

Death and its determinants

Declan French and Colin O'Hare, Queens University Management School¹

Abstract

Existing stochastic models of mortality are able to describe the dynamics of mortality but fail to explain and identify what has influenced historical trends and whether or not these trends will continue into the future. In this paper, we examine recent developments highlighted by the epidemiological literature and assess the evidence for their contribution to mortality decline across a number of OECD countries. We consider whether the space spanned by the latent factor structure in mortality data can be adequately described by developments in GDP per capita, public and private health expenditure and lifestyle-related risk factors using statistical techniques recently developed in macroeconomics and finance. We then judge the forecastability of historical trends and construct an epidemiologically-informed model for future mortality using the Bayesian hierarchical modelling approach of King and Soneji. Using suitable priors and the causal factors identified we compare the forecasting performance of this model against the benchmark atheoretical mortality model in the field, the Lee Carter model. We address the actuarial and capital market implications by demonstrating the effect on pricing of annuity business when taking causal factors into consideration.

1. Introduction

Mortality risk has been identified as one of the most significant risks that our society will face in the next number of years. Pension providers and insurance firms are directly exposed to this risk. However, many other aspects of modern life will also be affected by the substantial and sustained improvements in mortality experience that we are observing throughout the world. These improvements and the generally accepted significance of longevity risk have led to a large number of researchers dedicating time to the development of mortality models and to the forecasting of mortality rates.

Further, the tightening of regulation for insurers and pension providers has meant that it is now essential that mortality rates are modelled more accurately. In particular being able to forecast mortality rates with some level of confidence has become a matter of extreme importance. Accurate

Date: Latest version: August 19th 2011.

¹ Queen's University Management School, Riddel Hall, 185 Stranmillis Road, Belfast, Northern Ireland, UK
BT9 5EE, +44 (0) 28 9097 4671 Email: c.ohare@qub.ac.uk .

modeling and forecasting will enable advisors and policy decision makers to assess the financial implications of ageing populations more confidently.

Mortality rates show a stochastic characteristic and as a result there has been a significant amount of literature on modeling of mortality rates using stochastic methods. This approach avoids the need to explain why mortality improvements have occurred and focuses instead on allowing the data to drive the modelling process. The Lee and Carter (1992) model was the first to identify a common trend in the logarithm of age-specific mortality rates and modeled US male data using a one factor time series approach. Following this, many innovations of the Lee-Carter model have been developed including, Brounhs et al. (2002), Girosi and King (2005), Renshaw and Haberman (2003, 2006), Cairns et al. (2006), Currie et al. (2004), Currie (2006), Hari et al. (2008), and Plat (2009). Each of these innovations focuses on one or more perceived weaknesses of the Lee Carter model and improves the fitting quality as a result. For comparative purposes we use Lee Carter (1992) as the benchmark atheoretical model of mortality. This model effectively uses a principal components approach to modelling U.S. mortality identifying a single common trend in the data. All the methods outlined above are examples of the extrapolative approach to modelling mortality. This approach, which makes use of the regularities found the age and time profile of mortality data has been the most successful method to date (Booth and Tickle (2008)) but it fails to explain the drivers of mortality improvements. A second approach to modelling mortality is the explanatory approach which makes use of measured, exogenous variables to try to explain the trends seen in the mortality data and finally, there is an expectations approach making use of subjective, expert opinion on mortality data. Booth and Tickle (2008) outline the developments in the three approaches and note that the extrapolation approach has received the most attention from researchers. It relies on the assumption that trends seen in the past data will be continued into the future. Booth and Tickle (2008) accept that this is a reasonable approach but note that there is a place for explanatory methods to be applied to pick up aspects of mortality that are not present in past data. Looking at the explanatory approach, Booth and Tickle (2008) note that this field is yet to be fully developed, with a very limited range of models having been implemented. One notable model that they comment on is that of Girosi and King (2008) who extend the use of structural models in mortality forecasting to developing countries using Bayesian methods to smooth over time, age and country. This model has since been extended further by King and Soneji (2011) to incorporate lagged exogenous variables in a bayesian hierarchical model of mortality rates.

In many of the extrapolative models the innovation has been to add factors to explain newly identified aspects of the mortality profile or to improve the flexibility of the model to fit wider age range. Examples of these innovations include modeling the cohort effect, as in Renshaw & Haberman (2003, 2006), adding a second period effect, as in the Cairns et al (2006), widening of the model to fit ages 20-89, Plat (2009). Adding new factors in this way may improve the fit of the model but it also adds more complexity to the structure of the model which may or may not continue into the future.

Whilst these approaches are valuable in determining the direction of future mortality rates they give little insight into the reasons for secular trends. We should expect that certain aspects of lifestyle medical technology, healthcare provision and living conditions will contribute to the changes we see in

mortality rates but yet there is still a limited literature that incorporates these elements into a forecasting model.

In this paper, we attempt to identify appropriate exogenous determinants statistically and incorporate them into existing stochastic mortality models. The hope is that mortality forecasts conditional on forecasts of identified exogenous determinants will be more informative than the existing atheoretical approach. To demonstrate the superior fitting and forecasting quality of a model which incorporates forecast exogenous variables we compare our results with the benchmark Lee Carter (1992) model and with the Girosi and King (2008) model. To incorporate forecast exogenous variables we apply the King and Soneji (2011) approach as this method gives excellent results and already has the functionality to add exogenous variables.

There are many possible explanations for recent changes in mortality rates. The second half of the twentieth century saw a huge increase in life expectancies in developed countries with very significant reductions in older age mortality (Leon, 2011). Trends for male mortality in Western Europe, US and Japan have followed broadly similar trends. Cardiovascular disease has declined simultaneously at all ages in each country due to a reduction in risk behaviours and improvements in treatment (Kuulasmaa et al. 2000; Unal et al., 2004). Other determinants include the social, political and economic contexts within which people live although causal mechanisms are still not well understood. Forecasts for future mortality are also difficult to make as, for example, the detrimental effect of trends in obesity may overwhelm reductions in mortality due to lower smoking rates and technological progress may continue or slow down.

The health production function approach where health is proxied by mortality provides a framework for understanding the determinants of mortality. The model below is a modification of the seminal health production model used in a cross-sectional analysis of US states by Auster et al. (1969):

$$\ln M_i = c_i + \alpha Z_i + \beta X_i + \gamma HC_i + \delta E_i + u_i$$

where M_i are (standardised) mortality rates by state, Z_i socio-economic status (income, education), X_i lifestyle inputs (alcohol, tobacco), HC_i are healthcare inputs (drugs, doctors, hospital capital stock), E_i captures environmental variables (urbanization, industrialization) and u_i is a random element. In a revision to this work, Thornton et al. (2002) expanded lifestyle factors to include marital status, expanded environmental factors to include crime and added race, gender to the above model. The surprising consistent result is that health care expenditure seems to have little effect on variations on mortality. However, there is evidence that health care expenditure does become important in explaining variations in older adult mortality. Miller and Frech (2000) in a study of OECD countries found pharmaceutical expenditure to have little effect on life expectancy at birth but significantly positively related to remaining life expectancy at middle and older ages.

At any point in time all countries are using similar health technologies. In studying trends in mortality over time we must acknowledge the role of advances in medical technology. Cutler and Meara (2004) estimated that the decline in US mortality in the second half of the twentieth century was mainly due to a decline in infant mortality and older-age mortality. The former they attributed to advances in neonatal

care (ventilators, drugs, lab testing)² and the latter they attributed largely to medical advance in cardiovascular disease treatment (new drugs, new surgical procedures and specialised equipment) and to a lesser extent to a reduction in smoking rates. In the absence of suitable medical technology variables, secular increases in health care expenditure will capture technological improvement over time to a degree.

Taking into account increased consumption of goods injurious to health such as alcohol and tobacco it would be presumed that higher income would imply better health.³ Higher incomes allow people to spend more on both health inputs such as better doctors and hospitals in systems where healthcare coverage is not universal or where part payment is required (US and Japan) . But even in universal health care systems (e.g. National Health System in the UK) socio-economic inequalities persist as the wealthy can purchase more non-health care inputs that benefit health such as housing and nutritious food and they are less stressed by their economic circumstances (Wilkinson and Marmot, 2003). The choices that individuals make in relation to their health are also put them at the risk of disease. Cutler et al (2009) highlight smoking, obesity (due to the availability of cheap food) and drinking as the three most common risk factors in their US study. Thornton et al. (2002) found tobacco consumption increased mortality. Miller and Frech (2000) found alcohol consumption explained life expectancy at birth, middle and older ages. Leon (2011) attribute the stagnation in female life expectancy in Denmark, Netherlands and USA principally to the smoking epidemic there.

The role of other possible determinants has been posited. Economic instability has been found to increase the risk of premature death from heart disease in epidemiological studies (Bethune, 1997; Iverson et al, 1998) although Zweifel et al. (2009) conclude that the statistical evidence supporting such a relationship is weak in aggregate studies. Environmental air pollution increases deaths due to respiratory diseases which are concentrated in the elderly (Schwarz and Dockery, 1992) but suitably long time series data of air quality are difficult to find. Education levels although very important to health are not explored in this time analysis study as they have not varied substantially over the period considered.

The work presented in this paper can be divided into three parts. In the first part we use a principal components approach to identify the factor structure of the mortality data for the U.S. U.K. and Japan. The second stage takes the latent factors, and tries to explain these factors by observed, exogenous factors (GDP, health expenditure, smoking levels, alcohol consumption etc) using appropriate statistical techniques and using stopping rules to prevent the model become over-parameterised. Finally, having identified the most appropriate exogenous determinants we forecast the exogenous variables using ARIMA techniques and build the forecasted exogenous variables into a model using the King and Soneji (2011) approach to improve forecasts. Whereas King and Soneji (2011) used lagged exogenous variables to explain mortality, and so avoid the need to forecast these variables, we will forecast the exogenous variables separately to produce a model capable of allowing for forecast exogenous variables. We calculate the results for the Girosi and King (2008) approach with no exogenous variables as well as results where our forecast exogenous variables are included ie a variant of the King and Soneji (2011)

² Dealt with more fully in Cutler and Meara (2000)

³ Ruhm (2004) takes a contrarian position arguing that there are less motor vehicle accidents and people adopt healthier lifestyles in economic downturns.

approach. The main difference between standard multiple linear regression and Girosi and King (2008) model is how the regression coefficients are estimated. The model is framed in a Bayesian hierarchical structure with regression coefficients being drawn from posterior distributions.

The remainder of this paper is laid out as follows. In section 2 we discuss the data that has been used in this study. The methodology is discussed in section 3. The results are presented and analysed in section 4 and section 5 concludes.

2. Data

The mortality data used is taken from the Human Mortality Database collated by the Department of Demography at the University of California, Berkeley and the Max Planck Institute for Demographic Research in Rostock, Germany. Death rates, a ratio of the death count by single age and year divided by an estimate of the exposure-to-risk in the same interval, for males in the US, UK and Japan over the period 1970-2006 were selected. Models were estimated over the period 1970-2000 and mortality rates for the remaining 6 years, 2001-2006, were retained for comparison with forecasts. These countries were chosen so as to provide a variety of results. On the one hand, being non-tropical countries with developed health care systems similar factors should determine trends in mortality. On the other hand, they are distinct in terms of culture, diet, and the importance of private versus public provision in health care which should generate distinct results. Due to the exponential nature of mortality rates we model the logarithmically transformed mortality rates.

Data on possible determinants of health were taken from OECD Health data 2009. Data availability dictated the candidate variables chosen: Alcohol consumption, Tobacco consumption, Total fat intake, Fruit and vegetable consumption, GDP per capita and Health expenditure per capita. Definitions and descriptive statistics are given in Table 1. GDP and Health expenditure have been both logged in the statistical analysis. The obesity time series are short and patchy and to some extent this information is captured by the food measures included. Data on pharmaceutical expenditure and medical technology capital stock (CT and PET scanners, MRI units, radiation therapy etc) are insufficient and are captured crudely by aggregate health expenditure. Air quality emissions data (SO_x, NO_x and CO) are inadequate.

Not all determinants of mortality are contemporaneous. Barker (1992) provided evidence that insults to foetal health had life-long consequences based on an analysis of the risk factors for cardiovascular diseases found in adults who were born at the time of the WW2 Dutch famine. The short time series data considered in our study precludes the inclusion of variables of large lag length. These effects may be relatively minor compared with cardiovascular risk factors experienced in adulthood as Cutler et al. (2006) indicate.

