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ARE RECESSIONS GOOD FOR YOUR HEALTH?

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ABSTRACT

This study examines the relationship between economic conditions and health. Fixed-effect models are estimated using state level data for the 1972-1991 time period. Health is proxied by total and age-specific mortality rates, as well as by 10 particular causes of death. Total mortality and nine of the ten sources of fatalities exhibit a procyclical variation, with suicides representing the important exception. The fluctuations in mortality are larger for 20-44 year olds than for older individuals. The predicted relationship between personal incomes and health is quite weak and is sensitive to the choice of model specifications, time periods and dependent variables. These findings suggest the possible importance of cyclical variations in the time costs of medical care or healthy lifestyles and of negative health effects of job-holding.

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Are Recessions Good For Your Health?

Economic contractions are costly in a variety of ways. Output declines and persons losing jobs frequently take new positions which are inferior to their previous employment. Thus, five years after permanent layoffs, dislocated workers earn at least 10 percent less than if the terminations had been avoided, with still larger decreases in the period immediately following displacements (Ruhm, 1991). Some unemployed workers lose employer-based health insurance (Horvath, 1987) and recessions may also reduce opportunities for promotions and result in deteriorating working conditions, decreased asset values, and higher crime rates. In addition, economic downturns may adversely affect health. Seminal, but widely disputed, research by Harvey Brenner (e.g. Brenner, 1973; 1975a; 1975b; 1979) indicates that unemployment rates are positively correlated with admissions to mental hospitals, infant mortality rates, and deaths due to cardiovascular disease, cirrhosis, suicide, and homicide.

This study provides new evidence on the relationship between macroeconomic conditions and health. Fixed-effect (FE) models are estimated using longitudinal data for the 1972-1991 time period. The unit of observation is the individual state and intrastate variations in unemployment rates and personal incomes are focused upon. Health is proxied by total and age-specific death rates, as well as by 10 particular causes of mortality. The use of fixed-effect models reduces potentially serious biases present in most previous time-series or cross-sectional analyses, resulting from the inability to control for omitted characteristics which influence health status and are spuriously correlated with economic conditions over time or across locations at a point in time.

The findings are quite surprising. Unemployment rates are *negatively* and significantly related to total mortality and to nine of the ten specific causes of fatalities, with suicides representing the important exception. The procyclical variation in death rates is stronger for 20-44 year olds than for older individuals. Since young adults are most involved in the labor force and likely to experience unemployment, this provides further evidence that macroeconomic conditions are causally related to health. Finally, while personal incomes are negatively associated with some types of mortality, the estimated effects are frequently statistically insignificant and are sensitive to the choice of specifications, samples, and outcomes.

The primary conclusion, that health improves as the economy deteriorates, raises questions about simple economic models emphasizing health capital or psychological theories focusing upon the stress induced by recessions. Instead, the results point to a potentially important role for cyclical fluctuations in the time costs of medical care or healthy lifestyles and raise the possibility that employment itself sometimes has adverse effects on health.

1. Economic Conditions and Health Status

Previous studies of the relationship between macroeconomic conditions and health usually focus on psychological determinants, generally hypothesizing that higher rates of joblessness create increased stress and risk-taking, which cause detrimental changes in physical and mental health.[1] It is surprising that more attention has not been paid to economic factors,

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[1] For example, Brenner and Mooney (1983, p. 1128) write: "recession increases the probability of a variety of losses and social changes that potentially threaten health in at least three ways: 1) poverty or lack of material resources to meet the ordinary requirements and extraordinary problems of life .... 2) psychological stress associated with (the) loss ... 3) attempts to alleviate psychological distress by medication with alcohol or legal and illegal drugs ... (which) tend to exacerbate existing morbidity and produce additional health problems."

given research on the demand for medical care, the choice of lifestyle behaviors, and health outcomes in the context of human capital investments (e.g. the seminal research by Grossman, 1972) and, more recently, of the economic determinants of addictive behaviors (e.g. Becker & Murphy, 1988; Akerlof, 1991; Chaloupka, 1991). In economic models, health is one argument in the utility function and is determined by stochastic shocks as well as by economic factors such as incomes and the relative price of medical care.

Health may deteriorate during economic contractions because earnings decline and health insurance is lost. Using similar reasoning, health is likely to be positively related to incomes when considering cross-sectional data at a point in time. However, since the utility function contains many non-health inputs, it is possible that at least some aspects of health will improve during recessions. For example, lower earnings reduce the consumption of normal goods such as drinking and driving, with the result that alcohol-involved vehicle fatalities fall (Ruhm, 1995).[2]

Moreover, there are several reasons why even broad measures of health could improve as the economy worsens. First, employment may have a direct adverse effect on health due to hazardous working conditions or job-related stress.[3] Second, the time price of medical care may fall if individuals who are not employed or are working fewer hours find it easier to schedule medical appointments for themselves or their dependents.[4] Similarly, some aspects of healthy

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[2] Skog (1986) and Wagenaar & Stef (1989) provide additional evidence that liquor consumption is positively correlated with incomes.

[3] Research emphasizing the stressful nature of work includes Holt (1982), Baker (1985), Karasek & Theorell (1990), and Lennon (1994). Fenwick & Tausig (1994) simultaneously examine the role of job-holding and unemployment as stressors. There is a vast economic literature on hazardous working conditions (see Viscusi, 1993 or Tolley, Kenkel, & Fabian, 1994 for reviews). French & Dunlap (1995) provide a rare economic study which focuses on job stress.

[4] Seminal studies examining the time price of medical care include Phelps & Newhouse (1974) and Acton (1975). There is some empirical evidence supporting the possibility that the time-

lifestyles (e.g. regular exercise) may be time-intensive and so easier to undertake during periods of reduced work hours. Third, low-income persons losing jobs which lack employer-provided health insurance may sometimes become eligible for Medicaid, lowering the money price of medical care.[5]

2. An Economic Model of Health

This section develops a simple model indicating the potential relationships between macroeconomic conditions and health, with special attention paid to the division of time between market uses and nonmarket activities. Also included are several extensions to the basic model which are relevant to the empirical analysis that follows.

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costs of medical care rise during periods of high employment. For instance, Mwabu (1988), shows that agricultural workers in a rural health district in Kenya are less likely to see medical providers care during the busy season, when wages and work hours are high, than during the rest of the year, when earnings and labor supply are lower. Mullahy (1996) finds that employment and work hours are negatively related to the probability that individuals in the U.S. receive flu shots (a preventive health measure), after controlling for education, self-reported health status, and a variety of demographic characteristics. He also reports a set of instrumental variable results in which this finding is reversed. However, the IV estimates may be suspect since the state unemployment rate, which is the key instrument causing the sign reversal, may be correlated with the health outcome in his cross-sectional data. Supporting this possibility, the overidentification test performed by Mullahy rejects the null hypothesis of exogeneity at the .06 level for employment status and at the .16 level for work hours, in specifications where the only instruments are the state unemployment rate and its square.

[5] I am unaware of research directly examining this possibility. The closest to it is Yelowitz's (1995) analysis of the effects on labor supply of the "Medicaid notch" in the budget constraint of persons with low earnings potential. There is evidence that persons holding low-wage jobs have exceptionally high probabilities of being uninsured. For instance, 36% of nonelderly individuals in households with family incomes between $10,000-$14,999 lacked health insurance, in 1994, as compared to 28% of their counterparts with incomes of $5,000-$9,999 (Fronstin & Rheem, 1996). The difference occurs because higher probabilities of obtaining employer-based health insurance (26% vs. 13%) are more than offset by reductions in Medicaid eligibility (29% vs. 51%). Conversely, jobs leavers sometimes lose health insurance (Gruber & Madrian, 1995). Currie & Gruber (1996) show that Medicaid eligibility is positively correlated with the utilization of medical care and negatively related to rates of child mortality.

Individuals maximize a utility function U(H,Z), where H is health, Z a composite consumption good, and (with subscripts denoting partial derivatives) U_H and U_Z (U_{HH} and U_{ZZ}) are positive (negative). Health depends on baseline status (B), nonwork time (R), and medical care (M), with H_B, H_R, and H_M greater than zero. The budget and time constraints are $Y = WL = P_ZZ + P_MM$ and $R = T - L$, for Y, T, L, W, P_Z and P_M indicating income, total hours available, work hours, the hourly wage rate, and prices of the consumption good and medical care.[6]

Substituting in for L, the optimization problem is described by:

$$(1) \qquad \max_{M,R,Z} \; U(H(M,R,B),Z) \qquad \text{subject to} \quad W(T-R) = P_ZZ + P_MM \; .$$

First order conditions imply choosing M, R, and Z such that $U_HH_M/P_M = U_HH_R/W = U_Z/P_Z$, at which point the health improvement associated with the last dollar of medical care or leisure time provides the same marginal utility as the final dollar spent on the consumption good.

