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# 2 Methods for Projecting the Future Size and Health Status of the U.S. Elderly Population

Kenneth G. Manton, Eric Stallard, and Burton H. Singer

## 2.1 Introduction

Reform of the U.S. health-care system has focused on payment systems and insurance for the elderly, a high consumption group, and for 36 million uninsured and 60 million underinsured, younger U.S. residents. For these efforts, forecasts of the consequences of public health programs are needed, as well as of the effects of population risk factor trends and diagnostic and treatment innovation (Blackburn 1989). Because actuarial projections (e.g., Spencer 1989; Wade 1987) do not use information on health change prior to death, their use in designing service delivery, acute and long-term care (LTC) insurance, and reimbursement systems is limited, as is their ability to anticipate "turning" points in population growth and health (Myers 1981). Health forecasts are also needed to design interfaces for private insurance and Medicare and Medicaid coverage and for long-term market planning by drug and medical equipment manufacturers.

Changes in the size and health of the U.S. adult population are determined by chronic disease morbidity and mortality. Lifestyle and behavior (e.g., physical activity, smoking, and diet) influence the natural history of many chronic diseases. Improvements in the population distribution of risk factors and treatment have reduced U.S. mortality of those above age 65 (Blackburn 1989). After plateauing from 1982 to 1988, mortality of persons aged 85+ declined 8.6 percent from 1989 to 1991 (National Center for Health Statistics [NCHS]

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1992). To anticipate changes, health and mortality time-series data must be used. In this chapter we (i) introduce integrated models of risk factor dynamics and mortality processes, calibrated from longitudinal data, to forecast preventive and curative intervention effects; (ii) compare actuarial forecasts with those based on multivariate stochastic processes; and (iii) introduce models integrating disability dynamics with mortality processes, as a step toward integrating the dynamics of multiple biological levels (Lipsitz and Goldberger 1992).

In section 2.2, we review (2.2.1) the rationale of model specifications and introduce (2.2.2), time-inhomogeneous, multidimensional physiological variable processes. The dependency of mortality on the diffusion processes is defined in subsection 2.2.3 as a quadratic function of physiological variables multiplied by an exponential term representing "senescence." Dependent competing risks for multiple-cause mortality are discussed in subsection 2.2.4, where dependence is represented by risk factor trajectories generated by the diffusion process. In subsection 2.2.5 we introduce Grade of Membership (GoM) concepts to identify profiles of disabilities as vertices of a unit simplex within which individual disability dynamics operate as bounded diffusion processes. Positions in the simplex are defined by "scores" (i.e., coordinates in the convex space) representing the "degree of similarity" of an individual's traits to each "vertex" (i.e., profile of disabilities). Finally, we present discrete time approximations in subsection 2.2.6 for estimation and forecasting.

Section 2.3 presents projections based on scenarios about health interventions. We then discuss active life expectancy (ALE) projections and exogenous economic and social interventions. In section 2.4, we briefly discuss research needed to extend and refine models.

## 2.2 Methodology

### 2.2.1 Overview

We model mortality as influenced by the temporal dynamics of physiological (or, more generally, "state") variables. The mortality rate is expressed as a product of a quadratic function of measured physiological variables and an exponential function of age. The quadratic implies that there is an increasing risk of death with the movement of one or more physiological variables from an optimum homeostatic value (the minimum point of the function). The restriction to quadratic—as opposed to more complex—surfaces recognizes that data will seldom be sufficient to statistically discriminate between quadratic and higher-order polynomial surfaces. The exponential term, $\exp(\theta\ \text{age})$, is interpreted as the contribution to mortality of "senescence." By "senescence" we mean the age-specific average effect of currently "unknown" factors on mortality; senescence does not include any of the measured variables in the quadratic hazard. Although senescence is discussed in most theories of aging

(e.g., Medvedev 1990) as a decline in one or more biological functions, it has not been mathematically rigorously defined.

Evolution of physiological (state) variables in the quadratic hazard is assumed to be governed by stochastic differential equations with linear drift, i.e., dynamics are "Markovian." Although state variable processes may exhibit non-Markovian dependence, we assume that they can always be respecified to represent cumulative experience to approximate a Markovian process.

Forecasts using the stochastic differential equations focus on

1. The mean vector and covariance matrix of state variables at times/ages beyond the limits of data used to estimate parameters. Forecasts can be based on functional extrapolation assuming parameters do not change. Alternatively, parameters can be altered to represent scenarios about different interventions.

2. Life expectancy at birth, and specific ages, as measures of the effect of interventions on state dynamics and mortality.

3. Life tables for a range of interventions.

Total mortality is represented as a sum of "crude" cause-specific mortality rates, each operating in the presence of (i.e., competing with) all other causes (Yashin, Manton, and Stallard 1986). The mortality rate for a cause is correlated with rates for other causes through a vector of common physiological processes which represent the dependence of risks. Computational details are in subsection 2.2.4.

Mortality can also be modeled as a function of disability—both physical and cognitive—whose excursion from levels of performance associated with the lowest mortality risk are associated with the underlying state processes *causing* disability (see Manton, Stallard, and Singer 1992a). We use Grade of Membership (GoM) concepts to construct profiles of disabilities whose co-occurrence is biologically plausible. The profiles define fuzzy partitions using individual scores to represent the degree of similarity to each profile. The disability dynamics are modeled by a stochastic differential equation operating in the unit simplex whose vertices are the disability profiles. Mortality at a given age/time is represented by a quadratic function of the time-varying "scores," i.e., solutions for the stochastic differential equations in the simplex. In this formulation, high mortality rates are associated with excursions of the score vector away from $(0, \ldots, 0, 1, 0, \ldots, 0)$, where 1 is associated with the profile having "mild" (or no) disabilities (i.e., the "origin" of the space is a priori specifiable as the "state" having no "dis"-ability). Senescence is the average effect of unobserved variables at a given age. The hazard is $Q(\mathbf{g}) \exp(\theta \cdot \text{age})$ where $Q(\mathbf{g})$ is a quadratic function of the score vector, $\mathbf{g}$.

Ideally, the hazard would contain both physiological variables and disabilities. A model for a comprehensive set of state variables representing multiple levels of biological organization is beyond this paper's scope. We do illustrate a model where exogenous factors are allowed to influence the disability dynamics. For a discussion of issues in creating multilevel models, and their use in forecasting, see Manton (1993).

### 2.2.2 Physiological Dynamics

Assume a vector of $J$ variables, $\mathbf{x}(t)$, is governed by stochastic differential equations

$$d\mathrm{x}(t) = \mathrm{a}(t, \mathrm{x}(t))\, dt + \mathrm{b}(t, \mathrm{x}(t))\, d\mathrm{W}(t), \tag{1}$$

where $\mathbf{W}(t)$ is a $J$-dimensional Brownian motion process independent of initial values $\mathbf{x}(0)$, $\mathbf{b}(t, \mathbf{y})$ is a bounded matrix-valued function whose entries are scale factors governing the size of random fluctuations around $\mathbf{y} = \mathbf{x}(t)$, and $\mathbf{a}(t, \mathbf{y})$ is a vector governing drift in the neighborhood of $\mathbf{y} = \mathbf{x}(t)$. Equation (1) describes dynamics for a cohort; hence, age and time are confounded. To unconfound age and time, $\mathbf{a}$ and $\mathbf{b}$ must be parameterized by age or, equivalently, by birth cohort, $c$, with age $= t - c$, where $t$ is calendar time. Values of $\mathbf{x}(t)$ are deviations (excursions) about an optimal (minimum) risk vector of state variable values.

For the current example, we restrict $\mathbf{a}(t, \mathbf{y})$ to be

$$\mathrm{a}(t, \mathrm{y}) = \mathrm{a}_0(t) + \mathrm{a}_1(t)\mathrm{y}, \tag{2}$$

where $\mathbf{a}_1(t)$ is a restoring ("homeostatic") effect. We assume $\mathbf{b}$ depends on age/time and *not* on the level, $\mathbf{y} = \mathbf{x}(t)$. Equation (1) reduces to

$$d\mathrm{x}(t) = [\mathrm{a}_0(t) + \mathrm{a}_1(t)\mathrm{x}(t)]\, dt + \mathrm{b}(t)\, d\mathrm{W}(t). \tag{3}$$

### 2.2.3 Mortality

Let $T$ be a random age/time-at-death variable, with survival, conditional on $T$,

$$P(T > t \,|\mathrm{x}(s), 0 \le s < t) = \exp\left[-\int_0^t \mu(s, \mathrm{x}(s))\, ds\right], \tag{4}$$

where

$$\mu(s, \mathrm{x}(s)) = \left[\mu_0(s) + \mathrm{b}^{\mathrm{T}} \cdot \mathrm{x}(s) + \frac{1}{2}\mathrm{x}^{\mathrm{T}}(s)\,\mathrm{B}(s)\,\mathrm{x}(s)\right] e^{\theta s}. \tag{5}$$

Here $e^{\theta s}$ is senescence. The quadratic describes mortality risk due to excursions of state variables away from values with minimum risk, e.g., $x_0$. The dynamics for an individual evolve according to the diffusion process (3) for a random length of time, $T$, where conditional survival is governed by equations (4) and (5). If the initial vector, $\mathbf{x}(0)$, is Gaussian, then the Gaussian property propagates to $\mathbf{x}(t)$ for all $t > 0$, even with mortality selection (Woodbury and Manton 1977). When $\mathbf{a}_0$, $\mathbf{a}_1$, and $\mathbf{b}$ are constants, then (3) is the Ornstein-Uhlenbeck process, a unique time-homogeneous Gaussian diffusion process.

### 2.2.4 Cause-Specific Mortality and Competing Risks

Let $c_1, \ldots, c_K$ be causes of death, and $T_1, \ldots, T_K$ random variables representing ages/times at death from $c_1, \ldots, c_K$; $T$ = age/time at death = min $(T_1, \ldots, T_K)$. With the joint survival function $S(t_1, \ldots, t_K) = P(T_1 > t_1, \ldots, T_K > t_K)$, observe that $S(t) = P(T > t) = S(t, \ldots, t)$. The *net* hazard for $c_k$ in the absence of other causes is $\lambda_k(t) = -\left.\frac{dP(T_k > t)}{dt}\right/ P(T_k > t)$. The crude hazard for $c_k$ in the presence of other causes is

$$\mu_k(t) = -\frac{1}{S(t, \ldots, t)} \left.\frac{\partial S(t_1, \ldots, t_K)}{\partial t_k}\right|_{t_1 = \cdots = t_K = t}. \tag{6}$$

The rate $\mu(t) = -\frac{d}{dt} S(t, \ldots, t)/S(t, \ldots, t)$ is the sum of crude hazard rates, $\mu_k(t)$, by the definition of the total differential; i.e.,

$$\mu(t) = -\frac{d}{dt} S(t, \ldots, t)/S(t, \ldots, t) = \tag{7}$$

$$-\frac{1}{S(t, \ldots, t)} \sum_{k=1}^{K} \left.\frac{\partial S(t_1, \ldots, t_K)}{\partial t_k}\right|_{t_1 = \cdots = t_K = t} = \sum_{k=1}^{K} \mu_k(t).$$

The survival function, $S(t) = P(T > t)$, is

$$S(t) = \exp\left[-\int_0^t \mu(s)\, ds\right] = \exp\left[-\sum_{k=1}^{K} \int_0^t \mu_k(s)\, ds\right] = \tag{8}$$

$$\prod_{k=1}^{K} \exp\left[-\int_0^t \mu_k(s)\, ds\right].$$

If we assume $T_1, \ldots, T_K$ are independent, then

$$S(t) = \prod_{k=1}^{K} P(T_k > t) = \prod_{k=1}^{K} \exp\left[-\int_0^t \lambda_k(s)\, ds\right]; \tag{9}$$

i.e., net and crude mortality rates are equal, assuming independence.

