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How slowing senescence translates into longer life expectancy

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Mortality decline has historically been largely a result of reductions in the level of mortality at all ages. A number of leading researchers on ageing, however, suggest that the next revolution of longevity increase will be the result of slowing down the rate of ageing. In this paper, we show mathematically how varying the pace of senescence influences life expectancy. We provide a formula that holds for any baseline hazard function. Our result is analogous to Keyfitz's 'entropy' relationship for changing the level of mortality. Interestingly, the influence of the shape of the baseline schedule on the effect of senescence changes is the complement of that found for level changes. We also provide a generalized formulation that mixes level and slope effects. We illustrate the applicability of these models using recent mortality decline in Japan and the problem of period to cohort translation.

Keywords: mortality; lifetable; ageing; senescence; mathematical demography; entropy; semi-parametric survival models

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It is a curious thing that there is no word in the English language that stands for the mere increase of years: that is, for ageing silenced of its overtones of increasing deterioration and decay.... We obviously need a word for mere ageing, and I propose to use ageing itself for just that purpose. Ageing hereafter stands for mere ageing, and has no other innuendo. I shall use the word senescence to mean ageing accompanied by that decline of bodily faculties and sensibilities and energies which ageing colloquially entails. (P. B. Medawar 1957, p. 46)

1. Introduction

Recently, there have been a number of calls for tackling the problem of ageing head-on by reducing the rate at which people get old. In one prominent example, published in the *British Medical Journal*, researchers on ageing argue for medical research to turn away from the disease-specific approach and instead focus on slowing the ageing process: 'The most efficient approach to combating disease and disability is to pursue the means to modify the key risk factor that underlies them all—ageing itself' (Butler et al. 2008). Some observers, such as de Grey and Rae (2007) in *Ending Aging*, are optimistic about the possibility of dramatically slowing the ageing process in the near future.

In this paper, we explore what would happen to life expectancy if senescence were slowed by reducing the pace at which mortality rates rise with age. We establish formally how large the pay-off to such a breakthrough would be. Our aim in this study was to provide new formal relationships between different kinds of stylized mortality decline and in particular to gain insight into why slowing the rate of ageing could provide dramatic improvements in average longevity. Our results complement the classic work of Keyfitz (1977) on the effect of changing the level of mortality on life expectancy, adding slope changes and combined slope-and-level changes to the demographer's formal toolkit.

Among a variety of meanings, senescence has been defined as the increase of mortality risks that accompany the weakening of an organism with age (Medawar 1957; Finch 1990; Partridge 2010). Thus, one way to think about slowing senescence is as a reduction in the speed with which the risk of death increases with age. In the Gompertz case, when hazards are exponential, $\mu(a) = \alpha e^{\beta a}$, slowing senescence is equivalent to reducing β. More generally,

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for any pattern of increasing hazards, a slowdown in senescence can be modelled by letting the hazard at age a be equal to the hazard observed in a baseline schedule at age θa. For example, if $\theta = 1/2$, then an individual with slowed senescence is exposed at age 60 to the hazard observed in the baseline schedule at age 30, at age 80 to the original hazard at age 40, and so on. Although the mathematics of what follows applies to any baseline hazard, regardless of whether hazards increase or fall with age, the interpretation of θ depends on the baseline schedule. When hazards are rising, then $\theta = 1/2$ slows senescence. If hazards are falling, $\theta = 1/2$ increases the amount of time it takes for hazards to fall. Thus, the model would delay 'maturity' or slow 'growth' (Baudisch 2008).

Increases in longevity that come from what we call 'senescence-slowing' can be contrasted with increases that come from declines in the level of hazards (proportional hazards) and from increases that come from transforming the timing of the distribution of deaths (accelerated failure time). Our results show how much of an impact slowing senescence has on life expectancy, clarifying the relationship between these different sources of mortality change. In particular, the results allow us to see why, in modern populations, reducing the pace of senescence by 1 per cent, for example, will have a much larger effect than reducing mortality levels by 1 per cent. Our findings echo those of lifetable entropy (Keyfitz 1977; Mitra 1978; Goldman and Lord 1986; Vaupel 1986), except that we consider the case of changing the 'slope'—the pace of ageing—rather than the level of mortality. As lifetable entropy falls, declining mortality levels have a smaller and smaller effect on life expectancy, but a declining pace of senescence has a larger and larger effect.

