RECTANGULARIZATION REVISITED: VARIABILITY OF AGE AT DEATH WITHIN HUMAN POPULATIONS*

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Rectangularization of human survival curves is associated with decreasing variability in the distribution of ages at death. This variability, as measured by the interquartile range of life table ages at death, has decreased from about 65 years to 15 years since 1751 in Sweden. Most of this decline occurred between the 1870s and the 1950s. Since then, variability in age at death has been nearly constant in Sweden, Japan, and the United States, defying predictions of a continuing rectangularization. The United States is characterized by a relatively high degree of variability, compared with both Sweden and Japan. We suggest that the historical compression of mortality may have had significant psychological and behavioral impacts.

It is a fact of life that some people die in childhood, some in their adult years, and others in old age. Obviously, age at death is variable in all human populations, although the extent of this variability changes over time and differs across societies for a variety of reasons. Most of the common indices of mortality merely report some form of statistical average. For example, life expectancy at birth is simply the average age at death in a stationary population. Nevertheless, another aspect of mortality worth considering is its *variability* within a population. In this paper we examine the variability of mortality in terms of the distribution of ages at death in the life table.

Several interesting questions are related to this issue of variability. For instance, how has the variability of age at death changed historically? Likewise, how does it differ across populations, especially for populations at comparable levels of mortality? What are *causes* of observed changes and differences in the variability of age at death? What are social and psychological *consequences* of historical changes in this variability? For example, how does variability in age at death affect individuals' perceptions of their own mortality and their long-term life plans? Furthermore, what relationships, if any, exist between the level and the variability of human mortality? Is there some minimum level of variability that can be achieved within a population? If so, are biological limits in the variability of human mortality related to limits in its level? Finally, how should this variability be measured and analyzed?

Although the scope for research on the variability of age at death in humans is potentially quite broad, past work on this subject has been concerned mostly with the *compression-rectangularization hypothesis*, which was proposed in a well-known article by James Fries (1980). Yet the idea that the human survival curve becomes more rectangular with decreasing levels of mortality did not begin with Fries, who himself noted two earlier mentions of the term *rectangularization* (Comfort 1979; Upton 1977). Myers and Manton (1984) trace the idea back even further, to the work of Raymond Pearl ([1923] 1940).

As we demonstrate in this paper, rectangularization of human survival curves is associated with a reduction in the variability of age at death. As deaths become concentrated in an increasingly narrow age range, the slope of the survival curve in that range becomes steeper, and the curve itself begins to appear more rectangular. According to Fries, the rectangularization of survival curves observed in industrialized countries suggests that human life expectancy is approaching its maximum potential value. Fries predicted that this rectangularization would make possible a *compression of morbidity*, such that the period of debilitating disease in late life is minimized.

Since Fries's article appeared in 1980, other investigators have examined his various claims and predictions. Some of these studies suggest that rectangularization is "a myth" (Myers and Manton 1984) or an "ill-posed question" (Manton and Tolley 1991), or even that mortality is now undergoing an expansion, rather than a compression, at the oldest ages (Rothenberg, Lentzner, and Parker 1991). Others agree with Fries that rectangularization has been an important characteristic of historical mortality change in humans, although sometimes they observe that rectangularization has become slower in recent decades (Eakin and Witten 1995; Nusselder and Mackenbach 1996; Wilmoth, Curtsinger, and Horiuchi 1995). Finally, some researchers have challenged Fries's claim about the compression of morbidity (e.g., Schneider and Brody 1983).

To make sense of this discussion, we must recognize several important distinctions. First, the compression of *mortality*, which refers to an increasing concentration of ages at death and thus to a more rectangular survival curve, must not be confused with the compression of *morbidity*, which

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refers to an increasing concentration of illness and disability in the latter years of life (Manton and Singer 1994). Although Fries predicted that the two sorts of compression would occur simultaneously, they are logically separable phenomena. In this paper we consider only the compression of mortality, or rectangularization of the survival curve.

Second, an historical view of the compression of mortality may depend critically on the age range and time frame of the analysis. Variability of age at death may decline in some period when the full age range is considered, but may remain stable if the analysis is limited to older ages (Myers and Manton 1984). Moreover, there is no reason to expect that any process of compression (of morbidity or mortality) should continue indefinitely. Either a compression or an expansion of mortality may occur, depending on the characteristics of mortality change that define an era.

Third and last, the *average* length of life, or life expectancy at birth, is generally independent of its *variability*. Therefore limits to life expectancy and the rectangularity of the survival curve should be considered as separate issues in general. In some special circumstances, however, these two concepts may be interrelated. For example, Fries's argument about an upper limit for human life expectancy, which he thought was around 85 years, relied on a specific premise linking these two concepts. In particular, Fries believed that the maximum human life span is fixed; he claimed that "despite a great change in average life expectancy, there has been no detectable change in the number of people living longer than 100 years or in the maximum age of persons dying in a given year" (Fries 1980:130). If this premise were true, then the distribution of ages at death would be bounded on the right. In such a situation, as mortality declines and death is delayed, the distribution of ages at death *must* be compressed and the survival curve *must* become more rectangular. Thus, although rectangularization does not prove the existence of a fixed upper limit to the human life span, the opposite is a logically valid proposition: If there exists a fixed maximum age beyond which no human can live, rectangularization is a necessary consequence of a sustained decline in mortality.

Although this argument is logically sound, two important empirical findings inform our understanding of these topics. First, some research has revealed that rectangularization, or the compression of mortality, has not continued in recent decades for the elderly population, at least in the United States. Myers and Manton (1984), for example, showed that the standard deviation of deaths above age 60 in the United States *increased* rather than decreased between 1962 and 1979. Rothenberg et al. (1991) confirmed this result in their analysis of ages at death in the United States between 1962 and 1984. A fixed maximum human life span must result in a continued compression of mortality as death rates decline; therefore the failure to observe such a compression suggests either that no limit exists or that it is not currently in sight. Second, and more directly, recent analyses of international data on extremely long-lived individuals have disproved the assertion that the proportion of centenarians in a population has been constant historically or that the maximum age at death is unchanging over time (Vaupel and Jeune 1995; Wilmoth and Lundström 1996).

Taken together, these two sets of findings undermine the empirical basis for arguing that rectangularization is a necessary concomitant of mortality decline. They call into question Fries's broader conclusion about limits that affect the maximum or average human life span.[1] Apart from these specific criticisms, however, it is unfortunate that the notion of rectangularization of the survival curve ever became a central part of the discussion about biological limits to human mortality, because the connections between variability of age at death and limits in mortality level, or life expectancy, are largely indeterminate (Wilmoth 1997).

While most research on the variability of age at death has focused on this issue of biological limits, a number of relevant issues have been neglected. First, the literature shows little agreement about how to measure rectangularity and variability. Fries and other early researchers depended essentially on visual inspection. Since that time, several different measures have been used (Eakin and Witten 1995; Myers and Manton 1984; Nusselder and Mackenbach 1996), but no systematic effort has been made to list and compare the various possible measures.

Second, beyond the debate about whether mortality is becoming more or less compressed at present, it is important to investigate long-term trends in this important historical phenomenon, both to identify periods when rectangularization occurred and when it did not, and to clarify the causes of differences in patterns of change.

Third, previous work did not consider differences across contemporary populations in the variability of age at death. Although it is expected that variability should be low in countries with high life expectancies, populations with comparable mortality levels still may differ notably in the variability of age at death, perhaps as a reflection of differences in social structure and/or disease environments.

Finally, long-term changes in the variability of age at death may have had significant effects on perceptions, attitudes, and behaviors of individuals and, in turn, on society in general. These broad consequences of the historical compression of mortality merit a more fully detailed analysis.

In this article we first present and compare a number of measures of variability or rectangularity. Then, using our preferred measure, the interquartile range (*IQR*) of life table ages at death, we analyze data from Sweden, Japan, and the United States as a means of documenting historical trends and international differences in the variability of age at death. Using a new method of decomposition, we estimate the relative importance of reductions in death rates at various ages for historical changes in the life table *IQR*. Finally, we discuss briefly the possible psychological and behavioral

1. In our usage, the maximum life span is a theoretical concept and refers to the highest age attainable by any member of the population. Average life span is an empirical concept and refers to the average age at death for some (real or hypothetical) cohort. Thus, average life span is equivalent to life expectancy at birth.

effects of the enormous reduction in the variability of age at death that has occurred during the mortality decline of the past 250 years.

EMPIRICAL MEASURES

The notion that human survival curves have become more rectangular over time began as an intuitive concept with no formal definition. The increasing rectangularity of these curves is apparent from a casual inspection of Figure 1, which shows changes in life table parameters for the population of the United States in 1900 and 1995. Three pairs of curves illustrate parallel sets of changes: (1a) Death rates have fallen more sharply (in relative terms) at younger than at older ages; (1b) the distribution of ages at death has shifted to the right and has become less variable and less obviously bimodal; and (1c) the survival curve has become more rectangular as more individuals survive through childhood and young adulthood, but then die off rapidly in old age.

With the scale of change shown in Figure 1, these features of the three pairs of curves are unmistakable and can easily be detected visually. For detailed comparisons, however, we need more precise measures of either the variability of the age distribution of deaths or the rectangularity of the survival curve. Several means of measuring these changes have been proposed, but usually with little discussion of alternatives. We show here that most of the candidate measures are highly correlated. We also discuss some of the fundamental theoretical issues involved in choosing a measure of variability (of age at death) or rectangularity (of the survival curve). Finally, we propose a single measure, the interquartile range (*IQR*), which is favored both for its ease of calculation and for its straightforward interpretation.

Rectangularity Versus Variability

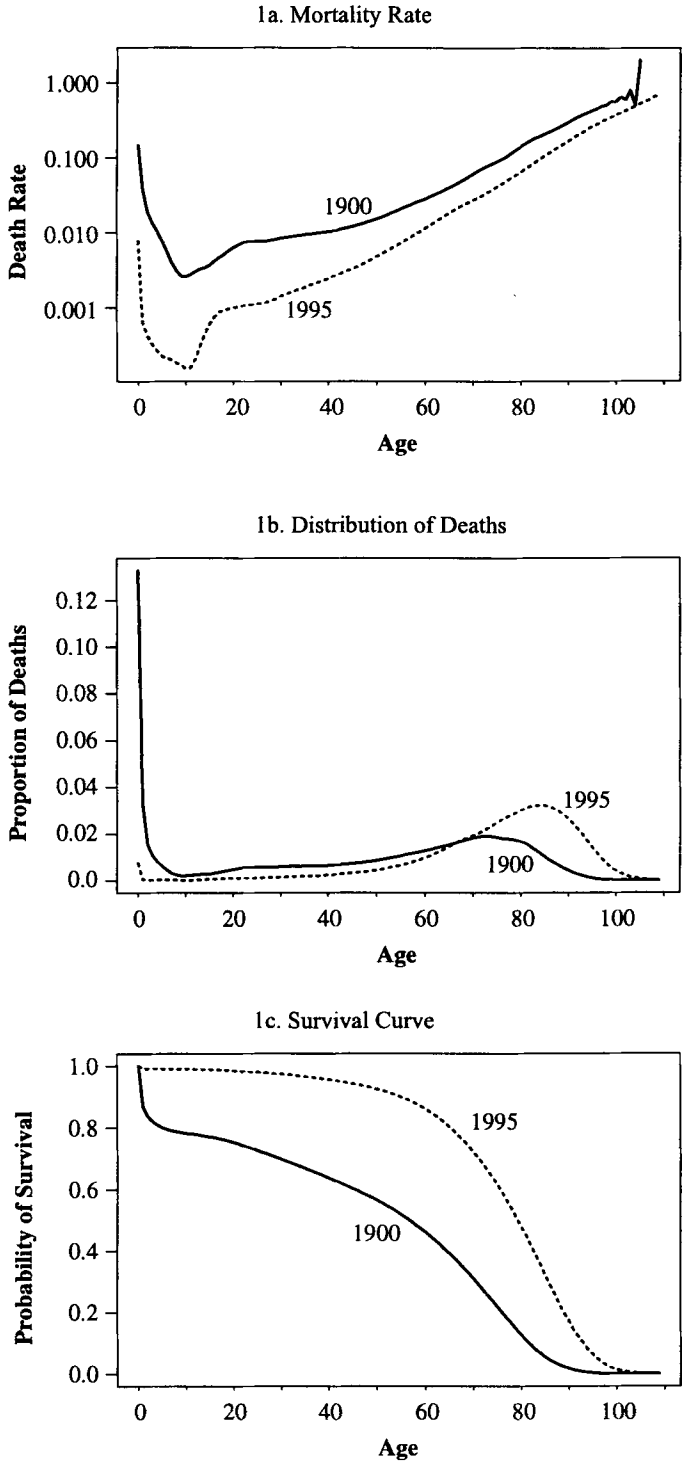
Real human survival curves are never truly rectangular: Such a situation would require that all individuals survive to some maximal age, ω, and then die suddenly at that uniform age. Formally, a truly rectangular survival curve would be written

$$S(x) = \begin{cases} 1 \text{ if } 0 \leq x \leq \omega \\ 0 \text{ if } x > \omega \end{cases}, \quad (1)$$

where $S(x)$ gives the probability of survival (or the expected proportion surviving) to age x.[2] In this situation, there would be no variability in age at death; thus perfect rectangularity is equivalent to zero variability. Although it may be unrealistic to expect that human survival curves could ever be perfectly rectangular, or that age at death could ever be perfectly predictable, it is still possible to define measures of the *degree* of rectangularity exhibited by a survival curve, or the *degree* of variability in the distribution of deaths by age.

