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The Influence of the Business Cycle on Mortality

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Abstract

We analyze the impact of short-run economic fluctuations on age-specific mortality using Bayesian time series econometrics and contribute to the debate on the procyclicality of mortality. For the first time, we examine the differing consequences of economic changes for all individual age classes. We employ a recently developed model to set up structural VARs of a latent mortality variable and of unemployment and GDP growth as main business cycle indicators. We find that young adults noticeably differ from the rest of the population. They exhibit increased mortality in a recession, whereas most of the other age classes between childhood and old age react with lower mortality to increased unemployment or decreased GDP growth. In order to avoid that opposed effects may cancel each other, our findings suggest to differentiate closely between particular age classes, especially in the age range of young adults. The results for the U.S. in the period 1956–2004 are confirmed by an international comparison with France and Japan. Long-term changes in the relationship between macroeconomic conditions and mortality are investigated with data since 1933.

JEL classification codes: C11, C32, E32, I10, J10

Keywords: Age-specific Mortality, Business Cycle, Unemployment,
Bayesian Econometrics, Health, Epidemiology

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1 Introduction

Most people are happy and willing to work hard, when they find a new job during an economic upturn after a phase of economic hardship. Commentators emphasize the improved situation and express the hope for a long duration. In the face of the common satisfaction, is it possible that there is also a hidden dark side of the boom with an increased risk of death? If such a relationship between mortality and the business cycle exists, it potentially affects the entire population, not only the working. Everyone might be subject to unexpected severe implications of economic fluctuations without even knowing about.

The principle concern for the interactions of economy and mortality is certainly not new. In the historical context of a society living at the subsistence level, a Malthusian relationship between mortality and the economy is often assumed, in which shrinking real wages caused by population growth or a crop failure inevitably increased mortality.[2] However, this paper is not about history, but rather about the United States and other industrialized countries in recent decades. There is a broad literature relating mortality to the state of the economy in modern times, which is discussed in detail later on.[3] In particular, Brenner (e.g., 1971, 1979) claimed that the traditional connection of low mortality in good and high mortality in bad economic times is still effective. This conventional socio-epidemiological wisdom of countercyclical mortality has recently been reversed by Ruhm (2000), who found evidence for procyclicality.

In general, the literature neglects age-specific differences in mortality fluctuations. Moreover, in most cases unemployment is the only business cycle indicator. We extend the analysis by an adequate inclusion of the age dimension of mortality and discriminate between the impact of changes in unemployment and in the growth of the gross domestic product (GDP). In the main, we analyze mortality changes along the business cycle in the United States since 1956 on an annual basis. In addition, we include data since 1933 covering the long aftermath of the Great Depression and World War II. Identical analyses for France and Japan enable us to draw an international comparison of the particular relations between mortality and the business cycle in countries with different economic and demographic experiences and very different institutional settings. We always take a macro perspective and do not use micro data on individual life courses, i.e., we do not claim that exactly the same persons are hit by unemployment and changed mortality, but the society as a whole.

Of course, only some people lose their job in a recession. Nevertheless, many more may be affected indirectly if unemployment is correlated with changing working conditions or working habits of the still employed. Diffusion of lifestyle changes via social networks forms another possible channel, how job losses of some can alter health and mortality of many. As well as other aspects of human behavior, the health related risky or preventive behavior of individuals is influenced by the example of others. Smoking, alcohol abuse, excessive calories intake or deficient physical activity are obvious examples of risky behavior changing

[2]The idea goes back to Malthus (1798).

[3]Cf. Section 2.

collectively over time. The reason is not only that people share many activities in their peer group. Social norms determine, what is widely considered as usual or at least acceptable in a community and what is not. For example, Christakis and Fowler (2007) show that the risk of obesity is connected with the prevalence of obesity in the peer group of an individual, but also transmitted in the social network between distant people, who have no direct contact at all. This results in *social infections* or *social epidemics*. Thus, life style changes of some people, who are especially affected by economic shocks, may have an influence on the many others not affected directly, and general mortality may change in line with general economic conditions.

An objection against economically induced mortality fluctuations might be that the wave length of the business cycle is short compared not only with the entire human life cycle, but also with the courses of many lethal diseases. At a first glance, this might indeed challenge any major relation between mortality and the business cycle. Nevertheless, some reasoning about actual causes of deaths refutes this argument. It is self-evident that the incidence of deaths from external causes like infections, accidents or violence may change in principle at least as rapidly as the state of the economy does. Even if the incidence of these fatalities is quite low, they still may account for a significant variation in mortality. For many other causes of deaths it might seem less plausible to assume short-run fluctuations. For example, myocardial infarction as a major preventable cause of death is typically associated with a long course of gradual deterioration of the health status, often related to decades of unhealthy behavior. However, we have to distinguish between long-run causes of a precarious health status and very short-run incidents which finally trigger the infarction itself. The importance of short-run circumstances is demonstrated by significant variations of the incidence of myocardial infarctions depending on the day of the week.[4] Altogether, there is strong evidence that mortality from preventable diseases, which accounts for a big portion of all fatalities, is subject to short-run influences as well.

So far, we have argued that the analysis of short-run interactions of mortality and the economy, both considered in a macro perspective, is reasonable. In this paper, we apply a Bayesian time series approach to demographic modeling, which is presented in detail in Reichmuth and Sarferaz (2008). The model specified therein is characterized by an appropriate embodiment of age-specific characteristics and can be estimated by a sophisticated Markov Chain Monte Carlo (MCMC) algorithm. A latent mortality variable, which drives the common part of the development of age-specific mortalities, and the two business cycle indicators as covariates are jointly modeled in a vector autoregression (VAR). Complemental autoregressions prevent sudden changes of the coefficients, which link these variables with particular age-specific mortalities. The VAR model accounts for potential endogeneity of all variables. The estimated VAR-parameters allow for a structural analysis of the actual interrelations of the latent mortality variable and the different covariates by

[4]For example, Willich et al. (1994) and Spielberg et al. (1996) find these variations in both West and East Germany prior to reunification. Anson and Anson (2000) find weekly cycles of mortality from external as well as from internal causes like circulatory diseases in Israel. As a further example, Sargent et al. (2004) analyze the effect of a local smoking ban in Montana and find that the incidence of infarctions is significantly reduced already in the first six months after a prohibition of public smoking.

means of impulse response functions. Based on these mutual interactions and the respective coefficients linking the variables to age-specific mortality, we calculate the full pattern of age-specific reactions to shocks in the economic variables. We trace both the reactions at a fixed age and the reactions of a real cohort aging by time. The first choice corresponds to a cross section and the second one to a diagonal section in the three dimensional surface of mortality reactions. In all cases, the Bayesian estimation approach yields not only point estimates, but information on the whole distribution of the results. Thus, we present error bands with probability masses corresponding to different percentiles of the responses of age-specific mortality.

We contribute to the debate on the effects of the business cycle on mortality triggered by recent findings of procyclical mortality contradicting conventional socio-epidemiological wisdom. For the U.S in the period 1956–2004 we find that the reaction of the male twenty to thirty years olds to macroeconomic shocks constitutes an exception. While most other age classes react negatively to a shock in unemployment and positively to a shock in GDP growth, the 25 years olds react with reversed signs. We confirm these findings with international data from France and Japan and observe that in both countries, in addition to the young male adults, even the 30 to 40 years olds react differently. This suggests that when examining the relationship between business cycles and mortality, data including all single age classes should be used, in order to avoid spurious results. To investigate a change in the relationship between macroeconomic variables and mortality rates we use data for the period 1933–1969. We find that that the relationship has indeed evolved over time. In the earlier period, all age classes react procyclical in the short-term and mostly countercyclical in the mid-term.

The rest of the paper is organized as follows. Section 2 provides a summary of relevant literature on mortality and the business cycle. In Section 3 the model is stated, while Section 4 briefly addresses the estimation procedure. The data are described in section 5. The empirical results are presented in Section 6. Finally, Section 7 concludes.

2 Literature on Mortality and the Business Cycle

In this section, we review the main literature on interactions of economic conditions and mortality in the modern world.[5] Evidence for a negative correlation in the long-run and in cross-section studies is compiled with a number of findings of procyclical mortality with respect to the business cycle. A short digression on the possible long-run implications of economic conditions completes the section.

2.1 Negative Correlation of Mortality and Good Economic State

Due to the impressive decline of mortality accompanied with an enormous rise of economic output and individual wealth since the 19th century, the correlation between mortality and good economic conditions is clearly negative in the long run.[6]

[5]We do not consider the literature on pre-industrialized societies of the Malthusian era.

[6]For the rise of life expectancy associated with the fallen mortality cf. Oeppen and Vaupel (2002).

