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Mortality Increase in Late-Middle and Early-Old Age: Heterogeneity in Death Processes as a New Explanation

Ting Li · Yang Claire Yang · James J. Anderson

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Abstract Deviations from the Gompertz law of exponential mortality increases in late-middle and early-old age are commonly neglected in overall mortality analyses. In this study, we examined mortality increase patterns between ages 40 and 85 in 16 low-mortality countries and demonstrated sex differences in these patterns, which also changed across period and cohort. These results suggest that the interaction between aging and death is more complicated than what is usually assumed from the Gompertz law and also challenge existing biodemographic hypotheses about the origin and mechanisms of sex differences in mortality. We propose a two-mortality model that explains these patterns as the change in the composition of intrinsic and extrinsic death rates with age. We show that the age pattern of overall mortality and the population heterogeneity therein are possibly generated by multiple dynamics specified by a two-mortality model instead of a uniform process throughout most adult ages.

Keywords Mortality acceleration · Sex differences · Two-mortality processes · Vitality

Introduction

The highly regulated, yet complex, age-specific pattern of overall mortality rate has long suggested the potential for a universal law of mortality (Carnes et al. 1996;

T. Li (✉)
Center for Population and Development Studies, Renmin University of China, No. 59 Zhongguancun Street, Haidian District, Beijing 100872, China
e-mail: tingli@email.unc.edu

Y. C. Yang
Carolina Population Center, Lineberger Comprehensive Cancer Center, and Department of Sociology, University of North Carolina, Chapel Hill, NC, USA

J. J. Anderson
Center for Studies in Demography and Ecology and School of Aquatic and Fishery Sciences, University of Washington, Seattle, WA, USA

Olshansky and Carnes 1997). The most important finding relating to this is arguably the Gompertz law, which shows an exponential rise in death rate with age (Gompertz 1825). Because the law is considered to be a good approximation of the mortality pattern that occurs between sexual maturity and old age, recent research has paid more attention to what happens beyond rather than within the predictable age interval of the law. Researchers have generally observed that a leveling off of the mortality rate at very old ages (later than age 85)—the so-called old-age plateau—would directly follow the exponential increase (Carey and Liedo 1995; Horiuchi and Wilmoth 1998; Vaupel et al. 1998), although controversy remains regarding the age to which the Gompertz law can be extended (Gavrilov and Gavrilova 2011). However, a relatively subtle but systematic deviation from the law between late-middle and early-old ages (50–70) was identified in a few studies, which found that the rate of mortality increase became either faster or slower than the predicted exponentially increasing trajectory (Ekonomov et al. 1989; Himes et al. 1994; Horiuchi 1983, 1997; Horiuchi and Coale 1990; Horiuchi and Wilmoth 1998; Milne 2007; Pakin and Hrisanov 1984).

Such a deviation within the predictable age range of the law has been largely ignored in the literature (Milne 2007). As a result, the mechanism underlying this phenomenon remains unclear. The lack of discussion on the phenomenon may be in part because the pattern of deviation varies between the two sexes and across periods; females exhibited consistent mortality acceleration in the 50–70 interval in all periods of a recent 60-year period (Horiuchi 1997; Milne 2007; Pakin and Hrisanov 1984), whereas males exhibited significant diversity in the same age interval across periods. In early periods (e.g., 1950–1979), male mortality did not accelerate notably and even decelerated in some countries (Horiuchi and Wilmoth 1997; Pakin and Hrisanov 1984); in more-recent periods, their mortality pattern has appeared to reverse and converge to the female pattern (Milne 2007).

Just as the old-age mortality plateau has stimulated a variety of theories in both demography and biodemography (Gavrilov and Gavrilova 1991, 2001; Mueller and Rose 1996; Vaupel et al. 1979; Yashin et al. 2001), the late-middle and early-old age mortality deviations are also likely to be informative about the dynamics of the adult mortality process. Because national mortality patterns based on vital statistics are quite robust, consistent deviations from the expected Gompertz trajectory are unlikely to be caused by random fluctuations. Rather, they are likely to reflect underlying mechanisms of mortality beyond the extant understandings and interpretations of the Gompertz law. In particular, the sex-differential and time-varying deviations from the expected mortality trajectory provide a unique opportunity for developing hypotheses about the changing dynamics of human mortality and sex differences in aging and longevity.

In this study, we first explore both previous hypotheses and our new hypothesis for the complex patterns of mortality increase observed in late-middle and early-old age. We next examine the patterns in 16 low-mortality countries, and test the newly proposed hypothesis based on a two-mortality dynamics model (Li and Anderson 2013).

Previous and Newly Proposed Hypotheses

The first early hypothesis considers the deviations at ages 50–70 to be a continuation of a mortality acceleration at early-middle ages (25–35), which is well recognized in

the literature as the consequence of a constant background mortality (Makeham term) adding to the exponentially increasing senescent mortality (Horiuchi and Wilmoth 1998; Makeham 1860). Because senescent mortality becomes dominant with aging (above age 40), the acceleration pattern should quickly diminish and the mortality trajectory ought to return to the Gompertz schedule at around age 40. In contrast, at ages 50–70, mortality exhibits another round of either acceleration or deceleration; therefore, these patterns cannot be attributed to an age-dependent masking of a background process similar to that explaining the early-middle age pattern of mortality acceleration. Empirically, extant Gompertz-like models with a constant Makeham term still cannot precisely capture the mortality pattern in early-old age (Bongaarts 2005), which serves as further evidence against the first hypothesis.

A second hypothesis considers later-life mortality deviations in terms of changes in sex hormone profiles with age. Based on the sex-differential patterns of mortality in the 1970s, this hypothesis postulates that the acceleration in female mortality is due to the loss of estrogen and hence loss of protection from various physiological disorders at post-reproductive ages (Horiuchi 1997). Compared with the gradual decline in male fecundity, female menopause may trigger an abrupt acceleration in senescence and consequently an acceleration in mortality. Considering age-related increases in senescence and mortality as "a consequence of biological processes calibrated to the reproductive biology of a species" (Carnes et al. 1996:232), this hypothesis provides a plausible explanation for the female mortality acceleration at postmenopausal ages. However, it explains neither the decelerating trends for male populations in early periods nor the recent convergence of male and female patterns. In essence, the second theory is inadequate to account for all the observed variations.

Given the convergence of male and female patterns in recent years, a third hypothesis, based on a concept of "repair senescence" (Horiuchi 2003), proposes that the observed mortality acceleration is a result of age-dependent changes in the rate of individual aging for both males and females. Specifically, it assumes that the rate of individual physiological deterioration (aging) increases in early-old age because of a decline in the rate of damage repair. Consequently, the rate of mortality increase, which is presumably associated with the pace of aging, accelerates. However, this hypothesis is still unable to explain the sex and period variations in the acceleration patterns. Essentially, a mathematical framework quantifying these differences is missing. In addition, because there is no well-established metric or battery of biomarkers to measure such changes in the basic aging rate, this hypothesis is also hampered by its intangibility.

In light of these deficiencies, we propose an alternative hypothesis. Instead of a senescent mortality process acting uniformly throughout most adult ages, as assumed by some mortality and aging theories that applied the simple exponential form on the adult mortality pattern (e.g., Strehler and Mildvan 1960), multiple processes may jointly shape the pattern of senescent mortality, thereby contributing to deviations from the exponential increase trajectory. The age patterns of mortality increase therefore reflect changes in the contributions of different mortality processes with age.

To explore this hypothesis, we borrow insights from a recent model developed by Li and Anderson (2013), which partitions adult mortality into intrinsic and extrinsic categories. In this model, the intrinsic category represents death from chronic damage accumulation, and the extrinsic category refers to death caused by acute forces that

destroy the capacity to survive. Assuming that both mortalities change with age, this two-mortality framework more flexibly accommodates the changing dynamics of mortality than was possible with previous frameworks. Thus, in principle, this framework can better address the sex and period variations observed at ages 50–70.

Besides testing the hypothesis that the late-middle and early-old mortality patterns involve multiple mortality processes, we also expect to arrive at findings that will inform a more general model of heterogeneous death processes that bear specific biological or social relevance.

Patterns of Mortality Increase in Late-Middle and Early-Old Age

As a first step, a thorough exploration of the change in the age pattern of mortality increase is crucial to test our hypothesis. In this regard, previous studies have several limitations. First, early studies were limited in historical coverage and, in particular, lacked the most recent data. As a result, they could not capture the changes in dynamics across time (Ekonomov et al. 1989; Himes et al. 1994; Horiuchi 1983; Horiuchi and Coale 1990; Pakin and Hrisanov 1984). Second, although a recent study by Milne (2007) covered longer periods, the analysis relied on a qualitative comparison of mortality shapes between two discrete age bands rather than a quantitative analysis of continuous changes in mortality with age. Finally, the works noted previously were primarily based on period data and thus lacked a consideration of the confounding effects of cohorts. A growing body of literature suggests that cohort variations in smoking behavior, nutrition status, obesity level, and other risk factors may produce notable differences between period and cohort patterns (Fogel 1994; Preston and Wang 2006; Yang 2008). Therefore, to comprehensively understand the mortality deviations in late-middle and early-old age, it is necessary to characterize both period and cohort age patterns of mortality increase to the most recent years.

Data and Methods

We used data from the Human Mortality Database (HMD n.d.). The overall death rates of males and females are available in the database from ages 0 to 110 or older, with one-year increments denoted as M_x, where x indicates single year of age. We selected both period and cohort data from 16 industrialized countries: Sweden, the United Kingdom, Italy, Japan, the Netherlands, Spain, the United States, France, Switzerland, Denmark, Australia, Canada, Belgium, Norway, Finland, and Austria. For each country, we examined six periods (1950–1959, 1960–1969, 1970–1979, 1980–1989, 1990–1999, and 2000–2007/2008/2009) and four cohorts spanning a decade each (1880–1889, 1890–1899, 1900–1909, and 1910–1919). These samples cover most industrialized countries that have relatively reliable population mortality data. We used only data from the post-1950 period, when background mortality (i.e., the age-invariant mortality defined as the Makeham term) was relatively small. By doing so, we sought to minimize the effects of background mortality on the later-life senescence-related mortality patterns.

