



# **The Impact of Unemployment on Heart Disease and Stroke Mortality in European Union Countries**

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## **I. Summary**

This paper examines the relation between unemployment and CVD mortality in European Union countries during the first decade of the 21<sup>st</sup> century. Two separate studies are summarized, focussing on increased heart disease and stroke mortality rates as potential outcomes of the greatly extended unemployment rate during 2000 – 2010 and especially the Great Recession of 2007 – 2009.

Unemployment rates and mortality from two cardiovascular illnesses are viewed in pooled cross-sectional analyses over the range of EU countries. In addition to the unemployment rate, other economic variables, expressing the Recession, are studied in relation to cardiovascular mortality over multiple years in a pooled cross-sectional time-series analysis with random effects.

The principal finding is that increases in the unemployment rate are related to increased heart disease and stroke mortality. Controlling for other labour market variables, such as labour force participation, as well as gross domestic product (GDP) per capita—the principal indicator of recession and economic growth.

In the first study of heart disease, the association with unemployment rates is analysed contemporaneously and at lags of 1, 2, 3, 4 and 5 years. Without adjusting for any control variables, only the unemployment rate relationships lagged at years 1, 2, 3, 4, 5 are statistically significant. With adjustment for GDPpc, however, the contemporaneous (0 lagged year) for the unemployment rate also becomes significant and positive. The unemployment rate lagged 2 years shows the strongest relation to IHD mortality rates. This is confirmed for age groups 35-44, 45-54, 55-64, 65-74, 75-84 and over 85. The relations of the unemployment rate to IHD mortality rates can be seen using age-specific unemployment rates for the age groups 35-44 to 65-74. The relations between the unemployment rate and the IHD mortality rates remain significant in the presence of as few as one or as many as eleven control variables. Variables under control include GDPpc, Southern Europe (binary variable), labour force participation, self-employment, national measures of poverty and smoking prevalence. Labour force participation and self-employment are considerably stronger labour market predictors (inverse) of IHD mortality rates than is the unemployment rate (positive).

These findings are consistent with the epidemiological literature which shows positive relations between unemployment and cardiovascular (CVD) mortality, and inverse relations between income and CVD mortality. These findings are also consistent with national level time-series analyses of unemployment and other macroeconomic variables in relation to CVD during the 20<sup>th</sup> century. The evidence is clear that the unemployment rate has been an important risk factor for IHD mortality since the start of the 2008-2009 Recession in Europe.

The second study of the impact of unemployment rates on stroke mortality replicates and extends the analysis of the impact of unemployment rates on heart disease mortality (for 28 EU countries during the period preceding and covering the Great Recession 2000 – 2011). In the second study, stroke mortality rates were found to increase as unemployment rates climb, with lags ranging over 2-4 years for both sexes and age groups over 45 years. The peak lag of 3 years following unemployment is slightly higher in its prediction of stroke mortality than the initial 2-year lag.

For males, the range of impact of the unemployment rate covers the working ages 45 – 64, whereas for females it covers those ages as well as 65 -84! It is similarly noteworthy that the impact of the labour force variables, unemployment rates and labour force participation rates, have a greater impact on stroke mortality in women. This is somewhat surprising from the classic social science perspective that males are thought to have a greater intrinsic involvement with careers, earnings and the economy in general than females. In the case of stroke outcomes, this is not borne out. Females show an effect of the labour force variables that occurs both earlier and later in life than males.

This study also finds that the labour force participation rate, a more comprehensive business cycle indicator that takes discouraged workers into account, predicts reduced stroke mortality contemporaneously with elevated labour force participation—i.e., without any lag (within the course of a year). The labour force participation rate shows at least twice the impact on stroke mortality of the unemployment rate (i.e., in terms of effect size). Additionally, GDP per capita, the most prominently used measure of the presence of recessions (or economic recoveries) by economic specialists, is very strongly related to stroke mortality declines and has around three times the impact of the unemployment rate. GDP per capita can be shown to influence stroke mortality declines contemporaneously—i.e., with zero year's lag—and with further lags that can be estimated over 1 – 10 additional years (at least). This means that the impact of GDP per capita on stroke mortality also involves a secular trend of at least 10 years duration.

The fact that unemployment rate increases predict elevated stroke mortality rates beyond the usual working life (i.e., over age 65) for women (including ages 65 – 84), and that the labour force participation rate predicts increased stroke mortality from ages 65 to over 85 (!) for each sex demonstrate powerful “spread effects” of these labour force variables. This means that the labour force variables influence health well beyond the normal working life and therefore have an especially large effect on life expectation in older ages.

These findings on the effects of labour force variables—especially the unemployment rate—on increased heart disease and stroke mortality is consistent with the epidemiological literature on unemployment in relation to cardiovascular mortality in individual-level epidemiological studies. This study raises the question of whether it is cost-effective to reduce unemployment, or its financial implications, in order to minimize health and health care costs.

## **II. Introduction**

Recently, an analysis was performed for the European Commission examining the relation between unemployment rates and heart disease mortality in EU countries over the period leading up to and including the Great Recession (2000 – 2010). The importance of singling out unemployment during the Great Recession is that it has an intrinsic meaning for policy makers as well as the general public. There is also an extensive literature in epidemiology at the individual level demonstrating this relation covering the past 40 years. In this document, analysis is presented of a replication and extension of this statistical relation to encompass stroke mortality among EU countries over the period 2000 – 2011.

The central problem of this analysis is to elucidate this relationship between unemployment and stroke mortality for the macro economy, which makes it directly pertinent to national and international economic and social welfare policy. There are three econometric issues to be dealt with in this analysis. The first is that it is important to control for gross domestic product per capita (GDP pc), which involves long-term economic growth, and is also the most widely used indicator of the presence of recessions. In fact, the relation between GDP and the unemployment rate is so fundamental and causal that classic estimates have been made of the precursor status of GDP growth to unemployment rate reduction (Okun's Law).

The second issue is that the officially calculated unemployment rate, as estimated by EUROSTAT and the ILO, as well as the US Census, omits crucial aspects of job loss. Most important is that the measurement of unemployment requires that the officially unemployed must be physically capable of, and actively seeking, work. However, many who lose jobs, but become discouraged about finding work, stop seeking work and are thus no longer officially counted as unemployed. Yet, these longer-term but discouraged workers are most vulnerable to psychological stress and poverty and thus to subsequent cardiovascular reactions.

A third issue with the unemployment rate as used in national statistics, lies in its implicitly "natural" inverse (i.e., counter-cyclical) relation to the business cycle. However, as the business cycle moves into the upturn phase, the unemployment rate actually increases, because, coming out of the recession, some people who are not in the labour force, enter the labour force expecting to find work but until they do find work remain unemployed. This is one reason the unemployment rate is known as a lagging indicator of the business cycle. For this reason it is sometimes assumed that the zero time lag (i.e., exactly contemporaneous) relation of the unemployment rate to a damaging health outcome (such as stroke) is often unreliable.

In analysing unemployment, specifically job loss, in relation to stroke mortality, it is therefore also logical to utilize the unemployment rate together with the labour force participation rate. In so doing, it is also important, as indicated above, to control for GDP per capita. But clearly, the three economic indicators—GDP per capita, unemployment rate, and labour force participation rate—are most appropriately placed at different lags, because they are causally related to each other, and at the same year of lag—e.g. lag zero—induce multi-collinearity.

Aside from such econometric issues associated with the nature of economic indicators, there are equally important considerations that have to be taken into account when economic indicators are used to predict health outcomes. Principal among these is the need to account for other health risk or beneficial factors that bear an important relation to the outcome in question. In the case of stroke mortality, as in other types of cardiovascular disease mortality, smoking is an important damaging predictor; similarly, vegetable fat consumption (and that of vegetable consumption generally), is a potentially beneficial factor.

Other factors that ideally should be brought under statistical control in order to focus on the GDP per capita and employment related variables, as indicators of recession, are



measures of chronic poverty. These are especially interesting when one examines the economic situation of European populations, since, in principle, they are generally politically well-protected by highly developed social welfare systems. Nevertheless, an indicator of a chronically very low living standard is the absence of complete access to potable water and sanitation. Further, an important measure of the extent of intergenerational poverty, and difficulty of social mobility, is the adolescent fertility rate. In the latter case, both adolescent mothers and their offspring are frequently subject to low educational attainment, low-wage employment and life-long weak earning capacity as well as high unemployment rates.

In the present analysis, the project began in an attempt to replicate the findings on the relation of unemployment to heart disease mortality to observe whether they apply to mortality due to stroke (or cerebrovascular disease). It is assumed in this paper that it is logical to try a nearly identical modelling process in the analysis of stroke mortality, compared to that which has been successful in the models prepared for heart disease mortality. The rationale behind this approach regarding the similarities of models predicting the two causes of death, rests on the assumption that the two phenomena are closely related. These assumptions are based on three points of similarity: (1) similarity of epidemiological predictors; (2) similarity of biological mechanisms; and (3) similarity of dietary and environmental risk factors.

### **III. Literature background**

#### **Socioeconomic status and population health**

European and United States' epidemiologic studies have shown since the 1970s that socioeconomic status (SES) is a stable risk factor for illness and mortality in individual persons (Galobardes, Smith and Lynch, 2006; Frankel, Smith, and Gunnell, 1991; Kaplan and Keil, 1999). Since heart disease, more specifically IHD, also referred to as Coronary Heart Disease, is the largest segment of mortality in industrialized countries, it is a central component of the more general, and nearly universal, relation between SES and overall mortality (and severe morbidity) – also referred to as the “social gradient” in disease. The higher the SES the lower the morbidity and mortality rates.

The significance of SES involves its components – income, education, occupational skill-level and their causal interrelations. Thus, parental SES tends to predict the educational level of children. Children's education, in turn, predicts their occupational skill-level, which then gives rise to their initial and permanent income levels. These epidemiological studies, also showing a positive relation between unemployment and CVD illness has been confirmed since the 1970s through time-series analysis of CVD mortality in the US and nine additional European countries (Brenner and Mooney, 1982; Gallo, Teng, Falba, Kasl et al., 2006; Fillate, Johansen, et al., 2003; Hendriksson, Lindblad, Agren et al., 2003; Morris, Cook, Shaper, 1994; Sorlie and Rogot, 1990; Crombie, Kenicer, et al. 1989; Moser, Fox, et al., 1986).

At the national (or macroeconomic) level, recession influences overall employment (and unemployment) and the creation of new employment via entrepreneurship, especially self-employment. Further, under conditions of unemployment lasting more than e.g. six months, it is common for the unemployed to leave the labour force altogether, as “discouraged workers,” never to return to the labour force, or to subsequently accept positions at much lower skill levels, wages or hours (i.e., underemployment). Thus, longer-term unemployment has implications for decreased labour force participation and underemployment. Further, national level unemployment rates can be mitigated by heightened self-employment, but only if family or other financing is available. Thus, the impact of unemployment on health should ideally be estimated in the context of GDP per capita, labour force participation and self-employment (Brenner, 1971; 1987b; 1997; 2005; 2012).

#### **Impact of unemployment on population health: national versus individual-level studies**

Unemployment is an important risk factor for mortality and morbidity—especially if the unemployment is of long duration (Tausky et al., 1967/8; Hallsten et al., 1999; Kasl et al., 2000). Brenner (2011) reviewed European Commission studies dated 1998-2004 and examined the time-series relations between mortality and economic growth and unemployment. These studies covered the 15 original European Union countries and the US over the period 1960-2000 (Brenner, 1979; 1984; 2000; 2002) and are extensions of original work done in the 1970s and early 1980s (Brenner 1984). Short-term positive relationships between unemployment and mortality are also seen within a year following increased unemployment rates. In a pooled cross-sectional study of metropolitan areas of the US, with a lag of two years following increased unemployment, and inclusion of poverty and income inequality as control variables, the standard positive relation was found for heart disease, stroke and homicide (Merva and Fowles, 1999), which is consistent with the findings of virtually all epidemiological studies in Europe and North America over the last thirty years (Kasl, 2000). In another pooled cross-sectional study of the US, based on US states, without any lags and without the usual epidemiological controls, these inverse relations were not replicated (Ruhm, 2000). Most recently, studies based on the Health and Retirement Survey have shown strong effects of unemployment on heart disease mortality (Gallo et al, 2004, 2006; Dupre et al., 2012) as well as overall

mortality (Noelke et al., 2014). Over the past decade, several European studies have been concentrating on the impact of firm restructuring with downsizing (i.e., job losses). The studies have uniformly found increased unemployment to be related to increased morbidity and mortality. In a technical report to the European Commission (Brenner, 2009; 2011) reviewed the empirical literature and identified unemployment as a key socioeconomic determinant of health, particularly for men. Within the last year two studies have shown marked increases in depression in multi-country studies of firm downsizing in Europe (Brenner et al., 2014) and industrialized countries (Reeves, McKee, Stuckler, 2014).

## **IV. Hypotheses**

### **Key Economic Variables**

- A higher unemployment rate predicts higher levels and increases in the cardiovascular mortality rate. Levels refer to cross-sectional heart disease and stroke mortality rate differentials among EU countries. Changes refer to trends and fluctuations over 2000 – 2010 in the case of heart disease and 2000 – 2011 for stroke. Unemployment refers to persons experiencing job loss and new entrants into the labour market who are seeking, and are capable of, work.
- A higher labour force participation rate predicts lower levels and decreases in the cardiovascular mortality rate. The “labour force” includes both employed and unemployed individuals. Unemployed persons who stop seeking work (and usually do not receive unemployment insurance benefits) because they do not believe they could find (suitable) employment are referred to as “discouraged” workers and are not counted in the labour force. The emotional situation of “discouragement” in job losers who no longer seek work (and the related loss of subsidized income), as well as the fact that discouraged workers tend to be among the uncounted long-term jobless would predict a larger and more immediate effect on stroke mortality.
- GDP per capita predicts lower cardiovascular mortality rates in the short, medium and long-term. GDP per capita is the basic indicator of national income and wealth, as well as the level of economic development. Economic growth is a function of the development of physical and human capital and thus of innovation, education, and knowledge development. European societies are heavily dominated by service industries. GDP per capita is the basis of expenditure on health, education and welfare, social insurance and investments in new technologies that elevate worker and environmental safety. Thus, GDP per capita is a fundamental source of increased population health and life expectancy. It is also the primary (positive) indicator of business cycles and thus, inversely, of recession. It is therefore also a principal predictor of (reduced) unemployment rates and therefore of economic instability.

### **Control variables**

- Chronic poverty  
Chronic poverty should predict higher cardiovascular mortality rates. Persons in chronic poverty are epidemiologically at higher risk for cardiovascular disease and its outcomes. This is assumed to result from stress which could directly influence psychophysiological reactions, or contribute to smoking, poor diets and exposure to noxious environments.

In this study, an indicator of regions with extraordinarily low levels of income are those with limited access to potable water and sanitation facilities. This condition

is relatively unusual but is nevertheless measurable to some degree in several EU countries.