The same negative correlation is found in cross-sections of countries with different development levels, e.g., in the World Mortality Report,[7] or of individuals with different socio-economic positions within one country. This link between mortality and economic well-being turns out to be superior to many other attempts to explain differential mortality. For example, MENCHIK (1993) finds monotonic negative influence of economic status on mortality in the U.S. and largely disproves genotype differences of ethnic groups as reason for mortality differences. DEATON (2003) discards inequality of income as a major determinant of mortality, but underscores the importance of income level as health determinant.[8] VON GAUDECKER AND SCHOLZ (2007) also find a large, monotonic impact of lifetime earnings on mortality in Germany and disprove at the same time sustained effects of having lived under the opposed institutional environments of either or West Germany prior to reunification. MACKENBACH ET AL. (2003) point out that in previous studies for all countries with available data, mortality turns out to be higher for those with low socio-economic position, no matter whether this is indicated by educational achievement, occupational class or income level.

Nevertheless, these unchallenged and well-known facts are about correlation, but not necessarily about causation. Furthermore, neither the look at long-run developments nor a simple comparison of different objects of investigation without adequate time perspective can reveal effects of short-run fluctuations. In the context of the business cycle, this means that conflicting settings in which mortality is either countercyclical, acyclical or even procyclical would all be perfectly compatible with these facts as long as mortality follows an downward time trend, whereas economic output follows an upward time trend beside their particular short-run fluctuations, i.e., time acts as a confounding factor.

Although a positive correlation of mortality and the business cycle was already discovered by OGBURN AND THOMAS (1922) for the United States 1900–1920 and six states of the U.S. 1870–1920, this result was widely ignored later on.[9] Instead, the most influential work on the interrelation of changes of economic conditions and mortality is done by BRENNER (e.g., 1971, 1979), who finds countercyclical mortality in aggregated time series. However, these studies are challenged by a number of authors for methodological reasons.[10]

2.2 Recent Findings of Procyclical Mortality

More recently, several panel studies deliver strong evidence that mortality is procyclical with respect to the business cycle. In most cases, the unemployment rate is used as economic variable. Since unemployment is countercyclical with respect to the business cycle, mortality changes are called procyclical if they are reverse to changes in unemployment as explanatory variable.

[7]Cf. UNITED NATIONS (2006).

[8]This finding is about income inequality, not about different ranks in the social environment.

[9]The authors themselves were skeptical about their findings. Decades later, similar results are found. EYER (1977) already addresses the different causes of death and their changes over the business cycle, while HIGGS (1979) looks at data from large U.S. cities 1871–1900 and relates mortality to procyclical immigration.

[10]RUHM (2000) gives an overview.

The most prominent results are due to RUHM (2000), who applies a fixed-effect model to state level data for 1972–1991 from the U.S. and finds that a sustained increase of unemployment instantaneously leads to significantly decreased mortality persisting for several years. Among adults, mainly the younger age class of 20–44 year olds is affected and much less the older classes, in which most of the fatalities occur. Among ten major causes of death only suicides increase and fatalities from cancer stay unchanged, whereas all other types of mortality decrease. The decrease is particularly strong for vehicle accidents and homicide, but also not negligible for other accidents, heart diseases and infant mortality.[11] In general, the more strongly fluctuating causes of deaths predominantly strike younger people.[12] A supplementary analysis of micro data for 1987–1995 from the annual Behavioral Risk Factor Surveillance System (BRFSS) survey yields that reported lifestyle changes are consistent with the resulting changes of mortality.[13] RUHM (2003) complements with the analogous finding that morbidity is procyclical, too, in particular with regard to acute health problems and to people in the prime-working age.

The findings of RUHM (2000) are confirmed by NEUMAYER (2004), who uses state-level data of unemployment from Germany in the period 1980–2000. He obtains some differing results concerning the individual causes of deaths, but also finds procyclical general mortality, in particular for the age classes below 45 and above 65.[14] A study on the country-level with data from 23 OECD countries in 1960–1997 by GERDTHAM AND RUHM (2006) also confirms the procyclical effect of changes of unemployment on mortality.

In contrast to these panel studies, TAPIA GRANADOS (2005b) applies a time series approach.[15] The change rates in age-adjusted general mortality as well as the change rates in specific mortality for population sub-groups, different age classes and particular causes of death are regressed on the change rates in unemployment and GDP in the United States for 1900–1996. Independent of the choice of the indicator for the business cycle, mortality is found to be procyclical for the entire period as well for all examined sub-periods.[16] He

[11] The decomposition of predicted cyclical fluctuations of mortality yields that due to their high elasticity, car accidents and other external causes (such as other accidents, suicide and homicide) account for a high percentage of the variation in spite of their relatively low incidence of fatalities. Deaths from cancer form the other extreme with high incidence, but low fluctuations, whereas so-called preventable deaths (from heart or liver diseases as well as from infectious diseases) exhibit the highest weight in both variation and incidence.

[12] With respect to deaths from coronary heart disease as the biggest single cause of death, RUHM (2006) finds working-age and older people to be affected similarly. These fatalities are procyclical and, most notably, react faster than other disease-related causes of death to the business cycle.

[13] RUHM AND BLACK (2002) and RUHM (2005) also analyze behavioral changes over the business cycle. They regress individual data from the BRFSS (1987–1999/2000) on state-level unemployment and find that tobacco and alcohol consumption as well as obesity are procyclical, whereas leisure-time physical activity is countercyclical. All changes in the prevalences are concentrated among people with worrying health related behavior, who are either heavy users, severe obese or completely physical inactive.

[14] His main results are robust to replacing the unemployment rate by GDP growth as economic indicator.

[15] In an earlier time series study, GRAHAM ET AL. (1992) find a countercyclical effect of consumption expenditures and a procyclical effect of unemployment on age-adjusted mortality in the United States in 1950–1988. They also report the cyclical patterns of major causes of death.

[16] In an even longer perspective on Sweden, TAPIA GRANADOS AND L. IONIDES (2008) find that a negative effect of economic growth on mortality in the first half of the 19th century ultimately turns into a positive

estimates that a time trend of falling mortality more than offsets the mortality raising effect of growing GDP.[17] In a similar approach, Hanewald (2008) regresses changes of mortality in (West) Germany in 1956–2004 on changes in a multitude of economic, environmental and behavioral time series. With respect to economic indicators, she finds an instantaneous positive effect of GDP on mortality, which is clearly more important than the negative effect of unemployment.[18] Unlike Ruhm (2000) and Neumayer (2004), who distinguish only three crude age classes 20–44, 45–64 and 65+, Tapia Granados (2005b) uses seven age classes from 0 to 84 and Hanewald (2008) uses eight age classes between 25 and 99. The regressions for the particular age classes are always independent from each other by construction.

In contrast to the results for the OECD, Bhalotra (2007) and Baird et al. (2007) find counter-cyclical infant mortality in India and the developing world. The effect of economic circumstances on old-age mortality is analyzed by Snyder and Evans (2006). They exploit the quasi natural experiment of a cut in social security in the U.S. and find that those born after the key date, who get lower payments and do at the same time more post-retirement work, have lower mortality than those born before.

2.3 Long-run Impact of Economic Conditions on Mortality

Adult mortality can even be affected by economic conditions much earlier in the life cycle. Barker (1992) advocates effects of fetal malnutrition on adult health via epigenetic programming of the unborn for the best fit to the current state of the world.[19] Some natural experiments in history deliver evidence for drastic long-run effects. For example, van den Berg et al. (2007) find higher mortality among older men, who were exposed to the Dutch Famine 1846–1847 in their perinatal period, and Almond et al. (2007) find negative effects of the Chinese Famine 1959–1961 on a whole bunch of socio-economic characteristics later on in life. These findings support the point that in addition to age and time their combination as cohort effect is sometimes relevant for mortality, too. Actually, long-run effects on mortality via fetal and neonatal programming are not limited to such rare extreme events. For example, Doblhammer and Vaupel (2001) show the effect of the month of birth on mortality above age 50 caused by the nutrition of the womb. With respect to the business cycle, significant negative effects of a good economic state at birth on mortality later on in life are found by van den Berg et al. (2006) for the birth cohorts 1812–1912 in the Netherlands.[20] Nevertheless, in this paper we focus on short-run interactions of economic conditions and mortality and not on possible long-run consequences.

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one in the second half of the 20th century.

[17]This article has sparked a vigorous debate in the *International Journal of Epidemiology* between supporters of the conventional view of Brenner that improved socio-economic conditions improve health and supporters of Tapia Granados in line with Ruhm.

[18]Other economic indicators turn out to be insignificant.

[19]This results in a reduced body size or a thrifty metabolism appropriate for poor nourishment, which increases the risk of cardiovascular and other diseases in adult life. The sex ratio is possibly also shifted towards more females. This programming constitutes a fast adaption mechanism within on generation supplementary to the evolutionary genetic adaption.

