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ABSTRACT

Using data from multiple sources, over the 1976-2009 period, I show that total mortality has shifted over time from strongly procyclical to being essentially unrelated to macroeconomic conditions. The relationship also shows some instability over time and is likely to be poorly measured when using short (less than 15 or 20 year) analysis periods. The secular change in the association between macroeconomic conditions and overall mortality primarily reflects trends in effects for specific causes of death, rather than changes in the composition of total mortality across causes. Deaths due to cardiovascular disease and transport accidents continue to be procyclical (although possibly less so than in the past), whereas strong countercyclical patterns of cancer fatalities and some external sources of death (particularly those due to accidental poisoning) have emerged over time. The changing effect of macroeconomic conditions on cancer deaths may partially reflect the increasing protective influence of financial resources, perhaps because these can be used to obtain sophisticated (and expensive) treatments that have become available in recent years. That observed for accidental poisoning probably has occurred because declines in mental health during economic downturns are increasingly associated with the use of prescribed or illicitly obtained medications that carry risks of fatal overdoses.

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Although health is usually thought to worsen when the economy weakens, substantial recent research suggests that mortality actually *declines* during such periods. Following Ruhm (2000), most of these studies utilize information for multiple locations and points, and use panel data techniques to control for many confounding factors, including time-invariant location-specific determinants and characteristics that vary over time in a uniform manner across locations.[1] Using data from a variety of countries and time periods, these investigations provide strong evidence of a procyclical fluctuation in total mortality and several specific causes of death.[2] In Ruhm's (2000) study of U.S. data for 1972-1991, a one percentage point increase in the unemployment rate was estimated to decrease total mortality by 0.5% an deaths due to motor vehicle accidents and cardiovascular disease (CVD) by 3.0% and 0.5%, with reductions also predicted for mortality from influenza/pneumonia, liver disease, non-vehicle accidents and homicides. By contrast, there was no effect for cancer mortality and suicides were estimated to rise by 1.3%.[3] Using similar empirical methods, the procyclicality of total mortality has been confirmed using data for Germany (Neumayer, 2004), Spain (Tapia Granados, 2005), France (Buchmueller, et al., 2007), Mexico (Gonzalez & Quast, 2011), Canada (Ariizumi & Schirle, 2012), OECD countries (Gerdtham & Ruhm, 2006), and Pacific-Asian nations (Lin, 2009).[4]

[1] By contrast, earlier studies (e.g. Brenner, 1971, 1979) typically used time series data for a single geographic location. This research has been criticized on methodological grounds (e.g. Kasl, 1979; Gravelle et al., 1981) and suffers from the fundamental problem that any lengthy time-series may contain omitted confounding factors that are spuriously correlated with health. Ruhm (2012) provides a detailed discussion of these issues.
[2] Mortality rates are the most common proxy for health because they represent the most severe negative health outcome, are objectively and relatively well measured, and since diagnosis generally does not depend on access to the medical system (in contrast to many morbidities). However, changes in non-life-threatening health conditions will not be fully accounted for. Due to limited data availability, few analyses examine how macroeconomic conditions affect morbidity. Exceptions include Ruhm (2003) and Charles & DeCicca (2008).
[3] This suggests that mental health and physical health may move in the opposite directions, as discussed below.
[4] Economou et al. (2008) find that total mortality is negatively but insignificantly related to unemployment rates for 13 EU countries but that the unemployment coefficient reverses sign when controlling health behaviors (smoking, drinking, calorie consumption) and other potential mechanisms (like pollution rates in the model).

Motor vehicle and CVD fatalities are also procyclical in almost all studies but with more variation in mortality from other causes.[5]

However, some investigations incorporating more current data raise the possibility that mortality has become less procyclical or even countercyclical in recent years. Using empirical methods and data similar to those in Ruhm (2000), Stevens et al. (2011) find that a one percentage point increase in the state unemployment rate was associated with a 0.40% reduction in total mortality from 1978-1991, but a smaller 0.19% decrease when extending the analysis through 2006.[6] McInerney & Mellor (2012) estimate that a one-point rise in joblessness lowered the mortality rates of persons aged 65 and over by 0.27% during 1976-1991, but *increased* them by 0.49% from 1994 to 2008. Svensson (2007) uncovers a positive relationship between Swedish unemployment rates and heart attack deaths from 1987 to 2003.

Changes in health behaviors provide a potential mechanism for the mortality response. Consistent with this, reductions in drinking, obesity, smoking and physical inactivity during bad economic times have been demonstrated by Ruhm & Black (2002), Ruhm (2005), Gruber & Frakes (2006), Freeman (1999) and Xu (2013), among others, while Edwards (2011) shows that individuals spend more time socializing and caring for relatives during such periods. However, research using recent data again raises questions about the strength and direction of these behavioral changes. For instance, Charles & DiCicca (2008) indicate the male obesity is countercyclical (females are not examined), Arkes (2009) obtains a similar result for teenage girls (but not boys), and Arkes (2007) shows that teenage drug use increases in bad times. Dávlos

[5] Stuckler et al (2009) obtain evidence from 26 EU countries of positive, negative and neutral relationships between unemployment rates and suicides, deaths from transport accidents, and total mortality; however, the statistical methods focus on rates of changes in mortality and unemployment, so that the results difficult to compare with other related research. It is worth noting that analyses undertaken as early as the 1920s have uncovered positive relationships between economic activity, total mortality and several specific causes of death (Ogburn & Thomas, 1922; Thomas, 1927; Eyer, 1977), as have some recent analyses using different methods (e.g. Fishback et al., 2007;Tapia Granados & Diez Roux, 2009).

[6] The estimated reduction rises to 0.33% over the 1978-2006 period when using age-adjusted mortality rates.

et al. (2012) uncover a countercyclical pattern for some types of alcohol abuse and dependence, while Colman & Dave (2011) suggest that increased leisure-time exercise during periods of economic weakness is more than offset by reductions in work-related physical exertion. Such findings are provocative although, as shown below, they should be viewed with some skepticism because the analysis periods are too short (eight years or less) to provide definitive results.

Using U.S. data over the 1976-2009 period, the present analysis examines whether the relationship between macroeconomic conditions and mortality has changed over time. An effort is made to identify potential mechanisms for the observed secular trends and to maximize comparability with previous related work by using empirical methods that conform closely to that research.[7] Four primary results emerge. First, total mortality has shifted from being strongly procyclical at the beginning of the analysis period to being essentially unrelated to macroeconomic conditions at the end of it. The evidence from prior research that deaths decline when the economy weakens reflects the inclusion of early sample years, when this was the case. Also, estimates obtained using relatively short (e.g. less than 15 year) analysis periods show considerable instability and so probably should be viewed as unreliable. Second, the overall trend masks considerable heterogeneity across specific sources of mortality. Deaths due to cardiovascular disease and transport accidents continue to be strongly procyclical (although possibly less so than in the past), whereas countercyclical patterns of cancer and some external sources of death (particularly accidental poisonings) have emerged over time. Third, secular changes in the relationship between macroeconomic conditions and overall mortality primarily reflects these trends in effects for specific causes of death, rather than changes in the composition of deaths across causes. Fourth, the emerging countercyclicality of cancer mortality may partially

[7] One exception is the use of an uncommonly detailed set of age controls, included since age and mortality are so closely related.

reflect the increasing importance of financial resources used to purchase sophisticated (and expensive) treatments that have become available in recent years, while that for accidental poisoning may be due to an increased association between mental health problems and the use of medications presenting risks of death classified as being due to poisoning.

1. Research Design

To maximize comparability with prior related research, this analysis uses variations of previously employed panel data methods (e.g. by Ruhm, 2000) to analyze the relationship between macroeconomic conditions and mortality rates. The basic estimating equation is:

$$ln(M_{jt}) = \alpha_j + X_{jt}\beta + U_{jt}\gamma + \lambda_t + \varepsilon_{jt}, \quad (1)$$

where M_{jt} is the mortality rate in state j at time t, U is the average annual state unemployment rate, X is a vector of covariates, α is a state fixed-effect, λ a general time effect, ε is the error term, and $\hat{\gamma}$ provides the estimated macroeconomic effect of key interest.

The year effects (λ_t) hold constant determinants of death that vary uniformly across locations over time (e.g. advances in widely used medical technologies or behavioral norms); the location fixed-effects (α_j) account for those that differ across states but are time-invariant (such as persistent lifestyle disparities between residents of Nevada and Utah); and the impact of the macroeconomy is identified from within-location variations in mortality rates relative to changes in other states.[8] The supplementary characteristics include the shares of the state population who are female, nonwhite, Hispanic and in 7 age groups (<1, 1-19, 45-54, 55-64, 65-74, 75-84 and ≥85 years old). Since these do not necessarily control for all time-varying determinants of death, most models reported below also include a vector of state-specific time trends. State-year

[8] The impact of national business cycles is absorbed by the time effects, so that discussions of macroeconomic effects refer to changes within locations rather than at the national level.

population weights are also generally incorporated, since the influence of residents of small states on the average treatment effect would be overstated with unweighted data. However, I also show how the main results are affected by these choices. In addition, I provide a preliminary examination of potential mediating factors, like home prices and per capita incomes. The 1976-2009 analysis period reflects the availability of consistent data on state unemployment and mortality rates.[9]

This analysis investigates whether the effects of macroeconomic conditions on mortality have changed over time. One way of doing so involves examining whether the predicted effects differ across sub-periods. However, as shown below, such estimates are quite sensitive to the precise choice of starting or ending years. For this reason, two alternative strategies are employed. The first specifies analysis periods of fixed duration and then sequentially estimates models for *all* alternative sample windows permitted by the data. For instance, using 20-year windows, results can be obtained for 15 periods ranging from 1976-1995 to 1990-2009. Second, the models will be estimated over the entire (34-year) period but with the addition of an interaction between the unemployment and a time trend according to:

$$ln(M_{jt}) = \alpha_j + X_{jt}\beta + U_{jt}\gamma + U_{jt}\times T_t\delta + \lambda_t + \varepsilon_{jt}, \quad (2)$$

where T is a linear trend taking the value of zero in the first sample year (generally 1976) and one in the last one (usually 2009).[10] The macroeconomic effect can then be estimated as $\hat{\gamma}$ in 1976 and $\hat{\gamma} + \hat{\delta}$ in 2009, with the p-value on $\hat{\delta}$ indicating whether the relationship has changed significantly over time.

[9] Unemployment rates are used to *proxy* macroeconomic conditions; however, a procyclical variations in mortality does not imply that the loss of a job improves health. To the contrary, Sullivan & von Wachter (2009) show that job loss is associated with increases in individual mortality rates.
[10] Thus, when using data from 1976 to 2009, $T_t = (t - 1976)/33$.

The two methods have complementary strengths (and weaknesses). Estimates based on equation (2) provide a single easily comprehensible summary of secular changes in the macroeconomic effects but are restrictive in their assumption of a linear trend. Those obtained using the alternative sample windows do not make assumptions about the parametric form of the trend but are harder to summarize and may be sensitive to the length of the analysis window.

A portion of the trend in macroeconomic effects on overall mortality rates could reflect secular changes in the share of deaths accounted for by specific age groups or causes. This possibility will be examined using a variation of the decomposition method developed by Oaxaca (1973) and Blinder (1973). To do so, mortality shares from source k in period τ ($\pi_{k\tau}$) will be calculated for the first and last five years of the data (1976-1980 and 2005-2009), respectively, with τ equal to 1 and 2 during these periods. Using estimates from equation (2), predicted unemployment coefficients for total mortality equations will be calculated for the midpoints of these two periods (1978 and 2007) as $\gamma_1 = \hat{\gamma} + \frac{2}{33}\hat{\delta}$ and $\gamma_2 = \hat{\gamma} + \frac{31}{33}\hat{\delta}$. Corresponding estimates for deaths from specific source k will be denoted by γ_{k1} and γ_{k2}.

Changes in the number of deaths, at time τ, predicted by a one-percentage point increase in the unemployment rate are:

$$\Delta D_\tau = (e^{\gamma_\tau} - 1)D_\tau \approx \gamma_\tau D_\tau \tag{3}$$

where D_τ is the total number of deaths. The approximation on the right-hand-side of (3) is almost exactly correct since estimated values of γ are near zero and, for ease of exposition, it will be treated as being precisely accurate below.[11] The number of deaths can be written as:

$$D_\tau = M_\tau P_\tau \tag{4}$$

[11] For instance, for the coefficient estimate γ = -0.02, the approximation implies that a one percentage point reduction in unemployment reduces the predicted mortality rate by 2.00%, whereas the actual decrease is 1.98%.

for M_τ the total mortality rate and P_τ the population at time τ. Adding *k* subscripts to (3) and (4) indicates corresponding relationships for specific sources of mortality.

The secular change in the predicted impact of a one-point rise on unemployment on the number of deaths is:

$$\Delta D_2 - \Delta D_1 = \gamma_2 D_2 - \gamma_1 D_1 \tag{5}$$

or

$$\Delta D_2 - \Delta D_1 = (\gamma_2 - \gamma_1)D_2 + \gamma_1(D_2 - D_1) \tag{5'}$$

The first term on the right-hand-side of (5') shows the change over time in the effect of macroeconomic conditions on deaths, which is the focus of this analysis. The second-term indicates that the total effect also depends on overall number of deaths that, in turn, vary with population and baseline mortality rates,[12] which are not the focus of interest here and so will be ignored. Evaluating the number of deaths at the average value for the later (2005-2009) period:

$$\Delta D_2 - \Delta D_1 = (\gamma_2 - \gamma_1)D_2.^{13} \tag{5''}$$

Since fatality rates are approximately linearly related to unemployment, as shown in (3), the impact on total mortality is a weighted average of the effects on specific sources of death:

$$\gamma_\tau = \textstyle\sum_k \gamma_{k\tau}\pi_{k\tau} \qquad \text{where} \quad \textstyle\sum_k \pi_{k\tau} = 1, \tag{6}$$

and π_k indicates the share of deaths from cause *k*. Substituting (6) into (5'') gives

$$\Delta D_2 - \Delta D_1 = (\boldsymbol{\gamma}_2\boldsymbol{\pi}_2 - \boldsymbol{\gamma}_1\boldsymbol{\pi}_1)D_2, \tag{7}$$

where: $\boldsymbol{\gamma}_\tau\boldsymbol{\pi}_\tau = \sum_k \gamma_{k\tau}\pi_{k\tau}$. Equation (7) can then be decomposed as:

$$\Delta D_2 - \Delta D_1 = [(\boldsymbol{\pi}_2 - \boldsymbol{\pi}_1)\boldsymbol{\gamma}_1 + (\boldsymbol{\gamma}_2 - \boldsymbol{\gamma}_1)\boldsymbol{\pi}_2]D_2. \tag{8}$$

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[12] Since $D_\tau = M_\tau P_\tau$, $D_2 - D_2 = M_2 P_2 - M_1 P_1 = M_2 (P_2 - P_1) + (M_2 - M_1)P_1$.
[13] Alternatively, 5'') could be evaluated using using earlier period (1976-1980) death rates, since 5') can be rewritten as: $\Delta D_2 - \Delta D_1 = (\gamma_2 - \gamma_1)D_1 - \gamma_2 (D_2 - D_1)$.

The first-term on the right-hand-side of (8) shows the portion of the change in the macroeconomic effect that is due to shifts in the composition of deaths across alternative sources. The second term indicates the change resulting from variation over time in the coefficients on the unemployment rate.[14]

2. Data and Descriptive Statistics

Primary data sources for this project include location-specific unemployment rates from U.S. Department of Labor's *Local Area Unemployment Statistics (LAUS) Database* and mortality rates and location-specific demographic characteristics from the Center for Disease Control and Preventions' *Compressed Mortality Files (CMF)*. These two data sources, as well as some issues for their use in the analysis, are described below. Data are used from a variety of additional sources, detailed later, are used when exploring potential mediating factors.

Annual average state unemployment rates are the main proxies for macroeconomic conditions. These data come from the *LAUS* database (www.bls.gov/lau/lauov.htm), a Federal-State cooperative effort providing monthly estimates of total employment and unemployment rates for approximately 7,300 areas including: census regions and divisions, states, metropolitan statistical areas, counties, and some cities.[15] Concepts and definitions underlying the *LAUS* data come from the Current Population Survey.

[14] The alternative decomposition: $\Delta D_2 - \Delta D_1 = [(\boldsymbol{\pi}_2 - \boldsymbol{\pi}_1)\boldsymbol{\gamma}_2 + (\boldsymbol{\gamma}_2 - \boldsymbol{\gamma}_1)\boldsymbol{\pi}_1]D_2$ uses 2005-2009 coefficients as the base. Although generally similar results are obtained, this decomposition seems less appropriate for an analysis of why the macroeconomic response has changed from what it was in an earlier time period.

[15] Some recent studies of macroeconomic patterns of health behaviors have analyzed county-level or MSA data (e.g. Charles & DeCicca, 2008; An & Liu, 2012). This has potential advantages (e.g. examining smaller regional economies) and disadvantages (e.g. greater measurement error). For this investigation, the major disadvantage is that a consistent data series of county unemployment rates only begins in 1990 and the Department of Labor cautions against using county level data prior to that time. Preliminary analysis revealed similar results using state and county data for the 1990-2009 period.