The model we call 'senescence-slowing' has been recently introduced into the statistical literature by Chen and Wang (2000). They call it the *accelerated-hazards* model, contrasting it with *proportional-hazards* (Cox 1972) and *accelerated-failure-time* (e.g., Kalbfleisch and Prentice 2002) models. (All three of these models are 'semi-parametric' in the sense that the modelled hazard schedule is a parametric transformation of an arbitrary baseline schedule.) Because the new literature on accelerated-hazards models is primarily concerned with multivariate estimation, it appears that the result we present for expected values has not been previously stated in an explicit way. Another contribution of the paper is the new relation between Keyfitz's entropy and the change in life expectancy that would follow from slowed senescence. A final contribution of our paper is to provide results for life expectancy under a 'hybrid' model that merges the proportional-hazards and senescence-slowing models.

Section 2 states our main results and Section 3 contains derivations of these formulae. Section 4 starts with a numerical illustration of slowed senescence, followed by a formal comparison of several semi-parametric models of mortality change. Section 5 provides two applications of the model—an empirical application to recent mortality decline in Japan and a theoretical application to the relationship between period and cohort life expectancy. We refer to the accelerated-hazards model as 'senescence-slowing', and the accelerated-failure-time model as 'death-delaying', linking hazards to senescence and failures to deaths.

2. The effect of slowing senescence on life expectancy

Keyfitz considered the effect of a proportional change $\theta = 1 + \delta$ in hazards at all ages such that the new hazard of death μ^* was a multiple of the baseline hazard μ_0:

$$\mu^*(a) = (1 + \delta)\mu_0(a). \tag{1}$$

He found that the effect on life expectancy of such a change in the level of mortality could be approximated by

$$\frac{\Delta e^*(0)}{e(0)} \approx -H\delta \tag{2}$$

where $\Delta e^*(0)$ refers to a small change in life expectancy at age 0 and the 'entropy' quantity H is defined as

$$H = \frac{-\int [\log \ell(a)]\, \ell(a)\, \mathrm{d}a}{e(0)}. \tag{3}$$

In a typical modern lifetable for adults, H is in the range of 0.1–0.2, and so reducing hazards by 1 per cent will increase life expectancy by only about 0.1–0.2 per cent. Some have used this formal property to argue that it will be difficult to make rapid progress in improving longevity (Olshansky 2001).

We model a change in the rate of senescence by letting the hazard μ^{**} at age a be the hazard

observed in the baseline schedule μ_0 at the age $a\theta$, so that

$$\mu^{**}(a) = \mu_0(a\theta)$$

for non-negative θ. Our first result, derived below, is that the new life expectancy at birth is

$$e^{**}(0) = \frac{1}{\theta}\int_0^\infty \ell(a)^{1/\theta}\,\mathrm{d}a \tag{4}$$

where $\ell(a)$ is the survival function associated with the baseline hazard schedule. This result is general in the sense that no restrictions are made on the baseline lifetable.

Our second result, also derived below, is that the relative change in life expectancy produced by accelerating senescence by δ can be given in terms of entropy H. Paralleling Keyfitz's notion by letting $\theta = 1 + d$, we find

$$\frac{\Delta e^{**}(0)}{e(0)} \approx -(1-H)\delta \tag{5}$$

where H is Keyfitz's entropy given above.

Equation (5) allows us to estimate the effect of slowing senescence. Considering again entropy levels in the range 0.1–0.2, a 1 per cent slowdown in the pace of senescence would increase adult life expectancy by 0.8–0.9 per cent, as contrasted with the 0.1–0.2 per cent increase from a similar decline in the level of mortality. The two effects are complementary, summing to one. As entropy varies, the larger the effect of proportional hazards the smaller the effect of slowing senescence, and vice versa.

