Lives vs. Livelihoods:
The Impact of the Great Recession on Mortality and Welfare

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November 26, 2024

Abstract

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 percent, 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 pro-cyclical mortality into a standard macroeconomics framework substantially reduces the welfare costs of recessions, particularly for people with less education, and at older ages.

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1 Introduction

Recessions damage the economy and prompt substantial government intervention. The macroeconomics literature has calibrated their welfare costs, focusing on their impacts on the level and volatility of consumption (e.g., Lucas 1987, 2003; Krebs 2007; Krusell et al. 2009). Yet recessions may also have important impacts on health. Indeed, an empirical literature in health economics has found mortality to be pro-cyclical 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 impacts 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 U.S. 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 one-percentage-point increase in a Commuting Zone's (CZ) unemployment rate between 2007-2009, its age-adjusted mortality rate fell by 0.5 percent. 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 percent for at least 10 years. These estimates imply that the Great Recession provided one in twenty-five 55-year-olds with an extra year of life.

We explore heterogeneity in the mortality impacts of the Great Recession by cause of death and by demographics. Recession-induced mortality declines are entirely concentrated among the half of the population with a high school degree or less but are otherwise pervasive across demographic groups. They appear across many causes of death, including cardiovascular disease (the largest cause of death), 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 roughly equi-proportional impacts (i.e., 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 overall mortality reduction comes from averted deaths among those ages 65 and over, roughly the same as their share of pre-recession 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 due to the Great Recession.

We investigate potential mechanisms behind the recession-induced 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—which we estimate can explain at least 20 percent 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., non-externality) mechanism, whereby reduced labor market activity frees up time for beneficial health behaviors (as in Ruhm 2000, 2005), and for two other external channels: reduced spread of infectious disease (as in Adda 2016), or improved quality of nursing home care (as in Stevens et al. 2015).

To assess the quantitative importance of mortality declines for the welfare consequences of recessions, we examine how they alter standard welfare analyses of recessions that are based solely on their impacts on consumption. Extending the Krebs (2007) model of the consumption-based welfare cost of facing a lifetime risk of recessions, we find that accounting for pro-cyclical mortality substantially reduces the welfare cost of recessions. For example, accounting for pro-cyclical 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 response to recessions. Lastly, 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, this 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 nationwide impacts of the Great Recession. Our estimates thus exclude, for example, any mortality impacts from the nationwide collapse of the stock market, or any nationwide increase in malaise.[1] In this sense, 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 impact 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

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[1] For example, exploiting variation in interview dates in the 2008 Health and Retirement Survey, McInerney et al. (2013) find that the October 2008 stock market crash caused immediate declines in subjective measures of mental health, although not in clinically-validated measures.

find evidence of a non-linear relationship between the size of the economic shock and the mortality decline. Third, our analysis focuses primarily on mortality impacts, 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 impacts at even longer time horizons.[2] Finally, we view our welfare analysis as a useful way to benchmark the magnitude of our mortality estimates, as we find that traditional analyses of the welfare costs of recessions that do not account for endogenous mortality and consider only consumption impacts may be substantially over-stated, especially for the elderly and the less educated. However, our analysis falls far short of a comprehensive analysis of the welfare impacts of recessions, given that 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; Jackson et al. 2021; Oreopoulos et al. 2012). These limitations notwithstanding, our paper sheds new light on the existence, nature, and causes of recession-induced mortality declines, and suggests that recognition of the mortality impact of recessions can have quantitatively important implications for their welfare consequences, both overall and across demographic groups.

Our paper 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 et al. 2005; Murphy and Topel 2006; Hall and Jones 2007; Jones and Klenow 2016; Brouillette et al. 2021). There has been relatively less attention, however, to incorporating cyclical fluctuations in health into welfare analyses of business cycles.[3]

Our findings 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 et al. 2006; Costa 2015; Chetty et al. 2016; Cutler et al. 2016),

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[2] For example, Schwandt and Von Wachter (2020) find that a temporarily higher state unemployment rate at the age of labor market entry (ages 16 to 22) is associated with long-run declines in earnings and increased mortality several decades later.

[3] Two exceptions are Edwards (2009) who extends Lucas (1987) to allow for cyclical mortality, and Egan et al. (2014) who contrast fluctuations in GDP to fluctuations in mortality-adjusted GDP. They reach different conclusions, with Edwards (2009) finding little effects from incorporating cyclical mortality into the analysis of business cycles, and Egan et al. (2014) finding substantial effects.

although the causal evidence of the impact of income on mortality is limited and mixed.[4] There is also evidence that job loss increases mortality (Sullivan and Von Wachter 2009), that sustained reductions in economic prospects contribute to "deaths of despair" (Case and Deaton 2021), and that 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 et al. 2019).

However, 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 paper of Ruhm (2000) have documented a negative contemporaneous association between area unemployment rates and mortality in area-year panel data.[5] However, in the decades before the Great Recession, the relationship between local unemployment and mortality weakened in the US (McInerney and Mellor 2012; Ruhm 2015). Moreover, studying almost three dozen countries over two hundred years, Cutler et al. (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 impact on health has produced mixed results.[6] When we surveyed over 300 experts in spring 2023 on the likely impact of the Great Recession on the U.S. mortality rate, 50 percent of respondents predicted that the Great Recession would increase mortality, and only 27 percent predicted a decrease; moreover, 93 percent of respondents provided a predicted impact on mortality larger than our (negative) point estimate, and 82 percent provided a prediction larger than the upper bound of our 95 percent confidence interval (Appendix A).

Our empirical approach follows in the spirit of Bartik (1991), Blanchard and Katz (1992), and especially Yagan (2019) in exploiting differences across areas of the country in their exposure

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[4] For example, Cesarini et al. (2016) find no impact from lottery winnings on adults' mortality up to 10 years later, while Dobkin and Puller (2007) and Evans and Moore (2012) find that mortality from substance abuse rises within a month when cash benefits are paid out, suggesting that the impacts of income (or at least liquidity) may differ across time horizons and populations.

[5] This relationship appears in the US (e.g. Ruhm 2000; Miller et al. 2009; Stevens et al. 2015), in Canada (Ariizumi and Schirle 2012), and in several European countries (Neumayer 2004; Granados 2005; Buchmueller et al. 2007).

[6] In the time-series, Seeman et al. (2018) find that blood pressure and blood glucose levels worsened for adults during the Great Recession, and Lamba and Moffitt (2023) find that pain increased. Yet when examining the relationship between health and the area unemployment rate using two-way fixed effect models, Currie et al. (2015) find that between 2003 and 2010, increases in the state's annual unemployment rate worsened contemporaneous physical and mental health of disadvantaged women, but may have improved the health of more advantaged women, while Strumpf et al. (2017) find that between 2005 and 2010, increases in a metropolitan area's annual unemployment rate decreased contemporaneous mortality. When measuring the Great Recession by its impact on the housing market, Currie and Tekin (2015) estimate that increases in neighborhood foreclosure rates from 2005 to 2010 increased visits to the emergency room or hospital in four states that were among the hardest hit by the foreclosure crisis, while Cutler and Sportiche (2022) find no impact of Great Recession-induced changes in house prices on the average mental health of pre-retirement adults (ages 51-61) in the Health and Retirement Survey. Closest to our own approach and results, Salinari and Benassi (2022) use an event study analysis to compare changes in life expectancy across European countries that were differentially hit by the 2008 economic crisis. They find that the countries that were harder hit experienced increases in life expectancy relative to those that did not experience an economic crisis and that these gains persisted for many years post-crisis.

to a large, aggregate economic shock. This complements the existing literature on the mortality impacts of recessions which analyzes the relationship between an area's mortality rate and its contemporaneous unemployment rate, controlling for area and year fixed effects. Relative to this literature, we offer several innovations. First, our use of a single, spatially differentiated shock allows us to examine the lag structure of the impact of the recession on mortality rather than assuming that any impact of unemployment on mortality is contemporaneous. Second, as emphasized by Arthi et al. (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 pre-recession 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 impacts of recessions from potential confounding factors that could both increase the local unemployment rate and also directly affect health. [7]

The rest of our paper proceeds as follows. Section 2 presents our data and empirical strategy. Section 3 presents our empirical estimates of mortality impacts. Section 4 investigates potential mechanisms behind these results. Section 5 explores their implications for the welfare analysis of recessions. Section 6 provides a brief conclusion.

2 Data and Empirical Strategy

2.1 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 Commuting Zones (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 Appendix B provides more detail on the underlying data sources and variable construction.

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

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[7] Examples of potential confounds include increased access to disability insurance, increased unemployment insurance generosity, or increases in the minimum wage; for each, there is evidence that they may both increase unemployment and improve health (Autor and Duggan 2003; Gelber et al. 2017; Johnston and Mas 2018; Kuka 2020; Flinn 2006; Ruffini 2022).

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 percent 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 and date of death (if any), along with 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 in the Medicare data. These data are available for all Medicare enrollees. In addition, for the approximately three-quarters of the elderly who are enrolled in Traditional Medicare, we also observe detailed, annual information about their healthcare use|including doctor visits, hospital admissions, and nursing home stays|and whether they were diagnosed with one of 20 chronic conditions in the last 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. This addresses the well-known challenge with most other US mortality data in which the numerator (deaths) and the denominator (population) come from different datasets, creating concerns about consistency between the two sources as well as potential mis-estimation 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. This allows us to address a concern with many existing estimates of pro-cyclical mortality that results may be confounded by endogenous migration in response to economic shocks (Blanchard and Katz 1992; Arthi et al. 2022). Third, we can use the (lagged) data on enrollee health conditions to analyze heterogeneous impacts 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 healthcare and estimate heterogeneous impacts by whether the individual lives in a nursing home.

Economic indicators. We use publicly available local economic indicators to trace the Great Recession across areas and years from 2003-2016. We construct the CZ-year unemployment 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 sub-sample 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 (durable and non-durable) 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.

Air pollution. We focus primarily on fine particulate matter (PM2.5), which is measured in micrograms per cubic meter ($\mu g/m^3$). We obtained granular, annual data on PM2.5 concentration levels from van Donkelaar et al. (2020) and use it to construct county-year measures of PM2.5 that cover 99.3% of (population-weighted) counties in the U.S. 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 U.S. We discuss these data—which have recently been used in several studies of pollution as an input or output (e.g., Molitor et al. 2023; Jha and Nauze 2022; Gould et al. 2023)—in more depth in Appendix B.3. In Appendix C.9, we show that when we limit to counties that also have AQS measures of PM2.5, the van Donkelaar et al. (2020) measures and the AQS measures produce similar findings.

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 the impacts of the Great Recession on 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 the impact of the Great Recession on a range of nursing home staffing measures as well as other characteristics such as patient volume and composition. Third, we draw on restricted-use data from the Health and Retirement Survey for 2002-2014—a nationally representative, bi-annual survey of older adults—to explore the impact of the Great Recession at the state level (the finest geographic information available) on self-reported measures of formal and informal care received by individuals 65 and older.

2.2 Empirical Strategy

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

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

where $SHOCK_c$ is a measure of the economic impact of the Great Recession on area c, $1(Year_t)$ is an indicator for calendar year t, ${}_c$ and ${}_t$ 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 ${}_t$'s; they measure impacts on the outcome y_{ct} in year t across areas differentially impacted by the Great Recession. In this equation (and throughout the paper unless indicated otherwise), we omit the interaction with the shock variable in 2006 so that all ${}_t$ coefficients are relative to 2006. Because population varies greatly across different areas in the US (Appendix Figure OA.1), we weight each area-year by its 2006 population, as in prior literature 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.[8] 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-hand side of equation (1). The mortality rate is defined as the share of the population in area c and 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.[9]

We also perform many analyses by sub-group, in which we estimate a fully-saturated model:

$$y_{ctg} = {}_{tg}[SHOCK_c \quad 1(Year_t) \quad 1(Group_g)] + {}_{cg} + {}_{tg} + \varepsilon_{ctg}, \tag{2}$$

where y_{ctg} is an area-year-group outcome, $1(Group_g)$ are indicators for sub-groups, ${}_{cg}$ are area-group fixed effects, ${}_{tg}$ are year-group fixed effects, and ε_{ctg} is the error term.

For both 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 impact of the Great Recession. We will investigate the plausibility of this assumption by examining the pre-trends in the event study results. Of course, finding similar mortality trends before the Great Recession in areas that are differentially impacted does not guarantee that these areas would have been on similar trends in the absence of the Great

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[8] Specifically, we add one to the mortality rate to avoid taking logs of zeros, although in practice this is never binding for the aggregate CZ-level analysis. Even when we disaggregate by cause of death or various demographics, mortality rates of zero are rare. The age group with the largest share of (population-weighted) CZs with zero deaths in a CZ-year is ages 5-14, with a share of zero deaths of only 1.6 percent; the cause of death with the largest share of (population-weighted) CZs with zero deaths in a CZ-year is homicides, with a share of zero deaths of 1.5 percent, and by race and ethnicity, it is Non-Hispanic Other, with a share of 0.8 percent. 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 Appendix C.6.

[9] We calculate the age-adjusted mortality rate in a CZ by averaging over the mortality rate in each of 19 age bins within the CZ, weighting each age bin by the national share of the population in that age bin in 2000. This 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+.

Recession, and this assumption becomes less plausible the further out in time we go past the Great Recession.

Measuring the Great Recession Shock. Our empirical strategy relies on the large spatial variation in the economic impact of the Great Recession. This has been previously leveraged to study the impact of the Great Recession on outcomes such as employment (e.g. Yagan 2019; Rinz 2022), time use (Aguiar et al. 2013), consumption (Mian et al. 2013), and educational attainment (Charles et al. 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 ${}_t$ in equation (1) captures the percent change in the mortality rate in CZ c and year t (relative to that CZ's 2006 mortality rate) associated with a one-percentage-point increase in the unemployment rate from 2007 to 2009 in that CZ.

Figure 1a 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. Yet some areas were much harder hit than others: the bottom quartile of CZs experienced an average 2.9 percentage-point increase in the unemployment rate, compared to a 6.7 percentage-point increase in the highest quartile. Areas that were especially hard hit include the so-called \sand states" of Florida, Arizona, Nevada, and parts of California (where the pre-recession housing and construction booms were concentrated) and the manufacturing states in the Midwest 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 multi-faceted economic shocks and can be parameterized in different ways. We therefore examine four different measures of the Great Recession: unemployment rate, EPOP ratio, log GDP per capita, and log house prices. The spatial variation in the 2007-2009 shock as measured by these different variables is highly, but imperfectly, correlated (Appendix Figure OA.2). In the national time series (Appendix Figure OA.3) they all flatten out between 2006 and 2007 and then worsen through 2009; however, the national aggregate trends in the 2010-2016 period look fairly different across these indicators, which is why we also consider other measures of the Great Recession besides the unemployment rate in Section 3.

Mortality Patterns Across Areas. Mortality rates vary widely across the United States (e.g. Chetty et al. 2016; Finkelstein et al. 2021). Figure 1b documents the variation in age-adjusted mortality rates across CZs in 2006, immediately prior to the Great Recession. Mortality rates were particularly high in the Southeastern United States and low in the Western United States. Figure

1c shows no correlation between the magnitude of the 2007-2009 Great Recession shock in each CZ and its 2006 (age-adjusted) mortality rate.

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 2 plots age-adjusted mortality rates from 2003 through 2016 for the CZs in the lowest quartile of the 2007-2009 unemployment 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 this 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, however, 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.

At the same time, the aggregate slowdown in mortality declines after the Great Recession shown in Figure 2 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 impact of differential local labor market shocks produced by the Great Recession, not the overall impact of the Great Recession on mortality. The slowdown in the aggregate mortality decline shown in Figure 2 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 impacts of the Great Recession on mortality that would not be captured by our empirical strategy. This is the well-known \missing intercept" problem for macro-economic counterfactuals.[10]

3 Mortality Impacts of the Great Recession

We present estimated mortality impacts overall and across different sub-populations and causes of death. After presenting initial event study results, for most of the subsequent analyses in the paper 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 Appendix D.

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

3.1 Overall Mortality Estimates

Figure 3 shows the results from estimating equation (1) for log age-adjusted mortality, with the $_{2006}$ 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 (Appendix Figure OA.4). The slightly positive pre-trend in the mortality estimates from 2003 through 2006 (also visible in Figure 2) indicates that before the Great Recession, areas that were subsequently harder hit were experiencing a slight relative increase in mortality. This opposite-signed pre-trend is consistent with our findings that recessions reduce mortality, as areas that were subsequently harder hit by the Great Recession experienced a relative rise in economic indicators in the preceding years (see Yagan (2019) as well as Appendix Figure OA.4). The opposite-signed pre-trend in the mortality estimates also suggests that by measuring the mortality impact of the Great Recession relative to the mortality level in 2006, we may be underestimating the extent of the 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 one-percentage-point increase in the local area unemployment rate between 2007 and 2009 was associated with a 0.50 percent (standard error = 0.15) decline in the area's annual age-adjusted mortality rate in 2007-2009 relative to its 2006 level. Over the following seven years (2010-2016), a one-percentage-point increase in the unemployment rate between 2007 and 2009 was associated with a 0.58 percent (standard error = 0.34) decline in the annual, age-adjusted mortality rate relative to 2006. Compared to the estimates for 2007-2009, the estimates for 2010-2016 are much less precise and are only marginally significant ($p = 0.08$ for 2010-2016, compared to $p = 0.001$ for 2007-2009); however, we cannot reject that the estimates for the two time periods are identical ($p = 0.78$).[11]

The Great Recession on average increased local area unemployment by about 4.6 percentage points between 2007 and 2009, implying that an increase in the local area unemployment rate of the magnitude of the Great Recession reduces average mortality by 2.3 percent per year, with effects persisting for at least ten years. Based on the standard population life table, the ten-year estimates suggest that 1 in 25 of 55-year-olds gained an extra year of life from this sized local shock (see Appendix Table OA.2). As another benchmark, the mortality declines from the average Great

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[11] Although there is work looking at lagged impacts 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 impacts 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 Appendix C.1, we show that a larger initial economic shock continued to translate into larger mortality declines in 2010-2016, both 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 3, these 2010-2016 estimates also lack precision, and the two areas' estimated impacts in 2010-2016 are not statistically distinguishable from zero, or from each other.

Recession shock are equivalent to the average, two-year secular mortality improvement over the half-century before the Great Recession.[12]

3.2 Unpacking the Overall Mortality Decline

Mortality rates vary substantially across demographic groups and reflect several underlying causes (Appendix Table OA.3).[13] In 2006, the elderly (65 and older) accounted for almost three-quarters of deaths, although they were only 12 percent of the population; individuals with a high school degree or less comprise about half (52 percent) of the population but account for 70 percent of deaths. Mortality also reflects several underlying causes. The two most common causes of (age-adjusted) deaths were cardiovascular disease (34 percent of deaths) and malignant neoplasms|i.e., cancer (23 percent). In this section, we examine the nature of the mortality decline across causes of death and various demographics and briefly explore impacts on morbidity. Because the patterns are often similar in the 2007-2009 period and the 2010-2016 period, we focus most of the discussion on the 2007-2009 period, where we have greater precision. We frequently summarize the average estimates; all underlying event studies are shown in Appendix D.

By cause of death. Figure 4a shows the estimated 2007-2009 mortality impacts for each of the top 11 causes of death (arranged in descending order of prevalence in 2006) and a final residual category for all other causes (Appendix Figure OA.7a does the same for estimated 2010-2016 impacts). Because the underlying mortality rates differ greatly by cause, Figure 4b 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.

Figures 4a and 4b show that cardiovascular disease accounts for both the largest share of deaths and the largest share of the estimated total reduction in deaths. A one-percentage-point increase in the 2007-2009 local area unemployment reduces the mortality rate from cardiovascular disease by 0.65 percent (standard error = 0.21). Since cardiovascular disease accounted for over one-third of total mortality in 2006, this implies that nearly half (48 percent) 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 percent of 2006 mortality, and so their contributions to the total recession-induced mortality decline are only 6.9 percent and 2.6 percent, respectively.[14]

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[12] Over the half-century preceding the Great Recession, average annual age-adjusted mortality declined by 1.1 percent per year (see Appendix Figure OA.10 and also Ma et al. (2015)).

