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HAS ACTUARIAL AGING "SLOWED" OVER THE PAST 250 YEARS? A COMPARISON OF SMALL-SCALE SUBSISTENCE POPULATIONS AND EUROPEAN COHORTS

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G.C. Williams's 1957 hypothesis famously argues that higher age-independent, or "extrinsic," mortality should select for faster rates of senescence. Long-lived species should therefore show relatively few deaths from extrinsic causes such as predation and starvation. Theoretical explorations and empirical tests of Williams's hypothesis have flourished in the past decade but it has not yet been tested empirically among humans. We test Williams's hypothesis using mortality data from subsistence populations and from historical cohorts from Sweden and England/Wales, and examine whether rates of actuarial aging declined over the past two centuries. We employ three aging measures: mortality rate doubling time (MRDT), Ricklefs's ω, and the slope of mortality hazard from ages 60–70, m'_{60-70}, and model mortality using both Weibull and Gompertz–Makeham hazard models. We find that (1) actuarial aging in subsistence societies is similar to that of early Europe, (2) actuarial senescence has slowed in later European cohorts, (3) reductions in extrinsic mortality associate with slower actuarial aging in longitudinal samples, and (4) men senesce more rapidly than women, especially in later cohorts. To interpret these results, we attempt to bridge population-based evolutionary analysis with individual-level proximate mechanisms.

KEY WORDS: **Biodemography of aging, England, hunter–gatherers, life-history theory, mortality, senescence, Sweden, Williams's hypothesis.**

Evolutionary theories of aging assign great importance to extrinsic mortality for determining the optimal allocation of energetic resources to growth, reproduction, maintenance, and repair functions. Allocation to the latter largely determines the normative length of the life span (Williams 1957; Edney and Gill 1968; Kirkwood and Holliday 1979; Abrams 1991; Austad 1992; Stearns 1992; Charlesworth 1993; Partridge and Barton 1993; Cichón 1997). Extrinsic mortality accounts for most preadult deaths, and therefore affects the proportion of individuals in a population that will reach old age, and the intensity of natural selection with age (Williams 1957; Hamilton 1966). If extrinsic mortality is high, average survival time will be short and selection should favor greater investment in early reproduction rather than in maintenance and soma repair. When extrinsic mortality is low, as in protected or buffered environments, greater incentives to build and maintain the soma should lead to a longer life span.

The concept of extrinsic mortality risk has been essential in discussions about life-history evolution. Extrinsic mortality includes causes of death whose origin is external to the individual organism at risk (Carnes and Olshansky 1997). "Intrinsic," or endogenous, mortality reflects deaths due to a deterioration in physiological processes (Charlesworth 1980; Hayflick 1994;

Ricklefs 1998). Predation, accidents, and infectious disease are typically classified as extrinsic causes, whereas cancers, heart disease, and other degenerative illnesses are considered intrinsic. An intrinsic mortality profile is difficult to observe in wild populations where many individuals may die from extrinsic causes. It is not easy to partition total mortality into meaningful components, but attempts to do so have nonetheless provided rich insights pertinent to the study of senescence (Carnes et al. 1996; Carnes and Olshansky 1997). In this article we take an alternative approach that does not require information on causes of death, but instead partitions mortality into age-independent (extrinsic) and age-dependent (intrinsic) components through the use of competing hazard models. In empirical investigations, extrinsic mortality has been operationalized as age-independent mortality (Gage 1989; Partridge 1989) and as the lowest preadult mortality rate (Promislow 1991) because most deaths at these pre-adult ages are due to extrinsic causes (Carnes et al. 2006). Specifying extrinsic mortality as age-independent is a useful heuristic, even if any particular "extrinsic" cause of death may be age-dependent (Carnes et al. 2006).

This popularized relationship between extrinsic mortality and senescence, labeled the "Williams hypothesis" by Abrams (1993), has been widely cited by evolutionary researchers over the past several decades. As summarized in a recent review, "Williams's hypothesis stands as the main predictive tool used by comparative biologists when investigating how senescence schedules are shaped in the wild" (Williams et al. 2006:458). The proposal that extrinsic mortality structures the life history of an organism and shapes aging rates has motivated studies of variation in life span and senescence among animals. For example, larger body size and protective shells, as among tortoises and turtles, reduce extrinsic mortality and extend life span by decreasing risk of predation, parasitism, starvation, or weather-induced stress (Sacher 1959; Calder 1984; Keller and Genoud 1997; Dudycha 2001). A now-famous quasi-experiment comparing mainland and island populations of opossums experiencing different predation rates (and hence extrinsic mortality risk), revealed longer life spans and delayed maturation in the less predated island population (Austad 1993).

Despite these and other observations that empirically support Williams's hypothesis (e.g., Stearns et al. 2000 on fruit flies; Dudycha 2001 on daphnia; Bryant and Reznick 2004 on guppies), several theorists have demonstrated that population dynamics (density-dependence) and interactions between senescent traits and susceptibility (condition-dependence) can modify the expected relationship between mortality and evolved senescence. Abrams (1993) shows that a positive relationship between extrinsic mortality and senescence requires density-dependent population growth that impacts fertility equally at all ages or that preferentially impacts juvenile survival. A positive, negative, or neutral relationship between extrinsic mortality and aging rate in any particular test may reveal more about underlying population dynamics or condition-dependence than about selective influences on aging. Additional confusion stems from disparate definitions and approaches for measuring both extrinsic mortality and aging (Bronikowski and Promislow 2005). The difficulty of grouping causes of death into mutually exclusive categories of extrinsic and intrinsic mortality has long been recognized (Pearl and Miner 1935; Carnes et al. 2006) and different definitions of aging can lead to conflicting results concerning Williams's hypothesis (Williams et al. 2006).

To describe age-related changes associated with the aging process we use the oft-cited definition of actuarial aging or senescence as the increase in mortality rate or decrease in reproductive rate with age (Abrams 1991; Rose 1991; Holmes and Austad 1995; Ricklefs and Scheuerlein 2001). A critical assumption is that changes in late-age mortality reflect underlying progressive, physiological decline in vital capacity with age (McClearn 1997). Aside from anecdotal reports of various chronic diseases among select members of past populations (e.g., Magee 1998; Greaves 2000), much of our ability to understand details of the aging process throughout human history requires a focus on mortality rates. There is general support at the cellular level that actuarial senescence at the population level relates to biological senescence at the individual level, giving justification to the demographic focus on mortality rates in the absence of other data (Austad and Fischer 1991; Sohal et al. 1993; Promislow et al. 1995; Martin et al. 1996; Ogburn et al. 1998; Kapahi et al. 1999; Kirkwood 2002; but see Yashin et al. 2002b).

Our goal in this article is threefold. We first examine whether rates of actuarial senescence among remote, small-scale societies differ from those encountered in modern nation-states. Modern hunter–gatherers are not living fossils nor do they represent the diversity in past foraging populations. They do, however experience economic, health and mortality conditions resembling those of ancestral human environments. Their study thus offers one window into the selective context of aging among prehistoric humans. We employ the largest and most complete set of small-scale hunter–gatherer and forager–horticulturalists that currently exists (Gurven and Kaplan 2007). Although the analysis is cross-sectional, longitudinal data exist for several populations, allowing us to additionally compare mortality and aging rates before and after phases of acculturation.

Second, we explore evidence for changes in the rate of actuarial senescence over the past several hundred years in two European populations. Here we use mortality life tables from Sweden over the past 250 years, the longest record of mortality courtesy of the Human Mortality Database (HMD 2007). We also include England and Wales over the past 160 years as a comparative dataset.

Finally we test whether change in actuarial senescence is associated with longitudinal change in extrinsic mortality (Williams's hypothesis) and whether this relationship differs significantly among males and females. It has been argued that future changes in the rate of physiological aging may require substantial achievements in regenerative medicine, including embryonic stem cell-based organ replacement and telomerase gene alteration. It is important to consider how much change has already occurred due to reductions in extrinsic mortality from improvements in hygiene, sanitation, nutrition, and medical technology, and from the waning of infectious disease. Does the temporal decline in extrinsic mortality throughout recent history associate with a decline in the rate of aging as suggested by Williams's hypothesis, and if so, is this result indicative of evolutionary change? Our analyses provide an important first test of a life-history approach to the evolution of senescence in humans that until now has been applied only to birds, fish, and mammals.

The following section summarizes empirical evidence for changes in rates of senescence among humans, and is followed by a discussion of different approaches of measuring actuarial aging or senescence. We then outline the specific predictions tested in the article, followed by a description of our methods and data, then results of our tests. We first examine regional patterns in extrinsic mortality and several measures of aging among small-scale subsistence societies, and then focus on temporal patterns in these same variables among Swedish and English men and women.

SENESCENCE IN HUMANS

Human life expectancy has continued to increase over the past 150 years (Oeppen and Vaupel 2003), and modal age of adult death has increased as well (Cheung et al. 2005). This shift in the modal age of adult death describes a lengthening of the human life span for an increasing proportion of the population. Although life expectancy gains in the first half of the 20th century were largely due to improvements in infant and child survivorship, subsequent gains in the latter half of the 20th century resulted from adult mortality decline, including late age mortality (Preston 1976; Gage 2005). Currently in the United States, the largest growing sector of the population is that of age 65+ individuals (NIA/NIH 2007), with remaining life expectancy at age 65, e_{65}, having nearly doubled during the past century in the United States and much of the developed world. Since the mid-18th century in Sweden, e_{50} has increased by 12 years, e_{60} by about 10 years and e_{70} by 6 years. By the year 2050, there will be a million centenarians in the United States, a 20-fold increase over the roughly 50,000 identified in the 2000 census (U.S. Census Bureau 2001).

That mortality rates have declined at older ages and remaining life expectancy has improved is not surprising or controversial. However, whether this overall mortality decline indicates a slowing of the aging process itself, such that a 55-year-old woman today is biologically "younger" than a 55-year old a hundred years ago, is still an unresolved question. Individuals of disparate ages clearly show similar levels of a variety of aging biomarkers. Tsimane forager–horticulturalists by age 40 have been exposed to the same number of years of high levels of the acute-phase protein marker of inflammation, C-reactive protein, as Americans by age 55 (Gurven et al. 2008). However, decreases in adult mortality may also bring higher rates of chronic morbidity and disability among survivors at later ages. Alternatively, the initial onset of these conditions may be compressed to later in the life span (Fries 1980, 1989). A third and likely possibility is that the prevalence of many chronic conditions may increase at late ages but the effects of these chronic diseases are now less debilitating than at the turn of the century (Freedman et al. 2002; Crimmins 2004).

Fogel's and Costa's studies of Union Army veteran medical records provide a unique opportunity to compare physiological conditions of older adults before the 20th century with those of older adults now. These show earlier onset of chronic conditions, and higher rates of chronic respiratory problems, valvular heart disease, arteriosclerosis, joint problems, and functional limitation among veterans. Furthermore, these conditions became less disabling over time (Fogel et al. 1993; Fogel and Costa 1997; Manton et al. 1997; Costa 2000, 2002). Roughly one-fifth of this decline has been linked to infectious conditions experienced by the soldiers during wartime, such as malaria, typhoid, and rheumatic fever, with another third accounted for by the shift from manual to nonmanual occupations (Costa 2000). These trends may not be linear; recent evidence suggests that cardiovascular deaths declined continuously since the 1960s in the United States but may have increased again after 2000 due to complications of increasing obesity and type 2 diabetes (Nemetz et al. 2008).