We examined the shape of mortality increase with age, which can measure deviations from the Gompertz law, by calculating the life table aging rate (LAR). The

LAR is defined as the percentage change in mortality rate at each age (Carey and Liedo 1995; Horiuchi and Coale 1990):

$$LAR(x) = \frac{1}{\mu(x)}\frac{d\mu(x)}{dx} = \frac{d\log\mu(x)}{dx}, \quad (1)$$

where $\mu(x)$ is the force of mortality at age x. When mortality curves are fit to the Gompertz model, $\mu(x) = a\exp(bx)$, the LAR corresponds to the slope parameter b, which is constant over age. Empirically, the LARs vary across ages, suggesting deviations from the Gompertz law. For example, a decline of the LAR indicates mortality deceleration, and an increase of the LAR indicates acceleration. The age pattern of LARs has been less frequently studied than that of mortality rates. However, an LAR plot can be more sensitive to the change of underlying dynamics of aging than a logarithmic plot of mortality rates against age, because a nearly straight line of log death rates could have LARs that change substantially with age (Horiuchi and Coale 1990; Horiuchi et al. 2003).

The LAR can be estimated using discrete data of age-specific death rate M_x by (Horiuchi and Coale 1990)

$$\hat{LAR}(x) = [\log M_x - \log M_{x-\Delta x}]/\Delta x, \quad (2)$$

where, for our analysis, the age interval is $\Delta x = 1$ year. We smoothed the sequence of the LAR by taking moving averages of seven successive values to reduce the stochastic variation in death rates. This method tends to "flatten original patterns to some extent by lowering peaks and raising troughs" (Horiuchi and Coale 1990:247), but as long as the observed trends are not driven by random processes, they should persist under the graduation procedure.

Results

The age patterns of the LARs in all 16 countries by time period are summarized in Fig. 1. In this study, we focus only on the pattern between ages 40 and 85. The LARs are not constant with age as would be expected if mortality followed the Gompertz law. In fact, they vary from 0.06 to 0.13 across countries and periods, implying that death rates rise at varying exponential rates between 6 % and 13 % per year of age.

Consistent with Horiuchi's (1997) finding, all female populations, except that in the United Kingdom in 1990s, exhibit a remarkable increase in the LARs around ages 50–70, indicating an acceleration of mortality increment in this age band. The pattern persists across all examined periods. Although the starting ages of increasing LARs vary from population to population of females, there is a general tendency of increasing delays in those ages over time. In contrast, the male populations show greater diversity across years. From the 1950s to the 1970s in many male populations, LARs tended to rise between ages 40 and 50, which is earlier than for their female counterparts. The LARs then reached a plateau (in Sweden, Japan, Spain, and Norway) or declined (in all other countries), corresponding to the late-middle and early-old age mortality decelerations reported in earlier studies (Himes et al. 1994; Milne 2007). Beginning in the 1980s, several male populations (e.g., those of France and Japan) showed late-life LAR increases. In all countries at the most recent period

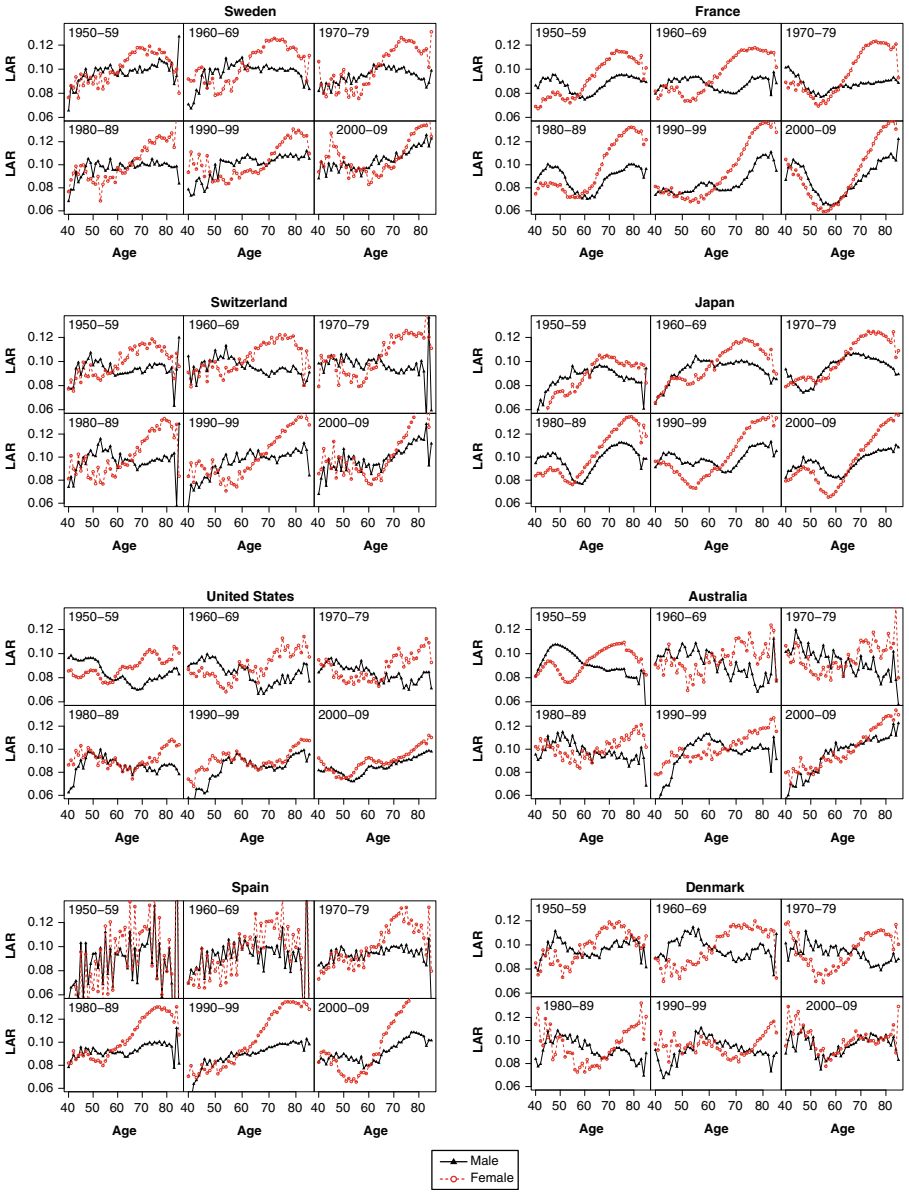


Fig. 1 Age-specific pattern of LAR from 16 selected industrialized countries in 6 periods (1950–1959, 1960–1969, 1970–1979, 1980–1989, 1990–1999, 2000–2007/2008/2009)

(the 2000s), LARs tended to increase at similar ages for males and females. We found significant increases in LARs for 9 out of 16 countries—Sweden, Switzerland, France, Japan, Spain, Belgium, Norway, Finland, and Austria—and moderate increases in the other countries. The age patterns in LARs across six periods suggest that the acceleration of mortality in late-middle and early-old age persisted for females across time, but the acceleration pattern also became more evident in male populations in recent periods.

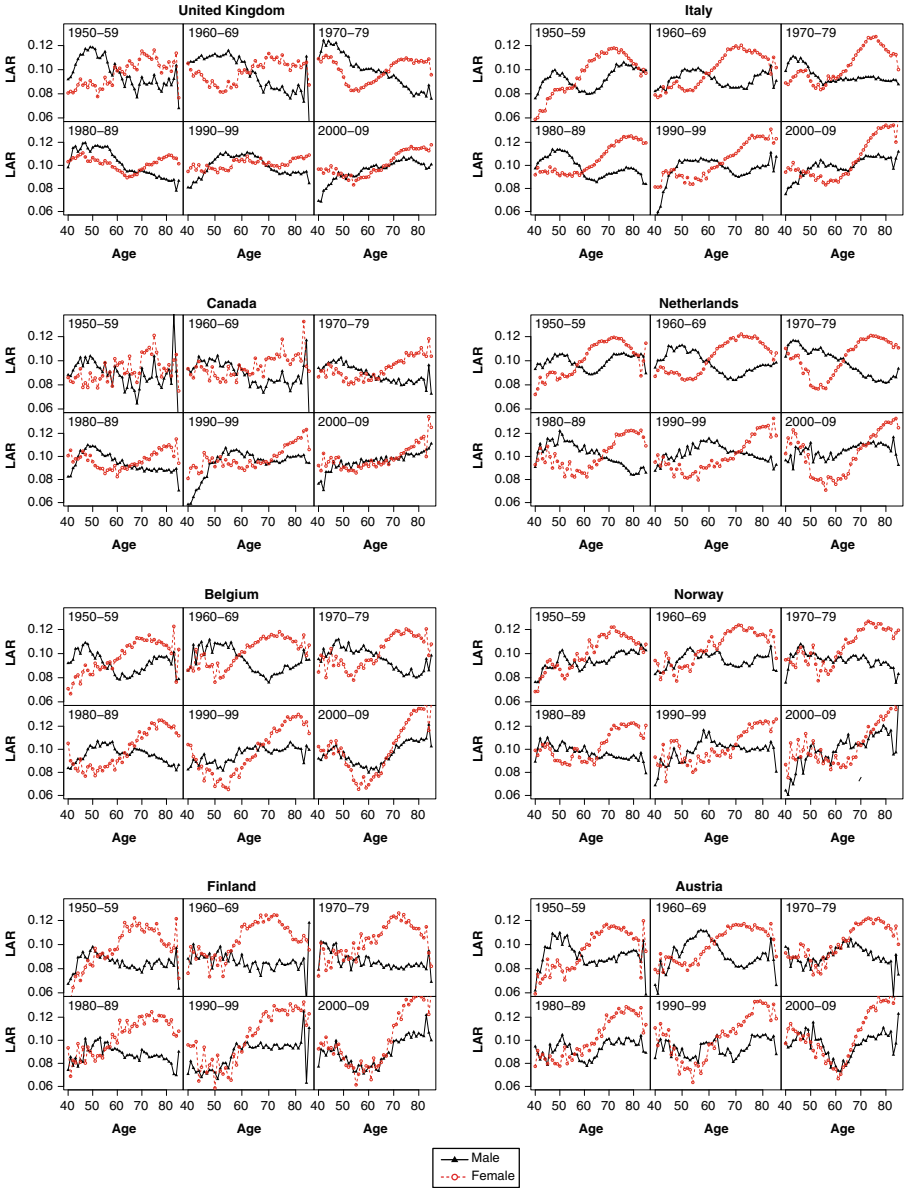


Fig. 1 (continued)

