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Aging in the Context of Cohort Evolution and Mortality Selection

Hui Zheng

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Abstract This study examines historical patterns of aging through the perspectives of cohort evolution and mortality selection, where the former emphasizes the correlation across cohorts in the age dependence of mortality rates, and the latter emphasizes cohort change in the acceleration of mortality over the life course. In the analysis of historical cohort mortality data, I find support for both perspectives. The rate of demographic aging, or the rate at which mortality accelerates past age 70, is not fixed across cohorts; rather, it is affected by the extent of mortality selection at young and late ages. This causes later cohorts to have higher rates of demographic aging than earlier cohorts. The rate of biological aging, approximating the rate of the senescence process, significantly declined between the mid- and late-nineteenth century birth cohorts and stabilized afterward. Unlike the rate of demographic aging, the rate of biological aging is not affected by mortality selection earlier in the life course but rather by cross-cohort changes in young-age mortality, which cause lower rates of biological aging in old age among later cohorts. These findings enrich theories of cohort evolution and have implications for the study of limits on the human lifespan and evolution of aging.

Keywords Rate of demographic aging · Rate of biological aging · Cohort evolution · Mortality selection · Strehler and Mildvan model

Introduction

In the past two centuries, life expectancy has more than doubled from 30–40 years to approximately 80 or more years in many developed countries (Oeppen and Vaupel 2002). The epidemiologic transition (Olshansky and Ault 1986; Omran 1971) is thought to be the key mechanism behind the increase in human life expectancy. Epidemiologic transition theory portrays four stages through which advanced societies pass, starting with the age of pestilence and infectious diseases that characterizes most

H. Zheng (✉)
Department of Sociology, The Ohio State University, 107 Townsend Hall, 1885 Neil Avenue Mall, Columbus, OH 43210, USA
e-mail: zheng.64@sociology.osu.edu

of the human history, entering the age of receding pandemics around the middle of the nineteenth century, and advancing to the age of degenerative and human-made diseases (e.g., cardiovascular disease) in the early twentieth century, and to the age of delayed degenerative diseases around the 1960s. In the early stages of the epidemiologic transition, mortality at young ages declines because of better sanitation and living standards; in later stages, the elderly experience a substantial mortality decline following improvements in medical technology. The epidemiologic transition theory attributes mortality decline to a changing mix of socioeconomic development, lifestyle changes, and medical innovations in each period.

By contrast, other theories emphasize change over cohorts rather than across periods. Working from life course and cohort replacement perspectives, these theories attribute old-age mortality decline in later stages of the epidemiologic transition to mortality decline at younger ages for each cohort. One such theory describes the "cohort morbidity phenotype," proposing that cohorts experiencing lower exposure to infection and inflammation during early childhood reap a lower mortality risk later in their lives (Finch and Crimmins 2004). Another theory, describing a trend of "technophysio evolution," argues that later cohorts are endowed with better health capital at birth and thus enjoy lower rates of health capital depreciation over the life course because of increasing control over the environment, improved food and energy production, other technological innovation, and economic growth (Fogel and Costa 1997). Conversely, detrimental conditions in early life would jeopardize survival in later life—a relationship that has been framed as "the physiological scarring effect" (Preston et al. 1998) or the "critical period" in epidemiology (Ben-Shlomo and Kuh 2002). Herein, I collectively refer to these theories as the "cohort evolution perspective," although I am mainly concerned with the theory of cohort morbidity phenotype and the theory of technophysio evolution, which explicitly attribute historical declines in mortality to improvements in morbidity phenotypes or health capital endowment across cohorts.

Although cohort evolution theories illuminate how cohort change can lead to mortality declines, two questions remain. First, are cohort evolution theories correct? Although theories of cohort morbidity phenotype and technophysio evolution have been supported by much evidence, neither theory takes into account possible changes in patterns of mortality selection, which is another mechanism linking early-life circumstances to health and mortality in later life (Preston et al. 1998). The theory of heterogeneity conceptualizes populations as composed of individuals or subpopulations with different physiological vulnerability to mortality, referred to as "frailty" (Vaupel et al. 1979). Later cohorts experience lower risks of infection and inflammation and have better nutrition and health capital during childhood, according to cohort evolution theories, so a smaller proportion of frail individuals are selected out of the population during young age. This, in turn, would cause a larger proportion of frail individuals to survive into old age and would increase the cohort's overall mortality risk at older ages. In this case, old-age mortality risk may be potentially higher for later cohorts than earlier cohorts. Such differences in selective survival have been used to explain the crossover in mortality rates between white and black Americans: despite lower mortality rates at younger ages, whites' mortality risk exceeds that of blacks at very old ages (e.g., Manton and Stallard 1981). If selection of frail individuals out of the population at younger ages has indeed declined across cohorts, cohort evolution

theories may not explain historical declines in old-age mortality risk in advanced societies after the third stage of the epidemiologic transition. Therefore, cohort evolution theories should be supplemented with a comprehensive investigation of the changing pattern of mortality selection across cohorts.

Second, cohort evolution theories predict that later cohorts enjoy better health in old age. Does that mean that aging slows down in later cohorts? We may conceptualize two indicators of aging: the rate of demographic aging and the rate of biological aging. The rate of demographic aging refers to the slope of the mortality curve (Gompertz slope)—the extent of acceleration in the mortality rate across ages. Gompertz's (1825) classical law of mortality models the increase in mortality rates over adulthood in an exponential pattern: $R_t = R_0 e^{\alpha t}$, where R_t is the mortality rate at age t, R_0 is the initial mortality rate, and α refers to the rate of increase in the mortality rate, alternately described as "mortality acceleration" or the rate of demographic aging. The mortality acceleration parameter α is affected by both the variance of the frailty distribution in the population (Vaupel 2010a; Yashin et al. 2002b) and by the initial mortality rate (Strehler and Mildvan 1960). Therefore, to understand the change in demographic aging (mortality acceleration) across cohorts, we must explore whether different cohorts experience different mortality selection processes.

The rate of biological aging is conceptually distinct from the rate of demographic aging; although sometimes the latter is used to approximate the former, it is misleading to use the two interchangeably (Yashin et al. 2002b). The rate of biological aging describes the decline of a living organism's "physiological and biological capacities with age, accompanied by an increase in the chances of death" (Yashin et al. 2002b:206). Changes in mortality patterns notwithstanding, has the rate of biological aging changed across cohorts? If the rate of biological aging has changed, we might ask how it is affected by cohort evolution and mortality selection processes; how the rates of demographic aging and biological aging are related; and whether the two rates converge or diverge in more recent cohorts. Following this reasoning, my study seeks to test whether cohort evolution and mortality selection theories adequately describe historical changes in mortality patterns, and to contrast trends in biological and demographic aging in seven advanced societies that have gone through the epidemiologic transition.

Cohort Evolution Theories

Theories of cohort evolution describe correlation across cohorts in the age dependence of mortality rates. I focus on two such theories: the theory of the cohort morbidity phenotype and the theory of technophysio evolution. Rather than emphasizing period trends in economic development or medical advancement, as the epidemiologic transition theory does, the cohort morbidity phenotype theory proposes that "the reduction in lifetime exposure to infectious diseases and other sources of inflammation—a cohort mechanism—has made an important contribution to the historical decline in old-age mortality" (Finch and Crimmins 2004:1736). Specifically, as subsequent cohorts experience a lower risk of inflammation during early childhood, this leads them to exhibit lower mortality rates later in life. Thus, cohorts with a mortality advantage over earlier cohorts at a young age maintain this advantage of lower mortality at any other stage in life.