Explanations for the decline in old age mortality, later onset of chronic disease, and decreased disability at older ages reflect historical changes in nutrition (McKeown 1976; Fogel and Costa 1997), infectious disease (Crimmins and Finch 2006), improvements in public health infrastructure and medical technology (Fogel 1997), and changes in health-related behaviors such as cigarette smoking (Cutler et al. 1998). These changes may have directly improved health and survivorship of older adults, but also indirectly through the cascading effects of improved nutrition and absence of infectious disease early in the life course (Elo and Preston 1992; Barker 1994; Gluckman and Hanson 2006; Finch 2007). An additional but related explanation includes the cumulative exposure to lipid oxidation and cumulative inflammatory processes as a significant cause of adult cardiovascular disease and cognitive aging (Plutsky 2001; Finch and Crimmins 2004).

MEASURING THE RATE OF AGING

Measuring senescence rate is not an easy task; most attempts involve some measure of mortality increase in adulthood. The most common mathematical function used to describe adult mortality patterns is the Gompertz equation, $m_x = m_g \exp(\gamma x)$, where γ is the exponential rate of mortality increase. With this parameterization, all subsequent mortality rates are multiplied by the initial adult mortality rate, m_g. The mortality rate doubling time (MRDT) $\ln 2/\gamma$, often used as a species-typical measure of aging (Finch et al. 1990), does not seem to vary much within species. MRDT estimates from both low and high mortality human populations usually fall in the range of 7–9 years (Finch et al. 1990). Comparisons of birds in the wild versus captivity, where extrinsic mortality is lower, reveal similar MRDTs in both cases (Finch et al. 1990; Ricklefs and Scheuerlein 2002). Promislow (1991) similarly found no association across mammals between extrinsic mortality (defined as minimal adult mortality rate) and the Gompertz rate parameter.

The Makeham modification adds an age-independent mortality component, m_0, such that the baseline mortality rate becomes $m_0 + m_g$, where m_g is the coefficient on the exponential term. A problem with the Gompertz–Makeham (GM) specification, however, is that m_g is embedded within the initial mortality hazard when $t = 0$ and at later ages. Due to the problem of interdependency between intrinsic and extrinsic components with the Gompertz model, Ricklefs (1998) promotes the use of a Weibull model. The Weibull model is defined as $m_x = m_0 + \alpha x^{\beta}$, with shape parameter β and scaling constant α. In both models, m_0 is considered a measure of extrinsic mortality whereas the other term estimates intrinsic mortality; such a crude distinction is necessary in the absence of biological data on causes of death, as is the case in this article.

Ricklefs (Ricklefs 1998; Ricklefs and Scheuerlein 2001; Ricklefs and Scheuerlein 2002) defines a different aging measure that makes use of both parameters in the intrinsic mortality term, and has units of 1/time: $\omega_w = \alpha^{1/(\beta+1)}$. A corresponding rate of aging for the Gompertz model is $\omega_G = \sqrt{m_g \gamma}$ (Ricklefs and Scheuerlein 2001). Ricklefs and Scheurlein (2001) define m_g as the coefficient multiplier on the exponential term of the Gompertz equation without the Makeham modification. Another option is to use the Makeham term (m_0), although m_0 is presumably not a component of senescent mortality, and ω would no longer be independent of m_0 with such a parameterization. For consistency, we choose the original Ricklefs specification of ω for the GM model even though it produces the paradoxical results that ω decreases with m_0, and ω is strongly positively correlated with MRDT. These paradoxical results are due to the statistical trade-off affecting estimation of the Gompertz parameters.

Both measures of ω reflect the magnitude of mortality increase and its acceleration, independent of m_0, rather than the specific rate, or doubling time, as with MRDT. In its focus on doubling time rather than absolute rates of mortality increase, MRDT is a rigid measure of actuarial aging. Ricklefs and Scheuerlein (2002) show through simulations that under most circumstances, Gompertz and Weibull models fit mortality data reasonably well and that aging measures (ω) are highly correlated across the two models.

Although both statistical models adequately fit adult mortality data, their specifications suggest alternative means of interpreting old age mortality. In the Gompertz model, initial mortality is multiplied by an exponential factor and so is consistent with the notion that older individuals are increasingly more vulnerable with age to similar causes of death that affect younger individuals (Ricklefs and Scheuerlein 2001). For the Weibull model, intrinsic mortality is separable from extrinsic age-independent mortality, such that deaths are more likely due to degenerative diseases and system failure. A somewhat similar interpretation follows with the GM model. In this article, we apply both GM and Weibull models to adult mortality. We estimate aging or senescence using Ricklefs's ω, and for comparison we include MRDT. We also consider the slope of the estimated mortality function (based on Weibull and GM). The slope, m'_{60-70}, describes the instantaneous rate of mortality change from ages 60 to 70.

PREDICTIONS (P)

If MRDT is a species-typical characteristic, a baseline prediction is that (P1) MRDT should be similar among our set of small-scale subsistence populations, including those that vary in their level of integration with national society and market affiliation. If declines in extrinsic mortality are too recent for evolutionary change to have impacted actuarial senescence, (P2) there should be no significant differences in MRDT across Swedish and English cohorts, and (P3) there should be no difference in MRDT between small-scale subsistence populations and modern nation states like Sweden. The alternative predictions, extending Williams's hypothesis to intraspecies comparisons, would state that the MRDT should vary negatively with the level of extrinsic mortality, m_0. Although changes in frequencies of the myriad genes impacting senescence are unlikely to have occurred in the past several hundred years, rates of genetic change will depend on the intensity of selection and extent of genetic variation. It is more likely, however, that as conditions improve and extrinsic mortality declines, existing flexibility in the genome could allow somatic improvements so that at the population level, actuarial senescence slows accordingly but with minimal genetic change.

We additionally test the above predictions using ω and m'_{60-70}, instead of MRDT, from both GM and Weibull models. Due to the interdependence problem of the GM model described in the previous section, we place more weight on results from the Weibull model.

We divide our analyses by sex in the European samples to compare rates of aging among women and men and the historical trajectory of change in these rates. Among most mammals, including humans, males exhibit higher mortality rates at most ages across the life span and have lower life expectancies at birth (Hazzard 1986). Evidence that males physiologically senesce at higher rates than women is less consistent, with women often reporting more morbid conditions (Idler and Benyamini 1997; Case and Paxson 2005). We make the tentative null predictions that (P4) men and women will show similar actuarial aging rates and that (P5) temporal changes in rates of actuarial aging will be similar for men and women.

Methods

DEMOGRAPHIC DATA

The sample of small-scale subsistence foraging and horticultural populations consists of four hunter–gatherer groups, four forager–horticulturalist group, and five acculturated hunter–gatherer groups (see Gurven and Kaplan 2007 for sample information and ethnographic descriptions). Whereas forager–horticulturalists have engaged in horticulture for many generations, acculturated hunter–gatherers have either recently begun adapting horticulture to their economy, and/or have some exposure to medicines, markets, and other modern amenities. The groups in our ethnographic sample of hunter–gatherers have had minimal or no exposure to modern medicine, and minimal or no inclusion of horticulture and market-derived foods in their diet. Survivorship and mortality profiles for these populations are based on actual deaths from prospective or retrospective studies, rather than on census data or incomplete mortality data fit to model life tables that already assume a certain age structure.

The European sample consists of 33 sex-specific Swedish life-tables for cohorts born 1751 through 1910 and 15 from England and Wales from 1840 through 1910, downloaded from the Human Mortality Database website in January 2007. We use quinquennial tables with one-year age gaps up until age 95 years. Due to data quality concerns with Swedish data before 1800 (Glei et al. 2007), we perform our analyses on Swedish data using start dates of both 1751 and 1800.

ANALYSIS

Although some forms of biological aging begin at birth or before maturity (Milne 2006), we base our analyses starting at age 15 for subsistence populations and age at lowest mortality for European populations (range: 10–18). We ignore early life periods because we are interested in mortality rate increase at adult ages that are not confounded by causes of juvenile death leading to high infant and child mortality.

Calculation of MRDT and ω is based on parameter estimates from nonlinear regression (PROC NLIN in SAS ver. 9.1) of mortality hazards using GM and Weibull model specifications. Mortality hazards, m_x, were calculated as $\ln\ (l_x/l_{x+n})/n$, where n refers to the age interval (5 years for subsistence populations and 1 year for Europeans), and l_x is probability of survivorship from ages 0 to x. The rate of change in mortality from ages 60 to 70, $m'_{60–70}$, is calculated as $[\hat{m}_g(\exp(70\hat{\gamma}) - \exp(60\hat{\gamma}))]/10$ for the GM and $[\hat{\alpha}(70^{\hat{\beta}} - 60^{\hat{\beta}})]/10$ for the Weibull model. Extrinsic mortality, m_0, was defined as the age-independent term in both the GM and Weibull models, and alternatively as the lowest mortality rate across the life course. The Pearson correlations between the model-based measure of extrinsic mortality and lowest mortality rate range from 0.82 to 0.97 using the Weibull-based m_0 and 0.60 to 0.93 using the GM-based m_0. To simplify presentation, we only use the model-based estimates of m_0, although no results change when using the other measure.

Due to the relatively small sample sizes of deaths and risk-years that comprise the life-tables for the small-scale subsistence populations, we weight these regressions by the number of risk-years in each age category. This approach is preferable to modeling survivorship (l_x) schedules, as done by others (e.g., Ricklefs and Scheuerlein 2001), due to the nonindependence of l_x among ages.

Statistical comparison of aging indicators within and across populations involved three steps. First, standard errors were obtained for MRDT with the delta method used to estimate the variance of a function of a random variable when formal evaluation is impossible (see Bishop et al. 1975). Second, standard errors for GM and Weibull $m'_{60–70}$ and ω were computed using a technique of resampling with repetition in the parameter subspace. GM and Weibull parameters were allowed to vary randomly based on their approximate standard errors attained from the nonlinear regression procedure. Resampled parameters provided simulated sampling distributions for respective aging indicators. Standard errors for $m'_{60–70}$ and ω were provided by the sampling variance of these distributions. Finally, aging indicators were compared using standard difference-of-means tests, with sample sizes corresponding to the number of datapoints in the original model regression. Statistical comparisons among subsistence populations are given in Table A1 of the Appendix, between Sweden and England/Wales by cohort and sex are given in Table A2 of the Appendix, and between European countries and subsistence populations in Table A3 of the Appendix.

Results

SUBSISTENCE SOCIETIES

MRDT, ω, and $m'_{60–70}$ for hunter–gatherers, forager—horticulturalists, and acculturated hunter–gatherers are presented in Table 1. Both Weibull and GM analyses show that extrinsic mortality is similar among hunter–gatherers and forager–horticulturalists, but is about one-half as high among acculturated

hunter–gatherers. Despite these differences in extrinsic mortality, there are few cases in which ω or $m'_{60–70}$ are consistently statistically distinguishable using both hazard models (Table A1), but MRDT is significantly higher by about 1–3 years among acculturated foragers. Ricklefs's ω is lowest among forager–horticulturalists, and $m'_{60–70}$ is almost double that of the other two categories. Within each of the macrocategories, there is significant variability in mortality parameters among specific populations according to the GM model. However, all aging measures are statistically indistinguishable according to the Weibull model.

