BUSINESS CYCLES AND HEALTH

HOW THE LEVEL OF DEVELOPMENT OF A COUNTRY AFFECTS THE RELATIONSHIP BETWEEN THE STRENGTH OF ITS ECONOMY AND THE HEALTH OF ITS POPULATION

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ABSTRACT

Recent research has overwhelmingly concluded that recessions are beneficial to the health of a developed society. This study tests the hypothesis that such a relationship is sensitive to the level of development of the country. Using panel data from the OECD, the association between total mortality and unemployment is shown to be positive but negatively dependent on the level of GDP per capita. Furthermore, the tipping point between an overall positive and negative relationship between health and unemployment is found to occur at PPP-adjusted GDP per capita level of $10,000. Similar patterns are established for six major causes of death, and in addition, male mortality is shown to be more sensitive to changes in unemployment and GDP than is female mortality. The results of this study imply that while long-run economic growth is beneficial to the health of both developed and developing countries, short-run growth only improves the health of the latter.

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I. INTRODUCTION

It is common knowledge that long-run economic growth has a positive impact on the health of a population. More ambiguous is the impact that short-run fluctuations in economic activity have on health. When research on this topic began, it was believed that short-run growth affected health in the same way as long-run growth, but recent research has found the opposite to be true in developed countries. Paradoxically, data has shown that when unemployment increases during economic contractions, the health of the population, as measured by a wide range of indicators, improves.

Many explanations for this phenomenon have been proposed. During times of economic expansion, the opportunity cost of not working increases, meaning that people will, on average, choose to invest less time in health-producing activities, such as exercise, in favor of spending those hours at work. Hazardous working conditions, job-related stress and the physical exertion associated with employment may have negative effects on health, and they will become particularly pronounced when working hours are increased, as is likely during periods of economic growth. During expansions, furthermore, there will be an increase in work-related injuries, as these are concentrated among procyclical industries like construction. Finally, when the economy is strong in one geographic location, there is likely to be an influx of migrants to that area in search of employment. This mobility can lead to higher mortality due to imported diseases and overcrowding (as in hospitals, on the road, etc.). Figure A.1. provides a pictorial mapping of these pathways.

The link between business cycles and health has been extensively studied among countries in the Organization for Economic Cooperation and Development (OECD), but there have been no studies showing how this relationship changes across countries with different

levels of economic development. However, a few have examined how the relationship changes over the development experience of a single country and others have examined this relationship in solely developing countries.

This paper substantiates the hypothesis that the negative association between economic performance and health weakens as the level of development decreases, and eventually, actually become positive. That is, in the most developed countries, an increase in unemployment is associated with an improvement in the health of a society, but the opposite is shown to be true in countries with lower levels of GDP per capita. The tipping point between these two situations is estimated to occur at a GDP per capita level of $10,000 in PPP-adjusted 2000 dollars.

The rationale for this hypothesis comes from the fact that in developing countries, people are more often likely to be on the brink of survival. The income effect, which is negligible in rich countries, is likely to be much stronger in developing countries. In rich countries, there are numerous social programs to keep income from falling below base levels necessary for survival. If unemployment increases in a less developed country, however, many people may no longer have sufficient income to survive or may have to work longer hours to earn the minimum amount of income needed to sustain their families. Both of these outcomes would lead to worsened health of the population. Also, during economic downturns, the price of food will rise relative to people's incomes, lowering the level of nutrition of the population, thereby deteriorating health outcomes. Finally, in developing countries, social programs are much weaker and an economic recession could lower spending on them so much as to increase exposure to infectious diseases and decrease the medical services available to the population. These pathways are summarized by Figure A.2.

The study proceeds by reviewing the relevant literature in this area of economics in Section II. Section III. explains the model and the theory behind it, Section IV. outlines the data and its source, and Section V. describes the estimation techniques used in the analyses. Section VI. interprets and discusses the results of the models estimated in Section V. Section VII. suggests areas for further research and Section VIII. concludes the paper. Following these are appendices and references.

II. LITERATURE REVIEW

i. *SHORT-RUN VERSUS LONG-RUN*

It has long been known that permanent economic growth leads to improved health. Early research in the study of the relationships between health and business cycles hypothesized that short-run economic growth would affect health in the same manner as long-run growth. This school of thought was championed by Brenner (1973, 1975, 1979), who found a countercyclical variation in admissions to mental hospitals, infant mortality rates, and deaths due to cardiovascular disease, suicide and homicide in the United States. In the years since, his papers have been widely discredited due to errors in his analysis, as pointed out by Gravelle, Hutchinson and Stern (1981), Wagstaff (1985), and Cook and Zarkin (1986). In addition, researchers who tried to replicate Brenner's findings were unable to ascertain the same results. Among these were Forbes and McGregor (1985), Joyce and Mocan (1993), and McAvinchey (1988).

McAvinchey (1988), in trying to verify Brenner's findings, found results contradictory to those of his predecessor. Unemployment and mortality data was collected for the years 1959 to 1982 for five European countries: France, West Germany, Italy, Sweden and Ireland. Separate

analyses were conducted for each sex in each country, and the optimal lag time between unemployment and mortality was calculated for each model using maximum likelihood estimation. The mean value of the optimal lag times was 4.9 years, with a maximum of seven years for Swedish women and a minimum of two years for German men. McAvinchey contended, like Brenner, that during recessions, health would decline, as was the prevailing theory at the time. He was unable to reject the null hypothesis that unemployment has no effect on mortality. Contradictory to his and Brenner's hypotheses, the relationship was in fact negative for seven out of the ten subgroups, with the exception of German women and the French. McAvinchey, however, proposed that the sign of this relationship was actually explained by fluctuations in income, for which unemployment is a proxy. However, later papers would show that variation in income is insufficient to explain the degree of the association between unemployment and health (Ruhm 2003 and 2004). The results of McAvinchey's study also showed that, in general, women seem to benefit more from a decrease in unemployment than men. In addition, there was some evidence to suggest that different age groups are affected unequally by short-run economic fluctuations, with the results showing that mortality was most sensitive to a change in unemployment in countries with a younger population. According to McAvinchey, this finding follows intuitively, as younger people are more likely to lose their jobs during a recession than people of other age groups.

In the years since Brenner's original research and the work of his contemporaries, the prevailing theory has been that health is negatively associated with the performance of the economy. Rather than affecting health through the same mechanisms as long-run growth, short-run growth has been hypothesized in recent research to affect health through different pathways. Johansson (2003) stresses the distinction between the two types of economic growth. Transitory

strengthening of the economy leads to more intensive use of the existing stock of human and physical capital, whereas permanent growth results from improvements in technology that expand production capacity, which decreases the stress on human capital. Thus, in short-run expansions, people are more likely to be overworked and therefore experience decreased health. Moreover, long-run economic improvements are associated with better medical technology and infrastructure, which result in a sustained lower mortality rate.

ii. *UNITED STATES*

The first conclusive evidence that economic expansions decrease the health of a population was published by Ruhm (2000). He collected longitudinal, state-level data from 1979 to 1991, using mortality rates as a measure of health status and unemployment as a measure of the state of the economy.

Results from Ruhm's analysis showed that a one percentage-point increase in the unemployment rate is associated with a 0.5 to 0.6 percentage point decrease in the overall mortality rate, which amounts to around 10,000 deaths annually. Strong evidence was also found that an increase in unemployment generates a decrease in deaths from all preventable causes of death, or those that respond to short-run changes in environment and behavior. Suicide and homicide, however, were shown to be countercyclical. The same association between suicides and homicides and business cycle fluctuations was also documented by Brenner (1973, 1975, 1979), though the main conclusions of the two authors with regard to overall health are opposing. Furthermore, deaths from cancer had no significant correlation to unemployment.

Attempting to explain the fluctuations seen in preventable causes of death, Ruhm (2000) performed a separate analysis on health-related behaviors using data from the Behavioral Risk Factors Surveillance Survey (BRFSS). The results showed that tobacco and alcohol use increase

during periods with low unemployment, which are also associated with more obesity and less leisure-time exercise.

In subsequent years, Ruhm has written a number of papers following up on his 2000 paper and exploring other topics within the field. Ruhm (2004) repeated the BRFSS analysis with data extended to the year 2000. Once again, smoking, drinking and obesity were found to decline during temporary economic downturns while leisure-time physical activity was found to increase. Furthermore, the drop in smoking was disproportionately strong among heavy smokers, as was the decline in drinking particularly strong among heavy drinkers. Similarly, a drop in unemployment caused the largest change in exercise time among the completely inactive and the largest change in weight among the severely obese. Males and minorities also experienced a larger decrease in obesity during economic contractions. Finally, little evidence was found to suggest an important role of income-effects on mortality rates.

More in-depth investigation into the relationship between business cycles and drinking, a behavior associated with many health problems, was carried out in Ruhm and Black (2002). Again, BRFSS data was used for the years 1987 to 1999. More attention was paid to higher amounts of drinking, as consuming two alcoholic beverages per day has been linked to lower coronary heart disease, so such levels of drinking would convolute any negative effect that would be expected to be observed between alcohol use and health. They found, in accordance with Ruhm (2000 and 2004), that drinking is procyclical and, moreover, that this effect is particularly pronounced among heavy drinkers. The data also suggested that a small part of the negative relationship between drinking and unemployment is due to decreased incomes during recessions, which reduces the amount of money that people are able or willing to spend on alcohol. A similar rationalization can be made for the variations seen in smoking (Ruhm 2000

and 2004). This finding helps to explain the negative relationship between health and unemployment as alcohol use in itself can contribute to more health problems (such as liver cirrhosis, studied in Ruhm 2000), but can also contribute to higher mortality from alcohol-related automobile accidents, which Ruhm (1995 and 2000) showed to increase during economic expansions.

Another study by Ruhm (2003) used a variety of less extreme medical conditions for the health outcome variable. Microdata from the National Health Interviews Survey (NHIS) were used for the years 1972 to 1981. Ruhm found that there is a countercyclical variation in physical health that is especially pronounced for individuals of prime working age, employed persons, and males, though he cautioned that this last relationship may have evolved over time due to increased female participation in the labor force. This result is in contradiction to McAvinchey (1988), who found that women are most affected by changes in unemployment. Furthermore, Ruhm's analysis showed that there is a strong relationship between unemployment and acute rather than chronic conditions, similar to the findings of Ruhm (2000). These results were robust when income was accounted for, meaning that changes in income associated with short-run economic fluctuations cannot fully explain the relationship between business cycles and health. Using three-year lags, Ruhm tested whether the effects of unemployment on health were sustained over time, a hypothesis that his results rejected. This is also in contrast to McAvinchey (1988), who found an average optimal lag of 4.9 years for Europeans. Finally, Ruhm (2003) found some evidence that mental health improves and the use of medical services increases during economic expansions, possibly an indication that people are seeking care for their decreased health.

In yet another investigation into health and business cycles in the United States, Ruhm (2007) compared state-level unemployment rates to deaths from coronary heart disease, a well-documented and understood condition that appears to respond rapidly to employment stressors, health-related behaviors and environmental conditions. Using the same model as Ruhm (2000), the mortality rate of coronary heart disease was found to be negatively associated with unemployment, with little difference across age groups. Due to the aforementioned nature of coronary heart disease, this result indicates that it may indeed be behavioral factors associated with changes in work hours or intensity that lead to adverse health effects during times of economic growth. Moreover, the effects of unemployment on health seemed to have completely dissipated after five years. In a subsequent analysis, Ruhm found that advanced medical treatments for coronary heart disease decrease during economic expansions, perhaps due to the increased time spent at work.

iii. *OTHER DEVELOPED COUNTRIES*

In addition to the United States, the relationship between health and unemployment has been strongly documented for other countries in the OECD, in both cross-national and single-country studies. The overwhelming majority of these studies corroborate the results of Ruhm (2000), with only a few apparent contradictions: Gerdtham and Johannesson (2003 and 2004) and Johansson (2004).

One of the most exhaustive transnational studies of business cycles and health was Gerdtham and Ruhm (2006), which used aggregate panel data from 23 OECD countries between 1960 and 1997, adjusted for income and types of health care provision. They found that an increase in the national unemployment rate decreases mortality rates from cardiovascular disease, influenza and pneumonia, liver disease, motor vehicle accidents, and other accidents, all

of which were shown to be procyclical in Ruhm (2000). Deaths from cancer were largely unaffected by business cycles, and there was some evidence to suggest that suicides and homicides decline during good economic times. These results are in accordance with those of Ruhm (2003), who showed that chronic diseases had little association with unemployment, and Ruhm (2000), who showed that suicide and homicide rates appeared to be positively correlated with unemployment. Finally, the procyclical fluctuation in mortality was found to be much stronger in countries with weak social insurance programs, as approximated by the percent of GDP devoted to public social expenditure.

Suicide rates were investigated further by Viren (1995 and 2005). Viren (1995) analyzed aggregate Finnish time-series data from 1878 to 1994. The results showed that the number of suicides is negatively related to the change in growth rate of GDP, though not to the growth rate itself, and positively related to bankruptcies and unemployment. The fact that the growth rate of GDP is not correlated to mortality is in contrast to Tapia Granados and Ionides (2008), who showed that the growth rates of both GDP and GDP per capita are associated with mortality in recent years. This contradiction, however, might be due to the fact that Viren only used suicide rates whereas Tapia Granados and Ionides used overall mortality rates. The results of Viren (1995) also showed that nominal income seems to matter more than real income in relation to suicide rates.

Viren (2005) expanded his earlier research, to include stock prices as an additional economic indicator. Analyses of Finnish aggregate time-series data from 1878 to 1999 provided strong evidence that the expected growth rate of income determines suicide rates. The expected rate of change of stock prices also appeared to have a very strong effect on suicide rates, though not the prices themselves, with higher expected stock prices lowering the incidence of suicides.

