# Macroeconomic fluctuations and mortality in postwar Japan[1]

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**Abstract** — Recent research has shown that, once the long-term declining trends are excluded, mortality rates in industrial countries tend to rise in economic expansions and fall in recessions. In the present work the comovements 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 due to trends, series were detrended either with the Hodrick-Prescott filter or through differencing. As previously found in other industrial economies, in Japan general mortality and age-specific death rates tend to increase in expansions and drop in recessions, both for males and females. The effect is slightly stronger for males, and 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, and deaths attributed to diabetes and hypertensive disease, making up about 4% of total mortality, 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.

## INTRODUCTION

In recent years a number of publications have shown that mortality rates, once long-term declining trends are excluded, tend to oscillate with the so-called *business cycles* or *trade cycles*, increasing during expansions of the economy and dropping during recessions, fluctuating procyclically. Though 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), there are reports suggesting that this parallel fluctuation of death rates and the economy can also be observed in recent decades in some medium-income 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). A possible

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[1] Besides minor editorial changes, this is a slightly expanded version of the paper published in *Demography,* Vol. 45, No. 2, pp. 323-343, May 2008. The section of supplementary tables and figures on pages 29-34 of the present paper includes tables and figures (all with the prefix "S") that either were suppressed in the *Demography* version, or are expanded versions of the tables in the body of the paper (for instance, table S-2 is an expanded version of table 1). This paper benefited from comments of two anonymous reviewers. Chiho Kabeya and Mieko Yoshihama kindly helped with linguistic issues. Their valuable help is here acknowledged.

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exception is Sweden, where an effect in the opposite direction was recently reported (Gerdtham and Johannesson 2005).

That mortality fluctuated with the economy, rising in expansions and falling in recessions, was already found by early 20th century researchers such as William Ogburn and Dorothy Tomas (Ogburn and Thomas 1922; Thomas 1927). From the 1970s onward, that finding was replicated by Joseph Eyer (Eyer 1977a) and other researchers (Higgs 1979; Graham, Chang, and Evans 1992). Since these findings show a tendency of mortality to rise in periods of economic expansion, they call into question the ideas of authors who have argued that economic expansions have beneficial effects on health and recessions have harmful effects (Brenner 1971, 1973, 1977, 1979a, 1979b, 1983, 1995; Catalano 1983, 1997), and they have raised controversy (Brenner 2005; Catalano and Bellows 2005; Edwards 2005; McKee and Suhrcke 2005; Neumayer 2005; Ruhm 2005a; Tapia Granados 2005c).

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

Though 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 are more frequent 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 intake, and atmospheric pollution (figure 1) might similarly contribute to trigger these deaths. Ruhm (2003, 2004, 2005a) has 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).

Though Japan was included in a panel study of 23 industrialized countries belonging to the Organization for Economic Cooperation and Development (OECD) in which a procyclical oscillation of mortality was found (Gerdtham and Ruhm 2002 and 2006; Johansson 2004), to date there has been no specific investigation of 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 had high and sustained rates of economic growth and very low levels of unemployment (figure 2). For these reasons it seems particularly interesting to ascertain if, as 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 during recent decades. As in other industrialized economies, during the second half of the 20th century in Japan mortality tended to deviate up from trend in periods of strong economic growth and to deviate down from trend during economic downturns.

The next three sections present the data and methods used in the study, the results of the statistical analysis for correlation and regression models, and the corresponding discussion. Appendix A discusses some theoretical issues and results when multivariate models are used, and appendix B discusses models using the share of health expenditure in GDP in explanatory models of the change in mortality.

## DATA AND METHODS

All data analyzed in this study were taken online from the Japanese Statistics Bureau (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 and a number of mortality rates, including age-and-sex specific and cause-and-sex specific death rates, were used in the analysis. Since both mortality rates and economic indicators (even unemployment) have long-term trends in this sample (figure 2), any correlation or regression model using untransformed series is strongly dependent on the trend and tell us 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 filter, or by transformation of the series into first differences ($x_t - x_{t-1}$). Correlation and regression models with variables transformed into 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 will not be reported.

The Hodrick-Prescott (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 is the series produced; in the HP filter, the larger the smoothing parameter, the smoother is the resulting trend. For annual data, a smoothing parameter $\gamma = 100$ is the usual option (Backus and Kehoe 1992), and $\gamma = 100$ was also used in this investigation.

Pearson correlation coefficients were computed as cross-correlations between series of deviations of the original data with respect to the HP trend. These correlations are used to ascertain the concomitant variation, i.e., 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), and is founded in John Stuart Mill's principle of concomitant variation, a theoretical principle in the study of causality (Mackie 1974) that has also been applied in natural time-series experiments (Glass, Wilson, and Gottman 1975).

The detrending through HP filtering or differencing takes care of trends due to changes in the demographic structure of the population and affecting particular mortality rates (crude mortality and cause-specific death rates) not adjusted for age. Since the changes in mortality due to population aging are long-term trends, the analysis of detrended series allows us 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 and, 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, the analyses were repeated for eight business cycle indicators, including two more traditional indicators—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.[3] However, to save space, only the results for GDP, GDP lagged one year, unemployment, and the labor participation ratio will be 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 will be only briefly mentioned.

As a summary index of a multiplicity of business, commercial, and government activities, GDP is considered a key economic indicator. In the analyzed period of the Japanese economy, HP-filtered GDP shows a strong negative and very significant negative correlation (– 0.73, $P < 0.001$) with the unemployment rate equally filtered. The HP-filtered labor force participation ratio, reflecting the proportion employed or looking for work among all those of working age, correlates with detrended GDP significantly at lag zero (0.32, $P = 0.04$), but its strongest correlation with GDP is when this is lagged one year (0.35, $P = 0.02$). This shows that the participation ratio is a leading indicator.[4]

Correlation models provide an indication of the strength and character (parallel or mirroring) of the association between the oscillations in two series, but from cross-correlations between HP-filtered series it is difficult to quantify the impact of macroeconomic conditions on mortality levels. Data filtered with a smoothing filter often have high positive autocorrelation and 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 were used. Ordinary least squares (OLS) were used to estimate equations of the type $\Delta m_t = \alpha + \sum_{i=0}^{k} \beta_k \cdot \Delta e_{t-i} + \varepsilon_t$, in which $\Delta m_t = m_t - m_{t-1}$ is the change in age-specific mortality from year $t$-1 to year $t$, $\alpha$ is a constant, and $\Delta e_{t-i}$ is the change in an economic indicator, from lag zero to lag $k$.

[3] Unemployment is countercyclical; the seven other indicators are all procyclical in this sample.

[4] Showing how difficult is to put together the scheme of business cycles provided by different indicators, when these three indicators are converted into rate of change, the participation ratio correlates only – 0.01 with GDP and – 0.12 with unemployment. When both are in rate of change, unemployment correlates – 0.58 ($P < 0.001$) with GDP.

The observations for most analyzed series cover about half a 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 if earlier macroeconomic fluctuations had a different impact on mortality than more recent ones, regressions were computed splitting the available time series samples in half. To formally test the possibility of structural change, the Chow test (Green 1993) was used.

## 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 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 participation ratio. In other studies (Neumayer 2004; Ruhm 2000; Tapia Granados 2005a, 2005b) it has been seen that the impact 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.

Judging by the correlations of male and female mortality with economic indicators, the macroeconomic fluctuations seem to have a stronger effect on male mortality. At least that is what is suggested by the correlations of business cycle indicators with sex-specific crude mortality and sex-specific mortality at ages 20–44 and 45–64 (table 1).

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 in a few cases which will be discussed below. Deaths attributed to heart disease,[5] accidents, liver disease, and pneumonia show significant negative correlations with unemployment, and positive correlations with the procyclical indicators, GDP and the participation ratio, i.e., 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 (– 0.61, both for male and female mortality), as well as positive correlations with GDP (0.35 and 0.37, respectively, for male and female mortality), and other procyclical

[5] In the Japanese statistics this category of heart disease mortality excludes hypertensive disease mortality.

indicators. Transportation accidents[6] (2.9% of all deaths in 1990) are intensely procyclical for both males and females, as shown by almost every economic indictor, while deaths due to "all accidents" (5.6% of all deaths) are much more intensely procyclical in males. Since this category includes injuries in the work-place, 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 pneumonia and liver disease mortality. However, mortality attributed to senility[7] appears only very weakly procyclical, with marginally significant negative correlations with unemployment and positive but indistinguishable from zero correlations with all other procyclical indicators.