A second and, it is assumed, more prevalent indicator of chronic poverty is the adolescent fertility rate. Both mothers and children in this circumstance are known to have relatively low family incomes and education levels. Thus, initial family poverty rates are often maintained through the subsequent generation involving low-skilled jobs and relatively high long-term unemployment rates as well as unstable low-wage employment.

- Smoking and diet

Smoking prevalence is predicted to increase cardiovascular mortality, but the time lag is uncertain. It is possible that reduced smoking prevalence would predict decreases in heart disease and stroke mortality in highly vulnerable age groups—in the short term. However, there is greater consensus in the literature on the impact of smoking in cohorts, ranging as long as 20 years in duration. In this study smoking prevalence is used as a control only in short-term time lags, given data availability and the requirement to focus on unemployment and labour force participation as key predictor variables.

Dietary factors potentially influencing cardiovascular mortality have traditionally tended to be focused on fat consumption and vegetable consumption. Saturated fat consumption has been identified as a harmful risk factor, whereas vegetable fat consumption (especially as identified in the Mediterranean diet and including olive oil) has been found to reduce cardiovascular illness, including stroke. Fruit and vegetable consumption in general has also been found epidemiologically to be an important source of reduction of chronic disease mortality. It is therefore predicted that vegetable fat consumption, even at short-term lags, will be seen to reduce heart disease and stroke mortality rates.

## **V. Research Method**

This study uses pooled cross-sectional time-series analysis (often referred to as panel analysis) as the basis for modelling the effect of unemployment rates on heart disease and stroke mortality rates in European countries. This method combines traditional cross-sectional analysis and temporal analysis over multiple years—in this case 2000 – 2010 for heart disease and 2000 – 2011 for stroke. The analyses thus include space and time components simultaneously. The cross-sectional aspect of this method allows a comparison, at a point in time, among countries, of how different levels of unemployment rates, under different national conditions of severity, duration, welfare compensation, Active Labour Market Policies and health care availability, can influence cardiovascular mortality. In fact, it is often assumed, that, given the extensive social safety net and welfare state systems of European countries, sufficient protection is available to fundamentally prevent unemployment rates from influencing mortality rates in a significant manner.

Multiple years (of cross-sectional analysis) are included for several reasons: (1) to assure that the results of any specific year's analysis are not unusual (i.e., idiosyncratic) with respect to the relation of unemployment to heart disease and stroke mortality. (2) The analysis also includes a time-series analytic component—as well as time trends, cycles, and other fluctuations in unemployment and heart disease/ stroke mortality over 11 years. This especially allows us to observe much of the impact of the Great Recession prior to its onset and during its occurrence—in terms of its effects on unemployment and its health implications. (3) Several control variables are included in the model. These take account of overall GDP—the most powerful trend affecting mortality rates—and most frequently used measure by economists to indicate the starting and ending dates and severity of recessions.

Another control variable is labour force participation, which supplements the unemployment rate by taking account of persons who have lost employment, but may no

longer be counted among the unemployed because they have stopped seeking work due to discouragement and have, thus, through standard counting methods, left the labour force. Also under control is a measure of chronic, intergenerational poverty such as exhibited by the adolescent fertility rate. And finally, smoking prevalence is controlled for—as the single most important behavioural risk factor influencing cardiovascular mortality. These variables are placed in the model to assure that the measured effects of unemployment are really not due to other factors that are associated both with unemployment and, independently, with cardiovascular mortality.

For example, a population subject to chronic poverty is likely to be vulnerable to unemployment as well as to heart disease and stroke. This would raise the question as to whether the effects attributed to unemployment are actually partly, or entirely, due to chronic poverty. Similarly, smoking is more frequent among the unemployed and is a principal risk factor for heart disease and stroke. Thus, one would want to assure that the cardiovascular effects attributed to unemployment are really not a complete or partial by-product of smoking.

In order to include these control variables in the predictive models for heart disease and stroke mortality, we require sufficient degrees of freedom to perform adequate tests of significance. Clearly, there are important restrictions in the degrees of freedom available from one year of cross-sectional analysis with a sample size of 28 units (i.e., countries). Using 11 years of cross-sections provides the expanded degrees of freedom.

### **Random versus fixed effects**

Two standard methods of modelling with a pooled cross-sectional time-series designs are commonly used. The choice of method depends largely on the nature of the variables used with respect to their time-series properties. In brief, if the model contains variables that do not vary at all (e.g. dichotomous) or are “slow moving” in the sense that they vary little from one time period to another or have very strong time trends, then the random effects method is required (Wooldridge, 2002).

### **Deductive logic of model construction**

We start with the most ubiquitous and pervasive finding in all of modern epidemiology, referred to as the “social gradient” in health. This finding is that increased socioeconomic status (SES) measured by income, educational level, occupational skill level (in graduated scales)—and most recently by unemployment rates (inverse relation)—is closely related to lower levels of illness and mortality, regardless of diagnosis, age, sex, and nationality (Galobardes, Smith and Lynch, 2006; Frankel, Smith, and Gunnell, 1991; Kaplan and Keil, 1993; Brenner and Mooney, 1982; Gallo, Teng, Falba, Kasl et al., 2006; Fillate, Johansen, et al., 2003; Hendriksson, Lindblad, Agren et al., 2003; Morris, Cook, Shaper, 1994; Sorlie and Rogot, 1990; Crombie, Kenicer, et al. 1989; Moser, Fox, et al., 1986).

These findings are at micro—or individual—level. The position taken in this paper that findings at the macro-level—if they are to represent, and elaborate, well-established causal relations, cannot contradict findings at the micro (individual) level, at which the effects of unemployment and mortality are thought to occur.

Continuing with this logic, if declines in income, or increases in unemployment, are found to occur at the individual level, or aggregated to the level of the population, then it follows that declines in population health should similarly occur. Such declines in population health should involve the major diagnostic categories of illness and mortality. Heart disease and stroke are the first and third most important cause of illness and mortality in industrialized societies, including all EU member countries.

In summary, given the extensive individual-level epidemiologic evidence of the inverse relation of income and employment to cardiovascular illness and mortality, it follows that national increases in unemployment and the declines in income are plausibly followed by increased cardiovascular illness and mortality rates at the national level.

In order to construct a comprehensive model that follows the logic of the universal SES-health relation, it would be important to include as many socioeconomic variables as practical on economic and statistical grounds. These would involve, at the national level, unemployment rates, labour force participation rates and GDP per capita as having short- to medium-term impacts on cardiovascular mortality.

Labour force participation rates would be included (in addition to unemployment rates) to assure that the potential findings based on unemployment rates would not be distorted as a result of the fact that measured unemployment does not include longer-term job losers who, through discouragement at not finding new employment, would leave the labour force rather than remain (technically) unemployed.

In the overall heart disease and stroke models, we also wish to control for population trends that influence poverty rates, and the extent of long-term trends of low-income employment, and unemployment, due to such factors as family instability. A prominent factor of this type is adolescent fertility (Hulme, D, Moore, K and Shepherd, A, 2001; OECD Social Policy Division, 2010).

Further controls to be tested in the heart disease and stroke models would involve income inequality—which could also reflect poverty (Sen, 1986)—and which has been argued to negatively influence health (De Maio, 2007) although this remains controversial.

A final type of control for potential confounding would be the most important behavioural risk factor associated with cardiovascular mortality, namely smoking prevalence. It should also be mentioned that there are other less well-established behavioural risk factors for cardiovascular illness such as high alcohol consumption, poor nutrition, and overweight. However, the major socioeconomic control variables (GDP per capita, adolescent fertility, Gini), should in principle adjust for these potential behavioural risks, since they are associated with low SES.

### **Further deductive approach: Replication of the heart disease model**

The deductive approach outlined above, in which a macro (national-level) model was developed from a large literature on health risks, based on micro (individual-level evidence), was used in an initial report to the EU based on a study investigating the link between unemployment and heart disease mortality rates in EU countries (Brenner, 2013).

Extending this deductive logic further, it was assumed that stroke mortality would be predicted by a similar model to that which successfully predicted heart disease mortality. The basis of this assumption is that stroke and ischemic heart disease have essentially the same behavioural risk factors (e.g. psycho-social stress, diet, hypertension, arteriosclerosis, aging) since they are part of the same cardiovascular disease process, which also includes kidney disease. (WHO, Media Centre Cardiovascular diseases, 2015)

Thus, in the present study, the heart disease model was replicated using the same independent variables of unemployment, labour force participation and GDP per capita and controlling for similar potential confounders. The stroke model also took account of the fact that GDP per capita needed to show a lag (to stroke mortality) that involves a minimum of five years, in order that multi-collinearity not render insignificant the relation to unemployment (at lags 0 – 3) due to Okun's law by which GDP is strongly inversely related to unemployment rates (especially in recessions). It had been found that the

optimal heart disease mortality model involved the unemployment rate at two year lag and GDP per capita at a seven year lag.

### **Adding GDP while taking account of Okun's Law**

A minimal model for heart disease or stroke mortality in industrialized countries would at least include one major proxy variable for aggregate population SES in addition to the unemployment rate. At the national level GDP per capita is the closest we come to an aggregate variable that represents the national standard of living as well as the sources of national investment. As an aggregate measure, the GDP per capita does considerably more than measure the "average" of SES for a national population. It also signifies the potential for investment in national health services, education and social welfare, including pensions and support of the disabled.

The great problem, however, in adding GDP per capita to a model of mortality already involving the unemployment rate is that, like the unemployment rate itself, the GDP is a fundamental indicator of business cycle fluctuations. Thus, concurrently – i.e., at zero years' lag – GDP per capita and the unemployment rate will be directly and robustly inversely related to each other. The principal reason is that even decline in the growth rate of GDP per capita has a causal relation to increased unemployment due to the decline in demand for goods and services. Declines in consumer demand signal to employers that fewer employees will be needed for firms to remain profitable at their previous rate.

So regular is the relation between GDP per capita and the unemployment rate that a rule of thumb stating the extent of GDP growth that is needed to produce a one percent decrease in the unemployment rate has been in use since the 1960s. It is referred to as Okun's Law, offered by Arthur Okun who proposed the relationship in 1962. The relationship varies depending on the country and the time period under consideration. In Okun's original statement of his law, a two percent increase in output corresponds to a one percent decline in the rate of cyclical unemployment; a 0.5 percent increase in labour force participation; a 0.5 percent increase in hours worked per employee; and a one percent increase in output per hours worked (labour productivity).

We therefore have the situation that GDP per capita declines can have a damaging effect on health through their impact on the unemployment rate. In that case, GDP per capita is the more distal causal variable while unemployment is the more proximal cause of health decline. In this scenario, however, unemployment is an intermediate component of the more general relation of GDP to health. This common cyclical concurrence produces difficulty for adding GDP per capita to the unemployment rate in order to produce a more complete and plausible model for e.g. IHD.

Our solution is based on the fact that the relationship of GDP per capita to cardiovascular mortality occurs over multiple years – i.e., there are strong lengthy, medium- and short-term relations of GDP per capita to cardiovascular mortality. This is because GDP per capita is a foundational variable which includes aggregate SES at the national level. This relation involves implications for long-term investments, such as in education, health care and infrastructure, as well as short-term consequences for personal household and government expenditures.

On the other hand, the most important effects of unemployment on health are empirically observed at 2-4 year's lag, and especially at a two-year lag. Therefore, we observe empirically that if we wish to examine a short-medium term relation of unemployment rates to stroke mortality–i.e., at 2-4 year's lag – and include the medium-long term impact of GDP per capita in the model, then we usually require at least a five-year lag of the GDP to mortality. If we place both the GDP per capita and the

unemployment rate at 2-4 year's lag, the presence of the GDP will force the unemployment rate into insignificance (due to multi-collinearity).

### **Logic of the sequence of analysis**

As mentioned above, the first full modelling exercise for stroke was to replicate the initial heart disease mortality model, for EU countries, repeating the two year lag of unemployment to stroke mortality and the GDP per capita lag of seven years.

The second type of model aimed to reproduce aspects of the initial replication, but removed the labour force participation rate in order to observe the completely independent impact of the unemployment rate alone at different lags.

The third model type sought to reproduce aspects of the initial replication, but removed the unemployment rate, in order to examine the completely independent impact of the labour force participation rate alone at zero lag.

With the forth model type, the issue was to determine the complete lag structure of GDP per capita in relation to stroke mortality in the absence of both the unemployment rate and the labour force participation rate.

The fifth model repeated the initial replication and expanded the GDP lag to 10 years, in order to accommodate the potential for investment in pharmaceutical development and other mechanisms of physical and human capital formation.

In the sixth and final model, the unemployment rate is tested at a lag of three years, with the remainder of the control variables included and the GDP per capita maintained at a lag of ten years.

### **VI. *Databases and analysis***

The databases from which the variables are extracted include, for the labour force and other economic variables, EUROSTAT supplemented by the International Labour Organization, World Bank, and the International Monetary Fund. Data on heart disease and stroke mortality for the EU countries are drawn from EUROSTAT with verification of reliability from the World Health Organization (WHO, Geneva). Data on international smoking prevalence, access to water and sanitation facilities, and adolescent fertility, are from the WHO. Vegetable food supply per capita is drawn from the Food and Agriculture Organization (FAO) databases. Statistical analysis is performed under the pooled cross-sectional time series method, with random effects (Wooldridge, 2002, p. 286), using STATA/SE for large databases.



## VII. Findings

### • Analysis of relation between unemployment and heart disease mortality

The overall problem is to construct a minimally plausible model of Ischemic Heart Disease (IHD) – on epidemiological grounds – that will also permit us to assess the contribution of unemployment rates. As an absolutely minimal IHD model for Europe, we can rely on a variable (or variables) that will serve as a marker for socioeconomic status (SES). The rationale is that, as discussed below, SES is epidemiologically the most pervasive and ubiquitous – i.e., generalizable – predictor of mortality at all ages, both sexes, nationalities and ethnic groups. In industrialized countries, SES is an especially sensitive risk factor in cardiovascular diseases.

As we shall see, prediction of total age-adjusted IHD mortality rate across European countries by unemployment rates alone will yield between 9-13 percent of variance explained. This percentage is probably insufficient to create a reasonable epidemiological IHD model, even though, for some middle-aged groups, variance explained by unemployment rates will rise to 20-30 percent.

### First step: unemployment rate as the sole predictor

Nevertheless, for illustrative purposes, we begin by exploring the simplest relation of the overall unemployment rate, as is normally taken as a marker of economic recession, to variation in the age-adjusted IHD mortality rate across all countries of the European Union and across time over the period 2000-2010.

We observe that at 0 lag, (i.e., concurrently), the adjusted  $R^2$  (i.e., variance explained) is .07 – but the relation is not statistically significant, yet the significant relation at 2 years lag shows a coefficient of 3.79. See Table 1.