The results for GDP, unemployment and bankruptcies were consistent with Viren (1995). In addition, the results of the 2005 analysis showed that the total number of bankruptcies have more predictive power than personal bankruptcies as a determinant of suicide rates. The results of both Viren (1995) and (2005) were consistent with all other papers studying the relationship between suicide and business cycles; unfailingly, research has shown that the number of suicides increases during economic contractions, regardless of the country.

Tapia Granados (2008) examined the relationship between unemployment and mortality in postwar Japan. Like Viren (1995 and 2005), he experimented with multiple indicators for the state of the economy, including GDP and unemployment, manufacturing activity (average hours, aggregate hours, employment and output), and the general level of employment (employment/population ratio and labor force participation). The results were consistent across all eight indicators, though Tapia Granados only went into detail for GDP, unemployment and labor force participation in his study.

Tapia Granados found that overall mortality is procyclical in postwar Japan. Deaths attributable to hypertension, diabetes and suicides are countercyclical, though the deaths from these causes accounted for only four percent of the total number of deaths. 41 percent of deaths were attributable to causes that were found to be procyclical, while the remaining half showed no correlation to business cycles. Again, the findings are consistent with other research showing that suicides are countercyclical (Ruhm 2000, 2003, Gerdtham and Ruhm 2006, Viren 1995 and 2005). Contrary to past research (Ruhm 2000, 2003 and Gerdtham and Ruhm 2006), however, cancer showed some evidence of being procyclical, though this relationship only showed up if the economy was lagged by one year.

A stronger relationship between mortality and economic fluctuations was found for males and for those between the ages of 45 and 64. These results are in stark contrast to McAvinchey (1988), who found that the relationship was strongest for women and countries with a lower average age, but in agreement with Ruhm (2003), who documented a stronger relationship for men and those of prime working age. In particular, Tapia Granados found that suicides in men are more sensitive to changes in the economy than for women, which is reflective of the particular gender roles in Japanese society. Overall, the results on the effect of business cycles on health across demographic variables such as sex and age have been contradictory. As such, the subject area warrants further investigation, though much of the variation is likely due to societal differences and may therefore be difficult to resolve.

Johansson (2004) used Gerdtham and Ruhm's (2006) model and a sample of 21 OECD countries from 1960 to 1997 to examine whether a shorter workweek improves health. One of the main explanations proposed for why periods of high unemployment are associated with better health is that the opportunity cost of not working increases during economic expansions, meaning that people work more. This hypothesis holds that when a person works more, he experiences more stress and more work-related injuries, and has less time to devote to health-producing activities such as leisure-time physical activity. According to this explanation, a longer workweek should be associated with higher mortality. However, the result of Johansson (2004) was the opposite of what past research contended: longer workweeks are associated with lower mortality.

Johansson hazarded that this could be due to correlation between the duration of workweeks and income or some other underlying variable, though when the former is controlled for, the results do not change much. Again, this is not surprising, as income has been shown to

have little effect on the relationship between health and business cycles (Ruhm 2000, 2004, and Ruhm and Black 2002). The results of this paper are convoluting, as they seemingly contradict many of the theoretical explanations of the mechanisms through which business cycles affect the employment status of the individual respondents. Instead, most researchers have generally used some sort of measure of the greater economy in the region in which the respondent lived as a proxy for the economic situation that the respondent was facing at the time of the survey (such as in Ruhm 2000). Perhaps the effect is only visible for aggregate levels of unemployment because it is not, on average, the act of becoming unemployed that increases health, but rather decreased – yet still positive – work hours that lead to increased health. This possible resolution, however, is undermined by Johansson (2004), who showed that longer workweeks are associated with decreased health. On the other hand, given Sweden's historically generous unemployment benefits, it is difficult to explain how unemployment could actually worsen health. Indeed, Gerdtham and Ruhm (2006) showed that the relationship between unemployment and mortality was weakest in the countries with the best social insurance programs, Sweden included. The results of these two papers deserve attention in further research in order to reconcile their conflicting conclusions with the rest of the body of literature.

Gerdtham and Johannesson followed up in 2004 using the same dataset as Gerdtham and Johannesson (2003). They found that mortality decreases significantly as individual income rises. This result is also in contradiction to past literature, the majority of which found little role for income in explaining the relationship between unemployment and health (Ruhm 2000, Ruhm and Black 2002). No significant relationship between mean community income and community income inequality could be found.

iv. *DEVELOPING COUNTRIES*

The vast majority of research on the relationship between business cycles and health has focused on the developed world, as described in Sections III and IV. However, there have been no significant papers studying how this relationship changes across countries with diverse levels of development, though some papers have focused on specific regions with lower levels of per capita GDP and on variations in the level of development of a single country over time.

The relationship between short-run economic fluctuations and demographic variables in developing countries was studied by Palloni et al. (1996). The authors hypothesized that pre-industrial mortality, fertility and nuptuality should respond in significant ways to business cycles. In particular, increases in the relative price of food and other commodities, and the contraction of economic opportunities as a consequence of reduced incomes during economic downturns, exert downward pressures on marriages and births but raise mortality, particularly among young children and the elderly, the frailest members of society. Palloni et al. contended that deteriorating living standards can lower nutritional intake and, eventually, nutritional status. This will increase the body's susceptibility to infectious disease and compromise its ability to fight off the effects of pathogens, disabling the mechanisms for recovery. In addition, deterioration of the country's infrastructure from reductions in social spending will increase exposure to infectious disease and curtailment of government-provided health services, leading to a decreased ability for recovery from these diseases. They hypothesized that certain diseases, such as cholera, measles and tuberculosis, which are now extremely rare in OECD countries, will show particularly large responses to economic fluctuations.

To test their hypothesis, Palloni et al. collected data on 11 Latin American countries from as early as 1908 and ending around 1989. The health outcomes used were natality, nuptuality,

and infant and adult mortality, and the economic indicator was detrended average GDP. Using a model similar to that of Ruhm (2000), they performed a separate analysis for each country but obtained mixed results. In only a handful of their models did the coefficient on GDP come back significant. The magnitudes of the significant coefficients in these were quite small, and they were all in the expected direction: positive for nuptuality and fertility, and negative for mortality. Evidence also suggested that the marriage and birth sensitivity to business cycles decreased after 1955, while that of infant mortality might have increased. During the time period investigated, and indeed even now in many cases, the countries studied in Palloni et al. were still developing. The results then indicate that countries with lower levels of economic development experience a positive relationship between health and economic fluctuations.

The first paper that was highly indicative of the relationship between business cycles and health across different levels of development was Reher and Sanz-Gimeno (2000). They analyzed Spanish mortality data broken down by cause from 1850 to 1990 using a stepwise regression technique. This data were coupled with data on GDP to determine how the effect of economic fluctuations on health changed over time – or as Spain became increasingly developed.

In the pretransitional period, mortality was shown to be countercyclical, though after the Spanish Civil War, this relationship began to change and was completely dissipated by the 1960s and 1970s. At that point, Reher and Sanz-Gimeno concluded that the process of economic modernization in Spain was complete. Furthermore, in earlier data, there was little or no lag between a fluctuation in GDP and its resulting effect on mortality, but the lag length increased as time progressed. The effect of economic conditions on health was found to change over the course of modernization, though not monotonically. There were transient periods when the sensitivity to mortality rose, though at the end of the analysis (late 1980s), there appeared to be

no correlation whatsoever between GDP and mortality. However, Tapia Granados (2004) documented a procyclical variation in mortality in Spain in recent years, suggesting that since the final years of Reher and Sanz-Gimeno's dataset, the relationship between health and GDP in Spain continued to evolve, eventually becoming negative. Furthermore, Reher and Sanz-Gimeno also found that sensitivity to unemployment increases as age increases and is higher for men than for women. Again, the body of research is undecided as to how the relationship between health and business cycles varies across sex and age. Finally, similar results were shown for the prevalence of contagious diseases as for mortality.

Perhaps the most conclusive research in respect to how business cycles affect health in developing countries is Tapia Granados and Ionides (2008), who showed a similar change in the relationship across time in Sweden. They collected aggregate Swedish data from 1800 to 2000 on GDP, GDP per capita, unemployment and the crop index. They also used infant mortality by sex, age-specific mortality by sex, and life expectancy at birth as health outcomes.

Tapia Granados and Ionides found that in the first half of the 19th century, GDP growth was positively related to health progress. This was followed by a reversal of the relationship, becoming negative in the second half of the 20th century. Furthermore, their analyses allowed them to calculate the tipping point – the year in which the relationship reversed. Depending on the health outcome and economic indicator used, the tipping points calculated ranged from 1880 to 1987. As Reher and Sanz-Gimeno (2000) found, there was almost no lag between health and business cycles in the 19th century, and in the latter half of the 20th century, the lag had increased to one or two years. Furthermore, in the 19th century, Tapia Granados and Ionides found a Malthusian relationship between mortality and GDP, with declines in mortality stimulating

economic growth three or four years later, but these effects were no longer visible by the second half of the 20th century.

These studies compromise the greater part of literature concerning the association between business cycles and health in countries other than those with the highest levels of economic development. No major inferences have been drawn about how the nature of the relationship between business cycles and health is manifested in developing countries, which, as discussed in Section I. will not necessarily exhibit the same patterns as their richer counterparts. This paper seeks to do build on the existing literature by filling this hole by verifying this hypothesis with empirical data.

III. METHODOLOGY

For my analysis, I will use a similar model to the one used by Ruhm (2000) and Gerdtham and Ruhm (2006), with the country as the level of observation. The rough specification for my model, with i and t representing the country and year, is as follows:

(1) $H_{it} = \beta_0 + \beta_1 U_{it} + \beta_2 (U_{it} \times GDP_t) + \beta_3 GDP_{it} + \beta_4 X_{it} + v_{it}$

where H_{it} is the mortality rate, U_{it} is cyclical unemployment rate, GDP_{it} is per capita level of deterministic GDP per capita, X_{it} is a vector of supplementary regressors and v_{it} is the error term. A fixed-effects model will be used to account for time-invariant country effects, ts the error term can be decomposed into $v_{it} = u_{it} + c_i$, where u_{it} is the random error component and c_i is the country fixed-effect. X_{it} will include controls for population age and gender distributions, lifestyle factors, and the level of social benefits, as all of these could conceivably affect mortality.

$\beta_2(U_{it} \times GDP_{it})$ is an interaction term, allowing the effect that unemployment has on mortality to vary across levels of development. Then, the relationship between cyclical unemployment and mortality rates for a given level of GDP, say GDP_0, is $\beta_1 + \beta_2 GDP_0$, or the slope on cyclical unemployment. My hypothesis is that β_2 will be negative, meaning that as GDP rises, the relationship between unemployment and mortality becomes more negative. The point at which the relationship switches direction is therefore $-\beta_1/\beta_2$.

FIGURES 1 & 2. Hypothesized Effects of Unemployment and Development on Mortality

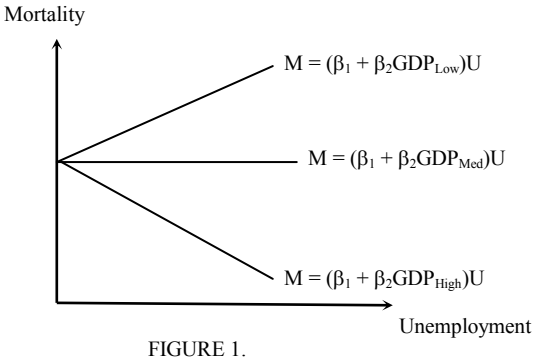


FIGURE 1.

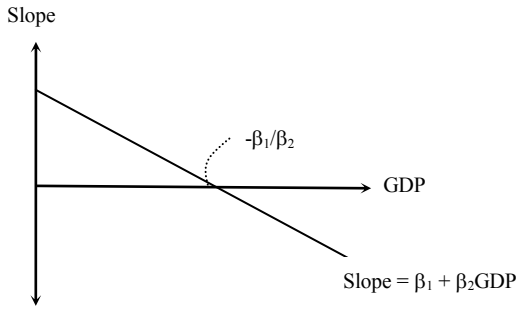


FIGURE 2.

A simplified version of this model is summarized in Figure 1. Each curve represents the relationship between unemployment and mortality for a different level of development. In this study, it is hypothesized that developing countries will exhibit a positive relationship, marked by GDP_{Low}, and developed countries will have a negative relationship, denoted by GDP_{High}. The slope of these lines is $\beta_1 + \beta_2 GDP$, again where GDP is a different level for each curve. This can also be visualized in Figure 2., which graphs the slope of the relationship between unemployment and mortality, or the slope of the curves in Figure 1. The graph shows how the relationship changes as GDP increases; it eventually becomes negative when GDP passes $-\beta_1/\beta_2$, which can be calculated from the results of the regressions in Section V.

