A Two-Process Mortality Model with Extensions to Juvenile Mortality, Population Dynamics and Evolution

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Population Association of America Meeting, 2015

Abstract

A vitality framework partitions mortality into intrinsic and extrinsic parts representing age-dependent loss of survival capacity and environmental challenges to the capacity. We extend a previous 4-parameter model suitable to modeling adult mortality to a 6-parameter form that includes vitality challenges to early life and thus is suitable for modeling survival from birth. The framework merges force of mortality and stochastic vitality models and shifts the perspective from explaining how the force of mortality changes with age to how the underlying intrinsic and extrinsic vitality processes change. Extensions of the model to population ecology and natural selection are discussed.

Keywords: mortality, two-process model, vitality, survivorship, juvenile survival

Introduction

Survival in human and animal populations is formed by processes acting on a multitude of scales from high frequency environmental challenges, to intermediate frequencies that characterize life history strategies and to the low frequency processes shaping species genomes. A challenge in biodemography is to develop models that capture contributions of these processes in shaping patterns of survival across taxa and in particular the patterns in human populations (Wachter and Finch 1997). While a number of models address individual scales, such as population level changes in abundance or age-dependent patterns of survivorship, the models are often based on different principles and incorporate different dimensions which precludes a clear understanding of their commonalities as well as differences. In this paper we begin with a brief review of several important approaches to modeling survivorship. We then illustrate their differences and how they fit into a tractable framework for characterizing survival in terms of population and individual level processes across multiple temporal scales.

We begin with the premise that survivorship models are all extensions of the exponential equation characterizing the decline of identical individuals subjected to a constant mortality rate or force of mortality, μ. The rate of change of survival is $dS/dx = -\mu S$ and age-dependent survivorship is $S = \exp(-\mu x)$. Of course individuals are neither identical nor do they experience constant mortality and deviations from a pure exponential curve is a result of violations of the assumption of a homogeneous population and a uniform mortality rate. Areas of study concerned with survival refine the model in different ways. A predominant class of models, common in ecology, assumes a homogeneous population and characterizes changes in the mortality rate in terms of resources and predators. The classic examples are the logistic equation proposed by Verhulst which relates mortality to the fraction of population carrying capacity K as $\mu \sim S/K$ and the Lotka-Volterra equation which relates prey mortality to predator density P as $\mu \sim P$. Models common to demography, again assume a homogeneous population and characterize the mortality rate as a function of age, not population density as is done in ecological models. The classic Gompertz (1825) model characterizes the mortality rate of adults as an exponentially increasing function of age giving $\mu = a\exp(-bx)$. A number of additional terms have been added to the model to better fit the deviations of the mortality rate with age. Notable examples include a background

mortality rate (Makeham 1890), adjustments for childhood mortality (Siler 1979) and young adult risk taking (Heligman 1980).

The assumption of heterogeneity of individuals within the population has been addressed in two ways. In a demographic context Vaupel et al. (1979) expressed heterogeneity as an adjustment to the force of mortality through the idea that individuals are born with a fixed level of frailty that affects their survival. Assuming frailty in the population has a gamma density distribution that scales the Gompertz *a* parameter, the mortality rate decelerates at old age due to the elimination of frailer individuals. Frailty models consider the unobserved lifetime distribution of heterogeneity which can be formulated in a variety of ways. The gamma-Gompertz model with gamma-distributed frailty is perhaps the most important form. A second approach to modeling heterogeneity dispenses with the traditional concept of the mortality rate as a fundamental property and characterizes mortality as the absorption of age-declining vitality, a measure of survival capacity, into a zero boundary representing death. Denoted a process point of view (Aalen and Gjessing 2001), in its most common form vitality is represented as a stochastic Wiener process characterized by a mean rate of loss and a stochastic variability on the rate. The mortality rate is defined by the first passage time of vitality to the zero boundary (Anderson 2000, Aalen and Gjessing 2001, Weitz and Fraser 2001). Vitality models thus characterize the mortality rate in terms of the stochastic dynamics of vitality, which only yield closed form solutions if the mean and variability in the vitality rate are constant with age and the initial vitality is constant. However, analytical approximations can be developed for initial distributions of vitality (Li and Anderson 2009). Nonlinear forms of vitality decline have also been proposed (Skiadas and Skiadas 2010).

