SPECIAL REPORT

A Potential Decline in Life Expectancy in the United States in the 21st Century

S. Jay Olshansky, Ph.D., Douglas J. Passaro, M.D., Ronald C. Hershow, M.D., Jennifer Layden, M.P.H., Bruce A. Carnes, Ph.D., Jacob Brody, M.D., Leonard Hayflick, Ph.D., Robert N. Butler, M.D., David B. Allison, Ph.D., and David S. Ludwig, M.D., Ph.D.

SUMMARY

Forecasts of life expectancy are an important component of public policy that influence age-based entitlement programs such as Social Security and Medicare. Although the Social Security Administration recently raised its estimates of how long Americans are going to live in the 21st century, current trends in obesity in the United States suggest that these estimates may not be accurate. From our analysis of the effect of obesity on longevity, we conclude that the steady rise in life expectancy during the past two centuries may soon come to an end.

The trend in the life expectancy of humans during the past thousand years has been characterized by a slow, steady increase[1,2] — a pattern frequently punctuated by a volatility in death rates caused by epidemics and pandemic infectious diseases, famines, and war.[3,4] This volatility was dramatically curtailed in the mid-19th century as infectious diseases yielded swiftly to improved living conditions, advances in public health, and medical interventions. During the past 30 years, the rise in life expectancy at birth in the United States decelerated relative to this historical pattern, and gains in life expectancy at older ages are now much smaller than they were in previous decades.[5]

How much higher can life expectancy rise? This is not just an academic question. The answer formulated today will have substantial influence on the rate at which taxes are levied and on the potential solvency of age-entitlement programs. Some scientists answer this question by extrapolating from historical trends, which has led to the recent prediction that life expectancy at birth will rise to 100 years in the United States and other developed nations by the year 2060.[6] The United Nations used a similar method but different assumptions to arrive at a projected life expectancy of 100 years for males and females in most countries by the year 2300.[7] The Social Security Administration (SSA) arrived at a more tempered but still optimistic view that life expectancy in the United States will continue its steady increases, reaching the mid-80s later in this century.[8]

A recently convened panel of advisers,[9] and some mathematical demographers who advocate the use of extrapolation,[10] have advised the SSA to project an even more rapid rate of increase in life expectancy for the U.S. population beyond that already anticipated between now and the latter part of this century. The bases for this advice include a demonstration that the maximum life span in Sweden has increased since the mid-19th century,[11] the world record for life expectancy at birth in developed nations has been increasing by three months per year since 1850, mortality declines occurred at older ages in the Group of Seven industrialized nations during the latter half of the 20th century,[12] and the prediction that "negligible senescence" will be scientifically engineered for humans in this century.[13] Negligible senescence is defined as age-specific mortality rates that remain constant throughout life as opposed to rising exponentially after puberty, which is common among humans and most other animals. This last point is important because it is the only "biologic" justification offered for the decision to raise forecasts of life expectancy.

Life-extending technology that might lead to much higher life expectancies does not yet exist and, should it be developed, must be widely implemented before it would influence statistics on population levels. We believe that potential forms of technology do not justify developing or revising forecasts of life expectancy. Extrapolation models fail to consider the health status of people currently alive and explicitly assume that the past can predict the future. Given that past gains in life expectancy have largely been a product of saving the young, and since future gains must result from extending life among the old, another quantum leap in life expectancy can occur only if the future is different from the past.[14]

An informed approach to forecasting life expectancy should rely on trends in health and mortality that may be observed in the current population. Forecasting life expectancy by extrapolating from the past is like forecasting the weather on the basis of its history. Looking out the window, we see a threatening storm — obesity — that will, if unchecked, have a negative effect on life expectancy. Despite widespread knowledge about how to reduce the severity of the problem, observed trends in obesity continue to worsen. These trends threaten to diminish the health and life expectancy of current and future generations.

THE RISE IN OBESITY

After remaining relatively stable in the 1960s and 1970s, the prevalence of obesity among adults in the United States increased by approximately 50 percent per decade throughout the 1980s and 1990s.[15] Two thirds of adults in the United States today are obese or overweight. In the United States, 28 percent of men, 34 percent of women, and nearly 50 percent of non-Hispanic black women are currently obese. The distribution of body-mass index (BMI, the weight in kilograms divided by the square of the height in meters)[16,17] has shifted in a skewed fashion such that the proportion of people with extreme obesity has increased at an especially rapid rate. These trends have affected all major racial and ethnic groups, all regions of the country, and all socioeconomic strata,[18] with the largest increases in obesity occurring among children and minorities.[19] It should be noted, however, that data from studies on the relation between BMI and mortality have been interpreted by some to suggest that current tables of ideal height and weight and, by extension, ideal ranges of BMI should be adjusted to include lower ideal weights for height or BMIs before age 40, as well as higher ideal weights for height or BMIs after age 40.[20] Redefining ideal weights for height in this way would increase the projected negative effect of obesity on life expectancy because of the large increases in obesity now observed among people under age 40.

