MACROECONOMIC FLUCTUATIONS AND MORTALITY IN POSTWAR JAPAN*

JOSÉ A. TAPIA GRANADOS

Recent research has shown that after long-term declining trends are excluded, mortality rates in industrial countries tend to rise in economic expansions and fall in economic recessions. In the present work, co-movements between economic fluctuations and mortality changes in postwar Japan are investigated by analyzing time series of mortality rates and eight economic indicators. To eliminate spurious associations attributable to trends, series are detrended either via Hodrick-Prescott filtering or through differencing. As previously found in other industrial economies, general mortality and age-specific death rates in Japan tend to increase in expansions and drop in recessions, for both males and females. The effect, which is slightly stronger for males, is particularly noticeable in those aged 45–64. Deaths attributed to heart disease, pneumonia, accidents, liver disease, and senility—making up about 41% of total mortality—tend to fluctuate procyclically, increasing in expansions. Suicides, as well as deaths attributable to diabetes and hypertensive disease, make up about 4% of total mortality and fluctuate countercyclically, increasing in recessions. Deaths attributed to other causes, making up about half of total deaths, don't show a clearly defined relationship with the fluctuations of the economy.

In recent years, a number of publications have shown that after long-term declining trends are excluded, mortality rates tend to oscillate with the so-called business cycles or trade cycles, increasing during expansions of the economy and dropping during recessions, thus fluctuating procyclically. Although this observation has been made in industrialized countries of high or medium income (Dehejia and Lleras-Muney 2004; Johansson 2004; Laporte 2004; Neumayer 2004; Ruhm 2000, 2003, 2004; Tapia Granados 2005a, 2005b), some reports suggest that this parallel fluctuation of death rates and the economy can also be observed in recent decades in some Latin American and Asian countries (Abdala, Geldstein, and Mychaszula 2000; De Rios Neto and Carvalho 1997; Khang, Lynch, and Kaplan 2005; Ortega Osona and Reher 1997), as well as in the turbulent 1990s in Russia (McKee and Suhrcke 2005). Conflicting results on the health effects of macroeconomic fluctuations in Sweden have been reported in recent years (Gerdtham and Johannesson 2005; Svensson 2007, forthcoming; Tapia Granados and Ionides forthcoming).

Mortality fluctuating with the economy—rising in expansions and falling in recessions—was documented by early twentieth-century researchers, such as Ogburn and Thomas (1922; Thomas 1927). From the 1970s forward, that finding was replicated by Eyer (1977a) and other researchers (Graham, Chang, and Evans 1992; Higgs 1979). Because these findings show a tendency of mortality to rise in periods of economic expansion, they call into question the argument that economic expansions have beneficial effects on health and that recessions have harmful effects (Brenner 1971, 1973, 1977, 1979a, 1979b, 1983, 1995; Catalano 1983, 1997) and thus have raised controversy (Brenner 2005; Catalano and Bellows 2005; Edwards 2005; McKee and Suhrcke 2005; Neumayer 2005; Ruhm 2005; Tapia Granados 2005c).

*José A. Tapia Granados, University of Michigan School of Social Work and Institute of Labor & Industrial Relations, 1111 East Catherine Street, Ann Arbor, MI 48109-0913; E-mail: jatapia@umich.edu. Two anonymous reviewers provided very insightful comments that substantially improved this paper. Chiho Kabeya and Mieko Yoshihama kindly helped with linguistic issues. Their valuable help is much appreciated. An expanded version of this paper provides more details of the analysis and is available on request.

Eyer (1977a, 1977b, 1984) was probably the first author who suggested a model in which specific pathways lead to the intensification of mortality during economic upturns; other pathways have been discussed more recently (Chay and Greenstone 2003; Dehejia and Lleras-Muney 2004; Neumayer 2005; Ruhm 2003, 2004, 2005; Tapia Granados 2005b). For example, the boost in motor-vehicle traffic and industrial activity during business upturns increases collisions and traffic deaths and also raises the level of atmospheric pollution, leading to cardiovascular events and increased deaths caused by respiratory processes.

Although cardiovascular disease and other chronic processes take years to develop, heart attacks peak on Mondays in the United States (Ruhm 2000) and on Sundays in Israel (Anson and Anson 2000), and deaths generally occur more frequently in the first week of each month (Phillips, Christenfeld, and Ryan 1999). This suggests that chronic processes can evolve to death with a very short lag because of intervening psychosocial or environmental factors. Occupational stress; lack of sleep; social isolation; increased consumption of tobacco, alcohol, and saturated fat; and atmospheric pollution (see Figure 1) might similarly contribute to trigger these deaths. Ruhm (2003, 2004, 2005) provided evidence showing harmful changes of these factors during expansions. In Japan, the popular notion of *karoshi*—that is, death owing to overwork—has been confirmed by medical studies (Sokejima and Kagamimori 1998).