Mortality due to suicide, diabetes, 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 suicide-unemployment much higher for males (0.66) than for females (0.40). 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 mellitus seems to be countercyclical, increasing in periods of high unemployment. Similarly, mortality attributed to hypertensive disease seems to increase slightly in recessions, though the only significant correlations are with GDP and all other correlations are only marginally significant or not significant.

Mortality attributed to gastric and duodenal ulcer, as well as renal failure shows some significant correlations with economic indicators, but the signs of the correlations are inconsistent and it is very difficult to ascertain a procyclical or countercyclical pattern. Cancer mortality is an even more difficult case, since 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 they are inconsistent with positive correlations with the 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 (the corresponding results were therefore excluded from table 3).

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[6] This is the terminology used in the Japanese Statistics Bureau website. Avoiding the term "accidents" has been repeatedly recommended in the public health field (Loimer and Guarnieri 1996), but regrettably the recommendation is often ignored.

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

**Regression models**

Models were computed in which a mortality rate is regressed on a constant and 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. Though 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, using different economic indicators and mortality rates, no lagged effects beyond lag one were found, and therefore there is no evidence of a delayed impact of economic change on mortality; the effect is rather coincidental, not lagged. Therefore, only the results of models with unemployment as the explanatory variable and without lagged terms will be 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, as well, on mortality attributed to heart disease, transport injuries, and other causes of death (table 4). For instance, for mortality 65–84 the estimated equation (standard errors in parentheses) is

$$\Delta m = \underset{(0.24)}{-0.84} \; \underset{(1.06)}{-2.61} \cdot \Delta u$$

so that the model predicts a drop in mortality of 0.84 deaths per 1,000 population (this is a mortality reduction of 2.7%, assuming the mortality level observed in 1990) when unemployment does not change ($\Delta u = 0$). Since 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 $\Delta u = -0.84/2.61 = -0.32$, that is to say, the model predicts that mortality 65–84 will not decrease whenever the unemployment rate diminishes by a 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), while the corresponding effects in age-specific mortality 45–64 and 65–84 in the years 1978–1996 are much weaker and not 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 for 2 degrees of freedom (d.f.) for the numerator, and 33 d.f. for the denominator, $P < 0.001$. Therefore, there is significant evidence of a structural change in the impact of business cycles on mortality 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, though the test result is only marginally significant ($P = 0.070$).

Consistent with the results of correlation models (table 1), regression models (table 3) show procyclical oscillations of mortality 45–64 and 65–84, though the negative effect of unemployment, i.e., the procyclical oscillation of mortality, is stronger in the earlier years. But is this 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 attributed to heart disease, suicide, and transportation injuries (note the high $R^2$ values), though the unemployment effect is negative for heart disease and transportation injury deaths, and positive for suicide. For instance, 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 it 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 reveal a procyclical oscillation of deaths due to heart disease and transportation injuries, and 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 due 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 ($R^2 \leq 0.04$). However, in the sample 1960–1977 the negative effect of unemployment is massive for both male and female mortality, and then, in the sample 1978–1998 the effect turns positive, though not significant. Plots of stroke mortality vs. unemployment (figure 4) do not reveal clear outliers in 1960–1977, but there are major outliers 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 in males, and from 102 to 121 in females. A further exploration of this issue goes 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, using either unemployment or GDP as the explanatory variable, the economy reveals a statistically significant effect on cancer mortality at lag one, negative for GDP and positive for unemployment, i.e., in a

countercyclical direction. Mortality due to cancer might therefore be countercyclical, though this effect only appears in regression models when "the economy" is lagged one year.

## DISCUSSION

The results presented here are similar to those obtained in studies from other countries (Neumayer 2004; Ruhm 2000, 2003, 2004, 2005a, 2005b; Tapia Granados 2005a, 2005b) in which most major causes of death have been found to be procyclical while suicides have been found to be generally countercyclical. While in former studies a procyclical fluctuation has been found in mortality caused by cardiovascular disease in general or acute ischaemic disease in particular (Ruhm 2005b), in this study heart disease mortality was procyclical while 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 above 2 (tables 3 and 4). Since $d = 2 \cdot (1 - \hat{a})$, where $\hat{a}$ is the estimated autocorrelation of the residuals, $d$ values above 2.0 indicate that the autocorrelation is negative and, therefore, the estimate for the standard error is expected to be biased, too large, which will bias the $P$-values down. 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. But these are only a few cases, and since 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.

Though 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, that, as we see, are acyclical or even countercyclical, were about twice the number of deaths attributed to heart disease, when in 1960 cancer deaths exceeded heart disease deaths by just a third. This and the increase in the countercyclical suicide share of total deaths seem to explain the fact that 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), while mortality rates clearly fluctuating countercyclically constitute only about 4% of all deaths. Mortality due 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, since procyclical causes of death have a larger weight

than countercylical deaths in total mortality. That 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 transport injuries, explains that mortality at ages 65–84 appears much less correlated with the economy than mortality at ages 45–64 (table 1), 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 though marginally significant positive effect of unemployment on mortality, i.e., a countercyclical fluctuation of this age-specific death rate. However, the correlations of HP-detrended mortality 20–44 with the HP-detrended participation rate and other procyclical indicators (table 1) seem to suggest a procyclical fluctuation. This discrepancy, though theoretically interesting, has not much practical importance because in recent years in Japan mortality at ages 20–44 is 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 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). When in this investigation 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). Though these results are not reported here, 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, that 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 macroeconomic 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 our knowledge, the countercyclical oscillation of diabetes mortality and the procyclical oscillation of mortality attributed to senility have never been previously reported. Though 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, 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, and 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 but requiring the attention of the younger ones.

More detailed studies using 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 only a few decades long, the present investigation has shown 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.

## APPENDIX A

## MULTIVARIATE ANALYSES OF THE IMPACT OF BUSINESS CYCLES ON MORTALITY

From the very early studies by Dorothy Thomas and William Ogburn in the 1920s to the most recent studies by Ruhm (2000, 2005a, 2005b) and many others (Ortega Osona et al. 1997; Abdala et al. 2000; Chay and Greenstone 2003; Dehejia and Lleras-Muney 2004; Laporte 2004; Neumayer 2005; Tapia Granados 2005a, 2005b), researchers have usually represented the dynamics of the economy in each specific statistical model by one particular economic indicator. Though unemployment has been the most commonly used, many other indicators have been tested; for instance, Thomas (1927) used indicators of investment in fixed capital, Ruhm (2000) used employment indicators, and Tapia Granados (2005a) used indicators of industrial activity such as average weekly hours in manufacturing and the index of industrial production. Money-based indicators such as GDP or state or provincial gross product have been also used (Ruhm 2000, Tapia Granados 2005a, 2000b), often providing weaker evidence of an impact of business fluctuations on mortality than that provided by models based in non-monetary business cycle indicators. To our knowledge, the only researcher who often used simultaneously several economic indicators with other covariates in models to analyze the impact of macroeconomic change on mortality has been Harvey Brenner (1983, 2005), who set complicated multivariate regressions that generated puzzlement and skepticism for many years (Eyer 1976, 1984; Kasl 1979, Winter 1983, Søgaard 1992) and have been more openly criticized recently (Ruhm 2004, Tapia Granados 2005c).

Regressions or correlations showing a statistical connection between a population variable and an economic indicator are just ecological models revealing ecological effects that can be different from individual effects (Diez Roux 2000). For instance, at the population level, years of high unemployment can associate with low mortality and years of low unemployment with high mortality, as has been shown in this and other investigations, while at the individual level unemployment status can be associated with a higher risk of death, compared with those working. Individual-level and population-level analyses focus on distinct questions. Individual-level studies assess the effects of individual-level unemployment on the health of those unemployed; in contrast, the regression effect of the national unemployment rate (or its change) on mortality (or its change) investigates the population-health impact of economic activity, with population-level unemployment serving as a proxy for the dynamic status of the economy. A harmful effect of unemployment on health at the individual-level is perfectly compatible with a beneficial health effect of unemployment at the population level (Eyer 1977b; Tapia Granados 2005c) . At a particular point in time, the unemployed are a minority compared with the large majority of employed plus those excluded from the labor market (because of retirement, housework, etc.). If accelerated economic activity, as indexed by lower unemployment, has a detrimental effect on everyone in the population, then the population-level association will reflect better health in times of high

unemployment even if, at the individual level, being unemployed is itself detrimental to health.