**Table 1:** Relation of unemployment rate to age-adjusted IHD mortality rate, by lagged years of unemployment 0-5

Predicting of ischaemic heart diseases (ICD-10 codes I20-I25) death rate per 100,000 of total both sexes combined population

Predictors	observ.	groups	obs./gr.	within	between	overall	Coef.	Std.Err	t	P>t	Beta
0 years lag of Unemployment as % of total labour force 15+	308	28	11	0.00	0.17	0.11	0.51	0.42	1.22	0.22	0.07
<b>1 year lag of Unemployment as % of total labour force 15+</b>	<b>307</b>	<b>28</b>	<b>11</b>	<b>0.08</b>	<b>0.14</b>	<b>0.11</b>	<b>2.28</b>	<b>0.45</b>	<b>5.03</b>	<b>0.00</b>	<b>0.28</b>
<b>2 years lag of Unemployment as % of total labour force 15+</b>	<b>306</b>	<b>28</b>	<b>10.9</b>	<b>0.19</b>	<b>0.11</b>	<b>0.10</b>	<b>3.79</b>	<b>0.47</b>	<b>8.12</b>	<b>0.00</b>	<b>0.42</b>
<b>3 years lag of Unemployment as % of total labour force 15+</b>	<b>306</b>	<b>28</b>	<b>10.9</b>	<b>0.15</b>	<b>0.11</b>	<b>0.10</b>	<b>3.54</b>	<b>0.50</b>	<b>7.05</b>	<b>0.00</b>	<b>0.37</b>
<b>4 years lag of Unemployment as % of total labour force 15+</b>	<b>306</b>	<b>28</b>	<b>10.9</b>	<b>0.09</b>	<b>0.11</b>	<b>0.10</b>	<b>2.78</b>	<b>0.53</b>	<b>5.26</b>	<b>0.00</b>	<b>0.29</b>
<b>5 years lag of Unemployment as % of total labour force 15+</b>	<b>306</b>	<b>28</b>	<b>10.9</b>	<b>0.04</b>	<b>0.10</b>	<b>0.09</b>	<b>1.94</b>	<b>0.52</b>	<b>3.72</b>	<b>0.00</b>	<b>0.21</b>

Legend: Red = Significance <.05  
 Bold Black = Significance <.10  
 Light Black = NS

Moving to lags of 1-5 years the relation is now always significant, with explained variance of 9-11 percent. The peak of this relation is at a lag of 2 years with an  $R^2$  of 50 percent, coefficient of 2.04 and standardized beta .24 (i.e., comparative estimate of impact of unemployment). See Table 2.

**Table 2:** Relation of unemployment rate to age-adjusted IHD mortality rate, adjusted for GDP per capita, by lagged years of unemployment 0-5 and lagged years of GDPpc 5-10

**Predicting of ischaemic heart diseases (ICD-10 codes I20-I25) death rate per 100,000 of total population (age standardized)**

Model 03b.1I5.2I0	asdr		AR(1)	Robust	Fixed
over. R <sup>2</sup>	0.51		0.51	0.51	0.51
Coefficients/Betas	Coef.	Betas	Coefficients		
5 years lag of GDP per capita at PPP in thousands of constant 2005 int. \$	-6.00	-0.76	-5.34	-6.00	-6.00
Unemployment rate as % of total labour force 15+ years old	0.77	0.11	-0.03	0.77	0.78

Model 03b.1I6.2I1	asdr		AR(1)	Robust	Fixed
over. R <sup>2</sup>	0.51		0.52	0.51	0.51
Coefficients/Betas	Coef.	Betas	Coefficients		
6 years lag of GDP per capita at PPP in thousands of constant 2005 int. \$	-5.42	-0.71	-5.21	-5.42	-5.38
1 year lag of Unemployment rate as % of total labour force 15+ years old	1.36	0.17	0.25	1.36	1.37

Model 03b.1I7.2I2	asdr		AR(1)	Robust	Fixed
over. R <sup>2</sup>	0.50		0.52	0.50	0.50
Coefficients/Betas	Coef.	Betas	Coefficients		
7 years lag of GDP per capita at PPP in thousands of constant 2005 int. \$	-4.79	-0.66	-5.00	-4.79	-4.71
2 years lag of Unemployment rate as % of total labour force 15+ years old	2.04	0.24	0.96	2.04	2.08

Model 03b.1I8.2I3	asdr		AR(1)	Robust	Fixed
over. R <sup>2</sup>	0.51		0.52	0.51	0.51
Coefficients/Betas	Coef.	Betas	Coefficients		
8 years lag of GDP per capita at PPP in thousands of constant 2005 int. \$	-4.81	-0.67	-4.90	-4.81	-4.72
3 years lag of Unemployment rate as % of total labour force 15+ years old	1.70	0.20	1.07	1.70	1.74

Model 03b.1I9.2I4	asdr		AR(1)	Robust	Fixed
over. R <sup>2</sup>	0.51		0.51	0.51	0.51
Coefficients/Betas	Coef.	Betas	Coefficients		
9 years lag of GDP per capita at PPP in thousands of constant 2005 int. \$	-4.89	-0.67	-4.46	-4.89	-4.79
4 years lag of Unemployment rate as % of total labour force 15+ years old	1.17	0.14	1.01	1.17	1.21

Model 03b.1I10.2I5	asdr		AR(1)	Robust	Fixed
over. R <sup>2</sup>	0.51		0.50	0.51	0.51
Coefficients/Betas	Coef.	Betas	Coefficients		
10 years lag of GDP per capita at PPP in thousands of constant 2005 int. \$	-4.81	-0.62	-3.74	-4.81	-4.67
5 years lag of Unemployment rate as % of total labour force 15+ years old	0.58	0.07	0.59	0.58	0.62

Legend: Red = Significance <.05  
 Bold Black = Significance <.10  
 Light Black = NS

**Second step: adding GDP while taking account of Okun's Law**

A minimal model for IHD in industrialized countries would at least include one major proxy variable for aggregate population SES in addition to the unemployment rate. At the national level GDP per capita is the closest we come to an aggregate variable that represents the national standard of living as well as the sources of national investment. As an aggregate measure, the GDP per capita does considerably more than measure the "average" of SES for a national population. It also signifies the potential for investment in national health services, education and social welfare, including pensions and support of the disabled.

The great problem, however, in adding GDP per capita to a model of mortality already involving the unemployment rate is that, like the unemployment rate itself, the GDP is a fundamental indicator of business cycle fluctuations. Thus, concurrently – i.e., at zero year's lag – GDP per capita and the unemployment rate will be directly and robustly inversely related to each other. The principal reason is that even decline in the growth rate of GDP per capita has a causal relation to increased unemployment due to the decline in demand for goods and services. Declines in consumer demand signal to employers that fewer employees will be needed for firms to remain profitable at their previous rate.

So regular is the relation between GDP per capita and the unemployment rate that a rule of thumb stating the extent of GDP growth that is needed to produce a one percent decrease in the unemployment rate has been in use since the 1960s. It is referred to as Okun's Law, offered by Arthur Okun who proposed the relationship in 1962. The relationship varies depending on the country and the time period under consideration. In Okun's original statement of his law, a two percent increase in output corresponds to a one percent decline in the rate of cyclical unemployment; a 0.5 percent increase in labour force participation; a 0.5 percent increase in hours worked per employee; and a one percent increase in output per hours worked (labour productivity).

We therefore have the situation that GDP per capita declines can have a damaging effect on health through their impact on the unemployment rate. In that case, GDP per capita is the more distal causal variable while unemployment is the more proximal cause of health decline. In this scenario, however, unemployment is an intermediate component of the more general relation of GDP to health. This common cyclical concurrence produces difficulty for adding GDP per capita to the unemployment rate (with both variables at zero lag) in order to produce a more complete and plausible model for e.g. IHD.

Our solution is based on the fact that the relationship of GDP per capita to IHD occurs over multiple years – i.e., there are strong lengthy, medium- and short-term relations of GDP per capita to IHD. This is because GDP per capita is a foundational variable which includes aggregate SES at the national level. This relation involves implications for long-term investments, such as in education, health care and infrastructure, as well as short-term consequences for personal household and government expenditures.

On the other hand, the most important effects of unemployment on health are empirically observed at 0-3 year's lag, and especially at a two-year lag. Therefore, we observe empirically that if we wish to examine a very short-term relation of unemployment rates to IHD – i.e., at lag zero – and include the medium-term impact of GDP per capita in the model, then we usually require approximately a five-year lag of the GDP to mortality. Similarly, optimally, tests of the one-year lag of unemployment rates to IHD should include a six-year lagged GDP per capita, assuming that we want to examine the health effects of unemployment rate as thoroughly as possible while observing the medium-term effects of GDP per capita. If we place both the GDP per capita and the unemployment rate at lag zero, the presence of the GDP will force the unemployment rate into insignificance (due to the fundamental relation of GDP per capita to the unemployment rate).

We now examine the relation of unemployment rates to IHD covering lags 0-5, holding constant GDP per capita at a five-year temporal distance. Thus, GDP per capita at lag 5 is added to unemployment lag zero, GDP per capita lag 6 is combined with unemployment rate lag 1, GDP per capita lag 7 with unemployment rate lag 2 and so on (see Table 2). Multi-collinearity due to the intrinsic relation between GDP per capita and unemployment rates, at the same lag, will not allow unbiased analysis of the relation of unemployment rates to IHD. Models with the type of lag separation discussed above allow us to see the effect of unemployment rates at 0-5 year lags, while including substantial influence of the GDP per capita variable.

Now, if we fix the unemployment rate at 2 years lag, we can observe the effects of GDPpc with a very short negative impact on IHD mortality, – at lag 0 – a negative medium-term impact – at lags 5-6 or 7 – and a negative longer-term impact at lag 10. We find that even at all three lag relations of the GDPpc, the relation between GDPpc and IHD mortality is negative, statistically significant and robust. Nevertheless, in this experiment, the relation between the unemployment rate at 2 years lag, and the IHD mortality rate remains significant. Thus, there is no necessary arbitrariness in selecting the appropriate lag for GDPpc in order to estimate the relations of the unemployment rate to IHD mortality (Table 3).

Yet if we wish to optimize the relation of the unemployment rate to IHD mortality, considering the presence of GDPpc in the model, we will select the unemployment rate at 2 years lag and the GDPpc perhaps at 7 years lag. The reason is that (1) at GDPpc lag 5 the model R<sup>2</sup> is maximized at .70, but the coefficient for unemployment rate lagged 2 years is 1.22; (2) on the other hand, the coefficient for unemployment rate lagged 2 years is highest, i.e., 2.6, at GDPpc lagged 10 years; (3) thus, taking into account both the R<sup>2</sup> for the model and the magnitude of the unemployment rate coefficient lagged 2 years, the optimum middle ground is around GDPpc lagged 7 years, where the model R<sup>2</sup> is .69 and the unemployment rate lagged 2 years coefficient is 2.03 (Table 3).

**Table 3:** Relation of unemployment rate lagged 2 years to age-adjusted IHD mortality rate, adjusted for GDPpc lagged 0-10 years and Southern Europe

#### Predicting of ischaemic heart diseases (ICD-10 codes I20-I25) death rate per 100,000 of total population (age standardized)

<b>Coefficients/Betas</b>	<b>Coef.</b>	<b>Betas</b>
<b>Model 03b.1I0.2I2</b>	<b>over. R<sup>2</sup>=0.66</b>	
0 years lag of GDP per capita at PPP in thousands of constant 2005 int. \$	<b>-5.6</b>	<b>-0.6</b>
<u>2 years lag of Unemployment rate as % of total labour force 15+ years old</u>	<u><b>1.52</b></u>	<u><b>0.17</b></u>
Dummy (1=Southern Europe, 0=other states)	<b>-81.7</b>	<b>-0.17</b>
<b>Model 03b.1I1.2I2</b>	<b>over. R<sup>2</sup>=0.67</b>	
1 years lag of GDP per capita at PPP in thousands of constant 2005 int. \$	<b>-5.94</b>	<b>-0.7</b>
<u>2 years lag of Unemployment rate as % of total labour force 15+ years old</u>	<u><b>0.57</b></u>	<u><b>0.06</b></u>
Dummy (1=Southern Europe, 0=other states)	<b>-80.4</b>	<b>-0.16</b>
<b>Model 03b.1I2.2I2</b>	<b>over. R<sup>2</sup>=0.68</b>	
2 years lag of GDP per capita at PPP in thousands of constant 2005 int. \$	<b>-6.22</b>	<b>-0.8</b>
<u>2 years lag of Unemployment rate as % of total labour force 15+ years old</u>	<u><b>-0.26</b></u>	<u><b>-0.03</b></u>
Dummy (1=Southern Europe, 0=other states)	<b>-79.3</b>	<b>-0.14</b>
<b>Model 03b.1I3.2I2</b>	<b>over. R<sup>2</sup>=0.69</b>	
3 years lag of GDP per capita at PPP in thousands of constant 2005 int. \$	<b>-5.99</b>	<b>-0.78</b>
<u>2 years lag of Unemployment rate as % of total labour force 15+ years old</u>	<u><b>0.11</b></u>	<u><b>0.01</b></u>
Dummy (1=Southern Europe, 0=other states)	<b>-78.2</b>	<b>-0.14</b>
<b>Model 03b.1I4.2I2</b>	<b>over. R<sup>2</sup>=0.69</b>	
4 years lag of GDP per capita at PPP in thousands of constant 2005 int. \$	<b>-5.82</b>	<b>-0.74</b>
<u>2 years lag of Unemployment rate as % of total labour force 15+ years old</u>	<u><b>0.65</b></u>	<u><b>0.07</b></u>
Dummy (1=Southern Europe, 0=other states)	<b>-77.2</b>	<b>-0.15</b>
<b>Model 03b.1I5.2I2</b>	<b>over. R<sup>2</sup>=0.70</b>	
5 years lag of GDP per capita at PPP in thousands of constant 2005 int. \$	<b>-5.56</b>	<b>-0.7</b>
<u>2 years lag of Unemployment rate as % of total labour force 15+ years old</u>	<u><b>1.22</b></u>	<u><b>0.13</b></u>
Dummy (1=Southern Europe, 0=other states)	<b>-76.5</b>	<b>-0.15</b>
<b>Model 03b.1I6.2I2</b>	<b>over. R<sup>2</sup>=0.69</b>	
6 years lag of GDP per capita at PPP in thousands of constant 2005 int. \$	<b>-5.09</b>	<b>-0.67</b>
<u>2 years lag of Unemployment rate as % of total labour force 15+ years old</u>	<u><b>1.78</b></u>	<u><b>0.2</b></u>
Dummy (1=Southern Europe, 0=other states)	<b>-75.7</b>	<b>-0.15</b>
<b>Model 03b.1I7.2I2</b>	<b>over. R<sup>2</sup>=0.69</b>	
7 years lag of GDP per capita at PPP in thousands of constant 2005 int. \$	<b>-4.82</b>	<b>-0.66</b>

<u>2 years lag of Unemployment rate as % of total labour force 15+ years old</u>	<b>2.03</b>	<b>0.24</b>
Dummy (1=Southern Europe, 0=other states)	<b>-74.95</b>	<b>-0.15</b>
<b>Model 03b.1I8.2I2</b>	<b>over. R<sup>2</sup>=0.69</b>	
8 years lag of GDP per capita at PPP in thousands of constant 2005 int. \$	<b>-4.76</b>	<b>-0.66</b>
<u>2 years lag of Unemployment rate as % of total labour force 15+ years old</u>	<b>2.12</b>	<b>0.25</b>
Dummy (1=Southern Europe, 0=other states)	<b>-74.2</b>	<b>-0.15</b>
<b>Model 03b.1I9.2I2</b>	<b>over. R<sup>2</sup>=0.68</b>	
9 years lag of GDP per capita at PPP in thousands of constant 2005 int. \$	<b>-4.7</b>	<b>-0.64</b>
<u>2 years lag of Unemployment rate as % of total labour force 15+ years old</u>	<b>2.3</b>	<b>0.28</b>
Dummy (1=Southern Europe, 0=other states)	<b>-73.8</b>	<b>-0.15</b>
<b>Model 03b.1I10.2I2</b>	<b>over. R<sup>2</sup>=0.66</b>	
10 years lag of GDP per capita at PPP in thousands of constant 2005 int. \$	<b>-4.53</b>	<b>-0.58</b>
<u>2 years lag of Unemployment rate as % of total labour force 15+ years old</u>	<b>2.6</b>	<b>0.31</b>
Dummy (1=Southern Europe, 0=other states)	<b>-73.7</b>	<b>-0.16</b>

Legend: Red = Significance <.05; Bold Black = Significance <.10; Light Black = NS

### Third step: inclusion of additional variables in models relating unemployment rates to IHD

We now have a basis for including additional variables in the IHD model to observe whether the basic relation between unemployment rate and IHD remains significant if we control for other factors that are related to unemployment or are thought to influence IHD as a result of epidemiological risk factor status. We begin with the well-known observation that Mediterranean countries tend to show lower rates of IHD than other European geographic areas, other things equal. This phenomenon is usually attributed to especially healthy Mediterranean diets – high in fruits and vegetables, fish, olive oil, and red wine and low in saturated fats (Estruch, et al, 2006).

