

Variance in Death and Mortality Decline

Shripad Tuljapurkar and Ryan D. Edwards *

September 20, 2007

The expansion of human life in the past century (1; 2) and its socio-economic implications have stimulated efforts to analyze and forecast mortality trends (3). A natural focus of these efforts is the period expectation of life at birth, e_0 (a life expectancy computed from death rates in a particular period). Mortality change is commonly summarized in terms of trends in e_0 , and mortality models are evaluated on their ability to match historical trends in life expectancy. These uses of e_0 gained considerable support from two recent findings: that e_0 has increased at a nearly constant rate in many industrial countries since 1955 (4), and that since 1840 annual world record female e_0 has also increased at a nearly constant rate (5). Some have argued that such constancy is fundamental in analyzing mortality change (6; 7), and one researcher (8) has extended a simple model (9) to forecast mortality change. But e_0 is only the mean of the distribution of ages at death, and we show here that the variance of this distribution provides important additional information. In particular, change in the variance in age at adult death is not captured by simple models, and constrains how we should conceptualize and analyze mortality change.

This paper is organized in four parts. First, we discuss the change in the age distribution of period life-table deaths at very young versus older ages, and show that historical increases in e_0 in the industrialized countries have been accompanied by equally striking decreases in the variance of the age of adult death. These trends show clearly that mortality decline over

*Tuljapurkar: Morrison Professor of Population Studies, Department of Biological Sciences, Stanford University, tulja@stanford.edu. Edwards: Queens College and the Graduate Center City University of New York, redwards@qc.cuny.edu. Tuljapurkar acknowledges support from NIA grant 1P01AG22500. Edwards acknowledges support from the Morrison Institute and from NIA grant T32 AG000244.

time has compressed the variance between individuals at the same time as it has increased average life expectancy. Second, we show how the variance in age of adult death can be approximately computed for any reasonable model of mortality rates, and illustrate this with three commonly used models, the Gompertz, the logistic, and models with multiplicative frailty. We also show that any generalization of the Bongaarts-Vaupel translation argument yields an unchanging variance in the age at adult death. These results imply that the Bongaarts model does not capture a major qualitative aspect of mortality change in many industrialized countries. Fourth, we present results on world-record trends in the variance in age of adult death, and discuss the implications of our results for understanding secular mortality change.

Our focus here is on the variability at age of death using period distributions of the age at death. Our results are related to work on the “rectangularization” of the survivorship schedule (10; 11) and on the existence of a maximum age at death (12; 13; 14), questions which explore the possible limiting forms of the distribution of age at death. Our analysis makes no assumptions or deductions about such a limit, but aims to illuminate the nature and significance of trends in the variability of age at death.

Distributions of Age at Death

The age pattern of life table deaths in any period is found from mortality rates $\mu(a)$ by age a in that period. The survivorship $l(a) = \exp\{-\int_0^a ds \mu(s)\}$, and the probability density of death at age a is $\phi(a) = \mu(a) l(a)$. Figure 1 (a) displays this density for Swedish females in 1999, illustrating the early spike at due to infant and child deaths and the much larger probability of death at much older ages. The majority of deaths occur at ages much later than infant and early childhood, a qualitative pattern typical of industrialized countries in the past half century and more. To focus on the older ages at which most death occurs, we choose a cutoff age A which separates infant and early childhood deaths from later deaths. A suitable value of A lies in the range of ages at which probabilities of death are near their minimum, but is otherwise arbitrary. For choices of $A = 10$ and 20 years, Figure 1 (b) shows that the proportion of female deaths over age A in Sweden has been over 92% since 1940; a similar pattern obtains for most industrialized countries. In the rest of this paper we use $A = 20$ years; changing the cutoff to age 10 has little effect on our results. Taking some literary license, we refer to deaths at ages

over A as “adult” deaths, and earlier deaths as “young” deaths.

Decomposing Life Expectancy

How do young deaths (at ages $\leq A$ years) and adult deaths (at ages $> A$) contribute to the average age at death in the period life table? Write T for the random age of death of an individual in a hypothetical cohort following a period life table. Let p_-, p_+ be the probabilities of young death ($T \leq A$) and adult death ($T > A$) respectively. Then

$$e_0 = p_- e_- + p_+ e_+,$$

where e_-, e_+ are (conditional) average ages of death for those who die young or die as adults, respectively. In the industrialized countries in the last five or six decades, e_- is much below 1 year, and p_- is well under 10%, so the main determinant of e_0 is the timing of adult death. Of course, declines in young deaths still matter to e_0 , but their effect is proportional to the value of e_+ . Consistent with this observation, Wilmoth and Horiuchi (10) used different methods to show that mortality change at adult ages has been the main contributor to changing e_0 in recent decades.

Decomposing Variance in Age at Death

Consider again random ages at death T in a hypothetical cohort following a period life table. The variance of age at death can be written

$$\begin{aligned} \text{Var}(T) &= p_- V_- + p_+ V_+ \\ &+ p_- (e_- - e_0)^2 + p_+ (e_+ - e_0)^2, \end{aligned} \tag{1}$$

where V_-, V_+ are (conditional) variances of age at death for those who die young or die as adults, respectively. For the industrialized countries, only the second and third terms matter. The first term is small because both its components are small, and the last term is small because e_0 has become increasingly close to e_+ . The third term contributes substantially only because e_- is small: this difference is not informative about substantive variation in the adult ages at which most deaths occur. This argument is illustrated by calculations of the contributions in equation (1): we illustrate these in Figure 2 for Swedish females over the period 1920 to 2003. Thus the term in that most matters to understanding variability in age at death is V_+ . Consistent

with this analysis, (15) show that differences between countries in the distribution of age at death are increasingly determined by differences in V_+ rather than in e_0 . From here on, we measure variability in age at death by V_+ with a cutoff age of 20 years, or equivalently the standard deviation $S_{20} = \sqrt{V_+}$.

Trends in Variance in Age at Adult Death

How has the variance in age at adult death changed over time, especially in comparison to changes in e_0 ? To provide historical perspective, we plot S_{20} against e_0 for Swedish females in three epochs, an early period from 1751 to 1891 (Figure 3(a)), a middle period from 1892 to 1953 (Figure 3(b)), and the recent period from 1954 to 2003 (Figure 3(c)). In the earliest period (Figure 3 (a)) e_0 and the variability S_{20} in adult death changed in apparently chaotic fashion. Although e_0 changed over a large range, changes in S_{20} were relatively modest. In the middle period (Figure 3 (b)) the chaotic change of the early period was replaced by a striking pattern in which S_{20} declined steadily whereas e_0 increased steadily. In this middle period, variability S_{20} fell by 49% over a much larger range than in Figure 3(a), and did so in almost perfect negative correlation with e_0 , which increased by 42%. The correlated pattern of declining variability and increasing life expectancy continued but slowed in the final period (Figure 3 (c)), the past five decades. Here S_{20} declined by 7% while e_0 increased by 11%.

Figures 4(a) and (b) plot S_{20} against e_0 for sexes-combined period mortality data for several industrialized countries over the past five decades. Considerable heterogeneity is apparent. Japan’s experience since 1955 looks much like that of Sweden during 1892 to 1953, a long and steady decline in variance coupled with gains in e_0 (Figure 4(a)). Denmark, shown on the same axes, actually experienced a net rise in S_{20} overall, although its schedule almost looks like a level projection of Sweden’s. Other countries, like France, the U.S., and Canada until about 1980, appear to have followed no clear trend at all since 1955 (Figure 4(b)). The remarkable, sudden divergence of Canada from the French path seems to be unique among countries in our dataset, cannot be linked clearly to discrete events in national health policies, and remains a provocative subject for future research.

