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HUMAN ACTUARIAL AGING INCREASES FASTER WHEN BACKGROUND DEATH RATES ARE LOWER: A CONSEQUENCE OF DIFFERENTIAL HETEROGENEITY?

Kristen Hawkes,[1,2] Ken R. Smith,[3,4,5] and James K. Blevins[1,6]

[1]*Department of Anthropology, University of Utah, Salt Lake City, Utah*

[2] *E-mail: hawkes@anthro.utah.edu*

[3]*Department of Family and Consumer Studies, University of Utah, Salt Lake City, Utah*

[4]*Huntsman Cancer Institute, University of Utah, Salt Lake City, Utah*

[5]*E-mail: ken.smith@fcs.utah.edu*

[6] *E-mail: jim.blevins@utah.edu*

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Many analyses of human populations have found that age-specific mortality rates increase faster across most of adulthood when overall mortality levels decline. This contradicts the relationship often expected from Williams' classic hypothesis about the effects of natural selection on the evolution of senescence. More likely, much of the within-species difference in actuarial aging is not due to variation in senescence, but to the strength of filters on the heterogeneity of frailty in older survivors. A challenge to this differential frailty hypothesis was recently posed by an analysis of life tables from historical European populations and traditional societies that reported variation in actuarial aging consistent with Williams' hypothesis after all. To investigate the challenge, we reconsidered those cases and aging measures. Here we show that the discrepancy depends on Ricklefs' aging rate measure, ω, which decreases as mortality levels drop because it is an index of mortality level itself, not the rate of increase in mortality with age. We also show unappreciated correspondence among the parameters of Gompertz–Makeham and Weibull survival models. Finally, we compare the relationships among mortality parameters of the traditional societies and the historical series, providing further suggestive evidence that differential heterogeneity has strong effects on actuarial aging.

KEY WORDS: **Frailty, Gompertz, mortality rate doubling times, Ricklefs' omega, Strehler–Mildvan correlations, Weibull.**

Mortality Rates and Senescence

Increasing age brings senescence, inevitable deterioration in physiological state and functional performance. A measure of this deterioration is the rising risk of death. Gompertz (1825), studying nineteenth century European mortality records, recognized that human mortality rates increase geometrically across most of adulthood. A model bearing his name has been found to fit that increase in mortality hazard with age not only for humans but for other animals as well (Finch 1990). Following Gurven and Fenelon (2009:1020), the two-parameter Gompertz model is:

$$m_x = m_g \exp(\gamma x),$$

where, m_x is the mortality rate at age x, m_g is the age-independent adult mortality rate, and γ is the rate of increase in adult mortality with increasing age x. This applies only to adults, so $x = 0$ at the beginning of adulthood. At that age the hazard is m_g, designated

as the initial mortality rate (IMR) by Finch (1990). When logarithmically transformed, the Gompertz model gives the log of mortality risk as a straight line across adult ages with $\ln(m_g)$ its intercept, and γ its slope. To characterize the rate of increase with age more intuitively, the slope is transformed to give a mortality rate doubling time (*MRDT*) (Sacher 1977; Finch 1990):

$$MRDT = (\ln 2)/\gamma.$$

Evolutionary biologists (e.g., Ricklefs 1998), gerontologists (Sacher 1977; Finch 1990), and demographers (e.g., Olshansky and Carnes 1997) have conceptualized the parameters of the Gompertz model as incorporating two different mortality components, one of them, m_g or the IMR, set by general species characteristics and local extrinsic threats that affect all ages, and the other, γ, the slope of the increase with age, set by intrinsic vulnerabilities that accumulate across adulthood due to physiological senescence. Extrinsic mortality is associated with risks such as accidents, disease, predation, climatic hardships, and food shortages. These hazards set the mortality "level" for all members of a population, moving the whole survival schedule up or down. Intrinsic mortality is associated with the progressive physiological deterioration that begins after maturity and results in actuarial aging, the "increase in mortality risk with age" across most of adulthood.

Evolutionary theoreticians disagree about whether to expect mortality to increase geometrically with adult age as a general outcome of natural selection on senescence (e.g., Abrams and Ludwig 1995; Mueller and Rose 1996), but there is a venerable history of appealing to mortality level to explain the evolution of the wide variation in senescence rates across species. Williams (1957:403–404) referred to the "phylogeny of senescence" and deduced that organisms with an evolutionary history of lower adult mortality should senesce more slowly whereas those with a legacy of higher mortality should have faster senescence rates. Gurven and Fenelon (2009:1017) recently aimed to test "Williams's 1957 hypothesis...that higher age-independent, or "extrinsic," mortality should select for faster rates of senescence... using mortality data from subsistence populations and from historical cohorts from Sweden and England/Wales... [to] examine whether rates of actuarial aging declined over the past two centuries." They concluded "that actuarial senescence has slowed in later European cohorts, [and] reductions in extrinsic mortality associate with slower actuarial aging in longitudinal samples" (p. 1017).

Yet the opposite pattern has often been found. The relationship between mortality level and rate of increase in mortality with age among populations of the same species can be strongly negative. Strehler and Mildvan (1960) were prompted to investigate this relationship among human populations by their theory of aging which assumed that Gompertz parameters could be associated with the parameters in an equation for the distribution of kinetic energy in atoms and molecules (Strehler 2000; Golubev 2009). They predicted, and found, a negative correlation between the two parameters of Gompertz models constructed from a sample of national life tables drawn from the UN Demographic Yearbook for 1955. The same correlation has been shown for subpopulations of human societies, across human populations over time, and across different populations of other species (e.g., Gavrilov and Gavrilova 1991; Nam 1996; Riggs and Hobbs 1998; Pletcher and Neuhauser 2000; Yashin et al. 2001, 2002a). Gavrilov and Gavrilova (1991) noted errors in Strehler and Mildvan's original calculations, corrected them, and found that the strong correlation persisted nevertheless. They labeled this the compensation law of mortality. ''High mortality rates in disadvantaged populations (within a given species) are compensated for by low apparent ''aging rates'' (longer mortality doubling period)'' (Gavrilov and Gavrilova 2001:528).