In economic expansions workers work more hours, profits and wages generally increase, bank reserve ratios and business failures decrease, more sulfuric acid and electric power is consumed, and people sleep and exercise less and smoke more (Mitchell 1951, Ruhm 2003). Since all these effects are highly correlated, the attempt of disentangling the "individual effects" of these variables on, say, mortality, by putting several of them as explanatory variables in the same regression will usually render poor results, because of collinearity. This will be particularly true when working with national time series of annual data, in which the sample size rarely reaches 100 and is often below 50. The combination of a small sample and explanatory variables substantially correlated will frequently produce statistical results with very low credibility. A basic principle in multiple regression is that the ratio sample size/parameters in the model be as large as possible—for instance, Cohen and Cohen (1983) recommend that this ratio be at least 10 or even 20. With, say, $n = 40$, it is difficult to go much further than including an intersect and an explanatory variable in the model.

To illustrate how little is gained by complicating the model, the results are presented herein of several models in which the outcome variable is mortality at ages 65–84 in first differences. They were all estimated with OLS using the available data, $n$ is between 37 and 39, and the Durbin-Watson $d$ is greater than 2.63 and smaller than 2.73 in each model. $U$ is unemployment, $\Delta U$ is the change in unemployment, and $g$ is economic growth (percentage change of real GDP). Standard errors are below the parameter estimates, and coefficients that are statistically significant at a 95% significance level are highlighted in boldface:

$\Delta m_{65\text{-}84} = -\mathbf{0.84} - \mathbf{2.6}\,\Delta U$ (0.24) (1.1) $R^2 = 0.15$ [1]

$\Delta m_{65\text{-}84} = -0.97 + 2.6\,g$ (0.66) (9.6) $R^2 = 0.00$ [2]

$\Delta m_{65\text{-}84} = -0.20 - \mathbf{3.6}\,\Delta U - 10.6\,g$ (0.51) (1.3) (7.6) $R^2 = 0.15$ [3]

$\Delta m_{65\text{-}84} = -0.93 + 0.02\,U$ (0.82) (0.39) $R^2 = 0.00$ [4]

$\Delta m_{65\text{-}84} = -1.5 + 0.17\,U + 4.2\,g$ (1.4) (0.50) (8.6) $R^2 = 0.01$ [5]

$\Delta m_{65\text{-}84} = -1.4 - \mathbf{3.0}\,\Delta U + 0.000002\,\text{GDP}$ (1.4) (1.1) $R^2 = 0.18$ [6]

$\Delta m_{65\text{-}84} = -6.5 - \mathbf{3.1}\,\Delta U + 0.46\,\ln\text{GDP}$ (5.8) (1.2) (0.47) $R^2 = 0.17$ [7]

It is clear from these results how little is gained by complicating the model with colinear covariates or with covariates that are not colinear (*U* and GDP are not collinear, though Δ*U* is strongly colinear with *g*) but have trends (as is the case with both *U* and GDP in these sample). Model [1] is clearly the most parsimonious, and by including other variables there is almost no gain in $R^2$. In a further attempt to capture different aspects of economic conditions, the following model includes unemployment *U* in levels to index the depth of recessions, GDP growth *g* for speed of economic change, and income *i,* which is GDP per capita, for the standard of living:

$\Delta m_{65\text{-}84} = -1.5 - 0.12\,U + 5.0\,g + 69.8\,i$ (5.8) (0.63) (10.3) (498.1) $R^2 = 0.01$ [8]

Nothing is statistically significant and the explanatory power of the model is 1% of the total variance of mortality. A possible interpretation of these results is that, when properly adjusted for the different components of the macroeconomic change, it is not possible to observe any significant impact of macro variables on mortality. Indeed, this was concluded by Goff (1980) from the results of a similar model applied to United States data. A more plausible conclusion, however, is that multivariate models may be useless when the issue is to estimate the effect of a factor and the available explanatory variables are just different and correlated ways to gauge that factor.

## APPENDIX B

### 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 (γ = 100), and correlated with economic indicators similarly detrended, the health share proves to be strongly countercyclical, since it correlates 0.57 with unemployment and – 0.76 with GDP (for both correlations, $P < 0.001$).

Since mortality oscillates procyclically and the health share in GDP oscillates countercyclically, it is to be expected that the health share will correlate negatively with mortality rates. At lag zero, age-adjusted or age-specific mortality in first differences or HP-detrended does not show significant correlations with the health share in GDP similarly detrended, but 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, there is a significant negative effect 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 due to heart disease (at lag one), pneumonia (at lag one), suicides (at lag two but only for females), and traffic injuries (at lag zero). No statistically significant effects are found for any lag in regressions for diabetes and cancer mortality.

These findings could be interpreted as suggesting that a higher health share in GDP reduces mortality at lag zero or lag one for particular causes, though not for others. However, the strong correlation between the oscillations of the health share in GDP and the oscillations of the economy, the fact that for mortality due to traffic injuries crashes—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 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—rather 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. 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 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.

**REFERENCES**


Abdala, F., Geldstein, R. N., and Mychaszula, S. M. 2000. "Economic Restructuring and Mortality Changes in Argentina - Is There Any Connection?" Pp. 328-350 in *The Mortality Crisis in Transitional Economies,* edited by G. A. Cornia and R. Paniccià, New York: Oxford University Press.

Anson, J. and O. Anson O. 2000. "Thank God it's Friday: the Weekly Cycle of Mortality in Israel." *Population and Research Policy Review* 19:143–54.

Backus, D. K., and P. J. Kehoe. 1992. "International Evidence on the Historical Properties of Business Cycles." *American Economic Review* 82: 864-888.

Baxter, M., and R. G. King. 1999. "Measuring Business Cycles: Approximate Band-pass Filters for Economic Time Series." *Review of Economics and Statistics* 81(4): 575-593.

Brenner, M. H. 1971. "Economic Changes and Heart Disease Mortality." *American Journal of Public Health, 61*(3): 606-611.

_____. 1973. "Fetal, Infant, and Maternal Mortality During Periods of Economic Instability." *International Journal of Health Services 3*(2): 145-159.

_____. 1977. "Health Costs and Benefits of Economic Policy." *International Journal of Health Services 7*(4): 581-623.

_____. 1979a. "Mortality and the National Economy. A Review, and the Experience of England and Wales, 1936-76." *Lancet 2*(8142): 568-573.

_____. 1979b. "Unemployment, Economic Growth, and Mortality. *Lancet 1*(8117): 672.

_____. 1983. "Mortality and Economic Instability: Detailed Analyses for Britain and Comparative Analyses for Selected Industrialized Countries." *International Journal of Health Services 13*(4): 563-620.

_____. 1995 "Political Economy and Health." In *Society and Health,* edited by B. C. Amick III, S. Levine, A. R. Tarlov and D. C. Walsh. New York: Oxford University Press.

_____. 2005. "Commentary: Economic Growth is the Basis of Mortality Rate Decline in the 20th Century — Experience of the United States 1901–2000." *International Journal of Epidemiology 34:* 1214-1221.

Catalano, R. 1983. "Health Effects of Economic Instability: A Test of Economic Stress Hypothesis." *Journal of Health and Social Behavior, 24*(1): 46-60.

_____. 1997. "The Effect of Deviations from Trends in National Income on Mortality: The Danish and USA Data Revisited." *European Journal of Epidemiology 13*(7): 737-743.

Catalano, R., and B. Bellows, B. 2005. "Commentary: If Economic Expansion Threatens Public Health, Should Epidemiologists Recommend Recession? *International Journal of Epidemiology 34:* 1212-1213.

