Trajectories of Morbidity, Disability, and Mortality among the U.S. Elderly Population: Evidence from the 1984–1999 NLTCS

Eric Stallard*

Abstract

This article employs a longitudinal form of the Grade of Membership (GoM) model to specify and estimate a multivariate model of the trajectories of morbidity, disability, and mortality among longitudinally followed elderly respondents to the National Long-Term Care Survey (NLTCS) of 1984, 1989, 1994, and 1999. A distinct trajectory was constructed for each individual respondent to the survey. The trajectories described the progressive declines over time in physical and cognitive functioning among a nationally representative sample of the U.S. elderly population.

The model was structured to represent simultaneously the essential features of the fixed frailty model of Vaupel, Manton, and Stallard and Strehler and Mildvan's model of linearly declining vitality. Unlike those models, however, the longitudinal GoM model was designed for easy and direct application to existing longitudinal data sets.

The measurement space in the NLTCS application included from one to four sets of repeated measures for each survey respondent on 95 independent variables characterizing the nature and intensity of limitations in activities of daily living (ADLs), instrumental activities of daily living (IADLs), physical functioning, and cognitive functioning, as well as indicators of behavioral characteristics, medical conditions, subjective health, age, race, sex, institutional status, and survival status.

The application showed that the model can be fitted to existing data and that the results were interpretable as generalizations of fixed frailty with linearly declining vitality.

1. Introduction

Actuaries, demographers, gerontologists, and biomedical researchers have contributed significantly to our understanding of survival at advanced ages. Further advances are possible using the extensive morbidity, disability, and mortality data currently being collected on longitudinally followed populations. Full realization of the analytic potential of such data will require new models and methods.

To date, much of the research on survival at advanced ages has been performed using the life-table and related methodologies. Methods for generalizing the life table to represent the effects of observed and unobserved covariates have been proposed. Such methods are required for fully representing the effects of large numbers of time-varying covariates in sample data from longitudinally followed populations.

This article generalizes the life table to include the effects of observed and unobserved time-varying health-related covariates using a longitudinal form of the Grade of Membership (GoM) model to specify and estimate a multivariate model of the trajectories of morbidity, disability, and mortality among

* Eric Stallard, ASA, MAAA, FCA, is Research Professor in the Department of Sociology & Center for Population Health and Aging, P.O. Box 90408, Duke University, Durham, NC 27708-0408, eric.stallard@duke.edu

elderly respondents to the National Long-Term Care Survey (NLTCS) of 1984, 1989, 1994, and 1999. This model is shown to be a natural extension of currently available methods and one that is consistent with recent research findings on individual differences in mortality and morbidity risks and the impact of those differences on survival at advanced ages.

The article contains five sections:

- ''Background'' provides information on existing life-table methods and recent research results that are used to motivate the development of the longitudinal form of the GoM model.
- ''Data'' provides information on the NLTCS and related administrative files from Medicare that constitute the inputs to the longitudinal GoM model.
- ''Methods'' describes the longitudinal GoM model, the methods used for estimating its parameters, and the application of the model to the longitudinal data in the NLTCS.
- ''Results'' presents the parameter estimates and resulting generalizations of the life table to represent trajectories of individual health histories. Validation of the model was based on internal consistency as well as comparisons with published life-table models and other data.
- ''Discussion'' considers the implications of the results in the context of existing models for analyzing and forecasting future morbidity, disability, and mortality patterns in the United States.

2. BACKGROUND

2.1 Life-Table Parameters

A life table is a tabular array of age-specific survival probabilities and associated derived annual and cumulative measures (Chiang 1984). Three fundamental parameters are

1. q_a = probability of death within one year after attaining age a
2. p_a = probability of surviving at least one year after attaining age a
3. μ_a = age-specific death rate, average hazard rate, or average force of mortality for the corresponding one-year age interval from a to $a + 1$.

The three parameters are functionally related. Parameter 2 is the complement of parameter 1—that is, the sum of the two probabilities is exactly 1. Parameter 2 can be logarithmically transformed, with a sign change, to produce parameter 3 (assuming that the average force of mortality μ_a is defined as the integral of the instantaneous force of mortality from a to $a + 1$; Manton and Stallard 1984). Moreover, $\mu_a \geq q_a$ for every age a, with approximate equality for values of μ_a below 0.05, which applies to current U.S. mortality rates up to about age 75–80 years.

2.2 Gompertz's Law

The force of mortality exhibits exponential growth above about age 20. This was discovered by Gompertz (1825), who published the famous ''law of mortality'' that now bears his name. Under Gompertz's law, $\mu_a = \alpha e^{\beta a}$, a continuous function of age, a, with proportionality constant α and growth constant β. In practice, age is discretely measured using completed years, implying that the estimate of α subsumes a factor equal to $e^{\beta/2}$. The estimate of β is unaffected by this practice, and the estimate of μ_a closely approximates the average force of mortality from a to $a + 1$.

Gompertz's law has been confirmed in numerous studies of human (and nonhuman) populations (Wetterstrand 1981; Olshansky and Carnes 1997). Two limitations were that the Gompertz ''constants'' (α and β) differed from one population to another and over calendar time and cohort within a given population. Moreover, the parameter values for α and β were negatively correlated when calculated for populations with a broad range of mortality conditions (Strehler and Mildvan 1960; Gavrilov and Gavrilova 2001).

Beginning at about 80–90 years of age (for humans; at various postreproductive ages for nonhumans), the increases in observed mortality rates are less than that predicted by Gompertz's law (Perks

1932; Olshansky and Carnes 1997; Vaupel et al. 1998), which indicates that Gompertz's law should be viewed as a useful approximation rather than an immutable fact.

Two analytic models have been proposed that yield insight into the dynamics of mortality at advanced ages: one based on linearly declining vitality (Strehler and Mildvan 1960) and the other on fixed frailty (Vaupel, Manton, and Stallard 1979).

2.3 Declining Vitality

Strehler and Mildvan (1960) proposed linearly declining vitality as the underlying physiological mechanism that explained the exponential increase found in Gompertz's law. They defined *vitality* as

> the capacity of an individual organism to stay alive, as measured by an appropriately weighted average of the maximum rate of work output (power output) less the basal power output of all of the functional modalities contributing to survival in the normal environment. (p. 15)

They noted that vitality included

> only the reserve capacity of an organism to do work in overcoming challenges to its existence. It does not include the work it must do to maintain itself in the absence of challenge. The latter, appropriately weighted, is the basal work rate. (p. 18)

To validate these assumptions, they analyzed rates of decline in reserve capacities for eight physiologic functions (nerve conduction velocity, basal metabolic rate, maximal breathing capacity, standard cell water, standard renal plasma flow, vital capacity, standard glomerular filtration rate, and a cardiac index) and found that all of the declines were linear. Assuming that the force of mortality followed Gompertz's law and that the fraction of challenges that were lethal was a negative exponential function of vitality, Strehler and Mildvan (1960, p. 17) found that the implied rate of decline in vitality was ''in reasonable agreement'' with the observed declines in reserve capacities.

Strehler and Mildvan's (1960) model was developed and tested at the population level. This limited their results in two ways:

1. They did not validate their model using individual-level data on the relationships between measures of physiologic reserve capacities and subsequent mortality rates.
2. They did not account for the impact of individual differences in initial vitality or in the rates of decline in vitality.

2.4 Fixed Frailty

Vaupel, Manton, and Stallard (1979) developed life-table methods to study the impact of unobserved but persistent individual differences in mortality risks. These differences were represented through the effects of a multiplicative factor termed *frailty* that operated, by assumption, as a fixed multiplier on a standard schedule of age-specific forces of mortality faced by individuals in each birth cohort. Without loss of generality, average frailty was defined to have the value 1 at the initial age of observation (e.g., at birth or any other age at which cohort follow-up began). Thus, using z_i to denote the frailty value for the ith individual, the model can be specified as $\mu_a(z_i) = z_i \cdot \mu_a(1)$, where $\mu_a(1)$ is the standard force of mortality at age a. Because individual frailty values were fixed for life, the distribution of frailty was assumed to be affected only by the selective effects of differential mortality at the different levels of frailty.

The meaning of frailty employed by Vaupel, Manton, and Stallard (1979) differed substantially from the meaning of the same term that has been increasingly used in the gerontological literature to characterize a clinical syndrome among elderly persons that includes such characteristics as weight loss, exhaustion, grip strength, walking speed, and physical activity (e.g., see Fried et al. 2001, 2004; Bortz 2002). This use of the term may be closer to the concept of low vitality, as implied by Strehler and Mildvan's (1960) model of declining vitality. The concept of frailty proposed by Vaupel et al. was one in which frailty was a fixed characteristic of an individual; this was synonymous with a concept of relative susceptibility to death.

Part of the motivation for the fixed frailty model was to determine if the slowing of the rate of increase in the force of mortality observed at the oldest ages in a population was an artifact of differential mortality selection on a population in which the forces of mortality faced by individuals continued to increase according to Gompertz's law.

Vaupel, Manton, and Stallard (1979) assumed that frailty was gamma distributed. Hougaard (1984) proposed that the inverse Gaussian distribution be considered as an alternative to the gamma. Manton, Stallard, and Vaupel (1986) fitted gamma and inverse Gaussian distributions to U.S. death rates at ages 65–94, using both Gompertz and Weibull models for the standard schedule of age-specific forces of mortality faced by individuals in each birth cohort. The estimates of the standard deviation of frailty were in the range 0.3–0.8 and were larger for females than males, larger for the Gompertz than the Weibull model, and larger for the inverse Gaussian than the gamma distribution. However, because the mathematics of the mortality selection process ensured that the standard deviations were fixed over age for the gamma but declined over age for the inverse Gaussian, the standard deviations for the two distributions converged to equality at age 89 for males and age 93 for females, after which the standard deviations crossed over.

One major limitation of the analysis in Manton, Stallard, and Vaupel (1986) was the need to assume a parametric form such as the Gompertz or Weibull for the standard schedule of age-specific forces of mortality faced by individuals in each birth cohort. Because Strehler and Mildvan (1960, p. 19) argued that their theoretical model applied to individuals, the assumption of a Gompertz function for individual forces of mortality in Manton et al. was consistent with their model. If that assumption was wrong, however, then the estimate of the standard deviation of frailty could be seriously in error.

To test this assumption, one must apply the model to data for which the functional form of the individual forces of mortality is not restricted to any specific parametric family. Such data were available to Iachine et al. (1998), who applied a correlated gamma-frailty model to survival data on Danish, Swedish, and Finnish twins. They found that the standard schedules of age-specific forces of mortality faced by individuals in each birth cohort increased substantially faster than the Gompertz function and that the standard deviation of frailty was about 1.5, which was about 3 times larger than the comparable estimates in Manton, Stallard, and Vaupel (1986; i.e., 0.46 and 0.54, respectively, for males and females under the gamma-mixed Gompertz formulation).

Iachine et al.'s (1998) results clearly contradicted Strehler and Mildvan's (1960) assumption that their theoretical model applied to individuals. Iachine et al.'s results also affected the interpretation of the analysis of Manton, Stallard, and Vaupel (1986), given that part of the motivation for the fixed frailty model was to determine if the slowing of the rate of increase in the force of mortality at the oldest ages in a population compared to the rate of increase predicted under Gompertz's law was an artifact of mortality selection. As an alternative to the Gompertz function, Manton et al. considered the Weibull function as a model of the forces of mortality faced by individuals. However, when fitted to the same age-specific data, the Weibull function rose slower than the Gompertz function at older ages, so that the Weibull model was likewise contradicted by Iachine et al.'s results. Manton et al. did not consider any models in which the individual forces of mortality rose faster than the Gompertz function, in part because the need for such models was not recognized at that time.

Iachine et al. (1998) found that (1) the variability in individual frailty was substantially larger than expected and (2) the individual forces of mortality rose substantially faster than expected under the assumption that the Gompertz function was an accurate representation of the age trajectories of individual forces of mortality. These findings are relevant to our goal of accurately modeling trajectories of disability and mortality for individual respondents to the NLTCS.

2.5 Genetic and Nongenetic Factors

Because their analyses were based on the survival experience of monozygotic and dizygotic twins, Iachine et al. (1998) were able to partition the variability in individual frailty into genetic and nongenetic components. They reported country-specific sex-specific estimates of ''narrow-sense'' heritability in frailty (reflecting the fraction of the variance of frailty associated with additive genetic effects) in the range 0.36–0.60 and 0.37–0.54 and pooled estimates of 0.57 and 0.51, respectively, for males and females.

Combined with other analyses by the same researchers (e.g., McGue et al. 1993; Yashin and Iachine 1995; Yashin, Iachine, and Harris 1999; De Benedictis et al. 2001), these results indicated that, for both sexes, about 50% of the variability in individual frailty was genetic. Conversely, these results indicated that about 50% of the variability in individual frailty was due to nongenetic factors.

Because individual frailty values were assumed to be fixed for life, or at least approximately so, in the fixed frailty model, the finding that nongenetic factors accounted for about half of the variability in frailty meant that the source and stability of such nongenetic factors were relevant.

Stability was relevant because the assumption that frailty was effectively fixed by early adult ages was necessary for the effects of mortality selection to become manifest at older ages. That is, if individual differences in frailty do not persist, then the excess risks faced by any given individual at one set of times would tend to be canceled out by reduced risks at later times. Over time, no cumulative advantage or disadvantage would be experienced by such individuals; hence, the individual and population forces of mortality would be the same, on average, and the impact of nonpersistent individual differences in frailty would be trivial.

The sources of the nongenetic factors are relevant to our understanding of these factors, their stability over time, the persistency of their effects, and the potential for interventions that could mitigate their deleterious effects.

Important insight into the nature of the nongenetic factors was provided by McGinnis and Foege (1993), who introduced the concept of ''actual causes of death,'' which were 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 the standard concept of ''underlying causes of death'' through published relative risk estimates. According to these authors, approximately 50% of all deaths in the United States in 1990 were attributable to the nine factors studied.

Using similar methods, Mokdad et al. (2004) updated that study and found that 48% of all deaths in the United States in 2000 could be attributed to the same nine factors. 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 and high serum cholesterol. Moreover, they provided no information on morbidity and disability, which are important to the current analysis. Despite these limitations, the results of their analyses were consistent with the expectation from the twin studies that about 50% of the variation in frailty, and hence in mortality, was due to nongenetic factors. Also, the cumulative nature of the damage associated with the various lifestyle and risk factor behaviors could support an assumption that the resulting mortality differentials were stable over time.

2.6 Life Tables with Covariates

Hazard-rate (force of mortality) regression models with observed covariates are often specified using Cox's (1972) regression model. This model is similar to the fixed frailty model except that an exponential function of the covariates operates as a fixed multiplier on the age-specific standard force of mortality. For example, using $\mathbf{x}_i$ to denote the vector of covariates for the ith individual, the model can be specified as $\mu_a(\mathbf{x}_i) = \exp(\mathbf{x}_i'\mathbf{b}) \cdot \mu_a(\mathbf{0})$, where $\mu_a(\mathbf{0})$ is the standard force of mortality at age a, that is, resulting from the condition $\mathbf{x}_i = \mathbf{0}$. Moreover, because $\mu_a(\mathbf{0})$ does not appear in the partial likelihood equations used to estimate the regression parameters, it is not necessary to specify the functional form of the standard force of mortality. Nonparametric estimates of $\mu_a(\mathbf{0})$ can be generated after the regression parameters have been estimated.

Various generalizations of Cox's (1972) regression model have been proposed to allow estimation and testing of the hazard-rate regression coefficients and standard forces of mortality with time-varying covariates (e.g., see Therneau and Grambsch 2000). A common approach is to conduct estimation and testing conditional on the observed sequences of covariate values obtained from all individuals in the sample. This approach neither requires, nor uses, an explicit model of the processes governing the changes in the covariate values.

When the changes in the covariates are a major focus of the analysis, then an explicit model of the processes governing the temporal changes in the covariate values is required. In this case either the Cox regression model must be embedded within a more general covariate model or an alternative model must be developed.

Manton et al. (1994a) presented a two-component Gaussian stochastic process model describing (1) the changes in the observable covariates over age and (2) the impact of the age-varying covariates on the individual forces of mortality. Implementation of the model followed the specifications given in Woodbury and Manton (1977) and Tolley and Manton (1991), viz., linear dynamics of the covariates and a quadratic form for the force of mortality.

The changes in the vector of covariate values, $\mathbf{x}_{ia}$, for the ith individual from age a to $a + 1$ were modeled as a first-order autoregressive process, as follows:

$$\mathbf{x}_{i(a+1)} = \mathbf{u}_a + \mathbf{A}\mathbf{x}_{ia} + \mathbf{e}_{i(a+1)}, \tag{2.1}$$

where $\mathbf{u}_a$ was a vector of age-specific constant changes, $\mathbf{A}$ was a regression matrix representing the effects of $\mathbf{x}_{ia}$ on $\mathbf{x}_{i(a+1)}$, and $\mathbf{e}_{i(a+1)}$ was a vector of normally distributed residual values (i.e., errors or innovations) at age $a + 1$. Estimation of $\mathbf{u}_a$ and $\mathbf{A}$ was generally conducted via ordinary least squares procedures. One exception was the application of the model to GoM scores from the NLTCS, for which estimation of $\mathbf{A}$ (actually $\mathbf{A}_a$) was conducted via the minimum entropy algorithm described in Manton et al. (1992b, pp. 326–29).

The hazard function was modeled as an age-specific quadratic function of the current covariates:

$$\mu_a(\mathbf{x}_{ia}) = \mu_a(\mathbf{0}) + \mathbf{b}_a'\mathbf{x}_{ia} + \frac{1}{2}\mathbf{x}_{ia}'\mathbf{B}_a\mathbf{x}_{ia}, \tag{2.2}$$

with parameters $\mu_a(\mathbf{0})$, $\mathbf{b}_a$, and $\mathbf{B}_a$, where $\mu_a(\mathbf{0})$ was the standard force of mortality at age a, that is, resulting from the condition $\mathbf{x}_{ia} = \mathbf{0}$.