Interpreting these patterns with a single story is difficult. Different countries may be at different stages of a common variance transition (Japan vs. Sweden); they may have different “background” levels of variance (Den-

mark vs. Sweden, U.S. and France vs. others); and they may break with past trends altogether (Canada vs. France). In all cases the variability S_{20} does change over time, with the smallest change being about 3% and the largest about 8%. Our results on change in S_{20} are consistent with those of Wilmoth and Horiuchi's (10), who showed that interquartile ranges of the age at adult death in have narrowed in recent decades in the industrialized countries.

Our central finding is that temporal changes in mortality have substantially altered both life expectancy e_0 and the variability in the age at adult death, S_{20} . Over the long historical period, secular change has compressed the variation between individuals in age at death, just as it has increased life expectancy. Changes in variance in the age at adult death continue in most industrialized countries. Clearly these patterns should be reflected in useful models of mortality change. To understand whether and how they can be included in models of mortality change, we now turn to the manner in which commonly used mortality models describe variation in the age at death.

Models of Adult Mortality

How is the variance in age at adult death described by mortality models? The most celebrated model of adult age-specific mortality is the Gompertz. Recent work (16; 17) suggests that a logistic model describes old-age mortality more accurately than does a Gompertz model. The logistic can also be seen as a result of a model in which Gompertz mortality is modified by a multiplicative frailty (9). Frailty, if it occurs in this form, should clearly contribute to the variability in age at death. While it can, we do not find this channel to be an entirely compelling account of the historical trends in S_{20} . Overall, we will show that traditional models are not well-equipped to deal with variance, and we find that disturbing in light of its clear importance.

We now present analytical results showing how the variance in age at adult death depends on the parameters of mortality models. We consider in order a general mortality model, the Gompertz, the logistic, a general model with multiplicative frailty, and the Gompertz with multiplicative frailty. We close this section with an analysis of models of mortality translation (which we explain below) and of a more flexible framework that can be seen as a generalized Gompertz model, the Lee-Carter model (18).

General Mortality Model

Suppose that adult mortality $\mu(a)$ is an increasing positive function of age a . In terms of cumulative mortality $M(a) = \int_0^a ds \mu(s)$, the survivorship $l(a) = \exp\{-M(a)\}$ falls to zero as age a increases. The distribution of age at death for adults, $\phi(a) = \mu(a) l(a)$, increases at young adult ages and falls to zero at very high ages. We indicate derivatives with respect to age by a dash, so

$$\frac{d\phi}{da} = \phi' = \mu' l + \mu l'.$$

The change in the value of ϕ between age a_1 and a slightly larger age $a_1 + x$ is the sum of two terms. The first, $x \mu'(a) l(a)$, represents an increase in the probability due to the increase with age of the death rate μ ; the second, $x \mu(a) l'(a) = -x \mu^2(a)$, represents a decrease in the probability due to the decrease with age of survivorship $l(a)$. At the modal age at death, a_0 , these changes balance and

$$\mu'(a_0) = \frac{d\mu}{da} = \mu^2(a_0). \quad (2)$$

If the mortality curve steepens, the mode will shift to a younger age.

Near the mode a_0 , the age-at-death distribution $\phi(a)$ can be approximated (via Taylor expansion) by a quadratic function,

$$\phi(a) \simeq \phi(a_0) \left(1 - \frac{(a - a_0)^2}{2 \sigma^2} \right), \quad (3)$$

where

$$\sigma^2 = \left(\frac{\phi(a_0)}{|\phi''(a_0)|} \right) = \frac{\mu(a_0)}{|\mu''(a_0) - 2\mu^3(a_0)|}. \quad (4)$$

Here $\phi''(a_0)$ is the (negative) second derivative of $\phi(a)$ evaluated at the mode a_0 and $\mu''(a_0)$ is second derivative of $\mu(a)$ at the mode a_0 . When the distribution $\phi(a)$ is reasonably sharply peaked around the mode a_0 , we can approximate it by a normal distribution,

$$\phi(a) \simeq \phi(a_0) \exp\left(-\frac{(a - a_0)^2}{2 \sigma^2}\right). \quad (5)$$

This approximation provides a useful and often accurate estimate of the moments of $\phi(a)$ – we use it here and also check its accuracy by numerical

computation (**REF** asymptotic expansions). In particular, the variance in age at adult death is approximately given by the σ^2 appearing in equation (4). This variance depends on the curvature of the mortality function, i.e., whether the slope of mortality steepens or shallows around the modal age. If the curve steepens, then $\mu''(a_0) > 0$ and the variance is smaller than for a curve that shallows at the mode.

The Gompertz Model

We write the Gompertz mortality function as $\mu(a) = \mu_0 e^{\beta a}$. Equation (2) shows that the mode satisfies

$$\mu(a_0) = \beta,$$

so the modal age at death is

$$a_0 = (1/\beta) \log(\beta/\mu_0). \quad (6)$$

We expect a_0 to decrease if β increases, a property which holds for (6) so long as $a_0 > (1/\beta)$ which is true for any plausible human mortality pattern. The probability density of age at death for the Gompertz model is

$$\phi(a) = \mu(a) \exp\left(-\frac{\mu(a) - \mu_0}{\beta}\right).$$

Because the Gompertz mortality rises exponentially, the density ϕ falls steeply at very high ages.

The variance in adult age at death for the Gompertz model is found from equation (4) to be approximately

$$\sigma^2 \simeq \frac{1}{\beta^2}. \quad (7)$$

Thus the Gompertz variance in age at death depends (at least approximately) only on the slope parameter β and not on μ_0 . It is possible to obtain an exact expression for the variance by analytical integration in terms of special functions but the results are not especially illuminating. However, we have computed numerically the exact variance for a range of values of β and μ_0 that are appropriate for twentieth century human mortality. We find that the exact value of σ depends only weakly on μ_0 and that equation (7) is an accurate approximation. It follows that a Gompertz model can only describe changes in the variance of the adult age at death if the Gompertz slope parameter β changes with time.

The Logistic Model

We write the logistic model for mortality as

$$\mu(a) = \frac{e^{\beta a}}{C + e^{\beta a}},$$

and integration shows that the probability density of deaths is

$$\phi(a) = (C + 1)^{1/\beta} \frac{e^{\beta a}}{(C + e^{\beta a})^{(1+1/\beta)}}.$$

This density falls as a simple exponential e^{-a} for high ages, much more slowly than for the Gompertz model. For the logistic, the modal age at death is

$$a_0 = \frac{1}{\beta} \log(\beta C), \quad (8)$$

and the approximate variance from equation (4) is found to be

$$\sigma^2 = \frac{(1 + \beta)}{\beta^2}. \quad (9)$$

Thus the logistic also displays the remarkable property that the variance in age at death depends only on the slope parameter β . It follows that a logistic model can only describe changes in the variance of the adult age at death if the slope parameter β changes with time.

Note that if we fit a Gompertz model and a logistic model to a particular data set, the value of β must be similar in both (compare the two models near $a = 0$ which here indicates the start of adult age), so the logistic model would imply a slightly larger variance in age at adult death than the Gompertz. We expect this difference because the density ϕ for the logistic model shallows as age increases (see the discussion after equation (5)).

