Changing Concepts of Morbidity and Mortality in the Elderly Population

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THERE IS LITTLE DOUBT THAT THE AGING OF THE population of the United States is a demographic phenomenon that holds profound implications for both private and public American institutions. In order to best adapt those institutions to serve the aging of the population, it is necessary to understand the dynamics that underlie this phenomenon. One component of those population dynamics, mortality, is of particular importance since it has implications for both individual and institutional planning. At the individual level mortality determines the number of years of life a person can expect to live past a given age—an important factor in planning career, retirement, and investment goals. At the institutional level, mortality is important since it is the prime dynamic factor determining short-run changes in the size and age structure of the elderly population. Since health and social service requirements for individuals change dramatically and rapidly after the age of 65, accurate predictions of the changes is age distribution at advanced ages are especially important to social policy.

Another reason why it is important to understand mortality is that it involves consideration of basic human aging processes as well as the physiological basis of a wide range of diseases. Mortality therefore is biomedically complex and even a less well understood population

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dynamic than, for example, human fertility. In this paper, a review is made of current theories and their consistency with the current evidence. After this review we will present some possible alternative perspectives on human mortality and longevity and discuss both short- and long-run implications of various models of mortality.

Current Theories of Human Mortality and Longevity

A primary goal of a model of human mortality is to anticipate changes in mortality rates and human life expectancy. Many current models of human mortality predict that life expectancy in the U.S., with the present organization of medical science, is unlikely to increase much beyond present levels—a view that has strongly influenced forecasts of the rate of population aging and federal planning. Two mechanisms are proposed to explain this "ceiling" on life expectancy, one involving limitations on life span due to cellular processes of senescence and one involving an increased societal risk from chronic degenerative diseases. In the following we discuss basic principles of each type of model.

Biological Constraints on Human Mortality Changes

A number of theorists (Fries, 1980; Keyfitz, 1978; Hayflick, 1975) argue that mortality reductions and life expectancy increases in the U.S. population will cease in the near future because of biological constraints on the length of the human life span that are due to species specific processes of senescence. A prime implication of this perspective is that current efforts to increase life expectancy through disease control serve mainly to "rectangularize" the survival curve (Comfort, 1964). Thus, the curve describing the proportion of a cohort surviving to any given age will become nearly square with the surviving proportion remaining near 1.0 until the age range where mortality due to biological senescence occurs. Then the curve will drop rapidly to 0.0. Thus, life expectancy is increased by eliminating "premature death" due to specific diseases, so that large proportions of a cohort survive to their biologically determined life span to die a "natural death."

As Fries (1980:133) concludes, "The surprising fact is that we are already approaching the limits."

The argument that senescence will soon limit life expectancy change is based upon four types of evidence. *First,* historically, the maximum human life span has not been observed to change except in populations where age documentation is poor and the literacy rate is low (Fries, 1980; Hayflick, 1975). *Second,* the risk of death seems to increase as an exponential function of age with a doubling time of about eight years, so that the likelihood of observing persons at extreme ages is small (Fries, 1980; Sacher, 1977). *Third,* standard actuarial computations indicate that the elimination of cancer and heart disease would increase the average life expectancy, at most, by 20 years (Hayflick, 1975). *Finally,* there is experimental evidence to suggest that at least certain types of human cells are internally programmed for only a limited number of reproductions (Hayflick, 1965, 1975, 1977). The weight of this evidence leads to an important conclusion—that mortality is not necessarily linked to disease processes, or, in Fries's (1980:130) words:

> The bioscientific, medical model of diseases, our prevalent model, assumes that death is always the result of a disease process; if there were no disease, there would be no death. This view is hard to defend.

Therefore, once we are close to the elimination of disease-related, premature death, then the average life expectancy in the population is unlikely to change significantly unless we discover the key to altering the basic biological aging rate of human organisms—an accomplishment viewed as unlikely at least in the near future (Fries, 1980; Hayflick, 1975).

An important corollary to the model of biological constraints on population life expectancy is the implication it holds for the age distribution of chronic disease morbidity. Two basic principles of this model (i.e., that mortality and morbidity are not necessarily linked and that changing disease risks do not alter the underlying aging rate of the organism) give rise to distinct and contradictory views on future changes of chronic disease morbidity. It is useful to present and contrast these views.

A distinctly pessimistic perspective is presented by Kramer (1980)

and Gruenberg (1977; 1980:1304–1305), who suggest that chronic disease prevalence and disability will increase as life expectancy is increased. This conclusion is reached because increases in life expectancy are viewed as not being accomplished either by reducing the incidence or by retarding the rate of progression of chronic degenerative disease, but by controlling the lethal sequelae of those diseases (i.e., primarily early terminal infections such as pneumonia). Therefore, Gruenberg (1977:3) concludes that "the net effect of successful technical innovations used in disease control has been to raise the prevalence of certain diseases and disabilities by prolonging their average duration." This will lead to what Kramer (1980) has labeled as a "pandemic of mental disorders and chronic disease."

A more optimistic appraisal is offered by Fries (1980). He argues that, in analogy to the rectangularization of the survival curve, there can be a rectangularization of the age at onset of chronic degenerative diseases. Thus, although medical science is viewed as not being effective in increasing the human life span, hope is offered that personal participation in health maintenance can help "postpone chronic illness, to maintain vigor, and to slow social and psychological involution" (Fries, 1980:134). Consequently, while the growth of the elderly population will be limited by biological constraints on the human life span, the requirements for health and social services for the elderly can be reduced by eliminating or postponing chronic disease, so that smaller portions of the life span will be affected (Fries, 1980:130). Unfortunately, the optimism with which Fries's arguments have been accepted needs to be tempered due to a critical omission in Fries's arguments. Specifically, he does not indicate how society is to deal with the increased social and health service demands currently manifest, and emerging, between the present time and the time of occurrence of his "utopian" stage where chronic disease onset can be delayed till age 85 and beyond.

Despite the differences in the degree of optimism with which Gruenberg and Fries view the future, there are several important commonalities in their perspectives. Most important is their view that chronic illness and not mortality should be the prime focus of public health efforts. Each, however, has a somewhat different perspective on how we should proceed in this effort and the difficulty in achieving significant progress. Gruenberg views chronic diseases as distinct pathological states, and emphasizes the search in epidemiological studies

for preventable causes of chronic illness. Fries (1980:133) views chronic illness as a physiological process accelerating loss of organ reserve for which "postponement," rather than "cure," is likely to be effective. That is, for Fries a "cure" is achieved by delaying the age at which a disease reaches the symptomatic threshold beyond the age programmed for "natural death." Both Fries and Gruenberg suggest that alternatives to clinical treatment of chronic diseases need to be developed in the effort to control illness and disability; Gruenberg emphasizes the importance of epidemiology and prevention, while Fries emphasizes the role of geriatric medicine and personal responsibility for self care.

In part, the differences in optimism seem to be a function of what Fries and Gruenberg perceive to be our present state of knowledge concerning mechanisms for slowing chronic disease progression. For example, though one might agree that "personal autonomy" could be the "probable final common pathway to improved health" (Fries, 1980:134)—say by better nutrition, exercise, and reduced smoking—it is not clear from Fries's exposition how such personal responsibility is to be engendered in the population. For example, we know that in certain population groups such as the Mormons mortality rates are 30 percent below the nation as a whole—largely as a consequence of a religious and social ethic that emphasizes personal responsibility for health (Lew, 1980). Though the success of such groups in improving health is well known, fostering such a health ethic nationally is a difficult task. Fries gives few directions on how such goals are to be accomplished programmatically. Indeed, though Fries argues that we are near the elimination of premature death, he acknowledges that currently 80 percent of mortality, and a higher proportion of disability, are due to chronic illness. Thus, though he suggests that "present approaches to social intervention, promotion of health and personal autonomy" (Fries, 1980:135) may serve to compress morbidity and senescence, and points to recent declines in circulatory disease mortality as possible signs of such improvement, it must be recognized that current social conditions have led to the majority of our health problems being due to what Fries views as "preventable" causes. Given the difficulties in achieving reductions in such an apparent health hazard as smoking we would be inclined to agree with Gruenberg's and Kramer's more pessimistic outlook. Furthermore, Gruenberg points out that certain chronic conditions (e.g., Down's

syndrome) are genetically programmed. Consequently, there may be significant chronic morbidity that cannot be altered by personal choice. Such arguments reach a logical extreme in the thesis of P.R.J. Burch (1976) who argues that a major component of all chronic disease risks is genetically determined through individual variation in the immune system.

Fries and Gruenberg also seem to differ in their belief about future increases in life expectancy. Fries sees these as constrained by the biological processes of senescence. Gruenberg suggests an imbalance between life-saving technology and health-preserving technology so that continuing life expectancy changes presently serve to greatly increase the demand for health service. Thus, Gruenberg seems to argue for further life expectancy changes. However, it seems reasonable to assume that future improvements in life expectancy will be limited since the progression of chronic diseases is unaltered. Both views posit that chronic disease morbidity and at least certain components of mortality have no necessary connection.

Societal Constraints on Human Mortality Changes

A second theoretical position is that, historically, major declines in mortality have resulted from reductions in infectious disease risk due to improvements in lifestyle, hygiene, nutrition, and other public health factors—and not due to innovations in medical technology (Omran, 1971; McKeown, 1976; McKinlay and McKinlay, 1977). Omran proposed a model of epidemiological transition in which the correlation between the economic, demographic, and public health changes of a society were described as a series of stages, with the U.S. and other developed nations having reached an "end" stage, the "Age of Degenerative and Manmade Diseases." In this end stage, mortality slowly declines (to rates below 20/1000) and eventually approaches stability at relatively low levels, while life expectancy at birth increases slowly until it exceeds 50 years (Omran, 1971:517). At this stage, fertility "becomes the crucial factor in population growth" and heart disease, cancer, and other chronic diseases become the prominent public health hazards—with little said about the prospects for reducing chronic disease risks. Indeed, the nature of developed industrial societies is viewed by certain authors as having positive health risks for

chronic diseases due both to societal, public health factors—such as environmental deterioration or occupational stress—and to factors involving choice at the individual level, such as smoking (Dubos, 1965).

Models of societal determinants of mortality also have implications for aging changes in U.S. society because they imply that a societal health state has been reached in which major improvements in life expectancy in the near future are unlikely. In contrast to the view that life expectancy is biologically bounded, however, the potential for reductions in infectious disease and maternal mortality due to improvements in hygiene, nutrition, and sanitation is viewed as having been largely fulfilled, while societal factors relevant to chronic disease risks have recently shown either marginal improvements (e.g., smoking rates) or actual deterioration (e.g., environmental toxicological hazards). Thus, while there is nothing in the societal model to preclude the existence of a biologically determined limit, societal constraints on life expectancy are viewed as becoming operational before biological constraints. As in the biological model, medical science is argued to have little potential for increasing life expectancy through the treatment of chronic degenerative diseases. Consequently, it cannot serve to compensate for possible increases in societal risks. Often both perspectives are combined to suggest an even more limited potential for life expectancy change than could be projected under a pure biological model. Fries's optimism seems to result from his belief that societal constraints on both life expectancy increases and health improvement can be overcome.

A Critical Evaluation of Current Models of Human Mortality

In this section we evaluate the logical and empirical consistency of the models of human mortality described above.

The Implications of the Hayflick Limit for Human Mortality

A prime rationale for arguing for biological constraints on human life span was the observation made by Hayflick (1965, 1975) that repeated subculturing of human fibroblasts eventually produced cells incapable

of further division. This finding has been labeled the Hayflick "limit" (i.e., that cells from different animal species have the potential for only a fixed number of doublings—a number characteristic of the species). It also was found that the potential for the number of cell doublings was correlated with the longevity of species (i.e., short-lived species have the potential for fewer doublings).

Although Hayflick's findings have been used by others to argue that we have nearly reached the maximum obtainable average life expectancy in the U.S. (e.g., Fries, 1980:130), Hayflick is careful to emphasize that this clonal senescence phenomenon need not have a simple relationship to the aging of the organisms from which the cells were obtained (Strehler, 1977:40). There are at least three reasons for this caution. *First,* experimental evidence suggests that the regression coefficients of the age of a cell donor on the number of cell doublings achieved is only -0.20 (S.D. of 0.05; correlation of -0.50) (Martin et al., 1970). As the number of doublings projected for human fibroblasts is 40 to 60, and the regression coefficient suggests that it takes 100 years to exhaust 20 doublings, the evidence does not support the notion that the human life span will soon be limited by this mechanism. *Second,* there are many differences between the behavior of cells in a culture medium and in the human body. For example, manipulation of the density of cells in the culture medium allows the cells to achieve 20 additional doublings (Strehler, 1977:42). Therefore, differences between the cell medium and in the *in vivo* environment could lead to large differences in the life span that would be projected. *Third,* there are a number of types of cells in the human body (e.g., epidermis, lining of the intestines, cells in the circulatory system) whose ability to reproduce is not defined by the Hayflick limit (Strehler, 1977:42).

Beyond the lack of conclusive experimental results, arguments that life expectancy increases are constrained by cellular mechanisms of senescence are critized as being conceptually oversimplified by focusing upon only one aspect of the processes which govern mortality (Sacher, 1980). As Gordon et al. (1977:36) conclude: ". . . it is not surprising that various theorists have seized on particular subsets of the entire gamut of age related changes and have chosen to regard them as more fundamental, showing that these changes, in turn, lead to most, if not all, of the others."

