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**Demographic Perspectives
on Human Senescence**

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ESTIMATING THE UPPER BOUNDS to human longevity is an inherently fascinating quest that has drawn the attention of scientists from a number of disciplines. The possibility of modifying the rate of senescence and prolonging youth by altering the expression of age-related diseases remains a topic of great interest and speculation. Demographers and government planners face the more practical problem of forecasting survival and estimating the size of future cohorts of older persons.

Although there is no doubt that the aging of populations will occur (Kinsella and Taeuber, 1993; Olshansky, Carnes, and Cassel, 1993), current debate in demography concerns the extent to which individual aging (implied in the measure of life expectancy) may continue (for example, see Manton, Stallard, and Tolley, 1991; Olshansky, Carnes, and Cassel, 1990). The distinction between competing estimates of longevity has important policy implications. Some researchers predict that life expectancy in the United States will approach 100 years during the next century, while others suggest that we are already approaching the practical limits of human longevity. If the more optimistic scenarios of life expectancy are correct, the size of the beneficiary population—those aged 65 and older—will be significantly larger in the near future than anticipated by official government forecasts. Further, if current morbidity rates remain unchanged, the size of the most frail and disabled subgroup of the population—those aged 85 and older—will also be larger than anticipated. Thus, it is critically important to resolve differences between these competing estimates of human longevity.

Demographic approaches to modeling and forecasting mortality are most often based on the observation of short-term trends in death statistics,¹ with the underlying assumption that the future will be some variation of recently observed historical trends (Ahlburg and Vaupel, 1990; Bell, Wade, and Goss, 1992; Day, 1992; Guralnik, Yanagashita, and Schneider, 1988; Lee and Carter,

1992; Manton, Stallard, and Tolley, 1991; Olshansky, 1985; Vaupel and Gowan, 1986). The extrapolation method is also being used by the Social Security Administration (SSA) (Bell, Wade, and Goss, 1992) and the Bureau of the Census (Day, 1992) to estimate the size and age structure of the older population of the United States. The time frame used as the basis for these forecasts is the past quarter century—a period when death rates in the United States declined at older ages more quickly than during any previous period of comparable length. Is it reasonable to expect that these trends in old-age mortality can be sustained or possibly even accelerated, or are they an anomaly? More importantly, should this method of forecasting be continued?²

Some recent models of mortality change have suggested that the SSA and Census Bureau have adopted mortality forecasting assumptions that are too pessimistic (Ahlburg and Vaupel, 1990; Guralnik, Yanagashita, and Schneider, 1988; Manton, Stallard, and Tolley, 1991).³ The more optimistic scenarios offered by these researchers are based on the premise that future declines in old-age mortality will occur at a more accelerated pace than those observed in the past. Lee and Carter (1992) demonstrated that if empirical models are used to extrapolate past mortality trends (for the entire twentieth century) into the future, life expectancy will increase at a faster pace than that assumed by the SSA in their latest forecast. Other scientists, however, have argued that gains in life expectancy in low-mortality populations should begin to decelerate because of entropy in the life table—a phenomenon well documented in the literature (Keyfitz, 1985; Horiuchi, 1989; Olshansky, Carnes, and Cassel, 1990). As noted by Carnes and Olshansky (1993), theories of senescence from evolutionary biology imply that another law of diminishing returns may apply to human mortality—the hypothesized presence of genetically influenced declines in physiological maintenance and repair mechanisms.

Theoretical bounds to human longevity

Two basic arguments have been set forth to explain why life expectancy will be considerably greater in the future than is currently anticipated. One group (Ahlburg and Vaupel, 1990; Vaupel and Gowan, 1986) estimates that period life expectancy at birth in the United States will reach 100 years sometime in the next century and that cohort life expectancy at birth for children born after 1982 in the United States is already at 100. Their estimates presume that death rates will decline by 2 percent at every age for each of the next 100 years. The 2 percent assumption derives from the observation that death rates from vascular diseases have declined precipitously in the United States from 1968 to about 1982, and the belief that these unprecedented declines will continue at every age for each of the next 100 years. Another group (Manton, Stallard, and Tolley, 1991) also argues for a 100-year life expectancy in the near future. They reason that the higher life expectancies attained by subgroups exhibiting the most favorable

mortality schedules not only can be achieved, but can be improved upon by everyone in the population. The mortality assumptions made by these two schools of thought are considered by their proponents as not only biologically plausible, but realistically achievable in the next century—and needing therefore to be incorporated into official government forecasts. In this article, we consider each of these methods of estimating human longevity in the context of the observed mortality record and in terms of their biological plausibility in light of evolutionary theories of senescence.

Sustained mortality declines of 2 percent

The mortality record of the United States in the twentieth century can be summarized from life tables and age-adjusted central death rates published by the Social Security Administration (Bell, Wade, and Goss, 1992) and the National Center for Health Statistics (1992). Age-adjusted central death rates for males and females in the United States in 1900 were 2,415.5 and 2,198.7 per 100,000, respectively. By 1990, these rates had declined to 1,022.3 for males (a 57.7 percent decline) and 614.0 for females (a 72.1 percent decline). For the first 90 years of the twentieth century, the annual average decline in the central death rate was 0.98 percent for males and 1.37 percent for females. At the same time, the expectation of life at birth increased for males from 46.4 to 71.6 years (a 25.2-year increase) and for females from 49.0 to 78.7 (a 29.7-year increase). The majority of the gain in life expectancy at birth in the twentieth century can be attributed to rapid and sustained declines in mortality at younger ages (see Table 1).⁴

TABLE 1 Annual average percent decline in age-adjusted central death rates at selected ages and time periods: US males and females, 1900–90

	1900–36	1936–54	1954–68	1968–90	1900–90
Males					
0–14	2.89	4.74	1.66	3.75	3.28
15–24	1.83	3.14	–.32	1.46	1.67
25–64	.87	1.69	–.18	1.88	1.12
65+	.21	1.15	–.32	1.21	.56
Total	.81	1.60	–.19	1.48	.98
Females					
0–14	3.11	5.01	1.72	3.60	3.40
15–24	1.90	6.81	.31	1.46	2.55
25–64	1.07	3.30	.62	1.64	1.59
65+	.33	1.84	.79	1.27	.93
Total	.95	2.54	.79	1.45	1.37