3. Derivations

Equation (4) for life expectancy in terms of θ can be obtained via repeated substitution in the integral

$$e^{**}(0) = \int_0^\infty \exp\left[-\int_0^x \mu_0(a\theta)\mathrm{d}a\right]\mathrm{d}x \tag{6}$$

First define a new variable $w = a\theta$ to obtain

$$\begin{aligned} e^{**}(0) &= \int_0^\infty \exp\left[-\frac{1}{\theta}\int_0^{x\theta} \mu_0(w)\mathrm{d}w\right]\mathrm{d}x \\ &= \int_0^\infty \ell(x\theta)^{1/\theta}\mathrm{d}x \end{aligned} \tag{7}$$

and then define $v = x\theta$ to obtain

$$e^{**}(0) = \frac{1}{\theta}\int_0^\infty \ell(v)^{1/\theta}\mathrm{d}v = \frac{1}{\theta}\int_0^\infty \ell(a)^{1/\theta}\mathrm{d}a.$$

Equation (5) can be obtained by paralleling Keyfitz's analysis, approximating the effect of a small change in senescence on life expectancy as $\Delta e^{**}(0)$ with $\delta\cdot(\mathrm{d}e^{**}/\mathrm{d}\delta|_{\delta=0})$. Thus

$$\begin{aligned} \frac{\Delta e^{**}(0)}{e(0)} &\approx \frac{\delta\cdot \frac{\mathrm{d}e^{**}}{\mathrm{d}\delta}|_{\delta=0}}{\mathrm{e}(0)} \\ &= \frac{\delta\cdot\left[-\left(e(0) + \int[\log \ell(a)]\ell(a)\mathrm{d}a\right)\right]}{e(0)} \\ &= -(1-H)\delta \end{aligned}$$

where the last equality uses H as in equation (3).

The quantity $-(1-H)$, which measures the relative impact on life expectancy at age 0 of a small change in senescence, is also equal to $\left(\int \ell(a)(\mathrm{d}/\mathrm{d}a)e(a)\mathrm{d}a\right)/e(0)$, where $(\mathrm{d}/\mathrm{d}a)e(a)$ is the derivative of $e(a)$ with respect to age a. Thus $-(1-H)$ is a weighted average of the changes in age-specific life expectancy with weights given by the probabilities of surviving to each age. As mentioned earlier, typical values of H for adults are far less than 1, so the quantity $1-H$ is positive. Consequently, slowing senescence corresponds to greater longevity. A sufficient condition for H to be less than 1 is that hazards increase monotonically.

4. Comparing different kinds of mortality change

4.1. Numerical illustration of reducing the pace of senescence for Swedish females

To illustrate the consequences of reducing mortality via slowed senescence and other models, we used the 2007 period lifetable for Swedish females from the Human Mortality Database. In order to consider only the senescent portion of life, we focused on adult mortality, defined as mortality at ages 30 and above. Figure 1 shows how a contemporary lifetable would respond to different kinds of mortality change, and allowed us to compare and contrast these effects.

The first panel shows the observed age pattern of death rates, along with three kinds of mortality reductions. The proportional-hazards model, shown with the dotted line, reduces mortality rates by 20 per cent at all ages. The senescence-slowing model, shown with the heavy dashed line, reduces the change in mortality with respect to age by 20 per cent. The death-delaying model, shown with the lighter dot-dash line, combines these two effects.

