

The Compression of Morbidity: Near or Far?

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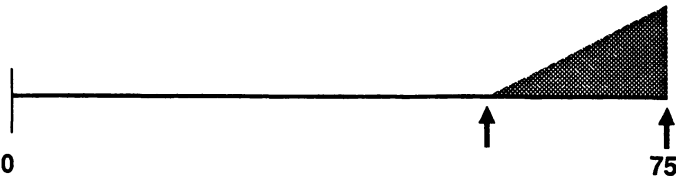
AN ATTRACTIVE MODEL FOR THE NATIONAL HEALTH consists of a collective, full, and vigorous life terminated by a swift and painless decline ending in death. The "compression of morbidity" hypothesis envisions reduction of the national illness burden by postponing the age of onset of chronic infirmity relative to average life duration so that the period of morbidity is compressed between an increasing age of onset and a relatively fixed life expectancy. The compression of morbidity thesis postulates that (a) if the morbid period is defined as that period from the onset of chronic infirmity until death, and (b) if the time of occurrence of such morbid events can be postponed, and (c) if adult life expectancy is relatively constant, then (d) morbidity will be compressed into a shorter period of time (Fries 1980, 1981, 1983, 1988).

The future national health may be represented by two alternative scenarios, one "hopeful" and one "pessimistic." In figure 1 a hypothetical current morbidity is schematically compared with that under these two future scenarios, each with an average life expectancy increased to 80 years. Technological or medical interventions employed after the onset of illness (scenario 1) may result in life extension, but since these interventions do not affect the onset of disease, morbidity is increased. On the other hand, primary prevention (scenario 2) might act to delay the onset of infirmity and thus to reduce morbidity.

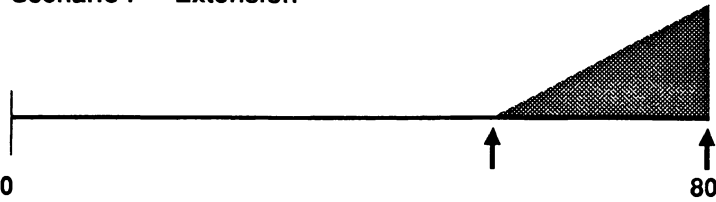
With chronic diseases accounting for the great majority of mor-

Compression or Extension of Morbidity

Hypothetical Present Morbidity



Scenario I — Extension



Scenario II — Compression

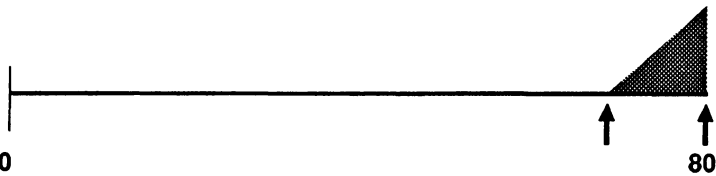


FIG. 1. Possible scenarios for future health. Since chronic morbidity is overwhelmingly concentrated in the later years, the national illness burden results from a dynamic interplay between relative movement of the two arrows, one representing the average of onset of illness or infirmity and the second representing the average age at death.

bidity, and with the “incubation period” for most of these chronic illnesses spanning many decades prior to first symptoms, the potential for preventive strategies is manifest. Less clear are the relative effects of such strategies upon morbidity and upon mortality; this represents the central question to be addressed here. The ten years since initial presentation of the compression of morbidity hypothesis (Fries 1980) provide a perspective from which to review recent trends; emerging data demonstrate a slowing of increases in life expectancy and a delay in the age of onset of major chronic diseases.

History and Predictions of the Model

Before 1980 the fraction of a typical life spent in ill health appears likely to have been increasing as chronic diseases replaced acute conditions as the primary causes of death (Fries 1981). Average life expectancy, after a dormant period in the 1960s, rose rapidly again in the 1970s. The gap between male and female life expectancy widened steadily (Faber 1986). On the other hand, there were hints that a change might lie ahead. The most important causes of mortality (atherosclerosis, lung cancer, automobile trauma) disproportionately affected males, and, thus, there were greater opportunities for improvement in males. The increasingly documented risk-factor models (Fries 1981) implied the potential for postponing the onset of chronic disease. National improvement in risk-factor prevalence was occurring and might be expected, after a lag period, to delay onset of disease. Increasing frailty (as opposed to specific disease) of the very old, observed clinically, argued against further marked increases in life expectancy at advanced ages (Fries 1981).

From this base, the compression of morbidity thesis suggested (Fries 1980, 1981) that, if these trends continued, life expectancy gains at advanced ages would slow, that the male/female life expectancy gap would narrow, that reductions in age-specific incidence rates for chronic illness would exceed reductions in age-specific mortality rates, and that randomized controlled trials of preventive interventions should show greater effects on morbidity than on mortality. These predictions were in contrast to standard projections (Faber 1986), which foresaw continued rapid gains in adult life expectancy and a widening male/female gap. Relevant data which address the validity of the model have emerged over the past ten years.