[20]These findings are confirmed by van den Berg et al. (2008) for the Danish birth cohorts 1873–1906.

3 A Bayesian State Space Model

To capture the interrelation between mortality and macroeconomic time series, we use the methods described in Reichmuth and Sarferaz (2008). The common components of age-specific demographic variables are modeled as latent variables and linked with macroeconomic data through a structural VAR (SVAR). To ensure a smooth development along the age dimension we assume for the coefficients, which link the explanatory variables with the age-specific demographic variables, to follow AR-processes. In the following we describe the model in more detail.

The observed demographic variables $d_{x,t}$ with age classes $x = 0, \ldots, A$ and time periods $t = 1, \ldots, T$, can be expressed as

$$d_{x,t} \;=\; \overline{d}_x + \beta_x z_t + \epsilon^d_{x,t} \tag{1}$$

with the arithmetic mean $\overline{d}_x = \frac{1}{T}\sum_{t=1}^{T} d_{x,t}$ and explanatory variables $z_t \equiv [\kappa_t \;\; Y_t]'$, where κ_t is a $K \times 1$ vector of unobservables and Y_t is a $N \times 1$ vector of observed covariates. The corresponding coefficient vector $\beta_x \equiv [\beta^\kappa_x \;\; \beta^Y_x]$ is $1 \times M$, where β^κ_x is a $1 \times K$ vector and β^Y_x is a $1 \times N$ vector with $M = K + N$. For the disturbances in Equation (1) we assume $\epsilon^d_{x,t} \sim i.i.d.\ \mathcal{N}(0, \sigma^2_d)$.

For the explanatory variables z_t we assume the following vector autoregressive process

$$z_t = c + \phi_1 z_{t-1} + \phi_2 z_{t-2} + \cdots + \phi_p z_{t-p} + \epsilon^z_t\,, \tag{2}$$

where c is a $M \times 1$ vector of constants and $\phi_1, \ldots, \phi_p$ are $M \times M$ coefficient matrices. The disturbances in Equation (2) can also be written as $\epsilon^z_t \equiv A\nu_t$, where A is a $M \times M$ coefficient matrix containing contemporaneous relations between the variables in z_t. For the $M \times 1$ vector of structural disturbances ν_t we assume $\nu^z_t \sim i.i.d.\ \mathcal{N}(0, I_M)$ and for the $M \times 1$ vector of reduced form disturbances ϵ^z_t we assume $\epsilon^z_t \sim i.i.d.\ \mathcal{N}(0, \Sigma_z)$, where $\Sigma_z = AA'$.

For the coefficient vector β_x we assume the following law of motion

$$\beta_x = \alpha_1 \beta_{x-1} + \alpha_2 \beta_{x-2} + \cdots + \alpha_q \beta_{x-q} + \epsilon^\beta_x \tag{3}$$

with $\epsilon^\beta_x \sim i.i.d.\ \mathcal{N}(0, \Sigma_\beta)$, where Σ_β is a $M \times M$ diagonal matrix. As the coefficient matrices $\alpha_1, \ldots, \alpha_q$ are also assumed to be diagonal, each component of β_x in fact follows an autoregressive process on its own. All disturbances are assumed to be independent of each other.

To identify the model uniquely we set the lower $K \times K$ block of β^κ_x to a diagonal matrix and the lower $K \times N$ block of β^Y_x to zero.

We choose the same priors as in Reichmuth and Sarferaz (2008). For the parameters in Equations (2)–(3) we assume Minnesota-type priors by centering the probability mass for the first lagged coefficient around one and for all subsequent lags around zero, whereby decreasing subsequently the uncertainty that the coefficients are zero for more distant lags. For the variance of the disturbance in Equation (1) we assume a quite diffuse inverted gamma distribution.

4 Estimation

We estimate the model described in equations (1)–(3) using Markov Chain Monte Carlo methods. More precisely we apply the Gibbs sampler. We draw from the joint distribution $\mathcal{P}(\Psi, z, \beta)$ by subdividing it into the conditional distributions $\mathcal{P}(\Psi \mid z, \beta)$, $\mathcal{P}(z \mid \Psi, \beta)$ and $\mathcal{P}(\beta \mid \Psi, z)$ and draw iteratively from them, where Ψ comprises all parameters of the model. Taken initialized values for $z^{(0)}$ and $\beta^{(0)}$ as given, we sample in the i-th iteration $\Psi^{(i)}$ from $\mathcal{P}(\Psi \mid z^{(i-1)}, \beta^{(i-1)})$, $z^{(i)}$ from $\mathcal{P}(z \mid \Psi^{(i)}, \beta^{(i-1)})$ and $\beta^{(i)}$ from $\mathcal{P}(\beta \mid \Psi^{(i)}, z^{(i)})$ successively. Under weak conditions and for $i \to \infty$ the Gibbs sampler converges and we obtain samples from the desired joint distribution $\mathcal{P}(\Psi, z, \beta)$.[21] For a more detailed description of the estimation procedure we refer to Appendix A of Reichmuth and Sarferaz (2008).

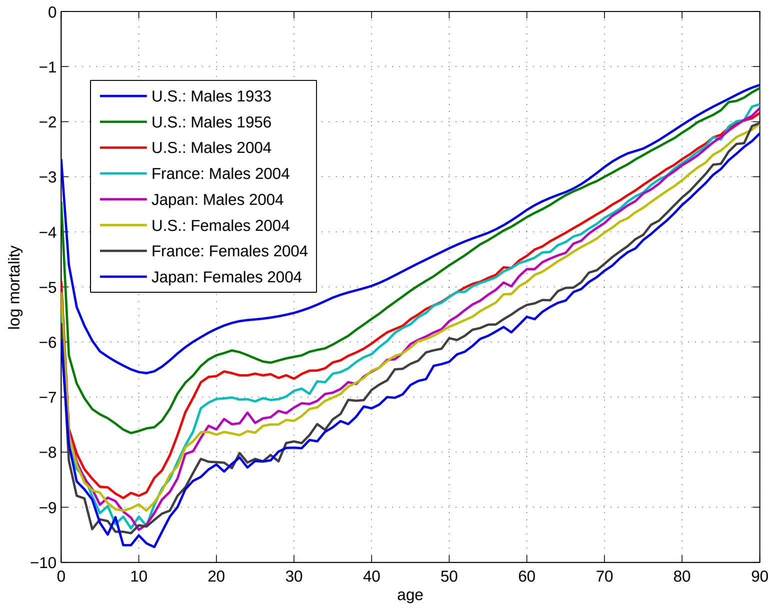

Figure 1: Age-specific log mortality for both sexes in the U.S., France and Japan.

5 Data

We analyze demographic-economic interactions in the United States in the periods 1956–2004 and 1933–1969 as well as in France and Japan in the period 1956–2004. The time series of logarithmized age-specific male and female mortality for 91 individual age classes from 0 to 90 are provided by the Human Mortality Database.[22] Figure 1 displays some examples of these age-specific mortalities.

[21] Cf. Geman and Geman (1984).

[22] Cf. Human Mortality Database (2008). In the Human Mortality Database obvious mistakes in the raw data are eliminated and death rates for the age classes 80 and above are smoothed by fitting a logistic function according to Thatcher et al. (1998) if the number of observations becomes too small. Wilmoth et al. (2007) supply a detailed method protocol. In the case of the U.S., population estimates 1940–1969 are adjusted and the extinct cohort method supposed by Kannisto (1994) is applied for the age classes 75 and above in the period 1933–1939. In the case of France, the data of infant deaths up to 1974 are corrected for false stillbirths.

As economic indicators for the business cycle we use time series of the unemployment rate and of GDP growth. The left column of Figure 2 displays the unemployment rates, which are measured as percentage of unemployed in the civilian labor force aged 16 or older in the U.S.,[23] as standardized unemployment rate in France[24] and as percentage of unemployed in the labor force aged 15 or older in Japan.[25] The right colum of Figure 2 displays the real GDP growth rates calculated from chained series provided by the Penn World Tables[26] and by the U.S. Census Bureau (2007) in case of the U.S. 1933–1969.

