Mortality and Macroeconomic Fluctuations in Contemporary Sweden

Mortalité et fluctuations macroéconomiques dans la Suède contemporaine

José A. Tapia Granados · Edward L. Ionides

Abstract Recent research has provided strong evidence that, in the United States in particular and in high- or middle-income economies in general, mortality tends to evolve better in recessions than in expansions. It has been suggested that Sweden may be an exception to this pattern. The present investigation shows, however, that in the period 1968–2003 mortality oscillated procyclically in Sweden, deviating from its trend upward during expansions and downward during recessions. This pattern is evidenced by the oscillations of life expectancy, total mortality, and age- and sex-specific mortality rates at the national level, and also by regional mortality rates for the major demographic groups during recent decades. Results are robust for different economic indicators, methods of detrending, and models. In lag regression models macroeconomic effects on annual mortality tend to appear lagged 1 year. As in other countries, traffic mortality rises in expansions and declines in recessions, and the same is found for total cardiovascular mortality. However, macroeconomic effects on ischemic heart disease mortality appear at lag two and are hard to interpret. Reasons for the procyclical oscillations of mortality, for inconsistent results found in previous studies, as well as for the differences observed between Sweden and the United States are discussed.

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Résumé   Des recherches récentes ont apporté de solides preuves que dans les économies présentant des revenus élevés ou moyens, et aux Etats-Unis en particulier, la mortalité tend à diminuer pendant les périodes de récession plutôt que pendant les périodes d'expansion. Il a été suggéré que la Suède pouvait être une exception à cette tendance générale. Cette recherche montre cependant qu'au cours de la période 1968–2003 la mortalité a oscillé de manière procyclique en Suède, déviant de ses tendances pour augmenter pendant les périodes d'expansion économique et pour diminuer pendant les périodes de récession. Ce schéma est mis en évidence par les variations de l'espérance de vie, de la mortalité totale, des taux de mortalité par âge et par sexe au niveau national, mais aussi des taux régionaux de mortalité des grands groupes démographiques au cours des récentes décennies. Les résultats sont robustes pour les différents indicateurs socio-économiques, les méthodes de décomposition des séries chronologiques pour éliminer les tendances et les modèles utilisés. Dans les modèles de régression avec retard, des effets macroéconomiques sur la mortalité annuelle tendent à apparaître avec un retard d'une année. Comme dans les autres pays, la mortalité par accidents de la route augmente pendant les périodes d'expansion et diminue pendant les périodes de récession. Il en est de même pour la mortalité cardio-vasculaire totale. Cependant les effets macroéconomiques sur la mortalité par cardiopathie ischémique apparaissent avec un retard de deux années et sont difficiles à interpréter. Les raisons des oscillations procycliques de mortalité, des résultats instables observés dans les études antérieures, ainsi que des différences observées entre la Suède et les Etats-Unis sont discutées.

Mots-clés   Mortalité · Espérance de vie · Suède · Cycles économiques · Fluctuations macro-économiques · Santé de la population

1 Introduction

In a number of countries and periods it has been found that over and above long-term declining trends, mortality rates tend to rise in expansions and decline in recessions. The main finding of the present investigation is that the same pattern is observed in Sweden during the period 1968–2003. Since mortality oscillates procyclically, life expectancy at birth (e_0) oscillates countercyclically and the annual gain in e_0 is greater in years of economic downturn than in years of economic expansion. This procyclical oscillation of mortality is shown in the present study using various statistical models, for a range of different mortality rates and business-cycle indicators. These results add to an emerging consensus that economic expansions rather than recessions have harmful effects for health.

   The next two sections review previous study on mortality and the business cycle, and potential pathways linking the economy with mortality. Sections 4 and 5 show the data and methods we have used for the investigation, and the results of our analyses. The last two sections include a discussion of the results of other

investigations, and a general discussion of our findings in the context of previous investigations.

2 Previous Research on Mortality and the Business Cycle

The relation between mortality and the business cycle was already investigated in the 1920s by Ogburn and Thomas (1922), who were surprised by finding a procyclical fluctuation of mortality in the United States and Britain during the business cycles preceding World War I. These early studies were largely forgotten for most of the twentieth century, probably because the idea that mortality evolves better during recessions than during expansions was too counterintuitive to be easily accepted.

The notion that economic hardship must be bad for health, and therefore associated with higher mortality, is deeply ingrained in social science. Many investigations in the fields of public health, demography, and economics have shown that low income is systematically associated with higher death rates (Isaacs and Schroeder 2005). Increasing mortality resulting from a worsening standard of living is the basis of Malthus's "positive check" to population growth, which is a frequent working hypothesis in demography and economic history. Historical studies of preindustrial societies have indeed shown that mortality upswings are associated, for instance, with bad harvests, increases in grain prices, or cold winters (Lee 1981; Schofield 1985; Thomas 1941). However, this link between mortality and food price inflation or bad harvests is considerably weakened with advances in economic development and industrialization (Galloway 1988). In Sweden, for example, the strong mortality responses to bad harvests observed in the eighteenth century and first half of the nineteenth century disappeared during the last decades of the nineteenth century (Thomas 1941; Tapia Granados and Ionides 2008).

In the 1970s and 1980s, Brenner repeatedly reported harmful effects of recessions on mortality, other health indicators, and social conditions (Brenner 1971, 1982, 1987). Brenner utilized relatively unconventional econometric techniques such as Fourier analysis, distributed lag regressions, and ARIMA models. These studies, mostly published in health journals, raised protracted controversy. While the results of a few investigations apparently supported Brenner's argument to some extent (Junankar 1991; McAvinchey 1984), other researchers criticized Brenner's studies for deficient presentation of data and methods, use of lags arbitrarily chosen, lack of statistical power with the number of observations being often close to that of covariates, improper methods of detrending, and inclusion of multiple covariates implying strong collinearity (Eyer 1977a; Kasl 1979; Gravelle et al. 1981; Winter 1983; Søgaard 1992; Wagstaff 1985). Meanwhile, with straightforward statistical methods Eyer (1977b), Higgs (1979), and Graham et al. (1992) again found a procyclical fluctuation of mortality in the United States.

In recent years a number of publications have reexamined whether macroeconomic fluctuations are related to short-term oscillations in mortality. In a groundbreaking study, Ruhm (2000) applied panel regressions to data from the 50 states of the United States during the years 1972–1991, showing that beyond long-term

trends, periods of expanding economic activity—gauged by low state unemployment levels—were associated with increased mortality, that is to say, that mortality oscillates procyclically. Other work using panel regressions has shown that the 1981–1982 recession induced substantial reductions of atmospheric pollution associated with significant drops in infant mortality (Chay and Greenstone 2003); that babies born during recessions in the United States have a reduced incidence of low birth weight and lower infant mortality (Dehejia and Lleras-Muney 2004); and that death rates oscillate procyclically in Japan (Tapia Granados 2008), the 16 German states (Neumayer 2004), the 50 Spanish provinces (Tapia Granados 2005a), and the 23 OECD countries (Johansson 2004; Gerdtham and Ruhm 2006). The procyclical oscillations of mortality in the United States have also been confirmed using time-series analyses (Tapia Granados 2005b) and individual-level data from the National Longitudinal Mortality Study (Edwards 2008). The evolution of mortality rates and life expectancy in the United States during the 1920s and 1930s shows that mortality-based health indicators evolved better during the Great Depression of 1929–1933 than during the economic expansions of the "roaring" 1920s and mid-1930s (Tapia Granados and Diez Roux 2009). In medium-income market economies mortality responses to macroeconomic downturns have been found to be erratic (Tapinos et al. 1997) or even clearly procyclical (Abdala et al. 2000; Khang et al. 2005; González and Quast 2010).

Sweden has been the focus of four recent studies by Gerdtham and Johannesson (2005), Tapia Granados and Ionides (2008), and Svensson (2007, 2010), which provide conflicting evidence on the relation between macroeconomic fluctuations and mortality in that country. The existence and nature of this relation in Sweden seems important because (a) Sweden is a world leader in health indicators and levels of income per capita; (b) the Swedish welfare state is one of the most developed in the world, with universal health care and generous income support for unemployed people provided by the State; and (c) the Swedish economy went through major fluctuations in the last decades of the twentieth century (see for instance the wide fluctuations of unemployment, Fig. 1).

The study by Gerdtham and Johannesson (2005) used a sample of 47,484 adults interviewed in Sweden in 1980–1996 and followed through the end of 1996, for a total of 6,571 deaths. Using a probit model Gerdtham and Johannesson modeled the probability of death as a function of age, the state of the economy (proxied by a business-cycle indicator), and a linear trend. They found mortality increasing during economic downturns and decreasing in upturns—i.e., a countercyclical fluctuation of mortality—though only for male mortality, and only for four business-cycle indicators (notification rate, capacity utilization, confidence indicator, and GDP growth) out of six that they considered (the aforementioned, plus unemployment and GDP deviation from trend).