Obesity is a multisystem condition associated with an elevated risk of type 2 diabetes, coronary heart disease, cancer, and other complications.[21,22] The effect of body weight on mortality has been studied extensively. In a study of more than a million U.S. adults, the lowest death rates were found among men with a BMI of 23.5 to 24.9 and among women with a BMI of 22.0 to 23.4.[23] Death rates from cardiovascular diseases were substantially elevated among people with higher BMIs. A prospective study of 6139 subjects in Germany found the greatest obesity-associated excess mortality to be among the young[24] — the standardized mortality ratio for people 18 to 29 years of age with a BMI of 40 or over was 4.2 in men and 3.8 in women. Fontaine et al. estimated the effect of obesity on years of life lost across the lifespan of adults.[25] For any degree of excessive body weight, young age was associated with greater years of life lost. Allison et al. used data from six cohort studies in the United States to determine that obesity causes approximately 300,000 deaths per year,[26] although a study by the Centers for Disease Control and Prevention (CDC)[27] may have overestimated deaths due to obesity.

Being overweight in childhood increases the risk among men of death from any cause and death from cardiovascular disease; it also increases the risk of cardiovascular morbidity among both men and women.[28] The lifetime risk of diabetes among people born in the United States has risen rapidly to 30 to 40 percent — a phenomenon presumably attributable to the obesity "epidemic."[29] Having diabetes in adulthood increases the risk of a heart attack by as much as having had a previous heart attack,[30] and the life-shortening effect of diabetes is approximately 13 years.[31] Evidence also suggests that at younger ages, disability rates have risen and fitness levels have declined dramatically in the United States, with both trends attributed, at least in part, to the rise in obesity.[32,33] The incidence of type 2 diabetes in childhood in the United States has increased many times over in the past two decades, an increase that is due almost entirely to the obesity epidemic[34,35]; shockingly, life-threatening complications, including renal failure, may develop by young adulthood in at least 10 percent of children with type 2 diabetes.[36]

If left unchecked, the rising prevalence of obesity that has already occurred in the past 30 years is expected to lead to an elevated risk of a range of fatal and nonfatal conditions for these cohorts as they age.[29] If the prevalence of obesity continues to rise, especially at younger ages, the negative effect on health and longevity in the coming decades could be much worse. It is not possible to predict exactly when obesity among the young will have its largest negative effect on life expectancy. However, in the absence of successful interventions, it seems likely that it will be in the first half of this century, when at-risk populations reach the ages of greatest vulnerability.

OBESITY AND FUTURE LIFE EXPECTANCY

Obesity has been shown to have a substantial negative effect on longevity, reducing the length of life of people who are severely obese by an estimated 5 to 20 years.[25] Although the life-shortening effect of obesity is evident for people who are obese, its negative effect on the future life expectancy of the population is also critically important to public policy.

We have estimated the effect of obesity on the life expectancy of the U.S. population by calculating the reduction in the rates of death that would occur if everyone who is currently obese were to lose enough weight to obtain an "optimal" BMI, which we defined as a BMI of 24. This calculation can be performed by linking data on the age-, race-, and sex-specific prevalence of obesity in the United States from the Third National Health and Nutrition Examination Survey[15] with estimates of race- and sex-specific rates of death for people 20 to 85 years of age who have BMIs that range from 17 to 45.[37] Complete life tables for the United States according to race and sex for 2000 were obtained from the National Center for Health Statistics.[38]

The calculations begin by estimating the conditional probability of death at age x of the nonobese population, $q(x_{no})$, as follows:

$$q(x_{no}) = [q(x) - q(x_o)\,P(x_o)] \div P(x_{no}),$$

where $q(x)$ is the conditional probability of death at age x for the entire population, $q(x_o)$ is the conditional probability of death at age x for the obese population, $P(x_o)$ is the proportion of the population at age x that is obese, and $P(x_{no})$ is $1-P(x_o)$, which is the proportion of the population at age x that is not obese.[15,37,38]