If the theoretical and experimental results are not conclusive in

determining the age at which a Hayflick type of limit becomes operational in the human population, then the validation of the model must rest upon the current population evidence of mortality and actuarial analyses. Hayflick (1975), when discussing possible future gains due to the control of diseases, suggests that an increase of 20 years in average life expectancy might be achieved before biological limits on longevity become operational. Fries (1980:132) projects a maximum average life expectancy of between 82.4 and 85.6 years for the U.S. population and concludes: "clearly, the medical and social task of eliminating premature death is largely accomplished." This conclusion seems somewhat at odds with his observation that currently 80 percent of all deaths are due to chronic illness. In addition, recent empirical evidence—such as significant reductions in the mortality risks of specific chronic diseases (e.g., cardiovascular and cerebrovascular) at very advanced ages (e.g., 85+) and the observation that individual death at advanced age is typically characterized by a multiplicity of chronic degenerative diseases—calls into question the proposition that mortality at the extremes of the life span is due primarily to "natural death" resulting from cellular senescence.

A number of questions of analysis and interpretation arise in efforts to explain the inconsistencies of these theories and data. Despite the theoretical interest in human mortality, surprisingly little attention is paid to the manner in which mortality data are analyzed, the nature of the linkage between morbidity and mortality, and the substantive implications of the assumptions of those analyses. In the following sections we review both current mortality data and the conceptual basis of analyses of these data.

Current Evidence on Increases in Life Expectancy at Advanced Ages

Models of social and biological constraints on human longevity suggest that the U.S. is rapidly approaching the maximum obtainable average life expectancy. Perhaps the strongest evidence for these positions was the observation that, after rapid declines in mortality in the 1940s and 1950s, little change occurred in the early 1960s. This was taken as evidence that the potential for life expectancy had been largely exhausted. Some analysts concluded that "the death rate for the United States has reached the point where further decreases as experienced

in the past cannot be anticipated" (National Center for Health Statistics, 1964:42). After this conclusion was reached, however, mortality decline accelerated again in the late 1960s. Therefore it is important to examine the age patterns of recent mortality reductions to determine if they substantiate the hypothesized rectangularization of the survival curves and to provide insight into the potential for further life-expectancy increases. For example, if we are approaching either biological or social constraints on life expectancy, persons at advanced ages—and the population group with the greatest life expectancy, white females—ought to show the greatest effects of a life expectancy "ceiling." If significant mortality reductions continue at advanced ages, and for white females, this would indicate that we are not currently approaching a life expectancy ceiling and imply a potential for life expectancy increase (Lew, 1980:1365). We shall restrict our attention to current mortality data, since that is the type of data usually examined (e.g., Fries, 1980). Cohort data generally suggest greater life expectancy and survival potential than current data (Jacobson, 1964; Myers, 1977).

An examination of Table 1 shows no evidence of a ceiling on the life expectancy. For each time period, the life expectancy at age 65 for white females has increased more rapidly than that for white males. Furthermore, while the rate of increase for white females after 1930 has averaged a little better than 0.5 percent per year, the rate of increase in life expectancy was 1.3 percent per year over the first seven years of the 1970s—nearly twice the average annual change in previous decades. One might argue that these statistics reflect sex-linked differences in longevity. Even if such sex linkage exists, the fact that the rate of mortality over age 85 is rapidly decreasing for both males and females (National Center for Health Statistics, 1980) suggests that each sex group is not near its own particular biological limits.

A second question is whether the reduction in mortality rates for white females is less at extreme ages, i.e., ages near a "limit" on life expectancy. In Table 2 we present the changes in white female mortality rates for ten-year age groups for three different periods from 1950 to 1979.

Table 2 shows that, after 1960, decreases in mortality rates for white females above age 85 are greater than for those aged 55 to 65. If biological constraints were operational, we should necessarily be seeing absolute increases in mortality rates at extreme ages (e.g., Lew,

TABLE 1
Changes in Life Expectancy at Age 65 for U.S. White Males and Females

Period	White Males		White Females	
	Change in Life Expectancy in Years	Percent Change in Life Expectancy	Change in Life Expectancy in Years	Percent Change in Life Expectancy
1900–1902 to 1929–1931	0.3	2.6 (0.87)	0.6	4.9 (1.63)
1929–1931 to 1939–1941	0.3	2.5	0.8	6.3
1939–1941 to 1949–1951	0.7	5.7	1.4	10.3
1949–1951 to 1959–1961	0.2	1.6	0.9	6.0
1959–1961 to 1969–1971	0.1	0.8	1.0	6.3
1969–1971 to 1977	0.8	6.7 (9.6)	1.5	8.9 (12.7)

Note: Figures in parentheses indicate the percent change in life expectancy adjusted for the different length intervals.

TABLE 2
Death Rates per 1000 for Older White Females in the U.S., 1950–1979

Age	1950	1960	Average Annual Percent Change	1970	Average Annual Percent Change	1979	Average Annual Percent Change
55–64	12.9	10.8	−1.63	10.1	−0.65	8.8	−1.43
65–74	32.4	27.8	−1.42	24.7	−1.12	20.1	−2.07
75–84	84.8	77.0	−0.92	67.0	−1.30	56.6	−1.72
85+	196.8	194.8	−0.10	159.8	−1.80	133.7	−1.81

Source: Monthly Vital Statistics: Annual Summary for the U.S., 1979. Births, Deaths, Marriages, and Divorces. Vol. 28, No. 13, Table 6, p. 21, November 13, 1980.

1980:1365) past age 85. This evidence indicates that past-age-85 mortality rates are decreasing rapidly.

A third way to detect the effect of a ceiling on life expectancy is to examine changes in patterns of the age specific probabilities of survival over the period 1940 to 1978. If a ceiling were operational for white females, we ought to see a rectangularization of these survival curves.

Figure 1 shows that the life-table-survival curves for 1978 and 1939–1941 (National Center for Health Statistics, 1980) diverge past age 50. At age 50 there are 9 percent more white females surviving in 1978; at age 85 there are 22 percent more females surviving in 1978. Thus, the proportion surviving to the most advanced ages has increased nearly 2 1/2 times as fast as at younger ages. The divergence is most striking between 1960 and 1978. For example, at age 50 the difference in survival was 7.3 percent between 1940 and 1960 and 1.8 percent between 1960 and 1978—an indication of a slowdown in mortality reductions at age 50 due to the fact that survival in 1978 was already very high (i.e., 94.3 percent.) However, the survival increase at age 85 between 1940 and 1960 was 11.5 percent or 1.58 times the change at age 50. The change from 1960 to 1978 (a slightly shorter period) was 10.5 percent or 5.8 times the change at age 50. Thus, though we show signs of approaching an irreducible mortality rate at earlier ages, there are presently no such indications at age 85. This more rapid rate of survival improvement at advanced ages is not consistent with the presence of a biological upper bound on life expectancy, i.e., one close to the present life expectancy. Indeed, it

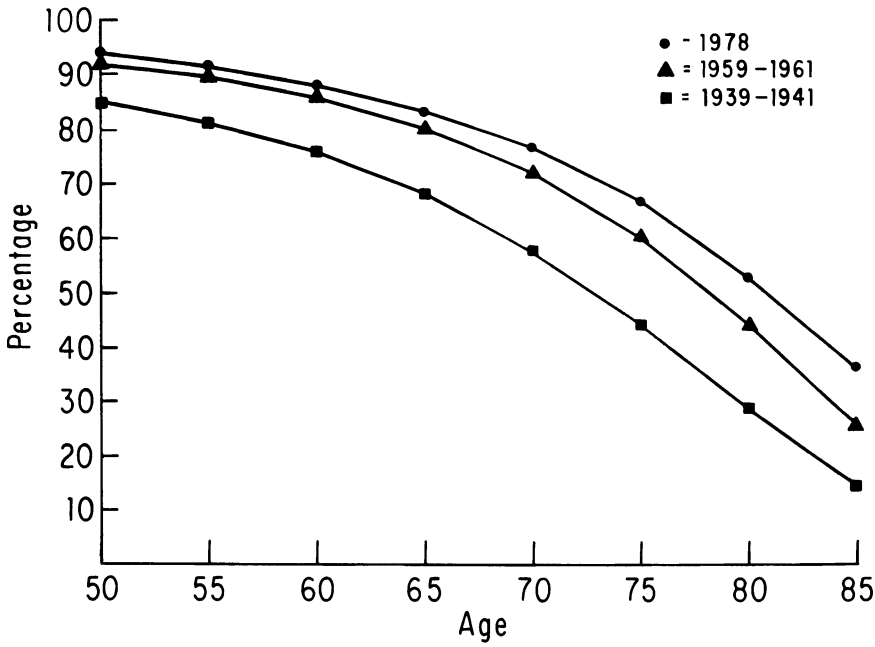

FIG. 1. Changes in the proportion of white females in the U.S. surviving for ages 50 to 85—1940 to 1978.

seems quite possible that real increases in life span are being manifest, and the terminal age of the survival distribution is increasing. To illustrate, the life expectancy for white females in the U.S. at age 85 increased only 0.56 years (from 4.10 to 4.66) from 1900 to 1960. However, from 1960 to 1978 life expectancy at age 85 has increased 2.04 years (from 4.66 to 6.7), or 44 percent. This implies that survivors to age 85 can expect to live, on average, to age 91.7 in 1978—up significantly from the expected terminal age of such individuals in 1960 of 89.67. Even more striking than the changes for white females are the changes in life expectancy for nonwhite females. From 1960 to 1978 nonwhite female life expectancy at age 85 increased from 5.4 to 9.9 years—an increase of 4.5 years or 82 percent. Though the quality of age reporting for nonwhite females at advanced ages might be questioned, this improvement has occurred during an era when data quality should have improved (due to social security enrollment), and persists even when adjustments are made for likely patterns of age misreporting. The improvement in life expectancy at advanced ages can be further demonstrated in Figure 2. In this figure

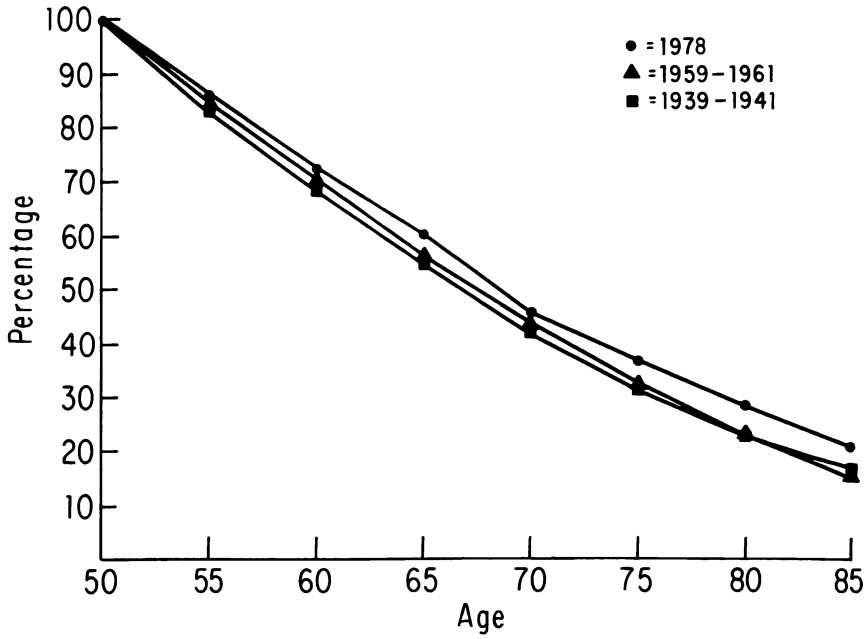

FIG. 2. Change in the proportion of the age 50 life expectancy remaining at subsequent ages: white females, 1940–1978.

we examine the proportion of the life expectancy at age 50 which remains at any given subsequent age. Thus, if the proportion at a given age increases from one time to the next, this implies that survival past age 50 had improved *relative* to the level of survival achieved at age 50 for the two time periods.

A comparison of the curves shows that the proportionate residual life expectancy increased markedly from 1939–1941 to 1978. Thus, *relatively* more of the life span is currently being preserved at advanced ages than in the past. This is again most evident in the period 1960 to 1978 where the proportionate increase at age 85 is 5.2 percent.

Another way to determine if rectangularization is occurring is to examine the variability of the age at death. If all diseases were eliminated, and persons died only of "natural death" at a species-specific biological limit, then one could expect less variance in the age at death (Fries, 1980). We examine this by computing the mean and variance of ages at death for white females in the U.S. past age 50 (to focus on chronic disease mortality) for 1960 and 1978 from standard U.S. life tables (National Center for Health Statistics, 1980). We found that while the average life expectancy past age 50 had

increased 9.3 percent, the variance of the age at death had increased 16.5 percent—77.4 percent faster than the mean. This too seems inconsistent with the hypothesized rectangularization of the survival curve.

From the age pattern and rate of current mortality declines, it seems reasonable to conclude that there is presently little evidence to substantiate the operation of biological or social constraints on life expectancy—especially at advanced ages. Furthermore, the increases in life expectancy at age 85 from 1960 to 1978 (e.g., 2.04 years for white females and 4.5 years for nonwhite females) are certainly more than "barely perceptible" as Fries (1980:131) has argued. Indeed, Hayflick (1975:11) holds that a life expectancy change of as little as five years could "rupture our present economic, medical and welfare institutions." Kramer (1980) similarly has argued that recent changes in life expectancy will greatly increase the demand for health services and that the future course of population aging will further increase those needs. Rice (1978) examined the prospects for continuing mortality reductions and determined that a continuation of the current rate of mortality reductions would lead to sharply higher expenditures for a broad variety of health services. Recent Social Security Administration (1981) forecasts demonstrate the implications of current mortality trends for the growth of the elderly population.

