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# The Strehler–Mildvan correlation from the perspective of a two-process vitality model

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*The Strehler and Mildvan (SM) general theory of ageing and mortality provides a mechanism-based explanation of Gompertz's law and predicts a log-linear relationship between the two Gompertz coefficients, known as the SM correlation. While the SM correlation is supported by data from developed countries before the second half of the twentieth century, the recent breakdown of the correlation pattern in these countries has prompted demographers to conclude that SM theory needs to be reassessed. In this paper we use a newly developed two-process vitality model to explain the SM correlation and its breakdown in terms of asynchronous trends in acute (extrinsic) and chronic (intrinsic) mortality factors. We propose that the mortality change in the first half of the twentieth century is largely determined by the elimination of immediate hazards to death, whereas the mortality change in the second half is primarily driven by the slowdown of the deterioration rate of intrinsic survival capacity.*

**Keywords**: SM correlation; Gompertz coefficients; mortality pattern; vitality; intrinsic and extrinsic mortality

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## Introduction

Strehler and Mildvan's (SM) general theory of ageing and mortality proposed a biological explanation of Gompertz's well-established law ($\mu(x) = a\,e^{bx}$, where $\mu(x)$ is the mortality rate at age $x$) (Gompertz 1825; Strehler and Mildvan 1960). The theory employed the analogy of chemical kinetics (Golubev 2009) to explain the exponentially increasing shape of the Gompertz curve which results from the interaction between the internal energy reserves of the organism and the external energy demands from environmental insults. An essential consequence of this theory is the SM correlation whereby the Gompertz coefficients are negatively correlated, that is, $\log a$ is a linear function of $b$. This regularity implies that in spite of continuous improvements in human longevity, the evolution of mortality curves should follow certain patterns (Yashin et al. 2001a). A number of studies (Riggs 1990; Riggs and Millecchia 1992; Prieto et al. 1996) have used data from the early decades of the twentieth century to confirm empirically the stable, negative relationship between the Gompertz parameters. However, a break in this relationship has been noted when recent data from industrialized countries such as Sweden, Japan, and France, were analysed (Yashin et al. 2001a, 2001b). These results have challenged the concepts of ageing and mortality that stem from SM theory, as well as the interpretation of the coefficients of models, such as Gompertz's, that are based on the rate of mortality.

In this paper, we demonstrate that the correlation between $\log a$ and $b$ can be readily explained by a two-process vitality framework developed by Li and Anderson (2013). In this new structure, the observed pattern of correlation of Gompertz coefficients is the consequence of the asynchronous trends over time in an intrinsic ageing process, and an extrinsic challenge process. Moreover, we show that changing age-specific adult mortality patterns over time are more complex than those characterized by a two-parameter Gompertz model. Instead, a two-process vitality model, based on four parameters, is the minimum needed to capture the observed changes. This new framework provides a flexible structure for describing the relationship between the physiological and demographic patterns of ageing and can be widely applied to topics such as longevity projection and sex/race differences in mortality.

## SM theory and the SM correlation

We begin with a brief introduction of SM theory that is derived from hidden processes described below. Organisms are assumed to have an initial survival capacity, termed $V$, which declines linearly with age $x$. Therefore, $V(x) = V_0(1 - Bx)$, where $B$ indicates the fraction of vitality loss per unit time and $V_0$ is the initial survival capacity. Over their life courses, animals experience random external challenges or insults with a mean frequency $K$. Challenges have random magnitudes, exponentially distributed with an average $D$, that express the deleteriousness of the environment. Death occurs when the magnitude of a challenge exceeds the remaining vitality. These assumptions produce the Gompertz law of exponentially increasing mortality with age. A detailed review of SM theory can be found in Finkelstein (2012).

The SM correlation derived from the theory describes a negative log-linear relationship between the two Gompertz coefficients. Therefore,

$$\log a = \log K - b/B \tag{1}$$

which, when substituted into Gompertz's law, expresses mortality in terms of the mean frequency of the random extrinsic challenges and the rate of loss of vitality. Therefore,

$$\begin{aligned} \log \mu(x) &= \log K + b(x - 1/B) \\ &= \log K + B/D(x - 1/B) \end{aligned} \tag{2}$$

where $a = K\exp(-1/D)$ and $b = B/D$, with $B$ and $D$ being normalized by $V_0$.

Equation (2) imposes strong regularity on mortality, such that given the assumptions of SM theory, no matter how human longevity changes over time, the shape of the mortality curves conform to a fixed relationship. Specifically, when K and B are constant, that is, both the challenge frequency and the fraction of vitality loss per unit time are stable, all mortality curves in log scale must intersect at a single point (1/B, log K) (Riggs and Millecchia 1992; Yashin et al. 2001a, 2002). With this restriction, as log a declines and b increases, the survival function defined as

$$l(x) = \exp\left(-\int_0^x \mu(t)\,\mathrm{dt}\right) \tag{3}$$

becomes increasingly rectangular over time, a development known as the rectangularization of the survival curve (Wilmoth and Horiuchi 1999; Yashin et al. 2001b).

SM theory has been applied to the analysis of population mortality data by obtaining both the vitality loss fraction $B$, which is considered to reflect genetic influences on survival, and the environmental parameters, $K$ and $D = B/b$, which are considered to reflect extrinsic conditions (Riggs 1990; Riggs and Millecchia 1992; Prieto et al. 1996; Zheng et al. 2011). However, because equation (2) is a linear function with two degrees of freedom but is defined by three parameters ($K$, $b$, and $B$) or ($K$, $D$, and $B$), it is impossible to specify all the parameters for a single mortality curve. In order to estimate these parameters, Strehler and Mildvan (1960) developed several methods that postulated different restrictions on equation (2) (e.g., the $K$ parameter is set to be 1) or any series of mortality curves (e.g., all the mortality curves share a common parameter $B$). The resulting estimates vary according to the method and are highly dependent on the specific assumptions.

However, all the estimation methods, as well as SM theory itself, fundamentally rely on the validity of the SM correlation pattern implied by equation (2). Although a series of experimental studies (Riggs 1990; Riggs and Millecchia 1992; Prieto et al. 1996) suggest a stable negative relationship between the Gompertz coefficients in the first half of the twentieth century, the pattern tends to break after 1950 for many industrialized countries (Wilmoth and Horiuchi 1999; Yashin et al. 2001a, 2001b; Golubev 2004). The first sign of deviation from the SM correlation pattern was demonstrated by Myers and Manton (1984), who found the tail of the survival curve of the US population tended to increase with years in the second half of the twentieth century. In effect, mortality trajectories in recent years do not cross the supposedly single intersection point ($1/B$, $\log K$), but tend to become parallel at old ages. Similar findings have been confirmed by a range of studies (Gavrilov and Gavrilova 1991; Manton and Tolley 1991; Kannisto 1994; Jeune and Vaupel 1995; Horiuchi and Wilmoth 1997, 1998; Wilmoth and Horiuchi 1999; Wilmoth et al. 2000; Robine 2001). In spite of using different terminologies and emphasizing different demographic phenomena, these studies all confirmed the tendency of derectangularization of survival curves and a corresponding breakdown of the SM correlation pattern. These results are summarized in Yashin et al. (2001a, 2001b) and the instability of the SM correlation is demonstrated in a plot of Gompertz $\log a$ vs. $b$ by Yashin et al. (2001a) in their Figures 3–6.

