



# Recessions lower (some) mortality rates: evidence from Germany

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## Abstract

In his article with the provocative title “Are Recessions Good for Your Health?”, Ruhm (J. Health Econ. 21(4) (2000) 659) has found robust and consistent evidence that the total mortality rate, age-specific mortality rates as well as most specific mortality causes are pro-cyclical. His finding that high unemployment rates are associated with lower mortality and vice versa stands in stark contrast to Brenner’s earlier work, who found the opposite effect, possibly after a time lag. Ruhm controls for state-specific effects in a panel of US states over the period 1972–1991, whereas Brenner’s work is based on time-series analysis. Extending and improving upon Ruhm’s original analysis, we analyse the effect of state unemployment and economic growth rates on mortality in the states of Germany over the period 1980–2000, both in a static and a dynamic econometric model. Controlling for state-specific effects, we find evidence that aggregate mortality rates for all age groups taken together as well as most specific age groups are lower in recessions. The same is true for mortality from cardiovascular diseases, pneumonia and influenza, motor vehicle accidents and suicides, but not for necessarily for other specific mortality causes. In particular, there is never a statistically significant effect on homicides, other external effects and malignant neoplasms. There are also few differences apparent between the effect on male and female mortality. If we do not control for state-specific effects, then we often arrive at the opposite result with higher unemployment being associated with higher mortality. This suggests that a failure to control for time-invariant state-specific effects leads to omitted variable bias, which would erroneously suggest that mortality rates move counter-cyclically. Overall, we can confirm Ruhm’s main finding for another country: recessions lower some, but not all, mortality rates in the case of Germany.

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## Introduction

The effect of short-term economic fluctuations on the state of health in general and mortality in particular has found the interest of scholars since Brenner’s (1973, 1975, 1979, 1987, 1995) path-breaking work. Brenner found with the help of time-series analysis that recessions are associated with deteriorating health in the United States, England and Wales and Sweden. Others have failed to find analogous evidence in a replication of his work on other countries or time-periods (Forbes & McGregor 1984; Wagstaff 1985; Joyce & Mocan 1993). The major advantage of Ruhm’s (2000) analysis of the

effect of unemployment rates on mortality rates in US states over the period 1972–1991 is the use of panel data. He finds that ‘state unemployment rates are *negatively* and significantly related to total mortality and eight of the ten specific causes of fatalities’ (Ruhm 2000, p. 617, emphasis in original). In other words, mortality behaves pro-cyclically as it moves with the business cycle. Panel data analysis has the advantage over time-series analysis that it can control for time-invariant state-specific effects and thus control for a potentially important source of omitted variable bias.

Gerdtham and Ruhm (2002) find similar evidence to Ruhm (2000) in pooling data from OECD countries. Ruhm (2001) himself has repeated his analysis on individual rather than aggregate data from the 1972–1981 US National Health Interview Surveys, coming to

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very similar conclusions as his analysis with aggregate data. In particular, he finds that the number of medical problems, the prevalence of acute morbidities and the number of reported “bed-days” all fall in economic recessions. Gerdtham and Johannesson (2002), on the other hand, find in their analysis of individual data from Sweden that some aspects of male mortality move counter-cyclically rather than pro-cyclically, whereas the business cycle is unrelated to female mortality. However, Tapia Granados (2003) in his analysis of the relationship between economic fluctuations and aggregate mortality in the 19th and 20th century does find a pro-cyclical movement in Swedish mortality rates. Tapia Granados (2002) finds similar results pooling data from the 50 Spanish provinces over the period 1980–1997.

The objective of this paper is to test the relationship between aggregate mortality and economic fluctuations for a different nation-state, namely Germany over the period 1980–2000. For Germany, no comprehensive individual data are available. Germany is an obvious candidate for an analysis of aggregate data, however, since similar to the US it is a federal nation-state, which allows panel data analysis, and as one nation-state, Germany is also more homogeneous in terms of population characteristics than a pool of nation-states, which helps to reduce omitted variable bias. We will show that economic recessions lower aggregate and some specific mortality rates, thus providing more support for Tapia Granados’ (2002, p. 41) contention that ‘the pro-cyclical character of mortality fluctuations is beginning to be a proven fact’.

Like Ruhm (2000) we also use fixed-effects estimation. At the same time, we improve on his analysis in a number of ways. First, we use standard errors, which are robust towards arbitrary heteroscedasticity and autocorrelation. Second, in sensitivity analysis we additionally make standard errors robust towards clustering such that observations are merely assumed to be independent across states, but not necessarily within states. Third, we use a dynamic model, which does not require the researcher to specify the number of time lags included in the model, a decision, which is always and by necessity somewhat arbitrary. Instead, we include the lagged dependent variable and correct for the correlation of the regressor with the error term with the help of Arellano and Bond’s (1991) generalized method of moments (GMM) estimator. Fourth, we look at gender-specific mortality rates to test for gender differences.

