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The SAINT mortality model:

Theory and application

Summary

Pro jecting the mortality rates for a small population is a challenging enterprise. The development of mortality rates for small populations often show great variability in improvement rates over time and across age groups. Consequently, simple extrapolatory techniques yield unreliable results which are very sensitive to the data period and age bands chosen.

In this paper we develop a mortality model which combines small population data with data from a larger reference population. Conceptually, we imagine that the long-term mortality development of the small population will follow that of the reference population but there may be substantial short- to middle-term deviations. Mathematically, we use the reference population to estimate an underlying parametric trend and a 3-dimensional time series to model the deviations from this trend over time and age groups. Mortality projections, including uncertainty assessment, are performed by a combination of trend extrapolation and standard time series methods.

The trend model in itself features an application of frailty theory which rests on the assumption that populations consist of genetically heterogeneous individuals some more frail than others. Generally, high age groups are more homogeneous than young age groups since frail individuals tend to die at younger ages. However, continuing improvements in nutrition, health care, medical advances etc. imply that the frailty composition of the different age
groups change over time. In effect, the model predicts that we will witness higher improvements rates in age-specific death rates for high ages in the future than seen historically.

The combination of a structured underlying trend and a stochastic model accounting for the deviations from the trend allows us to make biologically plausible, stable forecasts from noisy data. We apply the model to Danish mortality data with the reference population being a pooled international data set.

With this application in mind the model has been dubbed SAINT for Spread Adjusted INternational Trend. However, despite the name the reference data set need not represent an international trend. The proposed modelling approach is applicable whenever a suitable reference population can be found, ►

By Søren F. Jarner
Actuarial Department, ATP

Esben M. Kryger
Laboratory of Actuarial Mathematics
Institute of Mathematical Sciences
University of Copenhagen

e.g. the population of interest could be that of a life insurance or pension company with a national or regional data set as reference.

Introduction

Almost all developed countries have experienced steadily declining death rates throughout the 20th century. This development has continued in the first decade of the 21st century and there is no sign of death rates levelling out or improvement rates even slowing down. Even countries like Japan and France which enjoy some of the lowest death rates in the world continue to experience improvements.

Mortality projections, and an assessment of their uncertainty, are of vital importance in a number of areas ranging from public financing policymaking ensuring long-term sustainability of health care expenditures to individual pension saving decisions. The future development in death rates and life expectancy is also highly relevant for pension funds, in particular, those legally obliged to provide adequate reserves for life annuity type liabilities. A great number of mortality models have been proposed most of which can be characterized as purely statistical models extrapolating past trends. Undoubtedly, the most influential and popular model is the one proposed by Lee and Carter (1992) although numerous extensions and other model types have been proposed since then, see e.g. Brouhns *et al.* (2002); Lee and Miller (2001); Renshaw and Haberman (2006); de Jong and Tickle (2006); Currie *et al.* (2004); Cairns *et al.* (2006).

The Lee-Carter model in its original form assumes age-specific, relative rates of improvements which are constant over time. Age groups which has had low rates of improvements in the past will be pro jected to have low rates of improvements also in the future, and likewise for age groups with high past rates of improvement. This lack of structure may lead to biologically implausible forecast with e.g. pro jected death rates for high age groups crossing that of younger age groups. In fairness, the model was developed specifically for the US which, like other large populations, has had a regular improvement pattern across age groups over time (at least since 1950) in which case the intrinsic lack of structure of the Lee-Carter model does not become apparent.

However, for small populations, like Denmark, the mortality evolution has been much more erratic with great variability in rates of improvements over time and age groups in which case simple extrapolative techniques do not produce sensible forecasts.

Another shortcoming of the Lee-Carter and similar methods is the inability to forecast improvements in age groups which have not experienced improvements in the past (of course, some may argue that this is a virtue). Thus high age groups, say the 90-year-olds, which historically have had only very modest rates of improvements will be pro jected to have very low rates of improvement in the future also, while the death rates for younger age groups will improve at a higher rate thereby creating an increasingly steep mortality curve at high ages. This scenario is consistent with the existence of a fixed upper limit to human life spans which some do indeed believe to be the case.

There are, however, good reasons to believe that high age mortality will improve more markedly in the future than in the past. Reaching the age of 90 has historically been achievable for only the most robust individuals which have been relatively insensitive to the general level of health in the society. However, as reaching the age of 90 becomes more common this group will be more similar to the younger age groups and more responsive to future medical and other improvements. These ideas can be formalized by the use of frailty theory which assumes that people are born with an individual level of frailty.

The purpose of the work presented here is twofold. First, to produce stable long-term projections from volatile mortality data. Second, to ensure biologically plausible forecasts which allow for non-constant rates of improvement over time.

The first aim is achieved by a modelling framework in which an underlying trend is estimated from a larger reference data set while a time series model accounts for the observed deviations of the mortality data of interest from the trend. This structure guarantees stable long-term behavior while accommodating substantial local variation.

The second aim is achieved by choosing a parsimonious parametric model for the trend with biologically interpretable components, one of which being the amount of heterogeneity in frailty levels (at birth).

The paper is organized as follows. In section 2 we present the Danish mortality data along with the international reference data set and present the proposed modelling framework, section 3 contains a short exposition of frailty theory and develops the proposed parametric form for the underlying trend, the trend model and its estimated parameters are given in section 4, while the spread model and its estimated parameters are found in section 5.

2. Data description and modelling framework

2.1. **Data.** Data for this study originates from the Human Mortality Data-base[2] , which offers free access to updated records on death counts and exposure data for a long list of countries. The database is maintained by University of California, Berkeley, United Stated and Max Planck Institute for Demographics Research, Germany.

For each country and each sex data consists of death counts, $\{D(t,x)\}$, and corresponding exposures, $\{E(t,x)\}$, for a range of years t and ages x. $D(t,x)$ denotes the number of deaths occurring in calender year t among people aged x, and $E(t,x)$ denotes the total number of years lived during calender year t by people of age x. For readers familiar with the Lexis diagram, $D(t,x)$ counts the number of deaths in the square $[t,t+1)\times[x,x+1)$ of the Lexis diagram and $E(t,x)$ gives the corresponding exposure.