Because the distribution of BMIs is skewed toward higher levels, and the population is shifting toward higher BMIs, we provide a range of estimates for the effect of obesity on life expectancy by assuming that everyone who is obese has a BMI of 30 or 35. The effect of eliminating obesity on the life expectancy of the population (encompassed within this range) is then estimated by assuming that everyone who is obese acquires the mortality risk, $q(x_{24})$, of those with the optimal BMI of 24. The obesity-adjusted conditional probability of death, $q(x_a)$, at age x may then be obtained as follows:

$$q(x_a) = q(x_{no})\,P(x_{no}) + q(x_{24})\,P(x_o).$$

These obesity-adjusted conditional probabilities of death were estimated separately by race and sex. When a BMI of 30 or 35 did not lead to lower death rates than the optimal BMI of 24, it was assumed that there was no change in the conditional probability of death. This occurred for black males at 62 to 85 years of age at a BMI of 30 and at 67 to 85 years of age at a BMI of 35, and for black females at 60 to 85 years of age at a BMI of 30 and at 67 to 85 years of age at a BMI of 35. These calculations yielded a range of age-, race-, and sex-specific rates of death and hypothetical life tables for the year 2000 under the assumption that obesity has been eliminated.

A more detailed analysis of this kind could be performed using the full age distribution of BMIs and integrating these into the calculations at a finer level of detail. However, given that the methods required for such estimates are currently being reviewed by a panel of scientists convened by the Institute of Medicine, and in the interest of identifying plausible estimates rather than precise numbers, we took this simpler approach.

Our approach probably underestimates the negative effect of obesity on life expectancy at BMIs of 30 and overestimates it at BMIs of 35. Clearly, the absence of rates of death for obese people under 19 years of age (which forces the assumption that rates of death in this age range remained unchanged from the levels in 2000) leads to an underestimate of the overall effect of obesity on life expectancy. Finally, it is possible that the use of period (cross-sectional) rather than cohort data may also lead to substantial underestimates of the effects of obesity on life expectancy.[39]

Our conservative estimate is that life expectancy at birth in the United States would be higher by 0.33 to 0.93 year for white males, 0.30 to 0.81 year for white females, 0.30 to 1.08 years for black males, and 0.21 to 0.73 year for black females if obesity did not exist (Fig. 1). Assuming that current rates of death associated with obesity remain constant in this century, the overall negative effect of obesity on life expectancy in the United States is a reduction in life expectancy of one third to three fourths of a year. This reduction in life expectancy is not trivial — it is larger than the negative effect of all accidental deaths combined (e.g., accidents, homicide, and suicide),[40] and there is reason to believe that it will rapidly approach and could exceed the negative effect that ischemic heart disease or cancer has on life expectancy.

Several facts that suggest that the prevalence and severity of obesity and its complications will worsen and that rates of obesity-induced death will rise are as follows: current estimates of the effect of eliminating obesity are based on past trends, when the prevalence was much lower; the prevalence of obesity, especially among children, is likely to continue to rise; with obesity occurring at younger ages, the children and young adults of today will carry and express obesity-related risks for more of their lifetime than previous generations have done; a significant shift toward higher BMIs throughout the age ranges has occurred; death rates from diabetes have risen steadily in the past 20 years and are expected to rise further as younger cohorts age; and the medical treatment of obesity has been largely unsuccessful.[22] These trends suggest that the relative influence of obesity on the life expectancy of future generations could be markedly worse than it is for current generations. In other words, the life-shortening effect of obesity could rise from its current level of about one third to three fourths of a year to two to five years, or more, in the coming decades, as the obese who are now at younger ages carry their elevated risk of death into middle and older ages.

DISCUSSION

A rise in life expectancy to 100 years in the United States in this century would profoundly influence many aspects of society,[41] including the solvency of age-based entitlement programs and tax rates levied by the federal government. Even marginal increases in life expectancy beyond those anticipated by the SSA would markedly increase the number of octogenarians, nonagenarians, and centenarians that the SSA expects. However, in light of the obesity-driven trends in the health status of the U.S. popula-

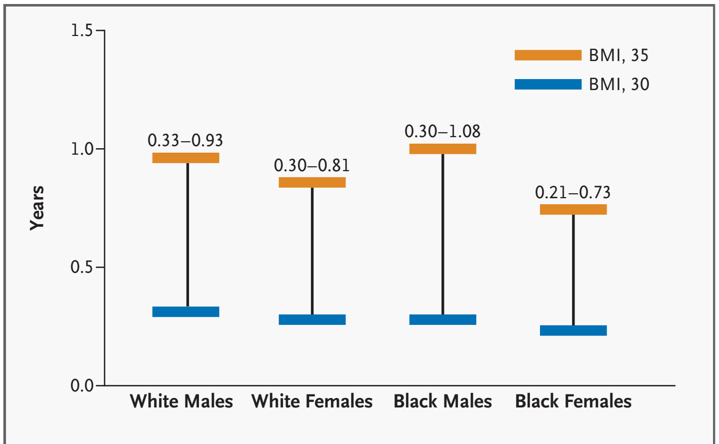

Figure 1. Life-Shortening Effect of Obesity According to Race and Sex in the United States in 2000.

