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Demographic Issues in Longevity Risk Analysis

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Abstract

Fundamental to the modeling of longevity risk is the specification of the assumptions used in demographic forecasting models that are designed to project past experience into future years, with or without modifications based on expert opinion about influential factors not represented in the historical data. Stochastic forecasts are required to explicitly quantify the uncertainty of forecasted cohort survival functions, including uncertainty due to process variance, parameter errors, and model misspecification errors. Current applications typically ignore the latter two sources although the potential impact of model misspecification errors is substantial. Such errors arise from a lack of understanding of the nature and causes of historical changes in longevity and the implications of these factors for the future. This article reviews the literature on the nature and causes of historical changes in longevity and recent efforts at deterministic and stochastic forecasting based on these data. The review reveals that plausible alternative sets of forecasting assumptions have been derived from the same sets of historical data, implying that further methodological development will be needed to integrate the various assumptions into a single coherent forecasting model. Illustrative calculations based on existing forecasts indicate that the ranges of uncertainty for older cohorts' survival functions will be at a manageable level. Uncertainty ranges for younger cohorts will be larger and the need for greater precision will likely motivate further model development.

Introduction

Longevity risk can be defined at individual and aggregate levels. At the individual level, longevity risk refers to the possibility of living longer than assumed in financial planning for the retirement of a single individual. At the aggregate level, longevity risk refers to the possibility of a higher average number of years of survival than assumed in designing a retirement security system for the aggregate. If the aggregate is a cohort of individuals who share a common year of birth, then, in theory, the longevity risk can be hedged with *longevity bonds* having coupons proportional to the number of cohort survivors at each anniversary after the issue date.

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Longevity bonds represent a potentially important new approach to the management of aggregate longevity risks. However, before such bonds or other similar instruments become practically feasible, a number of issues must be resolved.

This article focuses on demographic issues in longevity risk analysis relating to the measurement and modeling of survival and mortality. Actuarial (Cox, Lin, and Wang, 2006; Cairns, Blake, and Dowd, 2006), economic (Turner, 2006; Brown and Orszag, 2006), and financial issues (Blake et al., 2006; Milevsky, Promislow, and Young, 2006) are addressed elsewhere in this issue of the *Journal of Risk and Insurance*.

Among the demographic issues to be considered are the following:

1. Accurate measurements of the initial size and defining characteristics of each cohort, of decrements due to death, emigration, or other censoring events, and of increments due to immigration or other forms of cohort recruitment, are necessary to ensure that the longevity bond coupons accurately reflect the requirement that they be proportional to the number of survivors at each future date.
2. Accurate measurements of the inputs to the various models used for forecasting future survival and mortality are necessary for ensuring the validity of the outputs of those models.
3. New demographic models based on generalizations of the basic life table are needed for use in forecasting future survival and mortality. Factors to be considered in formulating such models include the following:
   - The impact of limits on the rate of increase in life expectancy and on the maximum value of life expectancy;
   - The changing nature of the mortality process;
   - The impact of individual-level risk covariates and their change over age, calendar time, and cohort;
   - The impact of technological innovation;
   - The stochasticity of life table parameters and its representation in forecasts.
4. Stochastic life table models with or without risk covariates produce forecasts of the distribution of the proportion of survivors at each future date, allowing the capital markets to set appropriate rates of investment returns for longevity bonds and similar instruments. The accuracy of such forecasts depends on the impact of estimation errors in the model's parameters and the risk that the selected model is not representative of future survival and mortality processes.

The article contains four sections:

- The Background section introduces basic concepts used by demographers in describing and measuring mortality.
- The Longevity Trends section discusses changes in longevity that have occurred in the past, the reasons that have been proposed to account for them, and the expected changes in longevity implied by the theoretical perspectives used by various researchers in explaining the historical trends.
- The Forecasting section discusses the use of deterministic and stochastic process models for forecasting the distribution of future survival outcomes for pricing models for longevity bonds for a set of closed cohorts.

- The Conclusion section finds that, although the necessary demographic data and mortality models currently exist, further methodological development is needed to improve the accuracy of the pricing models for longevity bonds and similar instruments that may be introduced into the capital markets.

BACKGROUND

Demography is the science of human populations. Its focus includes all aspects of the size, geographic distribution, and composition of human populations and changes in these characteristics over time. Composition includes sex, age, race/ethnicity, social, economic, and other relevant characteristics of the population. Modes of change include births, deaths, migration, marriage/divorce, and other relevant transitions. Sources of data for studying these changes include government-sponsored vital statistics systems and special surveys (see Shryock, Siegel, and Associates, 1971).

Two aspects of longevity bonds could benefit from consideration of the experience of demographers in measuring and modeling mortality.

First, the expectations of the capital markets are that the data underlying the cash flows of the longevity bonds will be accurate, valid, and reliable measurements of the cohort survival processes described in the bond prospectuses. Demographers would generally agree that the minimal requirements for meeting these expectations are as follows:

- The defining characteristics of each cohort must be clearly stated in a form that can be implemented consistently over time and verified by audit.
- The initial size of each cohort must be accurately assessed and recorded.
- Decrements to each cohort due to death, emigration, or other censoring events must be accurately and completely recorded in a timely manner that allows for accurate, periodic updating of the cohort survival function.
- In the case of a closed cohort (i.e., the cohort's membership is fixed at some date and age), the processes needed to meet these requirements are routinely implemented by life insurance and annuity carriers. Similar processes would be needed for longevity bonds, including incentives for reporting each death and continuous monitoring of vital statistics death reports.
- In the case of an open cohort, which is typical of national vital statistics data systems, a range of difficulties arise beyond those associated with the closed cohort processes familiar to life insurance and annuity carriers. For example, increments due to immigration or other forms of cohort recruitment must be accurately and completely recorded and the impact of such increments must be accounted for in the calculation of the cohort survival function. Survival functions for such cohorts can be calculated using the *period life table model* developed by demographers (Shryock, Siegel, and Associates, 1971). In this case, the development of the observed survival function requires a model which is distinct from the models used to predict the survival function at future times.

Based on the above considerations, the choice between open and closed cohort membership would be a fundamental design choice in the development of a longevity bond. Depending on the nature of the issuer, the administrative systems available to that issuer, and the confidence of the capital markets in those systems, one could make

a case for or against one or the other form of cohort definition. From a demographic perspective, both forms could be made to work.

Second, accurate measurement of the inputs to the various models used for forecasting future survival and mortality is necessary for ensuring the validity of the outputs of those models.

The details of these inputs depend on the specifications of the forecasting models. Typical inputs would include mortality counts and exposed population sizes by combinations of sex, age, race/ethnicity, social, economic, and other relevant characteristics of the population. Additional inputs could include stratifications of the mortality counts by cause of death and other characteristics not explicitly used to stratify the exposed population. Such data could be collected on an ongoing basis and may be supplemented by historical experience.

If the inputs are based on mortality experience data on select lives from life insurance and annuity carriers, then the models should be structured to account for the wear-off of selectivity and the credibility of the data provided by the life insurance and annuity carriers.

LONGEVITY TRENDS

This section discusses changes in longevity that occurred in the past and reasons proposed to account for them. The logic underlying this discussion is that understanding the reasons for historical changes is a necessary first step for formulating assumptions for forecasting future changes. Without an understanding of the reasons for prior changes, one can have little confidence in forecasts of future changes. For example, one could not assess whether the assumptions of any specific forecasting model were contradicted by past experience.

Study of historical changes in longevity requires a time frame appropriate to that of the forecasts needed for longevity bonds. Such a time frame could fall in the range of 50–75 years, assuming that 50 years is the youngest age at which a cohort longevity bond is issued. The lower bound of this range would follow this cohort to age 100, at which point the survival fraction could be 10 percent or less, whereas the upper bound would follow the cohort to age 125, an age that exceeds by 2.55 years the current world record for longevity (122.45 years; Allard, Lèbre, and Robine, 1998). Shorter time frames would be appropriate if the youngest issue age were greater than 50 years.

The appropriate time frame for understanding historical changes is less precise. On an evolutionary time scale, Judge and Carey (2000) estimated that *Homo habilis* had a life span of 52–56 years 2.0 million years ago, *Homo erectus* a life span of 60–63 years 1.7 million years ago, and *Homo sapiens* a life span of 72–91 years 100,000 years ago, compared to 122 years for *Homo sapiens* today. These authors concluded that postmenopausal survival among females is at least one to two million years old and is not a result of recent changes in human survival patterns. Postmenopausal survival is important to our analysis because almost all of the improvement in survival that would be relevant to longevity bonds will be postmenopausal. Given that natural selection has already put in place a mechanism for postmenopausal survival, one can

contemplate steps that could be taken to extend such survival with greater confidence than were such a mechanism not in place.

On the time scale of recorded history, Fogel and Costa (1997) considered the impact of the first and second agricultural revolutions (occurring about 9000 B.C.E. and 1700 C.E., respectively) on human longevity and proposed a new theory of *techniophysio evolution* based on "a synergism between technological and physiological improvements that has produced a form of human evolution that is biological but not genetic, rapid, culturally transmitted, and not necessarily stable" (p. 49). These authors used this new theory to explain changes in mortality over the past 300 years as functions of improved nutrition at all stages of development, increased body size, and greater efficiency and durability of vital organ systems.

