The Great Debate on the Outlook for Human Longevity: Exposition and Evaluation of Two Divergent Views

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No. 1. I'll bet you 100 to 1 that less than 1 person in 100 will ever reach age 100.
No. 2. But we have already passed that.
No. 1. Then I'll bet you 100 to 1 that less than 1 person in 100 will ever reach age 110.
No. 2. I wouldn't bet on it. —Anonymous, 2004

The biology of longevity has to be considered in the light of evolution, but it also has to be considered in the light of reliability engineering. And both evolution and reliability engineering have to be considered in the light of population thinking, that is, demography. —Vaupel, 2003a

Introduction

Several leading gerontologists are engaged in a spirited and even vituperous debate regarding the prospects for human longevity. The issue is what life expectancy will be just after mid-century in the industrialized countries, and, more particularly, in the United States in 2060. The debate on the future of life expectancy is closely linked to such issues as the possibilities for extending average recorded human life span, the existence of limits to human life span and life expectancy, the form of the trajectory of age-specific mortality rates at the highest ages of life and the utility of developing projections of mortality on the basis of causes of death, particularly as aggregated in the categories of intrinsic (or endogenous) and extrinsic (or exogenous) mortality.

This paper sets forth the elements of the two main positions and critically examines them. Many of the arguments on each side are cogent, and I found it hard to reconcile the two positions. I conclude, therefore, with an intermediate position that accepts the principal arguments of each side without fully embracing the conclusions of either. I illustrate some of the arguments on the basis of data for the United States.

1. Issues in the Debate

The first group espouses the more expansive view. It maintains that a life expectation (at birth) of 100 years will be reached in the industrialized countries by the year 2060. The second group supports the more conservative view. It maintains that life expectation is not likely to exceed 85 years by 2060 in the United States. Both views posit the same general cultural and technical environment as now exists, without assuming groundbreaking developments in molecular biogerontology, that is, without modifications of the basic processes by which humans age. The difference in life expectation is great and has tremendous social and economic consequences, particularly with regard to planning for health services and the management of the Social Security system.

At first view, the debate is between mathematical demographers and social biologists, but the sides do not line up so neatly inasmuch as scholars of both types appear on each side. On the expansive side are James Carey, James Vaupel, Shiro Horiuchi and John Wilmoth; on the conservative side are Leonard Hayflick, Jay Olshansky, Bruce Carnes and James Fries.

There are two other positions, each more extreme than those being compared here. The first I call "geriatric nihilism." It essentially argues that we ought not be so concerned about the longevity of very old people; too much treatment is wasted on

people who have only a few years to live. The other position not involved in the main contest is the view on the part of some molecular biologists, such as Cynthia Kenyon, Leonard Guarente and Michael Rose, that, with genetic engineering, aging can be drastically postponed in humans, as it has been postponed experimentally in nematode worms and yeast (Guarente, 2003; Guarente and Kenyon, 2000; Hall, 2003). I am not concerned with this position either, which I consider to be unrealistic at this time. Work in this area is still in its primitive stages and has been confined to very low-order animals.

2. The Demographers' View

Using data mostly for the European industrial countries, the demographers point to (1) the uninterrupted, long historical rise in life expectancy, (2) the acceleration in the rise of the numbers of older aged (85 and over) persons, (3) the increase in the numbers and percentages of centenarians and supercentenarians, (4) the steady rise in the maximum recorded life span, (5) the deceleration of the age trajectory of death rates at the very advanced ages of the life cycle, (6) the lack of a strong positive correlation between levels of mortality and rates of improvement of mortality and (7) the momentum of cohort succession. This group argues that the processes that have led to these developments will continue to prevail in the future.

2.1 Historical Trends in Longevity

Life expectancy (at birth) in the United States has steadily increased over the past century. In 1900 it was only 47; it reached 68 by 1950; and today it stands at 77.0 (Table 1). These numbers represent an annual average amount of gain of 0.3 year, or an annual average rate of gain of 0.5 percent, over a century. By exponentially extrapolating such a series on life expectancy, Oeppen and Vaupel (2002) predicted a life expectancy of 100 in 2060 in the United States. According to Vaupel (2003b), approximately half of the girls born in Germany today will live to experience the beginning of the 22nd century; that is, the median age at death of the birth cohort will be 100 or so.

TABLE 1
Comparison of Percent Declines in Age-Adjusted Death Rates With Percent Increases in Life Expectation at Birth: United States, 1900 to 2000, by Decades and by Tri-Decades

Year	Age-Adjusted Death Rate[b]	Expectation of Life at Birth (e_0)		Percent Change[a] Death Rate (e_0)
U.S. 2000 Population as Standard				
2000	869.0	76.9	-7.4	+2.0
1990	938.7	75.4	-9.7	+2.3
1980	1039.1	73.7	-15.0	+4.1
1970	1222.6	70.8	-8.5	+1.6
1960	1339.2	69.7	-7.4	+2.2
1950	1446.0	68.2	-19.0	+8.4
1940	1785.0	62.9	NA	NA
1970-2000	X	X	-28.9	+8.6
1940-1970	X	X	-31.5	+12.6
U.S. 1940 Population as Standard				
1940	10.8	62.9	-13.6	+5.4
1930	12.5	59.7	-12.0	+10.4
1920[c]	14.2	54.1	-10.1	+8.2
1910[c]	15.8	50.0	-11.2	+5.7
1900[c]	17.8	47.3	X	X
1900-1930	X	X	-29.8	+26.2
1910-1940	X	X	-31.6	+25.8

X Not applicable. NA Not available.
[a] For prior decade.
[b] Per 100,000 population for 1940-2000; per 1,000 population for 1900-1940.
[c] Death registration states only.

Source: Based on U.S. National Center for Health Statistics, *NVSR* 51(3), 2002; *NVSR* 50(15), 2002; and *Vital Statistics of the United States, 1950*, Vol.1, Part 1, 1954.

In the last several decades of the last century, the percent reductions of death rates and the percent increases in survival rates at the older ages have increased greatly in relation to those for the younger age groups (Table 2). Select any older age group and any younger age group and compute the differences between the ages in the percent reductions or increases, as in the examples at the lower panel of Table 2. As a result, survival rates at the higher ages have risen briskly and have shifted upward relative to those at the younger ages (Table 3). Table 3 also illustrates this trend with data on

changes in age-bounded life expectancies (i.e., average years lived in the age interval). Average years lived in the interval from age 65 to age 80 increased from 11.1 years to 12.5 years, that is, by 1.2 years, in the 50-year period from 1950 to 2000, whereas in the earlier half-century, average years lived in this age interval amounted to only 1.0 year. In the birth-to-65-year interval, the relative shifts were sharply in the opposite direction.

TABLE 2
Percent Change in 5-Year Mortality Rates and 5-Year Survival Rates: United States, 1900-1950 and 1950-2000

Terminal Age Group (Years)	Percent Reduction in 5-Year Mortality Rates[a]		Percent Increase in 5-Year Survival Rates	
	1900-1950[b]	1950-2000	1900-1950[b]	1950-2000
0-4	83.4	76.6	14.8	2.5
5-9	93.7	76.2	5.1	0.4
10-14	97.4	72.6	1.3	0.2
15-19	97.3	47.4	1.3	0.2
20-24	96.3	34.3	2.1	0.2
25-29	95.6	35.7	2.8	0.3
30-34	95.1	40.7	3.1	0.4
35-39	94.5	42.9	3.5	0.5
40-44	93.9	44.8	3.5	0.8
45-49	92.9	47.1	3.5	1.3
50-54	91.6	48.7	3.5	2.1
55-59	89.2	48.9	4.0	3.3
60-64	85.7	46.0	4.7	4.7
65-69	80.5	42.4	6.6	6.4
70-74	73.4	40.1	8.9	9.4
75-79	63.3	38.8	12.6	14.8
80-84	49.8	35.0	20.0	22.4
85-89	35.8	27.6	28.4	30.8
90-94	23.2	21.2	35.6	43.3
95-99	12.9	15.7	43.8	63.0
100-104	5.3	13.1	76.6	104.9
Selected Differences				
(25-29) – (75-79)	+32.3	-3.1	-9.8	-14.5
(30-34) – (80-84)	+45.3	+5.7	-16.9	-22.0

[a] Five-year mortality rates are the complements of 5-year survival rates.
[b] Life table for 1900-02 relates to white males in the Original Death Registration States.

Source: Based on life tables for 1900-02, 1949-51, and 2000, published by the U.S. National Center for Health Statistics or its predecessor agencies.