IV. DATA

Ideally, health data from developing countries as well as developed countries would be used to test the hypothesis of this paper. However, health data from developing countries is nearly nonexistent, and the data that does is exist is unreliable. Instead, data was collected only on OECD countries, which still allows for a sufficient amount of variation in levels of economic development (GDP per capita). All of the variables used in the analyses were collected from the OECD Health Data 2008 database, including demographic and economic indicators. 29 countries were used in the estimations, with Turkey being the only OECD country excluded because of its lack of data on mortality. Unbalanced panel data was used for all of these analyses, with the time variable reaching as many as 46 years to as few as nine, depending on the data available. The variables used are summarized in Table 1. and described in more detail in the following subsections. Summary statistics of each raw variable are located in Table B.2.

i. *HEALTH STATUS*

Like Ruhm (2000) and most other researchers, mortality data was used as a measure of the health status of a population. There is much more data available on mortality because it is easier to document than nonfatal sicknesses, and it also avoids the problem of accounting for varying levels of severity and determining what constitutes the endpoint of an illness. From the database, mortality rates were collected per 100,000 people in the population. In addition to total mortality, data was collected on mortality broken down by cause and by gender. The causes chosen to be studied were malignant neoplasms (cancer); mental and behavioral disorders; acute myocardial infarctions (heart attacks); influenza and pneumonia; bronchitis, asthma and emphysema; chronic liver diseases and cirrhosis; land transport (including motor vehicle accidents); and intentional self-harm (suicides). These causes of death were chosen because they

have been shown to correlate to business cycles in past research, with the exception of cancer, which has been shown to be independent of short-run economic fluctuations. Furthermore, these are among the leading causes of death in many of the countries studied, so there are more observations per each year and country, increasing the accuracy of the regression estimates.

ii. *LEVEL OF DEVELOPMENT*

As a proxy for the level of development of the countries, GDP per capita was chosen. While this measure may fail to account for many facets of development such as living standards, it is both widely-reported and easily quantified, and it gives a rough guide to the economic prosperity of the individual. This measure is also available from the OECD Health Data 2008 as PPP-adjusted GDP per capita in U.S. dollars indexed to the year 2000. It was necessary to remove the cyclical component of GDP, as this is an indicator of short-run business cycles, rather the level of development. The natural log was taken of each GDP variable, as they both appeared to be right-skewed. After the log was taken, the data appeared to be normally distributed. To attain only the deterministic component of GDP, the natural log of GDP per capita was regressed against the lagged natural log of GDP per capita individually by country. For each country, then, the following model was used to predict the fitted values of GDP per capita.

$$(2) \qquad \ln(GDP_t) = \beta_1 \ln(GDP_{t-1}) + \beta_0 + \varepsilon_t$$

The error term, ε_t, in this model denotes the stochastic shock that describes the deviation of GDP from its deterministic trend. Therefore, instead of using the straight GDP per capita values, the fitted values from the model above were used. It should be noted that for all of the countries, the models above had an Adjusted-R^2 greater than 0.95, and both β_1 and β_0 were significant at the

5% level. Therefore, it can be assumed that the use of this model of GDP growth was appropriate. Also, the constructed variable for GDP was then lagged by both one and two years.

iii. *BUSINESS CYCLES*

Like the majority of past research on this topic, cyclical unemployment rates were chosen as an approximation of business cycles. To get only the cyclical component, unemployment was locally normalized by subtracting the mean of all of the years available for each country and subtracting their standard deviation to get $\frac{u_{it} - \bar{u}_i}{\hat{\sigma}_i}$. This calculation, however, assumes that the systematic component remains stationary throughout time. This assumption seems to be appropriate, as there is little correlation between unemployment and year within countries (see Table B.1). As with GDP, unemployment was lagged by one and two years.

iv. *CONTROL VARIABLES*

Control variables included those used in past research such as male-to-female ratio and age distribution, which was captured by three variables: percent of the population between the ages of 0 and 14, percent between age 15 and 64, and percent aged 65 or older. Of course, any two of these variables implies the third, so it was necessary to exclude one of the three. The first variable was chosen to be excluded as children are the least likely to be affected by fluctuations in unemployment as they are rarely employed, thus the other variables would better lend themselves to theoretical interpretation. In addition, public and total health care expenditure and unemployment benefits in PPP-adjusted GDP per capita in 2000 U.S. dollars and as a percent of total GDP were used. These variables can be assumed to approximate the degree of social protection provided by the government, which can have an impact on mortality and mitigate the effects of unemployment on health. The natural logs of both unemployment variables were taken as they appeared to be right-skewed. Finally, lifestyle variables on fertility, alcohol

consumption and fat intake were collected, which were not included in past research on this subject.

v. *INTERACTION TERM*

As discussed in Section IV., an interaction variable between deterministic GDP per capita and detrended unemployment was created to allow the relationship between health and business cycles to change as the level of development of a country changes. Lags for both one and two years were created from this interaction variable.

TABLE 1**.** Description of Variables

Variable	Description
Dependent Variables	
mortality	Total mortality*
female	Female mortality*
male	Male mortality*
suicide	Mortality by self-inflicted harm*
landtransport	Mortality by land transport*
liver	Mortality by chronic liver disease and liver cirrhosis*
lungs	Mortality by bronchitis, emphysema and asthma*
flupneu	Mortality by influenza and pneumonia*
heartattack	Mortality by acute myocardial infarction*
mental	Mortality by mental and behavioral disorders*
cancer	Mortality by malignant neoplasms*
Variables of Interest	
unemp	Unemployment rate
dtunemp	Detrended unemployment rate
gdp	GDP per capita
loggdp	Log of GDP per capita
dtloggdp	Log of deterministic GDP per capita
dtloggdpxdtunemp	Interaction term between *dtunemp* and *dtloggdp*
Control Variables	
mfratio	Male-to-Female ratio, males per 100 females
pop0_14	Percent of Population between ages 0 and 14
pop15_64	Percent of Population between ages 15 and 64
pop65	Percent of Population age 65 or over
he	Total health expenditure as a percent of GDP
publiche	Public health expenditure as a percent of GDP
hce	Total health expenditure per capita
publichce	Public health expenditure per capita
ubgdp	Unemployment benefits as a percent of GDP
logubgdp	Log of unemployment benefits as a percent of GDP
ubppp	Unemployment benefits per capita
logubppp	Log of unemployment benefits per capita
fertility	Number of children per woman aged 15 to 64
alcohol	Liters of alcohol consumed per capita per day, ages 15 and over
fat	Grams of fat consumed per capita per day

* deaths per 100,000 people

V. ESTIMATION

i. *TOTAL MORTALITY*

Before more advanced statistical techniques are employed, evidence of the procyclical nature of mortality can be readily seen from Figure 3. Here, mortality and unemployment rates have been locally detrended and demeaned, and then averaged across each year. The graph seems to roughly indicate that mortality and unemployment are negatively related. It is not possible, however, to ascertain from this graph that the relationship between mortality and unemployment is dependent on the level of development. To test for this, fixed-effects regression analysis was used.

FIGURE 3. Mortality and Unemployment Rates over Times

Model 1. (Table 2.) displays some preliminary results of the estimation of Equation (1) (Section III.), using a simple OLS procedure without fixed effects on the variables summarized in Table 1. *Pop15_64*, *pop65*, *mfratio*, *publiche*, *dtloggdp*, *dtunemp* and *dtloggdpxdtunemp* are significant at the 1% level, and the model has an Adjusted-R^2 value of 0.819. Both *dtunemp* and

dtloggdpxdtunemp are significant and in the expected direction. The residuals of the model appear to be normally distributed in quantile and probability plots and seem to be scattered randomly around the reference line y = 0. No significant outliers are visible. However, Ramsey's test rejects the null hypothesis of no specification error on the 1% level. The possibility of omitted variables bias is particularly serious. To take this into account, it is necessary to add more explanatory power to the model.

TABLE 2. Models for Total Mortality

Variable	Model 1.	Model 2.	Model 3.
pop15_64	-4.86 (1.23)**	-9.89 (1.29)**	-9.89 (2.33)**
pop65	-12.38 (1.48)**	-20.86 (1.63)**	-20.86 (3.59)**
mfratio	-20.85 (1.20)**	-21.14 (1.83)**	-21.14 (2.24)**
publiche	15.22 (2.63)**	9.94 (2.57)**	9.94 (4.16)*
dtloggdp	-191.74 (6.68)**	-152.71 (6.32)**	-152.71 (10.66)**
dtunemp	412.52 (44.31)**	158.54 (29.45)**	158.54 (43.31)**
dtloggdp xdtunemp	-44.47 (4.65)**	-17.15 (3.10)**	-17.15 (4.41)**
intercept	5032.62 (153.53)**	5148.92 (208.25)**	5148.92 (251.36)**
R^2	0.82	0.917	0.917
Adjusted-R^2	0.819	-	-
N	836	836	836
Estimation Method	OLS	Fixed-Effects	Fixed-Effects, Driscoll and Kraay SE

Notes: Standard errors in parentheses; * significant at the 5% level; ** significant at the 1% level

Instead of accounting for omitted variables bias by including additional regressors, a fixed-effects model was chosen to account for possible correlation between country-specific effects and the error term, which will then have both a random component and a fixed-effects component. One of the problems with omitted variables bias is that the omitted variable may be correlated to the remaining explanatory variables, and this correlation will lead to biased estimators. In a fixed-effects model, the missing variables can often be captured by the fixed-effects component of the error term, and as this is allowed to be correlated to the regressors, the estimated coefficients will be unbiased.

A random-effects model could also serve to better specify the model, but this method was rejected in favor of a fixed-effects estimator. A random-effects model assumes that the error term consists of two components, one of which is the random error component just as in the fixed-effects model. The second component in this method, however, is considered to be a random variable instead of a time-invariant fixed value. By assuming that this term is a random variable, one is essentially assuming that the panels (countries in this case) are a random sampling of a larger population and that the errors are not correlated to the explanatory variables (Gujarati, 2003). Neither of these assumptions is plausible with the data at hand. First, the countries were chosen not randomly, but in the interest of data availability, so the first assumption is violated. Second, it is entirely feasible that the error term is correlated to the explanatory variables. Consider the multitude of variables that have not been accounted for in the model, such as climate, which can have an impact on mortality rates. For example, climate is associated with latitude, which is in turn correlated to GDP per capita as the northernmost countries also tend to have higher GDP per capita. Therefore, it is also probable that GDP per capita and climate will be correlated, so likewise, the error term will be correlated with at least one of the explanatory variables. Given this, a fixed-effects technique seems to be the appropriate method. Furthermore, a Hausman test between the fixed-effects model (Model 2.) and the analogous random-effects model, rejects the null hypothesis on a 5% significance level with a p-value of 0.035. Therefore, it can be concluded that the results between a random-effects model and a fixed-effects model differ significantly, corroborating the theoretical reasoning above. As such, a fixed-effects model was chosen as the appropriate method for the analyses.

There is one additional assumption of the fixed-effects model that must be met before proceeding (Wooldridge, 2002): strict exogeneity of the regressors. That is, the regressors must

be uncorrelated to the random components of the error terms across all time periods. A similar, but less restrictive, assumption must be met for the OLS model. This is checked in an ad-hoc method by re-estimating Equation (1) using the fixed-effects estimator. Model 2. summarizes the results, with all of the same variables turning up significant and with the same signs as in Model 1. Furthermore, the variables of interest, *dtunemp* and *dtloggdpxdtunemp*, are both significant and in the expected directions. The R^2 statistic of this model is greatly improved over its predecessor, with a value of 0.917. Then, to test whether the aforementioned assumption is met, the random error terms are extracted from the model, and lagged both forward and backward by up to six years. This range of years was chosen arbitrarily, but it is unlikely that if there is no significant correlations found for these thirteen years, that there will be any for higher order lags. The correlations between these and each of the explanatory variables in Model 2. are summarized in Table A.7. None of the lags exhibited a high correlation with any of the explanatory variables, so it can be assumed that the assumption of strict exogeneity has been met. *Pop65* had the highest correlation across all lags, with a correlation reaching as high as 0.19, but all other explanatory variables had correlations below 0.1. As such, it can be supposed that the assumption has been met.

In addition to the three major concerns of the simple OLS regression model – multicollinearity, heteroskedasticity and temporal autocorrelation – a fourth must be considered when using panel data (Wooldridge 2002): spatial correlation. All of these were tested for using either Models 1. or 2. Variance inflation factors (VIF) and tolerance values indicate that there is little or insignificant mutlicollinearity between any of the variables in Model 1. except *dtunemp* and *dtloggdpxdtunemp*. This is not worrisome, however, as one would expect these two variables to be related since one is a component of the other. When either of these variables is

removed, all of the VIF values fall below five, indicating that the level of multicollinearity is insignificant.

Heteroskedasticity, however, was found to be present in both models. For Model 1., both the White test and the Breusch-Pagan/Cook-Weisberg tests for heteroskedasticity reject the null hypothesis of homoskedasticity at the 1% level. Furthermore, using a Modified Wald's test for groupwise heteroskedasticity, the null hypothesis of homoskedasticity is rejected in Model 2. at the 1% level. Similar results were found for autocorrelation; a Wooldridge test for first-order autocorrelation returns a p-value of 0.0, which indicates that the null hypothesis of no autocorrelation should be rejected.

Spatial dependence, while not an issue in one-dimensional datasets (that is, ones that have only one indexing variable), is a major concern for panel data. Driscoll and Kraay (1998) and Hoeschle (2007) showed that failing to account for cross-sectional dependence can lead to seriously biased standard error estimates. Spatial dependence means that the error term of one panel is correlated with those of other panels, and it is almost always present when the panels are constituted of countries (Wooldridge 2002). In this situation, unseen societal factors that may influence the error term do not end abruptly at the border of a country. For example, the people of Spain and Portugal may share a similar genetic makeup, which has a theoretically strong impact on mortality. As this is not accounted for in any of the regressors, it will show up in the error terms, which will then be correlated. This relationship can also be manifested across time periods; for example, economic conditions in one year Germany may affect the economy of France in the next year, leading to error term correlation that is both cross-sectional and asynchronous.

Model 2. was tested for very general forms of spatial correlation using a Pesaran test for cross-sectional dependence and a Breusch-Pagan LM test of independence. The latter is possible to implement in this scenario as there are, on average, as many years as there are time periods; this method tends to be less accurate when the number of panels is large relative to the time dimension (Hoechle 2007). Both tests confirm the hypothesis of the theoretical discussion above, indicating a presence of cross-sectional dependence. As such, it is necessary to find an estimation method that accounts not only for heteroskedasticity but general forms of both temporal and spatial autocorrelation.

There are many methods to account for one or two of these, but few that account for all three. One such method uses a nonparametric technique to account for the three aforementioned problems by adjusting the standard errors of the fixed-effects model. The implementation of this method, which uses Driscoll and Kraay standard errors, allows for unbalanced panels, which is the case with this dataset. This method also allows for a small number of years in relation to the number of panels, though due to the asymptotic nature of the underlying theory of this model (see Driscoll and Kraay, 1998), it is still advisable to be cautious in these scenarios. Model 3. implements this technique and has a total of 836 observations, meaning that there is an average of 28.83 years per country or about the same number of years as countries, which, as mentioned earlier, is not considered to be low.