Strehler–Mildvan correlations came to our attention as we investigated demographic differences between humans and chimpanzees, expecting—on grounds of Williams' hypothesis—chimpanzees to show faster rates of aging than humans. Instead we found that both Gompertz parameters (mortality level and rate of increase in mortality risk with age) vary among populations of each species and the ranges of variation in both level and rate of increase overlap. Yet humans showed, and the limited chimpanzee data suggested, distinct, negative relationships between those parameters characteristic of each species (Hawkes et al. 2009).

Emphasizing that Strehler–Mildvan correlations are opposite to the classic expectation about lower adult mortality levels selecting for slower rates of physiological aging, we hypothesized that Strehler–Mildvan correlations do not measure variation in senescence rates but differential heterogeneity in adult age classes due to the strength of mortality filters. Given that individuals vary in their relative frailty, mortality selection will alter the composition of older age classes (e.g., Vaupel and Yashin 1985). When overall death risks are higher, more relatively frail individuals die younger, leaving older age classes disproportionately represented by those that were always at lower risk. Conversely, with lower mortality hazard at all ages, members of vulnerable subpopulations survive longer. Their relative risks contribute more to the average risk of older age classes (Hawkes et al. 2009; Hawkes 2010; Hawkes and Smith 2010). Under this differential heterogeneity hypothesis, equating the demographic aging rate of populations with the age-specific mortality risks of the individuals that compose them is an ecological fallacy (Robinson 1950; Wilmoth and Horiuchi 1999). These arguments, tracing back at least to Pearl (1922), may explain why human populations with lower mortality levels have higher rates of actuarial aging as

shown by Strehler and Mildvan (1960) and subsequently many others.

But the question has lingered: Does the Strehler–Mildvan correlation reflect a real empirical pattern or is it a modeling artifact? Using many of the same populations that were in our convenience sample (Hawkes et al. 2009), Gurven and Fenelon (2009:1017), reported the contrary pattern: "higher extrinsic mortality associates with faster aging." Here we address this contradiction, presenting additional analyses to explain the opposing results. Gurven and Fenelon use more than one measure of aging rate and note that answers depend on the measure used. We follow their lead and investigate the reasons for the difference.

We begin with the historical Swedish life tables, first applying a model-free measure to assess the modeling artifact question. Then we introduce the classic survival models Gompertz–Makeham (GM) and Weibull, that—unlike Gompertz—include a separate additive term for young adult mortality. We apply these models and the aging measures constructed from their parameters to the Swedish series. We focus on females because, as the model-free evaluation shows, the sexes differ in detail but not in the relationship of interest here. So including both sexes would double reported results while adding nothing to the analysis at hand. We show the relationships among the model parameters and the aging measures for the Swedish historical cohorts, and confront the apparent inconsistency that when background mortality falls, actuarial aging rates increase as measured by *MRDT*, but decrease as measured by ω (Ricklefs 1998). We explore the behavior of ω and show that it necessarily follows mortality level not rate of increase with age. Applying the two classic models to the same dataset reveals that in spite of the model differences, their parameters behave in closely corresponding ways. Then we consider the subsistence populations and compare the behavior of parameters reported by Gurven and Fenelon across this dataset to their behavior across the Swedish series. Although Strehler–Mildvan correlations are extremely strong in both cases, the relationships between the additive terms for young adult mortality and the actuarial aging terms differ. We explain how differential heterogeneity in the older age classes might account for this difference.

Model-Free Measures

Gurven and Fenelon used cohort life tables for Swedes born from 1751 through 1910 and report similar findings for period life tables from 1751 to 1930 drawn from the Human Mortality Database (www.mortality.org). Over the decades covered by these records, age-specific mortality rates declined substantially. Because parametric survival models might impose artifactual results, we first investigate model-free measures of both mortality level and rate of actuarial aging over the historical series.

Figure 1 (period life tables above the diagonal, cohorts below) plots results for Swedish females using two measures of mortality level, the mortality rate in the 30–34 year age class, m_{30-34}, and the fraction surviving to that age, $l_{30-34} \times 10^{-5}$ (l_x values are divided by 10,000 throughout to turn them into fractions). The figure also plots the relationship of these measures of mortality level to a simple model-free measure of actuarial aging: the ratio of the mortality rate at the age of 80 to the rate at age 30, m_{75-79}/m_{30-34}. This ratio is a direct empirical assessment of how much the mortality rate increases over these 50 years of adulthood. In addition, the figure includes starting year to show how the parameter values change over time. Figure 1 displays results for females only, although we report the findings for both sexes here. For period 5×5 life tables (five year age classes averaged over five years), the mortality rate in the 30–34 year age class for females fell from 0.015 to 0.004, for males it fell from 0.010 to 0.004. The fraction of females surviving to 30 climbed from a minimum of 0.48 to a maximum of 0.88, whereas for males the increase was from 0.53 to 0.87. The ratio of mortality rate at 80 to the mortality rate at 30 increased from 12.9 to 37.0 in the female period life tables and for male periods the ratio increased from 10.6 to 23.4. For female cohort 5×5 life tables (5-year age intervals averaged over five years)—below the diagonal—the mortality rate in the 30-34 year interval, m_{30-34} fell from a high of 0.013 to a low of 0.002. For male cohorts m_{30-34} fell from a high of 0.013 to a low of 0.003. The fraction of female birth cohorts surviving to age 30 ($l_{30-34} \times 10^{-5}$) increased from a minimum of 0.53 to a maximum of 0.84 whereas the male fraction surviving to 30 increased from 0.51 to 0.82. For cohorts, the ratio of mortality at 30 to mortality at 80, m_{75-79}/m_{30-34} went from 12.9 to 37.0 for females, 10.6 to 23.4 for males. By these model-free measures, actuarial aging rates increased as mortality levels fell in both sexes and period and cohort life tables. Yet Gurven and Fenelon concluded the opposite from their analyses. To investigate why, we introduce the models and measures they used and apply them to the same Swedish record. Then we evaluate the behavior of the aging rate measures and investigate why the measures give contradictory results. Following this we analyze the parameter values they reported from fitting the same models to 15 life tables from traditional societies and compare the patterns to those in the Swedish series. There are differences as well as similarities and we explore how the differential heterogeneity hypothesis may explain them both.