This figure shows the potential gain in life expectancy at birth for the U.S. population in 2000, by race and sex, if obesity were eliminated. The range of estimates is shown between the bars on the basis of the assumption that everyone who is obese has a body-mass index (BMI) between 30 (lower bar) and 35 (upper bar) and acquires the risk of death of people with a BMI of 24. The horizontal bars are not error bars.

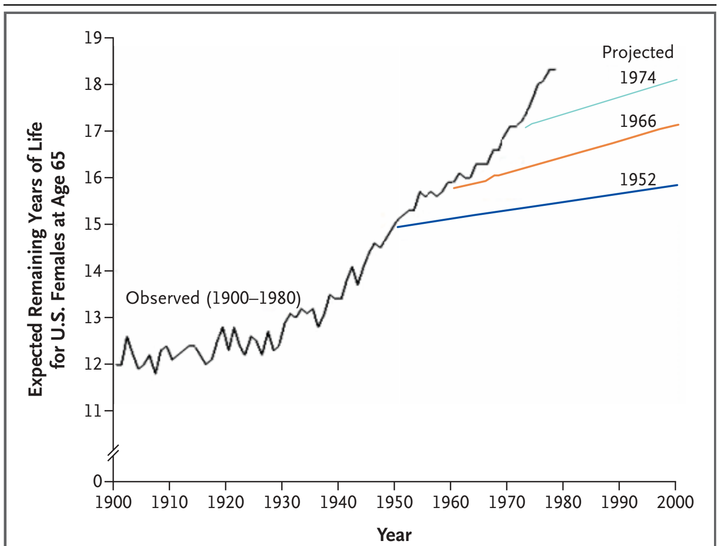

Figure 2. Observed and Projected Life Expectancy at Age 65 for U.S. Females (1900 to 2000).

Shown are observed changes, from 1900 to 1980,[42] in expected remaining years of life at age 65 for females in the United States, and projections of the expected remaining years of life at age 65 made by the SSA in actuarial studies published in 1952,[43] 1966,[44] and 1974.[45]

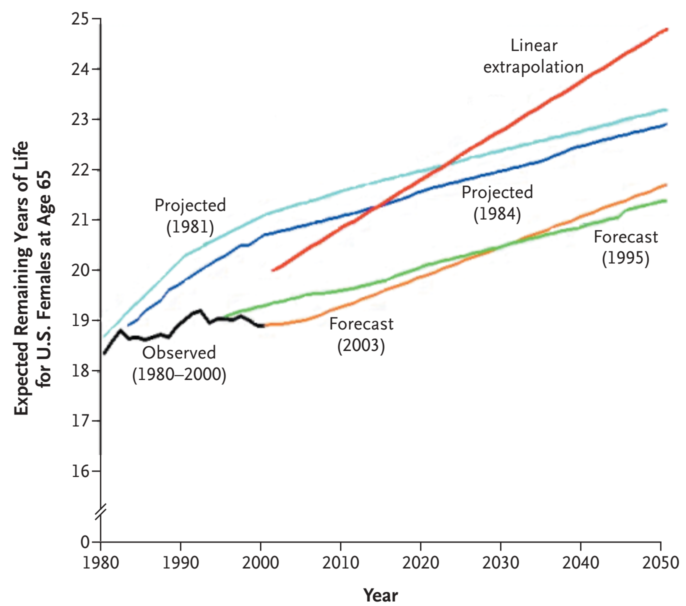

Figure 3. Observed and Projected Life Expectancy at Age 65 for U.S. Females (1980 to 2050).

Shown are observed changes, from 1980 to 2000,[46] in expected remaining years of life at age 65 for females in the United States, projections of the expected remaining years of life at age 65 made by the SSA in actuarial studies published in 1981[46] and 1984,[47] and forecasts based on the SSA's 1995 and 2003 Trustees Reports.[48,49] A forecast of the expected remaining years of life at age 65 for females in the United States, assuming the observed trend from 1940 to 2000 is extrapolated linearly from 2000 to 2050, is shown.

tion (especially the young), the bases for the SSA's recent decision to raise its midrange forecasts of life expectancy beyond the increases anticipated during the next 70 years merit reconsideration.