Chay, K. Y., and M. Greenstone. 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.
Cohen, J., and P. Cohen. 1983. *Applied Multiple Regression/Correlation Analysis for the Behavioral Sciences,* 2nd ed. Hillsdale, Lawrence Erlbaum,
De Rios Neto, E., and J. A. M. Carvalho. 1997. Demographic Consequences of Structural Adjustment: The Case of Brazil. Pp. 174-198 in *Demographic responses to economic adjustment in latin america*, edited by G. Tapinos, A. Mason and J. Bravo. Oxford: Clarendon Press.
Dehejia, R., and A. Lleras-Muney. 2004. "Booms, Busts, and Babies' Health. *Quarterly Journal of Economics 119*(3): 1091-1130.
Diez Roux, A. V. 2000. "Multilevel Analysis in Public Health Research." *Annual Review of Public Health* 21:171-192.
Diggle, P. J. 1989. *Time Series: A Biostatistical Introduction*. New York: Oxford University Press.
Edwards, R. D. 2005. "Commentary: Work, Well-being, and a New Calling for Countercyclical Policy." *International Journal of Epidemiology* 34*:* 1222-1225.
Eyer, J., 1976. "Rejoinder to Dr. Brenner." *International Journal of Health Services* 6, 157–168.
_____. 1977a. "Prosperity as a Cause of Death." *International Journal of Health Services 7*(1): 125-150.
_____. 1977b. "Does Unemployment Cause Death Rate Peak in Each Business Cycle? Multifactor Model of Death Rate Change." *International Journal of Health Services 7*(4): 625-662.
_____. 1984. "Capitalism, Health, and Illness." Pp. 23-59 in *Issues in the Political Economy of Health Care,* edited by J. B. McKinlay. New York: Tavistock.
Gerdtham, U.-G, and Johannesson, M. 2005. "Business Cycles and Mortality: Results from Swedish Microdata." *Social Science and Medicine, 60*(1): 205-218.
Gerdtham, U.-G., and Ruhm, C. J. 2002. "Deaths Rise in Good Economic Times: Evidence from the OECD." *NBER Working Paper, 9357*
Gerdtham, U.-G., and Ruhm, C. J. 2006. . "Deaths Rise in Good Economic Times: Evidence from the OECD." *Economics and Human Biology* 4: 298–316.
Glass, G. V., V. L. Willson, and J. M. Gottman. 1975. *Design and Analysis of Time-series Experiments*. Boulder: Colorado Associated University Press.
Goff, B.L. 1980. "Health and the Economy: Exogenous Versus Choice Variables." *Kyklos* 43(3):437-484.
Graham, J. D., B. Chang, and J. S. Evans. 1992. Poorer Is Riskier. *Risk Analysis 12:* 333-337.
Green, W. H. 1993. *Econometric Analysis*, 2nd ed. New York, Macmillan.
Higgs, R. 1979. "Cycles and Trends of Mortality in 18 Large American Cities, 1871-1900." *Explorations in Economic History 16:* 381-408.
Johansson, E. 2004. "A Note on the Impact of Hours Worked on Mortality in the OECD." *European Journal of Health Economics 4:* 335-340.

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* 69:784–9.

Khang, Y. H., J. W. Lynch, and G. A. Kaplan. 2005. Impact of Economic Crisis on Cause-specific Mortality in South Korea. *International Journal of Epidemiology 34*(6): 1291-1301.

Laporte, A. 2004. “Do Economic Cycles Have a Permanent Effect on Population Health? Revising the Brenner Hypothesis.” *Health Economics 13:* 767-779.

Loimer, H., and M. Guarnieri. 1996. “Accidents and Acts of God: A History of the Terms.” *American Journal of Public Health 86*(1): 101-107.

Mackie, J. L. 1974. *The Cement of the Universe: A Study of Causation*. Oxford: Clarendon Press.

McKee, M., and Suhrcke, M. 2005. “Commentary: Health and Economic Transition.” *International Journal of Epidemiology 34:* 1203-1206.

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 and Medicine 58*(6): 1037-1047 [erratum corrigendum in *Social Science and Medicine* 59(9):1993].

Neumayer. E. 2005. “Commentary: The economic business cycle and mortality.” *International Journal of Epidemiology* 34*:* 1221-1222.

Ogburn, W. F., and D. S. Thomas. 1922. “The Influence of the Business Cycle on Certain Social Conditions.” *Journal of the American Statistical Association 18:* 324-340.

Ortega Osona, J. A., and D. Reher. 1997. Short-term Economic Fluctuations and Demographic Behaviour: Some Examples from 20th-Century South america. Pp. 129-155 in *Demographic Responses to Economic Adjustment in Latin America,* edited by G. Tapinos, A. Mason and J. Bravo. Oxford: Clarendon Press.

Phillips, D. P., N. Christenfeld, and N. M. Ryan. 1999. “An Increase in the Number of Deaths in the United States in the First Week of the Month: An Association with Substance Abuse and Other Causes of Death.” *The New England Journal of Medicine 341*(2): 93-98.

Ruhm, C. J. 2000. “Are Recessions Good for Your Health? *Quarterly Journal of Economics 115*(2): 617-650.

_____ 2003. “Good Times Make You Sick.” *Journal of Health Economics 22:* 637-658.

_____ 2004. “Macroeconomic Conditions, Health, and Mortality. “Cambridge, MA: National Bureau of Economic Research, NBER Working Paper 11007.

_____ 2005a. “Commentary: Mortality Increases During Economic Upturns.” *International Journal of Epidemiology* 34: 1206-1211.

_____ 2005b. “Macroeconomic Conditions and Daths from Coronary Heart Disease.” Working Paper. Greensboro, NC: NBER and University of North Carolina.

Søgaard J. 1992. “Econometric Critique of the Economic Change Model of Mortality.” *Social Science and Medicine* 34:947–57.

Sokejima, S., and S. Kagamimori. 1998. “Working Hours as a Risk Factor for Acute Myocardial Infarction in Japan: Case-control Study.” *BMJ 317*(7161): 775-780.

Tapia Granados, J. A. 2005a. “Increasing Mortality During the Expansions of the US Economy, 1900-1996.” *International Journal of Epidemiology 34*(6): 1194-1202.

_____. 2005b. “Recessions and Mortality in Spain, 1980-1997.” *European Journal of Population 21*(4): 393-422.

Tapia Granados, J. A. 2005c. “Response: Economic Growth, Business Fluctuations and Health Progress.” *International Journal of Epidemiology* 34*:* 1226-1233.

Thomas, D. S. 1927. *Social Aspects of the Business Cycle*. New York: Knopf.

Winter, J., 1983. ‘Unemployment, Nutrition and Infant Mortality in Britain, 1920–50’, Pp. 232–256 in *The Working Class in Modern British History: Essays in Honour of Henry Pelling,* edited by J. Winter. Cambridge: Cambridge University Press.3

**Table 1. Correlations between annual series of three economic indicators and mortality rates, all detrended with the HP filter, Japan, *ca*. 1960 to recent years**

| Mortality rate | GDP | Unemployment | Labor force participation ratio | GDP lagged one year |
|---|---|---|---|---|
| Age-adjusted mort. | – 0.01 | – 0.28† | + 0.42** | + 0.18 |
| Crude mortality | – 0.03 | – 0.32* | + 0.32* | + 0.19 |
| Males | + 0.00 | – 0.42** | + 0.35* | + 0.25 |
| Females | – 0.03 | – 0.41* | + 0.23 | + 0.27† |
| Infant mortality | + 0.01 | + 0.13 | + 0.32* | + 0.23 |
| *Mortality at ages* | | | | |
| 1-14 | + 0.01 | – 0.24 | – 0.01 | + 0.13 |
| 10-19 | + 0.22 | – 0.07 | + 0.54*** | + 0.18 |
| Males | + 0.34* | – 0.12 | + 0.51*** | + 0.23 |
| Females | + 0.02 | + 0.04 | + 0.48** | + 0.06 |
| 20-64 | + 0.06 | – 0.29† | + 0.42** | + 0.28† |
| 20-44 | – 0.08 | 0.00 | + 0.47** | – 0.02 |
| Males | + 0.21 | – 0.16 | + 0.48** | + 0.28† |
| Females | + 0.00 | – 0.09 | + 0.43** | + 0.15 |
| 45-64 | – 0.02 | – 0.39* | + 0.18 | + 0.16 |
| Males | – 0.04 | – 0.37* | + 0.15 | + 0.12 |
| Females | + 0.00 | – 0.36* | + 0.25 | + 0.20 |
| 65-84 | + 0.03 | – 0.32† | + 0.26 | + 0.25 |
| Males | + 0.05 | – 0.32* | + 0.24 | + 0.23 |
| Females | + 0.01 | – 0.31† | + 0.25 | + 0.26† |

Note: Sample size $n = 40$ for correlations involving unemployment; $n = 44$ or more for the correlations with other economic indicators.

