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AGE PATTERNS OF MORTALITY FOR OLDER WOMEN: AN ANALYSIS USING THE AGE-SPECIFIC RATE OF MORTALITY CHANGE WITH AGE

SHIRO HORIUCHI

Laboratory of Populations
Rockefeller University
New York, NY 10021-6399

ANSLEY J. COALE

Office of Population Research
Princeton University
Princeton, NJ 08544

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The age-specific rate of mortality change with age, defined by $k(x) = d \ln \mu(x)/dx$, where $\mu(x)$ is the age-specific death rate at exact age x, is estimated for middle and old ages in ten selected populations that are considered to have relatively accurate age data. For females in each of the study populations, $k(x)$ follows a bell-shaped curve that usually peaks around age 75. In some of the populations, the age pattern of $k(x)$ for males is confounded with substantial cohort variations, which seem to reflect long-term impacts of their World War I experiences.

Among the mathematical models proposed by Gompertz, Makeham, Perks and Beard, only the Perks model is consistent with the bell-shaped pattern of $k(x)$. It is shown that, if the risk of death for every individual follows the Makeham equation and if the individual frailty is gamma-distributed, then the age-specific death rate follows the Perks equation.

KEY WORDS: Mortality, old-age mortality, aging, heterogeneity, Gompertz model, Perks model.

INTRODUCTION

In this paper we propose a mortality measure that seems useful in analyzing age patterns of death rates. The measure, which will be denoted by $k(x)$, indicates the *proportional* increase or decrease with age in the risk of death at a given age x, and is called the *age-specific rate of mortality change with age*.[1] In old ages, the risk of death grows with age. Since the increment itself tends to be larger at older ages of higher mortality, it seems useful to measure the mortality increase *relative to* the risk of death and examine its variations.

[1]This measure is concerned with age variations, not period variations. Age-specific rate of mortality change *over time* can be calculated in a similar way.

Little attention has been given, however, to the measurement of $k(x)$ partly due to the common exercise in mortality research to calculate the logarithms of age-specific death rates and plot them against age. In theory, the plot is expected to indicate patterns of proportional mortality change with age: a rising (declining) rate of mortality increase results in an upwardly (downwardly) concave curve, and a constant proportional growth results in a straight line. Usually, plotted points are narrowly scattered around a straight line over a wide range of middle and old ages. However, as shown later in Figures 1 and 2, the logarithms of death rates plotted against age tend to *appear* to fall on a straight line, even when the value of $k(x)$ changes substantially with age. Therefore, age variations in the proportional increment of death rate should be detected not by looking at graphs of the logarithms of death rates plotted against age but by calculating $k(x)$ directly.

This measure has at least four advantages in mortality research, which will be illustrated in this paper. First, it is useful in assessing mathematical models of mortality. Several equations describing the mortality increase with age among middle-aged and old persons have been developed over the period of more than one and a half centuries, including those by Gompertz, Makeham, Perks and Beard.

It is not a very simple task, however, to evaluate and compare the validity of those models. All of those models usually fit observed death rates and their logarithms well, making it difficult to determine which model fits the data best. Furthermore, those models have different numbers of parameters. In general, models with more parameters tend to fit data better, introducing additional difficulties in comparative assessment of those models. Those mathematical models, however, can be straightforwardly evaluated using $k(x)$, because age patterns of $k(x)$ implied by those models are significantly different.

Secondly, $k(x)$ is expected to provide some clues about the heterogeneity of a population with respect to mortality. A population may be considered to consist of subgroups that follow different mortality schedules. The proportions of those subgroups in the population vary with age because the groups exposed to higher risk tend to be reduced faster. Therefore, the age pattern of $k(x)$ for the entire population may be significantly different from those of its subgroups, giving some clues about the distribution in the population of the individual vulnerability to the risk of death. It will be shown later that the introduction of a heterogeneity model helps explain observed $k(x)$ patterns.

Thirdly, $k(x)$ seems useful in studying physiological aging. The risk of death increases as the human body degenerates, so that $k(x)$ may reflect age variations in the "pace of aging."

Finally, $k(x)$ is useful in detecting cohort mortality variations. For example, if a low mortality cohort is followed by a high mortality cohort, mortality increases with age from the younger group to the older group at a relatively low rate, so that $k(x)$ tends to be small. If the order is reversed, $k(x)$ tends to be large. Therefore, if a cohort experiences a relatively higher mortality schedule than its adjacent ones, low values of $k(x)$ at older ages tend to be followed by high values of $k(x)$ at younger ages, and the high-mortality cohort is likely to be located between those unexpectedly high and low values of $k(x)$.

In the following sections, we will investigate the $k(x)$ function in selected populations, identify their similarities and differences, examine the validity of those findings, analyze the observed $k(x)$ patterns in relation to existing mathematical models, and discuss a hypothetical underlying mechanism that could generate the observed $k(x)$ patterns.

METHODS AND MATERIALS

The force of mortality (or the instantaneous death rate) at exact age x is given by

$$\mu(x) = \frac{d\ln(l(x))}{dx}, \tag{1}$$

where $l(x)$ is the life-table function representing the proportion surviving from birth to age x. The rate of mortality change at age x is then defined by

$$k(x) = \frac{d\ln(\mu(x))}{dx}. \tag{2}$$

If data are available for single-year age groups, $k(x)$ may be estimated by

$$\overset{*}{k}(x) = \ln(M(x,1)) - \ln(M(x-1,1)), \tag{3}$$

where $M(x,a)$ is the central death rate from age x to $x+a$. Note that, in Eq. (3), mortality data from exact age $x-1$ to $x+1$ are used for estimating $k(x)$.

The empirical analysis of old-age mortality is complicated by two defects often found in recorded data. One defect is the small number of persons and deaths at very old ages, introducing an unavoidably large stochastic variation in death rates. Values of $\overset{*}{k}(x)$ estimated using Eq. (3), therefore, need to be smoothed to detect underlying patterns of its systematic variations. We smooth the sequence of $\ln(M(x,1))$ first by taking moving averages of five successive values, use Eq. (3) to compute $\overset{*}{k}(x)$ from the graduated sequence of $\ln(M(x,1))$, then smooth the sequence of $\overset{*}{k}(x)$ by taking weighted averages of nine successive values, the weight being distributed triangularly over nine values. Namely, $k(x)$ is estimated by

$$\hat{k}(x) = \sum_{n=-4}^{4} \frac{(5-|n|)}{25} \cdot \overset{*}{k}(x+n). \tag{4}$$

It should be noted that this method, like many other graduation procedures, tend to flatten resulting patterns to some extent, by lowering peaks and raising troughs.

Although this method consists of two steps, i.e., smoothing of $\ln(M(x,1))$ and that of $\overset{*}{k}(x)$, the second step is more crucial than the first step. The two-step procedure and smoothing using the second step only produce similar $k(x)$ curves, though the inclusion of the preliminary (first-step) smoothing of $\ln(M(x,1))$ deletes small wiggles.

Another widely-observed defect of old-age mortality data is a strong tendency toward age misreporting for very old persons, both in the ages of the living as

TABLE 1
List of the Populations Studied

Country	Year	The highest age for which data are available
Australia	1970–72	99
Austria	1960–62	97
France	1962,68,74	99
Federal Republic of Germany	1962,68,74	94
German Democratic Republic	1976	98
Germany	1910–11	100
Hungary	1975	84
Japan	1970	99
Sweden	1973–77	99
Taiwan	1931–35	95

recorded in censuses, and in the ages of decedents in records of death.[2] Only when age is determined from a long established register of persons, or when age can be confirmed from the date of birth listed on an identity card each individual carries, can reliable death rates by single years of age be obtained for very old ages.