[13] 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 Appendix B for more detail.

[14] Given the large (22.5 percent) share of deaths in the residual category, we also analyzed an alternative, standard cause-of-death grouping that groups causes of death into 10 mutually exclusive categories and yields a much smaller (3.5 percent) 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 Appendix C.2.

Most other point estimates in Figure 4a also indicate mortality declines, and no cause of death experiences a statistically significant increase in mortality. Several other causes of death—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 of death, we estimate a precise null effect of 0.02 percent (standard error = 0.11), which we interpret as reassuring that our results are picking up the causal impact of the Great Recession, rather than spurious factors correlated with the size of the Great Recession shock.

Figure 5 displays the full event-study estimates from 2003 to 2016 for several individual causes of death: cardiovascular disease, cancer, motor vehicle accidents, suicide, liver disease, and homicide (event studies for the remaining causes of death are in Appendix Figure OA.8). The effects on cardiovascular and liver mortality appear 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 the 2007-2009 period grows in magnitude in the 2010-2016 period to a statistically significant 1.7 percent decline (standard error = 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 below.

Not surprisingly in light of the recession-induced declines in both 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 the 2010-2016 period. A one-percentage-point increase in the 2007-2009 unemployment rate is associated with a 1.4 percent (standard error = 0.63) decline in deaths of despair from 2010-2016 (Appendix Figure OA.11a). Consistent with this, Case and Deaton (2017) note that there is no evidence of deaths of despair rising during the Great Recession, and interpret deaths of despair arising not from declines in income per se but rather from a more prolonged impact of cumulative disadvantage. However, our findings contrast with Pierce and Schott (2020)'s result that areas of the US more exposed to import competition from China experienced an increase in deaths of despair primarily among working-age populations.

By age. Figure 6 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. While the point estimates are larger at younger ages, they are also quite imprecise. Longer-term 2010-2016 estimates, as displayed in Figure OA.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 the years 2007-2009 is the same for ages 25-64 and for 65+ ($p = 0.30$).[15]

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 percent—of deaths averted by the Great Recession, roughly proportional to their 72.5 percent 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.

By education. Strikingly, the entire recession-induced mortality decline is concentrated among those with a high school degree or less (Panel (a) of Figure 7). Specifically, among those age 25 and over, we compare impacts separately for the roughly half of the population with a high school degree or less to those with more than a high school degree.[16] The point estimates indicate that in 2007-2009, a one-percentage-point increase in the local unemployment rate is associated with a statistically significant 0.80 percent (standard error = 0.26) decline in the mortality rate for those with high school or less, compared to a statistically insignificant 0.014 percent (standard error = 0.54) increase for those with more than high school. Although the mortality impacts by education are not statistically distinguishable ($p = 0.12$) in 2007-2009, they are statistically distinguishable ($p < 0.01$) in 2010-2016 (the point estimate is -1.48 (standard error = 0.69) for those with less education compared to 0.48 (standard error = 0.75) for those with more), as well as for the entire 2007-2016 period ($p < 0.01$; the point estimate is -1.3 (standard error = 0.56) for those with less education compared to 0.34 (standard error = 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 when we look within age groups (Appendix Figure OA.42). 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.[17] Third, given potential concerns that the differential impacts by education might in fact 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.[18] Finally, consistent with

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[15] 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 ($p = 0.01$) or for 65+-year-olds ($p = 0.03$).

[16] Due to data limitations explained in more detail in 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 impact on our estimates.

[17] When we dis-aggregate the lower education sample into those with less than a high school degree and those with exactly a high school degree, we see declines in both subgroups (Appendix Figure OA.41), although the estimates become much noisier and statistically insignificant for these sub-groups.

[18] The (population-weighted) mean state has 52 percent of the population with a high school degree 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 degree or less is 0.16, and not statistically significantly different from zero. Moreover, when we

mortality impacts that are concentrated among those with less education, we also find in the Medicare data that the mortality impacts on the elderly are much larger among the approximately 12 percent of the population on Medicaid (a proxy for low-income) in the prior year (Appendix Figure OA.43).

By gender and by race/ethnicity. We find no evidence of differential mortality impacts by gender, with nearly identical estimates for males and females (Figure 7 Panel (b)). And while recession-induced mortality declines appear to be more pronounced for non-White population groups (with particularly large point estimates for Hispanic individuals), we cannot reject equal impacts across groups in any time period (Figure 7 Panel (c)).

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 re-timing of deaths, a phenomenon often referred to as "mortality displacement" or "harvesting." Researchers tend to investigate this by looking at longer time horizons such as a month or a year (see e.g. Chay and Greenstone 2003; Deryugina et al. 2019). Displacement is therefore 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 5, 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 impact 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 six percent lower counterfactual remaining life expectancy than a typical decedent of the same age (see Appendix C.4 for more detail).

Morbidity. Like most of the literature in health economics, we focus on mortality as a measure of health since 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 Appendix Table OA.3). This raises the possibility that we are missing important non-mortality health effects at younger ages that might only translate into mortality effects decades later. These longer-run mortality impacts 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 negative mortality impacts.

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expanded our analysis to allow for calendar year effects to vary with the share of the state's population that in 2006 had a high school degree or less, we found the results unchanged (see Appendix Section C.6, and especially Appendix Figure OA.17).

In the spirit of Ruhm (2003), we therefore 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 commuting zones in the BRFSS, we estimate equation (1) at the state level.[19]

Figure 8 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 percent reduction (standard error = 0.47) of the morbidity index over the 2007-2009 period and a 1.19 percent reduction (standard error = 0.51) over the entire 2007-2016 period. This reflects declines in each measure of morbidity, although none of them is individually statistically significant. For example, in the 2007-2009 period, a one-percentage-point increase in the state unemployment rate is associated with a statistically insignificant 0.98 percent (standard error = 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 percent (standard error = 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+), although only statistically significant for the two younger age groups. Overall, we interpret these results as suggestive that morbidity is also pro-cyclical, with roughly similar magnitudes across age groups.

3.3 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, this could bias the estimated relationship between recessions and mortality. Arthi et al. (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 population that is 65 and over.[20] This raises the concern that what looks like fewer people dying in harder-hit areas might in fact reflect fewer people living in these places. One finding that mitigates against this driving our findings is that we estimate a precise

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[19] We show in Appendix C.6 that our baseline mortality estimates (Figure 3) are unchanged when switching from the commuting zone to the state level for analysis.

[20] See Appendix Figure OA.12 and also 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 out-migration (Yagan 2019; Monras 2020; Hershbein and Stuart 2024). We also show in Appendix C.5 (see especially Appendix Figure OA.13) that predicted mortality—based on gender, race, marital status, and education—does not change in areas that are more vs. less impacted by the Great Recession.

zero for declines in cancer mortality, the second leading cause of death (see Figure 4). If estimated declines in the mortality rate simply reflected unmeasured declines in population, we would expect this to show up as declines in mortality for all major causes of death.[21]

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 impact 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 et al. 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:

$$\log(m_{it}(a)) = a + {}_{t}[SHOCK_{c(i;t)} \quad 1(Year_t)] + {}_{c(i;t)} + {}_{t} + {}_{it}: \tag{3}$$

Once again, ${}_{t}$ are year fixed effects, and we cluster standard errors at the CZ level.

Figure 9a (and Table 1, row 1) shows estimates based on yearly location. The 2007-2009 estimate indicates that a one-percentage-point increase in the local area unemployment rate reduces the annual mortality rate by 0.51 percent (standard error = 0.16), which is nearly identical to our baseline estimate in Figure 3.[22]

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

$$\log(m_{it}(a)) = a + {}^{RF}_{t}[SHOCK_{c(i;2003)} \quad 1(Year_t)] + {}_{c(i;2003)} + {}_{t} + {}_{it}: \tag{4}$$

The key distinction is that we now measure both the location fixed effects ${}_{c(i;2003)}$ and the Great Recession shock $SHOCK_{c(i;2003)}$ based on individuals' location in 2003. This alleviates concerns about potential contamination from differential population flows into or out of areas that experience different shocks. The results (see Figure 9b; Table 1, row 2) continue to indicate a statistically significant decline in mortality from an increase in the unemployment rate. The 2007-2009 period

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[21] Of course, 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. For example, the share of patients who moved CZs three years before they died was 5.6 percent for cancer decedents compared to 6.0 percent for those who died of cardiovascular disease.

[22] In Appendix Figure OA.16, we show more systematically that for the 65+ population, our baseline analysis using equation (1) in the CDC data (as displayed in Figure 6), 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.

estimate indicates that a one-percentage-point increase in the local area unemployment rate reduces the annual mortality rate by 0.35 percent (standard error = 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:

$$SHOCK_{c(i;t)} \quad 1(Year_t) = a + {}^{FS}_{t}[SHOCK_{c(i;2003)} \quad 1(Year_t)] + {}_{c(i;2003)} + {}_{t} + v_{it} : \qquad (5)$$

The first stage is quite large (see Appendix Figure OA.14; Table 1, row 3), with an average coefficient of 0.95 (standard error = 0.003) in 2007-2009; therefore, not surprisingly, the reduced form is only slightly smaller than the control function estimate (see Figure 9c; Table 1, row 4) when we use the $\hat{v}_{it}$ residuals from equation (5) as an additional regressor in the following equation:

$$\log(m_{it}(a)) = a + {}_{t}[SHOCK_{c(i;t)} \quad 1(Year_t)] + {}_{c(i;2003)} + {}_{t} + \hat{v}_{it} + {}_{it} : \qquad (6)$$

The identifying assumption behind this control function approach is that while a person's 2003 location of residence may have a direct effect on their mortality|reflecting a combination of systematic variation in unobserved health determinants across the elderly in different CZs as well as any direct impact place of residence has on mortality as in Finkelstein et al. (2021)|the Great Recession shock experienced by the place a person lives in 2003 only affects their mortality through its correlation with the Great Recession shock experienced by the place they live in later years. The control function estimated mortality effect from 2007-2009 of -0:37 (standard error = 0:17) is smaller in absolute value|but not statistically distinguishable from|the estimate based on yearly residence in row 1. This difference may reflect the presence of unmeasured population declines in areas harder hit by the Great Recession. Finally, Table 1, row 5 (Appendix Figure OA.15) shows estimates based on yearly location (i.e., estimating equation (3)), limited to the 88 percent 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 (row 1).

Additional sensitivity analyses. In Appendix C.6, we explore the sensitivity of our baseline mortality estimate in Figure 3 to several alternative specifications. These include (i) the geographic unit of analysis (CZ vs. state vs. county), (ii) our choices regarding functional form for both 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 impacts of the geographically-concentrated fracking boom that occurred during our time period). The results are quite stable across these alternatives. We also engage in several additional analyses which lend support for the assumption in our baseline specification that the log mortality rate is linear in the size of the shock to the unemployment rate; for example, we find that 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.

4 Mechanisms

Recessions might reduce mortality via several potential channels. We group them conceptually 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.[23] Internal and external effects have different implications for the welfare consequences of our findings. External health benefits from reduced economic activity would suggest that recessions may have positive welfare effects that mitigate their negative welfare effects from reduced income and consumption, while the welfare implications of mortality reductions that arise from internal effects would be less clear-cut, and depend in part on whether individuals 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 in which we examine their implications for the welfare consequences of recessions.

4.1 Internal Effects

There are two main channels for internal effects discussed in the literature. First, with their increased non-labor time, the newly unemployed may have more time for self-care. This 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 pro-cyclical mortality effects emphasized in the original work by Ruhm (2000)—as well as potentially increased use of medical care.[24] Second, recession-induced consumption declines could improve health by decreasing health-harmful 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 3 are inconsistent with internal effects as the primary driver of the estimated mortality declines. First, three-quarters of the mortality reduction comes

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[23] We use the term external effects rather than externalities to indicate a broader set of health effects that come 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 seven percent of the total recession-induced mortality decline—likely reflect a mix of both internal effects (single car accidents) and external effects (multi-car accidents).

[24] Mitigating against any potential increased use in medical care is the loss in health insurance associated with employment losses and reductions in income (Coile et al. 2014).

from a reduction in elderly deaths, a group whom we estimate did not experience any direct income effects from Great Recession-induced local labor market declines (see Appendix C.8 and especially Appendix Figure OA.19).[25] Second, the time pattern of the mortality reductions—an immediate decline that does not grow larger over time (recall Figure 3)—is not consistent with an important role for changes in health behaviors; changes in exercise, diet, or smoking would be expected to impact mortality with a lag and to grow over time as health capital improves.[26]

When we look directly for evidence of internal effects, we find little evidence of a substantive role for these channels. We do not find a statistically significant impact of the Great Recession on self-reported health behaviors (Figure 8, Panel (b)) either individually or when we pool them to improve statistical power. Specifically, we examine the impact on the log share of individuals in the area who report that they currently smoke, that they smoke daily, that they currently drink, that they have consumed more than five drinks in one sitting in the past month, that they have not exercised within the past 30 days, that they did not receive a flu shot in the past year, or that they are currently overweight or obese. That said, while 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.[27] We also find no evidence of a substantively or statistically significant impact of the Great Recession on healthcare use among the elderly, measured in the Medicare data by physician visits, ER visits, or total expenditures (Appendix Figure OA.20).[28] 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 4 and Appendix Figure OA.11b), but combined this accounts for less than seven percent of the total reduction in mortality.

4.2 External Effects

We explore three main potential sources of positive external health effects from recessions suggested by prior literature: reductions in pollution (Chay and Greenstone 2003; Heutel and Ruhm 2016), increases in the quality of healthcare (Stevens et al. 2015), and reductions in the spread of infectious

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[25] 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, and much more modest and short-lived declines in elderly employment compared to impacts at younger ages (Rinz 2022).

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

[27] For example, we estimate that on average over the 2007-2009 period, a one-percentage-point increase in state unemployment from 2007-2009 decreases the share smoking by 1.2 percent (standard error 0.9 percent), increases the share excessively drinking by 0.6 percent (standard error 0.6), and decreases the share not exercising by 0.8 percent (standard error = 0.6 percent). Interestingly, although statistically insignificant, the point estimates are often similar in magnitude to those found in Ruhm (2000). Appendix Table OA.4 shows this more clearly by estimating the specification in levels and reporting the comparable estimates from Ruhm (2000).

[28] 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 percent per percentage-point increase in $SHOCK_c$) in the 2010-2016 period. Some of this may reflect compositional changes as elderly individuals who would have died are now at risk of hospitalization.

disease (Adda 2016). We find little support for a role for the latter two classes of external effects, but evidence consistent with a quantitatively important role for recession-induced reductions in air pollution, which may explain over one-third of the recession-induced mortality declines.

Reduction in air pollution. To examine the impact 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 if we measured pollution at the CZ level; we continue to measure the Great Recession shock at the CZ level since 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:

$$y_{ct} = {}_t[SHOCK_{cz(c)} \quad 1(Year_t)] + {}_c + {}_t + "_{ct}; \qquad (7)$$

where c now denotes county, cz denotes commuting zone, and $SHOCK_{cz(c)}$ is defined identically as in equation (1). Figure 10a confirms that our estimates of the impact of the Great Recession on mortality remain very similar to our baseline results in Figure 3 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 10b). Following the recent air pollution literature (e.g., Deryugina et al. 2019; Dedoussi et al. 2020; Currie et al. 2023; Heo et al. 2023), we focus on the pollutant PM2.5 (in µg/m^3) as the dependent variable when estimating equation (7). A one-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 micrograms per cubic meter (µg/m^3) (standard error = 0.039), and from 2010-2016 of 0.20 (standard error = 0.055); to put that in perspective, the average 0.18 µg/m^3 decline from 2007-2016 represents a 1.7 percent decline relative to the 10.5 µg/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 10b 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 impacts of (contemporary) pollution on (contemporary) mortality (see, e.g., Graff Zivin and Neidell 2013; Currie et al. 2014, for reviews). Second, the fact that both mortality declines and PM2.5 declines persist to the end of our study period is also consistent with this same body of evidence. 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 by the bloodstream. This can impair cardiovascular function (EPA 2004) and—perhaps more surprisingly—increase motor vehicle mortality (Burton and Roach 2023), and also reduce mental health and increase rates of suicide (Jia et al. 2018; Persico and Marcotte 2022; Molitor et al. 2023), all areas where we found statistically significant mortality declines (recall Figure 4). Fourth, our finding that the mortality declines are concentrated in the half of the population with a high school degree or less is consistent with evidence that less educated and lower-income individuals are disproportionately exposed to greater levels of air pollution both overall (Bell and Ebisu 2012; Hajat et al. 2015; Jbaily et al. 2022) as well as within cities (e.g., Hajat et al. 2013).

Assessing the quantitative importance of recession-induced pollution declines for recession-induced mortality declines is more challenging. Our three complementary approaches are suggestive of pollution being a quantitatively important channel behind the estimated mortality declines. First, we combine existing estimates from Deryugina et al. (2019) of the impact of daily PM2.5 exposure on elderly mortality with our estimates of the impact of an increase in the unemployment rate on the levels of PM2.5 to make a back-of-the-envelope calculation that the recession-induced pollution declines can explain about 17 to 35 percent of the 2007-2009 total recession-induced mortality declines, depending on which mortality estimates we use from Deryugina et al. (2019). This back-of-the-envelope calculation imposes the assumption that one year of increased exposure to PM2.5 has 365 times the impact 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 Appendix Section C.10).

Second, to more directly gauge the quantitative importance of the pollution channel, we take advantage of the fact that while counties that were harder hit by the Great Recession on average experienced a larger decline in pollution (Figure 10b), there is substantial heterogeneity in this relationship (Figure 10c). We therefore examine how much the estimated impact of the Great Recession on mortality changes when we control for changes in pollution; under the (admittedly strong) assumptions that the Great 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, e.g., MacKinnon et al. 2002; Fagereng et al. 2021). Specifically, we estimate:

$$y_{ct} = {}_t[SHOCK_{cz(c)} \quad 1(Year_t)] + {}_t[PM2.5\_SHOCK_c \quad 1(Year_t)] + {}_c + {}_t + \varepsilon_{ct}; \qquad (8)$$

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 to 2010 change in PM2.5 levels in county (with positive numbers reflecting a decline).[29]

Figure 10d shows the estimates of $_t$ from equation (8). Controlling for the pollution shock attenuates the estimated impact of the Great Recession on mortality from 2007-2009 by about 20 percent, from a one-percentage-point increase in unemployment reducing mortality by 0.50 percent (Figure 10a) to 0.39 percent (Figure 10d).[30]

Third, to isolate exogenous variation in the PM2.5 shock—as well as to correct for potential classical measurement error in the PM2.5 shock which might bias downward its estimated impact on mortality—we also 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 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 recession-induced mortality declines. Whereas with the OLS analysis, the recession-induced pollution declines explain about 20 percent of the recession-induced mortality declines from 2007-2009, with the IV analysis they appear potentially able to explain them entirely.[31]

Reduction in the spread of infectious disease. Influenza and pneumonia accounted for only two percent of deaths in 2006, and the associated mortality declines from the Great Recession are statistically insignificant (Figure 4).

Improved quality of nursing home care for the elderly. Tighter labor markets may result in improved quantity and quality of healthcare workers. This seems particularly likely for direct care workers providing home care and nursing home care for the elderly, which does not require much

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[29] We parameterize $PM2.5\_SHOCK_c$ as the negative 2006 to 2010 change because this is highly correlated with $SHOCK_{cz(c)}$ (Figure 10c), 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 Great Recession on mortality may be mediated by the estimated impact of the Great Recession on pollution.

[30] Appendix C.9 shows that we get 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 10. Using the EPA data we find no significant impacts of the Great Recession on other pollutants, specifically carbon monoxide and ozone.

[31] 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-percent reduction in TSP results in a 0.35 percent decline in the infant mortality rate—to our setting. Since we estimate a 1.5 percent reduction in PM 2.5, based on their estimate, we would expect a 0.52 percent decline in mortality, which is nearly identical to our baseline estimate.

formal training and may therefore be relatively elastically supplied.[32] Given widespread concerns about worker shortages in these sectors (e.g., Geng et al. 2019; Grabowski et al. 2023), this in turn 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 counter-cyclical.

