

Dynamics of Health Changes in the Oldest Old: New Perspectives and Evidence

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THE AGING OF THE UNITED STATES POPULATION IS a phenomenon that has serious and wide-ranging implications for both social and health policy. Certain dimensions of these implications are well understood and well documented. For example, the elderly population has grown by 9.3 million, or 55.6 percent, from 1960 to 1980 and is projected to continue to grow to 36.3 million by the year 2000, and to 67.3 million by 2040. We can anticipate many of the changes produced by this growth (Myers and Manton 1983), such as an increase in the proportion of the gross national product devoted to health care from 5.3 percent in 1960 to 9.5 percent in 1980—a figure projected to reach 12.0 percent by 1990 (Rice 1980; Rice and Feldman 1983; Freeland and Schendler 1983).

Aspects of the current aging trends in the United States population are, however, historically unique and hence are neither well understood nor documented. Four of the historically unique aspects of current aging trends are (1) the rapid growth of the oldest old (aged 85 and older) population, (2) increases in life expectancy at advanced ages, (3) the predominance of females at advanced ages, and (4) reductions in the age-specific mortality rates of certain major chronic degenerative

diseases (e.g., stroke, ischemic heart disease). These historically unique elements of current population aging suggest that we need to examine carefully the dynamics of health changes in the oldest old (aged 85 and older) for their number is projected to increase rapidly (to 5.4 million in 2000; to 13.3 million in 2040) and to become a significant proportion of the total population (4.0 percent by 2040). The improvement in survival at advanced ages strongly suggests that important changes in health and the natural history of disease processes may be occurring concurrently.

Consequently, in assessing health changes at later ages, it is important to employ concepts of health and disease flexible enough to describe newly emerging patterns. Given the highly selective nature of survivors to extreme old age, it is also important to distinguish clearly between changes in the mechanisms of aging and morbidity at the individual level and the pattern of morbidity and mortality rates expressed at the population level. For example, while the age-specific risk of certain types of cancer may increase throughout life, there may be a tendency for the age trajectory of population mortality rates to flatten, or even decline, at advanced ages due to systematic removal by mortality at relatively young ages of high-risk persons (e.g., heavy smokers). Thus, we cannot simply infer the age dependence of morbidity processes for individuals from population rates. A related problem has affected certain physiological studies of aging since physiological parameters were sometimes estimated from a study population which contained a mixture of "morbid" and "healthy" elderly. Consequently, in our assessment we must be aware of new scientific findings about the nature of the physiology of aging and disease at advanced ages—especially with regard to the extreme individual variation and increased chances for intervention in those processes (Minaker and Rowe 1985).

In the following pages we will conduct an assessment of individual health changes at advanced ages using a broad range of vital statistic and epidemiological data and data from longitudinal aging studies. In conducting this assessment we will deal with the issues raised above, i.e., the need for new concepts of aging and disease, the heterogeneity of health changes, and new scientific insights. To begin, we will present a general model of health changes based upon cohort and life-course perspectives. This model will serve as a tool for describing the relation of morbidity, disability, and mortality and the changes of those relations over age. One of the principal values of such a

model is that no single data source contains detailed and nationally representative time-series information on all three types of health outcomes (i.e., specific types of morbidity, disability by type and level, and cause-specific mortality) for large numbers of persons over age 85. Consequently, we must rely on combining information from a multiplicity of studies each of which will be limited in scope, quality, or size. To develop a coherent picture from such a wide range of data sources we need a model that can function as an integrating mechanism.

The first type of data discussed is national cause-specific mortality data for the period 1968 through 1980. Though describing health status only at the time of death and being subject to data-quality questions such as artifacts due to physician custom in filling out death certificates, these data have the advantages of being nationally representative, reporting on large numbers of events at age 85 years and older (e.g., over 350,000 deaths in 1980), and of being available for an extensive time series. Furthermore, recent studies (e.g., Lubitz and Prihoda 1983) show that treatment costs during the final year of life are a large proportion of total Medicare disbursements (28 percent in 1978) so that the medical conditions reported at death are important determinants of acute-care costs among the elderly. The relatively short life expectancy at age 85 and above (i.e., 5 to 6 years) would also suggest that medical conditions reported at death are more closely associated with disease prevalence and incidence than at younger ages. We will examine multiple-cause mortality data, where all conditions reported by the physician are recorded; hence, changes in the mortality patterns will be informative about underlying health changes.

The second type of data we will discuss is data on individual and population morbidity and disability characteristics of the oldest old. In this portion of the analysis we will focus upon data of three types. The first is from a longitudinal study of normal aging conducted at Duke University from 1955 to 1976. Though the study population was quite small, the data encompass a broad range of physiological and psychological measurements repeated 11 times on an extremely elderly population (mean age at entry was 71.3 years). This data set is used to examine the nature of aging trajectories at the individual level. The second data source examined is the 1982 National Long-Term Care Survey. This survey collected extensive data on functional disabilities in the elderly (aged 65 and older) noninstitutionalized population. Using this survey we can examine the national distribution

of chronic disability in the noninstitutionalized population and the association of those disabilities with self-reported medical problems. The third source of data are the 1964, 1969, 1973, and 1977 National Nursing Home Surveys (National Center for Health Statistics 1967, 1974, 1977, 1981) from which we can assess the health characteristics of the nursing home population—a population component that is particularly important for the population aged 85 and over because of its high rate of institutionalization (Soldo and Manton 1985).

Finally, we compared patterns of mortality changes in the United States and Japan—a country that is also experiencing rapid population aging and life-expectancy increases. Given limitations on cross-temporal data in the United States, the international comparison will be useful to determine the variability of aggregate health conditions at advanced ages in different social and economic contexts. This type of comparison may tell us if there is potential for further increases in life expectancy at advanced ages, as might be implied if two countries with similarly high life expectancies had very different cause-of-death patterns—suggesting that the mortality rates for certain diseases in one country may be lowered to the level observed in the other country.

A Model of Health Changes at Advanced Ages

There is considerable debate over the implications of recent mortality changes for the aggregate health characteristics of the elderly population (e.g., Feldman 1982; Brody 1983, 1985; Manton 1982, 1983; Fries 1980, 1983). Much of this debate could perhaps be resolved, or at least focused, if a common conceptual framework general enough to describe health changes at later ages were available. To aid in our discussion we present such a model, developed at a World Health Organization Scientific Group meeting on the “Epidemiology of Aging” in January 1983 (World Health Organization 1984). The model utilizes a set of concepts from a standard tool for analyzing survival known to epidemiologists, public health researchers, and policy analysts—the life table. Basically, the model is constructed from a series of life-table survival curves that describe the change in the proportion of a cohort that can expect to survive to a given age without one of three basic types of health events occurring—morbidity, disability, or mortality. The model can be simply illustrated.

In figure 1 the horizontal axis represents age and the vertical axis

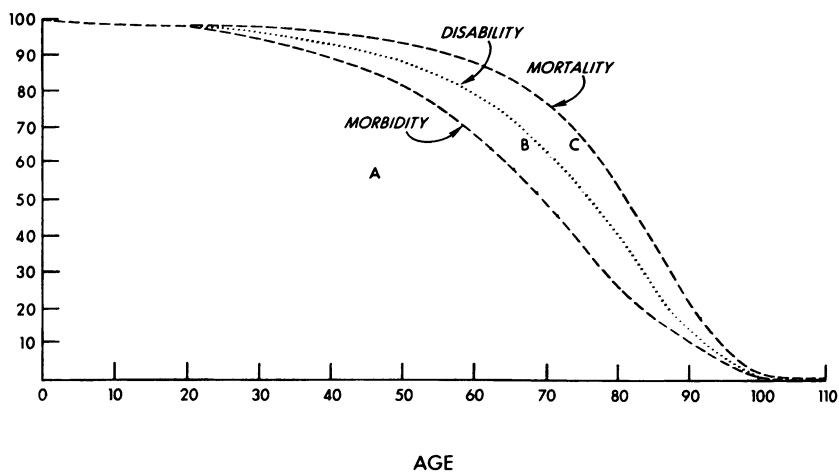


FIG. 1. The mortality (observed), morbidity (hypothetical), and disability (hypothetical) survival curves for U.S. females in 1980.

describes the probability (expressed as a percent) of surviving to a given age without suffering one of the three critical health events. The spatial relations of the three curves have substantive interpretations in terms of the changing health burden on society of age-related morbidity and disability. Specifically, the areas in the figure are defined by a product of age (time) and the *average* probability (for an individual; frequency in a population) of being in a given health state. Consequently, the areas describe the numbers of person-years spent by the cohort or life-table population in specific health states. Thus, the area marked A represents the number of person-years spent free of disease, the area marked B represents the number of person-years spent with chronic disease but unimpaired, and area C represents the number of person-years a given cohort can expect to be disabled. Areas A and B combined represent a measure of the potentially productive or active life expectancy.

The reason for presenting this model is not simply as an exercise to compare survivorship and life-table models. Indeed, the mathematical theory of survivorship is a mathematical theory about the life table. Instead, it is presented as a conceptual device which represents all the variable interactions that are necessary to describe aging and health changes at advanced ages, but which are too complicated to be portrayed without a model. The model is necessary to use the information-organizing power of mathematics to develop a comprehensive theoretical framework within which complex aging processes can be described.

Naturally, one measure of the utility of the model is the uses to which it can be applied. For different types of information, and for different questions, it can be utilized in different ways. For example, as in any model of survivorship, it can be used to portray the mortality risks of a cohort. Often, however, cohort data are unavailable. In these cases we use period mortality rates to construct a hypothetical life table. Though such a life table reflects the cross-sectional experience of many cohorts at a single point in time, we still can derive useful information from it such as the life expectancy at birth that would obtain under the mortality rates for a given period. Similarly, the model described above can be applied to cohort or longitudinal data when they are available. When such data are not available, the model can be applied to cross-sectional data to provide summary measures of what the current disability, morbidity, and mortality rates imply about health status at advanced ages. We will examine three possible uses of the model: (1) as a conceptual framework to put different theories of aging-related health changes into a common perspective, (2) as a measure of population health status, and (3) as an actuarial tool for describing the need at the population level for specific types of health services.

As a Tool for Reviewing Different Theories of Health Changes among the Elderly

The first use we will make of figure 1 is to examine six different perspectives or models of health changes at advanced ages.

Strehler (1975), and more recently Walford (1983), have speculated about the possibility that basic research into the aging process may yield interventions that could increase the human life span by 25 years or more. Strehler (1975) suggested that such a breakthrough could occur within 35 years, i.e., by 2010. In discussing the health changes likely to be associated with such life-span extension, Strehler argues that, since interventions would be made into basic aging processes which determine the age at onset of disease and disability, the same amount of time would be spent in terminal decline (i.e., the size of area A would increase but areas B and C would be fixed). Consequently, only a productive life span would be lengthened. In terms of our model this would imply that the three curves would be maintained in a fixed relation but that they would be shifted 25 or more years outward along the age axis.

Gruenberg (1977) and Kramer (1981) have also discussed the recent course of health changes in the United States. They suggest that though there have been significant increases in life expectancy, there is little evidence of changes in the age at onset of morbidity and disability. Thus, while the morbidity and disability curves in figure 1 remain relatively fixed (i.e., area A unchanged), the mortality curve would be shifted to the right increasing the number of person-years expected in morbid and disabled states (i.e., areas B and C would expand). This, they suggest, is a result of an imbalance between expenditures on disease prevention versus the clinical management of disease.

Fries (1980, 1983) has argued for the possibility of the "compression of morbidity" by appropriate lifestyle interventions. Specifically, he has suggested that we, in the United States, are rapidly approaching the point where the survival curve would become "rectangularized," i.e., where most persons would survive to their biologically endowed life span and die of "natural death." He suggests that, though the mortality curve has become nearly fixed, it is possible, with appropriate interventions, to move the morbidity and disability curves toward the mortality curve, i.e., that the age at onset of morbidity and disability would converge toward a relatively fixed target. This would compress areas B and C and significantly reduce the number of person-years spent in health-impaired states.

Feldman (1982) prepared a report for the recent commission on restructuring Social Security in which he advised caution in changing the entitlement age of the system. The proposed changes in entitlement age (i.e., to age 67 by the year 2000) were accepted on the argument that there must have been age-specific improvements in health status associated with recent increases in life expectancy. He proposes a simple model of health-state transitions, such as those which must underlie the survival curves in figure 1, which he uses to illustrate how life expectancy could have increased but also with a net increase in the prevalence of chronic morbidity and disability. He argues that the available time-series data are currently inadequate to answer the question of whether health status has truly improved.

Golini and Egidi (1984) conducted simulations of the effects on population health status of different interventions on a similar set of health-state transitions to those postulated by Feldman (1982), i.e., disease incidence, disease duration, and case fatality. They used a simple one-disease model, an experience broadly representative of

Italian morbidity and mortality changes at advanced ages, to examine the relative impact on survival, population structure, and disease prevalence of different health interventions. Thus, they were, in effect, examining the types of health transitions that would produce a particular age configuration of morbidity, disability, and mortality curves. What they found was that reasonable changes in case-fatality rates and disease duration tended to dominate reasonable or likely changes in disease incidence. That is, a one-year increase in disease duration for a disease like cancer with initially a two-year average survival increased disease prevalence by 50 percent. They suggested that it would be unreasonable to postulate a decrease in incidence of 50 percent so they projected an inevitable increase in prevalence of chronic degenerative diseases in developed aging societies.

Riley and Bond (1983) reviewed a wide range of recent scientific evidence on aging and found both a high degree of individual variability in rates of aging changes and a relatively intact maintenance of certain physiological functions, even to the eighth decade of life (Lakatta 1983). Basically, these findings suggest that many of the physiological changes associated with aging can be identified with age-related pathological changes and are not directly tied to intrinsic aging processes. The evidence suggests that (1) the three health outcomes are interrelated, (2) changes or interventions at one level will have feedback to other health outcomes (i.e., improvement in morbidity or slowing the rate of progression of chronic disease will have an impact on mortality), and (3) there are greater opportunities for specific interventions into select dimensions of age-related health changes than have been heretofore recognized (e.g., World Health Organization. Regional Office for Europe 1982). This suggests that all three curves can be moved, and that, for example, an appropriate allocation of resources can compress the morbid and disabled period, but not with an absolutely fixed mortality curve. Manton (1982) described this type of interdependence as a dynamic equilibrium of morbidity and mortality—an equilibrium that is played out on each of multiple interacting dimensions of physiological aging changes. These last perspectives, which reflect recent scientific insights into aging processes and health changes at advanced age, suggest that, though figure 1 is a useful conceptual model, its empirical application must consider two further factors.

One factor involves the extreme heterogeneity of aging rates and health changes in the oldest old population. Riley (1981) recognizes this in terms of cohort differences (i.e., that the life-course experience

of a given cohort will be an important determinant of rates of health change at later ages) and in the observation that the oldest old population is composed of highly selected survivors from the cohort. It also suggests that extreme care be used in discriminating between changes in life chances at the individual level and the aggregate implications of those changes at the population level. For example, we may find that individuals with specific chronic diseases have those diseases better controlled with a net improvement of life quality; this, however, may lead to increased age-specific prevalence rates due to improved survival and possibly to greater aggregate demands for health services.

A second factor found in recent scientific investigation is that aging changes at advanced ages are multidimensional and that the different dimensions proceed along different aging trajectories. For example, while certain cardiac-output parameters show little degradation with age in the absence of explicit disease (Lakatta 1983), other physiological functions such as renal function show slow linear declines. Also, most manifestations of aging at advanced ages apparently represent a gradual, but relatively constant, accumulation of aging changes and not an acceleration of aging processes at advanced ages. To determine the effect of specific interventions one must assess the systematic feedback between interventions for specific organ systems and determine how this will affect the rate of accumulation of aging changes.

As a consequence of these two factors, empirical application of the survival-curve model in figure 1 requires analytic procedures that recognize (1) the population heterogeneity of age-related health changes, and (2) the multidimensionality of aging processes at advanced ages. Such procedures have been developed and applied both to cause-specific mortality data (e.g., Manton and Stallard 1984) and to community epidemiological data (e.g., Manton, Stallard, and Woodbury 1985). The purpose of these efforts is to generate empirical estimates of the transition rates between the various health states as categorized in figure 1. By understanding the health state transitions one can determine how a population changes health status over time and age (Feldman 1982). This is important in understanding the physiological mechanisms generating those changes, identifying factors affecting the transitions between health states, and projecting the future health status of the population at different ages and for different times (e.g., Manton and Liu 1984a; Manton, Stallard, and Tolley 1983; Manton 1984). Examples of such efforts are discussed in this paper.

A Static Measure of Health Status

The second use of the survival-curve model is as a measure or index of health status in elderly populations. In such an application one is concerned with the cross-sectional relation of morbidity, disability, and mortality and not directly with estimating the transitions between those states. Such an application is illustrated in figure 2 where the health status of Japanese males and females in 1979 is described.

