LIVES VERSUS LIVELIHOODS: THE IMPACT OF THE GREAT RECESSION ON MORTALITY AND WELFARE*

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We leverage spatial variation in the severity of the Great Recession across the United States to examine its impact on mortality and explore the quantitative implications. We estimate that an increase in the unemployment rate of the magnitude of the Great Recession reduces the average annual age-adjusted mortality rate by 2.3%, with effects persisting for at least 10 years. Mortality reductions appear across causes of death and are concentrated in the half of the population with a high school degree or less. We estimate similar percentage reductions in mortality at all ages, with declines in elderly mortality thus responsible for about three-quarters of the total mortality reduction. Recession-induced mortality declines are driven primarily by external effects of reduced aggregate economic activity on mortality, and reduced air pollution appears to be a quantitatively important mechanism. Incorporating our estimates of procyclical mortality into a standard macroeconomic framework substantially reduces the welfare costs of recessions, particularly for people with less education, and at older ages. *JEL codes:* E3, I1.

I. INTRODUCTION

Recessions damage economies and prompt substantial government intervention. The macroeconomics literature has

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calibrated their welfare costs, focusing on their effect on the level and volatility of consumption (e.g., Lucas 1987, 2003; Krebs 2007; Krusell et al. 2009). Recessions may also have important effects on health. Indeed, an empirical literature in health economics has found mortality to be procyclical in the 1970s and 1980s (e.g., Ruhm 2000; Stevens et al. 2015), although perhaps less so in the subsequent two decades (Ruhm 2015). Incorporating the mortality effects of recessions could have important implications for their welfare consequences, both overall and across demographic groups.

We consider this possibility in the context of the 2007–2009 Great Recession in the United States. At the time, the Great Recession produced the largest decline in U.S. employment since the Great Depression. Following Yagan (2019), we leverage spatial variation in the economic severity of the Great Recession across the United States to provide new empirical evidence on the impact of recessions on mortality and to explore implications for the welfare consequences of recessions.

We find that the Great Recession substantially reduced mortality. For every 1 percentage point increase in a commuting zone's (CZ) unemployment rate between 2007 and 2009, its age-adjusted mortality rate fell by 0.5%. These mortality reductions appear immediately and persist for at least 10 years, although the point estimates become less precise and statistically insignificant over time. Since the average national unemployment rate increased by 4.6 percentage points between 2007 and 2009, our estimates imply that an increase in the unemployment rate of the magnitude of the Great Recession reduces the average, annual age-adjusted mortality rate by 2.3% for at least 10 years. These estimates imply that the Great Recession provided 1 in 25 55-year-olds with an extra year of life.

Recession-induced mortality declines are entirely concentrated among the half of the population with a high school diploma or less but are otherwise pervasive across demographic groups. They appear across many causes of death, including cardiovascular disease, motor vehicle accidents, liver disease, and suicide; no cause of death experiences a statistically significant increase in mortality, and we estimate a precise zero for cancer mortality, the second largest cause of death. We find similar percentage reductions in mortality rates across gender, race/ethnicity, and age groups. However, because mortality is so much higher among the elderly, about three-quarters of the over-

all mortality reduction comes from averted deaths among those ages 65 and over, roughly the same as their share of prerecession mortality. The single largest cause of death, cardiovascular mortality, accounted for about one-third of deaths in 2006 and about half of the estimated mortality declines.

Several pieces of evidence suggest that the primary driver of the mortality declines are externalities from reduced aggregate economic activity, holding constant own employment or consumption. First, averted deaths are concentrated in the elderly population—who experienced little if any direct income effects from the Great Recession–induced local labor market decline. Second, we find a quantitatively important role for a particular external channel—recession-induced declines in air pollution; like the mortality declines, recession-induced pollution declines persist throughout our study period and may be able to explain at least 20% and potentially all of the recession-induced mortality declines. By contrast, we find little evidence for other mechanisms discussed in the literature—a key direct (i.e., nonexternality) mechanism, whereby reduced labor market activity frees up time for beneficial health behaviors (as in Ruhm 2000, 2005) and two other external channels, the reduced spread of infectious disease (as in Adda 2016) and improved quality of nursing home care (as in Stevens et al. 2015).

The recession-induced mortality declines are quantitatively important for estimates of the welfare effects of recessions. Extending the Krebs (2007) model of the consumption-based welfare cost of facing a lifetime risk of recessions, we find that accounting for procyclical mortality substantially reduces the welfare cost of recessions. For example, accounting for procyclical mortality reduces the willingness to pay to avoid future recessions by more than half for a 45-year-old with a coefficient of relative risk aversion of two and a value of a statistical life-year of five times annual consumption. Willingness to pay to avoid future recessions declines even more dramatically at older ages. Viewed through the lens of our stylized macro model, recessions may even be welfare-improving for the elderly, who benefit from mortality reductions while exhibiting limited consumption responses to recessions. Last, endogenous mortality also has important distributional implications: because the mortality declines from recessions are concentrated entirely among those with a high school degree or less, endogenous mortality substantially mitigates—

and at older ages even reverses—the regressive nature of recessions that is found when focusing exclusively on consumption.

These findings come with some important caveats. First, our design will not pick up any aggregate effects of the Great Recession—for example, any nationwide mortality effects from the stock market collapse (see McInerney, Mellor, and Nicholas 2013), or any nationwide increase in malaise. Our estimates may be more applicable to the more "typical" local recessions studied in the literature than to aggregate, national downturns. Relatedly, our design does not fully capture impacts of the Great Recession that are spatially differentiated but not perfectly correlated with local labor market declines, such as declines in house prices, or declines in air pollution that may originate from declines in local labor markets but affect other areas due to wind patterns. Second, while the Great Recession helps identify the impact of local area recessions on mortality, those impacts may not generalize to other (particularly milder) recessions; that said, we do not find evidence of a nonlinear relationship between the size of the economic shock and the mortality decline. Third, our analysis focuses primarily on mortality effects, yet recessions may also have important morbidity impacts, particularly for those at younger ages with very low mortality. Our limited evidence indicates that the Great Recession also caused roughly equi-proportional morbidity reductions across ages, suggesting that our focus on mortality may underestimate the extent of recession-induced health improvements. Fourth, although we analyze the 10-year impact of the Great Recession shock, our analysis does not measure effects at even longer time horizons, which may be very different (see Schwandt and von Wachter 2020). Finally, while we view our welfare analysis as a useful way to benchmark the magnitude of our mortality estimates, the analysis falls far short of a comprehensive analysis of the welfare effects of recessions; it does not incorporate other potential channels for welfare impacts, such as reduced job satisfaction, subjective well-being, and public resources for education, or the career costs of recessions for new labor market entrants that have been highlighted in other work (e.g., Akerlof et al. 1988; Kahn 2010; Oreopoulos, von Wachter, and Heisz 2012; Jackson, Wigger, and Xiong 2021). These limitations notwithstanding, our article sheds new light on the existence, nature, and causes of recession-induced mortality declines, and suggests that recognition of the mortality effect of recessions

can have quantitatively important implications for their welfare consequences, both overall and across demographic groups.

This study extends the macroeconomics literature on the welfare cost of business cycles (e.g., Lucas 1987, 2003; Krebs 2007; Krusell et al. 2009) to incorporate our estimates of endogenous mortality over the business cycle. Our approach is in the spirit of existing work in macroeconomics that has incorporated secular improvements in health into welfare comparisons across countries and welfare analyses of economic growth within and across countries (e.g., Nordhaus 2002; Becker, Philipson, and Soares 2005; Murphy and Topel 2006; Hall and Jones 2007; Jones and Klenow 2016; Brouillette, Jones, and Klenow 2021). There has been relatively less attention to incorporating cyclical fluctuations in health into welfare analyses of business cycles.[1]

We also contribute to a much larger empirical literature on the relationship between the economy and health. A considerable body of evidence suggests that improvements in the economy are good for health, based on which one might expect that recessions increase mortality. There is a well-documented negative relationship between income and mortality within countries, across countries, and over time (e.g., Cutler, Deaton, and Lleras-Muney 2006; Costa 2015; Chetty et al. 2016; Cutler, Huang, and Lleras-Muney 2016), although the causal evidence of the impact of income on mortality is limited and mixed (Dobkin and Puller 2007; Evans and Moore 2012; Cesarini et al. 2016). There is also evidence that job loss increases mortality (Sullivan and von Wachter 2009), sustained reductions in economic prospects contribute to "deaths of despair" (Case and Deaton 2021), and counties exposed to greater job loss from trade liberalization with China experience both increases in fatal drug overdoses among the working-age population (Pierce and Schott 2020) and increased mortality of young men relative to young women (Autor, Dorn, and Hanson 2019).

The existing empirical work on the relationship between recessions and mortality raises questions about what to expect for the Great Recession. For the decades before the Great Recession, a series of papers starting with the influential work of Ruhm (2000) have documented a negative contemporaneous association

1. Two exceptions are Edwards (2009) who extends Lucas (1987) to allow for cyclical mortality, and Egan, Mulligan, and Philipson (2014), who contrast fluctuations in GDP to fluctuations in mortality-adjusted GDP. They reach different conclusions.

between local area unemployment rates and mortality in area-year panel data both in the United States (e.g., Ruhm 2000; Miller et al. 2009; Stevens et al. 2015), and in Canada and several European countries (Neumayer 2004; Granados 2005; Buchmueller, Jusot, and Grignon 2007; Ariizumi and Schirle 2012). However, in the decades before the Great Recession, the relationship between local unemployment and mortality weakened in the United States (McInerney and Mellor 2012; Ruhm 2015). Moreover, studying almost three dozen countries over 200 years, Cutler, Huang, and Lleras-Muney (2016) conclude that while small recessions are associated with reduced mortality, large recessions are associated with increased mortality.

Reinforcing the uncertainty about the impact of the Great Recession on mortality, the existing literature studying its effect on health has produced mixed results (Currie, Duque, and Garfinkel 2015; Currie and Tekin 2015; Strumpf et al. 2017; Seeman et al. 2018; Cutler and Sportiche 2022; Salinari and Benassi 2022; Lamba and Moffitt 2023). When we surveyed over 300 experts in spring 2023 on the impact of the Great Recession on the U.S. mortality rate, 50% of respondents predicted that the recession would increase mortality, and only 27% predicted a decrease; moreover, 93% of respondents provided a predicted impact on mortality larger than our (negative) point estimate, and 82% provided a prediction larger than the upper bound of our 95% confidence interval (Online Appendix A).

Our empirical approach follows Bartik (1991), Blanchard and Katz (1992), and especially Yagan (2019) in exploiting regional differences in exposure to a large, aggregate economic shock. We complement existing work, which analyzes the relationship between an area's mortality rate and its contemporaneous unemployment rate, by controlling for area and year fixed effects. Relative to this literature, we offer several innovations. First, we use a single, spatially differentiated shock, allowing us to examine the lag structure of the impact of the recession on mortality rather than assuming that any such effect is contemporaneous. Second, as emphasized by Arthi, Beach, and Hanlon (2022), a key limitation to the existing literature is the potential for contamination from unobserved migration in response to recessions. For some of our analyses, we leverage individual-level panel data in which we can instrument for current location with prerecession location and confirm that our results are not spuriously driven by endogenous migration or unmeasured changes in the local population.

Third, our empirical approach helps isolate the causal effects of recessions from potential confounding factors that could increase the local unemployment rate and also directly affect health, such as increased access to or generosity of disability insurance or unemployment insurance.

The rest of the article proceeds as follows. Section II presents our data and empirical strategy. Section III presents our empirical estimates of mortality effects. Section IV investigates potential mechanisms behind these results. Section V explores implications for the welfare analysis of recessions. Section VI provides a brief conclusion.

II. Data and Empirical Strategy

II.A. Data

We restrict our analysis to people in the 50 states and the District of Columbia from 2003 to 2016. Following Yagan (2019), we begin all of our analyses in 2003 to avoid contamination from the 2001–2002 recession. Our primary analysis is across CZs, which are a standard aggregation of counties that partition the United States into 741 areas designed to approximate local labor markets; we also perform some analyses at the county or state level. We briefly describe our main data sources here, and Online Appendix B provides more detail on the underlying data sources and variable construction.

1. *Mortality.* We use two major sources of mortality data. First, following Ruhm (2016), we construct mortality rates by combining death records from the restricted-use mortality microdata from the Centers for Disease Control and Prevention (CDC) on the universe of U.S. mortality events from 2003 to 2016 (National Center for Health Statistics 2023) with population data from the National Cancer Institute's Surveillance Epidemiology and End Results (SEER) program. For each decedent, we observe county of residence, exact date of death, cause of death, and demographic information including age in years, race, ethnicity, gender, and education. The population data provide annual, county-level population estimates by single year of age, race, ethnicity, and gender.

Second, we use mortality records from a 20% random sample of all Medicare enrollees aged 65+ in the United States from 2003

to 2016. The enrollee-level panel data contain information on ZIP code of residence each year, date of death (if deceased), and demographic variables such as race, ethnicity, gender, and annual enrollment in Medicaid (a proxy for low income). Unfortunately, we do not observe the cause of death. However, for the approximately three-quarters of the elderly who are enrolled in traditional Medicare, we observe detailed, annual information about their health care use—including doctor visits, hospital admissions, and nursing home stays—and any diagnoses with 1 of 20 chronic conditions in the past year, such as lung cancer, diabetes, or depression. We analyze two primary Medicare samples: a panel of 2003 Medicare enrollees ages 65–99 in 2003, and a repeated cross section of individuals ages 65–99 each year, often further restricted to individuals who were enrolled in traditional Medicare in the prior or current year.

The Medicare data offer several advantages over the CDC mortality data, albeit for the 65 and older population only. First, they provide a well-defined population denominator in which mortality can be directly observed. Observing mortality and the population denominator in the same data addresses the well-known challenge with most other U.S. mortality data in which the numerator (deaths) and the denominator (population) come from different data sets, creating concerns about consistency between the sources and potential misestimation of the denominator during intercensal years (Currie and Schwandt 2016). Second, the individual-level panel nature of the Medicare data allows us to define a cohort of individuals based on their initial location and follow them over time. Assigning individuals to their prerecession locations allows us to address a concern with many existing estimates of procyclical mortality that results may be confounded by endogenous migration in response to economic shocks (Blanchard and Katz 1992; Arthi, Beach, and Hanlon 2022). Third, we can use the (lagged) data on enrollee health conditions to analyze heterogeneous effects on mortality by health status, which is not recorded in the CDC data. Finally, we use the Medicare data to analyze the impact of the Great Recession on the consumption of health care and estimate heterogeneous effects by whether the individual lives in a nursing home.