The National Health Habit Experiment

In 1964 the surgeon general officially declared that cigarette smoking was hazardous (U.S. Department of Health, Education, and Welfare 1964). At about the same time, the diet/heart disease hypothesis began to amass strong supportive evidence (Farquhar 1987). A series of national pronouncements (LaLonde 1974; U.S. Department of Health, Education, and Welfare 1979) urged good health habits, particularly smoking cessation, decrease in dietary fat, moderate alcohol consump-

tion, seat belt use, and regular exercise. The evidence for causal relations between habits and health, validating the Framingham and other risk-factor models, became strong (Farquhar 1987).

The public response, while inadequate in the lower-income groups, has been dramatic (U.S. Department of Health and Human Services 1987). Per capita tobacco consumption is down by 40 percent since 1963 and the rate of decline is accelerating, aided by public restrictions on smoking behavior. Use of butter has declined by more than one-third, whole milk and cream by one-quarter, and animal fats and oils by 40 percent. Use of vegetable fats and oils is up by 60 percent and fish consumption by over 25 percent (Kaplan 1984). Over 30 million Americans report that they practice regular aerobic activity. Automobile seat-belt use, once rare, exceeds 70 percent in many regions. Particulate air pollution is down by two-thirds from its peak, lead exposure nearly by 10-fold, and carbon monoxide by one-third (U.S. Department of Health and Human Services 1987).

The magnitude of these "interventions" suggests that a natural experiment is underway. Indeed, these national changes, occurring slowly but on a large scale, are equal to or greater than those changes achieved in the randomized trials of primary prevention discussed below. One should be able to use results in closely observed experimental groups to develop an expectation for results of the ongoing national "experiment." The early years (1970s) of the "experiment" were associated with striking decreases in mortality rates, particularly in the senior years. As average life expectancy increased, however, mortality changes (discussed below) have slowed markedly in the 1980s. The randomized-controlled trial data (also discussed below) suggest that in populations and years when life expectancy is already good, the gains from improved health habits are expressed primarily in terms of prevention of morbid effects, with only small mortality effects.

Decreasing Age-specific Incidence of Certain Major Chronic Diseases

Data on trends in morbidity are weak; consequently, interpretations of available data have been controversial (Kovar 1987; Schneider and Brody 1983). Studies employing interview data through the late 1970s generally suggest little change in health status, although some have argued worsening (Verbrugge 1984) and others, using transformations of

the data, improvement (Palmore 1986). The data do not speak convincingly to either conclusion, yet the pessimistic view is most frequently voiced. Barsky (1988) recently has termed this tendency for declining satisfaction with personal well-being despite dramatic improvement in health "the paradox of health."

Yet, despite the absence of hard evidence many of the major chronic diseases *must* be occurring later in life if the prevailing risk-factor models are accurate, because these models stipulate that if the accumulation of toxic exposures (e.g., pack-years of cigarette smoking, total animal fat intake) is slower, the clinical threshold must be reached later in life (Farquhar 1987; Stern 1979; Ueshima, Tatara, and Asakura 1987).

For more precise data one must look to studies of specific problems. It is appropriate to focus first on those problems which make the largest contributions to national morbidity, such as atherosclerosis (the single largest contributor), lung cancer (the most frequent major malignancy), and automobile accidents (the largest cause of major trauma). The preponderance of data demonstrates that most of the recent improvement in these disease conditions has been in age-specific incidence rather than in improved survival after occurrence (Goldman and Cook 1984; Pell and Fayerweather 1985). For other major diseases, such as Alzheimer's disease, Parkinson's disease, and urinary incontinence, no data on trends in incidence rates are available, and there obviously can be no assurance that the encouraging trends in some areas may be generalized to all disease areas.

Pell and Fayerweather (1985) studied trends in the incidence and mortality of myocardial infarction in employees of the DuPont company over a 26-year period and concluded that the major source of the decline in mortality was a reduction in incidence. A total decline of 28.2 percent in incidence was observed. The slope of the decline in incidence, calculated from the DuPont data, was significantly greater ($p < .01$) than declines in slopes of either 24-hour or 30-day mortality. As predicted by the compression of morbidity model, effects on incident age were greater than effects on survival, and declines were most striking at younger ages (rectangularization). Males averaging 30 years of age showed incidence-rate declines of 50 percent; those of age 40, 39.4 percent; those of age 50, 25.9 percent; and those of age 60, 22.3 percent. The incidence declined by 1.17 percent per year; adult male life expectancy over the same period increased by less than one-third of a percent per year.

