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Issues of Selection in Human Survivorship:
A Theory of Mortality Change from the Mid-Eighteenth
to the Early Twenty First Century

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Issues of Selection in Human Survivorship – A Theory of Mortality Change from the Mid-Eighteenth to the Early Twenty First Century

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Abstract

Is variation in empirical mortality across populations consistent with a hypothesis of selection? To examine this proposition an extended frailty mortality model is put forward; incorporating biological frailty; a common non-parametric hazard, joint for men and women, representing endogenous mortality in terms of degenerative aging (senescence); and environmental influence on survivorship. As the model is fitted to empirical cohort mortality exhibiting extreme variation, biological aging is identified up to a multiplicative factor. Mortality of elected cohorts born in Sweden, Denmark, and Iceland during the past 250 years and in Japan any ten years between 1950 and 1990 is approached appropriately by the model. Reduced natural selection may account for a substantial part of the empirical mortality change in the course of the demographic transition. Survivorship in the late nineteenth and the twentieth century ties selection to major medical advances and rapid recent mortality decline, probably with consequences for future health and survivorship.

Keywords: Biodemography; congenital frailty; selection; heterogeneity; cohort mortality; stochastic micro-simulation; longevity

JEL codes: C6, C8, I12, J1

Introduction

Genetic heritage instigate selection in survivorship. Is heterogeneity in empirical mortality over the past three centuries consistent with a hypothesis of selection? Modern highlights in the history of demographic and statistical modeling of heterogeneity in human survivorship include contributions of Gompertz (1825), Makeham (1860), Cox (1972), and Vaupel et al.

(1979). The Makeham law extends the Gompertz function by introducing an additive death risk which is independent of age. The Gompertz part of the Gompertz-Makeham law describes mortality as an exponential function of age. The Cox model broadens this view by introducing a shared unspecified baseline intensity depending on time or age; multiplicatively related to an exponential function admitting multivariate personal characteristics which may be fixed or vary over time.

An extension of multiplicative hazard modeling of the role of genetic heritage is due to Vaupel et al. (1979), who depict mortality as a multiplicative function of congenital biological frailty at the level of individuals and a joint baseline hazard depending on age. Several important questions are left open. For example, how do environmental factors during gestation influence the frailty distribution of live births in a cohort? Moreover, Vaupel et al. (1979) neither consider choice or interpretation of their baseline mortality, nor do they reflect on environmental interaction with human survivorship after birth.

In weighty empirical work Bourgeois-Pichat (1946, 1951) singles out so-called endogenous and exogenous elements in infant mortality. The embedded statistical model is basically a competing risks approach. Examining age-cumulated mortality $\Lambda_{[0,x]}$ as a function of age x , Bourgeois-Pichat noted that the transform $\ln \Lambda_{[0,x]}$ plotted against $\ln(\ln(x+1))$ would be linear over the age segment $[x_0, 1^{\text{st}} \text{ birthday}]$ for a very wide range of populations with reliable mortality data. This finding translates into the linear model $\ln(\Lambda_{[x_0,x]}) = b \ln(\ln(x+1))$. Usually age x_0 is set to one month with age x equal to 1st birthday and parameter b equal to 3. By back-projection to age 0 sharp from age x_0 noting that empirical mortality in age $[0, x_0]$ is dominated by endogenous biological factors, Bourgeois-Pichat (1951) determined endogenous mortality as the difference $\Lambda_{x_0} - \Lambda_0$. This finding based on essentially descriptive statis-

tics leads to important conclusions. First, endogenous biological factors dominate neonatal mortality while post-neonatal mortality is governed by exogenous environmental factors. Second, in populations with general access to steadily improved medical technology the endogenous element has emerged as the principal determinant of infant mortality. In old first-world countries this development dates back to the great medical advances by distinguished researchers such as Pasteur, Koch, and Lister in the late nineteenth century followed by Fleming and Salk in the early and mid-twentieth century. Like earlier statisticians and demographers, Bourgeois-Pichat ignored natural selection.

Un-modeled heterogeneity is a frequent source of error in prediction and forecasting of population change. Assessment of future mortality by mere extrapolation of time series of expected length of life based on cross-sectional data is risky. At a mortality crisis life expectancies may be lower than 20 years; for example from 1784 to 1786 succeeding extreme earthquakes and volcanism in Iceland around 1783 (Stephensen 1785; Thorarinsson 1968). Life expectancies may also prove deceptive during structural change and long term decrease of period mortality associated with the demographic transition. Cross-sectional Japanese survivorship in the late twentieth and early twenty-first century ties selection to rapid recent mortality decline and major medical advances.

With Bourgeois-Pichat's distinction between basic biological and environmental factors in infant mortality as a point of departure, I shall consider latent heterogeneity and selection in cohort-based mortality to explain human survivorship through the entire life course. Following Vaupel et al. (1979) the biological component is modeled as a personal congenital frailty and an age-dependent baseline hazard. I use *aging* and *senescence* as synonyms. Masoro (2006) defines senescence as "The progressive deterioration during the adult period of life that underlies an increasing vulnerability to challenges and a decreasing ability of the organism to survive". The baseline hazard governs aging over the life course. The model ac-

commodates environmental influence either with or without selection. Infections, hunger, personal life style, and medical technology are examples of environmental influence with selection. External mortality without selection occurs in situations where any member of a population-at-risk is under exposure to the same instantaneous death risk independently of personal congenital frailty. This particular type of death risk is more difficult to pinpoint empirically. It is associated with catastrophe, that is great and sudden disaster or misfortune (The Free Dictionary 2008).

I shall use this model to comment upon empirical mortality change over the past three centuries in modern industrialized societies. Does empirical cohort-based population mortality on reliable statistical record support the hypothesis of selection which is unobservable across persons and in the population? Is there a built-in general death factor? Or would we achieve immortality be if all diseases were defeated (Rubin 2007)? Can an age-dependent biological baseline be identified which is independent of sex and birth cohort? What is the environmental influence on human survivorship with selection? How will the likely future pace of biological and technological change affect human survivorship?

The countries chosen for application of the model must possess long historical time series or well-documented rapid recent mortality decline. Furthermore, to avoid confusing external mortality with selection as distinct from external mortality without selection, the countries must have suffered very few or no casualties as a direct consequence of major catastrophes on own ground from birth to extinction or right-truncation of the cohorts. Subject to these restraints I consider male and female cohorts born in Iceland (1767), Sweden (1751, 1801, 1851, 1901, 1944), Denmark (1835, 1901, 1944), and in Japan any ten years between 1950 and 1990. Cohorts born in the nineteenth century are observed up to 2005.

After presenting the frailty model, I apply it to mortality of the elected birth cohorts.

I show that it is possible to fit the extended frailty model to empirical mortality of the elected male and female cohorts using the same baseline hazard and the same trend for men and women. The main difficulty lies in identifying a suitable trend in epochs with rapid mortality change and affecting specific age classes in twentieth century Sweden and Denmark, notably in the wake of the Spanish Flu in 1918, and after the Second World War up through the early 1950s.

Model

In stochastic survivorship models, selection and heterogeneity is commonly introduced either as a covariate vector depicting individual congenital frailty (weakness) or as sojourns in specific states under exposure to forces of transition. Yashin et al. (2001) point out that hidden difference in the survival chances of individuals in a population may greatly influence the shape of mortality. To study heterogeneity and selection of survivorship I propose the mortality model.

$$m_i(x, t) = z_i \cdot m(x, z = 1) \cdot \varepsilon(t) + \delta(t) \quad (1)$$

The hazard $m(x, z = 1)$ refers to biological mortality; functions $\varepsilon(t)$ and $\delta(t)$, t denoting calendar time, represent the environmental effect on mortality. Statistic z_i , fixed over the life course, represents congenital frailty of individual i .

Congenital frailty remains unknown. Reproductive health varies across individuals and populations (Leridon 1977). As a function of biological frailty, the frailty distribution of live births in a cohort is select by environmental factors during gestation. In absence of information on survivorship during gestation I follow Vaupel et al. (1979) and assume Z to be a gamma distributed random variate. The gamma distribution is a simple and flexible tool to introducing heterogeneity in survivorship. The distribution has three parameters namely α

(shape), β (scale), and γ (location). With $\gamma = 0$ the gamma variate Z has mathematical expectation $E[Z] = \alpha\beta$ and variance $VAR[Z] = \alpha\beta^2$; $\alpha, \beta > 0$. The most successful survivors would be those with relatively low frailty values. The variable Z and the process $\varepsilon(t)$ are independent from another in the model. They cause selection in mortality represented by the cohort life table $\ell(x)$, the random element being time to death.

Following Prentice and Kalbfleisch (1980), Cox and Oakes (1984), and Andersen et al. (1993) I interpret $m(x, z = 1)$ as a baseline hazard shared by all individuals. I see the joint baseline hazard as an integral component of biological mortality, which is heterogeneous by the multiplicative relationship of the baseline hazard and frailty z_i . A person with $z = 1$ has a biological mortality equal to the baseline mortality. If baseline mortality has changed only little for centuries, it should be possible to describe a wide range of cohort-based mortality with the same baseline mortality.