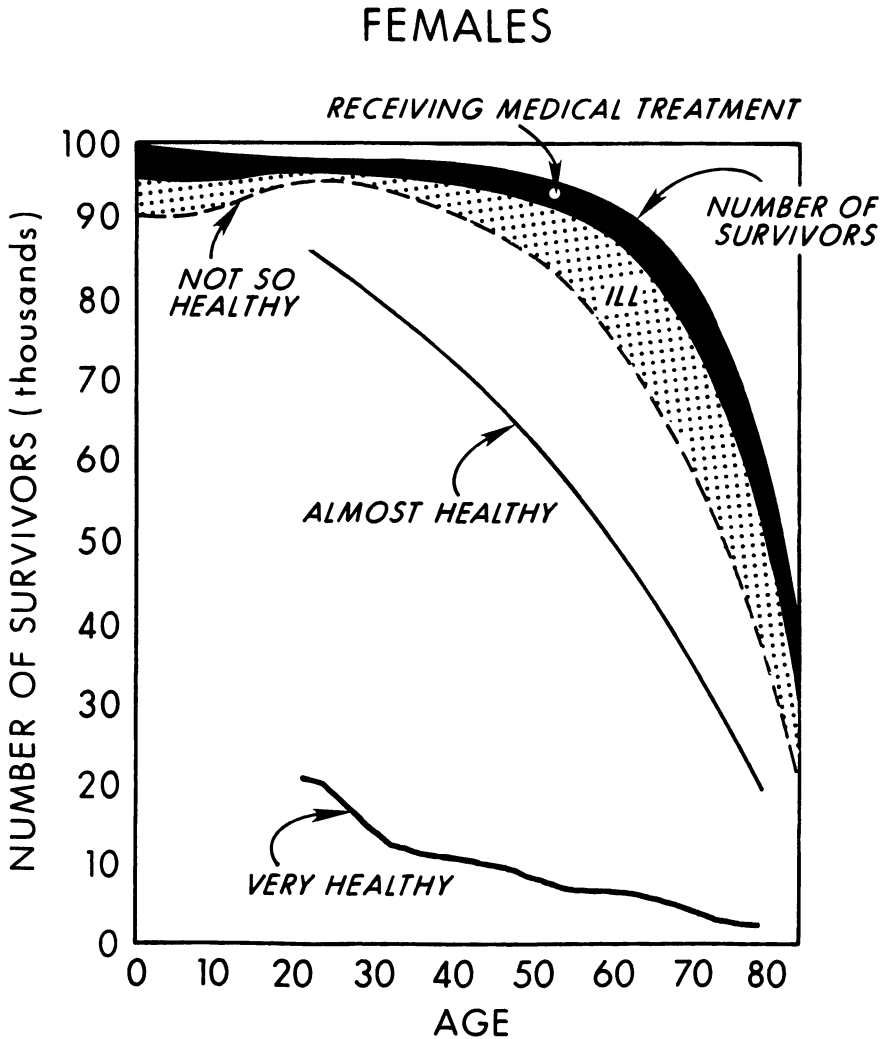


FIG. 2. Quality of life as seen from state of health first draft, Japanese males and females.

Source: Koizumi 1982, chap. 6, pp. 6-15, table 7.

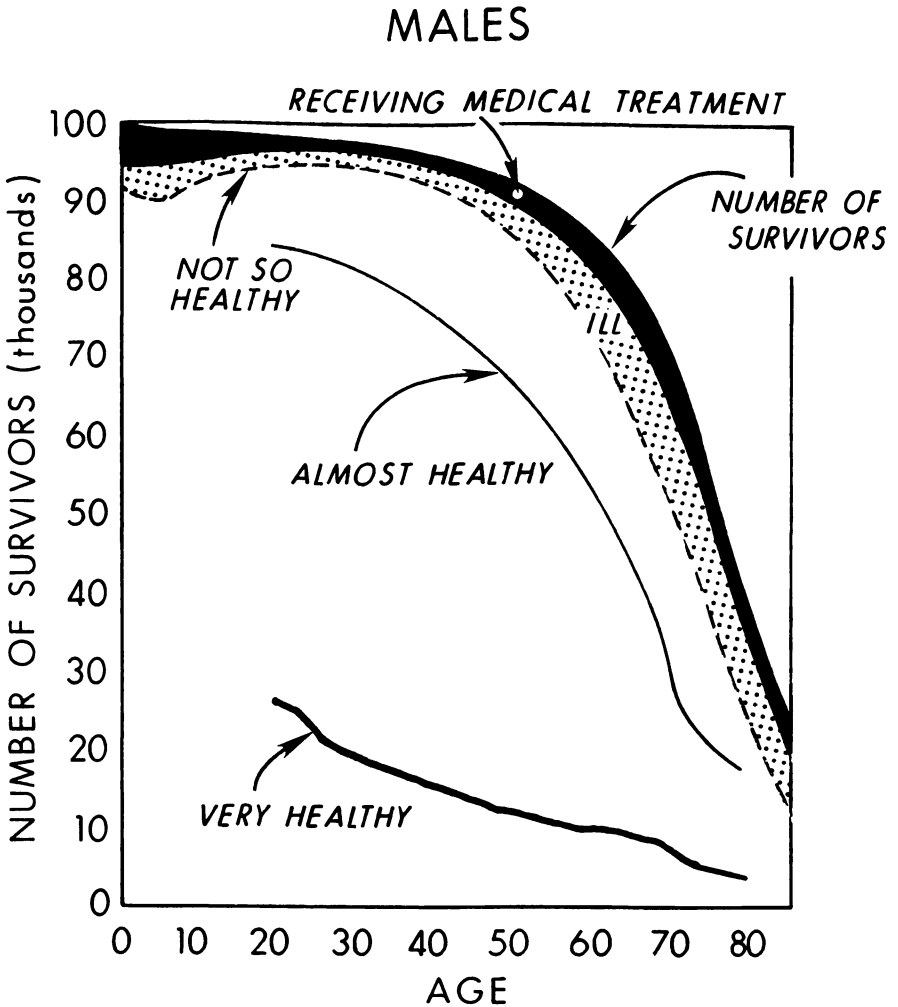


FIG. 2—Continued

The curves were derived from (1) life tables constructed from vital statistics, (2) a patient survey to determine the level of medical care utilization, (3) a national health survey to determine disease prevalence, and (4) the Basic Survey on health to determine the degree of health consciousness. There have been a variety of attempts to develop such life-expectancy measures adjusted or weighted for health or functional status. For example, the use of the survival curves as illustrated in figure 2 was motivated by the concept of "productive man-years" developed by Sanders (1964). Shepard and Zeckhauser (1977) proposed a "quality adjusted life year" (QALY). QALY involves assigning weights

to different disability states in terms of their relation to full functioning on one hand and death on the other. The QALY was used, for example, as a measure of health outcome in reviewing the impact of pneumococcal vaccine (Office of Technology Assessment 1979). Recently, Katz et al. (1983) have been developing and evaluating the use of "active life expectancy" measures as a tool for describing the relative change of functional status and life expectancy at advanced ages. Lalonde (1975) applied a similar concept in an assessment of health status in the elderly population in Canada. Though insights presuming the logic of a model like that in figure 2 have been made by a number of researchers, we, in our assessment, will focus on the implications of the model for the development of models of the *processes* of health changes at later ages rather than for the development of indexes or specific measures.

The parallel survival-curve concept was used by Koizumi (1982) to make two types of health comparisons. First, a cross-sectional comparison of sex differentials in mortality, morbidity, and health-service utilization was made. From figure 2 this suggested a pattern similar to that observed in the United States, i.e., the greater survival of females along with their greater reported prevalence of chronic conditions and higher health-service utilization (Verbrugge 1984). Cross-temporal comparisons within sex were also made. From 1965 to 1979, these comparisons showed a consistent improvement in survival but little consistent improvement in either disease prevalence or degree of medical care received on an age-specific basis. This implied that a major factor in the improvement of survival was a more successful management of chronic disease (such as suggested by Golini and Egidi 1984). This also implied that the number of person-years spent in the morbid and disabled states (areas B and C in figure 1) increased more rapidly than those spent free of disease (area A).

The Need to Modify Actuarial Measures to Reflect the Operation of Health Processes among the Elderly

A third use of the survival-curve model is as an actuarial tool. In one type of application, a particular form of this model is used to estimate health-state transition rates that are then used to project future disease costs (Tolley, Manton, and Stallard 1984). A more direct application of the survival-curve concept as an actuarial tool is in a recently

proposed modification of the "adjusted annual per capita cost" (AAPCC) formula which is used to reimburse health maintenance organizations (HMOs) under Medicare. The original formulation of the AAPCC was not explicitly calibrated for health-status differentials between HMO populations (Kunkel and Powell 1981). Among the proposals for introducing health status in the AAPCC were two involving disability indices and prior utilization of health services (Cookson 1983). A third formulation (Tolley and Manton 1985) involved adjusting the reimbursement level for total and cause-specific mortality differentials between geographic areas. This adjustment was based on the observation that a significant portion of Medicare disbursements is made in the final year of life (Lubitz and Prihoda 1983). The age-specific differentials in Medicare disbursements in the final year of life are summarized in table 1.

In the table we see that, for persons aged 67 and over, the ratio of expenditures between decedents and survivors is 6.2 to 1. This ratio decreases significantly with age, from 9.8 at ages 67 to 69, to only 3.7 over age 85. The decrease in the ratio with age is brought about both by a 50 percent increase in the costs of treating survivors and a 57 percent decline in the terminal-year costs of nonsurvivors.

Not only do terminal-year costs change significantly with age but there is also a significant difference in costs due to the nature of the terminal event. These differentials are presented in table 2 where we

TABLE 1
Medicare Reimbursement per Enrollee in 1978. According to Age

Age Interval	Reimbursement per enrollee Survival status		Decedents-to survivor ratio
	Decedent	Survivor	
67+	\$4,527	\$729	6.2
67-79	5,801	592	9.8
70-74	5,466	668	8.2
75-79	5,056	771	6.5
80-84	4,274	859	5.0
85+	3,285	889	3.7

Source: National Center for Health Statistics 1983, 17.

TABLE 2
Five General Causes of Death and the
Average Charge to Medicare in the
Year of Death

Cause	Medicare charge
IHD	\$4,400
CVD	4,400
CANCER	6,600
ACCIDENT	5,100
OTHER	5,300

Source: Tolley and Manton 1985, table 2.

see a 50 percent differential in terminal-care costs between cancer and stroke.

Thus, part of the age differential in terminal-care costs will be due to the change with age of the mix of health problems (e.g., cancer will be less significant at advanced ages). Significant differences in local-area health costs can be expected because the mixture of health problems varies significantly geographically (e.g., Mason et al. 1975; Kuller et al. 1969).

Since cause-specific mortality data are readily available, it was proposed that the mortality profile of the catchment area of an HMO, such as a county, could be used as an index of the differential morbidity and disability costs associated with the area's population. Hence, using data on the typical pattern of expenditures associated with specific terminal illnesses, one could adjust reimbursement levels to the HMO, assuming a fixed relation between specific causes of death and prior morbidity and disability (Tolley and Manton 1985). The measure itself was based upon a model of health-status transitions which is used to forecast the probability of certain health changes preceding specific types of mortality by using a wide range of epidemiological and clinical information on age-specific case-fatality and incidence rates (Hartunian, Smart, and Thompson 1981; Singer and Levinson 1976). These transitions can be translated into "recurrence tables" which, when combined with cost factors, produce the aggregate costs generated by a specific-cause-of-death profile (i.e., the set of age- and cause-specific mortality probabilities for the local area population). An adjustment based upon mortality data is difficult to manipulate and

relatively resistant to antiselection bias (i.e., the recruitment of only healthy persons to the HMO) since the health status of a selected population appears to rapidly converge to that of the more general population (Cookson 1983).

Such an actuarial usage of the basic survival-curve model can be extended from determining current reimbursement levels to long-range planning for HMOs. If it is true that there are major medical expenses associated with terminal illness, then an HMO with a very healthy population is likely to experience heavy levels of disbursements as their population progresses to advanced ages. Thus, though an HMO can help promote health and prevent disease, it will remain at risk to high expenditures as large proportions of its population survive to advanced ages where mortality risks eventually rise. This is illustrated in table 3 where, using national cost figures, we have calculated age-specific differences in costs due to expenditures on terminal illness for two United States counties (Davis County, Utah, and Benton County, Washington).

We see that the Washington county has significantly higher costs through age 77 whereas the Utah county, being heavily Mormon, has greater life expectancy and consequently higher medical expenditures at more advanced ages (age 78 or higher). The implication of this different age patterning of costs is significant. First, it suggests that

TABLE 3
Annual per Capita Costs Due to the Mortality Process Assuming Cause-specific Mortality Costs as Estimated by Lubitz and Prihoda (1983)

Age (1-year intervals)	Benton County, Washington	Davis County, Utah	Ratio Benton/Davis
70	\$319	\$312	1.02
71	374	331	1.13
72	438	347	1.26
73	502	363	1.38
74	550	381	1.44
75	576	405	1.42
76	563	437	1.29
77	526	470	1.12
78	478	514	0.93
79	452	559	0.81

Source: Tolley and Manton 1985, table 8.

the fiscal viability of an HMO will be strongly determined by the age structure of its population and the interaction of that structure with its mortality risk structure (i.e., its age- and cause-specific mortality rates). Second, even for the county with the Mormon population, a population exhibiting many of the healthy lifestyle practices promoted as preventative strategies, there will eventually be a period of high fiscal risk when the mortality rates for that population start to increase at advanced ages (Tolley and Manton 1985).

Mortality and Survival: Recent Trends

In the prior section we described a general conceptual model of health status changes at advanced ages and discussed three possible uses of that model. In this and following sections, we will discuss the vital statistic and epidemiological evidence on individual components of that model, i.e., morbidity, disability, and mortality at extreme ages. The first type of data we will examine is mortality data.

Total Mortality Changes at Advanced Ages: The Age-at-death Distribution

The first model element we examined is the survival curve. There has been considerable debate about whether the survival curve has become increasingly rectangular in recent years. The answer to that question is certainly yes if we examine the entire curve from birth. However, we know that the chronic disease processes which cause most deaths at later ages are different from the causes of infant and childhood mortality, and accidental mortality at early ages. Indeed, the apparent rectangularization of the survival curve seems more a function of mortality rates at early ages having reached low levels relative to the mortality rates among the elderly than of the mortality changes observed at advanced ages. Indeed, at advanced ages there is some evidence provided by the Social Security actuaries that the upper age bound to survival has increased significantly (~8 years) from 1900 to 1980 (Social Security Administration 1982). Thus, if we focus our attention on mortality changes at advanced ages, the evidence about the impact of rectangularization seems quite different. Consider the illustration in table 4.

TABLE 4
Means and Standard Deviations of Ages of Death at Ages 60 Years and
Over by Sex, United States, 1962–1979

Year	MALE		FEMALE		TOTAL		Number	% of deaths at all ages
	Mean	S.D.	Mean	S.D.	Mean	S.D.		
1962	74.1	8.5	76.8	8.9	75.3	8.8	1,209,810	68.9%
1967	74.2	8.6	77.2	8.9	75.6	8.8	1,298,800	70.2
1971	74.4	8.7	77.6	9.1	75.9	9.0	1,373,889	71.2
1975	74.5	8.8	78.0	9.2	76.2	9.2	1,387,422	73.2
1979	74.9	8.9	78.6	9.4	76.7	9.4	1,436,416	75.0

Source: National Center for Health Statistics mortality data tapes.

In this table we present the means and standard deviations of all deaths occurring in the United States at age 60 years and older in several years. It has been suggested that rectangularization, if operational, ought to be manifest in a decreasing standard deviation in the age at death, because of the truncation of the age-at-death distribution by a fixed upper bound to the life span (Fries 1980). This reduction should presumably be greatest for those who are observed to be closest to the bound, i.e., those who die at advanced ages. We see that not only has the mean age at death past age 60 increased but so has the standard deviation (Myers and Manton 1984).

Fries (1984) suggests that selecting a fixed age to make this comparison is inappropriate because of the shift of the distribution to more advanced ages. He suggests that one should examine the age-at-death distribution from a fixed percentage point, i.e., the age after which a certain proportion of the deaths will occur. These figures are presented in table 5.

In this table we see that, for the last 75 percent of deaths there has been a 0.4- and 0.7-year drop in the standard deviation for males and females respectively though this drop has been accompanied by increases of 2.2 and 3.6 years in the mean age at death. What is interesting is that the size of the drop in the standard deviation of the age-at-death distribution decreases as we move further into the tail of the distribution, i.e., presumably closer to a fixed upper bound. For males, the standard deviation decreases only negligibly after the 66.6 percentile while, for females, the decrease is negligible for the last 25 percent of deaths. Since we would expect the ages of death

TABLE 5
Means and Standard Deviations of Ages of Death Following Various
Percentile Points of Death by Sex, United States, 1962 and 1979

Year	Percentile	MALE		FEMALE		TOTAL	
		Mean	S.D.	Mean	S.D.	Mean	S.D.
1962							
	75.0	71.6	10.0	76.2	9.3	73.6	9.8
	66.6	73.6	8.7	78.0	8.0	75.6	8.6
	50.0	77.1	6.9	81.3	6.2	79.1	6.7
	33.3	80.5	5.6	84.5	4.9	82.3	5.4
	25.0	82.3	5.0	86.3	4.2	84.8	4.5
1979							
	75.0	73.8	9.6	79.8	8.6	76.4	9.6
	66.6	75.3	8.7	81.3	7.7	78.2	8.5
	50.0	79.0	6.9	84.7	5.8	81.8	6.6
	33.3	82.4	5.5	87.8	4.5	85.1	5.2
	25.0	84.3	4.9	89.0	4.1	86.8	4.5

Source: National Center for Health Statistics mortality data tapes.

at the most advanced ages to be most affected by a fixed upper bound on life span this suggests that, up to 1979, the effects of rectangularization had been minimal at the ages where one would expect to see it most in evidence.

An examination of the change in the mean age at death for the different groups in table 5 is interesting. It suggests that the 25 percent of all deaths occurring in the United States at the latest ages (nearly 500,000 of the total two million deaths in 1979) had a mean age of 84.3 for males and 89.0 for females. This represents an increase of 2.0 years for males and 2.7 years for females over the corresponding 1962 values. Thus, not only is the standard deviation fixed at advanced ages but the distribution has shifted significantly upward.

It should be noted that such evidence can only be used in a negative sense to evaluate rectangularization of mortality at advanced ages. That is, one can only confirm that rectangularization is not evident in these data. If the survival curve did appear to become more rectangular with this type of evidence we cannot tell to what degree environmental factors were involved in producing mortality at advanced ages and, hence, contributed to an apparent rectangularization.

