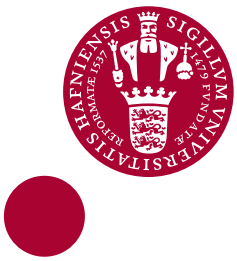


Issues of Selection in Human Survivorship

An Interpretation of Gross Change of Mortality Among Males Born in Sweden 1760 and 1900

Hansen, Hans Oluf

Published in:
Symposium i Anvendt Statistik 2013

Publication date:
2013

Document version
Early version, also known as pre-print

University of Copenhagen, Dept. of Economics
5 Øster Farimagsgade, Building 26, Room 26.0.24,
DK-1353 Copenhagen K, Denmark
Phone +45 35 32 32 61
E-mail HansOluf.Hansen@econ.ku.dk
home page http://www.econ.ku.dk/personal/usihoh/
Ftp-server: ftp://ftp.ibt.ku.dk/usihoh/

10 December 2012

Issues of Biological Selection and Heterogeneity in Human Survivorship -

An Interpretation of Gross Change of Mortality among Males born in Sweden1760 and 1900[1]

By

Hans O. Hansen
Assoc. Prof. (Demography) (Emeritus)

[1] To be presented at Symposium on Applied Statistics, Aarhus University, 28-29 January 2013. Forthcoming in proceedings.

Hans Oluf Hansen:

Issues of Biological Selection and Heterogeneity in Human Survivorship -

An Interpretation of Gross Change of Mortality among Males born in Sweden1760 and 1900

Abstract

The notion of selection is essential on understanding diversity and causation in social change. This study emphasizes selection and heterogeneity in human survivorship as a function of latent individual congenital frailty, a shared baseline hazard portraying biological attrition with age, and environmental change with impact for selection. Using the male Swedish cohorts born in 1760 and 1900 as examples individual life times are obtained by stochastic micro simulation in the simple life model, conditional on latent random frailty on birth. The arbitrary age limit is the ninety fourth birthday.

By disregarding latent heterogeneity popular parametric modeling such as the Lee-Carter model (1992) or classical log-linear modeling (Bishop et. al. 1975; Fienberg 1977; Agresti 2002) nearly always come up short on describing medium or long term change of empirical mortality. Adequate explanation of latent biological selection and heterogeneity in historical mortality change inevitably augments the number of parameters. The simulation model is fully parameterized. This is a viable and consistent prerequisite rather than a problem of a stochastic simulation study.

Lifelong biological frailty is a natural indicator of health. The study shows that the mean and standard deviation of diversity in terms congenital frailty among mature and elderly survivors is perceptibly greater in the 1900 cohort compared to the 1760 cohort when controlling for genetically conditioned frailty and biological aging. The difference lies with the period factors. The decline of Swedish infant and child mortality between 1760 and 1900 has led to massive further postponement of death, in particular of individuals with high congenital frailties, and hence to added heterogeneity of health in the mature and elderly ages. These results gives food for thought on analysis and projection of survivorship in an era of rapid population aging, persistent economic recession, and exploding health budgets.

Keywords: *Selection, heterogeneity, mortality, stochastic micro simulation, longevity*

What is selection?

On conception man is endowed with a personal biological vulnerability determined by genetic characteristics; some of which inherited and others possibly brought about by mutation. Furthermore, human survival is governed by biological attrition and period factors. Interaction with the physical, biological and social environment causes selection in survivorship. The importance of environmental influence upon human survivorship is indicated by mortality change. Human behavior is governed by numerous constraints, some of which self-inflicted and others imposed by the changing social or physical environment. Volitional or forced selection on joining or staying out of a group causes heterogeneity of the group. Rational behavior entails maximizing the recognized options open to the individual agent at any point in his/her life course. Hence, selection is essential on understanding diversity and causation in social change.

Objective of the study

This study offers an illustration and interpretation of selection in human survivorship as a function of latent congenital frailty in the framework of the simple life model. For an example of selection instigated by congenital frailty in generalized life models cf. Hansen (2011).

Data

Annual central death rates of Swedish males 1751 to 2010 by sex and one-year age groups. Source: Human Mortality Database (HMD). Cause-of-death is not reported in HMD.

Model and estimation

Model. Referring to Hansen (2008, 2011) the frailty model may briefly be summarized as follows.

$$m_{\upsilon}(x,t) = z_{\upsilon} m_s(x, z=1)\varphi(x,t) + \theta(x,t);\ \varphi(0,t_0) = 1 \qquad (1)$$

Mortality $m_{\upsilon}(x,t)$ of individual υ aged x at time t is seen as a function of latent congenital frailty z_{υ}; latent biological attrition indicated by a shared baseline hazard $m_s(x, z=1)$; and period factors $\varphi(x,t)$ and $\theta(x,t)$ respectively with/without impact for selection. In this study we assume $\theta(x,t) \equiv 0$. Consider radix $\ell(0,t_0)$ of a given birth

cohort born at time t_0; let z_υ denote latent personal frailty on live birth; and assume z_υ to be an outcome of a gamma distributed stochastic variable Z as follows.

$$Z = z_\upsilon \sim Gamma(\alpha, \beta, \gamma); \alpha, \beta > 0, \gamma = 0 \tag{2}$$

$$E[Z] = \alpha\beta \tag{3}$$

$$\begin{aligned} Var[Z] &= \alpha\beta^2 \\ &= \beta E[Z] \end{aligned} \tag{4}$$

Gamma parameters α, β, γ symbolize shape, scale, and location of the probability distribution. Throughout this study we set $\gamma = 0$. The latent gamma parameters have no independent biological interpretation. They serve to generate diversion in individual congenital frailty. Congenital frailties greater than mean indicate bad health while frailties lower than or equal to mean, designate mean health or health better than mean. Individual congenital frailty is taken to remain the same over the entire life course.