We can represent dependence among $T_1, \ldots, T_K$ generated by state processes, $\{\mathbf{x}(t), t \geq 0\}$. Let $X_0^t$ be the history of the process $\mathbf{x}(s)$ over $0 \leq s < t$. More formally, $X_0^t$ is the minimal $\sigma$-algebra generated by $\mathbf{x}(s)$ for $0 \leq s < t$. We assume that, conditional on $X_0^t$, $T_1, \ldots, T_K$ are independent. Then

$$P(T > t | X_0^t) = \prod_{k=1}^{K} P(T_k > t \,|X_0^t). \tag{10}$$

Conditional independence of $T_1, \ldots, T_K$, given $X_0^t$, means, that the process $\mathbf{x}(t)$ accounts for ("explains") the unconditional dependence of $T_1, \ldots, T_K$. If we

assume independence of $T_1, \ldots, T_K$ conditional on $X_0^t$, conditional net and crude rates are equal. The conditional survival function is

$$P(T_k > t|X_0^t) = \exp\left[-\int_0^t \sum_{k=1}^{K} \mu_k(s|\, X_0^s)\, ds\right], \tag{11}$$

where $\mu_k(s \mid X_0^s)$ is the crude mortality rate conditional on the history of the process to $s$. Forecasts in section 2.3 assume that $\mu_k(s \mid X_0^s) = \mu_k(s, \mathbf{x}(s))$; i.e., only current values of $\mathbf{x}(s)$ are informative. This is plausible if components of $\mathbf{x}(s)$ can include measures of the effect of past history to $s$. For specifications involving more complex dependence on process history, see Yashin, Manton, and Stallard (1986).

We parameterize $\mu_k(s, \mathbf{x}(s))$, analogous to equation (5), as

$$\mu_k(s, \mathbf{x}(s)) = [\mu_{0,k}(s) + \mathbf{b}_k^{\mathrm{T}} \cdot \mathbf{x}(s) + \frac{1}{2}\, \mathbf{x}^{\mathrm{T}}(s)\, \mathbf{B}_k \mathbf{x}(s)]\, e^{\theta s}\,. \tag{12}$$

Thus each $\mu_k$ is a quadratic function of $J$ state variables. Senescence has a common value of $\theta$ for all $c_k$; i.e., senescence is the age-specific average effect of unknown factors on death. The unconditional survival function is

$$P(T > t) = \exp\left[-\int_0^t \sum_{k=1}^{K} \bar{\mu}_k(s)\, ds\right], \tag{13}$$

where

$$\bar{\mu}_k(t) = E[\mu_k(t, \mathbf{x}(t)) \mid T > t] \tag{14}$$

(see Yashin et al. 1986). Here equation (14) is the unconditional crude hazard for $T_k$. To see, in a simple scalar case with $J = 1$, how the $\bar{\mu}_k(t)$ are related by $\mathbf{x}(t)$—i.e., the explicit form of dependence—suppose that $\mu_k(t, \mathbf{x}(t)) = h_k(t)\mathbf{x}^2(t)$. Then, after manipulation, it can be shown (Yashin et al. 1986) that

$$\bar{\mu}_k(t) = h_k(t)[\mathbf{m}^2(t) + \gamma(t)], \tag{15}$$

where

$$\mathbf{m}(t) = E[\mathbf{x}(t) \mid T > t]$$

and

$$\gamma(t) = \mathrm{Var}[\mathbf{x}(t) \mid T > t].$$

These quantities satisfy the system of ordinary differential equations

$$\begin{aligned} \frac{d\mathbf{m}(t)}{dt} &= [\mathbf{a}_0(t) + \mathbf{a}_1(t)\mathbf{m}(t)] - 2\gamma(t) \sum_{k=1}^{K} h_k(t)\mathbf{m}(t), \\ \frac{d\gamma(t)}{dt} &= 2\mathrm{a}_1(t)\, \gamma(t) + \mathbf{b}^2(t) - 2\gamma^2(t) \sum_{k=1}^{K} h_k(t). \end{aligned} \tag{16}$$

To generate forecasts with cause elimination, one sets $h_k^0(t) \equiv 0$ when cause $c_k^0$ is eliminated. Observe that $\mathbf{m}(t)$ and $\gamma(t)$ depend—via equation (16)—on all other rates, $h_k(t)$; $k \neq k^0$. The dependence of $\mu_k(t)$ on state variables is—for $\mu_k(t, \mathbf{x}(t)) = h_k(t)\, \mathbf{x}^T(t) \cdot \mathbf{x}(t)$—through $\mathbf{m}(t)$ and $\gamma(t)$ in equation (15). Although more general specifications (e.g., eq. [12]) are more complicated than equation (15), the dependence of competing risks still operates through differential equation systems for $\mathbf{m}(t)$ and $\gamma(t)$.

### 2.2.5 Disability and Grade of Membership (GoM) Models

Survey-based assessments of disability yield vectors of discrete responses, $\mathbf{x}$, for each individual. Commonly, among the elderly, many individuals have multiple disabilities—but no specific combination occurs with high frequency. The distribution of disabilities in a population is best described by constructing empirically (and biologically) defensible profiles of co-occurring disabilities to be the vertices of a unit simplex. Each individual is associated with a point, $\mathbf{g}_i$, in the simplex. Components of $\mathbf{g}_i = (g_{i1}, \ldots, g_{iK})$ are convex weights (where $g_{ik} \geq 0$ and $\sum_{k=1}^{K} g_{ik} = 1$) representing the "degrees of similarity" of individuals to each profile (or distances to each vertex). For example, a person with $\mathbf{g}_i = (0, 1, 0, \ldots, 0)$ has disabilities only found in the second profile. A person with $\mathbf{g}_i = (2/3, 1/6, 0, \ldots, 0, 1/6)$ has some disabilities from profiles 1, 2, and $K$; however, more of his conditions are in profile 1 (the score of 2/3) than in profiles 2 and $K$ (i.e., scores of 1/6).

The use of the $g_{ik}$ is related to incidental parameter estimation problems discussed by Neyman and Pearson, Neyman and Scott, and others. Resolution of the problem requires imposition of a "smoothing" operator on incidental parameters (e.g., Kiefer and Wolfowitz 1956). In GoM, the statistical properties of the $g_{ik}$ are derived from theorems due to Weyl (1949) on polyhedra. Specifically, such models are identifiable and parameters consistently estimated, because once $J$ discrete variables are selected, a space of potential responses, say $M$, constructed from $\sum_{j=1}^{J}$ basis vectors (i.e., containing only 0s or 1s) is fixed. The probabilities calculated from score estimates ($g_{ik}$), vertex coordinates ($\lambda_{kjl}$), and observed responses define a linear parameter space, $L_B$, bounded by $M$. The intersection $L_B \cap M$ yields the simplex, $B$, whose vertices define the profiles (i.e., $\lambda_{kjl}$ coordinates) and whose faces define the half-spaces for the $g_{ik}$ (Woodbury, Manton, and Tolley 1994). The convex constraints imposed on the $g_{ik}$ by $M$ mean that all individuals are represented on the boundary, or in the interior of $B$, and that each individual's coordinates are uniquely defined (given his responses) because of the definition of vertices, $\lambda_{kjl}$, by $L_B \cap M$. This differs from multivariate continuous variable models where coordinate systems are constructed to represent central mass points (equivalent to centers of gravity) of specific multivariate distribution functions. It also differs from contingency tables (Bishop, Fienberg, and Holland 1975) and latent class models (LCM; Lazarsfeld and Henry 1968) used for discrete variables. In those procedures, the $g_{ik}$ must be 0 or 1. In contingency tables, each person's group

is observed (i.e., which $g_{ik} = 1.0$ is known) so that only the $\lambda_{kjl}$ (for each of $K$ observed groups) are estimated—under the constraint that groups are discrete (i.e., $g_{ik}$ can only be 0 or 1). In LCM, groups are not observed, so that the $g_{ik}$ must be estimated. Again the groups are discrete, so the state variable scores, $g_{ik}$, can only be 0 or 1, though the probability of being in a group (i.e., $p_{ik} = P(g_{ik} = 1.0)$ is what is typically estimated. In GoM there is an additional within-group heterogeneity component, due to the continuous scaling of the $g_{ik}$, not represented in LCM. However, LCM is nested within GoM, so likelihood ratio tests of model specification can be made. In forecasting, the process is generally restricted to the unit simplex $B$. In forecasting using LCM, cases can only fall on the vertices, with all transitions being discrete. $B$ imposes constraints on the forecasts, although, if well-specified functions relate exogenous factors to the $\lambda_{kjl}$ and $g_{ik}$, it is possible to use those functions to predict changes in the unit simplex; e.g., new variables can become relevant, changing the space $M$.

To formalize this, response vectors are modeled as

$$P(\mathbf{X} = l) = \int_{S_K} P[\mathbf{X}^{(\mathbf{g}_i)} = l | \mathbf{g}_i = \boldsymbol{\gamma}_i] \, d\mu(\boldsymbol{\gamma}_i) , \tag{17}$$

where $\mathbf{X}^{(\mathbf{g}_i)}$ is a random response vector for an individual with score vector, $\mathbf{g}_i$, and $\mu(\boldsymbol{\gamma}_i)$ is a probability measure on the unit simplex with $K$ vertices, $S_K$. Dependence among coordinates in the response vector is modeled assuming: (i) conditional on $g_i$, coordinate variables are independent; i.e.,

$$P[\mathbf{X}^{(\mathbf{g})} = l \mid \mathbf{g}_i = \boldsymbol{\gamma}_i] = \prod_{j=1}^{J} P[X^{(\mathbf{g}_i)} = l_j \mid \mathbf{g}_i = \boldsymbol{\gamma}_i] ; \tag{18}$$

and (ii) the conditional marginal frequencies, $P[X_j^{(\mathbf{g})} = l_j \mid \mathbf{g}_i = \boldsymbol{\gamma}_i]$, are convex combinations of profile frequencies for the same variable; i.e.,

$$P[X_j^{(\mathbf{g})} = l_j \mid \mathbf{g}_i = \boldsymbol{\gamma}_i] = \sum_{k=1}^{K} \gamma_{ik} \, P[Y_{ij}^{(k)} = l_j] \equiv \sum_{k=1}^{K} \gamma_{ik} \, \lambda_{kjl_j}. \tag{19}$$

Here $Y_{ij}^{(\mathbf{k})}$ is a random variable describing responses to $j$ by $i$ with the characteristics of $k$.

Equations (17)–(19) describe the distribution of individuals at a fixed age/time. Disability dynamics are modeled as a diffusion process in $B$; i.e., the evolution of the $g_{ik}$ are described relative to a fixed set (or a fixed set conditional on exogenous factors) of $K$ profiles. For $K = 2$, scores evolve according to a diffusion process on an unit interval. With $\mathbf{g}_i = (g_{i1}, g_{i2}) \equiv (g_{i1}, 1 - g_{i1})$, the stochastic differential equation is

$$dg_{i1}(t) = [a_0(t) + a_1(t)g_{i1}(t)] \, dt + c \sqrt{g_{i1}(t) \, [1 - g_{i1}(t)]} \, dW(t), \tag{20}$$

where $W(t)$ is standard Brownian motion, to describe dynamics within the unit interval. If we assume that $\{0\}$ and $\{1\}$—points identified by two profiles—

are "reflecting" boundaries, then transition probabilities governing equation (20) in the closed interval [0, 1] are given by the "fundamental" solution of

$$\frac{\partial u}{\partial t} = c^2 x(1 - x)\frac{\partial^2 u}{\partial x^2} + [a_0(t) + a_1(t)\, x]\frac{\partial u}{\partial x}, \quad \text{for } (x, t) \in [0,1] \times [0, +\infty),\ u(0, x) = f(x) \in C^2[0, 1],$$

subject to

$$\left.\frac{\partial u}{\partial x}\right|_{x=1-} = \left.\frac{\partial u}{\partial x}\right|_{x=0+} = 0\,, \tag{21}$$

i.e., reflecting boundary conditions.

The "local" variance (diffusion-term) specification, $c^2x(1 - x)$, implies that variance in the region of $g_1 = x$ is the same as for Bernoulli trials. The age/time-dependent coefficient, $a_1(t)$, defines drift either toward or away from profile 1, depending on its sign. Forecasts in section 2.3 are high-dimensional generalizations of equations (20)–(21). The discrete-time analogue of the process used for estimation is in subsection 2.2.6. For a more extensive discussion of GoM, see Manton et al. (1992b), Woodbury et al. (1994), Berkman, Singer, and Manton (1989), or Singer (1989).