Economic slowdowns can affect health in at least three ways. First, the relative price of medical care may change. Second, baseline health may rise or fall (e.g. due to changes in stress or risk-taking). Third, the wage rate is likely to decline. An increase in P_M lowers the optimal amount of medical care and the level of health, except in the rare case where medical care is a Giffen good. A decrease in baseline health may cause the desired level of medical care to rise but generally not by enough to restore health status to its previous level. Finally, if the substitution effect dominates, a reduction in wages decreases desired work hours. This improves health by lower the time costs of medical care and other health preserving activities but also

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[6] The budget constraint can easily be modified to allow for nonlabor income.

reduces incomes, which operates in the opposite direction. Thus, the overall effect on health is ambiguous.[7]

If the consumption good has a direct effect on health, the health production function becomes H=H(M,R,B,Z) and the solution to the maximization problem is $U_H H_M/P_M = U_H H_R/W = (U_H H_Z + U_Z)/P_Z$. Consumption of Z now has an indirect effect on utility, through its impact on health. A decrease in W generally causes both M and Z to decline. If $H_Z > 0$, it therefore becomes more probable (than when $H_Z = 0$) that H will fall. Conversely, if $H_Z < 0$, a reduction in earnings potential is more likely to result in improved health.

Following Grossman (1972), the determination of health can be made dynamic by assuming that baseline health status is a function of previous investments in medical care and a contemporaneous error term. To illustrate, let baseline status at time t be determined by:

$$(2) \qquad B_t = B(B_{t-1}, M_{t-1}, \delta, \varepsilon_t) = (1-\delta)B_{t-1} + m_{t-1} + \varepsilon_t ,$$

where δ is the depreciation rate of health capital, ε a stochastic shock, and $m = M^{\alpha}$, with $0 < \alpha < 1$. Backwards substitution implies:

$$(3) \qquad B_t = (1-\delta)^n B_{t-n} + \Sigma_{i=0}^{n-1} (1-\delta)^i [m_{t-i-1} + \varepsilon_{t-i}] .$$

If n is large, the first term on the right-hand-side of (3) approaches zero and baseline status at t depends completely on the sequences of medical investments and shocks. In this model,

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[7] The model is easily extended to allow for multiple aspects of health. Redefine the utility function as U(**H**,Z), where $\mathbf{H} = \{H_1, H_2, ..., H_n\}$, for H_j the level of the jth health attribute. Utility is maximized by choosing **H** and Z such that $U_j M_j/P_j = U_H H_R/W = U_Z/P_Z$, for $U_j = \delta U/\delta H_j$ and $M_j = \delta H_j/\delta M$. The analysis of the effects of the macroeconomy remains essentially unchanged, although the possibility of differential effects on alternative health characteristics is introduced.

economic downturns have persistent effects on health. Changes in earnings and money or time prices affect medical care in the current period but also influence future baseline health.

Mortality rates are imperfect indicators of health. Nonetheless, fatality rates and health status are likely to be closely related. For instance, if death occurs when $H^* + \varepsilon \leq H_{min}$, for H_{min} a minimum threshold health level, H^* the expected level of health, and ε an error term, the probability of mortality is:

$$\Pr(\text{mortality}) = \Pr(\varepsilon \leq H_{min} - H^*) = F(H_{min} - H^*),$$

where F(.) is the cumulative distribution function of the error term. Implicitly, by their selection of M, R, and Z, individuals choose a likelihood of death and fatality rates vary with economic conditions in the same direction as H.

3. Methodological Issues

Studies using aggregate time-series data to examine the relationship between economic conditions and health typically estimate some variant of:

$$H_t = \alpha + X_t\beta + E_t\gamma + \varepsilon_t, \tag{4}$$

where H is the measure of health, E the indicator of economic conditions, X a vector of covariates, and ε is an error term. For instance, in Brenner (1975a), health outcomes are proxied by mortality rates, X contains controls for income and government spending, and E includes current and lagged unemployment rates. The key coefficients of interest are those on unemployment. Brenner uncovers a strong positive correlation between the rate of joblessness and mortality, leading him to conclude that health varies procyclically.

Previous researchers (e.g. Gravelle, et. al., 1981; Stern, 1983; Wagstaff, 1985; Cook & Zarkin, 1986) have pointed out serious technical flaws in Brenner's analysis and later studies

(e.g. Forbes & McGregor, 1984; McAvinchey, 1988; Joyce & Mocan, 1993) which correct for these problems fail to replicate his findings.[8] Instead, the results are sensitive to the choice of countries, time periods, and proxies for health. Significantly, elevated unemployment is frequently positively rather than negatively correlated with health. This fragility of results should not be surprising since research using aggregate time-series data contains a fundamental shortcoming which severely limits its usefulness. Any lengthy series is likely to contain omitted variables which are spuriously correlated with the regressors and outcome variables.[9]

To show this bias concretely, assume that the "true" model is:

$$H_t = \alpha + X_t\beta + E_t\gamma + Q_t + \phi_t \,, \tag{5}$$

where Q is unobserved and ϕ a "white noise" disturbance. If E and Q are related according to $Q_t = E_t\delta + \mu_t$, where μ is uncorrelated with E, the error term in (4) is $\varepsilon_t = E_t\delta + \mu_t + \phi_t$ and the estimated unemployment coefficient is $\hat{\gamma} = \gamma + \delta$, which is upwards (downwards) biased if the unobserved factor is positively (negatively) correlated with E.

Cross-sectional data suffers from a similar problem. For instance, Junankar (1991) estimates:

$$H_j = \alpha + X_j\beta + E_j\gamma + \varepsilon_j \,, \tag{6}$$

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[8] Criticisms include Brenner's method of choosing lag lengths, the hypothesized pattern of lag coefficients, choice of covariates, and the plausibility of his results. For instance, the strongest effects are observed for infants and the elderly, the groups who are least likely to work and so for whom macroeconomic conditions are expected to have the smallest health impact.

[9] For example, much of the variation in unemployment occurring during the four decades (beginning in the 1930s) covered by Brenner's research resulted from dramatic reductions in joblessness following the great depression. During this same period, mortality declined due to improvements in nutrition and increased availability of antibiotics. Failure to control for these factors leads Brenner to overstate the detrimental health impact of unemployment.

where mortality rates are again the dependent variable and j indicates a specific region/occupation population subgroup in Britain. In this case, $\hat{\gamma}$ is biased if region-group unemployment rates are correlated with unobservables which influence fatalities. For example, unskilled blue collar workers are likely to experience the most joblessness but also to have the highest mortality due to lack of education, unhealthy lifestyles, etc., which are associated with but not caused by differences in unemployment.

As an alternative, many researchers directly contrast the health of unemployed and employed persons (e.g. Moser, et. al. 1984; Hammärstrom, et. al. 1988; Dooley, et. al., 1988; Janlett, et. al., 1991; Pierce, et. al., 1994; Fichtenbaum, 1995). The use of microdata has the advantage of allowing for more fully specified models but introduces two other problems. First, macroeconomic conditions may affect the health of a broad cross-section of the population, not just those who are unemployed. For instance, jobs may become more stressful due to economic restructuring or because of increased fear of future layoffs. Second, health is unlikely to be exogenous to individual labor force status (e.g. if unhealthy individuals are more likely to experience unemployment).[10]

This study avoids many of the aforementioned problems by estimating fixed-effect models using pooled time-series cross-sectional data for the 50 states and the District of

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[10] To illustrate, assume that health and economic status are simultaneously determined by:

$$E_j = a_0 + a_1 H_j + \varepsilon_{1j}$$

and

$$H_j = b_0 + b_1 X_j + b_2 E_j + \varepsilon_{2j},$$

with all variables defined as above. Substituting and solving for E yields:

$$E_j = (1-a_1 b_2)^{-1}(a_0 + a_1 b_0 + a_1 b_1 X_j + a_1 \varepsilon_{2j} + \varepsilon_{1j}).$$

Estimates of the reduced-form health equation, or its multivariate counterpart specified by (6), therefore provide biased estimates of the effect of unemployment, since E and e_{2j} are correlated.

Columbia. One benefit of using state-level data is that fluctuations in local economic conditions are almost certainly exogenous to health. A disadvantage is that the set of covariates controlled for will be less comprehensive than if microdata were utilized. This latter shortcoming is partially surmounted by using FE techniques, which automatically account for interstate differences which remain constant over time, and by including explanatory variables for some population characteristics.

