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3 Longer Life Expectancy? Evidence from Sweden of Reductions in Mortality Rates at Advanced Ages

James W. Vaupel and Hans Lundström

Life expectancy at current mortality rates in Western Europe, the United States, Canada, Australia, New Zealand, and Japan exceeds 75 years; in Japan and the Scandinavian countries (and in some states of the United States, such as Hawaii and Minnesota) life expectancy for women is around 80 years. Many demographers, gerontologists, and others believe that life expectancy will continue to rise slowly until it reaches an upper limit of perhaps 85 years. Fries (1980) has helped popularize this idea, but as adumbrated below, numerous others have contributed to it. Demeny (1984), in making long-term population forecasts for the World Bank, assumed that even by the year 2100 there would be no country with a life expectancy above 82.5 years.

Other researchers are skeptical about the existence of an upper limit to life expectancy, at least at an age as early as 85. They foresee continuing and perhaps even accelerating progress in reducing mortality rates at all ages, including the most advanced ages (Manton, Stallard, Tolley 1991). Some projections suggest that the life expectancy of the current generation of children in the United States might be 100 years or more, if progress in reducing mortality rates continues over the next century (Vaupel and Gowan 1986; Guralnik, Yanagishita, and Schneider 1988.)

If life expectancy remains at about current levels, demographers can make fairly long-term forecasts of the future size of the elderly population with reasonable accuracy. Everyone who will be more than 60 years old in the year 2050 has already been born; if age-specific death rates remain more or less constant and if migration rates are low or predictable, then the current popula-

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tion can be projected forward to estimate the population of the elderly up through the middle of the next century.

Suppose, however, life expectancy rises so that children alive today live 100 years on average, instead of 75 years. The elderly population will increase dramatically in size and the oldest-old population of those above age 85 will explode in number (Ahlburg and Vaupel 1990).

Hence, theory and evidence concerning the prospects for longer life expectancy are of fundamental relevance in assessing the impact of population aging. In particular, major issues in the economics of aging hinge on projections of the old and oldest-old populations. These issues include trends in (1) healthcare demand and costs, (2) the financial soundness of social security systems and pension plans, (3) individual, corporate, and governmental decisions about the age of retirement, and (4) the nature and extent of intergenerational transfers of resources.

A major biomedical uncertainty lies at the core of the disagreement between those who foresee life expectancy leveling off at about 80 or 85 years and those who predict more radical increases to a century or more. Does the force of mortality (i.e., the age-specific hazard of death) (1) sharply and inexorably rise for the typical individual to extremely high levels around age 85 or (2) increase after age 85 at about the same rate or even at a slower rate than before age 85, with the likelihood that the rate of progress being made in reducing the force of mortality among the very old will be of the same order of magnitude as the rate of progress being made among the younger old?

The first perspective implies that life spans are limited. Individuals may differ somewhat in their maximum potential life spans, with some individuals having a potential of 100 years and others a potential of 75 years. On average, however, the typical individual's longevity is unlikely to exceed the natural limit of 85 years or so that has prevailed for millennia. Most of those who adhere to this perspective believe that continued progress in reducing mortality rates up to age 75 or so is likely to be made, so that death before age 75 will become rare. Consequently, life expectancy will approach the length of the typical maximum life span, i.e., about 85 years. Eventually, some extraordinary breakthroughs may be made that permit humans to live beyond their natural life spans, but when such breakthroughs will occur, if ever, is uncertain.

This general point of view is often illustrated with diagrams showing an increasing rectangularization of survivorship curves or showing bell-shaped distributions, centered around age 85, of what Fries (1983) describes as "natural death (due to senescent frailty)." Such survivorship curves and distributions of deaths imply that little or no progress can be made in reducing death rates after age 80 or so.

The second perspective implies that the force of mortality rises fairly smoothly to very advanced ages exceeding 100 years or more—there is no sharp increase for the typical individual around age 85, and there may even be some gradual lessening of the rate of increase after age 90 or so (as implied

by the power function or the logistic function used, instead of an exponential function, in some models of mortality). Furthermore, there is no discontinuity around age 85 in the rate of progress that is likely to be made in reducing the force of mortality, so that substantial reductions in mortality rates will probably be achieved at all ages. Consequently, life expectancy will continue to gradually but steadily increase and may rise to 90, 95, or even longer by the year 2050. Major biomedical breakthroughs are likely over the course of the next century, although the exact nature and significance of these breakthroughs cannot now be foreseen: these breakthroughs may result in some acceleration in the rate of progress made in reducing the force of mortality, so that a life expectancy of well over 100 years, less than 100 years from now, cannot be ruled out. In contrast to the limited-life-span paradigm, this might be called the mortality-reduction paradigm.

Given the current state of knowledge, no judicious researcher can claim to know for sure which of these two paradigms is more correct—or whether some combination of them or some entirely different perspective will eventually prove to be true. Furthermore, each of the two paradigms has numerous variants that have not yet been conclusively shown to be inconsistent with reliable empirical evidence.

Broadly speaking and with many caveats, the limited-life-span paradigm can be associated with the stream of research done by Pearson (1923), Pearl (1923), Clarke (1950), Bourgeois-Pichat (1952, 1978), Comfort ([1964] 1979), Ryder (1975), Hayflick (1977, 1980), Sacher (1977), Keyfitz (1978), Kohn (1982), and their colleagues. The most prominent recent advocate and popularizer of this general perspective is Fries (1980, 1983, 1984; Fries and Crapo 1981; Fries, Green, and Levine 1989); useful reviews are also provided by Rosenfeld ([1976] 1985) and Gavrilov and Gavrilova (1991). These researchers generally assume that there are biological barriers to longer life expectancy; in contrast, Olshansky, Carnes, and Cassel (1990) stress practical barriers that may effectively limit life expectancy to values less than 85 years or so. Whether the barriers are practical or genetic is, however, rarely explicitly addressed: in much of the gerontological literature it is simply accepted as a stylized fact that natural or senescent death implies that mortality rates cannot be substantially reduced at advanced ages. Harman (1991) and Lohman, Sankaranarayanan, and Ashby (1992) provide two recent examples of the strength and persistence of this point of view.