However, we do not find any evidence for this channel. Figure 11(a) shows results from re-estimating equation (1) in the Medicare data, separately for the 7 percent of the population that was in a nursing home in any given year or the previous year and the 93 percent that was not. A one-percentage-point increase in the unemployment rate from the Great Recession reduced mortality rates by the same 0.5 percent for each group. Individuals who were in a nursing home in the current or previous year have much higher mortality rates|this 7 percent of the elderly accounts for 32 percent of their annual deaths. However, Panel (b) shows no evidence of an increase in either the number or the skill mix of nursing staff hours in nursing homes in areas where the Great Recession hit harder.[33] Panel (c) also shows no evidence of an impact of the Great Recession on nursing home occupancy rates or resident characteristics. Finally, in Appendix Section C.7, we find no evidence of an impact of the Great Recession on whether elderly individuals receive more home healthcare either from a professional or from a spouse, child or relative, although the results are fairly noisy.[34]

5 Welfare Consequences of Recessions with Endogenous Mortality

To assess the quantitative importance of the estimated recession-induced mortality declines, we consider how incorporating these mortality declines affects the welfare consequences of recessions. To do so, we augment Krebs (2007)'s calibrated model of the welfare cost of facing a lifetime of possible recessions to allow mortality to also vary with the business cycle; this allows us to gauge the quantitative importance of our estimates of endogenous mortality on a \`standard" calibration of

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[32] Recession-induced declines in the price of elderly care may be part of a larger phenomenon of recession-induced price declines which could have health benefits. For example, Stroebel and Vavra (2019) find that retail prices declined more in areas that were harder hit by the Great Recession's housing bust.

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

[34] Another potential channel for improved quality of care could be recession-induced decreases in motor vehicle traffic and hence 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 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 welfare cost of recession risk. Our augmentation follows the approach in the existing literature that incorporates changes in life expectancy into welfare analyses (e.g., Becker et al. 2005; Jones and Klenow 2016) by assuming that gains in life expectancy represent improvements in well-being.

5.1 Model

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

$$U(c(t);m(t)) = E_0\left[\sum_{t=0}^{\infty}\beta^t S(m(t))u(c(t))\right] \tag{9}$$

where $c(t)$ is the agent's consumption in period t, $m(t)$ is the mortality rate (indexed by t because it is allowed to vary by time 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 (i.e., $T = \sum_{t=0}^{\infty} S(m(t))$).

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

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

where b governs the willingness to pay for additional years of life. Assuming that $\beta = 1$ and that consumption is constant over time, the value of a statistical life-year (VSLY) is given by:

$$\text{VSLY} = \frac{\partial U(c;m)/u'(c)}{\partial T} = bc^{\sigma} - \frac{c}{\sigma-1}; \tag{11}$$

which implies that the VSLY is increasing in c if $\sigma > 1$ (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); i.e., there is no saving, borrowing, or insurance.[35]

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 $1 - \pi_H$.

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[35] 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 one of our referees' 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.

Income in period $t = 0$ is normalized to 1, and evolves according to a stochastic process which allows for two types of persistent income shocks:

$$y_{t+1} = (1 + g)(1 + \epsilon_{t+1})(1 + \eta_{t+1})y_t \qquad (12)$$

where g is the exogenous growth rate in income that does not depend on the aggregate state. The first type of income shock ϵ_{t+1} does not depend on the aggregate state and is an iid random variable distributed as $\log(1 + \epsilon) \sim N(-\sigma^2/2; \sigma^2)$. 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:

$$\eta_{t+1} = \begin{cases} -d^{\omega} & \text{with probability } p^{\omega} \\ \frac{p^{\omega} d^{\omega}}{1 - p^{\omega}} & \text{with probability } 1 - p^{\omega} \end{cases} \qquad (13)$$

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 loss from job displacement, with $p^L > p^H$ and $d^L > d^H$. In other words, both 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.

Welfare cost of recessions. Again following Krebs (2007), we define the welfare cost of recessions λ^{dm} as the amount the representative agent would need to be paid,[36] 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:

$$\underbrace{E_0\left[\sum_{t=0}^{\infty} \beta^t S(m^{\omega}(t))\, u((1 + \lambda^{dm})y(t))\right]}_{\text{Expected Lifetime Utility with Stochastic Aggregate State}} = \underbrace{E_0^{\omega=H}\left[\sum_{t=0}^{\infty} \beta^t S(m^{\omega=H}(t))\, u(y(t))\right]}_{\text{Expected Lifetime Utility without Recessions}}; \qquad (14)$$

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 above 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 6—that a recession lowers the mortality rate by a constant percentage across all age groups. Thus:

$$m^L(t) = (1 + dm)\; m^H(t) \qquad (15)$$

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[36] We sometimes refer to this as willingness to pay rather than willingness to accept; for small amounts, these are equivalent in a neoclassical model.

for all t, and recall from our empirical estimates that dm (the percentage change in mortality caused by a recession) is negative.[37]

Intuition for the impacts 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 $\in \{L, H\}$ 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 and ^{dT}, respectively, we show in Appendix E.1 that, if we set $p^H = 0$ and take a first-order approximation of the formula for ^{dT}, we obtain:

$$^{dT} \qquad dT \left(\frac{VSLY}{c} + \frac{1}{\quad 1} \right) : \tag{16}$$

This formula indicates that the welfare cost of a recession with endogenous mortality (^{dT}) is equal to the welfare cost of a recession with exogenous mortality ( ) minus the welfare benefit from the percentage increase in life expectancy (dT) from the recession.[38] 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 lifespan as a share of annual consumption in the normal state ($VSLY = c$) plus an adjustment factor $1=(\quad 1)$.[39] This 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 $^{dT} < 0$, 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 impact on the welfare costs 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 impacts on the mortality rate across ages. Using the population mortality rates from the 2007 SSA life tables used in the calibration below, this implies that

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[37] In this setup, when $\omega = H$ for all time periods, lifetime consumption risk is reduced (since income shocks are larger and more likely in recessions compared to 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.

[38] 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.

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

recessions produce larger percentage gains in life expectancy (dT) at older ages (see Appendix Table OA.2). For example, at age 35, remaining life expectancy is 44 years, and the Great Recession increases life expectancy by 0.037 percent, while at age 65, remaining life expectancy is 18 years and the Great Recession increases life expectancy by 0.36 percent, i.e., by ten times as much.[40]

5.2 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 ($= 0.99$) compared to $= 0.96$ in Krebs (2007), so that 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 is based on an average 3.1 percentage point increase in the unemployment rate in a typical recession, combined with our estimates in Section 3 that a one-percentage-point increase in unemployment causes a 0.5 percent decline in the mortality rate and that this percent decline was quantitatively and statistically similar across ages in the age range we are modeling.[41] We ignore potential recession-induced morbidity improvements (see Figure 8), which would further mitigate the welfare losses associated with reduced consumption.

We report results for VSLYs that correspond to two, five, or eight times annual consumption at age 35 (which is normalized to 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 different sources described in Kniesner and Viscusi (2019). The low end of the range follows the assumed \$100k VSLY made by e.g., 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 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 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 $g = 0.02$, $= 0.01$, and ${}_H = 0.5$. We normalize $y(0) = c(0) = 1$, where time 0 corresponds to someone aged 35. We report results for a

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[40] For additional intuition, note that a proportional change in mortality rates has a larger relative impact on survival rates at higher (compared to lower) mortality rates. This leads to larger percentage gains in life expectancy (dT) at older ages for a given percentage decline in mortality rates across the age distribution.

[41] As discussed in Section 3.2, conditional on age, the marginal death averted has only about six percent 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.

range of risk aversion parameters (), allowing values of $= 1.5, 2,$ and 2.5. To calibrate equation (14), we numerically simulate the economy for a large number of individuals ($N = 1,000$).[42]

5.3 Results

Baseline results. Figure 12a 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.[43] The figure shows results for $= 2$ 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 percent of average annual consumption for the rest of their lives to avoid the risk of all future recessions.[44] This willingness to pay declines monotonically with age since older people have fewer years remaining and hence fewer periods in which they risk recession-induced consumption declines.

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 percent of average annual consumption to 1.63 percent, a decline of 0.73 percentage points (or about 30 percent), while for a 45-year-old, endogenous mortality lowers the welfare cost of recessions from 2.00 percent of average annual consumption to 0.91 percent (a decline of about 55 percent). 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 percent 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 Appendix Table OA.5). Intuitively, welfare costs of recessions are increasing in the assumed level of risk aversion () 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 percent of average annual consumption for risk aversion of 1.5 to 3.09 percent with risk aversion of 2.5. Holding risk aversion constant at $= 2$, 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.[45]

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[42] We choose $N = 1,000$. To increase the accuracy of our simulations, we carry out 1,000 independent simulations and calculate ^{dm} by solving equation (14) numerically in each simulation, and then calculate the simple average across the 1,000 simulations for each value of ^{dm} that we report in our figures and tables.

[43] As mentioned, Schwandt and Von Wachter (2020) finds that workers who are entering the labor market (i.e., ages 16 to 22) during a recession have higher mortality several decades later relative to cohorts that enter the labor market before or after the recession. Since our welfare analysis begins at age 35, it does not account for such potential earlier-life impacts of recessions.

[44] This is very close to Krebs (2007)'s estimate of a welfare cost of recessions of 2.4 percent of average annual consumption for $= 2$. Our baseline model differs from his because it accounts for (exogenous) mortality rather than assuming infinitely lived agents with a (different than what we assume) discount factor. Without these differences, we replicate his estimates.

[45] Consistent with the analytical results presented in Appendix E.2, Appendix Table OA.5 shows that the welfare cost of recessions with endogenous mortality is well-approximated by the welfare cost of recessions with exogenous

Heterogeneity by education. Recessions tend to more adversely impact consumption among those with less education (see, e.g., Guvenen et al. 2014; Mian and Sufi 2016). The heterogeneous mortality impacts of the recession by education (shown in Section 3) provide a countervailing force that mitigates this regressive nature of recessions. To study this through the lens of our model, we allow both the economic and mortality impacts 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 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 12b). For those with more than a high school degree, the welfare effects with exogenous and endogenous mortality are nearly identical, since we estimate effectively no mortality impacts 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 degree 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.

Accounting for retirement. The welfare analysis thus far has made the (extreme) assumption that the economic impacts of recessions are the same at all ages. This 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 impacts for the elderly (see e.g. Rinz (2022) as well as Appendix Figure OA.19). To assess the potential importance of this heterogeneity in economic impacts by age, Figure 12c displays welfare analyses under a different (extreme) assumption of no impact of recessions on income for agents aged 65 and over, i.e., everyone at this age is retired and on a fixed income. Once again, the figure displays results for = 2 and the value of b that corresponds to a VSLY of $250k, while Appendix Table OA.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 12a which ignore retirement and assume

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mortality and an additively separable term based on the welfare impact of the recession-induced mortality decline. At a given age, the difference in welfare costs of recessions with exogenous mortality (column 1) and endogenous mortality under an assumed VSLY is fairly similar as we vary because varying primarily affects the welfare cost of recessions under exogenous mortality but not the magnitude of the endogenous mortality adjustment.

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

Accounting for mortality effects of job displacement. Lastly, 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 Appendix E.5 for more detail).[46] The welfare cost of recessions is about twice as large for workers who are ever displaced compared to workers who are never displaced, and the gap in welfare costs of recessions between endogenous and exogenous mortality is larger for workers who are never displaced compared to the gap for all workers in our 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 non-displaced 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 to our Great Recession estimates.[47]

6 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 pro-cyclical mortality driven largely by the external health effects of reduced local economic activity; recession-induced pollution declines appear to be a quantitatively important mechanism. Accounting for pro-cyclical 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, our estimates do not incorporate

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[46] 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 vs. not displaced.

[47] We have followed Krebs (2007) in modeling recessions as being associated with increases in rates of job displacement. However, 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 above may not be netting out substantial increases in mortality from job displacement of the kind documented in Sullivan and Von Wachter (2009).

any national impacts of the Great Recession. For example, they do not capture any mortality impacts that operate through the nationwide changes in stock markets or interest rates. We may also miss important non-mortality 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 also 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 impacts. They also raise important questions for further work about whether we would find similar mortality impacts (and similar mechanisms behind them) from other economic shocks such as natural resource booms and busts (Black et al. 2005; Feyrer et al. 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 et al. 2013).

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Figures

Figure 1: Geographic Patterns and Correlation of Unemployment and Mortality

(a) 2007-2009 Change in Unemployment Rate

(b) 2006 Age-Adjusted Mortality Rate

(c) Correlation of Pre-Recession Mortality Rates and Shock

Notes: Figure 1a displays a heat map of the unemployment shock, i.e., the change in CZ unemployment rates from 2007-2009, binned into octiles. Figure 1b 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-hand corner of each figure. Figure 1c displays a scatterplot of the 2006 age-adjusted CZ mortality rate against the 2007-2009 change in 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.

Figure 2: Age-Adjusted Mortality Rate by Severity of Shock

Notes: 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; the mean among the lowest (population-weighted) quartile of CZs is displayed in blue. 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.

Figure 3: Impact of Shock on Log Mortality

Notes: This figure displays the yearly coefficients $_t$ 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-hand 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.

Figure 4: Impact of Shock on Log Mortality, by Cause of Death

(a) 2007-2009 Pooled Estimates

(b) 2007-2009 Decomposition

Notes: Figure 4a displays the group-specific average of 2007-2009 coefficients $_{tg}$ 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 which 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 Appendix Figure OA.7. Figure 4b decomposes the contribution of each 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 Figure 4a). The blue bars indicate each cause of death's share of 2006 mortality. The orange bars present the implied share of the mortality decline accounted for by a given cause of death. To construct these orange 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 3 of -0.50%. 95% confidence intervals for these estimates, clustered by CZ, are shown as vertical lines. N=741 CZs.

Figure 5: Impact of Shock on Log Mortality, by Cause of Death: Selected Event Studies

(a) Cardiovascular Disease

(b) Cancer

(c) Motor Vehicle Accidents

(d) Suicide

(e) Liver Disease/Cirrhosis

(f) Homicide

Notes: This figure displays the yearly coefficients $_{tg}$ 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 (six of which are displayed here; the remaining six are displayed in Figure OA.8). $SHOCK_c$ is the 2007-2009 change in the CZ unemployment rate. Figure 5a displays effects on the log mortality rate from cardiovascular disease; Figure 5b from cancer; Figure 5c from motor vehicle accidents; Figure 5d from suicide; Figure 5e from liver disease; and Figure 5f 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-hand 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.

Figure 6: Impact of Shock on Log Mortality, by Age

(a) 2007-2009 Pooled Estimates

(b) 2007-2009 Decomposition

*\All" Age Group estimate is of log age-adjusted mortality

Notes: This figure displays the group-specific average of 2007-2009 coefficients $_{tg}$ 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 Appendix Figure OA.7. Figure 6b decomposes the contribution of each of these 10 age groups to the overall estimated 2007-2009 pooled reduction in mortality (i.e. the estimate from Figure 6a). 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 3 of -0.50%. 95% confidence intervals for these estimates, clustered by CZ, are shown as vertical lines. N=741 CZs.

Figure 7: Impact of Shock on Log Mortality, by Education, Gender and Race

Notes: This figure displays the group-specific average of 2007-2009 and 2010-2016 coefficients $_{tg}$ from equation (2), where the outcome y_{ctg} is 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. Impacts by education are estimated on a restricted sample and at the state level, weighting by 2006 state population. Impacts 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 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.

Figure 8: Impact of Shock on Log Self-Reported Health and Health Behaviors

Notes: This figure displays the average of 2007-2009 and 2010-2016 coefficients $_t$ 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. 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.

Figure 9: Impact of Shock on Mortality Hazard Rate: Sensitivity to Yearly vs. Baseline Residence

(a) Yearly Residence (Full Sample)
($_t$, equation (3))

(b) 2003 Residence (Reduced Form)
($^{RF}_t$, equation (4))

(c) Control Function ($_t$, equation (6))

Notes: This figure displays yearly coefficients $_t$ from equation (3) (Figure 9a), $^{RF}_t$ from equation (4) (Figure 9b), and $_t$ from equation (6) (Figure 9c), with outcome log(m_{it} (a)) defined as the log of the individual-level mortality hazard rate at age a. In Figure 9a, individuals are assigned their yearly residence, while in Figure 9b, individuals are assigned their 2003 CZ of residence. The sample reflects a panel of 2003 Medicare beneficiaries, subject to the restrictions in Table OA.7. 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-hand 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. Control function standard errors are calculated via a Bayesian bootstrap procedure with 500 repetitions. Gray bars indicate the sample size by year (which is reduced each year due to mortality); scale is determined by the secondary y-axis. The areas shaded in red correspond to the timing of the Great Recession, adopting the NBER's business cycle dating. N=6,634,999 beneficiaries.

Figure 10: Impact of Shock on Log Mortality and Pollution

(a) Log Age-Adjusted Mortality Rate

(b) PM2.5 Levels (g/m^3)

(c) Unemployment and PM2.5 Shock Correlation

(d) Log Mortality, Mediating for PM2.5 Shock

Notes: Figures 10a and 10b display the yearly coefficients ${}_t$ from equation (7), where the outcome y_{ct} is the log age-adjusted county mortality rate per 100,000 (Figure 10a) or the annual county PM2.5 level (Figure 10b), and SHOCK_c is the 2007-2009 CZ change in unemployment rate. Figure 10c 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. Figure 10d displays the yearly coefficients ${}_t$ from equation (8) in grey, where the outcome y_{ct} remains the same. ${}_t$ 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 ${}_t$ from Figure 10a are plotted in black for reference. All analyses are restricted to the 3,107 counties (representing 99.3% of the US 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 Figure 10a for reference). These estimates (and corresponding standard errors) are reported in the lower left-hand corner, along with the corresponding estimate for the entire 2007-2016 period. Coefficients, standard errors, and confidence intervals are multiplied by 100 in Figures 10a and 10d 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 Figures 10a, 10b, and 10d correspond to the timing of the Great Recession, adopting the NBER's business cycle dating.

Figure 11: Impact of Shock on Log Characteristics of Nursing Home Care

Notes: This figure displays the average of 2007-2009 and 2010-2016 coefficients $_{tg}$ from equation (2) (Panel (a)) and coefficients $_{t}$ 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 utilize 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 nurse, licensed practical nurse, 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 impacts 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.

Figure 12: Impact of Endogenous Mortality on Welfare Costs of Recessions

(a) Baseline

(b) By Education

(c) With Retirement (No Income Variation After Age 65)

Notes: This figure displays the welfare cost of recessions, based on equation (14), at various ages under exogenous and endogenous mortality, assuming = 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 non-monotonic estimates following Chernozhukov et al. (2009) to improve efficiency. Figure 12a shows results for our baseline simulation. Figure 12b 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. Figure 12c shows results when we incorporate retirement by assuming there is no income variation for agents ages 65 and above.

Tables

Table 1: Sensitivity to Current vs. 2003 Location

Regression Specification	2007-2009 Period Estimate	2010-2016 Period Estimate	2007-2016 Period Estimate
Yearly Residence ($_t$, eq. 3)	-0.513	-0.533	-0.527
	(0.161)	(0.241)	(0.210)
2003 Residence (Reduced Form) ($^{RF}_t$, eq. 4)	-0.348	-0.269	-0.293
	(0.157)	(0.233)	(0.203)
First Stage ($^{FS}_t$, eq. 5)	0.945	0.916	0.925
	(0.003)	(0.005)	(0.004)
Control Function ($_t$, eq. 6)	-0.370	-0.326	-0.339
	(0.165)	(0.251)	(0.223)
Yearly Residence (Non-Movers) ($_t$, eq. 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. Table displays the average of yearly coefficients from 2007-2009, 2010-2016, and 2007-2016. Estimates are based on coefficients $^{FS}_t$ from equation (4) for the reduced form specification, on coefficients $^{FS}_t$ from equation (5) for the first stage regression where the dependent variable is the shock experienced in a given year, and on coefficients $_t$ 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 re-weighted sample. Coefficients and standard errors are multiplied by 100 throughout for ease of interpretation. The sample is all 2003 Medicare beneficiaries, subject to the restrictions in Appendix Table OA.7. The event studies for rows 1, 2, and 4 can be found in Figure 9, the event study for row 3 can be found in Figure OA.14, and the event study for row 5 can be found in Figure OA.15. N = 6,634,999 in all rows, except for the last row where we limit to non-movers, where N = 5,838,592.

