Are Recessions Harmful to Health After All? Evidence from the European Union

By

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Discussion Paper 2007-18
March 2007

Editor: Dr W David McCausland
www.abdn.ac.uk/business/
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Abstract: This paper investigates the effects of national unemployment rates on overall age and cause-specific mortality rates in a panel sample of 13 European Union countries. A fixed-effects model is used to control for unobserved time-invariant characteristics within countries. In addition, controls such as lifestyle risk factors, urbanisation and medical intervention indicators, for potential confounders are used. Contrary to some recent evidence this study shows that there is a strong, positive relationship between adverse economic conditions and the mortality.

JEL Classification Code: C23, C33, E24, I12

Keywords: Mortality, Unemployment, Fixed-Effects models

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1 The authors wish to thank the workshop participants of the SOCIOLD project and David McCausland for helpful comments. The financial support of the European Commission under the Fifth Framework Programme “Quality of Life and Management of Living Resources” (contract number: QLRT-2001-02292) is gratefully acknowledged.

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INTRODUCTION

The majority of studies that investigated the link between unemployment and health status, from 1960 onwards, provided consistent evidence that unemployment has a positive effect on mortality rates, i.e. increases in unemployment increase mortality. It was thought that high unemployment rates to not only threaten the social and economic cohesion, through their negative financial consequences on individuals, but also to be a risk factor for population health and thus mortality rates. However, recently, Ruhm (2000, 2003) contradicted conventional wisdom using U.S. data and suggested that recessions and thus high unemployment rates are beneficial to population health. He argues that in recessions health improves as individuals both improve their dietary habits and reduce lifestyle habits detrimental to health such as smoking and drinking.

This study revisits this issue questioning the universality of Ruhm’s recent results. It uses a sample of thirteen European Union countries. Following Ruhm (2000), it uses fixed effects models to control for unobserved, time-invariant characteristics within each country that are spuriously correlated with economic conditions across countries. Aggregate-level studies have been criticised for not being able to reveal the true relationship, because, at the population-level, other confounding factors exist which mediate in the relationship of interest namely the unemployment–mortality relationship. An innovation of this study is that it takes into account the confounding factors directly in order to overcome problems associated with confounding. It thus includes in the fixed effects models indicators of lifestyle factors such as smoking and drinking, dietary habits, medical intervention and the like.
OVERVIEW OF THE LITERATURE

Though the significant increase in life expectancy and the associated reduction in mortality rates are subject to multiple causes, nutrition, technological advancement and medical intervention, effective public health care services and rising living standards are commonly considered to be largely responsible for these gains in longevity (Illsley, 1990; Sans et al., 1997). However, longevity also appears to be affected by a number of socioeconomic factors. Specifically, unemployment has been found to negatively affect certain facets of health, ranging from psychological health status (Theodossiou, 1998) to physical symptoms and mortality (Gerdtham and Johannesson, 2003).

An issue of importance in investigating the unemployment-mortality relationship is the identification of the mechanisms that link a social phenomenon such as unemployment to a biological process such as health and mortality. In doing so, this section first briefly reviews recent studies which investigate the effect of unemployment rates on mortality.

Unemployment and mortality

A number of studies reviewed the existing evidence regarding the direct link and the multiple latent pathways that intervene in the unemployment-mortality relationship. Unemployment can be detrimental to the individual’s standard of living and financial resources. Restricted financial resources can lead to poor nutrition, and probable restriction to access to medical health care when needed. This may cause increased physical morbidity and even death.

Indeed, Junankar (1991) found a positive relationship between unemployment and mortality and Gerdtham and Johannesson (2003) found that unemployment is a health hazard. It increases the risk of dying by approximately 50%, even after controlling for income. Martikainen and Valkonen (1996) argued that individuals who experience unemployment were found to exhibit greater mortality rates compared to their employed counterparts, after controlling for demographic and SES indicators. Morris et al. (1994) showed that not only unemployment experience, but also the duration of unemployment spells increase the risk of dying after controlling for potential
confounders such as age, race, marriage, income, and occupational class. A number of studies found an age difference in mortality by showing that mortality was excessive for middle-aged unemployed men compared to their employed counterparts. Importantly, Junankar (1991), Ungváry et al. (1999), and Moser et al. (1986) point out the negative externalities that the families of the unemployed individual face. Importantly, the latter study showed that the wives of unemployed men have an increased risk of mortality. Yet, unemployment exerts a stronger effect on the mortality of men than of women. This is in line with Theodossiou (1998) who found a differential effect of unemployment on the health of men and women.

Interestingly, many studies have shown that health inequalities differ according to age. Ungváry et al. (1999) in a review of relevant studies showed that mortality of unemployed middle-aged men is more sensitive to unemployment compared to other age groups. Health inequalities due to unemployment by age were also found by Theodossiou (1998).

Snyder and Evans (2002) contradicted the earlier literature by finding that a higher income group (aged over 65 years) experience a higher mortality rate compared to a younger (by few months to one year) cohort. They also found that the younger cohort responded to lower incomes by working in part-time, post-retirement jobs. They interpreted this finding as highlighting the protective effect of moderate employment, which cancels out the feelings of exclusion, and social isolation that the elderly individuals may experience after retirement.