There is no significant relationship between ω and m_0 among subsistence societies in the Weibull analysis ($P = 0.48$, Fig. 1A). However, m_0 is inversely related to both ω and MRDT in the GM analysis ($P = 0.03$, $P = 0.006$, respectively, Fig. 1B). That is, higher extrinsic mortality associates with faster aging in terms of MRDT but slower aging in terms of ω. No significant relationship exists between m_0 and $m'_{60–70}$ based on either Weibull or GM models.

These analyses compare different subsistence populations cross-sectionally. For six of the populations, mortality data exist for two different time periods, reflecting an "early" period of little contact with national society and a more traditional economy, and a "later" period after substantial integration and acculturation. We compare m_0 and ω between the two time periods in Figure 2 using Weibull. For all but one of the groups (Herero pastoralists), m_0 is lower in the later period (Fig. 2A). For four of the groups, ω is lower in the later period, no different in one group (Hiwi) and higher among the Mucujai Yanomamo (Fig. 2B). Based on the GM analysis, all six groups show higher MRDT values in the later period (Fig. 2C). Four of the six groups show decreases in $m'_{60–70}$ with acculturation (Fig. 2D). There is no difference in $m'_{60–70}$ for the Herero whereas the Yanomamo show an increase in the later period. Although the majority of these within-population results are consistent with Williams's hypothesis, none of the relationships is statistically significant (Table A1).

SWEDEN AND ENGLAND/WALES

Figure 3 shows the temporal sequence of Weibull-based m_0, ω, and MRDT in Sweden (Fig. 3A) and England/Wales (Fig. 3B). Extrinsic mortality (m_0) shows no consistent pattern before the 1770 cohort in Sweden, then increases until 1800, and declines steadily thereafter. Extrinsic mortality in England and Wales shows a similar trend as in Sweden during the same time period starting in the 1840 cohort ($r = 0.87$, $P < 0.0001$ for both males and females). The inconsistency in extrinsic mortality before 1800 in Sweden is likely due to periods of high mortality due to famine and epidemics (Hofsten and Lundström 1976) although data quality is poorest for pre-19th century. Although extrinsic mortality was similar between Swedish and English males (parameter estimate $\beta = 1.075$ from regression of Swedish m_0 on English m_0), mortality among Swedish females was substantially higher than that of English females ($\beta = 1.892$). Male extrinsic mortality exceeds female extrinsic mortality in both nations, especially during late 19th and early 20th century. Comparison of the slopes from regressions starting with the 1795 Swedish birth cohort onward reveals that male m_0 declines at about 1.7 times the rate of female m_0 ($\beta = -9.1 \times 10^{-4}$ for males, $\beta = -5.4 \times 10^{-4}$ for females per decade). Swedish survivorship appears to have become increasingly more favorable over time for men than women. In England/Wales, the rate of decline in m_0 progressed at about the same rate for men and women (0.0008 per decade).

In Sweden, MRDT for men and women appears to decline from about 10 years during pre-19th century, remaining relatively flat at about 8 years until the end of the 19th century birth cohort, then declining again, especially among women (Fig. 3A). MRDTs are fairly similar among Swedish men and women until the 20th century when female MRDT drops precipitously toward 6 years. In England/Wales, MRDT remains unchanged for men at just under 9 years but declines at about 0.25 years per decade among women to below 7 years (Fig. 3B). Ricklefs's ω (from Weibull) shows a trajectory similar to MRDT and these two measures are positively correlated (e.g., Pearson's $r = 0.95$ for Swedish men and $r = 0.97$ for women), producing the contradictory result of more rapid aging over time according to MRDT and slower aging according to ω. Although MRDT is mostly flat among English/Welsh male cohorts, ω decreases slightly (6%) over the 70-year cohort span. ω decreases by about 11% for women over the same time period.

Values of $m'_{60–70}$ from Weibull and GM models are highly correlated ($r > 0.99$ for men and women from both Sweden and England/Wales) and so we present in Figure 4 temporal trends in $m'_{60–70}$ based only on the Weibull model. $m'_{60–70}$ declines steadily from the mid-18th century at a rate of 0.0008 per decade for male and female birth cohorts in Sweden and male cohorts in England/Wales. English/Welsh females exhibit a steeper decline of 0.0013 per decade. Although patterns of decline are roughly similar for both sexes in both countries, English/Welsh men show the highest absolute rate of mortality increase during their 60s in all cohorts. Differences in $m'_{60–70}$ among Swedish men and women are small until late 19th century birth cohorts, when old-age mortality then declined more rapidly among women than men. For both sexes and nations, $m'_{60–70}$ is strongly positively correlated with ω ($r > 0.95$) and somewhat less so with MRDT ($r > 0.82$). Due to relatively small standard error, most of these documented changes in European aging measures are statistically significant (Table A3). Actuarial aging appears more rapid among the English/Welsh than Swedish when comparing within the same birth cohorts, although the differences are small for ω and $m'_{60–70}$. Larger ω and $m'_{60–70}$ values suggest more rapid actuarial aging in England/Wales than in Sweden, although English/Welsh

Table 1. Parameter estimates for Weibull and Gompertz–Makeham (GM) models of adult mortality among subsistence societies. Aging measures include Ricklefs's ω, mortality rate doubling time (MRDT), and the slope of mortality from ages 60 to 70, m'_{60-70}. Extrinsic mortality here is given as the lowest mortality rate across the life span, $m_{0,low}$, and the model-based estimates, m_0. Bold cells emphasize the subgroup averages for hunter-gatherers (HGs), acculturated HGs, and forager-horticulturalists.

		Weibull					Gompertz–Makeham					
Group	$m_{0,low}$	m_0	α	β	ω	m'_{60-70}	m_0	m_g	γ	ω	MRDT	m'_{60-70}
Hadza	0.004	0.010	7.47×10^{-17}	8.02	0.016	0.0018	0.009	1.70×10^{-05}	0.113	0.0014	6.11	0.0017
Ache (forest)	0.010	0.011	1.25×10^{-11}	5.26	0.018	0.0024	0.010	1.49×10^{-04}	0.087	0.0036	7.98	0.0024
!Kung	0.006	0.011	6.62×10^{-12}	5.45	0.018	0.0029	0.008	4.44×10^{-04}	0.074	0.0057	9.43	0.0027
Hiwi	0.008	0.022	6.39×10^{-33}	17.13	0.017	0.0053	0.022	2.54×10^{-09}	0.264	0.0000	2.62	0.0051
HG average	**0.007**	**0.012**	$\mathbf{7.85 \times 10^{-12}}$	**5.37**	**0.018**	**0.0035**	**0.010**	$\mathbf{2.28 \times 10^{-04}}$	**0.080**	**0.0043**	**8.63**	**0.0035**
Hiwi (post-contact)	0.010	0.011	8.59×10^{-13}	5.78	0.017	0.0016	0.011	4.50×10^{-05}	0.098	0.0021	7.09	0.0015
Ache (reservation)	0.002	0.003	8.12×10^{-12}	5.24	0.017	0.0015	0.002	1.15×10^{-04}	0.083	0.0031	8.35	0.0014
Aborigines	0.002	0.003	4.6×10^{-10}	4.39	0.018	0.0021	0.000	7.16×10^{-04}	0.063	0.0067	10.98	0.0020
Kung 1963–74	0.003	0.001	2.79×10^{-11}	5.03	0.018	0.0020	−0.003	5.44×10^{-04}	0.066	0.0060	10.53	0.0019
Agta (peasant)	0.005	0.005	1.67×10^{-06}	2.64	0.026	0.0037	0.014	2.61×10^{-08}	0.252	0.0001	2.75	0.0237
Acculturated HG avg.	**0.004**	**0.006**	$\mathbf{1.33 \times 10^{-10}}$	**4.66**	**0.018**	**0.0027**	**0.004**	$\mathbf{4.00 \times 10^{-04}}$	**0.070**	**0.0053**	**9.90**	**0.0027**
Gainj	0.007	0.010	1.27×10^{-07}	3.31	0.025	0.0053	0.002	3.22×10^{-03}	0.059	0.0137	11.85	0.0063
Tsimane (1950–1989)	0.007	0.010	5.53×10^{-17}	8.26	0.018	0.0037	0.010	1.50×10^{-05}	0.125	0.0014	5.55	0.0033
Tsimane (1990–2002)	0.001	0.005	1.49×10^{-14}	6.70	0.016	0.0014	0.004	3.10×10^{-05}	0.100	0.0018	6.92	0.0013
Yanomamo (Mucajai, precontact)	0.006	0.013	6.22×10^{-14}	6.40	0.016	0.0016	0.013	3.10×10^{-05}	0.103	0.0018	6.76	0.0015
Yanomamo (Mucajai, post-contact)	0.003	0.010	6.17×10^{-13}	6.14	0.019	0.0052	0.008	1.46×10^{-04}	0.098	0.0038	7.08	0.0051
Yanomamo (Neel)	0.014	0.031	5.96×10^{-16}	7.62	0.017	0.0027	0.030	3.00×10^{-05}	0.110	0.0018	6.32	0.0024
Forager-horticulturist avg.	**0.006**	**0.011**	$\mathbf{5.81 \times 10^{-15}}$	**7.14**	**0.018**	**0.0058**	**0.010**	$\mathbf{4.30 \times 10^{-05}}$	**0.109**	**0.0022**	**6.38**	**0.0057**

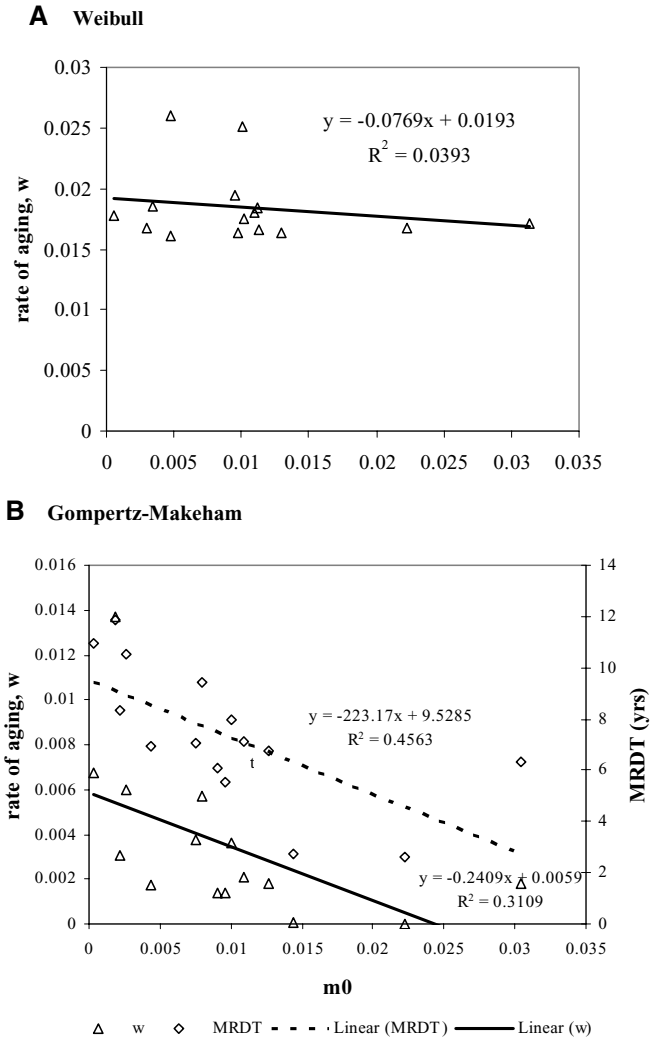

Figure 1. **Rates of aging as a function of extrinsic mortality among subsistence populations, using (A) Weibull model and (B) Gompertz–Makeham (GM) model.**

MRDT is greater than Swedish by over one year, suggesting the opposite.