Taking this problem into consideration, we have selected our study populations. In order to obtain relatively accurate single-year data on age at death and age of persons, preference was given to populations that have long histories of vital registration, those with high levels of literacy and education, and those with cultural backgrounds that are in favor of accurate age identification. We also attempted to include a few populations with relatively high levels of mortality, and introduce some cultural and regional diversities among study populations. Data collection was not so easy as initially expected, because a number of countries tabulate mortality data by single year of age only up to a certain age that is not sufficiently old (e.g., 84) and pool mortality data for older ages together (e.g., 85 and over).

Ten populations listed in Table 1 have finally been selected. Eight of the selected sets of data are for developed nations in the 1960s and 1970s, and the other two sets of data, for Taiwan, 1931–35, and for Germany, 1910–11, represent relatively high mortality.[3] For France and West Germany, three different periods are included for an investigation of cohort effects on the observed age patterns.

RESULTS

Logarithms of death rates from age x to $x+1$, $\ln(M(x,1))$, are plotted for ages 55 and over for males and females of the study populations. $M(x,1)$ is calculated by dividing the number of deaths between age x to $x+1$ in a year by the mid-year population between age x to $x+1$. $M(x,1)$ is accepted as an approximation of $\mu(x+0.5)$.[4]

[2]See for example, Horiuchi and Coale (1982) and Coale and Kisker (1986).

[3]Data in the 1980s are not included here because data collection for this research project was conducted in the late 1970s.

[4]It can be shown that if mortality increases exponentially at a rate k over the interval from x to $x+1$ in a stationary population, then

$$M(x,1) \simeq \mu(x+0.5)(1+k^2/24).$$

If $k = 0.1$, $M(x,1) \simeq \mu(x+0.5)(1.0004)$.

As illustrated in Figure 1, plotted points followed fairly straight lines. In most of the study populations, R^2 obtained from ordinary least squares regression of $\ln(\mu(x))$ on age between 55 and 95 is over 0.99.

The apparent linearity, however, does not necessarily suggest the lack of substantial variations in $k(x)$. Graduated values of $k(x)$ are presented in Figure 2. In all of the female populations, $k(x)$ does not remain constant, but changes systematically. It rises in younger old ages, reaches a peak, then declines in older old ages, resulting in a bell-shaped curve.

A closer look at Figure 2 reveals that the bell-shaped curves in the eight low-mortality female populations follow similar trajectories: the age at which $k(x)$ starts to rise is around 55, the peak is about age 75, and the value of $k(x)$ at its peak is close to 0.12. The curves are fairly symmetric around the peak. The similarity of those curves is clearly seen when the eight curves are plotted together in Figure 3.

Bell-shaped curves in two female populations with relatively high mortality, Taiwan, 1931–35, and Germany, 1910, are somewhat different from the others. Their curves have lower peaks at earlier ages, and spread over wider age ranges than those in the other populations.

Male populations do not uniformly reveal such bell-shaped patterns. Instead, fluctuating patterns of $k(x)$ are found for such countries as the Federal Republic of Germany, the German Democratic Republic, Austria and France, all of which were deeply involved in World War I. A closer look reveals that the timing of those fluctuations coincide with cohorts, as illustrated later in Figure 4. It seems that those $k(x)$ curves for males that might be similar to the female curves have been confounded with cohort mortality variations that reflect long-term impacts of World War I upon the health of its survivors. Since we have analyzed male $k(x)$ patterns elsewhere in order to test this hypothesis (Horiuchi, 1983), detailed results for males are not presented here.

DISCUSSION I: VALIDITY OF RESULTS

Before discussing implications of our results, the validity of the findings needs to be carefully examined. The following three questions might be raised:

1. Is the bell-shaped pattern attributable to *cohort* variations in mortality rather than *age* variations?
2. Is the shape of the curve an artifact of the smoothing procedure?
3. Is the pattern simply a reflection of some systematic errors in data?

First, if the bell-shaped curve shifts over time with cohorts, the pattern should be interpreted as representing cohort differences in mortality. Figures 4 and 5 show patterns of $k(x)$ in 1962, 1968 and 1974 for males and females, respectively, in France and the Federal Republic of Germany. The troughs and peaks of $k(x)$ curves for males in Figure 4 move with cohorts. The birth cohort of 1897 in France and that of 1894 in the Federal Republic of Germany are always at troughs. For females, however, curves observed in different years are similar to each other and almost no cohort effects are noticeable in Figure 5. Comparison of Figures 4 and 5 gives some support to the view that the female bell-shaped pattern reflects age variations rather than cohort variations of mortality.

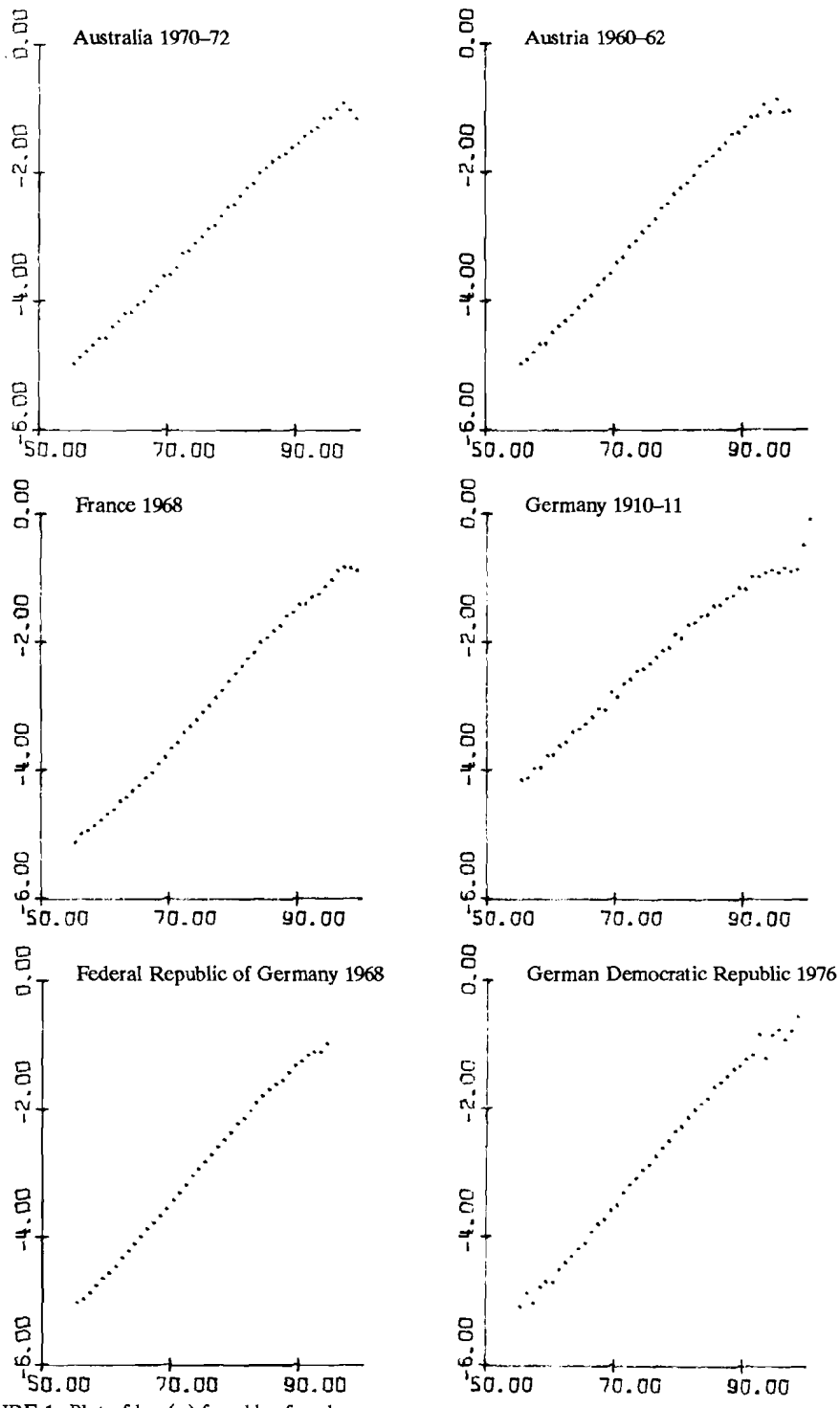

FIGURE 1. Plot of $\ln \mu(x)$ for older females.