The models estimated are of the form:

$$H_{jt} = \alpha_t + X_{jt}\beta + E_{jt}\gamma + S_j + \varepsilon_{jt}\,, \tag{7}$$

where the subscripts j and t index the state and time period, H and E are proxies for health and macroeconomic conditions, X is a vector of supplementary regressors, and ε is the regression disturbance. The fixed-effect, S_j, controls for time-invariant characteristics which differ across states, α_t accounts for time-varying factors which influence health throughout the country, and $\hat{\gamma}$ captures the impact of *within-state* deviations in economic conditions.[11]

4. Data

The data analyzed are for the 50 states and the District of Columbia and cover the 1972-1991 time period. The outcomes are total mortality rates, fatality rates for three age groups (20-44, 45-64, and $\geq$65 year olds), and deaths due to 10 specific causes: 1) malignant neoplasms; 2) major cardiovascular diseases, 3) pneumonia and influenza; 4) chronic liver disease and cirrhosis of the liver; 5) motor vehicle accidents; 6) other accidents and adverse effects; 7) suicide; 8)

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[11] Fixed-effect models have occasionally been utilized to investigate the relationship between economic conditions and specific aspects of health. For instance, Ruhm (1995) and Evans & Graham (1988) include FE specifications in their studies of motor vehicle fatalities and Cook & Tauchen (1982) do so when investigating cirrhosis mortality.

homicide and legal intervention; 9) infant mortality (deaths within the first year); and 10) neonatal mortality (deaths within the first 28 days).

The 10 specific sources of fatalities account for more than three-quarters of all deaths. Cancer, heart disease, and pneumonia/influenza represent three of the main sources of physical illnesses which cause mortality. Cirrhosis, suicides, and motor vehicle or other fatal accidents combine the effects of lifestyles and physical or mental health problems in various degrees. Homicides provide one indication of the interaction between crime and the economy. Finally, infant and neonatal mortality are partially determined by prenatal and postnatal care. Information on death rates is obtained from various issues of *Vital Statistics of the United States*, which is published annually by the U.S. Bureau of the Census.

The use of mortality rates has several advantages. They represent objective indicators of health, for which data are readily available and which have been widely used in this and other areas of previous research.[12] However, as Bound (1991) and others have pointed out, death rates imperfectly measure health status to the extent that they fail to capture the effects of illnesses (e.g. arthritis) or medical interventions (e.g. hip replacements) which influence the quality of life but have little or no influence on longevity or result from causes (e.g. accidents) which may be unrelated to previous health. These problems are partially overcome by considering various types of fatalities, rather than just the total death rate.

Unemployment rates and employment-to-population (EP) ratios are alternatively used as proxies for macroeconomic conditions. Unemployment rates furnish direct information on the

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[12] For instance, mortality data have been used by Anderson & Burkhauser (1985) and Burtless (1987) to examine the effects of health on retirement behavior and by Idler, et. al. (1990) to investigate the information content of self-evaluations of health.

ability of labor market participants to find jobs and have been frequently utilized in previous research. EP ratios are included because some economists (e.g. Clark & Summers, 1982) believe that they provide a more accurate measure of labor market conditions for groups, such as young workers, who frequently enter and exit the labor force. A consistent (unpublished) series on unemployment rates and employment-to-population ratios was provided to me by the U.S. Bureau of Labor Statistics. The data refer to the noninstitutionalized civilian population aged 16 and over, with information on some smaller states first provided in 1976. Per capita personal incomes, adjusted to $1987 using the implicit price deflator, are also incorporated in some models, with data obtained from U.S. Department of Commerce (1989) and various issues of the *Statistical Abstract of the United States*.

Many of the regressions also control for demographic characteristics. These include the percentage of the state population with three levels of educational attainment (high school dropout, some college, college graduate), in two ethnic groups (black, Hispanic), and two age categories (<5, ≥65 years old). *Ceteris paribus*, mortality rates are expected to be relatively low when the proportions of educated individuals, whites, and young persons are relatively high.

Ethnic status and age are measured over the full population whereas educational attainment refers to persons aged 25 and higher. These variables are constructed using census data for the years 1970, 1980, and 1990, obtained from various issues of the *Statistical Abstract of the United States* and, for the percentage of Hispanics in 1970, from U.S. Bureau of the Census (1973). Values for the non-census years were extrapolated by assuming a constant rate of growth between census periods. The method of reporting educational attainment changed between 1980 and 1990. In 1970 and 1980, information on years of college was provided; in

1990 the data indicated whether a college degree had been obtained. For this analysis, the "some college" group refers to persons with 1 to 3 years of university education in 1970 and 1980 and to those with more than one year of college but no bachelor's degree in 1990. The "college graduate" group are those with four or more years of college in the two earlier census years and with a bachelor's degree in the latest one. Given that a substantial fraction of students take more than four years to obtain a degree, this procedure is likely to moderately overstate the percentage of college graduates prior to 1990.

Summary statistics for the variables used in the analysis, weighted by the total resident population in each state, are displayed in Table 1 and are largely self-explanatory. The fatality rates refer to deaths per 100,000 persons, except for infant and neonatal mortality which are per 1000 live births. For cause-specific mortality, the listings in parentheses refer to the Ninth Revision of the International Classification of Diseases (ICD-9 categories).

Total mortality and most specific sources of fatalities declined substantially during the sample period. For instance, as shown in Figure 1, the overall death rate fell more than 8% over the 20 years (decreasing from 943.2 to 860.3 per 100,000 persons), with substantially larger 27% and 36% reductions for mortality due to heart disease and vehicle crashes respectively. By contrast, cancer fatalities increased 23% between 1972 and 1991.[13]

A preliminary indication of the relationship between macroeconomic conditions and health is provided in Figure 2, which displays *national* total mortality and unemployment rates in each year. The variables are detrended, using a linear time trend, and normalized by subtracting

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[13] Deaths from influenza/pneumonia, suicides, and homicides also rose over time (by 3%, 2%, and 12% respectively). Fatalities from all other specific causes trended downwards, as did age-specific mortality rates (falling by 18%, 30%, and 17% for 20-44, 45-64, and $\geq$65 year olds).

the mean value of the detrended variable and dividing by its standard deviation.[14] Previewing the econometric results to follow, the figure illustrates a strong *inverse* relationship between unemployment and fatality rates. Thus, deaths decline dramatically during the 1975 and 1982/3 recessions, while increasing throughout much of the economic recovery of the middle 1980s.[15] Although the national data may suffer from the aforementioned problems of confounding, these findings raise questions about the common belief that health declines during economic downturns.

The fixed-effect estimates exploit within-state variations in economic conditions and have the potential to improve on (aggregate) time-series analyses if there are substantial independent macroeconomic fluctuations across states over time. To illustrate that this condition is met, figure 3 summarizes the patterns of unemployment in four large states in different regions of the country (California, Illinois, New York, and Texas). As shown, Illinois had relatively low rates of joblessness during the 1970s but more elevated unemployment subsequently, whereas the opposite pattern is observed for California and New York (until 1991). Additional evidence of the heterogeneity of local economic performance is obtained by calculating the squared-correlation coefficient between national and state unemployment rates for each of the 50 states and the District of Columbia. The R^2 exceeds .9 in just 3 cases and is .75 or higher in only 14. Conversely, the R^2 are in the ranges of .5 to .75, .25 to .5, and <.25 for 17, 13, and 7 states.[16]

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[14] Unemployment exhibits virtually no time trend, decreasing by .003% per year. Conversely, total mortality is estimated to decline by 3.1 deaths per 100,000 persons annually.

[15] A regression of the natural log of total mortality on unemployment rates, using national data (n=20), yields an unemployment coefficient of -.0100 with a standard error of .0041.

[16] North Carolina's unemployment most closely follows the national rate (R^2=.94); the weakest association is for Wyoming (R^2=.04).

