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Articles

In Search of Methuselah: Estimating the Upper Limits to Human Longevity

S. JAY OLSHANSKY, BRUCE A. CARNES, CHRISTINE CASSEL

Estimates of the upper limits to human longevity have important policy implications that directly affect forecasts of life expectancy, active life expectancy, population aging, and social and medical programs tied to the size and health status of the elderly population. In the past, investigators have based speculations about the upper limits of human longevity on observations of past trends in mortality. Here the estimate of the upper bound is based on hypothesized reductions in current mortality rates necessary to achieve a life expectancy at birth from 80 to 120 years and an expectation of life at age 50 from 30 to 70 years. With the use of conditional probabilities of death from complete life tables for the United States, reductions in mortality required to achieve extreme longevity (that is, 80 to 120 years) were compared with those resulting from hypothetical cures for all cardiovascular diseases, ischemic heart disease, diabetes, and cancer. Results indicate that in order for life expectancy at birth to increase from present levels to what has been referred to as the average biological limit to life (age 85), mortality rates from all causes of death would need to decline at all ages by 55%, and at ages 50 and over by 60%. Given that hypothetical cures for major degenerative diseases would reduce overall mortality by 75%, it seems highly unlikely that life expectancy at birth will exceed the age of 85.

SINCE THE MID-19TH CENTURY THE HUMAN POPULATION HAS experienced one of the most important and dramatic changes in the history of the species—a near doubling of the expectation of life at birth from 40 to near 80 years (1). Most of the mortality declines and increases in life expectancy that occurred early in this century were a result of rapidly declining neonatal, infant, and maternal mortality. Today, mortality rates in younger and middle age groups are so low in the United States that the complete elimination of mortality before the age of 50 (about 12.4% of all deaths) would increase life expectancy at birth by only 3.5 years. Thus, the potential for additional increases in longevity relies on progress in dealing with the diseases of the elderly.

While there has always been a segment of the human population that survived to older ages, from ancient times until this century

only marginal improvements seem to have been made in extending life for the population aged 50 and older. Interestingly, in the last 25 years in the United States, age-adjusted death rates from the major cardiovascular diseases declined by more than 34% (2). Most of the declines in mortality and gains in life expectancy during this recent mortality transition were achieved in the elderly population—a phenomenon so unexpected and unexplained that it has been referred to as a new stage in the epidemiologic history of developed nations (3–5).

Having made such rapid advances in the extension of life during the last 100 years, researchers are now beginning to ask why this recent transition in mortality took place, how long it can continue, and what factors might contribute to future gains in life expectancy (6)? These are crucial policy questions because they address estimates of the size, proportional distribution, and health status of the elderly population in the future.

A related issue of critical importance is whether further declines in mortality would lead to an increased active life expectancy or an expanded period of frailty and dependency. Unless active life expectancy is improved from present levels, the combination of population aging, a larger than predicted elderly population, and possible shifts in the distribution of frailty conditions among the oldest-old, would have an enormous adverse impact on government-funded programs such as Social Security and Medicare. Many interdisciplinary studies are currently addressed to this issue (7–12).

In this article we examine the methods that have been used to estimate the upper limits to human longevity and determine precisely what age-specific mortality declines would be required to achieve specified life expectancy targets. Insights gained from pursuing the second line of inquiry should help clarify issues concerning active life expectancy.

Terminology

Since research on human aging and longevity is an interdisciplinary effort, the terms used to describe these phenomena (for example, life expectancy, active life expectancy, life-span, average life-span, maximum life-span potential) are often used interchangeably (6). To avoid problems of definition, the terms we use are defined as follows: (i) “life expectancy” is the average number of years of life remaining for a population of individuals, all of age x , and all subject for the remainder of their lives to the observed age-specific death rates corresponding to a current life table. This is referred to in demography as “period life expectancy” because it is based on the risks of mortality that are present during a single time period (13). Although life expectancy may be calculated for any age, it is most often presented as life expectancy at birth. However, when the concern is mainly with older ages, the expectation of life at some