The force of mortality and process point of view appear on the surface to be distinct and possibly incompatible approaches to modeling survivorship. However, they can be merged into a common framework by partitioning mortality in distinct intrinsic and extrinsic forms that capture the demarcation of mortality resulting from chronic processes associated with aging and acute processes typically associated with infectious disease and accidental death (Carnes and Olshansky 1997, Carnes et al. 2006). The merged framework (Li and Anderson 2013) characterizes the absorption of stochastic vitality into the zero boundary as intrinsic mortality and environmental challenges to the remaining vitality at age as extrinsic mortality. Thus, intrinsic mortality is represented by the end point of the cumulative loss of survival capacity and extrinsic mortality is represented as an acute challenge exceeding the remaining vitality. Li and Anderson

(2013) illustrated extrinsic challenges to stochastic vitality trajectories preferentially eliminate lower vitality paths which reshapes the distribution of vitality. While this model does not yield a closed solution, by expressing the extrinsic mortality process as challenges to the mean rate of loss of vitality, a closed form is obtained. Furthermore, parameter estimates of the approximate model fit to data generated by simulations of the full model are within 10% of the true parameters.

As noted by (Li and Anderson 2013, 2015), challenges to remaining average vitality can be viewed in the Strehler and Mildvan (1960) interpretation of the Gompertz model in which an exponentially increasing mortality rate is produced by random challenges to a linearly declining vitality. Thus, the resulting two-process vitality model, with distinct intrinsic and extrinsic processes, combines the stochastic vitality model and Gompertz model and thus merges heterogeneity and age-dependent classes of survival models into a single form. However, the model is limited because it does not address other important processes that shape survivorship curves, such as childhood mortality. Nor has the two-process model been considered in the context of population level processes. The following section expands the model with juvenile or ontogenescent mortality and discusses how other age-dependent and population processes can be addressed.

Model formulation

Building on the two-process model (Li and Anderson 2013), first define independent intrinsic and extrinsic mortality processes. As developed previously, intrinsic mortality results from the exhaustion of vitality. Next, separate extrinsic mortality into independent juvenile and adult components. Then subscripting the intrinsic process, *i*, and adult and juvenile extrinsic processes (*e*, *a*) and (*e*, *j*) respectively the total mortality rate is

$$\mu(x) = \mu_i(x) + \mu_{e,a}(x) + \mu_{e,j}(x) \tag{1}$$

Figure 1 illustrates juvenile and adult vitalities and challenges. Intrinsic mortality results when a stochastic vitality trajectory reaches the zero boundary. Extrinsic adult mortality results when a random environmental challenge exceeds the mean age-dependent adult vitality. Extrinsic juvenile mortality results when a random environmental or developmental challenge exceeds the mean age-dependent juvenile vitality.

