Basis risk modelling: a co-integration based approach

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BASIS RISK MODELLING: A CO-INTEGRATION BASED APPROACH.*

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Abstract

Most mortality models are generally calibrated on national population. However, pensions funds and annuity providers are mainly interested in the mortality rates of their own portfolio. In this paper we put forward a multivariate approach for forecasting pairwise mortality rates of related population. The investigated approach links national population mortality to a subset population using an econometric model that captures a long-run relationship between both mortality dynamics. This model does not lay the emphasis on the correlation that the two given mortality dynamics would present but rather on the long-term behaviour, which suggests that the two time-series cannot wander off in opposite directions for very long without mean reverting force on grounds of biological reasonableness. The model additionally captures the short-run adjustment between the considered mortality dynamics.

Our aim is to propose a consistent approach to forecast pairwise mortality and to some extent to better control and assess basis risk underlying index-based longevity securitization. An empirical comparison of the forecast of one-year death probabilities of portfolio-experienced mortality is performed using both a factor-based model and the proposed approach. The robustness of the model is tested on mortality rate data for England & Wales and Continuous Mortality Investigation assured lives representing a sub-population.

Keywords: Longevity risk, national and insured mortality rates, co-integration, basis risk.

1 INTRODUCTION

National mortality indices provide a straightforward means to ease an overall view of longevity evolution. Such indices play a key role in advancing the understanding of national populations ageing process but also other different sub-populations as they are a major public and available source of information about the longevity improvements at the national level. Moreover, through some quantitative assessments, mortality of a group of individuals that are part of the whole population can be studied using national datasets. Indeed, a sub-population is likely to share some characteristics with the "parent" population and thus offer scope for the comprehension and the assessment of each through the acquaintance of the other. This potential relationship supported by the presence of common factors that would affect mortality rates across multiple populations in a similar way.

Most notably, with regard to a population and its sub-partitions, some exogenous factors might affect similarly each mortality patterns. For example, the benefits from economic progress which would induce a significant decrease of mortality is fully shared by the population at all levels. Also any medical development leading to greater well-being might impact all the segments of a national population but with an incidence that depends on their access to healthcare. Mortality rates across these populations might also be affected in a similar fashion by pandemics, heat waves or other environmental changing conditions. Dowd et al. (2011) invoke the importance of such features in modeling mortality. They suggest that a *biologically reasonable* model should allow taking into account such behaviours and for interdependence of the mortality rates of sub-populations with regard to the parent population. To a tackle this issue many approaches were proposed in the academic literature: Plat (2009), Cairns et al. (2011) and Dowd et al. (2011) among others.

These methodologies that aim at modeling two different related populations are also motivated by the need for insurers to better understand and predict the mortality underlying their own annuitants or pension portfolios. In such a case, experienced mortality has some specific features that characterize the particular population of annuitants and pensioners. More generally, these are known to show a low mortality profile compared to the national population. This is due particularly both to adverse selection phenomena and to the fact that annuitants are wealthier in comparison with the average population. Generally, the reasons for this mismatch include heterogeneity of socioeconomic status (assessed by income, occupation or education), nutritional standards or sanitation (see Barrieu et al. (2012) for further details on the heterogeneity of longevity risk).

Recently, with the development of the so-called longevity-linked securities market, a specific intent was devoted to the development of quantitative proxies and methodologies that intend to formalize the already observed relationship between the national mortality and its sub-populations, i.e. insured mortality.

Traditionally, practitioners were used to pull-down the national mortality by some fixed ratio to derive the insured ones. This is based upon the stability of the ratio of the two related mortality time-series over time. These practices, nevertheless, are more and more questioned especially by the new European regulation that promotes the use of stochastic frameworks in order to circumvent this issue. Simultaneously, other techniques have been considered in practice like relational models (see Brass (1971)). They offer an alternative to the deterministic approach and suggest to link the two mortality time-series through a regressive model taking into consideration the logistic transform of the time-series, see Hannerz (2001) and Delwarde et al. (2004) for further details.

It should be noted that mortality patterns have been decreasing during the last century and the quantity of data that we already detain about mortality in most industrialized countries should be useful for understanding their sub-populations mortality dynamics. The idea underlying the approach of Brass (1971) and its extensions are making sense knowing that the recent development in the modeling of sub-populations mortality rates argues the need to take account of their dependencies to "parent" population, especially when these are subject to common influences. However, the adequacy of such a model especially with regard to dynamic properties of mortality rates may be questioned. Indeed, this framework implicitly endorses a parallel evolution of the two related mortality rates and that might be inadequate if we are considering, for example, the quantification of the so-called basis risk. This risk emerges from the mismatch between national mortality and the mortality to which pension funds or annuity providers are exposed for instance. Typically, this emerges when those potential mortality-dependent risk hedgers choose to transfer their exposure using an index hedge instrument predicated on some national mortality.

On the basis of the empirical findings of Cairns et al. (2011) and Dowd et al. (2011), we can intrinsically assume that two related populations may share similar global improvement and are subject to common influences. As outlined before, this is likely to be the case for our considered data in extent to which one of the considered populations is an integral part of the national one. Such time-series are hence intended to move together sharing some global movement. This may be apparent when the both time-series are placed side by side. Accordingly, we are looking at the joint econometric features of these time-series and analysis the presence of common stochastic trends. To put this another way, we check out that the two mortality rates have comparable long-run dynamic properties. Next, we propose an econometric model to link the two underlying mortality rates by assuming the existence of common stochastic trends.

This approach is concerned with the estimation of the state variables of the underlying time-series on the long-term. Traditionally, such a framework is used by economists, which could predict long-run behaviour linking up the evolution of one or more state variables. This was used, for example, by Johansen and Juselius (1990) and Dickey et al. (1991) to estimate the money demand function. The underlying methodology, the so-called cointegrated process, was first introduced by Engle and Granger (1987) and is based on the existence of a relationship linking two integrated processes of the same order in such a way that the resulting process is also integrated, but of lower order. In the following, we focus both on the pairwise age-specific mortality and the entire mortality table in order to better understand the behaviour of the common factors across ages or bucket of ages. Moreover, we emphasize the use of such a methodology to enhance the understanding of experienced mortality but also to forecasting future death rates. In the recent years, cointegration analysis has been used to understand mortality dynamics in various countries as to enhance the Lee and Carter (1992) model and advance the understanding of the underlying assumptions related to the number of factors driving the mortality; see e.g. Lazar and Denuit (2009) and Njenga and Sherris (2009), but also the modeling of mortality by cause of death, Gaille and Sherris (2010); and the understanding of the impact of macroeconomic fluctuations on the mortality, see Hanewald (2010).

The primary focus of this paper is then the characterisation of existing relationship and the presence common factors among the considered populations. We conduct a cointegration analysis in order to understand the relationship, if any, between these populations. The nature of this relation is thoroughly analysed as well as the common factors driving changes in mortality rates and of the number of significant factors to include in a factor-based modeling framework briefly discussed in Section 2.

As an important preliminary remark, we want to stress that the adequacy of a model can be assessed from two rather different standpoints. The first one, which we shall adopt here, is to see how well our approach describes the dynamics of the primary object, namely the mortality dynamics. The second one, more concerned with prediction performances, asks how accurate and robust are the model's projections across several time horizons and more precisely with regards to the basis risks.