The possibility that the mortality-reduction paradigm may be more correct is implied by most of the process models of mortality developed from Gompertz (1825) onward. This viewpoint has been cogently argued by Manton (1982; Manton and Soldo 1985; Manton and Woodbury 1987; Myers and Manton 1984; Manton et al. 1991) and is supported either explicitly or implicitly by Schatzkin (1980), Schneider and Brody (1983), Peto, Parish, and Gray (1986), Vaupel and Owen (1986), Vaupel and Gowan (1986), Schneider and Guralnik (1987), Poterba and Summers (1987), and Rowe and Kahn (1987).

The key reason that the controversy between the limited-life-span and mortality-reduction paradigms has not been resolved is that there is relatively little reliable data on mortality rates over age, time, and sex among the oldest old (i.e., those over aged 85). Indeed, it is remarkable how little is known, considering the rapidly increasing population at advanced ages and the high life-table probability, approaching 50 percent for females in some countries, of survival past age 85.

Very few published human life tables extend past age 85, and the population and death counts that are available for the oldest old tend to be suspect. As reviewed (and bewailed) by numerous demographers (including Shryock and Siegel 1976; Mazess and Forman 1979; Rosenwaike 1981; Horiuchi and Coale 1983; Spencer 1986; Coale and Kisker 1986, 1990; Kannisto 1988), various kinds of gross errors are common in reported age-specific deaths and population sizes above age 85. These errors—such as age heaping caused by rounding off of ages to the nearest age divisible by five or ten, the tendency of some older people to exaggerate their ages, the fact that a relatively few errors in misclassifying younger people as very old people can swamp actual counts of very old people, or failures to remove the deceased from population registers so that the dead appear to survive eternally—may represent systematic biases across populations. Hence it may be impossible to reduce these errors by the usual statistical expedient of examining many data sets and either formally or informally averaging them. It is consequently essential that large, reliable databases on oldest-old human mortality be assembled and analyzed.

The most reliable data on mortality rates up to the most advanced ages over a long period of time pertain to Sweden. Excellent data exist for Sweden since 1750; superlative data have been archived since 1895. The published Swedish data that are readily available are highly accurate, but even these data have some deficiencies at advanced ages. In particular, much of the published data is smoothed by actuarial methods after age 90 or so, and the most widely available mortality rates are based on aggregated data on several years of age and time rather than on single years of age and time. Furthermore, the data, once published, have not been revised as new information (from censuses or cohort death counts) has become available.

Using unpublished information in the archives of Statistics Sweden, one of us (Lundström) is in the process of meticulously verifying, correcting, and computerizing the death counts and population counts needed to estimate mortality rates at advanced ages in Sweden from 1750 to 1992. For this article, we made use of a nearly completed version of the Lundström database for 1895 to 1990. A few minor changes may be made to a few of the death and population counts in this database, but the version we used is undoubtedly extremely close to the final version.

3.1 Force of Mortality at Ages 85, 90, and 95

Figure 3.1 plots the force of mortality for Swedish females at ages 85, 90, and 95, from 1900 through 1990. Other ages between 80 and 100 show similar patterns.

The force of mortality, also known as the hazard or intensity of death, is a measure favored by demographers to capture the level of mortality. It is defined, at age x and time y, by

$$\mu(x) = -\frac{ds(x, y)/dx}{s(x, y)}, \quad y = y_0 + x,$$

where $s(x, y)$ is the proportion of the cohort born x years ago that is surviving at time y, and y_0 is the time the cohort was born. The Swedish data are available by single years of age and time, so a discrete approximation must be used to estimate μ. We used the standard approximation

$$\mu(x, y) = -\ln(1 - D(x, y)/N(x, y)),$$

where $D(x, y)$ represents the number of deaths among the cohort of people who were between exact ages $x - 1$ and x on January 1st of year y, and $N(x, y)$ represents the number of people in this cohort on January 1st. Note that the

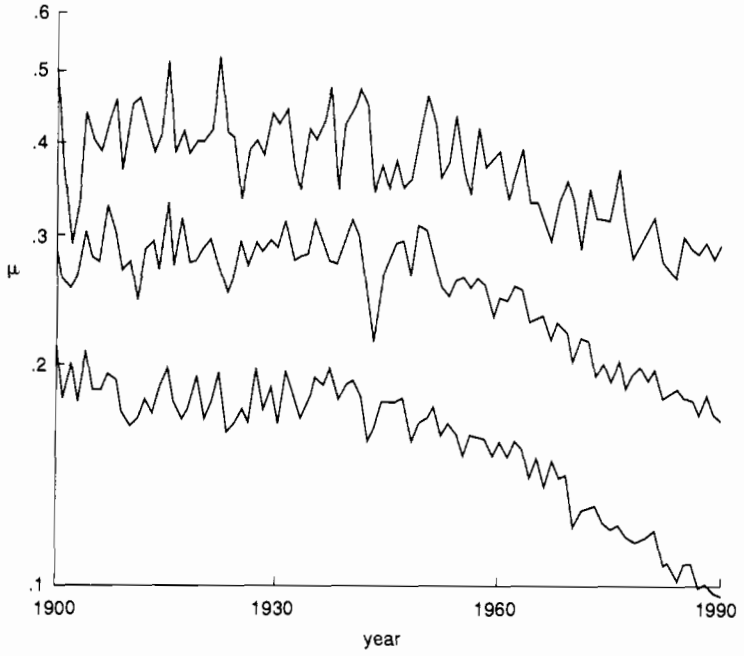

Fig. 3.1 Force of mortality for females, ages 85, 90, and 95: Sweden, 1900–90

members of this cohort attain exact age x (i.e., celebrate their xth birthday) over the course of year y. Also note that in-and-out migration is ignored: net migration is negligible in Sweden after age 80.

Population sizes are small, especially at age 95, so the trajectories in figure 3.1 show considerable random fluctuation. The overall trends, however, are clear. There was little progress in reducing the force of mortality at advanced ages before 1940 or 1950. Afterward, the force of mortality declined considerably, even at age 95. At age 85, the force of mortality declined from about .2 to about .1. At age 90, the decline was from a level of about .3 to about .2. An absolute decline on the order of magnitude of .1, from about .4 to about .3, is also apparent at age 95.

As shown in figure 3.2, the trends for Swedish males are roughly similar, although less dramatic. It is clear that the force of mortality for very old males in Sweden was substantially lower in 1990 than it was in 1900, although the reduction was less than for females and the levels of mortality are higher for males than for females. At each age, the absolute decline for males was on the order of magnitude of .05, in contrast to the decline of roughly .1 for females.