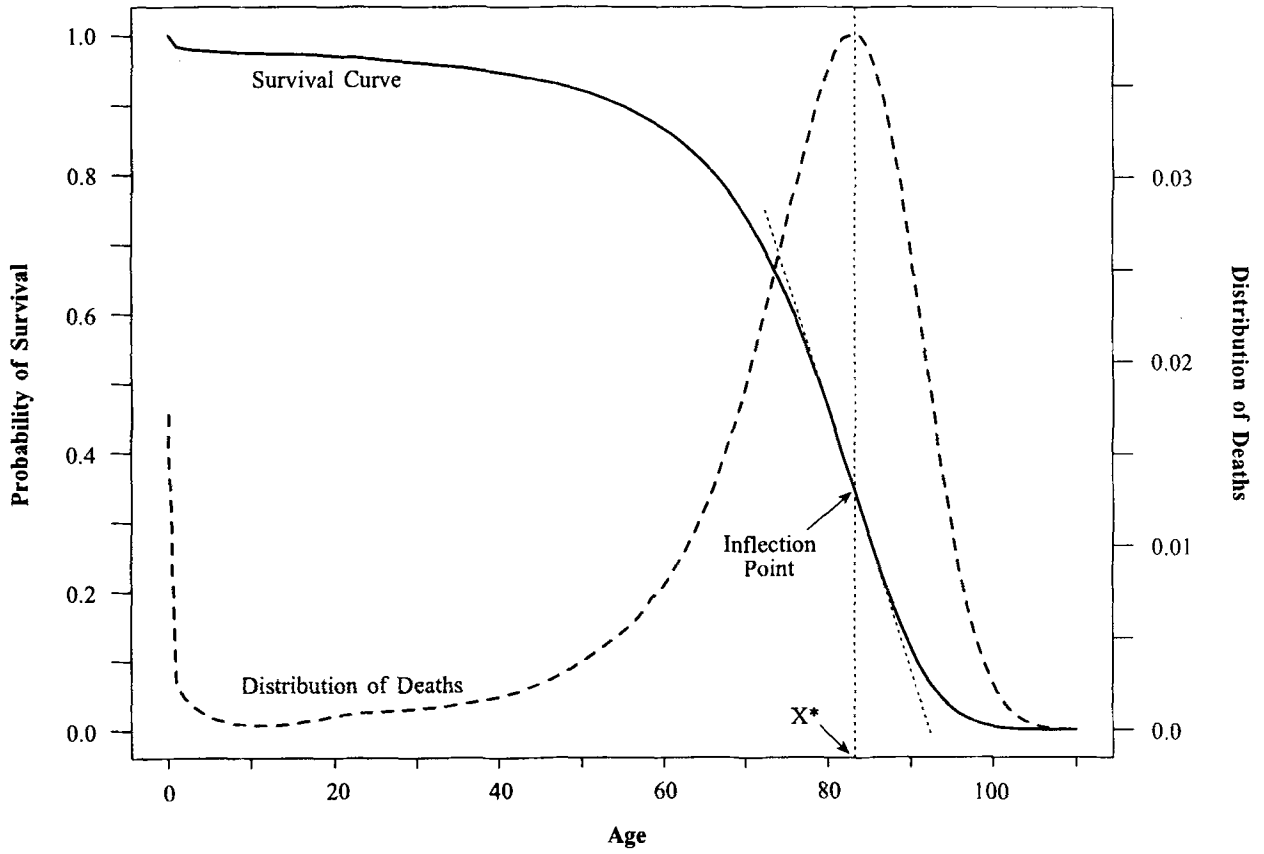
2. Here we employ the standard notation for the statistical analysis of survival data. Let X be a positive random variable, and let $P[X \leq x]$ represent the probability that X takes on a value less than or equal to x. Then $S(x) = P[X > x]$ is the survival curve, $F(x) = 1 - S(x) = P[X \leq x]$ is the cumulative distribution function, $\lambda(x) = -S'(x)/S(x)$ is the hazard function, and $f(x) = S(x)\lambda(x)$ is the probability density function of X.

FIGURE 1. COMPARISON OF THREE LIFE TABLE FUNCTIONS, U.S. WOMEN, 1900 AND 1995



Source: Office of the Chief Actuary, Social Security Administration (Bell et al. 1992; data available through the Berkeley Mortality Database, http://demog.berkeley.edu/wilmoth/mortality).

FIGURE 2. ILLUSTRATION OF FASTEST DECLINE (*FD*) MEASURE OF THE RECTANGULARITY OF SURVIVAL CURVES



A simple measure of rectangularity is the maximum downward slope of $S(x)$ in the adult age range. This measure is illustrated in Figure 2. Increasing rectangularity according to this measure implies increasing verticality in the survival curve at older ages. In formulas, the slope of $S(x)$ at the point of *fastest decline* is

$$\begin{aligned} FD &\equiv \max_x\{-S'(x)\} \\ &= \max_x\{S(x)\lambda(x)\} \\ &= \max_x\{f(x)\} \end{aligned} \qquad (2)$$

for $x > 15$ only. As this formula indicates, the downward slope of $S(x)$ at its point of fastest decline equals the height of the density function at its modal value. This relationship is also depicted in Figure 2.[3]

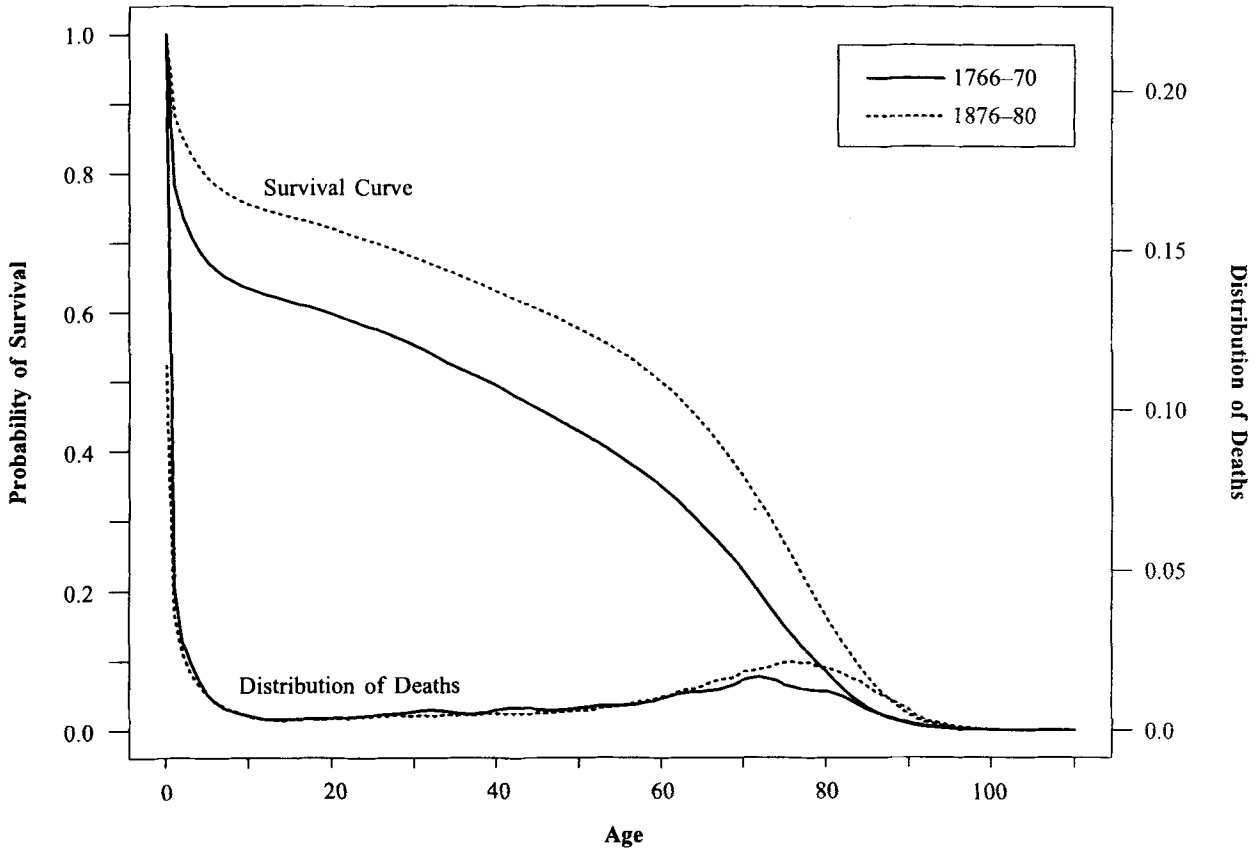
Intuitively it appears that measures of rectangularity and variability should be related inversely: As the distribution of age at death becomes less variable, the survival curve should become increasingly rectangular. For example, what is the relationship between *FD* and some measure of variability—say, the standard deviation, σ (defined formally in Appendix A)? Under certain conditions, it is possible to show that the expected inverse relationship holds true. For example, suppose that a random variable is part of a family of random variables that differ only by a linear transformation. Thus, define a standardized random variable, X_S, such that $X_S = (X - \mu)/\sigma$, where μ and σ respectively are the mean and the standard deviation of X. Suppose further that the distributions of X and X_S are symmetric. Obviously these assumptions are unrealistic for human mortality curves in general, but they are a reasonable approximation in situations of low mortality, when the distribution of age at death resembles a normal distribution (admittedly, however, with a long left tail).

In this restricted case of a family of symmetric curves linked by linear transformations, the inverse relationship between *FD* and σ has a very simple form. Let x^* be the mode of X. Thus $FD = f(x^*)$. Since the distribution of X is symmetric, we know that $x^* = \mu$. Therefore

$$FD = f(x^*) = \frac{1}{\sigma} f_s\left(\frac{x^* - \mu}{\sigma}\right) = \frac{1}{\sigma} f_s(0) = \frac{1}{\sigma} FD_s. \qquad (3)$$

3. In situations of very high mortality, the modal age at death is sometimes below 15 years. Thus, by restricting this calculation to ages greater than 15, we ensure that the *FD* measure refers to the degree of rectangularity in the survival curve at *older* ages.

FIGURE 3. COMPARISON OF TWO LIFE TABLE FUNCTIONS, SWEDISH WOMEN, 1766–1770 AND 1876–1880



Source: Swedish life tables reconstructed using data from various sources (Berkeley Mortality Database, http://demog.berkeley.edu/wilmoth/mortality).

Thus the maximum downward slope of the survival curve (or the height of the density function at the mode) equals that same value for the standard curve divided by σ. In this case, then, the correlation between *FD* and σ is always negative. Empirically this inverse relationship does not hold in general; as we show below, however, it usually holds at least approximately. The major exceptions we have noted involve severe violations of the assumption of symmetry, such as in situations of very high infant mortality. For example, Figure 3 shows survival curves and distributions of ages at death for Swedish women in 1766–1770 and 1876–1880. Although standard deviations are identical (in each period, σ was 32.8 years), the survival curve in the latter period is noticeably more vertical (*FD* increased from 0.017 to 0.021).

High Correlation

Theoretical results describing the relationships among various measures of rectangularity and variability are usually difficult (and sometimes seemingly impossible) to derive analytically. Therefore we have compared 10 such measures empirically, using historical mortality data for three national populations (Sweden, Japan, and the United States). These measures are based on various intuitive notions of rectangularity or variability; many have appeared previously in the literature. They are described fully in Appendix A, but here we wish merely to document the high degree of correlation among the 10 measures, as illustrated in Table 1.

Briefly, the 10 measures are as follows:

1. Fixed rectangle. In a standard plot of a survival curve, imagine a rectangle with a height of 1 and a right boundary at (say) age 100. The measure equals the proportion of this rectangle that lies below the survival curve. As the curve becomes more rectangular, this quantity increases.

2. Moving rectangle. Suppose that the right boundary in this rectangle changes, depending on survival probabilities at the oldest ages. Suppose, for example, that the right boundary always equals the age at which only 1/1,000 of the original cohort is still alive. Again, the measure equals the proportion of the rectangle that lies below the survival curve.

3. Fastest decline. This measure equals the (negative) slope of the survival curve at its point of fastest decline in the adult age range. As the survival curve becomes more rectangular, this value increases.