Cohort Mortality: Analytic Models of Mortality Changes at Advanced Ages

An advantage of examining the temporal change in the distribution of ages at death as part of the study of mortality at advanced ages is that the results are not affected by inconsistency of age reporting between death certificate and population data. A disadvantage in studying changes in the cross-sectional age-at-death distribution is that changes in the distribution can be affected by the aging of different-sized birth cohorts. Furthermore, in examining the change in the standard deviation of the distribution of ages at death past any point one does not know whether the standard deviation is increasing because the tail of the distribution is getting thicker or because it is becoming elongated. Though it is useful to examine the age-at-death distribution to determine if rectangularization is occurring because other investigators have proposed and performed similar studies, an analytically superior approach is statistically to fit specific models of the age increase in mortality rates to cohort mortality data. In this regard a study of the cohort-specific mortality of the population from 1968 to 1978 is relevant (Manton, Stallard, and Vaupel 1985). In this study 20 male and 20 female birth cohorts aged 65 through 84 in 1968 were followed for 11 years to ages 75 to 94 in 1978. For each of these cohorts a variety of models describing the age-related increase in mortality were fit. In this study Medicare data on mortality and population were used (Wilkin 1982), data which are viewed as being less prone to age-reporting problems than census data.

The findings of the analyses are informative in that the well-known Gompertz model of adult mortality (Fries 1983) was found not to fit these data because it predicted mortality rates that were significantly too high at advanced ages. Indeed, the failure of the Gompertz function, a special class of mathematical curves often used to describe life-table survival past age 30 (e.g., Spiegelman 1969), to reproduce the observed mortality-rate increases at advanced ages, ages where one might expect "natural death" to predominate, is consistent with numerous other empirical studies of mortality-rate increases at advanced ages (e.g., Wilkin 1982; Perks 1932). In addition to the Gompertz model, Weibull (1951) hazard models with a less-rapid rate of increase in mortality were tried. The Weibull and Gompertz models represent alternative theories about survival. The Weibull model was also found not to describe the data.

Traditional theories of mortality view populations as homogeneous. We departed from that perspective by conceptualizing the population as being heterogeneous, with different survivor curves describing the mortality experience of different subpopulations. The modification of demographic and actuarial models to reflect such risk-heterogeneity may also be viewed as a first approximation to reconcile population models of survival with the extensive epidemiological and clinical data on the existence of strong and measurable differentials in disease and mortality risks. The simplest modification of survival models to represent heterogeneity is to assume that the increase with age of the mortality risks of each individual is described by the same type of mathematical function (e.g., the Gompertz or Weibull functions), but that the age trajectory of mortality for the population is described by a model where there is a distribution over individuals of the parameters of those functions—a distribution that is systematically changed by mortality (Spiegelman 1969; Perks 1932). The net effect of introducing such heterogeneity in aging rates into the model is that the age rate of increase in mortality is less rapid at advanced ages due to the systematic early removal of “high risk” individuals (i.e., individuals with rapid rates of “aging”).

Introducing the effects of heterogeneity in the Gompertz model resulted in a significantly better fit—especially at advanced ages where the simple Gompertz function predicted too rapid an increase in mortality risks. In this regard it is useful to examine the change, across birth cohorts, of the estimated parameter controlling the curvature of the Gompertz function. There is a systematic decrease in this parameter from the birth cohort of 1883 to that of 1902. For males, the decline was from 0.1037 for the 1883 cohort to 0.0734 for the 1902 cohort. Since this parameter can be interpreted as the annual relative increase in the mortality rate we see that there is estimated to be a decrease of 29.2 percent in the rate of increase of mortality risks at these very advanced ages. This suggests that the interventions which have reduced cause-specific mortality risks have served to alter the age rate of mortality increases *even* at advanced ages where it is suggested by Fries (1980) that “natural death” should predominate.

In addition to fitting the generalized Gompertz function to represent heterogeneity, we also fit a similarly modified form of the Weibull function. The generalized Weibull function actually described the data better. It is theoretically significant that the generalized Weibull function described the data better since the Weibull function describes

a process where death is generated by multiple, cause-specific death processes (Rosenberg et al. 1973; Economos 1982). The parameter describing the curvature of the Weibull function (i.e., m) also decreased across cohorts from the oldest to the youngest. The excellence of the fit of the generalized Weibull model is demonstrated in figure 3.

Cause-specific Mortality Trends from 1968 to 1980

Of course, a study restricted to total mortality is not useful in telling us how morbidity and mortality processes are linked. To examine the nature of this linkage, we will focus on two types of associations of specific diseases with the risk of death. The first association is with a specific disease identified as the underlying cause of death. This is the usual type of cause-specific mortality statistic reported and is sometimes used as the basis for ranking diseases in terms of their

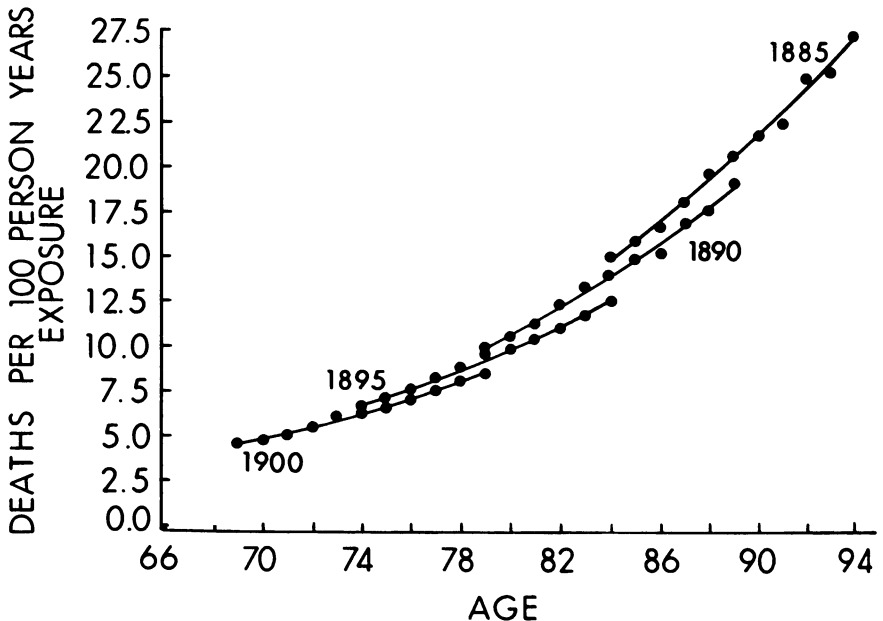


FIG. 3. The fit of the generalized (i.e., gamma mixed) Weibull hazard function to cohort Medicare population and mortality data. Mortality rates for the male birth cohorts of 1885, 1890, 1895, and 1900 are plotted as are the predicted values (continuous lines) from the model.

Source: Wilkin 1982.

public health importance. The underlying cause of death may not be the most appropriate statistic for examining the relation of diseases to the risk of death at advanced ages where the prevalence of multiple, possibly interacting, chronic diseases is high because there is, *by definition*, only one underlying cause coded from the death certificate. To describe mortality at such advanced ages we prefer to examine multiple-cause mortality data, i.e., data representing all medical conditions reported on the death certificate (Manton and Stallard 1984); the second association is with a specific disease identified anywhere on the death certificate.

In examining changes in cause-of-death patterns we will need procedures that control for changes in population structure—especially given the rapid recent growth of the elderly and oldest old populations. Thus, instead of simply examining the proportion of all deaths attributed to a given condition at a specific age, we will construct multiple-decrement life tables, i.e., life tables describing the age-specific survival experience of persons dying from the medical condition of interest (Preston, Keyfitz, and Schoen 1972). In such a life table, instead of ℓ_0 (the size of the initial life-table population) being an arbitrary prespecified number like 100,000 it is the proportion of deaths that can be expected at all ages in the life-table population from the selected condition. Thus, if 20 percent of all deaths were due to cancer, the multiple-decrement life-table radix at birth would be $0.20 \times 100,000$ —or 20,000. If by age 65, 25 percent of the cancer deaths had occurred (i.e., 5 percent or 0.25×0.20) then ℓ_{65} will be 15,000, suggesting that 15 percent of the total deaths in the population will be from cancer after age 65. We can compare these life-table statistics at specific ages over time to see how the proportion of deaths attributable to a given condition after a specific age changes. In the following analyses we focus on white male and female mortality since these groups predominate at ages 85 years and over. Also, because of space limitations, we will report statistics only from 1968 and 1980. We analyzed data for all years 1968 through 1980 and report significant trends in the text.

In our examination of cause-specific mortality we will examine variation in cause-specific mortality risks over two dimensions. The first is time. We know that major declines in the mortality from specific chronic diseases have occurred from 1968 onward. Thus, we will wish to evaluate how cause-of-death patterns have changed in

conjunction with those reductions—especially in terms of multiple-cause patterns at advanced ages. Second, we can meaningfully group causes according to different natural histories and modes of operation. Consequently, we examined three groups of selected causes of death: (1) complications of morbidity and debilitation (pneumonia, septicemia), (2) major age-related disease processes (cancer, stroke, heart disease), and (3) long-term initiating conditions (hip fracture, diabetes mellitus).

Complications of Morbidity and Debilitation. Septicemia and pneumonia/influenza are infectious disease processes that are primarily lethal for persons with impaired health and thus would be expected to be important causes of death for the very elderly (Besdine and Rose 1982). We present in table 6 parameters estimated from life tables for the underlying-cause (UC) and total-mention (TM) occurrence of septicemia and pneumonia/influenza.

The statistics in table 6 represent the percentage of deaths due to the specified condition that can be expected after a given birthday (exact age) in 1968 and 1980. Each entry is the ratio of the multiple-decrement survival probability for the disease to the probability of surviving to the given exact age, multiplied by 100.

For example, the value of 5.96 for female pneumonia/influenza deaths suggests that after age 85, 5.96 percent of deaths will be due to pneumonia/influenza. This is an increase of 45.4 percent over the proportion of all deaths (i.e., 4.1 percent) that can be expected to be due to pneumonia/influenza. Thus, on average, pneumonia and influenza deaths are more concentrated among the oldest old.

Septicemia is of particular interest since it has increased as an underlying cause of death from 2,285 deaths in 1968 (0.118 percent of all deaths) to 7,477 deaths by 1980 (0.375 percent of all deaths). This increase, moreover, occurred despite changes in the 9th revision of the International Classification of Diseases (ICD) where some of the deaths that had been previously assigned to septicemia were reassigned to diseases of infancy, leading to a jump in the mean age at death in the condition and a comparability ratio significantly less than 1.0 (i.e., 0.85 [National Center for Health Statistics 1980, 12]). This shift was also accompanied by a major increase in the mean age at death expected in the life-table population. For example, in 1968 the mean age at death from septicemia for white males (56.9 years) was much lower than the life expectancy (67.8 years). By 1978 the mean

TABLE 6
 Age-specific Contributions to Mortality of Complicating Conditions:
 Percentage of Deaths Beyond Stated Exact Age Due to (U.C.) or
 Associated with (T.M.) Pneumonia/Influenza and Septicemia
 in 1968 and 1980, U.S. White Population

AGE	1968		1980	
	U.C.	T.M.	U.C.	T.M.
PNEUMONIA AND INFLUENZA				
Females				
0	4.10	12.38	3.58	9.67
45	4.04	12.48	3.66	9.88
65	4.31	13.16	3.99	10.52
75	4.84	14.35	4.53	11.53
85	5.96	16.41	5.55	13.32
N=	27,929	84,832	24,573	69,736
Males				
0	3.64	12.09	2.96	9.31
45	3.63	12.47	3.11	9.81
65	4.12	13.92	3.63	11.03
75	4.92	15.76	4.57	12.88
85	6.46	18.51	6.28	15.65
N=	33,560	109,860	23,838	78,050
SEPTICEMIA				
Females				
0	0.12	1.08	0.48	2.88
45	0.10	1.01	0.48	2.88
65	0.09	0.93	0.49	2.83
75	0.09	0.85	0.50	2.76
85	0.07	0.69	0.49	2.55
N=	1,036	8,979	3,797	23,765
Males				
0	0.12	1.08	0.41	2.67
45	0.10	1.03	0.43	2.73
65	0.10	1.03	0.45	2.79
75	0.10	1.00	0.49	2.87
85	0.11	0.92	0.52	2.93
N=	1,249	10,655	3,680	24,262

Source: National Center for Health Statistics mortality data tapes.

age at death from septicemia had reached 66.5 years which, in 1979, jumped to 74.0 years. Thus, the change from the 8th to the 9th revision accentuated the role of septicemia as a cause of death at advanced ages. Independent of the effects of changes in the ICD revision, there was a major increase in the role of septicemia at advanced ages within the 8th revision tenure (1968 to 1978). For example, in 1968 septicemia accounted for 11.3 percent (0.118/0.106) more male mortality at all ages than after the 85th birthday. In 1978, it was 10.4 percent more important after exact age 85. By 1980, after the changes in the ICD revision, it was 20.5 percent more important as a cause of death after exact age 85.

Septicemia occurs very often as an associated cause of death and hence is greatly underreported in underlying-cause-of-death data. In 1968 it occurred almost nine times as often on the death certificate (19,634 times) as it was recorded as the underlying cause of death. By 1980 its total occurrence increased 2.4 times (to 48,027) but it was recorded relatively more often as an underlying cause. Consequently, in 1980 it was recorded only a little more than 6.4 times as often as it was recorded as an underlying cause (rather than the 8.6 to 1 ratio in 1968). This suggests that septicemia (1) was reported to be a far more important cause of death in 1980 than in 1968, (2) was reported as being relatively more lethal in 1980 than in 1968, (3) was viewed as increasingly important as a cause of death at advanced ages, and (4) that part, but probably not all, of the trend to become more significant at advanced ages was due to changes in the ICD revision.

One would expect pneumonia and influenza to behave in a similar fashion to septicemia, i.e., to increase in significance past age 85. The pattern is generally similar except that, though the comparability ratio is also fairly low (93 percent [National Center for Health Statistics 1980, 12]), there does not appear to be as large an effect in the shift from the 8th to the 9th revision. Furthermore, pneumonia/influenza is even more predominant as a cause of death at advanced ages—a pattern that existed in 1968 but increased by 1980. For example, the mean age at death for white males from pneumonia/influenza was 2.6 years higher than the life expectancy in 1968. By 1980 this difference had increased to 8.6 years with pneumonia/influenza being over twice as important as a cause of death above exact age 85 as at all ages.

In examining the total occurrence of pneumonia/influenza at death we see that over 12 percent of white male deaths in 1968 were associated with pneumonia/influenza—a figure that increased to 18.5 percent for deaths at age 85 and above. In contrast to septicemia, however, the significance of pneumonia/influenza in terms of total occurrence for white males dropped nearly 25 percent, i.e., it affected 9.3 percent of all deaths in 1980 and only 15.7 percent after exact age 85. Thus, overall, pneumonia/influenza has declined in significance as a complicating factor in mortality.

Since both pneumonia/influenza and septicemia are often lethal complications of other chronic, debilitating diseases, and because there are effects due to the change in ICD revisions for both diseases, it is useful to examine their total combined occurrence to see if the drop in pneumonia/influenza deaths is compensated for by the rise in septicemia deaths. In 1968 there were 214,326 deaths where either disease was reported. In 1980 this dropped to 195,813. Furthermore, if we examine the proportion of deaths affected by both diseases after exact age 85 we see little change. Thus, despite the rise in septicemia, there is little evidence of an increase in the combined impact of these complicating infectious diseases at advanced ages. This would run counter to the notion that increased survival has created a large population of extremely frail persons at ages 85 and over who are at high risk of such infectious processes.

Major Age-related Mortality Causes. Three diseases—heart disease, stroke, and cancer—account for roughly 70 percent of deaths in the life-table population. The underlying-cause and total-mention occurrence of these three diseases are recorded in table 7.

Cancer (solid tumors only) is of particular interest because it is one of the few major types of chronic disease whose death rates increased over the period 1968 to 1980. For white males, cancer deaths increased from 14.6 percent of all deaths in 1968 to 19 percent in 1980. Much of the increase is attributable to increased risks from one specific type—lung cancer. Similar increases were noted for white females for whom cancer deaths increased from 13.9 to 16.6 percent of all deaths.

In interpreting mortality trends for cancer it is important to remember that cancer is a term applied to a broad range of neoplastic diseases with very different characteristics and age-specific risks. Furthermore, the mix of cancer types is very different between males and females. In general, however, cancer is relatively less important as a cause of

TABLE 7
Major Age-related Mortality Causes: Percentage of Deaths Beyond Stated
Exact Age Due to (U.C.) or Associated with (T.M.) Cancer, Stroke,
and Heart Disease Death in 1968 and 1980, U.S. White Population

Exact Age	1968		1980	
	U.C.	T.M.	U.C.	T.M.
SOLID TUMORS				
Females				
0	13.87	15.74	16.60	19.05
45	13.87	15.81	16.64	19.16
65	11.05	13.06	13.59	16.15
75	8.58	10.57	10.47	12.99
85	5.77	7.62	7.14	9.55
N =	117,604	130,807	154,097	173,431
Males				
0	14.62	16.98	19.00	22.23
45	15.47	18.04	20.02	23.49
65	14.27	17.29	18.55	22.46
75	11.66	14.99	15.47	19.74
85	7.86	11.13	11.01	15.44
N =	138,569	159,227	179,985	207,353
HEART DISEASE				
Females				
0	42.00	52.92	42.59	60.45
45	44.01	55.23	44.10	62.19
65	46.51	58.14	46.65	64.98
75	47.59	59.30	48.58	66.98
85	48.29	59.45	50.38	68.56
N =	286,217	364,086	319,010	460,908
Males				
0	42.42	52.74	41.14	58.25
45	45.50	56.41	43.70	61.50
65	45.92	57.80	44.41	63.25
75	46.19	58.30	45.21	64.74
85	46.61	58.26	47.09	66.87
N =	387,382	481,301	365,429	518,013
STROKE				
Females				
0	15.18	22.70	12.16	19.73
45	15.85	23.66	12.56	20.38
65	17.18	25.59	13.55	22.01
75	18.61	27.33	14.79	23.89
85	19.43	27.55	15.71	25.05
N =	100,467	152,488	88,710	144,630

TABLE 7 (Continued)

Exact Age	1968		1980	
	U.C.	T.M.	U.C.	T.M.
STROKE				
Males				
0	9.64	15.03	7.28	12.52
45	10.42	16.16	7.77	13.36
65	12.40	19.13	8.94	15.40
75	14.67	22.21	10.50	17.82
85	16.49	23.79	11.71	19.43
N=	83,602	131,586	60,175	104,090

Source: National Center for Health Statistics mortality data tapes.

death at advanced ages; the proportion of female deaths due to cancer above exact age 85 is less than half of what it is at all ages.