GM and Weibull Models

Gurven and Fenelon use standard survival models to measure changes in rates of actuarial aging. Makeham (1867) added a separate age-independent component, m_0, to the classic two-parameter Gompertz model and Gurven and Fenelon employ it. We attach the

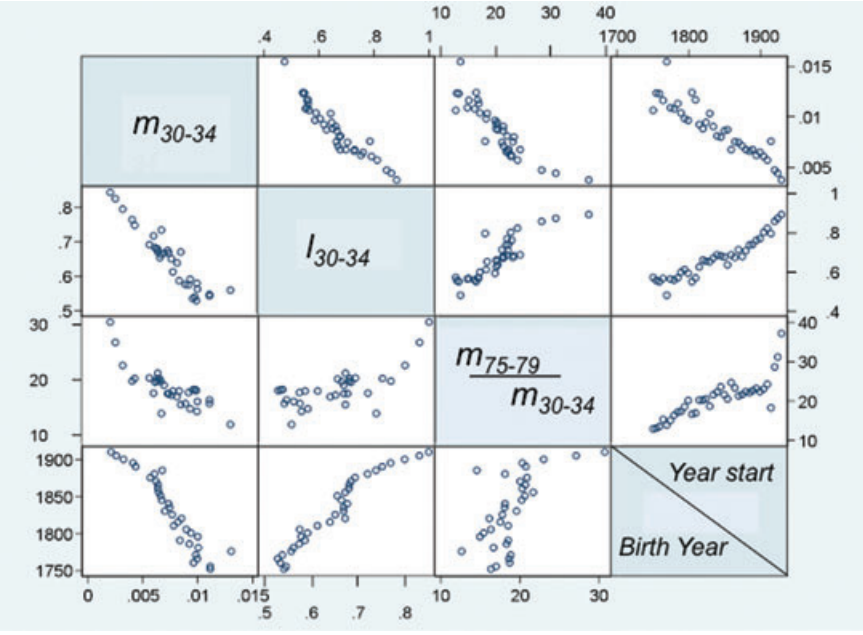


Figure 1. Nonparametric mortality indices for Swedish females by year: m_{30-34}, l_{30-34}, m_{75-79}/m_{30-34}. Period life tables are above the diagonal, cohort life tables below.

subscript "*k*" (for Makeham) to the GM parameters to distinguish them in model comparisons:

$$m_x = m_k \exp(\gamma_k x) + m_{0k}.$$

In GM, as in the Gompertz model the exponent, γ (γ_k in GM) indicates the rate of increase in mortality with increasing adult age x. The mortality rate doubling time (*MRDT*) (Sacher 1977; Finch 1990) in GM is

$$MRDT_k = (\ln 2)/\gamma_k.$$

Gurven and Fenelon also use the Weibull model argued by Ricklefs (1998) to be preferable to Gompertz for characterizing rates of aging because it "separates the initial mortality rate computationally and characterizes aging related mortality by two parameters, one (β [dimensionless]) controlling shape and the other (α [time$^{-(\beta+1)}$]) magnitude for a given shape" (Ricklefs 1998). We add the subscript w to the additive term in this model to distinguish it from the additive term in GM

$$m_x = \alpha x^{\beta} + m_{0w}.$$

Failure (or death) is a power function of age in the Weibull model in contrast to the Gompertz and GM models where death (failure) is an exponential function of age.

As an alternative to *MRDT*, which is defined by the Gompertz exponent and so can only be calculated from Gompertz or GM models, Ricklefs (Ricklefs 1998; Ricklefs and Scheuerlein 2002, 2003) developed other indices for the rate of aging, labeled ω. Omega can be calculated from the parameters of any of the previous models. For the Gompertz and GM models:

$$\omega_g = (m_g \gamma)^{1/2} \text{ and } \omega_k = (m_k \gamma_k)^{1/2}.$$

For the Weibull model

$$\omega_w = \alpha^{1/(\beta+1)}.$$

Swedish Historical Demography

Gurven and Fenelon estimated GM and Weibull models from the historical life tables using adult age classes up to 95 years. We exclude ages over 80 because departures from Gompertz at older ages are widely recognized (Vaupel et al. 1998), including by Gompertz himself (Olshansky 1998). We use 5-year (5×5) instead of 1-year age classes (1×5) to provide some smoothing, spanning age 30 to 80 with mortality level indexed by m_{30-34}. We fit model parameters to the life tables using SAS as Gurven and Fenelon did and evaluate the strength and direction of associations with Pearson correlation coefficients.

Gurven and Fenelon focused on birth cohorts. Students of Swedish historical demography have found that the subsequent mortality rates of those born at the same time reveal an impact of shared early life conditions (Kermack et al. 1934; Fridlizius 1989; Bengtsson and Lindstrom 2000, 2003; Finch and Crimmins 2004). But period effects are also substantial across these centuries (Barbi and Vaupel 2005; Murphy 2010). Gurven and Fenelon (2009:1024) say, "Based on analyses of period data (not shown),

we find the same significant relationships but with stronger correlations between aging measures... and m_0." We also found similar patterns in the relationship between rate of increase with age and mortality level in both cohort and period life tables as shown in our model-free measures (Fig. 1). The two sexes show similar relationships between mortality level and rates of increase in mortality with age so we report data on females and focus on cohorts to more closely parallel Gurven and Fenelon's analysis.