### **The impact of economic fluctuations on health**

There are many theories of the impact of economic fluctuations on health conditions. However, one can perhaps distinguish between two main perspectives. As

the main objective of this paper is an empirical analysis, we will merely sketch the arguments here. The reader is referred to Brenner and Mooney (1983), Watkins (1985), and Ruhm (2000) and the many references cited therein for a more extensive discussion. One perspective focuses on the social and psychological aspects of the hardship caused by economic downturns (see, for example, Watkins, 1985). The material losses associated with unemployment and the material insecurity for those who manage to stay in employment, but are at risk of losing their job in recessions, lower personal health-related expenditures and possibly lead to unhealthy diets. The stress, anxiety and psychological hardship connected to loss of job or fear of loss of job are also detrimental to health as affected individuals resort to medication, alcohol and other drugs to alleviate their stress and hardship. Novo, Hammarström, and Janlert (2001) report that employed young persons aged 21 report more somatic and psychological symptoms during economic downturn than during economic upturn. Unemployed people not only lose materially, they also potentially lose access to social networks, self-esteem, self-confidence, a scheduled life structure, a sense of identity and possibly a purpose for their lives (Brenner & Mooney, 1983; Watkins, 1985; Winkelmann & Winkelmann, 1998). There is evidence that people who become unemployed suffer from deteriorating mental and physical health and wellbeing (Warr, 1987; Jahoda, 1988; Wadsworth, Montgomery, & Bartley, 1999; Flatau, Galea, & Petridis, 2000).

The other perspective is derived from an explicitly economic model of utility maximization. In this model economic upturns can have negative and recessions can have positive effects on health for at least four major reasons: First, in economic upturns the opportunity costs of leisure time increase as individuals work more and gain more. As a consequence, less time will be spent on health-preserving activities and routine medical check-ups. Less time is available for cooking lower-calorie and better quality meals at home and more calorie-rich prepared food will be consumed (Chou, Grossman, & Saffer, 2002). Second, job-related stress will increase during periods of economic upturn, particularly if hours of working and work pressure are increased due to expanding economic activity. Whilst individuals might resort to increased tobacco use, alcohol, medication and drugs in times of economic downturn, they might equally well resort to the same means of seeming relief to cope with the stress of periods of economic expansions. Third, work-related accidents increase in periods of economic expansion (Tapia Granados, 2002). Some sectors, which tend to move pro-cyclically such as construction works, are particularly prone to high accident rates (Ruhm, 2002). Fourth, temporary increases in income due to economic expansion might increase the consumption of health-damaging

goods such as alcohol and tobacco (Freeman, 1999; Ruhm, 2002; Ruhm & Black, 2002).

Note that the two perspectives and the theories associated with them need not be inconsistent with each other. Instead, they could capture two different aspects of a complex impact of economic fluctuations on health and mortality. If the two effects are of approximately equal strength, then our empirical analysis will fail to find any statistically significant effect. If one effect is stronger than the other, then we will find an either positive or negative statistically significant effect. Even if this is the case, it does not imply that the other effect is non-existent. Instead, it merely means that one effect is so much stronger than the other that the overall effect goes into one direction.

## Research design

### *The dependent variables*

Like Ruhm (2000) we include total mortality for all age groups taken together as well as total mortality for three specific age groups (20–44, 45–64, older than 65) and ten specific mortality causes: malignant neoplasms (ICD 140–208), cardiovascular diseases (ICD 390–459), pneumonia and influenza (ICD 480–487), chronic liver diseases (ICD 571), motor vehicle accidents (E810–825), intentional self-damage (suicide) (E950–959), murder and manslaughter (homicide) (E960–969), other external effects (E800–809, E826–949), neonatal mortality (death within 28 days after birth) and infant mortality (death within the first year after birth). In extension to Ruhm (2000) we also look at gender-specific mortality rates to see whether there are any differences between the sexes. The data have been purchased from the German federal statistical office as a special analysis (“Sonderaufbereitung”) of their health statistical database. They have been transformed into mortality rates with state population data taken from Statistisches Bundesamt (various years). Codes refer to the ninth version of the International Classification of Diseases (ICD-9). Codes changed in the update to ICD-10, which took effect in the German health statistics in 1998, but the categories stayed the same. Our period of analysis covers the years 1980–2000. The ten specific mortality causes are responsible for between 73% and 88% of total mortality depending on the year, which is similar to the US case, where they account for around 80% of total mortality on average (Ruhm, 2000, p. 622).

Of course, as has been noted by many (Wagstaff, 1985; McAviney, 1988; Ruhm, 2000) it would be better if we could use morbidity rather than mortality data as mortality captures only the extreme fatal consequence of bad health. Unfortunately, however, no such data are available and we are therefore left with

mortality data. Similarly, in principle it would have also been better to use data of individuals rather than aggregates due to the so-called ‘ecological fallacy’ problem. However, no such comprehensive data are available for Germany. In its absence, aggregate data analysis is still informative, even if one needs to be careful in drawing conclusions from the aggregate level to individual behaviour (Gravelle, Wildman, & Sutton, 2002).