The *CMF* (www.cdc.gov/nchs/data_access/cmf.htm) contain information for every death of a U.S. resident including: state and county of residence, year of death, race and sex, Hispanic origin (after 1998), age group (16 categories), underlying cause of death (ICD codes and CDC recodes). The number of records is reduced by aggregating those with identical values for all variables and adding a count of the number of such records. The *CMF* also contains population estimates for state and county resident populations, as well as for subsamples stratified by race, sex, Hispanic origin, and 13 age groups. Data prior to 1988 are publically available while those from 1989 on require special agreement with the CDC, which was obtained for this research.

The *CMF* mortality and population data were used to create the main analysis dependent variables. In addition to total annual mortality rates, these include sex-specific death rates and fatality rates for six age groups (<45, 45-54, 55-64, 65-74, 75-84 and ≥85 year olds) and four major causes: cardiovascular disease (CVD), malignant neoplasms (cancer), other diseases, and external (non-disease) causes.[16] A variety of more specific causes or age- by cause-specific rates were also examined, as described below. The *CMF* was additionally used to construct independent variables for the sex, age and race structure of the state population. Specifically, the econometric models include controls for the share of the state population who are female, nonwhite, Hispanic, and aged <1, 1-19, 45-54, 55-64, 65-74, 75-84 and ≥85 years old.[17]

The analysis of cause-specific mortality introduced complications. From 1976-1978, cause of death was categorized using the 8th revision of the International Classification of Diseases (ICD-8 codes). ICD-9 codes were used between 1979 and 1998, and since 1999, mortality classifications have been based on ICD-10 categories. Crosswalks have been

[16] I examined other age-specific death rates, including infant mortality rates, in preliminary analysis, but focus on these age groupings since the large majority of deaths (and changes in deaths) occur to those who are relatively old.
[17] Thus, the reference group is the share of 20-44 year old non-Hispanic white males. Identification of the Hispanic population is only provided in the *CMF* data beginning in 1998. Therefore, Hispanic population shares were calculated using the *Surveillance Epidemiology and End Results (SEER)* Program of the National Cancer Institute (http://www.seer.cancer.gov/data), which provides information on Hispanics throughout the entire analysis period.

established between the ICD-8 and ICD-9 coding systems as well as between ICD-9 and ICD-10 codes; however, the correspondence is imperfect. These issues are typically minor when looking at broad causes of death (e.g. those from cardiovascular disease) but are sometimes important for specific sources of mortality. To provide information on this, the National Center for Health Statistics has calculated "estimated comparability ratios" indicating the relative number of deaths in 1996 attributed to a specific cause using ICD-9 and ICD-10 classifications (Anderson, et al., 2001) and, similarly, for 1976 when using ICD-8 versus ICD-9 codes (Klebba & Scott, 1980).

When the estimated comparability ratios are close to one (i.e. a similar number of deaths are reported using either set of ICD codes), issues of data comparability are likely to be minor and well captured by the inclusion of year fixed-effects in the estimation models. For example, the estimated comparability ratios are 1.013 and 1.003 for fatalities due to CVD and cancer, when using ICD-8 and ICD-9 codes, and 0.998 and 1.007 when using ICD-9 and ICD-10 categories. However, the potential problems are greater for some numerically important causes of death, as well as for some others that have been analyzed in previous research. For instance, the ICD-9 and ICD-10 code comparability ratio is 0.698 for influenza/pneumonia, indicating that 30% fewer deaths are recorded from this cause using the more recent coding system. On the other hand, the reported number of deaths due to for kidney disease (nephritis, nephrotic syndrome, nephrosis) or Alzheimer's Disease is much higher when using ICD-10 rather than ICD-9 categories – the estimated comparability ratios are 1.232 and 1.554. In such cases, the inclusion year dummy variables may not adequately account for the coding changes.

Due to concerns about comparability across time, the analysis of detailed disease causes of death is largely restricted to subcategories of cardiovascular disease – heart disease, cerebrovascular disease (stroke) and other CVD – and specific types of malignant neoplasms –

cancer of the digestive organs, lung/respiratory system, genital organs, lymphatic system, and all other cancers. In addition, deaths from external causes are divided into those due to transport accidents, other (non-transport) accidents, intentional self-harm (suicide), and homicide/legal intervention. Because non-transport accidents will be shown to have important explanatory power, some separate analysis is conducted for important components of them including deaths due to: falls, drowning/submersion, smoke/fire/flames, and poisoning/exposure to noxious substances.[18]

I also examined deaths from specific diseases such as diabetes, Alzheimer's, chronic lower respiratory, and kidney disease. Several additional steps were taken when conducting these analyses. First, because the ICD-8 and ICD-9 categories were often not comparable, the analysis begins in 1979 (the first period ICD-9 codes were used) rather than 1976. Second, based on CDC analyses of classification changes between ICD coding systems (Anderson et al., 2001), deaths from pre-senile dementia (ICD-9 code 290.1) during 1979-1998 were classified as Alzheimer's disease and those due to end-stage renal disease (ICD-9 code 593.1) were placed with kidney disease (Nephrititis/Nephrosis/Nephrotic Syndome). Even so, there is reason to doubt whether deaths from these sources are comparable across time, and so they receive limited attention.[19]

A variety of potential mediating determinants of the macroeconomic effects were examined including: 1) the industrial composition of employment, as measured by the percent of jobs in manufacturing, construction and farming industries); 2) average per capita incomes and wealth, where the latter is proxied by median-single family home prices; 3) nonmarket time, decomposed into annual weeks worked and usual work hours conditional on some employment;

[18] These account for 65% of deaths due to non-transport accidents. The most important remaining category, deaths due to "other and unspecified transport accidents and their sequelae" is not comparable over time

[19] To provide one example, the national number of deaths from Alzheimer's disease rose from 1,010 in 1979 to 82,435 in 2008. Although much of the increase was due to population aging (Alzheimer's is a disease primarily affecting the old), some of it almost certainly reflects increases in reporting and diagnosis (Hoyert, 1996).

4) highway vehicle miles driven; 5) the percent of 25-60 year olds with any health insurance and (separately) public or private health insurance; 6) current smoking, body mass index (BMI), obesity (BMI $\geq$ 30) and severe obesity (BMI $\geq$ 40).[20] Each of these might plausibly be related to mortality through their direct influence on health (e.g. smoking and obesity), the ability to purchase health inputs including medical care (e.g. income, wealth and health insurance), or because they present direct fatality risks (e.g. miles or employment in industries with high accident rates).[21]

Industry employment data come from the *Bureau of Economic Analysis* (www.bea.gov), those on health insurance and employment weeks and hours from the *March Current Population Survey* (http://cps.ipums.org/cps), housing prices from the *Freddie Mac House Price Index* (www.freddiemac.com/finance/fmhpi), highway miles from the Federal Highway Administration *Highway Statistics*, and those on smoking, BMI and obesity are from the *Behavioral Risk Factor Surveillance System* (www.cdc.gov/brfss). The data on mediating factors are available over the full 1976 through 2009 period, except for the health behaviors where the first year is 1987 and health insurance where it is 1988.[22]

Appendix Table A.1 provides details on the ICD codes used to classify specific causes of death. Appendix Table A.2 supplies means and sample standard errors for mortality rates (per

[20] Body mass index is weight, in kilograms, divided by the square of height in meters.

[21] The role of health behaviors and income as mediating factors has received extensive attention in related previous research, as well as in a literature examining the role of short-term income fluctuations on mortality and (e.g. Evans & Moore, 2012). The health effects of housing wealth, house prices and foreclosures have also been studied (Currie & Tekin, 2011; Fichera & Gathergood, 2013). Vehicle miles driven have traditionally been closely correlated with real personal income, but this relationship appears to have weakened in recent years (Memmott, 2007). Declines in the availability of private health insurance during economic downturns may be offset by increased eligibility for public insurance (e.g. Medicaid) but this may have become less true it than it used to be because state and local revenues have become more volatile over time (General Accountability Office, 2011). Work hours also declined more during the 2007-2009 downturn than in other recent periods of economic weakness, but it is not clear whether this represents a change in the responsiveness to macroeconomic conditions or if it was simply due to the severity of the recent recession (Kroll, 2011). This analysis does not investigate some other potential mechanisms, such as the changes in nursing home staffing hypothesized to be important by Stevens, et al. (2011).

[22] The *BRFSS*, began in 1984 but with only 15 states. By 1987, there were 32 states plus the District of Columbia in the sample, with number increasing to 44 plus DC in 1990, and with all 50 states and DC participating after 1995.

100,000 population), state characteristics (age, sex and race-ethnicity shares), and for the potential mediating factors.

Appendix Table A.3 illustrates how the sources of death have changed over the analysis period by showing numbers and shares of deaths from specific sources during the first and second halves of the sample period (1976-1992 and 1993-2009). As expected, given increasing life expectancies, the proportion of mortality accounted for by the elderly has grown over time: for example, 27% of deaths were among those ≥85 and and 55% for those ≥75 during 1993-2009, versus 20% and 45% from 1976-1992. Conversely, shares of death to those <65 (<45) fell from 31% (11%) to 26% (9%) of the total.

The causes of mortality also changed substantially over time (partly due to population aging). Cardiovascular deaths declined from 47% of the total in 1976-1992 to 38% in 1993-2009. Conversely, cancer accounted for a slightly larger share of fatalities in the later years (rising from 22% to 23%), during which time there was also a rapid rise in deaths from other disease (from 24% to 35%), with substantial increases deaths due to diabetes, Alzheimers', chronic lower respiratory, liver and kidney disease. The share of deaths from external sources fell slightly over time (from 7.4% to 6.8%), with reductions for mortality from transport accidents and homicides being offset by an increased share of mortality due to non-transport accidents.

3. The Declining Procyclicality of Total Mortality

The relationship between macroeconomic conditions and mortality has changed markedly over time, essentially resulting in the elimination of the previously observed procyclical relationship. Table 1 shows initial econometric findings obtained for the entire (1976 – 2009) sample period, with the natural log of the total mortality rate as the dependent variable. All models control for the sex, race/ethnicity and age composition of the state population, as well as for state and year dummy variables. Columns (1a) and (2a) show results from estimating equation (1), where the macroeconomic effect is treated as constant over time. Columns (1b) and (2b) provides corresponding findings from equation (2), which adds interactions between the unemployment rate and a linear time trend. As discussed, the trend variable equals zero in 1976 and one in 2009, so that the predicted macroeconomic effects are proxied by $\hat{\gamma}$ and $\hat{\gamma}+\hat{\delta}$ in these years. The specifications shown further vary according to whether or not the data are weighted by size of the state population (columns 2a and 2b versus 1a and 1b) and if linear state time-trends are also controlled (the top versus bottom panels).

The preferred specifications, focused upon in the subsequent analysis, are in columns (2a) and (2b) of the bottom panel of Table 1.[23] These estimates weight the data by the size of the state population and include vectors of state-specific linear trends. Weighting is important if the macroeconomic effects are heterogeneous between small and large states and we are interested in the overall average treatment effect, since unweighted models overemphasize the influence of small states.[24] Controlling for state time trends may be useful if there are omitted time-varying confounding factors (which becomes more likely as the analysis period lengthens) and because most mortality rates have trended sharply downwards over time.[25]

Using the preferred specifications, a one percentage point increase in the unemployment rate is estimated to reduce the total mortality rate by 0.30%, which is in line with previous

[23] Table A.4 displays coefficients for the other state level covariates (but not state or year dummy variables or trends) for the models in column (2b) of Table 1.
[24] For instance, this may occur if measurement error and migration flows are greater in small than large states.
[25] Mortality trends vary considerably across sources of death, with large reductions over time observed for mortality from cardiovascular disease and external sources, a relatively flat trend for cancer fatalities, and an increase for deaths from diseases other than CVD and cancer.

estimates.[26] With the unemployment rate-time trend interaction also included, the one point rise in joblessness is predicted to lower total mortality 0.54% in 1976 but to increase it by a statistically insignificant 0.08% in 2009.[27] The average effect is not very sensitive to whether or not the data are weighted or state time-trends are included; the time-varying estimates are somewhat more so. However, in none of these cases does unemployment coefficient predicted for 2009 differ significantly from zero (in either a statistical or economically meaningful sense).

A potential concern is that unemployment rates now provide different information on macroeconomic conditions than they did in previous decades and that this could be a source of the secular trends just reported.[28] To investigate this possibility I estimated models equivalent to those above but with controls for nonemployment rather than unemployment rates, where the former are defined as the percentage of the civilian population (aged 16 and over) who are not working, whether because they are unemployed or out of the labor force. These models provided quite similar estimates of the relationship between macroeconomic conditions and total mortality: in the preferred specification, a one percentage point increase in the nonemployment rate predicted a statistically significant 0.45% reduction in total mortality in 1976 but an insignificant 0.16% increase in 2009.[29]

Figure 1 provides three alternative way of showing that the procyclicality of total mortality has diminished over time, by providing sets of estimates of equation (1), without

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[26] Ruhm (2000) obtains a slightly larger 0.5% reduction in total mortality but the current estimate is close to the 0.3% predicted decrease obtained in Stevens et al.'s (2011) preferred specification.
[27] Here and throughout, statistical significance refers to p-values of 0.05 or less.
[28] For instance, declines in labor force participation rates were particularly pronounced during the "great recession" that began in 2007, when compared to other economic downturns (Shierholz, 2012).
[29] Changes in interstate migration rates over time are also unlikely to explain the results. Migrants tend to move from areas of higher to lower unemployment rates and healthy individuals are more likely to migrate than are those in poorer health (Halliday, 2007). This will tend to introduce a countercyclical mortality effect. Secular reductions in the procyclicality of mortality might then occur if migration rates were increasing over time. However, migration, after rising throughout the first three-quarters of the 20th century, peaked around 1980 and rates have instead fallen sharply since 1980 (Molloy, et al., 2011).

unemployment rate time trend interactions, for varying time periods. The solid lines show point estimates and dotted lines the 95-percent confidence intervals. Figure 1A displays unemployment rate coefficient estimates where the analysis period begins in 1976 and ends in years that vary between 1985 and 2009. The magnitude of the estimated macroeconomic effect initially increases, as the time period extends beyond 1985, and reaches a peak when the final year is 1991 – rising from -0.0021 to -0.0040.[30] There is little sensitivity to the choice of ending years between 1990 and 2004 – where the unemployment coefficients range between -0.0036 and -0.0040 – but for analyses continuing beyond 2004, the estimated unemployment rate effect weakens, falling to -0.0030 for the entire 1976-2009 period, although still statistically significant. The results of Figure 1A are substantially similar to those obtained in previous research and do not alter the conclusion that mortality is procyclical, although less so now than in the past.

The sensitivity of results to the choice of sample periods can be seen more explicitly in Figure 1B, where the final sample year is always 2009 but the starting analysis year varies between 1976 (the full period) and 2000. Here we see that the unemployment coefficient declines from -0.0030 for the full sample period to between -0.0005 and -0.0014 when beginning the analysis between 1980 and 1997 (although it again increases for the relatively short and recent samples, beginning after 1997). Perhaps more importantly, we fail to reject the null hypothesis of *no* macroeconomic effect for any analysis periods beginning after 1978.

Figure 1C displays the results obtained using 20-year sample windows, beginning in the specified year. For example, the left-most entry shows that the unemployment coefficient for 1976-1995 is -0.0039, while that furthest to the right entry shows that the corresponding

[30] For ease of exposition, I will frequently refer to the implied magnitudes of the effects, while ignoring that the absolute changes in coefficients take the opposite patterns when the coefficient has a negative sign. For instance, I may state that the unemployment effect "weakens" or "decreases" when the coefficient changes from -0.0040 to -0.0030, even though the coefficient actually less negative (and so larger) in this case.

coefficient for 1990-2009 is -0.0005. Notably, while total mortality is estimated to be significantly procyclical (negative unemployment rate coefficients) for all 20-year windows beginning between 1976 and 1986, the size of the predicted effect diminishes fairly steadily thereafter and is small and insignificant for 20-year windows beginning after 1986.

The choice of 20-year sample windows in Figure 1C is somewhat arbitrary and may conceal an increased procyclical variation of mortality towards the end of the data period, that was suggested in right-hand-side of Figure 1B. This possibility is investigated in Figure 2, which replicates the analysis in Figure 1C, but with analysis periods that vary in duration between 5-years and 20-years. Two main findings deserve mention. First, the estimates become more volatile and less precise as the sample duration shortens. Thus, when using 5-year windows, the unemployment coefficients fluctuate wildly with even relatively small changes in the analysis window (for instance, from 0.0055 for 1995-99 to -0.0116 for 1999-2003) but we almost always fail to reject the null hypothesis of no macroeconomic effect. Second, the standard errors have typically increased for more recent samples. As a result, the estimates obtained using 10-year or 15-year analysis windows, while less volatile than those using 5-year periods, still lack sufficient precision to allow us to determine whether the apparent partial reversion of the macroeconomic effects (towards more procylical mortality) in recent years is real or simply reflects noise in the estimates.[31] Another important implication is that the findings of some recent investigations of macroeconomic variations in health outcomes or behaviors should be viewed with extreme caution because the analysis periods are too short to provide reliable estimates.[32]

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[31] For example, when using 10-year periods, the average standard error is over twice as large for analysis windows beginning between 1989 and 2000 as for those starting between 1976 and 1988 (0.0019 vs. 0.0009).

