

The Time-Varying Relationship Between Mortality and Business Cycles in the U.S.*

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Abstract

Our paper explores the relationship between mortality among working-age and non-working adults and the business cycles in the United States. We first present a theoretical model to outline the transmission mechanisms from the business cycles to health status. We use our theoretical model to motivate our empirical framework. We find overwhelming evidence of structural breaks in the relationship between mortality and the business cycles. We use a model that features time-varying parameters and variance to capture the salient fact that macroeconomic variables and their volatility have changed over time. We find that there is a strong link between mortality and the business cycles. Mortality is pro-cyclical for most age groups and the causes of death we examine in the paper. Moreover this relationship is highly time-varying. We also find that the relationship between total mortality and the business cycles has strengthened over time, especially in the late 1980s and subsequent periods. This may be partly explained by the structural change the U.S. economy experienced in the 1990s and the rise in the number of hours worked.

KEYWORDS: Mortality, Business Cycles, Time-Varying Parameters

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1 Introduction

Since the paper by Ruhm (2000), there has been a renewed interest in the relationship between health status, in particular mortality and the business cycles. Ruhm (2000, 2003) finds that mortality is procyclical, that is the secular decline in mortality accelerates during a recession and decelerates during a boom. He argues that a 1% increase in the unemployment rate leads to a 0.54% decline in the overall mortality rate. His findings contradict earlier results by Brenner (1979, 1987) who found that mortality is counter-cyclical. Brenner's results, however, have been discounted on the basis of methodological problems (see Wagstaff, 1985). The recent literature overwhelmingly also finds that mortality is pro-cyclical (Tapia Granados, 2005, Neumayer, 2004 and Miller et al., 2009). However estimates of the relationship between mortality and the business cycles vary greatly across studies.

The empirical literature on the relationship between mortality and the business cycles either use individual or aggregate data. Most, if not all the papers in this literature have modelled the relationship between mortality and the business cycles using a linear model that does not allow the parameters and/or the variances to change over time.¹ Yet, there is ample evidence that many macroeconomic time series, including the unemployment rate, real GDP and the mortality rate have been subject to important structural changes over time. For example, the volatility of many macroeconomic series in the U.S., in particular output and inflation, has declined over the years, leading to a so-called "Great Moderation". This phenomenon is well documented in the literature and there is some consensus that the Great Moderation occurred in the middle of the 1980s (McConnell and Perez-Quiros, 2000). The mortality rates for all age groups and for various causes of deaths in the U.S. have also seen a substantial decline in the last decades. Major advances in technology, medicine, and the availability of better nutrition over the years have all contributed to a significant decline in the mean and volatility of the mortality rate (Cutler, Deaton and Lleras-Muney, 2006).

The focus of our paper is to examine the time-varying relationship between mortality and measures of the business cycles which has so far remained unexplored in the literature. We model the relationship between mortality and business cycles for the U.S. over the period 1962-2006 using a time-varying parameter model (TVP) for adults 16 years and over. We use time-series data on

¹A notable exception is Tapia Granados (2005) who uses time-series for the US for the period 1900-1996 and provides estimates of the relationship between mortality and the business cycles both for the entire sample and for selected sub-periods.

total deaths, as well as age-specific and cause-specific mortality on accidents and cardiovascular diseases and examine how these correlations vary with two measures of the business cycles, namely the deviation of real GDP and unemployment from their respective trends. The framework that we use allows the relationship between mortality and the business cycles to change over time. Moreover, the TVP model that we use can also explicitly take into account the presence of heteroskedasticity, thus allowing the variance of the shocks to change over time.

Our study is motivated by the overwhelming evidence of structural change between these macroeconomics variables. Using tests for structural breaks, we find that the relationship between mortality and the business cycles has not remained constant over time. On the contrary, we find strong evidence of structural breaks and the latter provides the motivation to use a model that allows the relationship between mortality and the business cycles to change over time. Using a fixed-coefficient framework to model the relationship between these macroeconomic series would be unsuitable given the widespread evidence of instability. Doing so, that is ignoring the structural changes can lead to biased results, wrong inferences and misleading policy recommendations. Put simply, postulating that the structural relationships between mortality and the business cycles have remained unchanged, as assumed in most of the other studies in the literature that use fixed-coefficient in the face of technological advances, improvements in medicine and changes in the delivery of health care, is essentially a leap of faith.

The TVP model we employ in this paper, by allowing the coefficients to evolve stochastically over time, is very appealing since it can be applied to time series models with parameter instability. In our paper, the time variation is modeled as driftless random walks as in the work of Cooley and Prescott (1976) and is estimated using the median-unbiased estimator proposed by Stock and Watson (1998). This framework has often been used in the literature on monetary policy rules as in Cogley and Sargent (2001) in the context of a VAR or in Boivin (2006) in a single equation context.

Our paper is directly related to several other papers (Tapia Granados (2005, 2008), Neumayer (2004), Laporte (2004) and Wagstaff (1985)) that have estimated the relationship between total deaths, age-specific and cause-specific mortality and measures of the business cycles using time-series data. However, our paper differs in three important ways from the previous literature. First we examine the relationship between mortality and the business cycles using a time-varying parameter (TVP) model. Most if not all previous studies have used models that do not allow such a relationship to change over time. Second, we allow for the possibility that the variance of the shocks of the model may be changing over time. There is ample evidence that the volatility of

many macroeconomic series and the mortality rates of all age-groups have fallen over time. Previous studies have assumed that the variance of the shocks have remained constant over time. Third, we describe the various channels through which the ups and down of the economy can affect mortality using a structural model that is similar to the canonical model Grossman (1972) and to the recent papers by He, Huang and Hung (2011) and Feng and Gomis-Porqueras (2011) to motivate our empirical framework.

We find that total mortality is strongly procyclical except for the age-groups 35-54 where the evidence is mixed. The relationship between total deaths and the business cycles displays significant time-varying relationship. Our results reveal that the relationship between total mortality and the unemployment rate, especially for adults older than 35, has been stronger in the 1980s and subsequent years. This time variation in U.S. mortality can be linked to the shift and changes that occurred in the U.S. economy during that period. While there were important gains in productivity in the U.S. in the last part of the 1980s and the 1990s, there was also at the same time an increase in the number of average hours worked.² This structural change and increases in the number hours worked may have left individuals with permanently less time to devote to health enhancing activities such as home-cooked meals and exercises, thus leading to a stronger relationship between mortality and the business cycles. In contrast, before this period, mortality is found to be procyclical but the magnitude of the effect of the business cycles is smaller. We also find that deaths due to motor vehicles accidents, accidental deaths (on-the job related accidents) and deaths related to cardiovascular disease is strongly procyclical. Our results also indicate that the relationship between these causes of deaths and the business cycles has in general become smaller over the 1980s and 1990s.

The paper is organized as follows. Section 2 provides a brief review of the literature. Section 3 describes our theoretical framework. Section 4 describes the data we used in the paper. Section 5 describes the TVP model that we use and present evidence of instability in the parameters and variance of the model. We discuss our results in section 6 and section 7 concludes.

2 Literature Review

²See Cociuba and Ueberfeldt (2010). They argue that between 1980s and 2007, average hours worked in the U.S. increased by 13%

The literature on the relationship between mortality and the business cycles has been heavily influenced by a series of papers by Brenner (1979, 1987) and Ruhm (2000, 2003, 2005). In his various studies, Brenner (1979, 1987) concludes that mortality is strongly counter-cyclical, that is mortality rises in bad times. These findings have, however, been discounted because of serious statistical and methodological problems such as the choice of lag lengths and the failure to control for important determinants of mortality (see for example Laporte, 2004, Wagstaff, 1985).³

When these statistical problems are addressed, Brenner's results are often found to be very fragile. Much of Brenner's results are driven by the correlation between unemployment and mortality after the 1930s depression. The secular decline in mortality during that period was largely caused by the availability of antibiotics and improved nutrition, thus factors that may be correlated with the fall in the unemployment rate but not caused by the ups and downs of the economy. This is confirmed by Cutler, Deaton and Lleras-Muney (2006) in their research. They show that approximately two thirds of the decrease in cardiovascular mortality since 1950 can be explained by medical advances, namely increased use of non-acute medications and intensive medical therapies. They also state that reduced mortality between 1800 and 1940 came almost completely from decreases in infant and child mortality, which became half as common by 1950 than it was at the beginning of the century.

The recent literature has been heavily influenced by several papers from Ruhm (2000, 2003, 2005). He uses state-level data on unemployment and mortality for the U.S. covering the period 1972-1991 and he shows that most causes of mortality, especially cardiovascular deaths, are strongly pro-cyclical. He finds that a 1% increase in the unemployment rate leads to a 0.54% decrease in total mortality rate. He also presents evidence of the procyclicality of mortality for many cause-specific diseases and deaths, namely heart disease, pneumonia, infant and neonatal deaths, motor-vehicle and on the job accidents.

Ruhm (2000) argues that the secular decline in mortality slows down during a boom mainly because the opportunity cost of leisure increases. Individuals during an economic boom take advantage of the favourable labour market by working more. As a result, they devote less time to leisure, health enhancing activities such as exercising, cooking meals at home, sleeping and caring for the sick and elderly but more time to unhealthy activities such as drinking and smoking. Ruhm (2000, 2003, 2005) argues that this opportunity cost of time is an important transmission channel from business cycles to mortality.

³A more comprehensive summary of the literature can be found in Ruhm (2005).

He also argues that elevated stress levels and longer working hours during an economic upturn lead to more job-related fatal accidents and injuries. Higher stress levels may also have a direct negative impact on health status as it may accelerate the rate at which our stock of health depreciates much in the same way as a machine would depreciate more rapidly if used more intensively. Ruhm (2000, 2003, 2005) also finds that motor-vehicles accidents tend to increase during an economic expansion as the volume of highway traffic tend to increase during these periods.

Recent papers that use time-series rather than panel data have corrected many of the statistical problems that plagued Brenner's analysis and have found similar results as Ruhm (2000). Laporte (2004) uses an error-correction model and finds that increases in unemployment are associated with reduced mortality risks. Tapia Granados (2005) uses annual data for the U.S. from 1900 to 1996 and finds that the age-specific and cause-specific mortality over that period is pro-cyclical. In this paper, he also conducts sub-sample estimation and provides some analysis of the stability of the coefficients across periods. To our knowledge, it is the only paper employing time-series data that conducts such an exercise to verify for the potential of structural breaks.