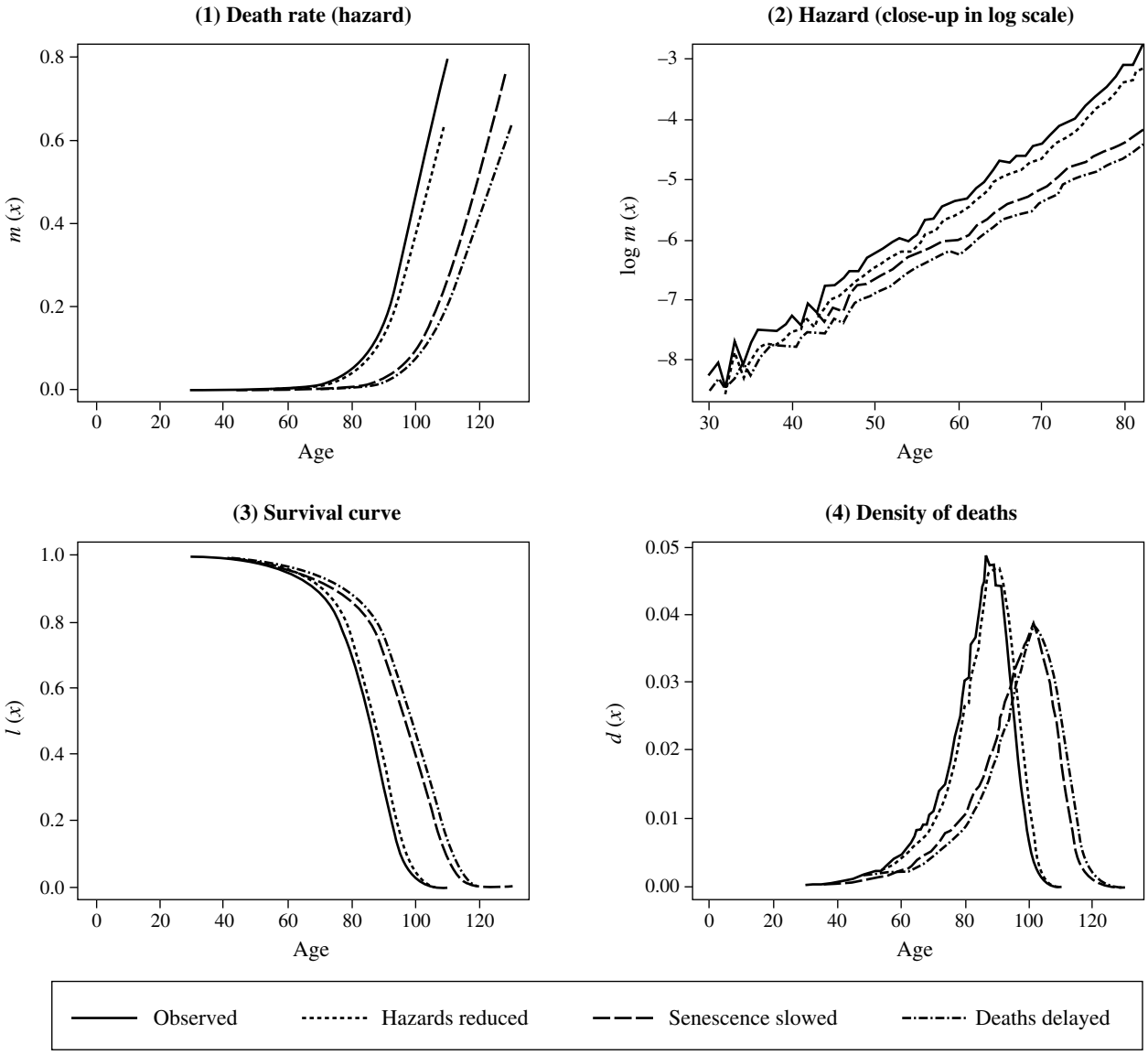

Figure 1 Comparison of the effect of various kinds of stylized mortality decline for Swedish females over 30 years of age
Source: 2007 period lifetable from the Human Mortality Database.

The second panel shows a close-up view of the same mortality rates across a more limited range of ages and in the logarithmic scale. The near linearity of these logarithmic curves means that mortality is increasing approximately exponentially with age, as in the Gompertz model. The close-up view shows us that the senescence-slowing and death-delaying models change the slope of mortality increase with age, whereas the proportional-decline model retains the slope of the original observations, but at a lower level. It is in this sense that senescence-slowing can be considered a slope change. The third and fourth panels of the figure show the impact of changing mortality rates on the survival curve and distribution of deaths. We see that the change in the survival curve is large for the senescence-slowing and death-delaying models but small for the proportional-decline model. The distribution of deaths is moved to older ages as a result of the proportional change in deaths, a well-known result of applying proportional hazards to the Gompertz model (e.g., Vaupel 1986; Goldstein and Wachter 2006). In addition to a change in 'location', the slowing of senescence also broadens the distribution of deaths.