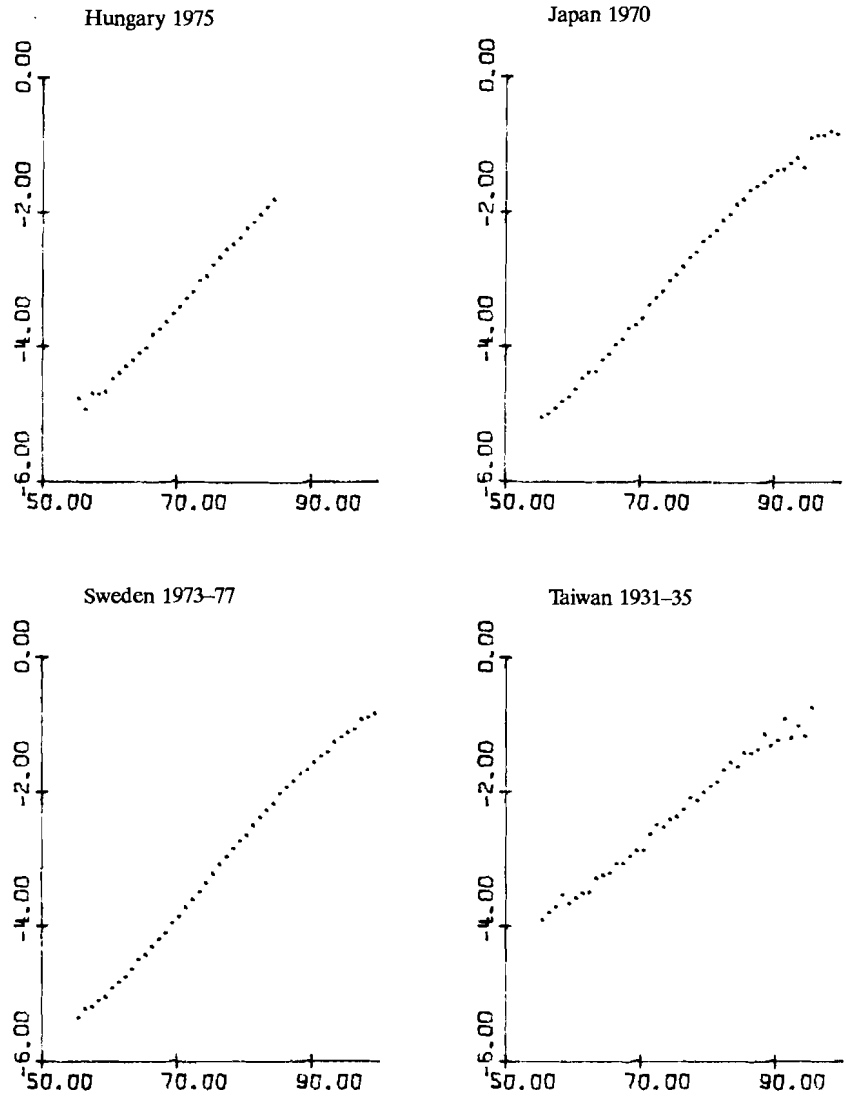

FIGURE 1. (*Continued*).

Secondly, in order to test if the bell-shaped pattern is a statistical artifact of our method of graduation, we have estimated the $k(x)$ function in two ways that are different from our original procedure and compared the results. The first method uses the $l(x)$ function in the abridged life table. An average rate of mortality change from age $x-5$ to $x+5$ is calculated from $l(x-5)$, $l(x)$ and $l(x+5)$ as

$$k(x) \simeq 0.2 \ln \frac{\ln(l(x+5)/l(x))}{\ln(l(x)/l(x-5))}. \tag{5}$$

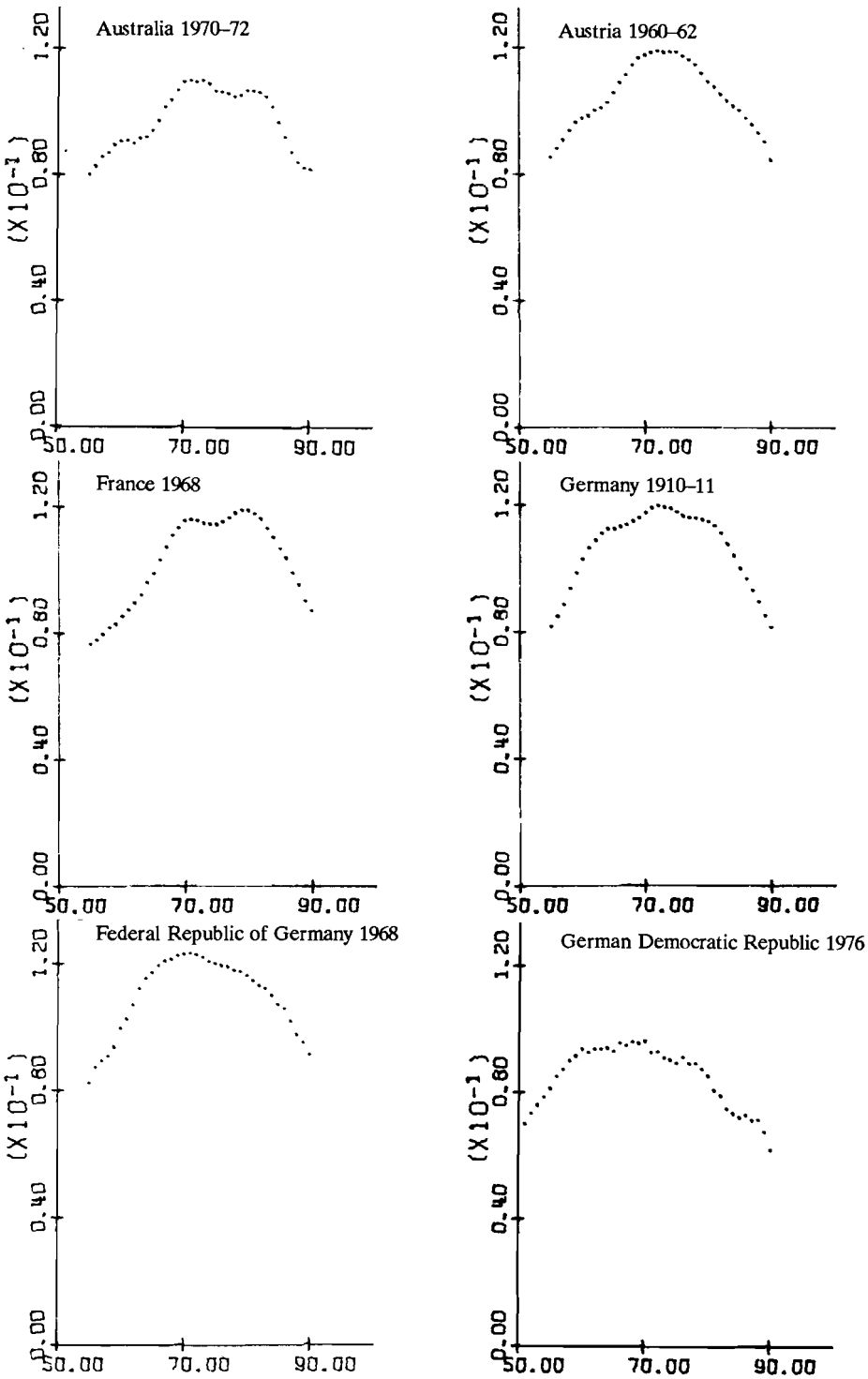

FIGURE 2. Plot of $k(x)$ for older females.

