Pursuing Longevity: Delay vs Elimination of Degenerative Diseases

S. JAY OLSHANSKY, PHD

Abstract: Using a new model which allows for projection of mortality change resulting from preventive health care measures, prospective changes in longevity for the resident United States population in 1978 were compared with projections of longevity gains occurring under a standard single cause-elimination model. Results indicate that equal or greater gains accrue from the prevention or delay of several major degenerative diseases, than from the complete elimination of some single major degenerative diseases. Observed declines in mortality from 1960 to 1978 have resulted in gains in longevity equivalent to the successful elimination of some major degenerative diseases. (*Am J Public Health* 1985; 75:754–757.)

Introduction

Research indicating that the hypothetical elimination of a single major degenerative cause of death would lead to only relatively small gains in life expectancy at birth[1–3] has been challenged by Schatzkin.[4] He suggested that because degenerative diseases share common risk factors,[5,6] and simultaneous/multiple cause-elimination models would yield relatively substantial gains in life expectancy as a result of improved livestyles and preventive health care measures, we have reason for optimism. The main issue of interest, however, as Keyfitz[7] has pointed out, should not be optimism vs pessimism or the exact location of the biological limit to life,[8] but what direction should be taken by scientific research, and public health measures, to ensure that both mortality and morbidity are compressed into advanced ages at nearly the same rate in the future.[9,10]

Until recently, the only way to assess the relationship between improving life conditions, advances in medical technology, and prospective gains in life expectancy was to use extrapolation models based upon past trends in overall or cause-specific mortality.[11–14] These models have been criticized because of their inability to realistically model the relationship between multiple risk factors and dependency among various causes of death.* Recent developments in the modeling of mortality change, however, provide for more valid estimates of the demographic consequences of delaying** or preventing degenerative causes of death.[15] These are referred to as cause-delay models for projecting mortality. In this study, gains in longevity resulting from the hypothetical elimination of selected degenerative diseases are compared to projected gains in longevity using this new model.

Methods

Details of the rationale and assumptions of the delay methodology, including assumptions about delays in selected causes of death, may be found in Olshansky* and in Manton *et al*,[15] (see Appendix). The premise here is that prospective changes in mortality over time have been shown to be a reflection of differences in age-race-sex-cause-specific mortality rates between successive age groups at a given point in time. Overall mortality rates may then be projected into the future based upon assumptions of how long it would take for degenerative diseases most likely to decline in the future, to experience a given hypothetical delay in the forces of their mortality. [A delay in the force of mortality for a given degenerative disease means that successive birth cohorts approaching the same age range (for example 60–64) will experience the observed mortality rates of successively younger age groups (e.g., 55–59 year olds in the case of a 5-year delay) of the period from which the original measurements were taken.]

Based upon observed trends in cause-specific mortality in the United States from 1960 to 1978, and a test of the SIMCAD model on 1960 data for the US, it was decided that a five-year delay for major cardiovascular diseases (8th ICDA: 390–448), diabetes mellitus (250), and some cancers (190–199, males only: 170–174, females only: 170–173, 160–163) would produce percentage changes in mortality over successive 20-year time periods from 1978 that are consistent with historical patterns of change in these diseases. Mortality rates for other causes of death were presumed to remain stable at their observed 1978 levels.

Projected delays were calculated only for the population aged 20 and over. Delays of three-years and seven-years were also calculated in order to provide for a margin of error or a range of possible mortality change projected into the future. The age-race-sex-cause-specific mortality counts for the resident population of the US were provided by the National Center for Health Statistics. Age-race-sex population data were drawn from the latest intercensal estimates of the resident population of the United States provided by the US Census Bureau. [Data are based on revised intercensal estimates of the 1978 resident population that are adjusted for the undercount of Blacks exposed in the 1980 Census.] The mortality and population counts used in the SIMCAD projection model are for the year 1978.