The remainder of the paper is organized as follows. First, in Section 2 we show how the Lee and Carter model can be interpreted using the econometric techniques using the dynamic factor models and the common stochastic trends approach. We further point out the equivalence between the common stochastic trends framework and the cointegration approach. Finally, in Section 3, the dynamic relationships among the series of age-specific death rates are investigated with the help of the Johansen (1995) maximum likelihood methodology. The forecast of mortality rates are generated using a vector error-correction model (VECM) and comparisons are made with the original Lee and Carter's approach. Meanwhile, we discuss the cause and effect relationship between the related populations.

2 FROM SINGLE POPULATION MORTALITY MODELS TO ECONOMETRIC ANALYSIS

Booth and Tickle (2008) and **?** provide an exhaustive overview of the recent development of stochastic mortality modeling. They discussed mainly the emergence of a new class of models starting with the well-known Lee and Carter (1992) model. The latter has a wide spread recognition in the academic literature and is widely used by life insurance and pension practitioners. The original framework has seen several extensions [see among others: Lee and Miller (2001), Brouhns et al. (2002), Renshaw and Haberman (2003) and more recently Cairns et al. (2009a)] and was used to forecast mortality in various countries. Among the proposed extensions many contributions have discussed the validity of the single time-varying factor driving the entire mortality table. It is, for example, shown that mortality patterns could differ from an age or a bucket of ages to an other and thus lead to encounter for more than a factor in projecting mortality, see Cairns et al. (2006a) for instance.

Lazar and Denuit (2009) show that such a model does not offer a universal framework to handle the mortality modeling issue. In fact, the model has been shown to violate the hypothesis that assumes a unique shared factor over ages for mortality evolution for some different periods and populations. This implicitly suggests that in some cases the mortality patterns are governed by more than one-dimensional factor. In this regard, it is reasonable to introduce models where mortality evolution depends on multiple factors. Cairns et al. (2006b) lead some empirical analyses on the behaviour of the logistic transform[1] of mortality, logit $q_t(x)$, and shows that for fixed time t, it evolves linearly in x. Consequently, they assumed that for each t

$$\text{logit } q_t^n(\mathbf{x}) = \kappa_t^1 + \kappa_t^2\mathbf{x} + \varepsilon_t(\mathbf{x}), \tag{2.1}$$

where κ^1 and κ^2 are two stochastic factors.

On the other side and motivated by the approximation in the Lee and Carter approach that investigated the singular value decomposition of the centered logarithm of mortality, more and more statistical studies on goodness-of-fit of models including second and higher terms are made (see Cairns et al. (2009a) among others). Booth et al. (2002) argued that a full expanded model shall be written as follows[2]

[1] The logistic transform is defined for $x \in [0, 1]$ as logit $(x) = \log(x/(1-x))$.
[2] The original proposition is given in terms of the centred death rates, i.e. $\log m_t(\mathbf{x})$.

$$\text{logit}\ q_t(\mathbf{x}) = \alpha(\mathbf{x}) + \sum_{i=1}^{r} \beta^i(\mathbf{x})\kappa_t^i. \tag{2.2}$$

Most of these works are focusing on the single population modeling. However, in practice, the growing use of longevity-linked derivatives and especially the index-based securities, initiated works on the joint modeling of two or more populations; see e.g. Li and Lee (2005), Biatat and Currie (2010), Cairns et al. (2011), Plat (2009) and Dowd et al. (2011). There are in particular models that are based on extensions of the factorial approach of Lee and Carter (1992). Indeed, Li and Lee (2005) extend this model by assuming a similar improvement process of the considered populations. More formally, a time-varying factor drives both mortality rates while the idiosyncratic variations around the shared process are mean-reverting. Biatat and Currie (2010) investigate the modeling issues of small national population's mortality, i.e. Denmark, together with a much larger population, i.e. Europe-wide.

In Cairns et al. (2011), a new framework is introduced to handle the joint development over time of mortality rates of two related populations with the primary aim of producing consistent mortality forecasts for the two populations. It is achieved using an extended version of Lee and Carter model which incorporates a mean-reverting stochastic spread that allows for different trends in mortality improvement rates in the short-run, but with parallel improvements in the long run.

This paper is in the same vein as Plat (2009) and Cairns et al. (2011) in the sense that we aim at linking two mortality dynamics on a stochastic framework. The starting point of our analysis is a vector autoregressive (VAR) specification of the joint mortality rates, which is a way to estimate dynamic relationships among jointly endogenous variables without specifying any particular structural relationships or exogeneity of the variables. Next, we look for any specific equilibrium that may exist and links the two time-series on the long-run.

With this aim in mind, we adopt the following useful notation. We denote by $\mathbf{x} = (x_{\min}, \cdots, x_{\max})$ the age groups that constitute the basis of our dataset and N_x the number of these groups. Here, $x_{\min}$ and $x_{\max}$ are the first and the last observed individual age or group of ages. Let $\mathbf{t} = (t_{\min}, \cdots, t_{\max})$ be the vector of calendar years and N_t the number of calendar years, i.e. $N_t = t_{\max} - t_{\max} + 1$[3]. Similarly, we introduce the mortality table denoted by $N_x \times N_t$-matrix $\mathbf{q}^i = (q_t^i(x))_{\mathbf{x},\mathbf{t}}$ where the subscript i indicates the underlying population, $i = \text{nat}$ for national population mortality and $i = \text{ins}$ for the experienced mortality or the insured population mortality. Finally, we consider the pair-

[3] As we assumed to work on a 5-year age buckets, the notation makes sense to the extent that $x_{\min} = 60 - 64, x_{\min} = 65 - 70, \cdots, x_{\max} = 84 - 89$ and consequently $N_x = 6$.

wise age-specific mortality $\mathbf{Q}(x) = (q_t^{\text{nat}}(x) \quad q_t^{\text{ins}}(x))_{\mathbf{t}}^\top$ and the $(N_x + N_x) \times N_t$-matrix of the whole mortality surface $\mathbf{Q} = (\mathbf{Q}(x))_{\mathbf{x}}$.

On the basis of the above notation we specify the following generating process and that $\mathbf{Q}_t$ and $\mathbf{q}_t(x)$ can be described respectively by a $2 \cdot N_x$-dimensional and a 2-dimensional Gaussian VAR(p) process, taking into account up to p lags, of the following type:

$$\mathbf{Q}_t = \boldsymbol{\gamma}_t + \sum_{j=1}^{p} \boldsymbol{\Phi}_j \mathbf{Q}_{t-j} + \boldsymbol{\epsilon}_t \quad \text{and} \quad \mathbf{q}_t(x) = \boldsymbol{\mu}_t + \sum_{j=1}^{p} \boldsymbol{\Xi}_j \mathbf{q}_{t-j}(x) + \mathbf{u}_t(x), \tag{2.3}$$

where $\boldsymbol{\epsilon}_t$ is a $2 \cdot N_x$-dimensional Gaussian white noise with $\mathcal{N}(0, \boldsymbol{\Sigma})$ distribution, $\mathbf{u}_t$ denotes a 2-dimensional Gaussian noise with $\mathcal{N}(0, \boldsymbol{\sigma})$ distribution[4]. For $j = 1, \cdots p$, Φ_j and Ξ_j are respectively $(2 \cdot N_x \times 2 \cdot N_x)$-dimensional and (2×2)-dimensional matrices.

