Stroke-Attributable Death Among Older Persons During the Great Recession

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Highlights

- During the Great Recession, stroke accounted for 16.6% higher than expected fraction of deaths in California among older white men but not among other older Californians.

- However, overall mortality did not increase among white men, and therefore we infer that the Great Recession did not accelerate mechanisms that led to death among older Californians, but may have shifted more than expected white men into mechanisms that led to death by stroke.

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Abstract
This study assesses how the Great Recession influenced the distribution of mortality among older Californians. Specifically, this study tests the hypothesis, suggested by individual risk factor research, that the ratio of stroke, the fourth leading cause of death in the US, to other deaths would increase in the Recession. We applied interrupted time series methods to data from the California Department of Public Health Vital Statistics Section (January 2000 – November 2007) to determine whether the ratio of stroke deaths to other deaths differed from values expected from history. Our findings indicate that among white men, the odds of stroke-attributable death increased between May 2008 and December 2010 by 16.6% over the expected levels from the preceding 95 months starting January 2000 and ending November 2007. Our results did not show an increase in odds of stroke death among white women, black men and women, or Hispanic men and women. We infer the Recession may have affected social, biologic, and behavioral risk factors and thereby altered the life histories of older white men in ways that shifted mortality risk toward stroke.

JEL Classification

E20; I10; I12

Keywords. Stroke; Mortality; Great Recession

1. Introduction

The Great Recession, which officially extended from December 2007 to June 2009, (National Bureau of Economic Research, 2014), affected virtually all sectors of the U.S. economy and induced the deepest downturn in employment since the end of World War II (Goodman and Mance, 2011). Despite the intuition that such a circumstance would adversely affect public health, studies of the relationship between the Great Recession and mortality in the U.S. and elsewhere have produced inconsistent findings (Stuckler et al., 2014). Indeed, this inconsistency characterizes the larger research studying the association between mortality and economic contraction (Catalano et al., 2011). Some research has found that mortality from several common causes falls when labor markets weakens (e.g., (Gerdtham and Ruhm, 2006), while other research reports deaths rising during economic contraction (e.g., (Stuckler et al., 2009).

Reviews (Catalano et al., 2011; Stuckler et al., 2014) of the literature connecting recessions to mortality have noted that the disparate findings likely arise from heterogeneity in the spatial and temporal mix of mechanisms that set older persons, who disproportionately contribute to mortality, on trajectories to death. While some of these mechanisms may accelerate in response to economic circumstances, others may slow. This possibility suggests a research strategy that focuses, at least at the outset, on causes of mortality for which recession would likely "harvest" deaths among older persons whose trajectories would otherwise have spread their deaths more evenly over time or across causes. For reasons described below, we suspect that economic contraction may accelerate death by stroke and thereby "harvest" deaths from other causes or from the future. We test this suspicion using stroke deaths in California before and during the Great Recession.

Although each individual has a unique history of genetic endowment as well as of social experiences and environmental exposures, commonalities among these histories allow epidemiologists and demographers to identify groups that differ significantly in morbidity and longevity (Olshansky et al., 2005; Vaupel, 2010; Yoon et al., 2014). The distribution of older individuals across these groups, in turn, predicts the frequency and causes of death among the elderly (Galea, 2007; Halfon and Hochstein, 2002; Vasunilashorn and Crimmins, 2009; Worthman and Kuzara, 2005). The Great Recession may have changed this distribution in the United States and elsewhere by affecting, among other factors, income, time use, access to medical care, exposure to work-related hazards, as well as by inducing stress and anxiety.

The causes of death suspected to increase during bad economic times include those responsive to stressful experiences. The biological precursors of stroke, for example, likely develop over the life course but the timing of an ischemic event appears affected by "triggers" including stressful experiences (Guiraud et al., 2013). This circumstance has, in fact, led to the suspicion that job loss may trigger stroke. Research supporting this hypothesis shows that older workers who suffer layoff exhibit a twofold increase in stroke compared to similar workers who remain employed (Gallo et al., 2004). The association presumably arises because late-career job loss decreases income and savings and severs social interactions thereby inducing significant anxiety and psychological stress while reducing social and material support (Gallo et al., 2004). Non-transferable firm or industry-specific skills can, moreover, lead to poor prospects of reemployment with equivalent wages and benefits (Brand et al., 2008).