We will use both Danish data and a pooled international data set consisting of data for the following 18 developed countries: USA, Japan, Germany, UK, France, Italy, Spain, Australia, Canada, Holland, Portugal, Austria, Belgium, Switzerland, Sweden, Norway, Finland and Iceland.

The subsequent analysis uses data from the years 1935 to 2004 and ages 20 to 100. As far as the time dimension is concerned the cut points are determined by the availability of US data. Concerning the age span the analysis could in principle be based on all ages from 0 to 110. However, since the prime focus is adult mortality and since the mortality pattern at young ages differs markedly from adult mortality all ages below 20 have been excluded. For very high ages the quality of data is poor and sometimes based on calculated quantities and for this reason all ages above 100 have also been excluded.

In the following, $D_{Int}(t,x)$ and $E_{Int}(t,x)$ denotes, respectively, the total number of deaths and the total exposure in calender year t among people aged x in those of the 18 countries for which data exists for that year. The same quantities for Denmark will be denoted $D_{DK}(t,x)$ and $E_{DK}(t,x)$, respectively. From the death counts and exposure data we form the (crude) death rates

$$(1)\quad m(t,x)=\frac{D(t,x)}{E(t,x)} \qquad \text{for } t=1935,\ldots,2004,\ x=20,\ldots,100.$$

The pooled data set has for women a total of 180 million deaths with an exposure of about 15 billion person years and for men a total of 197 million deaths with an exposure of about 14 billion person years. For Danish females there are a total of 1.6 million deaths with an exposure of 120 million person years and for Danish males there are a total of 1.7 million deaths with an exposure of 114 million person years. Thus the international data set is approximately 100 times larger than the Danish data.

2.2. **Historic development.** The evolution of age-specific death rates in Denmark can be seen on Figure 1. On first sight it seems that death rates have been steadily declining throughout the period. However, a closer look at the numbers reveals that there has been considerable variation in the pace of improvements over time and across age groups. A thorough descriptive analysis of the evolution in Danish death rates from 1835 to present, including a life expectancy decomposition analysis, can be found in Jarner *et al.* (2007). Here we will give only a brief overview of the development in Danish mortality and compare it with the international development.

The period under study can informally be divided into four subperiods: 1935-1950, 1950-1980, 1980-1995 and 1995-2004, each having a distinct improvement pattern. In the first period (which in fact goes back to 1900) there were considerable improvements in death rates for both sexes and most age groups, except for the oldest old. The annual rate of improvement in agespecific death rates were about 3% for ages below 30, just below 2% for ages between 30 and 60, and about 0.5% for ages above 60. For a precise definition of how these numbers are calculated see Jarner *et al.* (2007).

[2] See www.mortality.org

Figure 1: Historic development in female (top panel) and male (bottom panel) log death rates in Denmark from 1935 to 2004 for the age groups 20, 30, . . . , 100. The lines represent the age groups in decreasing order with the 100-year-olds at the top and the 20-year-olds at the bottom

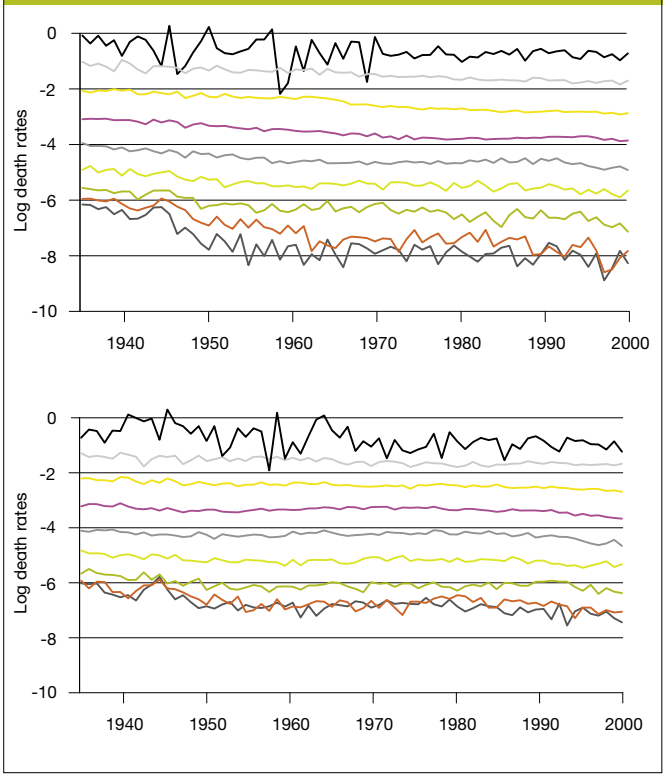
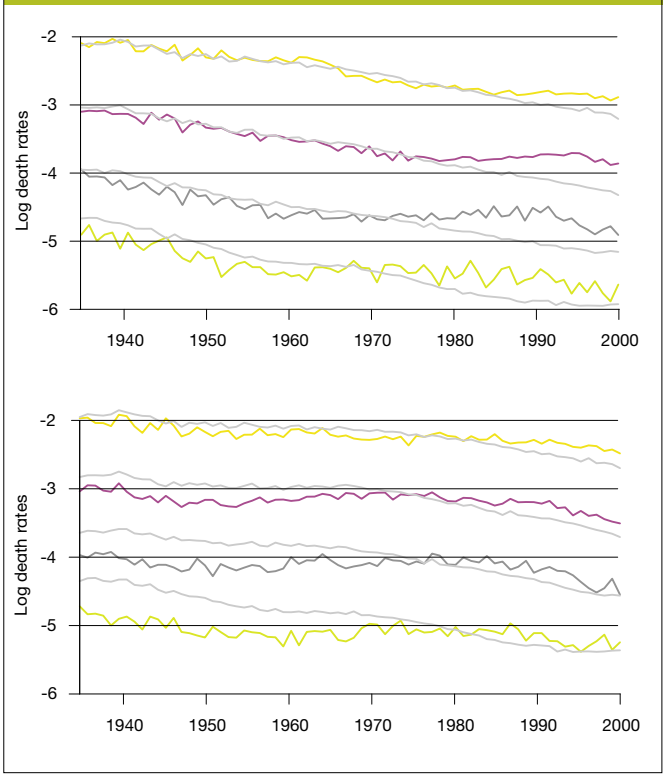


Figure 2. Danish and international development in female (top panel) and male (bottom panel) log death rates from 1935 to 2004 for the age groups 50, 60, 70 and 80. For all age groups Danish rates start below and end above the international rates.