Differentiating Equation 2.3 leads to the following vector error-correction model (VECM) specification:

$$\Delta\mathbf{Q}_t = \boldsymbol{\gamma} + \sum_{i=1}^{p-1} \Psi_i \Delta\mathbf{Q}_{t-i} + \Pi\mathbf{Q}_{t-1} + \boldsymbol{\epsilon}_t \quad \text{and} \quad \Delta\mathbf{q}_t(x) = \boldsymbol{\mu} + \sum_{i=1}^{p-1} \Gamma_i \Delta\mathbf{q}_{t-i}(x) + \tilde{\Pi}\mathbf{q}_t(x) + \mathbf{u}_t(x). \tag{2.4}$$

Let us now focus on the non-differentiated terms in Equation 2.4. While supposing that $\mathbf{Q}_t$ is a vector of non-stationary and integrated I(1) variables, all remaining differentiated term $\Delta\mathbf{Q}_t$ are integrated I(0) and $\mathbf{\Pi Q}_t$ must be integrated of order 0 to avoid spurious regression.

Henceforth, we focus particularly on the term $\mathbf{\Pi Q}_t$ which is of great importance since the time-series $\mathbf{Q}_t$ is integrated. The $\mathbf{Q}_t$ being integrated is almost the case of the mortality data as we will test later. The non-stationary feature is a specific characteristic of mortality that arises from the improvements of mortality over years. Hence, the fact that the time-series are integrated, and thus not stationary, could convey the presence of the so-called cointegrating relationship in the vector $\mathbf{Q}_t$. As noted earlier, the cointegration implies sharing a common stochastic trend in the vector $\mathbf{Q}_t$ and thus makes it possible to find a vector β such that $\boldsymbol{\beta}^T\mathbf{Q}_t$ is stationary.

For instance, there are three cases when this requirement that $\mathbf{\Pi Q}_t \sim I(0)$ is met. First, the case where $bl\Pi$ has full rank, i.e. there exist two linearly independent columns. That would lead to a logical inconsistency in **??**. This can be seen by considering $\mathbf{\Pi} = \mathbf{I}$ as a simple full rank matrix. In this case, **??** would define a stationary variable $\Delta\mathbf{Q}_t$ to be equal to a non-stationary variable, $\mathbf{Q}_{t-1}$ plus a stationary lagged variables and a stationary error term. Thus the case where $\mathbf{\Pi}$ is of full rank is not consistent with the

[4] $\boldsymbol{\Sigma}$ and $\boldsymbol{\sigma}$ denotes respectively the $(2 \cdot N_x \times 2 \cdot N_x)$ and (2×2) variance-covariance matrices.

hypothesis of integrated logistic transformation of mortality time-series.

Second, if the rank of $\mathbf{\Pi}$ is zero, which implies that there is no stationary combination of $\mathbf{Q}_t$. In fact, the model in **??** is reduced to a VAR(p) model. More precisely, it means that the insured population and national mortality rates are not linked on the long-run and no equilibrium may be found.

Third, the case where matrix $\mathbf{\Pi}$ has a rank equal to r, i.e. a stationary linear combination exists within the vectors of logit $Q_t(x)$, i.e. a co-integration relationship exists. Conversely, there exists a stochastic common factor that drives the pattern of the two death rates. Furthermore, in this case, we denote $\Pi = \alpha\beta^T$, where α and β are (2×1) matrices. The vector β contains the co-integration coefficient such that βlogit $Q_{t-1}(x)$ is integrated of order 0, i.e. β^Tlogit $Q_{t-1}(x) \sim I(0)$. Under the hypothesis that logit $Q_t(x) \sim I(1)$, all stochastic component are stationary in the VEC model (i.e. **??**) and the system is now logically consistent. Here, α and β stand respectively for the speed of adjustment to disequilibrium and the long-run coefficients.

3 EMPIRICAL ANALYSIS

3.1 SOURCE OF DATA

We focus on England & Wales national mortality data and the Continuous Mortality investigation (CMI) assured lives data-set. The datasets consist of the number of deaths and the central exposure to risk at each age from 60 to 89 in each calendar year $1947 - 2004$.

The CMI male dataset represents the mortality experience of male assured lives holding endowment or whole life assurance policies with UK life insurance companies that have contributed to the CMI's investigation over the concerned period. In the sequel, we make an essential assumption relative to the composition of the CMI assured lives data. In fact, we suppose that subset of the assured males population upon which the mortality rates are computed remains unchanged in the future. The portfolio of assured lives is considered closed to new entries and other exits except those due to deaths. We further consider aggregate mortality statistics for death records and exposure based on 5 year group of ages.

Figure 1 shows the mortality of both national and assured lives. First, the mortality rates of assured lives are lower than those of the national population, which is explained in Cairns et al. (2011) and emphasized by the fact that people holding this type of policy are generally coming from a richer social stratum than average population, or are at least being able to purchase a life insurance policy. At first sight, we can also notice

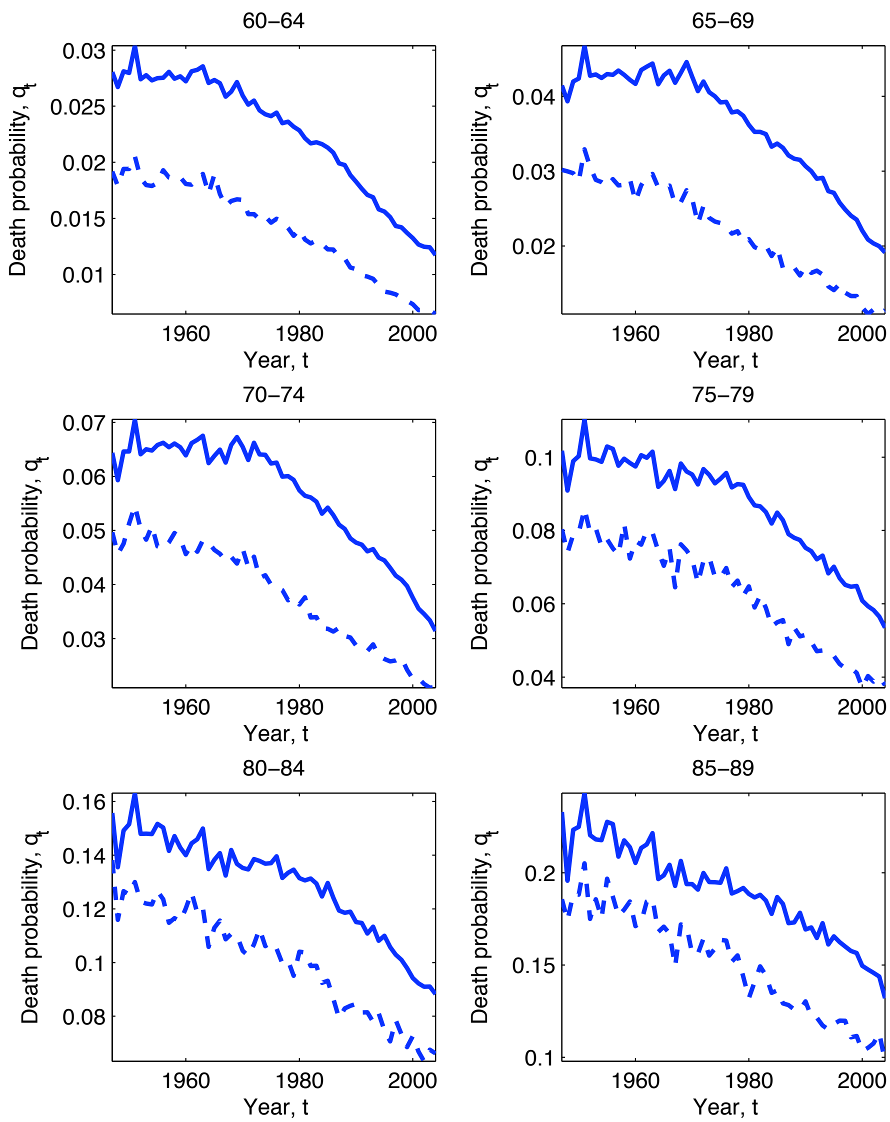

Fig. 1: *One-year death probability for both CMI assured lives population (dashed line) and E&W national population (solid line) for different age buckets.*

from Figure 1 the strong co-movement of the joint mortality rates at each age level. This is also highlighted by Cairns et al. (2011) and Dowd et al. (2011) and might underpin the presence of similar forces that drive the evolution of mortality rates. This specific characteristic is widely recognized in the demographic literature as being the consequence of the normalization of human being conditions across industrialized countries. **?** and **?**, among others, provide evidence for convergence in global mortality levels due to convergence of social and economic factors. Consequently, separate modeling and analyses of these related mortality rates shall compromise this empirical feature and omit any long-run relationship or equilibrium. While doing so, we tend to exaggerate the short-term differences that lead to diverging projections of mortality rates, which seems highly implausible in view of the empirical similarities, see Cairns et al. (2011).