To facilitate estimation of the quadratic hazard, it was necessary to impose two types of constraints. One set of constraints was needed to ensure that the quadratic form was nonnegative definite. The second set was needed to reduce the number of parameters to a manageable number. This was done by replacing each age-specific term in the quadratic hazard with an exponential function of age, as follows:

$$\mu_a(\mathbf{0}) = \mu(\mathbf{0})e^{\beta a}, \tag{2.3}$$

$$\mathbf{b}_a = \mathbf{b}e^{\beta a}, \tag{2.4}$$

$$\mathbf{B}_a = \mathbf{B}e^{\beta a}, \tag{2.5}$$

where the exponential growth constant, β, was the same for all three types of parameters (see Manton, Stallard, and Singer 1992a). The expression for $\mu_a(\mathbf{0})$ was identical to Gompertz's formula with the substitution $\alpha = \mu(\mathbf{0})$. Substitution for $\mu_a(\mathbf{0})$, $\mathbf{b}_a$, and $\mathbf{B}_a$ in the expression for $\mu_a(\mathbf{x}_{ia})$ yielded

$$\mu_a(\mathbf{x}_{ia}) = \left(\mu(\mathbf{0}) + \mathbf{b}'\mathbf{x}_{ia} + \frac{1}{2}\mathbf{x}_{ia}'\mathbf{B}\mathbf{x}_{ia}\right)e^{\beta a}, \tag{2.6}$$

a multidimensional generalization of Gompertz's law with the substitution $\alpha = \mu(\mathbf{0}) + \mathbf{b}'\mathbf{x}_{ia} + \frac{1}{2}\mathbf{x}'_{ia}\mathbf{B}\mathbf{x}_{ia}$ reflecting multiplicative and interaction effects of observable age-varying covariates. The resulting expression allowed the individual forces of mortality to rise as fast as, faster than, or slower than the Gompertz function, given that the individual forces of mortality were controlled by (1) changes in $\mathbf{x}_{ia}$, which can move the individual up or down the sides of the quadratic function, and (2) the exponential growth constant, β, which increased the height of the quadratic function by a constant factor at each age.

Manton et al. (1994a) found that the exponential growth constant, β, was reduced substantially when covariates were included in the model. Moreover, in comparing the performance of functional status measures from the NLTCS with cardiovascular disease risk factors from the Framingham Heart Study, the authors found that the functional status measures accounted for higher proportions of the variance in mortality risks (79–87% vs. 70–71%) and produced substantially greater reductions in the exponential growth constant, β, than did the cardiovascular disease risk factors (35–48% vs. 14–19%).

As the exponential growth constant declined toward 0, one would have greater confidence that the changes in the covariates accounted for changes in mortality over age, that is, because the movement of the covariates to high risk values would be the only mechanism in the model that could account for the increase in mortality risks among affected individuals.

One limitation of the two-component Gaussian stochastic process model was that the changes in the observed covariates were represented in equation (2.1) as an autoregressive process with individual covariate values tending to regress toward age-specific cohort mean values rather than to individually estimated covariate trajectories. This limitation increases in importance if the analysis is directed toward individual level survival, not population or cohort survival. However, generalizations of the model to represent individually estimated covariate trajectories are yet to be done.

A second limitation of the two-component stochastic process model was that the residual values in equation (2.1) were specified as a Gaussian diffusion process (Woodbury and Manton 1977). Application of this model to more general forms of covariate changes required ad hoc procedures. In the case of Manton et al.'s (1994a) analysis of the GoM scores from the NLTCS, ad hoc constraints had to be imposed on (1) the regression matrix $\mathbf{A}$, to maintain constraints on the sums and the signs of the GoM scores, and (2) the covariance matrices of the elements of $\mathbf{e}_{i(a+1)}$, to maintain the restricted ranges of the variances and covariances of the GoM scores.

The remainder of this article presents a new approach to calculating individually estimated covariate trajectories based on the specification of an explicit temporal structure for the GoM model. The approach is substantially simpler and easier to apply than the prior approach, which adapted the two-component Gaussian stochastic process model to the analysis of the changes in the GoM scores estimated from each wave of the NLTCS. Moreover, with fixed covariate trajectories, the approach logically follows as an integration and generalization of the fixed frailty and the declining vitality models.

3. DATA

The longitudinal GoM model was estimated using data from the second through fifth waves of the National Long-Term-Care Survey, which was conducted in 1982, 1984, 1989, 1994, 1999, and 2004. The second through fifth waves (1984–99) contained longitudinal and cross-sectional data on a nationally representative sample of 36,992 U.S. elderly persons (15,102 males and 21,890 females) who were enrolled in Medicare and were aged 65 years or older at some point during 1984–99. The sixth wave initiated field operations in late 2004 and was not available for this analysis.

Data from the four waves were pooled and organized according to the age at which an individual first completed the NLTCS interview (defining that individual's first observation). Each individual could be observed a maximum of one to four times depending on the date of the first observation (for first observation in 1999, the maximum was one time; for first observation in 1994, two times; for 1989, three times; and for 1984, four times) and whether the individual was alive or dead at each successive observation date. The distribution of the sample by attained age, observation number, and vital status at follow-up is shown in Table 1.

The 36,992 respondents were observed a total of 82,324 times. All respondents in the current analysis had complete interviews at the time of their first observation; subsequent observations shown in Table 1 included respondents who were alive at the date of the next survey, even if they did not complete an interview at that time. This was necessary to account for selected respondents aged 70–74 years for whom second observation interviews were omitted by design. It also ensured that subsequent nonrespondents provided at least one complete interview.

In addition to the observations for surviving respondents, 17,030 deaths (7,234 males and 9,796 females) were recorded during the 1984–99 study period, with 6,800 deaths occurring before the scheduled date of the second observation, 6,165 occurring after the second observation but before the scheduled date of the third observation, and 4,065 occurring after the third observation but before the scheduled date of the fourth observation. Deaths occurring after the 1999 survey are not shown in Table 1.

The NLTCS covered both institutionalized and noninstitutionalized persons. The wave-specific sample sizes during 1984–99 ranged from 17,286 to 22,139 persons, with from 3,112 to 4,954 persons disabled and living in the community, and 1,036 to 1,764 persons in institutional residence.

The response rates were excellent for the first five waves of the survey (95–97%: see Manton, Corder, and Stallard 1993, 1997; Manton and Gu 2001; Freedman et al. 2004). All institutionalized respondents were designated for a detailed interview except in 1982. A screener interview targeted noninstitutionalized disabled respondents for further study using a detailed community interview. At the time of each survey, a replenishment sample of 5,000–5,500 persons who attained their 65th birthday in the period following the prior survey was added to the surviving sample to replace the deaths occurring since the prior survey and to ensure that the new sample was representative of the entire elderly population aged 65 years or older.

Table 1

Distribution of 1984–1999 NLTCS Sample by Attained Age, Observation Number, and Vital Status at Scheduled Date of Follow-up

Attained Age	Observation Number 1	2	3	4	Total
	Alive				
65–69	20,549				20,549
70–74	6,717	14,024			20,741
75–79	3,941	5,013	8,856		17,810
80–84	2,656	2,807	2,806	4,129	12,398
85–89	1,639	1,596	1,576	1,652	6,463
90–94	676	789	657	670	2,792
95–99	697	221	222	163	1,303
100–104	117	78	36	37	268
Total	36,992	24,528	14,153	6,651	82,324
	Dead				
65–69					
70–74		1,922			1,922
75–79		1,097	2,046		3,143
80–84		1,063	1,249	1,425	3,737
85–89		1,048	1,193	1,099	3,340
90–94		843	932	884	2,659
95–99		454	562	480	1,496
100–104		373	183	177	733
Total		6,800	6,165	4,065	17,030

Source: Author's calculations based on data from the NLTCS.

The NLTCS provided data on age, sex, race, residence type (community vs. institutional), height, weight, alcohol and cigarette use, exercise, 30 major medical conditions, vision, subjective health status, seven activities of daily living (ADLs), nine instrumental activities of daily living (IADLs), seven functional limitation items (Nagi 1976), cognitive status (Short Portable Mental Status Questionnaire [SPMSQ] 1984–94; Mini–Mental State Examination [MMSE] 1999), short-term memory (1999), and aberrant behaviors. A detailed listing of these items is provided in the Appendix.

The NLTCS detailed interview questionnaire items used to assess ADL limitations identified activities in which the respondent received active physical help from another person during the week prior to the interview. This provided an objective anchor against which the use of standby help or special equipment to cope with lower levels of limitation can be compared. The detailed community questionnaire also probed for activities in which help was needed but not received, so that the entire spectrum of ADL limitations was represented in the NLTCS. A subjective response occurred when the respondent reported that help was needed but not received. This latter category of limitation was not counted as an ADL limitation in either the traditional NLTCS disability classification algorithm or in the HIPAA classification algorithm (Stallard 2000, 2001; Stallard and Yee 2000). It was included as a mild disability in the five-state Markov chain models developed in the two Stallard papers and that of Stallard and Yee.

The assessment of IADL limitations was based on detailed community questionnaire items that established that the respondent cannot perform the activity because of a disability or health problem. This removed socially defined roles as reasons for not performing activities such as cooking, housework, or managing bills (Freedman and Martin 1998).

One frequent misconception about the design of the NLTCS is that the detailed community interview covered only chronically disabled respondents. This is not true. The NLTCS included 4,207 distinct respondents who received a total of 5,734 detailed community interviews in which they were classified as nondisabled at least one time during the 1984–99 study period. This group contained 2,911 respondents with exactly one such interview, 1,109 with exactly two such interviews, 143 with exactly three such interviews, and 44 with exactly four such interviews.

The size and composition of the nondisabled subgroup receiving the detailed community interview changed over time as the survey evolved. The NLTCS classification protocols in the detailed community interviews identified 980, 916, 1,806, and 2,032 respondents who were nondisabled in 1984, 1989, 1994, and 1999, respectively. The increase between 1989 and 1994 reflected the introduction in 1994 of a special ''healthy'' subsample of 922 nondisabled respondents who otherwise would not have received the detailed community interview; an additional ''healthy'' subsample of 284 nondisabled respondents was included in 1999.

Nondisabled respondents who were not included in the detailed community sample provided information only on a limited set of variables: age, sex, race, residence type, seven ADLs, nine IADLS, and survival status, with negative responses for the ADLs and IADLs. Forty-five percent of the sample (16,671 nondisabled respondents) completed the study without receiving a detailed interview, with one-third of those respondents (5,677) dying during the study period. The inclusion of 4,207 nondisabled respondents in the detailed community sample meant that responses to the full set of variables in the NLTCS were available for respondents at all levels of morbidity and disability, including nondisabled respondents.

The NLTCS had large sample sizes at ages 85 and older, ranging from 2,425 respondents in 1984 to 2,940 respondents in 1999, with a total of 10,826 observations at ages 85 and older during the 1984–99 study period. The 1994 and 1999 surveys introduced supplementary samples of the population aged 95 and older, so that the respondent sample sizes at ages 95 and older increased from 244 in 1989 to 573 in 1994 and 562 in 1999, with a total of 1,571 observations at ages 95 and older during the 1984–99 study period.

All NLTCS records were linked to Medicare vital statistics and beneficiary claims data for calendar years 1982–2002, which provided virtually complete tracking of mortality and beneficiary medical costs during and beyond the 1984–99 study period. This linkage resolved concerns about bias in mortality rates due to the 5% per wave nonresponse rate, which can be significant in traditional longitudinal designs in which respondent rosters were not linked to an administrative record system such as Medicare. This linkage also permitted assessment of the cumulative impact of nonresponse on the study population. Among the 14,153 respondents alive at the third observation, 12,778 (90.3%) had a complete screener or detailed interview; similarly, 5,846 (89.7%) of 6,651 respondents alive at the fourth observation had a complete screener or detailed interview.

Age at last birthday on the date of the NLTCS interview was queried on the interview and verified against the age computed from the Medicare vital statistics data. Discrepancies were resolved using the Medicare data, which were accurate up to ages 95–99 during the 1980s (Kestenbaum 1992) and were consistent from one survey to the next, because the computed ages were based on recorded dates of birth.

4. METHODS

4.1 Overview

This section generalizes the basic Grade of Membership technique, a categorical data procedure that allows large numbers of variables to be simultaneously analyzed (Woodbury and Clive 1974; Woodbury, Clive, and Garson 1978; Manton and Stallard 1988; Woodbury et al. 1994; Manton, Woodbury, and Tolley 1994b), to estimate individually defined covariate trajectories from longitudinal data.

The GoM technique was appropriate as the starting point for such a task because GoM generates scores for each individual person included in the analytic data set. The GoM scores were an essential building block for defining covariate trajectories. Moreover, because the GoM scores were derived from data on morbidity, disability, and other health-related characteristics, they represented individual measures of fixed frailty, and the age changes in those scores represented individual measures of declining vitality.

The GoM scores can be included in a system of linked equations to predict the probabilities of future covariate outcomes based on the observed distributions of covariates in the analytic data set. The method permits predictions of morbidity, disability, and other health-related characteristics such as the use, cost, and intensity of acute health care and long-term-care (LTC) services for the elderly.

The expressions for the age changes in GoM scores can be written in the form of an age-inhomogeneous K-state Markov chain in which the K-element vector of GoM scores for the ith individual, denoted by $\mathbf{g}_i$, with elements g_{ik}, $k = 1, \ldots, K$, represents the initial state vector for the K states.

In conventional Markov chain models, an individual occupies only one state at a time, and, with this restriction, one can say that the states are ''crisp'' (Manton, Woodbury, and Tolley 1994b).

When GoM techniques are combined with Markov models, however, two things are different. First, each GoM state is described in relation to imaginary, ideal individuals, or ''pure types.'' Second, the GoM states are not ''crisp''; instead they are ''fuzzy'' (Zadeh 1965; Singpurwalla and Booker 2004). Thus, each individual is characterized by combinations of the GoM states. The extent to which the individual is characterized by any one pure-type state is referred to as the individual's grade of membership (GoM) score for that state.

Each individual has a GoM score for each of K states; the scores fall between 0 and 1, inclusive, and they sum to 1. These constraints imply that the vector of GoM scores is a *stochastic* vector. The GoM scores, however, are not *probabilities* of membership in each of the K states; instead they are *grades* of membership. If they were probabilities, then the GoM model would simplify to a latent class model (Lazarsfeld and Henry 1968), and the initial state vector, $\mathbf{g}_i$, would be a probability distribution vector.

Differences between the GoM and latent class models are manifest when the likelihoods are compared and when the posterior probabilities of classification are considered (Manton et al. 1992b). Under the latent class model, as more information is obtained on each individual, the posterior probabilities of classification tend toward 1 for the correct class and 0 for all other classes. Under the GoM model, as more information is obtained on each individual, one obtains more precise estimates of the GoM scores without convergence to the boundary values, 0 or 1.

Geometrically, the boundary constraints on, say, a four-pure-type GoM model imply that the entire process of GoM scores occurs within (or on the boundaries of) a three-dimensional object shaped as a regular tetrahedron (i.e., triangular pyramid). The base and side faces of this object are equilateral triangles.

The temporal trajectories of the GoM scores represent paths within this regular tetrahedron along which individuals travel as they age. For example, if the vertex opposite the base corresponds to the healthiest pure type, then a typical trajectory for a healthy 65-year-old person would begin near the top vertex and gradually move toward the base as the person aged. Movement toward one or another base vertex would depend on the nature of the medical conditions and disabilities that the person acquired as well as the correspondence of each base vertex with the other pure types and the likelihood that these pure types would manifest specific medical conditions and disabilities.

Similarly, the boundary constraints on a three-pure-type GoM model imply that the entire process of GoM scores occurs within (or on the sides of) a two-dimensional object shaped as an equilateral triangle; while the constraints on a two-pure-type GoM model imply that the entire process occurs on a one-dimensional line segment. Two is the minimum number of pure types needed to model fixed frailty with declining vitality. The use of four pure types in the current analysis allows for substantial heterogeneity in initial frailty as well as in the trajectories of declining vitality.

Given the initial stochastic state vector of GoM scores, subsequent values of the stochastic state vector are obtained via matrix multiplication by a sequence of stochastic transition matrices, for which the elements in each row (assuming postmultiplication) are nonnegative and sum to 1. These operations, which maintain the constraints on the state vectors of GoM scores, are identical to the operations that constitute a conventional age-inhomogeneous K-state Markov chain.

Thus, the set of K GoM scores defines the K-element vector $\mathbf{g}_i$. Disease and disability progression are represented as changes in the GoM score vector from one wave of the NLTCS to the next as the individual ages, possibly moving from the community to an institution, developing impairments, changing behaviors, or dying.

The merging of the fixed frailty and declining vitality models is accomplished in the current article by constraining the transition matrices to have an upper-triangular form, ensuring that individual GoM scores can only move away from the healthiest and toward the more debilitated pure types as individuals age.

The remainder of this section discusses the details of the GoM model and its application to longitudinal data analysis.

4.2 Basic GoM Model

The longitudinal GoM model is most easily understood when described as an extension of the basic GoM model (Woodbury and Clive 1974).

The basic GoM model is used to analyze data for I persons, indexed by i, $i = 1, \ldots, I$, with measurements or observations on J multinomial covariates, indexed by j, $j = 1, \ldots, J$, each of which is measured with L_j distinct outcomes or response levels. The jth covariate for the ith individual is typically denoted as x_{ij}. However, given that the outcomes are discrete (or can be coded as discrete), it is more convenient to employ binary coding for the data such that $y_{ijl} = 1$ if $x_{ij} = l$ and $y_{ijl} = 0$ if $x_{ij} \neq l$. By convention, this coding rule sets $y_{ijl} = 0$ for all values of l associated with the relevant index j if the value of x_{ij} is unknown or is missing from the data, or if x_{ij} is undefined because the jth covariate was properly skipped because of the ith individual's response on a prior screening question.

From the J covariates, K latent dimensions can be identified, where K is the number of pure-type individuals or ideal states in the specified model. In the longitudinal application, K is the number of components or elements of the fixed frailty vector. Generally, $K < J/2$ is a sufficient condition for identifiability of the GoM model (Woodbury et al. 1994). Practically, K-values very much smaller than $J/2$ lead to satisfactory models (Wachter 1999).

For example, the longitudinal application in this article uses $K = 3$, 4, or 5 with $J = 95$, implying that $K \leq J/19$ (where $19 = 95/5$). For the four-pure-type model, the NLTCS data set provided 946,493 responses to support the estimation of 46,112 parameters for males and 1,625,882 responses to support 66,476 parameters for females (see Appendix). Although the number of parameters is large, the number of responses used in estimating those parameters is substantially larger by factors of 20.5 and 24.5, respectively.

Two types of parameters are estimated in basic GoM. The first are the λ_{kjl}'s, which are the *pure-type probabilities;* the second are the g_{ik}'s, which are the *GoM scores*. Both are indexed using $k = 1, \ldots, K$.

Like the y_{ijl}'s, the λ_{kjl}'s are matched to the indexes (j and l) of the responses to the J covariates. The λ_{kjl}'s for the jth covariate are multinomial probabilities, where each parameter λ_{kjl} is the probability that (only) the lth response is observed for pure type k (or for an individual i who is exactly like pure type k) on variable j, subject to the convexity constraints $0 \leq \lambda_{kjl} \leq 1$ and $\Sigma_{l=1}^{L_j} \lambda_{kjl} = 1$.

The g_{ik}'s are state variables that quantify how well each individual's observed state is described by each of the K pure-type dimensions or ideal states. An individual with $g_{ik} = 1$ for some index k is exactly like the kth pure type. The g_{ik}'s are convexly constrained scores for individuals, that is, $0 \leq g_{ik} \leq 1$ and $\Sigma_{k=1}^{K} g_{ik} = 1$ (Woodbury, Manton, and Tolley 1994).