The above circumstances lead us to hypothesize that death among older persons occurred more frequently than expected by stroke than by other causes during the Great Recession. If we find support for our hypothesis, we then ask whether all-cause incidence of death differed from

expected among older persons during the Recession. The argument leading to our hypothesis implies, as noted above, that stroke "harvested" deaths from other causes, or from the future, during the Recession. If so, we would find no change or a decline in the overall incidence of death.

We test our hypothesis among 2,268,288 deaths to persons over 50 years old in California for the 132 months starting January 2000 and ending December 2010. We test the hypothesis separately among men and women of non-Hispanic white (hereafter white), Hispanic, and non-Hispanic black (hereafter black) race/ethnicity, given prior research indicating that these groups have significantly different risks for stroke mortality (Ayala et al., 2001).

2. Methods

2.1 Data

The California Department of Public Health, Center for Health Statistics & Informatics, Vital Statistics Section provided the data for this test (California Department of Public Health, 2012). Each year, the Vital Statistics section produces a file that includes data from all the death certificates registered in California as well as death certificates for California residents who die in other states. For our study period, causes of death were classified according to the International Classification of Diseases, Tenth Revision (ICD-10) (World Health Organization, 1993). We considered the following ICD-10 codes as stroke deaths: intracerebral hemorrhages (I61.0 - I61.9), cerebral infarctions (I63.0 - I63.9), and stroke not specified as hemorrhage or infarction (I64.0 -I64.9). All other causes of death, including unknown or missing causes, were coded as non-stroke mortality.

2.2 Analyses

Our test follows the logic of Weiner-Granger causation (Bressler and Seth, 2011) in that it asks whether the ratio of stroke deaths to other deaths (i.e., odds a death will result from stroke) among older Californians differed during the Great Recession from values predicted from history. Consistent with much literature in the physical, biological, and behavioral sciences, we define "predicted from history" as statistically expected values estimated from models that describe autocorrelation in a time series. Autocorrelation intuitively refers to secular trends or seasonal cycles but also includes the tendency of a series to remain elevated or depressed, or to oscillate, after high or low values. We modeled autocorrelation in the ratio of stroke deaths to other deaths with methods devised by Box and Jenkins (Box et al., 2008). These well-developed methods appear widely accepted in the epidemiologic literature (Catalano and Serxner, 1987; Zeger et al., 2006). Box-Jenkins methods mathematically express various filters through which time series without known patterns can pass. Each filter, or combination of filters, imposes a unique pattern. The Box-Jenkins approach uses an iterative model-building process by which the researcher infers the filter that imposed the observed pattern. The error terms of these models (i.e., the differences between the values predicted by the inferred model and the observed series) express the "unexpectedness" of observed values. The general form of a Box-Jenkins model is as follows.

$$\nabla_d (S_t / O_t)^e = c + \frac{\left(1 - \theta B\right)\left(1 - \theta_n B^q\right)}{\left(1 - \phi B\right)\left(1 - \phi_n B^p\right)} a_t$$

∇_d is the "difference operator" that indicates a series exhibits secular trends or cycles and requires differencing (i.e., value at time t-d subtracted from that at time t) to render the series stationary in its mean. S_t is the number of stroke-related deaths in California at month t. O_t is the

number of other deaths in California at month t. c is a constant. ϕ is an autoregressive parameter that measures a series' tendency to remain elevated or depressed after a perturbation. θ is a moving average parameter that measures the tendency of perturbations to be present for more than one period. B is the "backshift operator" or value of the variable it conditions at month t – q or at t – p. a_t is the error term at month t.

Our analyses proceeded as follows. First, we used Box-Jenkins methods to arrive at the best fitting models for the natural logarithm of the ratio of stroke to other deaths for Hispanic, black, and white males and females aged more than 50 years and residing in California. We transformed the ratios to natural logarithms to reduce any bias introduced by variation in variability over time and to allow us to express our results in the familiar effect on odds metric. We derived separate models for each group due, as noted above, to their widely noted differences in stroke mortality. We estimated these models for the 95 months beginning January 2000 (1st month) and ending November 2007 (95th month), prior to the beginning of the Great Recession. Second, we applied the models and model parameters estimated in step 1 to the entire 132 months of data. These models estimate a value for each month from all the observed values of preceding months using the parameters from the pre-recession months. The error terms of these estimations (i.e, the difference between the estimated and observed values) express the degree to which the observed odds of death attributable to stroke over the entire test period differed from the values expected from the circumstances that produced the values observed in the first 95 (i.e., pre-recessionary) months of the period. Third, we used the approach of Alwan and Roberts (Alwan and Roberts, 1988) to detect shifts above the 99.5 percent (2-tailed test) confidence interval of the error terms from the model estimated in step 2. By shift we mean a series of values that rise above the confidence interval and persist, on average, at high levels

through the end of our data series. If the Great Recession increased the odds of stroke-attributable death, the Alwan and Roberts methods would find error terms persistently above their 99.5 percent confidence interval among the 96th through 132nd months of our data (i.e. December 2007 through December 2010).