Before performing the cointegration analysis, we should keep in mind that the latter implies a causal linkage between the related mortality rates, at least in one direction. In Table 2 we report the results of the Granger causality test which aims at testing the need of one time-series to forecast the other. This test highlights the presence of at least a unidirectional causality linkages as an indication of some degree of integration. This unidirectional causality informs about leader-follower relationships in terms of mortality improvements. On the basis of Granger causality test results presented in Table 2, unidirectional causality from E&W mortality to CMI mortality is detected. Indeed, an examination of the results indicates changes in national mortality rates leads that of CMI rates. However, the presence of a similar relation in the opposite direction is denied. This finding echoes the implicit hypothesis of Dowd et al. (2011) model, so co-called the gravity model, which considers that the national population "[the larger population] exerts a pull on the insured population [smaller one], but the pull of the smaller population on the larger one is negligible". The gravity model focuses on the interdependency between the two mortality rates by assuming that the period component in Equation 2.2 are mean reverting and a gravitational force is imposed to the evolution of the experienced population mortality. This framework as stated by Dowd et al. (2011) does not discuss the adequacy of the underlying mortality model but rather concentrates on the linkage between the populations. In our case, the underlying model is not a priori specified but we may, for example, conclude from Table 1 that models that are based on up to five temporal factors are not well suited to capture the entire uncertainty on the CMI mortality table. However, this is enhanced by the introduction of the E&W population, see the right panel in Table 1, where the cumulative proportion of variance explained by factor-based models raised while reducing the number of needed factors. For example, the cumulative proportion of variance explained a three-factor model is roughly 95% for the joint populations while does not exceed 92% for the CMI population considered separately.

Insured mortality			**National mortality**			**Joint mortality**		
Std	**% of var.**	Cumul	**Std**	**% of var.**	**Cumul**	**Std**	**% of var.**	**Cumul**
5.31	91.00%	91.00%	5.48	96.80%	96.80%	7.6	93.00%	93.00%
0.57	1.06%	92.05%	0.71	1.60%	98.37%	0.9	1.30%	94.37%
0.53	0.92%	92.97%	0.36	0.42%	98.79%	0.75	0.90%	95.27%
0.5	0.82%	93.79%	0.24	0.19%	98.98%	0.55	0.50%	95.77%
0.45	0.65%	94.44%	0.19	0.11%	99.10%	0.52	0.44%	96.21%
0.45	0.64%	95.08%	0.17	0.09%	99.19%	0.5	0.41%	96.61%
0.42	0.58%	95.66%	0.16	0.08%	99.27%	0.46	0.34%	96.95%
0.39	0.50%	96.17%	0.15	0.07%	99.34%	0.43	0.29%	97.24%
0.38	0.47%	96.63%	0.14	0.07%	99.41%	0.4	0.26%	97.50%
0.36	0.42%	97.05%	0.14	0.06%	99.47%	0.37	0.23%	97.73%

Tab. 1: *Percentage of the observed variation explained by the eigenvectors using principal component analysis.*

The integration of the national population reduced the number of factors that are needed to effectively project the CMI mortality. However, the nature and the role of these factors is not fully understood at this stage. In fact, we should further investigate the relationship between the two considered mortality rates and especially in terms of the econometric behaviour that these may exhibit. The aim of the sequel is the characterization of the interdependency of the mortality rates at each bucket of age. The analysis also aims at advancing the understanding of the two-population dynamics.

		CMI						E&W					
		60-64	**65-69**	**70-74**	**75-79**	**80-84**	**85-89**	**60-64**	**65-69**	**70-74**	**75-79**	**80-84**	**85-89**
CMI	**60-64→**	—	12.03*	9.54*	19.71*	17.45*	15.66*	0.8695	2.6913	1.0742	5.03*	8.60*	2.7662
	65-69→	2.746	—	9.42*	11.60*	18.80*	10.67*	0.5407	0.5716	0.4531	1.6035	3.518	1.42998
	70-74→	4.181	10.68*	—	28.14*	22.16*	24.47*	1.5677	1.3435	1.2054	3.814	4.69*	1.1471
	75-79→	4.065	6.99*	0.077	—	9.17*	9.55*	0.5169	0.7918	1.6957	0.1492	0.8019	2.9532
	80-84→	1.011	4.26*	5.63*	8.95*	—	10.91*	1.0369	0.658	0.4356	2.4555	3.275	1.062
	85-89→	4.061	7.00*	6.25*	5.76*	6.47*	—	1.2393	0.5693	0.8556	0.007	0.4612	1.4221
E&W	**60-64→**	10.77*	11.05*	4.75*	18.44*	16.53*	13.51*	—	3.4182	1.4479	7.6878	13.6312	26.835
	65-69→	3.075	8.02*	6.55*	14.69*	14.39*	12.44*	0.5614	—	3.4552	7.72*	10.12*	26.45*
	70-74→	2.797	3.078	5.17*	12.93*	12.67*	12.34*	0.5299	1.6894	—	12.26*	9.94*	28.98*
	75-79→	3.101	3.431	2.709	8.35*	10.59*	8.69*	0.5299	2.1771	3.1994	—	3.5321	17.33*
	80-84→	2.016	3.946	1.348	5.05*	6.14*	6.87*	0.9109	1.6031	2.0245	0.4338	—	15.57*
	85-89→	1.545	2.778	1.288	2.853	4.83*	6.06*	1.7848	4.47*	6.05*	7.99*	10.19*	—

Tab. 2: *Granger causality test. Each mortality rate is tested for causality on other rates in sense of Granger. The (*) means that the test is significant at 5% based on the F-statistic of the test: H_0: 'x does not cause y'. The table reads as follow "E&W '80-84'→ CMI '75-79'" whose F-statistic is above the 5% critical value, hence mortality rates at the age group '80-84' for E&W population causes the mortality rates at age-bucket '75-79' for the CMI population.*

3.2 VECM specification

3.3 UNIT-ROOT TESTS

As outlined above, Figure 1 suggests that each of the time-series from the log-death rates vectors $\mathbf{q}_t(x)$ and $\mathbf{Q}_t$, taken individually, is nonstationary. Then, the first step of our modeling starts studying the presence of unit roots in the CMI assured lives and E&W national mortality time-series.

We apply classical unit root tests and we verify the presence of unit roots in the time-series we analyze, see Table 3. The hypothesis of unit root is accepted at 5% level and for every test when a constant is included in the test regression. When, also a linear time trend is included in the test regression, the hypothesis of unit root in each time-series is accepted at 5% level by the ADF test, and also at 5% level by the PP test. Furthermore, when we consider the efficient unit root tests, the hypothesis of unit root is always accepted at 5% level and for each test. Also we run the tests for the first differences of the variables, and the null hypothesis of a unit root is rejected at the 5% level.

