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Public Policy Implications of Declining Old-Age Mortality

Recent years have witnessed dramatic improvements in the longevity of the elderly population. Life expectancy for women at age 65 increased by 3.3 years, a 22 percent change, between 1950 and 1980.[1] Age-specific death rates fell by 29 percent for white females over 85 and by 19 percent for white males over 85.[2] Continuation of these trends could have major implications for public policy toward the elderly. If mortality rates continue to drop, the elderly population will be substantially larger than if the rates remain constant at their current level. The number of very elderly, those over age 85, could rise especially rapidly.

This paper explores some policy implications of the dramatic longevity gains that have occurred and are likely to continue. We focus on the potential burden, through demand for medical care and other resources, that the elderly are likely to create. Different views on this issue are possible. A pessimistic outlook would hold that improvements in life expectancy are likely to be associated with large increases in the costs of supporting the elderly. The elderly will grow more numerous, particularly at very old ages where support costs are greatest. These costs may be

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1. U.S. Department of Health and Human Services, Social Security Administration, Office of the Actuary, *Social Security Area Population Projections, 1984,* Actuarial Study 92 (Government Printing Office, 1984), table 9b.

2. Calculations are based on data in table 1. Mortality rates for nonwhites show an even greater decline, although they may be contaminated by measurement errors. These data problems are discussed in Ansley Coale and Ellen Kisker, "Mortality Crossovers: Reality or Bad Data?" (Princeton University, Office of Population Research, 1985).

particularly high for "marginal survivors"—those who would have died at earlier ages were it not for recent progress in reducing mortality. A more optimistic view is also possible. It would argue that the same forces that have led to recent declines in mortality might also be expected to lead to reduced morbidity and increased ability to function. Mortality reductions lower the number of the aged who are within a year or two of death. Since these are the years when support costs are highest, especially for health care, the social burden per aged person might actually decline.

The relative importance of these two effects is an empirical question that cannot be resolved by a priori argument. The choice of which view is more appropriate is, however, clearly an important policy issue. Projections of future medicare costs are extremely sensitive to the number and expected needs of potential beneficiaries. While much attention has focused on efforts to reduce medicare costs by reforming reimbursement procedures and changing the health care delivery system, there are limits to the savings available from these individual devices. Moreover, because of the typically one-shot character of individual cost-reduction measures, their impact on projected future costs is ultimately smaller than demand-related factors, such as health status, which cumulate year after year.

This paper surveys some relevant evidence and presents some new calculations bearing on the effects of mortality improvements. While the available data permit only tentative conclusions, it appears that reductions in morbidity associated with declining mortality have been counterbalanced by high morbidity rates among marginal survivors. As a consequence, the health needs of elderly persons at given ages have not changed very much.

Mortality Trends among the Aged

Mortality among the aged has declined dramatically since 1950. Life expectancy for white women at age 65, after rising by 1.9 years between 1950 and 1970, rose by 1.6 years between 1970 and 1980. For white men the change was 0.2 years between 1950 and 1970 and 1.2 years between 1970 and 1980 (see table 1). These increases in life expectancy were caused by dramatic reductions in mortality rates at all ages. Between 1950 and 1980 the death rate of male 85-year-olds fell from 221 per 1,000 to 178 per 1,000, or nearly 20 percent.

The table describes progress in reducing mortality among the aged over

Table 1. Death Rates and Life Expectancy, by Age and Sex, Selected Years, 1950–80

Measure	*1950*	*1960*	*1970*	*1980*
Crude death rate (per 1,000 people)				
Men				
55–59	18.8	17.8	17.7	13.9
65–69	40.7	40.5	40.5	33.2
75–79	90.1	87.0	86.9	80.7
85 and over	221.2	217.5	185.5	178.2
Women				
55–59	10.2	8.3	8.3	7.2
65–69	25.2	21.5	19.2	16.5
75–79	69.9	60.8	53.5	45.6
85 and over	196.8	194.8	159.8	140.4
Life expectancy (years)				
Men				
55	19.1	19.5	19.5	21.2
65	12.8	13.0	13.0	14.2
75	7.8	7.9	8.1	8.8
85	4.4	4.3	4.6	5.0
Women				
55	22.6	23.8	24.9	26.5
65	15.0	15.9	16.9	18.5
75	8.9	9.3	10.2	11.5
85	4.8	4.7	5.5	6.3

Sources: Death rates for 1950–70 from U.S. Department of Health, Education, and Welfare, National Center for Health Statistics, *Vital Statistics of the United States*, vol. 2: *Mortality*, pt. A (Government Printing Office, 1950, 1960, and 1970 issues). Death rates for 1980 and all life expectancies from Department of Health and Human Services, NCHS, *Vital Statistics of the United States, 1980*, vol. 2: *Mortality*, pt. A, sec. 6. Data are for white men and women only.

the past thirty years. We present data on white men and women, because demographic data for nonwhites at extreme ages are somewhat unreliable. Several patterns emerge from the mortality data. First, mortality reductions have been greater for women than for men, despite convergence between the sexes in patterns of employment, life-style, and rates of smoking. Second, mortality reductions do not exhibit any pattern across age groups, and there have been significant reductions even among the extreme aged. Despite forecasts of ultimate limits on life expectancy and the rectangularization of the life table, there is no evidence of rising mortality rates as more and more people reach extreme ages.[3] Third, the pace of mortality reductions has accelerated, with especially rapid progress being made during the 1970s.

3. For a discussion of the evidence on mortality among the extreme aged, see Kenneth G. Manton, "Changing Concepts of Morbidity and Mortality in the Elderly Population," *Milbank Memorial Fund Quarterly*, vol. 60 (Spring 1982), p. 192.

The second part of the table illustrates the substantial changes in life expectancy during this period. For women aged 75, life expectancy increased from 8.9 to 11.5 years between 1950 and 1980, an increase of 29 percent. The gain for men at age 75 was only 1 year during this period. Even at age 85, there were dramatic improvements: a 14 percent increase for men and a 31 percent gain for women. In 1950 an 85-year-old woman's life expectancy was only 0.4 years greater than that of an 85-year-old man. In 1980 the difference was 1.3 years.

The sources of these dramatic declines in mortality are not well established. The bulk of the increase in life expectancy at older ages appears to be the result of reduced death rates from cardiovascular diseases. The most recent report of the Social Security Administration's Office of the Actuary indicates that between 1968 and 1980 mortality from heart disease among men 65–69 declined at a 2.2 percent annual rate and mortality from vascular disease fell at a 4.9 percent rate. For women 65–69 the corresponding improvement rates were 2.5 and 4.9 percent. There have also been substantial reductions in death rates from digestive disease and diabetes for both sexes. Partially reflecting these changes, cancer death rates actually rose during this period at a 1.0 percent annual rate for men aged 65–69 and a 1.4 percent rate for women this age.[4]

The cause of these mortality reductions is far from clear. One possible explanation is improvements in access to medical care due to the enactment of medicare and medicaid in 1965. The timing of the acceleration in the mortality rates' decline supports this possibility. An alternative possibility is improved medical procedures for treating hypertension and heart attacks. Still another possible cause is improved diet and exercise among the aged. In all likelihood the decline in mortality can be traced to some combination of these and other factors.

Declines in mortality have potentially important effects on the composition of the aged population. The most obvious effect is that the average age of the elderly population will increase as more and more people survive to older ages. A more subtle but probably more important effect of reduced mortality is that the population at any given age will include marginal survivors who will be less healthy and less self-sufficient than the rest of the population.

The magnitude of the mortality declines is well conveyed by the percentage of the 1980 population at various ages who, if they reached age

4. Social Security Administration, *Projections, 1984*, p. 25.

50, would not have been alive had they faced the mortality rates of cohorts born ten, twenty, thirty, or forty years earlier. We focus on persons who would have reached age 50 to highlight changes for the elderly and to avoid contaminating our results with changes in infant mortality or other factors affecting younger persons.

The shares of marginal survivors are calculated using cohort life tables for persons born in the first year of each decade between 1850 and 1910. These data should be distinguished from those in synthetic life tables, the type commonly used in calculating life expectancies. In a synthetic table, the death rates at each age correspond to the probability that a person of that age in year t would die in year t. The death rates for different ages therefore correspond to different birth cohorts. In a cohort life table, a single birth cohort is followed throughout its life. The entry for age 65 in the 1910 cohort table would report the fraction of 65-year-olds in 1975 who died within one year.

We calculated the number of marginal survivors as follows. Let q_t^w denote the probability that a person born in year w dies between birthdays t and $t + 1$, conditional on living to age t. The probability of living to age t in birth cohort w, conditional on reaching age 50, is therefore:

$$S^w(t) = \prod_{i=50}^{t-1} (1 - q_i^w).$$

For persons of age $a > 50$ in 1980, a fraction $S^{1980-a}(a)$ of the members of the birth cohort who reached age 50 are still alive. If these persons had been born m years earlier, the comparable fraction would have been $S^{1980-a-m}(a)$. The proportion of the 1980 population that is accounted for by marginal survivors relative to the cohort m years earlier, $MS(a,m)$, is therefore:

$$MS(a,m) = \frac{[S^{1980-a}(a) - S^{1980-a-m}(a)]}{S^{1980-a}(a)}.$$

The results of the calculations are shown in table 2. Particularly at old ages, the share of marginal survivors in the population is very high. Fifteen percent of the 80-year-old men alive in 1980 and 35 percent of the 80-year-old women would have reached age 50 but not have been alive at 80, given the mortality experience of the cohort that preceded them by

Table 2. Percentage of Persons Alive in 1980 Who Would Not Have Been Alive Given Mortality Rates of Earlier Birth Cohorts, by Age and Sex

Earlier birth cohort (number of years preceding)	*Age in 1980*							
	60		*70*		*80*		*90*	
	Men	*Women*	*Men*	*Women*	*Men*	*Women*	*Men*	*Women*
10	3.9	0.9	3.4	2.9	5.0	10.8	16.2	30.4
20	3.8	1.4	6.0	8.0	8.0	22.2	36.3	53.0
30	5.4	3.6	8.5	14.4	15.1	35.2	58.4	72.3
40	6.5	6.1	10.5	20.7	25.1	46.2	58.7	76.2

Sources: Authors' calculations based on cohort life tables provided by Metropolitan Life Insurance Company, and supplemented with data from DHEW, NCHS, *Vital Statistics of the United States*, vol. 2: *Mortality*, pt. A, for years since 1974. Data are for white men and women only.

thirty years. The share of marginal survivors rises rapidly with age. For example, given the mortality experience of the cohort that preceded them by ten years, 0.9 percent of the 60-year-old women would not have been alive in 1980, 2.9 percent of the 70-year-olds, 10.8 percent of the 80-year-olds, and 30.4 percent of the 90-year-olds.

