Discussion Papers
Department of Economics
University of Copenhagen

No. 15-16

Modeling and projecting mortality
A new model of heterogeneity and selection in survivorship

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ISSN: 1601-2461 (E)

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2015-09-22

Modeling and projecting mortality[+]

A new model of heterogeneity and selection in survivorship

By

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+) An earlier version of this research was presented at Symposium on Applied Statistics, Technical University of Denmark (DTU), January 26, 2015

Abstract

The demographic and epidemiological literature offers abundant examples of a range of shortcomings of statistical modeling to describe mortality by sex, age, time/cohort, and cause-of-death. Statistical modeling of mortality operating with implicitly homogenous sub-groupings exposed to mortality risk fails to consider latent biological heterogeneity at the level of individuals, and thereby important biological and social selection of survivorship. Defined on the state space of the simple life model, this study presents a proportional hazard model that makes up for such drawbacks as far as latent biological heterogeneity is concerned. The model describes heterogeneity and selection in individual survivorship by iterative stochastic micro simulation using cohort-based population mortality as an empirical benchmark. The model offers efficient linkage between past assorted mortality, on one hand, and informed anticipation of future heterogeneous survivorship, on the other hand. The combination of stochastic micro-simulation and log-linear modeling of the period effect or trend uncovered under the model makes the new Heterogeneity and Selection Model a powerful analytic and predictive tool of survivorship. Postulating a trend independent of age makes the popular Lee-Carter model (1992) unfit for professional demographic and actuarial use. Moreover, by sweeping latent biological heterogeneity under the rug, mortality analysis and projection based on central rates such as the Lee-Carter model (1992) underrates mortality in the mature and elderly ages. This is demonstrated by comparing current official mortality projections of Sweden, Denmark, and England & Wales to a set of alternative mortality projections under the Heterogeneity and Selection Model.

Keywords: *biodemography, heterogeneity and selection, stochastic micro-simulation, projection of survivorship*

JEL J1, J11, J110, J14, J17

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Introduction

Living organisms interact with environment in continuous time. The interaction involves adaptation and quest for survival. Personal human genetics and biological aging makes individuals special and groupings of people heterogeneous. Furthermore, never ceasing exposure to the aggregate physical, social and cultural environment renders waiting time to failure (death) differential across persons; which, again, distorts the genetic and non-biological composition of groupings of survivors in the course of age and time. The pruning of lives before a certain age is called *selection*; persons surviving a given age are said to be *select*. Human societies have an obvious interest in coherent inference on survivorship and social allocation of life time – time is money.

Personal genetics constituting vitality and biological attrition of cells with age are real, though normally unobservable phenomena embedded in data on time of failure (age-at-death). Starting off from given time series of empirical mortality, recovery of the latent biological agents and their interaction with the physical and social environment is certainly not straightforward. Coherence and transparency of argument call for statistical modeling.

Statistical modeling of mortality in demographic and actuarial settings has developed over a long period of time. Well-known early modern examples are John Graunt (1662), Benjamin Gompertz (1825), and William Makeham (1860). For reviews of the literature cf. for example Olshansky, Carnes, Casel (1990), Olshansky and Carnes (1997), Booth and Tickle (2008), Olshansky et al. (2011), and Hatzopoulos and Haberman (2015). The uttering “But he doesn’t have anything on!” of the small child in “The emperor’s new clothes” (Andersen 1837) comes to mind at this point. Despite plenty of examples of

impressive technical showoff ranging from singular value decomposition (SVD) of matrices (Lee & Carter 1992), random walk with drift (Girosi & King 2007, Haldrup et al. 2014) to modeling individual failure times as first-passage times in discrete space Markov chains and continuous space diffusion processes (Aalen 1994, Aalen & Gjessing 2001, Li et. al. 2009, 2011, 2013a-b), none of the analysts, quite surprisingly, considers modeling of mortality as a function of biological heterogeneity and human interaction with environment.

In my view, survivorship modeling should - as a minimum – acknowledge and respect the following undeniable facts:

a. Humans are genetically different.

b. Human survivorship is constituted by interacting biological and environmental factors.

c. By molecular biological attrition of cells, the human survival potential diminishes with age.

d. Genetic heterogeneity governs selection in survivorship.

Part I of this study discusses and compares a new hazard model to the mortality models of Vaupel et al. (1979), Gavrilov (1991, 2001), Li et al. (2009, 2013a-b), and Lee & Carter (1992).

In addition to consistent recovery of the micro-foundation of historical population mortality the Heterogeneity and Selection Model enables autonomous anticipation of mortality by recovery of individual failure times of existing projections e.g. current official mortality projections.

Part II presents an application of the model featuring trend analysis of the latent period effect 1960-2011 recovered under the Heterogeneity and Selection Model from cohort-based population mortality 1901-2011; and recovered from current official mortality projections of Denmark, Sweden, and England & Wales; to varying degrees produced under the Lee-Carter model (1992). The popular Lee-Carter approach is blind to latent biological heterogeneity and undervalues mortality in the mature and elderly ages by leaving latent heterogeneity and hidden selection aside like other mortality models based on central demographic rates. This is demonstrated by comparing current official mortality projections of Sweden and England & Wales with projections under the new Heterogeneity and Selection Model. The Swedish and English official mortality projections exhibit a natural and rather sober extension of cohort-based historical mortality development. The historical support of the optimism regarding anticipated development of the official Danish

projection is less persuasive. Based on mediocre analysis of the historical mortality trend and inconsiderate use of the Lee-Carter approach, as it seems, rather than on professional demographic analysis and argument, the Danish projection, in particular, is overly optimistic; with negative impact for the official population projection; and for the derived statutory baseline mortality of life insurance (Forsikringstilsynet c. 2014).

Part I

Presentation of the Heterogeneity and Selection Model

1 Problem and purpose

Mortality $m_i(x,t)$ alias intensity or instant probability of death defined on state space $S=\{\text{alive, dead}\}$ is the centerpiece of analytical interest in the present context. With x indicating age, x_i age at failure (death) of person i, and t time, the probability that person i born at time t_0 will survive at least to time $t=t_0+x$ is then given by,

$$p_i\big((0,t_0),(x,t)\big)=\exp\left(-\int_{u=0}^{x} m_i(u,t_0+u)\,du\right) \qquad (1)$$

At population level the joint probability of surviving at least to (x,t) if born at t_0 is,

$$\begin{aligned} p\big((0,t_0);(x,\mathrm{t})\big) &= \prod_{\forall i} p_i\big((0,\mathrm{t}_0);(x,t)\big) \\ &= \exp\left(-\sum_{\forall i}\left(\int_{u=0}^{x} m_i(u,t_0+u)\,du\right)\right) \end{aligned} \qquad (2)$$

Needless to say, the probabilities are conditional on live birth.

This study proposes a hazard model emphasizing observable population mortality as a weighted sum of individual proportional mortality featuring a probability distribution of latent genetically related frailty alias vitality on conception; a function of biological attrition of cells independent of sex; and a period effect as a function of time and age. Lack of data and hidden heterogeneity makes classical parameter estimation such as maximum likelihood approaches inappropriate. As a powerful alternative I resort to quantification off the beaten track by iterative stochastic micro-simulation with cohort-based population mortality as empirical benchmark.

2 Modeling and issues of quantification

With x indicating age, x_i age at failure (death) of person i at time t, and *emp* representing empirical and *mod* modeled mortality I search for a model that minimizes the squared deviation of modeled from empirical population mortality i.e. a model subject to the following restraint.

$$Min\left\{\left(m(x,\mathrm{t},emp)-m(x,\mathrm{t},\mathrm{mod})\right)^2\right\}, x\in\left[0,\max(x_i)\right] \qquad (3)$$

The issue involves definition of notions, methodological approaches, and availability of failure data. Since people differ from one another, biologically and because of unique lifelong personal interaction with the physical and social environment, modeling and demographic interpretation of health and survivorship obviously should be at the level of individuals. Modeled population mortality can be established by individual failure times using standard approaches known from elemental demography (Hansen 2009) and introductory theoretical statistics. To approach the modeling issue let us first introduce state space $V=\{\text{alive, dead by cause } j\}$, $j=endo,exo$ of the competing risks model with terms *endo* referring to endogenous or intrinsic biological mortality and *exo* to exogenous or extrinsic mortality instigated by environmental non-biological causes (Bourgeois-Pichat 1951a-b).

2. Modeling heterogeneity and selection in survivorship

2.1. The contribution by Vaupel, Manton and Stallard (1979)

Vaupel, Manton and Stallard (1979) model individual mortality as $z_i\mu(x)$, z_i denoting a gamma distributed personal frailty and $\mu(x)=\mu(x,z=1)$ representing a chosen standard mortality. The model implies that the probability $\ell_i(x,z_i)$ that individual i survives at least to age x is equal to $\left(\ell_i(x)\right)^{z_i}$. Indicating genetically conditioned vitality, the notion of biological *frailty* appears to have been introduced in the biological literature long before the application by Vaupel, Manton and Stallard (1979) (Oral communication by L.A. Gavrilov, Estonia 2012). The authors' use of mixed statististical distributions along with the flexible gamma density to describe latent biological heterogeneity was probably among

the first specific demographic applications of well-established statistical techniques of old vintage. The authors' application targets *individual stationary mortality* leaving aside biological aging and environmental influence upon mortality; and hence as a matter of fact selection in survivorship. Despite such shortcomings and notwithstanding the fact that the model does not appear to have ever been properly tested against data, the demographic application by Vaupel, Manton and Stallard (1979) has attracted widespread attention among population analysts; nearly always operating at population level and commonly on cross-sections of cohorts like Vaupel, Manton and Stallard (1979).

2.2. Age-parity-cohort models

Equation (2) links individual survivorship to population mortality. Assuming that population mortality $m(x,t) = m(x+\tau, t+\tau), \tau \in [0,t[$ is a piecewise constant log linear function of an age effect *A*, a period effect *P*, and a cohort effect *C*, we have the socalled Age-Parity-Cohort or APC model; cf. for example Carstensen & Keiding (2005), O'Brien et al. (2008)), and Carstensen (2011). For a comprehensive historical review of APC-modeling cf. Hobcraft et al. (1982). As *period=cohort+age*, the model is over-parameterized which rules out unique identification of effects. More importantly, still, the implied assumption of cohort homogeneity is unrealistic. Under this model a *cohort* is tacitly perceived as a group of biologically homogeneous (cloned) individuals.

2.3. Competing risk modeling versus life modeling with environmental interaction

Let total population mortality $m_i(z_i, x, t)$ of individual i be a mixture of an endogenous effect $m_i(z_i, x, t, \text{endo})$ and an exogenous effect $m_i(z_i, x, t, \text{exo})$. How are the two effects related? Which operator should be chosen? If the two effects are additive and thereby statically independent we have, both at levels of individuals and population,

$$\ell(x) = \prod_j \ell^j(x), \text{ with } \ell^j(x=0) = 1, j = \text{endo, exo} \qquad (4)$$

This version is the competing risks model proposed by Li et al. (2009, eq. 1; 2013a-b) and in fact by Bourgeois-Pichat (1951).

If, on the other hand, the factors are multiplicative or proportional then, more realistically, we have interaction in continuous time between endogenous alias intrinsic biological mortality and a period effect with concerted multiplicative impact upon survivorship; reflecting the fact that no individual or population exists out of the thin air: there must be a material basis and hence a certain level of technology, a social organization, and a material and spiritual culture, altogether constituting the period effect. At population level the period effect translates into a trend effect.

2.4. A model of heterogeneity and selection in survivorship

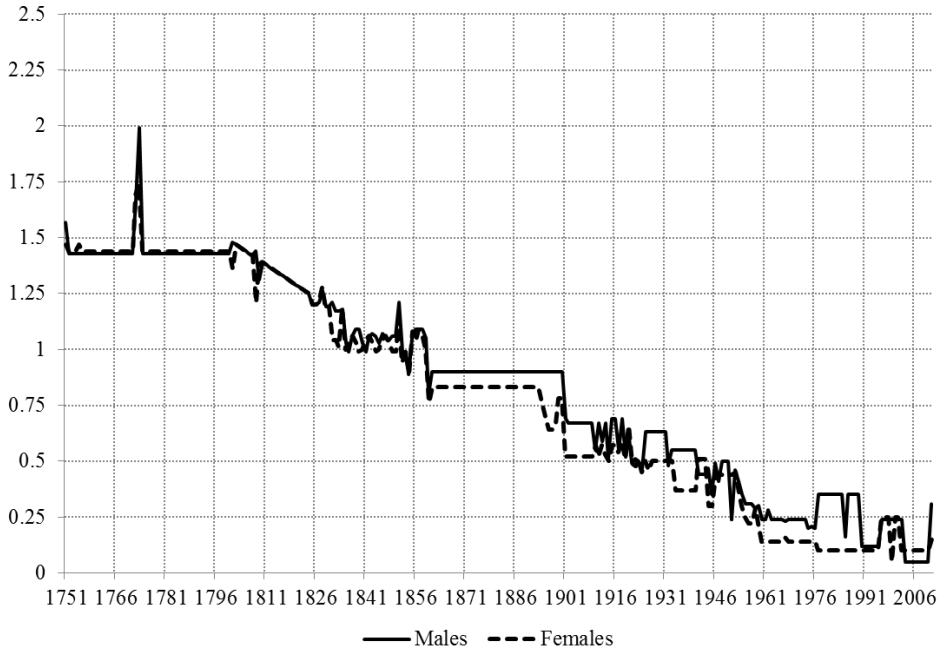
Keeping *sex* as a background variable, I model *individual mortality* $m_i(z_i, x, t)$ as a function of genetically conditioned vitality in terms of biological *frailty* z_i; of monotonously increasing biological *aging* $\eta(x)$; and of a mortality modifying external *period effect* $\varphi(x,t)$ linking individual mortality $m_i(z_i, x, t)$ to population mortality $m(x,t)$. Putting it all together while respecting the aforementioned irrefutable facts (cf. Introduction) and noting the shortcoming of the competing risks model to describe interaction, I propose the following proportional hazard model:

$$m_i(x,t) = z_i \eta(x) \varphi(x,t) \qquad (4)$$

The model describes interaction in continuous time between individual biological survivorship and environment. I shall assume that all individuals share the same function of biological aging. Conditional on *sex* the period or trend effect also depends on age. Contrary to frailty and biological aging the period effect is contingent on time and cleansed of latent biological heterogeneity under the proportional hazard model.

