IMPLICATIONS OF POPULATION AGING FOR GERIATRIC HEALTH

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ABSTRACT

The U.S. population has been aging for more than a century. The changes in health that accompany increasing longevity have been characterized by competing theories that rest upon assumptions about the relationships among old-age mortality, morbidity, and disability. The four theories evaluated in this chapter are: the pandemic of chronic disease, the compression of morbidity, life span expansion, and dynamic equilibrium. Each of these theoretical approaches has distinct implications for the shape of mortality change and for morbidity-mortality linkages. Evidence regarding recent changes in morbidity, disability, and mortality in the United States is reviewed in terms of each of the theoretical perspectives. Existing evidence is found to most closely support the theory of dynamic equilibrium, confirming that a compression of morbidity is not a foregone conclusion or an inevitable result of increased longevity.
INTRODUCTION

Aging is both an individual and a population level process. Just as each additional year of life marks the aging of an individual, increases in the absolute numbers of older persons and a rise in the relative share of the population that is considered "old" reflect the aging of a population. Whereas older individuals face increased risks of death, disease, and impairment, aging populations are marked by a higher prevalence of chronic disease and disability, and rising age at death.

The U.S. population has been aging for more than a century, though the pace has accelerated over the last 20 to 30 years. As recently as 1970, less than 10 percent of the population was 65 and older; by 1997, this figure had grown to 12.7 percent; and conservative estimates suggest this figure will exceed 20 percent by 2050 (1). During the same period, life expectancy at birth has increased from 67 to 72.5 years for males and from 75 to 79 years for females (2). Projections suggest that by the year 2050 men and women will live on average to age 80 and 84 respectively (1).

The elderly population itself also is aging rapidly. Presently, 34 million persons are aged 65 or older and over 11 percent of these are considered the oldest-old (aged 85 or older). By 2050 this proportion is expected to rise to almost one in four. In numbers this means an increase from about 3.8 million persons aged 85 or older today to over 18 million in 2050 (1).

Population aging is driven by the three basic components of population growth: fertility, mortality, and migration. In the first half of the twentieth century, declines in fertility were responsible for increasing the relative share of elderly persons in the U.S. population, though the baby boom slowed this trend for a while. Since the 1960's, however, reductions in mortality at older ages (Figure 1) have accelerated population aging (3,4). These declines in mortality are largely attributable to reductions in cardiovascular and cerebrovascular mortality (5,6). The National Center for Health Statistics reports that diseases of the heart, cerebrovascular disease, and artherosclerosis were responsible for 80 percent of the increase in life
expectancy between 1984 and 1989(6). In contrast, migration has had a minimal influence on the rate of population aging, since the amount of net migration in any year is far less than the number of births and deaths. Nevertheless, migration has contributed to the changing composition of America's elderly—particularly in terms of increasing racial and ethnic diversity.

The prominent role of mortality trends in explaining population aging in recent years and the strong links between mortality and morbidity, population aging in the U.S. can be expected to have important consequences for the health of the older population. Yet, the linkages between population aging and geriatric health are complex. Theories abound about how morbidity rates change in concert with declining mortality; each perspective lends itself to a different interpretation about the future of geriatric medicine. At one extreme, if all persons were to live their lives in good health and die of "natural" causes at a very old age, then geriatric practice would be reduced to routine monitoring of general health. At the other extreme, if increased longevity were to add only years of frailty, then geriatric practitioners would by necessity focus more on palliative care for chronic conditions.

The purpose of this chapter is to provide insight into the implications of population aging for geriatric health. The next section begins with a conceptual model of population health. This discussion sets the stage for a review of the major theories of morbidity-mortality linkages. To evaluate these theories, evidence regarding recent changes in morbidity, disability, and mortality in the U.S. is presented, and the likely implications of population aging for the future of geriatric health are discussed.