The factor $\varepsilon(t)$ is the trend of exogenous mortality with selection and hence an integral component of mortality; the magnitude of $\varepsilon(t)$ has shifted over time. Bourgeois-Pichat (1952, 1978) emphasized the importance of exogenous causes of death such as infectious diseases. In traditional societies environmental factors would instigate selection from an early age. Conversely, in developed societies with modern health prophylaxis and general public access to medical treatment, environmentally induced selection has been stalled to a high degree: The prospect that a frail person would live on to mature or old age is greater among current generations than among cohorts born two or three centuries ago. Reducing the trend factor $\varepsilon(t)$ defers selection to ages with higher biological mortality. However, individuals live also to extreme ages even in traditional societies. This is accommodated by the probabilistic nature of the model.

Environmental mortality $\delta(t)$ refers to shocks of mortality where members of a risk set are exposed to the same instantaneous extra death risk independently of personal congenital frailty. The direct casualties of the tsunami in South-East Asia Christmas 2004 and the immediate victims of the earthquakes that struck northern Pakistan in October 2005 are examples of environmental shocks that may have caused little or no selection of the risk populations.

Evaluating selection in human survivorship with the extended frailty model

Caused by latent individual frailty and environmental change, how far can selection explain variation in empirical mortality? We minimize the deviation of the model-based cohort mortality $m_{x_k}(z_{\{i\}}, m(x_k, z=1), \varepsilon(t), \delta(t))$ from the estimated empirical cohort mortality \hat{m}_{x_k} :

$$\sum_{\forall k} \left(m_{x_k}(z_{\{i\}}, m(x_k, z=1), \varepsilon(t), \delta(t)) - \hat{m}_{x_k} \right)^2 \quad (2)$$

The model-based cohort mortality $m_{x_k}(z_{\{i\}}, m(x_k, z=1), \varepsilon(t), \delta(t))$ in Eq. (1) is computed using a set of personal lifetimes $\{X_i\}$ obtained by stochastic micro-simulation as appropriate empirical event histories are unavailable. The associated survivor process is defined on the state space $S = \{\text{Alive}, \text{Dead}\}$. The lifetime X_i of individual i is a random outcome governed by hazard $m_i(x_k, t)$. Mortality and life table statistics result from the set $\{x_i\}$ of simulated individual life times (Hansen 2000).

To make the model-based mortality $m_{x_k}(z_{\{i\}}, m(x_k, z=1), \varepsilon(t), \delta(t))$ operational we postulate a multiplicative relationship between congenital frailty $Z = z_i$ and environmental effect $\varepsilon(t)$. Let t_0 denote time at birth. Being select by biology and environment prior to live

birth, the distribution of z_i as of time t is not purely biologically determined. Furthermore, identification of the effect of z_i and $\varepsilon(t_0)$ is only possible up to a multiplicative factor.

Therefore we choose to fix $\varepsilon(t_0)$ to 1.

As a linear function of α and a polynomial function of β , $VAR[Z]$ is the main determinant of selection of survivorship, and thereby of hidden heterogeneity in mortality. A given value of $VAR[Z]$ corresponds to many combinations of parameters α and β .

The initial mean of the gamma distribution is obtained by dividing $\hat{m}(0, t_0)$ (empirical rate of infant mortality) and infant mortality $m(0, z = 1)$ according to the baseline hazard. From Eq. (1), for $x = 0$ with $\varepsilon(t_0) = 1$ and extrinsic mortality $\delta(t_0) = 0$,

$$\begin{aligned} E[z_i] &= \hat{m}(0, t_0) / m(0, z = 1) \\ &= \alpha\beta \end{aligned} \tag{3}$$

The expectation $E[z_i]$ being fixed, the possible values of α, β are located on a hyperbola in the first quadrant. Fitting Eq. (1) to empirical cohort-based mortality by iteration boils down to evaluating Eq. (2) for pairs (α, β) , $\alpha, \beta > 0$, $\alpha\beta = \text{constant}$, as a first approximation. Fine tuning is done by keeping α fixed while changing $VAR[Z]$ at appropriate step lengths. The resulting β is $(Est. VAR[Z = z] / \alpha)^{1/2}$. I create the stochastic frailty $z_i = Z_i$ by drawing a random number $v_i \in [0, 1]$ from the uniform probability distribution. Let

$$v_i = P(Z_i \leq z_i | \alpha, \beta, \gamma = 0), \quad z_i > 0, \text{ and solve with respect to } z_i \text{ or } z_i = P^{-1}(v_i | \alpha, \beta, \gamma = 0).$$

Baseline hazard $m_s(x, z = 1)$ is unobservable. If the shape of intrinsic mortality has changed little it should be possible to isolate a non-parametric baseline hazard general enough to make Eq. (1) comply with known cohort-based mortality experience.

A simple and natural estimator of trend $\varepsilon(t)$ of mortality with effect on selection is,

$$\varepsilon(t) = \hat{m}(t) / \hat{m}(t_0) \quad (4)$$

t_0 denoting time at the birth of the cohort, and $\hat{m}(t)$ referring to sex-age standardized period mortality at time $t, t \geq t_0$. Empirical demographic time series are normally reported in years.

Alternatively, $\varepsilon(t)$ may be estimated by fitting a log-linear intensity model with the factors *age* and *time* to a time series of occurrence/exposure data for a period of length n years, nor-

malized so that $n = \sum_{t=t_0}^{n+t_0} \varepsilon(t)$, cf. Hoem (1987) for example. High values of $\varepsilon(t)$ should in-

crease selection. Decreasing values of $\varepsilon(t)$ reduce temporal selection by postponing death; diffusion of modern hygiene, prophylaxis, and deployment of modern medical and biotechnical know-how would have such an effect.

Depending on past survivor experience, the population at risk may respond differently to external mortality at time t . Persons selected by having survived momentarily high period mortality earlier in life may react differently from persons having been less exposed. Estimated by Eq. (4) trend $\varepsilon(t)$ of period mortality with selection is an average of such age-specific responses. If age is interacting with time, est. $\varepsilon(t)$ may underrate the trend of mortality of young people; conversely the trend of mortality of elderly persons might be somewhat overrated. Medical advances or change of life style may influence exogenous mortality with selection differently across gender and age.

To avoid confusing $\delta(t)$ with $\varepsilon(t)$ in the applications to follow, aiming at fitting Eq. (1) to mortality of the elected cohorts as a joint baseline $m_s(x, z = 1)$ is identified, $\delta(t)$ is set to nil. I focus on populations with long historical time series where it is safe to expect that extrinsic mortality $\delta(t)$ with no effect on selection has played a negligible role for centuries. Once the

baseline has been established evaluating cohort survivorship for values of $\delta(t)$ greater than nil is straightforward.

Data

To evaluate the capability of the model to approach empirical mortality, I fit Eq. (1) to the elected birth cohorts presenting extreme variation in empirical mortality across cohorts. The Swedish, Danish, and Japanese data were retrieved from the Berkeley Mortality Database supplemented with data from national statistical data banks. As a matter of fact subject to extensive interpolation and smoothing (Wilmoth et al. 2006) I shall assume that the Berkeley data portrait empirical mortality. The unpublished Icelandic data were collected by Hansen (1966). Embedded in scarce annual distributions of deaths by sex and age between 1783 and 1870, empirical Icelandic mortality was uncovered by the author in 2004 using iterative proportionate fitting and projection back and forward in time with the population censuses of 1801, 1835, and 1845 as starting points. Adapting Eq. (1) to the empirical adult mortality of the Icelandic 1767 cohort offers a probably unique opportunity of checking the capacity of the extended frailty model to describe extreme mortality with selection as an aftershock of a major environmental disaster. The lava flow did not directly cause any loss of life (Thorarinson 1969). All cohort data is grouped by sex and one-year age intervals. To evaluate the coherence of official anticipations of future mortality, the Swedish and Danish time series have been updated with official anticipations of mortality between 2005 and 2050.

Figure 1 shows the demographic transitions of Sweden and Denmark with indication of the birth years of the elected birth cohorts of each population. Period mortality of the birth years is strongly heterogeneous as the cohort-based mortality experience of contemporaneous survivors differs widely over time.