For early cohorts (born in 1880–1899) (Fig. 2), males and females show a surprisingly similar rise in the LARs from middle age onward, even prior to the age of menopause. But for more-recent cohorts (born in 1900–1919), the LAR pattern exhibits a more significant and earlier "hump" for males than for females. These cohort patterns are complex because they are also coupled with period effects that changed more dramatically in the examined populations (Horiuchi et al. 2003). However, the pattern of increasing LARs in middle or older ages for both sexes generally holds and even occurs simultaneously for nearly all early cohorts (born in 1880–1900). Therefore, the

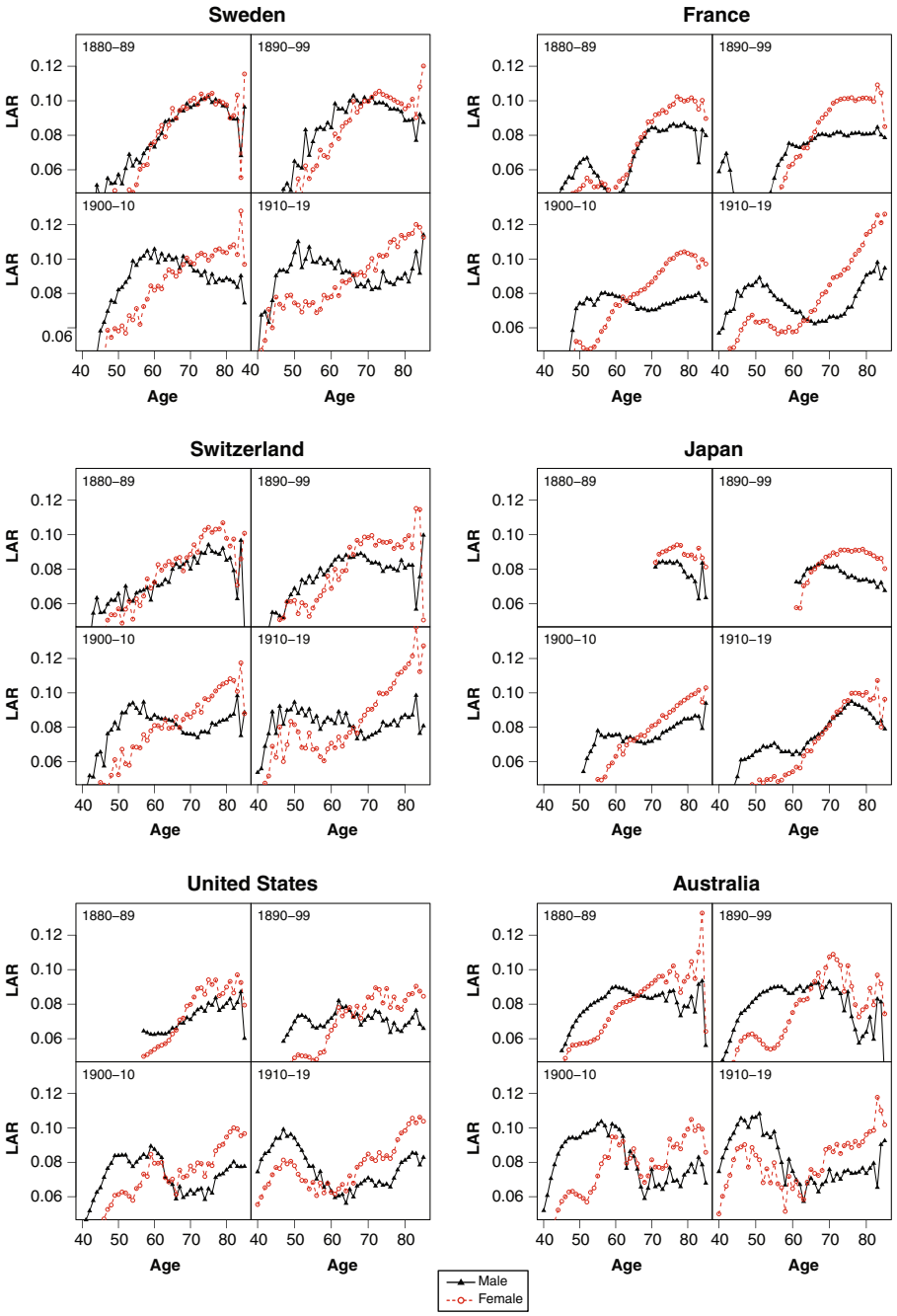
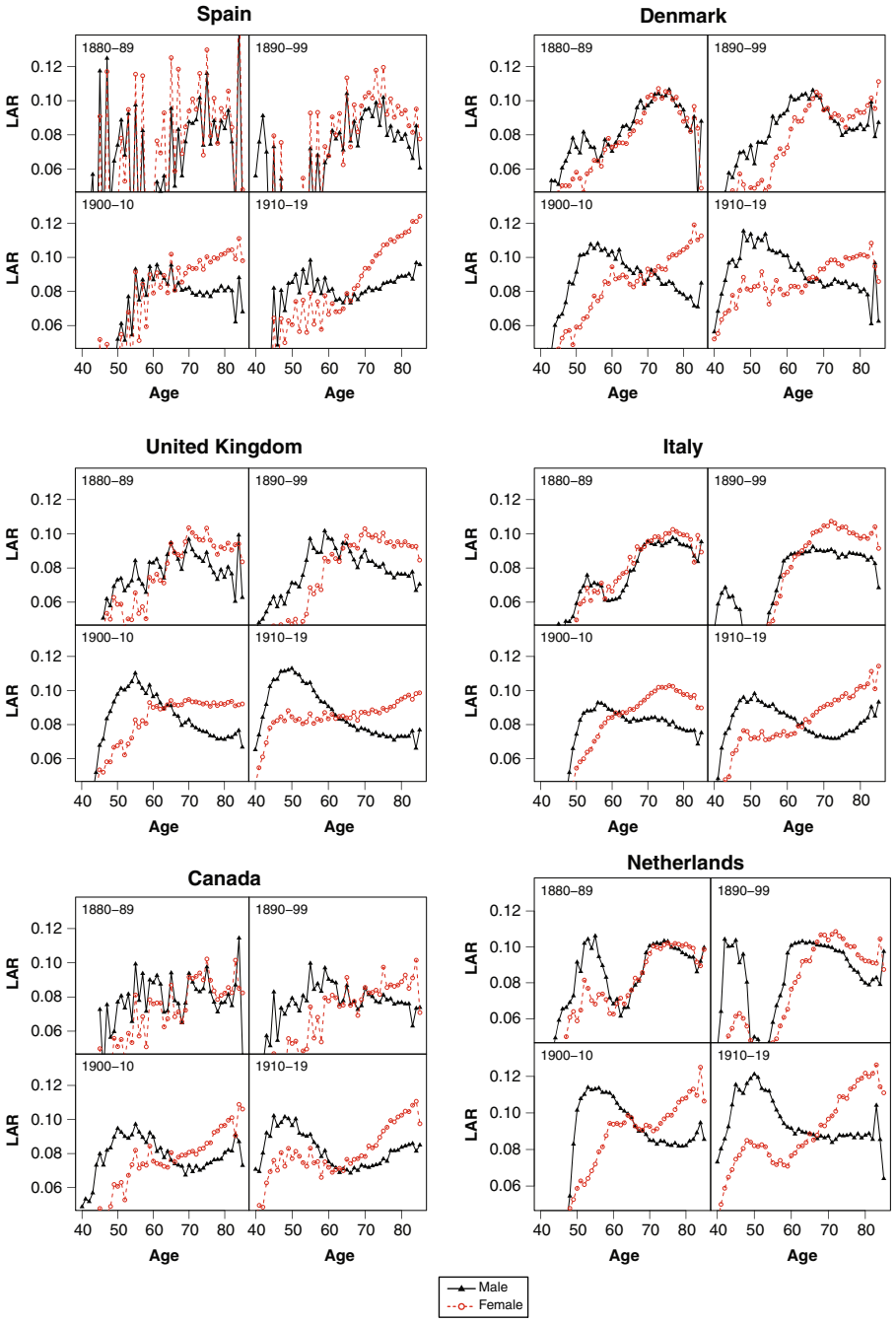


Fig. 2 Age-specific pattern of LAR from 16 selected industrialized countries in four cohorts (1880–1889, 1890–1899, 1900–1910, 1910–1919)

patterns of mortality increase, especially the acceleration, are not likely to be the consequence of pure cohort or period effects, but rather reflect underlying dynamics of aging that are common to both sexes.