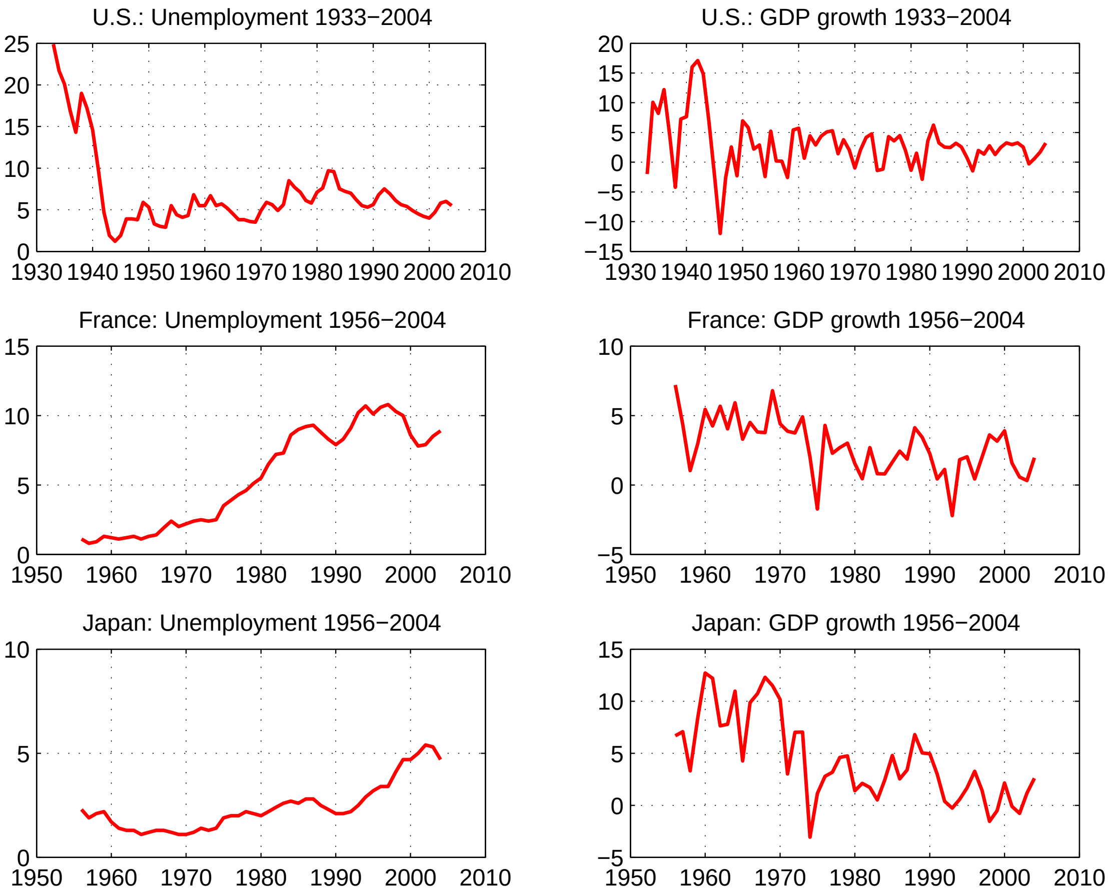

Figure 2: Unemployment rate and real GDP growth rate in the U.S., France and Japan. Note, that the scales are different, and use the uniform grid lines for convenient comparisons.

[23] C.f U.S. Census Bureau (2007). The pre 1947 unemployment figures refer to persons aged 14 or older, but this minor change causes no jump in 1947, when both definitions yield the same number.

[24] Cf. OECD (2008).

[25] Cf. Japan Statistics Bureau (2008a) and Japan Statistics Bureau (2008b).

[26] Cf. Heston et al. (2006).

6 Empirical Results

For the empirical results we use a lag length of $p = 4$ for the z's and $q = 4$ for the β's. To ensure convergence of the Gibbs sampler we restart the algorithm several times using different starting values drawn from an overdispersed distribution and compare the results. We observe that our sampler reaches convergence already after a few thousand draws. To avoid influences of the starting values we discard the first half of the chain as burn-in phase.

In the following we begin our empirical analysis with U.S. data for the period 1956–2004 as our main application. Afterwards, we compare the result to data for 1933–1969, in order to detect whether the relationship between macroeconomic variables and mortality rates evolves over time. Then we draw an international comparison with data from France and Japan.

6.1 Identification

We apply the Choleski-decomposition for the identification of structural shocks in the mutual impulse response functions of the three variables. This identification scheme builds on a triangularization of the covariance matrix Σ_z, which implies that not all variables can react instantaneously to an impulse in a particular variable. So, the ordering of the variables is crucial for the results. Of course, this affects the presented responses of age-specific mortality, too, which are derived from these mutual interactions. As already discussed in the introduction, mortality often reacts very quickly to short-run influences like changing economic conditions. In today's western industrialized countries with their low and quite stable mortality, we assume the reverse effect to be small and less fast.[27] Thus, in the context of mortality and the business cycle, mortality has to be placed last to allow for instantaneous reactions to the economic variables placed before it. The main business cycle indicators GDP growth and unemployment are negatively correlated and the mutual effects are partly contemporaneous. Nevertheless, the unemployment rate is known as lagged business cycle indicator. The labor market is subject to many frictions and search and matching problems, which impede fast adjustments. Figure 2 shows that the unemployment rate is by far less volatile than the GDP growth rate. Hence, in the ordering of the variables

$$z_t \equiv [\,unemployment,\, GDPgrowth,\, \kappa\,]_t{}'$$

unemployment has to be placed before GDP growth to account for the more important instantaneous reactions of GDP growth on unemployment.

6.2 U.S. 1956–2004

Now we describe our empirical results for the post World War II U.S. data set. Figure 3 shows the surface of median mortality responses to an unemployment shock. The big differences regarding the responses of different age classes are an interesting aspect of this figure. While the 20 to 30 years olds react positively, all other age classes react negatively.

[27] We do not consider possible major mortality crises caused by pandemics, large-scale natural disasters, war, terrorism, etc., but the small fluctuations related to the business cycle.

As it is discussed below, these differences are found for responses to a GDP growth shock as well, suggesting that relevant information might get lost by crude age grouping instead of using single age class mortality rates.

There are two ways to illustrate our findings for a particular age class. First, we cut a plane parallel to the time axis out of the mortality surface described in Figure 3 and plot it with the corresponding error bands. We denote this as *age impulse response function* (AIRF). Second, we cut a plane out of the mortality surface along the diagonal of the age and time axis. We denote this as *cohort impulse response function* (CIRF). It turns out that the CIRFs and the AIRFs are very similar. Hence, for convenience we report the CIRFs only and state it explicitly when they deviate from each other. We construct CIRFs and AIRFs of age-specific female mortality rates as well. Since they are quite similar to the results we obtain for male mortality, we do not show the results for female mortality and state it explicitly when they are different from those for male mortality.

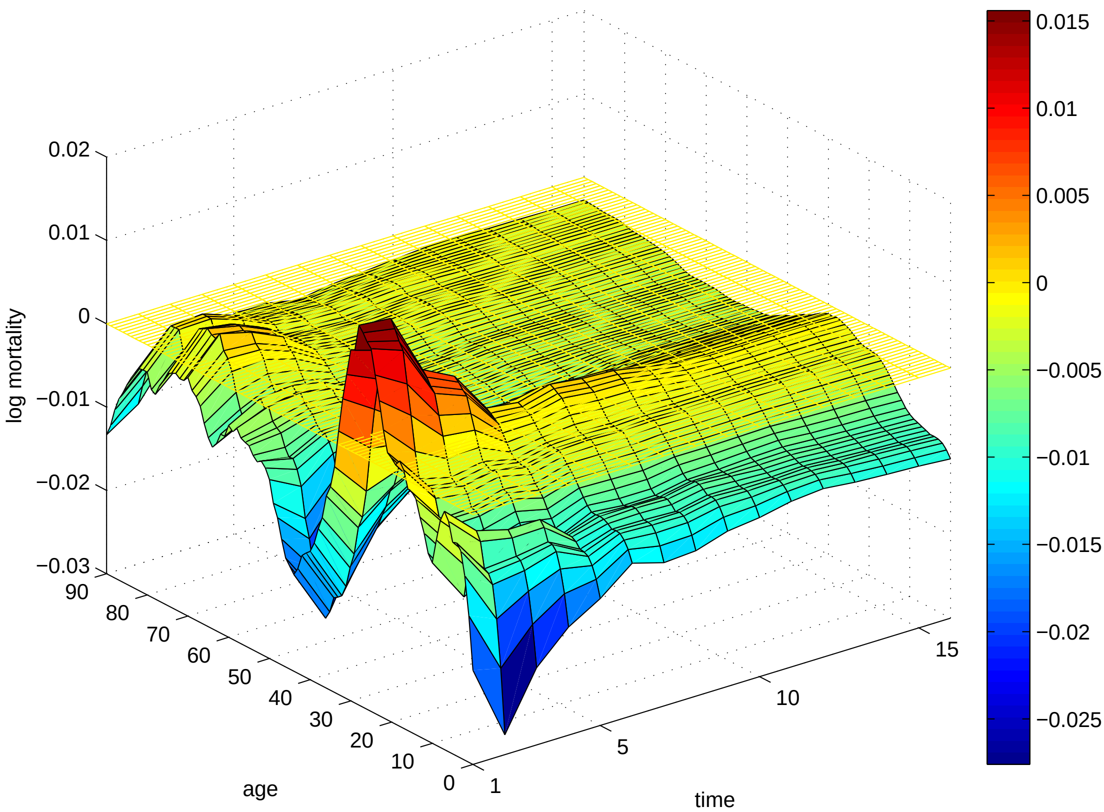

Figure 3: Surface of the median response of logarithmized age-specific male mortality to a one standard deviation shock in unemployment in the U.S. 1956–2004. The time axis refers to the time elapsed since the impulse and the yellow grid marks the zero plane.