Following Yashin et al.'s (2001a) analysis, Figure 1 illustrates the estimated Gompertz $\log a$ vs. $b$ for adult mortality (from ages 40 to 80) in France,

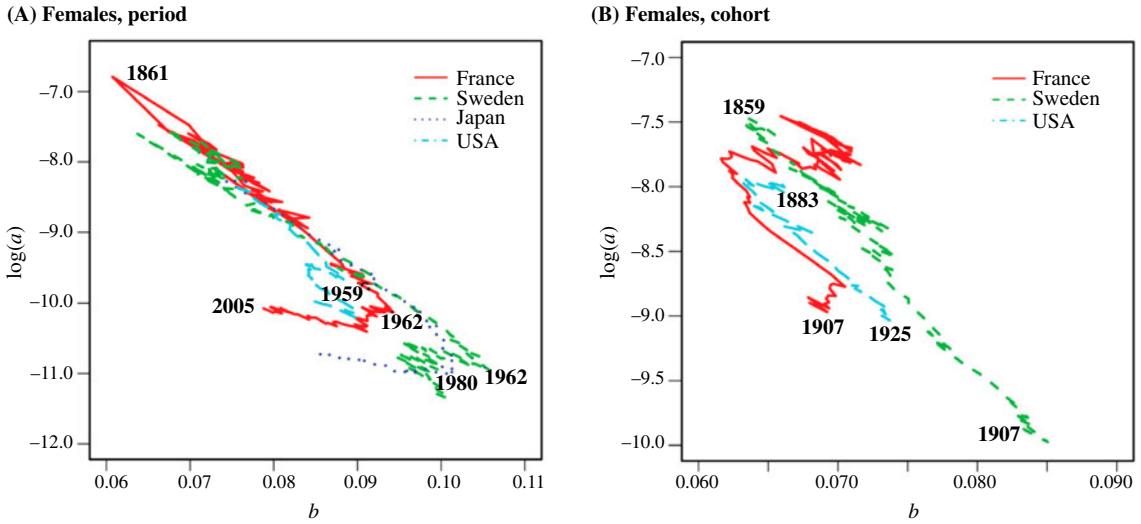


**Figure 1** Patterns of SM correlation: (A) females, period, France (1861–2005), Sweden (1861–2005), Japan (1950–2000), and USA (1938–2005); (B) females, cohort, France (1859–1917), Sweden (1821–1915), and USA (1883–1927)

*Note*: For consistency with other studies (Figures 3 and 5 in Yashin et al. 2001a) mortality for ages between 40 and 80 were used.

*Source*: Human Mortality Database (2011).

Japan, Sweden, and USA. Note that a similar pattern can also be found in the male populations as shown in Yashin et al. (2001a). In this paper, we only use the female populations to demonstrate. Until the second half of the twentieth century, period data have, as predicted by SM theory, a negative linear relationship with the curves moving over time from top-left to bottom-right of the graph. Thereafter, 'hooks' emerge and the curves flatten for France, Sweden, and USA around the 1960s and for Japan around 1980, thereby confirming that the SM correlation breaks down. More complex patterns are revealed by the cohort data, as the slope changes sign multiple times. The complex patterns in cohort data may involve other mechanisms. However, the important point here is that the SM theory assumptions break down for the years where the curves change slope and flatten.

While SM theory establishes an attractive connection between the intrinsic (organism-specific) and extrinsic (environmental) forces that shape mortality patterns, its deterministic structure is insufficient to characterize the interaction between the internal physiological ageing processes of individuals and environmental challenges. More importantly, the effect of the intrinsic vitality decline is revealed only indirectly through its interaction with extrinsic challenges, which makes it difficult to disentangle the two processes. Specifically, in the SM framework, the three process parameters $B$, $K$, and $D$ are not independent of each other and rely on additional constraints to specify the relationship. Therefore, assuming that the basic intrinsic–extrinsic division is reasonable, the irregular patterns of the SM correlation in recent decades are likely to be caused by changes in the relationships of the three parameters over time. The essential point here is that the simple relationship implied by the SM model is violated, and this is what compelled Yashin et al. (2001a, 2001b) to call for the development of new concepts in mortality theory and the relationship between the physiological and demographic patterns of ageing.

## The compensation law of mortality and the Gompertz–Makeham model

Another way of interpreting the SM correlation is known as the compensation law of mortality (Gavrilov and Gavrilova 1991), according to which the negative log-linear relationship between the two Gompertz coefficients suggests that a reduction in the level of mortality ($a$) is always compensated by an increase in the relative rate of mortality growth ($b$). That is, a higher survival capacity in younger age groups results in a faster ageing rate at older ages. The compensation law of mortality has the same form as equation (1) and the parameters $1/B$ and $K$ are defined as the species-specific lifespan and the species-specific mortality, respectively (Gavrilov and Gavrilova 1991). Both the compensation law of mortality and the SM correlation

identify a negative relationship between the Gompertz parameters $a$ and $b$, although the former implies the correlation based on the Gompertz–Makeham (GM) model, while the latter does not adjust the so-called age-invariant background mortality (Gavrilov and Gavrilova 1991; Strulik and Vollmer 2013).

The GM model is an extension of the Gompertz model, derived by adding a constant mortality term ($M$) known as background mortality (Makeham 1860). Thus,

$$\mu(x) = M + a\,e^{bx}. \tag{4}$$

For mortality data over a long period of time, the GM model has been shown to improve the Gompertz model fit significantly, especially between ages 30 and 90. Such observations led Gavrilov and Gavrilova (1991) to conclude that ignoring background mortality could generate an artificial dependency between the Gompertz coefficients that coincides with the real correlation pattern. They therefore recommended using Makeham-adjusted Gompertz coefficients to derive the compensate law of mortality or the equivalent SM correlation.