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Neither the period nor the cohort patterns in the LARs between ages 40 and 85 are precisely consistent with the Gompertz law prediction that the rate of

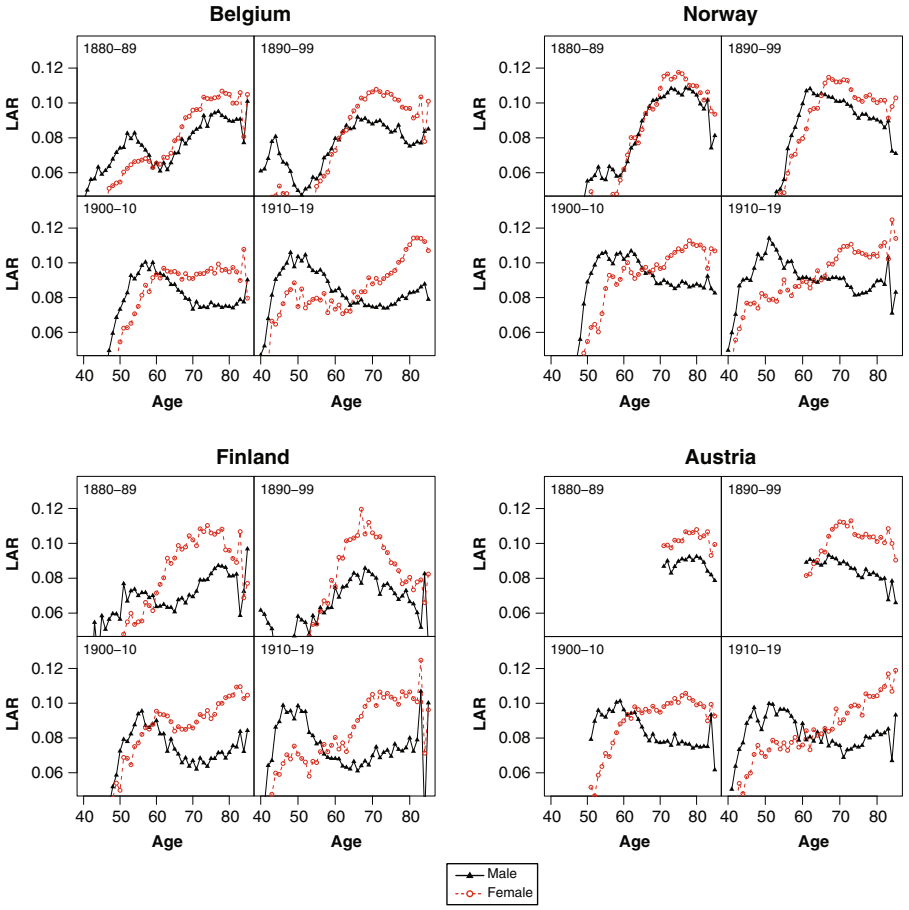

Fig. 2 (continued)

mortality increase is invariant with age. And neither set of patterns entirely supports the estrogenic hypothesis that age patterns of fecundity decline and the sex hormone profiles are the major causes of the sex differences in the LAR patterns. Also, the "repair senescence" hypothesis alone is insufficient to explain the diversity of the patterns. Two questions arise: (1) what is the fundamental cause for the patterns of mortality increase in late-middle and early-old age, and (2) why do these patterns differ by sex in some populations but tend to converge for the two sexes in others? Extant mortality theories cannot adequately address these questions, possibly because many of them rest on an assumption that mortality and its interaction with aging are uniform throughout adulthood (Horiuchi et al. 2003). Here we present a two-process view of mortality that provides a new conceptual framework for understanding the observed patterns of mortality increase during the latter half of the age range in the Gompertz model prediction. These patterns have been less frequently studied but potentially can yield important insights into the biology of aging and mortality in humans.

The Two-Mortality Hypothesis

Previous studies based on the cause-of-death (COD) data have shown that whereas middle-age mortality is dominated by acute and infectious diseases, many old-age mortalities are related to the progressive decline or failure in various physiological functions (Carnes et al. 2006; Gessert et al. 2002, 2003; Horiuchi et al. 2003). As Horiuchi et al. (2003) and Gessert et al. (2003) suggested, these patterns imply that the processes leading to death may be fundamentally different for these two life stages. The late-middle and early-old age patterns of mortality increase, therefore, are likely to be a phenomenon associated with the transition between the two stages. However, the boundary between the two causes of death is rather blurred because mortality rates of both processes increase with age (Carnes et al. 2006, 2008). It is problematic to consider the middle-age mortality to be unrelated to senescence or purely determined by external factors, because the frailty to external stresses also depends on the aging-related internal stability. Instead of relying on an ambiguous classification for the specific cause of death, we focus on illustrating the different processes that lead to death and how they produce the overall mortality patterns.

Method: The Two-Mortality Model

A two-mortality Markov process model developed by Li and Anderson (2013) makes it possible to mathematically address the two adult mortality processes together. The model is based on the premise that an individual is born with a fixed survival capacity (i.e. *vitality*), which is randomly depleted throughout life through degenerative processes. Death occurs with complete vitality depletion from either intrinsic damage accumulation or when a large, discrete extrinsic challenge momentarily depletes the current store of vitality (Fig. 3). Following convention (Carnes and Olshansky 1997; Carnes et al. 2006), the former death type is denoted as intrinsic mortality, $\mu_i(x)$, and the latter type is denoted as extrinsic mortality, $\mu_e(x)$. Total mortality, then, is the combination of the two

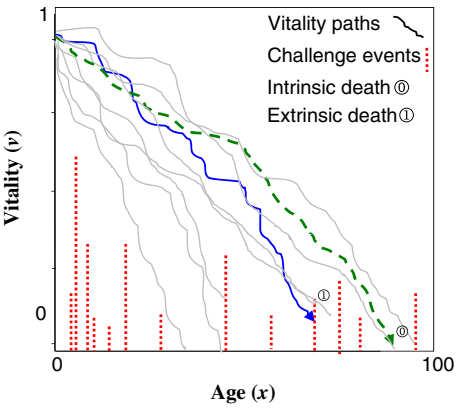

Fig. 3 Individual vitality declines stochastically and death occurs when vitality is exhausted through senescence or when a random extrinsic challenge exceeds the remaining vitality

mortality sources. Next, we illustrate in detail how these two types of deaths are produced by two stochastic processes.

The first process, intrinsic diffusion, is based on the stochastic decline in vitality with age toward an absorbing boundary at which intrinsic mortality occurs (Aalen and Gjessing 2001; Anderson 1992, 2000; Anderson et al. 2008; Li and Anderson 2009; Sacher and Trucco 1962). The process is mathematically specified by a Wiener process characterized by a mean vitality loss rate and a variation term to reflect the heterogeneity within the population that evolves with age. The intrinsic mortality rate is then derived from the death time distribution—that is, the distribution of first arrival time of the Wiener process (marked by the encircled zero in Fig. 3), which in this case is the inverse Gaussian distribution. This process summarizes the gradual degeneration in physiological functions (Shock 1957) through many mechanisms, such as the accumulation of free radicals (Harman 1956), RNA mistranscription (Wiegel et al. 1973), and the shortening of telomeres (Passos et al. 2007). The damage eventually accumulates to the point that it exceeds the body's self-repair capabilities and death ensues.

The second process, extrinsic challenge, is realized with a structure similar to that in the Strehler and Mildvan (1960) general theory of mortality and aging. Extrinsic death occurs when the external challenge magnitudes exceed the remaining vitality (marked by the encircled 1 in Fig. 3). The occurrence of challenges is assumed to follow a random Poisson process, and the distribution of challenge magnitudes is assumed to be exponential. The extrinsic mortality rate can be approximated by an exponentially increasing function with age as in the Strehler and Mildvan theory.

Adult overall mortality is represented as the sum of the two mortality rates:

$$\mu(x) = \mu_i(x) + \mu_e(x). \tag{3}$$

Equation (3) does not imply that these two mortality components are independent from each other. Because they share a common underlying process and parameters describing vitality functions, changing one mortality component would affect the other. Analytical solutions can be derived for both $\mu_i(x)$ and $\mu_e(x)$, and thus for $\mu(x)$, which is characterized by four parameters. We applied the total mortality Eq. (3) to the age-specific overall mortality rates obtained from the HMD. Parameters were estimated via maximum likelihood estimation, through which we were able to generate separately the intrinsic and extrinsic mortality curves with age. Although the model is built on the concept of vitality, we did not utilize an empirical measure but instead used a pure mathematical realization of the underlying vitality processes. The mathematical details and development of this model are described elsewhere (Li and Anderson 2013) and are summarized in the appendix.

In essence, both types of mortalities are aging-related, but they differ from each other in their final pathways to death. Extrinsic mortality involves an acute force that destroys the capacity for living, whereas the intrinsic mortality results from a chronic wearing down of the potential vitality that occurs as a biological destiny. In other words, extrinsic death can be potentially prevented by the elimination of challenges or thwarted by treatments. In contrast, intrinsic death usually cannot be avoided. Thus, the model is a realization of the concept

of heterogeneous death processes by which we can avoid the uncertainties in a case-by-case classification of death.

An Explanation of the Mortality-Increase Patterns

Figures 4 and 5 compare the two-mortality model-estimated measures with period and cohort demographic data for males and females in various years. The age-specific mortality rates with one-year increments were obtained from the HMD for the U.S. and Swedish populations as examples. The fitting algorithm is described in the appendix. The upper panels illustrate age-dependent mortality rates for observed and model fits as well as model-derived intrinsic and extrinsic rates. The middle panels show observed LARs calculated from Eq. (2) and model-fitted mortality rates transformed to LAR by Eq. (1). The lower panels show ratios of model-fitted intrinsic mortality rates to overall mortality rates.