### *The independent variables*

Like Ruhm (2000) we take the state unemployment rate as our main indicator of economic fluctuations. It is also the indicator most often used by researchers (Watkins, 1985). In sensitivity analysis, we use the growth rate in real GDP as an alternative indicator. As further control variables, we use personal available income per capita in real prices of 1995.<sup>1</sup> Presumably, health care is a normal good such that all other things equal mortality rates should go down if real incomes rise as individuals will spend more money on health-preserving investments. There is also substantial empirical evidence for this (Pritchett & Summers, 1996; Ettner, 1996).<sup>2</sup> On the other hand, many health-damaging consumption expenditures such as drinking and smoking are also likely to be normal goods (Freeman, 1999; Ruhm & Black, 2002). The effect of the average income level on aggregate mortality rates is therefore ambiguous. Note that this does not contradict the empirical evidence of cross-sectional studies showing that low-income social classes have higher mortality rates than high-income social classes at any moment of time (see, for example, Townsend, Davidson, & Whitehead, 1992).

Both real GDP and nominal personally available income data are taken from Statistisches Landesamt Baden-Württemberg (2002), which were converted into real terms using the German GDP deflator. Like Ruhm (2000), in order to control for differences in age structure, we take the percentage of the population under 5 years as well as those aged 65 or over as two further control variables. Ruhm (2000) further uses the percentage of population that is black or Hispanic as these ethnic minorities are likely to have higher mortality rates. The closest we can have in the German

<sup>1</sup> Gravelle, Hutchinson, and Stern (1981) argued against this variable, which was also used by Brenner (1979), saying that it ignores public health-relevant expenditure and that therefore gross domestic product (GDP) per capita might be the more relevant variable. Our results do not change if personal income is replaced by GDP.

<sup>2</sup> Note that this would not contradict the hypothesized negative impact of economic upturns on mortality as one needs to distinguish short-term perturbations from long-term income trends.

case is the percentage of foreigners among the total population. All these population characteristics data are taken from [Statistisches Bundesamt \(various years\)](#). The only control variables included in [Ruhm \(2000\)](#), for which we have no equivalent, are the ones capturing the status of education. Whilst such data exist in principle for some years in the so-called German microcensus, they have been aggregated from the individual to state aggregates only for the years 1999 and 2000. Whilst regrettable, the lack of such variables are likely to cause concern only if we believe that the state unemployment rate is strongly correlated with the omitted variables of education status. We have no reason to believe this is the case. Education status is relatively homogenous across German states due to free access to public schools and universities, the quality of which varies only little across states and time.

As a further control variable we include the Gini coefficient as a measure of income inequality. Such data are only available from 1985 onwards, which is why this variable is included only in sensitivity analysis. Data were kindly provided by Peter Krause from the German Institute for Economic Research, Berlin.

#### *The estimation technique*

We will estimate both a static model, with contemporaneous effects only, and a dynamic model, which allows for lagged effects of unemployment on mortality. In a static context, we estimate the following panel data model:

$$y_{it} = \lambda + \beta_1 \chi_{it} + \gamma_t + \varepsilon_{it}, \quad \text{where } \varepsilon_{it} = u_i + v_{it}, \quad (1)$$

where the subscript  $t$  stands for time and  $i$  for each state. The  $y$  is the (logged) mortality rate, the matrix  $x$  contains the explanatory variables, including the state unemployment rate. There are  $(t-1)$  year-specific dummy variables, which capture decreases in mortality rates over time due to, for example, improved health technology, healthier lifestyles and so on. The  $u_i$  are unobserved state-specific time-invariant fixed effects and  $v_{it}$  is a stochastic error term.

There are basically two estimators available for estimating Eq. (1), namely the fixed-effects and random-effects estimator. The fixed-effects estimator subtracts from the equation to be estimated, the over-time average of the equation for each state. Because of this so-called within transformation the individual state-specific fixed effects  $u_i$  are wiped out and the coefficients are estimated based on the time variation within each cross-sectional unit. The random-effects estimator, on the other hand, is based on the assumption that the fixed effects are random effects such that they can be included in the stochastic error term.

The big advantage of the fixed-effects estimator is that any potential correlation of the explanatory variables

with the fixed effects is avoided since the fixed effects and therefore their correlation with the explanatory variables are wiped out from the equation to be estimated. As a consequence, the estimation is unbiased even if the explanatory variables are correlated with the unobserved state-specific time-invariant effects as is likely to be the case here—one of the reasons to prefer fixed-effects over random-effects estimation. Also, given that our sample covers all states of a country rather than a random sample of it, the fixed-effects specification is the more natural one. We therefore use the fixed-effects estimator.

Like [Ruhm \(2000\)](#) we will weight observations by the square root of the state population in order to mitigate potential heteroscedasticity problems. In extension to [Ruhm \(2000\)](#), we also employ standard errors, which are robust towards arbitrary autocorrelation (as well as heteroscedasticity). It is not quite clear, why [Ruhm](#) did not use robust standard errors. Given that his panel is cross-sectionally dominated since his data draw from many more states than our analysis, he is perhaps less concerned about autocorrelation than we are.