Setting up parameters. From (3) we learn that a given value of $E[Z]$ determines a hyperbola in the first quadrant of an orthogonal coordinate system. See figure 1 for an example. This means that there may be many combinations of shape and scale parameters obeying the restraint. For $x = 0$ sharp $\varphi(x = 0, t_0) = 1$. We may then estimate $E[Z]$ from (1) by,

$$E[Z] = \frac{m(0, t_0)}{m_s(0)} \tag{5}$$

with $m(0, t_0)$ representing empirical cohort mortality.

The latent baseline $m_s(x, z = 1)$ and the gamma parameters used in the application to follow have been uncovered by extensive experimenting based on elected samples reflecting extreme variation in empirical cohort mortality across the demographic transition in Iceland, Denmark, Sweden, and Japan. The results suggest that the baseline $m_s(x, z = 1)$ and the gamma parameters could be fixed in time (Hansen 2008, 2011).

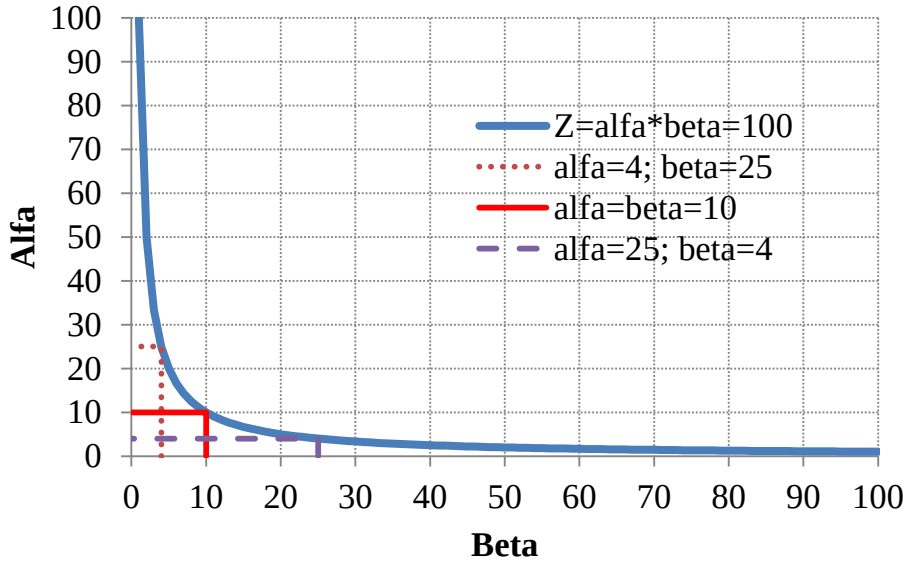
There has been a profound long term change in the cause-of- death structure of infants and young children in contemporary first-world countries. This change is best described by the classification by Bourgeois-Pichat (1946, 1951) of infant mortality into additive endogenous and exogenous mortality. We believe that the case is stronger for seeing the change as a period effect rather than as a shift of biological attrition with age. The interpretation of latent heterogeneity to follow rests on this proviso.

A model application targeting latent selection and heterogeneity in survivorship

How has the modern long term decline of mortality affected health and mortality in the course of the demographic transition? And what can we expect? In the following we address the issues under model (1) using data from HMD.

Figure 1

An example of different combinations of gamma parameters (α, β) generating dissimilar Levels of selection in survivorship in presence of fixed expectation.



The presentation of results proceeds as follows. We set out by comparing the recovered period factors of the Swedish male cohorts born around 1760 and 1900 i.e. $t_0 = 1760$. To present and comment on heterogeneity associated with latent congenital frailty among survivors we group the live births by elected fractile intervals of the gamma distribution describing the random allocation of the individual frailties. Furthermore, we compare mean and standard deviation of congenital frailty among survivors as functions of age across the two birth cohorts. The article closes with a summary and some perspectives on results.

Uncovered latent period factors

Linear mortality change by age may be described by period factor $\varphi(x,t \mid \forall t) = \alpha x + 1$ with $\alpha = 0$ indicating stationarity and $\alpha \neq 0$ representing proportional change, either steadily growing ($\alpha > 0$)or declining ($\alpha < 0$). In other terms, deviation from stationary or proportional change translates into period factors hovering around straight lines. Non-linear period factors indicate greater complexity of empirical mortality change.