† $P < 0.1$ * $P < 0.05$ ** $P < 0.01$ *** $P < 0.001$

**Table 2. Correlations between cause-and-sex-specific mortality rates (M = males, F = females) and three indicators of the Japanese economy, all variables HP-filtered**

| Cause-and-sex-specific mortality | | GDP | Unemployment | Labor force particip. ratio | GDP lagged one year |
|---|---|---|---|---|---|
| Heart disease | M | + 0.35* | – 0.61*** | + 0.20 | + 0.48*** |
| | F | + 0.37* | – 0.61*** | + 0.13 | + 0.48*** |
| Transport accidents | M | + 0.32* | – 0.27† | + 0.44** | + 0.14 |
| | F | + 0.46** | – 0.31* | + 0.53*** | + 0.24 |
| All accidents | M | + 0.15 | – 0.03 | + 0.45** | + 0.00 |
| | F | – 0.04 | + 0.23 | + 0.22 | – 0.07 |
| Liver disease | M | + 0.44** | – 0.45** | + 0.32* | + 0.53*** |
| | F | + 0.49*** | – 0.47** | + 0.30* | + 0.45*** |
| Pneumonia | M | + 0.14 | – 0.47** | + 0.17 | + 0.28† |
| | F | + 0.04 | – 0.37* | + 0.18 | + 0.14 |
| Senility | M | + 0.06 | – 0.28† | + 0.23 | + 0.13 |
| | F | + 0.01 | – 0.30† | + 0.08 | + 0.11 |
| Suicide | M | – 0.33* | + 0.66*** | + 0.09 | – 0.16 |
| | F | + 0.02 | + 0.40* | + 0.10 | + 0.08 |
| Diabetes | M | – 0.23 | + 0.35* | – 0.01 | – 0.25† |
| | F | – 0.28† | + 0.38* | – 0.04 | – 0.36* |
| Hypertensive disease | M | – 0.36* | + 0.02 | – 0.32* | – 0.23 |
| | F | – 0.33* | – 0.01 | – 0.28† | – 0.12 |
| Cancer | M | – 0.16 | + 0.29† | + 0.17 | – 0.37* |
| | F | – 0.15 | + 0.31† | + 0.26† | – 0.42* |
| Gastric & duodenal ulcer | M | – 0.14 | – 0.20 | + 0.07 | – 0.14 |
| | F | – 0.22 | + 0.17 | + 0.35* | – 0.14 |
| Renal failure | M | – 0.03 | – 0.17 | – 0.35* | – 0.13 |
| | F | – 0.13 | – 0.01 | – 0.27 | – 0.03 |

Note: *n* as in former table.

† $P < 0.1$ * $P < 0.05$ ** $P < 0.01$ *** $P < 0.001$

**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 ages 20–44 | | | Mortality ages 45–64 | | | Mortality 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 test | $F = 13.82$, $P < 0.001$ | | | $F = 14.65$, $P < 0.001$ | | | $F = 2.89$, $P = 0.070$ | | |

*Note:* $\Delta m_t$ = change in mortality from $t$-1 to $t$; $\Delta u_t$ = change in unemployment; $\varepsilon_t$ = error term; SE ($\hat{\beta}$) = standard error of the estimated unemployment effect $\hat{\beta}$; SSE = sum of square errors.

* $P < 0.05$ † $P < 0.01$.

**Table 4. Models in which the change in cause-specific mortality is regressed on a constant and the change in unemployment**

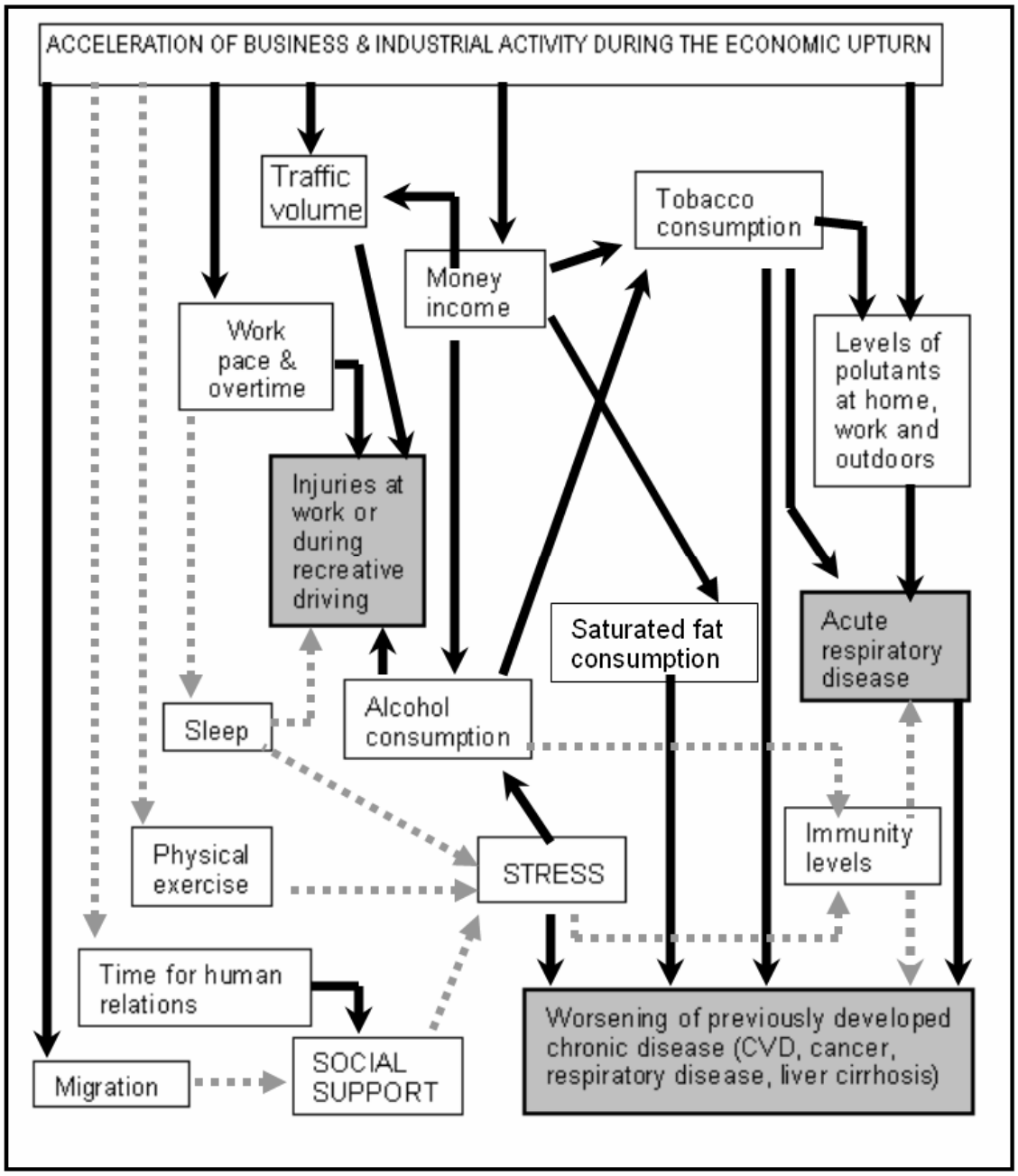
| | Cause-specific mortality | Sex | Unemployment effect (standard error) | | $R^2$ | Durbin-Watson *d* |
|---|---|---|---|---|---|---|
| Sample 1960-1998 ($n$ = 39) | Heart disease | M | – 9.99** | (3.18) | 0.21 | 1.70 |
| | | F | – 9.98** | (3.55) | 0.18 | 1.67 |
| | Cerebrovascular disease (stroke) | M | – 4.71 | (3.96) | 0.04 | 1.63 |
| | | F | – 2.79 | (3.10) | 0.02 | 1.89 |
| | Transportation accidents | M | – 2.27† | (1.14) | 0.10 | 1.62 |
| | | F | – 0.75* | (0.33) | 0.12 | 1.51 |
| | Liver disease | M | – 1.06** | (0.38) | 0.17 | 1.48 |
| | | F | – 0.51† | (0.28) | 0.08 | 2.02 |
| | Pneumonia | M | – 3.73 | (2.88) | 0.04 | 2.06 |
| | | F | – 2.26 | (2.35) | 0.02 | 2.30 |
| | Senility | M | – 0.81 | (1.01) | 0.02 | 2.04 |
| | | F | – 1.76 | (1.57) | 0.03 | 1.89 |
| | Suicide | M | + 6.29*** | (1.16) | 0.44 | 1.65 |
| | | F | + 1.73** | (0.51) | 0.23 | 1.56 |
| Sample 1960-1977 ($n$ = 18) | Heart disease | M | – 7.59† | (4.20) | 0.17 | 2.62 |
| | | F | – 6.74 | (4.04) | 0.15 | 2.41 |
| | Cerebrovascular disease (stroke) | M | – 16.57*** | (3.52) | 0.58 | 1.59 |
| | | F | – 10.35*** | (2.17) | 0.59 | 2.17 |
| | Transportation accidents | M | – 4.86† | (2.64) | 0.17 | 2.01 |
| | | F | – 1.21 | (0.76) | 0.14 | 1.55 |
| | Liver disease | M | – 0.91 | (0.52) | 0.16 | 1.46 |
| | | F | – 0.47 | (0.50) | 0.05 | 2.11 |
| | Pneumonia | M | – 4.03 | (4.86) | 0.04 | 2.60 |
| | | F | – 3.51 | (4.00) | 0.05 | 2.60 |
| | Senility | M | – 3.08 | (2.01) | 0.13 | 2.32 |
| | | F | – 5.43† | (2.99) | 0.17 | 2.20 |
| | Suicide | M | + 3.64** | (1.01) | 0.42 | 1.22 |
| | | F | + 1.44 | (0.88) | 0.14 | 1.20 |
| Sample 1978-1998 ($n$ = 21) | Heart disease | M | – 12.17* | (4.90) | 0.24 | 1.42 |
| | | F | – 12.96* | (5.71) | 0.21 | 1.56 |
| | Cerebrovascular disease (stroke) | M | + 3.77 | (6.09) | 0.02 | 2.13 |
| | | F | + 2.99 | (5.07) | 0.02 | 2.23 |
| | Transportation accidents | M | – 1.10* | (0.50) | 0.21 | 2.08 |
| | | F | – 0.61* | (0.21) | 0.30 | 2.60 |
| | Liver disease | M | – 0.79 | (0.51) | 0.11 | 1.54 |
| | | F | – 0.60 | (0.35) | 0.13 | 1.97 |
| | Pneumonia | M | – 6.38† | (3.30) | 0.16 | 2.04 |
| | | F | – 3.84 | (2.67) | 0.10 | 2.66 |
| | Senility | M | – 0.41 | (0.77) | 0.01 | 2.29 |
| | | F | – 1.07 | (1.37) | 0.03 | 2.36 |
| | Suicide | M | + 7.76*** | (1.89) | 0.47 | 1.73 |
| | | F | + 1.80* | (0.68) | 0.27 | 1.85 |