As noted above, the binary coded 0–1 variables for the lth response to the jth variable are indicated by y_{ijl}. The fundamental equation of the basic GoM model generates the probability of each discrete outcome (i.e., the probability that $y_{ijl} = 1$ or $x_{ij} = l$) from the "continuous" state variables (g_{ik}) using an inner product form

$$\text{Prob}(y_{ijl} = 1) = \sum_{k=1}^{K} g_{ik}\lambda_{kjl}. \tag{4.1}$$

For a given set of observations, the likelihood, L, is expressed as the product over i, j, and l of the set $\{\text{Prob}(y_{ijl} = 1)\}$:

$$L = \prod_i \prod_j \prod_l \left(\sum_k g_{ik}\lambda_{kjl} \right)^{y_{ijl}}. \tag{4.2}$$

The product form of the likelihood over the index i represents the standard assumption that individuals are statistically independent. The product form over the index j represents the fundamental assumption of the GoM model that the J covariates are statistically independent conditional on the responses to any prior screening questions, and conditional on the GoM score vector $\mathbf{g}_i$. The binary coding of the y_{ijl}'s ensures that all but the one term indexed by $l = x_{ij}$ will have unit value for each observed covariate, j, and hence will not affect parameter estimation.

Parameter estimation follows Manton and Stallard (1988) or Woodbury, Manton, and Tolley (1994). Parameter estimation may be conducted using fixed-point iteration procedures to update existing estimates of g_{ik} and λ_{kjl} as follows:

$$g_{ik} \leftarrow \frac{g_{ik} \times \sum_j \sum_l y_{ijl}\lambda_{kjl}/p_{ijl}}{\sum_j \sum_l y_{ijl}} \quad \text{and} \quad \lambda_{kjl} \leftarrow \frac{\lambda_{kjl} \times \sum_i y_{ijl} g_{ik}/p_{ijl}}{\sum_{l'} \sum_i y_{ijl'} g_{ik}\lambda_{kjl'}/p_{ijl'}}, \tag{4.3}$$

where

$$p_{ijl} = \sum_k g_{ik}\lambda_{kjl}. \tag{4.4}$$

If the final values of the g_{ik}'s are known, then the formula for the λ_{kjl}'s may be used to solve for the final λ-values iteratively. Each iteration is structured so that the update computations are run for all combinations of the subscripts k, j, and l using a fixed set of λ-values, after which the λ_{kjl}'s are updated as a set.

If the final values of the λ_{kjl}'s are known, then the formula for the g_{ik}'s may be used to iteratively solve for the final g-values. Each iteration is structured so that the update computations are run for all combinations of the subscripts i and k using a fixed set of g-values, after which the g_{ik}'s are updated as a set.

If both the g_{ik}'s and λ_{kjl}'s are unknown, the formulas may be used by alternating between the g-values and λ-values.

Once the GoM score vectors, $\mathbf{g}_i$, are estimated, they can be used as supplementary input data to conditional probability models for resource utilization, costs of services, and other health-related or survival-related measures.

4.3 Longitudinal GoM Model

Under a longitudinal GoM model, each individual observed at multiple times in a longitudinal survey is viewed as following a distinct trajectory as he or she ages. The 1984–99 NLTCS provides the opportunity to evaluate 15 years of experience along these trajectories, sufficient time to characterize the trajectories for many disabled persons.

To generalize the basic GoM model for longitudinal data, one first needs to add an age (or time) index to the basic GoM score vector, $\mathbf{g}_i$, so that this vector is respecified as $\mathbf{g}_{ia}$ for individual i at age a. For the NLTCS the index a increases by five years with each new wave of the survey; for notational simplicity, however, one-year (or one-unit) age increments are used in the following development.

One way to estimate a sequence of $\mathbf{g}_{ia}$'s for individuals in the NLTCS is to apply the basic GoM model to pooled data from the available waves. In this case the observed data for each individual at each wave are jointly indexed by i and a; the observations corresponding to distinct combinations of i and a are treated as independent observations in the basic GoM likelihood function. In effect, the index i is replaced by the index pair ia in the model's equations. For the data in Table 1, this approach increases the sample size from 36,992 individuals to 82,324 observations.

This approach was used in Manton, Stallard, and Singer (1992a) and Manton et al. (1994a). In both cases follow-up assessments of the changes in $\mathbf{g}_{ia}$ over age were required to complete the analyses. This was done using a linear dynamic form for the covariate changes, as assumed in the two-component Gaussian stochastic process model. Thus, equation (2.1) was respecified as

$$\mathbf{g}_{i(a+1)} = \mathbf{A}_a \mathbf{g}_{ia} + \mathbf{e}_{i(a+1)}, \tag{4.5}$$

where $\mathbf{A}_a$ was a generalized Markov-chain transition matrix governing transitions in $\mathbf{g}_{ia}$ from age a to $a + 1$ and $\mathbf{e}_{i(a+1)}$ was a vector of randomly distributed residual values (i.e., errors or innovations) at age $a + 1$. Because the distribution of $\mathbf{e}_{i(a+1)}$ was not Gaussian, ad hoc constraints were imposed on the diffusion process (see Manton, Stallard, and Woodbury 1991).

The alternative approach used in the current article dropped the residual terms from the updates in equation (4.5), obtaining

$$\mathbf{g}_{i(a+1)} = \mathbf{U}'_{a+1}\mathbf{g}_{ia} \quad \text{or, equivalently,} \quad \mathbf{g}'_{ia} = \mathbf{g}'_{i(a-1)}\mathbf{U}_a, \tag{4.6}$$

where $\mathbf{U}_a$ is the generalized Markov-chain transition matrix governing transitions in $\mathbf{g}_{i(a-1)}$ from age $a - 1$ to a. New notation is needed to distinguish $\mathbf{U}_a$ from $\mathbf{A}_a$, because of the transposed form of the revised update equation and the change in the age index.

Specification of the longitudinal GoM model is based on the sequence of updates to $\mathbf{g}_{ia}$ from age a_0 to age a. The model defines a fixed frailty GoM vector, $\mathbf{g}_i$, as an age-invariant vector of GoM scores or, equivalently, as an initial vector of GoM scores at age a_0. It follows that

$$\mathbf{g}'_{ia} = \mathbf{g}'_i \left\{ \prod_{\alpha=a_0}^{a} \mathbf{U}_\alpha \right\} = \mathbf{g}'_i \mathbf{V}_a, \tag{4.7}$$

where

$$\mathbf{V}_a = \prod_{\alpha=a_0}^{a} \mathbf{U}_\alpha, \tag{4.8}$$

with $\mathbf{U}_{a_0} = \mathbf{I}$, an identity matrix. The model uses the $\mathbf{V}_a$-matrices to represent declining vitality.

Operationally, the inner product form in the fundamental equation (4.1) of the basic GoM model is replaced with a bilinear form:

$$\text{Prob}(y_{ijla} = 1) = \mathbf{g}_i' \left\{ \prod_{\alpha=a_0}^{a} \mathbf{U}_\alpha \right\} \boldsymbol{\lambda}_{mjl} = \mathbf{g}_i' \mathbf{V}_a \lambda_{mjl}, \tag{4.9}$$

where the vector subscript m is used to index the combination (j, l), and the subscript a is used to index age. The likelihood is the product over i, j, l, and a of the set $\{\text{Prob}(y_{ijla} = 1)\}$:

$$L = \prod_i \prod_j \prod_l \prod_a \left(\mathbf{g}_i' \left\{ \prod_{\alpha=a_0}^{a} \mathbf{U}_\alpha \right\} \boldsymbol{\lambda}_{mjl} \right)^{y_{ijla}}. \tag{4.10}$$

Parameter estimation for the g-values and λ-values follows the fixed-point iteration procedures provided above in equations (4.3)–(4.4). To implement those procedures, one needs to rewrite equation (4.9) in an inner product form, isolating either the g-parameters or the λ-parameters as follows:

$$\text{Prob}(y_{ijla} = 1) = (\mathbf{g}_i' \mathbf{V}_a) \boldsymbol{\lambda}_{mjl} = \mathbf{g}_{ia}' \boldsymbol{\lambda}_{mjl} \tag{4.11}$$

and

$$\text{Prob}(y_{ijla} = 1) = \mathbf{g}_i' (\mathbf{V}_a \boldsymbol{\lambda}_{mjl}) = \mathbf{g}_i' \boldsymbol{\lambda}_{mjla}, \tag{4.12}$$

where $\boldsymbol{\lambda}_{mjla}$ is a vector of "adjusted" λ-parameters derived from the "unadjusted" λ-parameters as follows:

$$\boldsymbol{\lambda}_{mjla} = \mathbf{V}_a \boldsymbol{\lambda}_{mjl}. \tag{4.13}$$

For consistency, $\mathbf{g}_{ia}$ and $\mathbf{g}_i$ are termed "adjusted" and "unadjusted" GoM score vectors. In both cases the adjustment involves multiplication by the corresponding $\mathbf{V}_a$-matrix to produce an age-specific parameter vector.

Fixed-point iteration procedures can be implemented for the elements u_{kca} of $\mathbf{U}_a$, subject to the following convexity constraints: $0 \le u_{kca} \le 1$ and $\Sigma_{c=1}^{K} u_{kca} = 1$. The update equation for u_{kca} is

$$u_{kca} \leftarrow \frac{u_{kca} \times \sum_{t=a} \sum_i \sum_j \sum_l y_{ijlt}\, g_{ik(a-1)} \lambda_{cjl(a+1)t} / p_{ijlt}}{\sum_{c'} \sum_{t=a} \sum_i \sum_j \sum_l y_{ijlt}\, g_{ik(a-1)} u_{kc'a} \lambda_{c'jl(a+1)t} / p_{ijlt}}, \tag{4.14}$$

where

$$p_{ijlt} = \sum_k \sum_c g_{ik(a-1)} u_{kca} \lambda_{cjl(a+1)t}, \tag{4.15}$$

and $\lambda_{cjl(a+1)t}$ is the element in row c of the vector

$$\boldsymbol{\lambda}_{mjl(a+1)t} = \left(\prod_{s=a+1}^{t} \mathbf{U}_s \right) \boldsymbol{\lambda}_{mjl}. \tag{4.16}$$

The updating formulas may be used by alternating between the sets of g-values, u-values, and λ-values. All parameter updates are positive and meet the constraints of the model. Certain parameters converge to 0; these are boundary parameters that are identifiable in the fixed-point iterations as infinitesimal values. Convergence can be tested by perturbing the estimates and verifying that subsequent solutions converge to the same values. Testing convergence is important because the boundary constraints may "trap" the search algorithm at a local maximum likelihood solution. The solution in the current article was found only after establishing that the solution in an earlier version (Stallard 2005) was in fact a local maximum that was near to but not equal to the global maximum. Improvements to the estimation procedures based on modified Newton-Raphson procedures are being investigated.

4.4 Conditional GoM Model

Under a longitudinal GoM model, changes in the health statuses of individuals are described by changes over age or time of the GoM score vectors, and the utilization of resources, costs of services, and age-specific probabilities of death are described by a second model in which the GoM score vectors are assumed to be known. This second model is a conditional probability model because it depends on the first model to provide estimates of the GoM score vectors. The parameters of the conditional probability model can be estimated with a likelihood function identical in form to the likelihood function shown in equation (4.10), the only difference being that the numerical values of the g- and u-parameters are assumed to be known.

4.5 Incomplete Observations

Incomplete observations due to sample attrition and other forms of missing data are a pervasive problem in multivariate longitudinal data analysis. An incomplete observation occurs whenever a realized value, x_{ija}, for individual i for covariate j at age a is not observed or recorded in the analytic data set; in this case the data item is said to be ‘‘missing.’’

Sample attrition occurs whenever all covariate values, x_{ija}, $j = 1, \ldots, J$, are completely missing for one or more individuals i at age a. Table 1 indicates that death is a major cause of sample attrition in the NLTCS data, accounting for a loss of 46% of potential respondents at the second through fourth observation times. However, another 5% to 10% of potential respondents at each of the second through fourth observation times were completely missing for reasons other than death, and constitute unplanned missing data at those times.

The treatment of incomplete observations depends on whether or not the data are missing by design. The longitudinal GoM model readily accommodates planned missing data due to death and to various forms of survey ‘‘skip patterns,’’ and does so in a way that extends to the case where the data are missing other than by design.

Skip patterns occur when the interrogatories for given sets of questions depend on specific responses to prior ‘‘screening’’ questions. For example, the SPMSQ and MMSE questions were asked only to self-respondents in the NLTCS. If the relevant screening variable (Proxy Interview) was coded to indicate that a proxy was answering for the sample person, then all SPMSQ and MMSE questions were *coded as missing* using the binary coding rules described above (i.e., by setting $y_{ijla} = 0$ for all values of l associated with the relevant indexes j and a). Moreover, because all NLTCS surveys used either the SPMSQ or MMSE, but not both, the complementary questions were always coded as missing, by design.

Similarly, if an individual died in the interval preceding a given scheduled observation time, then all of the individual’s survey responses at that observation time were coded as missing and the relevant screening variable (Vital Status; more precisely, Retrospective 5-Year Survival Status) was coded to indicate that the individual was dead at that time, with complementary coding for individuals who were alive. It was neither necessary nor appropriate to code vital status for the first observation because all respondents were alive at that time. Hence, vital status was coded as missing at the first observation. For all observations scheduled beyond the time at which death was recorded, all survey responses and vital status were coded as missing.

If an individual died in the interval following a given scheduled observation time, then the relevant survival variable (Prospective 5-Year Survival Status) was coded to indicate that the individual died during the interval, with complementary coding for individuals who were alive at the end of the interval. The prospective 5-year survival variable was coded as missing for all observations obtained from the 1999 NLTCS because complete 5-year mortality follow-up from the 2004 NLTCS was not available for this analysis.

These coding rules implied that the information in the prospective and retrospective 5-year survival status variables was identical, the difference being that the prospective variable was linked to the GoM scores at the beginning of the interval while the retrospective variable was linked to the GoM scores at the end of the interval.

The effect of coding a covariate response as missing by setting $y_{ijl\alpha} = 0$ for all values of l associated with the relevant indexes j and α was to replace the corresponding term in the likelihood with the value 1. This was equivalent to dropping the missing covariate from the likelihood; it can be done without having to treat the missing response as an unknown parameter or having to integrate over some hypothesized distribution of missing responses (Orchard and Woodbury 1971; Manton, Woodbury, and Stallard 1994b).

The coding for skip patterns combined the screening variable with a set of dependent questions that were coded as missing for all respondents except those meeting the screening criteria. The joint probability of meeting the screening criteria and providing a given response to one of the dependent questions was represented in the likelihood as the product of the probability of meeting the screening criteria and the conditional probability of the given response, given that the screening criteria were met.

The conditional joint probability of providing a given set of responses to the set of dependent questions was represented in the likelihood as the product of the conditional probability of the each response. Multiplication of the resulting value by the probability of meeting the screening criteria yields the joint probability of meeting the screening criteria and providing the given set of responses.

Occasionally, a respondent's record failed to provide an answer to a question that should have been provided. These questions were coded as missing in the current analysis, but, because they were unplanned, there were no associated screening questions. This coding rule assumed that the missing data were randomly missing, given the values of the data that were recorded. This coding rule was applied both to sporadic missing data and to completely missing data for a given individual alive at the time of a scheduled interview.

An alternative treatment of unplanned missing data may be required if the data are not randomly missing. One approach would encode the presence/absence of a response to a given question in an auxiliary variable that defined the start of a new skip pattern with the screening criterion being the presence of a response. The affected question could then be coded as missing using the binary coding rules described above. Because this approach would add one auxiliary screening variable to the likelihood for each existing question and because there was no indication that the missing data patterns in the NLTCS were not random, the missing data in the current analysis were coded without such auxiliary screening variables.

5. Results

5.1 Model Selection

The longitudinal GoM model with three, four, and five pure types or ideal states (i.e., $K = 3$, 4, or 5) with age as the time dimension was independently estimated for 15,102 males and 21,890 females in the NLTCS using data on 95 independent variables characterizing the nature and intensity of limitations in ADLs, IADLs, physical functioning, and cognitive functioning, as well as indicators of behavioral characteristics, medical conditions, subjective health, race, institutional status, and survival status. This required a total of six sets of parameter estimates, with three sets of K-values for each sex.

The selection of three, four, and five pure types was based on prior experience in applying a related form of this model to represent the natural history of Alzheimer's disease in the period beginning immediately after the first symptoms (Kinosian et al. 2000, 2004). In the Alzheimer's application the temporal dimension was time since onset. In the present application the temporal dimension was age. The difference was that a large number of respondents in the NLTCS commenced follow-up after age 65–69, raising the possibility that the age-dependent parameters may be unstable.

Model selection was based on the likelihood ratio test statistics for $K = 1$ (the null model for a homogeneous population) versus $K = 3$, 4, or 5 using the standard χ^2 approximation (Wilks 1938). Combining the male and female results, the total χ^2 value for $K = 3$ was 601,826 with 74,790 degrees of freedom (d.f.), indicating that $K = 3$ was significantly better than $K = 1$. The incremental χ^2 value for $K = 4$ versus $K = 3$ was 85,179 with 37,416 d.f., which was statistically significant, indicating that $K = 4$ was significantly better than $K = 3$. The incremental χ^2 value for $K = 5$ versus $K = 4$ was 15,693 with 37,430 d.f., which was not statistically significant, indicating that $K = 5$ was too large. Hence, $K = 4$ was the preferred model.

All parameter estimates were constrained to lie within the closed interval [0, 1]. Self and Liang (1987) indicated that these constraints may imply more complex forms for the distributions of the likelihood ratios than derived by Wilks (1938). The additional complexity occurs when one or more parameters of the null model are on a boundary of the parameter space, which occurs frequently for models with $K > 1$. As a result, the incremental χ^2 tests described above for testing $K = 4$ or 5 versus $K = 3$ or 4 were conservatively biased with rejection regions that were smaller than they should have been (Manton, Woodbury, and Tolley 1994b).

Akaike (1974) introduced a model selection procedure that implied that the incremental χ^2 value for testing $K = 4$ or 5 versus $K = 3$ or 4 should be at least twice the degrees of freedom before rejecting the simpler and accepting the more complex model. Using Akaike's procedure, $K = 4$ was also the preferred model.

Logarithms of the likelihood ratios, χ^2 test statistics, and associated degrees of freedom for testing $K = 4$ versus $K = 1$ for the 95 variable are shown in the Appendix for males and females separately. The χ^2 test statistics were statistically highly significant at conventional levels for all 95 variables.

The independent estimation of the model by sex means that the GoM pure types are sex-specific. The pure types do not necessarily mean the same thing for males and females.