The dramatic importance of marginal survivors at extreme ages may be somewhat misleading, since the number of individuals alive at these ages is much smaller than at earlier ages. We therefore calculated the fraction of the population over 60 who would not have been alive if they had faced the life table of the cohort thirty years before them. Aggregating across age groups, more than 9 percent of the men and 16.9 percent of the women over age 60 in 1980 were marginal survivors. If people had faced the life table of those born forty years earlier, 12 percent of the men and 22 percent of the women would not have reached their current ages.[5]

Changes in mortality rates or other indicators of health status for the very old are difficult to interpret. The composition of the population has changed quite dramatically through time. Without general improvements in health, we would expect the large number of marginal survivors to reduce indices of health status at any given age. Of course, the dramatic reductions in mortality could have been accompanied by progress in lowering morbidity rates as well. We consider this possibility below. First we consider some implications of continuing reductions in mortality.

Demographic forecasts are notoriously difficult. The substantial mor-

5. We calculate the share of people alive in 1980 who would not have been alive if they faced the life table of m-years earlier as $[MS(60,m)*N_{60} + MS(70,m)*N_{70} + MS(80,m)*N_{80} + MS(90,m)*N_{90}]/(N_{60} + N_{70} + N_{80} + N_{90})$, where N_i is the number of persons of age i alive in 1980. We use data for every ten years because these are the only cohort life tables available to us.

tality gains of the last decade were largely unforeseen. Most observers expected a leveling off in the rate of decline of death rates among the elderly. Nonetheless, it is useful to consider the potential effects on the future population of continued mortality reductions. We rely on the Social Security Administration's Office of the Actuary, which computes three alternative scenarios reflecting different degrees of optimism about future mortality reductions. Alternative II assumes the continuation of current trends, with a gradual adjustment to moderate rates of progress in reducing mortality. Alternatives I and III, respectively, consider slower and faster progress.

Table 3 displays information on projected death rates under Alternative II. If current trends continue, the death rate at ages 75–79 for women is expected to decline 24 percent by 2000. At this point, it will be fully 48 percent below its 1960 level. By 2080 the death rates for women at all ages over 75 are projected to be approximately half their current level. For men progress is less dramatic but still implies a 40 percent mortality reduction at high ages between 1980 and 2080.

Table 4 shows the forecasts for life expectancy at age 65 under all three mortality scenarios. Dramatic improvements are clearly a possibility. Under Alternative II, the life expectancy for men at age 65 will rise by 22 percent, to nearly twenty years, by 2040. For women, even the pessimistic projections suggest life expectancies at 65 of more than twenty years by 2040. The optimistic scenario suggests more than twenty-five years by 2040 and over twenty-nine years by 2080.

These reductions in mortality have important implications for the size and structure of the aged population. In 1982 there were 27.5 million

Table 3. Actual and Projected Death Rates per 1,000 People, by Age and Sex, Selected Years, 1982–2080

	1982[a]		*2000*[b]		*2040*[b]		*2080*[b]	
Age	*Men*	*Women*	*Men*	*Women*	*Men*	*Women*	*Men*	*Women*
60–64	20.9	11.2	16.2	9.7	13.8	8.2	11.9	7.0
65–69	33.7	16.9	28.0	14.8	23.8	12.5	20.6	10.6
70–74	49.0	25.3	41.4	20.5	35.2	17.1	30.2	14.4
75–79	74.8	41.1	63.9	31.2	53.9	25.5	46.2	21.1
80–84	106.2	65.7	90.7	49.0	76.2	39.5	64.9	32.3
85–89	158.4	112.2	135.1	84.8	112.5	67.6	95.2	54.7
90–94	225.9	177.6	191.6	140.9	157.7	111.1	131.8	88.9

Source: U.S. Department of Health and Human Services, Social Security Administration, Office of the Actuary, *Social Security Area Population Projections, 1984,* Actuarial Study 92 (GPO, 1984). Projections assume continuation of current trends (Alternative II).

a. Actual.

b. Projected.

Table 4. Actual and Projected Life Expectancy at Age 65, by Sex, Selected Years, 1980–2080

	1980		2000		2040		2080	
Alternative[a]	*Men*	*Women*	*Men*	*Women*	*Men*	*Women*	*Men*	*Women*
Alternative I	14.0	18.4	14.8	19.5	15.6	20.6	16.4	21.5
Alternative II	14.0	18.4	15.7	20.7	17.1	22.5	18.5	24.3
Alternative III	14.0	18.4	16.6	21.8	19.9	25.5	23.3	29.1

Source: SSA, *Projections, 1984,* table 9b.

a. Alternative I assumes slower progress in mortality reduction than current trends; Alternative II assumes continuation of current trends; and Alternative III assumes faster progress than current trends.

persons aged 65 and over; this constituted 11.4 percent of the total population and 19.5 percent as many people as the population aged 20–64. By 2040 the Alternative II projections imply that those aged 65 and over will number 68.8 million, or 21.1 percent of the total population and 38.2 percent as many as those aged 20–64. The average age of those over age 65 will also rise. Even in the case of the pessimistic projections, the share of the aged population who are over 85 will rise from 9.7 percent in 1985 to 16.2 percent in 2040. The population at older ages will contain many marginal survivors. Of the 65-year-olds alive in 1965, 23.8 percent of the men and 44.6 percent of the women were still alive in 1985. The intermediate projections of the Social Security Office of the Actuary suggest that 39.6 percent of those between 65 and 70 in 2000 will be alive in 2020.[6] Less dramatic increases in the proportion of 65-year-olds living to be 75 can also be projected.

These data suggest that progress in reducing mortality is now having and will continue to have an important impact on the composition of the aged population. These effects are potentially important because there are great differences among the aged in the medical and institutional resources they require. In 1982, the most recent year for which data are available, per capita medical expenditures for persons over 85 were about twice as great as those for persons between 65 and 66.[7] The rate of institutionalization was 11.3 times as great for men over age 85 as for those between 65 and 74. For women the comparable ratio was 14.6.[8] These figures suggest that the dependency burden of the elderly population could increase sub-

6. Based on data in Social Security Administration, *Projections, 1984,* table 7b.

7. U.S. Department of Health and Human Services, Health Care Financing Administration, Bureau of Data Management and Strategy, *Annual Medicare Program Statistics, 1982* (GPO, 1984), p. 226.

8. Unpublished data from the 1982 National Master Facility Survey provided by the National Center for Health Statistics.

stantially with time. It also seems reasonable to expect that the health status of marginal survivors will be worse than that of the rest of the population.

The adverse effect of increased survivorship on the health status of the elderly population may of course be offset by improvements in the ability to treat chronic illness. We present a formal framework for thinking about the effects of reduced mortality on the health status of the population, and then examine the relative importance of improvements in managing chronic illness.

A Formal Model of Mortality Reductions

The interactions between reductions in mortality and the health status of the surviving population are complex. On the one hand, measures that lower mortality may also improve health status. Reductions in smoking, improvements in diet, and improved control of hypertension probably improve health at all ages. On the other hand, reductions in mortality may also raise morbidity by changing the composition of the surviving population. An obvious example is those whose lives have been extended through the widespread availability of kidney machines. Mortality reductions also raise morbidity by increasing the average age of the population.

The relative importance of these two effects has been the subject of some dispute. Victor Fuchs and James Fries take the optimistic view that health progress is likely to be associated with reduced morbidity.[9] Other authors, notably Kenneth Manton, take the opposite view and suggest that the burden of caring for the elderly population will rise as mortality falls.[10] The question of which view is correct depends on the source of mortality reductions. Kidney machines and exercise programs will differ in their effects on the health status of the aged population. Ideally, an analysis of

9. Victor R. Fuchs, " 'Though Much Is Taken': Reflections on Aging, Health, and Medical Care," *Milbank Memorial Fund Quarterly,* vol. 62 (Spring 1984), pp. 143–66; James F. Fries, "Aging, Natural Death, and the Compression of Morbidity," *New England Journal of Medicine,* vol. 303 (July 17, 1980), pp. 130–35; and James F. Fries and Lawrence M. Crapo, *Vitality and Aging: Implications of the Rectangular Curve* (San Francisco: W. H. Freeman, 1981).

10. Manton, "Changing Concepts of Morbidity and Mortality." A similar viewpoint is found in Lois M. Verbrugge, "Longer Life but Worsening Health? Trends in Health and Mortality of Middle-Aged and Older Persons," *Milbank Memorial Fund Quarterly,* vol. 62 (Summer 1984), pp. 475–519.

recent trends would focus on the differential sources of reduced mortality. However, it is notoriously difficult to isolate the reasons for declining mortality among the elderly. We therefore present a general framework that formalizes the effects of lower mortality rates on health status.[11]

Population Heterogeneity and Mortality

We present a model developed by Vaupel, Manton, and Stallard that permits decomposition of observed changes in death rates into two components: one due to health progress, which affects all individuals, and one due to the changing average frailty of the population.[12] The model is stylized in assuming that each individual is endowed with a "frailty" at birth that remains constant throughout life. While this assumption may be unrealistic, it successfully captures the notion that health progress that reduces the risk of death for all individuals will reduce the strength of the surviving population, especially at very advanced ages. This composition effect may partly mask mortality improvements. It may also lead increasing morbidity to be associated with mortality reductions.

We assume the force of mortality for individual i, in cohort j, at age t, $\mu_i^j(t)$, is the product of two terms:

$$\mu_i^j(t) = z_i \mu^j(t), \tag{1}$$

where $\mu^j(t)$ equals the cohort-specific force of mortality for persons of age t, and z_i is person i's frailty at birth.[13] The age-specific death rate, $q_i^j(t)$, is defined as the probability that person i in cohort j dies between ages t and $t + 1$, conditional on reaching age t. The force of mortality and $q_i^j(t)$ are linked by the approximation

$$\mu_i^j(t) = -\log[1 - q_i^j(t)]. \tag{2}$$

11. The issues here closely parallel those in the labor economics literature on heterogeneity versus state dependence. An overview of these questions may be found in James J. Heckman and George J. Borjas, "Does Unemployment Cause Future Unemployment? Definitions, Questions and Answers from a Continuous Time Model of Heterogeneity and State Dependence," *Economica,* vol. 47 (August 1980), pp. 247–83.

12. James W. Vaupel, Kenneth G. Manton, and Eric Stallard, "The Impact of Heterogeneity in Individual Frailty on the Dynamics of Mortality," *Demography,* vol. 16 (August 1979), pp. 439–54.

