Why do mortality variability trends for the young and old diverge? A Perturbation Analysis

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Abstract

Variability in longevity has recently been shown to follow strikingly different trends for the young and old: while overall mortality variation decreased as life expectancy at birth rose, survivors to older ages have become increasingly heterogeneous in their mortality risks. These diverging trends reflect changes in the underlying demographic parameters determining age-specific mortality. To understand these changes, we employ a Siler model – which describes the mortality hazard across the full lifespan and allows for the representation of distinct improvements in early-life, later-life, and background mortality. Using maximum likelihood parameter estimation techniques and newly-developed Markov-chain-based matrix calculus perturbation methods, we then quantify the sensitivity of age-specific mortality variance trends to the changing Siler model parameters. Our results suggest that the slower pace and later start of survival improvements in adulthood relative to those at younger ages form the dynamics that foster the growing inequalities observed for survivors to older ages.

Introduction

To understand the demographic transition of the past century and a half, researchers have analyzed the dynamics of population change using both empirical data and mathematical models. While providing insight into the dramatic increase in longevity, these analyses have also occasionally yielded

new puzzles, such as the diverging trends in mortality variability for survivors to young and older ages.

Disparities in the length of life within populations are a fundamental manifestation of health inequalities, and declining variation in longevity is a marker of reduced disparities in population health. Indeed, survival improvements have taken place at all ages, including the oldest (Wilmoth et al. 2001; Rau et al. 2008). However, age-specific trends in variance have not exhibited a uniform pattern: while overall mortality variation decreased as life expectancy at birth rose (Fries 1980, Wilmoth and Horiuchi 1999, Cheung et al. 2005), survivors to older ages have followed a conspicuously different trend, becoming increasingly heterogeneous in their mortality risks. The divergence in conditional variability measures can be summarized in a contour plot (see **figure 1** for an example), and are relatively consistent across industrialized nations with at least five decades of reliable vital statistics (Engelman et al. 2010).

The diverging trends in the contour plot raise important question about why the variation in the adult ages at death has not diminished along with the variation in overall mortality, and, furthermore, why it has increased over time despite progress in medical innovation, public health, standards of living, and aspects of social and economic development associated with rising life expectancy at all ages (McKeown 1976, Szreter 2004). Because the divergence in variance trends by age is visually striking, substantial, and persistent, it should be accounted for in mortality models and in characterizations of population health.

Our primary interest here is in whether mortality models are able to capture the differences in conditional variability trends at different ages. Specifically, we investigate the extent to which the models are able to depict the growing variability in mortality conditional on survival to older ages even while characterizing the declining variability in the overall mortality distribution. Below, we examine the empirical trends in mortality using the Siler model – which describes the hazard of mortality across the full lifespan and allows for the representation of distinct improvements in early-life, later-life, and background mortality. We then employ maximum likelihood parameter estimation techniques and Markov-chain based perturbation analysis to quantify the influence of each parameter in the Siler model on the age-specific variance trends. We conclude by arguing that the slower pace and later start of survival improvements in adulthood relative to those at younger ages form the dynamics that foster the growing inequalities in survival at older ages.

The Siler Model

In 1979, William Siler proposed a mathematical model that conceived of the lifetime mortality hazard trajectory as the result of three competing but non-interacting hazard trajectories that together shape the empirical pattern of mortality in animals. Siler argued that living beings are exposed to three types of hazards throughout the lifespan: (1) a hazard that decreases from birth onward as the animal adjusts to its environment, likely as a result of maturation; (2) a constant hazard, reflecting a set of risks present in the "background" and to which the animal does not adjust over time; and (3) a hazard that increases with age, reflecting the growing risk of death as a result of senescence. Combining these three components, Siler presented an additive model that allowed for a mortality hazard that decreases in early life, remains relatively flat between later childhood and young adulthood, and then increases monotonically at older ages:

$$\mu(x) = e^{\alpha_1 - \beta_1 x} + e^{\alpha_2 + \beta_2 x} + e^{\alpha_3}. \tag{1}$$

In applying the Siler model back to human populations, contemporary demographers (e.g. Canudas-Romo and Schoen 2005, Goldstein and Wachter 2006) thus treat mortality over the life course as having three components. The first term on the right hand side of the Siler model is described as a representation of the exponentially declining mortality hazard during childhood, the second term (which is a conventional Gompertz curve) as the exponentially increasing mortality hazard in adulthood, and the third term as a flat background mortality component, reflecting the overall (non age-dependent) level of mortality in a given time. The Siler model fits as well or better than most other models to human mortality data (Gage and Dyke 1986, Gage and Mode 1993), and causes of death are accounted for by the model in a manner consistent with the biological interpretations of the the distinct hazard components (Gage 1991). **Figure 2** demonstrates how the three components additively create a bathtub shaped hazard function for mortality across the lifespan.

Note that two potential shortcomings of the Siler model are that, like the Gompertz model, it does not capture the deceleration at older ages, nor does it account for the "hump" in early adult mortality (ages 15-30) characteristic in some populations and frequently attributed to accidental and maternal mortality. Nonetheless, the Siler model of mortality hazards across the life course can represent a wider array of mortality change scenarios – including the historical patterns that saw child mortality decline before adult mortality – and is thus particularly well-suited for answering our question.

Perturbation Analysis

Trends in the variability of longevity are the result of changes in the underlying demographic parameters determining age-specific mortality. The five-parameter Siler model provides a way to describe mortality hazards using scale parameters (α's describing the level of mortality at younger ages, older ages, and overall) and age-trend parameters (β's describing the slope of the hazard trajectory at younger ages and older ages). To better understand the diverging mortality variability trends, it is useful to quantify the sensitivity of these variability measures to changes in the Siler parameters. Defined broadly, perturbation analysis is a set of mathematical tools for quantifying the change in an outcome variable in response to a change in another variable or set of variables on which the outcome depends (Caswell 1978). In demography, perturbation analysis has been used to describe the sensitivity of population growth rates to changes in the environment or in vital rates (Demetrius 1969, Keyfitz 1971, Goodman 1971, Magnus and Neudecker 1988), and the sensitivity of life expectancy to changes in age-specific mortality rates (Keyfitz 1977, Pollard 1982, Vaupel 1986, Keyfitz and Caswell 2005).