5. Econometric Estimates of Total Mortality Rates

This section presents econometric estimates of the relationship between macroeconomic conditions and total mortality. Most of the regressions are fixed-effect models of the form specified by equation (7), with the dependent variable measured as the natural logarithm of the death rate (per 100,000 persons). Table 2 displays four specifications of the basic model, all of which include vectors of time and year dummy variables. Unemployment rates are the exclusive proxy of macroeconomic conditions in columns (1a) and (2a); models (1b) and (2b) also hold personal incomes constant. Specifications (2a) and (2b) control for population characteristics (age, education, and ethnic status), while (1a) and (1b) do not. Throughout the analysis, heteroscedasticity is corrected for by weighting observations by the square root of state populations.[17]

Unemployment rates are *negatively* and statistically significantly correlated with total mortality in all specifications. A one percentage point increase in joblessness reduces the predicted death rate by 0.8% in model (1a) and by 0.6% in column (2a). Personal incomes and fatality rates are also inversely related; however, the estimated effect is quite weak and is statistically insignificant when population characteristics are held constant (column (2b)). As a result, controlling for incomes does not substantially alter the predicted relationship between

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[17] I also experimented with using an alternative three-stage estimation procedure for the specifications shown in table 2. In the first stage, the equations were estimated by OLS. Second, the squared residuals from these models were regressed against a constant and the reciprocal of the state population. The constant term never differed significantly from zero while the population variable was always highly significant, as expected if the heteroscedasticity results from sampling error due to differences in population sizes. Third, the square root of the inverse of the predicted values from the second-stage regression were used as weights in a final set of estimates. The resulting unemployment and personal income coefficients were virtually identical to those displayed in the table.

unemployment and fatalities. The demographic coefficients generally conform to our expectations. Mortality increases with population age, there is some evidence that education has a protective effect on health, and blacks have higher death rates than whites; however, fatalities are negatively related to intrastate changes in the Hispanic population share.[18]

These results suggest that health improves during economic downturns and deteriorates during expansions. There are at least four reasons why this conclusion could be incorrect. First, unemployment rates may poorly proxy macroeconomic conditions. Second, the FE models might inadequately control for confounding factors. Third, the equations could be misspecified (e.g. by failing to account for dynamic effects). Fourth, total mortality rates could poorly indicate important aspects of health which fluctuate procyclically. The subsequent econometric analysis investigates whether any of these potential problems explains the observed countercyclical variation in health. The tentative but fairly strong conclusion is that they do not.

Table 3 summarizes 16 alternative specifications of the total mortality equation. The top and middle panels report results for models which are identical except that the former controls for unemployment rates while the latter holds constant the employment-to-population ratio. All of the equations include demographic characteristics and columns (1b), (2b), (3b), and (4b) also control for personal incomes. Vectors of state fixed-effects and year dummy variables or time trends are variously incorporated, as described at the bottom of the table.

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[18] The decline in the unemployment coefficient occurring when demographic characteristics are held constant is entirely due to controlling for age. This can be seen noting that the estimated unemployment effect is smaller, ranging between -.0049 and -.0053, when age is held constant but ethnic status and education are not, than when race and schooling are also included as regressors.

Since unemployment rates rise and employment-to-population ratios are negatively related, the coefficients on these variables should have the opposite signs. The parameter estimate of the latter is also expected to be smaller in magnitude than that of the former, since the EP ratio exhibits a greater cyclical variation. This occurs because departures from the labor force prevent some "discouraged" workers from being counted as unemployed.

The findings of Table 3 can be summarized as follows. First, mortality exhibits a statistically significant procyclical variation in all models which include state fixed-effects.[19] Second, this result is robust to the use of unemployment rates or employment-to-population ratios as the indicator of economic conditions. In the FE models, a one percentage point decrease (increase) in unemployment rates (EP ratios) raises expected mortality rates by between 0.5% and 0.6% (0.2% and 0.4%).[20] Third, the parameter estimate for personal incomes is sensitive to the choice of specifications and is often statistically insignificant.

Although the fixed-effect models automatically account for all time-invariant sources of heterogeneity, they do not purge potential biases resulting from within-state changes in omitted factors which are correlated with unemployment rates. As shown in Table 2, the inclusion of controls for a limited set of demographic characteristics reduces the estimated unemployment

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[19] Conversely, fatalities are uncorrelated with unemployment rates and positively associated with EP ratios when fixed-effects are excluded (see columns (2a) and (2b)), suggesting a positive correlation between cross-state variations in mortality and joblessness.

[20] Since mortality rates are restricted to range between zero and one, I also estimated grouped data logit models for equations with the full set of regressors. The dependent variable in the logit models is $H_{jt}/(1- H_{jt})$ and observations are weighted by $[H_{jt}(1- H_{jt})n_{jt}]^{-1/2}$, for n_{jt} the population of state j at time t. The coefficient (standard error) obtained was -.0058 (.0005) for the unemployment rate and .0032 (.0006) for the EP ratio. The magnitudes of the predicted effects are quite similar to those obtained for the log-linear models. For instance, evaluating the other regressors at their sample means, raising the unemployment rate from its average value to one percentage point higher is predicted to reduce total mortality by 0.6%,.

effect by approximately 30% (compare columns (1b) and (2b)). This indicates that at least some important covariates fluctuate within-states over time and raises the possibility that the remaining observed procyclical variation in mortality rates is the result of confounding.

Such a spurious relationship might exist if unemployment rates and unobserved characteristics share a common time trend. This possibility was dealt with in two ways. First, the equations were reestimated with the addition of controls for state-specific time trends.[21] The results are summarized in columns (1a) and (1b) of Table 4. Second, the sample was split into shorter time periods. This is expected to reduce the influence of the unobserved factors since fewer years of data will decrease the size of the trend component relative to the fluctuations around the trend. Specifications (2a) and (2b) display estimates for the 1971-1982 time span and (3a) and (3b) for 1983-1991 period.[22] Finally, columns (4a) and (4b) refer to a sample consisting of the 10 largest states (based on 1991 population). The rationale for eliminating small states is that measurement-error problems may be more severe for them.

Mortality varies procyclically in all of the specifications shown in Table 4. The inclusion of state-specific time trends has virtually no effect on the predicted health impact of unemployment; the coefficient is -.0058, which compares with -.0054 to -.0056 for corresponding specifications which exclude state time-trends (columns (2a) and (2b) of Table 2). The estimated effect of unemployment is smaller in the early period and for the 10 largest states than for the full sample. However, even in these cases, a one percentage point increase in

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[21] The regression model is: $H_{jt} = \alpha_t + X_{jt}\beta + E_{jt}\gamma + S_j + S_jT + \varepsilon_{jt}$, where T indicates the number of years since 1971 (i.e. T equals the survey year minus 1971).

[22] The cutoff point was chosen to obtain the most nearly equal sample sizes. The earlier period covers more years because unemployment data are missing for some states prior to 1976.

joblessness reduces predicted deaths by a statistically significant 0.3% (see models (2b) and (4b)). Moreover, personal incomes are *positively* and significantly related to fatalities for all of the stratified estimates, in contrast with the insignificant negative results obtained for the full sample. The income effect is especially strong for the 1971-1982 period and for the 10 largest states, resulting in a particularly sharp rise in the magnitude of the unemployment coefficient, when personal incomes are excluded from the model (specifications (2a) and (4a)).

6. Dynamic Effects

Up to this point, economic conditions have been assumed to have only a contemporaneous effect on mortality. For some types of fatalities (e.g. accidents or homicides) this restriction seems reasonable. However, for many diseases, death is likely to be the end result of a lengthy process, suggesting that changes in the macroeconomy may affect health over a period of several years.[23] These dynamic effects are considered in Table 5, which extends the analysis by allowing lags or changes in macroeconomic conditions to affect mortality. The regressions are estimated over a "balanced" panel of observations for which data are available on the contemporaneous and lagged variables.[24] All of the remaining econometric estimates include

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[23] This does not imply that current economic conditions have no effect. For instance, if death occurs when the stock of health capital falls below some minimum threshold level, contemporaneous shocks to baseline health status will result in changes in mortality rates. Empirical evidence indicates that changes in economic factors can have rapid and substantial effects on fatalities resulting from diseases which develop slowly over time. For instance, Cook & Tauchen (1982) show that cirrhosis mortality responds rapidly to changes in alcohol tax rates. Moreover, Willich, et. al. (1994) indicate that the heart attacks of working individuals peak on Mondays, suggesting that even extremely short-term changes in employment status can affect health.

[24] Thus, the estimates in columns (1a) and (1b) are equivalent to specifications (2a) and (2b) of Table 2, except that the sample excludes the year in which the unemployment data first becomes available (1972 for most states).

controls for state and time dummy variables and, unless otherwise noted, demographic characteristics.