[32] For instance, Charles & DiCicca's (2008) analysis of male obesity uses data from 1997-2001; Arkes' (2007, 2009) investigations of teenage body weight utilize information from 1997-2004, Dávlos et al.'s (2012) study of alcohol abuse and dependence compare 2001-02 and 2004-05, Colman & Dave's (2011) research on work and leisure-time physical activity covers 2003-2010, Cotti & Tefft's (2011) analysis of alcohol-related vehicle fatalities uses data

The results of subgroup analyses, examining gender- and age-specific sources of deaths, as well as those from diseases and external causes (accidents, homicides and suicides) are summarized in Table 2 and Figure 3. This and later figures show point estimates only, for 20-year analysis windows that begin in the year specified on the X-axis, with thicker lines indicating sources with relatively higher mortality rates.[33]

Gender differences are fairly minor. Over the entire period, a one percentage point increase in unemployment lowers predicted male mortality by 0.26% and the female death rate by 0.33%. The procyclicality of mortality disappears in later years for both sexes, with an insignificantly more positive unemployment rate coefficient in 2009 for men than women (0.0016 versus 0.0000). These patterns are replicated when using 20-year analysis windows (Figure 3A), with the one difference being that we are able to reject the null hypothesis of a zero unemployment coefficient in somewhat later 20-year sample periods for females than males.[34]

Secular changes in the macroeconomic effects appear to be relatively large for the youngest and oldest age groups, although imprecision of many of the estimates implies that these conclusions should be interpreted with caution. Conversely, there is relatively little change in the unemployment coefficients of 65-74 year olds, and this is the only group for whom the point estimate remains negative throughout the sample period.[35] Specifically, in 1976, a one percentage point increase in unemployment was predicted to *reduce* the death rates of <45, 45-54, 55-64, 65-74, 75-84 and ≥85 years old by 1.3%, 0.2%, 0.3%, 0.5% and 0.8%, whereas the

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from 2003-2009, and Tekin et al. (2013) investigate a variety of health outcomes and behaviors using data from 2005 to 2011.

[33] For example, in Figure 3B, the lines for 65-74 and 75-84 year olds are thick because they account for 20% and 28% of mortality, over the full period, whereas that for 45-54 year olds is thinner because this age group is responsible for less than 7% of deaths. Including 95-percent confidence intervals would make the figures difficult to interpret; however, they are discussed in the text where relevant.

[34] For women, we can reject the null hypothesis for all 20-year windows beginning earlier than 1987, whereas for men we are only able to do so for periods starting prior to 1983.

[35] However, we are unable to reject the null hypothesis of no macroeconomic effect in 2009 for any age group.

same change was estimated to have *increased* them by 0.9%, 0.5%, 0.3%, -0.2%, 0.1% and 0.4% in 2009 (see the fourth through ninth rows of Table 2).

The estimates obtained using 20-year windows, summarized in Figure 3B, are qualitatively similar but provide useful additional information. The 95-percent confidence intervals (not shown) exclude positive unemployment coefficients for all two-decade periods beginning prior to 1989 for 65-74 year olds and before 1985 and 1987 for 75-84 and ≥85 year olds. Conversely, the parameter estimate does not differ significantly from zero in any of the 20-year periods for 45-54 year olds and, for persons under 45, only does so where the initial year is 1976 or 1977. These findings provide the first hint that the overall changes in macroeconomic effects reflect diverse influences across causes of death.

The last two rows of Table 2, in combination with Figure 3C, confirm this possibility by demonstrating that levels and trends of the macroeconomic effects differ markedly for mortality from diseases versus those from external causes. A one point rise in joblessness lowered predicted disease mortality by 0.5% in 1976 while having no effect in 2009. By contrast, the estimates indicate a much larger corresponding 1.9% reduction in external deaths for the earlier year versus a statistically significant 1.2% increase in the later one. The results obtained using 20-year analysis windows suggest an almost monotonic but modest reduction over time in the unemployment coefficient for deaths from disease, with statistically significant negative estimates obtained for all two decade periods beginning before 1987. Conversely, while the estimated unemployment rate parameter estimate on mortality from external causes remains negative and fairly stable for 20-year periods whose first year is between 1976 and 1982, it attenuates sharply and steadily thereafter, with the data failing to reject a zero effect for windows starting between 1983 and 1988, and with significant positive effects obtained for those beginning in 1989 or 1990. These results help to explain the sharp reversal in the effects of macroeconomic conditions on deaths of persons <45 years old, since they accounts for 56% of all external deaths but less than 10% of total mortality.

4. Heterogenous Effects for Specific Sources of Death

Table 3 and Figure 4 provides further detail by stratifying mortality into three major types of diseases and four important external sources. The three disease categories: cardiovascular, cancer, and other disease, account for 42%, 23% and 29% of deaths over the whole sample period. The four external sources: transport accidents, other accidents, suicides and homicides are responsible for 2.2%, 2.4%, 1.4% and 0.9% of fatalities. Finally, four specific categories of other (non-transport) accidental deaths are considered – falls, drowning/submersion, smoke/fires/flames and poisoning/exposure to noxious substances – which constitute 0.7%, 0.2%, 0.2% and 0.5% of mortality.

4.1 Disease Mortality

There are striking disparities in both average and secular changes in the effects of macroeconomic conditions on deaths from different types of diseases. In particular, cancer mortality was unrelated to macroeconomic conditions in 1976 but became strongly countercyclical by 2009, whereas CVD and other disease mortality remains procyclical, with more modest changes occurring over time (see the first four rows of Table 3 and Figure 4A).

Research for earlier time periods (e.g. Ruhm, 2000; Neumayer, 2004; Miller et al., 2009) documents a strong procyclicality of CVD deaths but with little macroeconomic variation in cancer fatalities, and attributes this heterogeneity to the likelihood that short-term changes in behaviors (e.g. smoking, diet and exercise) more strongly influence the risk of death from cardiovascular disease than cancer. A possible explanation for the findings just described is therefore that the relationship between macroeconomic conditions and health behaviors has remained relatively stable over time, while cancer mortality has become more sensitive to the access to health care (which is procyclical in the U.S.) due to improvements in expensive medical treatments and technologies. This possibility is addressed in section 7.

Appendix Table A.5 and Figure A.1 provide a more detailed breakdown of deaths from disease: dividing CVD into heart disease, cerebrovascular disease (strokes) and other CVD (e.g. circulatory disorders); malignant neoplasms into cancers of the digestive organs, lung, breast, genital organs, leukemia/lymphatic cancer, and other cancers. The “other” disease category includes diabetes, Alzheimer’s, chronic lower respiratory, liver and kidney disease, which together account for just under 40% of disease mortality not attributable to CVD or cancer. As discussed, further analysis of deaths from other diseases is hindered by the lack of comparability between ICD-9 and ICD-10 codes; also 1979 is the first sample year for those that are investigated, to avoid the added complexity of comparing across ICD-8 and ICD-9 categories.[36]

The results confirm that the predicted effect of macroeconomic conditions on heart and cerebrovascular disease, the two major sources of CVD deaths, has remained essentially unchanged over time: a one percentage point increase in unemployment is predicted to reduce deaths from the former by around 0.2% and the latter by about 1.1%.[37]

Conversely, there is substantial variation across types of malignant neoplasms. Consider lung and digestive cancers, accounting for 28% and 24% of cancer mortality respectively. A one point rise in joblessness had essentially no estimated effect on lung cancer deaths in 1976 but

[36] The linear time trend variable is therefore set to range between zero in 1979 and one in 2009 for these analyses.
[37] The effects on deaths from other cardiovascular disease, which account for around 6% of CVD mortality, are somewhat less stable but do not change by statistically significant amounts over time.

was predicted to raise them by a statistically significant 0.8% in 2009. By contrast, digestive cancer fatalities were largely unresponsive to the macroeconomy in either period, and with no clear secular trend.[38] Mortality from breast, lymphatic and genital cancers (accounting for 8%, 10% and 11% of cancer deaths) may have also become less procyclical or more countercyclical over time, although we can we never reject the hypothesis that mortality from these sources is unrelated to macroeconomic conditions.

The data also hint at the possibility that deaths from some other diseases may have become less procyclical over time, while noting the cautionary caveats mentioned earlier. Specifically, the unemployment rate-trend interaction coefficients in Table A.5 are strongly positive for fatalities from diabetes, kidney and liver disease, although only the last of these approaches statistical significance and the data generally fail to reject the possibility of no macroeconomic effect in any analysis year.

4.2 External Sources of Death

As with major sources of disease, there is considerable heterogeneity in average macroeconomic effects and their trends for specific sources of external deaths (see the fifth through ninth rows of Table 3 and Figure 4B). One of the most consistent findings of previous research that transport fatalities are procyclical.[39] This effect persists, although it may have weakened somewhat over time, with a one percentage point rise in the unemployment rate predicting around a 2% reduction in 2009 versus a more than a 3% decrease in 1976. Also consistent with most (but not all prior studies), suicides increase with joblessness, and the effect may have strengthened over time, with the one point growth in unemployment being associated

[38] The results in Table A.5 raise the possibility of a small positive time trend but the analyses of 20-year windows, shown in Figure A.1, raise doubts that this has occurred.
[39] Previous analyses have often examined motor vehicle deaths, which constituted 94% of transport accident fatalities from 1976-2009. (The remainder includes other land transport, water, air, space and other unspecified transport accidents.) Transport deaths are considered here because they are coded more consistently across time.

with a 0.9% rise is suicides in 1976 versus a 2.4% growth in 2009.[40] The estimates for homicides are sensitive to the choice of models, with the results for equation (2) over the full time period, shown in Table 3, suggesting a strong procyclical pattern in 1976 but an acyclical or countercylical variation in 2009. Conversely, the 20-year analysis windows reveal no consistent evidence of a trend, nor does the unemployment coefficient ever differ signficantly from zero (see Figure 4B).

The most noteworthy finding is that deaths due to non-transport accidents have switched from being strongly procyclical to sharply countercylical: estimates of equation (2) indicate that a one point rise in unemployment *reduced* predicted fatalities from this source by 2.5% in 1976 but *increased* them 2.3% in 2009. Results obtained using 20-year sample windows, shown in Figure 4B, are entirely consistent with this, showing a monotonic increase in the unemployment coefficient over time – ranging from -0.0165 for 1976-1995 to 0.0251 for 1990-2009 – with a statistically signficant *negative* parameter estimate for all 20-year periods starting prior to 1982 and a significant *positive* coefficient for those beginning in 1988 or later.

The last five rows of Table 3 and Figure 4C further decompose the effects of non-transport accidents and show that the secular trends are dominated by changes in the effects of macroeconomic conditions on deaths due to accidental poisoning, where an extremely strong countercyclical pattern emerges for 20-year analysis windows beginning after the early 1980s. Conversely, there is little consistent evidence of a change over time in deaths resulting from falls, drowning or fires. Given these results, deaths due to accidental poisoning will receive considerable attention below.

[40] When using 20-year analysis windows, the unemployment coefficient for suicide is positive in all years, statistically significant for all periods beginning after 1980, while just missing being significant in earlier sample windows (Figure 4B).

5. Decompositions of Secular Changes in Macroeconomic Effects

Using the decomposition methods described previously, I next demonstrate that the declining macroeconomic responsiveness of total mortality results from secular changes in the effects for specific sources of mortality, rather than because of shifts over time in the composition of deaths (towards those that are less procyclical). All numerical calculations in the tables refer to the predicted effect of a one percentage point increase in the unemployment rate.

Table 4 summarizes decomposition results based on age and major cause of death. The first row shows the expected change, between 1976-80 and 2005-09, in the impact of macroeconomic conditions on total mortality. A one point rise in unemployment predicts a 12,271 reduction in deaths in 1976-80 versus a 1,011 increase in in 2005-09, or a total of 13,283 additional deaths per year in the later period.[41]

Discussion of the decompositions focuses on predicted secular *changes* in macroeconomic effects, rather than on their levels. For example, the last two entries in the second row of Table 4 indicate that declines, between 1976-80 and 2005-09, in the share of deaths accounted for by persons <45 years old resulted in 1,003 additional fatalities, from a one point rise in unemployment, in the later period compared to the earlier one. This occurred because the mortality rates of this age group were relatively procyclical at the beginning of the analysis period, so that a reduction in their share of all deaths decreases the procyclicality of total mortality. On the other hand, reduced procyclicality of mortality for this age group (i.e. Δ in the coefficients) predicts a corresponding increase of 3,851 deaths. For brevity, this is explained

[41] The unemployment coefficient in 1978 (midpoint of the 1976-80 period) was -0.00503, while that for 2007 (middle of 2005-09 timespan) was 0.00041. The resulting coefficient change of 0.00544 implies around 0.5% more fatalities, or 13,283 additional deaths per year, based on 2,441,428 deaths that occurred annually between 2005-09. This was calculated as: (0.0004143 − -0.0050263) × 2,441,428 = 13,283 deaths. As previously discussed, the decompositions are based on linear approximations from coefficients on log mortality rates. The exact estimates (using exp($\hat{\gamma}$)-1 rather than $\hat{\gamma}$), imply an increase of 13,319 deaths.

below using language along the lines of: “for persons < 45, changes in the shares predict an increase of 1,003 deaths while changes in the coefficients account for 3,581 additional deaths”.

Table 4 demonstrates that changes in the composition of mortality account for little of the secular shift in macroeconomic effects – 1% to 2% of the total, depending on whether decomposing by age or major cause of death. By contrast, changes in the unemployment rate coefficients for major causes account for 87% of the overall secular trend and those for age groups for over 100%.[42] When decomposing by age, changes in the coefficients for <45, 75-84 and ≥85 year olds play a particularly central role, predicting 3,851, 3,581 and 7,317 additional deaths from a one point rise in unemployment. The importance of the two oldest age groups is expected. Since they account for 43% of deaths in 1976-80 (and 56% in 2005-09), changes in the coefficients translate into large variations in the number of deaths. By contrast, fewer than 12% of fatalities involve persons under the age of 45 and the overall importance of this group results from the extremely large change in the unemployment coefficient (from -0.0120 in 1978 to 0.0079 in 2007). It also points to a likely role for external sources of death, which accounts for over 40% of mortality for this age group.

The bottom panel of Table 4, shows that changes over time in the unemployment coefficients predict around five thousand additional deaths annually from external causes, accounting for more than 35% of the total secular trend. This occurs even through only 7% to 8% of all deaths are due to external causes, and helps to explain the large change for <45 year olds just described.[43] Cancer is the other key contributor, with changes in the coefficients accounting for around three thousand additional deaths, or almost one-quarter of the total macroeconomic change. Cardiovascular disease plays a smaller role, even though it is the number one cause of

[42] Since separate (unconstrained) models are estimated for different sources of death, the total contributions of changes in coefficients on mortality shares can sum to more or less than the change predicted for total mortality.
[43] External sources account for over 40% of mortality involving persons under the age of 45.

death, because the secular change in the unemployment coefficient is modest and imprecisely estimated. The decline in CVD as a share of total deaths also plays a role but its effect is fully offset by the increased share of deaths from “other” diseases, so that changes in the disease composition of mortality explain virtually none of the overall effect.

Given the importance of external causes and cancer in explaining the overall trend in macroeconomic effects, Table 5 provides a more detailed decomposition for deaths from these sources, separately analyzing six types of cancer, four external causes and, among the external causes, subcategories of non-transport accidents. Changes in the unemployment coefficients, rather than shifts in the composition of deaths, are again of primary importance, accounting for 88% to 98% of the overall macroeconomic trend for cancer deaths and essentially all of the change in external fatailities and in those due to non-transport accidents. Coefficient changes for lung cancer are responsible for 30% of the overall cancer effect, with those for breast and lymphatic cancers accounting for 14% and 16% of the cacer total.

Non-transport accidents play the key role in explaining why external causes of death shift from being procyclical in 1976-80 to countercyclical in 2005-09. Specifically, the switch from a large negative to a sizeable positive unemployment coefficient accounts for over 3000 additional deaths annually, constituting almost two-thirds of the predicted rise in external mortality. Moreover, this increase is approximately equal, in terms of the number of deaths affected, to that for all types of cancer, even though these accidents only account for around 3% of all fatalities (versus over 20% for cancers). The bottom panel of Table 5 demonstrates that this is almost entirely explained by the huge change in the relationship between macroeconomic conditions and deaths due to accidental poisoning.

6. Age-Specific Mortality from External Causes and Cancer

The previous section highlighted the role of external causes of death, particularly those due to accidental poisonings, and cancer in explaining the elimination of the procyclical fluctuation in mortality over the last three decades. Since external causes disproportionately affect the relatively young and most cancer deaths involve person who are fairly old, I explore these relationships in greater detail by providing a separate analysis of these causes of mortality for three broad age groups: <45, 45-74 and ≥75 year olds. Table 6 summarizes the results.

The predicted effect of macroeconomic conditions on all three sources of fatalities exhibits a substantial secular trend for <45 year olds. In 1976, a one-point increase in joblessness was estimated to reduce deaths from cancer, external sources, and accidental poisoning by 0.9%, 2.2% and 2.7%%. This compares to predicted increases of 1.1%, 0.9% and 15.4%, in 2009.