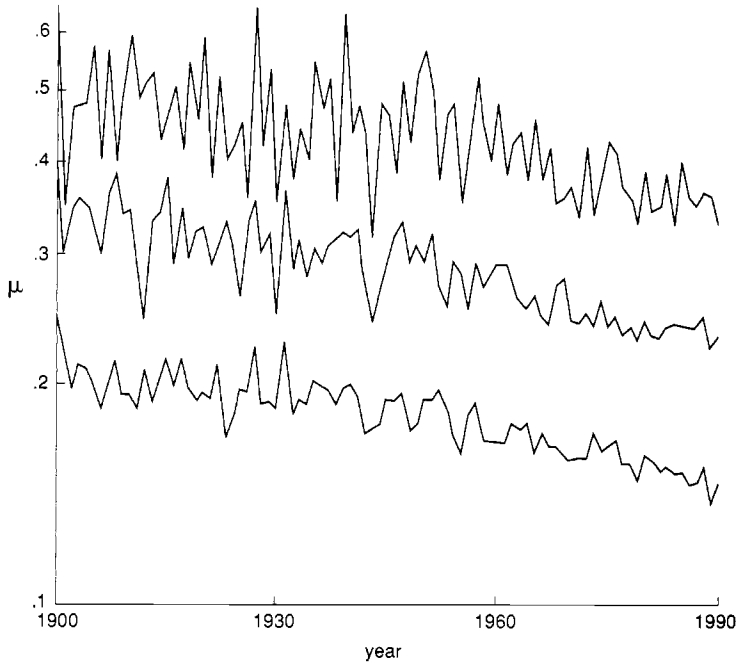

Fig. 3.2 Force of mortality for males, ages 85, 90, and 95: Sweden, 1900–90

3.2 Average Annual Rates of Progress in Reducing Mortality Rates

To summarize the overall pattern of reduction, table 3.1 presents average annual rates of progress in reducing the force of mortality for Swedish females and males over successive 20-year time periods and for people in their 60s and 70s as well as octogenarians, nonagenarians, and centenarians.

For males and for females, the average level of the force of mortality over a decade of time and age was calculated as follows:

$$\bar{\mu}(x_0, y_0) = \frac{\sum_{y=y_0}^{y_0+9} \sum_{x=x_0}^{x_0+9} \tilde{N}(x)\, \mu(x, y)}{\sum_{y=y_0}^{y_0+9} \sum_{x=x_0}^{x_0+9} \tilde{N}(x)} .$$

The $\tilde{N}$s are used to standardize the age composition of the population: we calculated the $\tilde{N}$s from the population of Sweden in the 1980s:

$$\tilde{N}(x) = \sum_{y=1980}^{1989} N(x, y) .$$

The values of $\mu(x, y)$ were calculated as described above. If the death count equaled the population count, then the standard approximation $\mu = 2$ was used. Occasionally, at ages greater than 100, it was impossible to estimate μ for some specific year, because no one was alive at that age and year. In such cases, the μ term was dropped from the numerator and a corresponding correction was made in the denominator. The average annual rate of progress in reducing the force of mortality was then calculated using

$$\rho(x_0, y_0) = -\left[\left(\frac{\bar{\mu}(x_0, y_0 + 20)}{\bar{\mu}(x_0, y_0)}\right)^{.05} - 1\right] .$$

Table 3.1 **Average Annual Rates of Progress in Reducing Mortality Rates by Age Category and Time Period**

		Time Period			
Sex	Age Category	1900–09 to 1920–29	1920–29 to 1940–49	1940–49 to 1960–69	1960–69 to 1980–89
Male	60–69	.50	.44	.28	.62
	70–79	.37	.20	.19	.62
	80–89	.36	.13	.36	.53
	90–99	.27	.11	.36	.56
	100+	1.76	−1.07	.97	.18
Female	60–69	.24	.61	1.88	1.63
	70–79	.18	.22	1.25	2.08
	80–89	.19	.10	.78	1.64
	90–99	.13	.03	.60	.94
	100+	.23	.41	.80	.49

Table 3.1 indicates that progress has been made in Sweden in reducing the force of mortality at all ages after 60 for both males and females. Estimated rates of progress fluctuate erratically for centenarian males, probably because there are so few observations for this category, but even so the general trend is toward a reduction in mortality rates. For females and for younger age categories, the picture is clear: mortality rates among the elderly are declining in Sweden and at a faster pace in recent decades than in the first decades of the century.

For males in the most recent time period, the rate of progress is roughly the same—about half a percent per year—for men in their 60s, 70s, 80s, and 90s. For females in the most recent time period, the rate of progress is about 2 percent for women in their 70s and half as much for women in their 90s. Note, however, that the rate of progress for women in their 80s is the same, 1.6 percent, as that for women in their 60s.

If rates of progress in the first 20 years of the century are compared with the most recent 20-year period, it is apparent that there has been a considerable acceleration of rates of progress. The acceleration is greater for females than for males. The acceleration is also greater in older age categories than in younger ones, at least in the age categories, below age 100, where there are substantial numbers of observations.

The overall acceleration in rates of progress and the greater acceleration at older ages may reflect actual changes on the individual level: the elderly today may be healthier than in the past, and they may be receiving better health care. A supplemental explanation was suggested by Vaupel, Manton, and Stallard (1979). Progress in reducing mortality rates at younger ages makes it more difficult to make progress at subsequent ages if the persons whose lives are saved are frail and vulnerable. In effect, progress in reducing cohort mortality rates at younger ages masks the true rate of progress (controlling for compositional changes) at older ages. However, as mortality rates in an age category decline, this effect diminishes in importance, resulting in an apparent acceleration in rates of progress.

3.3 Lexis Maps of Force of Mortality

Another way to summarize data concerning a surface of demographic rates over age and time is to present a Lexis map, i.e., a shaded contour map of the surface (Vaupel, Gambill, and Yashin 1987). Figure 3.3 displays a Lexis map of the force of mortality for Swedish females at ages 80–111 from 1900 through 1990. Figure 3.4 displays a corresponding map for Swedish males.