Figure 2 shows the historical trend in life expectancy at age 65 for females in the United States from 1900 through the inception of the Social Security program, in 1935, and formal projections of the rise in life expectancy at age 65 made by the SSA at various times since then. Before 1980, the SSA consistently underestimated the subsequent rise in life expectancy at age 65 because it assumed that recently observed gains could not be sustained. After 1980, this position was reversed, and the SSA began tracking and extrapolating more recently observed trends in life expectancy at age 65. Ironically, this change in approach occurred just when the rise in life expectancy began to stall (Fig. 3). The fact is, life expectancy at age 65 for females has remained largely unchanged for most of the past 20 years.

A central problem with the SSA's forecast is best illustrated by its projections for diabetes. From 1979 to 1999, rates of death from diabetes increased annually by an average of 2.8 percent for males and 1.8 percent for females. In 1990, diabetes decreased life expectancy by 0.22 year for males and 0.31 year for females,[40] but the negative effect of diabetes on life expectancy has grown rapidly since then. However, the negative effect of diabetes on the life expectancy of the population could now be several times as great as it was in 1990.[31] Given the rapidly rising prevalence of diabetes and the prospect that childhood obesity today will probably accelerate the rising prevalence of diabetes in the coming decades, it is difficult to justify the SSA's assumption that rates of death from diabetes will decline by 1.0 percent to 3.2 percent annually throughout the 21st century, beginning in the year 2010.[5]

We anticipate that as a result of the substantial rise in the prevalence of obesity and its life-shortening complications such as diabetes, life expectancy at birth and at older ages could level off or even decline within the first half of this century. This is in contrast to both the recent decision by the SSA to raise its forecast of life expectancy and what we consider to be the simple but unrealistic extrapolation of past trends in life expectancy into the future.

There are other realistic threats to increases in life expectancy. From 1980 to 1992 in the United States, the age-adjusted rate of death from infectious diseases rose by 39 percent, an increase fueled mostly by the AIDS epidemic; the overall rate of death from infectious diseases increased 4.8 percent per year from 1980 to 1995[50]; hospital-acquired infections have increased[51,52]; hospital-acquired and antibiotic-resistant pathogens have entered the community and our food supply[53,54]; and recent decreases in mortality related to the human immunodeficiency virus have leveled off.[55]

Infectious diseases could decrease life expectancy substantially if pandemic influenza strikes.[56] Developing and developed nations are far more vulnerable to a global pandemic of influenza today than in 1918, owing to an aging population, resistance to antibiotics, and more rapid transport of microbes, among other reasons. This heightened risk is balanced in part by better global surveillance and interventions already present.[57] Although estimating the negative effects of epidemics on the future course of life expectancy is problematic, it has been established that infectious diseases, when they do emerge, can wipe out a century's worth of gains in health and longevity in less than one generation.[58]

Other forces that could attenuate the rise in life expectancy include pollution, lack of regular exercise, ineffective blood-pressure screening, tobacco use, and stress.

Advances in the medical treatment of major fatal diseases, including the complications of obesity, are likely to continue. Unfortunately, recent trends in the prevalence of cancer and in the rates of death from cardiovascular diseases in the United States reveal only marginal gains in longevity in recent decades,[59,60] and even the gains produced from the elimination of any one of today's major fatal diseases[61] would not exceed the negative effects of obesity that appear to be forthcoming.

A leveling off or decline in life expectancy in the United States is not inevitable. We remain hopeful that the public health community and public policymakers will respond to the impending dangers that obesity poses to both the quality and the length of life. However, the negative effect on health and longevity of unchecked obesity is substantial according to statistics on health and mortality that can be observed for the generations currently alive, as has already been shown in Okinawa, Japan.[62] It is important to emphasize that our conclusions about the future are based on our collective judgment, as are all forecasts, and we acknowledge that forces that influence human mortality can change rapidly.

Finally, our forecast has other public-policy implications. Dire predictions about the impending bankruptcy of Social Security based on the SSA's projections of large increases in survival past 65 years of age appear to be premature. However, this "benefit" will occur at the expense of the economy in the form of lost productivity before citizens reach retirement and large increases in Medicare costs associated with obesity and its complications.[63] Presently, annual health care costs attributable to obesity are conservatively estimated at $70 billion to $100 billion.[64,65] With rapid increases in the prevalence of diabetes, and a decrease in mean age at the onset of diabetes, the cost of treating diabetes-related complications, such as heart disease, stroke, limb amputation, renal failure, and blindness, will increase substantially. A similar escalation of health care costs from other complications associated with obesity (e.g., cardiovascular disease, hypertension, asthma, cancer, and gastrointestinal problems) is inevitable. The U.S. population may be inadvertently saving Social Security by becoming more obese, but the price to be paid by obese people themselves and the economy is already high enough to justify considerably increased spending on public health interventions[66] aimed at reducing the incidence and severity of obesity.