2. *Economic Indicators.* We use publicly available local economic indicators to trace the Great Recession across areas and years from 2003 to 2016. We construct the CZ-year unemploy-

ment rate and employment-to-population (EPOP) ratio using data from the Bureau of Labor Statistics' Local Area Unemployment Statistics, and CZ-year real GDP per capita using data from the Bureau of Economic Analysis. For the subsample of counties for which it is available, we construct a CZ-level annual house price index from the Federal Housing Finance Agency's yearly House Price Index (HPI) public release. We obtain state-level annual data on total household expenditures on goods and services from the Personal Consumption Expenditures (PCE) surveys published by the Bureau of Economic Analysis; we use the PCE Index to adjust all expenditures to 2012 dollars and divide state-level annual expenditures by the SEER population data to obtain a measure of state-year real consumption per capita. We use data from the Current Population Survey to measure state-year earnings and income in the overall working-age population, as well as by education and age.

3. *Air Pollution.* We focus primarily on fine particulate matter (PM2.5), measured in micrograms per cubic meter ($\mu g/m^3$). Using granular, annual data on PM2.5 concentration from van Donkelaar et al. (2021), we construct county-year measures of PM2.5 that cover 99.3% of (population-weighted) counties in the United States. The authors generate estimates of PM2.5 by combining ground-level pollution monitor measurements from the EPA's Air Quality System (AQS) database with observations of visual occlusion from satellite images to produce estimates of PM2.5 for virtually the entire United States. We discuss these data—which have recently been used in several studies of pollution as an input or output (e.g., Jha and Nauze 2022; Gould et al. 2023; Molitor, Mullins, and White 2023)—in more depth in Online Appendix B.3. For counties that also have AQS measures of PM2.5, the van Donkelaar et al. (2021) and AQS measures produce similar findings (Online Appendix C.9).

4. *Other Outcomes.* We draw on several additional data sources to probe potential mechanisms behind our mortality findings and to explore impacts on non-mortality measures of health. First, we use data from the Behavioral Risk Factor Surveillance Survey (BRFSS) to examine self-reported health, health behaviors, and health insurance coverage at the state level (the finest geographic information available). Second, we use facility-level administrative data from annual certification inspections

of all nursing home facilities across the United States to measure nursing home staffing and other characteristics such as patient volume and composition. Third, we draw on restricted-use (state-level) data from the Health and Retirement Survey for 2002–2014—a nationally representative, biannual survey of older adults—to examine self-reported measures of formal and informal care received by individuals 65 and older.

II.B. Empirical Strategy

Our empirical strategy closely follows Yagan (2019), who exploits spatial variation in the effect of the Great Recession on local labor markets to study its long-term impacts on employment and earnings. Our main estimating equation is:

$$y_{ct} = \beta_t [SHOCK_c \times \mathbb{1}(Year_t)] + \alpha_c + \gamma_t + \varepsilon_{ct}, \tag{1}$$

where $SHOCK_c$ measures the economic impact of the Great Recession on area c, $\mathbb{1}(Year_t)$ is an indicator for year t, α_c and γ_t are area and year fixed effects, respectively, and ε_{ct} is the error term. We estimate equation (1) using OLS and cluster our standard errors at the local area c. The coefficients of interest are the β_t's; they measure effects on the outcome y_{ct} in year t across areas differentially affected by the Great Recession. Unless indicated otherwise, we omit the interaction with the shock variable in 2006 so that all β_t coefficients are relative to 2006. Because population varies greatly across areas in the United States (Online Appendix Figure A.1), we weight each area-year by its 2006 population, as in prior work examining effects of recessions on mortality (e.g., Ruhm 2000, 2015).

Also following this prior literature, we define our main outcome variable y_{ct} to be the log age-adjusted mortality rate in area c and year t.[2] For sufficiently low annual individual mortality rates, this specification is an approximation to a parametric individual-level survival model in which the individual's log odds of dying are given by the right side of equation (1). The mortality rate is defined as the share of the population in area c and

2. Specifically, we add one to the mortality rate to avoid taking logs of zeros, although in practice we never need to do so for the aggregate CZ-level analysis. Even when we disaggregate by cause of death or various demographics, mortality rates of zero are extremely rare. Our main results are very similar if we instead estimate a Poisson specification for age-adjusted mortality rates, as recommended by Chen and Roth (2024); see Online Appendix C.6.

year t at the beginning of year t who die during year t. In all of our analyses using the death certificate data (except those that disaggregate by age), we examine age-adjusted mortality rates, so that our analysis is not affected by different secular trends in mortality across age groups.[3]

We perform many analyses by subgroup, in which we estimate a fully saturated model:

$$(2)\quad y_{ctg} = \beta_{tg}[SHOCK_c \times \mathbb{1}(Year_t) \times \mathbb{1}(Group_g)] + \alpha_{cg} + \gamma_{tg} + \varepsilon_{ctg},$$

where y_{ctg} is an area-year-group outcome, $\mathbb{1}(Group_g)$ are indicators for subgroups, α_{cg} are area-group fixed effects, γ_{tg} are year-group fixed effects, and ε_{ctg} is the error term.

For these estimating equations, the key identifying assumption is that there are no shocks to mortality that coincide exactly with the timing of the Great Recession and are correlated with the size of the local area economic effect of the Great Recession. We investigate the plausibility of this assumption by examining pre-trends in the event-study results. Of course, finding similar mortality trends before the Great Recession in areas that are differentially affected does not guarantee that these areas would have been on similar trends in the absence of the Great Recession, and this assumption becomes less plausible the further out in time we go past the Great Recession.

1. *Measuring the Great Recession Shock.* Our empirical strategy relies on the large spatial variation in the economic impact of the Great Recession. This strategy has been previously leveraged to study the effect of the Great Recession on outcomes such as employment (Yagan 2019; Rinz 2022), time use (Aguiar, Hurst, and Karabarbounis 2013), consumption (Mian, Rao, and Sufi 2013), and educational attainment (Charles, Hurst, and Notowidigdo 2018). Following Yagan (2019), in our baseline specification we parameterize the impact of the Great Recession on area c (i.e., $SHOCK_c$) as the percentage point change in the CZ unemployment rate between 2007 and 2009. Thus β_t in equation (1) captures the percent change in the mortality rate in CZ c and

3. We calculate the age-adjusted mortality rate in a CZ by averaging over the mortality rate in each of 19 age bins in the CZ, weighting each age bin by the national share of the population in that age bin in 2000. This approach is in the spirit of Ruhm (2000), who controls for the share of the population in various age groups. The age bins are 0, 1–4, 5–9, and then every five-year age bin up through 80–84, with a final bin for 85+.

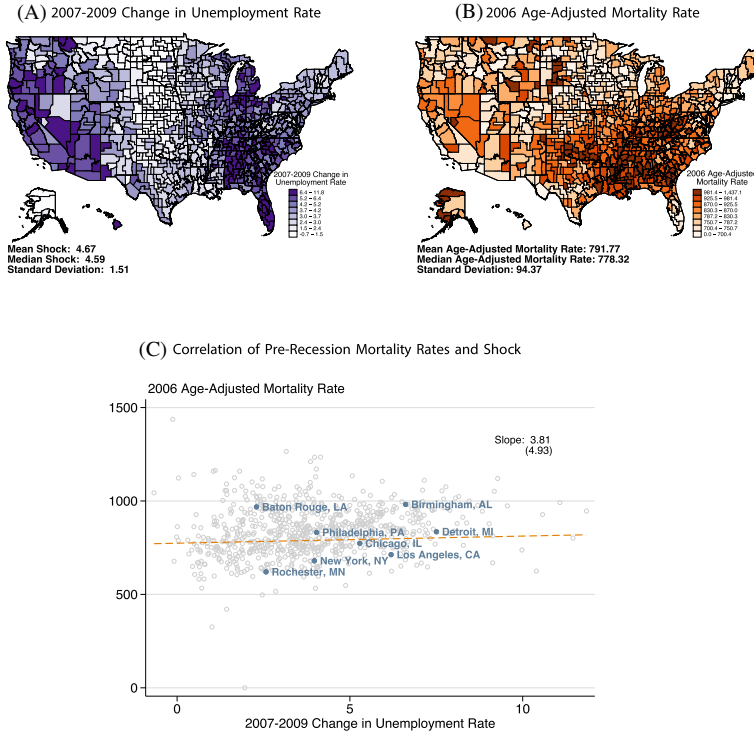

FIGURE I

Geographic Patterns and Correlation of Unemployment and Mortality

Panel A displays a heat map of the unemployment shock, that is, the change in CZ unemployment rates from 2007–2009, binned into octiles. Panel B displays a heatmap of 2006 CZ age-adjusted mortality rates per 100,000. The 2006 CZ population-weighted mean and standard deviation of the unemployment shock and mortality rate are reported in the lower left corner of each panel. Panel C displays a scatterplot of the 2006 age-adjusted CZ mortality rate against the 2007–2009 change in the CZ unemployment rate, with each circle representing one CZ. The linear fit between the 2006 mortality rate and the 2007–2009 change in the unemployment rate, weighted by the 2006 population, is plotted as a dashed orange line, with the slope and heteroskedasticity-robust standard error reported in the top right-hand corner of the figure. $N = 741$ CZs.

year t (relative to that CZ's 2006 mortality rate) associated with a 1 percentage point increase in the unemployment rate from 2007 to 2009 in that CZ.

Figure I, Panel A shows the spatial variation in this baseline measure of $SHOCK_c$. The median (population-weighted) CZ

experienced a 4.6 percentage point increase in the unemployment rate. Virtually every CZ in the country experienced an increase in unemployment between 2007 and 2009. Some areas were much harder hit than others: the bottom quartile of CZs saw an average 2.9 percentage point increase in the unemployment rate, compared to a 6.7 percentage point increase in the highest quartile. Especially hard-hit areas include the so-called sand states of Florida, Arizona, Nevada, and parts of California (where the prerecession housing and construction booms were concentrated) and Midwest manufacturing states such as Michigan, Indiana, and Ohio. By contrast, most of Texas, Oklahoma, Kansas, Nebraska, and the Dakotas were relatively unscathed.

Our use of the unemployment rate to parameterize the recession follows the existing literature analyzing the relationship between recessions and mortality (e.g., Ruhm 2000, 2003, 2005; Stevens et al. 2015). However, in practice, all recessions—including the Great Recession—are multifaceted shocks and can be parameterized in different ways. We examine four different measures: unemployment rate, EPOP ratio, log GDP per capita, and log house prices. The spatial variation in the 2007–2009 shock as measured by these variables is highly but imperfectly correlated (Online Appendix Figure A.2). In the national time series (Online Appendix Figure A.3) they all flatten out between 2006 and 2007 and then worsen through 2009; however, the national aggregate trends in 2010–2016 look fairly different across these indicators, which is why we consider other measures of the Great Recession besides the unemployment rate in Section III.

2. *Mortality Patterns Across Areas.* Figure I, Panel B documents the wide variation in age-adjusted mortality rates across CZs in 2006, immediately before the Great Recession. Mortality rates were particularly high in the Southeastern United States and low in the Western United States. Figure I, Panel C shows no correlation between the magnitude of the 2007–2009 Great Recession shock in each CZ and its 2006 (age-adjusted) mortality rate.

3. *Mortality Patterns Across Areas over Time.* To provide a preliminary look at how changes in mortality correlate with areas more or less hard hit by the Great Recession, Figure II plots age-adjusted mortality rates from 2003 through 2016 for the CZs in the lowest quartile of the 2007–2009 unemployment

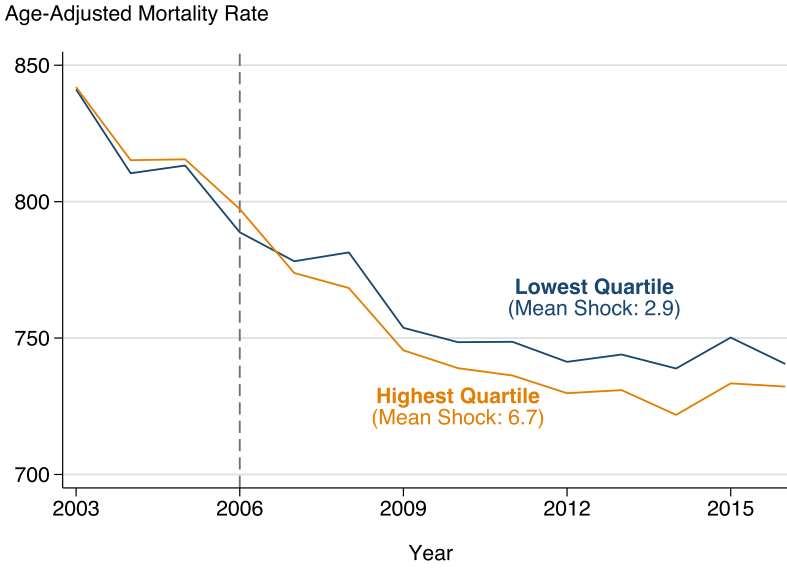

FIGURE II

Age-Adjusted Mortality Rate by Severity of the Shock

This figure displays trends in the (population-weighted) mean age-adjusted CZ mortality rate per 100,000 from 2003 to 2016. Mean mortality among CZs in the highest (population-weighted) quartile of the Great Recession unemployment shock is displayed in orange (light gray; color version available online); the mean among the lowest (population-weighted) quartile of CZs is displayed in blue (dark gray). Weights throughout are the 2006 CZ population. $N = 473$ CZs in total, 125 CZs in the top quartile, and 348 CZs in the bottom quartile.

shock (mean unemployment shock of 2.9 percentage points) and the CZs in the highest quartile (mean unemployment shock of 6.7 percentage points). Both exhibit decreasing mortality over the study period. Their mortality rates are indistinguishable in 2003; by 2006, the CZs that will be harder hit by the Great Recession have, if anything, experienced a relative increase in mortality. After 2006, there is an immediate and pronounced decline in age-adjusted mortality in the harder-hit CZs relative to the less hard-hit ones, creating a gap in age-adjusted mortality rates that persists through the end of the series in 2016.

The aggregate slowdown in mortality declines after the Great Recession shown in Figure II is an important reminder that our empirical strategy captures only differential mortality declines across local labor markets that are differentially affected by the

Great Recession. In other words, we are estimating the effect of differential local labor market shocks produced by the Great Recession, not the overall impact of the Great Recession. The slowdown in the aggregate mortality decline in Figure II could reflect changes in determinants of mortality unrelated to the Great Recession, such as the rate of progress in medical technologies, but it could also reflect aggregate effects of the recession on mortality that would not be captured by our empirical strategy. This limitation is the well-known "missing intercept" problem for macroeconomic counterfactuals.[4]

III. Mortality Effects of the Great Recession

We present estimated mortality effects overall and across different subpopulations and causes of death. After presenting initial event-study results, for most of the subsequent analyses we summarize the average event-study estimates for the 2007–2009 and 2010–2016 periods for ease of exposition; the underlying event studies are shown in Online Appendix D.