TABLE 3
Percent Surviving and Age-Bounded Life Expectancies:
United States, 1901 to 2000

Year	Percent surviving: Birth to 65	Age 65 to 80	Age 80 to 95	Average years lived in interval: Birth to 65	65 to 80	80 to 95	95+
	Percent Surviving			**Average Years Lived in Interval**			
Year	**Birth to 65**	**Age 65 to 80**	**Age 80 to 95**	**Birth to 65**	**Age 65 to 80**	**Age 80 to 95**	**Age 95+**
1901[a]	40.9	33.1	2.5	44.4	10.1	5.2	2.2
1939-41	60.4	37.9	3.7	55.9	10.6	5.6	2.6
1949-51	67.6	43.4	5.2	58.7	11.1	6.2	2.5
1959-61	71.1	47.2	4.5	59.7	11.4	6.3	2.4
1969-71	71.9	49.1	7.9	60.0	11.5	6.9	3.1
1979-81	77.1	56.0	11.7	61.1	12.0	7.6	3.3
1989-91	79.5	59.2	13.3	61.6	12.3	8.0	3.3
2000	82.1	62.1	14.2	62.2	12.5	8.1	3.5
Percent Increase							
1901 to 1949	65.3	31.1	108.0	32.2	9.9	19.2	13.6
1949-51 to 2000	21.4	43.1	173.1	6.0	12.6	30.6	40.0

[a] Original Death Registration States.
Source: Based on decennial life tables, 1901 to 1989-91, and life table for 2000 published by the U.S. National Center for Health Statistics or its predecessor agencies.

The rise in life expectancy at birth shows no signs of stopping. Currently, Japanese women are setting the national record for women, with a life expectancy at birth of 85 years, and Icelandic and Japanese men are setting the national record for men, with a life expectancy of 79 and 78 years, respectively (Population Reference Bureau, 2004).

As a result of the higher survival to age 85 and the reduction of death rates above age 85 in the United States in recent decades, there has been a sharp increase in the number of persons 85 years and over and 100 years and over (although the possible contributions to this trend of an increase in the size of birth cohorts 85 years ago and earlier and immigration need to be evaluated). (See Table 4.) There even appears to have been an *acceleration* in the rise in the numbers of older aged persons and centenarians (Tables 4 and 5). While being a centenarian is still relatively rare, the number of centenarians is estimated to have increased 22-fold in the United States between 1950 and 2000, from 2,300 to 50,000.

TABLE 4
Percentage Increase in the U.S. Population 85 Years and Over, by Age: 1980 to 2050

Age	Population (000)				Percent Increase		
	1980	1990	2000	2050 [a]	1980-90	1990-2000	2000-50 [b]
Total, 85 and over	2,240	3,049	4,240	20,861	36.1	39.1	392.0
85-89	1532	2,047	2,790	10,253	33.6	36.3	267.5
90-94	562	760	1,113	6,473	35.2	46.4	481.6
95-99	132	205	287	2,984	55.3	40.0	939.7
100 and over	14 [c]	37	50	1,150	164.3	35.1	2,200.0

[a] U.S. Census Bureau projections, middle series, based on 2000 census.
[b] Average decennial increases are 32 percent for 85 years and over, and 26, 35, 47, and 63 percent for the component age groups.
[c] Records of the Social Security Administration; the original census count, 32,000, was re-estimated.

Source: Internet, *www.census.gov*. Various census reports.

TABLE 5
Growth of U.S. Centenarian Population: 1950 to 2050

Year	Number	Percent Increase	Percent of Total Population
1950	2,300	X	--
1960	3,300	43.5	--
1970	4,800	45.5	--
1980	14,000	195.2	0.01
1990	37,000	164.3	0.01
2000	50,000	35.1	0.02
2050	1,150,000 [1]	2,200.0 [2]	0.29

-- Less than 0.005 percent.
X Not applicable.
[1] Census Bureau middle series of projections.
[2] The average decennial rate of increase is 62.7 percent.

Source: U.S. Census Bureau census reports; records of the Social Security Administration; Internet, *www.census.gov*; and Siegel and Passel (1976).

We cannot talk of trends in supercentenarians (people who have a verified age of 110 or more) in the United States because the figures are too unreliable, whether we consider census, vital statistics, Medicare data, or Social Security beneficiaries. The number of supercentenarians is rapidly increasing in Western Europe, however (Robine and Vaupel, 2002). While the first confirmed supercentenarians go back only to the 1960s, their numbers have increased exponentially since then.

Maximum recorded life span has been steadily increasing at least for 140 years in Sweden, and the rate of increase has been rising (Wilmoth and Robine, 2003). (See also Robine and Saito, 2003). Maximum recorded life span rose by 0.4 years per decade before 1969 and 1.1 years per decade thereafter. Maximum recorded life span in the "world" (Western Europe) has risen even more rapidly. It rose linearly since mid-19th century at an average "rate" of three months per year (2.5 years per decade) for females and at an average "rate" of 2.5 months per year (2.0 years per decade) for males (Oeppen and Vaupel, 2002). For every year since 1977, the oldest validated age at death was 110 or higher. For the seven countries with the most thoroughly validated data, maximum age at death rose 1.4 years per decade from 1977 to 2000 (Wilmoth and Robine, 2003). Wilmoth and Robine attribute the rise in the maximum age at death in Sweden largely to declines in mortality over age 70 and secondarily to increased numbers of survivors to old age (both larger birth cohorts and increased survivorship from birth to age 70).

2.2 Age Pattern of Mortality Rates

An examination of the age pattern of age-specific mortality reveals a slowing down of the rate of increase in death rates at the highest ages, that is, from about age 85. Between ages 30 and 85, age-specific death rates tend to rise roughly in accordance with the Gompertz curve, that is, at a fixed rate of increase. The rate of increase tends to fall above age 85, and even possibly, at the more extreme ages, to become zero or negative, although one cannot be certain of the latter because of the sparseness of the data above age 110. Several researchers have described this phenomenon: Robine and Vaupel (2002), Horiuchi and Wilmoth (1998), Wilmoth (1998), and Robine and Saito (2003). Robine and Vaupel (2002) reported, on the basis of the experience of the International Database on Longevity (IDL) list of validated supercentenarians (n=159), a probability of dying at age 110 of 0.52 and then virtual stability just below this level until age 114.

Table 6 presents a rough illustration of this generalization using mortality rates for the United States in 1979-1981 and 2000. The table shows the rate of mortality change between adjacent 5-year age groups (from 5-year age group to 5-year age group) based on official U.S. life tables for these years. The figures are roughly similar over the range of ages from 40 to 85. They resemble a modification of the growth parameter of

the Gompertz curve—for the Gompertz curve to fit these figures exactly, they would have to be constant—and are possibly described better by the Makeham model, which allows for extrinsic or exogenous mortality (i.e., mortality due to nonbiological or accidental causes). Table 7 shows rates of mortality change for single ages from age 85 to age 99 based largely on Medicare data. There is a substantial and steady decline of the rates over the whole range of these ages.

TABLE 6
Rate of Mortality Change Between Successive 5-Year Age Groups: United States, 1979-81 and 2000 Life Tables
[$k = \ln({}_5q_x) - \ln({}_5q_{x-5})$]

Ages of Initial Rate	Terminal Rate	1979-81	2000
Births/5	5/10	-2.303	-2.303
5/10	10/15	.027	.226
10/15	15/20	1.101	1.188
15/20	20/25	.333	.340
20/25	25/30	.089	.031
25/30	30/35	.061	.158
30/35	35/39	.284	.332
35/40	40/45	.417	.383
40/45	45/50	.460	.405
45/50	50/55	.449	.374
50/55	55/60	.410	.432
55/60	60/65	.413	.436
60/65	65/70	.374	.408
65/70	70/75	.372	.406
70/75	75/80	.357	.396
75/80	80/85	.385	.427
80/85	85/90	.337	.381
85/90	90/95	.265	.288
90/95	95/100	.181	.204
95/100	100/105	.090	NA
100/105	105/110	.044	NA

NA - Not available.

Note: Death rates for ages under 85 years are based on vital statistics and death rates for ages 85 years and over are based on the Medicare program.
Source: Based on U.S. National Center for Health Statistics, *NVSR* 50(15), "Deaths: Final Data for 2000," 2002; *U.S. Decennial Life Tables for 1979-81* 1(1), 1985.

TABLE 7
Age-Specific Rate of Mortality Change at Successive Ages Above Age 84
Based on Medicare Data: United States, 1997 and 1990
[$k_x = \ln(q_x) - \ln(q_{x-1})$]

Initial Terminal Ages	1997[a]	1990[b]
84-85	0.0926	0.0858
85-86	0.0902	0.0908
86-87	0.0878	0.0898
87-88	0.0855	0.0875
88-89	0.0831	0.0865
89-90	0.0807	0.0899
90-91	0.0783	0.0919
91-92	0.0759	0.0864
92-93	0.0736	0.0809
93-94	0.0712	0.0715
94-95	0.0688	0.0691
95-96	0.0664	0.0697
96-97	0.0640	0.0628
97-98	0.0616	0.0562
98-99	0.0593	0.0563
99-100	NA	0.0564

NA Not available.