It should be emphasized that, even though cancer death rates increased over this period, the mean age at death from cancer also increased paralleling the increase in the mean age at death for most major chronic diseases. Furthermore, even though cancer is often viewed as a highly lethal condition, it is reported 10 to 20 percent more often on the death certificate than it is recorded as an underlying cause of death—a pattern that remained relatively stable over time. This reporting of cancer as an associated cause of death is partly a function of the changing mixture of cancer types manifest over age. For example, above exact age 85 prostate cancer becomes highly prevalent for males in being reported on the death certificate nearly twice as often as it is recorded as the underlying cause of death. The decrease in the importance of cancer at advanced ages deserves attention since cancer is often viewed as a disease whose risks are age-determined. One possible explanation of the decline in cancer's significance at advanced ages is that the most highly susceptible portion of the population is removed by mortality selection. The loss of the most susceptible persons by advanced ages leads to a slower rise in cancer risks at those ages (e.g., Spiegelman 1969; Manton and Stallard 1982).

Heart disease mortality rates dropped significantly (21 percent for each sex) from 1968 to 1980. Despite the decline in mortality rates we see that, among white females, heart disease slightly increased in significance as an underlying cause of death (from 42.0 to 42.6

percent). Among white males, there was a slight decrease. For both sexes the proportion of deaths due to heart disease past exact age 85 increased from 1968 to 1980. This is because although heart disease mortality rates decreased, far more persons survived to advanced ages where heart disease was, relative to other causes of death, a more prominent mortality risk.

The occurrence of heart disease as a cause of death mentioned anywhere on the death certificate increased significantly from 1968 to 1980 at all ages (i.e., from 52.9 to 60.5 percent for white females and from 52.7 to 58.3 percent for white males). This is because there was a significant increase in the rate at which heart disease is reported as an associated condition perhaps reflecting the generally greater prevalence of chronic heart disease as a medical condition at death at later ages. This seems consistent with the medical evidence obtained from autopsy series of very elderly persons which showed high proportions with degenerative circulatory disease change (e.g., Jonsson and Hallgrimsson 1983).

Mortality rates for stroke have dropped dramatically over time (39 percent for white females and 41 percent for white males, from 1968 to 1980). Such reductions appear to have continued through to our most recent mortality-rate estimates (National Center for Health Statistics 1984). Except for hip fractures, stroke has the highest mean age at death recorded for any of the conditions we studied. Its importance as an underlying cause of death has declined more rapidly than its total occurrence on the death certificate, suggesting decreased lethality and higher prevalence at death.

For the two major circulatory diseases it is important to ascertain whether the mortality-rate declines resulted from reduced incidence or reduced case-fatality rates because of their different implications for health status and long-term care needs at advanced ages. Unfortunately, the evidence is not conclusive (Havlik and Feinleib 1979). There is evidence that declines in cardiovascular mortality had begun in California by 1960 (Borhani and Hechter 1964). Other evidence suggests that hypertension had begun declining before the dissemination of antihypertensive drugs (Kuller et al. 1979). There is evidence of stroke declines beginning in the 1940s (Miller and Kuller 1973).

It is probably safe to conclude that for stroke and heart disease, both incidence and case-fatality rates declined, but probably over different time periods and in different ways for the two diseases. For

stroke, long-term data on incidence are available, for example, from studies conducted at the Mayo Clinic in Rochester, Minnesota (Matsumoto, Whitsaut, and Kurland 1973). In those studies it appeared to certain investigators (e.g., Sherwin 1984) that initially the case-fatality rate for stroke was reduced—probably by better nursing management of chronically disabled stroke patients and better control of infectious processes through improved antibiotic therapy. This might explain reductions in stroke mortality rates before widespread dissemination of effective antihypertensive medications through national programs. Clearly, however, with the advent of the National High Blood Pressure program in 1972 we can document reductions in stroke incidence, e.g., of 15 percent between 1971 and 1976 (Robins and Baum 1981). It should be emphasized that even the data from the same sources have been interpreted differently by different investigators. Whisnant (1984) suggests that the treatment of hypertension may be the only significant contributor to the decline of stroke mortality. Hachinski (1984), in contrast, argues that a reduction in causes of death associated with stroke, such as heart disease, may account for the decline.

It is interesting to speculate if stroke mortality can continue to decline due to future improvements in the dissemination of hypertension control. Recent data for Maryland (Sherwin 1984) suggest that hypertension identification and control in that state had reached extremely high levels—especially for the elderly and for women. This would suggest that, though similar saturation may not yet have occurred in other states, future large declines in stroke mortality due to improved clinical control of hypertension may be more difficult to achieve through existing clinical practice—especially at advanced ages where the problem is apparently well recognized. There appears to be considerably more potential for improvement at younger ages. Alternately, future major reductions in stroke mortality might be achieved by recognizing that a large portion of stroke deaths occurs in persons with borderline hypertension—because, although the relative risk is low for borderline hypertension, the proportion of the population exposed to that risk is high (Sherwin 1984). Hence, the absolute number of *excess* stroke deaths produced by borderline hypertension may be quite high. Thus, further reduction may require better control of borderline hypertension through lifestyle and nutritional interventions rather than through drug management with its attendant risks.

An area of considerable recent interest in hypertension control among the elderly is that of isolated systolic hypertension (Minaker and Rowe 1985). We know, for example, that diastolic blood pressure in the population tends not to increase after late middle age. In contrast, systolic blood pressure tends to increase and is associated with elevated stroke risks. Currently studies are being conducted to determine if effective control of isolated systolic hypertension will, in fact, reduce stroke risks at advanced ages. Given the cause-of-death patterns just described, with the continuation of significant proportions of deaths at advanced ages being associated with stroke (and heart disease) this implies a large potential for further life-expectancy increases at advanced ages.

Long-term Initiating Conditions. In this section we describe two conditions that can produce mortality by causing long-run debilitation of the affected person. Diabetes mellitus operates over the long term to cause circulatory degeneration and is viewed as a risk factor for stroke and heart disease among others. Hip fracture operates over a shorter period of time and causes general debilitation, often leads to institutionalization, and can cause serious life-threatening, morbid events. The mortality data for these two diseases are reported in table 8.

The underlying-cause occurrence of diabetes mellitus above age 85 is stable over time, while the proportion of both male and female deaths due to diabetes actually decreased up to age 85. The total occurrence of diabetes mellitus at death is much greater, about four times, than its occurrence as an underlying cause of death. The total occurrence of diabetes has increased at all ages for males while increasing for females only after age 85. Female risks are about one third to one half higher than for males. Diabetes is a much more serious problem for black than for white females. The lower prevalence at death of diabetes mellitus at advanced ages is likely due to selection, i.e., the lower life expectancy of diabetics than of the general population.

In table 8, there are no underlying-cause statistics reported for hip fracture. This is a result of the way underlying causes of death are assigned for accidents. In these cases the external cause (E-code), rather than the nature of injury (N-code), is reported as the underlying cause. Thus, only in the multiple-cause-of-death data can one determine the national significance of hip fracture as a cause of death.

Among white males, hip fracture is predominantly a disease of

TABLE 8
 Conditions Initiating Long-term Degeneration and Potentially Lethal
 Processes: Percentage of Deaths Beyond Stated Exact Age Due to
 (U.C.) or Associated with (T.M.) Hip Fracture and Diabetes
 Mellitus, 1968 and 1980, U.S. White Population

Exact Age	1968		1980	
	U.C.	T.M.	U.C.	T.M.
HIP FRACTURE				
Females				
0		2.21		1.49
45		2.33		1.55
65		2.66		1.74
75		3.19		2.04
85		4.05		2.53
N =		12,895		9,830
Males				
0		0.81		0.68
45		0.88		0.73
65		1.15		0.90
75		1.63		1.23
85		2.57		1.94
N =		6,514		4,982
DIABETES MELLITUS				
Females				
0	2.42	8.32	1.97	7.80
45	2.49	8.67	2.00	8.04
65	2.45	8.75	1.96	8.05
75	2.09	7.50	1.76	7.34
85	1.30	4.76	1.37	5.53
N =	18,793	63,371	16,750	65,437
Males				
0	1.48	5.49	1.32	5.76
45	1.54	5.89	1.37	6.12
65	1.56	6.15	1.38	6.27
75	1.46	5.65	1.32	5.91
85	1.13	4.17	1.12	4.78
N =	13,744	50,372	12,136	52,220

Source: National Center for Health Statistics mortality data tapes.

advanced old age having a mean age at death of 81.5 years in 1968 and 84.1 years in 1980. This is reflected in the age-specific proportion of deaths affected by hip fracture, being only 0.7 percent of all mortality in 1980 but 2 percent of mortality past exact age 85. The decline in male (and female) hip fracture mortality was evident after 1975. The female mortality rates of hip fracture are higher than for males being, in 1980, 1.5 percent of all deaths—rising to 2.5 percent past exact age 85 (4 percent in 1968). As for males the mean age at death is high—85.2 years in 1968, and increasing to 86.9 years in 1980. The decline in the number of deaths where hip fractures are reported may indicate that an improvement occurred in the mortality rates for the chronic conditions with which hip fractures are associated. Promising treatments for osteoporosis, the degenerative process underlying the risk of hip fracture, are a relatively new phenomenon. Consequently, improved clinical management of osteoporosis is unlikely to have yet had an impact on hip fracture incidence or mortality. Associated with the 15,410 deaths with hip fracture reported in 1980 were an estimated 200,000 incident cases of hip fracture with a mean age of 79 years (Brody 1985). Under current incidence rates, the number of hip fractures in 2000 is projected to reach 330,600. By 2050, this number is projected to reach 650,000, with 340,000 over age 85 (Brody 1985).

Overall Changes in the Cause-of-death Pattern at Advanced Ages. The seven causes of death described above accounted for 80 percent of deaths past age 85 in 1980. Thus, they describe the majority of changes in mortality conditions over age 85. Though there were clear trends in the relative significance of the seven causes past age 85 the overall impression is that the mix of causes of death at 85 was relatively stable from 1968 to 1980. An important change affecting all causes was a two- to three-year increase in the mean age at death for the major causes of death. Furthermore, it is notable that several of the conditions for which we have identified major risk factors at earlier ages (e.g., cancer, stroke) remain important at advanced ages, at least suggesting the possibility of further mortality reduction at advanced ages through risk-factor intervention. Also important in interpreting mortality patterns was the potential significance of dependent competing risks. That is, for several diseases mortality rates appeared to have declined because of an association with another chronic condition whose mortality rate had declined (Hachinski 1984). Given the high

prevalence of multiple chronic conditions at advanced ages such cause dependency is potentially a very important factor in mortality changes.

It should be noted that certain very important morbid states are not reflected in the mortality statistics. For the population 85 years of age and over the most important such condition is Alzheimer's disease—a condition which Katzman (1976) estimated might affect 90,000 deaths in 1970 (and current estimates suggest that 120,000 deaths may be affected in 1980). Brody (1985) estimates that there were 2 million people with Alzheimer's disease or related disorders in 1980, with a mean age of about 80 years. The prevalence of the disease past age 85 is estimated to be at least 20 percent. By 2000 the number of persons affected is projected to be 3.8 million; by 2050 the projection is 8.5 million, of whom nearly 5 million will be over 85 (Brody 1985).

Morbidity at Advanced Ages

In the preceding section we studied the association of specific diseases with mortality using cause-specific mortality data and examining its change over time. A different type of data is available from longitudinally followed populations. In such longitudinal studies it is possible to characterize more fully the physiological aging and morbidity changes within elderly individuals. One type of longitudinal study is designed to examine the risk factors associated with specific chronic diseases (e.g., Framingham, Evans County [Kessler and Levin 1970]). A second type of longitudinal study was designed specifically to describe individual aging changes. A classic study of this type is the first Duke Longitudinal Study of Aging (1955 to 1976) which involved the application of an extremely broad range of physiological, clinical, and psychological instruments to a relatively small ($N = 267$) but extremely elderly (mean age at study entry was 71.3 years) population at eleven times over a 21-year period. The purpose of the study was to describe normal aging changes and to discriminate between those changes and changes due to explicit age-related pathology (Palmore 1970; 1974). A number of other longitudinal studies of aging are available (e.g., the study population followed at National Institute on Aging Gerontological Research Center in Baltimore) which we did not review. However, the Duke Study should serve to illustrate certain important basic principles about health changes at advanced ages.

In analyzing these and other data we employed a multivariate analytic strategy called "grade of membership" (GOM) analysis. The aim of the GOM analysis is to identify subpopulations that have similar physiological characteristics, similar demographic attributes, and similar responses to physical measurements as they relate to morbidity (Woodbury and Manton 1982; Clive, Woodbury, and Siegler 1983). The subpopulations are described by two types of parameters. The first type represents the probabilities that persons in a given subpopulation have a particular attribute or quality. The probabilities for each of five subpopulations defined in this way are presented in table 9. These five states can be examined to determine if they describe the trajectory of aging changes in individuals but without requiring that the total study population follow a specific distribution, or that the trajectory of change is of a specific parametric form. The second type of parameter represents how well individuals are described by each of the typical characteristics of the analytically identified subpopulations. Hence they represent individual differences not captured by the multivariate descriptions of the subpopulations. In the current analysis, we were less interested in the population distribution over these states than in the description of the aging trajectories implied by the distribution of attributes within each subpopulation. Hence, we did not discuss the individual-level parameters inasmuch as, for our current purposes, they were "nuisance" parameters whose primary function was to ensure that the subpopulation definitions were not confounded with distributional artifacts.

In table 9 the variables (e.g., pulse pressure), and the response intervals (e.g., pulse pressure less than 50) are described on the left, the proportions of the sample with specific attributes are described in column 2, and the next five columns describe the proportions that can be expected to have a particular attribute in each of the five empirically generated subgroups. We can see that the first four groups are roughly ordered in terms of decreasing health with the fifth group having manifest morbid changes. We also see that not only are there consistent patterns in terms of physical attributes, but that the last two groups have significantly poorer intellectual performance.

Given these groups defined on physical health and intellectual function, it is important to assess their characteristics in terms of age, survival time, and social status. These three sets of variables were *not* used to form the groups. Thus, they represent information that is independent of the group definitions. The first four groups are

TABLE 9
The Probability of Five Analytically Defined States ($K = 5$) Having Specific Physiological Characteristics

Variable	Sample proportion	1	2	3	4	5
PULSE PRESSURE						
<50	0.135	0.324	0.000	0.000	0.000	0.303
50-60	0.173	0.427	0.000	0.000	0.142	0.184
61-70	0.181	0.222	0.206	0.100	0.398	0.000
71-80	0.187	0.000	0.794	0.000	0.000	0.514
81-90	0.123	0.027	0.000	0.220	0.459	0.000
90+	0.201	0.000	0.000	0.680	0.000	0.000
DIASTOLIC BLOOD PRESSURE						
<86	0.134	0.414	0.000	0.000	0.000	0.000
86-95	0.610	0.586	1.000	0.000	1.000	0.000
96-115	0.208	0.000	0.000	1.000	0.000	0.550
116+	0.049	0.000	0.000	0.000	0.000	0.450
INFARCTION (EKG)						
Yes	0.142	0.000	0.000	0.000	0.000	1.000
INJURY (EKG)						
Yes	0.022	0.000	0.000	0.000	0.000	1.000
ISCHEMIA (EKG)						
Yes	0.243	0.000	0.000	0.000	0.000	1.000
ARTERIOSCLEROTIC ETIOLOGY						
Yes	0.486	0.000	0.000	1.000	0.000	1.000
HYPERTENSIVE ETIOLOGY						
Yes	0.294	0.000	0.000	1.000	0.000	1.000
RHEUMATIC ETIOLOGY						
Yes	0.068	0.000	0.000	1.000	0.000	0.000
CHOLESTEROL						
<206	0.246	0.000	0.692	0.000	0.804	0.000
206-255	0.410	0.602	0.308	0.396	0.196	0.311
256-305	0.270	0.319	0.000	0.440	0.000	0.610
306+	0.074	0.080	0.000	0.165	0.000	0.079
HEMATOCRIT						
<40	0.253	0.000	0.000	0.557	0.659	0.000
40-41	0.199	0.372	0.000	0.250	0.000	0.402
42-45	0.386	0.439	0.546	0.194	0.341	0.425
46+	0.161	0.189	0.454	0.000	0.000	0.173

Table 9 (continued)