The results presented above and summarized in Table 3 suggest that CMI mortality and E&W national mortality are I(1) time-series, thus, $\mathbf{Q}_t$ and $\mathbf{q}_t(x)$ are I(1) processes[5]. The purpose of the sequel is to search for long-run equilibrium relationships (common stochastic trends) among the components of $\mathbf{q}_t(x)$, using cointegration techniques.

	ADF test			**PP test**		
	E&W	**CMI**	**5% C.V.**	**E&W**	**CMI**	**5% C.V.**
60-64	-1.0658	-2.5745	-3.5262	-2.0045	-2.5906	-3.5190
65-69	-1.2256	-2.9771	-3.5263	-2.1105	-2.7472	-3.5191
70-74	-0.6193	-2.3202	-3.5264	-1.7584	-2.0338	-3.5192
75-79	-0.4816	-1.8891	-3.5265	-1.6076	-2.2697	-3.5193
80-84	-0.2289	-3.0830	-3.5266	-2.0470	-1.7384	-3.5194
85-89	-1.2691	-3.1641	-3.5267	-1.5196	-2.6291	-3.5195

Tab. 3: *Unit root tests for univariate age-dependent mortality rates for CMI and E&W populations. The ADF unit root test, is based on the regression* $\Delta q_t^i = c + b_0 q_{t-1}^i(x) + \sum_{j=1}^{p-1} b_j \Delta q_{t-j}(x)^i + \epsilon_t(x)$*, with* $\epsilon_t(x)$ *are i.i.d.* $\mathcal{N}(0, \sigma(x)^2)$*-distributed. Table displays the t-statistic, to test the hypothesis* $b_0 = 0$*.*

[5] In the Engle and Granger (1987) sense, that is, a vectorial process in which all univariate components are integrated of the same order.

3.4 COINTEGRATION ANALYSIS AND STATE DYNAMICS SPECIFICATION

We study the presence of cointegrating relationships among the two population time-series using the VAR-based Johansen (1988, 1995) *Trace* and *Maximum Eigenvalue* tests.

The VAR(p) specification is a way to estimate dynamic relationships among jointly endogenous variables without specifying any particular structural relationships or exogeneity of the variables. The number of lags, i.e. p, in this model is selected minimizing some criteria to have a parsimonious model with as few lags as possible. Based on the results reported in Table 6, the lag length is selected (see p^* in Table 6). Then we can re-formulate the Gaussian VAR(2) model in the equivalent vector error correction model (VECM) representation as follows:

$$\Delta \mathbf{Q}_t = \boldsymbol{\gamma} + \Psi \Delta \mathbf{Q}_{t-1} + \Pi \mathbf{Q}_{t-1} + \boldsymbol{\epsilon}_t \quad \text{and} \quad \Delta \mathbf{q}_t(x) = \boldsymbol{\mu} + \Gamma \Delta \mathbf{q}_{t-1}(x) + \tilde{\Pi} \mathbf{q}_t(x) + \mathbf{u}_t(x). \tag{3.1}$$

We focus particularly on the terms $\tilde{\Pi}\mathbf{q}_{t-1}(x)$ and $\Pi \mathbf{Q}_{t-1}$. The fact that the time-series are integrated, and thus not stationary, could convey an existing so-called cointegration relationships in the vectors $\mathbf{q}_t(x)$ and $\mathbf{Q}_t$. The cointegration implies sharing one or more stochastic trends in the vector respectively in $\mathbf{q}_t(x)$ and $\mathbf{Q}_t$ and thus makes it possible to find a vector β and $\tilde{\beta}$ such that $\beta^T \mathbf{q}_t(x)$ and $\tilde{\beta}^T \mathbf{Q}_t$ are stationary.

We determine the rank of the matrices Π and $\tilde{\Pi}$ using the (likelihood ratio) trace and maximum eigenvalue tests. The rank() gives the number of cointegrating relations (the so-called cointegrating rank, that is, the number of independent linear combinations of the variables that are stationary), and $(2-r)$) and $(N_x - r)$ are the number of unit roots (or, equivalently, the number of common trends) driving respectively the time-series $\mathbf{q}_t(x)$ and $\mathbf{Q}_t$.

3.4.1 PAIRWISE AGE-SPECIFIC COINTEGRATION

The results, presented in the top panel of Table 4, indicate that both trace and maximum eigenvalue tests accept the presence of one cointegrating relation ($r = 1$) at 5% level, and, thus, they decide for the presence of two unit roots in the vector $\mathbf{q}_t(x)$, for x in $\{60-64, 65-69, 70-74, 75-79, 80-84, 85-89\}$. Consequently, we can write $\tilde{\Pi} = \alpha\beta^\top$, where α and β are (2×1) vectors (the second part of Table 4 provides the maximum likelihood parameter estimates of these matrices), and $\beta^* \mathbf{q}_t(x)$ will be I(0), see Engle and Granger (1987) and Johansen (1995).

If we look more closely at the parameters of the cointegration relationship, we can study the behaviour of the two mortality rates at an age (bucket of age) level. To sim-

	Trace		Max Eigenvalue					
	r = 0	**r <= 1**	**r = 0**	**r <= 1**				
5% C.V.	**19.96**	**9.24**	**15.7**	**9.24**	α		β	
60-64	41.58	4.53	37.1	4.53	-0.617 (0.132)	0.164 (0.093)	1	-1.080 (0.032)
65-69	38.62	5.93	32.7	5.93	-0.15 (0.02)	0.06 (0.04)	1	-1.24 (0.09)
70-74	33.12	2.99	30.1	2.99	-0.36 (0.01)	0.15 (0.05)	1	-1.59 (0.11)
75-79	19.29	3.02	16.3	3.02	-0.19 (0.04)	0.11 (0.06)	1	-1.07 (0.10)
80-84	23.36	5.12	18.2	5.12	-0.27 (0.06)	0.21 (0.08)	1	-0.87 (0.05)
85-89	16.12	5.37	10.8	5.37	-0.14 (0.09)	0.52 (0.11)	1	-0.74 (0.04)

	r = 11	**r = 10**	**r = 9**	**r = 8**	**r = 7**	**r = 6**
5% C.V.	4.12	12.28	24.28	40.07	59.74	83.36
Test stat.	2.70E-3	7.29	24.07	45.45	69.83	100.88
	r = 5	**r = 4**	**r = 3**	**r = 2**	**r = 1**	**r = 0**
5% C.V.	110.72	142.22	177.8	217.23	260.68	307.16
Test stat.	140.64	214.85	309.46	431.02	564.56	740.99