The first cell above the diagonal of Figure 2 shows that $MRDT_k$ and the Gompertz exponent, γ_k, are strongly negatively correlated ($r = –0.997$) as they must be by definition (r is not a perfect –1 because Pearson is a linear correlation). While the GM and Weibull parameterizations are quite different, the exponents in the two models (γ_k and β) fitted to the same data are perfectly positively correlated (although values of the Weibull β are 41-fold larger than the GM γ_k)—the next cell along and above the diagonal. So the Weibull exponent is also strongly negatively correlated with $MRDT_k$ ($r = –0.996$). In addition, the multiplicative terms of the two models, m_k and α, are positively correlated with each other ($r = 0.929$). The multiplicative terms and the exponents are negatively correlated with each other in both models ($r = –0.942$ for GM, $r = –0.895$ for Weibull). As is evident in Figure 2, neither correlation is linear so these Pearson correlations underestimate the strength of their association.

Cells in the right column show that $MRDT_k$ and the two multiplicative terms, m_k and α, are positively correlated with mortality level as indexed by m_{30-34} ($r = 0.634$, 0.749, and 0.584 respectively; $P < 0.001$ in all cases). The exponents are the conventional measures of the rate of change in mortality rate with age in both GM and Weibull (Kleinbaum and Klein 2005). Both are negatively correlated with mortality in the 30–34 year age class ($r = –0.635$ for γ_k and –0.618 for β; $P < 0.001$ in both cases). By these measures, the rate of actuarial aging is faster, the slope is steeper, the doubling time is shorter as the level of mortality declines—opposite to Gurven and Fenelon's conclusion.

Cells below the diagonal, based on cohorts, show associations of $MRDT_k$ with the ω values and additive terms for both GM and Weibull parameterizations plus calendar year to indicate how these change over time. $MRDT_k$ values are positively correlated with ω_k ($r = 0.930$) and with ω_w ($r = 0.771$) as well as with both additive terms, m_{0k} ($r = 0.467$) and m_{0w} ($r = 0.660$); $P < 0.001$ in all cases. The two omegas, ω_k and ω_w, are very closely correlated with each other ($r = 0.949$) and both are negatively correlated with birth year ($r = -0.900$ and -0.971, respectively). So, if ω is a measure of aging rate, that rate goes down over time in this sample. Yet paradoxically the omegas are positively correlated with $MRDT_k$. By $MRDT_k$, the actuarial aging rate increases over time as mortality levels decline, whereas by ω it decreases. Gurven and Fenelon note this "contradictory result of more rapid aging over time according to *MRDT* and slower aging according to ω" (2009:1022). To understand this contradictory result we first consider ω_k.

Cursory inspection of the definition makes it look as though ω_k should vary directly with γ_k, but it cannot because the multiplicative term, m_k, and the exponent, γ_k, are negatively correlated with each other and it is m_k that dominates. This domination of m_k is evident in the natural logarithm of ω_k:

$$\ln(\omega_k) = (\ln[m_k] + \ln[\gamma k])/2.$$

Because both m_k and γ_k have values less than 1, their logarithms are negative; and, because m_k is smaller, the absolute value of $\ln(m_k)$ is larger. Consequently it is $\ln(m_k)$ that has the larger effect on $\ln(\omega_k)$.

As with ω_k in GM, ω_w in Weibull is positively correlated with m_{30-34} ($r = 0.934$) and positively correlated with the multiplicative term, in this case, α ($r = 0.727$). Logging ω_w clarifies those relationships:

$$\ln(\omega_w) = \ln(\alpha/(\beta+1)).$$

Although the behavior of this nonlinear function is complex, made more so by the strong negative linear correlation between $\ln\alpha$ and β; ($r = –0.990$)—the Strehler–Mildvan correlation mentioned above and discussed again below—this transformation shows that ω_w must be positively associated with α and negatively with β, the Weibull slope. As is the case with ω_k, ω_w is an index of mortality level not actuarial aging rate.

Although specifications differ, GM and Weibull models both fit the Swedish data well. Pearson correlation coefficients between observed and predicted mortalities for the 37 female 5×5 periods from 1751 to 1930 and the 33 female cohorts from 1751 to 1910 are all greater than $r = 0.99$. To illustrate the fit and highlight the difference between mortality level and actuarial aging rate, Figure 3 plots the observed age-specific mortalities for two periods (top panel) and two cohorts (bottom panel) chosen to represent high and low mortality levels. GM and Weibull models of these data are plotted as well. Between periods 1770–1774 and 1930–1934 in the upper panel, m_{30-34} declines from 0.015 to 0.004, the proportion surviving to 30 increases from 0.48 to 0.88, and the ratio of the mortality rate at the age of 80 to the rate at 30 years, m_{75-79}/m_{30-34} climbs from 13.7 to 37.0. Slope parameters of GM and Weibull models fitted to these data both increase over time, γ_k from 0.077 to 0.109, and β from 3.23 to 4.50. In the lower panel of Figure 3, the cohort birth years are 1775–1779 and 1910–1914. Between them mortality in the 30–34 year age class declines from 0.013 to 0.002, the proportion surviving to 30 increases from 0.56 to 0.84, and the ratio of mortality at 80 to mortality at 30 rises from 12.7 to 30.8, while γ_k rises from 0.100 to 0.108, and β from 3.89 to 4.43. Both panels show that mortality rates at all ages are shifted upwards when young adult mortality is higher. They