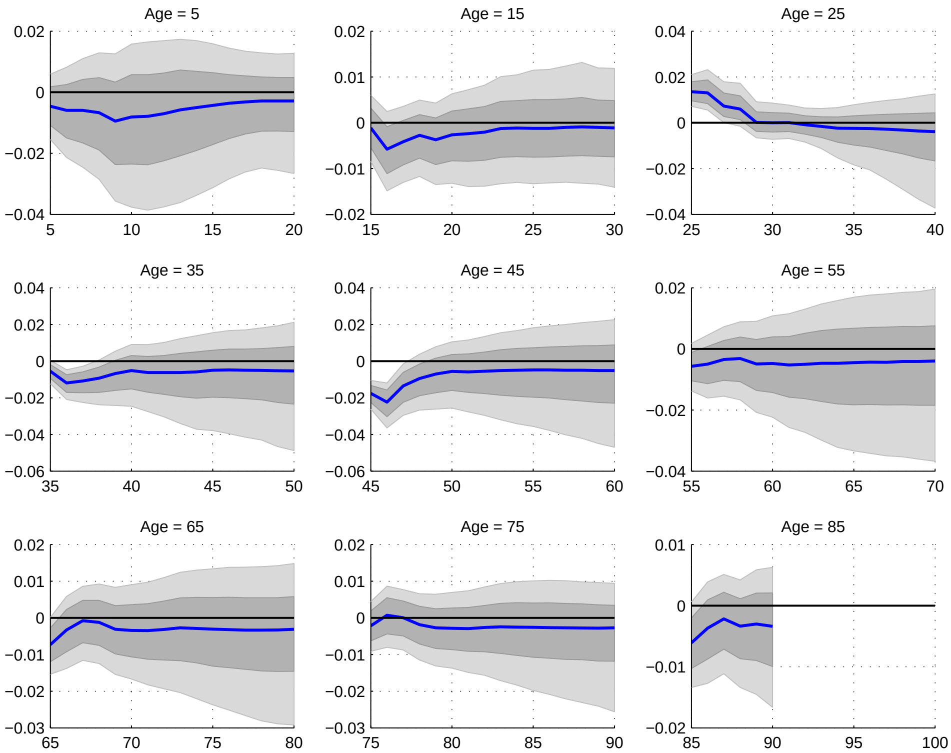

Figure 4: Responses of log mortality in the further life of some male cohorts to a one standard deviation shock in unemployment occurring at the labeled age. The entire gray shaded area around the blue median represents 90% and the dark gray shaded area respresents 68% of the posterior probability mass.

Figure 4 plots the male CIRF of an unemployment shock. Looking at the responses of child and adolescent mortality we find that the probability mass of the responses center around zero. For the age class of the 25 years olds we observe a positive response to an unemployment shock, which persists with most of the probability mass above zero up to the age of 28. For the age classes of 35 and 45 years olds we observe negative responses, which peter out after four years. For the age classes of 55 to 75 we find responses centered very much around zero. Finally, the age class of 85 years olds exhibits a negative contemporaneous reaction to an unemployment shock. Evidently, Figure 4 reveals that the age classes from 25 years olds to 45 years olds are strongly exposed to a shock in unemployment. This seems to be plausible, since a large part of the U.S. work force is included in this range. It should be noted that the anomaly we observe for the male mortality rates of the 25 years olds does not show up for female mortality rates.

Figure 5 reports the CIRF of a shock to GDP growth. The age group of 25 years olds represents an exception like in Figure 4. While all other age classes show a positive and persistent reaction, in the group of 25 years olds it is anticyclical in the beginning and turns positive after about five years. This indicates that the mortality responses of the 20 to 30 years olds are driven by different factors than that of all other age groups.

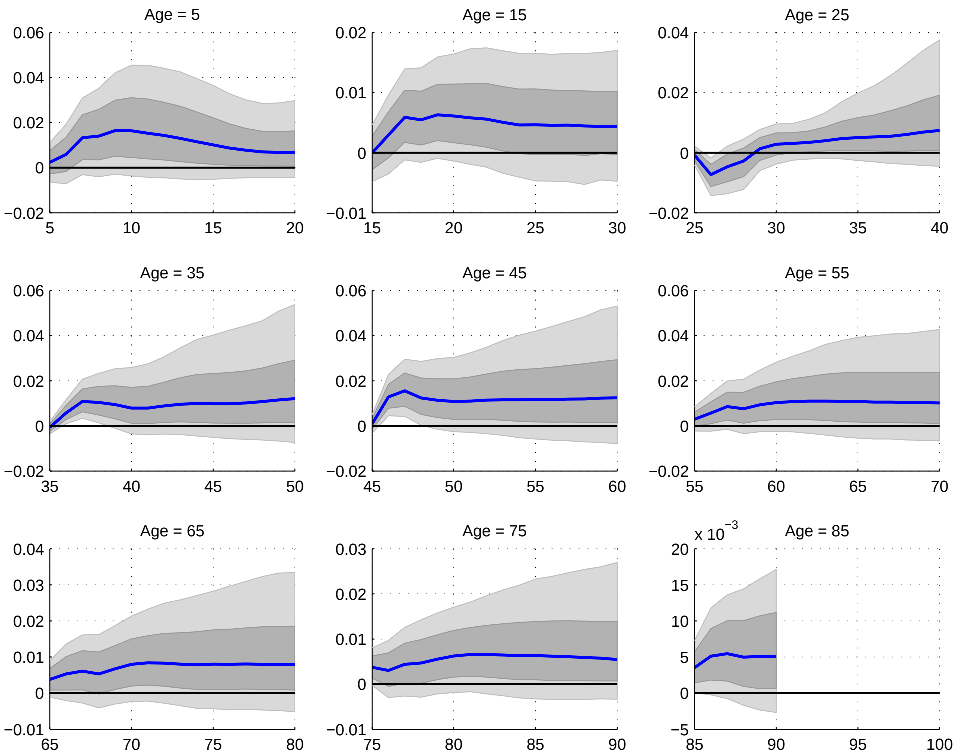

Figure 5: Responses of log mortality in the further life of some male cohorts to a one standard deviation shock in GDP growth occurring at the labeled age. The entire gray shaded area around the blue median represents 90% and the dark gray shaded area respresents 68% of the posterior probability mass.

Overall, the findings presented in Figure 4 and 5 confirm for almost all age classes the evidence described in RUHM (2000), whereas the reaction of male 25 years olds poses an exception, which is in line with the evidence found in BRENNER (e.g., 1971, 1979). This discrepancy in the results for particular age classes implies that studies, which rely on crude age grouping, may miss important features of the data, because opposed effects possibly cancel each other. The anomaly in the mortality reactions of young adults coincides with the anomaly in the mortality level known as *accident hump*, which is associated with risky attitudes. According to this, a careless attitude of heavily discounting future consequences, leading to increased risk taking in the pessimism of an economic downturn, delivers a possible explanation for rising mortality of young adults. This is complemented by the possibility that they are in fact more exposed to economic hardship, because they have not yet accumulated sufficient resources to smooth consumption and may also suffer from stronger fluctuations of youth unemployment. The lack of an own family reinforces both arguments due to less responsibility and missing support. Another possible explanation for differential mortality is that chronic diseases are rare among young adults and, unlike older people, they do not suffer from increased numbers of infarctions triggered by stress in a boom. Hence, compared to the rest of the population, they may have health disadvantages in a recession, but advantages in a boom.

6.3 Change over time: U.S. 1933–1969

The U.S. economy underwent dramatical changes during the period 1933–2004. Some major events, possibly altering the relationship between macroeconomic variables and mortality, are the Great Depression of the early 1930s and World War II. Looking at the upper-left panel of Figure 2 we observe an enormous decline of the U.S. unemployment rate during the 1930s and 40s, indicating that the decreasing unemployment and mortality rates coincide. To analyze these event separately we make use of the sub-period 1933–1969 and construct CIRFs.