Of importance in all of these forecasts is the ability to *anticipate* future changes in mortality trends. In this regard, the relatively more rapid improvement of survival at advanced ages from 1960–1978 than from 1940–1960 is significant. This phenomenon is important because from 1940–1960 there were large reductions in infectious disease risk at early ages—reductions that had begun to be played out in the early 1960s—and which lead to the conclusion that there was little potential for future life-expectancy increases (National Center for Health Statistics, 1964). However, the changes between 1960 and 1978 suggest that mortality reductions began in a new age domain and were unanticipated because they were due to an entirely different set of health hazards and accomplished through different mechanisms.

The Age Trajectory of Human Mortality: The Population Rate of Aging

The recent decline in mortality risks at advanced ages and the possible increase in life span due to the control of chronic diseases lead us to

examine a second important question—associated changes in age-specific disability and morbidity in the population. This evidence will help us determine if improvements in life expectancy are due to control of acute complications of chronic diseases, as argued by Gruenberg (1977), or, perhaps, to actual retardation of the age rate of progression of chronic diseases.

One way to examine these changes is to study nationally representative data on morbidity and disability at various ages. Data on the civilian noninstitutionalized population are consistent in showing no evidence of increases in disability or morbidity over age 65 (Department of Health, Education, and Welfare, 1979). For example, between the Health Interview Surveys of 1972 and 1977, the percentage of persons reporting their health as fair or poor decreased from 31.0 to 29.9 while the proportion reporting no limitation in major activities increased slightly from 56.8 to 57.0 percent. The number of bed-disability days due to chronic conditions increased only slightly from 14.1 to 14.5 (persons 45 to 64 increased from 7.9 to 8.2 days), while the number of restricted-activities days remained at 36.5 for both 1972 and 1977. Neither the number of restricted-activities days nor the number of bed-disability days due to acute conditions showed much change over the period 1969 to 1977. An analysis of similar measures made over the 1960s produced the same conclusion, i.e., though more persons were surviving to advanced ages where chronic diseases should be more prevalent, little evidence existed to suggest that elderly persons of a given age were more disabled than in earlier decades (Riley and Foner, 1968:215).

The possibility exists that the reason why disability and morbidity have not increased in the noninstitutionalized population is that they greatly increase the risk of institutionalization. The data on the institutionalized population also suggest little deterioration in the average health state of the elderly population in the U.S. due to life-expectancy increases. For example, the age-specific rate of institutionalization might be viewed as a general measure of severe disability in the population. Thus, if increases in life expectancy had been achieved not by altering the severity of chronic conditions but by preventing death due to those conditions, these increases in life expectancy should be associated with an increased rate of institutionalization. The most recent data, from the 1973–1974 and 1977 National Nursing Home Surveys, show that the nursing home population (the predominant type of long-term care facility for the elderly) grew

17.1 percent from 1973–1974 to 1977 (Department of Health, Education, and Welfare, 1979:149). However, the over-65 population also increased significantly during this period (about 10 percent). As a consequence the rate of institutionalization increased by only 6 percent. Of even greater interest is the rate of institutionalization for the over-85 population—the age group for which the effects of life expectancy increases on disability are expected to be greatest. The rate of institutionalization for the over-85 group, which comprised 40 percent of the total nursing home population, has actually dropped 15 percent from 253.7 per 1,000 in 1973–1974 to 216.4 per 1,000 in 1977. Though such a major decline for this group could have many sources, the decline does not suggest that recent increases in life expectancy at advanced ages results in such severe disability that these persons find nursing home care mandatory.

The measures described above can be criticized since they reflect a number of subjective decisions about health care utilization and subjective evaluations of the level of disability associated with objective health states. Clearly, it would be preferable to use clinical indices of health states. However, clinical measures of population health from nationally representative samples of the elderly comparable over time are rare. Several are available from the Health and Nutrition Examination Survey (HANES) and Health Examination Survey (HES). The first measure, serum cholesterol above 260 mg/100ml, shows declines from 1960–1962 to 1971–1974 for all ages—including persons 65 to 74. Hypertension (systolic pressure above 160 mm Hg or diastolic of 95 mm Hg) shows an even more dramatic decrease. The percent of hypertensive individuals in the 65 to 74 population has dropped from 49.2 to 40.7 percent (Department of Health, Education, and Welfare, 1979). This contrasts with persons aged 25 to 64 where the proportion with hypertension has increased.

Thus, the available nationally representative data on disability, activity restriction, self-appraisal of health, and the current rate of long-term institutionalization at advanced ages and selected clinical measures show no marked deterioration in health status among persons aged 65 or more through much of the 1960s and 1970s despite significant increases in life expectancy at advanced ages. Therefore, if a pandemic of chronic diseases is occurring—a fact which might be debated—something must have been accomplished to reduce the severity and associated disability of these diseases at specific ages.

One should be careful to distinguish between the use of these rates

to suggest an overall improvement in health status at specific ages, and the use of the same data to forecast future absolute demands for health service. For example, the severity of chronic diseases could be reduced significantly and yet there still could be significant increases in the absolute demands for health services due to the simple growth of the elderly population (Kramer, 1980). Rice (1978:12) has projected that, under constant mortality assumptions, the number of nursing home residents could increase by 57 percent, from 1.3 million to 2.1 million, over the twenty-five-year period 1978 to 2003. If mortality rates continue to decline as they have from 1968 to 1978, this increase would be 112 percent, or to 2.8 million persons, if the mortality changes were accomplished with *no* compensating decrease in the age-specific rates of institutionalization. It seems likely, however, that significant mortality declines would not occur without some change in the age-specific institutionalization rate. The correlation of the two rates will be the result of a mortality and health status equilibrium to be discussed later in this paper. We believe that an understanding of mechanisms linking disease-severity reduction and mortality decreases would produce more precise forecasts of the absolute level of future health service demands.

It should be recognized, however, that national data on disability and health, though important in determining whether aggregate health status has changed, lack adequate age detail to examine possible mechanisms underlying health changes at more advanced ages (Liu et al., 1982). To do this, given that the disability data suggest no major deterioration in health status, we must return to the use of the mortality data. McKinlay and McKinlay (1977:410) suggest mortality data "are the only types of data which are readily accessible for the examination of time trends simply because comparable morbidity and disability data have not been available." However, exploiting mortality data to examine age changes in population health and aging rates involves the use of models and cause-specific measures of disease severity. In the following subsections we present several possible measures.

Age Change in Mortality Rates: An Indicator of Population Aging. Sacher (1980:13; 1977) argued that the age trajectory of mortality in a human population reflects the biological rate of aging for individuals. If the rate of biological aging is assumed to be correlated with functional declines and disability, the mortality data can be used to infer detailed

age changes in disability. In order to interpret what the age pattern of population mortality risks implies for the biological aging processes at the individual level, it is necessary to fit biologically motivated actuarial models to the mortality data. Fries (1980:131) concludes that the "best mathematical models relate the linear decline in organ function to the exponential mortality rate." An exponentially increasing mortality rate is the type of age trajectory of mortality risks associated with the well-known Gompertz function which has often been used in models of aging and mortality (see Strehler, 1977, chapter 5; Sacher, 1977).

There are two problems with the application of the Gompertz model. First, recent evidence on Medicare recipients shows that mortality rates at advanced ages (90 and over) are lower than the rates predicted from the Gompertz function. Indeed, the age trajectory of these rates becomes increasingly flatter with age until, at age 100, the mortality hazard is approximately constant (Wilkin, 1981). These observations were confirmed in a cohort of 50,000 persons selected for study by the American Cancer Society (Lew, 1980:1365). Thus, the observed age trajectory of mortality risks does not strictly militate against observing persons at advanced ages as argued by Fries (1980:131) in accepting the Gompertz model. Furthermore, it is difficult to resolve Fries's acceptance of the Gompertz function as a model of natural death with his assumptions that the "biologic distribution" described by mortality data is normal, i.e., the Gompertz hazard function will not produce a normal distribution of ages at death. This logical inconsistency is troubling since the assumption of a normal distribution was involved in Fries's forecast of a maximum life expectancy of about 85 years. However, an even more important problem, both logically and empirically, with the Gompertz model is that it can be used to infer the age trajectory of mortality risks for individuals from the age-specific mortality rates of a cohort *only* for homogeneous populations (e.g., Sacher, 1977:584). If there are individual differentials in mortality risks in human populations, as Fries argues, then a function describing the age trajectory of risks of a cohort does not describe the age trajectory of risks for any individual in the cohort (Vaupel et al., 1979), and by inference does not describe his loss of "vigor" (Fries, 1980:131). This divergence of the age-specific mortality risks in a population from the age trajectory of individual aging changes is due to systematic selection of "high" risk

persons by mortality. The cohort age-specific mortality rates describe only the *average* risk among survivors to a given age. We can examine the implication of heterogeneity for the use of models of human mortality as indicators of population-aging rates in two ways.

First, one can examine the relation of the age trajectory of mortality risks for individuals with that for cohorts under plausible (i.e., consistent with epidemiological studies) assumptions about individual variation in endowment for longevity. One possible model of longevity differentials is based on the assumption that individual differences in the endowment for longevity can be summarized by a single index, say z, and that the z_i's for individuals are fixed over age. With this asssumption, the following equation relates cohort mortality risks, $\bar{\mu}$, to individual mortality risks, $\bar{\mu}$ (Vaupel et al., 1979):

$$\bar{\mu} = \bar{z} \cdot \mu. \tag{1}$$

The symbol $\bar{z}$ represents the mean of the z_i's. This equation indicates that the age change of mortality risks for individuals with different values of z are proportional to one another. Since the value of $\bar{z}$ decreases with age as individuals with high z values die at earlier ages, the form of the age trajectory of mortality risks (and any age specific functions of these risks such as life expectancy) for cohorts is different than that for individuals. This can be made explicit by observing that $\bar{z}$ may be replaced by $(\bar{s})^{1/k}$, the proportion surviving to a given age raised to the $1/k$ power, where a) z_i is assumed to be gamma distributed, and b) k is the gamma shape parameter (where the squared coefficient of variation of z_i is equal to $1/k$). For example, if we assume that the endowment for longevity, z_i, is exponentially distributed, then k will be equal to 1.0. This means that for an *individual* with the average endowment for longevity, the risk of dying is twice the observed cohort force of mortality at the point at which 50 percent of the cohorts had died. That is, from equation (1), $\bar{\mu} = (0.5)^1 \cdot \mu$ so that $\mu/\bar{\mu} = 2.0$. Thus, if the z_i's are exponentially distributed, individuals with the average endowment for longevity (i.e., $\bar{z}$) will die out of the population relatively rapidly, and by the time 50 percent of the cohort has died, the average cohort risk will be far below the risk for a person with the average initial value of z_i. In Figure 3, we compare the age trajectory of the probability of death (Prob $= 1 - e^{-\mu}$) for persons with average mortality risks ($z = 1.0$; circles and triangles) with the cohort probability of death

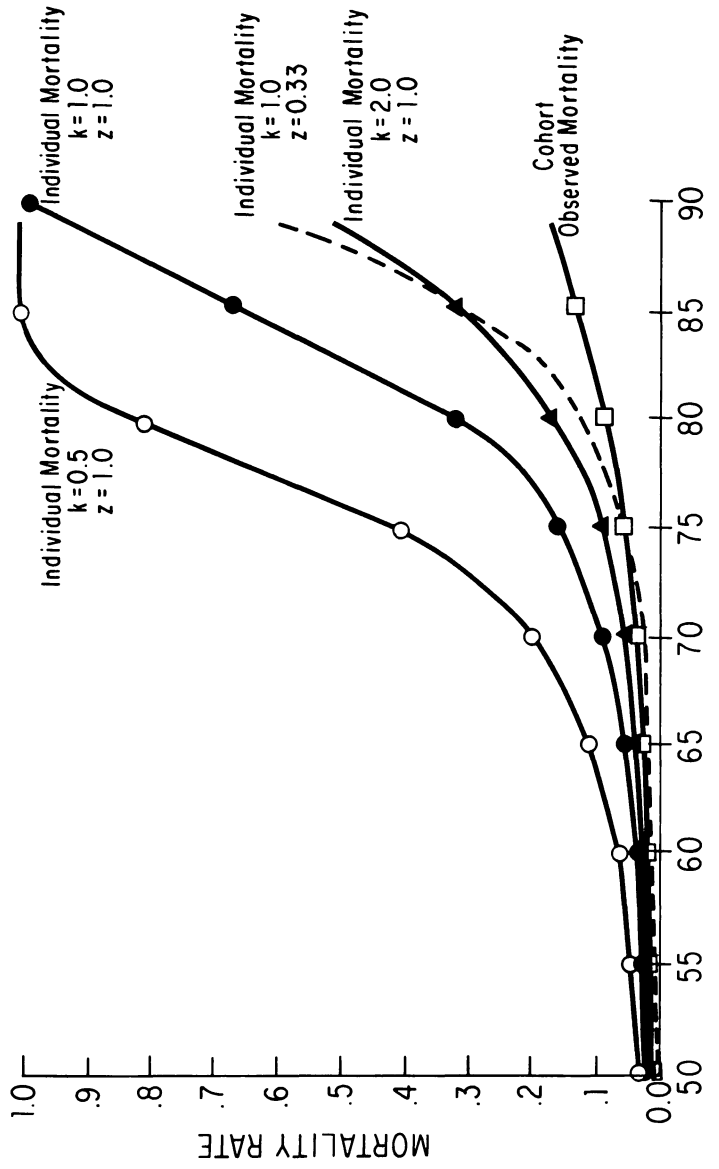

FIG. 3. Observed and individual age-specific mortality rates after adjustment for population heterogeneity, white females in the U.S., 1875 birth cohort.