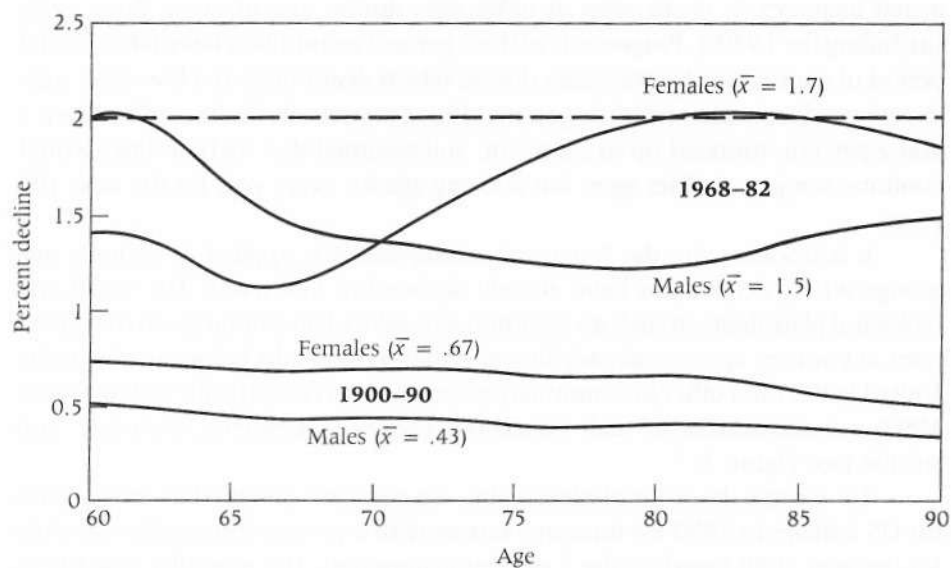
Although the mortality trend in the twentieth century has been toward declining death rates, the observed mortality record of the United States also illustrates that the pattern of mortality change across time and within age groups has not been uniform (Brody, 1985). For example, in the first half of this century large absolute and sustained declines in the death rate were concentrated at younger ages. A second trend emerged from 1954 to 1968, when death rates declined at a much slower pace for females and actually increased for males. After 1968 a third trend emerged, characterized by rapid and unexpected declines in death rates at most ages. The 1968–82 period was unique in that the mortality reductions were concentrated at ages 60 and older, primarily reflecting delays in mortality from vascular diseases (Olshansky and Ault, 1986).⁵ This brief and unique period of US mortality has captured the attention of demographers and epidemiologists and is the foundation for the optimistic assumptions used to predict future mortality.⁶ Between 1968 and 1982, annual average declines in age-adjusted central death rates for males and females in the United States were 1.63 and 1.90, respectively. More recently, from 1982 to 1990, a deceleration of the mortality decline has occurred, with the annual average decline in death rates reduced to 0.93 percent for males and 0.27 percent for females.

After a century of sustained declines in death rates at younger and middle ages in the United States, about 92 percent of all babies born today will survive up to and beyond 50 years of age. Consequently, any further declines in mortality at younger and middle ages will result in only small increases in life expectancy (Olshansky, Carnes, and Cassel, 1990; Vaupel, 1986). Future increases in life expectancy will primarily result from declines in death rates where they still remain at high levels—in the oldest ages (mostly between ages 60 and 90).

The historical trend in mortality at older ages reveals an even less stable pattern than that observed at younger and middle ages. From 1900 to 1990, the average percentage decline in the annual conditional probability of death, $q(x)$, for the population aged 60 to 90 years of age was 0.43 for males and 0.67 for females (see Figure 1).⁷ During the period of most rapid decline (1968–82), the annual average decline in $q(x)$ was 1.7 percent for females and 1.5 percent for males. In twentieth-century US history, 2 percent annual declines in $q(x)$ for males and females have occurred only briefly for a few older age groups, and the pattern of change at older ages has been uneven throughout the century. In fact, death rates between ages 60 and 90 have actually increased during about one of every three years—including several increases after the period of rapid declines observed from 1968 to 1982.⁸ Although the general trend in US mortality throughout this century has been downward, fluctuating mortality has been the rule rather than the exception among every older age group (see Figure 2).

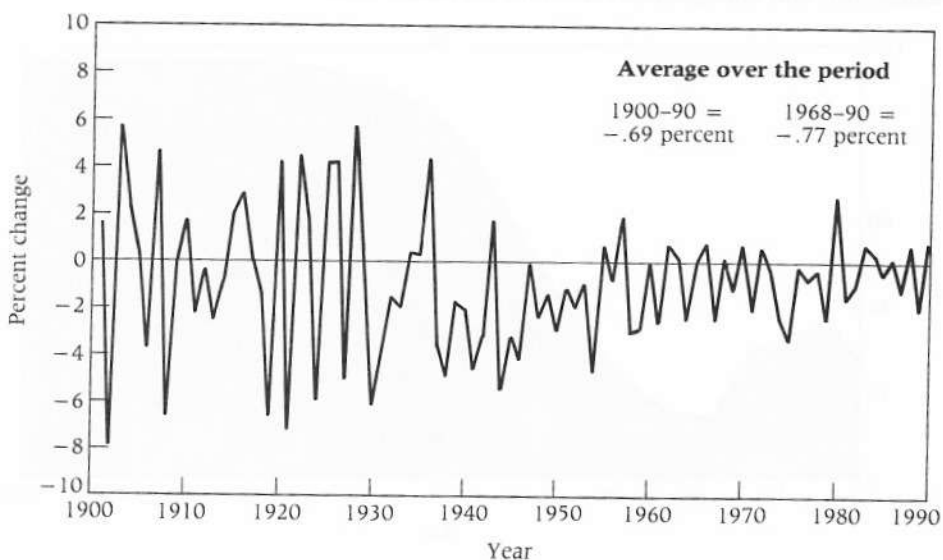
The observed mortality record of the United States simply does not support the assumption that death rates have declined by 2 percent annually at every age. Instead, the observed mortality record shows (1) fluctuating patterns of change in death rates across time and within age groups; (2) observed annual average

FIGURE 1 Annual average percent declines in the conditional probability of death $[q(x)]$ by single year of age between ages 60 and 90 for US males and females, 1900-90 and 1968-82, and average declines in $[q(x)]$ over the age span 60 to 90 for the same time periods



SOURCE: Calculated from data published by Bell, Wade, and Goss (1992).

FIGURE 2 Annual percent change in the conditional probability of death $[q(x)]$ at age 65: US females, 1900-90



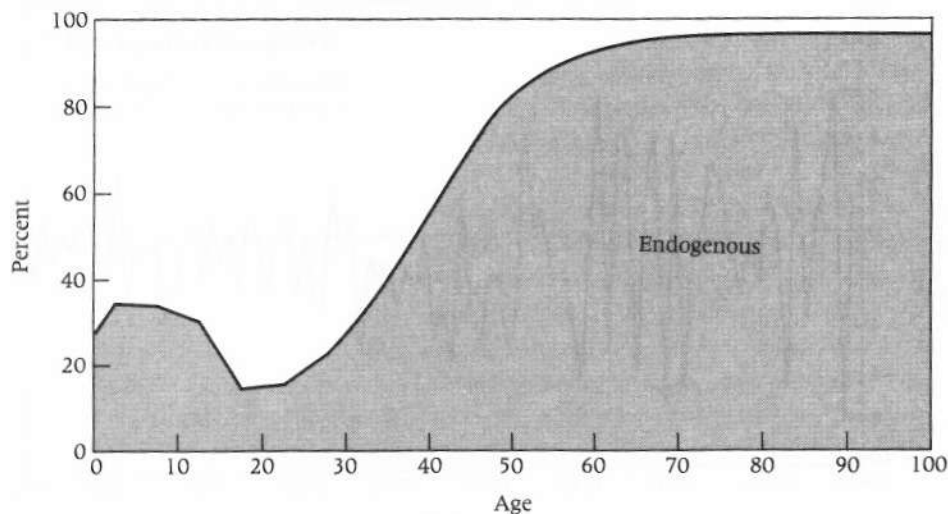
SOURCE: Bell, Wade, and Goss (1992).

declines in mortality of about 0.5 percent throughout this century for the population 60–90 years of age; (3) maximum declines of 1.6 percent during the period of most rapid declines in death rates at older ages (1968–82); and (4) actual increases in death rates at older ages during one of every three years (including the 1980s). Proponents of the 2 percent assumption have taken a brief period of anomalous US mortality during which death rates at a few older ages experienced an uncharacteristic pattern of near monotonic declines of between 1 and 2 percent, rounded up to 2 percent, and assumed that such declines would continue not just at older ages, but at every age for every year for the next 100 years.