Other studies support these observations. Hospital discharge survey data show steadily declining rates of hospitalization for myocardial infarction (National Center for Health Statistics 1968). The Kaiser-Permanente study in Northern California confirmed decreased incidence rates of acute myocardial infarction over time (Friedman 1979). The Minnesota Heart Survey showed a significant decline in hospitalization rates for acute myocardial infarction between 1970 and 1980 (Gillum et al. 1983). Unlike most other studies, an increase in four-year survival after heart attack was also noted, attributed in part to better short-term survival coupled with secondary prevention (e.g., exercise, diet) and continued medical care. Analysis of short-term stays in United States hospitals over time suggests that the average age of hospitalization for cardiovascular diseases increased by four years over a 13-year period while life expectancy from young adult (pre-heart attack) ages (age 30) increased only two years over the same period (National Center for Health Statistics 1968).

Lung cancer incidence rates also appear to have declined. Cure rates for these tumors have remained essentially constant (Bailar and Smith 1986); hence, incidence rates parallel mortality rates. Recently, declining mortality rates for lung cancer in men have been documented (Horn and Kessler 1986). A decrease of four percent occurred from 1982 to 1983, and the decline is continuing. Again in harmony with the compression of morbidity model, declines have been more marked at earlier ages; the decline for men under the age of 45 began in 1973, and for men aged 45 to 54 in 1978. Thus, changes in overall incidence rates are not evenly spread throughout the life span but occur earlier and to a greater degree in younger individuals. The lung cancer risk-factor model, with a lag association with smoking behavior, predicts that female lung cancer incidence should begin to decrease over the next few years.

Obviously, not all diseases are associated with modifiable risk factors, but the demonstration of the phenomenon of delayed onset in even a few of the most prevalent conditions is heartening. Atherosclerosis and lung cancer together account for 51 percent of mortality and a major amount of morbidity (National Center for Health Statistics 1989). It would seem likely that the same effects could be achieved in other risk-factor-driven chronic conditions such as emphysema, cirrhosis, colon cancer, breast cancer, oral cancers, and others. Effects of automobile seat belt laws are discussed below.

Randomized Controlled Trials of Primary Prevention

The compression of morbidity model requires that the effects of preventive interventions must be greater on morbid events than on mortality. A number of major, large, randomized controlled trials of primary prevention in cardiovascular disease provide data on this point.

Two critically important conclusions from these studies have gone largely unnoticed. The first is that effects on total mortality have been small. The second is that effects upon morbid events have been very large. Four of the most prominent studies (table 1) serve to illustrate these points. The MRFIT study (Multiple Risk Factor Intervention Trial Research Group 1982, 1986) examined the results of smoking cessation counseling, treatment of hypertension, and dietary fat reduction. The Lipid Research Clinics (LRC) (1984) study employed cholestyramine for cholesterol reduction, the Physician's Aspirin Study (Steering Committee of Physicians' Health Study Research Group 1989) used one aspirin tablet every other day, and the Helsinki Heart Study (Frick et al. 1987) used gemfibrosil for cholesterol reduction.

Total mortality was nearly identical in intervention and control groups in all of these studies; indeed, two of the four studies had more deaths in the intervention group. Coronary deaths sometimes were reduced but were compensated by increased numbers of fatalities in other categories. In contrast, measures of morbidity, often representing chronic symptomatic conditions such as angina pectoris and intermittent claudication, were consistently and highly significantly reduced. Table 1 lists all morbid events tabulated in these studies; unfortunately, study design issues of tabulating morbidity effects were not completely addressed and results are obviously incomplete. Nevertheless, taking these studies together there were 595 deaths in the intervention groups (int.) and 600 in the control groups (cont.). For serious morbid conditions, there were 2,556 in the intervention groups and 3,116 in the control groups. These differences are consistent from study to study.

A similar pattern is present in all other studies of primary prevention, but these are less instructive because of failure of the intervention to have a consistent effect, obvious toxicity of the intervention, failure to report total mortality, or failure to assess morbidity at all (Hebert et al. 1988; Hjermmann et al. 1981; Peto et al. 1988; Turpeinen et al.

TABLE 1
Major Randomized Trials of Primary Prevention

	Number of men	Duration	Deaths			Coronary deaths			Morbidity events			
			Int.	Cont.	Diff./ (%)	Int.	Cont.	Diff./ (%)	Int.	Cont.	Diff./ (%)	Morbidity/mortality
MRFIT ¹	12,866	7 yrs	265	260	-5(-2)	115	124	9(7)	1,366	1,628	262(16.1)***	262/-5
LRC ²	3,806	7 yrs	68	71	3(4)	44	32	12(27)	906	1,112	206(18.5)***	206/3
Physicians ³	22,071	5 yrs	217	227	10(4)	10	28	18(36)**	239	305	66(22)**	66/10
Helsinki ⁴	4,081	5 yrs	45	42	-3(-7)	14	19	5(26)	45	71	26(37)*	26/-3

Note: Int. = intervention group, Cont. = control group, Diff. = difference * = p < .05, ** = p < .01, *** = p < .001.