5.2 Description of the Four Pure Types

Table 2 displays selected λ-parameters and various summary measures based on the λ-parameters for the 95 variables, by pure type and sex, along with the observed and predicted marginal probabilities. In all cases the observed and predicted probabilities either match exactly or exhibit minor differences. This allows one to focus on the patterns of the various measures over the four pure types which are indexed by Roman numerals (I–IV).

Type I generally had the lowest and Type IV the highest probabilities of adverse outcomes on ADL, IADL, and cognitive impairments, functional limitations, institutionalization, and death. Type I had the highest use of alcohol and female smoking rates, but combined this with the highest rates of exercise.

Type II had the poorest subjective health status, largest number of medical conditions, highest current and prior body mass indexes (BMIs), and highest male smoking rates, but combined this with zero rates of institutionalization and mortality.

Type III was distinguished by its high mortality rates in combination with relatively good subjective health, relatively few medical conditions, no ADL or IADL impairments, low current and prior BMIs, relatively good vision and cognition, and zero rates of institutionalization.

Types III and IV both exhibited high mortality rates; however, only Type IV was subjected to high levels of physical and cognitive disability and institutionalization. Results below show that membership in Type III at older ages reflected transitions from Type I, whereas membership in Type IV at older ages reflected transitions from Types II and III.

Together these results indicate that the four pure types can be understood by pairing Types I and II at younger ages with no or low mortality and disability and then considering transitions at older ages, respectively, to Types III and IV with high mortality, but with disability only occurring for Type IV. Further insight into these dynamics can be gained by examination of the GoM scores and the transition/vitality matrix parameters.

5.3 GoM Scores and Transitions

Table 3 displays the average GoM scores for the four-pure-type model with stratifications by sex and age at initial interview. Type I had the highest average GoM scores, averaging 64.1% for males and 53.9% for females. The other types shared the remaining portion of the membership.

Table 2

λ-Parameters and Derived Summary Measures Estimated from 95 Independent Variables in the NLTCS, by Sex

Variable Name / Description	No. of Observations	Pure Type				Observed Value	Predicted Value
		I	II	III	IV		
Males							
Dead within 5 years (proportion)[1]	24,199	0.00	0.00	0.77	0.87	0.30	0.31
Nursing home resident (proportion)	27,819	0.00	0.00	0.00	0.52	0.05	0.05
Proxy interview (proportion)	8,329	0.14	0.00	0.17	0.75	0.29	0.29
Subjective health status (proportion fair / poor)	6,543	0.05	1.00	0.21	0.87	0.49	0.49
No. of medical conditions (0–30)	6,964	1.11	8.94	1.56	5.77	4.12	4.11
No. of ADL impairments (0–7)	27,814	0.00	0.59	0.00	6.12	0.64	0.64
No. of IADL impairments (0–9)	26,554	0.00	0.23	0.00	8.40	0.66	0.58
No. of functional limitations (0–7)	6,914	0.14	4.27	0.90	6.09	2.54	2.60
See well enough to read (proportion cannot)	6,965	0.03	0.10	0.16	0.61	0.21	0.20
No. of errors on SPMSQ (0–10)	4,761	0.58	0.49	1.35	5.94	1.90	1.92
No. of errors on MMSE (0–30)	1,022	1.06	3.24	4.88	20.31	4.90	4.80
Delayed word recall (proportion with <3 correct)	714	0.14	0.32	0.69	0.72	0.36	0.35
Naming animals (proportion with <5 in 1 minute)	723	0.02	0.03	0.04	0.17	0.03	0.03
No. of behavioral disturbances (0–4)	6,957	0.08	0.60	0.13	0.73	0.36	0.36
Alcohol use (proportion with any)	3,483	0.54	0.15	0.28	0.01	0.30	0.31
Cigarette use (proportion with any)	3,491	0.09	0.27	0.05	0.07	0.12	0.12
Light exercise (proportion with any)	3,364	0.97	0.90	0.96	0.10	0.80	0.81
Moderate exercise (proportion with any)	3,397	1.00	0.27	0.79	0.00	0.62	0.63
Vigorous exercise (proportion with any)	3,424	0.66	0.00	0.12	0.00	0.28	0.29
BMI at age 50 (proportion ≥ 28 kg / m^2)	3,113	0.12	0.62	0.06	0.21	0.23	0.23
BMI 1 year prior (proportion ≥ 28 kg / m^2)	3,250	0.19	0.76	0.02	0.07	0.25	0.25
Current BMI (proportion ≥ 28 kg / m^2)	3,349	0.21	0.73	0.01	0.05	0.25	0.25
Height (proportion $\geq$ 68″ (M) or 64″ (F))	3,400	0.80	0.65	0.56	0.59	0.68	0.68
Race (proportion white)	39,047	1.00	0.28	1.00	0.95	0.91	0.91
Females							
Dead within 5 years (proportion)	38,226	0.00	0.00	0.54	0.82	0.26	0.26
Nursing home resident (proportion)	43,764	0.00	0.00	0.00	0.75	0.09	0.10
Proxy interview (proportion)	17,633	0.00	0.00	0.17	0.75	0.26	0.26
Subjective health status (proportion fair / poor)	12,782	0.11	0.87	0.13	0.72	0.48	0.48
No. of medical conditions (0–30)	13,428	1.81	7.96	1.59	5.46	4.49	4.50
No. of ADL impairments (0–7)	43,753	0.00	1.77	0.00	6.31	1.09	1.09
No. of IADL impairments (0–9)	39,735	0.00	1.56	0.00	8.38	0.93	0.89
No. of functional limitations (0–7)	13,268	0.56	5.31	1.44	6.22	3.33	3.39
See well enough to read (proportion cannot)	13,413	0.02	0.16	0.18	0.65	0.23	0.22
No. of errors on SPMSQ (0–10)	10,659	0.62	0.67	1.25	7.45	2.22	2.25
No. of errors on MMSE (0–30)	2,115	1.26	3.65	6.16	21.39	5.75	5.62
Delayed word recall (proportion with <3 correct)	1,314	0.06	0.25	0.51	0.89	0.27	0.26
Naming animals (proportion with <5 in 1 minute)	1,341	0.00	0.00	0.09	0.32	0.04	0.04
No. of behavioral disturbances (0–4)	13,376	0.10	0.34	0.07	0.68	0.29	0.29
Alcohol use (proportion with any)	6,578	0.34	0.04	0.16	0.02	0.16	0.16
Cigarette use (proportion with any)	6,588	0.12	0.08	0.02	0.01	0.07	0.07
Light exercise (proportion with any)	6,291	0.97	0.71	0.83	0.09	0.73	0.73
Moderate exercise (proportion with any)	6,410	1.00	0.29	0.63	0.00	0.56	0.56
Vigorous exercise (proportion with any)	6,450	0.40	0.00	0.05	0.00	0.14	0.15
BMI at age 50 (proportion ≥ 28 kg / m^2)	5,635	0.09	0.60	0.09	0.23	0.25	0.25
BMI 1 year prior (proportion ≥ 28 kg / m^2)	5,983	0.23	0.79	0.00	0.07	0.30	0.31
Current BMI (proportion ≥ 28 kg / m^2)	6,218	0.22	0.78	0.00	0.06	0.29	0.30
Height (proportion $\geq$ 68″ (M) or 64″ (F))	6,392	0.57	0.45	0.29	0.39	0.44	0.44
Race (proportion white)	59,687	0.97	0.61	1.00	0.93	0.91	0.92

[1] Death probabilities were computed using GoM scores at both the start and end of each follow-up interval to approximate the GoM score changes during the interval; the values in this table are averages of the two sets of probabilities.
Source: Author's calculations based on data from the NLTCS.

Table 3
Average GoM Scores in Four Pure-Type Model, by Sex and Age at Initial Interview

Initial Age	No. of Respondents	Average GoM Score by Type			
		I	II	III	IV
Males					
65–69	9,194	0.667	0.111	0.189	0.034
70–74	2,863	0.589	0.140	0.196	0.074
75–79	1,472	0.667	0.167	0.166	0.000
80–84	864	0.587	0.194	0.219	0.000
85–89	416	0.566	0.188	0.246	0.000
90–94	151	0.456	0.544	0.000	0.000
95–99	122	0.513	0.487	0.000	0.000
100–104	20	0.353	0.647	0.000	0.000
Total	15,102	0.641	0.137	0.187	0.035
Females					
65–69	11,355	0.559	0.105	0.306	0.030
70–74	3,854	0.555	0.174	0.213	0.058
75–79	2,469	0.586	0.250	0.163	0.000
80–84	1,792	0.476	0.287	0.237	0.000
85–89	1,223	0.422	0.272	0.306	0.000
90–94	525	0.404	0.596	0.000	0.000
95–99	575	0.434	0.566	0.000	0.000
100–104	97	0.390	0.610	0.000	0.000
Total	21,890	0.539	0.184	0.251	0.026

Source: Author's calculations based on data from the NLTCS.

Comparisons across ages indicate that only Types I and II were represented at all ages, with noticeable discontinuities in the age trends for Types II and III at age 90–94, at which age Type III ''disappeared.'' Type IV disappeared at age 75–79, but this had minimal impact on the other pure types because of its small average score. The disappearance of Types III and IV at these ages was attributable to an identifiability problem due to the lack of observations on these individuals at younger ages in combination with the specific form of the vitality matrices ($\mathbf{V}_a$) estimated for these ages.

Table 4 summarizes the joint distribution of the GoM scores for 15 patterns based on combinations of the boundary values 0 and 1 with each other and with values in the open interval (0, 1). The table shows that 58.5% of males and 51.1% of females were exact pure type matches, that is, they had a GoM score of 1 on one of the four pure types, with Type I being the most frequent pure type, and 97.9% of males and 98.3% of females had at least one GoM score equal to zero.

The five most frequent patterns, covering 84.9% of males and 85.3% of females, were as follows:

1. An exact match to Type I
2. Combinations of Type I and II
3. An exact match to Type III
4. Combinations of Types I, II, and III
5. Combinations of Types I and III.

Exact matches to Type II and IV were relatively rare for both sexes with 2.2% each of males and females matching Type II and 0.6 and 0.5% (males, females) matching Type IV.

Positive GoM scores on Type I were estimated for 81.8% of males and 74.9% of females; the corresponding percentages for Types II–IV were, respectively, 32.9%, 35.2%, and 9.5% for males and 42.0%, 43.2%, and 8.5% for females.

Tables 5 and 6 display the age-specific upper-triangular GoM transition matrices ($\mathbf{U}_a$) and the resulting upper-triangular vitality matrices ($\mathbf{V}_a$) for the four-pure-type model for males and females, respectively. The transition matrices govern the age changes in the GoM scores in a manner identical to a conventional age-inhomogeneous K-state Markov chain: that is, the GoM scores for each individual

Table 4
Summarized Joint Distribution (%) of GoM Scores in Four Pure-Type Model, by Sex[1]

Rank	Pure Type and Value or Range[2]				Sex		Both Sexes
	I	II	III	IV	Male	Female	
1	**1**	**0**	**0**	**0**	**47.6**	**34.1**	**39.6**
2	(0, 1)	(0, 1)	0	0	11.0	17.0	14.6
3	**0**	**0**	**1**	**0**	**8.0**	**14.3**	**11.7**
4	(0, 1)	(0, 1)	(0, 1)	0	8.9	12.0	10.7
5	(0, 1)	0	(0, 1)	0	9.3	7.9	8.5
6	0	(0, 1)	(0, 1)	0	3.3	4.0	3.7
7	0	(0, 1)	(0, 1)	(0, 1)	2.5	2.5	2.5
8	**0**	**1**	**0**	**0**	**2.2**	**2.2**	**2.2**
9	(0, 1)	(0, 1)	(0, 1)	(0, 1)	2.1	1.7	1.9
10	(0, 1)	(0, 1)	0	(0, 1)	1.7	1.3	1.5
11	0	(0, 1)	0	(0, 1)	1.0	1.3	1.2
12	**0**	**0**	**0**	**1**	**0.6**	**0.5**	**0.5**
13	(0, 1)	0	(0, 1)	(0, 1)	0.5	0.5	0.5
14	(0, 1)	0	0	(0, 1)	0.5	0.4	0.4
15	0	0	(0, 1)	(0, 1)	0.5	0.4	0.4
Total					100.0	100.0	100.0
No. of Respondents					15,102	21,890	36,992

[1] Boldface font denotes exact pure types.
[2] The open interval (0, 1) excludes the boundary values 0 and 1.
Source: Author's calculations based on data from the NLTCS.

respondent to a given NLTCS interview can be arranged in a row vector that is postmultiplied by the appropriate age-specific transition matrix in Tables 5 or 6 to generate the GoM scores at the next interview.

Each age-specific vitality matrix is a cumulative product of the transition matrices for all ages up to and including the specified age. Examination of the vitality matrices shows that the "membership mass" began to move away from Types I and III at age 75–79 and, respectively, toward Type III and IV.

By age 90–94 all of the membership for males and females in Types II and III had transitioned to Type IV, making it impossible to estimate distinct GoM scores for Types II–IV for individuals whose initial interview was at this age or later. The convention used in the current analysis was to resolve this identifiability problem in favor of the lower numbered pure type, which explains the disappearance of Types III and IV at older ages in Table 3 noted above. This convention had no impact on the predictions of the model for the individual survey response probabilities.

Membership in Type I persisted to age 90–94 for males and 85–89 for females, with transitions to Type III beginning for both sexes at age 75–79. Membership in Type III at age 75–79 and older was solely a result of transitions from Type I.

These transitions produced vitality matrices at and above age 90–94, in which the only nonzero elements were in the first row and last column, with a pattern resembling the number "7" or, upon transposing the matrices, the letter "L."

Given that the transition and vitality matrices at age 65–69 were diagonal in form, by assumption, the transformation to an L-pattern was a remarkably simple outcome. Understanding the implications of this transformation can yield significant insight into the nature of the aging process.

5.4 Mortality Patterns

An important motivation for the current analysis was the ability of the longitudinal GoM model to elucidate the mechanisms by which the combination of fixed frailty and declining vitality contributed to the age-specific patterns of mortality faced by populations, and the differences between these patterns and those faced by individual members of those populations.

Table 7 displays the annual probabilities of death by age and sex. These were estimated as age-specific λ-values using the conditional form of the longitudinal GoM model with the g- and u-parameters fixed

Table 5
GoM Transition and Vitality Matrices, Four Pure-Type Model, Males

From . . .	5-Year Transition Matrix U[1] To . . . I	II	III	IV	Attained Age	Vitality Matrix V I	II	III	IV
					65–69				
I	1.000	0.000	0.000	0.000		1.000	0.000	0.000	0.000
II	0.000	1.000	0.000	0.000		0.000	1.000	0.000	0.000
III	0.000	0.000	1.000	0.000		0.000	0.000	1.000	0.000
IV	0.000	0.000	0.000	1.000		0.000	0.000	0.000	1.000
					70–74				
I	1.000	0.000	0.000	0.000		1.000	0.000	0.000	0.000
II	0.000	1.000	0.000	0.000		0.000	1.000	0.000	0.000
III	0.000	0.000	1.000	0.000		0.000	0.000	1.000	0.000
IV	0.000	0.000	0.000	1.000		0.000	0.000	0.000	1.000
					75–79				
I	0.788	0.000	0.212	0.000		0.788	0.000	0.212	0.000
II	0.000	1.000	0.000	0.000		0.000	1.000	0.000	0.000
III	0.000	0.000	0.000	1.000		0.000	0.000	0.000	1.000
IV	0.000	0.000	0.000	1.000		0.000	0.000	0.000	1.000
					80–84				
I	0.613	0.000	0.387	0.000		0.483	0.000	0.517	0.000
II	0.000	1.000	0.000	0.000		0.000	1.000	0.000	0.000
III	0.000	0.000	1.000	0.000		0.000	0.000	0.000	1.000
IV	0.000	0.000	0.000	1.000		0.000	0.000	0.000	1.000
					85–89				
I	0.705	0.000	0.295	0.000		0.340	0.000	0.660	0.000
II	0.000	0.389	0.000	0.611		0.000	0.389	0.000	0.611
III	0.000	0.000	1.000	0.000		0.000	0.000	0.000	1.000
IV	0.000	0.000	0.000	1.000		0.000	0.000	0.000	1.000
					90–94				
I	0.101	0.439	0.460	0.000		0.034	0.149	0.816	0.000
II	0.000	0.000	0.000	1.000		0.000	0.000	0.000	1.000
III	0.000	0.000	1.000	0.000		0.000	0.000	0.000	1.000
IV	0.000	0.000	0.000	1.000		0.000	0.000	0.000	1.000
					95–99				
I	0.000	0.375	0.625	0.000		0.000	0.162	0.838	0.000
II	0.000	1.000	0.000	0.000		0.000	0.000	0.000	1.000
III	0.000	0.000	1.000	0.000		0.000	0.000	0.000	1.000
IV	0.000	0.000	0.000	1.000		0.000	0.000	0.000	1.000
					100–104				
I	—	—	—	—		0.000	0.073	0.576	0.351
II	0.000	0.450	0.000	0.550		0.000	0.000	0.000	1.000
III	0.000	0.000	0.688	0.312		0.000	0.000	0.000	1.000
IV	0.000	0.000	0.000	1.000		0.000	0.000	0.000	1.000

[1] — denotes cells with average adjusted GoM scores equal to zero for which transition rates cannot be estimated.
Source: Author's calculations based on data from the NLTCS.

at the values in Tables 3, 5, and 6. To exploit the available data fully, the annual mortality probabilities were estimated from 20,428 deaths (for 8,583 males and 11,845 females) in the 18-year period following the 1984 NLTCS, rather than just for the four one-year periods following each survey (i.e., 1984, 1989, 1994, and 1999). This was done by linearly interpolating the individual GoM scores to single years of age (i.e., age at last birthday) and time, and then regrouping the annual observations into the eight quinquennial exposure-age categories shown in Table 7. Respondents aged 65–69 in 1999 were excluded from these conditional analyses because they provided only one observation to the main analysis with missing data for the five-year survival variable.

For both sexes Type IV had the highest death probabilities, and Type III ranked second. Types I and II had probabilities close to 0, indicating that these were extremely healthy types, except for males aged 95–99, where the results were less credible because of small sample sizes. For females the corresponding probabilities at age 95–99 were not estimable because the average GoM scores were equal to 0 within those cells.

