284.9	35.3
Infectious and parasitic diseases	-3.2	-8.0	14.5	-19.6	-29.4	21.9	7.4	5.0	15.0	10.1	4.7	30.1
Cancers	-115.8	-130.8	-110.2	-276.6	-311.3	-193.2	-85.5	-98.4	-53.6	-98.8	-121.7	-16.5
Cardio and metabolic diseases	-131.3	-147.1	-146.2	-231.8	-285.3	-80.7	-41.8	-46.8	-58.9	-147.4	-171.6	-104.4
Substance use and mental health	47.3	33.0	87.5	18.8	1.4	69.2	28.3	20.4	55.9	13.7	7.1	35.4
Opioid drug deaths	28.2	20.2	57.5	50.9	39.9	102.5	16.9	12.7	32.8	18.0	14.2	35.1
Suicide	11.0	9.2	14.6	-1.4	-2.2	2.2	2.9	2.4	4.7	-0.2	-0.3	0.3
Other body systems	37.9	20.2	68.0	-23.0	-43.8	47.4	35.6	20.9	69.4	17.8	3.3	71.5
Other causes of death	6.3	-0.2	13.2	-32.6	-46.2	4.3	4.7	0.7	13.8	-1.1	-6.7	19.2
YLL, age 25-84	-1.0	-1.5	-0.1	-3.5	-4.2	-0.9	-0.2	-0.6	1.1	-1.6	-2.0	0.1
N (Commuting zones)		707			311			707			311	

Notes: Authors' calculations based on data from the United States Decennial Census, the American Community Survey(ACS),(31) and National Vital Statistics System.(44) Death counts for 1990 and 2017 are based on vital statistics data averaged over 1989–1991 and 2016–2018, respectively. Population measures for these periods were based on the 1990 Decennial Census and the 5-year ACS file covering 2015–2019. White and Black populations exclude persons of Hispanic ethnicity. The "High Ed" group includes persons in the top three quartiles of the education distribution by sex, race, and birth cohort. The "Low Ed" group includes those in the bottom quartile of the education distribution. Age-standardized death rates used the national age distribution of combined non-Hispanic white and Black populations in 1990. All calculations were weighted by commuting zone populations by race, sex, age, and education as of 1990.

Table 2. Effect of 10 percentage point increase in predicted employment rate on change in age-standardized death rates at ages 45-64 between 1990 and 2017

	WHITE MALE				BLACK MALE				WHITE FEMALE				BLACK FEMALE			
	Total	High Ed	Low Ed	P-val	Total	High Ed	Low Ed	P-val	Total	High Ed	Low Ed	P-val	Total	High Ed	Low Ed	P-val
All causes	-11.1	-0.5	-79.4	0.00	-20.3	-2.1	-73.7	0.00	-18.8	-10.6	-52.9	0.00	-12.9	-2.9	-28.7	0.36
Standard error	*7.4*	*6.8*	*14.8*		*18.8*	*18.6*	*28.0*		*5.1*	*4.6*	*9.8*		*9.6*	*9.6*	*28.1*	
Infectious and parasitic diseases	-5.7	-5.5	-3.1	0.07	-8.4	-6.9	-13.2	0.06	-1.2	-0.8	-2.2	0.01	-1.2	-0.6	-3.9	0.05
	1.6	*1.7*	*1.2*		*2.7*	*3.1*	*3.6*		*0.3*	*0.3*	*0.6*		*0.6*	*0.7*	*1.6*	
Cancers	-3.6	0.4	-26.9	0.00	-10.3	-4.6	-35.1	0.02	-7.3	-4.2	-19.7	0.00	-7.2	-4.7	-20.3	0.04
	1.8	*1.8*	*4.6*		*4.8*	*4.5*	*14.0*		*1.7*	*1.7*	*4.1*		*2.8*	*2.9*	*8.0*	
Cardio and metabolic diseases	5.5	8.8	-23.4	0.00	3.9	13.8	-19.7	0.00	-0.1	0.4	-8.6	0.00	1.8	5.8	11.2	0.81
	2.7	*2.7*	*5.7*		*6.8*	*6.3*	*10.7*		*1.8*	*1.7*	*3.6*		*4.5*	*4.7*	*22.1*	
Substance use and mental health	-0.5	-0.7	-0.3	0.85	9.0	7.9	12.0	0.61	-1.3	-0.6	-1.8	0.21	2.5	2.6	1.1	0.59
	1.9	*1.6*	*3.3*		*4.9*	*4.4*	*10.5*		*0.7*	*0.6*	*1.1*		*1.9*	*1.5*	*4.0*	
Opioid drug deaths	-2.5	-1.4	-3.6	0.13	2.6	1.5	8.6	0.19	-1.1	-0.3	-2.3	0.01	0.8	0.5	1.8	0.38
	1.0	*0.7*	*2.0*		*4.1*	*3.1*	*8.4*		*0.5*	*0.3*	*1.0*		*1.3*	*1.0*	*2.4*	
Suicide	0.1	-0.2	1.0	0.07	0.5	0.5	0.9	0.72	0.0	0.1	-0.3	0.18	-0.1	-0.1	0.1	0.65
	0.4	*0.4*	*0.7*		*0.4*	*0.5*	*0.9*		*0.2*	*0.2*	*0.3*		*0.2*	*0.2*	*0.2*	
Other body systems	-6.0	-3.1	-18.7	0.00	-11.7	-9.1	-15.8	0.19	-7.7	-4.9	-17.0	0.00	-9.4	-7.6	-14.2	0.04
	1.8	*1.5*	*3.6*		*3.1*	*2.9*	*5.9*		*1.6*	*1.3*	*3.2*		*2.5*	*2.3*	*4.2*	
Other causes of death	-0.8	-0.4	-7.0	0.00	-2.8	-3.1	-1.9	0.75	-1.1	-0.5	-3.5	0.00	0.7	1.4	-2.6	0.07
	0.8	*0.7*	*1.8*		*2.6*	*2.8*	*3.9*		*0.4*	*0.4*	*1.1*		*1.0*	*1.1*	*2.1*	
N (Commuting zones)		707				311				707				311		