MODELING POPULATION HEALTH

The changes in health that accompany increasing longevity have been characterized by competing theories that rest upon assumptions about the relationships among old-age mortality, morbidity, and disability. A useful model for thinking about these linkages was developed by the World Health Organization (7) and is shown in Figure 2. Extrapolating from the basic survival function of the life table, this model represents graphically the age structure of morbidity, disability, and mortality in a population. The X-axis is age in years and the Y-axis shows the proportion of a cohort that survives
to a specific age without experiencing a particular event, either the onset of disease (A), disablement (B), or death (C). Curve C is the survival function derived from U.S. age-specific mortality rates for females in 1990. Curves A and B represent the hypothetical distribution of survival to each age without disease or disability, respectively.

This formulation lends itself to the calculation of summary measures of the health and mortality of the population in the form of expectations (or averages). For example, life expectancy at birth ($L^0$) and at age 65 ($L^{65}$) are shown as points on curve C. Comparable measures of healthy and active life expectancy, which represent the average number of years lived before the onset of chronic morbidity and disability, respectively, also can be represented. Healthy life expectancy is described by the area under curve A, representing the total number of person-years of good health in the population. Active life expectancy is the area under curve B, and the difference between the two curves shows the average amount of time spent with illness, but without disability.

THEORIES ON THE RELATIONSHIP BETWEEN MORBIDITY AND MORTALITY

The WHO model formalizes the relationships among morbidity, disability, and mortality, and provides a useful framework for describing and evaluating theories about the morbidity and disability changes that accompany increases in longevity. In this section we describe in detail the four most prominent theories of morbidity-mortality linkages.

**Pandemic of Chronic Disease.** One of the first theories relating mortality changes in the 20th century to patterns of morbidity was Grubenbergs's Pandemic of Chronic Disease model (8,9). This theory asserts that the invention of anti-infectious drugs such as sulfa, antibiotics, and insulin in the early part of this century led to the increased survival of persons with chronic morbid conditions. That is, frail individuals with reduced immune function who would have died of pneumonia or other opportunistic infections before the invention of such drugs were now able to survive longer, though in relatively poor health. Conditions that "benefited" from the elimination of fatal sequelae include arteriosclerosis, hypertension, diabetes, Downs syndrome, and organic brain disease. Such gains in survival also may have
increased the prevalence of mental disorders, such as dementia, which typically have a higher incidence at older ages (9).

This theory assumes that the age at onset of chronic diseases will remain the same but that mortality will be postponed until older ages. Consequently, the number of years spent morbid or disabled will expand with increases in longevity. In terms of Figure 2, we would expect to see the mortality curve shift outward as improvements in survival take place, but would observe no changes in the disability or morbidity curves, because the onset of disease and disability are assumed to remain the same.

**Compression of Morbidity.** Probably the most well known theory inspired by increases in longevity is the "compression of morbidity" theory espoused by James Fries. Fries argues that the average length of the human life span is fixed according to some predetermined biological endowment (11,12). In his original description of the theory, he assumes life expectancy is fixed is 85 (11). In more recent articles, he has allowed this maximum to move up as far as 86.9 years, but has not entertained further increases in maximum average life span (13). Further, he contends that the age of onset of chronic disease will of necessity increase, as we approach a time when all causes of premature mortality are eliminated.

In terms of Figure 2, the mortality curve does not shift outward but instead "rectangularizes" as more deaths occur close to the maximum average life span. The morbidity and disability curves follow a similar course as the infirm period of life is deferred to later ages. As the area between the mortality and morbidity curves decreases a compression of morbidity takes place.

**Life Span Expansion.** Several researchers, including Roy Walford, have proposed that Fries’ contention of a fixed maximum lifespan resulting from an unalterable rate of aging is biologically inaccurate and encourages fatalism among medical practitioners (10). He argues that it is possible to intervene in the underlying process of senescence, increasing life expectancy by as much as 25 years. In this model, slowing the aging process and therefore pushing back the age at onset of disease and disability would expand the proportion of active life. Walford, in particular, expects that these changes could result from dietary manipulations, especially from caloric restriction, which has
been related to longevity in mice. He argues that animals reared with dietary restriction not only live longer, but experience a shift towards later ages in the onset of diseases such as cancer, cardiovascular disease and auto-immune conditions (10).