Table 6

GoM Transition and Vitality Matrices, Four Pure–Type Model, Females

	5–Year Transition Matrix U[1] To . . .					Vitality Matrix V			
From . . .	I	II	III	IV	Attained Age	I	II	III	IV
					65–69				
I	1.000	0.000	0.000	0.000		1.000	0.000	0.000	0.000
II	0.000	1.000	0.000	0.000		0.000	1.000	0.000	0.000
III	0.000	0.000	1.000	0.000		0.000	0.000	1.000	0.000
IV	0.000	0.000	0.000	1.000		0.000	0.000	0.000	1.000
					70–74				
I	1.000	0.000	0.000	0.000		1.000	0.000	0.000	0.000
II	0.000	1.000	0.000	0.000		0.000	1.000	0.000	0.000
III	0.000	0.000	1.000	0.000		0.000	0.000	1.000	0.000
IV	0.000	0.000	0.000	1.000		0.000	0.000	0.000	1.000
I	0.836	0.000	0.164	0.000	75–79	0.836	0.000	0.164	0.000
II	0.000	1.000	0.000	0.000		0.000	1.000	0.000	0.000
III	0.000	0.000	0.000	1.000		0.000	0.000	0.000	1.000
IV	0.000	0.000	0.000	1.000		0.000	0.000	0.000	1.000
					80–84				
I	0.590	0.000	0.410	0.000		0.493	0.000	0.507	0.000
II	0.000	1.000	0.000	0.000		0.000	1.000	0.000	0.000
III	0.000	0.000	1.000	0.000		0.000	0.000	0.000	1.000
IV	0.000	0.000	0.000	1.000		0.000	0.000	0.000	1.000
					85–89				
I	0.611	0.000	0.389	0.000		0.301	0.000	0.699	0.000
II	0.000	0.595	0.000	0.405		0.000	0.595	0.000	0.405
III	0.000	0.000	1.000	0.000		0.000	0.000	0.000	1.000
IV	0.000	0.000	0.000	1.000		0.000	0.000	0.000	1.000
					90–94				
I	0.000	1.000	0.000	0.000		0.000	0.301	0.699	0.000
II	0.000	0.000	0.000	1.000		0.000	0.000	0.000	1.000
III	0.000	0.000	1.000	0.000		0.000	0.000	0.000	1.000
IV	0.000	0.000	0.000	1.000		0.000	0.000	0.000	1.000
					95–99				
I	—	—	—	—		0.000	0.278	0.607	0.114
II	0.000	0.924	0.037	0.039		0.000	0.000	0.000	1.000
III	0.000	0.000	0.853	0.147		0.000	0.000	0.000	1.000
IV	0.000	0.000	0.000	1.000		0.000	0.000	0.000	1.000
					100–104				
I	—	—	—	—		0.000	0.163	0.411	0.427
II	0.000	0.584	0.032	0.384		0.000	0.000	0.000	1.000
III	0.000	0.000	0.662	0.338		0.000	0.000	0.000	1.000
IV	0.000	0.000	0.000	1.000		0.000	0.000	0.000	1.000

[1] — denotes cells with average adjusted GoM scores equal to zero for which transition rates cannot be estimated.
Source: Author's calculations based on data from the NLTCS.

Table 8 displays the death probabilities after adjustment for declines in vitality, using the formula $\boldsymbol{\lambda}^{adj}_{mjla} = \mathbf{V}_a \boldsymbol{\lambda}_{mjla}$ to make the adjustment. Types II and III converged rapidly to the high death probabilities of Type IV. Type I maintained its advantage throughout the entire age range.

Figures 1 and 2 display the observed and predicted annual probabilities of death for males and females by age at last birthday at the start of the one-year follow-up period. The predicted probabilities were calculated by multiplying the age-specific probabilities in Table 8 by the corresponding age-specific average (interpolated) GoM scores. The observed and predicted probabilities are virtually identical.

Also shown in Figures 1 and 2 are the age-specific probabilities reported in the U.S. decennial life tables (USDLT) for 1989–91 (NCHS 1997), a period selected because it was close to the 1993 midpoint of the 18-year period following the 1984 NLTCS. The plotted points from the USDLT were annual probabilities of death for persons whose exact ages at last birthday at the start of the one-year follow-up period were at the midpoints of each quinquennial category. The comparisons with the observed/predicted rates indicate that the NLTCS mortality rates closely reproduced the age patterns of increase in the USDLT, which approximates Gompertz's law.

Table 7

Probabilities of Death within One Year in Four Pure-Type GoM Model, by Sex and Attained Age at Time of Exposure

Exposure Age	No. of Person-Years at Risk[2]	Annual Probability by Type[1] I	II	III	IV	Observed Probability	Predicted Probability
Males							
65–69	20,323	0.000	0.000	0.132	0.138	0.031	0.032
70–74	38,255	0.002	0.005	0.194	0.246	0.043	0.044
75–79	31,291	0.000	0.013	0.178	0.319	0.067	0.067
80–84	19,170	0.000	0.023	0.184	0.340	0.105	0.106
85–89	8,117	0.000	0.000	0.192	0.330	0.154	0.155
90–94	2,728	0.000	0.204	0.205	0.323	0.228	0.229
95–99	793	0.503	1.000	0.076	0.434	0.301	0.301
100–104	155	—	0.000	0.325	0.528	0.400	0.401
Total	120,832	0.001	0.007	0.180	0.314	0.071	0.072
Females							
65–69	25,424	0.000	0.000	0.081	0.140	0.017	0.017
70–74	52,008	0.001	0.003	0.108	0.223	0.027	0.027
75–79	48,498	0.000	0.003	0.111	0.249	0.043	0.043
80–84	35,563	0.000	0.005	0.116	0.267	0.070	0.070
85–89	20,404	0.000	0.000	0.128	0.271	0.115	0.115
90–94	9,577	0.000	0.000	0.182	0.272	0.183	0.184
95–99	3,804	—	0.446	0.000	0.388	0.264	0.264
100–104	992	—	0.000	0.148	0.499	0.325	0.324
Total	196,270	0.001	0.002	0.117	0.280	0.060	0.061

[1] — denotes cells with average adjusted GoM scores equal to zero for which probablilites cannot be estimated.
[2] Includes up to four observations per respondent; excludes respondents aged 65–69 in 1999.
Source: Author's calculations based on data from the NLTCS.

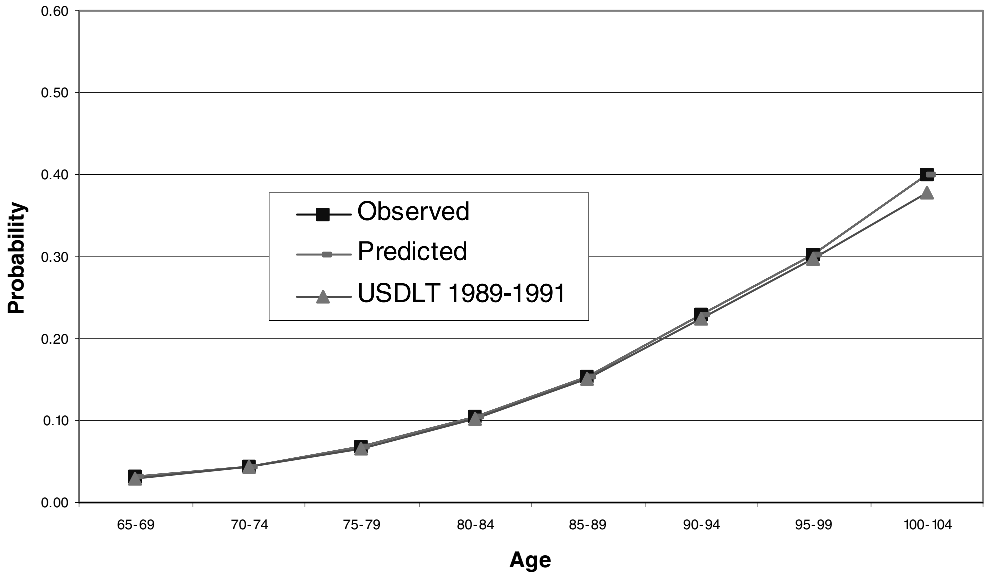
Table 8

Probabilities of Death within One Year in Four Pure-Type GoM Model, Adjusted for Declines in Vitality, by Sex and Attained Age at Time of Exposure

Exposure Age	No. of Person-Years at Risk[1]	Annual Probability by Type I	II	III	IV	Observed Probability	Predicted Probability
Males							
65–69	20,323	0.000	0.000	0.132	0.138	0.031	0.032
70–74	38,255	0.002	0.005	0.194	0.246	0.043	0.044
75–79	31,291	0.038	0.013	0.319	0.319	0.067	0.067
80–84	19,170	0.095	0.023	0.340	0.340	0.105	0.106
85–89	8,117	0.127	0.202	0.330	0.330	0.154	0.155
90–94	2,728	0.198	0.323	0.323	0.323	0.228	0.229
95–99	793	0.226	0.434	0.434	0.434	0.301	0.301
100–104	155	0.372	0.528	0.528	0.528	0.400	0.401
Total	120,832	0.041	0.033	0.253	0.271	0.071	0.072
Females							
65–69	25,424	0.000	0.000	0.081	0.140	0.017	0.017
70–74	52,008	0.001	0.003	0.108	0.223	0.027	0.027
75–79	48,498	0.018	0.003	0.249	0.249	0.043	0.043
80–84	35,563	0.059	0.005	0.267	0.267	0.070	0.070
85–89	20,404	0.089	0.110	0.271	0.271	0.115	0.115
90–94	9,577	0.127	0.272	0.272	0.272	0.183	0.184
95–99	3,804	0.168	0.388	0.388	0.388	0.264	0.264
100–104	992	0.274	0.499	0.499	0.499	0.325	0.324
Total	196,270	0.036	0.037	0.201	0.239	0.060	0.061

[1] Includes up to four observations per respondent; excludes respondents aged 65–69 in 1999.
Source: Author's calculations based on data from the NLTCS.

Figure 1
Probability of Death within One Year, Males



An exact match was not expected because the initial waves of the NLTCS oversampled the disabled population, a group that had mortality rates higher than the general population. On average, the excess mortality was about 2.9% for males and 4.4% for females. The NLTCS provided sampling weights that could be used to reweight the individual characteristics to match population characteristics exactly. However, such weights were neither needed nor used in the current analysis because the primary focus was on individual trajectories of disability and mortality, not population averages.

Figures 1 and 2 demonstrate that the averages of the individual trajectories followed known age patterns (i.e., with increases approximated by Gompertz's law) for human mortality above age 65. The

Figure 2
Probability of Death within One Year, Females

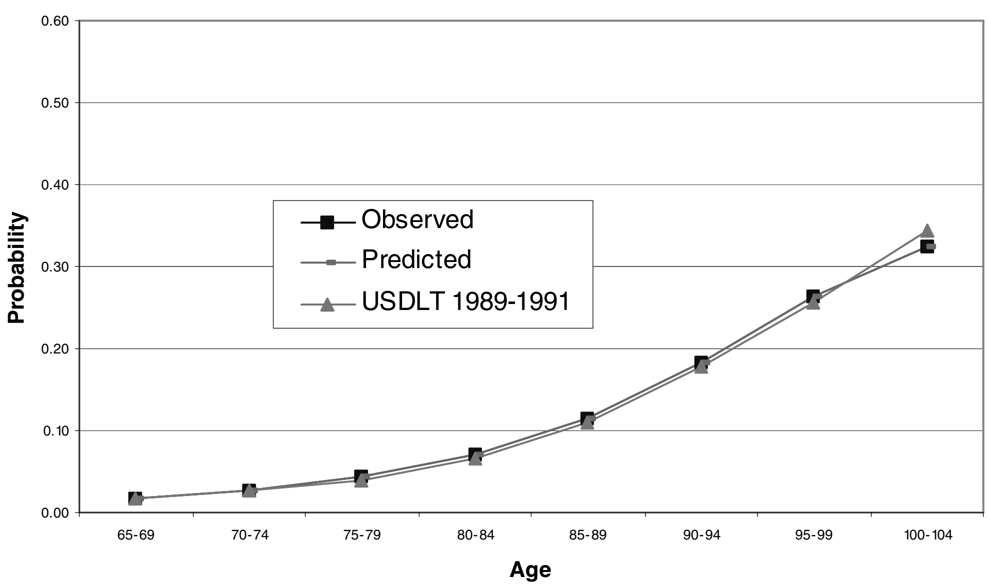
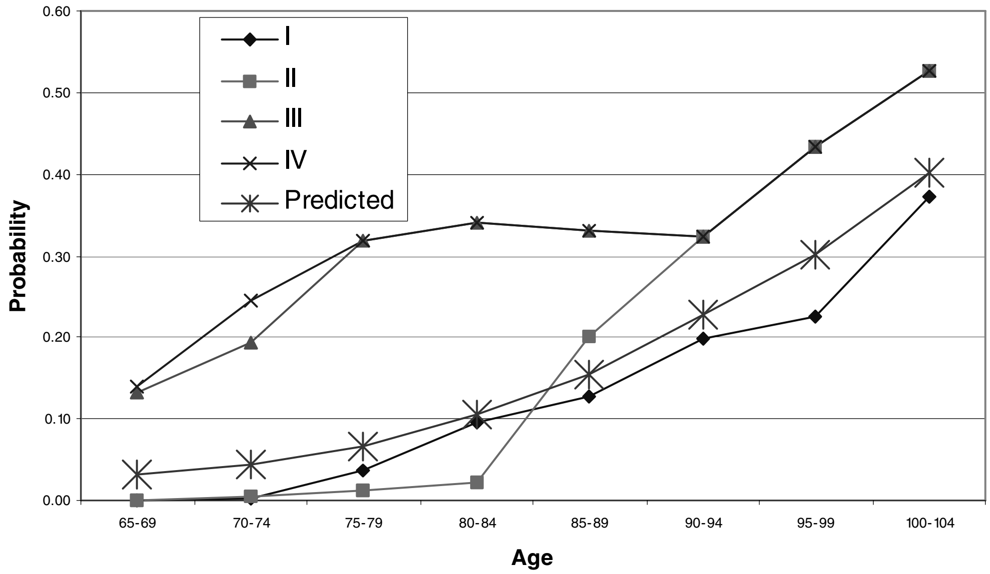


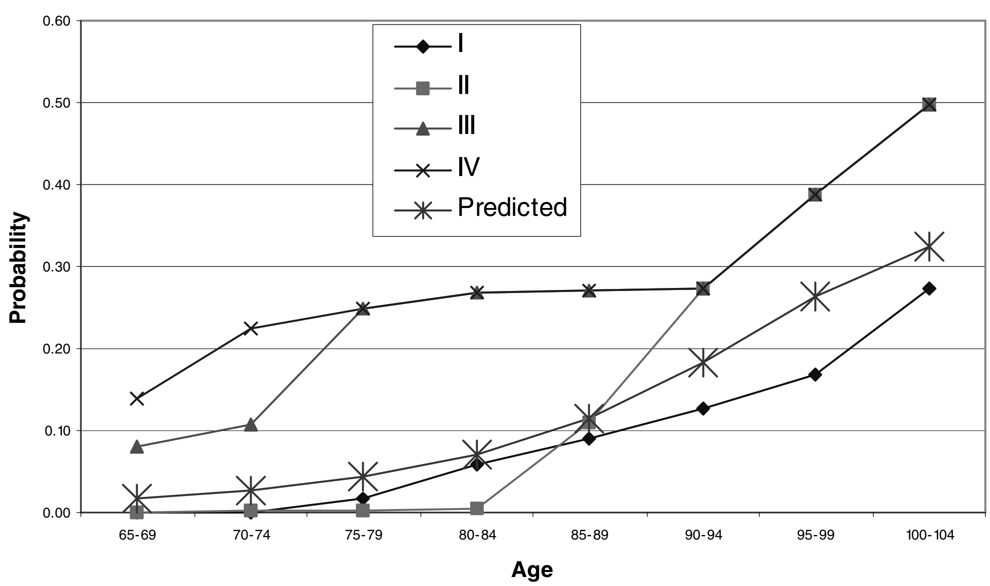
Figure 3
Adjusted Annual Probabilities of Death in Four Pure-Type Model, Males



averages of the individual trajectories also provided a basis for comparison with the age patterns for individual mortality above age 65, which were not well known.

Figures 3 and 4 display the adjusted individual annual probabilities of death shown in Table 8 for four categories of pure-type males and females by age at last birthday at the start of the one-year follow-up period. Also included in the figures for comparison with Gompertz's law are the average predicted probabilities for the NLTCS sample shown in Figures 1 and 2. The figures show that the pure-type probabilities of death had specific age ranges during which they increased slower than, faster than, or about the same rate as predicted by Gompertz's law, after adjusting for declines in vitality.

Figure 4
Adjusted Annual Probabilities of Death in Four Pure-Type Model, Females



The probabilities of death for Type IV were the highest at all ages. The probabilities of death for Type III were second highest and converged to those for Type IV over the age range 65–69 to 75–79 at a rate greater than or equal to that predicted by Gompertz's law, after which they were relatively constant and, hence, did not follow Gompertz's law.

The probabilities of death for Type I were the lowest at all ages, approaching but never quite reaching the average level of the predicted probabilities. Their rate of increase over the age range 70–74 to 80–84 was much faster than predicted by Gompertz's law; beyond that range, however, their rate of increase roughly matched that predicted by Gompertz's law.

The probabilities of death for Type II were low up to age 80–84, after which they increased precipitously at a rate much faster than predicted by Gompertz's law, leading to their overtaking and altering the trajectory for Type IV when the two pure types merged at age 90–94, increasing at a rate that roughly matched that predicted by Gompertz's law.

These results were broadly consistent with the expectations from Iachine et al. (1998) that (1) the variability in individual frailty with respect to mortality would be large and (2) the individual forces of mortality would rise substantially faster than the Gompertz function. However, the plateaus in the probabilities of death for Types III and IV combined with Gompertz-type increases for Types I and II after ages 80–84 and 90–94, respectively, indicated that the use of any simple parametric function will fail to capture the complexities of the patterns of individual mortality risks adequately.

Figures 5 and 6 display the unadjusted individual annual probabilities of death shown in Table 7 for the four categories of pure-type males and females by age at last birthday for ages 65–69 to 90–94 at the start of the one-year follow-up period. Also included in the figures are the average predicted probabilities for the NLTCS sample shown in Figures 1 and 2. The unadjusted pure-type probabilities of death were relatively constant over age, except for initial increases for Types III and IV and a large increase for Type II for males at age 90–94.

The relative constancies of these probabilities were consistent with the expectations from Manton et al. (1994a) that the rates of increase in individual mortality probabilities would be reduced substantially when time-varying covariates were included in the model. One implication is that the four pure types adequately represent the variation in individual mortality risks in a way that is relatively invariant over age.