Notes: Authors' calculations based on data from the United States Decennial Census, the American Community Survey (ACS),(31) and National Vital Statistics System.(44) Death counts for 1990 and 2017 are based on vital statistics data averaged over 1989–1991 and 2016–2018, respectively. Population measures for these periods were based on the 1990 Decennial Census and the 5-year ACS file covering 2015–2019. White and Black populations exclude persons of Hispanic ethnicity. The "High Ed" group includes persons in the top three quartiles of the education distribution by sex, race, and birth cohort. The "Low Ed" group includes those in the bottom quartile of the education distribution. P represents the p-value on the two-tailed test that the coefficients of high- and low-education groups in each race and sex category are equal. Age-standardized death rates used the national age distribution of combined non-Hispanic white and Black populations in 1990. Calculations are weighted by commuting zone population by race, sex, age, and education as of 1990. Standard errors were corrected for spatial correlation, allowing for correlations between commuting zones with geographic centroids within 100 km of one another.(47) We also report P-values for the test of the equality of coefficients on the Bartik variable between the high and low education groups in the 4, 8, 12 and 16th column of the table .

Table 3. Effect of 10 percentage point increase in predicted employment rate on change in YLL at ages 25 to 84 between 1990 and 2017

	WHITE MALE				BLACK MALE				WHITE FEMALE				BLACK FEMALE			
	Total	**High Ed**	**Low Ed**	**P-val**	**Total**	**High Ed**	**Low Ed**	**P-val**	**Total**	**High Ed**	**Low Ed**	**P-val**	**Total**	**High Ed**	**Low Ed**	**P-val**
YLL, age 25-84	-0.21	-0.11	-0.64	0.00	-0.20	-0.08	-0.46	0.00	-0.18	-0.09	-0.48	0.00	-0.14	-0.06	-0.33	0.00
	0.07	*0.06*	*0.12*		*0.13*	*0.13*	*0.17*		*0.04*	*0.03*	*0.08*		*0.07*	*0.06*	*0.12*	
N (Commuting zones)		707				311				707				311		

Notes: Authors' calculations based on data from the United States Decennial Census, the American Community Survey (ACS),(31) and National Vital Statistics System.(44) Death counts for 1990 and 2017 are based on vital statistics data averaged over 1989–1991 and 2016–2018, respectively. Population measures for these periods were based on the 1990 Decennial Census and the 5-year ACS file covering 2015–2019. White and Black populations exclude persons of Hispanic ethnicity. The "High Ed" group includes persons in the top three quartiles of the education distribution by sex, race, and birth cohort. The "Low Ed" group includes those in the bottom quartile of the education distribution. P represents the p-value on the two-tailed test that the coefficients of high- and low-education groups in each race and sex category are equal. Calculations are weighted by commuting zone population by race, sex, age, and education as of 1990. Standard errors were corrected for spatial correlation, allowing for correlations between commuting zones with geographic centroids within 100 km of one another.(47)

