

THE ECONOMY AND HEALTH[†]

Why Are Recessions Good for Your Health?

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A series of influential papers by Christopher J. Ruhm (2000, 2003, 2005, 2008) documents that recessions are “good for your health”—or, more specifically, that state-level mortality rates are strongly procyclical. The magnitude of the correlation is economically meaningful: a typical estimate from the literature suggests that a 1 percentage point increase in a state’s unemployment rate is associated with a 0.54 percent *reduction* in that state’s mortality rates. If this reflects a causal relationship that is also valid at the national level, then a 1 percentage point increase in the unemployment rate would translate (based on 2004 mortality rates) into about 12,000 fewer deaths per year. These findings are frequently interpreted as resulting from the rising opportunity cost of time that accompanies better labor market opportunities, and some empirical support exists for this interpretation. For example, Ruhm (2000) shows that obesity and smoking also exhibit a procyclical pattern,

and that diet and exercise improve when the unemployment rate rises—patterns that are consistent with changes in the value of time associated with working. On the other hand, research linking individual job displacements to individuals’ own mortality find that individuals who experience a job loss have higher probabilities of dying (Daniel G. Sullivan and Till M. von Wachter 2007). These results would be at odds with those based on state-level analyses unless the increase in state-level mortality rates that corresponds to improvements in the economy is driven by factors other than changes in individuals’ own labor supply and health behavior.

The purpose of this study is to advance our understanding of the mechanisms that are most likely to contribute to the procyclical relationship between macroeconomic conditions and mortality rates. In particular, we aim to distinguish between health changes resulting from changes in an individual’s own work and health behaviors and health changes that are related to “externalities” associated with the business cycle. While some of these possibilities have been explored in Ruhm’s earlier work, we bring additional light to bear on the question by focusing on more detailed mortality rate decompositions by age, sex, race, and cause of death, and by investigating the relationship between a particular demographic group’s mortality and the unemployment rate of that group relative to the unemployment rates of other demographic groups.

I. Data and Methodology

Our basic regression equation follows Ruhm (2000) and takes the following form:

$$(1) H_{j,t} = \alpha_t + \mathbf{X}_{j,t}\beta + U_{j,t}\gamma + S_j + S_jT + \varepsilon_{j,t},$$

where H is the natural log of the mortality rate in state j and year t , \mathbf{X} is a vector of state-year

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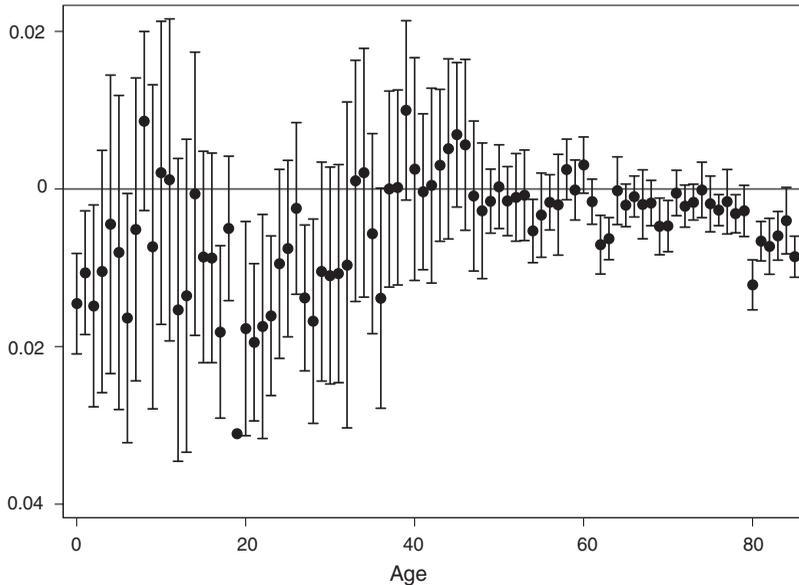