Results

Table 1 shows the gains in life expectancy that are projected to the years 2000 and 2020 for selected ages, by race and sex, based upon the three, five, and seven-year delay assumptions for selected causes of death. The data indicate that even small differences in delay assumptions frequently produce significantly larger gains in life expectancy. An additional set of delays projected from the year 2000 to 2020 also show relatively large gains in life expectancy at all ages.

Table 2 shows gains in life expectancy that would occur with the hypothetical elimination of six selected causes of

*Olshansky SJ: A social epidemiological model for projecting prospective mortality change. Department of Sociology, University of Utah. Submitted for publication.

**In this paper, the term "delay" is considered preferable to "prevention", since it is more realistic to expect that preventive health care measures will redistribute the risk of dying from degenerative diseases from younger to older ages, rather than reducing the risk to zero.

Address reprint requests to Professor S. Jay Olshansky, Department of Sociology, University of Utah, 301 Social/Behavioral Science Building, Salt Lake City UT 84112. This paper, submitted to the Journal November 26, 1984, was revised and accepted for publication February 15, 1985.

TABLE 1—Gains in Life Expectancy (in years) at Selected Exact Ages with the Hypothetical Delay of Selected Causes of Death

	White Males (1)	White Females (2)	Nonwhite Males (3)	Nonwhite Females (4)	White Males (5)	White Females (6)	Nonwhite Males (7)	Nonwhite Females (8)	White Males (9)	White Females (10)	Nonwhite Males (11)	Nonwhite Females (12)
	SIMCAD model (Major Cardiovascular Diseases, Diabetes, and some Cancers) projected to 2000											
Ages	3-Year Delay				5-Year Delay				7-Year Delay			
0	1.8	2.1	1.6	1.9	2.7	3.1	2.3	2.9	3.6	3.9	3.1	3.8
20	1.9	2.1	1.7	2.0	2.8	3.2	2.4	2.9	3.7	4.0	3.2	3.9
40	1.9	2.1	1.7	1.9	2.8	3.2	2.4	2.9	3.7	4.0	3.2	3.9
60	1.6	2.0	1.4	1.7	2.4	3.0	1.9	2.5	3.1	3.8	2.5	3.4
85	1.0	1.3	0.7	0.9	1.3	1.9	1.0	1.4	1.8	2.2	1.4	1.8
	SIMCAD model (Major Cardiovascular Diseases, Diabetes, and some Cancers) projected to 2020											
Ages	3-Year Delay				5-Year Delay				7-Year Delay			
0	3.5	3.9	3.0	3.6	5.0	5.6	4.4	5.3	6.2	6.9	5.5	6.7
20	3.6	4.0	3.2	3.7	5.2	5.7	4.6	5.4	6.4	7.1	5.7	6.9
40	3.6	4.0	3.2	3.6	5.2	5.7	4.6	5.4	6.5	7.1	5.8	6.8
60	3.2	3.8	2.6	3.2	4.5	5.5	3.8	4.7	5.7	6.9	4.8	6.0
85	1.9	2.5	1.3	1.7	2.6	3.6	1.9	2.5	3.2	4.5	2.3	3.1

death in the United States. [These are based upon cause-elimination models applied to mortality data for the US from 1969 to 1971. It is not necessary to apply the cause-elimination model to 1978 data because: 1) the differences in projected gains in life expectancy resulting from the elimination of selected causes of death at two points in time so close to one another would be relatively small; and 2) a more general comparison of delay vs elimination is being considered in this paper.] A comparison of the data in Tables 1 and 2 demonstrates that fairly moderate delays in deaths attributable to major degenerative diseases over the next 20 years would achieve the same or greater extension of life, as that which would have been achieved had we successfully eliminated some major degenerative diseases in 1970. For example, a five-year delay of major cardiovascular diseases, diabetes, mellitus, and some cancers, [Assumptions incorporated into the SIMCAD model include projected increases in some cancer death rates. This was done to conform with recent historical patterns in some causes of death where it is likely that they would continue to increase in the coming decades. This was most apparent for lung cancer death rates among women.] projected to occur by the year 2000, would achieve greater gains in life expectancy at almost all ages, and for all four race-sex groups, than that which would have been achieved had all cancer deaths in 1970 been eliminated. If this same medium-range delay in the same causes of death were achieved from the years 2000 to 2020, we would be closely approximating, and in some cases exceeding, the gains in life expectancy that would occur with the elimination of ischemic heart disease (34.7 per cent of all deaths in 1970).