General Mortality, Multiplicative Frailty

Following Vaupel (9), suppose that every individual has a random frailty Z and that $g(z) dz$ is the probability that Z takes values between z and $z + dz$. Mortality is determined by frailty Z and a baseline mortality function $\mu(a)$ as the product $Z \mu(a)$. Conditional on frailty, the probability distribution of age at death is

$$\phi(a|Z) = Z \mu(a) \exp(-Z M(a)),$$

with $M(a) = \int_0^a ds \mu(s)$. The usual specification of a frailty distribution assumes that average frailty is 1, and that the distribution of frailty has some variance $s^2 > 0$. The population probability distribution of age at death is the average over frailty,

$$\phi(a) = \mathcal{E} \phi(a|Z) = \int dz g(z) \phi(a|z). \quad (10)$$

Relative to individuals with a frailty of 1, less frail individuals will have a higher modal age at death and a larger variance in age at death.

We now obtain the mode and approximate variance of the general model. To simplify the equations below, it is convenient to define the following averages with respect to frailty,

$$h_j(a) = \mathcal{E} [Z^j e^{-Z M(a)}], \text{ for } j = 1, 2, 3. \quad (11)$$

In the population, the modal age at death satisfies the condition

$$\mu'(a) = \mu^2(a) \frac{h_2}{h_1}. \quad (12)$$

Note that if every frailty were set equal to 1, we would have $h_2 = h_1$ and this equation would reduce to our earlier equation (2). To approximate the variance in age at death, we use equation (4) and obtain

$$\phi''(a_0) = h_1 \mu'' + \mu^3 \left\{ h_3 - 3 \left(\frac{h_2^2}{h_1} \right) \right\}, \quad (13)$$

where the h_i are evaluated at the mode a_0 , and then set

$$\sigma^2 = \phi(a_0)/|\phi''(a_0)| = h_1(a_0)\mu(a_0)/|\phi''(a_0)|.$$

In these expressions, if every frailty is set equal to 1, we have $h_3 = h_1$ and the variance σ^2 reduces to the value in equation (4).

General Mortality, Gamma Multiplicative Frailty

The expressions we present above are not as illuminating as one might hope about how frailty would affect the mode or the variance in age at death. To obtain a qualitative sense of the effect of frailty, we consider the case when

frailty Z follows a gamma distribution (9). In this case, the probability that Z lies between w and $w + dw$ is assumed to be $g(w) dw$ with

$$g(w) = \frac{k^k}{\Gamma(k)} w^{k-1} e^{-kw}. \quad (14)$$

The average frailty is 1 and the variance of frailty is $\text{Var}(Z) = s^2 = (1/k)$. This distribution is convenient, as Vaupel et al. pointed out, because we can use it with any baseline mortality $\mu(a)$, to find an explicit expression for the average distribution of age at death in equation (10), in the form

$$\phi(a) = \mu(a) \left(\frac{k}{k + M(a)} \right)^{k+1}. \quad (15)$$

We can straightforwardly differentiate the above $\phi(a)$ to find that the modal age at death is defined by the condition

$$\mu' = \left(\frac{1 + s^2}{1 + s^2 M} \right) \mu^2. \quad (16)$$

Notice that if all individuals have the same frailty so that $s^2 = 0$, the equation for the mode reduces to the simpler equation (2). Qualitatively, the denominator on the right describes how frailty alters the rate of change of average mortality and survival depending on how much selection acts against more frail individuals. The magnitude of selection depends on both the variance s^2 in frailty, and the cumulative mortality hazard $M(a)$. Strong selection will act to decrease the modal age at death.

At the modal age, the second derivative of the age distribution of deaths can be found by differentiation and then substituted into the approximation equation (3) to obtain the variance in age at death,

$$\begin{aligned} \sigma^2 &= \left(\frac{\phi(a_0)}{|\phi''(a_0)|} \right), \\ &= \frac{\mu(a_0)}{|\mu''(a_0) - \mu^3(a_0) \{(1 + s^2)(2 + s^2)\} / \{(1 + s^2 M(a_0)^2)\}}|. \end{aligned} \quad (17)$$

Note again the selection effect in the denominator of (15) in which s^2 is multiplied by the cumulative mortality $M(a_0)$ which has occurred at ages below the mode. Strong selection (via a large $M(a_0)$) will combine with variance in frailty s^2 to reduce the denominator of equation (17) and thus to inflate the variance σ^2 in age at death.

Gompertz Mortality, Gamma Multiplicative Frailty

The gamma frailty model provides a little insight into how multiplicative frailty affects the mode and variance of age at death. But we can learn much more by combining a Gompertz baseline mortality $\mu(a) = \mu_0 e^{\beta a}$ with multiplicative gamma-distributed frailty. The modal age at death for this model is found using equation (16) with the Gompertz mortality, and yields the condition

$$\mu(a_0) = (\beta - s^2 \mu_0),$$

which explicitly gives us the mode as

$$a_0 = (1/\beta) \log \left(\frac{\beta}{\mu_0} - s^2 \right). \quad (18)$$

Compare this with equation (6) for the standard Gompertz and observe that frailty acts to reduce the modal age at death.

The variance in age at death is obtained using equation (17) and equation (16) and a little algebra, and yields the remarkably simple result that

$$\sigma^2 = \frac{(1 + s^2)}{\beta^2}. \quad (19)$$

Comparing this with equation (7) for the standard Gompertz shows that frailty simply amplifies the variance in age at death. An important conclusion is that a Gompertz model with gamma frailty can only describe changes in the variance of the adult age at death if either the Gompertz slope parameter β or the variance s^2 in frailty or both change with time.

Mortality Translation

The model of mortality translation due to Bongaarts and Feeney (7) provides an appealingly simple description of mortality change. We can describe it simply in terms of a hypothetical cohort following a period life table. Let T_1 be the random age at death of an individual in this cohort in period t_1 , e.g., $t_1 = 1990$. In a later period t_2 , suppose that the effect of mortality change between the two periods is completely described by delaying each death by the same amount. Thus, each random age at death T_1 in the first period is replaced in the later period by the random age at death $T_1 + D$, where $D > 0$ is fixed. We see at once that the average age at death increases from

$e_{01} = \mathcal{E} T_1$ in period t_1 to $e_{01} + D$ in the later period t_2 . If we shift the mean age at death at some fixed annual rate, we have found a model of mortality change that describes a constant trend in e_0 . We use the term mortality translation for any such model.

Notice that translation only affects the mean age at death and not its variance. Shifting every random age at death from T_1 to $T_2 = T_1 + D$ for a fixed D results in a constant variance, $\text{Var}(T_1) = \text{Var}(T_2)$. In fact, translation leaves unchanged all the central moments of the random age at death. Put geometrically, translation necessarily implies that the shape of the distribution of age death does not change.