13. We normalize the average frailty at birth to equal 1, as in ibid.

The probability that a type z_i individual will survive to age m, $S_i^j(m)$, is

$$S_i^j(m) = \exp\left[-\int_0^m \mu_i^j(t)dt\right] = \exp\left[-z_i \int_0^m \mu^j(t)dt\right]. \tag{3}$$

The population force of mortality at each age, $\bar{\mu}^j(t)$, is just a weighted average of individuals' $\mu_i^j(t)$'s. It depends upon both the distribution of frailties among those who are alive and the cohort-specific force of mortality $\mu^j(t)$. From equation 1,

$$\bar{\mu}^j(t) = \bar{z}^j(t) \cdot \mu^j(t), \tag{4}$$

where $\bar{z}^j(t)$ equals the mean frailty of survivors in cohort j at age t. The rate of morbidity, $\bar{\nu}^j(t)$, can also be modeled as a function of average frailty and a cohort-specific morbidity function, $\nu^j(t)$:

$$\bar{\nu}^j(t) = \phi[\underset{\sim}{z}^j(t)] \cdot \nu^j(t). \tag{5}$$

The ϕ function translates mortality-relevant frailties into morbidity-relevant ones. The cohort-specific morbidity function is designed to capture various factors that affect morbidity, such as medical progress.

To make this model operational, we must make some assumption about the distribution of frailties at different ages. We define $f^j(z,t)$ as the probability density function for frailties of individuals in cohort j at age t. Vaupel, Manton, and Stallard assume that frailties at birth follow a gamma distribution.[14] The gamma is sufficiently flexible to allow for a wide variety of distribution patterns. It also has the appealing property that if frailties at birth are gamma distributed, then so are frailties of the survivors at all subsequent ages. We postulate that

$$f^j(z,0) = \lambda_j^k z^{k-1} e^{-\lambda_j z}/\Gamma(k), \tag{6}$$

which is the gamma density with parameters λ_j and k. Its mean is k/λ_j, which equals 1, the average frailty of individuals at birth. This implies $k = \lambda_j$. The variance equals k/λ_j^2. Vaupel, Manton, and Stallard show that the density of frailties for age t survivors is

$$f^j(z,t) = [\lambda_j(t)]^k z^{k-1} e^{-\lambda_j(t)z}/\Gamma(k). \tag{7}$$

14. Ibid., p. 442.

The parameters of this gamma distribution are $\lambda_j(t) = \lambda_j - \log \bar{S}^j(t)$ and k. $\bar{S}^j(t)$ is the fraction of cohort j surviving to age t. Using equation 7, the average frailty of age-t survivors is therefore

$$\bar{z}^j(t) = k/[\lambda_j - \log \bar{S}^j(t)] = k/[k - \log \bar{S}^j(t)], \tag{8}$$

and the variance in frailties at age t is

$$\sigma_j^2(t) = k/[\lambda_j - \log \bar{S}^j(t)]^2. \tag{9}$$

Average frailty declines as a cohort ages, since death is more likely to remove frailer members of the population at earlier ages. The variance of frailties is also a declining function of age. This is intuitively reasonable, since at very advanced ages only the strongest members of the original population, those with the lowest z's, will remain alive.

The effect of selection-induced changes in average survivor frailty is largest at extreme ages. This can be illustrated by considering a reduction in mortality that lowers the cohort-specific force of mortality by a constant fraction (δ) at all ages:

$$\tilde{\mu}(t) = \delta \cdot \mu(t) \qquad \delta < 1. \tag{10}$$

This mortality improvement will affect the mean frailty of the very old by more than that for other groups, but since the average frailty of people who survive to very old age has been raised, the age-specific death rates for the very elderly will show least improvement as a result of the general mortality gain. This is because the change in mortality at each age has two components:

$$\frac{d\bar{\mu}(t)}{d\delta} = \frac{d[\delta\mu(t)\bar{z}(t)]}{d\delta} = \bar{z}(t)\mu(t) + \mu(t)\,\frac{d\bar{z}\,(t)}{d\delta}\,, \tag{11}$$

where $\delta = 1$ initially. The first term yields a reduction in mortality rates as δ falls. It corresponds to the direct reduction in the mortality rate for persons who survive to each age. The second term has the opposite effect; as δ falls, it shows that average frailty at each age will rise, causing some *increase* in the observed age-specific death rate. At all ages average frailty rises as δ falls. This effect is largest at old ages. Since the direct reduction in mortality rates is a constant proportion at all ages, the observed response to an improvement such as equation 10 will be smallest at high

ages. This is what one would expect intuitively. The selection effect of mortality improvements cumulates through time, and so has its greatest impact at high ages.

The model we have sketched imposes several strong restrictions on the nature of mortality reductions. For example, a reduction in the cohort's baseline mortality, $\mu^j(t)$, affects very frail individuals much more than those who were initially healthy. There are undoubtedly some forms of medical progress that affect healthier individuals more than those who are extremely frail, and it would be desirable to allow for such progress in a more general framework. There also may be errors introduced by our assumptions about the functional form of the frailty distribution, although it is difficult to assess their impact. Finally, when we make comparisons across cohorts born at different ages, we assume that the average frailty at birth is the same for each cohort. This seems a natural starting point, although further work might examine the extent to which changing patterns of neonatal care could influence the value of $\bar{z}^j(0)$ across cohorts.

Application to Recent Mortality Gains

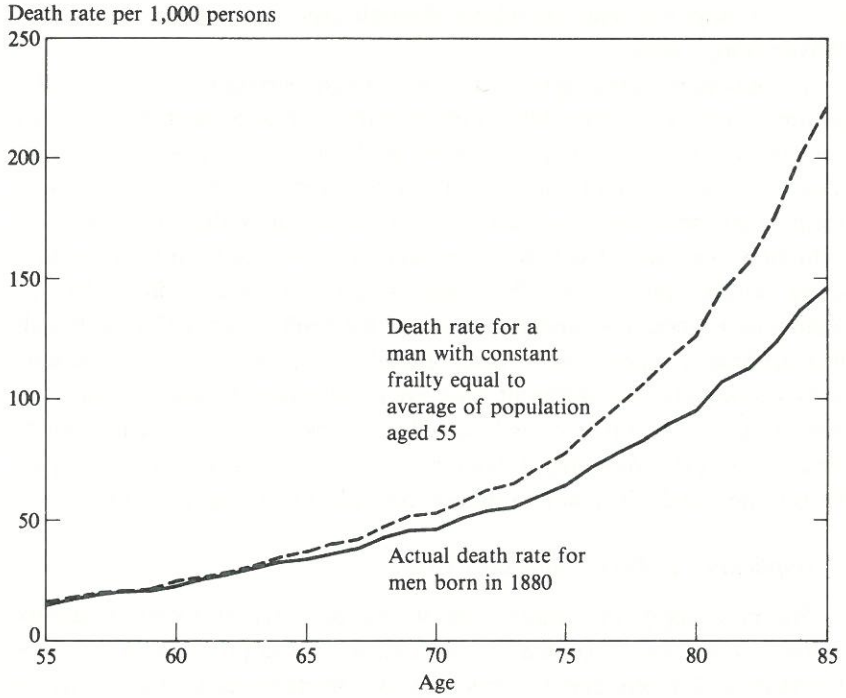
We now apply this framework to the analysis of recent mortality gains.[15] It allows us to measure the extent of true progress in reducing mortality at different ages by removing the effects of reduced mortality on the frailty of the remaining population.

When we normalize the mean cohort frailty to unity at birth, parameterizing the frailty distribution reduces to the problem of choosing k. The variance of frailties at birth is equal to $1/k$. As k rises, the dispersion of frailties declines until in the limiting case of $k = \infty$, there are no differences among cohort members. Manton, Stallard, and Vaupel analyze mortality data on the cohorts of white men and white women born in the United States in five-year intervals between 1850 and 1880. They estimate that k equals 3.93 for the male population and 2.84 for women.[16]

15. Parallel issues arise in analyzing the efficacy of medical techniques. These are discussed in Donald S. Shepard and Richard J. Zeckhauser, "The Choice of Health Policies with Heterogeneous Populations," in Victor R. Fuchs, ed., *Economic Aspects of Health* (University of Chicago Press, 1982); and Shepard and Zeckhauser, "Long-Term Effects of Interventions to Improve Survival in Mixed Populations," *Journal of Chronic Diseases*, vol. 33 (1980), pp. 413–33.

16. Kenneth G. Manton, Eric Stallard, and James W. Vaupel, "Methods for Comparing the Mortality Experience of Heterogeneous Populations," *Demography*, vol. 18 (August 1981), pp. 389–411. The authors also found some cases with much more heterogeneity, corresponding to k of less than 1.

Figure 1. Death Rates for Cohort and Constant-Frailty Person



Our analysis assumes $k = 4.0$ for both men and women; this probably overstates k and understates the dispersion of frailty.[17] These parameters imply that 81.5 percent of men at birth have frailties between one-half and twice the average frailty. To illustrate the difference between the population mortality experience and that facing an individual of constant frailty, we compute for each year the probabilities of dying for a man who was born in 1880 with a frailty of 0.56, the average frailty for those who survived to age 55. We then compare the probabilities that he will die in each year after age 55 with the observed cohort death rates for these ages. The two sets of death rates, denoted $\bar{q}^j(t)$ and $q^j(0.56, t)$, are plotted in figure 1. As time elapses after age 55, the difference between the constant frailty individual's probability of dying and that for the cohort as a whole

17. We tried to make our own estimates of k, the parameter that determines the variance of frailties at birth, using our data on cohort-specific mortality rates. When the maximum likelihood algorithm converged, the estimates always implied $k = \infty$, suggesting the complete absence of heterogeneity and contrasting starkly with the results of Vaupel, Manton, and Stallard using American and Swedish mortality data. The differences in our findings may have resulted from our particular assumptions about the functional forms of the individual hazard functions. Further research should attempt to resolve these differences.

widens. At age 60, for example, the observed death rate is 23.7 per 1,000 persons, while that for the constant-z individual is 24.5. By age 75, the difference is more dramatic: 64.5 versus 77.8 per 1,000 persons. These trends reflect the declining average frailty of the surviving population; the constant-frailty person is increasingly among the frailest members of the surviving cohort. At age 60, he is frailer than 58.3 percent of the surviving cohort; by age 75, 68.3 percent are less frail.

