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A duality in aging: the equivalence of mortality models based on radically different concepts

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Abstract

Several alternative mortality models fit Swedish old-age mortality data equally well. The models build on two different concepts of the heterogeneity of individuals in a population. The first concept concerns fixed, genetic differences among individuals in their risk of death. The second concept involves acquired susceptibility to death due to physiological changes and environmental influences. We show that alternative mortality models based on either of these two concepts or some mix of them lead to the same parametric form of observed age-specific death rates. We discuss this duality property of mortality processes and show that even when a mortality model fits the data, the concepts used to construct the model may not be correct.

Key words: Models of mortality; Gompertz law; Sweden; Heterogeneity; Frailty

1. Introduction

Human death rates increase exponentially with age from about age 30–35 to about age 80–85. At more advanced ages, however, the rate of increase slows and the trajectory of mortality falls off from a Gompertz curve [1,2]. Two large studies of fruit fly mortality found a similar deceleration of death rates [3,4]. In this article, we compare several alternative mortality models that can account for the observed pattern. The models build on two different concepts of the heterogeneity of individuals in a population. The first concept concerns fixed, genetic differences among individuals in their risk of death. The second concept involves acquired sus-

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ceptibility to death due to physiological changes and environmental influences. Surprisingly, it turns out that alternative mortality models based on either of these two concepts or on some mix of them can lead to the same parametric form of observed age-specific death rates. This duality implies that it is impossible to determine the validity or relative importance of the two concepts on the basis of mortality data alone.

2. Genetic differences and observed mortality

In 1960, Strehler and Mildvan [5] showed that heterogeneity among individuals can produce an observed mortality pattern in a population that deviates from the underlying age-trajectory of mortality for individuals. One possibility is that individuals in a cohort have different chances of survival because of fixed, innate differences in frailty. Following Beard [6] and Vaupel, Manton and Stallard [7], let the force of mortality (or hazard of death) of individuals of frailty Z at age x be given by:

$$\mu(x,Z) = Zae^{bx} + c \tag{1}$$

where a, b, and c are parameters to be estimated. If frailty Z is a gamma distributed random variable with mean 1 and variance σ^2, then average frailty among survivors at age x is given by:

$$\bar{Z}(x) = \frac{1}{1 + \sigma^2 \frac{a}{b}(e^{bx} - 1)} \tag{2}$$

and observed mortality $\bar{\mu}(x)$ in the population is given by:

$$\bar{\mu}(x) = \frac{ae^{bx}}{1 + \sigma^2 \frac{a}{b}(e^{bx} - 1)} + c \tag{3}$$

The model in Eqs. 1 and 3 might be called a gamma-Makeham model. It corresponds to the case of fixed continuously distributed frailty. The observed mortality (3) corresponds to a logistic mortality curve.

It has been shown [8,9] that this model fits mortality data significantly better than the traditionally used Gompertz-Makeham mortality model, where

$$\mu(x) = ae^{bx} + c \tag{4}$$

We confirmed this finding by fitting the gamma-Makeham and the Gompertz-Makeham models to Swedish data using the maximum likelihood method [10]. The likelihood ratio test indicates that for every male and female cohort born from 1850 to 1879, the gamma-Makeham model gives the better fit to the data at the significance level of 0.01.

Fig. 1a,b plots the empirical, Gompertz-Makeham and gamma-Makeham death rates starting from age 50 for illustrative 1861 Swedish female and male cohorts. Graphs for other cohorts look similar.