The cohort mechanism described by the cohort morbidity phenotype theory links the old-age decline in mortality observed in the third and fourth stages of the epidemiologic

transition (the ages of degenerative and human-made diseases, and then of delayed degenerative diseases) to the young-age mortality declines experienced by cohorts born during the second stage of the transition (the age of receding pandemics). This link helps explain decreased mortality rates for the major degenerative diseases (e.g., heart disease, cancer, and stroke) in the 1960s despite the lack of significant medical breakthroughs during that period (Crimmins and Finch 2006). Much evidence supports the enduring effect of early-life inflammation over the life course (Finch and Crimmins 2004). For example, childhood streptococcal infections increase the risk of rheumatic heart disease in adulthood (Jones 1956); respiratory infections in early life are linked with late-life lung impairments (Bengtsson and Lindstrom 2003; Shaheen et al. 1994); and a reduction in lifetime exposure to infections and inflammation retards the atherosclerotic process (Crimmins and Finch 2006).

The link between health in early and later life is reflected in the theory of technophysio evolution (Fogel and Costa 1997). Unlike genetic evolution, which relies on natural selection, technophysio evolution proposes that technological change is synergistic with physiological improvements across cohorts, producing a form of human evolution that is biological but not genetic (Fogel 2005). This theory applies only to the past 300 years of human history—the span over which human technology has developed the potential to significantly improve health and longevity (Fogel and Costa 1997). According to this theory, technological innovation endows later cohorts with more health capital at birth, and leads them to experience lower rates of health capital depreciation as they age. Innovations that make this possible include humans' increasing control over the environment, improved food and energy production, and economic growth (Fogel 2004a; Fogel and Costa 1997).

Rather than considering inflammation to be the mechanism linking childhood mortality to old-age mortality, technophysio evolution theory emphasizes the role of nutrition—*in utero*, during infancy, and in early childhood. This is consistent with the influential work of Barker and colleagues, who identified maternal malnutrition (which retards fetal growth and causes permanent organ damage) as the major mechanism linking childhood conditions to adult morbidity (Barker 1992, 1994, 2004; Barker et al. 1991). As the result of technological change, economic growth, and increasing food supplies, humans' body size has increased by more than 50 %, and the robustness and capacity of vital organ systems has greatly improved over the past three centuries (Fogel and Costa 1997). Thanks to greater physiological capacity, human beings are able to work more efficiently, intensively, and longer, and further contribute to technological changes.

The cohort evolution theories emphasize different mechanisms linking early- and later-life mortality, but both suggest a positive correlation between the two. Furthermore, Finch and Crimmins (2004:1737) argued that, "the major declines in mortality have had little effect on the basic rate of mortality acceleration during aging, as shown for cohorts by parallel linear slopes of mortality on semi-logarithmic plots." In other words, in spite of the trend in overall mortality across cohorts, the mortality slope (Gompertz slope) appears to be relatively stable. From this, I derive the following two hypotheses:

> *Hypothesis 1a: According to cohort evolution theories, young-age and old-age mortality rates are positively correlated across cohorts.*

Hypothesis 1b: According to the cohort morbidity phenotype theory, the acceleration of mortality during aging (e.g., after age 70) should be constant across cohorts: that is, it is not affected by young-age mortality rate.

Population Heterogeneity, Mortality Selection, and the Strehler and Mildvan Theory

The theory of population heterogeneity proposes that populations are composed of individuals or subpopulations with different physiological vulnerability to mortality, or *frailty* (Vaupel et al. 1979). Individual frailty is assumed to be fixed at birth, and mortality tends to remove frailer individuals from the population at earlier ages. This contributes to the deceleration of mortality at late ages: the rate at which the mortality hazard changes with age levels off after age 110 (Thatcher et al. 1998; Vaupel 2010b; Vaupel et al. 1998). The deceleration of mortality in very late life might be due to the underregistration of deaths or age uncertainty among very old adults, and these problems might plague even the best historical data (Crimmins and Finch 2006; Gavrilov and Gavrilova 2011). Keeping this possible limitation in mind, I aim to study how population heterogeneity may shape cohort patterns in mortality acceleration before very late ages (e.g., age 95). Yashin and colleagues (2002b) and Vaupel (2010a) have suggested that mortality acceleration (i.e., the rate of demographic aging) is related to the variance of the distribution of frailty in the population. "The slope of the resulting mortality rate increases when the variance of heterogeneity distribution declines" (Yashin et al. 2002b:209) because when a smaller proportion of frail individuals are selected out of the population at an early age, the mortality curve in later ages becomes steeper. This theory implies that later cohorts having a smaller proportion of frail individuals in early life (i.e., smaller variance in the distribution of frailty)—as cohort evolution theories would predict—would have a more pronounced acceleration of mortality in late life than earlier cohorts, in which a larger proportion were frail.

That decreased cohort frailty would exacerbate the acceleration of mortality in later life is consistent with Strehler and Mildvan's (SM) (1960) general theory of mortality and aging. This theory provides a biological and physical explanation for Gompertz's (1825) law of exponential increases in adult mortality by linking internal reserves of vitality to external environmental stress. The theory posits that the initial mortality rate $\ln(R_0)$[1] and the slope α of the logarithm of the Gompertz mortality curve ($R_t = R_0 e^{\alpha t}$) (i.e., the rate of mortality acceleration, or the rate of demographic aging) are negatively correlated. This correlation is expressed as $\ln(R_0) = -1/B\alpha + \ln(K)$, where B is the fractional loss each year of original vitality, and K denotes the frequency of environmental variations. In other words, if a later cohort has a lower initial mortality rate than a preceding cohort, it should experience greater acceleration in mortality (i.e., α) over the life course. Although the initial mortality rate in the SM theory refers to the intercept of the logarithm of the Gompertz curve, the mortality selection mechanism

[1] The initial mortality rate is denoted by $\ln(R_0)$, with subscript 0 indicating that this is the intercept of the logarithm of the Gompertz mortality curve. The initial mortality rate should not be confused with the infant mortality rate, which is denoted by $\ln(R_{0-1})$ in this article.

expressed in the SM correlation would suggest a negative correlation between young-age mortality rate and the rate of mortality acceleration. Although the SM theory conforms to Gompertz's Law of Mortality—an exponential increase of mortality with age t over adulthood—the theory of population heterogeneity proposes that mortality decelerates at very late ages. Both theories, however, imply that mortality selection operates throughout the life course and leads to an inverse relationship between young-age mortality rates and the magnitude of mortality acceleration (until very late age) across cohorts. This leads to the following hypothesis:

> *Hypothesis 2a: According to the theory of heterogeneity and the SM model, young-age mortality rate and mortality acceleration (i.e., rate of demographic aging, up until very late age: e.g., age 95) are negatively correlated across cohorts.*