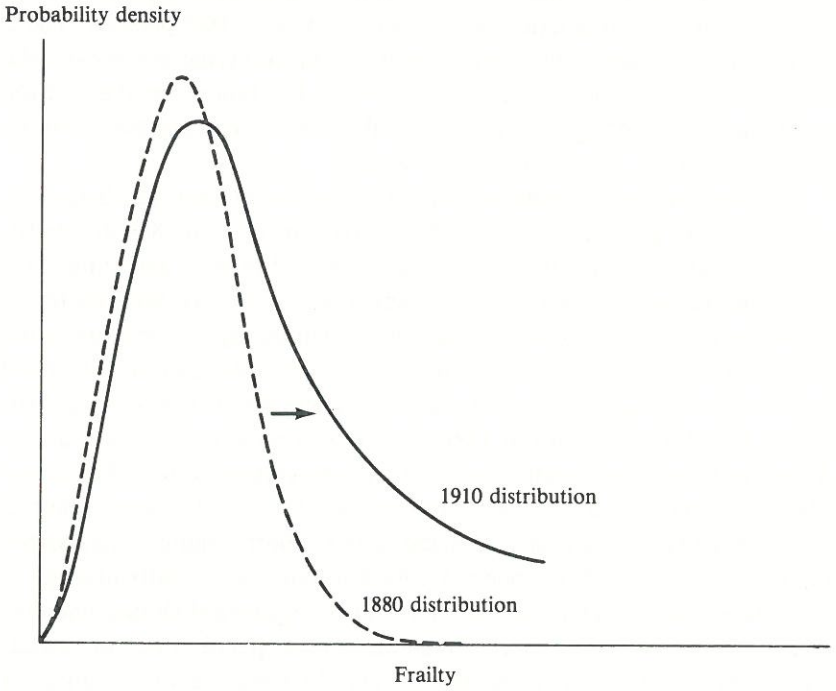
We use the Vaupel-Manton-Stallard technique to analyze changes in mortality rates and life expectancy for individuals born in 1880 and 1910. First, we calculate the observed change in mortality rates and compare it with the change that would have taken place if the average frailty of survivors in the 1880 cohort had applied to similar-aged survivors in the 1910 cohort. The underlying logic of our calculation can be illustrated graphically. Mortality rates are lower at each age for members of the 1910 cohort than for those born in 1880, so the distribution of frailties among those surviving to any particular age in the two cohorts differs. For example, the average frailty among survivors aged 65 in the later cohort is *higher* than that for 65-year-olds in the earlier cohort. Figure 2 is a stylized representation of the difference in frailty distributions at a particular age.

It shows two gamma distributions with the rightward-shifted one corresponding to the 1910 cohort. Given the cohort-specific force of mortality, $\mu^{1880}(t)$ and $\mu^{1910}(t)$, the two frailty distributions imply different observed mortality rates. The distribution of mortality hazards looks the same as the frailty distribution, since $\mu_i^j(t) = z_i \cdot \mu^j(t)$. Our calculations ask what the observed mortality rate at particular ages would have been for the 1910 cohort if it had had the same mortality distribution at those ages as the 1880 cohort did.

We also compute the changes in life expectancy at each age between the two cohorts, again making corrections for movements in average frailty. This enables us to identify the ages at which substantial mortality gains have taken place. Computing life expectancies requires data on the probability of death at ages up to 100 because the exact age at which people die is important. Unfortunately, the maximum age reported in our cohort life tables is 85. For the 1910 cohort, data are available on persons up to 72 years of age. We extended our tables to age 100 by fitting a Gompertz curve, a standard functional form relating age and the force of mortality, to our data on each cohort's death rates at ages 55–85.[18] We then use this

18. A discussion of the Gompertz function and its uses may be found in Shiro Horiuchi and Ansley J. Coale, "A Simple Equation for Estimating the Expectation of Life at Old Ages," *Population Studies*, vol. 36 (July 1982), pp. 317–26.

Figure 2. Change in Frailty Distribution at Age 65 as a Result of Mortality Improvements at Earlier Ages



curve to predict values of death rates at ages greater than 85. The Gompertz curve specifies that the force of mortality rises exponentially over time: $\mu^j(t) = \mu^j(a)e^{\beta(t-a)}$, where a in our estimates equals 55. This specification implies a simple regression model for mortality rates:

$$\log\{-\log[1-q^j(t)]\} = \alpha_j + \beta_j(t-55) + \epsilon_{jt}. \tag{12}$$

The results of our estimates for each cohort are shown in table in A-1.

The probabilities of death that would have been observed for the 1910 cohort if average frailty at each age had equaled that for the 1880 cohort are

$$\tilde{q}^{1910}(\bar{z}^{1880}(t), t) = 1 - \exp\{-k\log[1-\tilde{q}^{1910}(t)]\bar{z}^{1880}(t)/\bar{z}^{1910}(t)\}. \tag{13}$$

This expression depends upon $\bar{z}^{1910}(t)$, the average frailty at age t for the survivors in the 1910 cohort, and $\bar{z}^{1880}(t)$, the average frailty for survivors

from the 1880 cohort when they were t years old. These average frailties are functions of survival probabilities at earlier ages; see equation 8 above.

The results of the mortality rate calculations are shown in table 5, which reports the actual and the frailty-adjusted death rates at five-year intervals for both men and women. The table shows that for men the death rates at age 65 declined from 34.1 to 29.1 between the 1880 and 1910 cohort. At age 80 the decline was more pronounced, from 95.4 to 57.8. The table also shows that the decline would have been even larger at both ages if the average frailty of the respective populations had remained constant at its 1880 level. The change at age 65 would have been 6.7 persons per 1,000, compared with the actual 5.0, while at age 80 the constant-frailty decline in death rates equals 43.0, rather than 37.6. Figure 3 shows the reductions in death rates, with and without our frailty adjustment, for men of all ages between 55 and 85. The figure vividly demonstrates that the largest reductions in mortality occurred at very advanced ages. While a male survivor's probability of dying falls by nearly one-fourth at age 55, it is reduced by roughly 50 percent at all ages above 80.

The table shows that even more pronounced changes have occurred for women. At age 65 the observed mortality rates declined from 22.9 to 13.5 between 1945 and 1975, the dates when women in the 1880 and 1910 cohorts turned 65. Adjusting for changes in frailty yields a relatively small additional improvement, converting the 1975 mortality rate to 12.5. At older ages, the frailty adjustment matters somewhat more. At age 85, for example, observed death rates fell from 119.8 to 36.3, while keeping the 1880 cohort's frailty level would have lowered the death rate to 30.2.

Studying changes in death rates is one way to identify the ages at which the most progress has been made against mortality. However, the claim that substantial gains have occurred at extreme ages may be of little significance if the fraction of the population that lives to these ages is trivial. An alternative measure of where gains have been made is the change in life expectancy at a given age between two cohorts.

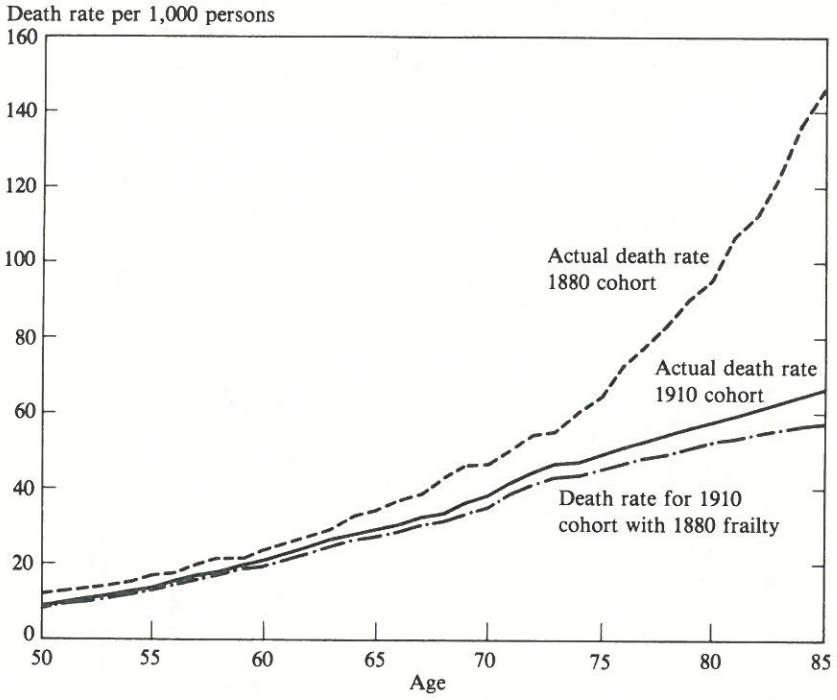
Table 6 shows the actual and adjusted life expectancies at ages 55 through 85 for individuals born in 1880, 1890, 1900, and 1910. The changes in life expectancy for both men and women occur disproportionately between the last two cohorts. For example, a man born in 1880 who lived to age 75 had a life expectancy of 8.3 years. If he had been born in 1900 and reached age 75, his life expectancy would have been 9.3 years. However, an individual born just ten years later, in 1910, would have a life expectancy of 12.5 years at age 75. The absolute gain in life expectancy is

Table 5. Actual and Frailty-Adjusted Death Rates per 1,000 Persons, 1880–1910 Birth Cohorts, by Age and Sex

			Death rate improvements							
	Death rate, 1880 cohort		*1880–90 cohort*		*1890–1900 cohort*		*1900–1910 cohort*		*Death rate 1910 cohort*	
Age	*Men*	*Women*	*Men*	*Women*	*Men*	*Women*	*Men*	*Women*	*Men*	*Women*
Actual death rate										
55	16.5	11.7	0.9	2.3	2.1	2.5	−0.1	0.4	13.6	6.5
60	23.7	15.7	1.6	3.5	1.4	2.3	−0.3	0.4	21.0	9.5
65	34.1	22.9	2.5	5.4	0.2	2.3	2.3	1.7	29.1	13.5
70	46.6	30.7	1.3	4.5	−1.1	2.5	8.4	5.1	38.0	18.6
75	64.5	45.0	−1.1	4.8	1.2	4.4	15.4[a]	9.3[a]	49.0[a]	26.5[a]
80	95.4	72.1	0.4	9.4	6.1	9.4	31.1[a]	21.9[a]	57.8[a]	31.4[a]
85	146.1	119.8	18.0	24.5	20.0[a]	29.4[a]	41.5[a]	29.6[a]	66.6[a]	36.3[a]
Death rate assuming 1880 frailty level										
55	16.5	11.7	1.1	2.5	2.5	2.7	0.2	0.5	12.8	6.1
60	23.7	15.7	1.9	3.7	2.0	2.6	0.1	0.6	19.8	8.8
65	34.1	22.9	3.0	5.8	1.1	2.8	2.7	1.9	27.3	12.5
70	46.6	30.7	2.0	5.2	0.2	3.3	8.9	5.2	35.5	17.0
75	64.5	45.0	−0.2	6.1	2.9	5.6	16.4[a]	9.4[a]	45.4[a]	23.9[a]
80	95.4	72.1	1.7	11.7	8.5	11.4	32.8[a]	21.5[a]	52.4[a]	27.6[a]
85	146.1	119.8	20.2	29.0	24.0[a]	31.8[a]	44.3[a]	28.8[a]	57.6[a]	30.2[a]

Source: Authors' calculations based on cohort life table data for white men and women provided by Metropolitan Life Insurance Company. See text for details of calculations.
a. Based on extrapolated death rates.