The Driscoll and Kraay method of calculating standard errors is a variation on the standard heteroskedasticity and autocorrelation consistent covariance matrix techniques, such as those developed by Newey and West (1987). It requires no prior knowledge of the exact form of the contemporaneous and lagged cross-unit correlations. When applying this method, however, it is necessary to specify the maximum lag, m(T), where autocorrelation follows an AR(m)

process. If none is specified, the maximum lag, m(T), is often chosen somewhat arbitrarily by the formula $\text{floor}[4(T/100)^{2/9}]$, which is the first step in Newey and White's (1994) derivation of the optimal lag for covariance matrix estimation. Another method to determine the maximum lag is to test for autocorrelation at multiple lag lengths and use the highest one for which it and all other lags less than it exhibit autocorrelation. Discussion A.1. outlines the method used to test for the presence of higher order autocorrelation, a combination of a Breusch and Godfrey LM test, which can test for higher-order autocorrelation in single-dimensional (not panel) datasets, and the Wooldridge test for first order autocorrelation. From this, an optimal lag of three years was estimated, which, incidentally, is the same as was calculated via the formula $\text{floor}[4(T/100)^{2/9}]$.

Both Models 3. and 4. (Table A.2) fit a fixed-effects model with Driscoll and Kraay's standard errors, though Model 3. includes *publiche*, which is significant in this model with a p-value of 0.024. Removing *publiche* raises the number of total observations to 996 and so the R^2 value rises from 0.917 to 0.920 between Models 3. and 4. However, *dtloggdp* and *publiche* have a correlation of 0.706 indicting that if *publiche* were to be excluded from the model, there would be misspecification, which leads to biased estimations of the parameters. Therefore, Model 3. was chosen as the final model for overall mortality.

Model 3. is well-fit with an R^2 of 0.917. This model appears to be normal from examining quantile, probability and residual plots, and as in Model 1., there are no visible outliers. The model uses a maximum lag of three years when accounting for autocorrelation, as mentioned above. All of the variables are significant at the 1% level, except for *publiche*, as discussed. The variables of interest, *dtunemp* and *dtloggdpxdtunemp*, are both significant with p-values of 0.001. Furthermore, their coefficients have the expected sign: positive for *dtunemp* and

negative for *dtloggdpxdtunemp*. *Dtloggdp* is also significant and negative. The interpretation of these three variables is as follows. A 1% change in the level of cyclical unemployment changes mortality by (158.54 – 17.15) = 141.39 deaths per 100,000 holding all other variables constant. Likewise, a 1% change in GDP per capita lowers mortality by (152.71 – 17.15) = 169.87 deaths per 100,000.

From these coefficients, it is possible to calculate the level of GDP per capita at which the slope on unemployment, 158.54 – 17.15*dtloggdp*, changes sign. This is calculated by setting the slope equal to zero and solving for *dtloggdp*. This gives a GDP per capita level of $10,327.73. Calculation of a confidence interval for this statistic is much less straightforward. There have been numerous methods proposed for estimating the variance of a quotient of normally-distributed variables, though the Fieller method (Fieller, 1932) was used in this paper as it has been shown to consistently outperform other methods, including the Delta, Bayesian and bootstrap techniques (Hirschberg and Lye, 2007). Using this method, a 95% confidence interval for the level of GDP per capita at which the relationship between unemployment and mortality changes direction is $8,185.96 to $13,103.34.

In a variation on Model 3., lagged variables for *dtunemp*, *dtloggdp* and *dtloggdpxdtunemp* were included in the model to determine whether there is a latency period between a fluctuation in the business cycle and its associated effect on mortality. These three variables were lagged both one and two years. However, none of these variables came up significant using Driscoll and Kraay standard errors. It can therefore be concluded that there is no delayed effect of unemployment on mortality, at least at one and two year lags.

ii. *MORTALITY BY CAUSE*

As was done for total mortality, fixed-effect models with Driscoll and Kraay standard errors using a three-year lag were estimated for each cause of mortality. Models 5. through 12. use *suicide*, *landtransport*, *liver*, *lungs*, *flupneu*, *heartattack*, *mental* and *cancer*, respectively, as measures of health status. Each regression contained some combination of the age distribution variables, one or both of *he* and *publiche*, and possibly *mfratio* in addition to the three variables of interest, *dtloggdp*, *dtunemp* and *dtloggdpxdtunemp*. None of these models were as well-fit as the total mortality models, with R^2 values ranging from as high as 0.745 in Model 10., *heartattack*, to as low as 0.104 in Model 6., *landtransport*. This is not surprising, as mortality, on average, can be explained well by broad measures of the economy and population demographics, but individual causes are much more associated with specific behaviors, which are not included in this dataset. The weaker goodness-of-fit of these models do not nullify the results of the regressions, but they do indicate that any inferences drawn should be regarded with some degree of skepticism.

The variables of interest, *dtunemp* and *dtloggdpxdtunemp,* were significant in Models 5., 7., 11. and 12. In Models 8. and 10., they were jointly significant, as determined by Wald tests. The coefficients on these variables were in the same direction as in Model 3. except for Models 8. and 11., which showed the opposite pattern. The regressands in these two models were *lungs* and *mental*. A summary of the level at which the sign of the slope of unemployment changes for each cause is included in Table A.5.

iv. *MORTALITY BY GENDER*

Models for gender-specific mortality rates, as with total and cause-specific, were estimated via fixed-effects regression with Driscoll and Kraay standard errors, accounting for

temporal correlation as an AR(3) process. *Male* is used as the regressand in Model 13., as is *female* in Models 14. and 15 (summarized in Table A.4.).

Model 13. shows the same pattern in the variables of interest as in Model 3. All of the same variables are significant, in addition to *he*, and the coefficients are in the same direction. The magnitudes of the coefficients on *dtunemp* and *dtloggdpxdtunemp* are much larger than in Model 4. It should be noted, however, that the average mortality rate of males across all countries and years is 1,104 men per 100,000 males compared to the average mortality rate of entire population of 868. The larger magnitudes of these coefficients, then, could be due to the difference in the scale of the explanatory variable. The R^2 value in the model, unlike those in the cause-specific models, is reasonably high at 0.873.

Model 14. also shows a similar pattern. Again, all of the coefficients are in the same direction, though not all are significant. *He* is significant in Model 14., unlike in Model 3., but has a positive sign. *Dtunemp* and *dtloggdpxdtunemp* are not significant in this model, though they are in the same direction as Models 3. and 13. A Wald's test, however, indicates that the two variables cannot be omitted without significantly worsening the R^2. This statistic is high in Model 14. at 0.930, indicating that the model is well-fit.

As a very rough approximation of the degree of gender equality in society (as time goes on, gender equality progresses), an interaction term between *year* and *dtunemp* was included in Model 15., in a variation on Model 14. The coefficient was positive, indicating that as time goes on (i.e. gender equality increases), so does the impact of unemployment on female mortality. However, *dtunemp* and the two interaction variables turned up insignificant in Model 15., though a Wald test indicated that removing any of them would significantly worsen the goodness-of-fit.

From Models 13. and 14., the level of GDP per capita at which the sign on the slope of unemployment changes can be calculated, as was done earlier. For Model 13., this is $12,253.80 with a 95% confidence interval of $10,426.22 to $12,253.80, calculated using the Fieller method. In Model 14, this level is $2,627.50 with a 95% confidence interval of $1,244.42 to $5,903.262.

VI. INTERPRETATION AND DISCUSSION

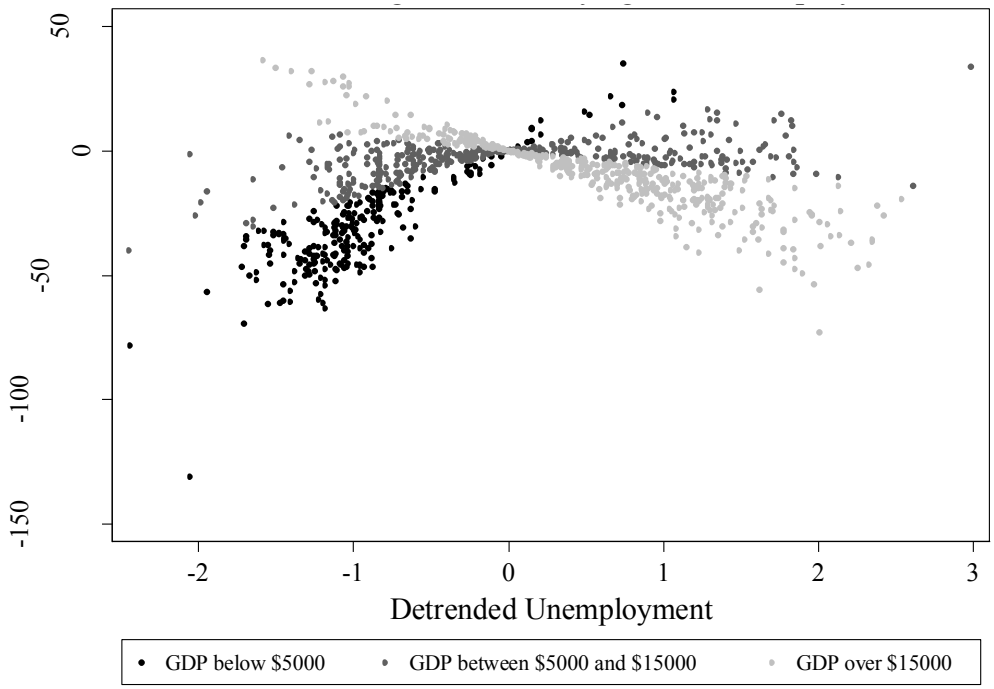
i. *TOTAL MORTALITY*

The estimation results in Section V. show that cyclical unemployment and the interaction term, *dtloggdpxdtunemp*, are significant in predicting mortality, which supports the hypothesis outlined in Section III. Not only is the mortality rate positively related to cyclical unemployment, but this relationship is negatively dependent on the level of GDP per capita. This pattern is illustrated in Figure 4., which displays the predicted change in mortality given a specific level of unemployment and GDP.

On the horizontal axis of Figure 4. is cyclical unemployment and on the vertical axis is the predicted impact on mortality for each observation given its level of cyclical unemployment and deterministic GDP per capita. It should be noted that the shape of this graph comes from the coefficients on *dtunemp* and *dtloggdpxdtunemp*, rather than directly from the data. Each point represents an observation for one year and country calculated by the formula (152.71 – 17.15*dtloggdp*)*dtunemp*. The graph displays the same conical shape as was expected in Figure 1. The shade of each point reflects the level of GDP of that country in that year. The black points indicate that the country has a deterministic GDP per capita of $5,000 or lower. The dark gray denotes countries with GDP per capita levels between $5,000 and $15,000. Finally, light gray

denotes the observations with the highest GDP per capita, over $15,000. The graph then shows that as the level of GDP increases, the slope on unemployment decreases.

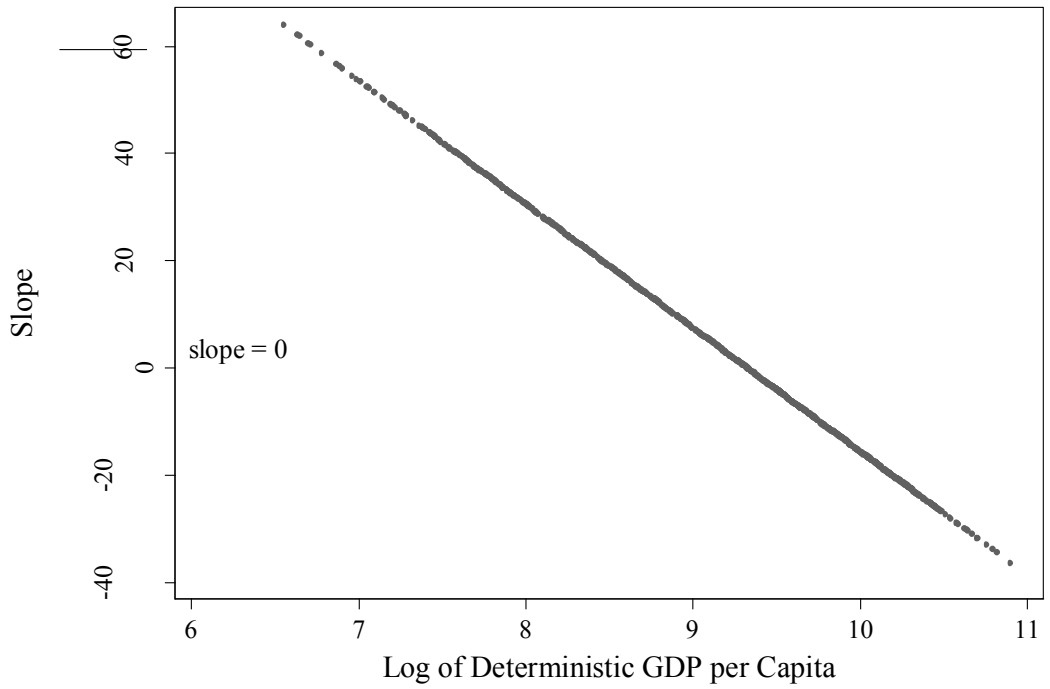
FIGURE 4. Predicted Change in Mortality given Unemployment and GDP



The slope on unemployment alone is graphed against the log of GDP per capita in Figure 5., as was theorized in Figure 2. The intersection of the line of observations and reference line at y = 0 marks the point at which the slope on unemployment becomes negative. An exact calculation of the tipping point can be computed from the regression coefficients, as was done in Section V. This occurs at a log of deterministic GDP per capita of 9.24, which gives an absolute GDP per capita level of $10,362.65. This level roughly corresponds, for example, to the United States in 1978 or Mexico in 2004. This has the implication that some countries, such as Mexico, have just entered the stage in which they experience the positive relationship between unemployment and mortality. Others, like the Czech Republic, whose GDP per capita fell below the tipping point in 1991, have recently fallen out of this stage. Still others, such as the United

States, have had sufficient levels of development to experience a positive relationship for many decades.