Mortality, influenced by disability, is represented by the survival function

$$P(T > t|\mathbf{g}(s),\ 0 \le s < t) = \exp\left[-\int_0^t \mu(s,\ \mathbf{g}(s))\, ds\right],$$

where

$$\mu(s,\ \mathbf{g}(s)) = [\frac{1}{2}\,\mathbf{g}^{\mathrm{T}}(s)\ \mathbf{B}\ \mathbf{g}(s)]e^{\theta s}\,. \tag{22}$$

Thus, mortality is governed by excursions of $g_{ik}(s)$ and the family of quadratic functions defined by (22), together with the average age-specific effect ($\theta$) of unobserved factor(s).

### 2.2.6 Discrete-Time Approximations, Likelihood, and Forecasting Algorithms

Since the data used to estimate parameters in equations (3)–(5) are often collected in multiwave panel designs, observations on physiological variables and disabilities are of the form $\mathbf{x}(t_l)$, $l = 1, 2, \ldots$, (number of assessments), where $t_l$ denotes the $l$th survey date. The discrete-time analogue of equation (3) is

$$\mathbf{x}_{t+1} = u_t + R_t\,\mathbf{x}_t + \mathbf{e}_t\,, \tag{23}$$

where $R_t - I$ is the analogue of $\mathbf{a}_1(t)$, $\sum_t = E(\mathbf{e}_t\,\mathbf{e}_t^T)$ corresponds to $\mathbf{b}(t) \cdot \mathbf{b}^T(t)$, and $\{\mathbf{e}_t\}$ are independent, Gaussian distributed vectors with mean $\mathbf{O}$ and covar-

iance matrix $\sum_t$. For estimation we generalize equation (23) to identify individuals in specific cohorts; i.e.,

(24)
$$\mathbf{x}_{t+1} = \mathbf{u}_0 + \mathbf{u}_1 \text{ age}_t + \mathbf{R}_1 \mathbf{x}_t + \mathbf{R}_2 \mathbf{x}_t \text{ age}_t + \mathbf{R}_3 \cdot \mathbf{z}_t + \mathbf{e}_t (\text{age}_t)^d ,$$

where $\text{age}_t$ means "age of the individual at calender time $t$," $\mathbf{z}_t$ is a vector of exogenous variables, and $\mathbf{u}_0$ is a vector of genetically determined levels on $J$ physiological variables. The age/time-dependent mortality rate is

$$\mu(\text{age}_t, \mathbf{x}_t) = (\mu_0 + \mathbf{b}^T \cdot \mathbf{x}_t + \frac{1}{2} \mathbf{x}_t^T \mathbf{B} \mathbf{x}_t) \exp(\theta \text{ age}_t) . \quad (25)$$

The time scale for equations (23)–(25) is the intersurvey interval. This is reasonable when the time between surveys is "small" relative to the time required for "substantial" change on state variables. Alternatively, if observations are made at time points that are widely spaced relative to rates of change in underlying processes, then one must evaluate how well discrete time observations can be embedded in a continuous-time diffusion process, equation (1) (see Singer and Spilerman 1976; Frydman and Singer 1979, who discuss the problem for finite-state Markov chains). This is a substantive issue about the model specification used to estimate parameters of the theoretical process of interest given available data—and about its limitations. Two approaches are useful for this problem. First, if there is variation in the time of assessment (i.e., it is triggered by changes in health—as may be the case in studies of LTC delivery systems), then the process can be divided into the smallest possible time unit (e.g., a month), and, for GoM, the $g_{ik \cdot t}$ can be assumed to be unchanged until a new assessment is made (i.e., until there is a jump in information). Then the $g_{ik \cdot t}$ are recalculated. The vertices ($\lambda_{kjl}$) are assumed constant over all time, so that the $g_{ik \cdot t}$ at any time are comparable. Then the monthly process, which more accurately approximates continuous time, may be used. This was used to evaluate the performance of Social/Health Maintenance Organizations (Manton et al. 1994). Since an assessment is done (in theory) as often as health changes, the approximation of the continuous time process should be good. A second strategy can be used for surveys with list samples (e.g., the National Long-Term Care Survey [NLTCS]), where administrative records provide partial information on the continuous time process. This was done using the 1982 and 1984 NLTCS where mortality occurring within 3, 6, or 12 months of assessment could be defined. Changes in the mortality rate over, say, five years can be compared, for GoM, with the $g_{ik \cdot t}$'s relation to mortality over three or six months. Changes in mortality over five years gives ancillary information on likely aggregate changes in disability using maximum likelihood estimates of the mortality disability relation for shorter intervals (Manton, Stallard, and Woodbury 1991).

The likelihood, based on equations (24) and (25), using the time scale defined by intersurvey periods, is

$$
\begin{aligned}
L = \prod_{i=1}^{I} \{ & \phi(\mathbf{x}_{it_0} | \text{age}_{it_0}) \\
& \times \prod_{t=t_0+1}^{T_i} \phi(\mathbf{x}_{it} | \mathbf{x}_{it-1}, \text{age}_{it-1}, \delta_{it-1} = 0) \exp(-\mu(\mathbf{x}_{it-1}, \text{age}_{it-1})) \\
& \times \exp(-\mu(\mathbf{x}_{1T_i}, \text{age}_{iT_i}))^{(1-\delta_{iT_i})} [1 - \exp(-\mu(\mathbf{x}_{iT_i}, \text{age}_{iT_i}))]^{\delta_{iT_i}} \}.
\end{aligned} \tag{26}
$$

In equation (26) $\mathbf{x}_{it}$ and age $_{it}$ are observed $(T_i + 1) - t_0$ times for person $i$. At $t + 1$ survival is assessed: $\delta_{it} = 1$ if $i$ dies before $t + 1$; $\delta_{it} = 0$ otherwise. Initial conditions are the distribution of $\mathbf{x}_{i0}$, conditional on age $(\phi(\mathbf{x}_{i0}|\text{age}_{i0}))$, assuming random sampling. If sampling is nonrandom within age or if the model is applied to a new population, $\phi(\mathbf{x}_{i0}|\text{age}_{i0})$ can be reweighted to eliminate bias (Dowd and Manton 1990). Second (and subsequent) observation(s) on a person define the second term in equation (26)—a multivariate time series, where $\phi(\mathbf{x}_{it}|\mathbf{x}_{it-1}, \text{age}_{it-1}, \delta_{it-1} = 0)$ is the density of $\mathbf{x}_{it}$ conditioned on $\mathbf{x}_{it-1}$, $\text{age}_{it-1}$, and $\delta_{it-1} = 0$; $I$ = number of persons in the population. One can see that the likelihood varies from that in standard time-series models (e.g., Box Jenkins) where mortality selection is not modeled.

Cohort life tables, and forecasts of their parameters beyond the bounds of the data, are based on recurrence formulas. First we set $l_t = P(T > t)$, then

$$
l_{t+1} = l_t \left| \mathbf{I} + \mathbf{V}_t \mathbf{B}_t \right|^{-1/2} \exp\left[ \frac{\mu_t(\boldsymbol{\nu}_t) + \mu_t(\boldsymbol{\nu}_t^*)}{2} - 2\mu\left( \frac{\boldsymbol{\nu}_t + \boldsymbol{\nu}_t^*}{2} \right) \right], \tag{27}
$$

where $\mu_t(\cdot)$ is the mortality rate, (25), with the exponential term absorbed into $\mu_{0t}$, $\mathbf{b}_t$, and $\mathbf{B}_t$; $\boldsymbol{\nu}_t$ and $\mathbf{V}_t$ are the mean vector and covariance matrix, respectively, of state variables at $t$; $\boldsymbol{\nu}_t^*$ and $\mathbf{V}_t^*$ are adjusted for survival to $t + 1$. $\boldsymbol{\nu}_t$ and $\mathbf{V}_t$ satisfy

$$
\begin{aligned}
\boldsymbol{\nu}_t^* &= \boldsymbol{\nu}_t - \mathbf{V}_t^* (\mathbf{b}_t + \mathbf{B}_t \boldsymbol{\nu}_t), \\
\mathbf{V}_t^* &= (\mathbf{I} + \mathbf{V}_t \mathbf{B}_t)^{-1} \mathbf{V}_t, \\
\boldsymbol{\nu}_{t+1} &= \mathbf{u}_t + \mathbf{R}_t \boldsymbol{\nu}_t^*, \\
\mathbf{V}_{t+1} &= \mathbf{R}_t \mathbf{V}_t^* \mathbf{R}_t^{\mathrm{T}} + \Sigma_t,
\end{aligned} \tag{28}
$$

where

$$
\begin{aligned}
\mathbf{u}_t &= \mathbf{u}_0 + \mathbf{u}_1 \, \text{age}_{t-1}, \\
\mathbf{R}_t &= \mathbf{R}_1 + \mathbf{R}_2 \, \text{age}_{t-1},
\end{aligned}
$$

and, for the Framingham data,

$$
\mathbf{R}_3 \equiv \mathbf{O}.
$$

For analysis of disability, we used $\mathbf{R}_3$ to model the effects of income and education on disability transitions. Equation (28), for physiological variables,

ensures that $\mathbf{X}_{t+1}$ is normally distributed with mean vector $\boldsymbol{\nu}_{t+1}$ and covariance matrix $\mathbf{V}_{t+1}$—i.e., $N(\boldsymbol{\nu}_{t+1}, \mathbf{V}_t)$—given that $\mathbf{X}_t \underline{L} N(\boldsymbol{\nu}_t, \mathbf{V}_t)$. Furthermore, $\mathbf{X}_t \underline{L} N(\boldsymbol{\nu}_t^*, \mathbf{V}_t^*)$ is the conditional distribution of $\mathbf{X}_t$ given survival to $t + 1$. For a derivation of these relations, see Woodbury and Manton (1983) and Manton, Stallard, and Woodbury (1986). For disability, the process is not Gaussian due to constraints on **B.** Diffusion ($\Sigma_t$) is a time-dependent variable with variance related to that of Bernoulli trials.

To represent multiple causes of death, we use the crude mortality rate for $c_k$ at $t$, $u_{kt}(\mathbf{x}_t) = \mu_{0k} + \mathbf{b}_k^T \cdot \mathbf{x}_t + \frac{1}{2} \mathbf{x}_t^T \mathbf{B}_k \mathbf{x}_t) \exp(\theta \text{ age}_t)$ and, using $\mathbf{B}_{kt} = \mathbf{B}_k \exp(\theta \text{ age}_t)$, observe

$$\bar{\mu}_{kt} = E[\mu_{kt}(\mathbf{x}_t) \mid T > t] = 2\,\mu_{kt}[(\boldsymbol{\nu}_t + \boldsymbol{\nu}_t^*)/2] - [\mu_{kt}(\boldsymbol{\nu}_t) + \mu_{kt}(\boldsymbol{\nu}_t^*)]/2 + \frac{1}{2} \ln \mid \mathbf{I} + \mathbf{V}_t \mathbf{B}_t \mid \text{tr}[\mathbf{V}_t \mathbf{B}_{kt}] / \text{tr}[\mathbf{V}_t \mathbf{B}_t] \tag{29}$$

(see Manton, Stallard, et al. 1992), with life tables generated by

$$l_{t+1} = l_t \exp\left(-\sum_{k=1}^{K} \bar{\mu}_{kt}\right). \tag{30}$$

To represent the effects of $l_t$ of eliminating cause $k$ in the dependent competing risk framework, we set the force of mortality for the $k$th cause, $\mu_{kt}$, equal to zero in

$$\mu(\text{age}_t, \mathbf{x}_t) = (\mu_0 + \mathbf{b}^{\mathrm{T}} \cdot \mathbf{x}_t + \frac{1}{2} \mathbf{x}_t^{\mathrm{T}} \mathbf{B}\, \mathbf{x}_t) \exp(\theta \text{ age}_t),$$

where

$$\mu_0 = \sum_{k=1}^{K} \mu_{0k}, \quad \mathbf{b} = \sum_{k=1}^{K} \mathbf{b}_k, \quad \text{and } \mathbf{B} = \sum_{k=1}^{K} \mathbf{B}_k.$$

For disability dynamics, instead of equation (26), parameters are obtained by maximizing the conditional (on the $g_{ik}$) likelihood

$$L = \prod_i \prod_j \prod_l \left(\sum_k g_{ik} \cdot \lambda_{kjl}\right)^{x_{ijl}}. \tag{31}$$

Here

$$x_{ijl} = \begin{cases} 1 & \text{if individual } i \text{ has response } l \text{ on variable } j, \\ 0 & \text{otherwise,} \end{cases}$$

and $\mathbf{g}^{(i)} = (g_{i1}, \ldots, g_{iK})$, where $g_{ik} \geq 0$ and $\sum_{k=1}^{K} g_{ik} = 1$. For a discussion of computation, see Manton and Stallard (1988).