In a recent paper, Miller and al. (2009) have shed more light on Ruhm's results. They argue that for working-age adults, the bulk of the positive correlation between mortality and business cycles is explained by motor-vehicles accidents and not work-related accidents or the labour-leisure choices made by individuals. Moreover, they also find that the majority of the increase in mortality due to cardiovascular diseases during an economic boom, a very important cause of mortality in the U.S., occur among adult individuals who are not typically in the labour force, that is those aged 65 and older. This result also suggests that the main driving force behind the rise in mortality above its trend during an economic boom may not be related to less healthy lifestyle or elevated stress level but more to factors that are indirectly linked to the business cycles, such as pollution and disruptions in social support and social networks.

3 Theoretical Model

This section describes our theoretical model that will serve as motivation for our empirical framework. The model that we employ is similar to Grossman (1972), and recent papers in the literature by He, Huang and Hung (2011) and Feng and Gomis-Porqueras (2011). The model is very similar to a standard real business cycles framework except that it features endogenous health. In the spirit of Grossman (1972), there is a demand for health since consumers value health directly since it brings direct utility and increases production opportunities. Health is an input in the production

process as it increases the efficiency of workers, reflecting the human capital aspect of health.

3.1 The model

The economy consists of a representative agent who derives utility from consumption, c , leisure, ℓ and health h . The representative agent maximizes lifetime utility

$$\sum_{t=0}^{\infty} \gamma^t u(c_t, h_t, \ell_t) \quad (1)$$

subject to the sequence of budget constraints:

$$c_t + m_t + i_t \leq y_t \quad (2)$$

$$n_t + \ell_t = 1 \quad (3)$$

$$y_t = e^{a_t} k_t^\omega (n_t h_t)^{1-\omega} \quad (4)$$

$$k_{t+1} = i_t + (1 - \delta_k) k_t \quad (5)$$

$$h_{t+1} = (1 - \delta_h - \delta(h_t)) h_t + (m_t^\phi \ell^{1-\phi}) \quad (6)$$

$$a_{t+1} = \rho a_t + \epsilon_{t+1} \quad (7)$$

where n_t is the amount of hours worked, m_t the amount of medical services purchased in period t , i investment in physical capital, y output produced and k_t the stock of physical capital. The final good in the model can be either consumed (c), invested into medical care or health stock (m) or into physical capital (i).⁴ In the model each individual is endowed with one unit of time that is devoted to either working or enjoying leisure. The production function includes physical capital and effective labour represented by the term (nh) . Health in the production function can be interpreted as human capital, as an increase in the stock of health increases the productivity of workers in the model. Moreover, as shown by He, Huang and Hung (2011), including health in the production function is important to capture the procyclicality of job-related accidents. In the model, as hours worked, n , increases, everything else equal, the stock of health h falls because of the substitution between these two terms in the production function. One can argue that this decrease in the health stock represents higher on the job accidents as production increases. The capital accumulation identity in the model is standard (equation 5).

⁴The consumption good can be a composite good comprising of goods that enhance health such as nutritious food and other goods that leads to the deterioration of health, such as drinking and smoking.

Following Grossman (1972), we assume that the health stock depends on last period's health stock and on the time and effort allocated by each agent to replenish their health stock. Equation 6 describes how the health stock evolves over time. The stock of health depreciates over time and this depreciation depends on a constant rate (due to age for example) and also on how intensive the workers work. Similar to models with variable capital utilization where the stock of capital depreciation depends on how intensive capital is used, we argue that the rate at which the health stock depreciates also depends on how intensive the agent is working. In this case, we follow Burnside and Eichenbaum (1996) and the Real Business Cycles literature and assume that $\delta(h_t) = \frac{n_t^\psi}{\psi}$. As individuals work more, this increases the depreciation of their health stock. This channel captures Ruhm's idea that longer working hours increases the level of stress of individuals, thus exerting a negative effect on their health status.

To summarize the various channels through which health is affected in the model, let us assume a positive technology shock that leads to an increase in output and hours worked. In the model, the higher output leads to an increase in expenditure devoted to health care (m_t increases). As a result, higher medical expenditures have a positive impact on the health stock of the representative agent. On the other hand, the positive technology shock leads to higher wages. This in turn has two effects on the demand for leisure. There is an income effect that increases the demand for leisure but at the same time the higher wages increases the opportunity cost of leisure and agents substitute away from leisure and work more. As agents work more, this has a detrimental effect on health in the model through two channels. First, longer working hours implies that the utilization rate $\delta(h_t)$ increases, leading to a faster depletion of the health stock. Moreover, longer working hours, implies that the stock of health h falls because of the substitution between these two terms in the production function. Second, as hours worked increase due to the positive productivity shock, this leaves less time for health enhancing activities as leisure falls. Since the latter is a direct input in the production of health, the stock of health thus declines as a result. Health status is procyclical if the negative consequences of the positive technology shock on health outweighs the positive benefits it conveys.

Therefore the objective of the consumer is to maximize utility subject to the constraints given by equations (2-7) and the initial conditions. The first order conditions of the model are,

$$c_t : \gamma^t u_{c_t} - \lambda_{1t} = 0 \tag{8}$$

$$k_{t+1} : -\lambda_{1t} + \lambda_{1t+1} \left[\omega \left(\frac{y_{t+1}}{k_{t+1}} \right) - (1 - \delta_k) \right] = 0 \tag{9}$$

$$m_t : -\lambda_{1t} + \lambda_{2t} \left[\phi m_t^{\phi-1} \ell_t^{1-\phi} \right] = 0 \quad (10)$$

$$n_t : -\gamma^t u_{n_t} + \lambda_{1t} (1 - \omega) \left(\frac{y_t}{n_t} \right) - \lambda_{2t} \left[n_t^{\psi-1} h_t + (1 - \phi) m_t^\phi \ell_t^{-\phi} \right] = 0 \quad (11)$$

$$h_{t+1} : \gamma^{t+1} u_{h_{t+1}} - \lambda_{2t} + \lambda_{1t+1} (1 - \omega) \left(\frac{y_{t+1}}{h_{t+1}} \right) + \lambda_{2t+1} \left(1 - \delta_h - \frac{n_{t+1}^\psi}{\psi} \right) = 0 \quad (12)$$

where λ_{1t} is the Lagrange multiplier on the resource constraint and λ_{2t} is the Lagrange multiplier on the health accumulation identity.

The first-order conditions can be combined and we obtain:

$$u_{c_t} = \gamma u_{c_{t+1}} \left[(1 - \delta_k) + \omega \left(\frac{y_{t+1}}{k_{t+1}} \right) \right] \quad (13)$$

$$\frac{u_{n_t}}{u_{c_t}} = (1 - \omega) \left(\frac{y_t}{n_t} \right) - \left[\frac{n_t^{\psi-1} h_t}{\phi m_t^{\phi-1} \ell_t^{1-\phi}} + \left(\frac{1 - \phi}{\phi} \right) \frac{m_t}{\ell_t} \right] \quad (14)$$

$$\frac{u_{h_{t+1}}}{u_{c_{t+1}}} = \left[\frac{(1 - \delta_k) + \omega \left(\frac{y_{t+1}}{k_{t+1}} \right)}{\phi m_t^{\phi-1} \ell_t^{1-\phi}} \right] - \left[(1 - \omega) \left(\frac{y_{t+1}}{h_{t+1}} \right) \right] - \left[\frac{1 - \delta_h - \frac{n_{t+1}^\psi}{\psi}}{\phi m_{t+1}^{\phi-1} \ell_{t+1}^{1-\phi}} \right] \quad (15)$$

Equation 13 is the Euler condition for consumption. Equation 14 is the intratemporal condition that relates the optimal choice between leisure and work. In the standard RBC model, the second term in square bracket in equation 14 is zero. This second term in our model reflects the fact that an additional unit of leisure conveys an additional benefit in the model. An increase in leisure not only increases utility directly but the latter can also be used to increase the stock of health capital since leisure is an input in health production. As a result, the marginal benefit of an extra unit of leisure is actually greater in this model.

Equation 15 reflects the intertemporal condition between health and consumption. An additional unit of spending on health care has two benefits according to equation 15. First, it increases directly labour productivity since health is an input in production. This is represented by the second term in square bracket on the right hand side of the equation. Second, an additional unit of health expenditure contributes directly to the current and future stock of health. This in turn increases health stock and output and consumption in the future. This effect is captured by the third term in square bracket on the right hand side of the equation. An increase in health expenditure has however an opportunity cost in terms of forgone physical investment and future output and consumption. This is represented by the first term in square bracket on the right hand side of the equation.

4 Empirical Model

Our empirical specification is motivated by equation 15. According to equation 15, health status will depend on output (y), health care expenditure (m), and hours worked n_t . Health care expenditures and hours worked are both positively correlated with output. In our baseline model, we use two measures of the business cycles, the deviations of output and unemployment from trend to capture the effects of these variables on health status which is the mortality rate in our empirical framework. Instead of using a model with constant parameters and constant variance, we employ a model with time-varying parameters (TVP) with heteroskedastic variance. As explained in the introduction, there is overwhelming evidence of structural breaks in the relationship between mortality and the business cycles. Ignoring these structural changes can lead to biased results, wrong inferences and misleading policy recommendations.

The TVP model with heteroskedastic variance that we use has many advantages over a framework that assumes that the correlation between mortality and the economy and the volatility of the shocks have remained constant over time or even over a framework that model structural breaks using a discrete break model (mostly by splitting the sample into several periods). Split-sample estimates as used in Tapia Granados (2005) imply that all parameters in the model are simultaneously affected by the estimated discrete change and that the variance across periods does not change over time. However, in practice, the parameters of the model may evolve differently over time making the TVP model more appealing since the latter is able to uncover how each parameter is slowly changing over time independently of each other.