While it is generally assumed that the GM model is always superior to Gompertz, few studies have examined this hypothesis using recent data and only one has briefly identified a consistent deviation observed from the fit of a GM-type model (Bongaarts

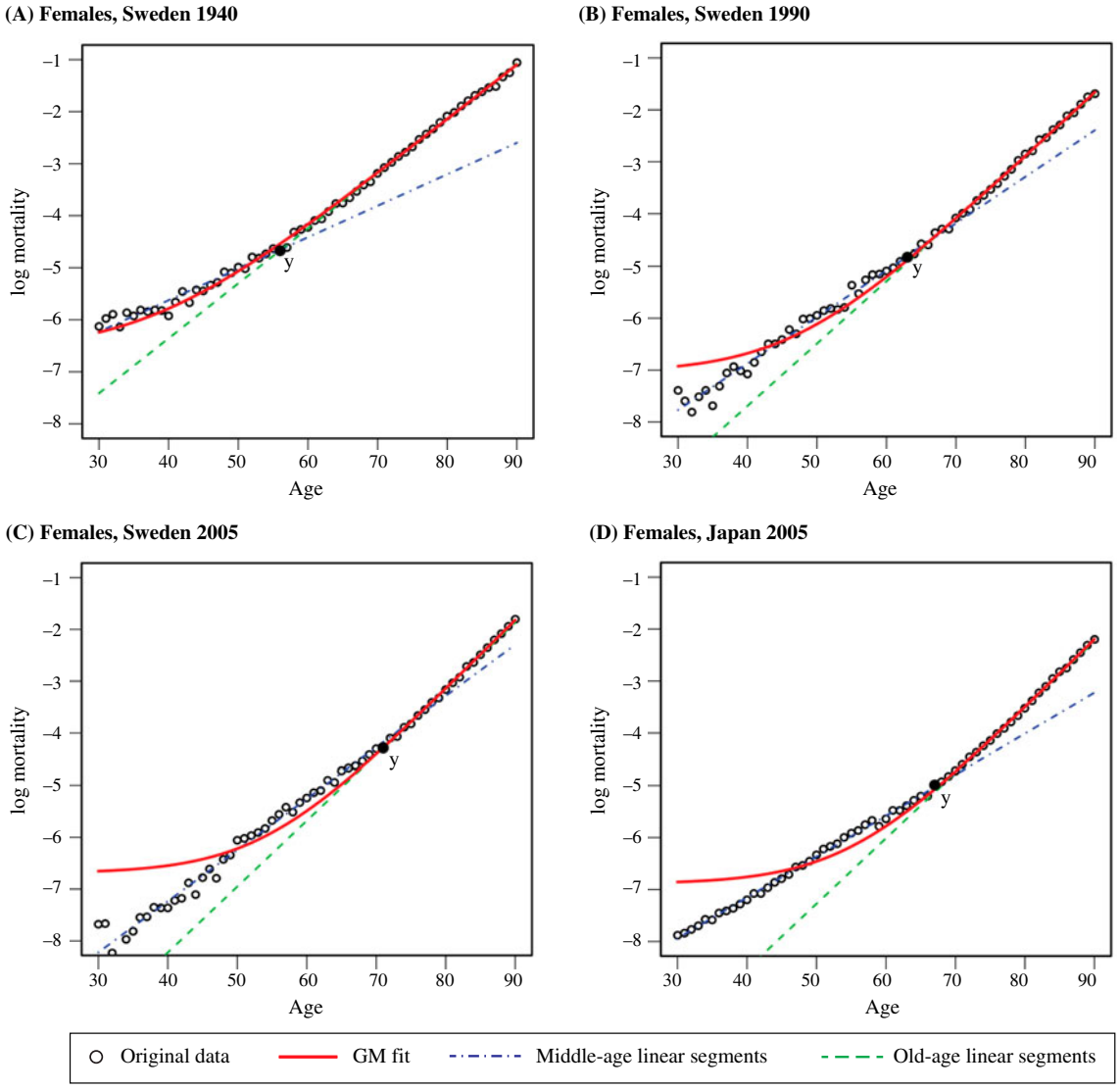


**Figure 2** Gompertz–Makeham model fitted to selected period mortality data: (A) females, Sweden, 1940; (B) females, Sweden, 1990; (C) females, Sweden, 2005; (D) females, Japan, 2005

*Note*: The data (○) are fitted to the GM model (---) using conventional weighted least squares methods. A piecewise linear model that transitions between middle-age (—) and old-age (---) linear segments at age $y$ is demonstrated in (A) and (C).

*Source*: As for Figure 1.

2005). Figure 2 illustrates the fit of the GM model to early and late period mortality data (ages 30–90) based on the conventional weighted least squares method with exposure determined by the weights (using the 'nls' function in R). Results using other algorithms such as the built-in function 'fitGM' in the R package 'fmsb' were similar. As expected, the GM model provides a good fit to the period mortality data before 1950. Nevertheless, discrepancies between the model fit and the data emerge after1950; see especially Figures 2C and D. Specifically, the GM model tends to overestimate mortality for ages between 30 and 50 and underestimate it for ages between 50 and 70. Such deviations become more significant over time and, as Figure 2 shows, are consistently observed in developed countries such as Sweden and Japan. Note that the Swedish female populations were taken as examples of the GM model fit over period years and the Japanese female population (2005) was added to demonstrate that the lack of fit from the GM model in more recent data can be widely observed in other countries.

The GM deviations can be explained as a mismatch of the nonlinear properties of the GM model and the piecewise linear nature of log-mortality curves. As illustrated in Figures 2A and C, log-mortality curves, to a first approximation, can be described by two straight-line segments, one fitting the middle-age portion of the data, the other fitting the old-age portion (Milne 2007). The intersection point where the two lines meet ($y$ in Figure 2) shifts from 58 in 1940 to 70 in 2005. Fitting log-mortality data, the GM model tends to fit the old-age linear piece. However, because the model is highly curved at younger ages, fitting the older-age segments will underestimate mortality between 50 and 70 and overestimate it below 50. In effect, in order to fit the mortality pattern in old age, the GM model has to raise the value of the background mortality, which results in a poor fit to early-age mortality. For further discussion of the mortality increase in early old age, see Li et al. (2013) who, using a two-process vitality model, analyse the transition as a shift from extrinsic to intrinsic mortality processes.

While the background mortality ($M$) in the GM model might be expected to decline over the long term owing to improvements in public health and increases in longevity, Figure 3 illustrates that this is not the case. A dramatic decline of $M$ during the first half of the twentieth century corresponds to the overall mortality reduction across all ages. However, a surprising increase in $M$ from about

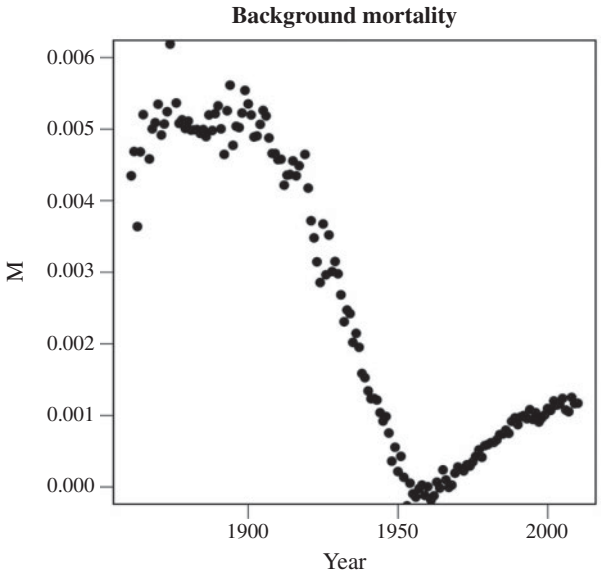


**Figure 3** Gompertz–Makeham background mortality ($M$) derived by weighted least squares of fitting period data for Swedish females aged 30–90
*Source*: As for Figure 1.