With multiple $g_{ik \cdot t}$ for each individual (i.e., eq. [31] is expanded by disaggregating individual observations into episodes based on assessment at each $t$), and $\lambda_{kjl}$s fixed over $t$, the discrete time analogue of diffusion in $B$ is

(32) $$\mathbf{g}_{i(t+1)} = \mathbf{C}_t\, \mathbf{g}_{it} + \mathbf{e}_{i(t+1)},$$

where $\mathbf{C}_t$ is—for each $t$—a $K \times K$ matrix of coefficients which are functionally equivalent to regression coefficients subject to constraints that each $\mathbf{C}_t$ be a stochastic matrix and $g_{ik(t+1)} \geq 0$, $\sum_{k=1}^{K} g_{ik} = 1$. For $h \neq k$, $c_{hkt}$ is the movement in $B$—away from profile $k$ and toward $h$ during $[t, t+1]$. Similarly, $1 - c_{kkt}$ is the total age/time-dependent movement from $k$ during $[t, t+1]$.

## 2.3 Forecasts

### 2.3.1 Data

The first analysis uses as state variables physiological risk factors measured in the Framingham Study (Dawber 1980) for 2,336 males and 2,873 females aged 29–62 years in 1950. The risk factors, measured biennally, were age (years), sex, diastolic blood pressure (DBP; mm Hg), pulse pressure (PP; mm Hg), serum cholesterol (SC; mg/dl), vital capacity index (VCI; cl/m$^2$), hemoglobin (Hb; dg%) or hematocrit (Ht), smoking (CIG; cigarettes per day), body mass index (BMI; hg/m$^2$), blood sugar (BLDS; mg%), ventricular (heart) rate (VRATE), and left ventricular hypertrophy (LVH).

Disability assessments were obtained from the 1982, 1984, and 1989 National Long-Term Care Surveys (NLTCS). Twenty-seven disability measures, listed in table 2.1, were obtained from all chronically disabled persons interviewed in the two community samples of the 1982 and 1984 NLTCS ($N$ = 11,535). These were used in a GoM analysis to produce a six-profile solution defined by the $\lambda_{kjl}$ in table 2.1. The 1989 NLTCS is used to confirm forecasts based on 1982 and 1984.

The profiles in table 2.1 may be interpreted as follows:

- 1 is "healthy" with few chronic impairments,
- 2 has no Activities of Daily Living (ADL) and few physical impairments but has Instrument Activities of Daily Living (IADL) impairments associated with cognition (e.g., phoning, managing money, and taking medication),
- 3 has no ADL and few IADL impairments but moderate physical limitations (e.g., climbing stairs and holding, reaching for, and grasping objects),
- 4 has problems with bathing, several IADLs, and more physical functions,
- 5 has several ADL and IADL impairments (but not involving cognition, cf. profile 2; profile 4 had more upper body impairment), and
- 6 is highly impaired on multiple ADLs and IADLS.

Thus, the profiles describe different dimensions of function, e.g., cognitive impairment (profile 2), upper (profile 4) and lower body function (profile 5), and mixed or combined disability and frailty. There is a rough tendency for disability to increase across profiles.

**Table 2.1** **Estimates of Response Profile Probabilities ($\lambda_{kjl} \times 100$) for the Combined 1982 and 1984 NLTCS Sample (11,535 complete detailed interviews)**

| | | Profiles | | | | | |
|---|---|---|---|---|---|---|---|
| Variable | Observed Frequency | Healthy (1) | Moderate Cognitive Impairment (2) | Mild Instrumental and Physical Impairment (3) | Serious Physical Impairment (4) | Moderate ADL and Serious Physical Impairment (5) | Frail (6) |
| ADL—Needs help: | | | | | | | |
| Eating | 6.1 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 46.2 |
| Getting in/out of bed | 26.3 | 0.0 | 0.0 | 0.0 | 0.0 | 76.7 | 100.0 |
| Getting around inside | 40.6 | 0.0 | 0.0 | 0.0 | 0.0 | 100.0 | 100.0 |
| Dressing | 19.8 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 100.0 |
| Bathing | 44.0 | 0.0 | 0.0 | 0.0 | 42.0 | 100.0 | 100.0 |
| Using toilet | 21.3 | 0.0 | 0.0 | 0.0 | 0.0 | 41.5 | 100.0 |
| Bedfast | 0.8 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 5.3 |
| No inside activity | 1.4 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 9.8 |
| Wheelchair-fast | 3.4 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 23.0 |
| IADL—Needs help: | | | | | | | |
| With heavy work | 76.8 | 24.1 | 100.0 | 100.0 | 100.0 | 100.0 | 100.0 |
| With light work | 24.2 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 100.0 |
| With laundry | 46.1 | 0.0 | 100.0 | 18.2 | 100.0 | 45.3 | 100.0 |
| With cooking | 33.0 | 0.0 | 100.0 | 0.0 | 0.0 | 0.0 | 100.0 |
| With grocery shopping | 63.3 | 0.0 | 100.0 | 0.0 | 100.0 | 100.0 | 100.0 |
| Getting about outside | 63.5 | 0.0 | 52.7 | 55.1 | 100.0 | 100.0 | 100.0 |
| Traveling | 61.6 | 0.0 | 100.0 | 0.0 | 100.0 | 100.0 | 100.0 |
| Managing money | 29.7 | 0.0 | 100.0 | 0.0 | 0.0 | 0.0 | 100.0 |
| Taking medicine | 24.6 | 0.0 | 93.3 | 0.0 | 0.0 | 0.0 | 100.0 |
| Making telephone calls | 17.5 | 0.0 | 83.0 | 0.0 | 0.0 | 0.0 | 96.0 |

| | | | | | | | |
|---|---|---|---|---|---|---|---|
| Function limitations— | | | | | | | |
| How much difficulty do you have: | | | | | | | |
| Climbing (one flight of stairs) | | | | | | | |
| None | 15.8 | 45.3 | 20.3 | 0.0 | 0.0 | 0.0 | 0.0 |
| Some | 28.9 | 54.7 | 79.7 | 22.1 | 0.0 | 0.0 | 0.0 |
| Very difficult | 33.0 | 0.0 | 0.0 | 77.9 | 53.1 | 67.1 | 7.0 |
| Cannot at all | 22.3 | 0.0 | 0.0 | 0.0 | 46.9 | 32.9 | 93.0 |
| Bending (e.g., putting on socks) | | | | | | | |
| None | 41.4 | 100.0 | 100.0 | 0.0 | 0.0 | 100.0 | 0.0 |
| Some | 28.5 | 0.0 | 0.0 | 100.0 | 0.0 | 0.0 | 0.0 |
| Very difficult | 19.0 | 0.0 | 0.0 | 0.0 | 100.0 | 0.0 | 0.0 |
| Cannot at all | 11.1 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 100.0 |
| Holding a 10 lb. package | | | | | | | |
| None | 26.4 | 77.4 | 45.2 | 0.0 | 0.0 | 0.0 | 0.0 |
| Some | 17.8 | 22.6 | 54.8 | 21.0 | 0.0 | 22.9 | 0.0 |
| Very difficult | 16.7 | 0.0 | 0.0 | 79.0 | 0.0 | 0.0 | 0.0 |
| Cannot at all | 39.0 | 0.0 | 0.0 | 0.0 | 100.0 | 77.1 | 100.0 |
| Reaching overhead | | | | | | | |
| None | 54.0 | 100.0 | 100.0 | 0.0 | 0.0 | 100.0 | 0.0 |
| Some | 21.8 | 0.0 | 0.0 | 100.0 | 0.0 | 0.0 | 37.6 |
| Very difficult | 14.7 | 0.0 | 0.0 | 0.0 | 77.5 | 0.0 | 0.0 |
| Cannot at all | 9.5 | 0.0 | 0.0 | 0.0 | 22.5 | 0.0 | 62.4 |
| Combing hair | | | | | | | |
| None | 69.8 | 100.0 | 100.0 | 0.0 | 0.0 | 100.0 | 0.0 |
| Some | 17.1 | 0.0 | 0.0 | 100.0 | 35.1 | 0.0 | 37.4 |
| Very difficult | 7.6 | 0.0 | 0.0 | 0.0 | 65.0 | 0.0 | 0.0 |
| Cannot at all | 5.5 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 62.6 |
| Washing hair | | | | | | | |
| None | 53.4 | 100.0 | 100.0 | 0.0 | 0.0 | 100.0 | 0.0 |
| Some | 15.2 | 0.0 | 0.0 | 100.0 | 0.0 | 0.0 | 0.0 |
| Very difficult | 10.0 | 0.0 | 0.0 | 0.0 | 100.0 | 0.0 | 0.0 |

(*continued*)

**Table 2.1** (continued)

| Variable | Observed Frequency | Profiles | | | | | |
|---|---|---|---|---|---|---|---|
| | | Healthy (1) | Moderate Cognitive Impairment (2) | Mild Instrumental and Physical Impairment (3) | Serious Physical Impairment (4) | Moderate ADL and Serious Physical Impairment (5) | Frail (6) |
| Cannot at all | 21.4 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 100.0 |
| Grasping an object | | | | | | | |
| None | 64.8 | 100.0 | 100.0 | 0.0 | 0.0 | 100.0 | 27.3 |
| Some | 20.8 | 0.0 | 0.0 | 100.0 | 0.0 | 0.0 | 33.6 |
| Very difficult | 10.5 | 0.0 | 0.0 | 0.0 | 94.4 | 0.0 | 10.8 |
| Cannot at all | 3.9 | 0.0 | 0.0 | 0.0 | 5.6 | 0.0 | 28.3 |
| Can you see well enough to read a newspaper? | 73.1 | 100.0 | 0.0 | 100.0 | 71.3 | 100.0 | 46.4 |
| Mean scores ($\overline{g}_k \times 100$) | | 33.7 | 11.9 | 13.5 | 9.0 | 16.5 | 15.2 |

We reanalyzed the 27 items, using the 16,485 respondents to the combined 1982, 1984, and 1989 NLTCS. The $\lambda_{kjl}$ for the three surveys are in table 2.2. There is a high degree of similarity in the six profiles between the two solutions; i.e., $B_{82-84}$ (table 2.1) and $B_{82-89}$ (table 2.2) are similar. The primary difference is fewer IADL impairments involving outside mobility for profile 3 in table 2.2. Variables whose coefficients are on the boundary of $B$(i.e., $\lambda_{kjl} = 1.0$ or 0.0) are highly stable because the solution, given the constraints of $M$, is "hyper"-efficient. Thus, $B$ is not changed much by the extension to 1989. The trait-weighted prevalence of the six profiles, 1982–89, at the bottom of table 2.2 for both solutions, also shows a high degree of similarity.

Finally, national population counts, together with growth rates, $r_t$, in year $t$, were derived from census estimates for ages 30–100 for 1986. Projections of the population aged 30–31 between 1988 and 2080 (Spencer 1989) determined the size of new cohorts at they "age in."

### 2.3.2 Risk Factor Projections/Simulations

For projections, we need initial conditions, descriptions of two-year changes in risk factors (24), and hazard rates for cancer, cardiovascular disease, and "other" causes ($K = 3$). In table 2.3 we present sex-specific life-table parameters for selected ages, for dependent *and* independent "elimination" of CVD generated using equation (28).

Under independence, age-specific risk factor means do *not* change. With dependence, means change due to decreased *selection* of persons with adverse CVD risk factor values. For males aged 90, mean BLDS rose to 115.0 (from 111.9) because CVD elimination allows diabetics to live longer. Mortality for causes dependent on the same risk factors increase. Thus, independence overstates the effect of eliminating CVD, with bias increasing as $l_t$ decreases. By age 90, the bias for males is 39 percent (i.e., 3.1 vs. 4.3 years); 17.6 percent for females.