Tapia Granados and Ionides (2008) investigated the relation between economic growth and health progress in Sweden during the nineteenth and twentieth centuries. They found that annual economic growth was positively associated with mortality decline throughout the nineteenth century, though the relation became weaker as time passed and was reversed in the second half of the twentieth century, during which higher economic growth was associated with lower drops in mortality. While
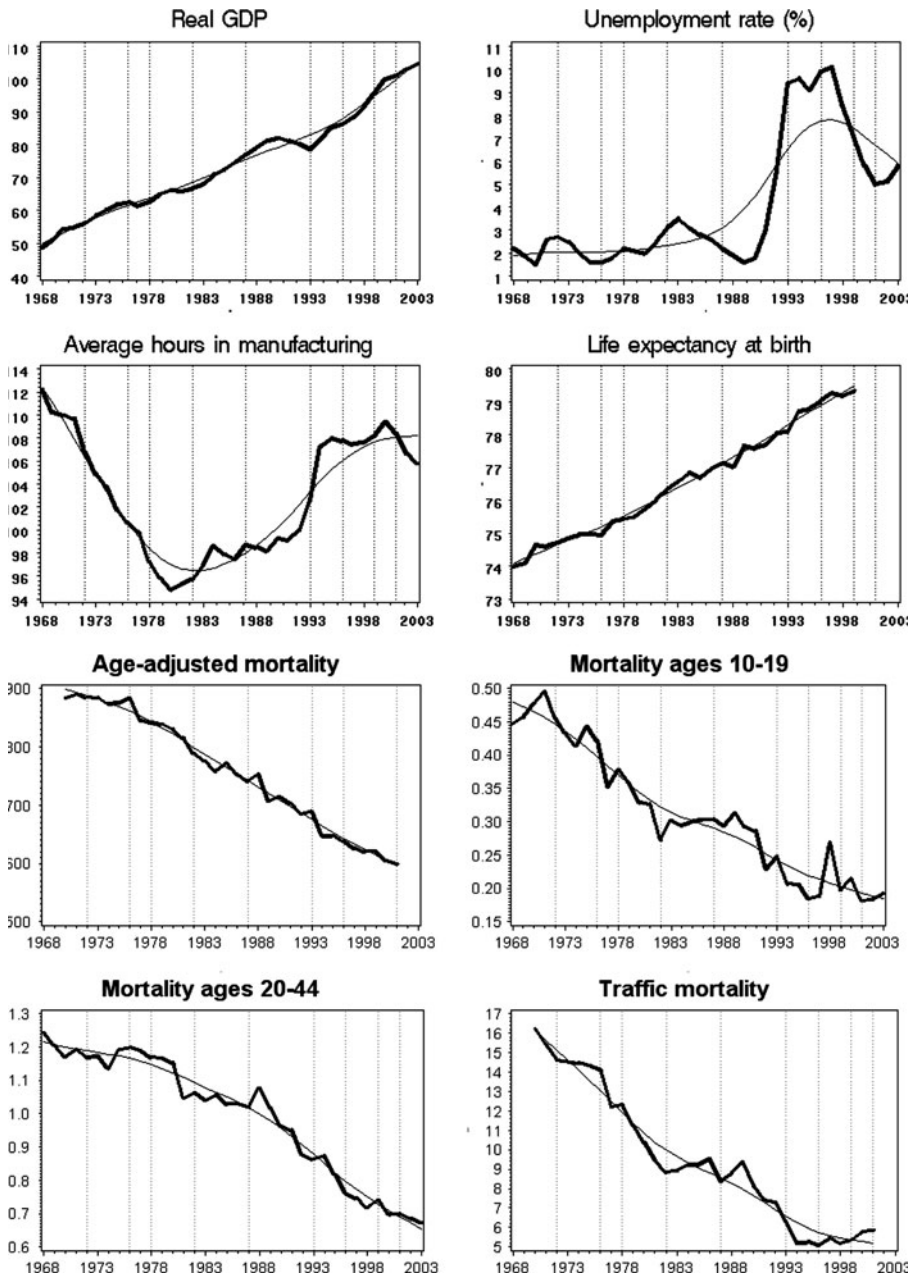
Fig. 1 Selected variables used in the study. The thin curves are Hodrick–Prescott trends computed with a smoothing parameter $\gamma = 100$. *Notes*: Real GDP and average hours indexed to 100 for 2000 and 1992, respectively. Mortality is per 100,000; traffic mortality is an age-standardized rate. Vertical dotted lines are business-cycle troughs (OECD chronology)

the economy-related effect on health occurs mostly at lag zero in the nineteenth century, the effect is lagged up to 2 years in the twentieth century. These results implying a procyclical fluctuation of mortality rates after 1950 were found to be robust across a variety of mortality rates (sex-, age-, and age-and-sex-specific), statistical procedures (including linear regression, spectral analysis, and lag regression models), and economic indicators (GDP per capita growth, unemployment, and some measures of inflation). This study did not investigate effects for periods shorter than a half century or for cause-specific mortality, but its results indicate that macroeconomic changes (indexed by annual GDP growth or the annual change in unemployment) have a lagged effect on the annual change in mortality, mostly at lag one.

In two different studies Svensson (2007, 2010) has employed panel regressions to analyze annual rates of mortality and unemployment in the 21 Swedish regions during 1976–2005. He could not detect any relation between mortality (neither total nor sex-specific) and unemployment that was robust to different specifications including year-fixed effects, region-specific linear trends or other methods to avoid potential bias. Therefore, he was unable to replicate the countercyclical oscillation of male mortality reported by Gerdtham and Johannesson (though Svensson downplays this inconsistency between his results and those of Gerdtham and Johannesson). The only robust procyclical oscillation found by Svensson is for mortality due to work-related injuries, which increase in expansions. Other cause-specific mortality rates were found to be procyclical or countercyclical depending on the specification. Svensson found ischemic heart disease mortality to be consistently countercyclical, though only among those aged 20–49 and at the 0.1 level of statistical significance.

3 Pathways Linking the Economy and Mortality

Potential pathways leading to a procyclical fluctuation of mortality have been extensively discussed elsewhere (Ruhm 2006; Tapia Granados 2005a; Eyer 1984). They will only be briefly summarized here.

Mortality caused by diseases of the circulatory system, that is, cardiovascular disease (CVD) mortality, is usually the main component of total mortality in most countries. It has been hypothesized that procyclical oscillations of CVD mortality may result from a variety of CVD risk factors that increase during expansions. These would include stress in the working environment—because of overtime and increased rhythm of work (Sokejima and Kagamimori 1998)—, reduced sleep time (Liu et al. 2002), atmospheric pollution, smoking, and consumption of saturated fat and alcohol (Ruhm 2006). Many of these factors might increase the risk of death of persons who had a previously developed chronic disease, such as atherosclerosis cancer, diabetes, a respiratory illness, or a chronic infection. The increase in traffic deaths during economic booms and the decrease in recessions is a robust finding across countries (Eyer 1977b; Ruhm 2000; Tapia Granados 2005a, 2008; Khang et al. 2005). The strong procyclical fluctuation of traffic-related injuries and deaths is intuitively explained by the fact that commuting, commercial, and recreational

traffic rapidly expand in economic upswings, and the effect is probably aggravated by the procyclical change in alcohol consumption and by sleep deprivation in expansionary periods in which overwork is frequent (Liu et al. 2002).

Increased infant mortality in expansions has been related to economic activity through atmospheric pollution (Chay and Greenstone 2003) and healthier behaviors of pregnant women during recessions (Dehejia and Lleras-Muney 2004). In addition, in high-income economies injury deaths are a quite large proportion of the small annual number of infant and children deaths. Thus, economic expansions could be associated with an above-trend level of infant and child mortality through car crashes as well as domestic or school injuries related to overworked parents or caretakers.

4 Data and Methods

In the present investigation crude, and sex- and age-specific mortality rates were computed from deaths and population in 5-year age strata taken from Statistics Sweden (2005). Data on annual unemployment rates, real GDP, industrial production, and the confidence indicator for the manufacturing industry were obtained from the same source. Infant mortality and age-standardized mortality rates were taken from the WHO-HFA (2005) database.[1] Four indices of Swedish manufacturing activity (average hours, aggregate hours, manufacturing output and manufacturing employment) were obtained from the U.S. Bureau of Labor Statistics (2005). Data on life expectancy at birth are from the Human Mortality Database (2005).

Our main analysis employs data from Swedish annual statistics (Fig. 1), from 1968 to the early 2000s (usually until 2003, with *n* equal to 35 or 36 in most series). This time frame was chosen for two reasons: first, because it is possible that the relation between macroeconomic changes and health progress changed over time, and the focus of this investigation is contemporary Sweden; second, because a number of cause-specific and age-adjusted mortality rates for Sweden are available from the WHO Health-For-All database beginning in 1968 or 1970. The reference chronology of the Organisation for Economic Co-operation and Development (OECD 2006) indicates that there were 10 business cycle troughs in Sweden (Fig. 1) between 1968 and 2003.

Population health is indexed by life expectancy at birth (e_0, "life expectancy" for brevity) and a variety of age-, sex-, and cause-specific mortality rates. Since life expectancy is the best mortality-based summary index of population health, unaffected by the age structure of the population and allowing intertemporal and cross-sectional comparisons, we based many of our analysis in Δe_0, the annual gain in life expectancy, as a major indicator of health progress.

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[1] In the HFA database age-standardized rates are computed with the direct method, applying age-specific rates for 5-year age-strata (0–4, 5–9, etc.) to a standard European population. For instance, the standardized rate of cancer mortality at ages 0–64 represents what the age-specific cancer mortality rate at ages 0–64 would have been if the Swedish population aged 0–64 had the same age distribution as the standard European population.

Analyses in this article are either (*a*) simple correlation models between health and economic indicators, or (*b*) lag regressions in which a health indicator is regressed on contemporaneous and lagged values of an economic indicator. Strong collinearity between business cycle indicators precludes using combinations of these indicators in the same multivariate regression model. Since both the indicators of population health and the economic indicators used in the study have obvious trends (Fig. 1), *all* variables were detrended before analysis to produce trend-stationary series. Several procedures to detrend variables were used; only results obtained either with "HP detrended" series (i.e., series obtained by subtracting the trend computed with the Hodrick–Prescott filter[2] from the actual value of the variable) or with variables transformed into first differences ($\Delta x_t = x_t - x_{t-1}$) or rate of change ($\Delta x_t / x_{t-1}$) are presented. Other detrending procedures[3] were employed in sensitivity analyses and yielded similar results.

