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Recessions, healthy no more?

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ABSTRACT

Over the 1976–2010 period, total mortality shifted from strongly procyclical to being weakly or unrelated to macroeconomic conditions. The association is likely to be poorly measured when using short (less than 15 year) analysis periods. Deaths from cardiovascular disease and transport accidents continue to be procyclical; however, countercyclical patterns have emerged for fatalities from cancer mortality and external causes. Among the latter, non-transport accidents, particularly accidental poisonings, play an important role.

1. Introduction

Health is usually thought to worsen when the economy weakens, but substantial recent research suggests that mortality actually *declines* during such periods. Following Ruhm (2000), most recent studies utilize longitudinal data and panel techniques to control for many confounding factors, including time-invariant area-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 procyclical fluctuations in total mortality and several specific causes of death[2]. In Ruhm's (2000) analysis of the U.S., covering 1972–1991, a one percentage point increase in the state unemployment rate was estimated to decrease total mortality by 0.5% and motor vehicle and cardiovascular disease (CVD) deaths by 3.0% and 0.5%, with reductions also observed for fatalities from influenza/pneumonia, liver disease, non-vehicle accidents and homicides. By contrast, cancer mortality was unaffected and suicides were estimated to rise by 1.3%[3]. Using similar empirical methods, the procyclicality of total mortality has been confirmed for Germany (Neumayer, 2004), Spain (Tapia Granados, 2005), France (Buchmueller et al., 2007), Mexico (Gonzalez and Quast, 2011), Canada (Ariizumi and Schirle, 2012), OECD countries (Gerdtham and Ruhm, 2006), and Pacific-Asian nations (Lin, 2009)[4]. Motor vehicle and CVD fatalities are procyclical in almost all studies, with more variation in mortality from other causes[5].

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[1] I thank seminar participants at the College of William and Mary, New School for Social Research, National Bureau of Economic Research, Bureau of Economic Analysis, American Health Economics Conference and the Southeastern Health Economics Study Group for helpful comments and the University of Virginia Bankard Fund for financial support.

[1] Earlier investigations (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: they represent the most severe negative health outcome, are well measured and diagnosis generally does not depend on access to the medical system. However, changes in non-life-threatening health conditions are not accounted for. Due to limited data availability, few analyses examine how macroeconomic conditions affect morbidity. Exceptions include Ruhm (2003) and Charles and DeCicca (2008).

[3] Thus, mental health and physical health may move in the opposite direction.

[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).

[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, making it difficult to compare the results with other related research. Analyses undertaken as early as the 1920s uncovered positive relationships between economic activity, total mortality and several specific causes of death (Ogburn and Thomas, 1922; Thomas, 1927; Eyer, 1977), as have some recent analyses using different methods (e.g. Fishback et al., 2007; Tapia Granados and Diez Roux, 2009).

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Some investigations suggest that mortality has become less procyclical or countercyclical in recent years. Using methods and data similar to 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 to 1991, but a smaller 0.19% decrease when extending the analysis through 2006[6]. McInerney and Mellor (2012) estimate that a one-point rise in joblessness lowered the mortality rates of persons 65 and over by 0.27% during 1976–1991, but raised them 0.49% from 1994 to 2008. Svensson (2007) uncovers a positive relationship between Swedish unemployment rates and heart attack deaths from 1987 to 2003[7].

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 (Ruhm and Black, 2002; Ruhm, 2005; Gruber and Frakes, 2006; Freeman, 1999; Xu, 2013), and 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 relationships. Charles and DeCicca (2008) indicate that male obesity is countercyclical; Arkes (2009) obtains a similar result for teenage girls (but not boys); Arkes (2007) shows that teenage drug use increases in bad times; Dávlos et al. (2012) uncover a countercyclical pattern for some types of alcohol abuse and dependence; Colman and Dave (2013) 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 skepticism because the analysis periods are too short (eight years or less) to provide definitive results.

Using U.S. data covering 1976–2010, the present study examines whether the relationship between macroeconomic conditions and mortality has changed over time. Comparability with previous investigations is maximized by using empirical methods that conform closely to that research[8]. Three primary results emerge. First, total mortality has shifted from being strongly procyclical to being weakly related or unrelated to macroeconomic conditions. Evidence from prior research that deaths decline when the economy deteriorates largely reflects the inclusion of early sample years, when this was the case. Second, the results obtained using relatively short (less than 15 year) periods show considerable instability and should probably be viewed as unreliable. Third, fatalities due to cardiovascular disease and, to a smaller degree, transport accidents continue to be procyclical, whereas strong countercyclical patterns for cancer and some external sources of death (particularly accidental poisonings) have emerged.

2. Research design

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 estimating equation is:

$$\ln\left(M_{kjt}\right) = \alpha_{kj} + X_{jt}\beta + U_{jt}\gamma + \lambda_{kt} + T_{jkt} + \varepsilon_{kjt} \quad (1)$$

where M_{kjt} is the mortality rate from source k in state j at year t, U is the state unemployment rate, X a vector of covariates, α a state fixed-effect, λ a general time effect, T a state-specific linear time trend, ε is the error term, and $\hat{\gamma}$ provides the estimated macroeconomic effect of key interest[9].

The year effects (λ_{kt}) 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_{kj}) account for those that differ across states but are time-invariant (such as persistent lifestyle disparities between residents of Nevada and Utah). Since the supplementary time-varying state characteristics (X_{jt}) do not necessarily control for all time-varying determinants of death, the models also include state-specific trends (T_{jkt})[10]. The 1976–2010 analysis period reflects the availability of consistent data on state unemployment and mortality rates. The macroeconomic impact is then identified from within-location variations in mortality rates, relative to changes in other states and after controlling for demographic characteristics and state-trends. Since the impact of national business cycles is absorbed by the time effects, discussions of macroeconomic effects refer to changes within-states rather than at the national level.

One way of investigating whether the impact of macroeconomic conditions on mortality has changed is to compare predicted effects differ across sub-periods. However, since such estimates are often sensitive to the choice of starting or ending years, two alternative strategies are employed. First, models for total mortality are estimated with differing starting and ending dates, and with varying lengths of the analysis period. The second, and main, method specifies analysis periods of fixed duration and then sequentially estimates models for *all* alternative sample windows permitted by the data. Most commonly, 20-year periods are used with results obtained for 16-windows ranging from 1976–1995 to 1991–2010.

Figures are frequently provided with point estimates (and sometimes confidence intervals) on the unemployment rate coefficient presented for each analysis window. Tables are also supplied showing unemployment coefficients and standard errors for the first and last of the 20-year periods (1976–1995 and 1991–2010), denoted by $\hat{\gamma}_\tau$ and $\hat{s}_\tau$, respectively, where τ equals 1 (2) in the first (last) period. I test whether the macroeconomic effect has changed by providing estimates for $\Delta\hat{\gamma} = \hat{\gamma}_2 - \hat{\gamma}_1$.

