

# Cohort Changes in Active Life Expectancy in the U.S. Elderly Population: Experience From the 1982–2004 National Long-Term Care Survey

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**Objectives.** To understand declines in chronic disability prevalence in the U.S. elderly population, we examined cohort changes in active life expectancy, a health measure relating population disability and longevity dynamics.

**Methods.** We computed active life expectancy and life expectancy using the six National Long-Term Care Surveys done from 1982 to 2004 and linked to continuous-time Medicare service data for the same time period by using a stochastic process model based on disability scores calculated using grade of membership analyses. We simultaneously estimated continuous-time disability dynamic and mortality functions to calculate life tables for specific disability states and for temporally changing mixtures of disability states.

**Results.** Disability dynamics, measured as changes in grade of membership scores, showed significant variation across two birth cohorts followed for 24 years. Disability dynamics and disability-specific hazard functions were significantly improved in the younger cohort (persons aged 65–74 in 1982).

**Discussion.** Our results, supporting the hypothesis of morbidity compression, indicate that younger cohorts of elderly persons are living longer in better health. The methods describe individual disability transitions and mortality and other factors associated with disability changes, making it possible to better evaluate interventions to promote future declines in disability.

**Key Words:** Active life expectancy—Disability dynamics—Grade of membership—Fokker-Planck.

ACTIVE life expectancy (ALE) is often used to measure the person-year burden of functional disability both in the U.S. elderly population and for international comparisons of developed countries with relatively high life expectancies and aging populations (Robine & Michel, 2004; Robine, Romieu, & Michel, 2003). The World Health Organization recommends ALE as an important health measure for comparing developed countries with aging populations because this measure better reflects health status and quality-of-life issues that, in those countries, may vary more than total life expectancy (LE).

Often ALE is calculated by using cross-sectional life table methods (Sullivan, 1971) applied to demographic data on mortality (e.g., national life tables) and age-specific disability prevalence rates calculated from national health surveys. Cross-sectional estimates of ALE calculated at several points in time can be used to track changes in the overall performance of the U.S. health care delivery system because they reflect the health and functional status of the entire U.S. elderly population at each date (e.g., Manton, Gu, & Lamb, 2006b; Mathers & Robine, 1997; Robine & Michel, 2004). Measures of health status other than disability can also be used to refine the decomposition of total LE. For example, chronic morbidity and acute health service use have been used as health state identifiers, as have various types of institutional and long-term care service use (Ogawa, 1982). Assuming the shape of the population age trajectories of survival and disability

intensity remain similar over time, period changes in LE and ALE may carry useful information about cohort health changes (Goldstein & Wachter, 2006).

Another important issue in calculating ALE is how the health or functional state (here defined in terms of loss of various self-maintenance functions) is defined. Many analysts have used simple disability measures (e.g., activities of daily living [ADLs], instrumental ADLs [IADLs] based on counts of disabilities made at specific points in time; Manton et al., 2006b). Others have used various interview and subjective expert elicitation procedures to determine the perceived utility of being in a specific health-impaired state defined across a 0 (death) to 1 (healthy) scale. Such procedures have been used to calculate quality-adjusted life years, disability-adjusted life years, and health-adjusted life years (e.g., Äijänseppä et al., 2005).

Much of the interest in ALE in the United States is attributable to recent changes in adult LE. From 1954 to 1968, male LE in the United States declined due to increases in cardiovascular disease (CVD) risks. After 1968, male LE began to increase due to declines in male CVD mortality. By 1980 it became clear that the male LE increase was not a fluke—it was due to persistent declines in both heart disease and stroke mortality.

A general acceptance that disability declines were occurring in the U.S. elderly population did not occur until data from the

1994 National Long-Term Care Survey (NLTCs) became available and were analyzed (Freedman & Soldo, 1994; Manton, Corder, & Stallard, 1997; Singer & Manton, 1998). Scientific acceptance that disability declines were occurring internationally came even later (e.g., Robine et al., 2003). In 1983, a strategy was implemented to increase the normal retirement age for Social Security from age 65, starting in 2003, to age 67 by 2027. Recently it has been proposed that the normal retirement age might be raised even further, for example to age 69 in Britain (by 2050) and to age 73.2 in Japan (under current survival conditions). Manton, Lowrimore, Ullian, XiLiang, and Tolley (2007) estimated the effect of extending the Social Security eligibility age coupled with the decline in disability on workforce participation.