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middle age (such as age 50) is a far more useful index. (ii) “Active life expectancy at age x ” is the average number of years of life remaining in an independent state (free from significant disability) for a population of individuals, all of age x , and all subject for the remainder of their lives to the observed age-specific risks of disability. (iii) “Life-span” is the genetically endowed limit to life for a single individual if free of all exogenous risk factors. It is not possible to actually observe or estimate the life-span of an individual. Therefore, life-span is a theoretical construct primarily used to estimate the theoretical upper limits to life and contrast prevailing mortality conditions (as measured by period life expectancy) with those that are theoretically achievable. (iv) “Average life-span” is the average of individual life-spans for a given birth cohort. Because life-span refers to individuals in a heterogeneous population, it is inappropriate to use the term “life-span” for an entire population. In fact, the life-spans for a given birth cohort could range from 1 day of life to over 120 years. Thus, the average life-span of a population is also a theoretical construct that can be estimated (14–18) but not measured directly. (v) “Verified longest lived individual” is a member of a given species whose maximum length of life has been observed and verified. Although this term implies a maximum upper age limit for every member of the population, it is more appropriate to consider this age as a statistical outlier given a heterogeneous distribution of individual life-spans. In the gerontology literature, the verified age of the longest lived individual represents an operational definition of the maximum life-span potential (MLSP) for a species (19).

To give visual meaning to these terms, data for the United States is presented in Fig. 1. First, note that life expectancy at birth has increased from 47 years in 1900 to about 75 years in 1988. Presenting the distribution of deaths for hypothetical cohorts (of 100,000 persons) with the observed mortality risks for the two time periods is a simple way to describe why a rapid increase in life expectancy has occurred. The 1900 curve illustrates that (i) just under 12 of every 100 female babies born in that year died before their first birthday, (ii) there was a pronounced increase in mortality among women during their reproductive years, and (iii) the survivors of high infant, child, and maternal mortality tended to die between the ages of 50 and 80. The dashed line illustrates that for females born in 1985, the vast majority of their deaths will be concentrated between the ages of 70 and 90. The differences between these two curves is a clear illustration of the mortality transition that took place during this century. That is, deaths from infectious and parasitic diseases were rapidly replaced by deaths from chronic degenerative diseases.

If we assume an 85-year average life-span for women, the fact that

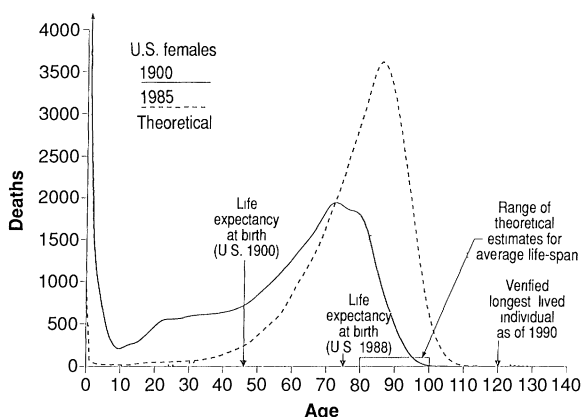


Fig. 1. Observed and theoretical distribution of life table deaths for females in the United States, 1900 and 1985.

the modal age at death is near 85 suggests that the upper limit to longevity has already been approached and that further significant declines in mortality are unlikely (14, 15). If true, the current distribution of deaths for U.S. females (dashed line, Fig. 1), approximates the actual distribution for individuals experiencing their genetically endowed limit to life. Thus, the 78-year average age at death for this hypothetical cohort represents an estimate of the average life-span for the population. If, however, average life-span was closer to 100 years with a similar scatter of individual life-spans about the mode (13, 19, 20), the distribution of deaths would look more like the dotted line in Fig. 1.

As of 1990, the oldest verified age that an individual has survived is just over 120 years. Cases of such extreme longevity are expected in the coming decades as the statistical probability of an outlier surviving past the average life-span increases with larger cohorts surviving to older ages. Note, however, that claims of extreme longevity (for example, beyond the age of 120) for some subgroups of the population have been shown to be false (21–24). Evidence suggests a tendency for age exaggeration in societies where persons of advanced age are shown increased deference.

Estimating the Upper Limits to Human Longevity

Several attempts have been made to estimate the average life-span of the human population. With the use of a cumulative survival distribution, one method operationally defines the average life-span as the age beyond which only 0.1% of the population (of a given cohort) has survived (13). Although arbitrary, this method permits change in the estimate as newly observed mortality data modifies the estimated survival distribution. Based on observed mortality rates from 1985, the average life-span of the U.S. population is 108 years (based on data published by the Social Security Administration) (25).

A second method is based on the construction of a composite mortality profile made up of the lowest age-sex-cause-specific mortality rates that are presently observed in developed countries during a single time period (17). With data from developed countries in 1974, the upper limits to human life expectancy at birth were estimated to be 79.4 years for females and 73.8 years for males.