The second period from 1950 to 1980, however, saw only very modest improvement rates for most age groups. For women the age group from 30 to 60 had annual rates of improvement of 0.7%, while the same age group for men had a slight *increase* in death rates over that period. Also men above 60 had virtually no improvements in mortality rates, while the same age group for women had sizeable improvements of about 1.5% per year.

For women the slow rates of improvements from 1950 to 1980 were reduced even further from 1980 to 1995. The age group from 30 to 60 had annual rates of improvements of 0.8%, while the age group above 60 had rates of improvement of just 0.1%. For men the corresponding rates were, respectively, 1.0% and 0.4%, thus only marginally higher.

Since 1995 improvements have picked up again and both sexes are enjoying historically high improvement rates. For men annual rates of improvement are about 2% for all age groups above 30, and for women rates are about 3.4% for ages from 30 to 60 and about 1.7% for ages above 60.

In summary, the younger age groups have had higher annual rates of improvements over the period than the older age groups, but rates of improvement for the older age groups have been increasing over the last 20 years.

Figure 2 shows the evolution in Danish age-specific death rates for ages 50, 60, 70 and 80 together with the same rates for the international data set. Two features in particular stand out. First, the evolution in international rates seems to have been much more steady than the Danish evolution. This is hardly surprising considering that the international data set consists of data from 18 different countries around the globe. Most national and even regional effects are likely to be smoothed out when pooled. The only feature from the Danish data which is also present in the pooled data is the lower pace of improvements from 1950

to 1980, although the slowdown is much less pronounced. Arguably, the increased rates of improvements among the oldest over the period is also visible in the international data, especially for men.

Second, the relative worsening of Danish death rates compared to international rates is striking (and, from a Danish perspective, alarming). Death rates for Danish females were about the same as the international level up to 1980. Over the next 15 years Danish female death rates stagnated while the international rates continued to decline and a sizeable gap was established. From 1995 onwards the gap has reduced somewhat but Danish women still suffer substantial excess mortality compared to the international level. Danish men started out having substantially lower death rates than the international level but their lead was eroded, in particular during the period from 1950 to 1980, and in 1980 they were at the international level. From 1980 Danish male death rates have improved but by less than the international rates such that Danish males today have a slight excess mortality. Thus, for both sexes Denmark has lost ground compared to the international development, but might be starting to catch up.

2.3. **Modelling framework.** Compared to Denmark the international development in death rates has been much more stable with near-constant annual rates of improvement. Denmark, on the other hand, has experienced alternate periods with high and almost no improvements. Still, over long horizons the development in Danish rates seems to follow that of the international community.

In our view a good mortality model must be able to quantity both the likely drift in mortality in the future and the uncertainty associated with the drift. In most prevailing models these two aims are achieved by the same time series model, often a random walk with drift. As a consequence, if data has shown large variability around a trend this translates into large uncertainty about the long-term trend.

However, although Denmark has had a much less stable development than the international community as a whole it is unlikely that the long-term trends will deviate from each other. Furthermore, one must also assume that there are limits to how far Danish rates can fall behind, or get ahead of, international rates.

With this in mind we propose a two-component modelling framework in which we separately model the underlying trend and deviations from it. We shall refer to the deviations as the spread. Schematically we have the structure

(2) Danish mortality = international trend + spread.

This is the structure which has lead to the name SAINT for Spread Adjusted INternational Trend. We implicitly assume that the spread will fluctuate around zero (or perhaps another fixed level), but that it does not have a trend component of its own. With this decomposition we can separate the uncertainty about the underlying long-term trend from (the uncertainty of) shorter term deviations. Thus we can allow substantial short-term variability and still have stable long-term behavior.

In the following we will develop a model which conforms with this general framework. Specifically, we will propose a parametric model for the trend based on frailty theory and a 3-dimensional VAR-model for the spread. Clearly many other models will fall into the proposed framework and enjoy the same long-term stability properties.

3. Frailty theory

3.1. **Notation.** In this section the object of study will be the time- and age-dependent gender-specific force of mortality, $\mu(t,x)$; for ease of readability we will leave out the sex-dependence from the notation. We shall also refer to μ as the mortality function or intensity. The mortality intensity represents the instantaneous rate of dying for a person aged x at time t, i.e. the probability that the person will die between time t and $t+dt$ is $\mu(t,x)dt$.

The survival function, $\bar{F}(t,x)$, denotes the proportion of the cohort aged x at time t still alive or, more mathematically, the probability that a person born at time $t-x$ is still alive at time t (at age x). In terms of the force of mortality the survival function is given by

$$\bar{F}(t,x) = e^{-\int_{t-x}^{t}\mu(u,u-t+x)du}. \tag{3}$$

Conversely,

$$\mu(t,x) = -\frac{d}{d\delta}\log\bar{F}(t+\delta,x+\delta)|_{\delta=0} \tag{4}$$

3.2. **Old age mortality.** Assume for now that the force of mortality is time-invariant, i.e. $\mu(t,x) = \mu(x)$. This is the starting point for classical mortality modelling and various parametric forms of μ have been proposed in this situation. Classical forms include the ones named after Gompertz

$$\mu(x) = \alpha e^{\beta x},$$

and Makeham

$$\mu(x) = \alpha e^{\beta x} + \gamma.$$

Although widely used there seems to be no underlying reason why these simple forms should describe human mortality. In fact, several studies indicate that the exponential form overestimate the mortality at high ages. Some empirical studies even find a mortality plateau at high ages at which the intensity essentially remains constant, others find a sub-exponential growth rate at high ages.