Appendices

A Expert Survey

We designed and implemented a survey of experts to assess their priors on the direction and magnitude of change in the average annual U.S. mortality rate due to the Great Recession. The survey was hosted on Qualtrics and publicized via three channels: (i) a personalized email from co-author Matthew Notowidigdo, (ii) Twitter (now known as X) posts, and (iii) the Social Science Prediction Platform. Notowidigdo sent a personalized email to each of the NBER affiliates in the Health Care, Health Economics, Economic Fluctuations and Growth, and Labor Studies programs (737 total). Notowidigdo also advertised the survey on Twitter, particularly targeting users identifying as experts in healthcare, labor markets, macroeconomics, public health, epidemiology, or medicine.

Anonymous survey responses were collected with IRB approval (MIT COUHES protocol E-4838) between March 29 and April 11, 2023. In total, we received 249 responses from the NBER group, 126 responses from Twitter, and 5 responses from the Social Science Prediction Platform.

Survey Design. The survey first asked for educational background and field of research or specialization. After providing information on the magnitude of the change in the aggregate U.S. unemployment rate during the Great Recession (i.e., \The aggregate U.S. unemployment rate increased by 4.6 percentage points from 2007-2009."), we elicited a multiple-choice prediction of whether the Great Recession increased, decreased, or did not impact the average annual mortality rate in the United States from 2007{2009. We then asked respondents for their predicted magnitude of the percent change in the annual mortality rate from 2007 to 2009 caused by the Great Recession. Finally, we elicited a multiple-choice prediction of whether the Great Recession increased, decreased, or did not impact the average annual mortality rate in the United States from 2007{2009 separately for three age bins: individuals aged 0{24, 25{64, and 65 and above. After this, we posed several free-response questions. First, we asked (in a free response box) what factors had influenced the respondent's predictions. We also asked respondents to indicate whether they had heard or seen any results from our study before the time of response, so we could exclude responses of participants with prior knowledge of our paper from our analysis. Respondents were finally invited to note any outstanding questions, comments, or suggestions.

Analysis Sample. We discarded 17 responses with no prediction for the direction of change in mortality, as well as 9 responses from participants who indicated that they were aware of early-stage results from our paper. The remaining analysis sample consisted of 354 responses: 237 NBER responses, 112 Twitter responses, and 5 Social Science Prediction Platform responses. Of these respondents, 56 percent self-identified as health economists, 20 percent as macro-economists, and 25 percent as other economists or researchers. Approximately 84 percent of respondents identified as faculty or post-doctoral researchers. Of the 354 responses, 317 responses provided a guess for the magnitude of change.

Results. Figure OA.6 shows the distribution of the direction of change in mortality rates predicted by respondents in the analysis sample. Panel (a) indicates that nearly half of all respondents predicted an increase in mortality, while Panel (c) shows differences in the predicted direction of change by age group. Panel (d) shows heterogeneity in predictions by respondent subfield: macroeconomists are more likely to predict an increase than health economists.

Panel (b) describes the distribution of the predicted direction and magnitude of change in the cumulative distribution function (with answers below the 5th percentile and above the 95th percentile trimmed from the figure for visual clarify, though these respondents are included in the following statistics). We find that 93 percent of the respondents provided a predicted impact on mortality larger than our (negative) point estimate, and 82 percent provided a prediction above the upper bound of our 95 percent confidence interval.

B Data

Throughout, we restrict our analysis to people in the 50 states and the District of Columbia from 2003 to 2016.[48]

B.1 Mortality Data

CDC Data. The CDC mortality data are derived from state death certificates which in turn are completed by physicians, coroners, medical examiners, and funeral directors (Office of Disease Prevention and Health Promotion n.d.). Information on how to apply for the CDC restricted-use microdata is available at https://www.cdc.gov/nchs/nvss/nvss-restricted-data.htm .

These microdata offer several key advantages over the publicly-available CDC mortality data, which can be found at https://wonder.cdc.gov/wonder/help/ucd.html . In particular, the public data report only coarse age bins, do not allow an analysis of mortality for combinations of sub-groups (e.g. certain causes of death within a certain age group), omit certain demographics such as education, and suppress mortality information for cells with less than ten deaths; this threshold can prevent the publication of county-level data for groups with low mortality rates (e.g. younger individuals), or small population shares (e.g. less common causes of death or demographic groups). We confirmed that we can replicate our aggregate findings in the public-use data.

To compute mortality rates using the CDC microdata, we use population data from the National Cancer Institute's Surveillance Epidemiology and End Results (SEER) program. More information about these data can be found here: https://seer.cancer.gov/popdata/ . The SEER population estimates are a modification of the US Census Bureau's intercensal population estimates. As noted by e.g., Ruhm (2015), they are designed to provide more accurate population estimates for intercensal years. In practice, we have verified that our results are not sensitive to our choice of the SEER or Census population measure.

To measure cause of death, we use the cause of death recodes from the Department of Vital Statistics' List of 39 Selected Causes of Death for the "underlying cause of death" variable. This gives a single, mutually exclusive cause of death for each decedent; for further information see "Part 9 - Understanding Cause-of-Death Lists for Tabulation Mortality Statistics" from the National Vital Statistics System Instruction Manual (National Center for Health Statistics 2023). We then follow the NCHS-provided hierarchy of classifications and collapse the 39 causes to 23 by combining all malignant neoplasms into a single category and all forms of major cardiovascular disease into a single category.

Medicare data. We use the Medicare data to analyze mortality for a 20 percent random sample of the near-universe of Americans 65 and older.[49]

We observe death records, annual zip code of residence, and demographic variables for all Medicare enrollees, regardless of whether they are enrolled in Traditional Medicare or Medicare Advantage. Medicare Advantage is a program in which private insurers receive capitated payments from the government in return for providing Medicare beneficiaries with health insurance. Insurance claims (and hence healthcare

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[48] In the CZ-level analyses, we exclude eight counties in Alaska (five of which did not exist until 2014) because it is not straightforward to map them to CZs; these counties account for less than 0.006 percent of the population.

[49] Although the data also contain information on under-65 Medicare enrollees—in particular, recipients of Social Security Disability Income (SSDI)—we exclude these individuals from our analysis since both the number and composition of SSDI recipients change during recessions (Carey et al. 2022).

utilization measures or health measures which are based on diagnoses recorded by physicians) are not available for enrollees in Medicare Advantage.

The death records that we use in the Medicare data come primarily from the Social Security Administration. Specifically, we use the mortality information in the Master Beneficiary Summary File. More information on the source of the mortality data in this file can be found in Jarosek (2022). The Social Security Administration in turn receives death reports directly from sources "including family members, funeral homes, financial institutions, postal authorities, States and other Federal agencies" (Social Security Administration 2023).

For the approximately three-quarters of the elderly who are enrolled in Traditional Medicare, we also observe detailed information about their healthcare use and health diagnoses. Specifically, we observe doctor visits, emergency room visits, inpatient hospitalizations, and nursing home stays; we also observe annual indicators capturing the presence of 20 specific chronic conditions that the patient could have been diagnosed with, such as lung cancer, diabetes, or depression.[50]

We analyze two primary Medicare samples. First, we analyze a panel of 2003 Medicare enrollees aged 65-99 in 2003; we make a few other minor sample restrictions described in Appendix Table OA.7. We use this enrollee-level panel to explore the sensitivity of our findings to accounting for potentially endogenous movements of individuals across areas. Second, we analyze a repeated cross section of individuals aged 65-99 each year, often further restricting to individuals who were enrolled in Traditional Medicare in the previous or current year; again, we make a few other minor sample restrictions described in Appendix Table OA.8. Summary statistics for each of these samples can be found in Appendix Table OA.9. In addition, Appendix Figure OA.16 illustrates that the estimates of the impact of the Great Recession on mortality for the 65+ population are similar when using Medicare data as when using CDC mortality data, and are also similar across our repeated cross-section of Medicare beneficiaries (N=13,705,511) and our panel of 2003 beneficiaries who are then followed forward through 2016 (N=6,634,999).

B.2 Economic Data

We obtain monthly data on the county-level unemployment rate and counts of employment from the Bureau of Labor Statistics' Local Area Unemployment Statistics (LAUS, available at https://www.bls.gov/lau/). Following Yagan (2019), we construct CZ-year estimates of the unemployment rate in each month by summing the number of unemployed individuals across all counties in each CZ-month and taking the average across all months in a year, and then dividing by the annual population (for ages 16+) in that CZ from the Census (available at https://www2.census.gov/programs-surveys/popest/datasets/2010-2019/counties/asrh/).[51] Similarly, to construct CZ-year estimates of the employment-to-population (EPOP) ratio, we sum the monthly employment counts across counties within a CZ and then average the monthly employment counts across months within a year; we transform these annual CZ employment counts into estimates of the CZ-year EPOP ratio using the same annual counts of CZ-level population aged 16+ from the Census as the denominator.

For our baseline $SHOCK_c$ measure in equation (1), we also follow Yagan (2019) and measure it as the percentage point change in the CZ unemployment rate between 2007 and 2009. We obtain this

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[50] Chronic conditions are measured for those enrolled in Traditional Medicare for one to three prior years (depending on the condition). We focus on the 20 chronic conditions that have a look-back period of one year.

[51] Intercensal estimates are obtained by measuring population change since the previous Census, based on births, deaths, and migration. See https://www2.census.gov/programs-surveys/popest/technical-documentation/methodology/2010-2020/methods-statement-v2020-final.pdf

directly from the replication package in Yagan (2019), who calculates annual CZ unemployment rates in the manner we do above—i.e. by summing monthly county-level counts of the unemployed (and also the number of people in the labor force) across counties within the CZ to construct monthly CZ unemployment rates which he then averages across months to obtain annual estimates.

We obtain annual county-year real GDP from the Bureau of Economic Analysis (see the CAGDP1 time series at https://apps.bea.gov/regional/downloadzip.cfm). We aggregate this to the CZ-year level by summing the real GDP and population for all counties within a CZ, then dividing to obtain real GDP per capita for that year. For a sub-sample of counties for which it is available, we also obtain annual county-year house pricing data from the Federal Housing Finance Agency's yearly House Price Index (HPI) public release. This data release is available at https://www.fhfa.gov/DataTools/Downloads/Pages/House-Price-Index-Datasets.aspx. The HPI is a weighted repeat-sales index of single-family house prices with mortgages purchased or securitized by Fannie Mae or Freddie Mac since 1975 (see Bogin et al. (2019) for details). We average these county-year data to the CZ-year level, weighting the counties with observed HPI by their 2006 population from the SEER data.

State-level household total consumption expenditures on (durable and non-durable) goods and services are from the Personal Consumption Expenditures (PCE) surveys published by the Bureau of Economic Analysis. Consumption data are provided directly at the state and year level as the sum of all expenditures within a state for different expenditure categories in the PCE. These data are available at https://apps.bea.gov/regional/downloadzip.cfm.

Finally, we use data from the Current Population Survey (CPS), administered jointly by the U.S. Census Bureau and the Bureau of Labor Statistics, to study the impact of the Great Recession on earnings (overall and by education group) as well as income (overall and by age group). The CPS is a monthly survey administered to a nationally representative sample of individuals aged 15 years or older and not in the Armed Forces. The survey excludes institutionalized people, such as those in prisons, long-term care hospitals, and nursing homes. To match the yearly structure of our data, we use the CPS survey results from March of each year. We measure individual annual earnings, adjusted to 2015 dollars using the CPI. We also measure inflation-adjusted individual annual income (censoring negative values to be zero); individual income includes wage income, retirement income, unemployment insurance, and business and farm income (see https://cps.ipums.org/cps-action/variables/INCTOT#comparability_section).

B.3 Air Pollution Data

For our primary measure of PM2.5, we use the granular, annual data on PM2.5 concentration levels from van Donkelaar et al. (2020). The authors generate estimates of PM2.5 by feeding observations of visual occlusion from satellite images into a chemical transport model, and combining the predictions with ground-level pollution monitor measurements from the EPA's Air Quality System (AQS) database using a Geographic Weighted Regression model. These data have recently been used by economists studying such topics as the impact of pollution on suicide (Molitor et al. 2023) and the impact on air pollution of US embassy air-quality tweets (Jha and Nauze 2022) or of transitioning from gas to electric cooking (Gould et al. 2023). They have also been used to construct a national Census-linked individual-level dataset of pollution exposure (Voorheis et al. 2023).

The estimates from van Donkelaar et al. (2020) are extremely granular, generated over a 0.01 degree by 0.01 degree grid (roughly 1 km at the equator), and we use the annual frequency mean PM2.5 estimates for North America. Thanks to the high granularity, we are able to assign each grid block to a Census

Block Group and weight the grid blocks in a county by their 2010 Census population when averaging PM2.5 to the county level. Doing the same aggregation for every year of our sample period, we construct a county-year level PM2.5 data set that covers 99.3% of (population-weighted) counties in the U.S.

As a check on the van Donkelaar et al. (2020) measures of PM2.5, as well as to be able to look at other air pollutants, we also use data on air pollution obtained directly from the EPA's Air Quality System (AQS) database (available at https://www.epa.gov/aqs). We average pollution monitor readings within a monitoring site to the site-year level, weighting by the number of daily pollution readings for each monitor if there are multiple monitors at the same site. We then average these data to the county-year level, weighting sites by the number of daily pollution readings from the monitors within those sites. Although the EPA monitor data are commonly used to measure air pollution (e.g. Isen et al. 2017; Deryugina et al. 2019; Alexander and Schwandt 2022), they are limited in several respects for our purposes. First, our estimates of the impact of pollution in contributing to recession-induced mortality declines may be biased downward by classical measurement error, since the monitor data produce rather coarse geographic measures of air pollution, whose effects on health may be much more local than the county (Currie et al. 2023). Second, a well-known limitation is that the pollution monitoring network is sparse (Fowlie et al. 2019). Indeed, to study the impact of the Great Recession on air pollution or the impact of recession-induced air pollution on mortality using the EPA data, we limit our analysis to the approximately two-thirds of (population-weighted) counties which have a pollution monitor in 2006 and in 2010. Moreover, even when monitors exist in counties, they are unlikely to be representative of the whole county, both because pollution can vary a lot within a county and because there is evidence that monitors are endogenously placed to avoid the worst-polluting spots (Grainger and Schreiber 2019). Reassuringly, we show in Appendix C.9 below that, when we limit to counties in which both satellite and EPA measures of PM2.5 are available, results look very similar.

B.4 BRFSS Data

The Behavioral Risk Factor Surveillance Survey is an annual telephone survey administered to approximately 400,000 individuals aged 18 or older across the United States. The survey modules elicit demographic information and responses to a series of questions covering self-reported health, health behavior, and healthcare access. These data are collected by state departments of health in coordination with the CDC. Survey questions are divided between core modules (which are in principle always asked) and optional modules (which may or may not be asked, according to state discretion). The BRFSS is designed to produce representative estimates of these responses at the state level. Initial sampling is conducted via random digit dialing, and each data release includes post-stratification weights.

We analyze the BRFSS sample from 2003-2016. For each variable of interest, we generate the state-year mean according to the BRFSS final respondent weights. Our analysis then proceeds at the state-year level, weighting estimates by the 2006 SEER state population.[52]

The BRFSS methodology was refined in 2011 to incorporate reports from cell phone users and to improve survey weighting. While this change increased the reach and representation of the survey, it also generates a potential confound when comparing raw survey tabulations from before and after the 2011 adjustment. For several variables (share who smoke or drink; share with very good or excellent health; share obese) we observe these changes reflected as sharp, though generally small, changes in the aggregate

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[52] Note that the core questionnaire was not asked in Hawaii in 2004; otherwise, our BRFSS sample includes all 50 states and the District of Columbia.

time series from 2010-2011. However, our empirical approach includes year fixed effects which should take a first step towards mitigating these effects, and we are comforted by the observation that our event study results do not include similar discrete jumps at 2011.

BRFSS Variable Definitions. Our analyses examine several BRFSS measures of self-reported health, health behavior, and healthcare. We describe each self-report and (if necessary) our modifications in detail below:

- **Poor subjective health:** We construct an indicator for whether the respondent describes their current state of health as less than "Very Good" or "Excellent" (i.e. "Good", "Fair", or "Poor").
- **Mental health:** We construct an indicator for whether the respondent reports any days out of the past 30 in which their "mental health, which includes stress, depression, and problems with emotions," was not good.
- **Ever had diabetes:** Respondents report whether a doctor has ever told them that they have diabetes.
- **Currently have asthma:** Respondents report whether a doctor has ever told them that they have asthma. If they respond affirmatively, they are subsequently asked if they still have asthma. We define "currently having asthma" as an affirmative response to both questions (i.e. we define this variable as zero for both individuals who have never had asthma and those who were previously diagnosed but do not currently have asthma).
- **Weight:** From respondent self-reported height and weight, the BRFSS constructs BMI (as weight in kilograms divided by the square of height in meters). Following BRFSS documentation, we define individuals as overweight or obese if they have a BMI greater than or equal to 25, and as obese if they have a BMI greater than or equal to 30.
- **Currently smoke/smoke daily:** Respondents are asked if they have smoked at least 100 cigarettes before in their life. If they respond affirmatively, they are asked if they currently smoke every day, some days, or never at all. From these two questions, the BRFSS defines an indicator for whether the respondent currently smokes cigarettes (i.e. every day or some days, vs. not smoking). From the same set of questions, we define "smokes daily" as an indicator for whether the respondent smokes every day (unconditionally—i.e. smoking daily instead of some days or never).
- **Currently drink/binge drinking:** We report an indicator for whether individuals currently drink (alcohol), which corresponds to a question in the BRFSS asking whether respondents have had any alcoholic beverage in the past 30 days. Respondents are subsequently asked how many times in the past 30 days they have consumed at least five drinks (for men) or four drinks (for women). The BRFSS then constructs an indicator for binge drinking in the past month, defined as one for a positive response to having 4/5 or more drinks at a time in the past month and as zero for individuals who have not (whether they report any alcohol consumption or not).
- **Exercise:** We lift directly from the BRFSS a question asking whether respondents "participate[d] in any physical activities or exercises such as running, calisthenics, golf, gardening, or walking for exercise" during the past month.

- Flu shot: Similarly, respondents report whether they had a flu shot in the past 12 months, and we lift this variable directly.
- Health insurance: We define currently having health insurance as an affirmative response to "Do you have any kind of healthcare coverage, including health insurance, prepaid plans such as HMOs, or government plans such as Medicare?" This question is asked of all respondents.

Appendix Table OA.10 shows the means of these various outcomes overall and separately by age group.

B.5 Nursing home data

We use the Online Survey Certification and Reporting (OSCAR) and Certification and Survey Provided Enhanced Reporting (CASPER) databases to measure nursing home staffing. In particular, we use the data compiled by the Shaping Long-Term Care in America Project at Brown University (LTCFocus 2023), which compiles the OSCAR/CASPER data with aggregate facility-level measures from CMS's Minimum Data Set (MDS). These are facility-level administrative data obtained from certification inspections of nursing homes; they are the same data that were previously used by Stevens et al. (2015) to study the impact of recessions on the quantity and quality of nursing home staffing. We take CZ-level means of each measure, weighting facility observations by that facility's total number of beds in that year. We then take the log of these CZ-level aggregates before proceeding with analysis. In particular, we measure:

- The number of nursing home staff hours. We observe the number of direct care staff hours per resident-day, where direct care workers include registered nurses, licensed practical nurses, and certified nursing assistants.
- The skill mix of nursing home staff hours, defined as the ratio of registered nurse full-time equivalents divided by the number of registered nurse and licensed practical nurse full-time equivalents in nursing homes.
- The volume of nursing home patients, measured by the occupancy rate (occupants per bed).
- The average age of nursing home residents.
- The share of residents that are female.