Other hypotheses offered to explain the relatively low IHD death rates in Southern European countries include an absence of cold winter temperatures, such as prevail in Northern Europe, and more closely knit family ties – offering greater social cohesion or “social capital” (McGregor, Watkin and Cox, 2004). Our analysis shows that this “dummy” variable – i.e., 0/1 binary variable – for Southern European countries indeed contributes substantially to the explained variance of our IHD model (see Table 4).

**Table 4:** Relation of unemployment rate to age-adjusted IHD mortality rate, adjusted for GDPpc and Southern Europe by lagged years of unemployment 0-5 and lagged years of GDPpc 5-10

**Predicting of ischaemic heart diseases (ICD-10 codes I20-I25) death rate per 100,000 of total population (age standardized)**

Model 03b.1I5.2I0	asdr		AR(1)	Robust	Fixed
over. R <sup>2</sup>	0.70		0.70	0.70	0.51
Coefficients/Betas	Coef.	Betas	Coefficients		
5 years lag of GDP per capita at PPP in thousands of constant 2005 int. \$	-6.00	-0.76	-5.41	-6.00	-6.00
Unemployment rate as % of total labour force 15+ years old	0.77	0.10	-0.01	0.77	0.78
Dummy (1=Southern Europe, 0=other states)	-76.25	-0.15	-75.06	-76.25	

Model 03b.1I6.2I1	asdr		AR(1)	Robust	Fixed
over. R <sup>2</sup>	0.70		0.70	0.70	0.51
Coefficients/Betas	Coef.	Betas	Coefficients		
6 years lag of GDP per capita at PPP in thousands of constant 2005 int. \$	-5.44	-0.71	-5.28	-5.44	-5.38
1 year lag of Unemployment rate as % of total labour force 15+ years old	1.36	0.17	0.27	1.36	1.37
Dummy (1=Southern Europe, 0=other states)	-75.35	-0.15	-74.20	-75.35	

Model 03b.1I7.2I2	asdr		AR(1)	Robust	Fixed
over. R <sup>2</sup>	0.69		0.70	0.69	0.50
Coefficients/Betas	Coef.	Betas	Coefficients		
7 years lag of GDP per capita at PPP in thousands of constant 2005 int. \$	-4.82	-0.66	-5.07	-4.82	-4.71
2 years lag of Unemployment rate as % of total labour force 15+ years old	2.03	0.24	0.96	2.03	2.08
Dummy (1=Southern Europe, 0=other states)	-74.95	-0.15	-73.88	-74.95	

Model 03b.1I8.2I3	asdr		AR(1)	Robust	Fixed
over. R <sup>2</sup>	0.69		0.70	0.69	0.51
Coefficients/Betas	Coef.	Betas	Coefficients		
8 years lag of GDP per capita at PPP in thousands of constant 2005 int. \$	-4.84	-0.67	-4.98	-4.84	-4.72
3 years lag of Unemployment rate as % of total labour force 15+ years old	1.69	0.19	1.07	1.69	1.74
Dummy (1=Southern Europe, 0=other states)	-74.02	-0.15	-73.37	-74.02	

Model 03b.1I9.2I4	asdr		AR(1)	Robust	Fixed
over. R <sup>2</sup>	0.69		0.68	0.69	0.51
Coefficients/Betas	Coef.	Betas	Coefficients		
9 years lag of GDP per capita at PPP in thousands of constant 2005 int. \$	-4.93	-0.67	-4.57	-4.93	-4.79
4 years lag of Unemployment rate as % of total labour force 15+ years old	1.17	0.13	1.01	1.17	1.21
Dummy (1=Southern Europe, 0=other states)	-73.15	-0.16	-72.93	-73.15	

Model 03b.1I10.2I5	asdr		AR(1)	Robust	Fixed
over. R <sup>2</sup>	0.66		0.65	0.68	0.51
Coefficients/Betas	Coef.	Betas	Coefficients		
10 years lag of GDP per capita at PPP in thousands of constant 2005 int. \$	-6.09	-0.65	-3.91	-4.88	-4.67
5 years lag of Unemployment rate as % of total labour force 15+ years old	1.15	0.12	0.59	0.59	0.62
Dummy (1=Southern Europe, 0=other states)	-81.87	-0.17	-72.90	-72.68	

Legend: Red = Significance <.05; Bold Black = Significance <.10; Light Black = NS

The next set of variables for testing as potential confounders includes other labour market measures – i.e., labour force participation and self-employment.

The next most important variables are those indicating high rates of poverty, relatively low income and/or the potential for low life-time earnings. These variables include the percentage of a population with access to water and sanitation (inverse indicator of a high poverty rate), adolescent fertility rates and proportion of the labour force engaged in agriculture.

Finally, potentially the most important source of IHD mortality, namely smoking prevalence, is included for epidemiological completeness.

## **Detailed findings**

- If unemployment is lagged over zero – 5 years in relation to age-adjusted heart disease mortality, without any control variables, then unemployment at lags 1 – 5 will be statistically significant and the highest coefficient for unemployment is at lag 2. The overall R square is approximately 0.10 (See table 1).
- When unemployment is related to age-adjusted heart disease mortality at zero – 5 years lag, with a control for GDP per capita at lags ranging from 5 – 10 years (in order to avoid multicollinearity with unemployment and maintain medium-long-term relations of GDP per capita to age-adjusted heart disease mortality), the 2-year lag for unemployment has the highest coefficient, with GDP per capita lagged 7 years. This will be the case regardless of the method of calculating the regression coefficient—i.e., random effects, random effects with AR (1) adjustment, robust random effects, or fixed effects (See table 2).
- The optimum lag for unemployment in its relation to age-adjusted heart disease mortality is at 2 years, and that for GDP per capita is 7 years, where an additional control variable—dummy for Southern Europe—is added to the model. The overall R square now rises to approximately 0.70 (See table 4).
- However, if the unemployment relations' lag to age-adjusted heart disease mortality is fixed at 2 years, and GDP per capita as well as the dummy for Southern Europe are in the model, then the optimum lag for GDP per capita is 5 years since the highest overall R square is 0.70 for the model (See table 3).
- The relation between unemployment and age-adjusted heart disease mortality at 2 years' lag for unemployment controlling for GDP per capita and Southern Europe (dummy) can be seen for ages 35 – 44, 44 – 54, 55 – 64, 65 – 74, 85 and over, but not for under age 35 (See table 5).



Table 5: Relation of unemployment rate lagged 0 or 2 years to IHD mortality rates, by age, adjusted for economic and life style variables

Predicting of ischaemic heart diseases (ICD-10 codes I20-I25) death rate per 100,000 of total population.  
Pooled analysis for 11 years (2000-2010), 28 EU countries.

Model 03b.1110.215	15-24		25-34		35-44		45-54	
over. R <sup>2</sup>	0.36		0.64		0.76		0.88	
Coefficients/Betas	Coef.	Betas	Coef.	Betas	Coef.	Betas	Coef.	Betas
7 years lag of GDP/cap. at PPP in thousand of cons. 2005 int. \$	-0.01	-0.21	-0.03	-0.13	-0.20	-0.17	-1.30	-0.32
Total pop. served with Improved water & Sanitation (%)	-0.01	-0.09	-0.10	-0.29	-0.20	-0.12	-0.97	-0.15
Labour force participation as % of total population (age specific, lagging*)	0.00	-0.12	-0.03	-0.07	-0.30	-0.16	-1.49	-0.19
Self-employment as a proportion (%) of total employment 15+	0.03	0.33	0.06	0.21	-0.06	-0.04	-0.69	-0.13
Unemployment rate as % of total labour force (age specific, lagging**)	0.01	0.11	0.04	0.16	0.20	0.13	1.17	0.21
Employment in Agriculture as a proportion (%) of total LF 15+	-0.03	-0.34	-0.08	-0.23	0.65	0.32	2.12	0.30
Adolescent fertility rate (births per 1,000 women ages 15-19)	0.02	0.28	0.09	0.32	0.22	0.15	0.42	0.08
Gini index of inequality in household net disposable income			-0.03	-0.08	0.06	0.03	0.84	0.11
Inflation, consumer prices (annual %)	0.01	0.12	0.06	0.28	0.16	0.11	0.24	0.05
Proportion of regular daily smokers in the male pop. age 15+	0.00	-0.08	0.02	0.09	0.14	0.13	0.45	0.13
Dummy 1 (1=Southern Europe, 0=other states)	-0.04	-0.03	0.21	0.03	-4.57	-0.14	-26.10	-0.23
Dummy 2 (1=Poland, Bulgaria & Romania, 0=other states)	-0.19	-0.08	-1.20	-0.12	-10.78	-0.22	-46.92	-0.27
Age specific LFPR yes/no (lagging: number of years)*	yes	2	no	0	yes	0	no	0
Age specific UER yes/no (lagging: number of years)**	no	0	yes	0	yes	2	yes	2

Model 03b.1110.215	55-64		65-74		75-84		85+	
over. R <sup>2</sup>	0.91		0.92		0.80		0.71	
Coefficients/Betas	Coef.	Betas	Coef.	Betas	Coef.	Betas	Coef.	Betas
7 years lag of GDP/cap. at PPP in thousand of cons. 2005 int. \$	-4.55	-0.42	-15.26	-0.50	-39.22	-0.59	-53.59	-0.27
Total pop. served with Improved water & Sanitation (%)	-4.32	-0.25	-6.38	-0.12	-42.86	-0.27	-129.89	-0.22
Labour force participation as % of total population (age specific, lagging*)	-3.85	-0.18	-8.06	-0.14	-4.07	-0.07	-77.65	-0.22
Self-employment as a proportion (%) of total employment 15+	-1.60	-0.11	-5.37	-0.13	8.53	0.09	-34.49	-0.13
Unemployment rate as % of total labour force (age specific, lagging**)	2.54	0.16	3.78	0.08	7.41	0.09	43.59	0.19
Employment in Agriculture as a proportion (%) of total LF 15+	4.04	0.21	12.40	0.24	-25.28	-0.24	32.69	0.10
Adolescent fertility rate (births per 1,000 women ages 15-19)	0.67	0.05	3.46	0.09	-1.81	-0.02	14.86	0.05
Gini index of inequality in household net disposable income	1.30	0.07	-2.28	-0.04	-29.11	-0.26	-64.99	-0.19
Inflation, consumer prices (annual %)	1.09	0.09	0.61	0.02	0.89	0.02	-3.86	-0.03
Proportion of regular daily smokers in the male pop. age 15+	0.94	0.10	4.62	0.19	8.99	0.18	14.89	0.10
Dummy 1 (1=Southern Europe, 0=other states)	-89.45	-0.28	-248.80	-0.25	-672.10	-0.20	-2179.78	-0.17
Dummy 2 (1=Poland, Bulgaria & Romania, 0=other states)	-164.41	-0.34	-481.56	-0.32	-953.55	-0.19	-3796.14	-0.19
Age specific LFPR yes/no (lagging: number of years)*	no	0	no	0	no	0	no	0
Age specific UER yes/no (lagging: number of years)**	yes	2	yes	2	no	2	no	2

Legend: Red = Significance <.05  
Bold Black = Significance <.10  
Light Black = NS