We used three steps to answer the question of whether all-cause incidence of death differed from expected in any group for which we found a shift in the odds of death attributable to stroke. The first two steps mimicked those described above for our hypothesis test. In step 1 we derived the Box-Jenkins model and parameters from the months before the shift in the odds of death attributable to stroke appeared. In step 2 we applied the model and its parameters to all 132 months of the all cause death time series. In step 3, we regressed the error terms of the model estimated in step 2 on a binary variable scored 1 for all months after the level shift appeared for the test group and 0 otherwise. The coefficient for the binary variable would differ significantly (i.e., $p < .05$; 2 tailed test) from 0 if the incidence of all-cause death rose or fell below expected levels during the same months that the odds of stroke attributable death rose or fell below values expected from pre-recession autocorrelation.

3. Results

Table 1 shows the best fitting ARIMA models, the product of step 1 above, for the first 95 months (i.e., pre-recession months) for the six test groups. As suggested by the difference operators shown in Table 1, the ratio of stroke deaths to other causes of death trended downward for most of the test period only for white men and women. Table 1 also shows that the ratios for all groups except black men exhibited autocorrelation such that values in month t-1 affected those in month t. Ratios for black and Hispanic men and women all exhibited delayed

autocorrelation in that values in month t predicted those at month t+7, t+8, or t+11. None of the ratios, however, exhibited strong seasonality.

Table 1

Box-Jenkins Models for the Log Odds of Stroke-attributable Death in the Indicated Groups for 95 Pre-recession Months (i.e, January 2000 through November 2007). All Coefficients Exceed Twice their Standard Errors.

Group	Best Fitting Box-Jenkins Model
Men	
Black	$(S_t/O_t)^e = -3.1185 + \frac{1}{\left(1-0.3288B^8\right)}a_t$
Hispanic	$(S_t/O_t)^e = -3.1144 + \frac{1}{\left(1-0.2861B\right)\left(1-0.3291B^{11}\right)}a_t$
White	$\nabla(S_t/O_t)^e = -0.0058 + \left(1-0.8333B\right)a_t$
Women	
Black	$(S_t/O_t)^e = -2.8068 + \frac{1}{\left(1-0.3538B\right)\left(1-0.2851B^7\right)}a_t$
Hispanic	$(S_t/O_t)^e = -2.8340 + \frac{\left(1+0.2658B^8\right)}{\left(1-0.4142B\right)}a_t$
White	$\nabla(S_t/O_t)^e = \frac{1}{\left(1+0.5300B\right)}a_t$

The Alwan and Roberts outlier detection analysis, step 3 above, found only one group for which monthly error terms after November 2007 persisted outside their 99.5 percent confidence interval. The error terms for older white men shifted above the confidence interval in May 2008 (i.e., the 101st month in our series) and persisted above expected levels through 2010. The downward trend in the odds of stroke-attributable deaths, and the post-April 2008 exceptions to it, can be seen in Figure 1, which shows the expected and observed values for our test months.