Figure 1 The demographic transition of Sweden and Denmark

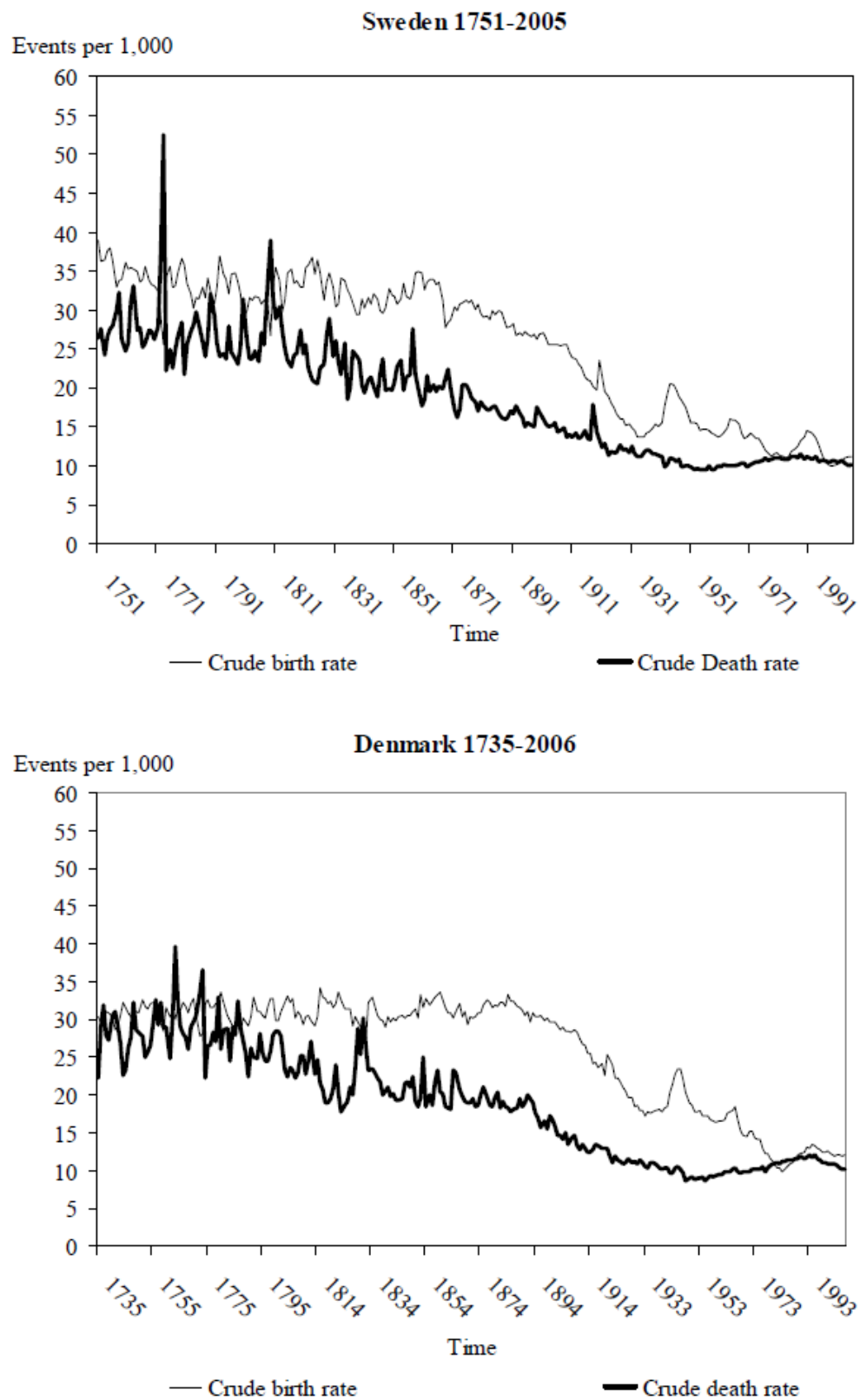
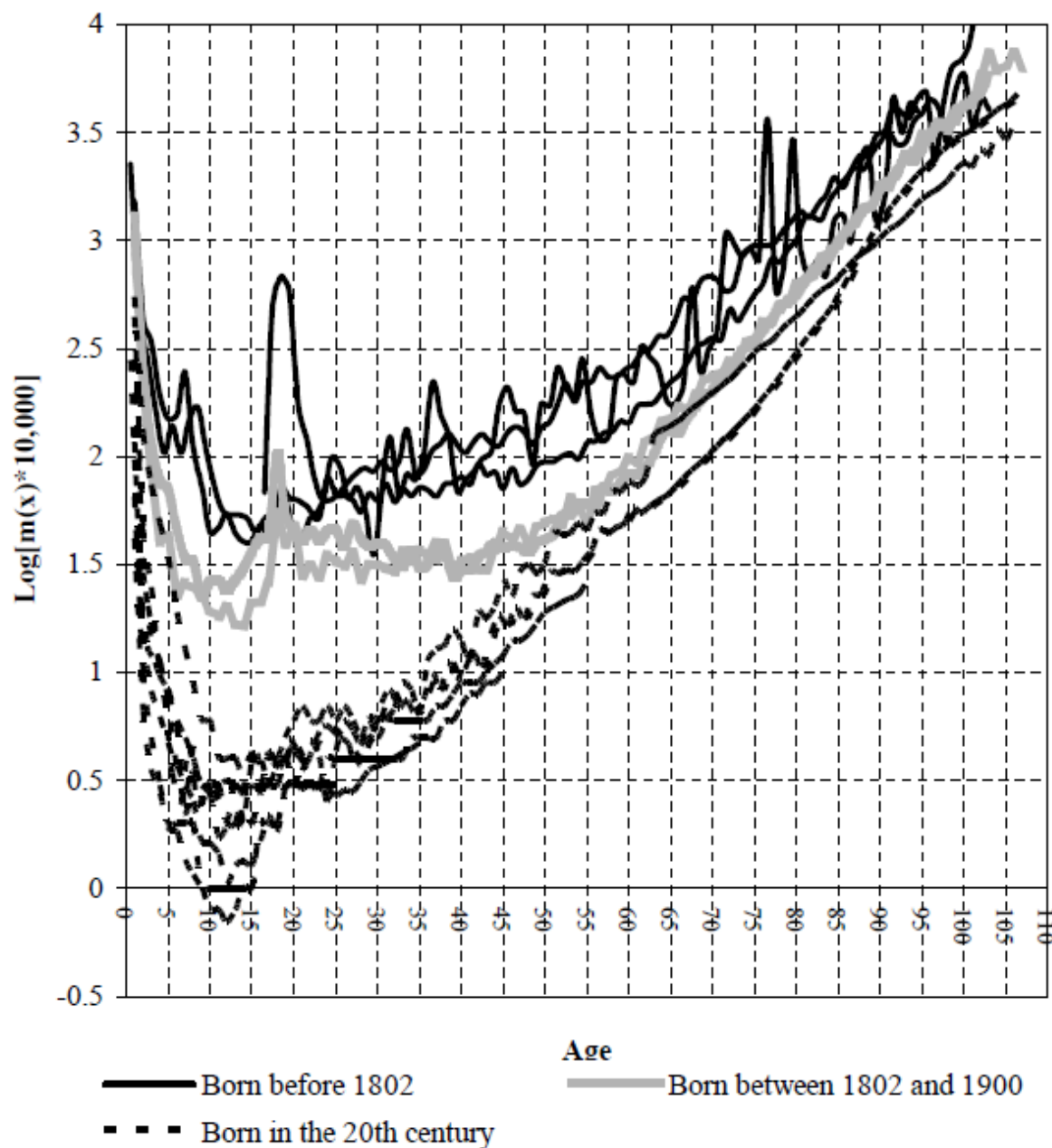


Figure 2 Empirical mortality of elected female cohorts born before 1802 and in the course of the nineteenth and twentieth centuries (Semi-logarithmic scale)



Source.

Based on data from Berkeley Mortality Data Base and mortality recovered by Hansen 2004.

Note.

Cohorts born before 1802: Iceland 1767; Sweden 1751, 1801

Cohorts born between 1802 and 1900: Denmark 1835; Sweden 1851

Cohorts born in the twentieth century: Sweden and Denmark 1901, 1944; Japan 1950, 1960, 1970, 1980, and 1990

Figure 2 underscores that chances of survival were very different over the elected births cohorts. Men have higher mortality than women. The elected cohorts may naturally be regrouped into three: those born up to 1801, those born in course of the nineteenth century, and those born after 1901. The difference between any pair of graphs indicates proportionality because of the logarithmic scale for mortality.

As a member of the group of cohorts born before the modern long term decline of mortality, the Icelandic 1767 cohort (Figure 3) shows extreme mortality beyond human control. In the latter half of the eighteenth century Swedish mortality was much lower than mortality in Iceland. Up to the late nineteenth century, health and living conditions in Iceland may best be described as a wintering medieval regime.

The mortality of the Swedish and Danish cohorts born in the first half of the nineteenth century (Figure 2) is transitory in the sense that the cohorts spent their childhood and teen-years in epochs with relatively high mortality; however, they lived to reap the benefits of innovation and accelerating medical and other life-preserving technological advances later in the century. The cohorts born in the latter half of the nineteenth century and beyond came of age in an era with increasing human control of exogenous mortality with selection. The difference of Swedish and Danish cohort mortality is surprisingly small before 1970. Japanese mortality of the cohorts born any ten years between 1950 and 1990 is low and exhibits substantial decrease in the latter half of the twentieth century.

Results

After numerous stochastic micro simulations using the empirical population mortality of the elected birth cohorts (Figure 2) as targets, attempts of determining a latent baseline shared by men and women and all elected cohorts turned out successful. The joint baseline $m(x, z = 1)$

Table 1. Gamma parameters obtained on fitting Eq. (1) to empirical mortality of the elected birth cohorts

Country, Gender, and Birth Year	Initial estimate of Congenital Mean Frailty	Gamma distribution			
		Shape	Scale	$E[Z = z]$	$VAR[Z = z]$
T_0	$\hat{z} = \frac{\hat{m}(x=0, T_0)}{m(x=0, z=1)}$	α	β	$\alpha\beta$	$\alpha\beta^2$
1	2	3	4	5	6
Sweden					
Men					
1751	90.319	1.57	57.274	89.919	5,150
1801	97.739	1.44	69.472	100.040	6,950
1851	70.841	0.85	118.57	100.784	11,950
1901	45.875	0.97	47.294	45.875	2,170
1944	14.274	0.62	24.429	15.146	370
Women					
1751	79.636	1.54	52.533	80.901	4,250
1801	85.546	1.07	89.391	95.648	8,550
1851	57.842	1.03	61.927	63.785	3,950
1901	37.581	0.64	58.721	37.581	2,207
1944	10.537	0.36	27.889	10.040	280
Denmark					
Men					
1835	89.809	1.03	90.575	93.293	8,450
1901	62.655	0.51	133.578	68.125	9,100
1944	21.683	0.62	24.429	15.146	370
Women					
1835	70.667	0.92	86.916	79.963	6,950
1901	49.175	0.51	101.460	51.745	5,250
1944	16.828	0.30	57.009	17.103	975
Japan					
Men					
1950	23.587	1.20	19.656	23.587	463.6
1960	12.969	0.55	23.581	12.969	305.8
1970	5.802	1.15	5.045	5.802	29.3
1980	3.110	1.15	2.704	3.110	8.4

Table 1 Continued ...