Moreover, the discrete break model assumes that the change in the parameters is sudden. There is no guarantee, however, that this is the case. For example, the constant improvement in medical technology and care would most likely imply that the estimated relationship between mortality and the business cycles is gradually changing over time. This relationship would be better represented by continuous drifts in the estimated parameters rather than by a sudden change in parameters. Another advantage of the TVP model over a model that uses a discrete break, is that with the TVP model, we do not need to find when the break occurs, an exercise that involves considerable uncertainty.⁵ Moreover, if one is using a framework that allows for multiple break dates, it may be simply infeasible to estimate the model if the number of breaks is large relative to the sample size or if the break dates are close to each other.

Compared to the constant parameter model, the TVP model that we use in this paper may be

⁵We will, however, need the break dates if we assume that there are changes in the volatility of the shocks.

more robust to model misspecification, in particular if structural breaks in the mean or variance or non-linearities are the causes of the misspecification. For example, the secular decline in mortality in some periods may accelerate or decelerate independently of how the economy is behaving. It may be due to the discovery of a new vaccine that eradicates or reduces deaths from a certain disease. In that case, the relationship between mortality and the business cycles becomes weaker which implies a non-linear relationship and/or a change in the coefficients of the model. In both cases, the TVP model is well suited to deal with such changes since it allows each coefficient to change independently of each other and to take any time paths.

The model that we use is given by:

$$\begin{aligned} M_t &= \alpha_t + \theta_t M_{t-1} + \beta_t X_t + \epsilon_t \\ M_t &= \Phi_t' Z_t + \epsilon_t \end{aligned} \tag{16}$$

where M_t is the deviation of the log of the mortality rate from its trend estimated using an HP filter and X_t is either the log deviation of real GDP from its trend (output-gap) or the log deviation of the unemployment rate from its trend level (unemployment gap) also estimated using a HP filter. All the parameters of the model are indexed with a time subscript indicating that they are time-varying. The vector Φ contains the time-varying parameters of the model and the vector Z the corresponding regressors. In our baseline model, we set θ_t to zero and do not include the lagged mortality rate as a regressor. We include the lagged mortality rate in our second specification to account for the possible dynamics in the mortality rate but also to purge the equation from any serial correlation.⁶

The time variation is modeled as driftless random walks:

$$\Phi_t = \Phi_{t-1} + \eta_t \tag{17}$$

where $E(\eta_t) = 0$

The parameters of the TVP model, including the variance of η_t can be jointly estimated by maximum likelihood estimation (MLE) using the Kalman filter. As explained by Stock and Watson (1998), this procedure can run into the so-called pile-up problem if the standard deviations of the innovations of the random-walk components, that is η_t are very small. The pile-up problem typically occurs if the variation of the parameters from period-to-period is small. This may happen even if

⁶The paper focuses on the model with no lagged dependent variable. As a robustness check, we have performed several simulations by including the lagged dependent variable as a regressor. We find that the explanatory power of the business cycles indicator is weakened.

the time variation in the parameters is statistically significant and important. The pile-up problem leads to a signal-to-noise ratio $\frac{\sigma_\epsilon}{\sigma_\eta}$ that is zero. In this case, maximum likelihood methods will mistakenly yield an estimate that is zero even if the true value is greater than zero. To deal with the pile-up problem, we use a heteroskedasticity-robust version of Stock and Watson (1998) median-unbiased estimate that is described by Boivin (2006).

First, we rewrite the time-varying policy parameters expressed in equation (17) as:

$$\Delta\Phi_t = \eta_t = \tau\nu_t \tag{18}$$

where η_t and ν_t are serially and mutually uncorrelated zero mean random disturbance terms and τ is a scale parameter. To focus on the estimation of τ when it is close to zero, Stock and Watson (1998) consider the following parameterization:

$$\tau = \frac{\lambda}{T} \tag{19}$$

where T is the sample size and λ can be inferred by inverting the heteroskedasticity-robust version of the Quandt Likelihood Ratio (QLR_T) test by dropping the first and last 15% of observations from the sample and using the tables provided by Stock and Watson (1998). Once λ is known, estimates of the variance of the parameters, that is $var(\Delta\Phi_t)$ and the time varying parameters can then be obtained following the methodology outlined in Boivin (2006).

The variance matrix of the TVP is pre-estimated using the median-unbiased estimator:

$$var(\widehat{\Delta\Phi_t}) = \left(\frac{\hat{\lambda}}{T}\right)^2 \left(\frac{\hat{Z}'\hat{Z}}{T}\right)^{-1} \Omega \left(\frac{\hat{Z}'\hat{Z}}{T}\right)^{-1} \tag{20}$$

where the heteroskedasticity consistent estimate of Ω is obtained using the White estimator of $E(Z_t\epsilon_t\epsilon_t'Z_t')$, based on the OLS residuals of the fixed coefficient regression, that is equation (16). The time series for Φ_t is then obtained using a standard MLE approach and using the Kalman smoother conditional on the median unbiased estimates of the variance of Φ_t . To allow for possible changes in the variance of the residuals, we estimate the above separately over different regimes using the OLS residuals.

5 Data Description

The time-series data on the unemployment rate, real GDP growth rate and mortality runs from 1962Q1 to 2006Q3. We use two popular measures of the business cycles in the paper: the deviations

of real GDP and the unemployment rate from trend⁷. Since we are interested in the behaviour of these time-series at business cycles frequencies, that is movements roughly between 6 and 32 quarters, we detrend our data and focus only on business cycles frequencies.

The mortality data is from the NBER's web site.⁸ These data are from the Multiple Cause-of-Death Mortality Data from the National Vital Statistics System of the National Center for Health Statistics and cover the period going from 1959 to 2006. Each death across the United States is accounted for and detailed information, such as the age of the individual, the state they lived in (available for most years), the cause of death, their race, education, occupation, etc. are available.⁹

We counted how many deaths occurred per quarter and used these numbers and data from the US Census Bureau on the population per quarter to calculate death rates for different age-groups and for two specific causes of deaths: cardiovascular and circulatory problems and accidents (which include violent deaths such as murders). The data from the Census Bureau were yearly population counts taken on July 1st. To convert the data to quarterly frequency, we use a liner extrapolation and the quarterly population data is then used to calculate the death rates.

To calculate the deviations of all the variables from their trend, we employ two methods to detrend the data. We use the popular Hodrick-Prescott (H-P) filter as well as the Baxter-King filter (B-K) to detrend our data.¹⁰ The different mortality rates by age and causes of deaths are shown in Figures 1-3. It is clear from Figures 1-3 that the mortality rates for all age groups have experienced a substantial decline since the 1970s. Advances in technology, medicine and the availability of better nutrition have all contributed to reduce the mean and variance of the mortality rate over time.

Figures 4-6 respectively plot the detrended unemployment rate against the detrended total mortality rate, accidental deaths rate and deaths due to cardiovascular disease. The graphs have been normalized so that the scales matched. As Ruhm (2000) points out in his paper, the procyclicality of mortality is very striking in these graphs. Figure 4 shows that there is a strong negative relationship between mortality and total deaths. This relationship seems to have however weakened in the last part of the sample, especially for the age group 25-54. We can see from Figure 4 that the relationship between detrended unemployment and total mortality turns positive at the end of the

⁷The data for unemployment and real GDP growth are from the Federal Reserve Bank of St.Louis FRED website. We collected monthly data on the U.S. unemployment rate and transformed them into quarterly data (we averaged the monthly unemployment rate over the 3 months of each quarter, weighing each month's unemployment rate equally)

⁸<http://www.nber.org/data/vital-statistics-mortality-data-multiple-cause-of-death.html>

⁹Except for 1972, for which only a half-sample was collected. We have multiplied the numbers for that year by two for our estimations

¹⁰We use the HP filter in our baseline estimates and the BK filtered estimates as robustness check.

sample for this age group. We get as similar result for deaths due to accidents. Mortality due to accidents is clearly procyclical but in this case also, the relationship with detrended unemployment seems to have changed for certain age groups (25-54) at the end of the sample. Deaths due to heart attacks and cardiovascular disease also display a strong procyclical pattern as shown in Figure 6. In this case also, the relationship between this cause of death and unemployment seem to have changed for certain age groups at the end of the sample.

6 Empirical Results

6.1 Evidence of structural breaks

We first provide evidence whether the relationship between total deaths as well as age-specific and cause-specific mortality and different measures of the business cycle has changed over time. We use a heteroskedasticity robust version of the Quandt Likelihood Ratio (QLR_T) test with 15% trimming as in Boivin (2006) to detect whether there has been a break or multiple breaks in the regression coefficients.¹¹

We apply the test for the model with and without a lagged dependent variable. Results for the QLR_T tests for age-specific mortality due to all causes and the two specific causes of deaths (cardiovascular and circulatory problems and accidents) are shown respectively in Table 1-3. We only report the results for the model without a lagged dependent variable and using the two indicators of business cycles. The first column of each table lists the coefficients that we are testing and the breakdown by age, column two uses the unemployment rate as the regressor and respectively shows the p -value for the QLR_T statistic testing the stability of all parameters and when the break occurs and column three is similar to column two but uses the deviations of real GDP from trend as the regressor. In each case, the date at which the break occurs is determined by taking the maximum value of the F statistic, that is the date at which the $sup - F$ test is at its maximum. In the baseline model, we allow for only one break.¹²

The results of the QLR_T statistic testing the stability of all parameters are shown in Figures 7-9. We find evidence of structural breaks in an overwhelming majority of cases when we employ

¹¹The null is that the intercept and the dependent variables have remained constant over time and this is tested against the alternative that there is a break in all or some of the coefficients at a given unknown date. The trimming percentage that we use and which is common in the literature implies that the test applies only to 70% of the data.

¹²We have also estimated the model with more than two breaks if the latter was justified. The results are very similar to cases where we only allow for one structural break.

data on age-specific total deaths, as well as cause-specific deaths. The results are robust whether or not we use unemployment or real GDP as indicators of the business cycles. The joint stability test on all coefficients implies a median unbiased estimate of λ that varies between 4 and 10 depending on the model used. This indicates that the period-to-period variation in the parameters of the model is small but statistically significant (see Stock and Watson, 1999). In almost all cases, we find strong evidence in favour of the time-varying model that allows for heteroskedastic variance.