Mortality translation is appealing because it can be used with any mortality model. Bongaarts and Feeney (7) and Bongaarts (8) used translation for a Gompertz and a logistic model. Vaupel (9) used a Gompertz model in essentially the same way although he did not explicitly refer to translation. Take any reasonable adult mortality function $\mu(a)$. In period t_1 suppose that the corresponding mortality $\mu_1 > 0$ for ages greater than some cutoff age A_1 . In a later period t_2 define adult mortality to be a translation of the original mortality schedule,

$$\mu_2(a) = 0, \text{ for } A_1 \leq a < (A_1 + D),$$

and

$$\mu_2(a) = \mu_1(a - D), \text{ for } a \geq (A_1 + D).$$

It follows automatically that the probability distribution of ages at death is also translated: if ϕ_1 and ϕ_2 are the distributions in the two periods, then

$$\phi_2(a) = \phi_1(a - D), \text{ for } a \geq (A_1 + D).$$

This is simply an alternative statement of the translation of the random age at death T_1 distributed as ϕ_1 to $T_1 + D$.

It is obvious that mortality translation, by construction, cannot describe changes in the variance in the probability distribution of age at death (or for that matter, of other central moments of ϕ related to the shape of the distribution, such as skewness or kurtosis).

Generalized Gompertz: Lee-Carter

Lee and Carter (18) proposed a parsimonious, three-parameter model that explains temporal trends in mortality well in industrialized countries (19).

Using the singular value decomposition, they estimate

$$\log \mu(a, t) = \alpha(a) + \beta(a) k(t), \quad (20)$$

where $\mu(a, t)$ is the mortality at age a in period t , $\alpha(a)$ and $\beta(a)$ are constant age profiles, and $k(t)$ is a random walk with negative drift.

The age profile $\alpha(a)$ is an average and so will be approximately Gompertz or logistic in shape. But the $\beta(a)$ profile is not necessarily constant with age, as it would be in a Gompertz model with a fixed age slope over time. Indeed, fits of $\beta(a)$ typically reveal that mortality declines at different rates at different adult ages across industrialized countries (20). In consequence the slope and curvature of mortality in this model are free to evolve over time, leading in general to changes in the variance of age at death. The singular value decomposition of Equation (20) produces optimal fits of age-specific mortality rates, however, not necessarily of the moments of the distribution of ages at death.

Implications and Discussion

Mortality Models

Over the last two centuries, the variance in age at adult death, measured here by the standard deviation S_{20} , has declined by about 50%. If we used a Gompertz model to describe period mortality at ages over 20, then the slope of the Gompertz model would have to increase by about 40% in order to replicate observed trends in S_{20} . A logistic model for period mortality would require a larger increase, about 50%, in the slope. It is true that in the past 50 years declines in S_{20} have been much slower, but they are still about 10%. Corresponding increases in the slope of the Gompertz model are about 11%, and for the logistic about 16%.

These findings, that the Gompertz slope cannot be constant over time and still match trends in variance, stand in stark contrast to traditional interpretations and uses of the Gompertz model. A common perspective is that the Gompertz slope is a constant parameter, similar even across many species, that is dictated by biology, while the intercept may vary according to external influences (21). We believe the evidence suggests that instead, both nature and nurture must affect the Gompertz slope, at least in human populations and potentially in other species.

Mortality translation models do not allow any change in the variance of adult death and should not be used to capture changes in the age pattern of deaths, even if they describe changes in e_0 . It is curious that translation models nonetheless provide a good statistical fit to mortality patterns in industrialized countries since 1950 (8). Our results suggest that analysis of the age distribution of deaths provides an important diagnostic test for mortality models that should be incorporated into active use.

The addition of multiplicative frailty to a Gompertz model does not address changing variances in age at death, unless we assume that frailty distributions have been changing quite rapidly over time. Temporal change in frailty has not been a feature of mortality models, and it is not clear why the distribution of such frailties would narrow over time.

By construction, the Lee and Carter (18) forecasting model can predict changes in the variance in the age at death. In practice, we have found that its implicit forecasts of S_{20} appear to be extrapolations of average trends over the forecast interval. This is consistent with the spirit of the Lee-Carter framework and mirrors patterns in Lee-Carter forecasts of e_0 . But since the historical paths of S_{20} have been considerably more convoluted than those of e_0 in every industrialized country, the simple extrapolation of the long-term average trend in S_{20} produced by Lee-Carter seems incongruous. We conclude that while the flexibility of Lee-Carter makes it a valuable model of mortality, it does not capture trends in variance as well as we believe they should be.

Components of Variability in Ages at Death

The variance $V_+ = S^2(20)$ in age at death which we study here is a sum of variability due to all individual differences. Suppose that individuals can be divided into groups (using e.g., socioeconomic risk factors, genes, and so on) and we partition the total variance into a sum of within-group variances and between-group variances. Equation (1) is an example of such a decomposition. Values of S_{20} provide significant constraints on any such decomposition. In particular, a change (or lack of it) in S_{20} over a period of time implies directly a consistent change in either within-group variance or between-group variance.

As one example, persistent educational differences in adult mortality have been documented over long periods of time (22). More broadly, socioeconomic gradients in mortality have existed throughout this period, and

remain significant today. In this long run, both e_0 and e_+ , the conditional expectation of life after reaching adult ages, have risen, and S_{20} has declined. If socioeconomic differences in mean age at adult death have stayed roughly constant over time, then the variance within groups must have declined because the overall S_{20} has declined. A precise decomposition of population variability by subgroups should be possible along the lines of this argument. Over the past half century, we have found (Figure 4) differences between countries in the pattern of change of S_{20} relative to e_0 . These provide a basis for a comparative analysis of trends in age at death by socioeconomic, genetic, or other factors.

Long-Term Trends in Ages at Death

A striking result about long run mortality change is the demonstration by (5) that world-record high e_0 has risen at a remarkable linear rate over the past 160 years. Such uniformity suggests that upper bounds on life span, prognosticated and then consistently broken throughout this period, are not as clear as some currently believe, and that the pace of human development and achievement measured in this way has been rapid and surprisingly steady across several distinct periods of socioeconomic and epidemiological transitions.

Elsewhere, we describe how long-term trends in the record-low variance paint a very different picture regarding the gains in human well-being along the dimension of mortality (15). True, progress against the Gompertz slope, has indeed been achieved, contrary to the opinions of those who may have viewed it as immutable. But long-term gains have come more in fits and starts rather than continuously, and this highlights the remaining challenges, as does the considerable heterogeneity across countries this century in progress against variance. We do not fully understand the sources of variance in life spans, nor the underlying health inequalities they presumably reflect, and this is a problem for policy as well as for modeling and forecasting mortality.