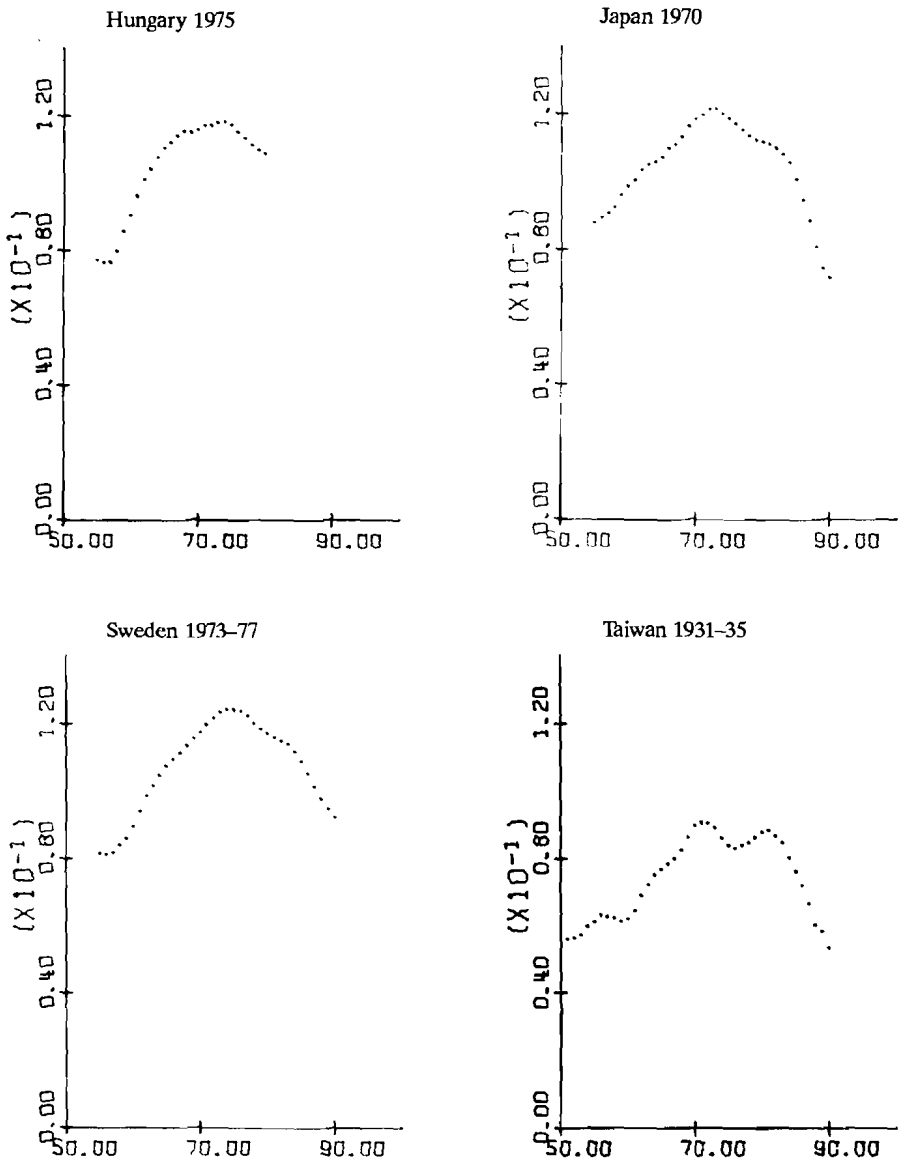

FIGURE 2. (*Continued*).

The other method uses central death rates by five-year age-groups. Namely, $k(x)$ is approximated by the proportional increase in the death rate from age group $(x-5,x)$ to the next one $(x,x+5)$:

$$k(x) \simeq \frac{\ln(M(x,5)) - \ln(M(x-5,5))}{5}. \tag{6}$$

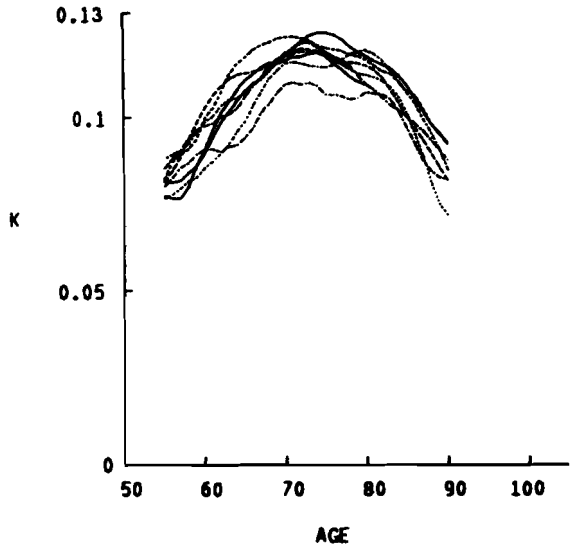

FIGURE 3. Plot of $k(x)$ for older females in eight selected populations[a]

As illustrated for Swedish females in Figure 6, the patterns of $k(x)$ estimated using these simpler methods are nearly identical to our original results. In addition, if the bell-shaped pattern is an artifact of the smoothing method, such curves could be found for males as well as females. However, the bell-shaped pattern was not observed for the majority of the male study populations.

Finally, consideration should be given to the possibility that a substantial amount of systematic age misreporting might distort observed $k(x)$ curves. Data sets used in this study may not be completely accurate. Age-specific death rates for Taiwan, 1931–35, and Germany, 1910–11, are more erratic than those in the other populations and show some symptoms of digit preference, suggesting that data quality for those populations may not be as good as the others. This applies to Australia, 1970–72, though to a lesser extent.

However, most of the study populations are nearly completely literate and well-educated, and have relatively long histories of vital registrations. In addition, our data set includes Sweden, which is known as one of the providers of the best quality of demographic data for an extended period of time, and Japan, where people are highly concerned with accurate identification of ages of older persons because special celebrations of their long lives are held at ages 60 (*kanreki*-celebration), 70 (*koki*), 77 (*kiju*) and 88 (*beiju*). Furthermore, it seems unlikely that all of those female populations with different cultures have the same pattern of age misreporting that result in the bell-shaped curve of $k(x)$.

Kannisto (1988) examined the quality of mortality data on centenarians in seventeen countries and selected data from thirteen countries as reliable. Although the ages focused in his study are older than those in this study, it does not seem unreasonable to expect good quality of data on old persons in populations that have

[a]All study populations in figure 2 except Germany 1910–11 and Taiwan 1931–35.