(squares) under different assumptions (i.e., different values of k) about the magnitude of heterogeneity of individual risks.

In Figure 3 we see that a) the age increase in the probability of death for individuals has a much sharper bend than for cohorts, and b) the degree of divergence between the individual and cohort curves is positively correlated with the initial heterogeneity of the cohort, i.e., the divergence increases as k decreases (since k and the variance of individual risk levels are inversely related). Note that three of the individual curves in Figure 2 are for persons with the average endowment for longevity scaled, for convenience, so that $\bar{z} = 1.0$. A comparison of the two curves with $z = 1.0$ and 0.33 for $k = 1.0$ shows how the age specific mortality probabilities for persons from the same distribution, but with different levels of longevity endowment, vary. Although the magnitude (i.e., values of k) of individual heterogeneity in a cohort might be debated, the direction of the divergence between cohort and individual risks is a product of the existence of any degree of heterogeneity (Vaupel et al., 1979) and results regardless of the shape of the distribution of individual risks (Manton and Stallard, 1981). Thus, if individuals have different endowments for longevity, the cohort age-specific probabilities of death will increase less rapidly with age than will the age-specific risk of death for any individual. This is simply a result of the fact that the age-specific probability of death for a cohort represents the *average* risk for all persons who survive to a specific age. Mortality will cause the survivors to a given subsequent age to have a lower average risk due to the systematic early selection by mortality of high risk individuals.

The importance of examining the hypothetical age trajectory of risk for individuals rather than the cohort age trajectory of risks is that the individual curves imply stronger homeostatic forces at younger ages. That is, since the age curve of mortality risks rises more rapidly for individuals, the age at death for an individual is more precisely determined than is suggested by the cohort survival curves. This implies that individual mortality risks remain low until reaching a narrow age "window" and then, upon entering the window, physiological homeostasis deteriorates rapidly so that mortality risks for the individual rise very rapidly. The fact that the age loss of function occurs more rapidly and occupies a shorter time prior to death for individuals than indicated by cohort or cross-sectional data suggests

that at younger ages the individual is more resistant to environmental stress. As a consequence, more of the life span is spent in a physiological state where restorative forces can maintain function in the face of the loss of physiological reserve than in the Gompertz model. This suggests that the loss of function for individuals is not strictly linear with age—a finding consistent with longitudinal data on function loss (Upton, 1977) and with the observation of a "terminal drop" in cognitive functioning in longitudinal data (Riegel, 1971). Linear declines tend to be observed in cross-sectional data, possibly due to the effects of systematic "selection" (e.g., Shock, 1960).

If the shape of the individual age trajectory of mortality risks is preserved under life span extension, then productive life span will be extended as age-specific mortality is reduced instead of the morbid period prior to death. At least one theorist suggests that "it will take about as long to go through a terminal illness at age 150 as it does at 65 today. The real effect will be to increase the total number of years an individual spends in a healthful, and hopefully productive, state" (Strehler, 1975:6). The implication of such a mechanism is that age-specific mortality reductions will be positively correlated with reductions in disease severity and disability at corresponding ages. Therefore, as mortality rates decline at a given age, there would be some compensating decline in the rate of utilization of certain health services (e.g., nursing home care) before that age. In fact, such models might be used as the basis for improving projections of health service utilization by providing estimates of the likely change in health-service-utilization rates associated with a given mortality reduction.

These and other observations (e.g., Baltes and Schaie, 1974) reflect much new evidence on the nature of aging changes in individuals. The weight of this evidence indicates that not only is the rate of aging highly variable among individuals, but that our stereotype of elderly persons seriously underestimates their ability to maintain functional capacity at older ages. This is consistent with disability data which show no current increases in disability associated with life expectancy increase at advanced ages and with the rates of institutionalization over age 85.

A second way to examine the effects of heterogeneity is to investigate differences in cause-specific mortality risks. We can empirically assess the effects of heterogeneity on the mortality risks of specific diseases by calculating modified-life-table functions which do not assume per-

sons are at equal risk to all causes of death. That is, one examines the change in the survival distribution realized by theoretically eliminating the disease from those persons who actually died from it. The modified-life-table functions can then be compared with the standard-life-table functions used by Hayflick (1975) which are based on the assumption that life extension due to the elimination of a given disease will be averaged over all persons. The two life expectancy measures are related (Manton et al., 1980b) as:

$$\text{(life expectancy change in target group)} = \frac{\text{(average life expectancy change)}}{\text{(proportion of population in target group)}} \quad (2)$$

In Table 3, we present estimates of the change in life expectancy due to the elimination of chronic respiratory disease among white males in 1977.

The first column contains estimates of age-specific life expectancy for white males in the U.S. in 1977. The second contains the age-specific life expectancy for white males in the U.S. who died of chronic respiratory disease in 1977. Note that, at birth, the life expectancy for white males dying of chronic respiratory diseases is over 3½ years greater than the life expectancy of the general population because to die of this disease, one must survive other diseases. By age 50, the life expectancy of the general population surpasses that of persons dying of chronic respiratory diseases. The third column of Table 3 contains the average life-expectancy gain due to the elimination of chronic respiratory diseases calculated in the standard way (Chiang, 1968). Column 4 shows that the gain in life expectancies among persons who die of the disease is over 25 times greater than the life expectancy gain in the third column. The life expectancy for persons cured of chronic respiratory diseases is the sum of the corresponding age-specific quantities in columns 2 and 4. Thus, the life expectancy at birth of white males in the U.S. who died of the disease in 1977 could be increased by 10.43 years, to 84.09 years, by eliminating the disease. This gives quite a different perception of the effects on life expectancy of eliminating chronic respiratory diseases than we get from examining the standard measures (column 3). The standard measure confounds the survival distribution, either observed or modified by cause elimination, of individuals afflicted with the disease

TABLE 3
Observed and Hypothetical Cause Elimination Age-Specific Life Expectancy for White Males in the U.S. in 1977

Age	(1) Observed Life Expectancy	(2) Life Expectancy for Those Dying of Chronic Respiratory Diseases	(3) Age Specific Average Gain in Life Expectancy Due to Total Elimination of Chronic Respiratory Disease	(4) Age Specific Gain in Life Expectancy of Chronic Respiratory Disease for Those Who Died of the Disease
0–4	69.97	73.66	0.43	10.43
5–9	66.18	68.81	0.43	10.31
10–14	61.31	63.82	0.43	10.30
15–19	56.43	58.84	0.43	10.28
20–24	51.82	53.87	0.43	10.26
25–29	47.30	48.89	0.44	10.24
30–34	42.67	43.91	0.44	10.23
35–39	38.01	38.95	0.44	10.20
40–44	33.40	34.00	0.44	10.17
45–49	28.93	29.12	0.45	10.09
50–54	24.69	24.35	0.45	9.94
55–59	20.73	19.81	0.45	9.66
60–64	17.09	15.67	0.43	9.18
65–69	13.89	12.07	0.41	8.47
70–74	11.04	9.11	0.35	7.54
75–79	8.62	6.81	0.28	6.50
80–84	6.74	5.19	0.20	5.40
85–89	5.24	4.08	0.12	4.39
90–94	4.04	3.33	0.07	3.56
95–99	3.25	3.57	0.04	3.02
100+	3.22	3.09	0.02	2.29

with the survival distribution of persons dying of all other causes of death.

New Models of Cause-Specific Mortality: Age-Specific Measures of Disease Severity. Any model of human mortality used to analyze population aging must accommodate concepts of cause-specific mortality that portray the increasing importance of chronic degenerative diseases in causing death. Unfortunately, existing conceptualizations of cause-specific mortality were originally developed to represent the primary mortality risks of a society with vastly different social, economic, and

health conditions than those which exist in contemporary society in the U.S. These models were designed to portray mortality risks in a population with a relatively *young* age structure, a low prevalence of chronic degenerative diseases, and a situation in which infectious disease and causes of infant and maternal mortality were the primary health risks. Under such societal conditions, it was believed that the medical conditions determining the age at death could be described by the single medical condition that precipitated the sequence of morbid events which led to death. This type of model therefore is referred to as the "underlying-cause-of-death" model. The use of this model has continued because it simplifies the tabulation of mortality statistics and can be analyzed by simple statistical methods.

The underlying-cause-of-death model may be insufficient to describe contemporary U.S. mortality patterns. With increases in the prevalence of chronic degenerative conditions, mortality at advanced ages is frequently a product not only of immediately lethal events, but also of chronic degenerative conditions. The failure of underlying-cause mortality statistics to discriminate between deaths with and without contributory chronic conditions is a handicap in describing the health state of the national population through mortality statistics, especially the health state of the elderly.

As a consequence, it is necessary to develop new mortality data and concepts. Fortunately, the basic mortality data collection instrument, the U.S. standard death certificate, provides the basis for collecting such information since it permits the reporting of causes of death which are associated in various ways with the underlying cause. This potential is often utilized, for, in 1969, 5.2 million conditions were reported for 1.9 million deaths—an average of 2.7 conditions per death certificate. This additional information, much of it concerning associated chronic conditions, can yield insight on the age-specific association of mortality reduction and morbidity. For example, "multiple-cause" mortality data might be used to test arguments made by Kramer and Gruenberg that life-expectancy increases are due to the elimination of the mortality consequences of diseases associated with chronic illness, e.g., pneumonia. In the multiple-cause data we find that in 1969, 60,000 death certificates listed pneumonia as an underlying cause whereas 150,000 additional death certificates listed pneumonia as an associated cause of death. These 150,000 deaths presumably could have been "delayed" by the elimination of pneu-

monia under Kramer and Gruenberg's argument. Thus, by analyzing deaths where pneumonia is an associated condition one could determine the potential for increasing life expectancy by delaying such deaths. These data will also be useful in assessing arguments that mortality at very advanced ages will increasingly be due to "general failure without specific disease" (Strehler, 1975:6). For example, Strehler argues that life expectancy will change markedly over the next 35 years and suggests that such changes will result from an enhanced capacity to alter biological aging rates. If the frequency of chronic diseases continues to increase at very advanced ages, as life expectancy increases, this would suggest that life-expectancy increase was due to a reduction in the rate of progression of chronic disease processes, thereby increasing their duration and prevalence, and not due to retardation of a generalized aging process.

Multiple-cause mortality data have been coded in the U.S. by the National Center for Health Statistics since 1968 and are the source for national underlying-cause mortality statistics. With such data we can, at specific ages, examine the change over time in the role of a chronic degenerative disease from an underlying cause of death to an associated cause of death. This shift could imply a reduction in the severity of the chronic disease process when associated with a decreasing risk as an underlying cause of death and correlated with a greater age at death. One way to measure this shift is to calculate the ratio of the number of death certificates on which the chronic disease was recorded to the number of death certificates on which that disease was the underlying cause of death (Wing and Manton, 1981). These ratios could then be compared on an age-specific basis to the risk of dying from the condition as an underlying cause of death. A reduction in severity could be indicated if the underlying-cause-of-death risk of the disease decreased over time and the nonunderlying-cause-of-death reporting of the condition increased. In Table 4 we present these ratios and the underlying rates for selected major causes of death for the years 1968 and 1977 for white males in the U.S.

