Health and the Great Recession

How did mortality rate change as a result of the financial crisis?

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Abstract

Although the Great Recession originated in the financial sector, it ended up having broad-based implications that bled into nearly every sector of the "real economy." These impacts ended up affecting nearly all economic actors in some way or another. This paper examines the impact of the crisis on death rate and its leading causes, using a structural model adapted from other work. It also synthesizes previous literature, which suggests mixed findings on the relationship between economic indicators and health outcomes, both during crises and during less severe fluctuations. Our findings show that the Great Recession did not increase death rates on the whole, in line with other research studying mortality data in the 21st century. There was, however, variance in certain causes, as suicides were shown to increase after the crisis ended. We also conclude that results are highly sensitive to the specific start and end date definitions of the financial crisis.

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Introduction

When one thinks about the Great Recession and the conversations that surround it, especially in a finance or policy context, one's thoughts are immediately drawn to topics we discussed in class. People generally think of Lehman Brothers, interest rates, over-leveraged banks, mortgages, or housing. Few consider the non-financial impacts realized by individuals. While effects on savings, property, assets, income, or jobs are often studied, broader effects, such as happiness and health status remain largely unexplored. We seek to explore impacts to the "real economy" via health outcomes, specifically mortality. Throughout the paper, we seek to explore this different side of the financial crisis by examining the academic literature surrounding health impacts of the financial crisis. Additionally, we produce our own empirical analysis of the impacts of the financial crisis on mortality rates by cause. We hope to contribute to the understanding of how this crisis impacted individuals and see how consistent the health impacts are with other crises and macroeconomic downturns.

Literature Review

This literature review will seek to synthesize the existing academic literature to provide a foundation for interpreting our quantitative analysis. This literature review seeks primarily to understand what is already known about the health effects of the Great Recession, and more broadly, the relationship between health and economics, and crises more specifically, so as to contextualize our understanding of the recession's effects. The literature review proceeds as follows. Section I looks at the general research into the association between health and the business cycle. Section II examines whether crises differ from general fluctuations in terms of health effects, and

Section III examines Great Recession specific research on mortality, physical health, mental health, and individual behavior.

Section One: Health and the Business Cycle

There is a long history of research on the relationship between wealth and health. In developing countries, and throughout much of history, the positive relationship between economic growth and health is undisputed in the academic literature (Pritchett & Summers, 1996). However, the relationship between health and economic conditions within industrialized nations is subject to much debate.

Prevailing thought throughout much of the 20th Century was that industrialized nations were much like their developing counterparts — health improves as the economy improves (Ruhm, 2015). Probably the foremost scholar of this school of thought was then-John Hopkins professor Harvey Brenner, who used a time series analysis to study the relationship between mortality and unemployment (often used as the standard metrics for measuring the association between health outcomes and the economy). Brenner found a strong procyclical relation between unemployment and death rate (Brenner, 1971; Brenner, 1979). He estimated that, in the United States, an increase in unemployment rate of one percentage point results in 36,887 deaths over the course of 6 years (Brenner, 1976). Since publication, however, Brenner's methods have been called into question (Kasl, 1979; Gravl et al., 1981; Wagstaff, 1985; McAvinchey, 1988). While some of the criticism levied is specific to Brenner's analysis, even subsequent researchers who have improved on his work using time-series analysis fail to find robust support for his findings that mortality rate is countercyclical with unemployment (McAvinchey, 1988). University of Virginia Professor

Christopher Ruhm asserts that time-series analyses of any variety struggle to account for omitted variables correlated with economic conditions and health (Ruhm, 2015).

Ruhm, in his own work, finds evidence contradicting the long-held belief that economic growth and improved health go hand in hand. Using panel data (a modified variation of his model is explained below in the "Quantitative Analysis" section), Ruhm shows that in his period of analysis (1972-1991) unemployment and mortality are negatively correlated — in economic downturns people are actually healthier (Ruhm, 2000). Ruhm's findings were supported by studies across numerous countries and time periods. A procyclical relationship between mortality and unemployment has been shown in Germany between 1980 and 2000 (Neumeyer, 2004), in Spain from 1980-1997 (Tapia Granados, 2005), in France from 1982-2003 (Buchmueller et al., 2007), in Canada from 1977-2009 (Ariizume & Schirle, 2012), in Mexico from 1993-2004 (Gonzalez & Quast, 2004), in Norway from 1977–2008 (Haaland & Telle, 2015), and across OECD countries from 1960-1997 (Gerdtham & Ruhm, 2006).

However, more recent analyses conducted using similar aggregate models of health, show that this procyclical relationship between economic growth and health has been greatly diminished, with some studies finding no correlation at all. Ruhm himself finds that the relationship has been weakened in recent years, using data from 1976-2009 in the United States (Ruhm, 2015b). At the aggregate level, researches have shown that a one percentage point increase in unemployment reduced mortality rate by 0.40 percent from 1978-1991, but only by 0.19% when extending from 1978-2006 (Stevens et al., 2011). Similarly, a one percent increase in joblessness among seniors lowered mortality rate 0.3 percent from 1976-1991 but raised it 0.5 percent from 1994-2008. Ruhm argues that this can be explained by the increasing importance of cancer as a cause of mortality,

which is countercyclically related to economic growth, as expensive technological developments have improved cancer treatment, but are likely less affordable in worse economic conditions (Ruhm, 2015b). Out of these findings, an emerging academic consensus finds that there is no stable relationship between economic indicators and well-being in industrialized countries. The association instead depends on sociological context (Burgard & Kalousova, 2015).

Other researchers have studied the association between recessions and health changes on the individual instead of the aggregate, and as a consequence, can identify mechanisms through which these aggregate-level associations can vary depending on the context. In terms of leading mortality causes, traffic-related fatalities and deaths from cardiovascular disease are both strongly procyclical, and thus decrease in recessions (Ruhm, 2015b). Both can be explained by working less, as fewer individuals are commuting to work, and job strain increases the risk of cardiovascular problems (Schall et al., 1994). Suicide, on the other hand, has been shown to be countercyclical in the majority of cases (Catalano et al., 2011; Modrek et al., 2013). These causes may counter each other depending on their prominence in given contexts. On the individual level, researchers suggest that fluctuations in the business cycle can affect health through different behavioral mechanisms as well, with both positive and negative implications. Reduced working hours can lead to changes in time use, as well as changes in stress levels. Whether these changes affect health positively or negatively depends on context. Additionally, weaker economies can change both wealth levels and government spending levels, which can induce consumption changes. Thus, both individual-level and aggregate drivers of changes in health effect are contradictory, meaning their overall effect is likely to be context specific (Burgard & Kalousova, 2015).

Section Two: Health and Economic Crises

While the above literature describes the relationship between economic indicators and health outcomes, it does not differentiate between normal downturns and crises. For instance, there is no reason to expect, as most models do, that the relationship between unemployment and health is linear. The difference on health between one and two percent unemployment may be dramatically different than the impact of moving from 14 to 15 percent. Thus, to understand the Great Recession's health effects, supplementary literature on the health effects of crises will be useful. The great depression is the most prominent crisis in American history. Studies have found aggregate health improvements during the great depression, largely driven by a decrease in deaths from respiratory infection. However, the only effect that can be causally attributed during the Great Depression is that suicides occurred at a higher rate in regions with more bank suspensions (Stuckler et al., 2012). Given the context-specific nature of the association between health and recessions, the Great Recession may well differ from findings in previous crises.

Section Three: Health and the Great Recession

While few studies have been conducted measuring aggregate effects of the Great Recession, studies of unemployment and mortality in the E.U. during the Great Recession, similar in structure to the studies done by Ruhm, showed no effect of unemployment on mortality, although the expected increase in suicides and decline in traffic-related fatalities was confirmed (Stucker et al., 2011; Toffolutti & Suhrcke, 2014). In terms of physical health, individual-level studies measuring physical health in the Great Recession reported that self-reported health (SRH) measures declined (Burgard et al., 2012; Cannuscio et al., 2012; Drydakis, 2015; Reile et al., 2014; Yilmazer et al., 2015; Schootman et al., 2012). Aggregate-level studies generally supported this, with perhaps the most

robust study (quarterly data was reported, making estimates more precise) showing an initial increase in SRH but long-run declines for individuals in the United Kingdom (Astell-Burt & Feng, 2013). Individuals were also more likely to report morbidity factors such as suffering from disability or ailments such as backache, diarrhea, fatigue, and sleeping problems during the recession (Cannuscio et al., 2012). A study of an individual firm showed that even those who survived layoffs faced a cost, as two Stanford professors studied a large aluminum plant that underwent cutbacks and found that employees not laid off still reported increased hypertension and diabetes (Modrek & Cullen, 2013). Additionally, a neighborhood level study of Detroit showed that an increase in abandoned homes was associated with a decrease in immune system function, measured through thymic aging (McClure et al., 2018). In all, while limited aggregate-level studies showed that the Great Recession had no effect on physical health and mortality, individual-level research showed negative effects.