To disentangle biological selection and heterogeneity from "pure" period influence we fit model (1) to empirical cohort mortality by iterative adjustment of period factor $\varphi(x,t)$ under the restraints of fixed frailty distribution and permanent biological attrition with age. When "cleansed" of disturbing biological influence the adjusted period factor becomes an informative indicator of environmental influence upon survivorship with

impact for biological selection and heterogeneity; such as epidemics in traditional societies or progress in medical and hygienic technology in emerging modern or contemporary first-world societies with rising or high mass consumption of goods and services of vital necessity.

Figure 2 shows the estimated period factors of the cohorts born in about 1760 and 1900. The fully drawn graph of the 1760 cohort and the dotted graph of the 1900 cohort portrays the period factors after adjustment to empirical cohort mortality. The peak at age 12-13 of the 1760 cohort refers to the hunger crisis of 1772-73 while the top at age 18-20 of the 1900 cohort is associated with the Spanish Flue around 1918. Excepting the turmoil associated with the Spanish Flue the trend of the 1760 cohort is much higher than that of the 1900 cohort. Furthermore, the 1760 graph deviates from the 1900 graph by fluctuating around 1 while reflecting several minor crises. Excepting the major crises of 1772-73 and 1918-21, the 1900 cohort - in striking contrast to the 1760 generation - exhibits nearly steady decline of mortality across age and time; indicating that mortality had come under much greater human control in the course of the twentieth century. Via calibration by eq. (5) infant mortality in about 1900 is almost half its level around 1760.

High infant mortality and sudden upsurge of mortality associated with epidemic infection may have significant impact on selection and biological diversity of survivors. This hypothesis is substantiated below.

Allowing for randomness associated with micro simulation of remaining life time (truncated by age 94) combined with iterative assessment of environmental (period) influence $\varphi(x,t)$, modeled mortality (dashed curves) approaches empirical cohort mortality (fully drawn curves) closely which should be expected as model (1) is fully parameterized (figure 3).

Survivorship impacts of congenital mean frailty.

What differential survivorship impacts of subgroupings of congenital frailty can be detected under model (1) in presence of joint prenatal biological and gestational factors combined with alternative series of postnatal period factors? To gain some insight into the trend of development of differential survivorship impacts under the *ceteris paribus* restraint we focus on the cohorts born in 1760 (34,693 live births) and 1900 (70,840 live births).

The micro simulated data sets. The congenital frailties of the modeled mortality of the 1760 and 1900 cohorts are drawn from the gamma probability distribution with form parameter (alpha) equal to 1.43, scale parameter (beta) equal to 84.91, and location parameter (gamma) equal to nil. The risk set of predicted mortality is therefore

Figure 2.
Estimated series of cohort-based period factors of the male generations born in 1760 and 1900

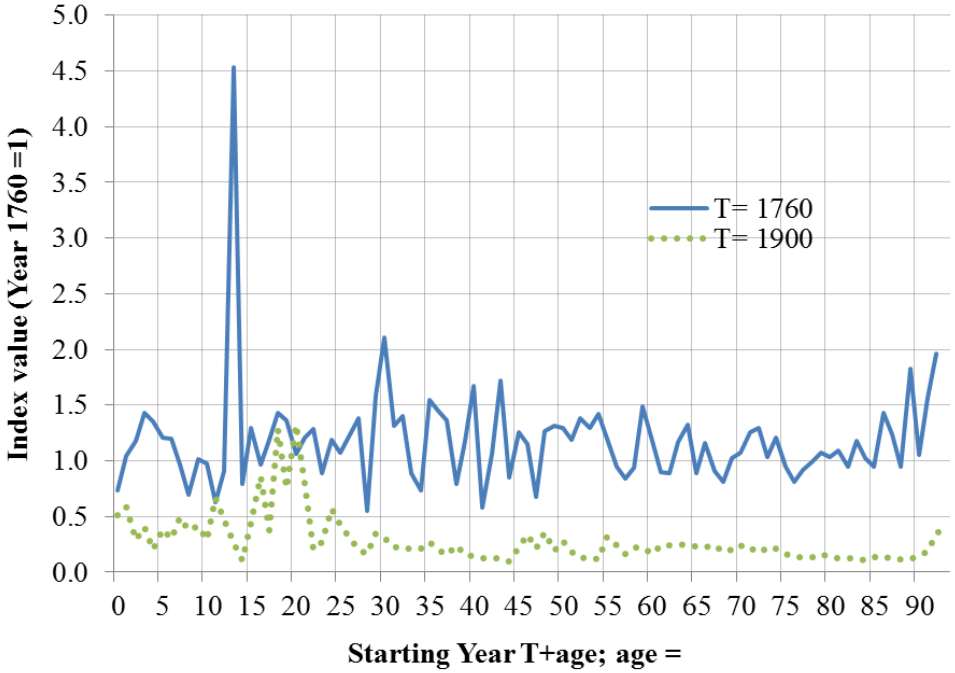
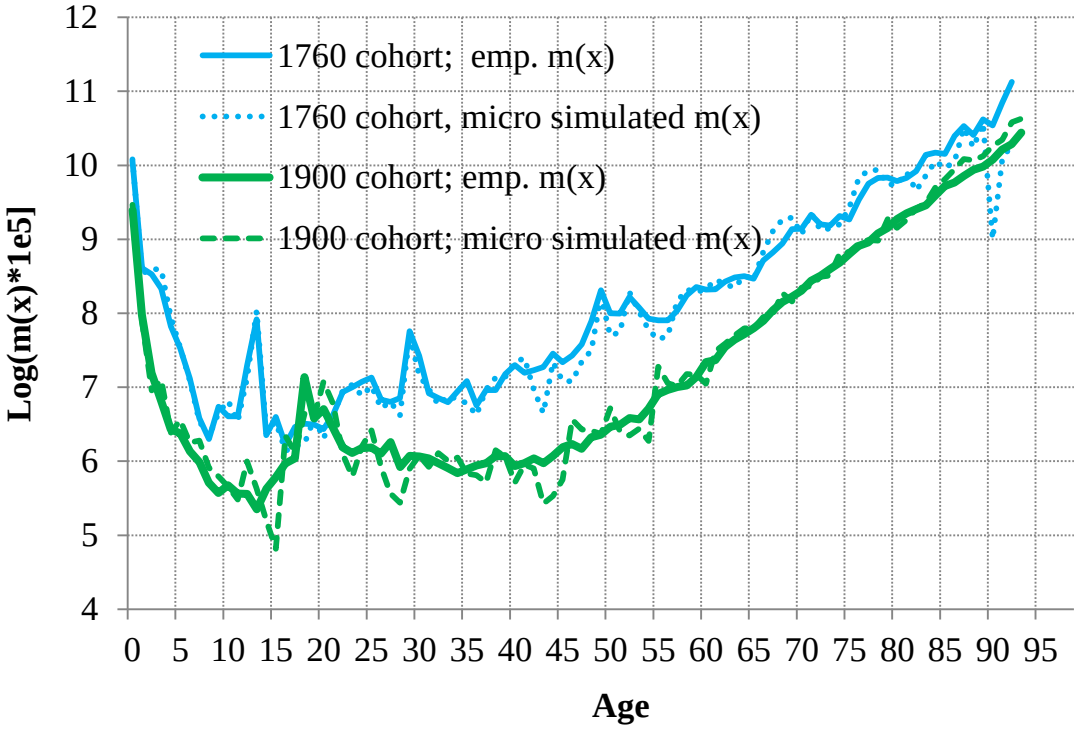