A panel study of 23 industrialized countries belonging to the Organization for Economic Cooperation and Development (OECD), including Japan, found a procyclical oscillation of mortality (Gerdtham and Ruhm 2002 and 2006; Johansson 2004). To date, however, no specific investigation has been undertaken to assess the potential association of death rate oscillations with the fluctuations of the economy in Japan.

Japan is the world leader in life expectancy (78.6 years for males and 85.6 for females in 2004), and the Japanese economy in the postwar period has seen high and sustained rates of economic growth and very low levels of unemployment (Figure 2). For these reasons, it seems particularly interesting to ascertain whether, like in other industrialized countries, expansions and contractions of the economy in Japan have a noticeable covariation with mortality rates.

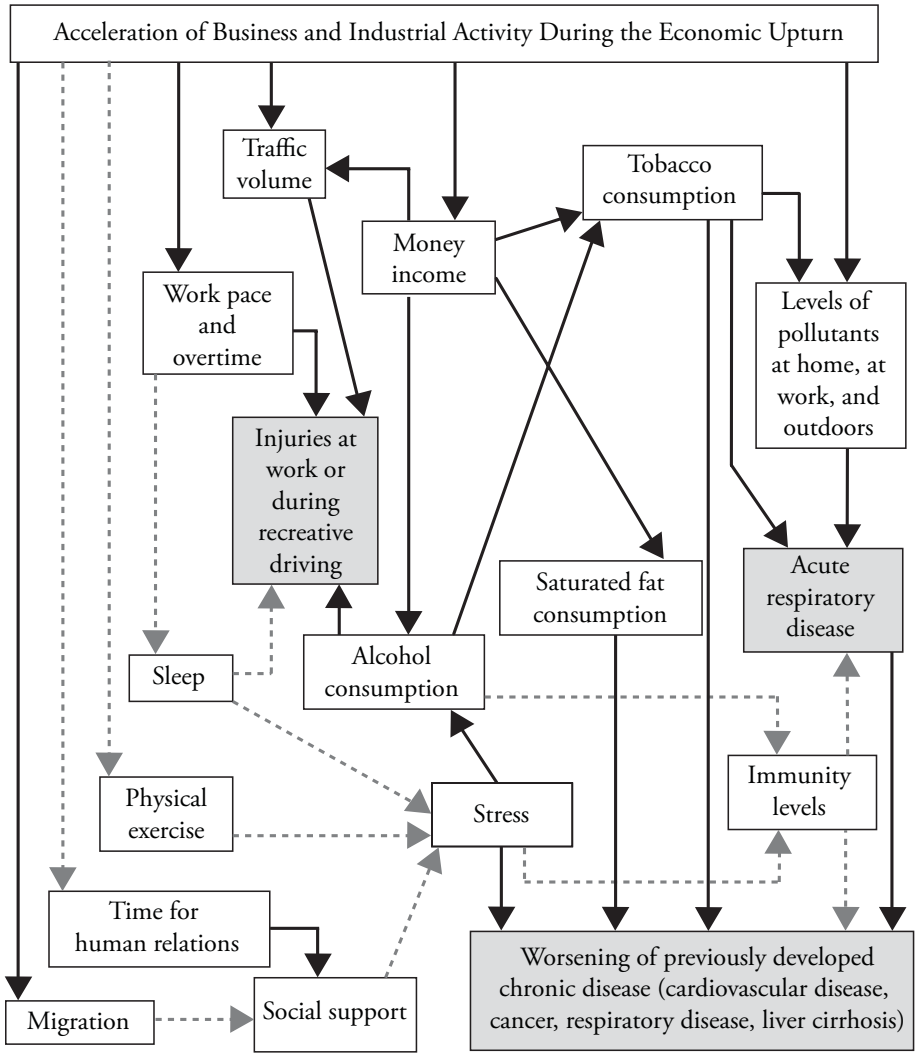
The results reported in this paper show that the association between economic fluctuations and recurrent changes in mortality found in other countries is also observable in Japan in the decades after 1950. Similar to other industrialized economies, in Japan mortality tended to deviate from its secular trend upward in periods of strong economic growth and downward during economic downturns.

DATA AND METHODS

All data analyzed in this study were obtained online from the Japanese Statistics Bureau (http://www.stat.go.jp/english/index.htm). The series of economic indicators and mortality rates are annual, starting in the 1950s (different series start in different years) and generally ending between 1995 and 2002.