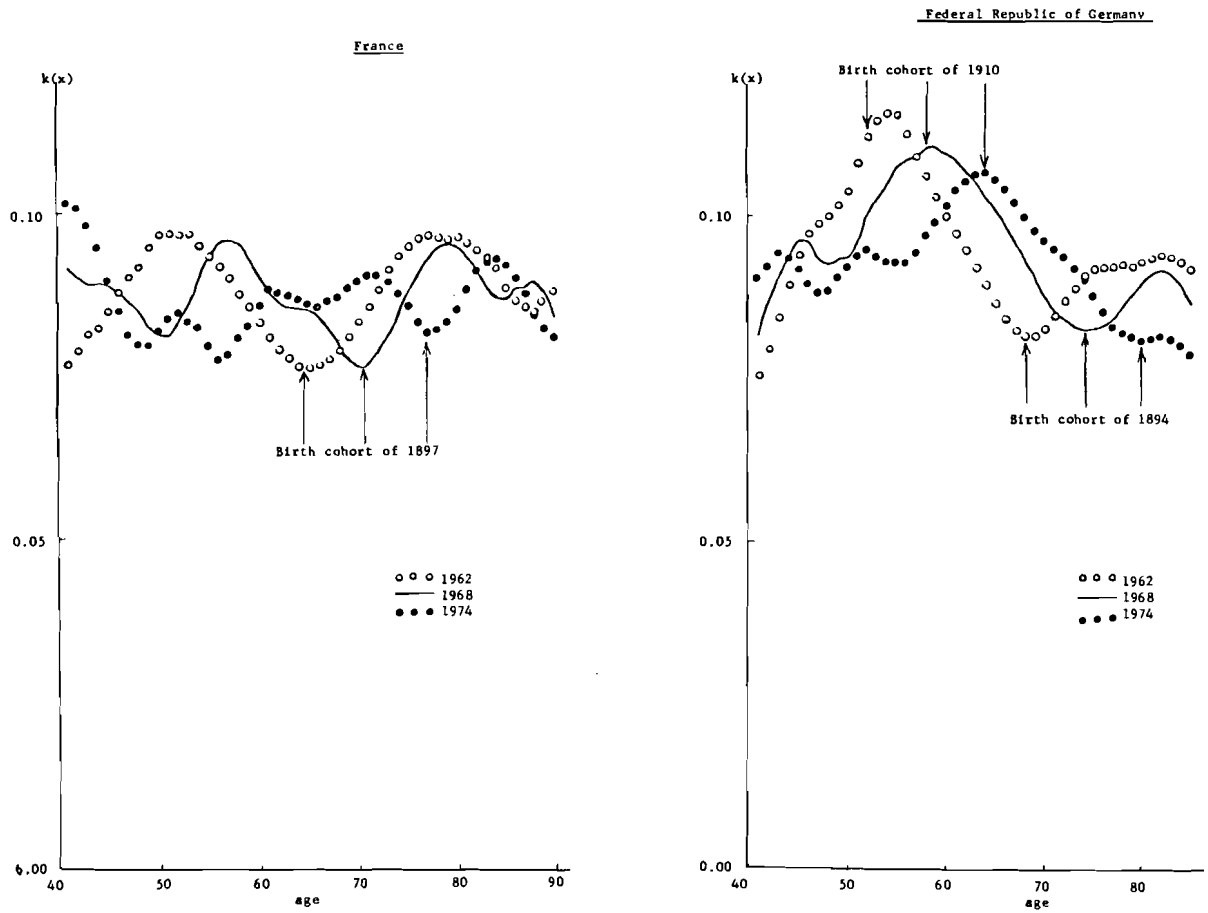

FIGURE 4. Plot of $k(x)$ for French Males and German Males.

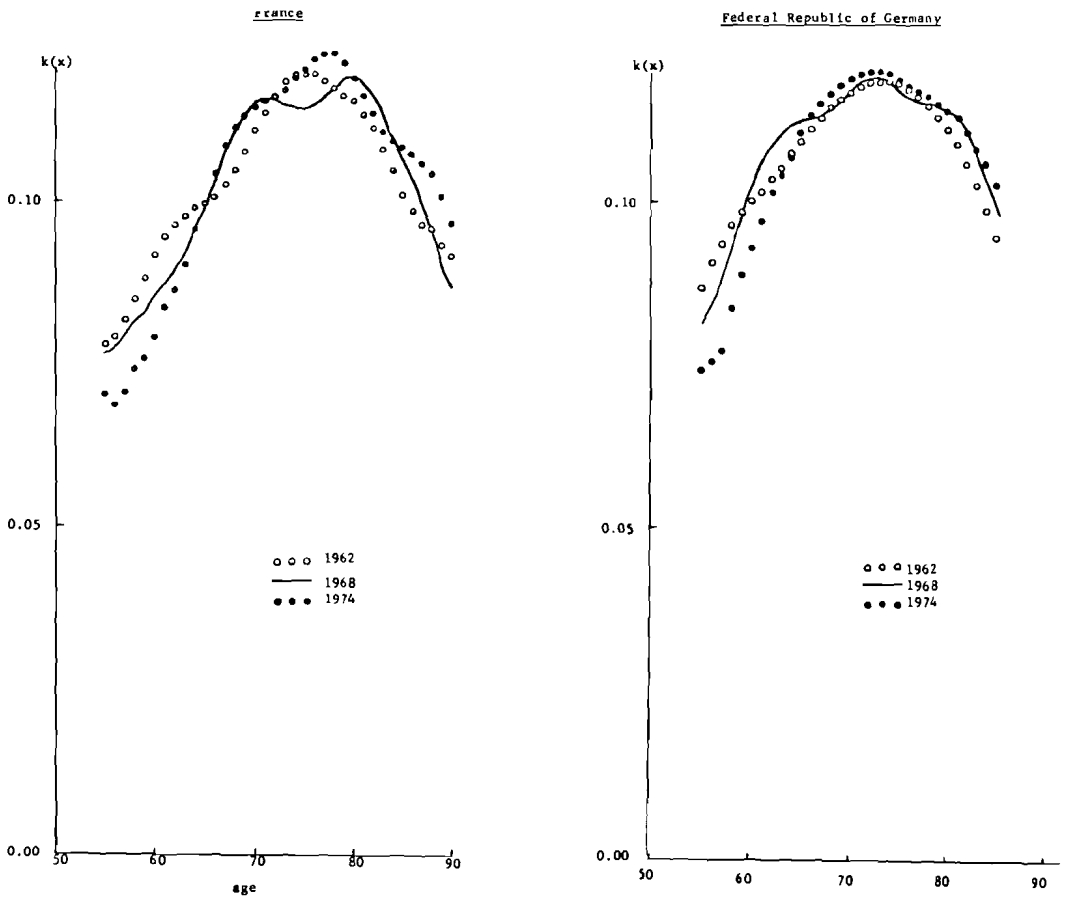

FIGURE 5. Plot of $k(x)$ for French Females and German Females.

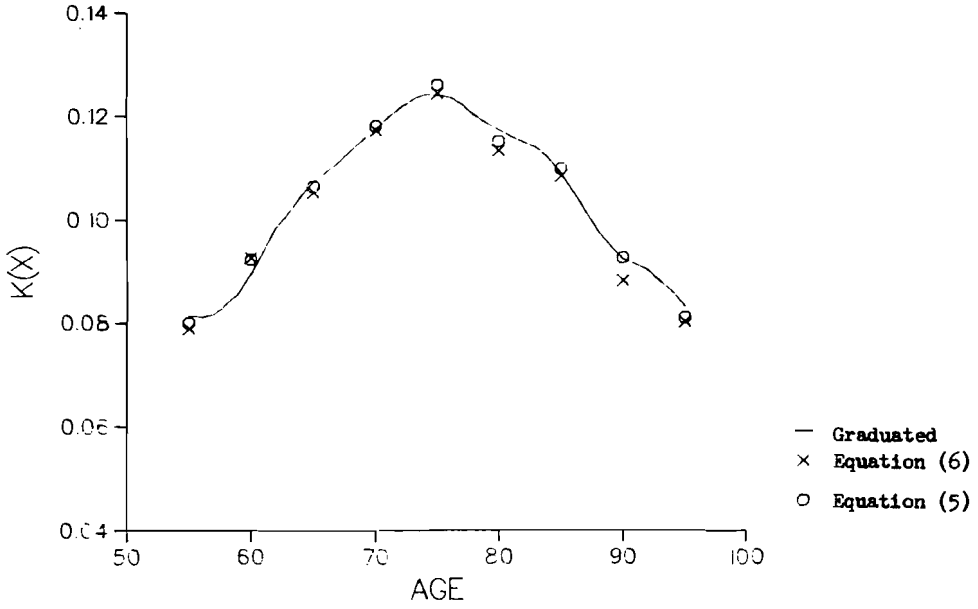

FIGURE 6. Values of $k(x)$ calculated using three different procedures: Swedish females, 1973–1977.

reliable records on centenarians. Kannisto's original 17-country data set includes six countries in our data set (Australia, Austria, Federal Republic of Germany, France, Japan and Sweden), all of which were assessed by him as reliable. Although the periods covered for the six countries are not identical between the two studies, they overlap with each other except for Austria (1960–62 in this study and 1967–83 in Kannisto's study.)