Macroeconomic fluctuations are not easy to identify and delimit, and not all the different business cycle indicators gauge them in the same way. In the present study, eight economic indicators were used: GDP, the unemployment rate, the index of industrial production, the confidence indicator and four indices of activity in manufacturing: average working hours, aggregate hours, output and employment. All of them show significantly correlated fluctuations except the confidence indicator which fluctuates without a definite relation to the others. For the sake of brevity results for only three business cycle indicators—real GDP, the unemployment rate, and the index of average working hours in manufacturing—will be presented here. At lag zero, detrended unemployment and GDP have a strong negative correlation in this Swedish sample (-0.75 when both series are in rate of change, -0.85 when both are detrended with the Hodrick–Prescott filter, for both correlations $P < 0.001$). The index of average hours worked in manufacturing correlates positively and significantly with unemployment (0.40, $P = 0.01$, both series HP-detrended) at lag zero. Therefore, both unemployment and average hours are countercyclical, while GDP is procyclical.[4]

Cross-correlations and distributed lag regression models were used to ascertain the concomitant variation between "health" and "the economy" or the coincident or lagged effect of the latter on the former. Cross-correlations between detrended series have been used to analyze the historical properties of business cycle indicators (Backus and Kehoe 1992), biological and medical data (Diggle 1990), and time-series experiments (Glass et al. 1975). Distributed lag regressions are a common tool in econometrics. For the sake of consistency, the two variables correlated or used as outcome and explanatory variables in a regression were always

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[2] We used a smoothing parameter $\gamma = 100$ (Fig. 1), as advised for annual data (Backus and Kehoe 1992).

[3] We used the HP filter applied with $\gamma = 10$, and the band-pass filter BP(2,8), recommended by Baxter and King (1999).

[4] Business cycle data for the United States and other industrialized countries show average working hours in manufacturing as a procyclical indicator; in Sweden, however, its strong positive correlation with unemployment shows that it is clearly a countercyclical one. We comment on the potential causes of this difference in the discussion.

detrended by the same method.[5] In lag regressions we used the Akaike information criterion (AIC) as an objective approach to choose among the multiple models that we investigated. Although AIC has its strengths and flaws relative to other model selection criteria, such as BIC and adjusted R^2 (Claeskens and Hjort 2008), to minimize AIC is a common and generally accepted procedure to choose "the best" model among models with different lags.

In supplementary analyses, utilizing panel regression models we also analyzed Swedish mortality rates and unemployment rates for the 21 Swedish regions in the years 1976–2005. These data were kindly shared with us by Mikael Svensson, and correspond to the data used in his two articles (Svensson 2007, 2010). We obtained the population data for the regional analysis from Statistics Sweden.

5 Results

5.1 Correlation Models

For the whole population and for large age groups, and with series HP-detrended, mortality has positive correlations with GDP and negative correlations with unemployment and average hours (Table 1). Life expectancy at birth, e_0, follows the same pattern with reversed signs, as is to be expected if mortality is procyclical.[6] Out of 54 correlations of detrended economic indicators with detrended health indicators (Table 1), only four correlations have a sign that is inconsistent with a procyclical oscillation of mortality.

When age- and sex-specific mortality rates are correlated with the economic indicators, with variables either HP-detrended (Table 2, bottom panel) or in rate of change (Table 2, top panel), the correlations of the HP-detrended variables with the economic indicators are much stronger. However, the direction of the correlations is consistently similar in analyses involving rate of change or HP-detrended series, suggesting that the HP-filtering is not introducing spurious results in terms of the sign of correlations of economic indicators with mortality rates. Leaving aside the correlations that are statistically indistinguishable from zero, almost without exception the correlations of age- and sex-specific mortality rates with unemployment and average hours are negative, while the correlations of mortality rates with GDP are positive (Table 2). As observed for larger demographic groups (Table 1), the pattern of signs in the correlations of age- and sex-specific mortality rates (Table 2) indicate a procyclical oscillation of mortality for most age groups. The high correlations between detrended business cycle indicators and detrended

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[5] In fact, there is an exception to this rule, because in some models first differences in e_0 or mortality are regressed on the rate of growth of GDP. GDP growth is a major dynamic index of business conditions and, moreover, because of the exponential growth of real GDP, first differences tend to present exploding heteroskedasticity when the series exceeds a few observations.

[6] The other three manufacturing indicators (aggregate hours, output, and employment) and the index of industrial production correlate similarly with mortality and e_0. The confidence indicator has erratic correlations with both the other business cycle indicators and with the health indicators.

Table 1 Cross-correlations between three economic indicators, life expectancy, and selected mortality rates for Sweden, 1968–2003

Health indicator	Unemployment	GDP	Average hours in manufacturing
Life expectancy at birth	0.35*	−0.15	0.51**
LEB, males	0.37*	−0.18	0.48**
LEB, females	0.30[†]	−0.10	0.49**
Age-standardized mortality rate	−0.27	0.21	−0.49*
ASMR, males	−0.19	0.12	−0.37*
ASMR, females	−0.31[†]	0.26	−0.38*
Crude mortality rate	−0.24	0.11	−0.39*
CMR, males	−0.17	0.08	−0.37*
CMR, females	−0.27	0.14	−0.38*
Age-specific mortality rates			
Infant mortality	−0.41*	0.24	−0.16
0–4	−0.48**	0.36*	−0.34*
5–9	−0.01	−0.08	−0.34*
10–19	−0.42**	0.52**	−0.12
20–44	−0.42**	0.36*	−0.21
25–64	−0.30[†]	0.17	−0.34*
45–64	−0.25	0.11	−0.33*
55–64	−0.26	0.07	−0.42**
65–84	−0.03	−0.13	−0.37*
85 and over	0.16	−0.19	−0.10

Notes: Correlations with mortality rates are based on $n = 36$ or close to 36 in most cases; in LEB correlations, $n = 32$. All variables detrended with the Hodrick–Prescott filter, $\gamma = 100$

[†] $P < 0.1$; * $P < 0.05$; ** $P < 0.01$

mortality for ages 10–19 and 20–44 are apparent in the fluctuations of these death rates following the swings of the economy (Fig. 2).

During the period of the study, the unemployment rate oscillated very moderately in Sweden before 1990, never exceeding 3%, but in the early 1990s unemployment rose to around 10%, remaining there for several years (Fig. 1). Correlations between mortality rates and economic indicators computed in split samples corresponding to the years in which unemployment was low (1968–1989) or high (1989–2003) reveal that the results are quite robust (Table 3). In split samples the correlations of life expectancy and mortality rates with the countercyclical unemployment and average hours and with the procyclical GDP all show an increase in death rates during expansions. However, with smaller samples fewer results are significant and in some cases even coefficient signs are unstable.

The zero-lag correlations of detrended series of cause-specific mortality with detrended economic indicators (Table 4) indicate a strong procyclical oscillation of traffic mortality, which shows strong positive correlations with GDP and strong negative correlations with unemployment and average hours. CVD mortality does not reveal any clear relation with business fluctuations at lag zero. One of its

Table 2 Cross-correlation between economic indicators and age- and sex-specific mortality rates for large age strata

Age-specific mortality	Unemployment rate	GDP	Average hours in manufacturing
A. Series in rate of change			
Male mortality			
10–19	−0.09	0.15	−0.04
20–44	−0.08	0.05	−0.04
45–64	−0.06	0.11	−0.56**
65–84	0.27	−0.23	−0.30†
85 and over	0.09	−0.13	−0.08
Female mortality			
10–19	−0.16	0.15	−0.10
20–44	−0.19	0.20	−0.14
45–64	−0.19	0.24	0.00
65–84	0.10	−0.23	−0.16
85 and over	0.18	−0.20	−0.19
B. HP-detrended series			
Male mortality			
10–19	−0.41*	0.50*	−0.04
20–44	−0.42*	0.31†	−0.17
45–64	−0.14	0.01	−0.38*
65–84	0.06	−0.16	−0.28†
85 and over	0.21	−0.17	0.07
Female mortality			
10–19	−0.28†	0.34*	−0.20
20–44	−0.30†	0.31†	−0.19
45–64	−0.31†	0.20	−0.15
65–84	−0.11	−0.08	−0.40*
85 and over	0.05	−0.14	−0.21

In each of the two panels, the series are either in rate of change or HP-detrended

† $P < 0.1$; * $P < 0.05$; ** $P < 0.01$

components, ischemic heart disease mortality or "heart attacks"—causing about one half of total CVD deaths—correlates negatively with GDP (−0.29) and positively (0.28) with unemployment, at marginal or not significant levels when considering all ages. However, at ages over 64 the correlations have the same sign, but they are much stronger, all of which suggests that ischemic heart disease at advanced ages fluctuates countercyclically in Sweden *when considering lag zero effects only*. Cerebrovascular disease (stroke) mortality comprises about one fifth of all CVD deaths and does not reveal any definite relation with business cycle indicators at lag zero.[7]