Figure 5
(Unadjusted) Annual Probabilities of Death in Four Pure-Type Model, Males

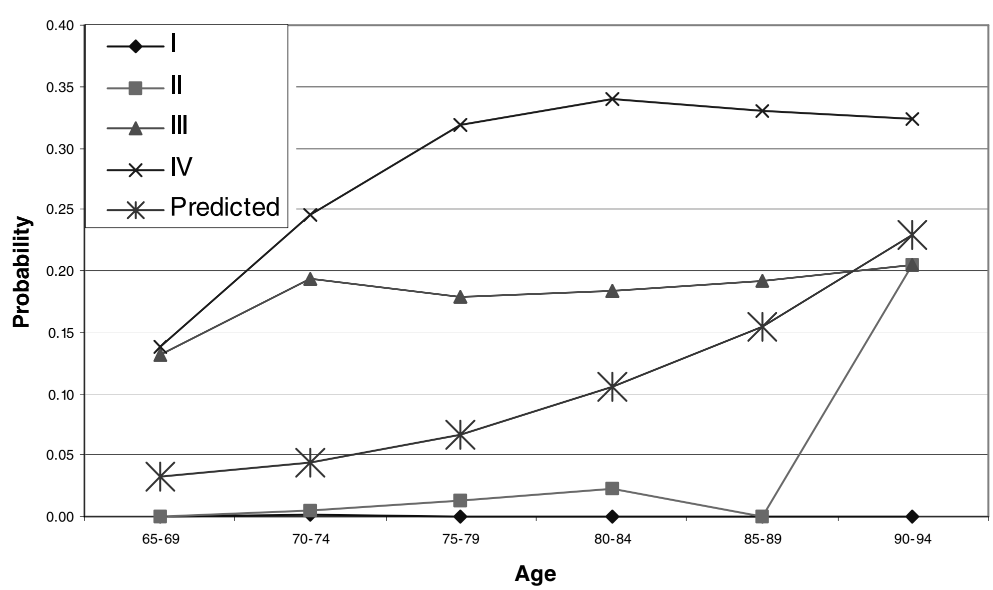
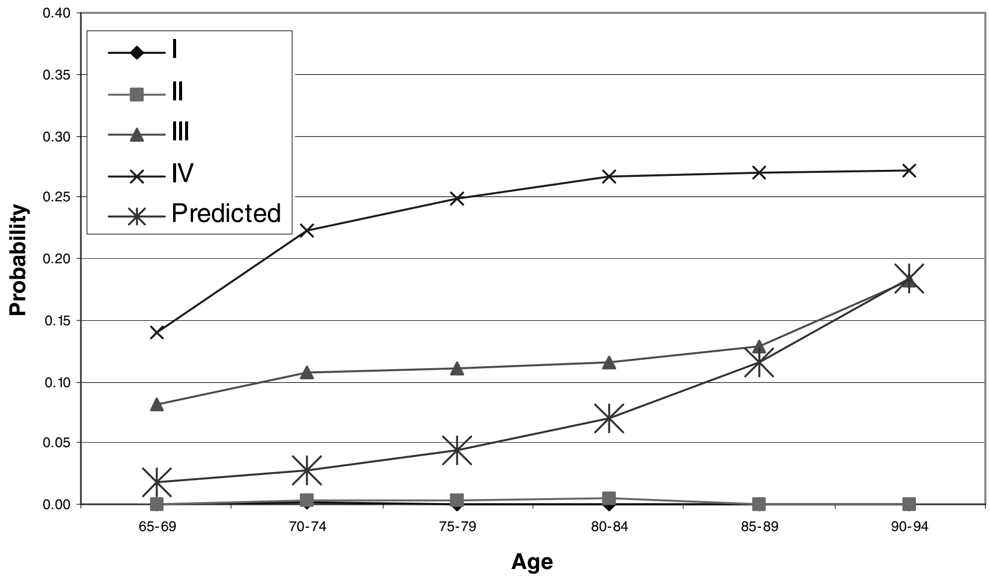


Figure 6
(Unadjusted) Annual Probabilities of Death in Four Pure-Type Model, Females



5.5 Adjusted GoM Scores

Table 9 displays the average adjusted GoM scores in the four-pure-type model for males and females by age at initial interview based on the same cases as shown in Table 3. The averages are displayed graphically in Figures 7 and 8 using a 100% stacked-line format to highlight the depletion or exhaustion of Type I over age, and the corresponding increases in Types II–IV over age. The GoM scores were constrained to sum to 1.0 (100%) so that the reductions in the combined average scores for Types I and II necessitated corresponding increases in the combined average scores for Types III and IV.

The declines in the average GoM scores for Type I and for the combination of Types I and II reflected the declining vitality of the NLTCS sample population over age in a manner consistent with expectations from Strehler and Mildvan (1960) that measures of individual vitality would decline approximately linearly with age. The results for females showed a moderate increase for the combination of Types I and II between ages 65–69 and 75–79, after which the decline was approximately linear.

For comparison, Figures 9 and 10 display the average unadjusted GoM scores shown in Table 3 for the four-pure-type model for males and females by age at initial interview for ages 65–69 to 90–94, using a 100% stacked-line format to highlight the age pattern. Ideally, one would have expected the average unadjusted GoM scores for Types I and II to increase over age because of differential mortality selection. Under such selection persons who survived to the oldest ages were those who faced the lowest mortality probabilities at younger ages. This expectation was fulfilled for Types I and II combined, but not for Type I alone.

5.6 Medicare Costs

Table 10 displays the average annual Medicare costs in the four-pure-type model based on Medicare program payments during the one-year period following each NLTCS interview, stratified by sex and type of Medicare service.

Methodologically, the cost variables were recoded to discrete measures using variable-sized categories covering the range of observed costs with cut-points based on multiples of 10 and \$2, \$5, and \$10; λ-values were estimated for these categories using the conditional form of the longitudinal GoM model with the g- and u-parameters fixed at the values in Tables 3, 5, and 6. The average costs were computed using the λ-weighted averages of the mean costs for each category. The differences between the

Table 9

Average Adjusted GoM Scores at Initial Interview in Four Pure-Type Model, by Sex and Age at Initial Interview

Initial Age	No. of Respondents	Average GoM Score by Type			
		I	II	III	IV
Males					
65–69	9,194	0.667	0.111	0.189	0.034
70–74	2,863	0.589	0.140	0.196	0.074
75–79	1,472	0.525	0.167	0.141	0.166
80–84	864	0.283	0.194	0.304	0.219
85–89	416	0.193	0.073	0.374	0.361
90–94	151	0.016	0.068	0.372	0.544
95–99	122	0.000	0.083	0.430	0.487
100–104	20	0.000	0.026	0.203	0.771
Total	15,102	0.590	0.125	0.201	0.084
Females					
65–69	11,355	0.559	0.105	0.306	0.030
70–74	3,854	0.555	0.174	0.213	0.058
75–79	2,469	0.490	0.250	0.096	0.163
80–84	1,792	0.235	0.287	0.241	0.237
85–89	1,223	0.127	0.162	0.295	0.416
90–94	525	0.000	0.122	0.282	0.596
95–99	575	0.000	0.121	0.264	0.615
100–104	97	0.000	0.063	0.160	0.776
Total	21,890	0.469	0.152	0.258	0.121

Source: Author's calculations based on data from the NLTCS.

observed and predicted costs were less than 1% while the differences between the total cost summed over the four types of Medicare services and the direct estimate of the costs of all Medicare services were less than 2%. These comparisons indicate that the discrete recoding of the costs to the variable-sized categories did not bias the estimates.

Table 10 shows that males used an average of $5,085 in Medicare services over the one-year period following the NLTCS interview, with the lowest costs ($2,729) for Type I and the highest ($11,979) for

Figure 7

Adjusted Age-Specific GoM Distribution, Males

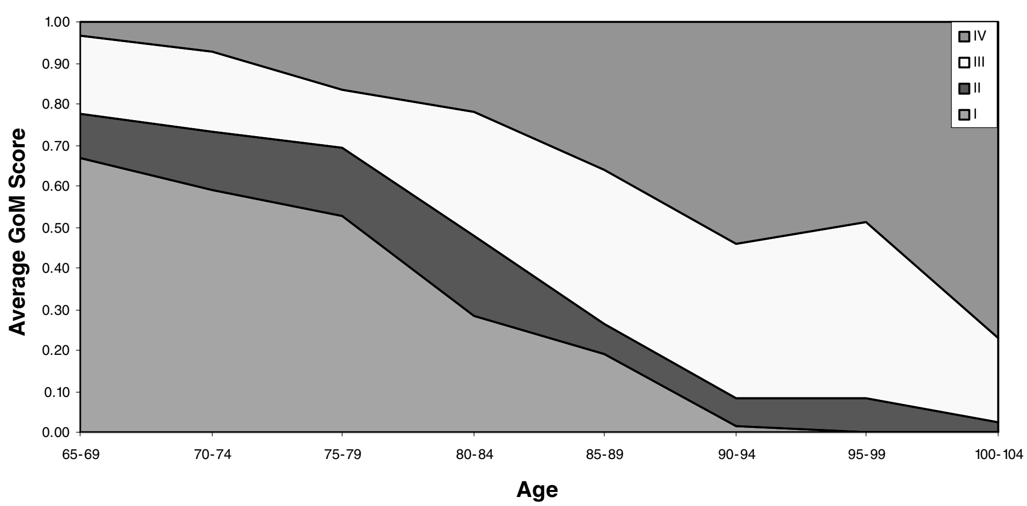


Figure 8
Adjusted Age-Specific GoM Distribution, Females

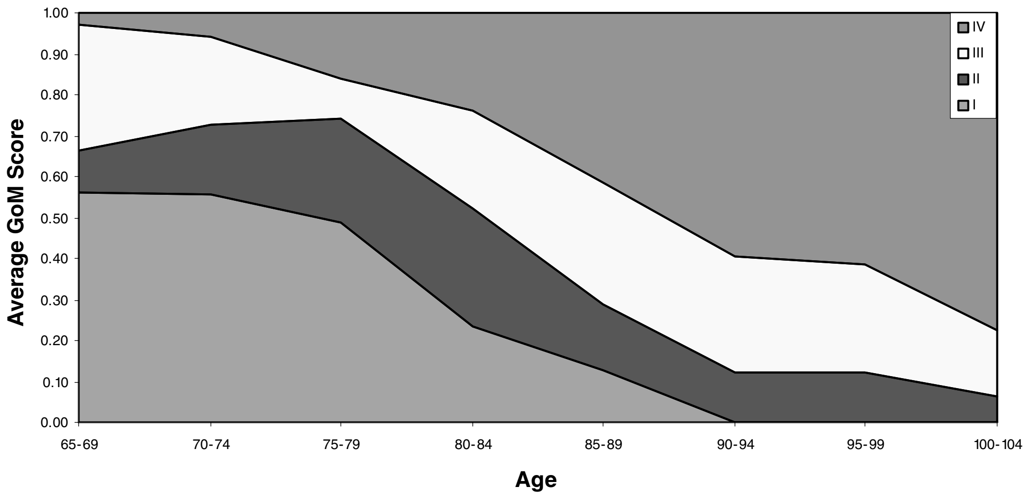


Figure 9
Unadjusted Age-Specific GoM Distribution, Males

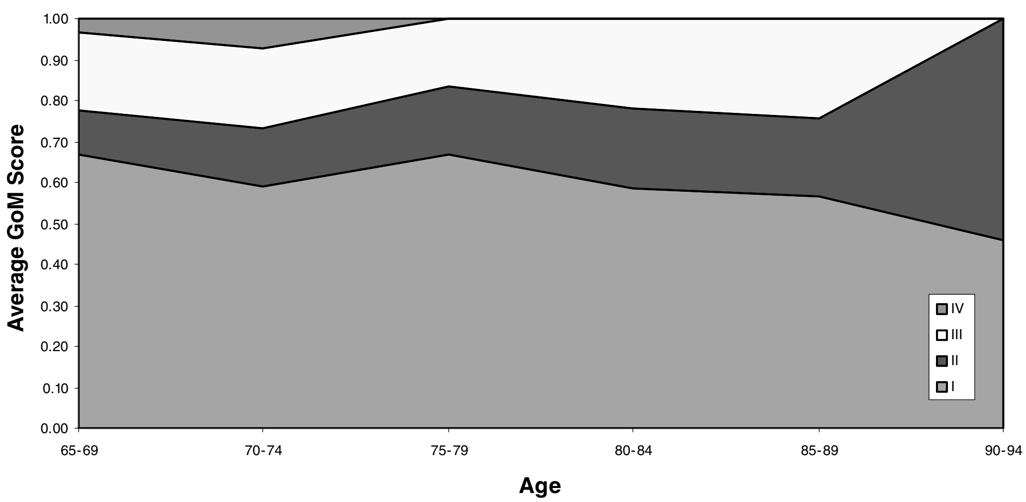


Figure 10
Unadjusted Age-Specific GoM Distribution, Females

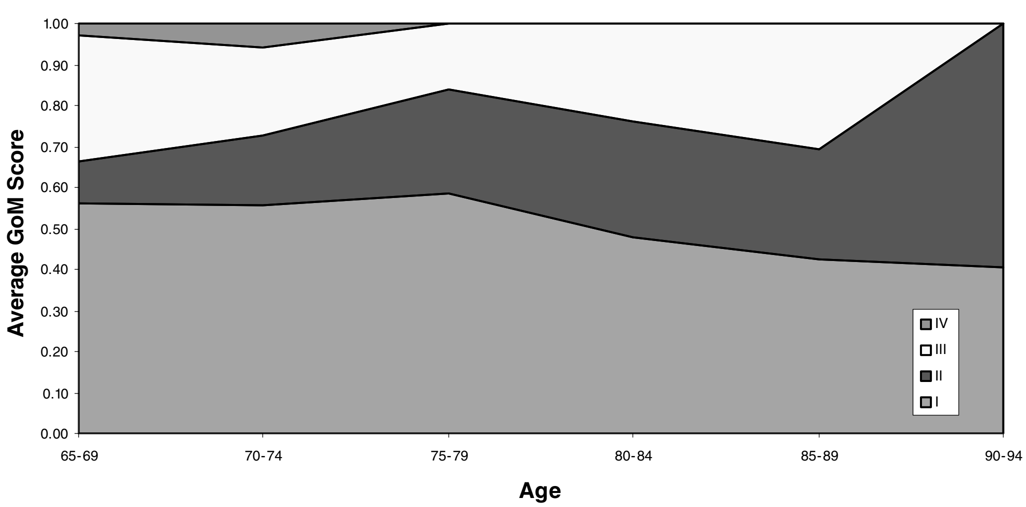


Table 10

Per Capita Costs for Services Provided under Medicare's Fee-for-Service Program within One Year after NLTCS Assessment; Four Pure-Type GoM Model, by Sex and Type of Service

No.	Service	Number of Person–Years at Risk[2]	Annual Cost by Type[1] I	II	III	IV	Observed Cost	Predicted Cost	Ratio Obs./Pred.
			Males						
1	Home health	23,250	21	136	242	1,614	$255	$253	1.009
2	Hospice	23,250	13	8	59	224	45	45	1.000
3	Skilled nursing facility	23,250	25	63	258	1,070	194	194	1.003
4	All other care	23,250	2,640	4,882	6,713	9,061	4,590	4,570	1.005
5	Total 1–4	23,250	2,699	5,089	7,272	11,969	5,085	5,062	1.005
6	All Medicare services	23,250	2,729	5,017	7,212	11,979	5,085	5,055	1.006
	Ratio of 6 to 5		1.011	0.986	0.992	1.001	1.000	0.999	
			Females						
1	Home health	37,339	34	690	313	1,321	$386	$386	1.000
2	Hospice	37,339	1	7	59	190	41	41	1.001
3	Skilled nursing facility	37,339	12	167	378	1,101	272	271	1.005
4	All other care	37,339	1,903	5,783	5,812	6,675	4,087	4,078	1.002
5	Total 1–4	37,339	1,951	6,647	6,561	9,287	4,787	4,776	1.002
6	All Medicare services	37,339	1,958	6,767	6,515	9,173	4,787	4,774	1.003
	Ratio of 6 to 5		1.004	1.018	0.993	0.988	1.000	0.999	

[1] All costs were converted to 2004 dollars using the CPI-U to inflate the costs for 1984, 1989, 1994, and 1999.
[2] Includes up to four observations per respondent; excludes respondents aged 65–69 in 1999.
Source: Author's calculations based on data from the NLTCS.

Type IV, with a ratio of 4.39 to 1. Similarly, females used an average of $4,787 in Medicare services, with the lowest costs ($1,958) for Type I and the highest ($9,173) for Type IV, with a ratio of 4.68 to 1. The excess costs for Type IV occurred for all four types of services. For males, all costs except hospice care increased monotonically across Types I to IV; for females, Type II had higher total costs and higher home health costs than Type III.

Hospice care was restricted to persons who were terminally ill and who enrolled in the hospice program. The average costs were similar for males and females, and the services were primarily restricted to Types III and IV. The costs for Type III were the same for males and females; the costs for Type IV were 3.80 and 3.22 times larger, respectively, for males and females. These results were consistent with the differences in subjective health status and number of medical conditions between Types III and IV in Table 2.

Care in a skilled nursing facility (SNF) was restricted to short-term skilled nursing and rehabilitative care following a hospital stay. The average costs were higher for females than for males by a factor of 1.40, but this was not a uniform differential. Costs for females were 2.65 times larger for Type II, 1.47 times larger for Type III, and 1.03 times larger for Type IV. Compared to Type III, the costs for Type IV were 4.15 and 2.91 times larger, respectively, for males and females.

The one-year SNF utilization rates were 14.8% for Type IV males and 14.5% for Type IV females. The corresponding nursing home residence rates were 51.6% for Type IV males and 75.2% for Type IV for females in Table 2, primarily reflecting custodial care in a long-term care institution at the start of the one-year follow-up period for which the Medicare costs were tabulated. Both sets of rates were consistent with the high levels of morbidity and disability experienced by Type IV.

Home health care included a range of skilled services provided to homebound individuals. The average costs were higher for females than for males by a factor of 1.51, but this also was not a uniform differential. Females had higher costs for Types II and III and lower costs for Type IV. Compared to Type III, the costs for Type IV were 6.67 and 4.22 times larger, respectively, for males and females.

The high home health cost for Type II females distinguished this service from hospice and SNF care, but was consistent with the high cost for the residual category (All Other Care) for Type II females.

6. DISCUSSION

This article had two goals: to introduce the reader to a broad range of research on survival at advanced ages, and to integrate the findings of that research into a coherent model of the trajectories of change in health and survival characteristics of individuals. The model was structured to represent simultaneously the essential features of the fixed frailty model (Vaupel et al. 1979) and the model of linearly declining vitality (Strehler and Mildvan 1960). Unlike those models, however, the new model was designed for easy and direct application to existing longitudinal data sets.

The application was successful in characterizing the health and survival experience of respondents to the NLTCS. The model was structured to represent the effects of variables that directly measured individual health, disability, and health-related behaviors. The results of the analysis indicated that the four-pure-type longitudinal GoM model is a parsimonious and powerful model of the changes in health and survival characteristics of individuals.

Several findings were noteworthy.