FIGURE 1. COEFFICIENTS ON UNEMPLOYMENT IN POISSON MODELS OF MORTALITY FOR EACH YEAR OF AGE

demographic controls, α , is a vector of year fixed effects, and S_j controls for time-invariant state characteristics. State-specific time trends, S_jT , are also included. The main indicator of a state's economic health, U , is the state unemployment rate. We have replicated Ruhm's (2000) analysis, which is based on data from 1972–1991, and then build on his work by utilizing several additional sources of data and extending the analysis through 2004. In order to get a consistent measure of the unemployment rate over time, most of our analyses begin with 1978. Our basic model incorporates mortality data from Centers for Disease Control and Prevention (CDC) Multiple Cause of Death Data, and population denominator data from the National Health Interview Survey (NHIS) Cancer Surveillance, Epidemiology and End Results Program (SEER). We also use the Cancer-SEER data to create control variables for the fraction of the population age 0–4, 5–17, 18–30, and 65+, and for the fraction black. Monthly Current Population Survey (CPS) data are used to create measures of the state's Hispanic population and education. Our regressions are weighted by population, and we cluster our standard errors at the state level. Finally, we estimate most of our models using a Poisson count data model because when we analyze subgroups we sometimes have cells

with zero mortality counts. Taken as a whole, these extensions/changes have a very limited impact on the estimated association between macroeconomic conditions and health. Our preferred specification suggests that a 1 percentage point increase in the unemployment rate leads to a 0.43 decrease in the mortality rate, compared to Ruhm's estimate of 0.54.

II. Decompositions by Age and Cause

Next, we begin to investigate the relative importance of “own” versus “other” factors by estimating the Poisson analogue to equation (1) separately by single year of age. Figure 1 shows the estimated coefficients on the unemployment rate and their associated confidence intervals for each age. Echoing Ruhm's earlier work, we find that young adults have the most cyclical mortality rates. However, the figure also makes three additional points. First, perhaps because we use more recent years of data, the typical semi-elasticity in the 20- to 44-year-old age range is much less than 2 percent (Ruhm's previous estimate). Second, the strong procyclical pattern among young adults is mostly driven by those at the younger end of the 20- to 44-year-old age range. Indeed, those age 35–44 have, on average, positive coefficient estimates. Finally,

TABLE 1—ESTIMATED RELATIONSHIP BETWEEN UNEMPLOYMENT AND AGE-SPECIFIC MORTALITY—ALL CAUSES

Age	Average beta	Predicted additional deaths
0–85	–0.0047	–11,803
0–0	–0.0146	–407
1–17	–0.0095	–173
18–24	–0.0167	–451
25–34	–0.0076	–300
35–44	0.0006	92
45–54	–0.0005	–87
55–64	–0.0018	–476
65–69	–0.0024	–407
70–74	–0.0018	–392
75–79	–0.0024	–761
80–84	–0.0072	–2,668
85+	–0.0086	–5,773

the larger magnitude of the cyclical among young adults extends to children as well. Since children are unlikely to be working, this finding suggests that the large coefficient estimates among young adults may result from something beyond the direct effect of their own employment experiences. Our estimates also confirm Ruhm's finding that the elasticity of the mortality rate with respect to the unemployment rate is lower (in absolute value) among those most likely to be retired. Among those older than 60, the estimated coefficient on the unemployment rate is negative but generally much smaller than the estimates for younger age groups.

Table 1 shows the weighted averages of the age-specific coefficient estimates from Figure 1 for each of 11 age groups, where we weight by the total number of deaths in each age cell. We also show the total increase in deaths that would be predicted from a 1 percent increase in the unemployment rate. As suggested by Figure 1, the largest coefficient estimates are for those age groups that are unlikely to be working. The average coefficient estimate for those under age 15 is -0.015 , but drops to -0.005 or less during the prime working ages of 35–65. The coefficient estimates increase slightly for those over age 65, another group that has limited labor force participation.