3. Methodology

Many of the approaches to mortality modelling used in the actuarial literature are based on a principal component analysis (PCA) of time series of mortality data by single age. The Lee-Carter model is a one – PC model and other multifactorial derivatives of this model add further cohort terms or additional factors to capture younger or older age mortality (e.g. Renshaw and Haberman, 2006; Cairns et al. 2006; Plat 2009) . Yang et al. (2010) building on previous PCA studies of mortality (Bell, 1997; Hyndman and Ullah, 2005) considers a two-PC model.

An econometric literature on factor analysis is well-developed. Factor analysis has been used extensively in economic forecasting, modelling business cycles and analysing contagion effects of economic crises. In order to put an economic interpretation on latent factors extracted in these cases, Bai and Ng (2006) developed a statistical test for large cross section (N) and large time dimension (T) datasets to test the adequacy of observed variables as proxies for the unobserved factors. These tests take into account that latent factors are not known but must be estimated.

Assuming that a set of N variables, x_{it} , can be described by a weighted linear combination of r (smaller than N) factors, F_t , we can apply Factor Analysis to the datasets. This statistical technique accounts for the maximum amount of data variance with a small number of factors while best reproducing the observed correlations between the variables.

$$x_{it} = \lambda_i' F_t + e_{it} \quad i = 1, \dots, N, t = 1, \dots, T \quad (1)$$

In classical factor analysis the error terms e_{it} are presumed to be independent across i and t. In approximate factor analysis this condition is relaxed.

Using principal components as estimates for the factors, the matrix of factor estimates $\tilde{F} = (\tilde{F}_1, \dots, \tilde{F}_T)'$ is given by the r eigenvectors associated with the largest eigenvalues of the matrix $XX'/(NT)$. The factor loadings $\Lambda = (\lambda_1, \dots, \lambda_N)'$ are given as $\Lambda = X' \tilde{F}/T$. In order to determine r, we use the stopping rule for principal component analysis of the approximate factor model developed by Bai and Ng (2002). A number of variants of the information criteria are given with the most popular statistic being:

$$IC_p(r) = \log \tilde{\sigma}^2(r) + r \cdot \frac{N+T}{NT} \ln [\min(N, T)] \quad (2)$$

where $\tilde{\sigma}^2(k) = \frac{1}{NT} \sum_{i=1}^N \sum_{t=1}^T \tilde{e}_{it}^2$ and the tilde(~) indicates estimation by PCA. The number of factors r for which this criterion is minimised gives the estimated number of factors \hat{r} .

Given a matrix G_t of m observed variables, we want to know if they are a linear combination of the r latent variables F_t . Tests have been developed for testing each variable of G_t singly and for testing G_t as a group. Considering the single tests, each variable of G_t may be an exact factor i.e. $G_{jt} = \delta_j' F_t \forall t$ or an approximate factor $G_{jt} = \delta_j' F_t + \varepsilon_{jt} \forall t$. Let $\hat{\delta}_j$ be the least squares estimate of δ_j . Two tests have been developed for the exact case. Letting $\hat{G}_{jt} = \hat{\delta}_j' \tilde{F}_t$ and $\tau_t(j) = \frac{\hat{G}_{jt} - G_{jt}}{(\text{var} \hat{G}_{jt})^{1/2}}$,

We count the proportion of the time series for which \widehat{G}_{jt} deviates from G_{jt} by more than ϕ_α , the α percent critical value of the limiting distribution of $\tau_t(j)$.

This gives the statistic

$$A(j) = \frac{1}{T} \sum_1^T 1(\widehat{\tau}_t(j) > \phi_\alpha). \quad (3)$$

We test how far \widehat{G}_{jt} is from G_{jt} giving the statistic

$$M(j) = \max_{1 \leq t \leq T} |\widehat{\tau}_t(j)|. \quad (4)$$

This is a more stringent test as it demands that \widehat{G}_{jt} be close to G_{jt} at every point in time. Here, e_{it} must be serially uncorrelated for the limiting distribution of $\tau_t(j)$ to be asymptotically normal.

In the approximate case, we use two goodness of fit statistics:

The noise to signal ratio

$$NS(j) = \frac{\widehat{\text{var}}(\widehat{\varepsilon}(j))}{\widehat{\text{var}}(\widehat{G}(j))}. \quad (5)$$

The coefficient of determination

$$R^2(j) = \frac{\widehat{\text{var}}(\widehat{G}(j))}{\widehat{\text{var}}(G(j))}. \quad (6)$$

Testing the group G_t as a set, the canonical correlations between G_t and F_t are considered. The first canonical correlation, ρ_1 , is the largest correlation that can be found for linear combinations of G_t and F_t . The second canonical correlation, ρ_2 , is the largest correlation that can be found from linear combinations of G_t and F_t uncorrelated with those giving the first canonical correlation, and so on. Having to estimate F_t has no effect on the sampling distribution of the canonical correlations. For $k=1, \dots, \min[m,r]$ and $(F'_t, G'_t)'$ identically independently normally distributed,

$$(\rho_k^{2-}, \rho_k^{2+}) = \left(\tilde{\rho}_k^2 - 2\phi_\alpha \frac{\tilde{\rho}_k(1-\tilde{\rho}_k^2)}{\sqrt{T}}, \tilde{\rho}_k^2 + 2\phi_\alpha \frac{\tilde{\rho}_k(1-\tilde{\rho}_k^2)}{\sqrt{T}} \right) \quad (7)$$

where $\tilde{\rho}_k$ is the k^{th} canonical correlation between G_t and \tilde{F}_t . If all the m variables in G_t are exact factors then the canonical correlations will all be unity. If the m variables are linearly dependent then the number of non-zero canonical correlations will be less than m . Any single variables in G_t may be found to be exact or approximate factors from the single tests but may be a linear combination of other observed variables as indicated by the group tests.

Having identified the most appropriate exogenous factors to build into our model of mortality we take the models of Girosi and King (2008) and its extension to allow for exogenous variables King and Soneji (2011) as a starting point to build our epidemiologically informed model of mortality. Girosi and King (2008) developed a method of modelling mortality rates across ages, years and countries which uses a Bayesian hierarchical approach to information pooling. Their objective in doing this was to make use of

beliefs that data across neighbouring ages, years or countries should show similar characteristics. For example, we might expect that the mortality rate experienced by a 20 year old in a given year should be similar to that experienced by the 21 year old or the 19 year old in the same year. Similarly, the mortality rate in say 2000, for a given age should be similar to the mortality rate for that same age in 1999 or in 2001. By using the hierarchical approach Girosi and King (2008) were able to smooth mortality rates for a single country across ages and time and so produce realistic forecasts of mortality that do not break the norms in terms of age and time going forward (for example, mortality rates increasing with age and improving in time). Considering the logarithmically transformed mortality rate during year t for life aged x as $m_{x,t}$ they set out the following model specification:

$$m_{x,t} \sim N\left(\mu_{x,t}, \frac{\sigma_x^2}{b_{x,t}}\right) \quad x = 1, \dots, N, t = 1, \dots, T$$

$$\mu_{x,t} = Z_{x,t} \beta_x$$

This specification only differs from a standard linear regression model in the $b_{x,t}$ weighting that is applied to the variance and in the approach to defining the parameters β_x and σ_x^2 . The specification above provides the basic building block of the Bayesian hierarchical approach in which we now interpret the coefficients β_x and standard deviations σ_x^2 as random variables with their own prior distributions. The prior on the coefficients β_x which depends on its own “hyper-parameter” θ is denoted $P(\beta | \theta)$ with prior on the hyper-parameter $P(\theta)$. The prior for the variance random variable σ is denoted $P(\sigma)$. The functional form of the priors is chosen to be tractable and diffuse so as not to influence the results with a gamma or inverse gamma density function being used.

The prior for the coefficient β is chosen to reflect the “similarity” belief across cross sections. This is formalised by introducing a density function for the prior defined as:

$$P(\beta | \theta) \propto \exp\left(-\frac{1}{2} H^\beta [\beta, \theta]\right)$$

where

$$H^\beta [\beta, \theta] \equiv \frac{1}{2} \sum s_{i,j} \|\beta_i - \beta_j\|_\theta^2$$

where the notation $\|\beta_i - \beta_j\|_\theta^2$ denotes a weighted Euclidean norm and where the symmetric matrix s is called the adjacency matrix.

It’s entries reflect the “proximity” of cross section i to cross section j and so the weight put on the relationship between the coefficients of cross section i and cross section j . Using this approach the fitted

model shows forecasts that are smooth in the age and time dimension and that do not violate the smoothness beliefs across age and time that “may” be violated by using multiple regression methods.

King and Soneji (2011) noted that linear regression could be a useful framework for including potentially informative covariates, either a ‘cohort effect’ (e.g., a cohort’s earlier smoking patterns) or a ‘period effect’ (e.g., the 1964 US Surgeon General’s Report on Smoking and Health (Office of the Surgeon General 1964)). Further by doing this within the Girosi and King (2008) model they also incorporate the empirical regularities of smoothing by age and time imposed in this set-up. The method that King and Soneji (2011) used to develop their model with exogenous covariates was to identify links between mortality rates and lagged covariates, specifically smoking habits and obesity. They argue against using contemporaneous relationships in favour of lagged relationships and from the literature determined the optimal lag period to be 25 years in the case of smoking. They also argue that the additional forecasting step required to project the exogenous variables would lead to additional uncertainty in the model. We argue in this paper that this is not the case where the most appropriate covariates are first identified by objective statistical criteria. Furthermore, although it may be appropriate to use current data to determine future mortality in the case of smoking rates this approach does not facilitate the inclusion of more contemporaneous variables such as current GDP, health expenditure, alcohol consumption or diet. Therefore we attempt to forecast these variables here while acknowledging better forecasts could be obtained using more adequately specified structural models or more sophisticated statistical techniques.

In our model we forecast the identified exogenous variables using ARIMA methods and, taking the resulting forecasts, we build a model of mortality with exogenous variables using the King and Soneji (2011) approach.

4. Results

The pattern of mortality change over the last sixty years can be seen in figures 1-3 for the UK, US and Japan. Although not labelled, it can be inferred that mortality rates at younger ages are near the bottom of the graphs and older age mortality rates at the top. A secular decline in mortality at all ages can be seen in each country. The most noticeable differences between the graphs is at the younger ages. UK death rates for those aged 20-40 are subject to more noisy fluctuations than those at other ages. Figure 2 shows mortality rates for these ages increasing gradually around 1970, decreasing over the next decade and increasing again around 1990. Younger male mortality rates have decreased in Japan much more quickly than mortality rates at older ages especially over the period 1950-1980.

We first of all try to get a sense of the latent factor structure of the mortality data for males in each country over the fitting period 1970-2000. This shorter time period was chosen due to data constraints imposed by the availability of suitable exogenous variables. The number of factors is first determined and these factors are analysed to check their association with younger or older age mortality variation.

Many of the stopping rules for terminating extraction of principal components were developed in psychometric literature and are not used by econometricians as they require the time dimension to be much larger than the number of variables (Breitung and Eickmeier, 2005). Bai and Ng (2002) have developed a statistical test based on information criteria (equation 2) with appropriately chosen penalties to determine the number of static factors for the approximate factor model. Applying this rule we find a similar factor structure for each of the three countries: the estimated number of factors is $\hat{r}=2$ for the UK and captures 86% of the variation in the data while $\hat{r}=4$ for the US and Japan capturing 98% of the data variation in both cases. A Lee-Carter model with one factor or other early derivatives of this model would be therefore inadequate to capture all the common variation in the US and Japanese data while more recent models such as Plat (2009) or Cairns et al. (2006) provide a factor structure of a more suitable dimension for model fitting.

Before associating the factors extracted from the data with real-world trends, the *communality* (the percentage of the variation explained) at each age is estimated and graphed in figure 4. In US and Japan male mortality data, the four principal components extracted explain almost all the variation in the data at every age. In the UK data, older age mortality is also almost completely explained by the three principal components. However younger age mortality variation is only partially explained by common factors and is subject to factors peculiar to particular ages or very small groups of ages (as was mentioned above in relation to figure 4). Causes of death among younger males in the UK are different at different ages with cancers and circulatory diseases accounting for a large proportion of deaths among 35-39 year-olds while among 20-24 year-olds external causes such as transport accidents, suicides and violence are more significant.⁴ The factors extracted for each country tend to be associated with particular ages. From the factor loadings graphed in figures 5 and 6, we see that factors are either associated with younger or older age mortality. For the UK (figure 5) male mortality over 45 years of age is explained by factor 1 while the other factor explains younger age mortality. From figure 6, it can be seen factors 1 and 4 explain US male mortality at older ages while the other two factors explain younger age mortality. This would indicate that we need at least two types of exogenous factors to explain the variation in mortality rates: perhaps lifestyle-related factors to explain younger age mortality (e.g. alcohol consumption) and factors related to health treatment improvements to explain older age mortality. Japanese mortality rates behave differently with most factors not particularly associated with any particular ages except perhaps factor 4 on which middle age mortality ages are more heavily loaded. This would indicate that we require exogenous factors associated with mortality improvements at every age (e.g. income) or alternatively a large set of exogenous factors which together explain each principal component extracted.