One theory offering some insight into the phenomenon of slower increase in the force of mortality at high ages is frailty theory. This theory assumes that the population is heterogeneous with each person having an individual level of susceptibility. As frail individuals are more likely to die first the composition of the population will change over time such that the fraction of robust individuals will increase. This selection mechanism causes the overall mortality intensity to be more and more influenced by the mortality intensity of the more robust individuals.

The following simple model illustrates the idea. Assume that the ith person of a population has his own Makeham intensity:

(5) $$\mu_i(x) = \mu(x; z_i) = z_i \alpha e^{\beta x} + \gamma,$$

where z_i is an individual frailty parameter, while α, β and γ are shared by all persons in the population. Assume furthermore that Z follows a scaled Γ-distribution with mean 1 and variance σ^2, i.e. its density is given by

(6) $$f(z) = \frac{\lambda^\lambda}{\Gamma(\lambda)} z^{\lambda-1} e^{-\lambda z}$$

where $\lambda = 1/\sigma^2$ is the shape parameter.

The individual survival function is

(7) $$\bar{F}(x; z_i) = e^{-\int_0^x \mu_i(u)du} = e^{-z_i \alpha \int_0^x e^{\beta u} du - x\gamma} = e^{-z_i \alpha (e^{\beta x} - 1)/\beta - x\gamma},$$

and the survival function at the population level thus becomes

$$\begin{aligned}
\bar{F}(x) &= \int_0^\infty \bar{F}(x; z) f(z) dz \\
&= \frac{\lambda^\lambda}{\Gamma(\lambda)} \int_0^\infty e^{-z\alpha(e^{\beta x} - 1)/\beta - x\gamma} z^{\lambda-1} e^{-\lambda z} dz \\
&= \frac{\lambda^\lambda}{\Gamma(\lambda)} e^{-x\gamma} \int_0^\infty e^{-z(\lambda + \alpha(e^{\beta x} - 1)/\beta)} z^{\lambda-1} dz \\
&= \frac{\lambda^\lambda}{(\lambda + \alpha(e^{\beta x} - 1)/\beta)^\lambda} e^{-x\gamma} \\
&= \left(\frac{1}{1 + \sigma^2 \alpha (e^{\beta x} - 1)/\beta} \right)^{1/\sigma^2} e^{-x\gamma}.
\end{aligned}$$

From this expression the force of mortality for the population can now be derived

(8) $$\mu(x) = -\frac{d}{dx} \log \bar{F}(x) = \frac{\alpha e^{\beta x}}{1 + \sigma^2 \alpha (e^{\beta x} - 1)/\beta} + \gamma.$$

Note that

(9) $$\mu(x) \to \frac{\beta}{\sigma^2} + \gamma \text{ for } x \to \infty.$$

Hence, although each individual intensity is exponentially increasing the selection mechanism is so strong that the population intensity has a finite asymptote. It is instructive to see how the level of heterogeneity, as measured by σ^2, affects the asymptotic level. If σ^2 is very large the first term in (9) essentially vanishes. In this case the population is very heterogeneous at birth and contains a sizeable fraction of very robust individuals, i.e. individuals with very low frailty and thus near-constant intensity $\mu_i(x) \approx \gamma$. The robust individuals will dominate the "limit population" and the limit value of the population intensity thus also becomes close to γ. If, on the other hand, σ^2 is small the fraction of very robust individuals is smaller and less dominating in the "limit population" and the asymptotic value of the intensity therefore higher. In the limit case with $\sigma^2 = 0$ the population is homogeneous and the intensity equals the individual intensity, $\mu(x) = \alpha \exp(\beta x) + \gamma$.

The model here described is the so-called logistic model or 'gamma-Makeham' model, which has been used by e.g. Thatcher (1999) to model old age mortality. The combination of individual mortality intensities of Makeham-form and Gamma-distributed frailties makes the model mathematically appealing and allows explicit calculations. However, the qualitative aspects of the model holds in more generality and under other assumptions about both the form of individual intensities and the shape of the frailty distribution.

3.3. **Frailty model with time-varying living conditions.** The logistic form (8) has been used as the basis for mortality models by letting some or all of the parameters be time-dependent. This approach has been shown to fit historic data well but the resulting intensity surface cannot be interpreted as the result of selection in a heterogeneous population. To achieve this interpretation one has to introduce the time-variation at the level of the individual intensities and carry out the calculations to get to the population intensity. This results in a time-varying population intensity with a more involved structure than the ones normally seen.

Below we will derive the population intensity for a general frailty model with time-varying living conditions. Assume that the mortality intensity for an individual with frailty z is given by

$$\mu(t,x,z) = z\alpha H\left(\int_{t-x}^{t} g(s,s-t+x)ds\right)\kappa(t,x) + \gamma(t,x), \tag{10}$$

for some increasing function H.

The idea is that the individual mortality intensity is affected by four components

(1) a frailty parameter affecting the general susceptibility of the individual;

(2) a "wear-out" index, $\int_{t-x}^{t} g\,ds$, which accumulates the general living conditions in the society during the period lived by the person. The rate $g(t,y)$ can be interpreted as the "rate" of aging at time t at age y. In this terminology, $\int_{t-x}^{t} g\,ds$ can be interpreted as the "biological" age of a person which in general will differ from his physical age, x;

(3) a time- and age-dependent effect, $\kappa(t,x)$, which can be interpreted as the current level of treatment and medical care;

(4) a time- and age-dependent effect, $\gamma(t,x)$, which can be interpreted as the current rate of accidents.

Note that letting $H(y) = e^{\beta y}$, $g(t,x) \equiv 1$, $\kappa(t,x) \equiv 1$ and $\gamma(t,x) \equiv \gamma$ we are back at the time-invariant, simple frailty model from the previous section.