The association between business fluctuations and the oscillations of mortality rates over and above the general declining trend were ascertained through cross-correlations and distributed lag regressions. Eight major indicators of the Japanese economy as well as a number of mortality rates, including age-and-sex specific and cause-and-sex-specific death rates, were used in the analysis. Because both mortality rates and economic indicators (even unemployment) have long-term trends in this sample (Figure 2), any correlation or regression model that uses untransformed series is strongly dependent upon the trend and tells nothing about the association between the oscillations in the two correlated variables. For that reason, all analyses presented in this paper correspond to detrended series, computed either with the Hodrick-Prescott (HP) filter, or by transformation of the series into first differences ($x_t - x_{t-1}$). Correlation and regression models with variables transformed into

Figure 1. Potential Causal Pathways Linking Economic Fluctuations to Mortality



Notes: Black, solid arrows represent positive effects. Gray, dashed arrows represent negative effects. (For instance, a drop in alcohol consumption raises immunity levels and decreases the risk of injuries.) The three shaded rectangles represent the final steps leading to death. Many other possible links (e.g., physical exercise directly improving immunity or releasing metabolites inhibiting heart disease) and bidirectional pathways (alcohol consumption increasing tobacco consumption and vice versa) have to be omitted to simplify the scheme.

rate of change ($[x_t - x_{t-1}] / x_{t-1} \approx \ln x_t - \ln x_{t-1}$) rendered similar results to models in first differences and are not reported.

The HP filter is a smoothing tool, increasingly used by economists, that generates a series not very different from that produced by a moving average (Figure 2). With a moving average, the wider the averaging window, the smoother the series that is produced; with the HP filter, the larger the smoothing parameter, the smoother the resulting trend. For annual data, a smoothing parameter $\gamma = 100$ is the usual option (Backus and Kehoe 1992). In this investigation, I also used $\gamma = 100$.

Pearson correlation coefficients were computed as cross-correlations between series of deviations of the original data with respect to the HP trend. These correlations were used to ascertain the concomitant variation: namely, parallel or mirroring movements of the health indicator series and the economic indicator. This procedure has been used in economics (Backus and Kehoe 1992; Baxter and King 1999), biology and medical science (Diggle 1989), and epidemiology (Tapia Granados 2005a). The procedure is founded on John Stuart Mill's principle of concomitant variation, which is a theoretical principle in the study of causality (Mackie 1974) that has also been applied in natural time-series experiments (Glass, Willson, and Gottman 1975).

The detrending through HP filtering or differencing takes care of trends that are due to changes in the demographic structure of the population and that affect particular mortality rates (crude mortality and cause-specific death rates) not adjusted for age. Because the changes in mortality attributable to population aging are long-term trends, the analysis of detrended series makes it possible to focus on mortality fluctuations as they relate in the short run to business-cycle upswings and downswings of the economy.

Macroeconomic fluctuations, or business cycles, are intuitively quite obvious, but they are difficult to describe and delimit with precision. Indeed, different economic indicators often provide a relatively different view of these swings of the market economy (Mitchell 1951). To verify the robustness of the results, I repeated the analyses for eight business-cycle indicators: the two more-traditional indicators, gross domestic product (GDP) and unemployment; four indicators of manufacturing activity, average hours, aggregate hours, employment, and output; and two indicators of the general level of employment, the employment/population ratio and the labor force participation ratio.[1] However, to save space, only the results for GDP, GDP lagged one year, unemployment, and the labor force participation ratio are presented in detail for the correlation models. The results for the other indicators are also consistent with the conclusion that mortality in Japan fluctuates procyclically, but are only briefly mentioned herein.

As a summary index of a multiplicity of business, commercial, and government activities, GDP is considered a key economic indicator. In the period I analyzed the Japanese economy, HP-detrended GDP shows a strong negative and statistically significant negative correlation (–.73, $p < .001$) with the unemployment rate equally filtered. The HP-detrended labor force participation ratio, reflecting the proportion employed or looking for work among all those of working age, correlates with HP-detrended GDP significantly at lag zero (.32, $p = .04$), but its strongest correlation with GDP is when this is lagged one year (.35, $p = .02$). This shows that the labor force participation ratio is a leading indicator.[2]

Correlation models provide an indication of the strength and character (parallel or mirroring) of the association between the oscillations in two series. However, from cross-correlations between HP-filtered series, it is difficult to quantify the effect of macroeconomic conditions on mortality levels. Data detrended with a smoothing filter often have high positive autocorrelation; therefore, they are not appropriate for regression models. Correlations are also inappropriate for exploring effects with distributed lags. For quantifying effects and exploring the simultaneous impact of coincidental and lagged variations of the economy on mortality, regression models are used. Ordinary least squares (OLS) are used to estimate equations of the following type:

$$\Delta m_t = \alpha + \sum_{i=0}^{k} \beta_i \cdot \Delta e_{t-i} + \varepsilon_t,$$

1. Unemployment is countercyclical; the seven other indicators are all procyclical in this sample.

2. Showing how difficult it is to put together the scheme of business cycles provided by different indicators, when these three indicators are converted into rate of change, the labor force participation ratio correlates only –.01 with GDP and –.12 with unemployment. When both are in rate of change, unemployment correlates –.58 ($p < .001$) with GDP.

in which Δm_t is the change in age-specific mortality from year $t-1$ to year t, α is a constant, and Δe_{t-i} is the change in an economic indicator from year $t-i-1$ to year $t-i$.