Suitable variables describing obesity, medical technology and pharmaceutical advances were not available for these countries over this period. Due to data constraints, only the following exogenous factors could be tested against the principal components extracted from the mortality data : Alcohol consumption (for those aged 15+), Tobacco consumption (15+), Total fat intake , Fruit and Vegetables consumption, Gross domestic product per capita (in 2000 prices) and total expenditure on health per capita(in 2000 prices). These variables are graphed for each country in figures 8-13.

⁴ See for example mortality data for 2000 in Office for National Statistics (UK), Mortality Statistics, Series DH2 No. 27. Table 3.

Alcohol consumption in the US and Japan has peaked and declined while it continues to increase in the UK. The US decline although not exactly concurrent with declines in younger age mortality observed in figure 5 indicate at least a partial explanation for trends there. Even at 2000 levels, UK annual consumption at 10.4 litres of pure alcohol per capita was moderate compared to other countries in the OECD – Luxembourgers consumed 15.4 l per head in the same year - although the more harmful pattern of binge drinking is more common among young adults in the UK than in many countries (Kuntsche et al., 2004).

A decline in smoking (figure 9), economic growth (figure 12) and steep increases in health expenditure (figure 13) may explain declines in mortality at all ages for all countries. Diets have been improving in the UK with less fat and more fruit and vegetables being consumed. The opposite is true in Japan with fruit and vegetable consumption declining slightly and fat intake increasing dramatically although it was still second lowest in the OECD in 2000. Good diet behaviours have been negated by bad ones in the US with both more fat and fruit and vegetables being consumed.

Although we have actual values of all potential covariates for 2001-06 we decided that using predictions of these values would make a fairer assessment of any gains in forecastability. Forecasts were made using the Box-Jenkins methodology. The usual approach of deciding on the number of auto-regressive (AR) and moving average parameters (MA) using information criteria gives poor forecasts. The inclusion of moving average terms generally tends to worsen forecasts. We presume that a researcher would realise this in practice by testing their model. Therefore, the Schwarz information criteria were used to decide between models with various AR terms. As GDP is generally found to be non-stationary (e.g. Westerlund, 2007), this variable was first-differenced and consequently health expenditure per capita also. In summary, the ARIMA(p,d,q) models were UK GDP (2,1,0), US health expenditure (1,1,0), US Alcohol (3,0,0), Japan health expenditure (0,1,0), Japan alcohol (1,0,0) and Japan fat (1,0,0). Assessing prediction errors post-hoc, these models did not necessarily provide the best forecasts but reflect the level of uncertainty encountered in practice.

Each of the proposed exogenous determinants of mortality are, in turn, compared to the principal components extracted from the data using the statistical tests outlined in (3) and (4). Results for the UK are given in Table 2. None of the proposed exogenous determinants is an exact factor as $A(j)$ which should be 5% if factor j is an exact factor. Health expenditure per capita comes closest to being a linear combination of the extracted principal components with $A(j) = 0.484$. The $A(j)$ statistic allows the relationship between the exogenous determinants of mortality and the latent factors not to hold at some points in time. The $M(j)$ statistic is a stronger test and requires that at every point in time the relationship must hold within a small degree of error. Using a 5% significance level with $T=50$ the critical value is 3.28. Not surprisingly the test rejects all of the proposed factors. Bai and Ng (2006) note that if anything both tests are underpowered so we can safely conclude that none of the proposed factors are exact factors. Allowing for a close relationship between proposed factors and latent factors as opposed to an exact relationship is more realistic where variables are measured with error, the statistical indicators do not reflect the underlying construct accurately, the relationship might not be exactly linear or the relationship might be moderated by other factors. The goodness of fit statistic ($R^2(j)$) and the noise to signal ratio ($NS(j)$) indicate how far the proxies are from the true factors. Bai and Ng (2006)

suggest that if $NS(j) > 0.5$ and/or $R^2(j) < 0.95$ then errors in the linear relationship between the proposed factors and the latent factors are non-negligible and the proposed factors are not strong proxies for the latent factors. According to these measures, Health expenditure per capita and GDP per capita are particularly strong proxies and Total fat intake is a particularly poor proxy. Of course, numerous studies have found cointegration between national income and health expenditure (Freeman, 2003; Westerlund, 2007; Moscone and Tosetti, 2010) and using the two variables may provide little extra information than simply using one.

The squared canonical correlations are given in the final column. The first value indicates that there is a linear combination of the proposed proxies and a linear combination of the two latent factors that are highly correlated. The second value indicates that any linear combinations orthogonal to those already found are much less correlated (the confidence interval for the second canonical correlation almost includes zero). There is therefore only one well-defined relation between the two sets and the set of six proxy factors as a whole does not span the latent factor space. As both GDP per capita and health expenditure per capita are both individually highly associated with the latent factors either variable can be used to improve forecasting models.

The results for the US are given in Table 3. None of the proposed exogenous determinants is an exact factor $A(j)$ - alcohol consumption followed by health expenditure per capita are closest to being exact factors according to both the $A(j)$ and $M(j)$ statistics. The importance of alcohol consumption in the US contrasts with its relative unimportance in explaining variations in the UK mortality data. This bears out the correlation noted above between variations in US younger age mortality and alcohol consumption. Allowing for some deviation between the observed variables and the latent factors (columns 4 and 5) and using the rule of thumb from before, Health expenditure, Tobacco consumption, Alcohol consumption and GDP are all strong proxies for the four latent factors. However, the set of six proposed factors does not span the latent factor space as there are only two well-defined relations. The squared canonical correlations between the latent factors and a set of just the two variables Alcohol consumption and Health expenditure per capita are 0.997 and 0.934 suggesting that little is gained by adding the extra four variables. For the purposes of forecasting, these two variables - one a lifestyle variable and the other a medical care variable - appear to be strongly associated with mortality trends and are sufficiently orthogonal to provide distinct forecasting power.

In the case of Japan (Table 4), none of the proposed exogenous determinants is an exact factor. Almost all the variables except tobacco consumption and fruit and vegetable consumption are strong proxies for the four latent factors. This finding is in keeping with figure 7 where a more complicated latent factor structure than for the other two countries was observed. Nevertheless, the set of factors considered does not encompass the latent factor structure. There are two well-defined relations between proxies and latent factors although the third canonical correlation is large. As health expenditure and GDP cointegrate, the three variables - alcohol consumption, fat intake and health expenditure - provide an appropriate basis for forecasting models. This set has squared canonical correlations of 0.995, 0.851 and 0.416 with the latent factors which when compared to column 6 of Table 3 indicates some information is lost by focusing on this smaller subset.

Having now identified the most appropriate exogenous factors to build into the King and Soneji (2011) approach we must look at the dynamics of each variable. We have data for each identified exogenous variable from 1970 – 2006. From this we take the data from 1970-2000 and use ARIMA methods to forecast the values of the exogenous variables from 2001-2006. The exogenous variables can be seen in graphical form in figures 8-13. Taking the forecast exogenous factors from above we apply the King and Soneji (2011) approach to forecast mortality rates. We do this using the freely available YourCast software developed by Girosi and King⁵. For U.K. we use GDP as the only exogenous variable to be built into the model while for the U.S. health expenditure and alcohol consumption are used. Finally for Japan we use health expenditure, alcohol consumption and fat intake. We present the fitting and forecasting results of our model in tables 5-10, for the U.S., U.K., and Japan. We also present the results of fitting and forecasting the mortality data using the Lee Carter (1992) model and the Girosi and King (2008) model with no exogenous variables for comparison. We measure the fitting and forecasting quality using the three measures E1, E2 and E3 outlined below where we take the standardized error to be $\text{Error}_x = (\text{Projected } q_x - \text{Actual } q_x) / (\text{Projected } q_x)$;

(i). The average error E1 – this equals the average of the standardized errors, i.e. Error_x/n , where n = the number of ages included in the summation, that is the mean of the differences. This is a measure of the overall bias in the projections.

(ii). The average absolute error E2 – this equals the average of $|\text{Error}_x|$, that is the mean of the absolute differences. This is a measure of the magnitude of the differences between the actual and projected rates.

(iii). The standard deviation of the error E3 – this equals the square root of the average of the squared errors (Error_x^2), the root mean squares of the differences between the projected and actual rates.

The results are presented in tables 5-7 (forecasting results) and 8-10 (fitting results) and draw a range of comments. We first comment on the comparison of atheoretical models ignoring the use of exogenous variables and then secondly on the value added by incorporating epidemiological information in the King and Soneji (2011) model focusing on the forecasting results and commenting briefly on the fitting results.

Forecasting results

The first, and most important point to note is that our technique, using forecast exogenous variables in a structured model (labelled King and Soneji (2011) in Tables 5-7) performs best across all the countries in the study and using all three measures of fit quality. The improvement over the atheoretical models of Lee Carter (1992) and Girosi and King (2008) is of the order of 1-2% for the U.K. and the U.S irrespective of error measure and 4-4.5% for Japan.

⁵ For more details on the YourCast software used in this study and developed by King and Soneji go to <http://www.gking.harvard.edu/yourcast>.

The improvement in forecasting quality appears to be more pronounced where we have been able to identify more exogenous variables to explain the latent factor structure. This can be seen in the marked improvement in the case of Japan over Lee-Carter where the statistical analysis identified three exogenous variables to explain the latent factor structure and where the improvement in forecasting is 4% on E2 and 4.5% on E3. In the case of U.K. where the statistical analysis identified only one exogenous variable to explain the latent factor structure the improvement in forecasting over Lee Carter is more modest at 1.4% on E2 and 2.4% on E3. Note this is still a substantial improvement over the benchmark model.

When comparing with the Girosi and King method our model still outperforms on all three countries and across all measures of fit quality. In most cases the Girosi and King method outperforms the Lee Carter model but this is not universal, indeed for the Japanese case, the Girosi and King model results in poorer forecasts when compared with the Lee Carter model (E1-E3 in Table 7). This may be due to the more complex factor structure of the Japanese data and the associated cost in terms of fit quality when applying the smooth method employed by the Girosi and King methods.

The three measures E1, E2 and E3 are measuring differing aspects of the forecasting and fitting quality. E1 measures the level of bias seen in the forecast whereas E2 and E3 measure the level of spread seen in the forecast with E3 only differing from E2 in the significance placed on outliers. When looking at the forecasting results on E1 only again our model outperforms across all countries suggesting that the level of bias is lower in the King and Soneji model than in the atheoretical models. The difference when compared with Lee Carter in the case of Japan is particularly marked at 3.8% (Table 7). It is worth noting here also that in some cases the measures for E1 and E2 are the same suggesting that the fitted mortality rates for some models and some countries are overestimated for every age and every year. This indicates that true mortality rates are improving at a faster rate than the atheoretical models can accommodate and suggests that perhaps using a logarithmically transformed mortality rate is no longer sufficient to linearize the data before applying these types of models.

Looking at figures 14-16 we can see the fitted and forecast mortality rates along with the actual mortality rates for each of the models considered in this paper. The first point to note is that we can see a high level of volatility in the actual mortality rates (the solid bold lines) for 20 and 40 year olds the reasons for this were discussed earlier in this paper and include links between incidence of death and the economy for example. The profile for 60 and 80 year olds is less volatile where deaths are less linked to current lifestyle and economic influences and are more determined by a lifetime accumulation of health shocks. In each case the Lee Carter model (blue line) appears to overestimate the mortality rate and this is particularly pronounced in the case of Japan (see Figure 16a for example). The Girosi and King (2008) model appears to underestimate mortality rates for most countries particularly where there has been an upward spike in the most recent mortality experience (Figure 15a). This can be attributed to the smoothing mechanism of the Girosi and King specification and an expectation that future mortality rates may come back down again. Incorporating exogenous variables in our model rectifies some of this underestimation. A particularly good example of our approach improving upon the Girosi and King approach is in the case of Japan where the inclusion of exogenous variables leads to forecasts which are very accurate (Figures 16a and 16b). Again, this is possibly due to the fact we were able to identify more

exogenous variables that were linked to the latent factor structure in the case of Japan. With more data and a bigger spanning set of exogenous variables we may be able to repeat the outperformance shown in the Japanese case for the U.K. and U.S. and other countries that haven't been considered here. A more detailed analysis of the performance of the King and Soneji model at each individual age and year (not shown, available on request) shows it gives particularly accurate forecasts for the middle-ages, 30-60 years of age, for the U.K. and this is consistent across all measures of fitting quality. For the U.S. the story is different, where here the forecasting performance appears superior over the older ages, 55-90 years of age. In Japan where we had the largest set of exogenous determinants, performance is superior across many young and many older ages. We therefore conclude that the larger the set of exogenous variables identified the broader the age range over which forecasts are better.