Reversal of the procyclicality of mortality from these three causes is more muted for persons 45 and over. The one point rise in joblessness predicts a 0.0% and 1.3% declines in mortality from cancer and external sources for 45-74 year olds in 1976, and a 1.2% increase in poisoning deaths, versus increases of 0.3%, 1.7%, and 10.5% in 2009. For persons ≥75, the predicted mortality reductions in 1976 are 0.0%, 1.6%, and -1.3% in 1976 while, in 2009, cancer and external deaths are estimated to increase by 1.0% and 0.4%, whereas poisoning fatalities fall by a statistically insignificant 0.4%.

Less pronounced secular changes in the effects of macroeconomic conditions on fatality *rates* for older (versus younger) individuals need not imply smaller impacts on the *number* of deaths, since mortality rises so rapidly with age. This can be seen in Table 7, which shows decomposition results for age- and cause-specific mortality. Once again, changes in the coefficients, rather than in the composition of deaths, accounts for most of the overall secular trend in macroeconomic effects. Moreover, this exercise illustrates that secular changes in cancer deaths are dominated by ≥75 year olds while those for external causes, particularly poisonings, primarily occur among those under the age of 45.

Specifically, cancer accounts for 23% of the overall trend in macroeconomic effects, with changes in the unemployment coefficients for person ≥75 being responsible for 71% of this. Conversely, the elderly account for little of the corresponding change due to external sources of death and none that resulting from accidental poisonings. Instead, these are dominated by shifts in the coefficients for <45 year olds, with 45-74 year olds playing a non-trivial role. This is particularly true for accidental poisoning, where the youngest group accounts for 67% of the mortality trend due to changes in unemployment rate coefficients, and 45-74 year olds for 27%.

7. Mechanisms

I next provide a preliminary two-step exploration of potential reasons for the declining procyclicality of mortality. In the first stage, equations (1) and (2) are estimated using potential mechanisms, instead of mortality rates, as outcomes. The goal is to identify possible mediating factors whose macroeconomic fluctuations have changed over time. For instance, income variations may have become more or less procyclical in recent years. In the second-stage, these mechanisms are added to the mortality equations, to examine whether their inclusion attenuates the unemployment-time trend interaction coefficient. This portion of the analysis focuses on deaths from cancer and external causes, since they were shown above to explain most of the weakening procyclicality of overall mortality.

Table 8 summarizes the first-stage results. The average unemployment coefficients in model (a) generally conform with expectations: unemployment rates are negatively correlated with manufacturing and construction (but not farming) employment, incomes and home prices, weeks and hours worked, highway vehicle miles, and overall or private (but not public) health insurance coverage, while the predicted effects for bodyweight, obesity and smoking are statistically insignificant.

More relevant for this analysis are the unemployment-trend interactions shown in model (b). The most important findings are that per capita incomes and median housing prices have become more cyclically sensitive over time, while the reverse is true for highway vehicle miles: a one percentage point increase in unemployment reduced estimated incomes, home prices and miles driven by 0.7%, 0.7% and 1.5% in 1976 versus 1.9%, 7.7% and 0.3% in 2009. Construction employment has also become more cyclically sensitive but jobs in manufacturing industries less so. The interaction coefficients are small and statistically insignificant for the other potential mechanisms. For health behaviors and insurance coverage, this may reflect the relatively short period over which the data are available, although the lack of an effect on healthy behaviors is consistent with evidence that the CVD fatalities (which may be strongly influenced by them) have not changed dramatically over time.

I next examine whether the unemployment rate main effect and trend interaction coefficients, in models examining cancer and accidental poisoning mortality, are attenuated when adding potentially important mechanisms as supplemental regressors. The main findings are displayed in Table 9, with the original specifications in columns (a) and (b), median home prices added in specifications (c) and (d), and per capita incomes also controlled for in models (e) and (f). The results for other potential mechanisms are briefly discussed below.

Financial resources appear to have gained importance as protection against cancer deaths. As shown in the top panel of Table 9, a one percent increase in per capita incomes reduces overall cancer fatalities by 2% to 3%, with a slightly smaller but more precisely estimated 2%

decrease for corresponding growth in median home prices. Moreover, their inclusion in the model eliminates the 0.2% average reduction in cancer mortality predicted by a one point rise in unemployment (compare columns a and e) and substantially attenuates the unemployment-trend interaction coefficient: without controlling for incomes and home prices, a one point increase in unemployment is predicted to reduce cancer fatalities by a statistically insignificant 0.1% in 1976 versus a significant rise of 0.5% in 2000 (column b); when they are included. the same 0.1% percent drop in cancer mortality is anticipated in 1976 but a considerably smaller 0.3% increase in 2009 (column e). Similar patterns are obtained for digestive, lung, and breast cancer fatality rates (not shown). In each case, higher housing prices predict lower mortality (and the same is true of incomes, except for breast cancer) and their inclusion eliminates or attenuates both the average and trend component of the macroeconomic effect.[44] These results are consistent with an explanation that cancer mortality has become more countercyclical in recent years due to an increasing sensitivity of incomes and home prices to macroeconomic conditions.

I also estimated models with controls for public and private insurance coverage. Although insurance coverage was generally negatively related to cancer deaths, its inclusion did not materially affect the unemployment rate coefficients. This could reflect limitations of the insurance data, which did not begin until 1988, or the near universality of insurance coverage through Medicare for the elderly who account for the vast majority of cancer mortality.[45]

Deaths due to accidental poisoning are examined in bottom panel of Table 9. There is an extremely strong negative effect of median home prices on this source of mortality and

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[44] Conversely, these controls had little effect on predicted deaths from other types of cancer.

[45] I also tested whether the results for all, digestive, lung or breast cancers were sensitive to adding controls for vehicle miles driven and the industrial structure of employment (with health insurance coverage excluded so that data were available for the full sample period). The coefficients on the associated coefficients were generally insignificant – exceptions included a significant positive coefficient of manufacturing employment on lung cancer deaths and a negative effect of vehicle miles on breast cancer death – and their inclusion did not materially affect the coefficients on the unemployment rate main effects or the unemployment-trend interactions.

controlling for them attenuates the average unemployment effect by 35% and the unemployment-trend interaction by 18% (compare columns a vs. c and b vs. d). This suggests that wealth is protective against the risk of poisoning fatalities; however, there are two reasons question the extent to which this relationship is causal. First, the coefficients on home prices are implausibly large, indicating that at least some of the observed effect may be due to uncontrolled confounding factors. Second, the coefficients on per capita incomes, added in columns (e) and (f), have the wrong sign (for this explanation) although they are imprecisely estimated.[46]

8. Discussion

Total mortality has switched from being strongly procyclical during the 1970s and 1980s to being essentially unrelated to macroeconomic conditions during the first decade of the 21st century. However, such patterns are not uniform across causes of mortality. For instance, cardiovascular fatalities continue to be procyclical, although possibly less strongly so than in previous years, whereas cancer deaths have moved from being unrelated to macroeconomic conditions to being substantially countercyclical. Similarly, external sources of death have switched from procyclical to countercyclical, with accidental poisoning fatalities the key contributor to this change.

These conclusions should be interpreted with some caution, since the estimates can be sensitive to changes in the starting and ending dates of analysis. Such parameter instability is a particular problem when the analysis window is short (probably anything less than 15-years) and raises even larger concerns about the findings of a number of recent investigations that have used relatively brief (often less than 10-year) periods. One contribution of this study is to provide

[46] I also examined, but found little support in the data, for the possibilities that the cyclical patterns of transport mortality were explained by changes over time in the effects of macroeconomic conditions on vehicle miles driven and that those due to falls resulted from secular changes in the economic responsiveness of manufacturing, construction and farming employment.

parsimonious methods of illustrating the sensitivity of the results the length of the analysis windows, as well as to the first and last years examined.

The results for cancer may reflect the increasingly important role of financial resources in facilitating the purchases of sophisticated but expensive treatments that have played a role in extending the lives of many cancer patients. Figure 5 provides evidence of this progress, showing age-adjusted mortality rates for lung and breast cancer, as well as from colorectal and prostate cancer (the most important sources of deaths from digestive and genital cancers) and non-Hodgkin's lymphoma and leukemia (which together account for most fatalities from lymphatic cancers).[47] It is noteworthy that death rates from lung, breast, prostate cancer and non-Hodgkin's lymphoma peaked in 1993, 1989, 1993 and 1997, at which point they were 34%, 4%, 24% and 56% above the 1976 levels. These peaks roughly coincide with the timing of important advances in the "war on cancer".[48] Mortality rates declined substantially thereafter, falling by 18%, 33%, 44% and 30% between the peak year and 2009, consistent with an increasing role for sophisticated medical and surgical treatments developed during the last 25 years.[49]

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[47] Breast cancer deaths in the figure refer to females only and prostate cancer mortality just to males.

[48] Cutler (2008) highlights the role of improvements in medications and chemotherapy agents developed in the late 1980s and 1990s for lung and prostate cancer and during the 1990s and early 2000s for colorectal cancer, as well as the rapid expansion of screening tests for colon cancer (colonoscopies), breast cancer (mammographies), and prostate cancer (PSA tests) in the 1980s and 1990s. Berry et al. (2005) indicate that increased screening and advances in treatments – chemotherapy and hormonal therapy and the targeted drug trastuzumab (Herceptin) – played approximately equal roles in accounting for the 21% decline in age-adjusted breast cancer mortality between 1975 and 2000. National Cancer Institute (2013) credits substantial reductions in deaths from advanced colon cancer to combining the use of an existing drug, 5-fluorouci1 (5FU), with the chemotherapy agents irinotecan (Camptosar) and oxalipatin (Eloxatin) which first became available in 1996 and 2002, as well as the antibody treatments cetuximab and bevacizumab, made available in 2004. Philipson et al. (2012) note that the U.S. cost per cancer case rose from $47,000 in 1983 to $70,000 in 1999. During the same period, they cite major changes in the treatment of prostate cancer (higher rates of radical prostatectomy, improvements in radiation therapy, hormone agonists, and drugs reducing testosterone levels), and the 1988 approval of trastuzumab (Herceptin) as major breakthrough for breast cancer. Dotan et al. (2010) credit rituximab (Rituxan), a monoclonal antibody approved for use in the U.S. in 1997, as a "paradigm shift" for treating certain types of non-Hodgkin's lymphoma, leading to substantial increases in overall and progression-free survival.

[49] Overall age-adjusted cancer fatality rates rose 6% between 1976 and 1991 and then fell 20% from 1991 to 2009. Colorectal cancer fatalities were only 55 percent as high in 2009 as in 1976, but declined steadily throughout the analysis period. Leukemia, deaths peaked in in 1980 and the decline fairly slowly (13% between 1976 and 2009).

The findings for accidental poisoning mortality are particularly interesting. Deaths from this source have increased dramatically for persons under the age of 75 since 1990s, with particularly rapid growth starting in the late 1990s (see Figure 6). This reflects higher rates of poisoning fatalities resulting from drug use, particularly those due to opioid analgesics such as morphine, hydrocodone (e.g. Vicodin), and oxycodone (e.g. OxyContin).[50] The growth in drug poisoning deaths has accompanied the rapid rise in opioid use for the treatment of chronic non-cancer pain, in part spurred by new pain management standards introduced by the Joint Commission on the Accreditation of Health Care Organizations (JCAHO) in 2000, liberalization of prescribing laws by state medical boards and aggressive marketing by the pharmaceutical industry (Manchikanti, et al., 2012).[51]

Although opioids are primarily prescribed to treat pain, there are strong linkages between pain, mental health problems and the legal or illicit use of narcotic analgesics.[52] Therefore, it seems likely that the increased procyclicality of poisoning partly deaths reflects the unintended consequences of illicit or prescribed use of opioids used to treat mental health problems, which become more prevalent during economic downturns. This need not reflect a change in the effects of macroeconomic conditions on mental illness – economic weakness has long been associated with increased rates of suicides or non-psychotic mental disorders and diminished mental health

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Not all of the declines are due to medical treatments. For example, reductions in smoking (particularly for males) are an important source of reductions in deaths from lung cancer.

[50] The share of poisoning deaths that were due to drugs rose from 56% to 89% between 1980 and 2008, and the overall poisoning fatality rate tripled during this period, with the number of deaths identified as being due to opioid analgesics rising by 270% (Warner et al., 2011). These figures include intentional drug poisoning deaths (suicides), which constituted 13% of the total in 2008, and deaths of undetermined intent (which could be accidents or suicides), accounting for 9% of the total. The remaining 77% were identified as accidental drug poisoning deaths.

[51] As a result of these changes, 238 million narcotic analgesic prescriptions were written in 2011, including 136.7 million for hydrocodone alone (Manchikanti et al., 2012).

[52] Depression and other forms of mental illnesses increase the experience of pain; pain is associated with more depressive symptoms and the two share many of the same biological pathways (Bair et al. 2003). Given this, persons with depression, dysthymia and generalized anxiety or panic disorders use narcotics at relatively high rates (Sullivan et al., 2006; Brennan Braden et al., 2009), and opioids have been shown to have a palliative effect on mental health problems such as depression and obsessive-compulsive disorder (Bodkin, et al. 1995; Koran, et al., 2005).

(Ruhm, 2000; Ruhm, 2003; Charles & DeCicca, 2008) – but instead with the increased availability of the risky drugs used to treat them. Thus, accidental poisoning appears to be a physical manifestation of what was previously primarily a mental health problem. Consistent with this, home prices are protective accidental poisoning mortality and appear to explain a portion of both the average and secular trend in the macroeconomic effect.

These findings are not only relevant for our understanding of the mechanisms by which health is produced but also for measuring the size and effects of business cycle fluctuations. For instance, Egan et al. (2013) argue that procyclical patterns of mortality imply that business cycle fluctuations are milder than they appear when using standard GDP measures. However, the analysis above suggests that this may no longer be true, at least for the United States.

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Table 1: Estimated Macroeconomic Effects on Total Mortality Using Different Specifications

Regressor	**(1a)**	**(1b)**	**(2a)**	**(2b)**
State Time Trends Excluded				
Unemployment Rate	-0.0024**	-0.0034*	-0.0021**	-0.0020
	(0.0011)	(0.0020)	(0.0010)	(0.0018)
Unemployment × Trend		0.0030		-0.0003
		(0.0040)		(0.0039)
State Time Trends Included				
Unemployment Rate	-0.0037***	-0.0051***	-0.0030***	-0.0054***
	(0.0007)	(0.0013)	(0.0009)	(0.0011)
Unemployment × Trend		0.0037		0.0062***
		(0.0027)		(0.0018)
Population Weights	No	No	Yes	Yes

Note: Dependent variable is the natural log of the total mortality rate in the state, obtained from the *Compressed Mortality Files*, for the period 1976 to 2009 (n=1,734). Table shows the coefficient on the state unemployment rate and (where specified) the interaction between the unemployment rate and a time trend that takes the value zero in 1976 and one in 2009. Observations are weighted by the state population in the third and fourth columns. The regressions also include vectors of state and year dummy variables, state-specific linear time trends (in the second panel) and controls for the share of the state population who are: female, nonwhite, Hispanic, and aged <1, 1-19, 45-54, 55-64, 65-74, 75-84 and ≥85 years old. Robust standard errors, clustered at the state level, are shown in parentheses. *** p<0.01, ** p<0.05, * p<0.1.

Table 2: Estimated Macroeconomic Effects on Specific Sources of Mortality

Type of Mortality	(a) Unemployment Rate	(b) Unemployment Rate	(b) Unemployment × Trend
All	-0.0030**	-0.0054***	0.0062***
	(0.0009)	(0.0011)	(0.0018)
Sex-Specific			
Males	-0.0026**	-0.0052***	0.0068***
	(0.0011)	(0.0014)	(0.0025)
Females	-0.0033***	-0.0054***	0.0054***
	(0.0007)	(0.0010)	(0.0017)
Age-Specific (Years)			
<45	-0.0045	-0.0133***	0.0226***
	(0.0035)	(0.0043)	(0.0062)
45-54	0.0007	-0.0021	0.0071
	(0.0017)	(0.0029)	(0.0063)
55-64	-0.0003	-0.0026*	0.0058*
	(0.0009)	(0.0014)	(0.0030)
65-74	-0.0037***	-0.0048***	0.0027
	(0.0005)	(0.0009)	(0.0022)
75-84	-0.0028***	-0.0052***	0.0062**
	(0.0010)	(0.0010)	(0.0029)
≥85	-0.0031**	-0.0076***	0.0116***
	(0.0013)	(0.0014)	(0.0041)
Cause-Specific			
Diseases	-0.0026***	-0.0045***	0.0047**
	(0.0009)	(0.0011)	(0.0018)
External Sources	-0.0068***	-0.0187***	0.0301***
	(0.0024)	(0.0032)	(0.0067)

Note: Dependent variable is the natural log of the specified mortality rate in the state, obtained from the *Compressed Mortality Files*, for the period 1976 to 2009 (n=1,734). Observations are weighted by the state population. Table shows the coefficient on the state unemployment rate and, in model (b), the interaction between the unemployment rate and a time trend that takes the value zero in 1976 and one in 2009. The regressions also include vectors of state and year dummy variables, state-specific linear time trends, and controls for the share of the state population who are: female, nonwhite, Hispanic, and aged <1, 1-19, 45-54, 55-64, 65-74, 75-84 and ≥85 years old. Robust standard errors, clustered at the state level, are shown in parentheses. *** $p<0.01$, ** $p<0.05$, * $p<0.1$.