TABLE 1. CORRELATION COEFFICIENTS FOR 10 MEASURES OF RECTANGULARITY OR VARIABILITY, BASED ON 154 PERIOD LIFE TABLES FOR SWEDEN, JAPAN, AND THE UNITED STATES

	FR	*MR*	*FD*	*SC*	*QP*	*PI*	*SD*	*IQR*	*G*	*H*
Fixed Rectangle	1.000									
Moving Rectangle	0.997	1.000								
Fastest Decline	0.975	0.974	1.000							
Sharpest Corner	0.837	0.828	0.922	1.000						
Quickest Plateau	0.923	0.922	0.972	0.927	1.000					
Prolate Index	0.967	0.967	0.990	0.912	0.963	1.000				
Standard Deviation	−0.926	−0.918	−0.923	−0.835	−0.868	−0.906	1.000			
Interquartile Range	−0.956	−0.959	−0.926	−0.785	−0.856	−0.919	0.936	1.000		
Gini Coefficient	−0.993	−0.997	−0.959	−0.797	−0.895	−0.951	0.918	0.967	1.000	
Keyfitz's *H*	−0.987	−0.994	−0.948	−0.775	−0.886	−0.942	0.883	0.948	0.996	1.000

Sources: U.S. life tables: Office of the Chief Actuary, Social Security Administration (Bell et al. 1992). Swedish and Japanese life tables: Berkeley Mortality Database, http://demog.berkeley.edu/wilmoth/mortality.

Notes: The 154 life tables were divided evenly between male and female tables and consisted of 98 tables for Sweden (for periods 1751–1755 through 1991–1995), 18 for Japan (for periods 1951–1955 through 1991–1995), and 38 for the United States (1901–1905 through 1991–1995). The 10 measures of rectangularity or variability are defined in Appendix A.

4. Sharpest corner. This measure equals the (negative) second derivative of the survival curve at the point where it turns downward most quickly in the adult age range. As the survival curve becomes more rectangular, this value increases.

5. Quickest plateau. This measures equals the second derivative of the survival curve at the point where it levels off most quickly at very high ages. As the survival curve becomes more rectangular, this value increases.

6. Prolate index. This measure, proposed by Eakin and Witten (1995), is a sophisticated means of measuring the steepness of the slope of the survival curve at older ages. (See Appendix A for a precise definition.)

7. Interquartile range. This measure equals the distance between the lower and the upper quartiles of the distribution of ages at death in a life table. As age at death becomes less variable, this measure decreases.

8. Standard deviation. This measure equals the standard deviation of the distribution of ages at death in a life table. As age at death becomes less variable, this measure decreases.

9. Gini coefficient. This measure reflects the degree of inequality in age at death in a life table population. Its value decreases as age at death becomes less variable.

10. Keyfitz's *H*. This quantity was developed to approximate the dynamic relationship between the force of mortality by age and life expectancy at birth. It also expresses the "degree of concavity" in the survivorship curve (thus the opposite of rectangularity) and the increasing concentration of deaths at older ages.[4] As deaths become more concentrated, the value of Keyfitz's *H* declines.

The first six of these measures reflect different conceptualizations of the rectangularity of survival curves (fixed rectangle, moving rectangle, fastest decline, sharpest corner, quickest plateau, and prolate index). The next three measures reflect variability in the distribution of ages at death (standard deviation, interquartile range, and Gini's coefficient of dispersion). The last measure, Keyfitz's *H*, is the most difficult to characterize, but because *H* tends to decline with increasing levels of life expectancy, we classify it here, for convenience, among the measures of variability.

Thus we have six measures of rectangularity and four measures of variability, reflecting different notions of rectangularization or mortality compression. Table 1 documents the presence of strong positive correlations within each of these two groups, and strong negative correlations across groups. The weakest correlations are generally those involving the sharpest corner (*SC*) and quickest plateau (*QP*) measures. With only one exception, the absolute values of all correlations not involving this pair of measures are above 0.9. Therefore it appears that one can choose a single measure from among the eight remaining measures on the basis of convenience and ease of interpretation. Below we argue in favor of using the interquartile range (*IQR*) for this purpose.

A Single Measure

The interquartile range (*IQR*) has a twofold appeal as a single measure of variability in the life table. First, it is very simple to calculate because it equals the difference between the ages where the survival curve, $S(x)$, crosses 0.25 and 0.75. Second, being the length of the span of ages containing the middle 50% of deaths, it possesses a simple interpretation. On the basis of the correlations shown in Table 1, the *IQR* would not be our first choice. Nevertheless, its convenience and its clear meaning make it an optimal measure for the kinds of analyses we wish to pursue.

The most important argument in favor of the *IQR* is that it is one of only two measures considered here which are ex-

4. Some authors refer to *H* as a measure of "entropy" in the life table (e.g., Demetrius 1976).

pressed in units of years of age. The standard deviation (*SD*) shares this advantage, but it is more difficult to compute and is less strongly correlated with almost all of the other measures listed in Table 1 than is the *IQR*.

Other Methodological Issues

In addition to selecting a single measure, we must choose between at least four ways of applying that measure to real data. The first choice concerns the relevant age range for a particular analysis. A second possible modification involves standardizing the age scale. Figure 4 shows trends in the *IQR* for the Swedish population during 1751–1995 based on different combinations of the age range and the age scale. The pros and cons of these choices are discussed below.

Left-censoring. For some purposes it may seem appropriate to consider only the adult age range (which could be defined as age 15 and above, as 30 and above, or by some other formula). In discussions about the rectangularization of the survival curve in relation to the limits of human longevity, some authors have argued that at least childhood mortality should be ignored, because it is not the result of senescent processes (Myers and Manton 1984). For most other purposes, however, such as studies of the psychology of death or social inequality in the face of death, we see little advantage in restricting the age range used in the analysis.

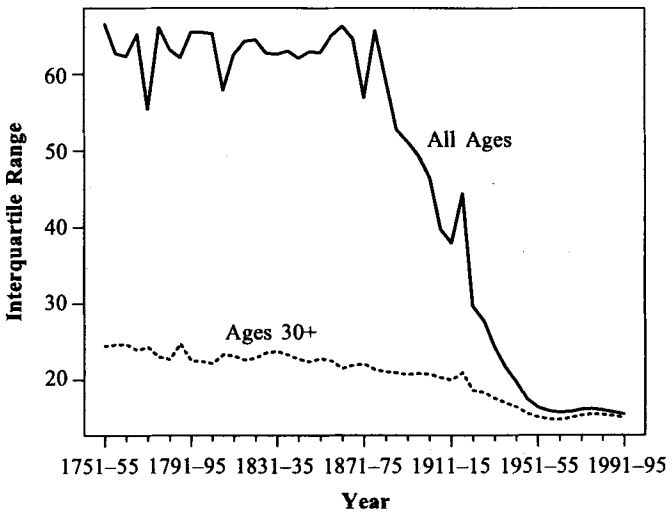
Historically the life table *IQR* for the entire age range has declined greatly, as seen in Figure 4a for Sweden. The same graph depicts a much smaller drop in the *IQR* for ages 30 and above. Most of the decline in the *IQR* over all ages was highly concentrated during a 75-year period from the late 1870s to the early 1950s, during an era of rapid reductions in infectious disease. In comparison, the decline in the *IQR* for ages 30 and above was smaller (in absolute or relative terms) and somewhat more gradual. The convergence of the two trends in recent decades reflects the near-elimination of deaths below age 30. Regardless of the choice of age range, however, the life table *IQR* ceased to decline or to change significantly after the early 1950s in Sweden.

Standardized age. There is a strong argument for using a standardized age variable when comparing variability or rectangularity across species, because the raw time scales would not be comparable (Eakin and Witten 1995). Similarly, it may be appropriate to use a standardized age in historical studies of humans, given the large increase in life expectancy, so that variability in age at death is expressed in relation to the average. The method of standardization used here is simply to divide the original ages at death by life expectancy (within the age range considered), and then to compute a measure of variability, such as the *IQR*. If the *IQR* based on original ages at death is roughly constant (for example, during more than a century after 1750 in Sweden), the *IQR* based on standardized ages decreases merely as a function of increasing life expectancy (see Figure 4b). Thus the effect of standardization is to combine changes in absolute variability with changes in life expectancy; as a result, it is more difficult to separate these two effects and to interpret observed changes. For this reason we do not favor the use of standardized ages in historical studies of the variability of mortality in humans.

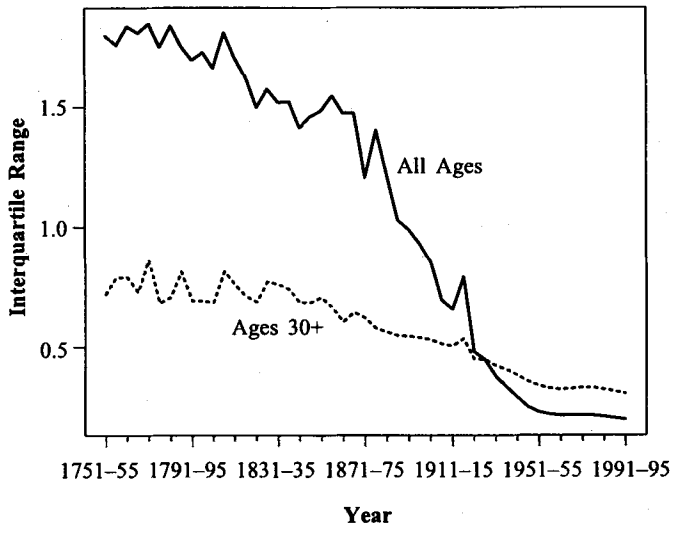
In light of these various considerations, we have chosen to measure variability in the age at death using the inter-

FIGURE 4. COMPARISON OF FOUR METHODS FOR COMPUTING THE INTERQUARTILE RANGE (*IQR*) OF AGES AT DEATH, SWEDEN, SEXES COMBINED, 1751–1995

4a. Original Age, All Ages Versus Ages 30+



4b. Standardized Age, All Ages Versus Ages 30+



Source: Swedish life tables reconstructed using data from various sources (Berkeley Mortality Database, http://demog.berkeley.edu/wilmoth/mortality).

Note: Standardized age equals original age divided by life expectancy (from birth or age 30).

quartile range (*IQR*) applied over all ages, without standardizing based on some average measure of longevity. This measure is employed for all subsequent graphs and calculations in this paper. We turn now to the results that emerge from an analysis of variability in ages at death based on the *IQR*.

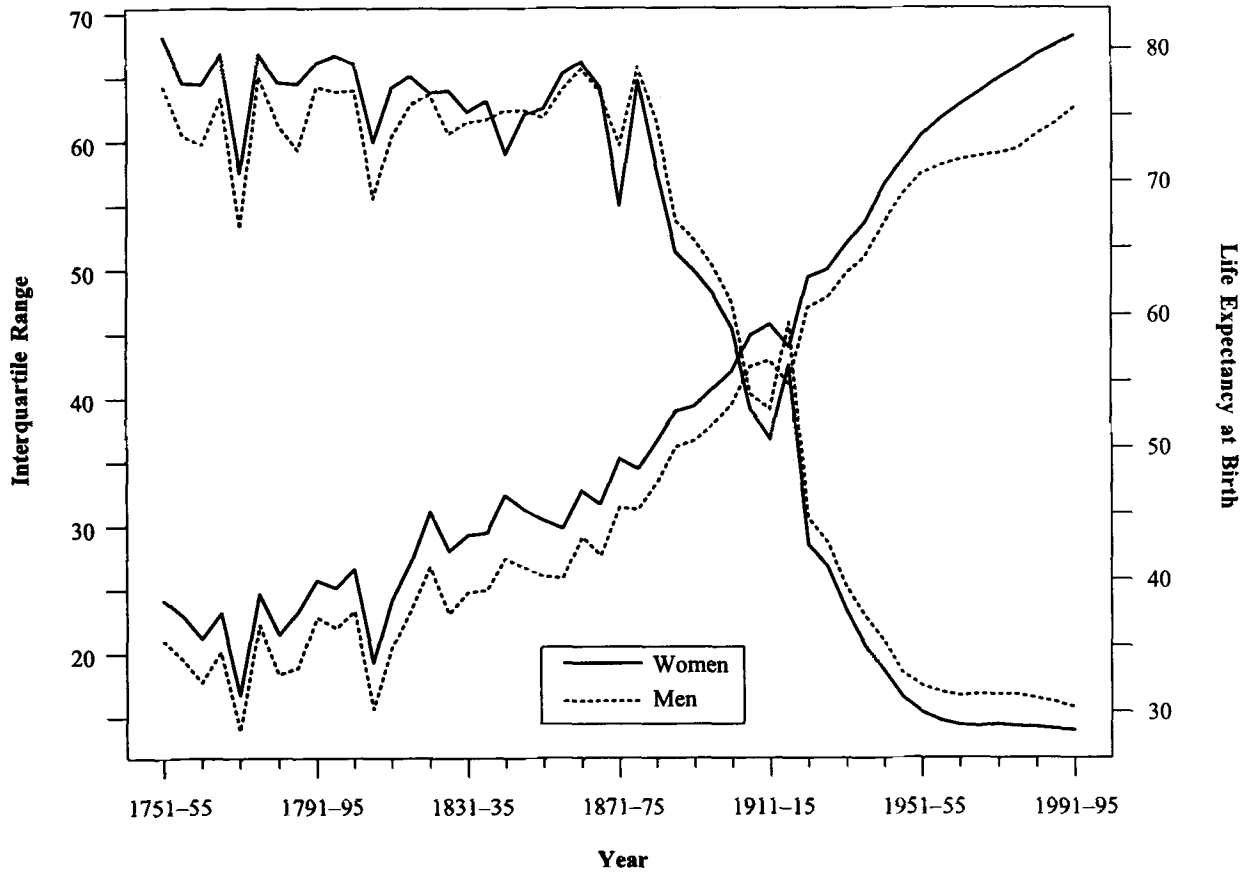
VARIABILITY OF AGE AT DEATH IN HISTORICAL PERSPECTIVE

Death has always been certain, but certainty regarding the timing of death has varied widely in historical perspective. Based on data for Sweden, Figure 5 illustrates the enormous change that has taken place in the variability of death across the age range during the industrial era. As life expectancy rose from the mid-thirties to the high seventies, the life table *IQR* declined from the mid-sixties to around 15. Thus, at the dawn of the industrial era, a life expectancy of about 35 years masked an enormous variability in ages at death because the middle half of deaths was spread over ages from less than 5 years to almost 70 years. Life was not only short in this period; in addition, its timing was highly uncertain and indeed bimodal.