As a next step, we will allow for a lagged effect of unemployment on mortality as well, using a dynamic econometric model. There are two basic ways to account for such lagged effects of the explanatory variable, namely via finite distributed lag (FDL) or infinite distributed lag (IDL) models. The FDL model assumes that the explanatory variable impacts upon the dependent variable over a finite time period. The simplest way to account for this is to include both the contemporaneous explanatory variable and a number of lags of this variable. The problem with this approach is the high multicollinearity amongst the lagged variables and the need to choose how many lags are included. Imposing a polynomial structure on the lagged variables such that the effect is declining linearly (polynomial of order one) or non-linearly (polynomial of second order or higher) circumvents the multicollinearity problem ([Hill, Griffiths, & Judge, 1997](#)). It does leave the researcher with the problem of choosing the correct lag length, however. [Brenner \(1979\)](#), for example, chose a 10-year lag. [Ruhm \(2000, p. 634\)](#) uses a 4-year lag because the coefficient on the fifth lag in the total mortality estimation was insignificant at the 95% level. Such a decision rule is of course essentially arbitrary and therefore problematic.

In the IDL model no lag length needs to be chosen, as by definition an infinite number of lags is included. The IDL model can be written as

$$y_t = \alpha + \sum_{j=0}^{\infty} \beta_j \chi_{t-j} + e_t. \quad (2)$$

Note that for simplicity and for the time being we ignore the fact that we have panel data and we look at a

pure time-series problem. Later on, we will revert back to our panel data context. Clearly, in its general form, Eq. (2) cannot be estimated as it implies an infinite number of coefficients to be estimated. It turns out, however, that similar to the FDL model, the problem can be circumvented if some structure is imposed on the lags. Koyck (1954) showed that with a geometric lag structure, Eq. (2) can be transformed into the following model

$$y_t = \lambda + \beta_1 y_{t-1} + \beta_2 \chi_t + \varepsilon_t. \quad (3)$$

If we put Eq. (3) into a panel data context then it is written more generally as follows:

$$y_{it} = \lambda + \beta_1 y_{it-1} + \beta_2 \chi_{it} + \gamma_i T_i + \varepsilon_{it}, \text{ where } \varepsilon_{it} = u_i + v_{it}. \quad (4)$$

The short-run or contemporaneous effect of the explanatory variable on the dependent variable is simply given by  $\beta_2$ , whereas the long-run effect can be computed as  $\beta_2/(1 - \beta_1)$ . As Eq. (4) is estimated for each mortality cause, the lag with which unemployment affects mortality can of course differ from cause to cause.

Estimation of Eq. (6) with either ordinary least squares (OLS) or a fixed-effect or a first-differenced panel estimator is problematic. This is because of the inclusion of the lagged dependent variable as a regressor. Since  $y_{it}$  is a function of  $u_i$ , so is  $y_{it-1}$ . The correlation of a regressor with the error term renders the OLS estimator both biased and inconsistent. The same is true for the fixed-effect or first-differenced estimator. Whilst in the process of estimation the  $u_i$  are wiped out, biasedness and inconsistency is a consequence of the correlation between  $y_{it-1}$  and  $v_{it-1}$  (Baltagi, 1995, p. 126).

There are two ways to estimate Eq. (6) without bias and consistently. One is to follow Anderson and Hsiao (1981) and to use a two-stage least squares (2SLS) first-differenced estimator, that is, a first-differenced estimator with instrumental variables. First-differencing wipes out the  $u_i$  and using either  $y_{it-2}$  or  $\Delta y_{it-2}$  (that is,  $y_{it-2} - y_{it-3}$ ) as an instrument for  $y_{it-1}$  solves the problem since neither instrument is correlated with  $\Delta y_{it}$ . In addition, further lags can be included. Alternatively, one can use the so-called Arellano and Bond (1991) GMM estimator. The basic idea of this estimator is to use all prior dependent variables that are valid instruments, not just  $y_{it-2}$ . We will use the Arellano and Bond dynamic panel estimator as it is more efficient than the 2SLS first-differenced estimator together with heteroscedasticity-robust standard errors.

### The sample

The sample consists of all 11 Western German states until 1990 and all 16 German states from 1991 onwards, with Western Berlin becoming Berlin after re-unifica-

tion. We thus have a sample of 281 observations. Note that in using Arellano and Bond's (1991) GMM estimator, we lose the first 2 years of data as the lagged dependent variable is one of the explanatory variables and needs to be instrumented for with a further lag. Ruhm (2000) provides estimates with and without year-specific dummy variables as well as with and without available income per capita included. There is little justification for estimates without year-specific effects and without income per capita. The year-specific effects capture exogenous changes in the mortality rate unrelated to our explanatory variables, e.g. progress in life-saving medical technology, and excluding income per capita might lead to omitted variable bias. Hence we only report estimations with both included, but our results do not change much if we apply one of the other specifications.<sup>3</sup> We take the natural log of the dependent variable to render its distribution less skewed and to allow an easy to understand elasticity interpretation of the results. However, our results do not change much if the estimate is in levels of the dependent variable, rather than in natural logs.