Table 4. Effect of 10 percentage point increase in Bartik coefficient on mortality risk factors for the period 1990-2017

	White Male			Black Male			White Female			Black Female		
	Total	**High Ed**	**Low Ed**	**Total**	**High Ed**	**Low Ed**	**Total**	**High Ed**	**Low Ed**	**Total**	**High Ed**	**Low Ed**
Age 45-64												
Current Smoking	-0.0061	-0.0066	-0.0051	-0.0063	-0.0052	-0.0154	-0.0121	-0.0112	-0.0152	-0.0064	-0.0051	-0.0063
(standard error)	*0.0039*	*0.0036*	*0.0039*	*0.0058*	*0.0064*	*0.0091*	*0.0054*	*0.0049*	*0.0043*	*0.0027*	*0.0028*	*0.0046*
Heavy Drinking	-0.0047	-0.0049	-0.0037	0.0001	-0.0003	0.0041	-0.0016	-0.0011	-0.0011	-0.0021	-0.0028	0.0023
	0.0032	*0.0036*	*0.0036*	*0.0043*	*0.0043*	*0.0050*	*0.0022*	*0.0023*	*0.0022*	*0.0035*	*0.0030*	*0.0070*
Obesity	-0.0112	-0.0117	-0.0109	0.0040	0.0057	-0.0120	-0.0065	-0.0072	-0.0051	0.0002	0.0014	-0.0083
	0.0031	*0.0035*	*0.0035*	*0.0036*	*0.0034*	*0.0086*	*0.0032*	*0.0031*	*0.0051*	*0.0056*	*0.0057*	*0.0085*
Health Insurance	0.0136	0.0132	0.0274	-0.0038	-0.0053	0.0110	0.0040	0.0065	0.0108	-0.0039	-0.0085	0.0138
	0.0035	*0.0032*	*0.0057*	*0.0089*	*0.0088*	*0.0130*	*0.0037*	*0.0032*	*0.0063*	*0.0066*	*0.0064*	*0.0131*
Age 25-84												
Current Smoking	*-0.0015*	*-0.0018*	*-0.0020*	*-0.0029*	*-0.0042*	*-0.0047*	*-0.0087*	*-0.0077*	*-0.0110*	*-0.0039*	*-0.0025*	*-0.0067*
(standard error)	*0.0028*	*0.0025*	*0.0033*	*0.0041*	*0.0038*	*0.099*	*0.0034*	*0.0031*	*0.0045*	*0.0027*	*0.0018*	*0.0044*
Heavy Drinking	*-0.0039*	*-0.0041*	*-0.0029*	*-0.0030*	*-0.0041*	*0.0022*	*-0.0021*	*-0.0020*	*-0.0016*	*-0.0001*	*-0.0006*	*0.0019*
	0.0026	*0.0029*	*0.0027*	*0.0025*	*0.0017*	*0.0075*	*0.0020*	*0.0021*	*0.0023*	*0.0015*	*0.0014*	*0.0026*
Obesity	*-0.0077*	*-0.0082*	*-0.0048*	*0.0029*	*0.0028*	*0.0009*	*-0.0069*	*-0.0075*	*-0.0011*	*-0.0079*	*-0.0084*	*-0.0057*
	0.0029	*0.0032*	*0.0028*	*0.0031*	*0.0033*	*0.0062*	*0.0024*	*0.0027*	*0.0018*	*0.0057*	*0.0060*	*0.0046*
Health Insurance	*0.0129*	*0.0121*	*0.0345*	*-0.0011*	*-0.0017*	*0.0074*	*0.0064*	*0.0079*	*0.0197*	*-0.0029*	*-0.0054*	*0.0093*
	0.0034	*0.0030*	*0.0056*	*0.0077*	*0.0070*	*0.0116*	*0.0032*	*0.0028*	*0.0057*	*0.0056*	*0.0053*	*0.0091*

Notes: For current smoking, heavy drinking, and obesity, we use BRFSS data from 1994 to 2017. For the health insurance variable, we use the ACS from 2008 to 2018. Regressions control for 1990 per capita income in the commuting zone, by race.

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