Table 3: Estimated Macroeconomic Effects on Cause-Specific Mortality

Cause of Death	(a) Unemployment Rate	(b) Unemployment Rate	Unemployment × Trend
Diseases	**-0.0026*** (0.0009)**	**-0.0045*** (0.0011)**	**0.0047** (0.0018)**
Cardiovascular Disease	-0.0038*** (0.0012)	-0.0044** (0.0017)	0.0015 (0.0038)
Cancer	0.0015** (0.0006)	-0.0009 (0.0010)	0.0063*** (0.0021)
Other Diseases	-0.0048*** (0.0017)	-0.0062*** (0.0022)	0.0035 (0.0055)
External Causes	**-0.0068*** (0.0024)**	**-0.0187*** (0.0032)**	**0.0301*** (0.0067)**
Transport Accidents	-0.0263*** (0.0035)	-0.0320*** (0.0045)	0.0145** (0.0070)
Other Accidents	-0.0066 (0.0046)	-0.0245*** (0.0053)	0.0454*** (0.0116)
Suicides	0.0147*** (0.0045)	0.0088 (0.0075)	0.0150 (0.0107)
Homicides	-0.0108* (0.0062)	-0.0249*** (0.0084)	0.0361* (0.0204)
All Other Accidents	**-0.0066 (0.0046)**	**-0.0245*** (0.0053)**	**0.0454*** (0.0116)**
Falls	-0.0164*** (0.0056)	-0.0206** (0.0100)	0.0105 (0.0170)
Drowning/Submersion	-0.0068 (0.0060)	-0.0148* (0.0088)	0.0203 (0.0155)
Smoke/Fire/Flames	-0.0173*** (0.0057)	-0.0296*** (0.0099)	0.0314 (0.0242)
Poisoning/Noxious	0.0431* (0.0231)	-0.0118 (0.0344)	0.1399*** (0.0520)

Note: Dependent variable is the natural log of the specified mortality rate in the state, obtained from the *Compressed Mortality Files*, for the period 1976 to 2009 (n=1,734). Observations are weighted by the state population. Table shows the coefficient on the state unemployment rate and, in model (b), the interaction between the unemployment rate and a time trend that takes the value zero in 1976 and one in 2009. The regressions also include vectors of state and year dummy variables, state-specific linear time trends, and controls for the share of the state population who are: female, nonwhite, Hispanic, and aged <1, 1-19, 45-54, 55-64, 65-74, 75-84 and ≥ 85 years old. Robust standard errors, clustered at the state level, are shown in parentheses. *** $p<0.01$, ** $p<0.05$, * $p<0.1$.

Table 4: Decomposition of Predicted Effect of Macroeconomics Conditions on Changes in Deaths

Source of Death	Share of Deaths: 1976-80 (a)	Share of Deaths: 2005-09 (b)	Δ in Deaths Due to: Δ in Shares (c)	Δ in Deaths Due to: Δ in Coefficients (d)
All Deaths	1.00000	1.00000		**13,283**
Age of Death (Years)				
<45	0.11378	0.07950	**1,003**	**3,851**
45-54	0.07313	0.07597	-12	1,164
55-64	0.15152	0.11823	**181**	**1,484**
65-74	0.23418	0.16222	818	950
75-84	0.25886	0.26935	**-124**	**3,581**
≥85	0.16821	0.29464	**-2,139**	**7,317**
Total Explained			-273	18,347
% Explained			-2.1%	138.1%
Cause of Death				
CVD	0.50297	0.33350	1,800	1,088
Cancer	0.20548	0.23062	**-34**	**3,094**
Other	0.21011	0.36268	-2,214	2,691
External	0.08144	0.07320	**338**	**4,732**
Total Explained			-110	11,606
% Explained			-0.8%	87.4%

Note: Predicted changes are for a 1% point increase in the unemployment rate. The total average annual number of deaths is 1,928,101 for 1976-1980 and 2,441,428 for 2005-2009. Entries in bold are those where the unemployment-time trend interaction is significant at the 0.1 level or better. The change in deaths due to changes in shares and coefficients are calculated as $\gamma_1(\pi_2-\pi_1)]D_2$ and $(\gamma_2-\gamma_1)\pi_2 D_2$, respectively, for γ_τ, π_τ and D_τ indicating the predicted unemployment coefficient (detailed below), share of deaths and total number of deaths in period τ, where τ=1 in 1976-80 and τ=2 in 2005-2009. The total change in deaths is $(\gamma_2-\gamma_1)\pi_1 D_2$. γ_1 and γ_2 are the predicted unemployment coefficients in 1978 and 2007, midpoint of the 1976-80 and 2005-09 periods. These are calculated as the unemployment main effect coefficient plus the unemployment-trend interaction evaluated in 1978 and 2007.

Table 5: Detailed Decomposition of Cancer and External Causes of Death

Source of Death	Share of Deaths 1976-80 (a)	Share of Deaths 2005-09 (b)	Δ in Deaths Due to: Δ in Shares (c)	Δ in Deaths Due to: Δ in Coefficients (d)
All Deaths	1.00000	1.00000		**13,283**
All Cancers	0.20548	0.23062	**-34**	**3,094**
Digestive	0.05496	0.05323	-1	289
Lung	0.05165	0.06653	**53**	**937**
Breast	0.01798	0.01686	**6**	**448**
Genital	0.02341	0.02238	9	380
Lymphatic	0.01902	0.02256	**-20**	**482**
Other	0.03845	0.04906	-25	519
Total Explained			23	3,057
% Explained			0.7%	98.8%
All External	0.08144	0.07321	**339**	**4,732**
Transport Accident	0.02865	0.01847	**774**	**576**
Other Accident	0.02535	0.03200	**-352**	**3,119**
Suicide	0.01420	0.01421	0	458
Homicide	0.01108	0.00751	198	581
Total Explained			620	4,735
% Explained			13.1%	100.1%
All Other Accidents	0.02535	0.03200	**-352**	**3,119**
Falls	0.00706	0.00917	-102	207
Drowning	0.00302	0.00145	52	63
Fires	0.00318	0.00125	131	84
Poisoning	0.00260	0.01179	**-75**	**3,536**
Total Explained			5	3,891
% Explained			0.2%	124.7%

Note: See note on Table 4. The average annual number of cancer deaths is 396,179 during 1976-80 and 563,034 during 2005-2009. Average number of external deaths is 157,033 and 178,709. Average number of other (non-transport) accidental deaths is 48,875 and 78,115.

Table 6: Estimated Macroeconomic Effects on Age-Specific Mortality From Specified Causes

Regressor	<45 Years Old		45-74 Years Old		≥75 Years Old	
	(a)	(b)	(a)	(b)	(a)	(b)
Cancer Deaths						
Unemployment Rate	-0.0011 (0.0021)	-0.0088*** (0.0027)	0.0010 (0.0007)	-0.0003 (0.0010)	0.0039*** (0.0010)	-0.0000 (0.0018)
Unemployment × Trend		0.0196*** (0.0045)		0.0033 (0.0026)		0.0100** (0.0038)
Deaths from External Causes						
Unemployment Rate	-0.0099*** (0.0031)	-0.0219*** (0.0048)	-0.0009 (0.0025)	-0.0126*** (0.0033)	-0.0084*** (0.0030)	-0.0162*** (0.0053)
Unemployment × Trend		0.0306*** (0.0093)		0.0297*** (0.0073)		0.0200 (0.0123)
Deaths from Poisoning Accidents						
Unemployment Rate	0.0397 (0.0274)	-0.0273 (0.0392)	0.0468** (0.0231)	0.0124 (0.0363)	0.0061 (0.0121)	0.0128 (0.0180)
Unemployment × Trend		0.1704*** (0.0569)		0.0877 (0.0527)		-0.0171 (0.0373)

Note: See note on Table 3. Mortality rates refer to the specified cause of deaths for <45, 45-74, or ≥75 years olds. Sample size is 1,734, except for accidental poisoning deaths, were the sample sizes are 1,733, 1,715 and 1,600 for <45, 45-74, or ≥75 years olds.

Table 7: Detailed Decomposition of Age-Specific Mortality from Specified Causes

Source of Death	Share of Deaths		Δ in Deaths Due to:	
	1976-80	2005-09	Δ in Shares	Δ in Coefficients
	(a)	(b)	(c)	(d)
All Deaths	1.00000	1.00000		**13,283**
All Cancers	0.20548	0.23062	**-34**	**3,094**
Age: <45	0.01136	0.00820	**58**	**345**
Age: 45-74	0.13111	0.11995	3	837
Age: ≥75	0.06299	0.10247	**57**	**2,193**
All External	0.08144	0.07320	**339**	**4,732**
Age: <45	0.04916	0.03444	**722**	**2,264**
Age: 45-74	0.02294	0.02528	**-62**	**1,613**
Age: ≥75	0.00924	0.01345	-155	576
Poisoning	0.00260	0.01179	**-75**	**3,536**
Age: <45	0.00168	0.00652	**-201**	**2,385**
Age: 45-74	0.00075	0.00503	185	946
Age: ≥75	0.00017	0.00023	2	-8

Note: See note on Table 4. The average annual number of cancer, external and accidental poisoning deaths are 396,179, 157,033 and 5,008 during 1976-80 and 563,034, 178,709 and 28,774 during 2005-2009.

Table 8: First-Stage Estimates of the Effect of Macroeconomic Conditions on Potential Determinants of Mortality

Regressor	(a)	(b)	(a)	(b)	(a)	(b)
	Manufacturing Job Share		**Construction Job Share**		**Farming Job Share**	
Unemployment Rate	-0.0023***	-0.0032***	-0.0021***	-0.0016***	0.0003***	0.0004**
	(0.0003)	(0.0006)	(0.0003)	(0.0003)	(0.0001)	(0.0002)
Unemployment × Trend		0.0022*		-0.0013***		-0.0002
		(0.0011)		(0.0005)		(0.0003)
	ln(Per Capita Income)		**ln(Median Home Price)**		**Annual Weeks Worked**	
Unemployment Rate	-0.0117***	-0.0071***	-0.0358***	-0.0070	-0.3575***	-0.3554***
	(0.0012)	(0.0016)	(0.0036)	(0.0073)	(0.0192)	(0.0374)
Unemployment × Trend		-0.0117***		-0.0732***		-0.0055
		(0.0024)		(0.0197)		(0.0859)
	Usual Work Hours/Week		**ln(Vehicle Miles)**		**Health Insurance**	
Unemployment Rate	-0.2535***	-0.2361***	-0.0103***	-0.0150***	-0.0038***	-0.0036**
	(0.0202)	(0.0259)	(0.0024)	(0.0031)	(0.0010)	(0.0016)
Unemployment × Trend		-0.0441		0.0120*		-0.0005
		(0.0630)		(0.0068)		(0.0026)
	Private Health Ins.		**Public Health Ins.**		**Current Smoker**	
Unemployment Rate	-0.0062***	-0.0055***	0.0011	0.0021	0.0004	-0.0031*
	(0.0010)	(0.0019)	(0.0010)	(0.0015)	(0.0014)	(0.0017)
Unemployment × Trend		-0.0018		-0.0022		0.0069
		(0.0033)		(0.0020)		(0.0044)
	ln(BMI)		**Obese**		**Obese: Class 3**	
Unemployment Rate	0.0001	0.0002	-0.0008	-0.0009	0.0002	0.0003
	(0.0003)	(0.0005)	(0.0005)	(0.0010)	(0.0002)	(0.0003)
Unemployment × Trend		-0.0003		0.0000		-0.0001
		(0.0009)		(0.0017)		(0.0006)

Note: Dependent variables are as specified above the coefficient estimates. All specifications are estimated by least squares. Observations are weighted by the state population. Table shows the coefficient on the state unemployment rate and, in model (b), the interaction between the unemployment rate and a time trend that takes the value zero in the first year data on the dependent variable is available and one in 2009. The regressions also include vectors of state and year dummy variables, state-specific linear time trends, and controls for the share of the state population who are: female, nonwhite, Hispanic, and aged <1, 1-19, 45-54, 55-64, 65-74, 75-84 and ≥85 years old. The first data year is 1976 except for body weight and smoking (1987) and health insurance (1988). Sample sizes are 1,734 except for construction and manufacturing share of jobs (1,730 and 1,732), health insurance (1,173) and body weight/smoking (1,115). Robust standard errors, clustered at the state level, are shown in parentheses. *** p<0.01, ** p<0.05, * p<0.1.

Table 9: Estimated Effects on Cancer and Accidental Poisoning Mortality With and Without Controls for Home Prices and Income

	(a)	(b)	(c)	(d)	(e)	(f)
Cancer						
Unemployment Rate	0.0015**	-0.0009	0.0006	-0.0011	0.0004	-0.0012
	(0.0006)	(0.0010)	(0.0006)	(0.0010)	(0.0008)	(0.0010)
Unemployment × Trend		0.0063***		0.0047**		0.0046**
		(0.0021)		(0.0020)		(0.0020)
Ln(Median Home Price)			-0.0244***	-0.0207**	-0.0221***	-0.0190***
			(0.0078)	(0.0080)	(0.0067)	(0.0070)
Ln(Per Capita Income)					-0.0277	-0.0214
					(0.0449)	(0.0453)
Accidental Poisoning						
Unemployment Rate	0.0431*	-0.0118	0.0278	-0.0142	0.0313	-0.0105
	(0.0231)	(0.0344)	(0.0228)	(0.0343)	(0.0278)	(0.0369)
Unemployment × Trend		0.1399***		0.1152*		0.1186**
		(0.0520)		(0.0575)		(0.0585)
Ln(Median Home Price)			-0.4268**	-0.3367*	-0.4601*	-0.3808
			(0.1789)	(0.1955)	(0.2324)	(0.2459)
Ln(Per Capita Income)					0.4001	0.5612
					(1.2186)	(1.2240)

See note on Table 3.

Figure 1: Unemployment Coefficients for Total Mortality Using Different Analysis Samples