Using Eq. (1), $(e^{\hat{\gamma}k} - 1) \times 100\%$ provides the predicted percentage change in mortality from source k resulting from a one percentage point increase in the unemployment rate. While these estimates show the *relative* size of the macroeconomic effect, they do not directly indicate changes in the *absolute* number of predicted fatalities because, for example, large relative effects may imply small absolute changes for sources that are responsible for few deaths. These relative effect sizes are translated into absolute numbers through estimates of:

$$\left(e\Delta^{\hat{\gamma}k} - 1\right) \times \pi_k D \quad (2)$$

where $\Delta\hat{\gamma} = \hat{\gamma}_{2k} - \hat{\gamma}_{1k}$, D is the average annual number of deaths (2222,313) and π_k is the share of deaths due to source k over the 1976–2010 period.

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

[7] Using time-series methods for the U.S. from 1961 to 2010, Lam and Piérard (2014) also argue that total and cardiovascular mortality have become less procyclical over time, while motor vehicle fatalities remain strongly procyclical.

[8] One exception is the use of an uncommonly detailed set of age controls.

[9] Unemployment rates are used to *proxy* macroeconomic conditions; however, a procyclical variation in mortality does not imply that the loss of a job improves health. To the contrary, Sullivan and von Wachter (2009) show that job loss is associated with increases in individual mortality rates.

[10] Mortality trends vary considerably across sources of death, with large secular reductions for total mortality and that from cardiovascular disease and external sources, a relatively flat trend for cancer, and an increase for other disease deaths. State-year population weights were also sometimes incorporated but unweighted estimates are generally preferred (Wooldridge, 1999; Butler, 2000; Solon et al., 2015) and so are focused upon below.

A potential concern is that calculations based on (2) do not account for the possibility that a portion of the trend in macroeconomic effects on overall mortality rates could reflect secular changes in the *shares* of deaths due to specific sources. This was examined using a variation of the Oaxaca (1973) and Blinder's (1973) decomposition method. The method and results, which are summarized in the online appendix, indicate that almost all of the macroeconomic effect was due to changes in the coefficients, rather than in mortality shares, so that predictions obtained from (2) are useful.

3. Data and descriptive statistics

Annual average state unemployment rates, the main proxies for macroeconomic conditions were obtained from the U.S. Department of Labor's *Local Area Unemployment Statistics Database* (www.bls.gov/lau/lauov.htm), which provides monthly estimates of total employment and unemployment rates for census regions and divisions, states, metropolitan statistical areas, counties, and some cities[11]. Concepts and definitions underlying the *LAUS* data come from the Current Population Survey. Mortality data are from the Center for Disease Control and Prevention' *Compressed Mortality Files* (*CMF*) (www.cdc.gov/nchs/data_access/cmf.htm), which 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). Data prior to 1988 are publicly available; those from 1989 to 2010 were obtained by special agreement with the CDC. Population data (the denominator in the mortality rate calculations) from 1981 on come from the National Cancer *Surveillance Epidemiology and End Results (SEER)* program (http://www.seer.cancer.gov/data)[12]. These were supplemented by census estimates, included in the *CMF* files, for 1976–1980.

In addition to total annual mortality rates, sex-specific death rates were constructed, as were fatality rates for five age groups (<25, 25–44, 45–64, 65–74, and ≥75 year olds) and deaths from major diseases and external causes[13]. The *SEER* data were additionally used to construct independent variables for the share of the state population who were female, nonwhite, Hispanic, and aged <1, 1–19, 45–54, 55–64, 65–74, 75–84 and ≥85 years old[14].

The analysis of cause-specific mortality introduces complications. From 1976 to 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 ICD-10 categories since 1999. Crosswalks have been established between ICD-8 and ICD-9 and between ICD-9 and ICD-10 coding systems; 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 important for many 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 using ICD-8 versus ICD-9 codes (Klebba and Scott, 1980).

When the estimated comparability ratios are close to one (i.e. a similar number of deaths are reported using either ICD system), issues of data comparability are likely to be minor and well captured by the inclusion of regression year fixed-effects. For example, the estimated comparability ratios are 1.013 and 1.003 for CVD and cancer fatalities, when using ICD-8 and ICD-9 codes, and 0.998 and 1.007 for ICD-9 and ICD-10 categories. However, the potential problems are greater for some numerically important causes of death, and for others that have been analyzed in previous research[15]. Due to these concerns, the analysis of disease mortality is restricted to the major categories of CVD and malignant neoplasms, as well as a generic grouping of all other disease types[16]. A fuller investigation is provided for subcategories of external deaths, including those from transport accidents, non-transport (other) accidents, intentional self-harm (suicide), and homicide/legal intervention. Because non-transport accidents will be shown to be particularly important, separate analysis is conducted for the subcomponents: falls, drowning/submersion, smoke/fire/flames, and poisoning/exposure to noxious substances[17].

Appendix Table A1 details the ICD codes used to classify causes of death. Means and sample standard errors for mortality rates (per 100,000 population)and state characteristics are detailed in the online appendix. Appendix Table A2 illustrates how the sources of death changed over the analysis period, showing numbers and shares of fatalities during 1976–1995 and 1991–2010. As expected, given increased life expectancy, the proportion of mortality accounted for by the elderly has grown substantially. Declines in the share of cardiovascular deaths has been offset by increases in mortality from other diseases. The fraction from external sources changed little, with reductions from fatal transport accidents and homicides being compensated for by increases in non-transport accidents, particularly poisoning deaths.

4. The declining procyclicality of total mortality

Fig. 1 supplies three ways of examining whether the procyclicality of total mortality has diminished over time, by estimating Eq. (1) for different time periods. Solid lines show point estimates and dotted lines the 95% confidence intervals.

Fig. 1A displays unemployment rate coefficients where the analysis period begins in 1976 and ends in years ranging between 1995 and 2010. The magnitude of the estimated macroeconomic effect declines monotonically but modestly as the sample is extended to more recent periods, ranging from −0.0043 when the last year is 1995 to −0.0034 when it is 2010. All of the coefficients are significantly different from zero and these results, which are largely

[11] Some recent studies of macroeconomic patterns of health behaviors have analyzed county-level or MSA data (e.g. Charles and DeCicca, 2008; An and 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. Also, Lindo (2015) provides evidence that the health effects of macroeconomic conditions are understated when using more disaggregated (e.g. county rather than state) data. Preliminary analysis revealed similar results using state and county data starting in 1990.

[12] The *SEER* data are designed to supply more accurate population estimates for intercensal years than standard census projections, and to adjust for population shifts in 2005, resulting from Hurricanes Katrina and Rita. Differences between the *SEER* and *CMF* population estimates are miniscule prior to 2000 but are sometimes reasonably large (up to 3%) after 2003.

[13] 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 occur to those who are relatively old.

[14] Hispanic population shares are not provided prior to 1981. Therefore, shares for 1976–1980 were extrapolated as a linear trend for changes occurring between 1981 and 1986.

[15] For instance, the ICD-10 to ICD-9 comparability ratios are 0.698, 1.232 and 1.554 for influenza/pneumonia, kidney disease (nephritis, nephrotic syndrome, nephrosis) and Alzheimer's disease.

[16] Ruhm (2013) provides a preliminary analysis examining three sub-components of CVD, five categories of malignant neoplasms and five types of other diseases.