Although rate of demographic aging is affected by the extent of population heterogeneity (Vaupel 2010a; Yashin et al. 2002b) and the initial mortality rate (Strehler and Mildvan 1960), "all older humans share a similar, and perhaps essentially the same, rate of increase in mortality with age" (Vaupel 2010b:539). This implies that individuals' biological aging process is constant across cohorts. This insight is consistent with the SM theory's proposition that the rate of decline in the vitality index, denoted as B, is fixed. The SM theory assumes a linear decline of vitality index $V_t = V_0(1 - Bt)$ with increasing age, where vitality V_t is the capacity of an individual organism to stay alive at age t, and the coefficient B is the yearly decrement from the original vitality V_0. The coefficient $B = b + f(D)$ is a function of both a normal aging component, b, and the impact $f(D)$ of environmental factors as determined by a summary measure of relative environmental deleteriousness, D. Based on mortality data from 32 countries from the mid-1950s, Strehler and Mildvan (1960:16–17) concluded that B "appears to be nearly constant regardless of the environment . . . . It thus appears that B is dominated by (the normal aging process) b, or in other words that the rate of loss of vitality during the aging process is largely independent of the environment." This leads to the following hypothesis:

> *Hypothesis 2b: According to the theory of heterogeneity and the SM model, the rate of biological aging is fixed across cohorts.*

Relation Between Cohort Evolution and Mortality Selection Mechanisms

Mortality selection mechanisms (expressed in the theory of heterogeneity and SM model) are not necessarily in conflict with the cohort evolution perspectives (encompassing both the cohort morbidity phenotype and technophysio evolution theories). In fact, both processes may coexist. Cohort evolution theories suggest a positive correlation between young-age mortality *level* and old-age mortality *level* across cohorts, and mortality selection mechanism suggests a negative correlation between young-age mortality *level* and the *rate* of mortality acceleration (i.e., α from the equation $R_t = R_0 e^{\alpha t}$, or the slope of the log mortality curve) across cohorts. Although cohort evolution theories do not take mortality selection mechanisms into

account, they imply that any mortality selection process cannot undo the positive correlation between young- and old-age mortality rates. To test this aspect of cohort evolution theories, we need to consider whether mortality selection changes or reverses the correlation between young- and old-age mortality rates.

Figure 1 portrays four scenarios represented by log mortality curves for one hypothetical earlier cohort 1 and another hypothetical later cohort 2, where each scenario may support or dispute cohort evolution theories and mortality selection mechanisms. Here, $\ln(R_t)$ represents the logarithm transformation of the age-specific mortality rate at age t. Panel A supports both cohort evolution and mortality selection theories, given that cohort 2 has lower young- and old-age mortality rates than cohort 1 and the mortality acceleration (i.e., α, the slope of the log mortality curve) is stronger for cohort 2 than for cohort 1. In other words, the levels of young- and old-age mortality rates are positively correlated, and mortality acceleration is negatively correlated with young-age mortality rate across cohorts. Panels B and C support cohort evolution theories but dispute mortality selection mechanism: cohort 2 has lower young- and old-age mortality rates compared with cohort 1, but the slope of the mortality curve is not steeper. In this case, young- and old-age mortality levels are positively correlated, but mortality acceleration is either uncorrelated or positively correlated with the young-age mortality rate across cohorts. Panel D disputes cohort evolution theories but supports mortality selection mechanism: cohort 2 has higher old-age mortality rate compared with cohort 1, despite lower mortality rate at younger ages. In this case, mortality acceleration and young-age mortality rate are negatively correlated, and young- and old-age mortality levels are also negatively correlated across cohorts. Therefore, Panel A represents the only scenario in which both cohort evolution theories and mortality selection mechanism are supported.

One of the cohort evolution theories—the cohort morbidity phenotype theory—further suggests that mortality acceleration during old age (e.g., after age 70) should be constant regardless of the young-age mortality rate. It is not clear whether this theory refers to the rate of demographic aging or the rate of biological aging, given that some studies use the slope of the logarithm of the empirical mortality curve to approximate

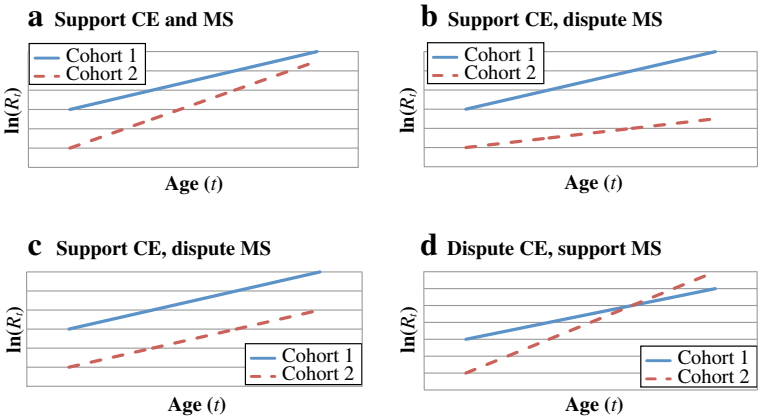

Fig. 1 An illustration of mortality selection mechanism (MS) and cohort evolution theory (CE). $\ln(R_t)$ represents logarithm transformation of age-specific mortality rate at age t

the rate of individual biological aging (Yashin et al. 2002b). If cohort morbidity phenotype theory refers to the rate of demographic aging, then it is in conflict with the mortality selection mechanism on this point because the latter implies that the rate of demographic aging is negatively correlated with the young-age mortality rate: that is, differential mortality selection processes lead to different patterns of demographic aging across cohorts. If the former theory refers to the rate of biological aging, then it coincides with population heterogeneity theory and SM's proposition that the rate of biological aging at the individual level is fixed. But is the rate of biological aging truly fixed? Studies claiming a fixed senescence process are based on period data collected no earlier than the mid-twentieth century (Strehler and Mildvan 1960; Vaupel 2010b). Does a fixed process of senescence (biological aging) adequately characterize cohorts born before the twentieth century, when infectious diseases and epidemics were still prevalent? Or, instead, does cohort evolution encompass evolution in the process of biological aging; and if so, is the latter affected by patterns of mortality selection? If we allow that the rate of biological aging may have varied in the past, we may further test whether this rate is related to the rate of demographic aging and, if so, whether the two diverge or converge across cohorts.

Data and Methods

This study investigates cohort trends in the rates of demographic and biological aging, testing the preceding hypotheses, using cohort age-specific mortality data from the Human Mortality Database. The data cover the following countries and cohorts: Sweden, 1751–1915; Netherlands, 1850–1914; Iceland, 1838–1915; France, 1816–1914; England, 1841–1912; Denmark, 1835–1914; and Norway, 1846–1914. The SM theory assumes that vitality declines linearly over the life course. In other words, the rate of biological decline (the slope of the decline in vitality) is constant over the life course. Some later studies have suggested that the decline in vitality is actually nonlinear (Arbeev et al. 2005). Rather than making the strong assumption of a linear decline in vitality, and also for the purpose of focusing on the aging process in old age, I confine the analysis to the decline in vitality after age 70.[2] Because of problems of death underregistration and age uncertainty at very old ages, even in the best historical data (Crimmins and Finch 2006; Gavrilov and Gavrilova 2011), I restrict the upper end of age in my analysis to 94.