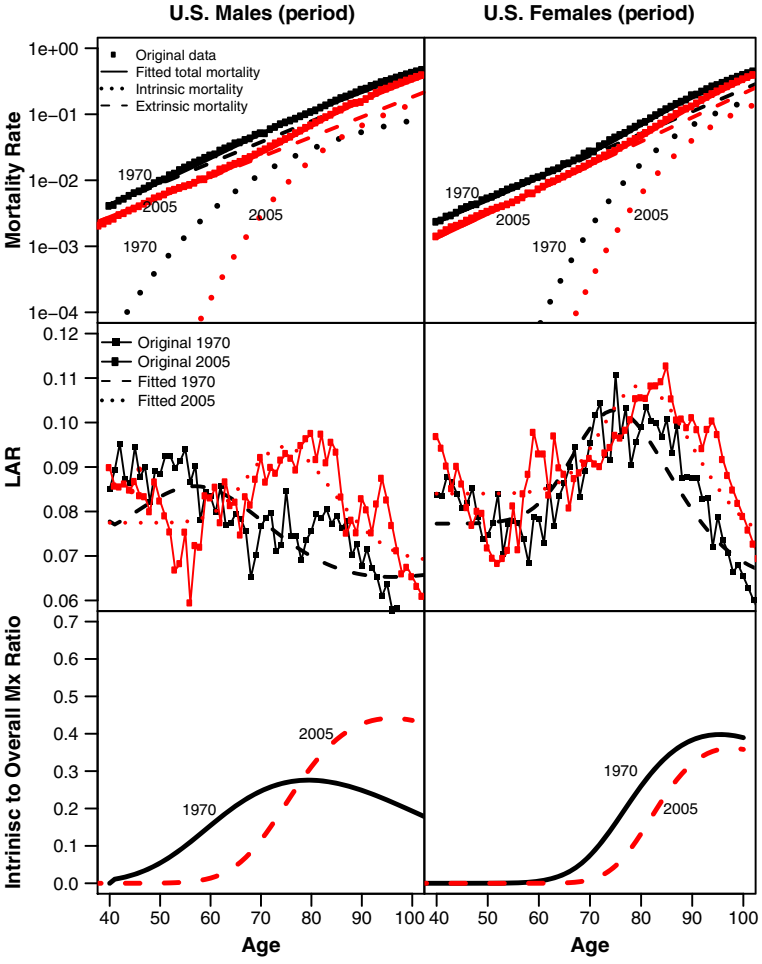

Fig. 4 The two-process mortality model fit to the U.S. period data from 1970 and 2005 for males and females, the corresponding fitted LARs, and the intrinsic mortality proportions

As shown in Figs. 4 and 5, the two-mortality model fits both period and cohort data from the U.S. and Swedish populations remarkably well and, as indicated by the root mean square error and the *F* test (Table 1) (Gallant 1987), the model performs significantly better than the Gompertz model. In particular, the model captures both the acceleration and deceleration patterns in the mortality rise, which are often ignored when data are simply fit with the Gompertz function. We include an age span from 40 to 100 years to show the continuation of the patterns throughout life. In the framework of the two-mortality model, middle- and old-age mortality acceleration is interpreted as the emergence of the intrinsic mortality rate adding to the exponentially increasing extrinsic mortality rate. Derived from the Wiener process with an absorption boundary (see the appendix), intrinsic mortality has a concave shape with age: it first increases, but the rate of increase in late-middle and early-old age decelerates because of the effect of selective survival, and thus of population heterogeneity, at later ages, as frailty theory would suggest (Vaupel et al. 1979). That is, the

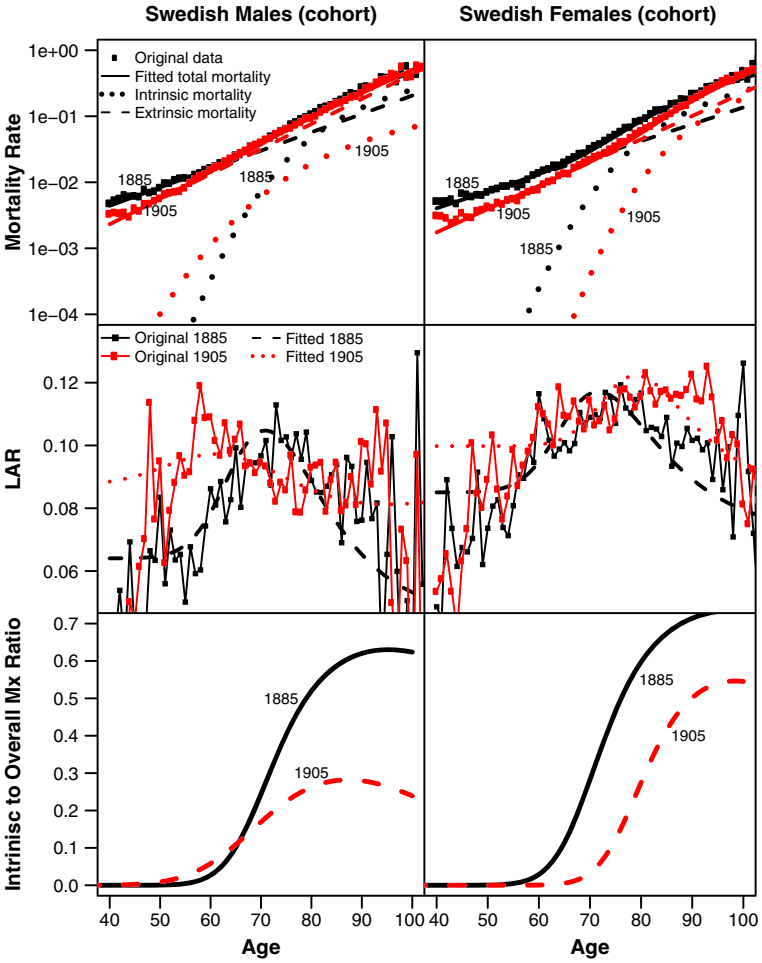

Fig. 5 The two-mortality-process model fit to the Swedish cohort data from 1885 and 1905 for males and females, the corresponding fitted LARs, and the intrinsic mortality proportions

Table 1 Root mean square error (RMSE) for the two-process model and the Gompertz model of log mortality data between ages 40 and 100

Population\Model	Two-Process Model	Gompertz Model	p Value[a]
U.S. Males, Period 1970	0.057	0.069	<.001
U.S. Females, Period 1970	0.048	0.075	<.001
U.S. Males, Period 2005	0.048	0.052	.001
U.S. Females, Period 2005	0.058	0.090	<.001
Swedish Males, Cohort 1885	0.083	0.131	<.001
Swedish Females, Cohort 1885	0.096	0.168	<.001
Swedish Males, Cohort 1905	0.105	0.105	.125
Swedish Females, Cohort 1905	0.134	0.184	<.001

[a] p value is calculated from the F test that compares the fit of the two models.

Markov structure of the vitality process ensures that frailer individuals die early and that stronger, higher-vitality individuals die later, which leads to the accumulation of more-robust individuals at very old ages and hence to the mortality plateau (Aalen and Gjessing 2001; Li and Anderson 2009; Steinsaltz and Evans 2004, 2007; Weitz and Fraser 2001).

Total mortality, the sum of intrinsic and extrinsic death rates, first displays a convex shape when the intrinsic mortality emerges to play a more important role; it then changes to a concave shape because of the selective survival properties of intrinsic mortality. Since the shape of extrinsic mortality is assumed to be invariant in this model, the shape of total mortality is primarily determined by intrinsic mortality. Correspondingly, LARs have bell shapes at middle and old ages for all populations. However, the starting and ending ages of the LAR bell shapes depend on how intrinsic mortality progresses with age, and the degree of the curvature depends on the proportion of overall mortality that is intrinsic mortality. Therefore, the sex and period differences in the intrinsic process are keys to explaining the different patterns of mortality increase between the two sexes and across periods. We illustrate these properties in the next few paragraphs.

For the U.S. period mortality data, sex differences in the mortality-increase pattern primarily lie in the year 1970 (Fig. 4). Consistent with previous findings (Fig. 1), females exhibit an increase in LAR between ages 50 and 70, whereas males in the same age span show a decrease in LAR following an early increase. The distinct patterns for males and females result from their different progressions of intrinsic mortality: for males, intrinsic death appears much earlier (at about age 40), and the leveling off of the rate also occurs earlier (about age 60), corresponding to a higher degree of population heterogeneity. The early exposure to intrinsic death for some males in this period was possibly due to unhealthy lifestyles and habits, such as smoking (Preston and Wang 2006; Yang 2008), that induced chronic physiological damage and began an earlier internal process of wear and tear. The large population variation in intrinsic mortality can also be attributed to the tobacco-use epidemic that greatly differentiates individuals in the male population in the 1970s (Diderichsen

1990; Diderichsen and Hallqvist 1997; Vagero and Lundberg 1993). In essence, a large proportion of males were smokers, while the remaining nonsmokers lived healthier lifestyles. A considerable proportion of males died from intrinsic causes early, and the surviving males were a robust group of nonsmokers, thereby ensuring the early achievement of mortality plateaus for male populations. Correspondingly, the faster intrinsic deterioration also puts the male population at a higher risk of extrinsic mortality. This associated high extrinsic mortality rate further clouds the total mortality pattern, such that the resulting percentage increase in total mortality is not as apparent for males as it is for females in the same period. In contrast, females in 1970 had a later, more-concentrated onset of intrinsic mortality and relatively lower associated extrinsic mortality; thus, the old-age mortality acceleration (i.e., the increase in LAR) for females was quite evident between ages 50 and 70.