The life expectancies at age 30 associated with the four curves are 53.5 for the observed lifetable, 55.4 for the proportional reduction, 64.3 for senescence-slowing, and 66.7 for delayed-deaths.

4.2. A more formal comparison, along with a flexible hybrid model

We show a formal comparison of selected lifetable functions under various semi-parametric models of mortality change in Table 1. The first column gives the name of the model as used in this paper. The 'baseline' row provides the definitions of the baseline hazards $\mu(a)$ and survival $\ell(a)$ used in the rest of the table. The standard proportional-hazards results are shown in the next row. The 'senescence-slowing' row summarizes the results given in equations (4) and (5), which were derived in Section 3. The 'death-delaying' row gives the definition of the standard lifetable functions according to the accelerated-failure-time model (e.g., Kalbfleisch and Prentice 2002) in terms of the common parameter θ. The derivations for this model are

$$\begin{aligned}\text{survival} &= \exp\left[-\int_0^a \theta\mu_0(x\theta)dx\right] \\ &= \exp\left[-\int_0^{a\theta} \mu_0(w)dw\right] = \ell(a\theta), \\ \text{life expectancy} &= \int_0^a \ell(x\theta)dx = \frac{1}{\theta}\int_0^\infty \ell(a)da,\end{aligned}$$

and

$$\begin{aligned}\frac{\Delta e(0)}{e(0)} &\approx \frac{\delta}{e(0)}\cdot\left[\frac{\partial}{\partial\delta}\left(\frac{1}{1+\delta}\int_0^\infty \ell(a)da\right)|_{\delta=0}\right] \\ &= \frac{\delta\cdot\left[-\int \ell(a)da\right]}{e(0)} = -\delta.\end{aligned}$$

Table 1 allows us to see that the death-delaying model can be thought of as a particular combination of the proportional-hazards and senescence-slowing models. These three approaches to changing mortality—proportional reductions in hazards, senescence-slowing, and death-delaying—can be subsumed in a more general model. We introduce this more general model, which we call the 'hybrid model', in the last row of Table 1. In this case we modify the baseline mortality schedule $\mu_0(a)$ with two parameters θ_1 and θ_2 to get

$$\mu(a) = \theta_1\mu_0\ (\theta_2 a)$$

where the constant θ_1 provides a proportional change in mortality and θ_2 changes the rate of senescence. Within this framework, the proportional-hazards model is the case where $\theta_2 = 1$, the senescence-slowing model corresponds to $\theta_1 = 1$, and the death-delaying model refers to the particular circumstance with $\theta_1 = \theta_2$. The hybrid model highlights how the death-delaying model is a limited special case of something more general. We present an application of the hybrid model to recent mortality decline in Japan below.

The final column in Table 1 gives the proportional effect on life expectancy of a small change in θ, with $\delta = \theta - 1$. For the hybrid model we use $\delta_1 = \theta_1 - 1$ and $\delta_2 = \theta_2 - 1$. In terms of the proportional effect of a change in the parameters on life expectancy, we see that the death-delaying model is the combined effect of senescence-slowing and proportional hazards since $-H\delta + (-(1-H)\delta) = -\delta$. The effect of the hybrid model is also the combination of these two models but allows unequal values of δ_1 and δ_2.

We have used the parameter θ to measure the pace of senescence. For example, if a 30 year old faces the mortality rates previously known to a 60 year old, then it is natural to use the number 2 to describe the speed of senescence. There are however other descriptions of changing senescence. For example, the rate of change in age-specific mortality with age, introduced by Horiuchi (1983) and now called the lifetable-ageing rate (LAR), is given by

$$\frac{d\log(\mu(\alpha))}{da} = \frac{\frac{d}{da}\mu(\alpha)}{\mu(a)}.$$

Using the models in Table 1, we find that the LAR for the proportional hazards is the same as the LAR

Table 1 Summary of mathematical results for various semi-parametric models of changing mortality