There is some evidence that the economic conditions influence health over a period of years. In particular, lagged unemployment is positively related to current mortality whereas the reverse is true for personal incomes at year t-1. Nevertheless, there is little reason to believe that the failure to control for these dynamic effects explains the observed procyclical variation in mortality rates. In particular, the predicted effect of current unemployment, in models with lags excluded, is similar to the sum of the parameters at t and t-1, when both are controlled for. For instance, the year t unemployment coefficient in specification (1a) is -.0050, while the sum of the lagged and current parameters is an almost identical -.0047 in column (2a). Similarly, the contemporaneous income effect is .0014 in model (1b), whereas the sum of the personal income coefficients at t and t-1 is -.0019 in specification (2b). Moreover, as shown in columns (3a) and (3b), growth in unemployment between years t-1 and t is associated with reductions in mortality, while rising personal incomes are predicted to have the opposite effect. This provides a further indication that health deteriorates as economic conditions improve.[25]

7. Age-Specific Death Rates

The macroeconomy could affect the health of persons of all ages. For instance, resources to support Medicare, Medicaid, and other programs which pay for medical services received by

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[25] The models were also estimated with longer lags or changes in economic conditions. These regressions suggest that the macroeconomy affects mortality up to three years in the future and the pattern of coefficients indicates that changes in rather levels of prior economic conditions are most important. Thus, increases in unemployment at t, t-1, or t-2, compared to the previous year, are associated with lower rates of year t mortality, whereas corresponding income growth is correlated with higher fatality rates. Conversely, the coefficients on levels of prior unemployment or personal incomes are quite sensitive to the specification of the lag structure.

senior citizens may vary with the state of the economy. Nevertheless, if economic fluctuations cause changes in health, the latter are expected to be concentrated among prime-age workers and particularly on relatively young adults, who have the highest rates of labor force participation and joblessness. Conversely, evidence of stronger impacts for older individuals would raise concern that the observed relationship is spurious.

Table 6 reports the results of total mortality equations estimated separately for persons aged 20-44, 45-64, and $\geq$65 years old. The specifications conform to those in Table 2. Since the youngest of the three age groups is most involved in the labor force, the cyclical fluctuations in fatality rates are anticipated to be the largest for them.[26] The econometric estimates confirm this expectation. Controlling for personal incomes and demographic characteristics, a one percentage point rise in unemployment lowers the predicted death rate of 20-44 year olds by 1.3%, while having no effect on persons aged 45-64 and reducing the fatalities of individuals 65 years old and over by just 0.3% (see column (2b)).[27]

When personal incomes are left out of the model, the estimated unemployment effect rises by around 40% for 20-44 year olds (i.e. a one percentage point increase in unemployment

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[26] In 1994, 82.7% of 20-44 year olds participated in the labor force, as compared to 71.5% of those aged 45-64 and 12.4% of persons 65 and older (U.S. Department of Labor, 1995). In the same year, unemployment rates were 6.1%, 4.0%, and 4.0% for the three age groups.

[27] Once personal incomes are held constant, the age-specific unemployment coefficients are essentially unaffected by the inclusion of demographic characteristics (compare columns (1b) and (2b)). This suggests that the estimated unemployment effects are unlikely to be substantially reduced by the addition of controls for other characteristics (e.g. sex) whose within-state variations over time are highly correlated with age. The crudeness of mortality as a proxy for health may explain why deaths of the oldest group exhibit greater cyclical variation than those of 45-64 year olds. In particular, a given increase in disease severity is more likely to be fatal for older, and typically frailer, individuals than for their middle-age counterparts. A reduction in health status is even less likely to cause death for still younger persons but, as shown below, a substantial portion of their mortality is from causes other than disease.

reduces expected mortality rates by 1.8%) but remains essentially unchanged for 45-64 or ≥65 year olds (see column (2a)). This occurs because incomes are strongly *positively* correlated with death rates for the youngest group, while being unrelated or negatively associated for the two older categories. Thus, a $1000 rise in incomes increases the predicted fatalities of 20-44 year olds by 3.4%, while decreasing those of persons aged 45-64 and ≥65 by 0.6% and 0.1%.

8. Cause-Specific Mortality Rates

This section investigates how deaths from 10 specific causes vary with macroeconomic conditions. The analysis reveals that the procyclical fluctuations in mortality are broadly distributed, rather than being limited to a few sources of fatalities, with the largest effects observed for those types of death experienced relatively often by young adults. Examination of the correlations of regression residuals, across causes of death, demonstrates that the cyclical changes are unlikely result from common omitted variables which generate a spurious relationship between state unemployment rates and the various types of fatalities.

The age-distribution of mortality, in 1990, is displayed in Table 7. Deaths from most causes are concentrated among the elderly. Seventy-two percent of all fatalities occur to persons beyond 64 years of age and 89% among individuals 45 and older. Over 90% of mortality from heart disease, cancer, and influenza or pneumonia involves persons past the age of 44. By contrast, younger individuals account for more substantial shares of some types of deaths. Thus, 69% of fatal motor vehicle crash victims are younger than 45, as are 42% of those involved in other deadly accidents, 56% of suicides, and 83% of homicides.

Fixed-effect estimates for the 10 causes of mortality are summarized in Table 8, with the specifications corresponding to those in Table 2. As mentioned, deaths from heart disease,

cancer, and influenza/pneumonia are concentrated among the elderly and so are expected to be relatively weakly associated with economic conditions. Conversely, stronger relationships are anticipated for fatalities from liver ailments, motor vehicle and other accidents, suicides, and homicides, since they afflict a greater share of prime-age workers. Substantial cyclical fluctuations in infant and neonatal mortality rates are also expected, since prenatal and postnatal care is largely provided by relatively young adults.

Unemployment rates are negatively and significantly related to nine of the ten causes of death, confirming that many aspects of health deteriorate as the economy improves. Moreover, those sources of mortality most likely to be experienced by 15-44 year olds exhibit greater cyclical variation than those dominated by more elderly individuals. For instance, a one percentage point increase in the state unemployment rate is predicted to reduce fatalities from motor vehicle crashes, other accidents, homicides, and liver ailments by 2.4%, 1.7%, 1.5%, and 0.8% respectively (in model (2b)), whereas expected deaths from cardiovascular disease, malignant neoplasms, and influenza or pneumonia fall by just 0.2% to 0.5%.[28] Infant and neonatal mortality represent a middle ground, declining 0.5% to 0.8% per one percentage point increase in unemployment.

In contrast to the other outcomes, suicides are predicted to *increase* by 0.7%, in specification (2b), for each one percentage point rise in state unemployment rates. This

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[28] The findings are consistent with patterns of disease progression over time. For instance, a stronger unemployment effect is observed for heart disease than for cancer, which makes sense since the former is more responsive to short-term changes in medical care or lifestyles than is the latter. Differential effects of the macroeconomy could also occur for reasons unrelated to the age-distribution of deaths. For example, motor vehicle fatalities may vary over the business cycle because persons *of all ages* drink and drive less during recessions, rather than because young adults are most likely to be involved in deadly crashes.

divergent result is particularly interesting because many of the other dependent variables primarily proxy physical health or non-disease sources of death, whereas suicides are among the most widely studied indicators of mental health.[29] It therefore raises the possibility that worsening economic conditions have negative effects on at least some facets of mental health, while improving most aspects of physical well-being. Equally important, the finding suggests that previous analyses of suicides should be interpreted cautiously and may provide little indication of the effects of the macroeconomy on more general aspects of health.

Table 8 supplies further evidence of the ambiguous relationship between personal incomes and mortality rates. When demographic characteristics are controlled for, the income coefficients are negative and statistically significant for three causes of death (heart disease, cancer, and neonatal mortality), positive and significant for two (influenza/pneumonia and motor vehicle fatalities), and differ insignificantly from zero for the remaining five sources (see model (2b)).

The cause-specific mortality equations can be further used to examine whether the observed macroeconomic effects are the result of common omitted variables. To illustrate, consider two types of mortality which are determined by:

(8a) $$H_1 = X\beta_1 + E\gamma_1 + Q\delta_1 + \phi_1 \qquad \text{and}$$

(8b) $$H_2 = X\beta_2 + E\gamma_2 + Q\delta_2 + \phi_2 ,$$

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[29] Theoretical and empirical research on the relationship between economic conditions and suicide dates back to Durkheim (1897). More recent studies include Hammermesh & Soss (1974); Brenner, (1979); Moser, et. al. (1984); Dooley, et. al. (1989) and Yang (1992), among many others. Some of the other sources of mortality (e.g. motor vehicle crashes and certain types of heart disease) may also have a mental health component.

where the subscript indexes the type of death, Q is a common unobserved factor, and ϕ_1 and ϕ_2 are "white noise" error terms. The state and year subscripts, intercept term, fixed-effects, and time-effects are all excluded for convenience.