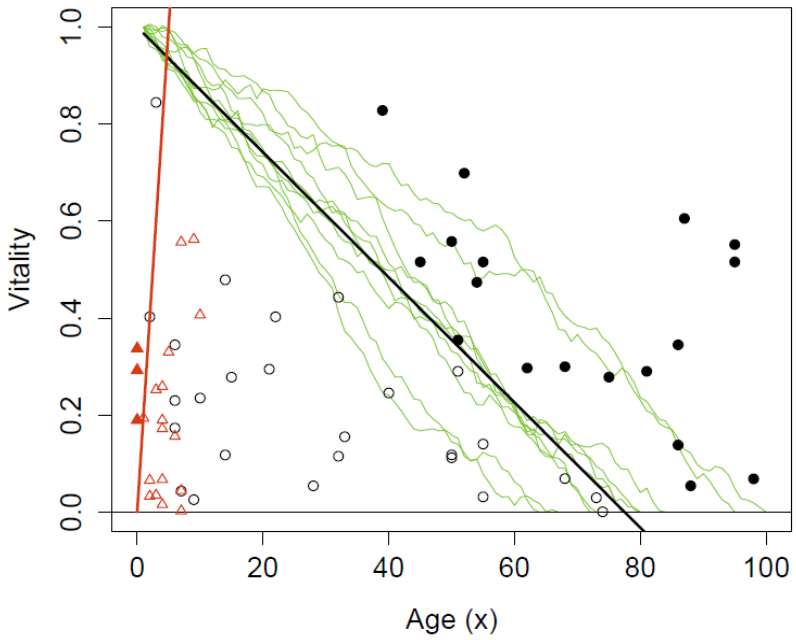

Fig. 1. Patterns of vitality and challenges for 10 numerical simulations. Arrival of stochastic vitality trajectories (green lines) to the zero-vitality boundary demark intrinsic mortality events. Adult challenges (filled black points) exceeding the deterministic vitality (black line) represented adult extrinsic mortality. Challenges less than the deterministic vitality (open points) represent non-lethal event. Deterministic juvenile vitality begins at age zero and increases (red line). Red triangles represent juvenile challenges. Filled triangles depict juvenile extrinsic mortality events and open triangles depict non-lethal challenges. Model parameters generating curves correspond to period data for Swedish females in 1900: r = 0.0129, s = 0.0126, λ = 0.045, β = 0.40, γ = 0.15, α = 1.

Intrinsic mortality

Following (Anderson et al. 2008) define the stochastic loss of vitality as an abstract index of adult survival capacity starting at initial value v_0 and declining by cell degradation processes such as production of reactive oxygen species in respiration (Beckman and Ames 1998, Yin and Chen 2005) and telomere attrition in cell replication (Aubert and Lansdorp 2008). The rate of loss of vitality described by a Wiener process becomes

$$\frac{dv}{dx} = -\rho + \sigma\varepsilon_x \qquad (2)$$

where ε_x is a white noise process, ρ is the mean rate of loss of vitality in a population and σ is the variability in the rate. Intrinsic mortality occurs in an individual when its vitality trajectory reaches the zero-vitality boundary representing death. The probability distribution of first-passage time of vitality to the death boundary is defined by the inverse Gaussian distribution (Chhikara and Folks 1989) as

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

where the normalized deterministic rate and stochastic intensity of vitality loss are $r = \rho / v_0$, $s = \sigma / v_0$. The intrinsic survival to age *x*, characterized by the fraction of vitality trajectories in a cohort that have not yet reached the death boundary, is

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

and the intrinsic mortality rate is

$$\mu_i(x) = f(x)/l(x) \tag{3}$$

Note by assuming a unit initial vitality the model does not account for heterogeneity in the initial vitality distribution. However, the model captures the effects of initial heterogeneity in the evolving heterogeneity expressed by *s* (Li and Anderson 2009). In practice, it is difficult to resolve the contributions of initial and evolving heterogeneity to vitality trajectories. In fact, any pattern of first arrival time can be generated by a combination of initial and evolving heterogeneities (Steinsaltz and Evans 2004). Decomposing contributions of initial and evolving heterogeneities to intrinsic mortality requires specifying the initial distribution form (e.g. Li and Anderson 2009).

Extrinsic adult mortality

Following (Li and Anderson 2013) characterize extrinsic mortality as the result of random challenges exceeding adult vitality. Challenge frequency λ is characterized by a homogeneous Poisson distribution such that a challenge at age *x* does not depend on the previous history of challenges (Finkelstein 2012). Challenge magnitude *Z* is a random process with an exponential distribution and mean magnitude βv_0 where β scales challenges relative to the initial vitality. The challenge cumulative distribution becomes

$$\varphi(z) = 1 - \mathrm{e}^{-z/v_0\beta} \tag{4}$$

To formally define the extrinsic mortality rate denote $v(x)$ the realization of the random vitality process at age *x*, then the conditional extrinsic mortality rate is

$$m_e\left(x \mid v = v(x) \right) = \lambda \Pr\left[Z > v(x) \right] = \lambda\left(1 - \varphi(v(x)) \right) = \lambda e^{-v(x)/v_0\beta} \tag{5}$$