The data available to us include death counts by year of birth as well as by current age and year. Furthermore, the data include population counts of those attaining a specific age in some year (e.g., the number of those who celebrated their 85th birthday in 1970) as well as counts of the number of people at a given age on January 1st of a given year. Hence it is possible to estimate the

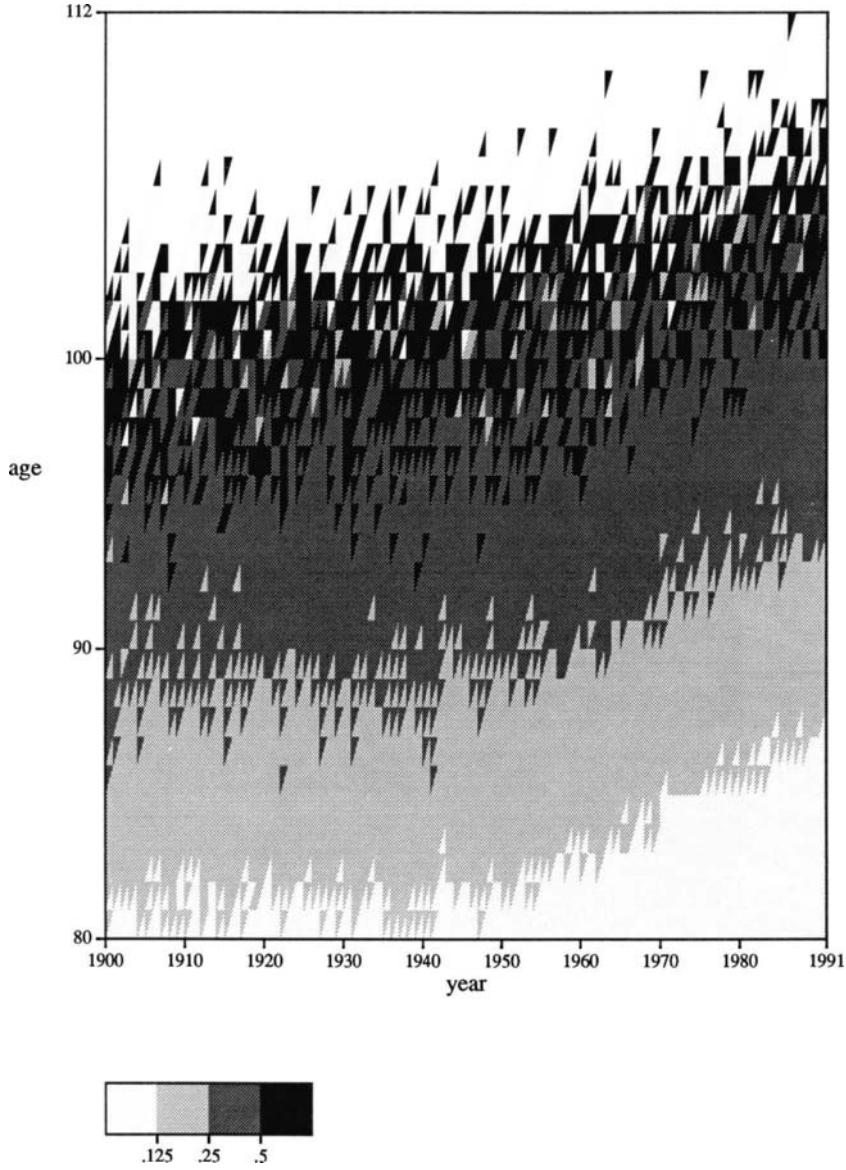

Fig. 3.3 Force of mortality for females 80–111: Sweden, 1900–90

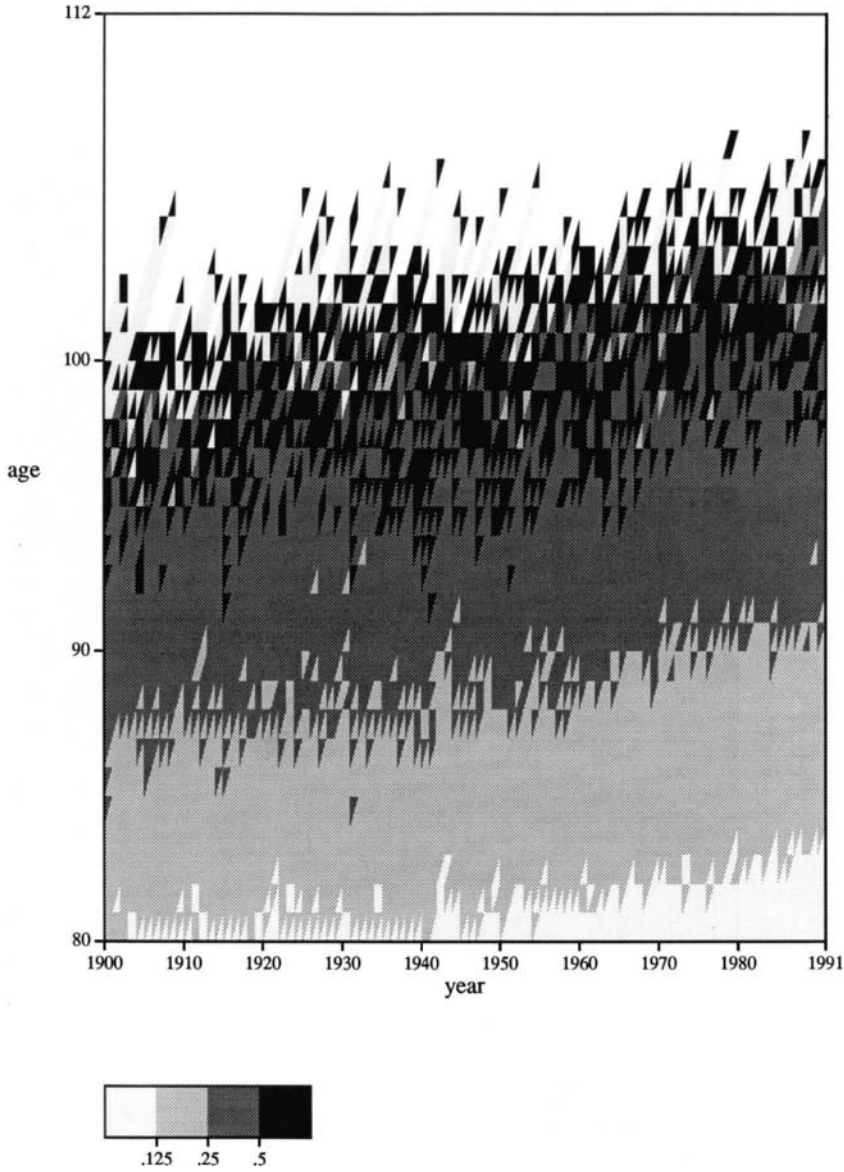

Fig. 3.4 Force of mortality for males 80–111: Sweden, 1900–90

force of mortality for triangular categories of age and time. Let $q = D/N$ be the ratio of the death count to the population at risk, in one of these triangles. To convert this into an annual probability of death, let

$$q^* = 1 - (1 - q)^2 .$$

Then, analogous to the formula used earlier, the force of mortality can be estimated by

$$\mu = -\ln(1 - q^*).$$

The four shades of gray in figures 3.3 and 3.4 represent four levels of this estimated force of mortality. The light gray tones along diagonals above age 100, terminated by a black triangle, generally represent cohorts with one remaining member: the force of mortality is zero until this person dies.