Model	Hazard	Survival	Density	Life expectancy	$\frac{\Delta e}{e(0)}$
Baseline	$\mu(a)$	$\ell(a)$	$\mu(a)\ell(a)$	$\int \ell(a)\mathrm{d}a$	0
Proportional-hazards	$\theta\mu(a)$	$\ell(a)^\theta$	$\theta\mu(a)\ell(a)^\theta$	$\int \ell(a)^\theta \mathrm{d}a$	$-H\delta$
Senescence-slowing	$\mu(\theta a)$	$\ell(\theta a)^{1/\theta}$	$\mu(\theta a)\ell(\theta a)^{1/\theta}$	$\frac{1}{\theta}\int \ell(a)^{1/\theta}\mathrm{d}a$	$-(1-H)\delta$
Death-delaying	$\theta\mu(\theta a)$	$\ell(\theta a)$	$\theta\mu(\theta a)\ell(\theta a)$	$\frac{1}{\theta}\int \ell(a)\mathrm{d}a$	$-\delta$
Hybrid model	$\theta_1\mu(\theta_2 a)$	$\ell(\theta_2 a)^{\theta_1/\theta_2}$	$\theta_1\mu(\theta_2 a)\ell(\theta_2 a)^{\theta_1/\theta_2}$	$\frac{1}{\theta_2}\int \ell(a)^{\theta_1/\theta_2}\mathrm{d}a$	$-H\delta_1 - (1-H)\delta_2$

Note: $\delta = \theta - 1$, and in the hybrid model $\delta_1 = \theta_1 - 1$ and $\delta_2 = \theta_2 - 1$. The rightmost column represents approximations that are sufficiently accurate if δ is small.

for the baseline schedule. This is not surprising since the proportional-hazards model does not change the slope of mortality. Similarly, the death-delaying and senescence-slowing models both influence LAR in the same way, as does the hybrid model (assuming $\theta_2 = \theta$). Again, this is to be expected since each of these models changes the slope of mortality hazards in the same way.

5. Applications

5.1. An empirical example of the 'hybrid' model to slowing senescence with additional declines in mortality level in Japan

We now consider an application of the 'hybrid' model to a recent example of mortality decline. From the many patterns of observed mortality decline over time and across populations, we chose the example for Japanese adult males for 1990 and 2005 shown in Figure 2. It can be seen from the figure that although mortality fell at all ages, the decline was substantially faster at older ages than at younger ages. The fit of the 'hybrid' model is shown in the dashed line, with $\hat{\theta}_1 = 0.928$ and $\hat{\theta}_2 = 0.961$. It can be seen from the close fit of the hybrid estimate and the observed 2005 mortality rates that a decline in both level and slope appears to be a good description of the change in mortality in this period. In particular, the hybrid model fits very well from ages 30 to 90. Only at the oldest ages does the slowing of senescence appear to be less than the model values.

From 1990 to 2005, the life expectancy of Japanese males at age 30 rose from 47.17 to 49.42 years. The 'hybrid' model can show how much of this increase was due to decline in mortality level (δ_1) and how much to slowing senescence (δ_2). We estimated $\hat{\delta}_1 = -0.072$ and $\hat{\delta}_2 = -0.039$. In words: the level of mortality fell by about 7 per cent while the pace of ageing fell by about 4 per cent. (We estimated $\hat{\delta}_1$ and $\hat{\delta}_2$ using the non-linear least squares function nlm() in R. We minimized the sum of squared residuals of log-mortality rates plus the sum of the squares of the difference in life expectancy calculated from fitted and observed age schedules. More robust methods could certainly be developed.)

Under the hybrid model, the first-order Taylor series estimate of the change in life expectancy relative to life expectancy is

$$\frac{\Delta^{(1)} e(0)}{e(0)} = -H\delta_1 - (1 - H)\delta_2.$$

From the 1990 lifetable we observe an increase of 2.25 years in life expectancy at age 30. Using the lifetable we calculate that H is approximately 0.21, which gives us an estimated 2.17-year increase in life expectancy at age 30, of which 0.71 years was due to decline in level and 1.46 years was due to decline in slope.