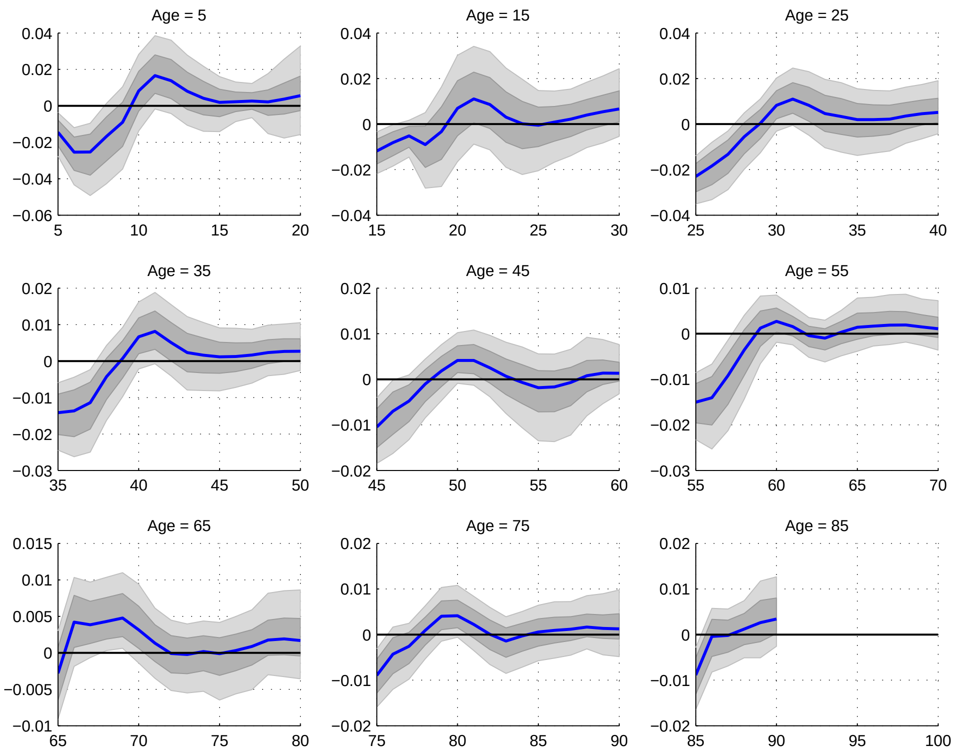

Figure 6: Responses of log mortality in the further life of some male cohorts in the period 1933–1969 to a one standard deviation shock in unemployment occurring at the labeled age. The entire gray shaded area around the blue median represents 90% and the dark gray shaded area respresents 68% of the posterior probability mass.

Figure 6 and 7 report the CIRFs for the period 1933–1969. As opposed to the post World War II period, all age classes react in a similar fashion to a shock in unemployment and GDPgrowth. While all age classes react negatively to a shock in unemployment in the first four years, after four years all responses switch signs and turn positive. The responses to a shock in GDP growth are positive and last about five years. Two outcomes in Figure 6 and 7 are striking. First, on the one hand Figure 6 indicates that the short term responses of the mortality rates are similar to results found in Ruhm (2000).[28] On the other hand it also

[28]Cf. Section 2.2

indicates that the mid-term responses are line with Brenner (e.g., 1971, 1979). Second, whereas the response of the age class of the 25 years olds is procyclical in the 1954–2004 period, it turns anticyclical for 1933–1969. This suggests that transmission channels from macroeconomic variables to age-specific mortality rates might have changed over time.

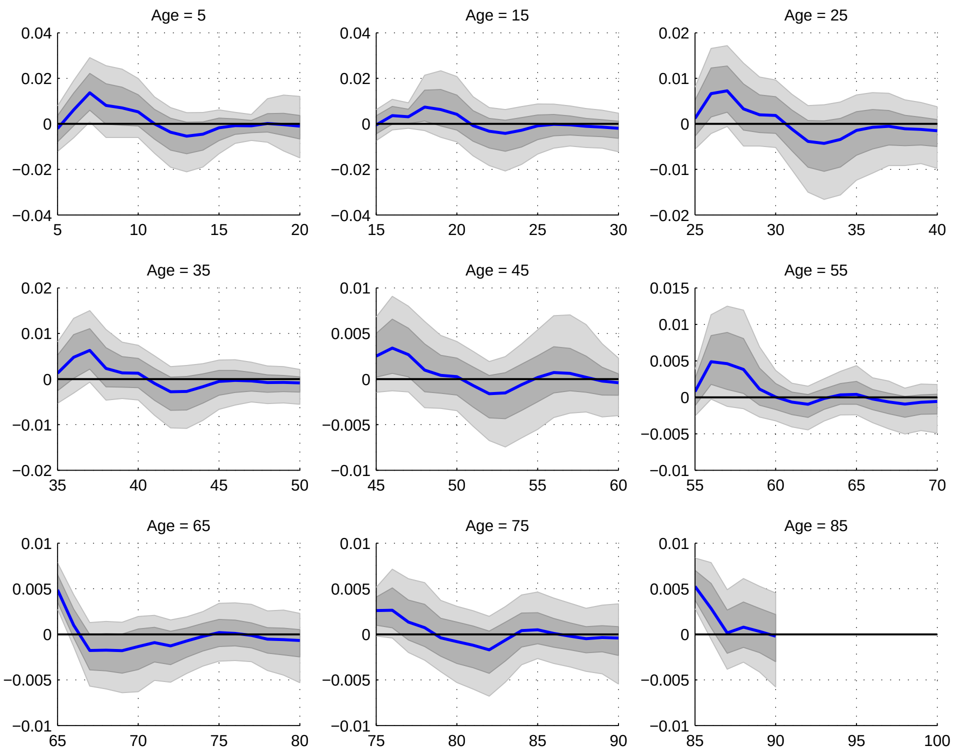

Figure 7: Responses of log mortality in the further life of some male cohorts in the period 1933–1969 to a one standard deviation shock in GDPgrowth occurring at the labeled age. The entire gray shaded area around the blue median represents 90% and the dark gray shaded area respresents 68% of the posterior probability mass.

6.4 International Comparison: France and Japan 1956–2004

The influence of the business cycle on mortality may not only vary by time, but also across countries. To draw an international comparison, we analyze data from France and Japan in the period 1956–2004 in addition to the U.S. These examples of western industrialized countries from three continents differ in many economic, institutional and demographic aspects, so that they are well suited to detect possible differences, but also common features in the interrelation of economic fluctuations and mortality.[29]

[29]Figure 1 shows that the level of current mortality at almost all age classes is highest in the U.S. and lowest in Japan.

In fact, the reactions patterns after an unemployment shock for France and Japan are quite different from that for the U.S. Figure 8 and 9 present male CIRFs for these countries. The main common feature in all three countries is the temporarily increased mortality of young adults around age 25. In France and Japan, the rise actually appears for both sexes and in a wider age range than in the U.S. The reactions of most of the other age classes exhibit definite discrepancies. Unlike to the U.S., no patterns of pronounced short-lived negative responses in the age groups 35 and 45 exist. Most of the probability mass for the 35 years olds is temporarily above zero, which is in line with the mortality responses of the even younger adults. The mortality reactions of all age classes between 45 and 85 are clearly and persistently negative in France. In Japan, at age 65 most of the probability mass is below zero, too, and the reactions are clearly negative at the highest ages of 75 and 85.

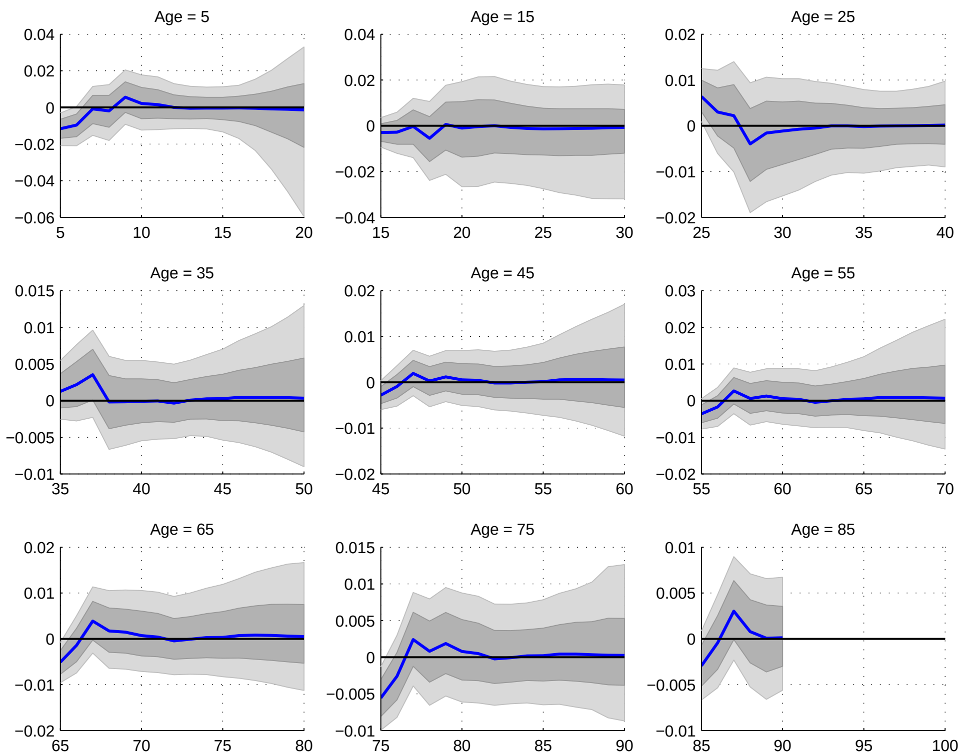

Figure 8: Responses of log mortality in the further life of some male cohorts in France to a one standard deviation shock in unemployment occurring at the labeled age. The entire gray shaded area around the blue median represents 90% and the dark gray shaded area represents 68% of the posterior probability mass.