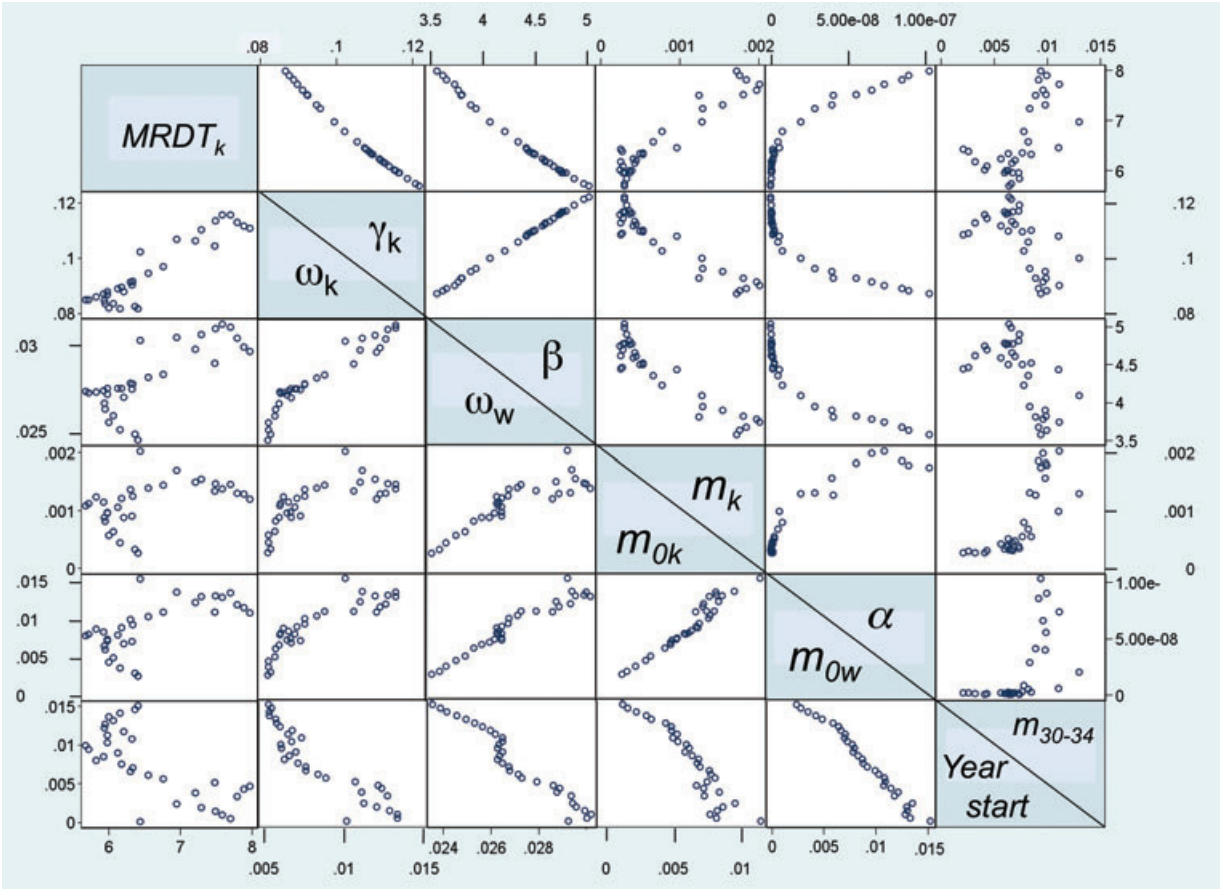


Figure 2. Parameter relationships among and between Gompertz–Makeham ($m_x = m_k \exp(\gamma_k x) + m_{0k}$) and Weibull ($m_x = \alpha x^{\beta} + m_{0w}$) models as well as the aging measures, *MRDT* (= (ln2)/γ_k) and the omegas ($\omega_k = (m_k \gamma_k)^{1/2}$ and $\omega_w = \alpha^{1/(\beta+1)}$) defined by them.

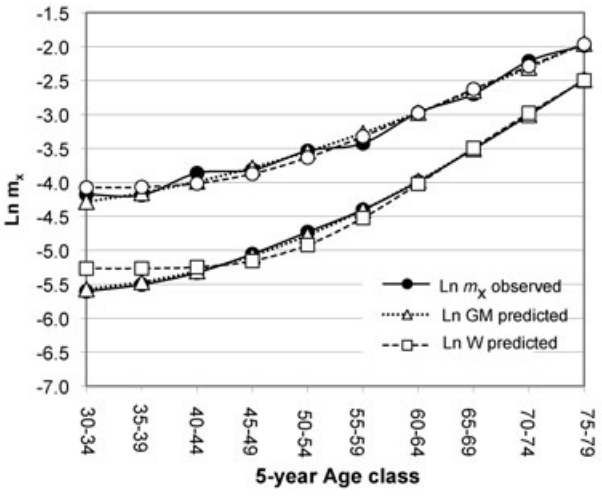
also show that the difference between mortality rates at older ages is smaller than the difference at younger ages. The mortality curves are not parallel. Higher mortality rates at younger ages are associated with shallower slopes: slower subsequent increase in mortality.

Traditional Societies

The Swedish data come from a large population with well-controlled national records. This reduces the distortion of chance effects on demographic processes and minimizes errors in identifying ages at death. But Swedish mortality experience may not represent that of other human populations. Many suspect that age-specific risks and relationships among mortality parameters might be quite different for people in subsistence societies, especially hunter–gatherers who confront ancient death risks more similar to those faced by everyone before the origins of agriculture. The mortality experience of such populations is especially difficult to capture because samples are small and death ages must be painstakingly estimated. But careful demographies of small-scale societies have been accumulating. We agree with Gurven and Fenelon that the similarity in mortality patterns these studies reveal between traditional societies and eighteenth and nineteenth century Europe are important and underappreciated lines of evidence about the evolved life history of our species (Hawkes 2003; Gurven and Kaplan 2007). Howell (1979) made this point decades ago, although it continues to be widely ignored (Hawkes and Blurton Jones 2005).

Gurven and Fenelon explore the relationships between mortality level and actuarial aging for a sample of traditional small-scale societies similar to the one used by Gurven and Kaplan (2007). They calculated GM and Weibull parameters for 15 life tables: four for hunter–gatherers, five for acculturated hunter–gatherers, and six for forager–horticulturalists—some from the same society at different time periods (2009:1023). Across this sample "m_0 [our m_{0k}] is inversely related to both ω and *MRDT* in the GM analysis ($P = 0.03$, $P = 0.006$, respectively, ...)" (p. 1022). For reasons explained above, we expect $MRDT_k$ and ω_k to be positively correlated with each other. $MRDT_k$ declines by definition as the Gompertz exponent (γ_k) increases; and although the definition of ω seems to imply it will vary directly with γ, it cannot if the component terms of ω_k are both less than 1 and the

Upper Panel. Swedish female 5X5 periods – higher lines represent the high mortality period 1770-74, lower lines the low mortality period 1930-1934.