In Table 4 we see that diabetes, pneumonia, ischemic heart disease, and stroke show declines in their rate of occurrence as underlying causes of death over the ten-year period. The third column shows that proportionate declines in these rates are, in general, quite large. Only the underlying-cause-of-death mortality rates for cancer and chronic respiratory disease over 65 show increases. The rates for 1968 are the

TABLE 4
Changes in Age-Specific Underlying Cause and Total Mention Rates for White Males in the U.S., 1968–1977, Rates Per 100,000

	Underlying Cause (UC)			Total Mention (TM)			TM/UC		"Severity" Index
Age	1968	1977	1977/1968	1968	1977	1977/1968	1968	1977	1977/1968
				Pneumonia and Influenza					
<65	15.0	7.7	0.52	46.4	31.7	0.68	3.10	4.10	1.32
65–74	149.8	93.9	0.63	597.0	436.7	0.73	3.99	4.65	1.17
75–84	443.6	347.9	0.78	1583.6	1285.6	0.81	3.57	3.70	1.04
85+	1343.8	1029.4	0.77	3980.0	3156.8	0.79	2.96	3.07	1.04
				Cancer					
<65	81.2	80.4	0.99	87.4	86.7	0.99	1.08	1.08	1.00
65–74	988.0	1058.2	1.07	1137.2	1211.9	1.07	1.15	1.15	1.00
75–84	1545.6	1732.5	1.12	1943.0	2156.2	1.11	1.25	1.24	0.99
85+	1853.7	2107.7	1.14	2611.7	2972.1	1.14	1.41	1.41	1.00

				Diabetes					
<65	6.1	4.3	0.71	19.1	16.1	0.84	3.15	3.72	1.18
65–74	85.1	63.3	0.74	341.5	297.9	0.87	4.01	4.70	1.17
75–84	168.4	124.8	0.74	664.1	597.5	0.90	3.94	4.79	1.21
85+	242.0	197.5	0.82	896.5	876.7	0.98	3.71	4.44	1.20
				Ischemic Heart Disease					
<65	147.8	110.8	0.75	162.8	123.1	0.76	1.10	1.11	1.01
65–74	2118.1	1659.8	0.78	2465.1	1960.2	0.80	1.16	1.18	1.01
75–84	4472.1	3612.1	0.81	5308.4	4410.9	0.83	1.19	1.22	1.03
85+	8962.8	7410.0	0.83	10525.8	8985.9	0.85	1.17	1.21	1.03
				Stroke					
<65	19.3	12.6	0.66	32.3	22.4	0.69	1.68	1.77	1.06
65–74	448.5	294.6	0.66	723.1	512.8	0.71	1.61	1.74	1.08
75–84	1431.4	997.7	0.70	2226.2	1664.9	0.75	1.56	1.67	1.07
85+	3452.5	2417.4	0.70	5001.5	3828.8	0.77	1.45	1.58	1.09
				Chronic Respiratory Disease					
<65	11.6	9.0	0.78	24.3	21.7	0.89	2.11	2.41	1.14
65–74	234.8	237.5	1.01	531.5	566.3	1.07	2.26	2.38	1.05
75–84	365.4	470.7	1.29	935.2	1168.4	1.25	2.56	2.48	0.97
85+	362.5	529.7	1.46	1101.1	1501.0	1.36	3.04	2.83	0.93

standard cause-specific mortality rates reported in U.S. vital statistics; the rates for 1977 are age standardized to the 1968 population. The next three columns contain mortality rates based on the total number of occurrences of the condition on the death certificate. We see, by comparing the sixth column with the third column, that the pattern of increases and decreases for the underlying-cause mentions of a disease parallel the increases and decreases in the rate of total mentions of the condition. Thus, the population mortality risks from these conditions associated in any way with death are also, in general, declining. It may be, however, that the amount of increase or decrease in the total mention rate is more or less than the underlying-cause mention rate. The relative rates of increase or decrease are reported in the last column of the table and reflect the relative increase or decrease of the significance of the condition, at specific ages, as a factor associated with death. An examination of these ratios suggests a decrease in severity of the chronic illnesses—a decrease occurring at all ages. There are only three instances where the underlying-cause rate of mention has fallen less rapidly (yielding an index less than 1.0) than the total mention rate, i.e., for cancer at ages 75 to 84 and for chronic respiratory disease above age 75. This same fact can be examined in the cross-temporal changes of the ratio of total mentions to underlying-cause mentions (TM/UC) of the disease on the death certificate.

This measure does not directly reflect the severity of the chronic disease in the population since, obviously, such an assessment requires knowledge of the actual incidence and prevalence of the disease in the population for the desired dates. For example, if the incidence of these diseases had radically dropped over the period, and prevalence subsequently decined, the rate at which these diseases are mentioned on the death certificate as a function of total prevalence may have increased, thus implying increased severity. However, if it is reasonable to presume that incidence had not declined markedly (i.e., less than the decline of the total mention rate), then the decrease in the proportion of deaths with a given disease as the underlying cause would imply a reduction in severity. The fact that these mortality ratios are obtained from national vital statistics data, meaning that they are available on an annual basis for the entire population, makes the ratio a useful device for monitoring the national population for signs of changes in chronic illness severity.

In Table 5 we present the same set of rates and ratios for nonwhite males as was provided for white males in Table 4.

A comparison of Tables 4 and 5 shows that, though the magnitude of the underlying-cause-of-death rates varies between white and nonwhite males, the age and disease-specific patterns of increases and decreases are similar. For example, for both male groups, the underlying-cause rates for pneumonia, diabetes, ischemic heart disease, and stroke have decreased at all ages. For both male groups, the chronic respiratory disease underlying-cause rates increased after age 65 with a strong positive gradient with age in that increase. For cancers, the increases at all ages are greater for nonwhite males. The absolute levels of the nonwhite rates for chronic respiratory diseases and ischemic heart disease, however, are lower at all ages. For the other four diseases we note an interesting pattern where nonwhites have higher rates at earlier ages and lower rates at advanced ages, though this cause-specific "crossover" tends to occur at later ages in 1977 than in 1968 (Manton, 1982).

In examining the total mention rates we begin with the expectation, because of their lower life expectancy, that the ratio of total to underlying-cause mentions ought to be lower for nonwhite males reflecting a greater severity of these conditions at younger ages. One might raise an objection to such an interpretation on the basis that this differential in the rate of reporting of associated conditions might be due to such factors as inadequate prior medical care and medical records for nonwhites, carelessness and lack of interest on the part of the physician in completing the death certificate, etc. However, if such measurement factors are operational, it is clear that they must operate differentially across chronic diseases. For example, for both diabetes and pneumonia, the ratio of total to underlying-cause mentions is much larger for white males. However, the ratio for chronic respiratory and ischemic heart disease are quite similar. It is also interesting to note that, even for diabetes and pneumonia, diseases where the ratio of total to underlying-cause mentions is discrepant, the relative rate of change of total to underlying-cause mentions is comparable. Indeed the cause and age-specific pattern of this index is in general agreement between white and nonwhite males. Thus, it seems that explanations of the age-specific differences between demographic groups in the role of chronic diseases as causes of death might be useful in evaluating race and sex differences in health status

TABLE 5
Changes in Age-Specific Underlying Cause and Total Mention Rates for Non-White Males in the U.S., 1968–1977, Rates Per 100,000

	Underlying Cause (UC)			Total Mention (TM)			TM/UC		"Severity" Index
Age	1968	1977	1977/1968	1968	1977	1977/1968	1968	1977	1977/1968
				Pneumonia and Influenza					
<65	40.5	18.6	0.46	87.2	56.6	0.65	2.15	3.05	1.42
65–74	248.3	152.5	0.61	800.9	570.2	0.71	3.23	3.74	1.16
75–84	485.4	392.8	0.81	1415.6	1250.3	0.88	2.92	3.18	1.09
85+	989.4	735.8	0.74	2481.9	1974.2	0.80	2.51	2.68	1.07
				Cancer					
<65	81.1	87.3	1.08	87.0	93.5	1.07	1.08	1.07	0.99
65–74	1162.5	1289.3	1.11	1311.9	1439.5	1.10	1.11	1.10	0.99
75–84	1433.1	1883.3	1.31	1702.4	2229.2	1.31	1.19	1.18	1.00
85+	1425.7	1648.7	1.16	1804.8	2096.5	1.16	1.27	1.27	1.00
				Diabetes					
<65	8.9	6.6	0.75	21.1	20.5	0.97	2.38	3.09	1.30
65–74	126.1	101.4	0.80	353.5	339.0	0.96	2.80	3.35	1.19
75–84	159.1	158.2	0.99	470.6	518.1	1.10	2.96	3.27	1.11
85+	183.6	155.0	0.84	541.2	567.0	1.05	2.95	3.66	1.24

				Ischemic Heart Disease					
<65	119.1	91.1	0.76	134.4	103.2	0.77	1.13	1.13	1.00
65–74	2044.8	1510.1	0.74	2395.7	1788.8	0.75	1.17	1.18	1.01
75–84	3243.6	2868.7	0.88	3851.3	3476.1	0.90	1.19	1.21	1.02
85+	5824.4	4118.7	0.71	6737.6	4979.6	0.74	1.16	1.21	1.05
				Stroke					
<65	43.5	25.8	0.59	66.5	42.0	0.63	1.53	1.63	1.06
65–74	869.4	513.6	0.59	1328.3	833.3	0.63	1.53	1.62	1.06
75–84	1464.4	1125.1	0.77	2250.9	1763.8	0.78	1.54	1.57	1.02
85+	2503.3	1583.6	0.63	3623.9	2452.2	0.68	1.45	1.55	1.07
				Chronic Respiratory Disease					
<65	10.3	7.6	0.74	20.0	17.6	0.88	1.93	2.31	1.20
65–74	131.2	137.6	1.05	315.0	330.5	1.05	2.40	2.40	1.00
75–84	193.6	239.5	1.24	451.4	610.2	1.35	2.33	2.55	1.09
85+	164.5	252.6	1.54	507.8	702.4	1.38	3.09	2.78	0.90

along a number of dimensions, such as utilization of medical services, implications of general health status factors (e.g., nutrition and stress) for susceptibility to chronic disease, differential risk factor distributions, and possibly differential biological rates of aging (Manton, 1980). Clearly, the population data offer a unique opportunity for monitoring the change of health-status differentials over age. This would be especially important in identifying critical age ranges where health status is undergoing rapid changes. It would appear highly cost-effective to use the population data to identify these critical ages before the investment in detailed epidemiological studies is made.

Multiple-cause data offer some useful insights into the health changes underlying the current mortality reductions. First, they suggest that Kramer (1980) and Gruenberg (1977) are correct in arguing that population aging implies a much greater prevalence of chronic disease. This seems inconsistent with Fries's (1980) arguments that persons at advanced ages are more likely to die of "natural death" within presently observed life spans, and that elimination of premature death involves the "postponement" of chronic disease symptoms until after the age of natural death. This observation is also inconsistent with Strehler's (1975) proposition that current life-expectancy changes might be due to unanticipated progress in altering the basic aging rate. The data suggest, however, that Fries may be correct in arguing that medical science can reduce the severity of such conditions at a given age by slowing the rate of progression of those disorders. Persons with chronic diseases survive to advanced ages, then die of other causes of death with the chronic disease operating in a contributory role. This would seem to be inconsistent with Kramer and Gruenberg, who argued that mortality reductions were accomplished, not through slowing the progression of a chronic disease, but by eliminating its lethal sequelae. It may be, as Kramer and Gruenberg argue, that the elimination of lethal sequelae leave a health-compromised group at a much elevated *risk* of serious disability and other forms of morbidity. Formally, such phenomena might be characterized as a type of "dependent" competing risk (Manton and Stallard, 1980). Though one can certainly cite examples of such phenomena, it is also clear that health-promoting behavior (e.g., smoking cessation and hypertension control) can have an impact on the risks of a broad range of diseases so that control of one disease or risk factor can reduce the risk of many others (Schatzkin, 1980). It seems clear that a basic challenge

to epidemiologic research is to go beyond the study of simple disease/risk factor associations and develop integrated approaches which study the effects of prevention and therapy on multiple diseases (World Health Organization, 1980). It suggests, along with our review of disability data, evidence on longitudinal aging changes and analysis of the age trajectory of mortality risks, that mortality reductions extend the productive life span of individuals not by eliminating chronic disease, but by reducing its severity at any given age.

Cause-Elimination Models and Forecasts of Life-Expectancy Change

Many of the arguments holding that control of chronic degenerative disease will have little impact on life expectancy are derived from cause-elimination life tables. Although useful first approximations of the health impact of a given disease, these procedures are probably not appropriate for making forecasts of future life-expectancy change since they involve a series of assumptions not likely to be physically realized. For example, they assume that all persons have equal susceptibility to all causes of death—a finding inconsistent with epidemiological evidence.

A second important assumption is that individuals who are observed to have died of a given disease at a given age not only are assumed not to have died of the disease, but also that they are not subjected to any risk from the disease after that age. The obvious problem with such an assumption is that chronic diseases are often "controlled" (e.g., Fries, 1980:133) but not eliminated, so that the person will remain at risk of the disease. In fact, the risk might continue to increase with age, but more slowly, so that the age at which the disease reaches a lethal stage is delayed. In this case, for a given chronic disease delayed m years, the age-specific probability of death at ages x to $x + 1$, denoted q_x, decreases to the value $q_x^{(m)}$ where:

$$q_x^{(m)} = 1 - (1 - q_{x-m})^{d_x - m,c/d_x - m} \cdot (1 - q_x)^{(d_x - d_{x,c})/d_x}. \qquad (3)$$

In equation (3), d_x and $d_{x,c}$ denote the age-specific total and cause-specific number of deaths, respectively, at ages x to $x + 1$; d_{x-m} and $d_{x-m,c}$ are corresponding quantities at ages $(x - m)$ to $(x - m + 1)$. The cause-delay life table calculations differ from standard life table calculations in that q_x is replaced by $q_x^{(m)}$ (Manton et al., 1980a).

The cause-delay calculations avoid a serious logical difficulty apparent in the cause-elimination computations. That is, if control of a major risk factor like smoking has a discernible effect on all major causes of death, one obviously cannot forecast life-expectancy changes by simpler cause elimination since this would imply an infinite life span. In order to carry out cause-elimination calculations where all major causes of death are eliminated, the analyst has to define an underlying base mortality rate. This obviously entails critical assumptions. For example, Strehler (1975:7) obtains an estimate of 15 years for the maximum life-expectancy increase obtainable by eliminating diseases by defining the death rate due to accidents as a "measure of general homeostatic capacity." The assumption that accidents define a baseline mortality rate, presumably insensitive to environmental factors, is clearly tenuous. Schatzkin (1980) has pointed out that similar situations arise in forecasting the mortality implications of changes in major chronic-disease risk factors such as smoking, hypertension, and nutrition that affect a broad range of diseases.