Tab. 4: *Top panel: Johansen cointegration tests for the vector $\mathbf{q}_t(x)$ observed from 1947 to 2004. The null hypothesis is for both tests H_0 : rank$(\Pi) = r$, where $r = 0, 1$. Both test statistics accept at 5% the hypothesis rank$(\Pi) = 1$ (we use MacKinnon et al. (1999) p-values). Under the restriction $r = 1$, the second half of the table provides the estimates of the adjustment parameters $\alpha = (\alpha_1 \quad \alpha_2)^\top$ (t-values are in brackets) and the cointegrating vector $\beta = (1 \quad \beta_2)^\top$. Bottom panel: Johansen cointegration trace test for the vector $\mathbf{Q}_t$. The null hypothesis is H_0 : rank$(\Pi) = r$ where $r \in 0, 1, \cdots, 11$. The critical values used are given in Trenkler (2003). The trace test accepts the hypothesis rank$(\Pi) = 9$.*

plify, we consider the mortality time-series for two age buckets $60 - 64$. As we can see the behaviour the time-series of each population is similar except in the speed of adjustment. The cointegration relations for this time series induced by the component $\Pi\mathbf{q}_t(x)$, for the two populations, are given by:

$$-\underset{(0.132)}{0.617}\left(-\underset{(0.0006)}{0.003} + q_t^{\text{nat}}(60-64) - \underset{(0.032)}{1.080}\, q_t^{\text{ins}}(60-64)\right) \tag{3.2}$$

$$\underset{(0.093)}{0.164}\left(\underset{()}{-0.0039} + q_t^{\text{nat}}(60-64) - \underset{(0.032)}{1.080}\, q_t^{\text{ins}}(60-64)\right) \tag{3.3}$$

We shall recall that the cointegration relations act as an explanatory variable in the VECM formulation for each pairwise mortality time-series. In Equation 3.2 and in view of the adjustment coefficient $\alpha = (-0.617, 0.164)$ we see that the mortality, in logarithmic scale, for both assured lives and national population is error-*correcting* at the group age $60-64$. In other words, for any deviation from the equilibrium of the national mortality $q_t^{\text{nat}}(60-64)$, say for example that $q_t^{\text{ins}}(60-64)$ is above the equilibrium so that $-0.003+q_t^{\text{nat}}(60-64)-1.080q_t^{\text{ins}}(60-64)>0$, follows an adjustment toward this equilibrium due to the negativity of $\alpha_1=-0.617$. The same argument applies to assured lives mortality which also adjust to equilibrium but with different *speed*. For national population at age bucket $60-64$ the proportion of the deviation from the equilibrium that is corrected each year is 60%. In other words, 60% of deviation is removed each year. On the other hand, the correction for the assured lives corresponds to 16% per period. On the real scale the speed of adjustment is inverted due to the logarithmic transformation of initial data.

Aside from the particular joint influence of the two populations, a simple interpretation of this behaviour - the adjustment - can be argued to explain the differentials. First, we should notice that the CMI assured lives is a sub-population of the England & Wales national population and represent roughly 10% in size[6]. The adjustment of the assured lives to the equilibrium is partly attributed to this fact. The assured population is in fact adjusting because of the aggregate effect that is related to the global environment in which the sub-population evolves. So, the sub-population benefits of improvement at the national level. The same argument is raised by Li and Lee (2005) and Cairns et al. (2011) and suppose that the factor - or the factors - driving the mortality of the two population are mean reverting. Li and Lee (2005) introduce the idea of a global improvement process plus idiosyncratic variations for each population that are mean reverting. In the long run, the global improvement process dominates, resulting in consistent long-term developments in different population. The mean reverting hypothesis assumed by Cairns et al. (2011) is verified by our the error correction model since the insured mortality is correcting with regards to the national mortality and thus will remain correspondingly lower in the future than that of the national population.

Regarding the national error-correction we can argue that mortality is adjusting deviations, caused by increasingly rapid improvements of assured, due to so-called diffusion theory in demography, see e.g. Rogers and Adhikarya (1979). Indeed, as it was outlined by Evandrou and Falkingham (2000), changes in health risk behaviour are adopted first among the middle classes and then diffuse through the population. For example, people in higher socioeconomic classes, who are more likely to be "assured lives", gave up smoking sooner than those in lower socioeconomic classes. The effect of such a change is first observed in assured lives mortality then the national mortality

[6] The CMI data are a subset of the UK population, so are not strictly a subset of the population of England & Wales. However, in the context of this analysis the difference is small.

adjust due the diffusion effect.

On the other hand, the speed of adjustment of the national shows that the proportion of deviation that is corrected each period is slower compared to the adjustment of the insured population (the pace is inverted due to the logistic transformation). In Willets (2004) and Willets et al. (2004) it is shown for example that *cohort effect* was first observed in the CMI assured lives dataset and was "propagated" to national population few years later.

To recap, the insured and national mortality time-series exhibit a long run equilibrium through a cointegration relationship. The time-series error correct and do not wander too far away from each other with different adjustment. For all age buckets the adjustment is quickest for assured lives whereas national population show a slow pace to react to disequilibrium. Note also that changes on the national mortality, i.e. a shock to the residuals in the VECM, will be transmitted more quickly to insured lives than those affecting the insured population.

The purpose in the sequel is to search for long-run equilibrium relationships (common stochastic trends) among the components of $\mathbf{Q}_t$, using cointegration techniques. The objective is fully understand how the common trends at group of age levels interact and more precisely how the common trends are shared in the whole mortality tables.

3.4.2 AGGREGATE AGE-SPECIFIC COINTEGRATION

So far, the time-series of age-specific death rates exhibit a tendency to move together in the long term. At an age level x the time-series $\mathbf{q}_t(x)$ share one common stochastic trend. This is ascribed to the cointegration relationship. In fact, the concepts of common trend and cointegration are two sides of the same coin, being equivalent from the mathematical point of view (see Johansen (1995)).

To fully understand the mechanism of stochastic trends driving the whole mortality tables, i.e. assured lives and national mortality, we should lead a $(2 \times N_x)$-dimensional analysis of cointegration. This is on order to find out which age-specific mortality rates share the same common factors, or how those are shared among group of ages, an analysis of the full system of time-series is required, i.e. looking for cointegrating time-series in $\mathbf{Q}_t$. The single age-specific mortality analysis can be of interest in case, for example, the insurer enter into longevity linked security based on the so-called, for example a *q-forward* contract. In this case, the analysis conducted so far allows to assess the relationship that links the index, i.e. national mortality index, and the insurer own portfolio index, i.e. CMI assured lives.

The Lee and Carter (1992) model assumes that the log-death rates time-series share one common stochastic trend. This implies that there are $N_x - 1$ cointegrating relationships in the N_x-dimensional vector of national population mortality. Furthermore, if we assume that the Lee and Carter (1992) model could fit the insured mortality and that the stochastic trend, i.e. κ, is the same as in the national mortality we end up with a system of $2.N_x - 1$ cointegrating relationships, namely $\mathbf{\Pi}$ is of rank $2 \cdot N_x - 1$ such that $\boldsymbol{\beta}^\top \mathbf{Q}_t$ is stationary ($\boldsymbol{\beta}$ is a $2 \cdot N_x \times (2 \cdot N_x - 1)$ matrix).

Based on results reported in the bottom panel in Table 4, the trace test indicates the presence of 9 cointegrating relations ($r = 9$) at 5% level. Consequently, the vector $\mathbf{Q}_t$ contains $r = 9$ cointegration relations, being driven by $N_x - r = 3$ common stochastic trends. Recall that the Lee and Carter model is based on the hypothesis that there is only one common stochastic trend driving the mortality surface. The present analysis, thus, contradicts this assumption. This shows that the basic assumption of the Lee-Carter model is violated over the $1947 - 2004$ period.

A first approach for extracting factors from the data is to use Principal Components Analysis (PCA). This technique transforms the original multivariate problem to another multivariate setting with less number of time-series. It reduces the dimensionality of the original dataset into a substantially smaller set of uncorrelated variables that captures the most of the information in the original problem, see e.g. Booth et al. (2002) and Koissi et al. (2006).