The observations for most analyzed series cover about one-half century and correspond to a period in which the Japanese economy underwent major transformations. Structural changes modifying the relation between variables cannot be ruled out *a priori.* To ascertain whether earlier macroeconomic fluctuations had a different effect on mortality than more recent ones, I computed regressions by splitting the available time-series samples in half. To formally test the possibility of structural change, I used the Chow test (Green 1993).

The fact that business-cycle indicators in first differences or in rate of change are highly correlated is a theoretical objection to using two or more of them in multivariate regressions to estimate the effect of macroeconomic change on mortality. However, to verify that this theoretical consideration also implies poor results in practice, I estimated some multivariate regressions in which "the economy" was represented by more than one variable, including unemployment and GDP, in levels or in change. Because the use of multiple economic variables did not substantially improve model fit, the results of these models are not reported here.

RESULTS

Correlation Models

The correlations of GDP, unemployment, and the labor force participation ratio with crude, age-specific or cause- and sex-specific mortality rates (Tables 1 and 2) show that general mortality and mortality attributed to the major causes of death oscillate procyclically. With few exceptions, age-specific death rates (Table 2) reveal positive correlations with GDP and the labor force participation ratio, and negative correlations with unemployment, indicating a procyclical oscillation of death rates. In absolute value, the correlations of death rates with GDP (both at lag zero and with GDP lagged one year) are weaker than the correlations of death rates with unemployment or the labor force participation ratio. As shown in other studies (Neumayer 2004; Ruhm 2000; Tapia Granados 2005a, 2005b), the effect of the business-cycle oscillations on mortality is usually captured by employment-based indicators much better than by GDP, and this seems also to be true in the case of Japan.

Given the small sample size, it is not surprising that many correlations don't reach statistical significance at the usual levels. The positive correlations of crude, age-adjusted, and age-specific mortality for all age strata except infant mortality and mortality at ages 1–14 with the other five procyclical business indicators (not shown) provide additional evidence that mortality oscillated procyclically in Japan during the study years.

The correlations of business cycle indicators with sex-specific crude mortality and sex-specific mortality at ages 20–44 and 45–64 (Table 1) suggest that the macroeconomic fluctuations have a stronger effect on male mortality. The correlations of cause- and sex-specific mortality rates with economic indicators (Table 2) are, for each cause, quite similar for male and female mortality except for a few cases that I discuss later. Deaths attributed to heart disease,[3] accidents, liver disease, and pneumonia show significant negative correlations with unemployment, and positive correlations with the procyclical indicators, GDP, and the labor force participation ratio: that is, they tend to increase in expansions and drop in recessions, procyclically. Mortality attributed to heart disease grows intensely in economic expansions and falls in recessions (Figure 3), and therefore shows strong negative correlations with unemployment (–.61, both for male and female mortality) as well as

3. In the Japanese statistics, this category of heart disease mortality excludes hypertensive disease mortality.

Table 1. Correlations Between Annual Series of Three Economic Indicators and Mortality Rates, All Detrended With the HP Filter: Japan, Circa 1960 to Recent Years

Mortality Rate	GDP	Unemployment	Labor Force Participation Ratio	GDP, Lagged One Year
Age-Adjusted Mortality	–.01	–.28†	.42**	.18
Crude Mortality	–.03	–.32*	.32*	.19
Males	.00	–.42**	.35*	.25
Females	–.03	–.41*	.23	.27†
Infant Mortality	.01	.13	.32*	.23
Mortality				
At ages 1–14	.01	–.24	–.01	.13
At ages 10–19	.22	–.07	.54***	.18
Males	.34*	–.12	.51***	.23
Females	.02	.04	.48**	.06
At ages 20–64	.06	–.29†	.42**	.28†
At ages 20–44	–.08	.00	.47**	–.02
Males	.21	–.16	.48**	.28†
Females	.00	–.09	.43**	.15
At ages 45–64	–.02	–.39*	.18	.16
Males	–.04	–.37*	.15	.12
Females	.00	–.36*	.25	.20
At ages 65–84	.03	–.32†	.26	.25
Males	.05	–.32*	.24	.23
Females	.01	–.31†	.25	.26†

Note: $n = 40$ for correlations involving unemployment; $n = 44$ or more for the correlations with other economic indicators.
†$p < .10$; *$p < .05$; **$p < .01$; ***$p < .001$

positive correlations with GDP (.35 and .37, respectively, for male and female mortality), and other procyclical indicators. Transportation accidents[4] (2.9% of all deaths in 1990) are intensely procyclical for both males and females, as shown by almost every economic indictor. Deaths attributable to "all accidents" (5.6% of all deaths) are much more intensely procyclical in males. Because this category includes injuries in the workplace, this sex difference is probably a manifestation of the larger participation of men in the labor force of the Japanese industrial sector.

Also clearly procyclical are the oscillations of mortality from pneumonia and liver disease. However, mortality attributed to senility[5] appears only very weakly procyclical; its correlation with unemployment is marginally significant and negative, and its correlations with all other procyclical indicators are positive but indistinguishable from zero.