Figure 1A. Sample Begins in 1976 and Continues through Specified Year

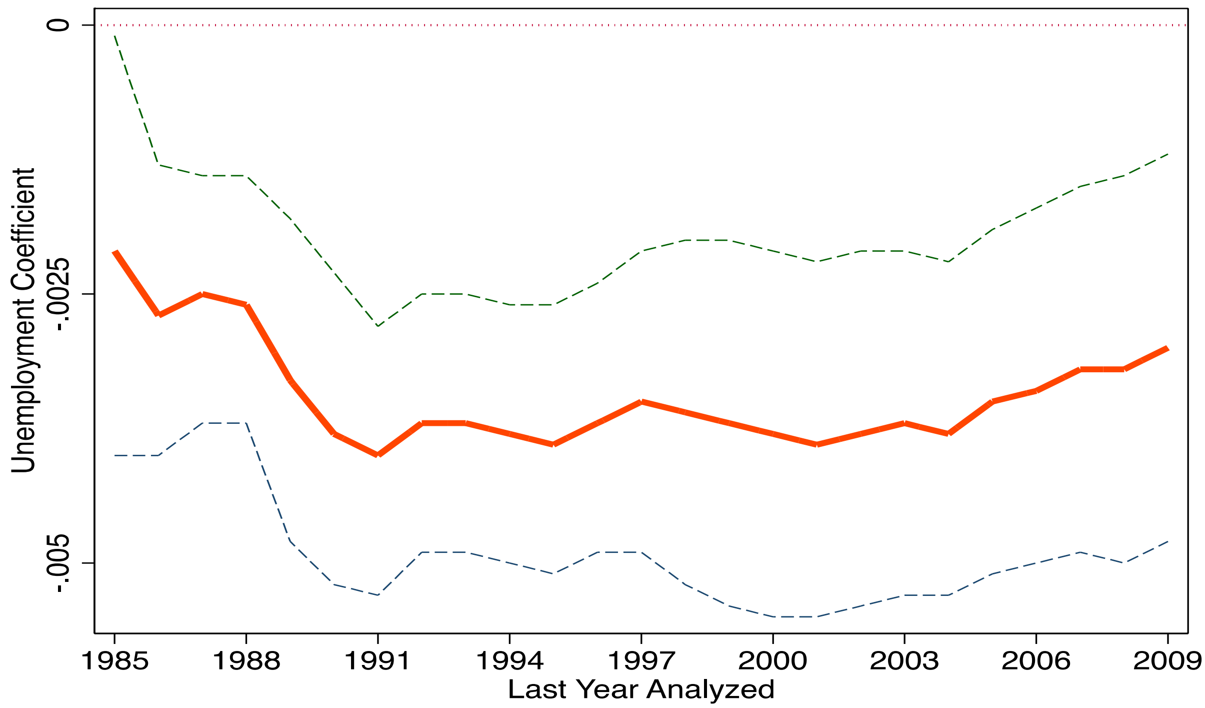


Figure 1B. Sample Begins in Specified Year and Continues through 2009

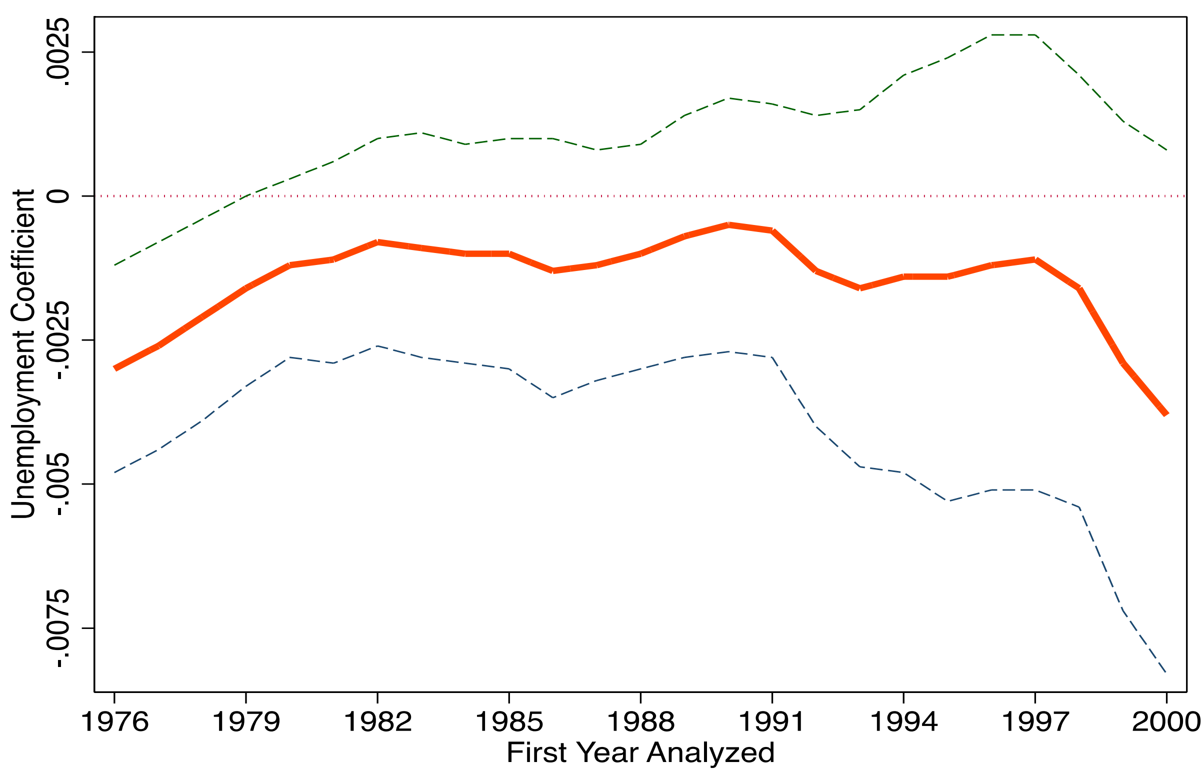
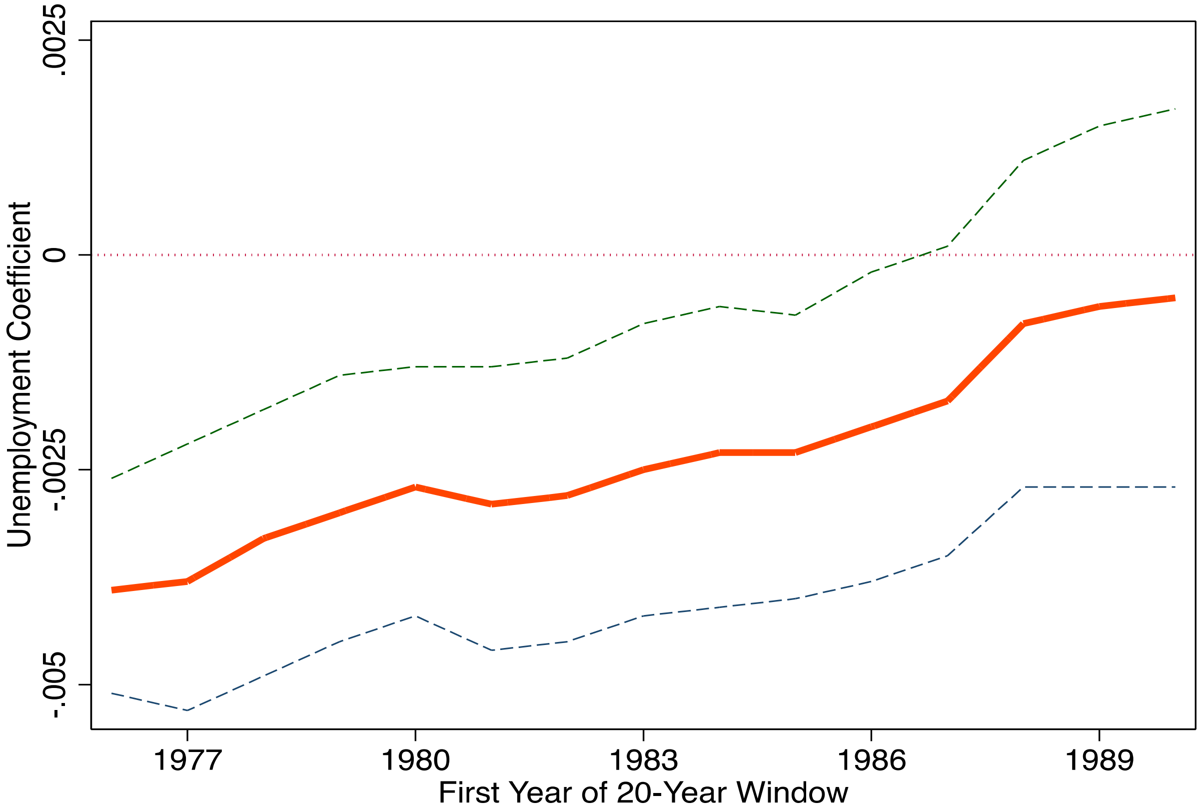


Figure 1C. Analysis Sample Covers 20-Year Period



Note: Figure shows estimates obtained from models where the dependent variable is the national log of the total mortality rate in the state. The solid line indicates the estimated unemployment coefficient and the dotted lines show the 95-percent confidence interval calculated from robust standard errors clustered at the state level. The regressions also include vectors of state and year dummy variables, state-specific linear time trends, and controls for the share of the state population who are: female, nonwhite, Hispanic and aged <1, 1-19, 45-54, 55-64, 65-74, 75-84 and ≥85 years old.

Figure 2: Unemployment Coefficients for Total Mortality Using Different Sample Windows

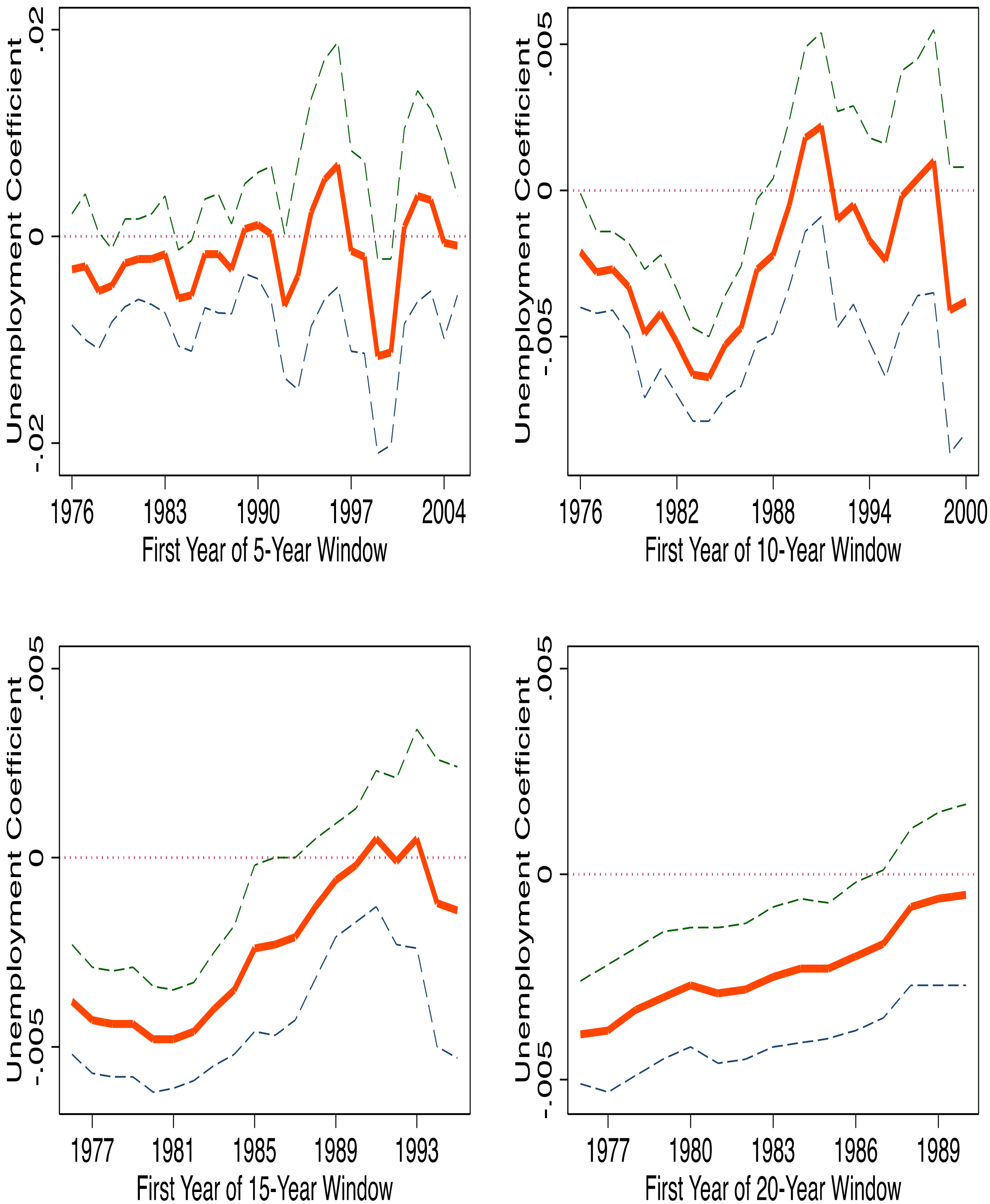


Figure 3: Unemployment Coefficients for Sex-Specific and Age-Specific Mortality, and Major Causes of Death

Figure 3A. Sex-Specific Mortality

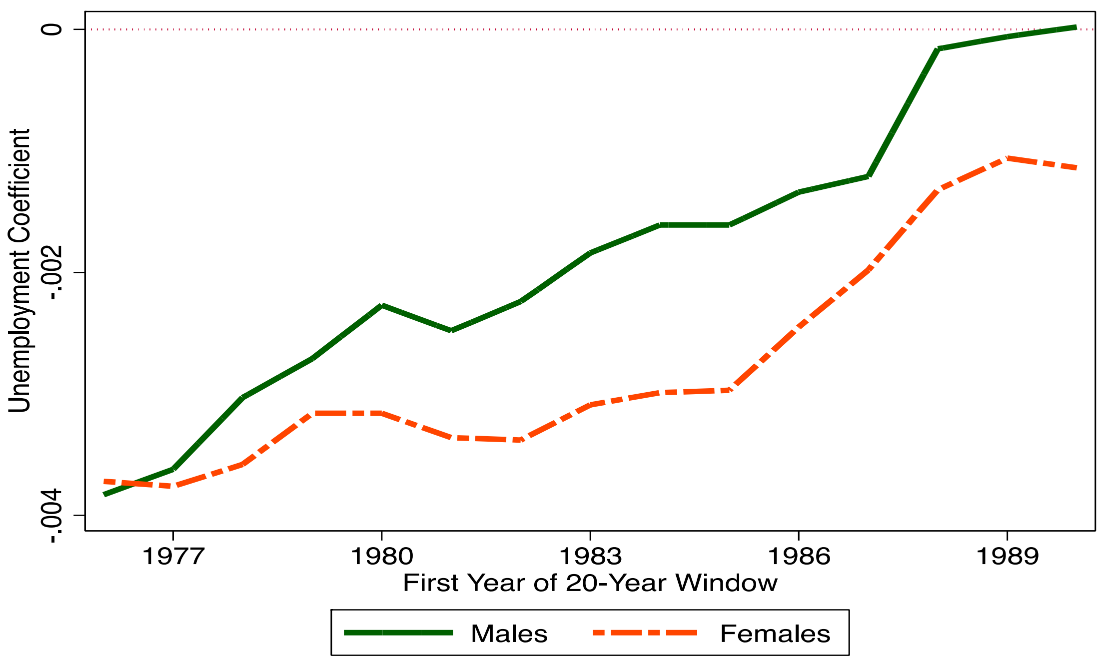


Figure 3B. Age-Specific Mortality

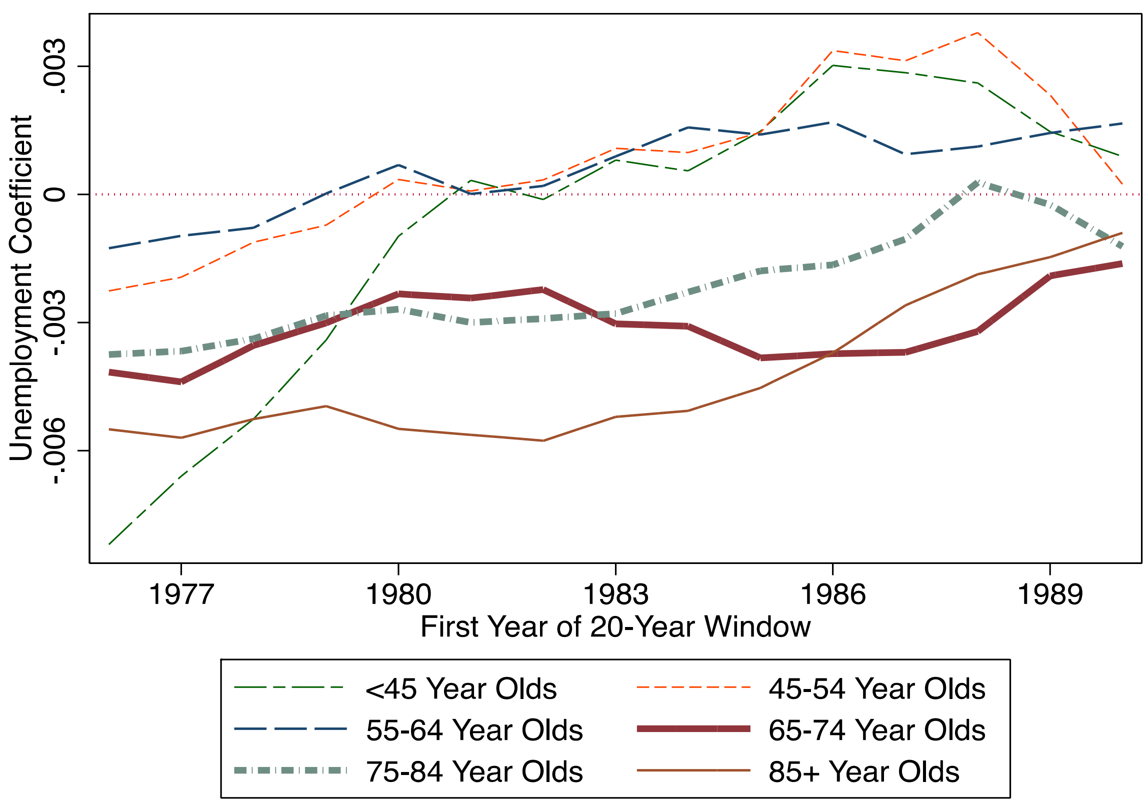


Figure 3C. Cause-Specific Mortality

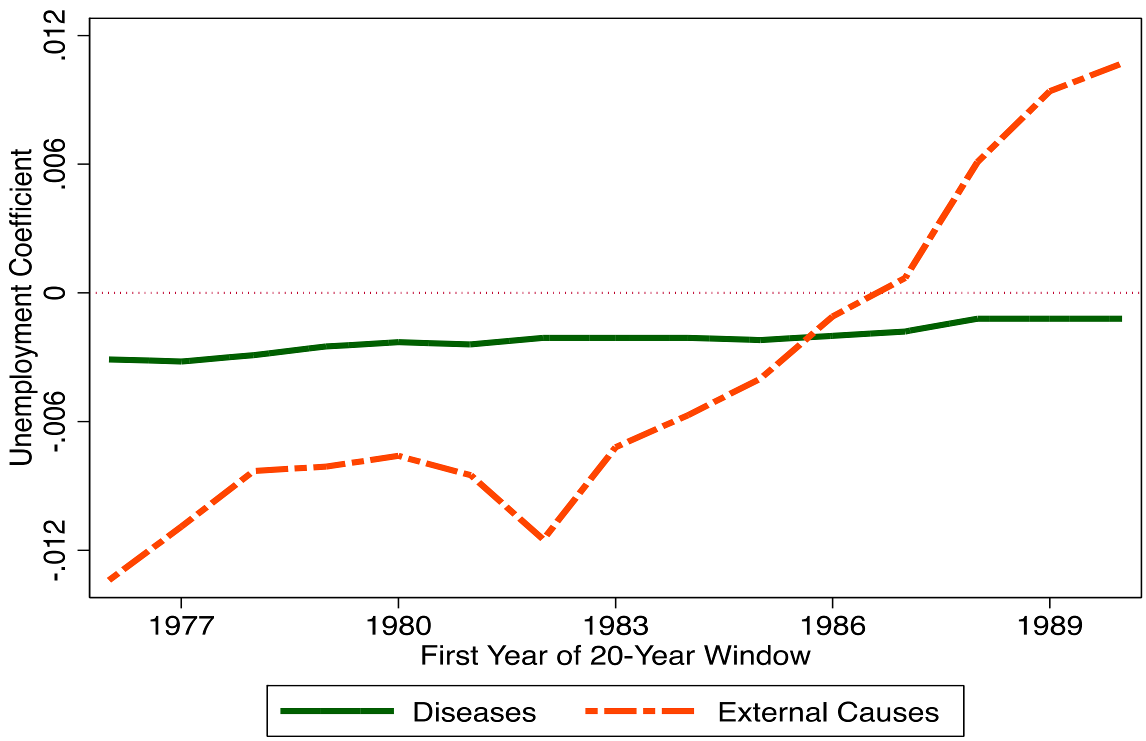


Figure 4: Unemployment Coefficients for Deaths from Specific Diseases and External Causes