B.6 Health and Retirement Study Data

Data and Sample The Health and Retirement Study (HRS) is an ongoing longitudinal study of individuals in the United States born between 1924 and 1965. The HRS is sponsored by the National Institute on Aging (grant number NIA U01AG009740) and is conducted by the University of Michigan. Survey respondents are divided into cohorts based on the year in which they were first interviewed; the HRS began interviewing cohorts in 1992 and has added additional cohorts four times since, in 1998, 2004, 2010, and 2016. Households are sampled according to a multi-stage area-probability sampling procedure which first draws Primary Sampling Units (metropolitan areas, counties, or groups of counties), Census Divisions within these units, and then households from within those divisions (Lee et al. 2021). Over the survey's history, eligibility has been determined by screeners of housing units, the Medicare enrollment files, or some combination of the two (HRS Staff 2011). The HRS over-samples Hispanic and Black individuals as well as residents of Florida.

The HRS interviews these sampled respondents and their spouses/partners (if applicable), regardless of whether spouses are themselves age-eligible. Each interview covers demographic, financial, health, cognitive, housing, employment, and insurance data for respondents, their households, and their spouses. Our data comes from the RAND HRS Longitudinal File, a dataset based on the HRS core data. This file was developed at RAND with funding from the National Institute on Aging and the Social Security Administration.

We obtained access to a restricted-use version of the HRS that allows us to observe state of residence for interviews conducted bi-annually between 2002 and 2014. Our analyses therefore focus on a bi-annual, repeated cross-section of HRS respondents from 2002-2014. We restrict each year's sample to respondents from households where both the respondent and their spouse (if present) are at least 65 years old in that year. Note that this permits individuals to \age in" to the sample, even if they were previously interviewed for the HRS and would have been excluded based on this age criteria. We do not consider households interviewed outside of the 50 US states and the District of Columbia.

HRS Variable Definitions We analyze four measures of home care in the HRS. First, we examine the number of individuals from whom respondents report receiving help with their activities of daily living (ADLs), with their instrumental activities of daily living (IADLs), or with managing their finances, henceforth referred to as \helpers". We also examine separate indicators for whether respondents report any paid helpers, unpaid helpers, or either.

Respondents in the HRS are first asked whether they have ever received help with ADLs, IADLs, or finances during any period and, if affirmative, they are asked who helped them. In a separate section of the survey, respondents are then asked for details about each of these helpers, including the frequency of help in the past month and whether each helper was paid in the past month, except those who are employees of institutions. RAND then takes this \helper list" and computes the number of helpers as the number of individuals on the helper list who helped in the past month and are not employees of institutions (because the \number of employees of an institution cannot be accurately counted due to the nature of institutional care" (Bugliari et al. 2022)).

The RAND HRS Longitudinal File reports directly the number of helpers each respondent reports in the past month (including zero) and the number of helpers who were paid (again including zero). From these reports, we additionally define any helpers as an indicator for whether the number of helpers is non-zero or zero; any paid helpers as an indicator for whether the number of paid helpers is non-zero or zero; and any unpaid helpers as an indicator for whether the number of helpers is (strictly) greater than the number of paid helpers.

C Additional Details on Empirical Analyses

C.1 Lag Structure of the Impact of the Economy on Mortality

Although there is work looking at lagged impacts 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 try to investigate possible lagged impacts 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 impact. Specifically, we estimate:

$$y_{ct} = \sum_{q \in \{L,H\}} \beta_{qt}\left[SHOCK_c \times 1(Year_t) \times 1\{Recovery_{q(c)}\}\right] + \alpha_c + \gamma_t + \varepsilon_{ct}, \tag{17}$$

where $1\{Recovery_{H(c)}\}$ is an indicator that CZ c has an above-median 2010-2016 recovery rate among CZs in the same decile of $SHOCK_c$, and $1\{Recovery_{L(c)}\}$ is an indicator that it has a below-median recovery. Because the unemployment rate is a notoriously challenging measure of recovery—as worker exit from the labor force can produce a decline in unemployment without any corresponding increase in the employment-to-population ratio—we measure the recovery by the change in the area's EPOP ratio between 2010-2016, rather than the change in the area's unemployment rate.[53] For symmetry, we also measure the initial economic shock ($SHOCK_c$) in equation (17) by the percentage point change in the area's 2007-2009 EPOP ratio. Using the EPOP ratio instead of the unemployment rate for the measurement of $SHOCK_c$ in equation (1) produces very similar results to our baseline specification (see Appendix Figure OA.21). Estimation of equation (17) thus exploits the substantial dispersion across CZs in the rate of the 2010-2016 EPOP recovery *within* each decile of the 2007-2009 EPOP shock (see Appendix Figure OA.22). The above- vs. below-median recovery CZs (conditional on the size of the initial shock) are distributed fairly evenly across the United States (Appendix Figure OA.23) and display no systematic relationship with pre-recession demographic characteristics (Appendix Table OA.11).

The top two panels of Figure OA.24 show the results of estimating equation (17) using the EPOP ratio as the dependent variable. While in above-median recovery CZs (Panel (b)) the EPOP ratio has returned to the pre-recession level by 2016, in the below-median recovery CZs (Panel (a)) it has regained only about half of the estimated decline. The bottom two panels show the impact of the 2007-2009 shock on log mortality, separately for CZs that subsequently experienced below-median recovery (Panel (c)) and above-median recovery (Panel (d)). As expected, the impact of the Great Recession is similar for these two types of CZs in the 2007-2009 period, with a one-percentage-point decline in the EPOP ratio associated with a 0.4 percent decline in mortality.

The results for 2010-2016 are more puzzling. Qualitatively, we see similar mortality declines persist in both the above- and below-median recovery areas, despite the fact that the above-median recovery areas have completely recovered by the end of our study period, while the below-median places have only partly recovered. Lagged mortality impacts of past economic declines could potentially explain the persistent mortality results in the above-median areas. However, we would also expect *larger* (in absolute value) mortality declines in the below-median recovery areas; consistent with this, the point estimates indicate

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[53] For example, in the national time series, the unemployment rate starts to recover after 2009 and by 2016 is essentially back to pre-recession levels, while the three other economic indicators remain substantially below their 2006 levels (Appendix Figure OA.3). The same phenomenon appears when comparing more vs. less affected areas (Appendix Figure OA.4).

that over the 2010-2016 period, the below-median recovery places do experience an approximately 20 percent larger mortality decline (relative to 2006) than the above-median recovery places. However, like the overall analysis in Figure 3, the estimated 2010-2016 mortality declines are not statistically significant in either the above- or below-median recovery areas (nor are they statistically distinguishable from each other). The imprecision of the results therefore admits a range of possibilities, making firm conclusions difficult.

C.2 Mortality Impacts by Cause of Death

Our baseline analysis by cause of death examines the 11 most common causes of death in the ICD10 39-group classification and groups all other deaths into the residual category. Since this residual comprises roughly a quarter of 2006 deaths, as well as a quarter of the estimated 2007-2009 mortality reduction, we also examined results for an alternative cause of death categorization with a smaller residual. The alternative definition uses the top 10 most common causes of death as given by the ICD10 chapters (which approximately correspond to disease of various body systems) classification system and groups the rest into a residual category that now comprises only 3.5% of 2006 deaths.

Table OA.12 displays the confusion matrix for the two different classifications of causes of deaths. It shows a generally very close correspondence between the baseline and alternative cause of death categories; the exceptions are the alternative category of respiratory disease, which encompasses both the lower respiratory disease and influenza/pneumonia baseline causes, and the alternative category of external causes, which encompasses the motor vehicle accident, suicide, and homicide baseline causes; one of the reasons we prefer our baseline specification is that it breaks out these|very different|external causes. However, Table OA.12 also shows the advantage of these alternative cause of death categories: most of our baseline residual is allocated to one of the alternative causes.

Figure OA.9 displays both the share of 2006 deaths each alternative category comprised and each category's share of the 2007-2009 mortality reduction (analogous to Figure 4b for the baseline cause of death categories).

Table OA.13 shows the 2007-2009, 2010-2016, and 2007-2016 estimates for mortality reduction by baseline cause of death, and Table OA.14 shows the same thing by alternative cause of death. The causes in Table OA.14 are ordered to be roughly analogous to the order of causes in Table OA.13 (ex. Cardiovascular disease is the first cause in Table OA.13, and circulatory diseases is the first cause in Table OA.14), but as described above, the baseline and alternative causes of death do not exactly map one to one.

C.3 Mortality Impacts by Education

The NCHS mortality data contain information on education, which we can use to obtain the number of deaths in each education-age-location-year bin. However, the SEER population data do not contain population counts by education. To construct the population denominator for mortality impacts by education, we therefore turn to the American Community Survey (ACS). The ACS is sent by the U.S. Census Bureau to approximately 3.5 million U.S. households each year, and it collects information including participants' age, years of education, and location. Since we use the publicly available ACS data, the only non-suppressed location variable is each individual's state of residence; as a result, we conduct the analysis by education at the state level, using ACS data from 2003-2016. We also limit our analysis to individuals aged 25 and over so that we can observe completed education.

Specifically, we compute the number of surveyed individuals who fall into categories defined by five-year age bins (with the first bin ages 25-29, and the last bin 85+), education bins (high school or less, some college, and college or more), and state of residence. Since the ACS only surveys a subset of Americans, we then compute the share of individuals in each category (adjusting for survey weights) and multiply by the total population in each year according to the SEER data to obtain an estimate of the number of individuals falling in each age/education/state/year bin. Combining these data with the NCHS data allows us to produce a state-year panel of age-adjusted mortality rates for each education bin, which we use to conduct our analysis.

Note, however, that the education level is missing for a small share (4.5 percent) of deaths in the NCHS data. Furthermore, these deaths are concentrated in specific state-years. We therefore drop any state for which at least one state-year is missing education information for over 45 percent of its deaths. In practice, this means that we exclude Georgia, New York, Rhode Island, and South Dakota from the sample; together, these four states account for 52.6 percent of the deaths with missing education information. The state-year with the next largest share of deaths with missing education information after excluding these four states is Maine in 2011, and this share is just 10.6 percent.

These sample restrictions do not meaningfully affect our results. As seen in Figure OA.41a, which estimates our main specification at the state-level using 47 states, the 2007-2009 period estimate is -0.66 (standard error = 0.25), which is very similar to the corresponding estimate of -0.62 (standard error = 0.25) using all 50 states and the District of Columbia and all ages in Table OA.15.

C.4 Health Status of Marginal Life Saved

To analyze the health status of the marginal life saved, we closely follow Deryugina et al. (2019). Specifically, we turn to the Medicare data and limit our analysis to the approximately three-quarters of the overall Medicare sample that is on Traditional Medicare in every month of the prior year and for whom, as explained in Section 2, we can therefore observe measures of health. This analysis is thus, by necessity, limited to the elderly population; as we have seen, they account for three-quarters of the estimated mortality decline. We estimate an auxiliary model of mortality as a function of individual demographics and health conditions at the beginning of the year and use this model to predict counterfactual, remaining life expectancy for each individual in each year.

C.4.1 Predicting Remaining Life Expectancy

The rich, detailed information on individual demographics and health conditions in the Medicare data allows us to estimate a mortality model and use it to generate predicted counterfactual remaining life expectancy for each decedent in our data. Specifically, in addition to age, race, and gender, the Medicare data contain measures of individual health conditions derived from health diagnoses recorded in claims data.[54]

To estimate remaining life expectancy, we follow the standard approach in the literature (e.g. Olshansky and Carnes 1997; Chetty et al. 2016; Finkelstein et al. 2021), and adopt a Gompertz specification in

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[54] As documented by Song et al. (2010) and Welch et al. (2011), these claims-based measures of health reflect both the enrollee's underlying health as well as a large measurement error component that varies systematically by place, as places that tend to treat patients more aggressively are also more likely to diagnose and record underlying conditions. However, since our analysis looks at within-area differences in the impact of the Great Recession by measured health, such place-specific measurement error is unlikely to bias our analyses.

which the log of the mortality hazard rate for individual i in year t ($\log(m_{it})$) is linear in age a:

$$\log(m_{it}(a)) = a + X_{i(t-1)} + {}_{it} \tag{18}$$

We estimate this prediction model on the mortality experience of 2002 Medicare enrollees who were also enrolled in Traditional Medicare (Part B) in 2001. We do so for three different definitions of $X_{i(t-1)}$: (i) no covariates (i.e. only age), (ii) demographic covariates (race, gender), and (iii) demographic covariates plus chronic condition indicators, where we restrict our attention to the 20 chronic conditions that have a look-back period of one year. We also specify a prediction model with constant remaining life expectancy, which we set to the average of 2002 enrollees' predicted remaining life expectancies in the specification with no covariates (only age).

We then use the estimates from equation (18) to predict remaining life expectancy for each patient-year in the sample from 2003-2016 where, recall, the sample is limited to individuals who are alive at the beginning of the year and were on Traditional Medicare for all months of the previous year. Specifically, given the Gompertz assumption, we can estimate remaining life expectancy conditional on being alive at age $a = A_0$ as:

$$LE_{it} = \int_{A_0}^{\infty} \exp\left[\frac{e^{X_{i(t-1)}}}{} (e^{a} - e^{A_0}) \right] da. \tag{19}$$

As expected, as we add additional covariates to the prediction model, the predicted counterfactual remaining life expectancy among those who die in the following year declines (see Appendix Figure OA.46). If we assume that decedents would have had the remaining life expectancy of the average Medicare enrollee, we predict their counterfactual remaining life expectancy to be 11 years. Accounting for age—i.e. that the typical Medicare decedent is older than the typical Medicare enrollee—reduces that counterfactual remaining life expectancy by about 30 percent, to 7.9 years. Further accounting for demographics and chronic health conditions reduces counterfactual remaining life expectancy by another 20 percent, to 6.5 years.

C.4.2 Analyzing decline in life-years lost

We define life-years lost for each individual in the data at the beginning of the year to be zero if they survive, and to be equal to their predicted remaining life expectancy at the beginning of the year if they die that year. We then re-estimate equation (1) with the dependent variable now the log number of life-years lost in CZ c and year t per hundred thousand beneficiaries. Specifically, we define life-years lost per hundred thousand beneficiaries (LYL_{ct}) as

$$LYL_{ct} = 100{,}000 \quad \frac{\sum_{i \in S_{ct}} LYL_{it}}{|S_{ct}|}; \tag{20}$$

where LYL_{it} is the life-years lost for individual i in year t and S_{ct} represents the set of beneficiaries living in CZ c during year t.

The key object of interest is how our estimate of the impact of the Great Recession on life-years lost varies as we use increasingly rich covariates to predict each individual's remaining life expectancy. Appendix Table OA.16 shows the results from re-estimating equation (1) for the dependent variable log life-years lost ($\log(LYL_{ct})$). For comparison, Column (1) shows the results where the dependent variable is the log of the (non age-adjusted) mortality rate per 100,000; it indicates that a one-percentage-point increase in the unemployment rate reduces the mortality rate by 0.6 percent. As expected, we see in Column (2) that estimating equation (1) with log life-years lost as the dependent variable yields very similar results to Column (1) if we assume that decedents have the same remaining life expectancy of the average Medicare enrollee. In particular, Column (2) suggests that a one-percentage-point increase in the unemployment rate reduces the life-years lost in a CZ by about 0.62 percent. Columns (3) through (5) explore how this changes as we incorporate richer covariates into our prediction of counterfactual remaining life expectancy among decedents. Column (3) shows that accounting for age reduces the estimate of life-years lost by about 8 percent to 0.57 percent. Accounting for differences in demographics and health conditions further reduces the estimate of life-years lost by about another 5 percent (to 0.54 percent); none of these differences are statistically distinguishable.

The analysis in Appendix Table OA.16 suggests that the Great Recession-induced mortality reductions came from individuals who had only slightly lower counterfactual life expectancy than typical decedents of the same age; since we are comparing how the percent change in life-years lost varies across sets of controls, we are effectively normalizing by the \typical" decline in life-years due to mortality or, in other words, to decedents. We can also ask whether the marginal life saved has substantially lower life expectancy than a typical Medicare enrollee of the same age. To do this, we re-estimate equation (1) with the level of life-years lost (LYL_{ct}) as the dependent variable. Table OA.17 shows the results. Column (1) indicates that, for a one-percentage-point increase in the Great Recession shock, we observe a mortality rate reduction of 29 per 100,000 in the set of beneficiaries covered by Traditional Medicare in the previous year. Columns (2) through (5) then show our estimates of the impact of the Great Recession on life-years lost in this population, as we use more and more covariates to predict counterfactual remaining life expectancy for decedents. Assuming that each decedent's counterfactual remaining life expectancy is equal to the predicted mean across all Medicare enrollees in this sample (11 years), we obtain a decline in life-years lost of 326 per 100,000 beneficiaries (that is, 326 life-years gained). Incorporating age reduces the decline in life-years lost substantially, to 212 (column 3) or by about 35 percent. Further incorporating demographic and health covariates reduces the decline in life-years lost by another 20 percent, to 167; in other words, the marginal life saved has about 80 percent the remaining life expectancy of a typical Medicare enrollee of the same age, when modeling life expectancy off of age, demographics, and chronic conditions. This is not surprising as the decedent population in general has a lower remaining life expectancy than the general population.

C.5 Assessing Potential Demographic Changes Associated with the Great Recession

We examined whether changes in the demographic composition of the population associated with the timing and magnitude of the Great Recession Shock could spuriously drive findings of recession-induced mortality declines. A priori, we considered this unlikely since areas that were harder hit by the Great Recession experienced an increase in the age of the population (Appendix Figure OA.12). However, to investigate other potential demographic changes correlated with mortality rates, we use data from the National Health Interview Survey (NHIS) from 2003-2016, which contains information on individuals' year of death, gender, race, marital status, and education, to estimate a logistic regression that predicts an individual's probability of death in a given year based on those demographic characteristics.[55] Since all

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[55] We do not use age as a characteristic because our main outcome is the age-adjusted mortality rate and, because, as noted, the increase in age in areas harder hit by the Great Recession biases against our finding of a recession-induced mortality increase.

of these variables are also available in the American Community Survey (ACS), we use the estimates from this regression to generate a predicted mortality rate for every person-year in the ACS from 2003-2016, and then aggregate this predicted mortality measure to the state-year level. Figure OA.13a shows that there is dispersion in our predicted state-year mortality rate.

Figure OA.13b shows the estimates of the impact of the Great Recession from equation (1) on both the log mortality rate (dashed gray line) and the log predicted mortality rate (black line); both analyses are run at the state-year level rather than the CZ-year level because we are only able to generate predicted mortality at the state level. Unlike the recession-induced decline in actual mortality, we find no impact of the Great Recession on predicted mortality, suggesting that any changes in demographic composition associated with the Great Recession shock are unlikely to contribute to the estimated mortality declines in more impacted areas.

C.6 Additional Sensitivity Analysis

We explored the sensitivity of our baseline estimates in Figure 3 to a number of alternative specifications. Table OA.15 summarizes the findings. The first row replicates our baseline estimates from estimating equation (1), as shown in Figure 3; subsequent rows present one-off deviations from this baseline. We continue to focus our discussion primarily on the 2007-2009 period estimates where we have greater precision. These results are quite stable.

Geographic unit. Estimates are very similar when we re-estimate equation (1) at the state level or county level instead of the CZ level (Panel (a)). For example, for the 2007-2009 period, our baseline estimate is that a one-percentage-point increase in the CZ unemployment rate decreases mortality by 0.50 percent (standard error = 0.15). At the state level, the estimate increases slightly to 0.62 percent (standard error = 0.25), and at the county level, it decreases slightly to 0.49 percent (standard error = 0.10).

Functional form. Estimates are also very similar across functional form choices (Panel (b)). If we replace the dependent variable with the age-adjusted mortality rate in levels in year t, we obtain very similar results. For example, for the 2007-2009 period, we estimate that a one-percentage-point increase in the CZ unemployment rate decreases mortality by 3.7 deaths per 100,000 (standard error = 1.0) or about 0.47 percent relative to 2006 mortality of 790 per 100,000. The next row shows what happens if we estimate our specification with a Poisson regression using the age-adjusted mortality rate in levels as the outcome. Specifically, we estimate:

$$y_{ct} = \exp(\quad {}_t[SHOCK_c \quad 1(Year_t)] + \quad {}_c + \quad {}_t); \tag{21}$$

where all variables are defined as in equation (1). The 2007-2009 estimate of -0.45 percent (standard error = 0.14) is very similar to our baseline result.

