# Demographic Analysis of Aging and Longevity

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The populations of most of the world's countries are growing older. This shift is creating a new demography, a demography of low fertility and long lives. The rapidly growing populations of the elderly are putting unprecedented stresses on societies, because new systems of financial support, social support, and health care have to be developed and implemented. I will focus on a particular research thrust, namely, demographic analyses of survival and longevity. I will start with a review of the remarkable improvements in survival at older ages in recent decades.

"There is one and only one cause of death at older ages. And that is old age. And nothing can be done about old age." This verbal pronouncement by Leonard Hayflick,[1] a pioneering gerontologist, captures the gist of a prevalent syndrome of beliefs. Because deaths at younger ages are now unusual in developed countries, this view implies that human life expectancy in the developed countries, and in China and many other developing countries with low mortality, is close to the limit imposed by biology. The population of older people will grow as the baby boomers age, but if this view is correct, governments need not worry that enhanced survival at older ages might accelerate the growth. Furthermore, the view that mortality at older ages is intractable leads to the conclusion that health-care resources and biomedical research should increasingly be directed toward improving "the average well being of the population" rather than extending "the average lifespan" (P. H. M. Lohman et al., 1992; see S. Jay Olshansky et al., [1990] for a subtler conclusion).

Mortality at older ages is, however, by no means intractable (John R. Wilmoth, 1997). In fact, remarkable progress has been made since 1950 and especially since 1970 in substantially improving survival at older ages, even the most advanced ages. Despite this compelling evidence, the belief that old-age mortality is intractable remains deeply held by many people. Because of its implications for social, health, and research policy, the belief is pernicious. Because the belief is so prevalent, forecasts of the growth of the elderly population are too low, expenditures on lifesaving health care for the elderly are too low, and expenditures for biomedical research on the deadly illnesses of old age are too low.

The fact is that mortality at older ages has fallen dramatically since 1950 in developed countries and most developing countries as well. For instance, over the half century from 1900 to 1950, central death rates for 85-, 90-, and 95-year-old Swedish women hovered around 0.2, 0.3, and 0.4, respectively. By 1995 these death rates had fallen below 0.1, 0.2, and 0.3. Among female octogentarians and nonagenarians in England and Wales, France, Sweden, and Japan in 1950 there were about 180 deaths per 1,000 population. By 1995 there were less than 90 deaths per 1,000 population in all four of these countries. Similar progress was achieved in most other developed countries and in many developing countries. Improvements were also made for males, although male gains have generally been smaller than female gains (Vaino Kannisto, 1994, 1996; Kannisto et al., 1994).

Another, longer-term perspective is provided in Table 1, which documents the acceleration of mortality improvements for females in the Nordic countries of Denmark, Finland, Norway, and Sweden, countries for which reliable mortality data at older ages are available well back into the 19th century. Note the rapid improvements in mortality in recent decades, especially for women in their seventies and eighties.

Table 2 displays death rates by age and time for females in the Nordic countries. The in-

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[1] Talk given on 28 August 1988 at the American Association of Retired People (AARP) headquarters, Washington, DC ("The Likely Health, Longevity, and Vitality of Future Cohorts of Mid-Life and Older Persons").

TABLE 1—AVERAGE ANNUAL RATES OF IMPROVEMENT IN FEMALE MORTALITY (PERCENTAGES) FOR AGGREGATION OF DENMARK, FINLAND, NORWAY, AND SWEDEN, FOR SEXAGENARIANS, SEPTUAGENARIANS, OCTOGENARIANS, AND NONAGENARIANS, OVER SUCCESSIVE 20-YEAR PERIODS

| | Age category | | | |
|---|---|---|---|---|
| Time period | Sixties | Seventies | Eighties | Nineties |
| 1900's–1920's | 0.3 | 0.2 | 0.1 | 0.0 |
| 1920's–1940's | 0.7 | 0.4 | 0.2 | 0.0 |
| 1940's–1960's | 1.7 | 1.0 | 0.6 | 0.5 |
| 1960's–1980's | 1.5 | 2.1 | 1.7 | 1.2 |

*Source:* See Kannisto et al. (1994) for description of how average annual rates of improvement are calculated.