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

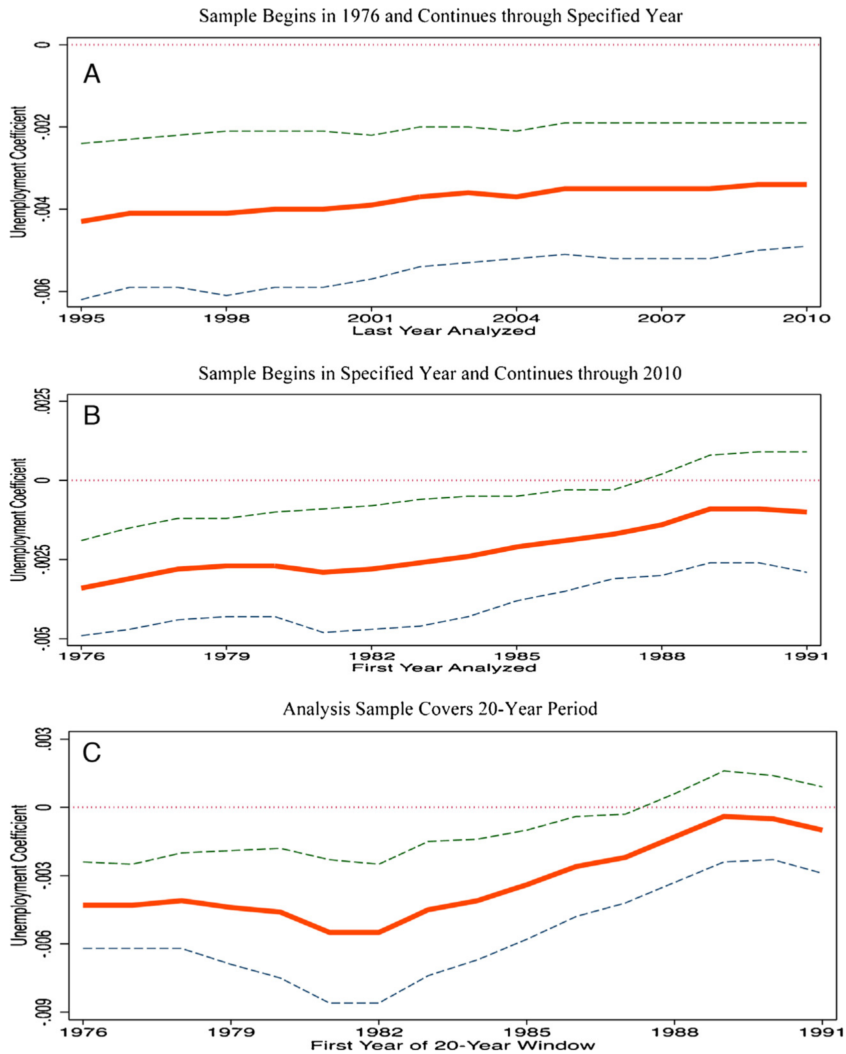

Fig. 1. Unemployment coefficients for total mortality using different analysis samples. (A) Sample begins in 1976 and continues through specified year. (B) Sample begins in specified Year and Continues through 2010. (C) Analysis sample covers 20-year period.

similar to those of previous research, do not alter the conclusion that mortality is procyclical.

The sensitivity of findings to the choice of sample periods can be seen more explicitly in Fig. 1B, where the sample always ends in 2010 but the starting year varies between 1976 and 1991. The unemployment coefficient attenuates from −0.0034 for the entire sample period to between −0.0029 and −0.0009 when the starting year is 1978 or later[18]. Perhaps more importantly, the data fail to reject the null hypothesis of *no* macroeconomic effect for periods beginning after 1988.

Fig. 1C displays results using 20-year sample windows beginning in the specified year. The left-most entry shows that the unemployment coefficient for 1976–1995 is −0.0043, while the farthest right result shows that it is −0.0010 for 1991–2010. Total mortality is significantly procyclical (negative unemployment rate coefficients) for all 20-year windows starting between 1976 and 1987, but the predicted effect diminishes steadily for windows beginning after 1982 and is small and insignificant for those starting in 1988 through 1991. This pattern is *not* caused by especially negative health consequences of the great recession of 2007–2009.

The choice of 20-year sample windows is arbitrary and may conceal an increased procyclical variation of mortality toward the end of the data period. This possibility is investigated in Fig. 2, which replicates Fig. 1C, but for periods of between 5-years and 20-years. Two findings deserve mention. First, at shorter durations, the estimates become more volatile and less precise. For instance, when using 5-year windows, the unemployment coefficients fluctuate wildly for even small changes in timing (e.g. from 0.0120 for 1996–2000 to −0.0077 for 1999–2003) but 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 determine whether the possible partial reversion of the macroeconomic effects in recent years (toward more procyclical mortality) is real or reflects statistical noise[19]. An important implication is that the findings of

[18] The full sample estimate is in line with previous results. Ruhm (2000) obtains a slightly larger 0.5% reduction in total mortality but the current estimate is close to the 0.3% decrease obtained in Stevens et al.'s (2011) preferred specification.

[19] When using 10-year periods, the average standard error is 46% larger for analysis windows beginning between 1989 and 2001 than for those starting between 1976 and 1988 (0.0018 versus 0.0013).