[a] Ages 85 to 99 based on Medicare data.

[b] Ages 95 to 99 based on Medicare data; ages 85 to 94 based on a shifting blend of Medicare rates and rates based on vital registration.

Source: U.S. National Center for Health Statistics, reports on life tables for 2000 (*NVSR* 51(3), 2002) and 1989-91 (*U.S. Decennial Life Tables for 1989-91* 1(1), 1997).

The proponents of the demographic position conclude from the historical trends and the age trajectories described that no limit can now be set for human longevity, either as measured by life expectancy or by life span. They maintain that the pattern of decelerating death rates at the later phases of the age cycle, and especially of unchanging or even declining death rates, is inconsistent with the assumption of a limit to life expectation or life span.

There is a more narrowly focused debate as to whether the deceleration of the hazard rates at the oldest ages can be accounted for entirely by the selective elimination of the more vulnerable members of the cohort. Otherwise stated, the members who have poorer health die off, leaving the more healthy persons as survivors at the highest ages. The evidence seems to support the view that heterogeneity of the older population with respect to health does not fully explain the deceleration (Wachter, 2003). Some of the deceleration could then reflect real declines in mortality at the "individual" level or for the component "health status" subgroups. In an exceptional turn to biology by the demographers, a revision of classical evolutionary theory has been co-opted to explain the deceleration of the rates, but the arguments are inconclusive (Wachter, 2003).

2.3 Momentum of Cohort Succession

There is emerging evidence that the improvements in health in youth are maintained into the later ages (Elo and Preston, 1992; Haywood and Gorman, 2004). Given the considerable class mobility in the United States, the generally improving health of the younger population is reflected in better health at the older ages, which results in more healthy children and then more healthy older years (Marmot, 1986). In other words, upward mobility leads to better health for the families of the upwardly mobile, which they pass on by example, education and provision of better care to their children, who pass on these practices to their children.

2.4 The Role of Medical and Other Human Interventions

In the past, medical and other human interventions have contributed greatly to the extension of human life. We have benefited from immunizations, water purification, cleaner air and a greater supply of nutritious food, as well as coronary bypass surgery, organ transplantation and new medications for chronic diseases. The demographic protagonists in this debate maintain that we must not dismiss the possibilities of considerable further progress in both biomedicine and social medicine.

The rate of progress in the reduction of death rates from the major chronic diseases of later life, especially the cardiovascular diseases, achieved in the 1970s and

1980s, was spectacularly large, unanticipated and unprecedented (U.S. National Center for Health Statistics (NCHS), 2004). Even though the rate of progress diminished somewhat in the 1990s, the death rates from the major cardiovascular diseases fell by 31 percent between 1970 and 2000. Even if the advances achieved through human intervention in the last quarter-century helped only to manage chronic diseases, without dealing with their underlying causes or eliminating them, they contributed to the tremendous reduction we have seen in the death rates of cardiovascular and selected other diseases in this period. The demographers argue that we may anticipate further medical developments that will result in a continuation of these declines, as suggested below.

Gains could come from further progress in regenerative medicine genetic engineering, tissue engineering, nanotechnology and sonocytology. Regenerative medicine is a rather general concept referring to the use of human, organic materials to stimulate the body's healing power, and includes such procedures as bone marrow transplantation, joint replacement, organ transplantation and cartilage cell transplants. Genetic engineering includes recombinant DNA technology (i.e., gene splicing), in which drugs based on human proteins, such as recombinant human insulin (for diabetes) and interferon alpha (for boosting the immune system in response to hepatitis and cancer) are used to treat common diseases. The sequencing of the human genome poses new possibilities for progress through gene therapy, even though at this time the functions of most genes are unknown, as are the combinations of genes needed to perform most cellular and tissue functions. For example, gene therapy could be used to stimulate bone growth (osteogenesis) or growth of blood vessels (angiogenesis). Embryonic stem cells could be programmed to metamorphose into specialized cells and tissues. Therapeuting cloning could become the route by which some of our most intractable illnesses could be treated. Other possibilities include the development of new drugs and new medical devices, such as new medical imaging devices, sensor-driven drug delivery, remote monitoring devices, improved coronary stent systems and implantable neurostimulation systems.

There are vast uncertainties with regard to the chances of success and timing of any successes in these programs, but they all could be interpreted as human interventions short of interventions in the fundamental processes of aging, that is, programs that would slow the rate of biological aging.

2.5 Biological Considerations

The demographers have some biological arguments as well, although these arguments are not central to their position. The classical evolutionary theory of

senescence generally supports the view of an intrinsic tendency for the mortality rates at the higher ages in most species to rise steadily, possibly exponentially. However, as I have noted, several researchers have demonstrated and as I have illustrated, the rise in the curve of mortality rates of humans tends to slow down after about age 85 and may even peter out or reverse direction above age 110. Carey et al. (1992) and Curtsinger et al. (1992) have shown that the hazard rates at extreme ages in several subhuman species (medflies, nematode worms and yeast) also do not continue to rise exponentially. In fact, in numerous species the fundamental shape of the hazard curve is approximated by the Gompertz curve, that is, by unchanging rates of increase from young adulthood up to advanced age, and then the rates of increase decelerate (Finch, 1990). This interspecies similarity suggests that the same biological patterns apply to humans and subhuman species. On the other hand, there is considerable variability among species and within species in the manifestations of senescence.

3. The Social Biologists' View

To counter the claims of the demographers that there are no limits to human longevity, the biological position places considerable weight on (1) the epidemiological transition, particularly the current dominance of the endogenous causes of death, (2) the limited prospects of eliminating or even sharply reducing the endogenous causes, (3) the massive reductions in mortality rates necessary to produce even small increases in life expectation in low-mortality countries and (4) the evidence of considerable age-related somatic deterioration in humans (and most animal species) after the peak fertility years.

3.1 Demographic Considerations

During the epidemiological transition that occurred in the last century, the predominance of infectious and parasitic diseases was replaced by the predominance of the degenerative diseases of later life, such as cancer, cardiovascular diseases and diabetes. The former are typically acute diseases with an early outcome of complete recovery or death. The latter are typically chronic and progressive. Whereas the former diseases were essentially externally caused and relatively easy to treat once vaccines for them were developed and immunization against them became widespread, the latter are difficult to treat and essentially incurable, having an etiology that is quite complex and essentially biologically based. Alternatively stated, the extrinsic (exogenous) causes of death result largely from external forces such as microbes and violence, and the intrinsic (endogenous) causes result essentially from biological or genetic causes.

While the 1970s and 1980s saw sharp reductions in the death rates from several endogenous diseases, these high rates of decline did not continue during the 1990s (U.S. NCHS, 2004). The decline in the death rate from the major cardiovascular diseases in the 1990s was less than 8 percent, as compared with 15 percent in the 1970s and 13 percent in the 1980s. The death rate from cancer increased 24 percent (not age-adjusted) between 1970 and 2000. The protagonists on the biological side maintain that it is unreasonable to expect the death rates from the endogenous diseases to decline during the next several decades at the same rate as they declined during the last several decades, or to expect age-specific death rates as a group to decline in this century at the same rate as they declined during the last century (when life expectation at birth increased by nearly 30 years, or over 60 percent). In fact, we cannot confidently assume that death rates at the higher ages will continue to fall. The trends can decelerate, come to a halt and even change direction. Any extrapolation of the trend of life expectancy at birth on the basis of its past trends could easily overstate the prospects for its increase.

As stated, Oeppen and Vaupel's (2002) prediction of a life expectancy (at birth) of 100 in 2060 in the United States corresponds roughly to an exponential extrapolation of life expectancy in 2000 on the basis of the 1900-2000 annual rate of increase. Olshansky, Carnes and Cassel reported in 1990 that achieving a life expectancy of 100 in the United States would require, for example, a uniform reduction of about 90 percent in current age-specific death rates. Golini reported in 2002 that a reduction of 85 percent would be required to achieve a life expectancy of 100. The answer depends on the mortality base for the projection and the age pattern assumed for the reduction. I estimate that obtaining a life expectancy of 100 from the level of the U.S. age-specific death rates of 2000 under the assumption of a uniform reduction of these rates would require a decline of 82 percent in age-specific mortality. The following mini-table relating the (uniform) percent reduction in death rates and percent increase in life expectancy at birth, beginning with the U.S. life table for 2000 (e_0 = 76.9 years), suggests a curvilinear relationship between these measures:

Percent Reduction in Age-Specific Death Rates	**Percent Increase in Life Expectancy**	**Resulting Life Expectancy**
0	0	77 (base value)
45	10.5	85
50	12	86
80	25	96
82	30	100
90	71	132
100	∞	∞

Olshansky et al. (1990) consider that attaining a figure of 100 in 2060 is implausible, if not impossible, because it would require the elimination of all deaths from endogenous causes. This presumably assumes that exogenous mortality continues roughly at the present level. Much exogenous mortality would certainly remain with us. Deaths from violence (i.e., accidents, suicide and homicide), which make up 6 percent of deaths today, may be expected to continue, as would some types of infectious and respiratory diseases, and complications of pregnancy, childbirth and the puerperium. Accordingly, these analysts concluded that life expectation was unlikely to rise above 85 years (88 for females and 82 for males) unless the aging process itself could be modified, which is not expected for the foreseeable future. This is consistent with a uniform reduction of age-specific death rates in 2000 of about 45 percent. (See also Fries, 1980; Carnes, Olshansky and Grahn, 1996; Olshansky, Carnes and Désesqelles, 2001; and Olshansky, Carnes and Grahn, 1998.)