Here, we use perturbation methods to explore the sensitivity of variance measures conditional on survival to successive ages (s_a) to changes in the parameters of the Siler model of mortality across the full age spectrum. Because trends in the conditional variance measures depend on the changing distribution of mortality, perturbation analysis offers an appealing way to characterize and quantify the influence of each Siler model component on mortality variability patterns across both age and time. This type of analysis has not been done previously, and is now possible due to newly-developed matrix calculus methods based on a Markov-chain population model (Caswell 2006, 2008, 2010). The Markov chain formulation of mortality and longevity analysis assumes that individuals move through a set of transient states – in this case, ages – over their life cycle and eventually die, or, in the multi-state terminology, enter an absorbing state in which they remain thereafter. Since absorption – or mortality – is certain for all individuals, analyses of conditional longevity measures are analogous to investigating how long it takes until absorption occurs and what the distribution of absorption times is given different initial states, or ages. Matrix calculus provides a notational framework and permits the consistent differentiation of functions of scalar, vector or matrix arguments.

The transition matrix representing the probabilities of survival and mor-

tality from one age to the next can be written as

$$\mathbf{P} = \left(\begin{array}{c|c} \mathbf{U} & \mathbf{0} \\ \hline \mathbf{M} & \mathbf{I} \end{array} \right), \tag{2}$$

where $\mathbf{U}$ is the matrix of survival (or transition) probabilities, p_x, between transient states (i.e. the conditional probability of survival from age $x-1$ to age x in the life table). p_x is a column vector of survival probabilities:

$$p_x = e^{-\mu(x)} \tag{3}$$

Notably, the transition probabilities p_x that make up the matrix $\mathbf{U}$ are a direct function of the mortality hazard function $\mu(x)$, which in our case is represented by the five-parameter Siler model.

Given that the HMD life tables provide age specific probabilities of mortality and survival for 111 ages (0-110+ years), $\mathbf{U}$ can be represented as a square matrix of transition probabilities among the transient states:

$$\mathbf{U} = \begin{pmatrix} 0 & \dots & \dots & 0 \\ p_1 & & & \\ & \ddots & & \\ & & p_{110} & 0 \end{pmatrix}. \tag{4}$$

Note that the last entry of $\mathbf{U}$ is zero, as no one survives beyond the final (absorbing) age category in the life table.

$\mathbf{M}$ is the matrix of transition probabilities from transient states to the absorbing state (i.e. the probability of mortality at age x), and can be written as

$$\mathbf{M} = \begin{pmatrix} 1-p_1 & & & \\ & 1-p_2 & & \\ & & \ddots & \\ & & & 1 \end{pmatrix}. \tag{5}$$

Note that $\mathbf{M}$ has the same number of columns (representing starting ages) as $\mathbf{U}$, but one less row (representing ages reached), as all members of the life table "survive" into the initial age category.

$\mathbf{I}$ gives the probability of remaining in the absorbing state, and $\mathbf{0}$ gives the probability of moving from an absorbing state to a transient state. Since the absorbing state is death, the probability of remaining in the absorbing state is 1 (represented by the identity matrix, $\mathbf{I}$), and the probability of leaving the absorbing state, $\mathbf{0}$, is zero.

Within this framework, remaining life expectancy at any age x may be analyzed in terms of how long it takes a person in that state x to enter the absorbing state. If ν_{ix} represents the number of visits to state i by someone in state x before death, then the fundamental matrix $\mathbf{N}$ represents the expected length of stay in each transient state:

$$\mathbf{N} = E(\nu_{ix}) = (\mathbf{I} - \mathbf{U})^{-1} \tag{6}$$

Thus, if η_x is the number of visits to all transient states by an individual in state x prior to absorption, then the life expectancy of an individual aged x is the expectation of η_x. The expected longevity at all ages in the life table may be obtained as the column sums of $\mathbf{N}$:

$$E(\boldsymbol{\eta})^{\mathsf{T}} = \mathbf{1}^{\mathsf{T}}\mathbf{N}, \tag{7}$$

where $\mathbf{1}$ is a column vector of ones.

Similarly, under the Markov formulation, $V(\boldsymbol{\eta})^{\mathsf{T}}$ represents the vector of variances for longevity conditional on survival to each age:

$$V(\boldsymbol{\eta})^{\mathsf{T}} = \mathbf{1}^{\mathsf{T}}\mathbf{N}(2\mathbf{N} - \mathbf{I}) - E(\boldsymbol{\eta})^{\mathsf{T}} \circ E(\boldsymbol{\eta})^{\mathsf{T}}. \tag{8}$$

Note that this equation is based on the classic definition of variance $Var[X] = E[X^2] - (E[X])^2$, and $\circ$ denotes the element by element multiplication of the two life expectancy matrices.

By taking the derivative of the variance and applying the chain rule successively to link $\mathbf{U}$ with the Siler hazard model, we can obtain the sensitivity of the vector of conditional variances to changes in the five Siler parameters $\boldsymbol{\theta} = \{\alpha_1, \beta_1, \alpha_2, \beta_2, \alpha_3\}$:

$$\begin{aligned}\frac{dV(\boldsymbol{\eta})}{d\boldsymbol{\theta}^{\mathsf{T}}} = \quad & [2(\mathbf{N}^{\mathsf{T}} \otimes \mathbf{1}^{\mathsf{T}}) + 2(\mathbf{I} \otimes \mathbf{1}^{\mathsf{T}}\mathbf{N}) - (\mathbf{I} \otimes \mathbf{1}^{\mathsf{T}}) - 2\left(\operatorname{diag}\left(E(\boldsymbol{\eta})\right) \otimes \mathbf{1}^{\mathsf{T}}\right)] \\ & \times (\mathbf{N}^{\mathsf{T}} \otimes \mathbf{N}) \tfrac{d\operatorname{vec}\mathbf{U}}{d\boldsymbol{\theta}^{\mathsf{T}}},\end{aligned} \tag{9}$$

Note that the vec function stacks the columns of a matrix into a single vector, and $\otimes$ denotes a Kronecker product.