Variable		Sample proportion	1	2	3	4	5
ARTERIOSCLEROTIC RETINOPATHY							
None	0.162	0.000	0.999	0.000	0.000	0.000	0.000
Grade 1	0.527	0.997	0.000	0.999	0.000	0.000	0.000
Grade 2	0.286	0.000	0.000	0.000	0.942	0.893	
Grade 3	0.025	0.000	0.000	0.000	0.057	0.107	
Grade 4	0.001	0.003	0.001	0.001	0.001	0.000	
HYPERTENSIVE RETINOPATHY							
None	0.625	0.682	1.000	0.556	0.537	0.000	
Grade 1	0.263	0.318	0.000	0.444	0.463	0.000	
Grade 2	0.097	0.000	0.000	0.000	0.000	0.870	
Grade 3	0.015	0.000	0.000	0.000	0.000	0.130	
OVERALL CARDIOVASCULAR FUNCTIONAL STATUS							
No disease	0.546	1.000	1.000	0.000	0.000	0.000	
Definite dis- ease not limiting	0.280	0.000	0.000	0.693	0.000	0.512	
Mod. to se- vere disease	0.174	0.000	0.000	0.307	1.000	0.488	
TOBACCO USE							
Yes	0.253	0.000	1.000	0.000	1.000	0.000	
SUGAR							
Absent	0.965	1.000	0.963	0.918	0.965	0.961	
Trace	0.006	0.000	0.012	0.000	0.000	0.039	
1+	0.010	0.000	0.025	0.000	0.035	0.000	
2+	0.019	0.000	0.000	0.082	0.000	0.000	
ALBUMIN							
Absent	0.877	1.000	0.924	1.000	1.000	0.230	
Trace	0.105	0.000	0.000	0.000	0.000	0.770	
1+	0.012	0.000	0.076	0.000	0.000	0.000	
2+	0.006	0.000	0.000	0.000	0.000	0.000	
SUBJECTIVE HEALTH							
Very poor	0.029	0.000	0.000	0.000	0.000	0.311	
Poor	0.073	0.000	0.000	0.000	0.371	0.000	
Fair for my age	0.037	0.000	0.000	0.070	0.124	0.000	
Fair	0.202	0.011	0.215	0.000	0.506	0.569	
Good	0.280	0.314	0.366	0.560	0.000	0.000	
Good for my age	0.181	0.363	0.000	0.370	0.000	0.000	
Excellent	0.108	0.312	0.000	0.000	0.000	0.120	
Excellent for my age	0.090	0.000	0.420	0.000	0.000	0.000	

Table 9 (continued)

Variable		Sample proportion	1	2	3	4	5
VERBAL SCALED SCORES							
<31	0.114	0.000	0.000	0.000	0.251	1.000	
31-50	0.296	0.000	0.000	0.000	0.749	0.000	
51-70	0.295	0.000	1.000	0.000	0.000	0.000	
71-90	0.253	0.798	0.000	1.000	0.000	0.000	
91+	0.042	0.202	0.000	0.000	0.000	0.000	
PERFORMANCE SCALED SCORES							
<26	0.412	0.000	0.000	0.000	1.000	1.000	
26-40	0.410	0.421	1.000	0.643	0.000	0.000	
41-55	0.165	0.521	0.000	0.358	0.000	0.000	
56-70	0.014	0.058	0.000	0.000	0.000	0.000	
HEIGHT (INCHES)							
<60	0.094	0.000	0.000	0.452	0.077	0.000	
60-62	0.253	0.000	0.000	0.000	0.923	0.000	
63-65	0.273	0.610	0.000	0.548	0.000	0.340	
66-68	0.221	0.390	0.243	0.000	0.000	0.660	
69+	0.160	0.000	0.757	0.000	0.000	0.000	
OBESITY							
2	0.053	0.000	0.000	0.000	0.000	0.375	
3	0.672	0.770	0.557	0.804	1.000	0.000	
4	0.237	0.230	0.443	0.083	0.000	0.519	
5	0.038	0.000	0.000	0.113	0.000	0.105	

Source: Duke Longitudinal Study data files.

ordered in terms of an increasing average age (i.e., 75.0, 75.6, 79.3, 83.2 for groups 1 to 4). The last group has the lowest expected age (74.8). The first four groups appear to describe the gradual physiological decline with age for the same set of persons followed over the course of the study. The fifth group appears to describe a different set of persons who entered the study physiologically impaired. Though the fourth group is the oldest (83.2) it has fewer explicit pathological changes than the fifth and youngest group. We also find that the fifth group has the shortest expected survival time. Thus, it appears to be an initially morbid group who died out of the study population fairly rapidly.

It is interesting to examine the association of these groups with socioeconomic status and race. What is intriguing is that both the oldest group and the young, physically impaired group are of lower socioeconomic status and proportionately more likely to be black.

These patterns confirm at the individual level two types of mortality selection processes that have been argued to occur at advanced ages in vital statistics data (Manton, Stallard, and Vaupel 1985). Specifically, the existence of a relatively young, morbid subgroup that dies out rapidly (i.e., before age 80) is consistent with models that describe mortality patterns at advanced ages (aged 85 and older) as a result of the rapid mortality selection, or exhaustion, of impaired lives. It should be noted that the impairment may either be intrinsic (e.g., genetic) or acquired. Both types of impairment will produce the same population mortality patterns if (1) they are permanent and (2) mortality is analyzed after the age at which the impairment is acquired. This observation is also consistent with the concept that the population aged 85 years and over is a highly selected group of survivors with special attributes (Riley and Bond 1983). The fact that both the oldest and most morbid groups have high proportions of blacks is consistent with explanations of black-white mortality crossovers or convergences (Manton 1980) as due to adverse mortality selection of socioeconomically deprived persons—an observation also made in the Evans County study (Wing et al. 1985).

The significance of these findings for our conceptual model is that they describe, using a very flexible procedure (i.e., making no assumptions about the distribution of the sample or the form of the hazard rate), both heterogeneity in aging processes and its effects on differential survival chances; and the longitudinal association of morbidity and mortality as a process on the individual level.

One of the limitations of this analysis is the smallness of the data set and hence the stability of the estimates. This is a general problem in that such longitudinally followed study populations do tend to be small. It should be remembered, however, that our task here was not to try to estimate age-specific morbidity rates but to look at broad features of an individual-level process the mechanisms of which determine the transitions in the population-level model. We also found, empirically, that the basic nature of the five groups was not altered by small changes in the set of variables. Furthermore, when the number of subpopulations that the analysis was asked to identify was changed

the multivariate descriptions of the new subpopulations tended to represent aggregations or decompositions of the descriptions presented in table 9. The stability of the analysis seems to be a result of the adjustment of the patterns for distributional effects effected by the individual-level parameters.

Disability

The Noninstitutional Population

The one component of the conceptual model we have not yet discussed in detail is disability. In figure 1 we assumed that disability results from the progression of chronic, degenerative disease. This concept of a process is explicit in the World Health Organization's (1980) model of impairments, disabilities, and handicaps, the origin of which can be examined in Susser (1973). Since these concepts are basic to efforts to generate functional assessments using standard terminology it is useful briefly to compare these concepts with those in the standard medical-epidemiological model (table 10).

The impairments are the result of pathological changes in the organism's physiology. Disabilities represent functional limitations in the organism's ability to perform basic self-care and other functions (e.g., eating, bathing, toileting), while handicaps refer to defects in the ability to perform certain social functions. In this analysis we will assume that disabilities can be measured by Katz's (Katz and Akpom 1976) "activities of daily living" (ADL) and the "instrumental activities of daily living" (IADL), detailed in table 14, part B.

The disablement process described above is generally accepted in geriatric medicine. However, it is often argued that there is not necessarily a direct mapping of the degree and type of functional disability with the identity of the underlying morbid process (Besdine 1984). Indeed, it is suggested that, at advanced ages, it is more typical for chronic disease to be manifest in terms of functional limitations than in terms of the more usual symptomatology manifest for the same disease in younger patients (Besdine 1984). As a consequence of a lack of a strong correlation between specific medical problems and functional limitations, we will examine the distribution of disability within the population separately from specific types of morbidity.

TABLE 10
 A Comparison of the Logic of the Medical/Epidemiological
 Model of Disease and the Extension of the Model
 for Assessing Disease Consequences

-
1. Medical/Epidemiological Model
 Etiology → Pathology → Manifestation
 2. Extension of Medical Model for Disease Consequences
 Disease → Impairment → Disability → Handicap
 where

Impairments are "concerned with abnormalities of body structure and appearance and with organ or system function, resulting from any cause; in principle, impairments represent disturbances at the organ level."

Disabilities are "reflecting the consequences of impairments in terms of functional performance and activity by the individual; disabilities thus represent disturbances at the level of the person."

Handicaps are "concerned with the disadvantages experienced by the individual as a result of impairments and disabilities; handicaps thus reflect interaction with and adaptation to the individual's surroundings."

Source: World Health Organization 1980, 14.

Projections of Disability in the Community. A recent survey that provides detailed information on disability in the noninstitutional population is the National Long-Term Care Survey (NLTCs), conducted by the Health Care Financing Administration (HCFA) in 1982. In this survey a telephone screen of 36,000 persons drawn from the HCFA's Health Insurance Master File was conducted. This screen yielded 6,393 noninstitutionalized persons over age 65 who reported limitations in either "instrumental activities of daily living" (IADL) or of "activities of daily living" (ADL). Upon completion of a household interview, 87 percent (or 5,580 persons) were identified as having such limitations. These 5,580 persons represent roughly 4.6 million persons over age 65 and in the community, who had IADL or ADL limitation. Approximately 1,050 of the respondents were over age 85 representing 818,000 persons of whom 654,000 (or 80 percent) were female. As a result of the relatively small numbers of persons aged 85 and over in the survey, certain of the rate estimates will be highly

variable—especially as the population aged over 85 is decomposed. Hence, certain of the following estimates and projections, though they provide potentially unique information on disability in the oldest old, should be interpreted with caution.

To use the information on disability from the survey, we decided to construct a scale representing degree of disability. Since the ADL limitations are based upon a sociobiological model of development (Katz and Akpom 1976), they are viewed as hierarchically ordered so that they may be summed into a simple scale ranging from 1 (least disabled) to 6 (most disabled). We divided the ADL score into 3 groups (i.e., 1 to 2 limitations; 3 to 4 limitations; 5 to 6 limitations). Persons with an IADL limitation but no ADL limitation were put into a separate category. To understand the pattern of increase of functional limitation with age we present, separately for males and females, age-specific disability levels adjusted to reflect the proportion expected to survive to age x based upon the period life tables prepared for 1980 by Social Security actuaries.

An examination of the two sets of curves shows that both the prevalence of disability at all levels and the rates of institutionalization are lower at most ages for males than for females. For example, of persons who survive to age 90, 63 percent of males either report disability or are in nursing homes compared to 71 percent for females. The increased prevalence of disability among females up to age 90 is correlated with the greater survival of females. The growth of the risk of disability with age is clearly illustrated in the figures, with 24 percent of men and 35 percent of women who are either in institutions or have three or more ADL limitations by ages 85 to 89. It should be noted, however, that the proportion of the community population of females over age 85 who report disability does not increase. Indeed, the proportion of *noninstitutionalized* females disabled at any level of disability actually drops from 38 percent at ages 85 to 89 to 34 percent over age 90. However, if one adds in the proportion of females in nursing homes at these ages (i.e., 22 percent at ages 85 to 89 versus 37 percent above age 90), the proportion then increases (from 60 percent to 71 percent). Since females predominate at these later ages this suggests that the proportion of the survivors in this very old noninstitutional population which remains functionally capable is decreasing but even above age 90 is sizeable (i.e., 29 percent).

Thus, the apparent stability of disability rates in the community

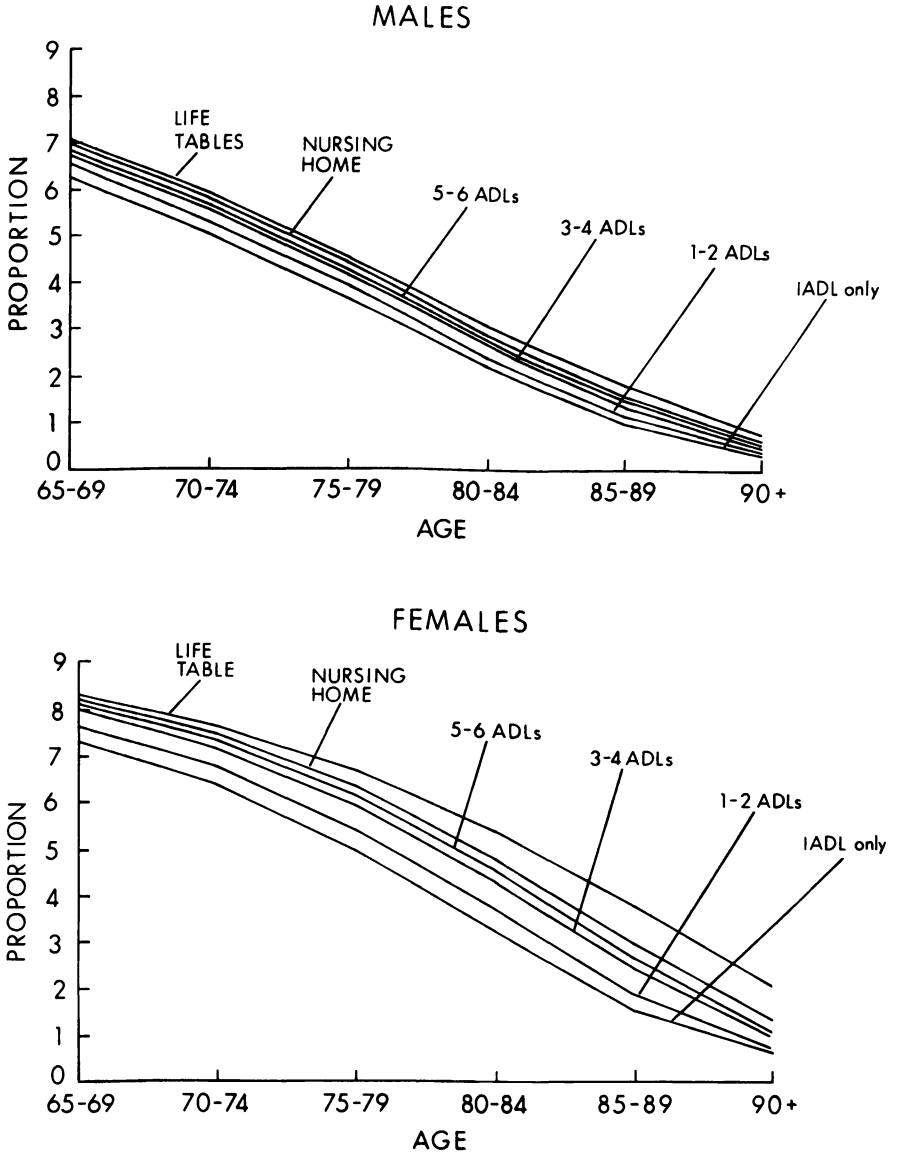


FIG. 4. The observed mortality and hypothetical morbidity, disability, and long-term care service use survival curves for U.S. males and females, 1980. Sources: NNHS data tapes; U.S. Census Bureau, *Current Population Reports* P25, no. 917, table 2, 1982; NLTCs data tapes; Social Security Administration 1982.

at later ages is due to the fact that mortality and institutionalization have selected out highly disabled persons. As a consequence of the selection process, noninstitutional female survivors to ages 90 and over may be members of a highly select, very special subpopulation

who do not experience the same degenerative processes at the same rates as experienced by persons who died at younger ages. It is interesting to speculate whether, for younger cohorts for whom higher proportions can expect to survive to advanced ages, the increasing heterogeneity at advanced ages will cause the prevalence of disability in the community to increase or if the proportion of the population which is institutionalized will increase. In this regard it is interesting to note that, while female survival at advanced ages increased, the proportion of the population over age 85 in nursing homes decreased substantially (i.e., by 15 percent, from 253.7 to 216.4 [table 16]) between 1973 and 1977. To select between the two possibilities cited above it will be necessary to wait for the 1984 round of the NLTCs where the mortality and institutionalization risks of individuals surveyed in 1982 will be assessed.

In table 11 we present projections of the noninstitutionalized disabled population, a population directly associated with need for "long-term care" (LTC) services. The projections are of the numbers of persons, specific to marital status, age, and sex, at the four disability levels from 1980 to 2040—the year by which the post-World War II baby-boom cohort reaches age 85.

These projections are of the simple, static-component type where we assumed that the age, sex, marital status, and disability-specific rates estimated from the NLTCs for the noninstitutionalized elderly population were constant over time. These rates were applied to population projections prepared by the Social Security actuaries (adjusted to United States resident census level) after the population had had subtracted from it an estimate of the institutional population based upon rates estimated from the 1977 National Nursing Home Survey.

In table 11 the rapid growth of the disabled elderly population is evident. In the year 2000 we can expect 2.4, 1.0, 1.3, and 2.0 million persons at ADL levels 1 to 2, 3 to 4, 5 to 6, and with an IADL limitation only. By the year 2040 these numbers increase dramatically so that there are 13.1 million persons in the community with at least an IADL limitation (4.6 million with 3 or more ADL limitations). These projections are based on the assumption that the current rate of institutionalization can be maintained—an assumption that many would question, though the growth of the numbers of nursing home beds needed to maintain the 1977 utilization rates is only 2.1 percent per annum compared to the 6 percent growth rate observed from 1969 to 1977. If it cannot, nursing home bed constraints (perhaps aggravated by the effects of diagnosis-related group (DRG)

TABLE 11
 Projections of the Noninstitutionalized Long-term Care Population and Institutionalized Population by Age, Sex, Marital Status, and Disability Level, 1980-2040 (numbers in thousands)

Year	Noninstitutionalized						Noninstitutionalized						Total*	Nursing home	Total*
	IADL			Disability level			IADL			Disability level					
	1-2 ADL	3-4 ADL	5-6 ADL	1-2 ADL	3-4 ADL	5-6 ADL	1-2 ADL	3-4 ADL	5-6 ADL	1-2 ADL	3-4 ADL	5-6 ADL			
MARRIED MALES															
Age 65-74															
1980	217	167	79	79	136		143	170	76	85	20		20	492	
2000	256	197	94	161			175	207	93	104	25		25	604	
2040	399	308	146	250			288	343	153	171	41		41	996	
Age 75-84															
1980	133	114	49	89			76	73	47	52	29		29	277	
2000	230	198	84	154			150	145	94	102	58		58	549	
2040	452	389	165	303			310	299	193	211	120		120	1,133	
Age 85+															
1980	19	46	28	25			11	14	9	12	14		14	60	
2000	42	98	60	53			20	24	16	21	24		24	106	
2040	112	263	162	143			57	68	46	59	68		68	298	
MARRIED FEMALES															
UNMARRIED MALES															
UNMARRIED FEMALES															
Age 65-74															
1980	59	56	30	20			260	231	86	75	118		118	771	
2000	79	74	39	27			277	247	92	80	126		126	822	
2040	153	144	76	52			413	368	137	119	188		188	1,225	

Age 75-84												
1980	53	65	16	30	90	254	283	365	138	137	339	1,261
2000	82	101	25	47	139	393	416	536	202	201	498	1,853
2040	194	239	59	111	331	933	755	972	367	364	903	3,361
Age 85 +												
1980	39	47	18	19	77	201	111	204	85	130	407	937
2000	67	80	31	33	132	343	253	463	193	294	924	2,127
2040	176	212	82	87	347	904	585	1,069	445	680	2,133	4,912

Source: Unpublished tabulation of National Long-Term Care Survey and 1977 NNHS.