Figure 4A. Specific Diseases

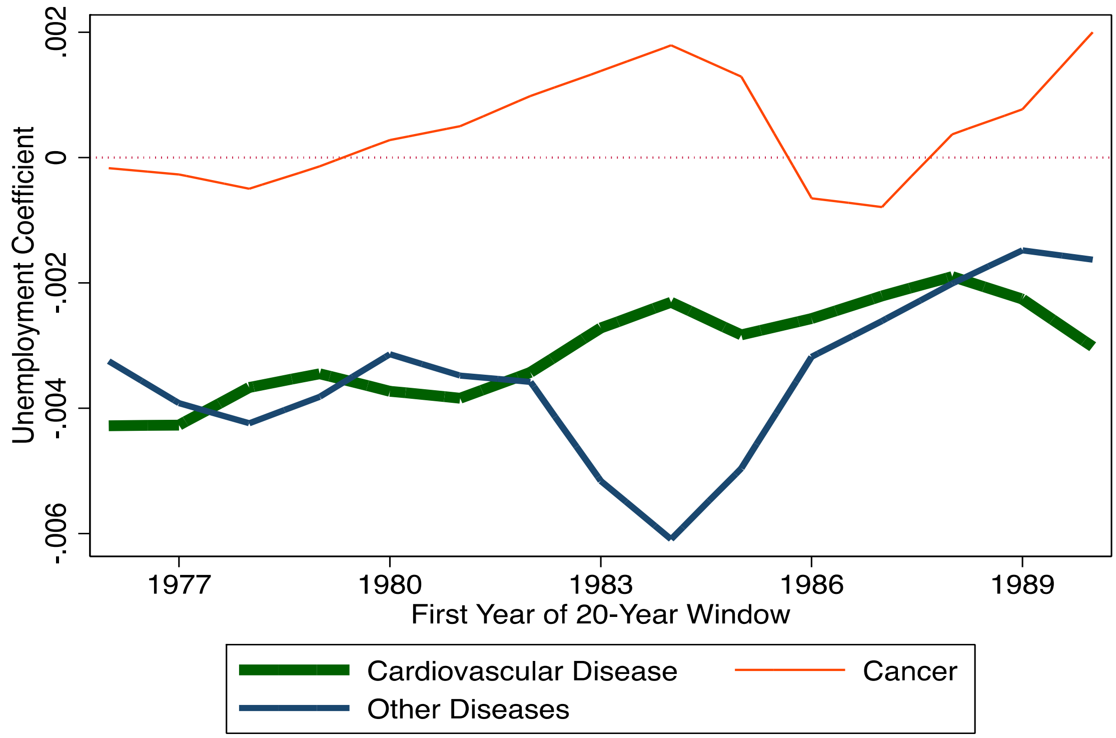


Figure 4B. External Causes

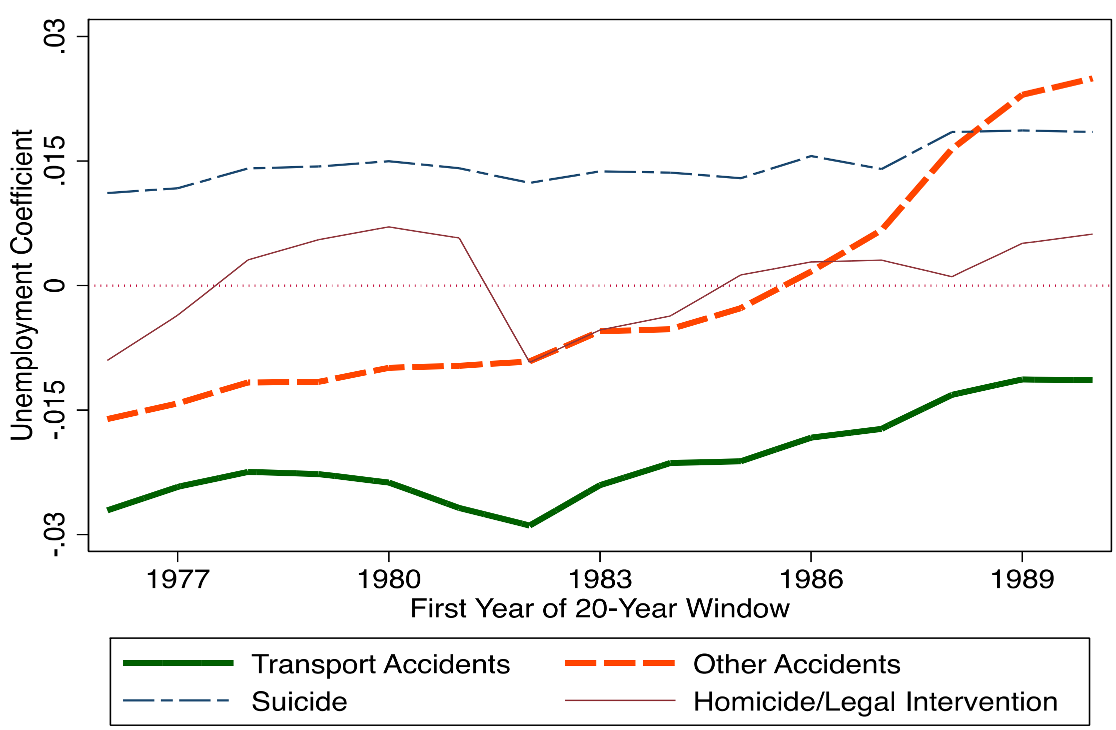


Figure 4C. Other Accidents

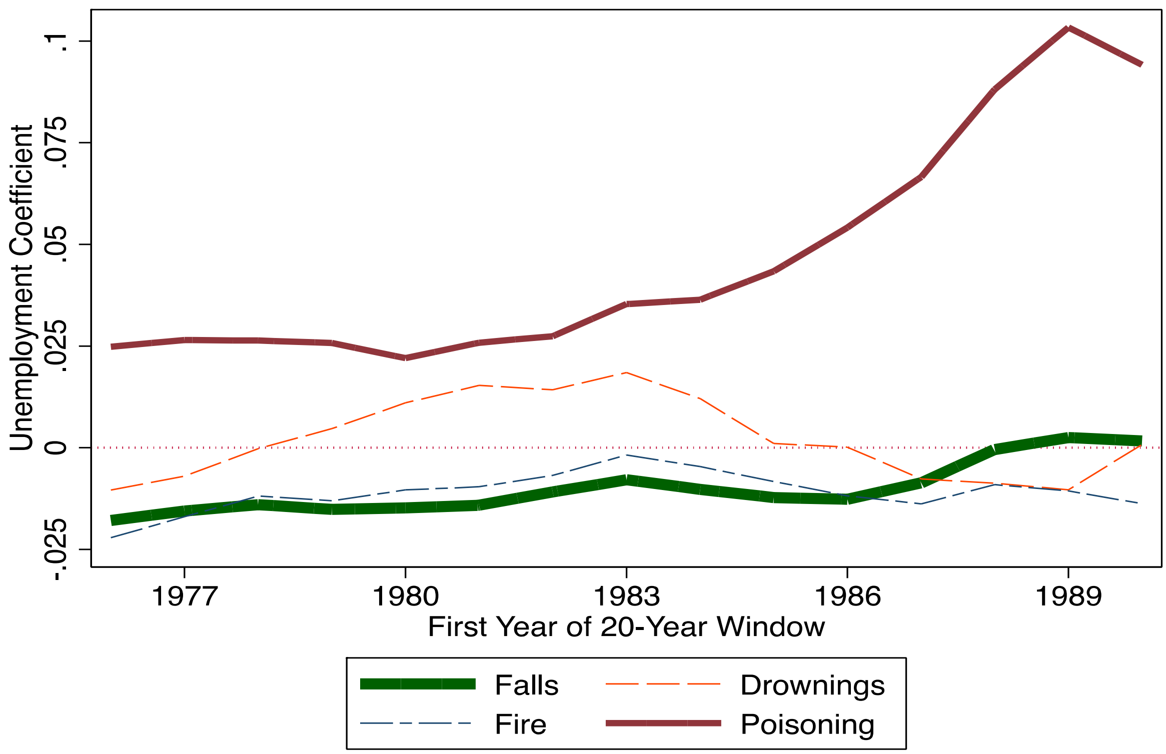
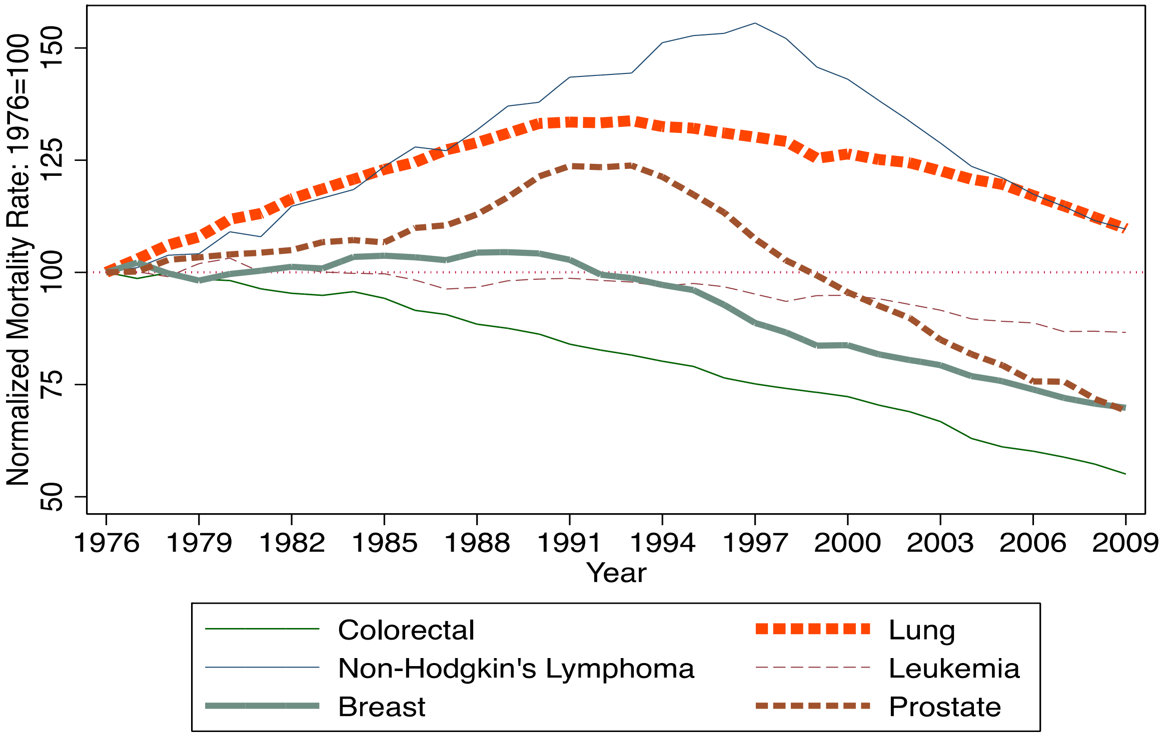
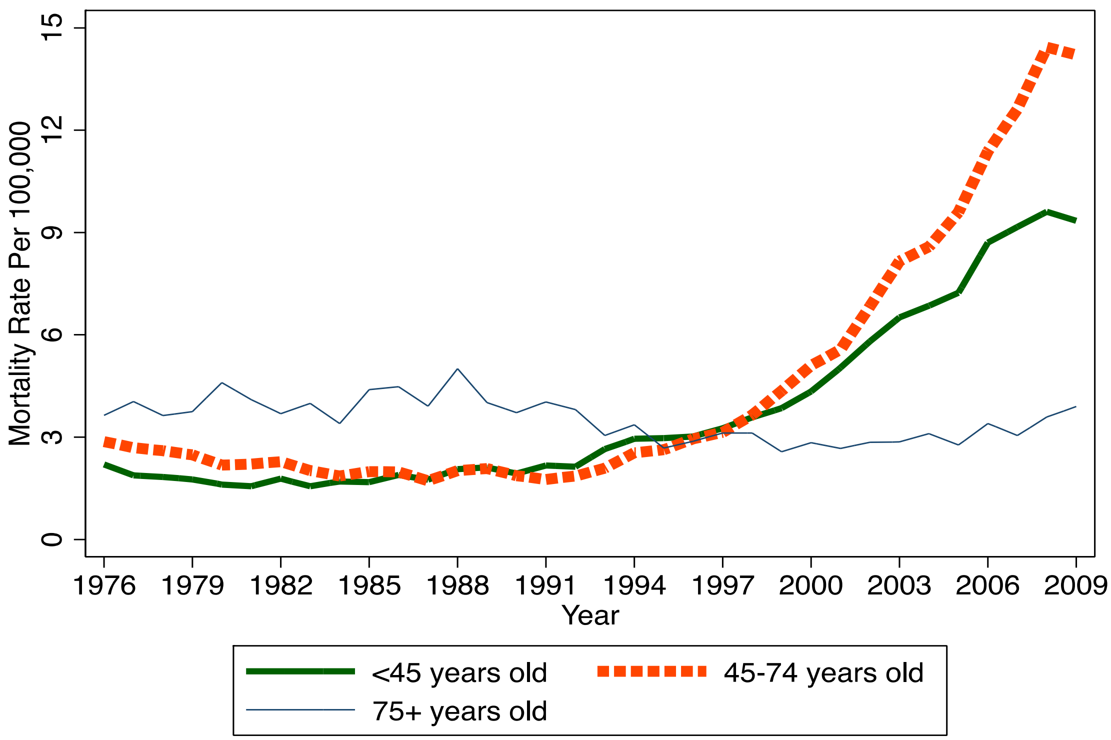


Figure 5: Trends in Age-Adjusted Cancer Mortality Rates



Note: Figure shows age-adjusted mortality rates from the specified type of cancer for both sexes, except for breast and prostate cancer where the rates refer to females and males only. Mortality rates are normalized such that values in 1976 equal 100.

Figure 6: Trends in Accidental Poisoning Mortality, By Age



Appendix A.1: Definitions of Specific Causes of Mortality

Variable	Description	ICD-8 (1976-1978)	ICD-9 (1979-1998)	ICD-10 (1999-2009)
Cancer	Malignant Neoplasms	140-209	140-208	C00-C97
Digestive	Malignant Neoplasms: Digestive Organs & Peritoneum (e.g. stomach, colon, pancreas)	150-159	150-159	C15-C25
Lung	Malignant Neoplasms: Respiratory System	160-163	160-165	C32-C34
Breast	Malignant Neoplasms: Breast	174	174-175	C50
Genital	Malignant Neoplasms: Genital Organs (Cervix, Ovary, Prostate)	180-187	179-187	C53-C61
Lymph	Malignant Neoplasms: Leukemia, Lymphatic/Hematopoietic Tissues	200-209	200-208	C81-C96
Diabetes	Diabetes Mellitus		250	E19-E14
Alzheim	Alzheimer's Disease		290.1, 331	G30
CVD	Major Cardiovascular Diseases	390-448	390-448	I00-I78
Heart	Diseases of the Heart	390-398, 402, 404, 410-429	390-398, 402, 404-429	I00-I09, I11, I13, I20-I51
Stroke	Cerebrovascular Disease	430-438	430-438	I60-I69
Resp	Chronic Lower Respiratory Disease		490-496	J40-J47
Liver	Cirrhosis, Chronic Liver Disease		571	K70-K74
Kidney	Nephritis, Nephrosis, Nephrotic Syndrome		580-589, 593.9	N00-N07, N17-N27
External	External Sources	800-999	800-999	U01-U03, V01-Y98
Transport	Transport Accidents	800-848, 940-941	800-848, 929.0, 929.1	V02-V99, Y85
Nontran	Other (Non-Transport) Accidents	850-939, 942-949	850-928, 929.2-949	W00-X59, Y86
Othacc	Specific Accidents: Falls, Drowning, Fires, Poisoning, Firearms (unintentional)	850-899, 910, 922, 924	850-869, 880-899, 910, 922, 924.1	W00-W19, W32-W34, W65-W74 X40-X49
Falls	Accidents: Falls	880-887	880-888	W00-W19
Drowning	Accidents: Drowning/Submersion	910	910	W65-W74

Fires	Accidents: Smoke/Fire/Flames	890-899	890-899	X00-X09
Poison	Accidents: Poisoning/Noxious Substances	850-879, 924	850-869, 924.1	X40-X49
Suicide	Suicide (Intentional Self-Harm)	950-959	950-959	X60-X84, Y87.0
Homicide	Homicide & Legal Intervention	960-978	960-978	X85-Y09, Y87.1, Y35, Y89.0

Note: Non-Transport Accidents include deaths due to falls, drowning/submersion, smoke/flames/fires, poisoning/exposure to noxious substances, and accidental discharge of firearms. (Other and unspecified nontransport accidents are excluded.)