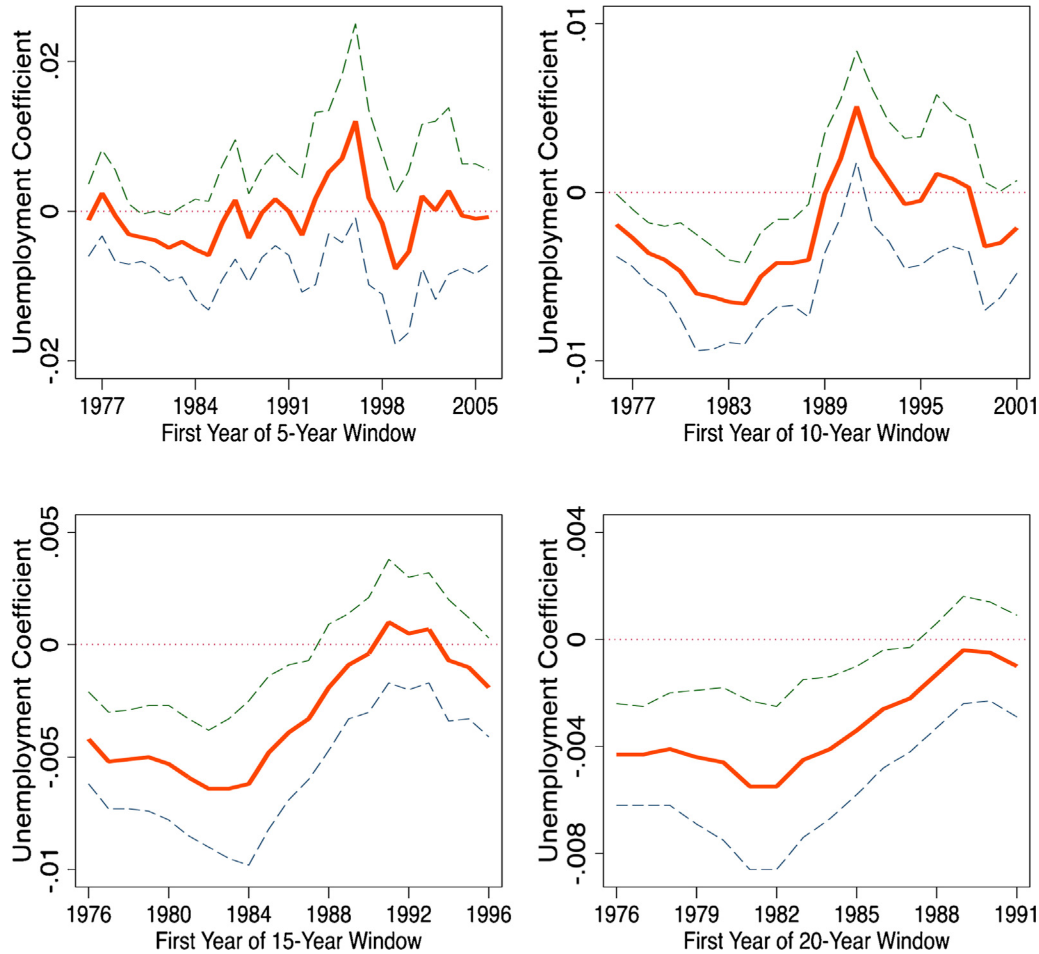

Fig. 2. Unemployment coefficients for total mortality using different sample windows.

many recent investigations of macroeconomic variations in health outcomes and behaviors should be viewed with extreme caution because the analysis periods are too short to provide reliable estimates[20].

Fig. 3 shows that the estimated change in the relationship between macroeconomic conditions and total mortality is fairly insensitive to controlling for state-specific linear time trends or weighting the data. (This figure, and those that follow, show point estimates for 20-year analysis windows that begin in the year specified on the *X*-axis.) In all cases, the estimated procyclicality of mortality declined over time, with most of the change dating from the early to mid-1980s and with statistically insignificant unemployment rate coefficients for some or all recent analysis windows. Removing state-specific trends makes this effect more pronounced than in the "preferred" specifications, which use unweighted data and control for state trends; weighting the data and including trends makes it somewhat less so[21]. In all cases, the change in the unemployment coefficient between 1976–1995 and 1991–2010 is positive and statistically significant.

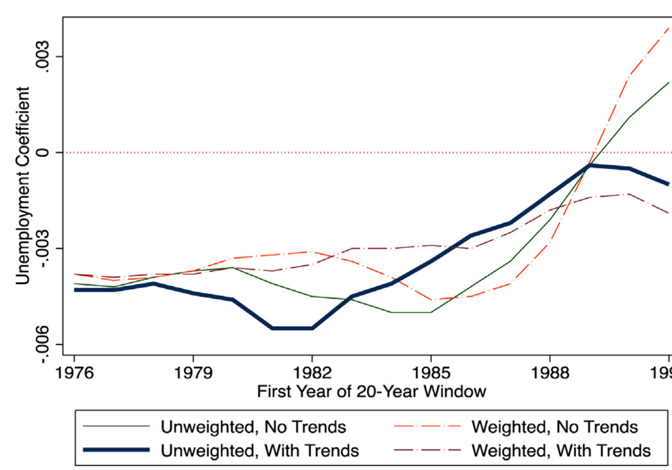

Fig. 3. Unemployment coefficients for total mortality using alternative estimation methods.

Table 1 shows the unemployment coefficients, from estimating Eq. (1), for total, sex-specific and age-specific mortality. Results

[20] Charles and DeCicca's (2008) analysis of male obesity used data from 1997 to 2001; Arkes' (2007, 2009) investigation of teenage body weight utilized information from 1997 to 2004, Dávlos et al.'s (2012) study of alcohol abuse and dependence compared 2001–2002 and 2004–2005, Colman and Dave's (2013) research on work and leisure-time physical activity covered 2003–2010, Cotti and Tefft's (2011) analysis of alcohol-related vehicle fatalities used data from 2003 to 2009, and Tekin et al. (2013) investigated a variety of health outcomes and behaviors from 2005 to 2011.

[21] For example, the unemployment rate coefficient (standard error) for the 1991–2010 analysis window is 0.0022 (0.0017), 0.0039 (0.0020), −0.0010 (0.0010) and −0.0019 (0.0009) for unweighted data without trends, weighted data without trends, unweighted data with trends and weighted data with trends.

Table 1
Estimated macroeconomic effects on specific sources of mortality.

Type of mortality	1976–1995	1991–2010	Difference
All	−0.0043 (0.0009)***	−0.0010 (0.0010)	0.0033 (0.0012)***
Sex-specific			
Males	−0.0044 (0.0010)***	−0.0001 (0.0012)	0.0043 (0.0015)***
Females	−0.0041 (0.0010)***	−0.0018 (0.0011)	0.0022 (0.0014)
Age-specific (Years)			
<25	−0.0165 (0.0025)***	0.0024 (0.0035)	0.0189 (0.0037)***
25–44	−0.0078 (0.0028)***	0.0016 (0.0035)	0.0094 (0.0042)**
45–64	−0.0020 (0.0011)*	0.0018 (0.0013)	0.0038 (0.0017)**
65–74	−0.0037 (0.0009)***	−0.0019 (0.0012)	0.0018 (0.0014)
≥75	−0.0041 (0.0010)***	−0.0019 (0.0013)	0.0022 (0.0012)*

Note: Dependent variable is the natural log of the specified state mortality rate, obtained from the *Compressed Mortality Files*, for 1976 to 2010 ($n = 1785$). The first two columns show the coefficient on the state unemployment rate for 20-year subsamples ($n = 1020$) covering 1976–1995 and 1991–2010. 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 third column shows the difference between the unemployment coefficients for the 1991–2010 and 1976–1995 subsamples. Robust standard errors, clustered at the state level, are shown in parentheses. *** $p < 0.01$, ** $p < 0.05$, * $p < 0.1$.

in the first column refer to 1976–1995, those in the second to 1991–2010, with the final column showing the difference between the two. A one point rise in unemployment predicts a statistically significant 0.43% reduction in total mortality during 1976–1995 compared to a small and insignificant 0.10% decrease in 1991–2010 (see the first row). The 0.33% difference between these two periods is statistically significant and indicates that the procyclicality of mortality has largely disappeared in recent years[22].

To address the possibility that the observed secular trends reflect a change in the relationship between unemployment rates and macroeconomic conditions, rather than in the health effects of economic conditions, I estimated specifications that controlled for nonemployment (the percentage of the 16 and over civilians unemployed or out of the labor force) rather than unemployment rates[23]. In these models a one percentage point increase in the nonemployment rate predicted a statistically significant 0.39% reduction in total mortality during 1976–1995 and an insignificant 0.02% decrease in 1991–2010[24]. The highly significant 0.37% difference is slightly larger than that obtained using unemployment rates.

The remainder of Table 1 and Fig. 4 summarize subgroup analyses, stratified by gender and age. In Fig. 4, and subsequently, thicker lines indicate sources with relatively high mortality shares[25]. The evidence suggests larger secular changes in macroeconomic effects for men than women. In 1976–1995, a one point rise in unemployment predicted a 0.44% reduction in male mortality and a 0.41% decrease for females. This effect completely disappeared by 1991–2010 for men but fell only half as much for women. The declining procyclicality of mortality has been particularly pronounced for males since 1982, while showing a steadier reduction for females (see Fig. 4A).