The individual survival function is given by

$$\bar{F}(t,x;z) = e^{-\int_{t-x}^{t}\mu(u,u-t+x;z)du} = e^{-z\alpha I(t,x)-\int_{t-x}^{t}\gamma(u,u-t+x)du}, \tag{11}$$

where $I(t,x) = \int_{t-x}^{t} H\left(\int_{t-x}^{u} g(s,s-t+x)ds\right)\kappa(u,u-t+x)du$. Assuming frailties to follow a (scaled) Γ-distribution with mean 1 and variance σ^2 the survival function for the population becomes

$$\begin{aligned}\bar{F}(t,x) &= \int_0^\infty \bar{F}(t,x;z)f(z)dz \\ &= \frac{\lambda^\lambda}{\Gamma(\lambda)} e^{-\int_{t-x}^{t}\gamma(u,u-t+x)du}\int_0^\infty e^{-z(\lambda+\alpha I(t,x))}z^{\lambda-1}dz \\ &= \left(\frac{1}{1+\sigma^2\alpha I(t,x)}\right)^{1/\sigma^2} e^{-\int_{t-x}^{t}\gamma(u,u-t+x)du},\end{aligned}$$

and hence

$$\begin{aligned}\mu(t,x) &= -\frac{d}{d\delta}\log\bar{F}(t+\delta,x+\delta)|_{\delta=0} \\ &= \frac{d}{d\delta}\left(\frac{1}{\sigma^2}\log\left(1+\sigma^2\alpha I(t+\delta,x+\delta)\right) + \int_{t-x}^{t+\delta}\gamma(u,u-t+x)du\right)|_{\delta=0} \\ &= \frac{\alpha H\left(\int_{t-x}^{t} g(s,s-t+x)ds\right)\kappa(t,x)}{1+\sigma^2\alpha I(t,x)} + \gamma(t,x). \qquad (12)\end{aligned}$$

Example. With $H(y) = e^{\beta y}$, $g \equiv 1$ and $\kappa(t) = e^{c(t-t_0)}$ one gets

$$\mu(t,x) = \frac{\alpha e^{\beta x}\kappa(t)}{1+\sigma^2\alpha\kappa(t)(e^{\beta x}-e^{-cx})/(\beta+c)} + \gamma(t,x).$$

3.4. **Computation**. The expression in (12) is hard to evaluate analytically for general specifications of g and κ. To facilitate computation we will therefore assume that g and κ are constant on "integer" squares, i.e. g is assumed to take the form

$$g(t,x) = g(i,k) \text{ for } t \in [i,i+1), x \in [k,k+1), i,k \in \mathbb{N},$$

and likewise for κ. Under this assumption integrals over g are piecewise linear functions which (for most specifications of H) makes it possible to compute I, and hence μ, explicitly.

For $H(y) = e^{\beta y}$ we find for integer values of t and x, and $u \geq t-x$:

$$H\left(\int_{t-x}^{u} g(s,s-t+x)ds\right) = e^{\beta\sum_{i=t-x}^{\lfloor u\rfloor-1} g(i,i-t+x)+\beta g(\lfloor u\rfloor,\lfloor u\rfloor-t+x)(u-\lfloor u\rfloor)},$$

where $\lfloor u\rfloor$ denotes the largest integer smaller than or equal to u. Inserting this in the expression for $I(t,x)$ yields

$$
\begin{aligned}
I(t,x) &= \int_{t-x}^{t} H\left(\int_{t-x}^{u} g(s,s-t+x)ds\right)\kappa(u,u-t+x)du \\
&= \sum_{j=t-x}^{t-1}\int_{j}^{j+1} H\left(\int_{t-x}^{u} g(s,s-t+x)ds\right)\kappa(u,u-t+x)du \\
&= \sum_{j=t-x}^{t-1}\int_{j}^{j+1} e^{\beta\sum_{i=t-x}^{\lfloor u\rfloor-1} g(i,i-t+x)+\beta g(\lfloor u\rfloor,\lfloor u\rfloor-t+x)(u-\lfloor u\rfloor)}\kappa(u,u-t+x)du \\
&= \sum_{j=t-x}^{t-1} e^{\beta\sum_{i=t-x}^{j-1} g(i,i-t+x)}\kappa(j,j-t+x)\int_{j}^{j+1} e^{\beta g(j,j-t+x)(u-j)}du \\
&= \sum_{j=t-x}^{t-1} e^{\beta\sum_{i=t-x}^{j-1} g(i,i-t+x)}\kappa(j,j-t+x)\frac{e^{\beta g(j,j-t+x)}-1}{\beta g(j,j-t+x)}.
\end{aligned}
$$

For integer values of t and x the intensity can thereby be written as

$$
\mu(t,x) = \frac{S(t-x,t)}{1+\sigma^2\sum_{j=t-x}^{t-1} S(t-x,j)\frac{e^{\beta g(j,j-t+x)}-1}{\beta g(j,j-t+x)}} + \gamma(t,x), \tag{13}
$$

where

$$
S(t-x,j) = \alpha e^{\beta\sum_{i=t-x}^{j-1} g(i,i-t+x)}\kappa(j,j-t+x), \quad j = t-x,\ldots
$$

It is also possible to write down an expression valid for all t and x, but it becomes rather messy. The general expression has a number of "edge-effect" terms arising from the misalignment of the integrals with the intervals over which g and κ are assumed constant.

4. Trend modelling

4.1. **Likelihood function.** As previously announced we will use the international data set to estimate the underlying intensity surface. For a given specification of μ we will assume that the $D_{Int}(t,x)$ are independent with

$$
D_{Int}(t,x) \sim \text{Poisson}\left(\bar{\mu}(t,x)E_{Int}(t,x)\right), \tag{14}
$$

where $\bar{\mu}(t,x)$ is the average value of μ over the square $[t,t+1)\times[x,x+1)$. As an approximation to this average we will use

$$
\bar{\mu}(t,x) = \frac{1}{4}\left(\mu(t,x)+\mu(t,x+1)+\mu(t+1,x)+\mu(t+1,x+1)\right). \tag{15}
$$

Assuming a parametric form of g, κ and γ and collecting all parameters (including α, β and σ^2) in the vector θ the log-likelihood function becomes

$$
\begin{aligned}
l(\theta) &= \sum_{t,x} D(t,x)\log(\bar{\mu}(t,x)E(t,x)) - \log(D(t,x)!) - \bar{\mu}(t,x)E(t,x) \\
&= \sum_{t,x} D(t,x)\log(\bar{\mu}(t,x)) - \bar{\mu}(t,x)E(t,x) + constant, \qquad (16)
\end{aligned}
$$

where the last term does not depend on θ.