Country, Gender, and Birth Year	Empirical Infant Mortality	Sum of Squared Deviations by age 80	Life expectancies $e_{[0,100]}$			Model Based Infant Mortality Est. m_0
			Emp. Life table	Model Based Life Table	Standard Deviation of Model based Life Expect- ancy	
T_0	$\hat{m}(x=0, T)$	SSD				
1	2	3	4	5	6	7
1990	0.005	*	*	85.4	0.861	0.005
Women						
1950	0.055	*	*	75.1	0.793	0.053
1960	0.028	*	*	82.3	0.848	0.027
1970	0.012	*	*	83.3	0.846	0.012
1980	0.007	*	*	86.8	0.877	0.006
1990	0.004	*	*	88.0	0.886	0.004
Iceland						
Men						
1767	*	0.0571	*	26.9	0.393	0.445
Women						
1767	*	0.0448	*	31.6	0.447	0.425

Sources and note cf. Table 1.

Sources.

Sweden, Denmark, and Japan up to 2005: Computations based on the Berkeley Mortality Data Base. Sweden 2005-2050: Statistics Sweden (2006). Denmark 2005-2050: Unpublished data from Statistics Denmark. Iceland 1767: Unpublished mortality recovered by Hansen in 2004.

Note.

Symbol *: Statistic undefined.

is non-parametric. As a non-parametric function of age, the baseline accounts for aging over the human life course. The expected length of life at birth of the identified baseline hazard $m(x, z=1)$ is 85.6 years.

The age pattern of the baseline hazard is the focus of interest. It is defined as follows.

$$m_{NORM}(x, z=1) = \left(m(x, z=1) / \sum_{x=0}^{Max(x)} m(x, z=1) \right) * 10^6 \quad (5)$$

Table 2. Demographic results obtained on fitting Eq. (1) to empirical mortality of the elected birth cohorts

Country, Gender, and Birth Year	Empirical Infant Mortality	Sum of Squared Deviations by age 80	Life expectancies $e_{[0,100]}$			Model Based Infant Mortality Est. m_0
			Emp. Life table	Model Based Life Table	Standard Deviation of Model based Life Expect- ancy	
T_0	$\hat{m}(x=0, T)$	SSD				
1	2	3	4	5	6	7
Sweden						
Men						
1751	0.238	0.0069	36.6	37.6	0.475	0.220
1801	0.258	0.0062	36.2	37.2	0.481	0.246
1851	0.187	0.0039	43.9	42.8	0.542	0.227
1901	0.121	0.0015	56.8	55.6	0.634	0.115
1944	0.038	0.0006	75.5	74.5	0.784	0.039
Women						
1751	0.210	0.0074	39.8	40.2	0.497	0.202
1801	0.226	0.0029	40.6	40.3	0.511	0.221
1851	0.153	0.0035	47.5	49.7	0.590	0.155
1901	0.099	0.0017	61.8	60.7	0.679	0.093
1944	0.028	0.0003	80.9	80.4	0.832	0.026
Denmark						
Men						
1835	0.237	0.0025	42.2	41.3	0.522	0.224
1901	0.165	0.0115	56.3	54.7	0.643	0.159
1944	0.057	0.0006	75.2	74.5	0.784	0.055
Women						
1835	0.186	0.0027	45.3	45.1	0.552	0.190
1901	0.130	0.0009	61.7	58.9	0.673	0.122
1944	0.044	0.0037	75.9	77.0	0.812	0.044
Japan						
Men						
1950	0.062	*	*	70.5	0.754	0.062
1960	0.034	*	*	78.0	0.811	0.033
1970	0.015	*	*	81.5	0.831	0.015
1980	0.008	*	*	83.7	0.847	0.007

Table 2 Continued ...

Country, Gender, and Birth Year	Empirical Infant Mortality	Sum of Squared Deviations by age 80	Life expectancies ${}^0e_{[0,100]}$			Model Based Infant Mortality Est. m_0
			Emp. Life table	Model Based Life Table	Standard Deviation of Model based Life Expect- ancy	
T_0	$\hat{m}(x=0, T)$	SSD				
1	2	3	4	5	6	7
1990 Women	0.005	*	*	85.4	0.861	0.005
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1960	0.028	*	*	82.3	0.848	0.027
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1767	*	0.0448	*	31.6	0.447	0.425

Sources and note cf. Table 1.

The actual baseline identified in this study (Figure 9) belongs to a set M of multiplicative transformations of the normalized baseline $m_{NORM}(x, z=1)$:

$$M = \{m(x, z=1) = \nu \cdot m_{norm}(x, z=1)\}, \forall x, \nu > 0 \quad (6)$$

The crude and the normalized baseline hazards are proportional on the metric scale; the non-negative factor ν of proportionality being independent of age.

A summary of results on fitting Eq. (1) to the empirical mortality of the elected cohorts is shown in Tables 1-2.

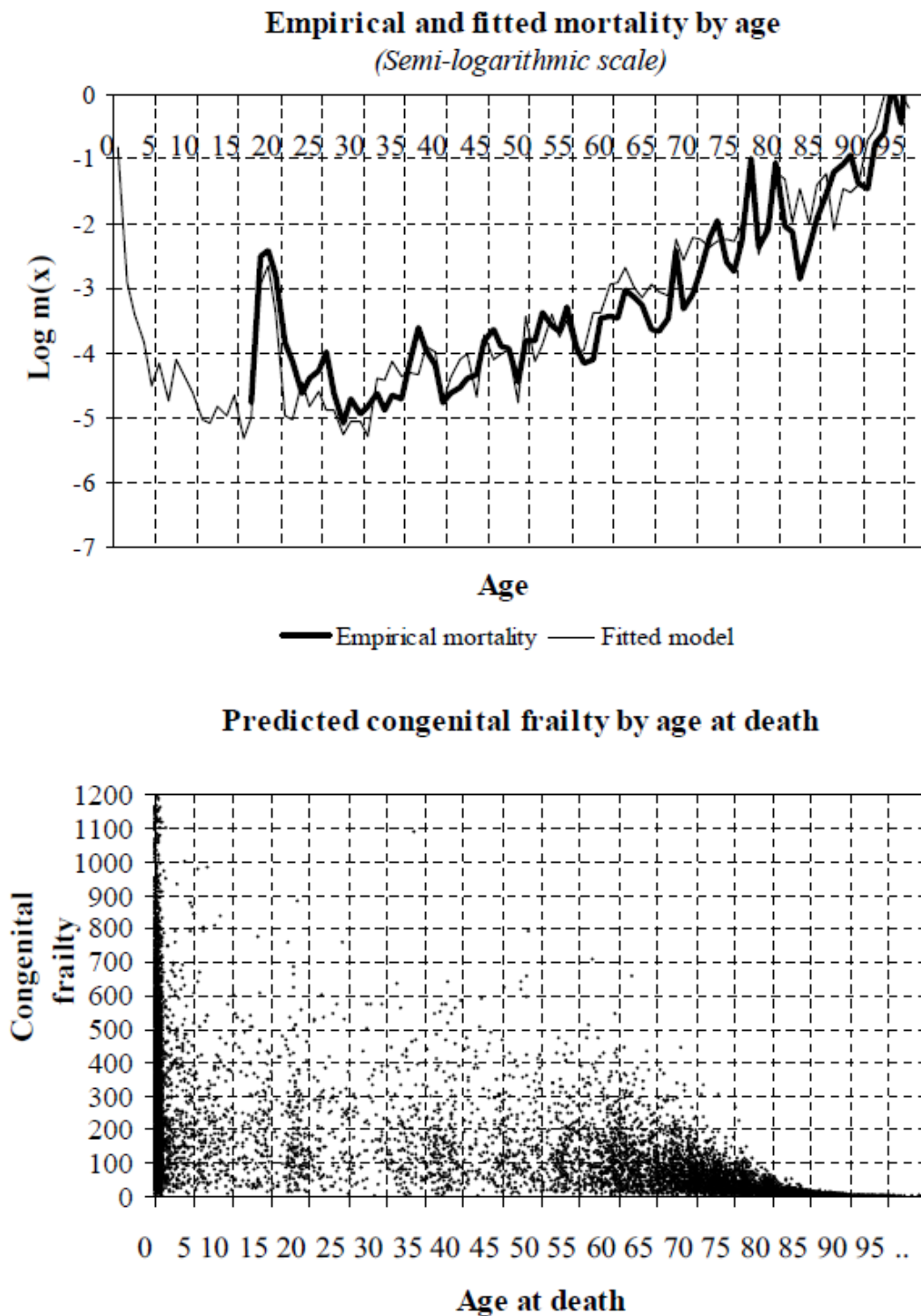
An intermediate estimate \hat{z} (Table 1, column 2) of the mean frailty $E[Z]$ is obtained by Eq. (3). Subject to this restraint I find values of the gamma parameters α and β that together with the shared trend $\varepsilon(t)$ and the joint baseline $m_s(x, z=1)$ minimizes Eq. (2). Keeping the resulting α -value fixed (Table 1, column 3) I fine-tune the fit by calibrating $\text{VAR}[Z]$ (Table 1, column 6) using values of β differing by a suitable step-length. The final β -values and the final z -values appear in Table 1, columns 4 and 5.