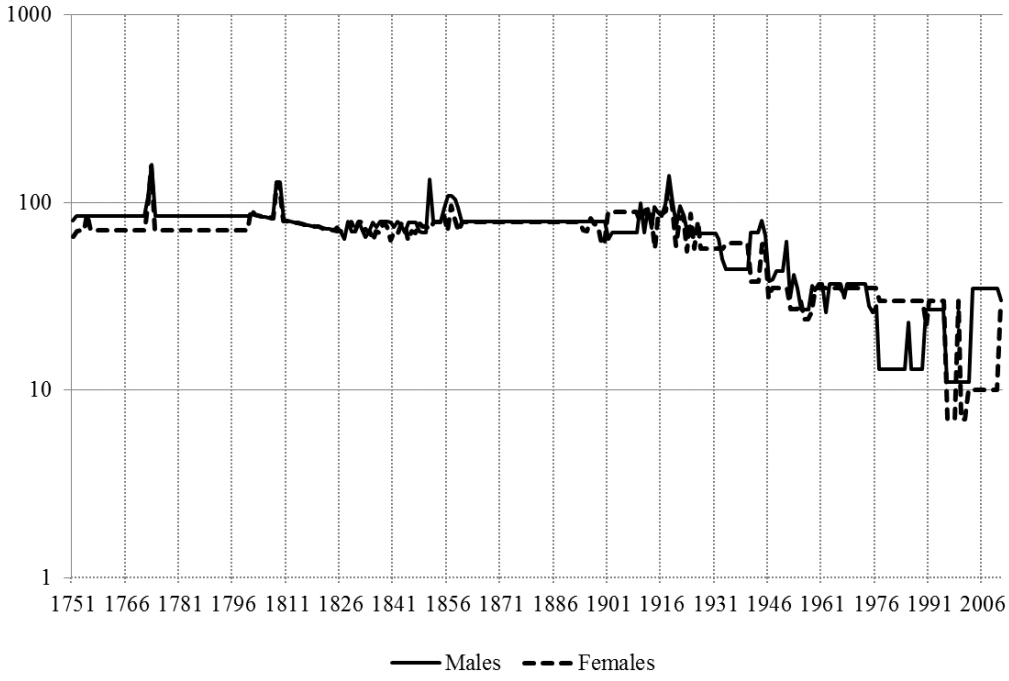
Linking to the aforementioned competing risks modeling (Section I.2.3), biological aging $\eta(x)$ represents intrinsic mortality $m_i(x,t,endo)$. Equation 4 sees $\varphi(x,t)$ as a multiplicative scaling of intrinsic biological mortality rather than as an independent extrinsic death risk; hence $\varphi(x,t) \neq m_i(x,t,exo)$. Formally, $\varphi(x,t)$ may be interpreted as a parameter that scales intrinsic mortality to external influence upon mortality across age and time. Under the Heterogeneity and Selection Model exposure to continuous external impact upon survivorship is hypothesized joint for all individuals.

Figure 1. Uncovered shape (α) parameter values of gamma distributed frailties. By sex



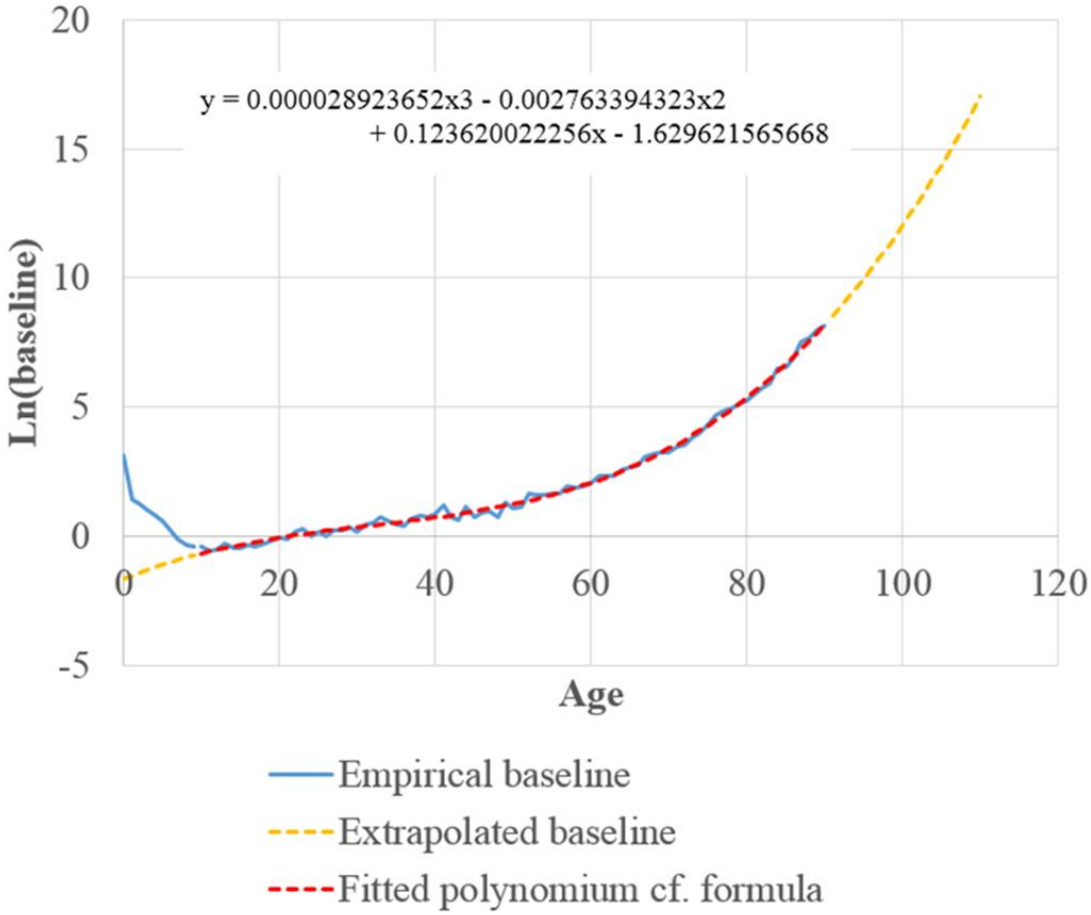
1) Obtained by minimizing the deviation of modeled from observed cohort-based population mortality. Swedish cohorts born between 1751 and 2011

Figure 2. Uncovered scale parameter (β) values of gamma distributed frailties by sex[1]



1) Note. Cf. figure 2, note 1.

Figure 3. Empirical, graduated and extrapolated biological aging



Linking to the proportional hazard model of Vaupel, Manton, and Stallard (1979) we have $\mu(x) = \eta(x)\varphi(x,t)$; which makes the standard mortality $\mu(x)$ proposed by Vaupel et al. (1979) a function of unknown biological aging of an individual interacting with environment in continuous time. In conclusion, by targeting individual mortality the proportional hazard model represented by eq. (4) is more realistic than earlier models. The model by Vaupel et al. (1979) operates with too few parameters; and the model by Li et al. 2009, 2013a-b, in addition to tacit assumption of homogeneous vitality, is based on misapprehension of the competing risks model as a tool to describing real world interaction in continuous time.

3. Normation of the frailty and selection model

Subject to two restraints viz. empirical cohort-based population mortality and invariability of the latent biological components across geography and time, two out of the three components of the frailty and selection model must be normed i.e. brought to a common level of comparison before the model can be deployed. Note also that if $\varphi(x,t) \equiv 1$ then the frailty and selection model (eq. 4) is *stationary* and formally equivalent to the one proposed by Vaupel, Manton, and Stallard (1979); leaving us with an un-normed product of generic components viz. frailty on conception and biological aging.

3.1.1. Biological frailty $\{z_i\}$

It is a well-established empirical fact that the sex proportion on live birth is positive in the disfavor of females. For an enlightening technical discussion of a range of important related biological issues cf. Boklage (2005), Gutiérez-Adán (2005), and Jongbloet (2005). The sex difference could be substantially higher on conception than at birth because of selection in the course of gestation. This, in itself, leads me to expect that males could be somehow more frail that females. See also Hansen (1996).

How do we assess intrinsic mortality $\eta(x)$ *per se*? Gavrilov and Gavrilova (1991, 2001) study deterioration and failure with age using reliability theory. They see aging as a direct consequence of systems redundancy. Inspired by explorative research by Aalen and Gjessing (2001), Li and Anderson (2009) study vitality-dependent mortality by modeling individual failure times as first-passage times in continuous space diffusion processes (Wiener processes). As a crucial abstraction from reality none of the authors consider biological heterogeneity associated with individual biological diversity instigating selection in survivorship. Li et al. (2009, 2013a-b), moreover, do not allow for interaction in continuous time between environment and intrinsic mortality (cf. Section 2.3). To make up for such drawbacks I propose an alternative and more empirical approach targeting joint quantification of a sex-specific gamma distribution on one hand, and intrinsic mortality on the other hand. My approach may be summarized as follows.

Since generic attrition with age is part of the biological package on conception and because the variance of male frailty no doubt is somehow greater than female frailty it is natural to expect that mortality is differential by sex. I shall assume individual frailty to

obey some probability distribution defined on the non-negative axis of the real numbers. This makes population mortality a weighted average of individual mortality. Following Vaupel, Manton and Stallard (1979) I shall assume individual frailty upon conception to be gamma distributed with *shape* or *form* parameter α, *scale* parameter β, and a *location* parameter equal to zero; these parameters together endow the gamma distribution with great functional flexibility. The gamma distribution has mean $E[Z]=\alpha\beta$ and variance $Var[Z]=\alpha\beta^2$.

3.1.2. Biological aging $\eta(x)$

According to Sharpless and DePinto (2007) "we age, in part, because our self-renewing stem cells grow old as a result of heritable intrinsic events, such as DNA damage, as well as extrinsic forces, such as changes in their supporting niches…" Hence, we should expect biological decay to be a monotonously increasing and otherwise homogeneous function of age.

A working hypothesis of a proportional relationship between a fixed age pattern of mortality and gamma parameters varying with time actually finds support by empirical population mortality divided by sex and one-year age groupings among one-year cohorts born in Sweden 1751-2011 and Denmark 1835-2011. The result rests upon heuristic least-square fitting of the non-parametric product $\eta(x)\varphi(x,t)$ to empirical population mortality via stochastic micro-simulation (cf. Appendix A) of individual failure time. Figures 1-2 indicate that for all practical purposes the form and scale parameters are statistically independent of time up to the end of the eighteenth century, an epoch where empirical cohort mortality was nearly stationary on a long term basis. It appears that the shape values (figure 1) are about the same for males and females while the scale values (figure 2) are slightly but systematically somehow higher for males compared with females; which makes the variance of the male frailty distribution slightly higher than the variance of the female frailty distribution.

The significant time dependence of the uncovered gamma parameters is clearly unrealistic. Moreover, rejuvenation – decrease of mortality with age - rules out the disclosed age pattern of mortality (figure 3, fully drawn line) as a realistic representative of intrinsic mortality $\eta(x)$, at least before age ten; since biological aging is a monotonously increasing

function of age (Sharpless and DePinto 2007). Needless to say, this observation is vigorously empirically supported. In other terms, postulating independence of time regarding frailty and aging discards the naïve frailty model proposed Vaupel, Stallard, and Manton (1979) as a valid description of individual mortality and cohort-based population mortality in the course of the demographic transition of Sweden and Denmark.

The uncovered mortality pattern can be interpreted either as a baseline mortality $\mu(x)$ with survivor function $\ell(x)$ under the frailty model of Vaupel et al. (1979) or as $\ell(x)=\eta(x)\varphi(x,t),\varphi(x,t)\equiv 1$ under the Heterogeneity and Selection Model (eq. 4). The outcome in terms of declining intrinsic mortality before age ten along with the significant time dependency of the gamma parameters speaks strongly against interpretation in the framework of the model proposed by Vaupel et al. (1979).

Opting for interpretation under the Heterogeneity and Selection Model (eq. 4) and accepting the uncovered mortality as a rather close approximation to intrinsic mortality in the age span 10-93: how do we remove inherent selection of the uncovered mortality pattern before age ten? And how should we approach intrinsic mortality beyond age 93 with the given uncovered information?

The fully drawn line graph (figure 3) displays the uncovered age pattern of mortality which I interpret as a close proxy of intrinsic mortality $\eta(x)$ in the age span 10-93. Because of lack of trustworthy empirical information the pattern has been truncated by age 94. To make up for the problem of a decreasing rather than a monotonously increasing function before about age ten I fit a cubic spline to the age segment 10-93; with a very satisfactory result, indeed; cf. the boldfaced dashed line in figure 3. Extending the polynomium upwards to age 110 and downwards to age 0, I obtain the final version of endogenous mortality $\eta(x)$ applied throughout this study. The correction makes the period effect greater than one before age 1 under stationarity.

For future use I select (shape, scale) values equal to (1.43, 84.91) of males and (1.46, 70.54) of females based on the uncovered values before 1800 cf. figures 1-2. These gamma distributions are likewise deployed all through this study.

4. Robustness of the chosen normation and parametrization

The robustness of the chosen parametrization of the Heterogeneity and Selection Model may be explored by considering the Heterogeneity and Selection Model (eq. 4) supported by stochastic micro-simulation using cohort-based population mortality as empirical benchmark. Here follows a summary of results.

4.1. Biological aging $\eta(x)$ and period effect $\varphi(x,t)$

From eq. (4) we note that the product $\eta(x)\varphi(x,t)$ is fixed across individuals, and hence at population level. This means that the product is neutral both to proportional change $k = k(x), x \in [0, \max(x)]$ and to disproportional change $k \neq k(x)$; the latter will change the age patterns of the factors albeit not the value of the product. Keeping biological aging $\eta(x)$ fixed, whatever its age pattern, ensures comparability of the period effect over time. This insight is, indeed, quite reassuring considering the latency of intrinsic mortality in terms of biological aging.

4.2. Impacts for biological aging $\eta(x)$ and period effect $\varphi(x,t)$ of choice of alternative gamma distribution of latent biological frailty

Note that realistic two-sex solutions impose certain restraints upon the choice of sex-specific gamma parameters. The chosen relationship between male and female mortality is given by the one of empirical stationary mortality used to quantify intrinsic mortality $\eta(x)$. Changing the shape or form parameter while keeping the scale parameter fixed instigates inverse proportional change of $\eta(x)$ and $\varphi(x,t)$, cf. section 4.1.

Changing the scale parameter, no matter the shape or form parameter, impacts on heterogeneity of frailty and thereby on inverse disproportional change of $\eta(x)$ and $\varphi(x,t)$, cf. section 4.1. In either case the pace of change of the uncovered period effect will be statistically neutral.