Fitting results

Turning to the fitting results (Tables 8-10) our model is generally superior across all countries in the study and all measures of fit quality. The one exception is the UK where the fit is poorer on the E2 and E3 measures but as we have seen it nevertheless gives superior forecasts.

Looking at the fitting quality of the Girosi and King approach against the benchmark Lee Carter model it can be seen that the Girosi and King model shows less bias (shown by the lower value of E1) for all three countries considered in the study. However, the Girosi and King model shows a poorer fit quality for all three countries measured on the E2 and E3 measures (e.g. 0.4% worse on E2 and 0.1% on E3 for the UK). This can be explained by the smoothing mechanism in the Girosi and King model which improved smoothness at the cost of a more accurate fit. Finally, when we include forecast exogenous variables the fit is improved. For example, comparing the King and Soneji model with Girosi and King for Japan there is much less bias (0.072% compared with 0.238%) and forecasts show less variation around the actual realised mortality rates (the E2 measured is reduced by 2.1% and E3 is reduced by 2.6%). Of course we expect a better fit for models with a greater number of explanatory variables. Therefore the forecasting results are of greater consequence in this study.

Implications for annuity pricing and product development

In this paper we have shown that the use of exogenous variables in modeling mortality rates can lead to more accurate forecasts of mortality. At the same time, and with further study, identifying the drivers of mortality improvements could lead to a more dynamic approach to forecasting future mortality rates based on improvements in healthcare, or changes in lifestyle not yet seen in the data. This in turn could lead to the development of more accurately priced mortality hedging, derivative-based products which incorporate allowances for changes in the drivers of mortality not just in the changes in mortality themselves.

In this final section we look at annuity pricing using the models discussed in the paper to see how the prices of immediate and deferred annuities would vary when compared to the price using the Lee carter fitting mortality rates.

An annuity of £1 per year payable in advance can be valued using a discounted cashflow model. Assuming an interest rate of 6% (this has been chosen arbitrarily and does not affect the ratio figures) we calculate the value of an annuity as:

$${}_n|a_x = \sum_{k=0}^{89-n-x} v^{n+k} {}_{n+k}p_x$$

Where ${}_{n+k}p_x$ is the probability of a male aged x surviving the next $n+k$ years. We can calculate the probabilities ${}_{n+k}p_x$ using the fitted single year mortality rates q_x using the fact that the probability of surviving one year is $p_x = 1 - q_x$. We then iteratively calculating the probability of surviving the full $n+k$ years. Finally, for comparison purposes we calculate the annuity ratio, AR as:

$$AR = \frac{\sum_{k=0}^{89-n-x} v^{n+k} {}_{n+k}p_x^X}{\sum_{k=0}^{89-n-x} v^{n+k} {}_{n+k}p_x^{LC}}$$

Where ${}_{n+k}p_x^X$ is the probability of a life currently aged x surviving the next $n+k$ year, calculated using either the Girosi and King (2008) method with no exogenous variables or the King and Soneji (2011) method using our forecast exogenous variables and where ${}_{n+k}p_x^{LC}$ is the equivalent probability calculated using the Lee Carter (1992) fitted mortality rates. We look at annuities purchased between the ages 20 and 75 with deferral periods between 0 years and 65 years. Figure 17 shows the resulting contour maps for the ratio of annuity prices under the Girosi and King (2008) approach and the King and Soneji (2011) approach against the Lee Carter method. For the U.K. and U.S. both methods employed in this paper result in higher annuity prices than those predicted by the Lee Carter model. For the case of the U.K. the average increase in annuity prices is 2.2% (Girosi and King) and 3.1% (King and Soneji). For the U.S. the corresponding results are 8.7% and 1.1%. In the case of Japan annuities priced using the Girosi and King method are on average 0.2% higher, whilst using the King and Soneji approach they are 6.6% higher. The significant difference in prices for the U.S. when comparing Lee Carter (1992) to the Girosi and King (2008) approach appears to be coming from the compounding effects of deferred annuity prices where the deferral period is long. The same conclusion applies for the King and Soneji (2011) approach when applied to annuity prices for Japanese data. Using the Girosi and King method for Japanese data there is some underpricing for short deferral periods so on average the effect is not pronounced. At the key ages of 60 and 65 the analysis shows that on average, using the Girosi and King (2008) method the annuity price will be 2.3% and 3% higher respectively across all the countries considered in this study whilst using the King and Soneji (2011) approach with our identified exogenous variables the difference is of the region 2.2% and 3.4% respectively. In all cases using the King and Soneji

(2011) approach leads to higher annuity prices than would be quoted otherwise based on Lee Carter (1992).

5. Conclusion

In this paper we have taken an approach of Bai and Ng (2006) to develop links between the latent factor structure of mortality data and observable factors. We have employed stopping rules to manage the number of factors and so avoid overfitting models. We explain the latent factors by comparing with exogenous factors deemed plausible by public health literature. The techniques employed in this paper are novel to this literature although well known as an econometric method.

We focus on data from the U.S., U.K. and Japan and note that in each of the cases, employing the methods of Bai and Ng (2006) that a differing number of exogenous variables seem to explain the mortality variation in each case. In the case of U.K. we identify one exogenous wealth factor, for U.S. 2 factors are identified one wealth and one lifestyle, finally for Japan, we identify 3 factors two lifestyle and one wealth. We forecast these exogenous factors using ARIMA methods and apply a model which incorporates these exogenous factors using the methods of King and Soneji (2011). Forecasts can be dramatically improved using this approach. We believe that there is scope for further improving forecasts by developing better methods for forecasting the exogenous determinants. We note that the forecasts are improved by the inclusion of these factors.

Further work will involve expanding the set of the exogenous variables that could be useful in explaining mortality variation and using age-specific variables instead of aggregate measures, considering the inclusion of lagged variables where appropriate and considering the ability of the model over a longer term forecasting period.

Tables

Table 1: Descriptive statistics, 1970-2000 (in order UK, US and Japan).

J	Mean	Standard Deviation	Definition
Alcohol	(UK) 9.3 (US) 9.5 (Japan) 7.8	0.6 0.8 1.0	Annual consumption of pure alcohol in litres, per person, aged 15 years and over
Tobacco	2349 2645 3227	516 667 147	Annual consumption of tobacco items (eg cigarettes, cigars) in grams per person aged 15 years and over
Fat	138.9 133.5 73.1	3.0 10.0 8.9	Total fat (grams per capita per day)
Fruit & Veg	151.8 219.7 173.1	14.3 17.6 8.8	All fruit and vegetable consumption (except wine) in kilos per capita
GDP	11896 25420 2,994,819	2275 4712 707,685	Gross domestic product per capita in national currency units at 2000 price levels
Health exp	712 2787 191,957	222 1098 63,732	Total health expenditure (private and public) per capita in national currency units at 2000 price levels

Table 2: Testing the factors in UK male crude mortality rates by single age 20-89, 1970-2000

j	A(j)	M(j)	R ² (j)	NS(j)	$\hat{\rho}(k)^2$
Alcohol	0.839	49.14	0.546 (0.310, 0.782)	0.832	0.991 (0.987, 0.997)
Tobacco	0.871	24.62	0.809 (0.688, 0.930)	0.236	0.323 (0.052, 0.594)
Fat	0.645	26.43	0.313 (0.042, 0.584)	2.195	-
Fruit & Veg	0.871	28.02	0.815 (0.698, 0.933)	0.227	-
GDP	0.645	10.43	0.967 (0.944, 0.990)	0.035	-
Health exp	0.484	14.80	0.970 (0.949, 0.991)	0.031	-

A(j) is the frequency that $|\hat{\tau}_t(j)|$ exceeds the 5% asymptotic critical value. M(j) is the value of the test. R² is defined in 6, NS(j) defined in 5 and $\hat{\rho}(k)^2$ is the vector of canonical correlations G_t with respect to F_t.

Table 3: Testing the factors in US crude mortality rates by single age 20-89, 1970-2000

j	A(j)	M(j)	R ² (j)	NS(j)	$\hat{\rho}(k)^2$
Alcohol	0.323	5.94	0.976 (0.960, 0.993)	0.024	0.999 (0.998, 1.000)
Tobacco	0.710	7.53	0.991 (0.984, 0.997)	0.009	0.951 (0.918, 0.985)
Fat	0.806	25.82	0.911 (0.850, 0.971)	0.098	0.366 (0.096, 0.636)
Fruit & Veg	0.935	44.99	0.878 (0.798, 0.959)	0.139	0.130 (-0.091, 0.352)
GDP	0.806	14.56	0.975 (0.958, 0.992)	0.025	-
Health exp	0.419	5.20	0.997 (0.994, 0.999)	0.003	-

A(j) is the frequency that $|\hat{\tau}_t(j)|$ exceeds the 5% asymptotic critical value. M(j) is the value of the test. R^2 is defined in 6, NS(j) defined in 5 and $\hat{\rho}(k)^2$ is the vector of canonical correlations G_t with respect to F_t .

Table 4: Testing the factors in Japan crude mortality rates by single age 20-89, 1970-2000

j	A(j)	M(j)	$R^2(j)$	NS(j)	$\hat{\rho}(k)^2$
Alcohol	0.484	9.494	0.978 (0.962, 0.993)	0.023	0.995 (0.992, 0.999)
Tobacco	0.484	6.893	0.804 (0.680, 0.928)	0.244	0.938 (0.895, 0.980)
Fat	0.581	8.961	0.992 (0.986, 0.998)	0.008	0.786 (0.652, 0.919)
Fruit & Veg	0.839	39.868	0.795 (0.666, 0.924)	0.258	0.310 (0.039, 0.580)
GDP	0.484	8.338	0.993 (0.988, 0.998)	0.007	-
Health exp	0.548	7.130	0.992 (0.987, 0.998)	0.008	-

A(j) is the frequency that $|\hat{\tau}_t(j)|$ exceeds the 5% asymptotic critical value. M(j) is the value of the test. R^2 is defined in 6, NS(j) defined in 5 and $\hat{\rho}(k)^2$ is the vector of canonical correlations G_t with respect to F_t .

Table 5: Forecasting results for the U.K. for ages 20-89 and years 2001-2006

Model	E1	E2	E3
Lee Carter (1992)	10.600%	10.652%	13.920%
Giroso and King (2008)	10.349%	10.362%	12.413%
King and Soneji (2011)	9.254%	9.254%	11.551%

Table 6: Forecasting results for the U.S. for ages 20-89 and years 2001-2006

Model	E1	E2	E3
Lee Carter (1992)	8.268%	8.325%	11.161%
Giroso and King (2008)	7.462%	7.700%	10.219%
King and Soneji (2011)	6.661%	7.016%	8.841%

Table 7: Forecasting results for the Japan for ages 20-89 and years 2001-2006

Model	E1	E2	E3
Lee Carter (1992)	8.579%	8.579%	10.256%
Giroso and King (2008)	8.790%	8.793%	11.501%
King and Soneji (2011)	4.729%	4.729%	6.007%

Table 8: Fitting results for the U.K. for ages 20-89 and years 1970-2000

Model	E1	E2	E3
Lee Carter (1992)	0.247%	3.501%	4.900%
Girosi and King (2008)	0.138%	3.903%	5.134%
King and Soneji (2011)	0.117%	3.757%	4.940%

Table 9: Fitting results for the U.S. for ages 20-89 and years 1970-2000

Model	E1	E2	E3
Lee Carter (1992)	0.333%	3.832%	5.784%
Girosi and King (2008)	0.040%	4.119%	5.986%
King and Soneji (2011)	-0.089%	2.551%	3.402%

Table 10: Fitting results for the Japan for ages 20-89 and years 1970-2000

Model	E1	E2	E3
Lee Carter (1992)	0.394%	3.857%	5.051%
Girosi and King (2008)	0.238%	4.994%	6.461%
King and Soneji (2011)	0.072%	2.939%	3.853%

Figures

Figure 1 UK male crude mortality rates, 1950-2009 (log scale).