The Great Recession also had an effect on mental health. At the individual level, those who reported loss of job, income, or wealth were at higher risk of distress in both the United States and Europe (Drydakis, 2015; Brenner et al., 2014; Riumallo-Herl et al., 2014; Urbanos Garrido & Lopez-Valcarcel, 2015; McIerney et al., 2013). And although those directly affected suffered the most, uncertainty led to negative effects for those who remained employed as well (Sargent-Cox et al., 2011; Tekin et al., 2013). One study showed that mental health effects among the general population were worse in the United States than Europe, asserting that the lack of social safety net in the United States was the cause (Riumallo-Herl et al., 2014). Others, however, attribute this difference to the nature of the housing crisis (the health impacts of which are discussed in more depth later), which was unique to the United States (Houle, 2014). The decrease in mental health can be seen beyond reported measures as well, affecting the number of psychiatric disorder diagnoses

and observed suicidal behavior. Aggregate level studies on differences in diagnosable depression and anxiety, find substantial increases across countries, including the United States (Mehta et al., 2015), Europe (Economou et al., 2013; Gili et al., 2013), and Asia (Lee et al., 2010). Numerous studies show that the likelihood of suicidal behavior -— both deaths and attempts — has been shown to increase in the aftermath of the Great Recession as well, particularly among working-aged men, consistent with aggregate-level mortality rate findings discussed above (Reeves et al., 2014; Reeves et al., 2015; Corcoran et al., 2015; De Vogli et al., 2013; De Vogli et al., 2014; DeFina & Hannon, 2015; Gemmill et. al, 2015; Nortsröm et al., 2015; Phillips and Nugent, 2014; Fowler et al., 2015).

The Great Recession affected various health-related behaviors as well, which could help inform our analysis on what drives changes in health outcomes. One positive change was in time use, where researchers found that roughly 50 percent of hours gained from reduced work went toward leisure activities. Significant increases were found in home production (30 percent of all hours), caring for children, and time spent on education as well (Aguiar et al., 2013). However, working conditions for those with jobs have been shown to worsen during recessions, increasing stress (Tausig & Fenwick, 1999). The recession affected important consumption habits as well, including nutrition, alcohol consumption, and smoking. In the United States, both aggregate and individual level studies showed that unemployment was associated with less healthy nutrition decisions and more calories purchased (Ng et al., 2014; Macy et al., 2013). While aggregate-level analyses shows a decrease in alcohol consumption, (Tekin et al., 2013; Bor et al., 2013; Harhay et al., 2014; Nandi et al., 2013) individual-level studies show that for those who lost their jobs or experienced housing distress, alcohol consumption and problematic drinking increased (Richman et al., 2012; Vijayasiri et al., 2012; Richman et al., 2014; Mulia et al., 2014; Murphy et al., 2014). This

implies differential effects on alcohol consumption depending on how the crisis affected you — while those affected most drank more, the population as a whole drank less. Smoking evidence is similarly mixed. Individual-level studies showed that those experiencing economic strain were more likely to smoke (Kalousova & Burgard, 2014), while a closer analysis reveals that this increase was much higher for those were were less educated (Macy, 2013) and that this education gap was noticed similarly in differential odds of quitting smoking for younger adults (Benson, 2015). Behavior changes were mixed, with positive time-use changes for those with reduced work offset by stress increases for those whose employment remained unchanged. Consumption behavior was mixed as well, as even though nutrition worsened, the effect on drinking and smoking depended on the population.

While thus far, most of the research treats the crisis holistically, some authors try to discern the mechanisms that could have made the Great Recession unique from other crises, with a potential mechanism being the housing crisis. Individual-level studies show that those affected by the housing crisis had negative mental health effects (Cagney et al., 2014; McLaughlin et al., 2012; Osypuk et al., 2012; Burgard et al., 2012, Cannuscio et al., 2013; Yilmazer et al., 2015) and increased suicide rates (DeFina & Hannon, 2015; Fowler et al., 2015; Houle, 2014). Studies on physical health, however, were mixed (Yilmazer et al., 2015; Arcaya et al., 2014). We could not find aggregate-level studies attempting to differentiate between the effects of unemployment and the housing crisis. While this area of study remains incomplete, the early evidence shows that the housing crisis may have been a significant differentiator between the Great Recession and crises that originated in other sectors.

Although long-term impact is outside the scope of research we're capable of conducting, yet another mechanism that could have affected health outcomes is increased inequality. Inequality has increased since the recession (Cynamon & Fazzari, 2016), potentially caused by the crisis itself, and decisions made by lead policy actors to prioritize rescue over relief in response. Inequality has been associated with worse health outcomes across the income distribution (Kaplan et al., 1996; Kennedy et al., 1996; Kennedy et al., 1998; Lynch et al., 1998; Waitzman and Smith, 1998; Soobader and LeClere, 1999). Additionally, while much of the research has focused on average impact, there has been evidence the financial crisis disproportionately negatively affected racial minorities (Tekin et al., 2013; Houle, 2014; Lo and Cheng, 2014). Although the literature in this area is sparse, it suggests that inequality resulting from the Great Recession may well reduce health outcomes for everyone.

Section Four: Summation

In sum, while aggregate-level analyses once showed a clear procyclical relationship between the economy and mortality rate, most analyses of the 21st century show no effect. The prevailing wisdom is instead that the relationship is entirely context specific. Additionally, while crises may differ from general fluctuations, the great depression showed mild health benefits, not discernibly different that our understanding of the 20th century relationship between health and the business cycle in the United States generally. Early evidence from the Great Recession specifically however is mixed. Aggregate-level studies showed no effect on mortality in Europe, and generally muted impacts on health overall, whereas individual-level studies showed those most affected by the crisis faced negative health consequences. We could not find aggregate-level studies of mortality rate in the United States, so our analysis will attempt to quantify the Great Recession's effect on mortality in the United States, and show how it fits within the broader academic consensus.

Quantitative Analysis

Section One: Introduction and Motivation

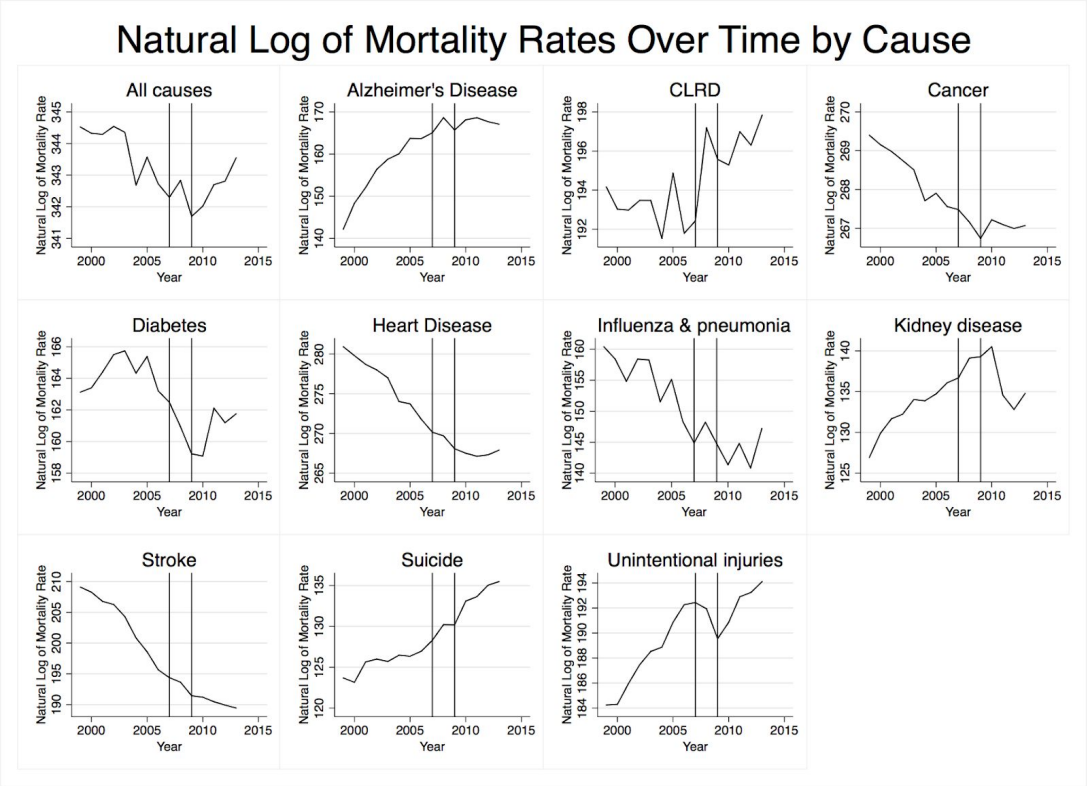
The main question we seek to answer through this analysis can be stated quite simply as: "What is the causal effect of the Great Recession on mortality?" More specifically, this analysis attempts to confirm the general consensus from aggregate-level research on the association between economic indicators and health and apply it to a new set of years, and tries to ascertain the overall effects of the recession on health, both in comparison to other studies of crises, and in a vacuum.