Part II

Prediction of survivorship under the Heterogeneity and Selection Model

5. Introduction

Historical mortality development has bearings upon future survivorship. As the frailty and selection model features failure time of individuals living thru particular historical or future epochs the natural observational plan under the model is longitudinal. Individual failures mark the end of life-courses anchored in time. I base interpretation of historical population mortality upon the Heterogeneity and Selection Model (eq. 4) on assumption of fixed latent distributions of frailty and biological aging across time and geography along with shared exposure to environmental influence upon individual survivorship. At population level period factor $\varphi(sex, age, time)$ operates as a trend cleansed of biological heterogeneity under the model. This makes the period factor or trend the centerpiece of interest on analysis of historical and anticipated pace and structure of mortality change.

5.1 Purpose

Despite a range of shortcomings (cf. Lee & Miller 2000 for examples) the macro-scopic mortality model of Lee & Carter (1992) still enjoys acceptance in insurance and pension circles and in certain quarters of public administration engaged with planning and population projection. Is the Heterogeneity and Selection Model competitive to the Lee-Carter approach as an instrument of mortality projection? A key issue in both models is the notion of *trend* describing level of mortality at time t. Keeping sex as a background variable and only depending on time, the Lee-Carter approach operates with a univariate trend; while the trend of new Heterogeneity and Selection Model is a multivariate function of *sex, age*, and *time*, including all two-factor interactions. Projection of mortality rests on prediction of the trend, and hence of mortality change per unit of time. Once the trend has been predicted projection of mortality can take place under either model.

The new Heterogeneity and Selection Model is fully parameterized. It can therefore be brought to statistically comply, not only with historical population mortality, but also with

future population mortality as anticipated, for example, by official national mortality projections. This feature makes it an instrument of consistent interpretation of the micro foundation and trend of historical and anticipated mortality development.

Among first-world countries with long historical time series of empirical mortality divided by sex, year, and one-year age groupings I shall focus on Sweden, Denmark, and England & Wales as examples in the following. The national cohorts to be considered are born between 1900 and 2011. The period of reference for historical evaluation and prediction of trends is arbitrarily set to 1960-2011. As we shall see, projections based on models or approaches sweeping latent heterogeneity under the rug undervalues mortality in age segments with intense biological selection such as the mature and elderly ages in contemporary low-mortality countries. For examples of biological heterogeneity and selection in human survivorship under the new micro model (eq. 4) cf. Appendix C, figure C.1 and Hansen (2014).

I shall first consider the historical anchoring and the realism of the (heterogeneous) pace of expected future development of mortality according to the official contemporary projections of Sweden, Denmark, and England & Wales. Special emphasis is given to cross-sectional trend analysis 1960 to 2011 and to the expected trend of official mortality projections 2015-2110 (Sweden), 2015-2049 (Denmark), and 2015-2062 (England & Wales). On the background of the national empirical mortality trends 1960 to 2011, what future mortality trends are anticipated by the official mortality projections of Sweden, Denmark, and England and Wales from 2015 and beyond? How do the national trends compare? And are they realistic? All national official projections considered anticipate continued decrease of mortality in the twenty-first century. By fitting the heterogeneity model to the official mortality projections, the embedded trends may be uncovered and analyzed as a function $\varphi(sex, age, time)$ of sex, age, and time. As a natural continuation of historical development the uncovered expected Swedish trend to 2110 turns out to be by far the most convincing and businesslike.

The Danish and English official projections less convincingly both anticipate accelerated mortality decline to converge towards the expected Swedish level in 2049 (Denmark) and 2062 (England & Wales) from markedly higher levels of empirical cross-sectional mortality around 2011. No justification for these optimistic expectations is given. These official mortality outlooks call for reconsideration.

So, secondly, I carry out set of alternative national mortality projections 2015-2110 under the Heterogeneity and Selection Model. Assuming continued persistent exponential decline of mortality 2012 to 2110 as observed of Sweden 1960 to 2011, what is the expected level of Swedish mortality as of 2110? What annual exponential mortality decline of Denmark and England & Wales is required to approach the anticipated Swedish level as of 2110? How do these expectations compare with the official national mortality projections? I let each national set of the alternative mortality projections comprise a main scenario and two auxiliary scenarios serving to illustrating less dramatic expected mortality decreases than anticipated under Scenario #1 and the main alternative of the official projections.

Thirdly, to study the undervaluation of mature and old-age mortality according to the official mortality projections I compare the official mortality projections (elected years) to the national projections under the Heterogeneity and Selection Model (eq. 4) based on the aforementioned assumptions regarding trend. Full and detailed reporting of results is not possible in the framework of this article. For an overview of files, results, and some codebooks cf. Appendix B.

Before discussing the main issues some preliminaries need to be considered viz. stochastic micro-simulation and data; the notion of *trend*; cross-sectional versus cohort projection of mortality in the framework of the new Heterogeneity and Selection Model vs. the original Lee-Carter model of 1992; and set-up of assumptions for predictions under the Heterogeneity and Selection Model.

5.2. Stochastic micro-simulation and Data

For a technical outline of fitting the Heterogeneity and Selection Model to time series of cohort-based population mortality by stochastic micro-simulation cf. Appendix A.

A necessary prerequisite on fitting the model to data is access to long empirical time series on population mortality by sex, age, and time. Cross-sectional one-year groupings of population mortality converted to birth cohorts will do for the present research purpose. Such information is easily available from Human Mortality Database (HMD), University of California, Berkeley; for Sweden (SE, 1749-2011), Denmark (DK, 1835-2011), and England & Wales (EW, 1841-2011). For a review of sources and applied methodology on creating the HMD cf. Wilmoth et al. (2007). For all practical purpose I shall assume graduations and interpolations of the HMD data to be close approximations to real-world mortality.

Using the normalization and assumptions presented in Section 3.1 individual failure time and period effects have been uncovered for annual cohorts born 1751-2011 (Sweden), 1835-2011 (Denmark), and 1841-2011 (England & Wales), extended with official mortality projections of the same three countries, each generation comprising 100,000 failure times x_i, $x_i \in [0,110[$. Due to questionable data quality of empirical population mortality among the oldest old, all results from age 94 and beyond, say, should be taken *cum grano salis*; questionable data beyond age 94 is of limited social significance, anyway.

For easy descriptive reference summaries of annual cross-sectional mortality in terms of life expectancies are computed for all mortality projections at population level. Supplementary advanced pivot applications allow detailed visualization of any underlying combination of mortality patterns by sex, age, birth date, date of observation, and projection; cf. the overview in Appendix B.

5.3 Trends

A trend model describes variation of a system as a function of time and statistically important time-dependent interactions. The anticipated trend of mortality should exhibit a natural extension of careful demographic analysis of historical development. Well-argued modifications makes mortality prediction a *projection* or a *forecast*.

Let $T(0)$ be the value of some system at time 0 and $T(t)$ the value of the system at time t. For future use let us briefly consider the following three trend definitions.

Exponential trend

An exponential growth system may be described as,

$$\begin{aligned} T(t) &= T(0)\exp\left(a + \int_0^t r(u)du\right) \\ &= T(0)\exp(a + rt) \end{aligned} \tag{5}$$

If $r = r(u), u \in [0,t[$.

Depending on the sign of the exponent, the system is increasing if $a + rt > 0$; decreasing if $a + rt < 0$; and stationary if $a + rt = 0$.

Log-linear trend

Conversion of eq. (5) to a log linear model is straightforward,

$$\ln\left(T(t)/T(0)\right) = a + \int_0^t r(u)du = a + rt \qquad (6)$$

If r constant over time $\left[T(0), \mathrm{T}(t)\right[$.

It is easily seen that exponential trend models may be transformed and generalized to log linear models. For a relevant example of a log linear three-factor model including all interactions consider,

$$\ln\varphi\left(sex(I), age(J), time(K)\right) = I + j + K + IJ + IK + IJK \qquad (7)$$

Eq. (7) evidently allows a much richer representation of the trend than eqs. (5) or (6). Eq. (7) is readily identified as a model of the period factor uncovered under the Heterogeneity and Selection Model (eq. 4); hence cleansed of biological influence; and naturally interpretable as a multi-factorial trend at population level.

Lee-Carter trend

Many producers of official mortality projections and forecasts today subscribe to the Lee-Carter model. This is true, for example, of Denmark and to some extent Sweden. The model, moreover, enjoys great popularity among life insurance and pension companies. Scratching the surface by ignoring latent genetic factors in survivorship Lee & Carter (1992) model population mortality as,

$$\log m(x,t) = a(x) + \mathrm{b}(x)k(t) + e(x,t) \qquad (8)$$

With normation $1 = \sum_{\forall x} b(x)$ and $0 = \sum_{\forall t} k(t)$. This makes vector $\{a(x)\}$ an average of the log rates. Starting off from a data matrix $\{m(x,t)\}$ of cross-sectional population mortality, vectors $\{\mathrm{b}(x)\}$ and $\{k(t)\}$ are found by single-valued decomposition (SVD) of matrix $\{\mathrm{b}(x)k(t)\}$. Statistic $\{k(t)\}$ is a trend vector. Statistic $e(x,t)$ is a random element. Note that the Lee-Carter trend is an exponential trend only depending on time (eq. 5).

Figure 4. Design matrix of the log-linear the-factor model with sub-models cf. eq. (7)

Model	Factors present in model (1: yes; 0: no)						
#	I	J	K	IJ	IK	JK	IJK
1	1	1	1	1	1	1	1
2	1	1	1	1	1	1	0
3	1	1	1	0	1	1	0
4	1	1	1	1	0	1	0
5	1	1	1	1	1	0	0
6	1	1	1	0	0	1	0
7	0	1	1	0	0	1	0
8	1	1	1	1	0	0	0
9	1	1	0	1	0	0	0
10	1	1	1	0	1	0	0
11	1	0	1	0	1	0	0
12	1	1	1	0	0	0	0
13	0	1	1	0	0	0	0
14	1	1	0	0	0	0	0
15	1	0	1	0	0	0	0
16	0	0	1	0	0	0	0
17	0	1	0	0	0	0	0
18	1	0	0	0	0	0	0
19	0	0	0	0	0	0	0

Girosi & King (2007) show that this model is a special case of a considerably simpler, and less often biased, random walk with drift model, and prove that the age profile forecast from both approaches will always become less smooth and unrealistic after a point.

Analysis of the period factor alias trend $\varphi(sex, age, time)$

Indicating level of mortality and inversely related to human control of survivorship the period effect $\varphi(sex, x, t)$ calibrates the interaction of the endogenous biological effects and the exogenous physical and social world. As the biological effects may safely be assumed fixed over time and geography, the period effect is crucial on projecting mortality under the frailty and selection model. Mortality change $r(sex, x, t) = d\varphi(sex, x, t)/dt$ per unit of time modifies $\varphi(sex, x, t)$. Note that $\varphi(sex, x, t)$ and $r(sex, x, t)$ are statistically independent: different levels of mortality may be associated with statistically the same rate of mortality change, and mortality development may be different for populations starting at statistically identical levels cf. eq. (5). This gives added weight to $r(sex, x, t)$ both on analysis of historical mortality change and on evaluation of future mortality development.

The estimated period effect is biologically homogeneous under the Heterogeneity and Selection Model (4) with fixed sex-specific distributions of frailty and progressing cellular aging. To what extent does a time series of values of $\varphi(sex, x, t)$ referring to a chosen period of reference, support a working hypothesis of a proportional or multiplicative relationship between *sex*, *age*, and *time*? Are there important interactions?

Rational evaluation of the *working hypothesis* calls for modeling by iterative proportional fitting of the (simulated) data to a log-linear three-factor model. A likelihood is a joint probability distribution of a collection of random variables. In the situation at hand we have no random variables, and therefore no distribution of random variables. By pragmatic interpretation, let the (simulated) outcome of each cell of the three-dimensional contingency table be a continuous variable per unit of time. We may compute a surrogate of a likelihood (SL) referring to the specific contingency table. By comparing the SL-value of a given sub-model and the SL-value of the full (saturated) three-factor model a decision may be reached whether the specific sub-model may describe the "data" without important loss of information. Allowing evaluation of the main effects and interactions included in each of the eighteen sub-models, the pragmatic approach dictates graphical model control rather than decision-making based upon statistical testing. An appropriate analytic framework is log-linear modeling of the period factor $\varphi(sex, x, t)$ featuring the variables *I, J, and K;* interactions *IJ, IK,* and *JK;* a joint off-set value a_{ijk} ; and a random residue e. The full (saturated) model is given by eq. 7.

I estimate the model using iterative proportional fitting (IPF) and graphical model control. In addition to the saturated model, the log-linear three-factor model comprises eighteen sub-models cf. figure 4. A simple pragmatic test of independence of interactions entails graphing the K-factor (time) derived under models #2-11 against time. Interactions tend to be small. Omitting interactions with *K* makes $\varphi(t)$ equal to $\exp(K(t))$.

5.4 Cross-sectional versus cohort projection of mortality in the framework of the new Heterogeneity and Selection Model versus the Lee-Carter Model

Projection of population mortality under the Heterogeneity and Selection Model (HSM) rests on prediction of individual failure times in a life cycle perspective; in dazzling contrast

to mortality projection based on analysis of cross-sectional population mortality such as the Lee-Carter model (1992) (LCM). Here follows a brief comparison of characteristics of the two model cf. eqs. (4) and (8).

Observational plan

LCM: Cross-sectional

HSM: Longitudinal

Level of aggregation

LCM: Population

HSM: Individuals

Parameters

LCM: age, time, sex (background variable)

HSM: Individual biological frailty (latent), biological aging (latent), age, time, sex (background variable)

Trend

LCM: Heterogeneous univariate function of *time*, given *sex* (background variable)

HSM: Biologically homogeneous multivariate function of *time*, *age*, and *sex* including all interactions

Projection of trend

LCM: Time series analysis with one deterministic and possibly one or several additional stochastic factors (cf. Haldrup et al. 2014 and their references).

HSM: Deterministic prediction of statistically important factors and interactions in the framework of log-linear three-factor modeling.

Discussion

As the HSM model is fully parameterized all information regarding the historical past of individuals or groups of individuals is maintained. Conditional on this information the remaining life time symbolized by stochastic variate T to individual failure is evaluated in presence of the separately projected collective external exposure from time t and beyond; cf. Section 7.2.1.

The LCM trend represents an extrapolation in the framework of some time series model of factor $k(t)$ obtained by single valued decomposition of a matrix with reference to a

given historical cross-section of central demographic mortality rates; cf. eq. 8. Such a matrix is strongly heterogeneous by the observational plan i.e. latent heterogeneity and lack of memory of the historical past of the overlapping cohorts. Furthermore, as the LCM approach is blind to biological selection it undervalues mortality in life segments with great selection.

Both approaches leave social selection out of consideration; which may induce further error on assessment of future mortality.

5.5 Simulation results

The simulation results occur in two forms:

a. A register of individual failure times with information on sex, personal frailty, birth date, and exact age-at-death.
b. A register of aggregate information on sex, empirical and model-based population mortality, model-control in terms of squared deviation between empirical and model-based population mortality, and period effect $\varphi(sex, x, t-x)$, t denoting temporal time.