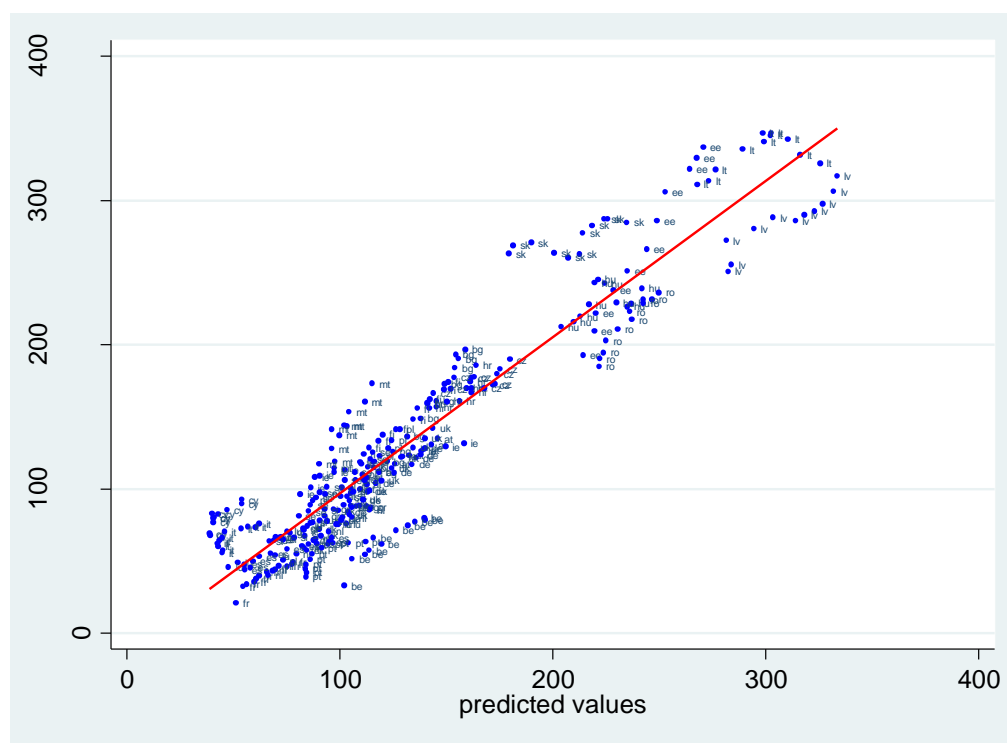
- But if the relation between unemployment and age-adjusted heart disease mortality controls for 11 economic and life-style variables, then a significant coefficient can be seen for all age groups from 15 – 24 to 85 +. For age groups 15 – 24 and 25 – 34, the lag for unemployment is at zero, whereas for all age groups over 35, the unemployment lag is at 2 years. The control variables are GDP per capita, total population served with improved water and sanitation, labour force participation as per cent of total population 15+, employment in agriculture as a proportion of total labour force 15+, adolescent fertility rate (births per 1,000 women ages 15 – 19), Gini index of inequality in household net disposable income, inflation (consumer prices annual per cent), proportion of regular daily smokers in the male population ages 15+, Dummy 1 (1 = Southern Europe, 0 = other states), Dummy 2 (1 = Poland, Bulgaria, and Romania, 0 = other states). (See table 5).
- As can be seen in table 5, the coefficients for unemployment continually increase as population's age: 15 – 24 = 0.01, 25 – 34 = 0.04, 35 – 44 = 0.20, 45 – 54 = 1.17, 55 – 64 = 2.54, 65 – 74 = 3.78, 75 – 84 = 7.41, 85 and over = 43.59. These rising coefficients by increasing age are consistent with higher levels of heart disease prevalence by age in the European population. In general, the R square and fit of the overall model, R square, also tends to improve with age, except for the oldest age groups. Thus, 15 – 24 = 0.3602, 25 – 34 = 0.6402, 35 – 44 = 0.7629, 45 – 54 = 0.8848, 55 – 64 = 0.9888, 65 – 74 = 0.9162, 75 – 84 = 0.8018, 85 and over = 0.7129.
- Finally, we now compare the impact of unemployment on age-adjusted heart disease mortality with lags for unemployment at 0, 1 and 2 years in models controlling for the 11 economic and life style variables. In this comparison all three lags for unemployment are statistically significant, but the highest coefficient and smallest confidence interval is at a lag of 2 years. However, the overall R square for the model is highest when unemployment is at lag 0 = 0.8884, somewhat lower at lag 1 = 0.8774 and lower still at lag 2 = 0.8687. See tables 6, 7, 8 and accompanying figures.

**Table 6:** Relation of unemployment rate lagged 0 years to age-adjusted IHD mortality rate, adjusted for economic and life style variables.

Predicting of ischaemic heart diseases (ICD-10 codes I20-I25) death rate per 100,000 of total population. Pooled analysis for 11 years (2000-2010), 28 EU countries.

Random-effects GLS regression		Number of obs	=	307
Group variable:	numcod	Number of groups	=	28
R-sq: within	= 0.6609	Obs per group: min	=	10
between	= 0.8982	avg	=	11.0
overall	= 0.8884	max	=	11
Wald chi2(10)	= 737.62	corr(u_i, X)	= 0 (assumed)	Prob > chi2
sigma_u	26.861826	sigma_e	10.389635	rho
to u_i)				.86986833 (fraction of variance due

Predictors	Coef.	Std.Err	t	P>t	Beta
<b>5 years lag of GDP/cap. at PPP in thousands of cons. 2005 int. \$</b>	<b>-3.95</b>	<b>0.42</b>	<b>9.40</b>	<b>0.00</b>	<b>0.49</b>
<b>Total pop. served with Improved water &amp; Sanitation (%)</b>	<b>-2.49</b>	<b>0.75</b>	<b>3.31</b>	<b>0.00</b>	<b>0.14</b>
<b>Labour force participation as % of total population 15+</b>	<b>-2.25</b>	<b>0.55</b>	<b>4.05</b>	<b>0.00</b>	<b>0.16</b>
<b>Self-employment as a proportion (%) of total employment 15+</b>	<b>-1.98</b>	<b>0.56</b>	<b>3.54</b>	<b>0.00</b>	<b>0.18</b>
<b>0 years lag of Unemployment rate as % of total labour force 15+</b>	<b>0.74</b>	<b>0.25</b>	<b>2.90</b>	<b>0.00</b>	<b>0.09</b>
<b>Employment in agriculture as a proportion (%) of total LF 15+</b>	<b>3.69</b>	<b>0.81</b>	<b>4.57</b>	<b>0.00</b>	<b>0.29</b>
<b>Adolescent fertility rate (births per 1,000 women ages 15-19)</b>	<b>1.12</b>	<b>0.49</b>	<b>2.28</b>	<b>0.02</b>	<b>0.10</b>
<b>Proportion of regular daily smokers in the male pop. age 15+</b>	<b>0.67</b>	<b>0.26</b>	<b>2.57</b>	<b>0.01</b>	<b>0.11</b>
<b>Dummy 1 (1=Southern Europe, 0=other states)</b>	<b>-75.09</b>	<b>12.41</b>	<b>6.05</b>	<b>0.00</b>	<b>0.21</b>
<b>Dummy 2 (1=Poland, Bulgaria &amp; Romania, 0=other states)</b>	<b>143.76</b>	<b>19.88</b>	<b>7.23</b>	<b>0.00</b>	<b>0.27</b>
<b>Constant</b>	<b>594.82</b>	<b>81.88</b>	<b>7.26</b>	<b>0.00</b>	<b>0.03</b>



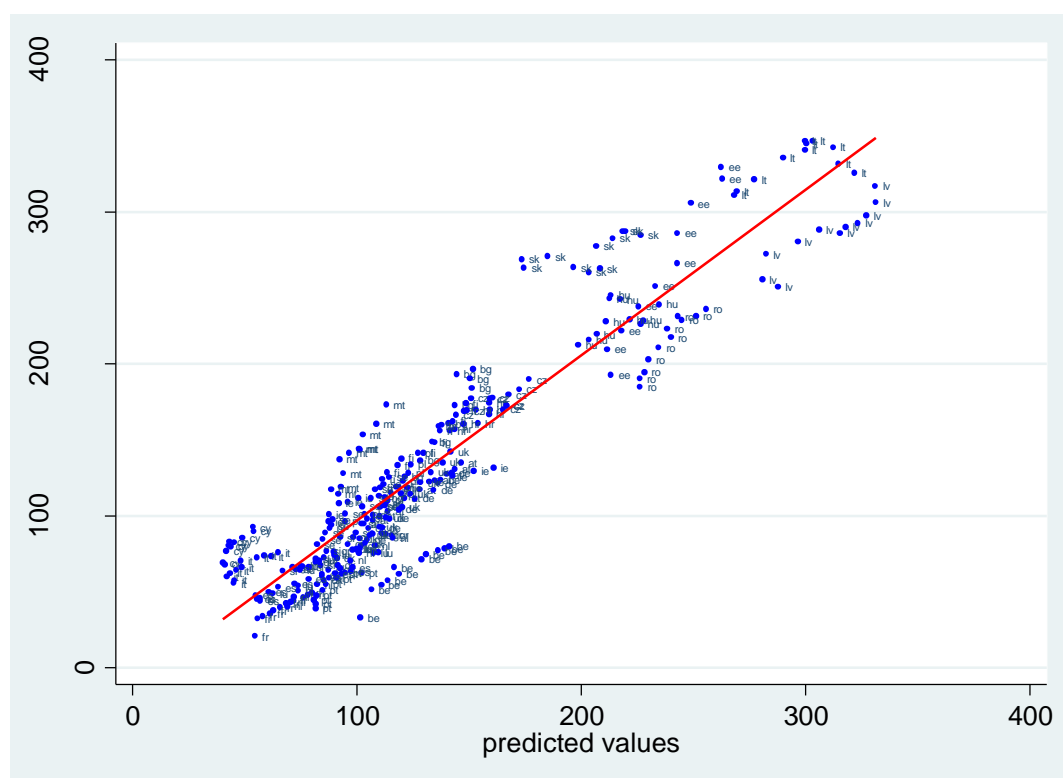
Legend: Red = Significance <.05; Bold Black = Significance <.10; Light Black = NS

**Table 7:** Relation of unemployment rate lagged 1 year to age-adjusted IHD mortality rate, adjusted for economic and life style variables

Predicting of ischaemic heart diseases (ICD-10 codes I20-I25) death rate per 100,000 of total population. Pooled analysis for 11 years (2000-2010), 28 EU countries.

Random-effects GLS regression			Number of obs	=	304
Group variable:	numcod		Number of groups	=	28
R-sq: within	= 0.6623		Obs per group: min	=	10
between	= 0.8870		avg	=	10.9
overall	= 0.8774		max	=	11
Wald chi2(10)	= 728.75	corr(u_i, X) = 0 (assumed)	Prob > chi2	=	0.0000
sigma_u	26.295798	sigma_e	10.059353	rho	.87234043 (fraction of variance due to u_i)

Predictors	Coef.	Std.Err	t	P>t	Beta
6 years lag of GDP/cap. at PPP in thousands of cons. 2005 int. \$	-3.54	0.39	-8.98	0.00	-0.46
Total pop. served with Improved water & Sanitation (%)	-2.83	0.75	-3.76	0.00	-0.16
Labour force participation as % of total population 15+	-2.15	0.54	-3.95	0.00	-0.15
Self-employment as a proportion (%) of total employment 15+	-1.94	0.55	-3.52	0.00	-0.18
1 year lag of Unemployment rate as % of total labour force 15+	0.84	0.30	2.84	0.01	0.10
Employment in agriculture as a proportion (%) of total LF 15+	3.54	0.82	4.33	0.00	0.28
Adolescent fertility rate (births per 1,000 women ages 15-19)	1.04	0.48	2.15	0.03	0.09
Proportion of regular daily smokers in the male pop. age 15+	0.80	0.25	3.18	0.00	0.13
Dummy 1 (1=Southern Europe, 0=other states)	-72.32	12.25	-5.90	0.00	-0.21
Dummy 2 (1=Poland, Bulgaria & Romania, 0=other states)	-138.71	19.65	-7.06	0.00	-0.26
Constant	605.84	81.43	7.44	0.00	0.06



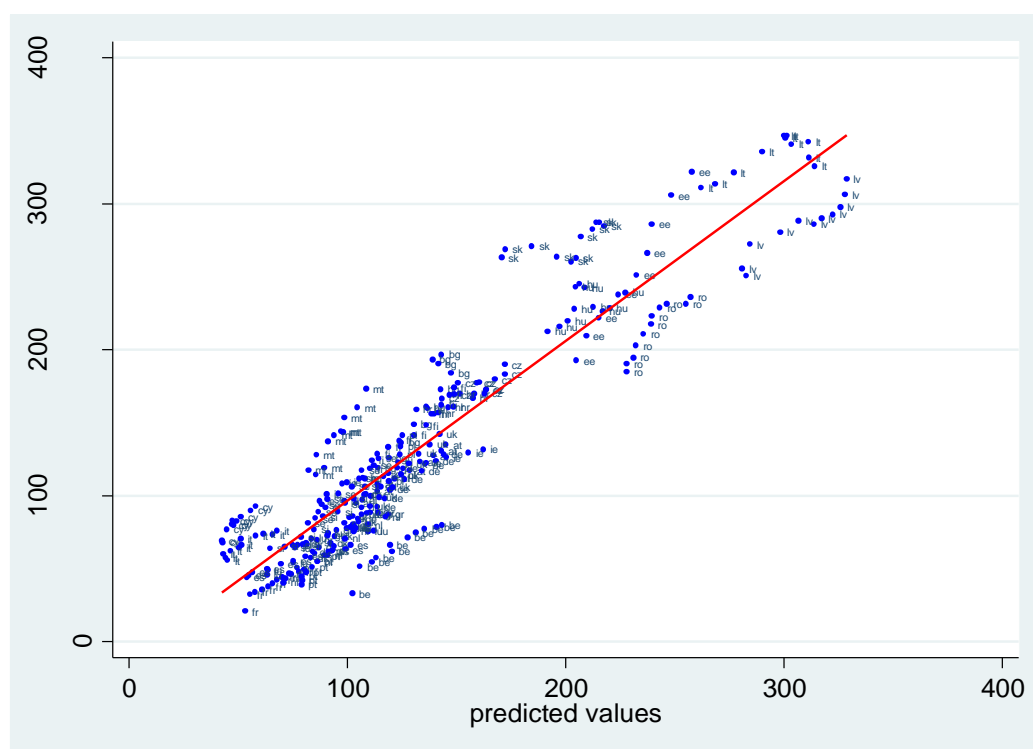
Legend: Red = Significance <.05; Bold Black = Significance <.10; Light Black = NS

**Table 8:** Relation of unemployment rate lagged 2 years to age-adjusted IHD mortality rate, adjusted for economic and life style variables

Predicting of ischaemic heart diseases (ICD-10 codes I20-I25) death rate per 100,000 of total population. Pooled analysis for 11 years (2000-2010), 28 EU countries.

Random-effects GLS regression			Number of obs	=	301
Group variable:	numcod		Number of groups	=	28
R-sq: within	= 0.6746		Obs per group: min	=	9
between	= 0.8781		avg	=	10.8
overall	= 0.8687		max	=	11
Wald chi2(10)	= 745.17	corr(u_i, X) = 0 (assumed)	Prob > chi2	=	0.0000
sigma_u	25.974699	sigma_e	9.5995131	rho	.87982999 (fraction of variance due to u_i)

Predictors	Coef.	Std.Err	t	P>t	Beta
7 years lag of GDP/cap. at PPP in thousands of cons. 2005 int. \$	-3.27	0.36	-9.01	0.00	-0.43
Total pop. served with Improved water & Sanitation (%)	-3.14	0.76	-4.15	0.00	-0.19
Labour force participation as % of total population 15+	-1.87	0.54	-3.49	0.00	-0.14
Self-employment as a proportion (%) of total employment 15+	-1.61	0.54	-2.97	0.00	-0.16
2 years lag of Unemployment rate as % of total labour force 15+	1.14	0.35	3.25	0.00	0.12
Employment in agriculture as a proportion (%) of total LF 15+	3.02	0.83	3.63	0.00	0.25
Adolescent fertility rate (births per 1,000 women ages 15-19)	0.99	0.47	2.11	0.04	0.09
Proportion of regular daily smokers in the total pop. age 15+	0.88	0.24	3.70	0.00	0.15
Dummy 1 (1=Southern Europe, 0=other states)	-71.35	12.12	-5.89	0.00	-0.21
Dummy 2 (1=Poland, Bulgaria & Romania, 0=other states)	-134.44	19.49	-6.90	0.00	-0.26
Constant	604.39	80.26	7.53	0.00	0.11



Legend: Red = Significance <.05; Bold Black = Significance <.10; Light Black = NS

### • Analysis of relation between unemployment and stroke mortality

#### Stroke mortality rate model as replication of heart disease mortality rate model

A stroke mortality rate model is developed that can essentially reproduce the findings for heart disease mortality as originally created. In this original heart disease mortality model the principal lag of the unemployment rate is at 2 years, that for the labour force

participation rate is at zero, and the lag for GDP per capita is at an optimal 7 years. Smoking and dietary controls are included at lag zero. Smoking prevalence is utilized only for males because prevalence for females was not a statistically significant predictor of stroke mortality. Food supply from vegetable sources is the dietary control and is statistically significant. Controls for chronic poverty include (lack of) availability of potable water and sanitation, and the adolescent fertility rate at lag zero for both variables were significant. The Gini index is also used as a control for enlarged income inequality.