On a shorter time scale, Oeppen and Vaupel (2002) analyzed life expectancies from period life tables in the various countries and found that the top-ranked values increased linearly from 1840 to 2000, at a rate of 2.43 years per decade (ypd) for females ($r^2 = 0.992$) and 2.22 ypd for males ($r^2 = 0.980$) with no indication of either an acceleration or deceleration in the rates of change. These authors argued for a continuation of these rates of increase in life expectancy forecasts for future years, hypothesizing that countries that lagged significantly behind the top ranked countries would catch up as the gaps between their performance and that of the top ranked countries are closed. These results support the analyses of Fogel and Costa (1997) and confirm the findings that significant changes in human longevity have taken place over the past one to two centuries.

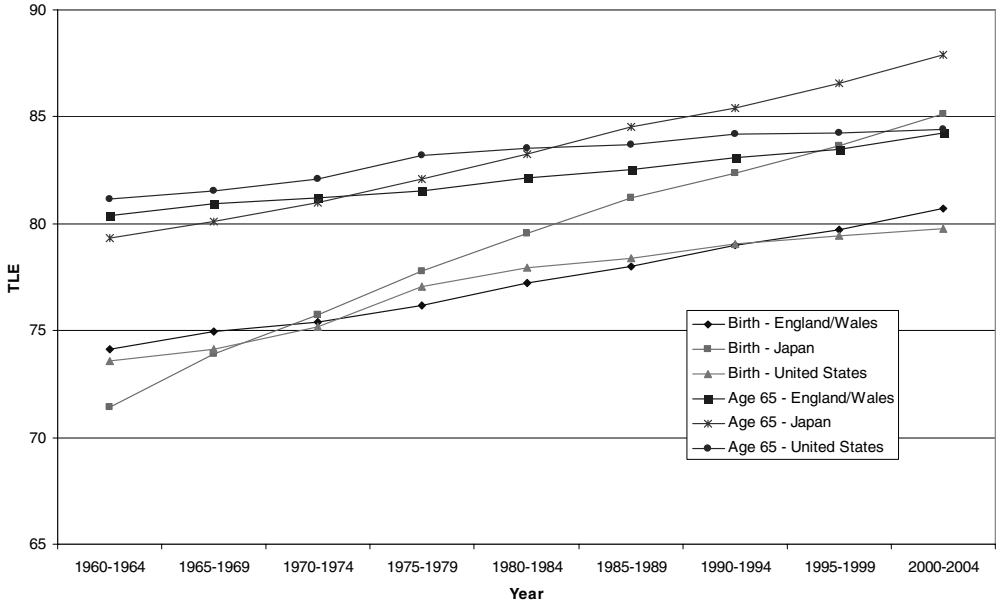
Fogel and Costa (1997) estimated that more than half of the reduction in mortality over the past two centuries was associated with increases in average body mass index (BMI) and maximum adult height. Not all increases are beneficial, however, as noted by Olshansky et al. (2005) who estimated that extant levels of obesity (which is associated with heart disease, cancer, stroke, diabetes, and hypertension) may result in a reduction in current life expectancy of 0.33–0.75 years over what would be the case in the absence of obesity, and who speculated that the life-shortening impact of obesity could increase to as much as 2–5 years in the coming decades. More importantly, these authors caution that the increases in obesity could halt or reverse the improvements in life expectancy by 2050.

Maximum Life Expectancy

Using a range of physiological data and concepts of biological warranty periods, Carnes, Olshansky, and Grahn (2003) argued that human life expectancy was limited to a range of about 85–95 years. Given that female life expectancy in Japan in 2004 was 85.6 years (Human Mortality Database, 2006), it is apparent that the lower end of Carnes' range is too low. Moreover, given the existence of such a limit, one would have expected to see a slowdown in the female improvements in Japan, the top-ranked gender in the top-ranked country since 1986 (Oeppen and Vaupel, 2002), especially if the limit were closer to age 85 than to age 95.

Figure 1 shows that this is not the case. Indeed, the rates of increase in total life expectancy at birth and at age 65 years were faster in Japan than in the United States and England & Wales, with the result that Japanese life expectancy at birth

FIGURE 1
Total Life Expectancy at Birth and Age 65 Years, Females, in England & Wales, Japan, and the United States, 1960–1964 to 2000–2004



Source: Human Mortality Database; 2000–2002 and 2000–2003 projected to 2000–2004.

exceeded United States and English & Welsh total life expectancies at age 65 in 2000–2004.

With female life expectancy in the United States in 2003 at 80.1 years (Hoyert et al., 2006), and Olshansky's warning that life expectancy may decrease in the United States in the next several decades, the range of about 85–95 years as a hypothesized upper limit appears less imminent. On the other hand, if Oeppen and Vaupel's finding that life expectancy is increasing 2.43 ypd continues, then the US female life expectancy will reach 85.0 years in 2023 and 94.8 years in 2063.

Olshansky et al. (2005) criticized the Social Security Administration (SSA) for assuming "large increases in survival past 65 years of age" (p. 1143). The Board of Trustees (2006) indicated that their best estimate of female life expectancy in 2080 was 85.1 years (2006, p. 82), 0.5 years less than that of Japanese females in 2004. This 76-year lag is significant because, in theory, the United States could match the extant achievements of Japan without new scientific or medical breakthroughs. The Trustees' best estimate of total life expectancy (TLE; $\text{TLE}_a = a + e_a$, where e_a is the residual life expectancy at age a) for females at age 65 in 2080 was 87.8 years, a value that was also 0.5 years less than that of Japanese females in 2004. Not surprisingly, Oeppen and Vaupel (2002) also criticized the SSA, but their concern was that the SSA had "recalcitrantly assumed that life expectancy will increase slowly and not much further"(p. 1031).

There is a substantial range of opinion among demographers concerning the reasons for changes in longevity and the implications of these trends for future improvements

in longevity. Olshansky and Vaupel and their respective associates represent the range of plausible opinions among professional demographers. It is not surprising then that the best-estimate forecasts of the Social Security Trustees fall in between these two extremes. The Trustees' 2080 low–high range for female life expectancy was 82.2–89.1 years, which was within the narrowest range of 80.1–94.9 years formed from Olshansky and Vaupel's various estimates. The Trustees' range was based on consideration of inputs from a number of professional demographers and actuaries (e.g., see Technical Panel on Assumptions and Methods, 1999, 2003).

The linear extrapolation proposed by Oeppen and Vaupel (2002) leads to female life expectancy projections for 2080 in the range 94.9–103.9 years, exceeding the upper bound of the Trustees' range for female life expectancy in 2080 by 5.8–14.8 years. Oeppen and Vaupel did not provide a basis for assessing the plausibility of their method of projection. This contrasts with Fries (1980, 1989) who hypothesized that the intersection of the linear extrapolations of TLEs at birth and age 65 years would yield an estimate of the maximum life expectancy under four assumptions:

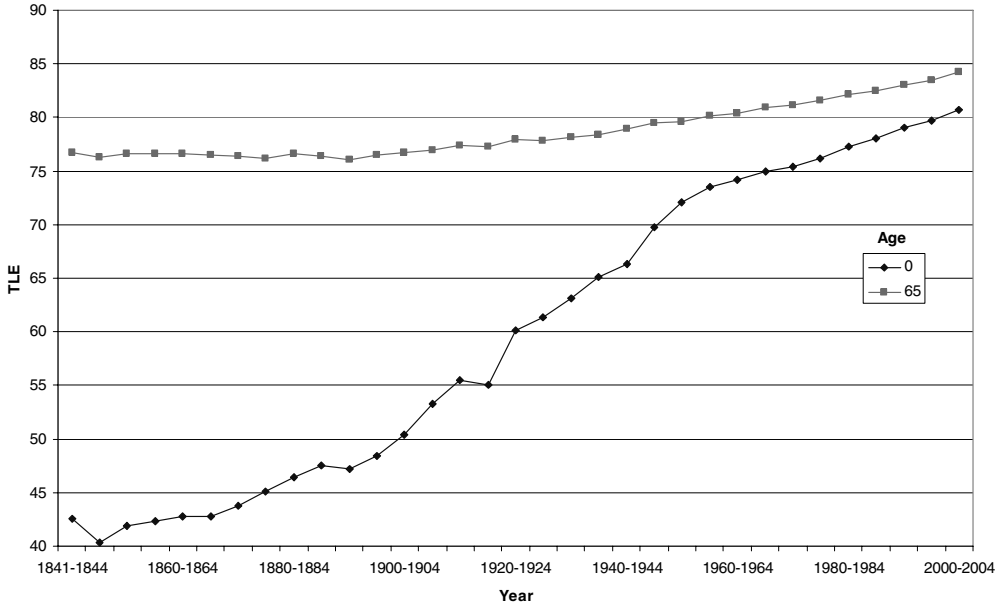
1. All deaths prior to age 65 are premature.
2. All premature deaths after age 65 will be eliminated in the same year as all premature deaths before age 65.
3. Linear extrapolation of life expectancy is reasonable.
4. Human life expectancy has a maximum value; once this maximum is reached, no further increases are possible.

Using this method, Fries (1980) produced an estimate of the maximum life expectancy of 85.0 years in 2045; Fries (1989) produced two estimates, 85.1 and 86.3 years, for an average of 85.7 years. Figure 1 displays the TLEs at birth and age 65 years for US females by quinquennium from 1960–1964 to 2000–2004. Using 1980–1984 to 2000–2004 as the calibration period for extrapolation at 0.93 and 0.42 ypd at birth and age 65 years, respectively, produces an estimate of the maximum life expectancy of 88.2 years in 2090–2094. Application of the same procedure to the corresponding data for English & Welsh females in Figure 1 with an extrapolation at 1.74 and 1.04 ypd at birth and age 65 years, respectively, produces an estimate of the maximum life expectancy of 89.4 years in 2050–2054.