Does m_0 track changes in MRDT, ω, and $m'_{60–70}$? Table 2 reports the results of regressions of each aging measure on model-based m_0 in Sweden and England/Wales. MRDT does not vary with m_0 in any of the analyses in Table 2, nor does ω with m_0 for three of the four GM analyses. However, ω (Weibull) and $m'_{60–70}$ (both GM and Weibull) are both significantly positively related to m_0. Although restricting Swedish cohorts to those after 1795 reduces some of the variation and improves R^2 in all of the regressions, it does not significantly alter the parameter estimates nor make the GM analyses statistically significant. We also performed the same regressions using period data for Sweden. Based on analyses of period data (not shown), we find the same significant relationships but with stronger correlations between aging

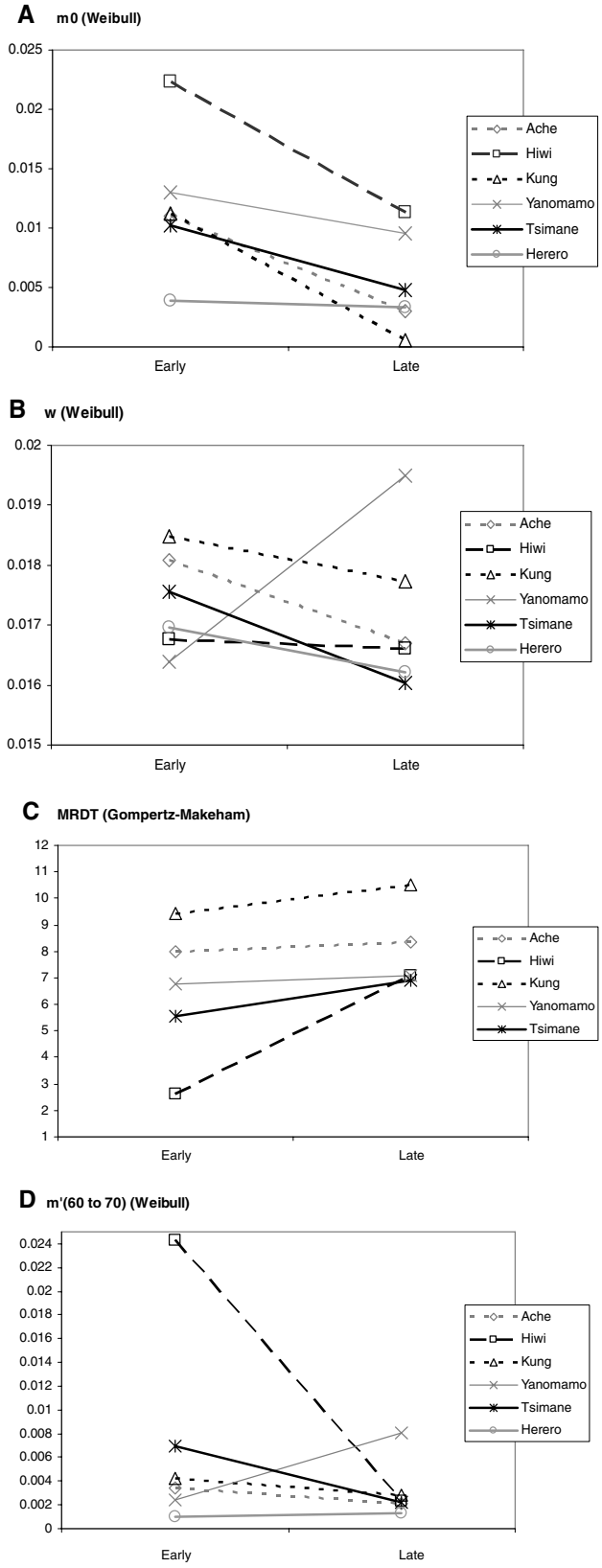

Figure 2. **Comparing extrinsic mortality (m_0) and aging measures (ω, MRDT, and $m'_{60–70}$) between early periods of little to no outside contact and later periods of contact or acculturation among a subsample of subsistence populations.**

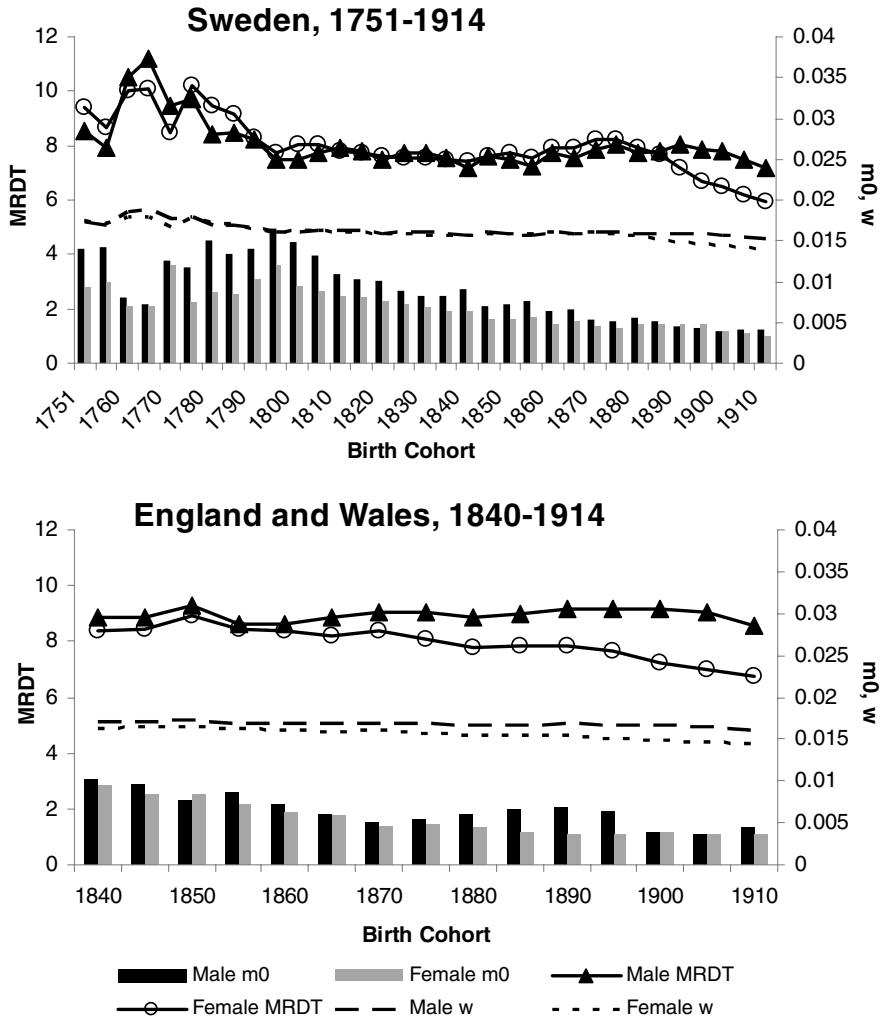

Figure 3. Trends in extrinsic mortality (m_0) and aging measures, ω and MRDT, among men and women for (A) Swedish cohorts, 1751–1914 and (B) England/Wales, 1840–1914.

measures (ω, $m'_{60–70}$) and m_0. Actuarial aging derived from period data reflects senescence experienced during a 5 year time interval, instead of by a cohort of individuals.

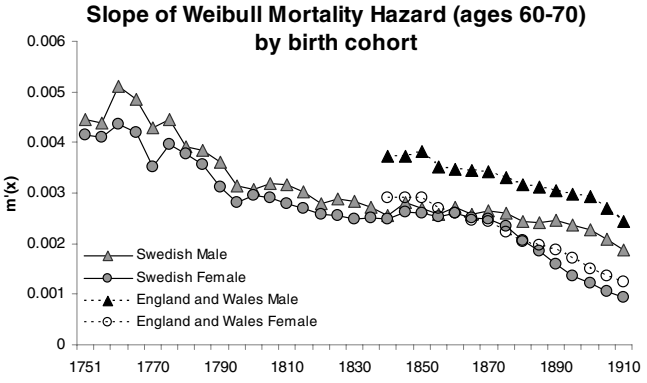

Figure 4. Trends in our third aging measure, $m'_{60–70}$, for Swedish and English/Welsh cohorts.

Figure 5 illustrates several of the typical regressions from Table 2 using examples for each of the aging measures. Ricklefs's ω declines about three times more rapidly among both Swedish and English/Welsh women than among men with each decrement in m_0 (Weibull). The rate of decline in $m'_{60–70}$ with decreases in m_0 was about 1.5–2 times greater in women than in men.

COMPARISON OF SUBSISTENCE AND EUROPEAN SOCIETIES

How does actuarial aging in small-scale subsistence societies compare with that of Sweden and England/Wales? Aging measures among subsistence societies and the earliest Swedish cohorts are similar, yet diverge when comparing across successive European cohorts that show progressively slower aging (Table A2). For example, the difference in ω increases fourfold and in $m'_{60–70}$ increases about threefold (based on Weibull) over the 150 years. Differences in MRDT are less consistent and show

Table 2. Estimates of slope parameter from regressions of aging parameters on extrinsic mortality, done for male and female cohorts from Sweden and England/Wales using both Gompertz–Makeham and Weibull models.

	Gompertz–Makeham, Males						Gompertz–Makeham, Females					
	Sweden			England/Wales			Sweden			England/Wales		
	MRDT	ω	$m'_{60–70}$	MRDT	ω	$m'_{60–70}$	MRDT	ω	$m'_{60–70}$	MRDT	ω	$m'_{60–70}$
β	−2	0.057	0.131	−49	0.039	0.144	130	0.240	0.216	231	0.406	0.289
P-value	0.9654	0.5618	0.0019	0.2211	0.5899	0.0268	0.2770	0.19333	0.0190	0.0646	0.0189	0.0030
Pearson correlation	0.008	0.105	0.521	0.336	0.152	0.569	0.195	0.232	0.406	0.489	0.596	0.710
	Weibull, MALES						Weibull, FEMALES					
	Sweden			England/Wales			Sweden			England/Wales		
	MRDT	ω	$m'_{60–70}$	MRDT	ω	$m'_{60–70}$	MRDT	ω	$m'_{60–70}$	MRDT	ω	$m'_{60–70}$
β	–	0.094	0.133	–	0.093	0.157	–	0.267	0.266	–	0.264	0.247
P-value	–	0.0172	0.0002	–	0.0117	0.0008	–	1.32E−04	9.53E−06	–	5.44E−05	6.17E−06
Pearson correlation	–	0.412	0.607	–	0.631	0.771	–	0.617	0.688	–	0.852	0.896

little progressive change in comparisons of different cohorts with subsistence societies.