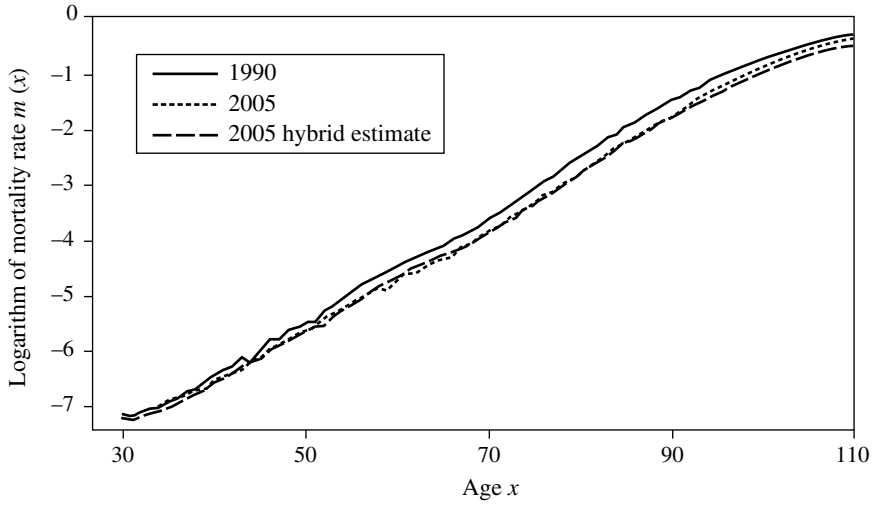

Figure 2 Fit of hybrid model combining slowing-senescence and proportional-hazards decline for mortality rates of Japanese adult males, 1990 and 2005, with hybrid model fit to 2005
Note: The hybrid model was fit by transforming the 1990 schedule using non-linear least squares to pick the most appropriate values of θ_1 and θ_2 (equivalently, δ_1 and δ_2) with the additional objective that the life expectancy of the fitted 2005 schedule be very close to that of the observed 2005 schedule.
Source: Human Mortality Database.

The decomposition can be improved by using a second-order Taylor series approximation as

$$\frac{\Delta^{(2)}e(0)}{e(0)} = \left[-H\delta_1 + H_2\frac{\delta_1^2}{2}\right] + \left[-(1-H)\delta_2 + (2-4H+H_2)\frac{\delta_2^2}{2}\right] + [(2H-H_2)\delta_1\delta_2]$$

where $H_2 = \left(\int \log(l_x)^2 l_x dx\right)/e_0$ and δ_1^2 and δ_2^2 are the squares of δ_1 and δ_2. Using the 1990 lifetable again we get a value for H_2 that also rounds to 0.21. In this case, the estimated life expectancy increase is 2.27 years (as compared to the 2.25-year change observed), with 0.73 years due to falling levels given by the first bracketed term, 1.51 years due to declining slope given by the second bracketed term, and 0.03 years due to the interaction of slope and level effects given by the third bracketed term. The small value of the interaction term suggests that the decomposition into additive slope and level effects is a nearly complete description of the change in life expectancy.

The hybrid model thus allows us to say that in the case of recent improvements in the mortality of Japanese males, changes in level were responsible for about one-third of adult life expectancy improvement, whereas the apparent slowing of senescence was responsible for about two-thirds. Although the changes in level were nearly twice the magnitude of the changes in slope, the low entropy value (≈ 0.2) meant that the slope change had a bigger impact on life expectancy than the change in level.

In this application, we chose an example in which both slope and level effects contributed to longevity increases. The hybrid model can also describe effects that go in opposite directions, such as the decline in level and increase in slope that describes much of the mortality decline observed until recently.

5.2. Cohort–period translation as slowing senescence

A second, more theoretical application of the senescence-slowing model is to the problem of the relationship between period and cohort life expectancy (Canudos-Romo and Schoen 2005; Goldstein 2006; Goldstein and Wachter 2006; Rodríguez 2006). Here we show that when mortality is shifting steadily to older ages, the cohort lifetable is a senescence-slowed version of the period lifetable.