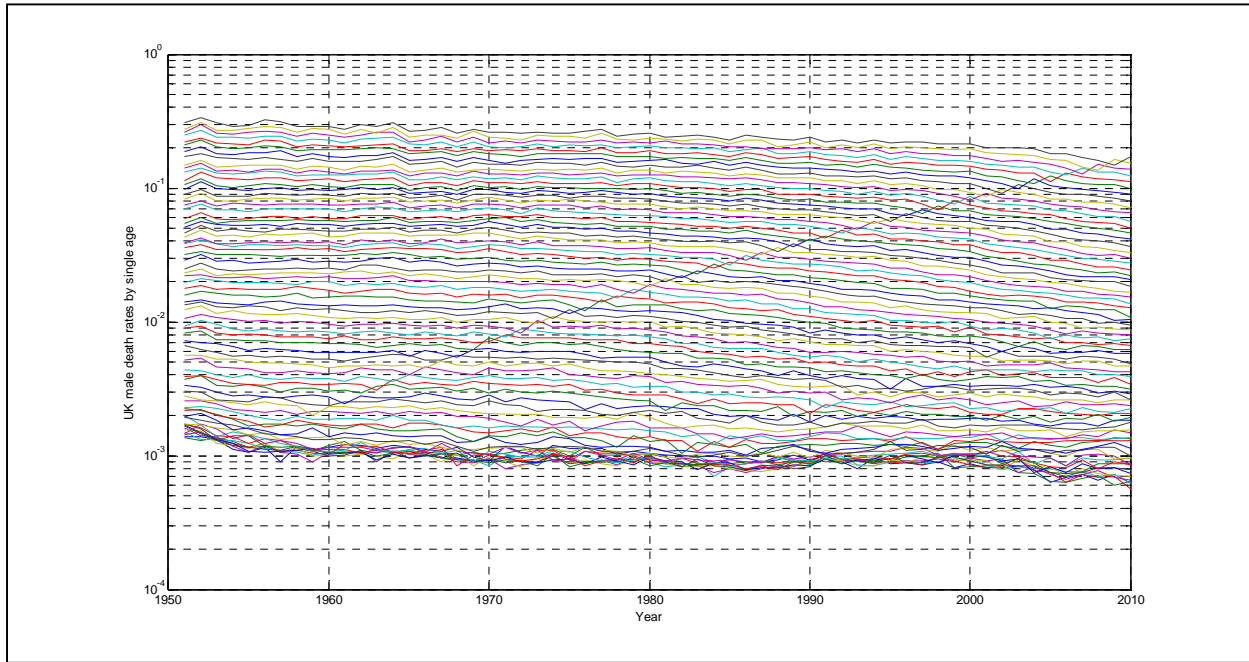


Figure 2 US male crude mortality rates, 1950-2009 (log scale).

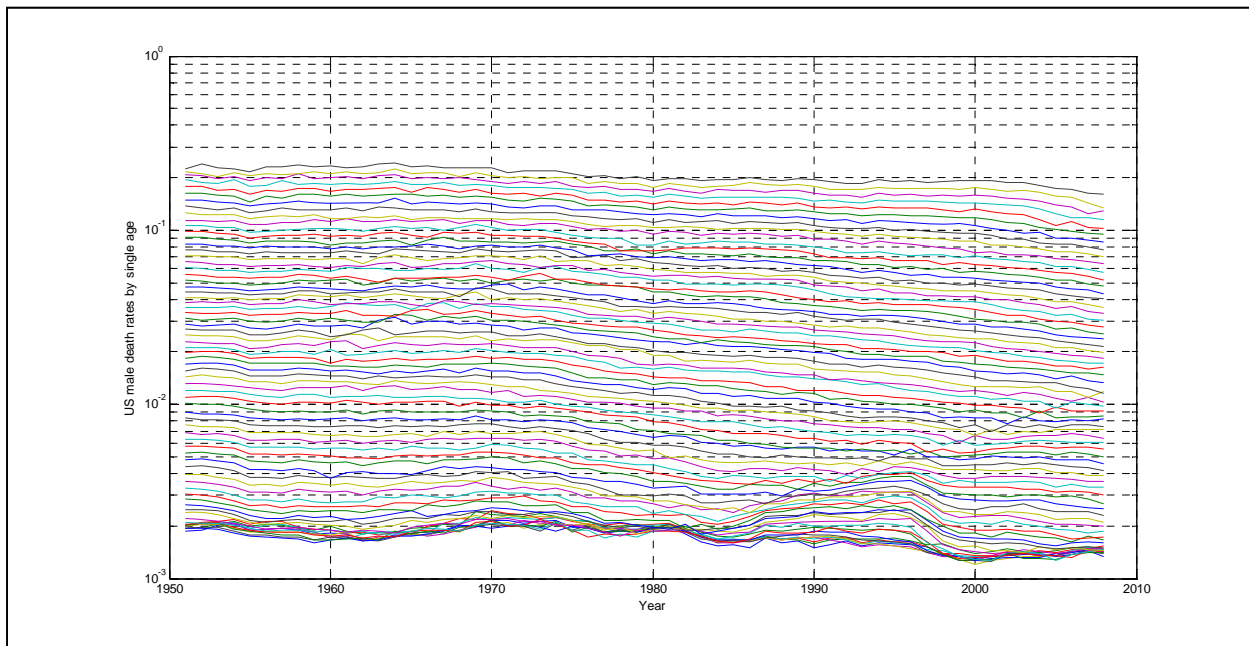


Figure 3 Japan male crude mortality rates, 1950-2009 (log scale).

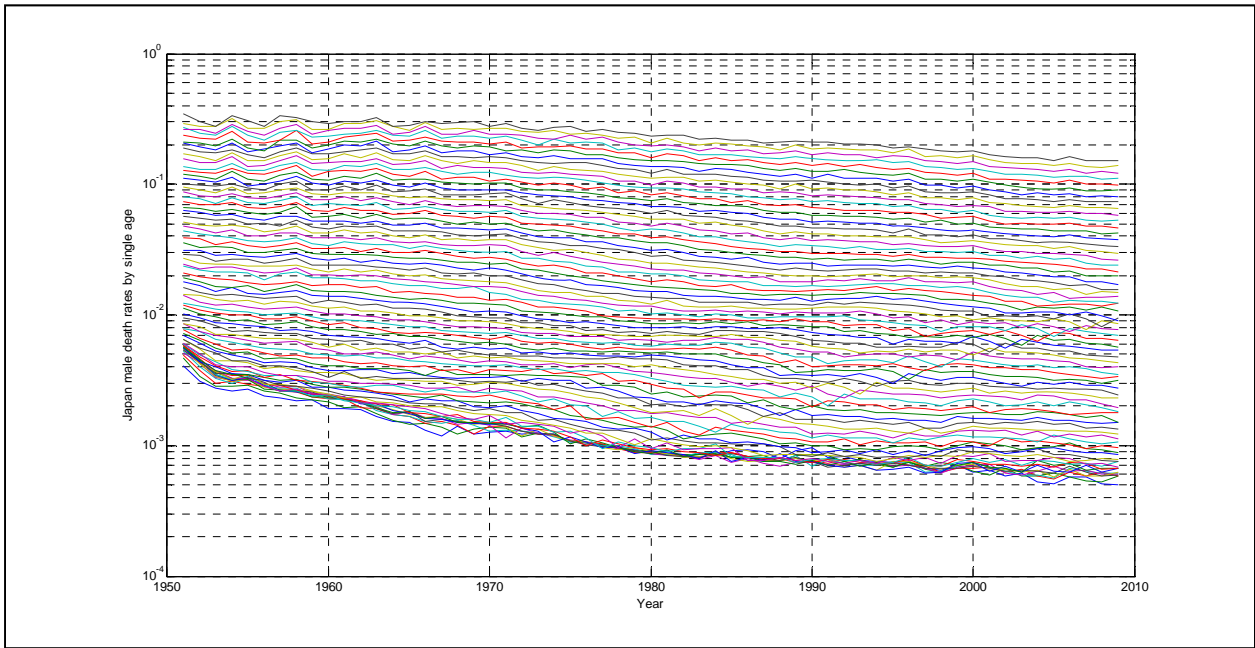


Figure 4 : Proportion of variance explained by principal component extraction (communality) by age for male crude mortality rates, 1970-2000.

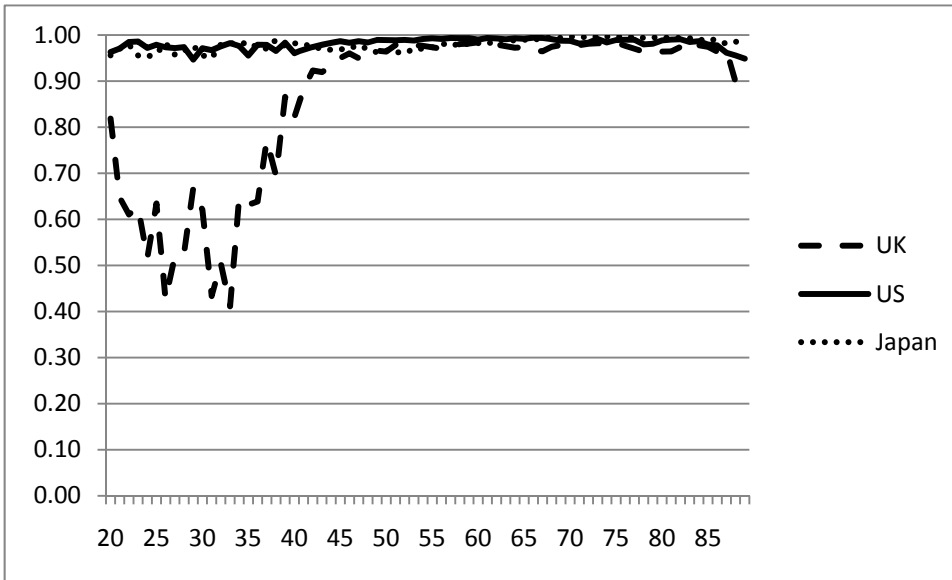


Figure 5 : Rotated factor loadings by age for UK male crude mortality rates, 1970-2000.

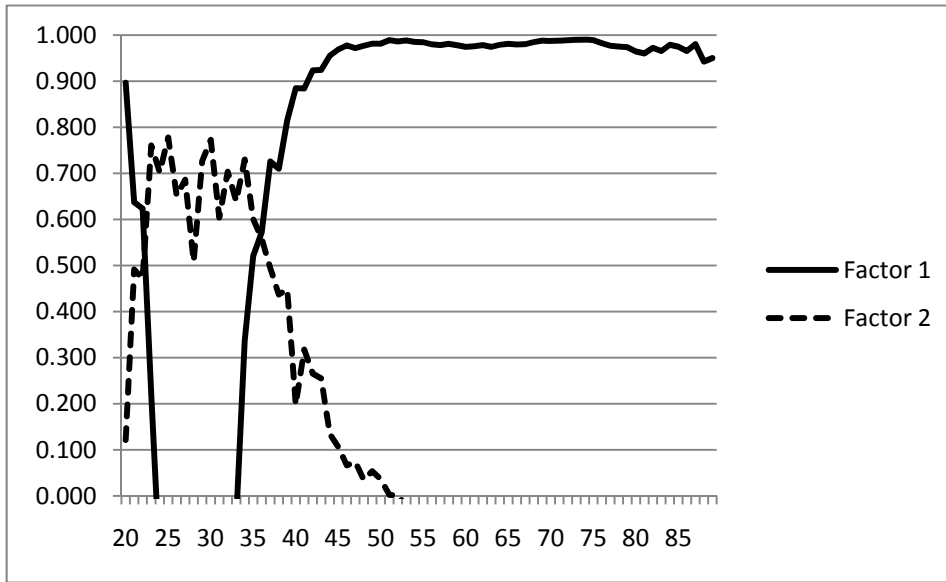


Figure 6 : Rotated factor loadings by age for US male crude mortality rates, 1970-2000.



Figure 7 : Rotated factor loadings by age for Japan male crude mortality rates, 1970-2000.