¹ Morbidity events angina pectoris, intermittent claudication, congestive heart failure, peripheral vascular disease, stroke, accelerated hypertension, left ventricular hypertrophy, impaired renal function, total nonfatal coronary events.

² Morbidity events definite or suspect nonfatal coronary, positive exercise test, angina, coronary bypass surgery, congestive heart failure, intraoperative myocardial infarction, resuscitated coronary collapse, TIA, brain infarct, intermittent claudication.

³ Morbidity events nonfatal coronary, nonfatal stroke.

⁴ Morbidity events nonfatal coronary.

1979; Committee of Principal Investigators 1978; Veterans Administration 1970). Some studies of secondary prevention by antihypertensive treatment, particularly in severe hypertension, have shown total mortality reduction, but again the effects upon morbidity have been greater (Hebert et al. 1988). Other reviewers have recently discussed these same observations (Oliver 1988; McCormick and Skrabanek 1988).

The failure of experimental, primary prevention studies to demonstrate reduction in overall mortality rates has several possible explanations. First, the association between national habit changes and declining national mortality rates suggests that an effect on mortality is likely to be present; thus, it should be seen if enough people were studied for long enough. Second, unforeseen toxicity of the intervention (e.g., treatment of mild hypertension in the MRFIT study, aspirin-induced hemorrhagic stroke) may have produced some offsetting deaths; this was certainly true in the clofibrate study (Committee of Principal Investigators 1978). Third, all of the studies could have had "bad luck."

Perhaps more important, the "competing risk" statistical models commonly used assume that the individual "saved" from a coronary death is at *average* risk for death from other causes—an assumption which is not likely to be true. The "competing risk" of senescence is not included in these models. (While age distributions of study deaths have not been reported, study deaths are likely to have been concentrated in the older individuals, and contributions of senescence seem likely.) Competing risk-model projections in the United States usually project a life expectancy increase of five to ten years in life expectancy from birth from total elimination of atherosclerosis (Manton 1987; Metropolitan Life 1989), yet in Japan, where the prevalence of atherosclerosis is extremely low, the benefit seems to be more on the order of one year. Cerebrovascular disease (arteriosclerosis) is prevalent in Japan, and deaths from this cause tend to make up for the decreased amount of cardiovascular disease. Biologically, intimal plaques (atherosclerosis) and hardening of the arteries (arteriosclerosis) are quite different processes, with the latter showing more characteristics of a manifestation of senescence. With decreasing salt intake and better control of hypertension in Japan, it will be of considerable interest to follow trends in longevity and cerebrovascular disease in that country. Similar insights may be gained by inspection of data from different states in our own country; lifestyles are arguably best in Utah due to the dominant presence

of the Mormon church, yet longevity in Utah is only slightly better than average from age 65 (1980: 17.2 versus 16.5) and is actually below average from age 85 (5.9 years versus 6.0) in that state (Metropolitan Life 1987).

The central points here are, first, that it has been experimentally shown to be easier to decrease morbidity than to decrease mortality in some major diseases, and, second, that it is likely that health habit changes (such as reduction of smoking or saturated fat intake) will have less effect on life expectancy than commonly projected by statistical models which do not include a function for senescence per se and which assume independence between probabilities of different fatal events in the same individual.

Associations between Health Habits and Morbidity

An increasing number of studies now link better health habits with substantially lower morbidity, contradicting the occasional assertion that better health habits might lead primarily to longer life and thus to increased need for medical care. Control Data Corporation prospectively studied approximately 15,000 employees over a three-year period, using medical claims costs to represent morbidity (Milliman and Robertson, Inc. 1987). Persons not exercising at the outset of the study had 114 percent higher nonmaternity claims costs, used 30 percent more hospital days, and were 41 percent more likely to have annual claims over \$5,000 than those who exercised the equivalent of walking 1.5 miles four or more times a week. Those more than 30 percent above desirable weight had 11 percent higher medical claims, 45 percent more hospital days, and included 48 percent more people with major claims. Smokers, those with high cholesterol levels, and those rarely using automobile seat belts had similar increases. Similar results were found with number of claims, dollar amount of claims, and number of claims over \$5,000.