FIGURE 5. Slope of Mortality against Unemployment Given GDP



It is interesting that no lagged variables came up significant in the estimation of Model 4. Past research has offered evidence of lags, as discussed in Section II. Tapia-Granados (2008) found a one-year lag in postwar Japan and McAvinchey (1988) calculated a lag of 4.9 years in five European countries, though Ruhm (2003) found no evidence of a prolonged effect of unemployment on mortality. Of course, since all of the variables in the dataset are aggregated to the year, it is possible that there is a lag under a year in length that is not visible from the data.

ii. *MORTALITY BY CAUSE*

The models for cause-specific mortality are not nearly as well-specified as those for total mortality, so any inferences made from these models must be regarded with caution. The models indicate that *suicide*, *landtransport*, *liver*, *flupneu*, *heartattack* and *cancer* are all procyclical and

furthermore that the relationships between these variables become more negative as the level of development of a country rises. The fact that *cancer* (malignant neoplasms) and *suicide* are among these variables is surprising. Past research has overwhelmingly shown that suicide is countercyclical (Gerdtham and Ruhm 2006, Viren 1995 and 2005, and Tapia-Granados 2008). Theoretical explanations also hold that suicide should by countercyclical; people are more likely to commit suicide during recessions due to the emotional stress of losing a job, meaning that suicide should be countercyclical. This theory is validated by the model for *mental*, as deaths from mental and behavioral causes are found to be countercyclical, which is perhaps indicative of mental health improving during economic expansions. These results are consistent with the past literature on suicide and mental health (Ruhm 2003).

Furthermore, Gerdtham and Ruhm (2006) showed that cancer was unaffected by cyclical unemployment, though Tapia-Granados showed a procyclical relationship, but only when the economic indicator variable was lagged by one year. This study, however, finds deaths from cancer to be procyclical. Cancer is a disease that is not sensitive to short-run conditions but rather long-term lifestyle factors, and therefore should not be affected by business cycles. However, it is possible that while cancer is a condition that is not likely to be contracted because of short-run conditions, it is possible that the business cycles can weaken the body's immune system, leading to more deaths from this pre-existing condition.

The theory that chronic illnesses should not be correlated to business cycles (Ruhm 2000), however, is substantiated by the model for *lung*: deaths from asthma, emphysema and bronchitis are shown to be unaffected by business cycles. While this broad category can include acute asthma attacks and bronchial infections, the majority of the causes grouped into this

category are chronic, so the insignificance of the variables *dtunemp* and *dtloggdpxdtunemp* is not surprising.

In both models with the regressands *landtransport* and *flupneu*, *dtunemp* and *dtloggdpxdtunemp* come up insignificant. Ruhm (2000 and 2002) and Gerdtham and Ruhm (2006) show that there is a significant increase in motor vehicle accidents during economic expansions. Furthermore, it would be expected that influenza and pneumonia would be impacted by short-run business cycles because they are so-called "preventable" causes of death, meaning that they are responsive to short-term changes in behavior and environment. Perhaps the already cyclical nature of influenza epidemics confounds the analysis, as flu season follows a sinusoidal trend, peaking in the winter and ebbing in the summer. This could be accounted for by performing the analysis at the month-level rather than at the year.

It should finally be noted that because of the classification of these health status variables, some of the true associations between cause-specific mortality and unemployment may be hidden. For example, *liver* includes both liver cirrhosis and chronic liver problems, which means that is a combination of both acute and chronic illnesses, and only the former are expected to be affected by short-run economic fluctuations.

iii. *MORTALITY BY GENDER*

The results of the regressions with *male* and *female* as the regressands are consistent with past research. Male mortality shows much higher sensitivity to unemployment than does female mortality. This difference in the relationship between business and health across genders is most likely due to gender roles in the societies examined. There are two major factors that are likely to influence the magnitudes of the relationships for each gender.

Men tend to be the principal earners in a household, therefore if a woman loses her job during a recession, she is likely to have a very different experience than a man. In countries with lower levels of GDP per capita, unemployment can often leave a man with no source of income, leading to sickness, and on aggregate, a higher mortality rate (see Figure A.2.). A woman who becomes unemployed during a recession, however, is often supported by a secondary household income and therefore her experience of the economic downturn will be much less severe.

In developed countries, an analogous pattern is likely to occur. During an economic expansion, a man – whose social standing tends to be more dependent on his vocation and is probably already working longer hours than the average woman – will likely increase his work hours so much as to significantly lessen the time he can invest more time in health-producing activities. In aggregate, this will lead to higher male mortality. The same will happen for women, but to a lesser extent. An average woman is likely to work fewer hours, so increasing hours during an expansion will not have as much of an impact on the time she can invest into her health. Furthermore, a female social status is less dependent on profession as much as male standing, so a woman may not experience as much increased stress during an expansion.

These two factors imply that female health will not be affected as strongly by cyclical fluctuations and will also be less sensitive to changes in GDP per capita. Furthermore, the level of GDP per capita at which the direction of the relationship between mortality and unemployment reverses differs greatly across the two genders. For women, this level is $2,628 whereas for men it is $12,254. Figures A.9. through A.12. graphically represent this relationship. Finally, the relationship for women has been shown to change over time, becoming more like that of men. Because year is a rough proxy for the level of gender equality, it can be

inferred that as a society becomes more equal, the way female health is associated to business cycles becomes more and more like that of male health.

iv. *OTHER OBSERVATIONS*

It is interesting to note which control variables did not come up significant. Much of the theory on this subject suggests that the reason that mortality in OECD countries is procyclical is because these countries also have very good social programs. As such, when a person becomes unemployed in one of these countries, he is supported by government unemployment benefits. In less developed countries, unemployment benefits are weak if not completely absent, meaning that when a person loses becomes unemployed, he may not have enough money to afford basic sustenance and medical care, thereby raising the mortality rate on aggregate. It would be expected, then, that unemployment benefits would have an impact on the level of mortality. However, neither unemployment benefits per capita (*logubppp*) nor as a percentage of GDP (*logubgdp*) came up significant in any of the regressions. Perhaps this is due to a high correlation to other variables used in the models, though neither had a correlation greater than 0.6 with any other variable.

Also interesting is the fact that per capita health care expenditure, whether public or total, was not significant in any of the models. This might be due to the fact that these variables had significantly fewer observations than the other variables used in the model, thereby decreasing the degrees of freedom of the models. Total health care expenditure as a percent of GDP was significant in many of the models, and public health care expenditure as a percent of GDP was significant in a few more. While the coefficient on *he* is always negative, one counterintuitive result is that the coefficient on *publiche* is almost always positive. It would be expected that higher public spending on health care would lower the mortality rate, but the sign on this

coefficient indicates the opposite is true. However, *publiche* is correlated to *dtloggdp* with a correlation of 0.618, meaning that the GDP variable might capture some of the explanatory power of *publiche*. Indeed, *publiche* is negatively correlated to mortality, as is *dtloggdp*, so this result is not surprising.

Another counterintuitive result is that the coefficient on *mfratio* is always negative, except in Model 11. This means that the higher the ratio of males to females in the population, the lower the mortality rate is. This seems somewhat paradoxical as men, on average, have lower life expectancies than women (this is shown by their higher mortality rate in Table B.2.). It is possible that the countries that the longest male life expectancies are also the ones that have the most males as a percent of the total population, as fewer men have died off at earlier stages of life. Interestingly, *mfratio* is negatively correlated – albeit slightly – to both *dtloggdp* and *mortality*. This lends support to the explanation that perhaps countries whose men live longer have higher life expectancies, as life expectancy is related to mortality. However, life expectancy is also likely to be correlated to GDP per capita, which is not correlated to *mfratio*.

Finally, the variables *alcohol*, *fat* and *fertility* came up insignificant in many of the models, though theoretically, a case can easily be made for why each would affect mortality. It is common knowledge that consumption of high amounts of alcohol and fat is associated with lower life expectancies. Fertility is not only mathematically related to mortality, but it also serves as an indicator of societal norms, which in turn can impact mortality rates. The most apparent reason for these variables turning up insignificant is that they had many fewer observations than the other variables in the dataset, so any model that included one or more of them would have many fewer degrees of freedom. More evidence of this is that in the models that included these variables, the R^2 values were very low.

VII. FURTHER RESEARCH

The study fills a gap in the body of literature surroundin health and business cycles. Until now, there have been no studies that examine the relationship between unemployment and mortality across countries with different levels of development. Still, there are improvements that could be made to the model, the major one of which would be to expand the dataset. The sample used in this study was only a subset of the world's most developed countries, the OECD, rather than a representative sample. Still, the amount of variation in GDP per capita levels, both across countries and years, provided adequate variation in development levels to substantiate the hypothesis of this study. At the same time, any extrapolations made to countries outside of the sample must be made caustiously as the sample does not include any countries with Africa and few from Asia. As data from developing countries improves and expands, the models estimated in this paper can be repeated, giving a more global result as well as smaller standard errors. Also, decreasing the unit of analysis from the year to the month could offer additional insight. No lags came up significant in this study, but perhaps there is a lag under a year in length that would become apparent if monthly data were used.

In addition, more research into the differing impacts on mortality from unemployment could benefit the body of literature. Interaction variables between gender-equality and unemployment would be insightful, as *year* is only a rough proxy of equality and its interaction term with unemployment came up only jointly significant in Model 16. Gender equality could be measured by a number of variables, such as percent of women in the workforce, the average female's income as a percent of the average male's, or the percent of women holding a public office.

Finally, while the existence of an association between business cycles and health has been well-documented, the mechanisms through which this relationship manifests itself are much less studied. In fact, the little evidence there is on this subject (Johansson 2004) contradicts the theoretical explanation that people work fewer hours during recessions and can therefore devote more time to health-producing activities. This could be particularly insightful when trying to understand why the relationship evolves as the level of development of a country increases. Studying this, however, is much more complicated as it most likely necessitates the use of micro-level data, which is not as accessible as aggregate data.

VIII. CONCLUSION AND POLICY IMPLICATIONS

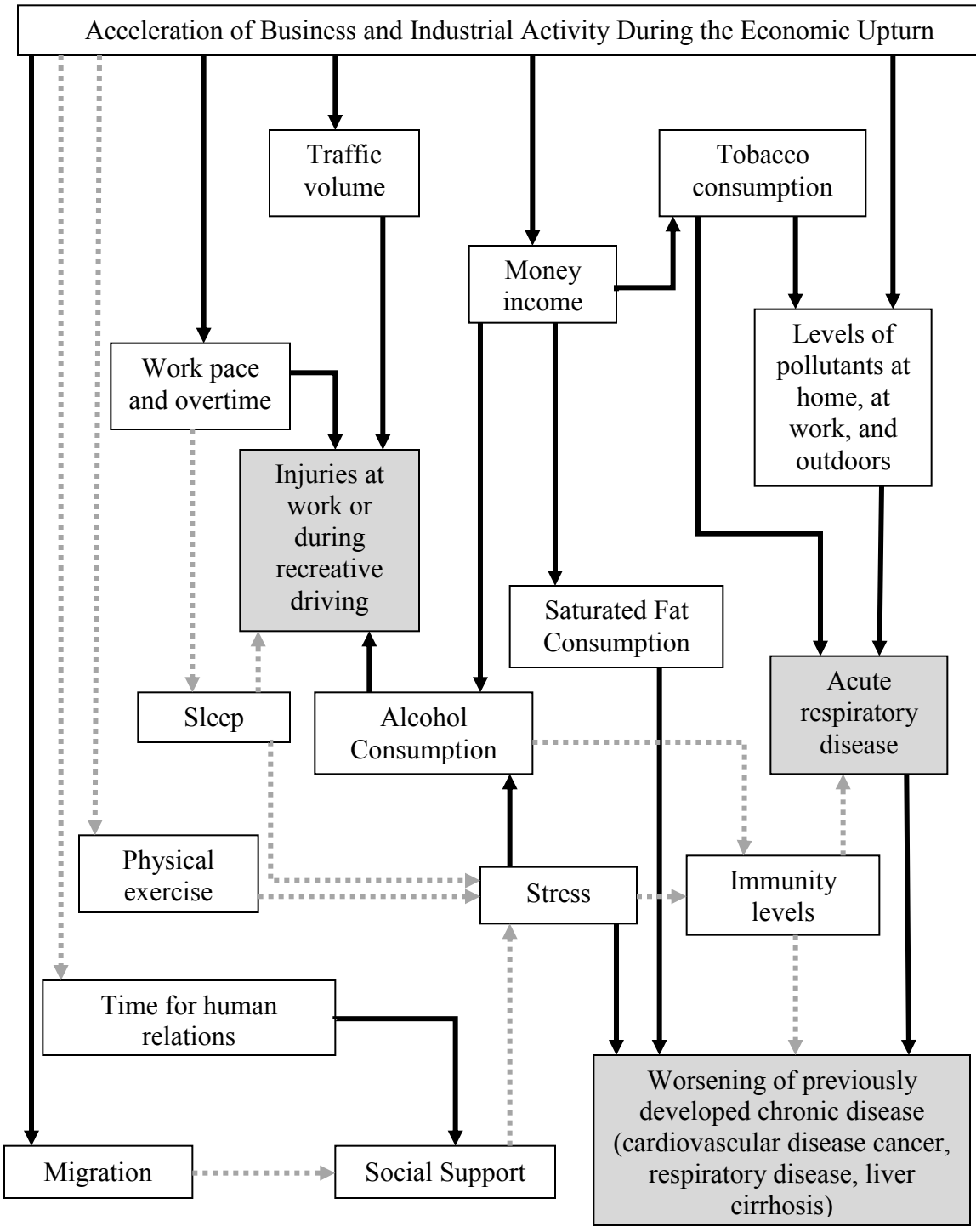
As this paper set out to establish, the level of development of a country affects the relationship between health and business cycles. Past research showed that there was a procyclical trend in mortality in OECD countries, but this paper shows that this is not true across all countries and years. In fact, the results of the analyses in this study indicate that the point at which the relationship becomes negative is a GDP per capita level around $10,000. Furthermore, as the literature suggests, male mortality is much more sensitive to changes in cyclical unemployment than is female mortality. Also, the level of per capita GDP at which the relationship between health and business cycles switches direction is much lower for women than for men. Deaths from suicide; land transport accidents; liver cirrhosis and chronic liver disease; influenza and pneumonia; myocardial infarctions; and cancer are shown to procyclical while deaths from mental and behavioral disorders are countercyclical. Bronchitis, asthma and emphysema-related deaths appear to bear no association to business cycles. The results of cause-

specific mortality, however, must be regarded with caution as their respective models were not well-specified, possibly leading to biased results.