4. "Transportation accidents" is the terminology used on the Japanese Statistics Bureau Web site. Avoiding the term "accidents" has been repeatedly recommended in the public health field (Loimer and Guarnieri 1996), but regrettably the recommendation is often ignored.

5. According to an anonymous reviewer, "dementia" might be a more proper term for this category that is termed "senility" by the Japanese Statistics Bureau. However, the Japanese term used in the table of Japanese statistics, *rosui*, refers to death by natural aging in cases in which no specific ailment is found, but an individual "runs out of life juice." It seems therefore proper to translate it into English as "senility."

Mortality attributable to suicide, diabetes mellitus, and hypertensive disease correlate positively with unemployment and negatively with GDP and all the other procyclical indicators. Therefore, these cause-specific rates oscillate countercyclically, increasing in recessions and falling in expansions. Among these three causes of death revealing a countercyclical oscillation, suicide is the salient one (Figure 3), with the correlation between suicide and unemployment much higher for males (.66) than for females (.40). The larger increase of suicide in males during recessions seems quite consistent with the role of males and females in modern society in general and Japanese society in particular.

Mortality attributed to diabetes seems to be countercyclical, increasing in periods of high unemployment. Similarly, mortality attributed to hypertensive disease seems to increase slightly in recessions, although the only significant correlations are with GDP; all other correlations are only marginally significant or are not significant.

Mortality attributed to gastric and duodenal ulcer, as well as to renal failure, shows some significant correlations with economic indicators. Because the signs of the correlations are inconsistent, however, it is very difficult to ascertain a procyclical or a countercyclical pattern. It is even more difficult in the case of cancer mortality: its marginally significant positive correlations with unemployment and its negative correlations at both lag zero and lag one with GDP seem to suggest a countercyclical movement, but the correlations are inconsistent with positive correlations with the labor force participation ratio and other procyclical indicators (not shown).

In correlation models with variables HP-detrended, mortality attributed to stroke (cerebrovascular disease), chronic bronchitis and emphysema, asthma, and tuberculosis did not reveal any significant correlations with economic indicators. Therefore, the corresponding results were excluded from Table 3.

Regression Models

I computed models in which a mortality rate was regressed on a constant and on coincidental and lagged values of an economic indicator—unemployment or GDP—with both the dependent and the independent variables converted either into first differences or into rate of change. Although some significant effects at lag one were found in models using GDP as the economic indicator, for models with unemployment as regressor, the lagged values of unemployment were not significant. In general, when using different economic indicators and mortality rates, I found no lagged effects beyond lag one. Therefore, there is no evidence of a delayed impact of economic change on mortality: the effect is rather coincidental, not lagged. Consequently, only the results of models with unemployment as the explanatory variable and without lagged terms are reported here.

The regression results show significant negative effects of unemployment on age-specific mortality rates at ages 45–64 and 65–84 (Table 3) and also on mortality attributed to heart disease, transportation injuries, and other causes of death (Table 4). For instance, for mortality at ages 65–84, the estimated equation (with standard errors in parentheses) is

$$\Delta m = -0.84 - 2.61 \cdot \Delta u,$$
$$\qquad (0.24)\ (1.06)$$

so that the model predicts a drop in mortality of 0.84 deaths per 1,000 population (a mortality reduction of 2.7%, assuming the mortality level observed in 1990) when unemployment does not change ($\Delta u = 0$). Because unemployment shows an increasing trend during the study period, and the mean change in the unemployment rate during the period was 0.05 percentage points, the expected mean annual variation in mortality at ages 65–84 is $\Delta m = -0.84 - 2.61 \cdot 0.05 = -0.97$; that is, a mortality reduction of about 1 death per 1,000 per year. Mortality will not change year to year ($\Delta m = 0$) when $\Delta u = -0.84 / 2.61 = -0.32$: in other words, the model predicts that mortality at ages 65–84 will not decrease

Table 3. Results of Models $\Delta m_t = \alpha + \beta \cdot \Delta u_t + \varepsilon_t$, in Which Age-Specific Mortality Is Regressed on Unemployment

	Mortality at Ages 20–44			Mortality at Ages 45–64			Mortality at Ages 65–84		
Sample	$\hat{\beta}$	$SE(\hat{\beta})$	SSE	$\hat{\beta}$	$SE(\hat{\beta})$	SSE	$\hat{\beta}$	$SE(\hat{\beta})$	SSE
1960–1996	0.07*	0.03	0.068	–0.14	0.12	0.859	–2.61*	1.06	71.89
1960–1977	0.09†	0.05	0.029	–0.36*	0.16	0.322	–5.17*	1.92	48.72
1978–1996	0.01	0.02	0.008	–0.13	0.09	0.133	–0.80	0.88	12.44
Chow *F* Test (*p* value)		13.82 (.001)			14.65 (.001)			2.89 (.070)	

Note: Δm_t = change in mortality from $t-1$ to t; Δu_t = change in unemployment; ε_t = error term; $SE(\hat{\beta})$ = standard error of the estimated unemployment effect $\hat{\beta}$; and SSE = sum of square errors.