It is unclear why the 2 percent assumption was applied to younger age groups where death rates have already declined to low levels. The social and biological plausibility of such an assumption is difficult to defend given that death rates at younger ages are already low and that most deaths before age 30 in the United States (and other low-mortality populations; World Health Organization, 1992) are attributable to such exogenous causes as accidents, homicide, and suicide (see Figure 3).⁹

To address the issue of plausibility, we compare observed mortality rates for US females in 1990 for three age ranges with a projected mortality schedule for the year 2080 based on the 2 percent assumption. The mortality schedule is separated into three age ranges so that the magnitude of the mortality reduction

FIGURE 3 Percent of total mortality at each age attributable to exogenous and endogenous causes for US males, 1988



SOURCE: Calculated from data published by the National Center for Health Statistics (1992); US males, 1988.

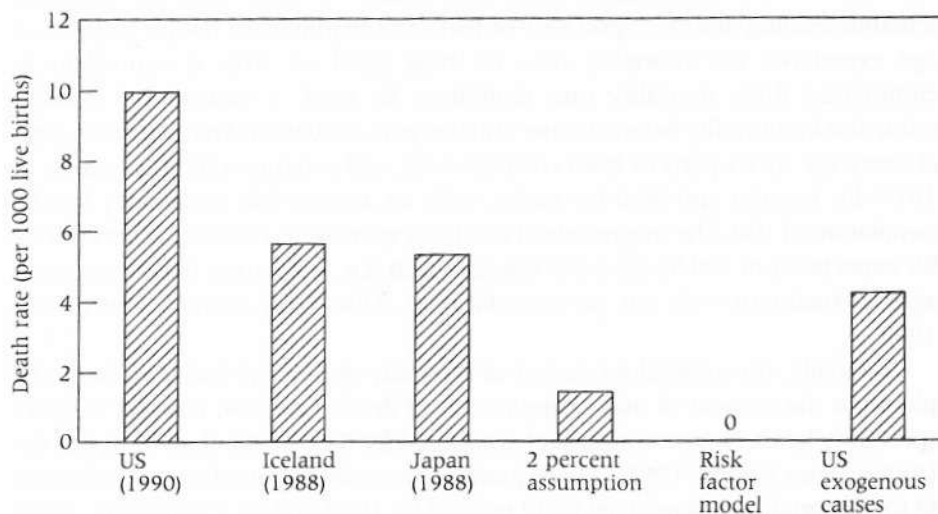
associated with the 2 percent assumption is evident, and so that the proximity of the 2080 mortality schedule to a death rate of zero is obvious.

A sustained 2 percent reduction in mortality for ages less than 30 years produces death rates near zero by the year 2080—with infant mortality reduced to a rate of 1.4 per thousand live births (see Figure 4). The lowest infant mortality rates ever observed anywhere are in Japan (5.3) and Iceland (5.6) (World Health Organization, 1992). Although advances in neonatal intensive care and therapeutic interventions have been impressive over the past quarter century, it is not currently possible to eliminate the conception and/or birth of children with life-threatening congenital anomalies that terminate life early, nor has it been possible to eliminate external causes of death among infants. The infant mortality rate in the United States from congenital anomalies and external causes¹⁰ (4.2 per 1000 live births) probably represents a practical threshold below which infant mortality rates cannot fall in the short term¹¹ (National Center for Health Statistics, 1992).

For the rest of the population under age 30, the 2 percent assumption leads to mortality rates less than one-half the lowest mortality rate observed at any age—the rate for those at ages just prior to puberty (see Figure 5). The 2 percent assumption would require the elimination of all endogenous mortality and the near complete elimination of exogenous mortality before age 30.

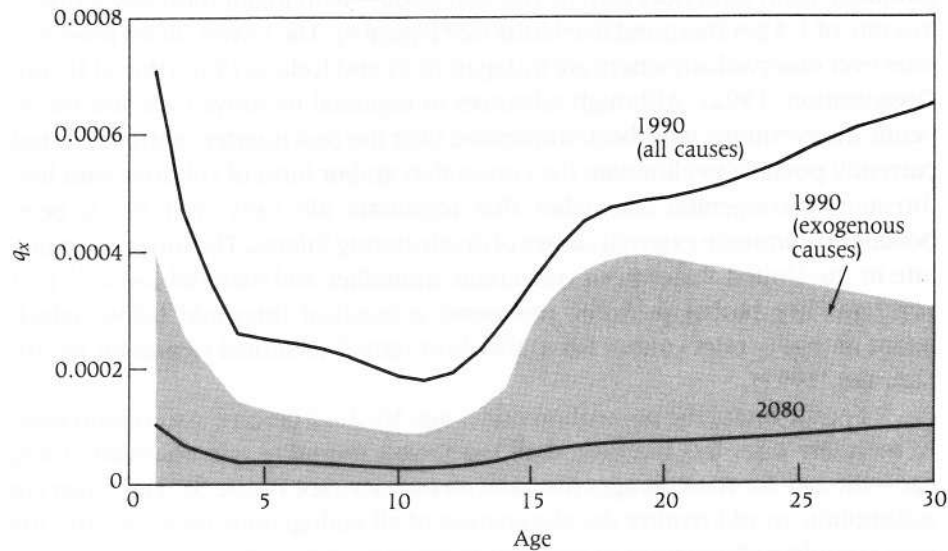
By the year 2080, the 2 percent assumption also leads to mortality risks for people between ages 30 and 70 that are comparable to those observed for young

FIGURE 4 Observed infant mortality rates in Iceland and Japan and in the United States under various assumptions



SOURCE: National Center for Health Statistics (1991); World Health Organization (1992).

FIGURE 5 Conditional probability of death [$q(x)$] for US females aged 1–30: Observed rates for total mortality and endogenous causes in 1990, and projected rates in 2080 assuming an annual decline of 2 percent at each age between 1990 and 2080