The trends also appear to be relatively pronounced for the young and middle-aged. We are unable to reject the null hypothesis of a zero unemployment rate effect in 2010 for all age groups. Specifically, a one percentage point increase in joblessness *reduced* the predicted death rates of <25, 25–45, 45–64, 65–74, and ≥75 years old by 1.6%, 0.8%, 0.2%, 0.4%, and 0.4% in 1976–1995 but *increased* them by 0.2%, 0.2%, 0.2%, −0.2%, and −0.2% in 1991–2010. We are unable to reject the null hypothesis of a zero unemployment rate effect in 1991–2010 for all age groups. The patterns for all possible 20-year windows are qualitatively similar (see Fig. 4B). The 95% confidence intervals (not shown) exclude positive unemployment rate coefficients for all two-decade periods beginning prior to 1986 for ≥75 year olds, before 1989 for <25 year olds, and earlier than 1983 for those aged 65–74. Conversely, a zero coefficient is rarely rejected for 45–64 year olds.

5. Heterogenous effects across sources of death

Table 2 and Figs. 5 and 6 stratify disease versus external sources of death, and then separately examine three disease and four external causes. The three disease categories: cardiovascular, cancer and other diseases, accounted for 42%, 23% and 29% of deaths over the 1976–2010 period. The four external sources: transport accidents, other (non-transport) accidents, suicides and homicides were responsible for 2.2%, 2.4%, 1.4% and 0.9%. Finally, four specific types of non-transport accidents are considered – falls, drowning/submersion, smoke/fires/flames and poisoning/exposure to noxious substances – which constituted 0.7%, 0.2%, 0.2% and 0.5% of fatalities.

Levels and trends of the macroeconomic effects differ markedly for mortality from disease versus external causes. A one point rise in joblessness lowered predicted disease mortality by 0.33% in 1976–1995 versus 0.14% in 1991–2010, a stastically insignificant change. By contrast, a much larger 1.5% reduction in external deaths was estimated for the earlier years versus a statistically significant 0.8% increase in the later ones, and the difference is highly significant. Fig. 5 suggests an almost monotonic but modest attenuation over time in the unemployment coefficient for deaths from disease, with statistically significant negative estimates obtained for all two decade periods starting before 1986. Conversely, the predicted effect for external causes was negative and fairly stable for 20-year periods beginning between 1976 and 1982, but attenuated steadily for analysis windows starting from 1982 to 1990, with a statistically insignificant effect for those with first years between 1984 and 1989, and significantly positive unemployment rate coefficients obtained for those initiating in 1990 or 1991. These results help to explain the sharp reversal in the effects

[22] Coefficients on the other time-varying state level covariates are provided in the online appendix.

[23] 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).

[24] A one percentage point increase in the unemployment rate predicts a 0.73 percentage point rise in nonemployment over the full period, in models controlling for state demographic characteristics, time trends, and state and year dummy variables. Changes in interstate migration are also unlikely to explain the results. Migrants tend to be healthy and to move from areas of higher to lower unemployment rates (Halliday, 2007), introducing a countercyclical mortality effect. Mortality might therefore have become less procyclical if migration rates were increasing over time. However, migration rates instead peaked around 1980 and have fallen sharply since then (Malloy et al., 2011).

[25] For example, in Fig. 4B, the line for ≥75 year olds is thick because they account for 51% of mortality, from 1976 to 2010, whereas that for <25 year olds is thin because they are responsible for less than 4% of deaths.

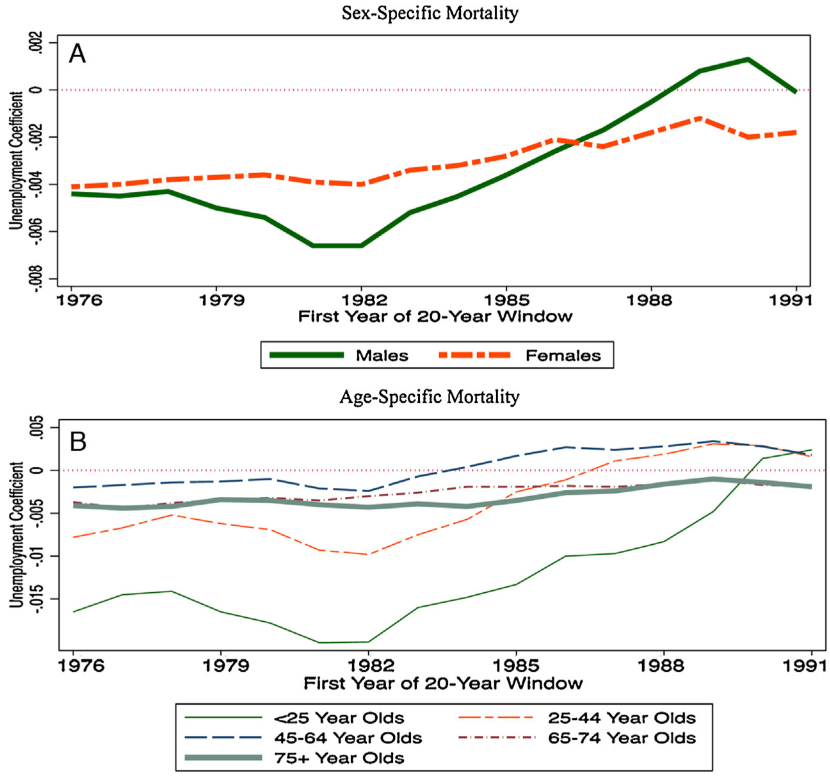

Fig. 4. Unemployment coefficients for sex-specific and age-specific mortality. (A) Sex-specific mortality. (B) Age-specific mortality.

Table 2
Estimated macroeconomic effects on cause-specific mortality.

Cause of death	1976–1995	1991–2010	Difference
Diseases	**−0.0033 (0.0010)*****	**−0.0014 (0.0010)**	**0.0019 (0.0013)**
Cardiovascular disease	−0.0036 (0.0013)***	−0.0041 (0.0017)**	−0.0005 (0.0019)
Cancer	0.0002 (0.0013)	0.0027 (0.0011)**	0.0024 (0.0011)**
Other diseases	−0.0060 (0.0020)***	−0.0011 (0.0036)	0.0048 (0.0027)*
External causes	**−0.0148 (0.0020)*****	**0.0078 (0.0028)*****	**0.0226 (0.0023)*****
Transport accidents	−0.0265 (0.0035)***	−0.0086 (0.0048)*	0.0180 (0.0030)***
Other accidents	−0.0173 (0.0033)***	0.0086 (0.0049)*	0.0259 (0.0048)***
Suicides	0.0041 (0.0031)	0.0171 (0.0046)***	0.0130 (0.0054)**
Homicides	−0.0063 (0.0065)	0.0160 (0.0115)	0.0223 (0.0100)**
Other accidents	**−0.0173 (0.0033)*****	**0.0086 (0.0049)***	**0.0259 (0.0048)*****
Falls	−0.0119 (0.0053)**	0.0016 (0.0084)	0.0135 (0.0095)
Drowning/submersion	−0.0015 (0.0065)	0.0143 (0.0155)	0.0159 (0.0147)
Smoke/fire/flames	−0.0308 (0.0099)***	−0.0019 (0.0179)	0.0289 (0.0169)*
Poisoning/noxious	−0.0148 (0.0161)	0.0422 (0.0251)*	0.0570 (0.0290)**

Note: See note on Table 1. *** $p < 0.01$, ** $p < 0.05$, * $p < 0.1$.