To assess the quality of the fit I compare the intermediate (Table 1, column 2) and the final estimates of $E[Z]$ (Table 1, column 5); empirical and model-based life table statistics such as infant mortality (Table 2, columns 2 and 7); life expectancy (Table 2, columns 4 and 5); sums of squared deviation SSD (Table 2, column 3). The life expectancies are truncated by age 100 owing to data limitations. As a result of right-truncation by 2005 only model-based life expectancies can be calculated for the Japanese cohorts and the Swedish and Danish 1944 cohorts. All differences tend to be small. The standard deviation of the model-based life expectancy (Table 2, column 6) rests on 10,000 micro-simulated lifetimes.

Fitting the model to empirical mortality of the Icelandic 1767 cohort involves special efforts due to data deficiencies before 1783. Hence the empirical mortality is left-truncated by age 16. As the empirical birth cohort is much smaller than the simulated birth cohort, empirical risk time is smaller than simulated time-at-risk. Hence the age profile of empirical mortality is more rugged than the age pattern of simulated mortality. As already indicated by Table 2, fitting the model to empirical mortality is actually quite successful (Figure 3).

The predicted infant mortality is very high. This is in keeping with empirical infant mortality in Iceland between 1838 and on 1860 on reliable statistical record. Furthermore, the model predicts extreme selection among infants and young children. This interpretation applies on a general basis up to at least 1870. The model appropriately anticipates extreme selection

Figure 3 Fit of Eq. (1) to empirical mortality of the Icelandic female cohort born in 1767 and congenital frailty by predicted age at death



Source. Based on data recovered by Hansen (1967).

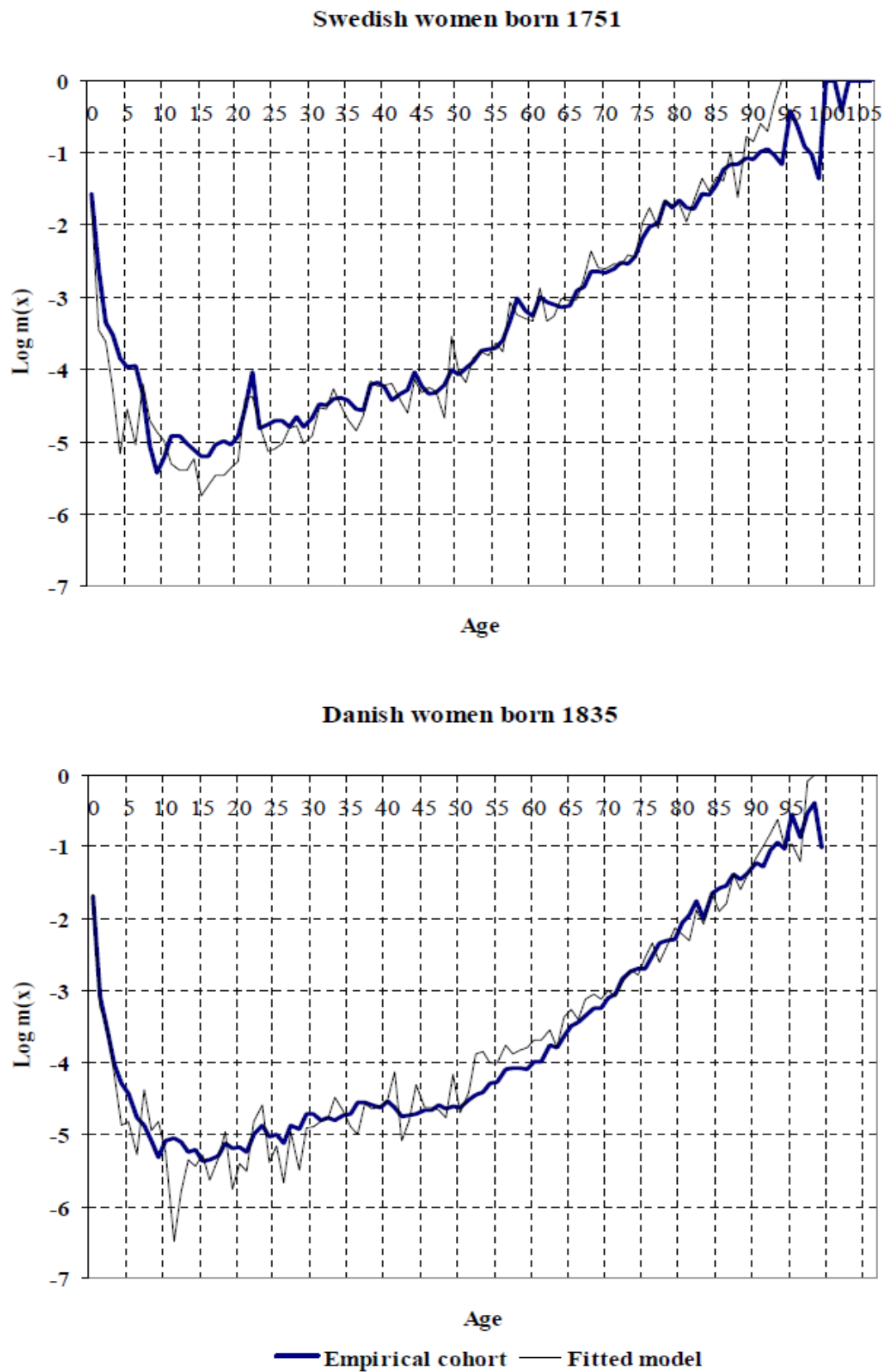
associated with acute mortality in the train of the environmental disaster in 1783. As the birth cohort gets pruned of lives with high congenital frailty, mortality becomes less heterogeneous (Figure 3). By Eq. (1) deceleration of mortality at advanced ages is a natural consequence of selection.

The capacity of Eq. (1) to describe empirical mortality of the other elected birth cohorts, using the shared baseline, is also quite successful (Table 2). Some graphical examples are shown in Figures 4 and 5. The deviation of modeled from empirical mortality in general, is small and unsystematic up to age 90 (Figure 4). The data currently in the Berkeley Mortality Database are too weak for serious model assessment beyond this age.

Figures 5 and 6 illustrate changes in period mortality from 1917 to 1924 and between 1944 and 1952. The secular trend $\varepsilon(t)$ is not fully adequate on describing mortality of teenagers and younger adults. The 1901 cohort was more depleted by the Spanish Flu around 1918 than older birth cohorts, possibly due to reduced selection brought about by decreasing mortality since the 1890s. In addition to selection, an effect of immunity cannot be ruled out. Conversely, the 1944 cohorts suffered more from the upsurge of mortality towards the end of the Second World War and gained more from the succeeding mortality decline between 1945 and 1952 than older cohorts. The causes of this mortality change are complex. The increase of mortality towards the end of World War II may have selected the survivors. Furthermore, rather large contingents of war refugees, many with bad health, were repatriated after 1945. And last but not least, infectious diseases could now be combated more efficiently as penicillin and other medical advances became available to the public after the war. The small deviations of fitted from observed cohort mortality during the Spanish Flu in 1918 and in the train of World War II are discernible on the logarithmic but hardly on the metric scale.