In the same spirit as Lazar and Denuit (2009) we can reformulate the above problem, Equation 2.2 in a state-space basis with one or more common stochastic factors. The state-space theory, based on the Kalman filtering technique, has been used to estimate the model by Lazar (2009) and Hari et al. (2007). The concepts of common trend and cointegration are two sides of the same coin, being equivalent from the mathematical point of view. Both methodologies are extrapolative approaches, based on the reduction of the data dimensionality. Working in an appropriate inferential statistical framework for projecting death rates is essential, in order to achieve correct inferences, to derive the properties of estimators and to capture the actual dynamics of the death rates. These issues are essential to efficiently manage the longevity risk. In the next section we approach the problem of mortality modeling using the cointegration approach. We first look for an equilibrium in the pairwise mortality time-series. Then we tackle the problem of the number of common factors to include by analysis the cointegration of the whole time-series.

Another way to extract and forecast the common stochastic trends shared by the set of log-death rates time-series, the common factors can be modelled as a multivariate random walk with drift. Since the dynamics of $\mathbf{Q}_t$ can be reformulated as a state space model given by:

$$\begin{cases} \mathbf{Q}_t = \boldsymbol{\beta}\boldsymbol{\gamma}_t + \boldsymbol{\varepsilon}_t, \\ \boldsymbol{\gamma}_t = c + \boldsymbol{\gamma}_{t-1} + \boldsymbol{\rho}_t, \end{cases} \tag{3.4}$$

where $\boldsymbol{\beta}$ is a $(N_x \times 3)$-matrix of loadings parameter and $\boldsymbol{\gamma}_t$ is (3×1)-vector of the common factors.

Notice that the fact that the mortality tables are driven by 3 common factors is consistent with some empirical studies. Indeed, Cairns et al. (2009b) shows that the so-called *M7 model* fits quite well the England & Wales mortality surface. In this model, national mortality time-series are driven by 3 stochastic factors.

4 BACKTESTING AND MODEL PERFORMANCE

We assess the performance of the proposed cointegration model with error correction. For that, an out-of-sample forecasting exercise is performed in the second half of our sample, i.e. starting in 1975 while fixing the parameters at their estimated values. At each time horizon 5, 10, 15 and 20 step ahead forecasts are computed by iterating forward through time. In order to evaluate the prediction performances of the VECM model, we measure the error magnitude by means of the root mean squared error, denoted RMSE. Our forecasts are compared to the original Lee and Carter (1992) model where the parameters are estimated separately on datasets of the assured lives and national population. The relative performance of the VECM model is reported compared to the Lee and Carter model. The results of the out-of-sample forecasting comparison are reported in Table 5.

The conclusion standing out from Table 5 is that the VECM generally outperforms the Lee and Carter model over any forecasting horizon for both population except at the shorter term ($h = 5$ and $h = 10$) for high ages. The Lee and Carter model appears to be more accurate at older ages for the shorter horizon than the VECM. We notice that the differences in model performance tend to increase with the length of the forecast horizon, with the largest divergence corresponding to lower ages, where RMSE over these age-buckets for the Lee and Carter approach is approximately 12 percent greater than for the VECM(1) model for 15-20 horizons for both populations. As far as CMI mortality is considered, VECM(1) model forecasts tend to outperform the Lee and Carter approach in most cases, where roughly of the mortality forecasts involve underpredictions. The same pattern holds true for E&W population, where most Lee and Carter forecasts underpredict mortality except for high ages, i.e. $80 - 84$ and $85 - 89$.

Since, in this work, one of the main objectives is to project the mortality for both

Forecast horizon	CMI					
	60-64	**65-69**	**70-74**	**75-79**	**80-84**	**85-89**
5	53.20%	45.56%	37.46%	13.33%	11.30%	9.15%
10	37.34%	23.33%	31.15%	10.03%	9.51%	6.52%
15	23.91%	20.71%	17.04%	7.90%	8.76%	5.12%
20	12.08%	9.84%	11.03%	5.71%	3.73%	3.11%
	E&W					
	60-64	**65-69**	**70-74**	**75-79**	**80-84**	**85-89**
5	79.30%	77.30%	73.18%	91.43%	112.3%	102.2%
10	65.20%	72.90%	67.10%	87.20%	108.0%	109.1%
15	62.80%	67.20%	63.34%	82.37%	91.70%	83.31%
20	51.50%	56.19%	57.77%	91.21%	88.30%	81.22%

Tab. 5: *Out-of-sample root mean squared errors analysis. Table displays the VECM model's root mean squared errors out of the Lee and Carter's RMSE. The Lee and Carter's model is estimated and projected separately. We implicitly assume that the mortality of any of one population are independent of the other.*

populations in the long term the error correction model and the cointegration relations have significant forecast performances over the Lee and Carter model. The VECM performance comes from its ability to capture the long run equilibrium between the two population. Moreover, as we have seen earlier the Lee and Carter model fails to capture the whole information in the data. In fact, the one-factor specification of mortality evolution is not suitable to model and project the mortality in period considered so far.

Finally, Figure 2 shows the point projection of mortality rates at age-bucket $60-65$. The figure depicts the mortality content of the 95-percent forecast confidence intervals generated by the models over 35 forecast horizon. For this age level, it is evident that over all the projection period, the Lee and Carter yields interval projections that exhibit greater probability content than the VECM(1) model, but are also much wider. The Lee and Carter model seems more likely to generate mortality intervals that are too wide. Conversely, the VECM(1) model tends to produce intervals that are too narrow. On the other hand, the VECM(1) model tend to overestimate the mortality improvements in comparison to the Lee and Carter approach, see Figure 2.

In order to further improve the projection performance of the considered approach we should focus on the entire mortality table.

5 CONCLUDING REMARKS

We have proposed a way to jointly model the evolution of national and policyholders mortality. In this paper we have used and developed an econometric model to study

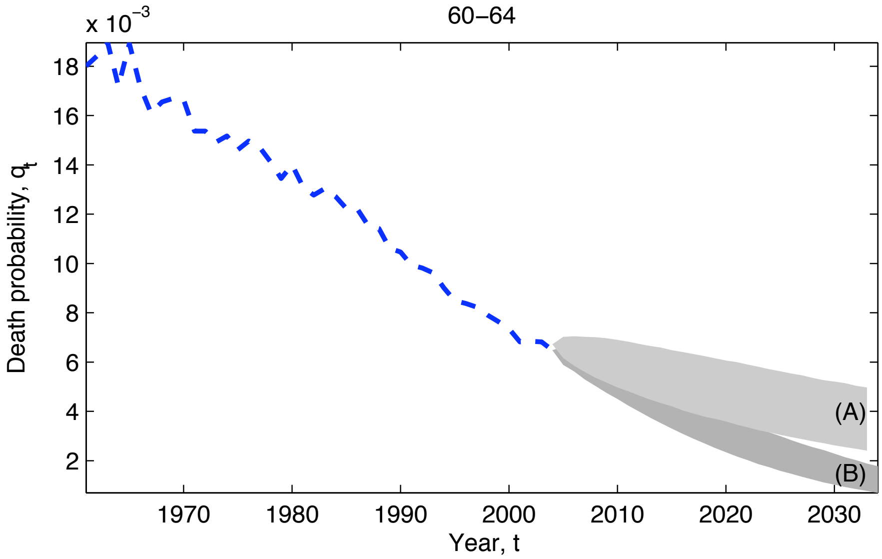

Fig. 2: *One-year death probability for both CMI assured lives population (dashed line) and E&W national population (solid line) for different age buckets.*

issues concerned with the dynamic relationships between mortality rates for two related populations. As far as joint mortality models are concerned, we have used the theory of cointegrating time-series to build the inter-dependence of mortality rates dynamics of the England & Wales population and the CMI assured lives population. In addition, this inter-dependency is analysed in terms of the causality between the two populations and the performance of the projections across different horizons using the cointegration relationships is investigated as well.