Ruhm (2000) reexamined the issue of the unemployment-mortality relationship by applying a fixed-effects model in a state-level study for US. His findings strongly contradicted the literature reviewed above. The results showed that economic upturns, by decreasing the unemployment rate, have a negative effect on physical health; contrary to the notion that unemployment worsens health, physically and mentally. Ruhm argues that there are at least three reasons as to why increasing employment rates negatively affect individual health: (i) non-market leisure decreases, reducing the possibility of health enhancing activities such as exercise; (ii) health could be considered as “an input” into the generation of services and products. Thus, due to a greater requirement of inputs in a growing economy, there should be an increase in
hazardous working conditions, physical exertion and in working hours; (iii) the availability of higher income increases the propensity of individuals to take risks and to indulge in activities such as smoking drinking and excessive eating of high fat diets. The main conclusion was that during economic downturns, as approximated by the unemployment rate, both overall and cause-specific\(^3\) mortality rates fall. The mortality rates used in the study were death rates from cardiovascular disease, influenza/pneumonia, motor vehicle fatalities and other accidents. Subsequent studies by Gerdtham and Ruhm (2003) and Neumayer (forthcoming) confirmed the Ruhm (2000) findings.

All in all, the above review of the recent research highlights a remarkable lack of consistency regarding both the sign of the unemployment-mortality relationship and the mechanisms that link these two phenomena. The present study attempts to shed some further light and reexamine this issue in an attempt to reach some conclusion. In doing so it takes into account directly the effects of confounding factors that mediate in the above relationship. It thus control for some important indicators of lifestyle factors such as smoking and drinking, dietary habits, medical intervention and the like.

Confounding factors and the Unemployment-Mortality relationship

Research has shown that confounding factors mediate in the economic conditions/unemployment-mortality relationship. Thus, in order that the true effects of economic conditions or unemployment on mortality be revealed the mediating confounding factors should be taken into account.

Some lifestyle behaviours promote health, while others are detrimental to it. For instance, medical research shows that smoking is responsible for a significant share in the incidence of heart diseases and cancer. Often, unemployment is associated with the adoption of unhealthy lifestyles such as increased smoking. Stern (1983) argues that this link is probably established due to the increased psychological “burden” and the stress the unemployed individuals feel. Wood et al. (1999) argued that people of lower SES are significantly more likely to adopt a sedentary lifestyle, to be overweight, and to smoke. Morris et al. (1992) found that increased bodyweight was associated with unemployment. Other studies showed that smoking and drinking is
heavier and nutrition is worse among the unemployed. On the contrary, Ruhm (2000, 2003) argues that unemployment is inversely related to smoking and obesity.

Suicide and self-inflicted injuries are leading causes of injury-related deaths worldwide (Chisiti et al., 2003). High levels of unemployment rates seem to be accompanied with higher incidence of psychological and behavioural disorders, psychosomatic diseases, and suicide or parasuicide. Individuals’ reactions to unemployment has been examined in a number of studies which suggest that economic recessions appear to trigger acts of violence, suicide and homicide, through mediating destructive psychosocial pathways. Intrinsically, Lester (2001) applied a meta-analysis approach to regional correlates of homicide rates in eight nations (England, India, Japan, Russia, Sweden, Taiwan, Ukraine, USA). He found consistent evidence for the effect of unemployment rates on homicide rates. Other covariates, such as population size, were also found to be strong determinants of homicides. Lewis and Sloggett (1998) asserted that the association between suicide and unemployment is much stronger in comparison to other SES indicators.

Finally, Hitiris and Posnett (1992) used a pooled sample of 20 OECD countries and showed the impact of health care spending in reducing mortality inequalities whereas Or (2000), who approximated health status with premature mortality, confirmed that an improvement in economic conditions reduces mortality rates and importantly highlighted the strong impact of environmental factors deterioration in increasing mortality.

**ECONOMETRIC METHODOLOGY**

Fixed-effects models are used for all specifications in this study. To illustrate the methodology in a two variable case, consider the variables $Y_{it}$, $X_{it}$ which are observed for $T$ periods. Thus, the $(Y_{it}, X_{it})$ form a panel where the $i,...,N$ is the cross-sectional dimension and the $t,...,T$ is the temporal dimension of the panel.

For every cross-section number $i$, the general form of the panel data regression, with a single explanatory variable, is:
\[ Y_{it} = a_i + bX_{it} + u_{it} \]  
(1)

where \( b \) is the slope parameter, \( Y_{it} \) is the dependent variable and \( X_{it} \) is the explanatory variable, \( a_i \) are the unobserved cross-sectional effects which are different between the cross-sections. Thus, the fixed-effects model takes into account the so-called ‘unobserved heterogeneity’. First differencing is one way to eliminate the fixed effects \( a_i \). An alternative way is to use the fixed effects or ‘within’ transformation. The fixed effects estimator allows \( a_i \) to differ across cross-section units by estimating different constants for each cross-section. By averaging equation (1) over time it follows that:

\[ \bar{Y}_i = a_i + b\bar{X}_i + \bar{u}_i \]  
(2)

The fixed effects are computed by subtracting equation (2) (known as the ‘within’ mean for each variable) from equation (1) and then estimating equation (4) with pooled OLS. Thus, the independent variables that are constant across all \( t \) periods are swept away by the fixed-effects transformation.

\[ Y_{it} - \bar{Y}_i = b(X_{it} - \bar{X}_i) + (u_{it} - \bar{u}_i) \]  
(3)

where \( \bar{Y}_i = \sum_{t} Y_{it} / T \), \( \bar{X}_i = \sum_{t} X_{it} / T \), \( \bar{u}_i = \sum_{t} u_{it} / T \).

\[ \hat{Y}_{it} = b\hat{X}_{it} + \hat{u}_{it} \]  
(4)

where \( \hat{Y}_{it} = Y_{it} - \bar{Y}_i \), \( \hat{X}_{it} = X_{it} - \bar{X}_i \) and \( \hat{u}_{it} = u_{it} - \bar{u}_i \) is the time-demeaned data on \( Y \), \( X \), and \( u \) respectively.