The gamma-Makeham model is based on the assumption that differences in sus-

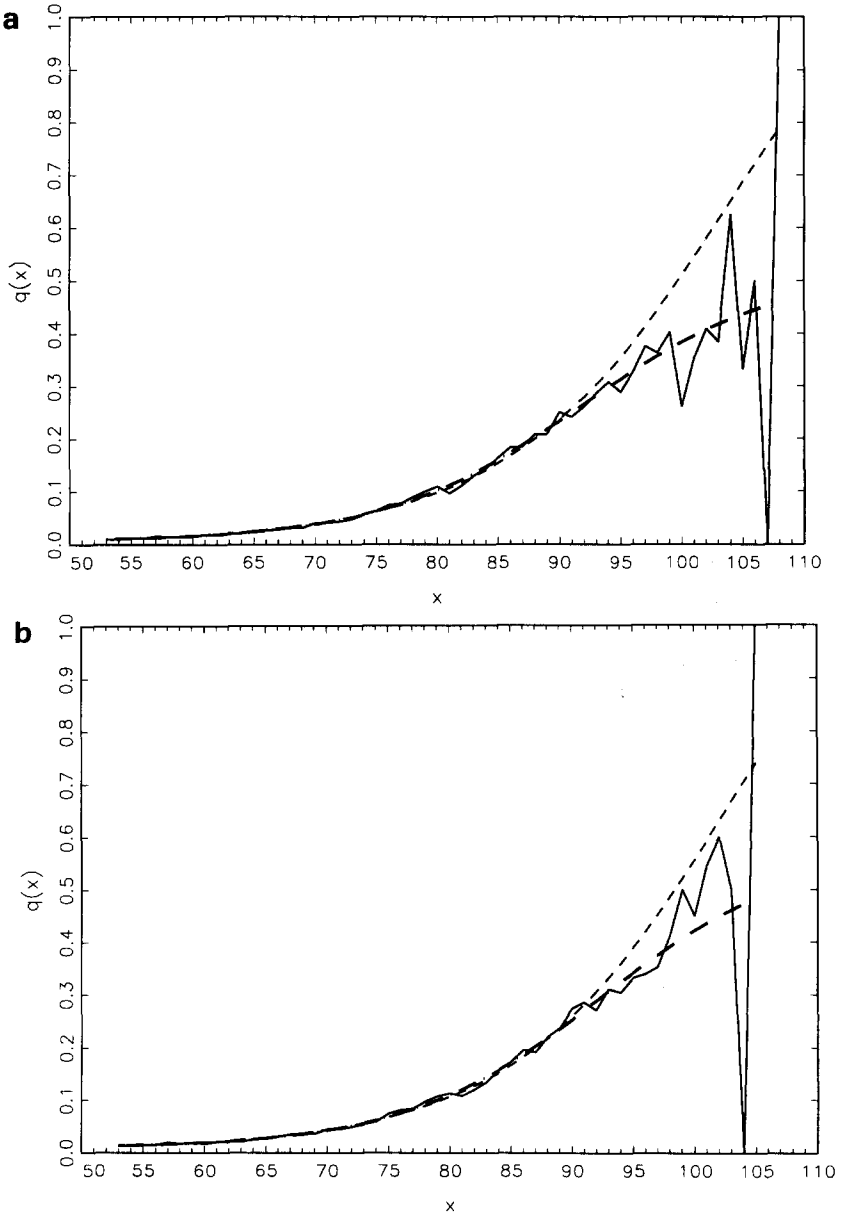


Fig. 1. Annual probability of death $q(x)$ by age x for females (a) and males (b) born in Sweden in 1861. The solid line gives the observed mortality trajectory, the heavy dashed line the gamma-Makeham fit, and the light dashed line the Gompertz-Makeham fit.

ceptibility to death are genetically predetermined and are characterized by frailty Z. Individuals with the same frailty have the same survival chances: death rates change deterministically with age in accordance with the underlying hazard function. A conceptual weakness of this model is that it does not explicitly include the random changes in individual frailty that occur due to environmental influences or stochastic processes of physical deterioration. These components are hidden in the underlying hazard function in some implicit averaged form. As an alternative to this fixed frailty model, a model of randomly changing frailty and stochastic aging can be considered.

3. Effects of stochastic aging

Assume that at age x an individual can be in one of states $0,...,n$ corresponding to $n + 1$ levels of frailty. These states, for example, may be associated with different health states. In any state $i < n$, a person faces a hazard of death and a hazard of moving to state $i + 1$ where the chances of survival are lower. Cohort mortality and survival functions can be analytically calculated if this general model is restricted along the lines suggested by Le Bras [11] and modified by Gavrilov and Gavrilova [12], as described below.