Figure one shows the trends in mortality (the natural log of the mortality rate) for all states during the period of analysis (1999-2013). The vertical bars represent the beginning and end of the declared portions of the financial crisis in 2007 and 2009[1], in order to identify areas of focus for our primary analysis. In 2013, these top ten leading causes of death accounted for 73.6 percent of all deaths in the United States (Kochanek, Murphy, Xu, & Arias, 2014).

The rest of the paper proceeds as follows. Section II provides a description of the data we use in our analyses. Section III goes over the theoretical models we use and our identification strategy framework. Section IV discusses our primary results. Section V provides robustness checks based on various time-dependent definitions of the financial crisis. Section VI briefly explores differential effects of mortality by state. Finally we conclude with a discussion of results, including caveats, and recommends of other areas for potential research.

[1] The authors recognize that defining a single start date for the financial crisis does not reflect the cascade-like manner in which the crisis unfolded. However, a definite start date is necessary for the analysis. While 2007 is used as the base definition, alternate specifications are tested under "Supplemental Analyses"

Figure 1: Mortality Rates by Cause Before, During (2007-2009), and After Great Recession



Note: Figure plots collective (all states and DC) log mortality rate by cause (accounting for the top 10 causes of mortality and all causes combined). Data from CDC Mortality files.

Section Two: Data

The data for our analysis comes from three sources. First, we utilize data from the National Center for Health Statistics (NCHS) and the Centers for Disease Prevention and Control (CDC) containing information on the ten leading causes of death in the United States as categorized from the tenth version of the International Classification of Diseases, Tenth Revision (ICD-10 codes) (Murphy, Xu, Kochanek, Curtin & Arias, 2017). This data contains the number of individuals who die each year in each state and Washington, DC for each of the given ten causes — Alzheimer's Disease, chronic lower respiratory diseases (CLRD), cancer, diabetes, heart disease, influenza and pneumonia, kidney disease, strokes, suicide, and unintentional injuries — as well as an aggregate death count for all causes.

In addition to these mortality data, we use data from the National Cancer Institute's Surveillance, Epidemiology, and End Results Program (SEER) (SEER, 2017). We use the adjusted SEER data that are adjusted for population shifts due to hurricanes Katrina and Rita in order to compute population levels for each state that we ultimately use to compute mortality rates per state per year, as well as a suite of demographic controls. We define mortality rate to be the number of deaths in a state per 100,000 people. The demographic controls we utilize, derived from SEER data include percentage of the population that is female, dummy variables for percent of the population that falls into various age ranges (age zero, five year buckets from 1-84, and then 85 and over), and race characteristics (white, black, and other).

Lastly, we use the Bureau of Labor Statistics (BLS) Local Area Unemployment Statistics (LAUS) data as a proxy for other macroeconomic effects. These data contain the average annual unemployment rate (civilian noninstitutionalized individuals age 16 or older who do not have a job, are currently available for work, and have actively looked for work in the prior four weeks or are waiting to be recalled to a job for which they have been laid off) for states for each year.

For all of these data sources, we limit our analysis between the years 1999-2013. Summary statistics can be found in Appendix Table One. We specifically make the decision to limit our analysis to before 2013, as after that date, many provisions of the Affordable Care and Patient Protection Act of 2010 (also known as the ACA or Obamacare) go into effect which we believe may have a significant impact on some measures of mortality by granting individuals better access to health insurance.

Section Three: Theoretical Model and Methods

As described above, for years, the conventional wisdom from aggregate level analyses showed a fairly robust procyclical relationship between economic indicators and mortality rate (Ogburn & Thomas, 1922; Ruhm, 2000). However, the most recent literature shows this procyclical relationship to be diminishing. Our base model will, in line with the historical literature relating economic fluctuation to health outcomes, attempt to assess whether this finding of no relation holds in the era of the financial crisis.

In addition to this general trend, we seek to determine a causal estimate of the impact of the 2008 financial crisis on mortality. While many 20th century studies attempting to estimate the effects of recessions on morbidity and mortality relied heavily on national time-series analysis (Brenner, 1971; Brenner, 1979), these methods have been criticized in recent years, as time-series regressions may have biased estimates due to omitted factors that are spuriously correlated with economic conditions and health (Ruhm, 2015a).

In light of these criticisms of time-series analyses as well as other potential serial correlation issues, we instead adapt a model from Ruhm (2015) that uses panel data containing measures of mortality outcomes for multiple states over several time periods (annual data from 1999-2013):

$$\ln(M_{cst}) = \alpha_s + X_{st}\beta + U_{st} + \pi_{st} + \delta_{ct} + \varepsilon_{cst} \qquad (1)$$

In model 1 above, the dependent variable is the natural log of the mortality rate M, which is defined as the number of deaths per 100,000 people in state *s* for cause *c* at time *t*. α represents state fixed effects for all 50 states and the District of Columbia, X represents a vector of demographic characteristics for a state in a given year, U represents our goal of trying to capture the separate

macroeconomic effects of the annual unemployment rate for a given state in a given year, π represents a state-time trend, and δ represents the parameter of interest, a variable indicating that the crisis has begun (indicates years 2007 and on). δ, therefore, represents the impact of the financial crisis beyond through changes in unemployment alone on the natural log of the death rate for a given cause of mortality. Normally, we would add year fixed effects, however, they would be collinear with our time-dependent indicator of the financial crisis. We utilize state-clustered, heteskedatic robust standard errors.

To further estimate the impact, we consider a series of other, similar models. Consider specification (1'), below:

$$\ln(M_{cst}) = \alpha_s + X_{st}\beta + \pi_{st} + \delta_{ct} + \varepsilon_{cst} \qquad (1')$$

This specification is nearly identical to (1) above, with the only difference being the elimination of the separate macroeconomic effect (U), the unemployment rate. One considerable mechanism for mortality in the financial crisis could be via job loss. Schaller and Stevens (2014) assert that job loss in the United States is associated with long-term increases in mortality rates, including lower self-reported health and mental health. Therefore, we may want to see the macroeconomic effects absorbed in our coefficient estimating the effect of the crisis, especially since these effects are mechanisms by which individuals experience impacts of the financial crisis, which would be captured in δ in this specification.

We also consider alternate specifications with an indicator for when the recession is taking place (during), and one for after the recession (post) to examine the differential treatment effects, and potential lagged effects of the financial crisis on mortality. In specification (2) below, D is an indicator variable representing the years during the crisis, which we define to be 2007-2009,

inclusive, for the sake of our analysis. P is an indicator variable representing the years post-crisis, i.e. 2010 onward.

$$\ln(M_{cst}) = \alpha_s + X_{st}\beta + U_{st} + \pi_{st} + D_{ct}\lambda + P_{ct}\delta + \varepsilon_{cst} \qquad (2)$$

Specification (2') below, is similar to (2) in its inclusion of both a during and post-crisis indicator variable, but also foregoes a separate indicator for macroeconomic trends, similar to specification (1').

$$\ln(M_{cst}) = \alpha_s + X_{st}\beta + \pi_{st} + D_{ct}\lambda + P_{ct}\delta + \varepsilon_{cst} \qquad (2')$$

Section Four: Results

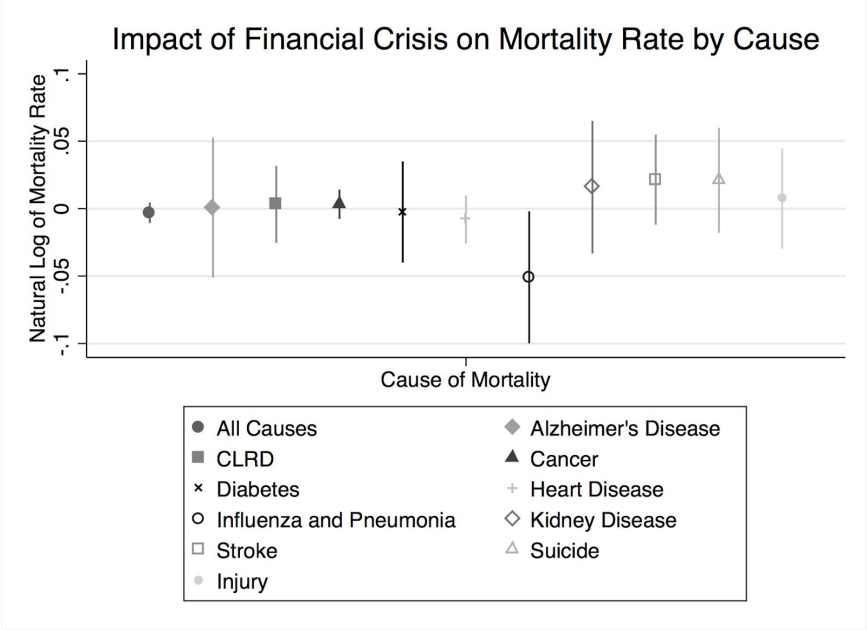
Consider specification (1), where the outcome of interest is the indicator for being under the influence of the financial crisis (years 2007 and on). Table One shows the coefficient of Interest, mortality rates by cause, attempting to estimate the causal effect of the financial crisis on each type of mortality. We clearly observe in this model that there the financial crisis has little impact on mortality rates. Figure 2a shows that the only statistically significant change in mortality once individuals are under the impact of the financial crisis is a slight decrease in mortality due to influenza and pneumonia of about five percent.