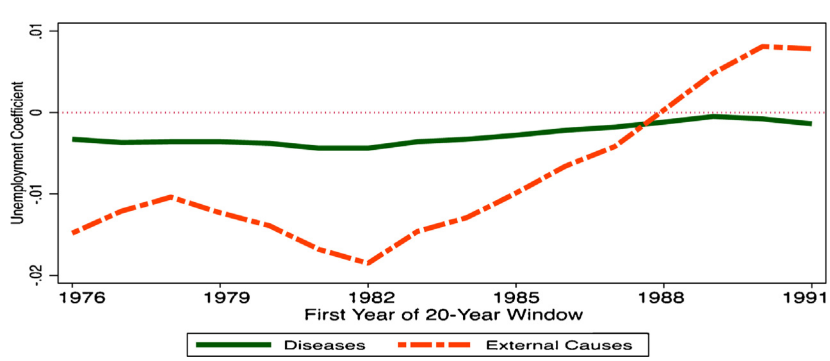

Fig. 5. Unemployment coefficients for disease versus external of deaths.

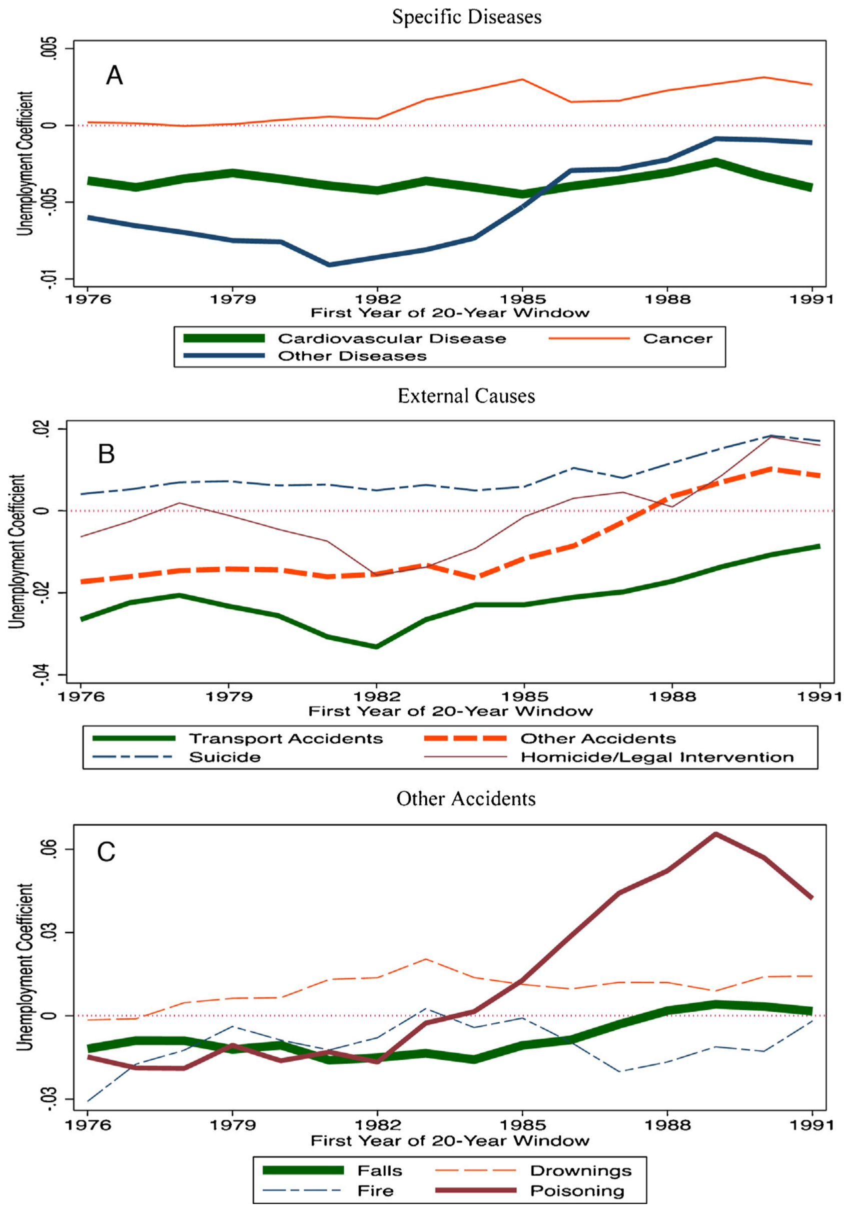

Fig. 6. Unemployment coefficients for deaths from specific diseases and external causes. (A) Specific diseases. (B) External causes. (C) Other accidents.

of macroeconomic conditions on deaths of younger persons, who disproportionately die from external causes[26].

5.1. Diseases

There are striking disparities across types of diseases. Cancer mortality was unrelated to the economy in 1976–1995 but strongly countercyclical by 1991–2010, whereas CVD mortality remained strongly procyclical throughout (see the top panel of Table 2 and Fig. 6A). The procyclicality of other disease mortality declined over time but the change between 1976–1995 and 1991–2010 was not quite significant at the 0.05 level[27].

Research for earlier time periods (e.g. Ruhm, 2000; Neumayer, 2004; Miller et al., 2009) documents a strong procyclicality of cardiovascular deaths but with little macroeconomic variation in cancer fatalities, and attributes this to the likelihood that short-term behavior changes (e.g. smoking, diet and exercise) more strongly influence the risk of CVD than cancer deaths. A conceivable explanation for the findings just described is that the relationship between macroeconomic conditions and health behaviors has remained relatively stable, while cancer mortality has become more sensitive to the availability of financial resources and access to (procyclical) health care due to improvements in expensive medical treatments and technologies[28].

[26] For example, <45 year olds accounted for 56% of external deaths (from 1976 to 2010) but less than 10% of total mortality.

[27] This result is sensitive to weighting. Using weighted data, the unemployment rate coefficient was −0.0029 in 1976–1995 and −0.0020 in 1991–2009. The difference was 0.0009 with a standard error of 0.0031.

[28] The cost per cancer case rose from $47,000 in 1983 to $70,000 in 1999 (Philipson et al., 2012), with many expensive new medical treatments and chemotherapy agents coming into use in the 1990s and early 2000s (Cutler, 2008). The continued procyclicality of CVD mortality could also occur for other reasons, such as a (stable) deleterious health effect of air pollution (Heutel and Ruhm, 2013).

Table 3
Change in effect of macroeconomic conditions on predicted number of deaths.