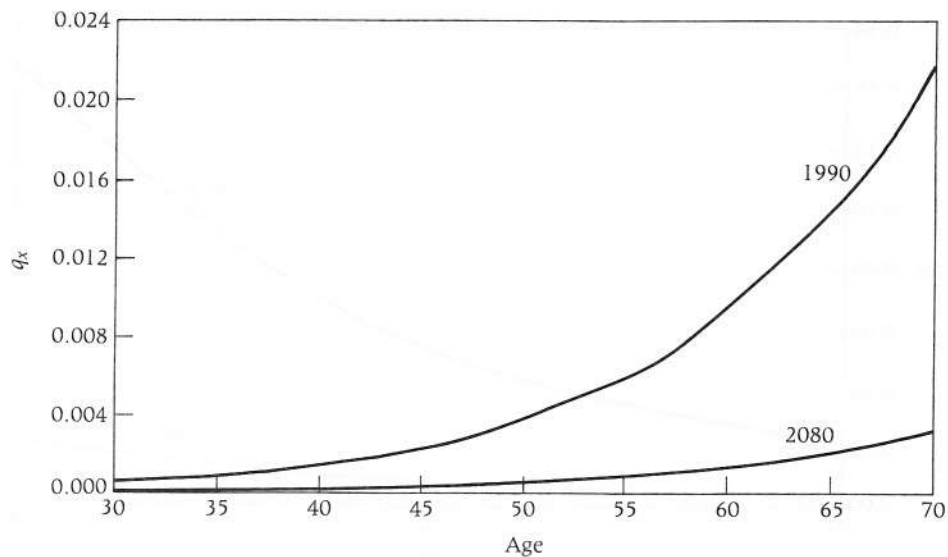


SOURCE: Bell, Wade, and Goss (1992); National Center for Health Statistics (1992).

children and teenagers (see Figure 6). For those aged 70–90, mortality would be comparable to that experienced by individuals who are today in their prime reproductive and childrearing years (see Figure 7). Note that the mortality schedule leading to a life expectancy of 100 years requires that people 90 years of age experience the mortality risks of those aged 65. This is equivalent to eliminating three mortality rate doublings. In total, a sustained 2 percent reduction in mortality between now and the year 2080 would reduce death rates at every age by 85 percent from current levels, and produce a life expectancy of 101.9 for females and 98.0 for males, with an average life expectancy for the population of 100. The magnitude of mortality reductions necessary to produce a life expectancy of 100 by the 2 percent approach (i.e., 85 percent declines at every age) is consistent with our previous findings (Olshansky, Carnes, and Cassel, 1990).

Finally, the anomalous period of mortality declines (1968–82) should be placed in the context of underlying causes of death. In 1968, vascular diseases (primarily heart disease and stroke) accounted for 56 percent of all deaths in the United States (Wade, 1992). Subsequently, the contribution of vascular diseases to total mortality was reduced to 49 percent by 1982 and 42.7 percent by 1990. Deaths and death rates from all forms of cancer combined, however, have increased almost every year since 1968 in the US, contributing 16 percent to total

FIGURE 6 Conditional probability of death [$q(x)$] for US females aged 30–70: Observed rates for total mortality in 1990 and projected rates in 2080 assuming an annual decline of 2 percent at each age between 1990 and 2080



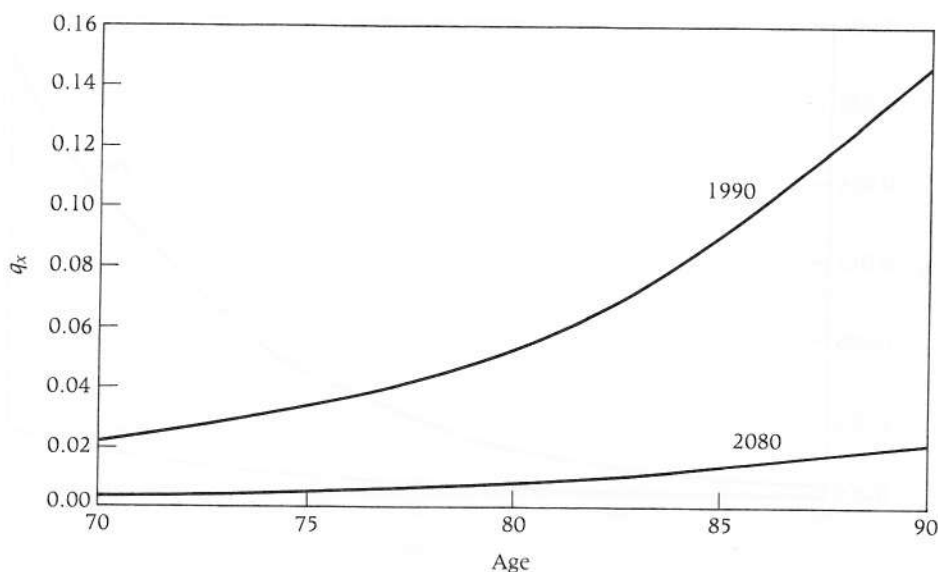
SOURCE: Bell, Wade, and Goss (1992).

mortality in 1968, 22.5 percent by 1982, and 24.2 percent by 1990 (Wade, 1992). Since declining death rates from vascular diseases are characteristic of declining mortality in other low-mortality populations, assumptions about future mortality should reflect the probable coincident rise in death rates from cancer.

If future annual declines in total mortality of 2 percent are concentrated among vascular diseases, the only major disease category showing declining death rates at older ages in this century, then all vascular diseases would be eliminated by the year 2010 (see Figure 8).¹² With mortality declines from vascular diseases exhausted, subsequent 2 percent reductions would have to come from cancer. However, cancer is the only major cause of death that has actually risen throughout the twentieth century, with no evidence that it will decline in the future. Further, it is possible that declining mortality from vascular diseases could lead to rising cancer death rates.

Even if the annual average changes in death rates observed for the population aged 60 and over during the period of rapid declines from 1968 to 1982 were to continue at their sex-specific observed rate of decline at every age (the 2 percent assumption) for every year until 2080 (i.e., 1.7 percent declines for females and 1.5 percent declines for males), life expectancy at birth for the entire population would be 94.7 years—not 100 years. Given that it is practically and biologically implausible to eliminate all deaths in the first 30 years of life, a 100-

FIGURE 7 Conditional probability of death [$q(x)$] for US females aged 70–90: Observed rates for total mortality in 1990 and projected rates in 2080 assuming an annual decline of 2 percent at each age between 1990 and 2080



SOURCE: Bell, Wade, and Goss (1992).

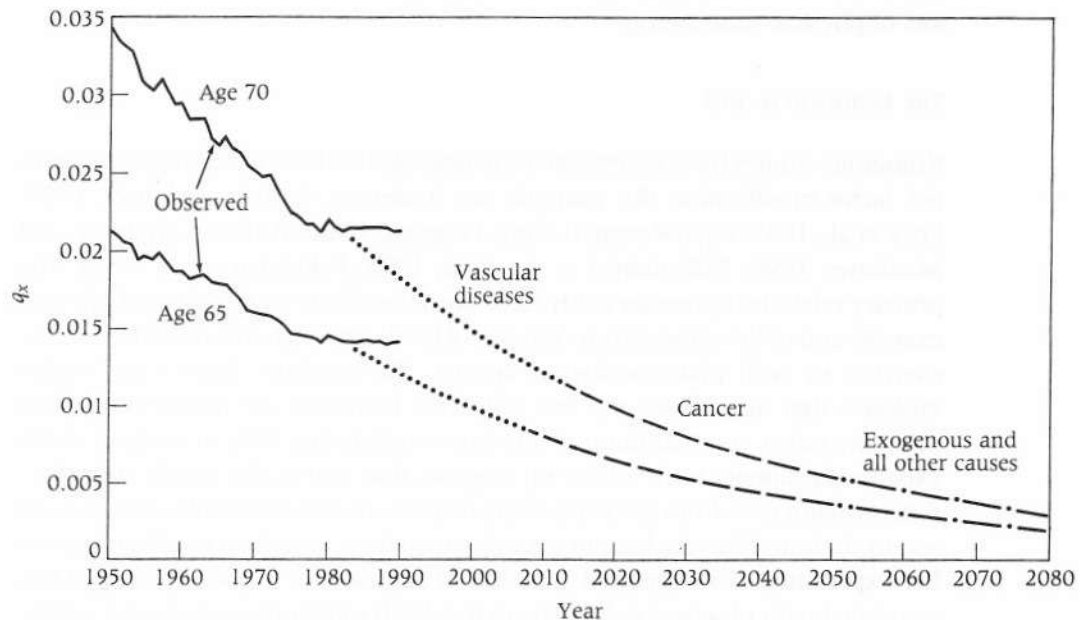
year life expectancy in the next century would actually require larger mortality declines for the older population than the 85 percent decline resulting from the 2 percent assumption. The difference between the 1.6 percent decline actually observed from 1968 to 1982 in the United States and the 2 percent assumption is only 0.4 percent annually. This small difference, however, leads to vastly different mortality schedules in the year 2080, with a 5.3-year difference in the expectation of life at birth and an enormous difference in the estimated size of the older population.