Discussion

We have shown mixed evidence against the null prediction of no geographical or temporal differences in actuarial aging (Predictions 1 and 2). Results vary depending on the specific aging measure used and the statistical model upon which parameter estimation is based. Among small-scale subsistence populations, there is strong evidence that extrinsic mortality risk declined following acculturation and integration into national society, and modest cross-sectional evidence (based only on MRDT) that higher extrinsic mortality associates with faster rates of actuarial aging. The diachronic comparisons in six groups show more support for the notion that actuarial aging rates decline with acculturation.

Actuarial aging rates among small-scale subsistence populations overlap with those from early Swedish cohorts and to some extent with the earliest cohorts in England/Wales. Contrary to popular claims (Vallois 1961; Weiss 1981), Williams (1957), and paleodemographic reconstructions of prehistoric agriculturalists (e.g., Lovejoy et al. 1977), hunter–gatherers surviving beyond reproductive maturity do not have exceptionally short life spans nor do they age more rapidly than most pre-industrial human populations (Blurton Jones et al. 2002; Gurven and Kaplan 2007).

Actuarial aging has slowed over time in Sweden and England/Wales although not in continuous steady decline. It appears that an initial decline occurred for individuals born during the 18th century followed by a century of relatively little change, then a greater decline for those born toward the end of the 19th century. Because the time span of the English cohort sample is smaller than the Swedish, beginning only in 1840, the temporal decline in English/Welsh aging rates is a more uniform decrease than that observed in Sweden.

Aging rates are highly correlated with extrinsic mortality rate in both nations for two of the three aging measures, consistent with Williams's hypothesis. MRDT is significantly related to m_0 only among subsistence societies. The stronger test focusing on longitudinal trends within two nations does not lend support to Williams's hypothesis based on MRDT, but does for ω and $m'_{60–70}$. We have shown that MRDT varies less across changing conditions, whereas ω and $m'_{60–70}$ are not species-typically invariant, but vary predictably among and within populations over time. These patterns are more consistently revealed with the Weibull than with the GM model (Table 2), perhaps due to the inseparability of extrinsic and intrinsic mortality components in the GM. Williams's hypothesis and subsequent treatments are silent about the magnitude of expected change in actuarial aging for each unit decrease in mortality, but Finch (1990) estimates that species-typical patterns should not vary by more than 25%. The variation in aging measures observed in this article falls within that range. To our knowledge, this is the first explicit test of Williams's hypothesis to be made using human mortality data.

Although our use of multiple aging measures and two estimation procedures (GM and Weibull) complicate any scenario concerning extrinsic mortality and actuarial aging, employing different approaches is an important exercise to obtain robust results.

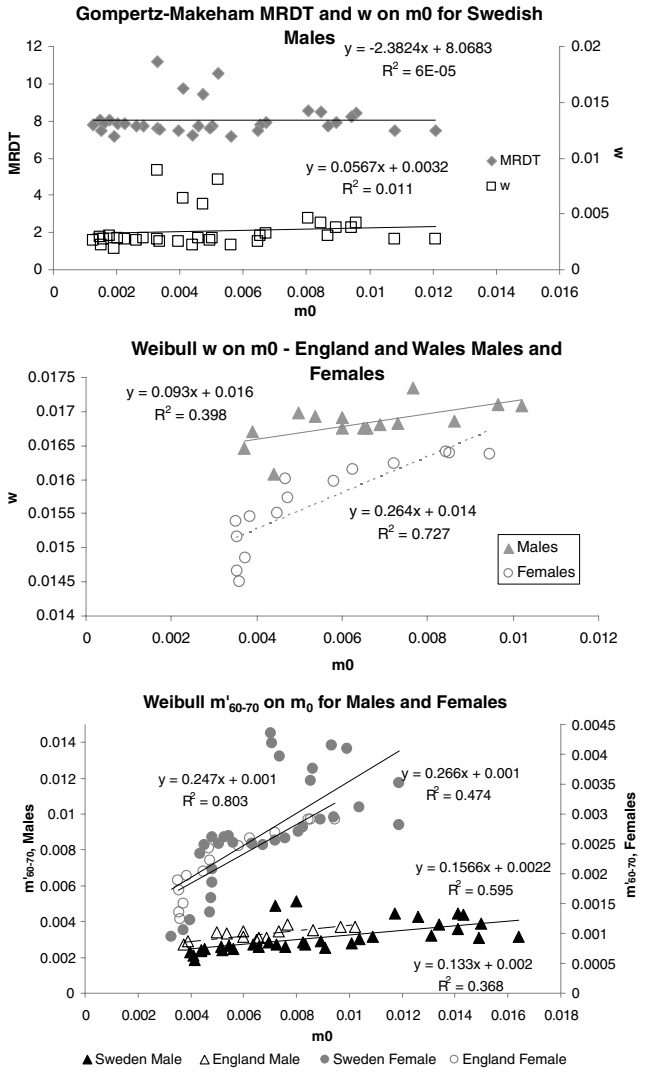

Figure 5. Relationship between aging measures and extrinsic mortality for a subset of the analyses presented in Table 2.

If we had used only the traditional MRDT from a GM model, we would have (1) found support for Williams's hypothesis among subsistence populations but not for the longitudinal trends in European nations, (2) concluded that aging has accelerated, rather than slowed down, in more recent cohorts, (3) found substantial variation in aging (> 2 year difference) among subsistence societies but little variation among sexes within cohorts (< 1 year) and among cohorts from the two nations (1–2 years).

Although our aging measures correlate strongly with one another, each tells a different story about the mortality increase at later ages. A noteworthy result is that MRDT "declines" over time in both Sweden and England/Wales and is not consistently related to extrinsic mortality rates. A similar decline in MRDT was also reported by Carnes et al. (1996) when comparing mortality rates over a 30-year period in Japan, the United States, The Netherlands, and Australia. Declines in MRDT over time have also been documented in association with mortality compression and the rectangularization of the survivorship function in several developed countries (Yashin et al. 2002b).

We suspect that declines in MRDT coupled with declines in ω and $m'_{60–70}$ may not be so incongruent. Declining extrinsic mortality and the compression of adult mortality and morbidity to older ages suggests a later age at the onset of intrinsic mortality. Temporal trends in the ages at which intrinsic mortality (i.e., ignoring m_0) reaches 0.01 and 0.02 show recent cohorts reaching these levels up to 15 years later than their ancestors born in the 1750s, and up to a decade later than those born in the 1840s (Fig. 6). Cohort improvements in the median age of death from intrinsic mortality (Carnes et al. 1996) reveal the same pattern. Mortality rates have decreased at all ages over the past two centuries but less so in absolute terms among those over age 50 (Kannisto et al. 1994, 1999). The later onset of intrinsic mortality increase coupled with a lower baseline adult mortality rate and fewer improvements in late age mortality means that the rate of mortality increase at later ages has accelerated over time. In other words, the mortality rate appears to accelerate and hence double more rapidly when baseline mortality is very low and adult mortality is increasingly compressed to later ages.

An alternative explanation for the positive relationship between m_0 and MRDT is that mortality declines over time have increased heterogeneity in underlying phenotypic condition (frailty) among survivors. Frailty heterogeneity has been an explanation for slower population-level actuarial aging at oldest ages (>90) (Vaupel et al. 1979), and for increases in morbidity (Crimmins 2001), but is perhaps unlikely to explain changes in earlier adult mortality, e.g., ages 40 to 60.

WHICH AGING MEASURE BEST REFLECTS SENESCENCE?

We agree with Bronikowski and Promislow (2005) and Williams et al. (2006) who argue that no single measure can accurately capture aging as conceptualized in the Williams hypothesis and that effort should be directed to understanding the effects of exogenous changes on fitness costs expressed at different ages (e.g., Pavard and Metcalf 2007). Our results based on the popular MRDT describe a pattern of more rapid senescence over time and with declines in m_0, although our other aging measures suggest the opposite. None of the aging measures we use here is derived from theory concerning the aging process but instead each is a statistical composite that describes some aspect of mortality increase based on a descriptive model. Ricklefs (1998) provides justification for why ω may be better than MRDT, especially in that MRDT relies on the problematic GM model. Our introduction of $m'_{60–70}$ was based on the intuition that the rate of increase at particular late ages may be more instructive than the absolute

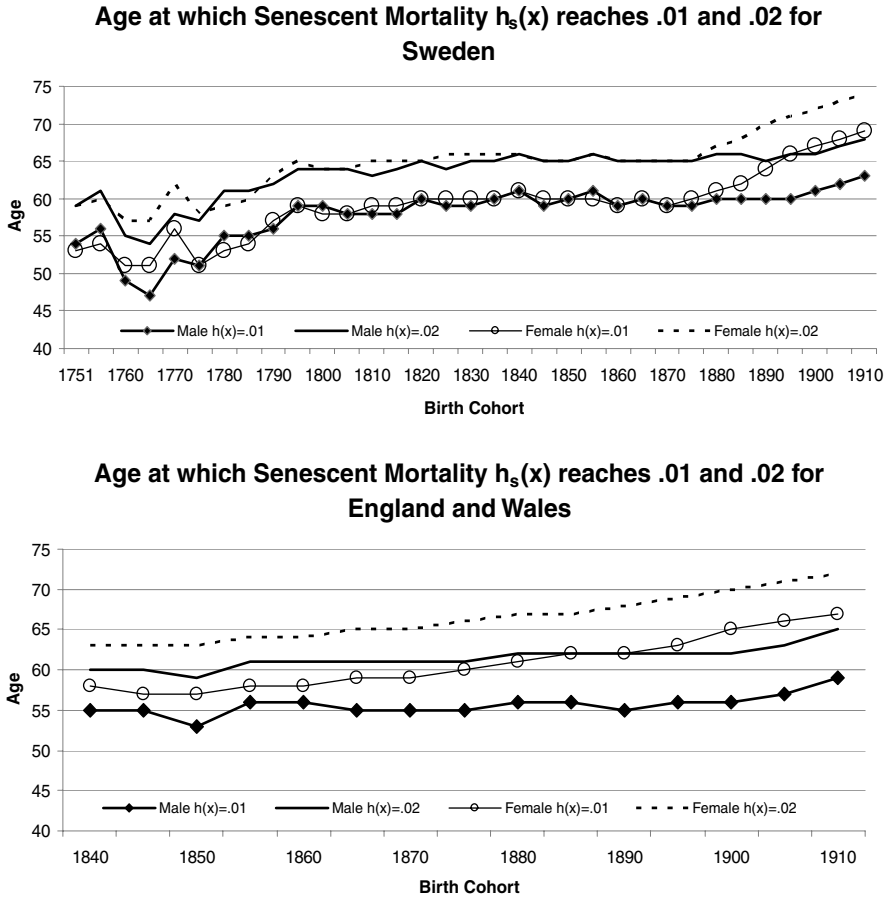

Figure 6. **The age at which senescent mortality component of the Weibull model reaches 0.01 and 0.02 among cohorts from (A) Sweden and (B) England/Wales.**

mortality rate itself. To a large extent, ω and $m'_{60–70}$ tell the same story in our analyses whereas proper interpretation of the MRDT results would require additional analysis to insure that they are not artifacts of the statistical estimation procedure. To the extent that MRDT provides the most rigid definition of aging (doubling time independent of absolute magnitude), MRDT may be useful for species-typical estimation and for cross-species comparison, but is unreliable as a summary measure of aging rate for the purpose of within-species comparison. Our results suggest that MRDT is a poor measure for evaluating secular trends in actuarial aging within populations.

EVIDENCE FOR SELECTION?