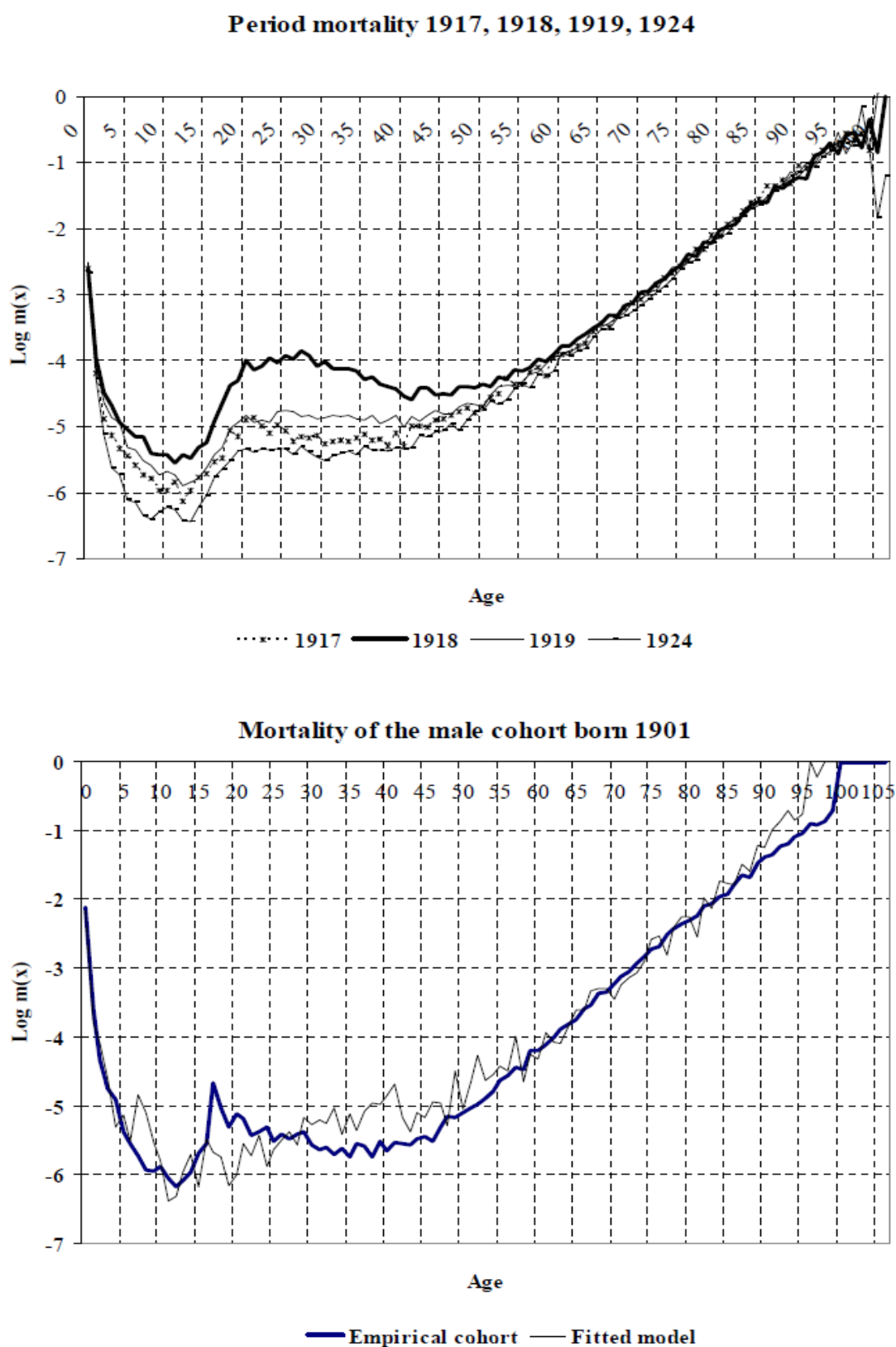
As indicated by Table 2, quantifying $\varepsilon(t)$ by Eq. (4) works well. A sample of trends is shown in Figure 7. Low values of $\varepsilon(t)$ indicate great human control of mortality. The

Figure 4 Empirical and fitted mortality by age. Swedish women born in 1751 and Danish women born in 1835



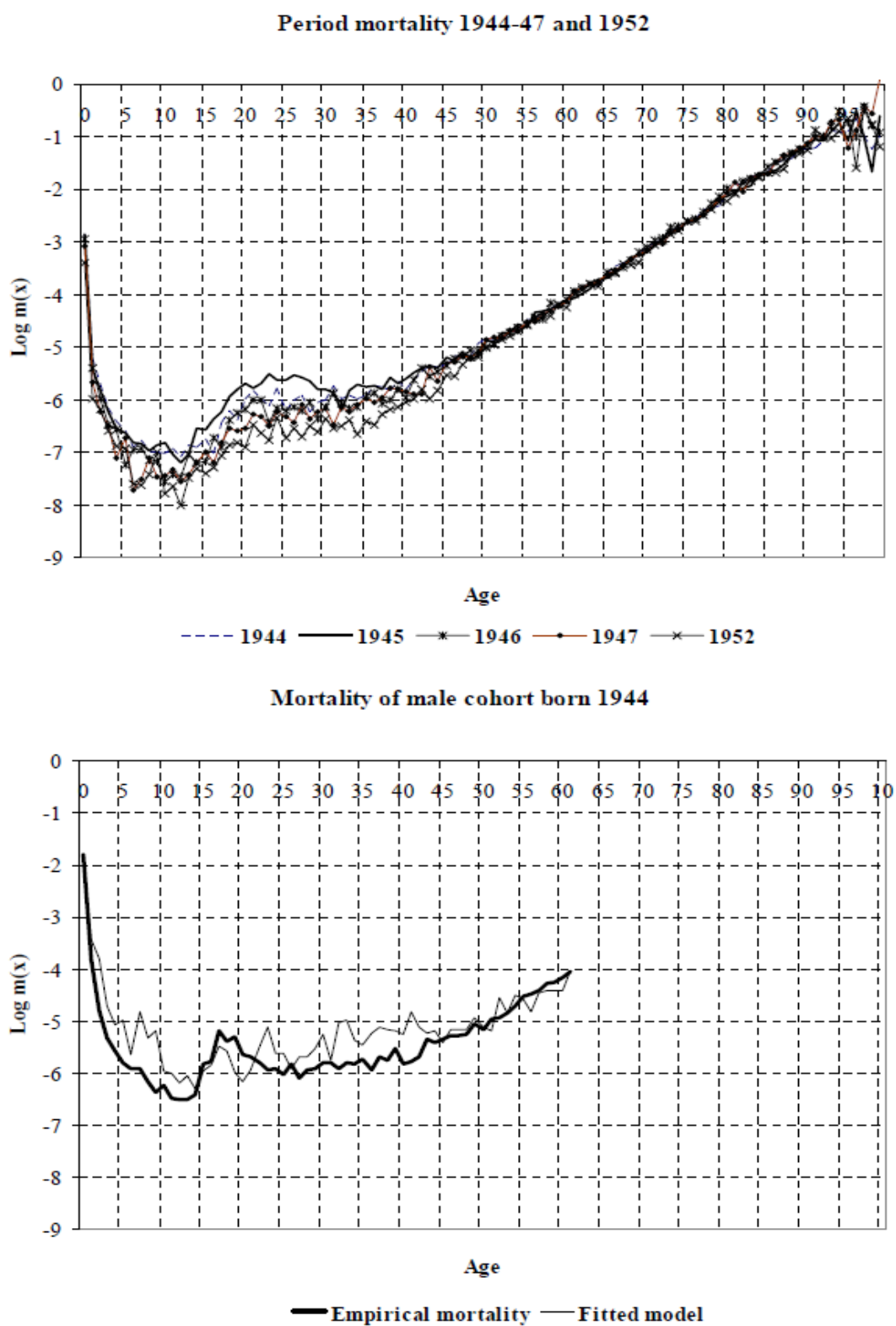
Source. Empirical mortality: Berkeley Mortality Data Base.

Figure 5 Period mortality of Swedish men between 1917 and 1924 and empirical and fitted mortality of men born in Sweden 1901



Source. Berkeley Mortality Data Base

Figure 6 Period mortality of Danish men between 1945 and 1952 and empirical and fitted mortality of men born in Denmark 1944

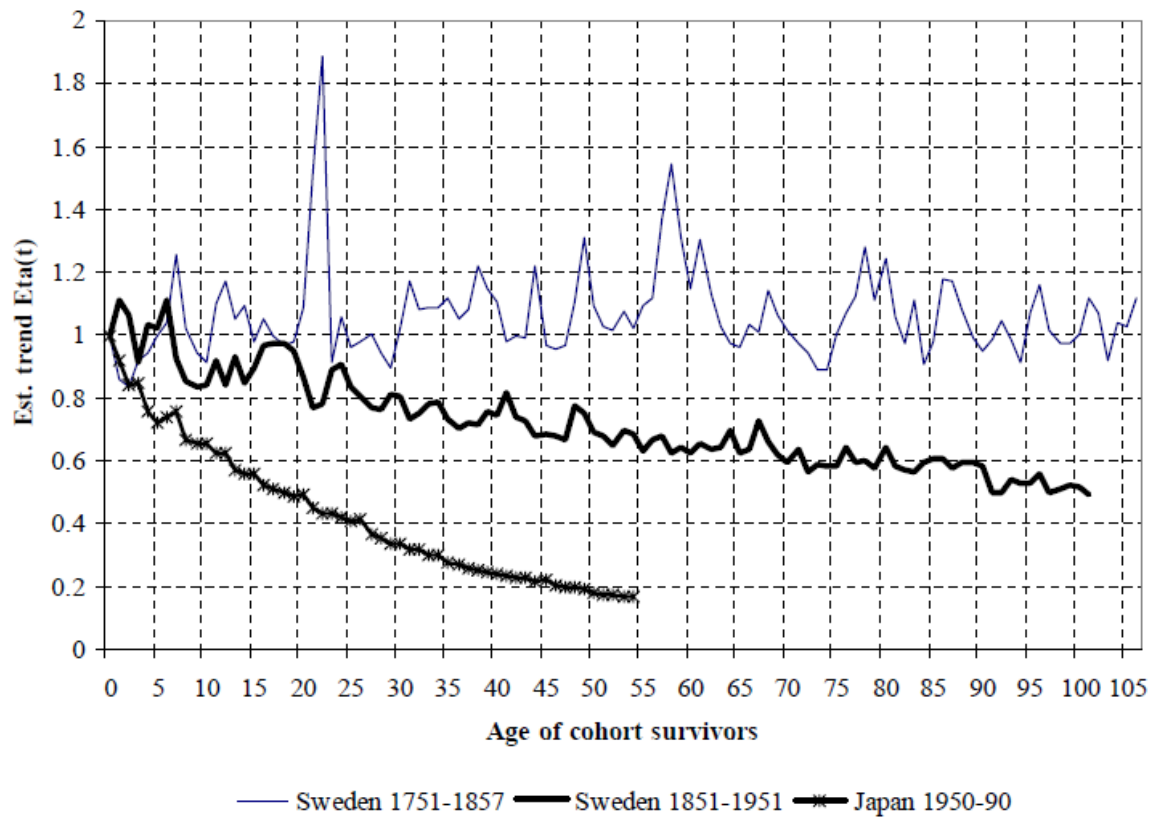


Source. Berkeley Mortality Data Base

trends of the Icelandic 1767 and the Swedish 1751 cohorts exhibit fierce uncontrolled short term variation in external mortality with selection. The trends of the cohorts born in Sweden 1851 and in Denmark 1835 depart from this line of development: The oscillations dampen and the long term trends show distinct decrease, in particular up through the latter half of the nineteenth century. The mainstream of social and technological change behind this development is complex. Comparing the Swedish and Danish trends of the 1901 and the 1944 cohorts, technological change appears to have been somewhat faster after the Second World than earlier in the century. Up to 1970 the trend of the Danish 1944 cohort was slightly lower than that of the Swedish cohort born in 1944. After 1970 the Swedish decline has been faster than the Danish, probably due to greater investment and more goal oriented organization of public health. According to mortality assumptions of recent official population projections, the trends should level off between 2005 and 2050, with Danish mortality declining faster than Swedish mortality. By 2050 the anticipated trend values are roughly the same.