Strehler and Mildvan (1960) outlined several methods to estimate the value of the biological aging coefficient B. I use the second method, which is the most straightforward for my purposes, and perform the following calculation for each country-cohort case.[3] First, I calculate the initial mortality rate $\ln(R_0)$ at age 70 and the rate of mortality acceleration from age 70 to age 94, α, using the equation $\ln(R_t) = \ln(R_0) + \alpha t$. This equation is the logarithm transformation of the equation $R_t = R_0 e^{\alpha t}$, where age-specific mortality rates R_t are available in the data. The terms $\ln(R_0)$ and α represent the

[2] I also considered ages 65 and 75 as the starting ages for calculating the rates of demographic and biological aging, but these changes to my method did not change the main findings.

[3] I did not use the other two methods outlined by Strehler and Mildvan because they either produced unrealistically low values of K or were not appropriate for heterogeneous human populations (Strehler and Mildvan 1960).

intercept and slope of the log mortality curve, respectively. Then, I calculate the coefficient B from age 70 to age 94, using the equation $\ln(R_0) = -1/B\alpha + \ln(K)$. I assign a value of K (K = 1) as suggested by Strehler and Mildvan (1960). Following this procedure, I obtain two estimated parameters for each country-cohort case: α, the slope of the log mortality curve from age 70 to age 94 (i.e., the parameter of mortality acceleration), and the coefficient B of vitality attrition from age 70 to age 94. The parameter α represents the rate of demographic aging, and the parameter B represents the rate of biological aging. I also calculate the rates of mortality acceleration from ages 40, 45, 50, 55, and 60 to 94 for the purpose of testing mortality selection hypotheses. The data used for the analysis are country-cohort panel data comprising 628 country-cohort cases. Each country-cohort case includes measures of age-specific mortality rates from age 0–1 to age 90–94; rates of mortality acceleration from age 40, 45, 50, 55, and 60 to age 94; and the values of the parameters α and B.

I conduct separate analyses of the data from each country. I also analyze the pooled sample of all countries by using country fixed-effects models to eliminate unobserved heterogeneity among countries (Wooldridge 2002). Although random-effects models are generally more statistically efficient than fixed-effects models, the Hausman test suggests that unobserved time-constant unit effects are correlated with explanatory variables in my data, thus warranting the use of fixed-effects models. Because of space constraints, this article presents regression findings from the pooled sample of countries and graphical illustrations of key results using data from select individual countries.

Results

Relation of Young-Age Mortality Rate to Old-Age Mortality Rate and Mortality Acceleration

Before examining cohort trends in the rates of demographic and biological aging, I first test Hypotheses 1a and 2a. Figure 2 describes the age-specific mortality rate from ages 0–1 to ages 90–94 for five select Swedish cohorts born between 1751 and 1915.

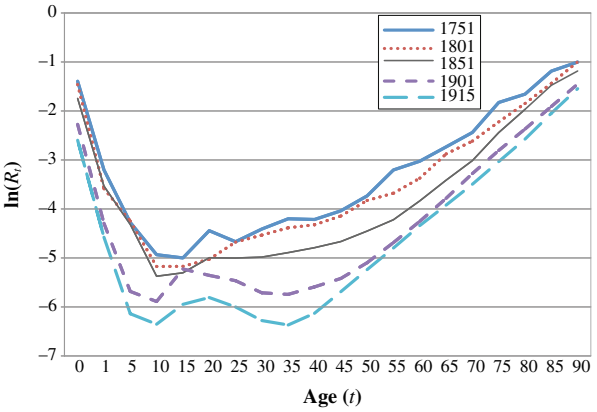

Fig. 2 Age-specific mortality rate over the life span, Sweden, 1751–1915 birth cohorts. $\ln(R_t)$ represents logarithm transformation of age-specific mortality rate at age t

(Figures for the other six countries are available upon request.) The general pattern is a downward shift in the mortality curve for later cohorts, which is consistent with Panel A in Fig. 1. In other words, later cohorts have both a lower young-age mortality rate and a lower old-age mortality rate than earlier cohorts. I also observe a mortality selection effect: in later cohorts, characterized by a lower young-age mortality rate, the acceleration of mortality after age 40 is steeper, which is also consistent with Panel A in Fig. 1.

To test whether these patterns represent statistically significant relationships, I regress the old-age mortality rate and the rate of mortality acceleration on young-age mortality rates. Table 1 shows the unstandardized coefficients obtained by regressing the old-age mortality rates between the ages 70 and 94 on young-age mortality rates in seven countries. Each mortality rate for a five-year span above age 70 is positively correlated with mortality rates from ages 0–1 to ages 10–14. This supports Hypothesis 1a, derived from cohort evolution theories.

Table 2 shows the unstandardized coefficients obtained by regressing the rate of mortality acceleration between ages 40 and 94 on young-age mortality rates in seven countries. Mortality acceleration parameters, α, after ages 40, 45, 50, 55, and 60 to 94 are negatively correlated with young-age mortality rates in most cases. This finding supports Hypothesis 2a, derived from population heterogeneity theory and the SM model. Together, these findings suggest that cohort evolution and mortality selection mechanisms coexist, as shown in panel A in Fig. 1.

The Trend in Mortality Acceleration (Rate of Demographic Aging)

I next explore whether mortality acceleration (or the rate of demographic aging) after age 70 is constant (Hypothesis 1b), as implied by the cohort morbidity phenotype theory. Figure 2 shows that the log mortality curves of five Swedish cohorts are essentially parallel after age 70, which is consistent with Finch and Crimmins' (2004) argument for fixed mortality acceleration across cohorts. However, a more formal test of cohort trends in mortality acceleration is required. In Fig. 3, I plot the trend in this parameter across cohorts in seven countries and find that it varies from cohort to cohort. The longest line

Table 1 Unstandardized coefficients for regression of old-age mortality rates age 70–94 on young-age mortality rates among seven countries (standard errors in parentheses)

	$\ln(R_{70–74})$	$\ln(R_{75–79})$	$\ln(R_{80–84})$	$\ln(R_{85–89})$	$\ln(R_{90–94})$
$\ln(R_{0–1})$	0.258***	0.190***	0.092***	0.058**	0.029
	(0.025)	(0.024)	(0.020)	(0.020)	(0.018)
$\ln(R_{1–4})$	0.032	0.074***	0.120***	0.113***	0.051**
	(0.024)	(0.023)	(0.019)	(0.018)	(0.017)
$\ln(R_{5–9})$	0.116***	0.143***	0.170***	0.163***	0.149***
	(0.021)	(0.020)	(0.016)	(0.016)	(0.015)
$\ln(R_{10–14})$	0.253***	0.219***	0.174***	0.155***	0.164***
	(0.025)	(0.024)	(0.019)	(0.019)	(0.018)
R^2	.65	.67	.76	.74	.70

Notes: $\ln(R_{number–number})$ represents logarithm transformation of age-specific mortality rate within each age group. Age-specific mortality rates are directly available in the Human Mortality Database.