Since Q is unobserved, the error terms for regressions on the observable characteristics, $\varepsilon_1 = Q\delta_1 + \phi_1$ and $\varepsilon_2 = Q\delta_2 + \phi_2$, will be positively correlated as long as δ_1 and δ_2 have the same sign. If Q and E are related, the unemployment coefficients will also be biased, providing a potential explanation for the observed relationship between joblessness and health. In particular, if the procyclical variation in mortality rates is the result of common excluded characteristics, we would expect the cross-equation residuals to be strongly positively correlated (except possibly for suicides). Conversely, negative associations or weak positive relationships suggest that shared confounding factors do not explain the cyclical fluctuations.[30]

The matrix of cross-equation residual correlations, displayed in Table 9, provides little indication of substantial bias caused by common omitted factors. More than two-fifths (23 out of 55) of the residual correlations have negative rather than positive signs and the (unweighted) average value of the 55 correlation coefficients is just .049. It may be desirable to exclude the total mortality and suicide equations from these counts, since the former represents the aggregation all individual causes of death and the latter exhibits a countercyclical variation. When this is done, 14 of 36 residual correlations are negative and the average correlation coefficient is .045. Even the positive relationships are generally quite weak; the correlation

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[30] The strength of any positive correlation in the cross-equation residuals will depend on the relative sizes of the variances of the omitted variables and the independent regression disturbance terms. Positive correlations could also arise for reasons unrelated to confounding (e.g. common sources of measurement error).

coefficient exceeds .707 (implying an R^2 above 0.5) in only 2 of 32 cases and surpasses 0.5 (R^2 greater than 0.25) in just 7. Furthermore, the error terms for some causes of death which vary procyclically are *negatively* correlated with those of most other sources of mortality. For instance, the influenza/pneumonia residuals are negatively associated with those for total fatalities and for 6 of the 9 specific causes of death.[31]

9. Conclusion

This study documents a strong inverse relationship between within-state fluctuations in unemployment and most types of mortality. The preferred specifications suggest that a one percentage point rise in joblessness is associated with a 0.5% decrease in the total death rate. The finding of a procyclical variation in fatalities is robust to changes in model specifications or time periods and is concentrated among young adults, whose employment is most affected by macroeconomic conditions. Deaths from 9 of 10 specific causes examined also rise as the economy improves, with the predicted increase in mortality, per one percentage point decline in the unemployment rate, ranging from a low of 0.2% for malignant neoplasms to a high of 2.4% for fatal motor vehicle accidents. The one exception is that suicides decline during expansions.

The association between personal incomes and health is quite tenuous. The predicted effects are often statistically insignificant and depend on the choice of specifications, outcomes, and time periods. A possible explanation for this fragility is that mortality and incomes may

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[31] The cross-equation residual correlations are expected to rise if important determinants of mortality are intentionally deleted from the models. To test whether this occurs, the fatality equations were reestimated with the macroeconomic and demographic variables dropped (so that only time and state effects are controlled for). When this was done, the average correlation coefficient tripled (rising from .049 to .144), with a still larger increase (from .045 to .154) when the total mortality and suicide residuals were excluded.

only be strongly related at relatively low levels of the latter (e.g. as in developing countries).[32] The estimated income effects also vary substantially by age and cause of death.

These findings raise questions about conventional economic and psychological models of the determinants of health. The former typically emphasize how prices, incomes, and stochastic shocks influence the stock of health capital; the latter generally focus on the effect of the macroeconomy on stress and risk-taking. In both cases, rising unemployment is usually predicted to have negative health effects, in contrast to the findings of this paper.

There are three possible interpretations for the econometric results. First, taken at face value, the estimates suggest that health improves during economic downturns and deteriorates during expansions. Second, mortality rates could vary with macroeconomic conditions while other measures of health do not (or not in the same direction). Third, the procyclical fluctuations of fatalities might reflect inadequacies of the data or econometric methods, rather than any true causal effects of the state of the economy.

Based on the evidence presented above, I believe that the first explanation is by far the most likely -- that health worsens as the economy improves. Although fatalities resulting from motor vehicle crashes, other accidents, or homicides may be unrelated or only weakly related to broader indicators of health, it is difficult to imagine that the same holds true for deaths due to heart disease, cancer, liver ailments, or influenza and pneumonia.

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[32] Fuchs (1979) claims that there is no systematic relationship between income and health in the United States, except possibly at the deepest levels of poverty. Evidence from the RAND Health Insurance Experiment indicates that medical expenses in the U.S. are virtually unrelated to income, although low (high) income households use relatively more inpatient (ambulatory) health care (Manning, et. al. 1987).

There are several reasons to doubt that the observed relationship between mortality rates and economic conditions is spurious. First, the fixed-effect models account for all time-invariant heterogeneity and the inclusion of controls for ethnic status, education, and particularly age reduce other sources of confounding. Second, the finding of a procyclical fluctuation in fatalities is robust to changes in the time period, the proxy for economic conditions, or the restriction of the sample to large states. Third, the strongest effects are observed for young adults, whose health is expected to be most strongly affected by the macroeconomy, and among those types of deaths disproportionately experienced by them. Fourth, the regression residuals for specific sources of mortality are seldom highly positively correlated with each other, in contrast to what would be expected if the cyclical fluctuations resulted from common omitted variables.

It is fairly obvious why some types of mortality rise during expansions. For instance, individuals consume more alcohol and drive greater distances when the economy improves, elevating the probability of involvement in motor vehicle crashes. It is less apparent, however, why deaths caused by disease also increase. As discussed, job-related stress and the time cost of medical care of healthy lifestyles may rise during periods of high employment. If stress is involved, however, its effect must be complicated since suicides, which are certainly correlated with mental health, decrease when unemployment rates fall. The effects of transitory and permanent changes in economic conditions may also be quite different. For instance, individuals may be more likely to postpone medical care during periods of temporarily high work intensity than if employment is permanently increased.

Are recessions good for your health? Surprisingly, the answer appears to be yes. Further research is needed to better understand why this is so.

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Table 1: Definitions and Summary Statistics of Variables Used in Analysis

Variable	Mean	Standard Deviation
Death Rate Per 100,000 Population Due To:		
All Causes	879.8	106.9
All Causes: 20-44 Year Olds	165.4	32.0
All Causes: 45-64 Year Olds	934.2	141.8
All Causes: ≥65 Year Olds	5240.0	417.7
Malignant neoplasms (140-208)	186.7	29.4
Major cardiovascular diseases (390-448)	423.9	74.5
Pneumonia and influenza (480-487)	27.1	5.9
Chronic liver disease and cirrhosis of the liver (571)	12.6	4.2
Motor vehicle accidents (E810-E825)	21.3	5.9
Other accidents and adverse effects (E800-E807, E826-E949)	22.2	5.4
Suicide (E950-E959)	12.3	2.8
Homicide and legal intervention (E960-E978)	9.5	4.2
Death Rate Per 1000 Live Births:		
Deaths Within First Year (infant mortality)	12.4	3.3
Deaths Within First 28 days (neonatal mortality)	8.5	2.7
Explanatory Variables:		
Civilian Unemployment Rate in %	6.9	2.1
% of Civilians Employed	59.6	4.0
Per Capita Personal Income (in thousands of 1987 dollars)	14.2	2.4
% of Population Under 5 Years Old	7.5	0.8
% of Population Aged 65 and Over	11.5	2.0
High School Dropouts (% of Persons 25 and over)	33.0	8.6
Some (1-3 years) College (% of Persons 25 and over)	25.0	6.9
College Graduate (>4 Years of College, % of Persons 25 and over)	16.6	4.0
% of Population Who are Black	11.7	8.0
% of Population Who are Hispanic	6.9	7.8

Notes: All variables are weighted by state populations. Employment statistics refer to civilians aged 16 and over. For the mortality rates, entries in parentheses refer to category listings from the Ninth Revision of the International Classification of Diseases.

Table 2: Fixed-Effect Estimates of the Determinants of Total Mortality Rates

	(1a)	(1b)	(2a)	(2b)
Unemployment Rate	-.0077 (.0007)	-.0083 (.0008)	-.0054 (.0005)	-.0056 (.0006)
Personal Income (in thousands)		-.0026 (.0012)		-.0012 (.0013)
Population <5 years old			-.0007 (.0020)	-.0006 (.0021)
Population aged 65 and over			.0485 (.0019)	.0485 (.0019)
High School Dropout			.0002 (.0006)	4.0E-5 (.0007)
Some College			-.0004 (.0005)	-.0004 (.0005)
College Graduate			-.0018 (.0011)	-.0011 (.0014)
Black			.0023 (.0013)	.0023 (.0013)
Hispanic			-.0019 (.0008)	-.0017 (.0008)

Note: The dependent variable is the natural logarithm of the total mortality rate per 100,000 population. The sample includes annual observations for the 50 states and District of Columbia covering the period 1972-1991. Missing data on unemployment rates for some states in the early years of the time period reduce the sample size to 930. Observations are weighted by the square root of the state population. All specifications also include vectors of state and year dummy variables. Standard errors are in parentheses.