Table 1: Regression Results for Effect of Financial Crisis on Mortality for Specifications (1) and (1') (Impact defined as 2007-onward, inclusive)

Type of Mortality	(1) Coefficient indicating under influence of crisis δ in (1)	(2) Coefficient indicating under influence of crisis δ in (1')
All causes	-0.00304	-0.00137
	(0.00381)	(0.00395)
Alzheimer's Disease	0.000787	0.00836
	(0.0258)	(0.0243)
CLRD	0.00313	0.00381
	(0.0142)	(0.0140)
Cancer	0.00330	0.00268
	(0.00542)	(0.00536)
Diabetes	-0.00250	-0.000594
	(0.0187)	(0.0183)
Heart Disease	-0.00801	-0.00644
	(0.00885)	(0.00881)
Influenza and Pneumonia	-0.0509**	-0.0471*
	(0.0243)	(0.0235)
Kidney Disease	0.0159	0.00688
	(0.0245)	(0.0242)
Stroke	0.0215	0.0214
	(0.0166)	(0.0162)
Suicide	0.0210	0.0186
	(0.0194)	(0.0196)
Unintentional Injury	0.00745	0.0129
	(0.0185)	(0.0180)
Control for Unemployment	Yes	No
Observations per Regression	765	765

Note: Table shows regression results where dependent variable is the natural log of the mortality rate per 100,000 people. Sample period is 1999-2013. Reported coefficients are the indicator variable for the crisis being "active" in the specifications which is 2007-onward, inclusive. Each reported coefficient represents a single regression. Regressions control for state demographics (5 year age buckets, percent female, and racial composition) and a linear state-specific time-trend. The left panel runs specification (1), the right panel runs specification (1'). Heteroskedastic robust standard errors, clustered at the state level are reported in parentheses. *** p<0.01, ** p<0.05, * p<0.1.

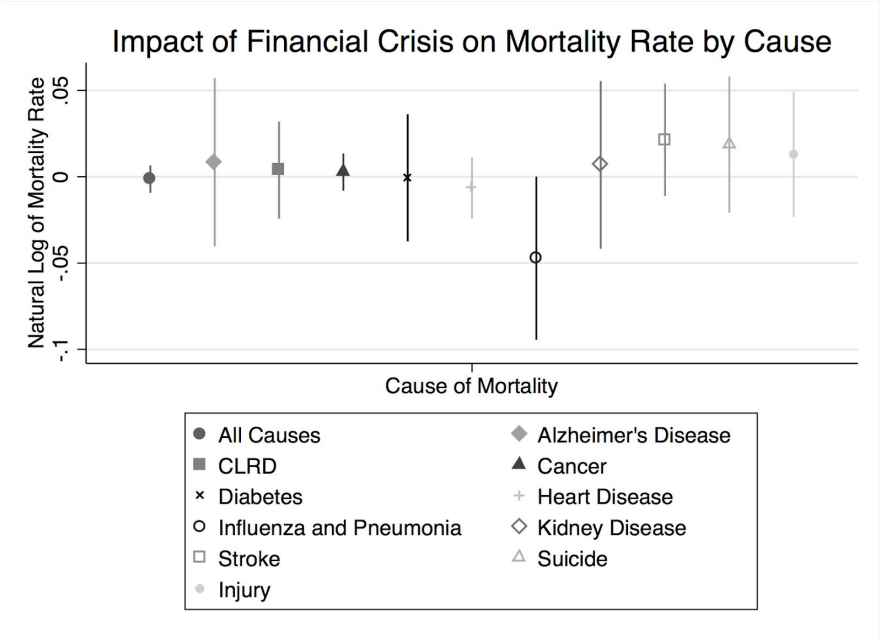
Figure 2(a): Coefficients from Specification (1) for Crisis Years 2007-Onward by Cause



Note: See left panel of table one. Plots coefficients from specification (1) as listed in table one. Bars represent a 95 percent confidence interval.

To see if the results are heavily dependant on the controlling for macroeconomic effects (state unemployment rates, in our case), we estimate specification (1') under the same conditions and time definition, with results are reported in table one and figure 2b. We find that the exclusion of the macroeconomic controls, has no practically no effect on the estimated impact of the crisis on mortality rates. The only change is the decrease in mortality rates due to influenza and pneumonia being significant at 5% level in specification (1), compared to a 10% level in specification (1'), with the new coefficient indicating a 4.7 percent decrease in the mortality rate of influenza and pneumonia.

Figure 2(b): Coefficients from Specification (1') for Crisis Years 2007-Onward by Cause



Note: See right panel of table one. Plots coefficients from specification (1') as listed in table one. Bars represent a 95 percent confidence interval.

We transition our analyses to looking at separate effects of "during crisis" impacts on mortality as well as "after crisis" impacts on mortality. There are a number of reasons why there may be different impacts from the immediate crisis period (2007-2009) compared to the aftermath, 2010-onward. Consider the following potential mechanisms, since the outcome we are measuring is mortality, we may expect the crisis to cause several acute changes in health behavior that may result immediately in mortality. However, there is a potential for there to be a lagged effect of the crisis on mortality, i.e. morbidity may increase during the crisis period, but mortality may not set-in until after the crisis, which would increase the mortality rate. However, if the government were to prioritize relief and recovery, such as they did when Congress passed the American Recovery and Reinvestment Act (ARRA) of 2009, which provided over $8 billion in increases to Supplemental Nutrition Assistance Program (SNAP) recipients and $150 million for Emergency Food Assistance programs (United States Department of Agriculture, 2010), there is potential for a marked decrease

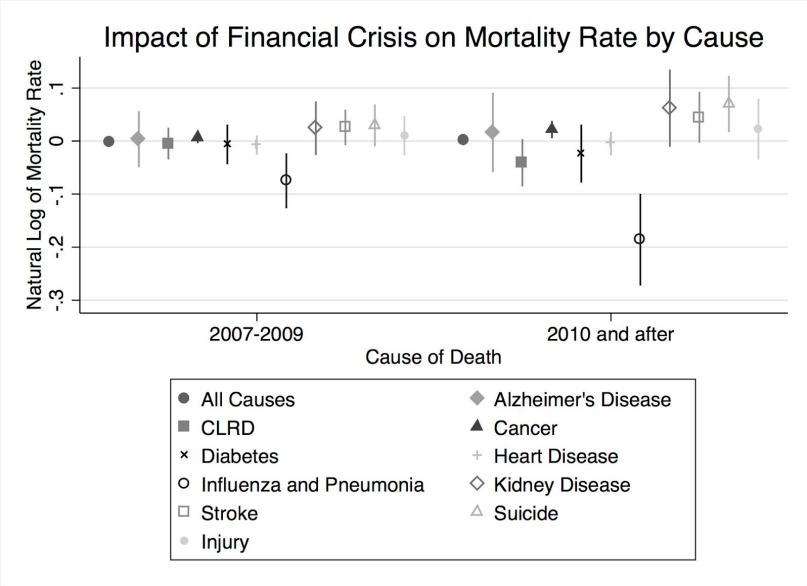
in mortality in the post-crisis period, relative to the other periods. Figure 3a below plots the coefficients for D, indicating "during the crisis" (2007-2009), and P, "post-crisis" (2010-onward) from specification (2).