Integrating over possible vitality states, the population-level extrinsic mortality rate is

$$\mu_e(x) = \int_0^\infty m_e(x \mid v) g(v) dv \tag{6}$$

where the age-dependent distribution of vitality in the population $g(v)$ depends on both the loss of vitality through the stochastic process and its modification by the preferential elimination of low-vitality individuals because of extrinsic challenges. Therefore, $g(v)$ changes by both intrinsic and extrinsic processes and it has no simple closed form solution. To obtained a closed form approximation to eq. (6) Li and Anderson (2013) represented the vitality being challenged as deterministic, i.e. $\sigma = 0$. Then the extrinsic mortality rate is approximated

$$\mu_e(x) = \lambda e^{-(v_0 - \rho x)/v_0 \beta} = \lambda e^{-(1-rx)/\beta} \tag{7}$$

Note that the linear approximation of vitality gives the Strehler and Mildvan (1960) interpretation of the Gompertz law: $\mu = a \exp(-bx)$ with $a = \lambda e^{-1/\beta}$ and $-b = \rho / v_0 \beta = r/\beta$ (Li and Anderson 2013, 2015).

Extrinsic juvenile mortality

Across nearly all species, juvenile stage mortality rates are high but rapidly decline as the individual matures. This phenomena has been termed ontogenescent mortality to distinguish it from the increasing senescent mortality in adults (Levitis 2011). Here we use the terms ontogenescent and juvenile interchangeably according to emphasis on the process or the life stage. Ontogenescent mortality is thought to involve a number of processes including increasing robustness and size (Levitis 2011), immune function development (Chandra 1997, Holt and Jones 2000), the decline in early life transcriptional developmental and environmental transitions (transitional timing hypotheses) and the decline in risk taking as animal size increases (growth tradeoff hypothesis) (Levitis 2011). Importantly, ontogenescent mortality involves environmental challenges, for example the leading cause of death for children is infectious disease including acute respiratory infections, diarrhea, malaria, etc., which account about two-thirds of the total death under age 5 (World Health Organization 2010).

We formulate a vitality-based representation of the ontogenescent mechanisms. Whereas, adult vitality declines through degradation of cellular function, juvenile vitality

increases as a result of ontogenesis. Assume initial juvenile vitality is zero and increases linearly as $v' = \rho' x$. As in adults, characterize juvenile challenges with a Poisson distribution of random frequency γ and an exponentially distributed magnitude scaled by β'. Using the arguments of eq. (5) through (7) the conditional mortality rate becomes $m_e = \gamma e^{-v'/\beta'}$ and the ontogenescent mortality rate from extrinsic juvenile challenges is

$$\mu_c(x) = \gamma e^{-x/\alpha} \tag{8}$$

where γ is a base juvenile mortality rate and $\alpha = \beta'/\rho'$ scales the juvenile stage duration.

Note that eq.(8) is equivalent to childhood mortality in the Siler (1979) equation, although cast in the vitality framework it embodies properties of ontogenesence proposed by Levitis and Martínez (2013) in which processes controlling juvenile mortality are distinct from those controlling adult mortality, i.e. the frequency and magnitude of juvenile challenges are independent of adult challenges. In actuality, both juveniles and adults would be susceptible to a common subset of environmental challenges such as infectious diseases. However, the juvenile and adult challenges have distinct patters. In this formulation juvenile vitality increases without limits which limits the duration of ontogenescent mortality, since the vitality eventually exceeds the magnitude of juvenile challenges. The half-life of ontogenescent mortality is $T_o = -\log(0.5)\beta'/\rho' = 0.69\alpha$. In human populations we can view ρ' as an intrinsic factor characterizing the rate of ontogenesis associated with prenatal and some postnatal factors and β' as an extrinsic factor characterizing postnatal conditions. The half-life of ontogenescent mortality thus depends on both intrinsic and extrinsic factors.