Table A.2: Descriptive Statistics for Selected Analysis Variables

Variable	Mean	Standard Error
Mortality Rates		
All Deaths	851.5	2.8
Males	893.8	3.3
Females	811.3	2.8
<45 Year Olds	123.3	0.6
45-54 Year Olds	477.8	2.2
55-64 Year Olds	1,118.6	5.2
65-74 Year Olds	2,548.3	9.9
75-84 Year Olds	5,873.1	16.9
≥85 Year Olds	15,072.2	24.9
Major Cardiovascular Disease (CVD)	356.6	1.9
Heart Disease	275.3	1.6
Cerebrovascular Disease (Stroke)	59.8	0.3
Other CVD	21.5	0.1
Malignant Neoplasms (Cancer)	192.5	0.7
Digestive Cancer	46.3	0.2
Lung Cancer	54.3	0.3
Breast Cancer	15.6	0.1
Cancer of Genital Organs	20.6	0.1
Leukemia/Lymphatic Cancer	18.6	0.1
Other Cancers	37.1	0.1
Other Diseases	242.3	1.2
Diabetes	20.8	0.1
Alzheimer’s Disease	12.8	0.2
Chronic Lower Respiratory Disease	37.4	0.2
Cirrhosis/Chronic Liver Disease	10.2	0.1
Kidney Disease	11.7	0.1
External Causes	60.1	0.3
Transport Accidents	18.8	0.1
Other (Non-Transport) Accidents	20.4	0.1
Falls	5.7	4.7E-2
Drowning/Submersion	1.6	2.0E-2
Smoke/Fires/Flame	1.6	2.2E-2
Poison/Noxious Substance	4.4	0.1
Suicide	11.7	0.1
Homicide/Legal Intervention	8.1	0.1
State Characteristics		
% Female	51.1	1.7E-2
% Nonwhite	16.8	0.2

% Hispanic	10.5	0.3
% <1 Year Old	1.5	4.4E-2
% 1-19 Years Old	27.8	0.1
% 45-54 Years Old	11.8	4.8E-2
% 55-64 Years Old	9.2	2.9E-2
% 65-74 Years Old	6.8	2.5E-2
% 75-84 Years Old	4.1	2.0E-2
% ≥85 Year Olds	1.3	9.3E-3
Unemployment Rate (%)	6.2	4.7E-2
Potential Mechanisms		
Manufacturing, % of Jobs	12.8	1.3E-2
Construction, % of Jobs	5.5	2.7E-2
Farming, % of Jobs	2.3	4.4E-2
Personal Income Per Capita (2009 $, 1000's)	33.2	0.2
Median Single Family Home Price (1000's 2009 $)	145.9	1.7
Weeks Worked/Year	38.7	5.4E-2
Usual Weekly Work Hours	33.3	4.4E-2
Highway Miles Driven (millions)	95,987	1,905
% With Health Insurance	80.7	0.4
% With Private Health Insurance	75.9	0.2
% With Public Health Insurance	11.3	0.1
% Current Smoker	24.0	0.1
Body Mass Index (BMI)	26.3	3.0E-2
% Obese (BMI≥30)	18.8	0.2
% Obese Class 3 (BMI≥40)	2.1	3.5E-2

Note: Data are for 1976-2009. Mortality rates are per 100,000 population. Other diseases refer to deaths not due to cardiovascular disease, cancer or external causes. Obervations are weighted by state population. Due to non-comparability between ICD-8 and ICD-9 codes, data for liver, diabetes, kidney, Alzheimer's and chronic lower respiratory disease starts in 1979. During 1979-1998, pre-senile dementia (ICD-9 code 290.1) classified with Alzheimer's disease and end-stage renal disease (ICD-9 code 593.1) classified with kidney disease (Nephritis/Nephrosis/Nephrotic Syndrome). Data on annual weeks worked, weekly work hours and health insurance refer to 25-60 year olds. Information on body weight and smoking begins in 1987, with missing data for some states in early years; that on health insurance begins in 1988.

Data Sources. Mortality rates and age/sex/race-ethnicity population shares: CDC *Compressed Mortality Files* (http://www.cdc.gov/nchs/data_access/cmf.htm); unemployment rates: DOL *Local Area Unemployment Statistics* (LAUS) database (http://www.bls.gov/lau/data.htm); personal income, manufacturing/construction/farming percent of total full-time and part-time jobs: *Bureau of Economic Analysis* (www.bea.gov); health insurance, weeks worked/year, usual weekly work hours, *March Current Population Survey*; *Integrated Public Use Microdata Series* (http://cps.ipums.org/cps/); housing prices, *Freddie Mac House Price Index* (www.freddiemac.com/finance/fmhpi/) and U.S. Census Bureau (www.census.gov/hhes/www/housing/census/historic/values.html); highway miles driven, Federal Highway Administration *Highway Statistics* (www.fhwa.dot.gov/policyinformation/statistics.cfm); smoking, BMI, and obesity, *Behavioral Risk Factor Surveillance System* (www.cdc.gov/brfss).

Table A.3: Sources of Death by Time Period

	1976-1992		1993-2009	
Source of Death	**#**	**%**	**#**	**%**
All Deaths	*2,045,826*	*100.0%*	*2,384,322*	*100.0%*
Males	1,085,240	53.0%	1,185,229	49.7%
Females	960,586	47.0%	1,199,093	50.3%
Age of Death (Years)				
<45	221,241	10.8%	208,396	8.7%
45-54	126,010	6.2%	163,765	6.9%
55-64	277,909	13.6%	256,557	10.8%
65-74	470,449	23.0%	434,750	18.2%
75-84	551,326	26.9%	672,339	28.2%
≥85	398,262	19.5%	648,164	27.2%
Cause of Death				
Cardiovascular	*958,913*	*46.9%*	*896,349*	*37.6%*
Heart	745,073	36.4%	687,369	28.8%
Cerebrovascular	158,645	7.8%	152,249	6.4%
Other CVD	55,194	2.7%	56,731	2.4%
Cancer	*450,758*	*22.0%*	*550,779*	*23.1%*
Digestive	113,957	5.6%	127,018	5.3%
Lung	122,898	6.0%	159,600	6.7%
Breast	39,091	1.9%	42,151	1.8%
Genital	50,305	2.5%	56,826	2.4%
Lymph	41,826	2.0%	55,090	2.3%
Other	82,681	4.0%	110,093	4.6%
Other Disease	*484,811*	*23.7%*	*775,844*	*32.5%*
Diabetes	39,705	1.9%	67,464	2.8%
Alzheimer’s	9,996	0.5%	53,275	2.2%
Chronic Lower Respiratory	73,293	3.5%	119,854	5.0%
Chronic Liver/Cirrhosis	27,187	1.3%	26,920	1.1%
Kidney	22,150	1.1%	37,971	1.6%
External Causes	*151,344*	*7.4%*	*161,335*	*6.8%*
Transport Accidents	51,465	2.5%	46,247	1.9%
Other Accidents	45,284	2.2%	60,961	2.6%
Falls	12,579	0.6%	17,237	0.7%
Drowning/Submersion	4,855	0.2%	3,532	0.1%
Smoke/Fires/Flame	5,178	0.3%	3,364	0.1%
Poison/Noxious Substance	5,421	0.3%	17,526	0.7%
Suicide	29,076	1.4%	31,986	1.3%
Homicide	22,198	1.1%	19,627	0.8%

Note: Table shows average deaths per year for specified age group or from specified cause. Data for diabetes, Alzheimer’s, lower respiratory, liver and kidney disease starts in 1979. Proportions of all deaths for these diseases in the earlier period refers to 1979-1992 rather than 1976-1992.

Table A.4: Additional Results for Total Mortality Regressions

Regressor	(a)	(b)
Unemployment rate	-0.00197	-0.00540***
	(0.00184)	(0.00107)
Unemployment × Trend	-0.00034	0.00619***
	(0.00393)	(0.00182)
Share <1 year old	8.96898	2.46405
	(5.75513)	(1.63251)
Share 1-19 years old	-0.70308	-0.86896***
	(0.50385)	(0.22800)
Share 45-54 years old	-0.06659	0.65966
	(0.99522)	(0.48782)
Share 55-64 years old	3.68763***	2.63094***
	(0.89545)	(0.61171)
Share 65-74 years old	2.61528***	2.59912***
	(0.87990)	(0.53425)
Share 75-84 years old	5.14847***	8.13035***
	(1.42735)	(1.19943)
Share >=85 years old	8.84547***	13.87427***
	(2.53111)	(3.21083)
Share female	1.23512	-4.57840***
	(3.38398)	(0.94855)
Share nonwhite	-0.92450**	1.28477***
	(0.43360)	(0.36837)
Share Hispanic	-0.18030	0.35460
	(0.22049)	(0.37904)
State Trends Included	No	Yes

Note: Table shows additional parameter estimates for model (2b) in Table 1, without and without the inclusion of time trends.

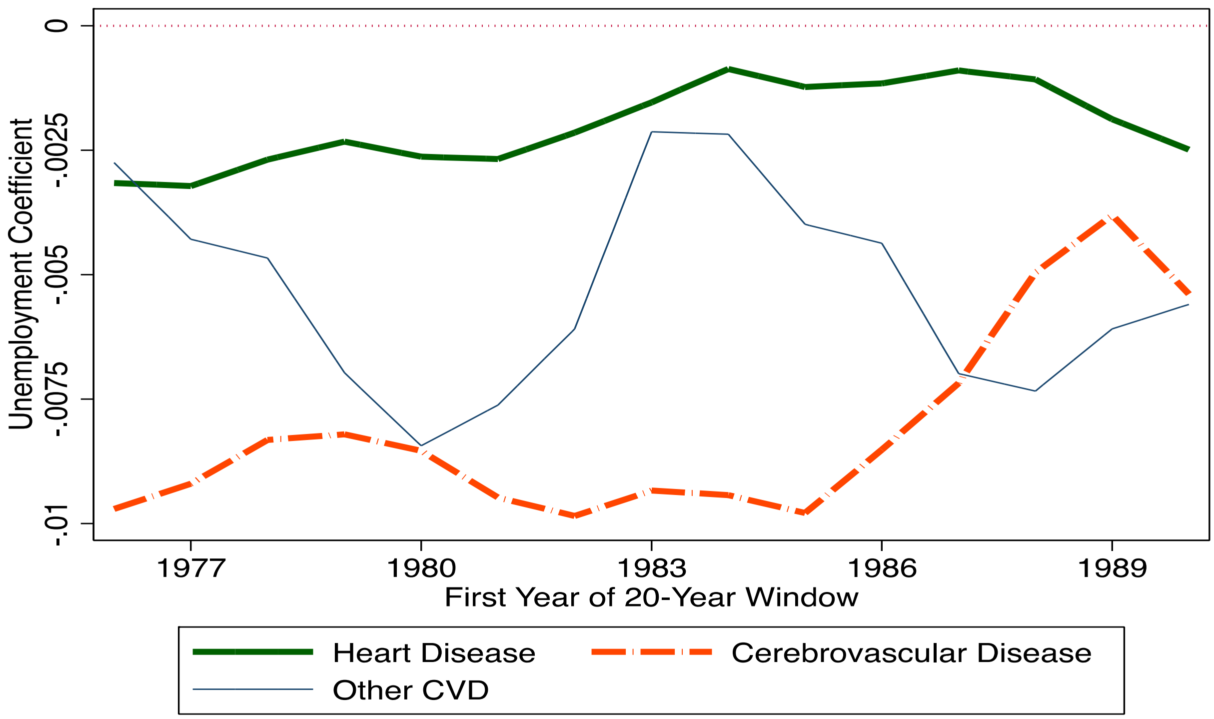
Table A.5: Estimated Macroeconomic Effects on Deaths From Specific Diseases

	(a)	(b)	
Cause of Death	**Unemployment Rate**	**Unemployment Rate**	**Unemployment × Trend**
All Diseases	**-0.0026*** (0.0009)**	**-0.0045*** (0.0011)**	**0.0047** (0.0018)**
Cardiovascular Disease	**-0.0038*** (0.0012)**	**-0.0044** (0.0017)**	**0.0015 (0.0038)**
Heart Disease	-0.0021* (0.0012)	-0.0023 (0.0018)	0.0005 (0.0047)
Cerebrovascular Disease	-0.0114*** (0.0025)	-0.0112*** (0.0032)	-0.0005 (0.0068)
Other CVD	-0.0026 (0.0028)	-0.0048 (0.0046)	0.0057 (0.0111)
Cancer	**0.0015** (0.0006)**	**-0.0009 (0.0010)**	**0.0063*** (0.0021)**
Digestive	0.0011 (0.0011)	0.0001 (0.0014)	0.0025 (0.0030)
Lung	0.0036*** (0.0009)	0.0011 (0.0017)	0.0066* (0.0037)
Breast	0.0017 (0.0013)	-0.0031 (0.0025)	0.0124** (0.0060)
Genital	-0.0010 (0.0016)	-0.0041* (0.0024)	0.0079 (0.0051)
Leukemia/Lymphatic	0.0010 (0.0010)	-0.0029 (0.0021)	0.0100** (0.0043)
Other Cancer	0.0007 (0.0016)	-0.0013 (0.0027)	0.0049 (0.0050)
Other Diseases	**-0.0035* (0.0021)**	-0.0042 (0.0028)	0.0018 (0.0055)
Diabetes	0.0021 (0.0048)	-0.0037 (0.0080)	0.0140 (0.0140)
Alzheimer’s	0.0009 (0.0070)	0.0013 (0.0128)	-0.0009 (0.0234)
Chronic Lower Respiratory	-0.0027 (0.0030)	-0.0029 (0.0029)	0.0003 (0.0069)
Liver	0.0002 (0.0035)	-0.0063 (0.0055)	0.0159* (0.0090)
Kidney	-0.0142** (0.0060)	-0.0201** (0.0093)	0.0145 (0.0205)

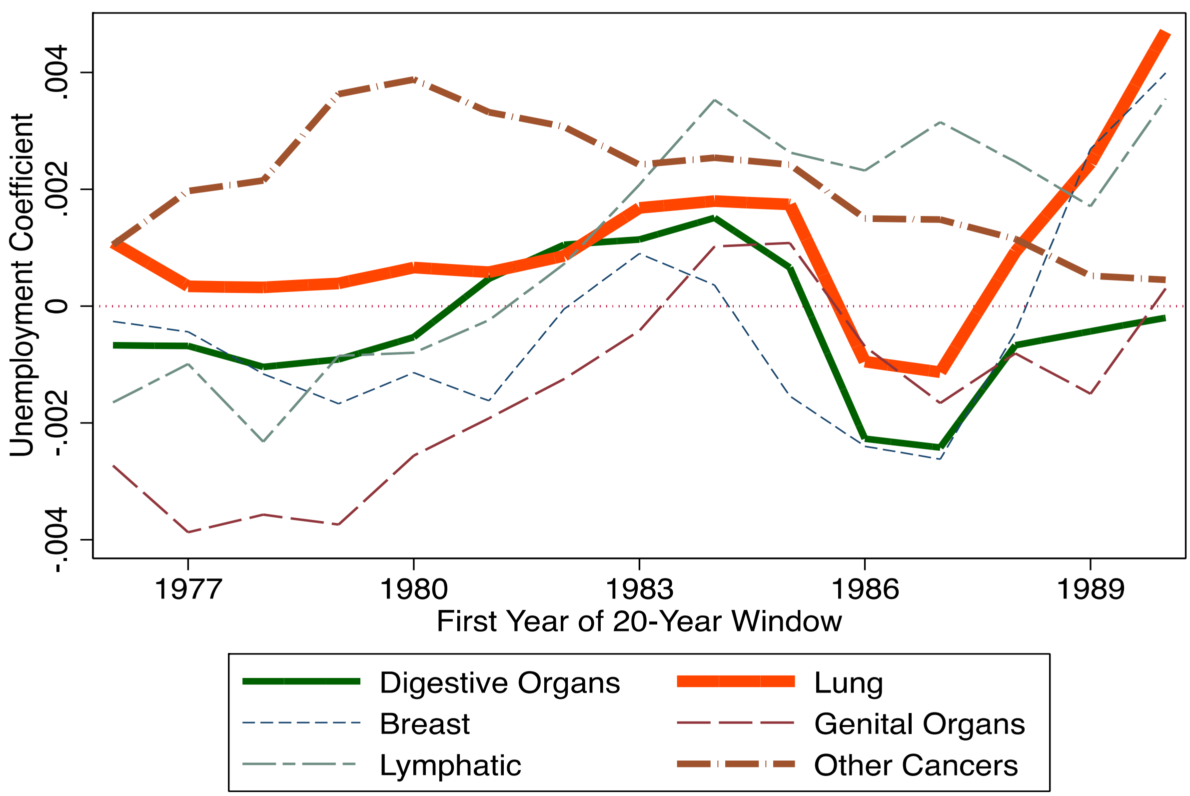
Note: See note on Table 3. Data for “other diseases” starts in 1979 (n=1,581) and trend variable in these cases scaled to equal zero in 1979 and one in 2009.

Figure A.1: Unemployment Coefficients for Deaths from Specific Diseases

Cardiovascular Disease



Cancer



Other Diseases