Figure 3.
Empirical and modeled mortality (logarithmic scale) by age. Swedish males born 1760 and 1900



heterogeneous. Stochastic micro simulation of individual life times under exposure to alternative period factors results in sequential data sets with variables (1) personal Id; (2) birth date; (3) age at death (truncated on the ninety-fourth birthday); (4) congenital frailty. At present birth date is scored by birth year but seasonal variation of the live births could be imposed along with date of death (derived variable) or classification of congenital frailty (derived variable) if so desired.

Differential survivorship impacts. A natural working hypothesis would assume human life time to be strongly dependent upon level of congenital frailty. To document differential selection and heterogeneity at various levels of aggregation from the micro simulated data sets we proceed as follows. We choose to operationalize level of congenital frailty by differences between percentiles z_F in the incomplete gamma probability distribution from where the frailties were drawn. To do so we compute

$$z_F = gamma^{-1}(Z = z \le F \mid \alpha = 1.43, \beta = 84.91, \gamma = 0) \qquad (6)$$

with F =.05, 10, 25, 50, 75, 90, 95 denoting percentiles alias fractiles and z_F indicating frailties for which $P\left(Z \le z_F \mid \alpha = 1.43, \beta = 84.91, \gamma = 0\right) = F$. For each frailty group we count the number of live births. Furthermore, for each sub-group we compute expectation value of *age* along with standard deviation and maximum value of this variable.
The descriptive statistics are listed in table 1.

Substituting period factors 1760 to 1854 with period factors 1900 to 1994, all other components fixed, increases the estimated expected life time at birth in the age segment 0-93 from 32.4 to 54.9 years. Both mean values are strongly heterogeneous with latent life expectancies varying widely across the subgroups of congenital frailty. The mortality decrease is greatest in the younger mature ages as also indicated by figure 3. Furthermore, as mortality falls we observe a systematic shift in life expectancy with increase from low to high congenital frailties (figure 4). Under model (1) environmental impact $\varphi(x,t)$ is multiplicative; meaning that personal death risk is correlated with personal congenital frailty: the higher the frailty of a live birth, the greater the absolute change in personal death risk. When mortality declines and life expectancy goes up progress will be better among persons with frailties above mean than among persons with congenital frailties below mean. Conversely, live births with high frailties will be among the first to perish if environmentally induced mortality goes up. This is very clearly illustrated by table 1 and figure 4.

Consolidated survivorship impacts of recovered congenital mean frailty. As the birth cohort gets pruned of weak lives we should expect the mean and the standard distribution of congenital frailty among survivors to decrease. The pace of the decline may be quite different, however, depending on the mean frailty on live birth.