A 2 percent annual decline in mortality at every age sustained over a century results in the elimination of almost all endogenous mortality at older ages, and almost all endogenous and exogenous mortality at younger ages—leaving death rates close to zero. Strong justification is required for assumptions that lead to the elimination of all vascular diseases, reversing historical trends in mortality from cancer, and achieving the near elimination of mortality before age 30.

Estimating human longevity by modeling risk factors

According to the risk factor model of human mortality adopted by Manton and colleagues (1991), the expectation of life at birth in the United States could reach 100 years sometime early in the next century. This theoretical estimate of the

FIGURE 8 Conditional probability of death [$q(x)$] for US females at ages 65 and 70: Observed rates 1950–90 and projected rates 1983–2080



SOURCE: Special tabulation of conditional probabilities of death, by year, from the Social Security Administration.

upper limit to life expectancy was arrived at by two different methods. First, standardized mortality ratios (SMRs) were calculated for selected subgroups of the population known to have low mortality rates relative to the standard US population in 1986. The most favorable SMRs observed among these selected subgroups were then collected and applied to the entire population assuming that everyone could achieve these favorable SMRs.

In the second approach, the relation of mortality and time-varying covariates was modeled as two interrelated processes. Essentially, this was accomplished by redefining senescence from a largely immutable force to an exogenous process that is modifiable by controlling disease processes. The newly defined exogenous disease processes were then hypothetically eliminated under the assumption that scientific breakthroughs would transform senescence to an exogenous cause. Central to this theoretical estimate of the upper limit to life expectancy are the assumptions that death rates under age 30 will be reduced to zero, the variance of all risk factors will be reduced to zero (i.e., heterogeneity for mortality risks will be eliminated), and that risk factor profiles observed for the most healthy subgroup of the 30-year-old population from the cohorts of the Framingham, Massachusetts Heart Study will have the same beneficial effect on the rest of the population and be held constant at those levels for everyone for the

duration of life (Manton, Stallard, and Tolley, 1991: 624). Further, it is assumed that everyone will adopt a "perfect" lifestyle from birth to death and avoid the loss of physical functioning.

The historical record

Numerous studies have investigated the possible health benefits associated with risk factor modification (for example, see Anderson, Castelli, and Levy, 1987; Frick et al., 1987; Lipid Research Clinic Program, 1984; Muldoon, Manuck, and Matthews, 1990; Paffenbarger et al., 1986, 1993; Pekkanen et al., 1992). The primary relationships under study have been the effects on mortality of physical exercise and of lowering serum cholesterol levels (through dietary modification, exercise, or with pharmacological agents). The evidence from these studies indicates that after about the first ten years following the intervention, total mortality either stays constant or increases—producing little or no gain in life expectancy. Longer term follow-up suggests that statistically significant reductions in death rates from coronary artery disease are associated with reductions in serum cholesterol levels, but not enough to produce more than marginal gains in life expectancy. The apparent paradox of stagnant or higher total mortality associated with observed reductions in the death rate from heart disease (attributable to the interventions) occurs because of increasing death rates from other causes (referred to as competing causes)—particularly cancer and exogenous mortality (e.g., accidents, homicide, and suicide).

Recent studies indicate that it is premature to determine whether there is a causal relationship between lowering serum cholesterol levels and elevated mortality from cancer and exogenous causes. Epstein (1992: 9) concluded that ecological, prospective, and intervention studies do not definitively support the claim that serum cholesterol reduction—while reducing cardiovascular mortality—also increases the risk of dying from other causes. The absence of a plausible mechanism linking cancer and exogenous causes of death to lower serum cholesterol levels was the reason given for this conclusion. However, Epstein (p. 7) also acknowledged that "the increase [in mortality] overtly exists for cancer and violent deaths," and that the issue of causality "does not enter into these considerations because the data have to be taken at face value for the purpose of explaining the balance between specific causes of death and total mortality." Muldoon and colleagues (1990) demonstrated with a meta-analysis that in almost all of the clinical trials that included both dietary modification and drug treatment, all-cause mortality either remained constant or increased—with the increase attributable to statistically significant jumps in mortality from cancer and exogenous causes. Although the link between low serum cholesterol levels and cancer disappears with extended follow-up, the effect on exogenous mortality remains.

Studies that link physical activity to coronary heart disease and total

mortality demonstrate that the difference in life expectancy between the most physically active segment of the population and otherwise comparable individuals who are sedentary throughout life is only one to two years (Paffenbarger et al., 1986, 1993). Other studies have also shown that subgroups of the population adhering to multiple health practices live longer than those with few or no health practices (Breslow and Breslow, 1993), but with differences in life expectancy that are much smaller than those estimated from risk factor models (Manton, Stallard, and Tolley, 1991). Even the healthiest subgroup of these study populations, however, did not have life expectancies that exceeded the mid-80s. In recent studies using healthy subgroups from the Longitudinal Study on Aging, researchers have shown that among those who adopt the most health practices and avoid loss of physical functioning throughout life, the maximum life expectancy achieved was 87–95 years (Grigsby and Bailey, 1993; Rogers and Carrigan, 1993). These studies demonstrated that improved risk factor profiles have neither a synergistic nor an additive effect as predicted by the risk factor model, but instead follow an apparent law of diminishing returns that is fundamentally different from the known phenomenon of entropy in the life table (Horiuchi, 1989; Keyfitz, 1985; Olshansky, Carnes, and Cassel, 1990).

The historical mortality record from clinical trials and retrospective studies on the mortality benefits associated with adopting selected optimum risk factor profiles (e.g., exercise, reductions in serum cholesterol through dietary modification or pharmacological agents, and the adoption of healthy lifestyles and avoidance of the loss of physical functioning) indicates that even if the entire population were to adopt optimum risk factor profiles, mortality declines from some causes of death may occur, but such declines alone would not be of sufficient magnitude either to produce large increases in life expectancy or to compensate for increasing death rates from cancer and exogenous causes.