A third important assumption made in standard cause-elimination calculations is that each death is caused by *one* of a set of independently operating diseases. Clearly, deaths, especially at advanced ages, are often due to multiple, possibly interacting, diseases. Thus, reducing stroke mortality may affect life expectancy in two ways: a) by delaying deaths due to stroke as the primary cause of death, and b) by reducing the mortality risks of stroke as a contributory cause of death where another disease was the primary cause.

In order to examine the implications of the three assumptions of standard cause-elimination calculations described above, we have produced in Table 6 measures of life-expectancy changes at three ages (birth, ages 65, and 75), a) under standard cause elimination assumptions; b) for underlying versus multiple-cause occurrence of disease; c) for the population affected by the disease; and d) under different assumptions about the degree to which the progression of the disease can be delayed.

Table 6 yields a variety of insights. First, a large portion of the potential mortality risk of chronic disease is in nonunderlying-cause occurrences. For example, the difference between eliminating diabetes as an underlying cause of death at birth and eliminating its total effect on mortality (i.e., all deaths where it was mentioned on a death certificate) is 0.47 years in the total population (0.64–0.17, or the

TABLE 6

Alternate Estimates of Life-Expectancy Changes (in Years) Due to the Elimination or Delay of Diabetes or Stroke for White Males in the U.S. in 1969

	Diabetes				Stroke				Stroke/Diabetes			
	Underlying Cause		Total Mention		Underlying Cause		Total Mention		Without Diabetes		With Diabetes	
Years Delay	1	2	3	4	5	6	7	8	9	10	11	12
					At Birth							
1	0.01	0.96	0.06	0.97	0.09	0.96	0.15	0.96	0.13	0.96	0.01	0.96
5	0.06	4.17	0.24	4.17	0.38	3.98	0.62	4.08	0.56	4.05	0.05	4.02
10	0.10	6.96	0.40	6.98	0.61	6.37	1.00	6.65	0.92	6.58	0.08	6.47
15	0.13	8.77	0.50	8.71	0.74	7.75	1.24	8.19	1.13	8.09	0.10	7.85
Eliminated	0.17	11.75	0.64	11.10	0.91	9.49	1.57	10.39	1.43	10.26	0.11	9.27
					At Age 65 Years							
1	0.01	0.55	0.04	0.56	0.09	0.73	0.14	0.73	0.13	0.73	0.01	0.66
5	0.04	2.60	0.17	2.61	0.38	3.13	0.61	3.18	0.56	3.17	0.05	3.02
10	0.07	4.50	0.30	4.65	0.63	5.09	1.02	5.29	0.93	5.24	0.08	5.09
15	0.09	5.81	0.39	6.08	0.77	6.23	1.27	6.58	1.15	6.48	0.10	6.36
Eliminated	0.13	7.92	0.53	8.24	0.93	7.61	1.58	8.22	1.42	8.05	0.12	7.70
					At Age 75 Years							
1	0.01	0.37	0.02	0.31	0.08	0.54	0.12	0.52	0.11	0.53	0.01	0.36
5	0.03	1.67	0.09	1.48	0.34	2.33	0.52	2.32	0.49	2.36	0.03	1.69
10	0.04	2.93	0.17	2.83	0.57	3.94	0.90	4.05	0.84	4.06	0.05	3.28
15	0.06	4.04	0.24	4.04	0.71	4.94	1.15	5.19	1.07	5.16	0.07	4.49
Eliminated	0.09	5.98	0.37	6.17	0.89	6.18	1.49	6.69	1.36	6.58	0.09	5.97

Columns 1,3,5,7,9,11 = life-expectancy change averaged over total population
Columns 2,4,6,8,10,12 = life-expectancy change averaged only over persons dying of selected conditions

difference between columns 1 and 3). Furthermore, in the multiple-cause data, we can identify the underlying causes of death with which the chronic disease was associated. Thus, a large part of the non-underlying-cause effect of diabetes can be attributed to the role of diabetes as a contributing factor in stroke-related deaths. This contribution is about a quarter (0.11 of 0.47 years) of the potential change in life expectancy due to the elimination of all nonunderlying-cause mentions of diabetes. Second, the "cause-delay" model indicates the change in survival due to the hypothetical "delay" of deaths due to a chronic disease. At younger ages, a large portion of the total delay can be realized whereas at advanced ages, competing causes of death prevent much of the delay from being manifest. The delay model permits the analyst to assess the implications of changes in the rate of progression of a broad range of diseases. Third, by examining the effects of disease-delay only among persons affected by the diseases we can identify the potential of intervention in chronic disease processes on individual survival. Finally, for all the methods a large proportion of the effects of chronic diseases on life expectancy persists to advanced ages (e.g., age 75.)

Thus, we see that standard cause-elimination calculations represent only one aspect of the mortality implications of controlling a chronic disease. Controlling a chronic disease among persons susceptible to death from the disease results in large increases in life expectancy for them at advanced ages. This suggests that standard calculations may seriously underestimate the impact on mortality risks of controlling chronic diseases on individual survival and thus, the potential for life-expectancy increases. Elimination of the indirect effects of a disease as a contributory cause of death suggests that there is a variety of ways mortality can be reduced. Furthermore, under the cause-delay calculations, it is possible to posit mortality reductions for a broad range of diseases. These computations could not be carried out using standard cause-elimination strategies. The net implication of these alternate models of mortality reduction is that the estimate of a change in life expectancy of 20 years provided by Hayflick (or 15 years by Strehler, 1975) is a result of a specific and biologically unrealistic model of the relation of disease to mortality. More realistic estimates would necessarily focus upon specific diseases and population groups and, for deaths at advanced ages, would take into account the likely multiplicity of chronic conditions affecting individuals.

New Mechanisms in Human Mortality Changes

Much of the data in support of both social and biological constraints on human life-expectancy change is historical. Unfortunately, historical patterns may be a poor basis from which to forecast future human mortality changes because prior changes in human life expectancy have been the result of decreases in the risk of infectious diseases and maternal and infant mortality, much of which has already been eliminated (Omran, 1977). Significant medical progress against major chronic disease has been a very recent phenomenon.

Progress has been most notable in the reduction of circulatory disease mortality and might be attributed to both lifestyle changes and innovations in the clinical treatment of circulatory diseases. For example, lifestyle changes, such as increases in action-oriented leisure time, reduction of dietary cholesterol, and reduction in smoking, may have reduced the rate of progression of circulatory disease in the population. However, it would be difficult to argue that all of the reduction in circulatory disease mortality is due to lifestyle changes because it is unclear whether all the race, age, and sex groups with significant declines in circulatory disease mortality have equally experienced these changes (Crimmins, 1981). Consequently, the reduction may partly be attributable to clinical innovations in the management of chronic circulatory disease risk. The fact that the proportion of persons aged 65–74 with hypertension has not increased demonstrates progress in medically controlling the condition in the population (Department of Health, Education, and Welfare, 1979) as does the dramatic reduction in cerebrovascular mortality. Other more recent innovations are improvements in diagnostic procedures (e.g., angiograms, ultrasound imaging, nuclear scanning, positron emission tomography) which help to identify specific, latent circulatory defects. Such diagnosed circulatory defects can then be clinically treated by such innovative procedures as balloon angioplasty, clot dissolving streptokinase, bypass and vascular surgery, implanting of defibrillators, medical treatment of atherosclerosis, and calcium blockers. It is possible that these new treatment modalities can resolve the circulatory defect before it becomes manifest in a potentially lethal, acute, circulatory event. Thus, death from a localized, life-threatening situation may be averted without significant residual disability.

A second important class of medical innovations involves the management of an acute, life-threatening circulatory event once that event has occurred. These innovations involve educational programs to teach the general public about cardiopulmonary resuscitation, the establishment of special medical units to bring critical, emergency care in the very first hours of an acute circulatory event, and intensive care afterwards, and the development of new surgical and medical treatments for the management of myocardial infarction. Development of such specialized care has led to important insights in the treatment and nature of myocardial infarcts. The discovery, for instance, that cardiac arrhythmia is often the primary cause of death, instead of destruction of cardiac tissue, suggests that successful management of potentially lethal arrythmias may allow the person to live a significant number of additional years with little or no residual disability.

Although recent developments in the management of circulatory diseases are impressive, there are also signs of progress in the treatment of specific cancers like childhood leukemia and Hodgkin's disease (Axtell et al., 1976). Though total cancer mortality has been increasing (a phenomenon partly due to the reduction of circulatory disease mortality), there have been a number of developments in the very early detection of tumors through chemical markers and advances in chemotherapy. For example, the use of chemical reservoirs and mechanical pumps to increase the local therapeutic dosage to liver tumors promises to increase the survival time of such patients by months and perhaps years. Long-term survival of terminal cancer patients (those whose disease can be managed but not cured) has increased despite the lack of significant progress against cancer mortality. One sign of progress is the serious consideration of the actuarial risks of cancer patients for insurance purposes (Raven, 1970; Ringertz, 1970).

Though these innovations in medical science are significant, in order to have an effect on the nation's health it is necessary that the medical care system be organized to provide these innovations on a mass basis. Thus, a possible prime factor in reductions of circulatory disease mortality has been the aggressiveness of federal programs to upgrade medical manpower and facilities and to provide extensive low- or no-cost medical care to the poor and elderly (Department of Health, Education, and Welfare, 1979; Rogers and Blendon, 1977). Such programs have already had a demonstrable impact on the nation's

health, but considerable potential for futher improvement exists both by more complete diffusion of proven medical treatments (e.g., a large proportion of hypertension remains untreated) and in further medical innovations. Not inconsequential in the potential for improvement is the long-term care experience gained through the application of biomedical approaches to controlling chronic disease. For example, much has been learned about long-term management of dialysis patients and medical treatment of hypertension to greatly increase the efficacy of treatment. On these and other grounds, there is reason to project further future increases in life expectancy at very advanced ages due to the control of specific chronic diseases.

An Alternate View of Human Mortality and Population Aging

In the previous sections we have reviewed current evidence on mortality conditions in the population of the U.S. and found that no single model adequately explained all of the relevant data. Consequently, such models must be modified before we can expect to accurately forecast changes in life expectancy, the growth of the elderly population (especially at advanced ages), and changes in the health status of the elderly population.

Changes to models of human mortality can be based on several simple principles:

1. The human organism is a complex multicomponent system, each component having its own aging rate. The death of the individual organism therefore will be determined by the fastest aging rate of the individual components.
2. The failures of components can be identified with major chronic degenerative diseases. One of the factors in chronic degenerative diseases is the effect of cellular senescence on the physiological capacity of a given component or major organ system.
3. The propensity for failure of individual component systems is only partly dependent on the propensity for failure of other components. This suggests that if an effective intervention can be made to delay the failure of a given component, death of the organism can be delayed. Since the diseases are partially interdependent, reduction

in the rate of failure of one component may also help to retard the rate of failure of another component (Schatzkin, 1980).

4. Mortality is a property of individuals, yet data on the risk of death are always based on the behavior of populations. Hence, analyses of mortality must link the biological mechanisms of individual aging and mortality with the measures of population risk.

These four principles simply formalize what we view as a common sense recognition that human aging and mortality are complex phenomena. To be explained, they must be viewed as dynamic multidimensional processes in which chronic degenerative diseases play an essential role. Such a model can be easily derived by the examination of surveys of the biological literature on aging processes in complex biological systems such as man (e.g., Strehler, 1977; Finch and Hayflick, 1977).

Although these four principles seem reasonable, they run counter to much of the current literature on mortality and aging models. For example, biological models (e.g., Fries, 1980; Strehler, 1975) seem to suggest that the nature of mortality will simplify as life expectancy increases and we approach hypothesized biological constraints on life span. This "simplification" results because chronic degenerative diseases are viewed as irrelevent to improving survival at advanced ages where, it is argued, death is primarily determined by a unidimensional process of cellular senescence. Theorists who developed models that viewed aging and mortality as multidimensional (e.g., Sacher, 1980; Sacher and Trucco, 1962) frequently analyzed empirical data on human mortality as if mortality were unidimensional (e.g., Sacher, 1977) and not related to specific disease processes. Even when aging and mortality were viewed as primarily determined by multiple chronic degenerative disease processes, there were few predictions of mortality reductions due to the control of these diseases (e.g., Gruenberg, 1977). When mortality reductions are forecast, they are attributed to unanticipated reductions in the aging rate rather than control of individual chronic disease processes (Strehler, 1975).

One might reasonably ask why there is such a disjuncture between the current evidence on mortality and disability and current models. One reason was the relatively static mortality conditions in the 1960s that strongly influenced the development of the current models of mortality. A second was historical evidence on the relative importance

of public health factors versus clinical innovations as mechanisms for reducing mortality. Although seldom discussed, a further reason was that the disease definitions and concepts that had been developed for acute diseases did not accurately portray the "natural history" of a chronic disease process or adequately distinguish between catastrophic events and the disease process underlying the risk of those events.

To illustrate, acute myocardial infarction is an acute circulatory catastrophe which is generally the result of underlying chronic degenerative circulatory disease. If the underlying disease is identified as generalized atherosclerosis, we are faced with the fact that atherogenesis is concomitant with "aging" and nearly universally prevalent in white males past the age of 30. If atherogenesis were completely controlled, we would see significant major increases in population life expectancy, for elimination of atherogenesis would imply significant delay, if not elimination, of a broad range of chronic diseases. This effect would be both direct, by decreasing the age-specific risks of stroke and coronary heart disease (as well as renal and other diseases) and indirect, by increasing the circulatory vitality of the organism and thereby increasing its ability to withstand the assaults of cancer and other noncirculatory diseases. Direct effects could be viewed as altering the aging of a given component and indirect effects as changes in the ability of the organism to maintain physiological homeostasis due to component interdependence.