According to the current life table for the United States in 2000, about 1.8 percent of an initial birth cohort survive to age 100 (U.S. NCHS, 2000b). The mean age at death is 77 and the median age at death is 80. The Oeppen-Vaupel projection of mortality, having posited that the mean (life table) age at death in 2060 will be 100, implies that about 56 percent of the initial cohort will survive to age 100, about 50 percent of the initial cohort will survive to about age 103 (the corresponding median age at death), and the complete expectation of life at age 100 (e_{100} + 100) will be about 106 years. The biologists maintain that this kind of change in the next half-century is simply not credible.

It is evident that at low levels of mortality, a considerable decline in age-specific death rates is required to produce even a small increase in life expectancy. Table 1 compares, for the period 1900 to 2000, the percent reductions in age-adjusted death rates (standard population = 2000 for 1940-2000, and standard population =1940 for 1900-1940) with the percent increases in life expectancy at birth for each decade. As expected, for every decade in this period, the percent increase in life expectation at birth is smaller than the associated percent decrease in death rates. However, we see only a rough indication of an inverse correlation between decennial changes in age-adjusted death rates and life expectation over this 100-year period. When the figures are grouped in 30-year intervals, the indications of this pattern are stronger. At e_o = 71 years in 1970, a decline of the age-adjusted death rate of 29 percent between 1970 and 2000 corresponds to a rise of only 9 percent in e_o (Table 1). In the previous 30-year period, with an initial expectation figure of 63 years, the changes were 32 percent and 13 percent. A similar relation is shown (except at the oldest ages) between percent declines in 5-year mortality rates from life tables and the corresponding percent increases in 5-year survival rates (Table 2). (See also Vaupel, 1986.)

3.2 Biological Aspects

As I interpret the views of the social biologists, "normal aging" in humans is a road map to disaster in later life. With normal aging there is senescence, i.e., molecular and cellular pathogenesis that degrades functional integrity and homeostasis of the body. Senescence limits life expectancy and life span. As a result of the biological processes and evolutionary influences noted, there is an average 50-to-80-percent loss in the functioning of the various physiological systems by one's 80th year. These relate to heart function, immune system function, endocrine system function, lung capacity, kidney function and bladder capacity (U.S. National Institute on Aging (NIA), 2002). There are sharp declines in muscle strength, bone density, aerobic capacity, glucose tolerance and the heart's maximum pumping rate. The declines in psychological functioning are also enormous by the 10th decade (Baltes, 2002).

No chronic degenerative disease has been eliminated, or even nearly eliminated, in spite of all the efforts in this direction. There is no evidence that the age of onset of any major endogenous disease has been raised, although, admittedly, establishing the age of onset of a disease is extremely difficult. We have been able only to manage some of these conditions so that their disabling effects are postponed and the quality of life for persons having the conditions is improved. Some additions to life expectancy may clearly result from medical interventions, but at this stage there are narrow limits as to how far these interventions can extend life expectancy. The death rates from various forms of violence were reduced by only one-third between 1970 and 2000, so these causes may still be expected to contribute substantially to future mortality. The death rate for cancer, as well as for several other leading endogenous diseases (e.g., nephritis, chronic obstructive pulmonary diseases, Alzheimer's disease, septicemia), rose in this period.

3.3 Epidemiological Considerations

The resurgence of old infectious diseases and the emergence of new ones may make progress more difficult. The last few decades have seen the reappearance in the United States of several infectious diseases that had largely been obliterated—measles, tuberculosis, whooping cough, diphtheria—and the appearance of new infectious diseases, primarily HIV/AIDS, but also SARS, monkeypox, and West Nile virus. Moreover, the death rate from influenza has increased significantly in recent decades. This trend is due to the decreasing effectiveness of many established antibiotics, the mutation of viruses and the possible transfer of viruses from animals to humans.

A more ominous threat is the recent sharp increase in overweight and obesity (37 percent between 1977 and 1999), with their numerous negative health implications, such as increased risk for diabetes, hypertension, stroke, osteoarthritis and various types of cancer (U.S. NCHS, 2004, Internet). Olshansky and Carnes have estimated that the current impact of obesity on life expectancy in the United States is approximately 3.5 years—a loss to life expectancy about the same as that caused by all types of cancers, and they expect the impact to at least double in the next few decades (Olshansky, 2004).

3.4 Evolutionary Perspectives

A basic argument of the group espousing a limit for human life expectancy and life span rests on the role of evolutionary biology in aging and senescence. Evolutionary theory informs us that animals have responded to the high levels of extrinsic mortality (i.e., accident, disease, starvation and predation) throughout their existence on earth by experiencing early reproduction of the species (35 years of age or younger for humans), and then experiencing failure of their bodily structures, which are not designed for extended operation. This evolutionary experience dictates an age beyond which the probability of continued survival is extremely low, defining a "biological warranty period," as Carnes, Olshansky, and Grahn (2003) have called the extended period allowed by evolution for human survival.

According to evolutionary theory, as Olshansky has stated, "natural selection operates primarily on traits that affect an organism's ability to reproduce; accordingly, one would not expect evolution to favor genes that extend an organism's life much beyond its reproductive years" (quoted in *Smithsonian*, March 2004, by Stephen S. Hall.). For Carnes, Olshansky and Grahn (2003), the duration of human life needed to achieve maturation and reproduction—ages 13 to about 40—helps define their biological warranty period.

Today most people survive beyond the ages of reproduction, but recall that only a century ago, life expectancy hardly extended through the reproductive years. Three factors account for the extension of life expectancy beyond the prime reproductive years. First, it would appear that evolution has endowed the body with a reserve capacity so as to assure the completion of its reproductive mission. It would be inefficient to risk failure in this mission, given the ever-present extrinsic factors vying for the life of an animal, by not building in such a reserve capacity. Next, evolution has discovered a "reproductive" role for older people. Most parents become grandparents and are thus available to nurture and train the young, who may go on to achieve

successful reproduction (Vaupel, 2003a; Austad, 1997). Finally, trends in life expectancy suggest that human intervention may have contributed to the success of many people in approximating, and of some people in exceeding, their biological warranty period. Survival into post-reproductive years has been achieved by human ingenuity and self-discipline, including public health innovations, medical developments, environmental improvements and especially lifestyle changes (Carey, 2003). I should also note that, according to reliability engineering theory, the fact that humans have many redundant parts could also be used to explain the extended life expectancy of humans after menopause (Gavrilov, 2003).

The biologists claim that there are no longevity genes in humans, and they question the relevance for humans of the research on aging of nematode worms, now being vigorously pursued by some evolutionary biologists. "Gerontogenes," or longevity assurance genes, have been found only in a few invertebrate species, such as nematode worms. Carnes and Olshansky (1993) consider age 80 as the limit of the biological warranty period for humans and 85 as the limit of life expectancy, barring pioneering scientific developments in understanding the fundamental processes of aging and ways of modifying them (e.g., modifying the biological rate of human aging). Survival much beyond these years, however, will be limited until such knowledge is secured (Hayflick, 1998, 2000). Hayflick also doubts that life span can be significantly prolonged by genetic engineering. He argues that extending human life span can only be achieved by probing into the causes of the aging of cells, i.e., by determining why older cells are more likely to fail than younger cells (Hayflick, 1998).

4. Comments and Critique

The arguments presented on each side are cogent even though they lead to different conclusions. Unlike the protagonists in this debate, I am inclined to be restrained in my comments on the two positions because I have no emotional commitment to either. I am concerned that the two sides have become so doctrinaire in their positions that they cannot recognize the merits of the opposing arguments. A review of some of the issues by a panel of the National Academy of Sciences revealed how ambivalent the situation remains with respect to forecasting mortality(Stoto and Durch, 1993).

The first position may be characterized as one of demographic determinism. It is strong on analysis of demographic trends but lacks a theory of demographic change. It lacks a theory for projecting mortality but finds the past statistical record of mortality change alone an adequate basis for determining future levels of mortality. Several pieces of historical evidence are offered for the position that there are no foreseeable

limits to life expectancy and life span, but we are not offered a convincing explanation of why the changes that have occurred should continue. If compositional/heterogeneity changes and classical evolutionary theory do not fully explain the deceleration of hazard rates at the very advanced ages, another theory is needed (Wachter, 2003).