In fixed-effects models the strict exogeneity assumption must hold. This implies that in order for the fixed-effects estimator to be unbiased, the idiosyncratic error \( u_{it} \) should be uncorrelated with each of the explanatory variables across all time periods. However, this estimator allows for arbitrary correlations between \( a_i \) and the independent variables in any time period. The fixed effects estimator \( \hat{b} \) is the coefficient of interest and it can be expressed as (Wooldridge, 2001):

\[ \hat{b} = \left( \sum_{i=1}^{N} \sum_{t=1}^{T} \hat{X}_{it}' \hat{Y}_{it} \right)^{-1} \left( \sum_{i=1}^{N} \sum_{t=1}^{T} \hat{X}_{it}' \hat{Y}_{it} \right) \]
The estimated intercepts $\alpha_i$ are swept away in the time-demeaned equations. They are viewed as omitted variables controlled through the within transformation (Wooldridge, 2000). The $\alpha_i$ can be estimated along with the $b$ by taking the traditional view of the fixed effects model, that is the dummy variable regression model. In this case, in equation (1), $\alpha_i$ are to be estimated after including a dummy variable for each cross-sectional observation together with the independent indicators and run a pooled OLS regression. In the dummy variable regression model the same $\hat{b}$ estimators, as with the fixed effects model, are obtained. Furthermore, the estimated intercepts can be computed as:

$$\hat{\alpha}_i = \bar{Y}_i - b\bar{X}_i$$

The specification, which will be used in this study, is a fixed-effects model in the form:

$$MR_i = a_i + b_1UR_i + b_2ADM_i + b_3POPDENSITY_i + b_4CIG_i + b_5ALC_i + b_6CALORIES_i + S_i$$  \hspace{1cm} (5)

The subscripts $i = 1, \ldots, 13$ and $t = 1, \ldots, 20$ are the country and the year period indicators respectively. $a_i$ is the country-specific fixed-effects intercepts. $MR_i$ denotes the various facets of mortality rates per 100,000 individuals and $S_i$ is an index of time effects across the 13 countries included in the sample, a year-specific dummy for 20 time periods (the omitted year is 1977).

The set of independent variables is as follows:

- $UR_i$: National unemployment rates (%)
- $ADM_i$: In-patient care admissions (%)
- $POPDENSITY_i$: Population density (residents per square km)
- $CIG_i$: Number of cigarettes consumed annually
- $ALC_i$: Annual alcohol consumption
- $CALORIES_i$: Average number of calories a person receives each day
Fixed-effects models provide the advantage of controlling for time-invariant characteristics that are different among cross-sections. In all regressions the results are corrected for heteroskedasticity, by applying White’s heteroskedasticity consistent covariance matrix estimator. This estimator provides correct estimates of the coefficient covariance in the presence of heteroskedasticity of unknown form.

**Independent variables**

The national unemployment rate is the primary independent variable of interest for this study. Unemployment rate reflects the economic cycle and therefore the economic conditions in the country. Furthermore, this study controls for a number of other variables in order to reduce sources of confounding in the unemployment-mortality relationship.

Preston (1975) highlighted that medical technological change can explain much of the observed increase in life expectancy. He argued that inequalities in health-care spending and healthy practices among individuals could partially explain mortality differences across countries. To capture this effect, in-patient care admissions (as % of the population), are included in (5) as a regressor. This variable is calculated as the sum of the hospitalized cases for patients admitted in inpatient facilities for at least 24 hours. However, the measure excludes day-cases, the release of a patient for the weekend, cases where hospital treatment is provided at the patient's home and newborns. In-patient care admissions could be expected to contribute to the reduction of preventable deaths.

Population density was found to be correlated to health in many ways. Thus, Adelman (1963) suggests that population density is a crude index of population pressure upon available resources. Furthermore, to some extent, the urbanisation reflects the economic development and degree of industrial growth (Pritchard and Evans, 1997). However, Groot (1997) argues that in highly populated regions communicative diseases are expected to flourish. Bruinsma et al. (2003) suggests that population density is an important determinant in the development of antibiotic resistance, which impacts negatively in the prevention of illnesses. Highly urbanized areas are expected to exhibit a higher incidence of mental and psychosomatic diseases, due to the rapid rhythms in the way of living and the increased stress. Studies, such as Mahoney et al.
(1990), Pritchard and Evans (1997) and Lester (2001), found a harmful effect of population density on cancer mortality and suicide. In view of the above, the variable of population density approximated as inhabitants per square kilometer, is included in the model (5).

Controls for lifestyle factors are also included in the model, in the form of smoking and drinking incidence. As the reviewed literature revealed, smoking is harmful and associated with a wide range of diseases, such as ischaemic heart disease, stroke, chronic bronchitis, and cancers. Smoking probably represents the largest single determinant of avoidable deaths.

Alcohol consumption is also an important determinant of health which is included in the model (5). Overall mortality rates, homicides, heart diseases, and cancers are believed to be influenced by alcohol drinking patterns (Romelsjö and Leifman, 1999; Vogel, 2002). Specifically, Poikolainen (1995) in a review of relevant studies pointed out that most studies reviewed concluded that a curvilinear, J-shaped relationship exists between alcohol and mortality.

Obesity due to unbalanced dietary habits seems to be a growing problem in industrialised countries. Dietary patterns are important in disease prevention and health promotion. A rich calories diet has serious long-term effects on health, such as circulatory diseases. Due to the unavailability of long time-series data on average body mass index, this study attempts to capture the dietary habits by using as a regressor the average number of calories per person.