The standard deviation conditional on survival to each age is the square root of the variance taken element-wise and its sensitivity is represented as:

$$\frac{dSD(\boldsymbol{\eta})}{d\boldsymbol{\theta}^{\mathsf{T}}} = \frac{1}{2}\operatorname{diag}\left(SD(\boldsymbol{\eta})\right)^{-1}\frac{dV(\boldsymbol{\eta})}{d\boldsymbol{\theta}^{\mathsf{T}}}. \tag{10}$$

Note that $SD(\boldsymbol{\eta})$ is analogous to the s_a notation used above to describe the standard deviation conditional on survival to age a.

Now, returning to the Siler model specifically and recalling the relationships above, $d\text{vec}\,\mathbf{U}$ can be written as:

$$d\text{vec}\,\mathbf{U} = \text{diag}\,(\text{vec}\,\mathbf{J})(\mathbf{1} \otimes \mathbf{I})dp \tag{11}$$

where dp, the derivative of each transition probability, is in turn a function of the derivative of the mortality hazard (a Siler trajectory in this case) with respect to a set of parameters $\boldsymbol{\theta}$:

$$dp = -\text{diag}\,(p)\frac{d\mu}{d\boldsymbol{\theta}^{\intercal}}, \tag{12}$$

with $\mathbf{J}$ denoting a square matrix with ones on the sub-diagonal and zeros elsewhere.

Finally, to obtain the derivative of the Siler hazard function with respect to the set of all parameters, $\frac{d\mu}{d\boldsymbol{\theta}^{\intercal}}$, we rewrite the three component Siler model as:

$$\mu(x) = e^{w_1} + e^{w_2} + e^{w_3}, \tag{13}$$

with the corresponding derivatives:

$$d\mu = e^{w_1}dw_1 + e^{w_2}dw_2 + e^{w_3}dw_3, \tag{14}$$

where

$$\begin{aligned} dw_1 &= d\alpha_1 - (d\beta_1)x \\ dw_2 &= d\alpha_2 + (d\beta_2)x \\ dw_3 &= d\alpha_3. \end{aligned} \tag{15}$$

Using the chain rule, this perturbation analysis allows us to determine and quantify the sensitivity of our outcome of interest (the standard deviation of the mortality distribution conditional on survival to successive ages) to unit changes in each of the five Siler hazard model parameters. The question we aim to answer with this analysis is whether the pattern of mortality improvement over time – as reflected in the Siler model parameters – differentially affects the trends in mortality variability for the young and the old.

Sensitivity of Variability Measures to Mortality Change

Maximum likelihood estimation (MLE) procedures allow us to simultaneously characterize all the parameters of the four mortality models under

consideration. Applied successively to each year of data, the MLE procedure can generate parameter trends that offer an added perspective on the demographic transition.

Figure 3 presents maximum likelihood estimates for the 5 parameters of the Siler model. All three α parameters, representing the overall levels of child, adult, and background mortality hazards show a decline over time. However, while the overall levels of child and background mortality (α_1 and α_3, respectively) have declined in a relatively linear fashion, the α_2 trend indicates a period of slightly increased adult mortality between 1900-1930 before the beginning of a subsequent linear decline. The magnitude of α_3 (background) at any year is greater than that of α_1 (child mortality) but less than that of α_2 (adult mortality).

The β_1 parameter, representing the age-trajectory of mortality decline in childhood, has increased over time, suggesting a faster pace of decline in the mortality hazard during the first years of life – a result consistent with the known improvements in infant survival. The Trend in β_2, the slope of the age trajectory of adult mortality hazard, is somewhat less intuitive. Mirroring the α_2 trend, the parameter value declines between 1900-1930, followed by a subsequent increase, indicating (as above) that the slope of the adult hazard trajectory may have grown steeper over time even as age-specific mortality hazards declined. The absolute magnitude of the change in the β parameters is very modest and considerably smaller than the change in the α parameters. This is particularly true for β_2, whose absolute magnitude is lowest.

Perturbation analysis offers a precise way to characterize and quantify the sensitivity of age-specific variance measures (and their trends over time) to changes in all model parameters using derivatives. As can be seen in figure 4 and figure 5, the Siler components differ in their influence on the pattern of variability by both age and time.

Figure 4 presents the sensitivity of s_a (where a ranges from 0 to 110) to a change in each Siler parameter for 6 selected years. Notably, all five Siler parameters have a consistently positive (α_1, α_3) or consistently negative (α_2, β_2, β_1) effect. The patterns for the parameters representing infant mortality (α_1 and β_1, left column) mirror each other, as the sensitivity to α_1 declines with age and the sensitivity to β_1 increases, with both approaching 0 as age increases. The two parameters are only influential for those variability measures that include the complete distribution and survivors to the first few years of life but not for measures conditional on survival to subsequent ages. The symmetrical pattern of the two parameters means that either an increase in the child mortality level or a decrease in age-trajectory of the

mortality hazard will increase overall mortality variability (s_0) or variability conditional on survival to very young ages. Notably, while in 1900 the magnitude of the sensitivity for both α_1 and β_1 was relatively high and each parameter's impact extended up to age 5, by later years the sensitivity declined markedly, and the two parameters' influence was confined to the first year of life.