3.1. Equal chances — different lives

Assume that all newborn individuals in a cohort start from the state 0. This means that everybody in the cohort has the same chances of survival when they are 0 years old. Let λ_0 and μ_0 be the transition rates from state 0 to state 1 and to death, respectively. For the i^{th} state let these transition rates be $\lambda_0 + i\lambda$ and $\mu_0 + i\mu$. Note that these hazards do not depend on age, but that they do increase from state to next state. Denote by $P_i(x)$ the probability that an individual age x will be found in the i^{th} state. As shown in [11]:

$$P_0(x) = P_0(0)e^{-(\lambda_0 + \mu_0)x}$$

and

$$P_i(x) = \frac{P_0(x)}{i!}\left[\frac{\lambda - \lambda e^{-(\lambda+\mu)x}}{\lambda + \mu}\right]^i \prod_{k=1}^{i}\left(\frac{\lambda_0}{\lambda} + (k-1)\right), \; i > 0 \tag{6}$$

The number of individuals alive at age x is given by the survival function $S_n(x)$:

$$S_n(x) = \sum_{i=0}^{n} P_i(x) \tag{7}$$

Now assume that the model has an infinite number of states. As the number of states tends to infinity the survival function $S_n(x)$ tends to the limit $S(x)$:

$$S(x) = e^{-(\lambda_0 + \mu_0)x}\left(\frac{\lambda + \mu}{\mu + \lambda e^{-(\lambda+\mu)x}}\right)^{\frac{\lambda_0}{\lambda}} \tag{8}$$

Taking the logarithmic derivative of $S(x)$ we get the formula for observed mortality $\bar{\mu}(x)$:

$$\bar{\mu}(x) = \mu_0 + \frac{\mu\lambda_0[1 - e^{-(\lambda+\mu)x}]}{\mu + \lambda e^{-(\lambda+\mu)x}} \tag{9}$$

Fig. 2a,b shows how parameters of the Le Bras model for female and male Swedish cohorts change with the year of birth of the cohort. Parameters were estimated by the maximum likelihood method. The parameter estimates are clearly correlated, some pairs positively, others negatively. A negative correlation between the parameters of the Gompertz model (when $\mu(x) = ae^{bx}$) was highlighted by Strehler and Mildvan [5]. Riggs [13,14] used this property for analyzing mortality data via reparametrized Gompertz curves. Gavrilov and Gavrilova [12] found a similar negative correlation in the Gompertz-Makeham model. The negative correlation between μ_0 and μ, and between λ and μ in Le Bras model is illustrated by Fig. 3. The nature and properties of these correlations merit further attention.

When $\mu \ll \lambda$, then $\bar{\mu}(x)$ may be approximated by

$$\bar{\mu}(x) = \left(\mu_0 - \frac{\mu\lambda_0}{\lambda}\right) + \frac{\mu\lambda_0}{\lambda} e^{(\lambda+\mu)x} \tag{10}$$

which is equivalent to the Gompertz-Makeham mortality model. The general four-parameter model can accommodate a variety of mortality trajectories, including trajectories where mortality rates level off at advanced ages.

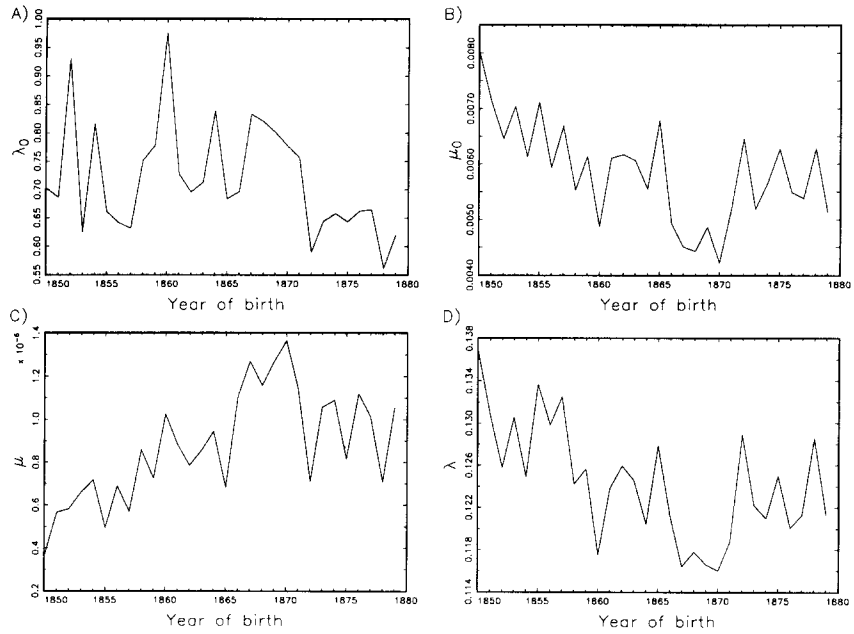
3.2. Average biological age

The various states in the Le Bras model of mortality can be associated with biological age. This interpretation is useful because it permits analysis of cohort differences, male-female differences, and cross-species differences in terms of health-state distributions among survivors. Such a perspective might also be useful in the analysis of prehistorical demographic data when chronological age at death can not be measured directly but must be estimated from the physical condition of skeletal remains. Expressions 5 and 6 allow us to calculate the average biological age (i.e. health state i) among survivors $\bar{i}(x) = \mathrm{E}(i \mid T > x)$. Because