Table 3: Alternative Specifications of Total Mortality Rate Equations

	(1a)	(1b)	(2a)	(2b)	(3a)	(3b)	(4a)	(4b)
Unemployment Rate	-.0054	-.0056	-.0002	-7.6E-5	-.0063	-.0056	-.0060	-.0063
	(.0005)	(.0006)	(.0010)	(.0010)	(.0004)	(.0005)	(.0004)	(.0005)
Personal Income		-.0012		.0026		.0031		-.0013
		(.0013)		(.0014)		(.0013)		(.0014)
Employment-to-Population Ratio	.0026	.0024	-.0016	-.0018	.0039	.0024	.0043	.0038
	(.0005)	(.0006)	(.0005)	(.0006)	(.0005)	(.0005)	(.0005)	(.0006)
Personal Income		.0010		.0034		.0079		.0025
		(.0014)		(.0014)		(.0013)		(.0016)
State Effects	Yes		No		Yes		Yes	
Time Effects	Yes		Yes		No		No	
Time Trend	No		No		No		Yes	

Note: See note on table 2. All specifications control for demographic characteristics.

Table 4: Fixed-Effect Estimates of Total Mortality Rate Equations for Alternative Samples

	Full Sample (with state-time trends)		1971-1982		1983-1991		10 Largest States	
	(1a)	**(1b)**	**(2a)**	**(2b)**	**(3a)**	**(3b)**	**(4a)**	**(4b)**
Unemployment Rate	-.0058	-0058	-.0039	-.0033	-.0061	-.0054	-.0056	-.0030
	(.0005)	(.0005)	(.0007)	(.0007)	(.0007)	(.0008)	(.0009)	(.0012)
Personal Income		-.0001		.0053		.0040		.0090
		(.0014)		(.0020)		(.0022)		(.0030)
Sample Size	930		471		459		200	

Note: See note on table 2. All specifications include vectors of state and year dummy variables and controls for demographic characteristics. The ten largest states refer to rankings in 1991 and include, in descending order of size, California, New York, Texas, Florida, Pennsylvania, Illinois, Ohio, Michigan, New Jersey, and North Carolina.

Table 5: Fixed-Effect Estimates of Total Mortality Equations with Lags or Changes in Macroeconomic Conditions

	(1a)	(1b)	(2a)	(2b)	(3a)	(3b)
Unemployment Rate	-.0050	-.0047	-.0059	-.0057	-.0045	-.0037
	(.0005)	(.0006)	(.0008)	(.0008)	(.0006)	(.0007)
Lagged Unemployment			.0012	.0016		
			(.0008)	(.0008)		
Δ in Unemployment					-.0143	-.0168
					(.0053)	(.0055)
Personal Income		.0014		.0047		.0025
		(.0016)		(.0020)		(.0017)
Lagged Personal Income				-.0028		
				(.0014)		
Δ in Personal Income						.0494
						(.0239)

Note: See note on table 2. All specifications include vectors of state and year dummy variables and controls for demographic characteristics. Samples are restricted to state-year observations with data available for lags and changes in unemployment rates and personal incomes (n=879). Lagged unemployment rates and personal incomes refer to the values of those variables in the previous year. The changes in unemployment rates are calculated as $(UN_t - UN_{t-1})/UN_{t-1}$, for UN_t the unemployment rate in the state at year t. Changes in personal incomes are calculated in a corresponding manner.

Table 6: Fixed-Effect Estimates of Total Mortality Equations For Different Age Groups

	(1a)	(1b)	(2a)	(2b)
		20-44 Year Olds		
Unemployment Rate	-.0225	-.0135	-.0183	-.0131
	(.0018)	(.0018)	(.0017)	(.0017)
		.0383		.0334
Personal Income		(.0029)		(.0041)
		45-64 Year Olds		
Unemployment Rate	.0007	-.0017	.0009	-.0001
	(.0009)	(.0010)	(.0010)	(.0010)
		-.0101		-.0062
Personal Income		(.0016)		(.0024)
		≥65 Year Olds		
Unemployment Rate	-.0019	-.0024	-.0030	-.0031
	(.0005)	(.0005)	(.0005)	(.0005)
		-.0022		-.0010
Personal Income		(.0009)		(.0012)

Note: See note on table 2. All specifications include vectors of state and year dummy variables. Specifications (2a) and (2b) also control for education levels and ethnic status.

Table 7: Age-Distribution of Mortality in 1990

Source of Mortality	# of Deaths	Age Group (in Years):			
		<15	15 to 44	45 to 64	≥65
All Causes	2,122,553	2.5%	8.4%	17.3%	71.7%
Cardiovascular Diseases	904,041	0.2	2.2	14.0	83.5
Malignant Neoplasms	499,427	0.3	4.7	26.7	68.3
Influenza/Pneumonia	78,307	1.2	3.1	7.2	88.6
Liver Disease/Cirrhosis	25,620	0.1	17.7	41.9	40.2
Motor Vehicle Accidents	46,467	6.7	62.2	15.6	15.4
Other Accidents	44,836	8.7	33.0	16.3	41.8
Suicide	30,572	0.8	55.4	23.0	20.7
Homicide	24,744	4.9	78.0	11.8	5.0

Source: *Vital Statistics of the United States, 1990* (table 1-30).

Table 8: Fixed-Effect Estimates of Cause-Specific Mortality Equations

	(1a)	(1b)	(2a)	(2b)	(1a)	(1b)	(2a)	(2b)
	Major Cardiovascular Diseases				**Malignant Neoplasms**			
Unemployment Rate	-.0061 (.0010)	-.0088 (.0011)	-.0043 (.0009)	-.0052 (.0009)	.0007 (.0010)	-.0025 (.0010)	-.0004 (.0007)	-.0019 (.0007)
Personal Income		-.0118 (.0017)		-.0062 (.0021)		-.0135 (.0017)		-.0102 (.0016)
	Influenza & Pneumonia				**Liver Disease & Cirrhosis**			
Unemployment Rate	-.0136 (.0024)	-.0141 (.0026)	-.0066 (.0024)	-.0046 (.0026)	.0033 (.0025)	-.0006 (.0027)	-.0069 (.0025)	-.0083 (.0027)
Personal Income		-.0020 (.0043)		.0134 (.0060)		-.0167 (.0044)		-.0091 (.0061)
	Motor Vehicle Accidents				**Other Accidents**			
Unemployment Rate	-.0253 (.0022)	-.0206 (.0024)	-.0289 (.0024)	-.0241 (.0025)	-.0161 (.0020)	-.0148 (.0021)	-.0164 (.0021)	-.0168 (.0022)
Personal Income		.0201 (.0039)		.0321 (.0058)		.0055 (.0035)		-.0026 (.0051)
	Suicide				**Homicide & Legal Intervention**			
Unemployment Rate	.0141 (.0022)	.0122 (.0023)	.0070 (.0022)	.0072 (.0024)	-.0224 (.0038)	-.0133 (.0040)	-.0167 (.0039)	-.0147 (.0041)
Personal Income		-.0079 (.0038)		.0016 (.0055)		.0386 (.0066)		.0133 (.0096)
	Infant Mortality				**Neonatal Mortality**			
Unemployment Rate	-.0059 (.0016)	-.0072 (.0017)	-.0043 (.0017)	-.0054 (.0019)	-.0086 (.0022)	-.0078 (.0024)	-.0058 (.0024)	-.0079 (.0025)
Personal Income		-.0057 (.0028)		-.0079 (.0043)		.0033 (.0039)		-.0134 (.0058)

Note: See note on table 2. All specifications include vectors of time and year dummy variables. Specifications (2a) and (2b) also control for demographic characteristics. All dependent variables are natural logs of mortality rates per 100,000 population, except infant and neonatal mortality which are per 1,000 live births. Sample sizes are 930 for death rates other than homicides, where missing values reduce the sample size to 922.