The relationship between the age-specific coefficient estimates and changes in the overall mortality rate depends on the number of deaths in each age group. Even though the coefficient estimates are largest among the young, they may not contribute much to overall mortality fluctuations because deaths among children and ado-

lescents are rare. To explore this issue further, we utilize the estimates from Figure 1, along with 2004 mortality data, to answer the question of how many “procyclical deaths” there are for each year of age. We do so by multiplying the estimated semi-elasticity for each year of age by the number of 2004 deaths for that age. We then aggregate these numbers for various age groups to assess the relative importance of each group in explaining aggregate mortality fluctuations; these results are shown in column 2 of Table 1.

There were 2,397,269 deaths in the United States in 2004. The overall average semi-elasticity is -0.0047 , and we estimate that a 1 percent rise in the unemployment rate would lead to approximately 12,000 additional deaths in the population. The bulk of those additional deaths, however, would occur among those with relatively weak labor force attachment: only 7 percent of the additional deaths from an increase in the unemployment rate would occur among those between the ages of 25 and 64. In contrast, 71 percent of the additional deaths are predicted to occur to those over age 80. The fact that the vast majority of deaths occur among those unlikely to be working suggests that individuals' own labor market involvement is not the key mechanism behind procyclical fluctuations in the overall mortality rate. While work, leisure, and health behaviors over the business cycle may play some role in generating procyclical mortality, the concentration of most of these “cyclical” deaths outside of typical working ages suggests that other factors, perhaps reflecting business cycle externalities, must also be very important.

TABLE 2—ESTIMATED RELATIONSHIP BETWEEN UNEMPLOYMENT AND CAUSE-SPECIFIC MORTALITY

Cause of death	Average beta	Predicted additional deaths
All causes	−0.0047	−11,803
Cardiovascular	−0.0047	−4,260
Cancer	0.0019	1,019
Respiratory	−0.0118	−2,771
Infections	−0.0200	−1,453
Degenerative brain	−0.0166	−2,686
Kidney	−0.0153	−683
Motor vehicle accidents	−0.0294	−1,285
Other accidents	−0.0103	−603
Suicide	0.0168	641
Homicide	−0.0162	−290
Other	−0.0138	−1,587
Nutrition, birth defects, gastrointestinal	−0.0046	−832
All non-motor vehicle accidents	−0.0043	−10,755

Another clue to the mechanisms driving procyclical mortality comes from disaggregating the relationship according to the cause of death. Table 2 shows the results of estimating equation (1) separately by cause of death. Like Ruhm, we find that the largest estimated coefficient, by far, is that for motor vehicle accidents, (the weighted average estimate is -0.029). Focusing on the number of additional deaths generated by a reduction in the unemployment rate, cardiovascular causes make up the largest category, with more than 4,000 additional deaths, or more than one-third of total deaths. The large coefficient on motor vehicle accidents is consistent with mechanisms other than individual work or leisure choices playing a prominent role in overall procyclicality. In contrast, (as has been emphasized in earlier work by Ruhm) the fact that more than one-third of the cyclically induced deaths are due to cardiovascular factors could point toward work-related stress or other time allocation choices as a key part of the story.

Looking more closely at the distribution of cardiovascular deaths, however, casts doubt on the role of individual work and health behaviors for overall cyclicality. Specifically, we have estimated equation (1) by age and cause of death. In Table 3, we summarize the number of predicted deaths for each of the top six causes of death (from Table 2) by age group and cause. Note that among prime working-age individuals, only a trivial number of cardiovascular deaths are induced by business cycle changes. The age-specific pattern of cardiac deaths does not support the notion that such deaths result

from work-related stress, or from substitution between work and health-related behaviors: 96 percent of the additional cardiac deaths that are related to the business cycle occur among those over age 65.