Sample of CZs. Results are robust to dropping various subsets of CZs from the analysis (Panel (c)). A particular concern is that the fracking boom occurred during our time period of interest and was concentrated in particular geographic areas—such as parts of Texas, Oklahoma, North Dakota, Colorado, Pennsylvania, and West Virginia—where it may have had direct impacts on economic activity and mortality (Bartik et al. 2019). Reassuringly, the results are robust to omitting the 56 CZs (representing about nine percent of the population) that include any county defined as \treated" by fracking in the Bartik et al. (2019) analysis of the impacts of fracking. To further probe concerns that different parts of the country may be differentially experiencing other shocks or secular trends, the next row shows results when we allow each of the year fixed effects to differ across each of the nine census divisions. The estimated 2007-2009 average impacts are somewhat attenuated to -0.38 percent (standard error = 0.14), but still show statistically significant mortality declines.[56] Finally, because population is very right-skewed across CZs (see Appendix Figure OA.1), the penultimate row of Panel (c) confirms the robustness of our results to dropping the ten most populous CZs.

Measurement of Shock. We explored the sensitivity of our results to measuring the Great Recession shock by the change in the area's employment-to-population (EPOP) ratio between 2007 and 2009 (Panel (d)), rather than the change in the unemployment rate as done by Yagan (2019) in his analysis of the Great Recession and in prior analyses of the relationship between recessions and mortality (e.g., Ruhm 2000, 2003, 2005; Stevens et al. 2015). Using the EPOP ratio instead of the unemployment rate for the measurement of $SHOCK_c$ in equation (1) produces very similar results to our baseline specification (see Appendix Figure OA.21)

Linearity in Shock. Our baseline specification assumes that the log mortality rate is linear in the size of the shock to the unemployment rate. This is a substantive as well as statistical assumption. The average shock during the Great Recession was much higher than in a typical recession; if mortality effects are linear in the size of the shock, this would increase our confidence that our mortality findings generalize to more \typical" recessions.

We therefore undertook four additional analyses to assess whether it is a reasonable approximation to assume linearity of mortality impacts in the size of the economic shock. First, the last row of Table OA.15 Panel (c) shows that the baseline results are somewhat larger (in absolute value) if we drop the top and bottom decile of CZs by the size of the shock.

Second, we allowed the impact of the shock to vary based on whether the CZ experienced an above- or below-average shock. Under the assumed linearity, we should find the same impact of the shock on both sub-samples. To examine this, we adopt a modified version of equation (17), with $Recovery_{q(c)}$ substituted for an indicator of whether a CZ experienced an above or below (population-weighted) median CZ 2007-2009 unemployment shock. The results are shown in Appendix Figure OA.29; consistent with the linearity assumption, we find very similar estimated impacts of the shock in both sub-samples.

Third, we relaxed the linearity assumption by replacing the $SHOCK_c$ variable in equation (1) with indicators for which quartile of the (population-weighted) CZ unemployment rate shock distribution the CZ is in. Specifically, we estimate

$$y_{ct} = \sum_{j=2}^{4} \left[{}_{t}^{(j)} \, SHOCKQ_c^{(j)} \quad 1(Year_t) \right] + {}_{c} + {}_{t} + {}_{ct}; \qquad (22)$$

where $SHOCKQ_c^{(j)}$ is an indicator for the jth quartile of the 2006 CZ population-weighted CZ unemployment rate shock; we omit the 1st quartile (with a mean shock of 2.89) and report estimates of ${}_t^{(2)}$,

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[56] CZs are assigned to states (and resulting Census Divisions) according to the state with the plurality of the CZ population. To further probe sensitivity to the set of CZs, Appendix Table OA.18 presents results when each of the nine census divisions is dropped in turn.

${}^{(3)}_t$, and ${}^{(4)}_t$. The results are shown in Appendix Figure OA.26. We find that the impacts on mortality are increasing monotonically in the quartile of shock, although these effects are not perfectly linear in the average size of the shock by quartile. The results indicate that CZs in the second quartile (mean shock of 4.00) experience a substantially larger mortality decline than those in the first quartile, and CZs in the fourth quartile (mean shock of 6.66) experience an even larger mortality decline than those in the second quartile, but that CZs in the third quartile (mean shock of 5.18) experience roughly similar mortality declines to those in the second quartile.

Fourth, Appendix Figure OA.27 plots the relationship between the (population-weighted) average of $SHOCK_c$ in each ventile of the CZ-shock distribution against the average change (for CZs in that ventile) in the log mortality rate between 2006 and its average across several post-period years: 2007-2009, 2010-2016, and 2007-2016. The relationship is noisy but looks roughly linear. This provides some support for the linear specification in equation (1).

Fifth and finally, Appendix Figure OA.28 displays our main event study where $SHOCK_c$ is the percent unemployment change from 2007-2009 relative to the 2007 unemployment rate, rather than the 2007-2009 unemployment shock in percentage points. Under this alternative nonlinear functional form, we continue to see negative coefficients post-2007, with a 2007-2009 period effect of -0.026 (SE = 0.005) and a 2010-2016 period effect of -0.044 (SE = 0.009). These estimates imply a similar effect size to our main specification.[57]

Endogenous migration. Section 3.3 examines the sensitivity of our main results to endogenous migration, using Medicare panel data to examine the impact of the GR shock while assigning individuals to either their yearly CZ of residence or their CZ of residence in 2003; these results are presented in Table 1 and Figures 9 and OA.15. However, these results all adopt an individual-level Gompertz specification, in contrast to our primary CZ-level two-way fixed effects model. Therefore, as an additional robustness check, we first apply our main specification in equation (1) to Medicare panel data, collapsing individuals to CZs using their yearly CZ of residence. Figure OA.16 shows the results from applying this specification to the elderly population in the CDC data (panel (a)) and the elderly population in the Medicare data (panel (b)), indicating that the analysis is not affected by the change in data.

We then adopt a revised specification which instead applies the GR shock corresponding to individuals' 2003 CZ of residence. Specifically, we estimate:

$$\widehat{SHOCK}_{c(i;t)} = \sum_{I_{c;t}} \frac{SHOCK_{c(i;2003)}}{N_{ct}} \tag{23}$$

$$y_{ct} = {}_t[\widehat{SHOCK}_{c(i;t)} \quad 1(Year_{c(i;t)})] + {}_{c(i;t)} + {}_t + "_{c(i;t)} \tag{24}$$

where $c(i;t)$ refers to an individual's yearly CZ of residence, $I_{c;t}$ is the set of individuals living in CZ c in year t, $c(i;2003)$ refers to an individual's 2003 CZ of residence, and N_{ct} is the sample size for a given CZ-year. $\widehat{SHOCK}_{c(i;t)}$ therefore represents the mean 2003 CZ shock experienced by individuals in a given CZ-year. Equation (24) in effect represents the CZ-level impact of a GR shock using 2003 CZ of residence, while still applying fixed effects based on yearly CZ. This equation can therefore be understood

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[57] Specifically, the mean population-weighted CZ has a 2007 unemployment rate of 4.76 percent; therefore, for the typical CZ, a 1 percentage point increase in unemployment is equivalent to a $1/4.76 = 21$ percent increase. Thus, a 0.026 percent decrease in mortality from a 1 percent increase in unemployment roughly implies a $0.026 \times 21 = 0.546$ percent decrease in mortality from a 1 percentage point unemployment increase. This is quite similar to our main specification estimate of -0.501.

as a supplementary check for endogenous migration.

Figure OA.16 panels (c) and (d) present event studies following this specification, using first the yearly CZ (panel (c)) and then the 2003 CZ (panel (d)).[58] Results look similar, suggesting that our results are not meaningfully impacted by endogenous migration.

Heterogeneity by state-level educational attainment. In Appendix Figure OA.41, we first show that our main effect is heterogeneous by education level, with large negative effects among those with a high school degree or less and null effects among those with more than high school. However, one may worry that these results may be driven by the fact that the areas more or less impacted by the Great Recession may have systematically more or less educated populations. This may in turn spark broader concerns about confounding trends biasing our results.

To test directly for this phenomenon, we adopt an alternative specification in which we allow for year fixed effects to vary by state-level educational attainment. Specifically, we estimate:

$$y_{ct} = \beta_t[SHOCK_c \times 1(Year_t)] + \alpha_c + \gamma_t + \delta_t[x_{s,2006} \times 1(Year_t)] + \varepsilon_{ct}, \qquad (25)$$

where $x_{s,2006}$ is the proportion of individuals in 2006 aged 25+ in a given state with a high school education or less, and this proportion is interacted with state fixed effects. Recall that all education-related results are presented at the state level, as this is the level at which we have education data.

Figure OA.17 presents event studies both under our main specification in equation (1) and using equation (25). The two event studies are highly similar, indicating that our main education findings are driven by within-state variation rather than between-state variation.

C.7 Impacts on Paid and Unpaid Home Care (HRS)

We examined informal care provision, as this might increase in tight labor markets when adult children have lower opportunity costs of time.[59] We therefore turn to the Health and Retirement Survey (HRS) to estimate impacts of the Great Recession on the elderly's receipt of paid or unpaid home care. The data and variables are described in more detail in Appendix B.6.

We estimate a variant of our baseline estimating equation (1), where the unit of observation is now the individual, the Great Recession Shock is measured at the state level, and the data include even years only between 2002-2014 (as we are only able to match respondents to their state, not CZ, and the HRS is only administered every two years). Specifically, we estimate (for continuous outcomes):

$$y_{it} = \beta_t[SHOCK_{s(i,t)} \times 1(Year_t)] + \alpha_{s(i,t)} + \gamma_t + \varepsilon_{it}, \qquad (26)$$

where $s(i,t)$ indexes the state of respondent i in year t, observed from 2002-2014, and y_{it} is respondent i's report of e.g. the number of individuals who helped them last month. $SHOCK_{s(i,t)}$ denotes the 2007-2009 change in the state unemployment rate in state $s(i,t)$.

When we then turn to binary outcomes (e.g. any helpers in the past month, with helpers defined as anyone assisting with activities of daily living, instrumental activities of daily living, or finances), we

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[58] Note that the panel (c) and (d) results are based on the sub-sample of Medicare enrollees in panel (b) whom we can observe in 2003 and follow forward, while the panel (b) results use the repeated cross-section of Medicare data without requiring they be present in the data in 2003.

[59] Mommaerts and Truskinovsky (2020) find evidence using a state-year panel that informal care provided by adult children is counter-cyclical, but it appears to be offset by the pro-cyclicality of spousal informal care, with no overall effect on the amount of informal care received by the elderly.

instead estimate a logistic regression model of the form:

$$\ln \frac{P(y_{it}=1)}{1 \quad P(y_{it}=1)} = {}_t[SHOCK_{s(i;t)} \quad 1(Year_t)] + {}_{s(i;t)} + {}_t + {}_{it} : \tag{27}$$

As before, we report ${}_t$ in our coefficient plots (not the odds ratios $e^{\,t}$).

We estimate the regression at the individual level since the means of many of these variables at the state-year level would be zero, complicating a log specification. We weight each estimate by the HRS respondent weights,[60] and cluster standard errors at the state level.

Appendix Figure OA.30 displays the results. It shows no evidence of an impact of the Great Recession on any of the measures of receipt of paid home care or receipt of unpaid home care.

C.8 Impacts on Income, by age, in the CPS

We leverage individual-level data in the CPS on income and age to estimate the impact of the Great Recession on income by age. We estimate both our standard OLS analysis of the impact of the Great Recession (equation 1) at the state-year level, and also an individual-level Poisson regression of the impact of the Great Recession with the following Poisson regression:

$$y_{it} = \exp \quad {}_t \; SHOCK_{s(i;t)} \quad 1(Year_t) \; + {}_{s(i;t)} + {}_t + {}_{e(i;t)} + {}_{a(i;t)} + {}_{g(i)} \; ; \tag{28}$$

where y_{it} denotes income for individual i in year t; $SHOCK_{s(i;t)} \quad 1(Year_t)$, ${}_{s(i;t)}$, and ${}_t$ are defined just as in equation (1), where $s(i;t)$ gives the state individual i lives in during year t. The individual-level analysis allows us to reduce noise by including demographic controls; specifically we include fixed effects for the individual's detailed education category (${}_{e(i;t)}$), age (${}_{a(i;t)}$), and gender (${}_{g(i)}$). The educational categories we include are none, grades 1-4, grades 5-6, grades 7-8, grade 9, grade 10, grade 11, grade 12 (no diploma), high school, some college, Associate's degree (vocational), Associate's degree (academic), Bachelor's degree, Master's degree, professional school degree, or PhD. The coefficients of interest are the estimates of ${}_t$. We estimate equation (28) using the survey weights provided by the CPS and cluster standard errors at the state level.

Appendix Figure ?? shows the results. Panels (b), (d), and (f) display the estimates of ${}_t$ from estimating the individual-level Poisson equation (28), where the outcome is income for all individuals aged 25+, income for individuals aged 25-54, and income for individuals aged 65+. Panels (a), (c), and (e) show analogous results from estimating equation (1) by OLS for the state-year outcome log average income for individuals in the relevant age group. Reassuringly, the results look similar using either approach but, as expected, the estimates with the individual-level specification are noticeably more precise. We find that the recession-induced income reductions are limited to individuals ages 25-64 (Panel (d)); we find no evidence of recession-induced income declines for the 65 and older population (Panel (f)).

C.9 Impacts of the Great Recession PM2.5 and on Other Pollutants Using EPA Monitor Data

Figure OA.31 shows that the satellite measures of PM2.5 and the EPA monitor measures produce similar findings when we limit to the overlap counties. Unlike the results using satellite data from the full country

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[60] These person-level weights are designed to align the HRS waves with population estimates from the American Community Survey 1-year Public Use Micro Samples (Lee et al. 2021). Of note, respondents who are institutionalized (i.e. live in a nursing home), live outside the United States, or are out of the HRS age range have weights of zero.

(Figure 10b) which indicate that counties harder hit by the Great Recession experience a persistent decline in PM2.5 levels through the end of our study period, Figure OA.31a shows that using the EPA monitor data, counties that were harder hit by the Great Recession experienced declines in PM 2.5 through 2010, after which pollution levels began to increase and by 2016 had returned to pre-recession levels. Reassuringly, the discrepancy between these two findings appears driven by the limited set of counties for which EPA monitor data is available; Figure OA.31b, which uses satellite data but only in the sample of counties covered by the pollution monitor data, displays a similar pattern of an initial decline in PM2.5 in harder-hit counties, followed by a return to pre-recession levels by the end of the sample period. Not surprisingly, therefore, controlling for the PM2.5 shock has a similar mediating effect on impact of the Great Recession shock on mortality using either the EPA monitor data or the satellite data in the same counties (Figures OA.31c and OA.31d).

We also used the EPA monitor data to explore the impact of the Great Recession on other pollutants, as Heutel and Ruhm (2016) also find significant roles for decreased carbon monoxide (CO) and ozone (O_3) in explaining countercyclical mortality, in addition to decreased PM2.5. We found no effects of the Great Recession on either carbon monoxide or ozone. We summarize the results here.

Due to limitations in the number of counties for which EPA monitor data is available for these pollutants, we focus our analysis on two samples for each pollutant: (1) the sample consisting of all counties for which we can measure the change in pollution between 2006 and 2010 for that specific pollutant and (2) the sample consisting of all counties for which we can measure this change for all three pollutants. These samples consist of 524 counties (comprising 64.4 percent of the 2006 population) for PM2.5, 179 counties (comprising 44.1 percent of the 2006 population) for CO, 730 counties (comprising 70.7 percent of the 2006 population) for O_3, and 137 counties (comprising 39.0 percent of the 2006 population) for the overlap sample.

Appendix Figure OA.32 shows the results from estimating equation (7) with different pollutants as the dependent variable. In the left-hand column we show results for the maximum sample of counties that we have for that pollutant, and in the right-hand column for the overlap sample of counties where we observe all three pollutants. As previously seen, we find that areas more exposed to the Great Recession shock experience declines in PM2.5 (Panels (a) and (b)). However, we find no evidence of recession-induced changes in carbon monoxide (Panels (c) and (d)) or in ozone (Panels (e) and (f)). Not surprisingly, therefore, if we run the mediation analysis in equation (8) using the 2006-2010 change in a different pollutant as the mediating factor, it has little impact on our estimates of the impact of the Great Recession on mortality (see Appendix Figure OA.33). When we include all three pollutants' changes between 2006-2010 interacted with year fixed effects as potential mediators, we find that it reduces the estimated impact of a one-percentage-point increase in the unemployment rate on mortality by about one-third for the 2007-2009 period, which is quite similar to the mediating effect we find for PM 2.5 alone in the same sample (see Appendix Figure OA.32, panel (b)).

(see Appendix Figure OA.34).

C.10 Gauging the Impact of Estimated Pollution Reductions on Mortality

We use the evidence from the existing quasi-experimental literature on the impact of air pollution on mortality to perform a back-of-the-envelope calculation of what size mortality declines we would expect from our estimate of the recession-induced pollution decline. A key challenge for this exercise is that we estimate a multi-year pollution decline, while the literature has focused primarily on relatively short-run variation in pollution exposure, and studied impacts over relatively short time horizons, typically less than one year, and sometimes over a matter of days (see e.g. EPA (2004), Graff Zivin and Neidell (2013), and Currie et al. (2014) for reviews, or Deryugina et al. (2019) for more recent work).[61] Moreover, it is a priori unclear whether the impact of a prolonged change in pollution exposure will be proportional, larger, or smaller than a temporary change. A persistent change might have proportionally smaller effects if harvesting is a primary driver of the short-run impacts, or it might have proportionally larger effects if impacts accumulate over time and/or it is harder to avoid exposure to pollution when it persists over a longer period of time; Barreca et al. (2021) find evidence consistent with the latter.

These issues notwithstanding, we attempted to use the existing literature on the relationship between one-day changes in pollution exposure and short-run mortality changes to benchmark the potential importance of the Great Recession-induced air pollution decline for mortality. Specifically, we use the estimates from Deryugina et al. (2019) of the impact of PM2.5 on elderly mortality, combined with our estimates of the impact of an increase in the unemployment rate on the levels of PM2.5. Deryugina et al. (2019) estimate that a one microgram per cubic meter increase in PM2.5 exposure for one day causes 0.69 additional deaths per million elderly individuals over a three-day window (see their Table 2 Panel (b) column 1), and more than double that over a one-month window (see their Figure 6). We make the (heroic) assumption that one year of increased exposure to PM2.5 has 365 times the impact on mortality as one day of increased exposure. Under this assumption, our estimate in Figure 10b that a one-percentage-point increase in the unemployment rate is associated with an average annual PM2.5 reduction of 0.14 micrograms per cubic meter over 2007-2009 suggests that the pollution declines associated with a one-percentage-point increase in unemployment would cause a decline in elderly deaths of between 4 and 8 deaths per 100,000, depending on whether we use their three-day window estimates or their one-month window estimates. Since we estimated that a one-percentage-point increase in unemployment causes a 0.5 percent decline in the elderly mortality rate, or about 23 deaths per 100,000 given the 2006 elderly mortality rate of about 4,600 per 100,000, this decrease of 4 to 8 deaths per 100,000 represents about 17 to 35 percent of the 2007-2009 total estimated recession-induced mortality decline.

C.11 Instrumenting for PM2.5 exposure with Wind Direction

We estimate an alternative version of the pollution mediation analysis in Figure 10d, in which we instrument for the PM2.5 that a county is exposed to with the extent that they are downwind from commuting zones with substantial economic shocks from the Great Recession. Recall that commuting zones which experience a larger decline in unemployment from the Great Recession experience larger declines in PM 2.5 (see Figure 10b). The idea behind the instrument is that counties that are downwind of commuting zones that experience a large economic shock from the Great Recession may experience declines in pollution that are independent from the economic consequences of the Great Recession in the counties' own commuting zone. Prior work has used variation in wind direction as an instrument for both short-term variation in pollution exposure (Deryugina et al. 2019) and long-term exposure to pollution (Anderson 2020).[62]

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[61] Ebenstein et al. (2017) and Anderson (2020) are important exceptions.