The period change in U.S. mortality from 1970 to 2005 reflects a simultaneous reduction of extrinsic mortality and delay of intrinsic mortality to older ages for both males and females (Fig. 4). These changes are likely the result of improvements in medical technology and living standards over the decades. Additionally, intrinsic mortality for males demonstrates a more progressive compression. Such a concentration of intrinsic deaths to older ages is due to diminishing intrinsic heterogeneity, in part as a result of the decline of tobacco use among males over the decades. For males in recent periods, as the overall mortality pattern becomes more sensitive to variations in intrinsic mortality, the older-age mortality acceleration pattern is more visible. Females do not show further mortality compression, but the mode of the LAR shifts to older ages, corresponding to the continued delays of the onset of intrinsic mortality to later ages (Fig. 4).

The pattern of mortality increase in cohort data can also be explained by the two-mortality model, as illustrated by comparisons of Swedish cohorts (Fig. 5). In the 1885 cohort, both males and females exhibit similar intrinsic mortality trajectories (Fig. 5, upper panels) and thus similar age patterns in LARs (Fig. 5, middle panels). The sex differences appear only in the more-recent 1905 cohort. Similar to the findings for period changes, intrinsic death for females is delayed by about a decade between the 1885 and 1905 cohorts. Correspondingly, the onset of the increase in LAR shifts to the right over the decades examined. However, because the male cohort in 1905 shows a larger population variation and earlier manifestation of intrinsic death than the male cohort in 1885, the modes of the LARs appear at younger ages in the twentieth-century cohort than in the nineteenth-century cohort. The mechanism underlying the shift in intrinsic mortality between earlier and later cohorts is unknown but could possibly involve an increase in smoking rates in early twentieth-century cohorts. Despite the fact that, for males, the cohort change of mortality-increase pattern is in the opposite direction of the period change, the underlying mechanism for the change is essentially the same: it is the progression of intrinsic mortality that drives the pattern.

The plots of the ratio of intrinsic to total mortality are good summaries of the age-dependent change of the mortality dynamics (Figs. 4 and 5, bottom panels). Generally, the proportion of intrinsic mortality increases from 0 after age 40, when mortality acceleration begins, to 0.3~0.7 at very old ages, when a plateau is achieved. The two populations with younger-age mortality acceleration and earlier deceleration—that is, U.S. males in period 1970 and the Swedish males in cohort

1905—both demonstrate an early increase and an early arrival of the peak in the proportion. Essentially, a higher peak value of the intrinsic proportion is associated with a more evident mortality acceleration pattern.

In conclusion, the two-mortality model provides answers to the questions we raised. First, the mortality acceleration in late-middle and early-old ages results from the superposition of intrinsic mortality above age-increasing extrinsic mortality. Mortality acceleration is more evident in populations with a higher proportion of total mortality that is intrinsic in later ages. Second, in both period and cohort data, variations in the patterns of the LAR by sex are primarily due to the sex differences in the progression of intrinsic mortality with age. Females exhibit more-regular LAR patterns because the increase in intrinsic mortality with age is similar for all years in both period and cohort data. Males exhibit more-complex LAR patterns because of the significant change in their pattern of intrinsic mortality across years. For some male populations, the onset of mortality acceleration occurs early because of elevated intrinsic death rates for younger individuals. The deceleration in the male mortality rate (declining LAR starting about age 50) for these populations corresponds to a left shift in the mortality plateau, which can be attributed to a greater degree of heterogeneity within the population resulting from the removal of lower-vitality individuals prior to old age. The convergence of the male and female LAR patterns in the recent period data is mainly driven by the convergence of their death processes, particularly the intrinsic process.

Discussion

Since the discovery of the Gompertz law, population researchers have been highly interested in exploring the processes of aging and death and determining why they occur along predictable paths. This continuing interest has led to the emergence and refinement of the field of biodemography, which aims to "use biological arguments to investigate demographic phenomena" (Olshansky and Carnes 1997:11). The search for understanding involves developing biological theories to explain the patterns, which in turn provide explicit criteria for testing the theories. As a prominent example of the evolution of this relatively young field of population research, an intense debate on the determinants of the age pattern of mortality has stimulated the development of multiple biodemographic theories in the literature (Carnes et al. 1996; Olshansky and Carnes 1997). Although the Gompertz law remains the most parsimonious and influential approximation of mortality patterns, systematic deviations from the predicted exponential mortality increase in empirical data can play an essential role in refining the law by highlighting the complexity of the death process and enriching the underlying biological theories of mortality and aging (Gavrilov and Gavrilova 2001; Mueller and Rose 1996; Vaupel et al. 1979). The late-middle and early-old age mortality acceleration and the sex differences therein discussed in this work are good examples of deviations that deserve further exploration: they reveal important new insights into the interplay of aging and mortality dynamics that are beyond current applications, interpretations, and understandings of the Gompertz law.

Change-of-Aging-Rate Versus Two-Mortality Hypothesis

The major contribution of our work is in setting a new framework for understanding the mortality acceleration pattern in late-middle and early-old age. Although the dynamics behind this phenomenon have been the subject of much scholarly work (Horiuchi 1997, 2003; Horiuchi et al. 2003; Milne 2007), the focus has been primarily on the basic rate of individual aging. A common assumption in this body of research is that the interplay between aging and mortality is uniform, such that changes in the mortality pattern are directly associated with changes in the aging rate. Although various explanations along this line, such as changes of system repair capacity (Horiuchi 2003), cannot be entirely discounted, the lack of rigorous mathematical frameworks and tangible biological measures largely limit the applications of these change-of-aging-rate hypotheses.

In this article, we take a different approach in which mortality depends on two independent but interacting mortality processes: one mortality process is intrinsic and therefore tightly coupled to aging, and the other process is extrinsic to the individual but interacts with the intrinsic process. This two-process mortality hypothesis provides a biologically based framework that is not constrained by a rigid coupling of aging and mortality. Drawing on the idea of the interplay between intrinsic and extrinsic processes characterized by vitality, mortality acceleration becomes a phenomenon associated with changes in the strengths of two mortality rates based on the two processes with age. The fundamental implication of the hypothesis is supported by cause-of-death (COD) data: death from acute and infectious diseases dominates in the middle-age interval, whereas death from degenerative diseases is more common in the old-age interval (Carnes et al. 2006; Gessert et al. 2002, 2003; Horiuchi et al. 2003). The two-mortality model provides a mathematically tractable framework to quantify the interplay of intrinsic and extrinsic processes. In addition, we show in this article that the model can explain quantitatively the major variations in the observed patterns and hence is a meaningful tool for biodemographic studies.

It is worth noting that the absorbing boundary for the intrinsic process does not necessarily lead to an upper limit for human longevity. Rather, the dynamics of intrinsic mortality are flexible and can be constantly modified by exogenous factors, including medical, social, and behavioral factors. In fact, the right shift of the mode of LARs—that is, the older-age onset of mortality acceleration (e.g., U.S. females in Figs. 4 and 5)—reflecting the delay of intrinsic death at the population level, is a manifestation of advances in intrinsic processes achieved by recent populations. Such cumulative improvements can be attributed to better nutrition, healthier daily behaviors, and reduced inflammatory infections among later populations—all of which contribute to the slowing of the vitality depreciation rate (Fogel 2004). Although this article is not intended to address the controversy concerning the limits of human longevity, our findings suggest that the basic biological mortality schedule may be elastic to complex changes through time. A focus on the dynamics of this elasticity, we believe, is a key to understanding highly regulated, yet ever-changing mortality patterns in aging populations.

The article focuses on mortality patterns of only industrialized countries in the recent half-century because of data availability and quality constraints. However, the two-mortality model can also be applied to nonindustrialized countries and to other

periods. In fact, the model demonstrates good fit to recent Chilean and early Swedish mortality data (see the appendix). We hypothesize that the two-mortality dynamics will hold even when the mortality acceleration pattern at or after late-middle age is less evident. In this case, a high proportion of extrinsic mortality and a flatly distributed intrinsic mortality in the age span can mask the pattern.

Sex Differences in Mortality

The two-mortality model also provides a new insight into the sex differences in mortality patterns. Although the biological and behavioral differences between males and females may not be the fundamental reason for the *mortality acceleration* in late-middle and early-old age, they play a major role in explaining the sex differences in *mortality levels* (Yang and Kozloski 2012; Yang et al. 2012). The higher extrinsic mortality among males is likely associated with a less-robust physiological ability to resist stress, more-reckless behaviors, and harsher working conditions (Crimmins and Finch 2006; Nathanson 1984; Owens 2002; Waldron 1983), all of which may increase males' exposures and/or vulnerabilities to external challenges. Furthermore, less-healthy behaviors (e.g. drinking and smoking) and less benefit from the protective effects of sex hormones in males relative to females contribute to both higher extrinsic mortality and more progressive decline in intrinsic health (vitality) and consequently earlier intrinsic deaths in males. In addition, more-diverse health behaviors can contribute to greater heterogeneity in mortality in male populations.

It is interesting that although sex differences in the extrinsic mortality rates are relatively persistent, male-female differences in intrinsic mortality rates have largely diminished in recent periods (Fig. 4). Specifically, from 1970 to 2005, the male-to-female ratio of age-adjusted extrinsic mortality was reduced by only 5 % (from 1.54 to 1.46), whereas the ratio of intrinsic mortality dropped by almost 50 % (from 2.38 to 1.29). The reduction in the intrinsic mortality difference possibly can be attributed to the significant cessation of smoking among males (Preston and Wang 2006; Wang and Preston 2009) and an associated decline in the population-level heterogeneity of intrinsic mortality, which is expressed by the variance in the vitality loss rate (Li and Anderson 2013).

