# *Encyclopedia of*
# POPULATION


**EDITED BY**

PAUL DEMENY

GEOFFREY MCNICOLL


**VOLUME**

2

I-W

APPENDIX

INDEX


MACMILLAN
REFERENCE
USA™

THOMSON
GALE

New York • Detroit • San Diego • San Francisco • Cleveland • New Haven, Conn. • Waterville, Maine • London • Munich

# MORTALITY, AGE PATTERNS OF

The risk of death varies markedly with age. The death rate is high in the first month after birth, declines during the rest of infancy and childhood, remains low during adolescence and young adulthood, and then rises gradually in middle age and steeply in old age (see Figures 1A and 1B). The decrease and increase in mortality reflect the rise of physiological abilities and disease resistance during child development and their decline during senescence. This basic pattern has been observed for most human populations in different historical eras, for both males and females.

The age pattern of death rates determines the age distribution of the number of deaths and the age trajectory of the number of survivors. Because the death rate declines during childhood and rises later in life, the age distribution of the number of deaths usually has two peaks, one in the first year of life, and the other in old age (Figure 1C). The later peak is usually between 70 and 90 years of age in modern human populations, but it is estimated to have been between 20 and 40 years in Stone Age populations.

The number of individuals who survive from birth to a given age $x$ is a decreasing function of $x$. Usually, the number of survivors plummets during infancy and early childhood, decreases gradually at young adult and middle ages, and then falls steeply at old ages (Figure 1D).