* Totals may reflect rounding errors.

reimbursement which may force the earlier discharge of acutely ill patients to nursing facilities) are likely to cause the number of non-institutionalized elderly at the higher levels of disability to be even greater.

The above projections describe the potential population in need of long-term care (LTC) services. They clearly show that the greatest growth in the need for both community and institutional LTC services is for unmarried females 75 years of age and over. We see that the largest absolute and relative increases are for unmarried females over age 85. It is interesting that married males represent a larger population than do married females.

Future demographic factors (i.e., changes in family size and composition, changes in probability of survival of spouse) and policy will affect the actual level or type of LTC services available. However, it is useful to examine briefly the nature and mix of services currently received by persons at different disability levels and ages (Manton and Liu 1984b). In table 12 we present the total number of excess hours spent per week in providing informal care by different classes of caregivers for different disability levels for the population aged 85 and over.

As we can see, the sources of informal care are quite different for different marital statuses and for males and females. We see that males are much more dependent on the spouse for informal care and that females receive more care from offspring and relatives. The importance of the spouse as the source of care decreases with age—reflecting the decreasing likelihood of both members of a couple surviving to advanced ages. To illustrate the level of care on an individual level from table 12, we see that 83.532 million excess hours of care are delivered weekly to 8.472 million females—an average of 9.85 hours per week. With age the likelihood of formal care services increases—as it does with increasing disability. This probably reflects the greater demand for nursing services for the more seriously disabled person.

One subgroup of particular interest is the 1.1 million persons reporting that they received some formal care. This group is likely to increase in size because of (1) the increased survival of the oldest old, (2) the possible decrease of informal care resources with changing family patterns, and (3) possible future constraints on institutionalization causing more severely disabled persons to remain in the community. Of the 1.1 million persons who reported receiving some formal care

TABLE 12
 Projections of Total Excess Hours per Week (in thousands) Spent in Providing Informal Care for Disabled Elderly, Age 85 +,
 by Disability Level, Sex, and Marital Status

Year	MARRIED MALES			MARRIED FEMALES			Total*		
	IADL	1-2 ADL	3-4 ADL	5-6 ADL	IADL	1-2 ADL		3-4 ADL	5-6 ADL
Spouse is helper									
1980	377	991	705	630	117	183	227	141	668
2000	808	2,124	1,513	1,351	207	323	401	250	1,180
2040	2,176	5,719	4,072	3,636	581	908	1,127	702	3,318
Offspring is helper									
1980	34	232	189	354	163	104	83	236	586
2000	73	497	405	760	287	184	147	416	1,034
2040	197	1,339	1,091	2,046	808	517	413	1,170	2,908
Relative is helper									
1980	36	94	93	53	28	103	57	157	345
2000	78	203	199	114	49	182	100	277	609
2040	210	545	536	308	139	513	282	780	1,714
Nonrelative is helper									
1980	29	88	64	60	27	27	42	61	157
2000	63	189	137	129	48	48	73	108	278
2040	169	509	368	347	135	135	206	304	781

TABLE 12 (continued)

Year	IADL	UNMARRIED MALES			Total*	UNMARRIED FEMALES			Total*	
		1-2 ADL	3-4 ADL	5-6 ADL		1-2 ADL	3-4 ADL	5-6 ADL		
Offspring is helper										
1980	482	573	370	226	1,651	1,317	2,425	1,341	2,837	7,921
2000	819	973	629	385	2,805	2,990	5,508	3,045	6,443	17,986
2040	2,161	2,569	1,660	1,016	7,406	6,906	12,719	7,033	14,881	41,539
Relative is helper										
1980	384	338	203	202	1,128	592	1,383	745	1,383	4,103
2000	653	575	346	343	1,917	1,344	3,140	1,692	3,140	9,316
2040	1,724	1,518	912	905	5,060	3,103	7,253	3,909	7,251	21,516
Nonrelative is helper										
1980	95	217	86	203	601	247	718	397	881	2,242
2000	162	368	146	344	1,021	560	1,630	901	2,000	5,090
2040	427	973	386	909	2,695	1,293	3,764	2,081	4,619	11,756

Source: National Long-Term Care Survey and NORC Survey of Caregivers.

* Totals may reflect rounding errors.

in the 1982 NLTCS we know that 605,000 reported providing some portion of the expenditure for formal care from their own resources. These persons reported spending \$99.2 million per month on such services. Of the 605,000, about 465,000, or 77 percent, report paying for all of their formal care. This group reports paying \$65.5 million monthly or \$141 per person for care. In table 13, we see how these reported expenditures break down by age and disability level.

In table 13 we see that monthly out-of-pocket expenses reported for persons who use formal care increases from \$108 for persons aged 65 to 74 to \$193 for persons aged 85 and older. Much of this increase is due to the higher disability levels of the population aged over 85. We see that monthly expenses increased from \$82 to \$429 with disability level. Nonetheless, for persons aged 85 and older at the highest and next to highest disability levels, expenses are greater than for the average for all ages combined. If similar levels of expenditures were to be maintained (in constant 1980 dollars) this would imply

TABLE 13
Monthly Out-of-pocket Expenditures on LTC Services for Disabled Persons
Who Pay for All of Their Care

	Number of persons	Average expenditure
AGE		
65-74	166,657	\$108
75-84	197,737	\$142
85+	100,450	\$193
Total	464,844	
DISABILITY LEVEL		
IADL only	133,885	\$ 82
1-2 ADLs	180,990	\$ 70
3-4 ADLs	70,857	\$111
5-6 ADLs	79,112	\$429
85+		
IADL only	19,671	\$ 65
1-2 ADLs	34,200	\$ 66
3-4 ADLs	19,429	\$163
5-6 ADLs	27,150	\$466

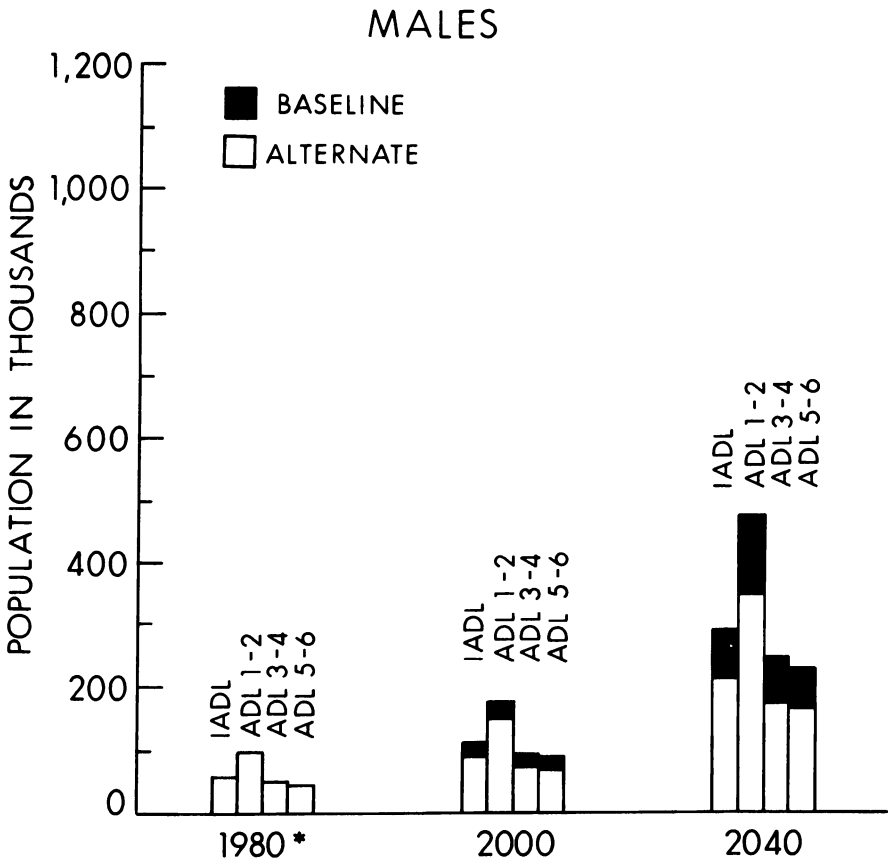
Source: National Long-Term Care Survey.

roughly \$107 million of out-of-pocket expenditures monthly for formal care in 2000 (an increase of 63 percent over 1982) and \$222 million in 2040 (an increase of 239 percent over 1982) with 41 percent of those expenditures being made for persons 85 and older in 2000 and 48 percent in 2040.

In some ways the above projections are a “worst case” scenario because we have assumed that age-specific disability rates remained constant, i.e., that the projected increase in life expectancy was not associated with changes in the disability rates. In effect this assumes that changes in the morbidity and disability curves in figure 1 are unrelated to changes in the mortality curve. It is also important to evaluate the implications of varying this assumption—e.g., of assuming that the projected improvement in life expectancy is a result of improvement in underlying health. For example, if we accept that recent reductions in stroke mortality (e.g., post 1972) are a result of reduced incidence due to improved control of hypertension, then this should also lead to a reduction of stroke-related disability in addition to the decrease in stroke mortality. The effect of a strong linkage between mortality reductions and disability rates is portrayed separately for males and females over age 85 in figure 5.

In figure 5 we have assumed that disability rates were reduced, on an age- and sex-specific basis, proportional to the projected mortality-rate declines utilized in the Social Security Administration (SSA) (1981) projections. In these projections there are large declines in the numbers of persons expected to be disabled, i.e., 17 and 23 percent reduction for males and females aged 85 and older in 2000, and 27 and 34 percent reductions in male and female disability rates in 2040. This translates, for example, into reductions of 318,000, 237,000, 519,000, and 289,000 persons aged 85 and older at disability levels 5 to 6, 3 to 4, 1 to 2, and IADL-only limitations. These projections show that, if the morbidity and disability curves move jointly with the survival curve, there would be much smaller increases in the disabled population. The most probable scenario is that there will be some improvement in health at advanced ages but not a perfect correlation between changes in the different curves. This suggests that the projections under the two scenarios (i.e., constant rates versus linked morbidity, disability, and mortality) represent upper and lower bounds on the likely future numbers of community-based elderly disabled—and hence bounds on the total need for LTC services.

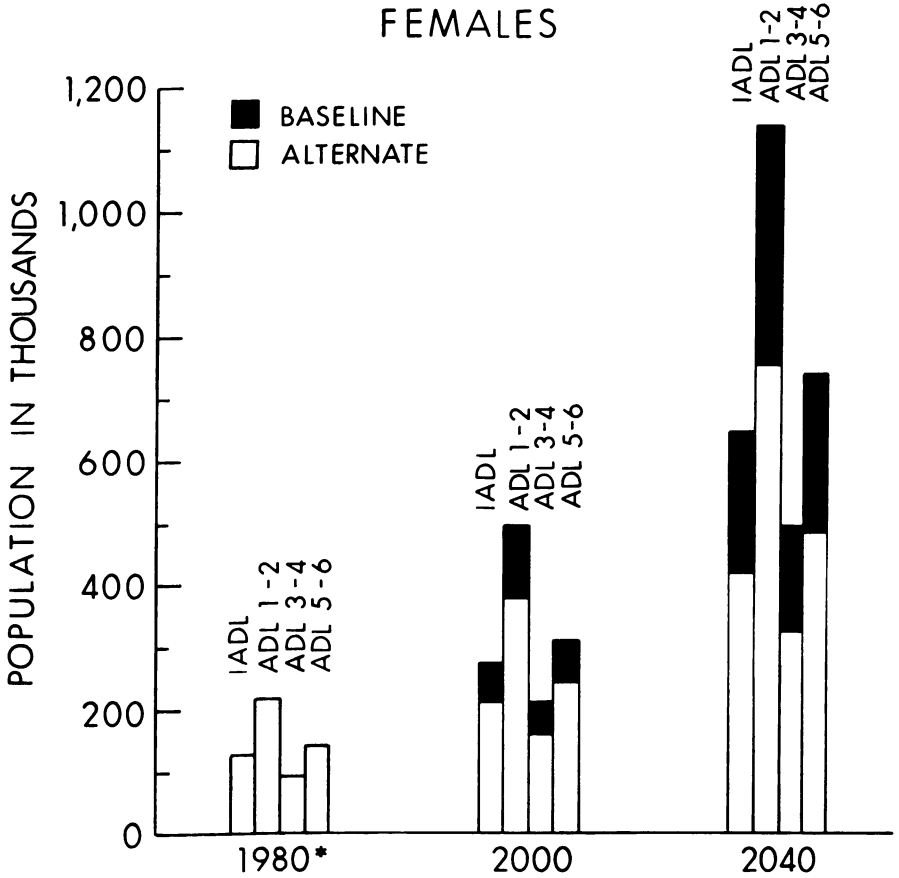
Identification of Subgroups in the Elderly Community-based Disabled Population. In the above analysis we have focused primarily on the quantitative growth of the United States LTC population. A different type of analysis can provide insights into the more qualitative dimensions of the LTC population. To accomplish this we performed the same type of analysis on the NLTC survey that was performed on the First Duke Longitudinal Study population. In particular, a methodology was applied that could identify subgroups in that population based upon sociodemographic factors and functional limitations. The purpose



* Baseline and alternate figures for 1980 are the same.

FIG. 5. Baseline and alternate projections for males and females aged 85 and older in the years 1980, 2000, and 2040 (population in thousands).

Source: National Long-Term Care Survey.



*Baseline and alternate figures for 1980 are the same.

FIG. 5—Continued

of the “grade of membership” (GOM) analysis of the NLTCS is different than that of our analysis of the Duke Longitudinal Study. For the Duke study we were interested in identifying trajectories as age changes in multivariate physiological states. Here we wish to identify subpopulations who represent differing target populations for LTC services. Later we will discuss the nature and mix of subpopulations identified in the NLTCS with subpopulations identified in the 1977 National Nursing Home Survey as a way of helping us to understand how various policy options might affect the mix of requirements for services. The results of the GOM analysis of the NLTCS are presented in table 14 (Manton and Woodbury 1984).

TABLE 14
Sociodemographic and Functional Limitation Response Profiles

Internal variables	Sample proportion	Pure type			
		1	2	3	4
SOCIODEMOGRAPHIC					
<i>Sex</i>					
Male	0.341	0.636	0.272	0.0	0.471
Female	0.659	0.364	0.728	1.0	0.529
<i>Age</i>					
65-69	0.189	0.344	0.0	0.146	0.192
70-74	0.217	0.302	0.0	0.307	0.211
75-79	0.219	0.262	0.054	0.314	0.217
80-84	0.186	0.092	0.310	0.233	0.153
85-89	0.127	0.0	0.459	0.0	0.122
90+	0.062	0.0	0.177	0.0	0.106
<i>Marital status</i>					
Married	0.414	0.778	0.0	0.0	0.652
Not married	0.586	0.222	1.0	1.0	0.348
<i>Education</i>					
Never attended school	0.055	0.0	0.226	0.0	0.068
Grades 1 through 8	0.216	0.132	0.410	0.211	0.188
Junior high school	0.332	0.298	0.164	0.495	0.311
Senior high school	0.278	0.385	0.149	0.222	0.297
College	0.102	0.154	0.050	0.065	0.113
Graduate school	0.017	0.031	0.001	0.007	0.023
<i>Employed</i> \geq 30 hours/week (binary)	0.014	0.043	0.0	0.0	0.0
<i>Income</i>					
\leq \$4999	0.185	0.0	0.0	0.567	0.061
\$ 5000 - \$6999	0.145	0.175	0.124	0.171	0.080
\$ 7000 - \$9999	0.161	0.221	0.067	0.104	0.222
\$10000 - \$14999	0.151	0.291	0.0	0.0	0.271
\$15000 - \$29999	0.125	0.151	0.190	0.0	0.212
\$30000 +	0.044	0.028	0.148	0.0	0.051
Refused to answer	0.059	0.061	0.089	0.054	0.041
Do not know	0.130	0.073	0.382	0.104	0.062
FUNCTIONAL STATUS					
<i>IADL or ADL respondent needs help with (individual binary variables):</i>					
Eating	0.073	0.0	0.0	0.0	0.392
Getting in/out of bed	0.296	0.0	0.0	0.247	1.0
Getting around indoors	0.442	0.092	0.0	0.636	1.0
Dressing	0.229	0.0	0.0	0.0	1.0

Table 14 (continued)

Internal Variables	Sample proportion	Pure type			
		1	2	3	4
Bathing	0.474	0.101	0.347	0.476	1.0
Getting to or using toilet	0.229	0.0	0.0	0.0	1.0
Bedfast	0.012	0.0	0.0	0.0	0.056
Did not get around inside at all	0.018	0.0	0.0	0.0	0.085
Wheelchairfast	0.036	0.0	0.0	0.0	0.173
Doing heavy work	0.829	0.490	1.0	1.0	1.0
Doing light work	0.278	0.0	0.0	0.0	1.0
Doing laundry	0.513	0.0	1.0	0.506	1.0
Preparing meals	0.370	0.0	1.0	0.0	1.0
Shopping for groceries	0.691	0.0	1.0	1.0	1.0
Getting around outside	0.686	0.266	0.618	1.0	1.0
Going places outside of walking distance	0.636	0.0	1.0	1.0	1.0
Managing money	0.312	0.0	1.0	0.0	0.819
Taking medicine	0.275	0.0	0.714	0.0	1.0
Making phone calls	0.206	0.0	0.646	0.0	0.652
<i>Difficulty climbing stairs</i>					
No difficulty	0.126	0.321	0.274	0.0	0.0
Some difficulty	0.288	0.679	0.726	0.0	0.0
Very difficult	0.342	0.0	0.0	0.806	0.132
Unable at all	0.244	0.0	0.0	0.194	0.868
<i>Difficulty bending for socks</i>					
No difficulty	0.370	0.769	0.775	0.0	0.0
Some difficulty	0.304	0.231	0.225	0.547	0.084
Very difficult	0.208	0.0	0.0	0.453	0.310
Unable at all	0.118	0.0	0.0	0.0	0.606
<i>Difficulty lifting and holding 10-lb. package</i>					
No difficulty	0.223	0.665	0.260	0.0	0.0
Some difficulty	0.177	0.335	0.522	0.057	0.0
Very difficult	0.183	0.0	0.218	0.426	0.0
Unable at all	0.417	0.0	0.0	0.517	1.0
<i>Difficulty reaching above head</i>					
No difficulty	0.494	1.0	1.0	0.0	0.0
Some difficulty	0.229	0.0	0.0	0.550	0.262
Very difficult	0.170	0.0	0.0	0.343	0.324
Unable at all	0.107	0.0	0.0	0.107	0.414
<i>Difficulty brushing or combing hair</i>					
No difficulty	0.666	1.0	1.0	0.158	0.0
Some difficulty	0.183	0.0	0.0	0.643	0.301

Table 14 (continued)

Internal Variables	Sample proportion	Pure type			
		1	2	3	4
Very difficult	0.085	0.0	0.0	0.199	0.275
Unable at all	0.066	0.0	0.0	0.0	0.424
<i>Difficulty washing hair</i>					
No difficulty	0.490	1.0	0.951	0.0	0.0
Some difficulty	0.161	0.0	0.049	0.596	0.0
Very difficult	0.114	0.0	0.0	0.404	0.038
Unable at all	0.235	0.0	0.0	0.0	0.962
<i>Difficulty grasping and handling small objects</i>					
No difficulty	0.630	1.0	1.0	0.204	0.328
Some difficulty	0.202	0.0	0.0	0.556	0.197
Very difficult	0.126	0.0	0.0	0.239	0.274
Unable at all	0.042	0.0	0.0	0.0	0.201
<i>Can see well enough to read newspaper with glasses</i>					
	0.707	1.0	0.0	1.0	0.533

Source: National Long-Term Care Survey.