The results of the analyses in this paper, and those of past research, indicate that while long-run growth is beneficial to the health of a population (that is, decreases mortality), short-run growth is detrimental to health in rich countries. As such, government programs to artificially boost economic growth leading to bubbles may actually harm the population's health. It is better, then, if a government is interested in minimizing mortality, for the government to invest in programs that lead to sustained long-run economic growth. This paper shows that this is not true in poorer countries, in particular, ones with GDP per capita levels below $10,000. In these countries, both long-run and short-run growth lower mortality rates, so any move that boosts economic output will improve the health of the country's people.

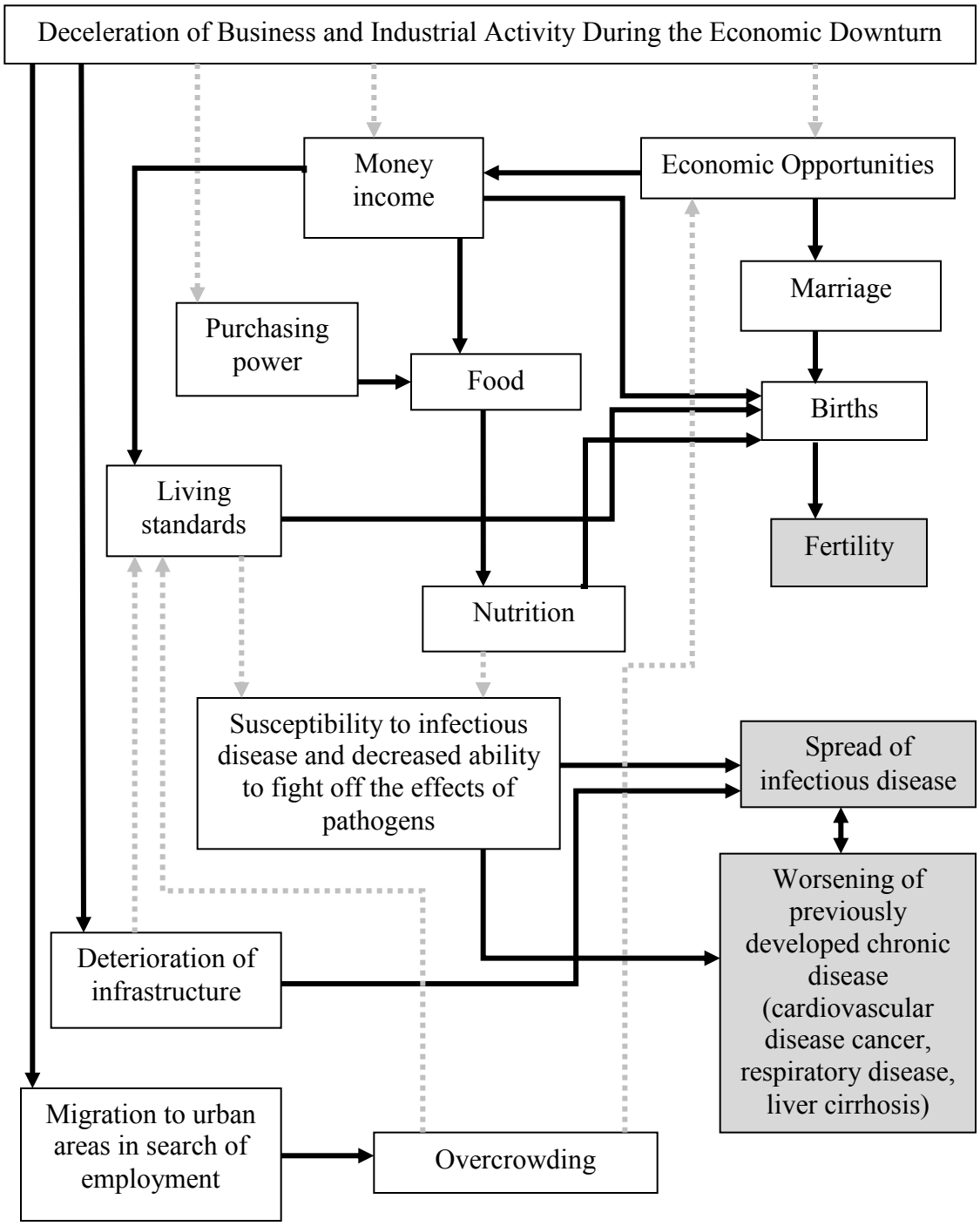
APPENDIX A. Theoretical and Empirical Supplements

FIGURE A.1. Pathways through which Economic Fluctuations Influence Mortality



Notes: Black, solid arrows represent positive effects, while gray, dashed ones represent negative effects. For instance, a drop in alcohol consumption raises immunity levels and decreases the risk f injuries. The shaded boxes indicate negative health outcomes that can directly lead to death. Many more possible pathways have been omitted for simplicity. Source: Tapia-Granados (2008).

FIGURE A.2. Pathways through which Economic Fluctuations Influence Mortality



Notes: Black, solid arrows represent positive effects, while gray, dashed ones represent negative effects. The shaded boxes indicate negative health outcomes that can directly lead to death. Many more possible pathways have been omitted for simplicity. Source: Palloni et al. (1996).

TABLE A.1. Models of Total Mortality

Variable	Model 1.	Model 2.	Model 3.	Model 4.
pop15_64	-4.86 (1.23)**	-9.89 (1.29)**	-9.89 (2.33)**	-9.62 (2.05)**
pop65	-12.38 (1.48)**	-20.86 (1.63)**	-20.86 (3.59)**	-17.17 (2,96)**
mfratio	-20.85 (1.20)**	-21.14 (1.83)**	-21.14 (2.24)**	-14.83 (1.31)*
publiche	15.22 (2.63)**	9.94 (2.57)**	9.94 (4.16)*	-
dtloggdp	-191.74 (6.68)**	-152.71 (6.32)**	-152.71 (10.66)**	-148.12 (12.26)**
dtunemp	412.52 (44.31)**	158.54 (29.45)**	158.54 (43.31)**	212.25 (31.32)**
dtloggdp xdtunemp	-44.47 (4.65)**	-17.15 (3.10)**	-17.15 (4.41)**	-22.85 (3.19)**
intercept	5032.62 (153.53)**	5148.92 (208.25)**	5148.92 (251.36)**	4486.33 (129.26)**
R^2	0.82	0.917	0.917	0.920
Adjusted-R^2	0.819	-	-	-
N	836	836	836	996
Estimation Method	OLS	Fixed-Effects	Fixed-Effects, Driscoll and Kraay SE	Fixed-Effects, Driscoll and Kraay SE

Notes: Standard errors in parentheses; * significant at the 5% level; ** significant at the 1% level

TABLE A.2. Models of Cause-Specific Mortality

Variable	Model 5.	Model 6.	Model 7.	Model 8.
	suicide	*landtransport*	*liver*	*lungs*
pop15_64	-	-	-0.64 (0.11)**	-
pop65	-	-	-1.13 (0.12)**	-
mfratio	-1.17 (0.12)**	-82.21 (11.83)**	-0.25 (0.10)*	-
publiche	1.00 (0.29)**	-	0.68 (0.33)*	2.31 (0.61)**
He	-0.55 (0.24)*	-	-1.36 (0.19)**	-
dtloggdp	-1.51 (0.37)**	-	2.84 (0.64)**	-15.49 (0.98)**
dtunemp	14.53 (1.39)**	335.07 (518.13)	8.64 (1.32)**	-7.05 (6.51)†
dtloggdpxdtunemp	-1.51 (0.14)**	-50.21 (54.27)	-0.88 (0.14)**	0.73 (0.69)†
intercept	139.10 (12.94)**	9160.30 (1135.40)**	71.97 (12.33)**	150.52 (8.58)**
R^2	0.267	0.104	0.363	0.614
N	836	1002	825	761

Notes: Standard errors in parentheses; * significant at the 5% level; ** significant at the 1% level; † jointly significant

TABLE A.3. Models of Cause-Specific Mortality

Variable	Model 9.	Model 10.	Model 11.	Model 12.
	flupneu	*heartattack*	*mental*	*cancer*
pop15_64	-1.24 (0.49)*	5.94 (1.14)**	-0.33 (011)**	-
pop65	-	10.66 (0.90)**	-	-
mfratio	-1.09 (0.53)*	5.69 (1.31)**	-	-4.29 (0.25)**
publiche	-	-	-1.98 (0.82)**	-
he	-	-	1.48 (0.42)**	-
dtloggdp	-10.06 (1.14)**	-105.64 (5.15)**	7.09 (0.91)**	-7.89 (2.13)**
dtunemp	14.26 (10.97)	23.08 (18.12)†	-13.94 (2.57)**	75.83 (9.86)**
dtloggdpxdtunemp	-1.58 (1.13)	-2.59 (1.88)†	1.49 (0.38)**	-7.94 (1.00)**
intercept	310.14 (57.79)**	19.12 (169.13)	-32.38 (7.13)**	672.35 (33.41)**
R^2	0.537	0.745	0.452	0.321
N	996	598	836	1001

Notes: Standard errors in parentheses; * significant at the 5% level; ** significant at the 1% level; † jointly significant

TABLE A.4. Models of Gender-Specific Mortality

Variable	Model 13.	Model 14.	Model 15.
	male	*female*	*female*
pop15_64	-12.01 (3.36)**	-7.32 (1.56)**	-7.16 (1.62)**
pop65	-24.65 (5.25)**	-19.33 (2.23)**	-19.55 (2.23)**
mfratio	-31.91 (3.61)**	-18.62 (1.47)**	-18.82 (1.41)**
publiche	44.24 (9.31)**	-	-
he	-27.62 (5.38)**	5.37 (1.91)**	5.48 (1.93)**
dtloggdp	-184.40 (18.63)**	-125.29 (6.80)**	-124.99 (6.99)**
dtunemp	316.37 (66.9)**	28.20 (32.73)†	-706.16 (584.81)†
dtloggdpxdtunemp	-38.39 (6.73)**	-3.58 (3.42)†	-8.49 (6.05)†
yearxdtunemp	-	-	0.39 (0.32)†
intercept	6930.81 (407.05)**	4296.34 (162.13)**	4305.24 (159.59)**
R^2	0.873	0.93	0.93
N	836	895	895

Notes: Standard errors in parentheses; * significant at the 5% level; ** significant at the 1% level; † jointly significant

FIGURE A.3. Quantile Plot of Model 1.

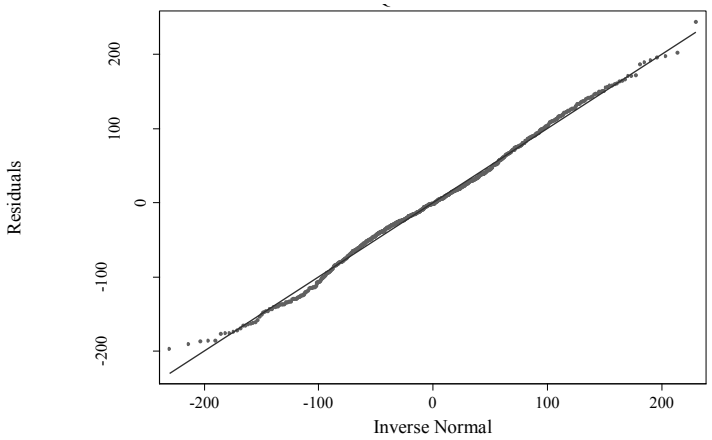


FIGURE A.4. Standardized Normal Probability Plot of Model 1.

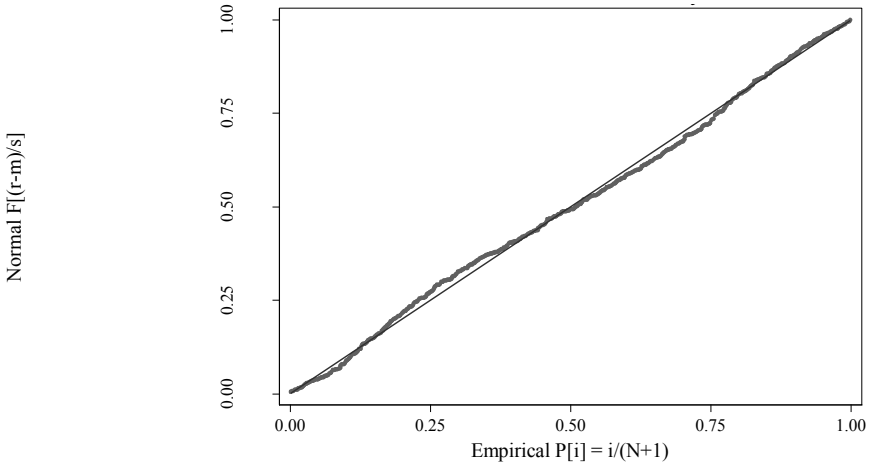


FIGURE A.5. Residual Plot of Model 1.