1955 is unlikely to reflect a real increase in background mortality. Instead, we suggest it indicates that the underlying mortality dynamics have changed such that the GM deviations from the actual pattern of mortality have been increasing. It is possible to employ another algorithm to force the GM model to fit the younger-age mortality and suppress the increase in $M$, but this results in bias in fitting older-age mortality (Bongaarts 2005). In either case, the GM model cannot generate unbiased estimates of mortality using recent data.

Because the GM model fails to characterize the mortality trajectory in recent decades, and in particular fails to capture the critical change in mortality shape between ages 40 and 80, we suggest that exploring the breakdown of the SM correlation based on the GM model adds additional complexity to interpreting the changing dynamics of mortality. To ensure a consistent and viable assessment of the SM correlation over time, we therefore employed the Gompertz model and restricted the data analyses to ages between 40 and 80, the interval in which the impact of background mortality is minimized and the slope of the log-mortality rate vs. age graph is approximately constant.

We also note that the failure of the GM model in fitting mortality data and the breakdown of the SM correlation occur during the second half of the twentieth century, with both phenomena reflecting a common and fundamental change in underlying mortality dynamics. We now demonstrate this by

treating the breakdown of the SM pattern as a shift between intrinsic and extrinsic mortality processes characterized by a vitality model.

## The two-process vitality model

Yashin et al. (2001a) concluded that changes in the SM correlation pattern over time indicate that there must have been changes in the frequency of extrinsic challenges, $K$, and changes in the rate of loss of vitality, $B$. While agreeing with this conclusion, we propose that the underlying mechanisms involve additional dimensions that the Gompertz and GM models cannot reveal, and that in order to understand the historical SM pattern we require a model that explicitly identifies the minimum number of dimensions needed to characterize mortality. We further propose that in its most reduced form, mortality involves intrinsic and extrinsic processes and that a minimum characterization of each process requires one dimension quantifying magnitude and a further dimension characterizing frequency. On this view, any mortality model requires a minimum of four dimensions, two characterizing extrinsic and intrinsic magnitudes and two their frequencies. In this paper, we do not specify the mathematical forms of these processes nor do we suggest that four dimensions are sufficient to fully describe the model.

The vitality model developed by Anderson (2000) and expanded by Li and Anderson (2013) into a full two-process framework (also described as a 'two-mortality model' in Li and Anderson 2013, p. 345) meets our criteria for exploring SM correlations and the changes that occurred during the second half of the twentieth century. Specifically, this new framework considers mortality resulting from stochastic depletion of intrinsic survival capacity, that is, vitality, and extrinsic challenges exceeding vitality. We describe the model here and demonstrate that the breakdown of the SM correlation is a natural consequence of independent changes in the intrinsic and extrinsic processes over time.

### *Intrinsic mortality*

In the original SM model, the intrinsic process is modelled as a linearly declining function of the organism's vitality in which death results when an external challenge exceeds the available vitality. In our model, death occurs from extrinsic challenges but also as the end point of the organism's gradual loss of vitality. Anderson et al. (2008) have discussed the biological basis of intrinsic death and Mitnitski and co-workers (Mitnitski et al. 2004, 2005; Rockwood and Mitnitski 2006, 2007) demonstrated that the frailty index, constructed as 'a proportion of all potential deficits (symptoms, signs, laboratory abnormalities, disabilities) expressed in a given individual', had a limit independent of age (0.65 ± 0.05) (Mitnitski et al. 2004, p. 627). In terms of vitality, their findings suggest a minimum value of survival capacity (i.e., the inverse of deficits accumulation) below which survival is unlikely, and this supports our notion of intrinsic mortality when vitality drops below a threshold. From a utility point of view, the inclusion of intrinsic mortality along with the more traditional extrinsic mortality allows for a quantitative estimate of the direct role of senescence in mortality.

We represent the stochastic loss of vitality leading up to intrinsic mortality in terms of a Markovian process (Anderson 2000; Aalen and Gjessing 2001; Weitz and Fraser 2001; Anderson et al. 2008; Li and Anderson 2009). Each individual begins with an initial vitality, $\nu_0$, that declines stochastically with age and results in death when its vitality reaches zero (see A in Figure 4). The random trajectory of vitality, $\nu_x$, between $\nu_0$ and 0 is described by the Wiener process with drift,

$$\nu_x = \nu_{x-1} - r + sW_x \quad x = 1, 2, 3 \ldots \tag{5}$$

where $W_x$ is a standard Wiener process modelled from a unit normal distribution N[0, 1]. When

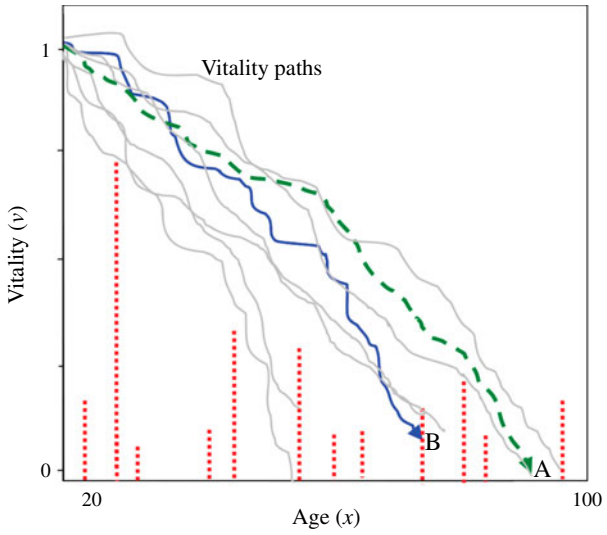


**Figure 4** Illustration of the two-process vitality model
*Note*: Vitality declines stochastically from an initial value of 1. Intrinsic mortality results when adult vitality is exhausted (A) and extrinsic mortality occurs when a random challenge exceeds the remaining vitality (B)
*Source*: Reproduced from Figure 1 of Li and Anderson (2013).

equation (5) is standardized to the initial vitality, then $r$ is the deterministic fraction of vitality lost in a unit increment of time and $s$ is the intensity of the random contribution to the rate of vitality loss. In this case, each normalized vitality trajectory starts from a single point $\nu_0 = 1$ and the differences in the initial values are reflected in the spread term $s$, which specifies the average combined variation in survival capacity from both inherent (initial) and acquired (evolving) sources per unit time. Without the presence of extrinsic mortality, the distribution of the time to death from intrinsic processes is the inverse Gaussian function (Cox and Miller 1965), which describes the first-passage time of vitality in equation (5) to the zero boundary.