Consider the age at which the force of mortality for females crosses the level of .125, as shown in figure 3.3. Until 1945 or so, this age is around 81; by 1990, the age is up around 87. One interpretation of this is that an 87-year-old Swedish female in 1990 was as healthy (at least in terms of probability of death) as an 81-year-old Swedish female in the first four decades of the twentieth century. The age at which the force of mortality for females crosses the level of .250 increases by about five years from a level fluctuating around 89 to a level of 94. Despite substantial statistical noise, a shift upward is also apparent at the level of .5, and there is also a clear increase in the maximum age attained. The record longevity is 111 years, attained by the grandmother of an employee of Statistics Sweden.

For males, as shown in figure 3.4, the surface of mortality rates is higher than for females. Furthermore, the upward shift is less substantial at the .125, .25, and .5 levels and in the maximum age attained. Nonetheless, it is clear that there has been a definite shift, on the order of three years or so. As noted above, this can be interpreted as the result of a downward shift in mortality curves or, alternatively, as a delay in the aging process: elderly Swedish males in 1990 can be considered to be three years "younger" (in terms of their risk of death) than Swedish males of the same age in the first part of this century.

3.4 Remaining Life Expectancy

A final perspective on the decline in oldest-old mortality rates in Sweden is presented in table 3.2. For the various decades from 1900 until 1990, the table gives, for males and females, the age at which remaining life expectancy is two years and the age at which remaining life expectancy is five years.

The numbers given are based on decennial life tables for each decade. The age-specific mortality rates, for single years of age, that form the basis of these life tables were calculated using the following standard formula:

Table 3.2 **Age at Which Remaining Life Expectancy is Two Years or Five Years**

	Two Years Left		Five Years Left	
Period	Males	Females	Males	Females
1900–09	93.9	95.3	80.5	81.7
1910–19	94.1	95.7	80.7	81.8
1920–29	95.3	96.1	81.2	82.0
1930–39	94.4	95.4	80.9	81.5
1940–49	95.0	96.9	81.6	82.3
1950–59	95.9	97.3	81.8	82.9
1960–69	96.3	99.0	82.5	84.0
1970–79	97.6	99.7	83.2	85.8
1980–89	98.1	100.1	83.7	86.7

$$q(x, y_0) = \frac{\sum_{y=y_0}^{y_0+9} D(x, y)}{\sum_{y=y_0}^{y_0+9} N(x, y)},$$

where, unlike above, $D(x, y)$ now represents the number of deaths within a year of time of people who attained exact age x in year y, and $N(x, y)$ represents the number of people who attain age x in year y.

Note in table 3.2 that, for both males and females and when remaining life expectancy is either two years or five years, there was little net change between the decade 1900–09 and the decade 1930–39. From the 1930s to the 1980s, however, the shifts were substantial. For males, the age at which two years of life expectancy are left increased by almost four years, from 94.4 to 98.1. For females, the corresponding shift was close to five years, from 95.4 to 100.1. The age at which remaining life expectancy is five years increased for males by almost three years, from 80.9 to 83.7. For females, the increase was five years, from 81.5 to 86.7.

As suggested earlier, one interpretation of these shifts is that the process of aging has been slowed or delayed in Sweden such that elderly Swedish men are effectively three or four years "younger" than they used to be and elderly Swedish females are five years younger. Caution is required because these figures are based entirely on mortality statistics, with no information about morbidity or disability. Nonetheless, treated judiciously, this perspective suggests that certainly mortality, and perhaps health more generally, is plastic even at the most advanced ages. It has been possible, at least in Sweden, to lower the force of mortality and to significantly postpone death even among the oldest old.

3.5 Discussion

Swedish life expectancy has been among the very longest in the world for many decades. If progress can be made in Sweden in lowering mortality rates at advanced ages, then the contention that oldest-old mortality rates cannot be significantly reduced seems questionable. Using highly reliable data, we presented four perspectives on mortality changes in Sweden since 1990 among the elderly. As shown in figures 3.1 and 3.2, the force of mortality at ages 85, 90, and 95 has substantially declined, especially since 1945 or so, and more for females than for males. As shown in table 3.1, rates of progress in reducing mortality rates among the elderly have accelerated over the course of the century and from the 1960s to the 1980s ran at an average annual rate of 1–2 percent for females and half a percent for males. As shown in figures 3.3 and 3.4, the ages at which the force of mortality attains the levels of .125, .25, and .5 have shifted upward substantially since 1945 or so, by about five years for females and three years for males. Finally, as shown in table 3.2, the ages at which remaining life expectancy reaches two years or five years have also shifted upward, by about five years for females and three or four years for males. These four perspectives are consistent with each other. They indicate that the belief that oldest-old mortality rates cannot be significantly reduced is incorrect.

A variety of other strands of evidence, reviewed by Manton et al. (1991), point in the same direction. Most of this evidence pertains to small special populations followed for short periods of time or is based on the results of sophisticated mathematical modeling. The evidence from Sweden is highly reliable, pertains to a sizable national population followed since 1900, and is so straightforward that it does not have to be smoothed or filtered through a statistical model.

The available evidence, taken together, suggests that, if historical rates of progress in reducing mortality rates continue to prevail in the future, newborn children today can expect to live about 90 years on average. If, as health and biomedical knowledge develops, progress accelerates so that age-specific mortality rates come down at an average rate of about 2 percent per year, then the typical newborn today in developed countries will live to celebrate his or her 100th birthday.