By contrast, modern mortality regimes are characterized by a high average age at death with significantly less variability than in the past. The distribution of ages at death is still bimodal, but the mode at younger ages has become less and less important. Today in economically developed countries, the middle half of the distribution of deaths by age is concentrated in a range of about 15 years, usually somewhere between ages 65 and 90. This increasing certainty in the timing of death must have had an enormous effect on the character of individual lives; later in this paper we offer some rather speculative comments about the historical significance of this change.

First, however, to analyze the mechanism by which this reduction in variability has been achieved, we decompose the

FIGURE 5. INTERQUARTILE RANGE (*IQR*) OF LIFE TABLE AGES AT DEATH, AND LIFE EXPECTANCY AT BIRTH ($\mathring{e}_0$), SWEDISH WOMEN AND MEN, 1751–1995



Source: Swedish life tables reconstructed using data from various sources (Berkeley Mortality Database, http://demog.berkeley.edu/wilmoth/mortality).

TABLE 2. LIFE EXPECTANCY AT BIRTH ($\dot{e}_0$), INFANT MORTALITY RATE (q_0), AND INTERQUARTILE RANGE (*IQR*) OF LIFE TABLE AGES AT DEATH FOR SWEDEN, JAPAN, AND THE UNITED STATES, SEXES COMBINED, 1861–1995

	Sweden			Japan			United States		
	$\dot{e}_0$	q_0	*IQR*	$\dot{e}_0$	q_0	*IQR*	$\dot{e}_0$	q_0	*IQR*
1861–1865	45.0	0.136	66.3	—	—	—	—	—	—
1871–1875	47.3	0.132	57.0	—	—	—	—	—	—
1881–1885	48.9	0.114	59.4	—	—	—	—	—	—
1891–1895	51.8	0.102	51.2	—	—	—	—	—	—
1901–1905	54.5	0.090	46.4	—	—	—	50.1	0.114	46.9
1911–1915	57.9	0.072	37.9	—	—	—	54.2	0.092	38.4
1921–1925	61.7	0.061	29.6	—	—	—	58.2	0.066	31.2
1931–1935	64.2	0.050	24.3	—	—	—	61.0	0.054	26.9
1941–1945	68.3	0.030	19.8	—	—	—	65.0	0.041	23.2
1951–1955	72.0	0.019	16.4	63.4	0.047	23.0	69.1	0.028	20.6
1961–1965	73.6	0.015	15.7	69.4	0.024	17.5	70.2	0.025	20.3
1971–1975	74.8	0.010	16.1	73.5	0.011	15.9	71.8	0.017	20.4
1981–1985	76.5	0.007	16.0	77.2	0.006	15.1	74.5	0.011	19.4
1991–1995	78.3	0.005	15.5	79.5	0.004	15.2	75.6	0.008	19.1

Sources: U.S. life tables: Office of the Chief Actuary, Social Security Administration (Bell et al. 1992). Swedish and Japanese life tables: Berkeley Mortality Database, http://demog.berkeley.edu/wilmoth/mortality.

change in the *IQR* as a function of mortality decline at different ages and consider the relationship between changing variability in age at death and the age pattern of mortality decline. We also examine similar changes from the perspective of birth cohorts, and document differences in the variability of age at death across national populations.

Contribution of Mortality Reductions at Different Ages

One might expect the reduction in the variability of ages at death to be due almost entirely to the decline of infant and child mortality. Indeed, our analysis demonstrates that decreasing mortality among the very young is responsible for much of the reduction in variability. To support this claim, we developed a technique for decomposing the change in the *IQR* into components attributable to the reduction in mortality rates at different ages. (The details of this method are described in Appendix B.)[5] For Sweden, the decomposition analysis begins with 1861–1865, or slightly before the start of the sharp decline in the *IQR* during the late 1870s. Table 2 shows actual values of life expectancy at birth, the infant mortality rate, and the *IQR* for Sweden (1861–1995), Japan (1951–1995), and the United States (1901–1995). The results of the *IQR* decomposition for these three countries are displayed in Tables 3–5. The total change in the *IQR* predicted by the decomposition (Tables 3–5, bottom row) matches closely the actual differences implied by the values listed in Table 2.

In Tables 3–5, the decomposition of the change in the *IQR* is presented both in absolute years (left-hand side of each table) and as a percentage of the total change over the full period for each country (right-hand side). A negative contribution (in absolute terms) indicates that the mortality reduction in that age range raised the lower quartile of the distribution more than it raised the upper quartile, thus contributing to a reduction in the *IQR*. Positive values, on the other hand, are associated with mortality reductions in an age range that is above the lower quartile but below the upper quartile. A mortality reduction in this age range raises the upper quartile without affecting the lower one and thus contributes to a lengthening of the *IQR*. Mortality reduction above the upper quartile does not affect the IQR and thus yields zeros in the decomposition. Overall the increase in the lower quartile is usually larger than the increase in the upper quartile; this results in a compression of the distribution of ages at death and thus a reduction in the *IQR*.

The change in Swedish mortality during 1861–1995 was enormous by any measure: Life expectancy increased by more than 30 years, infant mortality plummeted from more than 13% to less than 1%, and the *IQR* decreased by about 50 years (Table 2). About two-thirds of the reduction in the *IQR* during this period can be attributed to a decrease in mortality rates under age 5, although the relative contribution of mortality change in the adult age range became more and more important over time (Table 3). The importance of trends in infant and child mortality is enhanced by the fact that the

5. This method can be used to decompose changes in the median age at death and, more generally, changes in the pth percentile of the distribution of deaths by age (i.e., x_p, where $S(x_p) = 1 - p/100$). In this application we use it to decompose changes in the difference between the 75th and the 25th percentiles (i.e., the *IQR*). The method can easily be extended to decompose percentiles of ages at death, or the *IQR*, into components attributable to changes in cause-specific mortality, because all-cause death rates are simply the sum of cause-specific rates.

TABLE 3. DECOMPOSITION OF CHANGE IN LIFE TABLE *IQR* FOR SWEDEN, SEXES COMBINED, 1861–1995

	Predicted Change (in Years)					Percentage of Total Change				
Age	1861–65 to 1891–95	1891–95 to 1921–25	1921–25 to 1951–55	1951–55 to 1991–95	1861–65 to 1991–95	1861–65 to 1891–95	1891–95 to 1921–25	1921–25 to 1951–55	1951–55 to 1991–95	1861–65 to 1991–95
0	–6.5	–6.6	–2.8	–0.5	–16.4	12.8	12.9	5.4	0.9	32.1
1–4	–8.8	–7.1	–1.3	–0.1	–17.3	17.2	14.0	2.5	0.2	33.9
5–24	–2.6	–6.2	–3.1	–0.3	–12.1	5.1	12.2	6.0	0.5	23.8
25–49	0.4	–3.7	–5.1	–0.6	–9.0	–0.8	7.3	10.0	1.2	17.7
50–74	2.6	1.3	–1.4	–2.6	–0.2	–5.0	–2.5	2.8	5.0	0.3
75–89	0.1	0.5	0.3	3.1	4.0	–0.1	–1.0	–0.6	–6.0	–7.7
90+	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Total	–14.9	–21.9	–13.3	–1.0	–51.0	29.1	42.7	26.0	1.9	100.0

TABLE 4. DECOMPOSITION OF CHANGE IN LIFE TABLE *IQR* FOR JAPAN, SEXES COMBINED, 1951–1995

	Predicted Change (in Years)					Percentage of Total Change				
Age	1861–65 to 1891–95	1891–95 to 1921–25	1921–25 to 1951–55	1951–55 to 1991–95	1861–65 to 1991–95	1861–65 to 1891–95	1891–95 to 1921–25	1921–25 to 1951–55	1951–55 to 1991–95	1861–65 to 1991–95
0	–1.2	–0.4	–0.2	–0.1	–1.8	15.1	5.6	2.0	0.8	23.5
1–4	–0.9	–0.1	0.0	0.0	–1.0	11.2	1.3	0.6	0.2	13.3
5–24	–0.8	–0.2	–0.1	0.0	–1.1	10.4	2.3	1.5	0.5	14.7
25–49	–2.0	–0.6	–0.4	–0.2	–3.3	26.2	7.9	5.6	3.0	42.7
50–74	–0.7	–1.4	–1.9	–1.2	–5.2	9.5	18.3	23.9	15.3	67.1
75–89	0.1	1.1	1.8	1.7	4.8	–1.8	–14.7	–23.2	–21.6	–61.3
90+	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Total	–5.5	–1.6	–0.8	0.1	–7.8	70.7	20.7	10.5	–1.8	100.0

TABLE 5. DECOMPOSITION OF CHANGE IN LIFE TABLE *IQR* FOR THE UNITED STATES, SEXES COMBINED, 1901–1995

	Predicted Change (in Years)					Percentage of Total Change				
Age	1861–65 to 1891–95	1891–95 to 1921–25	1921–25 to 1951–55	1951–55 to 1991–95	1861–65 to 1991–95	1861–65 to 1891–95	1891–95 to 1921–25	1921–25 to 1951–55	1951–55 to 1991–95	1861–65 to 1991–95
0	–7.3	–1.5	–0.4	–0.3	–9.6	25.9	5.3	1.5	1.2	33.9
1–4	–5.0	–0.8	–0.1	0.0	–5.9	17.7	2.8	0.3	0.2	21.0
5–24	–4.7	–1.4	–0.1	–0.2	–6.3	16.6	5.1	0.2	0.6	22.5
25–49	–4.7	–4.0	–0.4	–0.6	–9.6	16.7	14.0	1.3	2.0	34.1
50–74	1.4	0.2	–0.4	–1.7	–0.6	–4.9	–0.6	1.5	6.1	2.1
75–89	0.1	1.1	1.2	1.5	3.8	–0.2	–4.0	–4.1	–5.2	–13.5
90+	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Total	–20.3	–6.4	–0.2	–1.4	–28.2	71.8	22.6	0.7	4.9	100.0

distribution of ages at death is bimodal. With the near-elimination of the mode at younger ages, the distribution of ages at death shifted upward very rapidly and became compressed in the region of the mode at older ages.

By various measures, the age pattern of mortality reduction in Sweden during approximately the first two-thirds of this period strongly favored younger age groups. In the last 40 to 50 years, however, an "aging of mortality decline" has

occurred, characterized by successively larger reductions in mortality rates at older ages, and by smaller reductions at younger ages (Horiuchi and Wilmoth 1995). As death rates have fallen more steeply at older ages, the older age range has made an increasing contribution to changes in the *IQR*. In the most recent period, however, the positive contribution from older ages (which lengthens the *IQR*) has been nearly as large as the negative contribution from younger ages (which shortens it). In other words, the distribution of ages at death has been increasing more uniformly in recent decades; thus the compression of mortality witnessed in earlier periods has nearly halted.

The mortality changes that took place in Japan during 1951–1995 were smaller, but unfolded more rapidly, than the changes in Sweden during 1861–1995. Roughly speaking, Japanese mortality patterns in the early 1950s resembled those of Sweden during the late 1920s, or about a quarter-century earlier. By around 1980, however, Japan had caught up with Sweden in life expectancy—as well as in most indicators of health or mortality—and was on its way to becoming the world leader in this domain.