**$p < .01$; ***$p < .001$

Table 2 Unstandardized coefficients for regression of rate of mortality acceleration from age 40 to age 94 on young-age mortality rates among seven countries (standard errors in parentheses)

	$\alpha_{40–94}$	$\alpha_{45–94}$	$\alpha_{50–94}$	$\alpha_{55–94}$	$\alpha_{60–94}$
$\ln(R_{0–1})$	–0.007***	–0.007***	–0.008***	–0.008***	–0.008***
	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)
$\ln(R_{1–4})$	–0.004***	–0.003***	–0.003***	–0.002**	–0.002**
	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)
$\ln(R_{5–9})$	0.000	0.000	0.000	0.001	0.001
	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)
$\ln(R_{10–14})$	–0.006***	–0.004***	–0.002**	–0.001	–0.000
	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)
R^2	.63	.58	.49	.38	.34

Notes: $\ln(R_{number–number})$ represents logarithm transformation of age-specific mortality rate within each age group. Age-specific mortality rates are directly available in the Human Mortality Database. $\alpha_{number–94}$ represents the rate of mortality acceleration from ages 40, 45, 50, 55, and 60 to 94. α is calculated using the equation $\ln(R_t) = \ln(R_0) + \alpha t$, where age-specific mortality rates R_t are available from the data. α is the slope of the log mortality curve.

**$p < .01$; ***$p < .001$

represents Sweden, and the dotted line represents Iceland, which has more random variation compared with other countries. The general pattern is that the mortality acceleration parameter $\alpha_{70–94}$ increased across cohorts until the mid-nineteenth century, decreased for about 10 years, increased again until the 1870s, decreased afterward, but resumed its increase in the early twentieth century and beyond. If the cohort morbidity phenotype theory proposes that mortality acceleration (the rate of demographic aging) is fixed across cohorts, then this proposition is not supported by my data.

What has driven the changes in the rate of demographic aging? Mortality selection mechanisms suggest that the rate of demographic aging should be negatively affected by young-age mortality. In other words, mortality acceleration in older ages is weak when young-age mortality is high and strong when young-age mortality is low. Figure 4 plots

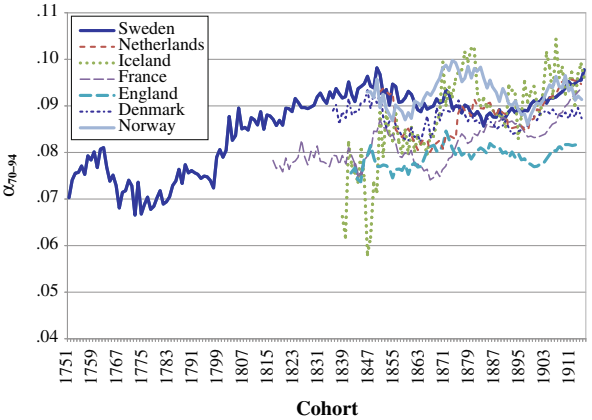

Fig. 3 The trend of rate of demographic aging between age 70 and 94 ($\alpha_{70–94}$) across cohorts. $\alpha_{70–94}$ represents the rate of demographic aging from age 70 to 94. α is calculated using the equation $\ln(R_t) = \ln(R_0) + \alpha t$, where age-specific mortality rates R_t are available from the data. α is the slope of the log mortality curve

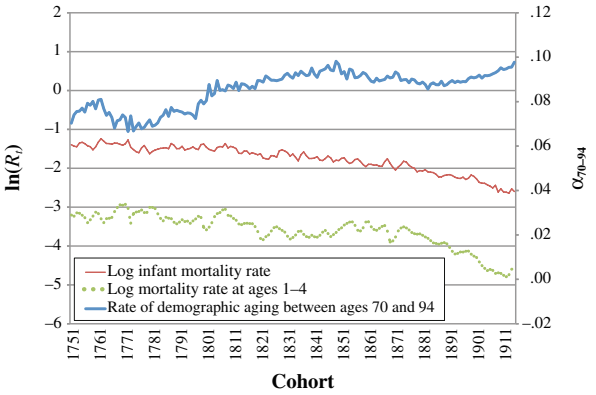

Fig 4 The trend of rate of demographic aging between age 70 and 94 ($\alpha_{70–94}$), log infant mortality rate, and log mortality rate at age 1–4 in Sweden across cohorts 1751–1915

the trends in mortality acceleration between age 70 and age 94; the log infant mortality rate; and the log mortality rate at ages 1–4 across Swedish cohorts born between 1751 and 1915. The rate of demographic aging $\alpha_{70–94}$ appears to be negatively correlated with $\ln(R_{0–1})$ and $\ln(R_{1–4})$. Regression results for all seven countries, presented in the $\alpha_{70–94}$ column in Table 3, suggest that mortality rates before age 15 explain only 28 % of the

Table 3 The unstandardized coefficients for regression of rate of demographic aging $\alpha_{70–94}$ and rate of biological aging $B_{70–94}$ on young-age and late middle-age mortality rates (standard errors in parentheses)

	$\alpha_{70–94}$		$B_{70–94}$	
$\ln(R_{0–1})$	–0.012***		–0.0002***	
	(0.001)		(0.0000)	
$\ln(R_{1–4})$	0.002		0.0001***	
	(0.001)		(0.0000)	
$\ln(R_{5–9})$	0.002		0.0002***	
	(0.001)		(0.0000)	
$\ln(R_{10–14})$	–0.005***		0.0001**	
	(0.001)		(0.0000)	
$\ln(R_{55–59})$		0.002		0.0001
		(0.002)		(0.0001)
$\ln(R_{60–64})$		0.002		0.0001
		(0.003)		(0.0001)
$\ln(R_{65–69})$		0.002		0.0000
		(0.003)		(0.0001)
$\ln(R_{70–74})$		–0.024***		0.0001
		(0.003)		(0.0001)
R^2	.28	.56	.52	.25

Notes: $\ln(R_{number–number})$ represents logarithm transformation of age-specific mortality rate within each age group. Age-specific mortality rates are directly available in the Human Mortality Database. $\alpha_{70–94}$ represents the rate of demographic aging from age 70 to 94. α is calculated using the equation $\ln(R_t) = \ln(R_0) + \alpha t$, where age-specific mortality rates R_t are available from the data. α is the slope of the log mortality curve. $B_{70–94}$ is calculated using equation $\ln(R_0) = (-1/B)\alpha + \ln(K)$ by assigning a value of K ($K = 1$) as suggested by Strehler and Mildvan (1960).

**$p < .01$; ***$p < .001$

variance in $\alpha_{70–94}$. On the other hand, mortality rates in late middle age (ages 55 to 69) are not significantly related with $\alpha_{70–94}$; but, together with the mortality rate at age 70, they explain 56 % of the variance in the rate of demographic aging $\alpha_{70–94}$. Although mortality acceleration during late life is affected by mortality selection in early life, it appears to be more directly affected by selection in late life, as indicated by a strong correlation between the mortality acceleration parameter spanning ages 70–94 and the mortality rate at the beginning of this time span, $\ln R(_{70–74})$.