The interventions envisioned by Walford would affect the risk of onset of disease, but not necessarily the disease process itself. The onset of morbidity (curve A) would shift back to later ages but the years lived with morbidity and disability would remain unchanged. In other words, all three curves in Figure 2 would shift proportionately to later ages, with the distance between the curves remaining the same.

**Dynamic Equilibrium.** Manton has proposed a fourth perspective on these relationships, dubbed “Dynamic Equilibrium” (14,15). Manton’s view makes explicit the complex interactions among morbidity, disability, and mortality. A fundamental assumption of his approach is that all three processes are mutable. That is, if there is a limit to life expectancy we have not yet come close enough to observe evidence of its existence. Further, he argues that the three processes are inter-related, so that interventions designed to affect one of the processes will inevitably alter the course of all three trajectories. For example, interventions in the late 1970s and early 1980s designed to control hypertension, not only reduced disability due to the disease, but also may have contributed to reductions in the incidence of myocardial infarction and consequent mortality. Declines in heart disease mortality, in turn, may have lead to increases in the prevalence of related disabling diseases, such as congestive heart failure (16).

The dynamic equilibrium model differs from the other approaches described above in that it assumes that years of life are gained through a combination of postponement of disease onset, reductions in severity of disease and speed of progression, and improved techniques for clinical management. It is also distinguished in assuming that chronic disease and mortality are related. For example, Manton suggests that decreasing or eliminating atherogenesis would not only decrease mortality by reducing the risk of acute incidents such as stroke and myocardial infarction, but also would strengthen the cardiovascular system in its ability to withstand other diseases in later life, such as cancer (14).
Within the WHO framework, dynamic equilibrium anticipates that all three curves will shift and change in shape as the cascading effects of changes in the disease process manifest themselves. Because all three curves shift, unlike the three previous theories, no conclusions can be drawn about the necessary relationship among the curves.

EVALUATING EVIDENCE OF MORBIDITY-MORTALITY LINKAGES

When the WHO model was first developed, empirical evidence was not available to test these theories, but in recent years, many longitudinal and time-series studies have been undertaken to assess the implications of increases in longevity for population health. To evaluate the various competing theories of morbidity-mortality linkages described above; we summarize existing studies in three areas. First, we review studies focusing on the rectangularization of mortality. Second, we review studies of trends in chronic disease and disability. Finally, we examine trends in life expectancy and active life expectancy in the U.S.

The Rectangularization of Mortality. Of the four theories reviewed above, only Fries' view asserts a limit to longevity. A number of studies have attempted to address whether the U.S. population is indeed reaching such a limit. Such studies are predicated on the assumption that even if we do not know the exact limit to human life span, we would expect to see certain patterns in mortality if the older population was approaching such a limit. First, we would expect mortality reductions in old age, but these reductions would be smaller for subgroups closer to the limit. That is, for very old subgroups with particularly high life expectancy, e.g., very old white women, we would expect to see improvements in mortality to be smaller than for the rest of the population who have not yet approached the limit. Second, if we were approaching the limit to longevity, we would expect to see less variation around the average age at death. That is, as more people approach the biological limit to life, and non-senescent causes of death are eliminated, the variation around the average age at death should decline.

What evidence is there for the rectangularization of mortality? Manton shows that from 1950 to 1977 white women experienced larger gains in life expectancy than white males, suggesting that they had not yet reached the
biological limits of life (14). Even white women aged 85 and older--those most likely to have reached the limit--experienced improvements in mortality over the same time period. More recently, Manton and Singer (16) also show patterns of mortality change that are inconsistent with the rectangularization of the survival curve. They compared the age to which the last 5 percent and last 1 percent of the population survived using Social Security Administration survival curves from 1900 to 1992. If compression had been occurring over this period, the age to which the last 5 percent survived should have increased more than the age to which the last 1 percent survived; yet the authors find both measures increased by the same amount.

Several researchers also have addressed the issue of the variability of age at death among the older population (14,17,18). Mean age at death for persons aged 50 and over for 1960 and 1978 for standard U.S. life tables show that the variance of the age at death had increased by 16.5 percent--over 75 percent faster than the mean age at death had increased (14). Even when deaths are disaggregated by both underlying and mentioned causes of death, the means and standard deviations for all conditions are increasing (17). Such patterns are inconsistent with a rectangularization of mortality, though it may be that we have not yet approached the possible maximum human age.