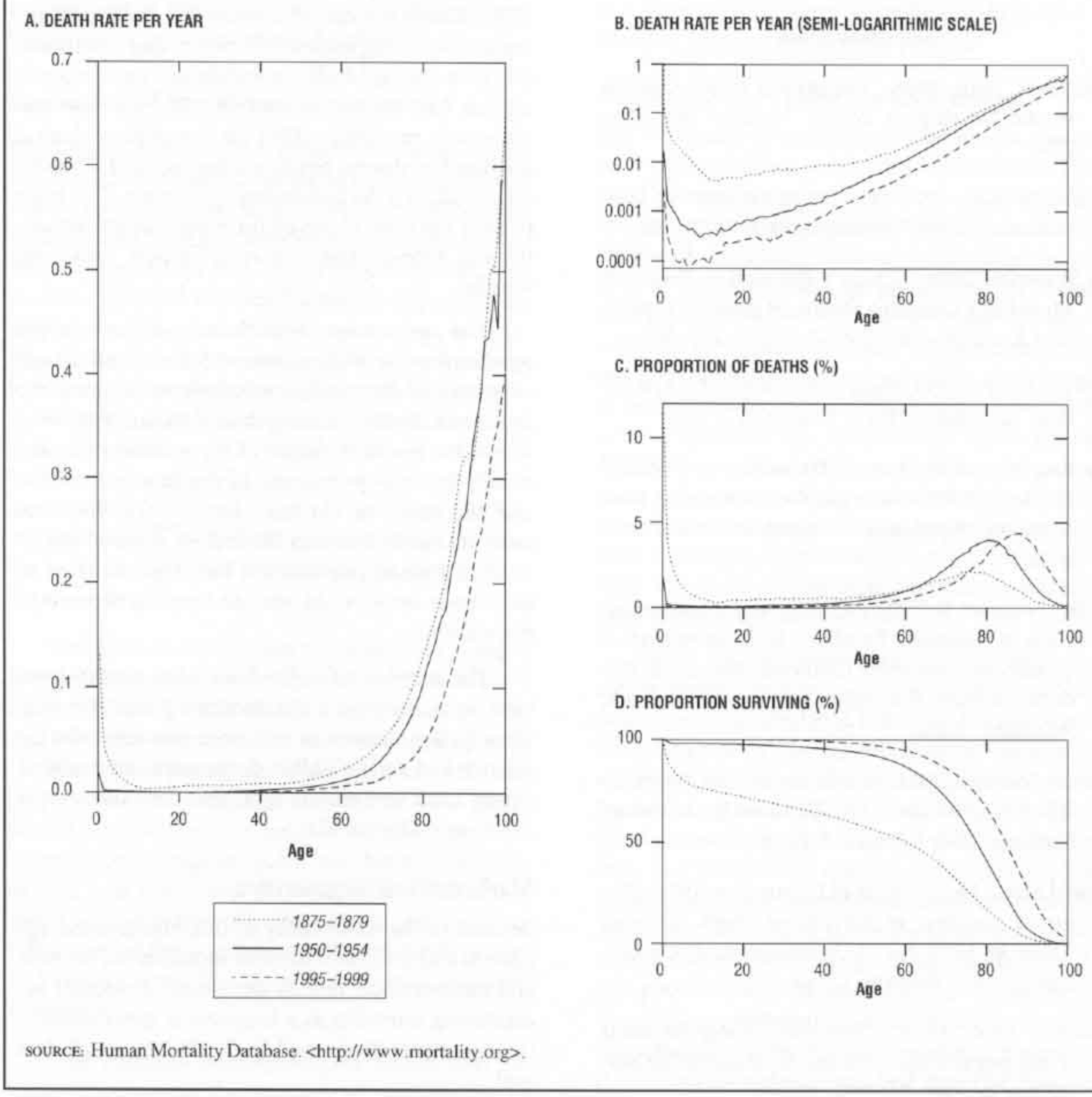
## Mathematical Regularities

Because of the universality of this fundamental age pattern and the smoothness of mortality curves, several mathematical models have been developed for expressing mortality as a function of age. Generally these mathematical models fit observational data well.

Some of these models (including the Thiele model, the Siler model, and the Heligman-Pollard model) cover the entire life span by combining separate components that represent mortality patterns in different stages of life. For example, in the Siler model, the death rate (or force of mortality) at exact age $x$, denoted by $m(x)$, is expressed as the sum of three terms: $m(x) = ge^{-hx} + c + ae^{bx}$ where $g$, $h$, $c$, $a$, and $b$ are parameters of the model. The three components ($ge^{-hx}$, $c$, and $ae^{bx}$) represent mortality decline in childhood, stable mortality during adoles-

**FIGURE 1**

**Age Patterns of Mortality among Swedish Females in Three Periods: 1875–1879, 1950–1954 and 1995–1999**




SOURCE: Human Mortality Database. <http://www.mortality.org>.

cence and young adulthood, and mortality rise at middle and old ages, respectively. The three components can be additively combined because in each life stage one of the three components largely determines the age-specific death rate with only small numerical contributions from the other two terms.

Most other models (including the Gompertz model, the Makeham model, the Weibull model, and different versions of the logistic model) are concerned with describing adult mortality only. The rising mortality curve at middle and old ages appears fairly straight on a semi-logarithmic scale (Figure 1B), suggesting that the death rate increases nearly exponentially with age. This exponential rise was discovered by the British actuary Benjamin Gompertz (1779–1865) in the early nineteenth century. In the Gompertz model, the death rate at exact age $x$ is expressed simply as: $m(x) = ae^{bx}$ where $a$ and $b$

are parameters of the model. The Gompertz model appears as one of the three components of the Siler model.

The parameter *b* of the Gompertz model, the slope of the logarithmic mortality curve, is called the Gompertzian rate of aging. In biodemographic research, the Gompertz model has been applied to mortality data of various species, and the Gompertzian rate of aging is widely used for comparing the pace of senescence among species, and also for studying effects of genetic and environmental factors on senescent processes.