Figure 3. Actual Death Rates for White Men Born in 1880 and 1910, and 1910 Rates Assuming 1880 Frailty Distribution



a smoothly declining function of age, while the percentage gain rises with age.

The difference between actual and frailty-adjusted life expectancies can be seen by comparing the top and bottom sections of the table. If the average frailty of the 1910 cohort at each age had equaled the same-age average frailty of the 1880 cohort, life expectancy would have been roughly one year greater. At age 55, for example, it would have raised male life expectancy from 22.4 to 23.5 years. At age 80 changes from 10.66 to 11.60 years for men and 13.92 to 14.87 years for women would be observed. Put another way, the marginal survivors at each age have lower life expectancies than those who would have lived to that age in the previous cohort. Since the difference between the actual and frailty-corrected estimates is approximately the same at all ages, the proportionate change induced by the frailty correction is largest at old ages.

These data cast doubt on the view of Fries and Crapo and others that

Table 6. Actual and Frailty-Adjusted Life Expectancies, 1880–1910 Birth Cohorts, by Age and Sex
Years

Age	*Life expectancy, 1880 cohort*		*Life expectancy improvements*						*Life expectancy, 1910 cohort*	
			1880–90 cohort		*1890–1900 cohort*		*1900–1910 cohort*			
	Men	*Women*	*Men*	*Women*	*Men*	*Women*	*Men*	*Women*	*Men*	*Women*
Actual life expectancy										
55	19.06	22.55	0.46	1.94	0.59	2.14	2.29	3.48	22.41	30.12
60	15.87	19.01	0.36	1.71	0.51	1.97	2.55	3.60	19.29	26.28
65	13.03	15.75	0.25	1.43	0.50	1.85	2.87	3.72	16.65	22.75
70	10.56	12.76	0.25	1.27	0.53	1.74	2.98	3.68	14.32	19.45
75	8.31	10.04	0.33	1.16	0.70	1.77	3.10[a]	3.68[a]	12.45[a]	16.65[a]
80	6.52	7.78	0.45	1.15	0.84	1.77	2.84[a]	3.21[a]	10.66[a]	13.92[a]
85	5.44	6.37	0.57	1.01	0.61[a]	1.43[a]	2.21[a]	2.30[a]	8.83[a]	11.11[a]
Life expectancy assuming 1880 frailty level										
55	19.06	22.55	0.61	2.30	0.93	2.66	2.85	3.89	23.45	31.40
60	15.87	19.01	0.50	2.06	0.82	2.46	3.12	4.00	20.31	27.54
65	13.03	15.75	0.38	1.78	0.80	2.34	3.44	4.10	17.66	23.96
70	10.56	12.76	0.37	1.61	0.81	2.21	3.57	4.03	15.31	20.61
75	8.31	10.04	0.45	1.49	0.96	2.21	3.71[a]	3.98[a]	13.42[a]	17.72[a]
80	6.52	7.78	0.57	1.46	1.10	2.18	3.42[a]	3.45[a]	11.60[a]	14.87[a]
85	5.44	6.37	0.70	1.30	0.84[a]	1.75[a]	2.68[a]	2.43[a]	9.66[a]	11.86[a]

Source: Authors' calculations based on cohort life table data for white men and women provided by Metropolitan Life Insurance Company. See text for details of calculations.
a. Based on death rate extrapolations.

longevity is currently pushing an upper limit.[19] Taking account of heterogeneity, it appears that life expectancy is increasing more at old than young ages. It is increasing more for women than for men, even though women already have longer life expectancies. We find little evidence to confirm the view that mortality is increasingly bunched at some specific age. When we remove the selection effect of reduced mortality at earlier ages on mortality at older ages, clear evidence against rectangularization emerges.

Our results suggest that if the framework sketched here is roughly accurate, the changing frailty mix between the 1880 and 1910 cohorts reduced the gains in both lower mortality rates and higher life expectancies that would have taken place if the survivors were of the same frailty as those in the 1880 cohort. These substantial changes in average frailty should also have had other effects. If a larger fraction of the population survives until age 65, then the variance of frailties at that age will increase. This should imply an increase in the variance of longevities after age 65. For men reaching age 65 in 1925, the variance of the remaining years of life was 40.7; for those reaching 65 in 1965, it was 66.5. The comparable figures for women in these two years were 45.2 and 87.0, respectively. This accords with the predictions of the heterogeneity model.

Evidence on Changing Morbidity and Health Needs

While it is difficult to assess the extent of heterogeneity in the population, the preceding analysis suggests that changes in mortality rates could result in important changes in the composition of the surviving population. If this were the only force acting on the health status of the aged, one would expect to see substantial deterioration, especially among those at high ages. This supports the pessimistic view of health progress. Another explanation could also be proposed to account for reductions in health status. Episodes of morbidity may weaken individuals. Recent progress may have allowed people to become more ill, and less resilient, without causing their death. If illness-induced reductions in resilience are persistent, then health progress may raise the average frailty of the surviving population.

Victor Fuchs's optimistic analysis of changes in health status does not

19. Fries and Crapo, *Vitality and Aging*.

consider the changing composition of the aged population.[20] Rather, he focuses on the effects of broadly defined health progress on the health status of a given aged person. He proposes an intriguing model for thinking about the linkages between aging and health. He argues that for medical care costs, disability, and institutionalization, age is better measured *backward* from death rather than forward from birth. Medical care costs, for example, have been shown to be highly concentrated in the year or two immediately preceding death. Since death rates at all ages have been declining, raising the average number of years till death, Fuchs's view would lead to the expectation that the health status of the elderly population should actually be improving. The fraction of the population at each age who are within one or two years of death has declined, as evidenced by changing death rates. Fuchs's view predicts that there should be greater improvements in health status at old ages than at younger ages because of the greater absolute reduction in death rates among the elderly. Any other view emphasizing the importance of medical developments or changes in life-style in reducing morbidity would also lead one to expect trend improvements in the health of the elderly.

These two views thus offer dramatically opposite predictions about trends in the health status of the elderly population and about the relative health status of persons at different ages. One cannot doubt the existence of both positive developments that reduce morbidity and changes in the composition of the population that tend to increase illness and disability. The central question is which effects predominate. To investigate this issue, we examine a number of indices of the health status of the elderly. When possible, we look at age-specific measures to avoid biases due to the aging of the population.

Table 7 reports the fraction of the aged population residing in nursing homes as reported in the five most recent surveys. While the overall level of nursing home usage is probably driven more by supply than demand factors, differences in institutionalization trends across different groups can provide information on their relative health status. For both men and women under 85, the data show a steady upward trend in the rate of institutionalization from 1963 until 1977 and then a small decline in 1982. There is a substantial increase in institutionalization rates at all ages between 1963 and 1969. The pattern among very old men and women, those over 85, is quite different, however. The rate of institutionalization

20. Fuchs, " 'Though Much Is Taken.' "

Table 7. Percentage of Population in Nursing Homes, by Age and Sex, Selected Years, 1963–82

	Age and sex					
	65–74		*75–84*		*85 and over*	
Year	*Men*	*Women*	*Men*	*Women*	*Men*	*Women*
1963	0.68	0.88	2.91	4.75	10.56	17.51
1969	0.99	1.29	3.60	6.23	13.08	24.76
1973	1.13	1.31	4.08	7.11	18.04	29.06
1977	1.27	1.59	4.74	8.06	14.00	25.15
1982	1.23	1.55	4.17	6.89	13.93	22.69

Sources: For 1963–77, NCHS, *Characteristics of Nursing Home Residents: Health Status and Care Received,* Vital and Health Statistics, series 13, no. 115 (DHHS, 1981), p. 4. For 1982, calculations are based on data from the National Master Facility Survey provided by the National Center for Health Statistics.

rises steadily until 1973 and then declines sharply through 1982. This pattern is rather surprising. Nonhealth determinants of the rate of institutionalization, such as the availability of public assistance or increased viability of remaining at home, would be expected to exhibit similar trends for all age groups. Thus these data suggest that the health status of the very old is improving relative to that of their younger counterparts.

An interesting feature of the data is that at all ages women have considerably higher rates of institutionalization than men, despite their longer life expectancy. This probably reflects their much greater likelihood of being widowed. The data also reject popular stereotypes about the pervasiveness of institutionalization. Even for the extremely aged, less than one-fifth of the population is in a nursing home. Only about 5 percent of the elderly population is institutionalized at any given time.[21]

An alternative indicator of health status is expenditures on medical care among persons of different ages. In our heterogeneous population model, the survivorship gains of the last two decades should have raised the average frailty among the extremely aged by more than that of the "young old" (those aged 55–70). This should correspond to increases over time in the relative health care needs of the extremely aged, provided more frail individuals experience both higher mortality and higher morbidity, as we have suggested. Data on medicare expenditure trends are presented in table 8. Because the overall level of medicare spending is driven by economic and noneconomic forces beyond the scope of this paper, we

21. However, one-fifth of all elderly persons will be in a nursing home at some point. See ibid., p. 161.

Table 8. Medicare Expenditure Ratios, by Age and Sex, Selected Years, 1966–82[a]

	1966		*1971*		*1977*		*1982*	
Age	*Men*	*Women*	*Men*	*Women*	*Men*	*Women*	*Men*	*Women*
65–66	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00
67–68	1.11	1.20	1.36	1.34	1.05	1.03	1.10	1.10
69–70	1.15	1.18	1.43	1.43	1.13	1.10	1.19	1.21
71–72	1.28	1.30	1.52	1.57	1.22	1.18	1.30	1.29
73–74	1.40	1.40	1.59	1.73	1.34	1.32	1.45	1.41
75–79	1.57	1.58	1.76	1.91	1.48	1.52	1.62	1.64
80–84	1.74	1.80	2.01	2.21	1.64	1.77	1.85	1.93
85 and over	1.97	2.02	2.09	2.24	1.87	1.97	1.96	2.10