During the crisis, we observe very little change in mortality rates, just a decrease in mortality due to influenza and pneumonia (roughly 7.5 percent decrease), which is consistent with our findings in specifications (1) and (1'), just slightly larger. After the crisis, we see a bit more variation in mortality rates by cause. We still see a statistically significant decrease in mortality due to influenza and pneumonia (increasing significantly in magnitude to a decrease of 18.6 percent), but also see a statistically significant increase in mortality due to cancer (roughly two percent). This increase in cancer is consistent with present literature finding that countercyclical patterns have emerged with cancer over time (Ruhm, 2015b), most likely reflecting the importance of financial resources to purchase sophisticated and expensive treatments and prescription drugs, which individuals have less capacity to do during economic downturns, such as after the crisis. We also find an increase in suicide rates after the crisis (roughly two percent), which is consistent with the literature indicating that suicide rates are countercyclical with economic conditions (Catalano et al., 2011; Modrek et al., 2013). The fact that we observe these conditions during the post period (2010 and after) rather than "during" suggests that there may be a lagged impact of the crisis effects that result in mortality. In aggregate, however, in both the "during crisis" and "post crisis" periods, we don't see a significant increase in all-cause mortality.

Figure 3(b) above shows results with specification (2') which find practically identical results to those in specification (2), indicating that the results are not sensitive to the inclusion of a control accounting for macroeconomic conditions via unemployment rate. As can be seen in Appendix

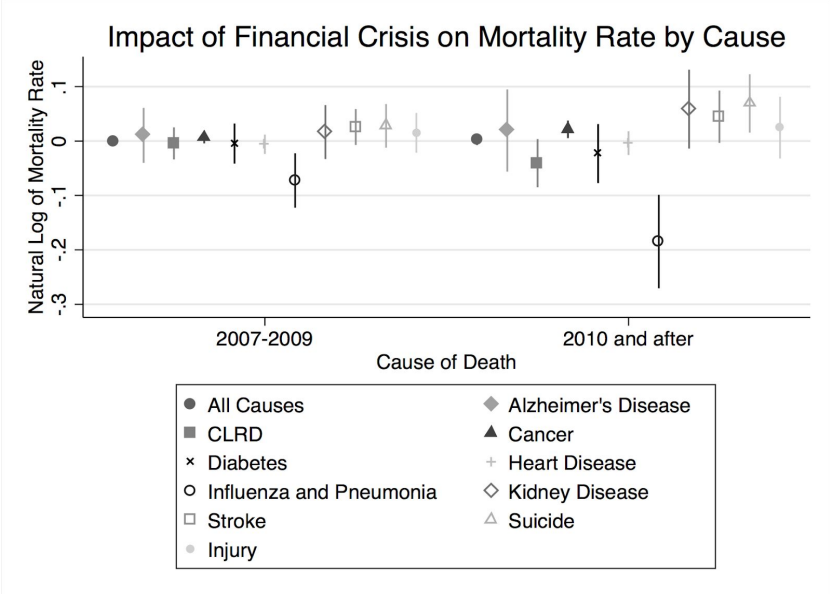
Table 2, with the exception of influenza and pneumonia during the crisis (which differs by .26 percentage points), the difference in point estimates for statistically significant mortality effects is less than one-tenth of a percentage point.

Figure 3(a): Coefficients from Specification (2) by Time Period and Cause



Note: Data comes from left panel of Appendix Table 2. Plots coefficients from specification (2). Bars represent a 95 percent confidence interval.

Figure 3(b): Coefficients from Specification (2') by Time Period and Cause



Note: Data comes from right panel of Appendix Table 2. Plots coefficients from specification (2'). Bars represent a 95 percent confidence interval.

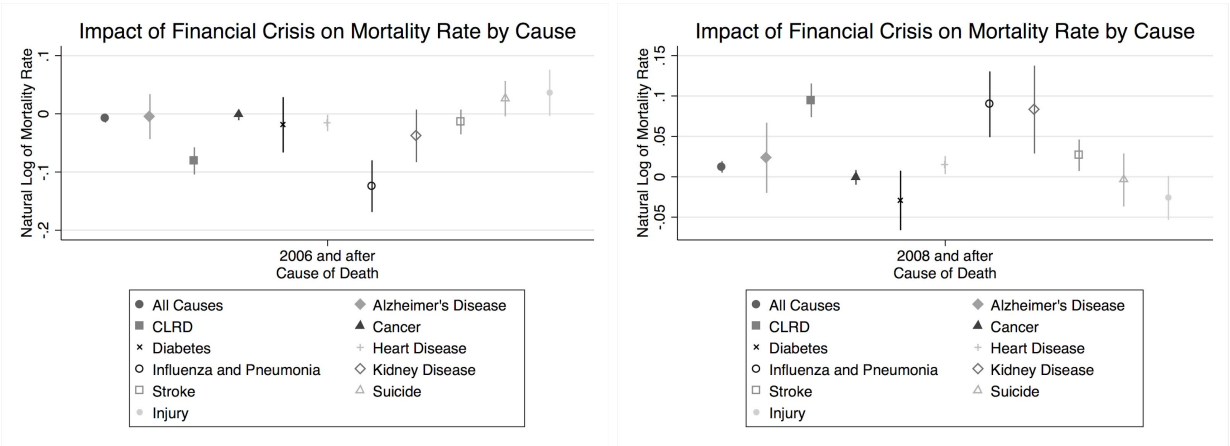
Supplementary Analyses

Section Five: Timing of Crisis Robustness Check

We are cognizant of the fact that there is significant debate regarding when the financial crisis began. Additionally, we realize the argument that the financial crisis didn't "begin" at a certain point, i.e. a pivotal turning point, but rather a cascade of events during the mid-2000s. In order to account for this and see if our results are sensitive to it, we vary the years we use to define the beginning and end of the financial crisis. Since our primary analyses found practically no impact of the inclusion for macroeconomic controls, we will continue this analysis with specifications (1') and (2'), that do not include LAUS data controlling for state unemployment rates.

We estimate specification (1') by redefining the cutoff for the effects of the crisis to being in 2006 and 2008 respectively in the models below:

Figures 4(a-b) Time-Dependent Robustness Checks of Specification (1')

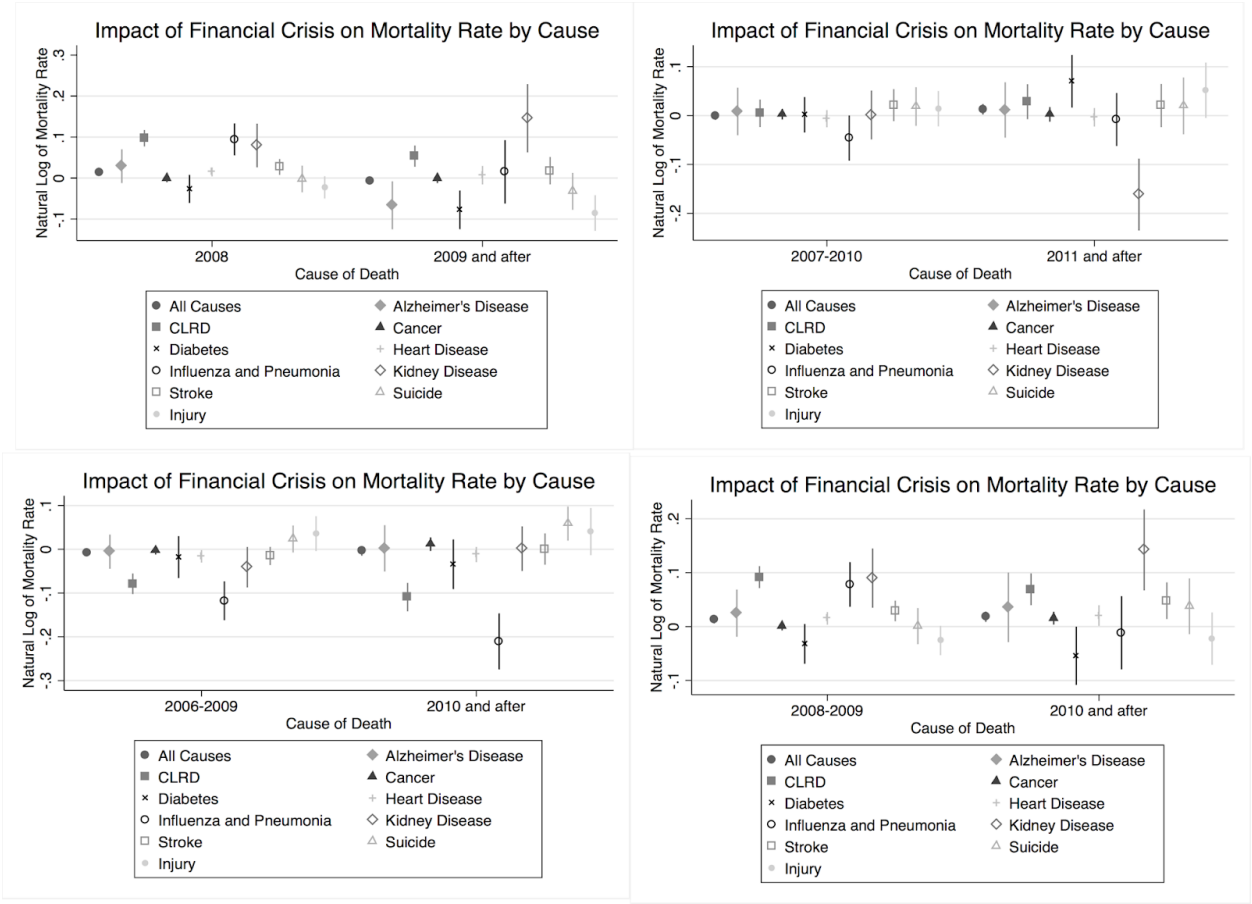


Note: Data comes from left panel of Appendix Table 3. Plots coefficients from specification (1') with varying definitions of crisis start and end times. Bars represent a 95 percent confidence interval.