In contrast, the parallel influences of α_2 and β_2, the two parameters defining the trajectory of mortality hazards in adulthood, extend across the full lifespan. Note that at all ages the sensitivity is negative, suggesting that an increase in either the overall level or age-trajectory of adult mortality would reduce variability, and, conversely, improved survival in adulthood will increase the age-specific variation. The sensitivity of the standard deviation of the mortality distribution at any age to β_2 is nearly an order of magnitude greater than its sensitivity to α_2, though the absolute magnitude of β_2 and its change over time is much smaller than that of α_2.

In 1900, the sensitivity of s_a to changes in both α_2 and β_2 had a high negative magnitude for measures conditional on survival to early ages, and approached 0 at higher ages. For both parameters, the sensitivity slope was steeper at older ages than it was at younger ages, with a plateau between (roughly) ages 50 and 70. Throughout the 20th century, the sensitivity to both parameters at younger ages declined (approaching zero), while the sensitivity at older ages increased slightly. The result, evident by the second half of the twentieth century, was a flat age-trajectory of sensitivity up to age 50, relatively more sensitivity for measures conditional on survival to age 50-75, and finally a steep reversal for measures conditional on survival to older ages.

The visual display of the sensitivity of s_a to α_3, the parameter determining background or overall mortality level, underscores the importance of this component to shaping the variability trends. The sensitivity is highest at young ages (particularly under 10) and approaches zero at older ages. This sensitivity has declined over time, but remains appreciable at young ages. Notably, at most ages, the conditional variance is more sensitive to changes in α_3 than changes in any of the other parameters, with the exception of β_2.

For another perspective on the relationship between the Siler parameters and conditional variability, **figure 5** presents the trends over time (1900-2007) in the sensitivity of the standard deviation of mortality for survivors at selected ages (0, 10, 50, and 75) to changes in each model parameter. Trends in the sensitivities of the unconditional standard deviation (s_0) to each parameter display clear differences between values in the first and second half of the twentieth century. For example, while the childhood

mortality parameters α_1 and β_1 were quite influential for s_0 in 1900, after six decades of steep decline it has remained relatively stable at very low levels since about 1960. A similarly steep decline followed by stable lower rate is apparent in the sensitivity of s_0 to α_3, the background mortality component, whose sensitivity trends display a parallel pattern to the time trends in the conditional variability measures themselves (see Engelman et al. 2010). The sensitivity of s_0 to the two adult mortality parameters, α_2 and β_2, also shows two distinct phases, where the negative sensitivity approaches zero at a rapid pace during the first part of the century, and then stabilizes (or continues to decline slightly) during the latter part of the century. Trends for the sensitivity of s_{10} to the same parameters follow a roughly similar pattern, consistent with the idea that the main advances in reducing childhood mortality took place in the first half of the twentieth century.

s_{50} and s_{75} are not sensitive to the childhood mortality parameters, and s_{50} (but not s_{75}) shows slight responsiveness to α_3 early in the twentieth century, but this sensitivity wanes over the decades. The main contrast between the two measures is apparent in their sensitivities to the adult mortality parameters α_2 and β_2, which were similar in 1900, but have diverged substantially since. The relative stability (despite fluctuations) in the sensitivity of s_{75} to the two parameters over time may seem counter-intuitive given the observed rise in mortality heterogeneity at older ages, but it is worth noting that this relative stability stands in contrast to the declining sensitivity in measures conditional on survival to younger ages: while s_{75} was relatively *less* sensitive to α_2 and β_2 in the first part of the twentieth century than s_0, s_{10}, and s_{50}, the mid-century cross-over has resulted in a *greater* contemporary sensitivity of s_{75} to α_2 and β_2 relative to its counterpart measures.

Discussion

An examination of trends in the Maximum-likelihood parameter estimates for Siler model indicates that the parameter representing the age-slope of mortality (β_2) is indeed increasing, suggesting the slope is getting steeper over time. This steeper slope also appears to be associated with the growing variance of the mortality distribution at older ages, a trend that has persisted despite the concomitant decline in the α levels characterizing adult mortality.

At first consideration, the change over time in the slope of the age-trajectory of mortality – even though small in magnitude – appears puzzling and counter-intuitive. For one, the early declines in the slope parameter take place in the first decades of the 20th century – before the notable declines in

adult mortality. At the same time, by the later decades in the century, when adult mortality was known to be declining, the slope began to grow steeper, potentially suggesting a higher rate of mortality hazard increase with age. The reason that this gradually steepening slope does not result in rising, rather than declining age-specific mortality is the faster pace of decline in the α-type intercept parameters representing overall mortality levels. But why are β and β_2 increasing?

One potential explanation is that for the cohorts aging through this period of demographic and epidemiologic change, progress in reducing mortality in adulthood has not fully counterbalanced the effect of improved survival in early life. We previously suggested that as a result of ongoing survival improvements at younger ages, delayed mortality selection has pushed health disparities from early to later life, where they manifest in the growing inequalities in late-life mortality (Engelman et al. 2010). Our current findings add support for the hypothesis that the empirical trends in mortality variation reflect changes in the underlying composition and health status of successive aging cohorts. If a growing number of frail people (who experience a higher mortality hazard at every age relative to their more robust counterparts) survives to every age over time, then, in line with the theory of heterogeneity (Vaupel et al. 1979, Vaupel and Yashin 1985), the observed mortality trajectory would reflect a constant or even slightly increased hazard at every age. The increase we observe in the β parameters over time may thus reflect a "failure of success."

As the parameters defining the trajectory of mortality change over time, so does the sensitivity of each age-specific variability measure. Perturbation analysis enabled us to quantify this relationship, showing the differential responses of variability measure conditional on survival to younger and older ages to the parameters defining the course of adult and background mortality levels. The third Siler model component, which is not considered to be age-dependent, nonetheless proved essential for the reproduction of the divergence, by age, of variability trends in our simulations. The perturbation analysis showed it to be among the most influential parameters in the Siler model in terms of defining the age-specific variability level for the young and middle-aged alike. Our results indicate that the timing and magnitude of declines in adult and background mortality influence the increased variability trend for survivors to adulthood and older ages and its divergence from the observed decline in variability for the complete distribution of ages at death. Thus, it is the slower pace and later start of survival improvements in adulthood relative to those at younger ages which form the dynamics that manifest in the growing inequalities in survival at older ages.