Because our data for Japan begin at a later point in that country's mortality history than the data for Sweden, the change in the life table *IQR* seen in Table 4 is also somewhat different. The most striking difference is that reductions in infant and child mortality were associated with a much smaller part of the reduction (whether absolute or relative) in the life table *IQR* for Japan during 1951–1995 than for Sweden during 1861–1995. On the other hand, the reduction in late-adult mortality (ages 50–74) had a much larger effect (again, either absolute or relative) on the *IQR* in Japan than in Sweden over these different periods. During most of the postwar period, the reduction in Japanese mortality rates in the age range 75–89 was outweighed by reductions at younger ages. In the most recent decade, however, mortality decline at the highest ages became dominant, resulting in a small increase in the *IQR*, as seen in Table 2 and Table 4. The decline in the *IQR* of ages at death began to slow considerably in the 1960s in Japan, about a decade later than in Sweden or the United States.

The change in the *IQR* for the United States resembles the pattern for Sweden in most respects, although the change is smaller (mostly because the time period is shorter). One important similarity is the dominant role of mortality decline below age 50 in the overall reduction of variability in the age at death. Another similarity between the American and the Swedish patterns is the slow rate of change in the *IQR* since the early 1950s. During the last four decades in both countries, most of the change in the *IQR* resulted from mortality reductions above age 50, although the positive and the negative contributions in this age range were balanced closely.

These decompositions suggest some generalities about the reduction of variability of ages at death within the life table. Over the long-term history of human mortality decline, most (perhaps about two-thirds) of the reduction in the life table *IQR* is attributable to mortality decline below age 5, which suppressed the "younger" mode of the bimodal distribution of ages at death. The rest of the change in the *IQR* is due mostly to mortality decline below age 50. Once a population has achieved moderately high levels of life expectancy (around 70 years), the secular decline in the *IQR* slows down. At this point, in a predominantly unimodal distribution with deaths concentrated at older ages, further reductions in mortality rates below age 50 have little effect on the *IQR*. Reductions above age 50 tend to show a balance of positive and negative impacts: In effect, the percentiles of the distribution of ages at death are raised in parallel.

Relationship to the Age Pattern of Mortality Decline

Figure 5 illustrates a rather curious historical finding. Whereas life expectancy rose gradually over the past 250 years in Sweden, variability of ages at death was reduced much more suddenly: almost entirely within a 75-year period from the late 1870s to the early 1950s. Both before and after this period of rapid change, the life table *IQR* shows little secular trend. Under what conditions, then, is a increase in life expectancy accompanied by a reduction in the variability of age at death? Conversely, when is variability stable even though life expectancy is increasing?

Presumably such questions should have neat mathematical answers, but unfortunately we have not succeeded in deriving useful analytical relationships except in some oversimplified special cases. Nevertheless Figure 6 provides some clues about the general character of the connection between trends in life expectancy and variability of age at death. Dividing the years from 1751 to 1995 into three periods (corresponding to the three parts of the historical *IQR* trend), we observe that the age pattern of mortality decline in Sweden differed significantly in these three eras. In general, during the two periods in which the *IQR* trend is flat, the average annual rate of mortality decline did not vary greatly across those ages that accounted for most of the deaths during each of those particular eras. Thus, during the period 1751–1755 to 1876–1880, the average rate of mortality decline was about 0.5% per year or less over most of the age range.[6] Similarly, rates of mortality decline have been about 1% per year since the 1950s for ages 30 and above, when most deaths now occur.

In contrast, during the period 1876–1880 to 1951–1955, the pace of mortality decline was unbalanced across the age range. In the first two decades of life, mortality risks decreased at annual rates of around 3% to 4% during this period, compared with rates of 1% or less at ages 50 and above. It appears, then, that this divergence in rates of mortality decline across the age range is the main cause of the decreasing variability in age at death. Although difficult to demonstrate mathematically, this conclusion is intuitively plausible: If

6. The presence of negative rates of mortality decline (supposedly indicating mortality *increase*) at ages 80 and above during this era probably reflects improvements in data quality (e.g., fewer exaggerated reports of extreme old age), which can cause a spurious pattern of mortality increase at older ages.

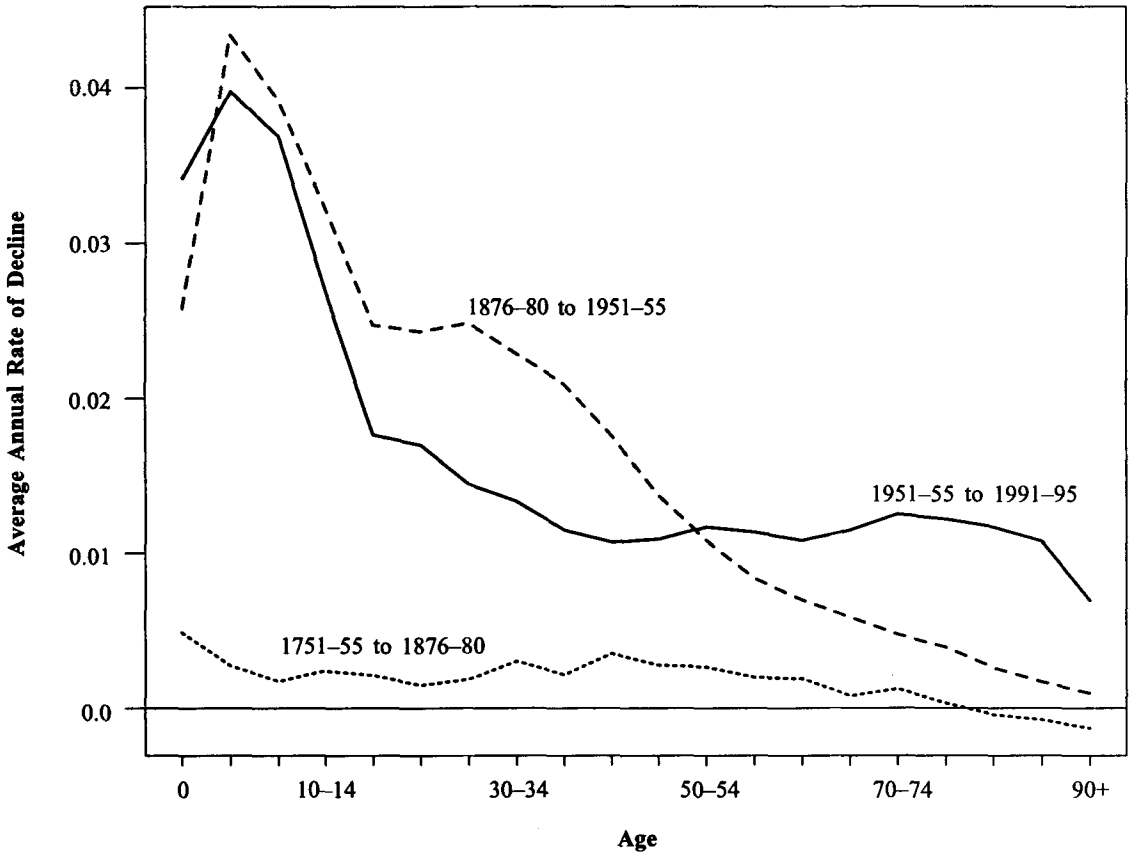
progress against mortality is concentrated in the earliest decades of life, deaths that would have occurred at younger ages are delayed more often than deaths that would have occurred at older ages. This differential process of delaying younger and older deaths results in an increasing concentration of ages at death. On the other hand, a more even pattern of mortality reduction across the age range (at least for those ages which account for most of the deaths) yields an increasing life expectancy with little change in the variability of age at death.

The youthful age pattern of mortality decline in the period 1876–1880 to 1951–1955 has been attributed to the "epidemiologic transition" (Omran 1971), in which there was a considerable decline in death rates due to infectious and parasitic diseases, perinatal and maternal disorders, and nutritional deficiencies. The age pattern of mortality decline flattened after the early 1950s because of unprecedented reductions in old-age mortality due to progress in combating degenerative diseases (Kannisto et al. 1994; Olshansky and Ault 1986). Thus the long historical decrease in the life table *IQR* was caused not by changes in senescent processes, but by a reduction in causes of early death that prevent senescence from occurring. On the other hand, the mortality decline of recent decades, which is linked more closely to changes in senescent processes, has not resulted in a continued rectangularization of the survival curve.

Variability of Age at Death for Birth Cohorts

All results presented thus far are based on period life tables. Therefore another question that emerges from Figure 5 is whether similar trends are evident for birth cohorts. A partial answer to this question is contained in Figure 7, which shows values of life expectancy at birth and displays the life table *IQR* for Swedish cohorts born from 1751–1755 through 1901–1905. Here again, although life expectancy rose gradu-

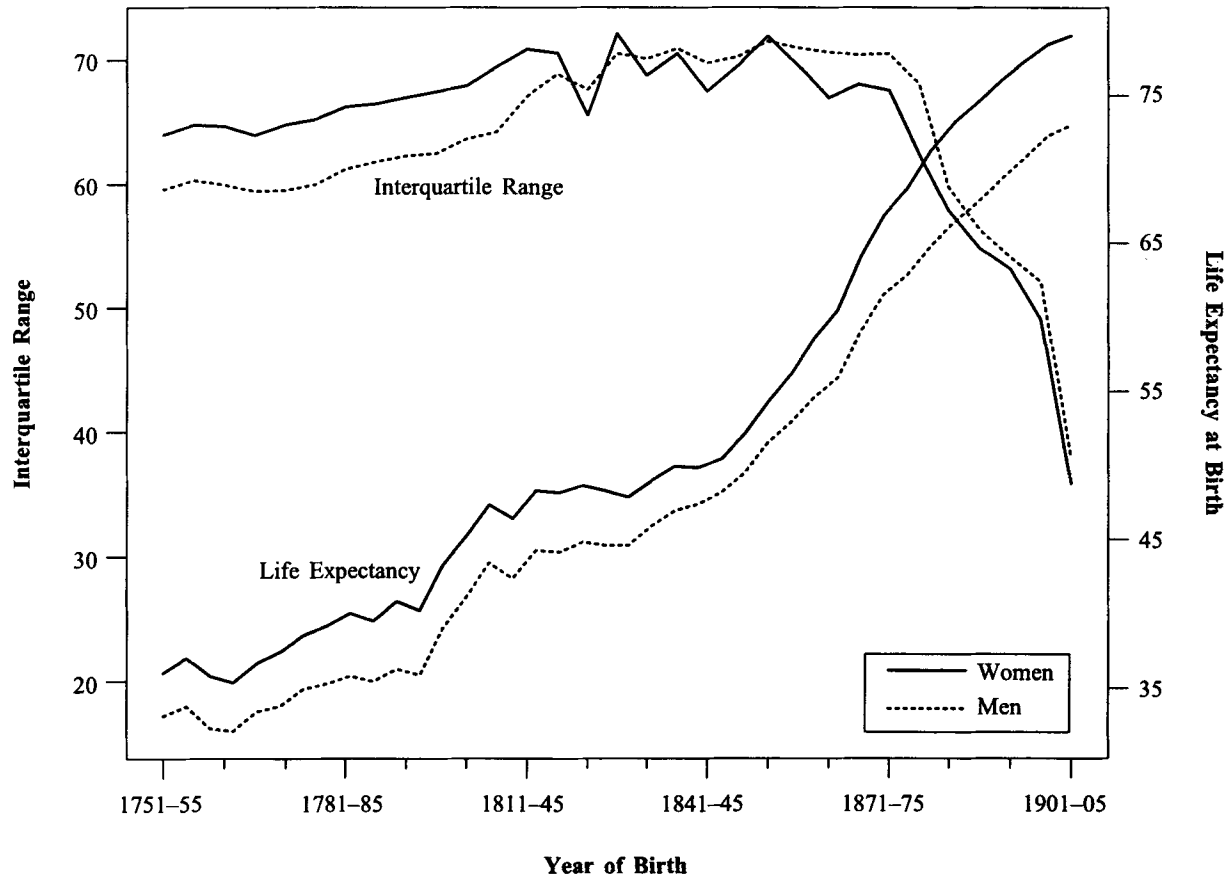
FIGURE 6. AVERAGE ANNUAL RATE OF MORTALITY DECLINE BY AGE, SWEDEN, SEXES COMBINED, THREE TIME INTERVALS DURING 1751–1995



Source: Swedish life tables reconstructed using data from various sources (Berkeley Mortality Database, http://demog.berkeley.edu/wilmoth/mortality).

Note: The average annual rate of mortality decline equals $-\ln(M_2/M_1)/(t_2 - t_1)$ where M_1 and M_2 are age-specific death rates for the earlier and the later time periods, respectively, and t_1 and t_2 are midpoints of the two time intervals.

FIGURE 7. INTERQUARTILE RANGE (*IQR*) OF LIFE TABLE AGES AT DEATH, AND LIFE EXPECTANCY AT BIRTH ($\mathring{e}_0$), SWEDISH WOMEN AND MEN, COHORTS BORN 1751–1905



Source: Swedish life tables reconstructed using data from various sources (Berkeley Mortality Database, http://demog.berkeley.edu/wilmoth/mortality).