The other position may be described as one of biological determinism. It is strong on biological theory, but its historical base for analyzing the trends in longevity is limited. It sets only rough guidelines for the future of life expectancy and life span and fixes no dates for the attainment of the "maximum" values hypothesized. For the most part, this position lacks a statistical model relating the biological processes at work to the demographic results proposed. Specifically, it lacks a formal statistical basis for developing projections of age-specific mortality.

Before presenting a more detailed evaluation of the two sides of the argument, I want to digress to consider three related topics that shed light on significant facts in the debate: the possible use of cause-eliminated life tables in judging the limits to life expectancy, the relation of mortality compression theory to the possibility of limits to life expectancy and the use of death rates disaggregated by cause in making projections of mortality.

4.1 Cause-Elimination Life Tables and Limits to Life Expectancy

Cause-elimination life tables provide another way of looking at the possibility of extending life expectancy. Cause-elimination life tables calculate life expectancy on the assumption that a particular cause of death or group of causes has been eliminated. Such tables have been computed by the National Center for Health Statistics every 10 years from 1959-61 to 1989-91. Tables are calculated for a few dozen causes, including the leading causes and an "all other causes" category; hence, all causes are nominally covered. They make the unrealistic assumption that the causes of death occur independently of one another; that is, they disregard the competing risks of the causes of death.

If we track the total life expectancy that would have been achieved between 1960 and 1990, assuming the complete elimination of each of the causes of death (and assuming independence of the risks of dying from the various causes), we would derive a series that approximates 90 or 91 years:

Years	Life Expectancy Without "Gains"	"Gains" in Life Expectancy	Life Expectancy Including "Gains"
1989-91	75.4	14.3	89.7
1979-81	73.9	18.6	92.5
1969-71	70.8	20.1	90.9
1959-61	69.9	18.9	88.8

Source: Based on U.S. National Center for Health Statistics decennial life tables for the indicated years.

For example, in 1989-1991, 14.3 years would be added to total life expectancy by the elimination of the various listed causes of death.[1] The sum of this number and the current expectation of life, 75.4 years, is 89.7. For 1959-61 the corresponding sum is 88.8. Inasmuch as this series of figures displays a fluctuating trend rather than a monotonic one and is not fully comparable, it is difficult to extrapolate it with confidence. Theoretically, we would expect the total years to be nearly constant if the data were accurate and the figures consistent and comparable; as life expectancy rises and age-cause-specific death rates fall, the increase in life expectation at birth during any period and the reduction in the total gains from eliminating the various diseases during the same period should approximately balance.

I have previously asserted that cause-of-death life tables tell us little or nothing about the future (Siegel, 1993); they give us only indications of the relative importance of various causes of death currently and inform public health policy in this way. Now I am deviating from this view to suggest that we can theoretically consider these figures as a current *maximum* estimate of human life expectancy. To understand why this may be so, we must recognize that independence of mortality risk is not the way mortality risks work in the real world. In actuality, all the causes of death compete actively with one another to achieve a person's demise.

Because of (1) the competing risks of death, (2) the multiplicity of causes that contribute to or are associated with each death and (3) the impossibility of human immortality, in the event of the elimination of any cause of death, or even its sharp reduction, the death rates from other causes—particularly those usually associated with the cause eliminated or with average ages close to that of the cause eliminated—will tend to rise. Obviously, death rates for all the listed causes cannot be reduced to zero. Stallard's (2002) study of underlying and multiple-cause mortality showed conclusively that "death is due not to just one single disease but to a complex set of interacting pathological processes. In these cases the designation of any single disease as the underlying cause of death provides a distorted description of the causal pathways." It is even conceivable that the sharp decline recorded for death rates from cardiovascular causes between 1970 and 2000 "contributed" to some extent to the lack of substantial progress in reducing cancer during this period, even though cancer is not usually reported as a contributing cause of death from cardiovascular diseases. Hayflick (1998) supports the main point here from the perspective of the molecular biologist.

On the other hand, it could be argued that if certain causes of death were eliminated, the death rates from some other causes would be lower than recorded because the specified eliminated cause could not have contributed to earlier deaths from these other causes. Hence, death rates from these other causes would also be lower. I believe, however, that the earlier of these two arguments is more cogent.

If two causes are eliminated in successive stages, first one cause and then the other, the gain made by the elimination of the second cause, allowing for the principle of competing risks, would tend to be greater than if it were eliminated independently. This is so because the number of survivors at each age in the life table is greater after the first cause is eliminated than when each of the two causes is eliminated independently and because of the competing risk principle. In other words, success in reducing some causes will exact a price on other causes, possibly resulting in a rise in the rates of these other causes. I am hypothesizing, therefore, that the total years added by the independent elimination of the various causes is a *maximum* in relation to probable life expectancy, since the real addition to life expectancy would be less because of the force of the competing risks of death.

4.2 The Compression of Mortality

Compression-of-mortality/rectangularization theory has been considered for its possible relevance in providing an indication of whether there are limits to life expectancy and life span (Wilmoth, 1997). Mortality compression theory may be relevant to this issue because a decreasing variation in the age distribution of deaths, or an increasing rectangularization of the survival curve, is more supportive of the view that there is a limit to life expectancy and life span. Many measures of the compression of mortality have been proposed (Wilmoth and Horiuchi, 1999), and I employ a few of them in the discussion here. It can be maintained, reversing the order of the arguments, that if average recorded life span is a fixed quantity such as 100, mortality will tend to become compressed because of the "ceiling effect," and if we assume that average recorded life span has been rising, then mortality compression may not be occurring. In the former case, the measure of compression can be life expectancy itself in the form of a percentage (for example, 1960, 69.9 percent; 1990, 75.4 percent; and 2000, 77.7 percent), where in a graphic presentation the percentage represents the share of the total rectangular area bordered by the x-axis running from 0 to 100 (age) and the y-axis running from 0 to100 (life expectancy). (See Siegel, 1993.)

In assessing the trend of variation in the distribution of deaths by age for 1940 to 2000, I use the relative interquartile range as derived from the appropriate U.S. life tables and recorded death statistics. The last column of Table 8 gives this measure based on life tables for 1939-41 to 2000. It is computed by dividing the interquartile range (the difference between the first and third quartile) by the second quartile, i.e., median age at death (expressed as a percent). The series shows a nearly steady decline in the percent variation around the median from 34 percent to 23 percent, and suggests, therefore, a pronounced trend of mortality compression. As stated, these results are more consistent with the fact that the rise in life expectancy is slowing down and may be approaching a fixed upper limit than that it will rise indefinitely. These results are mitigated by the data on average age at death in Table 8, however. In addition to the measure of variation, Table 8 displays several measures of central tendency. Not one of the three measures of central tendency shows a substantial falling off in its increase over the 60 years, as would be expected if the rise in longevity were slowing down. Similar measures based on recorded death statistics show a steady rise in the median age of deaths between 1950 and 2000, but the decline in the relative interquartile range from 39.0 percent to 27.0 percent suggests a marked trend toward mortality compression, as did the life table data (Table 9).

TABLE 8
Measures of Central Tendency and Dispersion of the Age Distribution of Life Table Deaths: United States, 1900 to 2000

Year	Mode[a]	Mean[b]	Median	1st Quartile	3rd Quartile	IQR[c]	RIQR[c]
2000	85.5	76.9	80.3	69.7	88.0	18.3	22.8
1989-91	83.5	75.4	79.0	67.9	87.2	19.3	24.4
1979-81	83.5	73.9	77.6	66.3	86.0	19.7	25.4
1969-71	80.5	70.8	74.9	63.1	83.5	20.4	27.2
1959-61	81.5	69.9	74.3	62.7	82.8	20.1	27.1
1949-51	78.5	68.1	72.8	60.6	81.5	20.9	28.7
1939-41	76.5	63.6	69.9	55.5	79.2	23.7	33.9
1900	72.5	47.7	56.4	20.2	73.1	52.9	93.8
Increase							
1970-2000	5.0	6.1	5.4	6.6	4.5	-2.1	-4.4
1940-1970	4.0	7.2	5.0	7.6	4.3	-3.3	-6.7
1900-1940	4.0	15.9	13.5	35.3	6.1	-29.2	-59.9

[a] Approximation.
[b] Mean age of death, or life expectation at birth.
[c] Interquartile range (IQR) and interquartile range as percent of median age at death (RIQR).

Source: Based on life tables published by U.S. National Center for Health Statistics or its predecessor agencies and by U.S. Office of the Chief Actuary (2002).