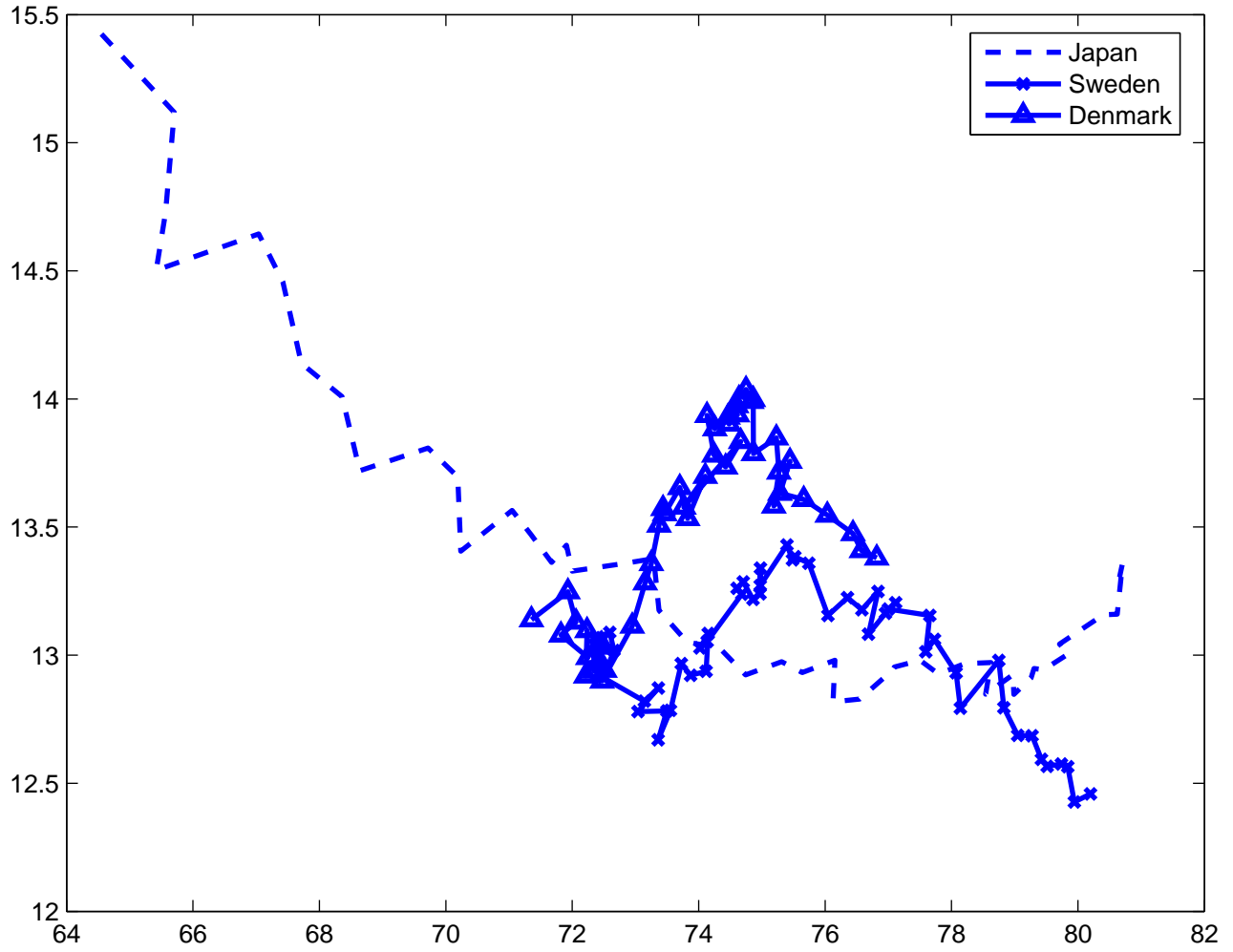
References

1. Preston, S. H. (1975) *Population Studies* **29**, 231–248.
2. Caldwell, J. C. (1976) *Population and Development Review* **2**, 321–366.

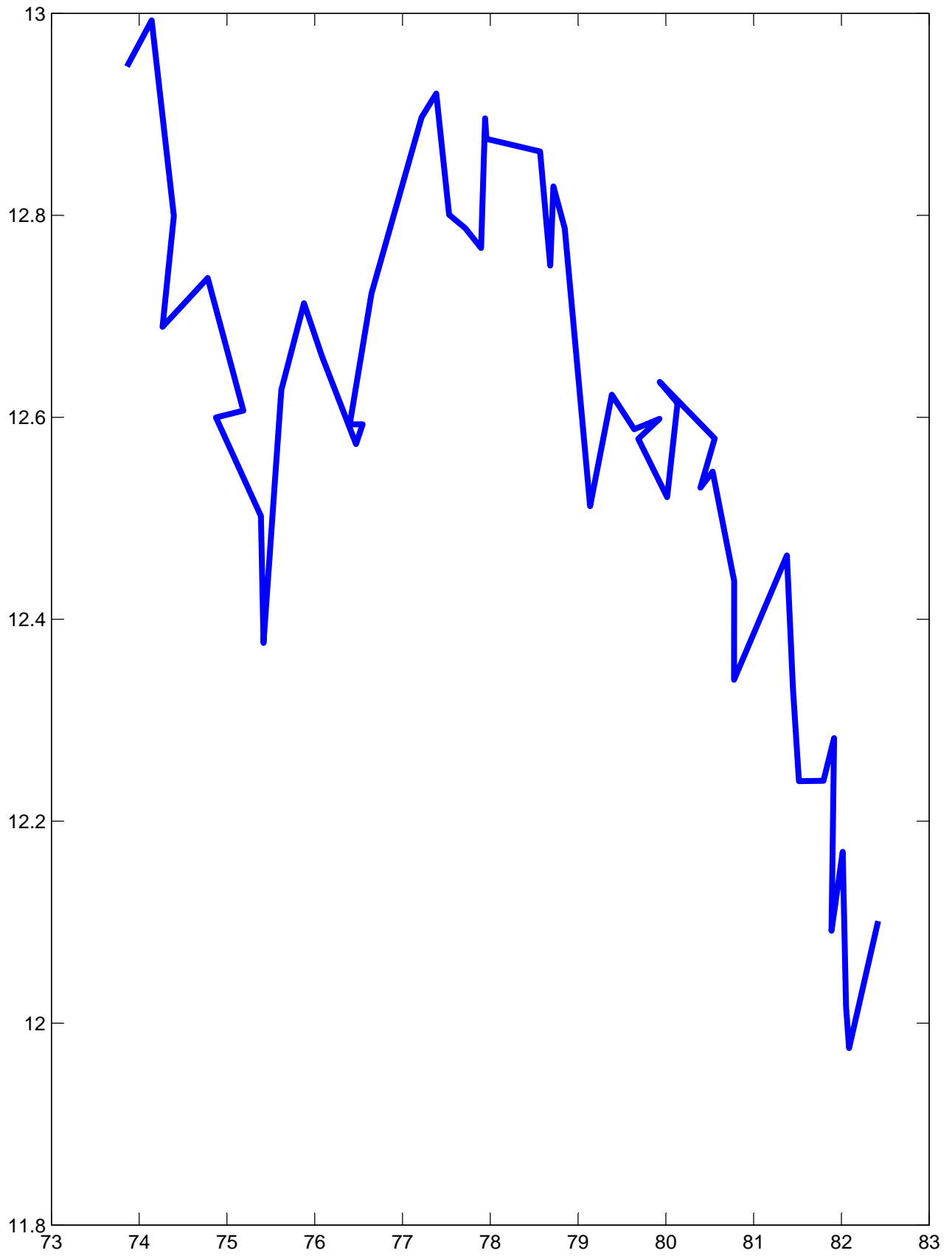
3. Tuljapurkar, S & Boe, C. (1998) *North American Actuarial Journal* **2**, 13–47.
4. White, K. M. (2002) *Population and Development Review* **28**, 59–76.
5. Oeppen, J & Vaupel, J. W. (2002) *Science* **296**, 1029–1031.
6. Bongaarts, J & Feeney, G. (2002) *Population and Development Review* **28**, 13–29.
7. Bongaarts, J & Feeney, G. (2003) *Proceedings of the National Academy of Sciences USA* **100**, 13127–13133.
8. Bongaarts, J. (2005) *Demography* **42**, 23–49.
9. Vaupel, J. W. (1986) *Population Studies* **40**, 147–157.
10. Wilmoth, J. R & Horiuchi, S. (1999) *Demography* **36**, 475–495.
11. Kannisto, V. (2000) *Demographic Research* **3**.
12. Fries, J. F. (1990) *New England Journal of Medicine* **303**, 130–135.
13. Olshansky, S. J, Carnes, B. A, & Cassel, C. (1990) *Science* **250**, 634–640.
14. Wachter, K. W & Finch, C. E, eds. (1997) *Between Zeus and the Salmon: The Biodemography of Longevity*. (Washington, DC: National Academy Press).
15. Edwards, R. D & Tuljapurkar, S. (2005) Inequality in life spans and mortality convergence across industrialized countries. Unpublished manuscript.
16. Vaupel, J. W, Carey, J. R, Christensen, K, Johnson, T. E, Yashin, A. I, Holm, N. V, Iachine, I. A, Kannisto, V, Khazaeli, A. A, Liedo, P, Longo, V. D, Zheng, Y, Manton, K. G, & Curtsinger, J. W. (1998) *Science* **280**, 855–860.
17. Thatcher, A. R, Kannisto, V, & Vaupel, J. W. (1998) *The Force of Mortality at Ages 80 to 120*. (Odense, Denmark: Odense University Press), No. 5.