Under this implementation of Fries' assumptions, the maximum life expectancy for English & Welsh and US females would be 88–89 years, a range consistent with Carnes, Olshansky, and Grahn's (2003) estimated range of 85–95 years, and 8–9 years above the 2003 levels. If this were in fact the true maximum, then it would greatly reduce the estimates of the initial risks faced by issuers of longevity bonds and the estimates of the continuing risks would gradually trend towards zero as the cohorts approached the maximum life expectancy, assuming that the cohort sizes were sufficiently large for the law of large numbers to apply to the survival fractions (Milevsky, Promislow, and Young, 2006).

Unfortunately, application of the same procedure to the corresponding data for Japanese females in Figure 1 with an extrapolation at 2.79 and 2.31 ypd at birth and age 65 years, respectively, produces an estimate of the maximum life expectancy of

FIGURE 2
Total Life Expectancy at Birth and Age 65 Years, Females, in England & Wales, 1841–1844 to 2000–2004



Source: Human Mortality Database; 2000–2003 projected to 2000–2004.

100.7 years in 2055–2059, which exceeds the upper bound of Carnes, Olshansky, and Grahn's (2003) estimated range by 5.7 years.

The Japanese data frustrate attempts to calculate the maximum life expectancy and indicate that such a maximum either does not exist or if it does exist is only minimally relevant within the 50–75 year time frame of the longevity bond forecasts for Japanese females, and likely totally irrelevant for English & Welsh and US females. In other words, if there is a maximum life expectancy, its value is not low enough to reduce the risks faced by issuers of longevity bonds over what would be the case in the absence of such a limit.

Epidemiologic Transition

Deeper insight into the types of changes that have occurred in the past can be gained by examination of Figure 2, which displays the TLEs at birth and age 65 years for English & Welsh females by quinquennium from 1841–1844 to 2000–2004, a period that overlaps that of Oeppen and Vaupel (2002). The TLEs at birth and age 65 years can be divided into three subperiods:

1. 1841–1844 to 1885–1889—with TLEs rising 1.11 ypd at birth and declining 0.08 ypd at age 65;
2. 1890–1894 to 1955–1959—with TLEs rising 4.06 ypd at birth and 0.63 ypd at age 65;

3. 1960–1964 to 2000–2004—with TLEs rising 1.64 ypd at birth and 0.96 ypd at age 65.

Tests of linearity for the TLEs at birth yielded $r^2 = 0.970$ for the entire period 1841–1844 to 2000–2004, $r^2 = 0.964$ for the period 1890–1894 to 2000–2004, and $r^2 = 0.993$ for the period 1960–1964 to 2000–2004. The corresponding r^2-values for the TLEs at age 65 were 0.860, 0.985, and 0.996 respectively.

The TLEs at birth and age 65 years for English & Welsh females for the period 1960–1964 to 2000–2004 were graphed in Figure 1. One can see that the plots are reasonably linear which is consistent with r^2-values above 0.990. In contrast, the plots in Figure 2 exhibit notable departures from linearity which might not have been expected given that the r^2-values for TLEs at birth were all above 0.960.

The patterns of change in the different subperiods in Figure 2 can be related to Omran's (1971) theory of the epidemiologic transition and its extensions by Olshansky and Ault (1986) and Rogers and Hackenberg (1987). The theory classifies changes in mortality and disease patterns as part of four successive stages of modernization:

1. An age of pestilence and famine—with life expectancies at birth fluctuating in the range of 20–40 years.
2. An age of receding pandemics—with life expectancies at birth steadily increasing in the range of 30–50 years.
3. An age of degenerative and man-made diseases—with life expectancies at birth continuing to increase in the range of 50–70 years.
4. An age of delayed degenerative diseases—with life expectancies at birth of 70 years and above with continuing increases.

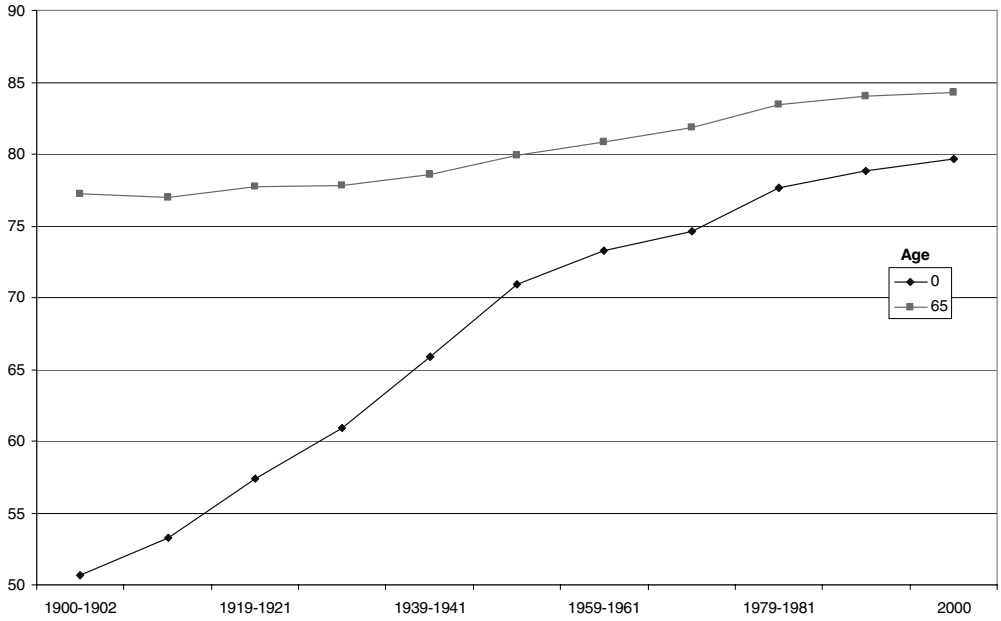
The timing of the stages of the epidemiologic transition depended on the demographic and socioeconomic conditions of the various countries. In England & Wales, the transition from stages 2 to 3 was completed by 1920, and the transition from stages 3 to 4 by 1960, which accounts for the increases seen in Figure 1. Similar transitions from stages 2 to 3 to 4 occurred in the United States, as shown in Figures 1 and 3.

Japan underwent a later but accelerated set of epidemiologic transitions, the end stages of which are seen in Figure 1 with Japan catching up with the United States and England & Wales by the 1970s. The continued rate of accelerated improvement in life expectancy in Japan beyond the 1980s, however, was not anticipated, and its impact is already large enough to undermine any reliance that one might have that an upper limit to life expectancy will serve to reduce the risk to issuers of longevity bonds.

Dynamic Equilibrium

Manton (1982) introduced the concept of a dynamic equilibrium between morbidity and mortality to explain the relationships between the declines in disease prevalence and the increases in life expectancy. On an intuitive level, the idea is that a population with improving health would more likely experience improving longevity than a population with static or declining health. Conversely, to explain why a population

FIGURE 3
Total Life Expectancy at Birth and Age 65 Years, Females, in the United States, 1900–1902 to 2000



Source: Anderson and Arias, 2003; Arias, 2006.

is experiencing improved longevity, one should look for related concurrent or prior changes in health.

The concept was proposed as an alternative to the conventional "pessimistic" view (Gruenberg, 1977; Kramer, 1980, 1983) that improvements in life expectancy would be accompanied by increased prevalence of major chronic diseases and disabilities, with an overall deterioration in average health. The concept shared some similarities but differed in a fundamental way from the "optimistic" view of Fries (1980, 1983) whose argument for the "compression of morbidity" was based on the hypothesis of a maximum life expectancy at about 85 years of age in combination with delays in the ages at which major chronic diseases became manifest. The concept of a dynamic equilibrium assumed no maximum life expectancy so it allowed for both compressions and expansions of specific measures of morbidity, but to a much lesser extent than expected by Kramer, Gruenberg, or Fries. Experience since those articles were published strongly supports the compression of morbidity hypothesis (Hubert et al., 2002; Stallard, Wolf, and Weltz, 2004), indicating that the hypothesis of a maximum life expectancy was unnecessary in Fries' (1980, 1983) original model.

To fully exploit the concept of dynamic equilibrium, one needs to provide an operational definition of "health" and specific procedures to measure it. In theory, if changes in health were more predictable than changes in mortality, or if changes in health preceded changes in mortality by significant numbers of years, one could use that information to improve forecasts of mortality by reducing the uncertainty of

future values and by anticipating bend points in future trajectories of mortality rates and life expectancies.

Motivation for this approach was provided by the "Barker hypothesis" (Barker, 1992; Hales and Barker, 1992) which states that diseases in late life (e.g., hypertension, stroke, heart disease, impaired glucose tolerance, and diabetes) are significantly influenced by developments very early in life including fetal growth rates, implying that measurable changes in health may precede observed changes in mortality by 80 years or more, a period longer than the 50–75 year time frame of the forecasts needed for longevity bonds.

Other influences during childhood development and early adult life could provide additional predictors with lagged effects within the 50–75 year time frame. Olshansky et al.'s (2005) analysis of obesity provides an example of such a predictor. These authors, however, did not address how changes in obesity would interact with changes in other synchronous and asynchronous health characteristics such as reductions in cigarette smoking and medical advances in the treatment of heart disease (Gregg et al., 2005). A complete model would account for all or a large fraction of salient interacting synchronous and asynchronous health characteristics tracked over the entire lifespan. A challenge for mortality forecasters involved in the development and pricing of longevity bonds will be to develop parsimonious models that adequately capture the behavior of the complete model using available data and methods.

Morbidity and Mortality

Comparison of trends in morbidity and mortality are best done using either age-specific or age-adjusted death rates.