* $P < 0.05$ ** $P < 0.01$ *** $P < 0.001$ † $P < 0.1$.

**Table 5. Proportion of all deaths attributed to specific causes of death, Japan, 1990**

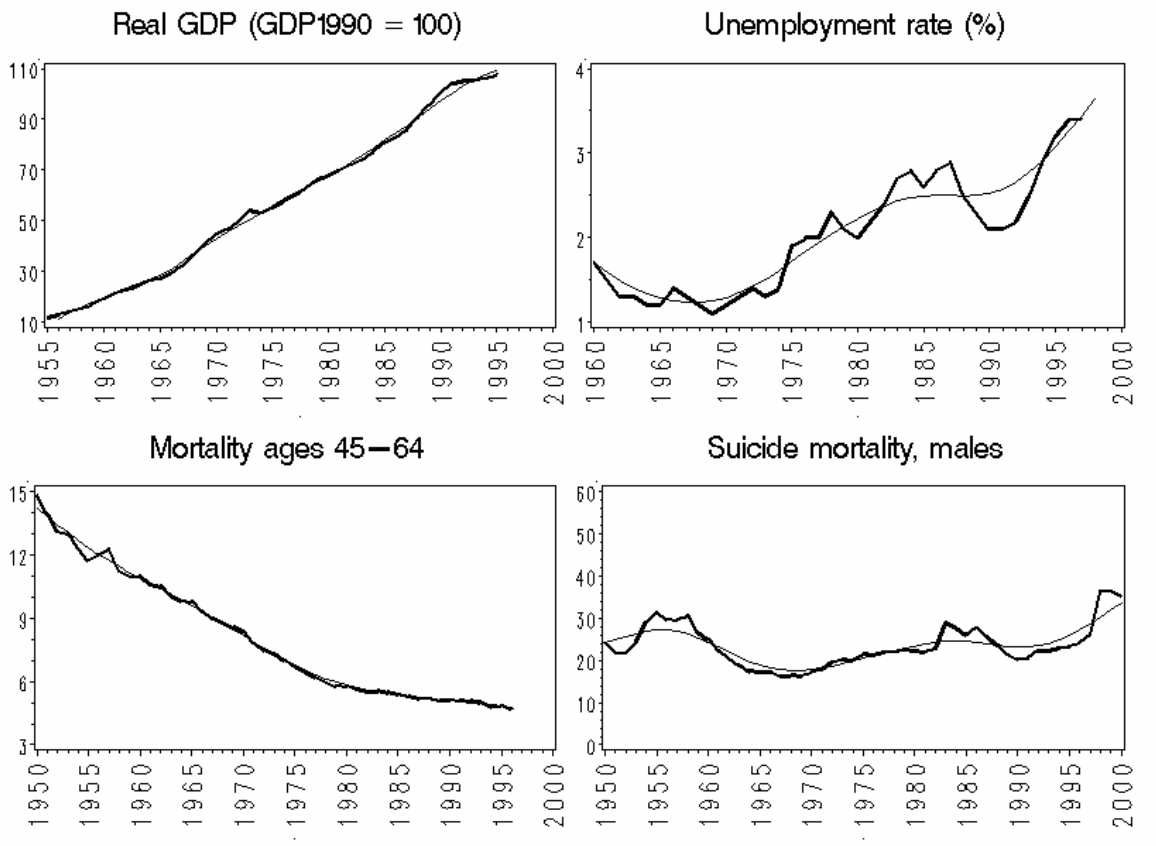
| | Relationship with economic fluctuations | % | |
|---|---|---|---|
| Heart disease (excluding hypertensive disease) | Procyclical | 20.6 | |
| Pneumonia | Procyclical | 9.8 | |
| All accidents (transport accidents) | Procyclical | 5.6 | |
| Diseases of liver | Procyclical | 3.4 | |
| Senility | Procyclical | 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* |