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[7] Table 4 and other tables omit the results for cancer, respiratory disease, infectious disease, flu, suicide and homicide, causes for which we could not find a conclusive relation with macro-economic fluctuations.
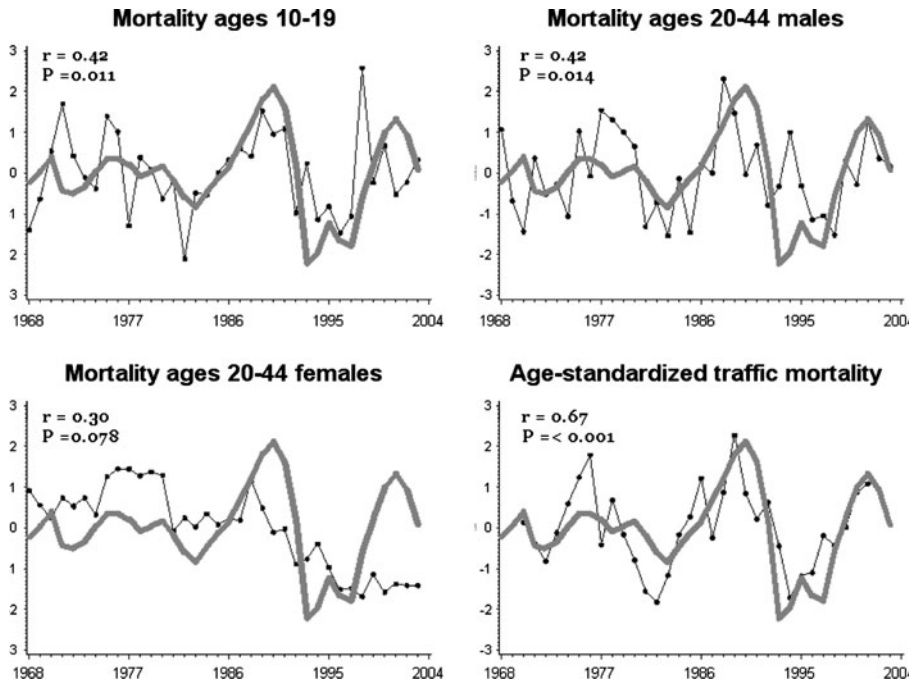
Fig. 2 Unemployment rate, inverted (thick *gray line* in the four panels) and four age- or cause-specific mortality rates. All series are annual data detrended with the Hodrick–Prescott filter ($\gamma = 100$) and normalized

5.2 Lag Regression Models

We explored the joint effect on health of coincidental and lagged changes in the economy with models of the type $H_t = \alpha + \sum_{i=0}^{p} \beta_i x_{t-i}$ including p lags, in which H_t is a health indicator at year t and x_{t-i} is an economic indicator at year $t - i$. In these models, health is indexed by e_0 or an age-specific mortality rate, and the explanatory variable is either unemployment, GDP, or average hours in manufacturing. Variables are detrended by conversion either in first differences or in rate of change. In general, in models using different methods of detrending, the estimated effects were consistent in sign. For a variety of health indicators we explored models in which the economy is indexed by an indicator (GDP, unemployment, or average hours) including several lags (Table 5). For life expectancy, crude mortality, or age-specific mortality for large age-groups (Table 5), AIC values indicate that models including a coincidental effect at lag zero and a lagged effect at lag one are to be preferred when the economic indicator used as regressor is GDP growth or the change in unemployment. However, when the regressor is average hours, the models including lag zero only are those that minimize AIC. All these distributed lag models in which either GDP or unemployment is the economic covariate (Table 5, panels A and B) show a significant effect on health at lag one, but when the economic indicator is average hours the effect is at lag zero. GDP

Table 3 Split sample cross-correlations of economic indicators with selected mortality rates and life expectancy

Health indicator	Unemployment	GDP	Average hours in manufacturing
A. Sample 1968–1989 ($n = 22$)			
Life expectancy	0.22	0.04	0.38[†]
Males	0.27	0.06	0.37[†]
Females	0.16	0.01	0.35
Crude mortality rate	−0.16	0.06	−0.29
Males	−0.07	0.01	−0.23
Females	−0.22	0.09	−0.34[†]
Infant mortality	0.30	−0.09	0.46*
Mortality 10–19	−0.47*	0.69***	0.16
Mortality 20–44	−0.61**	0.41*	−0.27
Mortality 45–64	0.11	−0.24	−0.32
Mortality 65–84	0.07	−0.18	−0.32
Mortality 85 and over	0.09	−0.20	−0.26
B. Sample 1989–2003 ($n = 15$)			
Life expectancy[a]	0.53	−0.29	0.73*
Males[a]	0.61[†]	−0.39	0.69*
Females[a]	0.44	−0.17	0.74*
Crude mortality rate	−0.20	0.01	−0.44[†]
Males	−0.10	−0.08	−0.45[†]
Females	−0.25	0.07	−0.39
Infant mortality	−0.78***	0.51[†]	−0.68**
Mortality 10–19	−0.50[†]	0.46[†]	−0.39
Mortality 20–44	−0.50[†]	0.47[†]	−0.24
Mortality 45–64	−0.30	0.18	−0.22
Mortality 65–84	0.06	−0.28	−0.34
Mortality 85 and over	0.36	−0.28	0.12
C. Sample 1981–1992 ($n = 11$)			
Life expectancy	0.53	0.40	−0.37[†]
Males	0.59[†]	0.49	−0.33[†]
Females	0.45	0.30	−0.40
Crude mortality rate	−0.28	0.25	−0.10
Males	−0.18	0.17	−0.01
Females	−0.36	0.31	−0.17[†]
Infant mortality	−0.41	0.15	0.46*
Mortality 10–19	−0.82**	0.85**	−0.31
Mortality 20–44	−0.72**	0.82**	−0.11
Mortality 45–64	0.39	−0.41	0.15
Mortality 65–84	−0.05	−0.08	−0.15
Mortality 85 and over	0.21	−0.27	−0.18

Notes: All variables detrended with the Hodrick–Prescott filter, $\gamma = 100$

[†] $P < 0.1$; * $P < 0.05$; ** $P < 0.01$; *** $P < 0.001$

[a] $n = 9$

Table 4 Correlations between economic indicators and cause-specific mortality rates for the years 1968–2002 in Sweden

Mortality rate	Unemployment	GDP	Average hours in manufacturing
Cardiovascular disease (I00–I99)[a]	0.14	−0.17	−0.14
Males	0.19	−0.13	−0.10
Females	0.06	−0.11	−0.16
Ages 55–64 males	−0.02	−0.15	−0.18
Ages 55–64 females	−0.16	0.06	0.05
Ages 65–74 males	0.21	−0.22	−0.16
Ages 65–74 females	0.13	−0.15	−0.17
Ages 75–84	0.01	−0.09	−0.15
Ischemic heart disease (I20–I25)[a]	0.28†	−0.29	−0.10
Males	0.27	−0.28	−0.03
Females	0.27	−0.29	−0.19
Ages 25–64 males	0.05	−0.10	−0.02
Ages 25–64 females	−0.37*	0.26	−0.18
Ages 35–44 males	0.15	−0.23	0.09
Ages 35–44 females	−0.34*	0.27	−0.19
Ages 45–54 males	0.11	−0.05	−0.02
Ages 45–54 females	−0.26	0.19	−0.24
Ages 55–64 males	−0.01	−0.07	−0.04
Ages 55–64 females	−0.24	0.16	−0.05
Ages 65 and older	0.33†	−0.32†	−0.10
Ages 65 and older males	0.32†	−0.29	−0.04
Ages 65 and older females	0.33†	−0.32*	−0.17
Ages 85 and older	0.39*	−0.36*	0.00
Cerebrovascular disease (stroke, I60–I69)[a]	−0.16	0.13	−0.18
Ages 25–64	−0.08	0.03	−0.17
Ages 25–64 males	0.01	−0.14	−0.33*
Ages 25–64 females	0.05	0.03	0.09
Ages 65–74	0.07	0.06	−0.19
Ages 65–74 males	0.21	−0.22	−0.16
Ages 65–74 females	0.13	−0.15	−0.17
Traffic-related mortality (V01–V99)[a]	−0.67***	0.69***	−0.27
Males	−0.53**	0.58***	−0.21
Females	−0.51**	0.50**	−0.30†
Ages 15–24	−0.65***	0.68***	−0.10
Ages 25–34	−0.53**	0.39*	−0.46**
Ages 35–44	−0.17	0.19	−0.12
Ages 45–54	−0.57***	0.60***	−0.25
Ages 55–64	−0.40*	0.41*	−0.29

Table 4 continued

Mortality rate	Unemployment	GDP	Average hours in manufacturing
Ages 65–74	−0.21	0.24	−0.02
Ages 75–84	−0.24	0.25	0.02

Notes: $n = 32$ or close to 32 for each correlation model. Variables detrended with the HP filter, $\gamma = 100$. Age-standardized death rates from the WHO-Europe HFA Database

[†] $P < 0.1$; * $P < 0.05$; ** $P < 0.01$; *** $P < 0.001$

[a] Codes according to the International Classification of Diseases, 10th edition (ICD10)

growth has a significant negative effect on life expectancy, while unemployment and average hours have significant positive effects. Similar patterns are observed for crude mortality or mortality for specific ages. Interpreting the sign of the net effect, most of these models suggest procyclical mortality, but considering the size of the net effect (adding up effects at lags zero and one), only the model for mortality at ages 45–64 regressed on GDP growth (Table 5, panel A) reaches marginal statistical significance (the net effect $0.003 + 0.022 = 0.025$ has an standard error SE = 0.013; $P = 0.09$). Models with average hours as economic indicator (Table 5, panel C) only require coincidental effects (at lag zero) and they suggest procyclical mortality when the economic indicator is either life expectancy, crude mortality, or mortality at ages 45–64.

In these regression models (Table 5), the Durbin–Watson d is usually either close to 2.0 or above 2.0. Since $d = 2[1 − r]$, where r is the estimated autocorrelation, $d \geq 2$ implies evidence against positive autocorrelation of the residuals which would result in underestimates of standard errors and spurious significance.[8]

We also examined some models in which an age-standardized mortality rate is regressed on coincidental or lagged GDP growth (Table 6). For all-cause mortality, sex-specific age-adjusted mortality for both males and females and sex-specific age-adjusted mortality in ages 25–64 (Table 6, panel A), the specification minimizing AIC is the one including effects at lags zero and one. With only a few exceptions in which lag-two effects are also needed, this is also true for CVD mortality in different demographic groups (Table 6, panel B). However, for traffic mortality (Table 6, panel B, bottom) the minimization of AIC occurs in specifications including just lag-zero effects.