As *birth date+ age=time of observation* all information may be converted back and forth for studies under longitudinal or cross-sectional observational plans. Simulation of individual failure times rules out trespassing personal integrity.

The libraries of simulated data offer rich potentials regarding model-based interpretation of heterogeneity and selection as a factor of population change in the course of the demographic transition; for example impact upon survivorship of epidemics and environmental disasters such as the Spanish Flue and their aftermath. Most analytic potentials are unexploited so far.

6. Analysis of empirical and anticipated mortality trends according to official national projections

Trend $\varphi(sex, age, time)$ is cleansed of latent heterogeneity under model (4). This makes it a better proxy of the *level of mortality* than population mortality $m(sex, age, time)$. As a prelude to formulating informed expectations regarding future mortality change it will be worthwhile to consider the quality of empirical anchoring 1960-2011 and anticipated

mortality change according to current official projections of Denmark, Sweden, and England & Wales. I obtain national estimates of $\varphi(sex, age, time)$ by fitting model (4) to cohort-based empirical population mortality extended with projected national cohort mortality; followed by log-linear analysis of $\varphi(sex, age, time)$ in the framework of the log-linear model (eq. 7.).

To ensure comparability over time the uncovered national mortality levels must be based on a system of consistent national weights at a suitable point in time, e.g. 1960. The calibration prompts fitting the log-linear three-factor model (eq. 7) to trend $\varphi(country, sex, age)$ keeping year 1960 as a background variable. Furthermore, establishing well-motivated expectations regarding future *change of mortality* under the Heterogeneity and Selection Model motivates comparison of the national growth regimes $\exp\left(a + \int_{T_0}^{T_t} r(u)\,du\right)$ (cf. eq. 7); using a base index equal to unity as of some chosen year.

6.1 Normalized national *mortality levels* as of 1960 by sex and age

I first consider long term development of *level of mortality* from 1960 to end year of the current national mortality projections of Denmark, Sweden, and England & Wales. Next I analyze empirical national *mortality change* from 1960 to 2011 and anticipated national mortality change from 2015 and beyond. The findings altogether leading to informed decisions regarding forestalled national mortality change to be incorporated in national mortality projections under the Heterogeneity and Selection Model (eq. 4).

Keeping $time = 1960$ as a background variable and carrying out the log-linear three-factor analysis I obtain the values of $T(country, t = 1960)$, $country = DK, SE, E\,\&\,W$, shown in

Table 1. Estimated standardized national mortality levels as of 1960+

Country	National mortality level 1960
DK	0.964433
SE	0.926103
EW	1.119615
Index	1

+) By canonical normalization the national levels factorize to 1.

Figure 5. Trend $\varphi(country, sex, age)$ as of $time = 1960$ under the Heterogeneity and Selection Model. Denmark, Sweden, and England & Wales.

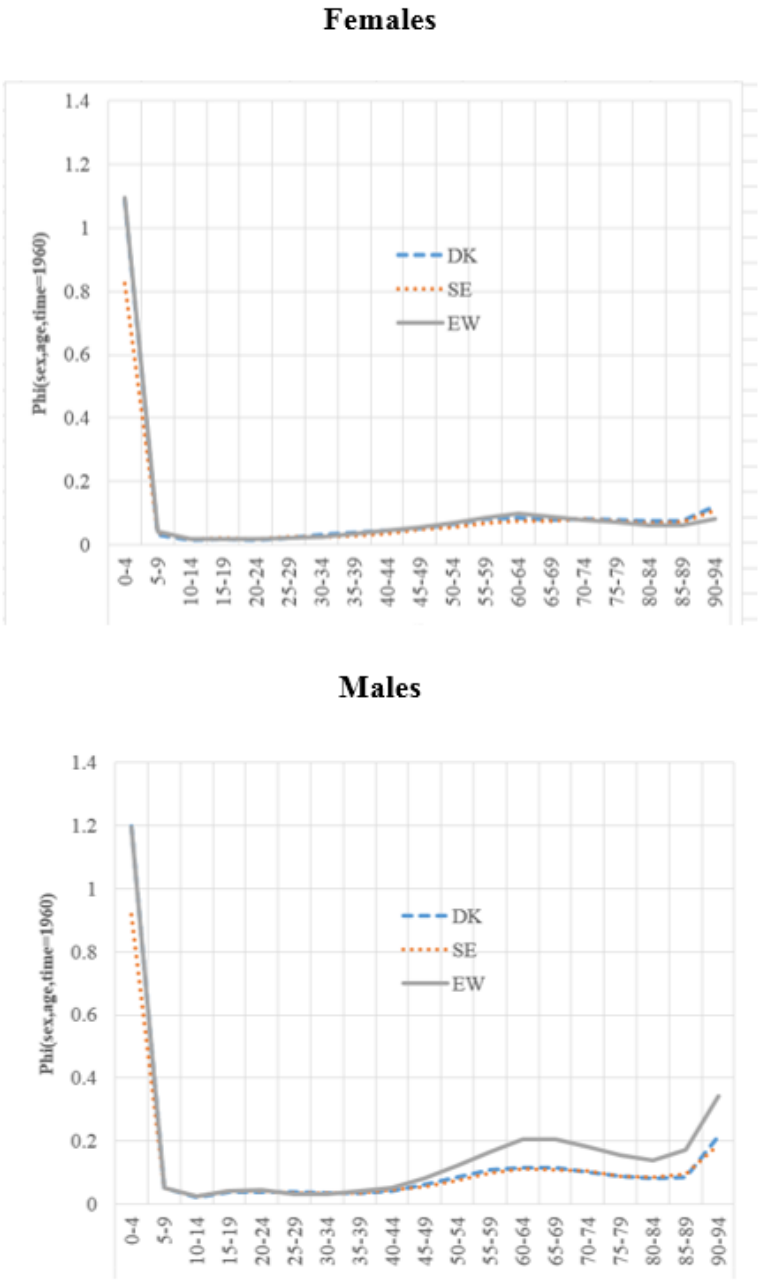
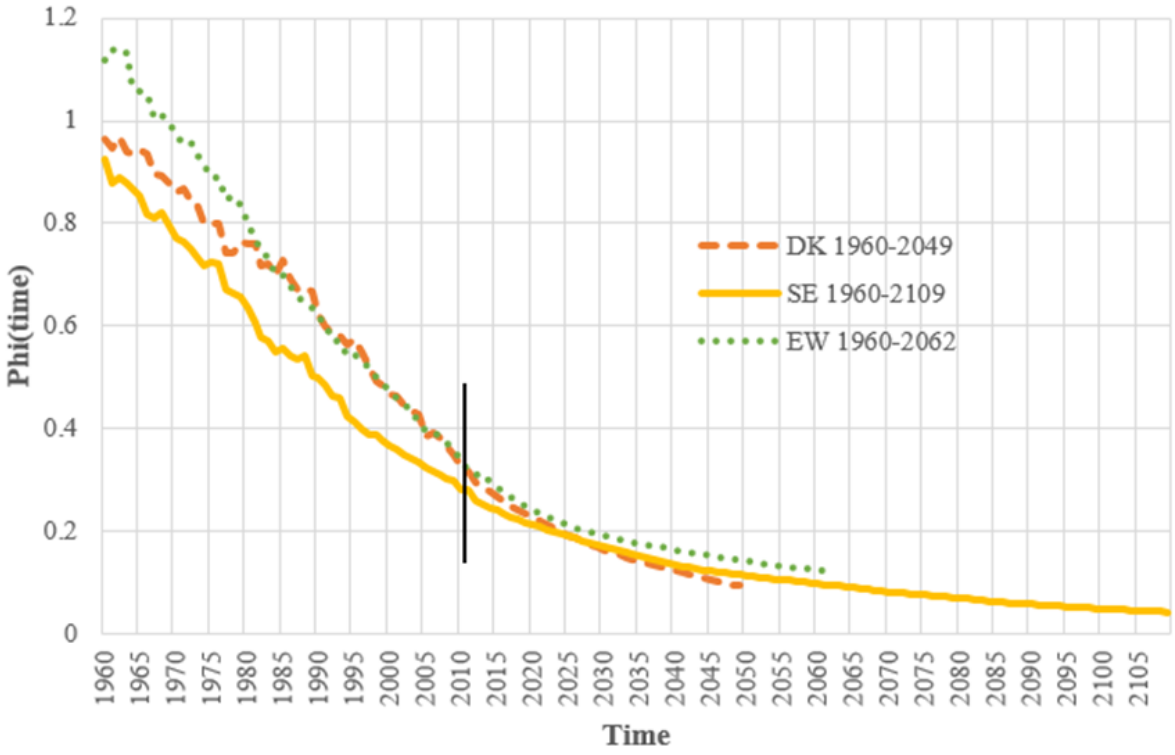


Table 1 after canonical normalization. The values should be seen as rather close approximations in presence of interactions, some of which of some statistical importance. With this proviso the table indicates that English mortality in 1960 was about 11 pct. above the joint level of cross-sectional mortality with Swedish and Danish mortality making up respectively about 92.6 pct., 96.4 pct. of the shared level. Figure 5 graphs $\varphi(country, sex, age)$ as of year 1960 against age; showing great similarity between Sweden

and Denmark across sex and age, and English females by age. Male mortality is higher than female mortality; and English male mortality is strikingly higher from around age 40 and beyond compared to Swedish and Danish male mortality. Furthermore, English infant and child mortality is distinctly higher than Swedish and Danish infant and child mortality.

Using the normalized national mortality levels as of 1960 (table 1) as indices (weights) figure 6 indicates national historical development from 1960 to 2011 along with anticipated advance from 2012 to the end year of the respective national mortality projections. The Danish and English official projections tend towards the projected mortality level of Sweden by the midst of the twenty-first century. Evaluation of these prophesies and their anchoring in the historical past since 1960 calls for further scrutiny.

Figure 6. Development of normalized national levels of mortality over time from 1960 to end Year of national mortality projections. Denmark, Sweden, and England & Wales



6.2 Normalized national *mortality change* by sex and age since 1960

6.2.1 Log-linear analysis of empirical mortality change 1960 to 2011

While the Swedish and English level of mortality exhibit a steady decline ever since 1960 Danish historical development has been rather rambling up to recent years though starting roughly on a par with Sweden in about 1960. This becomes particularly clear by log-linear analysis of the national annual change of mortality using $\varphi(sex,x,t), t \in [1960,2011[$ based

on simulation, keeping country as a background variable. It readily appears (documentation not reported) that interactions *IJK, IJ* and *IK* can be omitted as they add little to the overall description of the period effect. Interaction *JK* indicates the existence of an unsystematic and slightly positive relationship of *age x* and *time t* between 1960 and 2011 in each population. Hence $r(time) \simeq r(sex, age, time)$ and $\varphi(t) = \exp(K(t))\ t \in [1960, 2011[$. Comparing the pace of mortality change across nations in the period of reference calls for normation of $K(t)$ by indexing i.e. $K(t)/K(t_0 = 1960), t \in [1960, 2011]$.

Graphing the indexed time series against time (figure 7) documents a linear exponential decline of -1.4 per cent per year from 1960 to 2005 of Swedish mortality followed by a minor slow-down in recent years, also exponentially linear as it seems. From 1960 to c. 1975 mortality decline was somehow slower in Denmark and England & Wales. The timing and magnitude of the Swedish and the English growth patterns are almost identical from around 1975 to 2011; in sharp contrast to Danish development in the same epoch; cf. also figure 6. After nearly stagnation of Danish mortality from around 1975 to the early 1980s, a significant decline, starting in about 1984, gathers some momentum around 1994 to 2009. Since 1998 Danish mortality decline has been slightly faster than the Swedish decline.

Unhealthy Danish life style (e.g. smoking, eating habits, inadequate physical exercise) has not turned up all of a sudden in about 1975; nor can the distinctive Danish surplus mortality after 1960 be explained by postponed selection as the timing and extent of the modern general mortality decline is about the same in Sweden and Denmark (Hansen 2014). It is hard to avoid the conclusion that the Danish mortality decline in the 1970s and 1980s is a consequence of inadequate health policies and inefficient and sloppy political and administrative governance which society is now striving hard to make up for.

6.2.2 Anticipated national official mortality change from 2012 and beyond

To evaluate anticipated national official mortality change from 2012 and beyond I fit the Heterogeneity and Selection Model (HSM) to a sample of national official mortality projections from 2011 until the end of their time horizons. I summarize the results by approaching the uncovered log-linear trend with the exponential trend model (eq. 5) to obtain the results displayed in table 2.

Figure 7. Indexed period factors $\varphi(t), t \in [1960, 2011[$, (1960 = 1) in presence of all main factors and interactions of log-linear model #2

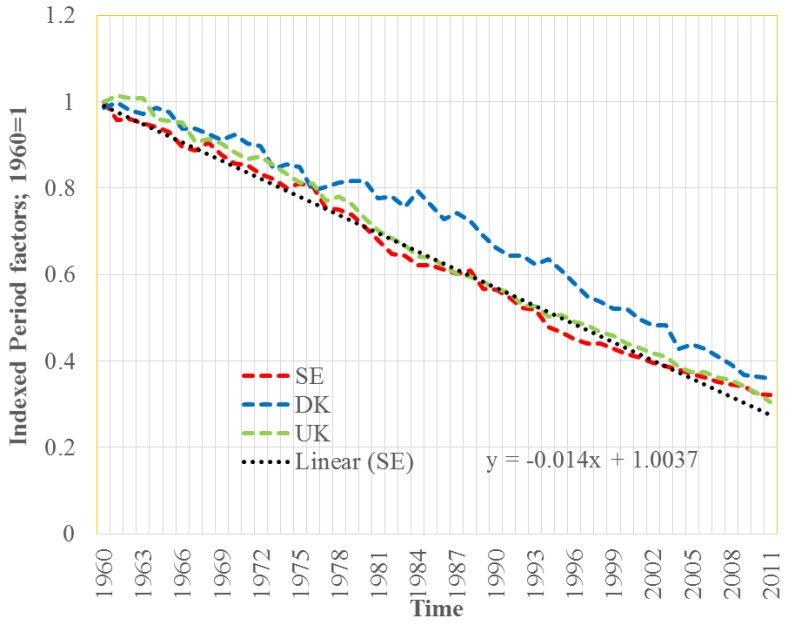


Table 2. Annual exponential mortality change *r* according to elected official mortality projections of Denmark, Sweden, and England & Wales

Official mortality projection	Mortality level as of 2011	Rate of annual mortality	Coefficient of determination R^2
SE 2011-2110	0.326	-0.016	0.974
DK DS/DREAM 2011-2110	0.358	-0.027	0.999
DK DS/DREAM 2014-2049	0.422	-0.027	0.921
DK DS/DREAM 2011-2110	0.358	-0.027	0.999
DK DS/DREAM 2011-2050	0.377	-0.032	0.999
SE SCB 2012-2110	0.294	-0.0183	0.994
EW 2012-2062	0.254	-0.024	0.959

The high values of the estimated coefficients R^2 of determination indicate that the exponential model with fixed growth r describes the anticipated national mortality trends adequately (graphical control omitted) with exception, perhaps, of the UK projection 2012-2062 and the DK/DREAM projection 2014-2049. The annual mortality changes deduced from the current official mortality projections all point to decline though at rather different pace. Sweden has the slowest and Denmark the fastest decline. Although less sufficiently described by the exponential trend model as indicated by the R^2 value, the pace of decline of the UK projection is slightly slower than the decrease foreseen by the Danish projection.