4.2. **Model**. The following nine parameter model has been selected (among a large number of candidate models) for its ability to fit the historic development in mortality in the international data with a relatively parsimonious structure. In particular, attention has been paid to the model's ability to capture the mortality of the 60+ years old as this part of the mortality surface has the largest potential for future improvement. We have not applied any formal tests in the model selection.

The model takes the form

$$
\begin{aligned}
\alpha &= 1 \\
\beta &= 1 \\
\sigma &= \theta_1 \\
g(t,x) &= \theta_2 + \theta_3 x + \theta_4(t-2000) \\
\kappa(t,x) &= \exp(\theta_5 + \theta_6 x + \theta_7(t-2000)) \\
\gamma(t) &= \exp(\theta_8 + \theta_9(t-2000))
\end{aligned}
$$

The substraction of (year) 2000 in the specification of g, κ and γ is for done interpretability reasons only. Thus $g(2000,0) = \theta_2$ is the "aging" of a newborn in year 2000, $\theta_3 x$ is the additional biological aging at age x, and θ_4 is the additional aging across ages for each calender year relative to year 2000. Similar interpretations can be given to the parameters in κ and γ.

4.3. **Estimation.** Table 1 contains maximum likelihood estimates and corresponding 95% confidence intervals for the model given above for women and men separately. Figure 3 illustrates the historic development in mortality and the fitted trend.

The model has three parameters $(\theta_4,\theta_7,\theta_9)$ to describe three different types of improvement in mortality over time. The main effect is carried by θ_7 which represents the annual improvement in mortality. This improvement is estimated to be about 1.8% for both women and men.

The improvement in "accidents" (θ_9) is estimated to about 9% for women. This is due to the dramatic decrease among the 20 to 30 years old in the beginning of the observation period, cf. top panel of Figure 3. However, since the accident level (θ_8) is already very low the high improvement rate will have virtually no effect on future mortality. For men, on the other hand, the accident level is not yet negligible and consequently the estimated improvement of about 2.5% per year will have some effect on the projected mortality at young ages.

Figure 3: Historic development in international female (top panel) and male (bottom panel) log mortality from 1935 to 2004 for the age groups 20, 30, . . . , 100. The lines represent the age groups in decreasing order with the 100-year-olds at the top and the 20-year-olds at the bottom. Model estimate of trend with parameters given in Table 1 is superimposed.

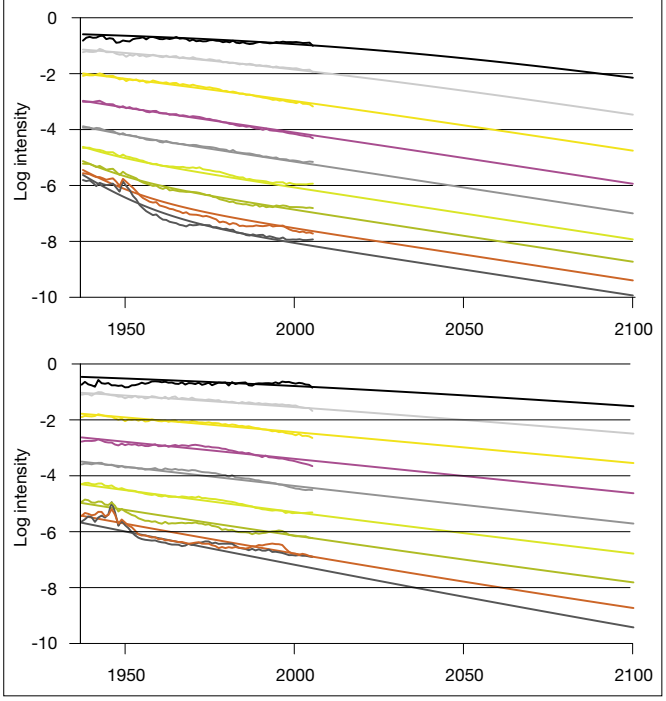


Table 1. Maximum likelihood estimates and 95% confidence intervals for the model given in Section 4.2. The estimation is based on international mortality data from 1935 to 2004 for ages 20 to 100 years.

Parameter	Women		Men	
	Estimate	95%-CI	Estimate	95%-CI
θ_1	$4.2805 \cdot 10^{-1}$	$\pm 3.4 \cdot 10^{-4}$	$2.7140 \cdot 10^{-1}$	$\pm 4.8 \cdot 10^{-4}$
θ_2	$2.7148 \cdot 10^{-2}$	$\pm 4.8 \cdot 10^{-6}$	$9.1032 \cdot 10^{-2}$	$\pm 4.8 \cdot 10^{-6}$
θ_3	$1.3046 \cdot 10^{-3}$	$\pm 1.2 \cdot 10^{-7}$	$6.2340 \cdot 10^{-5}$	$\pm 1.2 \cdot 10^{-7}$
θ_4	$-2.4000 \cdot 10^{-6}$	$\pm 7.6 \cdot 10^{-8}$	$8.1353 \cdot 10^{-5}$	$\pm 7.2 \cdot 10^{-8}$
θ_5	$-8.7838 \cdot 10^{0}$	$\pm 3.6 \cdot 10^{-4}$	$-1.0573 \cdot 10^{1}$	$\pm 3.4 \cdot 10^{-4}$
θ_6	$-6.1950 \cdot 10^{-3}$	$\pm 4.8 \cdot 10^{-6}$	$1.0912 \cdot 10^{-2}$	$\pm 4.8 \cdot 10^{-6}$
θ_7	$-1.8087 \cdot 10^{-2}$	$\pm 1.2 \cdot 10^{-5}$	$-1.7830 \cdot 10^{-2}$	$\pm 1.1 \cdot 10^{-5}$
θ_8	$-1.1803 \cdot 10^{1}$	$\pm 4.0 \cdot 10^{-3}$	$-7.5328 \cdot 10^{0}$	$\pm 1.8 \cdot 10^{-3}$
θ_9	$-9.0713 \cdot 10^{-2}$	$\pm 7.0 \cdot 10^{-5}$	$-2.5446 \cdot 10^{-2}$	$\pm 4.4 \cdot 10^{-5}$

Finally, θ_4 describes the annual improvement in "biological" aging. This effect is very small for women and only slightly larger for men. Interestingly, θ_4 is positive for men indicating a slight acceleration in aging, which however is dwarfed by the much larger general improvement.