Our study suggests that our method seems to perform quite well in the present environment. Of course, it may happen in the future that inequalities increase and that a 2-speed health system develops. In that case, it may happen that the general population does not benefit enough from some key advances of medical science. To take this into account, some stress tests could be performed, with some relation to the evolution of some socio-economic indicators. Some more work would be needed to study correlations between policyholders and national populations in several countries.

A Additional tables

	p	1	2	3		p	1	2	3
60-64	**AIC(p)**	-14.12	-14.34	-14.38*	**75-79**	**AIC(p)**	-12.33	-12.63	-12.64*
	HQ(p)	-14.03	-14.20*	-14.18		**HQ(p)**	-12.25	-12.49*	-12.44
	SC(p)	-13.90	-13.98*	-13.87		**SC(p)**	-12.11	-12.26*	-12.13
	FPE(p)	7.41E-07	5.90E-07	5.69E-07*		**FPE(p)**	4.42E-06	3.28E-06	3.24E-06*
65-69	**AIC(p)**	-12.94	-13.23	-13.35*	**80-84**	**AIC(p)**	-12.50	-12.56*	-12.56
	HQ(p)	-12.85	-13.09	-13.15*		**HQ(p)**	-12.42	-12.42*	-12.36
	SC(p)	-12.72	-12.87*	-12.84		**SC(p)**	-12.29*	-12.19	-12.05
	FPE(p)	2.40E-06	1.79E-06	1.59E-06*		**FPE(p)**	3.71E-06	3.53E-06	3.52E-06*
70-74	**AIC(p)**	-13.17	-13.34*	-13.26	**85-89**	**AIC(p)**	-11.84	-11.88*	-11.88
	HQ(p)	-13.09	-13.20*	-13.07		**HQ(p)**	-11.75*	-11.74	-11.68
	SC(p)	-12.95	-12.98*	-12.75		**SC(p)**	-11.62*	-11.51	-11.36
	FPE(p)	1.90E-06	1.61E-06*	1.74E-06		**FPE(p)**	7.23E-06	6.95E-06*	6.98E-06
Whole table $\mathbf{Q_t}(\mathbf{x})$									
	AIC(p)	-160.37	-160.43	-164.69*					
	HQ(p)	-158.16	-156.19	-158.42*					
	SC(p)	-154.67*	-149.48	-148.48					
	FPE(p)	2.52E-70	5.03E-70	9.29E-71*					

Tab. 6: *Criteria for VAR order selection. The criteria considered for VAR order selection are as follows: LR, FPE, AIC, HQ and SIC. Given the period of our dataset, N_t, and the N-dimensional Gaussian VAR(p) (n=N_x or $n = 2$) process with empirical white noise covariance matrix denotes, for each lag p, the sequential modified likelihood ratio (LR) test statistic, where m is the number of parameters per equation under the alternative, see ?????. The modified LR statistics are compared to the 5% critical values.* **FPE** $= [(T + np + 1)/(T - np - 1)]ndet(p)$ *denotes, for each lag p, the final prediction error criterion. If we denote by $\log(-L) = -(Tn/2)\log(2) + (T/2)\log(|(p) - 1|) - (Tn/2)$ the maximum value of the log-likelihood function associated to the VAR(p) model,* **AIC**$(p) = -2log - L/T + 2pn2/T$, **SIC**$(p) = -2\log -L/T + (\log(T)/T)pn^2$ *and* **HQ**$(p) = -2\log(-L/T) + (2\log(\log(T))/T)pn^2$ *denote, respectively and for each lag p, the Akaike, Schwarz and Hannan-Quinn information criteria. For each criterion, and starting from a maximum lag of $p = 3$, $(*)$ denotes the optimal number of lags.*

	Short-term parameter			**Long-term parameter**			
	$\mathbf{\Gamma}_1$		$\boldsymbol{\mu}$	$\tilde{\boldsymbol{\beta}}$		$\tilde{\boldsymbol{\alpha}}$	$\tilde{\boldsymbol{m}}$
60-64	-0.377	-0.078	0.0046	1	-0.9348	0.0164	2.6507
	0.1934	-0.5135	-0.0066			0.0113	
65-69	-0.1934	-0.1567	0.0016	1	-1.2439	0.0218	-7.7662
	0.6259	-0.6142	-0.0192			0.0108	
70-74	-0.3523	-0.1328	0.0039	1	-1.6341	0.023	7.8717
	0.1884	-0.4289	-0.0094			0.0158	
75-79	-0.2896	-0.1432	0.0039	1	-1.0795	0.0187	3.9888
	0.2865	-0.7125	-0.012			0.0143	
80-84	-0.289	-0.1145	-0.0026	1	-0.855	0.0175	-2.0313
	0.0236	-0.3524	-0.0163			0.0054	
85-89	-0.4267	0.0399	-0.0142	1	-0.7067	-0.0033	11.9987
	-0.415	-0.0707	-0.0051			0.0144	

Tab. 7: *Parameter estimates of the state dynamics* $\Delta\mathbf{q}_t(x) = \boldsymbol{\mu} + \mathbf{\Gamma}_1\Delta\mathbf{q}_{t-1}(x) + \tilde{\boldsymbol{\alpha}}^\top(\tilde{\boldsymbol{\beta}}\mathbf{q}_{t-1}(x) + \tilde{\boldsymbol{m}}) + \mathbf{u}_t(x)$ *with* $\mathbf{q}_t(x) = (q_t^1\ q_t^2)^\top$ *for* $x \in \{60-64, 65-69, 70-74, 75-79, 80-84, 85-89\}$.

	CMI						**E&W**					
	60-64	**65-69**	**70-74**	**75-79**	**80-84**	**85-89**	**60-64**	**65-69**	**70-74**	**75-79**	**80-84**	**85-89**
$\mathbf{z_{1t}}$	1	-0.515	-0.234	0.386	-0.031	-0.066	-0.306	-0.038	0.241	-0.320	-0.002	0.091
$\mathbf{z_{2t}}$	1	-0.112	0.140	-0.040	0.025	-0.055	-0.924	0.239	-0.077	0.026	0.092	-0.055
$\mathbf{z_{3t}}$	1	-5.350	1.266	-0.480	0.441	0.635	1.482	2.722	-1.653	-0.685	0.314	-0.396
$\mathbf{z_{4t}}$	1	0.190	-0.206	-0.012	-0.004	0.021	-0.987	0.231	-0.132	0.208	0.028	-0.092
$\mathbf{z_{5t}}$	1	-0.274	1.270	0.508	-0.631	0.099	-2.063	0.337	-1.709	1.276	0.665	-0.710
$\mathbf{z_{6t}}$	1	-0.024	0.015	-0.107	-0.138	0.020	-0.321	-1.149	1.052	-0.291	0.021	0.108
$\mathbf{z_{7t}}$	1	-0.401	0.343	-0.136	0.055	0.108	-5.739	3.609	-2.350	1.376	0.296	-0.171
$\mathbf{z_{8t}}$	1	-0.582	-0.164	-0.546	-0.035	-0.248	4.169	-0.447	-3.713	4.301	-1.980	0.411
$\mathbf{z_{9t}}$	1	-0.727	-0.436	0.004	-0.445	-0.033	0.942	3.714	-1.423	-1.122	-0.084	0.583

Tab. 8: *Cointegration relationships* $\mathbf{\Pi}$ *for the entire mortality rates* $\mathbf{Q}_t$. *The* $r = 9$ *relationships are reported.*

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