The intrinsic-extrinsic framework also provides a unique way to explore possible future sex gaps in mortality. Because the smoking patterns of males and females keep converging (Preston and Wang 2006), the intrinsic sex (male-to-female) mortality ratio is expected to drop further. Moreover, because intrinsic mortality reflects physiological degeneration processes that are common to both sexes, the sex gap in intrinsic mortality may eventually vanish. However, because the higher extrinsic mortality rate of males is associated with their physiological vulnerability to external challenges (e.g., infectious diseases) and innate risky behavior (Owens 2002), the sex difference in extrinsic mortality is likely to persist. This line of reasoning suggests that the male and female mortality trajectories in old age, where intrinsic mortality plays an important role, may continue to converge, whereas the mortality ratio in late-middle and early-old ages, where extrinsic mortality dominates, will likely persist.

Mortality Partition

Since the two-mortality structure is critical to the new model proposed in this article, the quantification approach that separates the two parts warrants clarifications. Although the development of the two-mortality hypothesis drawing from previous cause-of-death data analyses show that the causes of death could be different between middle and very old ages (Carnes et al. 2006; Horiuchi et al. 2003), our analysis used data exclusively on overall mortality rates and relied on a simplified mathematical partition of aging-related mortality.

The derivation of the two mortality types is based on a methodology that employs two stochastic processes. Although the two mortality types are mutually exclusive—that is, each death can be classified into only one of the two categories—the development of a single type of death can simultaneously involve both of the mortality processes. On one hand, the intrinsic deterioration process increases an individual's risk of extrinsic mortality; on the other hand, nonlethal extrinsic challenges that do not result in immediate deaths may still expedite the intrinsic degeneration rate, leading to a faster approach to the life limit. Although we do not explicitly measure the latter processes and model them only in terms of an increased rate of vitality loss in the intrinsic process, we do acknowledge the complexity of these processes and the ambiguity of distinguishing deaths from one cause to another. Our classification of mortality rates into intrinsic and extrinsic ones is rather a mathematical approximation. We distinguish these two mortalities according to whether acute forces or stimulus are involved at the final stage of death development. Many chronic diseases (e.g., the cardiovascular diseases) often result from long-term intrinsic damage accumulation, such that the intrinsic process plays a big role in disease development. However, deaths from these diseases are not necessarily classified as intrinsic mortality in the model. For example, although the long-term dysregulation of metabolic functions can put individuals at a much higher risk of myocardial infarction (MI) (Wilson et al. 1998), the death from MI may be actually triggered by intense physical and psychological exertions (Ueyama et al. 2008) or by an acute infection, such as pneumonia (Saikku et al. 1992). Therefore, death in this case may be categorized as extrinsic.

The conceptual underpinning of our mortality partition approach, which emphasizes the final moments of death, differentiates itself from that of previous studies. For example, the partition established by Carnes et al. (1996, 2006) utilized cause-of-death information, and the criterion is whether the primary cause of death originates from within the organism. This approach assumes that the initial development of a disease as the cause of death is more critical in classifying death and produces different patterns of age-specific intrinsic mortality rates. Specifically, compared with the findings based on the cause-of-death categorization by Carnes et al. (2006), intrinsic mortality in our study emerges much later—at ages 40–60 (vs. before age 15)—and surpasses or becomes comparable to extrinsic mortality at a relatively old age (vs. intrinsic

and extrinsic mortality rate crossover at middle age) (Figs. 4 and 5). These results should not be interpreted as being contradictory; they merely reflect different conceptualizations of the underlying cause-of-death processes and analytic approaches to extracting the corresponding information.

The mathematically derived mortality partition, although crude and perhaps idealized, has some advantages in empirical analyses. First, it requires much less information than that from cause-of-death data, which are often not available. Second, it avoids the inherent uncertainties associated with cause-of-death data, including the complexity of the etiology for some diseases, the opaqueness of the aging-disease interaction, and the imprecision in recording the actual cause of death, especially for older adults who possibly died from a combination of chronic conditions as well as environmental hazards (Gessert et al. 2003).

Finally, partitioning mortality into intrinsic and extrinsic components would not only enrich our understanding of mortality patterns but also help identify the effects of risk factors on each of the two death processes. Some recent studies have shown the promise of this approach by examining how the genetic variants would affect mortality rate and lifespan. Yashin et al. (2012), for example, employed the Strehler and Mildvan (SM) theory to explain the shape of mortality trajectories of population subgroups with different numbers of longevity alleles. In this pioneering work, they hypothesized that each longevity allele contributed to an increase in robustness and resistance to stress and thus the number of longevity alleles affected mortality risk through its influence on the initial survival capacity (i.e., vitality) of an individual. Because the original SM theory does not allow a direct assessment of the intrinsic components, this hypothesis has yet to be tested with a better analytic framework that allows for such assessment. In this sense, the two-mortality model is an extension to and improvement of the SM model because it is able to distinguish two different mortality processes and enables an empirical test of the genetic effects on the intrinsic components given additional genetic data.

Limitations

The two-mortality model summarizes many mechanisms into two simplified processes. Clearly, it is inadequate to explain the multitude of time-varying processes that ultimately determine the moment of death. The mortality categorization is particularly blurred at old age, when the absorption of an individual's remaining vitality into the death boundary and a challenge to his or her remaining vitality are essentially the same event in time.

Furthermore, this new model can be considered as a first crude approximation to a more general model of heterogeneous death processes. It certainly bears resemblance to frailty models (Hougaard 1995; Vaupel et al. 1979) by recognizing the heterogeneity in mortality, but the conceptual origin and focus of the models are quite different. The current model is more driven by biology

and intends to inform biological theories of aging and mortality, whereas frailty models are more statistically driven. One the other hand, the idea of further considering individual frailty in terms of individuals' different life experiences that characterize the model parameters could fit well in the framework. In the current model form, individual heterogeneity is represented only in the intrinsic stochastic diffusion process. A flexible structure that accounts for more sources of heterogeneity, such as allowing distributions for model parameters and age variations of the processes, would be worth exploring in the future.

Even with these limitations, we believe that the two-mortality model offers a valuable new analytic framework to reveal the complex and ever-evolving patterns of mortality in aging populations with widely available general mortality data.

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Appendix: Two-Mortality Model

The two-mortality model (Li and Anderson 2013) represents total mortality as the sum of two death sources that result from different processes. For additional details see Li (2011).

Intrinsic Mortality

The intrinsic process summarizes varied mechanisms by a single quantity called "vitality" to denote the remaining survival capacity of an organism. Each individual begins with an initial vitality, v_0, which stochastically declines with age. Intrinsic death occurs when the organism's vitality reaches zero (Fig. 3). The random trajectory of vitality, v_x, between v_0 and 0 is described by the following stochastic diffusion process with a negative drift r and a variance term s^2:

$$v_x = v_{x-1} - r + sW_x \qquad x = 1, 2, 3... \tag{A1}$$

where W_x is a standard Wiener process (Aalen and Gjessing 2001; Anderson 1992, 2000; Anderson et al. 2008; Li and Anderson 2009; Weitz and Fraser 2001). This process is characterized by three parameters (v_0, r, and s), but the distribution of the first arrival time to zero boundary, which regulates the mortality function, is determined by only two dimensions (Aalen and Gjessing 2001). Therefore, we need to normalize the process by one of the three

parameters. The choice of the denominator does not influence the mortality outcomes; it influences only the interpretations of the parameters after normalization. In the case where we standardize Eq. (A1) by the initial vitality v_0 (divide both sides of Eq. (A1) by v_0), the normalized parameter r and s separately represent the fraction of vitality loss and the fraction of vitality spread per unit of time. Under this condition, each standardized vitality trajectory seems to start from a single point $v_0 = 1$, but the actual differences in the initial values are reflected in both r and s. In fact, when the process is applied to a population where a single r is derived as the mean fraction of vitality loss per unit of time, s demonstrates the average combined variation from both inherent (initial) and acquired (evolving) sources per unit of time.

Another way to normalize the process is to use the s term as the denominator (Aalen and Gjessing 2001). While the evolving variation is fixed, the intrinsic death rate is determined by the initial vitality v_0 and the vitality loss rate r. This normalization can be applied when the initial vitality values are considered to be very different among populations; such differences are the focus of this study. Because all normalizations yield equivalent results, we will stick to the former approach here for easier interpretations.

The first-passage time of vitality to the zero boundary derived from Eq. (A1) is the inverse Gaussian function (Cox and Miller 1965):

$$f(x) = \frac{x^{-3/2}}{s\sqrt{2\pi}} \exp\left(-\frac{(1-rx)^2}{2s^2x}\right). \tag{A2}$$

By definition, the fraction of the total population that has not died from intrinsic causes at age x is equivalent to the probability that the individual's vitality has not reached zero by x. The survival pattern resulting from the intrinsic process can be expressed as

$$l(x) = 1 - \int_0^x f(x)dx = \Phi\left(\frac{1-rx}{s\sqrt{x}}\right) - \exp\left(\frac{2r}{s^2}\right)\Phi\left(-\frac{1+rx}{s\sqrt{x}}\right), \tag{A3}$$

and the intrinsic mortality rate is

$$\mu_i(x) = f(x)/l(x). \tag{A4}$$

Extrinsic Mortality

To model the extrinsic mortality, let Y_x with $x \geq 0$ be a random point process, with rate λ representing the occurrence of instantaneous extrinsic challenges, such as a natural disaster or infection. The parameter λ specifies the frequency of challenges. Each extrinsic event has a magnitude Z_x, where the cumulative distribution function $\varphi(z)$ denotes the intensity of the challenge. We assume that only when the challenge magnitude Z_x exceeds the current vitality level v_x does the extrinsic challenge result in death; that is, death occurs when

$\Pr(Z_x > v_x)$. Challenges not exceeding the current vitality level may also alter the vitality trajectory and, in effect, nonlethal challenges are subsumed into r and contribute to the lifetime-averaged rate of vitality change. This assumption couples the risk of death from external forces to the intrinsic age-dependent vitality level of the individual and insures that the effect of the extrinsic challenge changes with age. Assuming that Y_x is a history-independent Poisson process (Finkelstein 2007), the extrinsic mortality rate for each individual is

$$m_e(x) = \lambda \Pr\left(Z_x \geq v_x\right) = \lambda\left(1-\varphi\left(v_x\right)\right). \tag{A5}$$

If we further assume that the magnitude of the event is exponentially distributed such that most external events are small and the probability of large events declines relative to their magnitude (Strehler and Mildvan 1960), then the cumulative distribution function is $\varphi(z)=1-e^{-z/\beta}$, where β is a scale parameter. Now the conditional extrinsic mortality rate for an individual given the realization of the vitality trajectory v_x becomes

$$m_e\left(x \mid v_x\right) = \lambda e^{-v_x/\beta}, \tag{A6}$$

where β characterizes the environmental deleteriousness relative to the initial vitality of the organism. In essence, a larger β implies that high magnitude challenges occur more frequently.