### Results

We start with the static model with contemporaneous effects only. Table 1 presents results for aggregate mortality rates for all age groups, specific age groups as well as for both sexes. Note that for simplicity, only the coefficients of the unemployment rate is shown, whereas the coefficients of the other control variables and the year-specific time dummies are suppressed. We will come back to the other control variables further below. We can see that the unemployment rate is negatively and statistically significantly related to the overall mortality rate for all age groups taken together as well as to the mortality rate for all specific age groups and both sexes. Next, we look at specific mortality causes. The unemployment rate is negatively associated with mortality from cardiovascular diseases as well as suicide for males, females and both sexes taken together. A similar effect is observable for mortality from motor vehicle accidents for both sexes taken together and females, but not for males. Combined sex as well as male mortality from pneumonia and influenza is negatively associated to the unemployment rate, but not in the case of females. Similar to Ruhm (2000) we find no statistically significant impact of unemployment on deaths from

<sup>3</sup> Contrary to Ruhm (2000) we do not include state-specific time trends in some estimations or run the estimations for certain sub-groups of states. The reason is that we have a much smaller panel than Ruhm (2000) who can draw upon data from 50 states and if we replicate such analysis for 11–16 states, not surprisingly we get highly inefficient results.



Table 1

Static fixed-effects estimation results of the effect of the unemployment rate on mortality

	Both sexes	Female	Male
Mortality, all ages	–0.0110*** (4.16)	–0.0127*** (4.31)	–0.0091*** (3.79)
Mortality, 20–45 years	–0.0110*** (2.96)		
Mortality, 45–65 years	–0.0054* (2.16)		
Mortality, 65 years and older	–0.0124*** (4.19)		
Malignant neoplasms	–0.0013 (0.44)	–0.0026 (0.82)	0.0001 (0.03)
Cardiovascular diseases	–0.0175*** (5.37)	–0.0175*** (4.90)	–0.0177*** (5.66)
Pneumonia and influenza	–0.0307* (1.85)	–0.0258 (1.48)	–0.0365** (2.23)
Chronic liver diseases	0.0042 (0.67)	0.0021 (0.30)	0.0037 (0.51)
Motor vehicle accidents	–0.0131** (1.98)	–0.0205*** (2.63)	–0.0082 (1.01)
Suicide	–0.0138** (2.47)	–0.0190** (2.39)	–0.0098* (1.64)
Homicide	0.0032 (0.23)	0.0123 (0.62)	–0.0028 (0.16)
Other external effects	0.0168 (1.40)	0.0242 (1.50)	0.0102 (1.00)
Infant mortality	0.0018 (0.27)		
Neonatal mortality	–0.0193 (0.53)		

Note:  $N = 281$ . Coefficients of control variables and year-specific dummies not shown. Robust standard errors. Absolute  $t$ -values in parentheses.

\*Statistically significant at 0.1 level, \*\*0.05 level, \*\*\*0.01 level.

malignant neoplasms. Contrary to Ruhm (2000), however, is our result that mortality from liver diseases, homicide, other external effects as well as the infant and neonatal mortality rate are all statistically independent from the unemployment rate.<sup>4</sup> Contrary to Ruhm (2000) is also our result that mortality from suicide behaves pro-cyclically as this was the only mortality cause, for which he found a counter-cyclical behaviour.

Estimates from the dynamic model, for which results are reported in Table 2, are rather similar to the results from the static model in terms of sign of coefficient and statistical significance of the unemployment rate.<sup>5</sup> The

<sup>4</sup>Note that for the infant and neonatal mortality rate no gender-specific data are available.

<sup>5</sup>To save space, we suppress the estimates of the lagged dependent variable, the other control variables as well as the year-specific time dummies. For technical reasons, as explained in Arellano and Bond (1991, 281f), the GMM estimator becomes inconsistent in the presence of second-order autocorrelation. The relevant test fails to reject the hypothesis of no second-order autocorrelation at the 0.05 level in all regressions,

aggregate mortality rate for all age groups taken together is negatively associated with the unemployment rate as in the static model. The same is true for female mortality as well as mortality in the age groups 20–45 and older than 65, but the unemployment rate is highly insignificant for the age group 45–65 and for male mortality. Note, however, that in the latter case the unemployment rate remains statistically significant with a negative coefficient if the total male mortality is replaced by the sum of all specific mortality causes for males looked at here. As concerns these specific mortality causes, cardiovascular diseases are pro-cyclical for both sexes individually as well as taken together. The same is true for mortality from motor vehicle accidents, but as with the static model the unemployment rate is insignificant for males. Pneumonia and influenza as well as suicide are pro-cyclical for both sexes and females,

(footnote continued)

but mortality of people 65 years and older, aggregate female mortality as well as male mortality from suicides.