In fewer than 10 generations, we found changes in MRDT and ω that were somewhat modest in comparison to the changes in m_0, even though the association between ω and m_0 was strong. From the 1751 until 1910 cohorts there was a 68% decrease in Swedish m_0 and from 1840 to 1910 cohorts there was a 59% decrease in England/Wales m_0. However, MRDT decreased by 26% and 11% in Sweden and England/Wales, respectively, whereas ω declined by about 16% and 9%, and $m'_{60–70}$ showed a concomitant decline of 67% and 46%. Although the temporal decline in $m'_{60–70}$ appears to be linear (Fig. 4), changes in ω and MRDT seem mostly confined to cohorts born before 1800 and to those born after 1880 (Fig. 3).

Our results are mostly consistent with Williams's hypothesis, still we cannot evaluate whether the inferred slowing of the aging process is caused by the altered fitness costs and benefits that result from an increased tendency to survive from exogenous causes. The Williams's hypothesis does not specify the expected magnitude and timing of change in aging rates with declines in m_0. Rapid changes in extrinsic mortality may not have any evolutionary consequences on the aging process. It is likely that the time depth of our longitudinal data is too brief for adaptive genetic change to have occurred, although conclusive evidence would require an explicit population genetics model.

Explanations relating extrinsic mortality to aging rates via indirect effects of nutrition, infectious disease, and lifestyle at the individual level are complementary to the population-based evolutionary framework. Individual-level explanations point to proximate causal pathways that can explain the hypothesized change based on optimality-oriented thinking at the population

level. Improved conditions impacting individual lives likely lowered both extrinsic and intrinsic components of mortality. Extrinsic mortality, estimated by Carnes et al. (2006) using specific causes of death, declined by 30% from ages 15 to 65 between 1950 and 2000 in the United States, whereas intrinsic mortality decreased by 50% across all ages. Based on their classification scheme relying on specific causes of death, Carnes et al. (2006) find that extrinsic mortality is strongly age-dependent, contrary to the assumption of age-independence made here. However, the pathogenesis of many of the causes of death grouped together as extrinsic mortality (e.g., chronic liver diseases, kidney infections, inflammatory diseases of the urinary tract) likely involve "intrinsic" components that can influence age-related susceptibility, disease progression and fatality.

We briefly describe some proximate mechanisms that may explain how changes in mortality rates from specific causes may impact late-age survivorship. For cohorts born prior to 1800, trends in extrinsic mortality and aging rates are consistent with historical patterns documented for Finland (Kannisto et al. 1999), Sweden (Yashin et al. 2002a), and the United States (Costa and Steckel 1997) showing increases in mortality during the first half of the 19th century due to rapid urbanization, population growth, famines, and infectious disease (Fogel 1997). The secular decline in mortality in most Western countries began in the mid-19th century with improved hygiene and sanitation, as Omran's "age of pestilence and famine" drew to a close and as immunizations against common infectious diseases such as smallpox, measles, and pertussis became widespread. A later but smaller mortality decline coincided with antibiotic usage and improved medical intervention in the 20th century. Trends toward less physically demanding labor and earlier retirement during this time also led to reduced workplace trauma and fewer accident-related deaths in adulthood.

Reduced exposure to infection and trauma at the individual level can result in cascading effects over the life span that slow the aging process. Any decrease in disease exposure or wear-and-tear early in life, coupled with improvements in nutrition, can lead to greater allocation of bodily resources to cellular maintenance and repair functions. Overall mortality decline from reduced infections and their damaging sequela likely decreased late-age period mortality rates, and led to lower late-age mortality among subsequent cohorts. Crimmins and Finch (2006) and Finch and Crimmins (2004) have shown that early child mortality in Sweden, France, and England/Wales better predicts late-age cohort mortality than period rates, consistent with the hypothesis that early exposure to infectious conditions leads to more rapid senescence and higher mortality rates at late ages. Much of the reduction in late age mortality in the developed world during the second half of the 20th century was due to improved survival after diagnosis, rather than any substantial reduction in disease morbidity (Crimmins 2001). Kannisto et al. (1994) report that late 20th century declines in late-age mortality occurred simultaneously in 20 low mortality countries, suggesting that period factors may have overwhelmed localized cohort effects in recent years. Period effects, of course, are caused by contemporaneous interventions as well as by cohort-related changes. Additionally, recent cohorts in the United States and other countries have seen cohort declines in hypertension and period declines in smoking frequency, which also helped decrease mortality from cardiovascular disease (Lynch and Davey Smith 2005).

A coordinated and developed inflammatory response is adaptive in the infectious environments that characterized most of human history, even before the rise of agriculture (Pearce-Duvet 2006; Finch 2007). Chronic activation of the innate immune response, however, may carry pleiotropic costs late in life that foster the onset and development of chronic diseases. Inflammation has been linked to many chronic diseases of old age, such as atherosclerosis, vascular disease, diabetes, metabolic syndrome, Alzheimer's disease, and even cancer (see review in Finch 2007). Chronic inflammation likely decreased over time in parallel with reductions in mortality from infectious disease and with improvements in nutrition. Chronically elevated blood levels of inflammatory cells and proteins are independent factors involved in the atherosclerotic process and in immune system dysregulation, therefore reductions in lifetime levels of inflammation should slow cardiovascular and immune system deterioration with aging and delay the onset of many chronic diseases of old age. This proximate explanation is logically similar to the evolutionary model, in that a reduced assault rate decreases wear and tear on many internal systems and frees up energy for improved growth, repair, and immune function. These changes in energetic allocation support increased maintenance and improved health status and survivorship at older ages.

SEX DIFFERENCES?

We have shown that females display lower rates of actuarial aging than males and that temporal change in aging differs by sex. This slower female aging is characterized by lower values of ω, m'_{60-70}, and MRDT. Men and women reach any specific level of senescent mortality at similar ages for all cohorts up until those born in 1880 in Sweden (Fig. 6). The age gap increases thereafter, so that by the final 1910 cohort, women reach the same level of senescent mortality about 8 years later than do men in both Sweden and England/Wales. Certainly male extrinsic mortality has been more affected by wars (although less so in Sweden than in England) and by cigarette smoking, a prominent risk factor for lung cancer and cardiovascular disease (Godtfredsen et al. 2002). Extrinsic mortality was higher among men than women in all cohorts although the gap narrowed considerably over time (Fig. 3). Thus, although the differential in extrinsic mortality

rates has narrowed among the sexes, our results show that actuarial aging rates have diverged (Figs. 3 and 4). If these mortality patterns reflect underlying biological condition, then our results are at odds with the growing consensus that women display greater frailty (i.e., increased risk or vulnerability reflecting multisystem physiological change) than do men (Mitnitski et al. 2005).

Conclusion

In a recent review of biodemographic approaches to mortality modeling, Carnes and colleagues (2006) state that "a perfect classification of causes [of death] into extrinsic and intrinsic categories is not attainable" (196), yet they conclude that imperfect partitions provide more useful insight into senescence than exclusively focusing on total mortality rate. Despite some problems with the partitioning of mortality into age-independent (extrinsic) and age-dependent (intrinsic) components, our approach retains theoretical coherence with evolutionary approaches, allows comparability among primates and other mammals, and does not require information on causes of death. Rather than assuming independence between these two forms of mortality, we recognize that at the individual level, changes in exogenous causes, however defined, likely affect endogenous biological processes; they can influence investments in maintenance and repair capabilities and thereby impact rates of physiological decline later in life.

Declines in actuarial aging over the past several hundred years may be modest but are nontrivial and likely related to declines in age-independent mortality. These declines may not be linked through selective changes in gene frequencies, but instead illustrate the potential for phenotypic plasticity built into the human genome for delaying the onset of senescent mortality increase. Environmental change and medical interventions that alter the pathogenesis of intrinsic disease may be responsible for some of the changes in late-age mortality reported here. However, if interventions only delay senescent mortality without changing the onset of symptoms or disease, the gap between rates of physiological and actuarial senescence will widen. Despite the temporal changes in actuarial senescence reported in this article, it remains to be seen whether physiological senescence is similarly amenable to substantial improvement.

The selective context that shaped the "probabilistic expiration dates" for the "biological equivalent of a warranty period" in individual human lives (Carnes and Olshansky 2007:374) occurred during a long evolutionary history as hunter–gatherers. Despite having short mean life expectancies at birth, extant hunter–gatherers show adult mortality profiles and modal ages of adult death similar to those of preindustrial Europe (Gurven and Kaplan 2007), and here we showed that actuarial aging rates are also similar. It appears that few hunter–gatherers, forager–farmers, or preindustrial Europeans lived more than eight decades. These results suggest that similar age distributions of adult deaths may occur under a relatively broad range of environmental conditions. The same set of defenses and repair mechanisms that evolved to be phenotypically plastic in relation to ancestral environmental variation now permits a new distribution of deaths under modern conditions; modal age of adult death for most industrialized countries now surpasses eight decades (Kannisto 2001).

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LITERATURE CITED

Abrams, P. A. 1991. The fitness costs of senescence: the evolutionary importance of events in early adult life. Evol. Ecol. 5:343–360.

———. 1993. Does increased mortality favor the evolution of more rapid senescence? Evolution 47:877–887.

Austad, S. N. 1992. Primate longevity: its place in the mammalian scheme. Am. J. Primatol. 28:251–261.

Austad, S. N., and K. E. Fischer. 1991. Mammalian aging, metabolism, and ecology: evidence from the bats and marsupials. J. Gerontol. 46:47–51.

Barker, D. 1994. Mothers, babies, and disease in later life. BMJ Publishing, London.

Bishop, Y. M. M., S. E. Fienberg, and P. W. Holland. 1975. Discrete multivariate analysis. MIT Press, Cambridge.

Blurton Jones, N. B., K. Hawkes, and J. O'Connell. 2002. The antiquity of postreproductive life: are there modern impacts on hunter-gatherer postreproductive lifespans? Hum. Biol. 14:184–205.

Bronikowski, A. M., and D. E. L. Promislow. 2005. Testing evolutionary theories of aging in wild populations. Trends Ecol. Evol. 20:271–273.

Bryant, M. J., and D. N. Reznick. 2004. Comparative studies of senescence in natural populations of guppies. Am. Nat. 163:55–68.

Calder, W. A., III. 1984. Size, function and life-history. Harvard Univ. Press, Cambridge, MA.

Carnes, B. A., and S. J. Olshansky. 1997. A biologically motivated partitioning of mortality. Exp. Gerontol. 32:615–631.

———. 2007. A realist view of aging, mortality and future longevity. Pop. Dev. Rev. 33:367–381.

Carnes, B. A., S. J. Olshansky, and D. Grahn. 1996. Continuing the search for a law of mortality. Pop. Dev. Rev. 22:231–264.

Carnes, B. A., L. R. Holden, S. J. Olshansky, T. M. Witten, and J. S. Siegel. 2006. Mortality partitions and their relevance to research on senescence. Biogerontology 7:183–198.

Case, A., and C. Paxson. 2005. Sex differences in morbidity and mortality. Demography 42:189–214.

Charlesworth, B. 1980. Evolution in age-structured populations. Cambridge Univ. Press, Cambridge, UK.

———. 1993. Evolutionary mechanisms of senescence. Genetica 91:11–19.