### *Extrinsic mortality*

Extrinsic mortality is usually considered as deaths that are 'relatively preventable and treatable', including 'mortality mainly from infections and accidents' (Shryock and Siegel 1975, p. 405). We construct the extrinsic death process following SM theory, in which extrinsic mortality results when a random exterior event challenges the survival capacity of the organism. In SM theory, the survival capacity is deterministic; here we model the stochastic trajectory of vitality via equation (5) so that survival capacity is a stochastic variable. The interaction between the intrinsic ageing and extrinsic challenge processes resulting in death also have biological support. For example, the ageing-related degeneration of the immune system has been widely documented (Goidl et al. 1976; McElhaney et al. 1992). Older adults are more susceptible to fatal infectious diseases and neoplasia due to the senescence-related failure of immune functions (Miller 1996).

To express the extrinsic mortality rate at age $x \geq 0$, let a random point process $Y_x$ with mean frequency of challenges $\lambda$ represent an extrinsic challenge such as a natural disaster or infection. For each challenge, a variable $Z_x$ with a cumulative distribution function $\varphi(z)$ denotes the challenge magnitude. Assuming that death from an extrinsic cause occurs when the challenge magnitude $Z_x$ exceeds the current vitality level $\nu_x$ then death occurs when $\Pr[Z_x > \nu_x]$ (see Figure 4B). Further, assume that $Y_x$ is a history-independent Poisson process (Finkelstein 2007) and challenge magnitudes are exponentially distributed with a scale parameter $\beta$, such that most are small and the probability of large events declines relative to their magnitude as in SM theory. The conditional extrinsic mortality rate is therefore,

$$\begin{aligned} m_e(x|\nu_x) &= \lambda \Pr[Z_x > \nu_x] = \lambda(1 - \varphi(\nu_x)) \\ &= \lambda \, \mathrm{e}^{-\nu_x/\beta} \end{aligned} \tag{6}$$

where $\nu_x$ is a realization of the vitality process at $x$, and $\lambda$ and $\beta$ indicate the challenge frequency and the average challenge magnitude, respectively. The population-level extrinsic mortality rate can then be obtained by integrating equation (6) over vitality states $\nu_x > 0$.

### *Total mortality*

In this new model, mortality has two sources, an extrinsic one expressed by instantaneous challenges to the survival capacity and an intrinsic one expressed as the chronic wearing out of the survival capacity to an absorption boundary. Close-form solutions are possible for intrinsic mortality when every challenge kills or extrinsic death is ignored (Anderson 2000; Weitz and Fraser 2001) and for extrinsic mortality when the intrinsic process is linear (Strehler and Mildvan 1960). However, when the two processes interact stochastically, as assumed above, the age-dependent distribution of vitality in the population has no analytical solution because challenges preferentially eliminate low-vitality individuals thereby modifying the distribution derived from the Wiener process. In this case, the total mortality rate can either be approximated, as was done by Li and Anderson (2013), or calculated numerically using microsimulation. To maintain the interaction of processes and explore the consequences of the system, here we use microsimulation to derive the average macro-level characteristics from large sample simulations at the individual level (Manton et al. 2009).

First, we simulate vitality trajectories in a population of 10,000 individuals using equation (5). For each individual, a Poisson process, with rate parameter $\lambda$, generates a vector of random challenge times where the magnitude of each challenge is drawn from an exponentially distributed random process scaled by parameter $\beta$. Mortality occurs in the age interval $[x - 1, x]$ if the individual's vitality becomes zero or negative through the intrinsic loss process, or if a random challenge occurs with a magnitude exceeding the individual's vitality $\nu_{x\text{-}1}$. In either case, the individual's vitality trajectory is removed and the time to death recorded. Combining the ages of death of all individuals, we construct the

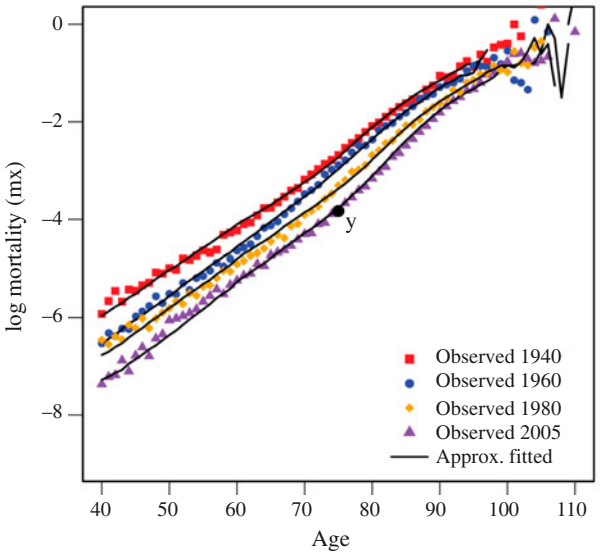


**Figure 5** Approximated fit of the two-process vitality model to selected period mortality curves for Swedish females, aged 40–110

*Note*: The approximated parameter values are: 1940 ($r = 0.0177$, $s = 0.0112$, $\lambda = 0.155$, $\beta = 0.202$); 1960 ($r = 0.0175$, $s = 0.011$, $\lambda = 0.152$, $\beta = 0.171$); 1980 ($r = 0.0164$, $s = 0.0102$, $\lambda = 0.142$, $\beta = 0.172$); and 2005 ($r = 0.0161$, $s = 0.010$, $\lambda = 0.12$, $\beta = 0.157$).

*Source*: As for Figure 1.

population life table by calculating age-specific survival rates, mortality rates, and the expected lifespan (Preston et al. 2001).

The mortality curves generated by microsimulation are determined by four parameters, with $r$ and $s$ characterizing the intrinsic process in terms of the average intrinsic degeneration rate and the variation in the rate, while $\lambda$ and $\beta$ determine the extrinsic challenge frequency and challenge magnitude, respectively. It is worth noting that, in principle, extrinsic challenges that do not result in mortality could incrementally alter the vitality of a survivor. In this model, we ignore this process but assume the effect is subsumed into the Wiener process.