III.A. Overall Mortality Estimates

Figure III shows results from estimating equation (1) for log age-adjusted mortality, with the β_{2006} coefficient normalized to zero. Places harder hit by the Great Recession experienced an immediate and pronounced decline in log age-adjusted mortality, which then remained at this lower level for at least 10 years. The immediate impact of the Great Recession on mortality in 2007 is consistent with economic indicators also beginning to deteriorate in 2007 in harder-hit areas (Online Appendix Figure A.4). The slightly positive pre-trend in the mortality estimates from 2003 through 2006 (also visible in Figure II) indicates that before the Great Recession, areas that were harder hit were experiencing a slight relative increase in mortality. This opposite-signed pre-trend is consistent with our finding that recessions reduce

4. As one suggestive way to gauge how large this missing intercept might be, Online Appendix Figure A.5 plots a time series of nationwide log mortality and unemployment from 1969 to 2019, residualized on a linear time trend and then standardized. Online Appendix Table A.1 displays coefficients from a time-series regression of log mortality on unemployment from 1969 to 2019, controlling for either a linear or a quadratic time trend; these indicate an even larger negative relationship than we find below when exploiting the spatial variation in the impact of the Great Recession.

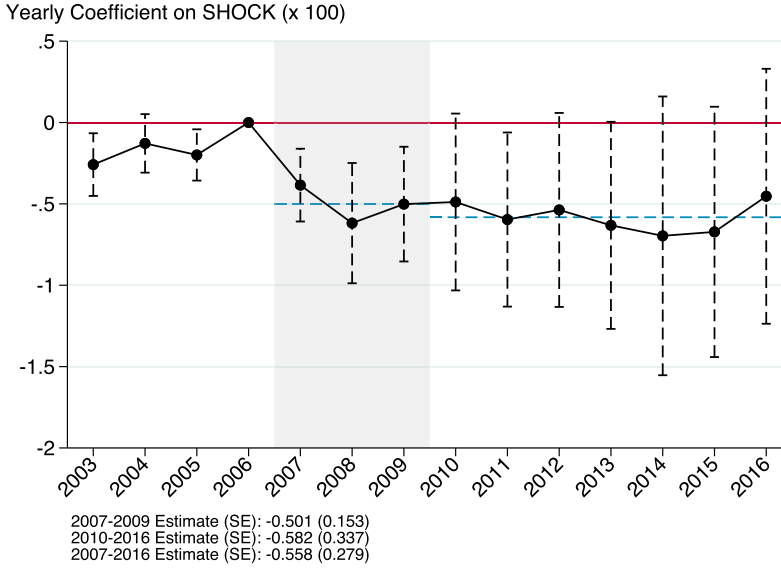

FIGURE III

Impact of the Shock on Log Mortality

This figure displays the yearly coefficients β_t from equation (1), where the outcome y_{ct} is the log age-adjusted CZ mortality rate per 100,000, and $SHOCK_c$ is the 2007–2009 change in the CZ unemployment rate. Observations are weighted by CZ population in 2006. Horizontal blue dashed lines indicate the point estimate for the average of coefficients from 2007–2009 and 2010–2016. These estimates (and corresponding standard errors) are reported in the lower left corner, along with the corresponding estimate for the entire 2007–2016 period. Coefficients, standard errors, and confidence intervals are multiplied by 100 throughout for ease of interpretation. Standard errors are clustered at the CZ level, and dashed vertical lines indicate 95% confidence intervals on each coefficient. The area shaded in gray corresponds to the timing of the Great Recession, adopting the NBER's business cycle dating. $N = 741$ CZs.

mortality, as areas that were harder hit by the recession experienced a relative rise in economic indicators in the preceding years (see Yagan 2019 and Online Appendix Figure A.4). The opposite-signed pre-trend in the mortality estimates suggests that by measuring the mortality effect of the Great Recession relative to 2006, we may be underestimating the extent of recession-induced mortality declines, if the pre-trend reflects unobserved forces that would have continued in the absence of the Great Recession.

The point estimates imply that a 1 percentage point increase in the local area unemployment rate between 2007 and 2009 was associated with a 0.50% (std. err. = 0.15) decline in the area's

annual age-adjusted mortality rate in 2007–2009 relative to its 2006 level. From 2010 to 2016, a 1 percentage point increase in the unemployment rate between 2007 and 2009 was associated with a 0.58% (std. err. = 0.34) decline in the annual, age-adjusted mortality rate relative to 2006. Compared to the shorter-run estimates, the longer-run estimates are much less precise and are only marginally significant ($p = .08$ for 2010–2016, compared with $p = .001$ for 2007–2009); however, we cannot reject that the two estimates are identical ($p = .78$).[5]

The Great Recession on average increased local area unemployment by about 4.6 percentage points between 2007 and 2009. An increase in the local area unemployment rate of this magnitude thus reduces average mortality by 2.3% a year, with effects persisting for at least 10 years. This decline is equivalent to the average, two-year secular mortality improvement over the half-century before the Great Recession (see Online Appendix Figure A.10 and Ma et al. 2015). Based on the standard population life table, the 10-year estimates suggest that 1 in 25 55-year-olds gained an extra year of life from this sized local shock (Online Appendix Table A.2).

III.B. Unpacking the Overall Mortality Decline

Mortality rates vary substantially across demographic groups and reflect several underlying causes (Online Appendix Table A.3).[6] In 2006, the elderly (65 and older) accounted for almost three-quarters of deaths, although they were only 12% of the population; individuals with a high

5. Although there is work looking at lagged effects of unemployment on mortality (e.g., Ruhm 2000), most of the existing literature on the relationship between recessions and mortality assumes that any such relationship is contemporaneous (e.g., Ruhm 2015; Stevens et al. 2015). To investigate possible lagged effects of economic downturns on subsequent mortality, we exploit spatial variation not only in the initial labor market impact of the Great Recession but also in the labor market recovery, conditional on the initial economic impact. In Online Appendix C.1, we show that a larger initial economic shock continued to translate into larger mortality declines in 2010–2016 in areas with below-median economic recoveries from 2010–2016 and in areas with above-median recoveries. However, like our overall estimates of mortality declines in Figure III, these 2010–2016 estimates lack precision, and the two areas' estimated impacts in 2010–2016 are not statistically distinguishable from zero or from each other.

6. We use the top 11 causes of death in 2006 from the Department of Vital Statistics' List of 39 Selected Causes of Death and group all remaining causes into a single residual category. See Online Appendix B for more detail.

school diploma or less make up about half (52%) of the population but account for 70% of deaths. Mortality also reflects several underlying causes. The two most common causes of (age-adjusted) deaths were cardiovascular disease (34% of deaths) and malignant neoplasms, that is, cancer (23%). In this section, we examine the nature of the mortality decline across causes of death and various demographics and briefly explore effects on morbidity. Because the patterns are often similar in 2007–2009 and 2010–2016, we focus most of the discussion on 2007–2009, for which we have greater precision. We frequently summarize the average estimates; all underlying event studies are shown in Online Appendix D.

1. *By Cause of Death.* Figure IV, Panel A shows the estimated 2007–2009 mortality effects for the top 11 causes of death (arranged in descending order of prevalence in 2006) and a final residual category for all other causes (Online Appendix Figure A. 7a does the same for 2010–2016). Because the underlying mortality rates differ greatly by cause, Figure IV, Panel B combines these estimates with 2006 cause-specific mortality rates to report the share of the recession-induced 2007–2009 mortality reduction accounted for by each cause of death.

Cardiovascular disease accounts for the largest share of deaths and the largest share of the estimated total reduction in deaths. A 1 percentage point increase in the 2007–2009 local area unemployment reduces the mortality rate from cardiovascular disease by 0.65% (std. err. = 0.21). Since cardiovascular disease accounted for over a third of total mortality in 2006, the estimate implies that nearly half (48%) of the deaths averted by the Great Recession would have been caused by cardiovascular disease. By contrast, while their percentage mortality reductions are large and statistically significant, motor vehicle accidents and liver disease each account for less than 2% of 2006 mortality, so their contributions to the total recession-induced mortality decline are only 6.9% and 2.6%, respectively.[7]

7. Given the large (22.5%) share of deaths in the residual category, we analyzed an alternative, standard cause-of-death grouping into 10 mutually exclusive categories which yields a much smaller (3.5%) share of deaths in the residual category. The two approaches code many causes similarly, so the results are quite similar. We discuss the alternative coding, how it compares to our baseline, and the results in more detail in Online Appendix C.2.

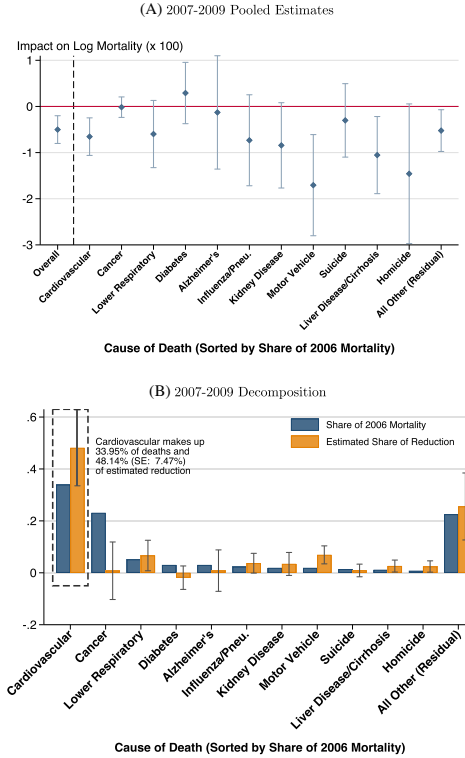

FIGURE IV

Impact of the Shock on Log Mortality, by Cause of Death

Panel A displays the group-specific average of 2007–2009 coefficients β_{tg} from equation (2), where the outcome y_{ctg} is the log age-adjusted CZ mortality rate per 100,000, groups g are defined as the 11 most common causes of death in the ICD10 39-group classification (presented in order of decreasing prevalence), and the final category is a residual category that captures all other mortality. Observations are weighted by CZ population in 2006. Coefficients and confidence intervals are multiplied by 100 throughout for ease of interpretation. Point estimates are displayed as diamonds; vertical bars indicate 95% confidence intervals, clustered at the CZ level. Analogous 2010–2016 estimates can be found in Online Appendix Figure A.7. Panel B decomposes the contribution of these 12 mutually exclusive and exhaustive cause of death categories to the overall estimated 2007–2009 pooled reduction in mortality (i.e., the estimate from Panel A). The blue (dark gray) bars indicate each cause of death's share of 2006 mortality. The orange (light gray) bars present the implied share of the mortality decline accounted for by a given cause of death. To construct these orange (light gray) bars, we multiply each estimated cause-of-death reduction in 2007–2009 by the number of deaths from that cause in 2006 and divide by the sum of estimated death reductions across all causes. Note that the implied "overall" mortality reduction from this exercise is −0.46%, very close to our estimate from Figure III of −0.50%. Ninety-five percent confidence intervals for these estimates, clustered by CZ, are shown as vertical lines. $N = 741$ CZs.

Most other point estimates in Figure IV, Panel A also indicate mortality declines, and no cause of death experiences a statistically significant increase in mortality. Several other causes—lower respiratory disease, influenza/pneumonia, kidney disease, and homicides—experience a percentage decline in their mortality rate similar to or larger than that of cardiovascular disease, but these declines are not statistically significant. For cancer deaths, which is the second largest cause, we estimate a precise null effect of 0.02% (std. err. = 0.11), which we interpret as reassuring that our results are picking up the causal effect of the Great Recession, rather than spurious factors correlated with the size of the shock.

Figure V displays the full event-study estimates from 2003 to 2016 for cardiovascular disease, cancer, motor vehicle accidents, suicide, liver disease, and homicide (event studies for the remaining causes of death appear in Online Appendix Figure A.8). The effects on cardiovascular and liver mortality seem persistent, with long-run (2010–2016) estimates similar to short-run (2007–2009) estimates but with larger standard errors. By contrast, the effects on mortality from motor vehicle accidents have entirely dissipated by 2016, while the modest decline in suicide due to the Great Recession over 2007–2009 grows in magnitude in 2010–2016 to a statistically significant 1.7% decline (std. err. = 0.5) for each percentage point increase in the 2007–2009 unemployment rate. This effect is striking given state-year panel estimates that increases in unemployment are associated with contemporaneous increases in suicides (Ruhm 2000; Harper et al. 2015) and may reflect recession-induced reductions in pollution as we discuss shortly.