18. Lee, R. D & Carter, L. R. (1992) *Journal of the American Statistical Association* **87**, 659–671.
19. Lee, R. D & Miller, T. (2001) *Demography* **38**, 537–549.
20. Tuljapurkar, S, Li, N, & Boe, C. (2000) *Nature* **45**, 789–792.
21. Finch, C. E, Pike, M. C, & Witten, M. (1990) *Science* **249**, 902–905.
22. Preston, S. H & Elo, I. T. (1995) *Journal of Aging and Health* **7**, 476–496.

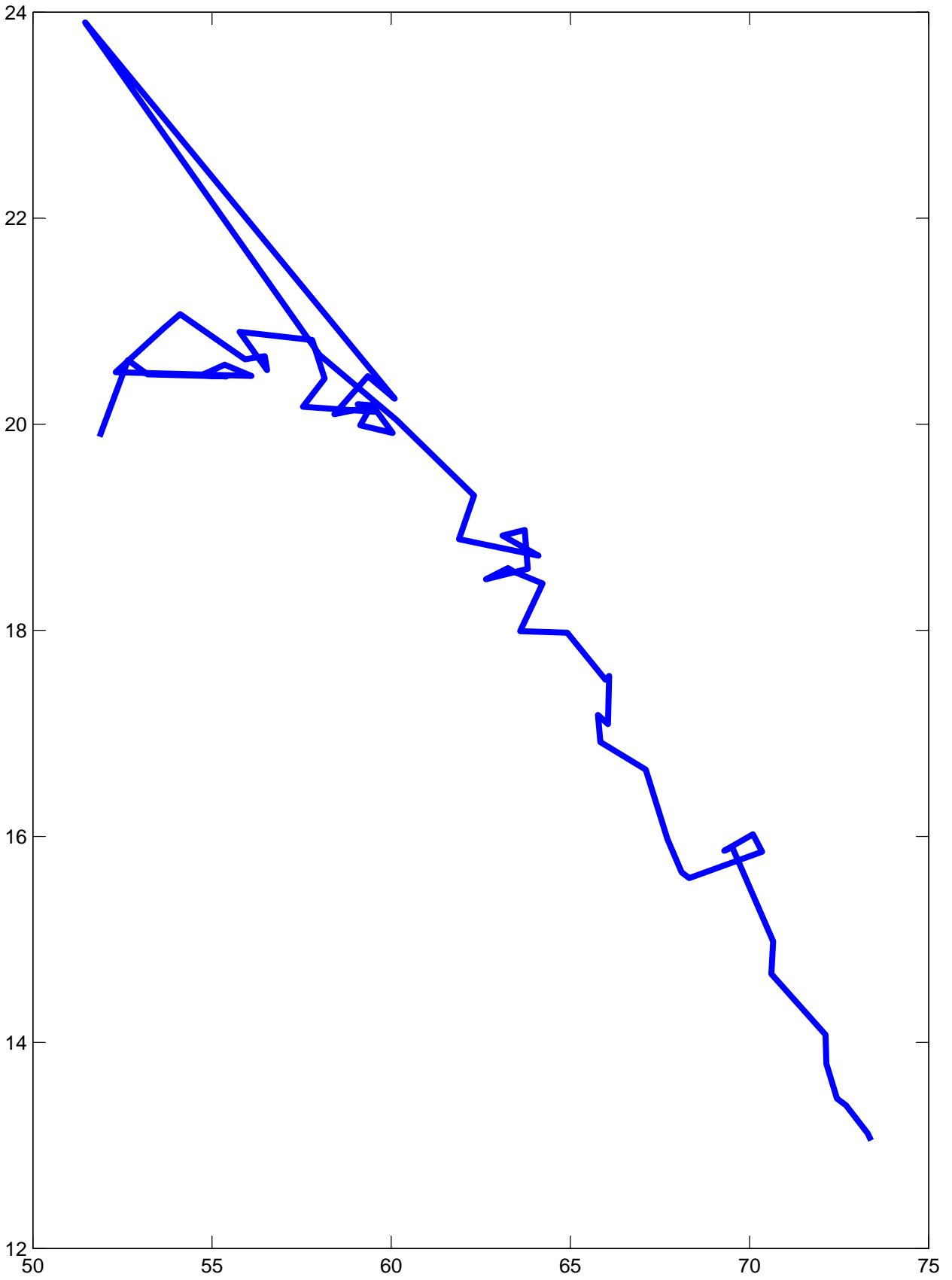
Both Sexes Combined $S(20)$ vs e_0 , 1954–present