With the seven-year delay assumptions, we may expect to achieve gains in life expectancy by the year 2020 that would exceed those achieved by the elimination of all deaths attributable to diseases of the heart (38.2 per cent of all deaths in 1970). Indeed, observed mortality declines and gains in life expectancy from 1960 to 1978 for the four race-sex groups

TABLE 2—Gains in Life Expectancy (in years) at Selected Exact Ages with the Hypothetical Elimination of Selected Causes of Death

	White Males (1)	White Females (2)	Nonwhite Males (3)	Nonwhite Females (4)	White Males (5)	White Females (6)	Nonwhite Males (7)	Nonwhite Females (8)	White Males (9)	White Females (10)	Nonwhite Males (11)	Nonwhite Females (12)
Ages	Malignant Neoplasms				Ischemic Heart Disease				Diseases of the Heart			
0	2.3	2.6	2.3	2.4	5.5	4.4	4.2	4.9	6.1	5.2	5.3	6.3
20	2.3	2.6	2.4	2.5	5.7	4.5	4.4	5.1	6.4	5.3	5.6	6.5
40	2.2	2.4	2.5	2.3	5.8	4.6	4.7	5.2	6.4	5.3	5.8	6.6
60	1.7	1.5	1.9	1.5	4.8	4.5	4.2	5.0	5.4	5.1	5.1	6.1
85	0.3	0.3	0.5	0.4	2.3	2.7	2.6	3.3	2.7	3.2	3.2	4.1
Ages	Cerebrovascular Disease				Acute Myocardial Infarction				Influenza and Pneumonia			
0	0.9	1.4	1.4	2.2	3.0	1.8	1.7	1.6	0.4	0.4	0.8	0.7
20	0.9	1.4	1.4	2.2	3.1	1.8	1.8	1.7	0.3	0.3	0.5	0.4
40	0.9	1.4	1.5	2.2	3.2	1.8	1.9	1.7	0.3	0.3	0.5	0.4
60	0.9	1.3	1.4	2.0	2.4	1.7	1.5	1.5	0.2	0.2	0.4	0.3
85	0.6	0.9	0.8	1.2	0.6	0.6	0.7	0.7	0.2	0.2	0.2	0.2

SOURCE: Calculated from data published by the National Center for Health Statistics.[16]

TABLE 3—Observed Gains in Life Expectancy (in years) at Selected Exact Ages by Race and Sex (United States; 1959/61 to 1978)

Ages	White Males (1)	White Females (2)	Nonwhite Males (3)	Nonwhite Females (4)
0	2.65	3.61	3.52	7.13
20	1.75	2.81	1.62	5.53
40	1.87	2.77	1.68	4.84
60	1.19	2.61	1.21	3.37
85	0.96	2.04	2.72	4.46

SOURCE: Calculated from data published by the National Center for Health Statistics.[17]

(equivalent to between the five- and seven-year delay assumption used in this study) exceed gains in life expectancy that would have occurred with the elimination of the number one cause of death in the United States, diseases of the heart (see Table 3). Thus, relatively small simultaneous delays in deaths attributable to several major degenerative diseases produce gains in life expectancy that are equal to, or greater than, the gains in life expectancy to be achieved by the elimination of some single major degenerative diseases.