Complete 6-parameter model

The final model describes the rate of mortality in terms of the three independent mortality processes in eq. (1) which are defined by eqs. (3), (7) and (8) giving

$$\mu(x) = \frac{x^{-3/2} e^{(1-rx)^2/2s^2 x}}{s\sqrt{2\pi}\left(\Phi\left(\frac{1-rx}{s\sqrt{x}}\right) - e^{2r/s^2}\Phi\left(-\frac{1+rx}{s\sqrt{x}}\right)\right)} + \lambda e^{-(1-rx)/\beta} + \gamma e^{-x/\alpha} \tag{9}$$

Eq. (9) describes the mortality rate from birth onward in terms of six parameters: *r* and *s*, characterize the intrinsic mortality, λ and β characterize the adult extrinsic mortality and γ and α characterize age-dependent ontogenescent mortality in juveniles.

Parameter estimates

The 6 parameters are estimated by fitting eq. (9) to mortality curves with a maximum-likelihood fitting routine (Salinger et al. 2003). The fitting package for a family of vitality models in R code is available at http://CRAN.R-project.org/package=vitality. The eq. (9) version is vitality.6ps.

Biases resulting from the approximation of eq. (7) in place of eq. (6) were evaluated in (Li and Anderson 2013) with simulated vitality trajectories generated according to a numerical representation of eq.(2) and challenges generated according to eq. (4). Regressions of estimated vs. simulated parameters gave the biases corrections

$$r \approx \frac{\hat{r}}{0.94+0.094\hat{\beta}} \quad s \approx \frac{\hat{s}}{1.39-1.03\hat{\beta}} \quad \lambda \approx \frac{\hat{\lambda}}{1.57-1.55\hat{\beta}} \quad \beta \approx \frac{\hat{\beta}}{1.02} \qquad (10)$$

Because juvenile stage extrinsic mortality is insignificant in the adult stage, bias corrections were not needed for α and γ.

Note that because uncertainties are introduced by the corrections of eq.(10), variance for adjusted parameters is larger than that directly obtained from the maximum likelihood algorithm (Fig. 3). In summary, the model slightly underestimates r and overestimates s and λ, but a correction can be applied to those parameters according to the strong relationships between the true and estimated parameters.

Example

The model is illustrated with fits to Swedish period mortality data (HMD) (Figs. 2 and 3). Figure 2 illustrates the age-dependent patterns of the three morality processes for Swedish females for the years 1820 and 2000. Juvenile mortality (μ_o) dominates early ages in both years. Its intensity rapidly declines and adult extrinsic mortality (μ_e), increasing log-linearly, dominates the middle portion of the curves. At old age, the intrinsic process (μ_i) dominates and the curves approaches a plateau due to the Wiener-process nature of intrinsic mortality (Weitz and Fraser 2001, Li and Anderson 2009). Note the transition from the dominance of extrinsic to intrinsic mortality (Fig. 2) occurs about the age of 60 in 1820 but is shifted to 80 by 2000 (Li and Anderson 2013). This transition is important to characterizing the complex patterns of mortality between late-middle and early-old age (Li et al. 2013). The initial juvenile mortality rate expressed through γ, was 26 times larger in 1820 than in 2000. Furthermore, the half-life of the juvenile mortality decreased from 10 to 3 months. The model captures the general

patterns of lifetime survival in both centuries. However, the model does not capture the well-established "accident hump" evident about age 20 for the year 2000 (Heligman 1980). We discuss model extensions for the accident hump and other processes below.