If there were no mortality change, the male population aged 85+ would grow 75 percent (to 1.4 million) by 2080 (the female population, 71 percent to 3.6 million) because of increased cohort size. This is less than the 9.8 million in the lowest Census Bureau Series 19 projection (mortality changes are 50 percent of the middle variant). Series 19 uses low, and Series 23 uses middle, fertility/immigration assumptions. We used fertility/immigration assumptions from Series 23. Life expectancy at age 30 (table 2.3) is similar to the United States in 1986 (i.e., males 43.8 vs. 43.9 and females 49.0 vs. 50.0). In 2080 the no mortality change scenario for the population aged 65+ is 19 percent lower than Census Bureau Series 23 (49.7 vs. 60.9 million). The relative difference for persons aged 85+ is larger (54 percent; 5.0 vs. 10.9 million). The Series 19 projections and the forecast with no mortality change are similar for the 65+ population (49.5 vs. 49.7 million).

Mortality is declining for the U.S. population aged 65+ and for those 85+ (i.e., for those aged 85+ it declined 8.6 percent from 1989 to 1991; NCHS

**Table 2.2** **Estimates of Response Profile Probabilities ($\lambda_{kjr} \times 100$) for the Combined 1982, 1984, and 1989 NLTCS ($N = 16{,}485$)**

| | | Profiles | | | | | |
|---|---|---|---|---|---|---|---|
| Variable | Observed Frequency | Healthy (1) | Moderate Cognitive Impairment (2) | Mild Instrumental and Physical Impairment (3) | Serious Physical Impairment (4) | Moderate ADL and Serious Physical Impairment (5) | Frail (6) |
| ADL—Needs help | | | | | | | |
| Eating | 7.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 55.2 |
| Getting in/out of bed | 39.9 | 0.0 | 0.0 | 0.0 | 0.0 | 100.0 | 100.0 |
| Getting around inside | 39.9 | 0.0 | 0.0 | 0.0 | 0.0 | 100.0 | 100.0 |
| Dressing | 19.4 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 100.0 |
| Bathing | 43.1 | 0.0 | 0.0 | 0.0 | 0.0 | 100.0 | 100.0 |
| Using toilet | 21.7 | 0.0 | 0.0 | 0.0 | 0.0 | 48.9 | 100.0 |
| Bedfast | 0.8 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 5.5 |
| No inside activity | 1.5 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 10.2 |
| Wheelchair fast | 7.0 | 0.0 | 0.0 | 0.0 | 0.0 | 19.9 | 25.8 |
| IADL—Needs help | | | | | | | |
| With heavy work | 71.9 | 14.5 | 100.0 | 100.0 | 100.0 | 100.0 | 100.0 |
| With light work | 22.6 | 0.0 | 35.5 | 0.0 | 0.0 | 0.0 | 100.0 |
| With laundry | 41.5 | 0.0 | 100.0 | 0.0 | 100.0 | 36.4 | 100.0 |
| With cooking | 29.8 | 0.0 | 100.0 | 0.0 | 0.0 | 0.0 | 100.0 |
| With grocery shopping | 56.9 | 0.0 | 100.0 | 0.0 | 100.0 | 100.0 | 100.0 |
| Getting about outside | 59.1 | 0.0 | 61.9 | 0.0 | 100.0 | 100.0 | 100.0 |
| Traveling | 52.9 | 0.0 | 100.0 | 0.0 | 100.0 | 100.0 | 80.3 |
| Managing money | 26.8 | 0.0 | 100.0 | 0.0 | 0.0 | 0.0 | 100.0 |
| Taking medicine | 23.5 | 0.0 | 100.0 | 0.0 | 0.0 | 0.0 | 100.0 |
| Making telephone calls | 16.0 | 0.0 | 87.3 | 0.0 | 0.0 | 0.0 | 85.5 |

| | | | | | | | |
|---|---|---|---|---|---|---|---|
| Function limitations—How much difficulty do you have: | | | | | | | |
| Climbing (one flight of stairs) | | | | | | | |
| None | 18.6 | 53.5 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 |
| Some | 29.1 | 46.6 | 88.5 | 33.8 | 0.0 | 0.0 | 0.0 |
| Very difficult | 31.4 | 0.0 | 11.5 | 66.2 | 50.7 | 73.0 | 10.9 |
| Cannot at all | 21.0 | 0.0 | 0.0 | 0.0 | 49.3 | 27.0 | 89.1 |
| Bending (e.g., putting on socks) | | | | | | | |
| None | 43.5 | 100.0 | 100.0 | 0.0 | 0.0 | 100.0 | 0.0 |
| Some | 27.9 | 0.0 | 0.0 | 100.0 | 0.0 | 0.0 | 0.0 |
| Very difficult | 18.0 | 0.0 | 0.0 | 0.0 | 100.0 | 0.0 | 0.0 |
| Cannot at all | 10.6 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 100.0 |
| Holding a 10 lb. package | | | | | | | |
| None | 29.6 | 84.2 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 |
| Some | 18.1 | 15.9 | 58.6 | 38.9 | 0.0 | 24.9 | 0.0 |
| Very difficult | 15.9 | 0.0 | 41.4 | 61.1 | 0.0 | 30.3 | 0.0 |
| Cannot at all | 36.4 | 0.0 | 0.0 | 0.0 | 100.0 | 44.7 | 100.0 |
| Reaching overhead | | | | | | | |
| None | 56.1 | 100.0 | 100.0 | 0.0 | 0.0 | 100.0 | 0.0 |
| Some | 21.2 | 0.0 | 0.0 | 100.0 | 0.0 | 0.0 | 34.3 |
| Very difficult | 13.9 | 0.0 | 0.0 | 0.0 | 76.8 | 0.0 | 14.1 |
| Cannot at all | 8.8 | 0.0 | 0.0 | 0.0 | 23.3 | 0.0 | 51.6 |
| Combing hair | | | | | | | |
| None | 71.6 | 100.0 | 100.0 | 0.0 | 0.0 | 100.0 | 0.0 |
| Some | 16.0 | 0.00 | 0.00 | 100.0 | 42.77 | 0.00 | 33.68 |
| Very difficult | 7.0 | 0.00 | 0.00 | 0.00 | 57.23 | 0.00 | 11.54 |
| Cannot at all | 5.4 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 54.8 |

(*continued*)

**Table 2.2** (continued)

| Variable | Observed Frequency | Profiles | | | | | |
|---|---|---|---|---|---|---|---|
| | | Healthy (1) | Moderate Cognitive Impairment (2) | Mild Instrumental and Physical Impairment (3) | Serious Physical Impairment (4) | Moderate ADL and Serious Physical Impairment (5) | Frail (6) |
| Washing hair | | | | | | | |
| None | 55.8 | 100.0 | 100.0 | 0.0 | 0.0 | 100.0 | 0.0 |
| Some | 14.8 | 0.0 | 0.0 | 100.0 | 0.0 | 0.0 | 0.0 |
| Very Difficult | 9.4 | 0.0 | 0.0 | 0.0 | 100.0 | 0.0 | 0.0 |
| Cannot at all | 20.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 100.0 |
| Grasping an object | | | | | | | |
| None | 66.0 | 100.0 | 100.0 | 0.0 | 0.0 | 100.0 | 24.6 |
| Some | 20.3 | 0.0 | 0.0 | 100.0 | 0.0 | 0.0 | 34.3 |
| Very Difficult | 10.1 | 0.0 | 0.0 | 0.0 | 95.5 | 0.0 | 14.8 |
| Cannot at all | 3.6 | 0.0 | 0.0 | 0.0 | 4.5 | 0.0 | 26.3 |
| Can you see well enough to read a newspaper? | 74.3 | 100.0 | 0.0 | 100.0 | 100.0 | 100.0 | 45.4 |
| Mean scores ($\bar{g}_k \times 100$) | | | | | | | |
| 1982–84 NLTCS[a] | | 33.7 | 11.9 | 13.5 | 9.0 | 16.5 | 15.2 |
| 1982, 1984, and 1989 NLTCS | | 34.4 | 11.6 | 13.1 | 9.1 | 16.7 | 15.3 |

[a]From table 2.1.

**Table 2.3** **Observed (baseline) and Cause-Elimination Life-Table Values Assuming Independence and Dependence of Competing Risks: CVD Elimination for Male and Females, Framingham Heart Study (20-year follow-up)**

| | age$_i$ | $e_i$ | PP | DBP | BMI | SC | BLDS | Hb | VCI | CIG |
|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | *Males* | | | | | |
| Baseline | 30 | 43.9 | 45.0 | 80.0 | 260.0 | 215.0 | 80.0 | 145.0 | 140.0 | 14.0 |
| Dependence | | 53.9 | | | | | | | | |
| Independence | | 54.8 | | | | | | | | |
| Baseline | 50 | 25.7 | 47.7 | 83.3 | 276.0 | 241.1 | 83.7 | 149.6 | 127.4 | 12.9 |
| Dependence | | 34.9 | 47.7 | 83.4 | 276.1 | 241.2 | 83.7 | 149.6 | 147.4 | 13.0 |
| Independence | | 35.8 | | | | | | | | |
| Baseline | 70 | 10.8 | 63.0 | 82.8 | 266.1 | 223.0 | 98.5 | 150.7 | 100.8 | 4.9 |
| Dependence | | 17.7 | 63.3 | 83.0 | 265.7 | 223.4 | 99.0 | 150.7 | 100.2 | 5.2 |
| Independence | | 18.8 | | | | | | | | |
| Baseline | 90 | 2.9 | 77.3 | 80.8 | 250.3 | 204.7 | 111.9 | 151.9 | 78.0 | 0.0 |
| Dependence | | 6.0 | 79.4 | 81.7 | 242.3 | 205.6 | 115.0 | 151.3 | 73.3 | 0.0[a] |
| Independence | | 7.2 | | | | | | | | |
| Baseline | 110 | 1.1 | 88.0 | 78.5 | 254.0 | 188.9 | 120.7 | 155.7 | 70.4 | 0.0 |
| Dependence | | 1.8 | 96.2 | 80.5 | 225.8 | 199.0 | 133.3 | 154.4 | 53.1 | 0.0 |
| Independence | | 2.7 | | | | | | | | |
| | | | | | *Females* | | | | | |
| Baseline | 30 | 50.0 | 45.0 | 75.0 | 235.0 | 200.0 | 80.0 | 125.0 | 115.0 | 8.0 |
| Dependence | | 56.9 | | | | | | | | |
| Independence | | 57.3 | | | | | | | | |
| Baseline | 50 | 30.4 | 48.9 | 80.0 | 256.0 | 246.2 | 81.9 | 135.4 | 105.7 | 10.1 |
| Dependence | | 38.0 | 48.9 | 80.0 | 256.1 | 246.3 | 81.9 | 135.4 | 105.7 | 10.1 |
| Independence | | 38.5 | | | | | | | | |
| Baseline | 70 | 13.7 | 68.8 | 83.0 | 252.5 | 255.9 | 94.2 | 141.8 | 78.1 | 6.8 |
| Dependence | | 20.3 | 68.9 | 83.0 | 252.7 | 256.1 | 94.3 | 141.9 | 77.8 | 6.9 |
| Independence | | 20.8 | | | | | | | | |
| Baseline | 90 | 3.6 | 86.9 | 83.9 | 234.7 | 263.6 | 105.1 | 146.1 | 53.5 | 1.1 |
| Dependence | | 7.0 | 88.2 | 84.7 | 233.4 | 262.7 | 106.1 | 146.5 | 51.0 | 1.4 |
| Independence | | 7.6 | | | | | | | | |
| Baseline | 110 | 1.1 | 99.6 | 80.2 | 220.1 | 275.6 | 113.0 | 148.3 | 41.4 | 0.0 |
| Dependence | | 1.8 | 105.9 | 84.3 | 210.6 | 271.2 | 118.8 | 149.2 | 29.4 | 0.0 |
| Independence | | 2.3 | | | | | | | | |

[a]Cigarette smoking was fixed at zero to prevent negative values.

1992). Declines are due, in part, to observed risk factor trends from 1960 to 1987, where smoking, cholesterol and hypertension among the U.S. elderly (age 65–74) population declined (e.g., Popkin, Haines, and Patterson 1992). Further improvement can be expected due to smoking reduction (Fiore et al. 1989), increased education (Feldman et al. 1989), and adoption of healthier life styles (e.g., more physical activity and improved nutrition) by elderly cohorts.