Though lacking detailed longitudinal health data in 1982, some authors nonetheless argued that the health problems of modern industrial societies at the third stage of the epidemiological transition would be dominated by chronic degenerative diseases (Omran, 1971) and a failure of medicine to effectively treat chronic disease (McKinlay & McKinlay, 1977). Other investigators suggested that the fundamental processes of chronic disease were not being altered—only LE in disabled states was being increased (Gruenberg, 1977; Kramer, 1980). In contrast, Fries (1980) suggested that ALE was increasing more rapidly than total LE, producing “morbidity compression.” Manton (1989) argued that morbidity and disability declines and longevity increases were all processes positively correlated in individuals so that there would be a dynamic (over time and age) equilibrium of morbidity- and disability-free life span with total longevity.

Unfortunately, nationally representative longitudinal data on U.S. morbidity and disability in the elderly population only began to become available in the 1980s (Feldman, 1983), so empirical longitudinal tests of the different models could not be done until the 1990s. Wilkins and Adams (1983) made early estimates of ALE for Canada. Others who made ALE estimates for the United States using other data, including longitudinal data from local studies, include Crimmins and Saito (2001), Guralnik, Land, Blazer, Fillenbaum, and Branch (1993), and Branch and colleagues (1991).

Manton and Land (2000) calculated ALE measures by using variants of the stochastic process procedures employed herein (Manton, Stallard, & Liu, 1993; Manton, Stallard, & Singer, 1992) for the 1982 to 1994 NLTCs. That analysis, however, did not explicitly deal with cross-cohort variation, because the 14-year time series (1982–1996) did not contain sufficient longitudinal follow-up to make meaningful cross-cohort comparisons. In general, those longitudinal results did point to morbidity compression occurring in the U.S. elderly population—a conclusion also strongly suggested by long-term cross-sectional analyses made using the 1982 to 1999 NLTCs (Manton et al., 2006b). Selected studies showed increases in ALE in other countries (Robine et al., 2003). For example, in a 15-year study of morbidity compression, morbidity expansion, and dynamic equilibrium in New Zealand, the evidence supported the more general dynamic equilibrium model (Graham, Blakely, Davis, Sporle, & Pearce, 2004). Jagger and colleagues (2007) in a British survey found evidence suggesting the potential for significant future morbidity compression.

We used data from the 1982 to 2004 NLTCs (and individual mortality experience to 2006) here to estimate cohort-specific changes in ALE and LE to determine whether part of the decline in chronic disability prevalence (Manton & Gu, 2001; Manton, Gu, & Lamb, 2006a) is attributable to cohort differences as opposed to period effects due to, for example, revisions of Medicare benefits. This makes the linkage of NLTCs to individual continuous-time Medicare expenditure and service use records important, both methodologically (e.g., the exact dates of death to be used in the ALE calculations at advanced ages taken from Medicare records are believed to be more accurate than the ages of death from death certificates; Kestenbaum, 1992; Manton & Gu, 2007) and because Medicare records (e.g., *International Classification of Diseases–9* diagnoses) contain significant additional health and health service use information recorded on a continuous-time basis to supplement the NLTCs health data and to explicate the relation of changes in Medicare and Medicaid use to ALE changes. For example, Medicare records contain the exact dates of use of skilled nursing facilities. Dates of diagnosis of Alzheimer’s disease and other dementing illnesses have been recorded in detail since 1991.

Many benefit changes occurred in the Medicare and Medicaid programs from 1982 to 2006. In 1983–1984, a Medicare prospective payment system was introduced to reimburse acute hospital stays; in 1989, Medicare regulations regarding home health and skilled nursing facility use were relaxed (e.g., *Duggan v. Brown*, 1988). In the Balanced Budget Act of 1997 (refined in 1999), innovations were made in Medicare reimbursement of home health agency and skilled nursing facility benefits (e.g., a skilled nursing facility prospective payment system was instituted that emphasized the provision of rehabilitation services based on Resource Utilization Groups, Version 3). In 2003, the Medicare Modernization Act was passed, which set the stage for payment for performance provisions and Medicare Part D—payment for outpatient drug use. Because these benefit changes target groups with different health states, the model must be capable of describing temporal interactions in its dynamic components.