Looking into the future: How well does model-based mortality of the 1944 cohort comply with official anticipations of future mortality development up to 2050 by the Central Bureaus of Sweden and Denmark (cf. Statistics Sweden 2005, 2006; Denmark: Unpublished)? Fitting Eq. (1) to empirical mortality of the 1944 cohorts using the identified joint baseline and assuming $\varepsilon(t)$ constant for values of t beyond 2005, the extended frailty model approaches the official mortality anticipations for the ages below 90 quite well (Figure 8). However, Statistics Denmark expects substantially lower mortality beyond this age than anticipated under the frailty model. Any clear justification of this prospect does not seem to exist. Major mortality decrease in such advanced ages has little influence on expected length of life at birth. Life expectancies at birth in the vicinity of 85 years of men and women combined are realistic in the light of historical mortality change in Iceland, Sweden and Denmark over the past three centuries, given the current level of medical and other technology.

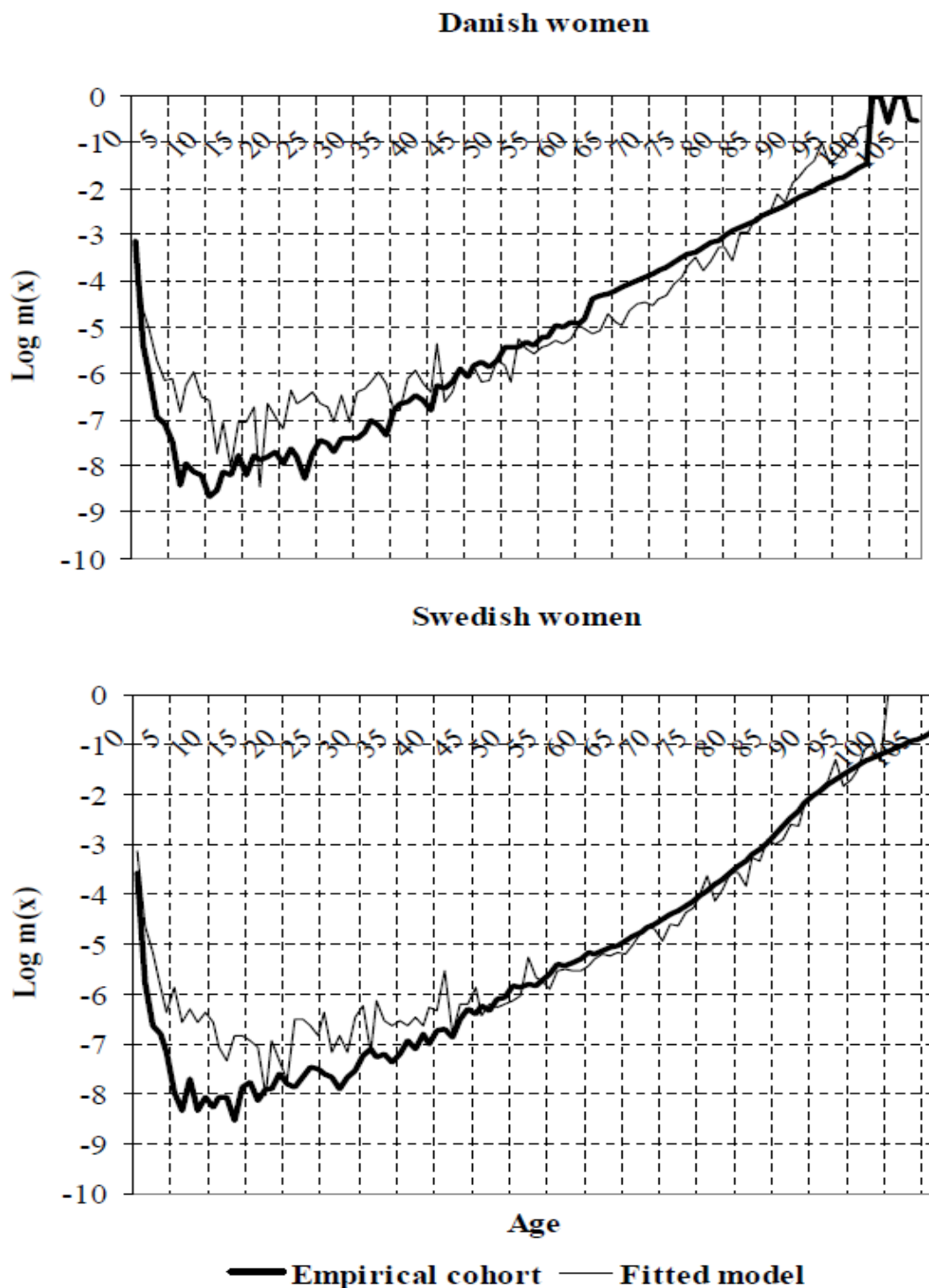
Figure 7 Estimated trends $\varepsilon(t)$ of the Swedish cohorts born 1751, 1851, and the Japanese cohort born in 1950¹



Source. Computations based on the Berkeley Mortality Data Base
 1) The trends are joint for men and women.

Starting from a different level Japan experienced about the same relative decrease of mortality in the second half of the 20th century as did Sweden and Denmark between 1801 and 2005 (Figure 7). A mortality decline of such intensity may have had very significant influence on heterogeneity and selection of current Japanese survivorship (Table 2). Fitting Eq. (1) with the shared non-parametric baseline hazard to elected Japanese cohorts born after the end of World War II emphasizes selection and survivorship in the younger ages. Comparing columns 2 and 7 of Table 2 shows that infant mortality is approached well by Eq. (1).

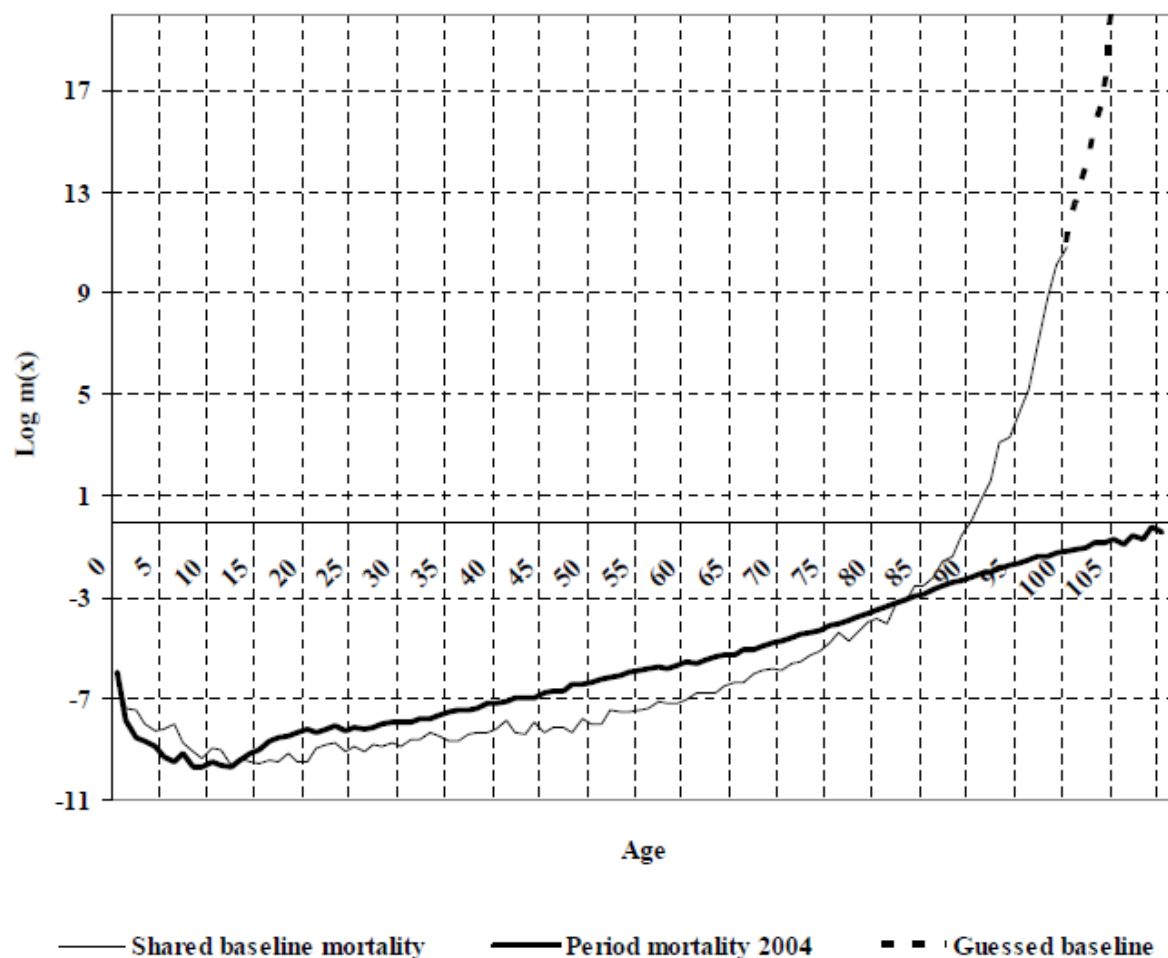
Figure 8 Empirical and predicted mortality of the female cohorts born in Denmark and Sweden 1944¹



Sources. Up to age 61: Computations based on data from Berkeley Mortality Data Base; age 61 and beyond: Computations based on anticipated mortality by Statistics Sweden (2205, 2006) and Statistics Denmark (unpublished).