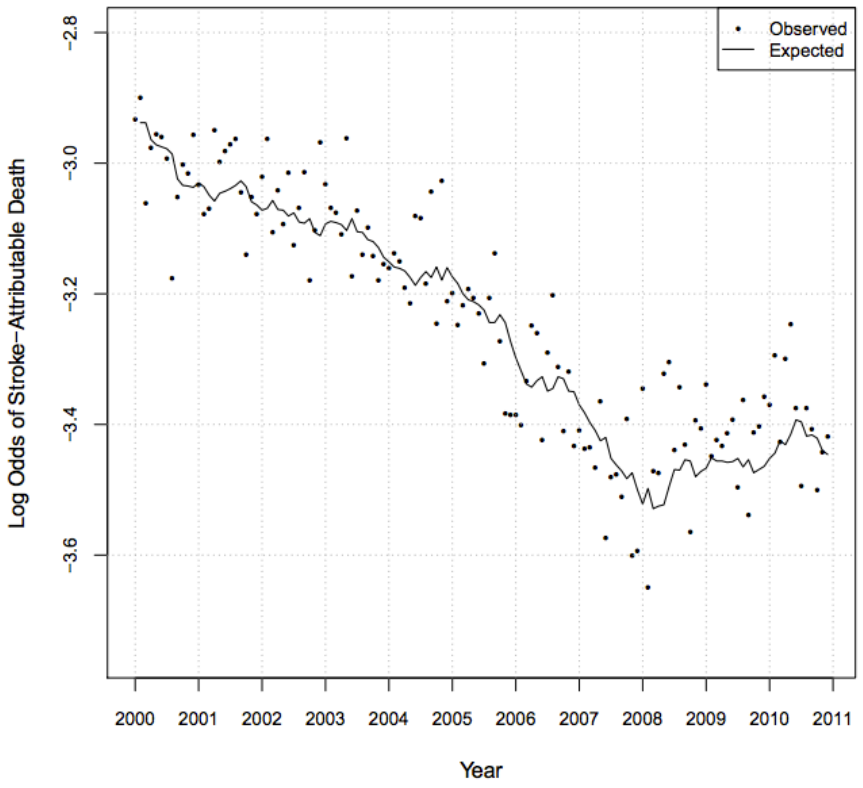

Figure 1. Observed and Expected Log Odds of Stroke-Attributable Death Among White Men for January 2000 through December 2010. Model is Based on 95 Pre-Recession Months Ending November 2007.

Figure 2 depicts the level shift detected by the Alwan and Roberts method for older white men. The points show the error terms from applying the best-fitting Box-Jenkins model for pre-recession months to all months. The lines shows the pre-May and post-April 2008 means of the residuals. The mean of the pre-May values (i.e., 0) would be the mean of post-April values if the Great Recession had no effect on the odds that a death among older white men arose from stroke as opposed to other causes. The mean (i.e., 0.0492) of the post-April values was, however, significantly ($p < .001$; 2- tailed test) greater than 0.

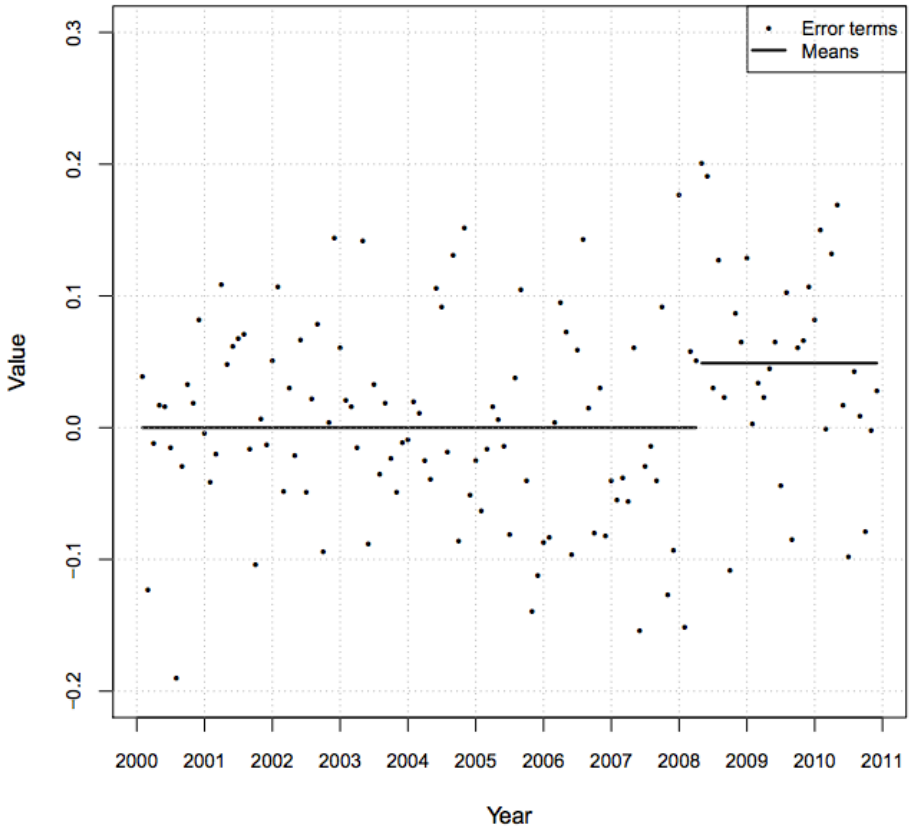

Figure 2. Means of pre-May and post April-2008 error terms from applying pre-Great Recession ARIMA model for older white men to entire test period.