References

[1] Canudas-Romo, V. and R. Schoen. 2005. Age-specific contributions to changes in the period and cohort life expectancy. *Demographic Research* 13(3):63-83.

[2] Caswell, H. 1978. A general formula for the sensitivity of population growth rate to changes in life history parameters. *Theoretical Population Biology* 14:215-230.

[3] Caswell, H. 2006. Applications of Markov chains in demography in A.N Langville and W.G. Stewart (editors), *MAM2006: Markov Anniversary Meeting*. Raleigh, North Carolina: Boson Books, pp. 319-334.

[4] Caswell, H. 2008. Perturbation analysis of nonlinear matrix population models. *Demographic Research* 18:59-116.

[5] Caswell, H. 2010. Perturbation analysis of longevity using matrix calculus. Paper presented at the 2010 meeting of the Population Association of America, Dallas, Texas.

[6] Cheung, S.L.K., J.M. Robine, E.J.C. Tu, and G. Caselli. 2005. Three dimensions of the survival curve: horizontalization, verticalization, and longevity extension. *Demography* 42(2): 243-252.

[7] Demetrius, L. 1969. The sensitivity of population growth rate to perturbations in the life cycle components. *Mathematical Biosciences* 4:129-136.

[8] Engelman, M., Canudas-Romo, V., Agree, E.M. 2010. The influence of increased survivorship on mortality variation among aging populations. *Population and Development Review* 36(3):511-539.

[9] Fries, J.F. 1980. Aging, natural death, and the compression of morbidity. *New England Journal of Medicine* 303: 130-135.

[10] Gage, T.B. 1991. Causes of death and the components of mortality: Testing the biological interpretation of a competing hazards model. *American Journal of Human Biology* 3:289-300.

[11] Gage, T.B., and Dyke, B. 1986. Parameterizing abridged mortality tables: The Siler three-component hazard model. *Human Biology* 58:275-291.

[12] Gage, T.B., and Mode, C.J. 1993. Some laws of mortality: How well do they fit? *Human Biology* 65:445-461.

[13] Goldstein, J.R., and K.W. Wachter. 2006. Relationships between period and cohort life expectancy: Gaps and lags. *Population Studies* 60(3): 257-269.

[14] Goodman, L.A. 1971. On the sensitivity of the intrinsic growth rate to changes in the age-specific birth and death rates. *Theoretical Population Biology* 2:339-354.

[15] The Human Mortality Database (University of California, Berkeley, and Max Planck Institute for Demographic Research, Germany) provides comprehensive mortality and population data for industrialized countries. (Data downloaded 7/1/2009, www.mortality.org).

[16] Keyfitz, N. 1971. Linkages of intrinsic to age-specific rates. *Journal of the American Statistical Association* 66:275281.

[17] Keyfitz, N. 1977. *Applied mathematical demography.* New York: Wiley.

[18] Keyfitz, N. and H. Caswell. 2005. *Applied mathematical demography.* Third edition. New York, NY: Springer-Verlag.

[19] Magnus, J.R. and H. Neudecker. 1988. *Matrix differential calculus with applications in statistics and econometrics.* New York: John Wiley & Sons.

[20] McKeown, T. 1976. *The Modern Rise of Population.* New York: Academic Press.

[21] Pollard, J.H. 1982. The expectation of life and its relationship to mortality. *Journal of the Institute of Actuaries* 109:225240.

[22] Rau, R. E. Soroko, D. Jasilionis, and J.W. Vaupel. 2008. Continued reductions in mortality at advanced ages. *Population and Development Review* 34(4):747-768.

[23] Siler, W. 1979. A competing risk model for animal mortality. *Ecology* 60(4):750-757.

[24] Szreter, S. 2004. Industrialization and health. *British Medical Bulletin* 69:75-86.

[25] Vaupel, J.W. 1986. How change in age-specific mortality affects life expectancy. *Population Studies* 40:147157.

[26] Vaupel, J.W., Manton K.G., Stallard E. 1979. The impact of heterogeneity in individual frailty on the dynamics of mortality. *Demography* 16(3): 439-454.

[27] Vaupel, J.W. and A. I. Yashin 1985. Heterogeneity's ruses: Some surprising effects of selection on population dynamics. *The American Statistician* 39(3); 176-185.

[28] Wilmoth, J.R. and S. Horiuchi. 1999. Rectangularization revisited: Variability of age at death within human populations. *Demography* 36(4):475-495.

[29] Wilmoth, J.R., L. J. Deegan, H. Lundstrm, and S. Horiuchi. 2001. Increase in maximum life span in Sweden, 1861-1999. *Science* 289:2366-2368.