The frailty parameter, θ_1, is relatively large for both women and men. This parameter controls the flatness of the mortality surface at high ages and the estimated level reflects the fact that the 80+ years old have not (yet) had the same rate of improvement as the younger age groups. However, as can be seen from the curved projections in Figure 3 the model predicts these age group to have larger improvements in the future.

The remaining parameters are shape parameters which are estimated to provide a good fit to the age profile of mortality.

The confidence intervals provide in Table 1 are calculated by a chi-square approximation to the log-likelihood function in (16). For a given parameter the interval represents the range of values which can be accepted (all other parameters kept fixed) in a test with a level of significance of 5%. The ranges are in all cases very narrow as one would expect in the current situation with a parsimonious model and a wealth of data. One could introduce stochastic components at the trend model level to better reflect the level of uncertainty in the projections. Here, however, we will take a simpler approach and place all stochastic components in the spread model.

5. Spread modelling

The real object of interest is Danish mortality which will be modelled as the international trend with an additional spread. The fundamental premise is that Danish mortality in the long run will develop similarly to the international trend but there will be short- to medium-term deviations. Thus we assumed that the spread will fluctuate around zero.

5.1. **Model.** Given the international trend, μ, we will assume that the $D_{DK}(t,x)$s are independent with

$$D_{DK}(t,x) \sim \text{Poisson}\left(\bar{\mu}_{DK}(t,x)E_{DK}(t,x)\right), \tag{17}$$

where

$$\begin{aligned} \bar{\mu}_{DK}(t,x) &= \bar{\mu}(t,x)\exp(a_t + b_t r_1(x) + c_t r_2(x)), \\ r_1(x) &= (x-60)/40, \\ r_2(x) &= (x^2 - 120x + 9160/3)/1000, \end{aligned} \tag{18}$$

with μ given by (15). The parametrization of the spread ensures that a_t can be interpreted as the level of excess mortality in Denmark compared to the international level, while b_t and c_t are higher order corrections to give a closer fit to the observed mortality structure in Denmark. The regressors, r_1 and r_2, are chosen to be orthogonal and normalized to (about) 1 at age 20 and 100.

The three-dimensional time series of spread parameters, $(a_t,\ b_t,\ c_t)$, is modelled as a VAR-process

$$\text{(19)} \qquad \begin{pmatrix} a_t \\ b_t \\ c_t \end{pmatrix} = A \begin{pmatrix} a_{t-1} \\ b_{t-1} \\ c_{t-1} \end{pmatrix} + e_t,$$

where A is a three by three matrix of autoregression parameters and the e_t's are three-dimensional i.i.d. normally distributed variates with covariance matrix Ω.

5.2. **Estimation.** In principle the VAR-parameters can be estimated directly by treating the spread parameters as so-called hidden variables. However, to ensure a simple and robust estimation procedure we will first treat the three-dimensional time series of spread parameters, $(a_t,\ b_t,\ c_t)$, as free parameters and estimate these from model (17). Note that the estimate of $(a_t,\ b_t,\ c_t)$ only depends on the (population) mortality for year t. Second, we will estimate the VAR-parameters by treating (the estimate of) the spread parameters as observations.

For women the model estimates are

$$\text{(20)} \qquad \begin{pmatrix} a_t \\ b_t \\ c_t \end{pmatrix} = \begin{pmatrix} 0.6670 & -0.2083 & -0.2827 \\ -0.1555 & 0.8549 & -0.1541 \\ -0.1651 & -0.1093 & 0.8208 \end{pmatrix} \begin{pmatrix} a_{t-1} \\ b_{t-1} \\ c_{t-1} \end{pmatrix} + e_t,$$

with

$$e_t \sim N_3(0, \begin{pmatrix} 0.001397 & -0.000895 & 0.000467 \\ -0.000895 & 0.003761 & -0.000873 \\ 0.000467 & -0.000873 & 0.001508 \end{pmatrix}),$$

and for men we get

$$\text{(21)} \qquad \begin{pmatrix} a_t \\ b_t \\ c_t \end{pmatrix} = \begin{pmatrix} 0.8211 & -0.1091 & -0.1338 \\ -0.0788 & 0.9114 & 0.0467 \\ -0.0532 & -0.0532 & 0.9081 \end{pmatrix} \begin{pmatrix} a_{t-1} \\ b_{t-1} \\ c_{t-1} \end{pmatrix} + e_t,$$

with

$$e_t \sim N_3(0, \begin{pmatrix} 0.002233 & -0.001717 & -0.000058 \\ -0.001717 & 0.005059 & -0.000001 \\ -0.000058 & -0.000001 & 0.003109 \end{pmatrix}).$$

5.3. **Forecasting and confidence intervals.** Forecasting in the VAR-model (19) is based on the conditional distribution of $(a_{t+h}, b_{t+h}, c_{t+h})$ given (a_t, b_t, c_t).

From the data generating equation and its expansion

$$\begin{aligned} \begin{pmatrix} a_{t+h} \\ b_{t+h} \\ c_{t+h} \end{pmatrix} &= A \begin{pmatrix} a_{t+h-1} \\ b_{t+h-1} \\ c_{t+h-1} \end{pmatrix} + e_{t+h} \\ &= A^h \begin{pmatrix} a_t \\ b_t \\ c_t \end{pmatrix} + A^{h-1} e_{t+1} + \ldots + e_{t+h} \end{aligned}$$

we obtain

$$\begin{pmatrix} a_{t+h} \\ b_{t+h} \\ c_{t+h} \end{pmatrix} \mid \begin{pmatrix} a_t \\ b_t \\ c_t \end{pmatrix} \sim N(m_h, V_h),$$

where m_h and V_h are given by either the recursive expressions

$$\begin{aligned} m_h &= A m_{h-1}, \quad m_0 = \begin{pmatrix} a_t \\ b_t \\ c_t \end{pmatrix} \\ V_h &= A V_{h-1} A^t + \Omega, \quad V_0 = 0, \end{aligned}$$

or the closed-form expressions

$$m_h = A^h \begin{pmatrix} a_t \\ b_t \\ c_t \end{pmatrix}, \quad V_h = \sum_{i=0}^{h-1} A^i \Omega (A^i)^t.$$

From these expressions forecasted values and corresponding (pointwise) 95%-confidence intervals are easily obtained as

$$\begin{pmatrix} \hat{a}_{t+h} \\ \hat{b}_{t+h} \\ \hat{c}_{t+h} \end{pmatrix} \mid \begin{pmatrix} a_t \\ b_t \\ c_t \end{pmatrix} = m_h \pm 1.96 \sqrt{\operatorname{diag}(V_h)}.$$

Figures 4 and 5 show the estimated spread parameters and their forecasted value with confidence intervals for women and men.