A special feature for male children in France illustrates possible differences between the mortality reactions at a fixed age (AIRF) and of an aging cohort (CIRF). The mortality reaction of 5 years olds in the AIRF is persistently negative.[30] After the first years, in which it is centered around zero, most of the probability mass of the response at age 15 is

[30]For the AIRF, cf. Figure 10 in the appendix.

below zero, too. In contrast, the responses in the presented CIRF get increasingly positive, when the originally 5 or 15 years olds enter the phase of young adulthood. This means that people can be subject to particular long-lasting mortality reactions in an age group, even if they enter this age group not until several years after the shock. Whereas these people have at least experienced the shock in an earlier phase of their life cycle, the finding from the AIRF actually implies that male children in France still profit from an unemployment shock many years before their own birth. In this case, the transmission to the children probably proceeds via lasting changes in the characteristics of parents and social environment. Of course, child mortality in today's industrialized countries is very low, so that even very little absolute changes matter a lot.[31] Nevertheless, it is noteworthy that mortality decreases after an unemployment shock are restricted to male children. At the age of 15, female mortality actually exhibits an increase in France and Japan, too.

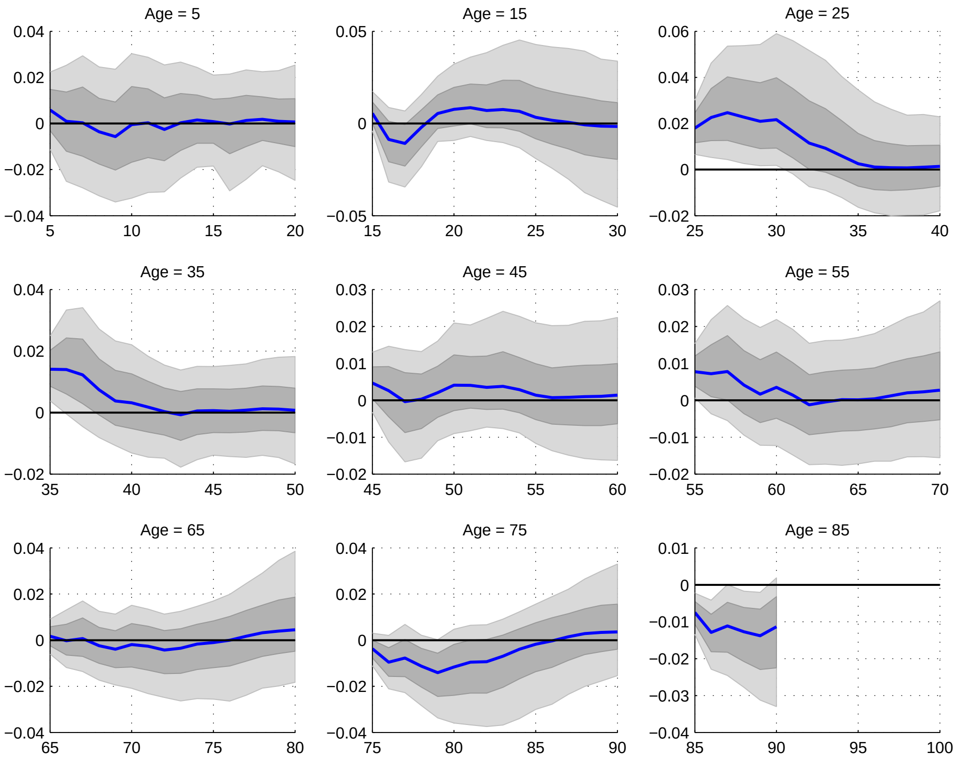

Figure 9: Responses of log mortality in the further life of some male cohorts in Japan to a one standard deviation shock in unemployment occurring at the labeled age. The entire gray shaded area around the blue median represents 90% and the dark gray shaded area represents 68% of the posterior probability mass.

[31] In each single age class of males between 5 and 12 only 30–50 cases of death occurred in France in 2004. The numbers for females are even lower. C.f. Human Mortality Database (2008).

The patterns of mortality reactions after a shock in GDP growth are relatively similar in all three countries.[32] Most age classes show a persistent increase, at least after the first one or two years. Only young adults in France and the U.S. exhibit decreased mortality. In the U.S., the effect is short-lived and restricted to the male age class of 25 years olds. In France, the effect lasts a little bit longer, and for 15 years olds after several years and for females in the first year, most of the probability mass is below zero, too.

All in all, the international comparison yields some differences, but as most striking result that in most cases a distinction has to be made between young adults and the rest of the population. With the exception of females in the U.S., young adults always suffer from increased unemployment. On the other hand, they often profit from increased GDP growth. Thus, their mortality is low in a boom and high in a recession. The rest of the population does not always react conclusively, but often profits from increased unemployment. These reactions are quite short in the U.S. and most widespread and persistent in France.[33] GDP growth always turns out to be harmful. Hence, for a large fraction of the population mortality is high in a boom and low in a recession.

6.5 Ethical Dilemma

The detected relationship with procyclical mortality of a large part of the population is a delicate issue. While young adults often profit, many people suffer at the same time from a small, but significant increase of mortality in a boom. This might be seen as ethical dilemma, because a good state of the economy is in general assessed as desirable. However, we think that this desirability still holds even if an economic upturn slightly increases mortality. From a theoretical point of view, which is most prevalent in economics, rational economic agents always make their decisions with the objective to maximize their own expected utility. The methodological individualism implicates that a high output and a high employment as well as all their direct and indirect implications result from these voluntary choices. Consequentially, the overall outcome has to be associated with high utility in average and is desired by the agents.[34] From an empirical point of view, there is also no contradiction in aiming for a boom despite its mortality side effect, because people uncoerced engage in all kinds of risky behavior in their private life, too. They ignore safety and health advices of all kinds and often trade off a slightly increased risk of death against various benefits in their own pursuit of happiness.[35] Nevertheless, with respect to both points of view, individuals might suffer from restricted information distorting their decisions. Hence, we think that in principle there is a role for health education to mitigate the adverse mortality effect of changed living conditions and individual behavior along the business cycle.

[32] Cf. figures 11 and 12 in the appendix.
[33] This is of course in line with the more flexible labor market in the U.S.
[34] Admittedly, there might be problems with external effects of individually optimal choices.
[35] Many people like to consume alcohol or tobacco, have an unhealthy diet, do hazardous sports, participate unnecessarily in traffic, visit dangerous places or expose themselves to sexually transmitted diseases.

7 Conclusion

In this paper we analyze the impact of short-run economic fluctuations on age-specific mortality. We contribute to the debate on the cyclicality of the effects of the business cycle on mortality triggered by recent findings of procyclical mortality by Ruhm (2000), which contradict conventional socio-epidemiological wisdom according to Brenner (e.g., 1971, 1979).

For the first time, we examine the differing consequences of economic changes for all individual age classes. To this end, we build on the model of Reichmuth and Sarferaz (2008) to set up structural VARs of a latent mortality variable and of unemployment and GDP growth as main business cycle indicators. Age-specific coefficients link these variables to the actual mortality of single age classes. Impulse response analyses show the age-specific mortality reactions to structural shocks in the economic variables.

For the U.S. in the period 1956–2004 we find that young male adults noticeably differ from the rest of the population. The 25 years olds exhibit increased mortality in a recession, whereas the age classes of 35 or 45 years olds react with lower mortality to increased unemployment and, like older people, with higher mortality to increased GDP growth. Thus, analyses of the cyclicality of mortality changes have to differentiate closely between particular age classes, especially in the age range of young adults, to avoid spurious results due to possible neutralization of opposed effects.