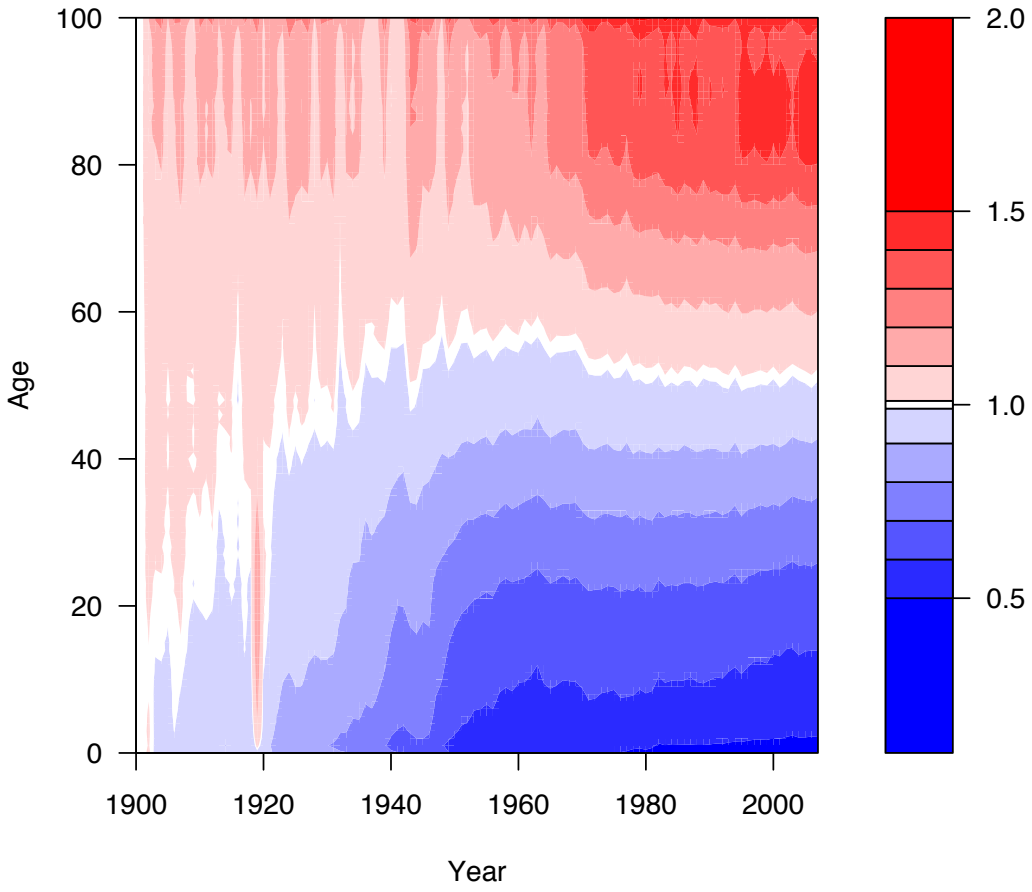

Figure 1: **Trends in age-specific standard deviations of the mortality distribution for Swedish females, 1900-2006.** Color is assigned according to the ratio of the standard deviation in the distribution of mortality for survivors to a given age (y-axis) in a given year (x-axis), relative to the age-specific value in 1900. White represents a ratio of 1 (no change); successively darker blues represent declining values < 1; successively darker reds represent increasing values > 1. *Source:* HMD 2009 and Engelman et al. 2010.

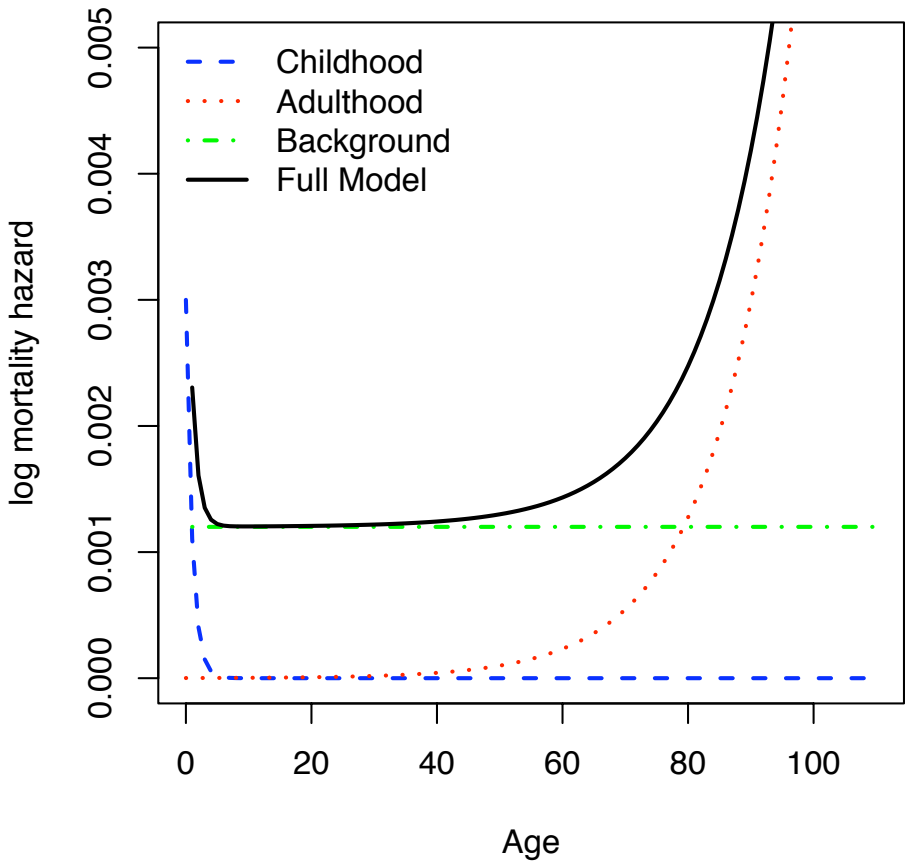

Figure 2: **The three-component Siler model**: $\mu(x) = e^{\alpha_1 - \beta_1 x} + e^{\alpha_2 + \beta_2 x} + e^{\alpha_3}$, where the first term on the right represents the mortality pattern dominant in childhood, the second term represents the mortality pattern dominant in adulthood, and the third term represents a background mortality level. Created with parameter values: $\alpha_1 = -2.4$, $\beta_1 = 0.6$, $\alpha_2 = -9.5$, $\beta_2 = 0.09$, and $\alpha_3 = -6.9$.