For total mortality, male mortality and female mortality and for male mortality at ages 25–64 predominantly positive and statistically significant effects of GDP growth on mortality at lag one indicate a procyclical oscillation of mortality rates. For CVD mortality at all ages and ages below 65, positive (and significant) effects of GDP growth at lag one also indicate a procyclical oscillation of this type of mortality. However, for CVD mortality at ages 65 and over what predominates is negative effects of GDP growth (at lags zero and two).

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[8] In only a few cases d is large enough to suggest negative autocorrelation (see footnote in Table 5). Adjusting for autocorrelation of the residuals did, as expected, produce similar parameter estimates with some additional statistical significance. To avoid the need to justify selection of a specific autocorrelated model, we simply present the conservative unadjusted values.

Table 5 Parameter estimates in models with a health indicator in first differences regressed on a constant, and coincidental and lagged values—except in panel C—of an economic indicator

Explanatory variable	Dependent variable	Lag 0	Lag 1	n	d	R^2	Model	Interpretation of the net effect
A. Economic growth (percent rate of change of real GDP)	Life expectancy	0.027 (0.019)	**−0.041** (0.019)*	31	2.70[a]	0.15	[1]	Procyclical mortality
	Crude mortality	−0.039 (0.023)	**0.048** (0.023)*	32	2.74	0.14	[2]	Procyclical mortality
	Mortality ages 20–44	0.003 (0.003)	0.000 (0.003)	35	2.46	0.02	[3]	
	Mortality ages 45–64	0.003 (0.012)	**0.022** (0.012)†	35	1.76	0.12	[4]	Procyclical mortality
	Mortality ages 65–84	**−0.173** (0.085)†	0.124 (0.085)	35	2.89	0.13	[5]	Countercyclical mortality
B. Unemployment rate in first differences	Life expectancy	−0.026 (0.042)	**0.076** (0.043)†	30	2.64	0.11	[6]	Procyclical mortality
	Crude mortality	0.076 (0.050)	**−0.119** (0.050)*	34	2.45	0.15	[7]	Procyclical mortality
	Mortality ages 20–44	−0.006 (0.007)	0.002 (0.007)	34	2.55	0.02	[8]	
	Mortality ages 45–64	−0.018 (0.025)	−0.018 (0.025)	34	1.62	0.07	[9]	
	Mortality ages 65–84	**0.327** (0.186)†	**−0.379** (0.187)†	34	2.45	0.13	[10]	Equivocal
C. Average hours in manufacturing, in first differences	Life expectancy	**0.043** (0.023)†		31	2.96[a]	0.11	[11]	Procyclical mortality
	Crude mortality	**−0.059** (0.027)*		35	2.93[a]	0.12	[12]	Procyclical mortality
	Mortality ages 20–44	−0.002 (0.004)		33	2.43	0.01	[13]	
	Mortality ages 45–64	**−0.033** (0.014)*		35	2.11	0.14	[14]	Procyclical mortality
	Mortality ages 65–84	−0.014 (0.105)		35	2.96[a]	0.05	[15]	

Notes: Standard errors in parentheses. Data include years from 1968 to the early 2000s. Estimates statistically significant at a confidence level of 90% or higher are highlighted in *boldface*. Mortality rates are deaths per 1,000 population, life expectancy in years. In similar models using age-specific mortality for ages 85 and over the 'best' model was the one including just the lag-zero effect, but the parameter estimates and its standard errors were approximately of the same size—that is, the effect was indistinguishable from zero—and the R^2 was 6% or lower

† $P < 0.1$; * $P < 0.05$; [a] $P < 0.05$ for the null hypothesis of no negative autocorrelation of the error term

Table 6 Regression estimates of the coincidental and lagged effect of GDP growth on the annual change in age-standardized mortality rates in models minimizing AIC among models including just lag zero, lags zero and one, or lags zero to two

Mortality rate	Lag 0	Lag 1	Lag 2	Oscillation of mortality according to the net effect
A. General mortality				
All ages	−0.97	3.35*		Procyclical
Males	−1.86	4.68*		Procyclical
Females	−0.36	2.38†		Procyclical
Infant mortality (age < 1)	−0.04	0.06†		Procyclical
Ages 25–64				
Males	−0.34	1.65*		Procyclical
Females	1.05†			Procyclical
B. Cause-specific mortality				
Cardiovascular disease				
All ages	−1.45	2.16*		Procyclical
Ages 0–64	−0.34	0.56**		Procyclical
Ages 45–54	−0.15	0.59		Procyclical
Ages 55–64	−1.67	2.71*		Procyclical
Ages 65–74	−1.83			Countercyclical
Ages 75–84	−15.80	26.08*	−13.85	Equivocal
Ischemic heart disease				
All ages	−0.95	1.65*		Procyclical
Males	−1.98	3.45*	−2.03	Equivocal
Females	−0.69	1.08*		Procyclical
Ages 25–64	−0.19	0.62*		Procyclical
Males	−0.45	0.94†		Procyclical
Females	0.07	0.30†		Procyclical
Ages 65+	−7.70	11.96†		Procyclical
Ages 85+	−57.47	81.72*	−47.04	Equivocal
Cerebrovascular disease (stroke)				
All ages	−0.10	0.46*		Procyclical
Ages 25–64	−0.16	0.30**		Procyclical
Ages 65–74	0.30			Countercyclical
Traffic injuries				
All ages	0.15*			Procyclical
Males	0.20*			Procyclical
Females	0.10			Procyclical
Ages 15–24	0.28			Procyclical
Ages 25–34	0.00			
Ages 35–44	0.08			Procyclical
Ages 45–54	0.13			Procyclical
Ages 55–64	0.22			Procyclical

Table 6 continued

Mortality rate	Lag 0	Lag 1	Lag 2	Oscillation of mortality according to the net effect
Ages 65–74	0.46[†]	−0.32		Procyclical
Ages 75–84	0.09			Procyclical

[†] $P < 0.1$; * $P < 0.05$; ** $P < 0.01$

To allow for an appropriate AIC comparison among models with different lags the sample was trimmed to the years 1970–2000 ($n = 31$) in all models. Except infant mortality (in deaths per 1,000 life births), all mortality rates are per 100,000 population and age-standardized by age intervals of 5 years

In regressions modeling coincidental and lagged effects of GDP growth on ischemic heart disease mortality (Table 6, panel B), almost without exception there are negative effects at lag zero and positive effects at lag one, and the positive effects at lag one are often statistically significant and predominant. However, for ischemic heart disease mortality in males the model minimizing AIC also includes effects at lag two and the net effect of GDP growth on this type of mortality in males is negative, i.e., ischemic heart disease mortality decreases with greater GDP growth. Overall, this indicates a countercyclical oscillation of ischemic heart disease mortality. However, the effects at lag two are difficult to interpret. Business cycles over recent decades in Sweden last approximately 4 years (according to the OECD chronology, the mean ± SD of the trough-to-trough distance is 3.8 ± 1.4 years). This implies that an effect of high GDP growth lagged 2 years will likely occur actually when GDP growth is already low in the next phase of the cycle. For ischemic heart disease at ages 85 and over, there are negative effects at lags zero and two and a significant positive effect at lag one, so that the net effect of GDP growth is negative, the positive effect for lag one is statistically significant but the net effect is not.

Overall, considering lag regression models (Tables 5, 6) and correlations (Tables 1, 2, 3, 4) for the period 1968–2003, the results are consistent for major health indicators. Results for life expectancy, total mortality, and crude or age-adjusted total mortality in large age groups indicate a procyclical fluctuation of death rates for the whole population and for both males and females. Less consistent are the correlations and regression results for age-specific mortality rates. The link between economic expansions (recessions) and increases (falls) in mortality rates seems to be stronger in adolescents, young adults and adults of middle age. Infant mortality also fluctuates procyclically. Results for ages 65–84 or over 65 are equivocal, and for mortality at ages 85 and over we could not find any statistically significant evidence of a link with business cycle fluctuations.

5.3 Size of the Effect

The net effects of GDP growth at lags zero) and lag one (−0.97 +3.35 = 2.32) on total age-adjusted mortality measured per 100,000 population (Table 6, panel A) imply that each extra percentage point in GDP growth will increase mortality in

about 2.32 deaths per 100,000. In recent years the age-standardized death rate is 600 per 100,000. Since the Swedish population is about 9 million, each percentage point increase in GDP growth would cause $[2.32/10^5] \times 9 \times 10^6 = 209$ extra deaths, or an increase of a slightly less than half a percent $(2.32/600 = 0.4\%)$ in age-adjusted mortality. During the period of study GDP growth oscillated between -2% in major downturns and 6% in major expansions. Therefore, a typical expansion in which GDP growth increases three or four percentage points would increase age-adjusted mortality between one and two percentage points, with an additional death toll of 600 to 800 fatalities.

Modeling the relation between macroeconomic change and health using the annual variations in unemployment (ΔU_t) and life expectancy $(\Delta e_{0,t})$ the estimated equation (error term omitted) is

$$\Delta e_{0,t} = 0.164 - 0.026 \cdot \Delta U_t + 0.076 \cdot \Delta U_{t-1}$$

(this is model [6] in Table 5). It implies a gain of 0.164 years in life expectancy from time $t - 1$ to time t after no change in unemployment from time $t - 2$ to time $t - 1$ and from time $t - 1$ to time t (that it, $\Delta U_t = \Delta U_{t-1} = 0$). On the other hand, the gain in life expectancy will be zero $(\Delta e_{0,t} = 0)$ with two successive equal drops in annual unemployment of 3.3 percentage points—which in macroeconomic terms means a brisk economic expansion—while greater annual drops in unemployment will *reduce* life expectancy. A recession in which unemployment grows 2 percentage points for two consecutive years $(\Delta U_t = \Delta U_{t-1} = 2)$ will be associated with an annual increase of 0.264 years in life expectancy, a gain 2.3 times greater than the 0.114 years gained in an expansion during which the unemployment rate consecutively drops one percentage point per year $(\Delta U_t = \Delta U_{t-1} = -1)$. For reference, it is important to note that during the period included in this investigation life expectancy at birth increased about 5 years during three decades, for an annual gain of 0.18 years, while the unemployment rate never increased more than 3.8 percentage point or dropped more than 1.3 points per year.