Not surprisingly in light of the recession-induced declines in suicides and deaths from liver disease, the Great Recession reduced "deaths of despair" (Case and Deaton 2015, 2017, 2021)—deaths from suicide, liver disease, and drug poisonings—in 2010–2016. A 1 percentage point increase in the 2007–2009 unemployment rate is associated with a 1.4% (std. err. = 0.63) decline in deaths of despair from 2010–2016 (Online Appendix Figure A.11a). Consistent with this, Case and Deaton (2017) note that there is no evidence of deaths of despair rising during the Great Recession; they interpret deaths of despair arising not from declines in income but from a more prolonged effect of cumulative disadvantage. However, our findings contrast with Pierce and Schott (2020)'s result that areas more exposed to import compe-

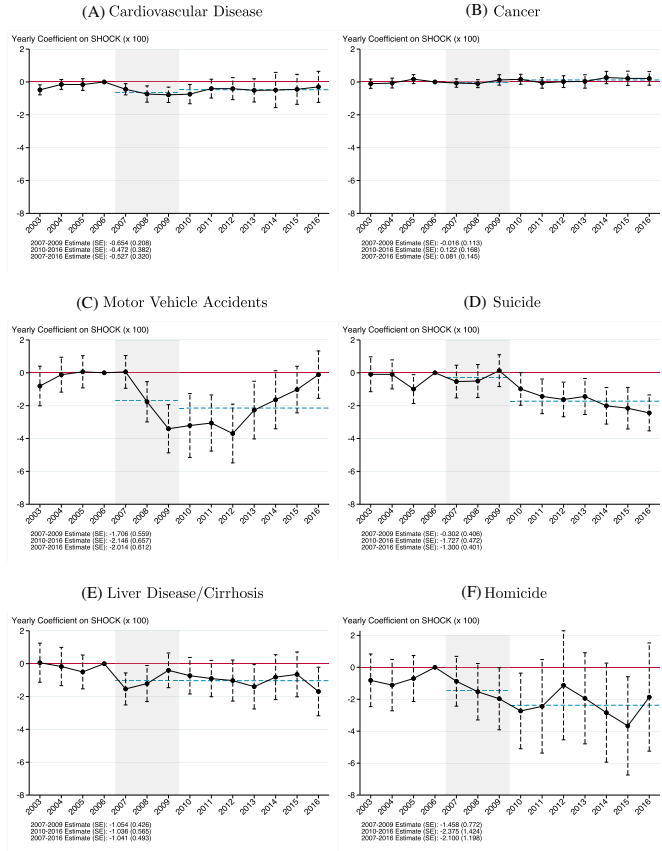

FIGURE V

Impact of the Shock on Log Mortality, by Cause of Death: Selected Event Studies

This figure displays the yearly coefficients β_{tg} from equation (2), where the outcome y_{ctg} is the log age-adjusted CZ mortality rate per 100,000, and g indicates 12 cause of death categories (6 of which are displayed here; the remaining 6 are in Online Appendix Figure A.8). $SHOCK_c$ is the 2007–2009 change in the CZ unemployment rate. Panel A displays effects on the log mortality rate from cardiovascular disease; Panel B from cancer; Panel C from motor vehicle accidents; Panel D from suicide; Panel E from liver disease; and Panel F from homicide. Observations are weighted by CZ population in 2006. Horizontal blue dashed lines indicate the point estimate for the average of coefficients from 2007–2009 and 2010–2016. These estimates (and corresponding standard errors) are reported in the lower left corner, along with the corresponding estimate for the entire 2007–2016 period. Coefficients, standard errors, and confidence intervals are multiplied by 100 throughout for ease of interpretation. Standard errors are clustered at the CZ level, and dashed vertical lines indicate 95% confidence intervals on each coefficient. The areas shaded in gray correspond to the timing of the Great Recession, adopting the NBER's business cycle dating. $N = 741$ CZs.

tition from China experienced an increase in deaths of despair primarily among working-age populations.

2. *By Age.* Figure VI displays 2007–2009 mortality declines for each age group, indicating that the Great Recession is associated with quantitatively and statistically similar percentage reductions in mortality rates across all (adult) age groups. The point estimates in Panel A are broadly similar across age groups, with many statistically significant. Though the point estimates are larger at younger ages, they are also quite imprecise. Longer-term 2010–2016 estimates, as displayed in Online Appendix Figure A. 7b, display similar trends. When we aggregate into larger age groups, we are unable to reject the hypothesis that the average percentage decline in mortality across 2007–2009 is the same for ages 25–64 and for 65+ ($p = .30$).[8]

Panel B combines the point estimates with mortality rates by age to show the contribution of different age groups to the estimated recession-induced reduction in total mortality. The elderly account for the majority (74.3%) of deaths averted by the Great Recession, roughly proportional to their 72.5% share of total mortality in 2006. The slightly larger percentage decline in mortality rates for 0–24-year-olds seen in Panel A has little quantitative significance for the total mortality declines, given the very low baseline mortality rate of this age group.

3. *By Education.* Strikingly, the entire recession-induced mortality decline is concentrated among those with a high school diploma or less (Figure VII, Panel A). Specifically, among those age 25 and over, we compare effects separately for the roughly half of the population with a high school diploma or less to those with more than a high school diploma.[9] The point estimates indicate that in 2007–2009, a 1 percentage point increase in the local unemployment rate is associated with a statistically significant 0.80% (std. err. = 0.26) decline in the mortality rate for those with high school or less, compared with a statistically insignifi-

8. By contrast, we can reject that the percentage decline in mortality for 0–24-year-olds is the same as either the percentage decline for 25–64-year-olds ($p = .01$) or for 65+-year-olds ($p = .03$).

9. Because of data limitations explained in more detail in Online Appendix C. 3, this analysis is conducted at the state rather than CZ level, is limited to individuals ages 25 and older, and excludes a few states with missing data. As shown in that appendix, these restrictions have little effect on our estimates.

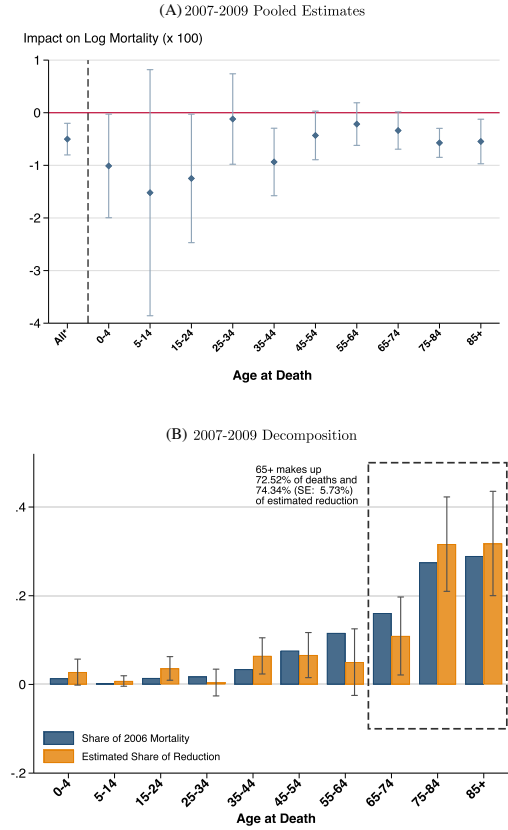

FIGURE VI

Impact of the Shock on Log Mortality, by Age

This figure displays the group-specific average of 2007–2009 coefficients β_{tg} from equation (2), where the outcome y_{ctg} is the log CZ mortality rate per 100,000 for a given age group, without any age adjustment, and groups g are defined by 10 age groups. Observations are weighted by CZ population in 2006. Coefficients and confidence intervals are multiplied by 100 throughout for ease of interpretation. Period estimates are displayed as diamonds; vertical bars indicate 95% confidence intervals, clustered at the CZ level. Analogous 2010–2016 estimates can be found in Online Appendix Figure A.7. Panel B decomposes the contribution of these 10 age groups to the overall estimated 2007–2009 pooled reduction in mortality (i.e. the estimate from Panel A). The blue bars indicate each age group's share of 2006 mortality. The orange bars present the implied share of the mortality decline accounted for by a given age group. To construct these orange bars, we multiply each estimated age group reduction in 2007–2009 by the number of deaths from that age group in 2006 and divide by the sum of estimated death reductions across all age groups. Note that the implied "overall" mortality reduction from this exercise is −0.50%, matching our estimate from Figure III of −0.50%. Ninety-five percent confidence intervals for these estimates, clustered by CZ, are shown as vertical lines. $N = 741$ CZs.

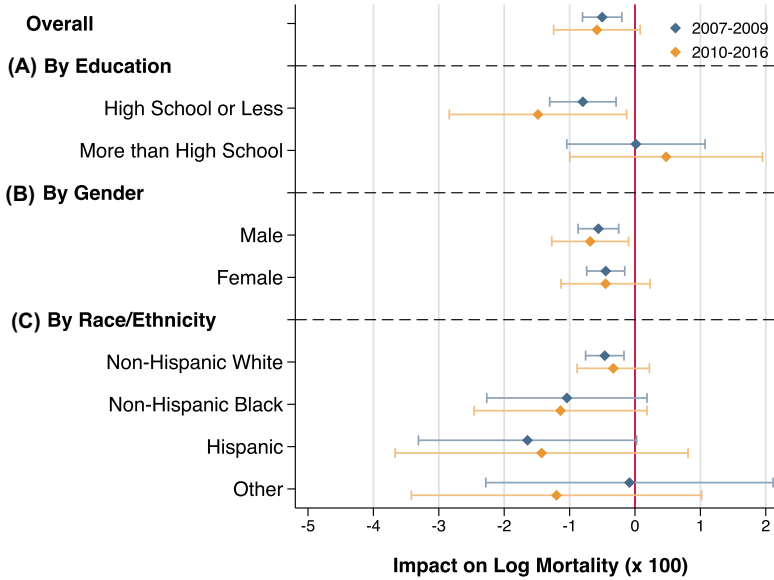

FIGURE VII

Impact of the Shock on Log Mortality, by Education, Gender, and Race

This figure displays the group-specific average of 2007–2009 and 2010–2016 coefficients β_{tg} from equation (2), where the outcome y_{ctg} is the log age-adjusted mortality rate per 100,000 and groups g are defined by education, gender, and race categories. The top row replicates the baseline estimates for the full sample, weighting by the 2006 CZ population. Effects by education are estimated on a restricted sample and at the state level, weighting by 2006 state population. Effects by gender and race are estimated at the CZ level, weighting by 2006 CZ population. Coefficients and confidence intervals are multiplied by 100 for ease of interpretation. Period estimates are displayed as diamonds; horizontal bars indicate 95% confidence intervals, clustered at the CZ level. $N = 741$ CZs for "overall" estimates. $N = 47$ states for estimates by education. $N = 739$ CZs (>99.9% of the total 2006 population) for estimates by gender, and $N = 434$ CZs (96% of the total 2006 population) for estimates by race. Note that calculating age-adjusted mortality rates separately for each racial group requires a CZ to have at least one person of each race in all 19 age bins in all years of our sample period. This requirement drops smaller, less diverse CZs but keeps the larger ones, hence most of the 2006 population is still covered despite dropping so many CZs.

cant 0.014% (std. err. = 0.54) increase for those with more than high school. Although the mortality effects by education are not statistically distinguishable ($p = .12$) in 2007–2009, they are statistically distinguishable ($p < .01$) in 2010–2016 (the point estimate is −1.48 (std. err. = 0.69) for those with less education compared with 0.48 (std. err. = 0.75) for those with more) and for the

entire 2007–2016 period ($p < .01$; the point estimate is −1.3 (std. err. = 0.56) for those with less education compared with 0.34 (std. err. = 0.68) for those with more education).

We performed several additional checks and analyses on these results by education. First, since the education distribution differs by age, we confirmed that the impact of the Great Recession is confined to those with high school education or less even within age groups (Online Appendix Figure A.43). Second, when we further disaggregate the higher education sample into those with some college and those with college or more, there is no evidence of mortality declines in either subgroup.[10] Third, given potential concerns that the differential effects by education might reflect differences across areas with different education shares, we confirmed that there is little variation in the share of a state's population with a high school degree or less, and little correlation between the state-level Great Recession shock and the education share.[11] Finally, consistent with mortality effects that are concentrated among those with less education, we find in the Medicare data that the mortality impacts on the elderly are much larger among the approximately 12% of the population on Medicaid (a proxy for low income) in the prior year (Online Appendix Figure A.44).

4. *By Gender and by Race/Ethnicity.* We find no evidence of differential mortality effects by gender, with nearly identical estimates for men and women (Figure VII, Panel B). While recession-induced mortality declines appear to be more pronounced for nonwhite population groups (with particularly large point estimates for Hispanic individuals), we cannot reject equal impacts across groups in any time period (Figure VII, Panel C).

10. When we disaggregate the lower education sample into those with less than a high school diploma and those with exactly a high school diploma, we see declines in both subgroups (Online Appendix Figure A.42), although the estimates become much noisier and statistically insignificant.

11. The (population-weighted) mean state has 52% of the population with a high school diploma or less, and the 10–90 range is only 0.46 to 0.58. The correlation between the state-level Great Recession shock and the share with a high school diploma or less is 0.16, and not statistically significantly different from zero. Expanding our analysis to allow for calendar year effects to vary with the share of the state's population with a high school diploma or less in 2006 yields unchanged results (see Online Appendix C.6, and especially Online Appendix Figure A.18).

5. *Health Status of Marginal Lives Saved.* When examining mortality effects over short time horizons—such as a day or three days—a natural question is whether they reflect a meaningful change in mortality over longer horizons or merely a slight retiming of deaths, a phenomenon often referred to as "mortality displacement" or "harvesting." Researchers tend to investigate this possibility by studying longer time horizons, such as a month or a year (see Chay and Greenstone 2003; Deryugina et al. 2019). Displacement is much less of a concern in our setting, where we study effects at the annual level that persist over 10 years.

Nevertheless, for our welfare analysis in Section V, it matters whether the remaining life expectancy of the marginal lives saved by the Great Recession differs from that of the typical decedent of the same age. Closely following Deryugina et al. (2019), we use the Medicare data to develop an auxiliary model of mortality as a function of individual demographics and health conditions at the beginning of the year. We use this model to predict counterfactual, remaining life expectancy for each individual in each year and analyze the effect of the Great Recession on life-years lost. The marginal life saved—when predicting life expectancy based on age, demographics, and chronic conditions—has only a statistically insignificant 6% lower counterfactual remaining life expectancy than a typical decedent of the same age (see Online Appendix C.4 for more detail).

6. *Morbidity.* We focus on mortality as a measure of health because it is not only important but also consistently and comprehensively measured. However, it is an imperfect measure of health, particularly at younger ages with low mortality (see Online Appendix Table A.3). The focus on mortality therefore raises the possibility that we are missing important nonmortality health effects at younger ages that might eventually translate into mortality effects decades later. These longer-run mortality effects need not be beneficial; for those who are entering the labor market (ages 16–22) during a recession, Schwandt and von Wachter (2020) find long-run mortality increases.

In the spirit of Ruhm (2003), we explore (where feasible) the impact of the Great Recession on measures of morbidity. We sign each measure so that—like mortality—higher values indicate worse health. Specifically, we analyze the impact of the Great Recession on the log share of respondents in the BRFSS with the following self-reported morbidity measures: (i) health that is less

than very good, (ii) any days in the last month with poor mental health, (iii) ever been diagnosed with diabetes, and (iv) currently have asthma. Since we cannot observe CZs in the BRFSS, we estimate equation (1) at the state level.[12]

Figure VIII shows evidence that the Great Recession reduced morbidity. Panel A shows the results for the measures of self-reported morbidity individually and for the average treatment effects, created by taking the simple average of treatment effects across the measures. The Great Recession caused a statistically significant 1.26% reduction (std. err. = 0.47) of the morbidity index over 2007–2009 and a 1.19% reduction (std. err. = 0.51) over the entire 2007–2016 period. This reduction reflects declines in each measure of morbidity, although none of them are individually statistically significant. For example, in 2007–2009, a 1 percentage point increase in the state unemployment rate is associated with a statistically insignificant 0.98% (std. err. = 0.59) decrease in the share of the population reporting themselves to be in less than very good health (i.e., fair, poor, or good health) and a 1.37% (std. err. = 1.13) decline in the share who report themselves as having asthma. The declines in average morbidity are similar across age groups (18–45, 46–64, and 65+), but only statistically significant for the two younger age groups. Overall, we interpret these results as suggestive that morbidity is also procyclical, with roughly similar magnitudes across age groups.