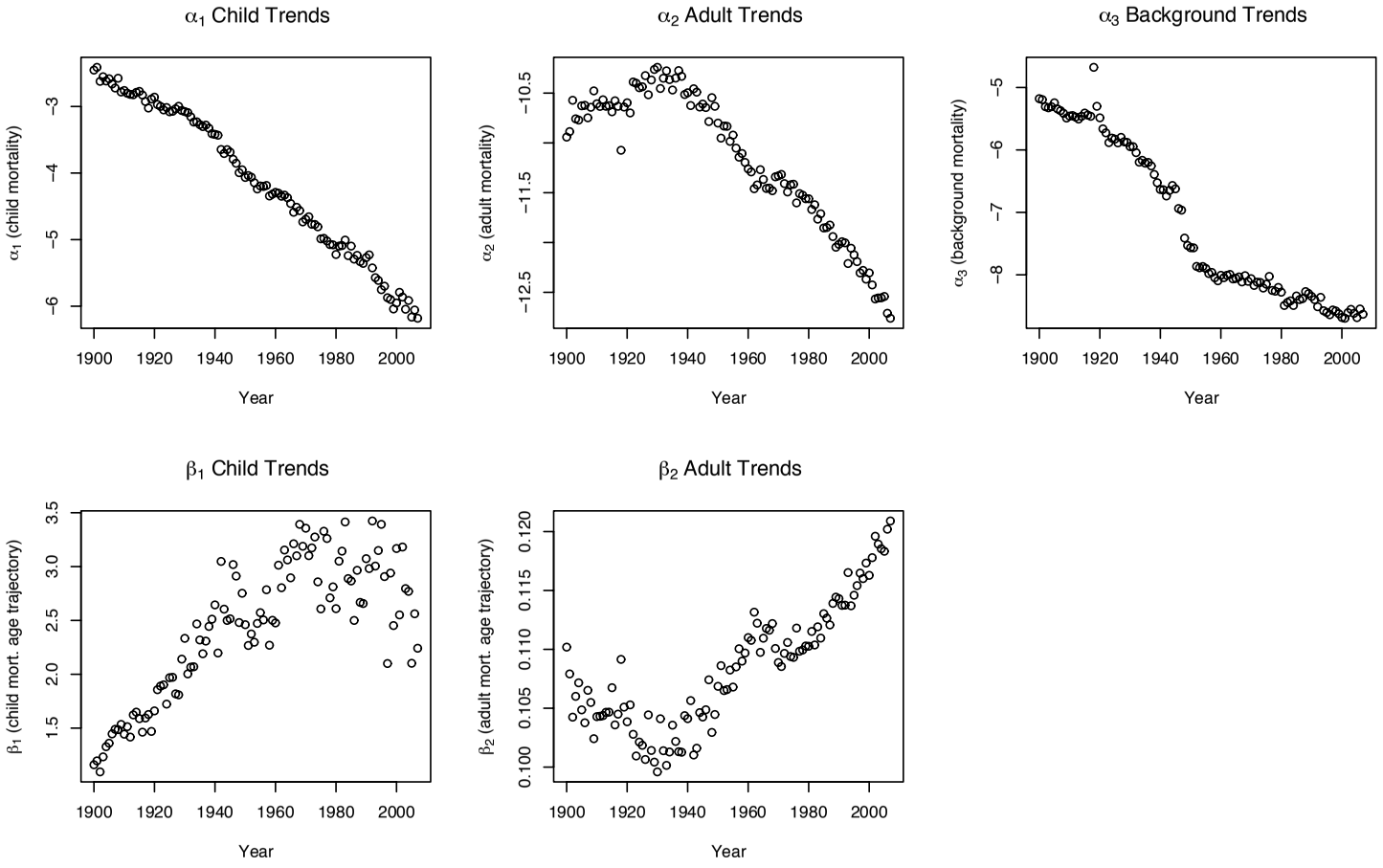

Figure 3: **Trends in maximum likelihood parameter estimates for the Siler model.** Note that the child mortality parameter β_1 is negative in the Siler equation, while all other parameters are positive. Based on life tables for Swedish females 1900-2007.