In keeping with historical development 1960 to 2011, Sweden maintains an expected annual decline around of -1.6 per cent all the way down to 2110. In striking contrast to Danish historical experience 1960 to 2011 on one hand, and expected Swedish development on the other hand, Statistics Denmark (DS) and Danish Rational Economic Agents Model (DREAM) without argumentation fancy an annual mortality decline of no less than about 2.7 per cent up to 2050, evidently in the wishful hope of reaching the anticipated level of Swedish mortality by the midst of the twenty-first century. An interesting open question is what DS/DREAM think will happen to this record-high mortality decline after 2050. Will the decline slow down? May mortality even rise again? Who and how will people be affected?

The official projection of England & Wales apparently is on a par with the anticipated Swedish mortality around 2062; which, spurs an expected annual English mortality decline of no less than about 2.4 per cent. No argumentation for the ambitious accelerated mortality decline of England & Wales after 2011 appears to be given.

7. National mortality projections under the Heterogeneity and Selection Model from 2012 to 2110

7.1 Setup of dynamic conditions for projection of mortality 2012-2110.

"It is tough to make predictions, especially about the future!" (Proverb of unknown origin but usually ascribed to the Danish humorist Storm P.). What should we actually expect regarding mortality change in Sweden, Denmark, and England & Wales between 2012 and 2110?

In terms of cross-sectional life expectancies around 2015, Swedish and English mortality is much lower than Danish mortality. Furthermore, as we have seen, Swedish and UK

mortality of Sweden and England & Wales has been subject to a linear decline of about -1.4 per cent annually 1960 to 2011; in striking contrast to the somehow rambling Danish mortality development in the same epoch.

Table 3. Indexed period effects $\varphi(t) \simeq \exp(K(t))$

Year t	Country		
	SE	DK	UK
1960	1	1	1
2011	0.322812	0.367905	0.305118
2110++	0.08	0.08	0.08

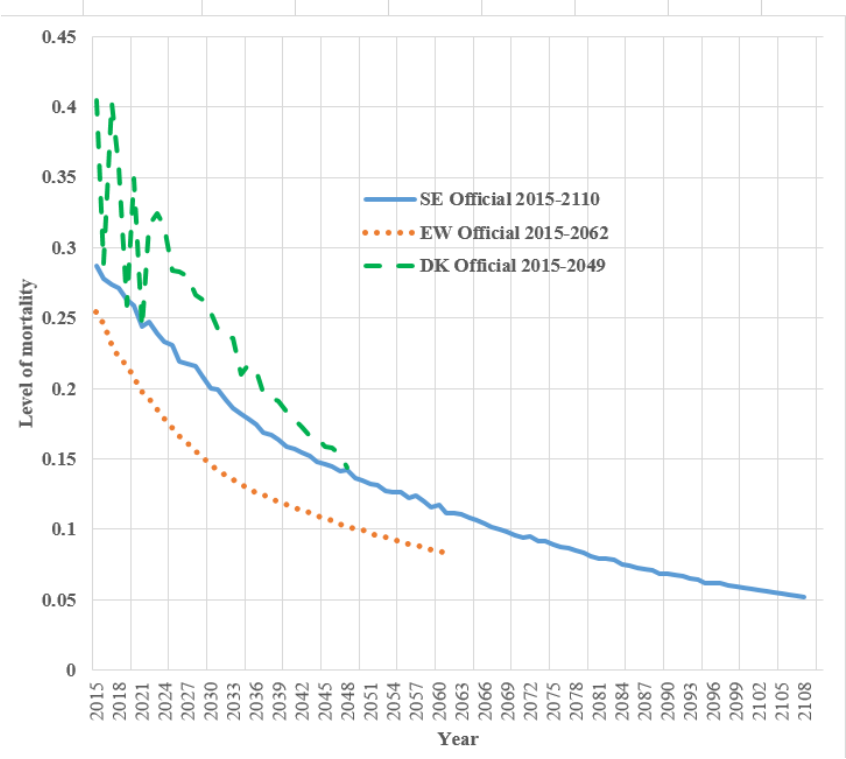
+) $\varphi(t)/\varphi(t=1960)$. Note that the national levels of mortality were different in the chosen base year of comparison.

++) Expected values based on fixed exponential Swedish mortality change 2012-2110. Statistic $K(t)$ is the log-linear factor *period.*

Table 3 shows the indexed national period effect $\varphi(t)$ as of year 2011 (base year of index: 1960) and the indexed shared target value of 0.08 as of 2110. It is immediately clear that starting from higher levels of mortality in 2011 than Sweden, Danish and English mortality decline must be faster to hit the assumed shared mortality levels in about 2110. See also figure 6.

Figure 8 exhibits the anticipated indexed levels of mortality (1960=1) according to official mortality projections of Sweden, England & Wales, and Denmark from 2015 and beyond. The slope of the curves indicate rate of annual change of mortality. For an overview of estimated rates of annual mortality change and associated determination coefficients to indicate the quality of fit of the exponential functions to the curves cf. table 2. The Danish curve starts at a much higher mortality level than the Swedish and the English curves which do not differ much in 2015. Comparing figures 7-8 and table 2 it readily appears that the trend of official Swedish mortality projection, by and large, represents a natural extension of the historical trend 1960 to 2011. The anticipated stronger English mortality decline 2015 to 2062, even though slowing down after c. 2040 appears somehow questionable as an extension of the preceding historical decline. No reasons are given for the anticipated Danish mortality decline 2011 to 2049. It is empirically ill supported 1960 to 2011.

Figure 8.. Anticipated indexed level of mortality according to official mortality projections of Sweden 2015-2110, England & Wales 2015-2062, and Denmark 2015-2049[1]



1. Cf. Table 1 for an overview of estimated parameters on fitting exponential functions with fixed annual growth rates to the curves. Base of index: $\varphi(t=1960)=1$.

Consider a main scenario and two auxiliary scenarios of future mortality change, each with fixed exponential growth targeting the same among three different levels of mortality in 2110. Let the lowest of the three levels define national scenario #1 and the two higher mortality levels scenarios #2-#3. Furthermore, let the lowest level be determined by persistent exponential decline of Swedish mortality 2012 to 2110 i.e. $\varphi(x,t=2110)=.08$ (Scenario #1); and let Scenarios #2-3 define slower mortality decline in terms of arbitrary target values of $\varphi(x,t=2110)$, respectively equal to .16 and .26. Table 4 shows the fixed national annual change that it takes to approach the respective target values of $\varphi(x,t=2110)$, given the national levels of $\varphi(x,t=2011)$. Note that the assumed annual mortality decline of scenario 1 of Sweden, is slightly slower than the one anticipated by Statistics Sweden (SCB, table 1). All-in-all I have now defined the setup of nine mortality projections to be carried out under the Heterogeneity and Selection Model using the annual change listed in table 3.

Table 4. Anticipated indexed period effects by 2110 and expected annual average change of mortality 2012-2110. By scenario and country.

Anticipated indexed period effects 2110 $\varphi(t_{1960}=1)$		Expected annual change $r=r(t)$ 2012-2110+		
Scenario #	Value	Sweden	Denmark	England & Wales
1	.08	-0.014091	-0.015412	-0.013522
2	.16	-0.007742	-0.009063	-0.007172
3	.26	-0.002582	-0.003903	-0.002013

+) $r=\frac{1}{2110-2011}\ln\left(\frac{K(2110)}{K(2011)}\right)$.

Assuming fixed exponential mortality decline from 2012 to 2110, table 4 exhibits the required national values of change $r=r(t), t\in[2012,2110]$ to hit the three alternative target values of 0.08, 0.16, and 0.26 in about 2110 leaving us with nine projections. Main scenario (Scenario #1) represents continued mortality change of SE and UK as of 1960 to 2011 and a somehow faster mortality decline of DK to hit the target value of .08 in about 2110. Target values 0.16 and 0.26 refer to Scenarios #2-3, respectively illustrating anticipated slower and low mortality decline.

7.2 A summary and comparison of official mortality projections with predictions under the Heterogeneity and Selection Model

Projection of population mortality under the Heterogeneity and Selection Model rests on prediction of individual failure times in a life cycle perspective; in glaring contrast to mortality projection based on analysis of cross-sectional population mortality such as the widely accepted Lee-Carter model (1992). The official mortality projections of Sweden and Denmark are all based on the Lee-Carter model; in Sweden only from age 50 and beyond (SCB 2012). For a summary of the Lee-Carter model (LC) cf. Section 5.3. The English projections rest on cohort-based extrapolation based on careful analysis of historical trends 1960-2011 (Office of National Statistics 2013; Gallop 2007, 2008). A brief review of

Table 5. Expected length of life at birth truncated at the ninety-fourth birthday[1]. By projection, sex, and elected years. Denmark, Sweden, and England & Wales[2]

Projection	Females				Males			
	2015	2049	2061	2110	2015	2049	2061	2110
DK_Insurance	84.1	86.8			81.2	85.3		
DK_Official	82.5	86.7			78.8	84.9		
HSM model:								
DK_Sc_1	82.3	85.0	86.0	89.2	78.5	81.5	82.6	86.4
DK_Sc_2	82.2	83.6	84.3	86.5	78.3	80.0	80.7	83.2
DK_Sc_3	82.0	82.4	82.7	83.9	78.1	78.7	78.9	80.2
SE_Official	83.7	87.0	87.9	90.5	80.5	85.3	86.3	89.6
HSM model:								
SE_Sc_1	83.5	85.9	86.7	89.3	80.0	82.5	83.4	86.7
SE_Sc_2	83.4	84.6	85.1	86.9	79.7	81.1	81.6	83.7
SE_Sc_3	83.3	83.5	83.6	84.3	79.6	79.8	80.0	80.7
EW_Official	82.9	86.6	87.4		79.5	84.2	85.3	
HSM model:								
EW_Sc_1	82.6	85.0	85.8	88.6	78.9	81.7	82.5	85.9
EW_Sc_2	82.4	83.6	84.0	85.9	78.7	80.0	80.5	82.6
EW_Sc_3	82.3	82.5	82.5	83.1	78.7	78.6	78.7	79.5

1). Notes. Based on cross-sectional observational schedules. DK Insurance stands for Danish Statutory Mortality for commercial use. The results named "official" refer to official projections. The alternative projections are based on the Heterogeneity and Selection Model (HSM) (eq. 4) and the log-linear three-factor model (eq. 5). EW stands for England & Wales.

prediction under the Heterogeneity and Selection Model (HSM) follows in Section 7.3. So how do the official projections differ from those obtained with the HSM methodology?

It goes without saying that a full reporting and comparing of results is far beyond the practical limits of the present report. At this place the presentation of cross-sectional life expectancies and comparison of sex-age specific population mortality is bound to be selective. A foretaste illustrating uncovered heterogeneity and selection in survivorship under the Heterogeneity and Selection Model will also be provided (Appendix C, figure C.1). All historical analyses and projections under the Heterogeneity and Selection Model model are fully documented elsewhere. For an overview cf. Appendix B.

7.2.1 The two-step projection procedure under the Heterogeneity and Selection Model

Using a two-step procedure, projecting mortality under the Heterogeneity and Selection Model is straightforward. First, the period factor at time $t > t_0$ is,

$$\varphi(sex,x,t) = \varphi(sex,x,t_0)\exp\left(\int_{t_0}^{t-t_0} r(x,u)\,du\right), t > t_0 \qquad (9)$$

Under the given assumptions, with $\varphi(x, t = 2011)$ referring to the saturated log-linear model (sub-model #1, figure 4), findings from log-linear analysis of the period factor 1960-2011, equation (9) simplifies to:

$$\varphi(sex,x,t) \simeq \varphi(sex,x,t_0)\exp\left(r(t-t_0)\right), t_0 = 2012 \qquad (10)$$

Once the time series $\varphi(x,t), t = 1900$ to 2109 has been established, converting to cohorts born between 1900 and 2109 is straightforward. The mortality projection may now be carried out under the Heterogeneity and Selection Model at the level of individuals i.e. in the framework of a prospective longitudinal observational plan.

7.3 A summary of projection results

Table 5 presents a summary of population mortality in terms of cross-sectional life expectancies at chosen years across official projections (boldfaced values) and the three scenarios under the HSM-model (italicized). The scenarios rest on the assumptions listed in table 4. The main findings may be summarized as follows.

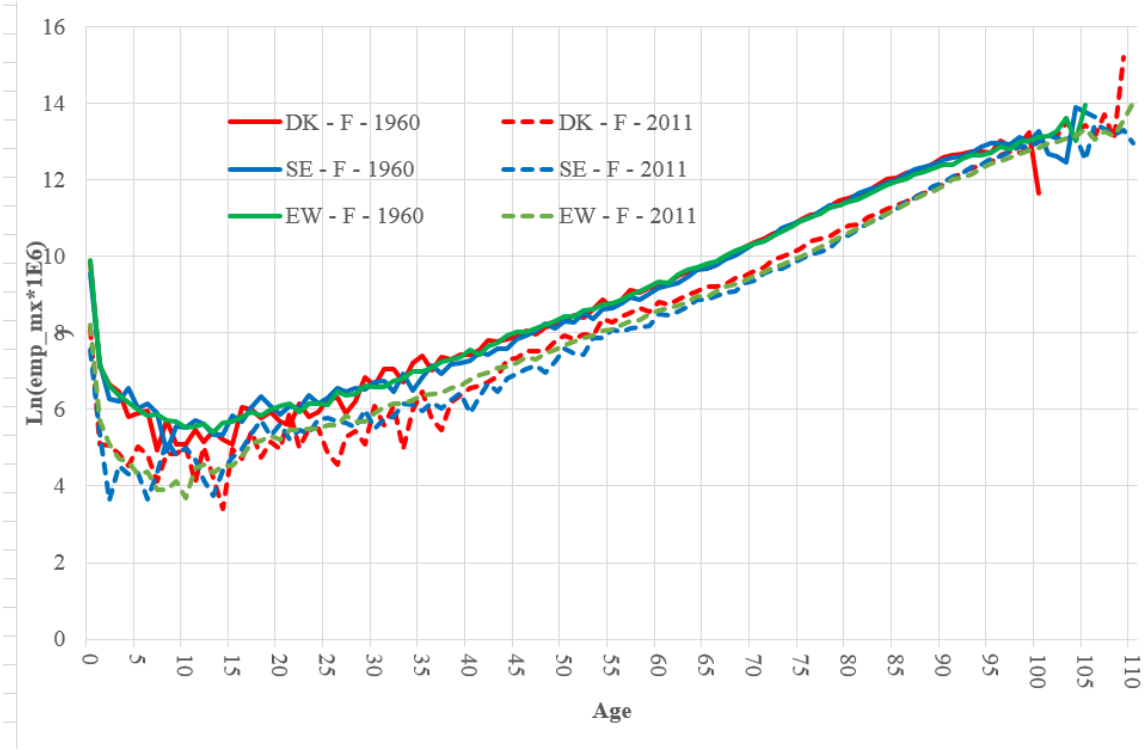
1. All projections agree on continued substantial decreases of mortality in the course of the twenty-first century.