For total stroke mortality, the unemployment rate, LFPR and GDP per capita are statistically significant. The R squared is at .80 (i.e., .7975). Examining the impact of the unemployment rate and the other economic variables among the age groups 35 – 44 to 85 +: (1) the unemployment rate is significant from 35 – 44 through 65 – 74; (2) the labour force participation rate is significant (negative) for ages 35 through 85 + (!); (3) GDP per capita is also significant for 35 – 85 +. (Male) smoking prevalence is significant for 35 – 44 and for 65 – 85 +; vegetable food supply is (negatively) significant for 35 – 85 +; potable water and sanitation availability is (negatively) significant for 35 – 85 +; and adolescent fertility is also significant for ages 35 – 85+. Finally, the Gini index is significant (with a positive sign) for ages 35 – 44, 55 – 64, 75 – 84 and 85 +.

Clearly stroke mortality follows the pattern of heart disease mortality in EU countries over 2000 – 2010 in the sense that it is partially predicted by the unemployment rate, labour force participation rate, GDP per capita and the control variables used in the original heart disease mortality model. See Table 9.

**Table 9:** Stroke mortality rate model as replication of heart disease mortality

Predicting of cerebro-vascular diseases (ICD-10 codes I60-I69) death rate per 100,000 of population (with GDP at lag 7 and UER at lag 2, by age) for the years 2000-2010

Predicting of cerebro-vasc. dis. DR/100,000 of total pop.	total	15-24	25-34	35-44	45-54	55-64	65-74	75-84	85+
Overall R square	0.80	0.25	0.64	0.73	0.78	0.84	0.84	0.78	0.56
7 years lag of GDP/capita at PPP in thousand of const. 2005 int. \$	-0.20	-0.03	-0.10	-0.13	-0.15	-0.17	-0.21	-0.29	-0.19
Labour force participation as proportion (%) of total populat. 15+	-0.22	-0.05	0.15	-0.14	-0.20	-0.15	-0.15	-0.16	-0.20
2 years lag of Unemployment rate as % of total labour force 15+	0.08	0.08	0.09	0.10	0.20	0.11	0.11	0.06	0.03
Total pop. served with Improved water & Sanitation (%)	-0.25	-0.04	-0.23	-0.25	-0.30	-0.40	-0.31	-0.19	-0.12
Food supply from vegetal products (kcal/capita/day)	-0.27	-0.07	-0.03	-0.17	-0.24	-0.23	-0.24	-0.19	-0.16
Proportion of regular daily smokers in male population age 15+	0.16	0.12	0.17	0.15	0.04	0.06	0.12	0.16	0.27
Adolescent fertility rate (births per 1,000 women ages 15-19)	0.16	0.20	0.28	0.23	0.18	0.18	0.17	0.11	0.03
Gini index of inequality in household net disposable income	0.04	0.01	0.05	-0.12	-0.02	-0.08	-0.04	0.07	0.18

## Tests of relations of principal economic variables to stroke mortality rates

### Unemployment rate without LFPR, but including other control variables

The next step was to examine the two major labour force indicators to determine whether they show predictable patterns in relation to stroke mortality while taking account of the other control variables.

The unemployment rate was examined for lag relations to stroke mortality at 0, 1, 2, 3, 4 and 5 years of lag, adjusting for the control variables described earlier. In this analysis 0, 1 and 5 years of lag were not statistically significant, while lags 2 – 4 showed statistical significance. This confirms that the most important initial lag of impact of the unemployment rate to stroke mortality is at 2 years. See Table 10.

Table 10: Unemployment rate without LFPR, but including other control variables

Predicting of cerebro-vascular diseases (ICD-10 codes I60-I69) death rate per 100,000 of population (by lagging of UER)

Years lagged for unemployment rate	0	1	2	3	4	5
Overall R square	0.81	0.80	0.80	0.79	0.79	0.80
10 y. lag of GDP/cap. at PPP in '000 of con. 2005 int. \$	-0.44	-0.43	-0.42	-0.40	-0.41	-0.42
...years lag of <u>Unemploym. rate as % of labour force 15+</u>	-0.02	0.03	0.08	0.12	0.10	0.06
Total pop. served with Improved water & Sanitation (%)	-0.14	-0.13	-0.13	-0.14	-0.14	-0.14
Food supply from vegetal products (kcal/capita/day)	-0.18	-0.18	-0.18	-0.19	-0.19	-0.19
Adolescent fertility rate (births per 1,000 women 15-19)	0.25	0.26	0.25	0.23	0.24	0.25
Gini index of inequality in h/hold net disposable income	0.01	0.01	0.02	0.03	0.02	0.02

Years lagged for unemployment rate	0	1	2	3	4	5
Overall R square	0.82	0.81	0.81	0.80	0.80	0.81
10 y. lag of GDP/cap. at PPP in '000 of con. 2005 int. \$	-0.42	-0.41	-0.40	-0.39	-0.40	-0.41
...years lag of <u>Unemploym. rate as % of labour force 15+</u>	-0.02	0.03	0.07	0.11	0.08	0.04
Total pop. served with Improved water & Sanitation (%)	-0.15	-0.15	-0.15	-0.15	-0.15	-0.15
Food supply from vegetal products (kcal/capita/day)	-0.17	-0.17	-0.17	-0.18	-0.19	-0.18
Adolescent fertility rate (births per 1,000 women 15-19)	0.25	0.25	0.25	0.24	0.24	0.24
Gini index of inequality in h/hold net disposable income	0.01	0.01	0.02	0.02	0.02	0.01
<b>Proportion of regular daily smokers in total pop. 15+</b>	0.06	0.06	0.05	0.04	0.04	0.05

Labour force participation without unemployment rate, but including other control variables

The LFPR is a far broader measure of labour market involvement of the population while the unemployment rate, as a measure of joblessness, has the disadvantage of not including jobless individuals who have given up seeking work.

It is clear that the labour force participation rate is a much more powerful labour market predictor (inverse) of stroke mortality than the unemployment rate (positive). First, the impact of the LFPR extends, for males, over the age groups 35 – 85 + while for females it extends over 25 – 85+. Second, the impact (i.e., effect size) of the LFPR on stroke mortality is often twice as great as that of the unemployment rate. This is the case even after adjusting for the control variables cited, and in the absence of the unemployment rate in the model predicting stroke mortality. See also Table 11.

**Table 11:** Labour force participation rate without unemployment rate, but including other control variables

Predicting of cerebro-vascular diseases (ICD-10 codes I60-I69) death rate per 100,000 of population (excluding UER, by sex and age)

Death rate per 100,000 of total population by age	asdr	<15	15-24	25-34	35-44	45-54	55-64	65-74	75-84	85+
Overall R square	0.79	0.35	0.37	0.63	0.66	0.78	0.82	0.82	0.77	0.56
10 y. lag of GDP/cap. at PPP in '000 of con. 2005 int. \$	-0.24	0.05	0.03	-0.18	-0.27	-0.20	-0.18	-0.21	-0.29	-0.20
Labour force participation as prop. (%) of total pop. 15+	-0.21	0.13	-0.09	-0.06	-0.15	-0.18	-0.18	-0.22	-0.19	-0.21
Total pop. <u>served</u> with Improved water & Sanitation (%)	-0.20	0.06	0.00	-0.12	-0.11	-0.23	-0.32	-0.25	-0.17	-0.12
Food supply from vegetal products (kcal/capita/day)	-0.22	0.05	-0.02	-0.12	-0.25	-0.27	-0.26	-0.28	-0.16	-0.13
Proportion of regular daily smokers in total pop. 15+	0.07	0.01	0.00	0.10	-0.04	-0.05	-0.01	0.03	0.06	0.22
Adolescent fertility rate (births per 1,000 women 15-19)	0.21	0.48	0.46	0.35	0.24	0.29	0.22	0.23	0.21	0.14

Death rate per 100,000 of male population by age	asdr	<15	15-24	25-34	35-44	45-54	55-64	65-74	75-84	85+
Overall R square	0.80	0.36	0.39	0.67	0.72	0.80	0.83	0.82	0.77	0.53
10 y. lag of GDP/cap. at PPP in '000 of con. 2005 int. \$	-0.25	0.05	0.04	-0.16	-0.26	-0.23	-0.18	-0.24	-0.29	-0.20
Labour force <u>particip.</u> as prop. (%) of male population 15+	-0.14	0.01	-0.13	0.03	-0.17	-0.14	-0.14	-0.15	-0.10	-0.13
Total pop. <u>served</u> with Improved water & Sanitation (%)	-0.17	0.06	-0.03	-0.16	-0.13	-0.20	-0.30	-0.21	-0.13	-0.06
Food supply from vegetal products (kcal/capita/day)	-0.22	0.11	0.01	-0.12	-0.21	-0.25	-0.25	-0.28	-0.17	-0.11
Proportion of regular daily smokers in male pop. 15+	0.16	0.07	0.04	0.15	0.00	0.03	0.10	0.11	0.16	0.27
Adolescent fertility rate (births per 1,000 women 15-19)	0.21	0.49	0.38	0.25	0.22	0.27	0.21	0.21	0.21	0.17

Death rate per 100,000 of female population by age	asdr	<15	15-24	25-34	35-44	45-54	55-64	65-74	75-84	85+
Overall R square	0.77	0.24	0.24	0.41	0.53	0.75	0.82	0.82	0.77	0.54
10 y. lag of GDP/cap. at PPP in '000 of con. 2005 int. \$	-0.21	0.02	0.04	-0.04	-0.13	-0.07	-0.13	-0.15	-0.26	-0.18
Labour force <u>particip.</u> as prop. (%) of female popul. 15+	-0.25	0.14	-0.10	-0.23	-0.15	-0.21	-0.21	-0.25	-0.24	-0.24
Total pop. <u>served</u> with Improved water & Sanitation (%)	-0.22	0.09	0.01	-0.09	-0.12	-0.30	-0.38	-0.28	-0.19	-0.13
Food supply from vegetal products (kcal/capita/day)	-0.22	0.04	-0.06	-0.10	-0.25	-0.28	-0.27	-0.28	-0.17	-0.14
Proportion of regular daily smokers in total pop. 15+	0.07	-0.02	0.01	0.12	0.11	0.01	-0.01	0.03	0.05	0.21
Adolescent fertility rate (births per 1,000 women 15-19)	0.20	0.35	0.39	0.34	0.21	0.29	0.24	0.24	0.19	0.12

GDP per capita without the unemployment rate and LFPR, but including other control variables

The third important recessional indicator is the most accepted as a business cycle and economic trend variable, namely, the GDP per capita. In this paper it can be observed that GDP per capita has a range of impact on stroke mortality starting at lag zero and extending over at least 10 years. This can be seen with adjustment for the control variables cited and in the absence of the labour market variables. It is especially important to omit the unemployment rate in this analysis, since GDP per capita is a major cyclical predictor of unemployment. See Table 12.



**Table 12:** GDP per capita without the Unemployment rate and LFPR, but including other control variables

Predicting of cerebro-vascular diseases (ICD-10 codes I60-I69) death rate per 100,000 of population (by lagging of GDP)

Years lagged for GDP/capita	0	1	2	3	4	5	6	7	8	9	10
Overall R square	0.81	0.82	0.82	0.81	0.81	0.81	0.81	0.81	0.81	0.81	0.8
... years lag of GDP/cap. at PPP in '000 of con. 2005 int. \$	-0.42	-0.53	-0.60	-0.66	-0.65	-0.64	-0.62	-0.58	-0.55	-0.50	-0.44
Total pop. served with Improved water & Sanitation (%)	0.06	-0.08	-0.07	-0.08	-0.08	-0.08	-0.07	-0.08	-0.09	-0.11	-0.14
Food supply from vegetal products (kcal/capita/day)	-0.14	-0.17	-0.15	-0.14	-0.14	-0.14	-0.14	-0.15	-0.15	-0.16	-0.18
Adolescent fertility rate (births per 1,000 women 15-19)	-0.19	0.19	0.14	0.07	0.08	0.09	0.12	0.15	0.17	0.21	0.25
Gini index of inequality in h/hold net disposable income	0.25	0.06	0.07	0.08	0.07	0.06	0.06	0.05	0.04	0.03	0.01

Years lagged for GDP/capita	0	1	2	3	4	5	6	7	8	9	10
Overall R square	0.83	0.83	0.82	0.81	0.80	0.80	0.80	0.80	0.81	0.81	0.81
... years lag of GDP/cap. at PPP in '000 of con. 2005 int. \$	-0.44	-0.51	-0.59	-0.67	-0.68	-0.67	-0.65	-0.61	-0.56	-0.50	-0.42
Total pop. served with Improved water & Sanitation (%)	-0.11	-0.09	-0.08	-0.08	-0.08	-0.07	-0.07	-0.08	-0.09	-0.12	-0.15
Food supply from vegetal products (kcal/capita/day)	-0.19	-0.17	-0.15	-0.13	-0.14	-0.14	-0.14	-0.15	-0.15	-0.16	-0.17
Adolescent fertility rate (births per 1,000 women 15-19)	0.22	0.19	0.14	0.08	0.08	0.10	0.12	0.15	0.18	0.21	0.25
Gini index of inequality in h/hold net disposable income	0.06	0.06	0.07	0.08	0.07	0.06	0.06	0.05	0.04	0.03	0.01
Proportion of regular daily smokers in total pop. 15+	0.08	0.05	0.02	-0.03	-0.04	-0.06	-0.05	-0.04	-0.02	0.02	0.06

### Expansion of model to include GDP per capita at 10 year's lag

In replicating the original heart disease mortality model to examine whether it applies to stroke mortality, the 7-year lag for GDP per capita was taken as the optimum lag originally found for heart disease mortality. However, it had subsequently been determined (see above) that the impact of GDP per capita on stroke mortality extends over at least a decade. Thus, the labour force variables predicting stroke mortality were retested in a model that contained GDP per capita at lag 10, the unemployment rate at lag 2, and LFPR at lag zero. All other control variables were included. Results were virtually the same as found when replicating the original heart disease model and GDP was estimated earlier at a lag of seven years. See Table 13.