[62] An alternative to using wind direction as an instrument would be to use an integrated assessment model such as the AP3 model (see Hollingsworth et al. (2022) for a recent paper taking this approach). The AP3 model captures emissions transportation over long distances but requires as inputs measures of emissions of individual pollutants. Since our analysis uses overall PM 2.5 pollution measures rather than emissions of individual pollutants, we use the wind instrument approach instead to predict downwind pollution changes. To the extent that our wind instrument approach does not capture all of

Construction of wind instrument We first confirm that a persistent, time-invariant wind direction exists for each county. To do so, we calculate annual measures of the prevailing wind in each county using 2003-2016 monthly data over a 0.25 degree by 0.25 degree grid of the entire United States; we obtained the raw prevailing wind data as grid-month averages from the European Centre for Medium-Range Weather Forecasts (ECMWF) Reanalysis v5 (ERA5) data set.[63] To aggregate up geographically to the county level, we map the center of each grid square to the Census Block Group (CBG); we weight each square by 2010 population when averaging to the county-month level. We then take a simple average of prevailing wind direction across months to obtain an annual, county-level measure of prevailing wind. Figure OA.35 confirms that there is a persistent wind direction. Specifically, it plots the county's wind direction (as defined by the angle θ) in year t, against the wind direction in $t-1$; the correlation is quite high. In what follows, we therefore use a single, time-invariant measure of the prevailing wind direction into each county, based on the average of the county-year measures during our 2003-2016 study period.

To construct the wind instrument, we follow Anderson (2020) and define a cone 45 degrees wide on both sides of the destination county's prevailing wind direction, extended outwards for a radius of 500 miles;[64] any neighboring counties whose centers fall within this cone are defined as upwind of the destination county.[65] We use the simple average of the CZ-level Great Recession shock in all upwind counties as instruments for the PM2.5 shock of the destination county. We exclude from the group of upwind counties any counties that share a CZ with the destination county and, due to data availability, we only consider Great Recession shocks for U.S. counties that fall within our defined cone and do not account for recession-induced changes in pollution in Canadian or Mexican regions that also fall within a cone.

Figure OA.36 illustrates examples of our instrument. The prevailing direction vector points towards the destination county and extends outward 500 miles from the destination county. The quarter-circle region defined as upwind is a cone that extends 45 degrees in either direction of the prevailing wind direction. Any counties whose centers fall within the defined upwind region are classified as upwind counties, and are highlighted in yellow.

First stage and placebo test We define the counties $\kappa \in K(c)$ to be the set of counties that are upwind of county c and are not in the same commuting zone as county c, with $N_{K(c)}$ denoting the number of counties in set $K(c)$ The first stage estimating equation is therefore:

$$PM2.5_{ct} = \sum_t \beta_t \left[\left(\frac{1}{N_{K(c)}} \sum_{\kappa \in K(c)} SHOCK_{cz(\kappa)} \right) \times 1(Year_t) \right] \\ + \sum_t \gamma_t [SHOCK_{cz(c)} \times 1(Year_t)] + \alpha_c + \delta_t + \varepsilon_{ct}; \quad (29)$$

---

the downwind effects that would be captured in the AP3 model, we will understate the overall role of pollution in explaining our mortality results.

[63] Data can be downloaded at: https://cds-beta.climate.copernicus.eu/datasets/reanalysis-era5-single-levels-monthly-means?tab=overview. In the raw data, wind direction is given as a u vector, the strength of wind along the east-west axis, and a v vector, the strength of wind along the north-south axis. After aggregating to the (time-invariant) county level, basic trigonometry allows us to convert the u and v vectors into a wind direction angle ranging from 0 to 360 degrees, where north is 0 degrees and south is 180 degrees.

[64] The 500 mile cutoff is ad hoc; we chose it to maximize the strength of the first stage.

[65] We calculate the coordinates of each county's center by averaging the coordinates of each Census Block Group within the county, weighting by 2010 population.

where $PM2.5_{ct}$ is the level of PM2.5 in a county-year for the destination county c, $SHOCK_{cz(c)}$ is the CZ-level Great Recession shock in county c, while $SHOCK_{cz(\,)}$ is the CZ-level Great Recession shock in some upwind county . ${}_t$ are the coefficients of the instrument, ${}_t$ are the coefficients on the destination county's own Great Recession shock, and ${}_c$ and ${}_t$ denote county and year fixed effects, respectively.

Since the wind direction data is not sufficiently granular to contain at least one observation in each county, we are able to define the wind instrument for 2,982 of the 3,107 counties (94.8 percent of the population-weighted counties) analyzed in Figure 10b). Figure OA.37a shows the first stage relationship between the instrument and PM 2.5 levels; specifically, it displays the coefficients ${}_t$ from equation (29).

To verify that wind is the actual channel for the instrument's power, and that the Great Recession shocks of other, non-upwind, neighboring counties would not be equally effective instruments for the destination county's PM2.5 shock, we run a placebo test in equation (30) where we add a placebo instrument in addition to the real instrument. The placebo instrument has the exact same definition as the real instrument, but with the prevailing wind direction rotated 90 degrees clockwise. A new cone is drawn out from the placebo prevailing wind direction, also 45 degrees on both sides and 500 mile radius, and $K'(c)$ is the set of placebo upwind counties comprised of individual counties $'$. Everything else is defined the same as in equation (29).

$$
\begin{aligned}
PM2.5_{ct} = {}_t &\left[\left(\frac{1}{N_{K(c)}}\sum_{\in K(c)} SHOCK_{cz(\,)}\right) \quad 1(Year_t)\right] \\
&+ {}_t\left[\left(\frac{1}{N_{K'(c)}}\sum_{'\in K'(c)} SHOCK_{cz(\,')}\right) \quad 1(Year_t)\right] \\
&+ {}_t[SHOCK_{cz(c)} \quad 1(Year_t)] + {}_c + {}_t + {}_{ct} \qquad (30)
\end{aligned}
$$

Note that while rotating the prevailing wind direction 180 degrees would be an intuitive choice of placebo, wind actually tends to blow along an axis, sometimes reversing direction, so a 90 degree rotation is a truer placebo than a 180 degree rotation (NOAA 2017).

Figures OA.37b and OA.37c show the results of the placebo test: the estimates of ${}_t$ and ${}_t$ from equation (30) respectively. Estimates of ${}_t$ remain very similar in the placebo test, while estimates of ${}_t$, the placebo instrument, are insignificant. This supports the contention that our wind instrument captures PM2.5 declines, and that these declines are driven by changes in economic activity in specifically upwind counties.

IV mediation analysis For the IV version of the mediation analysis in equation (8), we estimate a more parsimonious version of equation (8) in which we replace the interaction of each shock with year fixed effects with the interaction of each shock with either a single indicator $POST$ variable for years 2007-2016, or—in an alternative specification—with two indicator variables for the years 2007-2009 ($POST1$) and the years 2010-2016 ($POST2$). We do this to increase the power of the first stage.

Specifically, we estimate:

$$y_{ct} = [SHOCK_{cz(c)} \quad POST] + {}_c + {}_t + {}_{ct} \qquad (31)$$

and a mediated version:

$$y_{ct} = \quad [SHOCK_{cz(c)} \quad POST] + \quad [PM2{:}5\_SHOCK_c \quad POST] + \quad {}_c + \quad {}_t + \quad {}_{ct} \qquad (32)$$

or analogs in which the single indicator POST is replaced by two separate indicators for POST1 and POST2.

Table OA.19 shows the results. Columns (1) and (2) of Table OA.19 show that the results from estimating equations (31) and (32) look very similar to the more flexible specifications seen, respectively in Panels (a) and (d) of Figure 10.[66] As we saw previously, the OLS mediation analysis suggests that controlling for pollution reduces the recession-induced mortality declines by about 20 percent in 2007-2009, from an estimated impact of a 1 percentage point increase in unemployment on mortality of -0.530 percent (standard error = 0.155) in column 1 to an estimated impact of -0.425 percent (standard error = 0.161) in column 2 once the PM2.5 shock is controlled for. The role of recession-induced declines in PM2.5 in mediating the impact of the Great Recession on mortality becomes, if anything, more pronounced when we instrument for the pollution shock with upwind economic shocks in column (3). The IV analysis in column 3 indicates that controlling for the PM 2.5 shock essentially obliterates the impact of unemployment on mortality in 2007-2009, with the point estimate now a statistically insignificant -0.022 (standard error = 0.31). A similar pattern appears for the 2010-2016 estimates or the pooled 2007-2016 estimates. The substantially larger mediating impact of pollution in the IV analysis is consistent with the extensive discussions in the literature of the potential for substantial measurement error in PM2.5 (Currie et al. 2023; Fowlie et al. 2019; Grainger and Schreiber 2019).

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[66] Note that the estimates in column (1) and (2) differ slightly from the average of the pooled coefficients in Figure 10a and 10d, respectively, because the sample here is the subsample of 2,982 counties (from the original 3,107) for which we have the data necessary to construct the wind instrument.

D Compilation of Event Studies

In Section 3.2, we summarize the results from some mortality event studies in terms of the average estimated effects across years, particularly as they pertain to heterogeneity in mortality declines across groups and causes of death. We also refer to additional mortality analyses not presented in the main text. Here we present the underlying event studies behind these results:

- **Cause of death:** Figures 5 and OA.8 show results for the 11 most common causes of death, plus a residual category for all other deaths.
- **Deaths of despair:** Figure OA.11a shows results for deaths of despair, while Figures 5d, 5e, and OA.11b show the results for each of the three components of deaths of despair: suicide, liver disease, and drug poisonings (accidental or unknown).
- **By age:** Figures OA.39 and OA.40 show results for different age groups.
- **By education:** Figure OA.41 shows results by education. It shows results separately for the half of the population with a high school degree or less and the half of the population with more than a high school degree. It also disaggregates the results further to those with less than high school, exactly high school, some college but no four-year degree, and a four-year college degree or more.
- **By education, by age:** Figure OA.42 confirms that the finding that the Great Recession is confined to those with a high school education or less persists even when we look within age groups.
- **By Medicaid status:** Figure OA.43 shows results using the Medicare cross-sectional data for the elderly by whether or not individuals were enrolled in Medicaid during the previous year.
- **By race:** Figure OA.44 disaggregates results by Non-Hispanic White, Non-Hispanic Black, Hispanic, and Other populations.
- **By gender:** Figure OA.45 disaggregates results by gender.
- **Health status of marginal life saved:** Figure OA.46 shares average predicted counterfactual remaining life expectancy among decedents under a range of controls, and Tables OA.16 and OA.17 estimate the impact of $SHOCK_c$ on life-years lost under these counterfactual life expectancies.
- **Morbidity** Figures OA.47 and OA.48 show impacts on measures of morbidity from the BRFSS.
- **Motor vehicle mortality by age** Figure OA.51 shows results for motor vehicle mortality by age groups, as well as the share of the total mortality decline for various age groups that can be accounted for by declines in motor vehicle mortality. For the non-elderly, we find that a much larger share of the recession-induced mortality declines are accounted for by motor vehicle accidents. For example, while they account for only about seven percent of the overall recession-induced mortality decline, they account for almost one-quarter of the decline for 25-64 year olds and roughly half of the decline for those aged 15-44. By contrast, we find no evidence of recession-induced mortality declines due to motor vehicle accidents for the elderly, consistent with recessions not affecting their driving patterns.

In Section 3.3, we summarize the results from various event studies in terms of the average estimated effect of the Great Recession on mortality across years, particularly with regard to sensitivity analyses. Here we present the underlying event studies behind these results:

- Sensitivity to fixing population location in 2003: Figure OA.15 shows results restricting to non-movers in the Medicare panel data. Figures OA.16 and OA.18 confirm that our overall mortality estimates look similar among the Medicare sample to our main specification which uses CDC data to estimate mortality across all age groups, and that results look similar when assigning individuals to CZs based on their 2003 CZ of residence rather than their yearly CZ of residence.
- Sensitivity to geography: Figure OA.25 shows results changing the level of geography to counties and states rather than CZs, as well as dropping certain CZs from the sample (including especially populous CZs and CZs with significant fracking activity).
- Sensitivity to functional form: Figure OA.26 shows results under alternative functional forms.
- Linearity: Figure OA.27 plots the (population-weighted) average of $SHOCK_c$ in each ventile of the CZ-shock distribution against the within-ventile average change in log mortality, while Figure OA.29 adopts a modified version of equation (17), with $Recovery_{q(c)}$ substituted for an indicator for whether a CZ experienced an above or below (population-weighted) median unemployment shock, and displays separate event studies for above- and below-median CZs.

In Section 4 we summarize results from various event studies, particularly regarding potential mechanisms for mortality declines. Here we present the underlying event studies behind these results.

- Self-reported health behaviors: . Figures OA.49, and OA.50 show results for self-reported health behaviors in the BRFSS.
- Utilization of health services: Figure OA.20 examines utilization of health services among the Medicare population.
- Nursing home care: Figure OA.52 examines the effect of $SHOCK_c$ on mortality separately by individuals' nursing home utilization, while Figures OA.53 and OA.54 examine nursing home characteristics.
- Informal care provision: Figure OA.30 examines the effect of $SHOCK_c$ on measures of informal care provision recorded in the HRS.
- Pollution: Figures OA.32, OA.33, and OA.34 examine the mediation effects of other pollutants on our main results.

E Additional Details on Welfare Analyses

E.1 Simplified Welfare Model

We consider a simplified version of the model in Section 5 in which the aggregate state 2 f L; H g is drawn once and for all at $t = 0$, and there is no retirement. We consider two scenarios. In the first, mortality is exogenous to the aggregate economic state and individuals live for T periods. Under these assumptions, the agent's lifetime utility in the two states of the world is given by:

- Normal state. Expected lifetime utility if nature draws the normal state:

$$E[U(c;m)]^{normal} = p^H \quad T \quad u((1 \quad d^H)c) + (1 \quad p^H) \quad T \quad u(c) \tag{33}$$

- Recession. Expected lifetime utility if nature draws the recession state:

$$E[U(c;m)]^{recession} = p^L \quad T \quad u((1 \quad d^L)c) + (1 \quad p^L) \quad T \quad u(c) \tag{34}$$

We define the welfare consequence of a recession with exogenous mortality as , and it is thus given by:

$$E[U((1 + \ ) \quad c;m)]^{recession} = E[U(c;m)]^{normal} \tag{35}$$

Given the constant elasticity of marginal utility with respect to consumption in the per-period utility function, we can solve for the following closed-form expression for :

$$= \left(\frac{p^H(1 \quad d^H)^{(1 \quad)} + (1 \quad p^H)}{p^L(1 \quad d^L)^{(1 \quad)} + (1 \quad p^L)} \right)^{1=(1 \quad)} \quad 1 \tag{36}$$

This expression is increasing in p^L (the probability of job displacement in a recession) and d^L (the reduction in consumption in a recession), as expected.[67] The welfare cost of the recession is independent of b, the parameter which governs the VSLY, and of life expectancy T. Since life expectancy is assumed to be independent of the aggregate state, neither it nor the VSLY affects the agent's willingness to pay to avoid the recession state.[68]

In the second scenario, we allow for mortality to be endogenous to the aggregate state. In the normal state, life expectancy is T, while in the recession state, life expectancy is $T(1 + dT)$. Now we obtain the following expressions for expected lifetime utility in the two states:

$$E[U]^{normal} = p^H \quad T \quad u((1 \quad d^H)c) + (1 \quad p^H) \quad T \quad u(c) \tag{37}$$

$$\begin{aligned} E[U]^{recession} = {} & p^L \quad T(1 + dT) \quad u((1 \quad d^L)c) \\ & +(1 \quad p^L) \quad T(1 + dT)u(c) \end{aligned} \tag{38}$$

Using the above expressions, we can solve for the welfare cost of a recession in the case with endogenous

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[67] The expression is also increasing in $p^L \quad p^H$ and $d^L \quad d^H$ for all $0 < d^L < 1$ and $0 < d^H < 1$.

[68] We can also simplify the basic model even further by assuming $p^H = 0$ and $d^H = 0$. In this case, we have $= (p^L \quad (1 \quad d^L)^{(1 \quad)} + 1 \quad p^L)^{(1=(\quad 1))} \quad 1$. From this expression, we see that for $0 < p^L < 1$ and > 1, we have that as d^L goes towards 1 (holding p^L constant) we have going to 1 , implying that the agent is not willing to accept any amount of consumption to accept the recession state as the earnings consequences of job displacement grow large, as in Krebs (2007).

mortality (^{dT}):

$$ {}^{dT} = \left(\frac{dT \quad b = \bar{u}(c) + p^H (1 \quad d^H)^{(1 \quad)} + (1 \quad p^H)}{(1 + dT)(p^L (1 \quad d^L)^{(1 \quad)} + (1 \quad p^L))} \right)^{1=(1 \quad)} \quad 1 \qquad (39) $$

where $\bar{u}(c) = u(c) \quad b = \frac{c^1}{1}$, which transforms the per-period utility function into a standard CRRA utility function. Note that the expression for ^{dT} in equation (39) is valid for any value of dT and it simplifies to the expression for in equation (36) if dT = 0.[69]

We can build further intuition by setting $p^H = 0$ and then taking a first-order approximation around the left-hand side of equation (39) at $^{dT} = 0$, which leads to the following expression:

$$ 1 + (1 \quad) \quad {}^{dT} \qquad \frac{dT \quad b + \bar{u}(c)}{(1 + dT) \quad (p^L \quad (1 \quad d^L)^{(1 \quad)} \bar{u}(c) + (1 \quad p^L) \bar{u}(c))} \qquad (40) $$

$$ {}^{dT} \qquad dT \quad \frac{VSLY}{c} + \frac{1}{\quad 1} \qquad (41) $$

where is the welfare cost of a recession with exogenous mortality, and the second term is the adjustment for the percent change in life expectancy dT.[70]

E.2 Analytical Results for the Full Model

In the main text, we report simulation results of our model extending Krebs (2007) to allow for recessions to have mortality effects. The main reason we focus on the simulation results (rather than analytical results) is to be able to use realistic age-specific mortality rates which allow the welfare effects of recessions with endogenous mortality to vary by age.

In this section, we derive analytical results for the full model to show that the intuition from the simplified welfare model above carries through to our full model. To derive an analytical solution to our model, we make the following simplifying assumptions: we set = 1, and we assume that the mortality rate 1 q^S depends on the aggregate state but not age. We also continue to assume hand-to-mouth consumption so that consumption equals income each period.

In this setup, our goal is to solve for , which is implicitly defined as follows:

$$ \underbrace{E_0 \left[\sum_{t=0}^{1} (q^S)^t u((1 + \;) \quad y(t)) \right]}_{\text{Expected Lifetime Utility with Stochastic Aggregate State}} = \underbrace{E_0^{S=H} \left[\sum_{t=0}^{1} (q^H)^t u(y(t)) \right]}_{\text{Expected Lifetime Utility without Recessions}} ; $$

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[69] To see this, note that the dT $b\bar{u}(c)$ term in the numerator and the (1 + dT) term in the denominator in the expression for ^{dT} are the only differences with the expression for . This also means that if dT > 0, then ^{dT} < , meaning that a recession that is "good for your health" is less costly to the agent than an otherwise similar recession that has no impact on mortality risk (dT = 0). While the agent continues to dislike possible reductions in consumption during a recession, the agent values the increase in life expectancy associated with a recession, thus depressing their willingness to pay to avoid recessions.

[70] The derivation uses the fact that the per-period utility function $u(c) = \bar{u}(c) + b$ implies $VSLY = bc \quad c=(\quad 1)$, which can be re-written as follows:

$$ VSLY = bc \quad c=(\quad 1) $$
$$ VSLY=c + 1=(\quad 1) = b \quad c^{(\quad 1)} $$
$$ VSLY=c + 1=(\quad 1) = b=\bar{u}(c)=(1 \quad) $$

This expression shows that b needs to be "sufficiently large" to ensure that VSLY=c is positive, which is necessary for individuals to prefer lower mortality rates.