Table 1 displays the age-adjusted central death rates at ages 65 years and older in the United States for the total population, and by sex, for 1900 and 1999 (top panel) and 1910 and 1990 (bottom panel). As expected, male mortality was higher than female mortality in all years and the relative excess was smallest (8.8 percent) in the earlier years. The rightmost column of the table displays the annual rates of change in the

TABLE 1
Age-Adjusted Central Death Rates at Ages 65 Years and Older (per 100,000) in the United States

	Year		Annual Rate
Gender	1900	1999	of Change
Male	10,612	6,154	−0.55%
Female	9,749	4,157	−0.86%
Total	10,079	4,898	−0.73%
	1910	1990	
Male	10,444	6,526	−0.59%
Female	9,606	4,055	−1.07%
Total	9,937	4,986	−0.86%

Source: Bell and Miller (2002, Table 1).

TABLE 2
Prevalence of Chronic Conditions Among Elderly Male Veterans Aged 65 Years and Older in the United States (%)

	Population and Year		
Condition	Union Army 1910	NHIS 1985–1988	Annual Rate of Change
Digestive (hernia/diarrhea)	84.0	8.0	−3.03%
Genito-urinary	27.3	8.9	−1.45%
Circulatory	90.1	40.0	−1.06%
CNS, endocrine, metabolic, blood disorders	24.2	12.6	−0.85%
Musculoskeletal	67.7	42.5	−0.61%
Respiratory	42.2	26.5	−0.61%
Cancer	2.2	9.2	1.89%

NHIS is the National Health Interview Survey.
Source: Fogel and Costa (1997, Table 3).

TABLE 3
Prevalence of Chronic Conditions Among Elderly Males Aged 60–74 Years in the United States (%)

	Population and Year		
Condition	Union Army 1910	NHANES 1988–1994	Annual Rate of Change
Heart murmur	39.2	3.8	−2.84%
Irregular pulse	42.0	8.5	−1.95%
Decreased breath or adventitious sounds	37.8	10.8	−1.53%
Joint pain/tenderness/swelling	55.0	35.2	−0.55%

NHANES is the National Health and Examination Survey.
Source: Costa (2000, Table 1).

age-adjusted central death rates, where "–" denotes a decline. The smallest annual rates of decline were 0.55–0.59 percent per year for males; the largest were 0.86–1.07 percent per year for females. The rate of decline in male mortality was, after rounding to 0.6 percent per year, identical to the overall rate of decline in morbidity for males in this period reported by Fogel and Costa (1997), using health data from Union Army veterans in the early years (which limits the study of long-term trends to males).

Table 2 displays the prevalence rates for a range of chronic medical conditions among elderly male veterans in 1910 and 1985–1988 along with the annual rates of change in these prevalence rates. All conditions declined at a rate of at least 0.61 percent per year except cancer which increased 1.89 percent per year. Table 3 displays the prevalence rates for four additional conditions in 1910 and 1988–1994, the one difference being that nonveteran males were included in the latter period. Costa (2000, p. 56) noted that this change was made to increase the sample size and did

TABLE 4
Prevalence of Functional Limitations Among Elderly Males in the United States (%)

Age	Condition	Population and Year: Union Army 1900/1910[a]	Population and Year: NHANES 1988–1994	Annual Rate of Change
50–64 in 1900 or 1988–1994				
	Paralysis	4.8	0.9	−1.82%
	Difficulty bending	39.0	7.5	−1.80%
	Deaf (either/both ears)	2.9	1.4	−0.80%
	Difficulty walking	20.9	10.4	−0.76%
	Blind (either/both eyes)	2.8	1.5	−0.68%
60–74 in 1910 or 1988–1994				
	Difficulty bending	49.7	16.1	−1.38%
	Difficulty walking	30.9	13.8	−0.99%
	Paralysis	6.0	2.7	−0.98%
	Deaf (either/both ears)	3.8	2.7	−0.42%
	Blind (either/both eyes)	3.8	3.1	−0.25%

[a]Excludes wounded veterans, POWs, and disability discharges.
NHANES is the National Health and Examination Survey.
Source: Costa (2002, Table 3).

not bias the results. All four conditions declined at a rate of at least 0.55 percent per year.

Table 4 presents similar statistics for a range of functional limitations among middle-aged and elderly males in 1900/1910 and 1988–1994. All but two of the ten conditions declined at a rate of at least 0.68 percent per year.

The results in Tables 2–4 can be summarized as follows. Except for cancer, chronic disease prevalence rates for older males declined at a rate of 0.6 percent per year or faster. Except for sensory losses, functional limitation prevalence rates for older males declined at a rate of 0.6 percent per year or faster. The rates of decline for chronic disease and functional limitation prevalence rates were generally faster than the 0.6 percent per year rate of decline in mortality for older males over the same period. These results are consistent with the hypothesis that the compression of morbidity occurred throughout the entire 20th century.

Table 5 displays the mortality rates for all causes of death by sex and age at ages 65 and older in the United States in 1960 and 2000. The top three causes of death were heart disease, cancer, and stroke. Declines in heart disease and stroke death rates were major components of the mortality declines during 1960–2000, whereas cancer death rates increased over the three age groups 12.5 percent, 26.7 percent, and 55.7 percent, respectively, for males and 18.5 percent, 14.5 percent, and 15.2 percent for females (NCHS, 2005, p. 199). Table 6 shows, however, that the cancer increases were not enough to offset the other improvements.

The male cancer death rates were even higher in 1990 than in 2000 for all three age groups in Tables 5 and 6, as well as for the age range 15–64 years, indicating that cancer

TABLE 5
Death Rates (per 100,000), United States, by Sex and Age

Sex and Age	Year		Annual Rate of Change
	1960	2000	
Males			
65–74	4,914	2,980	−1.24%
75–84	10,178	6,973	−0.94%
85+	21,186	17,501	−0.48%
Females			
65–74	2,872	1,921	−1.00%
75–84	7,633	4,815	−1.15%
85+	19,008	14,719	−0.64%

Source: NCHS (2003, Table 35).

TABLE 6
Death Rates (per 100,000) for Cancer, Heart Disease, and Stroke, United States, by Sex and Age

Sex and Age	Year				
	1960		2000		
	Death Rate	Percent of All Deaths	Death Rate	Percent of All Deaths	Annual Rate of Change in Death Rate
Males					
65–74	3,713	76%	2,045	69%	−1.48%
75–84	7,688	76%	4,500	65%	−1.33%
85+	15,173	72%	10,625	61%	−0.89%
Females					
65–74	2,237	78%	1,253	65%	−1.44%
75–84	5,948	78%	2,976	62%	−1.72%
85+	13,985	74%	8,809	60%	−1.15%

Source: NCHS (2003, Tables 36–38).

mortality has begun to decrease and likely will continue to do so as these cohorts grow older. A similar pattern occurred for females except that the downturn did not apply to the two oldest cohorts (i.e., those aged 75–84 and 85+) in 2000.

Table 7 displays residual life expectancies for US males and females at age 65 in 1984 and 1999, and the corresponding expected years of disability based on two alternative definitions of disability. The absolute number of years of disability declined 1.01–1.29 percent per year, consistent with the hypothesis of the compression of morbidity. Females, however, had more than double the expected years of disability of males.

Thus, the long-term trends in morbidity and mortality can be characterized by declines in overall mortality rates, declines in mortality rates for specific causes of death, declines in the prevalence of specific chronic medical conditions that cause functional

TABLE 7
Residual Life Expectancy, HIPAA ADL Expectancy, and LTC Institutional/ALF Expectancy (in Years at Age 65), United States, 1984 and 1999, by Sex

Sex and Characteristic	Year		Annual Rate of Change
	1984	1999	
Males			
Residual life expectancy	14.43	15.66	0.55%
HIPAA ADL expectancy	1.22	1.01	−1.29%
LTC Institutional/ALF expectancy	0.58	0.50	−1.01%
Females			
Residual life expectancy	18.62	19.03	0.14%
HIPAA ADL expectancy	2.41	2.06	−1.05%
LTC Institutional/ALF expectancy	1.56	1.32	−1.10%

HIPAA is Health Insurance Portability and Accountability Act.
ADL is personal assistance in two of six activities of daily living.
LTC is long-term care.
ALF is assisted living facility.
Source: Author's calculations based on National Long-Term Care Survey and life tables from Bell and Miller (2002).

limitations, disabilities, and death, and declines in the prevalence of functional limitations. The results in Tables 5 and 6 indicate that the long-term trends in the compression of morbidity applied to females as well as males, consistent with the short-term trends in Table 7 and in Hubert et al. (2002).

Multiple Causes of Death

Although the fact of death is unambiguous, attempts to assign the cause(s) to each death are less so. The assignment of cancer, heart disease, stroke, or other diseases as causes of death (e.g., see Table 6) is based on a computerized review of the sequence of diseases, injuries, or complications listed on the medical condition field of the certificate of death and selection of one of the listed conditions as the "underlying cause"; all other conditions are "associated causes." Stallard (2002) found that the average number of medical conditions reported at death for aged decedents of both sexes during 1980, 1990, and 1998 was in the narrow range 1.99–2.06, indicating that the greater expected number of years of disability for females was not associated with a greater number of medical conditions at death.

The occurrence of two or more conditions raises the possibility that the conditions are not independent. Stallard (2002) evaluated the ratios of the observed to expected joint occurrences of pairs of conditions selected from among the top fifteen underlying causes of death in 1998 and 1999. The results for 1998 are summarized in Table 8.