The special role of young people with respect to mortality changes coincides with that in mortality levels known as *accident hump*. Possible explanations for the anomaly in the mortality reactions span from higher risk taking or actually more severe adverse effects on young adults in a recession to chronic diseases of older people facilitating acute myocardial infarctions in the stress of a boom.

In an analysis of an earlier period since 1933, all age classes react procyclical to economic changes. This may point to long-term changes of the channels between macroeconomic conditions and mortality. Admittedly, the aftermath of the Great Depression dominates this sample. An international comparison with France and Japan confirms the special role of young adults in the post-war period. Their countercyclical pattern of mortality reactions extends to both sexes and affects even a little wider age range than in the U.S. Most other age classes show a procyclical reaction pattern. This also holds for people in the retirement age, in particular outside of the U.S., who are not directly subject to the state of the labor market. In general, the mortality responses are most short-lived in the U.S. and most persistent in France, which suggests a relation to institutional differences. Nevertheless, the clear-cut contrast between countercyclical mortality of young adults and widespread procyclical mortality in the rest of the population is the most striking result.

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Appendix: Supplementary Figures

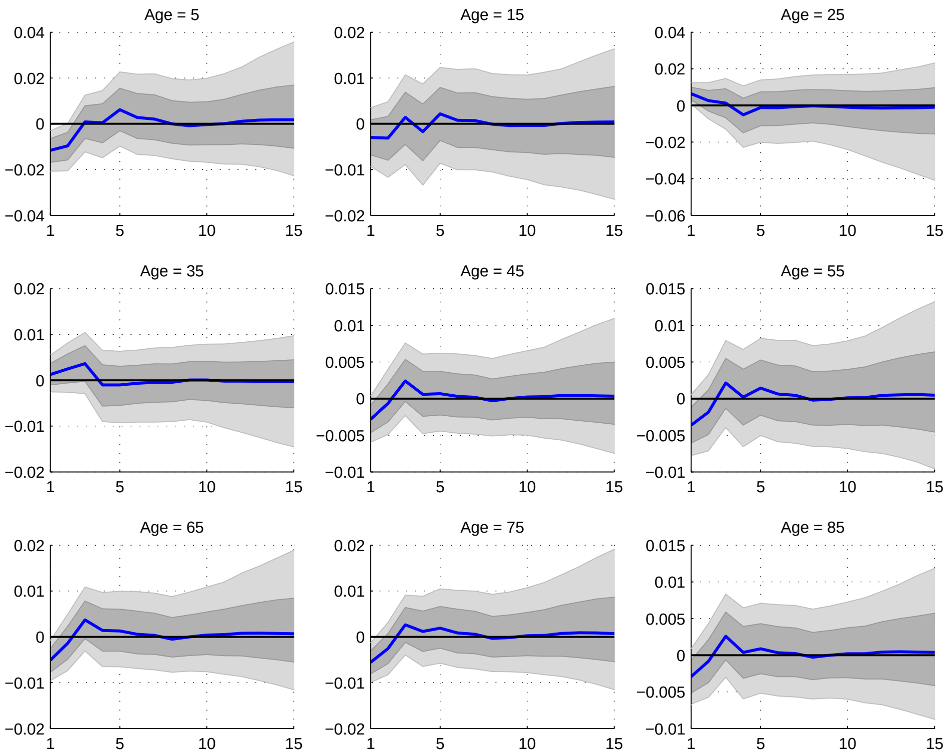

Figure 10: Responses of log mortality at some fixed ages of males in France to a one standard deviation shock in unemployment occurring in year 1. The entire gray shaded area around the blue median represents 90% and the dark gray shaded area respresents 68% of the posterior probability mass.

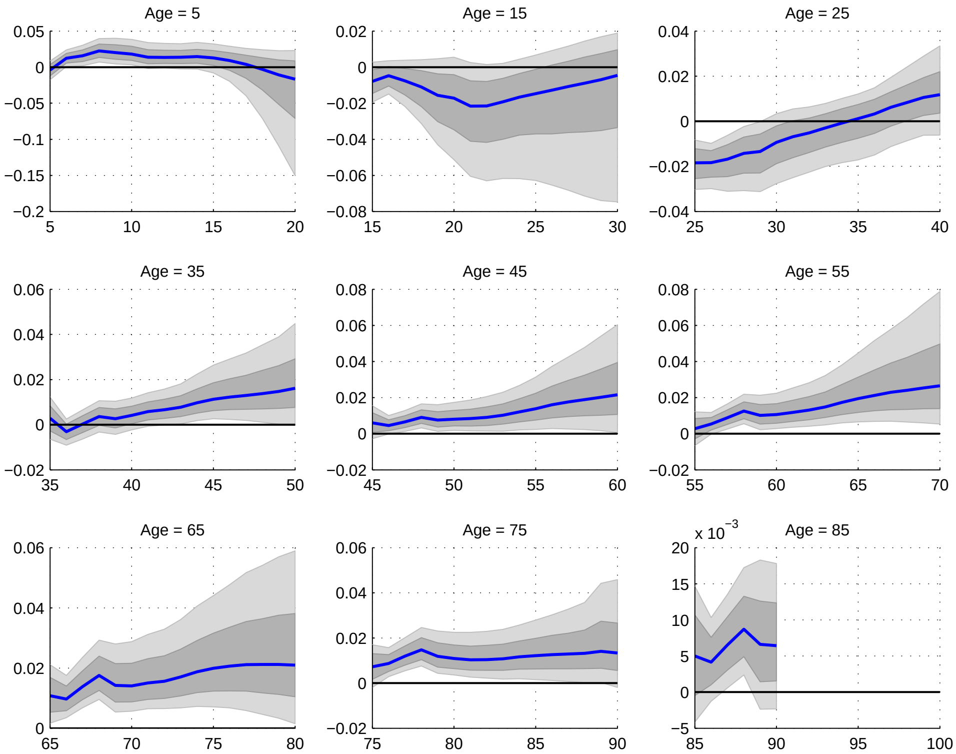

Figure 11: Responses of log mortality in the further life of some male cohorts in France to a one standard deviation shock in GDP growth occurring at the labeled age. The entire gray shaded area around the blue median represents 90% and the dark gray shaded area represents 68% of the posterior probability mass.

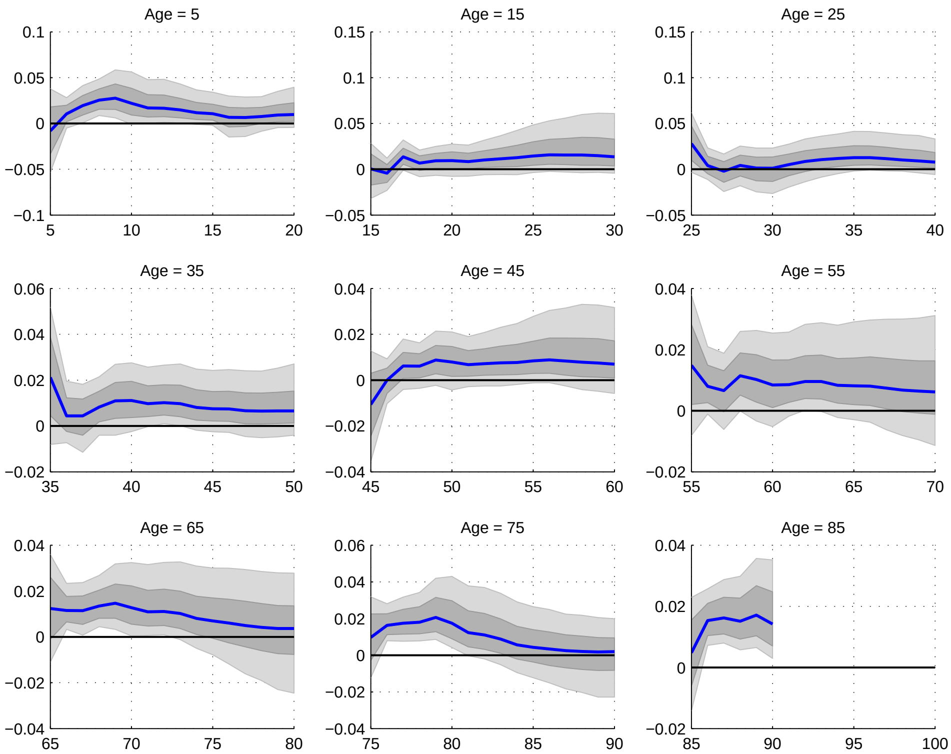

Figure 12: Responses of log mortality in the further life of some male cohorts in Japan to a one standard deviation shock in GDP growth occurring at the labeled age. The entire gray shaded area around the blue median represents 90% and the dark gray shaded area respresents 68% of the posterior probability mass.

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