III.C. Investigating Sensitivity to Population Changes: Medicare Panel Data

If recessions affect the size or composition of the local population in a way that is not captured by our population measures, such impacts could bias the estimated relationship between recessions and mortality. Arthi, Beach, and Hanlon (2022) suggest that this potential for endogenous, unmeasured changes in the local population in response to economic shocks is a key limitation of the existing literature on the impact of recessions on mortality. Consistent with such concerns, areas that were harder hit by the Great Recession experienced a relative decline in (measured) population, primarily reflecting an increase in the share of the

12. We show in Online Appendix C.6 that our baseline mortality estimates (Figure III) are unchanged when switching from the CZ to the state level for analysis.

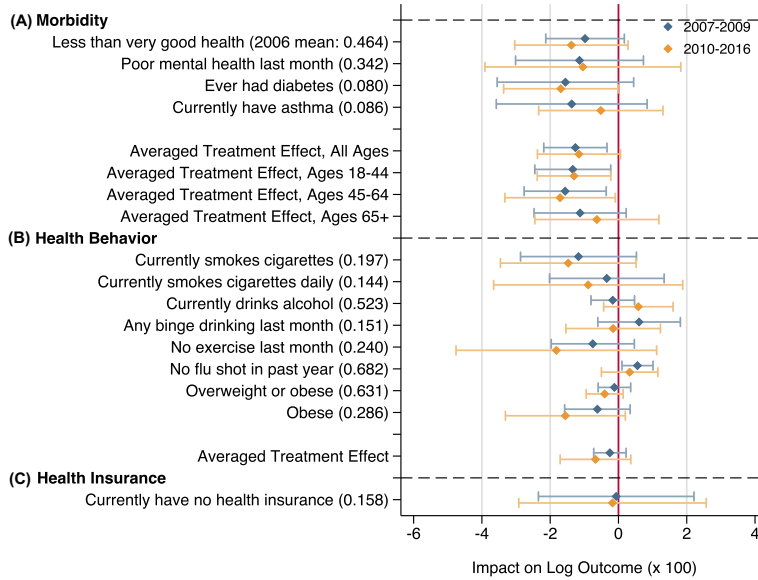

FIGURE VIII

Impact of the Shock on Log Self-Reported Health and Health Behaviors

This figure displays the average of 2007–2009 and 2010–2016 coefficients β_t from equation (1), where the outcome y_{ct} is the log share of respondents in each state who report the various rows' health conditions or health behaviors in the 2003–2016 BRFSS. Online Appendix B.4 provides more details on the sample and variable definitions. The averaged treatment effects are the average of the coefficients for each measure of health or health behavior, either for the sample as a whole or separately by age group as indicated. State averages are generated as the mean value of individual reports in a given state, weighted by BRFSS survey weights. Estimates are therefore all estimated at the state level, weighting by 2006 state population. Period estimates are displayed as diamonds; horizontal bars indicate 95% confidence intervals, clustered at the state level. Coefficients, standard errors, and confidence intervals are multiplied by 100 for ease of interpretation. The population average of each outcome (in levels, not logs) in 2006 is noted in parentheses next to each variable label (i.e., 2006 population-weighted means of each state estimate). $N = 51$ states.

population that is 65 and older.[13] This finding raises the concern that what looks like fewer people dying in harder-hit areas might

13. See Online Appendix Figure A.12 and Yagan (2019). The compositional change primarily reflects a decline in in-migration of prime-age workers to areas particularly affected by the Great Recession, rather than an increase in outmigration (Yagan 2019; Monras 2020; Hershbein and Stuart 2024). We show in Online Appendix C.5 that population composition based on gender, race, and education does not change in areas that are more versus less affected by the Great Recession (Online Appendix Figure A.13) and that predicted mortality—based on

in fact reflect fewer people living in these places. One finding that mitigates the estimated population declines driving our findings is that we estimate a precise zero for declines in cancer mortality, the second leading cause of death (Figure IV). If estimated declines in the mortality rate simply reflected unmeasured declines in population, we would expect to see declines in mortality for all major causes of death.[14]

To directly explore the sensitivity of our findings to unmeasured population changes, we turn to the individual-level panel data for the Medicare population. We analyze a panel of 2003 Medicare enrollees aged 65–99 in 2003 and examine how the estimated mortality effect of the Great Recession is affected by fixing their location at their 2003 location compared to allowing it to vary each year as it (implicitly) can in the preceding analyses using the death certificate data. We follow the standard approach in the literature (e.g., Olshansky and Carnes 1997; Chetty et al. 2016; Finkelstein, Gentzkow, and Williams 2021), and adopt a Gompertz specification in which the log of the mortality rate for individual i in year t ($\log(m_{it})$) is linear in age a. Once again, we focus our discussion primarily on the 2007–2009 results, where we have greater precision.

We begin by showing Gompertz estimates for the sample of Medicare enrollees we observe in 2003 and follow forward, using their yearly location. Specifically, we estimate:

$$\log(m_{it}(a)) = \rho a + \beta_t[SHOCK_{c(i,t)} \times \mathbb{1}(Year_t)] + \alpha_{c(i,t)} + \gamma_t + \epsilon_{it}. \tag{3}$$

Once again, γ_t are year fixed effects, and we cluster standard errors at the CZ level.

Table I, first row (and Online Appendix Figure A.15a) shows estimates based on yearly location. The 2007–2009 estimate indicates that a 1 percentage point increase in the local area unemployment rate reduces the annual mortality rate by 0.51% (std.

gender, race, and education—does not change in areas that are more versus less affected by the Great Recession (Online Appendix Figure A.14).

14. This logic presumes that migration rates are similar for individuals with different comorbidities. We confirmed in the Medicare data that people who died of cancer the year before the Great Recession were as likely to have lived in the same CZ in the years leading up to their death as people who died of other causes in that year.

TABLE I
SENSITIVITY TO CURRENT VERSUS 2003 LOCATION

Regression specification	2007–2009 estimate	2010–2016 estimate	2007–2016 estimate
Yearly residence (β_t, equation (3))	−0.513	−0.533	−0.527
	(0.161)	(0.241)	(0.210)
2003 residence (reduced form) (π_t^{RF}, equation (4))	−0.348	−0.269	−0.293
	(0.157)	(0.233)	(0.203)
First stage (π_t^{FS}, equation (5))	0.945	0.916	0.925
	(0.003)	(0.005)	(0.004)
Control function (β_t, equation (6))	−0.370	−0.326	−0.339
	(0.165)	(0.251)	(0.223)
Yearly residence (non-movers) (β_t, equation (3))	−0.559	−0.666	−0.634
	(0.179)	(0.244)	(0.218)

Notes. This table displays the point estimates and standard errors (in parentheses) of coefficients from various individual-level Gompertz hazard models of $\log(m_{it}(a))$, the log mortality rate at age a. The table displays the average of yearly coefficients from 2007–2009, 2010–2016, and 2007–2016. Estimates are based on coefficients π_t^{FS} from equation (4) for the reduced-form specification, on coefficients π_t^{FS} from equation (5) for the first-stage regression where the dependent variable is the shock experienced in a given year, and on coefficients β_t from equation (6) for the control-function specification and from equation (3) for yearly residence specifications. $SHOCK_c$ is defined as the 2007–2009 CZ change in the unemployment rate. Standard errors are clustered at the CZ level, except for the standard errors from estimating the control-function specification, which are calculated by performing a Bayesian bootstrap of the two-stage procedure with 500 repetitions so that first-stage residuals are redrawn for every reweighted sample. Coefficients and standard errors are multiplied by 100 for ease of interpretation. The sample is all 2003 Medicare beneficiaries, subject to the restrictions in Online Appendix Table A.7. The event studies for rows 1, 2, and 4 can be found in Online Appendix Figure A.15, the event study for row 3 can be found in Online Appendix Figure A.15c, and the event study for row 5 can be found in Online Appendix Figure A.16. $N = 6{,}634{,}999$ in all rows, except for the last row where we limit to non-movers, where $N = 5{,}838{,}592$.

err. = 0.16), which is nearly identical to our baseline estimate in Figure III.[15]

We then report results from estimating the reduced-form impact of the Great Recession based on individuals' location in 2003:

$$\log(m_{it}(a)) = \rho a + \pi_t^{RF}[SHOCK_{c(i,2003)} \times \mathbb{1}(Year_t)] + \alpha_{c(i,2003)} + \gamma_t + \epsilon_{it}. \quad (4)$$

The key distinction is that we now measure both the location fixed effects $\alpha_{c(i,2003)}$ and the Great Recession shock $SHOCK_{c(i,2003)}$ based on individuals' location in 2003. Measuring location prerecession alleviates concerns about potential contamination from

15. For the 65+ population, our baseline analysis using equation (1) in the CDC data (as displayed in Figure VI) looks similar to results from estimating equation (1) using the 65+ Medicare repeated cross-sectional data and using a subsample of Medicare enrollees whom we can observe in 2003 and follow forward (see Online Appendix Figure A.17).

differential population flows into or out of areas that experience different shocks. We continue to find a statistically significant decline in mortality from an increase in the unemployment rate (Table I, second row, and Online Appendix Figure A.15b). In 2007–2009, a 1 percentage point increase in the local area unemployment rate reduces the annual mortality rate by 0.35% (std. err. = 0.16).

This reduced-form impact of the Great Recession will be biased downward by any difference between the 2003 location and the contemporary location. To account for this, we estimate the first-stage equation relating the shock a person would have experienced each year based on her current location to the shock that she would have experienced based on her 2003 location:

$$SHOCK_{c(i,t)} \times \mathbb{1}(Year_t) = \rho a + \pi_t^{FS}[SHOCK_{c(i,2003)} \times \mathbb{1}(Year_t)] + \alpha_{c(i,2003)} + \gamma_t + v_{it}. \quad (5)$$

The first stage is large (Table I, third row, and Online Appendix Figure A.15c), with an average coefficient of 0.95 (std. err. = 0.003) in 2007–2009; not surprisingly, the reduced form is only slightly smaller than the control-function estimate (Table I, fourth row, and Online Appendix Figure A.15d) when we use the $\hat{v}_{it}$ residuals from equation (5) as an additional regressor in the following equation:

$$\log(m_{it}(a)) = \rho a + \beta_t[SHOCK_{c(i,t)} \times \mathbb{1}(Year_t)] + \alpha_{c(i,2003)} + \gamma_t + \phi\hat{v}_{it} + \epsilon_{it}. \quad (6)$$

The identifying assumption behind this control-function approach is that while a person's 2003 location of residence may directly affect their mortality—reflecting a combination of systematic variation in unobserved health determinants across the elderly in different CZs and any direct impact place of residence has on mortality as in Finkelstein, Gentzkow, and Williams (2021)—the Great Recession shock experienced by the place a person lives in 2003 only affects their mortality through its correlation with the shock experienced by the place they live in later years. The control-function estimated mortality effect from 2007–2009 of −0.37 (std. err. = 0.17) is smaller in absolute value—but not statistically distinguishable from—than the estimate based on yearly residence in the first row. This difference may reflect the presence of unmeasured population declines in areas harder hit by the Great Recession. Finally, Table I, fifth row (Online Appendix Figure A.

16) shows estimates based on yearly location (i.e., estimating equation (3)), limited to the 88% of the sample who does not move CZ from their 2003 location; these estimates are also quite similar to the estimates based on year residence for the full sample (first row).

1. *Additional Sensitivity Analyses.* In Online Appendix C.6, we explore the sensitivity of our baseline mortality estimate in Figure III to several alternative specifications. These include (i) the geographic unit of analysis (CZ versus state versus county), (ii) our choices regarding functional form for the dependent variable and the key independent $SHOCK_c$ variable, and (iii) the sample of CZs included in the analysis (to confirm, for example, that our findings do not spuriously reflect effects of the geographically concentrated fracking boom that occurred during our time period). The results are quite stable across these alternatives. Several additional analyses lend support to the assumption in our baseline specification that the log mortality rate is linear in the size of the shock; for example, the estimated impacts are similar whether estimated based on CZs that experienced an above-average or below-average unemployment shock. Since the average shock to the unemployment rate during the Great Recession was much higher than a typical recession, this linearity increases our confidence that our mortality findings may generalize to more "typical" recessions.

IV. Mechanisms

Recessions might reduce mortality through several channels. We group them into internal effects—whereby an individual's reduced employment or consumption reduces her own mortality—and external effects, which hold constant one's own employment and consumption and include any externalities from reduced aggregate economic activity on health.[16] Internal and external effects have different implications for the welfare consequences of

16. We use the term "external effects" rather than "externalities" to indicate a broader set of health effects from factors other than individual-level behavioral responses. Of course, some channels—such as the reduction in motor vehicle fatalities, which we find was responsible for about 7% of the total recession-induced mortality decline—likely reflect a mix of internal effects (single-car accidents) and external effects (multicar accidents).

our findings. External health benefits from reduced economic activity would suggest that the negative welfare effects from reduced income and consumption are mitigated by positive welfare effects from improved health. In contrast, the welfare implications of mortality reductions from internal effects would be less clear-cut and depend partly on whether people engage in privately optimal behavior. Our findings strongly point to external effects as the primary driver of the recession-induced mortality reductions, motivating our final section, where we examine their implications for the welfare consequences of recessions.

IV.A. Internal Effects

There are two main channels for internal effects discussed in the literature. First, with their increased nonlabor time, the newly unemployed may have more time for self-care, which may improve health by reducing stress (Brenner and Mooney 1983; Ruhm 2000) or improving health behaviors (Ruhm 2000, 2005). Under this scenario, we might expect to see improved diet, increased exercise, and increased smoking cessation—which was the mechanism behind the procyclical mortality effects emphasized in the original work by Ruhm (2000)—as well as potentially increased use of medical care. Second, recession-induced consumption declines could improve health by decreasing health-harming consumption such as alcohol, illegal drugs, and cigarettes (Ruhm 1995; Carpenter and Dobkin 2009; Evans and Moore 2012).