Table 1.
Descriptive statistics for variable *Age* given joint biology and shared prenatal survivorship
Swedish male cohorts born in 1760 and 1900

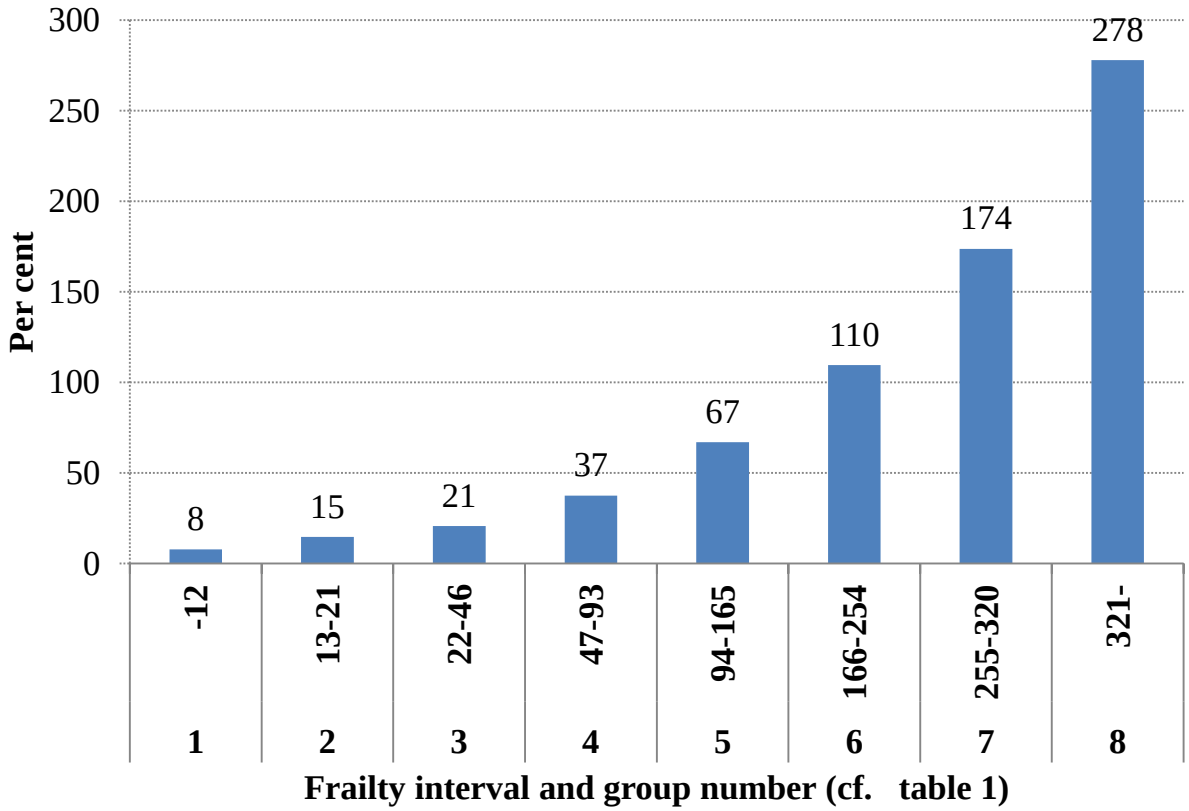
Group k	$P(X < z_k \mid \alpha, \beta) = F_k$		Statistics for Variable Age^+			
	F_k	$[z_k, z_{k+1}[$	N	MEAN	STD	MAX
1760 cohort, period factors 1760-1854						
1	0	-13.9	174	83.7	11.1	92.80
2	0.05	14.0-22.2	419	76.1	16.7	90.59
3	0.1	22.3-47.4	2,152	67.6	21.9	87.89
4	0.25	47.5-94.4	5,894	52.7	27.3	86.06
5	0.5	94.5-166.5	9,224	36.5	28.0	81.08
6	0.75	166.6-255.9	8,286	23.4	25.1	79.49
7	0.9	256.0-321.3	3,592	14.6	20.4	74.86
8	0.95	321.4-	4,946	7.6	14.5	71.69
Persons dying before age 94			34,687	32.4	30.3	92.80
Persons dying after age 94			6			
Total			**34,693**			
1900 cohort, period factors 1900-1994						
1	0	-13.9	260	90.1	6.9	93.98
2	0.05	13.0-22.2	853	87.2	12.7	93.99
3	0.1	22.3-47.4	4,193	81.6	16.3	93.91
4	0.25	47.5-94.4	12,128	72.4	22.6	93.06
5	0.5	94.5-166.5	19,087	60.8	27.7	90.87
6	0.75	166.6-255.9	16,599	49.0	30.1	88.60
7	0.9	256.0-321.3	7,361	40.0	30.1	85.51
8	0.95	321.4-	10,186	28.6	28.7	82.54
Persons dying before age 94			70,667	54.9	31.5	93.99
Persons dying after age 94			173			
Total			**70,840**			

+) N = number of live births. MAX refer to individual life time= age at death

To substantiate our anticipated postnatal selection and heterogeneity effects in human survivorship we graph individual congenital frailty by individual age at death of the 1760 and the 1900 cohorts (figures 5-6). The dashed horizontal grid lines indicate percentiles of congenital frailty distribution cf. table 1.