Discussion

If numerous major degenerative diseases share common risk factors, then this study suggests that measures designed to lessen the impact of these risk factors would extend life to an extent comparable with the discovery of a cure for some degenerative diseases. Previous research has demonstrated a link between such risk factors and mortality.[18–22] At the same time, one must bear in mind that when mortality declines are a result of preventive health care measures, morbidity may also be compressed into later ages.[22] That is, morbidity may increase in the future at rates that are faster than has already been projected if we fail to consider the importance of how gains in life expectancy are achieved.[23,24]

Thus, as mortality continues to compress toward the biological limit to life in later ages, it is possible that we will reap diminishing returns from health care dollars earmarked for declining mortality and increased longevity. To address the issue posed by Keyfitz,[7] one may conclude that the extension of years-to-life *and* life-to-years is an appropriate goal for the allocation of research and service funds. These data indicate that preventive health care measures should be a prime target.

APPENDIX

The premise behind the original single cause-delay model developed by Manton[15] was that improved lifestyles and advances in medicine will delay the mortality curves for major degenerative diseases and therefore redistribute the risk of dying from a single major degenerative disease from younger to older ages. In later research, this model was extended to a projection tool for overall and cause-specific mortality by providing for the simultaneous delay of more than one degenerative disease at a time. This is called the *SI*multaneous *M*ultiple *CA*use *D*elay (SIMCAD) method of projecting mortality. The difference between this and the original single cause-delay model is that the SIMCAD model conforms with the assumption of disease dependency and the nature of the epidemiology of mass disease while the single cause-delay model does not.

With the SIMCAD model one argues that successive birth cohorts are likely to experience more favorable life conditions throughout their lifetimes than previous cohorts passing through the same ages, and this will delay the age progression of diseases and result in uniformly lower mortality rates. The major advantage of this new approach is that it provides a more realistic representation of the mortality and life expectancy benefits currently being achieved by medicine, better access to health care, and improving lifestyles. For example, medical intervention is thought to delay death from degenerative diseases by forcing the clinical manifestations of disease into later ages, by earlier detection of diseases, and by longer survival with disease in the body. Improved lifestyles are thought to delay death by slowing the rate at which degenerative diseases progress. This model is thus a method of indirectly estimating the effects of successful efforts to delay the onset of major degenerative diseases.

While the SIMCAD model relies heavily upon assumptions about which degenerative diseases are most likely to decline in the future, and the extent of such declines, it has performed well as a predictor of mortality change. It provides a more reliable methodological representation of the epidemiologic transition as it has occurred historically, and as it is likely to occur in the near future, than other methods currently in use. It should be noted that since the SIMCAD model deals exclusively with the effects of degenerative diseases on gains in life expectancy, it is assumed that there will be no drastic changes in patterns of death from non-degenerative causes in the coming decades.

ACKNOWLEDGMENTS

A portion of these results were presented at the meeting of the Population Association of America held in Minneapolis, Minnesota, May 1984, and the American Sociological Association meeting held in Washington, DC, August 1985. I wish to acknowledge the valuable contributions made to this study by Drs. Evelyn Kitagawa, Philip M. Hauser, Bernice Neugarten, Elizabeth Kutza, and William Brustein, and the Department of Human Genetics, University of Utah. This work was sponsored by NICHD training grant No. 5T32HD07153, Population Research Center, University of Chicago.