Unless effective population-level interventions to reduce obesity are developed, the steady rise in life expectancy observed in the modern era may soon come to an end and the youth of today may, on average, live less healthy and possibly even shorter lives than their parents. The health and life expectancy of minority populations may be hit hardest by obesity, because within these subgroups, access to health care is limited and childhood and adult obesity has increased the fastest.[67] In fact, if the negative effect of obesity on life expectancy continues to worsen, and current trends in prevalence suggest it will, then gains in health and longevity that have taken decades to achieve may be quickly reversed. The optimism of scientists and of policymaking bodies about the future course of life expectancy should be tempered by a realistic acknowledgment that major threats to the health and longevity of younger generations today are already visible.

Supported by grants (K02 AG00785-05, to Dr. Olshansky, and AG00979-05, to Dr. Carnes) from the National Institute on Aging and a grant (1R01DK59240, to Dr. Ludwig) from the National Institute of Diabetes and Digestive and Kidney Diseases. Dr. Olshansky is also supported by the Institute of Government and Public Affairs at the University of Illinois at Chicago, and Dr. Ludwig is also supported by the Charles H. Hood Foundation.

Dr. Allison has disclosed multiple financial associations. These can be found at www.nejm.org with the full text of this article.

The assumptions and conclusions herein are the sole responsibility of the authors.

We are indebted to Mark Grant for constructive comments on an earlier draft of the manuscript.

From the University of Illinois at Chicago, Chicago (S.J.O., D.J.P., R.C.H., J.L., J.B.); University of Oklahoma Health Sciences Center, Oklahoma City (B.A.C.); University of California at San Francisco, San Francisco (L.H.); International Longevity Center, New York (R.N.B.); University of Alabama, Birmingham (D.B.A.); and Children's Hospital, Boston (D.S.L.). Address reprint requests to Dr. Olshansky at the University of Illinois, 1603 W. Taylor St., Rm. 885, Chicago, IL 60612, or at sjayo@uic.edu.

1. Macdonell WR. On the expectation of life in ancient Rome, and in the provinces of Hispania and Lusitania, and Africa. Biometrika 1913;9:366-80.

2. Faber JF. Life tables for the United States: 1900-2050. Actuarial study no. 87. Baltimore: Social Security Administration, 1982. (SSA publication no. 11-11534.)

3. Olshansky SJ, Carnes BA. Prospects for extended survival: a critical review of the biological evidence. In: Caselli G, Lopez AD, eds. Health and mortality among elderly populations. Oxford, England: Oxford University Press, 1996:39-58.

4. McNeill WH. Plagues and peoples. Garden City, N.Y.: Anchor Press, 1976.

5. Bell FC, Miller ML. Life tables for the United States Social Security area, 1900-2100. Actuarial study no. 116. Baltimore: Social Security Administration, 2002.