## Mortality Deceleration at Older Ages

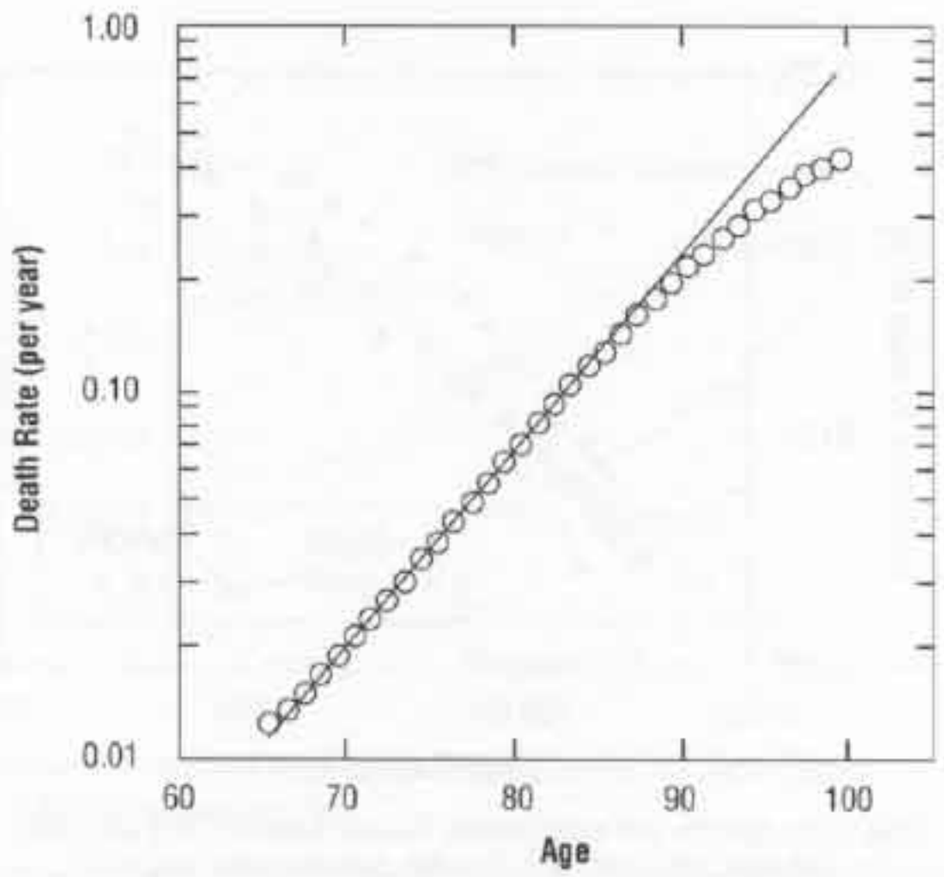
The exponential increase of adult mortality tends to slow at very old ages, as illustrated in Figure 2. Thus the logistic equation, which sets an upper limit to mortality rise, usually fits observed death rates at very old ages more closely than the exponential equation does. This mortality deceleration is observed in most large human populations as well as in several non-human animal species (including fruit flies, earthworms, wasps, and beetles) for which old-age mortality patterns have been examined in detail. Although the slowing-down of mortality increase can be clearly seen in human populations, it is less pronounced than in the non-human species. Some fruit fly populations even exhibit notable age-related *declines* of mortality at very old ages.

In modern human populations, the deceleration can be visually detected in the data for mortality above age 90, but age-specific rates of relative mortality increase (called *life table aging rates*) indicate that the slowing-down actually starts earlier, typically between ages 75 and 80. In populations with lower levels of old-age mortality, the deceleration tends to be delayed to higher ages.

The reason for the mortality deceleration is not fully known. One possible explanation is selective survival. Because less-healthy individuals are more likely to die at younger ages, survivors to older ages tend to have favorable health endowments and/or healthy lifestyles. This selection process could slow down the age-related increase in the death rate in the *population* data. Another possible explanation is that the age-related increase of mortality risk in each *individual* may slow down at old ages for physiological reasons.

**FIGURE 2**

**Age-Specific Death Rates among Women, Ages 65–99, France, 1971–1980**




Note: Circles are death rates per year for single years of age. The line was fitted to death rates between ages 70 and 80 by an ordinary least squares regression.

SOURCE: Human Mortality Database. <http://www.mortality.org>.

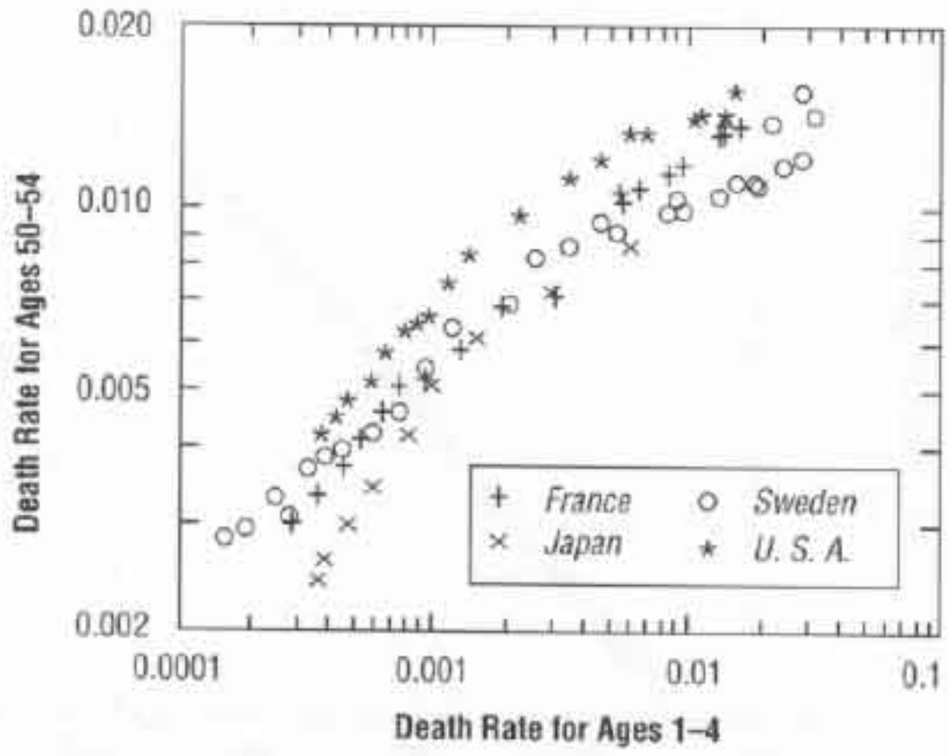
## Orchestrated Variations of Mortality Schedule