Sources: Authors' calculations based on U.S. Department of Health and Human Services, Health Care Financing Administration, *Medicare Program Statistics*, annual issues, and *Health Care Financing: Program Statistics*.

a. Ratio of average medicare expenditure per enrollee to expenditures per enrollee aged 65–66. Data are for white men and women only.

focus only on relative levels of expenditure on persons of different ages. The table reports the average medicare reimbursement per enrollee in each age group, scaled by the average reimbursement per enrollee aged 65–66. The data show very little variation over time in the age pattern of medicare expenditures. In 1966 the ratio of expenditures on enrollees over 85 to those between 65 and 66 was 1.97 for men and 2.02 for women. In 1982 the comparable values were 1.96 and 2.10, respectively. Similar patterns emerge in other years and at intervening ages.[22] Movements in the fraction of enrollees who receive some medical services also show a similar pattern. In 1966, 30.2 percent of medicare enrollees aged 65–66 received service, compared with 48.2 percent of those over 85. In 1982 the rates were 57.5 percent and 73.3 percent, respectively. The growth in utilization was larger for those at younger ages than for the very old.[23]

These data on both utilization and care levels suggest that the effects of health progress and the changing composition of the population have largely offset each other. They do not support the view that health progress inevitably carries with it huge expenditure burdens for marginal survivors, as the heterogeneity model would suggest. While close to half of the 1982 population of persons over 85 were marginal survivors, compared with a

22. One exception is 1971, when the unusual results appear to be due to unusually low expenditures on persons aged 65–66. Relative levels of expenditures between other ages are not out of line with the data from other years.

23. Data are drawn from the Health Care Financing Administration, *Annual Medicare Program Statistics, 1982*, and from U.S. Department of Health, Education, and Welfare, *Health Care Financing: Program Statistics, 1966.*

Table 9. Restricted Activity and Bed Disability among Aged, by Age and Sex, Selected Years, 1961–80

Health indicator and year	*Average days per person*			
	Age and sex			
	65–74		*75 and over*	
	Men	*Women*	*Men*	*Women*
Restricted activity days				
1961	31.9	34.8	36.1	46.2
1963	31.3	36.3	41.4	49.6
1965	30.9	30.7	36.0	41.9
1968	31.2	30.3	35.0	47.6
1971	26.6	30.6	38.8	44.9
1975	31.1	36.2	40.7	49.4
1980	34.2	39.2	36.0	46.6
Bed disability days				
1961	11.4	12.5	14.6	23.6
1963	10.8	11.3	17.6	20.0
1965	11.5	11.1	14.5	16.0
1968	12.0	11.6	16.3	20.9
1971	9.0	10.9	17.9	18.9
1975	9.8	10.6	17.0	17.7
1980	10.9	12.9	13.4	19.1

Source: NCHS, *Disability Days*, Vital and Health Statistics, series 10 (DHHS, various issues).

lower fraction in earlier years, relative medicare costs remained roughly constant.

Data on medicare expenditures and institutionalization have the virtue of being objective, but also have the problem of being influenced by a variety of factors other than the health status of the elderly. Another source of health status information is the extent of activity limitation and disability in the elderly population. Data on these health measures for the civilian noninstitutional population are collected in the Health Interview Survey, and tabulations are periodically published by the National Center for Health Statistics.[24] Table 9 presents information on the number of restricted activity days and bed disability days per year for subgroups of the elderly population for various years. Because the numbers refer only to the noninstitutionalized population, they may be heavily influenced by

24. Martin Neil Baily's chapter in this volume reviews a number of limitations of these data.

fluctuations in the institutionalization rate, especially since persons in institutions are likely to have substantial amounts of restricted activity.[25]

In general, the data on restricted activity display no clear trend. The reduced morbidity and frailty-composition effects of medical progress again seem to offset each other. The incidence of restricted activity days for both men and women aged 75 and over is nearly the same in 1980 as in 1961.[26] For those aged 65–74, there is slight evidence of an increase in restricted activity days in the 1980 data. These data confirm the inference drawn from the data on institutionalization: the health of the elderly has not worsened and may have improved in recent years. In particular, they offer no support for substantial reductions in health at extreme ages where the incidence of marginal survivors is greatest.

Similar conclusions are suggested by information on the incidence of bed disability days also shown in table 9. They also appear to be relatively constant between 1961 and 1980. For all age groups except women aged 65–74, the incidence of bed disability days was lower in 1980 than in 1961. In some cases, such as women aged 75 and over, the improvement suggests an average reduction of nearly one week per year of disability. For men over 75 a substantial improvement in health status is observed between 1975 and 1980, paralleling the observations made above. The principal gains for extremely old women occurred during the 1960s. An interesting aspect of the data is that the disability rate is higher for women than for men, particularly in the over-75 category. This tends to contradict Fuchs's view that time until death is a good indicator of health status, since women have longer life expectancies than men. It is consistent with the heterogeneity view, since a much larger fraction of women than men reach the age of 75.

These data suggest that improvements in morbidity associated with increases in life expectancy largely offset changes in the composition of the surviving population due to reduced mortality. As a result, improvements in life expectancy appear to have little effect on indicators of health status at advanced ages. While each of the indicators that we have

25. This may explain the presence of some outlying observations, such as the one for restricted activity days for women aged 75 and over in 1965.

26. One contrary finding on the incidence of disability is the fraction of new male social security recipients aged 62–64 who report health factors as the main reason for leaving their last job. This fraction has fallen from 54 percent in 1968 to 29 percent in 1982; see Sally R. Sherman, "Reported Reasons Retired Workers Left Their Last Job: Findings from the New Beneficiary Survey," *Social Security Bulletin,* vol. 48 (March 1985), p. 25. These data are unfortunately difficult to interpret because of the dramatic changes in retirement probabilities during this period.

examined—institutionalization, medical expenditures, restricted activity days, and bed disability days—are flawed as measures of health status, their problems are somewhat independent. As a result, this consistency provides fairly strong support for our basic conclusion that the "heterogeneity" and "reduced morbidity" effects of medical progress roughly cancel each other.

Microeconometric Evidence

The data presented thus far suggest that the health of the elderly population has not changed much through time despite the changing composition of the elderly population and progress in reducing morbidity. This suggests that these two effects have been roughly offsetting. We now present some very crude microeconometric evidence bearing on this question. We test Fuchs's hypothesis that "relevant age" should be measured backward from death rather than forward from birth by making use of longitudinal data from the Retirement History Survey.[27] A finding that years until death rather than age was the best predictor of health status would provide strong support for the optimistic view that increases in life expectancy will be associated with improvements in the health status of the aged. Conversely, a finding that controlling for time until death had little effect on the impact of age on health would support the more pessimistic view that increased morbidity will come with reduced mortality.

The Retirement History Survey includes information on individuals' ages as well as dates of death for the quarter of the original sample that died between 1969 and 1979. We compare the power of the age and time-till-death variables in explaining health-related activity limitations. Since this is only one indicator of health status, we also compare the performance of the two age variables as predictors of retirement decisions.

The Retirement History Survey suffers from several drawbacks as a source for an investigation of this type. First, it provides data on only the youngest part of the aged population.[28] Even at the end of the sample, the

27. The RHS data are described in Lola M. Irelan and others, *Almost 65: Baseline Data from the Retirement History Study,* U.S. Department of Health, Education, and Welfare, Social Security Administration, Office of Research and Statistics, Research Report 49 (GPO, 1976).

28. We initially tried to estimate equations for hospital costs. Unfortunately the limited age variation in our sample rendered the age effect on these costs statistically insignificant, precluding our test of whether any significant relationship is spuriously due to the time-till-death effect.

oldest person in the sample was only 73. Second, the information on activity limitation is self-reported and may therefore be subject to a number of biases.

We estimate activity-limitation equations of the form:

$$(14) \quad LIMIT_i = \alpha_0 + \alpha_1 MARRY_i + \alpha_2 EDUC_i + \alpha_3 SMSA_i + \alpha_4 RACE_i + \alpha_5 AGE_i + \alpha_6 YTD_i + \alpha_7 SURVIVOR_i + \epsilon_i,$$

where

LIMIT = a 0-1 variable that equals 1 if the respondent reported that health limitations restricted the kind or amount of work or housework that he could perform;
MARRY = a dummy variable equal to 1 for persons who were married;
EDUC = years of schooling;
SMSA = a dummy variable equal to 1 for persons residing within an SMSA;
RACE = 1 for nonwhites;
AGE = chronological age;
YTD = years to death.

Our goal is to compare the effect of increases in age with the effect of time until death on health-induced activity limitations. We control for several exogenous individual characteristics in an attempt to improve the precision of our estimates. The survey records both month and year of death, so we are able to measure this variable quite accurately. We also include *SURVIVOR*, a dummy variable equal to 1 if the respondent survived until the end of the sample period. We assigned these individuals the maximum value for *YTD* as well. No economic variables, such as wealth, were included in the equation because of the possibility that they were affected by individual choices based on knowledge of health status.

The results of several specifications of the activity-limitation equation are shown in table 10.[29] Increases in age and reductions in the number of years to death both raise the probability that a respondent would report health limitations. A one-year reduction in years to death has roughly the

29. All estimates were obtained using the Brookings Retirement History Survey extract, provided by Christine de Fontenay. We omitted one observation which claimed that the respondent had died in 1958.

Table 10. Estimates of Equations Explaining Health-Related Activity Limitations[a]

Variable	*Equation 1*	*Equation 2*	*Equation 3*
Constant	−0.588	0.926	−0.051
	(−0.186)	(0.031)	(−0.186)
MARRIED	−0.097	−0.090	−0.089
	(−0.016)	(−0.015)	(−0.015)
EDUCATION	−0.022	−0.021	−0.021
	(−0.001)	(−0.001)	(−0.001)
SMSA	−0.052	−0.047	−0.049
	(−0.011)	(−0.010)	(−0.010)
RACE	−0.004	−0.008	−0.003
	(−0.019)	(−0.018)	(−0.018)
AGE	0.021	. . .	0.016
	(0.003)	. . .	(0.003)
YEARS TO DEATH	. . .	−0.023	−0.023
	. . .	(−0.004)	(−0.004)
SURVIVOR	. . .	−0.088	−0.085
	. . .	(−0.022)	(−0.022)
SSR	1,729.3	1,676.4	1,670.5
R^2	0.046	0.075	0.079

a. All equations are estimated with 8,011 observations from the Brookings 1969 Longitudinal Retirement History Survey Extract File. Numbers in parentheses are standard errors.

same effect on the probability of reporting such limitations as one additional calendar year of age. The most important finding, however, is that the effects of age and time-till-death are largely independent. Although equations including the time-till-death variable fit significantly better than those with only age in the specification, controlling for time-till-death reduces the coefficient on the age variable by less than 25 percent. This suggests that the relationship between health limitations and age is not a spurious one, due solely to an underlying relationship between health care needs and time until death. It does suggest, however, that increases in life expectancy will reduce the age-specific activity limitation rates in the elderly population.