In another approach, the underlying causes of death observed for the longest lived subgroup of the human population are partitioned into two main categories: exogenous causes (infectious and parasitic diseases, accidents, homicide, suicide), and endogenous causes (mostly cardiovascular and cerebrovascular diseases and neoplasms). Standard life-table methods are used to hypothetically eliminate all exogenous causes of death (26). Lower bounds of endogenous mortality are then derived from the most favorable mortality schedule observed for a given country during a given time period (15). This approach assumes there are lower limits beyond which endogenous mortality cannot be reduced. Eliminating exogenous mortality and using presumed lower thresholds for endogenous mortality results in an average life-span estimate of 80.3 years for females and 73.8 years for males.

Linear extrapolation was used in a fourth approach to extend recently observed trends in period life expectancy at birth and at age 65 for males and females. Since life expectancy at birth cannot exceed life expectancy at age 65, the age at which these two trend lines converged was thought to represent an estimate of the average life-span for the human population (14). On the basis of the most recent mortality data for the United States and Japan, the average life-span for humans was estimated by this method to be between the ages of 85.1 and 86.3 (18).

In a fifth method, population level risk factors (such as smoking and symptoms of improper diet like high blood pressure and serum cholesterol levels) are modulated, and the subsequent changes these factors have on mortality rates and life expectancy are quantified (16). It is assumed that population risk factors were responsible for the mortality declines observed in developed nations in recent decades and that these same factors are likely to contribute to future declines in mortality. If lower bounds for mortality attributable to risk factors are assumed, theoretical manipulation of these factors should permit estimates of the upper limits to human longevity to be obtained. This model addresses a fundamental weakness in the other approaches which do not show a sensitivity to behavioral (that is, personal habit) changes that may propel life expectancy toward its biological limits. From an "optimal" risk factor profile, it was suggested that the average life-span of the human population could be as high as 99.2 years.

Finally, it should be noted that the Office of the Actuary at the Social Security Administration has projected what they called "ultimate mortality rates" (27) and more recently "ultimate rates of mortality decline" (28). It was stated that these projected mortality schedules were not subject to further reductions beyond the middle or latter part of the 21st century (27). Hence, they appeared to represent an Office of the Actuary estimate of the upper limits to human longevity. However, these mortality estimates are only one of three forecasted components of population change (mortality, fertility, and migration). The three components are combined to provide an estimate of future trends in overall population growth in the United States, and to estimate the growth of the elderly population. Recent publications by the Office of the Actuary no longer state that projected mortality rates are not subject to further reductions. Therefore, inferences concerning upper limits to longevity based on their approach, particularly for the entire human species, cannot be reasonably made.

Estimating realistic bounds on potential longevity in the future is the common theme in all the methods described so far. The fundamental question addressed in these studies is "what are the limits to mortality declines?" A useful alternative to asking what the effect of certain mortality changes would be on longevity, is to reverse the question and ask, "What mortality rates would be necessary to achieve selected life expectancy targets?" For example, what mortality schedules would be required if life expectancy at birth were between the ages of 80 and 120, or if the expected remaining years of life at age 50 were to rise to between 30 and 70 years? Furthermore, how would the predicted mortality schedules of such long-lived populations compare with current mortality schedules?

Identifying mortality schedules that would lead to specified levels of life expectancy makes it possible to assess whether the gap between observed mortality and that required for extreme longevity could plausibly be achieved. It would also be particularly useful to compare the required mortality declines with those that would exist if major fatal degenerative diseases could be eliminated while holding other rates constant. In a sense, this is an engineering approach because it permits us to define the magnitude of the public health and medical interventions required to achieve specified increases in life expectancy.

An alternative approach involves the comparative evaluation of increases in mortality risks that are observed among successive age groups. It may be shown, for instance, that the probability of dying doubles about every 8 years past the age of 30 (29). Because age-specific mortality rates in the United States have been declining throughout the age structure since the late 1960s, the mortality risks of older age groups have been shifted in the direction of those previously experienced by younger age groups (3). In effect, each successive cohort surviving into older ages appears younger (that is,

their risk of death is lower) than previous generations who survived to the same ages. The question now becomes, "How young (in terms of acquiring the risk of death of younger age groups) would the population need to become in order to achieve specified increases in longevity?" Additionally, given what is currently known about the effect of changes in risk factors on mortality rates (16, 30), and the potential effects of eliminating degenerative diseases on life expectancy (31), is it reasonable to expect that the risk of death for older age groups could be postponed to the extent required to produce extreme longevity?