$$\bar{\mu}(x) = \frac{\sum_{i=0}^{\infty} P_i(x)(\mu_0 + i\mu)}{\sum_{i=0}^{\infty} P_i(x)}$$

it follows that

$$\bar{\mu}(x) = \mu_0 + \bar{i}(x)\mu \tag{11}$$



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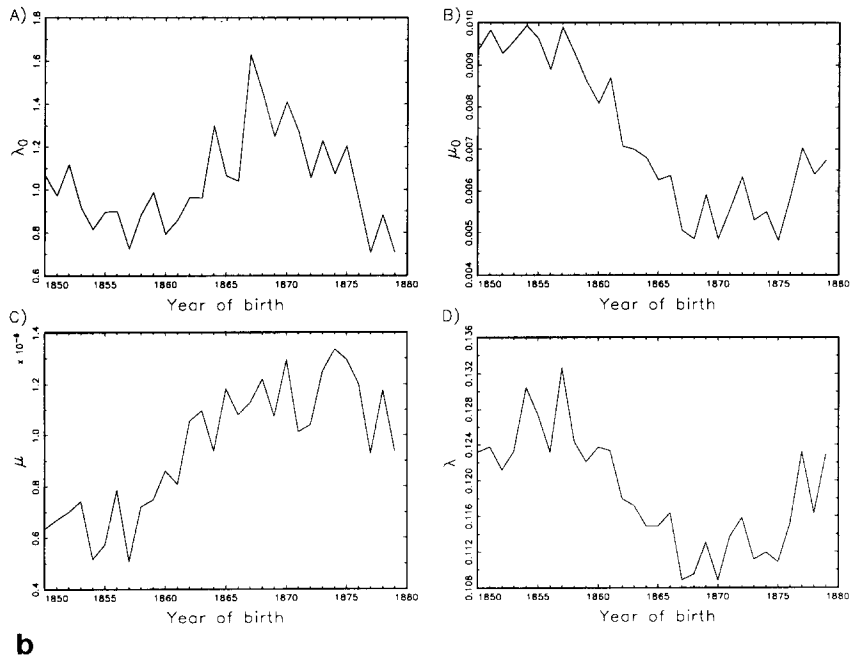

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Fig. 2. (a) Values of the Le Bras model parameters λ_0 A, μ_0 B, μ C, and λ D for Swedish females born in the period 1850–1879. (b) Values of the Le Bras model parameters λ_0 A, μ_0 B, μ C, and λ D for Swedish males born in the period 1850–1879.