TABLE 2—FEMALE CENTRAL DEATH RATES (PERCENTAGES) FOR AGGREGATION OF DENMARK, FINLAND, NORWAY, AND SWEDEN, FOR SEXAGENARIANS, SEPTUAGENARIANS, OCTOGENARIANS, NONAGENARIANS, AND CENTENARIANS, IN TWO PERIODS, 1930–1949 AND 1989–1993

| | Age category | | | | |
|---|---|---|---|---|---|
| Period | Sixties | Seventies | Eighties | Nineties | 100+ |
| 1930–1949 | 2.4 | 6.4 | 16.1 | 33.9 | 70.1 |
| 1989–1993 | 1.1 | 3.1 | 9.1 | 23.4 | 48.5 |
| Change: | 1.3 | 3.3 | 7.0 | 10.5 | 21.6 |

*Source:* Kannisto et al. (1994).

crease in death rates with age is striking. The decrease in death rates over time is also striking. If mortality is reduced, then the number of lives saved is proportional to the absolute decline rather than the relative decline. In the last row of Table 2, the absolute improvements in Nordic female mortality are displayed. The large absolute reductions in mortality among centenarians and nonagenarians is a remarkable achievement, at sharp variance with the view that old-age mortality is intractable.

If death rates at older ages were approaching a biological limit, then it might be expected that improvements in countries with the lowest death rates would tend to be slower than in countries with death rates further away from the irreducible minimum. There is, however, no correlation, either for males or for females, between levels of mortality and rates of mortality improvement. Furthermore, males suffer higher mortality than females, but rates of improvements for females are higher than for males (Kannisto et al., 1994).

Until recently, demographers have been wary of using U.S. mortality data at older ages because of concerns about the validity of age-reporting. New data sources now permit accurate estimation of U.S. death rates, at least up to ages in the late nineties for the white population of the United States (Bert Kestenbaum, 1992; Kenneth G. Manton and Vaupel, 1995; Laura B. Shrestha and Samuel H. Preston, 1995). It turns out that for octogenarians and nonagenarians U.S. death rates (for whites) are substantially lower than death rates in Western Europe or Japan. In the 1980's at age 90, for instance, female death rates in Europe and Japan were almost 50-percent higher than in the Upper Midwest region of the United States (0.19 vs. 0.13) and about 20-percent higher than in the Deep South region.

This is remarkable because mortality before age 65 or 70 is substantially higher in the United States than in Western Europe and Japan. Because the very old particularly benefit from medical care and salubrious behavior, it is possible that the U.S. advantage stems from better health conditions for the elderly. The U.S. mortality advantage at older ages might also be at least in part due to the immigration of large numbers of healthy migrants into the United States in the decades before 1920. Another possibility is that conditions during childhood have lingering effects on health at advanced ages: the United States was a world leader in childhood health at the beginning of this century. In any case, the gap between the United States, on the one hand, and Western Europe and Japan, on the other, is further evidence for the plasticity of mortality at older ages.

## I. Rapid Growth of the Elderly Population

I now turn to the impact of mortality reductions on the growth of the elderly population, starting with the population of centenarians. In the countries where reliable data are available on centenarians, the number of centenarians is increasing at an exceptionally rapid rate, about

8 percent per year on average. Demographers are used to population growths rates around 1 percent per year or so; an 8-percent growth rate seems more like an inflation rate. In England and Wales, an average of 74 persons per year reached age 100 between 1911 and 1920; by 1990 the number of people celebrating their 100th birthday had increased to almost 2,000, and in 1997 the number will be around 3,000 (Vaupel and Bernard Jeune, 1995). In China, Zeng Yi and I estimate that the number of centenarians is doubling every decade. In 1990 there were about 6,000 people age 100 and above in China. By the year 2000 there may be more than 12,000.