Cheung, S. L. K., J.-M. Robine, E. Jow-Ching, and G. Caselli. 2005. Three dimensions of the survival curve: horizontalization, verticalization and longevity extension. Demography 42:243–258.

Cichón, M. 1997. Evolution of longevity through optimal resource allocation. Proc. R. Soc. Lond. B 264:1383–1388.

Costa, D. L. 2000. Understanding the twentieth century decline in chronic conditions among older men. Demography 37:53–72.

———. 2002. Changing chronic disease rates and long-term declines in functional limitation among older men. Demography 39:119–137.

Costa, D. L., and R. H. Steckel. 1997. Long-term Trends in Health, Welfare, and Economic growth. Pp. 47–89 *in* R. H. Steckel, and R. Floud, eds. Health and welfare during industrialization. Univ. of Chicago Press, Chicago.

Crimmins, E. 2001. Mortality and health in human life spans. Exp. Gerontol. 36:885–897.

Crimmins, E. M. 2004. Trends in the health of the elderly. Annu. Rev. Public Health 25:79–98.

Crimmins, E. M., and C. E. Finch. 2006. Infection, inflammation, height, and longevity. Proc. Natl. Acad. Sci. USA 103:498–503.

Dudycha, J. 2001. The senescence of Daphnia from risky and safe habitats. Ecol. Letts. 4:102–105.

Edney, E. B., and R. W. Gill. 1968. Evolution of senescence and specific longevity. Nature 220:281–282.

Elo, I. T., and S. H. Preston. 1992. Effects of early-life conditions on adult mortality: a review. Population Index 58:186–212.

Finch, C. E. 1990. Longevity, senescence and the genome. Univ. of Chicago Press, Chicago.

Finch, C. 2007. The biology of human longevity. Academic Press, San Diego, CA.

Finch, C. E., and E. M. Crimmins. 2004. Inflammatory exposure and historical changes in human life-spans. Science 305:1736–1739.

Finch, C. E., M. C. Pike, and M. Whitten. 1990. Slow mortality rate accelerations during aging in animals approximate that of humans. Science 249:902–905.

Fogel, R. W. 1997. New findings on secular trends in nutrition and mortality: some implications for population theory. Pp. 435–486 *in* M. R. Rosenzweig, and O. Stark, eds. Handbook of population and family economics Elsevier science, Amsterdam.

Fogel, R. W., and D. L. Costa. 1997. A theory of technophysio evolution, with some implications for forecasting population, health care costs and pension costs. Demography 34:49–66.

Fogel, R. W., D. L. Costa, and J. M. Kim. 1993. Secular trends in the distribution of chronic conditions and disabilities at young adult and late ages, 1860–1988: some preliminary findings. National Bureau of Economic Research (NBER) Summer Institute, Economics of Aging Program, Cambridge, MA.

Freedman, V. A., L. G. Martin, and R. F. Schoeni. 2002. Recent trends in disability and functioning among older adults in the United States: a systematic review. J. Am. Med. Assoc. 288:3137–3146.

Fries, J. F. 1980. Ageing, natural death and the compression of morbidity. New Engl. J. Med. 303:130–136.

Fries, J. F. 1989. The compression of morbidity: near or far? Milbank Quarterly 67:208–232.

Gage, T. B. 1989. Bio-mathematical approaches to the study of human variation in mortality. Yearbook Phys. Anthropol. 32:185–214.

———. 2005. Are modern environments really bad for us?: revisiting the demographic and epidemiologic transitions. Yearbook Phys. Anthropol. 48:96–117.

Glei, D., H. Lundström, and J. Wilmoth. 2007. About mortality data for Sweden. Human Mortality Database, Berkeley, CA.

Gluckman, P. D., and M. A. Hanson. 2006. Developmental origins of health and disease. Cambridge Univ. Press, Cambridge.

Godtfredsen, N. S., C. Holst, E. Prescott, J. Vestbo, and M. Osler. 2002. Smoking reduction, smoking cessation, and mortality: a 16-year follow-up of 19,732 men and women from The Copenhagen Centre for Prospective Population Studies. Am. J. Epidemiol. 156:994–1001.

Greaves, M. 2000. Cancer: the evolutionary legacy. Oxford Univ. Press, Oxford.

Gurven, M., and H. Kaplan. 2007. Longevity among hunter-gatherers: a cross-cultural comparison. Popul. Dev. Rev. 33:321–365.

Gurven, M., H. Kaplan, E. Crimmins, C. Finch, and J. Winking. 2008. Lifetime inflammation in two epidemiological worlds: the Tsimane of Bolivia and the United States. J. Gerontol. 63A:196–199.

Hamilton, W. D. 1966. The molding of senescence by natural selection. J. Theor. Biol. 12:12–45.

Hayflick, L. 1994. How and why we age. Ballantine Books, New York, NY.

Hazzard, W. R. 1986. Biological basis of the sex differential in longevity. J. Am. Geriatr. Soc. 34:455–471.

Hofsten, E., and H. Lundström. 1976. Swedish population history. Main trends from 1750 to 1970. Statistiska Centralbyrån, Stockholm.

Holmes, D. J., and S. N. Austad. 1995. The evolution of avian senescence patterns: implications for understanding primary aging processes. Am. Zool. 35:307–317.

Human Mortality Database. University of California, Berkeley (USA), and Max Planck Institute for Demographic Research (Germany). Available at www.mortality.org or www.humanmortality.de (Accessed January 2007).

Idler, E. L., and Y. Benyamini. 1997. Self-rated health and mortality: a review of twenty-seven community studies. J. Health Social Behav. 38:21–37.

Kannisto, V. 2001. Mode and dispersion of the length of life. Population: An English Selection 13:159–171.

Kannisto, V., J. Lauritsen, R. A. Thatcher, and J. W. Vaupel. 1994. Reductions in mortality at advanced ages: several decades of evidence from 27 countries. Popul. Dev. Rev. 20:793–810.

Kannisto, V., M. Nieminen, and O. Turpeinen. 1999. Finnish life tables since 1751. Demographic Research 1.

Kapahi, P., M. E. Boulton, and T. B. L. Kirkwood. 1999. Positive correlation between mammalian life spans and cellular resistance to stress. Free Radic. Biol. Med. 26:495–500.

Keller, L., and M. Genoud. 1997. Extraordinary lifespans in ants: a test of evolutionary theories of aging. Nature 389:958–1060.

Kirkwood, T. B. L. 2002. Evolution of ageing. Mech. Age. Dev. 123:737–745.

Kirkwood, T. B. L., and R. Holliday. 1979. The evolution of ageing and longevity. Proc. R. Soc. Lond. B 205:531–546.

Lovejoy, C. O., R. S. Meindl, T. R. Pryzbeck, T. S. Barton, K. G. Heiple, and D. Kotting. 1977. Paleodemography of the Libben site, Ottawa County, Ohio. Science 198:291–293.

Lynch, J., and G. Davey Smith. 2005. A life course approach to chronic disease epidemiology. Annu. Rev. Public Health 26:1–35.

Magee, R. 1998. Arterial disease in antiquity. Med. J. Australia 169:663–666.

Manton, K. G., L. Corder, and E. Stallard. 1997. Changes in the age dependence of mortality and disability: cohort and other determinants. Demography 34:135–157.

Martin, G. M., S. N. Austad, and T. E. Johnson. 1996. Genetic analysis of aging: role of oxidative damage and environmental stresses. Nat. Genet. 13:25–34.

McClearn, G. 1997. Biomarkers of age and aging. Exp. Gerontol. 32:87–94.

McKeown, T. 1976. The modern rise of population. Academic Press, New York.

Milne, E. M. G. 2006. When does human ageing begin? Mech. Age. Dev. 127:290–297.

Mitnitski, A., X. Song, I. Skoog, G. A. Broe, J. L. Cox, E. Grunfeld, and K. Rockwood. 2005. Relative fitness and frailty of elderly men and women in developed countries and their relationship with mortality. J. Am. Geriatr. Soc. 53:2184–2189.

Nemetz, P. N., V. L. Roger, J. E. Ransom, K. R. Bailey, W. D. Edwards, and C. L. Leibson. 2008. Recent trends in the prevalence of coronary disease: a population-based autopsy study of nonnatural deaths. Arch. Int. Med. 168:264–270.

NIA/NIH. 2007. Why population aging matters: a global perspective. National Institutes of Health, Department of State and Department of Health and Human Services, Washington, DC

Oeppen, J., and J. W. Vaupel. 2003. Broken limits to life expectancy. Science 296:1029–1031.

Ogburn, C. E., S. N. Austad, D. J. Holmes, J. V. Kiklevich, K. Gollahon, P. S. Rabinovitch, and G. M. Martin. 1998. Cultured renal epithelial cells from birds and mice: enhanced resistance of avian cells to oxidative stress and DNA damage. J. Gerontol. 53:B287–B292.

Partridge, L. 1989. Lifetime reproductive success and life history evolution. Pp. 421–440 *in* I. Newton, ed. Lifetime reproduction in birds. Academic Press, New York, NY.

Partridge, L., and N. H. Barton. 1993. Optimality, mutation and the evolution of ageing. Nature 362:305–311.

Pavard, S., and C. J. E. Metcalf. 2007. Negative selection on BRCA1 susceptibility alleles sheds light on the population genetics of late-onset diseases and aging theory. PLoS ONE 2:e1206.

Pearce-Duvet, J. M. C. 2006. The origin of human pathogens: evaluating the role of agriculture and domestic animals in the evolution of human disease. Biol. Rev. 81:369–382.

Pearl, R., and J. R. Miner. 1935. Experimental studies on the duration of life. XIV. The comparative mortality of certain lower organisms. Q. Rev. Biol. 10:60–79.

Plutsky, J. 2001. Inflammatory pathways in atherosclerosis and acute coronary syndromes. Am. J. Cardiol. 88:10K–15K.

Preston, S. H. 1976. Mortality patterns in national populations, with special reference to recorded causes of death. Academic Press, New York.

Promislow, D. E. L. 1991. Senescence in natural populations of mammals: a comparative study. Evolution 45:1869–1887.

Promislow, D. E. L., M. Tatar, A. A. Khazaeli, and J. W. Curtsinger. 1995. Age-specific patterns of genetic variation in *Drosophila melanogaster*. I. Mortality. Genetics 143 839–848.

Ricklefs, R. W. 1998. Evolutionary theories of aging: confirmation of a fundamental prediction, with implications for the genetic basis and evolution of the life span. Am. Nat. 152:24–44.

Ricklefs, R., and A. Scheuerlein. 2001. Comparison of aging-related mortality among birds and mammals. Exp. Gerontol. 36:845–857.

———. 2002. Biological implications of the Weibull and Gompertz models of aging. J. Gerontol. Biol. Sci. 57A:B69–B76.

Rose, M. 1991. The evolutionary biology of aging. Oxford Univ. Press, Oxford.

Sacher, G. A. 1959. Relation of lifespan to brain weight and body weight in mammals. Pp. 115–133 *in* G. E. W. Wolstenhome, and M. O'Connor, eds. Ciba foundation colloquia on ageing. Churchill, London.

Sohal, R. S., H. H. Ku, and S. Agarwal. 1993. Biochemical correlates of longevity in two closely-related rodent species. Biochem. Biophys. Res. Commun. 196:7–11.

Stearns, S. C. 1992. The evolution of life histories. Oxford Univ. Press, Oxford.