The Trend in the Rate of Biological Aging

Has the rate of biological aging evolved similarly to the rate of demographic aging? Specifically, has the rate of biological aging increased for cohorts born after the late nineteenth century or early twentieth century? Figure 5 plots the rate of biological aging between ages 70 and 94 across the cohorts observed in each country. The longest line represents Sweden, and the dotted line represents Iceland, which exhibits more random variation than the other countries. The general pattern is that the rate of biological aging fluctuated widely until the mid-nineteenth century birth cohort, significantly declined, and then stabilized starting with the early twentieth century cohort. This pattern is not consistent with the hypothesis of a fixed rate of biological aging (Hypothesis 2b) derived from population heterogeneity theory and the SM model. The turning points in the evolution of the rate of biological aging coincide with the stages of epidemiologic transition. In the first stage (the age of pestilence and famine), mortality rates fluctuated greatly in response to epidemics, famines, and war. In the second stage, beginning in the mid-nineteenth century, young-age mortality risk declined as a result of receding pandemics. Since the late nineteenth century and early twentieth century, some advanced societies entered the third stage of the epidemiologic transition, and old-age mortality started declining. The coincidence of the trend in biological aging with the

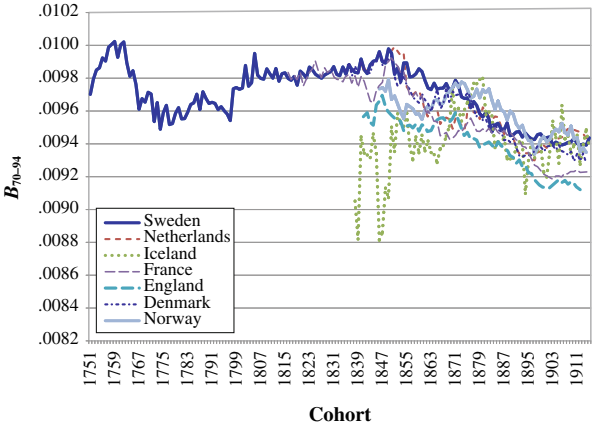

Fig. 5 The trend of rate of biological aging between age 70 and 94 across cohorts. $B_{70–94}$ is calculated using equation $\ln(R0) = (-1/B)\alpha + \ln(K)$ by assigning a value of K ($K = 1$) as suggested by Strehler and Mildvan (1960). The initial mortality rate $\ln(R_0)$ at age 70 and rate of mortality acceleration α from age 70 to 94 are calculated using the equation $\ln(R_t) = \ln(R_0) + \alpha t$, where age-specific mortality rates R_t are available from the data. $\ln(R_0)$ and α are the intercept and slope of the log mortality curve, respectively

stages of the epidemiologic transition suggests that the rate of biological aging is particularly affected by young-age mortality risk given that both have similar historical trends. The rate of biological aging is not affected as much by old-age mortality risk: in the third stage of the epidemiologic transition, old-age mortality risk declined but the rate of biological aging stabilized.

Figure 6 compares the trends in the rate of demographic aging $\alpha_{70–94}$ and the rate of biological aging $B_{70–94}$ in Sweden. These two parameters were close to each other before the 1859 birth cohort but began to diverge in subsequent cohorts born during the age of receding pandemics, when young-age mortality risk substantially declined. The parameter $\alpha_{70–94}$ continued gradually increasing in cohorts born after 1859 because of reduced mortality selection in both early and late life, but the parameter $B_{70–94}$ significantly declined and stabilized across cohorts born in the early twentieth century. The comparison of trends in the two parameters suggests that mortality selection does not contribute to the decreasing rate of biological aging because weaker mortality selection allows more frail individuals to survive to late age and potentially leads to higher rates of biological aging in later cohorts.

In order to test these two patterns, I regress the biological aging parameter $B_{70–94}$ on young- and old-age mortality rates for all seven countries. The results are presented in the $B_{70–94}$ column of Table 3. Mortality rates before age 15 are positively correlated with $B_{70–94}$, except for $\ln R(_{0–1})$, and explain 52 % of the variance in $B_{70–94}$. The negative coefficient for $\ln R(_{0–1})$ does not necessarily mean that a selection mechanism is in effect given that this mortality rate is positively and significantly correlated with $B_{70–94}$ before other young-age mortality rates are added to the model. In addition, mortality rates in late middle age and late age (ages 55 to 74) are not significantly related to $B_{70–94}$. Therefore, the rate of biological aging (B) is positively affected by young-age mortality rates but not by old-age mortality rates, and it is generally not affected by changing regimes of mortality selection. These findings stand in sharp contrast to my findings on the rate of demographic aging, which indicate that mortality selection is in effect and that the rate of demographic aging responds more strongly to the mortality rate at age 70 than to early-life mortality rates.

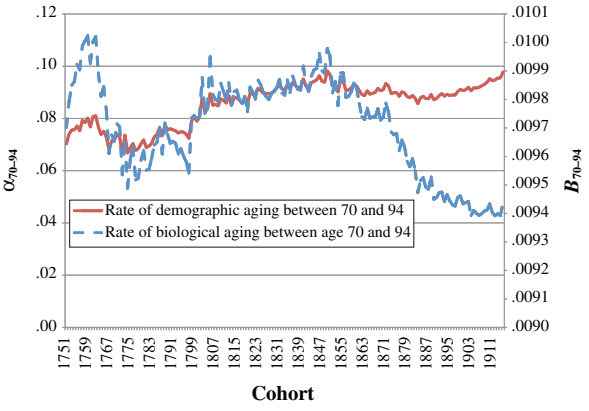

Fig 6 Comparison between rate of demographic aging $\alpha_{70–94}$ and rate of biological aging $B_{70–94}$ in Sweden

Discussion and Conclusion

This study investigates the aging process in the context of cohort evolution and mortality selection mechanisms. Using data from the Human Mortality Database on cohort age-specific mortality rates among seven advanced societies, this study begins by testing cohort evolution theories (predicted by the cohort morbidity phenotype and technophysio evolution theories) and mortality selection mechanism (implied by the theory of population heterogeneity and the SM model). Next, this study investigates trends in the rates of demographic and biological aging; the relationship between these two measures; and their responsiveness to cohort evolution and mortality selection processes. My results show a positive correlation between young- and old-age mortality rates, as predicted by cohort evolution theories; they also show a negative correlation between young-age mortality rates and mortality acceleration in late life, as predicted by mortality selection mechanism.

Taken together, these findings mean that the mortality selection mechanism does not override the process of cohort evolution. Although later cohorts experience a stronger acceleration of mortality in late age because of less heterogeneity in the distribution of frailty and weaker mortality selection over the life course, their old-age mortality rates remain lower than those of earlier cohorts, owing to substantially lower mortality risks early in the life course. This conclusion finds further support in prior empirical results. For example, Fogel and Costa (1997:56) found that "young adults born between 1822 and 1845 who survived the deadly infectious diseases of childhood and adolescence were not, as some have suggested, freer of degenerative diseases than persons of the same ages today; rather they were more afflicted."