Whether progress in reducing mortality rates will continue at historical levels or even accelerate is, of course, an open question. Even more uncertainty envelops an equally important question: if our children survive to become centenarians, what will their health be like during their extra life span? Will the added years be active, healthy years or years of decrepitude, disability, and misery? The answer to this question is central to forecasting the impact of population aging on health and social needs and costs, on retirement decisions and policies, and on other questions in the economics of aging, but very little is currently known about what the answer might be.

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Comment on Chapters 2 and 3

Peter Diamond

When David Wise invited me to discuss these two papers, he said that he wanted me to think about the policy implications of the findings. He said the same to Michael Hurd. Mike and I have divided up the policy world. He will talk about the relationship to Old Age and Survivors Insurance (OASI). I will talk about the relationship to Disability Insurance (DI). Since there are two other chapters about long-term care (LTC), we will not relate chapters 2 and 3 to that topic.

First, a simple overview of what these papers are about. Let $M(a,t)$ be the aggregate mortality rate as a function of age and time. We are interested in (at least) three things. How would you project M into the future? How would you examine the impact of medical interventions on M? How might you relate M to other issues such as the demand for LTC, the supply of DI recipients, the supply of labor?

There are two ways to go about projections. One is to examine aggregate statistics, whether for M or for mortality by cause, examine the history of trends, and think about extrapolation on that basis. This is what the Vaupel and Lundström chapter (chap. 3) is about. The second is to estimate mortality hazards on individual data and simulate to produce an aggregate projection. This is what the Manton, Stallard, and Singer chapter (chap. 2) is about.

One can interpret a major part of the Vaupel and Lundström paper as asking the following question. If one wants to project $M(a,t)$ based on selecting a functional form and estimating the parameters on aggregate data, what is a sensible (a priori) form for M? In particular, is it sensible to have a form consistent with declines in M at all ages that do not have an asymptotic minimum level of M bounded away from zero? The alternative is to assume that M stays

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very large at ages within the range we currently observe, that the asymptote, should the projection be extended indefinitely into the future, should not be zero.

The primary evidence brought directly to bear on this question is the presence of a trend in mortality rates at advanced ages. If there is an asymptote, it would appear that we are not sufficiently close to it for it to show up in the data, and therefore for it to play a role in functional form selection for estimations which are then used for projections out to the moderate term (100 years?).

The second topic in chapter 3 is the review of the basis for thinking that the mortality function should have an asymptote with a sharply rising section in the mid-90s. I have not read any of the literature being discussed. Without that background, I would hope that argument would be spelled out.

The Manton, Stallard, and Singer paper is of the micro simulation sort. It relates to Vaupel and Lundström's chapter in two ways. One is that disaggregated (by cause and individual) death rates should aggregate up to be consistent with our best sense of the shape of the aggregate mortality function. Second, the debate on functional form for the aggregate function is also relevant for the selection of functional form for disaggregated estimation. Unfortunately, in my reading of the Manton et al. paper, I could not tell how time entered in the functions to be estimated, so I could not tell how it related to the Vaupel and Lundström paper.

The methodology of chapter 2 involves two forms of disaggregation, if I have this right. One is by cause of death. The second comes from using individual data and introducing additional health variables into the estimation process. For example, if one has data on blood pressure at various times, one could estimate the stochastic time interdependence of blood pressures and the (lagged) stochastic structure of the relationship between blood pressure and mortality rates by cause. This is an extremely attractive way to proceed, since it opens up a way to address the additional questions I identified at the start. Disability is also identified as a powerful forecasting variable.

For example, if one wanted to consider the possible effects of a continued improvement in blood pressure and in the incidence of heart attacks in the population, one would proceed in different ways with the two different simulation models at hand. One might simply make an assumption on the extension of the trend in heart attack mortality. One would need to make some assumption (such as no effect) on the implication of the projected trend on other death rates. Alternatively, one could have an estimated relationship between blood pressure and different mortalities by cause and age and so derive the impact of blood pressure decline on all of the mortality rates by cause. Of course, one would then be making a different assumption: namely, that the interactions associated with the intervention are like those of the historical trend. That is, lowering blood pressure further is similar in its effects to previously caused improvements. If one is seeing improved dietary habits spreading to more of the population, this may be more plausible than if one is projecting a new drug

which may have side effects or other differences in effects from the historically given decline. That is, if the historical decline is diet driven, then the effects of diet on mortality by cause that do not go through blood pressure will be partially captured by blood pressure and partially captured by trend. The projection will be implicitly picking up some of this.

This is part of a familiar tension. Both macro- and micro-based projections involve assumptions, differently described assumptions. Which basis is more reliable for projection depends in part on the quality of the different assumptions. That is why it will remain useful for research to project in parallel on both macro and micro levels.

What about the relation of chapter 2 to DI? One might have a database that allowed estimation of DI receipt as a function of health variables. If these same health variables were part of this mortality estimation, one could then derive a DI-receipt simulation model. (It would also be necessary to recognize that varying administrative interpretations of DI standards have been a major cause of fluctuations in disability rates.) One would also need the mortality of DI recipients, which might use the basic model or might recognize that DI receipt was a variable of independent econometric value in predicting mortality. (One would also want a model of return to the labor force which is additional.) Getting more ambitious, one observes that retirement is often related to health variables. Combining this relationship with both the health evolution model and the mortality model, one has a potentially improved basis for projecting labor force and OASI benefits. More generally, I find this approach very attractive.

What about DI policy and the sense of the findings coming from Manton et al.'s paper? For this, I need to back up and talk about the place of DI in our panoply of programs. (In part I am drawing on my paper with Sheshinski [Diamond and Sheshinski, in press].) We have lots of programs to provide income to people with low earnings. There are welfare programs. There is UI. There is SSI. There is OASI. There is DI. There are also private programs (soup kitchens) that provide some benefits to the destitute. What is a potentially useful pattern here, and how do demographic changes affect how we might want to combine these programs as well as structure them? That is, changing demographics might call for changing the parameters of an individual program (thought of in isolation). For example, if health and labor supply improve "in proportion" to life expectancy, then one might want to simply change the parameters of OASI by changing, in proportion to life expectancy, the "normal retirement age," the age of eligibility for early benefits and, the age of eligibility for benefits independent of earnings. If life expectancy, health, and labor supply do not change in proportion, then one might contemplate a different pattern of change in the parameters.