Several other aspects of the risk factor model deserve further attention. First is the question of how plausible it is to assume that everyone in the population will actually decide to adopt (or practically be able to adopt) optimum risk factor profiles. The adoption of perfect lifestyles from birth to death for any subgroup of the human population (such as that of the United States) would probably require the near elimination of all racial, ethnic, social, and economic inequities, equal access to high-quality health care for all, "correct" decisions by everyone on when to seek health care, "correct" decisions on dietary habits that must be observed faithfully throughout life, and the presumption that it is possible to define individualized perfect lifestyles for a heterogeneous population.

As indicated earlier, a central assumption of the risk factor model is that physical functioning must be maintained for the duration of life by everyone in the population. The historical disability record of humans is characterized by numerous diseases and disorders that cause disability among the majority of survivors into older ages, and that are currently unaffected or marginally influenced by lifestyle changes or medical intervention (Brody, 1985; Cassel et

al., 1990). These include Alzheimer's disease, vision and hearing impairments, osteoarthritis, osteoporosis, and Parkinson's disease, to mention a few. The reclassification of senescence as an exogenous cause of death through rapid increases in scientific knowledge, as anticipated by Manton and colleagues (1991: 622), could occur only with knowledge that permits the elimination of all currently immutable diseases and disorders of senescence, and, once eliminated, the avoidance of any new (or infrequently observed) diseases and disorders of senescence associated with extended survival into advanced ages.

The mortality schedule resulting from the risk factor model that leads to a life expectancy at birth of 100 years contains several noteworthy differences from currently observed mortality schedules. All mortality for the population aged 30 and under would have to be reduced to zero, the proportion of each birth cohort surviving to age 65 would have to increase from an observed average of 83 percent to 97 percent, and the proportion surviving to their 85th birthday would have to increase from an average of 31 percent to 84 percent. Survival to age 85 would need to increase nearly fourfold for males (from 22 percent to 84 percent) and more than double for females (from 39 percent to 83 percent). Although further reductions in death rates at middle ages from endogenous causes could extend survival up to age 65, a survival of 97 percent of each birth cohort past that age requires the near elimination of all endogenous and exogenous mortality before age 65.

Finally, the risk factor model of Manton and colleagues (1991: 619) indicates that according to data from Kaplan et al. (1987), a selected subgroup of males from the Alameda County, California population "on average survive[s] to 98.0 years—24.2 years longer than the US male population at large." Manton and colleagues present hypothetical survival curves for this and other populations, illustrating that approximately 15 percent of the males in the Alameda County study population survive at least to the age of 115, and 5–10 percent of several other "healthy" populations survive past age 110 (Manton, Stallard, and Tolley, 1991: Fig. 1, p. 629). In fact, using the actual data from the Alameda County Study Population (kindly provided to us by Dr. George Kaplan of the Human Population Laboratory), we note that no member of this population has ever lived beyond the age of 109, and even among those with the healthiest lifestyles life expectancy did not exceed the mid-80s (Breslow and Breslow, 1993). The conflict between a "traditional" approach based on actual data and the "visionary" approach proposed by Manton and colleagues where senescence is hypothetically eliminated suggests that the validity and underlying assumptions of the risk factor model should be questioned.

Evolutionary perspectives on demographic models of mortality

One of the underlying premises of the evolutionary view of human mortality is the recognition that current mortality trends for humans (i.e., within the past 200

years) represent a significant departure from patterns that have been present since modern humans arose about 100,000 years ago—patterns characterized by high attrition at younger ages and the relatively infrequent expression of senescent mortality. From this perspective, the period of rapid declines in death rates at middle and older ages in the past quarter century represents a new mortality trend embedded within a broader period of new mortality patterns for the species. In this century alone, humans have accomplished what no other species has achieved. We have begun to control the forces of natural selection that have operated on our species for thousands of years by reducing early and middle-age mortality rates to the point where the vast majority of each birth cohort survives into older ages. As a result, in low-mortality countries close to 95 percent of each birth cohort survives past age 30, and over 85 percent of each birth cohort survives past age 60—twice the age required by our life history strategy for successful reproduction (Carnes and Olshansky, 1993). Further, in a single century the cause-of-death structure for humans has been completely transformed, with diseases and disorders of senescence replacing the fatal diseases that have historically precluded survival into older ages. We believe it is important for scientists involved in forecasting human mortality to expand their time frame of reference from the 15–20 years preceding their forecast to an evolutionary time frame encompassing the period from the origin of modern humans to the present. This broader time scale of some 100,000 years reveals the new and unusual nature of current trends in human mortality.

From evolutionary theories of senescence come several predictions about the magnitude and timing of senescent-related mortality that are relevant to demographic models of human mortality. According to these theories, there is a link between the reproductive period of the species and the time course of senescence. In a genetically heterogeneous population (such as humans), senescent-related mortality is predicted to follow a species-specific pattern beginning with the onset of the reproductive period. This may be referred to as a mortality signature for the species, a concept that dates back to Raymond Pearl (1922). We suggest that basic mathematical properties are associated with these signatures that involve the age-at-onset of senescent mortality, the rate at which senescent mortality increases, and the initial senescent death rate at the beginning of the reproductive period. These underlying patterns of senescent mortality should be largely immutable and should have mathematical properties (which can be estimated) that link species-specific senescent mortality patterns to the length and timing of the species' reproductive period. This relationship between reproduction and senescence is unique to each species and is thought to be a product of environmental conditions that prevailed when that species arose. The hypothesized inevitable presence of senescent mortality at the beginning of the reproductive period and its predictable rate of increase for humans conflict with demographic models that predict the elimination of all mortality before age 30 (Manton, Stallard, and Tolley, 1991), or the acceleration of mortality declines at older ages in the future (Ahlburg and Vaupel, 1990). The theory is also at odds

with extrapolation models adopted by the SSA and the Census Bureau, which choose as the basis of their forecasts the extension of the most rapid period of mortality declines ever observed for older segments of the human population. Furthermore, given that exogenous mortality has always been one of the primary forces that precluded survival into older ages for most living organisms, demographic models that predict the elimination of exogenous mortality (Manton, Stallard, and Tolley, 1991) or dramatic reductions in its occurrence at any age (Ahlburg and Vaupel, 1990) require strong justification.

The demographic models also predict that mortality risks for those aged 30–70 years will be transformed into risks typical for teenagers and young adults, and that those aged 70–90 will have the mortality risks of cohorts just beginning to experience the consequences of decline in somatic maintenance (i.e., 30–40-year-olds). Although humans have been able to achieve remarkable mortality declines by modifying the physical environment, altering lifestyles, and introducing modern medical technology, the one factor that remains beyond current control is the genome itself. From evolutionary theory one may predict that unless the genome or its expression can be controlled, the time-dependent but stochastically influenced expression of diseases and disorders associated with senescence is inevitable. At the individual level, humans who survive beyond the ages required by our species' life history strategy to ensure reproductive success cannot escape accumulated DNA damage and inevitable declines in somatic maintenance and repair that lead to death. At the population level, basic population genetics implies that once extended survival into older ages is achieved for almost everyone, genetic heterogeneity is increased—thus enhancing the opportunity for the expression of new or infrequently observed diseases.¹³

Without altering the basic rate of senescence, it is extremely difficult to envision mortality schedules that permit 30–60-year-olds to experience the mortality risks of teenagers who are the most protected from senescent mortality. Inevitable declines in somatic maintenance and repair make it even more improbable that humans experiencing 60 to 90 years of unavoidable DNA damage (see Boulikas, 1992; Lindahl, 1993) could miraculously achieve the mortality risks of those aged 30 to 40 who are just beginning to face the consequences of accumulated molecular damage and declining somatic maintenance and repair.