6. Oeppen J, Vaupel J. Broken limits to life expectancy. Science 2002;296:1029-31.

7. Department of Economic and Social Affairs. World population to 2300. New York: United Nations, 2004. (ST/ESA/SER.A/236.)
8. Cheng AW, Miller ML, Morris M, Schultz JP, Skirvin JP, Walder DP. A stochastic model of the long range financial status of the OASDI program. Actuarial study no. 117. Baltimore: Social Security Administration, 2004. (SSA publication no. 11-11543.)
9. 2003 Report to the Social Security Advisory Board. Washington, D.C.: Technical Panel on Assumptions and Methods, 2003.
10. Testimony before the trustees of the Social Security Administration by Dr. S. Jay Olshansky, Dr. John Wilmoth, and Dr. Ron Lee, September 13, 2002. Baltimore: Social Security Administration, 2002 (transcript).
11. Wilmoth JR, Deegan LJ, Lundstrom H, Horiuchi S. Increase of maximum life-span in Sweden: 1861-1999. Science 2000;289: 2366-8.
12. Tuljapurkar S, Li N, Boe C. A universal pattern of mortality decline in the G7 countries. Nature 2000;405:789-92.
13. de Grey ADNJ, Ames BN, Andersen JK, et al. Time to talk SENS: critiquing the immutability of human aging. Ann N Y Acad Sci 2002; 959:452-62.
14. Olshansky SJ, Carnes BA, Désesquelles A. Demography: prospects for human longevity. Science 2001;291:1491-2.
15. Flegal KM, Carroll MD, Ogden CL, Johnson CL. Prevalence and trends in obesity among US adults, 1999-2000. JAMA 2002;288: 1723-7.
16. Pietrobelli A, Heymsfield SB. Establishing body composition in obesity. J Endocrinol Invest 2002;25:884-92.
17. NHLBI Obesity Education Initiative. The practical guide to the identification, evaluation, and treatment of overweight and obesity in adults. Rockville, Md.: National Institutes of Health, 2000. (NIH publication no. 00-4084.)
18. Mokdad AH, Bowman BA, Ford ES, Vinicor F, Marks JS, Koplan JP. The continuing epidemics of obesity and diabetes in the United States. JAMA 2001;286:1195-200.
19. Ogden CL, Flegal KM, Carroll MD, Johnson CL. Prevalence and trends in overweight among US children and adolescents, 1999-2000. JAMA 2002;288:1728-32.
20. Andres R, Elahi D, Tobin JD, Muller DC, Brant L. Impact of age on weight goals. Ann Intern Med 1985;103:1030-3.
21. Must A, Spadano J, Coakley EH, Field AE, Colditz G, Dietz WH. The disease burden associated with overweight and obesity. JAMA 1999;282:1523-9.
22. Ebbeling CB, Pawlak DB, Ludwig DS. Childhood obesity: public-health crisis, common sense cure. Lancet 2002;360:473-82.
23. Calle EE, Thun MJ, Petrelli JM, Rodriguez C, Heath CW Jr. Body-mass index and mortality in a prospective cohort of U.S. adults. N Engl J Med 1999;341:1097-105.
24. Bender R, Jockel KH, Trautner C, Spraul M, Berger M. Effect of age on excess mortality in obesity. JAMA 1999;281:1498-504.
25. Fontaine KR, Redden DT, Wang C, Westfall AO, Allison DB. Years of life lost due to obesity. JAMA 2003;289:187-93.
26. Allison DB, Fontaine KR, Manson JE, Stevens J, VanItallie TB. Annual deaths attributable to obesity in the United States. JAMA 1999;282:1530-8.
27. Mokdad AH, Marks JS, Stroup DF, Gerberding JL. Actual causes of death in the United States, 2000. JAMA 2004;291:1238-45.
28. Must A, Jacques PF, Dallal GE, Bajema CJ, Dietz WH. Long-term morbidity and mortality of overweight adolescents: a follow-up of the Harvard Growth Study of 1922 to 1935. N Engl J Med 1992;327: 1350-5.
29. Koplan JP, Liverman CT, Kraak VI, eds. Preventing childhood obesity: health in the balance. Washington, D.C.: National Academies Press, 2005.
30. Haffner SM, Lehto S, Rönnemaa T, Pyörälä K, Laakso M. Mortality from coronary heart disease in subjects with type 2 diabetes and in nondiabetic subjects with and without prior myocardial infarction. N Engl J Med 1998;339:229-34.
31. Manuel DG, Schultz SE. Health-related quality of life and health-adjusted life expectancy of people with diabetes in Ontario, Canada, 1996-1997. Diabetes Care 2004;27:407-14.
32. Lakdawalla DN, Bhattacharya J, Goldman DP. Are the young becoming more disabled? Health Aff (Millwood) 2004;23(1):168-76.
33. Carnethon MR, Gidding SS, Nehgme R, Sidney S, Jacobs DR Jr, Liu K. Cardiorespiratory fitness in young adulthood and the development of cardiovascular disease risk factors. JAMA 2003;290: 3092-100.
34. Fagot-Campagna A, Pettitt DJ, Engelgau MM, et al. Type 2 diabetes among North American children and adolescents: an epidemiologic review and a public health perspective. J Pediatr 2000;136: 664-72.
35. Ludwig DS, Ebbeling CB. Type 2 diabetes mellitus in children: primary care and public health considerations. JAMA 2001;286: 1427-30.
36. Dean H, Flett B. Natural history of type 2 diabetes diagnosed in childhood: long term follow-up in young adult years. Diabetes 2002;51:Suppl 2:A24. abstract.
37. University of Alabama School of Public Health. Research tables. (Accessed February 24, 2005, at http://www.soph.uab.edu/statgenetics/Research/Tables/YLL.htm.)
38. Arias E. United States life tables, 2000. National and vital statistics. Vol. 51. No. 3. Hyattsville, Md.: National Center for Health Statistics, 2002:15-8, 21-4. (DHHS publication no. (PHS) 2003-1120 02-0644.)
39. Peeters A, Bonneux L, Barendregt J, Nusselder W. Methods of estimating years of life lost due to obesity. JAMA 2003;289:2941.
40. Anderson RN. U.S. decennial life tables for 1989-91. Vol. 1. No. 4. United States life tables eliminating certain causes of death. Hyattsville, Md.: National Center for Health Statistics, 1999. (DHHS publication no. (PHS) 99-1150-4.)
41. Beyond therapy: biotechnology and the pursuit of happiness. Washington, D.C.: President's Council on Bioethics, 2004.
42. Wade A. Social Security area population projections, 1986. Actuarial study no. 97. Baltimore: Social Security Administration, 1986. (SSA publication no. 11-11544.)
43. Myers RJ, Rasor EA. Illustrative population projections, 1952. Actuarial study no. 33. Baltimore: Social Security Administration, 1952.
44. Bayo F. United States population projections for OASDHI cost estimates. Actuarial study no. 62. Baltimore: Social Security Administration, 1966.
45. Bayo F, McKay SF. United States population projections for OASDHI cost estimates. Actuarial study no. 72. Baltimore: Social Security Administration, 1974.
46. Faber JF, Wilkin JC. Social Security area population projections. Actuarial study no. 85. Baltimore: Social Security Administration, 1981. (SSA publication no. 11-11532.)
47. Wade AH. Social Security area population projections, 1984. Actuarial study no. 92. Baltimore: Social Security Administration, 1984. (SSA publication no. 11-11539.)
48. Social Security Administration. 2003 OASDI trustees report. V. Assumptions and methods underlying actuarial estimates. (Accessed February 24, 2005, at http://www.ssa.gov/OACT/TR/TR03/V_demographic.html.)
49. Idem. Annual report of the board of trustees of the Federal Old-Age and Survivors Insurance and Disability Insurance Trust Funds. 1995. (Accessed February 24, 2005, at http://www.ssa.gov/history/reports/trust/1995/trtoc.html.)
50. Armstrong GL, Conn LA, Pinner RW. Trends in infectious disease mortality in the United States during the 20th century. JAMA 1999;281:61-6.
51. Diekema DJ, Beekmann SE, Chapin KC, Morel KA, Munson E, Doern GV. Epidemiology and outcome of nosocomial and community-onset bloodstream infection. J Clin Microbiol 2003;41:3655-60.
52. Osmon S, Warren D, Seiler SM, Shannon W, Fraser VJ, Kollef