Table 3 provides additional hints as to which mechanisms may be most important among working-age adults. Among this group, motor vehicle accidents account for the bulk of the cyclicality in mortality. This could reflect either changes in individual behavior or externalities associated with increased economic activity (there are likely to be more cars on the road). However, the fact that the estimated coefficient on motor vehicle accidents is of similar magnitude across age groups points to the latter explanation. The other major contributor to cyclical deaths among working-age individuals is the category of “other.” Future work will investigate more fully the nature of this residual category, which accounts for a relatively large number of additional deaths (approximately 1,300) among working-age individuals.

III. Direct Estimation of “Own” and “Other” Effects

We continue to investigate the relative importance of “own” versus “other” behaviors by estimating equation (1) for five-year demographic subgroups, and adding to each regression the subgroup’s own unemployment rate along with the state average. If most of the changes in the mortality rate are driven by changes in individuals’ “own” behaviors, then we would expect the

TABLE 3—ESTIMATED RELATIONSHIP BETWEEN UNEMPLOYMENT AND MORTALITY BY CAUSE OF DEATH AND AGE
(Predicted additional deaths from a 1 percentage point increase in the unemployment rate)

Age	Cardiovascular	Respiratory	Infections
<i>Panel A</i>			
0–85	–4,260	–2,771	–1,453
0–0	–3	–16	–25
1–17	6	–4	0
18–24	3	–5	–2
25–34	–4	–17	–67
35–44	11	–4	–156
45–54	–9	–111	–183
55–64	–177	–205	–18
65–69	–197	–150	–35
70–74	–246	–216	–37
75–79	–409	–274	–144
80–84	–1,087	–495	–281
85+	–2,147	–1,276	–506
Age	Degenerative brain	Motor vehicle	Other
<i>Panel B</i>			
0–85	–2,686	–1,285	–1,587
0–0	5	–6	–190
1–17	0	–92	–50
18–24	1	–298	–88
25–34	2	–252	–171
35–44	–9	–217	–456
45–54	–27	–177	–587
55–64	–54	–85	–48
65–69	16	–52	64
70–74	–64	–32	54
75–79	–161	–40	97
80–84	–362	–29	4
85+	–2,032	–6	–216

estimated coefficient on the group unemployment rate to be large and negative relative to the estimated coefficient on the state average. This exercise is similar in spirit to that undertaken by Douglas L. Miller and Christina H. Paxson (2006), who focus on cross-sectional and (1980–1990) decadal-change variation.

Most of the estimated coefficients on own-group unemployment rates are positive. This is the opposite direction from what one would predict if the procyclical mortality pattern were generated by individuals taking on less healthful behaviors. None of the estimated “own” coefficients is both negative and statistically different from zero. While the lack of statistical significance of many of the own-group coefficients could be due to measurement error, this would not explain the change in signs, or the positive and significant coefficients for certain age groups. In contrast, all of the coefficient estimates on the

overall state average continue to be negative, and many are statistically significant. The point estimates on overall unemployment are similar to those presented in Table 1. These effects are particularly strong among the elderly, who have relatively weak labor force attachment. Because unemployment rates may not be the best measure of labor market activity for the elderly, we have also repeated this exercise using employment-to-population ratios to capture the business cycle, and get qualitatively similar results.

IV. Conclusion

This paper begins to explore mechanisms behind the procyclical mortality pattern that is observed in the United States. Two conclusions emerge that should guide future work in this area. First, the primary causes of death contributing to cyclical mortality fluctuations among

working-age adults are not typically associated with stress levels or health behaviors. As a result, it seems unlikely that changes in individuals' own labor force status, work, or health behaviors are the key determinants of aggregate mortality changes across the business cycle. Cyclical changes in mortality among working-age individuals stem mostly from additional motor vehicle accidents. Second, decompositions by age (and by cause and age) make clear that understanding procyclical mortality requires understanding mortality patterns among the elderly. Among this group, own work behavior seems less likely to be an important mechanism. Other factors, including pollution changes and changes in the quality, quantity, and nature of health care inputs over the business cycle, form an important target for future research.

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