The number of survivors dying beyond a given age x is equal to the number of live births surviving at least to age x i.e. $\ell(x)$. Transition to death is a poor indicator of

Figure 4
Change of life expectancy by group of congenital frailty.
The male born in Sweden around 1900 in per cent of the 1760 cohort



health. The micro simulated datasets provide ample opportunities of studying hypotheses on health gradually and eventually leading to death.

On assumption of fixed frailty from conception to death, the greatest postnatal selection takes place among infants and young children and in the mature or elderly ages, from around age 45 and beyond (figures 5-6). Heavy selection of minors is a distinct feature of the 1760 cohort while the 1900 cohort emphasizes selection in the mature or elderly ages. Absence of effective human intervention evidently precipitates selection. Conversely, improved care and better living conditions tend to postpone selection, thereby increasing biological heterogeneity among survivors. External shocks such as infectious diseases like the one related to crop failure and famine around 1772-73 (figure 5) or epidemic infection like the Spanish Flue around 1918-21 (figure 6) may cause dramatic increased selection instantly with subsequent reduction of biological diversity. Individual frailties delimitated by the dashed horizontal grid lines constitute the expected length of life at birth by the frailty groups defined in table 1.

Figure 5.
Congenital frailty z by age at death. SE males born 1760
(The dashed horizontal grid lines indicate percentiles of congenital frailty distribution cf. table 1)