Estimating Extreme Cases of Longevity

The primary sources of data are complete life tables for the United States in 1980 and 1985, by sex, published by the Office of the Actuary at the Social Security Administration (25). The mortality schedules that would be required to produce a life expectancy at birth from 80 to 120 years of age (in 5-year increments based on 1985 rates) were estimated for two scenarios: (i) a proportional reduction in the conditional probabilities of death, $q(x)$, throughout the entire age structure, or (ii) a proportional reduction in $q(x)$ restricted to ages 50 and older. The proportional reductions were iterated until each of nine targeted levels of extended life expectancy at birth were achieved. It should be noted that the reduction of $q(0)$ was not allowed to decline below one-half of that observed in 1985. This constraint was imposed to conform with the likely biological reality that infant and child mortality cannot be reduced below five to six deaths per 1000 live births. It should be emphasized that life expectancy at birth is not sensitive to various assumptions about declining infant mortality because it is already so low.

A constant proportional reduction in the probabilities of death throughout the age structure causes larger absolute reductions at older ages where mortality rates are high. Proportional reductions also progressively shift the larger mortality rates further toward older ages. Both effects on $q(x)$ values are consistent with what is expected (3). When applied to the entire age structure, necessary reductions may be underestimated because the already low mortality rates observed for younger and middle age groups are unlikely to decline further. The second reduction scenario corrects this potential problem by restricting mortality reductions to ages 50 and older. Although not all inclusive, we think that the mortality scenarios presented here are biologically plausible.

The complete life tables published by the Office of the Actuary end at the age of 119. Any reductions in mortality will cause larger segments of the population to survive into the oldest age interval. A problem then arises because assumptions must be made about mortality rates beyond the oldest observed age interval. Survival scenarios for these hypothetically long-lived individuals would influence projected levels of life expectancy, the absolute size of the oldest old population, and trends in population morbidity and disability.

For each life expectancy target the mortality schedules for the elderly shifted to those experienced by younger age groups. The age shift required to achieve each of the nine life expectancy targets was determined for ages 55, 65, 75, 85, and 95. At these selected ages, the $q(x)$ values for each reduction scenario (applied to ages 50 and older) were compared to those observed for younger ages in the 1985 population. When a match with the 1985 schedule was identified, the difference in years represents the mortality age shift required to achieve the targeted increase in longevity for that specified age and reduction scenario.

Estimates of the levels of life expectancy at birth and at age 50 that would occur with the hypothetical elimination of selected chronic

degenerative diseases were arrived at by applying a cause-elimination life-table method (26) to the $q(x)$ values for the United States in 1985 (published by the Office of the Actuary). This model relies on an independence assumption where hypothetically eliminating one disease will not influence the risk of death from other diseases. However, it is known that degenerative diseases act interdependently. To correct this potential problem, a broadly defined disease category (encompassing interdependent diseases) was used for these calculations. Mortality counts published by the National Center for Health Statistics (32) for the United States in 1985 were used to partition overall mortality into the age-specific contributions from malignant neoplasms [9th International Classification of Diseases (ICD) 140-239], ischemic heart disease (ICD 410-414), diseases of the circulatory system (this includes primarily heart disease and stroke; ICD 390-459), and diabetes (ICD 250).

Mortality Reduction Required to Achieve Extreme Longevity

Extremely large declines in mortality for the entire population would be required for either sex to achieve life expectancies at birth between 80 and 120 years (Fig. 2). For example, a life expectancy of 80 years would require a 12% reduction in overall mortality for females, and a 48% reduction in mortality at all ages for males. A life expectancy of 85, the average biological limit hypothesized by Fries (33), would require a mortality reduction of 43% for females and 65% for males. Life expectancy at birth could reach 90 years and older only if mortality rates for 1985 were reduced by approximately 70%. A life expectancy of 105 would require reductions in excess of 90%. Although it is not expected that the gender gap in life expectancy will be eliminated, to do so would require larger mortality reductions for males because they have higher mortality rates than females throughout the age structure.

When mortality reductions are restricted to the 50-year and older age groups, the data (Fig. 2) suggest that only slightly larger proportional reductions are required to produce the same life expectancies as those achieved when reductions were applied to the entire age structure. Mortality reductions early in life have only a small impact on life expectancy at birth because mortality rates at younger ages are already low.