Lower Panel. Swedish female 5X5 cohorts – higher lines represent the high mortality cohort born 1775-79, lower lines the low mortality cohort born 1910-1914.

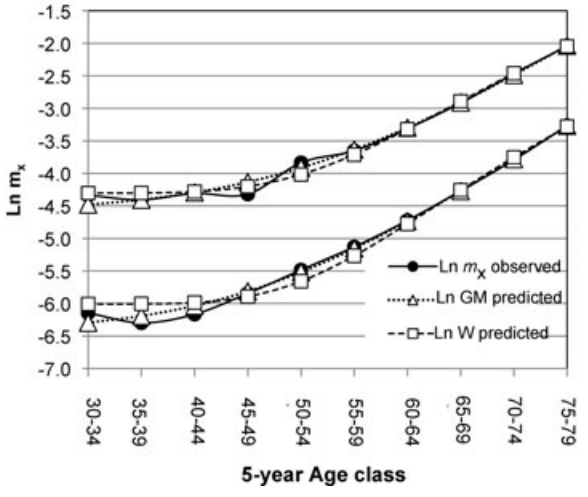


Figure 3. Observed age-specific mortality, m_x (filled symbols) and mortality predicted from Gompertz-Makeham, (GM—open triangles), and Weibull models (W—open squares), for two periods and two cohorts at two mortality levels, one high and one low.

value of m_k is smaller than in γ_k. The range of variation in m_k reported by Gurven and Fenelon for the traditional society sample covers six orders of magnitude with a maximum of 0.003 whereas the minimum value of γ_k across its 4.5-fold range is 0.059. As in the Swedish series, the domination of m_k in ω_k for the traditional societies is reflected in the close correlation between them ($r = 0.928$). Relationships between ω_w and Weibull parameter values are also similar to those in the Swedish series. However the Pearson correlation of ω_w with the Weibull slope β is not statistically significant ($r = -0.471$, $P = 0.076$), an apparent violation of the claim above that ω_w must be negatively related to the slope. This apparent contradiction is due to the nonlinearity of their relationship which is not captured by the Pearson correlation. When we fit a logarithmic regression the correlation of ω_w with β is $r = -0.70$ ($P = 0.004$), and as expected, ω_w is positively correlated with the multiplicative parameter α, $r = 0.744$ ($P = 0.002$).

There is, however, a difference between the Swedish series and the subsistence sample in the way the additive terms in both GM (m_{0k}) and Weibull (m_{0w}) are related to the other parameters. For Sweden, m_{0k} is positively correlated with both $MRDT_k$ and ω_k ($r = 0.402$, $P = 0.020$, and $r = 0.652$, $P < 0.0001$, respectively) although correlations are much stronger with the multiplicative term (m_k) ($r = 0.961$ with $MRDT_k$ and $r = 0.983$ with ω_k). As noted above, in the Swedish cohorts the additive term and the multiplicative term are positively correlated with each other, and each is positively correlated with observed mortality at age 30. Both are indices of mortality level.

In the subsistence societies, however, although the Strehler–Mildvan correlations between ln(m_k) and γ_k and between ln(α) and β are very strong, $r = –0.984$ and –0.999, respectively, parameters and estimates of mortality among young adults are not coordinated with the multiplicative terms, and not related to either $MRDT_k$ or ω in the same way. The additive GM term (m_{0k}) is not positively correlated with the multiplicative term (m_k), $r = –0.389$, $P = 0.151$, and whereas the additive term is positively correlated with observed initial mortality (m_{0k} with m_{low}, $r = 0.757$, $P = 0.001$), the multiplicative term is not (m_k with m_{low}, $r = –0.014$, $P = 0.959$). Also in contrast to the Swedish series, the additive term is *negatively* correlated with both $MRDT_k$ and ω_k ($r = –0.694$, $P = 0.004$, and $r = –0.562$, $P = 0.029$, respectively). And, as Gurven and Fenelon report, in Weibull there is not a significant relationship between the additive term (m_{0w}) and ω_w for this subsistence sample ($r = –0.202$, $P = 0.471$).

A differential heterogeneity hypothesis is a strong contender to explain this lack of coordination between mortality rates in young adults and the mortality experience of their elders. The subsistence society parameters are necessarily calculated from period life tables. If there have been large recent shifts in mortality levels, the only elders available to die are those who survived the previous mortality regime. Older age classes still retain the imprint of higher mortality levels in past. We turn to the issue of heterogeneity and then use it to further explore this feature of the subsistence sample.

Differential Heterogeneity

The association between lower overall mortality levels and faster actuarial aging in the Swedish data and implied by the Strehler–Mildvan correlations in the subsistence series may be explained, at least in part, by a differential heterogeneity of frailty hypothesis. When younger age classes experience lower mortality, heterogeneity in each succeeding age class is necessarily greater. The survival of more vulnerable members into older age classes raises the average risk for their age interval as a whole. We have in mind a combination of both level and slope heterogeneity (Wilmoth and Horiuchi 1999; Barbi et al. 2003). When the relative risks of more frail subpopulations are both higher at each age and increase faster with age, then lower overall mortality steepens the slope of the log of the hazard for the population as a whole (Hawkes et al. 2009; Hawkes 2010). The same hypothesis to explain pervasive Strehler–Mildvan correlations could explain why the relationship between mortality rates in young adults and the rate of increase in mortality with age across adulthood in the subsistence populations is different from the historical Swedish series. If differential heterogeneity does affect actuarial aging rates, then relatively large reductions in overall mortality as observed in some of the subsistence populations would not be accompanied by immediate shifts in the rate of increase in mortality with age. Gurven and Fenelon's set of life tables allows us to further explore that possibility.