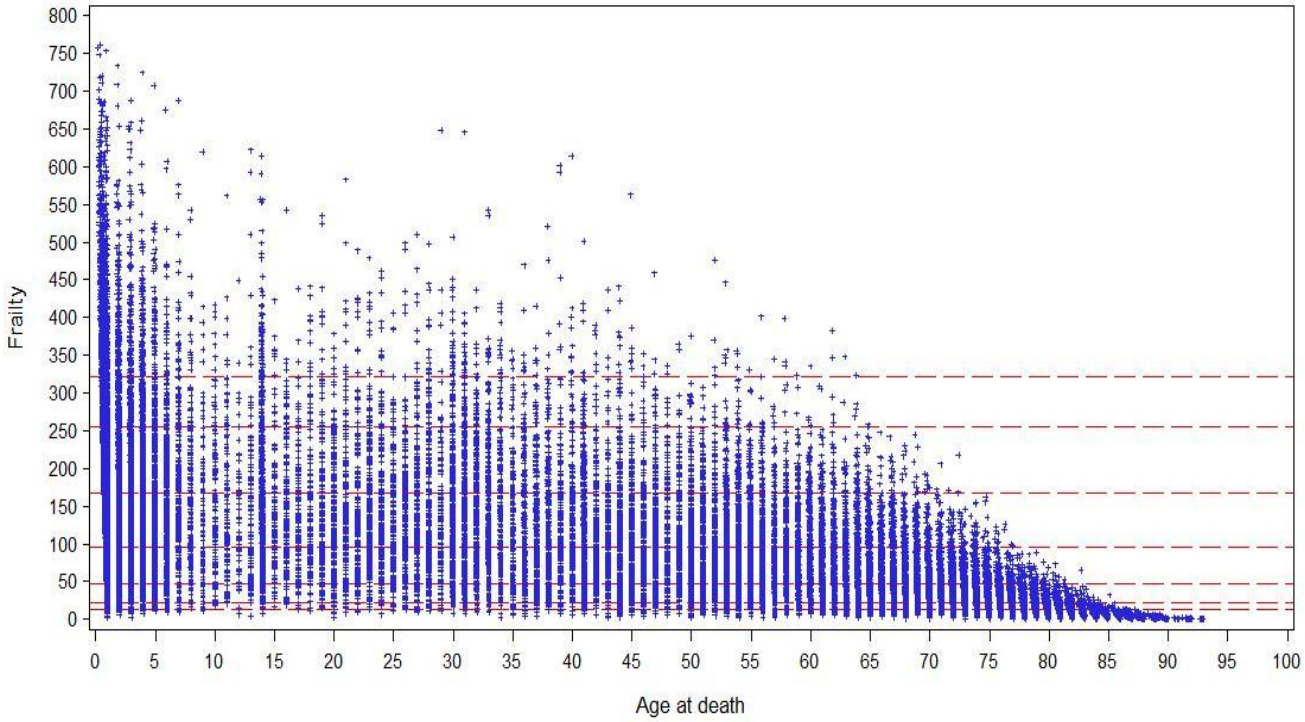


Figure 6.
Congenital frailty z by age at death. SE males born 1900

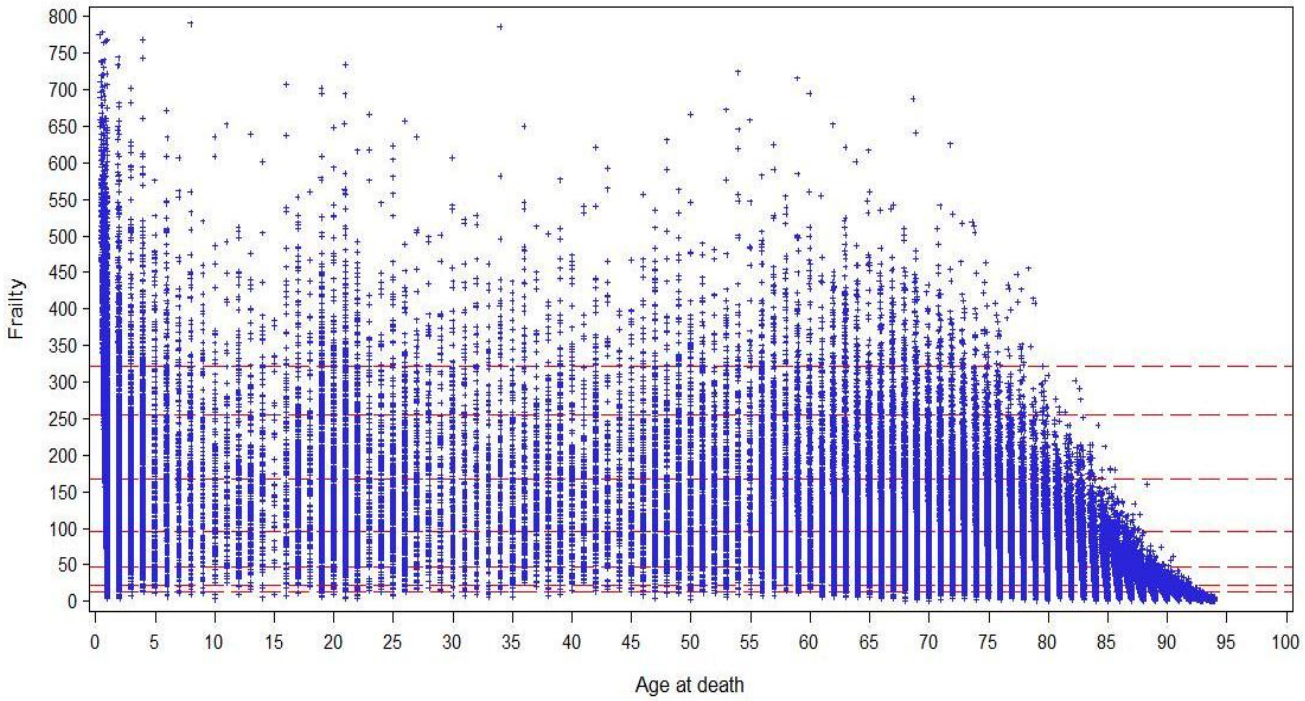


Figure 7

Estimated mean and standard deviation of latent micro-simulated congenital frailty among cohort survivors at age *x*. The Swedish male cohorts born in 1760 and 1900 (Cohort size 1760: 34,693 live births; 1900: 70840 live births)