The current level of excess mortality among Danish women compared to the international level is about 20% and this is forecasted to gradually fall back to zero over the next 40 years. Danish men, on the other hand, is very much in line with the international level. The confidence intervals are rather wide reflecting the observed variation in the spread over the estimation period.

Given a forecast of the spread, a forecast of Danish mortality is readily obtained from (18). Figure 6 shows fitted and forecasted Danish mortality as well as the estimated international trend. For women the Danish and international levels are currently quite far apart, while for men the two levels are almost identical.

Figure 4: Estimated and forecasted spread parameters for women with pointwise 95% confidence intervals.

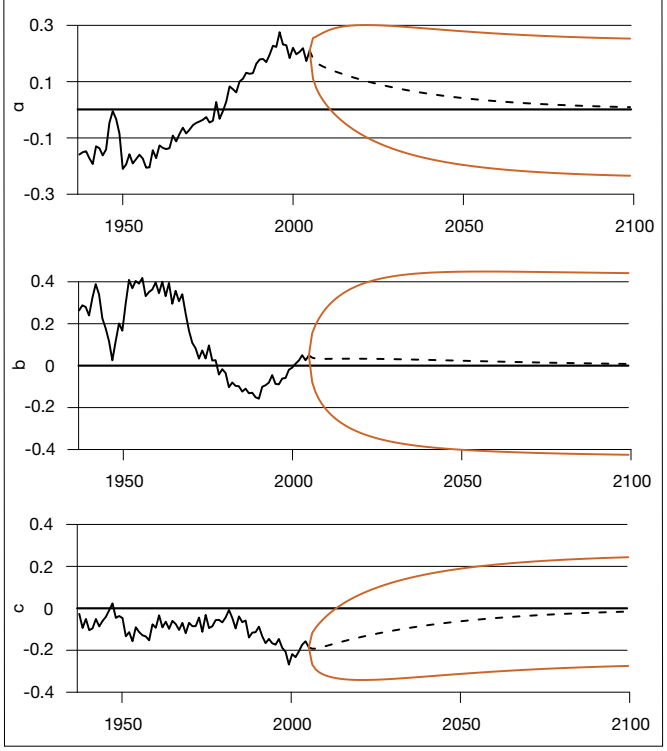


Figure 5. Estimated and forecasted spread parameters for men with pointwise 95% confidence intervals.

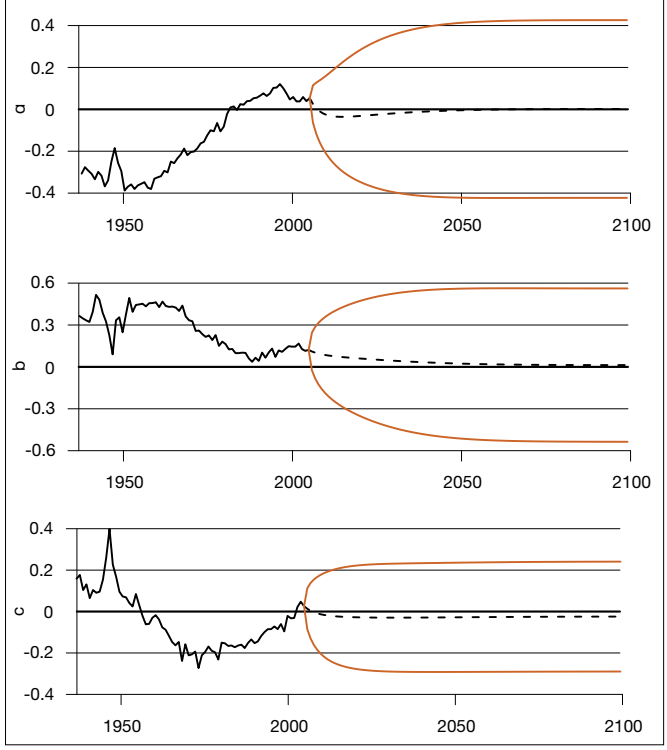
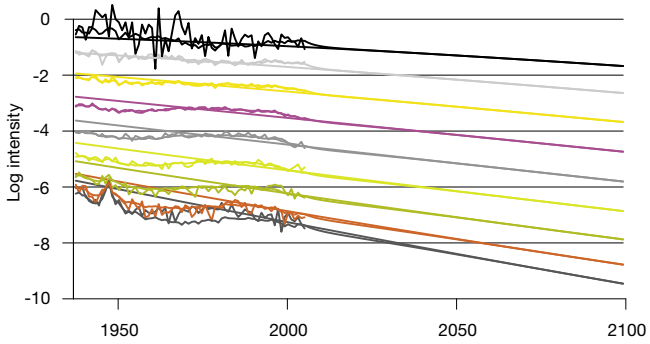


Figure 6. Historic development in female (top panel) and male (bottom panel) log mortality in Denmark from 1935 to 2004 for the age groups 20, 30, . . . , 100. The lines represent the age groups in decreasing order with the 100-year-olds at the top and the 20-year-olds at the bottom. Model estimate of both the international and the Danish level is superimposed.



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ATP, The Danish Labour Market
Supplementary Pension Fund
Kongens Vænge 8
DK-3400 Hillerød

Telephone +45 70 11 12 13
Telephone +45 48 20 37 34
sj@atp.dk
www.atp.dk