**Table 13:** Expansion of model to include GDP per capita at 10 year's lag

Death rate per 100,000 of total population by age	asdr	<15	15-24	25-34	35-44	45-54	55-64	65-74	75-84	85+
Overall R square	0.78	0.37	0.34	0.65	0.66	0.78	0.82	0.82	0.76	0.56
10 y. lag of GDP/cap. at PPP in '000 of con. 2005 int. \$	-0.21	0.07	0.03	-0.17	-0.24	-0.19	-0.16	-0.18	-0.25	-0.16
Labour force participation as prop. (%) of total pop. 15+	-0.20	0.16	-0.08	-0.07	-0.16	-0.16	-0.17	-0.20	-0.18	-0.22
2 y. lag of Unemploy. rate as % of total labour force 15+	0.09	0.13	0.02	-0.03	0.00	0.09	0.10	0.09	0.08	0.08
Total pop. served with Improved water & Sanitation (%)	-0.22	0.05	-0.01	-0.14	-0.14	-0.26	-0.36	-0.28	-0.18	-0.11
Food supply from vegetal products (kcal/capita/day)	-0.24	0.03	0.00	-0.14	-0.26	-0.28	-0.27	-0.29	-0.20	-0.16
Proportion of regular daily smokers in total pop. 15+	0.08	0.01	0.00	0.11	-0.03	-0.05	-0.01	0.04	0.07	0.22
Adolescent fertility rate (births per 1,000 women 15-19)	0.21	0.46	0.46	0.33	0.24	0.27	0.23	0.23	0.21	0.15
Gini index of inequality in h/hold net disposable income	0.05	-0.01	-0.09	0.05	-0.04	-0.05	-0.05	-0.02	0.10	0.15

Death rate per 100,000 of male population by age	asdr	<15	15-24	25-34	35-44	45-54	55-64	65-74	75-84	85+
Overall R square	0.78	0.42	0.35	0.64	0.69	0.77	0.81	0.80	0.75	0.52
10 y. lag of GDP/cap. at PPP in '000 of con. 2005 int. \$	-0.25	0.03	0.02	-0.17	-0.28	-0.26	-0.19	-0.24	-0.28	-0.19
Labour force participation as prop. (%) of total pop. 15+	-0.15	0.06	-0.13	0.01	-0.19	-0.13	-0.14	-0.15	-0.11	-0.15
2 y. lag of Unemploy. rate as % of total labour force 15+	0.07	0.13	-0.03	0.07	0.00	0.09	0.08	0.06	0.06	0.05
Total pop. served with Improved water & Sanitation (%)	-0.19	0.07	-0.07	-0.18	-0.13	-0.21	-0.31	-0.23	-0.15	-0.08
Food supply from vegetal products (kcal/capita/day)	-0.25	0.12	0.03	-0.16	-0.24	-0.26	-0.26	-0.31	-0.20	-0.13
Proportion of regular daily smokers in total pop. 15+	0.09	0.00	0.03	0.03	-0.11	-0.07	0.01	0.05	0.10	0.24
Adolescent fertility rate (births per 1,000 women 15-19)	0.23	0.47	0.38	0.25	0.22	0.26	0.22	0.22	0.24	0.18
Gini index of inequality in h/hold net disposable income	0.06	-0.16	-0.10	0.10	0.04	0.00	0.00	0.04	0.09	0.12

Death rate per 100,000 of female population by age	asdr	<15	15-24	25-34	35-44	45-54	55-64	65-74	75-84	85+
Overall R square	0.76	0.24	0.22	0.42	0.56	0.75	0.81	0.81	0.76	0.54
10 y. lag of GDP/cap. at PPP in '000 of con. 2005 int. \$	-0.17	0.09	0.05	-0.06	-0.13	-0.06	-0.11	-0.13	-0.22	-0.13
Labour force participation as prop. (%) of total pop. 15+	-0.24	0.16	-0.07	-0.25	-0.14	-0.18	-0.18	-0.23	-0.23	-0.23
2 y. lag of Unemploy. rate as % of total labour force 15+	0.11	0.11	0.10	-0.11	-0.05	0.05	0.11	0.10	0.10	0.09
Total pop. served with Improved water & Sanitation (%)	-0.23	0.07	0.02	-0.08	-0.16	-0.37	-0.42	-0.31	-0.20	-0.12
Food supply from vegetal products (kcal/capita/day)	-0.25	-0.02	-0.07	-0.10	-0.23	-0.26	-0.27	-0.29	-0.21	-0.18
Proportion of regular daily smokers in total pop. 15+	0.07	0.01	-0.02	0.18	0.15	0.02	-0.01	0.03	0.05	0.20
Adolescent fertility rate (births per 1,000 women 15-19)	0.21	0.33	0.40	0.30	0.23	0.30	0.25	0.25	0.20	0.15
Gini index of inequality in h/hold net disposable income	0.03	0.10	0.01	-0.01	-0.15	-0.11	-0.11	-0.07	0.10	0.14

### Expansion of model to include unemployment rate at 3 year's lag

It was found that there is a somewhat stronger relation between the unemployment rate and stroke mortality when the unemployment rate is at a lag of 3 rather than 2 years. This would mean that the unemployment rate lag effect for stroke occurs slightly later than for heart disease mortality. The stroke mortality model was then tested thoroughly for total mortality as well as by sex and age. The unemployment rate was included at lag 3, the LFPR at lag zero and the GDP per capita at lag 10. The unemployment rate was significant in the age groups 45 – 84 for total stroke mortality. For males, it was significant for the working ages 45 – 64 and for females 45 – 84. At the same time, for males the labour force participation rate influences stroke mortality (inversely) in the ages 35 – 85 +, while for females it influences stroke mortality over 25 – 85+. Clearly, the influence of labour market variables on stroke mortality is greater for females than for males. See Table 14.

Table 14: Expansion of model to include unemployment rate at 3 year's lag

Predicting of cerebro-vascular diseases (ICD-10 codes I60-I69) death rate per 100,000 of population (with UER at lag 3, by sex and age)

Death rate per 100,000 of total population by age	asdr	<15	15-24	25-34	35-44	45-54	55-64	65-74	75-84	85+
Overall R square	0.78	0.36	0.37	0.63	0.66	0.77	0.81	0.81	0.76	0.55
10 y. lag of GDP/cap. at PPP in '000 of con. 2005 int. \$	-0.24	0.05	0.04	-0.19	-0.26	-0.20	-0.18	-0.20	-0.29	-0.20
Labour force particip. as prop. (%) of total popul. 15+	-0.19	0.16	-0.07	-0.06	-0.15	-0.15	-0.16	-0.20	-0.17	-0.20
3 y. lag of Unemploym. rate as % of total labour force 15+	0.08	0.12	0.07	-0.02	0.01	0.11	0.09	0.08	0.08	0.04
Total pop. served with Improved water & Sanitation (%)	-0.20	0.06	0.01	-0.12	-0.11	-0.23	-0.32	-0.25	-0.17	-0.12
Food supply from vegetal products (kcal/capita/day)	-0.22	0.05	-0.02	-0.12	-0.25	-0.27	-0.26	-0.28	-0.17	-0.13
Proportion of regular daily smokers in total pop. 15+	0.06	-0.01	-0.01	0.10	-0.04	-0.06	-0.02	0.02	0.05	0.22
Adolescent fertility rate (births per 1,000 women 15-19)	0.21	0.47	0.46	0.35	0.24	0.28	0.22	0.23	0.21	0.14

Death rate per 100,000 of male population by age	asdr	<15	15-24	25-34	35-44	45-54	55-64	65-74	75-84	85+
Overall R square	0.78	0.38	0.40	0.62	0.69	0.77	0.80	0.80	0.76	0.53
10 y. lag of GDP/cap. at PPP in '000 of con. 2005 int. \$	-0.28	0.03	0.03	-0.20	-0.30	-0.27	-0.21	-0.27	-0.32	-0.22
Labour force particip. as prop. (%) of male popul. 15+	-0.13	0.04	-0.12	0.02	-0.17	-0.12	-0.13	-0.14	-0.09	-0.14
3 y. lag of Unemploym. rate as % of male labour force 15+	0.09	0.12	0.04	0.00	0.01	0.10	0.09	0.06	0.06	0.03
Total pop. served with Improved water & Sanitation (%)	-0.18	0.06	-0.04	-0.15	-0.12	-0.19	-0.28	-0.21	-0.15	-0.09
Food supply from vegetal products (kcal/capita/day)	-0.23	0.11	0.01	-0.13	-0.22	-0.25	-0.25	-0.28	-0.17	-0.10
Proportion of regular daily smokers in total pop. 15+	0.07	-0.01	0.01	0.03	-0.10	-0.07	-0.01	0.03	0.08	0.23
Adolescent fertility rate (births per 1,000 women 15-19)	0.23	0.48	0.38	0.27	0.23	0.27	0.21	0.22	0.23	0.18

Death rate per 100,000 of female population by age	asdr	<15	15-24	25-34	35-44	45-54	55-64	65-74	75-84	85+
Overall R square	0.76	0.23	0.23	0.43	0.54	0.74	0.81	0.81	0.76	0.53
10 y. lag of GDP/cap. at PPP in '000 of con. 2005 int. \$	-0.20	0.05	0.05	-0.02	-0.13	-0.05	-0.12	-0.14	-0.26	-0.18
Labour force particip. as prop. (%) of female popul. 15+	-0.22	0.18	-0.08	-0.23	-0.16	-0.18	-0.18	-0.22	-0.21	-0.22
3 y. lag of Unemploym. rate as % of female labour force 15+	0.09	0.09	0.08	-0.05	-0.04	0.09	0.08	0.09	0.09	0.06
Total pop. served with Improved water & Sanitation (%)	-0.21	0.08	0.01	-0.08	-0.13	-0.32	-0.38	-0.28	-0.19	-0.13
Food supply from vegetal products (kcal/capita/day)	-0.23	0.04	-0.07	-0.11	-0.25	-0.28	-0.27	-0.28	-0.18	-0.15
Proportion of regular daily smokers in total pop. 15+	0.06	0.01	-0.02	0.15	0.12	0.01	-0.02	0.02	0.04	0.20
Adolescent fertility rate (births per 1,000 women 15-19)	0.20	0.35	0.40	0.33	0.21	0.30	0.25	0.24	0.19	0.12

## ***VIII. Discussion and Interpretation***

The primary observation is that the unemployment rate has a positive relationship to stroke mortality that is similar to that found earlier for heart disease mortality in the EU. The only notable difference is that the unemployment rate can be seen to influence heart disease mortality within the first few months of an increase in unemployment (i.e., within the same contemporaneous year), while there is a lag of approximately two years after which increased stroke mortality follows a heightened unemployment rate. However, for both stroke and heart disease, increased labour force participation rates predict decreased mortality contemporaneously—i.e., as soon as the LFPR increases.

This raises interesting questions about the interpretation of the unemployment rate—as a measure of joblessness—versus that of the LFPR as measuring population involvement in economic activity. Clearly, these measures are not the inverse of each other. For example, as the EU unemployment rate continued to increase after 2009, the LFPR continued to increase, largely for women over 45.

The LFPR is certainly a more far-reaching indicator of labour market activity, compared with the unemployment rate, since the unemployment rate, as a measure of job loss, nevertheless excludes many of the long-term jobless who have left the labour force, being discouraged in their efforts to find employment. At the same time, the LFPR includes new and older entrants into the labour force who then comprise a part of the employment-to-population ratio. It is possible that being at work—as a health predictor—is not exactly the opposite of either losing work (or being jobless and seeking work)—as a health or illness predictor.

As to the mechanism(s) whereby job loss predicts heart disease and stroke mortality, the traditional perspective would point to the concept of “stress.” This mechanism could be manifested in psychophysiological responses to shock and loss, such as increased blood pressure, assuming the inability to cope over a sustained period of time (Matthews et al., 2001; Esch 2002). Similarly, the mental stress could result in behavioural responses including inability to stop smoking, excessive use of alcohol, weight gain or consumption of unhealthful diets (Montgomery et al. 1998). Additionally, major stress could engender depression, a risk factor for cardiovascular disease, withdrawal or hostile emotional responses which strain the stability of social relations among friends and family. Such potential damages to social ties are an important source of increased morbidity and mortality in the epidemiological literature (Seeman, 1996). Finally, it should simply be stated that impoverished conditions, often brought on by unemployment are well known to be associated with cardiovascular diseases, including stroke (Galobardes and Lynch, 2006).

The issues of economic life stress and emotional and behavioural responses to it are relevant to the question of why the unemployment rate, and the LFPR, should apparently influence heart disease and stroke mortality beyond the usual ages of working life—i.e., after 65, in EU countries with extensive pensions, government financed health care and other social welfare systems. This paper refers to these overarching influences of the labour force variables as “spread effects.” An interpretation would be that loss of employment for the principal (or any) family breadwinner affects the entire family (or extended family members). This extension refers to spouses or partners as well as to children and the elderly. Of course, cardiovascular illness, as a cause of death, is a health outcome that largely involves the elderly who are not frequently in the labour force. Thus, the possible mechanisms of disturbed financial support, or damage to social ties—and lack of social support (Uchino, 2006)—may plausibly be the main linkages of national patterns of employment loss to cardiovascular mortality. Also, it is possible that under significant conditions of stress, some persons do not seek appropriate health care, or do not comply with medical recommendations, even where such health care is otherwise accessible.

## **IX. Conclusions**

### **The unemployment – heart disease mortality relation**

The finding that the unemployment rate at lags of 0-5 years is positively related to Ischemic Heart Disease (IHD) mortality is consistent with epidemiological literature at the individual level of analysis. This substantial literature indicates that unemployment is a risk to heart disease morbidity and mortality. A modifying factor is that the unemployment rate at lag 0 is positively but not significantly related to IHD mortality – without control for other variables. However, with adjustment even for GDPpc, the unemployment rate at lag 0 is positively and significantly related to IHD mortality rates (with GDPpc lagged, e.g. 5 years).

The finding in this study that the unemployment rate lag 0 is positively related to IHD mortality is somewhat different from the results of 20<sup>th</sup> century time-series studies which show non-significant relations to IHD mortality of unemployment rates lagged 0 and 1 year, but strong positive relations of the unemployment rate at lags of 2 and 3 years. This finding is consistent with 20<sup>th</sup> century time-series studies showing the relation at unemployment rate lag 2, 3 and longer years, especially since the current work on IHD shows the peak relationship of unemployment rates to be at a lag of 2 years.

Somewhat different findings cited in this study on the relation of unemployment over 0-5 years to a major cause of mortality could be due to: (a) the usually large and intense effect of the current Great Recession, (b) the sample consisting of European Union countries only and (c) IHD mortality being used as the health outcome (as compared to other health measures).

With adjustment for GDPpc, Southern Europe and other economic and life style variables, the unemployment rate at lags 0, 1, 2 remains significantly related to IHD mortality and the unemployment rate lag 2 is shown to have the strongest relationship. However, GDPpc shows the strongest overall relation to IHD mortality (compared with any other variable in this study) with the highest R<sup>2</sup>, coefficient and standardized data for GDPpc lagged 0-10 years.

Labour market variables other than the unemployment rate show strong predictability to IHD mortality. Indeed, they are stronger than the assessed impact of the unemployment rate. These include labour force participation and self-employment at lag 0 with negative signs, and employment in agriculture as a percent of total employment with a positive sign at lag 0.