I also find that the rate of demographic aging (mortality acceleration) after age 70 is not fixed. This parameter increased across cohorts until the mid-nineteenth century, decreased for about 10 years, increased until the 1870s, decreased, and then increased again beginning in the early twentieth century. Mortality acceleration during late life is affected by mortality selection in early life—lower young-age mortality rates are associated with stronger mortality acceleration past age 70—but mortality acceleration is more directly affected by mortality selection in late life, or the mortality rate at age 70 (i.e., a strong SM correlation between rate of increase in mortality rate and initial mortality rate). Mortality acceleration past age 70 is not affected by mortality rates in late middle age (ages 55 to 69), which indicates that mortality acceleration in late age is not sensitive to mortality selection in late middle age, consistent with the findings of Janssen and colleagues (2005).

The rate of biological aging fluctuated widely until the mid-nineteenth century birth cohort, declined significantly, and then stabilized since the early twentieth century cohort. The turning points in the evolution of the biological aging rate are consistent with the stages of the epidemiologic transition. In particular, biological aging exhibits a trend break when young-age mortality substantially declined in the mid-nineteenth century, during the age of receding pandemics; and another trend break when old-age mortality significantly declined in the early twentieth century. This concordance implies that the rate of biological aging is affected more strongly by young-age mortality rates than by old-age mortality rates. A comparison of the trends in the rates of biological and demographic aging shows the two move together until the mid-nineteenth century birth cohort, and then diverge. This finding suggests mortality selection regimes do not

determine the rate of biological aging. If mortality selection were relevant to the rate of biological aging, the decrease in the young-age mortality rate since the mid-nineteenth century would have increased the rate of biological aging, but it did not. A fixed-effect regression analysis confirms these insights from the graphical analysis (Table 3).

My findings have several important implications. First, the rate of demographic aging—the mortality acceleration parameter α—might be used to approximate the rate of biological aging when young-age mortality rates are very high (e.g., because of pervasive epidemics). However, this approximation would be misleading for cohorts born in developed countries after the mid-nineteenth century, when young-age mortality rates substantially declined, because mortality selection over this time had altered the rate of demographic aging but not the rate of biological aging. The substitution of the rate of demographic aging for the rate of biological aging would be even more misleading in cohorts born after the early twentieth century. In these cohorts, old-age mortality rates significantly declined, and this altered their rate of demographic aging but not their rate of biological aging.

Second, my finding that the rate of biological aging is affected by young-age mortality rates but not by old-age mortality rates or mortality selection further enriches cohort evolution theories. The decline in young-age mortality risk, whether resulting from lower exposure to infection and inflammation or improved nutrition during infancy and childhood, not only reduces the level of mortality risk throughout the life course but also slows the rate of biological aging. This deceleration of biological aging at the individual level provides a micro-level mechanism that explains the positive correlation between young- and old-age mortality rates across cohorts.

Third, despite continuing reductions in the old-age mortality rate, the rate of biological aging has stabilized because of medical advancements and socioeconomic development since the early twentieth century. This finding is somewhat consistent with studies that claim a fixed senescence process based on period data collected since the mid-twentieth century (Strehler and Mildvan 1960; Vaupel 2010b). However, my study suggests that the rate of biological aging has not always been fixed. This finding also explains why B (the rate of biological aging) has decreased in period data after 1955, given that this decrease could be linked to decreases in B among cohorts born between the mid- and late-nineteenth century (Zheng et al. 2011). Although the rate of biological aging has stabilized, the mortality rate, especially in old age, continued declining in the twentieth century as people became increasingly likely to reach old age in better health. This phenomenon, described as delayed but not decelerated senescence, is attributable to improved living conditions and medical advancements (Vaupel 2010b).

Fourth, although I do not intend to join the debate on the limit of the human life span, my study may further fuel this debate. The stabilization in the rate of biological aging (B) may imply that the limit of the human life span exists, as Strehler and Mildvan's (1960) model states, that the maximum human life span is given by the inverse of B (i.e., $1 / B$). Because SM theory is deterministic in the tradition of classic life table and stable population theory in demography, the implied maximum human

life span (estimated by 1 / *B*) should be regarded as an expected value for a specific human population. In empirical applications, there will be stochastic variability around this expected value as individuals encounter environmental challenges to molecular bonds that may be sufficiently severe to cause death but vary in the timing of arrival. Thus, the expected maximum human life span for the population does not imply that all individuals must expire no later than that age (Zheng et al. 2011).

Applying SM's rule for determining the maximum human life span, my analysis suggests that age 115 may be the limit. The world's oldest living person at the time of this study was an American woman, Besse Cooper, who died in 2012 at age 116. She was one of eight people verified to live to age 116 (Swanepoel 2012). A French woman, Jeanne Calment, had the longest confirmed lifespan, living to age 122. Currently, there are approximately 500,000 living centenarians worldwide, and this number has increased by 7 % every year, but the number of living super-centenarians (older than age 115) has not changed (Ridley 2012). However, any statement about the limit of the human life span should be made carefully. In my study, young-age mortality rates explained about one-half of the variance in the biological aging parameter *B*, so *B* may decrease in the future because of the effect of unknown factors that contribute to the other half of the variance in *B*. Particularly, *B* may decline for cohorts born after the 1920s (for which I do not have data), and this would further increase the maximum human life span (estimated as 1 / *B*).

My study cannot reveal which mechanisms link a cohort's mortality risk at young ages to its rate of biological aging. It is unclear whether the declining rate of biological aging should be attributed to reductions in infection and inflammation during early childhood or instead to improved nutrition *in utero*, during infancy, or in early childhood. The theory of cohort morbidity phenotype emphasizes inflammation as the mechanism linking young- and old-age mortality risks and predicts that "once childhood infection is low, it can no longer be a factor in explaining old-age trends" (Crimmins and Finch 2006:499). This interpretation focuses exclusively on cohorts born before the twentieth century, when the levels of childhood infection were high. Similarly, if inflammation were the dominant mechanism linking young-age mortality risk to the rate of biological aging, a weakening correlation between these two factors would be evidenced among cohorts born since the early twentieth century. A stable rate of biological aging among cohorts born in the early twentieth century coincides with ongoing declines in young-age mortality rates (Figs. 4 and 6), meaning that young-age mortality risks cannot explain the trend in the rate of biological aging for these cohorts. A fixed-effects regression analysis further supports this conclusion (Table 4 in the Appendix). Based on these findings, we may suspect that reductions in infection and inflammation during early childhood have made a significant contribution to the decline in the rate of biological aging.

Reductions in infection and inflammation may slow the aging process because infection can cause permanent damage to vital organs, including the heart, lungs, and kidneys (Buck and Simpson 1982; Lunn et al. 1991; Mercer 1990; Preston et al. 1972). Similarly, having fewer infections at a young age reduces and delays the development of atherosclerotic and thrombotic

conditions by reducing the lifetime inflammatory burden (Crimmins and Finch 2006). The biological mechanism linking a reduction in early-life infections to a slower senescence is complicated. According to antagonistic pleiotropy theory, a single gene controls multiple traits, some of which may increase fitness in early life and others of which may be detrimental to fitness in late life, leading to senescence (Williams 1957). Selection does not eliminate this gene because it improves survival in early life; therefore, senescence may be the product of such selective pressures. A reduction in early-life infections may weaken the expression of this kind of gene at young ages, which may then slow senescence in late age, although this presumption awaits empirical testing. A reduction in early-life infections may also decrease the risk of detrimental mutations in late life, which, according to the mutation accumulation theory, cause aging (Medawar 1952).