Lane et al. (1987) found that regular long-distance runners had only two-thirds as many physician visits as matched community controls. Development of disability in individuals aged 52 to 74 was nearly imperceptible in these regular exercisers and occurred 10 times more rapidly in the control group. Appropriate controls for self-selection bias

were included. Runners had 40 percent more bone density in the lumbar spine by CT scan and both males and females maintained normal bone densities through the period studied; the morbidity target here is the 650,000 osteoporotic fractures occurring yearly (Lane et al. 1986; Brody 1985). The effects of exercise conditioning upon the ability to perform a variety of physical tasks at advanced ages has been repeatedly reported (Bairey 1986; Bortz 1982; Bruce 1984; Larson and Bruce 1986; Paffenbarger et al. 1986; Spirduso 1980). The social science literature abundantly documents both increased variability between individuals with age and the ability of well-conceived interventions to improve performance (plasticity) at any age. Improvements in cognitive function and social behavior associated with such interventions have been substantial, even with subjects well over 70 years of age (Baltes 1986; Berkman and Syme 1979; Plemons, Willis, and Baltes 1978).

The introduction of seat belt laws provides a complementary perspective upon the relationships of reductions in mortality and in morbidity. Introduction of such laws typically reduces automotive fatalities by about 25 percent, and automotive injuries by about 50 percent. Since serious injuries are much more common than deaths, the effect on morbidity is magnified. Illinois reported an estimated 55 to 60 lives saved and 8,000 serious injuries prevented in the first year after enactment of seat belt legislation. Safety belt wearers had a 60.1 percent reduction in severity of injury, a 64.6 percent decrease in hospital admissions, and a 66.3 percent decline in hospital charges (Orsay et al. 1988). Morbidity from automobile injuries is, of course, spread throughout the life span, but a substantial number of lifelong problems (e.g., paraplegia, whiplash) result. Moreover, reduction in morbidity by decreasing the effects of acute trauma at any age affects the broad goal of reducing total lifetime morbidity, as does prevention of nonfatal episodic medical events such as gall bladder disease or hernias.

Medical Care in the Last Year of Life

Economic projections for future medical costs generally assume that longer lives are necessarily more expensive per year of life; that is, that economic morbidity will be extended by increasing life expectancy. Some 18 percent of lifetime medical costs, however, occur in the last year of life. In many studies, notably those of Roos, Montgomery, and

Roos (1987) and Fuchs (1984), these effects are noted to be further concentrated in the last one or two months of life.

Recognition of this phenomenon is critical for projection of future health care costs, since it represents compression of economic morbidity. Medical expenses are closely tied to time of death, more loosely tied to time from birth. In simplified terms, at any given year of age two subpopulations are present, one made up of those who will die within the year and one of those who will not. The first subgroup is very expensive, the second relatively inexpensive. For each successive year of age, the relative proportions of these two subpopulations change so that there is a greater percentage of those high-cost subjects who will die within the year and a smaller percentage who will live through the year (Fuchs 1984). This phenomenon essentially accounts for increased health care costs per year of age above the age of 65; as demonstrated by Fuchs (1984), adjustment for the percentage who will die within the year removed the increases in hospital costs otherwise seen with each increasing year.

An important problem in many economic prophecies of future health care costs for the aged (usually very pessimistic) thus has been use of an assumption of *lower* age-specific mortality rates while at the same time assuming *constant* age-specific health care costs. If death is postponed, the costs of dying are likely to be postponed also. Interestingly, the data of Roos, Montgomery, and Shapiro (N.P. Roos personal communication 1987) demonstrate that hospitalization costs in the months prior to death are essentially constant for all age groups, including ages 75 to 84 and those aged over 85.

Changing Demographic Trends

Current life expectancy predictions, including the 1987 and 1988 predictions from the Social Security Administration, continue to project the trends of the 1970s into the future. In contrast to the compression of morbidity thesis, they thus predict steadily continuing gains in life expectancy over the next century and a gradual widening of the male/female gap in average life expectancy (Social Security Administration 1987, 1988). Recent national mortality data (U.S. Department of Health and Human Services 1988; Metropolitan Life 1988) suggest that