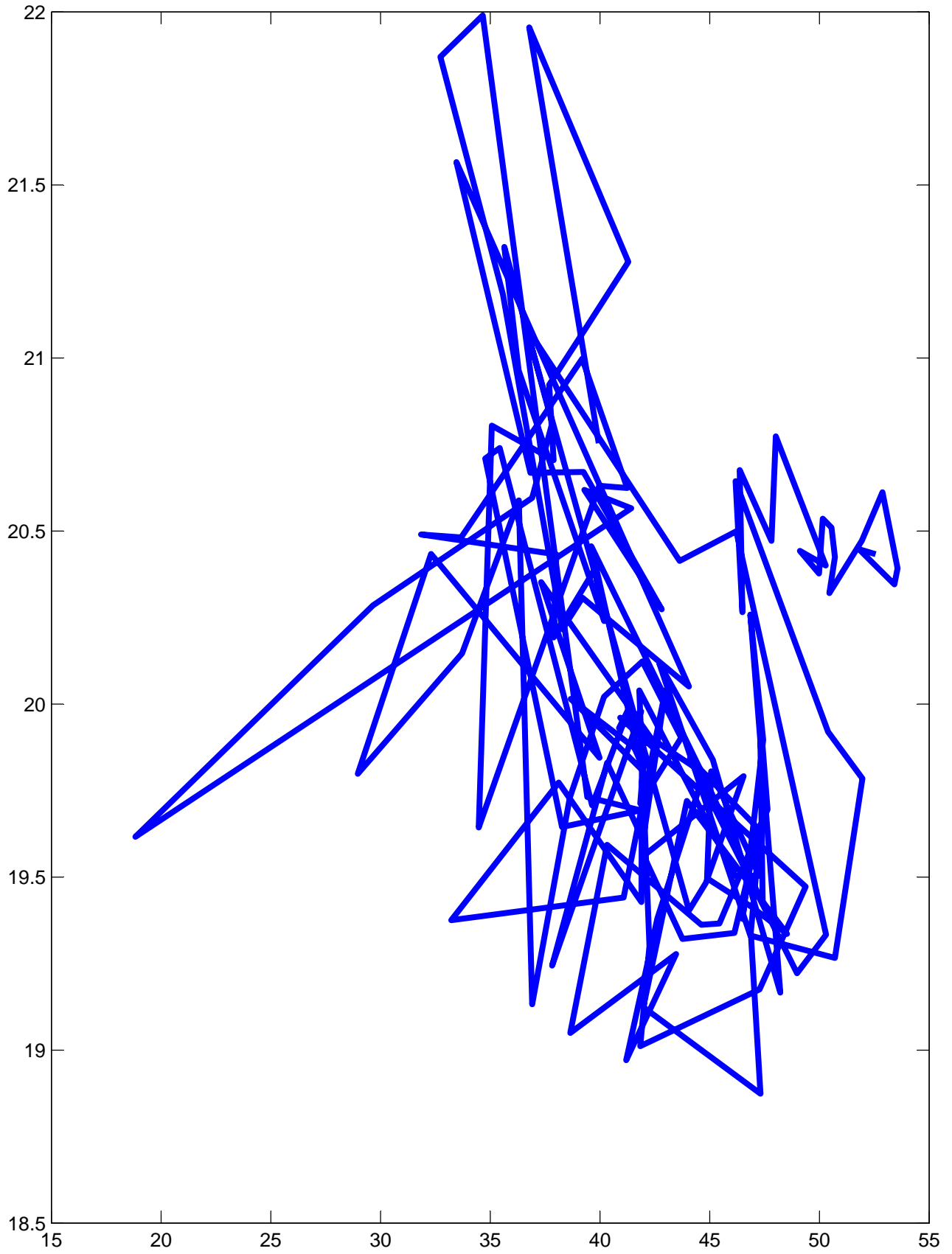
Sweden Female S(20) VS e_0 . 1954-2003

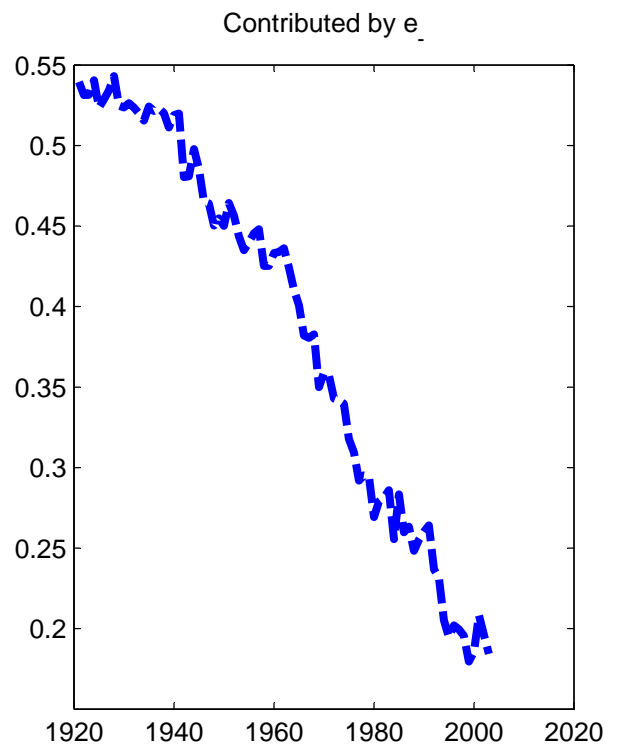
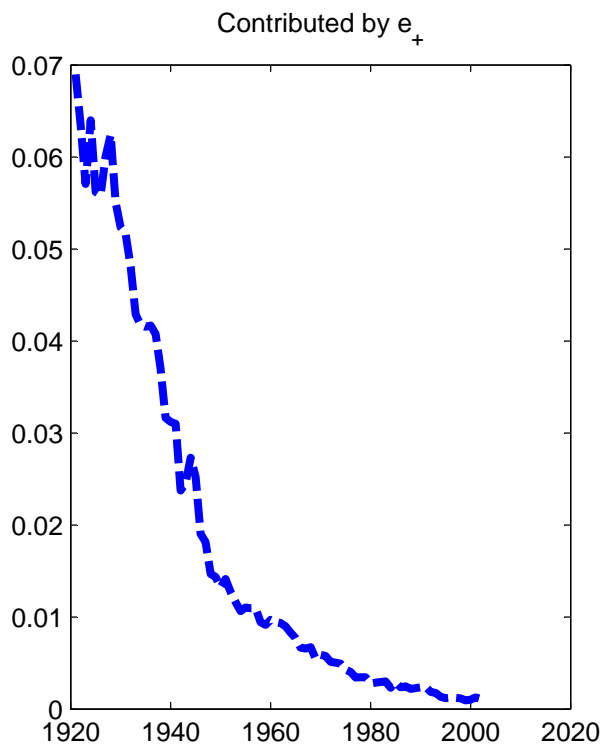
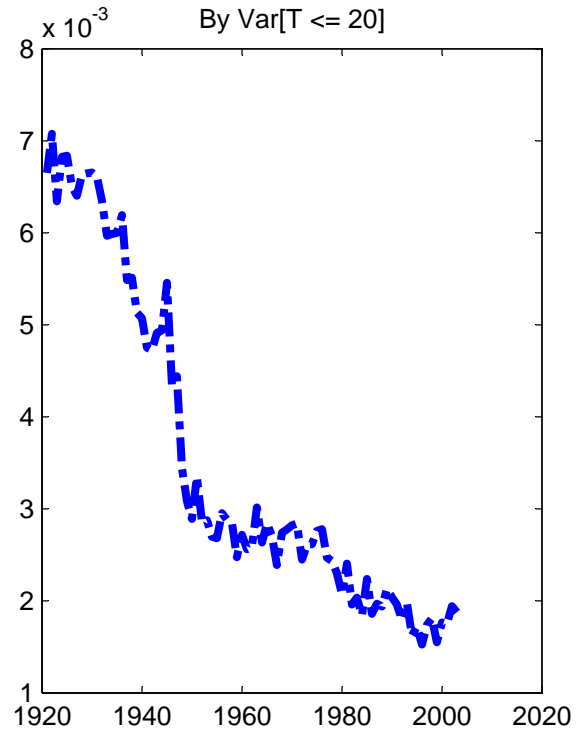
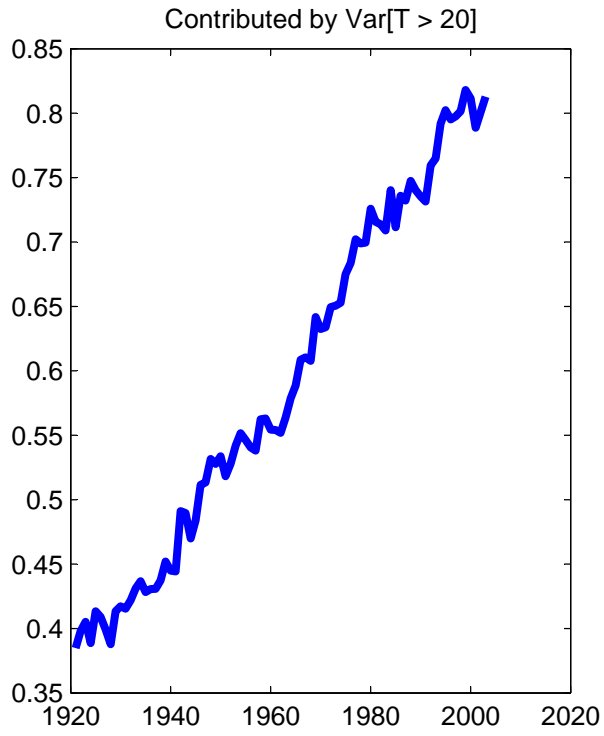