Figure 5 shows that the two-process vitality framework captures the adult mortality patterns of Swedish females (aged 40–110) obtained from the Human Mortality Database (2011) for the years: 1940, 1960, 1980, and 2005. Because the choice of ages influences the correlation of the Gompertz coefficients, we started with age 40 to ensure consistency with earlier studies (Yashin et al. 2001a, 2001b). The lines were generated from microsimulation with prescribed parameter values that matched the trend of the data. The nearly linear increase in the log-mortality rate in middle age results from the interactions between the intrinsic degeneration and extrinsic challenges. The mortality plateaus at very old age are a result of the properties of the Wiener process (Aalen and Gjessing 2001; Weitz and Fraser 2001; Li and Anderson 2009, 2013). The model also captures the acceleration in the log-mortality pattern beginning at old age, at point $y$ of the 2005 mortality curve in Figure 5 (Li et al. 2013). Of particular importance, Figure 5 illustrates that the two-process vitality model readily tracks the observed mortality patterns across the late twentieth century when the SM correlation breaks down.

## Explaining the SM correlation patterns

To explain the SM correlation in terms of the two-process model, note first that the deterministic rates of loss of vitality in the two models are equivalent, so $r = B$. Second, the models formulate the extrinsic challenges in the same way, so the representative magnitudes and mean frequencies are equivalent, that is, $D = \beta$ and $K = \lambda$. Thus, in the notation of the two-process vitality model, the SM correlation defined by equation (1) is simply,

$$\log a = \log \lambda - b/r. \tag{7}$$

That is, the log-linear relationship between $a$ and $b$ is preserved if $\lambda$ and $r$ are constant in the same way that SM theory requires $K$ and $B$ to be constant (Finkelstein 2012). In both the two-process vitality model and SM theory, the mid-twentieth-century breakdown of the log-linear relationship involves the parameters not being constant. However, the explicit linkage of parameters in the SM framework makes it difficult to explain how the parameters need to change to produce the breakdown. By contrast, in the two-process vitality model, $\lambda$ and $r$ are formed by independent processes that result in distinct forms of mortality (Li and Anderson 2013). Thus, evoking two distinct mortality processes allows for more flexibility in identifying the causes of the SM correlation and its breakdown.

To illustrate the SM correlation pattern and its breakdown in terms of the two-process model, we fit the Gompertz model to survival curves simulated with the two-process model. In this manner for each set of $r$, $s$, $\lambda$, and $\beta$, we derive the corresponding log $a$ and $b$ measures. It is then straightforward to identify what combinations of parameters generate and break the log-linear relationship of Gompertz parameters.

Figure 6 illustrates the Gompertz coefficient correlations (log $a$ vs. $b$) estimated from mortality curves

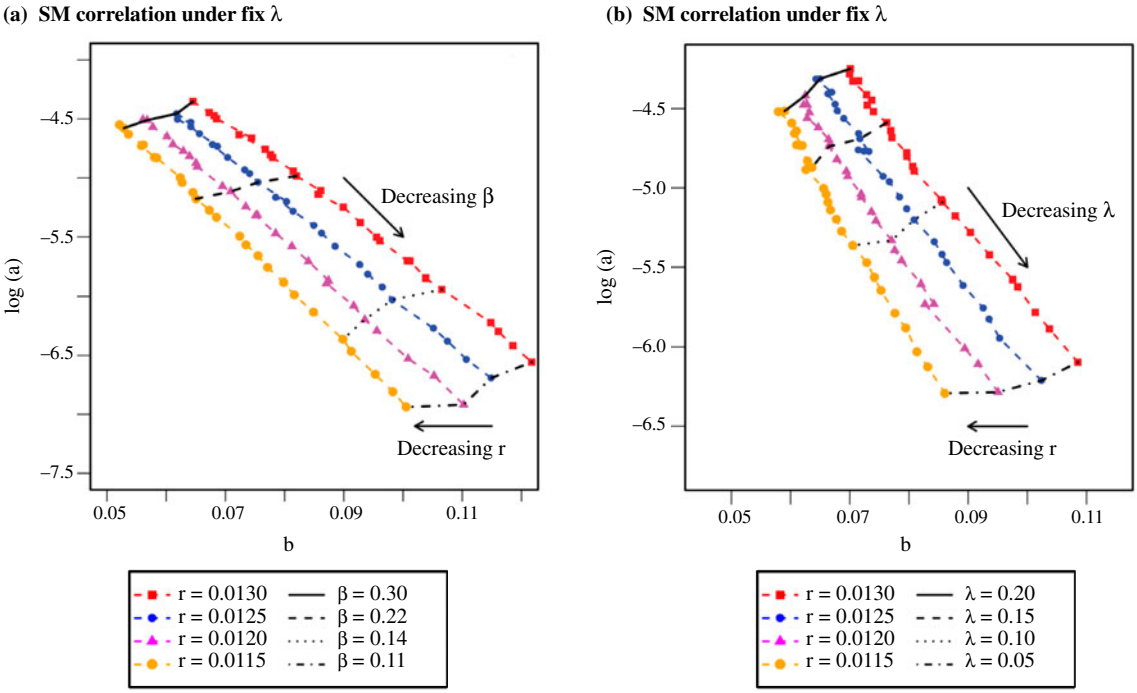


**Figure 6** Simulated SM correlation patterns
*Note*: Mortality curves are simulated under the fixed challenge frequency term $\lambda = 0.12$ in (A) and $\beta = 0.125$ in (B). For both (A) and (B) the curves have the same background variance structure in vitality, $s = 0.01$.

generated from the two-process vitality model. The parameters ($r, s, \lambda$, and $\beta$) used in simulating mortality trajectories are typical for adult mortality period data (ages 40–110). The $\log a$ vs. $b$ patterns are readily generated by fixing $\beta$ but varying $r$ and $\beta$ (Figure 6A), or fixing $\beta$ but varying $r$ and $\lambda$ (Figure 6B). For both patterns, the points forming each line, which move from the upper left to lower right of the graphs, are estimated by survival curves generated with a fixed $r$. The points forming each horizontal line, trending from right to left, are estimated from survival curves generated by fixing $\lambda$ (Figure 6A) or $\beta$ (Figure 6B) and decreasing $r$. Together, the plots illustrate how $\log a$ and $b$ change as $r$, $\lambda$, and $\beta$ change. As $r$ decreases, $b$ decreases but $\log a$ is relatively unchanged, while as either $\lambda$ or $\beta$ decrease, $b$ increases and $\log a$ becomes more negative. In both plots, $s$ is fixed because it has little impact on the patterns.

Now consider SM correlations in developed countries (France, Sweden, Japan, and USA) in terms of variations in $r$, $\lambda$, and $\beta$. A stable negative linear pattern of the Gompertz coefficients from about 1860 to 1960 in period survival data (Figure 1A) reflects changes in the extrinsic parameters $\lambda$ or $\beta$ dominating changes in the intrinsic parameter $r$. The reversal and flattening in the $\log a$ vs. $b$ curve since the mid-twentieth century illustrates that changes in the intrinsic parameter $r$ dominate changes in extrinsic parameters $\lambda$ or $\beta$. Thus, in terms of the two-process vitality model, throughout the first half of the twentieth century, improvements in the environment reduced the average challenge frequency, the magnitude or a combination of both. The break in the linear trend about 1960, and the subsequent bending backwards of the pattern, reflects a gradual shift from the dominance of improvements in environmental conditions to improvements in intrinsic accumulated health.