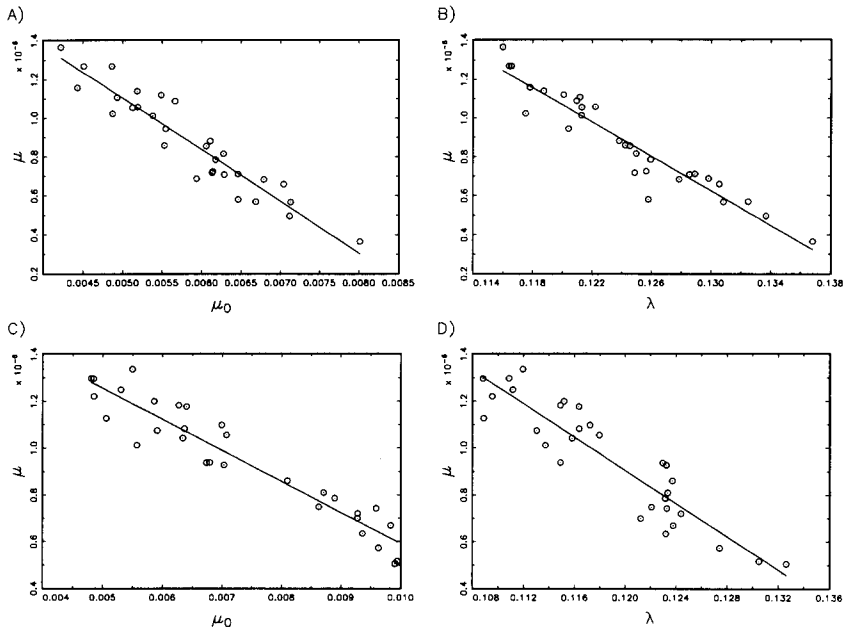

Fig. 3. Negative correlations in fits of Le Bras' model between μ and μ_0 for females A and males C and between μ and λ for females B and males D. Observed values for Swedish female and male cohorts born in the period 1850–1879 are given by circles; the line gives the least squares regression.

and hence

$$\bar{i}(x) = \frac{\bar{\mu}(x) - \mu_0}{\mu} \tag{12}$$

As $x \to \infty$, $\bar{\mu}(t) \to \mu_0 + \lambda_0$, so

$$\bar{i}(\infty) = \frac{\lambda_0}{\mu} \tag{13}$$

Biological age approaches this limiting value because there is eventually a balance between mortality selection on the one hand and debilitation on the other. Survivors cannot have too high a biological age and still be alive. Fig. 4 presents gr. ..is of the limiting average biological age among survivors for Swedish male and female cohorts born in the period 1850–1879. It is interesting to observe that the limiting average biological age declines with year of birth, i.e. survivors at advanced ages in the more recent cohorts are on average healthier. Further investigation from this perspective may provide another view on the possible compression of morbidity [15].

Note that $\bar{i}(x)$ is a measure of the average level of frailty among survivors in the Le Bras model of stochastic aging. This function, however, is different from the aver-

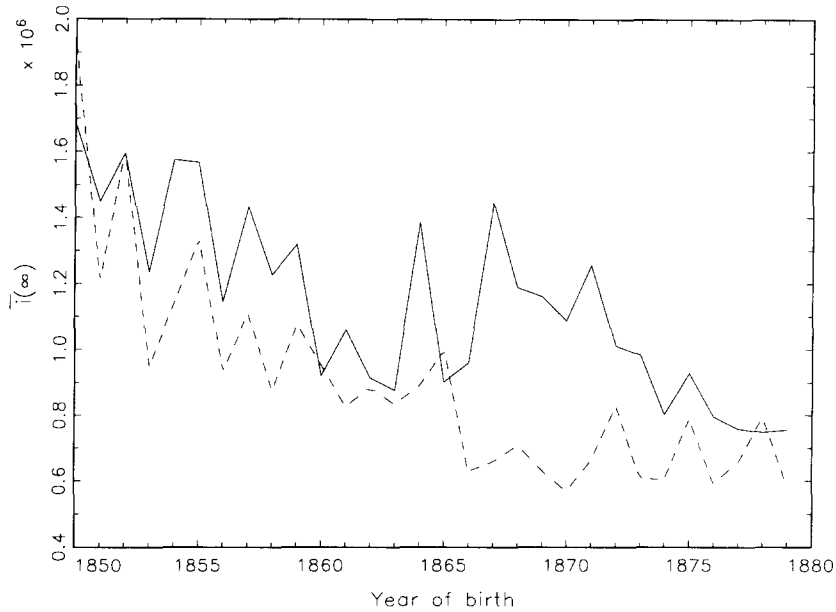

Fig. 4. Limiting average biological age $\bar{i}(\infty)$ for females (– – –) and males (——) born in Sweden in the period 1850–1879.

age frailty among survivors calculated in the case of the fixed frailty model in Eq. 2. Both frailty concepts capture an aspect of reality but in very different ways. It is unclear how important either or both will prove to be in understanding patterns of mortality and aging.