The evidence on the link between physical environment and health are still limited probably due to lack of data (Or, 2000). However, this study controls for an environmental degradation indicator, carbon dioxide emissions (CO₂). Carbon dioxide emissions exist naturally in the environment but human actions, such as the burning of fossil fuels (coal, oil, gasoline), elevate it to levels dangerous for human health. This indicator is of particular interest as a proxy of environmental deterioration due to human activities.
**Dependent variables**

**Age-standardised mortality rates**

The dependent variable primarily used in this study is total age-standardised mortality rates per 100,000 individuals for each of the 13 European Union countries included in the sample. The implicit assumption is that unemployment may have differential effects on the constituent part of the overall mortality rates. Thus, an exclusive focus on the overall mortality rates may hide some differential effects of the unemployment rate on the constituent parts that make up the overall mortality rate. Thus this study aims to highlight the different facets of mortality that can be more acutely affected than others from unemployment. The effect of the unemployment rate upon six causes of age-standardised mortality rates per 100,000 individuals is investigated, namely mortality from (i) ischaemic heart diseases, (ii) cancer of trachea, bronchus, and lung cancer, (iii) malignant neoplasms, (iv) motor vehicle accidents, (v) homicide and purposeful injury and (vi) suicide and self-inflicted injury.

In addition, the relationship of interest is examined with respect to age-specific mortality rates for six age groups: 25-34; 35-44; 45-54; 55-64; 65-74; and 75-84 years old.

**Unstandardised mortality rates**

Due to the unavailability of standardised mortality rates for males and females needed to study gender inequalities in mortality, the unstandardised death rates per 100,000 individuals are used namely (i) overall male crude death rates, (ii) overall female crude death rates.

In summary, the following facets of mortality are used in this study.

- All-cause age standardised mortality rates
- Age standardised mortality rates from ischaemic heart diseases
- Age standardised mortality rates from cancer of trachea, bronchus, and lung cancer
- Age standardised mortality rates from malignant neoplasms
- Age standardised mortality rates due to motor vehicle accidents
- Age standardised mortality rates from homicide and purposeful injury
- Age standardised mortality rates from suicide and self-inflicted injury
- Age standardised mortality rates of people aged 25-34 years
Age standardised mortality rates of people aged 35-44 years
Age standardised mortality rates of people aged 45-54 years
Age standardised mortality rates of people aged 55-64 years
Age standardised mortality rates of people aged 65-74 years
Age standardised mortality rates of people aged 75-84 years
All-cause male crude (unstandardised) death rates
All-cause female crude (unstandardised) death rates

THE DATA

There is a lack of consistent time series for health measures across countries. Information on mortality rates is one of the few exceptions. The relative availability of mortality rates from official data sources facilitates the analysis over long periods.

This study investigates the relationship between unemployment and mortality rates in 13 European Union countries (Austria, Belgium, Denmark, Finland, France, Greece, Ireland, Italy, Netherlands, Portugal, Spain, Sweden, and the UK) during the twenty-year period between 1977-1996. Germany and Luxembourg, the two remaining European Union countries are excluded from the analysis due to lack of data for the whole of the time period studied. The majority of data are derived from the European Database ‘Health for All’ of the World Health Organisation. The indicators of population density and the environmental degradation indicator (CO2 emissions) are drawn from the data set of Health, Nutrition, and Population Statistics of the World Bank data files. Both datasets are viewed to be reliable and contain all the appropriate variables for this study for a long time span.

Sample characteristics
The summary statistics of the variables included in the regressions are displayed in Table 1. Mortality trends changed significantly in the 20th century. The average overall age-standardised mortality rates in the countries studied, fell from 1977 to 1996 by approximately 24% (from approximately 971.8 to 734 deaths per 100,000 individuals). Specifically, middle-aged and older people exhibited substantial health gains from the observed reduction in all-cause mortality levels. While total average
mortality rates for individuals aged 25-34 years fell about 6%, the rate of decrease for the age group of 35-44 years was 17%. The biggest decreases in mortality rates are observed for the age groups of 45-54 and 55-64 years, with decrease rates of 28% and 29% respectively. As one would expect, the gains decrease marginally for older age groups. The longevity gains are substantial. For example, average mortality rates of the age group of 65-74 years declined roughly by 27%, while the decline in mortality of those aged between 75-84 years was 25%.

Ischaemic heart diseases, cancers, and neoplasms constitute some of the main causes of death, in contrast to the infectious diseases that were the main cause of death in the past. The average mortality rate of ischaemic heart disease for the thirteen European Union countries studied fell by approximately 37% in the 20-year time period. Cancer and malignant neoplasms do not show any signs of significant reduction. Specifically, mortality incidence due to malignant neoplasms fell by only 4% and death rates from cancer increased by roughly 4%.

The average rate of deaths from homicides increased substantially during these 20 years, by 12%. On the other hand, mortality rates from suicides decreased by about 10%. The mortality from car traffic accidents decreased by an impressive 39%, (from approximately 19.1 deaths per 100,000 individuals in 1977, to 11.6 in 1996).

THE RESULTS

All cause age-standardised mortality rates
In Table 2, column (a), the basic specification of the model is presented. The dependent variable is total age-standardised mortality rates per 100,000 individuals.

A strong, positive relationship between unemployment rates and mortality is obtained. This finding indicates the deleterious impact of joblessness on the health of individuals and it is consistent with a number of studies (Moser et al., 1987; Morris et al., 1994; Lavis, 1998). Based on the findings of this study, a 1% increase in national unemployment rates is expected to increase mortality rate by approximately 2.18, that is 2.18 deaths for every 100,000 inhabitants; a substantial increase.
A strong protective effect of in-patient care admissions on mortality is observed. In detail, if in-patient care admissions increase by 1%, mortality rates decrease by about 6.41, that is 6.41 deaths for every 100,000 inhabitants. This effect implies the importance of health-care provision. In-patient care admissions act as an important factor in the prevention of specific causes of death amenable to medical intervention and provision.