We extended our analysis of health status to consider also the effect of the two age measures on retirement decisions. Because the 1969 survey includes a relatively small fraction of retired persons, we report results for both it and the subsequent 1973 survey. The equation we estimate corresponds exactly to that for activity limitations above, except that the dependent variable now equals 1 if the person has retired and 0 otherwise. The results of estimating the retirement equation are shown in table 11. As one would expect, both *AGE* and *YTD* have significant effects on the probabil-

Table 11. Estimates of Equations Explaining Retirement Decisions[a]

Variable	1969 sample			1973 sample		
	Equation 1	*Equation 2*	*Equation 3*	*Equation 1*	*Equation 2*	*Equation 3*
Constant	−1.326	0.480	−1.030	−3.658	0.726	−3.529
	(−0.131)	(0.022)	(−0.133)	(−0.212)	(0.036)	(−0.088)
MARRIED	−0.098	−0.096	−0.095	−0.091	−0.093	−0.088
	(−0.011)	(−0.011)	(−0.011)	(−0.016)	(−0.017)	(−0.017)
EDUC	−0.009	−0.009	−0.008	−0.016	−0.015	−0.014
	(−0.001)	(−0.001)	(−0.001)	(−0.001)	(−0.002)	(−0.001)
SMSA	0.004	0.008	0.005	0.032	0.040	0.032
	(0.008)	(0.007)	(0.007)	(0.011)	(0.012)	(0.011)
RACE	0.016	0.010	0.016	−0.063	−0.078	−0.063
	(0.013)	(0.013)	(0.013)	(−0.020)	(−0.020)	(−0.020)
AGE	0.026	. . .	0.024	0.067	. . .	0.065
	(0.002)	. . .	(0.002)	(0.003)	. . .	(0.003)
YEARS TO DEATH	. . .	−0.002	−0.002	. . .	3.4×10^{-4}	3.0×10^{-4}
	. . .	(−0.002)	(−0.002)	. . .	(7.3×10^{-4})	(7.1×10^{-4})
SURVIVOR	. . .	0.008	0.013	. . .	−0.116	−0.097
	. . .	(0.016)	(0.015)	. . .	(−0.031)	(−0.030)
SSR	870.6	870.2	856.0	1,665.3	1,749.2	1,657.6
R^2	0.042	0.042	0.058	0.072	0.025	0.077
N	8,011	8,011	8,011	7,332	7,332	7,332

Source: See table 10.

a. Numbers in parentheses are standard errors. See text for further description.

ity of retirement. The 1973 estimates imply that an extra year of age increases the probability of retirement by 6.5 percent. Being a year closer to death has a trivial positive effect on this probability. This effect is also statistically insignificant. The important result is again that controlling for time until death does not change the effect of *AGE* on retirement for either the 1969 or 1973 samples, again suggesting that there is a genuine link between age and retirement. In results that are not reported here, we found that the *YTD* variable in both the retirement and activity-limitation equations had a highly nonlinear effect. Imminent death has a strong positive effect on the probability of retirement. Further research could usefully explore these effects in greater detail.

Conclusions

Our analysis of several types of data suggests that neither an extremely optimistic nor an extremely pessimistic view of the impact of declining old-age mortality is appropriate. Increased survivorship among relatively unhealthy members of the population has in the past been offset by general reductions in morbidity, leaving the age-specific health status of the population largely unchanged. While projections are difficult, there is no obvious reason to expect this pattern to change.

This suggests that future medicare costs or the costs of institutionalization can be estimated using current age-specific information. Projections of this type are somewhat ominous. On the basis of 1982 age-specific rates of institutionalization and intermediate mortality assumptions, one can forecast a 53 percent increase in the population of institutionalized men and a 67 percent increase for women by the year 2000. By 2020 the increase will be nearly 120 percent. Under the optimistic mortality assumptions, the corresponding figures are 64 percent for men and 79 percent for women by 2000, and by 2020 both populations would rise to more than 150 percent of their 1982 level. On the basis of the most recent profile of medicare costs by age, one can forecast a cost increase of 37 percent by the year 2000 under the intermediate mortality assumptions.[30] The increase would be 43 percent under optimistic assumptions. Since the

30. We calculated this value using the Office of the Actuary's Alternative II population projections, weighing the number of persons in each age group by the age-specific medicare costs shown in table 8.

intermediate mortality assumption implies that the population aged 65 and over will increase by 32 percent by the year 2000, these findings suggest that 22 percent of the increase in male institutionalization and 35 percent of the female increase will result from aging of the elderly population. For medicare, the comparable figures are 4 and 7 percent.

These increases in costs seem large relative to the savings attainable through improvements in the delivery of health care or the savings that might be possible through making consumers bear a larger fraction of health care costs. This suggests that we must inevitably plan on increases in the resources devoted to taking care of the dependent elderly, even if substantial improvements in the delivery of care are achieved.

Proposals to redefine the elderly are frequently advanced. The proponents suggest that the age of 65, originally set by Bismarck, is no longer an appropriate demarcation point for defining old age. More important, it is often argued that with increases in life expectancy the normal age of retirement should be increased so as to preserve the ratio of working years to retirement years for the average member of the population. Indeed, this principle was enshrined in the 1982 social security reform package, which calls for future increases in the social security retirement age. Our analysis suggests that these policy prescriptions are inappropriate. The data do not support Fuchs's view that age should be measured backward from death. Reductions in mortality do not seem to be associated with reductions in morbidity at each age. There is little reason to think that the health status of the typical 65-year-old twenty years from now will be better than it is now. Hence there is little basis for proposing a redefinition of the elderly.

Reductions in mortality, however, will be associated with increases in the variance of health status at any given age. Medical progress will make the best-off members of any given cohort still better off, while marginal survivors are likely to be in very poor health. This suggests the desirability of flexible policies when dealing with the aged population. Policies based on necessarily arbitrary age thresholds will become less and less satisfactory as the variance of health status in the population increases.

Our analysis has focused on the effects of mortality reduction taken as a whole, without distinguishing the cause. Further detailed investigations would be extremely valuable, since different types of reductions in mortality will have different effects. Reductions in accidents, for example, may not change the composition of the population in an unfavorable way, while the implantation of artificial hearts will increase dependency among the surviving population. At the margin, it would probably be desirable to tilt

medical progress toward policies of the first type. The criterion of maximizing total years of lifespan tends to favor policies directed at persons who, if saved, will be in good health. Maximizing the number of lives saved in the current year, another criterion, is more likely to lead to saving individuals who will be unhealthy and have a high risk of death.

Table A-1. Estimates of Gompertz Models for Mortality Rates, Age 55 and Over[a]

Birth cohort	*Constant(α)*	*Age − 55 (β)*	R^2	*N*
Men				
1880	−4.103	0.073	0.997	31
	(−0.013)	(0.0007)		
1890	−4.169	0.075	0.999	31
	(−0.009)	(0.0005)		
1900	−4.247	0.077	0.997	26
	(−0.013)	(0.0009)		
1910	−4.215	0.067	0.986	16
	(−0.019)	(0.0022)		
Women				
1880	−4.531	0.077	0.992	31
	(−0.023)	(0.0013)		
1890	−4.786	0.081	0.996	31
	(−0.016)	(0.0009)		
1900	−5.017	0.085	0.999	26
	(−0.006)	(0.0004)		
1910	−5.001	0.069	0.996	16
	(−0.011)	(0.012)		

a. Estimates are based on cohort life table data provided by the Metropolitan Life Insurance Company. The estimating equation is:

$$\log [- \log (1 - q_t)] = \alpha + \beta \times (t - 55) + \epsilon_t,$$

where t corresponds to chronological age. Estimation is by ordinary least squares; numbers shown in parentheses are standard errors.

Comment by Joseph Newhouse

Poterba and Summers have explored some consequences of the decline in mortality rates among the aged. In recent years the decline has been striking; for example, they calculate that almost one-fourth of white women who were age 80 in 1980 would not have survived if mortality rates had remained unchanged between 1960 and 1980.

It is not surprising that such a large demographic change—perhaps comparable to the postwar baby boom in both its unexpectedness and its consequences—would have potentially large implications for public policy. To take but one example, in 1982 nearly one-fourth of women over 85 resided in nursing homes and roughly half of nursing home expenditures

are paid for by medicaid. In part because of the decline in mortality rates, the number of women over 85 approximately tripled between 1960 and 1980 and is expected to double again by the year 2000. Little wonder that real expenditures on long-term care have risen by a factor of twelve in twenty years.

The decline in mortality among the elderly, although of obvious importance, has been little explored by economists. (Victor Fuchs is a clear exception.) The Poterba and Summers analysis is therefore welcome. Poterba and Summers reach a number of striking conclusions:

1. There continues to be considerable debate within the health policy community about what might be called the "one-hoss-shay" model of aging. This hypothesis, first put forward by Fries, suggests that there is a natural limit to the life cycle that we are beginning to approach.[31] Fries argues that we can therefore look forward to rather modest future gains in life expectancy. Medical progress, however, will continue to reduce morbidity and to improve the quality of life before that limit. Concomitant with the improvements in morbidity, there may well be reductions in medical care spending. In effect, disability and the costs of illness will become compressed into a brief interval near the end of life.

There is an opposite view, which holds that life expectancy will continue to increase as medical progress keeps alive individuals who would otherwise have died. These individuals, however, may not have very high-quality lives and may have above-average medical expenditures.[32] Thus, in marked contrast to the one-hoss-shay model, medical progress may be steadily extending life at increasing marginal cost and decreasing marginal quality of life. This view implicitly suggests that the return on biomedical research is steadily falling.

Poterba and Summers suggest that both these positions have some validity. Medical progress can both reduce costs and improve health for those who would have survived in any event, while it simultaneously may keep alive those who would have died ("marginal survivors") in a rather poor state of health and at high cost. Indeed, they go even further and suggest that quantitatively these two effects approximately offset. As a result, they conclude that future age-specific medical care costs can be projected using current age-specific information.