Sweden Female S(20) VS e_0 . 1891-1953

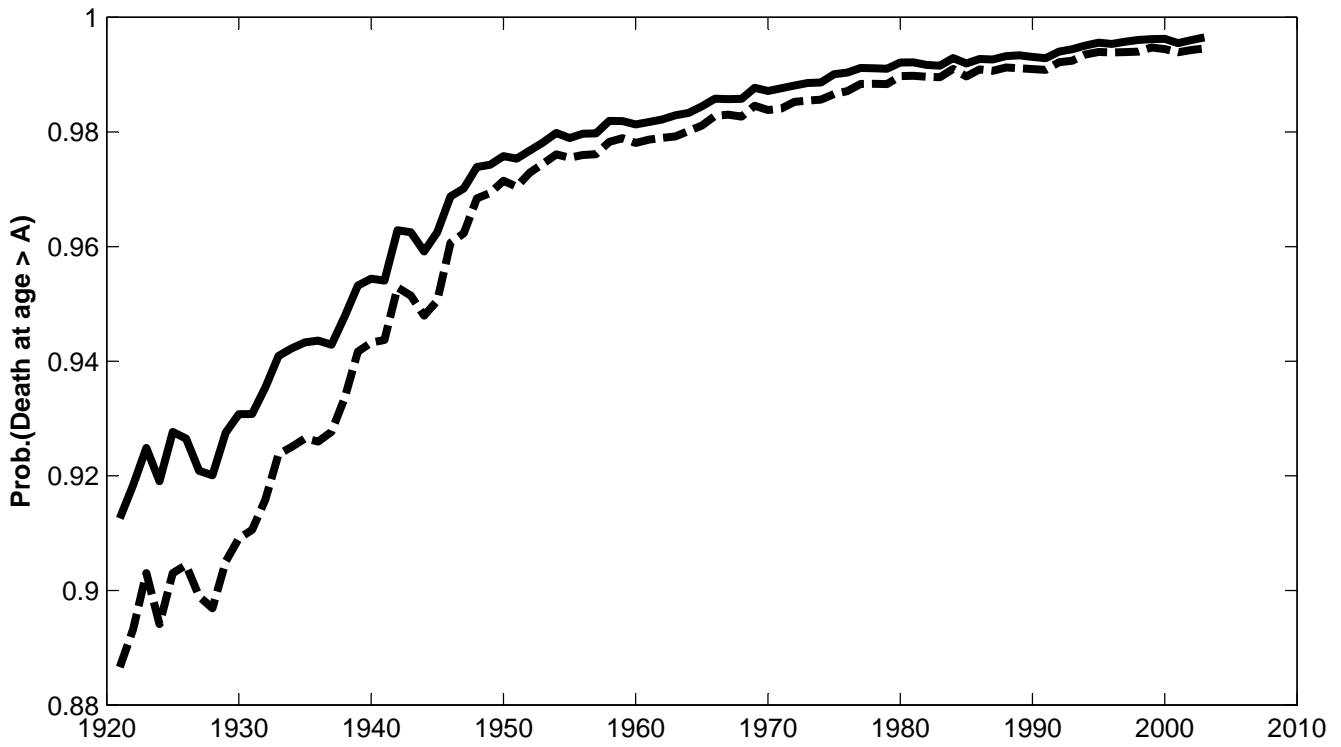
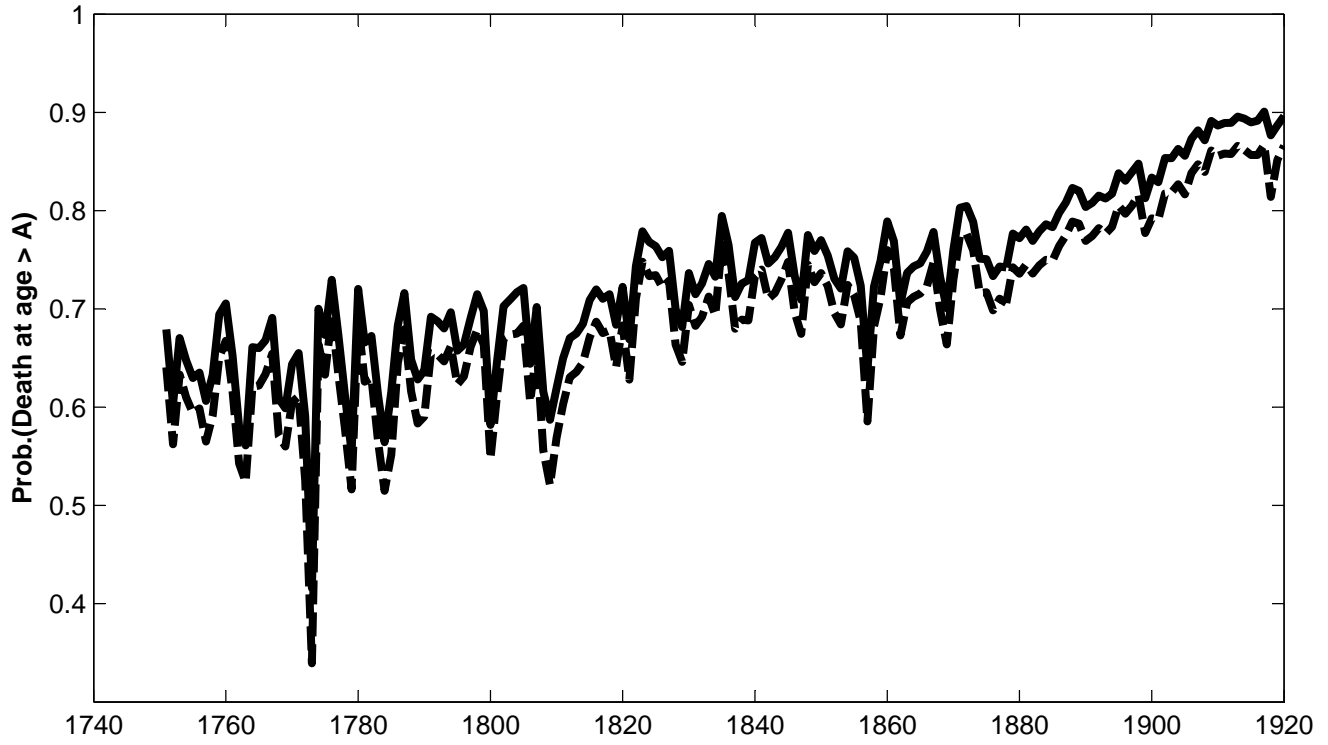


Sweden Female S(20) VS e_0 . 1751-1891





Sweden Deaths at Age >10(solid), >20(dashed)



Both Sexes Combined $S(20)$ vs e_0 . 1954–present