Finally, the risk factor model developed by Manton and colleagues (1991) predicts that the process of senescence will be transformed into an exogenous cause of death that can be eliminated by modern medical technology and improvements in lifestyles. This conclusion is at odds with evolutionary theory, which predicts that senescence is inevitable in all organisms that live beyond the reproductive period (Carnes and Olshansky, 1993; Kirkwood and Holliday, 1979; Kirkwood and Rose, 1991; Williams, 1957). There is no evidence that any species, or any single member of any species, has ever avoided the declines in somatic maintenance and repair that accompany survival into older ages and that eventually lead to death.

A challenge to demographic models

The current debate about upper limits to human life involves those who argue that life expectancy at birth cannot realistically exceed about 85 years (Fries, 1989; Olshansky, Carnes, and Cassel, 1990), and those who contend that life expectancy can reach 100 years early in the next century (Ahlburg and Vaupel, 1990; Manton, Stallard, and Tolley, 1991; Vaupel and Gowan, 1986). Demographers familiar with the relationship between old-age mortality and life expectancy know that the debate is not about the magnitude of mortality declines required to increase life expectancy beyond 85 years. We all agree on this point. The real issue is the plausibility of achieving these declines. In this article we have examined the demographic models in light of both the historical mortality record and prevailing theories of senescence.

The policy implications associated with these competing models are critically important because they involve the size and health status of future survivors to old age. A life expectancy of 100 years—if achieved at any time in the next century as some demographers anticipate—would result in increases in the size of the older population that far exceed the latest official government forecasts. Given the profound impact of the age structure of the population on society and the even larger implications for the health status of an older population, it is critical that we scrutinize the underlying assumptions of the competing schools of thought.

One school maintains that today's major fatal diseases and predisposing risk factors are interrelated, amenable to modification, and represent the primary cause of senescence. For example, smoking is a known risk factor for heart disease, stroke, and some forms of cancer. By reducing just one multiple risk factor such as smoking, mortality rates could theoretically decline simultaneously for several diseases—perhaps by amounts that when added together are equivalent to the elimination of a single disease. This process of improving population-level risk factors could theoretically continue until everyone in the population lived a perfect lifestyle from birth to death. This theory is not only logically appealing, it is also the underlying premise behind various health-promotion and disease-prevention programs in most countries. As a companion to the risk factor model, some demographers have argued that recent declines in mortality in the United States have been at about 2 percent annually at every age, and if such declines continue the expectation of life at birth would soon reach 100 years (Ahlburg and Vaupel, 1990; Vaupel and Gowan, 1986).

Theories developed from evolutionary biology form the basis for a second school of thought. The underlying premise is that senescence and disease do not arise from predetermined genetic programs, but instead are the inadvertent consequence of survival beyond the ages at which the forces of natural selection can operate. That is, the carefully controlled maintenance and repair functions that exist at the genetic level were designed to operate at a level of efficiency necessary to ensure reproductive success. Beyond that point, natural selection is

weak or nonexistent, thus permitting maintenance and repair functions to operate with declining efficiency until death ultimately occurs. Since living organisms are not genetically designed for immortality, senescence is inevitable. Given the selective forces that have historically operated on humans, one would predict that death rates from endogenous causes should first appear at the beginning of the reproductive period and become the dominant force of mortality between ages 30 and 40—a time frame that captures the probable reproductive window for an individual as well as an opportunity to assist in the reproductive success of offspring.

If the risk factor model is correct, then altering a population's lifestyles to favor "optimum" risk factor profiles could theoretically reduce the death rate from numerous diseases simultaneously. Taken together, such declines could work synergistically to reduce total mortality enough to achieve a life expectancy of perhaps 90 years (Grigsby and Bailey, 1993; Rogers and Carrigan, 1993).

If the evolutionary model of senescence is correct, some life-threatening disease or disorder associated with senescence would inevitably result from currently unavoidable declines in physiological function. As diseases and disorders associated with senescence are postponed or as survival improves for those with these diseases, new or infrequently observed diseases and disorders that may be more difficult to conquer should be revealed. Because there is no genetic program for extended survival, it is predicted that fighting diseases and disorders appearing in older ages will be a never-ending battle that becomes progressively more difficult as death rates decline. Although modifying risk factors may produce further reductions in death rates from fatal diseases (as has been observed in low-mortality countries), the longevity benefits should be neither synergistic nor additive. Instead, gains in life expectancy should be limited by the known phenomenon of entropy in the life table and a biologically based law of diminishing returns.

Proponents of the 2 percent approach make the historically inaccurate assumption that mortality declines have occurred at 2 percent annually and that they will persist in the future. Declines in death rates at older ages have reached 2 percent only for a few age groups, and then only for a brief period. The mortality schedules that result from the 2 percent assumption also require the elimination of all endogenous mortality and almost all exogenous mortality before age 30, and the elimination of most endogenous mortality after age 30. There are no observational or theoretical bases for these assumptions,¹⁴ nor are they consistent with theories from evolutionary biology that predict the inevitability of both exogenous causes of death and senescence.

We have a more fundamental disagreement with the risk factor model. Manton and colleagues (1991: 614) have argued that the theoretical limits to human life expectancy must lie near or beyond the expectation of life currently observed by the healthiest subgroup of the population. They further argue that

(1) the population is rapidly adopting optimum risk factor profiles (i.e., health practices and physiological parameters of senescence adhered to and experienced by long-lived subgroups of the population); (2) these profiles will have the same benefit for everyone; and (3) risk factor profiles that exist for exceptionally healthy 30-year-olds will remain at those levels for everyone in the population for the duration of life.

We disagree with the basic premises of the risk profile approach. We contend that in sexually reproducing organisms there is inherent diversity in the physiology of individual members of the population—a genetic diversity that is enhanced by declining old-age mortality and that forms the basis for evolution. A fundamental consequence of genetic heterogeneity is the certainty that individuals will exhibit varying degrees of disease expression, diverse responses to risk factors, and inevitable differences in life expectancy. The creation of a homogeneous population with identical mortality risks (as predicted by the risk factor model)—even if everyone in the population adopted the identical lifestyle that is “optimum” for a subgroup of the population—is biologically impossible. One demographic study has demonstrated that even among genetically identical members of a given species, in this case fruit flies, responses to risk factors can vary (Curtsinger et al., 1992). Rather than assuming that one set of “optimum” lifestyles applies to everyone, it appears reasonable to assume that individualized “optimum” lifestyles exist that correspond with the genetic complexity and diversity of the population.

Thus, although general patterns of senescence are observed within and between species, the stochastic component of senescence combines with genetic heterogeneity to produce inevitable variation in the rate and nature of senescence between individual members of the same species. This would explain (1) why genetically identical twins would not experience exactly the same life expectancy, (2) why a given set of risk factors would not have the same effect on every member of the population, and (3) why the observed age of the longest-lived member of a species cannot be achieved by every member of the population.