6.2 Results from TVP model

The methodology we discussed in section 4 is used to estimate the impact of business cycles on total mortality as well as mortality due to cardiovascular disease and accidents. The results are presented in Figures 10-15. Figure 10 shows the relationship between unemployment and total mortality. The time-varying point estimates are shown by the solid line whereas the dotted lines indicate the 95% confidence interval. The point estimates of the response of total mortality to changes in the unemployment rate indicate that mortality is pro-cyclical, that is mortality goes up in good times. This result is robust for all age categories and for the entire sample. Our point estimates vary from -0.01 to -0.06 depending on the age category and the time-period. In an overwhelming majority of cases and for most of the entire sample, our point estimates are significant at the 5% level. Our point estimates are very similar to Tapia Granados (2005) who also finds point estimates in that range for the U.S using a constant parameter model. If we assume that the trend unemployment rate is around 6%, then according to our point estimates, a one percentage decrease in the unemployment rate (relative to trend) would decrease the total death rate by approximately 0.17% to 1% depending on the age-group and the time period. Ruhm (2000) found that a 1% increase in the unemployment rate would decrease the overall mortality rate by around 0.54%. Our estimates are thus close to what Ruhm (2000) obtains using panel data. The mechanisms that generate this procyclical result thus imply that the costs on health that we outlined in the theoretical model (less time to invest in health production, faster depreciation of the health stock and lower health stock in the production process) outweigh any potential benefits (higher medical expenditure) that a good economy conveys.

Figure 10 also shows that our estimates for total mortality are generally bigger for younger adults (ages 25-44) compared to older adults who are still in the labour force (45-64) or compared to younger adults (less than 25). However, our estimates for individuals that do not participate normally in the labour force, that is those who are over 65, tend to be as large as those in the age group 25-44. This indicates that the pro-cyclical nature of mortality may not be entirely driven by

individual's own behaviour over the business cycles, such as working harder, being more stressed and exercising less during an economic expansion but also by external factors that are unrelated to behaviours, such as more elevated levels of pollution, more congested emergency rooms that leads to a lower quality of care. Our results thus support the recent findings of Miller and al. (2009). They argue that the main driving force behind an increase in mortality during an economic boom may not be completely related to less healthy lifestyles or elevated stress level but more to factors that are indirectly linked to the business cycles, such as pollution and disruptions in social support and social networks.

Figure 10 also reveals that for adults aged between 15-64, the relationship between unemployment and mortality has strengthened since the middle of the 1980s. This time variation in U.S. mortality can be linked to the shift and changes that occurred in the U.S. economy during that period. While there were important gains in productivity in the U.S. in the last part of the 1980s and the 1990s, there was also at the same time an increase in the number of average hours worked. Cociuba and Ueberfeldt (2010) argue that between 1980s and 2007, average hours worked in the U.S. increased by 13%. If this is the case, then the channels through which the business cycles affect mortality that we describe in our model may have become stronger in the 1990s, thus making this relationship even more compelling.

We obtain similar results when we employ the deviations of real GDP from its trend as a measure of business cycles. Figure 11 describes the time-varying relationship between mortality and GDP. Mortality is pro-cyclical except for the age category 35-44. This result is similar to Ruhm (2000), Tapia-Granados (2005) and Miller and al. (2009) who also find that mortality for this age-group is counter-cyclical.

Figure 12-13 show respectively the relationship between unemployment and deaths due to accidents and between gdp and deaths due to accidents. Mortality due to accidents is pro-cyclical. However, there are important and significant time-variation in the relationship between unemployment and deaths due to accidents. Our point estimates are bigger compared to total mortality. This result is not surprising as motor vehicle and on the job fatalities and on the go up dramatically when the economy does well. Accidental deaths even for those outside the labour force are significant and very procyclical. Our results also indicate that the relationship between deaths due to accidents and the unemployment rate may have weakened over the 1990s. One possible explanation is that the fundamental change in the composition of the U.S. economy in the 1990s with a move towards high-tech industries and the relocation of manufacturing industries overseas may have reduced the number of on-the-job accidents since the bulk of the additional jobs created

over that period were mostly concentrated in high-tech firms where the risk of getting injured or killed is much lower. This results warrant further investigation.

Finally, figures 14-15 show respectively the relationship between unemployment and deaths due cardiovascular deaths by age and between gdp and deaths due cardiovascular deaths by age. WE obtain similar results in this case also. Mortality due to cardiovascular diseases is strongly procyclical. We report the results for the age group that is 45 and older as the majority of the deaths occur within this age category. We find that the coefficients on unemployment and gdp display large and important time variation. Our results are similar to Ruhm (2000) who argues in his paper argue that cardiovascular deaths are very pro-cyclical. Tapia Granados (2005) also reports such a result.

7 Conclusions

Our paper shows that there is a robust link between mortality and the business cycle. We present overwhelming evidence that the strength of this relationship has changed over time. However, we find that mortality is very procyclical for the various causes of deaths we examine. Using a theoretical model, we are able to explain the mechanism through which changes in the economy affect mortality. Our model reveals that the mechanisms that lead to a deterioration of health status, such as less time to invest in health production, faster depreciation of the health stock and lower health stock in the production process outweigh any potential benefits (higher medical expenditure) that a good economy conveys. Our findings are similar to Ruhm (2000, 2003, 2005) and Tapia Granados (2005). Our results reveal that there are other factors at play that can explain why mortality accelerates during an economic boom. Individual factors play a large role in explaining this result but there are other indirect factors that lead to higher mortality rates in good times. More research should be devoted to examining these mechanisms.

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Table 1: QLR_T test with age-specific mortality rates - All causes

Parameters	Unemployment		GDP	
	p -values (sup -LM)	Break dates	p -values (sup -LM)	Break dates
$\{\alpha_{allages}, \beta_{allages}\}$	0.009	1993q1	0.064	1971q3
$\{\alpha_{15-64}, \beta_{15-64}\}$	0.076	1996q3	0.048	1996q3
$\{\alpha_{15-24}, \beta_{15-24}\}$	0.023	1997q1	0.054	1996q4
$\{\alpha_{25-34}, \beta_{25-34}\}$	0.162	1996q4	0.041	1996q4
$\{\alpha_{35-44}, \beta_{35-44}\}$	0.001	1996q4	0.002	1996q1
$\{\alpha_{45-54}, \beta_{45-54}\}$	0.003	1995q4	0.000	1974q2
$\{\alpha_{55-64}, \beta_{55-64}\}$	0.053	1973q4	0.066	1997q2
$\{\alpha_{65-74}, \beta_{65-74}\}$	0.000	1973q4	0.001	1973q2
$\{\alpha_{75-84}, \beta_{75-84}\}$	0.004	1973q4	0.002	1974q1
$\{\alpha_{85p}, \beta_{85p}\}$	0.000	1993q2	0.004	1971q2

Table 2: QLR_T test with age-specific mortality rates - accidents

Parameters	Unemployment		GDP	
	p -values (sup -LM)	Break dates	p -values (sup -LM)	Break dates
$\{\alpha_{15-64}, \beta_{15-64}\}$	0.002	1993q1	0.026	1994q2
$\{\alpha_{15-24}, \beta_{15-24}\}$	0.000	1995q2	0.014	1989q2
$\{\alpha_{25-34}, \beta_{25-34}\}$	0.151	1971q2	0.394	1970q3
$\{\alpha_{35-44}, \beta_{35-44}\}$	0.000	1993q1	0.004	1993q1
$\{\alpha_{45-54}, \beta_{45-54}\}$	0.012	1993q3	0.103	1994q3
$\{\alpha_{55-64}, \beta_{55-64}\}$	0.000	1972q3	0.022	1972q1
$\{\alpha_{65-74}, \beta_{65-74}\}$	0.005	1975q3	0.024	1971q4
$\{\alpha_{75-84}, \beta_{75-84}\}$	0.041	1996q4	0.167	1980q3
$\{\alpha_{85p}, \beta_{85p}\}$	0.003	1993q2	0.003	1974q1

Table 3: QLR_T test with age-specific mortality rates - Cardiovascular disease

Parameters	Unemployment		GDP	
	p -values ($sup-LM$)	Break dates	p -values ($sup-LM$)	Break dates
$\{\alpha_{15-64}, \beta_{15-64}\}$	0.149	1968q2	0.141	1968q2
$\{\alpha_{35-44}, \beta_{35-44}\}$	0.011	1991q4	0.120	1991q4
$\{\alpha_{45-54}, \beta_{45-54}\}$	0.005	1993q4	0.003	1978q3
$\{\alpha_{55-64}, \beta_{55-64}\}$	0.055	1968q2	0.098	1968q1
$\{\alpha_{65-74}, \beta_{65-74}\}$	0.080	1968q2, 1974q3	0.068	1968q2
$\{\alpha_{75-84}, \beta_{75-84}\}$	0.006	1993q1	0.082	1968q2
$\{\alpha_{85p}, \beta_{85p}\}$	0.000	1993q1	0.022	1993q1