Type of mortality	Share of deaths	Predicted Δ in no. of deaths	
		Point estimate	95% Conf. Interval
All deaths	1.00000	7253***	1882–12,637
Sex-specific			
Males	0.5121	4900***	*1625–8185*
Females	0.4879	2402	*−487–5298*
Age-specific (years)			
<25	0.0389	1655***	*1009–2305*
25–44	0.0572	1202**	*138–2276*
45–64	0.1865	1586**	*177–3000*
65–74	0.2031	799	*−454–2054*
≥75	0.5140	2565*	*−206–5343*
Diseases	0.9293	3988	*−1221–9210*
CVD	0.4155	−466	*−3846–2927*
Cancer	0.2263	1277**	*128–2329*
Other diseases	0.2875	3104*	*−326–6552*
External causes	0.0707	3582***	*2845–4322*
Transport accidents	0.0218	880***	*593–1168*
Other accidents	0.0243	1418**	*889–1942*
Suicide	0.0138	402**	*75–733*
Homicide	0.0094	469**	*56–891*
Other accidents	0.0243	1418***	*899–1942*
Falls	0.0069	207	*−78–497*
Drowning	0.0019	67	*−54–191*
Fires	0.0019	124*	*−18–271*
Poisoning	0.0054	709**	*1–1459*

Note: Predicted changes are for a one-percentage point increase in unemployment. Share of deaths is for 1976–2010. "Predicted Δ in # of deaths" is calculated as $(e^{\Delta\hat{\gamma}k}-1)\times\pi_k D$, where $\Delta\gamma\,\bar{}\,\gamma_2-\gamma_1$, for γ_τ the predicted unemployment coefficient in period τ, with $\tau=1$ in 1976–1995 and $\tau=2$ in 1991–2010; D is the average annual number of deaths during 1976–2010 (2222,313); π_k is the share of deaths from source k. 95% confidence intervals are estimated as $(e^{(\Delta\hat{\gamma}k\pm1.96\times s_k)}-1)\pi_k D$, for s_k the standard error on $\Delta\hat{\gamma}_k$.
*** $p<0.01$,
** $p<0.05$,
* $p<0.1$.

5.2. External causes

There is considerable heterogeneity in the effects for specific sources of external deaths (see the second panel of Table 2 and Fig. 6B). One of the most consistent previous research findings is that transport fatalities are procyclical[29]. This effect persists but has weakened recently, with a one percentage point rise in the unemployment rate predicting a 2.6% decrease in 1976–1995 versus a 0.9% reduction in 1991–2010. Suicides increase with joblessness, consistent with most prior studies, and this effect has strengthened over time: a one point growth in unemployment was associated with an insignificant 0.4% rise is suicides during 1976–1995 versus a highly significant 1.7% increase in 1991–2010[30].

The most noteworthy finding is that fatal non-transport accidents have switched from being strongly procyclical to sharply countercyclical: a one point rise in unemployment *reduced* predicted mortality rates by 1.7% in 1976–1995 but *increased* them 0.9% in 1991–2010, with nearly monotonic growth over time. The parameter estimates were negative and significant for all 20-year periods starting prior to 1985 but insignificantly *positive* for those beginning after 1987.

The bottom panel of Table 2 and Fig. 5C provide additional specificity on non-transport accident deaths, showing that the secular trends are dominated by changes for accidental poisonings, where the unemployment coefficient rose from −0.0148 in 1976–1995 to 0.0422 in 1991–2010, with a strong countercyclical pattern emerging for 20-year windows beginning after the early 1980s. There are more modest changes for deaths from falls or drownings and the procyclicality of fatalities from fires largely disappears in recent years but, as shown later, this is always a relatively minor source of mortality. Given these results, accidental poisonings receive special attention below.

6. Predicted changes in number of deaths

I next demonstrate that external sources of deaths, especially those for non-transport accidents and among these accidental poisonings, play a key role in explaining the declining macroeconomic responsiveness of total mortality. All numerical calculations are based on Eq. (2) and refer to a one percentage point increase in unemployment. The discussion focuses on predicted secular changes in macroeconomic effects, rather than levels for a single analysis period.

The first row of Table 3 shows that a one point increase in unemployment predicts 7253 more fatalities in 1991–2010 than in 1976–1995[31]. As noted, the procyclicality of mortality weakened over time more for men than women, so that males account for two-thirds of the overall change in the macroeconomic effect (see

[29] Previous analyses have often examined motor vehicle deaths, which constituted 94% of transport accident fatalities from 1976 to 2010. Transport deaths are considered here because they are coded more consistently across time.

[30] The unemployment coefficient was positive in all 20-year windows and statistically significant for those beginning after 1987.

[31] The unemployment coefficient was −0.00428101 for 1976–1995 and −0.00102266 in 1991–2010. The resulting difference of .00325834 implies around 0.33% more fatalities, or 7253 additional deaths per year, based on 2222,313 fatalities annually: (exp[−0.00102266−−0.00428101] − 1) × 2222,313 = 7252.86.

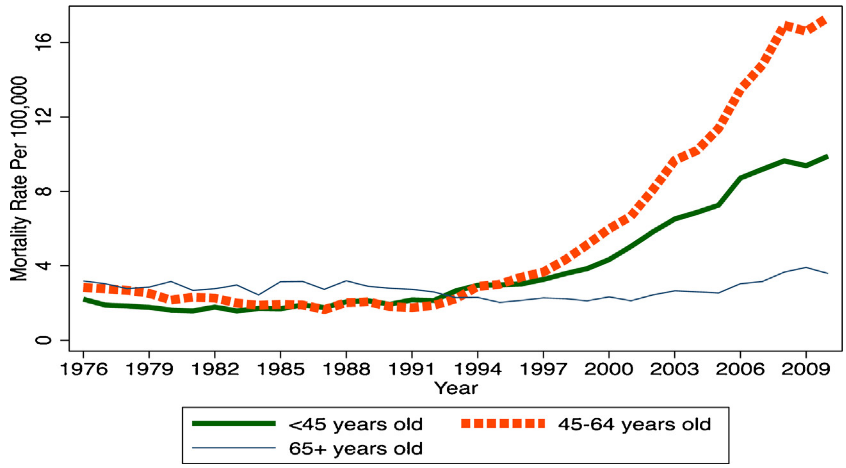

Fig. 7. Trends in accidental poisoning mortality, by age.

the second and third rows of Table 3)[32]. When decomposing by age, the most striking finding is the extent to which the declining procyclicality is concentrated among the relatively young: persons under <25 are responsible for less than 4% of deaths but 23% of the secular trend in the macroeconomic impact; those under 45 (65) comprise less than 10% (30%) of fatalities but almost two-fifths (over three-fifths) of the predicted change over time.

The remainder of Table 3 examines specific causes of mortality. A one percentage point rise in unemployment predicts almost 3600 more annual deaths from external sources in 1991–2010 than 1976–1995, or 49% of the change in total mortality. This occurs even through only 7% of all deaths are due to such causes, and helps to explain the large changes for males and <45 year olds (for whom external deaths account for over 40% of mortality). Cancer and other diseases also play a role, although the estimates are imprecise for the former and sensitive to the use of sampling weights for the latter[33]. By contrast, cardiovascular disease – the number one killer – explains none of the secular change, as the unemployment rate coefficient actually becomes more *negative* in later years.