To put the proportional reductions in perspective, consider the hypothetical elimination of major causes of death in the United States (Fig. 2). Eliminating all forms of cancer (22.45% of all deaths

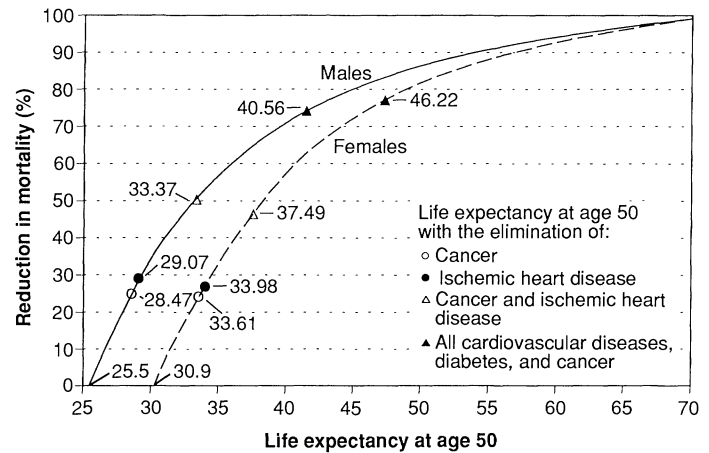


Fig. 3. Percentage of reduction in the conditional probability of death for the United States (from 1985 levels) required to produce a life expectancy at age 50 from 30 to 70 years.

in the United States in 1985) would increase life expectancy at birth by 3.17 years for females and 3.2 years for males. Eliminating ischemic heart disease (25.73% of all deaths in 1985) would increase life expectancy at birth by 3.0 years for females and 3.55 years for males. Eliminating both diseases together would increase life expectancy at birth by only 7.02 years for females and 8.1 years for males. If mortality attributable to the combination of all circulatory diseases, diabetes, and cancer was eliminated (71.34% of all deaths in 1985), life expectancy at birth would increase by 15.82 years for females and 15.27 years for males. It would require about a 75% reduction in mortality from all causes to equal the impact of eliminating these three major disease categories.

Life expectancy at age 50 has already exceeded 30 years for females (Fig. 3). However, male mortality rates would need to decline by about 35% from 1985 levels to achieve a 30-year expectation of future life at age 50. An increase in the expected remaining years of life at age 50 to 35 years (that is, an average life-span of 85 years) would require declines in mortality of 34% for females and 58% for males from 1985 levels. Eliminating cancer and ischemic heart disease together would increase the expected remaining years of life at age 50 above current levels by only 6.57 years for females and 7.83 years for males. Eliminating all circulatory diseases, cancer, and diabetes combined would increase the expected remaining years of life at age 50 above current levels by 15.3 years for females and 15.02 years for males. These increases are roughly equivalent to 75% reductions in mortality from all causes of death (that is, their combined contribution to all deaths) for the population aged 50 and older in 1985. These gains are similar to those described for life expectancy at birth because more than 94% of all deaths from these disease groups occur after the age of 50. To achieve a life expectancy of 50 years at age 50, mortality rates for either sex would need to decline by about 85% from 1985 levels (Fig. 3).

Barring major advances in the development and use of life-extending technologies or the alteration of human aging at the molecular level, the period of rapid increases in life expectancy in developed nations has come to an end. This does not mean that the size of the oldest old population will not continue to increase. Indeed, even if death rates remain at their current levels, the size of the population aged 65 and older in the United States will more than double by the third decade of the next century. This phenomenon, population aging, is the result of the aging of the large baby boom cohorts born in the United States between 1946 and 1964.

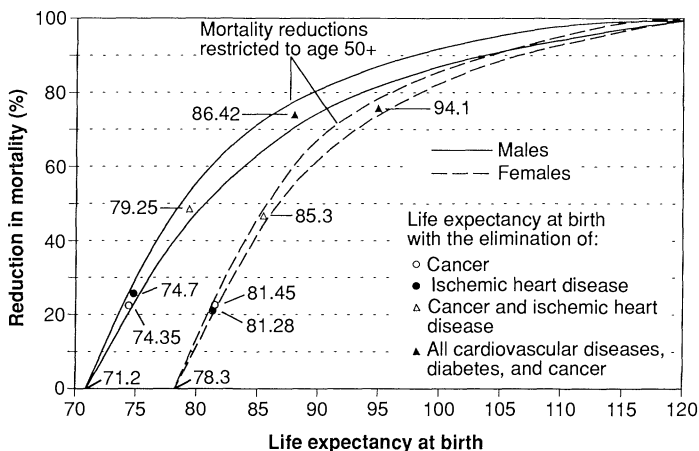


Fig. 2. Percentage of reduction in the conditional probability of death for the United States (from 1985 levels) required to produce a life expectancy at birth from 80 to 120 years.