As in the simplified model, we define $u(c) \quad \bar{u}(c) + b$, and we use this definition to re-arrange as follows:

$$E_0\left[\sum_{t=0}^{\infty}(q^S)^t(\bar{u}((1+\ )\ y(t))+b)\right] = E_0^{S=H}\left[\sum_{t=0}^{\infty}(q^H)^t(\bar{u}(y(t))+b)\right]$$

$$E_0\left[\sum_{t=0}^{\infty}(q^S)^t(\bar{u}((1+\ )\ y(t)))\right]+E_0\left[\sum_{t=0}^{\infty}(q^S)^t(b)\right] = E_0^{S=H}\left[\sum_{t=0}^{\infty}(q^H)^t(\bar{u}(y(t)))\right]+E_0^{S=H}\left[\sum_{t=0}^{\infty}(q^H)^t(b)\right]$$

We next define $T \quad E_0 \sum_{t=0}^{\infty}(q^S)^t$, $T^{S=H} \quad E_0 \sum_{t=0}^{\infty}(q^H)^t$, and $dT = T \quad T^{S=H}$, so that $dT > 0$ implies that eliminating recessions reduces mortality. We then have the following:

$$E_0\left[\sum_{t=0}^{\infty}(q^S)^t(\bar{u}((1+\ )\ y(t)))\right] + dT \quad b = E_0^{S=H}\left[\sum_{t=0}^{\infty}(q^H)^t(\bar{u}(y(t)))\right]$$

Following the derivations in Krebs (2007), we have the following expressions:

$$E_0\left[\sum_{t=0}^{\infty}(q^S)^t(\bar{u}((1+\ )\ y(t)))\right] = \frac{\bar{u}((1+\ )\ y_0)}{1 \quad \bar{q}E_0[(1+g)^{(1\quad)}(1+\ )^{(1\quad)}(1+\ )^{(1\quad)}]}$$

$$E_0^{S=H}\left[\sum_{t=0}^{\infty}(q^H)^t(\bar{u}(y(t)))\right] = \frac{\bar{u}(y_0)}{1 \quad q^H E_0[(1+g)^{(1\quad)}(1+\ )^{(1\quad)}(1+\ )^{(1\quad)}]}$$

where $\bar{q} = {}^{L}\ q^L + {}^{H}\ q^H$. Substituting in these expressions gives the following:

$$\frac{(1+\ )^{(1\quad)}}{1 \quad \bar{q}E_0[(1+g)^{(1\quad)}(1+\ )^{(1\quad)}(1+\ )^{(1\quad)}]} + dT \quad b/\bar{u}(y_0) = \frac{1}{1 \quad q^H E_0^{S=H}[(1+g)^{(1\quad)}(1+\ )^{(1\quad)}(1+\ )^{(1\quad)}]}$$

Note that if $dT = 0$ and $q^L = q^H = q$, then we have the following:

$$(1+\ )^{(1\quad)} = \frac{1 \quad qE_0[(1+g)^{(1\quad)}(1+\ )^{(1\quad)}(1+\ )^{(1\quad)}]}{1 \quad qE_0^{S=H}[(1+g)^{(1\quad)}(1+\ )^{(1\quad)}(1+\ )^{(1\quad)}]}$$

$$= \left(\frac{1 \quad qE_0[(1+g)^{(1\quad)}(1+\ )^{(1\quad)}(1+\ )^{(1\quad)}]}{1 \quad qE_0^{S=H}[(1+g)^{(1\quad)}(1+\ )^{(1\quad)}(1+\ )^{(1\quad)}]}\right)^{\frac{1}{1\quad}} \quad 1$$

which is the same expression as in Krebs (2007), setting $= q$.

To simplify the expression when $dT > 0$, we use the fact that $(VSLY/c) \quad (1 \quad\ ) \quad 1 = b/\bar{u}(c)$. We then go back to the above expression for and substitute. We also define $\quad (1 \quad \bar{q}E_0[(1+g)^{(1\quad)}(1+\ )^{(1\quad)}(1+\ )^{(1\quad)}])/(1 \quad q^H)$. We then have the following expression for ^{dT} (which we label as ^{dT} following the simplified model in the previous section and to distinguish from the defined in Krebs (2007)):

$$\boxed{(1+\ {}^{dT})^{(1\quad)} = \frac{1 \quad \bar{q}E_0[(1+g)^{(1\quad)}(1+\ )^{(1\quad)}(1+\ )^{(1\quad)}]}{1 \quad q^H E_0^{S=H}[(1+g)^{(1\quad)}(1+\ )^{(1\quad)}(1+\ )^{(1\quad)}]} \quad \frac{dT}{T^{S=H}} \quad ((VSLY/y_0) \quad (1 \quad\ ) \quad 1)}$$

As in the previous section, if we take a first-order approximation around ${}^{dT} = 0$, then we get $1 + (1 \quad\ ) \quad {}^{dT}$ on the left-hand side, which gives the following approximation formula:

$$\boxed{{}^{dT} \quad \frac{dT}{T^{S=H}} \quad \frac{VSLY}{y_0} + \frac{1}{\quad 1}}$$

The formula above is similar to the approximation formula in E.1 except for the additional term which is a necessary adjustment since the full model has a dynamic income process. The two boxed formulas show analytically that the welfare cost of recessions in the full model is approximately \separable" in the welfare cost coming from endogenous mortality.

E.3 Calibrations for Welfare Analysis of Adapted Krebs (2007) Model

- Mortality in \normal" times For mortality in \normal" times, we use the 2007 SSA mortality tables to calculate age-specific mortality rates for the $m^H(t)$ vector. The SSA reports mortality tables separately for men and women, available at https://www.ssa.gov/oact/HistEst/PerLifeTables/2022/PerLifeTables2022.html . We calculate the unisex mortality rate as the population-weighted average mortality rate using data from 2007. Specifically, for age a, male mortality rate $m^m(a)$, female mortality rate $m^f(a)$, and a male population share $s^m(a)$ we calculate $m(a) = s^m(a) \quad m^m(a) + (1 \quad s^m(a)) \quad m^f(a)$.

- Mortality decline from a typical recession To calibrate the mortality decline from a typical recession, we estimate that a typical recession produces a 3.1-percentage-point increase in the unemployment rate. We arrive at this estimate by using monthly data from the Federal Reserve (FRED { https://fred.stlouisfed.org/series/UNRATE) on the unemployment rate and the NBER's recession dating (https://fredhelp.stlouisfed.org/fred/data/understanding-the-data/recession-bars/). From this, we calculate the increase in unemployment in each post-World War II recession|excluding the Great Recession and the COVID Recession|as the difference between the minimum and maximum unemployment rate in the period starting 12 months before the official beginning of the recession or the end of the previous recession (whichever is later) and ending 12 months after its official end or when the next recession starts (whichever is sooner). We then combine this unemployment rate increase with our estimates in Section 3 that a one-percentage-point increase in unemployment causes a 0.5 percent decline in the mortality rate to calibrate the mortality decline from a typical recession of $dm = \quad 0{:}015$.

- VSLY We report results for a VSLY of \$100k, \$250k, and \$400k.[71] The high end of the range is based on several different sources described in Kniesner and Viscusi (2019). They report that a \$369,000 VSLY was used by the US Department of Health and Human Services and the Food and Drug Administration in 2016. They also note that much of the literature estimates a value of a statistical life (VSL), and explains that the VSLY can be calculated from an estimate of the VSL using the identity $VSLY = \quad r \quad VSL=(1 \quad (1+r)^{\ L})$, where L is life expectancy and r is the interest rate. They report that many government agencies use a VSL of about \$10 million; this is also the focal VSL estimate used in Viscusi (2018). Using what they say is the standard assumption in this literature of $r = 0{:}03$ and assuming that $L = 50$, we recover a VSLY of \$388,000. The low end of the range follows the assumed \$100,000 VSLY made by e.g. Cutler (2005) and Cutler et al. (2022). In a similar vein, Hall and Jones (2007) use as a baseline a VSL estimate of \$3 million, although they note it is at the low end of the range of estimates and they report sensitivity to higher values. Again assuming $r = 0{:}03$ and $L = 50$, this would imply a VSLY of \$117,000. Finally, we chose a VSLY of \$250,000 as the mid-point of the range of estimates.

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[71] We calculate the value of b given an assumed value of for each $VLSY$ value, and we report these values in Table OA.20.

E.4 Welfare Analysis by Education

Consider two educational groups where $e = \ell$ refers to individuals with a high school (HS) diploma or less and $e = h$ to individuals with more than a HS diploma. We have heterogeneity by education through three main channels in our welfare analysis: mortality rates $m_e(t)$, mortality effects dm_e, and income effects (which are jointly defined by the probability of being displaced d_e^{ω} and the displacement shock p_e^{ω}, where $\omega \in \{L, H\}$. Below we discuss how we estimate or calibrate each of these parameters.

- **Mortality rates by education** We allow mortality rates to vary by education, calibrating them based on Meara et al. (2008) so that the remaining life expectancy at age 25 is 14.1% higher for those with more than a high school degree.[72] Specifically, we use our unisex mortality rates, given by a population-weighted average of SSA gender-specific rates, and adjust it for education using the following algorithm: First, guess some $x \in (0, 1)$. Second, for each age a define $m_a^{\ell} = (1 + x)m_a$ and $m_a^{h} = (1 - x)m_a$, where m_a is the mortality rate at that age from the unisex table. Third, using these mortality rates and the methodology described in Appendix C.4.1, compute the remaining life expectancy at each age. Fourth, choose x such that the remaining life expectancy at age 25 is 14.1% higher for those with less than a high school degree.

- **Mortality effects by education** In Section 3, we report that a 1 percentage point increase in the local unemployment rate is associated with a statistically significant 0.80 percent (standard error = 0.26) decline in the 2007-2009 mortality rate for those with a high school education or less, compared to a statistically significant 0.014 percent (standard error = 0.54) increase in the mortality rate for those with more than a high school education. We use these estimates to calibrate the welfare effects of recessions by education by imposing that the ratios of the mortality effects in a typical recession relative to our estimated mortality effects during the Great Recession are the same by education.

  Specifically, we set up a system of two equations with two unknowns: the typical recession mortality effects for each education group e: $dm_{e=\ell}$ and $dm_{e=h}$. Define π_e as the population share for educational group.

$$\begin{cases} dm = \pi_{e=\ell}\, dm_{e=\ell} + (1 - \pi_{e=\ell})\, dm_{e=h} \\ \dfrac{dm_{e=\ell}}{dm^{GR}_{e=\ell}} = \dfrac{dm_{e=h}}{dm^{GR}_{e=h}} \end{cases}$$

  The first equation states that the overall mortality effect of a typical recession is given by the population-share-weighted average of the mortality effects by education. The second equation states that the ratio of the mortality effects (of a typical recession relative to the Great Recession) is the same by education group. We estimate in the CPS that 68 percent of the population has a high school degree or less, and 32 percent has at least some college education, so we set $\pi_{e=\ell} = 0.68$ and $\pi_{e=h} = 0.32$.[73] As noted in Section 5, we calibrate the mortality effect of a typical recession to be $dm = -0.015$ based on our baseline empirical estimates that a 1 percentage point increase in unemployment is associated with a 0.5 percent decline in the mortality rate, combined with the average 3.1 percentage point increase in the unemployment rate in a typical recession. Our Great

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[72] This number comes from Exhibit 1 of Meara et al. (2008), which shows that, in 2000, remaining life expectancy at 25 was 56.6 years for those with more than HS and 49.6 years for those with a HS diploma or less, a difference of 14.1%.

[73] Note that our analysis in this section pools CPS data from 1976-2007, which means that we re-estimate the population shares by education group for this longer time period. The population shares indicate lower average levels of education than for the population in the Great Recession sample because of secular trends in educational attainment in the US during the last several decades.

Recession estimates are $dm^{GR}_{e=\ell} = 0.0367$ and $dm^{GR}_{e=h} = 0.0006$ Using these estimates, we solve the two-equation system and find $dm_{e=\ell} = 0.023$ and $dm_{e=h} = 0.0004$.

- Job displacement effects by education To calibrate the welfare effects of recessions by education, we also need to calibrate education-group-specific job displacement parameters and education-group-specific change in income conditional on job displacement parameters. Existing evidence from Farber (2017) using the CPS Displaced Worker Supplement finds larger percentage reductions in income for workers with lower levels of education. Workers with lower levels of education also have higher unemployment rates in both recessions and normal times, partly due to higher job separation rates. As a result, we calibrate separate job displacement and income change (conditional on job displacement) parameters for the two education groups (i.e., separate p^S_e and d^S_e parameters for each education group in each aggregate state).

  To do this, we first calibrate the education-specific job displacement probabilities p^S_e. We estimate the education-group-specific average unemployment rate by state S ($\bar{\ }^S_e$) using data from the CPS from 1976 to 2007; we use the recession years within this time period to estimate $\bar{\ }^L_e$ and the non-recession years to estimate $\bar{\ }^H_e$ (based on the recession years defined by the NBER). This gives us estimates $\bar{\ }^L_{e=\ell} = 0.095$, $\bar{\ }^H_{e=\ell} = 0.083$, $\bar{\ }^L_{e=h} = 0.041$, and $\bar{\ }^H_{e=h} = 0.038$.

  Next, we assume that the ratios of unemployment rates by education (in state S) are the same as the ratio of the p^S_e job displacement probabilities needed in the model calibration. We also assume that the overall job displacement probability p^S is equal to the education-group-weighted average of the education-group-specific job displacement probabilities. This leads to the following two-equation system, which is defined for each aggregate state:

$$\begin{cases} p^S = \ _{e=\ell}\, p^S_{e=\ell} + (1 \quad _{e=\ell})\, p^S_{e=h} \\ \dfrac{\bar{\ }^S_{e=\ell}}{\bar{\ }^S_{e=h}} = \dfrac{p^S_{e=\ell}}{p^S_{e=h}} \end{cases}$$

  Following Krebs (2007), we have $p^L = 0.05$ and $p^H = 0.03$, which when combined with the estimates above allows us to solve for the four unknown parameters $p^L_{e=\ell} = 0.061$, $p^L_{e=h} = 0.027$, $p^H_{e=\ell} = 0.037$, and $p^H_{e=h} = 0.016$.

  The last set of parameters are the changes in income conditional on displacement, which we also want to allow to vary by education group and aggregate state (d^S_e). To calibrate these values, we use estimates of the earnings losses for full-time displaced workers by education from Table 2 in Farber (2017). We average the education categories (using population weights) to match our two broad education categories, and this gives us the average decline in income from job displacement by education (averaged across labor market states). To get state-specific estimates by education, we solve the following two-equation system for each aggregate state:

$$\begin{cases} d^S = \ _{e=\ell}\, d^S_{e=\ell} + (1 \quad _{e=\ell})\, d^S_{e=h} \\ \dfrac{d^S_{e=h}}{d_{e=h}} = \dfrac{d^S_{e=\ell}}{d_{e=\ell}} \end{cases} \tag{42}$$

  As with the mortality effects and the job displacement probabilities, the two equations assume a common ratio by education group and impose that the overall average income decline by state is equal to the education-group-weighted average income decline. Given the $d^L = 0.21$ and $d^H = 0.09$

values from Krebs (2007) and the Farber (2017) estimates, we calculate $d^L_{e=\ell} = 0.233$, $d^L_{e=h} = 0.161$, $d^H_{e=\ell} = 0.100$, and $d^H_{e=h} = 0.069$.

This gives us all of the parameters needed to calibrate the welfare model separately by education group.

E.5 Incorporating Mortality Effects of Job Displacement

Sullivan and Von Wachter (2009) find that job displacements substantially increase mortality. Our finding that the increase in unemployment from the Great Recession reduces mortality is a net result, which can mask the increase in mortality for the subset of people who lose their job. In our baseline welfare model calibrations, we do not allow for job displacements to affect mortality. This section describes how we adapt our model to accommodate this result by allowing for separate effects of recessions on mortality for those who do or do not experience job displacement, in order to incorporate the results in Sullivan and Von Wachter (2009).

Since Sullivan and Von Wachter (2009) find that mortality increases significantly for displaced workers with high tenure, we add a new state variable to our model so that agents have tenure ($\tau \in \{\ell, h\}$) drawn independently (across our simulated cross section of individuals each period), with the per-period probability of high tenure given by $p^{\tau=h}$. We define D to be an indicator that equals one at or after the first time a worker is displaced with high tenure. Mortality rates are affected by job displacement (for high-tenure workers only) and by the aggregate state (as in the baseline model) as follows: $m^L = (1 + dm^{L'}) \times (1 + dm^{D=1}) \times m^H$, where $dm^{L'} < 0$ is the overall "gross" effect of recessions and $dm^{D=1} > 0$ is the persistent effect of high-tenure job displacement on mortality.

To calculate $dm^{L'}$ and $dm^{D=1}$, we impose the condition that the ratio of expected mortality in the recession state and the normal state in this new model is the same as in our baseline model calibration. In other words, given a calibrated value for the displacement effect $dm^{D=1} > 0$, we set the "gross" mortality effect of recessions $dm^{L'} < 0$ so that the net impact of recessions on mortality remains the same as in the baseline model. More specifically, Sullivan and Von Wachter (2009) estimate that high-tenure displaced workers between age 35 and 50 lose around 1.5 years of life expectancy. Using the framework described above and the survival function in our main calibration, this is equivalent to a high-tenure displacement effect on mortality of $dm^{D=1} = 0.21$. Defining high-tenure as at least 6 years of tenure and using data from the CPS Displaced Workers Supplement, we find that the probability of being at or after the first high-tenure displacement probability in any period is given by 0.25, which allows us to calculate a gross recession effect on mortality of $dm^{L'} = -0.035$. This is 2.3 times larger than the dm^L used in our baseline calibration, which indicates how the large estimated mortality effects in Sullivan and Von Wachter (2009) indirectly affect the welfare cost of recessions for the "never displaced" workers according to our model (i.e., recessions benefit them 2.3 times as much as the average worker in our baseline calibration).

To see how this affects our welfare analysis, we calibrate this extended model allowing for mortality effects of job displacement, and we break out the workers who are "ever displaced" and "never displaced." Figure OA.38a reports the mortality cost of a job displacement as a percentage of average annual consumption. This welfare cost is defined as the willingness to accept an economy where job displacements affect mortality (i.e., $dm^{D=1} = 0.21$) relative to an economy where job displacements have no effect on mortality (i.e., $dm^{D=1} = 0$), holding constant the income and consumption consequences of job displacement (following Krebs (2007)). The large welfare effects therefore come entirely from the large effects of job displacement on mortality risk.

The second panel (Figure OA.38b) shows the welfare cost of recessions for the \ever displaced" and \never displaced" workers under exogenous and endogenous mortality. Given the Sullivan and Von Wachter (2009) age restrictions, we assume that only workers between ages 35 and 50 can experience the large mortality effects of a high-tenure job displacement. The results show that the welfare cost of recessions is about twice as large for the ever displaced workers compared to never displaced workers, and the gap between the welfare costs with endogenous and exogenous mortality is larger for the never displaced workers compared to our baseline calibration; this follows mechanically from the fact that $dm^{L'}$ is larger in magnitude than the dm^{L} used in our baseline calibration.

One implication of these results is that any recession that triggers an unusually large number of job displacements of high-tenure workers is likely to have smaller reductions (or even increases) in mortality in the aggregate as compared to our Great Recession estimates. Put another way, the mortality increases from job displacements in Sullivan and Von Wachter (2009) are substantially larger in magnitude than the overall mortality decreases that we estimate across the entire population. Interestingly, there is existing evidence in macroeconomics that recessions are primarily driven by reductions in the job finding rate rather than by increases in the job separation rate (Shimer 2012). This would make it much easier to simultaneously explain our results alongside the results in Sullivan and Von Wachter (2009). The Krebs (2007) model we use assumes that job displacement increases during recessions (and in our extension we assume that this increase in displacement probability is the same (proportionally) for high and low tenure workers), but if in fact recessions are not associated with an increase in job displacement, the two findings may not in fact be related.

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F Appendix Figures

Figure OA.1: 2006 Commuting Zone Population

Notes: This figure displays a histogram of 2006 CZ populations, in bins of 250,000. For visualization purposes, CZs with populations larger than three million are binned to three million. Descriptive statistics in the upper right-hand corner are reported for the full distribution. N=741 CZs.