**Trends in Chronic Disease and Disability.** Even if the geriatric population has not reached the limit to average life span, a change in the morbidity or disabled period could come about due to shifts in the morbidity and disability curves. What evidence do we have about the trend in chronic disease and disability among older Americans? Evidence from the 1970s and early 1980s suggested older Americans were experiencing longer periods of morbidity and disability than previous cohorts. Verbrugge (19) and Waidmann et al. (20), for example, found increases in self-reported chronic conditions by older Americans during the 1970s. Studies also showed increases in self-reported disability (19), even within five-year age groups (21). Whether such trends reflect actual changes in the underlying health of the older population is subject to debate. Waidmann et al. (20) attribute increases in hypertension during the 1970s and 1980s to a trend toward earlier detection rather than actual changes in the prevalence of the disease. Similarly they attribute increases in the numbers identifying themselves as unable to carry
out activities due to increased awareness of social programs with disability benefits.

Evidence from more recent years suggests longer life does not necessarily imply worsening health. Table 1 shows the prevalence of a variety of morbidity and disability measures as reported by older Americans from the 1984 and 1994 National Health Interview Survey. Overall, the comparisons across years paints a more benign picture of changes in the health of older Americans than earlier studies, even without controlling for differences in the age structure of the older population over time.

Over the ten-year period, fewer older men reported having cerebrovascular disease, diabetes, emphysema, hardening of the arteries, and visual impairments, and fewer older women reported diabetes, hardening of the arteries, heart disease, hypertension, and hearing, visual and osteopathic impairments. These findings are consistent with those reported by Waidmann et al. (20) and Manton et al. (22) who show declines or no change in most self-reported chronic conditions during the 1980s. Analysis of medical records of elderly HMO enrollees over two nine-year periods also suggests the incidence of heart disease was no greater from 1980-1989 than from 1970-1979 (23).

Nevertheless, some chronic conditions have increased in recent years. For women, cerebrovascular disease and emphysema increased from 1984 to 1994 (Table 1). At the same time, reports of heart disease and hypertension have increased for men. Further, both older men and women are more likely to report arthritis and cataracts in 1994 than in 1984. Such increases in chronic disease are driven in part by the aging of the population—that is, the fact that the age of the average geriatric patient has increased substantially over the past decade. Further, in some cases, changes in the detection and treatment of the specific disease in question might be fueling an increase. The increase in reports of cataracts at the same time vision impairment has declined is an obvious example of how changes in treatment (in this case cataract surgery) can have apparently contradictory effects on self-reported trends in health. In other cases, increases in disease might reflect changes in the risk factors for older men and women earlier in their lives; for example, the increase in emphysema in women is likely due at least in part to smoking trends among younger cohorts of women earlier in the century.
Irrespective of the trends in self-reported disease, Table 1 shows that in 1994 fewer older men describe themselves as having a limitation in their major activity, compared with 1984. Women, on the other hand, have experienced no change in their reports of activity limitations. The lack of increase in disability is consistent with other studies focusing on older Americans' ability to carry out personal care activities (such as bathing, dressing, feeding) and routine care activities (such as shopping, using the telephone and managing money). Crimmins, et al. (24), for example, show no trend in the prevalence of needing help with personal care but show declines in prevalence of needing help with routine care and declines in the incidence of both personal and routine care needs. Manton and colleagues (25,26) have put forth even stronger evidence for a decline in prevalence and incidence of old-age disability, describing a difference of 1.4 million in the number of older Americans who would have been disabled in 1996 if disability rates had not declined since 1982.

**Trends in Active Life Expectancy.** Evidence of declines in mortality, morbidity, and disability in isolation are insufficient to distinguish among theories of morbidity-mortality linkages. If the mortality curve shifts outward, but is not matched by shifts in the morbidity and disability incidence curves, for example, an expansion in morbidity and disability will occur along the lines of Gruenberg's Pandemic of Chronic Disease scenario. Alternatively, if shifts in the morbidity curve are matched by similar changes in disability and mortality, then no change in the morbid period will come about, as predicted by the Life Span Expansion theory.