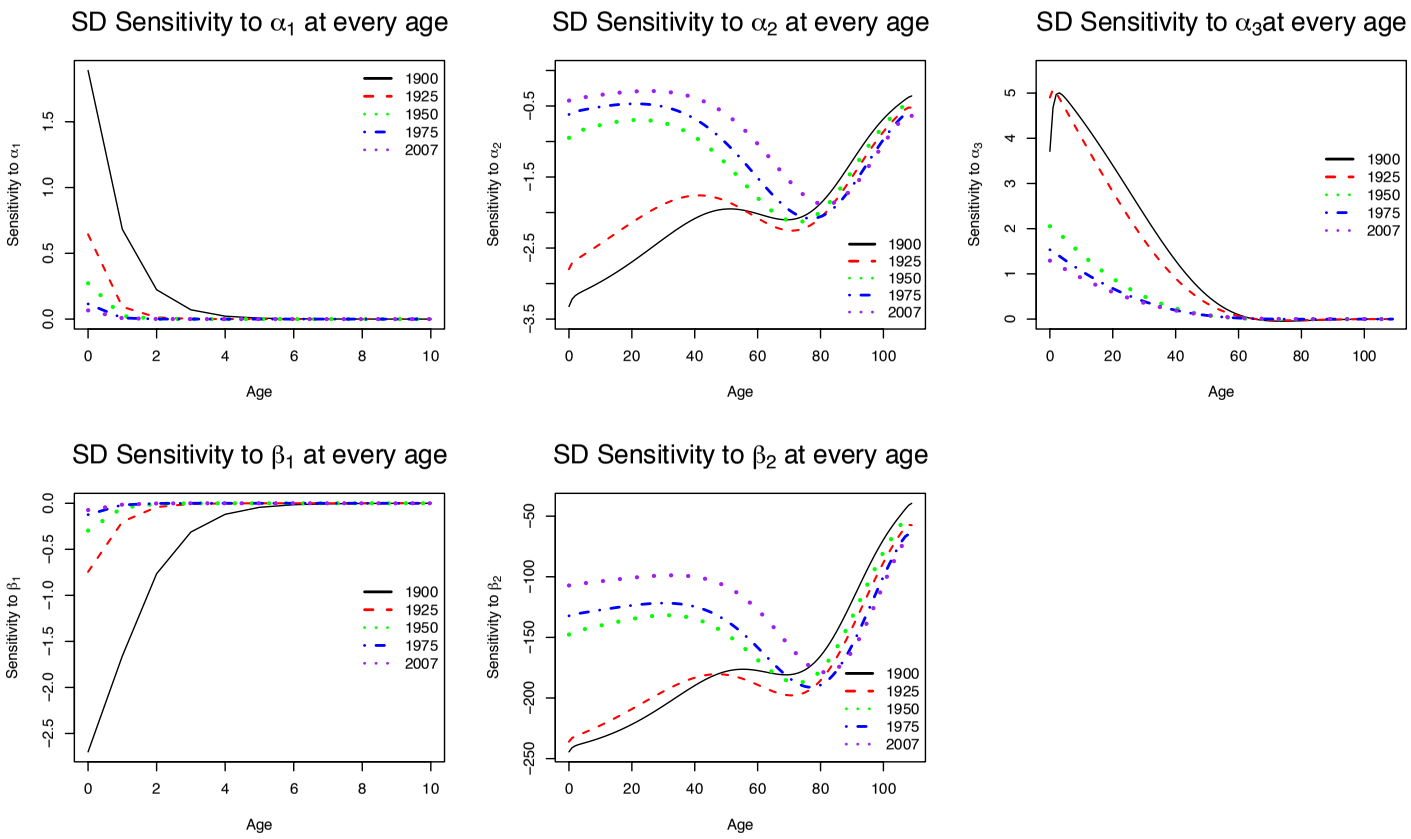

Figure 4: **Sensitivity of all age-specific (conditional) standard deviation measures to unit changes in each Siler model parameter for selected years.**

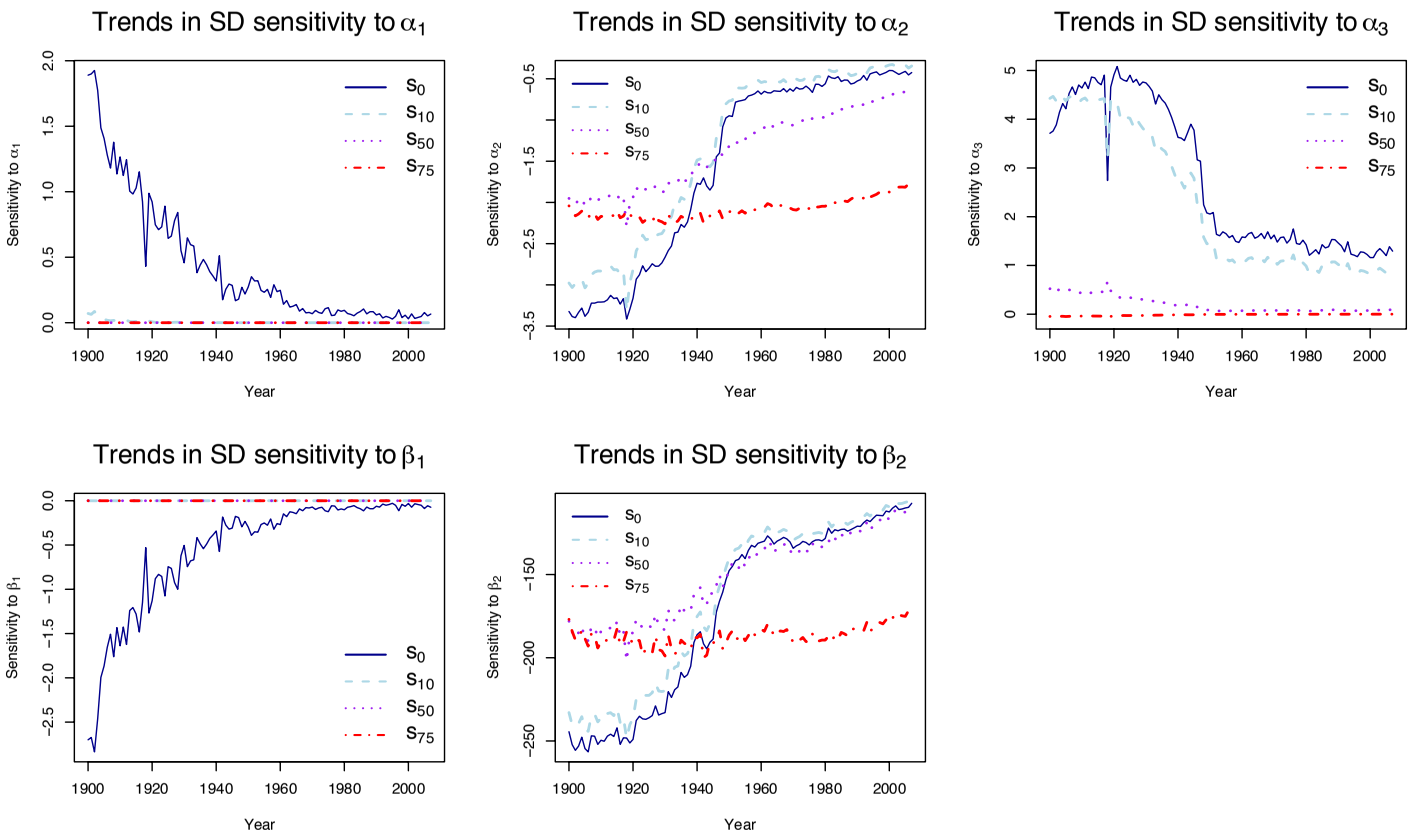

Figure 5: **Trends in the sensitivity of conditional standard variances to unit changes in each Siler model parameter for selected ages, 1900-2007.**