In summary, the observed age patterns of $k(x)$ for older women do not seem attributable to mortality variations of cohort origin, the method of graduation employed here, or inaccurately reported ages. The bell-shaped curves, therefore, are considered to reflect some underlying age structures of mortality.

DISCUSSION II: INTERPRETATION OF RESULTS

Several mathematical equations have been developed to represent age variations in mortality at middle and old ages. In particular, the following models by Gompertz, Makeham, Perks and Beard are well-known (Beard, 1963).

$$\text{Gompertz:} \qquad \mu(x) = Be^{ux}, \tag{7}$$

$$\text{Makeham:} \qquad \mu(x) = A + Be^{ux}, \tag{8}$$

$$\text{Perks:} \qquad \mu(x) = \frac{A + Be^{ux}}{1 + Ce^{ux}}, \tag{9}$$

$$\text{Beard:} \qquad \mu(x) = \frac{Be^{ux}}{1 + Ce^{ux}}, \tag{10}$$

where A, B, C and u are parameters taking positive values.[5]

Gompertz attributed the exponential increase of death rate in Equation (7) to the physiological deterioration that proceeds with age. He indicated that chance and degeneration were the basic two causes of death. Deaths by chance seem to include deaths by accident, suicide and homicide.

Since Gompertz did not include the chance factor in his equation, Makeham added an age-independent term for the risk of death by chance, i.e., A in Eq. (8), to the Gompertz model.

In some populations, however, observed death rates tend to be higher at young ages and lower at extremely old ages than the death rates estimated by fitting the Gompertz or Makeham equation to data. To overcome this problem, Perks proposed a logistic equation (9). Beard derived another logistic function (10) by modeling the physiological process of aging. Usually, all of these four equations fit data remarkably well.

However, these models imply very different $k(x)$ functions, as shown below.

$$\text{Gompertz:} \qquad k(x) = u, \tag{11}$$

$$\text{Makeham:} \qquad k(x) = \frac{u}{1 + De^{-ux}}, \tag{12}$$

$$\text{where} \quad D = A/B,$$

$$\text{Perks:} \qquad k(x) = \frac{u}{1 + De^{-ux}} - \frac{u}{1 + Ee^{-ux}} \tag{13}$$

$$\text{where} \quad D = A/B \quad \text{and} \quad E = 1/C,$$

$$\text{Beard:} \qquad k(x) = \frac{u}{1 + Ce^{ux}}. \tag{14}$$

The constancy of $k(x)$ is implied by the Gompertz model. Both the Makeham model and the Beard model imply that $k(x)$ follows logistic curves. A significant difference between the two logistic functions is that the Makeham logistic curve (12) rises monotonically from zero to u, whereas the Beard logistic curve (14) declines monotonically from u to zero.

According to the Perks model (13), $k(x)$ is the vertical difference of two parallel logistic curves. Both of the curves rise from zero to u, and their distance along the X-axis is always $\ln(B/AC)/u$. It should be noted that the vertical difference of two parallel logistic curves is a bell-shaped function that appears similar to the normal distribution.[6] In the case of Eq. (13), the function reaches its peak $u/(1+$

[5]The original Perks equation includes another exponential term in the denominator. Beard (1963), in discussing old-age mortality, excluded the term from the Perks equation, because the term was for approximating *child mortality*. In the present paper, we follow Beard's presentation of the Perks equation.

[6]The Perks model implies at least three bell-shaped functional relationships. Since this may cause some confusions, those relationships should be carefully distinguished. They are: a) $d\mu(x)/dx$ is a quadratic function of $\mu(x)$; b) $d\mu(x)/dx$ is a bell-shaped function of x that appears similar to the normal distribution curve; c) $d\ln(\mu(x))/dx$ (i.e., $k(x)$) is also a bell-shaped function of x with tails approaching zero at both ends. All of these functions have absolute maximums and are symmetric around their peaks.

The first function is given by

$$\frac{d\mu(x)}{dx} = \frac{u}{F - A}(\mu(x) - A)(F - \mu(x)), \qquad \text{where} \quad F = B/C.$$

TABLE 2
Results of Non-Linear Least Squares Regression Analysis for Swedish Females Aged 55–95, 1973–77

Parameters	Gompertz Model	Makeham Model	Perks Model	Beard Model
A	N.A.	.00073	.00239	N.A.
B	.00399	.00355	.00230	.00392
C	N.A.	N.A.	.00367	.00108
u	.11180	.11545	.13876	.11367
R^2 of $\ln\mu(x)$	.9980	.9983	.9998	.9981
R^2 of $k(x)$[a]	.0000	.2458	.9609	.1257

[a] The regression analysis was conducted to maximize R^2 of $\ln(\mu(x))$, but not R^2 of $k(x)$.

$\sqrt{AC/B}) - u/(1 + \sqrt{B/AC})$ at $x = \ln(\sqrt{A/BC})/u$, and the function is symmetric around the peak. (See Appendix A for more details.)

Therefore, among the four models, only the Perks model is consistent with the bell-shaped $k(x)$ patterns found for older women in the study populations.[7] This is illustrated for Swedish females in Table 2 and Figure 7. Table 2 presents parameter values of the four models that are estimated by fitting them to death rates for Swedish females. A non-linear least squares procedure (Sadler, 1975) was employed for regression of $\ln(\mu(x))$ for ages between 55 to 95. All of the models fit the data extremely well. As shown in the second row from the bottom of Table 2, R^2 for each model is above 0.998. Note that in those regression analyses, x is given as age measured with 55 as origin, i.e., the difference between the reported age and 55.

We have estimated the $k(x)$ sequence for the four models by substituting the estimated parameter values in Table 2 into the equations (11) to (14). Figure 7 compares those estimated $k(x)$ sequences with the observed one. The sequence of $k(x)$'s derived by fitting the Perks model (9) to the logarithms of age-specific death rates agrees fairly well with the smoothed sequence of observed $k(x)$ values, except for some departures at both ends. However, $k(x)$ values derived from the other models deviate substantially from observed values, in spite of the fact that those models fit the logarithms of observed death rates remarkably well.[8]

The compatibility of Eq. (13) with empirical data, however, does not necessarily imply that the proportional increase in the age-specific risk of death of *individuals* is well-approximated by the equation. It has been repeatedly pointed out that the age pattern of death rates for a population and the age pattern of mortality

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This function is a parabolic curve centering at the peak $u(F-A)/4$ at $\mu(x) = (A+F)/2$. The second function is represented as

$$\frac{d\mu(x)}{dx} = \frac{uC(F-A)e^{ux}}{(1+Ce^{ux})^2},$$

which reaches the maximum $u(F-A)/4$ at $x = -\ln C/u$. The third function is Eq. (13) in the text. It should be noted that if $\mu(x)$ is a logistic function, a) and b) always hold but c) does not hold for some kinds of logistic function.

[7] Our results seem to indicate the usefulness of a general strategy in data analysis: if two or more competing models appear to fit data well, the analyst may take the derivatives of the models, conduct numerical differentiation of the data, and test if the agreement between the models and data still holds.

[8] Because of the bell-shaped pattern of $k(x)$, the functional form $\mu(x) = B\exp(xu(x))$, where $u(x) = Ex^2 + Fx + G$, might fit the data well. However, unlike the Perks model, no theoretical justification could be found so far for this functional form.