The population of centenarians is growing, in part, because of the increase in births a century ago, the sharp decline in infant and childhood mortality, and the substantial decline in mortality at ages from childhood up to age 80. Demographic analysis demonstrates, however, that by far the most important factor in the explosion of the centenarian population (two or three times more important than all the other factors combined) has been the decline in mortality after age 80 (Vaupel and Jeune, 1995).

Centenarians are still unusual, but these findings do illustrate the fact that mortality reduction can have major impacts on population growth at older ages. The growth of the population of female octogenarians in England and Wales provides another telling example. The remaining life expectancy of 80-year-old females in England and Wales around 1950 was approximately six years. Currently the corresponding figure is about nine years, some 50-percent higher. As a result, the population of female octogenarians in England and Wales is roughly half again as big as it would have been if mortality after age 80 had remained at 1950 levels. Putting this in terms of population counts, more than a half million females aged 80+ are alive today in England and Wales who would have been dead if mortality after age 80 had not been reduced.

Table 3 provides information about the size of the older population of various countries, from age 60 and up, for both sexes combined. Estimates are also given for the size of these populations in 2025. The size of the older population shows substantial increases, not only in Europe, Japan, and the United States, but China, India, and other developing countries as well.

TABLE 3—PROPORTION OF POPULATION ABOVE AGE 60 (PERCENTAGE) AND POPULATION ABOVE AGE 60 (IN MILLIONS) FOR SELECTED COUNTRIES IN 1996 AND PROJECTED FOR 2025

| | Age 60+ (percentage) | | Age 60+ (millions) | |
|---|---|---|---|---|
| Country | 1996 | 2025 | 1996 | 2025 |
| Italy | 22 | 33 | 13 | 18 |
| Japan | 21 | 33 | 26 | 40 |
| Germany | 21 | 32 | 17 | 28 |
| France | 20 | 30 | 12 | 18 |
| United Kingdom | 21 | 29 | 12 | 17 |
| United States | 17 | 25 | 44 | 83 |
| China | 9 | 20 | 115 | 290 |
| Brazil | 7 | 16 | 11 | 31 |
| Mexico | 7 | 13 | 6 | 18 |
| India | 7 | 12 | 62 | 165 |
| South Africa | 7 | 10 | 3 | 6 |
| Egypt | 6 | 10 | 4 | 10 |

*Source:* U.S. Bureau of the Census (1997).

## II. Variation in Lifespan

The multiplication of the population of older people heightens interest in a fundamental question: why do some people die at 60, others at 80, and a few at 100? Why are the odds of dying at 80 rather than 60 increasing and the chance of surviving to 100 rapidly increasing (albeit from a very low level)? How important are genetic versus environmental, behavioral, and medical factors in determining how long an individual will live?

Studies of twins and other kinds of related individuals suggest that about 25 percent of the variation in adult lifespans appears to be attributable to genetic variation among individuals (Matt McGue et al., 1993; Anne Maria Herskind et al., 1996). Some research in progress by two of my colleagues (Anatoli Yashin and Ivan Iachine) suggests that an additional 25 percent may be attributable to nongenetic characteristics that are more or less fixed by the time a person is 30 or so: characteristics such as educational achievement, socioeconomic status, mother's and father's age at a person's birth, etc. Research on the relative importance for longevity of various candidate genes and nongenetic fixed attributes is, however, still at an early stage of development.

David J. P. Barker's (1992, 1995) "fetal-origins hypothesis" suggests that nourishment in utero and during infancy programs the development of risk factors for several important diseases of middle and old age. Other researchers have also concluded that nutrition and infections early in life have major effects on adult mortality (W. Kermack et al., 1934; Irma T. Elo and Preston, 1992; Fogel, 1993). To the extent that this is true, longevity may be determined by conditions in childhood and perhaps before birth. There is, however, conflicting evidence that suggests that current conditions (i.e., at older ages) may be much more important than conditions early in life. Kannisto (1994, 1996) finds period effects to be considerably more significant than cohort effects on mortality after age 80. Kaare Christensen et al. (1995) find that, from age 6 up to the oldest ages, twins (who tend to be born prematurely and at low birth weight) suffer the same age-specific death rates as singletons; and Kannisto et al. (1997) find no increased mortality in later life for cohorts born during the Finnish famine of 1866–1868. Pinning down the nature and magnitude of possible lingering effects of early-life conditions on survival at advanced ages is an important research priority.