In line with Mahoney et al. (1990) and Lester (2001), Table 2 reveals that population density is correlated positively to mortality levels, a finding that may suggest that in highly urbanized areas there may be a higher incidence of psychological diseases or that high population density facilitates the transmission of communicable diseases.

Table 2 reveals that lifestyle risk factors, such as increases in smoking and alcohol drinking increase mortality. On the contrary, the relationship between the nutrition indicator and mortality rates turned out to be insignificant.

In Table 2, column (2) the environmental degradation indicator is added as a regressor in the earlier model. The inclusion of the environmental conditions indicator does not significantly alter the results. Carbon dioxide emissions, have an insignificant effect on overall mortality rates.

In conclusion, the statistical analysis above has shown that high unemployment rates have a detrimental effect on mortality and thus health.

**Age-specific standardised mortality rates**

Table 3 exhibits the results of the regressions of mortality rates for six specific age groups against the set of independent variables and again before and after the inclusion of controls for carbon dioxide emissions. The consequences of unemployment rates on mortality show strong differences with respect to age. Importantly, the relationship between unemployment rates and mortality is positive and significant in all but the three age groups namely the 25-34, 65-74, and 75-84 years age groups. For all these groups the effect of unemployment rates on mortality is statistically insignificant. Since, in the European Union countries, individuals older
than 65 years of age are in retirement they are largely immune from the labour market conditions. Thus, one would not expect a significant impact of unemployment rates on the mortality rate of individuals above the 65 years of age threshold.

The statistical insignificance of the effect of the unemployment rate on mortality for the 25 to 34 years old supports the argument that the young are more able to cope with the effects of unemployment than their older counterparts. Indeed, though for young people unemployment may result in boredom or lack of purpose, the distress from unemployment is less among the young. Young age groups display a more optimistic view of the world, easily maintain social contacts and feel a lot less isolated, bored and inactive compared with their older counterparts. Clark and Oswald (1994) pointed out that this might be attributed to the fact that young people are less worried about unemployment because they recognise that it happens more to people like them. In turn, this may be attributed to the fact that ‘job shopping’ is more frequent among young age groups, which exhibit a high job-to-unemployment turnover (Nickell (1980). Therefore, overall one could expect that the effect of the unemployment rate on the health, and thus mortality of the younger individuals will be substantially less severe compared to older age groups. This is reflected in the results reported by the present study since it shows that although unemployment is not a significant mortality determinant in the younger age group, its impact on older age groups strengthens gradually. The 45 to 54 years of age group appears to be affected most by unemployment. This result is in line with earlier studies, which have shown a strong unemployment effect on the health of middle-age groups (Morris et al., 1994; Theodossiou, 1998; Ungváry et al., 1999).

The effect of population density on the mortality rates of the different age groups is positive for all but the youngest age group. Thus, harmful effects of population density do not seem to affect the health of the younger individuals who in fact appear to benefit from high population density. This effect might be attributed to the fact that the psychological benefits associated with urbanisation such as access to better employment opportunities, better opportunities for socialisation and the like outweigh the negative effects of high population density on health for the young age group. This is not surprising since young people exhibit more physical vigor, dynamism and resilience to disease compared to older individuals.
Smoking incidence as measured by the average daily cigarette consumption has detrimental effects on health as it increases mortality. However, its impact for the age groups after the age of 54 becomes statistically insignificant. Perhaps this reflects the fact that the percentage of those who smoke after the age of 54 decreases since the likelihood for the onset of health problems increases for the older age groups.

Studies such as Mukamal et al. (2003) and Barefoot et al. (2002) have shown that alcohol consumption may have beneficial effects on health. This study show that alcohol consumption appears to have beneficial effects for the youngest age group as it reduces the mortality rates. Yet, it seems to have detrimental effects on the health of the 45 to 54 and the 65 to 84 age groups. High calorie intake affects negatively individuals’ health and, thus, increases mortality for all but the oldest group. This may reflect the fact that at the later stages of the life cycle, individuals are more restricted and more careful with their nutrition habits compared to their younger counterparts due to the increased incidence of morbidity. The strong effect of high calories intake on mortality is in line with the findings of recent studies on the effect of obesity on health (Peeters et al., 2003; Sturm, 2001).

The introduction of the environmental degradation indicator in the regressions has not significantly altered the results. High carbon dioxide emissions appear to increase the mortality of the younger age group of 25-34 years. The effects are insignificant in the other age groups.

**Cause-specific standardised mortality rates**

Table 4, exhibits the results of the fixed-effects models regarding the mortality due to different causes of death. For each cause of death, two alternative models are shown namely one excluding the environmental indicator (column (a)) and one including it (column (b)). Importantly, for five out of the six causes of death investigated, it is found that unemployment rates have a consistent harmful impact on health and thus, increase mortality.

Specifically, a number of studies established a positive link between unemployment and ischaemic heart diseases (Junankar, 1999; Ungváry et al. 1999). Recent studies
contradict this finding by suggesting that increased unemployment rates lower deaths from ischaemic heart disease (Ruhm, 2000; Neumayer, forthcoming). The present study is in line with these former studies. The results clearly show a strong positive association between unemployment rates and mortality from ischaemic heart disease. Similarly, the unemployment rate turned out to affect mortality due to trachea, bronchus and lung cancers and due to malignant neoplasms. Furthermore, the results are in line with a number of studies suggesting the positive association of the unemployment rate with deaths from homicides and suicides. Mortality from traffic accidents is the only cause of death found not to be affected by unemployment rates. The above findings contradict the results of Ruhm (2000) and Neumayer (forthcoming).