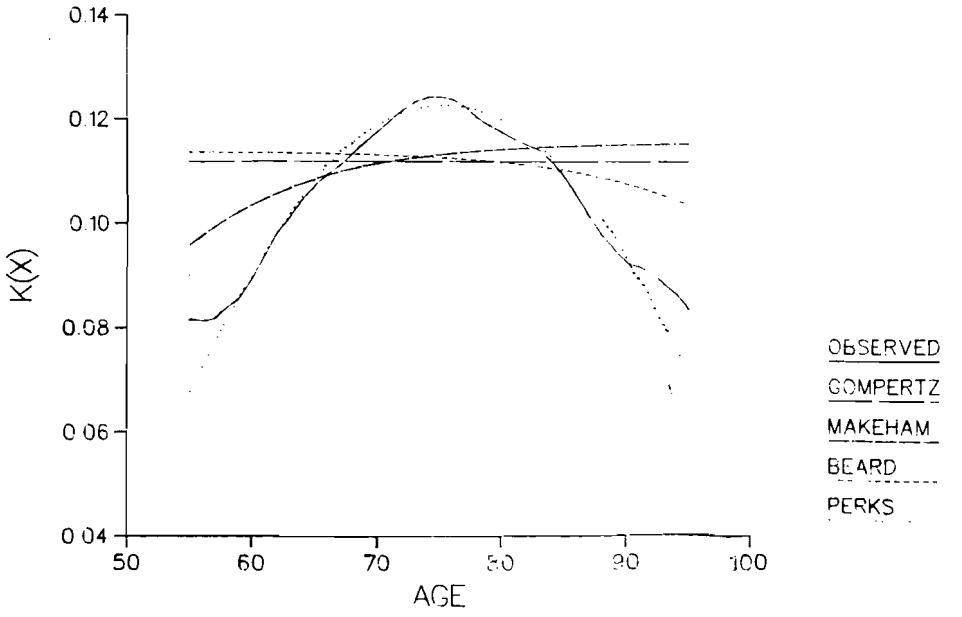

FIGURE 7. The sequence of $k(x)$ values estimated on the basis of different models: Swedish females, 1973–1977.

risk for individual members of the population could be substantially different because weaker subgroups tend to be reduced faster (Beard, 1963; Redington, 1969; Nam, Weatherby and Ockay, 1978; Shepherd and Zeckhauser, 1980 and 1982; Manton, Poss and Wing, 1979; Vaupel, Manton and Stallard, 1979; Keyfitz and Littman, 1980; Bourbeau and Legare, 1981; Manton and Stallard, 1981; Manton, Stallard and Vaupel, 1981).

The relationship between $k(x)$ for individuals and that for population are represented as follows. Suppose that z is a measure of frailty, $\mu_z(x)$ and $k_z(x)$ are the instantaneous death rate at exact age x and the rate of its change, respectively, for those with frailty z. (For a detailed definition of frailty, see Vaupel, Manton and Stallard, 1979.) It can be shown that

$$\mu_T(x) - k_T(x) = \int_0^\infty d_z(x)\{\mu_z(x) - k_z(x)\}\, dz, \tag{15}$$

where $d_z(x)$ is the proportion of all deaths at age x that occur to those with frailty z, i.e., $\int_0^\infty d_z(x)dz = 1$, and the subscript T refers to the entire population at a given age. Eq. (15) seems to suggest that the $k(x)$ pattern for individuals and that for populations could be significantly different.

It has previously been shown that, given the gamma-distribution of individual frailty, the age-specific death rate for a population is represented by the Beard logistic equation (10) if individuals follow the Gompertz equation (7), and by the Perks logistic equation (9) if individuals follow the Makeham function (8) (Beard, 1963). The proof is recapitulated in Appendix B. Therefore, the observed $k(x)$ patterns are consistent with the following model: the force of mortality at age x for an

individual with frailty z is given by

$$\mu_z(x) = A + ze^{ux}. \tag{16}$$

where z is a gamma-distributed variable, replacing the constant B in Eq. (8).

The equation (16) has two components: a constant A, and an age-dependent term ze^{ux}. The second term implies an exponential relationship, which has been derived from several mathematical models of aging and mortality (Strehler and Mildvan, 1960; Brillinger, 1961; Beard, 1963; Brown and Forbes, 1974; Abernethy, 1979). However, some types of mortality risk are relatively independent of the physical deterioration that proceeds with age. Although these kinds of risk may also vary with age, their age variations are considered to be significantly smaller than age variations in mortality risk related to degenerative aging, so that the risk may be approximated by the constant A in Eq. (16).

The Makeham chance factor A in Eq. (16) is necessary for the $k(x)$ curve to be bell-shaped. Not all monotonically increasing logistic $\mu(x)$ functions result in bell-shaped $k(x)$ patterns. If the factor A is excluded from the individual-level equation (16), then the resulting aggregate-level $\mu(x)$ function is Eq. (10), a logistic curve rising from zero to B/C, which does *not* lead to a bell-shaped $k(x)$ pattern.

The assumption of gamma-distributed individual frailty, which has been adopted in some mortality studies (Beard, 1963; Vaupel, Manton, and Stallard, 1979), does not seem unreasonable. As stated by Vaupel et al. (1979), the gamma-distribution has several advantages in modelling frailty. It is a very flexible function, taking on a variety of shapes; if the individual risk is expressed as the sum of two age-dependent terms, one being unrelated to frailty and the other being the product of z and an age-dependent function, and if the frailty is gamma-distributed at birth of a cohort, then the frailty is expected to be gamma-distributed within the cohort *at any age*, as shown in Appendix C; the gamma-distribution has been widely used for modelling individual differences.

Eq. (16) provides an intuitive interpretation of the bell shape as well as a mathematical derivation of the Perks model. The equation suggests two opposite effects on $k(x)$. On the one hand, the proportion of mortality risk that is chance-related becomes negligibly small for older persons. Therefore, $k(x)$ for an individual increases with age and approaches u, the rate of increase of the degeneration-related risk. On the other hand, the selective survival in a heterogeneous population makes the population composition less frail at older ages, thereby slowing down the mortality increase with age. The observed bell-shaped patterns seem to suggest that the first effect prevails at younger old ages and the second effect at older old ages.

The explanation of the observed $k(x)$ patterns in terms of Eq. (16), however, is not within limitations. First, although our data are *period* death rates in populations with *changing* mortality, the Perks model is derived from Eq. (16) for *cohort* death rates, or equivalently, period death rates in populations with *constant* mortality. Taking into consideration mortality changes over time and age variations in chance-related risk, Eq. (16) may be rewritten as

$$\mu_z(x,t) = A(x,t) + y(t)ze^{ux}, \tag{17}$$

where t denotes time and $y(t)$ is considered to reflect changes in such mortality determinants as medical technology, public health programmes, the standard of living and environmental contamination.

Although the Perks equation cannot be derived from Eq. (17), the observed $k(x)$ patterns are bell-shaped probably because age variations in mortality due to physiological degeneration are considerably greater than period variations. However, if there were extremely large period variations in the past, such as those due to the world wars, then the $k(x)$ pattern may not appear bell-shaped, as seen in some of the male populations.

Secondly, the difference between the two populations with relatively high mortality (Germany, 1910–11, and Taiwan, 1931–35) and the other eight populations may suggest that the bell shape tends to be narrower with a higher peak at an older age in populations with lower mortality. Eq. (16), however, does not provide a framework for analyzing temporal changes of the $k(x)$ pattern.

Thirdly, it may not be valid to assume that the value of the frailty parameter z of an individual remains constant throughout his life. The individual may become "debilitated" from severe diseases and injuries, malnutrition, unhealthy life styles and extended exposure to contaminated environments (Vaupel, Yashin and Manton, 1988). Such debilitation effects, however, are not taken into consideration in Eq. (16).

Finally, at least one alternative interpretation of the bell-shaped $k(x)$ patterns still remains possible. The above discussion is based on the speculation that there are large individual differences with respect to the susceptibility to death. However, if the individual differences are small, the bell-shaped pattern of $k(x)$ peaking around age 75 may simply be a reflection of "pace of aging" of individuals, suggesting that the pace of physiological aging is accelerating in younger old ages, say 55 to 75, and decelerating in older old ages, i.e., over 75.[9] This hypothesis should be investigated in the future from more physiological and geriatrical viewpoints.