## III. Trajectories of Mortality at Advanced Ages

Further insights into the determinants and plasticity of longevity can be gleaned by analyzing the trajectories of age-specific death rates at advanced ages, both for humans and for various nonhuman species (Vaupel, 1997). Benjamin Gompertz (1825) proposed that the force of mortality increased exponentially with age for humans, at least as a serviceable approximation over the range of adult ages for which he had data. Various subsequent researchers, especially in biology and gerontology, have viewed Gompertz's observation as a law that describes the process of senescence in almost all multicellular animals at all ages after the onset of reproduction. As a rough approximation at younger adult ages, Gompertz's exponential formula does capture the rise in mortality in a great variety of species (Caleb E. Finch, 1990). Human mortality, however, does not increase exponentially after age 80. Mortality decelerates, rising perhaps to a maximum or ceiling around age 110 (A. Roger Thatcher et al., 1998). Whether mortality is slowly increasing, level, slowly decreasing, or rapidly decreasing after age 110 is uncertain.

Humans are animals. Almost all animals show signs of aging, and for almost all animals death rates tend to rise after the age of maturity (Finch, 1990). Even researchers who are only interested in people may benefit from biological insights from studies of other species, because these insights may cast light on the biology of humans. I will give just one example. The largest nonhuman population followed to natural death consisted of 1.2 million medflies studied in a laboratory near Tapachula, Mexico. These flies were held in cages, each holding several thousand flies. As reported by James R. Carey et al. (1992), the trajectory of mortality rose, peaked, and then fell to a low level around which it hovers until the last fly died at an age of 171 days (compared with an average life span in the experiment of 21 days).

Why does mortality decelerate? One reason is that all populations are heterogeneous. Some individuals are frailer than others, and the frail tend to die first. This creates a fundamental problem—indeed, it seems to me the fundamental problem—for demographic analyses in general and for analyses of age-trajectories of mortality in particular. The individuals alive at older ages are systematically different from the individuals alive at younger ages. The age-trajectory of mortality reflects both the underlying age-trajectories of mortality for individuals in the population and the effects of compositional change as the frailer individuals drop out of the population (e.g., Vaupel et al., 1979; Vaupel and Anatoli I. Yashin, 1985; Vaupel and Carey, 1993).

Living organisms are complex systems. Reliability engineers and systems analysts have learned a great deal about the failure of complex systems. Automobiles are popular pieces of complicated equipment. They are sufficiently standardized that it is meaningfully possible to count their numbers on an age-specific basis. Then age-specific death rates can be calculated. It turns out that automobile mortality rises steeply at younger ages but levels off around age 10 or 12 (Vaupel and Cindy R. Owens, 1997). The question arises: is mortality a property of living organisms or a property of complicated systems? When it comes to death, how do people and flies differ from Toyotas? In particular, is the deceleration and leveling off of mortality a fairly general property of complicated systems? Better understanding of these questions may lead to new insights into aging and survival.

### IV. Conclusion

Over the past half century, and especially in the most recent decades, remarkable improvements have been achieved in survival at older ages, especially at the highest ages. This progress has accelerated the growth of the population of older people and has advanced the frontier of human survival substantially beyond the extremes of longevity attained in preindustrial times. The widely held position that mortality at older ages is intractable is untenable. However, little is yet known about why mortality among the oldest-old has been so plastic since 1950. There is considerable (but still inadequate) knowledge of why some people die in infancy or childhood and why some people die prematurely at adult ages before age 60 or 70. Much less is known about why some people survive to age 80, others to age 90, and a few to age 100. The little that is known has largely been learned within the past few years, and new findings (especially concerning genetic factors) are emerging at a rapid rate. A key finding is that mortality decelerates at advanced ages not only for humans, but for other biological species and for automobiles as well. The deceleration results from some mix of genetic, environmental, behavioral, bio-reliability, and heterogeneity forces and constraints, but the mix is not well understood.

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