Figure 1: Log of mortality rate - All causes

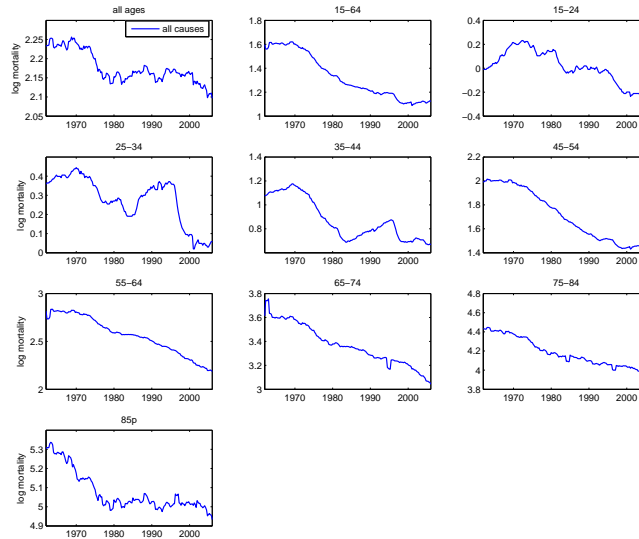


Figure 2: Log of mortality rate - Accidents

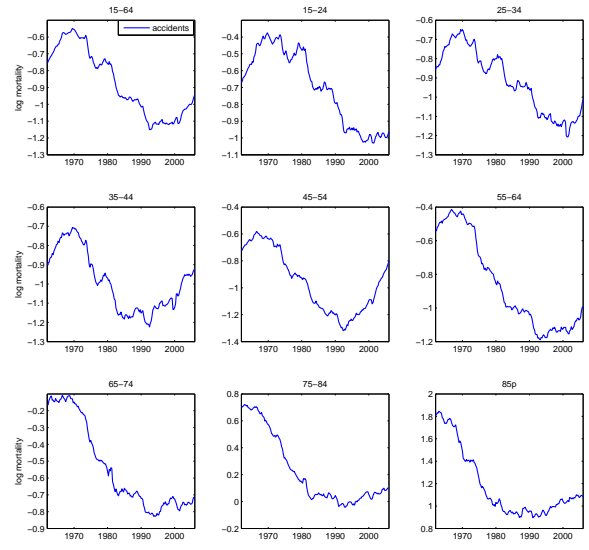


Figure 3: Log of mortality rate - Cardiovascular disease

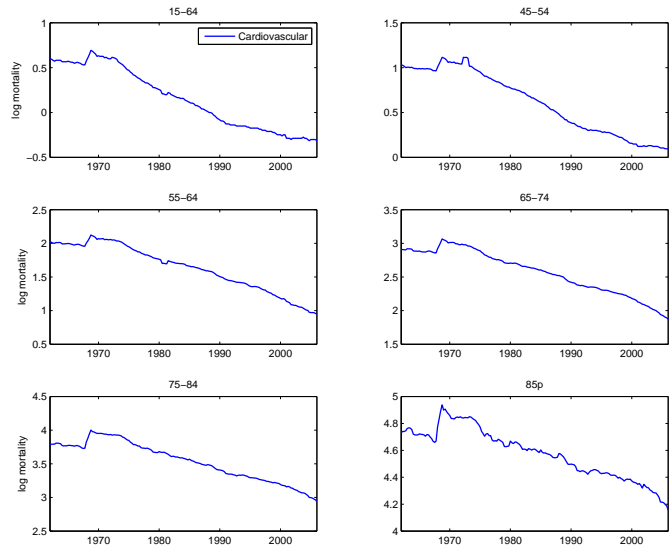


Figure 4: Correlation of unemployment and all causes of deaths

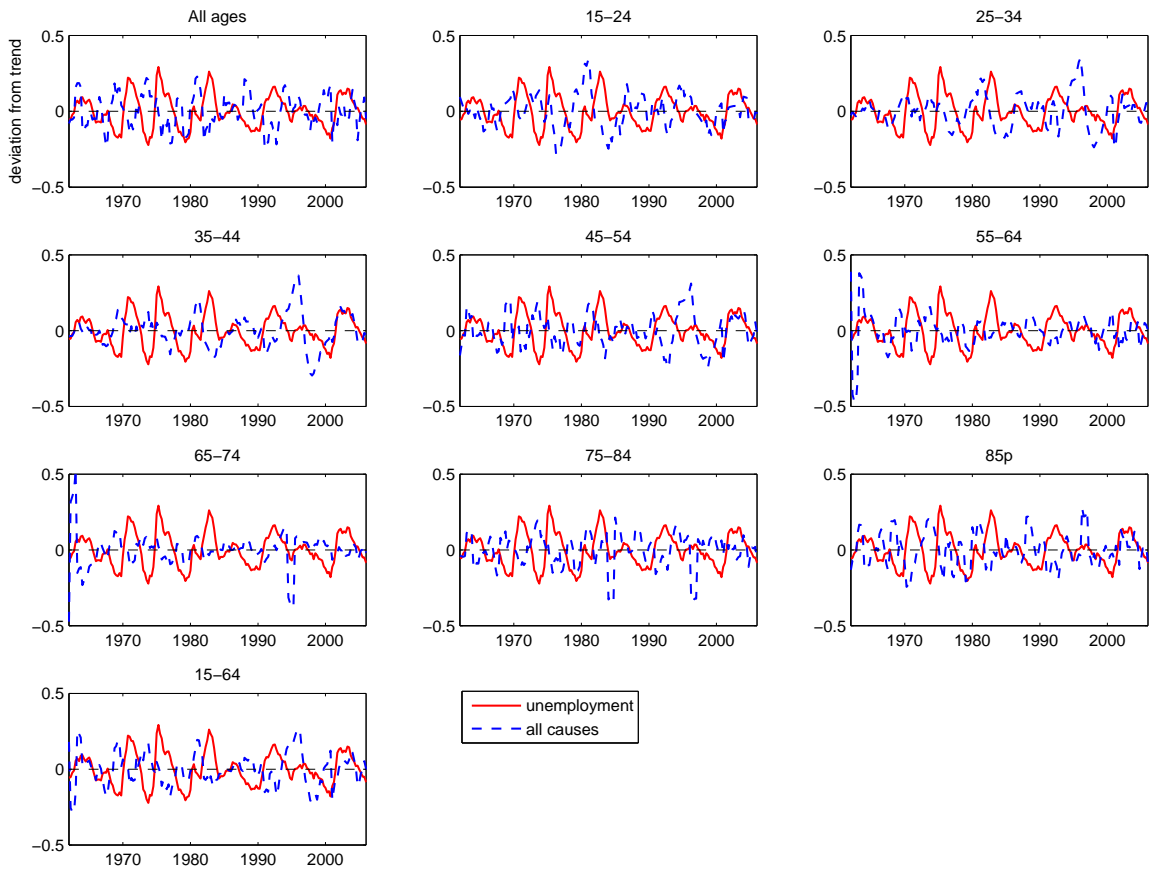