3.3. The balance between selection and debilitation

The observed mortality represented by 9 and 11 results from the influence of two important processes — stochastic aging and debilitation developing on the individual level and mortality selection developing on the population level. The mutual, balancing influence of these processes generates the distribution of surviving individuals. This distribution is given by the survival proportions $\pi_i(x)$. By definition these proportions can be calculated as

$$\pi_i(x) = \frac{P_i(x)}{S(x)}, \; i = 0,1,2,...$$

After substitution of 7, 8 and 14 into this formula we have

$$\pi_i(x) = \frac{1}{i!} V(x)^i (1 - V(x))^{\frac{\lambda_0}{\lambda}} \prod_{k=1}^{i} \left(\frac{\lambda_0}{\lambda} + (k-1) \right) \tag{14}$$

where

$$V(x) = \frac{\lambda - \lambda e^{-(\lambda+\mu)x}}{\lambda + \mu} \tag{15}$$

Since $V(x) \to \frac{\lambda}{\lambda+\mu}$ and $1 - V(x) \to \frac{\mu}{\lambda+\mu}$, we have for $\pi_i(\infty)$

$$\pi_i(\infty) = \frac{1}{i!}\left(\frac{\lambda}{\lambda+\mu}\right)^i \left(\frac{\mu}{\lambda+\mu}\right)^{\frac{\lambda_0}{\lambda}} \prod_{k=1}^{i}\left(\frac{\lambda_0}{\lambda} + (k-1)\right) \tag{16}$$

It is easy to check that $\sum_{j=0}^{\infty} \pi_j(\infty) = 1$

Note that the mean of the $\pi_i(\infty)$'s is $\bar{i}(\infty)$. Hence, Fig. 4 indicates that for most birth cohorts the limiting distribution of π for females is, on average, to the left of the distribution for males. Fig. 5 shows the entire distribution of $\pi_i(\infty)$ for males and females for an illustrative birth cohort, the cohort of 1868. It indicates that the female distribution is shifted to the left. This shift has an interesting interpretation. If i is taken as a measure of biological age, then surviving females at advanced ages are biologically younger (healthier) than surviving males. Since the female distribu-

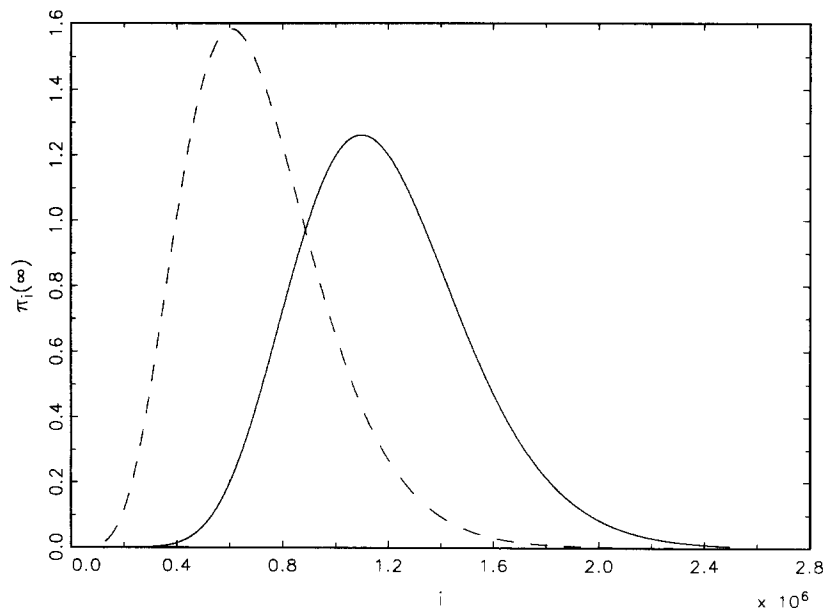

Fig. 5. Limiting distribution of biological age as described by proportions $\pi_i(\infty)$ of individuals in state i for females (– – –) and males (——) born in Sweden in 1868.

tion clearly lies to the left of the male distribution, it can also be concluded that females who die at advanced ages die at a younger biological age than males. Females tend to outlive males and to die at a higher chronological age, but females may die at a younger biological age. Looked at from another point of view, the model suggests that the aging process for females, i.e. the movement to worse states of health, develops more slowly than for males.

3.4. Does it really matter if frailty is genetic or acquired?

How important are genetic factors in determining mortality? How important are environmental influences and internal processes of aging? To capture genetic effects, fixed frailty models (like the gamma-Makeham model) have been developed. The influence of environmental factors and a randomly developing health deterioration process can be studied with the help of changing frailty or acquired heterogeneity models (like the Le Bras model). It turns out that mortality data alone does not permit a test of which kind of effect is more important. More exactly, the following statement holds true:

Proposition 1. Using survival data one can not distinguish between the Le Bras model of randomly changing frailty (9) and the gamma-Makeham model of fixed frailty (3).