Under the assumption that medical conditions reported on the death certificate are randomly associated, the expected joint probability of each pair of conditions was computed as the product of their observed marginal proportions. Observed/expected

TABLE 8
Ratios of Observed to Expected Age-Standardized Joint Frequencies of Multiple Causes: Unisex Mortality, United States, 1998, Age 65+

		Multiple Cause														
No.	Multiple Cause	1 Heart Dis.	2 Malign. Neopl.	3 CBV Dis.	4 COP Dis.	5 Pneu. Influ.	6 Diabt. Mellit.	7 Suicide	8 Nephri. Nephro.	9 Chr. Liv. Cirrho.	10 Septicemia	11 Alzhm. Dis.	12 Atheroscler.	13 Hypertens	14 Aortic Aneur.	15 Resid. Dis.
1	Diseases of heart	–	0.50	0.84	**1.02**	0.75	**1.33**	0.12	**1.12**	0.67	0.71	0.68	**1.15**	**1.20**	0.91	0.89
2	Malignant neoplasms	0.50	–	0.31	0.69	0.59	0.48	0.17	0.53	0.44	0.58	0.30	0.29	0.58	0.25	0.72
3	Cerebrovascular diseases	0.84	0.31	–	0.54	0.94	**1.41**	0.06	0.64	0.30	0.76	0.68	**1.95**	**2.13**	0.51	0.97
4	Chr. Obstructive Pul. Dis.	**1.02**	0.69	0.54	–	**1.50**	0.77	0.19	0.71	0.61	0.66	0.50	0.79	1.00	0.96	**1.19**
5	Pneumonia and influenza	0.75	0.59	0.94	**1.50**	–	0.87	0.03	**1.08**	0.63	**2.03**	**1.41**	0.51	0.67	0.35	**1.18**
6	Diabetes mellitus	**1.33**	0.48	**1.41**	0.77	0.87	–	0.12	**1.79**	**1.08**	**1.21**	0.75	**1.72**	**2.57**	0.33	0.94
7	Suicide	0.12	0.17	0.06	0.19	0.03	0.12	–	0.05	0.11	0.03	0.12	0.11	0.16	0.05	**2.18**
8	Nephritis/Nephrosis	**1.12**	.053	0.64	0.71	**1.08**	**1.79**	0.05	–	**1.70**	**2.20**	0.50	**1.35**	0.28	**1.14**	**1.15**
9	Chron. Liver Dis./ Cirrhosis	0.67	0.44	0.30	0.61	0.63	**1.08**	0.11	**1.70**	–	**1.29**	0.18	0.47	0.60	0.39	**1.58**
10	Septicemia	0.71	0.58	0.76	0.66	**2.03**	**1.21**	0.03	**2.20**	**1.29**	–	0.83	0.60	0.64	0.64	**1.52**
11	Alzheimer's disease	0.68	0.30	0.68	0.50	**1.41**	0.75	0.12	0.50	0.18	0.83	–	0.80	0.82	0.27	0.94
12	Atherosclerosis	**1.15**	0.29	**1.95**	0.79	0.51	**1.72**	0.11	**1.35**	0.47	0.60	0.80	–	**1.62**	**1.11**	0.94
13	Hypertension	**1.20**	0.58	**2.13**	1.00	0.67	**2.57**	0.16	0.28	0.60	0.64	0.82	**1.62**	–	**1.87**	**1.06**
14	Aortic aneurysm	0.91	0.25	0.51	0.96	0.35	0.33	0.05	**1.14**	0.39	0.64	0.27	**1.11**	**1.87**	–	0.93
15	Residual causes	0.89	0.72	0.97	**1.19**	**1.18**	0.94	**2.18**	**1.15**	**1.58**	**1.52**	0.94	0.94	**1.06**	0.93	–

Source: Stallard (2002).

ratios greater than 1.0, shown in boldface in Table 8, imply positive dependence; conversely, observed/expected ratios less than 1.0 imply negative dependence. One can see that heart disease and stroke (cerebrovascular diseases) are positively associated with hypertension, atherosclerosis, and diabetes, but negatively associated with each other. Negative associations in this model may occur because of competing risk effects or because one disease is in fact protective against another. The finding that both positive and negative associations between pairs of diseases occur in death certificate data falsifies the assumption that the top ranked causes of death behave as described in the standard form of the independent competing risk model, implying that the causes of death should not be forecasted independently.

Actual Causes of Death

McGinnis and Foege (1993) introduced the concept of "actual causes of death" to refer to nongenetic modifiable lifestyle and risk factor behaviors such as smoking, poor nutrition/physical inactivity, alcohol use, microbial agents, toxic agents, motor vehicle accidents, firearms, sexual behaviors, and illicit drug use that were quantitatively associated with standard sets of underlying causes of death through published relative risk estimates. These authors estimated that 50 percent of all deaths in the United States in 1990 were attributable to these nine factors. In a follow-up study, Mokdad et al. (2004) found that 48 percent of all deaths in the United States in 2000 could be attributed to the same nine factors. These results were consistent with complementary findings from twin studies that about 50 percent of the variation in frailty (i.e., susceptibility to death), and 25 percent of the variation in observed age at death, was due to genetic factors (Iachine et al., 1998).

Mokdad et al. (2004) identified several limitations to their methodology, including the use of relative risk measures from independent published analyses, and the lack of information and controls for the effects of high blood pressure, high serum cholesterol, multiple comorbidities, and disability, all of which could impact mortality forecasts based on their methods.

Given the substantial documented declines in the sequelae of the various modifiable lifestyle and risk factor behaviors, it is instructive to consider the changes that have occurred in these risk factors since 1960 and the potential impact of these changes on mortality forecasting models.

Table 9 shows that cigarette smoking among males and females at all ages above 45 years declined 0.38–2.69 percent per year, depending on age and sex, with the largest rates of decline occurring for males. Table 10 shows that hypertension declined 0.27–0.45 percent per year, except for males aged 65–74 years, where the prevalence was virtually constant. Table 11 shows that high serum-cholesterol declined 1.85–2.55 percent per year.

Table 12 shows that obesity increased 1.34–1.39 percent per year for females and 2.88–3.48 percent per year for males. Although this change has been well documented, the impact of the increase in obesity was not enough to offset the favorable impact of the declines in cigarette smoking, hypertension, and serum cholesterol, and other risk factors (Flegal et al., 2005). Olshansky et al. (2005) caution that this may not be the case in the future.

TABLE 9
Current Cigarette Smoking (%) at Ages 45–64 and 65 Years and Older, United States, by Sex

Sex	Year		Annual Rate of Change
	1965	2003	
	Age 45–64		
Male	51.9	23.9	−2.02%
Female	32.0	20.2	−1.20%
	Age 65+		
Male	28.5	10.1	−2.69%
Female	9.6	8.3	−0.38%

Source: NCHS (2005, Table 63).

TABLE 10
Prevalence of Hypertension (%; SBP $\geq$ 140, DBP $\geq$ 90, or Medicated) at Ages 55–64 and 65–74 Years, United States, by Sex

Sex	Year		Annual Rate of Change
	1960–1962	1999–2000	
	Age 55–64		
Male	60.3	50.7	−0.45%
Female	66.4	57.9	−0.36%
	Age 65–74		
Male	68.8	68.3	−0.02%
Female	81.5	73.4	−0.27%

Source: NCHS (2003, Table 66).

Fundamental Causes of Death

Link and Phelan (1995) introduced the concept of "fundamental causes" to explain the associations of socioeconomic status (SES) and social support factors with a broad range of lethal diseases. The specific epidemiologic risk factors that serve as intervening risk factors between SES and disease have changed significantly over the past several decades, even as the risks of specific diseases also changed, leading to the hypothesis that those who have access to greater amounts of money, knowledge, power, prestige, and social support will continue to have lower risks of disease and mortality and their advantages with respect to multiple risk factors and multiple disease outcomes will be maintained even as the mixture and nature of the major lethal diseases continues to change over time.

Preston and Taubman (1994) analyzed socioeconomic differentials in health and mortality using income, occupation, and education as the primary measures of SES. They concluded that "the bulk of black-white differences in mortality and health status are explicable in terms of the unequal distribution of the groups on variables such as

TABLE 11
Prevalence of High Serum Cholesterol (%; SC $\geq$ 240 mg/dL) at Ages 55–64 and 65–74 Years, United States, by Sex

Sex	Year		Annual Rate of Change
	1960–1962	1999–2002	
	Age 55–64		
Male	41.6	19.9	−1.85%
Female	70.1	25.6	−2.52%
	Age 65–74		
Male	38.0	13.7	−2.55%
Female	68.5	32.3	−1.89%

Source: NCHS (2005, Table 70).

TABLE 12
Prevalence of Obesity (%; BMI $\geq$ 30 kg/m^2) at Ages 55–64 and 65–74 Years, United States, by Sex

Sex	Year		Annual Rate of Change
	1960–1962	1999–2002	
	Age 55–64		
Male	9.2	35.5	3.48%
Female	24.4	42.1	1.39%
	Age 65–74		
Male	10.4	31.9	2.88%
Female	23.2	39.3	1.34%

Source: NCHS (2005, Table 73).

education and income" (p. 313). They found that educational differentials in mortality rates were on the order of 2.1 to 1 for whites and 2.4 to 1 for blacks at ages 25–64 but narrowed above those ages, especially among those who had not completed college.

Costa (2000) estimated that 29 percent of the decline in chronic diseases during 1900–1990 was due to occupational shifts from manual to white collar jobs. Rogot et al. (1992, p. 336) found that relative risks of death were on the order of 1.48 to 1 at ages 25+ years and 1.35 to 1 at ages 65+ years for twelve major occupations, with professional, technical, and kindred workers having the lowest death rates.