TABLE 2
Changes in Life Expectancy, U.S. 1976-1986

	1976	1977	1978	1979	1980	1981	1982	1983	1984	1985	1986	1987	1988
From birth													
All	72.9	73.3	73.5	73.9	73.7	74.2	74.5	74.6	74.7	74.7	74.8	74.9	74.9
Change	.3	.4	.2	.4	-.2	.5	.3	.1	0	0	.1	.1	0
Males	69.1	69.5	69.6	70.0	70.0	70.4	70.9	71.0	71.2	71.2	71.3	71.5	71.4
Females	76.8	77.2	77.3	77.8	77.4	77.8	78.1	78.1	78.2	78.2	78.3	78.3	78.3
Male/Female gap	7.7	7.7	7.7	7.8	7.4	7.4	7.2	7.1	7.0	7.0	7.0	6.8	6.9
From age 65													
All	16.1	16.4	16.4	16.7	16.4	16.7	16.8	16.7	16.8	16.8	16.9	17.0	16.9
Change	.5	.3	.0	.3	-.3	.3	.1	-.1	.1	0	.1	.1	-.1
Males	13.7	13.9	14.0	14.2	14.1	14.3	14.5	14.5	14.6	14.6	14.7	14.8	14.8
Females	18.1	18.3	18.3	18.6	18.4	18.6	18.7	18.6	18.6	18.6	18.6	18.6	18.6
Male/Female gap	4.4	4.4	4.3	4.4	4.3	4.3	4.2	4.1	4.0	4.0	3.9	3.8	3.8

Sources: U.S. Department of Health and Human Services 1988; Metropolitan Life 1987, 8-14; 1988, 18-23; 1989, 26-32; National Center for Health Statistics 1987.

this is unlikely to be the case; indeed, the 1982 predictions (Faber 1986) are already off by a year in terms of the male/female gap in life expectancy from birth, and by nine-tenths of a year in terms of female life expectancy from the age of 65.

Table 2 records actual changes in average life expectancy in the United States over the years of 1976 through 1988 from birth and from the age of 65 (U.S. Department of Health and Human Services 1988; Metropolitan Life 1989). Increases in male life expectancy have rather closely followed the 1982 projections (Faber 1986). Female life expectancy from birth, however, projected to be 79.3 in 1988 was instead 78.3, and female life expectancy from the age of 65 projected at 19.5 was in fact 18.6 (figure 2). Female life expectancy from birth thus has continued to rise, although more slowly, in the 1980s. Most significantly, however, female life expectancy from the age of 65 has not increased over the nine years since 1979. The compression of morbidity hypothesis predicts that female life expectancy at advanced ages will plateau as ultimate limits are first approached by females and that males, with lower life expectancies, will begin to plateau at a later point. Experience in this decade suggests that this may be occurring. Note in figure 2 the successive reductions in projections from the Social Security Administration.

It is premature to suggest that there will be no further increases in female life expectancy from the age of 65; indeed, our own projections of ideal life expectancy (table 3) suggest that an upper limit of 20 years or more might be eventually possible. But, it seems likely to this author, based on the randomized controlled trial observations which suggest what might be possible as well as the current plateau in longevity gains, that we will never again see the rates of growth in life expectancy at advanced ages that occurred during the years of major decline in chronic vascular disease in the 1970s, except perhaps in certain disadvantaged subgroups. It also seems likely that the current Social Security projections, meant to bracket all reasonably likely scenarios, may still be too high, at least for females.

Estimating the Limits to Life

In 1980, based on national data through 1976, we developed crude estimates of approximately 85 years for maximum average life expectancy (Fries 1980). Unlike many extrapolation models which assume constant

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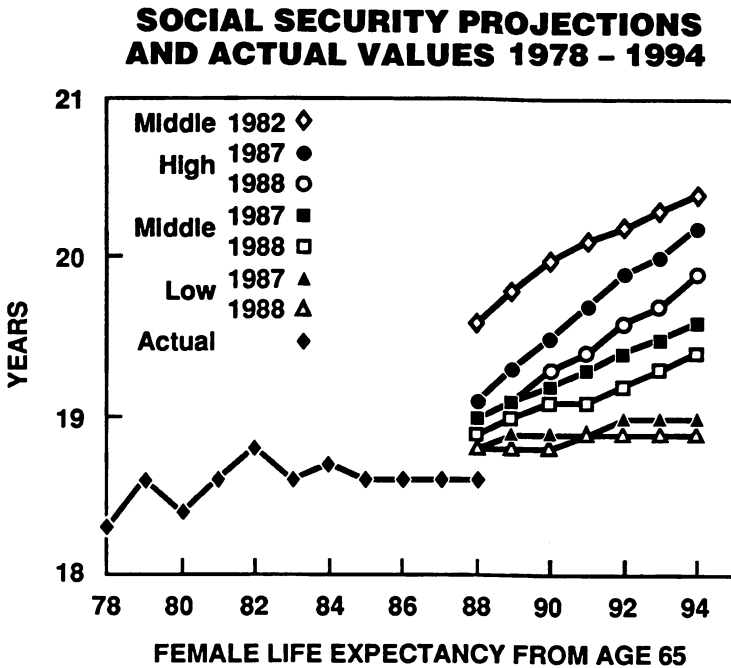


FIG. 2. Trends in female life expectancy from age 65. Despite predictions of rapid increase, data demonstrate late change over the past ten years. Male life expectancy from the age of 65 has continued to rise over this period. The Social Security Administration presents three series of estimates; the middle series is generally used in policy studies. More recent projections have been more conservative with regard to future improvement.