In the original specification of (1'), using 2007 as the crisis cutoff, we observe no statistically significant changes in any form of mortality. Based on the above figures, it appears that the model is very sensitive to the definition of the beginning of the crisis. When we define the start to be 2006, we observe statistically significant decreases in mortality due to chronic lower respiratory diseases (CLRD) (decrease of roughly 8 percent), heart disease (a roughly 1.5 percent decrease) and influenza and pneumonia (a roughly 12.4 percent decrease). When we definite the effects of the crisis to being in 2008, we see the overall impact of the crisis on mortality intensify. We observe an overall statistically significant increase in the all-cause mortality rate (around 1.22 percent), as well as cause-specific increases in the mortality rate for chronic lower respiratory diseases (9.47 percent), heart disease (around 1.5 percent), influenza and pneumonia (around 9 percent), kidney disease (around 8.3 percent), and stroke (around 2.7 percent).

In the original specification of (2'), defining the crisis between 2007-2009, and post-crisis effects in the years 2010 and after. During the crisis, we observe little change in mortality, with only a notable decrease in deaths due to influenza (around 7 percent). In the after crisis period (2010 and after), there were statistically significant increases in mortality due to cancer (around 2 percent) and suicide (around 7 percent), and decreases due to influenza and pneumonia (around 18.5 percent). As can be seen from the figures below, the specification of years drastically changes the coefficients identifying the effect of the financial crisis on various causes of mortality.

Figures 5(a-d) Time-Dependent Robustness Checks of Specification (2')



Note: Data comes from Appendix Tables 4a and 4b. Plots coefficients from specification (2') with varying definitions of crisis start and end times. Bars represent a 95 percent confidence interval.

When we restrict our definition of the financial crisis to 2008, we see a slew of statistically significant increases in mortality, perhaps indicating that this year was the most significant and why individuals perceive the Financial Crisis to be the "Crisis of 2008," based on when people experienced impacts relating directly to the "real economy." As can be seen in the upper left panel of figure 5 and Appendix Table 4a, we observe statistically significant increases in the mortality rates for all causes (1.34 percent), chronic lower respiratory disease (9.72 percent), heart disease (1.5 percent), influenza and pneumonia (9.43 percent), kidney disease (around 8 percent), and stroke (2.71 percent). In the years 2009 and after, we observe more mixed results with mortality. If we

expand our definition of the crisis by one year from our original (2') analysis to 2007-2010, we observe no statistically significant changes of mortality during the crisis, which is much more consistent with our original model. As can be seen in the upper right panel of figure 5, differences begin to appear in the post period, where we observe an increase in overall mortality (1.3 percent), and increases in mortality due to diabetes (around 7 percent) and a decrease in kidney disease (around 16 percent decrease). Based on our exploration of the literature, while aggregate analyses do not show aggregate increases to kidney disease and diabetes, an analysis of an individual firm showed an increase in diabetes among those who were laid off (Modrek & Cullen, 2013).

If we begin our crisis definition a year earlier (bottom left panel of figure 5), compared to our (2') analysis, we are more apt to find decreases in mortality rates, contributed to all causes (around 0.78 percent), CLRD (around 7.8 percent), heart disease (around 1.6 percent), and influenza and pneumonia (around 11.8 percent), as well as decreases in CLRD (10.9 percent), influenza (21.1 percent), and increases in suicide rates (around 5.9 percent) in the post period (2010-onward), which is largely consistent with the primary post-period findings of our primary (2') analysis. This could be because we are capturing more of the economic fluctuation by increasing our definition of the crisis, and magnifying cyclical variations shown to be prevalent in both suicide and respiratory disease, albeit with opposite effects (De Vogli et al., 2014; Miller et al., 2009). Finally, if we constrain our "during crisis" period to between 2008-2009, such as is the case in the bottom right panel, we observe treatment effects that are largely consistent in both the "during crisis and "after crisis" periods, with consistent increases in all cause mortality (1.29 percent), CLRD (9.17 percent), heart disease (1.52 percent), influenza and pneumonia (7.82 percent), kidney disease (9 percent), and stroke (2.91 percent). All cause (1.83 percent), CLRD (6.91 percent), cancer (1.56 percent), heart

disease (2.04 percent), kidney disease (14.2 percent), and stroke (4.8 percent) had statistically significant increases in mortality in the post period. We found diabetes to be decreasing by about 5.43 percentage points in the post period, as well.

Section Six: Modeling Differential State Effects

We sought to explore how the financial crisis impacted different states' mortality rates. A priori, we felt that there may be some states that were more harshly impacted than others, for example, we thought that states like New York that are heavily dependent on the financial sector, states like Michigan and Ohio, which are in the "rust belt," and states that were very impacted by housing prices such as Nevada and California, may have felt some of the hardest impacts and thus, had higher mortality rate changes. Additionally, we speculated that heavily agricultural dependant states may have been less impacted by the financial and wouldn't see as significant changes in mortality rates. We also think that states impacted by Hurricane Katrina could possibly have higher mortality rates as well. For this state differential analysis, we chose to focus on changes in suicide rates:

In order to establish a causal effect, we adapt specification (3) to include a state differential trend during the post period, since that is the period where we saw significant increases in the suicide rate in that specification:

$$\ln(M_{cst}) = \alpha_s + X_{st}\beta + \pi_{st} + D_{ct}\lambda + P_{ct}\delta + S * P_{ct}\theta + \varepsilon_{cst} \qquad (3)$$

The coefficient of interest here is θ which represents an interaction of each state S on the post indicator variable P. This creates a dummy variable for every state indicating its relative differential effect. For our analysis, we seek to explore state differential effects for suicide rates. In

the regression analysis, we exclude the interaction between the state that has the median state-specific suicide rate from this specification to determine the states that, on average, have significantly higher suicide rates and significantly lower suicide rates to see if they compare with our a priori speculation.

We find that the states with the top five lowest suicide rates (greatest negative differential effect) are Colorado, Maryland, Mississippi, Wisconsin, Minnesota, with Colorado being the most negative of the lot. The states that have the top five highest suicide rates (greatest positive differential effects) are Hawaii, Wyoming, New Jersey, Oklahoma, and Louisiana, with Hawaii being the most positive of the lot.

Ultimately, we conclude that this very naive state differential analysis doesn't seem to start to address a pattern in terms of which states were most disparately impacted. No region stands out as particularly more or less impacted. We recommend this as an area of more intense exploration, as there is a lot of literature surrounding the idea of disparate impacts of the financial crisis on mortality.

Discussion

Findings

We determine via our robustness checks that our results are highly dependent on the time-based definition of the financial crisis. This suggests that rather than an event with a discrete beginning and end, the crisis' impacts may have been more of a cascading effect. We see the mortality effects of the crisis being more pronounced in the years following the crisis. For example, we generally see more significant increases in mortality in our "crisis-active" and "post" effects when we run specification (2) and (2'), respectively, and see more pronounced effects when we define the start date of the crisis later for specification (1) (see column 2 of Appendix Table 3). The potential

implications of this are two-fold. First, we believe that the mortality impacts of the crisis may be lagged. This could either because of an increase in morbidity factors during the crisis, but mortality not setting in until after, or possibly that the impacts to the real economy may not have really hit substantially until after the peak of the financial crisis.

Our results with regards to an increase in all-cause mortality vary by specification and timing, however we never see a statistically significant decrease, suggesting that we may be able to effectively put a lower bound on our mortality estimates and determine that the financial crisis never decreased the mortality rate in aggregate.