†$p < .10$; *$p < .05$

whenever the unemployment rate diminishes by one-third of a percentage point. Mortality will increase with larger drops in unemployment.

Considering, respectively, the early and the late years of the study period, the effects on age-specific death rates are substantially different (Table 3). For instance, in 1960–1977, the unemployment effect on mortality is a statistically significant –0.36 for ages 45–64 and –5.17 for ages 65–84 (Table 3), and the corresponding effects on age-specific mortality at ages 45–64 and 65–84 in the years 1978–1996 are much weaker and not statistically significant.

The Chow test is just an *F* test to compare a whole sample model with the combination of two split samples: in this case, the periods 1960–1977 and 1978–1996. For mortality at ages 45–64,

$$F = [(0.859 - 0.322 - 0.133) / 2] / [(0.322 + 0.133) / 33] = 14.65,$$

(see the SSE values in Table 3), and with 2 degrees of freedom for the numerator and 33 degrees of freedom for the denominator, $p < .001$. Therefore, significant evidence exists of a structural change in the effect of business cycles on mortality at ages 45–64 before 1978 and in the most recent years. Applied to the same models with mortality for ages 65–84, the Chow test also suggests a significantly stronger effect for the years 1960–1977, although the test result is only marginally significant ($p = .070$).

Consistent with the results of correlation models (Table 1), regression models (Table 3) show procyclical oscillations of mortality at ages 45–64 and 65–84, although the negative effect of unemployment—that is, the procyclical oscillation of mortality—is stronger in the earlier years. The question, then, is whether this is consistent with the distribution of causes of death by age.

Models in which the change in cause- and sex-specific mortality is regressed on a constant and the change in unemployment (Table 4) reveal a very strong effect of macroeconomic fluctuations on death attributable to heart disease, suicide, and transportation injuries (note the high R^2 values). Since the unemployment effect is negative for heart disease and transportation injury deaths and positive for suicide, in the whole sample period 1960–1998, an increase of one percentage point in unemployment would reduce heart disease mortality by 9.99 deaths per 100,000 males but would raise male suicides by 6.29 suicides per 100,000. For heart disease deaths, transportation injury mortality, and suicides, the results of the regression models (Table 4) are therefore consistent with those of the correlation models with variables HP-detrended (Table 2). Both types of model

(Table 4, continued)

Cause-Specific Mortality	Unemployment Effect		R^2	Durbin-Watson *d*
	Coefficient	*SE*		
1960–1977 Sample (*n* = 18) (cont.)				
Suicide				
Male	3.64**	1.01	0.42	1.22
Female	1.44	0.88	0.14	1.20
1978–1998 Sample (*n* = 21)				
Heart disease				
Male	–12.17*	4.90	0.24	1.42
Female	–12.96*	5.71	0.21	1.56
Cerebrovascular disease (stroke)				
Male	3.77	6.09	0.02	2.13
Female	2.99	5.07	0.02	2.23
Transportation accidents				
Male	–1.10*	0.50	0.21	2.08
Female	–0.61*	0.21	0.30	2.60
Liver disease				
Male	–0.79	0.51	0.11	1.54
Female	–0.60	0.35	0.13	1.97
Pneumonia				
Male	–6.38†	3.30	0.16	2.04
Female	–3.84	2.67	0.10	2.66
Senility				
Male	–0.41	0.77	0.01	2.29
Female	–1.07	1.37	0.03	2.36
Suicide				
Male	7.76***	1.89	0.47	1.73
Female	1.80*	0.68	0.27	1.85

†$p < .10$; *$p < .05$; **$p < .01$; ***$p < .001$

reveal a procyclical oscillation of deaths attributable to heart disease and transportation injuries as well as a countercyclical oscillation for suicides. Regression models (Table 4) are also consistent with correlation models (Table 2) for mortality attributed to liver disease, pneumonia, and senility, all revealing a negative effect of unemployment. However, these effects revealing procyclical fluctuations of liver disease, pneumonia, and senility mortality are sensitive to period specification; they appear at statistically significant levels only in some samples; and, at any rate, they are much weaker than macroeconomic effects on heart disease, transportation accidents, and suicide mortality (note the low values for R^2).

Stroke mortality seems to be a special case. In regressions including the whole sample, the negative effect of unemployment on sex-specific death rates attributable to stroke (Table 4) is not significant—which is consistent with the correlation model results (Table 2)—and the models have a very poor fit ($R^2 \leq .04$). However, in the 1960–1977 sample, the negative effect of unemployment is massive for both male and female mortality. Then, in the 1978–1998 sample, the effect turns positive, although not significant. Plots of stroke mortality versus unemployment (Figure 4) do not reveal clear outliers in

1960–1977, but major outliers are noted for sex-specific stroke mortality in the years after 1977. For example, in 1994–1995, stroke mortality jumped from 91 to 114 deaths per 100,000 for males and from 102 to 121 for females. A further exploration of this issue is beyond the scope of this investigation.