Associated with the need to update disease concepts to reflect aging processes is the need to generalize the concept of prevention in a parallel manner. For example, Fielding (1978) identified three types of prevention on the basis of the nature of the intervention upon which the prevention was based. It seems clear that the concept of prevention has to be specialized to reflect the stage at which an intervention was made in a chronic disease process. The lack of a consistent terminology for generalizing the concept of prevention for chronic disease processes has generated considerable confusion both in assessing the role of clinical science in controlling chronic diseases and in forecasting the effects of such intervention.

Although mortality is an event that occurs in the individual organism, the development of an adequate model of human mortality must take into account the difficulties in differentiating between changes in individual mortality behavior and aggregate mortality patterns—the level at which data are generally analyzed. Because of

the failure to recognize these problems, analyses of aggregate mortality risks often are not consistent with the assumptions of the theories of individual mortality which are being tested. Since the ultimate test of these models is their ability to forecast human mortality, this is a critical deficiency.

Therefore, to successfully investigate a particular model of human mortality, one must have actuarial techniques which are consistent with its biological foundation. Such approaches, identifiable as "bioactuarial" techniques, should have several properties. First, they must consistently link individual health changes with aggregate measures of those changes by adjusting for mortality selection, cohort differentials in incidence rate, differentials in the rate of disease progression, and temporal changes in case-fatality rates (Manton and Stallard, 1982b). Second, they must link mortality with the morbidity history of individuals either by combining mortality and morbidity data or by using a theoretical model of the biological linkage of disease and death (e.g., Manton and Stallard, 1982a). This will allow forecasts about health changes, as inferred from mortality changes, to be made with the model and will offer an opportunity for validation of the behavior of the model from other data sources.

To understand the relation of morbidity and mortality as life expectancy increases, let us introduce the concept of a "dynamic equilibrium." This concept involves a modification of the basic epidemiological relation that prevalence is a function of incidence and duration. This basic relation suggests that if incidence is unchanged, then mortality reductions leading to life-expectancy increases can only occur by increasing the duration of the disease and, consequently, its prevalence. This phenomenon is a necessary consequence of the basic mathematical relation. However, for chronic diseases, it seems reasonable to ask substantive questions about the way in which duration is increased. One way to increase duration is to eliminate lethal sequelae, e.g., pneumonia (Gruenberg, 1977), and not to affect the basic rate of progression of the disease process. Another way to increase duration is to change the rate of progression of the primary disease process. In this latter case, duration, and hence life expectancy, is increased by reducing disease severity. For example, recalling our argument that individual homeostasis is stronger than implied by the cohort survival curve, life span extension may primarily increase productive life span with the period spent in a highly morbid state being

relatively constant. Maintenance of homeostasis may occur through clinical efforts (e.g., hypertension control) as well as health promoting behavior. Thus, though prevalence might increase, the average severity of the disease is decreased. Clearly, the duration of some diseases, and hence life expectancy, is increased by eliminating lethal sequelae. For example, Gruenberg (1977) explains how such a situation arises in Down's syndrome where improvements in survival have proceeded more rapidly than in the general population (Gruenberg, 1981). Other genetically determined diseases might also follow this mechanism. However, it seems that the bulk of recent life-expectancy changes has been effected through the second mechanism whereby the severity and rate of progression of the primary disease have been reduced. For example, diabetes mellitus and hypertension are two chronic pathological states of individuals which can be fairly well managed. Without treatment, mortality risks could rise; average duration, and hence prevalence, would fall. With treatment, though prevalence increases at the expense of health resources, the quality of life gained is often sufficiently good to justify the efforts. This position does not argue against the merits of the primary prevention of chronic diseases (i.e., incidence reduction) but rather suggests that positive societal benefits can accrue to disease management efforts.

The concept of equilibrium is consistent with many elements of Fries's and Kramer's views of the nature of chronic-disease risk in elderly populations. For example, as Fries argues, the concept of equilibrium indicates that much can be done to delay and minimize the effects of chronic disease. Furthermore, the concept of equilibrium is consistent with the operation of chronic diseases as manifestations of basic aging changes whose total prevalence will continue to increase due to the aging of human organisms. The concept of equilibrium, however, implies that the severity and rate of progression of chronic disease are directly related to mortality changes so that, correlated with mortality reduction, there is a corresponding reduction in the rate of progression of the "aging" of the vital organ systems of the body. Thus, we require our model of human mortality to have morbidity and mortality in equilibrium and to be dynamic and multidimensional.

In both Fries and Kramer, the concept of equilibrium is apparently rejected by arguing that chronic-disease mortality and morbidity were partly independent. In Kramer (1980) and Gruenberg (1977), life

expectancy increased even though chronic-disease prevalence *and* severity increased. In Fries (1980) the concept of equilibrium seemed to operate for chronic illness and "premature death"; however, it did not seem to apply to ultimate "natural death." Thus, the biological limit on life expectancy had nearly been reached even though a considerable prevalence of chronic degenerative disease remains. Given that we could find little current evidence of the operation of "natural death" even at the most advanced ages, it seemed parsimonious to argue that equilibrium presently applies at all ages. Our position is similar to that of Strehler (1975:6) that "there is no way to increase longevity in a hospitable environment except by improving the health state at all ages."

Equilibrium is an important demographic concept which has serious policy implications because mortality reductions are directly linked with the long-term management of chronic diseases—a component of primary health services that is likely to increase greatly. The concept of equilibrium suggests linkages in two directions. Health care costs will be incurred in reducing the severity of the chronic disease, or slowing its rate of progression, in order to reduce mortality risks. In return, it is expected that these costs will accelerate as life expectancy (and disease prevalence) increase. However, since life-span extensions are produced by reductions in the rate of progression of the disease, productive life span will also increase and *potentially* lead to greater economic productivity. Increases in health care costs will thus have to be balanced against greater economic productivity. Thus, equilibrium leads us naturally to a dynamic "cost/benefit" ratio.

Actually, such ratios are implicit in many actuarial analyses of health care expenditures in the calculation of direct (e.g., health care) and indirect (e.g., lost wages) cost of disease (Rice and Hodgson, 1981). However, the present formulas for calculating indirect costs of disease (i.e., lost wages and productivity due to premature death) minimize the economic importance of the elderly since indirect costs are calculated on the basis of current labor force and retirement patterns which suggest little economic productivity for persons over 65. It is unclear to what degree the lack of economic productivity implied in such analyses is a result of physical limitations or a function of institutional limitations on the labor-force participation of the elderly. If measures of the economic *potential* of the elderly were based on *physical* limitations, an entirely different cost/benefit picture could

result for chronic-disease control and life-expectancy increase. This conceptualization of equilibrium in aging, health status change, health care expenditure, labor-force participation of the elderly and life expectancy will be central in the following discussions.

Select Implications of Models of Human Mortality

In the preceding sections we have examined the theoretical and empirical bases of current models of human mortality and found them inadequate to explain recent mortality trends in the U.S. In this evaluation we identified elements of an alternative model which might better explain and forecast mortality changes. In this section, we will examine the implications of these alternative models for forecasting the rate and magnitude of population aging, the direction of biomedical research, and individual and institutional adaptations to population aging.

Forecasting the Growth of the Elderly Population

Some population theorists have argued that the rate and magnitude of population aging is largely determined by differential birth cohort size so that differences between theories of mortality have few implications for the quantitative aspects of population aging. However, an examination of recent efforts to forecast the growth of the elderly population shows that assumptions about mortality have had serious implications for both current estimates of the size of the elderly population and projections of its rate of increase.

For example, in 1953 the Census Bureau projected that there would be 20.7 million persons over age 65 in 1975. The estimated figure in 1975 was 22.4 million or nearly 8 percent more than the 1953 forecast. In December 1972, the over-65 population was projected to be 24.1 million in 1980. In fact, 25.7 million have been enumerated in the 1980 census. This is an error of 6.6 percent (1.6 million persons) over just eight years. Since all of the people over age 65 in 1980 were alive and had been enumerated in 1970, this discrepancy was due to the inability to anticipate the decrease of mortality rates.

Forecasts made as recently as 1978 (e.g., Siegel, 1978:17) were based on arguments that life expectancy will increase little at advanced ages. These estimates suggested that by the year 2000 there would be 31.9 million persons over age 65 in contrast to an estimate of 35.7 million based on current mortality trends (Crimmins, 1981).

Recently, Social Security actuaries have produced new forecasts of the over-65 population using mortality declines observed over the period 1968 to 1978 and a set of judgmental "ultimate annual percentage improvements in central death rates by sex and cause of death" (Social Security Administration, 1981:6). These "ultimate annual percentage improvements" are based on a number of assumptions about future mortality trends, and vary considerably from current trends. In general the ultimate annual percentage improvements are much less than currently observed trends of reduction (exception: cancer and residual causes for females). The observed annual improvements from 1968 to 1978 were used to determine improvements from 1981 to 2005 by gradually transforming the observed declines to the assumed ultimate annual improvements. After 2005 (to 2080) the purely judgmental rates are used. Projections were produced under three alternatives—alternative II representing the mortality declines of the mixed observed and judgmental estimates of decline, and alternatives I and III representing improvements averaging half and twice the alternative II improvements.

One can see that the different assumptions produce a difference in the forecast population size of 14 percent in 2000 and over 33 percent in 2040. In Table 7 we see the life expectancies at birth and age 65 that would be expected under the three scenarios. The female life expectancies at birth forecast under assumption III exceed Fries's estimates of the maximum obtainable life expectancy by the year 2020. The life expectancies at age 65 for females indicate that by 2000, nearly 20 years of life can be expected under the worst assumptions. These figures document the quantitative importance of mortality conditions for the future growth of the elderly population and the qualitative implication that extensions of current mortality conditions will soon bring us to and perhaps beyond Fries's projected limits. The most favorable mortality assumptions suggest about a 74 percent increase in life expectancy at age 65 by 2080 to levels (a mean of 97.6 years) that imply a significant lengthening of the human life span.

It should be emphasized that the primary difference between the

TABLE 7
Population Growth and Life-Expectancy Change from New Social Security Forecasts

A. Population Projections Under Three Mortality Assumptions*

Year	I Slowed Reduction Alternative	II Observed Reduction Alternative	III Accelerated Reduction Alternative
2000	34,651	36,251	39,409
2020	48,767	52,653	60,755
2040	60,211	67,527	82,119
2060	63,166	70,327	88,048
2080	73,043	75,934	87,832

B. Projected Life Expectancy

	e_0			e_{65}		
	I	II	III	I	II	III
			Male Life Expectancy			
1980	69.8	69.8	69.8	14.3	14.3	14.3
2000	71.4	72.9	75.9	15.0	15.8	17.4
2020	71.8	73.8	77.7	15.3	16.4	18.8
2040	72.2	74.6	79.4	15.6	17.0	20.1
2060	72.6	75.4	81.0	15.9	17.6	21.5
2080	73.0	76.1	82.6	16.1	18.2	22.8
			Female Life Expectancy			
1980	77.7	77.7	77.7	18.7	18.7	18.7
2000	79.4	81.1	84.9	19.8	21.1	24.2
2020	79.9	82.1	87.2	20.2	22.0	26.1
2040	80.3	83.1	89.5	20.6	22.8	28.2
2060	80.8	84.1	91.8	21.0	23.6	30.4
2080	81.2	85.0	94.2	21.3	24.4	32.6

* Figures denote thousands
Source: Social Security Administration. Office of the Actuary. 1981. *Social Security Area Population Projections, 1981.* Actuarial Study No. 85. SSA Pub. No. 11-11532, Table 18, p. 42.

recent Social Security forecasts, which show large increases in the elderly population, and earlier forecasts of much lower rates of growth is the period over which the rate of mortality reductions is estimated. The period 1968 to 1978 employed in the recent forecasts is an era with significant mortality reductions in contrast to the earlier periods used in estimating changes in mortality. The tremendous effect on the forecast population size of the period over which mortality trends

were evaluated might easily lead one to question the legitimacy of historical extrapolation. Use of such historical data can be defended on two grounds. First, it can be argued that one is using the most recent and reliable data (Rice, 1978). Second, one could defend the exercise on the basis that one is *projecting* the implications of current mortality trends for the growth of the elderly population. Projection must be carefully distinguished from actual forecasting in that only in forecasting does one actually predict the size of the population at a specified future date. It is clear, from the rapid changes in the "forecasts" of the size of the elderly population, which are due to the recent rapid change in mortality trends, that such simple extrapolation of historical trends and synthetic assumptions will not be sufficient for forecasting. Rather, biologically and epidemiologically motivated models of mortality trends must be developed so that changes in trends, such as we have recently experienced, can be anticipated. For example, we have shown that if individuals differ in their endowment for longevity, the proportion surviving to a given age will be correlated with the age-specific probability of death after that age. Thus, after a period of rapid mortality reductions, the proportion surviving to a given age will increase. In this larger group of survivors, as argued by Kramer and Gruenberg, there may be significant numbers of morbid and disabled persons. The consequence may be that the age-specific probabilities of death will increase because of the difficulty of keeping "impaired" persons alive at even more advanced ages. Thus, if forecasts were conducted using a heterogeneous population model, we might expect to see a cyclical pattern of mortality reductions, and then a slowing of reductions as the negative implications of prior mortality improvements accumulate in the population. These mortality reductions and "retrenchments" might occur at increasingly advanced ages. The point is that future mortality trends might be anticipated by such heterogeneous population models. The need to develop models which can anticipate changes in mortality trends is made clear by the tremendous costs to Social Security and Medicare of even small failures in forecasting.