Sources: Statistical Bulletin, Metropolitan Life Vol. 68 pp. 18-23, 1988. Statistical Bulletin, Metropolitan Life Vol. 69 pp. 26-32, 1989. Social Security Area Population Projections, 1987; Actuarial Study No. 99, SSA Pub. No. 11-115 46, Actuarial Study No. 102, 1988, Pub. 11-11549, Actuarial Study No. 87. SSA Pub. No. 11-11534, 1982.

percentage increases each year, the technique introduced does not assume a potentially infinite life expectancy. All techniques for predicting future longevity, including ours, require assumptions, and as recently and thoroughly discussed, these assumptions are necessarily arbitrary and reflect the opinions of the authors (Olshansky 1988). For example, the Office of the Actuary assumes a 1.2 percent annual increase and others an annual increase of as much as 2 percent (Guralnik, Yanagishita, and Schneider 1988). It is important to examine the assumptions as closely as the predictions themselves, since the assumptions determine the results.

The technique we introduced is based upon three assumptions: (1)

TABLE 3
Linear Regression Estimates of Maximum Average Life Expectancy

	Males		Females		All	
	Data through 1976	Data through 1986	Data through 1976	Data through 1986	Data through 1976	Data through 1986
From 1900						
Mean age	79.2	79.8	83.0	84.2	81.1	82.0
From 1950						
Mean age	81.1	83.0	91.7	90.6	86.4	86.8
Last 10 years						
Mean age	84.4	83.8	92.4	85.9	88.4	84.9
Last 20 years						
Mean age	83.5	84.3	94.6	89.1	89.1	86.7

that all deaths prior to the age of 65 are premature, (2) that at a point where all premature deaths before the age of 65 have been eliminated all premature deaths over that age will also have been eliminated and the natural maximal life span reached, and (3) that linear extrapolation is a reasonable approximation. Given these assumptions, calculation of the point at which the trend line of average expected age at death from birth (currently 74.8) will intersect with the trend line of average expected age at death having achieved age 65 (currently 81.9) yields an estimate of maximum average life expectancy. With this technique one might have found that the lines did not intersect (suggesting that life expectancy could be indefinitely increased), that the point of intersection was at advanced ages of perhaps 100 to 115 (suggesting that the theoretical limits were too distant to have much immediate relevance), or that the intersection point was relatively close at hand.

With ten additional years of data now available, and with the period of extrapolation thus shortened, such estimates should be more reliable. Table 3 presents a summary of linear regression calculations based on national life expectancy data for various periods since 1900 for males and females. Logically, the "age at death from birth" trend line and the "age at death having achieved age 65" trend line cannot actually cross; that is, the latter must always be above the former. As limits are approached, the rate of increase should decrease toward an asymptote

which represents a residual unavoidable burden of disease and accidents. Hence, linear models are not strictly appropriate. On the other hand, R square values for the fit of straight lines to these data are generally above .9, indicating that not much variability in the age estimates is left to be explained by a more complicated model. Any asymptotic curve drawn through the data will lie below the projected intersection point; hence, the estimates are in this sense conservative.

The range of estimates obtained gives a rough idea of the sensitivity of the model to differing selections of data periods for analysis. Results using more recent data are closely similar to earlier estimates; the grand mean of the several estimates was 86.3 years using United States data through 1976 and is 85.1 years using data through 1986. Thus, there is little reason to alter our earlier estimates significantly. With female life expectancy above the age of 65 already within 5 years of the theoretical limits and with an estimated standard of deviation of 7 to 8 years representing genetic variation, these projections suggest a possible explanation for the slowing of increases in female life expectancy.

A gap between male and female maximum life expectancies continues to be projected by these techniques but may have narrowed somewhat in the projections which use more recent data. Life expectancies currently are greatest in Japan; use of these same techniques with Japanese data suggests a difference of only 1.5 years between men and women, and intersection points are about a year higher than with United States data. Another line of reasoning suggests that the differences between men and women might ultimately disappear. Thus, male and female life expectancies converge when estimated from increasingly advanced ages, ultimately becoming identical. Life expectancy for both men and women from age 110 is 1.48 years (Faber 1986).

Slowing of the rate of increase in life expectancy above the age of 65 has important implications for estimating future morbidity. It is common to forecast that life expectancy increases also increase the risk of age-related diseases such as Alzheimer's disease, Parkinson's disease, and others (Guralnik, Yanagishita, and Schneider 1988). To be sure, the number of cases of such conditions is likely to increase as the cohort sizes of the older age groups increase. But, if life expectancy for the typical individual is relatively constant, then morbidity saved earlier in life can be truly saved unless the age-specific incidence of diseases such as Alzheimer's actually increases; the years not spent with symptoms of angina pectoris, dyspnea, or paraplegia are not replaced by subsequent

years in which these problems are complicated by chronic dementia. Under these constraints, if you get a chronic problem later in life, on average you will have it for a shorter period of time.