Table 2

Dynamic estimation results of the effect of the unemployment rate on mortality (GMM first-differenced)

	Both sexes	Female	Male
Mortality, all ages	−0.0059** (2.22)	−0.0078** (2.51)	−0.0034 (1.17)
Mortality, 20–45 years	−0.0103** (2.47)		
Mortality, 45–65 years	−0.0011 (0.41)		
Mortality, 65 years and older	−0.0073*** (2.90)		
Malignant neoplasms	−0.0014 (0.49)	−0.0041 (1.11)	0.0015 (0.49)
Cardiovascular diseases	−0.0093** (2.22)	−0.0095*** (3.25)	−0.0123*** (2.78)
Pneumonia and influenza	−0.0305* (1.74)	−0.0338** (2.08)	−0.0166 (0.75)
Chronic liver diseases	−0.0144 (1.44)	−0.0118 (1.00)	−0.0138* (1.71)
Motor vehicle accidents	−0.0122** (2.19)	−0.0199** (2.09)	−0.0108 (1.30)
Suicide	−0.0126*** (4.06)	−0.0197*** (2.57)	−0.0122 (1.24)
Homicide	0.0100 (0.78)	0.0037 (0.15)	0.0055 (0.33)
Other external effects	0.0011 (0.15)	0.0012 (0.08)	−0.0004 (0.05)
Infant mortality	−0.0002 (0.16)		
Neonatal mortality	0.0052 (0.13)		

Note:  $N = 254$ . Coefficients of the lagged dependent variable, control variables and year-specific dummies not shown. Absolute  $z$ -values in parentheses. Robust standard errors.

\*Statistically significant at 0.1 level, \*\*0.05 level, \*\*\*0.01 level.

but no longer for males. Deaths from malignant neoplasms, murder and manslaughter and other external effects as well as the infant and neonatal mortality rate are statistically independent from the unemployment rate. So is mortality from liver diseases for females and both sexes together, but in a dynamic context this mortality cause moves pro-cyclically for males.

What about the other control variables? Similar to [Ruhm \(2000\)](#), we find that the income variable is sometimes estimated with a positive significant coefficient, sometimes with a significant negative coefficient and is often found to be insignificant.<sup>6</sup> Higher income is associated with lower mortality from pneumonia and influenza, but higher total mortality as well as mortality

from cardiovascular diseases, malignant neoplasms and other external effects. For cardiovascular diseases, it seems likely that the increased demand for health-damaging goods such as excessive food, tobacco, alcohol and drugs following income increases more than compensates the effect of higher expenditures into health-preserving investments. In terms of aggregate mortality rates according to age groups, we find that higher income levels are not associated with mortality rates for the 20–45 year olds, are negatively associated with mortality rates for the 45–65 year olds and positively associated for mortality rates of those 65 years and older.<sup>7</sup>

<sup>6</sup>Our main results do not change if, following [Gravelle et al. \(2002\)](#), we add a squared income term to account for a potentially non-linear effect of income on mortality. We find no evidence for such a non-linear effect and the estimated coefficient of our unemployment variable is hardly affected.

<sup>7</sup>Interestingly, [Snyder and Evans \(2002\)](#) also find that individuals with higher retirement benefits due to a slightly earlier date of birth have higher mortality than those with lower retirement benefits due to a slightly later date of birth in their analysis of the so-called “notch” in US Social Security benefits based upon date of birth.

The population characteristic variables referring to age groups by and large test according to expectation. For example, a higher share of old people increases mortality in the case of cardiovascular diseases, malignant neoplasms, but decreases mortality in the case of other external effects and homicide. The share of foreigners is generally insignificant, apart from mortality from other external effects, for which a higher share of foreigners is associated with higher mortality. This is different from [Ruhm \(2000\)](#) who found more consistently significant effects for the share of blacks and hispanics. The reason for this difference is likely to be found in Germany's universal health coverage, which ensures that all foreigners also have full access to medication and medical treatment similar to Germans, whereas the health of ethnic minorities in the US often suffers from their inadequate access to health insurance.

### Sensitivity analysis

We will now test the sensitivity of our results with regards to model specification. For reasons of space, we will merely describe but not report detailed results, which are available from the author upon request, however. To start with, we include the Gini coefficient as a further control variable. As mentioned above, this variable is only available from 1985 onwards. Our results are hardly affected. The coefficient of the income inequality variable itself often has a positive sign, but it is never statistically significant. This is in accordance with other studies finding no robust evidence that income inequality is statistically significantly related to aggregate health ([Gravelle et al., 2002](#)).

So far, we have made standard errors merely robust towards arbitrary heteroscedasticity and autocorrelation. Going one step further, one can make standard errors also robust towards the clustering of observations. That is, observations are merely assumed to be independent across states, but not necessarily within states. One of the problems associated with making standards errors robust to clustering is the consequent loss in degrees of freedom, which means that the standard errors typically become larger and our estimations less precise. The major differences are that the total mortality rate between the ages 45 and 65, the traffic mortality rate and the mortality rate from pneumonia and influenza for both sexes as well as the male suicide rate become insignificant. Still, our main results are upheld. In particular, total mortality as well as mortality for other age groups, gender-specific mortality as well as the important mortality cause cardiovascular disease all move counter-cyclically.

To see whether German re-unification might represent a structural break causing problems for our estimations, we simply repeated our analysis of [Table 1](#) for the post-

reunification period only. Clearly, with a much shorter panel estimations become less precise and standard errors become higher. And yet, we still find that overall mortality rates and mortality from cardiovascular disease move counter-cyclically.