For our projection we had to establish "optimal" risk factor values for total

mortality. The total mortality function is generated as the sum of three cause-specific functions because certain risk factors (e.g., SC) had different relations with different causes (e.g., with CVD and cancer; Neaton et al. 1992). The cause-specific relations of risk factors is important in assessing "population" versus "high-risk" public health interventions. Indeed, "population" intervention for SC may increase mortality for a portion of the population (Frank et al. 1992). There have been questions raised about the evidence demonstrating the efficacy of SC reduction (Ravnskov 1992). There is less controversy about controlling SC by diet and exercise (which affects other metabolic parameters) than about the population use of SC-lowering drugs—especially those affecting liver enzymes (Oliver 1991). Consequently, we used the quadratic mortality model to determine the values that would increase life expectancy most, based on the 34-year Framingham follow-up. These are presented in table 2.4. Differences between the 20-year and 34-year data suggest what effects are like at the latter ages observed in the 34-year data.

There are differences between the optimal profiles for the 20-year and 34-year data on SC. This is because the quadratic surface for SC has a flat interior region due to the relations of SC to different diseases (Frank et al. 1992; Neaton et al. 1992). The additional 14 years of follow-up decreases the "optimal" male cholesterol value. The female cholesterol value remains higher (Epstein 1992). Part of the reason for the variability is the strong correlation over time of metabolic parameters. For example, BLDS, BMI, and VCI increased for males when SC declined.

The consequences of risk factor interventions, using parameters estimated from 20-year and 34-year Framingham data are in table 2.5. The mean and variance for CIG for smoking elimination are fixed at 0.0. This only modestly increases the population, because few smokers survive to age 80. Second, risk factor means were fixed at "optimal" levels for each cohort in 2006 (e.g., interventions for persons aged 30 in 1986 were introduced at age 50 in 2006, for those aged 50 in 1986 at age 70 in 2006, etc.). Life tables were calculated with $\boldsymbol{\nu}_{30}$ set to "optimal" risk factor levels, $\mathring{\mathbf{x}}_{it}$ and $\mathbf{R} = \mathbf{I}$, $\mathbf{u}_t = \mathbf{O}$, and $\Sigma = \mathbf{O}$. $\mathbf{V}_{t_0}$ was not changed. The male population in 2040 increased from 21.6 to 36.0 million at age 65 and from 1.6 to 9.8 million at age 85. Next, we partially (50% or 75%), or completely, eliminated variance by pre- and post-multiplying $\mathbf{V}_{t_0}$ by a diagonal matrix; $\Sigma$ is recalculated. The male population aged 65+ increased to 62.1 million by 2040. Males aged 85+ increased to 25.4 million by 2040, and to 36.0 million in 2080. Females had similar increases (i.e., 28.5 million in 2040 and 37.9 million in 2080). Projections based on the 20-year and 34-year optimal risk factor profiles are similar.

In table 2.6, the highest census projections (Series 9) are presented along with "optimal" projections based on 34-year data (profile 3, table 2.4).

In the "optimal" case, of 177 million persons aged 0–44 in 1986, 61 percent of females and 33 percent of males survive to age 85 in 2080. The male population aged 65+ is projected to be 74.6 million in the optimal case versus 52.5

**Table 2.4** **Risk Factor Means and Optimal Means (for eight variables, 20-year follow-up, and for ten variables, 34-year follow-up, Framingham data sets) Used in Projections**

| | Males | | | | Females | | | |
|---|---|---|---|---|---|---|---|---|
| Variable | Profile 1 (20-year) | Profile 2 (34-year) | Profile 3 (34-year) | Observed Means at Age 30 | Profile 1 (20-year) | Profile 2 (34-year) | Profile 3 (34-year) | Observed Means at Age 30 |
| 1. PP (mm Hg) | 32 | 35.5 | 27.8 | 45 (13.7) | 59 | 47.2 | 46.8 | 45 (15.5) |
| 2. DBP (mm Hg) | 82 | 74.4 | 80.5 | 80 (12.5) | 71 | 78.0 | 78.0 | 75 (12.3) |
| 3. BMI ($hg/m^2$) | 227 | 254.5 | 257.5 | 260 (34.4) | 274 | 267.3 | 267.6 | 235 (44.7) |
| 4. SC (mg/dl) | 260 | 211.8 | 172.8 | 215 (41.4) | 257 | 222.0 | 221.7 | 200 (42.9) |
| 5. BLDS (mg%) | 58 | 57.5 | 101.8 | 80 (29.6) | 107 | 124.9 | 124.4 | 80 (22.1) |
| 6. Ht (%)[a] | – | 46.8 | 47.5 | 47 (3.1) | – | 44.6 | 44.6 | 44 (3.0) |
| Hb (dg%) | 152 | – | – | 145 (10.2) | 133 | – | – | 125 (10.2) |
| 7. VCI ($cl/m^2$) | 152 | 145.9 | 160.1 | 140 (18.9) | 100 | 121.7 | 121.6 | 115 (17.0) |
| 8. CIG (cigarettes per day) | 0 | 0.0 | 0.0 | 14 (11.5) | 0 | 0.0 | 0.0 | 8 (8.1) |
| 9. LVH | – | 0.0 | 0.0 | 0.06 (0.0) | – | 0.0 | 0.0 | 0.1 (0.0) |
| 10. VRATE (per minute) | – | 61.3 | 67.4 | 77.0 (11.8) | – | 55.6 | 55.5 | 77.0 (11.6) |
| $e_{30}$ | | 70.9 | 82.5 | | | 73.2 | 73.5 | |

*Notes:* Standard deviations in parentheses.

[a]Hermatocrit value used in 34-year projections.

million in Series 9. The optimal male population aged 85+ is 38.3 million versus 13.9 million according to Series 9. Similar results occur for females aged 85+ (i.e., 39.7 vs. 20.0 million). Comparison of Series 5 and 9 projections showed that fertility/immigration produced 10 percent of the age 85+ and 18 percent of the age 65+ population increase in Series 9.

Projections of persons aged 85+ for 2040 by Guralnik, Yanagishita, and Schneider (1988), assuming a 2 percent per year mortality decline, are a third higher than Series 9 projections (i.e., 23.5 vs. 17.9 million). Ahlburg and Vaupel (1990) projected 72 million persons over 85 in 2080—similar to our optimal case (78.0 million). Their projections use a 2 percent per year mortality reduction and high fertility and immigration rates. Using middle fertility/

**Table 2.5** **Alternative Projections (in millions of Persons) for 2040, 2060, and 2080: 20-Year and 34-Year Framingham Data**

| | 2040 | 2060 | 2080 |
|---|---|---|---|
| | | *Males* | |
| *Age 65+* | | | |
| 20-Year data | | | |
| Baseline[a] | 21.6 | 21.6 | 20.7 |
| Smoking eliminated | 23.5 | 23.6 | 22.7 |
| Reduction of profile 1[b] risk factor variance by: | | | |
| 0%[c] | 36.0 | 36.8 | 35.8 |
| 50% | 51.5 | 55.5 | 54.3 |
| 75% | 58.7 | 66.3 | 65.0 |
| 100% | 62.1 | 72.0 | 71.0 |
| 34-Year data | | | |
| 100% Reduction in variance (profile 2[b]) | 63.3 | 72.9 | 71.7 |
| *Age 85+* | | | |
| 20-Year data | | | |
| Baseline[a] | 1.6 | 1.3 | 1.4 |
| Smoking eliminated | 1.7 | 1.5 | 1.6 |
| Reduction of profile 1 risk factor variance by: | | | |
| 0%[c] | 9.8 | 9.8 | 10.0 |
| 50% | 17.7 | 21.9 | 22.2 |
| 75% | 22.9 | 30.4 | 30.8 |
| 100% | 25.4 | 35.3 | 36.0 |
| 34-Year data | | | |
| 100% Reduction in variance (profile 2) | 26.4 | 36.2 | 36.7 |
| | | *Females* | |
| *Age 65+* | | | |
| 20-Year data | | | |
| Baseline[a] | 30.2 | 29.8 | 28.9 |
| Smoking eliminated | 31.4 | 31.1 | 30.2 |
| Reduction of profile 1 risk factor variance by: | | | |
| 0%[c] | 43.9 | 45.1 | 44.1 |
| 50% | 56.8 | 61.2 | 59.9 |
| 75% | 62.1 | 68.9 | 67.7 |
| 100% | 65.4 | 74.3 | 73.3 |
| 34-Year data | | | |
| 100% Reduction in variance (profile 2) | 67.3 | 74.4 | 73.3 |
| *Age 85+* | | | |
| 20-Year data | | | |
| Baseline[a] | 3.8 | 3.3 | 3.6 |
| Smoking eliminated | 4.1 | 3.7 | 4.0 |
| Reduction of profile 1 risk factor variance by: | | | |
| 0%[c] | 13.3 | 14.5 | 14.8 |
| 50% | 21.8 | 26.3 | 26.5 |
| 75% | 25.8 | 32.8 | 33.0 |
| 100% | 28.5 | 37.5 | 37.9 |
| 34-Year data | | | |
| 100% Reduction in variance (profile 2) | 31.2 | 37.8 | 37.9 |

[a]See table 2.3.

[b]Optimal values for 20-year and 34-year data are presented in table 2.4.

[c]Changes in risk factor means only.

**Table 2.6** **Comparison of Population Projections Based on Control of Multiple Risk Factors and on the High Census Bureau Variant (millions)**

| Age | Males | | | | | Females | | | | | Total | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | 1990 | 2010 | 2040 | 2060 | 2080 | 1990 | 2010 | 2040 | 2060 | 2080 | 1990 | 2010 | 2040 | 2060 | 2080 |
| *Risk Factor Control[1] (20-Year Delay)* | | | | | | | | | | | | | | | |
| 65+ | 13.0 | 24.9 | 68.1 | 75.6 | 74.6 | 18.9 | 26.5 | 70.0 | 77.5 | 76.3 | 31.9 | 51.4 | 138.1 | 153.0 | 151.1 |
| 85+ | 0.9 | 4.6 | 31.1 | 37.8 | 38.3 | 2.3 | 5.8 | 32.6 | 39.5 | 39.7 | 3.2 | 10.4 | 63.7 | 77.3 | 78.0 |
| Surviving to age 85 from 65[a] (%) | (6.7) | (18.5) | (45.7) | (50.0) | (51.3) | (12.2) | (21.9) | (46.6) | (51.0) | (52.0) | (10.0) | (20.2) | (46.1) | (50.5) | (51.6) |
| *Census Bureau Highest Variant (Series 9)[2]* | | | | | | | | | | | | | | | |
| 65+ | 11.8 | 18.1 | 37.1 | 43.1 | 52.5 | 17.5 | 24.4 | 45.5 | 51.7 | 61.4 | 29.3 | 42.5 | 82.6 | 94.8 | 113.9 |
| 85+ | 0.8 | 2.2 | 6.6 | 10.0 | 13.9 | 2.1 | 4.9 | 11.2 | 15.7 | 20.0 | 2.9 | 7.2 | 17.9 | 25.6 | 33.9 |
| Surviving to age 85 from 65[a] (%) | (6.8) | (12.2) | (17.8) | (23.2) | (26.5) | (12.0) | (20.1) | (24.6) | (30.4) | (32.6) | (9.9) | (16.9) | (21.7) | (27.0) | (29.8) |

*Sources:* For risk factor control, Duke University, Center for Demographic Studies; for Census Bureau highest variant, Spencer (1989).

[a]Figures in parentheses are percentage of persons over age 65 that are age 85+.

immigration assumptions, the 2 percent mortality decline projects 58 million persons aged 85+ in 2080. Thus, the optimal projections produce mortality declines averaging more than 2 percent per year. The 2 percent assumption generates a life expectancy of 100 years in 2080. "Optimal" interventions project life expectancies 3–12 years higher.