MH. The influence of infection on hospital mortality for patients requiring > 48 h of intensive care. Chest 2003;124:1021-9.
53. Uttley AHC, Collins CH, Naidoo J, George RC. Vancomycin-resistant enterococci. Lancet 1988;1:57-8.
54. McDonald LC, Kuehnert MJ, Tenover FC, Jarvis WR. Vancomycin-resistant enterococci outside the health-care setting: prevalence, sources, and public health implications. Emerg Infect Dis 1997;3:311-7.
55. Karon JM, Fleming PL, Steketee RW, De Cock KM. HIV in the United States at the turn of the century: an epidemic in transition. Am J Public Health 2001;91:1060-8.
56. Influenza pandemic plan: the role of WHO and guidelines for national or regional planning. Geneva: World Health Organization, 1999. (WHO/CDS/CSR/EDC/99.1.)
57. Olshansky SJ, Carnes BA, Rogers RG, Smith L. Infectious diseases — new and ancient threats to world health. Popul Bull 1997;52(2):1-52.
58. Economic and Social Affairs, Population Division. The impact of AIDS. New York: United Nations, 2004. (ST/ESA/SER.A/229.)
59. Decline in deaths from heart disease and stroke — United States, 1900-1999. JAMA 1999;282:724-6.
60. Jemal A, Tiwari RC, Murray T, et al. Cancer statistics, 2004. CA Cancer J Clin 2004;54:8-29.
61. Olshansky SJ, Carnes BA, Cassel C. In search of Methuselah: estimating the upper limits to human longevity. Science 1990;250:634-40.
62. Todoriki H, Willcox DC, Willcox BJ. The effects of post-war dietary change on longevity and health in Okinawa. Okinawan J Am Stud 2004;1:55-64.
63. Daviglus ML, Liu K, Yan LL, et al. Relation of body mass index in young adulthood and middle age to Medicare expenditures in old age. JAMA 2004;292:2743-9.
64. Wolf AM, Colditz GA. Current estimates of the economic cost of obesity in the United States. Obes Res 1998;6:97-106.
65. Allison DB, Zannolli R, Narayan KM. The direct health care costs of obesity in the United States. Am J Public Health 1999;89:1194-9.
66. Prevention of Childhood Obesity Act, S.2894, 108th Cong., 2d Sess. (October 5, 2004).
67. Strauss RS, Pollack HA. Epidemic increase in childhood overweight, 1986-1998. JAMA 2001;286:2845-8.