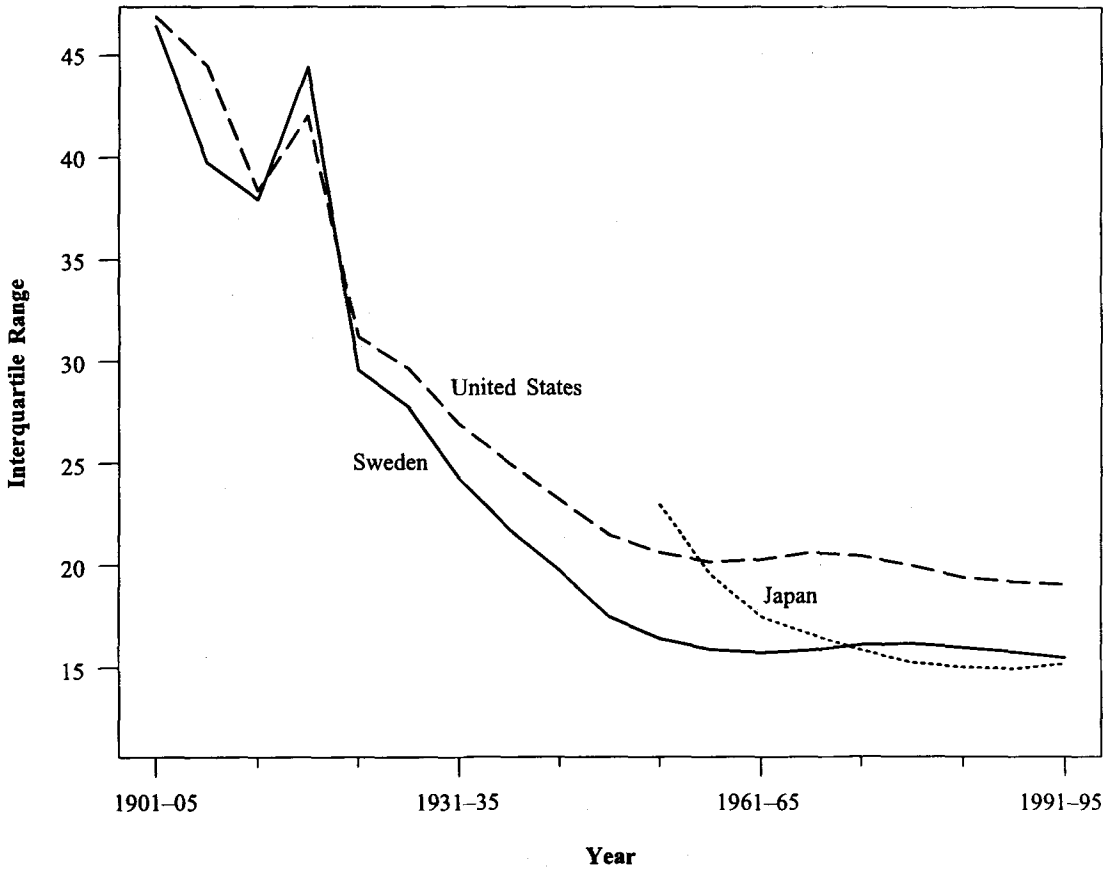
ally, the *IQR* declined sharply, beginning with cohorts born in the 1870s. These were the first cohorts to experience, as infants and children, the extremely rapid mortality decline that began in the last decades of the nineteenth century (compare Figure 6); again, this fact underlines the importance of rapid mortality decline at younger ages as the primary cause of the historical compression of mortality.

On the other hand, Figure 7 makes it evident that earlier cohorts underwent a modest "expansion of mortality." This expansion occurred because cohorts born from around 1800 to 1850 experienced the rapid mortality reduction of the late nineteenth century during the latter portion of their lives. Thus, from the perspective of these cohorts, the pace of mortality decline was greater at older than at younger ages, and resulted in an expansion, rather than a compression, of mortality.

The last cohort included in Figure 7 was born in 1901–1905. Today, in the late 1990s, some members of this cohort are still alive. Values of life expectancy for this cohort (and for the three preceding five-year cohorts) are based on the assumption that mortality rates after 1995 will be constant at the levels observed in 1991–1995. Thus life expectancies shown here for these last cohorts are probably slight underestimates of their ultimate values (if we assume a continuation of mortality decline after 1995). The *IQR* values for these cohorts are entirely accurate, however, because at least 75% of all cohorts shown in Figure 7 had died by 1995. (Indeed, 1901–1905 was the last five-year cohort for which the actual *IQR* could be computed with data through 1995.)

Therefore, given the problem of incomplete data for recent cohorts, it is impossible to determine when the life table *IQR* for Swedish cohorts may reach a lower plateau, like the *IQR* based on period life tables. Nevertheless, in light of the overall similarity between period and cohort patterns (due, as noted above, to the predominant role of infant and child mortality in the historical reduction of variability), it is reasonable to guess that the life table *IQR* may converge, at least temporarily, toward a level of about 15 years for Swedish cohorts born around 1950 and later. Obviously such a guess is speculative in view of uncertainties about future mortality trends.

FIGURE 8. INTERQUARTILE RANGE (*IQR*) OF LIFE TABLE AGES AT DEATHS OVER TIME, SWEDEN, JAPAN, AND THE UNITED STATES, 1901–1995



Sources: U.S. life tables: Office of the Chief Actuary, Social Security Administration (Bell et al. 1992). Swedish and Japanese life tables: Berkeley Mortality Database, http://demog.berkeley.edu/wilmoth/mortality.

This level of variability is considerably greater than predicted by Fries (1980) for his "ideally rectangular" mortality profile. Fries envisioned an ultimate scenario in which life expectancy would equal 85 years, and deaths would be distributed as in a normal distribution around age 85, with a standard deviation of about four years. For a normal distribution, a standard deviation of four years implies an *IQR* of 5.4 years. Thus, although we may be observing some convergence toward a more stable pattern of variability in the age at death, the degree of variability remaining is much greater than predicted by Fries, and variability has stabilized despite a continuing increase in life expectancy.

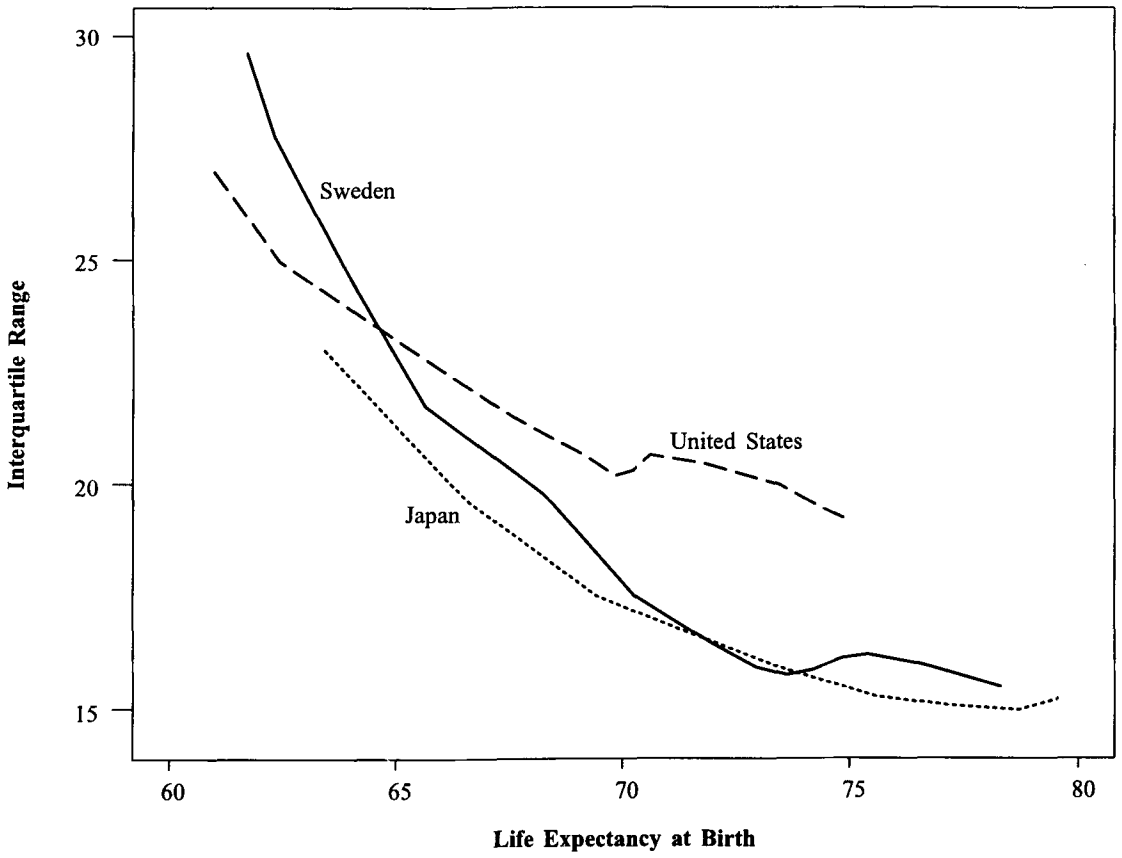
Variability of Age at Death Across Societies

Although decreasing variability of age at death is a characteristic of all three populations considered here, the absolute level of variability differs. Figure 8 shows that the variability of age at death in the United States during recent decades has been greater by almost five years (as measured by the life table *IQR*) than in either Sweden or Japan. Levels of variability were similar in the United States and in Sweden during the early part of this century but then diverged slowly. Among these three countries, variability in age at death was highest in Japan during the early 1950s, but then decreased quickly with the rapid rise in life expectancy.

We should expect variability in age at death to be relatively high for the United States in recent decades, because life expectancy has been lower there than in either Sweden or Japan during this period. Therefore Figure 9 plots the life table *IQR* against life expectancy at birth in these three countries. Here again, we see evidence of the unusual character of the age pattern of mortality in the United States.[7] For lev-

7. This anomalous age pattern of mortality in the United States (even when the age range is limited to ages 50 and above) was also noted by Himes (1994).

FIGURE 9. INTERQUARTILE RANGE (*IQR*) OF LIFE TABLE DEATHS VERSUS LIFE EXPECTANCY AT BIRTH ($\mathring{e}_0$), SWEDEN, JAPAN, AND THE UNITED STATES



Sources: U.S. life tables: Office of the Chief Actuary, Social Security Administration (Bell et al. 1992). Swedish and Japanese life tables: Berkeley Mortality Database, http://demog.berkeley.edu/wilmoth/mortality.

els of life expectancy above 65 years, the variability of age at death has been greater in the United States than in Sweden or Japan. For levels of life expectancy below 65 years, however, the life table *IQR* is highest in Sweden.

Therefore this preliminary analysis points to two phenomena that require explanation: the higher variability of age at death in the United States in recent decades, and the greater variability in Sweden during the early portion of this century. The diversity of the American population, with its greater socioeconomic inequality, seems the most likely cause of the former pattern. On the other hand, the higher variability of age at death in Sweden earlier in this century is a curiosity with no obvious explanation. This excess, however, is merely relative (in regard to life expectancy). From a purely historical perspective, as shown in Figure 8, age at death was no more variable in Sweden than in the United States during this period, although Swedish life expectancy was somewhat higher.

SIGNIFICANCE OF THE HISTORICAL COMPRESSION OF MORTALITY

The decline of human mortality during the industrial era is typically described in terms of increased life expectancy or decreased risks of death across the age range. In contrast, the decreased variability of age at death has received relatively little attention. Similarly, although some studies have been conducted on social and behavioral effects of the historical mortality decline (e.g., several articles in United Nations 1986), little research has focused on the consequences of reduced variability in age at death. Indeed, most analyses of the compression of mortality have been motivated by questions about biological limits to the human life span, with little discussion of the broader historical significance of this phenomenon.

In this paper we have documented a major historical change. Using Swedish data, we have shown that the enormous rise in life expectancy during the industrial era (from

the mid-thirties to the high seventies) was accompanied by an equally impressive reduction in the variability of age at death (from about 65 to 15 years, expressed in terms of the life table *IQR*). Although this change has little significance for the debate about potential limits to the human life span, it has important implications for individuals as they contemplate their own mortality and make decisions about how to structure their lives. Although a complete analysis of the topic is beyond the scope of this article, we offer the following speculative remarks with the hope that they will stimulate further discussion and research.

A fundamental issue is the connection between our fear of death and its timing within a population. Fear of death is an interesting topic not only for its psychological ramifications, but also in light of the social and economic decisions that individuals make in an attempt to avert death or mitigate its consequences. Presumably our expectations and our fears about death are formed in part by observing the mortality of those who surround us. For example, merely by noting the pattern of dying around them, individuals in modern industrial societies should recognize that they face only a modest threat of death before old age.