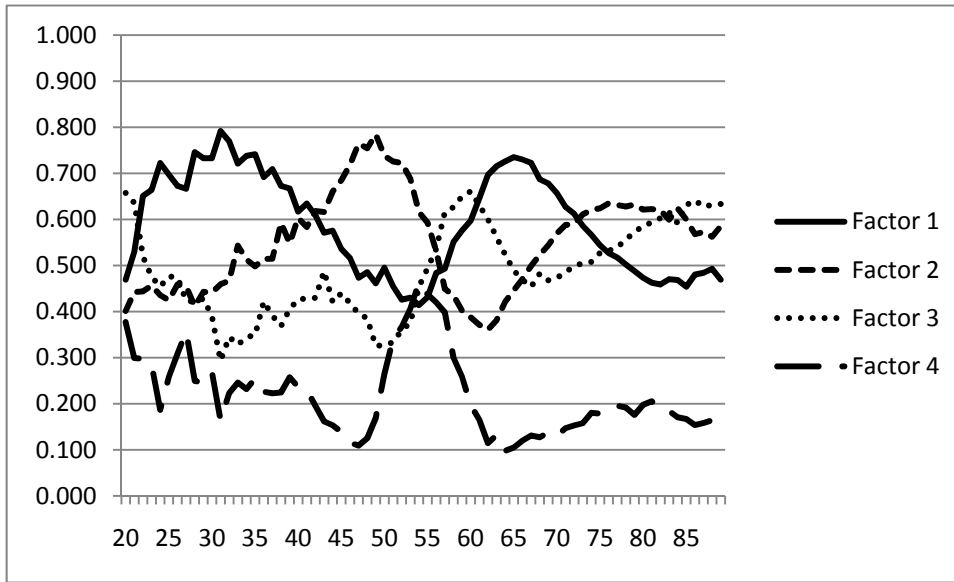


Figure 8 Alcohol consumption - Liters /capita (15+)

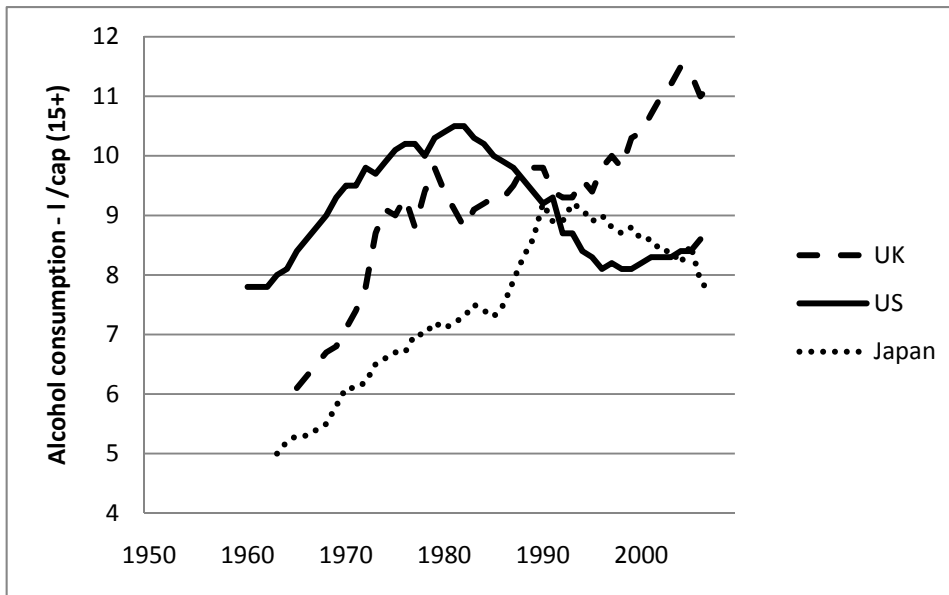


Figure 9 Tobacco consumption - Grammes /capita (15+)

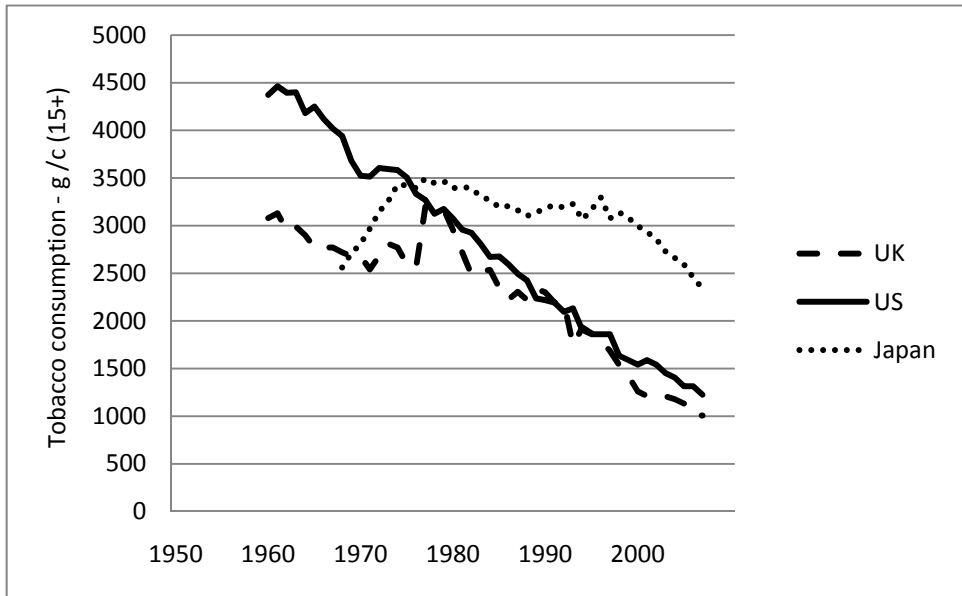


Figure 10 Total fat intake - grammes/capita/day

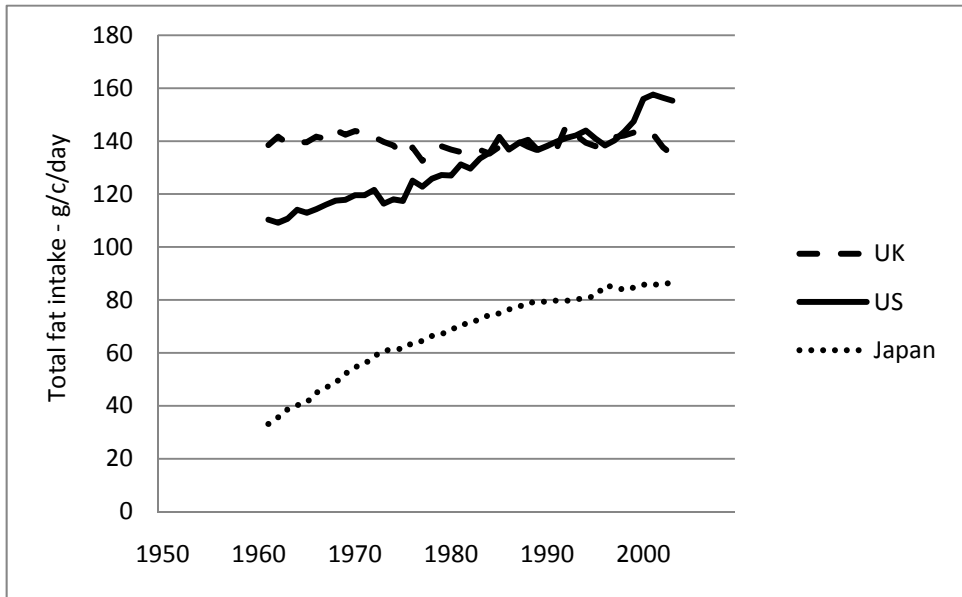


Figure 11 Fruit and Vegetables consumption - kilos per capita

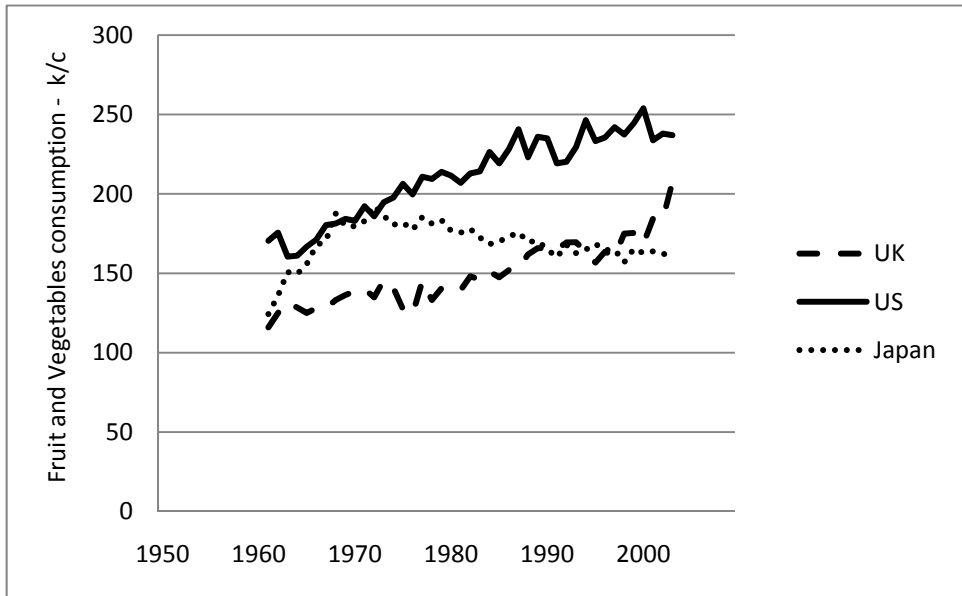


Figure 12 Gross domestic product - /capita at constant prices (1970 = 100)

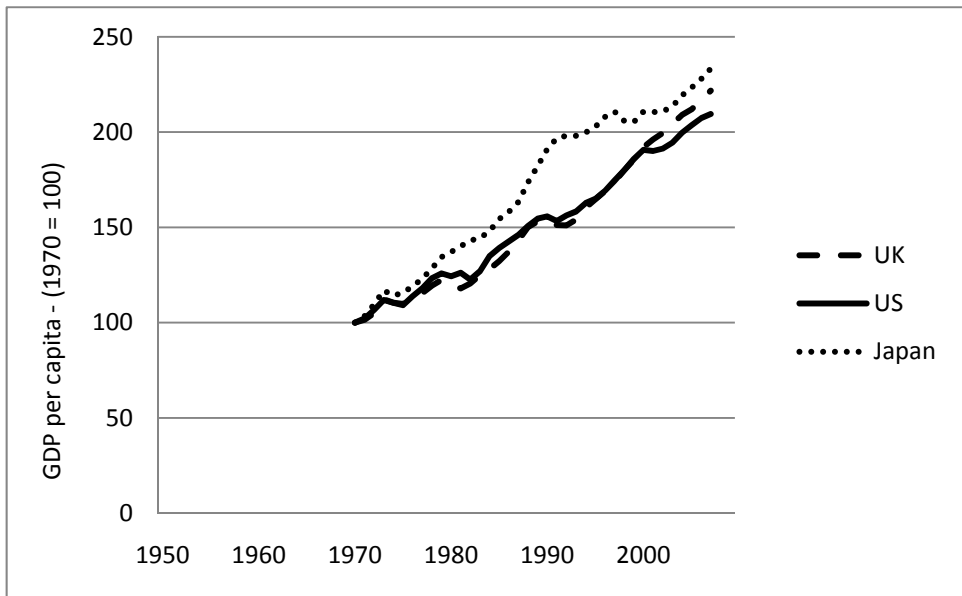


Figure 13 Total expenditure on health - /capita at constant prices (1970 = 100)

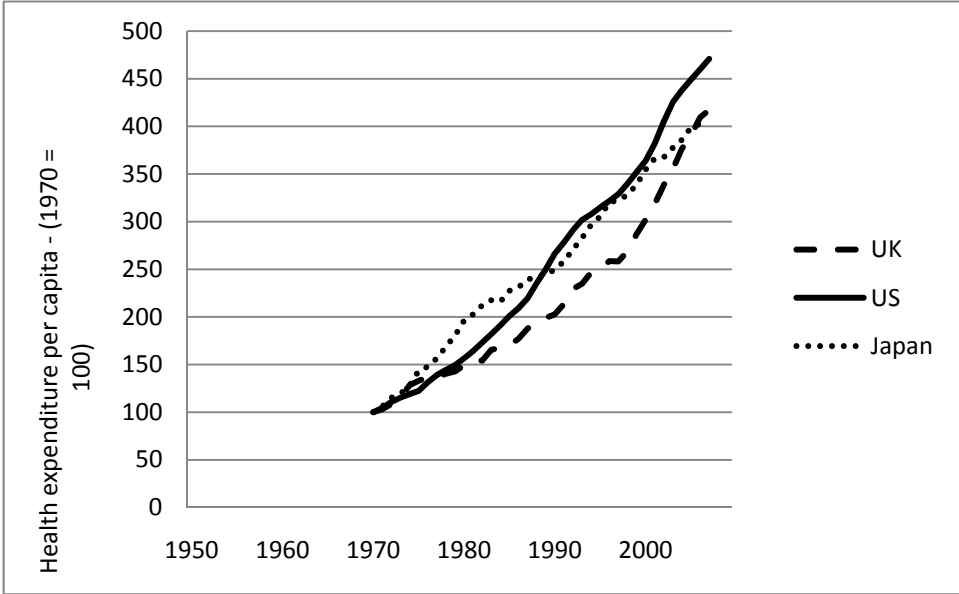


Figure 14 U.K. mortality rates fitted between 1970–2000, and forecast from 2001 - 2006 for the Lee Carter,(blue), Girosi and King (green), King and Soneji, (bold red dashed) models and actual mortality rates 1970-2006 (bold black) for males aged (a) 20, (b) 40, (c) 60 and (d) 80