Their subsistence dataset includes model parameters for five pairs of mortality schedules for the same societies, one earlier in time than the other. For those pairs the difference in mortality between early and late averages a 3.15-fold decrease at the lowest point, m_{low}. Compare this to the 4.18-fold decrease in mortality at age 30 for 5-year Swedish periods from the beginning ages of 1751 to 1930. That 4.18-fold change in Sweden took more than a century and a half while the early and late periods in the subsistence populations are either directly adjacent in time or separated only briefly. Consequently the older age classes used to calculate mortality rates in the later subsistence life tables have been previously filtered by the more severe earlier mortality regime. If actuarial aging rates reflect reductions in the heterogeneity of older age classes they will conform more to the earlier than to the later level of mortality.

It is consistent with this hypothesis that Gurven and Fenelon found no statistically significant differences between the *MRDT*s of the subsistence populations that were characterized both before and after substantial reductions in overall death risks. However, removing those, and the other "acculturated hunter-gatherers" does not change the negative relationship between the additive (m_{0k}) and multiplicative (m_k) terms noted for the traditional society sample as a whole. Nevertheless, Strehler–Mildvan correlations remain very high whatever subsets of this subsistence sample are considered showing the consistent pattern of faster actuarial aging when the mortality level indicated by the multiplicative term is lower. Perhaps recent variation in mortality level in all these cases makes the mortality represented by the additive term a misleading index of preceding mortality levels.

Discussion

We have shown that for the historical Swedish data, the simple model-free ratio of m_{75-79}/m_{30-34}, as well as widely used measures indicate that rates of actuarial aging increased over time as background mortality declined—except ω. We found that the contradictory behavior of this measure results from its domination by mortality level. Because ω tracks mortality level, it cannot reflect negatively correlated changes in actuarial aging rates. We also found unexpected correspondences among the parameters of GM and Weibull models by fitting them to the same dataset. Although Weibull has been characterized as an additive model of aging and Gompertz as proportional, with expected behavior of their aging terms distinguished accordingly (Ricklefs and Scheuerlein 2002), variation in the exponents of GM and Weibull fitted to the Swedish historical series are perfectly correlated with each other. Both models recover strong negative relationships between

changes in their exponents and changes in their corresponding multiplicative terms in the Swedish series as well as in the set of traditional societies. Strehler–Mildvan correlations recur across human populations, as mortality levels vary over both time and space. A likely explanation for this recurrence is differential heterogeneity in older age classes due to the varying strength of mortality filters.

Although Gurven and Fenelon's ostensible aim was to test "Williams 1957 hypothesis ... that higher age-independent, or "extrinsic," mortality should select for faster rates of senescence" (2009:1017), they also surmised, and we agree, that in the time frame of less than 10 generations they examined, "changes in extrinsic mortality may not have any evolutionary consequences on the aging process" (p. 1028). And, in spite of their contrary conclusions, Gurven and Fenelon recognize evidence from Sweden that actuarial aging rates have increased over time; e.g., on p. 1027 they note that:

> If we had used only the traditional *MRDT* from a GM model, we would have ... concluded that aging has accelerated, rather than slowed down, in more recent cohorts...
>
> *MRDT* "declines" over time in ... Sweden ... 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).

One reason Gurven and Fenelon conclude that there are "declines in actuarial aging over the past several hundred years" (p. 1030) is their reliance on ω. As our analyses show, this function is negatively correlated with the exponent, the standard index of the rate of increase in mortality risk with age in Gompertz, GM, and Weibull (Kleinbaum and Klein 2005). We are not the first to note that ω is a measure of mortality rate not changes in that rate with age—not actuarial aging. Ricklefs and Scheuerlein (2002: B72) say that for Gompertz (and by implication GM), "because ω_g is calculated from the product of m_0 [here m_g] and γ, reducing the value of m_0 tends to reduce the value of ω_g ... Thus, ω_g is sensitive to the value of m_0 and is therefore not a robust measure of the rate of aging." Similarly for Weibull, Ricklefs and Scheuerlein (2003:93) point out that "from the logarithmic form of the expression, $\log\omega = \log\alpha/(\beta+1)$, one can see that for a given value of β, the value of $log\omega$ is directly proportional to $log\alpha$." For a wide array of birds and mammals, Ricklefs and Scheuerlein (2002:B71) report that "β is often close to 3 in natural and captive populations and the value of ω_w is relatively insensitive to variation in the value of β." They explicitly conclude that "using ω derived from either the Gompertz or Weibull equation version, one defines aging according to the magnitude of the mortality rate at a particular age" (Ricklefs and Scheuerlein (2003:86). This means that at least for the human data canvassed here—and perhaps more generally—ω must vary "inversely" with rates of actuarial aging.

A second reason Gurven and Fenelon may be reluctant to rely on *MRDT* as a measure of actuarial aging is that they follow Finch in assuming that *MRDT* "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)" (Gurven and Fenelon 2009:1020). When Finch and Crimmins (2004) plotted the Swedish age-specific mortality (sexes not distinguished) over nearly the same time span we used in Figure 3, they emphasized the "stability of cohort mortality slopes...despite remarkable variability in overall mortality" (p. 1739). Our analysis counters their claim that "declines in mortality have had little effect on the basic rate of mortality acceleration during aging" (p. 1737). When the hazard is lower at all ages, the actuarial aging rate, the slope of the increase with age is significantly steeper. As Finch and Crimmins noted, changes in slope are larger in the period than in the cohort life tables, but as shown in Figure 3, lower level of mortality is accompanied by a faster rate of increase with age in both of them.