Competing theories can be distinguished, however, according to their predictions about absolute changes in life expectancy, and the number of years and relative amount of the lifespan expected to be spent in healthy or active states. In practice, measures of disability have been more readily available than measures of morbidity so we focus on active (rather than healthy) life expectancy measures here. Measures of active life expectancy are most often calculated from prevalence rates and may therefore be interpreted as the experience of a hypothetical cohort subject to rates prevailing during the time period in question, usually a single year. Period estimates, because they merge the experience of different cohorts, cannot tell us whether
individuals are living more years in ill or good health, and can lead to pessimistic conclusions about health at a given point in time (27). However, they provide a useful portrait over successive time periods of the trends in the health of a population (28). Measures of disability used to calculate active life expectancy vary widely from study to study, although measures of inability in carrying out roles or activities are used most frequently (29).

In Table 2 we present the expected direction of changes in four summary measures of population health for each of the four theories of morbidity-mortality linkages. The first column shows the expected direction for total life expectancy (or the average number of years of life). The second and third columns show expected directions for the average number of years spent in disabled and active states, respectively. Mathematically, estimates of the average number of years spent in disabled and active states sum to total life expectancy. Column four shows the expected change over time in the proportion of total life expectancy that is active (active life expectancy / life expectancy); this measure is particularly useful because it takes into account changes in both active and disabled years.

In terms of anticipated changes in active life expectancy, the rows of the table may be interpreted as follows: Gruenberg's Pandemic of Chronic Diseases implies increases in life expectancy and disabled life expectancy, but no change in active life expectancy. Consequently, we would expect to observe declines in the proportion of active life. Fries' theory of Compression of Morbidity is consistent with little or no change in life expectancy, but anticipates increases in active life expectancy offset by declines in disabled life expectancy. This theory also is consistent with expected increases in the relative amount of active life. If Walford's Life Span Expansion theory holds true, we would expect to see increases in life expectancy and active life expectancy, but no change in disabled life expectancy, also leading to an increase in the proportion of years spent in active life. Only Manton's theory of Dynamic Equilibrium asserts that increases in all three—life expectancy, active life expectancy, and disabled life expectancy—will occur. Unlike the three previous theories, this model can offer no prediction about changes in active life as a proportion of total life expectancy, as such forecasts can only be made when one of the three
parameters is assumed to be fixed.

Table 3 presents evidence on the direction these four summary measures of population health have taken in recent decades. Our estimates of change are calculated from the longest available time-series of life expectancy and active life expectancy for the U.S. (30), which are based on cross-sectional disability prevalence rates from the National Health Interview Survey for 1970, 1980 and 1990. In this table, disability is defined broadly and includes individuals in the community with long- and short-term limitations in their ability to carry out their normal activities because of a health condition and individuals institutionalized for the care of a mental or physical condition. The top panel shows changes for the earlier decade (1970 to 1980) and the bottom panel presents estimates for more recent changes (1980 to 1990). For each time period, we show changes in expectations calculated at birth and at age 65, for men and women separately. We focus on the measures for age 65, as they are less susceptible to the effects of changes in survival and health at the younger ages.

From 1970 to 1980 total life expectancy at age 65 increased for both men and women (1.2 and 1.6 years). Most of this increase was in disabled years: men experienced an additional 1.0 and women 1.4 years of disabled life after age 65. Years of active life expectancy also increased, but the increase was much smaller (0.2 years for both men and women). Despite increases in all three measures, the proportion of total life spent in an active state declined by 2.9 and 3.7 percentage points for men and women during this decade.

These trends may mask important variation over time in the distribution of older persons across different levels of disability. Multinational comparisons of trends in the 1970s show that when calculations of disabled life include only the most severely disabled, increases in life expectancy were matched by increases in active life expectancy (31). That is, the proportion of life spent in a severely disabled state did not increase during this time period. Thus, the declines in the proportion of life spent in a non-disabled state during the 1970s can be attributed to increases in moderate or light disability.