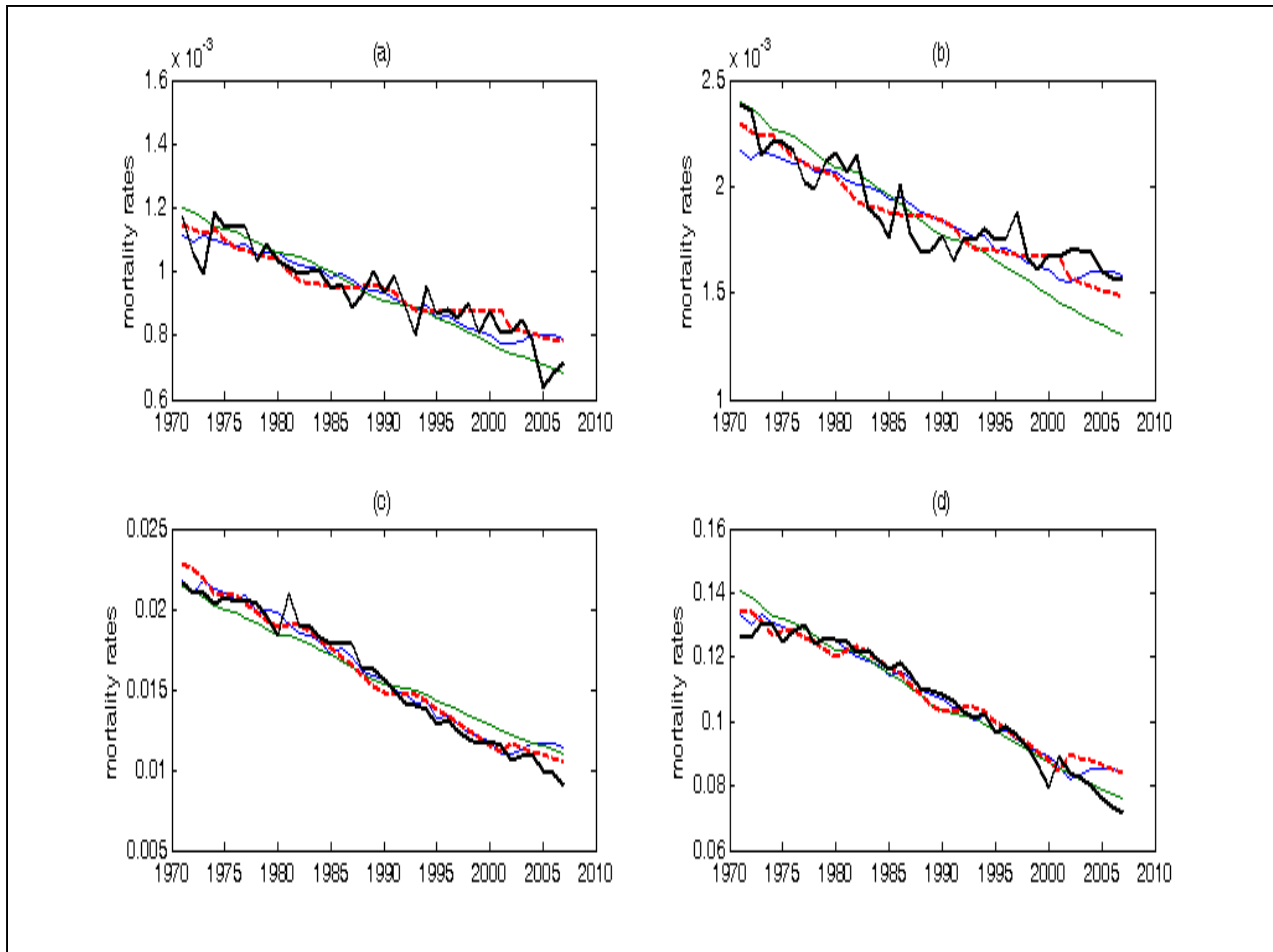


Figure 15 U.S. mortality rates fitted between 1970–2000, and forecast from 2001 - 2006 for the Lee Carter,(blue), Giroi and King (green), King and Soneji, (bold red dashed) models and actual mortality rates 1970-2006 (bold black) for males aged (a) 20, (b) 40, (c) 60 and (d) 80

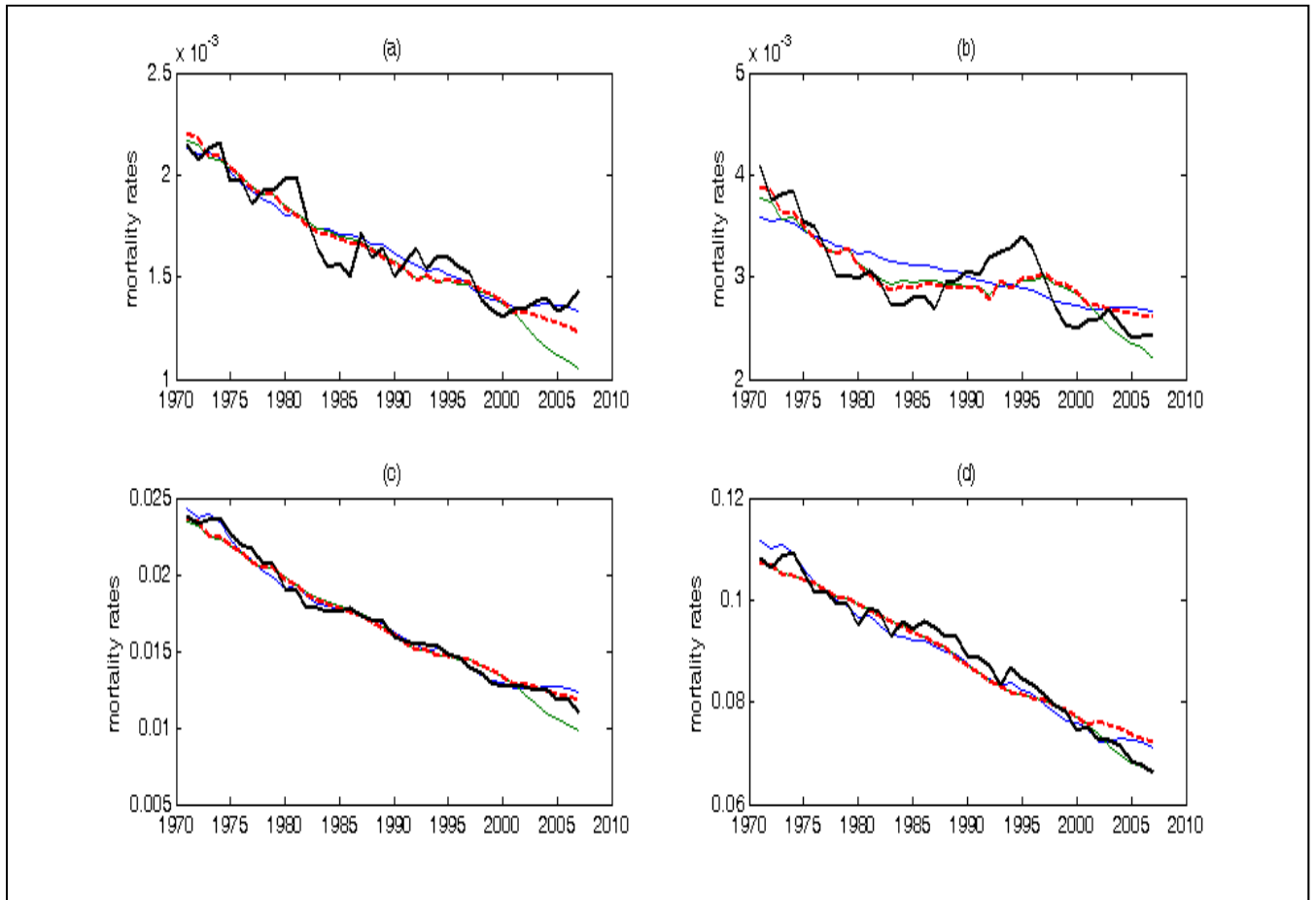


Figure 16 Japanese mortality rates fitted between 1970–2000, and forecast from 2001 - 2006 for the Lee Carter,(blue), Giroi and King (green), King and Soneji, (bold red dashed) models and actual mortality rates 1970-2006 (bold black) for males aged (a) 20, (b) 40, (c) 60 and (d) 80