We see virtually no effect of the crisis on Alzheimer's disease. Since this disease is much more common among elderly individuals, it begs the question if the impacts would be present in a differential treatment effect analysis by age, and whether the financial crisis exacerbated present trends. The results on chronic lower respiratory disease are relatively mixed across specifications and time periods, which may be reflective of its sensitivity to economic indicators. We typically see more intense increases in mortality rate estimates when we when we define the crisis to take place later (see Appendix Table 3, for example). This finding is relatively consistent with the literature, which finds that economic downturns are associated with lower rates of mortality due to respiratory issues. By defining the crisis later, we may be capturing less of the economic downturn and thus not absorbing the decrease in respiratory mortality that is associated with that.

We tend to see an increase in mortality due to stroke, especially post-crisis, but some specifications find the effect during the crisis as well. When we saw effects of cancer, we exclusively saw increases in the mortality rate in the post period using specification (2) and (2'). We believe that this finding is consistent with predominant academic theories regarding the increasing costs of

cancer treatment, as while new technologies can effectively combat cancer, they are relatively expensive (Ruhm, 2015b). We saw very mixed results on diabetes, heart disease, unintentional injury, and kidney disease.

Specifically with kidney disease, further analysis is warranted into the interactions with insurance. Throughout the time period we examine, many interactions were occuring in the insurance market, with universal healthcare being debated, issues with rising premiums, and the eventual passage of the Affordable Care Act. Those with kidney disease should've been relatively insulated from these impacts, given that individuals with End Stage Renal Disease are eligible for Medicare benefits regardless of age (Eggers, 2000). Future research should examine how the financial crisis impacted insurance coverage and the mechanisms by which it impacts mortality during crises. Universal coverage of kidney disease patients seems like an appropriate way to start thinking about the issue.

Changes in mortality due to influenza and pneumonia are by far one of the most significant changes as a result of the crisis. Mortality rate from respiratory diseases (such as influenza and pneumonia) has been shown to be strongly procyclical, however, research as to why is sparse. (Miller et al., 2009; Ionides et al., 2013) One justification for the mild negative effects during the crisis, but increased negative effects afterwards, is that during-crisis effects are mitigated due to the 2009 Swine Flu epidemic.

Perhaps the most interesting cause-specific mortality rate change was for suicide, where we saw significant increases in the suicide rate in a good number of our various specifications during the "post-crisis" period. This is in line with much of the literature cited above surrounding a decline in mental health and increased suicidal behavior as a result of the financial crisis (Reeves et al., 2014;

Reeves et al., 2015; Corcoran et al., 2015; De Vogli et al., 2013; De Vogli et al., 2014; DeFina & Hannon, 2015; Gemmill et. al, 2015; Nortsröm et al., 2015; Phillips and Nugent, 2014; Fowler et al., 2015).

Lastly, it's worth noting that going from specification (1) to (1') and (2) to (2') had very little impact on the point estimates for mortality rate. This leads us to conclude that trying to distinguish mortality through macroeconomic proxies compared to general crisis related phenomena is very difficult, as the two trend together very closely.

In summation, we view our primary findings as showing that there was no relation between unemployment and mortality during the Great Recession, supporting the emerging consensus in the literature that the relationship between mortality and unemployment is no longer procyclical. In terms of individual causes, pneumonia and the flu proved to highly procyclical in the majority of specifications (they decreased), and suicides increased in the aftermath of the recession. We believe both of these are in line with our findings from the literature as well.

Caveats

Reflecting on the analyses, one of the largest potential flaws that we see is with our identification strategy. The fact that our mortality findings are highly dependent on the specified time of the financial crisis suggests that our structural form model may not conclude the clearest causal estimate. Looking forward, we would have more confidence in a model that could leverage some sort of exogenous variation in crisis effects or intensity to determine these impacts on health outcomes. Additionally, we are cognizant of the fact that in multiple specifications we are imposing functional form on the relationship between unemployment and mortality. Aside from simplicity of analysis, we have no reason to believe the two variables are related in a linear fashion. Given the

above factors, individual or neighborhood level analyses may be more effective, as sensitivity to crisis definition makes us cautious about our findings.

Finally, we recognize that our findings do not show a causal relationship between the crisis and health outcomes. We would be interested in pursuing a reduced form oriented analysis to augment the structural work we have done, perhaps by leveraging similar firms with differential exposure to particularly impacted industries such as car manufacturing or real-estate, so as to derive better causal estimates. However, there was no reasonable path to pursue this route for the purposes of this paper.

Implications

Broadly, we believe our findings are in support of the majority of the literature published to date. Additionally, at the aggregate level it appears that the Great Recession in the United States does not appear to differ significantly from other crises in its relationship to the general trends of economic fluctuation on health outcomes observed during the surrounding years. Our analysis lends credence to the idea that, at an aggregate level, crises do not magnify health effects present in general economic fluctuations in any meaningful way.

Recommendations for Further Analysis

As can be seen from the literature review, there are no shortage of inquiries that tie economic conditions to health outcomes, yet relatively few are very specific to the Great Recession. We recommend a more in-depth analysis utilizing less aggregated, CDC Wonder Data, looking at mortality on a smaller level, rather than the aggregated level, as was done in this analysis. Using

individual level data would allow for more race and age specific analysis to establish better understanding of the differential effects, as preliminary studies have suggested some (Noelke & Belkfield, 2014). Additionally, with this data, the analysis would be able focus on smaller cities and counties, for example, we could compare financial hub New York City, to Hurricane Katrina impacted New Orleans, to rust-belt Ohio to gain a better understanding of how different geographic regions were impacted and how areas that specialize in different sectors suffered. We also think that using less-aggregated data would allow for cleaner causal identification strategies, such as creating synthetic controls to see how a specific area was impacted compared to what it would've looked like in absence of impacts from the financial crisis.

While mortality is an interesting and worthwhile metric of health outcomes, it is the most severe, and it may be interesting to examine health outcomes that may not be as drastic such as morbidity factors. Additionally, it may be worthwhile to examine the impacts of the financial crisis on health insurance coverage and how that impacted certain health outcomes.

In line with the suggestions above, we also would hope to explore in greater depth the weaknesses and strengths of aggregate-level analyses. If data limitations force us to continue with aggregate-level analyses of mortality rate, one path we would attempt to pursue is focusing our analysis on differentiating between housing and unemployment effects leveraging state-level foreclosure rate. Similarly, we would be interested in exploring whether aggregate-level analyses between other economic indicators and key health outcomes outside of unemployment and mortality rate support our preliminary findings. Additionally, as a sub-analysis, we would also like to ascertain the drivers behind the oft-observed procyclical nature of mortality from respiratory diseases such as the flu at the aggregate level.

Finally, as policy students, we would be interested in exploring how policies related to differential effects in different social/geographic contexts. Others have shown policies such as increased unemployment benefits have beneficial effects on mental health (O'Campo et al., 2015).

Ultimately, we conclude by determining that the relationship between health outcomes and financial crises are an important factor to consider when making regulations that could either reduce the probability or impact of a future crisis. Our analysis shows that there seems to be little effect in aggregate, but more research should be conducted in order to find causal and demographic-specific effects, so that we can craft better policy to support to most susceptible individuals in the event of future crises.

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Appendices

Appendix Table 1: Summary Statistics

Variable	Mean	Standard Deviation
Mortality Rates		
All Causes	847.73	125.96
Alzheimer's Diseases	25.03	8.59
Chronic Lower Respiratory Diseases (CLRD)	46.79	11.37
Cancer	193.52	29.62
Diabetes	24.98	10.72
Heart Disease	216.33	49.55
Influenza and Pneumonia	19.87	5.24
Kidney Disease	14.99	5.23
Stroke	49.97	11.76
Suicide	12.99	3.71
Unintentional Injury	42.50	9.80
State Characteristics		
State Unemployment Rate (%)	5.80	2.09
% Female	50.75	0.79
% White	81.71	13.78
% Black	11.81	11.42
% Other Nonwhite	6.58	10.24
% <1-Year-Old	1.32	0.16
% 1-4 Years Old	5.25	0.57
% 5-9 Years Old	6.65	0.64
% 10-14 Years Old	6.93	0.62
% 15-19 Years Old	7.19	0.52
% 20-24 Years Old	7.11	0.72
% 25-29 Years Old	6.64	0.80
% 30-34 Years Old	6.63	0.66
% 35-39 Years Old	6.88	0.77
% 40-44 Years Old	7.29	0.75
% 45-49 Years Old	7.35	0.57
% 50-54 Years Old	6.96	0.59
% 55-59 Years Old	5.99	0.83
% 60-64 Years Old	4.84	0.85
% 65-69 Years Old	3.80	0.60
% 70-74 Years Old	3.08	0.42
% 75-79 Years Old	2.51	0.39
% 80-84 Years Old	1.87	0.33
% Age 85 and older	1.70	0.40
Sample Size	**8,415**	

Note: Data cover the 1999-2013 and are for the 50 states plus the District of Columbia. Mortality rates are per 100,000 population. Data come from CDC Mortality Rates, Department of Labor Local Area Unemployment Statistics, and SEER Data.