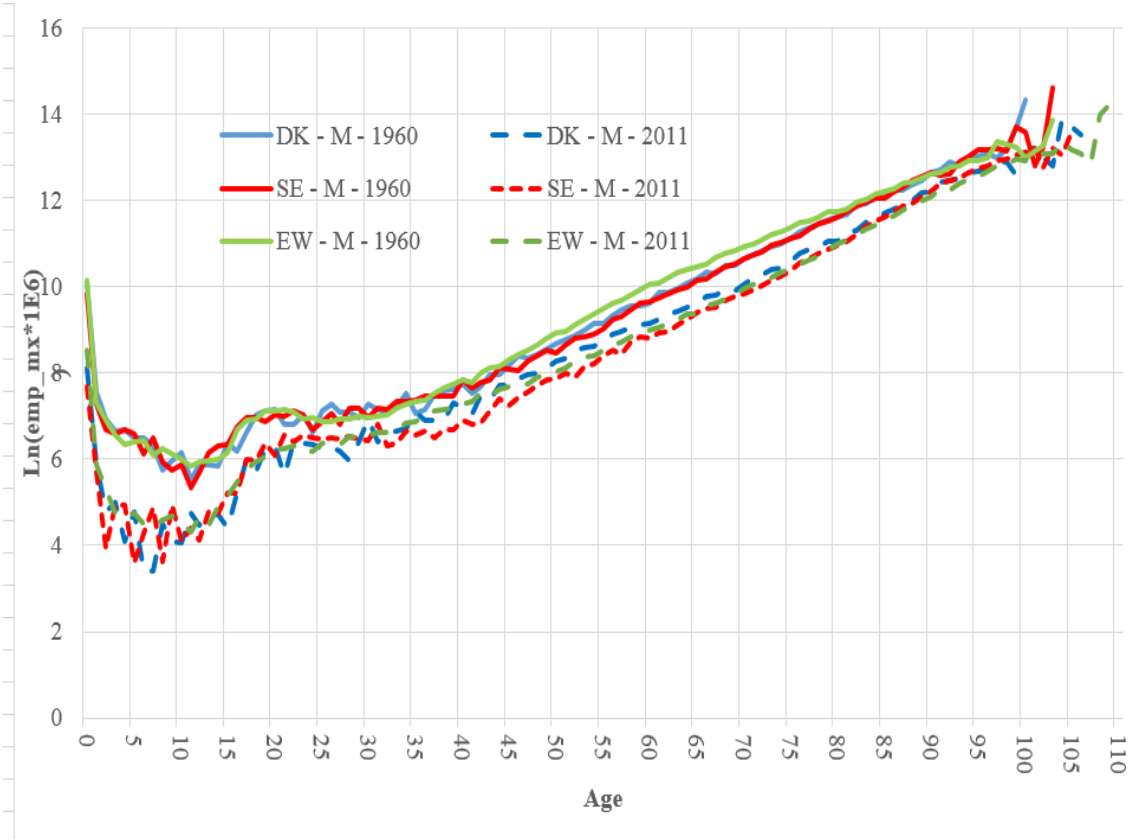
2. There is a remarkable general agreement and similarity across sex and timing of the Swedish and the UK results according to the official projections.

3. The gender gap diminishes over time as the male gains in life expectancy are systematically greater than the female gains up to each of the chosen years over the entire time horizon; in particular of the official projections over the scenarios.

4. The life expectancies according to the official projections are higher than those based on the Heterogeneity and Selection Model; in particular of males.

Figure 9. Empirical cross-sectional mortality 1960 and 2011 of Sweden, England & Wales, and Denmark. By sex (F, M). Semi-logarithmic scale[+].





Predictions (1) to (3) seem natural and plausible in a historical perspective. Declining mortality increases expected length of life at birth. Furthermore, declining mortality postpones biological selection which gives males an edge over females regarding gain in expected length of life. Hence the gender gap in survivorship diminishes over time.

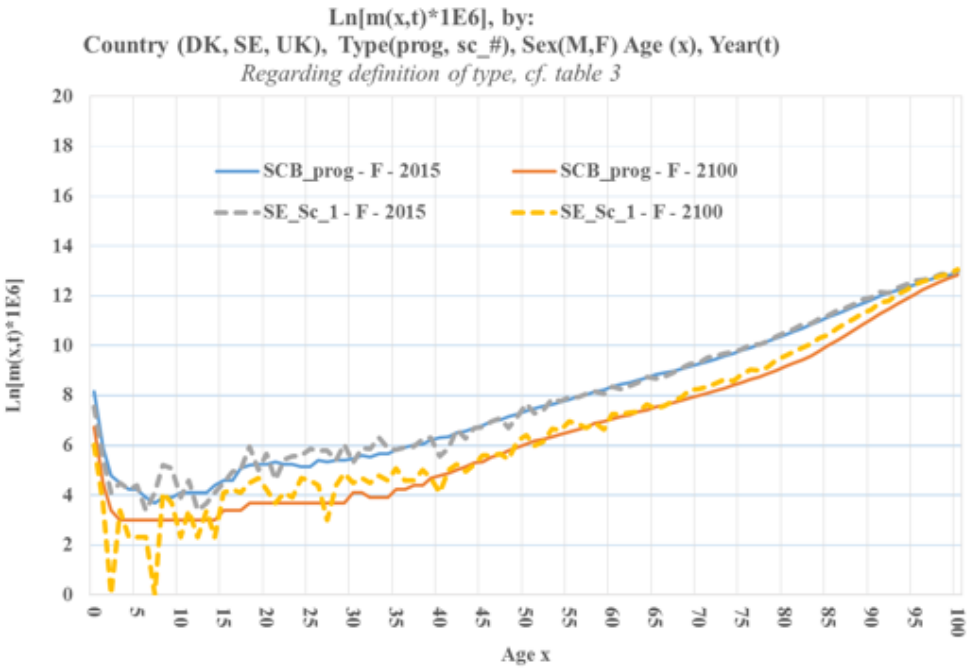
Conclusion (4) calls for further scrutiny. First, the official mortality projections assume faster mortality decline than expected in the projections based on the Heterogeneity and Selection Model. This in itself generates higher life expectancies according to the official mortality projections. Second, the Lee-Carter model and other mortality models based on population mortality sweeps biological diversity in survivorship under the rug contrary to the Homogeneity and Selection Model. In the course of the early-modern long term decline of mortality biological selection over the life course has undergone huge change in terms of ever-increasing postponement of latent biological selection. This is not an issue in the Lee-Carter universe. With decrease of mortality, biologically weak people stay alive longer due to postponement of selection. Hence, health of the risk set of survivors deteriorates; spurring slower mortality decline in the mature and elderly ages with fast progressing cellular decay. See figure 3; and figure C.1, Appendix C.

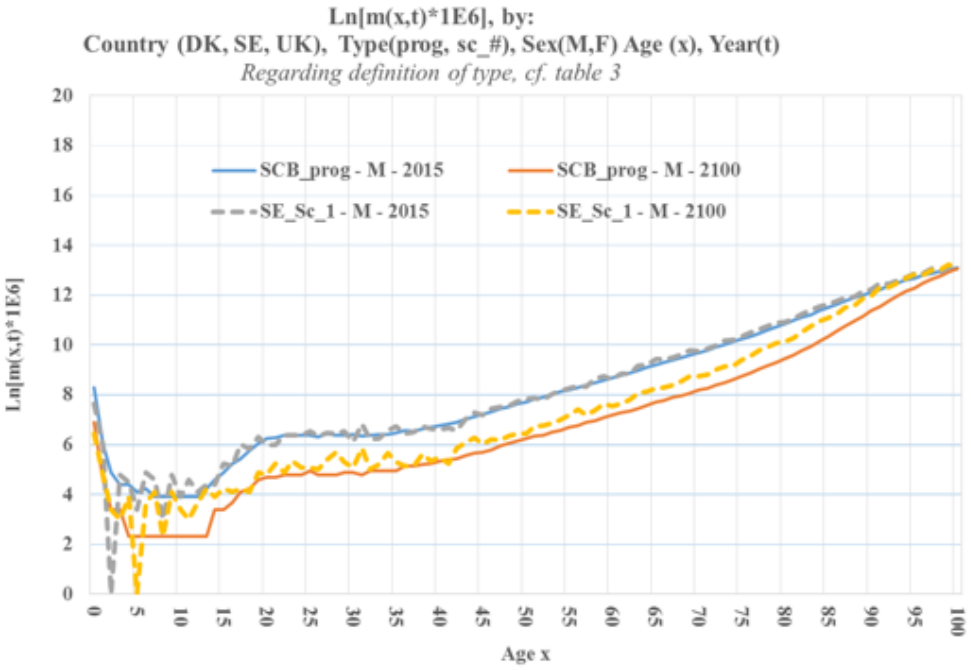
Gross change of national mortality by sex and age 1960 to 2011

Figure 9 compares gross change of national mortality by sex and age from 1960 to 2011 of Sweden, England & Wales, and Denmark. The levels and age patterns are basically quite similar, with Danish female mortality in high end both in 1960 and in 2011. There has been a sizeable mortality decline in the mature and elderly ages, and more so in the ages below 25. In other terms, empirical mortality change 1960 to 2011 has been non-proportional. This speaks strongly against utilizing a mortality model that is blind to age structural change and differential environmental impact in the course of time.

The official positions towards the utility of the Lee-Carter model (1992) as an instrument of mortality projection are mixed: In Sweden the Lee-Carter approach is used for projecting mortality from age 50 and older; in Denmark this methodology enjoys full and unrestricted acceptance – with devastating effects, alas. Incapacity to accommodate age-differential mortality change over time actually rules out use of the Lee-Carter approach in official English mortality projections (Gallop 2008). All three national agencies operate the level of population mortality in terms of central death rates; hence ignoring important latent heterogeneity with impact for selection.

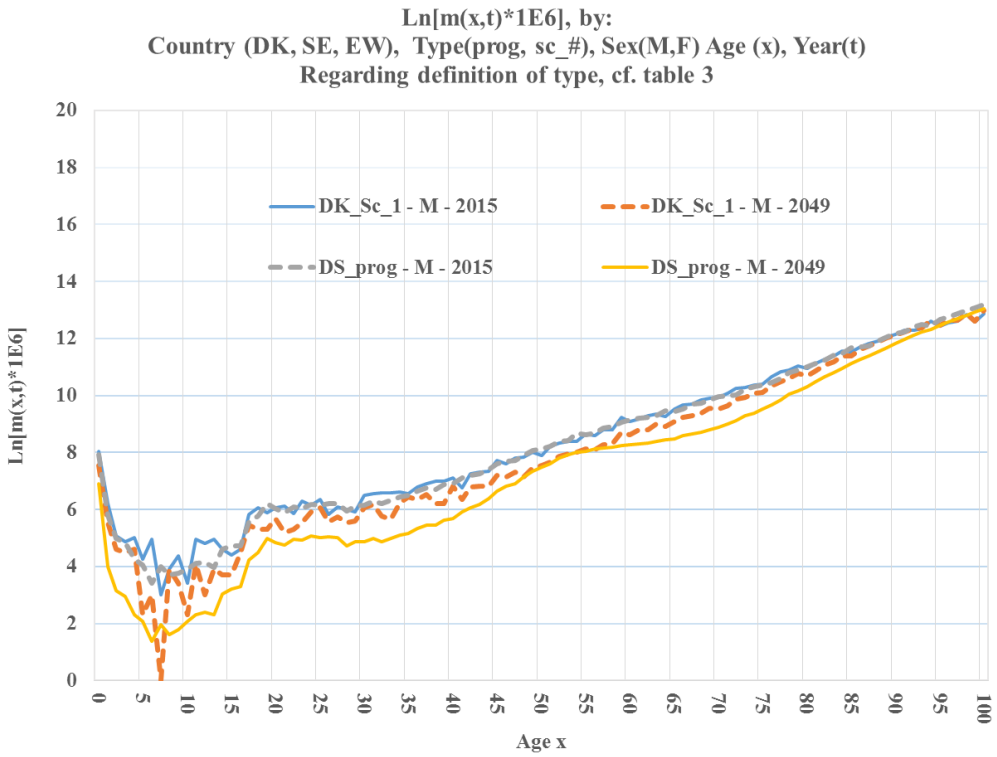
Figure 10. Projected crossectional mortality of Sweden 2015 and 2100. By sex (F, M) and projection method (SCB/LC, HSM Scenario 1). Semi-logarithmic scale+.