In table 14 we see that there are 33 sociodemographic and functional-limitation measures. In column 2 the probability of a particular attribute being found in the sample is listed. In the final four columns we find the probability of a particular attribute being found in the four groups identified by the analysis. The four types are very clearly distinguished by the patterns of association among attributes. Group 1 is a relatively young group (mean age about 73) of generally intact couples with relatively few functional limitations. Group 4 in contrast is seriously disabled and not distinguished by age (mean age 78). This group has a greater than average chance of being married—probably because such a disabled group would be unlikely to remain noninstitutionalized unless the spouse is present. Perhaps the most interesting groups are the second and third. Both groups are likely to be female and unmarried. The second group is extremely elderly (mean age 86) and relatively free of ADL limitations. They suffer from several IADL limitations, but, in terms of basic mobility (e.g., climbing stairs) they appear to

be in good shape. What is noticeable is that this very elderly group has difficulty in the IADLs of managing money, taking medication, and making phone calls. This suggests significant cognitive impairment in this group—relatively independent of serious physical impairment. This is in distinct contrast to the third group which, though much younger (mean age 76), has more ADL limitation and is considerably more limited in physical mobility. Though more limited physically this group has no impairment in terms of managing money, taking medication, or making phone calls. The relative independence of physical and cognitive impairment in these two groups suggests different policy options with respect to providing LTC services. Specifically, it would appear that the physical limitations of the younger, third group could be compensated for by appliances and special equipment while the cognitive impairments of the second group would seem more likely to require personal care assistance.

To confirm the association of the four subpopulations defined on disability with specific medical conditions, we examined the probability distribution of medical conditions for each of the four subpopulations. These patterns are reported in table 15.

We see that there is a strong association between the patterns of medical conditions reported and the degree and nature of disability. For example, the least-disabled population reports few of the most serious medical problems. In contrast, the fourth, most disabled population is also clearly the most morbid group with high reported rates of paralysis, heart attack, diabetes, cancer, stroke, and senility. This group is probably rapidly selected out by mortality and, if mortality improvements were concentrated among the highly debilitated, the age-specific prevalence of this group would be expected to increase. The oldest old group (mean age 86) reports low prevalence of certain major diseases but high prevalence of conditions associated with advanced age—arteriosclerosis, glaucoma, and, most important, senility. The low prevalence of diabetes may be a result of selective processes. The third group, consistent with its high level of disability, reports significant rates of diabetes and circulatory disease but no senility and low levels of stroke. Thus, the health status profiles of these four subpopulations seem consistent with their levels and types of reported disability and, though based only on cross-sectional data, seem to imply the operation of mortality selection.

TABLE 15
Association of Response Profiles with Medical Conditions

External variables	Sample proportion	Type			
		1	2	3	4
SUBJECTIVE HEALTH INDEX					
Excellent	0.105	0.188	0.237	0.003	0.024
Good	0.300	0.437	0.502	0.188	0.103
Fair	0.338	0.306	0.260	0.519	0.199
Poor	0.257	0.069	0.001	0.290	0.674
MEDICAL CONDITION					
Rheumatism or arthritis	0.732	0.652	0.463	0.979	0.691
Paralysis	0.098	0.049	0.0	0.040	0.336
Permanent numbness or stiffness	0.253	0.167	0.001	0.360	0.431
Multiple sclerosis	0.005	0.002	0.0	0.002	0.016
Cerebral palsy	0.005	0.005	0.004	0.0	0.011
Epilepsy	0.010	0.001	0.004	0.009	0.028
Parkinson's disease	0.033	0.007	0.031	0.032	0.077
Glaucoma	0.088	0.037	0.204	0.071	0.097
Diabetes	0.173	0.118	0.097	0.242	0.227
Cancer	0.062	0.040	0.025	0.057	0.131
Frequent constipation	0.338	0.196	0.204	0.466	0.480
Frequent trouble sleeping	0.420	0.294	0.227	0.616	0.502
Frequent severe headache	0.179	0.085	0.077	0.308	0.235
Obesity or overweight	0.233	0.297	0.0	0.376	0.152
Arteriosclerosis or hardening of arteries	0.334	0.181	0.424	0.309	0.530
Mental retardation	0.020	0.0	0.041	0.0	0.061
Senility	0.101	0.003	0.239	0.0	0.313
MEDICAL CONDITIONS EXPERIENCED IN LAST 12 MONTHS					
Heart attack	0.068	0.036	0.0	0.114	0.108
Other heart problems	0.300	0.196	0.162	0.446	0.369
Hypertension	0.477	0.460	0.206	0.672	0.460
Stroke	0.072	0.026	0.054	0.029	0.212
Circulatory trouble in arms or legs	0.550	0.378	0.270	0.767	0.722
Pneumonia	0.059	0.033	0.046	0.067	0.098
Bronchitis	0.127	0.127	0.006	0.184	0.149
Flu	0.172	0.139	0.092	0.264	0.162
Emphysema	0.109	0.149	0.023	0.100	0.125
Asthma	0.071	0.058	0.004	0.122	0.078
Broken hip	0.025	0.009	0.0	0.031	0.063
Other broken bones	0.056	0.030	0.0	0.093	0.091

Source: National Long-Term Care Survey.

The Institutional Population

Any assessment of disability in the population must include a study of the health characteristics of the nursing home population. In fact, a number of health policy issues may be considered in terms of the probability of exchange between different subpopulations in the community and institutions. To this end it is useful to consider two types of evidence on the institutional population.

Basic Trends in the Characteristics of the Elderly Institutional Population. The first type of evidence simply describes temporal changes in the health characteristics of the institutional population. The National Center for Health Statistics (1981) has conducted two national nursing home surveys (in 1973–1974 and 1976–1977) and three resident place surveys (in April–June 1963, May–June 1964, and June–August 1969) that are close enough in methodology that certain trends can be compared. One factor that can simply be compared is the age-specific utilization rates presented in table 16. We see that the rate of institutionalization among the elderly has nearly doubled, from 2.5 percent in 1963 to 4.8 percent in 1977. Among the oldest old, however, the rate dropped 14.7 percent between 1973–1974 and 1977.

To evaluate this more fully we present nine selected primary diagnoses for three surveys (1969, 1973–1974, 1977) in table 17.

In constructing these estimates we were faced with the methodological problem that we had to recode primary diagnoses to the relatively short list of 22 conditions provided in 1973 (44 conditions were coded in 1977; International Classification of Diseases, Adapted (ICDA) codes were provided in 1969). This clearly affects our ability to discuss trends. Nonetheless, we see consistent patterns in table 17, with major circulatory diseases (heart attack; stroke, especially over age 85), accidents (including hip fracture), and musculoskeletal and nervous system diseases declining at all ages. Cancer appeared to remain stable. Endocrine diseases, which include diabetes, increased as did respiratory diseases. The major increase was for hardening of the arteries.

Grade of Membership (GOM) Analyses of Nursing Home Populations. A second type of evidence involves the identification of subgroups using a GOM analysis as was reported for the NLTCs. We conducted such an analysis on the 1977 NNHS, separately for Medicare and non-Medicare recipients (Manton, Liu, and Cornelius 1985), where five

TABLE 16
Number of Nursing Home Residents
per 1,000 Population

1963	
Under 65 Years	0.6
65 Years and Over	25.4
65-74 Years	7.9
75-84 Years	39.6
85 Years and Over	148.4
1969	
Under 65 Years	0.9
65 Years and Over	37.1
65-74 Years	11.6
75-84 Years	51.7
85 Years and Over	203.2
1973-1974*	
Under 65 Years	0.6
65 Years and Over	45.1
65-74 Years	12.3
75-84 Years	59.4
85 Years and Over	253.7
1977	
Under 65 Years	0.9
65 Years and Over	47.9
65-74 Years	14.5
75-84 Years	68.0
85 Years and Over	216.4

Source: National Center for Health Statistics 1981, 4, table B.

* Excludes personal care home residents.

subpopulations were found which could broadly be identified with major diagnoses. It appeared that diagnosis was a better discriminator of subtypes within the shorter-stay Medicare population. In the non-Medicare population similar groups were found except for the emergence of a group whose primary limitation was in terms of mental illness and retardation.

The groups that were identified appeared to relate to certain subpopulations in the community population. For example, there existed, in both Medicare and non-Medicare reimbursed cases, a "cancer"

TABLE 17
Prevalence Rate per 1,000 Nursing Home Residents by Age and Primary Diagnosis at Last Examination:
1969, 1973, and 1977

Primary diagnosis	Age 65-74			Age 75-84			Age 85+		
	1969	1973	1977	1969	1973	1977	1969	1973	1977
Heart attack	74.9	41.1	14.8	104.5	55.3	14.1	120.4	64.1	26.5
Stroke	139.2	138.0	131.0	120.7	120.7	90.2	88.7	81.5	51.4
Hardening of arteries	60.9	151.7	106.4	113.2	237.1	227.8	138.7	293.8	293.3
Other circulatory diseases	69.9	31.4	81.0	92.0	39.9	95.3	100.3	40.4	126.1
Accidents, poisonings and violence	38.0	35.8	22.2	55.3	45.8	35.1	65.7	51.4	36.9
Musculoskeletal diseases	55.7	58.5	30.3	65.4	70.7	46.9	72.6	74.4	53.8
Endocrine, nutritional and metabolic disorders	69.9	59.5	73.5	53.5	46.9	63.6	33.6	37.1	41.5
Respiratory diseases	23.2	33.3	33.0	21.6	22.9	21.9	18.2	13.7	24.7
Cancer	37.3	29.4	31.1	27.2	23.6	23.0	21.7	20.7	20.3
Diseases of the nervous system and sense organs	94.3	78.4	55.7	60.7	49.3	36.6	39.5	35.4	25.3

Source: National Center for Health Statistics data tapes on the institutionalized population for 1969, 1973, 1977.

subpopulation with short lengths of stay and high mortality. This compared with our fourth group of highly morbid, potentially terminal persons in the community population. We also found stroke and hip fracture, acute care groups, and several groups identified by circulatory problems, multiple chronic diseases, and senility.

In this paper our focus is on the oldest old—a group where over 21 percent were institutionalized in the 1977 survey. To this end we conducted a separate GOM analysis of the population aged 85 years and over in a subsample of 1,618 cases randomly drawn from the three surveys of institutionalized persons (1969, 1973, 1977). We found that there was extreme heterogeneity even in the institutionalized population aged 85 and older. The three groups were defined on 15 variables (age, sex, marital status, medical condition, primary diagnosis, and functional limitation). In table 18 we present the probabilities of three groups for certain characteristics selected from the full set of characteristics employed in the analysis.

The third group is the youngest (i.e., aged 85 to 89), and was more likely to be male and married. The first group was very elderly (27 percent over age 94), female and unmarried. The second group is intermediate in age, but also tended to be female and unmarried. The three groups were very different in terms of health characteristics and level of care. The oldest group had senility, stroke, and hardening of arteries, required intensive nursing care, and was most prevalent in the more recent surveys. The intermediate group had a wide range of conditions, required lower levels of care, and was most prevalent in early surveys. The third group is interesting in that it is a predominantly male group found primarily in the most recent survey. This suggests significant changes in the oldest old nursing home population with the further aging of the female population and the recent emergence of an extremely elderly male group.

International Comparisons

It is instructive to consider how observations about health status at advanced ages made for the United States apply to other developed countries with comparable life expectancies. We have discussed how the conceptual model earlier described, applied to Japanese data, yielded comparable patterns of sex differences in survival and health

TABLE 18
Probability of Having Each of Certain Select Characteristics for Three
Analytically Determined Subgroups

Variable	Sample proportion	Pure type		
		1	2	3
AGE				
85-89	0.5896	0.3707	0.5944	1.0000
90-94	0.3028	0.3650	0.4056	0.0000
95 +	0.1075	0.2643	0.0000	0.0000
PRIMARY DIAGNOSIS				
Senility	0.1583	0.4370	0.0000	0.0000
Heart attack	0.0721	0.0000	0.1990	0.0000
Stroke	0.0624	0.1723	0.0000	0.0000
Hardening of arteries	0.2619	0.3852	0.0000	0.4441
Circulatory	0.1068	0.0000	0.2949	0.0000
Accident	0.0528	0.0000	0.1457	0.0000
Mental	0.0238	0.0000	0.0000	0.0864
Musculatory	0.0663	0.0000	0.1830	0.0000
Endocrine	0.0431	0.0000	0.0000	0.1565
Respiratory	0.0161	0.0000	0.0444	0.0000
Neoplasm	0.0245	0.0000	0.0675	0.0000
Nervous	0.0238	0.0000	0.0000	0.0864
Digestive	0.0148	0.0000	0.0409	0.0000
Infection	0.0026	0.0000	0.0040	0.0040
Genito-urinary	0.0116	0.0000	0.0000	0.0420
Skin	0.0045	0.0000	0.0101	0.0031
Blood	0.0058	0.0055	0.0105	0.0000
Other	0.0489	0.0000	0.0000	0.1775
LEVEL OF CARE				
Intensive	0.4520	0.7591	0.2213	0.3108
Other intensive	0.3870	0.2074	0.5121	0.4857
Personal	0.1474	0.0335	0.2420	0.1848
Neither	0.0136	0.0000	0.0246	0.0187
YEAR OF SURVEY				
1969	0.3090	0.2195	0.4824	0.1480
1973	0.3201	0.3698	0.3624	0.1603
1977	0.3708	0.4107	0.1552	0.6916

Source: National Center for Health Statistics data tapes on the institutionalized population for 1969, 1973, 1977.

status to those found in the United States. In this section we focus our comparisons on United States and Japanese total and cause-specific mortality trends. These comparisons are instructive for what they suggest about (1) the potential for further mortality reductions at advanced ages, (2) the role of cohort effects on mortality at advanced ages, and (3) the cause-of-death patterns at advanced ages.

The country that is of most interest to compare to the United States is Japan because of its extremely high life expectancy—in 1981 it was 73.8 years for males and 79.1 years for females (Koizumi 1982). In figure 6, however, we see that the Japanese are not advantaged at all ages.

Though Japanese mortality rates are lower at most ages, United States mortality rates are lower past age 85 where United States male and female rates are 83 and 76 percent of Japanese rates respectively. This suggests that (1) the lower United States life expectancy at birth is due to higher mortality at ages where lifestyle and environmental factors are important, and (2) if lifestyle modification reduced United States mortality rates at younger ages the United States would have life expectancy higher than current Japanese levels. We also calculated age- and sex-specific mortality ratios for cancer, stroke, and heart disease rates in 1978. These are presented in table 19.

For both sexes we see that cancer death rates are higher through

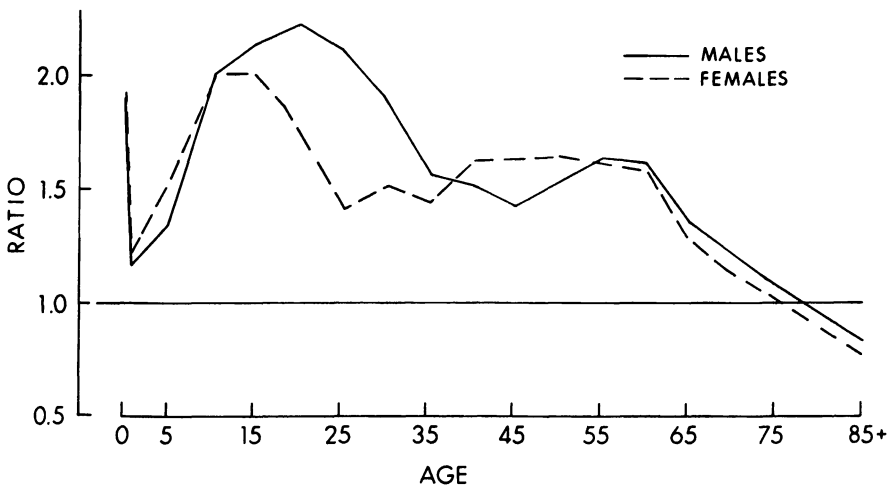


FIG. 6. U.S./Japan mortality ratio.