Table 9: Cross-Equation Correlations Of Mortality Regression Residuals

	Total	Heart	Cancer	Flu	Liver	Infant	Neonatal	Vehicle	Accident	Suicide	Homicide
Total	1.00										
Heart	0.82	1.00									
Cancer	0.69	0.45	1.00								
Flu	-0.15	0.11	-0.59	1.00							
Liver	0.38	0.10	0.84	-0.72	1.00						
Infant	0.13	-0.18	0.22	-0.41	0.32	1.00					
Neonatal	0.37	0.27	0.36	-0.52	0.35	0.60	1.00				
Vehicle	-0.03	-0.32	-0.02	-0.53	0.15	0.31	0.28	1.00			
Accident	-0.33	-0.39	-0.70	0.45	-0.63	0.02	-0.18	0.27	1.00		
Suicide	-0.38	-0.53	-0.40	0.25	-0.33	0.03	-0.52	0.24	0.50	1.00	
Homicide	0.39	0.05	0.63	-0.75	0.68	0.51	0.45	0.48	-0.31	-0.04	1.00

Note: Table shows the correlations between regression residuals for mortality equations which control for unemployment rates, personal incomes, demographic characteristics, and vectors of time and year dummy variables.

Fig. 1: *Trends in U.S. Mortality Rates*

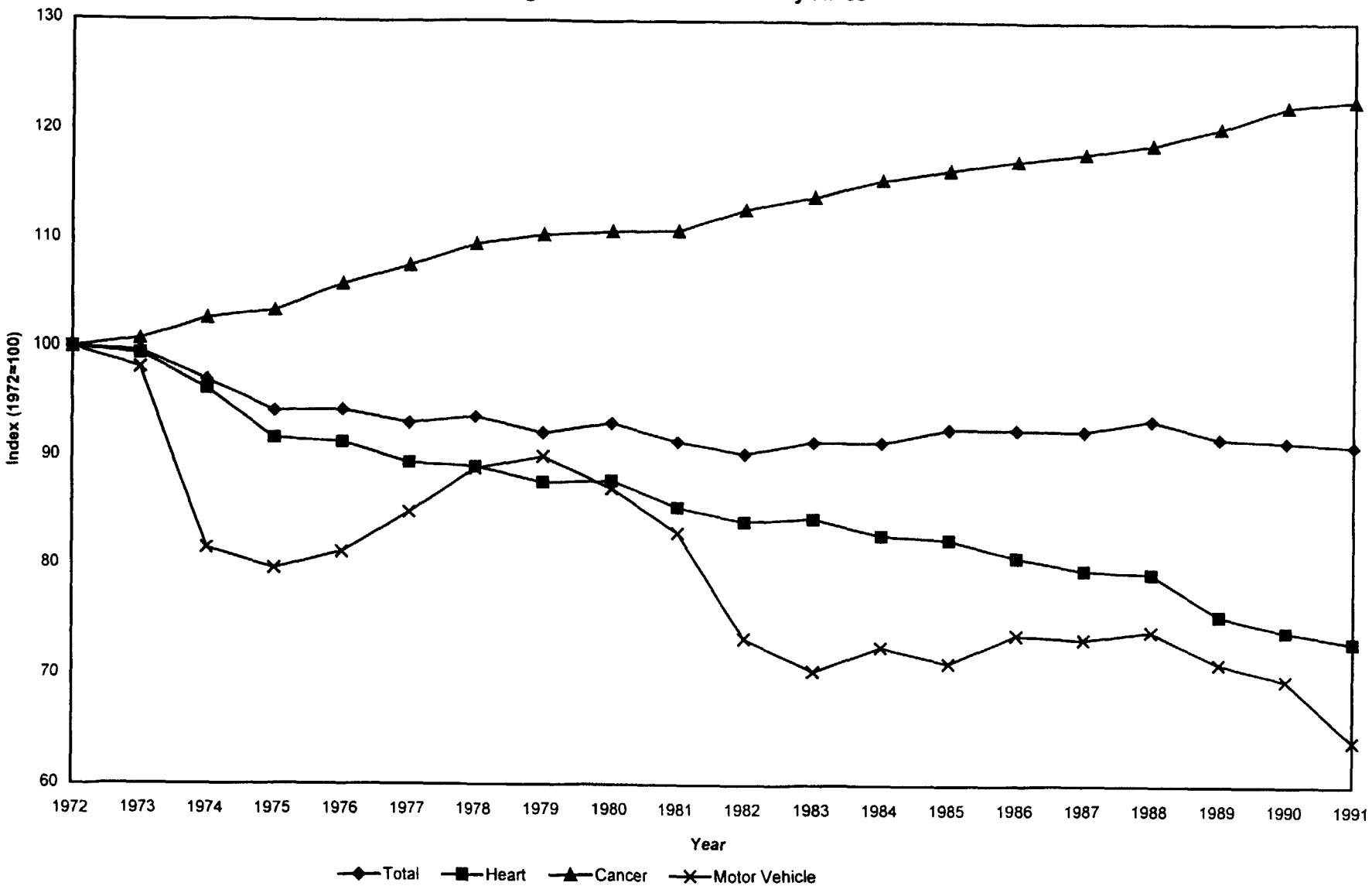

Fig. 2: Total Mortality and Unemployment Rates (Detrended and Normalized)

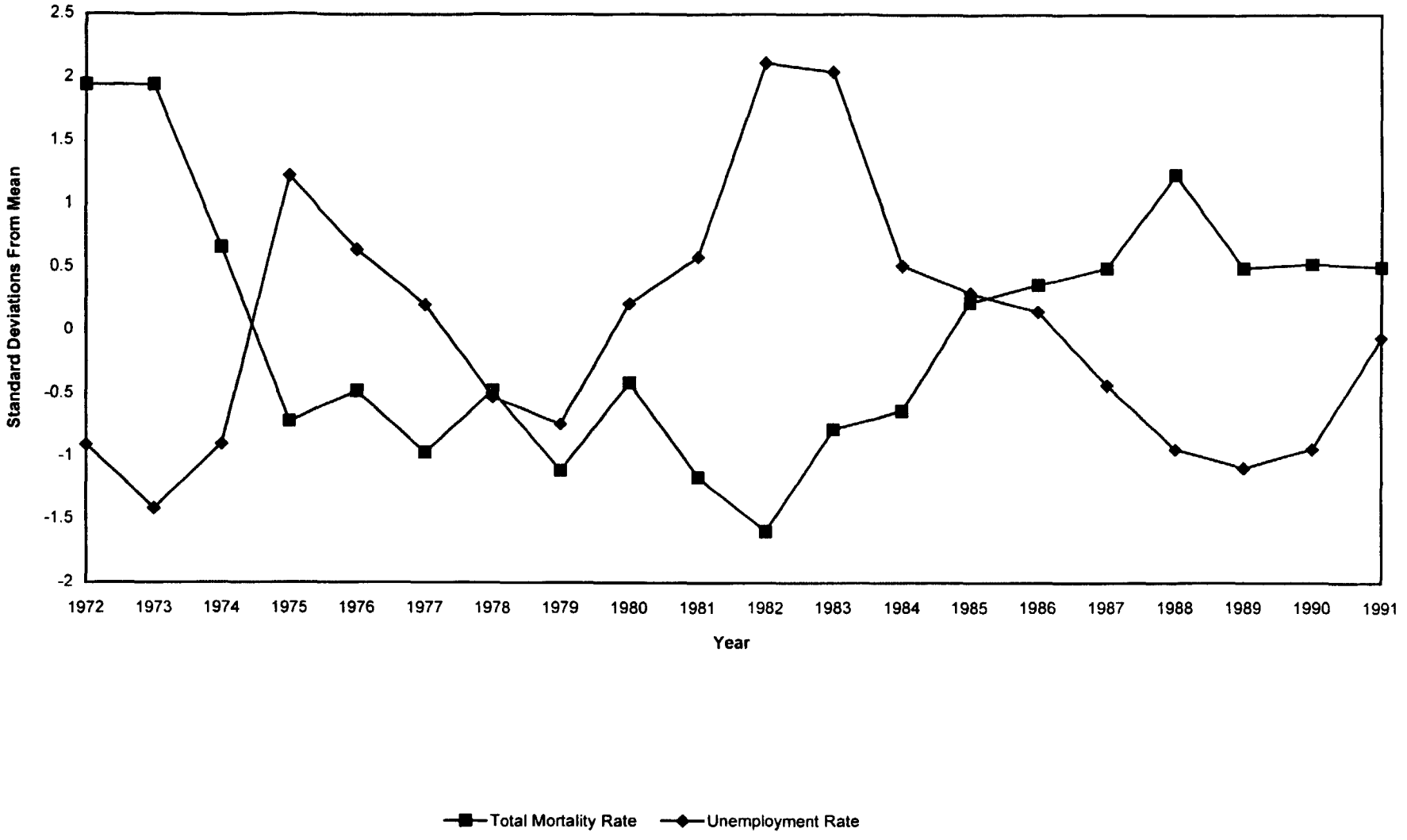

Fig. 3: Unemployment Rates in Selected States