We next answered the question of whether all cause incidence of death increased among white men after April 2008. To summarize the approach described in the Analysis section, we estimated a Box-Jenkins model and parameters from the months before the level shift appeared (i.e., May 2008). Next we applied the model and its parameters to all 132 months of the data. We then regressed the error terms of the model on a binary variable scored 1 for all months after the level shift appeared and 0 otherwise. The coefficient for the binary variable did not differ significantly (i.e., $p < .05$; 2 tailed test) from 0. We, therefore, infer that the Great Recession "harvested" stroke deaths from other causes of death among white men.

We converted our findings for white men into a summary statement by using the familiar effect-on-odds metric. We first created a binary variable scored 0 for the first 100 months (i.e., before May 2008) and 1 for the remaining 32. We then added this variable to the model shown in Table 1 for white men and estimated the equation again for all 132 months. The coefficient (i.e., 0.1540) for the binary variable was 3.51 times its standard error (i.e., .0439). Taking the antilog of the coefficient suggests that, among white men, the odds of stroke-attributable death increased between May 2008 and December 2010 by 16.6% over the level expected from history.

4. Discussion

Our findings show that stroke became a more frequent cause of death among older white men in California during the Great Recession than during better times. More specifically, we found a 16.6% increase over trend in the likelihood of stroke as a cause of death among these men during the Recession, starting in May 2008. Although the Recession officially began in late 2007, no change in stroke mortality is observed until later given that the Recession likely did not affect the lives of Californians until after national employment stopped growing (i.e., the

indicator which defines a recession). Indeed, disemployment, as opposed to unemployment, in California and elsewhere in the U.S. accelerated dramatically during the second quarter in 2008. We found no shift for women or for Hispanic or black men. The association with white men has implications for the overall mortality "landscape" in that stroke deaths among them accounted for 27% of all stroke deaths during our test period.

Many proximal mechanisms likely account for the association between the distal phenomenon of "recession" and stroke among older white men (Catalano and Dooley, 1983). Older white men may, for example, feel more connected to the equities or real estate markets than do other older men or women. They may have lost larger fractions of their income during the Recession than did other older persons. They may have lost more symbols of social status conferred by income.

Because not all segments of the economy felt the effects of the employment downturn equally (Hoynes et al., 2012), labor market attachment and earnings may also partly explain our findings. Research on job displacement and mortality, for example, indicates that mortality hazard is greater among those with higher earnings losses (Sullivan and von Wachter, 2009), and that stroke mortality hazard is significantly higher among those with higher income (Gallo et al., 2004). With white men—especially those ages 45 to 64—earning more on average compared to white women, black men and women, and Hispanic men and women (Bureau of Labor Statistics, 2011), the Great Recession may have disproportionately affected their stroke mortality risk.

We could have specified our test with multiple economic indicators such as stock prices, mortgage foreclosures, dis-employment rate, or unemployment rate and found which independent variables shared unique variance with death attributable to stroke over the "normal" range of their variation. We could have, alternatively, factor-analyzed a battery of California

economic indicators and tested whether variation in a molar, subsuming "economy" construct predicted shifts in the distribution of death across causes. These exercises would have missed, however, the point of our inquiry—to assess whether the Great Recession, as a distal societal force, shifted older Californians into mechanisms likely to end with death by stroke.

Would our results describe places other than California during the Great Recession? We know of no reason to believe older Californians differ from other older Americans, although only further research into the effects of the Great Recession will answer the question. The circumstances referred to collectively as the Great Recession, on the other hand, have been, and will hopefully remain, rare. We do not, therefore, expect to see the associations we found in our test period during more "normal" economic times.

5. Conclusions

Prior research exploring whether the Great Recession influenced the incidence of mortality has produced inconsistent results. Our study expands this literature by testing whether the Great Recession changed the distribution of deaths among older persons who, demographically speaking, exhibit the highest risk of mortality. Our results suggest that stroke mortality ratios among older white males significantly increased during the Great Recession. We infer these findings result from social, biologic and behavioral risk factors that altered life histories and, in turn, the distribution of the population across risk groups. Our analysis demonstrates how macro events such as economic contraction can alter and shape population health and aids in our understanding of the connection between life histories and health. We hope that future research examining the health effects of the Great Recession, or any other major ambient stressor, includes analysis of shifting risks and competing causes of death.

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