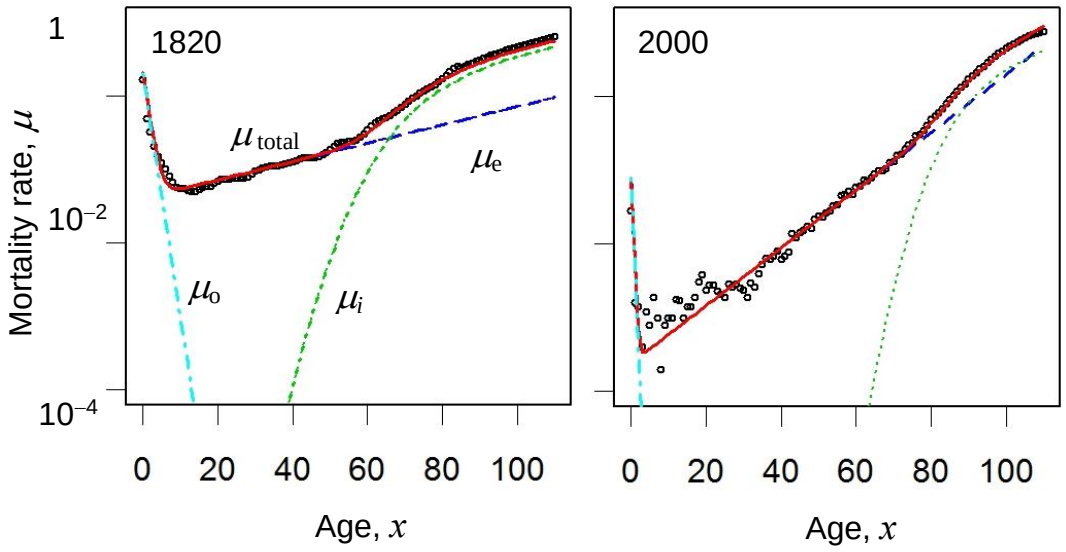

Fig. 2. Period mortality rate data (dots) of Swedish females for 1820 and 2000. Modeled total mortality rate is depicted by red lines. Green, dark blue and light blue lines depict mortality components μ_i, μ_e, and μ_o respectively. Model parameters are r = 0.0129, s = 0.0126, λ = 0.045, β = 0.400, γ = 0.2033, α = 1.35 for year 1820 and r = 0.0107, s = 0.0077, λ = 0.123, β = 0.115, γ = 0.0076, α = 0.39 for year 2000.

The historical patterns of the r, s λ and β, discussed in detail in (Li and Anderson 2013) clearly correspond to the patterns of improvements in human longevity over the past two centuries (Riley 2001) (Fig. 3). The analysis did not contain ontogenescent mortality and therefore the model was fit to period data left truncated at age 20. The historical patterns of parameters for models fit with or without juvenile extrinsic mortality are equivalent, except that the r and s parameters are smaller when fit to data from age zero onward. This difference is a result of the vitality parameters being scaled by the initial vitality which is lower for individuals of age 20 than age 0. Left truncation does not affect the estimates of the other parameters.

Changes in survivorship patterns over the two centuries reflect asynchronous across-year patterns in the intrinsic and extrinsic mortality processes. The vitality rate, which characterizes the intrinsic lifespan as approximately $1/r$, declines in gradual linear manner up to about 1950 (Fig. 3A); putatively because of improved nutrition and shelter associated with agrarian and industrial revolutions that are implicated in the reduced

mortality rates (Riley 2001, Sundin and Willner 2007). World War II marks further steepening in the slope of *r* and by inference an increase in the rate of improvement in living conditions. The 20th century decline in *s* quantifies a steady decrease in intrinsic heterogeneity within the female population. However, in males, *s* increases between 1950 and 1980, suggesting a rapid increase in heterogeneity (Fig. 3B) that coincides with class-stratified differences in smoking rates in adult males over this 30 year span (Diderichsen and Hallqvist 1997). Thus, we hypothesize that *r* quantifies the mean rate of ageing of individuals and *s* quantifies variability of ageing within a population. Together, they characterize how changes in heterogeneity of living conditions shaped the pattern of intrinsic lifespan over two centuries.

The pattern in extrinsic challenge intensity, β (Fig. 3C) and frequency λ (Fig. 3C) shifts from high-magnitude/low-frequency challenges to low-magnitude/high-frequency challenges about mid-20th century. This shift corresponds with the control of infectious diseases (Riley 2001, Omran 2005). Distinct spikes in challenge magnitude in 1808 and 1918 correspond with disease during the Finnish war (Mielke and Pitkänen 1989) and the influenza pandemic (Sundin and Willner 2007) respectively.