Two features of our findings in Section III are inconsistent with internal effects as the primary driver of the estimated mortality declines. First, three-quarters of the mortality reduction comes from a reduction in elderly deaths, a group we estimate did not experience any direct income effects from Great Recession–induced local labor market declines (Online Appendix C.8 and especially Online Appendix Figure A.20).[17] Second, the time pattern of the mortality reductions—an immediate decline that does not grow larger over time (recall Figure III)—is not consistent with an important role for changes in health behaviors; we would ex-

17. Consistent with our findings, other work using the same empirical strategy has similarly found little evidence of Great Recession–induced employment declines for the elderly. For example, Rinz (2022) finds much more modest and short-lived declines in elderly employment compared to effects at younger ages.

pect changes in exercise, diet, or smoking to affect mortality with a lag and grow over time as health capital improves.[18]

We find little direct evidence of a substantive role for internal effects. We do not find a statistically significant impact of the Great Recession on self-reported health behaviors (Figure VIII, Panel B) either individually or pooled to improve statistical power. Specifically, we examine the effect on the log share of individuals in the area who report that they currently smoke, smoke daily, currently drink, have consumed more than five drinks in one sitting in the past month, have not exercised within the past 30 days, did not receive a flu shot in the past year, or are currently overweight or obese. Although imprecise, some of the point estimates are consistent with potentially large improvements in certain health behaviors such as smoking and exercise, which might ultimately translate into important health improvements.[19] We find no evidence of a substantively or statistically significant impact on health care use among the elderly, measured in the Medicare data by physician visits, ER visits, or total expenditures (Online Appendix Figure A.21).[20] Finally, consistent with a role for declines in health-harmful consumption, we found declines (some statistically significant) in mortality from cirrhosis of the liver, homicide, suicide, and drug poisonings (see Figure IV and Online Appendix Figure A.11b). However, the combined decline in mortality that may be due to health-harmful consumption accounts for less than 7% of the total reduction in mortality.

18. For example, studies of the effect of smoking cessation on mortality find that effects grow gradually over a 10–15-year period and the effects in the first few years constitute only a small share of the total mortality declines (see Kawachi et al. 1993; Mons et al. 2015; U.S. Department of Health and Human Services 2020).

19. For example, we estimate that on average over the 2007–2009 period, a 1 percentage point increase in state unemployment from 2007–2009 decreases the share smoking by 1.2% (std. err. = 0.9%), increases the share excessively drinking by 0.6% (std. err. = 0.6%), and decreases the share not exercising by 0.8% (std. err. = 0.6%). Interestingly, although statistically insignificant, the point estimates are often similar in magnitude to those found in Ruhm (2000). Online Appendix Table A.4 shows this more clearly by estimating the specification in levels and reporting the comparable estimates from Ruhm (2000).

20. The one exception is inpatient visits, where there is a statistically significant increase in the share of patient-years with an inpatient admission (0.8% per percentage point increase in $SHOCK_c$) in the 2010–2016 period. This increase may reflect compositional changes, as elderly individuals who would have died are now alive and at risk of hospitalization.

IV.B. External Effects

We explore three main potential sources of positive external health effects from recessions suggested by prior literature: reductions in air pollution (Chay and Greenstone 2003; Heutel and Ruhm 2016), increases in the quality of health care (Stevens et al. 2015), and reductions in the spread of infectious disease (Adda 2016). We find evidence consistent with a quantitatively important role for recession-induced reductions in air pollution—which can explain at least one-fifth and potentially a much larger share of the recession-induced mortality declines—but little support for a role for the latter two classes of external effects.

1. *Reduction in Air Pollution.* To examine the effect of the Great Recession on air pollution and the extent to which this channel is responsible for the reduction in mortality, we conduct our analysis at the county level. This provides a better measure of a person's exposure to pollution than CZ-level analysis; we continue to measure the Great Recession shock at the CZ level because the local labor market is the more suitable area for the impact of that shock, and we continue to cluster our standard errors at the CZ level. We first estimate:

$$y_{ct} = \beta_t [SHOCK_{cz(c)} \times \mathbb{1}(Year_t)] + \alpha_c + \gamma_t + \varepsilon_{ct}, \tag{7}$$

where c now denotes county, cz denotes commuting zone, and $SHOCK_{cz(c)}$ is defined identically as in equation (1). Figure IX, Panel A confirms that our estimates of the impact of the Great Recession on mortality remain very similar to our baseline results in Figure III when we estimate equation (7) at the county level using the age-adjusted log mortality rate as the dependent variable.

Counties that were harder hit by the Great Recession also experienced larger declines in pollution, with these declines persisting through the end of our study period (Figure IX, Panel B). Following the recent air pollution literature (e.g., Deryugina et al. 2019; Dedoussi et al. 2020; Currie, Voorheis, and Walker 2023), we focus on PM2.5 (in $\mu g/m^3$) as the dependent variable when estimating equation (7). A 1 percentage point increase in the CZ-level unemployment rate from 2007–2009 is associated with an average reduction of PM2.5 from 2007–2009 of 0.14 $\mu g/m^3$ (std. err. = 0.039) and from 2010–2016 of 0.20 $\mu g/m^3$ (std. err. = 0.055); to put that in perspective, the average 0.18 $\mu g/m^3$ decline from

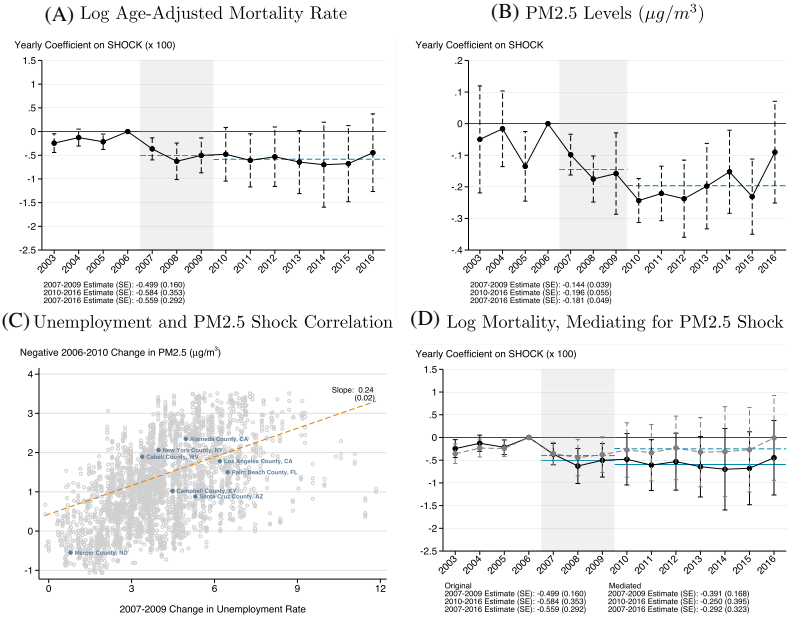

FIGURE IX

Impact of Shock on Log Mortality and Pollution

Panels A and B display the yearly coefficients β_t from equation (7), where the outcome y_{ct} is the log age-adjusted county mortality rate per 100,000 (Panel A) or the annual county PM2.5 level (Panel B), and $SHOCK_c$ is the 2007–2009 CZ change in unemployment rate. Panel C scatters the negative 2006–2010 change in the county PM2.5 level against the 2007–2009 change in its CZ's unemployment rate. The dashed line plots a linear fit, weighted by 2006 county population, with the corresponding slope and standard error to the right side of the figure. Panel D displays the yearly coefficients β_t from equation (8) in gray, where the outcome y_{ct} remains the same. β_t is the coefficient on the 2007–2009 change in the CZ unemployment rate interacted with calendar year when mediating for the negative 2006–2010 change in PM2.5 interacted with calendar year. The unmediated coefficients β_t from Panel A are plotted in black for reference. All analyses are restricted to the 3,107 counties (representing 99.3% of the U.S. population) for which we observe PM2.5 satellite data in both 2006 and 2010, and observations are weighted by county population in 2006. Horizontal blue dashed lines indicate the point estimate for the average of the mediated coefficients from 2007–2009 and 2010–2016 (and solid lines show the original coefficient averages from Panel A for reference). These estimates (and corresponding standard errors) are reported in the lower left corner, along with the corresponding estimate for the whole 2007–2016 period. Coefficients, standard errors, and confidence intervals are multiplied by 100 in Panels A and D for ease of interpretation. Standard errors are clustered at the CZ level, and dashed vertical lines indicate 95% confidence intervals on each coefficient. The areas shaded in gray in Panels A, B, and D correspond to the timing of the Great Recession, adopting the NBER's business cycle dating.

2007–2016 represents a 1.7% decline relative to the 10.5 $\mu g/m^3$ population-weighted national average level of PM2.5 in 2006. Consistent with existing work showing that recessions decrease air pollution (e.g., Chay and Greenstone 2003; Heutel 2012; Feng et al. 2015; Heutel and Ruhm 2016), this finding likely reflects recession-induced declines in major sources of air pollution such as industrial activity, electricity generation, and transportation.

Qualitatively, several pieces of evidence are consistent with the recession-induced pollution decline shown in Figure IX, Panel B driving at least some of the recession-induced mortality declines. First, the time pattern of the effects—with both PM2.5 and mortality declines showing up immediately in 2007—is consistent with a large existing literature indicating effects of (contemporary) pollution on (contemporary) mortality (see Graff Zivin and Neidell 2013; Currie et al. 2014, for reviews). Second, mortality declines and PM2.5 declines persist to the end of our study period. Third, the causes of death that are affected are also consistent with a pollution channel. PM2.5 is understood to affect mortality by reaching deep into the lungs and being absorbed into the bloodstream. This can impair cardiovascular function (EPA 2004) and—perhaps more surprisingly—increase motor vehicle mortality (Burton and Roach 2023) and reduce mental health and increase rates of suicide (Jia et al. 2018; Persico and Marcotte 2022; Molitor, Mullins, and White 2023), all areas where we found statistically significant mortality declines (recall Figure IV). Fourth, the recession-induced mortality declines are concentrated in the half of the population with a high school diploma or less, consistent with less educated and lower-income individuals being disproportionately exposed to greater levels of air pollution overall (Bell and Ebisu 2012; Hajat, Hsia, and O'Neill 2015; Jbaily et al. 2022) and in cities (e.g., Hajat et al. 2013).

Assessing the quantitative importance of recession-induced pollution declines for recession-induced mortality declines is more challenging. Three complementary approaches suggest that pollution is a quantitatively important channel behind the estimated mortality declines. First, combining estimates from Deryugina et al. (2019) of the impact of daily PM2.5 exposure on elderly mortality with our estimates of the effect of an increase in the unemployment rate on the levels of PM2.5, a back-of-the-envelope calculation suggests that the recession-induced pollution declines can explain about 17% to 35% of the 2007–2009

total recession-induced mortality declines, depending on which mortality estimates are used from Deryugina et al. (2019). This calculation imposes the assumption that one year of increased exposure to PM2.5 has 365 times the effect on mortality as one day of increased exposure; as we discuss, this assumption is surely heroic, but the sign of any bias is unclear (see details in Online Appendix C.10).

Second, to more directly gauge the quantitative importance of the pollution channel, we use the fact that while counties that were harder hit by the Great Recession on average experienced a larger decline in pollution (Figure IX, Panel B), there is substantial heterogeneity in this relationship (Figure IX, Panel C). We examine how much the estimated effect of the Great Recession on mortality changes when we control for changes in pollution; under the (admittedly strong) assumptions that the recession shock and the PM2.5 shock are independent conditional on covariates and that the PM2.5 shock is conditionally independent of any other unmeasured mediators of the treatment effect, this mediation analysis allows us to estimate the importance of the pollution channel (see MacKinnon et al. 2002; Fagereng, Mogstad, and Rønning 2021). Specifically, we estimate:

$$y_{ct} = \beta_t[SHOCK_{cz(c)} \times \mathbb{1}(Year_t)] + \phi_t[PM2.5\_SHOCK_c \times \mathbb{1}(Year_t)] + \alpha_c + \gamma_t + \varepsilon_{ct}, \quad (8)$$

where y_{ct} is the log age-adjusted mortality rate, $SHOCK_{cz(c)}$ is defined identically as in equation (7), and $PM2.5\_SHOCK_c$ denotes the negative 2006–2010 change in PM2.5 levels in county c (with positive numbers reflecting a decline).[21]

Figure IX, Panel D shows the estimates of β_t from equation (8). Controlling for the pollution shock attenuates the estimated impact of the Great Recession on mortality from 2007–2009 by about 20%, from a 1 percentage point increase in unemploy-

21. We parameterize $PM2.5\_SHOCK_c$ as the negative 2006 to 2010 change because this change is highly correlated with $SHOCK_{cz(c)}$ (Figure IX, Panel C), while other parameterizations, such as the 2006–2009 change in PM2.5 or the 2006–2016 change, are much less highly correlated, thereby leaving little room for PM2.5 as a mediator. Using measures of changes in PM 2.5 that are not highly correlated with the Great Recession shock seemed contrary to the spirit of the mediation exercise, which is designed to quantify in our setting how the estimated impact of the recession on mortality may be mediated by the estimated impact on pollution.

ment reducing mortality by 0.50% (Figure IX, Panel A) to 0.39% (Figure IX, Panel D).[22]

Third, to isolate exogenous variation in the PM2.5 shock—and avoid potential downward bias from classical measurement error in the PM2.5 shock—we estimate an instrumental variables version of equation (8). We instrument for a county's PM2.5 shock using the CZ-level Great Recession economic shocks in upwind neighboring counties outside of the county's CZ (see Online Appendix C.11 for more detail). Consistent with measurement error in the PM 2.5 variable, recession-induced pollution declines now appear to have a greater role in explaining the mortality declines. Whereas with the OLS analysis, the recession-induced pollution declines explain about 20% of the recession-induced mortality declines from 2007–2009, they appear potentially able to explain them entirely in the IV analysis.[23]

2. *Reduction in the Spread of Infectious Disease.* Influenza and pneumonia accounted for only 2% of deaths in 2006, and the associated mortality declines from the Great Recession are statistically insignificant (Figure IV).

3. *Improved Quality of Nursing Home Care for the Elderly.* Tighter labor markets may result in improved quantity and quality of health care workers. Such changes seem particularly likely for direct care workers providing home care and nursing for the

22. Online Appendix C.9 shows similar results when we focus on the subset of counties where we can measure PM 2.5 in both the EPA monitor data and the baseline data used in Panels B and D of Figure IX. Using the EPA data, we find no significant effects of the Great Recession on other pollutants, specifically carbon monoxide and ozone.