The likelihood ratio statistical tests comparing $K = 1$ with $K = 3$, 4, or 5 demonstrated that the elderly population is not homogeneous. This seems obvious, but it was a critical step in establishing the dimensionality of the longitudinal GoM model.

The distributions of the initial age-invariant GoM scores provided an effective and quantifiable implementation of the concept of fixed frailty. The GoM scores exhibited substantial variability over individuals; they were not exclusively clustered at the boundaries with values of 0 or 1. Pairwise and three-way memberships in Types I, II, and III were among the top five most frequent patterns of GoM scores. Approximately 25% of the initial age-invariant GoM membership was in Types III and IV, the pure types with the highest mortality rates. Membership in these pure types indicated impaired health at age 65.

The cumulative transition or vitality matrices provided an effective and quantifiable implementation of the concept of declining vitality. At age 90–94 and above the matrices had patterns in which only the first row and last column contained nonzero values, forming a pattern resembling a transposed ''L.'' A transposed L-shape also occurred for males and females with the three-pure-type model and for females with the five-pure-type model. For males the transposed L-shape also occurred with the five-pure-type model except for terms on the second diagonal (for Type II). Thus, the transposed L-shapes of the vitality matrices were robust with respect to the dimensionality of the model.

The combination of the initial distributions of GoM scores with the transposed L-shapes of the vitality matrices was consistent with a multistage process with random initial defects. The transposed L-shape arose because membership in Types II and III at older ages represented ''replacement'' membership drawn from Type I, not persisting initial membership in Types II and III.

The differences in mortality probabilities between the four pure types were substantial. At age 65 these differences implied life expectancies of 5.1 and 5.3 years, respectively, for Type IV males and females compared to 20.4 and 23.8 years, respectively, for Type I males and females. The substantially greater life expectancies for Type I were consistent with published data on centenarians indicating, on average, substantially better health and less disability in the decades prior to reaching age 100 than that of their contemporaries (Perls et al. 1999).

The adjusted mortality probabilities for the four pure types exhibited patterns of increase that were faster than predicted by Gompertz's law at selected ages (consistent with Iachine et al. 1998), that matched Gompertz's law at other ages (consistent with Strehler and Mildvan 1960), and that plateaued at high constant values at other ages (consistent with Perks 1932; also see Gavrilov and Gavrilova 2001). In the latter case the plateau was not a result of mortality selection on a heterogeneous population; instead it was a fundamental feature of individual mortality functions that deviated from Gompertz's law.

The unadjusted mortality probabilities were either relatively constant or increased over age at a rate generally slower than expected under Gompertz's law, consistent with Manton et al.'s (1994a) finding that the exponential growth constant of the Gompertz function in the two-component Gaussian stochastic process model was reduced substantially when covariates were included in the model. The faster-than-Gompertz increases in the adjusted mortality probabilities for Types I and II at selected ages reflected the impact of individual transitions from low to high mortality pure-type memberships.

Further refinement of the GoM model should focus on the statistical stability and smoothness of the progression of the age-specific parameter estimates. This could be done by imposing additional constraints on the GoM transition matrices, by introducing additional parameters to represent period and cohort effects in the study population, and by expanding the longitudinal follow-up of the individual respondents with data from the 2004 NLTCS.

Brown and McDaid (2003) conducted a comprehensive literature search, identifying 12 factors affecting retiree mortality that could be considered in developing risk classes for impaired life annuities in the individual annuity market. The 12 factors were age, sex, education, income, occupation, marital status, religion, smoking, alcohol, other health-related behaviors, obesity, and race/ethnicity. Seven of the 12 factors (age, sex, smoking, alcohol, other health-related behaviors, obesity, and race) were included in the current analysis; five factors (education, income, occupation, marital status, and religion) were excluded because they did not directly measure individual health, disability, and health-related behaviors. Four of the five factors (education, income, marital status, and religion) were provided as contextual measures in the NLTCS and could be included in further analyses of these data.

The Appendix included statistically significant tests for all 95 variables in the analysis along with rank orderings of the significance of each variable using ratios of χ^2 statistics to the associated degrees of freedom. The analysis was stratified by sex, with age having a special role in structuring the transition matrices. Five factors considered by Brown and McDaid (2003) were included in the Appendix and were rank ordered for males and females combined, respectively, as follows: smoking, #91; alcohol, #70; other health-related behaviors, #54 (for moderate exercise); obesity, #41; and race, #15. Although these five factors were statistically significant, the most significant factors were the measures of physical functioning based on the list of IADLs (#6; average item rank), ADLs (#17; average item rank), and functional limitations (#31; average item rank). Measures of cognitive functioning based on the Short Portable Mental Status Questionnaire (SPMSQ) were also highly significant (#33; average item rank).

The GoM model provided a solution to a problem identified by Brown and McDaid (2003, p. 41): how should one account for the joint effects of large numbers of risk factors that are potentially correlated or statistically dependent? The structure of the GoM model is such that the GoM scores are introduced as explanatory variables that resolve the correlations and statistical dependencies. Thus, conditional on the GoM scores, all risk factors are statistically independent. This condition was enforced in the current analysis via the product form of the GoM likelihood over all measured variables and tested by considering a sequence of models with increasing numbers of pure types (i.e., K-values). The statistical tests indicated that these conditions were achieved using a four-pure-type specification.

The NLTCS provided a broad range of data on the use, cost, and intensity of long-term-care and Medicare-funded acute care services at or following the time of each of the four NLTCS interviews. Large differentials were found between the pure types in acute and postacute Medicare costs, as shown in Table 10. Additional applications of the current model are being developed using the conditional form of the GoM model to analyze these measures for use in conjunction with the individual trajectories to estimate and project lifetime cost measures at the individual level. These individual level results can be aggregated into population estimates and projections using longitudinal sampling weights based on the NLTCS sampling design.

Applications of the GoM model extend beyond the NLTCS. The model can be applied to any longitudinal data with sufficient numbers of variables measured on each subject to support parameter estimation. For example, prior work with the Framingham Heart Study (e.g., Manton et al. 1994a) suggests that it would be feasible to develop a longitudinal GoM model for these data. The Framingham Heart Study conducted 25 biennial examinations with longitudinal follow-up over 48 years on a sample of 5,209 persons initially aged 28–62 years in 1948–50. Each examination included information on a broad range of cardiovascular disease risk factors and disease events. The later waves of the study (beginning at Exam 14) introduced health, disability, and cognitive functioning measures comparable to those in the NLTCS, suggesting that, with appropriate coding of the common variables, one could conduct pooled analyses of the two sets of data. Such analyses would double the size of the age range of the current model, with individual trajectories being tracked from age 30 onwards. This could improve the characterization of the early stages of the disablement process using traditional cardiovascular disease risk factors as precursors to the morbidity and disability measures jointly provided by the NLTCS and the later waves of the Framingham Heart Study.

APPENDIX

NLTCS VARIABLES, LOG-LIKELIHOOD VALUES, AND CHI-SQUARED STATISTICS BY VARIABLE, BY SEX

			Males						Females						Both Sexes	
No.	Variable Name / Description	No. of Response Levels	No. of Observations	Log of Likelihood Ratio for $K = 4$ vs. $K = 1$	Approximate Chi-Squared Statistic for $K = 4$ vs. $K = 1$	d.f. for Testing $K = 4$ vs. $K = 1^2$	Chi-Squared per d.f.	Rank Order of Chi-Squared per d.f.	No. of Observations	Log of Likelihood Ratio for $K = 4$ vs. $K = 1$	Approximate Chi-Squared Statistic for $K = 4$ vs. $K = 1$	d.f. for Testing $K = 4$ vs. $K = 1^2$	Chi-Squared per d.f.	Rank Order of Chi-Squared per d.f.	Chi-Squared per d.f.	Rank Order of Chi-Squared per d.f.
1	5-year survival status[1]	4	48,397	10,363.69	20,727.38	6	3,454.56	3	76,452	13,923.82	27,847.63	6	4,641.27	6	4,047.92	5
2	Proxy interview	2	8,329	799.96	1,599.93	3	533.31	20	17,633	2,644.64	5,289.27	3	1,763.09	15	1,148.20	17
3	Race	3	39,047	4,905.06	9,810.13	6	1,635.02	11	59,687	2,880.45	5,760.90	6	960.15	22	1,297.59	15
4	Residence type: institutional vs. noninstitutional	2	27,819	2,558.40	5,116.80	3	1,705.60	10	43,764	7,127.23	14,254.47	3	4,751.49	5	3,228.54	7
5	Height	5	3,400	50.62	101.24	12	8.44	92	6,392	61.16	122.32	12	10.19	93	9.31	93
6	Body Mass Index—current BMI class	4	3,349	343.71	687.43	9	76.38	63	6,218	704.14	1,408.28	9	156.48	60	116.43	61
7	Body Mass Index—BMI class at age 50 years	4	3,113	179.34	358.67	9	39.85	75	5,635	324.90	649.80	9	72.20	72	56.03	75
8	Body Mass Index—BMI class 12 months prior to interview	4	3,250	329.78	659.56	9	73.28	66	5,983	715.85	1,431.70	9	159.08	59	116.18	62
9	Alcohol use	3	3,483	181.30	362.60	6	60.43	69	6,578	237.12	474.24	6	79.04	70	69.74	70
10	Cigarette use	3	3,491	31.76	63.52	6	10.59	90	6,588	40.64	81.27	6	13.55	92	12.07	91
11	Exercise—hours / minutes of vigorous activities	5	3,424	438.85	877.70	12	73.14	67	6,450	535.93	1,071.86	12	89.32	68	81.23	67
12	Exercise—hours / minutes of moderate activities	5	3,397	760.26	1,520.52	12	126.71	52	6,410	1,248.46	2,496.92	12	208.08	54	167.39	54
13	Exercise—hours / minutes of light activities	5	3,364	540.17	1,080.34	12	90.03	61	6,291	812.81	1,625.63	12	135.47	63	112.75	64
14	Medical—rheumatism or arthritis	2	7,026	427.80	855.61	3	285.20	33	13,514	716.95	1,433.90	3	477.97	40	381.58	38
15	Medical—other permanent numbness or stiffness	2	7,021	454.97	909.95	3	303.32	29	13,487	603.15	1,206.29	3	402.10	43	352.71	40
16	Medical—paralysis	2	7,030	350.87	701.74	3	233.91	39	13,518	398.87	797.73	3	265.91	52	249.91	48
17	Medical—multiple sclerosis	2	7,028	12.78	25.57	3	8.52	91	13,520	30.07	60.13	3	20.04	87	14.28	89
18	Medical—cerebral palsy	2	7,029	6.42	12.84	3	4.28	94	13,517	10.43	20.86	3	6.95	94	5.62	94
19	Medical—epilepsy	2	7,023	16.38	32.76	3	10.92	89	13,514	27.19	54.39	3	18.13	88	14.53	88
20	Medical—Parkinson's disease	2	7,028	83.30	166.60	3	55.53	71	13,510	95.54	191.09	3	63.70	76	59.61	72
21	Medical—glaucoma	2	7,023	39.67	79.34	3	26.45	76	13,500	54.00	108.00	3	36.00	81	31.22	79
22	Medical—diabetes	2	7,026	112.50	225.01	3	75.00	64	13,509	301.58	603.15	3	201.05	55	138.03	57
23	Medical—cancer	2	7,023	17.16	34.31	3	11.44	88	13,499	23.00	46.01	3	15.34	89	13.39	90
24	Medical—frequent constipation	2	7,002	357.67	715.35	3	238.45	38	13,485	588.19	1,176.38	3	392.13	44	315.29	43
25	Medical—frequent trouble sleeping	2	7,017	534.60	1,069.19	3	356.40	26	13,491	714.72	1,429.44	3	476.48	41	416.44	34
26	Medical—frequent severe headaches	2	7,010	262.65	525.31	3	175.10	46	13,496	507.95	1,015.89	3	338.63	48	256.87	47
27	Medical—obesity or medically overweight	2	7,021	232.08	464.15	3	154.72	49	13,490	794.21	1,588.43	3	529.48	35	342.10	41
28	Medical—arteriosclerosis or hardening of the arteries	2	6,956	300.57	601.15	3	200.38	43	13,406	517.31	1,034.61	3	344.87	47	272.63	46
29	Medical—heart attack in 12 months prior to interview	2	7,010	76.71	153.43	3	51.14	72	13,477	98.38	196.76	3	65.59	74	58.36	73
30	Medical—any other heart problem in 12 months prior to interview	2	7,012	242.67	485.33	3	161.78	48	13,487	490.66	981.32	3	327.11	49	244.44	49
31	Medical—hypertension or high blood pressure in 12 months prior to interview	2	7,012	210.62	421.24	3	140.41	51	13,472	366.34	732.67	3	244.22	53	192.32	53
32	Medical—stroke in 12 months prior to interview	2	7,010	166.15	332.30	3	110.77	56	13,471	281.51	563.03	3	187.68	57	149.22	56
33	Medical—circulation trouble in arms or leg in 12 months prior to interview	2	7,003	949.60	1,899.20	3	633.07	19	13,464	1,507.17	3,014.34	3	1,004.78	19	818.92	20
34	Medical—pneumonia in 12 months prior to interview	2	7,010	111.00	222.00	3	74.00	65	13,457	98.44	196.87	3	65.62	73	69.81	69
35	Medical—flu or influenza in 12 months prior to interview	2	7,012	104.78	209.55	3	69.85	68	13,471	206.20	412.40	3	137.47	62	103.66	65
36	Medical—bronchitis in 12 months prior to interview	2	7,012	188.33	376.67	3	125.56	53	13,474	284.54	569.08	3	189.69	56	157.62	55
37	Medical—emphysema in 12 months prior to interview	2	7,015	217.17	434.35	3	144.78	50	13,471	124.39	248.78	3	82.93	69	113.85	63
38	Medical—asthma in 12 months prior to interview	2	7,015	187.99	375.98	3	125.33	54	13,483	188.56	377.12	3	125.71	64	125.52	59
39	Medical—broken hip in 12 months prior to interview	2	7,012	26.56	53.13	3	17.71	82	13,482	48.12	96.23	3	32.08	83	24.89	84
40	Medical—other broken bones in 12 months prior to interview	2	7,007	25.90	51.79	3	17.26	85	13,469	22.70	45.40	3	15.13	91	16.20	87
41	Medical—senility	2	7,181	442.93	885.86	3	295.29	30	14,176	1,197.41	2,394.81	3	798.27	29	546.78	30
42	Medical—Alzheimer's disease	2	5,175	158.48	316.97	3	105.66	57	10,342	442.94	885.88	3	295.29	50	200.47	52
43	Medical—mental retardation	2	7,182	59.86	119.72	3	39.91	74	14,180	111.94	223.89	3	74.63	71	57.27	74