The aggregated extrinsic mortality rate at a population level is

$$\mu_e(x) = \int_0^\infty m_e\left(x \mid v_x\right) g_x(v) dv_x = \int_0^\infty \lambda e^{-v_x/\beta} g_x(v) dv_x, \tag{A7}$$

where $g_x(v)$ is the normalized vitality distribution at age x, conditioning on (1) that the original vitality trajectory follows the intrinsic diffusion process as defined in Eq. (A1) and (2) that the extrinsic process constantly modifies the distribution by removing individuals who die from challenges. Note that $g_x(v)$ has no analytical solution because challenges preferentially eliminate low-vitality individuals and thus modify the distribution derived from the original intrinsic process. As another consequence of such a modification, the actual intrinsic mortality rate also deviates from Eq. (A4).

The lack of an analytical solution would certainly limit the application of the model. One conventional way to proceed is to modify the model assumptions so that they yield a closed formula. Our approach is to assume that extrinsic mortality depends on the mean vitality $\overline{v}(x)$ instead of the random variable v_x. That is, we replace v_x with the mean vitality $\overline{v}(x)$ in Eq. (A6), giving

$$\widetilde{m}_e\left(x \mid \overline{v}(x)\right) = \lambda(x) e^{-\overline{v}(x)/\beta}. \tag{A8}$$

To a first order, the expected value of vitality according to Eq. (A1) with an absorbing zero boundary evolves linearly with age until very old age (Anderson et al. 2008), giving

$$\overline{v}(x) \approx v_0 - rx = 1 - rx\,, \tag{A9}$$

where v_0 is the initial vitality and is equal to 1 in the normalized case. The conditional extrinsic mortality in Eq. (A7) then becomes independent of the distribution of vitality, and $g_x(v)$ integrates to 1. The extrinsic mortality rate is now approximated as

$$\mu_e(x) \approx \lambda e^{-(1-rx)/\beta}\,, \tag{A10}$$

which simply expresses the average external killing rate as an exponential function of the mean vitality loss trajectory defined by the two environmental parameters λ and β. Equation (A10) implies that at each age, all individuals are subjected to the same average extrinsic killing force; hence, the presence of extrinsic mortality does not change the vitality distribution resulting from the original intrinsic diffusion process. Extrinsic mortality alone in this approximated form resembles the Gompertz mortality function, which is in line with the Strehler and Mildvan theory (1960).

In essence, the intrinsic and extrinsic mortality rates are decoupled given that the extrinsic mortality force is assumed to be equal across the population at each age. Combining Eqs. (A4) and (A10), the total mortality rate can be represented as

$$\mu(x) = \mu_i(x) + \mu_e(x) = \frac{x^{-3/2} e^{-(1-rx)^2/2s^2 x}}{s\sqrt{2\pi}\left(\Phi\left(\frac{1-rx}{s\sqrt{x}}\right) - e^{2r/s^2}\,\Phi\left(-\frac{1+rx}{s\sqrt{x}}\right)\right)} + \lambda e^{-(1-rx)/\beta}. \tag{A11}$$

Simulation analyses show that this decoupled model (A11) slightly overestimates extrinsic mortality and consequently underestimates the intrinsic rate at very old ages. However, it does not affect the separation between the two mortality rates in earlier old age.

Model Fitting

The model fitting is cast as a maximum likelihood optimization, as developed by Salinger et al. (2003) to deal with interval-censored mortality data, in which mortalities are counted at the end of each time period rather than continuously. The likelihood function is constructed from the multinomial distribution based on the proportion of deaths in each time period:

$$\text{LogLik} = \sum_x \left(d_x \ln q_x + (n_x - d_x)\ln(1-q_x)\right), \tag{A12}$$

where d_x is the number of deaths at age x, n_x is the number of population at the beginning of age x, and q_x is the probability of death at age x. The

probability of death is derived from Eq. (A11). The algorithm estimates standard errors through the estimated variance matrix. Specifically, standard errors are obtained by taking the square root of the diagonal elements in the inverse of the Hessian of the negative log-likelihood, evaluated at the parameter estimates (Kendall et al. 1977). The fitting algorithm was published in R at the CRAN website (Passolt et al. 2013). Upon obtaining the four estimated parameters, we can generate the intrinsic, extrinsic, and total mortality trajectories based on Eq. (A11).

Model Fit to Mortality Data from Nonindustrialized Societies

In order to show that the two-mortality framework can be applied to nonindustrialized societies or developing countries, we illustrated the model fit to Swedish data in period year 1820 and Chilean data in period year 2005 in Fig. 6. The one-year interval death rate data are obtained from the HMD for these populations. A mortality acceleration pattern in late-middle and early-old age as well as a bell shape in the LAR is quite evident for all these data. A good fit from the model to both early Swedish and recent Chilean data further supports our hypothesis that the two-mortality dynamics could generally hold across populations.

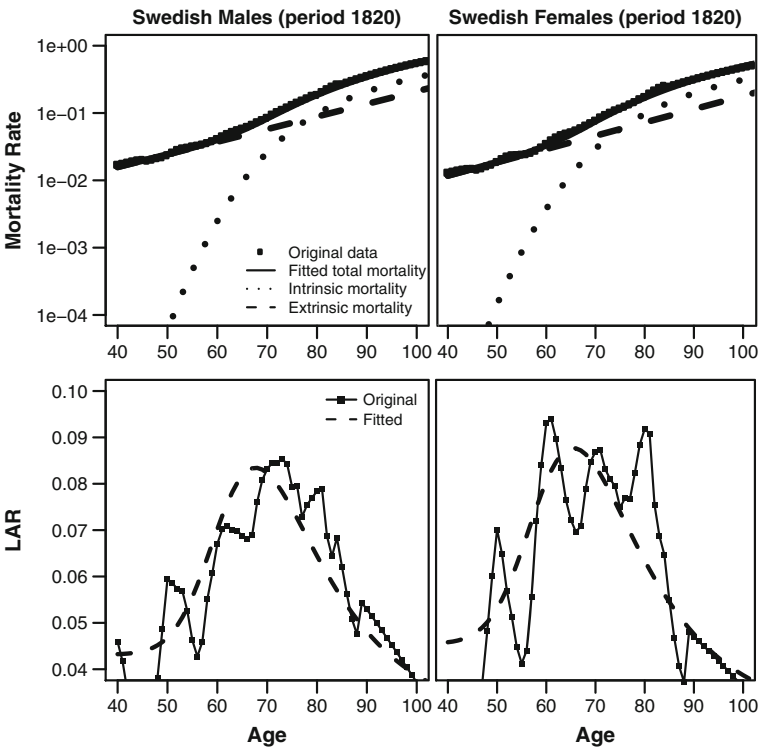

Fig. 6 The two-mortality-process model fit to the Swedish early period (1820) and Chilean current period (2005) mortality data and the corresponding fitted LARs

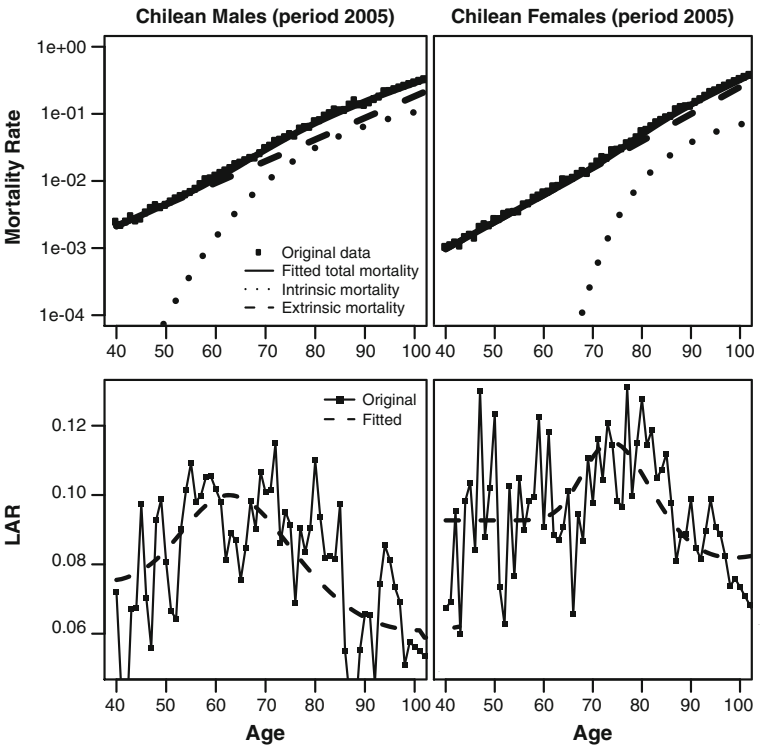

Fig. 6 (continued)

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