Figure 5: Correlation of unemployment and accidents

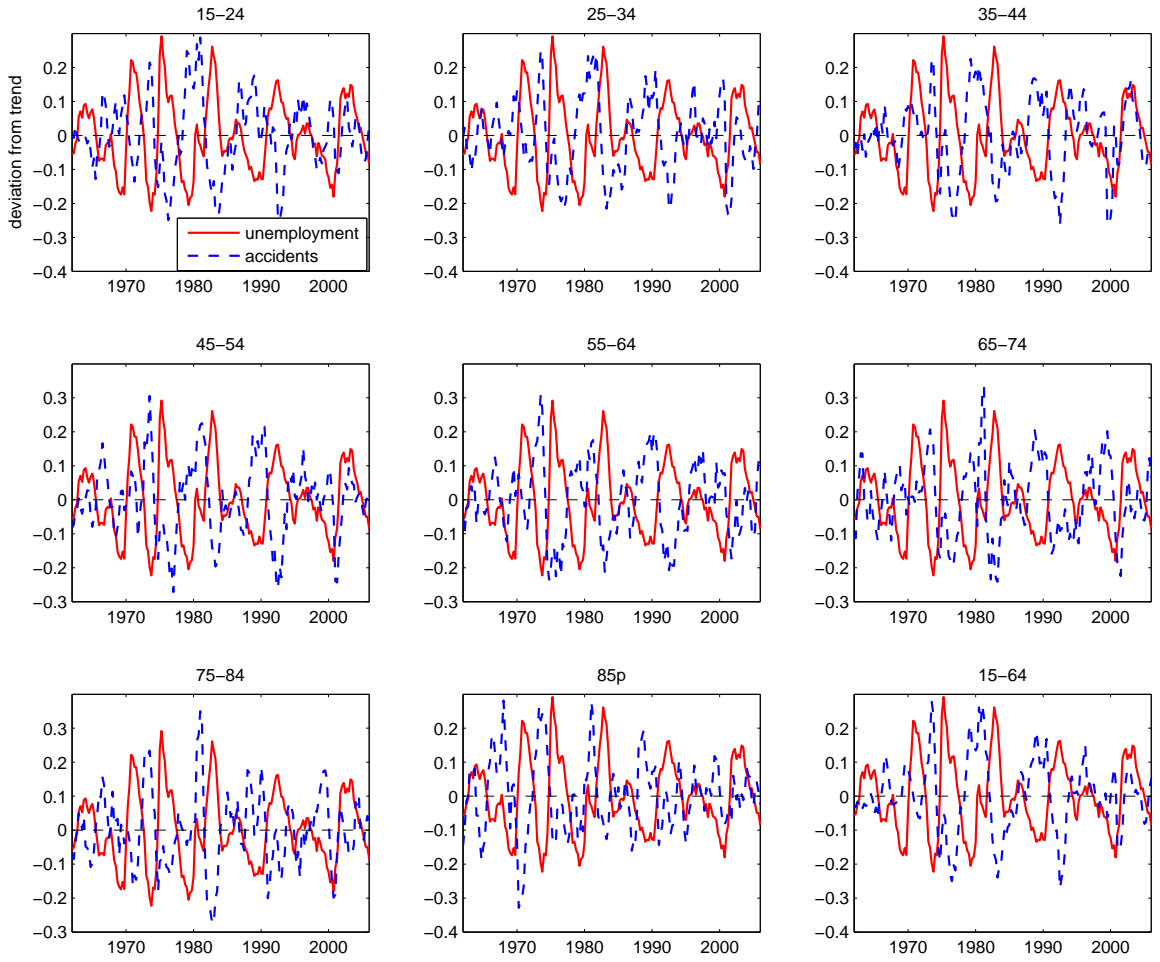


Figure 6: Correlation of unemployment and cardiovascular deaths

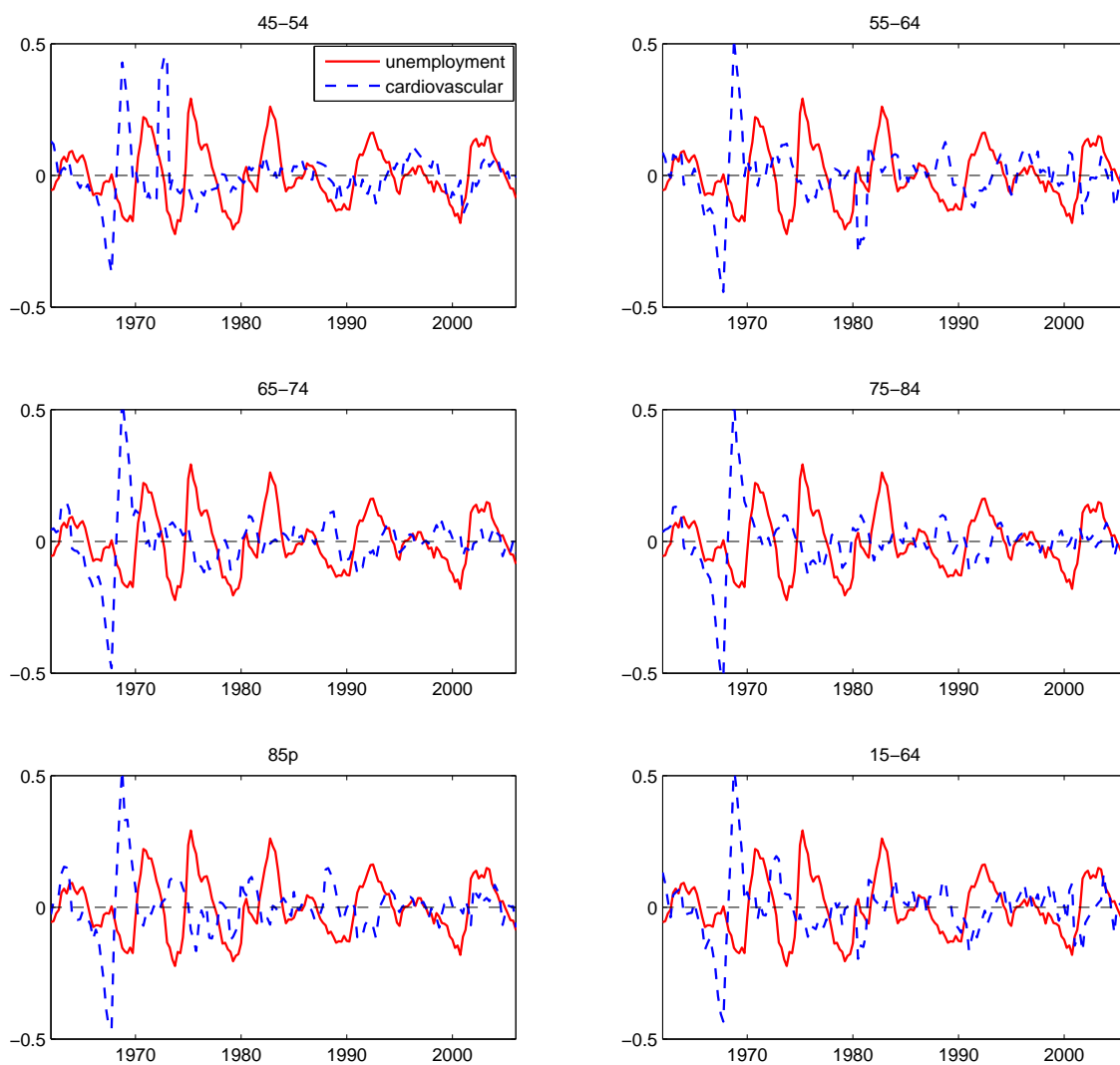


Figure 7: Total deaths by age - QLR_T test

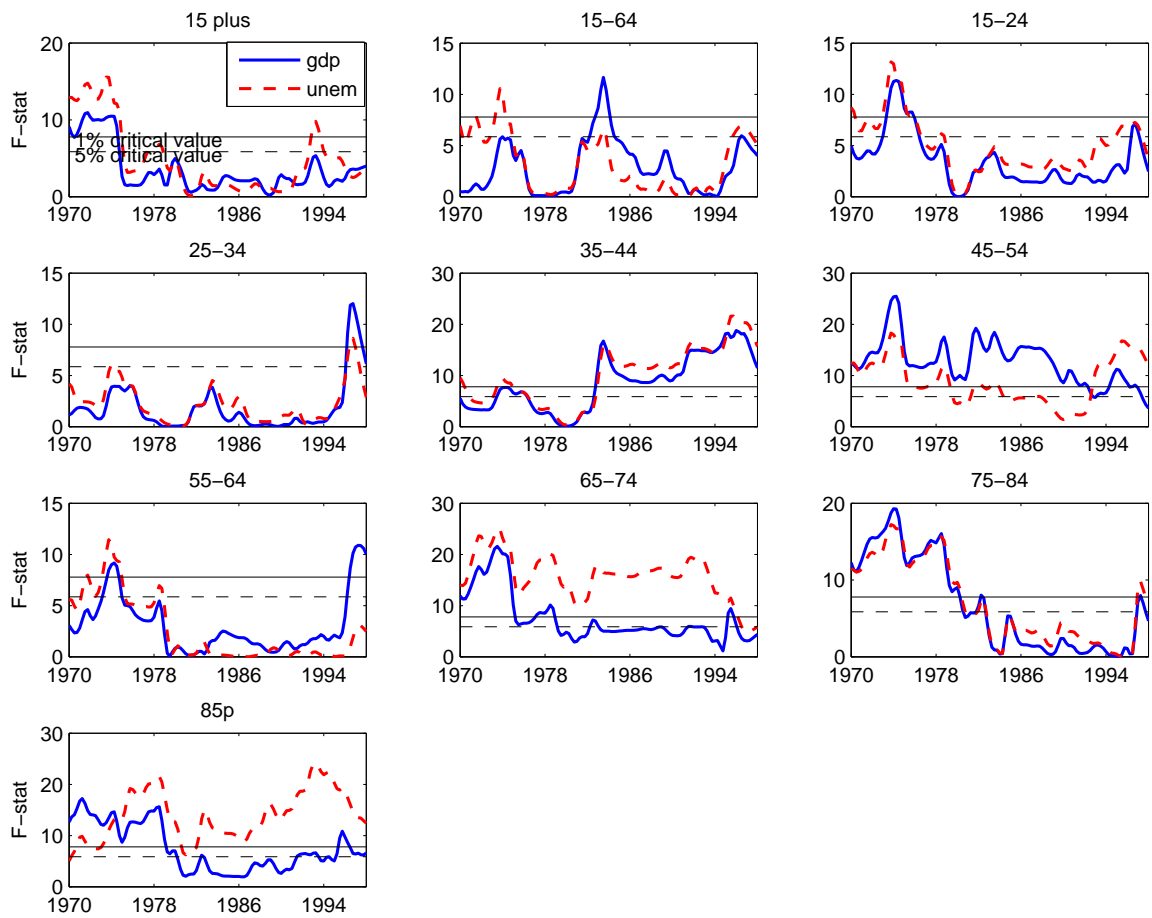


Figure 8: Deaths by accidents by age - QLR_T test

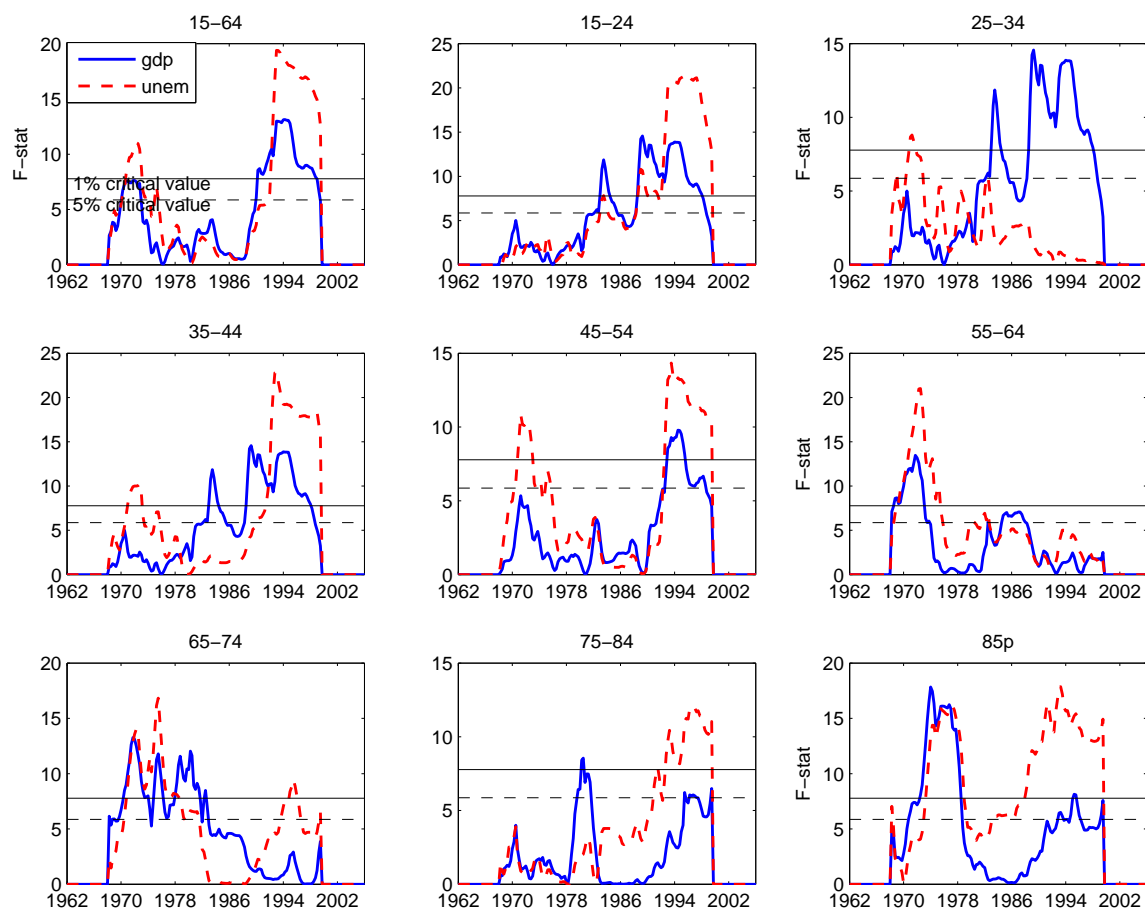