6 Results of Other Investigations on Sweden

The sample in Gerdtham and Johannesson's study is very large, including over 500,000 person-year observations (Gerdtham and Johannesson 2005, p. 205), However, their analysis only includes 6,571 deaths occurring over 16 years, while our analyses include *all deaths* that occurred in Sweden for each year of the study period, i.e., over 90,000 deaths per year for a total of over 3 million deaths during 1968–2002. National death rates give greater statistical power than a sample of 6,571 deaths to investigate various model specifications and, particularly, to check model consistency across population subgroups. The sample size limitations in the analyses reported by Gerdtham and Johannesson are illustrated for example by the fact that they had only 43 traffic-related deaths (p. 214), which implied that the business-cycle impact on this cause of death could not be analyzed. Moreover, though Gerdtham and Johannesson present some supplementary analyses using education and income as individual-level covariates, the only individual covariate

used in their primary analysis is the age at death. We also control for age in our analyses by using the aggregate measures of life expectancy and age-specific mortality rates.

According to Gerdtham and Johannesson (2005, p. 210), when quadratic and cubic trends were included in their models, some statistically significant results were weakened. Since linear trends may be insufficient to account for the long-term curvilinear trends in indicators of the Swedish economy in recent decades (Fig. 1), the imperfect control for these trends may have produced biased results. For instance, the enormous size of the effect of macroeconomic change on cancer mortality reported by Gerdtham and Johannesson may imply model misspecification. Similarly, their finding that results vary from procyclical to countercyclical fluctuation of mortality depending on the economic indicator used suggests instability in the results. There are also limitations to the indicators they used. For example, opinion-based variables such as the confidence indicator or the notification rate may be very imperfect indicators of the fluctuations of the real economy. Indeed, we have verified that the confidence indicator, one of the indicators rendering countercyclical results for mortality in Gerdtham and Johannesson's investigation, reveals a very erratic relation with GDP, unemployment, and all the other business indicators based on manufacturing, and is therefore likely to be a very unreliable index (neither procyclical nor countercyclical) of the changes in the real economy.

Another limitation of Gerdtham and Johannesson's investigation is the short period of data collection, which allows for few recurrent changes in the state of the economy. In their article they state (p. 209) that the relation between unemployment and probability of death is negative for unemployment (so that mortality is procyclical) over the whole study period, 1981–1996, but turns positive (counter-cyclical) for male, and zero for females, in the subperiod 1981–1991. Gerdtham and Johannesson consider the procyclical mortality result obtained for the years 1981–1996 to be biased by the high unemployment in the 1990s. However, if there is a real relation (whether procyclical or countercyclical) between the fluctuations of the economy and the mortality risk, the relation will tend to appear most clearly in samples covering longer periods and will tend to became unstable or to disappear as the analyzed period gets shorter and closer to the average duration of business cycles. This instability is shown, for instance, by our split-sample correlations of HP-detrended unemployment and mortality at ages 45–64 (Table 3 in this article). The correlation between these variables is positive (i.e., a countercyclical fluctuation of mortality) in the years 1981–1992 (the sample often used by Gerdtham and Johannesson), though it is negative (suggesting procyclical mortality) in other split samples (Table 3, panel B), and over the whole period 1969–2003 (Table 1). Business cycles may vary in length "from more than one year to ten or twelve years"—a classical definition by Mitchell (1951, p. 6) that fits very well with the data analyzed here, with macroeconomic fluctuations on average 4 years long. Therefore, 10 years may be too short a period to study any business-cycle-related issue. Indeed, the period 1981–1991 that Gerdtham and Johanesson often use to present results is one in which unemployment changed very little and GDP growth varied within very narrow limits (Fig. 1). Observing only part of the full range of

variation of a covariate is one of the potential causes of biased results in any statistical model.

Most of the results supporting a countercyclical oscillation of mortality (only for males) in Gerdtham and Johannesson's investigation come from models in which the only considered effect of the economy on mortality is a coincidental one. As reported by the authors, adding lagged variables with lags up to 4 years produced procyclical mortality effects at $t - 1$ for both unemployment and GDP deviation from trend. However, since the coefficients for the lagged economic indicator in these specifications are not reported by Gerdtham and Johannesson, it is not possible to evaluate the net effect.

It is also important to note that a positive impact of GDP growth and a negative impact of unemployment on the death rate in the next year is consistent with our results and indicates a procyclical oscillation of mortality.

In summary, we believe the results of Gerdtham and Johannesson (2005) are much less reliable than ours because insufficient consideration for lagged effects, use of unreliable business-cycle indicators, data collected during a very short period in terms of business cycles, and lack of power to detect effects due to few deaths in their sample.

We performed some analyses using the Swedish regional data used by Svensson (2007, 2010). We reproduced his results in several specifications of panel regressions. Then we reanalyzed his data, detrending the mortality and the unemployment regional data by either subtracting an HP trend ($\gamma = 100$) or by converting variables into first differences. We investigated models of the form

$$\mathrm{d}M_{r,t} = \alpha + \beta_i \cdot \sum_{i=0}^{k} \mathrm{d}U_{r,t-i} + \varepsilon_{r,t} + \psi_t$$

where $\mathrm{d}M_{r,t}$ is detrended mortality of region r at year t, $\mathrm{d}U_{r,t-i}$ is detrended unemployment for region r at year $t - i$, $\varepsilon_{r,t}$ is a normally distributed error term, and ψ_t is a normally distributed random effect for year. This random effect (Venables and Ripley 2002) adjusts for the spatial autocorrelation between regions (Layne 2007). To explore lagged effects we estimated models for $k = 0$, $k = 1$, $k = 2$, $k = 3$ and for each dependent variable we chose the model minimizing AIC. The best fit in terms of AIC was obtained when the model included effects at lags zero and one, or at lag zero only (Table 7). However, for some mortality rates, AIC is minimized in specifications including lags zero to two, or even lags zero to three. This and the effects with alternating sign that are difficult to explain makes some of these results puzzling, revealing the complexities of interpreting lag regression models. At any rate, what these regional models show is that negative and often statistically significant effects of regional unemployment predominate at lags zero or one, for total mortality and for sex-specific mortality, indicating a procyclical fluctuation of total and sex-specific mortality. With very few qualifications the same can be said for mortality at ages 45–64 and 65 and over, as well as traffic mortality. However, for ischemic heart disease mortality positive effects that predominate at lags zero and two make the interpretation of these effects equivocal.

Table 7 Effects of the regional unemployment rate on regional mortality in lag regression models

Mortality rate	Lag 0	Lag 1	Lag 2	Lag 3
A. Percentage change in mortality regressed on differenced regional unemployment				
Total mortality	0.05	−0.36†		
Male mortality	0.36	−0.66**		
Female mortality	−0.21			
Ages 20–44	−0.47			
Ages 45–64	0.08	−0.56		
Ages 65+	0.08	−0.40†		
Ischemic heart disease	0.72	−0.57	1.00*	
Traffic injuries	−2.65	1.29	−5.59**	3.40†
B. Regional mortality and regional unemployment as HP-detrended series, $\gamma = 100$				
Total mortality	−0.14	−3.73*		
Male mortality	3.88†	−8.29**	1.99	3.98†
Female mortality	−3.69†			
Ages 20–44	−0.90	0.70	−1.34*	
Ages 45–64	2.73	−4.59*		
Ages 65+	3.31	−18.18†		
Ischemic heart disease	2.80*	−2.27	4.36**	
Traffic injuries	−0.20†			

† $P < 0.1$; * $P < 0.05$; ** $P < 0.01$

For each dependent variable (mortality rate) the lag specification selected is that minimizing AIC out of the four possible lag lengths. Data for the years 1980–2005, in 21 Swedish regions. Models including a random effect for each year, to adjust for the spatial autocorrelation of the regional mortality rates. Heteroskedasticity over space in the panel regression was successfully stabilized by weighting regional observations by the square root of the population size

Considering all the results of regressions with regional data,[9] we interpret the negative signs of the predominant effects at lag one as evidence consistent with our analysis of national data indicating a procyclical oscillation of total, male and female mortality, as well as mortality at the three specific ages that Svensson investigated. We believe that Svensson's analysis of regional data missed the procyclical oscillation of death rates primarily because he did not consider lagged effects. In addition, his panel methods (including fixed effects for year, and sometimes region-specific linear trends too) could be less statistically powerful than our random effect specification.