These mechanisms notwithstanding, the decline in early-life infections is not an exclusive mechanism explaining the decline in the rate of biological aging. Improved nutrition and living standards during early childhood may also be very important factors in the deceleration of biological aging because improved nutrition can strengthen resistance to infection (Fogel 2004b), which may, in turn, weaken antagonistic pleiotropy and the accumulation of detrimental mutations. Moreover, improved energy intake will increase the resources available for the repair and maintenance of the body, perhaps then slowing senescence. According to the disposable soma theory, aging is the result of a compromise in energy allocation between repair and reproduction (Kirkwood 1977). The reason why the rate of biological aging stabilized despite continual improvements in living standards during the twentieth century may be because this rate has reached its minimum.

This study has several limitations. First, although the Human Mortality Database is considered to be of high quality and has been widely used for cross-national and historical research on old-age mortality (Ho and Preston 2010; Wilmoth and Horiuchi 1999; Yashin et al. 2001), I cannot totally dismiss the possibility that my results are biased by age misreporting in the death rates (e.g., Preston et al. 1999). I have tried to minimize this problem by limiting analyses to ages 94 and younger. As a cautionary example, Preston and colleagues (1996) found that the correction of age misreporting among older blacks could eliminate the mortality crossover between white and black adults at older ages. Similarly, greater mortality acceleration among later cohorts in this study may be caused by better data rather than by mortality selection.[4] In contrast to my findings, Himes and colleagues (1994) found that the slope of the age-specific mortality curve declines rather than increasing over successive periods.

For several reasons, though, I believe that my finding of an increasing slope of the mortality curve (greater mortality acceleration) across cohorts is credible. Unlike Himes and colleagues, I analyze the trend across cohorts rather than across periods. I performed a comparable analysis of this trend over historical periods using Human Mortality Database and found a pattern similar to the one reported by Himes and colleagues (1994): the slope of the mortality curve at older ages declines in successive periods. A discrepancy between cohort and period trends in the mortality curve has been previously reported by Finch and Crimmins (2004). Beltrán-Sánchez and

[4] I thank an anonymous reviewer for pointing out this potential problem.

colleagues (2012) also found that the Gompertz rate of mortality acceleration at older ages rises across 630 cohorts born throughout the nineteenth and early twentieth centuries in nine European countries. Finally, the amplification of mortality acceleration across cohorts starts at younger ages rather than older ages, as shown in Fig. 2. Therefore, my result for the trend in mortality acceleration is unlikely to be totally explained by changing patterns of old-age misreporting. However, the extent to which misreporting affects these conclusions is still unknown, and this problem merits further analyses using a different data set.

A second limitation of this study arises from my use of the SM model. This is an elegant biodemographic model of the age dependence of human mortality, and it links aggregate age-specific mortality rates, environmental insults, and individual energy reserves and biological aging. This model provides a single and straightforwardly derived parameter to estimate the rate of biological aging (B). Some of the studies examining the SM model have found that B is constant (Prieto et al. 1996; Riggs 1993; Riggs and Myers 1994), but others suggest that B is not constant (Yashin et al. 2000, 2001, 2002a; Zheng et al. 2011). This study extends prior studies by investigating how and why B may change across cohorts. I must note that B represents the average rate of biological aging within each cohort, meaning that there may be some stochastic variability around this expected value at the individual level. Moreover, B is only one measure of the rate of biological aging. Other studies have used biomarkers (e.g., allostatic load), the frailty index, and the vitality index as indicators of aging (Karlamangla et al. 2002; Levers et al. 2006; Mitnitski et al. 2005; Vasto et al. 2010; Yashin et al. 2007).

Using biomarkers to measure the rate of aging can yield a more detailed understanding of the aging process, and research in this area has advanced substantially in recent years. Still, the links between biomarkers and the aging process are very complex and not completely known. Some biomarkers may be positively related to aging, and others may be negatively associated with aging; moreover, the relationship between biomarkers and the aging process may be heterogeneous across individuals and may also vary over the life course (Yashin 2013). Furthermore, nonmonotonic age patterns of biomarkers (e.g., body mass index, which may rise and fall over the life course) introduce additional challenges for using biomarkers to measure biological aging (Yashin et al. 2013). Other important biomarkers of aging are unknown or cannot be measured (Piantanelli et al. 2001). Together, measured and unmeasured biomarkers characterize the biological mechanisms involved in the regulation of aging.

The research investigating the interrelation of biomarkers, the frailty index, the vitality index, and SM's estimate of biological aging (B) is limited. Zuev and colleagues (2000) found that the average life course decline of a metabolic rate indicator—a vitality index constructed from physiological indicators of metabolic activity—is very close to the rate of biological aging, B, in the SM model. This suggests that B is quite consistent with the rate of aging as derived from biomarkers, at least for this vitality index. Future analyses, however, should examine whether historical trends in the rate of biological aging, B, are consistent with trends in the rate of aging as measured by biomarkers. Historical data on the latter, however, are very limited, making SM's estimate of the rate of biological aging especially useful for analyses of past cohorts.

Aging is an extremely complicated process, driven by the interplay of genes and the environment. This study tries to understand aging in the context of cohort evolution and mortality selection. These two forces operate differently on demographic and biological aging. Demographic aging (the acceleration of the mortality rate in late life) is affected by mortality selection at young ages, and even more so by mortality selection at late ages. This causes later cohorts to have higher rates of demographic aging than earlier cohorts. Biological aging is not affected by mortality selection but rather by cohort evolution, whereby reductions in young-age mortality rates cause lower rates of biological aging in old age. The rate of biological aging has stabilized for cohorts born since the early twentieth century despite ongoing declines in young-age mortality risk. At this time, it is unknown whether this stabilization is due to diminished infections at young ages or due to the rate of biological aging reaching a minimum, such that it cannot be reduced further. Future research should investigate the mechanisms linking young-age mortality risk to the rate of biological aging, and ascertain whether stabilization in the rate of biological aging for cohorts born in the early twentieth century represents a culminating or a transitory stage.

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Appendix

Table 4 The unstandardized coefficients for regression of rate of biological aging $B_{70–94}$ on young age mortality rates since the early twentieth century birth cohort (standard errors in parentheses)

	$B_{70–94}$
$\ln(R_{0–1})$	.0000
	(.0000)
$\ln(R_{1–4})$	.0000
	(.0001)
$\ln(R_{5–9})$	.0001
	(.0000)
$\ln(R_{10–14})$	.0000
	(.0000)
R^2	.05

Notes: $\ln(R_{number\text{-}number})$ represents logarithm transformation of age-specific mortality rate within each age group. Age-specific mortality rates are directly available in the Human Mortality Database. $B_{70–94}$ is calculated using equation $\ln(R_0) = (-1/B)\alpha + \ln(K)$ by assigning a value of K ($K = 1$) as suggested by Strehler and Mildvan (1960). The initial mortality rate $\ln(R_0)$ at age 70 and rate of mortality acceleration α from age 70 to 94 are calculated using the equation $\ln(R_t) = \ln(R_0) + \alpha t$, where age-specific mortality rates, R_t, are available from the data. $\ln(R_0)$ and α are the intercept and slope of the log mortality curve, respectively.

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