Directions for Biomedical Research

A subject frequently discussed by authors of models of human mortality is the implication of their models for the future directions of

biomedical research. For example, a number of current mortality models are premised on the concept of a "rectangularization" of the human survival curve due to the determination of an upper bound on human life expectancy by basic biological processes of senescence characteristic of the human species. Because it is argued that we are currently near limits on life expectancy that are determined by basic biological processes of senescence, these theorists suggest that biomedical research be redirected from studies of specific disease processes to investigation of the basic biological processes of senescence. Only in this way, it is argued, can we continue to improve life expectancy. In our alternative model, we posit that aging can be defined in terms of a set of chronic degenerative disease processes, and, as a consequence, medical innovations in the management of chronic disease can continue to extend life span.

The evidence suggests that we have not exhausted the potential for life-expectancy changes due to the control of chronic diseases. Indications are, for the first time, that the human life span may have significantly increased through the control of chronic diseases. Furthermore, there are biologists who argue, in contrast to Fries (1980) and Hayflick (1975), that in the relatively near future (say 35 years) we may experience major reductions (up to 25 percent) in the rate of aging: "Unless the aging process differs in some mysterious and totally unforeseen way from other puzzles man has faced in the past, it is essentially inevitable that he will, before long, understand aging's sources, and with that understanding will come a considerable measure of control" (Strehler, 1975:5). Such a reduction in the rate of aging would produce an extension of life span from say 100 years to in excess of 125. Of course, the realization of much of this life span potential is dependent on the corresponding biomedical advances to allow individuals to survive diseases. Though current evidence indicates that our present life-expectancy changes at advanced ages are due to the control of chronic disease, the possibility of techniques for slowing the intrinsic aging rate implies a potential for considerable life-expectancy gain over the short run even if we were currently near a biologically determined ceiling on life expectancy.

Apart from qualitative implications for the long-term direction of biomedical research, there are serious short-run implications. For example, Kramer (1980) correctly identifies the implications of population aging if no steps are taken to reduce the absolute level of

disability and morbidity potentially associated with the growth of the elderly population. Gruenberg (1977) argues that relatively little effort is expended on "health saving" research. Fries (1980), though identifying what he feels are critical elements in health preservation, unfortunately offers us little guidance on how to deal with the current implications of changing mortality for the health state of the elderly. Thus, much biomedical research must be directed towards the development of a full range of programs dedicated to health preservation in the elderly population. Such programs will necessarily involve primary prevention and lifestyle modification as strategies to minimize both the severity as well as the incidence of chronic diseases. However, such efforts must also recognize political, cultural, and ethical constraints in altering behavior for health preservation and the necessary importance of clinical innovation in chronic disease management.

Structural Changes in American Institutions: Jobs and Careers

The quantitative implications for the magnitude of population aging and the qualitative implications for biomedical research lead us to ask a further critically important set of questions. These deal with the nature of both individual and institutional responses to population aging and whether adequate solutions will require basic structural innovations. Consideration of the possibility of structural changes seems to imply the notion of a "threshold," i.e., a limit on population aging beyond which responses within existing institutional structures no longer are adequate. Operationally, the concept of a threshold requires that we select a critical population variable which, upon reaching a given level, will imply the need for major structural changes. A number of such threshold variables might be selected, such as the proportion of the population over age 65, the absolute number of elderly persons, or an elderly "dependency" ratio.

In the context of our analysis of mortality, the variable we would select for evaluation is life expectancy, for it has clear implications both for the magnitude of population aging and for individual decisions about labor force participation and lifestyle. The evidence suggests that this life expectancy threshold already has been achieved. On the institutional level, our present estimates of the growth of the elderly population under current forecasts of life-expectancy change

indicate short-run difficulties due to economic conditions, and long-run difficulties due to demographic factors, in the maintenance of the present Social Security and Medicare systems. On the individual level, current life expectancies at birth approaching 80 years for white females (with a median expected life span of 84 years) suggests that the probability of surviving to advanced ages is currently high enough that specialized responses to the problems of the extreme elderly must be sought. Among gerontologists, recognition of this age heterogeneity within the over-65 population has already fostered subcategorization of that population into young-old (65–74) and old-old (75+) components. As life expectancy increases, further subcategorization will be required. If aging processes change qualitatively, in a way correlated with life-expectancy increases, as we have argued, then we must seriously reevaluate our perceptions of the needs and potential of groups such as the young-old. It has been suggested that we eschew chronological-age criteria entirely in favor of "functional age" (Kraegel, 1980:1355). Practically speaking, such proposals are premature since no really successful measure of functional age has been developed, and because functional age measures lead to a series of operational difficulties (e.g., they require extensive measurements to be determined; they rise and fall with changes in health status). So far, studies have shown that chronological age is the simplest and most generally valid measure of functional age currently available (Costa and McCrea, 1980).

Thus, if projections based on current forecasts of life expectancy suggest extreme difficulty in "patching" present institutions to cope with population aging, modified forecasts allowing for significant further increases in life expectancy suggest that only by structurally modifying societal institutions will we be able to cope with the extent of the aging of the population of the U.S. If we examine only the quantitative implications of population aging, the options for institutional change seem limited to a series of undesirable choices involving the reduction of entitlement criteria. In the best case, this will lead to short-run inequities. However, if proper attention is paid to the qualitative factors underlying population aging, then perhaps institutional changes can be devised to deal with population aging in positive ways. In the following, we shall consider how positive institutional changes could be made in one major section of society—the labor market.

For example, we might devise institutional structures which could

optimize the economic productivity of elderly individuals. One possibility is that life expectancy in the U.S. is sufficient to permit multiple-career/education cycles to be pursued within a single life span (Strehler, 1975). In 1978, for example, white males in the U.S. could be expected to live 14 years and white females 18.4 years after age 65, and from the evidence, much of the life span after age 65 for many persons is potentially productive. Viewed differently, a life expectancy of 14 years at age 65 implies a posteducational life span for males who survive to age 65 of over 54 years, a period of time adequate to permit the individual to plan for multiple-career cycles. It is possible that by offering the opportunity for multiple careers within one lifetime, the trend to early retirement could be reversed and elderly individuals kept in the labor force to more advanced ages.

The practicality of multiple-career cycles for individuals under the current level of life expectancy has already been demonstrated in the U.S. In the federal sector, such practices have nearly been institutionalized, with individuals entering a normal civil service track after completing 20 years of military service. It seems likely that similar multiple-career cycles could be accommodated in the private sector, with appropriate institutional changes. The required institutional changes will be both structural (i.e., easing administrative and statutory restrictions on the labor-force participation of the elderly) and attitudinal. Attitudinal changes will be particularly important among nonelderly labor-force participants because current expectations hold that early retirement will bring about rapid career advancement for younger workers. The introduction of career patterns with multiple-career cycles could help increase acceptance of elderly workers since each career cycle would have a fixed expectation of, say, 20 years. Consequently, with older persons entering new careers at midlife, advancement would not be so rigidly correlated with age. Thus, multiple-career cycles for individuals could maintain a high rate of circulation through jobs and career levels as well as permit career change and retraining (Strehler, 1975).

A second area where changes could be made to accommodate older workers is in job structure. Basically, jobs would be restructured on the basis of the principle of "compensation." Compensation implies that, though certain physical capacities such as neurological reaction time may decrease with age (at highly variable rates), there are compensating increases in skill, judgment, and stamina (McFarland,

1976). Evidence from longitudinal studies of elderly populations suggests the feasibility of compensation, especially in the case of intellectual functioning. While certain cognitive functions are simply maintained, others—such as vocabulary—can increase well into advanced years (Riegel, 1971). Compensation may well be implicit in the current structure of many jobs since many physical functions begin to decline significantly after age 30 (Upton, 1977). It seems reasonable that the principle of compensation can be extended further to utilize the full economic potential of the elderly.

Two factors suggest that there is considerable potential for the application of the principle of compensation in job restructuring. First, a large proportion of the U.S. economy is in tertiary (i.e., service) sectors. In the tertiary sector, there will be a greater potential for compensation to allow skill, experience, and intellectual capacity to be emphasized in jobs. Second, physical ability to work may be preserved to more advanced ages by the principle of compensation. Specifically, the ability to work has been found to be higher for those who are in professional occupations, are better educated, and of higher socioeconomic status (Sheppard and Rix, 1977:56–57). These observations can be attributed to a number of factors, but they suggest that as the elderly population increases in social and economic status, there will be an enhanced potential for continued economic productivity.

Of course even if one accepts the potential for increased economic productivity of the elderly through job restructuring and institutional accommodation of multiple-career cycles, there exists a large number of practical problems in implementing programs to realize this potential. For example, the number of female survivors to age 65 and older is much greater than the number of male survivors. Hence, one problem is to determine how, over the next 25 years, females who are currently survivors (in 1980) of cohorts born in 1915 and earlier can be integrated into the labor force. For such females, there will presumably be the need for special educational and training programs in order to compensate for the lower educational attainment and lack of work experience in these cohorts. These problems will be exacerbated because of the high rate of widowhood among females, with the implications of widowhood being fewer resources available through social networks and higher risks of institutionalization. The problem for elderly males may not be as difficult because: a) there are fewer male survivors to age 65 and over; b) most males will have acquired

some skills and work experience; and c) male rates of widowhood will be much lower. Certain problems will perhaps be even more acute for nonwhites (e.g., cohort differentials in education).

Furthermore, many of these problems will exist for some time into the future because of the lower educational advancement of the older birth cohorts. Thus, to fully utilize the potential of the elderly population, special career-training programs would have to be devised which take into account "educational level, attitudes, interest and functional capacities of survivors" (Kramer, 1981). Another series of problems would have to do with the practical difficulties of breaking up an individual's work life into several career cycles. Presumably, each cycle change would involve some retraining and possibly a limited "sabbatical" period. To facilitate such changes, it would be useful if, for example, one could make a partial withdrawal of pension payments already made at the time of the career shift. This would require a restructuring of present pension systems, but if it helped to retain workers in the labor force to more advanced ages, it might serve to alleviate the pressures on such pension systems by rapidly increasing life expectancies. From this discussion a number of obvious questions arise. Who will fund such programs? Can fiscal incentives be developed to involve the private sector in such programs? How much will government have to contribute? The one certain point is that failure to more fully utilize the productive potential of the elderly population becomes increasingly costly as the population ages, i.e., the need for action will not disappear—it can only grow.

If we accept that we can reduce the economic dependency of elderly populations by redefining jobs and careers, then we are faced with determining how to adapt entitlement to present government programs (e.g., Social Security, Medicare) and private pension funds. These questions will be tremendously complex, involving political and social equity issues as well as assessments of functional capacity. One response might be to change nothing, but current economic pressures make this increasingly difficult. These pressures are becoming so great because the present entitlement criteria were not developed systematically:

> The lore of gerontology tells us that Bismarck nearly a hundred years ago chose 65 as the retirement age under his social welfare system because Krupp advised him that most workers wouldn't live much beyond that, anyway, so the costs would not be burdensome.

> Can we truly continue to use a nineteenth century criterion as we move towards the twenty-first century—when persons at age 65 can be expected to live on the average at least fifteen years and perhaps longer if our interpretations of biomedical developments are correct? (Sheppard and Rix, 1977:73)

Even when the entitlement age is based on analysis, there has been little provision to systematically update the entitlement age to reflect new actuarial realities. For example, in Japan after World War II, the retirement age was set to age 55, based on prewar data in which the average age at death was only 50 (Maeda, 1980; Sheppard and Rix, 1977). Since that time, the average age at death has risen to 75—but the retirement age has not been adjusted. Similarly, in New York City, the pension-funding system was based on longevity calculations using pre-World War II mortality data—in some cases, 1918 mortality rates had been used (Sheppard and Rix, 1977:62). The resulting financial difficulties of both the Japanese and the New York systems are well known. In the U.S., current life-expectancy levels pose serious problems. If life expectancy increases in the fashion suggested by the Social Security forecasts in Table 7, the situation can only become more grave. As Strehler (1975:8) has emphasized:

> It is more than obvious that both the business (particularly insurance) sector and the public bodies that are parts of the equation should begin now, if not yesterday, to make more projections for the future. To base investment, government or private, on today's life span is to ignore clear handwriting on the palace wall, and the dislocation of retirement income crisis could make the 1974 energy crisis look like an afternoon tea.

Conclusion

In the preceding discussion we have attempted to accomplish three basic goals. The first is to alert us to the potential difficulties inherent in existing models and theories of human mortality and to point out how many of the current arguments are inconsistent with available data. Second, we have examined the implications of these various models for our perception of both quantitative and qualitative aspects of population aging. Finally, our third and overriding goal is to

stimulate research into the nature and implications of current mortality in the U.S. In past policy and research, mortality has often been treated as a topic of secondary interest. The evidence we have presented on the role of mortality in determining quantitative and qualitative aspects of population aging suggests that this will no longer be satisfactory. Instead, systematic mortality research, with an integrated multi-disciplinary perspective, is necessary to understand current mortality conditions and to anticipate future mortality trends.

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