**Figure 1. Potential causal pathways linking economic fluctuations to mortality.**



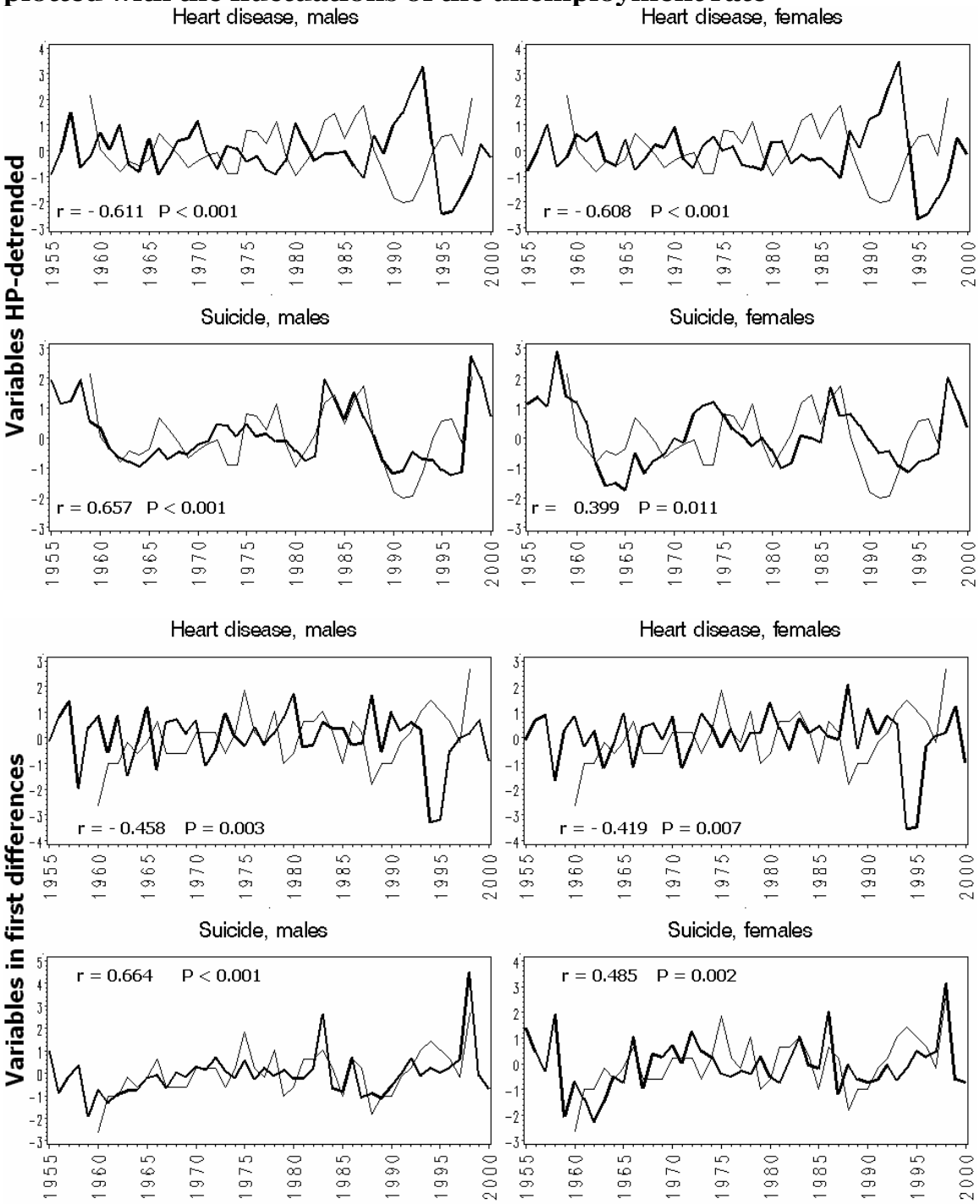

*Note:* Black solid arrows represent positive effects, gray dashed arrows, negative effects (for instance, a drop in alcohol consumption raises immunity levels and decreases the risk of injuries).

**Figure 2. Two economic indicators and two mortality rates used in the study**



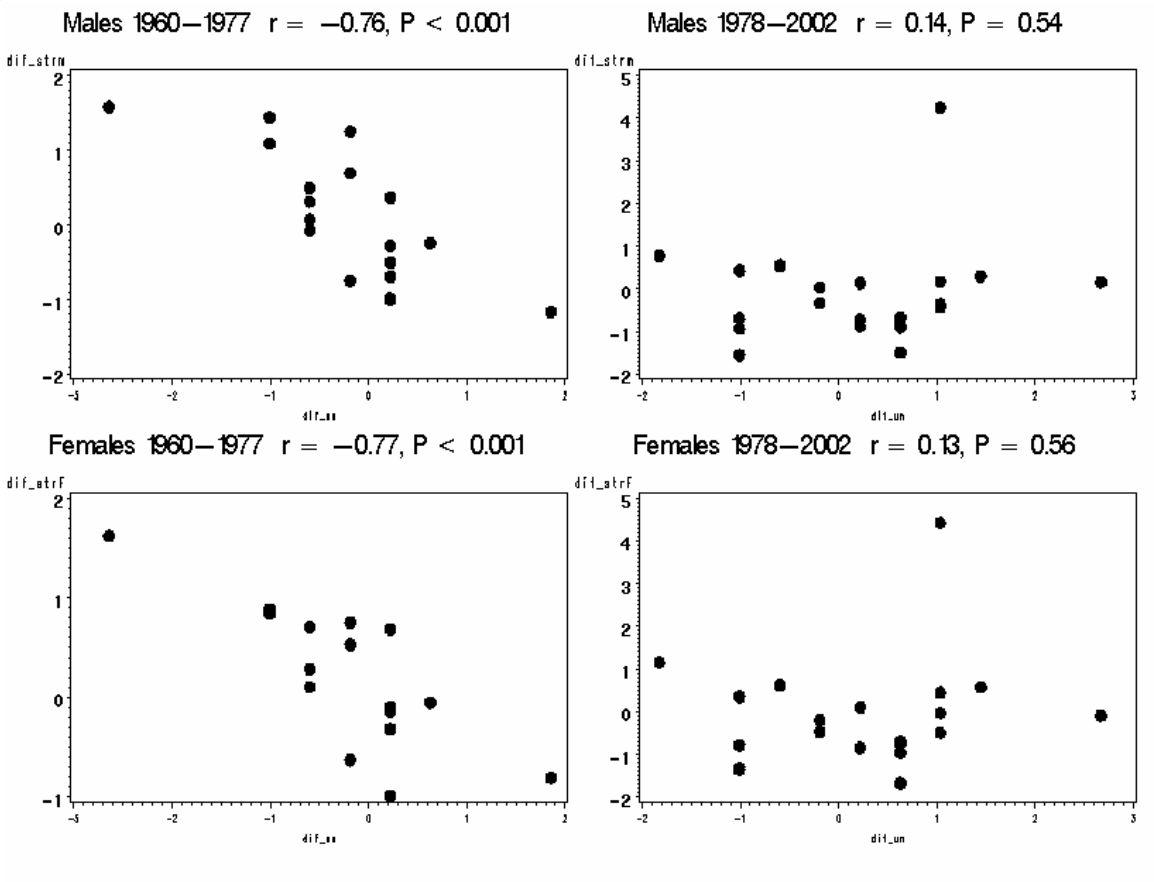

*Note*: The thin lines are Hodrick-Prescott trends, computed with a smoothing parameter γ = 100. Mortality for ages 45-64 is per 1000 population, the suicide rate is per 100,000 males.

**Figure 3. Fluctuations of four cause-and-sex-specific mortality rates plotted with the fluctuations of the unemployment rate**




*Note:* Unemployment is the thin line in all graphs. All series are normalized.

**Figure 4. Scatter plots of stroke mortality and unemployment in Japan, for males and females, and for the years 1960–1977 and 1978–1998**




Note: All variables are in first differences, and normalized.

# SUPPLEMENTARY TABLES AND FIGURES

**Table S-1.** Correlations between indicators of the Japanese economy, all detrended with the Hodrick-Prescott filter ($\gamma = 100$). Data correspond to series starting in different years, in the 1950s, up to recent years, with $n = 39$ for correlations involving unemployment, and $n = 43$ or more for the other correlations.

| | | Manufacturing indices | | | | Employment indices | |
|---|---|---|---|---|---|---|---|
| | Unemploy-ment | Average hours | Aggregate hours | Output | Employ-ment | Employment/ population. | Labor force particip. ratio |
| GDP | – 0.73*** | + 0.16 | + 0.60*** | + 0.90*** | + 0.60*** | + 0.48** | + 0.32* |
| Unemployment | | + 0.02 | – 0.56*** | – 0.71*** | – 0.65*** | – 0.43** | – 0.19 |
| Manuf. average hours | | | + 0.50*** | + 0.27 | – 0.08 | + 0.11 | + 0.12 |
| Manuf. aggreg. hours | | | | + 0.71*** | + 0.81*** | + 0.52*** | + 0.40** |
| Manuf. output | | | | | + 0.66*** | + 0.45** | + 0.28† |
| Manuf. employment | | | | | | + 0.56*** | + 0.41** |
| Employm./population | | | | | | | + 0.95*** |

† $P < 0.1$ * $P < 0.05$ ** $P < 0.01$ *** $P < 0.001$

**Table S-2.** Correlations between eight indicators of the Japanese economy and death rates, all variables detrended with the Hodrick-Prescott filter ($\gamma = 100$).Computations with $n = 40$ for correlations involving unemployment, and $n = 44$ or more for the correlations with other economic indicators.