The second decade shows smaller increases in total life expectancy at age 65 than the earlier period: an additional 0.9 and 0.5 years for men and
women, respectively. Increases also were observed in both disabled and active life expectancy at age 65; however, unlike the earlier period, a greater share of the increase in total life was due to increases in active life expectancy. As a result, the percent of total life expectancy spent in an active state actually increased by 1.1 and 1.4 percentage points for men and women, respectively, from 1980 to 1990.

Although at face value this evidence appears contradictory, suggesting an expansion of disability in the earlier period and a compression more recently, these results are consistent with Manton’s theory of Dynamic Equilibrium. During both time periods increases in total, active, and disabled life expectancy were observed, and such changes did not have a predetermined effect on the proportion of remaining life spent in an active state.

IMPLICATIONS

Taken together, recent trends in mortality, disability, and life expectancy offer little evidence that Pries’ Compression of Morbidity is taking place. Although evidence from the 1970s suggests that morbidity may have been expanding, the trend has not been unidirectional, and was followed in the 1980s by improvements in morbidity and disability at the oldest ages. Instead, existing evidence suggests that increases in survival that accompany population aging can best be understood as the result of a complex, dynamic process. Thus, whether increased life is accompanied predominantly by additional healthy or morbid years is not predetermined.

What are the implications of this characterization of population aging for geriatric practice? First and foremost, practitioners must recognize the vital role they play in determining the character of the lengthened lives of the older population. A compression of morbidity is not a foregone conclusion, or an inevitable result of increased longevity. There is a large role for medical practitioners in determining the direction of changes in the health status of the older population. Whether extra years of life are healthy and active or disabled and dependent is strongly related to the medical interventions employed by practitioners. A focus in earlier decades on reducing mortality from diseases such as myocardial infarction, kidney
disease and stroke added more years of unhealthy life, as individuals survived but remained dependent upon dialysis machines, pacemakers, or other medical technologies. In contrast, efforts aimed at preventing, reversing, and generally slowing the progression of disabling conditions such as diabetes, joint disease, osteoporosis, and arthritis, may have contributed to a compression of the morbid period in more recent years.

The dynamics of population aging also suggest an important role for recovery and rehabilitation, even among the very old. Currently recovery rates among the disabled geriatric population may be as high as 20 percent (32). Technological advances in areas such as bone and joint replacements; new surgical and drug therapies for neurological disorders such as Parkinson's disease; and advances in stroke rehabilitation all suggest that even greater increases in recovery and rehabilitation may be attainable. Demographers have demonstrated that additional gains in recovery rates would have a profound effect on reducing disability prevalence in the population (27,33).

Finally, although Fries' Compression of Morbidity theory suggests that the health of the older population will become more homogeneous as premature mortality is reduced and more individuals die of natural causes, the dynamic process of population aging described here suggests that, in the future, geriatric practitioners will serve an even more diverse older population. Other demographic trends will reinforce this trend toward increased heterogeneity. For example, substantial racial and ethnic differences in life expectancy and active life exist in the United States, though as better educated cohorts age, some of this disadvantage may be reduced (38). Further, even if the health of the older population improves, vast increases in the sheer number of older Americans with chronic disease and disability are expected between now and 2020 when the peak of the baby-boom generation will reach old age. As these compositional changes unfold, meeting the needs of an increasingly large and diverse patient population will certainly pose a challenge to geriatric practitioners.
References


Table 1. Rates for Select Chronic Conditions, Impairments, and Activity Limitations of the 65 and older Population, 1984 and 1994