7 General Discussion

The procyclical fluctuation of national Swedish mortality rates revealed by the results in earlier sections is consistent with results from other industrialized

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[9] In regional panel models not including a random effect for year, the unemployment effects were greater in size and much more significant than those reported in Table 7. However, we consider them probably biased by spatial autocorrelation.

countries; it is based on the period 1968–2003, which included eight or nine macroeconomic fluctuations; it is found in total, male, and female mortality, as well as age-specific mortality for most age groups; it is revealed by the correlations of mortality with seven economic indicators and also by lag models in which health indicators are regressed on an economic indicator; and, finally, it is consistent with the procyclical fluctuation of total and age-specific mortality rates (and the countercyclical fluctuation of life expectancy at birth) found by Tapia Granados and Ionides (2008) in time series for the second half of the twentieth century. In contrast, the countercyclical fluctuation of mortality reported by Gerdtham and Johannesson (2005) is inconsistent with other recent results from industrialized countries; is observed only for male mortality; appears only for selected business cycle indicators (some of questionable validity); and is sensitive to the inclusion of lagged terms in the regressions, or to the change in the time frame considered, often appearing only when the time frame is restricted to the expansionary years 1981–1991. Moreover, with regional data, Svensson (2007, 2008) was unable to reproduce Gerdtham and Johannesson's finding of a countercyclical fluctuation of male mortality, while our reanalysis of Svensson's regional data produced results that are basically consistent with the other results presented in this article, as well as with the results in Tapia Granados and Ionides (2008). Considering all the evidence, it must be concluded that in recent decades total mortality in Sweden, as well as male and female mortality individually, has oscillated procyclically.

CVD is the first cause of death in most countries and in Sweden about half of all deaths are attributed to this cause. CVD deaths have been found to be strongly procyclical at lag zero in the United States (Ruhm 2000; Tapia Granados 2005b), Germany (Neumayer 2004), and the OECD countries as a group (Gerdtham and Ruhm 2006), while in Spain they were found to be very slightly procyclical (Tapia Granados 2005c). In Sweden, the lag-zero correlations of CVD mortality with economic indicators (Table 4) do not reveal a comovement of both, but lag regression models indicate a predominantly positive net effect of GDP growth at lags zero and one on this type of mortality (Table 6). Therefore, overall CVD mortality in Sweden tends to rise in expansions, procyclically. The same pattern is observed for stroke mortality at all ages, or at ages below 65 (Table 6, panel B), though for ages 65–74 the preferred model only includes a positive effect at lag zero, what implies a countercyclical oscillation of stroke mortality at these ages.

Findings for ischemic heart disease are much more difficult to interpret. When modeled in lag regressions at the national level (Table 6, panel B) ischemic heart disease mortality reveals predominantly positive effects of GDP at lag one. This suggests a procyclical fluctuation, with heart attacks fluctuating upward 1 year after the start of an expansion and fluctuating downward 1 year after the start of a recession. However, in regional regression models (Table 7) positive effects of unemployment at lags zero and two predominate and cross-correlation analyses of national data also suggest positive effects of unemployment, i.e., countercyclical effects on ischemic heart disease mortality at ages 65 and over (Table 4). But most fatal heart attacks (i.e., deaths attributed to ischemic heart disease) occur at advanced age. Additional work is needed to better identify the effects of the

economy on ischemic heart disease mortality and why it may oscillate differently than other causes.

In our findings traffic injury mortality correlates strongly with the business cycle at lag zero (Fig. 2), particularly at young and middle ages (Table 4). Regression models in which the change in traffic mortality is regressed on coincidental and lagged GDP growth (Table 6, panel B, bottom) also reveal this procyclical oscillation of traffic mortality at all ages and for males, but at specific ages the regression coefficients are indistinguishable from zero and in panel models with regional data (Table 7) marginally significant effects appear at lag three. Two factors may explain these results. *First*, the differencing of a time series, converting it into a rate of change or a first difference series, largely filters out the fluctuations in business-cycle frequencies. This can result in failure to detect relations between variables at the business-cycle frequencies (Baxter and King 1999) and is one of the reasons for the increasing use of the HP filter and other detrending procedures instead of differencing. *Second*, strong associations at a larger level of aggregation (at all ages or at the national level) may weaken when mortality is disaggregated (by age strata or by regions) because of the introduction of statistical noise.

The strongly procyclical character of CVD mortality in general (Ruhm 2000; Tapia Granados 2005c) and heart attack mortality in the United States for all ages (Ruhm 2008) contrasts with the absence of a clear procyclical oscillation in ischemic heart disease mortality reported here and elsewhere (Svensson 2007) for Sweden. It has been previously proposed that the Swedish welfare state—with labor regulations and a labor market more friendly for employees than in the United States—could be a factor in explaining a lower impact of macroeconomic fluctuations on health in Sweden (Gerdtham and Ruhm 2006; Ruhm 2006; Svensson 2007) and this hypothesis deserves further investigation.

It is plausible that differences between institutional regulations of working conditions and labor markets between Sweden and the United States may perhaps account for the differential business-cycle dynamics of CVD mortality in both countries. The index of average hours worked in manufacturing has long been considered a leading business-cycle indicator (Mitchell 1951). Early in expansions firms demand overtime from workers and tend to hold off on hiring additional workers until managers are confident that demand is growing. Then average hours will increase early in an expansion and then will decrease. Similarly, early in a recession employers will reduce hours worked to reduce output and costs, and average hours will go down, but if the slowdown deepens into a recession, layoffs eventually will raise average hours. Differences in the ability of firms to hire and fire workers easily, or demand overtime from employees may well account for the fact that the index of average hours in manufacturing is procyclical in the United States and countercyclical in Sweden. The countercyclical character of this index, if present in other OECD economies—in many European countries mandatory overtime does not exist and there are generally more limitations for freely firing workers than in the United States (Chung 2007)—might explain why Johansson (2004), while getting results indicating an increase of mortality in periods of economic expansion, finds that an increase in hours worked per employed person

significantly decreases the mortality rate in a sample of 23 OECD countries during 1960–1997.

In this Swedish sample HP-detrended infant mortality significantly correlated negative with detrended unemployment (Tables 1, 3), and GDP growth has a marginally significant positive effect on infant mortality at lag one (Table 6), all of which suggest a procyclical oscillation of infant deaths. Potential pathways for procyclical infant mortality were mentioned in Sect. 3. On the other hand, since traffic-related deaths are intensely procyclical and a major cause of mortality in adolescents and young adults (Table 4), they are a very likely cause of the procyclical oscillation of deaths at ages 10–19 (Tables 1, 2).

We report only bivariate models. Therefore, our results can be challenged on the grounds of potential bias due to omitted variables. However, results based on the analyses of economic indicators covering a number of business cycles and converted into stationary series by filtering or differencing make it very unlikely that omitted variables may significantly bias the results with respect to business cycle fluctuations. Indeed, many variables change together with the levels of economic activity (for instance, overtime, average levels of daily sleep, volume of road traffic, atmospheric pollution, etc.), and these are exactly the candidate intermediary factors connecting "the economy" with the health outcomes. It is difficult to imagine real omitted variables that are not an intrinsic part of the aggregate fluctuations of economic activity indexed in this study by variables such as unemployment or GDP. A lurking variable seriously biasing the present results might be, for instance, technological changes having an immediate or short-lagged contractionary effect on mortality, and appearing or being implemented population-wide late in each expansion or early in each recession, inducing in this way a procyclical oscillation of mortality. But this kind of technological innovation is hard to imagine, and it is even harder to believe that it would have such an intense effect on mortality fluctuations.

Results of this investigation can also be challenged on the basis of criticisms of methods of detrending in general, the HP filter in particular, or the use of $\gamma = 100$ when dealing with annual data (Baxter and King 1999; Dagum and Giannerini 2006). However, the HP filter is now a standard method for detrending economic data, and $\gamma = 100$ is very regularly used with yearly observations and is the default value for annual data in econometric software packages. Furthermore, our overall conclusions are robust to variations in the choice of γ. Contrary to the view that HP-filtering introduces business-cycle frequency fluctuations in the data, so that the computed correlations between HP-detrended series are just "discovering" a spurious artifactual pulsation introduced by the filter, in this investigation, cross-correlations and distributed lag models with variables either HP-detrended or differenced produced similar qualitative results, revealing procyclical oscillations of death rates. Panel regressions (Neumayer 2004; Ruhm 2000; Tapia Granados 2005a) and analysis of time series (Tapia Granados 2005b) have produced exactly the same conclusion when they were applied in the past to study the relation between macroeconomic change and mortality in other countries.

Based on the results of a number of statistical models reported here as well as our examination of the results of other investigations, we conclude that in Sweden

mortality of both males and females oscillated procyclically during recent decades. Since the long-term trend of age-adjusted mortality rates is a falling one, its procyclical oscillation means that age-adjusted mortality declines in recessions, while during expansions it declines less rapidly, or even stagnates or increases. The fact that even in a country like Sweden, with a highly developed system of social services and publicly financed health care, mortality tends to evolve for the worse during economic expansions must be a warning call about the unintended consequences of economic growth.[10] The policy implications of procyclical mortality are too broad to be discussed here, but a clear implication is the need to further study the pathways connecting the fluctuations of the economy with the oscillations of death rates. Knowledge of these mechanisms may lead to the development of policies to reduce the harmful effects of macroeconomic fluctuations on health.

References

Abdala, F., Geldstein, R. N., & Mychaszula, S. M. (2000). Economic restructuring and mortality changes in Argentina—Is there any connection? In G. A. Cornia & R. Paniccià (Eds.), *The mortality crisis in transitional economies* (pp. 328–350). New York: Oxford University Press.

Backus, D. K., & Kehoe, O. J. (1992). International evidence on the historical properties of business cycles. *American Economic Review, 82*, 864–888.

Baxter, M., & King, R. G. (1999). Measuring business cycles: Approximate band-pass filters for economic time series. *Review of Economics & Statistics, 81*(4), 575–593.

Brenner, M. H. (1971). Economic changes and heart disease mortality. *American Journal of Public Health, 61*(3), 606–611.

Brenner, M. H. (1982). Economic change and sex-specific cardiovascular mortality in Britain 1955–1976. *Social Science and Medicine, 16*(4), 431–442.

Brenner, M. H. (1987). Relation of economic change to Swedish health and social well-being, 1950–1980. *Social Science and Medicine, 25*(2), 183–195.