REFERENCES

1. Keyfitz N: Improving life expectancy: an uphill road ahead. Am J Public Health 1978; 68:954–956.
2. Keyfitz N: What difference would it make if cancer were eradicated? an examination of the Taeuber paradox. Demography 1977; 14:411–418.
3. Tsai SP, Lee ES, Hardy RJ: The effect of a reduction in leading causes of death: potential gains in life expectancy. Am J Public Health 1978; 68:966–971.
4. Schatzkin A: How long can we live? A more optimistic view of potential gains in life expectancy. Am J Public Health 1980; 70:1199–1200.
5. Wingard DL, Berkman LF, Brand RJ: A multivariate analysis of health-related practices. Am J Epidemiol 1982; 116:765–775.
6. Berkman LF, Wingard DL: The impact of health practices on mortality. *In:* Berkman LF, Breslow L (eds): Health and Ways of Living: Some Social and Behavioral Predictors of Mortality. New York: Oxford Press, 1983.
7. Keyfitz N: What direction of research. Am J Public Health 1980; 11:1201.
8. Fries JF: Aging, natural death, and the compression of morbidity. N Engl J Med 1980; 303:130–135.
9. Havighurst R, Sacher GA: Prospects for lengthening life and vigor. *In:* Neugarten B, Havighurst R (eds): Extending the Human Life Span: Social Policy and Social Ethics. Chicago: University of Chicago Press, 1977; 13–18.
10. Manton K: Changing concepts of morbidity and mortality in the elderly population. Milbank Mem Fund Q 1982; 60:183–244.
11. US Census Bureau: Projection of the population of the United States: 1982 to 2050. Curr Popul Rep 1982: Series P-25, No. 22. Washington, DC: Govt Printing Office, 1982.
12. Bayo F, Faber J: United States population projections for OASDI cost estimates, 1980. Office of the Actuary 1980: Actuarial Study No. 82, SSA Pub. No. 11-11529.
13. National Center for Health Statistics: Changing mortality patterns, health service utilization, and health care expenditures: United States, 1978–2003. Public Health Service Series 3, No. 23. Washington, DC: Govt Printing Office, 1983.
14. Crimmins E: The changing pattern of American mortality decline, 1940–77, and its implications for the future. Popul Dev Rev 1981; 7:229–254.
15. Manton K, Patrick C, Stallard E: Mortality model based on delays in progression of chronic diseases: An alternative to cause elimination model. Public Health Rep 1980; 95:580–588.
16. National Center for Health Statistics. United States Life Tables by Cause of Death: 1969–1971. DHEW Pub. No. (HRA) 75-1150, Volume 1, Number 5, pp. 5–60/5–67. Washington, DC: Govt Printing Office, 1975.
17. National Center for Health Statistics: Life Tables. Vital Statistics of the United States, 1978, Volume 2-Section 5, p. 5–15. DHHS Pub. No. (PHS) 81-1104. Washington, DC: Govt Printing Office, 1978.
18. Hamburg DA, Elliott GR, Parron DD (eds): Health and Behavior. Frontiers of Research in the Biobehavioral Sciences 1982. Washington, DC: Institute of Medicine, National Academy Press, 1982.
19. Breslow L, Enstrom JE: Persistence of health habits and their relationship to mortality. Prev Med 1980; 9:469–483.

20. Belloc NB: Relationship of health practices and mortality. Prev Med 1973; 2:67–81.
21. Branch LG, Jette AM: Personal health practices and mortality analysis among the elderly. Am J Public Health 1984; 1126–1129.
22. Wiley JA, Camacho TC: Life-style and future health: evidence from the Alameda County Study. Prev Med 1980; 9:1–21.
23. Greunberg EM: The failures of success. Milbank Mem Fund Q 1977; 55:3–24.
24. Kramer M: The rising pandemic of mental disorders and associated chronic diseases and disabilities. In Epidemiologic Research as a Basis for the Organization of Extramural Psychiatry. Acta Psychiatr Scand (Suppl) 1980; 285, 62:382–397.

International Symposium on Health and Safety in Agriculture
CALL FOR ABSTRACTS

This symposium will be held at the University of Saskatchewan, Saskatoo, Canada from October 9–11, 1985. Its purpose is to summarize current information related to health and safety in agriculture and to provide a framework to discuss future research and health protection in this area. Sessions will be devoted to discussion of epidemiology, pathophysiology, clinical aspects and health promotion related to health risks in agriculture and related industries.

Deadline for abstracts is August 15, 1985. Abstracts should be in English. Both original and previously published material will be considered.

For more information write to:

The International Symposium on Health and Safety in Agriculture
Section of Respiratory Medicine
Department of Medicine
University Hospital
Saskatoon, Saskatchewan, S7N 0X0.