<table>
<thead>
<tr>
<th>Select Chronic Conditions</th>
<th>Men</th>
<th>Men</th>
<th>Women</th>
<th>Women</th>
</tr>
</thead>
<tbody>
<tr>
<td>Arthritis</td>
<td>405.7</td>
<td>428.6</td>
<td>547.7</td>
<td>553.5</td>
</tr>
<tr>
<td>Cataracts</td>
<td>101.5</td>
<td>129.6</td>
<td>183.6</td>
<td>192.4</td>
</tr>
<tr>
<td>Cerebrovascular disease</td>
<td>82.6</td>
<td>53.1</td>
<td>53.8</td>
<td>60.5</td>
</tr>
<tr>
<td>Diabetes</td>
<td>194.7</td>
<td>107.3</td>
<td>108.5</td>
<td>96.9</td>
</tr>
<tr>
<td>Emphysema</td>
<td>73.7</td>
<td>67.9</td>
<td>20.1</td>
<td>29.6</td>
</tr>
<tr>
<td>Hardening of arteries</td>
<td>93.0</td>
<td>69.1</td>
<td>84.8</td>
<td>41.3</td>
</tr>
<tr>
<td>Heart disease</td>
<td>334.9</td>
<td>360.5</td>
<td>309.7</td>
<td>299.4</td>
</tr>
<tr>
<td>Hypertension</td>
<td>311.2</td>
<td>319.5</td>
<td>471.9</td>
<td>395.8</td>
</tr>
</tbody>
</table>

Impairments / 1000 population

| Hearing impairment                           | 352.1 | 354.1 | 283.3 | 238.0 |
| Visual impairment                            | 114.9  | 94.6  | 91.3  | 75.3  |
| Osteopathic impairment/deformity             | 142.7  | 153.7 | 193.2 | 174.1 |

Activity limitation (% reporting)

|                                              | 38.9  | 36.9  | 39.0  | 39.1  |

Source: Tables 58, 68 and 70 (34; 35)
Table 2. Expected Changes in Population Health Measures
Under Four Theoretical Perspectives

<table>
<thead>
<tr>
<th>Theory</th>
<th>Total Life Expectancy</th>
<th>Disabled Life Expectancy</th>
<th>Active Life Expectancy</th>
<th>Active Life Expectancy/Total Life Expectancy</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pandemic of Chronic Diseases</td>
<td>↑</td>
<td>↑</td>
<td>0</td>
<td>↓</td>
</tr>
<tr>
<td>Compression of Morbidity</td>
<td>0</td>
<td>↓</td>
<td>↑</td>
<td>↑</td>
</tr>
<tr>
<td>Life Span Expansion</td>
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<td>↑</td>
<td>↑</td>
</tr>
<tr>
<td>Dynamic Equilibrium</td>
<td>↑</td>
<td>↑</td>
<td>↑</td>
<td>?</td>
</tr>
</tbody>
</table>

↑ = increase  ↓ = decrease  0 = no change  ? = unknown

<table>
<thead>
<tr>
<th>At birth</th>
<th>Change in Total Life Expectancy (in years)</th>
<th>Change in Disabled Life Expectancy (in years)</th>
<th>Change in Active Life Expectancy (in years)</th>
<th>Change in Active Life as Percent of Total Life Expectancy (in percentage points)</th>
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<tbody>
<tr>
<td></td>
<td>1970-1980</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Males</td>
<td>3.1</td>
<td>2.4</td>
<td>0.7</td>
<td>-2.7</td>
</tr>
<tr>
<td>Females</td>
<td>3.0</td>
<td>2.9</td>
<td>0.1</td>
<td>-3.1</td>
</tr>
<tr>
<td>At age 65</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Males</td>
<td>1.2</td>
<td>1.0</td>
<td>0.2</td>
<td>-2.9</td>
</tr>
<tr>
<td>Females</td>
<td>1.6</td>
<td>1.4</td>
<td>0.2</td>
<td>-3.7</td>
</tr>
<tr>
<td></td>
<td>1980-1990</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Males</td>
<td>1.7</td>
<td>0.1</td>
<td>1.6</td>
<td>0.3</td>
</tr>
<tr>
<td>Females</td>
<td>1.2</td>
<td>0.1</td>
<td>1.1</td>
<td>0.2</td>
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</tbody>
</table>

Source: Authors' tabulations based on (30)
Figure 1. Trends in Age-Specific Death Rates 1970-1995 by Sex

Source: (2)
Figure 2. Survival Curve for U.S. Females, 1990, and Hypothetical Disability and Morbidity Curves

Source: Bell, Wade, and Goss, 1992, and authors' tabulations.