[Brenner \(1995, p. 227\)](#) emphasizes that 'such variables as unemployment (...) are typically associated with increase in mortality rates 2–3 years following the lowest point in the business cycle...'. We have therefore replaced the contemporaneous unemployment variable in our static model with the unemployment rate lagged by 3 years. No positive association between the unemployment rate and mortality rates as suggested by Brenner is apparent. Indeed, in accordance with our dynamic estimation results we find the unemployment rate lagged by 3 years often to be negatively associated with mortality rates.

How sensitive are our results towards the indicator of economic fluctuations used? If we replace the unemployment rate with the growth rate in real GDP, then our results are generally rather similar. In particular, the aggregate mortality rate for all age groups taken together, for all specific age groups as well as mortality from cardiovascular diseases for both sexes are positively associated to changes in GDP.<sup>8</sup> The major difference is that pneumonia and influenza as well as suicide no longer react pro-cyclically. The infant and neonatal mortality rates, on the other hand, move pro-cyclically with changes in real GDP.

### Discussion

We have seen that economic downturns are associated with higher mortality in German states similar to the pattern observed by [Ruhm \(2000\)](#) for the states of the United States of America. The effect is statistically significant for aggregate mortality rates for all age groups taken together, all specific age groups and for both males and females. This is by and large true independent of whether the static or dynamic model is estimated and independent of whether the indicator for economic fluctuations is the unemployment rate or the real GDP growth rate. How strong is the effect? [Table 3](#) summarizes by how many percent the mortality rate decreases following a one percentage point increase in the unemployment rate in the static and the dynamic model.<sup>9</sup> Like [Ruhm \(2000\)](#) we find that the effect of unemployment on mortality is weakest in the age group

<sup>8</sup>Only in the dynamic model, the change in GDP variable becomes marginally insignificant for the age group 65 and older.

<sup>9</sup>Note that the estimated effect for the male mortality rate in dynamic estimation refers to the sum of specific mortality rates looked at here as the total mortality rate tested insignificantly.



Table 3

Percentage change in mortality rate due to one percentage point increase in state unemployment rate

	Static			Dynamic (short-run)			Dynamic (long-run)		
	Both sexes	Female	Male	Both sexes	Female	Male	Both sexes	Female	Male
Mortality, all ages	−1.10%	−1.27%	−0.91%	−0.59%	−0.78%	−0.55%	−0.79%	−1.09%	−0.69%
Mortality, 20–45 years	−1.10%			−1.03%			−1.29%		
Mortality, 45–65 years	−0.54%			n.s.			n.s.		
Mortality, 65 years and older	−1.24%			−0.73%			−1.11%		
Cardiovascular diseases	−1.75%	−1.75%	−1.77%	−0.93%	−0.95%	−1.23%	−1.43%	−1.51%	−1.80%
Pneumonia and influenza	−3.07%	n.s.	−3.65%	−3.05%	−3.38%	n.s.	−6.46%	−6.96%	n.s.
Chronic liver diseases	n.s.	n.s.	n.s.	n.s.	n.s.	−1.38%	n.s.	n.s.	−2.50%
Motor vehicle accidents	−1.31%	−2.05%	n.s.	−1.22%	−1.99%	n.s.	−1.63%	−2.02%	n.s.
Suicide	−1.38%	−1.90%	−0.98%	−1.26%	−1.97%	n.s.	−1.78%	−2.63%	n.s.

Note: n.s., not significant.

45–65. Contrary to [Ruhm \(2000\)](#) who found the effect to be by far strongest for the age group 20–44, both our static and dynamic effects show about the same effect for this and the age group 65 and older. It is not surprising that young adults are strongly affected by economic conditions given their high labour force participation, but the strength of the effect for the retired people is somewhat unexpected. With respect to the specific mortality causes, the effect of the unemployment rate is about three times stronger on mortality from pneumonia and influenza than for the other causes, whereas the size of the effect for the other mortality causes is approximately similar. This is also contrary to [Ruhm \(2000\)](#) who found the strongest effects with respect to traffic accidents, other external effects and homicides. What is comforting is that similar to [Ruhm \(2000\)](#) we find no significant effect of the unemployment rate on mortality from malignant neoplasms. It would be extremely disturbing if short-run economic fluctuations would impact significantly upon such a disease with a rather long-term determination.

Looking across the sexes, there are few differences apparent. No sex is clearly more consistently or more strongly affected by economic fluctuations than the other one. Lastly, [Table 3](#) shows that the main effect of economic fluctuations on mortality seems to occur in the short run as the long-run effect is often not that much bigger than the short-run effect. That the unemployment rate is less consistently and often less clearly statistically significantly negatively related to mortality is a consequence of the relative inefficiency of the dynamic GMM estimator. Whilst instrumenting the lagged dependent variable avoids its correlation with the error term, the estimation can be inefficient if the instruments are weak. In other words, estimation is inefficient if further lags are bad predictors of the lagged dependent variables ([Wooldridge, 2002](#)). In our case, further lags are actually quite good predictors, but still some loss of efficiency is often unavoidable if instruments need to be used.