The ontogenetic parameters, α and γ, have patterns distinct from the other parameters. The index of ontogenescent mortality duration α, with units of years, exhibits a peak in second half of the 19th century and then asymptotically declines over the 20th century (Fig. 3E). The term represents the ratio β'/ρ', and so the pattern is controlled by multiple processes. Interestingly, the asymptotic decline beginning at the end of the 19th century could involve a linear increase in ρ'. This could suggest a linear increase in the rate of ontogenesis development resulting from improved postnatal care associated with improved living conditions, e.g. better housing nutrition, hygiene and education (Köhler 1991). The historical pattern of the initial juvenile mortality rate γ (Fig. 3F) exhibits a steady decline over two centuries with slope changes about 1930 and 1950. It also strongly correlates with the initial adult extrinsic mortality rate, defined $\lambda e^{-1/\beta}$, e.g. over the two-century dataset of Swedish females $\gamma = -0.0001 + 0.017\lambda e^{-1/\beta}$ with R-sqr = 0.82. We suggest this correlation between zero-age juvenile and adult extrinsic mortality rates provides insights into the contributions of environmental and prenatal factors in the historical decline of childhood mortality. This proposition is explored further in (Sharrow and Anderson in review).

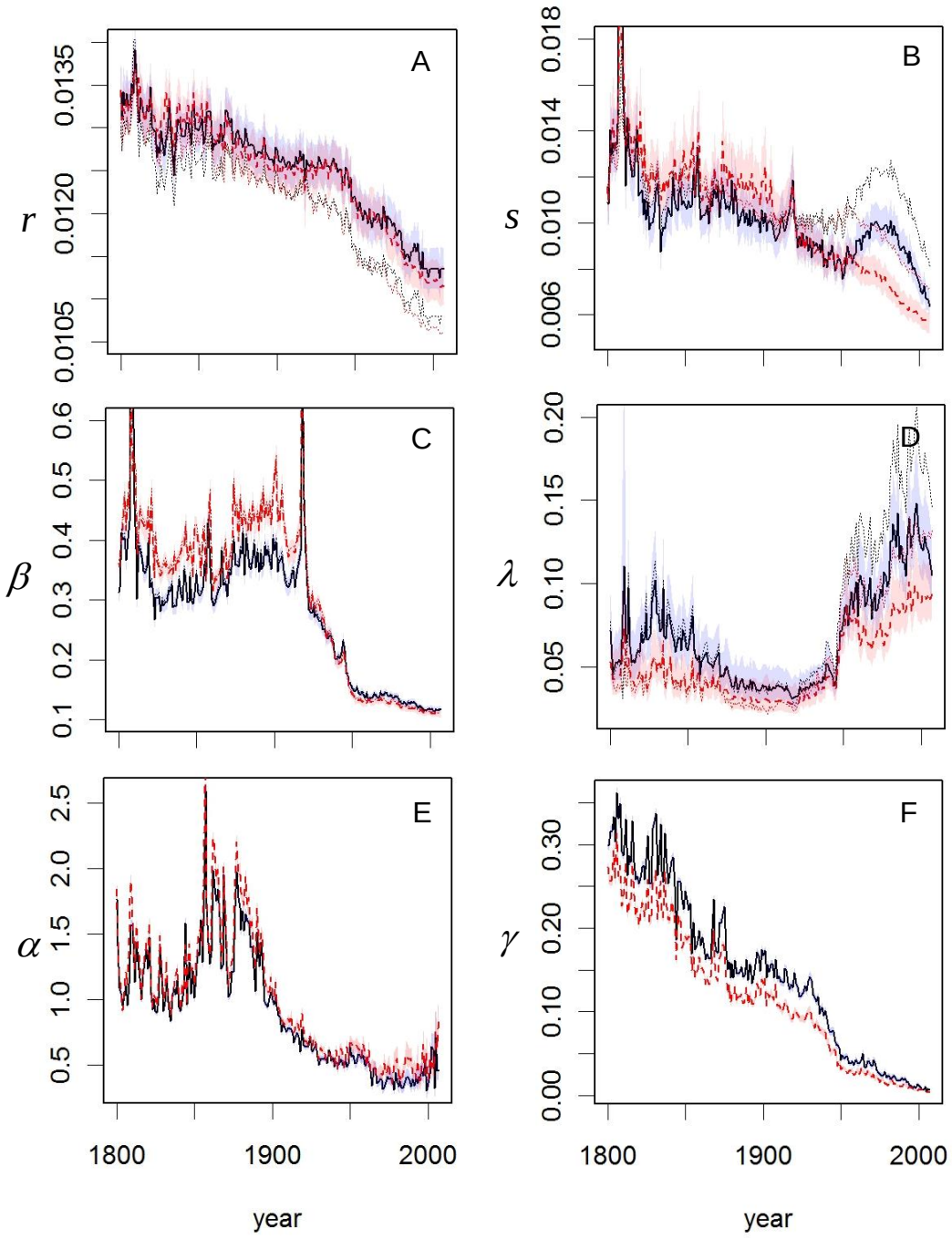

Fig. 3. Longitudinal patterns of bias-corrected vitality parameters for the Swedish population (1800-2007). Male and female patterns are depicted as solid (black) and dashed (red) lines separately. Shaded areas indicate approximated variation for the corrected parameters derived from Eq. 10. (A) vitality loss (yr^{-1}), (B) variation of vitality loss rate ($yr^{-1/2}$), (C) average adult challenge intensity, (D) average adult challenge frequency (yr^{-1}), (E) half-life of juvenile mortality (yr), (F) initial juvenile mortality rate (yr^{-1}). Dotted lines in A-D denote uncorrected parameters.

Discussion

The vitality framework partitions mortality into intrinsic and extrinsic parts representing age-dependent loss of survival capacity and environmental challenges to the capacity. The framework merges Gompertz-like force of mortality models and stochastic vitality models of mortality. The analytical solution requires an assumption of independence between intrinsic and extrinsic processes however, this can be relaxed using simulation forms of the equation (Li and Anderson 2013).