A *mortality schedule* is a set of age-specific death rates observed during a given period or over the lifetime of a cohort. As described earlier, empirical mortality schedules generally exhibit a three-phase pattern (downward, stable, and upward). Thus it might seem reasonable to expect mortality schedules that produce the same life expectancy at birth to be similar. However, very different combinations of age-specific death rates (e.g., a combination of relatively high child mortality and low adult mortality and a combination of relatively low child mortality and high adult mortality) can show the basic three-phase age pattern and produce the same life expectancy. Various hypothetical mortality schedules with the same life expectancy could be generated by adjusting the parameter values of, for example, the Siler model.

Nevertheless, data from various areas and countries in different periods suggest that age-specific death rates tend to change over time and vary among populations in fairly orchestrated ways. Death rates at even widely different ages are strongly positively

**FIGURE 3**

**Relationship between the Annual Death Rate for Females Aged 1–4 and the Annual Death Rate for Females Aged 50–54**




Note: Computed for five-year periods: France, between 1901 and 1995; Japan, between 1951 and 1995; Sweden, between 1861 and 1995; United States, between 1901 and 1995.

SOURCE: Human Mortality Database. <http://www.mortality.org>.

(though not linearly) correlated with each other (Figure 3). The reason that mortality schedules vary in orchestrated ways is probably that the overall level of socioeconomic and technological development in the population is reflected in various determinants of health and survival, including the standard of living, nutritional status, available medical technologies, and level of public health services. These factors, in turn, affect death rates of all age groups simultaneously.

High correlations among age-specific death rates make it possible to construct a set of *typical* mortality schedules corresponding to different levels of life expectancy. Such a typical schedule is called a model life table. Two well-known systems of model life tables are the Coale-Demeny model life tables and United Nations (Heligman-Preston) model life tables; both are widely used. The most typical patterns in the two systems are called the West model life tables in the Coale-Demeny system and the General Pattern in the U.N. system. Not surprisingly, the West life tables and General Pattern life tables are very similar.

However, some empirical mortality schedules depart systematically (though not greatly) from these typical patterns, probably reflecting mortality impacts of different natural and cultural environments. Thus both the Coale-Demeny and U.N. systems include additional sets of model life tables to describe these departures.

Demographers have developed several mathematical formulations of the relationships among age-specific death rates. These are called "relational models" and include the Brass logit model, the Heligman-Preston principal-component model, the Lee-Carter model, and Azbel's "law of survival." For example, in the Lee-Carter model, the death rate at age $x$ and time $t$, denoted by $m(x, t)$, is expressed as:

$$\log m(x,t) = a(x) + k(t)b(x)$$

where $a(x)$, $k(t)$, and $b(x)$ are estimated from a set of observed age-specific death rates for multiple periods. This implies that logarithms of age-specific death rates are linear functions of each other. The Lee-Carter model has been shown to closely fit mortality schedules of the United States between 1933 and 1987 and those of the G-7 countries (Canada, France, Germany, Italy, Japan, United Kingdom, and United States) during the second half of the twentieth century.