Poverty indices are positively related to IHD mortality. These include percent of the population (not) served with improved water and sanitation, the adolescent fertility rate and employment in agriculture as proportion of total employment. Southern Europe (as a dummy or binary variable) shows a negative relation to IHD mortality, supporting the hypotheses of healthy Mediterranean diet, relatively mild winter temperatures, and possibly stronger social (family and community) ties.

In summary, the unemployment rate at lags of 0-2 years show strong positive relations to IHD mortality over 2000-2010 in European countries. These relations hold over at least 35-74 years of age and even over 75-84 and 85+ years of age. These relations hold regardless of whether less than 3 or more than 10 variables are used as controls.

## **The unemployment – stroke mortality relation**

The study on stroke mortality rates similarly indicates that they have been significantly influenced by unemployment rates during the period preceding and including the Great Recession, over 2000 – 2011 in European Union countries. These results, also obtained via pooled cross-sectional time-series multiple regression analysis, are found even under controls for (1) other major economic indicators of recession—the labour force participation rate and GDP per capita—as well as (2) smoking and dietary risks to stroke, and (3) measures of severe chronic poverty such as (lack of) access to potable water and sanitation, and the adolescent fertility rate. It is again noteworthy that the labour force participation rate has a considerably greater impact on stroke mortality than the unemployment rate, which is the principal subject of this analysis. From this perspective, it is remarkable that the previous literature, both in epidemiology and in macroeconomic research, hardly ever considers the relation of labour force participation to health, although the relation of unemployment to damaged health is well attested in individual-level epidemiologic studies.

The macroeconomic relation of unemployment rates to stroke mortality rates is similar to that of unemployment rates to heart disease mortality rates, except that the lag between unemployment rates and stroke mortality rates appears to be longer. That the relations between unemployment rates and stroke should resemble that shown in heart disease mortality is not surprising in that both are cardiovascular illnesses and similar psychological, biological and medical mechanisms are probably involved in both types of illnesses.

It can now be seen that relations between unemployment and cardiovascular illness, that have been observed in epidemiological studies of individual persons, can be observed as well at the national level with macroeconomic variables, such as the unemployment rate, in contemporary times. This means that national and regional policies that could prevent or reduce unemployment rates, or mitigate the related economic losses, and finance active labour market policies, have a much broader function. Such policies could enhance population health and life expectancy, and probably reduce high levels of national health care expenditures.

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## ***XI. Appendix 1: Selected studies on unemployment, income and CVD***

### **Selected epidemiological studies on income, unemployment in relation to CVD**

#### *Income and CVD*

The review of the literature by Galobardes, Smith and Lynch (2006) found that the current evidence from individual-level studies shows that those who experienced less favourable childhood socioeconomic circumstances are at greater risk for developing and dying of CVD in adulthood. Thus, if the social patterning of CVD outcomes observed in adulthood is already determined by socioeconomic circumstances experienced in childhood, interventions that aim at reducing economic, political, social, and cultural disadvantages should start early in life.

Frankel, Smith, and Gunnell (1999) state that the relation between childhood socioeconomic position and adult cardiovascular mortality is examined in 3,750 individuals whose families took part in the Carnegie survey of family diet and health in England and Scotland between 1937 and 1939.

The study by Kaplan and Keil (1993) says researchers are still learning about different modifiable factors that may influence cardiovascular diseases. Socioeconomic status may provide a new focus. During 40 years of study there has been a consistent inverse relation between cardiovascular disease, primarily coronary heart disease, and many of the indicators of SES. There is some evidence that areas with the poorest socio-environmental conditions experience later onset in the decline in cardiovascular mortality.

Brenner and Mooney (1982) developed a multivariate model of the impact of more fundamental changes in the socioeconomic and bio-physical environments and applied it to cardiovascular disease mortality rates, by sex, in England and Wales and Scotland during 1955-1976. The predictive model includes factors associated with (1) long-term growth in the economy, (2) deleterious behavioural risk factors loosely associated with economic growth—especially cigarette consumption per capita, (3) economic instability—especially recession as indicated by factors related to unemployment, income loss, and recessionary declines in average weekly hours worked in manufacturing industries, (4) health care, and (5) physical environmental disturbances—especially very cold temperatures.

#### *Unemployment and CVD*

Gallo, Teng, Falba, Kasl, Krumholz and Bradley (2006) state that after controlling for established predictors of the outcomes, displaced workers had a more than twofold increase in the risk of subsequent MI and stroke relative to working persons. Physicians who treat individuals who lose jobs as they near retirement should consider the loss of employment a potential risk factor for adverse vascular health changes.

Filate, Johansen, Kennedy and Tu (2003) observed significant regional variations in CVD mortality rates per 100,000 populations. Newfoundland and Labrador had the highest CVD and IHD mortality rates, while Nunavut and the Northwest Territories had the lowest CVD and IHD mortality rates. Health region smoking and unemployment rates were identified as the most important factors associated with CVD and IHD mortality at the health region level.

The study by Henriksson, Lindblad, Agren, Nilsson-Ehle and Rastam (2003) was a prospective study of men, all the same age and without obvious individual threats of future job loss. It was found that cross-sectional associations between unemployment and CVD risk factors were lost in times of lower employment rates. Thus it is vital to consider general unemployment rates in society when the attributable risk of unemployment is estimated in health promotion strategies aimed at preventing cardiovascular disease.

Morris, Cook, Shaper (1994) indicate that after adjustment for a wide range of background variables including social class, health behaviour, and health status before loss of employment the mortality still remained significantly raised. This suggests a causal effect, but set against this is the non-specific nature of the effect, with the increased mortality involving both cancer and cardiovascular disease.

The study by Sorlie and Rogot (1990) found that employed persons aged 25-64 years were found to have standardized mortality ratios from 61% to 74% of the average, depending upon their sex and race. Unemployed men had standardized mortality ratios slightly above 100, but these values were 1.6 and 2.2 times higher than those for employed white men and black men, respectively.

Crombie, Kenicer, Smith and Tunstall-Pedoe (1989) indicate strong associations with several measures of social disadvantage, the strongest being with percentage of male unemployment. A fitted multiple regression model with mortality from coronary heart disease in men found independent effects of two social variables (percentage male unemployment and percentage social class III-V) and one climatic factor (rainfall).

The study by Moser, Fox, Goldblatt and Jones (1986) provides evidence which could be seen as supporting hypotheses about relationships between stress and overall mortality, with a marked excess for suicides. The evidence with respect to ischaemic heart disease is positive but less convincing with excess mortality from this cause principally occurring among younger unemployed men and among the wives of men who were seeking work in 1971. Given the sharp contrasts in the pattern and levels of unemployment between 1971 and 1981 it is difficult to extrapolate from these findings to the present day.

### **Selected aggregate time-series studies on unemployment, income and CVD mortality**

The time-series analysis work by Brenner (2012) shows that health expenditures, labour force participation, and GDP per capita, tend to decrease CVD mortality, controlling for consumption risk factors.

The study by Brenner (2005) shows that economic growth, cumulatively over at least a decade, is the central factor in mortality rate decline in the US over the 20th century. The net effect of increased unemployment is a substantial increase in mortality. This is also reflected in the entirely negative relation between the cumulative effects of the employment to population ratio and mortality rates over a decade.

Time-series regression analysis work by Brenner (1997) showed that, holding constant the effects of tobacco, animal fats and alcohol, increased income and social welfare expenditures are related to heart disease mortality rate declines, whereas increased unemployment and business failure rates are related to heart disease mortality rate increases over more than a decade.

Brenner's (1987b) data for nine countries in the Post World War II era were investigated: Australia, Canada, England and Wales, Denmark, Federal Republic of Germany, Finland, France, Sweden and the United States. In all nine countries unemployment and business failures are positively related to heart disease mortality, and in eight countries the trend of economic growth shows an inverse relationship.

The predictive model by Brenner and Mooney (1982) includes factors associated with (1) long-term growth in the economy, (2) deleterious behavioural risk factors loosely associated with economic growth—especially cigarette consumption per capita, (3) economic instability—especially recession as indicated by factors related to unemployment, income loss, and recessionary declines in average weekly hours worked in

manufacturing industries, (4) health care, and (5) physical environmental disturbances—especially very cold temperatures.

The findings of the study by Brenner (1971) clearly indicate that economic downturns are associated with increased mortality from heart disease and that, conversely, heart disease mortality decreases during economic upturns.

## **Selected epidemiological studies on the effects of unemployment on life style**

### *Unemployment and Alcohol*

Study results by Popovici and French (2013) show a positive and significant effect of unemployment on drinking behaviours, and the findings are robust to numerous sensitivity tests. Perhaps, macroeconomic policy decisions intended to stimulate the economy during economic downturns should also consider the avoided personal costs and externalities associated with alcohol misuse.

The study by Montgomery, Cook, Bartley and Wadsworth (1998) compared men who had never been unemployed. The adjusted relative odds amongst men with over three years of accumulated unemployment (after adjustment for possible confounding socioeconomic and behavioural factors measured prior to unemployment) were 2.15 for problem drinkers. Men who had experienced unemployment in the year prior to the interview, compared to those who had not, after adjustment, were significantly more likely to drink heavily and to have a drinking problem. Unemployment may play a significant part in establishing life-long patterns of hazardous behaviour in young men.

### *Unemployment and Diet*

The American Dietetic Association (2010) indicates that food insecurity rates in the United States parallel poverty rates, and food insecurity worsens in recessionary times. With the poor economic conditions in 2009 and 2010 in the United States, it is anticipated that food security will increase, further emphasizing the urgency of this health issue in the United States.

Dave and Kelly (2010) showed that a higher risk of unemployment is associated with reduced consumption of fruits and vegetables and increased consumption of “unhealthy” foods such as snacks and fast food. Among individuals predicted to be at highest risk of being unemployed, a one percentage point increase in the resident state’s unemployment rate is associated with a 2-8% reduction in the consumption of fruits and vegetables.

### *Unemployment and Smoking*

Novo, Hammarstrom and Janlert (2000) found that a low level of education, and among women also financial problems and motherhood, were associated with more frequent smoking. Unemployment was associated with tobacco consumption, especially among women and during the boom. Thus, smoking habits were found to be a question of both unemployment and tobacco trends in society.

Hammarstrom and Janlert (1994) showed that irrespective of early smoking, smoking habits developed more unfavourably among unemployed young people than among those with no unemployment during the period studied. The odds ratio of being a smoker at the age of 21 years when unemployed more than 20 weeks during the observation period, compared with those without or with short unemployment, was 2.44 for men and 3.45 for women. It seems that unemployment is a risk factor for development of tobacco smoking in young people, especially among women.

## *Unemployment and Social Relations*

McKee-Ryan, Song, Wanberg and Kinicki (2005) showed that unemployed individuals had lower psychological and physical well-being than did their employed counterparts. Within unemployed samples, work-role centrality, coping resources (personal, social, financial, and time structure), cognitive appraisals, and coping strategies displayed stronger relationships with mental health than did human capital or demographic variables.

The study by McLoyd, Jayaratne, Ceballo, and Borquez (1994) showed that maternal unemployment and work interruption, influences adolescent socioemotional functioning. Current unemployment, but not past work interruption, had a direct effect on depressive symptomatology in mothers. As expected, depressive symptomatology in mothers predicted more frequent maternal punishment of adolescents, and this relation was fully mediated by mothers' negative perceptions of the maternal role. More frequent maternal punishment was associated with increased cognitive distress and depressive symptoms in adolescents, and consistent with predictions, these relations were partially mediated by adolescents' perceptions of the quality of relations with their mothers. Adolescents who perceived their families as experiencing more severe economic hardship reported higher anxiety, more cognitive distress, and lower self-esteem.

## **Selected epidemiological studies on the influence of stress and depression on cardiovascular disease**

Whooley and Wong (2013) showed that depression is a robust risk factor for the development of cardiovascular disease in healthy populations, and is predictive of adverse outcomes (such as myocardial infarction and death) among populations with pre-existing cardiovascular disease. An important direction critical toward meaningful progress in the field of depression and cardiovascular disease is better understanding of how depression is related to cardiovascular disorders other than CAD, including cerebrovascular disease, peripheral arterial disease, atrial fibrillation, and other arrhythmias.

The review by Peterson and Kim (2011) identifies several unresolved issues that require answers, answers that can be found with further research. One crucial but unexplored issue is whether randomized controlled trials that increase psychological health assets protect against cardiovascular events. The field of behavioural cardiology should expand its focus and make room for psychological health assets when conceptualizing how psychological factors impact cardiovascular health.

The work by Goldston and Baillie (2008) showed the volume, strength and consistency of the association in depression and CHD, the dose-relationship of the risk and the plausibility of the potential pathophysiological mechanisms have led to the conclusion that depression is an independent etiological and prognostic risk factor in CHD. Furthermore, the magnitude of increased risk incurred by depression on both the etiology and prognosis of CHD is equal to many of the conventional cardiac risk factors such as smoking, high cholesterol and hypertension. It has been proposed that depression exerts its adverse impact on the cardiovascular system directly through biological mechanisms including increases in heart rate, blood pressure, cardiac arrhythmias, platelet aggregation and inflammation, and indirectly through its association with behavioural and lifestyle factors such as smoking, physical inactivity, lack of adherence to medical regimes and social isolation.

The European Heart Network (2006) study discusses some of the biological mechanisms of stress, including regulation of cortisol levels, heart rate variability, blood pressure, plasma fibrinogen levels, inflammatory responses and other immune system reactions, and testosterone/oestrogen levels. Regeneration after stress is crucial, and sufficient high-quality deep sleep is essential for regeneration.

The work by Zellweger, Osterwalder, Langewitz and Pfisterer (2004) indicates that evidence is growing that depression per se is an independent risk factor for cardiac events in a patient population without known CAD. There are several behavioural and pathophysiologic factors that seem to link depression with development of CAD. One may hypothesize that patients treated for their depression might better adhere to risk factor modifications, prescribed medications and rehabilitation programs.

Esch, Stefano, Fricchione and Benson (2002) found that stress has a major impact upon the circulatory system. It plays a significant role in susceptibility, progress, and outcome of cardiovascular diseases. In particular, stress may cause or exacerbate disease processes depending on the type of stressor involved (e.g. physical, chemical, biological, mental, psychosocial etc.) and/or the duration of its influence on an organism. Mental and psychosocial stressors have a profound impact, and in this regard, the sympathetic nervous system represents a generally important effector of the stress response with potentially deleterious influences on the cardiovascular system.

Findings by Matthews, Gump and Owens (2001) tested the influence of chronic stress on cardiovascular and neuroendocrine responses to and recovery from acute stressors and whether the effects are gender specific. Men's basal levels and acute stress responses may be more affected by environmental factors than are women's. Relative to women in midlife, men may be at heightened risk for CVD because of enhanced acute-stress responses as well as delayed recovery after cessation of acute stress.

Wheatley (1984) found that mental stress may directly influence coronary heart disease (CHD) and also a number of its etiologic risk factors. Out of a total of 77 patients treated with a placebo, there were five cases of myocardial infarction during the trial periods as compared to no such cases among 81 patients treated with the antianxiety drugs.

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