Appendix Table 2: Regression Results for Effect of Financial Crisis on Mortality for Specifications (2) and (2')

	Model with Unemployment Control		Model without Unemployment Control	
Type of Mortality	Coefficient on "During"	Coefficient on "Post"	Coefficient on "During"	Coefficient on "Post"
All causes	-0.00211	0.00216	-0.000598	0.00284
	(0.00371)	(0.00507)	(0.00385)	(0.00515)
Alzheimer's Disease	0.00354	0.0162	0.0104	0.0193
	(0.0263)	(0.0373)	(0.0251)	(0.0376)
CLRD	-0.00472	-0.0408*	-0.00436	-0.0406*
	(0.0149)	(0.0222)	(0.0147)	(0.0220)
Cancer	0.00657	0.0216***	0.00613	0.0214**
	(0.00530)	(0.00807)	(0.00521)	(0.00807)
Diabetes	-0.00630	-0.0238	-0.00472	-0.0230
	(0.0187)	(0.0272)	(0.0183)	(0.0270)
Heart Disease	-0.00738	-0.00449	-0.00596	-0.00385
	(0.00890)	(0.0110)	(0.00884)	(0.0109)
Influenza and Pneumonia	-0.0750***	-0.186***	-0.0724***	-0.185***
	(0.0258)	(0.0431)	(0.0249)	(0.0427)
Kidney Disease	0.0242	0.0622*	0.0164	0.0586
	(0.0252)	(0.0362)	(0.0247)	(0.0361)
Stroke	0.0256	0.0446*	0.0257	0.0447*
	(0.0167)	(0.0240)	(0.0164)	(0.0239)
Suicide	0.0298	0.0699**	0.0279	0.0691**
	(0.0197)	(0.0265)	(0.0200)	(0.0267)
Unintentional Injury	0.0101	0.0224	0.0151	0.0246
	(0.0187)	(0.0286)	(0.0182)	(0.0282)
Control for Unemployment	Yes		No	
Observations per Regression	765		765	

Note: See note on Table 1 for general regression information. Reported coefficients are the indicator variables for the crisis being "active" in the specifications which is 2007-2009, inclusive, and the after the 2010 for the "post crisis" period. Each reported coefficient represents a single regression. Left panel runs specification (1); right panel runs specification (1'). Heteroskedastic robust standard errors, clustered at the state level are reported in parentheses. *** p<0.01, ** p<0.05, * p<0.1.

Appendix Table 3: Regression Results for Robustness Checks of Specification (1') with Crisis Start Times of 2006 and 2008

Type of Mortality	**Crisis begins in 2006** Coefficient indicating under influence of crisis δ in (1)	**Crisis begins in 2008** Coefficient indicating under influence of crisis δ in (1)
All causes	-0.00748*	0.0122***
	(0.00378)	(0.00354)
Alzheimer's Disease	-0.00485	0.0235
	(0.0192)	(0.0216)
CLRD	-0.0810***	0.0947***
	(0.0117)	(0.0104)
Cancer	-0.00173	-0.000705
	(0.00465)	(0.00453)
Diabetes	-0.0189	-0.0293
	(0.0237)	(0.0183)
Heart Disease	-0.0158**	0.0145**
	(0.00705)	(0.00560)
Influenza and Pneumonia	-0.124***	0.0897***
	(0.0222)	(0.0203)
Kidney Disease	-0.0379*	0.0832***
	(0.0225)	(0.0271)
Stroke	-0.0141	0.0266***
	(0.0106)	(0.00970)
Suicide	0.0261*	-0.00394
	(0.0151)	(0.0163)
Unintentional Injury	0.0361*	-0.0263*
	(0.0198)	(0.0136)
Control for Unemployment	No	No
Observations per Regression	765	765

Note: See Table 1 for general regression information. Reported coefficients are the indicator variables for the crisis being "active" using specification (1'). Timing changes with varying definitions of crisis start and end times. Heteroskedastic robust standard errors, clustered at the state level are reported in parentheses. *** p<0.01, ** p<0.05, * p<0.1.

Appendix Table 4a: Regression Results for Robustness Checks of Specification (2')

	During: 2008 Post: 2009 and after		During: 2007-2010 Post: 2011 and after	
Type of Mortality	Coefficient on "During"	Coefficient on "Post"	Coefficient on "During"	Coefficient on "Post"
All causes	0.0134***	-0.00633	-0.000895	0.0130**
	(0.00336)	(0.00445)	(0.00395)	(0.00533)
Alzheimer's Disease	0.0290	-0.0665**	0.00846	0.0115
	(0.0205)	(0.0291)	(0.0242)	(0.0282)
CLRD	0.0972***	0.0535***	0.00463	0.0284
	(0.00999)	(0.0129)	(0.0140)	(0.0178)
Cancer	-0.000712	-0.000598	0.00267	0.00254
	(0.00463)	(0.00574)	(0.00534)	(0.00748)
Diabetes	-0.0264	-0.0773***	0.00176	0.0702**
	(0.0171)	(0.0235)	(0.0180)	(0.0268)
Heart Disease	0.0150***	0.00697	-0.00634	-0.00344
	(0.00543)	(0.0112)	(0.00877)	(0.00941)
Influenza and Pneumonia	0.0943***	0.0152	-0.0459*	-0.00808
	(0.0194)	(0.0385)	(0.0230)	(0.0270)
Kidney Disease	0.0794***	0.146***	0.00129	-0.162***
	(0.0265)	(0.0415)	(0.0249)	(0.0366)
Stroke	0.0271***	0.0180	0.0214	0.0205
	(0.00963)	(0.0166)	(0.0163)	(0.0220)
Suicide	-0.00221	-0.0324	0.0186	0.0198
	(0.0162)	(0.0224)	(0.0197)	(0.0289)
Unintentional Injury	-0.0227*	-0.0853***	0.0142	0.0519*
	(0.0135)	(0.0216)	(0.0180)	(0.0282)
Control for Unemployment	No		No	
Observations per Regression	765		765	

Note: See note on Table 1 for general regression information. Reported coefficients are the indicator variables for the crisis being "active" and for the "post crisis" period using specification (2'). Timing changes with varying definitions of crisis start and end times. Heteroskedastic robust standard errors, clustered at the state level reported in parentheses. *** p<0.01, ** p<0.05, * p<0.1.

Appendix Table 4b: Regression Results for Robustness Checks of Specification (2'), continued

	During: 2006-2009 Post: 2010 and after		During: 2008-2009 Post: 2010 and after	
Type of Mortality	Coefficient on "During"	Coefficient on "Post"	Coefficient on "During"	Coefficient on "Post"
All causes	-0.00777**	-0.00372	0.0129***	0.0183***
	(0.00373)	(0.00550)	(0.00351)	(0.00475)
Alzheimer's Disease	-0.00540	0.00222	0.0249	0.0357
	(0.0194)	(0.0264)	(0.0218)	(0.0321)
CLRD	-0.0788***	-0.109***	0.0917***	0.0691***
	(0.0117)	(0.0162)	(0.0102)	(0.0147)
Cancer	-0.00277	0.0117	0.00117	0.0156**
	(0.00460)	(0.00775)	(0.00419)	(0.00594)
Diabetes	-0.0177	-0.0341	-0.0322*	-0.0543**
	(0.0238)	(0.0283)	(0.0184)	(0.0269)
Heart Disease	-0.0161**	-0.0120	0.0152**	0.0204**
	(0.00711)	(0.00865)	(0.00584)	(0.00954)
Influenza and Pneumonia	-0.118***	-0.211***	0.0782***	-0.0115
	(0.0221)	(0.0319)	(0.0207)	(0.0339)
Kidney Disease	-0.0409*	0.00136	0.0900***	0.142***
	(0.0231)	(0.0254)	(0.0274)	(0.0374)
Stroke	-0.0152	0.000623	0.0291***	0.0480***
	(0.0104)	(0.0178)	(0.00951)	(0.0169)
Suicide	0.0236	0.0588***	0.000821	0.0377
	(0.0154)	(0.0194)	(0.0167)	(0.0258)
Unintentional Injury	0.0358*	0.0405	-0.0258*	-0.0223
	(0.0198)	(0.0268)	(0.0136)	(0.0241)
Control for Unemployment	No		No	
Observations per Regression	765		765	

Note: See note on Table 1 for general regression information. Reported coefficients are the indicator variables for the crisis being "active" and for the "post crisis" period using specification (2'). Timing changes with varying definitions of crisis start and end times. Heteroskedastic robust standard errors, clustered at the state level reported in parentheses. *** p<0.01, ** p<0.05, * p<0.1.