Turning to the remaining independent variables, the results do not substantially differ from the earlier findings presented in Table 2. Nevertheless, there are some notable differences. Thus, alcohol consumption appears to have some beneficial effects in reducing mortality due to ischaemic heart disease and cancer, which is in line with some medical research on this issue (Mukamal et al., 2003; Barefoot et al., 2002). The environmental degradation indicator increases mortality rates due to heart diseases, a rather expected finding.

**Overall gender-specific crude death rates**

Many studies have reported that unemployment has a differential effect on the health of males and females. Theodossiou (1998) and Ungváry et al. (1999) reported health gender differentials and in particular, the studies have found that women’s psychological health is less affected from unemployment in comparison to men.

Unfortunately age standardised mortality rates disaggregated by gender are, to our knowledge, not available. However, crude (unstandardised) death rates per 100,000 inhabitants are. Thus, fixed-effects models, separately for the crude mortality rates for men and women are estimated. The results for the variable of interest are reported in Table 5 but they should be viewed with caution given the shortcomings of the dependent variable of not being age-adjusted. Nevertheless, they confirm a differential effect of unemployment rate on the mortality rates for males and females. In particular, the mortality of men increases with increasing unemployment rates.
On the contrary, the effect of the unemployment rate on crude mortality rates for females though positive is statistically insignificant. The statistical insignificance of the unemployment rate effect on mortality for women suggests that women seem to be less affected by unemployment compared to men. This offers some evidence to support the argument that women seem to be less affected by unemployment than men because an alternative is always available to them, that is the return to domestic commitments that provide a time structure, some sense of purpose, status and activity (Jahoda, 1982).

CONCLUSIONS

This study reports a strong positive association between national unemployment rates and mortality levels in 13 European Union countries. Economic downturns do appear to harm the health after all contrary to recent findings by Ruhm (2000, 2003) for the U.S. Thus the basic model specification suggests that a 1% increase in unemployment rates is associated with a decrease of 2.18 deaths per 100,000 individuals. In addition, unemployment rates exhibit a positive and statistically significant effect on five out of the six specific causes of death examined in this study, namely, ischaemic heart diseases, cancer of trachea, bronchus, and lung cancer, malignant neoplasm, homicide and purposeful injury and suicide and self-inflicted injury. Similarly, unemployment rates are found to affect the mortality of individuals in the 35 to 64 year age range. Furthermore, despite their shortcomings, crude mortality rates were used to study the differential effect of the unemployment rate on the mortality of males and females. This study revealed that the mortality of men is severely affected by increased unemployment rates but mortality of women appears to be less affected by the unemployment rate.

An innovation of this study is the attempt to control for confounding factors, which may mediate in the relationship of interest namely the unemployment–mortality relationship, and thus, the true relationship cannot be revealed. To overcome the problems associated with confounding, this study took into account the important confounding factors directly. The effect of cofounders such as in-patient medical care, life-style factors and population density play an important and independent role in
determining the mortality rates of populations. The protective effect of in-patient medical care on mortality is shown in most cases of age, cause-specific and gender-specific mortality rates. Overall both obesity and smoking have detrimental effects on population health and thus mortality. Alcohol consumption does appear to have beneficial effects, particularly regarding the mortality from ischaemic heart disease. Finally, population density positively affects mortality but its effect is mainly concentrated on the older age groups.
References:


Table 1. Summary statistics

<table>
<thead>
<tr>
<th>Variables</th>
<th>Mean</th>
<th>Standard Deviation</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Dependent variables</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age-standardised total mortality rates per 100,000 population</td>
<td></td>
<td></td>
</tr>
<tr>
<td>All ages</td>
<td>846.6</td>
<td>76.5</td>
</tr>
<tr>
<td>25-34 years old</td>
<td>94.3</td>
<td>2.5</td>
</tr>
<tr>
<td>35-44 years old</td>
<td>176.5</td>
<td>10.47</td>
</tr>
<tr>
<td>45-54 years old</td>
<td>439.8</td>
<td>50.8</td>
</tr>
<tr>
<td>55-64 years old</td>
<td>1085.5</td>
<td>118.8</td>
</tr>
<tr>
<td>65-74 years old</td>
<td>2743.4</td>
<td>272.2</td>
</tr>
<tr>
<td>75-84 years old</td>
<td>7136.6</td>
<td>697.3</td>
</tr>
<tr>
<td>Age-standardised cause-specific mortality per 100,000 population</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Infant deaths (per 1,000 live births)</td>
<td>9.42</td>
<td>2.72</td>
</tr>
<tr>
<td>Ischaemic heart disease</td>
<td>157.2</td>
<td>22.2</td>
</tr>
<tr>
<td>Trachea/bronchus/lung cancer</td>
<td>39.6</td>
<td>0.99</td>
</tr>
<tr>
<td>Malignant neoplasms</td>
<td>196.3</td>
<td>3.0</td>
</tr>
<tr>
<td>Motor vehicle traffic accidents</td>
<td>15.3</td>
<td>2.2</td>
</tr>
<tr>
<td>Homicide and purposeful injury</td>
<td>1.3</td>
<td>0.1</td>
</tr>
<tr>
<td>Suicide and self-inflicted injury</td>
<td>14.1</td>
<td>0.9</td>
</tr>
<tr>
<td>Crude death rates per 100,000 population</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>1053.1</td>
<td>23.7</td>
</tr>
<tr>
<td>Female</td>
<td>945.8</td>
<td>14.3</td>
</tr>
<tr>
<td><strong>Independent variables</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Unemployment rates (%)</td>
<td>9.0</td>
<td>1.9</td>
</tr>
<tr>
<td>In-patient care admissions (%)</td>
<td>16.2</td>
<td>0.8</td>
</tr>
<tr>
<td>Population density (people per square km.)</td>
<td>140.8</td>
<td>2.8</td>
</tr>
<tr>
<td>Number of cigarettes consumed per person per day</td>
<td>1800.0</td>
<td>61.8</td>
</tr>
<tr>
<td>Pure alcohol consumption, litres per capita</td>
<td>9.7</td>
<td>0.4</td>
</tr>
<tr>
<td>Average number of calories available per person per day</td>
<td>3288.0</td>
<td>83.3</td>
</tr>
<tr>
<td>Carbon dioxide (CO\textsubscript{2}) emissions, metric tons per capita</td>
<td>7.9</td>
<td>0.4</td>
</tr>
</tbody>
</table>