Source: World Health Organization mortality and population data.

TABLE 19
Ratio of Mortality Rates for Cancer, Heart Disease, and Stroke, U.S. and
Japanese Males and Females, 1978

Age	Cancer	Heart disease	Stroke	All causes
MALES				
30	.92	3.84	48	1.71
35	.98	6.99	.39	1.55
40	1.06	6.89	.34	1.36
45	1.14	7.83	.30	1.38
50	1.27	8.02	.33	1.57
55	1.18	7.34	.34	1.53
60	1.21	6.25	.35	1.52
65	1.07	5.15	.32	1.28
70	1.04	4.34	.32	1.14
75	1.06	3.61	.33	1.05
80	1.15	3.08	.34	.97
85	1.44	2.96	.41	.83*
FEMALES				
30	.86	3.85	1.03	1.30
35	.98	5.05	.92	1.34
40	1.27	6.20	.73	1.49
45	1.42	6.70	.61	1.54
50	1.52	7.06	.58	1.54
55	1.48	6.43	.50	1.50
60	1.45	5.33	.47	1.45
65	1.19	4.05	.38	1.17
70	1.08	3.29	.36	1.02
75	1.06	2.99	.37	.95
80	1.06	2.52	.38	.86
85	1.38	2.89	.50	.76*

Source: World Health Organization mortality data file.

* Ratios of the final crude death rates.

age 40 in Japan. After age 40, cancer death rates are higher in the United States. The rates decline in early old age (i.e., ages 65 to 79) and then increase at advanced ages. The heart disease rates also show a nonlinear pattern with the peak ratios for both males and females occurring at age 50 with United States rates being 7 to 8 times higher than the Japanese. Stroke shows the opposite patterns with Japanese male mortality rates being 2 to 3 times United States levels. The

pattern for females is different with larger excesses in Japanese female risks emerging after age 40.

These cause-specific patterns suggest that the advantage of United States mortality rates at advanced ages is due to significantly elevated stroke risks in Japan and to stroke mortality as an important cause of death at advanced ages. These patterns also suggest considerable potential for improvements in life expectancy in Japan by reducing stroke mortality to United States levels. Likewise, if the United States were to match the Japanese mortality rates for heart disease and cancer in middle age, its life expectancy levels could be significantly increased.

One area of mortality comparison often overlooked is cohort differentials. A number of recent studies suggest that cohort differences are important factors in cross-national mortality differentials. For example, male mortality rates for cohorts born in Japan between 1925 and 1935 (i.e., for males aged 10 to 20 at the end of World War II) were elevated. This was attributed to nutritional shortages experienced in Japan during World War II (Okubo 1982). No comparable risk in cohort mortality rates was observed for Japanese females. Similar findings have been derived for male cohorts born in Germany between 1899 and 1904, i.e., those aged about 16 in 1918 (Horiuchi 1983). Similar patterns have been observed for World War I survivors in France and Austria and have begun to emerge for German males who were adolescents during World War II.

Cohort differences are also observed for cause-specific mortality. In table 20 we compare mortality for three diseases for the United States and Japan for cohorts born in 1893, 1903, and 1913 (aged 85, 75, and 65 in 1978).

In table 20 we have taken the ratio of the cause-specific mortality for different cohorts at comparable ages. If a ratio is consistently less than 1.0 this suggests a cohort effect in that, at all ages compared, the mortality rate for the younger cohort is less than that for the older cohort. The complementary pattern would be noted for cohort increases. For males we see clear evidence of a decrease in heart disease in the United States (see Patrick et al. 1982). In Japan, there is evidence of a decrease between the 1913 and 1903 cohorts while the pattern is mixed between the 1903 and 1893 cohorts. In the United States there are strong increases in cancer mortality for cohorts while the pattern is less consistent in Japan. This is probably due to the greater significance of lung cancer mortality in the United States. For

TABLE 20
 Ratios of Cause-specific Mortality for Different Cohorts at Comparable
 Ages, U.S. and Japanese Males and Females

Age	Heart disease		Cancer		Stroke	
	1913	1903	1913	1903	1913	1903
	1903	1893	1903	1893	1903	1893
MALES						
JAPAN						
45	0.83	—	1.00	—	1.27	—
50	0.93	—	0.99	—	0.89	—
55	0.71	1.01	0.95	1.06	0.68	1.38
60	0.57	1.18	0.90	1.05	0.54	0.99
65	0.72	0.79	0.95	1.02	0.56	0.85
70	—	0.63	—	1.08	—	0.66
75	—	0.85	—	1.13	—	0.68
U.S.						
45	0.97	—	1.00	—	0.79	—
50	1.00	—	1.07	—	0.86	—
55	0.92	0.95	1.11	1.13	0.86	0.76
60	0.81	1.01	1.10	1.10	0.72	0.84
65	0.77	0.93	1.05	1.14	0.64	0.85
70	—	0.84	—	1.15	—	0.79
75	—	0.88	—	1.20	—	0.74
FEMALES						
JAPAN						
45	0.67	—	0.88	—	0.85	—
50	0.76	—	0.93	—	0.65	—
55	0.40	0.83	0.86	1.00	0.62	0.97
60	0.44	0.95	0.86	0.96	0.54	0.82
65	0.60	0.67	0.91	0.92	0.56	0.74
70	—	0.59	—	0.95	—	0.64
75	—	0.85	—	1.00	—	0.67
U.S.						
45	0.80	—	1.00	—	0.64	—
50	0.85	—	0.99	—	0.68	—
55	0.85	0.76	1.07	0.88	0.78	0.67
60	0.74	0.89	1.09	0.96	0.70	0.76
65	0.70	0.86	1.00	1.04	0.60	0.80
70	—	0.78	—	1.06	—	0.77
75	—	0.84	—	1.11	—	0.75

Source: World Health Organization mortality data file.

stroke the difference between United States cohorts is large and consistent. For Japan there is a higher risk for younger cohorts at earlier ages.

The United States female patterns show the same cohort patterns as for males. In Japan the cohort differences are more consistent for females than for males—an observation of interest given their extremely high life expectancy levels. Indeed, for Japanese females even cancer risks show declines. The results suggest that much of the life expectancy increases in both Japan and the United States may be related to cohort differentials in cause-specific mortality risks. This also suggests that Japan will surpass even the current high life expectancy levels they have achieved.

It is informative to make a cross-national comparison of the age rate of increase of mortality at extreme ages similar to the study described earlier of mortality patterns for 20 United States cohorts for ages between 65 and 94 years (Manton, Stallard, and Vaupel 1985). In the United States study there was a decline in the age rate of increase in mortality rates past age 80—a slowing that could be modeled as due to the rapid selection of a heterogeneous birth cohort (see Wilkin 1982). In Horiuchi and Coale (1983) a study was made of female mortality patterns for 10 countries. In these international comparisons a slowing of the age rate of increase of mortality rates at advanced ages (i.e., age 75 and over) similar to that in the United States was observed. As in the American study this could be explained by modeling the population as being heterogeneous in its mortality risks (one of the same models, the gamma mixed Gompertz, was found to describe the data reasonably well). This was viewed as particularly significant in Japan because of the accurate reporting of age in that society due to the special cultural significance of the celebration of the 60th (Kanreki), 70th (Koki), 77th (Kiju), and 88th (Beiju) birthdays.

Comparisons of adult mortality between developed and less-developed countries are often made using cause-elimination life tables. This approach provides a hypothetical estimate of the effect on life expectancy of eliminating a given disease or cause of death. This estimate is usually expressed as the average additional number of years that can be expected to be lived in a population if the disease were totally eliminated (i.e., $\Delta e_{x,A}^0$). Typically, using such measures one finds that chronic diseases have less of an impact in developing countries than in developed countries. This is illustrated in table 21 for United States

TABLE 21

Average Life Expectancy Gains ($\Delta \hat{e}_{x,A}$) in the General Population; and Age-specific Percentage of Deaths ($\hat{c}_{x,A}$), Residual Life Expectancy ($\hat{e}_{x,A}$), and Average Life Expectancy Gain ($\Delta \hat{e}_{x,A}$) among Persons Observed to Die of Each of Three Diseases

	$\Delta \hat{e}_{x,A}$		$\hat{c}_{x,A}$		$\Delta \hat{e}_{x,A}$		$\hat{c}_{x,A}$		$\Delta \hat{e}_{x,A}$		$\hat{c}_{x,A}$		$\Delta \hat{e}_{x,A}$			
	1950	1978	1950	1978	1950	1978	1950	1978	1950	1978	1950	1978	1950	1978		
	MALES															
Cancer in U.S.	FEMALES															
Birth	1.89	3.01	13.40	20.78	67.13	69.88	14.07	14.50	2.48	3.25	15.22	18.42	67.39	71.38	16.32	17.65
Age 75	0.71	1.34	4.19	7.71	6.86	7.60	6.23	7.75	0.69	1.15	5.23	8.07	7.67	9.58	6.94	9.45
Cancer in Japan	FEMALES															
Birth	1.33	2.97	10.11	21.25	64.64	70.30	13.17	13.97	1.54	2.48	9.79	15.87	64.30	71.46	15.69	15.64
Age 75	0.35	1.01	1.94	8.18	5.37	6.74	4.90	6.81	0.35	0.78	2.29	7.27	6.33	8.05	5.69	7.65
Heart disease in U.S.	FEMALES															
Birth	6.27	5.81	40.02	36.89	71.14	73.89	15.67	15.76	5.72	6.41	38.94	38.09	76.87	82.31	14.70	16.83
Age 75	4.33	4.55	16.70	18.54	7.92	9.05	9.54	10.98	4.99	6.46	24.06	29.39	9.16	12.08	10.83	14.54
Heart disease in Japan	FEMALES															
Birth	0.91	1.06	8.15	10.98	69.32	77.71	11.20	9.64	1.17	1.20	9.99	12.99	72.65	82.68	11.68	9.20
Age 75	0.61	0.85	3.34	7.22	7.12	9.05	4.93	6.50	0.86	1.14	5.44	10.74	8.45	10.80	5.90	7.59
Stroke in U.S.	FEMALES															
Birth	1.16	0.85	10.85	8.04	73.48	77.20	10.70	10.52	1.69	1.59	14.81	13.34	76.01	82.76	11.38	11.94
Age 75	0.95	0.85	5.51	5.18	7.90	9.53	6.38	7.32	1.24	1.57	8.90	10.72	8.86	12.29	7.27	9.67
Stroke in Japan	FEMALES															
Birth	1.88	3.16	17.87	27.39	72.06	77.18	10.50	11.52	2.48	3.79	21.73	31.84	74.50	81.47	11.40	11.89
Age 75	1.76	2.61	7.85	17.68	6.78	8.61	6.05	8.15	2.26	3.42	11.80	25.21	7.71	10.33	7.18	9.70

Source: World Health Organization mortality data files.

and Japanese males and females from 1950 to 1978 for three conditions. In Japan in 1950 life expectancy levels at birth were similar to those in many developing countries. Hence, the cross-temporal comparisons of $\Delta e_{x,A}^{\circ}$ for Japan show the effects within a country of a rapid progression from developing to developed country life expectancy levels.

We see that the effect on male life expectancy of cancer increased 123 percent (from 1.33 to 2.97 years) from 1950 to 1978 and that the effect on female life expectancy of stroke increased 53 percent. These changes are potentially misleading since they involve cross-temporal variation of (1) the proportion of deaths due to the given condition, and (2) the mean age at death of the given condition. In order to unconfound these factors one can modify the cause-elimination life table computations in order to calculate the life expectancy gain only among persons observed to die of the condition (Greville 1948; Manton, Patrick, and Stallard 1980). These alternate life expectancy change estimates ($\Delta e_{x,A}^{\circ}$) are also presented in table 21 and show very different patterns. For example, among United States males observed to die of cancer, the theoretical gain in life expectancy was 14.1 years in 1950 and increased only marginally to 14.5 years in 1978. The additional columns in the table show the two factors that underlie the differences between the measures. The first, f_{xA} , describes the percentage of all deaths expected from the condition past exact age x . Hence, the 59 percent increase in $\Delta e_{x,A}$ for cancer in the United States from 1950 to 1978 is largely a product of an increase of 55 percent in the proportion of deaths expected to be due to cancer. Along with the 55 percent increase in f_{0A} , we also find significant increases in the mean age at death (e_{0A}°) from cancer from 67.1 to 69.9 years. The primary observation of importance in this table is the consistent increase in the mean age at death—even for cancer with its large increase in the proportion of deaths it causes and for stroke in Japan with its greater importance as a cause of death.

Conclusion

The aging of the United States population has presented new challenges to the development of health and social policy. In designing responses to these challenges planners have focused on the quantitative dimensions of population aging. This is a natural response given the magnitude

of the problems. A solely quantitative perspective, however, does not take into account that the current process of population aging in the United States is historically unique and has important qualitative features that will be important in developing policy.

One distinctive feature of the current process of population aging is the reduction of mortality at advanced ages which is leading to rapid growth of the population aged 85 and over—the population group with the highest per capita service needs. If the projections produced by the Social Security Administration (1982) are correct, and we have 5.4 million persons over age 85 in 2000 and over 13 million persons over age 85 in 2040, then policy responses that do not take into account qualitative dimensions may be overwhelmed or misdirected. Current policy has been formulated on the basis of concepts and evidence that inadequately reflect these qualitative factors. Presently we lack adequate concepts and models of the health changes that will be associated with increases in life expectancy at advanced ages. For example, Omran's (1971) theory of the "epidemiological transition" did not envisage a stage in which chronic-disease mortality would decrease in importance. Even our scientific understanding of the basic age trajectory of many physiological processes has been profoundly changed—just since 1982. The possibility of life expectancy increases at very advanced ages was not represented in most demographic and actuarial projections until recently (e.g., Social Security Administration 1981).

Developing strategies to allow policy makers to adequately respond to the qualitative dimensions of population aging requires two elements. The first is a broad conceptual framework to relate basic health and survival changes. Such a framework is presented in figure 1. This framework is necessary to organize the multiple interacting processes describing health changes at advanced ages into a coherent and readily comprehensible model. With such a conceptual model we can apply the information-organizing capacity of mathematics to analyses of a wide range of types of information and for different questions. The range of mathematical tools that will be required to utilize the model in different contexts will be broad—in some cases actual cohort survival can be described while in other cases only the experience of a cross-section is available. The mathematical tools may even be more detailed than the model presented representing individual differences in risk or a more complete set of health state transitions where disability and

morbidity may be reversed at the individual level. Nonetheless, all of these models can be developed from the basic concepts of the age correlation of morbidity, disability, and mortality as described in figure 1.

The second element is a comprehensive review of a broad range of data on the interrelation of morbidity, disability, and mortality changes at advanced ages. Naturally, one probable conclusion of any such review will be that our current knowledge of the health changes among the oldest old is seriously and systematically deficient. However, by *systematically* identifying gaps in our knowledge we can begin to develop a map for needed research initiatives. A requirement of such a review is that disciplinary boundaries be bridged so that a broad consensual view of the phenomena can be generated.

In the review we have just conducted, we were able to review only selected elements of the relevant data. However, even this cursory investigation of the data, and a review of the existing literature in several relevant disciplines (e.g., Riley and Bond 1983; Minaker and Rowe 1985) offered insights and suggested basic principles for further investigation and for policy development. First, it is clear that morbidity, disability, and mortality are generated by a multidimensional physiological process operating at the level of the individual organism. Second, it is clear that there is wide variability in the parameters of this process and that there is greater possibility for intervention than was previously thought. Because of the interlinkage of morbidity, mortality, and disability in an individual-level process, intervention must be evaluated with an eye to possible feedback in the system. Third, the process relating morbidity, disability, and mortality extends back over the full life of an individual. Thus, life-course and cohort perspectives should be introduced into policy development. Currently such concepts are seldom utilized in policy evaluations (e.g., Social Security Administration 1981). Fourth, the oldest old are a highly selected group of survivors. To understand their needs one must understand their special status. Finally, current stereotypes of degenerative health changes at advanced ages are inaccurate and must be changed.

With these and other insights, policy and planning to meet the health and social needs of the elderly can be improved in a number of areas. First, a better understanding of the age dependency of the transition between health states at advanced ages can greatly improve

our forecasts of life-expectancy change (Manton 1983) and changes in disease and disability prevalence (Manton and Liu 1984a). It may also be used to define new areas of needed research (e.g., identifying the effects of major risk factors on health at advanced ages). Second, a better understanding of the heterogeneity of the oldest old population and the qualitative aspects of their health status may help define new, more effective policy responses. This was illustrated in our analyses of the National Long-Term Care Survey (NLTC) where cognitive and physical impairments were found to occur somewhat independently and to be predictors of differential needs for service (i.e., special equipment seemed more useful for the younger, physically disabled group while personal assistance was more likely necessary for the older, cognitively impaired group). Third, the insights gained in such studies appear promising for the development of prospective payment systems for different types of health services. For example, the qualitative analysis of the NLTC survey suggested that reimbursement classification for LTC services must be multidimensional, going beyond simple functional classification to include mitigating socioeconomic and housing factors (Luce, Liu, and Manton 1984). Additional analyses also suggested the limited additional information in medical diagnoses for such services. Clearly, this preliminary study of the changing health characteristics of the oldest old suggests the need for intensive review of the concepts and evidence upon which we now base current and future policy.

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