Rogot et al. (1992, p. 206) found that relative risks of death were on the order of 1.79 to 1 at ages 25+ years and 1.44 to 1 at ages 65+ years for seven categories of family income. Preston and Taubman (1994) noted that part of these differentials may be due to reverse causation whereby persons with poor health are unable to work and, hence, have reduced income.

Lynch (2003) analyzed the effects of education on self-rated health and found that the effects increased over age and cohort, with the largest effects at the oldest ages

for the youngest cohorts. Interactions between age and cohort were not detectable in cross-sectional data for a single calendar year; joint analyses of multiple calendar years were required.

Lynch (2003) also considered the role of income and occupation as economic mediators of the impact of education on health. The increasing education of the younger cohorts may interact with the occupations that they enter to produce additional changes in mortality beyond those forecasted, assuming that education and occupation operate independently. Preston (1992) projected the changes in educational attainment of the oldest-old (age 85+) during the period 1990–2015 and found that the fraction of males and females who did not attend high school would decline from 58 percent to 20 percent and from 50 percent to 14 percent, respectively. Accounting for the impact of changes in educational attainment on future mortality rates will require a clearer understanding of these causal linkages.

Genetic Causes of Death

The link between genes and phenotypes is generally quantified by the heritability coefficient, defined as the percentage of the total phenotypic variance attributable to "additive" genetic variance (i.e., that portion of genetic variance acted upon by individual selection; Hartl and Clark, 1989, p. 464). Human longevity is such a phenotype: survival data from monozygotic and dizygotic twins indicate that heritability is about 50 percent for frailty (i.e., susceptibility to death) and 25 percent for observed ages at death (Iachine et al., 1998).

Human diseases are phenotypes that function as underlying or associated causes of death. Heritability estimates from Scandinavian twins are about 27 percent for female breast cancer, 35 percent for colon cancer, and 42 percent for prostate cancer (Willett, 2002). Heritability estimates for coronary heart disease among Swedish twins are about 57 percent for males and 38 percent for females (Zdrakovic et al., 2002).

Heritability estimates (including nonadditive effects) for disease risk factors, which can function as distal or intermediate causes of death, are about 85 percent for height, 62 percent for weight, 56 percent for systolic blood pressure, and 44 percent for diastolic blood pressure (Hartl and Clark, 1989, p. 471).

Heritability estimates for complex human behaviors, which can function as distal causes of death, are as high as 40–50 percent (Plomin, Owen, and McGuffin, 1994). Heritability estimates from the Minnesota Twin Study for a combination of five broad personality traits (extraversion, neuroticism, conscientiousness, agreeableness, and openness) were about 42 percent (Bouchard, 1994). Heritability estimates for general cognitive ability are about 20 percent in infancy, 40 percent in childhood, and 60 percent in adult life (Plomin, 1999).

Large gains in IQ in the United States over the period 1918–1995 reflect a significant impact of the environment on IQ and a reverse causation whereby IQ impacts a person's environment, which could account for the increase in heritability over age (Flynn, 1999; Dickens and Flynn, 2001). Turkenheimer et al. (2003) found that the heritability of IQ in young children varies with family SES, ranging from 0 percent in the most impoverished families to 60 percent in the most affluent families. A substantial part

of the temporal increase in IQ was attributed to increases in educational attainment (Uttl and Van Alstyne, 2003).

Thus, the proximate, intermediate, and distal causes of death identified above exhibit significant genetic dependence which accounts for a substantial part of the differences in mortality risks between individuals. Better understanding of these dependencies has the potential to lead to significant reductions in risk through a broad range of conventional and innovative interventions (e.g., see Schwartz, 1998; Arking, Novoseltsev, and Novoseltseva, 2004; Lucke and Hall, 2006).

FORECASTING

This section discusses the use of deterministic and stochastic process models for forecasting the distribution of future survival outcomes for pricing models for longevity bonds for a set of closed cohorts. Deterministic models have a long history of use in demographic forecasting where the uncertainties of the "best-estimate" forecasts are quantified using corresponding results from a range of alternative projections based on plausible alternative assumptions (e.g., see Alternatives I, II, and III in Board of Trustees, 2006). Stochastic process models have only recently been introduced in forms that are practical for use in demographic forecasting (Lee, 1998). An advantage of these models is that major components of the uncertainties of the forecasted quantities can be explicitly represented within the basic model structure. Additional consideration must be given to uncertainties relating to errors in parameter estimation and model misspecification, just as in the case of a deterministic model.

Parsimony and Credibility

There is a trade-off between the complexity of the selected forecasting model and the ability of that model to capture the impact of predictable trends and bend-points or reversals in proximate, intermediate, and distal causes of death. Examples of such predictable factors among the future elderly include increases in education and IQ, increases in average attained adult height, reversals in cancer death rates beginning in the 1990s, declines in cigarette smoking, and increases in obesity. Any forecasting model that omits (ignores) salient factors will be unable to represent the impact of changes in those factors in its outputs. This omission may or may not be significant to the utility of that model in forecasting for longevity-bond pricing. Assessment of the significance of the omission requires consideration of the differences in pricing between models with and without the specific factors.

Within a given model structure, additional trade-offs must be made with respect to the parameterization of the fitted data and projected values. Keyfitz (1984) considered such trade-offs in the context of selecting the number and form of parameters to forecast future values of life table survival functions, based on deterministic extrapolations of past trends in those parameters. Keyfitz recommended the use of the most parsimonious parameterization from among those that yielded forecasted survival functions that reflect credible patterns of age progressions in mortality rates.

The most parsimonious parameterization of human survival is the one-parameter form based on life expectancy at birth. Oeppen and Vaupel (2002) argued that deterministic forecasts of future life expectancy can be made by linear extrapolation of the 2.43 ypd rate of increase in female life expectancy observed during 1840–2000,

under the hypothesis that countries that lagged significantly behind the top ranked countries would catch up as the gaps between their performance and that of the top ranked countries are closed.

Presumably, the same argument could be made for forecasting residual life expectancy at age 65 years as a one-parameter summarization of the life table survival function above age 65 (or any other issue age for a longevity bond). However, as discussed above, the total life expectancies at birth and age 65 intersect (e.g., in the range 88–101 years for the three cases considered), which implies an impossible pattern of age progression in mortality rates in which no deaths can occur below age 65. Thus, forecasts based on life expectancy extrapolations violate Keyfitz's (1984) credibility criterion and, hence, are not recommended for use in longevity bond modeling.

Oeppen and Vaupel (2002) considered alternative models for forecasting based on extrapolations with constant and nearly equal percentage declines in age-specific death rates based on stochastic process models introduced by Lee and Carter (1992), as implemented in Tuljapurkar, Li, and Boe (2000). These models meet Keyfitz's (1984) dual criteria of parsimony and credibility without greatly distorting Oeppen and Vaupel's (2002) intent that forecasts of future life expectancy increase linearly. To make this point, Oeppen and Vaupel noted that:

> ... steady *rates* of change in mortality levels produce steady *absolute* increases in life expectancy. This relationship may underlie the linear trend of record life expectancy. (2002, Supplementary Material, p. 4)

Even in the case where the constant age-specific rates of decline are all equal, the relationship is still only an approximation (Vaupel, 1986): the error in the approximation is such that increases in total life expectancy are progressively slower at older ages and higher death rates. As death rates at older ages decline, the rate of increase in total life expectancy at these ages converges to the rate of increase in life expectancy at birth, implying that the intersection problems inherent in direct extrapolation of age-specific total life expectancies can be avoided by extrapolating age-specific death rates. In so doing, however, one should consider that the historical rates of decline in death rates tended to be lower at older ages than at younger ages (Bell and Miller, 2002; Lee, 2003). Historical rates of declines of 1–2 percent per year imply half-lives of projected death rates of 34–69 years; such death rates will not approach zero within the forecasting range of 50–75 years considered for longevity bonds.

Lee-Carter Model

The Lee-Carter model (Lee and Carter, 1992) for forecasting mortality rates has been used to demonstrate that changes in the logarithms of US national age-specific death rates during 1900–1989, and beyond, can be accurately represented as a random walk with drift, an especially simple type of time series structure. The basic equation of the model is:

$$\ln(m_{at}) = a_a + b_a k_t + \varepsilon_{at}, \tag{1}$$

where m_{at} is the death rate at age a in year t, k_t is the random walk with drift, a_a and b_a are parameters relating k_t to m_{at}, and ε_{at} represents the Gaussian error component of the model. The random walk with drift is modeled as:

$$k_t = k_{t-1} + u + \xi_t, \tag{2}$$

where u is the constant (the drift parameter) and ξ_t is the Gaussian stochastic innovation; ξ_t and ξ_s are independent for $s \neq t$.

The use of a one-dimensional random walk with drift meets Keyfitz's (1984) parsimony criterion, and, depending on the pattern of change over age in b_a, it may meet the credibility criterion for all or part of the forecasting range.

Two conclusions of Lee and Carter's (1992) analysis were notable:

1. The time series process could be described by a one-dimensional random walk that represented the changes in the entire vector of age-specific death rates.
2. The length of the mortality time series was not critical as long as it was more than about 10–20 years.

The second conclusion was amended by Lee and Miller (2001) who obtained better model fits by restricting the start of the calibration period to 1950, a date which roughly coincided with the bend-points in life expectancies shown in Figure 3. Booth, Maindonald, and Smith (2002) introduced procedures for selection of an optimal calibration period which identified the longest period for which the estimated "mortality index" parameter, $k_{t,}$, was linear. Because linear changes in k_t, produce approximately linear changes in age-specific life expectancies, these optimization procedures formalize Lee and Miller's (2001) *ad hoc* restrictions on the start date of the calibration period, yielding a start date that roughly matches the start date of the fourth stage of the epidemiological transition (see Figures 1–3).