| | | | Manufacturing indices | | | | Employment indices | |
|---|---|---|---|---|---|---|---|---|
| | GDP | Unemploy-ment | Average hours | Aggregate hours | Output | Employ-ment | Employm./ population | Labor force participation ratio |
| Age-adjusted mort. | – 0.01 | – 0.28† | + 0.17 | + 0.27† | + 0.10 | + 0.24 | + 0.43** | + 0.42** |
| Crude mortality | – 0.03 | – 0.32* | + 0.14 | + 0.26† | + 0.09 | + 0.25 | + 0.36* | + 0.32* |
| Crude mort. males | + 0.00 | – 0.42** | + 0.11 | + 0.34* | + 0.13 | + 0.35* | + 0.43** | + 0.35* |
| Crude mort. females | – 0.03 | – 0.41* | + 0.07 | + 0.25 | + 0.12 | + 0.28† | + 0.32* | + 0.23 |
| Infant mortality | + 0.01 | + 0.13 | + 0.16 | + 0.06 | + 0.07 | + 0.04 | + 0.23 | + 0.32* |
| Ages 1-14 | + 0.01 | – 0.24 | + 0.11 | + 0.22 | + 0.14 | + 0.21 | + 0.06 | – 0.01 |
| Ages 10-19 | + 0.22 | – 0.07 | + 0.41** | + 0.49** | + 0.15 | + 0.33* | + 0.49*** | + 0.54*** |
| Ages 20-64 | + 0.06 | – 0.29† | + 0.20 | + 0.42** | + 0.11 | + 0.39** | + 0.45** | + 0.42** |
| Ages 20-44 males | + 0.21 | – 0.16 | + 0.23 | + 0.57*** | + 0.12 | + 0.54*** | + 0.48** | + 0.48** |
| Ages 20-44 females | + 0.00 | – 0.09 | + 0.23 | + 0.36* | + 0.07 | + 0.30† | + 0.40* | + 0.43** |
| Ages 45-64 males | – 0.04 | – 0.37* | + 0.21 | + 0.39* | + 0.10 | + 0.32* | + 0.23 | + 0.15 |
| Ages 45-64 females | + 0.00 | – 0.36* | + 0.14 | + 0.33* | + 0.14 | + 0.31* | + 0.33† | + 0.25 |
| Ages 65-84 | + 0.03 | – 0.32† | + 0.06 | + 0.17 | + 0.09 | + 0.19 | + 0.31† | + 0.26 |

† $P < 0.1$ * $P < 0.05$ ** $P < 0.01$ *** $P < 0.001$

**Table S-3.** Correlations between sex-specific mortality rates (M = males, F = Females) and eight indicators of the Japanese economy in the years 1950-2002. All variables are HP-filtered series of annual data. The correlations are all of them based in samples including between 44 and 53 observations, except those involving unemployment that are often based in 40 observations only. All the correlations between mortality and the eight economic indicators were statistically zero for mortality caused by stroke, chronic bronchitis and emphysema, asthma, and tuberculosis—which therefore have not been included in this table

| | | | | Manufacturing indices | | | | Employment indices | |
|---|---|---|---|---|---|---|---|---|---|
| Cause-and-sex-specific mortality | | GDP | Unemploy-ment | Average hours | Aggregate hours | Output | Employ-ment | Employm./ population | Labor force particip. ratio |
| Heart disease | M | + 0.35* | – 0.61*** | – 0.04 | + 0.37** | + 0.22 | + 0.49*** | + 0.28† | + 0.20 |
| | F | + 0.37* | – 0.61*** | – 0.11 | + 0.33* | + 0.22 | + 0.49*** | + 0.22 | + 0.13 |
| Transport accidents | M | + 0.32* | – 0.27† | + 0.41** | + 0.62*** | + 0.38** | + 0.45** | + 0.48*** | + 0.44** |
| | F | + 0.46** | – 0.31* | + 0.37* | + 0.65*** | + 0.46*** | + 0.50*** | + 0.56*** | + 0.53*** |
| All accidents | M | + 0.15 | – 0.03 | + 0.33* | + 0.42* | + 0.26 | + 0.28† | + 0.43* | + 0.45** |
| | F | – 0.04 | + 0.23 | + 0.14 | + 0.05 | + 0.08 | – 0.01 | + 0.15 | + 0.22 |
| Liver disease | M | + 0.44** | – 0.45** | – 0.15 | + 0.28† | + 0.21 | + 0.47*** | + 0.36* | + 0.32* |
| | F | + 0.49*** | – 0.47** | + 0.00 | + 0.45** | + 0.26† | + 0.54*** | + 0.37* | + 0.30* |
| Pneumonia | M | + 0.14 | – 0.47** | – 0.18 | + 0.14 | + 0.10 | + 0.32* | + 0.21 | + 0.17 |
| | F | + 0.04 | – 0.37* | – 0.17 | + 0.07 | + 0.07 | + 0.23 | + 0.19 | + 0.18 |
| Senility | M | + 0.06 | – 0.28† | + 0.06 | + 0.09 | + 0.01 | + 0.07 | + 0.27 | + 0.23 |
| | F | + 0.01 | – 0.30† | + 0.03 | + 0.07 | – 0.02 | + 0.07 | + 0.14 | + 0.08 |
| Suicide | M | – 0.33* | + 0.66*** | – 0.14 | – 0.21 | – 0.31* | – 0.16 | – 0.12 | + 0.09 |
| | F | + 0.02 | + 0.40* | + 0.06 | – 0.01 | + 0.00 | – 0.02 | – 0.04 | + 0.10 |
| Diabetes | M | – 0.23 | + 0.35* | – 0.08 | – 0.15 | – 0.09 | – 0.13 | – 0.05 | – 0.01 |
| | F | – 0.28† | + 0.38* | – 0.10 | – 0.21 | – 0.19 | – 0.19 | – 0.09 | – 0.04 |
| Hipertensive disease | M | – 0.36* | + 0.02 | – 0.20 | – 0.28† | – 0.18 | – 0.18 | – 0.29† | – 0.32* |
| | F | – 0.33* | – 0.01 | – 0.29† | – 0.26† | – 0.14 | – 0.09 | – 0.24 | – 0.28† |
| Cancer | M | – 0.16 | + 0.29† | + 0.30* | + 0.02 | + 0.07 | – 0.18 | + 0.13 | + 0.17 |
| | F | – 0.15 | + 0.31† | + 0.42** | + 0.04 | + 0.03 | – 0.24 | + 0.17 | + 0.26† |
| Gastric & duodenal ulcer | M | – 0.14 | – 0.20 | + 0.10 | + 0.16 | + 0.04 | + 0.13 | + 0.12 | + 0.07 |
| | F | – 0.22 | + 0.17 | + 0.15 | + 0.19 | + 0.02 | + 0.14 | + 0.26† | + 0.35* |
| Renal failure | M | – 0.03 | – 0.17 | – 0.15 | – 0.21 | + 0.03 | – 0.14 | – 0.28† | – 0.35* |
| | F | – 0.13 | – 0.01 | + 0.01 | – 0.14 | – 0.03 | – 0.15 | – 0.23 | – 0.27 |

† $P < 0.1$ * $P < 0.05$ ** $P < 0.01$ *** $P < 0.001$

**Table S-4**. Results of regression models in which the annual rate of change of an age-specific mortality rate is regressed on an intercept and the rate of change in unemployment ($\Delta \ln m_t = \alpha + \beta \Delta \ln u_t + \varepsilon_t$)

| Age-specific mortality | Intercept | Unemployment | $R^2$ | $d$ |
|---|---|---|---|---|
| 20–44 | – 0.032*** | 0.025 | 0.02 | 1.62[a] |
| | (0.004) | (0.031) | | |
| 45–64 | – 0.022*** | – 0.064* | 0.15 | 1.79[a] |
| | (0.003) | (0.026) | | |
| 65–84 | – 0.019*** | – 0.122** | 0.21 | 2.79[b] |
| | (0.005) | (0.040) | | |

* $P < 0.05$ ** $P < 0.01$ *** $P < 0.001$.

[a] Durbin-Watson $d$ compatible with absence of residual autocorrelation, either positive or negative.

[b] This $d$ implies a $P = 0.007$ against the null hypothesis of absence of negative autocorrelation of the regression residuals, so it has to be assumed that the standard error of the unemployment coefficient estimate is too large (autocorrelation of the residuals enlarge or shrinks standard errors depending of being respectively negative or positive).

**Table S-5.** The 38 years of the period 1959-1997 divided by tertiles according to the rate of change of age-adjusted mortality

| | Years | Mean change in age-adjusted mortality (%) | Mean change in the unemployment rate (%) | Mean unemployment rate (%) | Mean real GDP growth (%) |
|---|---|---|---|---|---|
| Lowest third | 12 | – 5.4 | + 4.9 | 2.1 | 5.7 |
| Medium third | 13 | – 3.0 | + 2.9 | 2.0 | 5.8 |
| Highest third | 13 | – 0.3 | – 2.4 | 1.9 | 5.9 |

**Figure S-1**. Three indicators of the Japanese economy in the last decades of the 20th century. All variables are detrended with the Hodrick-Prescott filter, and then normalized. The oscillations of average hours (solid black) tend to lead two or even three years those of GDP (gray), and these in turn tend to lead one year those of inverted unemployment (dotted)