23. The possibility that recession-induced declines in pollution may explain the entirety of recession-induced declines in mortality is broadly consistent with the evidence in Chay and Greenstone (2003), who use geographic variation in reductions in air pollution caused by the 1981–82 recession to assess the impact of air pollution on infant mortality. Their analysis differs from ours in several ways, including their pollution measure—total suspended particulate (TSP) levels, which is a super-set of our air pollution measure of PM2.5 particles—their focus on infant mortality, and their exclusion restriction that the only way the recession affected infant mortality was via effects on pollution. With these caveats in mind, we can apply their headline estimate—a 1% reduction in TSP results in a 0.35% decline in the infant mortality rate—to our setting. Because we estimate a 1.5% reduction in PM2.5, based on their estimate, we would expect a 0.52% decline in mortality, which is nearly identical to our baseline estimate.

elderly, which does not require much formal training and may be relatively elastically supplied. Given widespread concerns about worker shortages in these sectors (e.g., Geng, Stevenson, and Grabowski 2019; Grabowski, Gruber, and McGarry 2023), increased availability of direct care workers could have meaningful health benefits for the elderly. Indeed, Stevens et al. (2015) provide evidence from state-year panel data from 1978–2006 that increases in the unemployment rate are associated with increases in the quantity and quality of nursing home staff and that deaths in nursing homes are particularly responsive to the state unemployment rate. Similarly, using county-year panel data, Konetzka et al. (2018) and Antwi and Bowblis (2018) find that the quality of nursing home staffing is countercyclical.

However, we do not find any evidence for this channel. Figure X, Panel A shows results from reestimating equation (1) in the Medicare data, separately for the 7% of the population that was in a nursing home in any given year or the previous year and the 93% that was not. A 1 percentage point increase in the unemployment rate from the Great Recession reduced mortality rates by the same 0.5% for each group. Individuals who were in a nursing facility in the current or previous year have much higher mortality rates—this 7% of the elderly accounts for 32% of their annual deaths. However, Panel B shows no evidence of an increase in the number or the skill mix of nursing staff hours in nursing homes where the Great Recession hit harder.[24] Panel C also shows no evidence of an impact of the Great Recession on nursing home occupancy rates or resident characteristics. Finally, in Online Appendix C.7, we find no evidence of a recession effect on whether elderly individuals receive more home health care either from a professional or from a spouse, child, or relative, although the results are fairly noisy.[25]

24. For example, the point estimates suggest that for every 1 percentage point increase in the local area unemployment rate during the Great Recession, there is a statistically insignificant 0.11% (std. err. = 0.22) decrease in direct care hours per resident-day during 2007–2009 and a 0.09% decrease (std. err. = 0.24) from 2010–2016. By contrast, Stevens et al. (2015) estimate that every 1 percentage point increase in the state-year unemployment rate increases employment in a nursing home by 3%.

25. Another potential channel for improved quality of care could be recession-induced decreases in motor vehicle traffic and thus reduced ambulance transport times. There is evidence that increased congestion increases ambulance transport times and increases the mortality of individuals admitted to the hospital with

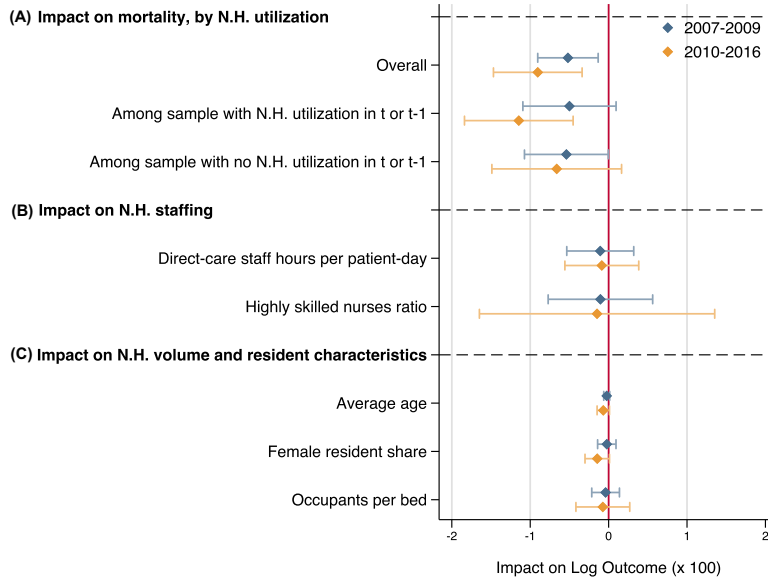

FIGURE X

Impact of the Shock on Log Characteristics of Nursing Home Care

This figure displays the average of 2007–2009 and 2010–2016 coefficients β_{tg} from equation (2) (Panel A) and coefficients β_t from equation (1) (Panels B and C), where outcomes y_{ctg} and y_{ct} include several facets of nursing home care. Panel A measures the log (non–age-adjusted) mortality rate per 100,000 separately among individuals who did and did not use nursing home care in the current or previous year, as well as across the whole sample of nursing home utilizers and nursing home non-utilizers. Panels B and C draw from a range of data sources that originally measure outcomes at the nursing home level. "Direct-care staff hours" is defined as the sum of the hours worked by registered nurses, licensed practical nurses, and certified nursing assistant staff per resident-day. "Highly skilled nurses ratio" is the number of registered nurse full-time equivalents divided by the number of registered nurse + licensed practical nurse full-time equivalents in nursing homes. These and other outcomes in Panels B and C are then aggregated to the CZ level, weighting by each nursing home's total number of beds, before being logged. All effects are therefore estimated at the CZ level, weighting by 2006 CZ population. Coefficients, standard errors, and confidence intervals are multiplied by 100 for ease of interpretation. Point estimates are displayed as diamonds; vertical bars indicate 95% confidence intervals, clustered at the CZ level. $N = 733$ CZs (covering $> 99.9\%$ of the 2006 Medicare population) in Panel A, with the sample of CZs limited to those with at least one beneficiary associated with nursing home utilization and one not associated with nursing home utilization in every year. $N = 716$ CZs (covering 99.8% of the overall 2006 population) in Panels B and C, with the sample of CZs limited to those with at least one nursing home.

V. Welfare Consequences of Recessions with Endogenous Mortality

To assess the quantitative importance of the estimated recession-induced mortality declines, we consider how incorporating these declines affects the welfare consequences of recessions. We augment Krebs (2007)'s calibrated model of the welfare cost of facing a lifetime of possible recessions to allow mortality to vary with the business cycle; this extension allows us to gauge the quantitative importance of our estimates of endogenous mortality on a "'standard" calibration of the welfare cost of recession risk. Our augmentation follows existing work that incorporates changes in life expectancy into welfare analyses (e.g., Becker, Philipson, and Soares 2005; Jones and Klenow 2016) by assuming that gains in life expectancy represent improvements in well-being.

V.A. Model

1. *Utility.* We consider a large N of ex ante identical agents. The representative agent's expected lifetime utility is given by:

$$U(c(t), m(t)) = \mathbb{E}_0 \left[\sum_{t=0}^{\infty} \beta^t S(m(t)) u(c(t)) \right], \tag{9}$$

where $c(t)$ is the agent's consumption in period t, $m(t)$ is the mortality rate (allowed to vary over the life cycle), and β is the agent's subjective discount rate. The cumulative survival rate $S(m(t)) = \prod_{\tau=0}^{\tau=t}(1 - m(\tau))$ is calculated using the vector of mortality rates up to time t, and life expectancy T is equal to the sum of the cumulative survival rates, that is, $T = \sum_{t=0}^{\infty} S(m(t))$.

The per period utility function $u(c)$ follows Hall and Jones (2007) and is given by

$$u(c) = b + \frac{c^{1-\gamma}}{1-\gamma}, \tag{10}$$

where b governs the willingness to pay for additional years of life. Assuming that $\beta = 1$ and that consumption is constant over time,

acute myocardial infarction or cardiac arrest (Jena et al. 2017). However, data on ambulance transport times are only available for a few states before the Great Recession, and annual, state-level information on vehicle miles traveled is inconsistently reported and of questionable reliability (Federal Highway Administration 2014).

the value of a statistical life-year (VSLY) is given by:

$$\text{VSLY} = \frac{\left(\frac{U(c,m)}{u'(c)}\right)}{T} = bc^{\gamma} - \frac{c}{\gamma - 1}, \tag{11}$$

which implies that the VSLY is increasing in c if $\gamma > 1$ (Hall and Jones 2007).

The agent receives income $y(t)$ when alive, and we assume that consumption always equals income in each period ($c(t) = y(t)$ for all t); that is, there is no saving, borrowing, or insurance.[26]

2. *Recessions and Income Processes.* Our model of recessions and income processes follows Krebs (2007) exactly. The aggregate state $\omega \in \{L, H\}$ affects the agent's stochastic income process and is drawn each period, with the probability of a normal state ($\omega = H$) given by π_H and the probability of a recession ($\omega = L$) given by $1 - \pi_H$. Income in period $t = 0$ is normalized to one, and evolves according to a stochastic process which allows for two types of persistent income shocks:

$$y_{t+1} = (1 + g)(1 + \theta_{t+1})(1 + \eta_{t+1})y_t, \tag{12}$$

where g is the exogenous growth rate in income that does not depend on the aggregate state. The first type of income shock θ_{t+1} does not depend on the aggregate state and is an iid random variable distributed as $\log(1 + \theta) \sim N\left(\frac{-\sigma^2}{2}, \sigma^2\right)$. The second type of income shock η_{t+1} represents job displacement; it has a discrete distribution that depends on the aggregate state as follows:

$$\eta_{t+1} = \begin{cases} -d^{\omega} \text{ with probability } p^{\omega} \\ \frac{p^{\omega} d^{\omega}}{1 - p^{\omega}} \text{ with probability } 1 - p^{\omega}. \end{cases} \tag{13}$$

The p^H and p^L values correspond to the approximate job separation rates during normal times and a recession, respectively, and the d^{ω} values likewise correspond to the average earnings

26. In Krebs (2007), the agent choosing consumption equal to income each period is derived as an equilibrium outcome. We instead assume $c(t) = y(t)$ at the outset to make it as easy as possible to compare our results to the original results in Krebs (2007). Based on a referee's comments, we conjecture that $c(t) = y(t)$ is also an equilibrium outcome in our extended model as long as the borrowing rate and lending rate differ due to exogenous financial intermediation costs (and the resulting interest rate spread is sufficiently large), and the intertemporal elasticity of substitution for consumption is sufficiently small. In this case, the agent will find it too costly to borrow when young to smooth consumption.

loss from job displacement, with $p^L > p^H$ and $d^L > d^H$. In other words, the risk of job loss and the reduction in income conditional on job loss are higher in the bad aggregate state. Since we assume the agent is engaging in hand-to-mouth consumption, any change in income translates one for one into a change in consumption.

3. *Welfare Cost of Recessions.* Again following Krebs (2007), we define the welfare cost of recessions Δ^{dm} as the amount the representative agent would need to be paid, calculated as a percentage of their average annual consumption, to accept the stochastic aggregate state relative to an otherwise similar economy that stays in state $\omega = H$ for all time periods:

$$\underbrace{\mathbb{E}_0\left[\sum_{t=0}^{\infty}\beta^t S(m^{\omega}(t))u((1+\Delta^{dm})y(t))\right]}_{\text{Expected Lifetime Utility with Stochastic Aggregate State}}$$

$$= \underbrace{\mathbb{E}_0^{\omega=H}\left[\sum_{t=0}^{\infty}\beta^t S(m^{\omega=H}(t))u(y(t))\right]}_{\text{Expected Lifetime Utility without Recessions}}, \tag{14}$$

where $m^{\omega}(t)$ is age-specific mortality risk in state ω (potentially endogenous to the aggregate state). If mortality is exogenous, then $m^{\omega=H}(t) = m^{\omega=L}(t) = m(t)$, and the expression simplifies to the expression in Krebs (2007), using age-specific rather than constant mortality rates. To incorporate endogenous mortality, we assume—consistent with the evidence in Figure VI—that a recession lowers the mortality rate by a constant percentage across all age groups. Thus,

$$m^L(t) = (1+dm)\cdot m^H(t) \tag{15}$$

for all t, and recall from our empirical estimates that dm (the percentage change in mortality caused by a recession) is negative.[27]

27. In this setup, when $\omega = H$ for all time periods, lifetime consumption risk is reduced (because income shocks are larger and more likely in recessions compared with normal times), and lifetime mortality is increased. Following Krebs (2007), mean consumption growth remains the same when recessions are eliminated; life expectancy decreases when recessions are eliminated because $dm < 0$. A natural extension would be to allow mean consumption growth to increase when recessions are eliminated.

4. *Intuition for the Effects of Endogenous Mortality: Simplified Model.* To build intuition for how endogenous mortality will affect the welfare cost of recessions, consider a simplified version of the above model in which the aggregate state $\omega \in \{L, H\}$ is drawn once and for all at $t = 0$. If mortality is exogenous to the aggregate economic state, individuals live for T periods; with endogenous mortality, life expectancy is T in the normal state, and $T(1 + dT)$ in the recession state. Denoting the welfare cost of a recession with exogenous mortality and endogenous mortality as Δ and Δ^{dT}, respectively, we show in Online Appendix E.1 that if we set $p^H = 0$ and take a first-order approximation of the formula for Δ^{dT}, we obtain:

$$\Delta^{dT} \approx \Delta - dT\left(\frac{\text{VSLY}}{c} + \frac{1}{\gamma - 1}\right). \tag{16}$$

This formula indicates that the welfare cost of a recession with endogenous mortality (Δ^{dT}) is equal to the welfare cost of a recession with exogenous mortality (Δ) minus the welfare benefit from the percentage increase in life expectancy (dT) from the recession.[28] The second term shows that an endogenous increase in life expectancy reduces the willingness to pay to avoid a recession by the percentage change in life expectancy (dT) times the value of this additional life span as a share of annual consumption in the normal state ($\frac{VSLY}{c}$) plus an adjustment factor $\frac{1}{\gamma-1}$.[29] This result implies that no matter how costly the recession is in terms of labor earnings, there always exists a value of the VSLY (given a change in life expectancy dT) where $\Delta^{dT} < 0$, meaning that the agent would have a positive willingness to pay for nature to draw the recession state.

The approximation formula allows us to anticipate that endogenous mortality will have a greater effect on the welfare costs

28. The additive separability—which we will find is a fairly good approximation of the full model—indicates that we do not have to incorporate any potential correlation within individuals between consumption declines and mortality changes, such as those implied by the Sullivan and von Wachter (2009) evidence that job loss itself increases mortality.