Figure 9: Cardiovascular deaths by age - QLR_T test

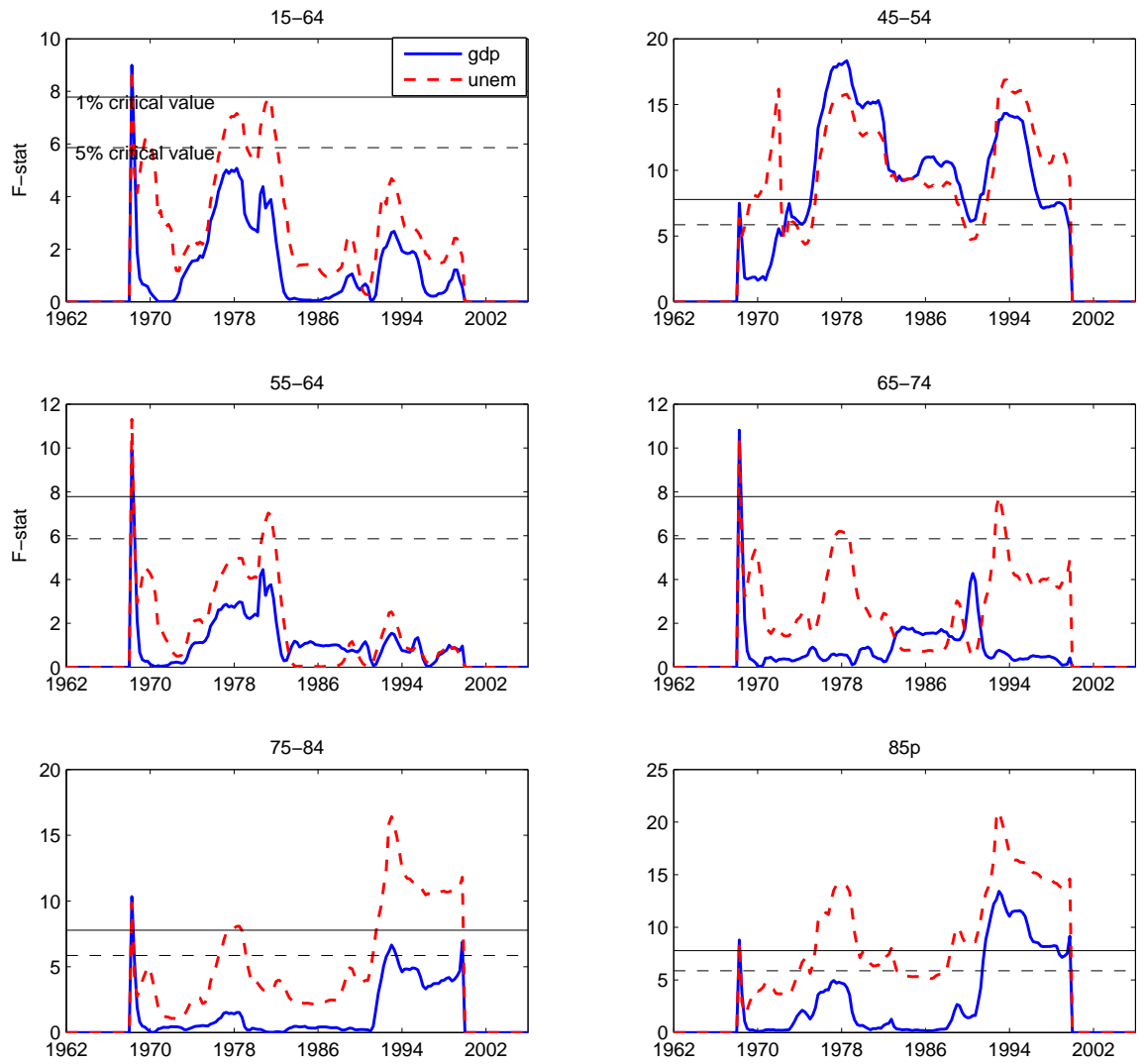


Figure 10: TVP estimates - All causes of deaths by age with unemployment

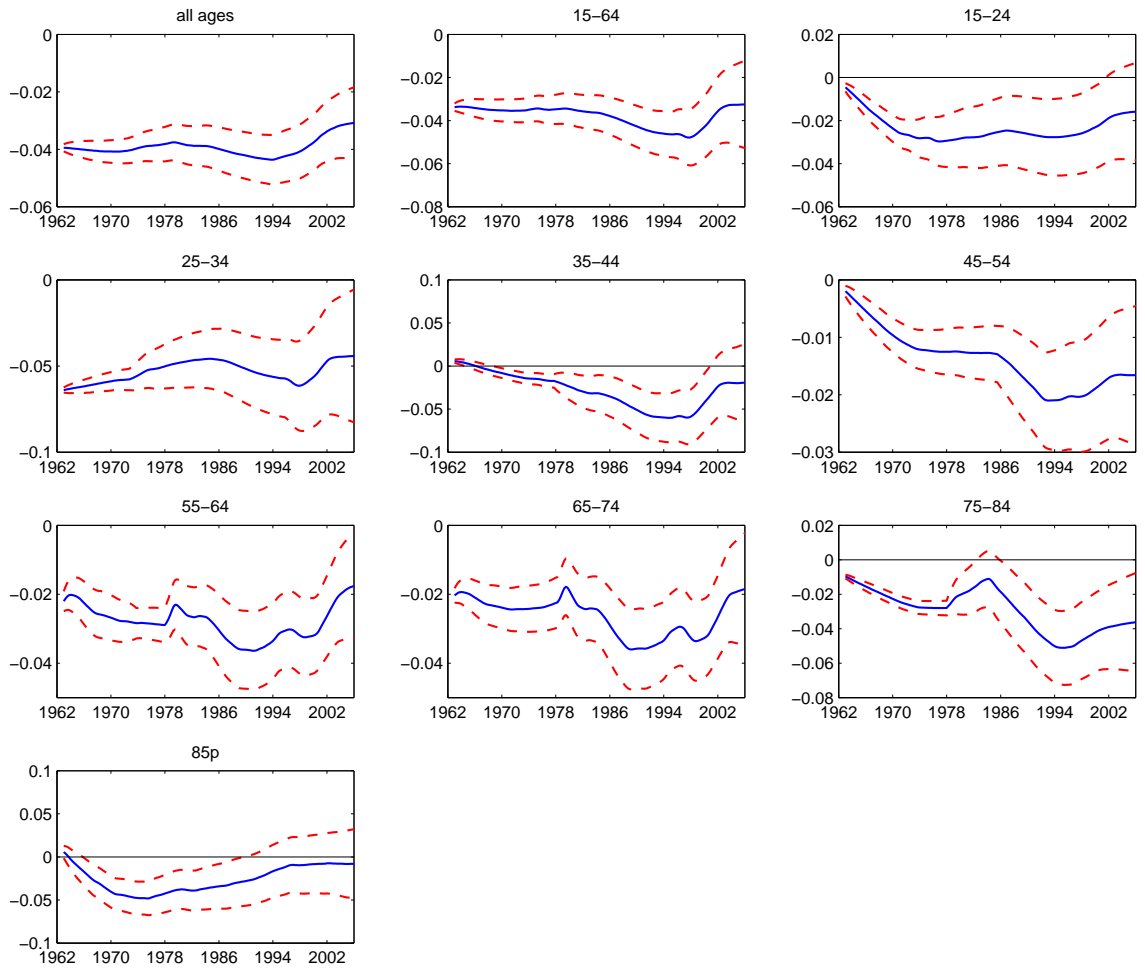


Figure 11: TVP estimates - All causes of deaths by age with real GDP

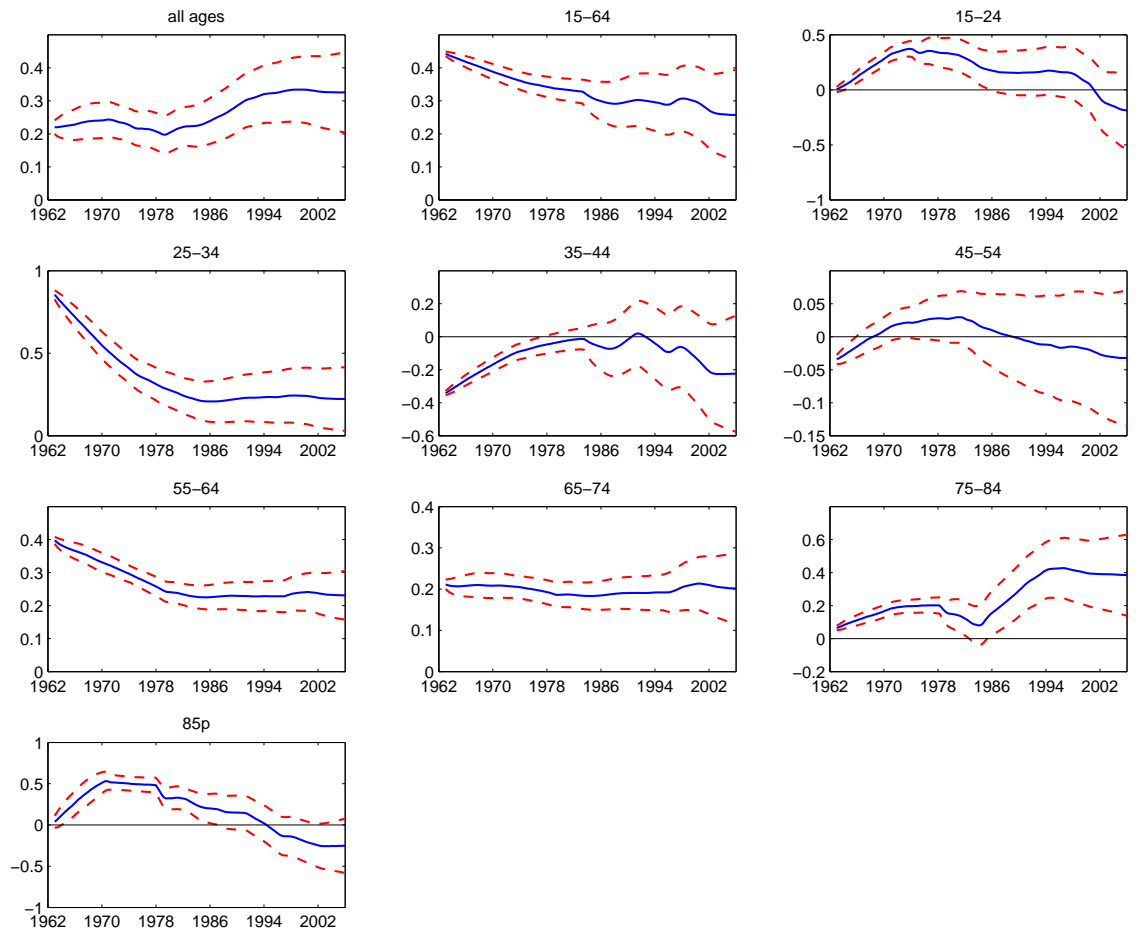


Figure 12: TVP estimates - Accidental deaths by age with unemployment