Table 4

Static estimation results of the effect of the unemployment rate on mortality (OLS without fixed-effects)

	Both sexes
Mortality, all ages	0.0092*** (4.78)
Mortality, 20–45 years	0.0210*** (6.13)
Mortality, 45–65 years	0.0299*** (15.32)
Mortality, 65 years and older	0.0037* (1.74)
Malignant neoplasms	0.0125*** (7.42)
Cardiovascular diseases	0.0026 (0.94)
Pneumonia and influenza	−0.0199** (2.20)
Chronic liver diseases	0.0125*** (2.67)
Motor vehicle accidents	−0.0585*** (6.92)
Suicide	−0.0161*** (2.67)
Homicide	0.0185* (1.80)
Other external effects	−0.0097 (0.90)
Infant mortality	0.0256*** (6.01)
Neonatal mortality	0.0137 (0.54)

Note:  $N = 281$ . Coefficients of control variables and year-specific dummies not shown. Robust standard errors. Absolute  $t$ -values in parentheses.

\*Statistically significant at 0.1 level, \*\*0.05 level, \*\*\*0.01 level.

What is the reason why [Ruhm \(2000\)](#) and our own analysis find that mortality moves pro-cyclically, whereas [Brenner's](#) analysis and those of others found a

counter-cyclical movement? The main contribution of [Ruhm \(2000\)](#) to the topic was the use of a fixed effects estimator, which ensures that the estimations do not suffer from omitted variable bias from variables, which are time-invariant. To see how a failure to control for fixed effects can lead to misleading results, [Table 4](#) presents OLS estimates of Eq. (1) without state-specific fixed effects included. The effect of unemployment rates on mortality is completely reversed for aggregate mortality for all ages and all specific age groups. A higher unemployment rate is now associated with higher mortality! The same is true for many specific mortality causes such as malignant neoplasms, liver diseases, homicides and the infant mortality rate. Only mortality from pneumonia and influenza, suicides as well as motor vehicle accidents still moves pro-cyclically. We therefore see how a failure to control for state-specific effects can erroneously suggest that aggregate mortality as well as many specific mortality causes move counter-cyclically.

## Conclusion

All in all, we have found confirmation for [Ruhm's \(2000\)](#) general result in our analysis of German states: recessions tend to lower mortality rates. We find consistent and robust evidence that recessions lower aggregate mortality rates for all age groups taken together as well as all specific age groups. We find less consistent evidence for specific mortality causes than [Ruhm \(2000\)](#) did. For both sexes taken together, we find the predicted effect for cardiovascular diseases, pneumonia and influenza, motor vehicle accidents and suicides in both static and dynamic model estimations. These mortality causes make up together between 43% and 66% of total mortality, with cardiovascular diseases being by far the most important one. However, contrary to [Ruhm \(2000\)](#) we find no statistically significant negative effect for liver diseases and other external effects. The same is true for infant and neonatal mortality, unless we replace the unemployment rate with the real GDP growth rate as our indicator of economic fluctuations. We also find that the size of the impact compared across age groups and specific mortality causes differs from the ones found by [Ruhm \(2000\)](#). The reasons for these differences are not quite clear. It could be that our estimations are less efficient given that we have only data for 11 (from 1991 onwards: 16) states available, whereas [Ruhm \(2000\)](#) can draw from a much bigger sample. Alternatively, the relationship between economic fluctuations and mortality could also be different in Germany due to socio-economic or other factors not captured in our estimations.

It is important to be aware of the caveats in interpreting our results. Whilst we show that overall mortality rates as well as some specific rates are counter-

cyclical, this does not automatically imply that the average health status is also counter-cyclical. It is perfectly possible that a recession lowers the mortality risk for some individuals whilst worsening the health status of the majority of other individuals, but short of increased mortality. One reason for this could be the negative health effects of recessions for the unemployed themselves. [Ruhm's \(2000\)](#) and our results as well as that of others therefore need not contradict the evidence for the negative effects of unemployment on some health aspects for at least some people.

This leads us to the policy implications of our result that economic upturns are associated with greater population mortality and vice versa for economic downturns. As [Tapia Granados \(2002, p. 41\)](#) points out, Brenner's work is often invoked as additional evidence for expansionary economic policies. Surely, the implication of our opposite findings cannot be that recessions are desirable even if, statistically speaking, they lower mortality rates. Instead, we need to focus on how the negative impact of economic upturns on mortality rates can be mitigated, if not avoided. There has been much focus on the negative health implications of economic downturns and rightly so. Maybe what we need is a similar focus on the negative health implications in terms of increased mortality rates in times of economic upturns.

For this we need to better understand the mechanisms that link economic upturns to health deterioration. [Ruhm \(2002\)](#) and [Ruhm and Black \(2002\)](#) could estimate the impact of economic conditions on such health-relevant activities as smoking and drinking habits, and physical exercise as well as various measures of height-adjusted weight from individual state-specific data. They show that lifestyles tend to become healthier in recessions: individuals smoke and drink less, are less severely obese and are more physically active. Unfortunately, for Germany no such individual data were available. In any case, more research is needed into why the positive impacts of recessions on mortality more than compensate the well documented negative health effects for certain sub-groups of the population, particularly the unemployed.

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