Stearns, S. C., M. Ackermann, M. Doebeli, and M. Kaiser. 2000. Experimental evolution of aging, growth and reproduction in fruit flies. Proc. Natl. Acad. Sci. USA 97:3309–3313.

U.S. Census Bureau, P. D. 2001. U.S. Interim Projections by Age, Sex, Race, and Hispanic Origin. http://www.census.gov/ipc/www/usinterimproj/.

Vallois, H. V. 1961. The social life of early man: the evidence of skeletons. Pp. 214–235 *in* S. L. Washburn, ed. Social life of early man. Aldine de Gruyter, Chicago.

Vaupel, J. W., K. G. Manton, and E. Stallard. 1979. The impact of heterogeneity in individual frailty on the dynamics of mortality. Demography 16:439–454.

Weiss, K. M. 1981. Evolutionary perspectives on human aging. Pp. 25–28 *in* P. Amoss, and S. Harrell, eds. Other ways of growing old. Univ. Press, Stanford, CA.

Williams, G. C. 1957. Pleitropy, natural selection and the evolution of senescence. Evolution 11:398–411.

Williams, P. D., T. Day, Q. Fletcher, and L. Rowe. 2006. The shaping of senescence in the wild. Trends Ecol. Evol. 21:458–463.

Yashin, A. I., A. S. Begun, S. I. Boiko, S. V. Ukraintseva, and J. Oeppen. 2002a. New age patterns of survival improvement in Sweden: do they characterize changes in individual aging? Mech. Age. Dev. 123:637–647.

Yashin, A. I., S. V. Ukraintseva, S. I. Boiko, and K. G. Arbeev. 2002b. Individual aging and mortality rate: how are they related? Soc. Biol. 49:206–217.

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Appendix

Table A1. Differences in aging indicators among subsistence populations. Bold cells indicate lack of significance ($\alpha = 0.05$).

		Gompertz–Makeham			Weibull	
		ω	$m'_{60–70}$	MRDT	ω	$m'_{60–70}$
Macrocategories						
HGs	FHs	0.0021	−0.0009	2.2494	**0.0002**	**0.0010**
HGs	Acc HGs	−0.0010	**0.0004**	−1.2701	**0.0001**	**0.0014**
FHs	Acc HGs	−0.0031	0.0013	−3.5195	**−0.0001**	**0.0005**
Comparison populations						
Hadza	Ache forest	−0.0022	**−0.0006**	−1.8640	**−0.0018**	**−0.0006**
Hadza	Tsimane 1990–2002	**−0.0004**	**0.0005**	**−0.8121**	**0.0003**	**0.0004**
Hadza	Ache Reservation	−0.0017	**0.0003**	−2.2388	**−0.0004**	**0.0003**
Hadza	Kung 1963–1974	−0.0029	**−0.0008**	−4.4217	**−0.0014**	**−0.0002**
Hadza	Yanomamo, Mucajai, post-	−0.0024	**−0.0033**	**−0.9677**	**0.0006**	**0.0007**
Ache forest	Tsimane 1990–2002	0.0018	**0.0011**	**1.0518**	**0.0021**	**0.0011**
Ache forest	Ache Reservation	**0.0005**	**0.0010**	**−0.3748**	**0.0014**	**0.0010**
Ache forest	Kung 1963–1974	**−0.0007**	**−0.0002**	−2.5578	**0.0004**	**0.0004**
Ache forest	Yanomamo, Mucajai, post-contact	**−0.0002**	**−0.0027**	**0.8962**	**0.0023**	**0.0013**
Tsimane 1990–2002	Ache Reservation	−0.0013	**−0.0001**	**−1.4266**	**−0.0007**	**−0.0001**
Tsimane 1990–2002	Kung 1963–1974	−0.0025	**−0.0013**	−3.6096	**−0.0017**	**−0.0006**
Tsimane 1990–2002	Yanomamo, mucajai, post-contact	−0.0020	**−0.0038**	**−0.1556**	**0.0003**	**0.0002**
Ache Reservation	Kung 1963–1974	**−0.0012**	**−0.0012**	**−2.1830**	**−0.0010**	**−0.0005**
Ache Reservation	Yanomamo, Mucajai, post-contact	**−0.0007**	**−0.0037**	**1.2710**	**0.0010**	**0.0003**
Kung 1963–1974	Yanomamo, Mucajai, postcontact	**0.0005**	**−0.0025**	3.4540	**0.0020**	**0.0009**
Acculturation						
Hiwi (<1960)	Hiwi (1961–1989)	**−0.0021**	**0.0036**	−4.4701	**0.0002**	**0.0038**
Tsimane (1950–1989)	Tsimane (1990–2002)	**−0.0004**	**0.0021**	−1.3705	**0.0015**	**0.0023**
Ache (<1978)	Ache (1978–1993)	**0.0005**	**0.0010**	**−0.3748**	**0.0014**	**0.0010**
!Kung (<1963)	!Kung (1963–1974)	**0.0015**	**0.0001**	**−1.1036**	**0.0008**	**0.0009**
Yanom. (1930–1956)	Yanom. (1982–1996)	−0.0020	**−0.0036**	**−0.3243**	**0.0004**	**0.0006**

Table A2. **Differences in aging indicators between subsistence populations and European countries. Bold cells indicate lack of significance ($\alpha = 0.05$).**

		Gompertz–Makeham			Weibull	
		ω	$m'_{60–70}$	MRDT	ω	$m'_{60–70}$
Subsistence populations vs. Sweden (males)						
1751–1754	HGs	0.0003	0.0005	**−0.1062**	**−0.0007**	**−0.0008**
1751–1754	FHs	0.0025	**−0.0003**	2.1432	**−0.0005**	**0.0002**
1751–1754	Acc HGs	−0.0007	0.0009	−1.3763	**−0.0006**	**0.0007**
1800–1804	HGs	−0.0015	**−0.0004**	−1.1304	−0.0019	**−0.0017**
1800–1804	FHs	0.0006	−0.0012	1.1190	−0.0017	**−0.0008**
1800–1804	Acc HGs	−0.0026	**0.0000**	−2.4005	**−0.0018**	**−0.0003**
1850–1854	HGs	−0.0017	−0.0006	−1.1304	−0.0021	**−0.0020**
1850–1854	FHs	0.0004	−0.0015	1.1190	−0.0019	**−0.0010**
1850–1854	Acc HGs	−0.0027	**−0.0002**	−2.4005	−0.0021	**−0.0005**
1900–1904	HGs	−0.0017	−0.0009	−0.8525	−0.0023	**−0.0021**
1900–1904	FHs	0.0004	−0.0018	1.3969	−0.0021	**−0.0012**
1900–1904	Acc HGs	−0.0027	**−0.0005**	−2.1227	−0.0023	**−0.0007**
1910–1914	HGs	−0.0024	−0.0011	−1.4640	−0.0028	**−0.0024**
1910–1914	FHs	−0.0003	−0.0020	0.7854	−0.0026	**−0.0015**
1910–1914	Acc HGs	−0.0034	−0.0007	−2.7341	−0.0028	**−0.0010**
Subsistence populations vs. Eng/Wales (males)						
1840–1844	HGs	0.0004	**0.0001**	**0.2318**	**−0.0009**	**−0.0011**
1840–1844	FHs	0.0025	−0.0008	2.4812	**−0.0007**	**−0.0002**
1840–1844	Acc HGs	−0.0006	**0.0005**	−1.0383	**−0.0009**	**0.0003**
1870–1874	HGs	0.0004	**−0.0001**	0.4288	**−0.0010**	**−0.0013**
1870–1874	FHs	0.0025	−0.0010	2.6782	**−0.0008**	**−0.0003**
1870–1874	Acc HGs	−0.0006	**0.0003**	−0.8414	**−0.0010**	**0.0001**
1890–1894	HGs	0.0003	**−0.0003**	0.5125	**−0.0012**	**−0.0015**
1890–1894	FHs	0.0024	−0.0012	2.7619	−0.0010	**−0.0005**
1890–1894	Acc HGs	−0.0008	**0.0001**	−0.7577	**−0.0012**	**−0.0001**
1910–1914	HGs	−0.0008	−0.0007	**−0.0640**	−0.0019	**−0.0020**
1910–1914	FHs	0.0013	−0.0016	2.1854	−0.0017	**−0.0010**
1910–1914	Acc HGs	−0.0018	**−0.0003**	−1.3342	**−0.0019**	**−0.0005**

Table A3. Difference in three aging indicators, comparing Sweden against England/Wales by sex, longitudinal change within countries, and by sex within cohorts by country. Bold cells indicate lack of significance ($\alpha = 0.05$).

		Gompertz–Makeham			Weibull	
		ω	m'_{60-70}	MRDT	ω	m'_{60-70}
Comparison populations						
Males						
Sweden 1840–1844	England 1840–1844	−0.0025	−0.0008	−1.7032	−0.0014	−0.0009
Sweden 1870–1874	England 1870–1874	−0.0018	−0.0005	−1.2019	−0.0010	−0.0006
Sweden 1890–1894	England 1890–1894	−0.0016	−0.0004	−1.1033	−0.0009	−0.0005
Sweden 1910–1914	England 1910–1914	−0.0016	−0.0004	−1.3999	−0.0009	−0.0005
Females						
Sweden 1840–1844	England 1840–1844	−0.0012	−0.0003	−0.9783	−0.0006	−0.0004
Sweden 1870–1874	England 1870–1874	−0.0001	**0.0000**	−0.1790	**0.0000**	**0.0000**
Sweden 1890–1894	England 1890–1894	−0.0007	−0.0002	−0.6745	−0.0004	−0.0002
Sweden 1910–1914	England 1910–1914	−0.0006	−0.0002	−0.8024	−0.0004	−0.0002
Trends in Sweden (males)						
1751–1754	1800–1804	0.0019	0.0009	1.0242	0.0010	0.0012
1800–1804	1850–1854	0.0002	0.0003	**0.0000**	**0.0002**	**0.0002**
1850–1854	1900–1904	0.0000	0.0003	−0.2778	0.0002	0.0002
1900–1904	1910–1914	0.0007	0.0002	0.6114	0.0003	0.0005
Trends in England/Wales (males)						
1840–1844	1870–1874	**0.0000**	0.0002	−0.1970	**0.0001**	**0.0002**
1870–1874	1890–1894	0.0002	0.0002	−0.0837	**0.0002**	**0.0002**
1890–1894	1910–1914	0.0011	0.0004	0.5765	0.0007	0.0005
Sex differences Sweden (male–female)						
1751–1754	1751–1754	−0.0010	0.0001	−0.8664	−0.0003	**0.0000**
1800–1804	1800–1804	−0.0005	0.0001	−0.5489	**−0.0001**	**0.0000**
1850–1854	1850–1854	−0.0002	0.0000	−0.2172	**0.0000**	**0.0000**
1900–1904	1900–1904	0.0015	0.0007	1.2832	0.0008	0.0007
1910–1914	1910–1914	0.0011	0.0006	1.2080	0.0012	0.0006
Sex Differences England/Wales (male–female)						
1840–1844	1840–1844	0.0011	0.0005	0.4722	0.0007	0.0006
1870–1874	1870–1874	0.0014	0.0007	0.6691	0.0010	0.0007
1890–1894	1890–1894	0.0021	0.0008	1.2945	0.0014	0.0009
1910–1914	1910–1914	0.0022	0.0008	1.8055	0.0016	0.0008