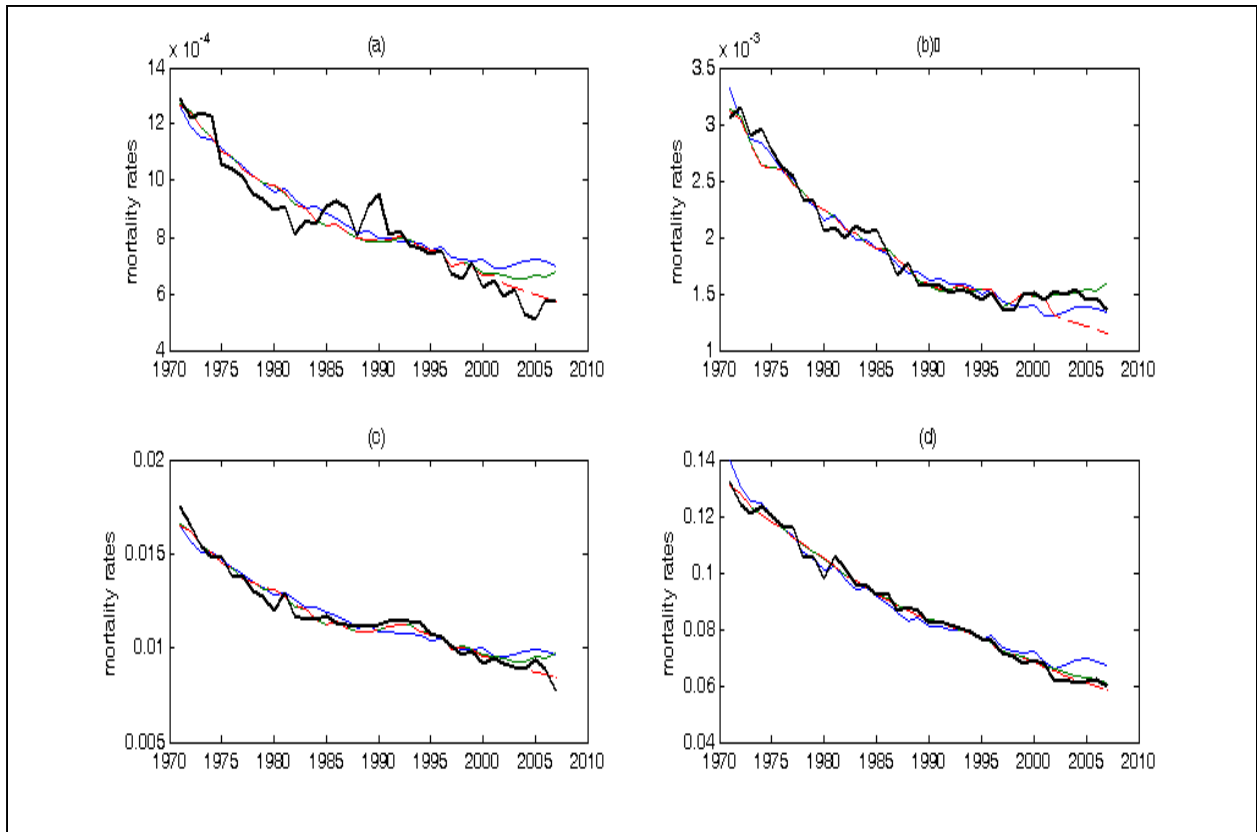
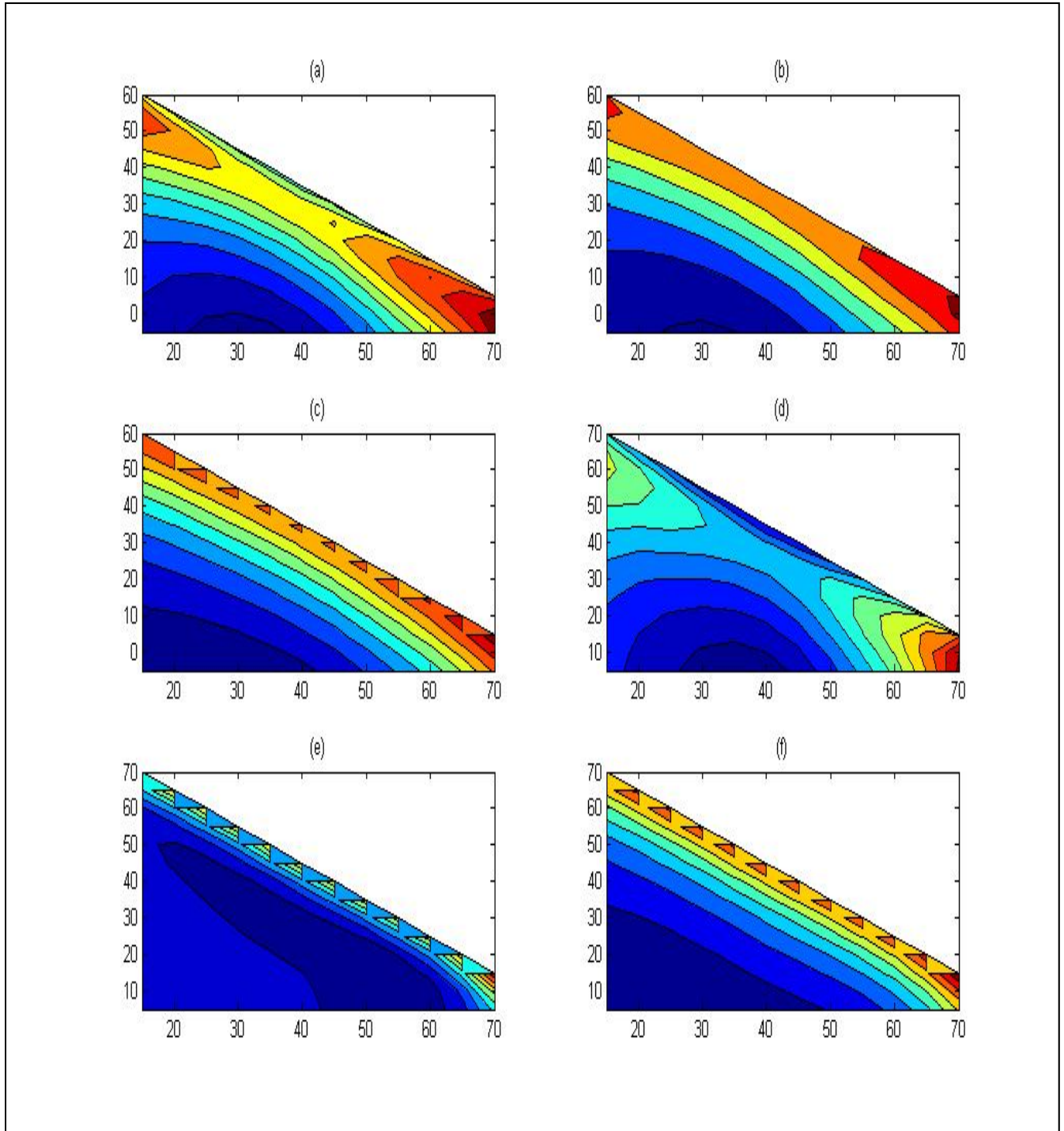


Figure 17 Annuity price ratios for U.K (a, b), U.S.(c, d) and Japan (e, f) using the Girosi and King (2008) model (left) and the King and Soneji (2011) model (right) for issuing ages from 20-70 (x-axis) and deferral periods from 0-65 years (y-axis).



References

- Auster, R., Leveson, I. & Sarachek, D. 1969, "The Production of Health, an Exploratory Study", *The Journal of Human Resources*, vol. 4, no. 4, pp. 411-436.
- Bai, J. & Ng, S. 2006, "Evaluating latent and observed factors in macroeconomics and finance", *Journal of Econometrics*, vol. 131, no. 1-2, pp. 507-537.
- Bai, J. & Ng, S. 2002, "Determining the Number of Factors in Approximate Factor Models", *Econometrica*, vol. 70, no. 1, pp. 191-221.
- Barker, D.J. 1992, *Fetal and infant origins of adult disease*, British Medical Journal.
- Bell, W. 1997, "Comparing and assessing time series methods for forecasting age-specific fertility and mortality rates.", *Journal of Official Statistics*, vol. 13, pp. 279-303.
- Bernanke, B.S., Boivin, J. & Eliasziw, P. 2005, "Measuring the Effects of Monetary Policy: A Factor-Augmented Vector Autoregressive (FAVAR) Approach", *The Quarterly Journal of Economics*, vol. 120, no. 1, pp. 387-422.
- Bethune, A. 1997, "Unemployment and mortality" in *Health inequalities*, eds. F. Drever & M. Whitehead, HMSO, London.
- Breeden, D.T., Gibbons, M.R. & Litzenberger, R.H. 1989, "Empirical Test of the Consumption-Oriented CAPM", *The Journal of Finance*, vol. 44, no. 2, pp. 231-262.
- Breitung, J. & Eickmeier, S. 2006, "Dynamic factor models", *Allgemeines Statistisches Archiv*, vol. 90, no. 1, pp. 27-42.
- Booth, H., & Tickle, L., 2008, "Mortality modeling and forecasting: A review of methods", *The Australian Demographic & Social Research Institute*.
- Brouhns, N., Denuit, M., and Vermunt, J.K., 2002. "A Poisson log-bilinear approach to the construction of projected lifetables". *Insurance: Mathematics and Economics* vol. 31 no. 3, pp. 373-393.
- Cairns, A.J.G., Blake, D. & Dowd, K. 2006, "A Two-Factor Model for Stochastic Mortality with Parameter Uncertainty: Theory and Calibration", *Journal of Risk and Insurance*, vol. 73, no. 4, pp. 687-718.
- Cutler, D., Glaeser, E.L. & Rosen, A.B. 2009, "Is the U.S. Population Behaving Healthier?" in *Social Security Policy in a Changing Environment*, eds. J.R. Brown, J.B. Liebman & D.A. Wise, National Bureau of Economic Research, University of Chicago Press, pp. 423-442.
- Cutler, D., Meara, E. 2000, "The Technology of Birth: Is It Worth It?", *Forum for Health Economics & Policy*, vol. 3, no. (Frontiers in Health Policy Research), Article 3.
- Cutler, D.M. & Meara, E. 2004, "Changes in the Age Distribution of Mortality over the Twentieth Century" in *Perspectives on the Economics of Aging* University of Chicago Press, pp. 333-366.
- Cutler, D., Deaton, A. & Lleras-Muney, A. 2006, "The Determinants of Mortality", *The Journal of Economic Perspectives*, vol. 20, no. 3, pp. 97-120.
- Freeman, D.G. 2003, "Is health care a necessity or a luxury? Pooled estimates of income elasticity from US state-level data", *Applied Economics*, vol. 35, no. 5, pp. 495-502.
- Giroi, F. and G. King 2005, *A reassessment of the Lee-Carter mortality forecasting method*, Harvard University.
- Giroi, F. and G. King 2008, *Demographic Forecasting*, Princeton: Princeton University Press.
- Hári, N., De Waegenaere, A., Melenberg, B. & Nijman, T.E. 2008, "Estimating the term structure of mortality", *Insurance: Mathematics and Economics*, vol. 42, no. 2, pp. 492-504.
- Human Mortality Database [University of California, Berkeley (USA), and Max Planck Institute for Demographic Research (Germany).], Available at www.mortality.org or www.humanmortality.de [Accessed 2011, 10th May].
- Hyndman, R.J. & Ullah, S. 2005, *Robust forecasting of mortality and fertility rates: A functional data approach (working paper)*, Department of Economics and Business Statistics, Monash University, Melbourne.

- Iversen, L., Andersen, O., Andersen, P.K., Christoffersen, K. & Keiding, N. 1987, "Unemployment and mortality in Denmark, 1970-80.", *British Medical Journal (Clinical research ed.)*, vol. 295, no. 6603, pp. 879-884.
- J., T. 2002, "Estimating a health production function for the US: some new evidence", *Applied Economics*, vol. 34, no. 1, pp. 59-62.
- King, G., and Soneji, S., 2011 "The Future of Death in America." *Demographic Research* 25: 1-38.
- Kuntsche, E., Rehm, J. & Gmel, G. 2004, "Characteristics of binge drinkers in Europe", *Social science & medicine*, vol. 59, no. 1, pp. 113-127.
- Kuulasmaa, K., Tunstall-Pedoe, H., Dobson, A., Fortmann, S., Sans, S., Tolonen, H., Evans, A. & Ferrario, M. 2000, "Estimation of contribution of changes in classic risk factors to trends in coronary-event rates across the WHO MONICA Project populations", *The Lancet*, vol. 355, no. 9205, pp. 675-687.
- Lee, R.D. & Carter, L.R. 1992, "Modeling and Forecasting U. S. Mortality", *Journal of the American Statistical Association*, vol. 87, no. 419, pp. 659-671.
- Leon, D.A. 2011=20, *Trends in European life expectancy: a salutary view*. *International Journal of Epidemiology*, vol. 40, no.2, pp. 271-277.
- Miller, R.D.J. & Frech, H.E.I. 2000, "Is There a Link Between Pharmaceutical Consumption and Improved Health in OECD Countries?", *PharmacoEconomics*, vol. 18, no. 3, pp. 33-45.
- Moscone, F. & Tosetti, E. 2010, "Health expenditure and income in the United States", *Health Economics*, vol. 19, pp. 1385-1403.
- Organization for Economic Cooperation and Development 2009, *OECD Health Data 2009: Statistics and Indicators for 30 Countries*, Paris.
- Ostro, B.D. 1983, "The effects of air pollution on work loss and morbidity", *Journal of Environmental Economics and Management*, vol. 10, no. 4, pp. 371-382.
- Plat, R. 2009, "On stochastic mortality modeling", *Insurance: Mathematics and Economics*, vol. 45, no. 3, pp. 393-404.
- Renshaw, A.E. & Haberman, S. 2006, "A cohort-based extension to the Lee-Carter model for mortality reduction factors", *Insurance: Mathematics and Economics*, vol. 38, no. 3, pp. 556-570.
- Ruhm, C.J. 2004, *Macroeconomic Conditions, Health and Mortality*, National Bureau of Economic Research.
- Schwartz, J. & Dockery, D.W. 1992, "Increased mortality in Philadelphia associated with daily air pollution concentrations", *American Review of Respiratory Disease*, vol. 145, pp. 600-604.
- Stewart, S.T., Cutler, D.M. & Rosen, A.B. 2009, "Forecasting the Effects of Obesity and Smoking on U.S. Life Expectancy", *New England Journal of Medicine*, vol. 361, no. 23, pp. 2252-2260.
- Stock, J.H. & Watson, M.W. 2002, "Macroeconomic Forecasting Using Diffusion Indexes", *Journal of Business & Economic Statistics*, vol. 20, no. 2, pp. 147-162.
- Unal, B., Critchley, J.A. & Capewell, S. 2004, "Explaining the Decline in Coronary Heart Disease Mortality in England and Wales Between 1981 and 2000", *Circulation*, vol. 109, no. 9, pp. 1101-1107.
- Westerlund, J. 2007, "Testing for Error Correction in Panel Data", *Oxford Bulletin of Economics & Statistics*, vol. 69, no. 6, pp. 709-748.
- Wilkinson, R.G. & Marmot, M.G. 2003, *Social determinants of health: the solid facts*, World Health Organization, Regional Office for Europe.
- Yang, S.S., Yue, J.C. & Huang, H. 2010, "Modeling longevity risks using a principal component approach: A comparison with existing stochastic mortality models", *Insurance: Mathematics and Economics*, vol. 46, no. 1, pp. 254-270.
- Zweifel, P., Breyer, F., Kifmann, M. & Kifmann, M. 2009, *Health Economics*, Springer.