TABLE 9
Measures of Centricity and Dispersion of the Age Distribution of Deaths Based on Vital Statistics: United States, 1950, 1970, and 2000

Measure	1950	1970	2000	Increase	
				1950-1970	1970-2000
Mode[a]	72.5	77.5	82.5	5.0	5.0
Median	66.2	70.3	77.3	4.1	7.0
1st quartile	51.0	56.9	64.9	5.9	8.0
3rd quartile	76.8	80.0	85.8	3.2	5.8
IQR[b]	25.8	23.1	20.9	-1.3	-2.2
RIQR[b]	39.0	32.9	27.0	-6.1	-5.9

[a] Approximation.
[b] Interquartile range (IQR) and interquartile range as percent of median age at death (RIQR).

Source: Calculated from U.S. National Center for Health Statistics vital statistics reports for 1950, 1970, and 2000.

A new measure of rectangularization, the ratio of life expectancy at birth to total life expectancy at age 100, that is, $e_0 / 100 + e_{100}$, takes account of the available information on superlongevity and removes the confining effect of the assumption of a fixed life span. The measure shows continued compression of mortality even though life expectation at age 100 is rising. Using life tables of the Social Security Administration, the series increases steadily through two centuries:

1900	48.2
1950	69.8
2000	77.6
2100	83.5

A total life expectancy at age 100 of about 107 would be required to have this measure begin to turn around. In sum, compression-of-mortality theory is more consistent with limits on life expectancy and life span, but is not a very probative argument for the reason that the theory essentially depends on a prior assumption regarding how life span shifts.

4.3 Disaggregating Deaths by Cause in Mortality Projections

A sub-plot in the debate regarding the future "terminal" levels of mortality relates to the data and methodology for making projections of mortality in connection with population projections. The issues are closely connected with those earlier discussed. One side, the social biologists (especially Carnes, 2004a; Carnes, 2004b; Carnes and Olshansky, 1997), argues for disaggregating deaths by cause, not into a full array of causes but into the two broad categories of endogenous (intrinsic) and exogenous (extrinsic) causes, in making projections of mortality. The demographers (especially J. Wilmoth, J. Vaupel, R. Lee and S.Tuljapurkar) see no necessary merit in this approach or in any use of cause-of-death data. In their projections of population, they have mainly used time-series analysis, either on the total population of each age/sex group or on the fertility and mortality components of population change without any further disaggregation of the data, except for age and sex (Lee, 1992). In spite of the major change in the cause-pattern of death rates in the last century, the rate of mortality decline has remained quite steady, as shown by the Lee-Carter (1992) index. The demographers note that the Actuary's Office of the Social Security Administration (SSA) has long used cause-of-death classes in projecting mortality and that its experience with this procedure has not been particularly favorable. SSA has more often overestimated future mortality levels than underestimated them. Moreover, the SSA method does not provide probabilistic confidence intervals for its projections, only low and high alternatives. For these reasons, these demographers argue for

abandoning the cause-of-death approach. To be sure, if there is a problem with the SSA projections, it is not the use of cause-of-death detail but the assumptions and applications made with the data.

The biologists want to incorporate the endogenous/exogenous dichotomy into the projection design. They consider endogenous mortality to be essentially different in character from exogenous mortality because of the former's mainly biological origins, argue that endogenous mortality has a persistent and characteristic pattern and feel that more accurate judgments regarding the future levels of mortality can be made for the two separate series than for the combined series. No actual projections of mortality have been made in this way, so a report on the method's performance cannot be made. Implementation of the endogenous/exogenous dichotomy in making mortality projections would raise some fundamental problems of classification and design, as suggested below.

A comparison of endogenous death rates for 1950 and 2000 in the United States showed that both the level and age pattern of endogenous death rates changed substantially between these years. The rates for endogenous causes had sharp declines at every age (10-year age groups except ages under 1, 1-4, and 85+) between 1950 and 2000, ranging from 28 percent (85+) to 92 percent (1-4). The rates for exogenous diseases showed declines at most ages, although less marked ones, and increases at ages 65-74, 75-84, and 85+. The summary figures (rates per 100,000) are as follows:[2]

			Change	
All Causes	**1950**	**2000**	**Amount**	**Percent**
All causes*	963.8	873.1	-90.7	-9.4
Endogenous mortality	815.8	712.4	-103.4	-12.7
Exogenous mortality	148.0	160.7	+12.7	+8.6
Sums of rates**				
Endogenous mortality	36,018.2	21,949.2	-14,069.0	-39.1
Exogenous mortality	4,507.3	4,100.5	-406.8	-9.0

* Crude rate for all deaths, endogenous deaths and exogenous deaths.

** The sum of the age-specific death rates for endogenous and exogenous causes from age under 1 to ages 85 and over.

Source: Based on U.S. National Center for Health Statistics, *Vital Statistics of the United States, 1950*, Vol.1, Part 1, 1954; and *National Vital Statistics Reports* 50 (15), "Deaths: Final Data for 2000," 2002.

The patterns of endogenous and exogenous deaths rates also changed in this period. This is shown by weighted indexes of dissimilarity for each of the years and the two categories of causes of death:[3]

Comparison of Patterns of Death Rates by Weighted Index of Dissimilarity

	1950 and 2000	**Endogenous and Exogenous**	
All causes	27.4		
Endogenous	28.9	1950	32.0
Exogenous	32.9	2000	18.3

The change in the pattern of endogenous mortality between 1950 and 2000 was substantial (29 percent). The change in the pattern of exogenous mortality was even greater (33 percent). Exogenous mortality would be expected to have more irregular fluctuations than endogenous mortality, both in level and pattern, but here the situation is mixed. Because the pattern of endogenous mortality in the two years was so different, human intervention (whether in the form of changes in lifestyle, medical developments or improvements in the environment) must have been involved. Hence, for humans it is hardly possible to establish an underlying, unchanging, mortality age "signature," as it has been for mice and other animals that have been subject to human care (Carnes, 2004a). In sum, human endogenous mortality and human exogenous mortality do not maintain a fixed level or pattern (except very broadly), so that while the trends of each of these classes of mortality may reasonably be extrapolated separately for purposes of population projections, the process is subject to the same uncertainties as other types of projections.

4.4 Other issues re the Endogenous/Exogenous Classification

As mentioned, the biologists argue for the use of cause-of-death data in projecting mortality, but not in the form of specific groups of causes of deaths like infectious diseases, heart disease, cancer, diabetes, nephrotic disease, etc. They maintain that the specific causes listed on death certificates are not the real causes of death, but only rough indications of the cause system. Death may better be viewed as the end result of the compromise of the integrity of the cells and organs of the body and the associated decline of physiological systems, particularly the endocrine and immune systems. This decline results from such processes and agents as oxygen free-radical molecules, protein crosslinking, reduced production of heat-shock proteins and a decrease in the production of natural hormones (U.S. NIA, 2002). Accordingly, these analysts argue that the natural way to group deaths for projection of mortality is along

biological lines, that is, into the broad categories of deaths denoted as endogenous/ intrinsic mortality and exogenous/extrinsic mortality. They maintain that the categorization of deaths as endogenous and exogenous is fundamental to understanding the past trend of mortality, the age pattern of mortality and the future prospects for mortality. The endogenous group of causes is deemed to have a more regular trend and age pattern than total mortality, and hence is deemed to provide the best basis for extending the age curve of mortality to the highest ages and projecting the trend of mortality over time. Once projections of endogenous mortality have been completed, the less-regular component, exogenous mortality, can be superimposed on them (Carnes, 2004a).

One logical flaw in this apparently straightforward notion is that the line of demarcation between endogenous/intrinsic and exogenous/extrinsic mortality is unclear, and experts would disagree as to the classification of many diseases. Another is that as changes in technology, community organization and medical knowledge occur, the levels and age patterns of both endogenous and exogenous death rates change. While there may be no disagreement that accidents, suicides and homicides, and even infective and parasitic diseases, are extrinsic, there are some specific types of chronic diseases of later life that may be characterized as "man-made," or at least as being caused in large part by external agents, such as lung cancer (from heavy smoking), rheumatic heart disease (from an infection, i.e., rheumatic fever), cirrhosis (from heavy drinking), cardiomyopathy (possibly from a virus), chronic obstructive lung disease (induced by early-life respiratory infections) and gastric carcinoma (possibly due to infection). Value judgments suggesting contributory negligence enter into the classification scheme. Few diseases are purely genetic (germ-line). Some so-called "genetic" diseases result from somatic mutations caused by environmental factors, e.g., radiation-induced cell mutations causing cancer. Oxygen free radicals, indicted as a cause of oxidative damage to cells and tissues, can be produced by tobacco smoke, sun exposure and other environmental factors. The International Classification of Diseases (ICD) codes do not permit a precise assignment to the two broad-cause categories proposed without a major overhaul.