All data (except for population density and carbon dioxide emissions) are drawn from the Health for All Database, WHO ([http://www.euro.who.int/hfadb](http://www.euro.who.int/hfadb)). Population density and carbon dioxide emissions were taken from the Health, Nutrition, and Population Statistics of World Bank data set ([http://devdata.worldbank.org/hnpstats/](http://devdata.worldbank.org/hnpstats/)).
Table 2. Fixed-effect estimates of overall age-standardised mortality equations

<table>
<thead>
<tr>
<th>Independent variables</th>
<th>Dependent variable</th>
<th>All-cause age-standardised mortality rates</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>(a)</td>
</tr>
<tr>
<td>Unemployment rates (%)</td>
<td></td>
<td>2.18 (2.22)</td>
</tr>
<tr>
<td>In-patient care admissions (%)</td>
<td></td>
<td>-6.41 (-3.20)</td>
</tr>
<tr>
<td>Population density</td>
<td></td>
<td>2.06 (4.47)</td>
</tr>
<tr>
<td>Per capita daily cigarettes consumption</td>
<td></td>
<td>0.01 (1.69)</td>
</tr>
<tr>
<td>Per capita alcohol consumption</td>
<td></td>
<td>5.57 (1.72)</td>
</tr>
<tr>
<td>Per capita daily average intake of calories</td>
<td></td>
<td>0.04 (1.22)</td>
</tr>
<tr>
<td>CO₂ emissions</td>
<td></td>
<td>-</td>
</tr>
<tr>
<td>Time effects</td>
<td></td>
<td>Yes</td>
</tr>
<tr>
<td>Fixed-effects</td>
<td></td>
<td>Yes</td>
</tr>
</tbody>
</table>

* t-statistics are included in the parentheses. The results are corrected for heteroskedasticity with White’s heteroskedasticity correction.
### Table 3. Fixed-effect estimates of age disaggregated standardised mortality equations

<table>
<thead>
<tr>
<th>Dependent variables</th>
<th>All-cause age-standardised mortality rates (in age groups)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>25-34 years old</td>
</tr>
<tr>
<td></td>
<td>(a)</td>
</tr>
<tr>
<td>Unemployment rates (%)</td>
<td>-0.40 (1.32)</td>
</tr>
<tr>
<td></td>
<td>-0.44 (-1.52)</td>
</tr>
<tr>
<td>In-patient care admissions (%)</td>
<td>-2.37 (-3.95)</td>
</tr>
<tr>
<td></td>
<td>-1.43 (-2.31)</td>
</tr>
<tr>
<td>Population density</td>
<td>-0.46 (-3.11)</td>
</tr>
<tr>
<td></td>
<td>-0.40 (-2.97)</td>
</tr>
<tr>
<td>Per capita daily cigarettes consumption</td>
<td>0.01 (2.98)</td>
</tr>
<tr>
<td></td>
<td>0.01 (2.15)</td>
</tr>
<tr>
<td>Per capita alcohol consumption</td>
<td>-3.96 (-5.56)</td>
</tr>
<tr>
<td></td>
<td>-4.49 (-6.03)</td>
</tr>
<tr>
<td>Per capita daily average intake of calories</td>
<td>0.02 (2.44)</td>
</tr>
<tr>
<td></td>
<td>0.01 (1.74)</td>
</tr>
<tr>
<td>CO₂ emissions</td>
<td>-3.26 (4.89)</td>
</tr>
<tr>
<td></td>
<td>-1.13 (1.25)</td>
</tr>
<tr>
<td>Time effects</td>
<td>Yes</td>
</tr>
<tr>
<td>Fixed effects</td>
<td>Yes</td>
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</tbody>
</table>

* t-statistics are included in the parentheses. The results are corrected for heteroskedasticity with White’s heteroskedasticity correction.
Table 4. Fixed-effect estimates of cause disaggregated standardised mortality equations