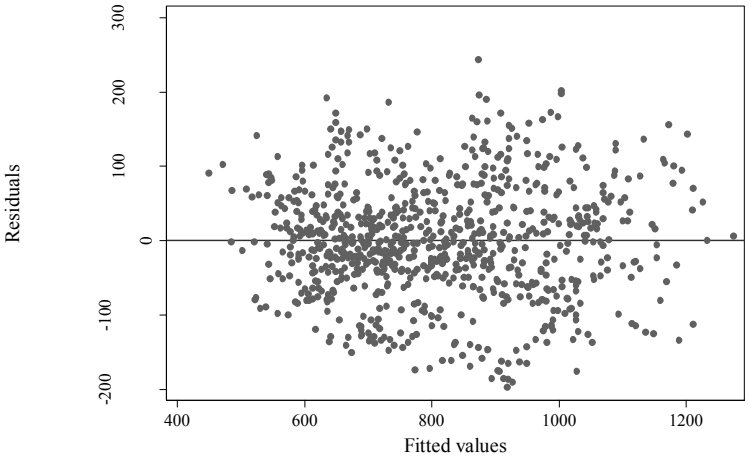


FIGURE A.6. Quantile Plot of Model 4.

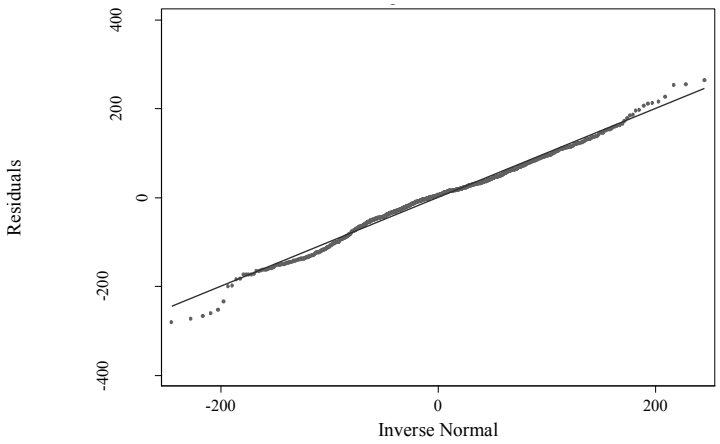


FIGURE A.7. Standardized Normal Probability Plot of Model 4.

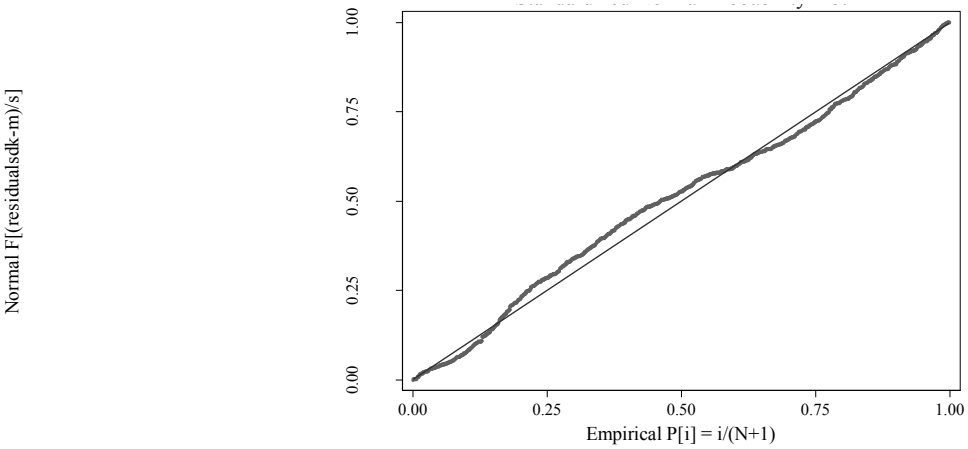


FIGURE A.8. Residuals Plot of Model 4.

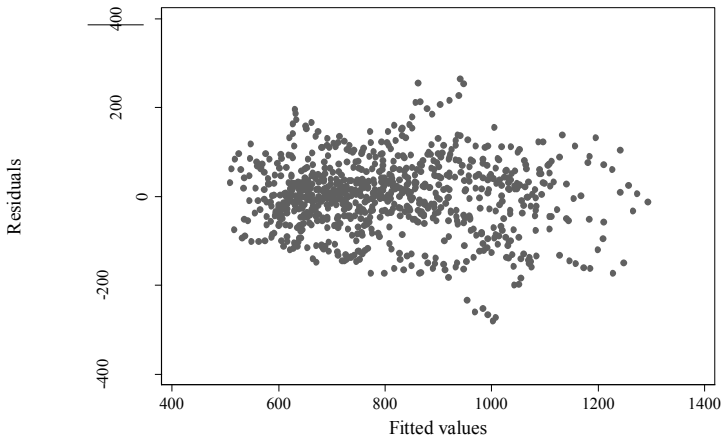


FIGURE A.9. Predicted Change in Male Mortality given Unemployment and GDP

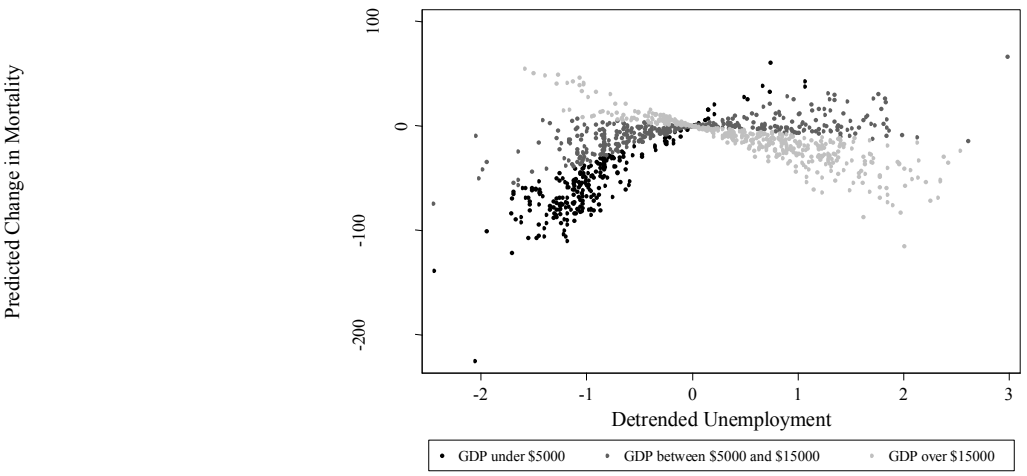


FIGURE A.10. Slope of Male Mortality against Unemployment given GDP

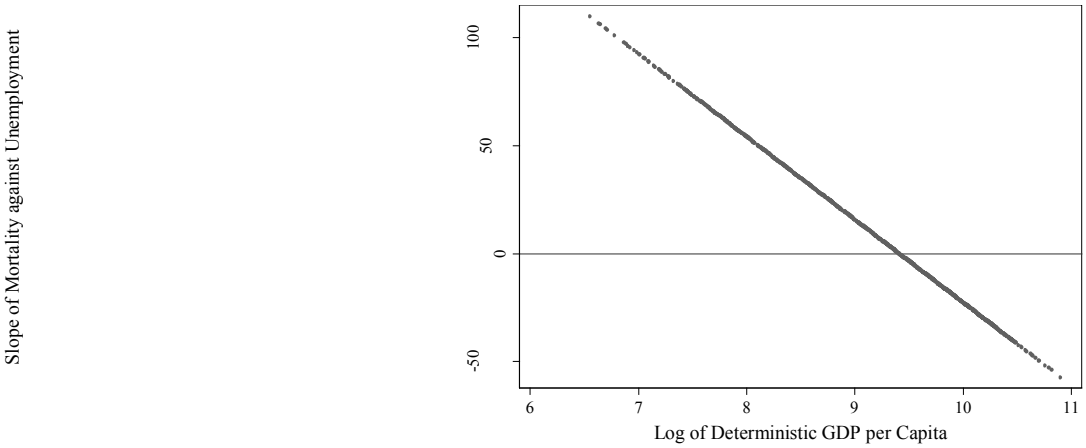


FIGURE A.11. Predicted Change in Female Mortality given Unemployment and GDP

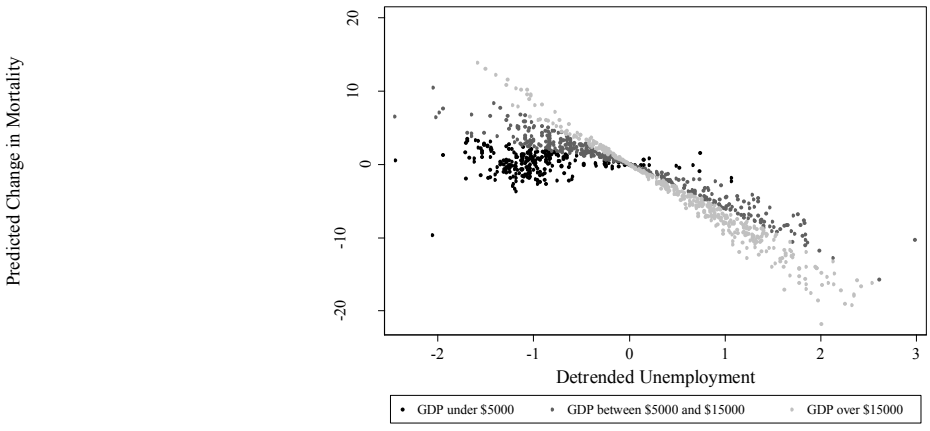


FIGURE A.12. Slope of Female Mortality against Unemployment given GDP

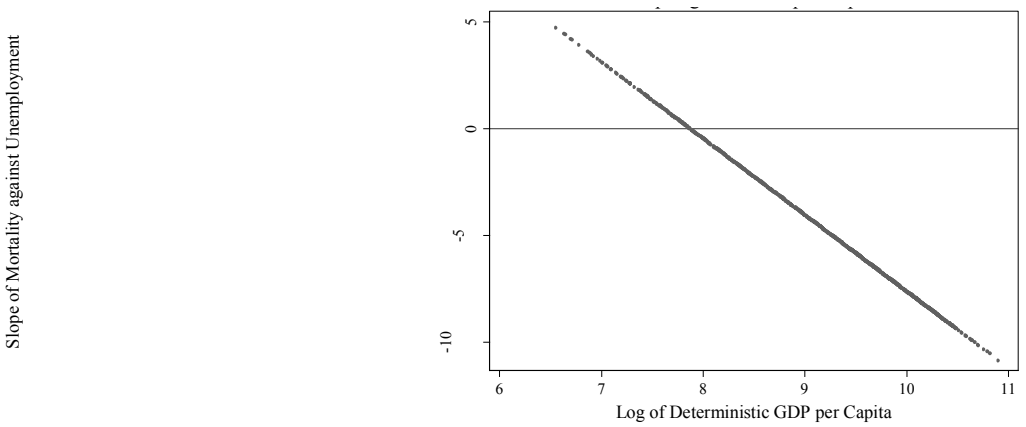


TABLE A.5. Estimated Tipping Points of GDP per Capita

Cause	GDP per capita estimate	95% confidence interval	
		Lower bound	Upper bound
suicide	$14,728.97	$13,496.84	$16,086.36
landtransport	$791.53	$373.01	$1,783.74
liver	$17,870.53	$15,459.60	$20,703.51
lungs	$15,132.69	$6,379.30	$39,169.20
*flupneu**	$8,173.63	$4,621.16	$14,978.09
heartattack	$7,453.60	$3,567.63	$16,548.62
mental	$11,663.01	$9,292.67	$14,720.75
cancer	$14,120.14	$12,694.42	$15,724.15

* *dtunemp* and *dtloggdpxdtunemp* were insignificant, so values may not be valid

DISCUSSION A.1. Testing for Higher Order Autocorrelation in Fixed-Effects Panel Data

Wooldridge (2002) developed a test for AR(1) temporal correlation, but not for higher order autocorrelation. What follows is a generalization of this technique to test for the presence of AR(m) processes for any m.

Model 2 estimates a fixed-effects model similar to that of equation (a), where y_{it} is the dependent variable and $\mathbf{x}_{it}$ is a vector of explanatory variables.

(a) $y_{it} = \beta_0 + \beta_{it}\mathbf{x}_{it} + v_{it}$, where t = 1,…,T and i = 1,..,N

Here, $v_{it} = c_i + u_{it}$. In Stata, this equation can be estimated and then u_{it}, the random error component, can be extracted by $v_{it} - c_i$. This variable, u_{it}, can then be lagged for an arbitrary number of years. In this implementation, six years was chosen. Then, equation (b) can be estimated, suppressing the constant and using robust standard errors, which Wooldridge (2002)

indicates is necessary when testing for autocorrelation in a fixed-effects model with panel data. This equation assumes a simple AR(m) process.