The end of rapid increases in life expectancy does not mean that mortality rates will necessarily stop declining, as some have argued (14). Additional mortality declines that do occur, even if concentrated in older ages as expected (3), will have a minimal effect on life expectancy at any age. That is, it will take increasingly larger reductions in mortality to produce equivalent incremental increases in life expectancy. This explains why increases in life expectancy at birth and at age 65 have tapered off in recent years among the longest lived subgroup of the population in the United States (white females), even though their mortality rates continue to decline. Therefore, life expectancy at birth is not an adequate metric of mortality declines when life expectancy at birth approaches 80 years. This particular relation between changes in mortality and life expectancy has been discussed by various authors (34–36).

Survival Curves

Survival curves for females (Fig. 4) in the United States for each of the proportional reduction scenarios were compared to the observed survival curve for females in the United States in 1980. Similar patterns of survival were projected for males. A survival curve describes the survival experience of a hypothetical cohort of 100,000 babies born during a single year, assuming their future mortality will conform to that observed for the other age cohorts in that year.

Note that the survival curves become progressively more rectangular in appearance as life expectancy at birth increases. Recall, however, that the 1980 survival curve can only be observed to the age of 119 because this was the age limit of $q(x)$ values provided by the Office of the Actuary. Beyond age 119, it is not possible to know the shape of the survival curve.

Recent evidence indicates that the U.S. population is currently experiencing declines in old age mortality and an increase in the variance of the average age at death. In subsequent discussion, the combination of these two phenomenon will be referred to as “the expansion of mortality” (37–38). If the risk of death is delayed without mortality compression, the declining portion of the survival curve will begin at later ages but retain its characteristic shape at older ages. The continued expansion of old age mortality currently observed is an indication that the expected mortality compression has not begun, and more people, therefore, will survive beyond the age of 85 than was previously thought possible. As a result, the survival curves shown here are shifting to the right with the declining portion of the curve moving off the graph. With declining old age mortality and a greater number of centenarians, it will be possible in the future to more precisely estimate the shape for this region of the survival curve.

With changing life expectancy, the median age at death (the age to which half of the original birth cohort would survive) will also change dramatically. For example, if life expectancy increased to 85 years, the median age at death would increase from its present level of 81 to 88 years of age. Thus, one half of all females born in the year during which life expectancy is 85, would live to see their 88th birthday. Similarly, one half of the females born in a year during which life expectancy is 100, would live to see their 104th birthday. If life expectancy increased to 110 years, about 80% of the original birth cohort would live to see their 102nd birthday. These extreme scenarios are improbable given that current (1985) mortality rates would need to be reduced by approximately 85% for the population aged 50 and older to live, on average, an additional 50 years.

The age shifts in mortality required to increase the expected remaining years of life at age 50 to between 30 and 70 years were calculated for both sexes at selected ages (Fig. 5). For example, to

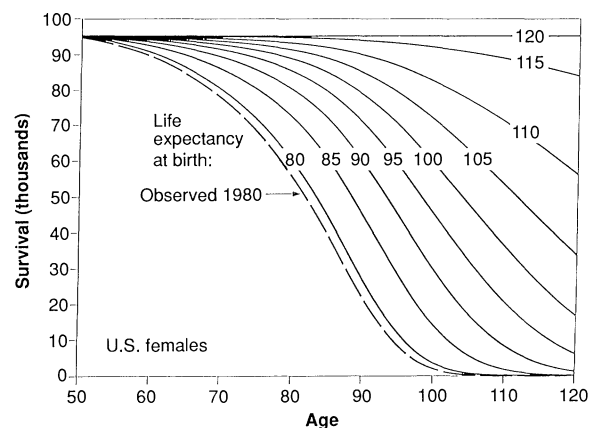


Fig. 4. Observed (1985) and projected survival curves for U.S. females at ages 50 and older with a life expectancy at birth from 80 to 120 years.

increase male life expectancy at age 50 by 6.25 years (from 23.75 years to 30 years), 55-year-old males would need an age shift in mortality of 5 years (the risk of death currently observed by 50-year-olds). An increase in life expectancy of about 16 years would require an age shift of 14 years (the mortality currently observed for males 41 years of age). It appears that for males between the ages of 55 and 75, each year of life expectancy gained requires an equivalent shift in years toward the mortality currently experienced by a younger age cohort. Beyond the age of 75, slightly larger shifts are required (Fig. 5). An expectation of remaining years of life at age 50 of 50 years would require the highly improbable scenario of a 55-year-old man experiencing the mortality risk of a teenager. Similar relationships hold for females.