In spite of these limitations, the combination of the Makeham equation for individuals and the gamma distributed frailty seems to provide a plausible explanation of the bell-shaped pattern, capturing major sources of variation in $k(x)$, though probably oversimplifying minor ones. In this study, we have found an interesting female $k(x)$ pattern, proposed an explanation for the pattern, and shown the usefulness of the measure in mortality research. Its usefulness should be explored further, probably with cause-specific mortality data as well as long-time series of age-specific death rates.

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[9]Interestingly, a study of old-age mortality in female populations by Weatherby et al. (1983) has also shown that the age of 75 is a cut-off point of old-age mortality pattern. They say "improvements in levels of economic development, distributional inequality, and basic primary health care tended to reduce mortality primarily before the age of 75. Reduction in mortality above the age of 75 may occur as improvements in medical technology make it possible to prolong the lives of persons with chronic illness."

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APPENDIX A: CHARACTERISTICS OF THE PERKS $k(x)$ FUNCTION

In the Perks model, the instantaneous mortality rate at exact age x is given by

$$\mu(x) = \frac{A + Be^{ux}}{1 + Ce^{ux}}, \tag{A.1}$$

where A, B, C and u are positive parameters. This equation represents a logistic curve bounded by A and B/C, that is, $\mu(x)$ approaches A as x decreases and $\mu(x)$

approaches B/C as x increases. Since adult mortality increases with age, we set $B > AC$.

The rate of mortality change with age is given by

$$k(x) = \frac{d\ln(\mu(x))}{dx} = \frac{u}{1+De^{-ux}} - \frac{u}{1+Ee^{-ux}}, \qquad \text{(A.2)}$$

where $D = A/B$ and $E = 1/C$.

Let $g_1(x) = u/(1+De^{-ux})$ and $g_2(x) = u/(1+Ee^{-ux})$. The $k(x)$ function is the difference of two logistic functions $g_1(x)$ and $g_2(x)$, both of which rise from zero to u, and have the same parameter u that determines the steepness of the logistic growth. The two logistic curves are parallel along the X-axis since

$$\begin{aligned} g_1(x) &= u/(1+De^{-ux}) = u/(1+Ee^{-ux-\ln E+\ln D}) \\ &= g_2(x+G), \qquad \text{(A.3)} \\ \text{where} \quad & G = (\ln E - \ln D)/u. \end{aligned}$$

Therefore, their distance along the X-axis is constantly G.

The $k(x)$ function in (A.2) has several important characteristics. First, it is positive for all x because $B > AC$ implies $E > D$. Second, $k(x)$ approaches zero as x approaches the positive or negative infinity.

Thirdly, the function has the absolute maximum. By differentiating (A.2) with respect to x, we get

$$\frac{dk(x)}{dx} = \frac{u^2e^{-3ux}DE(E-D)}{(1+De^{-ux})^2(1+Ee^{-ux})^2}\left(1-\frac{e^{2ux}}{DE}\right) \qquad \text{(A.4)}$$

so that the derivative of $k(x)$ is positive when $x < \ln(\sqrt{DE})/u$, zero when $x = \ln(\sqrt{DE})/u$ and negative when $x > \ln(\sqrt{DE})/u$. Substituting $x = \ln(\sqrt{DE})/u$ into (A.2), we get the maximum value of $k(x)$, that is, $u/(1+\sqrt{D/E}) - u/(1+\sqrt{E/D})$.

Finally, the function is symmetrical around the peak at $x = \ln(\sqrt{DE})/u$ since

$$\begin{aligned} k\left(\frac{\ln(\sqrt{DE})}{u} - y\right) &= \frac{u}{1+e^{uy}\sqrt{D/E}} - \frac{u}{1+e^{uy}\sqrt{E/D}} \\ &= \left\{u - \frac{u}{1+e^{uy}\sqrt{E/D}}\right\} - \left\{u - \frac{u}{1+e^{uy}\sqrt{D/E}}\right\} \\ &= \frac{u}{1+e^{-uy}\sqrt{D/E}} - \frac{u}{1+e^{-uy}\sqrt{E/D}} \\ &= k\left(\frac{\ln(\sqrt{DE})}{u} + y\right). \qquad \text{(A.5)} \end{aligned}$$

APPENDIX B: DERIVATION OF THE PERKS MODEL

PROPOSITION. Suppose that the instantaneous death rate at exact age x for persons with frailty z is given by

$$\mu_z(x) = A + ze^{ux} \tag{B.1}$$

and z at $x = 0$ is gamma-distributed:

$$f(z) = \frac{b^a z^{a-1}}{\Gamma(a)} e^{-bz}, \tag{B.2}$$

where $f(z)$ is the p.d.f. of z, $\Gamma(a)$ is the gamma function of a, and a and b are parameters of the gamma distribution. Then the death rate at age x for the population is represented by

$$\mu(x) = \frac{A + Be^{ux}}{1 + Ce^{ux}}, \tag{B.3}$$

where $B = (A + au)/(ub - 1)$ and $C = 1/(ub - 1)$.

PROOF. The proportion of persons with frailty z who survive up to age x is

$$l_z(x) = \exp\left\{-\int_0^x \mu_z(y)\,dy\right\} = e^{-Ax}\exp\left\{-\frac{z}{u}(e^{ux} - 1)\right\}. \tag{B.4}$$

The instantaneous death rate at age x for the population is represented as

$$\mu(x) = \frac{\int_0^\infty f(z) l_z(x) \mu_z(x)\,dz}{\int_0^\infty f(z) l_z(x)\,dz} \tag{B.5}$$

Substituting (B.1), (B.2) and (B.4) into (B.5), we have

$$\begin{aligned}
\mu(x) &= A + e^{ux}\frac{\int_0^\infty z^{a-1}\exp\{-z(b + (e^{ux} - 1)/u)\}z\,dz}{\int_0^\infty z^{a-1}\exp\{-z(b + (e^{ux} - 1)/u)\}\,dz} \\
&= A + e^{ux}\frac{a}{b + (e^{ux} - 1)/u} \\
&= \frac{A + \{(A + au)/(ub - 1)\}e^{ux}}{1 + \{1/(ub - 1)\}e^{ux}} \\
&= \frac{A + Be^{ux}}{1 + Ce^{ux}}.
\end{aligned}$$

Q.E.D.

APPENDIX C: GAMMA DISTRIBUTED FRAILTY AMONG SURVIVORS

PROPOSITION: Suppose the instantaneous death rate at age x for a person with frailty z is composed of two age-dependent functions, $A(x)$ and $H(x)$ in the following way:

$$\mu_z(x) = A(x) + zH(x). \tag{C.1}$$

We assume that frailty z at $x = 0$ is gamma-distributed as given by (B.2). Then z is gamma-distributed at any age.

PROOF. It is derived from (C.1) that

$$l_z(x) = \exp\left\{-\int_0^x A(y)dy\right\}\exp\left\{-z\int_0^x H(y)dy\right\}. \tag{C.2}$$

The p.d.f. of z at age x is given by

$$g_x(z) = \frac{f(z)l_z(x)}{\int_0^\infty f(z)l_z(x)dz}. \tag{C.3}$$

By substituting (C.1) and (B.2) into (C.3), we have

$$g_x(z) = \frac{z^{a-1}\exp\{-z\,(b + \int_0^x H(t)dt)\}}{\int_0^\infty z^{a-1}\exp\{-z\,(b + \int_0^x H(t)dt)\}\,dz}.$$

Setting $c = b + \int_0^x H(t)dt$ and $y = cz$ yields:

$$g_x(z) = \frac{z^{a-1}e^{-cz}}{\int_0^\infty z^{a-1}e^{-cz}\,dz} = \frac{c^a z^{a-1}e^{-cz}}{\int_0^\infty y^{a-1}e^{-y}\,dy},$$

which represents the gamma distribution with parameters a and c. Q.E.D.

APPENDIX D: DATA SOURCES

Australia

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Austria

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