In the case of cohort data (Figure 1B), the complex pattern suggests multiple changes in the dominance of intrinsic and extrinsic processes over time, but disentangling these changes is difficult and beyond the scope of our paper. For a further discussion of cohort and period effects in the model, see Li and Anderson (2013).

SM theory fails to explain the correlation patterns because it attributes all mortality to one process in which environmental challenges exceed the available vitality. Because it does not express mortality independent of environmental challenges, it does not differentiate between changes in survival resulting from improvements in chronic ageing-related processes and improvements in acute environmental processes. Including two mortality processes breaks these restrictions.

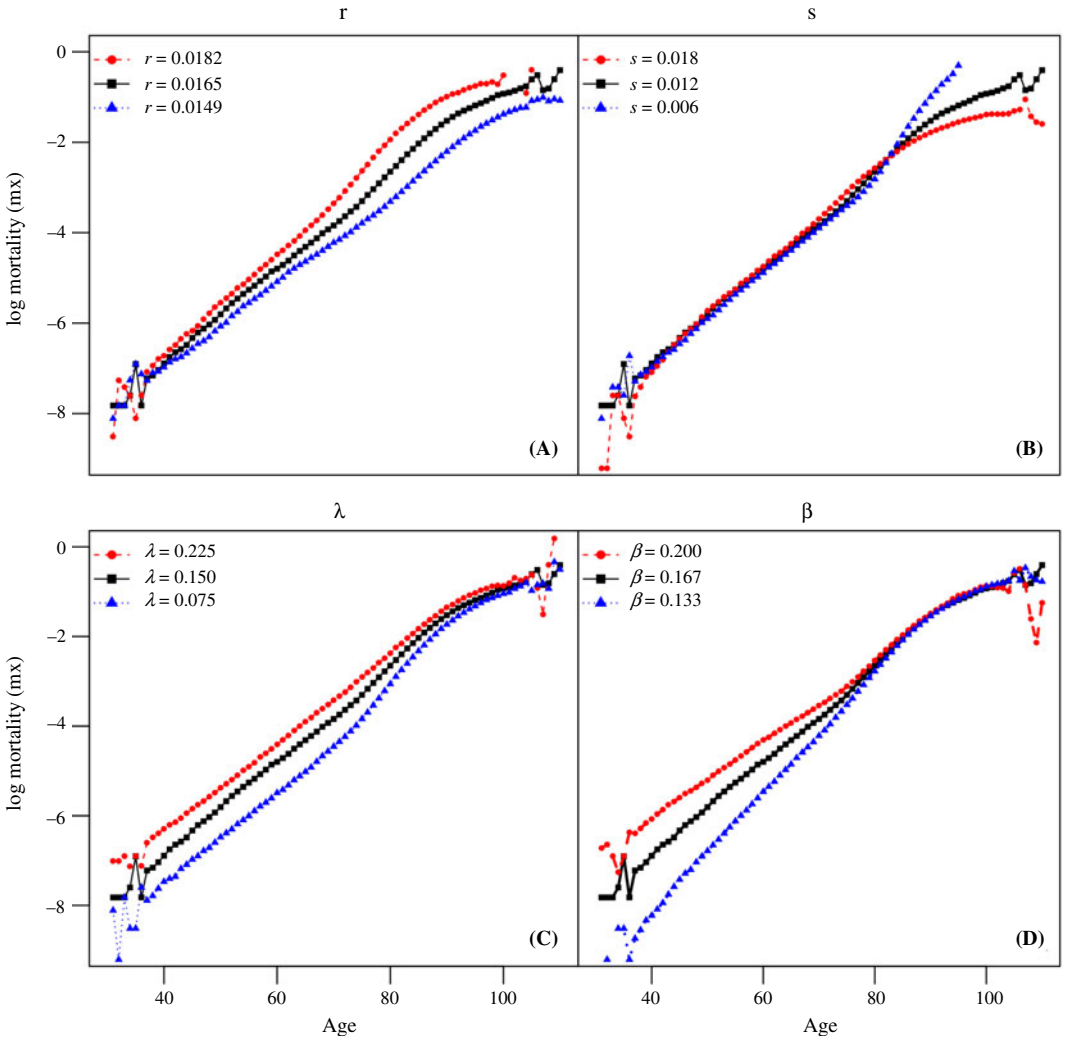


**Figure 7** Simulated patterns of log mortality under varying values of the two-process vitality model parameters

*Note*: (A) $r$ changes while $s$, $\lambda$, and $\beta$ are fixed at 0.012, 0.150, and 0.167, respectively; (B) $s$ changes while $r$, $\lambda$, and $\beta$ are fixed at 0.0165, 0.150, and 0.167, respectively; (C) $\lambda$ changes while $r$, $s$, and $\beta$ are fixed at 0.0165, 0.012, and 0.167, respectively; (D) $\beta$ changes while $r$, $s$, and $\lambda$ are fixed at 0.0165, 0.012, and 0.150, respectively.

## The shape of mortality

To explore further the complex effect of temporal changes in mortality processes on mortality curves we present simulated plots, generated as described in the two-process vitality model section, summarizing how parameters in the two-process vitality model affect the shape of log mortality (Figure 7). Each plot depicts the impact on log mortality of three different values of one model parameter with the other parameters remaining constant.

Figure 7 shows that all four parameters affect the shape of log mortality with age, but in distinctive ways. The intrinsic parameters $r$ and $s$ primarily affect $t$ mortality patterns in old age, in particular by determining the characteristics of mortality plateaus. Smaller values of $r$, which indicate slower rates of intrinsic deterioration, decrease the age of the onset of the plateau; for example, in Figure 7A the slope of log morality begins to decelerate at age ~80. Smaller values of $s$ increase the asymptotic height of the plateau (Figure 7B), but neither intrinsic parameters significantly affect early-age mortality. By contrast, the extrinsic parameters mostly affect early ages where the log-mortality relationship with age is linear. Increases in $\lambda$ tend to raise the intercept of the relationship (Figure 7C) while increases in $\beta$ tend to raise the intercept and lower the slope of the relationship (Figure 7D). At old age, the effects of both extrinsic parameters are small and the mortality curves quickly converge.