Even though the risks of death have been postponed in the United States such that successive generations appear “younger” (in terms of their probabilities of death) than previous generations, the dramatic age shifts in mortality required for increases in life expectancy beyond 85 years appear unlikely.

Conclusions

These results have important implications for the forecasting of life expectancy and active life expectancy, and the compression of morbidity hypothesis. Current methods for estimating gains in longevity rely on past trends in overall or cause-specific mortality rates (28, 39), or on estimates of the effects of improvements in risk factors at the population level (16). The data presented here indicate that life expectancy should not exceed 85 years at birth or 35 years at age 50 unless major breakthroughs occur in controlling the fundamental rate of aging. To achieve these levels of life expectancy, mortality declines would have to be concentrated among the major fatal degenerative diseases for the population aged 50 and older. Furthermore, as life expectancy increases, increasingly larger reductions in mortality will be required to produce equivalent increases in life expectancy. These observations suggest that the measure of period life expectancy becomes a less sensitive metric of mortality declines (particularly when they occur among older age groups) as life expectancy at birth approaches 80 years. It is our opinion that with existing medical technology, declines in mortality comparable to the total elimination of all circulatory diseases, diabetes, and cancer combined is highly improbable. Even if these diseases were eliminated, life expectancy at birth for the population of the United States would not exceed 90 years. However, we strongly suspect that major advances in genetic engineering and new life-extending

technologies are forthcoming, and these will be followed by commensurate declines in mortality and extensions of longevity.

From a policy perspective, it is therefore important to change our focus from the metric of life expectancy to the absolute number of people who are expected to survive to older ages in the future, and the morbidity and disability profiles they will experience. The size of the elderly population has historically been underestimated (40, 41), and prospects for additional mortality declines indicate that the size of this population could grow considerably even beyond present expectations. Forecasts of the size of the elderly population depend critically on the distribution of death among those who survive to the ages of 85 and older (that is, the slope of the decline phase of the survival curve), and it is these numbers that are important to estimates of social security outlays and health care costs. It should be emphasized that while declines in old age mortality produce small increases in life expectancy, they result in extremely large increases in the expected size of the elderly population.

Fries (33) hypothesized that mortality will be compressed against a fixed upper limit to life. However, recent evidence suggests that instead of mortality compression, there is currently an "expansion of old age mortality" in the United States (37, 38). This is an indication that mortality compression has not yet begun. Recent mortality declines for older age groups have not been accompanied by a commensurate extension of the tail end of the age distribution or an increase in the age of the verified longest lived individual. Thus, it would appear that a biological limit to life is operating. At present, there are insufficient data available on the survival trajectories of the extreme elderly population to improve forecasts of the size of the centenarian population.

A crucial societal issue arises as a consequence of continued expansion in old-age mortality. Will trends in population morbidity and disability remain constant, follow a course that parallels mortality, or be postponed and compressed against a fixed average life-span? If the expansion of old age mortality continues, much larger segments of the population will survive beyond the average life-span, and compression of morbidity seems much less likely. Several unknowns exist. Why is expansion of old age mortality occurring; when will it stop (that is, what is the average life-span of the population); and will the survivors to these older ages be more or less healthy than is the case today?

Addressing the first question is likely to shed light on the latter two (42). If improvements in risk factors for fatal degenerative diseases are responsible for the observed declines in old age mortality, then morbidity and disability may exhibit commensurate de-

clines. These declines would occur only if improvements in risk factors have the same effect on postponing the onset of morbidity and disability as they have on postponing mortality. However, advances in medical treatment more than improvements in risk factors, may be allowing elderly persons who are frail and who suffer from fatal degenerative diseases to survive longer after the onset of the diseases than was the case in the past. In this case, age-specific morbidity and disability rates and their duration would increase substantially. In fact, even if rates of morbidity and disability remain constant, the number of people surviving with conditions of frailty will definitely increase because of the rapid growth in the size of the elderly population resulting from population aging and declining old-age mortality.

Even with improved life-styles and health care at the population level, morbidity compression may not occur. The decline in old-age mortality may still be accompanied by fixed or even increasing rates of morbidity and disability. This could occur if the disabling diseases are different from the major causes of death in terms of their predisposing risk factors and basic pathology. Constant or increasing rates of morbidity and disability could also occur if the distribution of frailty conditions experienced at advanced ages shifts from disease-dependent causes to more inherent and currently unavoidable age-dependent physiological decrements.