Has this increased certainty of survival resulted in a diminished fear of death, not only as we contemplate our own mortality but also as we envision the departure of friends and loved ones? It seems possible and even likely that increased certainty of survival has strengthened individual expectations about a proper ordering of deaths within a family or social group. When these expectations are confirmed, our fear of death may be attenuated, as suggested by Kastenbaum:

> The old person tends to be seen as the most suitable selection in the pecking order of death. Sorrow may follow the death of the person whose name appears atop the list, but the very fact that one's expectations have been confirmed provides a measure of psychological security. Death is "playing the game" according to the rules that we would like to believe have been established. (1985:621)

According to this argument, when death strikes predominantly the elderly, the idea that younger people are less at risk is confirmed, and the young are reinforced in their comfortable belief that they are out of death's reach.

Another consideration is the connection between our fear of death and the social and economic disruption that it causes for both decedents and survivors. Death is disruptive not only for the individual involved but also for family, friends, and associates. After a death, survivors often face new circumstances and challenges. This effect may be greatest when death strikes a young adult, especially the parent of a young child. Conversely, an elderly person's death may be less disruptive to the lives of their children, who are typically grown and independent. Accordingly the historical extension of longevity and compression of mortality also may have diminished our fear of death by making it less disruptive overall from the survivors' perspective.

Although it may seem plausible that mortality compression has diminished the fear of death, there are reasons for caution. For example, it also seems possible that increased certainty of long life may have made premature death more tragic, evoking greater fear and encouraging more elaborate strategies of avoidance and preparation. An anthropological study of impoverished communities in northeastern Brazil illustrates the indifference to young people's death and suffering in a society where infant and child death is commonplace (Scheper-Hughes 1992). The Brazilian mothers' complacency and even complicity surrounding the death of a weak child may be shocking by American standards. Conversely, the enormous investment in health and medicine made by wealthy societies could be viewed, from an outside perspective, as reflecting an extraordinary fear of death.

How do individuals in these various settings perceive death, and in what different ways do they fear it? How is the psychology of death in a society related to the social and economic systems established to avert and manage death? How has the compression of mortality affected these relationships? In light of the enormous change in the variability of age at death that we have documented, cross-cultural and historical studies of these and similar issues seem warranted.

CONCLUSION

The key findings of this paper can be summarized as follows:

1. Ten measures of rectangularity (of the survival curve) or variability (of age at death) were presented and shown to be highly correlated. The interquartile range (*IQR*) of life table ages at death was chosen for this analysis because it is highly correlated with the other measures and because it is convenient and easily interpretable.

2. On the basis of an analysis of Swedish data, variability of age at death (as measured by the life table *IQR*) has decreased from 65 to 15 years, approximately, during the past 250 years. These changes in variability reflect three important phases of mortality decline: a period of slow mortality reduction across the age range, associated with high but stable levels of variability in age at death, from the mid-eighteenth century to the late 1870s (in Sweden); an era of rapid reduction in infant and child mortality, resulting in an enormous and rapid compression of mortality, during a 75-year period from the late 1870s to the early 1950s; and a period of unprecedented reduction in late-adult mortality, accompanied by near-constant levels of variability in ages at death, from the 1950s to the 1990s. Because of the limited availability of historical data, we cannot observe all three of these phases for most countries, although in the present analysis the transition from the second to the third phase is clearly visible for Japan and the United States as well.

3. Using a new method of decomposition, we showed that the historical compression of mortality can be attributed to declining risks of death across a broad age range (from birth to age 75), although the greater proportional reduction in mortality rates at younger ages (especially below age 5) has had a predominant influence. The historical reduction in the life table *IQR* occurred mostly during a period when rela-

tive rates of mortality decline were much greater at younger than at older ages. Empirically, when death rates decline at a similar pace across the age range, the variability of age at death changes little. Our analysis of cohort patterns of mortality is consistent with this conclusion.

4. In recent decades, the mortality pattern in the United States has been characterized by greater variability in ages at death than in both Sweden and Japan, even when we consider that levels of life expectancy are lower in the United States.

In light of these findings, what should we expect about future trends in the variability of age at death in low-mortality populations? If the current decline in mortality ceases, the distribution of ages at death (including levels of variability) will stabilize. Yet if mortality rates continue to decline rapidly at both younger and older adult ages, we should expect a continuing pattern of stability in the variability of ages at death, as the entire distribution shifts upward. On the one hand, Fries's prediction of a continuing rectangularization toward a *fixed* mortality pattern is inconsistent with the trends of recent decades. On the other hand, the hypothesis that we are undergoing an expansion of mortality (also called "derectangularization" by Gavrilov and Gavrilova 1991) receives only scant support from our analysis of data for these three countries. The small expansion of mortality that occurred in one instance (for Japan during the most recent decade) is trivial when compared with the enormous historical reduction in variability that preceded it.

From a broad historical perspective, rectangularization of the human survival curve is by no means a myth. In recent decades, however, variability in the distribution of deaths by age has stabilized. Moreover, the current pattern of mortality decline suggests that the level of variability observed today, with most deaths concentrated in an historically narrow age range, could be maintained for the foreseeable future, whether or not life expectancy continues to increase.

APPENDIX A

In this appendix we give definitions for six measures of the rectangularity of the survival curve and four measures of the variability of age at death.

1. Fixed rectangle. Choose a right endpoint, ϕ, and consider the rectangle bounded by the x- and the y-axis, and the lines $x = \phi$ and $y = 1$. Compute the proportion of this (fixed) rectangle that falls under the survival curve:

$$FR \equiv \frac{\int_0^{\phi} S(x)dx}{\phi}. \tag{A1}$$

In this study, we set $\phi = 100$. As the survival curve becomes more rectangular, FR increases.[8]

2. Moving rectangle. Choose a right endpoint, γ, such that $S(\gamma) = \varepsilon$ for the survival curve in question. Compute the proportion of this (moving) rectangle that falls under the survival curve:

$$MR \equiv \frac{\int_0^{\gamma} S(x)dx}{\gamma}. \tag{A2}$$

In this study, we set $\varepsilon = 10^{-3}$. As the survival curve becomes more rectangular, MR increases.

3. Fastest decline. Compute the largest negative slope for the survival curve in the adult age range (arbitrarily, above age 15):

$$FD \equiv \max_x\{-S'(x)\} \text{ for } x \geq 15. \tag{A3}$$

In empirical applications first differences are used in place of derivatives, and the $S(x)$ curve is smoothed before differencing in order to minimize the influence of random fluctuations.[9] This measure reflects the steepness of the survival curve's descent in the upper adult age range. The value of FD increases as the survival curve becomes more rectangular.[10]

4. Sharpest corner. Compute the largest *negative* second derivative for the survival curve in the adult age range:

$$SC \equiv \max_x\{-S''(x)\} \text{ for } x \geq 15. \tag{A4}$$

In applications, we used second differences of a smoothed $S(x)$ curve. This measure reflects the rapidity of the downward turn in the survival curve at advanced adult ages (*before* its steepest descent, as measured by FD). The value of SC increases as the survival curve becomes more rectangular. This measure is called the *minimum curvature* by Eakin and Witten (1995).

5. Quickest plateau. Compute the largest *positive* second derivative for the survival curve in the adult age range:

$$QP \equiv \max_x\{S''(x)\} \text{ for } x \geq 15. \tag{A5}$$

In applications, we use second differences of a smoothed $S(x)$ curve. This measure reflects the rapidity of the flattening in the survival curve at very high ages (*after* its steepest descent, as measured by FD). The value of QP increases as the survival curve becomes more rectangular. Eakin and Witten (1995) call this measure the *maximum curvature.*

6. Prolate index. Let x' and x'' be the ages corresponding to the sharpest corner and quickest plateau measures of rectangularity. Thus $SC = S''(x')$ and $QP = S''(x'')$. Let θ be the angle between the vertical line, $x = x''$, and a line connecting $(x', S(x'))$ with $(x'', S(x''))$, as shown in Figure A1. The prolate index, as defined by Eakin and Witten (1995), equals the cosine of θ:

8. Integrals involving $S(x)$ are computed in the standard fashion with life table values of l_x. Suppose that values of l_x are available for ages a and b (for $a < b$) but for no intermediate values. Then $\int_a^b S(x) \equiv (b-a)\frac{l_a + l_b}{2}$. The full integral is approximated by summing over individual age intervals across the range of integration. For life tables with single-year age intervals, $b - a = 1$. Thus, in this example,

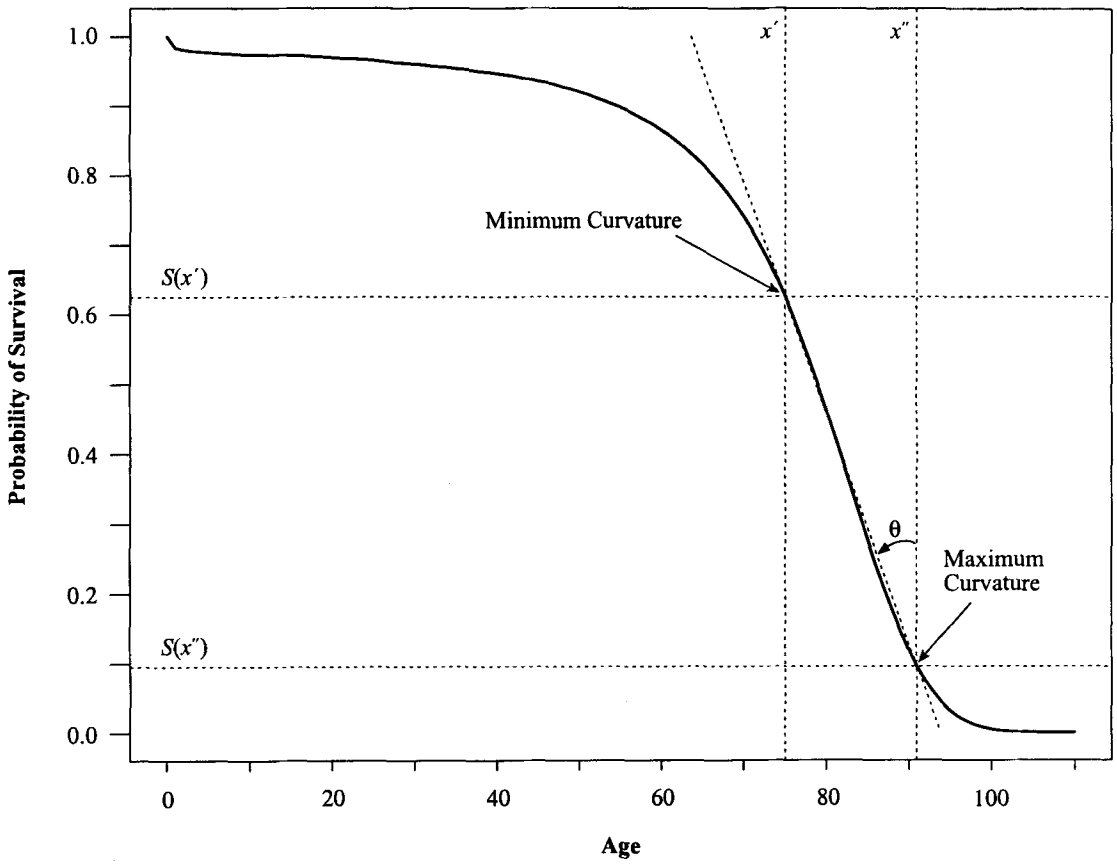
$$FR \equiv \frac{\int_0^{100} S(x)dx}{100} \equiv \frac{1}{100}\left[\frac{1}{2}(l_0 + l_1) + \frac{1}{2}(l_1 + l_2) + \ldots + \frac{1}{2}(l_{99} + l_{100})\right]$$

$$= \frac{1}{100}\left[\frac{1}{2}l_0 + \sum_{x=1}^{99} l_x + \frac{1}{2}l_{100}\right].$$

9. The smoothing algorithm used for FD, SC, and QP was 4(3RSR)2H, twice (Tukey 1977), available as the function, smooth(), in S-Plus.

10. As noted in the text, FD also equals the height of the density function, $f(x^*)$, at the mode of its distribution, x^*.

FIGURE A1. ILLUSTRATION OF *SC*, *QP*, AND *PI* MEASURES OF RECTANGULARITY



Note: See Appendix A for explanation.

$$PI \equiv \cos(\theta) = \frac{S(x') - S(x'')}{\sqrt{[S(x') - S(x'')]^2 + [x'' - x']^2}}. \quad \text{(A6)}$$

Since $S(x)$ is nonincreasing, θ must be between 0 and $\pi/2$; thus the values of *PI* lie between 1 and 0. As the survival curve becomes more rectangular, θ becomes smaller and thus *PI* increases.

7. Interquartile range of age at death. Compute the interquartile range (*IQR*) of the distribution of ages at death in the life table:

$$IQR \equiv x_2 - x_1, \quad \text{(A7)}$$

where x_1 and x_2 are ages such that $S(x_1) = 0.75$ and $S(x_2) = 0.25$. As the distribution of ages at death becomes less variable, $x_2 - x_1$ decreases.