Proof. Model 9 can be rewritten as

$$\bar{\mu}(x) = \lambda_0 + \mu_0 - \frac{\lambda_0(\lambda + \mu)}{\lambda + \mu e^{(\lambda + \mu)x}} \tag{17}$$

Let us represent (3) in a form similar to (17). After simple transformations we get

$$\bar{\mu}(x) = \frac{b}{\sigma^2} + c - \frac{\frac{b}{\sigma^2} - a}{\left(1 - \sigma^2 \frac{a}{b}\right) + \sigma^2 \frac{a}{b} e^{bx}} \tag{18}$$

Comparing the coefficients in 17 and 18 and solving the system of respective algebraic equations we get for λ, λ_0, μ, μ_0:

$$\begin{aligned} \mu_0 &= a + c \\ \lambda_0 &= \frac{b}{\sigma^2} - a \\ \lambda &= b - \sigma^2 a \\ \mu &= \sigma^2 a \end{aligned} \tag{19}$$

Solving these equations with respect to a, b, c and σ^2, we get

$$
\begin{aligned}
a &= \frac{\lambda_0}{\lambda}\mu \\
b &= \lambda + \mu \\
c &= \mu_0 - \frac{\lambda_0}{\lambda} \\
\sigma^2 &= \frac{\lambda}{\lambda_0}
\end{aligned}
\tag{20}
$$

Relations 19 and 20 establish the one-to-one correspondence between the parameters of Le Bras and gamma-Makeham mortality models. Thus, if one model gives a good fit to the data, the other model gives an equally good fit. This situation creates an important methodological problem: how should the results of statistical analysis be interpreted?

3.5. Compromise solution? — one more problem

One might expect that the truth lies in a compromise solution: a cohort is heterogeneous from the very beginning and then it undergoes a stochastic process of aging. So a superior model would allow for an initial distribution with respect to health states with further stochastic evolution starting from each of these states. For example, one could take the proportions $\pi_i(x_0)$, i = 0,1,...for some x_0 as an initial distribution of frailty for newborn individuals and allow for stochastic evolution in accordance with Le Bras' model. It is clear from this construction that the observed mortality rate $\tilde{\mu}(x)$ at age x for this population coincides with $\bar{\mu}(x_0 + x)$. It turns out that parametric description of this mortality curve is the same as the initial Le Bras model. More exactly the following statement holds true:

Proposition 2. Using survival data, the stochastic aging model in which all individuals in a cohort start from the '0' state is indistinguishable from a heterogeneous stochastic aging model in which individuals in the cohort start from different states with the initial distribution $\tilde{P}_i(0)$ defined as

$$\tilde{P}_i(0) = \pi_i(x_0)$$

where the $\pi_i(x)$ are defined by 14 for arbitrary x_0.

Proof. Note that observed mortality in the model of stochastic aging can be represented in the form

$$\bar{\mu}(x) = \omega - \frac{\gamma}{1 + \alpha e^{\beta x}} = f(x;\alpha,\beta,\omega,\gamma) \tag{21}$$

where $\omega = \lambda_0 + \mu_0$, $\beta = \mu + \lambda$, $\gamma = \lambda_0 \frac{\beta}{\lambda}$, $\alpha = \frac{\mu}{\lambda}$. It is enough to show that for some parameters α', β', ω', γ':

$$f(x_0 + x,\alpha',\beta',\omega',\gamma') = f(x,\alpha,\beta,\omega,\gamma) \tag{22}$$

It is clear from 21 that 22 will be satisfied, if $\beta' = \beta$, $\omega' = \omega$, $\gamma' = \gamma$, and $\alpha' = \alpha e^{-\beta x_0}$.

3.6. Which state do we start with?

Another possible modification of the Le Bras model deals with changes of the initial state. Note that the structure of this model is such that if all individuals in the cohort start from the j^{th} state, the parametric structure of the model remains the same with natural substitution of λ_0 and μ_0 by $\lambda_0(j) = \lambda_0 + j\lambda$ and $\mu_0(j) = \mu_0 + j\mu$ i.e.

$$\bar{\mu}_j(x) = \mu_0(j) + \frac{\mu\lambda_0(j)[1 - e^{-(\lambda+\mu)x}]}{\mu + \lambda e^{-(\lambda+\mu)x}}$$

Thus from the statistical point of view, it is impossible to say whether the cohort starts from state '0' or from state 'j'.