<table>
<thead>
<tr>
<th>Dependent variables</th>
<th>Ischaemic heart disease</th>
<th>Trachea/bronchus/ lung cancer</th>
<th>Malignant neoplasms</th>
<th>Motor vehicle traffic accidents</th>
<th>Homicides &amp; purposeful injury</th>
<th>Suicide &amp; self-inflicted injury</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>(a)</td>
<td>(b)</td>
<td>(a)</td>
<td>(b)</td>
<td>(a)</td>
<td>(b)</td>
</tr>
<tr>
<td>Unemployment rates (%)</td>
<td>1.20 (3.13)</td>
<td>1.09 (3.16)</td>
<td>0.18 (2.47)</td>
<td>0.18 (2.45)</td>
<td>0.38 (2.06)</td>
<td>0.34 (2.13)</td>
</tr>
<tr>
<td>In-patient care admissions (%)</td>
<td>3.19 (3.04)</td>
<td>5.75 (4.95)</td>
<td>-0.84 (-6.44)</td>
<td>-0.77 (-5.36)</td>
<td>-2.79 (-8.40)</td>
<td>-1.97 (-5.44)</td>
</tr>
<tr>
<td>Population density</td>
<td>0.47 (2.11)</td>
<td>0.63 (3.67)</td>
<td>-0.23 (-7.02)</td>
<td>-0.22 (-6.80)</td>
<td>-0.41 (-4.28)</td>
<td>-0.36 (-4.36)</td>
</tr>
<tr>
<td>Per capita daily cigarettes consumption</td>
<td>0.02 (3.24)</td>
<td>0.01 (2.20)</td>
<td>0.001 (0.82)</td>
<td>0.004 (0.54)</td>
<td>0.003 (1.40)</td>
<td>0.001 (0.88)</td>
</tr>
<tr>
<td>Per capita alcohol consumption</td>
<td>-5.75 (-5.80)</td>
<td>-7.21 (-7.86)</td>
<td>-0.95 (-5.21)</td>
<td>-0.99 (-5.19)</td>
<td>-0.70 (-1.67)</td>
<td>-1.17 (-2.78)</td>
</tr>
<tr>
<td>Per capita daily average intake of calories</td>
<td>0.04 (3.80)</td>
<td>0.02 (2.99)</td>
<td>0.01 (6.67)</td>
<td>0.01 (6.34)</td>
<td>0.02 (5.00)</td>
<td>0.02 (4.39)</td>
</tr>
<tr>
<td>Per capita CO₂ emissions</td>
<td>- 8.96 (7.95)</td>
<td>- 0.23 (1.41)</td>
<td>2.85 (5.87)</td>
<td>- 0.24 (1.77)</td>
<td>- 0.06 (0.41)</td>
<td>0.51 (1.36)</td>
</tr>
<tr>
<td>Time effects</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>Fixed-effects</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
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</table>

* t-statistics are included in the parentheses. The results are corrected for heteroskedasticity with White’s heteroskedasticity correction.
Table 5. Fixed-effect estimates of overall male and female crude death equations

<table>
<thead>
<tr>
<th>Dependent variables</th>
<th>All-cause male crude death rates</th>
<th>All-cause female crude death rates</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>CO₂ emissions not included</td>
<td>CO₂ emissions included</td>
</tr>
<tr>
<td></td>
<td>(a)</td>
<td>(b)</td>
</tr>
<tr>
<td>Unemployment rates (%)</td>
<td>2.25 (1.93)</td>
<td>2.13 (1.95)</td>
</tr>
<tr>
<td>Other controls</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>Time effects</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>Fixed-effects</td>
<td>Yes</td>
<td>Yes</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Dependent variables</th>
<th>All-cause female crude death rates</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>(a)</td>
</tr>
<tr>
<td>Unemployment rates (%)</td>
<td>0.81 (0.61)</td>
</tr>
<tr>
<td>Other controls</td>
<td>Yes</td>
</tr>
<tr>
<td>Time effects</td>
<td>Yes</td>
</tr>
<tr>
<td>Fixed-effects</td>
<td>Yes</td>
</tr>
</tbody>
</table>

* t-statistics are included in the parentheses. The results are corrected for heteroskedasticity with White’s heteroskedasticity correction. Control variables are: In-patient care admissions (%), Population density, Per capita daily cigarettes consumption, Per capita alcohol consumption, Per capita daily average intake of calories, Per capita CO₂ emissions.
ENDNOTES:

1 Stern, 1983; Creed, 1998; Ungváry et al., 1999.

2 Moser et al., 1987; Morris et al., 1994; Lavis, 1998.

3 10 specific causes of death were used namely, death rates from malignant neoplasms, cardiovascular disease, influenza/pneumonia, chronic liver disease and cirrhosis of the liver, motor vehicle fatalities, other accidents and adverse effects, suicide, homicide and legal intervention, infant deaths, and neonatal deaths.

4 Hammarström, 1994; Morris et al., 1994.

5 Morrell et al., 1994; Theodossiou, 1998.


7 Gerdtham and Johannesson, 2003; Morrell et al., 1993; Yang and Lester, 1995.

8 Some studies have used the country’s GNP as a regressor together with the unemployment rate. However, GNP also reflects the business cycle, and, as Okun’s law indicates one should expect that fluctuations of unemployment rate to be the mirror image of fluctuations in GNP. Thus, it was considered inappropriate to include the country’s GNP as a regressor in this study. Its inclusion would only have the effect of obscuring the effect of the unemployment rate on mortality.

9 European health for all database, WHO Regional Office for Europe, Copenhagen, Denmark, (http://www.euro.who.int/hfadb).

10 The data for population density and carbon dioxide emissions were taken from the Health, Nutrition, and Population Statistics of World Bank data set (http://devdata.worldbank.org/hnpstats/).

11 At the 10% level of statistical significance.

The fixed-effect models reported in tables 2, 3, and 4 were also estimated with the dependent variables being transformed in natural logarithms. The results were similar but are not reported for space consideration. They are available from the authors on request.

The usual control variables namely in-patient care admissions (%), population density, per capita daily cigarette consumption, per capita alcohol consumption, per capita daily average intake of calories, per capita CO₂ emissions are also included in the regressions.

For model (a) the P-value is 0.055 and for model (b) is 0.054.