8. Standard deviation of age at death. Compute the standard deviation, σ, of the distribution of ages at death in the life table:

$$\sigma^2 \equiv \int_0^\infty x^2 f(x)dx - \mathring{e}_0^2$$
$$= 2\int_0^\infty \int_x^\infty S(y)dydx - \mathring{e}_0^2, \quad \text{(A8)}$$

where

$$\mathring{e}_0 \equiv \int_0^\infty xf(x)dx = \int_0^\infty S(x)dx. \quad \text{(A9)}$$

As the distribution of ages at death becomes less variable, $\sigma = \sqrt{\sigma^2}$ decreases.

9. Gini coefficient. As demonstrated by Hanada (1983), Gini's concentration ratio can be computed with life table data:

$$G \equiv 1 - \frac{1}{\mathring{e}_0}\int_0^\infty [S(x)]^2 dx. \quad \text{(A10)}$$

Thus G is a measure of inequality in age at death. As the variability of age at death declines, the value of G decreases.

10. Keyfitz' s *H*. By definition,

$$H \equiv -\frac{\int_0^\infty S(x)\cdot \ln[S(x)]dx}{\int_0^\infty S(x)dx}. \quad \text{(A11)}$$

In its original application (Keyfitz 1985), a proportional reduction of size δ in the force of mortality across the age range yields an approximate relative increase of $H\delta$ in the expectation of life at birth, $\mathring{e}_0$. As the age at death becomes less variable, an equivalent proportional reduction in death rates yields a smaller relative increase in life expectancy. Thus, as variability of age at death declines, the value of H decreases (Nagnur 1986; Nusselder and Mackenbach 1996).

APPENDIX B

In this appendix we propose a method for decomposing a change (or difference) in the life table interquartile range (*IQR*). With this method, ΔIQR is decomposed into components attributable to mortality change (or difference) in distinct age groups.

Let $\mu(x,t)$, the force of mortality at age x and time t for some population, be a continuous function of age and time. Also, let $H(x,t)$ be the cumulative hazard function at time t:

$$H(x,t) = \int_0^x \mu(y,t)dy. \quad \text{(B1)}$$

Furthermore, let $x_p(t)$ be the pth percentile of the distribution of ages at death (i.e., within a period life table) at time t. Formally this percentile is defined in terms of the cumulative density function and its complement, the survival function. Thus $x_p(t)$ equals an age x such that

$$\begin{aligned} F(x,t) &= 1 - S(x,t) \\ &= 1 - e^{-\int_0^x \mu(y,t)dy} \\ &= 1 - e^{-H(x,t)} \\ &= \frac{p}{100}. \end{aligned} \quad \text{(B2)}$$

Finally, the life table *IQR* at time t is defined as the difference between the 25th and the 75th percentiles of the life table distribution of deaths:

$$IQR(t) = x_{75}(t) - x_{25}(t). \quad \text{(B3)}$$

As the theoretical basis for the decomposition of ΔIQR, we will show that

$$\begin{aligned} \Delta x_p(t_1,t_2) &= x_p(t_2) - x_p(t_1) \\ &= \int_{t_1}^{t_2}\int_0^\infty \frac{-\frac{\partial}{\partial t}\mu(x,t)}{\mu(x_p(t),t)}\cdot I_p(x,t)dxdt, \end{aligned} \quad \text{(B4)}$$

where

$$I_p(x,t) = \begin{cases} 1 \text{ if } x < x_p(t) \\ 0 \text{ else} \end{cases}. \quad \text{(B5)}$$

Thus

$$\begin{aligned} \Delta IQR(t_1,t_2) &= \Delta x_{75}(t_1,t_2) - \Delta x_{25}(t_1,t_2) \\ &= \int_{t_1}^{t_2}\int_0^\infty -\frac{\partial}{\partial t}\mu(x,t)\cdot\left[\frac{I_{75}(x,t)}{\mu(x_{75}(t),t)} - \frac{I_{25}(x,t)}{\mu(x_{25}(t),t)}\right]dxdt \\ &= \int_{t_1}^{t_2}\int_0^\infty \rho(x,t)\cdot\mu(x,t)\left[\frac{I_{75}(x,t)}{\mu(x_{75}(t),t)} - \frac{I_{25}(x,t)}{\mu(x_{25}(t),t)}\right]dxdt, \end{aligned} \quad \text{(B6)}$$

where $\rho(x,t) = \frac{-\frac{\partial}{\partial t}\mu(x,t)}{\mu(x,t)} = -\frac{\partial}{\partial t}\ln(\mu(x,t))$ is the relative rate of mortality decline at age x and time t.

Using this formula and numerical integration, one can estimate ΔIQR very accurately. Of course there is no reason to make these calculations merely as a means of finding ΔIQR, which can be computed directly from the two life tables. Rather, this formula is useful as a means of decomposing ΔIQR into components attributable to mortality change in specific age groups. For example, the portion of ΔIQR attributed to mortality change in the age interval from a to b equals

$$\int_{t_1}^{t_2}\int_a^b \rho(x,t)\cdot\mu(x,t)\cdot\left[\frac{I_{75}(x,t)}{\mu(x_{75}(t),t)} - \frac{I_{25}(x,t)}{\mu(x_{25}(t),t)}\right]dxdt. \quad \text{(B7)}$$

In the remainder of this appendix, we derive these formulas analytically and discuss numerical methods for approximating the integrals.

Derivation

The derivation of the above formulas depends on the following simple result:

For all values of $p \in (1,100)$ and $t \in \Re$,

$$\begin{aligned} \frac{\partial}{\partial t}x_p(t) &= \frac{-\frac{\partial}{\partial y}H(x_p(t),t+y)\Big|_{y=0}}{\mu(x_p(t),t)} \\ &= \int_0^{x_p(t)} \frac{-\frac{\partial}{\partial t}\mu(x,t)}{\mu(x_p(t),t)}dx. \end{aligned} \quad \text{(B8)}$$

Proof: Since $F(x_p(t),t) = 1 - S(x_p(t),t) = 1 - e^{-H(x_p(t),t)} = \frac{p}{100}$ is constant (by definition), the cumulative hazard for the pth percentile, $H(x_p(t),t)$, must be identical for all values of t. Thus, for example,

$$H(x_p(t),t) = H(x_p(t+dt),t+dt), \quad \text{(B9)}$$

where dt is some small, positive number.

Case 1: Suppose that $\frac{\partial}{\partial t}x_p(t) > 0$, so that $x_p(t+dt) > x_p(t)$. Then,

$$\begin{aligned} &H(x_p(t),t) = H(x_p(t+dt),t+dt) \\ &= H(x_p(t),t+dt) + \int_{x_p(t)}^{x_p(t+dt)}\mu(x,t+dt)dx \\ &= H(x_p(t),t+dt) + \mu(x^*,t+dt)\cdot\left[x_p(t+dt) - x_p(t)\right], \end{aligned} \quad \text{(B10)}$$

for some value, x^*, that lies between $x_p(t)$ and $x_p(t+dt)$. Rearranging this equation and dividing by dt yields

$$\frac{x_p(t+dt) - x_p(t)}{dt}$$

$$= -\frac{1}{\mu(x^*,t+dt)} \cdot \frac{H(x_p(t),t+dt) - H(x_p(t),t)}{dt}. \quad \text{(B11)}$$

Letting dt go to zero in this equation completes the proof for this case.

Case 2: Suppose that $\frac{\partial}{\partial t} x_p(t) < 0$, so that $x_p(t) > x_p(t + dt)$. Then,

$$H(x_p(t),t) = H(x_p(t+dt),t+dt).$$
$$= H(x_p(t),t+dt) - \int_{x_p(t+dt)}^{x_p(t)} \mu(x,t+dt)dx$$
$$= H(x_p(t),t+dt) - \mu(x^*,t+dt)\cdot\left[x_p(t) - x_p(t+dt)\right]. \quad \text{(B12)}$$

Rearranging this equation, dividing by dt, and letting dt go to zero yields the same result as in the previous case. QED

The main result (given earlier) follows easily from this lemma:

$$\Delta x_p(t_1,t_2) = x_p(t_2) - x_p(t_1)$$
$$= \int_{t_1}^{t_2} \frac{\partial}{\partial t} x_p(t)dt$$
$$= \int_{t_1}^{t_2} \frac{-\frac{\partial}{\partial y} H(x_p(t),t+y)\Big|_{y=0}}{\mu(x_p(t),t)} dt$$
$$= \int_{t_1}^{t_2} \frac{\int_0^{x_p(t)} -\frac{\partial}{\partial t}\mu(x,t)dx}{\mu(x_p(t),t)} dt$$
$$= \int_{t_1}^{t_2}\int_0^{\infty} \frac{-\frac{\partial}{\partial t}\mu(x,t)}{\mu(x_p(t),t)} \cdot I_p(x,t)dxdt. \quad \text{(B13)}$$

Numerical Methods

Suppose that we know the value of $\mu(x,t)$ for only two points in time, t_1 and t_2. If we assume that, for a given age x, $\rho(x,t)$ is constant during the interval $[t_1, t_2)$, then the relative rate of mortality decline at x between t_1 and t_2 equals

$$\rho(x) = -\frac{1}{\Delta t}\ln\left(\frac{\mu(x,t_2)}{\mu(x,t_1)}\right), \quad \text{(B14)}$$

where $\Delta t = t_2 - t_1$, and the force of mortality for some t in this interval equals

$$\mu(x,t) = \mu(x,t_1)\cdot e^{-(t-t_1)\rho(x)}. \quad \text{(B15)}$$

Alternative assumptions about the pattern of mortality change over time (for example, constant arithmetic change, or unusual patterns in which all the change occurs in the first half of the interval for some ages and in the second half for others) yield only slightly different results for this decomposition. Among the various possible assumptions, a constant relative rate of mortality decline is certainly the most consistent with patterns typically observed in real data (e.g., Lee and Carter 1992).

To compute the outer integral in the equation for $\Delta IQR(t_1,t_2)$, divide the time interval into N equal subintervals. Observe that the force of mortality at the midpoint of the nth sub-interval equals

$$\mu^n(x) = \mu(x,t_1 + \tfrac{2n-1}{2N}\Delta t) = \mu(x,t_1)\cdot e^{-\frac{2n-1}{2N}\Delta t\cdot\rho(x)}, \quad \text{(B16)}$$

where $n = 1,2,\ldots,N$. By standard numerical methods, then, we can approximate the outer integral as follows:

$$\Delta IQR(t_1,t_2)$$
$$\approx \sum_{n=1}^{N}\left\{\frac{\Delta t}{N}\int_0^{\infty}\rho(x)\cdot\mu^n(x)\cdot\left[\frac{I_{75}^n}{\mu^n(x_{75}^n)} - \frac{I_{25}^n}{\mu^n(x_{25}^n)}\right]dx\right\}. \quad \text{(B17)}$$

In most situations, a choice of $N = 20$ yields quite accurate results. When there is little difference between the two mortality schedules, however, smaller values of N may suffice.

The methods for approximating the inner integral in these formulas require only a brief explanation. Suppose that, instead of $\mu(x,t_1)$ and $\mu(x,t_2)$, we possess observed death rates, $m(x,t_1)$ and $m(x,t_2)$, based on discrete intervals of age and time. The discreteness of time presents no problem, so long as we remember that Δt equals the distance between the midpoints of the two time intervals. The discreteness of age also is unproblematic, so long as we make proper use of linear interpolation in approximating the integrals in the above formulas.

Consider a set of discrete death rates, $m(x)$. To find the pth percentile, x_p, first compute the implicit value of the corresponding cumulative hazard:

$$H(x_p) = -\ln\left(1 - \frac{p}{100}\right). \quad \text{(B18)}$$

Then find an age x such that

$$M_1 = \sum_{y=0}^{x} m(y) \le H(x_p)$$
$$\text{and } M_2 = \sum_{y=0}^{x+1} m(y) > H(x_p). \quad \text{(B19)}$$

Since $m(x)$ is the death rate over the interval $[x,x + 1)$, x_p must lie in the interval $[x + 1, x + 2)$. By linear interpolation, we approximate x_p as follows:

$$x_p \approx x + 1 + \frac{H(x_p) - M_1}{M_2 - M_1}. \quad \text{(B20)}$$

Finally, the estimation of $\mu(x_p)$ in the above formulas is based on linear interpolation as well. Recall that $m(x)$ gives a point estimate for $\mu(x + 0.5)$, not $\mu(x)$. Therefore find x such that $x + 0.5 \le x_p < x + 1.5$ (not necessarily the same x as in the previous paragraph) and estimate $\mu(x_p)$ by a simple linear interpolation of $m(x)$:

$$\mu(x_p) \approx (x + 1.5 - x_p)\cdot m(x)$$
$$+ (x_p - (x + 0.5))\cdot m(x + 1). \quad \text{(B21)}$$

These results are based on the assumption that observed death rates are available for single-year age intervals. If these rates are available only for broader age intervals (for example, 5- or 10-year age groups), the preceding approximation methods could be adapted easily.

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