44	See well enough to read newspaper	2	6,965	442.66	885.32	3	295.11	31	13,413	767.79	1,535.57	3	511.86	36	403.48	35
45	Subjective health status	4	6,543	1,469.75	2,939.50	9	326.61	27	12,782	1,961.39	3,922.77	9	435.86	42	381.24	39
46	ADL Personal Assistance Level—bathing	6	27,814	7,333.75	14,667.51	15	977.83	13	43,753	15,742.50	31,485.01	15	2,099.00	12	1,538.42	12
47	ADL Personal Assistance Level—dressing	6	27,814	5,384.09	10,768.19	15	717.88	17	43,753	11,344.22	22,688.45	15	1,512.56	18	1,115.22	18
48	ADL Personal Assistance Level—toileting	6	27,814	4,914.04	9,828.08	15	655.21	18	43,753	11,398.87	22,797.74	15	1,519.85	17	1,087.53	19
49	ADL Personal Assistance Level—transferring in / out bed	6	27,814	5,932.27	11,864.55	15	790.97	15	43,753	12,658.62	25,317.24	15	1,687.82	16	1,239.39	16
50	ADL Personal Assistance Level—eating	6	27,814	2,838.15	5,676.30	15	378.42	25	43,753	6,189.46	12,378.93	15	825.26	27	601.84	27
51	ADL Personal Assistance Level—continence	4	27,814	3,547.13	7,094.26	9	788.25	16	43,753	8,926.37	17,852.75	9	1,983.64	13	1,385.94	14
52	ADL Personal Assistance Level—indoor mobility	6	27,814	6,679.40	13,358.80	15	890.59	14	43,753	14,556.25	29,112.49	15	1,940.83	14	1,415.71	13
53	IADL Limitations—light housework	2	26,528	3,898.50	7,797.00	3	2,599.00	7	39,646	5,543.84	11,087.67	3	3,695.89	9	3,147.45	8
54	IADL Limitations—laundry	2	26,528	4,947.78	9,895.55	3	3,298.52	4	39,646	7,560.36	15,120.72	3	5,040.24	4	4,169.38	4
55	IADL Limitations—cooking	2	26,528	4,248.61	8,497.23	3	2,832.41	6	39,646	6,640.63	13,281.27	3	4,427.09	7	3,629.75	6
56	IADL Limitations—grocery shopping	2	26,528	5,329.14	10,658.28	3	3,552.76	2	39,646	9,136.14	18,272.28	3	6,090.76	2	4,821.76	2
57	IADL Limitations—outside mobility	2	26,758	5,436.69	10,873.37	3	3,624.46	1	40,447	10,537.88	21,075.77	3	7,025.26	1	5,324.86	1
58	IADL Limitations—travel	2	26,528	4,802.11	9,604.22	3	3,201.41	5	39,646	9,123.69	18,247.38	3	6,082.46	3	4,641.93	3
59	IADL Limitations—managing money	2	26,528	3,167.18	6,334.36	3	2,111.45	9	39,646	5,624.80	11,249.60	3	3,749.87	8	2,930.66	9
60	IADL Limitations—taking medicines	2	26,528	3,397.08	6,794.15	3	2,264.72	8	39,646	5,154.42	10,308.84	3	3,436.28	10	2,850.50	10
61	IADL Limitations—phoning	2	26,528	2,297.76	4,595.52	3	1,531.84	12	39,646	3,224.40	6,448.81	3	2,149.60	11	1,840.72	11
62	Functional Limitations—climbing one flight of stairs	4	6,590	2,080.02	4,160.04	9	462.23	22	12,625	3,470.97	6,941.95	9	771.33	30	616.78	23
63	Functional Limitations—bending to put on socks or stockings	4	6,933	1,964.61	3,929.21	9	436.58	23	13,287	3,440.34	6,880.67	9	764.52	31	600.55	28
64	Functional Limitations—lifting and holding a 10 lb. package	4	6,914	2,265.03	4,530.05	9	503.34	21	13,240	3,919.36	7,838.71	9	870.97	24	687.15	21
65	Functional Limitations—reaching above head	4	6,983	1,214.45	2,428.90	9	269.88	34	13,416	2,300.91	4,601.82	9	511.31	37	390.60	36
66	Functional Limitations—combing or brushing hair	4	6,998	976.83	1,953.66	9	217.07	42	13,450	2,467.03	4,934.06	9	548.23	34	382.65	37
67	Functional Limitations—washing hair	4	6,990	1,738.40	3,476.80	9	386.31	24	13,420	3,801.90	7,603.80	9	844.87	25	615.59	24
68	Functional Limitations—using fingers to grasp and handle small objects	4	6,987	986.35	1,972.70	9	219.19	41	13,439	1,584.68	3,169.37	9	352.15	46	285.67	45
69	SPMSQ—What is the date today?	2	5,361	433.71	867.41	3	289.14	32	11,874	1,385.83	2,771.66	3	923.89	23	606.51	25
70	SPMSQ—What day of week is this?	2	5,354	378.20	756.40	3	252.13	36	11,860	1,263.52	2,527.05	3	842.35	26	547.24	29
71	SPMSQ—What is your street address?	2	5,355	460.79	921.58	3	307.19	28	11,847	1,502.67	3,005.35	3	1,001.78	20	654.49	22
72	SPMSQ—In what state is this?	2	4,401	180.94	361.87	3	120.62	55	9,959	751.59	1,503.17	3	501.06	39	310.84	44
73	SPMSQ—How old are you?	2	4,407	338.44	676.89	3	225.63	40	9,961	1,093.34	2,186.69	3	728.90	32	477.26	32
74	SPMSQ—When were you born? (month, day, year)	2	5,132	253.90	507.79	3	169.26	47	11,295	1,022.08	2,044.16	3	681.39	33	425.33	33
75	SPMSQ—Who is the President of the United States now?	2	4,404	359.49	718.98	3	239.66	37	9,958	1,453.54	2,907.08	3	969.03	21	604.34	26
76	SPMSQ—Who was the President just before him?	2	4,403	391.43	782.86	3	260.95	35	9,960	1,213.54	2,427.08	3	809.03	28	534.99	31
77	SPMSQ—What was your mother's maiden name?	2	4,385	152.23	304.46	3	101.49	58	9,923	566.53	1,133.06	3	377.69	45	239.59	50
78	SPMSQ—Subtract 3 from 20 and keep subtracting . . .	2	4,409	269.08	538.16	3	179.39	45	9,951	752.49	1,504.97	3	501.66	38	340.52	42
79	Behavior—Lose temper and throw, kick, slam, destroy things	3	6,951	78.12	156.24	6	26.04	77	13,374	78.23	156.45	6	26.08	86	26.06	82
80	Behavior—Lose your way and not find your way back	2	6,964	117.78	235.55	3	78.52	62	13,385	143.69	287.39	3	95.80	66	87.16	66
81	Behavior—Take anything not yours without realizing	2	6,955	32.79	65.58	3	21.86	80	13,375	98.36	196.73	3	65.58	75	43.72	76
82	Behavior—Forget to do important things like eating	2	6,956	288.92	577.85	3	192.62	44	13,369	423.74	847.48	3	282.49	51	237.55	51
83	Memory—Name as many animals as possible in one minute	4	723	26.20	52.41	9	5.82	93	1,341	68.18	136.36	9	15.15	90	10.49	92
84	Memory—Delayed 12-word recall	12	714	61.13	122.26	33	3.70	95	1,314	97.67	195.33	33	5.92	95	4.81	95
85	MMSE—Orientation: day, date, month, year, season	6	1,022	116.72	233.44	15	15.56	86	2,115	300.86	601.71	15	40.11	79	27.84	81
86	MMSE—Orientation: country, city, street, floor #, address	6	1,022	164.48	328.96	15	21.93	79	2,115	356.16	712.32	15	47.49	77	34.71	77
87	MMSE—Registration: three-word memory	4	1,022	69.79	139.59	9	15.51	87	2,115	154.28	308.56	9	34.28	82	24.90	83
88	MMSE—Attention: subtract 7 from 100 and keep subtracting . . .	6	1,022	130.27	260.55	15	17.37	84	2,115	225.50	451.00	15	30.07	85	23.72	86
89	MMSE—Recall: three-word memory	4	1,022	78.72	157.44	9	17.49	83	2,115	173.24	346.48	9	38.50	80	28.00	80
90	MMSE—Language: point and name	3	1,022	53.50	107.00	6	17.83	81	2,115	94.71	189.42	6	31.57	84	24.70	85
91	MMSE—Language: repeat phrase	2	1,022	63.46	126.92	3	42.31	73	2,115	141.04	282.07	3	94.02	67	68.17	71
92	MMSE—Language: three-stage command	4	1,022	102.85	205.71	9	22.86	78	2,115	188.62	377.24	9	41.92	78	32.39	78
93	MMSE—Language: read and obey	2	1,022	84.74	169.49	3	56.50	70	2,115	155.46	310.92	3	103.64	65	80.07	68
94	MMSE—Language: sentence writing	2	1,022	137.12	274.23	3	91.41	60	2,115	234.52	469.05	3	156.35	61	123.88	60
95	MMSE—Language: figure drawing	2	1,022	145.94	291.87	3	97.29	59	2,115	253.18	506.36	3	168.79	58	133.04	58
	Total	287	946,493	120,124	240,247	573	419.28		1,625,882	223,379	446,758	573	779.68		599.48	

[1] 5-Year Survival Status was included in the analysis to code for missing data due to death at follow-up. For this one variable the predicted probabilities were computed using GoM scores at both the start and end of each follow-up interval to approximate the GoM score changes during the interval; the statistics in this table are the summed values for the two sets of computations. A separate survival analysis with age-specific annual probabilities of death was conducted using the outputs of the initial analysis. See text for details.

[2] Incremental degrees of freedom refer to the difference in the number of λ-values between the 4 pure-type model and the 1 pure-type model for the indicated variable. The total of the incremental d.f. does not include 45,306 d.f. (males) and 65,670 d.f. (females) for the GoM scores and 42 d.f. for the transition matrix parameters. The chi-squared statistics indicate the incremental effect of each variable assuming that it was the last variable added to the model. Equivalently, the chi-squared statistics indicate the effect of constraining the λ-values for each variable to be equal across the 4 pure types of the 4 pure-type model.

Source: Author's calculations based on data from the NLTCS.

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REFERENCES

AKAIKE, H. 1974. A New Look at the Statistical Model Identification. *IEEE Transactions on Automatic Control* AC-19(6): 716–23.

BORTZ, W. M. 2002. A Conceptual Framework of Frailty: A Review. *Journal of Gerontology: MEDICAL SCIENCES* 57A(5): M283–88.

BROWN, R. L, AND J. MCDAID. 2003. Factors Affecting Retirement Mortality. *North American Actuarial Journal* 7(2): 24–43.

CHIANG, C. L. 1984. *The Life Table and Its Applications*. Malabar, FL: Robert E. Krieger.

COX, D. R. 1972. Regression Models and Life-Tables. *Journal of the Royal Statistical Society: Series B* (*Methodological*) 34(2): 187–220.

DE BENEDICTIS, G., Q. TAN, B. JEUNE, K. CHRISTENSEN, S. V. UKRAINTSEVA, M. BONAFIDÈ, C. FRANCESCHI, J. W. VAUPEL, AND A. I. YASHIN. 2001. Recent Advances in Human Gene–Longevity Association Studies. *Mechanisms of Ageing and Development* 122: 909–20.

FREEDMAN, V. A., E. CRIMMINS, R. F. SCHOENI, B. C. SPILLMAN, H. AYKAN, E. KRAMAROW, K. L. LAND, J. LUBITZ, K. MANTON, L. G. MARTIN, D. SHINBERG, AND T. WAIDMANN. 2004. Resolving Inconsistencies in Trends in Old-Age Disability: Report from a Technical Working Group. *Demography* 41(3): 417–41.

FREEDMAN, V. A., AND L. G. MARTIN. 1998. Understanding Trends in Functional Limitations among Older Americans. *American Journal of Public Health* 88(10): 1457–62.

FRIED, L. P., L. FERRUCCI, J. DARER, J. D. WILLIAMSON, AND G. ANDERSON. 2004. Untangling the Concepts of Disability, Frailty, and Comorbidity: Implications for Improved Targeting and Care. *Journal of Gerontology: MEDICAL SCIENCES* 59(3): 255–63.

FRIED, L. P., C. M. TANGEN, J. WALSTON, A. B. NEWMAN, C. HIRSCH, J. GOTTDIENER, T. SEEMAN, R. TRACY, W. J. KOP, G. BURKE, AND M. A. MCBURNIE. 2001. Frailty in Older Adults: Evidence for a Phenotype. *Journal of Gerontology: MEDICAL SCIENCES* 56A(3): M146–56.

GAVRILOV, L. A., AND N. S. GAVRILOVA. 2001. The Reliability Theory of Aging and Longevity. *Journal of Theoretical Biology* 213: 527–45.

GOMPERTZ, B. 1825. On the Nature of the Function Expressive of the Law of Human Mortality, and on a New Mode of Determining the Value of Life Contingencies. *Philosophical Transactions of the Royal Society of London* 115: 513–83.

HOUGAARD, P. 1984. Life Table Methods for Heterogeneous Populations: Distributions Describing the Heterogeneity. *Biometrika* 71(1): 75–83.

IACHINE, I. A., N. V. HOLM, J. R. HARRIS, A. Z. BEGUN, M. K. IACHINA, M. LAITINEN, J. KAPRIO, AND A. I. YASHIN. 1998. How Heritable Is Individual Susceptibility to Death? The Results of an Analysis of Survival Data on Danish, Swedish and Finnish Twins. *Twin Research* 1: 196–205.

KESTENBAUM, B. 1992. A Description of the Extreme Aged Population Based on Improved Medicare Enrollment Data. *Demography* 29(4): 565–80.

KINOSIAN, B., E. STALLARD, J. LEE, M. A. WOODBURY, A. ZBROZEK, AND H. A. GLICK. 2000. Predicting 10-Year Outcomes of Alzheimer's Disease. *Journal of the American Geriatrics Society* 48(6): 631–38.

KINOSIAN, B., E. STALLARD, K. G. MANTON, D. L. STRALEY, A. ZBROZEK, AND H. A. GLICK. 2004. The Expected Outcomes and Costs of U.S. Patients with Incident Suspected Alzheimer's Disease (AD) over 15 Years. Abstract of Poster Session at the 9th International Conference on Alzheimer's Disease and Related Disorders, Philadelphia, PA, July 17–22.

LAZARSFELD, P. F., AND N. W. HENRY. 1968. *Latent Structure Analysis*. Boston: Houghton Mifflin.

MANTON, K. G., L. S. CORDER, AND E. STALLARD. 1993. Estimates of Change in Chronic Disability and Institutional Incidence and Prevalence Rates in the U.S. Elderly Population from the 1982, 1984, and 1989 National Long Term Care Survey. *Journal of Gerontology: Social Sciences* 48(4): S153–66.

———. 1997. Chronic Disability Trends in Elderly United States Populations: 1982–1994. *Proceedings of the National Academy of Sciences* 94: 2593–98.

MANTON, K. G., AND X. GU. 2001. Changes in the Prevalence of Chronic Disability in the United States Black and Nonblack Population above Age 65 from 1982 to 1999. *Proceedings of the National Academy of Sciences* 98(11): 6354–59.

MANTON, K. G., AND E. STALLARD. 1984. *Recent Trends in Mortality Analysis*. Orlando, FL: Academic Press.

———. 1988. *Chronic Disease Modelling*. New York: Oxford University Press.

MANTON, K. G., E. STALLARD, AND B. SINGER. 1992a. Projecting the Future Size and Health Status of the U.S. Elderly Population. *International Journal of Forecasting* 8: 433–58.

MANTON, K. G., E. STALLARD, AND J. W. VAUPEL. 1986. Alternative Models for the Heterogeneity of Mortality Risks among the Aged. *Journal of the American Statistical Association* 81(395): 635–64.

MANTON, K. G., E. STALLARD, AND M. A. WOODBURY. 1991. A Multivariate Event History Model Based upon Fuzzy States: Estimation from Longitudinal Surveys with Informative Nonresponse. *Journal of Official Statistics* 7(3): 261–93.

MANTON, K. G., E. STALLARD, M. A. WOODBURY, AND J. E. DOWD. 1994a. Time-Varying Covariates in Models of Human Mortality and Aging: Multidimensional Generalizations of the Gompertz. *Journal of Gerontology: BIOLOGICAL SCIENCES* 49(4): B169–90.

MANTON, K. G., M. A. WOODBURY, E. STALLARD, AND L. S. CORDER. 1992b. The Use of Grade of Membership to Estimate Regression Relationships. In *Sociological Methodology, 1992,* ed. P. Marsden, pp. 321–81. London: Basil Blackwell.

MANTON, K. G., M. A. WOODBURY, AND H. D. TOLLEY. 1994b. *Statistical Applications Using Fuzzy Sets*. New York: John Wiley & Sons.

MCGINNIS, J. M., AND W. H. FOEGE. 1993. Actual Causes of Death in the United States. *Journal of the American Medical Association* 270(18): 2207–12.

MCGUE, M., J. W. VAUPEL, N. HOLM, AND B. HARVALD. 1993. Longevity Is Moderately Heritable in a Sample of Danish Twins Born 1870–1880. *Journal of Gerontology: BIOLOGICAL SCIENCES* 48(6): B237–44.

MOKDAD, A. H., J. S. MARKS, D. F. STROUP, AND J. L. GERBERDING. 2004. Actual Causes of Death in the United States, 2000. *Journal of the American Medical Association* 291(10): 1238–45.

NAGI, S. Z. 1976. An Epidemiology of Disability among Adults in the United States. *Milbank Memorial Fund Quarterly—Health and Society* (fall): 439–67.

NATIONAL CENTER FOR HEALTH STATISTICS (NCHS). 1997. *U.S. Decennial Life Tables for 1989–91*. Vol. 1, no. 1. Hyattsville, MD: NCHS.

OLSHANSKY, S. J., AND B. A. CARNES. 1997. Ever since Gompertz. *Demography* 34(1): 1–15.

ORCHARD, R., AND M. A. WOODBURY. 1971. A Missing Information Principle: Theory and Applications. In *Proceedings of the Sixth Berkeley Symposium on Mathematical Statistics and Probability,* ed. L. M. LeCam, J. Neyman, and E. L. Scott, pp. 697–715. Berkeley, CA: University of California Press.

PERKS, W. 1932. On Some Experiments in the Graduation of Mortality Statistics. *Journal of the Institute of Actuaries* 63: 12–57.

PERLS, T. T., M. H. SILVER, AND J. F. LAUERMAN. 1999. *Living to 100*. New York: Basic Books.

SELF, S. G., AND K. Y. LIANG. 1987. Asymptotic Properties of Maximum Likelihood Estimators and Likelihood Ratio Tests under Nonstandard Conditions. *Journal of the American Statistical Association* 82(398): 605–10.

SINGPURWALLA, N. D., AND J. M. BOOKER. 2004. Membership Functions and Probability Measures of Fuzzy Sets. *Journal of the American Statistical Association* 99(467): 867–89.

STALLARD, E. 2000. Retirement and Health: Estimates and Projections of Acute and Long-Term Care Needs and Expenditures of the U.S. Elderly Population. In *Retirement Needs Framework,* SOA Monograph M-RS00-1, chapter 15. Schaumburg, IL: Society of Actuaries.

———. 2001. Estimates of the Incidence, Prevalence, Duration, Intensity, and Cost of Chronic Disability among the U.S. Elderly. Paper presented at the annual meeting of the Population Association of America, March 29–31, Washington, DC. Silver Spring, MD: PAA.

———. 2005. Trajectories of Disability and Mortality among the U.S. Elderly Population: Evidence from the 1984–1999 NLTCS. In *Living to 100 and Beyond Monograph,* Society of Actuaries Online Monographs.

STALLARD, E., AND R. K. YEE. 2000. *Non-insured Home and Community-Based Long-Term Care Incidence and Continuance Tables.* Actuarial Report Issued by the Long-Term Care Experience Committee. Schaumburg, IL: Society of Actuaries.

STREHLER, B. L., AND A. S. MILDVAN. 1960. General Theory of Mortality and Aging. *Science* 132(3418): 14–21.

THERNEAU, T. M., AND P. M. GRAMBSCH. 2000. *Modeling Survival Data: Extending the Cox Model*. New York: Springer.

TOLLEY, H. D., AND K. G. MANTON. 1991. Intervention Effects among a Collection of Risks. *Transactions of the Society of Actuaries* 43: 443–68.

VAUPEL, J. W., J. R. CAREY, K. CHRISTENSEN, T. E. JOHNSON, A. I. YASHIN, N. V. HOLM, I. A. IACHINE, V. KANNISTO, A. A. KHAZAELI, P. LIEDO, V. D. LONGO, Y. ZENG, K. G. MANTON, AND J. W. CURTSINGER. 1998. Biodemographic Trajectories of Longevity. *Science* 280(5365): 855–60.

VAUPEL, J. W., K. G. MANTON, AND E. STALLARD. 1979. The Impact of Heterogeneity in Individual Frailty on the Dynamics of Mortality. *Demography* 16(3): 439–54.

WACHTER, K. W. 1999. Grade of Membership Models in Low Dimensions. *Statistical Papers* 40: 439–57.

WETTERSTRAND, W. H. 1981. Parametric Models for Life Insurance Mortality Data: Gompertz's Law over Time. *Transactions of the Society of Actuaries* 33: 443–68.

WILKS, S. S. 1938. The Large Sample Distribution of the Likelihood Ratio for Testing Composite Hypotheses. *Annals of Mathematical Statistics* 9(1): 60–62.

WOODBURY, M. A., AND J. CLIVE. 1974. Clinical Pure Types as a Fuzzy Partition. *Journal of Cybernetics* 4(3): 111–21.

WOODBURY, M. A., J. CLIVE, AND A. GARSON. 1978. Mathematical Typology: A Grade of Membership Technique for Obtaining Disease Definition. *Computers and Biomedical Research* 11(3): 277–98.

WOODBURY, M. A., AND K. G. MANTON. 1977. A Random-Walk Model of Human Mortality and Aging. *Theoretical Population Biology* 11(1): 37–48.

WOODBURY, M. A., K. G. MANTON, AND H. D. TOLLEY. 1994. A General Model for Statistical Analysis Using Fuzzy Sets: Sufficient Conditions for Identifiability and Statistical Properties. *Information Sciences* 1: 149–80.

YASHIN, A. I., AND I. A. IACHINE. 1995. Genetic Analysis of Duration: Correlated Frailty Model Applied to Survival of Danish Twins. *Genetic Epidemiology* 12: 529–38.

YASHIN, A. I., I. A. IACHINE, AND J. R. HARRIS. 1999. Half of the Variation in Susceptibility to Mortality Is Genetic: Findings from Swedish Twin Survival Data. *Behavior Genetics* 29(1): 11–19.

ZADEH, L. A. 1965. Fuzzy Sets. *Information and Control* 8(3): 335–53.

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