The effects of the model parameters on log mortality depicted in Figure 7 helps to further explain the SM patterns revealed in Figure 6. Applying the Gompertz model (linear fit) to the log-mortality curves between ages 40 and 80, the change in $r$ significantly alters the Gompertz slope $b$ but not the intercept $a$. Therefore, a decrease in $r$ decreases $b$, which results in points in the $\log a$ vs. $b$ plot shifting leftwards (Figure 6). For the case of extrinsic parameters, a decline in $\lambda$ (Figure 7C) or $\beta$ (Figure 7D) significantly reduces the intercept $a$ and because of the convergence of mortality trajectories at old age, the slope $b$ correspondingly increases. This trend occurring over a period of time would produce a sequence of points in a $\log a$ vs. $b$ plot trending from the top-left to bottom-right as in Figure 6 and was also observed in the $\log a$ vs. $b$ plot using nineteenth- to mid-twentieth-century data (Figure 1A). Note that parameter $s$, which measures the intrinsic heterogeneity, has little impact on the log-mortality curve before age 80 and thus does not affect the SM correlation pattern.

The change in the empirical patterns of mortality revealed in Figure 5 is similar to the change shown in Figure 7. Comparing the period log-mortality curves of 1940 and 1960, the differences between the two curves diminish with age, which corresponds to the change when $\beta$ is reduced, and the almost parallel shift in the curve until very old age between 1980 and 2005 may indicate reductions in both $r$ and $\lambda$.

## Discussion

Understanding why human mortality patterns have changed over time has been the subject of many studies (Horiuchi and Wilmoth 1997; Wilmoth and Horiuchi 1999; Tuljapurkar et al. 2000; Bongaarts 2005) and is important for assessing the impacts of public health programmes and developing more accurate projections of future patterns of mortality. An important change in the pattern of mortality occurred in the mid-twentieth century when survival curves became derectangularized and shifted to the right. Of particular interest to demographers, this mid-century change broke what was assumed to be a quasi-universal demographic law which fixed the relationship between the Gompertz model coefficients (Yashin et al. 2001a, 2001b; Finkelstein 2012).

The SM general theory of ageing and mortality, as the first important mechanism-based justification of Gompertz's law, is well recognized in the demographic literature. Although generalizations to the framework, such as including a Makeham term to improve the model fit, have been proposed, historically SM theory has provided a good description of mortality patterns, at least between middle and early-old age. Therefore, the theory, as well as the correlation it defined, was considered to be a universal law and widely applied to many studies. However, the evidence exhibited in this and other studies (Yashin et al. 2001a, 2001b) clearly suggests that the SM framework, in its original form, is no longer suitable for analysing contemporary mortality data. Since there appears to be no superior alternative model, modifying the SM framework seems a more feasible option, not only because of the theory's historical influence, but also because it is considered necessary to use a single framework to quantify human mortality change. Such assumptions stem from the fact that the biological processes underlying human mortality and ageing have not changed dramatically over the past 100 years (Yashin et al. 2001b).

Several approaches to revising SM theory have been proposed. Zheng et al. (2011) analysed patterns in 42 countries and suggested retaining general SM theory, but allowing heterogeneity in the coefficients across demographic groups, in particular the rate of decline in vitality ($B$ in SM theory or $r$ in the two-process vitality model) and the amount of deleteriousness ($D$ in SM theory or $\beta$ in the two-process vitality model). Finkelstein (2012) explored deviations from SM theory with models including a gamma-Gompertz frailty model and a vitality-independent model that diminishes the frequency of late-life challenges (Gompertz $a$ parameter) through medical interventions. In our paper we explain the change in SM correlations by drawing on earlier work (Li and Anderson 2013) that builds on SM theory by adding a second mortality process and a fourth dimension. The resulting model captures heterogeneity in the rate of loss of survival capacity and includes intrinsic mortality from physiological wear in addition to the extrinsic or acute mortality of SM theory. This new framework removes the interdependence of the Gompertz parameters and allows a single coherent framework whereby historical patterns of mortality can be understood. It retains the basic structure of SM theory, which is broadly recognized within the literature, while becoming more flexible in adapting to current mortality change. In this way the new framework bridges our understanding of mortality dynamics in the past and our ability to predict future changes.

Within the two-process vitality framework, we explain the changes in period mortality of developed

countries in the first half of the twentieth century as a result of three developments: improvements in environmental conditions, including medical advances that eliminated many infectious diseases; modern technologies that prevented natural disasters; and increasing health knowledge that reduced acute risks. Additionally, the framework attributes further reductions in mortality and the corresponding increase in longevity during the second half of the twentieth century to reductions in chronic health risks. While it is unlikely that intrinsic improvements have a significant genetic contribution, it is possible that they involve physiological responses to advanced living standards, better nutrition, and healthier lifestyles that together reduce the rate of intrinsic vitality loss and delay the onset of organ malfunction and chronic diseases. Intrinsic improvements could also be due to medical interventions and life-saving technologies, such as replacement surgery that, in extending life expectancy, is equivalent to reducing the rate of physiological decay (Finkelstein 2012; Strulik and Vollmer 2013).

We suggest that the two-process vitality framework, with its natural and intuitive characterization of complex temporal trends in mortality across centuries, and in particular over the past half-century, provides a basis for studying other demographic problems (Li and Anderson 2013). First, partitioning of mortality into a slowing, but varying, chronic accumulative process and a rapidly varying acute challenge process can quantitatively characterize genetic, behavioural, and environmental contributions to mortality and longevity over different historical periods. Second, the framework, with appropriate extrapolations of the four parameters, should be ideal for projecting mortality and longevity contingent on a population's physiological status and future patterns of environmental conditions, such as climate, health resources, disease prevalence, and social–economic patterns. Moreover, cross-species and cross-habitat comparisons of patterns of intrinsic and extrinsic mortality should illuminate underlying biological and environmental mechanisms. For instance, varying lifespans within or between some species may be the result of differing rates of predation, not just differences in the rates of ageing. However, successful applications rely on good analytical approximations of the framework. For example, Li and Anderson (2013) addressed the problem of estimating model parameters by imposing constraints on the extrinsic processes.

The proposed framework is also flexible. For example, age-dependent extrinsic challenges can be represented by expressing the challenge frequency parameter $\lambda$ as a non-homogeneous Poisson process (Finkelstein 2008). Furthermore, other intrinsic process forms can be explored in place of the Wiener process decay to a boundary. Vitality dynamics could be expressed as a stochastic equilibrium process such as Ornstein–Uhlenbeck (Borgan et al. 2008), or a stationary random process that underlies the stress–strength model in reliability theory (Finkelstein and Cha 2013). While these and other stochastic forms are possible their usefulness ultimately rests on empirical confirmation and biological realism.

In summary, the two-process vitality framework is a simple approximation to the multi-dimensional processes that determine the moment of death. While it is invariably inadequate when higher levels of specificity of mortality are required, we suggest it is the minimum increment needed to extend the Gompertz framework to meet the challenges in understanding historical and future patterns of mortality.

## Notes

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