Caveats

All is not going well with the national health. AIDS is an important new problem without easy solutions. Intravenous drug abuse may be increasing. Violent death has become the leading cause of years of life lost in some subgroups (Massachusetts Medical Society 1989). Environmental problems remain, and some new ones are being uncovered. Lower socioeconomic groups are lagging seriously in terms of reducing health risks; it seems quite likely that major mortality improvements from both prevention and medical care are possible in disadvantaged populations. Nuclear, chemical, and biological weapons are stored in large stockpiles. The biological riddle of aging might be solved, yielding the possibility of greatly increased life expectancy with possibly a prolonged period of senescence. Differences in lifestyle and habit in individuals with differing education levels suggests a future increase in already apparent differences in health status (Pierce et al. 1989). Many important chronic diseases, such as rheumatoid arthritis, multiple sclerosis, Alzheimer's disease, and Parkinson's disease do not have identifiable risk factors.

The discussions above have been phrased broadly, emphasizing the possibility of substantially reducing the morbidity in large, perhaps dominant, disease and accident categories. There has not been an attempt to discuss all problems or to suggest that all have solutions. There will be trade-offs in the individual case, some adverse, as when a sudden death prevented in early life allows a subsequent chronic lingering illness. Morbidity from a shorter illness may sometimes increase more rapidly and be of greater magnitude. These instances are compensated by other instances where delay in onset of morbidity adds many years of fully functional well-being. Others have argued more pessimistic scenarios eloquently (Guralnik, Yanagishita, and Schneider 1988) and will undoubtedly find the discussions above to be incomplete or to lack balance. Some may fear that the compression of morbidity thesis is a disguised argument for reduction of funding for medical research (Guralnik, Yanagishita, and Schneider, 1988); this is not the case. The argument made here is that data demonstrate that

current preventive interventions reduce morbidity far more frequently than they do mortality in the major conditions for which data are available and that policies directed at delaying onset of symptoms thus, on balance, will decrease lifetime morbidity. The principal criticism made here is of predictions based upon assumptions of decreases in age-specific mortality rates, while at the same time (unreasonably) assuming that age-specific morbidity rates will remain constant.

Prevention versus Palliation

The medical demographics of successful aging require a decreasing period between two points: the average age at first infirmity and the average age at death. If the first increases faster than the second, there is compression of morbidity; if the reverse happens, there is extension of morbidity. Our traditional medical approaches, in essence, have neglected the point at which illness begins and focused attention instead on treating (or palliating) disease which has been clinically recognized. This approach tends to increase morbidity since life extension generally is attained at the cost of a loss in life quality. A second problem with this approach is that after-the-fact medical care is expensive and is likely to become increasingly so. Additional gains require higher technology and a higher price tag, and provide decreasing amounts of additional life. The cost of medical care at the margin inevitably increases.

The compression of morbidity model suggests a strategy to reduce the national morbidity. By focusing efforts upon moving the onset of the first chronic infirmity upward, years of healthy life may be gained. Indeed, any movement of this onset date to later ages will return health to the population compared with the alternatives. Even if total morbidity is not decreased, the rate of increase in morbidity will be slowed. Moreover, the behavioral and preventive initiatives required are relatively low in technological content and may prove to be less expensive than traditional medical approaches.

The future for the national morbidity thus largely depends upon a self-fulfilling prophecy. If we neglect preventive and behavioral issues, morbidity is likely to increase. The compression of morbidity arguments provide the theoretical basis for development of "successful aging" programs designed for reduction of morbidity and now are argued

strongly even by some of these initially critical of the concept (c.f., Guralnik and Kaplan 1989). If we continue to emphasize high technology and replacement of worn-out body parts, costs will increase. There is a potential option for a healthier and perhaps a less-expensive future, but our response to the choice is not yet recorded.

The important questions are hard to avoid. Is it time to develop and implement a national strategy for postponement of morbidity? Do we have the data that we need? Is the contemporary academic medical center appropriately structured for current national health needs? Do social incentives reward healthy behaviors? Are research resources for studies of prevention adequate? How can we extend the benefits of healthier lifestyles throughout all income groups?

The compression of morbidity is presently occurring, in part, and for some. Yet, further compression of morbidity is not inevitable; it requires the increasing attention of inertia-bound institutions to improvement of long-term outcomes and a decreasing emphasis upon technical process (Somers 1987, 1988). The public is increasingly aware of many of these considerations. The response of the health professions has been more sluggish. This is a time for reappraisal.

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