1) The ruptures in empirical mortality around 2005 are associated with data issues rather than with mortality change.

Figure 9 The baseline hazard¹ and period mortality of Japanese women 2004

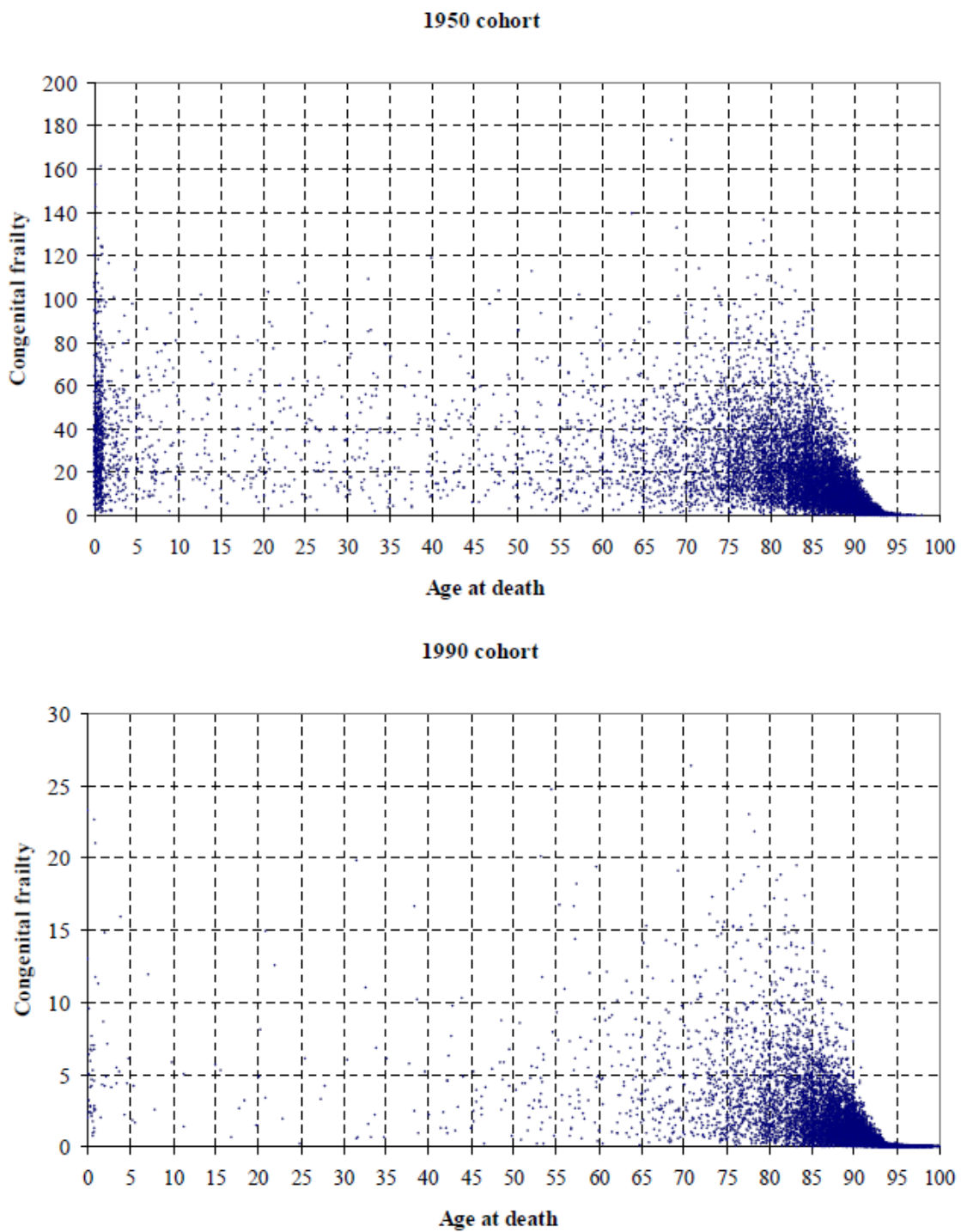


Sources. Computations based on the Berkeley Mortality Data Base.

1) The baseline hazard is common for men and women across all elected cohorts.

The period mortality of Japan is currently at a record low level. Comparing Japanese period mortality 2004 and the specimen of baseline mortality actually applied in this study (Figure 9), baseline mortality $m(x, z = 1)$ is systematically higher than empirical period mortality beyond age 85. This is an indication of substantial selection of Japanese survivors in advanced ages 2004 belonging to the generations born up to 1920. Empirical period mortality 2004 between age 15 and age 85 is systematically higher than baseline mortality, possibly as a

Figure 10 Congenital frailty by predicted age at death among Japanese women born in 1950 and 1990



Source. Computations based on data from Berkeley Mortality Data Base.

consequence of “postponed” or “delayed” mortality associated with steadily reduced selection of the cohorts born between 1920 and 1990 (Table 1). This trend is likely to have continued beyond 1990; which may explain why empirical period mortality 2004 below age 15 actually is lower than baseline mortality. The cross-section of Japanese birth cohorts observed through the tiny window of 2004, consequently, is likely to be highly heterogeneous. Assuming $\varepsilon(t)$ to remain constant and independent of $m(x, z = 1)$ for values of t greater than 2004, the extended frailty model predicts life expectancies of 85.4 years of males and 88 years of females for the Japanese cohorts born in 1990 (Table 2, column 5).

Due to unavoidable confusing of $\varepsilon(T)$, $m(x, z = 1)$, and Z (Eq. (1)) with the data at hand, determination of baseline mortality in very young ages is not perfect. Identifying the baseline hazard beyond age 92 is not viable with the available data, either. Empirical mortality in ages past the early nineties is less reliable due to estimation error associated with smallness of the risk sets, defective observational plans, and many kinds of potential data error.

Under Eq. (1) the notion “congenital frailty” is a prime indicator of health. The extended frailty model predicts dramatic negative changes in the health composition in the mature and elderly ages, as persons with relative high frailties now may live to higher ages than seen earlier in history. This may be illustrated by plotting individual frailty against predicted personal life time of survivors belonging to two cohorts, each comprising 10,000 live births; using the gamma distributed congenital frailties z_i (Table 1) and the trend values $\varepsilon(t)$ of the Japanese cohorts born in 1950 and 1990 on assumption of independence between $\varepsilon(t) = \varepsilon(2005)$ and $m_s(x, z = 1)$ (Figure 10). Rapid population aging due to sustained below-replacement reproduction in Japan since 1960 and increasing costs of keeping frailer individuals in advanced ages alive is likely to become economically stressful. This is already evident in Japan and other first-world countries.

Conclusion

A hypothesis that selection may be a major determinant of heterogeneity in human survivorship over the past two to three centuries is found to be consistent with empirical mortality of the elected cohorts. It is safe to expect that extrinsic mortality $\delta(t)$ with no effect on selection has played a negligible role for the survivor histories of these cohorts. This condition is essential to avoid confusing $\delta(t)$ and external mortality with selection while attempting to identify a joint baseline hazard representing biological aging as the model is fit to data.

Extensive efforts of fitting Eq. (1) to empirical cohort mortality actually have proven successful. The identified biological baseline has been identified mathematically up to a set of multiplicative transformations of a normalized age pattern.

Application of Eq. (1) supports the relevance of distinguishing between *exogenous* and *endogenous* death risk (Bourgeois-Pichat 1952) or *intrinsic* and *extrinsic* mortality (Olshansky et al. 1996, 2003). The base line hazard portrays a degenerative process accounting for endogenous or intrinsic mortality as a function of age; it gains momentum and tends to infinity from around age 90. Even though some people live to age 100, few centenarians to date survive to age 105 and less so to age 110. Hence, eradicating all diseases will not ensure immortality. Biological aging is rather to be seen as a natural process of the organism (Gavrilov and Gavrilova 2001, 2006).

Contemporary technology and bio-medical scientific progress does not appear to have had any substantial influence on biological aging so far. What technological and other progress the future will bring remains to be seen. The current survivor consequences of continued progress in medical technology – however impressive – are likely to remain marginal for some time in countries like Iceland, Sweden, Denmark, and Japan.

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