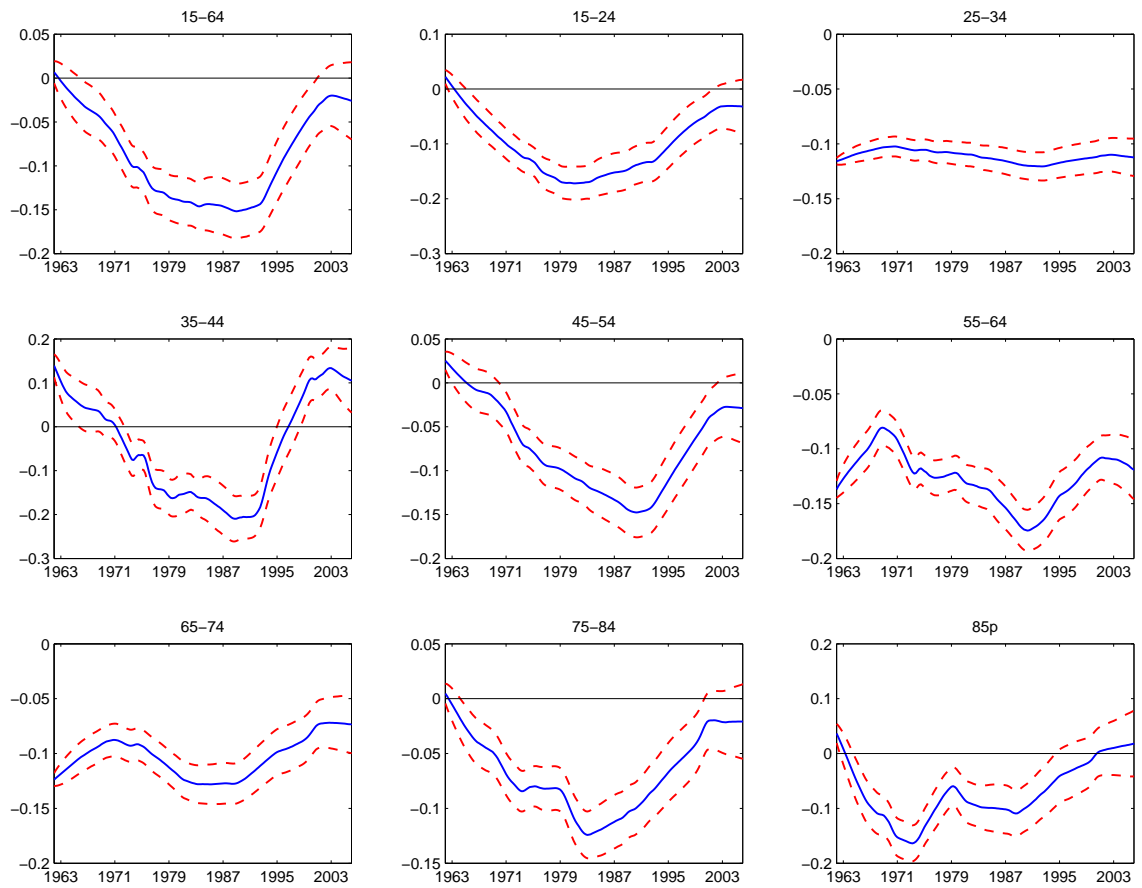


Figure 13: TVP estimates - Accidental deaths by age with GDP

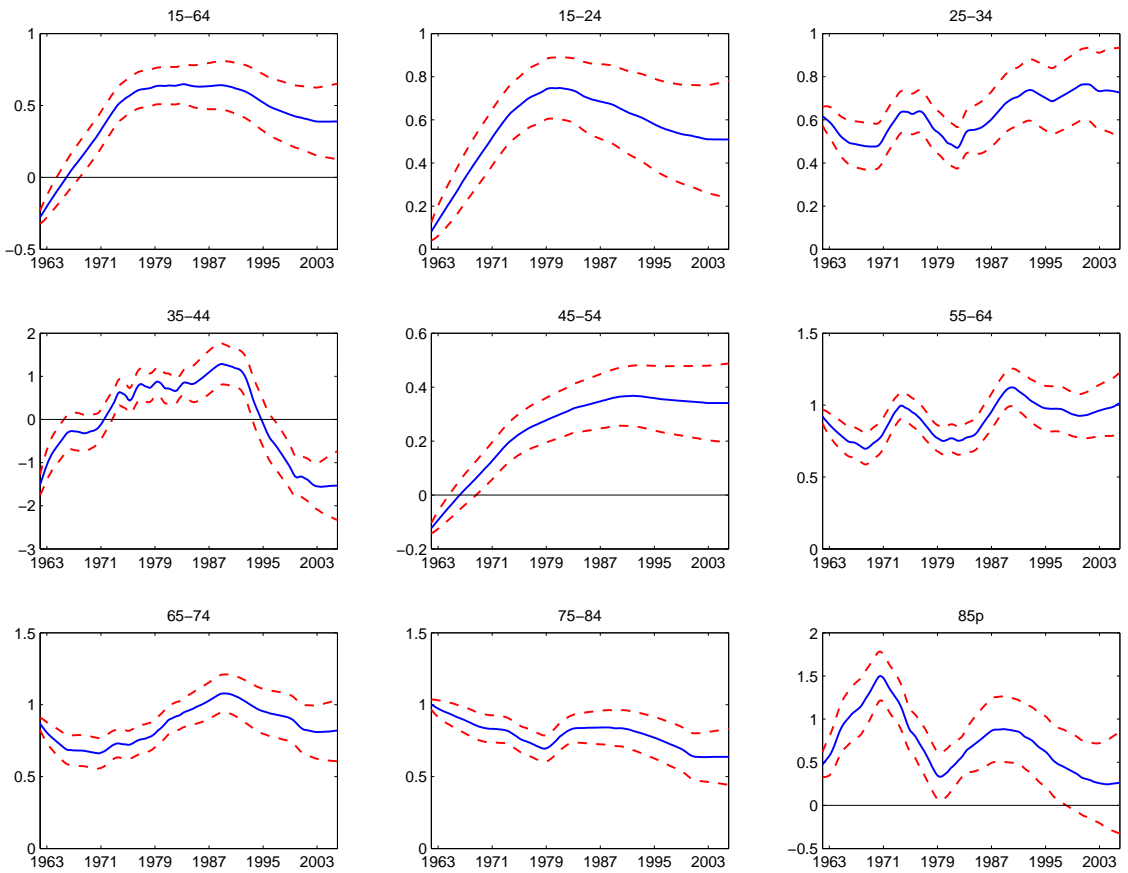


Figure 14: TVP estimates - Mortality due to cardiovascular diseases by age with unemployment

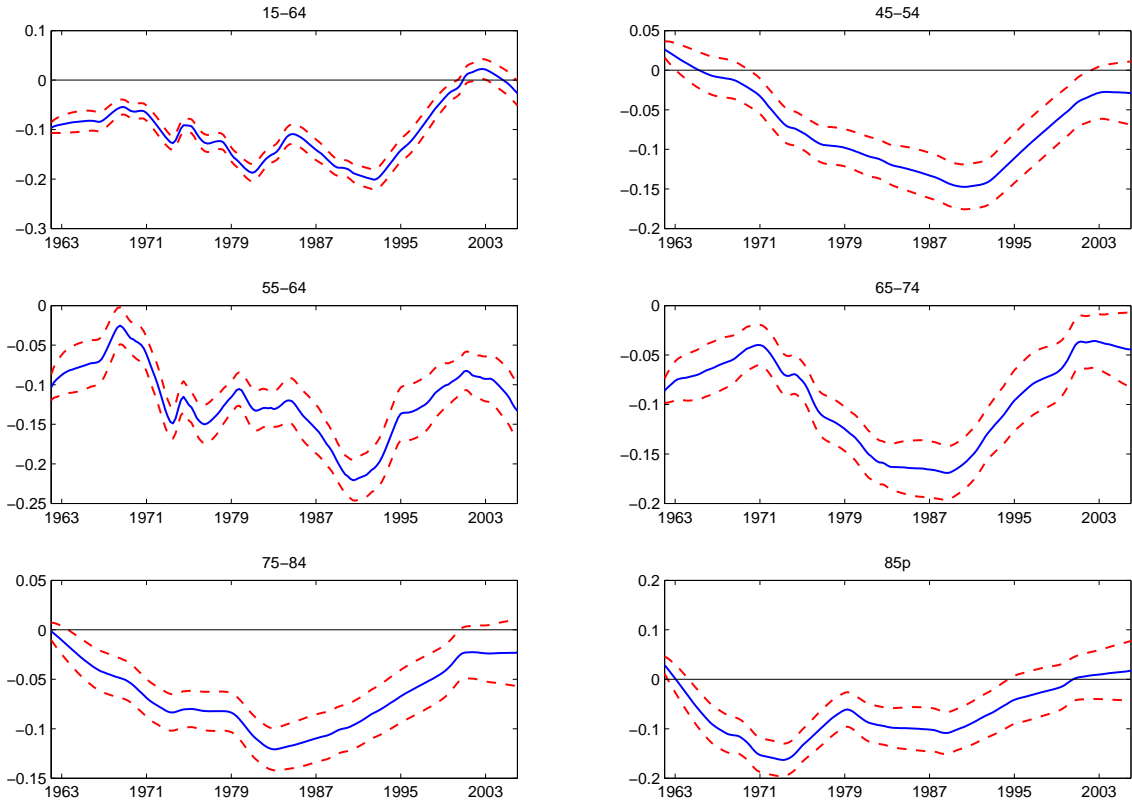


Figure 15: TVP estimates - Mortality due to cardiovascular diseases by age with GDP