We also contend that it is simply impractical to expect every member of the population to adopt an “optimum” risk factor profile—whether that profile is individualized or population based—even if such profiles could be defined and the means developed to permit individuals to achieve them. Although such an effort (if successful) would probably improve the quality of life and might even extend the length of life for some, the results of risk factor trials and retrospective studies suggest that such efforts have only marginal effects on life expectancy. Examination of the observed life expectancies for healthy subgroups of the population has demonstrated that risk factor modification has neither a synergistic nor an additive effect on mortality or life expectancy—a finding that is inconsistent with the risk factor model. Thus, we contend that a practical limit to

life expectancy must lie below that observed among a select few who have both the genetic composition and the economic means to maintain excellent health throughout life.

We are not arguing that diseases and disorders associated with senescence are necessarily immutable. Indeed, it is probable that the onset and progression of fatal diseases will be further postponed (even at older ages), and it may even be possible to do the same for disabling diseases and disorders that are currently immutable (Olshansky et al., 1991). From the biological perspective, however, senescence is inevitable and progressive. This means that as progress is made against earlier-occurring disorders, those that emerge at later ages should be increasingly difficult to conquer. While it may be interesting to speculate about longevity benefits derived from the mathematical elimination of senescence (Manton, Stallard, and Tolley, 1991), official government forecasts should not be based on the premise that senescence will soon be modifiable or eliminated, or on the assumption that everyone will soon adopt optimum risk factor profiles, avoid the loss of physical functioning, and retain the risk factor status of a 30-year-old for the duration of life. There is no scientific reason to believe that any of these conditions can be met, nor is there any indication that a sizable segment of the population is even remotely heading in the direction required by the risk factor model. Furthermore, there is no evidence in the historical disability record of human populations to support the assumption that the lifelong maintenance of physical functioning enjoyed by only a small fraction could be achieved by everyone.

To summarize, the 2 percent assumption and the risk factor model are not consistent with the observed mortality record, theories of senescence that predict progressive declines in somatic maintenance and repair, the broader historical context for recent trends in human mortality, or the evolutionary forces that determine the survival potential of living organisms.

Evolutionary theories of senescence suggest that short of major modifications of the genome to favor longevity, the life expectancy of living organisms should conform to a biological law of diminishing returns based on a unique species-specific relationship between reproduction and senescence. Although there is probably not a genetic program for death, the basic biology of our species, shaped by the forces of evolution acting on us since our inception, places inherent limits on human longevity. We suggest that some demographic models of human senescence produce mortality schedules that appear to be inconsistent with predictions about human mortality from evolutionary theory. Evolutionary theories of senescence have testable hypotheses. Since evolutionary biologists do not study humans, demographers are in a unique position to test some of the predictions about human mortality patterns derived from evolutionary theory. Of particular interest are the mathematical properties of patterns of senescent mortality, and the hypothesized species-specific link between the length of the reproductive period and the age-specific force of senescence.

Notes

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1 Because the pattern of human mortality has changed little during the 100,000 years that humans have inhabited the earth (with the exception of about the last 200 years), mortality trends observed during the past century are considered short-term. However, most official forecasts of mortality made by US government agencies have based their forecasts on trends in mortality observed during the 15–20 years preceding the exercise.

2 Several authors have raised questions about the appropriateness of using extrapolation methods for forecasting mortality; for example Alho and Spencer, 1990; Alho, 1992; van Poppel and de Beer, 1993; Olshansky, 1988.

3 Guralnik and colleagues (1988) did not predict that 2 percent declines in death rates would actually occur. Instead, they speculated about how the future size and age structure of the US population would be influenced by mortality declines of this magnitude. The basis for the 2 percent assumption used by Guralnik et al. was the conclusion by Vaupel and Gowan (1986) that this was possible.

4 The time periods chosen for Table 1 are believed to capture distinct plateaus and declines in mortality in the United States, particularly at older ages.

5 With the exception of a minor increase in mortality for males from 1971 to 1972.

6 Further evidence for this assumption is provided by Kannisto et al. (1993), who demonstrate that mortality declines at older ages have been near 2 percent in many low-

mortality countries for the period 1960 to 1980.

7 Mortality data at older ages in the United States are generally acknowledged to be defective, with errors of age misstatement and faulty assumptions about mortality rates for the population aged 95 and older (Coale and Kisker, 1990). Recent evidence suggests, however, that gender-specific mortality estimates for the total population under age 95 are reliable (Kestenbaum, 1992). The greatest problems with estimates of old-age mortality in the United States occur when the death rates are generated by race. The data presented here are sex-specific death rates for the total population up to age 90 without reference to race.

8 Trends in mortality observed over periods longer than one year (see Table 1) dampen fluctuations and reveal nonstochastic components of mortality trends. It is important, however, to recognize that stochastic processes are operating.

9 Exogenous mortality is defined as accidents, homicide, suicide, some forms of cancer, and infectious and parasitic diseases. Endogenous mortality is defined as all other causes. Similar distributions of endogenous and exogenous mortality exist for US females and for other low-mortality populations (WHO, 1992). We are now seeking to refine the distinction between exogenous and endogenous (senescent) mortality.

10 These include the following disease categories from the tenth international classification of diseases: congenital anomalies (740–759); maternal complications of pregnancy (761); complications of placenta, cord, and membranes (762); disorders relating to short gestation and unspecified low birthweight (765); birth trauma (767); intrauterine hypoxia and birth asphyxia (768); neonatal hemorrhage (772); accidents and adverse effects (E800–E949); and homicide (E960–E969).

11 It may eventually become possible to better diagnose life-threatening congenital anomalies during gestation. Selective induced

abortion could then nearly eliminate deaths from congenital anomalies. Nevertheless, there will always be some unavoidable early-age mortality from accidents and homicide.

12 Projections are for US females from 1983 to 2080 under the assumption that total mortality at ages 65 and 70 declines by 2 percent annually from levels observed in 1983. This assumption is consistent with predictions made by Ahlburg and Vaupel (1990) and Vaupel and Gowan (1986) about the future course of mortality in the United States.

13 Because senescence has always been a rare event, individuals who survived into older ages probably shared a common genetic heritage that permitted them to senesce at a slower pace. Declining mortality at all ages would be expected to permit subgroups of the population that are more genetically diverse to survive into older ages. That is, individuals who would have otherwise died early in life are now surviving to much older ages. Examples include individuals treated for end-stage renal disease, diabetes, vascular occlusion, and cancer (among many others). A greater diversity of alleles in a population that lives

beyond the reproductive period should result in an expansion of the number of diseases and disorders associated with senescence.

14 Proponents of the 2 percent assumption have argued that large reductions in mortality at all ages are possible in the future because declines of this magnitude occurred in the past in most low-mortality populations (Kannisto et al., 1993). The presence of high mortality from vascular diseases 20 years ago permitted large declines in death rates to occur. Future mortality declines must be considered within the context of underlying mortality schedules present today. Comparable percentage declines in mortality in the future require reductions in mortality from causes (primarily cancer) that have not exhibited trends comparable to that of vascular diseases. Additionally, halving the death rate from lower baseline mortality levels results in ever smaller absolute declines and therefore smaller increases in life expectancy. The entropy of the life table has been well documented in the literature (for example see Horiuchi, 1989; Keyfitz, 1985; Olshansky, Carnes, and Cassel, 1990).

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