The framework contains three temporal scales which we believe are valuable for addressing problems in population ecology and evolution of longevity. Short-term processes are characterized by extrinsic challenges set by λ, β for adults and γ, α for juveniles. While these parameters are constant in the current model, they can represent within-lifespan changes in extrinsic processes. For example, for human period data the early adult mortality increase, or "accident hump" (Heligman 1980), can be represented by an age-dependent Poisson challenge frequency $\lambda(x)$ in eq. (7). For population ecology, the challenge frequency could be expressed as a function of predator density to characterize the effects of predator regulation on age-specified prey population dynamics. Currently, population models typically ignore age-dependent effects, as in classic Lotka-Volterra predator-prey models, or incorporate age-specific effects through stage-specific Leslie matrix models that do not differentiate heterogeneity within the stages.

At an intermediate temporal scale *r*, which is ρ normalized by initial vitality v_0, characterizes the average lifetime vitality loss rate on a year-specific basis when fitting period data (Fig. 3). The pattern in *r*, across years provide a population level measure of changes in senescence as well as quantifying demographic phenomena such as epidemiological transitions (Li and Anderson 2013, Sharrow and Anderson in review) and across-year changes in survival patterns of late-middle and early-old age survivorship (Li et al. 2013).

The initial vitality v_0 is unique and can characterize the effect of initial conditions on survivorship curves in two different ways depending on whether $x = 0$ represents birth or the beginning of a life stage. For the latter case, Gosselin and Anderson (2013) demonstrated in fish that the mean and variance of v_0, manipulated by food level and competition in a growth stage, affected survivorship in a subsequent starvation stage. Importantly, competition under resource limitation increased the number of fish surviving

starvation; effectively competition culled weaker fish allowing more resources for the survivors. Variations in the mean and variance of v_0 when data begins at birth can produce a similar dynamic. However, in this case v_0 is determined by genetic inheritance. Thus, individuals inheriting larger values of v_0 from parents would experience diminished lethality of environmental challenges resulting in their higher survival than individuals inheriting smaller v_0 values. Thus, heritability of v_0 can potentially characterize some elements of natural selection. Further work on this issue is forthcoming.

In summary, an intrinsic/extrinsic vitality framework naturally merges what were seemingly distinct force of mortality and process point of view approaches to describing mortality. The merged framework shifts the perspective from explaining how the force of mortality changes with age and time to how the underlying intrinsic and extrinsic vitality processes change. We suggest this perspective has value in exploring how environmental and evolutionary processes can shape future survivorship patterns.

Acknowledgement: Funded under National Institute of Ageing grant 1R21AG046760-01

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