(b) $$u_{it} = \rho_1 u_{i,t-1} + \rho_2 u_{i,t-2} + \ldots + \rho_6 u_{i,t-6} + \varepsilon_t$$

As the real u_{it} are unknown $\hat{u}_{it}$, must be used instead. Wooldridge (2002) shows that for a fixed-effects estimator, using $\hat{u}_{it}$ is equivalent to using $\ddot{u}_{it} = u_{it} - \bar{u}_i$, the time-demeaned errors. He also shows, that in this situation, there is an expected correlation of $^{-1}/_{T-1}$ between $\ddot{u}_{it}$ and $\ddot{u}_{is}$ for any s $\neq$ t. Therefore, the estimated $\hat{\rho}$ should not be tested for whether they are significantly different than zero but whether they are significant different than $^{-1}/_{T-1}$. A similar result can be derived for unbalanced panels from the assumptions that (a) $E(u_{it}|\mathbf{x}_i,\mathbf{s}_i,c_i) = 0$ and (b) $E(u_i u_i'|\mathbf{x}_i,\mathbf{s}_i,c_i)=\mathbf{I}_T$ where t = 1,2,..,T and T is the maximum number of years for any panel (Wooldridge, 2002). Here, $\mathbf{s}_i$ is a vector of length T for year i, where 1 is in the t^{th} position if the observation exists in year t and zero otherwise. Then for years s $\neq$ t,

$$\mathrm{Corr}(\ddot{u}_{is}, \ddot{u}_{it}) = \frac{\mathrm{Cov}(\ddot{u}_{is},\ddot{u}_{it})}{\sqrt{\mathrm{Var}(\ddot{u}_{is},)}\sqrt{\mathrm{Var}(\ddot{u}_{it})}}$$

where $\mathrm{Var}(\ddot{u}_{is}) = \mathrm{Var}(\ddot{u}_{it})$

$$= \frac{\mathrm{E}(\ddot{u}_{is},\ddot{u}_{it})}{\mathrm{E}(\ddot{u}_{it}{}^2)} = \frac{\mathrm{E}[(u_{is}-\bar{u}_i)(u_{it}-\bar{u}_i)]}{\mathrm{E}[(u_{it}-\bar{u}_i)^2]} = \frac{\mathrm{E}(u_{is}u_{it})-\mathrm{E}(u_{it}\bar{u}_i)-\mathrm{E}(u_{is}\bar{u}_i)+\mathrm{E}(\bar{u}_i{}^2)}{\mathrm{E}(u_{it}{}^2)+\mathrm{E}(\bar{u}_i{}^2)-2\mathrm{E}(u_{it}\bar{u}_i)} = \frac{0-\frac{\sigma_u^2}{T}-\frac{\sigma_u^2}{T}+\frac{\sigma_u^2}{T}}{\sigma_u^2+\frac{\sigma_u^2}{T}-\frac{2\sigma_u^2}{T}}$$

where $E(u_{is}u_{it}) = E(u_{is})E(u_{it})$ since the null hypothesis assumes no serial correlation, or correlation between error term.

$$= \frac{-\sigma_u^2/T}{\sigma_u^2(1-\frac{1}{T})} = \frac{-1}{T-1}$$

The technique just described was implemented for Model 2. The results of the estimation of equation (b) are summarized in Table A.6. below.

TABLE A.6. Models of Higher Order Autocorrelation

Random Error Lag	Model A.		Model B.		Model C.		Model D.	
	ρ_i	Robust SE	ρ_i	Robust SE	ρ_i	Robust SE	ρ_i	Robust SE
u_{t-1}	0.606	0.055**	0.612	0.054**	0.633	0.053**	0.622	0.052**
u_{t-2}	0.245	0.059**	0.249	0.058**	0.227	0.055**	0.223	0.054**
u_{t-3}	0.195	0.068**	0.193	0.066**	0.169	0.063**	0.114	0.050**
u_{t-4}	0.007	0.071	0.000	0.066	-0.073	0.051	-	
u_{t-5}	-0.091	0.063	-0.101	0.050	-		-	
u_{t-6}	-0.010	0.050	-		-		-	
N	627		659		692		725	
Max(T_i)	40		41		42		43	
R^2	0.89		0.896		0.902		0.907	
H_0: ρ_i =	-0.026		-0.025		-0.024		-0.024	

Notes: * significant at the 5% level; ** significant at the 1% level

In Models A. through D., the p-values were calculated under the null-hypothesis that $\rho_i = \frac{-1}{T-1}$ where T is the maximum number of years per any country. This model indicates that only the random error components with lags for one to three years were significant. Removing both the error components with lags 4, 5 and 6 in Models B., C. and D., respectively, gives the same result: that a lag of three is optimal.

TABLE A.7. Correlations between Lagged Random Error Components and Explanatory Variables

Variable	*pop15_64*	*pop65*	*publiche*	*dtloggdp*	*dtunemp*	*dtloggdpxdtunemp*
u_{t-6}	-0.01	-0.18	-0.11	-0.02	-0.04	-0.04
u_{t-5}	0.00	-0.19	-0.11	-0.01	-0.02	-0.02
u_{t-4}	0.03	-0.18	-0.10	0.02	0.00	0.00
u_{t-3}	0.04	-0.19	-0.10	0.03	0.00	0.01
u_{t-2}	0.05	-0.19	-0.10	0.04	0.01	0.02
u_{t-1}	0.08	-0.19	-0.08	0.06	0.01	0.02
u_t	-0.01	-0.12	-0.07	-0.01	0.02	0.03
u_{t+1}	0.01	-0.13	-0.06	0.00	0.01	0.02
u_{t+2}	0.02	-0.13	-0.04	0.03	0.02	0.03
u_{t+3}	0.03	-0.14	-0.04	0.03	0.02	0.03
u_{t+4}	0.03	-0.15	-0.04	0.04	0.03	0.04
u_{t+5}	0.05	-0.15	-0.02	0.07	0.04	0.05
u_{t+6}	0.06	-0.15	-0.01	0.08	0.06	0.06

Notes: The u_{ti} represent the lagged random component of the error terms of Model 2.

APPENDIX B. Exploratory Data Findings

TABLE B.1. Correlations between Year and Unemployment by Country

Australia	0.057
Austria	0.076
Belgium	0.013
Canada	0.001
Czech Republic	0.126
Denmark	0.045
Finland	0.070
France	0.066
Germany	0.084
Greece	0.097
Hungary	0.110
Iceland	0.085
Ireland	-0.004
Italy	0.019
Japan	0.054
Korea	-0.044
Luxembourg	0.107
Mexico	0.094
Netherlands	0.031
New Zealand	0.088
Norway	0.062
Poland	0.125
Portugal	0.053
Slovak Republic	0.122
Spain	0.066
Sweden	0.074
Switzerland	0.118
United Kingdom	0.035
United States	-0.035

TABLE B.2. Summary Statistics of Variables Used

Variable	Mean	Standard Deviation	Min	Max
unemp	5.71	4.17	0	23.9
dtunemp	-2.37E-08	0.99	-2.45	2.99
gdp	13057.95	10218.32	496	59176
mfratio	96.73	3.07	87.2	105.6
pop0_14	23.42	6.33	13.6	47.2
pop15_64	64.80	3.67	48.2	71.9
pop65	11.79	3.48	2.9	20.8
publichce	1574.14	874.83	73	5759
hce	1138.32	625.44	16	3301
publiche	6.93	2.09	1.5	15.3
he	5.05	1.73	0.7	8.9
ubppp	239.62	213.12	0	1080
ubgdp	1.30	1.07	0	5.3
alcohol	10.38	3.58	2.5	20.8
fertility	2.13	0.87	1.08	7.26
fat	120.95	28.11	14.4	170.8
mortality	868.22	196.05	428	1358.8
suicide	13.49	6.84	0.9	42
landtransport	1470.69	2708.7	2	16084
liver	13.58	10.52	0	69.4
lungs	17.94	11.93	0.1	71.8
flupnu	32.06	20.28	3.9	154.4
heartattack	79.59	40.98	5.5	214.7
mental	7.61	6.29	0	38.2
cancer	181.43	27.49	83	259.4
male	1104.3	237.96	592.3	1668.9
female	690.76	172.47	303.7	1177.4

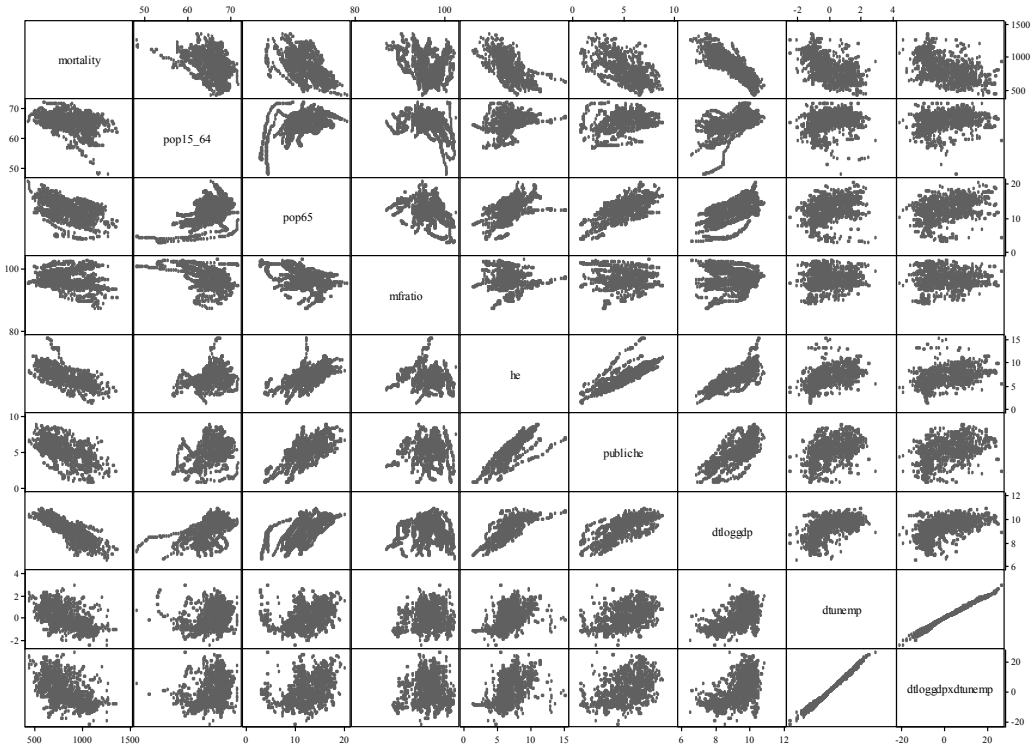
TABLE B.3. Correlations between Explanatory Variables

Variable	*dtunemp*	*dtloggdp*	*dtloggdpx dtunemp*	*mfratio*	*pop0_14*	*pop15_64*	*pop65*	*he*	*publiche*	*hce*	*publichce*	*logubgdp*	*logubppp*
dtunemp	1.00												
dtloggdp	0.05	1.00											
dtloggdpxdtunemp	1.00	0.06	1.00										
mfratio	0.12	0.23	0.12	1.00									
pop0_14	0.03	-0.38	0.01	0.46	1.00								
pop15_64	-0.15	0.15	-0.15	-0.25	-0.62	1.00							
pop65	0.09	0.36	0.10	-0.39	-0.77	-0.02	1.00						
he	0.04	0.58	0.05	-0.04	-0.24	-0.01	0.32	1.00					
publiche	0.14	0.46	0.16	0.02	-0.32	-0.13	0.51	0.58	1.00				
hce	0.00	0.82	0.02	0.14	-0.22	-0.01	0.30	0.87	0.52	1.00			
publichce	0.09	0.83	0.10	0.20	-0.29	-0.08	0.44	0.66	0.78	0.86	1.00		
logubgdp	0.38	-0.04	0.37	0.09	0.01	-0.16	0.12	0.06	0.33	-0.04	0.11	1.00	
logubppp	0.36	0.41	0.37	0.19	-0.16	-0.07	0.27	0.32	0.51	0.33	0.47	0.89	1.00

TABLE B.4. Correlations between Dependent and Explanatory Variables

Variable	*dtunemp*	*dtloggdp*	*dtloggdpx dtunemp*	*mfratio*	*pop0_14*	*pop15_64*	*pop65*	*he*	*publiche*	*hce*	*publichce*	*logubgdp*	*logubppp*	*alcohol*	*fat*	*fertility*
mortality	-0.17	-0.74	-0.18	-0.40	0.29	-0.09	-0.30	-0.38	-0.38	-0.57	-0.63	-0.03	-0.37	0.25	-0.14	0.31
male	-0.14	-0.74	-0.16	-0.45	0.22	-0.02	-0.27	-0.39	-0.36	-0.58	-0.62	0.01	-0.33	0.31	-0.12	0.19
female	-0.19	-0.70	-0.21	-0.35	0.33	-0.15	-0.31	-0.36	-0.38	-0.53	-0.60	-0.07	-0.40	0.18	-0.15	0.39
suicide	0.01	-0.02	0.01	-0.26	-0.05	0.16	-0.07	0.03	0.24	0.00	0.09	0.06	0.06	0.22	0.12	-0.17
landtransport	-0.08	0.16	-0.08	-0.15	0.06	0.04	-0.11	0.55	-0.13	0.48	0.06	-0.27	-0.18	-0.06	-0.06	0.10
liver	-0.12	-0.38	-0.13	-0.65	-0.25	0.31	0.07	-0.12	-0.11	-0.29	-0.31	-0.15	-0.30	0.27	-0.22	0.13
lungs	0.07	-0.55	0.06	-0.19	0.26	-0.13	-0.23	-0.31	-0.15	-0.36	-0.30	0.06	-0.19	0.13	-0.13	0.40
flupneu	0.03	-0.12	0.03	0.13	0.36	-0.39	-0.15	-0.27	-0.09	-0.17	-0.09	0.02	-0.05	-0.32	-0.25	0.46
heartattack	0.12	-0.30	0.11	0.16	0.46	-0.44	-0.24	-0.18	-0.08	-0.24	-0.22	0.29	0.10	-0.09	0.18	0.28
mental	0.05	0.42	0.06	0.18	-0.05	-0.11	0.16	0.15	0.24	0.33	0.40	0.30	0.48	-0.19	0.10	0.08
cancer	-0.07	-0.25	-0.08	-0.19	0.09	0.07	-0.17	-0.13	0.04	-0.23	-0.20	0.17	0.04	0.36	0.40	-0.32

FIGURE B.1. Scatter Plot Matrix of Variables Used



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