Lee and Carter (1992, p. 669) analyzed the error structure of forecasts based on their two-component random-walk time-series model. For a fixed jump-off year t (i.e., the latest data year of the calibration period) and a forecast range s (i.e., the length of the forecast period) the model can be rewritten as:

$$\ln\bigl(m_{a(t+s)}\bigr) = a_a + b_a \left(k_t + su + \sum_{r=1}^{s} \xi_r \right) + \varepsilon_{a(t+s)}. \tag{3}$$

Because the product $b_a \cdot k_t$ is determined only up to a multiplicative constant, one can rescale k_t so that $u \equiv 1.0$, in which case b_a is the annual rate of change in m_{at}. The constraints used by Lee and Carter (1992, p. 661) set $\sum_a b_a = 1$ and $\sum_t k_t = 0$.

Lee and Carter (1992, p. 669) modeled the variance of the forecast, conditional on k_t, as the sum of five terms which can be written as follows:

$$\begin{aligned} \operatorname{var}\bigl(\ln\bigl(\hat{m}_{a(t+s)}\bigr)\bigr) = {} & \operatorname{var}(\hat{a}_a) + \hat{b}_a^2 \operatorname{var}\left(s\hat{u} + \sum_{r=1}^{s} \xi_r \right) + (\hat{k}_t + s\hat{u})^2 \operatorname{var}(\hat{b}_a) \\ & + \operatorname{var}(\hat{b}_a) \operatorname{var}\left(s\hat{u} + \sum_{r=1}^{s} \xi_r \right) + \operatorname{var}\bigl(\varepsilon_{a(t+s)}\bigr). \end{aligned} \tag{4}$$

The variance of the forecast of the random walk with drift, $su + \sum \xi_s$, appearing in the second and fourth terms is a sum of two components: the diffusion variance,

$\text{var}(\sum \xi_r)$, a linear function of the forecast length, s; and the drift-parameter variance, $\text{var}(s\hat{u})$, a quadratic function of the forecast length, s. Because a quadratic function of s must eventually exceed any fixed linear function of s, it follows that there will be a forecast length beyond which the drift-parameter variance exceeds the diffusion variance.

In addition to the variance of the random walk with drift, there is additional variance due to estimation of the parameters, a_a and b_a, and the error of prediction of m_{at}. Lee and Carter (1992, p. 669) calculated that the second term accounted for at least 91 percent of the variance in the forecasted logarithms of the death rates after 75 years, suggesting that this term provided a suitable approximation to the total variance. In applications where the calibration time series is shorter or where the mortality data are supplemented with data on morbidity and risk factor processes, one would need to retain all five terms.

Lee (2000) indicated that the variances of cohort life table functions based on the projected age-time-specific death rates would require consideration of the autocovariance structure of the errors in k_t. Such variances could also be computed based on microsimulation techniques applied to the parameterized random walk with drift.

Lee (2000) identified various modifications/extensions of the model that could be used in forecasting for longevity-bond applications, including:

- disaggregation by sex, geography, or other grouping criteria used to define the targeted cohorts;
- disaggregation by cause of death;
- addition of parameters for cohort effects;
- lower bounds on the age-specific death rates;
- modified procedures for the jump-off mortality index, k_t;
- constraints on the drift parameter based on "leader countries" (e.g., Japan).

Several extensions are in current use. For example, the SSA's stochastic forecasting model is disaggregated by cause of death, using expert opinion to inform the specification of the future values of the age-specific "ultimate" annual rates of decline in death rates (i.e., drift parameters), with trending toward those values over a 25-year phase-in period (Cheng et al., 2004). The use of expert opinion allows the model to reflect expected changes in historical trends for specific causes of death (e.g., cancer, diabetes, and respiratory disease), based on predictable trends and bend-points or reversals in proximate, intermediate, and distal causes of death.

How can predictable trends and bend-points be estimated? If the characteristics are fixed at the individual level (e.g., education and adult height), then they may already be measured in a form that can be projected by standard demographic methods. Another possibility is to model time series data on proximate, intermediate, and distal causes of death using the Lee-Carter model. Given the dependence of mortality on various factors, one might also consider extensions of the Lee-Carter model to include lagged correlated random walks.

The leader-country (also termed avant-garde or best practice) model was introduced by Wilmoth (1998a) to use the estimated rate of decline in Swedish death rates as a

way to *slow down* the forecasted rate of decline in Japanese death rates. This contrasts with Oeppen and Vaupel's (2002) recommendation, in effect, to use the estimated rate of decline in Japanese death rates as a way to *speed up* the forecasted rate of decline in US death rates.

Wilmoth (1998b, 2000) argued for linear extrapolation in mortality forecasts due to the long-term stability of the historical declines and the complexity of the causative mechanisms. Even if one accepts this argument, the differences in forecasts due to differences in calibration periods, in expectations regarding ultimate cause-specific rates of decline, in expectations for the current leader country, etc., can be viewed as model misspecification errors. If a compelling case cannot be made for selection or rejection of one or more of the various forecasts, then all of the models may be retained and the uncertainty of the composite forecast calculated, by microsimulation if probabilities are provided for the various forecasting models or model assumptions using, for example, elicitation techniques to obtain such probabilities from panels of experts (see Lee (1998) and Lutz, Sanderson, and Scherbov (1998) for discussion and caveats).

The differences can be large. For example, Horiuchi (2000) compared official "medium" variant forecasts for the G7 countries for 2050 with corresponding central (50^{th} percentile) forecasts made by Tuljapurkar, Li, and Boe (2000) based on the Lee-Carter model and found gaps of 1.3–8.0 years (United Kingdom–Japan) in life expectancy at birth. Indeed, the official 1997 Japanese forecast for 2020 was 82.1 years, a value that was exceeded in 2004 (82.4 years; Human Mortality Database, 2006). Given the 1.0 year increase in Japanese life expectancy during 2000–2004, the official 1997 Japanese forecast for 2050 (83.0 years) may be exceeded as early as 2007.

The gap in life expectancy at birth in the US for 2050 was 2.5 years, exactly 50 percent of the official range of 5.0 years (Tuljapurkar, Li, and Boe, 2000), implying that the high official projection matched the central projection based on the Lee-Carter extrapolation. The 95^{th} percentile of the Lee-Carter extrapolation was 4.7 years above the central official projection. Additional sources of error not included in the Lee-Carter variances could increase this gap further.

Fortunately, the above comparisons are not directly applicable to longevity bonds. The relevant comparisons are those for a cohort, not those based on some future period (calendar-year) life table. Cohort life tables prepared by the Board of Trustees (2006) indicated that the high-low gap in projected US cohort life expectancy at age 65 in 2005 was 1.3 years for females and 1.1 years for males. The corresponding high/low ratios of 1.068 and 1.067, respectively, are upper bounds to the ratios of the present values in 2005 of the coupons associated with the sex-specific pairs of cohort survival functions. Given that adverse deviations of about 6–7 percent over expected experience could significantly erode pricing margins, these comparisons indicate that uncertainty in cohort survival is material, but not unmanageable. Larger deviations could occur with younger cohorts and with deferral periods for older cohorts.

Risk-Factor Models

Wilmoth's (1998b, 2000) argument for linear extrapolation of age-specific death rates was based on the long-term stability of the historical declines and the complexity of

the causative mechanisms. However, the use of expert opinion in official government forecasts (e.g., Bell and Miller, 2002) and the leader-country concepts advocated by Wilmoth (1998a) and Oeppen and Vaupel (2002) suggest that linear extrapolation based solely on the long-term stability of the historical declines is not that compelling. Lee (1998, 2003) indicated that such extrapolations be viewed as baseline forecasts with consideration given to alternatives, including the leader-country concept.

Tuljapurkar and Boe (1998) observed that extrapolation methods are incapable of dealing with the complexity of the causative mechanisms: new models are required to deal effectively with the interrelationships of morbidity, disability, and mortality, and antecedent risk factors. Extensions of the risk-factor model proposed by Woodbury and Manton (1977) could exploit the expanding scope of knowledge in the fields of demography, epidemiology, public health, biology, and health-related subfields of the social sciences, some of which was reviewed in the previous section. Tuljapurkar and Boe (1998) opined that the current development of these models was "probably at too early a stage for direct use in forecasts of mortality, but they can provide useful perspectives on the assumptions in mortality forecasts" (p. 41).

The following are the main properties of the Woodbury-Manton model, as extended by Yashin, Manton, and Stallard (1989):

1. The level of description is changed from the population to the individual, because death occurs to individuals, not populations.
2. The model also applies to other "end-point" events including death due to specific causes and the onset of specific diseases or disease events (e.g., heart attack or stroke).
3. The risk of death (i.e., mortality hazard rate) is represented as a function of measurable fixed and time-varying covariates (risk factors), because individual differences in one or more covariate values are associated with significant differences in the risk of death.
4. Age-specific cohort death rates are expressed as averages of the individual risks of death for cohort members who survive to each age.
5. Changes in the distributions of the covariates produce changes in the individual risks of death and, hence, in the cohort death rates.
6. Additional changes in the cohort death rates can occur due to changes in the mortality hazard rate functions which relate the risk of death to the measurable covariates.
7. Changes in the distributions of the covariates can occur between cohorts reaching a common issue age due to the impact of previous selective survival, and within cohorts due to changes in the interrelationships between covariates.
8. Additional changes in the distributions of the covariates and the mortality hazard rate functions can occur due to population-wide changes in the environment.
9. The model yields forecasts of the means and variances of the cohort survival functions that take into account the effects of all measurable covariates at the individual and population-wide levels.
10. The model structure provides an array of options for including expert opinion on the rates of change of both individual and population-wide parameters.