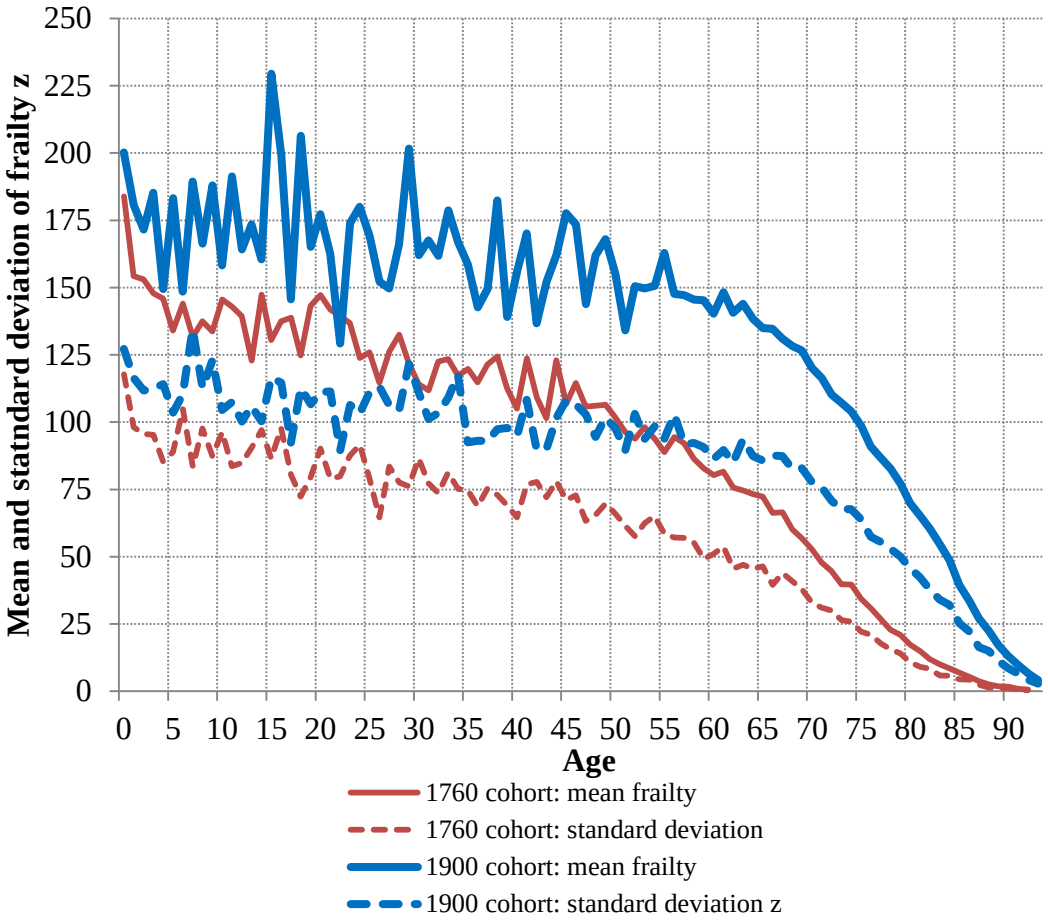


Figure 7 summarizes major features of figures 5-6; by displaying the estimated mean and standard deviation of congenital frailty among cohort members surviving at least to a given integer-valued exact age from birth to the ninety fourth birthday. Decline of mean frailty with age is faster and standard deviation smaller in the 1760 generation compared to the 1900 cohort; indicating that heterogeneity decreases much faster with age in the 1760 cohort. All graphs naturally converge to nil as the birth cohorts become extinct.

Closing remarks

This project has shown that a hypothesis of biological selection and heterogeneity subject to sustained environmental influence, is consistent with empirical mortality change. The study suggests that medical intervention *per se* may actually have been more successful

among mature and elderly people than indicated by biologically heterogeneous empirical life data in the course of the modern long term demographic transition.

Human life is finite - all good things come to an end. Because of biological and social diversity the expectation of a fixed (deterministic) upper age limit is hardly realistic. Infant and child mortality is a principal determinant of expected length of life at birth. In advanced contemporary first-world countries such as Sweden or Japan infant and child mortality is now so low that there is little scope for much further improvement. The potentials of added mortality reduction among people around age 50 or beyond in such countries are more difficult to assess, first and foremost because of insufficient information on health. Postponing death by medical intervention comes at a cost and may eventually lead to added expenditure when the mixture of latent frailty and biological attrition intensifies with age. Improvement of expected length of life in first-world countries will be marginal and costly. If the required economic and humanitarian resources can be mobilized there might still be some space for improvement, however. Extending the upper random age limit on a mass basis necessitates successful meddling with the human genome controlling biological aging, in model (1) represented by the baseline hazard. Whether this will be technically possible or even desirable remains to be seen as for now. Anticipation of sustained life expectancies far beyond eighty five years is commonplace in demographic wisdom. Such vistas are poorly supported by trustworthy cohort-based mortality experience worldwide up to now (2012).

Biological diversity has an impact on acquired social and economic heterogeneity. The approach adopted in this study has drawn on stochastic micro simulation of remaining time to death in the framework of the simple life model. It may readily be generalized to more complex stochastic life models describing interaction of biological and social change.

Finally, fitting the frailty and selection model to one-year cohorts over longer time spans with subsequent conversion to temporal time may help understand and explain transitory mortality change related to cross-sections of birth cohorts in the course of the modern long term demographic transition. Such experience may provide a useful basis for more informed projections of mortality and survivorship than seen so far.

References

Agresti, A. (2002). Categorical Data Analysis (John Wiley & Sons, Inc., Hoboken, New Jersey).

Bishop, Y., S.E. Fienberg, P.W. Holland (1975). Discrete multivariate analysis: Theory and practice (M.I.T. Press)

Bourgeois-Pichat, J. 1946. De la mesure de la mortalité infantile. Population (French Edition). 1, 1. 53-68

Bourgeois-Pichat, J. 1951. La mesure de la mortalité infantile. Population (French Edition). 6, 2. 233-248

Bourgeois-Pichat, J. 1952. La mesure de la mortalité infantile: II, Les causes de décès. Population (French edition). 6, 3. 459-480

Fienberg, S. E. (1977). The Analysis of Cross-Classified Categorical Data (M.I.T. Press)

Girosi, F.; G. King (2007). Understanding the Lee-Carter Mortality Forecasting Method (Internet. Link: http://gking.harvard.edu/gking/files/lc.pdf.

Hansen, Hans O. (2008). Issues of Selection in Human Survivorship: A Theory of Mortality Change from the Mid-Eighteenth to the Early Twenty First Century. Discussion Paper No. 2008-18. Department of Economics, University of Copenhagen. (Link: ftp://ftp.ibt.ku.dk/usihoh/Docs).

Hansen, Hans O. (2011). Selection as a factor in human survivorship over the past three centuries. Prepared for 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. IUSSP Working Paper.

Hansen, Hans O. (2012). Measuring Period Factors in Human Survivorship. Presentation prepared for the Tallinn Workshop, September 2012: New measures of mortality – what do they mean? Estonian Institute for Population Studies, Tallinn University, Estonia, Tallinn, 5 – 7 September 2012 .

Human Mortality Database. University of California, Berkeley (USA), and Max Planck Institute for Demographic Research (Germany). Available at www.mortality.org or www.humanmortality.de

Lee, Ronald D. and Lawrence R. Carter. 1992. "Modeling and Forecasting U.S. Mortality." Journal of the American Statistical Association 87(419, September).