## Historical Changes in Mortality Schedules

Two major health transitions affected age patterns of mortality differently. In Figure 1, these two transitions are illustrated by changes in Swedish female mortality from 1875–1879 to 1950–1954 and changes from 1950–1954 to 1995–1999. The first type of transition (called the epidemiological transition) is the significant reduction of mortality from highly contagious infectious diseases, nutritional disorders, and complications of pregnancy and childbirth. In many countries that are at high levels of economic and technological development, this transition occurred mainly in the nineteenth century and the first half of the twentieth century. Early childhood mortality fell considerably, but old age mortality declined only modestly (Figure 1A). Because the relative reduction of adult mortality was greater at younger ages, the slope of the logarithmic mortality curve became steeper (Figure 1B).

The epidemiological transition greatly reduced the proportion of deaths in early childhood and concentrated deaths into a relatively narrow range of old

age. Thus the earlier peak in the distribution of deaths fell and the later peak rose (Figure 1C). The transition made the survival curve more rectangular. The survival curve has become fairly flat from birth to middle age because of the low mortality in this age range, and slopes steeply downward in old age because of the high concentration of deaths (Figure 1D).

The second transition is the substantial decline of mortality from degenerative diseases, including heart disease, stroke, and chronic kidney disease. In economically developed countries, this change started in the third quarter of the twentieth century. The absolute reduction of mortality was greater at older ages (Figure 1A), and the relative reduction of mortality was fairly constant over adult ages, producing a nearly parallel downward shift of the logarithmic mortality curve without appreciably changing the slope (Figure 1B). Because of the decline of old age mortality, the peak of the distribution of adult deaths moved toward older ages instead of rising higher (Figure 1C), and the downward slope of the survival curve shifted horizontally to the right without becoming noticeably steeper (Figure 1D).

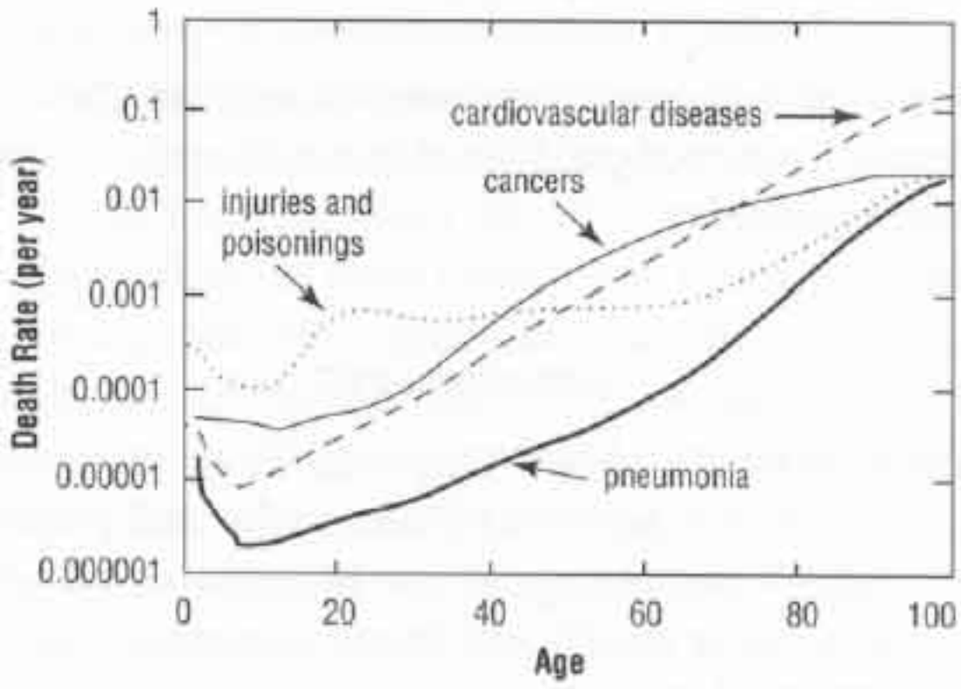
## Age Patterns of Cause-Specific Mortality

The age pattern of mortality differs among major causes of death. The differentials help in investigating the relationships between disease development and age-associated physiological changes, particularly the processes of senescence. Some typical age patterns of cause-specific mortality are shown in Figure 4. They are shown on a semi-logarithmic scale, because notable differences are found in the patterns of relative (rather than absolute) changes of mortality with age. Figure 4 displays data for France, but similar patterns are observed in other low mortality countries.