The National Institute on Aging has concluded that the environmental/lifestyle component of so-called "genetic" diseases accounts for a considerable percentage of the cause-structure of these diseases (U.S. NIA, 2002), but it does not provide precise quantitative measures for specific diseases. Some conditions are affected by large genetic contributions; others are affected by small ones. In each case, the contribution of lifestyle/environment may do much to modify the effect of the genetic component but cannot eliminate it. Even the rough quantification of the genetic/nongenetic etiology of endogenous diseases, as NIA has tended to make, is difficult to defend. That effort

seems to accept the earlier theories of behavior genetics, according to which heritability indexes can be calculated for non-Mendelian traits. The genetic/environmental/lifestyle interaction in the causation of diseases is believed to be far more complex than a simple exogenous/endogenous classification of deaths seems to imply. Vetta and Courgeau (2003) maintain that the genetic factor and the nongenetic factor cannot be separated for any human trait. It is virtually impossible to assign many diseases to either the endogenous or exogenous categories except arbitrarily. Even with deaths due to violence and adverse effects (accidents, homicides and suicides), we recognize the greater vulnerability of some age groups—the very young and the very old—over other age groups.

In general, the social biologists accept the fact that the distinction between two cause-categories is a simplification of reality (Carnes, 2004a). All models are simplifications, that is, ideal typical constructs, but the classification may still be of value for analytic and heuristic purposes. In trying to compare mortality levels and patterns between populations, we need to decide what are the most informative indicators of mortality we should compare, and the biological/nonbiological dichotomy is one useful way to draw information from the record. Admittedly, endogenous mortality is not a collection of purely genetically determined causes and includes diseases caused by genetic/lifestyle/environmental interactions. It is, however, "a collection of causes that are all related in some direct way to the biology of the organism" (Carnes, 2004a; Carnes, 2004b; Carnes and Olshansky, 1997).

4.5 Review of Projections of Mortality by Cause

To consider projections of mortality developed by disaggregating deaths by cause, we refer to the sets of U.S. population projections made by the Chief Actuary's Office, U.S. Social Security Administration. For a half-century, SSA has prepared its mortality projections by employing assumptions involving detail for cause-of-death classes as well as age, sex and marital status. Mortality is projected to a distant terminal date, by a process of expert judgment ("Delphic" method), in terms of the assumed reductions in the age-sex-specific rates for 10 groups of causes of death. Until recently, the various sets of mortality projections have been routinely criticized for being too conservative. In spite of the considerable disaggregation of the input data for the SSA mortality projections and the many years that SSA employed this method, the SSA

intermediate projections of life expectation were low for most projection years (Table 10). The projections of life expectancy prepared by the SSA in 2002 were adjusted upward in accordance with the recommendations of its 1999 Technical Panel on Assumptions and Methods (1999), but SSA continued the cause-of-death approach in spite of the recommendation of the panel to employ the Lee-Tuljapurkar stochastic forecasting method (Table 11).

TABLE 10
Comparison of Actual Values for U.S. Life Expectation and Values Projected by the U.S. Actuary's Office, for 1990,2000,and 2050
(Figures Shown are Intermediate Series or Averages of Low and High Series)

LIFE EXPECTATION

Pub.year no.	1990	2000	Actuarial 2050	Expectation at birth 1990	2000	2050	Expectation at age 65	(Base year)	report
Male									
1957(1954)		46		NA	71.4	71.4	NA	15.0	15.0
1966(1965)		62	69.4	70.3	70.3	13.9	14.3	14.3	
1974(1972)	72		68.6	69.0	NA	13.4	13.6	NA	
1978(1977)		77	69.6	70.3	71.6	14.2	14.6	15.4	
1983(1981)		88		72.3	73.4	75.8	15.1	15.7	17.5
1987(1986)		99	72.3	73.9	76.7	14.9	15.6	17.4	
1991(1989)	106	71.9	72.9	76.7	15.3	15.9	17.9		
1996(1994)	110	71.8	73.0	77.2	15.0	15.6	17.7		
2002(actual)		71.8		74.1	79.0[a]	15.1	16.3	18.8[a]	
Female									
1957(1954)	46		NA	77.1	77.1	NA	17.5	17.5	
1966(1965)		62		75.6	76.4	76.4	16.8	17.2	17.2
1974(1972)		72		76.3	76.9	NA	17.8	18.1	NA
1978(1977)		77		77.3	78.0	80.4	18.4	18.9	20.5
1983(1981)		88	79.8	81.0	83.8	19.9	20.8	23.1	
1987(1986)		99	79.3	80.8	83.7	19.2	20.1	22.4	
1991(1989)	106	78.8	79.9	83.1	19.0	19.6	21.7		
1996(1994)	110	78.8	79.7	83.0	19.0	19.4	21.4		
2002(actual)		78.8	79.5	83.5[a]	19.0	19.2	21.8[a]		

DEVIATIONS FROM ACTUAL

Male									
1957				NA	-2.7	-7.6	NA	-1.3	-3.8
1966				-2.4	-3.8	-8.7	-2.2	-2.0	-4.5
1974				-3.2	-5.1	NA	-1.2	-2.7	NA
1978				-2.2	-3.8	-7.4	-0.6	-1.7	-3.4
1983				+0.5	-0.7	-3.2	+0.5	-0.6	-1.3
1987				+0.5	-0.2	-1.3	+0.2	-0.7	-1.4

1991	+0.1	-1.2	-1.3	-	-0.4	-0.9
1996	-	-0.9	-1.8	-	-0.7	-1.1

Female

1957	NA	-2.4	-6.4	NA	-1.7	-4.3
1966	-3.2	-3.1	-7.1	-2.2	-2.0	-4.6
1974	-2.5	-2.6	NA	-1.2	-1.1	NA
1978	-1.5	-1.5	-3.1	-0.6	-1.3	-1.3
1983	+1.0	+1.5	+0.3	+0.9	+1.6	+1.3
1987	+0.5	+1.3	+0.2	+0.2	+0.9	+0.6
1991	-	+0.4	-0.4	-	+0.4	-0.1
1996	-	+0.2	-0.5	-	+0.2	+0.4

NA - Not available. - Rounds to zero.

[a] Intermediate projection in *2002 Annual Report of the Board of Trustees of the Federal OASI and SI and DI Trust Funds*, published in 2002.

Source: Selected *Actuarial Studies* of the U.S. Office of the Actuary, Social Security Administration. U.S. National Center for Health Statistics, *National Vital Statistics Reports*, Vol. 51, No.3, 2002.

TABLE 11
Alternative Projections of Life Expectation at Birth for the United States and Canada From Official and Private Sources: Base Year, 2030, 2050, and Terminal Year

Source	Base Year Male/Female	2030 Male/Female	2050 Male/Female	Terminal Year as Noted Male/Female
U.S. Census				
Low		76.5/82.6[a]	79.5/84.9	85.0/89.3 (2100)
Medium	74.1/79.8	77.6/83.6[a]	81.2/86.7	88.0/92.3 (2100)
High	(1999)	79.1/84.6[a]	83.8/88.4	92.3/95.2 (2100)
U.S. Actuary's Office				
Low cost series		75.4/80.4	76.4/81.4	77.7/82.3(2080)
Intermediate series	73.8/79.4	77.1/81.9	79.0/83.5	81.4/85.6(2080)
High cost series	(2002)	79.2/83.8	82.4/86.6	86.4/90.1(2080)
Ahlberg/Vaupel[b]				
Low				71.4/78.2 (2080)
Medium	71.4/78.2			84.0/89.0 (2080)
High	(1987)			96.0/100.0 (2080)
Olshansky/Furner[c]				
Lowest		75.3/82.5		
Middle		76.0/83.3		
Highest		77.0/84.9		
Lee/Tuljapurkar[d]				
Low				81/86 (2065)

Source	Base Year Male/Female	2030 Male/Female	2050 Male/Female	Terminal Year as Noted Male/Female
Medium				83/88 (2065)
High				86/90 (2065)
Tuljapurkar/Li/Boe				
Low		77.6 (2020)		80.7
Middle	75.5	79.3 (2020)		82.9
High	(1994)	80.9 (2020)		85.0
Statistics Canada				
Low		78.5/83.0 (2026)		
Medium	75.5/81.2 (1996)	80.0/84.0 (2026)		
High		81.5/85.0 (2026)		

[a] For the year 2025.
[b] Low: Mortality at 1987 level. Medium: 1% annual decline in mortality. High:2% annual decline in mortality.
[c] Non-black population.
[d] Stochastic time series forecasts. The low and high figures define the lower limit and upper limit of the 95% confidence interval.

Source:

U.S. House of Representatives, 107th Congress, 2nd Session: Communication from the Board of Trustees, Federal Old-Age and Survivors Insurance and Disability Insurance Trust Funds transmitting *The 2002 Annual Report of the Board of Trustees of the Federal Old-Age and Survivors Insurance and Disability Insurance Trust Funds*, U.S. Government Printing Office, Washington, 2002.