29. Intuitively, the adjustment factor comes from the fact that if $\gamma > 1$ and $b = 0$, then $VSLY < 0$, which perversely implies that individuals are willing to pay to reduce life expectancy. In Online Appendix E.2, we derive exact analytical results and a similar approximation formula for the full dynamic model developed. We find a similar approximation formula that includes an additional term coming from the income and consumption dynamics in the full model.

of recessions at older ages. To see this, note that equation (16) indicates that the impact of endogenous mortality on the welfare cost of a recession is increasing in the percent change in life expectancy (dT) caused by the recession. Next recall our empirical findings of (roughly) equi-proportional effects on the mortality rate across ages. Using the population mortality rates from the 2007 SSA life tables used in the calibration below, recessions produce larger percentage gains in life expectancy (dT) at older ages (see Online Appendix Table A.2). For example, at age 35, remaining life expectancy is 44 years, and the Great Recession increases life expectancy by 0.037%, whereas at age 65, remaining life expectancy is 18 years and the Great Recession increases life expectancy by 0.36%, that is, by 10 times as much.[30]

V.B. Calibration

We use the 2007 SSA mortality tables to calculate age-specific, unisex mortality rates for mortality in "normal" times (the $m^H(t)$ vector) and set $m^H(t) = 1$ starting at age 100. We choose a higher discount factor ($\beta = 0.99$) compared with $\beta = 0.96$ in Krebs (2007), so when we use realistic mortality rates, we end up with a welfare cost of recessions with exogenous mortality that is similar to Krebs (2007). For the mortality effect of a recession, we set $dm = -0.015$ for all ages. This calibration is based on an average 3.1 percentage point increase in the unemployment rate in a typical recession, combined with our estimates in Section III that a 1 percentage point increase in unemployment causes a 0.5% decline in the mortality rate and this percent decline was quantitatively and statistically similar across ages in the range we are modeling. We ignore potential recession-induced morbidity improvements (see Figure VIII), which would further mitigate the welfare losses associated with reduced consumption.[31]

We report results for VSLYs that correspond to two, five, or eight times annual consumption at age 35 (which is normalized to

30. For additional intuition, note that a proportional change in mortality rates has a larger relative effect on survival rates at higher (compared with lower) mortality rates. As a result, a given percentage decline in mortality rates across the age distribution leads to larger percentage gains in life expectancy (dT) at older ages.

31. As discussed in Section III.B, conditional on age, the marginal death averted has only about 6% lower counterfactual remaining life expectancy than a typical decedent, a difference sufficiently small (and statistically insignificant) that we do not account for it in our welfare analysis.

one by assumption). At an annual consumption of \$50k (roughly average expenditure for consumer units in the 2013 CEX; Foster 2015), these correspond to a VSLY of \$100k, \$250k, or \$400k, respectively. The high end of the range is based on several sources described in Kniesner and Viscusi (2019). The low end of the range follows the assumed \$100k VSLY made by, for example, Cutler (2005) and Cutler et al. (2022), and is also similar to the baseline VSLY in Hall and Jones (2007). Given an assumption for the VSLY, we compute the implied b in equation (11) for each value of γ assuming annual consumption of $c = \$50k$. Because of the assumed average annual growth in consumption ($g = 0.02$), the VSLY in the model calibration will also grow with age; however, for ease of exposition, we refer to them by the assumed value corresponding to consumption of \$50k. We discuss our calibration of mortality and the VSLY in more detail in Online Appendix E.3.

Finally, for our calibration of the income process, we follow Krebs (2007) exactly: we set $p^H = 0.03$, $p^L = 0.05$, $d^H = 0.09$, and $d^L = 0.21$, and we set $g = 0.02$, $\sigma = 0.01$, and $\pi_H = 0.5$. We normalize $y(0) = c(0) = 1$, where time 0 corresponds to someone aged 35. We report results for a range of risk aversion parameters (γ), allowing values of $\gamma = 1.5$, 2, and 2.5. To calibrate equation (14), we numerically simulate the economy for a large number of individuals ($N = 1{,}000$).[32]

V.C. Results

1. *Baseline Results.* Figure XI, Panel A shows our baseline estimates of the welfare cost of recessions for people starting at different ages between 35 and 75, with and without accounting for endogenous mortality. The figure shows results for $\gamma = 2$ and the value of b that corresponds to a VSLY of \$250k. With exogenous mortality, we find that a 35-year-old would be willing to pay 2.36% of average annual consumption for the rest of their lives to avoid the risk of all future recessions. This willingness to pay declines monotonically with age because older people have fewer years remaining and hence fewer periods in which they risk recession-induced consumption declines.

32. To increase the accuracy of our simulations, we carry out 200 independent simulations and calculate Δ^{dm} by solving equation (14) numerically in each simulation, and then calculate the simple average across the 200 simulations for each value of Δ^{dm} that we report in our figures and tables.

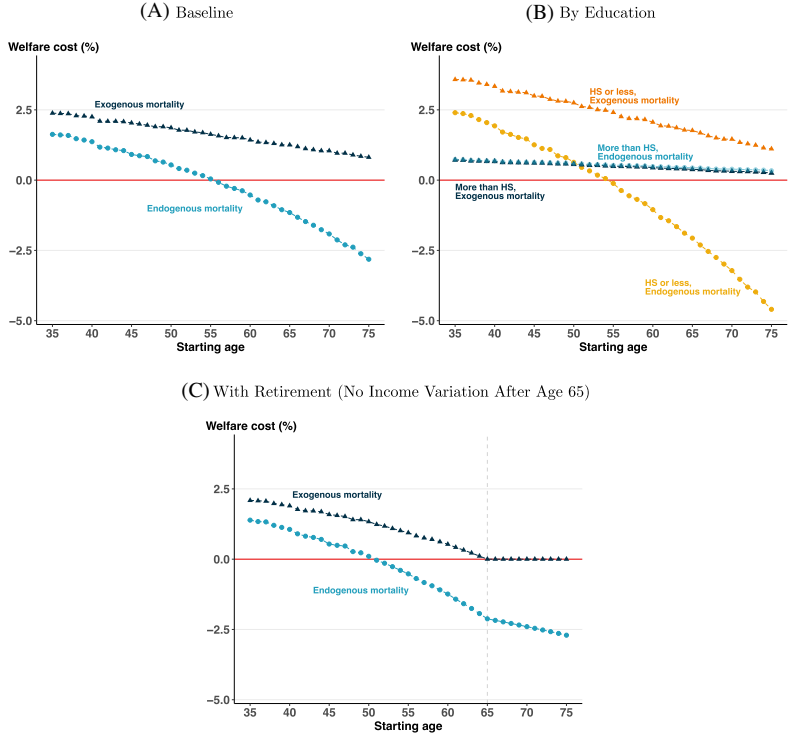

FIGURE XI

The Impact of Endogenous Mortality on the Welfare Costs of Recessions

This figure displays the welfare cost of recessions, based on equation (14), at various ages under exogenous and endogenous mortality, assuming $\gamma = 2$ and b corresponding to a VSLY of \$250k. The welfare cost is the amount an individual would need to be paid to accept the stochastic aggregate state relative to an otherwise similar economy that stays in the non-recession state for all time periods, measured as a percentage of average annual consumption. Because the true target function is monotonically decreasing in age, we rearrange the nonmonotonic estimates following Chernozhukov, Fernández-Val, and Galichon (2009) to improve efficiency. Panel A shows results for our baseline simulation. Panel B displays results when we allow for different income and mortality impacts of recessions for different education groups: those with a high school (HS) diploma or less and those with more than a HS diploma. Panel C shows results when we incorporate retirement by assuming there is no income variation for agents ages 65 and above.

Accounting for endogenous mortality lowers the welfare cost of recession at all ages and, as anticipated by the simplified model, more so at older ages. For a 35-year-old, accounting for endogenous mortality lowers the welfare cost of recessions from

2.36% of average annual consumption to 1.63%, a decline of 0.73 percentage points (or about 30%), whereas for a 45-year-old, endogenous mortality lowers the welfare cost of recessions from 2.00% of average annual consumption to 0.91% (a decline of about 55%). Starting at around age 55, accounting for endogenous mortality makes recessions welfare improving. At age 65, for example, eliminating recession risk reduces welfare by about 1.15% of average annual consumption.

Although these qualitative patterns are fairly robust, the specific numbers are naturally sensitive to our assumptions about risk aversion and the value of a statistical life-year (see Online Appendix Table A.5). Intuitively, welfare costs of recessions are increasing in the assumed level of risk aversion (γ), and the impact of endogenous mortality on these welfare costs is increasing in the assumed value of a statistical life-year. Under exogenous mortality, the welfare cost of recessions for a 35-year-old ranges from 1.74% of average annual consumption for risk aversion of 1.5% to 3.09% with risk aversion of 2.5. Holding risk aversion constant at $\gamma = 2$, accounting for endogenous mortality lowers the welfare cost of a recession for a 35-year-old by 0.30 percentage points for a VSLY of \$100k and by 1.16 percentage points for a VSLY of \$400k.

2. *Heterogeneity by Education.* Recessions tend to more adversely affect consumption among those with less education (Guvenen, Ozkan, and Song 2014; Mian and Sufi 2016). The heterogeneous mortality impacts of the recession by education (shown in Section III) provide a countervailing force that mitigates this regressive nature of recessions. To study this through the lens of our model, we allow the economic and mortality effects of recessions to vary with education based on our empirical estimates of the mortality effects of recessions by education, as well as calibrated education-specific mortality rates and education-specific job displacement probabilities and earnings losses conditional on displacement (see Online Appendix E.4 for more details).

Accounting for the differential endogenous mortality by education mitigates—and ultimately reverses—the regressivity of recessions under exogenous mortality (Figure XI, Panel B). For those with more than a high school diploma, the welfare effects with exogenous and endogenous mortality are nearly identical, since we estimate effectively no mortality effects of recessions for

this group. For those with a high school education or less, the welfare cost of recessions with exogenous mortality is substantially higher than for those with more education, reflecting the greater economic impact on the less educated group. However, accounting for endogenous mortality reduces the welfare cost of recessions for those with a high school education or less; as individuals age, the impact of endogenous mortality for the less educated becomes so large that it closes and ultimately reverses the finding under exogenous mortality that recessions are more costly for those with less education. With exogenous mortality, the welfare cost of recessions for those with a high school diploma or less is about five times as large as it is for those with more than a high school degree between ages 35 and 55. However, with endogenous mortality, the welfare costs of recessions converge for the two education groups by about age 50, and after that are less costly for those with less education.

3. *Accounting for Retirement.* The welfare analysis thus far has made the (extreme) assumption that the economic effects of recessions are the same at all ages. This assumption is unlikely to be true. Indeed, in the context of the Great Recession's local labor market shocks, the evidence suggests much smaller (or perhaps even no) economic effects for the elderly (see Rinz 2022; Online Appendix Figure A.20). To assess the potential importance of this heterogeneity in economic effects by age, Figure XI, Panel C displays welfare analyses under a different (extreme) assumption of no impact of recessions on income for agents aged 65 and over, that is, everyone at this age is retired and on a fixed income. Once again, the figure displays results for $\gamma = 2$ and the value of b that corresponds to a VSLY of \$250k, whereas Online Appendix Table A.6 shows results for a range of assumptions about risk aversion and the value of a statistical life-year.

As expected, relative to the baseline results in Figure XI, Panel A that ignore retirement and assume the same income process for all ages, welfare costs of recessions are now lower because income is unaffected by recessions starting at age 65. With exogenous mortality, welfare costs of recessions are now (mechanically) zero starting at age 65. With endogenous mortality, recessions now become welfare improving around age 50 rather than around 55 when we ignore retirement. Indeed, in a model with endogenous mortality and retirement, eliminating recession risk at age 55 reduces welfare by about 0.52% of average annual con-

sumption, and eliminating it at age 65 reduces welfare by about 2.12% of average annual consumption.

4. *Accounting for Mortality Effects of Job Displacement.* Last, we extend our model to allow for job displacements to increase mortality among the displaced and we calibrate the extended model to match the empirical results in Sullivan and von Wachter (2009), who find large effects of job displacements on mortality for high-tenure workers (see Online Appendix E.5 for more detail).[33] The welfare cost of recessions is about twice as large for workers who are ever displaced compared with workers who are never displaced. The gap in welfare costs of recessions between endogenous and exogenous mortality is larger for workers who are never displaced compared with the gap for all workers in the baseline model calibration. Intuitively, this is because our baseline mortality estimates are net of any countervailing mortality increases caused by job displacements, so once we account for the mortality effects of job displacements, recessions must cause even larger mortality reductions for nondisplaced workers to match our main empirical results. One implication is that any recession that triggers an unusually large number of job displacements of high-tenure workers is likely to have smaller reductions (or potentially even increases) in mortality in the aggregate compared with our Great Recession estimates.[34]

VI. Conclusions

We examined the impact of the Great Recession on mortality and explored its implications for the welfare consequences of recessions. We find evidence of procyclical mortality driven largely by the external health effects of reduced local economic activity; recession-induced pollution declines appear to be a quantitatively

33. In contrast to previous analyses that consider ex ante differences in welfare costs across workers with different characteristics, we now compare the ex post welfare costs of workers who are displaced versus not displaced.

34. We have followed Krebs (2007) in modeling recessions as being associated with increases in rates of job displacement. If instead recessions are primarily driven by reductions in the job-finding rate rather than by increases in the job-separation rate as documented by Shimer (2012), then the recession-induced reductions in mortality documented here may not be netting out substantial increases in mortality from job displacement of the kind documented in Sullivan and von Wachter (2009).

important mechanism. Accounting for procyclical mortality substantially reduces estimates of the welfare costs of recessions, with effects more pronounced for those with less education and for those at older ages.

These findings naturally come with some caveats. In particular, the estimates do not incorporate any national effects of the Great Recession. They do not capture any mortality effects that operate through the nationwide changes in stock markets or interest rates. We may also miss important nonmortality health impacts, particularly at younger ages where mortality may be a worse proxy for overall health.

Nonetheless, our findings suggest important trade-offs between economic activity and mortality, adding to the growing literature suggesting that GDP is an incomplete proxy for welfare (e.g., Stiglitz et al. 2009; Jones and Klenow 2016). Our results highlight the importance of considering the link between changes in economic activity and mortality when evaluating the welfare consequences of recessions or of potential public policies designed to blunt their effects. They also raise important questions for further work about whether we would find similar mortality effects (and similar mechanisms behind them) from other economic shocks, such as natural resource booms and busts (Black, McKinnish, and Sanders 2005; Feyrer, Mansur, and Sacerdote 2017), adoption of industrial robots (Acemoglu and Restrepo 2020), the North American Free Trade Agreement (Choi et al. 2024), and increased import competition from China (Autor, Dorn, and Hanson 2013).

Supplementary Material

An Online Appendix for this article can be found at *The Quarterly Journal of Economics* online.

Data Availability

The data underlying this article are available in the Harvard Dataverse, https://doi.org/10.7910/DVN/DUOILE (Finkelstein et al. 2025).

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