In the "optimal" case, senescence is assumed unchanged ($\theta$ is not altered) and no diseases are "cured." The Gompertz in equation (5) ($\theta$ is 8.05 percent for males, 8.12 percent for females) limits the life expectancy for persons with optimal risk factor profiles. Without using the 10 risk factors in the mortality function, the $\theta$ for the 34-year data was 9.4 percent for males and 10.0 percent for females. Thus, the 10 risk factors significantly reduced (by 14.4 percent and 19.0 percent) the effects ($\theta$) of unobserved variables on the age dependence of mortality. Since $\theta$ is a nonlinear parameter, the proportion of the age dependence explained by the 10 risk factors is much higher than the decline in $\theta$; about 62 percent of male and 69 percent of female age dependence of mortality was due to the risk factors.

Without $\theta$, the coefficients of the mortality function are not only biased, but do not represent the age variable equilibrium of the process (e.g., Manton 1988) because the risk factors contain "age" effects that bias them away from the true homeostatic point. Since more persons survive to advanced ages, and with improved risk factor profiles, the probability of an individual living to ages higher than currently observed increases.

### 2.3.3 Projections Based on Disability Dynamics

Equations (31) and (32) in section 2.2.6 represent cohort changes in disability with age and mortality after an index age (e.g., $t_0 = 65$). To project the distribution of the disabled population to a future time, multiple cohort projections are needed. Specifically, for, say, 2020 we might consider the active life expectancy (ALE) for all persons aged 65+ at that date. To do this, we need to evaluate life-table equations for a cohort aged 65 in 1990 (i.e., the 1925 birth cohort), a cohort ages 67 in 1990 (i.e., the 1923 birth cohort), and so forth, up to the oldest age (e.g., the cohort aged 115 in 1990, or the birth cohort of 1875). The cohort life-table parameters weighted by its size in 1990 can, for the appropriate age and date, be assembled to form the cross-sectional population. Specifically, cohort calculations must be made from age-specific start points in, say, 1990 and run to 2020 using equations (27) and (28). For the population aged 65 in 1990, life-table calculations (population weighted) must be run from age 65 (in 1990) to age 95 (in 2020). In 2020, the ratio $l_{95}/l_{65}$, generates the number of persons surviving to 2020. Multiplying by $\nu_{k(95)}$, the mean of scores on profile $k$, generates the number of survivors aged 95 in 2020 in each disability class. For the population aged 70 in 1990, $l_{100}/l_{70}$ generates the number of survivors to age 100 in 2020; multiplied by $\nu_{k(100)}$, this produces the number in each disability class. Similar calculations are performed for other age groups. These are summed to get the total population aged 65+ in a given disability state at a given date.

In interpreting the projections, it is important that individual membership in disability states are graded and multidimensional. This stabilizes projections but means that "counts" are sums of the $g_{ik}$, not the number of individuals with nonzero scores. If the average score among persons with nonzero $g_{ik}$ is, say, .5, then the projected count is 50 percent of the number of such persons. Alternatively, two persons, each with 50 percent disability, are equivalent to one person with 100 percent disability; i.e., counts are weighted by traits associated with each profile. Since the $k$ profiles form partitions for each individual, the sums of the $g_{ik}$ partition the projected population.

Additionally, we do not want parameters for cross-sectional life tables. We need to simulate a current cohort's future experience. Estimates from the 1982, 1984, and 1989 NLTCS suggest that the proportion of the elderly population remaining nondisabled increased. The population aged 65+ grew 10 percent from 1984 to 1989, while the disabled population grew 6.8 percent (Manton, Corder, and Stallard 1992). The problem is to modify $\mathbf{C}_t$ to reflect a reasonable cohort scenario. The scenario is implemented by altering sample weights to reflect assumptions about cohort disability changes. In our cohort scenario, we assumed that half of 80 percent of the transitions to a disability state from the nondisabled, screened population were prevented and that two-thirds of 20 percent of that population with changes had disability prevented. The two adjustments, implemented by adjusting sample weights, imply 53 percent of the disability occurring in the younger nondisabled population is prevented. By imposing interventions in the screened, nondisabled population we simulated the prevention of disability in a population that (*a*) is younger than the NLTCS (Medicare-eligible elderly disabled) population on average, (*b*) has not had disability for a long time, i.e., it must be newly incident, and (*c*) tends to be at a relatively low level of disability. This produces a life table that matches the Social Security Administration (SSA) cohort life expectancy projected for persons age 65 in 1984 (Social Security Administration [SSA], 1983). Since the second NLTCS was done in 1984, the scenario produced results very close to the SSA projections. The cohort life expectancies also match period life tables projected by the SSA (1989) for the approximate midpoint (i.e., 2005) of the projection interval. The intervention produced dynamics ($\mathbf{C}_t$) (see eq. [32]) consistent with the monthly disability dynamics estimated from the Medicare component of the Social/Health Maintenance Organization (S/HMO) evaluation. Thus, the scenario accurately reflects short-term disability changes.

Projections can be altered by modifying the $g_{ik \cdot t}$ and generating new $\mathbf{C}_t$. Thus, the effects of disease intervention on disability may be forecast. In the projections, we did not change disease prevalence, but we assumed that the income and education distribution for persons aged 65–69 would be applied for persons at all ages in the cohort life table.

Table 2.7 contains sex-specific life-table parameters for selected ages for (*a*) cohort simulations, (*b*) the forecasting model calibrated with the 1982, 1984,

**Table 2.7** **Simulation, Baseline Cohort Life Tables, and Age-Specific Meaning $g_{ix} \times 100$**

| Age | | $l_x$ | $\Delta C/S$[a] $\Delta I/S$ | $e_x$ | Profiles 1 | 2 | 3 | 4 | 5 | 6 | Institutional |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | *Males* | | | | | | |
| 65 | Simulation | 100,000 | | 15.4 | 92.7 | 0.7 | 1.1 | 0.8 | 2.2 | 1.5 | 1.0 |
| | Cohort | 100,000 | 0.0% | 15.6 | 92.2 | 4.0 | 0.7 | 0.7 | 1.1 | 1.0 | 0.3 |
| | Income and education | 100,000 | 0.0% | 16.8 | 92.7 | 0.7 | 1.4 | 0.9 | 1.9 | 1.7 | 0.7 |
| 75 | Simulation | 66,272 | | 10.7 | 91.6 | 1.3 | 1.1 | 0.7 | 1.9 | 1.7 | 1.8 |
| | Cohort | 68,956 | 4.1% | 10.3 | 85.4 | 8.5 | 0.8 | 71.1 | 1.8 | 1.3 | 1.2 |
| | Income and education | 68,958 | 4.1% | 12.2 | 90.2 | 2.1 | 1.2 | 0.6 | 2.4 | 2.0 | 1.5 |
| 85 | Simulation | 32,587 | | 6.6 | 78.5 | 4.6 | 2.0 | 1.7 | 3.9 | 4.2 | 5.1 |
| | Cohort | 32,886 | 1.0% | 6.1 | 73.1 | 12.1 | 1.3 | 2.6 | 3.5 | 2.4 | 5.1 |
| | Income and education | 37,374 | 14.7% | 8.1 | 75.7 | 6.2 | 2.1 | 1.5 | 5.5 | 4.8 | 4.2 |
| 95 | Simulation | 7,061 | | 4.4 | 65.3 | 6.0 | 2.7 | 1.4 | 6.5 | 7.3 | 10.9 |
| | Cohort | 6,262 | −11.3% | 3.2 | 56.8 | 12.6 | 1.6 | 5.6 | 6.6 | 5.8 | 11.1 |
| | Income and education | 10,894 | 54.3% | 5.8 | 63.6 | 7.4 | 2.9 | 1.3 | 7.9 | 8.1 | 8.9 |
| 105 | Simulation | 655 | | 3.5 | 68.1 | 6.0 | 2.7 | 1.3 | 6.3 | 6.6 | 9.0 |
| | Cohort | 198 | −69.8% | 2.2 | 50.0 | 19.1 | 0.2 | 4.8 | 12.6 | 3.3 | 10.0 |
| | Income and education | 1,780 | 171.8% | 4.8 | 65.2 | 7.3 | 2.9 | 1.2 | 7.9 | 7.7 | 8.0 |

| | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | | | *Females* | | | | | | |
| 65 | Simulation | 100,000 | | 20.5 | 91.2 | 0.9 | 1.7 | 2.2 | 1.4 | 1.4 | 1.1 |
| | Cohort | 100,000 | 0.0% | 20.6 | 92.1 | 3.4 | 2.0 | 0.7 | 1.2 | 0.8 | 0.8 |
| | Income and education | 100,000 | 0.0% | 23.7 | 91.8 | 0.9 | 1.7 | 1.7 | 1.5 | 1.3 | 1.2 |
| 75 | Simulation | 80,560 | | 14.2 | 87.6 | 1.5 | 2.3 | 1.8 | 2.7 | 1.7 | 2.5 |
| | Cohort | 82,201 | 2.0% | 13.9 | 84.0 | 7.8 | 1.3 | 1.1 | 2.2 | 1.4 | 2.2 |
| | Income and education | 84,340 | 4.7% | 17.3 | 83.4 | 3.7 | 2.6 | 1.6 | 3.3 | 3.2 | 2.2 |
| 85 | Simulation | 53,931 | | 8.5 | 68.8 | 4.4 | 3.6 | 2.5 | 5.3 | 4.0 | 11.5 |
| | Cohort | 54,235 | 0.6% | 8.4 | 65.1 | 12.1 | 2.2 | 2.7 | 5.5 | 3.4 | 9.0 |
| | Income and education | 59,797 | 10.9% | 11.9 | 65.1 | 7.2 | 3.0 | 2.0 | 6.1 | 7.0 | 8.0 |
| 95 | Simulation | 18,192 | | 5.5 | 46.8 | 8.2 | 4.1 | 3.1 | 5.9 | 8.3 | 23.4 |
| | Cohort | 18,757 | 3.1% | 4.9 | 48.8 | 10.6 | 1.8 | 4.6 | 5.8 | 6.1 | 22.4 |
| | Income and education | 26,725 | 46.9% | 8.9 | 52.2 | 10.1 | 3.7 | 2.5 | 5.7 | 11.8 | 14.0 |
| 105 | Simulation | 2,916 | | 4.5 | 49.6 | 8.5 | 4.3 | 3.2 | 6.1 | 8.6 | 19.7 |
| | Cohort | 2,246 | −23.0% | 3.6 | 50.0 | 9.9 | 1.2 | 4.4 | 4.9 | 5.0 | 24.5 |
| | Income and education | 8,111 | 178.2% | 7.5 | 53.2 | 10.7 | 3.9 | 2.6 | 5.8 | 12.3 | 11.6 |

*Source:* 1982 and 1984 NLTCS.

[a]For each pair of numbers in this column, the top is the percentage increase in survival ($l_y$) for 1982–89 cohort relative to simulation, and the bottom is the percentage increase in survival for income and education adjustment relative to simulation.

and 1989 data, and (*c*) the cohort simulation with the income and education distribution adjusted.

The six profiles are augmented with an "Institutionalized" group to represent the entire U.S. Medicare-eligible population aged 65+. Life expectancy ($e_t$) for the cohort simulation is higher at age 65 than in the 1986 U.S. cross-sectional life tables produced by the U.S. Bureau of the Census (Spencer 1989; e.g., for females, 20.5 years vs. 19.0 years, and for males, 15.4 years vs. 14.8 years). Mortality decreases at later ages (from that observed in the period life table) because nondisabled persons have lower mortality. Overall, the life expectancy is nearly identical to that of the 1919 cohort (i.e., persons aged 65 in 1984) life tables prepared by the SSA (i.e., for males, 15.3 vs. 15.4 years, and for females, 20.6 vs. 20.5 years; SSA 1983). In table 2.7 we also present life tables calculated using the declines in disability observed from 1982 to 1989 (with mortality followed from 1982 to 1991; with Bayesian unit weights applied to each year's sample). The projected life expectancy at age 65 is again close for males (i.e., 15.4 vs. 15.6 years) and females (20.5 vs. 20.6 years). The simulations are similar to the 1982–89 life tables for males to age 85 and for females to age 95. The fact that the simulation provides a higher life expectancy at later ages than the 1982–89 data is because (a) the 1982–89 life tables do not reflect disability declines after 1989 and (b) since we weighted each survey year equally (a conservative approach), the θ for the 1982–84 interval is smaller because of the shorter interval (i.e., $\theta_M = 4.0$ percent and $\theta_F = 3.6$ percent) than for the 1982–89 estimates (i.e., $\theta_M = 5.5$ percent and $\theta_F = 4.4$ percent). This is because there are more unobserved disability transitions in the five-year interval 1984–89. We also show the effect of controlling income and education using the simulated cohort as the base. This increases life expectancy 1.4 years for males and 3.2 years for females. The life expectancy trajectories of the three scenarios are presented in figure 2.1.