Above age 85, the curvatures of most cause-specific trajectories are concave (i.e., the gradients of the curves diminish with age), as seen for the four causes of death in Figure 4. Under age 85, the patterns are more variable. The death rate from cardiovascular diseases keeps rising steeply and exponentially throughout the adult ages. The death rate from cancers increases sharply at middle ages (the 30s and 40s), but slows down markedly at older ages, making the mortality curve of adult cancers concave already from around age 40. Most site-specific cancer death

**FIGURE 4**

**Death Rates per Year from Cancers, Cardiovascular Diseases, Pneumonia, and Injuries and Poisoning, by Age, Both Sexes Combined, France, 1979–1994**




SOURCE: National Institute of Demographic Studies (France). <http://matisse.ined.fr>.

rates follow this pattern as well. In contrast, the death rate from pneumonia increases relatively slowly around age 30 but the increase accelerates at old ages. Thus the mortality curve for adult pneumonia tends to be convex in the entire adult age range below 85.

Concave and convex curvatures on a logarithmic scale indicate deceleration and acceleration, respectively, of age-related relative increases in mortality. Overall, the curvature of the mortality age-pattern seems related to the selectivity of the disease. Concave patterns are likely to be seen for diseases that develop in persons with specific genetic, environmental, and lifestyle risk factors. Convex curvatures tend to be found for diseases to which most persons are vulnerable when they become old and frail. Concave patterns are seen for acute myocardial infarction, hemorrhagic stroke, and chronic liver diseases. In addition to cancers, convex patterns are observed for congestive heart failure, -infarctive stroke, chronic kidney diseases, and some infectious diseases such as influenza and septicemia.

A very different pattern is seen for mortality from injuries and poisoning, including accidents, homicide, and suicide. The death rate remains stable from age 20 to 60. In this age range, the risk of external injury is strongly related to behavioral patterns but not directly to physiological changes in the processes of senescence. At older ages the death rate

from injury and poisoning increases fairly rapidly. This is mainly due to rising mortality from accidents such as falls and the inhalation or ingestion of harmful substances, reflecting the weakening of the musculoskeletal system and diminishing effectiveness of neural control of the body.

**See also:** *Aging and Longevity, Biology of; Causes of Death; Epidemiological Transition; Gompertz, Benjamin; Life Tables.*

**BIBLIOGRAPHY**

Azbel, Mark Y. 1999. "Empirical Laws of Survival and Evolution: Their Universality and Implications." *Proceedings of the National Academy of Sciences of the United States of America* 96(26): 15368–15373.

Coale, Ansley J., and Paul Demeny. 1983. *Regional Model Life Tables and Stable Populations,* 2nd edition. New York: Academic Press.

Finch, Caleb E. 1990. *Longevity, Senescence, and the Genome.* Chicago: University of Chicago Press.

Gage, Timothy B. 1990. "Variation and Classification of Human Age Patterns of Mortality: Analysis Using Competing Hazards Models." *Human Biology* 62(5): 589–617.

Gavrilov, Leonid A., and Natalia S. Gavrilova. 1991. *The Biology of Life Span: A Quantitative Approach.* Chur, Switzerland: Harwood.

Heligman, Larry, and John H. Pollard. 1980. "The Age Pattern of Mortality." *Journal of the Institute of Actuaries* 107: 49–80.

Horiuchi, Shiro, and John R. Wilmoth. 1997. "Age Patterns of the Life-Table Aging Rate for Major Causes of Death in Japan, 1951–1990." *Journal of Gerontology: Biological Sciences* 52A: B67–B77.

Horiuchi, Shiro, and John R. Wilmoth. 1998. "Deceleration in the Age Pattern of Mortality at Older Ages." *Demography* 35(4): 391–412.

Kohn, Robert R. 1985. "Aging and Age-Related Diseases: Normal Processes." In *Relations Between Normal Aging and Disease,* ed. Horton A. Johnson. New York: Raven Press.

Lee, Ronald D., and Lawrence R. Carter. 199? "Modeling and Forecasting U.S. Mortality. *Journal of American Statistical Association* 8? 659–671.

United Nations. 1982. *Model Life Tables for Developing Countries.* New York: United Nations.

Vaupel, James W., et al. 1998. "Biodemographic Trajectories of Longevity." *Science* 280(5365): 855–860.

Vaupel, James W., Kenneth G. Manton, and Eric Stallard. 1979. "The Impact of Heterogeneity in Individual Frailty on the Dynamics of Mortality." *Demography* 16: 439–454.

Wilmoth, John R., and Shiro Horiuchi. 1999. "Rectangularization Revisited: Variability of Age at Death within Human Populations." *Demography* 36(4): 475–495.

SHIRO HORIUCHI