Similarly, the desired relationship between programs might change because of demographic changes. We have programs that are universal (along the lines of a negative income tax). We have programs that are targeted at a group with

a relatively easy to measure target variable (age as a basis for OASI, at least after the start-up period, so that documentation of age happens well before eligibility). We have programs that are based on difficult to measure variables (such as DI based on the ability to work). Programs also differ in the cost of verification. Akerlof (1978) has written about the improved trade-off between distributional goals and disincentive costs that come from such targeted programs. But targeted programs are subject to both type I and type II errors. Stern (1982) has written about the choice between a targeted and a universal program as a function of the disincentives elements and the magnitude of the errors. Diamond and Sheshinski (in press) have written about combining both types of programs, incorporating awareness of both types of errors and disincentives in the choice of parameters for the two programs.

Workers become eligible for OASI at age 62. Workers remain eligible for DI until age 65. Thus there is a three-year overlap period during which workers are eligible for both programs. With the delay in the normal retirement age (but not the age of eligibility for OASI), the overlap period will grow. The overlap period is an accident of legislative history. Moreover the relative parameters of the two programs are a result of the adaptation of the parameters of the retirement program to generate a disability program, not the result of a conscious optimization over both programs. This should probably change. In particular, growing life expectancy together with health and labor supply improvements, which, I think, will not improve in proportion, will increase the range of differences in outcomes across people and so increase the importance of coordinating both programs. However, such a move is in the opposite direction to pressures in the United States and in many other countries.

In particular, following the Chilean example, there is a move toward forced savings programs that do not provide insurance for variation in the length of working life. Provision of such insurance is likely to have increased importance in the future, with growing life expectancy at older ages. The extent to which this is important is related to the impact on life expectancy of the sort of events that lead to early retirement. It would be wonderful to have a full-fledged estimation of the parameters of such interaction along the lines of Manton et al.'s chapter. Longer life expectancy, labor supply held constant, is a source of relatively lower living standard and so, on utilitarian lines, a reason for the receipt of income redistribution. The link with disability is more complicated because of the correlation with life expectancy. That is, recognizing that the groups of people who are more likely to receive disability are more likely to die young is relevant for designing programs which have given ex ante redistribution elements. I think that a detailed calculation of the conditional probabilities is important for design of disability programs relative to retirement programs. A similar approach may be a fruitful basis for design of LTC insurance as well, but that is a separate subject.

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Comment on Chapters 2 and 3

Michael D. Hurd

Understanding the determinants of the mortality risk of the elderly and how those determinants will change over time is important for public policy. For example, the future costs of the Social Security retirement program depend directly on length of life following retirement: an underestimate of life expectancy at age 65 of, say, 10 percent translates directly into an underestimate of costs of 10 percent in steady state.

Because the elderly consume medical services at about four times the rate of the nonelderly, the amount we spend on health care depends in an important way on the life expectancy of the elderly, although the link is complicated because of the bunching of medical expenditures just before death. Trends in medical expenditures make this particularly important: health-care consumption rose from 9.1 percent of GNP in 1980 to 12.2 percent in 1990, because of sustained inflation in medical services in excess of CPI inflation and because of sharp increases in age-specific use per capita. Future increases in medical prices and in age-specific use will interact with demographic changes in a way that can produce very large increases in total consumption of medical services and, of course, in the costs of the Medicare and Medicaid programs.

In this comment I will focus on the variability of forecasts. I will use the forecasts of Manton, Singer, and Stallard (chap. 2), official forecasts from the Social Security Administration, and results from the Vaupel and Lundström paper (chap. 3) to argue that our uncertainty about the course of mortality risk is great, in the sense that the range of mortality outcomes spanned by the forecasts implies a wide range of tax and transfer outcomes.

Uncertainty in Population Forecasts.

The Office of the Actuary of the Social Security Administration forecasts both the elderly and nonelderly populations from assumptions about the future course of age-specific mortality rates, rates of immigration, and fertility rates. The rates are based on expert opinion: they are not based on a theoretical

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model that has been fitted to historical data. Table IIC.1 has forecasts of the elderly population in 2040, life expectancy at age 65, and the elderly dependency ratio (the ratio of the number of elderly to the number aged 20–64) for three alternative assumptions about the course of mortality rates.[1] Alternative II is considered to be the best estimate, and it typically is used as the basis for forecasts of the cost, revenues, and balances of the trust funds. It embodies a 27 percent decline in age-specific mortality rates by 2040. Alternatives I and III are based on mortality rate declines of 13 and 40 percent, respectively, and forecasts of the trust funds that use these alternatives are often thought to bound the possible outcomes.

Alternative I has 7.5 percent fewer elderly in 2040 than the baseline (alternative II), and alternative III has 8.6 percent more. Under alternative III, life expectancy is 11–13 percent higher than under alternative II, so that in steady state costs would be 11–13 percent higher.

The baseline forecast of Manton et al. gives 51.7 million elderly in 2040 (table 2.5). I do not know of any way to analyze the causes of the difference between this number and the Social Security baseline (69.6 million); rather I would like to focus on the variation in forecasts. The forecast of Manton et al. is substantially outside the interval bounded by alternatives I and III. Furthermore, Manton et al. give simulations in which their risk factors are controlled. If the mean of each risk factor is put at its optimum but the variance is not changed, many individuals will have risk factors that are far from optimal. Nonetheless, controlling the mean in this way will increase the number of elderly to 79.9 million by 2040 (table 2.5), an increase of 54 percent over baseline. If, in addition, the variance of the risk factors is put to zero, so that the risk factors of each individual are at their optima, Manton et al. forecast 127.5

Table IIC.1 Number of Elderly, Life Expectancy, and Elderly Dependency Ratio

Year and Alternative	Mortality Reduction (%)	Number (millions)	Life Expectancy at Age 65		Dependency Ratio
			Male	Female	
1990	na	31.9	15.0	18.8	.21
2040					
I	13	64.4	15.6	19.4	.33
II	27	69.6	17.2	21.2	.39
III	40	75.6	19.3	23.5	.47

Source: OASDI Board of Trustees (1990).

1. The table has the Social Security area population, which differs slightly from the U.S. population because it includes some additional geographic areas such as Puerto Rico. The alternatives differ by assumptions about fertility and immigration as well as mortality rates, but variation in the fertility and immigration assumptions will have only marginal effects on the elderly population in 2040.

million elderly by 2040, an increase of 246% over baseline (table 2.5). These give, of course, enormous variation in the number of elderly compared with the forecasts of the Office of the Actuary.