3.7. Mortality in sovereign populations

Assume that in the compromise model (i.e. in the model with initial heterogeneity and subsequent stochastic aging), we select a subpopulation which at time zero is in state i. Starting from this time, we are interested in the future of only this subpopulation. The observed mortality for this subpopulation is given by the formula

$$\bar{\mu}_i(x) = \mu_0(i) + \frac{\mu\lambda_0(i)[1 - e^{-(\lambda+\mu)x}]}{\mu + \lambda e^{-(\lambda+\mu)x}} \tag{23}$$

with $\lambda_0(i) = \lambda_0 + i\lambda$, $\mu_0(i) = \mu_0 + i\mu$ and with the same λ and μ. This means that the functional forms of the observed mortalities for subpopulations are the same and differ only by parameter values.

3.8. Survival in the empire

Assume that in the compromise model we mark the subpopulations which are located in each state at time zero. Starting from this time we are interested in the evolution of these (infinite number) of marked subpopulations. Each of these subpopulations evolves in accordance with its own mortality law $\bar{\mu}_i(x)$ given by (23) and survival function

$$S_i(x) = e^{-\int_0^x \bar{\mu}_i(u)du} \tag{24}$$

The survival function for the total population can be represented as

$$S(x) = \sum_{i=0}^{\infty} \pi_i(x_0) S_i(x) \tag{25}$$

We already know that the survival function $S(x)$ corresponds to a logistic mortality

rate $\tilde{\mu}(x) = f(x;\alpha,\beta,\omega,\gamma)$. Thus a population with logistic observed mortality can be represented as a mixture of populations with exactly the same (i.e. logistic) functional forms of mortality.

4. Discussion

Assume that there are two groups of researchers who deal with mortality at older ages in Sweden. Both groups would like to investigate the mechanisms of mortality and aging. The first group believes that genetic factors play a key role in the aging process. The second group believes that a stochastic process of debilitation underlies the observed mortality pattern. To analyze the Swedish data, these two groups try to use an appropriate model of aging that is consistent with their assumptions. The first group prefers the gamma-Makeham model. The second group chooses the Le Bras model.

Both groups fit their model and discover that their model fits the data well. In their reports, the groups provide interpretations of their findings. Researchers in the first group infer that they have found evidence of the presence of unobserved fixed heterogeneity in human mortality. They argue that this heterogeneity can be interpreted as reflecting genetic factors. Representatives of the other group believe that they have found evidence that observed patterns of human mortality result from a process of stochastic aging and changing frailty. They conclude that health deterioration and environmental influences are responsible for the observed Swedish mortality pattern.

Suppose now that at some stage of their studies the groups of researchers discover that the two different concepts used in their research not only fit the data equally well but also yield exactly the same model of observed mortality. Moreover, they find that several other concepts lead to the same parametric structure of the observed mortality rate. What should the researchers do? The situation resembles the problem of non-identifiability in proportional-hazard models with fixed frailty: as discussed by Hoem [16], many underlying mortality rates and many frailty distributions produce the same mortality pattern. Our results show that changing frailty models cannot be distinguished from a fixed frailty model (the gamma-Makeham model). This feature demonstrates an important duality in aging, analogous to the duality in physics between wave and particle theories. The observed pattern of aging can be explained in at least two different ways, which involve two different concepts of changing chances of death with age on the individual level. The concepts are not contradictory but complimentary. They focus attention on different aspects of the complicated process of survival.

5. Conclusion

In the statistical analysis of data, results and conclusions depend not only on the data per se but also on basic assumptions about the mechanisms which generated the data. It is very important to know how much a data set itself 'can say'. Researchers often become disappointed when they learn that data sets frequently tell us surprisingly little, or, at least, not so much as we expected. In this article, we have

shown that mortality data alone cannot solve an important problem concerning the nature of survival mechanisms. In particular, we proved that without additional covariates or assumptions, the fixed frailty gamma-Makeham mortality model cannot be distinguished from the Le Bras model of stochastically changing frailty, and several changing frailty models cannot be distinguished from each other. These examples illustrate that even when a model fits the data, the concepts used to construct the model may not be correct.

6. Acknowledgement

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