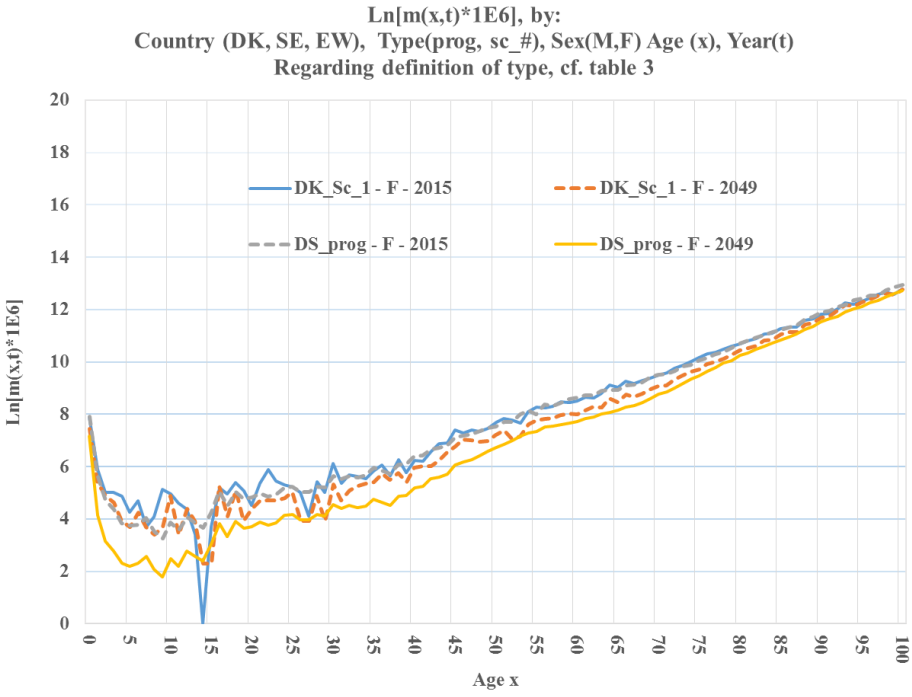




+) Cf. the life expectancies in table 3.

Figure 11. Projected crossectional mortality of Denmark 2015 and 2049. By sex (F, M) and projection method (DS/DREAM/LC, HSM Scenario 1). Semi-logarithmic scale





+.

Sweden (SE)

The Swedish official mortality projections are based on rather careful empirical and model-based trend analyses (Statistics Sweden 2012). All analyses emphasize mortality change across *sex, age, time* and to some extent *cause-of-death*. Drawing on discussions by Lee & Miller (2000), along with experience from earlier official mortality projections, the model by Lee & Carter (1992) is given special attention by Lundström & Quist (2004). Using 1985-2011 as the period of reference, the official projection beyond age 50 draws on the Lee-Carter approach as the latter is found to under valuate mortality in the younger ages. Empirical cross-sectional mortality development on a gross basis 1960 to 2011 (figure 7) strongly supports this suspicion. The problems arise as the Lee-Carter model, unlike the Heterogeneity and Selection Model, fails to effectively separating latent biological heterogeneity and *trend*.

Assuming a slightly slower mortality decline than Statistics Sweden (SCB) (tables 2, 4) how do the SCB projection and the HSM projection, alternative 1, compare across sex, years 2015 and 2100? Matching the expected age profiles as of year 2100 (figure 11) one additional drawback of the Lee-Carter model comes to surface viz. that this model systematically under values mortality in the mature and elderly ages by not taking biological heterogeneity and selection into consideration. The underrating of mortality is more pronounced of males than of females. Unsupported optimism of an official mortality projection could have far reaching economic impact for individuals and society.

The historical (1960-2011) and officially anticipated (2012-2110) annual trends of mortality are very nearly fixed (figures 5-6, table 3). Furthermore, the record low level of empirical Swedish mortality since the late twentieth century is well documented. This historical steadiness, incorporated in the official mortality projection, endows Swedish health care with respect and current official Swedish mortality projections with credibility. This, with good reasons, makes Swedish achievements an aspiration for other countries such as Denmark and England & Wales (table 3).

Denmark (DK)

The Danish official mortality projection is based on the Lee-Carter approach with extensions described by M.F. Hansen et al. (2006) and M.F. Hansen (2010). The projection is carried out by DREAM and distributed thru Statistics Denmark. No specific assumptions are reported regarding the mortality projection 2015-2049. Statistics Denmark (link

Documentation) simply refer to an alleged "extensive documentation" at the homepage of DREAM (link DREAM) which turns out to be the afore-mentioned working papers of M.F. Hansen et al. (2006, 2010)!

In virtual absence of grounds and arguments for the applied assumptions, the official mortality projection 2015 to 2049 is rather problematic (figure 11); first of all because of the prediction of unrealistically low expected mortality in the ages below 50. The precise period of historical reference for estimating the applied Lee-Carter model for Denmark is unreported. Its age component (cf. eq. 8) almost surely has been based on mortality experience covering some, if not all, of the epoch 1980 to 2011; hence favoring very low mortality among children and younger adult persons in striking contrast to mortality of mature and elderly persons. Estimating the age component by single valued matrix decomposition of historical data; and extending the age component using an empirically unsupported extreme annual global mortality decrease (cf. table 3); not surprisingly altogether prompts devastating results in terms of a distorted age structure of anticipated mortality.

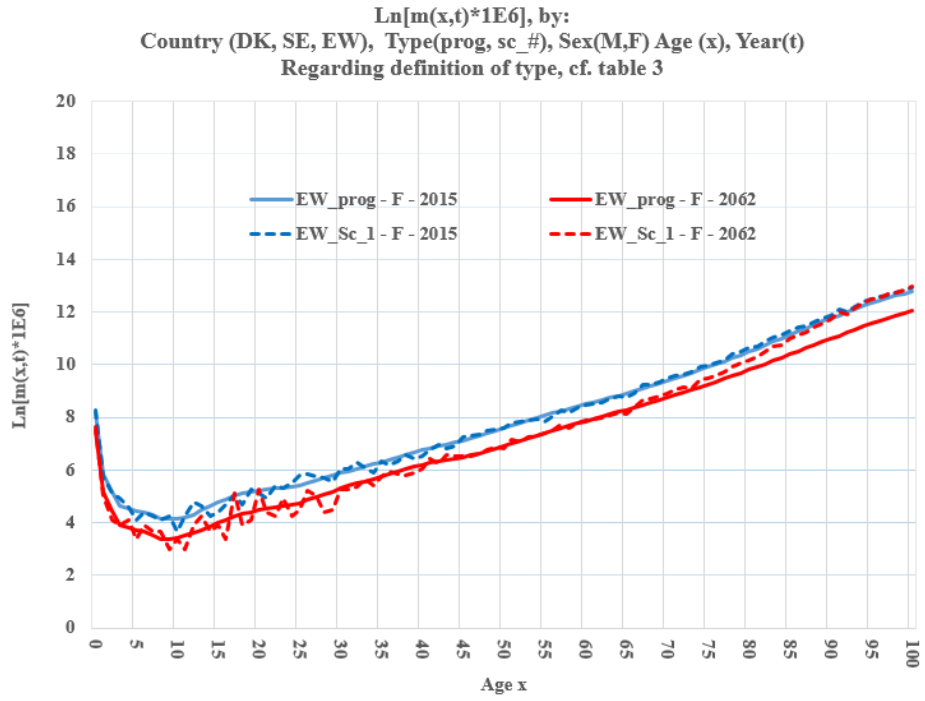
The estimated age pattern of the LC-model – whatever its factual data base - combined with the expected extreme decline of the mortality trend 2015 to 2049 (table 3) leads to the projection result based on the LC-model examplified in figure 11. Projection of Danish mortality under the longitudinal HSM-model presents a strong and constructive alderrnative to the LC-approach; cf. the fully drawn blue curve of figure 11 and the life expectancies in table 3. Further commenting on the professional quality of the Danish official mortality projection 2015 to 2049 by contrasting of the LC- and the HSM-approaches seems pointless.

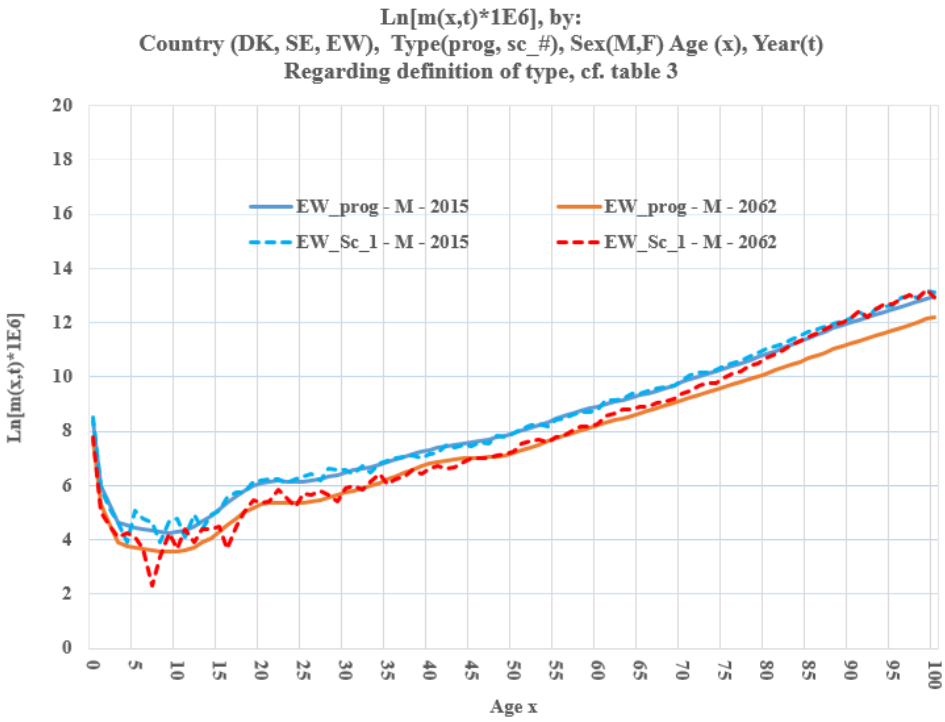
England and Wales (EW)

The assumptions of the official mortality projections of United Kingdom (England, Wales, Scotland, and Northern Ireland) are documented by Office of National Statistics (2013). For a discussion of the key forces likely to influence U.K. mortality in the twenty-first century, and a description of the methodology and assumptions used in the latest projections of English mortality cf. Gallop (2008). Assumed improvements in projected mortality rates are based on historical trends in mortality by sex, age, and birth cohort prior to the projection(s).

Figure 12 compares the official mortality projection of England & Wales, on one hand, and Scenario 1 under the HSM-model; both differentiated by sex and age. Interestingly,

Figure 12. Projected crossectional mortality of England & Wales 2015 and 2062. By sex (F, M) and projection method (Official projection, HSM Scenario 1). Semi-logarithmic scale[+].





careful cohort-based prediction based on population mortality in terms of central death rates underrates old-age mortality by leaving selection out of consideration. Note that the official projection of England and Wales operates with lower annual age-specific mortality trends than Scenario 1 under the HSM model (cf. tables 3 and 4). This should imply somehow higher old-age mortality under the HSM model, Scenario 1.

8. Summary and closing remarks

Modeling survivorship must respect three conditions; first, human existence is genetically unique; second, the body cells wear down by biological attrition over the life course; and third, individual biology interacts with the social and physical environment subject to change over time. Biological heterogeneity spurs selection in survivorship. Statistical modeling of mortality representing the data-generating process in idealized aggregated form, commonly on tacit assumption of homogeneity, fails to capture biological heterogeneity and environmental interaction and hence, selection in survivorship; the latter impacting on the shaping of the age-pattern of mortality. So far, no statistical modeling, whatever the number of parameters, has managed to make up for this problem. Absence of data on the (latent) biological forces leads to poor statistical parameter estimation.

Could quantification off the beaten track using iterative stochastic micro-simulation with cohort-based population mortality as empirical benchmark be a workable alternative strategy to quantify a model capturing latent biological heterogeneity of mortality? To investigate this hypothesis I propose a multiplicative three-factor model incorporating genetic individual frailty, biological ageing, and a period factor depending on *time*, *age*, and *sex*. The biological factors may safely be assumed fixed in time. Furthermore, long term mortality prior to the demographic transition up thru the eighteenth and early nineteenth centuries was nearly stationary. Subject to the restraints of empirical cohort mortality and fixed biological ageing shared by men and women: can a set of parameters of an appropriate probability distribution and a biological ageing function be approached to make the model-based cohort mortality statistically equivalent to empirical mortality? The answer is affirmative if we condition on stationarity of empirical mortality in traditional societies such as Sweden and Iceland prior to the demographic transition. Keeping the probabilistic frailty distributions fixed beyond the emerging mortality decline in the course of the demographic transition provides the rationale for introducing a period factor at the

level of individuals. The period factor translates into a trend factor at population level void of biological heterogeneity under the proportional hazard model.

How does mortality projection under the new biological Heterogeneity and Selection Model compare with official mortality projections and with models and algorithms currently used for projection of mortality? Two issues appear to be of basic importance at this point viz. the quality of anchoring in past survival experience and informed anticipation of the future mortality trend.

By operating on human life courses the new proportional three-factor model is longitudinal in character. Switching back and forth between longitudinal and the cross-sectional observational plans is straightforward. Furthermore, absence of biological heterogeneity of the trend factor under the main model makes the log-linear three-factor model a natural analytic workbench on evaluation and prediction of the trend factor as a function of sex, age, time, and all (two-factor) interactions. The new Heterogeneity and Selection Model prevails greatly over statistical mortality models based on empirical estimation of parameters; one case in point is the Lee-Carter model which has been widely accepted by the demographic and actuarial folklore over the past two decades. In addition to the faults and weaknesses of the Lee-Carter model, listed by Lee & Miller (2001), can be added undervaluation of late adult and old-age mortality by postponement of selection over the life course, not least in epochs of rapid mortality decrease as of present in first- and second-world countries. This is a general problem of all models and approaches based on central death rates by sex and age.

Fitting the proportional hazard model to historical and officially anticipated mortality of Sweden discloses a steady annual historical and anticipated mortality decrease of about 1.6 per cent between 1960 and 2110. This is on a par with English historical mortality decrease between 1960 and 2011. The empirical Danish mortality trend exhibits a roller-coaster development in the same period. Rather much out of keeping with past development since 1960 Denmark anticipates drastic accelerated mortality decline in the official projections to bring them at the level with Sweden by 2050. By 2062 English mortality is expected to come close to the Swedish level. Poor, inconsiderate, and mechanical use of the Lee-Carter approach discards the Danish result. The English projection quite appropriately draws on cohort differentiated trends based on central sex-age specific death rates; which, however,

also makes it blind to latent heterogeneity and therefore prone to undervaluation of old-age mortality.

The Heterogeneity and Selection Model presented in this study has proven strongly competitive to conventional statistical modeling and approaches that ignore latent biological factors such as frailty and ageing. Period factor or trend $\varphi(sex, age, time)$ is recoverable under the model. Purged of biological heterogeneity makes it the centerpiece of interest on studying social and economic heterogeneity in survivorship across people or groups of individuals. Rather than biological dissimilarities, the differences in level of mortality across the three countries considered in this study may reflect national dissimilarities e.g. in equal and affordable access to effective health care, including modern medical high-tech technology; in public awareness of the importance of healthy life style; and in quality of public governance at large; to mention just a few. Tenable inference on empirical social mobility confounded with latent biology calls for simulation of multi-dimensional stochastic processes in the framework of longitudinal observational plans. For an advanced example cf. Hansen (2000).