Chay, K. Y., & Greenstone, M. (2003). The impact of air pollution on infant mortality: Evidence from geographic variation in pollution shocks induced by a recession. *Quarterly Journal of Economics, 118*(3), 1121–1167.

Chung, H. (2007). Flexibility for employers or for employees? A new approach to examining labour market flexibility across Europe using company level data. In H. Jørgensen & P. K. Madsen (Eds.), *Flexicurity and beyond: Finding a new agenda for the European social model* (pp. 243–277). Copenhagen: DJØF.

Claeskens, G., & Hjort, N. (2008). *Model selection and model averaging*. Cambridge: Cambridge University Press.

Dagum, E. B., & Giannerini, S. (2006). A critical investigation on detrending procedures for non-linear processes. *Journal of Macroeconomics, 28*(1), 175–191.

Dehejia, R., & Lleras-Muney, A. (2004). Booms, busts, and babies' health. *Quarterly Journal of Economics, 119*(3), 1091–1130.

Diggle, P. J. (1990). *Time series—A biostatistical introduction*. Oxford: Clarendon Press.

Edwards, R. (2008). Who is hurt by procyclical mortality? *Social Science and Medicine, 67*(12), 2051–2058.

Eyer, J. (1977a). Does unemployment cause a death rate peak in each business cycle? *International Journal of Health Services, 7*(4), 625–662.

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[10] The potentially harmful consequences of economic growth for social welfare were already discussed decades ago from a variety of perspectives, for instance by Mishan (1970), Zolotas (1981), Eyer (1984), and Georgescu-Roegen (Randolph Beard and Lozada 1999).

Eyer, J. (1977b). Prosperity as a cause of death. *International Journal of Health Services, 7*(1), 125–150.

Eyer, J. (1984). Capitalism, health, and illness. In J. B. McKinlay (Ed.), *Issues in the political economy of health care* (pp. 23–59). New York: Tavistock.

Galloway, P. R. (1988). Basic patterns in annual variations in fertility, nuptiality, mortality, and prices in pre-industrial Europe. *Population Studies, 42*(2), 275–302.

Gerdtham, U.-G., & Johannesson, M. (2005). Business cycles and mortality: Results from Swedish microdata. *Social Science and Medicine, 60*(1), 205–218.

Gerdtham, U.-G., & Ruhm, C. (2006). Deaths rise in good economic times: Evidence from the OECD. *Economics & Human Biology, 4*(4), 298–316.

Glass, G. V., Willson, V. L., & Gottman, J. (1975). *Design and analysis of time-series experiments*. Boulder: Colorado Associated University Press.

González, F., & Quast, T. (2010). Macroeconomic changes and mortality in Mexico. *Empirical Economics,*. doi:10.1007/s00181-010-0360-0.

Graham, J. D., Chang, B., & Evans, J. (1992). Poorer is riskier. *Risk Analysis, 12*, 333–337.

Gravelle, H. S., Hutchinson, G., & Stern, J. (1981). Mortality and unemployment: A critique of Brenner's time-series analysis. *Lancet, 2*(8248), 675–679.

Higgs, R. (1979). Cycles and trends of mortality in 18 large American cities, 1871–1900. *Explorations in Economic History, 16*, 381–408.

Human Mortality Database. (2005). *A project of the University of California, Berkeley, and the Max Planck Institute for Demographic Research*, http://www.mortality.org/. Accessed 2005.

Isaacs, S., & Schroeder, S. A. (2005). Class—The ignored determinant of the nation's health. *New England Journal of Medicine, 351*(11), 1137–1142.

Johansson, E. (2004). A note on the impact of hours worked on mortality in the OECD. *European Journal of Health Economics, 4*, 335–340.

Junankar, P. N. (1991). Unemployment and mortality in England and Wales: A preliminary analysis. *Oxford Economic Papers, 43*, 305–320.

Kasl, S. (1979). Mortality and the business cycle: Some questions about research strategies when utilizing macro-social and ecological data. *American Journal of Public Health, 69*(8), 784–789.

Khang, Y. H., Lynch, J., & Kaplan-Oso, G. (2005). Impact of economic crisis on mortality in South Korea. *International Journal of Epidemiology, 34*(6), 1291–1301.

Layne, L. J. (2007). Appendix 4: Spatial autocorrelation. In S. J. Kunitz (Ed.), *The health of populations*. New York: Oxford University Press.

Lee, R. (1981). Short-term variation—Vital rates, prices, and weather. In E. A. Wrigley & R. S. Schofield (Eds.), *The population history of England 1541–1971—A reconstruction* (pp. 356–401). London: Edward Arnold.

Liu, Y., Tanaka, H., & Fukuoka Heart Study Group. (2002). Overtime work, insufficient sleep, and risk of non-fatal acute myocardial infarction in Japanese men. *Occupational and Environmental Medicine, 59*(7), 447–451.

McAvinchey, I. D. (1984). Economic factors and mortality: Some aspects of the Scottish case 1950–1978. *Scottish Journal of Political Economy, 31*(1), 1–27.

Mishan, E. J. (1970). *Technology and growth—The price we pay*. New York: Praeger.

Mitchell, W. C. (1951). *What happens during business cycles—A progress report*. New York: National Bureau of Economic Research.

Neumayer, E. (2004). Recessions lower (some) mortality rates—Evidence from Germany. *Social Science & Medicine, 58*(6), 1037–1047 [corrigendum in *Social Science & Medicine, 59*(9)].

OECD. (2006). Organisation for Economic Co-operation and Development (OECD). *OECD composite leading indicators: reference turning points*. http://www.oecd.org/document/41/0,2340,en_2649_34349_1891177_1_1_1_1,00.html. Accessed 2006.

Ogburn, W. F., & Thomas, D. S. (1922). The influence of the business cycle on certain social conditions. *Journal of the American Statistical Association, 18*, 324–340.

Randolph Beard, T., & Lozada, G. (1999). *Economics, entropy and the environment—The extraordinary economics of Nicholas Georgesçu-Roegen*. Northampton, MA: Edward Elgar.

Ruhm, C. J. (2000). Are recessions good for your health? *Quarterly Journal of Economics, 115*(2), 617–650.

Ruhm, C. J. (2006). Macroeconomic conditions, health, and mortality. In A. M. Jones (Ed.), *The Elgar companion to health economics*. Cheltenham, UK: Edward Elgar.

Ruhm, C. J. (2008). Macroeconomic conditions, health, and government policy. In R. F. Schoeni, J. S. House, G. Kaplan, & H. Pollack (Eds.), *Making Americans healthier: Social and economic policy as health policy*. New York: Russell Sage.

Schofield, R. (1985). The impact of scarcity and plenty on population change in England, 1541–1871. In R. I. Rotberg, et al. (Eds.), *Hunger and history: The impact of changing food production and consumption patterns on society*. New York: Cambridge University Press.

Søgaard, J. (1992). Econometric critique of the economic change model of mortality. *Social Science and Medicine, 34*(9), 947–957.

Sokejima, S., & Kagamimori, S. (1998). Working hours as a risk factor for acute myocardial infarction in Japan: A case-control study. *BMJ, 317*, 775–780.

Statistics Sweden. (2005). www.scb.se. Data downloaded in 2005–2009.

Svensson, M. (2007). Do not go breaking your heart: Do economic upturns really increase heart attack mortality? *Social Science and Medicine, 65*, 833–841.

Svensson, M. (2010). Economic upturns are good for your health but watch out for accidents: A study on Swedish regional data 1976–2005. *Applied Economics, 42*(5), 615–625.

Tapia Granados, J. A. (2005a). Recessions and mortality in Spain, 1980–1997. *European Journal of Population, 21*(4), 393–422.

Tapia Granados, J. A. (2005b). Increasing mortality during the expansions of the US economy, 1900–1996. *International Journal of Epidemiology, 34*(6), 1194–1202.

Tapia Granados, J. A. (2005c). Economic growth, business fluctuations and health progress. *International Journal of Epidemiology, 34*(6), 1226–1233.

Tapia Granados, J. A. (2008). Economic fluctuations and mortality in postwar Japan. *Demography, 45*(2), 323–343.

Tapia Granados, J. A., & Diez Roux, A. V. (2009). Life and death during the Great Depression. *Proceedings of the National Academy of Sciences of the USA, 106*(41), 17290–17295.

Tapia Granados, J. A., & Ionides, E. L. (2008). The reversal of the relation between economic growth and health progress: Sweden in the 19th and 20th centuries. *Journal of Health Economics, 27*, 544–563.

Tapinos, G. P., Mason, A., & Bravo, J. (1997). *Demographic responses to economic adjustment in Latin America*. New York: Clarendon Press.

Thomas, D. S. (1941). *Social and economic aspects of Swedish population movements, 1750–1933*. New York: Macmillan.

U.S. Bureau of Labor Statistics. (2005). http://www.bls.gov/fls/home.htm. Accessed 2005.

Venables, W. N., & Ripley, D. D. (2002). *Modern applied statistics with S* (4th ed.). New York: Springer.

Wagstaff, A. (1985). Time series analysis of the relationship between unemployment and mortality: A survey of econometric critiques and replications of Brenner's studies. *Social Science and Medicine, 21*(9), 985–996.

WHO-HFA. (2005). Heath for All Database, European Regional Office, World Health Organization. http:\\www.euro.who.int/hfadb. Data downloaded between 2005 and 2010.

Winter, J. (1983). Unemployment, nutrition and infant mortality in Britain, 1920–50. In *The working class in modern British history: Essays in honour of Henry Pelling*. Cambridge: Cambridge University Press.

Zolotas, X. (1981). *Economic growth and declining social welfare*. Athens/New York: Bank of Greece/New York University Press.