Disability represents an actual loss of function for a person, rather than a risk factor out of range. Thus, it is a better predictor of mortality (see Grand et al. 1990; Campbell et al. 1985). For example, 92 percent of the age dependence of mortality for males, and 94.5 percent for females, is explained by functional level (compared to 62 and 69 percent for the risk factors). However, disability is not only an outcome of disease processes. Often, loss of function (implying decreased activity and worsened nutrition) is an etiological factor in mortality at advanced ages; e.g., 56 percent of deaths in one autopsy series were due to CHF, pulmonary embolism, or pneumonia, all of which are stimulated by lack of activity and poor nutrition. Only recently have mechanisms underlying the effect of functioning on health been specified. It was discovered, for example, that impaired heart muscle produced enzymes down-regulating the activity of skeletal muscle to keep them within the range of activity supportable by the remaining cardiac function (Drexler 1992). Many metabolic parameters are affected by activity—even to extreme ages (e.g., age 107 in Lindsted, Tonstad, and Kuzma 1991). Likewise, higher education and higher income not only

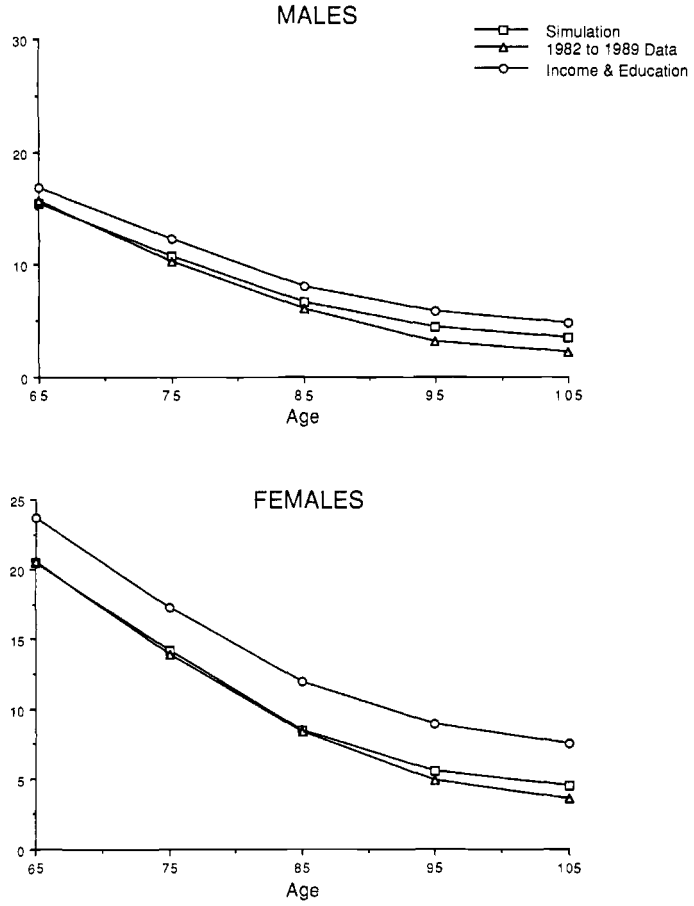


**Fig. 2.1 Life expectancy at selected ages for males and females under three different scenarios: cohort simulation, 1982–89 life tables; and income and education adjustment of cohort simulation**

imply improved access to medical care but also better lifestyle and higher expectations about health and functioning at later ages.

In addition, we can examine the distribution of frailty at each age. The value for $\bar{g}_1$ represents ALE at a given age. For males this declines more rapidly in the 1982–89 data (e.g., to 56.8 percent by age 95). For females the level of ALE is about the same at each age. In general the group that increases most rapidly in the 1982–89 data is the second group with mild cognitive impairment. For females, income and education greatly reduce the institutional population.

In table 2.8 we present cross-sectional distributions of males and females in each disability state for 1990 and 2020 and changes in the size of those populations (based on cohort simulations).

**Table 2.8** **Distribution of Persons in Each Disability State, 1990 and 2020 (thousands)**

| Age Group | Profile 1 | 2 | 3 | 4 | 5 | 6 | Institutional |
|---|---|---|---|---|---|---|---|
| | | | *Males* | | | | |
| Baseline: 1990 | | | | | | | |
| 65+ | 11,047.41 | 184.75 | 173.79 | 109.99 | 305.40 | 280.60 | 255.44 |
| 85+ | 617.95 | 40.73 | 19.36 | 11.01 | 47.49 | 44.95 | 65.50 |
| Baseline: 2020 | | | | | | | |
| 65+ | 20,490.82 | 352.32 | 316.48 | 202.72 | 592.51 | 522.48 | 545.93 |
| 85+ | 1,511.63 | 104.99 | 50.49 | 27.73 | 121.37 | 114.76 | 166.61 |
| Variance control: 2020 | | | | | | | |
| 65+ | 20,479.09 | 375.91 | 336.81 | 225.83 | 635.87 | 604.89 | 721.86 |
| 85+ | 1,416.83 | 107.02 | 52.70 | 32.35 | 129.78 | 140.02 | 258.59 |
| | | | *Females* | | | | |
| Baseline: 1990 | | | | | | | |
| 65+ | 15,228.11 | 343.02 | 437.46 | 352.48 | 513.50 | 396.42 | 722.42 |
| 85+ | 1,298.12 | 109.68 | 84.70 | 64.38 | 125.97 | 116.39 | 330.76 |
| Baseline: 2020 | | | | | | | |
| 65+ | 26,037.34 | 679.84 | 790.24 | 634.12 | 940.28 | 784.75 | 1,535.03 |
| 85+ | 2,912.99 | 298.53 | 206.44 | 157.22 | 307.05 | 323.71 | 903.86 |
| Variance control: 2020 | | | | | | | |
| 65+ | 25,915.74 | 679.66 | 803.76 | 661.15 | 959.60 | 865.16 | 2,009.09 |
| 85+ | 2,702.47 | 285.19 | 201.36 | 166.21 | 299.58 | 368.13 | 1,288.55 |

*Source:* 1982 and 1984 NLTCS.

The baseline population change from 1990 to 2020 reflects the growth of the age 85+ population. The changes for 2020 with, and without, variance control reflect increases in the size of the most disabled populations when disability heterogeneity is eliminated; i.e., this reflects what projections with discrete groups (e.g., using LCM categories, $g_{ik} = 0.0$ or 1.0) would produce. The effects are considerable at advanced ages; e.g., at ages 85+ in 2020 the institutional population increases 55 percent for males and 43 percent for females with variance control. The most fundamental problem is that age trajectories of disability are distorted by the use of homogeneous categories.

The variance control intervention shows that the effect of reduced mortality is eventually overwhelmed by mortality selection (i.e., reversing the mortality differentials). This demonstrates that the average age trajectory of risk factors (i.e., for an average individual) is not the same as the age trajectory observed for a heterogeneous population. In a population, mortality "prunes" the tails of the risk factor distribution, leaving a residual subpopulation with lower risks. The variance control intervention collapses the tails of the distribution to a single point mass, eliminating the effects of selection from the projections.

## 2.4 Discussion

We modeled mortality as a function of risk factor histories prior to death. The diffusion process describing the evolution of state variables and history-dependent mortality rates are used to forecast means and covariances of state variables and life tables and life expectancies for several scenarios. This is different from demographic forecasts (Spencer 1989; Wade 1987; Alho and Spencer 1990a, 1990b), which use only terminal-state information—i.e., age and cause of death. An advantage of the diffusion models is that one can ascertain the time scale and role of intermediate health processes prior to death. Methods that use only terminal-state information can assess interventions only when their effects on mortality are already manifest.

We also introduced a model of disability dynamics and mortality based on profiles of disabilities identified with the vertices of a unit simplex. Individual disability dynamics are represented via a diffusion process for score vectors, whose components are interpreted as the "degrees of similarity" of an individual's response to each profile of conditions. With mortality rates constructed in terms of $g_{ik}$, we produced history-dependent mortality rates which reflect physical and cognitive functioning of individuals prior to death. Ideally, both physiological and disability variables would be represented in a model. However, an integrated model, involving multiple levels of biological organization, lies in the future.

We forecast population size and health using the diffusion mortality processes with physiological variables. Changes in population produced by risk factor interventions were simulated. A stochastic limit to life expectancy was imposed by representing senescence in the mortality function. Risk factor values were not assumed to change until 2006. No change in case fatality or aging rates was assumed. Large population increases above age 85 resulted.

Federal population projections, useful for many purposes, do not make health forecasts. Models based on health processes sometimes make unrealistic assumptions, e.g., that risk factors operate independently (Tsevat et al. 1991). Multivariate stochastic process models, calibrated with longitudinal data, represent the interaction of risk factors, age, and mortality and may anticipate "turning points" that, without a model, may take years to identify (Myers 1981). Past mortality declines were presaged by risk factor changes in the population between 1960 and 1987. The projections illustrate (*a*) risk factor–based forecasts, (*b*) estimates of upper bounds to future population growth based on risk factor effects, and (*c*) variation of forecasts. They also show that Census Bureau projections are achievable by controlling known risk factors. In Census Bureau projections, however, mortality improvements are "front-end" loaded (they decline to an ultimate rate in 2012). We assumed no improvement in the first 20 (or 30) years.

The risk factor projections also suggest that uncertainty in the growth of the U.S. elderly population and changes in its health are greater than currently

envisioned. To understand how uncertainty propagates in forecasts, research is needed on: (1) integration of multiple data sources with different error structures (e.g., superpopulation sampling models; Cassel, Sarndal, and Wretman 1977), (2) data with long-term follow-up and more experience at advanced ages, (3) biologically realistic models of health processes, (4) effects of error of parameter estimates, and modes of reducing it, on forecast uncertainty, and (5) effects of functional impairment on mortality.

In addition, the analyses identified risk factor dynamics important for mortality at late ages. For example, reduction of SC and BMI at later ages may be due to a significant prevalence of malnutrition (Williams 1992). Popkin et al. (1992), in analyzing risk factor trends, showed that the population aged 65–74 responded to public health initiatives. However, those initiatives emphasize risk factor avoidance appropriate to middle-aged persons—goals that may not be optimal at later ages. What is needed are recommendations of positive actions specifically for elderly persons. This model is one way to assess the content of public health programs designed specifically for the elderly. For example, forecasts of risk factor means to very advanced ages show nonlinear trajectories due to the interaction of state dynamics and mortality. Specifically, at some advanced age the mean of a risk factor must start moving to more optimal values because of the exponential increase in the force of mortality due to unobserved factors represented by the Gompertz. When sufficient numbers of risk factors are represented that $\theta$ is "small," the cross-temporal covariances of observed risk factors will describe optimal trajectories.

Using disability assessments from the NLTCS, we produced projections of ALE for males and females. Disability was represented by scores describing multiple dimensions of disability. The use of a dynamic model with graded scores predicted a very different distribution of disability in 2020 than if discrete disability groups are used. Specifically, variance within disability categories tended to increase mortality risks—especially for the most highly disabled groups. At the same time, it reduced the average $g_{ik,t}$ for high disability dimensions, so that the mortality in later years declined. This is why variance "control" initially increased life expectancy (i.e., at ages 65 and 75) and then decreased it (i.e., at ages 85 and above).

We also investigated an intervention whereby certain disability transitions (to low levels, at early ages among persons with no prior impairment) were modified to simulate a cohort. The cohort scenario focused on short-term changes in incidence. This is an area requiring further substantive and methodological research. Specifically, the NLTCS detects disability of 90+ days duration at two points in time. To take point prevalences as fixed to calculate disability effects may overestimate the length of disability episodes and underestimate the number of persons experiencing disability. The number of short-term stays, and the likelihood of disability reversal, may be underestimated. To deal with data limitations, a model accurately describing continu-

ously changing disability states, their interaction with mortality, and time of observation is needed.

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