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Appendix A

Iterative stochastic fitting of the Heterogeneity and Selection Model to empirical cohort mortality

A.1 Stochastic appraisal of individual frailty and individual failure time

Frailty z_i

Define,

$\gamma \in [0,1]$: Uniformly distributed random number

z_i : Frailty on conception of individual # *i*

α : Form parameter of gamma distribution

β : Scale parameter of gamma distribution

Assume,

$$z_i = Gamma^{-1}\left(P\left(Z \le \gamma | \alpha, \beta\right)\right) \qquad (1)$$

Failure time x_i

Define,

t: Time

x: Age

$\overset{*}{x}_i$: Individual failure time

$\eta(x)$: Biological attrition with age of body cells

$\varphi(x,t)$: Environmental period factor

$m_i\left(z_i \eta(x) \varphi(x,t)\right)$: Mortality of individual *i*

$\tau \in [0,1[$: Expected remaining life time at age *x* in the course of age $[x, x+1[$

Assume,

$$m\left(z_i,\eta(x),\varphi(x,t)\right)=m\left(z_i,\eta(x+\tau),\varphi(x+\tau,t)\right),\tau\in[0,1[$$

$\exp\left(-m\left(z_i,x,t\right)\right)>\gamma$: Probability of surviving at least to age *x+1*

$\exp\left(-m\left(z_i,x,t\right)\right)\le\gamma$: Probability of dying in the course of age $[x,x+1[$

In the event of "dying" statistic $\tau\in[0,1[$ may be assessed as follows.

$$\tau=-\frac{1}{m\left(z_i\eta(x)\varphi(x,t)\right)}\ln\left(1-\exp(-\gamma m\left(z_i\eta(x)\varphi(x,t)\right)\right) \qquad (2)$$

A.2. Fitting the model to empirical population mortality by iterative stochastic micro simulation

Model:

$$m\left(z_i,\eta(x),\varphi(x,t)\right)=z_i\eta(x)\varphi(x,t) \qquad (3)$$

Keeping *sex* as a background variable population mortality $m(.,\mathrm{x},\mathrm{t})$ is a weighted average of individual mortality $m(\mathrm{i},\mathrm{x},\mathrm{t})$ i.e.

$$m(.,x,t)=\int_0^1 f\left(z_i\right)\eta(x)\varphi(x,t) \qquad (4)$$

$f(z)$ denoting the density function of the gamma probability distribution with parameters α and β. Let $m(emp,x,t)$ indicate empirical population mortality and $m(\mathrm{mod},x,t)$ modeled population mortality at age and time $(x,t)dxdt$ and assume again piecewise constant mortality i.e. $m(x,t)=m(x+\tau,t+\tau)$, $\tau\in[0,1[$. The task then boils down to minimizing the squared deviation between empirical and modelled cohort-based population mortality by iterative stochastic microsimulation keeping the gamma distribution fixed across lives pertaining to a specific cohort while keeping biological aging globally fixed i.e.

$$Min\left\{\left(m(emp,x,t)-m(\mathrm{mod},x,t)\right)^2\right\} \qquad (5)$$

Let top script *(n)* indicate number of iteration. Then with $\varphi^{(0)}(x,t)=1,\forall(x,t)$, we proceed as follows.

Step 1 Compute $m^{(n)}(\text{mod},x,t)$

Step 2
$$\varphi^{(n+1)}(x,t)=\varphi^{(n)}(x,t)\frac{m^{(n)}(\text{mod},x,t)}{m(emp,x,t)} \tag{5}$$

Step 3
$$m^{(n+1)}(\text{mod},x,t)=\varphi^{(n+1)}(x,t)m^{(n)}(\text{mod},x,t) \tag{6}$$

Repeat step 1-3 until an arbitrary minimum (random) tolerance has been achieved. Normally conversion of $m^{(n)}(\text{mod},x,t)$ to $m(emp,x,t)$ is fast, $n\le 30$ should be more than enough.

Appendix B

A Summary of Historical Mortality, Projections and Graphical Pivot Table Applications Used and Produced in This Project

Data

- Central mortality rates (one-year groups)
    - Source: Human Mortality Database, University of Berkely, USA
        Sweden 1751-2011
        Denmark 1835-2011
        England & Wales 1842-2011
    - Variables: sex, age, year
    - Observational plan: cross-sectional
- Official mortality projections
    - Source: National Statistical Offices
        Sweden (SCB) 2015-2110
        Denmark (DS) 2015-2049
        England & Wales 2015-2062
    - Variables: sex, age, year
    - Observational plan: cross-sectional
- Derived statistics
    - Life tables, cohort-based and cross-sectional, age [0, 94[

Interpretations and projections under model (eq. 4)

- Individual failure times, historical mortality plus official projections:
    - Stochastic micro-simulation,
        Number of iterations: 30
        Cohort size: 100,000 individuals
    - Variables: iteration #,ID, sex, frailty, birth date (year), age-at death
    - Countries,
        Sweden (SCB) 1901-2110
        Denmark (DK) 1901-2110
        England & Wales 1901-2110
    - Three scenarios
- Statistics based on individual failure times (derived statistics):
    - Central death rates by birth year, sex, and age
    - Life tables, age [0, 94[, cohort-based and cross-sectional
    - Graphics,
        Individual frailty against individual failure time (point swarms)
        Power pivoting with graphics

Appendix C

Examples of postponement of mortality over the life course in Epochs of historical and anticipated long term decline of mortality. By level of aggregation.

C.1 Individual mortality

Figure C.1 exhibits a sample of micro simulated gamma distributed biological frailties graphed against individual failure time (age-at-death). The dashed horizontal lines indicate deciles of the gamma density. The frailties refer to Swedish male cohorts born in 1900 and 1958, and expected to be born in 2017 according to official mortality projection.

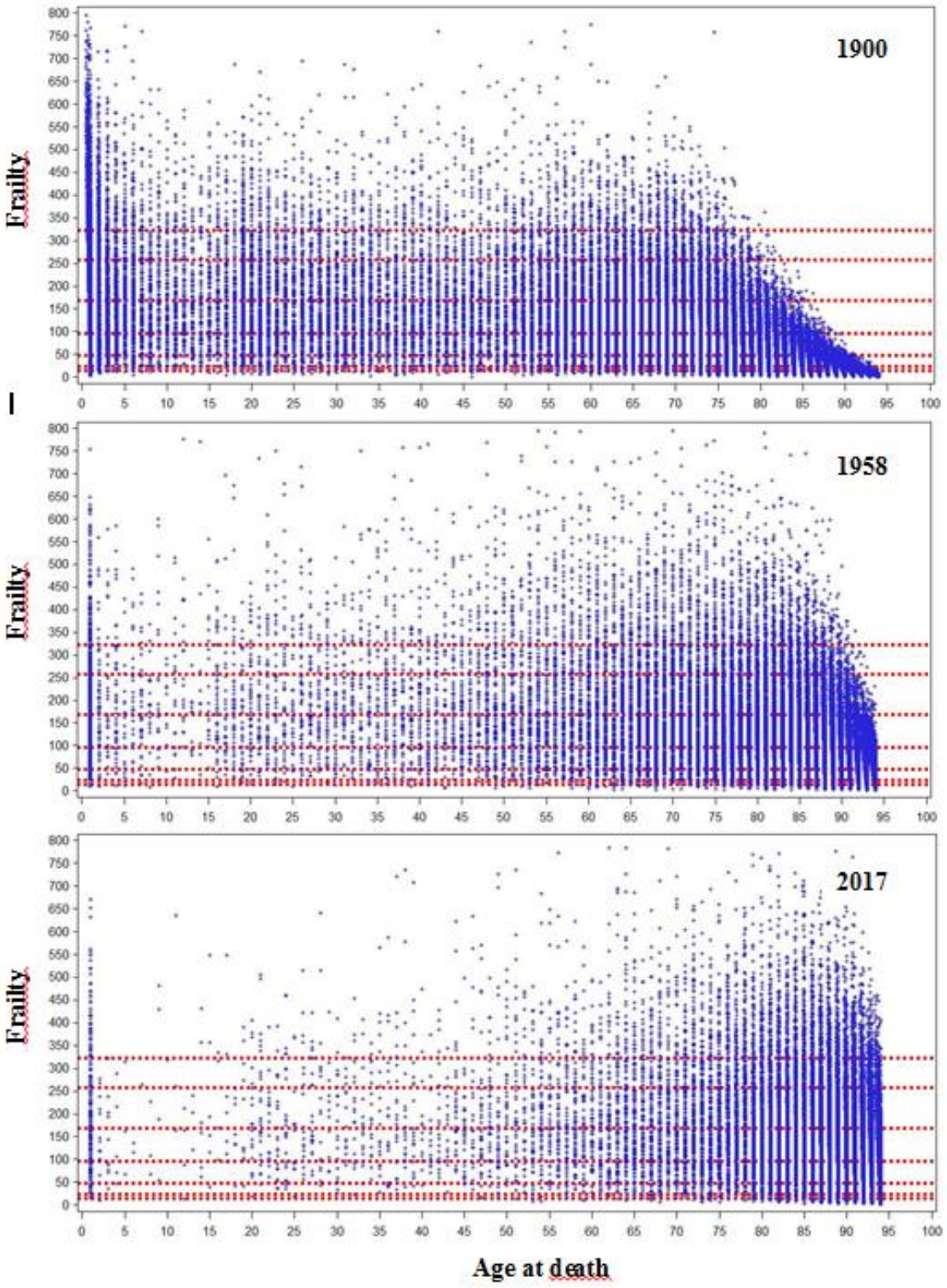
- Individuals with high frailties evidently profit the most from the general mortality decline in the course of the twentieth century due to postponement of selection.
- Frailty most probably is positively correlated with health. The mortality effects of aggravating public health to not a small extent could be expected to be counteracted and dampened by deployment of effective public access to modern health care and medical technology.
- As the official mortality projection underlying the expectation of the 2017 cohort does not account for biological heterogeneity it probably undervalues mature-age and old-age mortality. Hence the picture drawn by 2017 cohort may be somehow optimistic regarding capacity to staying alive to very old ages

C.2 Historical population mortality

Figure C.2 shows elected cohort schedules of historical population mortality divided into three groups. The first group refers to cohorts born and becoming extinct before the general long term decline of mortality; the second group illustrates transition from high to low mortality; and the third group cohorts exposed to comparatively low mortality throughout their entire existence.

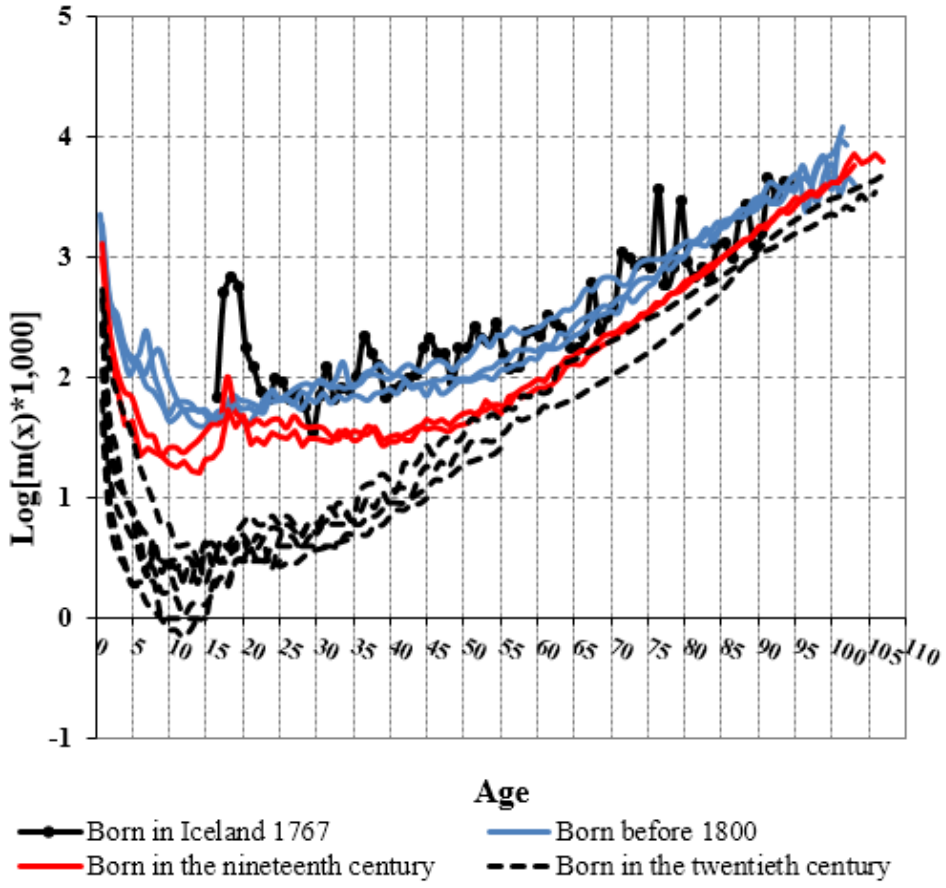
In striking contrast to infants, children, and younger adults who have profited immensely from the modern long term decline of mortality, this is in general by no means the case with persons in their mature and old ages. As indicated by figure C.1 in any population there will be some that live to very old ages viz. those with low biological frailties. The lesson is that the national populations have become a lot frailer in the course of the demographic transition.

Figure C.1. Personal frailty plotted against individual age at death. Swedish male cohorts born 1900, 1958, and 2017. With indication of decile intervals (horizontal dashed lines).



Source: Hansen (2014).

Figure C.2.. Empirical mortality of elected female cohorts born before 1800 and in the course of the nineteenth and twentieth centuries (semi-logarithmic scale)



Note

- Cohorts born before 1802:
  Iceland 1767;
  Sweden 1751, 1801
- Cohorts born between 1802 and 1900:
  Denmark 1835 and 1851;
  Sweden 1851
- Cohorts born in the twentieth century:
  Sweden and Denmark 1901, 1944, 1952;
  Japan 1950, 1960, 1970, 1980, and 1990

Sources

Sweden, and Denmark

- Berkeley Mortality Data Base

Iceland

- Icelandic mortality recovered by Hansen 2004.

Japan

- Berkeley Mortality Data
- Base Statistics and Information Department, Minister's Secretariat, Ministry of Health, Labor and Welfare "Vital Statistics" (Japan)
- Statistics Bureau, the Director-General for Policy Planning (Statistical Standards) and the Statistical Research and Training Institute (Japan)

Source: Paper presented at the Seminar on Lifespan Extension and the Biology of Changing Cause-of-Death Profiles: Evolutionary and Epidemiological Perspectives, organized by the IUSSP Scientific Panel on Evolutionary Perspectives in Demography, Rauischholzhausen, Germany, 13-15 January 2011.