The *MRDT*s that Gurven and Fenelon report for their subsistence sample show a larger range, from 2.62 to 11.85. Removing the three extreme values, the range is still from 5.55 to 10.98. In Gurven and Kaplan's (2007:327) analysis of high mortality human populations, *MRDTs* range from 2.8 to 17.3. For our convenience sample (Hawkes et al. 2009) the range of *MRDT*s was 8.2 to 32.7. Removal of the latter extreme value leaves the next largest, 17.9, still more than a twofold range. Shortly after Finch (1990; Finch et al. 1990) suggested the stability of actuarial aging rates within species, Wood et al. (1994) mounted an empirical challenge. Turning to the 1991 UN Demographic Yearbook, Wood and colleagues calculated Gompertz models for mortality schedules from 27 countries for ages 15 to 84 years and reported that, "The average MRDT is 8.5 ± 7.6 (mean $\pm$2s.d.) for females (with a range from 4.8 to 19.8) and 9.8 ± 9 for males (range = 6.1–26.4)..." (Wood et al. 1994:40). Claims that *MRDT*s vary little within species are correct only in the sense that the exponent that defines them varies less than the Gompertz (or GM) multiplicative term. It is the variation in human *MRDTs*, and its correlation with mortality level that gives ubiquitous Strehler–Mildvan correlations.

The data in Wood et al. 1994 (Table 2–4) allow calculation of the Strehler–Mildvan correlations for their 1991 UN Yearbook sample. As usual the Strehler–Mildvan correlations are very high: $r = -0.97$ for both females and males. Zheng et al. (2011) used data for 42 countries from the UN Demographic Yearbook 1955–2003 to calculate Strehler–Mildvan correlations from Gompertz

models for 5 × 5 male periods from age 30 to an open ended 85+ interval. They report that the strength and even direction of the Strehler–Mildvan correlations varies among countries, but the aggregated set of 462 country-period observations gives a correlation between $\ln m_g$ and γ of $r = -0.92$. Golubev (2009), having noted that Strehler–Mildvan correlations can be inflated by failure to incorporate the additive Makeham term, constructed GM models for 18 developed countries for which the Makeham term was "negligibly small" (2009:11). Using female life tables across the age range 25–80 years for the period 2000–2004 from the Human Mortality Database, he found a Strehler–Mildvan correlation of $r = -0.98$. These analyses add to the accumulating evidence that falsifies the claim Ricklefs (2000:108) made that ". . .rate of aging in human populations is independent of differences in the baseline mortality rate among nations." Instead, γ, which defines *MRDT* for Gompertz and GM, and β, which gives the Weibull equivalent, vary regularly with baseline mortality rates among human populations and within them over time.

Our favored hypothesis that this Strehler–Mildvan pattern results from mortality filters on the heterogeneity of frailty in older age classes may be clarified by a more extreme example. Finch and colleagues (1990) drew attention to striking similarity in the rate of increase in mortality hazard with age between Australian soldiers in Japanese prisoner-of-war camps and Australian civilians during the same period of World War II. In the horrendous conditions of the prison camps, age-specific death rate was at least 10 times higher than among the civilians. But this difference was accompanied by no difference in the effect of age on risk, no difference in *MRDT* between the prisoners and the civilians. Finch and colleagues interpreted this as evidence that *MRDT* is essentially a species characteristic, "stable under lifelong adverse or good conditions" (1990:903). We considered this argument when we confronted the wide variation in *MRDT*s in our nine-society convenience sample (reflecting the systematic variation in the Gompertz exponent, γ, with mortality level that is captured in Strehler–Mildvan correlations). In light of the evident variation in *MRDT* across our sample, we surmised that the similarity in the slope of the log of the hazard with age between the Australian prisoners and civilians could reflect the shared heterogeneity filters between soldier and civilian populations. "Exposure to the harsh conditions of the camps raised the overall mortality. But, each age class of Australian soldiers who became prisoners was drawn from the civilian population and reflected the civilian heterogeneity; so, the rate of increase in mortality with age was unchanged" (Hawkes et al. 2009).

The same point applies to life tables for the subsistence populations. As in the case of Australian prisoners versus civilians, many individuals in the earlier and later periods of the subsistence populations have experienced the same heterogeneity filters. In the Tsimane case for example, lowest mortality fell sevenfold between the early (1950–1989) and late (1990–2002) periods (Gurven and Fenelon 2009:1023). All those who were adults from 1990 to 2002 had to have survived the mortality filters operating before 1990. Most of the adults available to die in that later period not only grew up in, but lived most of their adulthood before 1990. Reductions in the heterogeneity of frailty during the early period had to persist into the later one contributing to the persistence of past rates of actuarial aging.

Conclusions

Human rates of increase in mortality across adulthood vary systematically so that the inverse relationship between mortality level and rate of actuarial aging in human populations is remarkably strong. As mortality levels for many human populations have declined over the past two centuries lowering the hazard for both 30- and 80-year-olds, the difference between the risks of death in these age classes has increased. Strehler–Mildvan correlations capture this robust pattern. The function ω cannot detect it because ω reflects the decline in mortality level itself, not changes in the rate of increase in mortality with age. Recognition of the actual empirical pattern is important but does not explain it. Differential heterogeneity might. As mortality levels fall, growing fractions of more frail individuals survive to contribute to the hazard at subsequent ages. This hypothesis could also explain some contrasts between the historical Swedish series and the subsistence sample where older adult age classes must retain the imprint of mortality regimes in recent past. Of particular importance, the differential heterogeneity hypothesis also highlights the likelihood that variation in actuarial aging rates among populations of the same species are unrelated to the rates of physiological senescence among individuals.

We considered death risk only to 80 years. In many populations, better diets and less exposure to disease in early life allow more people to arrive at a healthier old age. At the same time, medical and technological advances diminish the prevalence and severity of various deficits and morbidities and also make reduced levels of physical competence compatible with survival, all weakening biases against more frail subpopulations. Yet, even where most individuals survive to ages rarely attained by our ancestors, advancing years still bring deterioration in physiological state and functional performance. Senescence remains a legacy of our evolution (Williams 1957; Hamilton 1966; Kirkwood 2008). And with inevitable heterogeneity, mortality selection still operates, now placing some limit on the rate of expansion in the surviving fraction of disabled elderly (Mitnitski et al. 2005; Christensen et al. 2008; Manton 2008; Kulminski et al. 2008).

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