The third panel of Table 3 provide a more detailed decomposition of external deaths. Non-transport accidents are of special interest because the unemployment coefficient switches from large and negative to positive, accounting for over 1400 additional deaths annually, or 40% of the predicted rise in external cause mortality. This increase is 11% larger than for all cancers, even though non-transport accidents constitute only 2% of all fatalities, versus 22% for malignant neoplasms. Transport accidents are also important, explaining 880 extra deaths per year. The effects on suicides and homicides are in the same direction but of considerably smaller magnitude.

The bottom panel of Table 3 presents separate results for four sources of non-transport accidents, which together explain around two-thirds of deaths from this cause. The role of accidental poisonings is remarkable. Although accounting for just 0.5% of deaths, a one point rise in unemployment is predicted to result in 709 more annual poisoning fatalities in 1991–2009 than in 1976–1995, or half the change in non-transport accident fatalities, 20% of that for external deaths and almost 10% of the overall mortality effect.

7. Discussion

The strong procyclical pattern of mortality present in the 1970s and 1980s has been largely eliminated in recent years. The pattern varies across sources of deaths, with much larger secular changes observed for external than disease causes. All types of external deaths became less procyclical or more countercyclical during the analysis period, with particularly large changes for non-transport accidents, and within this category, for accidental poisonings. Among diseases, cardiovascular mortality continues to fall sharply when the economy deteriorates, whereas cancer deaths have became substantially countercyclical. These findings are relevant not only for understanding of the production of health but also for measuring the size and effects of business cycle fluctuations. Egan et al. (2013) argue that procyclical mortality implies that business cycle fluctuations are milder than when calculated using standard GDP measures, but this may have become less true in recent years.

Some estimates are sensitive to changing the starting and ending dates of analysis. Such parameter instability is particularly problematic when the sample window is short – probably anything less than 15 years – raising concerns about the findings of many recent related investigations that have used brief (often less than 10 year) timespans. One contribution of this study is to provide parsimonious methods of illustrating the sensitivity of the results the length of the analysis window, and to the first and last years examined. Another caveat is that specific sources of death are implicitly treated here as being independent of each other, although some prior research (Yeung et al., 2014) identifies potential correlations between them. This is not an issue for the analysis of total mortality but may be important when considering specific causes of death as competing risks.

Mechanisms for the previously observed procyclical variation in mortality remain poorly understood, so it is speculative as to why the relationship has changed in recent years. Two possibilities are intriguing. First, the change dates to (20-year) analysis periods beginning in the early 1980s, which precisely coincides with the reduction in macroeconomic volatility that has been referred to as the "Great Moderation" (Stock and Watson, 2003; Bernanke, 2004)[34]. This raises the possibility that the mortality patterns here are part of a broader change in the effects of short-term changes

[32] Since separate (unconstrained) models are estimated for different sources of death, the total contribution of changes in predicted group-specific mortality can sum to more or less than the effect predicted for total mortality.

[33] With weighted data, a one point unemployment increase predicts 580 more deaths from other diseases in 1991–2010 than in 1975–1995

[34] Productivity also shifted from procyclical to acyclical or slightly countercyclical at about the same time (Galí and van Rens, 2010).

in macroeconomic performance, or in the role of unemployment rates as a proxy for macroeconomic conditions. With regards to the latter, it is noteable that the residual variation in state-year unemployment rates after including controls (one minus the R-squared from regressing unemployment rates on state and year dummy variables, state-specific time trends, and the time-varying state demographic characteristics) fell from 0.177 in 1976–1995 to 0.094 in 1999–2010. There is also suggestive evidence that the procyclicality of mortality might have increased slightly in the most recent analysis periods, that include the severe 2007–2009 recession.

Second, the emerging importance of accidental poisoning fatalities occurred at the same time that deaths from this source increased dramatically for young and middle-aged adults (see Fig. 7). Over 90% of poisoning fatalities are now due to drug overdoses, with particularly important roles for prescription opioids (such as hydrocodone and oxycodone) and benzodiazepines (Warner et al., 2011; Ruhm, 2015). The higher death rates reflect greater availability of these drugs raising the ease of self-injury and accidental death during bad economic times[35]. Moreover, economic weakness has long been associated with diminished mental health (Ruhm, 2000, 2003; Charles and DeCicca, 2008; Bradford and Lastrapes (2014) and, to the extent these drugs are now being taken to address this, the increased procyclicality of poisoning deaths may be a physical manifestation of what was previously a mental health problem[36].

Appendix A. Appendix

Tables A1 and A2.

Appendix B. Supplementary data

Supplementary data associated with this article can be found, in the online version, at http://dx.doi.org/10.1016/j.jhealeco.2015.03.004.

Table A1
Definitions of specific causes of mortality.

Variable	Description	ICD-8 (1976–1978)	ICD-9 (1979–1998)	ICD-10 (1999–2010)
Cancer	Malignant neoplasms	140–209	140–208	C00–C97
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
Transport	Transport accidents	800–848, 940–941	800–848, 929.0, 929.1	V02–V99, Y85
Other Ac	Other (non-transport) accidents	850–939, 942–949	850–928, 929.2–949	W00–X59, Y86
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 and legal intervention	960–978	960–978	X85–Y09, Y87.1, Y35, Y89.0

Table A2
Sources of death by time period.

Source of death	All Years		1976–1995		1991–2010	
	#	%	#	%	#	%
All deaths	2222,313	100.0	2081,936	100.0	2367,352	100.0
Males	1138,011	51.2	1097,329	52.7	1181,266	49.9
Females	1084,302	48.8	984,607	47.3	1186,086	50.1
Age of death (years)						
<25	86,549	3.9	97,528	4.7	74,871	3.2
25–44	127,161	5.7	126,536	6.1	134,595	5.7
45–64	414,460	18.6	399,677	19.2	418,713	17.7
65–74	451,301	20.3	472,501	22.7	437,723	18.5
≥75	1142,362	51.4	985,091	47.3	1301,093	55.0
Cause of death						
Cardiovascular	923,419	41.6	957,085	46.0	892,420	37.7
Cancer	502,882	22.6	463,278	22.3	548,661	23.2
Other diseases	638,973	28.8	510,290	24.5	765,354	32.3
External causes	157,039	7.1	151,283	7.3	160,917	6.8
Transport accidents	48,545	2.2	50,581	2.4	45,750	1.9
Other accidents	54,044	2.4	45,419	2.2	60,350	2.5
Falls	15,225	0.7	12,721	0.6	17,217	0.7
Drowning/submersion	4181	0.2	4676	0.2	3565	0.2
Smoke/fires/flame	4229	0.2	4984	0.2	3402	0.1
Poison/noxious substance	12,090	0.5	5938	0.3	17,225	0.7
Suicide	30,755	1.4	29,391	1.4	32,171	1.4
Homicide	22,560	1.1	22,560	1.1	20,117	0.8

Note: Table shows average deaths per year for the specified age group or cause.

[35] For example, per capita opioid sales more than tripled between 1999 and 2010 (Paulozzi, 2012).

[36] See Ruhm (2013) for a more extensive discussion of these issues.

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