U.S. Bureau of the Census: Internet, 2004: www.census.gov. See also *Population Division Working Paper*, *No. 38*, by F.W. Hollmann, T.J. Mulder and J.E. Kallan, 1999.
Ahlburg/Vaupel: D.A. Ahlburg and J.W. Vaupel, "Alternative Projections of the U.S. Population," *Demography*, 27(4), November 1990.

Olshansky/Furner: Interagency Forum on Age-Related Statistics, *Occasional Paper from the National Center for Health Statistics*, "Projections of Health Status and Use of Health Care of Older Americans," January 1996.

Lee/Tuljapurkar: R.D. Lee and S. Tuljapurkar, "Population Forecasting for Fiscal Planning: Issues and Innovations," unpublished manuscript, September 1998.
S. Tuljapurkar, N. Li., C.Boe, "A Universal Pattern of Mortality Decline in the G7 Countries," *Nature*, 405:789-792, 2000.

Statistics Canada: Canada, Statistics Canada, *Population Projections for Canada, Provinces, and Territories: 2000-2026*, by M.V. George, S. Loh, R.B.P. Verma and Y.E. Shin, 2001.

Several other sets of mortality projections essentially approximate those of the SSA. As Table 11 suggests, the projections of life expectancy of the U.S. Census Bureau, the U.S. Actuary's Office, Lee-Tuljapurkar, Statistics Canada and Olshansky/Furner are roughly similar for 2025-2050. On the other hand, the projections of Ahlberg-Vaupel, Oeppen-Vaupel (not shown) and Manton-Stallard-Tolley (not shown) offer high figures for life expectancy at birth that reach 100 before the end of this century. I think, on balance, that the more conservative projections of life expectancy for mid-century of the two federal agencies, Olshansky and Furner, and Lee-Tuljapurkar are to be preferred to the more radical ones proposed or supported by Oeppen-Vaupel, Manton et al. and Wilmoth.

5. Conclusion

The probability of few or no survivors at the very advanced ages is still very high, and supercentenarians are extremely rare. Simply because certainty of death is not explicitly reached does not preclude a near-zero probability of survivorship, and, with present mortality rates, this situation is reached approximately by ages 100-105. Moreover, the fact that one cannot prove that life expectancy and life span have no current ceilings does not prove that they have no current ceilings. If death rates at the oldest ages decline over time and, as a result, relatively more people survive to these ages, life expectation will tend to rise and one may then reasonably hypothesize that an immediate limit to life expectation is not in sight. Even so, this outcome is not necessarily probative of the notion that there is no practical limit to life expectancy or life span. As Wilmoth (1997) recognizes, the link between the patterns of mortality change that we have seen recently and the hypothesis that there is no limit to mortality decline "relies on intuition rather than on firm theoretical results."

One explanation offered for the steady rise in maximum recorded life span, namely the increasing size of national populations (or the increasing size of birth cohorts), has limited probative value. The conclusion depends in part on the assumption that death rates do not rise at the highest ages. Disregarding the factor of immigration, the argument is true if (1) the number of survivors to advanced ages is increasing as a result of an increase in the size of birth cohorts and/or unchanging or falling death rates up to the advanced ages and (2) death rates at the highest ages remain unchanged or fall. It is not true if (1) the number of survivors to advanced ages falls because of declining size of birth cohorts or an increase in mortality up to the advanced ages and (2) mortality at the highest ages rises. It mainly depends on what happens to mortality rates at the upper end of the age scale, and we do not know with certainty what will happen. This is evident from a comparison of two life tables with

greatly different mortality rates, e.g., the SSA life tables for 2000 and 2100 (U.S. Office of the Chief Actuary, 2002). With initial cohorts of equal size (100,000), the last survivor in the first table is age 113, and the last survivor in the second table is over age 120—all based on the assumption of declining mortality over the century.

Focusing on the size of birth cohorts instead of survival rates, if birth cohorts fall, as in many countries of Western Europe, then we should expect maximum recorded life span to move in reverse. One can also argue that the phenomenon of a rising maximum age at death is partly a statistical artifact. Consider the analogy to statistical sampling error in relation to the size of the sample population. The sampling error of an "estimate" of the age of the oldest person from a small "sample" population would be wider than the "estimate" from a large "sample" population. Hence, the confidence interval of the first estimate may very well overlap with the confidence interval of the second estimate and may even encompass it.

We have to be mindful of the insights provided by evolutionary biology to research on human longevity, but we must not be too ready to apply findings about subhuman species to humans, especially the findings about the longevity genes of the few lower-order invertebrates that have been studied. Knowledge remains very limited with respect to the range of species studied under natural conditions from which generalizations can be made. Moreover, while humans have the capacity to apply accumulated knowledge about themselves to themselves and, to a limited extent, determine their own biological destiny, they still embody reliability systems that are products of biological evolution and that expire under appropriate conditions.

There is considerable logic in projecting past trends of mortality in one form or another, because it incorporates the observed and unobserved wisdom of the past and the effect of changing influences of the past on mortality (e.g., epidemiological transition), and it makes possible separate projective judgments about important compositional/etiological elements (e.g., endogenous/exogenous mortality). On the other hand, projection of past trends cannot allow for the emergence of new factors, the turning points and the unexpected future changes that are sure to occur. Our considerable experience with population projections should remind us that overconfidence with any doctrinaire position can be risky. Recall how demographers failed to predict the baby boom after 1945, the baby bust after 1965, the lack of progress in mortality between 1954 and 1968, the sharp decline in mortality from heart disease and cerebrovascular disease after 1968, and the general lack of progress in reducing cancer in the last several decades. Twenty years ago, we could not have predicted the AIDS epidemic, the obesity epidemic and the re-emergence of old infectious diseases that are occurring today. Surprises will not stop occurring; trends in mortality, as in

fertility, may decelerate sharply, accelerate sharply or even change direction. Unexpected influences can emerge that make our complex technical manipulations and demographic logic ineffective as predictors of the future.

The issue of the detail of disaggregation to be used in any projection is a perennial one, and so far, detail by cause of death has not proved to be a solution of the problem of the accuracy of population projections. The future is simply too hard to predict with confidence, no matter how complex we structure our projections, however we disaggregate the data and how logical our demographic analysis. There is no way of developing forecasts of life expectation with an acceptable confidence interval, say 95 percent, that is narrow enough to provide meaningful guidance to policymakers. We can strive for maximum accuracy, but at best we can achieve greater analytical utility.

Finally, I am not arguing against research on methods of projecting mortality. We must proceed with studies comparing alternative methods of projecting mortality and alternate constellations of the data, employing both global measures of mortality and measures defined by various degrees of disaggregation. In addition, research on competing causes may lead to more fundamental designs for structuring the causes of death. Such research may add to the analytical utility of projections, but do not count on it to add to their accuracy. At the same time, to support the goal of providing the most useful information to policymakers, it is important to continue to explore ways of conveying the degree of uncertainty in the projections prepared, whether in the form of alternative assumptions providing a range of figures, alternative methods with variable weighting of the results, stochastic demographic forecasting, simulation modeling and ex post facto evaluations of projections.

Notes

[1] Because the exact figure for the number of years added depends on the detail of the cause-of-death classification for which cause-elimination life tables are available, it is not a unique number and should be considered only an estimate of the total years added. For 1969-71, I had to make an estimate of gains for "all other causes."

[2] Although this comparison is affected by changes in the classification from the Sixth Revision of the International List of the Classification of Diseases to the Tenth Revision of the International List, the effect of any bias is largely negated by the broad grouping of deaths by cause.

[3] The Index of Dissimilarity for the 1950-2000 comparison indicates the extent of disagreement between the two distributions (patterns) in these two years, with limits of 0 percent and 100 percent. It is calculated as follows: (1) The age-specific death rates for all endogenous causes combined (and all exogenous causes combined) are summed over all ages in each of the years 1950 and 2000, (2) the percent distribution of the endogenous (and exogenous) age schedule is computed for 1950 and 2000, (3) the algebraic differences between the endogenous (and exogenous) series for 1950 and 2000 are taken and (4) the sums of the positive and negative differences over all ages between 1950 and 2000 are obtained. The Index of Dissimilarity is the positive or negative sum (the two being equal) of the differences:

$$ID = \frac{1}{2} \left[\sum_a (r_{a50} - r_{a00}) \right]$$

where r_a represents the age-specific death rate (as percent of total) in the indicated year.

The very high rates at the older ages tend to dominate the percent distributions, giving them undue influence on the resulting indexes. To adjust for this limitation, the age-specific rates were first weighted by the population distribution in each year, the 1950 rates being weighted by the 1950 population and the 2000 rates being weighted by the 2000 population. Then, the percent distributions of the rates in step (2) were calculated as preliminary to steps (3) and (4).

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