31. Fries, "Aging, Natural Death, and the Compression of Morbidity."

32. Joseph P. Newhouse, "The Erosion of the Medical Marketplace," in Richard M. Scheffler, ed., *Advances in Health Economics and Health Services Research*, vol. 2 (Greenwich, Conn.: JAI Press, 1981), pp. 1–34.

Their projections using current age-specific information are quite ominous. The number of institutionalized persons is estimated to grow around 60 percent by the year 2000. They point out the low likelihood that the costs of such large growth could be offset either by greater efficiencies in the delivery system or by greater cost sharing. (I agree, although some might argue that a more efficient delivery system could in fact offset them.)

2. Because Poterba and Summers argue that the health status of the average aged person is not likely to change (health status gains among those who would have survived anyway are offset by increased survival of those with greater risk of death, or to use their term, greater frailty), they conclude that there is little basis for increasing the retirement age to more than 65 years.

3. Finally, they point out that it would be desirable to "tilt medical progress" to take account of the quality of lives saved by medical advances; moreover, the maximal number of lives saved may not be an appropriate criterion in appraising proposed innovation. Reductions in accidents from improved highway safety, for example, may not increase the average frailty at any given age, but implantations of artificial hearts may well do so; if the cost and benefits of a measure to reduce accidents equals those of an artificial heart, the former would be preferred.

The Argument

I suspect many of Poterba and Summers's conclusions are correct, although the supporting evidence is far from compelling. The first evidence introduced is the Vaupel, Manton, and Stallard model of heterogeneity of risks. This model illustrates how, if mortality rates fall generally, the surviving population will contain a higher fraction of those at greater risk (higher average frailty) than if mortality rates are constant. Poterba and Summers use this model to suggest what life expectancies might have been if the average frailty in the population did not tend to rise over time (that is, if selection did not operate). The difference between actual life expectancies and frailty-corrected life expectancies is about the same in absolute terms at all ages; hence the percentage gains are greater at older ages (where life expectancies are less). In the context of the Vaupel, Manton, and Stallard model, these percentage gains reflect the percentage fall in mortality rates facing a cohort at a given point in time; actual

mortality rates also include the selection effect and hence show less of a fall. Because the percentage gain in frailty-corrected rates is greater at older ages, Poterba and Summers infer that "these data cast doubt on the view of Fries and Crapo and others that longevity is currently pushing up against an upper limit." Put another way, corrected for the selection effect, the greatest percentage improvements in mortality rate appear to be at older ages, contrary to the one-hoss-shay hypothesis.

The difficulty with Poterba and Summers's quantitative conclusion from the Vaupel, Manton, and Stallard model is that it is dependent on both the structure of the model and the parameter used to operationalize it. Although it will seem plausible to almost any economist that there is some heterogeneity, so that the qualitative argument of Poterba and Summers is correct, the conclusion of offsetting effects obviously depends on the amount of heterogeneity. Here it is somewhat disconcerting that their own estimates suggest an absence of heterogeneity, a result they dismiss as unreasonable. The quantitation in the paper, therefore, uses an estimate of heterogeneity made by Vaupel, Manton, and Stallard. This estimate may, of course, be correct (Poterba and Summers regard it as conservative), but no formal estimate of the uncertainty surrounding this parameter is given. In light of Poterba and Summers's independent estimate of no heterogeneity, however, the uncertainty appears to be considerable. Uncertainty surrounds not only this parameter, but also whether the structure of the model is correct; that is, whether frailty is in fact distributed with a gamma distribution. This is important because one must specify how much the frailty of the marginal survivor increases as death rates fall. Because the quantitative conclusion leans heavily on that assumption, it would be better if the argument that the factors offset could be sustained on the basis of other evidence, and Poterba and Summers offer some.

They note that if heterogeneity is present, the increase in average frailty, all else being equal, increases morbidity—although of course medical progress could also be working to reduce morbidity as well as mortality. They contrast this view with a recent analysis by Fuchs, which did not account for heterogeneity (or selection). Fuchs argued that health may be better measured by years until death rather than by age. The basis of his argument was that medical expenditure is concentrated in the two years before death; essentially the argument is that health dramatically worsens just before death. Because years till death is increasing among the aged, this argument implies that average health should be increasing. Fuchs went on to suggest that the conventional retirement age of 65 might be

increased as a result. Poterba and Summers point out that Fuchs has ignored the selection effect.

The data they cite do not show much of a trend in morbidity: (1) the institutionalization rate generally increased after 1963 but fell from 1977 to 1982 (and in some age groups from 1973 to 1977); (2) age-specific medicare expenditures showed little trend between 1966 and 1982: the percentage of the medicare population seeking care increased at all ages between 1966 and 1982, but increased more for the young than the old; and (3) disability days showed little trend between 1961 and 1980. Poterba and Summers conclude from this relative constancy that the selection effect and medical progress in reducing morbidity may be offsetting.

Again I am sympathetic to their general point that heterogeneity may be important and should be accounted for, but I find their empirical evidence less than compelling. Essentially, the argument centers around whether one would have observed a fall in morbidity had not improved life expectancy led to survival of the less fit.

The problem with the evidence at hand is, as Poterba and Summers acknowledge, that many factors that are not controlled for affect the measures of morbidity; thus interpretation is difficult. For example, coverage of long-term care services by medicaid undoubtedly increased institutionalization rates, whereas the increased availability of home health services probably decreased them. The spread of supplementary insurance policies (medigap) has undoubtedly contributed to the percentage of medicare enrollees who obtain services. Disability days may reflect primarily the incidence of acute illnesses and have little to do with frailty or medical progress. It is not clear that any of these omitted variables are age-neutral. As a more nitpicking comment, table 8, which presents age-specific medicare expenditure patterns, should probably also include expenditure on long-term care. Even with long-term care included, however, I doubt that the patterns would look very different. The main point, however, is straightforward: because changes in factors other than average frailty were occurring, the data are simply not very decisive one way or the other.

Finally, Poterba and Summers consider data from the Retirement History Survey to examine Fuchs's claim that years to death may be a better measure of health status than age. They use both years to death and age to explain hospital costs in 1968 for the sample included in this survey. They find that age and years to death exert independent and marginally significant effects on hospital costs. They derive a similar (but statistically stronger) finding for the retirement decision.

As a minor technical suggestion, I think they might do better using the log of hospital costs, given the strong skewness of the distribution of hospital expenditure. But I find their conclusion that there are independent effects of these variables quite plausible. Age may be a proxy for accumulating chronic conditions from the aging process, while proximity to death may indicate a sudden worsening of one's health. These effects, however, can occur independently of selection; thus they cannot be used to infer selection.

The Conclusions

I find Poterba and Summers's conclusion that future medicare costs and costs of institutionalization can be predicted from current age-specific information consistent with the model and the empirical work, but in practice too sweeping because it ignores the advent of the medicare prospective payment system. First, and quite mundanely, the federal government is attempting to cap outlays on medicare, whereas previously it had been rather passive. Future medicare expenditure patterns and even age-specific patterns may thus be quite different, depending on the details of the payment system. For example, if a procedure used heavily by the younger aged (such as surgery) is downweighted, age-specific patterns would be affected. Second, and with greater economic content, the previous cost reimbursement method of financing hospital use may have induced much of the technological change that Poterba and Summers find of dubious merit. Because insurance paid almost fully for any marginal test or procedure, the economic calculus that previously applied to an innovation in inpatient care was (approximately) whether its expected benefit was greater than zero.[33] The advent of prospective payment changes this calculus; cost-saving innovation is rewarded, but innovation of new products (such as the artificial heart) may or may not be rewarded, according to government decisions about how to reimburse for these new technologies. Such uncertainty may well retard new product innovation and hence the rate of expenditure increases. Moreover, this too may not be age-neutral. Thus there is a high degree of uncertainty surrounding any predictions of future medicare costs simply because of the changed reimbursement system; current age-specific patterns may not be a good guide.

33. Edward L. Schneider and Jacob A. Brody, "Aging, Natural Death, and the Compression of Morbidity: Another View," *New England Journal of Medicine*, vol. 309 (October 6, 1983), pp. 854–56.

Second, Poterba and Summers argue that there is little basis for proposing a redefinition of the elderly because the health status of the typical 65-year-old twenty years from now is likely to be about the same as it is now (rather than better, in which case one might advance the conventional retirement age to some higher figure). I also have some doubts about this argument. First, it seems to accept that 65 is the appropriate retirement age given current health status. In light of how the convention surrounding age 65 arose, however, this is a doubtful proposition. But suppose one accepts that at current levels of health status 65 is the optimal retirement age. Then if their premise is correct that average health status will remain unchanged, should the retirement age stay fixed? I am not persuaded for two reasons. The first is Poterba and Summers's own emphasis on heterogeneity, which straightforwardly seems to imply that the retirement age should not be uniform. The second is that the effect of selection on frailty may weaken. Continued increases in frailty at age 65 depend on continuing reductions in mortality rates before age 65, but mortality rates cannot fall below zero (or more realistically, there is probably some irreducible minimum above zero). Hence the increase in frailty is bounded. Moreover, depending on the actual distribution of frailty in the right tail, as mortality rates fall toward zero, the increase in average frailty can in principle fall (that is, the second derivative can be negative), although this is not the case if frailty follows a gamma distribution, as Poterba and Summers assume. Although the increase in frailty is bounded, improvement among the health status of those aged 65 who survive—whether from improvements in medicine such as the artificial hip or from changes in life-style such as changed diets—is not obviously bounded. Thus, even if one accepts the argument that selection and medical progress have approximately offset each other in the past twenty years, they may well not do so in the next twenty.

Third, Poterba and Summers argue that at the margin it would be desirable to reorient medical progress toward policies that are less selective in their effects on mortality. It is not clear that this is possible; that is, it is not clear whether existing allocations already account for this, so that what they are talking about is a reduction in research and development. In any event, one must consider willingness to pay for these advances; it is possible that individuals (including frail individuals) are willing to pay for progress that has selective effects. This certainly may be true of individuals' willingness to pay in their own behalf. Additionally, it appears difficult for the American political process not to save identifiable lives when

the means are available to do so. This implies that there is likely to be a market for technology that saves such lives, once developed, and in turn research on such technologies may be supported by private monies—witness the current support of the artificial heart by a private firm. Thus any attempt to reorient federal research and development policy may be ineffective in reorienting the total research and development effort.

Although these comments have emphasized the uncertainties that surround these issues, I close by emphasizing that the issues Poterba and Summers are dealing with are difficult empirically—indeed, it is hard to offer constructive suggestions that would make major improvements in their analyses—and their principal point concerning the importance of selection effects is quite likely to be correct.