In the regression models, the results for diabetes mellitus were erratic in sign and not statistically significant in any sample. Unemployment effects for cancer were positive but not statistically significant, even at the 90% confidence level in any sample. (Results for both cancer and diabetes were excluded from Table 4.) However, when either unemployment or GDP is used as the explanatory variable, the effect of the economy on cancer mortality at lag one is statistically significant and negative for GDP and positive for unemployment—that is, in a countercyclical direction. Mortality attributable to cancer might therefore be countercyclical, although this effect appears only in regression models when "the economy" is lagged one year.

Mortality and Share of Health Expenditures in GDP

The health expenditure share in the Japanese GDP rose from 2.9% in 1960 to 7.4% in 1998. When the series of health share in GDP is detrended by converting it in deviations from an HP-trend, and correlated with economic indicators similarly detrended, the health share proves to be strongly countercyclical because it correlates .57 with unemployment and –.76 with GDP.

In regressions in which the change in age-adjusted or age-specific mortality for ages 45–64 or 65–84 is regressed on the change in health share, a significant negative effect is noted on mortality at lag one. There are no significant effects at greater lags.

When the change in cause- and sex-specific mortality rates is regressed on a constant, plus coincidental and lagged values of the change in health share, negative significant effects are found for the health share on mortality attributable to heart disease (at lag one), pneumonia (at lag one), suicides (at lag two, but only for females), and traffic injuries (at lag zero).

These findings could be interpreted as suggesting that a higher health share in GDP reduces mortality for particular causes, although not for others. However, three factors suggest that the oscillations of the health share in GDP and the fluctuations in mortality are both dependent on the business cycle, without any causal dependence between them: (1) the strong correlation between the oscillations of the health share in GDP and the oscillations of the economy, (2) the fact that for mortality due to traffic injuries—in which health expenditure is likely to have a low or null impact—there is a statistically significant regression coefficient of the health share at lag zero, and (3) the lack of statistically significant effects of the health share on mortality due to cancer and diabetes—which *a priori* would seem much more susceptible to the influence of medical care. Multivariate models, including the coincidental or lagged values of unemployment *and* the health share that were significant in univariate regressions, do not disentangle this confusion. For mortality at ages 65–84, the health share remains significant, and unemployment is no longer significant. For heart disease mortality of both males and females, unemployment is the significant factor in the multivariate regression. All these models are just gauging ecological effects, and the colinearity between variables with such small samples does not allow separation of the effects.

DISCUSSION

The results presented here are similar to those obtained in studies from other countries (Neumayer 2004; Ruhm 2000, 2003, 2004, 2005, 2007; Tapia Granados 2005a, 2005b) in which most major causes of death have been found to be procyclical, whereas suicides have been found to be generally countercyclical. In previous studies, a procyclical fluctuation has been found in mortality caused by cardiovascular disease in general or acute ischemic disease in particular (Ruhm 2007). In this study, however, heart disease mortality

is procyclical, stroke deaths are unclearly related to macroeconomic fluctuations, and the residual category of hypertensive disease mortality looks weakly countercyclical.

In this study, the regression models in which mortality is set as a function of "the economy," with both variables detrended through conversion into first differences, have values of the Durbin-Watson d not very far from 2, and often greater than 2 (Table 3). Because $d = 2 \cdot (1 - \hat{a})$, where $\hat{a}$ is the estimated autocorrelation of the residuals, d values greater than 2.0 indicate that the autocorrelation is negative. Therefore, the estimate for the standard error is expected to be biased—too large—which will bias the p values upward. No spurious statistical significance is therefore to be expected in these models. In some of the models in which $d \leq 2$, the d values are small enough to suspect that the positive autocorrelation of the residuals may be biasing the results toward spurious statistical significance. However, these are only a few cases. Because the solution to the problem of autocorrelation of residuals—to introduce lagged values of the dependent variable as covariates—would strongly complicate the models and its interpretation, it was not attempted.

Although for age-specific mortality rates, the impact of macroeconomic change seems to be clearly weaker in the years 1978–2002 compared with the earlier years of the study period (Table 3), for particular causes of death (such as heart disease and suicide), the effect of business fluctuations (in opposite directions) is clearly stronger in the most recent years (Table 4). Around the year 2000, cancer deaths, which are acyclical or perhaps even countercyclical, were about twice the number of deaths attributed to heart disease; in 1960, cancer deaths exceeded heart disease deaths by just one-third. This and the increase in the countercyclical suicide share of total deaths seem to explain why for ages 45–64 and 65–84, the procyclical fluctuation of death rates is much weaker in the later years of the study period.