It is instructive to consider the basic structure of the model and identify some existing applications. New applications and improvements will be facilitated by the continuing collection of the necessary longitudinal datasets and motivated by the increasing need for more accurate forecasts and more reliable quantification of the uncertainties of such forecasts as longevity bonds and other instruments are introduced to the capital markets.

Woodbury and Manton (1977) specified a random-walk model using just two assumptions:

$$d\,x_{ia} = u(x_{ia})\,da + d\xi(x_{ia}) \tag{5}$$

$$d\,S(x_{ia}) = -\mu(x_{ia})S(x_{ia})\,da\,, \tag{6}$$

where

- x_{ia} is the covariate vector for individual i at age a;
- $u(x_{ia})$ is a constant vector (a vector of drift parameters);
- $d\xi(x_{ia})$ is a Gaussian stochastic (random-walk) innovation vector—$d\xi(x_{ia})$ and $d\xi(x_{i(a+s)})$ are independent for $s \neq 0$;
- $\mu(x_{ia})$ is the mortality hazard rate function (note the distinction between μ and u);
- $S(x_{ia})$ is the marginal survival function for individual i at age a with respect to the distribution of the paths (trajectories), denoted $X_i \equiv X^a_{ia_0}$, of the covariate process $\{x_{i(a_0+s)}\}$ joining x_{ia_0} and x_{ia} from age a_0 to age a; hence, $S(x_{ia}) = E(S(X^a_{ia_0}) \mid X^a_{ia} = x_{ia})$, where X^a_{ia} is the endpoint of the path at age a.

Conditional on the observed initial values, x_{ia_0}, at age a_0, the random walk generates an unnormalized distribution of x_{ia}, with density function denoted $f(x_{ia})$, for all $a > a_0$ which is governed by a Fokker-Planck equation with one additional term for mortality:

$$\begin{aligned}\frac{\partial f(x_{ia})}{\partial a} &= -\mu(x_{ia})\,f(x_{ia}) - \sum_j u_j(x_{ia})\frac{\partial f(x_{ia})}{\partial x_{ia,j}} \\ &\quad - f(x_{ia})\sum_j \frac{\partial u_j(x_{ia})}{\partial x_{ia,j}} + \frac{1}{2}\sum_j\sum_k \sigma_{jk}(x_{ia})\frac{\partial^2 f(x_{ia})}{\partial x_{ia,j}\,\partial x_{ia,k}},\end{aligned} \tag{7}$$

where $\sigma_{jk}(x_{ia})$ is the j, k-element of the diffusion covariance matrix generated from $d\xi(x_{ia})$.

The survival function for individual i is the integral of the unnormalized density:

$$S_{ia} = \int \cdots \int f(x_{ia})\,d\,x_{ia}\,, \text{ with } S_{ia_0} = 1. \tag{8}$$

The variance of the survival function for individual i is defined as:

$$\operatorname{var}\big(S\big(X^a_{ia_0}\big)\big) = E\big(S^2\big(X^a_{ia_0}\big)\big) - S^2_{ia}, \tag{9}$$

which can be evaluated using the methods in Yashin, Manton, and Stallard (1989, pp. 132–133).

The survival function for a cohort of size N is the average of the N individual survival functions:

$$S_a = \sum_{i=1}^{N} S_{ia}/N. \tag{10}$$

This expression allows for heterogeneity among individuals in each cohort to be represented by an initial distribution of x_{ia_0}. In the case of a closed cohort with individual underwriting, the initial distribution of x_{ia_0} might be the only information available to construct a forecast, in which case the model parameters would have to be estimated from data on other similar cohorts.

The variance of the survival function for a randomly chosen individual in the cohort is a marginal variance defined as:

$$\operatorname{var}\left(S\left(X^a_{a_0}\right)\right) = \operatorname{var}(S_{ia}) + \sum_{i=1}^{N} \operatorname{var}\left(S\left(X^a_{ia_0}\right)\right)/N, \tag{11}$$

which is the sum of the between-individual variance and the average of the within-individual variances, where the between-individual variance is defined as:

$$\operatorname{var}(S_{ia}) = E\left(S^2_{ia}\right) - S^2_a, \tag{12}$$

and the within-individual variance is defined in Equation (9). The marginal variance, $\operatorname{var}(S(X^a_{a_0}))$, is additive over individuals and hence its impact on the uncertainty of the forecasted cohort survival function will converge to zero at a rate of $1/N$ as the cohort size N increases.

Yashin, Manton, and Stallard (1989) extended the Woodbury-Manton model to reflect more realistic conditions in which dependencies occur between individual survival functions due to population-wide changes in the environment (e.g., medical progress, scientific breakthroughs, pharmaceutical innovations, public health initiatives, etc.). These common environmental changes were allowed to differentially impact each individual through interactions with his or her individually-unique covariate values, thereby increasing the variances of the cohort survival functions in such a way that they would not tend to zero as the cohort size increases but instead would converge to positive lower-bound values that would be comparable to the variance estimates for the same cohort obtained from the Lee-Carter model.

Yashin, Manton, and Stallard (1989, pp. 134–135) presented procedures for computing the variances of these extended survival functions. The logic of the extension can be described by considering a common exogeneous vector process, $\{y_t \equiv y_{a_0+s}\}$, with path denoted $Y \equiv Y^a_{a_0}$, which is observable at all times up to the time the cohort reaches age a_0 and can be forecasted at least to the time the cohort reaches age a. The time-specific values of $Y^a_{a_0}$ are appended to each individual covariate path $X^a_{ia_0}$ (or vector $x_{i(a_0+s)}$) joining x_{ia_0} and x_{ia} from age a_0 to age a by matching external time, t, to cohort age, $a_0 + s$.

The original Woodbury-Manton formulas were modified by introducing Y as a conditioning covariate process, i.e., $S_a \leftarrow S_a(Y)$, etc. With these changes, the survival

function for a cohort of size N is the expected value with respect to Y of the modified form of Equation (10):

$$\bar{S}_a = E\left(S_a\left(Y^a_{a_0}\right)\right). \tag{13}$$

The variance component attributable to the exogeneous vector process is:

$$\mathrm{var}\left(S_a\left(Y^a_{a_0}\right)\right) = E\left(S^2_a\left(Y^a_{a_0}\right)\right) - \bar{S}^2_a, \tag{14}$$

which is the desired positive lower-bound of the variance of the forecasted cohort survival function. As with other forecasting models considered herein, the forecast of Y can be structured to incorporate as much or as little expert opinion as deemed appropriate.

The conceptual issues addressed in Yashin, Manton, and Stallard's (1989) extension are analogous to those considered by Milevsky, Promislow, and Young (2006).

Application of the basic Woodbury-Manton model to forecasting the size, health status, and survival of the US population was illustrated in Manton, Stallard, and Singer (1992). This application focused on the impact of hypothetical interventions in which all persons within a cohort had their morality hazard rates reduced to the lowest age-specific values for individuals within that cohort. The interventions were imposed directly on individual risk factor values, taking account of risk factor interactions, with the mortality reductions obtained as a by-product. Such calculations could be used to inform expert opinion with respect to the specification of best-estimate forecasts under this model, or to better estimate trends and bend-points for specific causes of death in aggregate models such as the SSA's implementation of the Lee-Carter model.

A more recent application used the model to assess the potential impact on life expectancy of bone-marrow derived progenitor-cell therapy for atherosclerosis. Under the conditions assumed for the efficacy of the therapy, males at age 30 would gain 5.9 years; females 3.7 years (Kravchenko et al., 2005).

Other methods for forecasting future survival as a function of risk factors based on inputs from individual-level longitudinal datasets have been implemented using microsimulation models (e.g., Goldman et al., 2005) and Grade of Membership techniques (e.g., Stallard, 2005).

CONCLUSION

Further methodological development is needed in the area of forecasting cohort survival functions and their variances to improve the accuracy of the pricing models for longevity bonds and similar instruments that may be introduced into the capital markets. The necessary demographic data and mortality models currently exist for such development. The illustrative calculations indicated that the likely ranges of uncertainty produced from such models will be large enough to warrant serious attention, but not large enough to produce unacceptably high longevity-risk loadings. The uncertainty in cohort survival functions for longevity bonds issued for older cohorts in the current year will be substantially less than the uncertainty for younger cohorts, with or without a deferral period.

An appropriate starting point for these efforts would be to refine the Lee-Carter model to explicitly represent all sources of error in forecasts of cohort survival functions, including process variance, parameter errors, and model misspecification errors.

One major challenge will be the elicitation and justification of expert opinion in formulating alternative assumptions about future trends in total and cause-specific mortality, and the corresponding probability distributions needed for modeling the uncertainties in those trends. One approach could involve risk-factor models that were structured to provide in-depth analysis of the assumptions underlying the various opinions of the experts and feedback to the experts on the future implications of those assumptions.

Further development of risk-factor models as practical supplements or alternatives to the Lee-Carter model and its extensions will be motivated by the need for greater precision in the forecasts of the means and the variances of the cohort survival functions due to: (1) the ability of these models to reflect the impact on future mortality of changes in the distributions of risk factors that have already occurred; and (2) the richer set of parameters available to experts attempting to opine on the impact of biomedical innovations targeted directly to disease risk factors (i.e., prevention), rather than to disease treatments or cause-specific mortality reductions.

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