Fries (33) also hypothesized that morbidity will decline and become compressed into a shorter duration of time before death. However, improved life-styles at younger ages and medical technology will continue to strip away lethal processes that terminate life early. Left behind will be a rapidly growing elderly population that lives longer but whose additional years of life may be dominated by nonfatal but highly disabling conditions of frailty (such as arthritis, osteoporosis, sensory impairments, and Alzheimer's disease). Therefore, a prudent course of action for the medical community would be to vigorously seek ways to ameliorate the disability associated with nonfatal age-dependent conditions of frailty that are prevalent in the oldest ages (43–45), while continuing the effort to postpone the onset of fatal degenerative diseases.

A failure to concentrate on what appear at this time to be immutable age-dependent causes of frailty, could result in longer life but worsening health—that is, declining active life expectancy (46–48). Conversely, a successful effort to postpone morbidity and simultaneously lessen the adverse effects of nonfatal but highly disabling conditions would result in a reduction in the duration of frailty for that part of the population that has already approached its biological limits; a reduction in morbidity and disability rates among those who experience long-term nonfatal conditions; and an overall increase in active life expectancy.

In sum, we have shown that further gains in life expectancy will occur but these gains will be modest, even if major causes of death (that is, cancer and heart disease) are eliminated. However, it is not clear whether a longer life implies better health. In fact, we may be trading off a longer life for a prolonged period of frailty and dependency—a condition that is a potential consequence of successfully reducing or eliminating fatal degenerative diseases. Current research efforts by the medical community are focused on prolonging life rather than preserving and improving the quality of life. An obvious conclusion, therefore, is that the time has come for a shift toward ameliorating the non-fatal diseases of aging.

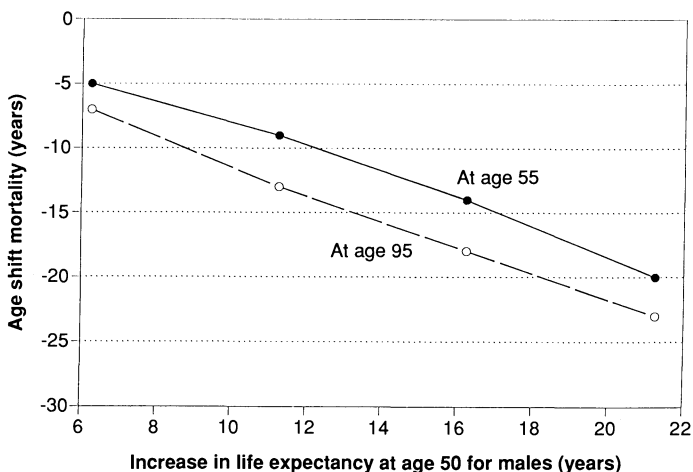


Fig. 5. Age shift in mortality for U.S. males aged 55 and 95 (from 1985) required to increase life expectancy at age 50 to 30 to 70 years.

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The Interdiffusion of Polymers

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In contrast to interdiffusion in simple liquids, interdiffusion of polymeric chains is dominated by their intertangement and their large size. These properties profoundly reduce both the molecular mobilities and the role of entropy in driving the mixing. The resulting diffusional processes have only recently been studied. Such studies reveal a wide spectrum of behavior ranging from acceler-

ated interdiffusion (for strongly compatible chains) to its suppression below the critical point for phase separation. Effects that are still poorly understood include the initial disposition at interfaces of the chains' ends (through which diffusion proceeds by reptation) and the need for cooperative motion, which can strongly magnify local friction.

MIXING OF LIQUIDS IS COMMONPLACE. AT THE MOLECULAR level, it takes place by diffusion: to what extent and how rapidly liquids interdiffuse depend on their chemical affinity and on the mobility of their molecules. For simple liquids (composed of small molecules), mixing is classical and well understood (1, 2). In the case of polymers, on the other hand (both synthetic chains and flexible biopolymers), the large size of the molecules together with their intertangled nature result in extremes of sluggishness and molecular incompatibility (or, in rare cases, extreme compatibility). For these reasons, it is only recently that

insight into the process of mixing different polymers, based on direct measurements of their interdiffusion, is emerging.

The connected, chain-like nature of polymer molecules leads to entanglements. These arise because the backbones of the polymer chains cannot cross through each other (Fig. 1); motion involving such mutual crossing is forbidden. This effect is most marked in undiluted polymeric liquids (3), where every chain is highly interpenetrated by others. In such a polymer melt (N monomers per chain), each chain adopts on average an open, random, coil-like conformation (4, 5). For typical values of N in the range 10^3 to 10^4 , only about 1% of the volume of such a coil is occupied by the monomers of the chain itself: the rest of the coil volume is interpenetrated and filled by segments belonging to other chains.

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