Sorting the causes of death according to the results shown, mortality rates fluctuating procyclically constitute about 41% of all deaths (Table 5), whereas mortality rates clearly fluctuating countercyclically constitute only about 4% of all deaths. Mortality attributable to cancer and all other analyzed causes of death showing an undefined relationship with economic fluctuations constitute about 52% of all fatalities. It is therefore consistent with these results that total mortality fluctuates procyclically because procyclical causes of death have a larger weight than countercylical deaths in total mortality. The higher the age, the higher the proportion of acyclical or countercyclical malignancy deaths and the lower the proportion of fatalities caused by procyclical heart disease or transportation injuries. This explains why mortality at ages 65–84 appears much less correlated with the economy than mortality at ages 45–64 (Table 1), even though the unemployment regression effect is much higher (Table 3) because mortality is much higher at these ages.

The results of correlation models based in HP-detrended series (Tables 1 and 2) are not always consistent with those of regression models in which variables are in first differences (Tables 3 and 4). Some significant associations of specific mortality rates (for instance, for ages 10–19 or for pneumonia) are not revealed by regression models in which variables are in first differences. The models in which annual change in mortality at ages 20–44 is regressed on annual change in unemployment (Table 3) suggest a very weak, although marginally significant, positive effect of unemployment on mortality: that is, a countercyclical fluctuation of this age-specific death rate. However, the correlations of HP-detrended mortality at ages 20–44 with the HP-detrended participation rate and other procyclical indicators (Table 1) seem to suggest a procyclical fluctuation. This discrepancy is theoretically interesting but does not have much practical importance: for instance, in 1996 in Japan, mortality at ages 20–44 was about six times smaller than that at ages 45–65 and about 32 times smaller than at ages 65–84.

The discrepancies between correlation models with variables HP-detrended and regression models with variables in year-to-year change seem to be due to the fact that the transformation of a time series into first differences filters in the short-term high-frequency

Table 5. Proportion of All Deaths Attributed to Specific Causes of Death: Japan, 1990

Cause of Death	Relation With Economic Fluctuations	Percentage
Heart Disease (excluding hypertensive disease)	Procyclical	20.6
Pneumonia	Procyclical	9.8
All Accidents (transportation accidents)	Procyclical	5.6
Liver Disease	Procyclical	3.4
Senility	Procyclical	2.0
All procyclical causes		41.4
Suicide	Countercyclical	2.4
Diabetes Mellitus	Countercyclical	1.2
Hypertensive Diseases	Countercyclical	0.9
All countercyclical causes		4.5
Malignant Neoplasms	Undefined or countercyclical	32.9
Cerebrovascular Disease (stroke)	Undefined	14.5
Chronic Bronchitis and Emphysema	Undefined	1.5
Asthma	Undefined	0.9
Gastric and Duodenal Ulcer	Undefined	0.5
Tuberculosis	Undefined	0.7
Renal Failure	Undefined	1.9
All other major causes of death		20.0

components of the fluctuation at the expense of filtering out the lower-frequency components that are typical of so-called business cycles (Baxter and King 1999). In this investigation, when correlations of age-specific or cause-specific mortality rates and the eight economic indicators with variables in first differences or rate of change were tested, the results (not shown) were generally much more frequently erratic or weaker (Figure 3, first and third rows for heart disease) than those with HP-filtered series (as discussed earlier; Tables 1 and 2). These results are not reported here, but the pattern of correlation signs obtained was also very suggestive of a procyclical oscillation of general mortality, sex-specific mortality, age-specific mortality at different ages, and a number of cause-specific mortality rates except suicide, which also appeared to be fluctuating countercyclically. Suicide seems to be also a special case in that the strength of the association between its variations and the macro-economic changes is basically identical at the low frequency of the HP-detrended series and at the high frequency of series in first differences (Figure 3, second and fourth rows).

To my knowledge, the countercyclical oscillation of diabetes mortality and the pro-cyclical oscillation of mortality attributed to senility have never been previously reported. Although neither of these two cause-specific mortality rates shows a strong association with the fluctuations of the economy, both are processes that can last years, and sometimes decades, until death. As mentioned in the introduction, a business-cycle-associated oscillation of this type of death may be just a manifestation of how chronic processes can be aggravated and accelerated into death by contemporary events. If the countercyclical oscillation of diabetes mortality suggested by these Japanese results is confirmed in other studies, this finding may throw some light on the pathways contributing to the worsening

and death of diabetic patients. Pandemic obesity seems to be causing higher incidence and prevalence of diabetes almost worldwide; therefore, theoretical and practical knowledge to deal with this disease is increasingly demanded.

The dependency of frail, elderly people on the economically active generation for personal care and attention might contribute to the procyclical oscillation of deaths attributed to senility or other chronic processes common in the aged. If so, the economic boom would increase the risk of death of the overworked adults, and indirectly that of those no longer working yet requiring the attention of younger people.

Studies using more detailed panel data from the Japanese geographical regions may be needed to confirm these results and to examine the pathways involved for each cause of death. Within the limitations of national data that are only a few decades long, the present investigation shows that in Japan, as in other industrial nations, death rates tend to fluctuate with the aggregate economy, increasing during expansions and decreasing during economic downturns, over and above the secular trend.

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