A way to judge the importance of the variation is in terms of its effects on Social Security tax rates. The Trustees' Report has a sensitivity analysis, which gives the ceteris paribus change in net trust fund income resulting from a change in mortality assumptions. Moving from alternative I to alternative III, which increases the elderly population by 17 percent, requires that taxes increase by about 0.85 percent of taxable payroll each year from now to 2039 (OASDI Board of Trustees 1990, table B2, 50-year balance). Taking the baseline of Manton et al. and assuming a constant response of payroll taxes to percentage changes in the elderly population, I estimate that under the first forecast of Manton et al. (mean risk factors at their optimum) the tax would have to be 2.7 percent of taxable payroll greater than under baseline; under the forecast that puts the variance of risk factors to zero, the tax would have to be 7.3 percent of taxable payroll greater. The latter figure is rather large, about a 50 percent increase in the payroll tax. While the former figure, an increase of 2.7 percent, may seem moderate, it should be kept in mind that it is only moderate because it is levied over each of the next 50 years: if the tax is not levied until the baby-boom generation begins to retire, it will be much greater. Furthermore, it is greater by a factor of 3.2 than the variation in the tax rate between alternatives I and III.

Although Vaupel and Lundström have no population forecasts, the basic point of their paper is the same as that of Manton et al's: our uncertainty about the upper bound of the future elderly population is great. If the Fries hypothesis is correct, life expectancy can increase but not much beyond those figures given in my table IIC.1 under alternative III. Vaupel and Lundström argue that Fries is wrong and that there is no theoretical limit to life expectancy: even if the risk factors in Manton et al. are put to their optima, their population forecasts could be too low. In this event, the effects on the Social Security retirement system and the health-care system would be practically devastating.

Population Forecasts and Changes in Risk Factors.

In the forecasting model of Manton et al., the actual population could differ from the forecast population for at least two reasons: the choice of the level and variance of the risk factors used in the simulations could be different from actual future risk factors, or the model of mortality risk may not give the right change in mortality risk for a change in risk factors because it is incorrectly specified. The preceding section discussed how the forecasts vary as the risk factors vary. In this section, I will give an example that shows how difficult it is to find a correctly specified model. If the model is not correctly specified, we should be even more uncertain about the future elderly population because, even if we know with certainty the future course of the risk factors, we would

still be uncertain about the course of mortality risk. The example will consider the relationship between mortality rates and exercise.

Suppose, as in Manton et al., we want to use epidemiological (nonexperimental) data such as the Framingham data to find the effects of exercise and cholesterol on mortality rates. Following them, we would fit a vector ARMA to the risk factors (exercise and cholesterol) and a mortality hazard which would depend on the risk factors and possibly on their past levels. Other risk factors such as age, sex, and marital status would also be used, but they are not necessary for this example. This system could be used to forecast values of exercise and cholesterol and, hence, mortality rates, and, therefore, it could forecast the population. We could get good forecasts if other unobserved determinants of risk factors and mortality evolve as they have in the past.

Now suppose we want to forecast the response to a change in a risk factor, say, exercise. As in Manton et al. this would involve changing a risk factor and, through simulation, finding the new forecast population. To illustrate the range of outcomes, I introduce two health models.

In health model A, exercise does affect mortality risk because it affects unmeasurable healthiness, which, in turn, affects mortality risk. People choose exercise levels by whim. In model B, exercise has no effect on healthiness or on mortality risk. However, individuals with differing levels of healthiness face differing costs of exercise: exercise is unpleasant or even painful for unhealthy individuals, and they would tend to exercise less. We would, therefore, observe a negative correlation between exercise and mortality under either model, but, of course, the effects in the population of requiring everyone to take up exercise would be completely different: under model A mortality risk would decline; under model B it would be unchanged.

A statistical method for controlling for unobserved healthiness can be based on panel data: the individual effect (healthiness) can be accounted for in a number of ways, such as by taking deviations from individual means in a linear model or by modeling the distribution of the individual effect in a nonlinear model. For simplicity, take the case of a linear model. Then, model parameter estimates will depend on variation in the time path of exercise at the individual level and any associated mortality events. Under model A, those individuals who decrease their exercise level will eventually have higher mortality rates, whereas individuals who do not decrease their exercise levels will have unchanging mortality rates. If healthiness is static, under model B any variation over time in exercise would not be associated with any variation in mortality rates. The two models predict different relationships between the time paths of exercise and mortality. That difference can be used to identify the true model, which will lead to the correct prediction about the effects of changing exercise on mortality risk.

However, the point of the model of Manton et al. is that risk factors evolve, and it surely follows that healthiness also changes over time. Under model A,

mortality risk would vary as healthiness varies, but, on average, individuals who choose declining levels of exercise would have increasing mortality rates. Under model B, individuals whose healthiness fell would reduce exercise, and their mortality rates would also rise. Thus, the empirical outcomes would be the same under either model A or B: unchanging exercise is associated with unchanging mortality rates; falling exercise is associated with increasing mortality rates. Without further specification of what would amount to a structural model, no empirical methods could separate the models, and so we should not have much confidence that changing exercise in the population will have an effect on mortality risk.

This example is a gross simplification of the model of Manton et al., but it does, I believe, illustrate why I have reservations about their assessment of the effects of altering risk factors. I do not mean to be overly critical because this is the same kind of empirical problem economists face with nonexperimental data, and everyone knows how difficult it is to find convincing results.

Conclusion

The paper by Manton, Singer, and Stallard, the paper by Vaupel and Lundström, and the population forecasts by the Social Security Administration should lead practically anyone to the conclusion that the actual future elderly population could well be very different from the forecast population. The difference is large as measured by the variation in the impact on the Social Security retirement system. Although I have no quantitative measures, I am sure the effects on health-care expenditures and on the Medicare and Medicaid programs vary in a similar way. How policy should react to the uncertainty is not at all obvious, but because it will be practically catastrophic to the retirement and health-care financing systems should the actual population reach the upper levels of the forecasts, we should be thinking now of policies to cover those cases. Of particular importance is reducing the rate of growth in medical costs, because of the interaction of medical costs with the elderly population.[2]

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2. See my comment on Shoven, Topper, and Wise's chap. in this volume.