Bongaarts (2005) considers a shift-based model of adult mortality, namely

$$\mu_s(a,t) = \mu_s(a - S(t), 0)$$

where $S(t)$ is the amount of shift in years up or down the age axis, and the subscript s distinguishes the part of mortality that varies with age from the background mortality that does not change with age. The case of linear shifts is given by $S(t) = rt$.

Cohort life expectancy under linear shifts corresponds to a slowed-senescence version of period life expectancy with $\theta = 1 - r$. To see this, note that the cohort born in year $t = 0$ experiences hazards $\mu_s(a,a) = \mu_s(a(1-r),0)$ which in senescence-slowing terms is $\mu_0(\theta a)$, with $\theta = 1 - r$. Thus, for any pattern of mortality improvement based on linear shifts, regardless of the baseline hazard, cohort life expectancy will be given exactly by the equivalent senescence-slowed version of period life expectancy. Furthermore, the same relationships involving entropy apply, with cohort life expectancy being bigger than period life expectancy by a proportional amount approximated by $r(1-H)$ and an absolute gap approximated by $e_0 r(1-H)$.

In the special case of a Gompertz mortality schedule with continuous proportional decline, the slowing rate of senescence of the cohort relative to the period can be seen directly. When

$$\mu(a,t) = \alpha(t)e^{\beta a} = \alpha_0 e^{-kt}e^{\beta a}$$

the cohort born in year t experiences hazards $\mu(a,t+a) = \alpha(t)e^{(\beta - k)a}$. This is equivalent to changing the speed at which hazards rise with age, and thus analogous to the senescence-slowing model of $\mu(a) = \mu_0(\theta a)$, with $\theta = 1 - k/\beta$. Applying our result (5) for slowing senescence to the Gompertz case, the gap between cohort and period life expectancy (cf., Goldstein and Wachter 2006) is approximately $(k/\beta)(1-H)e_0$, since here $-\delta = k/\beta$.

To get a sense of the magnitude of the difference between period and cohort life expectancy, let $\beta \approx 0.1$ and $\alpha \approx 0.0001$. The Gompertz schedule then gives us $e_0 \approx 63$ years and $H \approx (1/e_0\beta) \approx 0.16$. The 1 per cent annual decline in hazards ($k = 0.01$) produces a cohort life expectancy $(0.01/0.1)(1 - 0.16) \approx 8$ per cent longer than period life expectancy, corresponding to a value of 69 years, or 6 years longer than period life expectancy.

6. Discussion

The complementarity between the entropy H of the proportional-hazards model and the effect of changing the rate of senescence $(1 - H)$ given in (5) means lifetables with a large response to changing the level of hazards will have a small response when changing the rate of ageing, and vice versa. For example, when hazards are constant, $1 - H = 0$, which makes sense since, under constant hazards, transforming age from a to θa makes no difference. At the other extreme, if hazards are zero until some age when they become infinite, all deaths will be concentrated at this age. In this case, $H = 0$, since proportional changes in the level of hazards taking the value of zero or infinity are without consequence. Any change in the age at which hazards become infinite, however, will be perfectly reflected in a change in life expectancy, with $1 - H = 1$.

Current human lifetables in low-mortality countries have values of H approximately from 0.1 to 0.2. A 10 per cent decline in hazards at all ages would increase life expectancy by 1–2 per cent. A 10 per cent slowdown in the pace of senescence would increase life expectancy by 8–9 per cent. As H gets smaller, the distinction between the senescence-slowing model and the death-delaying model becomes less important. This is because the additional effect of changing the level of hazards has a smaller and smaller impact relative to the effect of changing the slope.

Historically, entropy for adults has fallen over time, from about 0.3 in nineteenth-century Sweden to about 0.1 in contemporary low-mortality populations. This decline in H means that the relatively greater impact of slope vs. level changes has itself increased over time. Whereas in the nineteenth century the benefit of slowing the ageing process would have been perhaps 2–3 times the benefit of lowering the level of mortality, today the benefit is nearly 10 times as large. The mathematics of mortality change thus provides good reasons why researchers on ageing want to focus now, more than ever, on slowing ageing itself. It remains to be seen how difficult or costly it will be to slow ageing, but the mathematics shows us that even small reductions in the pace of ageing could lengthen lives considerably.

Notes

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