Additionally, many health improvements occurred due to period-specific public health interventions (e.g., the health effects of reductions in smoking, initially stimulated by the 1962–1964 U.S. Surgeon General’s reports, became strongly manifest in the 1990s). Cutler, Landrum, and Stewart (2006) attributed much of the recent decline in chronic disability to the diffusion of medical advances in the treatment of major circulatory diseases. Lichtenberg (2007) suggested that advances in chemotherapy have contributed to recent (e.g., post-1990) declines in cancer mortality.

By determining cross-cohort differences in the disability and mortality dynamics used to calculate ALE, experts can use those differences in parameters to improve long-range forecasts and projections and to better identify shift points in the processes. By assessing cohort-specific contributions to disability, researchers will have a stronger empirical base on which to examine the health mechanisms associated with recent U.S. disability declines and to improve intervention strategies (Manton, Lamb, & Gu, 2007).

Table 1. Percentage of Assisted Living and Non-Assisted Living by Disabled Group

Group	Subgroup	NLTCs		
		1994	1999	2004
Assisted living	Nondisabled		41.30	39.06
	Comm disabled		43.53	32.63
	Institution		15.17	28.31
	Total		2.30	3.42
Non-assisted living	Comm nondisabled	76.75	80.32	82.24
	Comm ADLs	12.76	12.32	14.56
	Comm IADL only	4.50	3.21	2.37
	Institution	5.98	4.16	3.26
	Total	100.00	97.70	96.58

Note: NLTCs = National Long-Term Care Survey; Comm = community; ADL = activity of daily living; IADL = instrumental ADL.

## DATA

The NLTCs is a large, nationally representative longitudinal survey started in 1982. It was conducted again 2 years later in 1984 and then every 5 years to 2004. Interviewing was done in two stages. In 1982, a sample large enough to identify about 6,000 chronically disabled (90+ days) individuals was screened. In subsequent survey years, the sample was maintained at roughly 20,000 persons. In later NLTCs, only persons in the sample who had not previously expressed chronic disability, or who were newly sampled, were screened. All persons who had evidenced chronic disability in a prior NLTCs, or who were in institutional residence, were automatically “screened in” at all subsequent waves starting in 1984; hence, both positive and negative changes in disability can be assessed. For all persons identified as chronically disabled, and for persons identified as being in a special nondisabled sample drawn from persons who screened out as not disabled (starting in 1994 and repeated in 1999 and 2004), a detailed community residential or institutional interview was conducted.

In each NLTCs from 1984 on, a supplementary sample was drawn of Medicare enrollees who had passed age 65 and become Medicare-eligible since the prior NLTCs. This supplementary sample of 5,000 to 5,500 persons aged 65 to 69 roughly maintained the cross-sectional sample size of about 20,000 persons in the next NLTCs by “replacing” the loss of a similar number of deaths at all ages occurring since the prior wave. This also ensured that the entire Medicare elderly (65+) population was represented in each NLTCs. Starting in 1994, oversamples of persons aged 95+ were drawn (i.e.,  $N = 540$  in 1994;  $N = 600$  in 1999;  $N = 1,584$  in 2004). This improved the precision of disability and mortality estimates at extreme ages for which there were relatively few data on health transitions and individual mortality risks but for which chronic disability changes may play an important role in long-term (75-year) forecasts of Medicare and Medicaid expenditures (Manton, Lamb, et al., 2007).

The NLTCs screener and community interview sections on disability measures and health conditions have been preserved largely unchanged from 1982 to 2004. In addition, the basic sample structure of the NLTCs has been maintained (i.e., the 173 primary sampling units drawn from the full set of 376

primary sampling units defined for the Current Population Survey were used for all NLTCs). For those 173 primary sampling units, a list sample of elderly Medicare enrollees was drawn. Thus, 100% of persons drawn for any NLTCs are linked to a continuous-time history of Medicare costs and service use and, since 1991, *International Classification of Diseases-9* diagnoses on both Part A and Part B.

Screener response rates remained high (+95%) across all NLTCs until 2004. In 2004, the NLTCs response rate dropped to 91% due to difficulty tracking persons aged 65 to 74 with low levels of Medicare service use. To deal with possible response rate bias in longitudinal disability rate estimates, the Medicare service use and expenditure files, which are available for 100% of the NLTCs sample (both respondents and non-respondents) in all waves, were used to adjust for temporally emergent bias in disability prevalence rate estimates. The correlation of the increased 2004 nonresponse rate with Medicare service use in 2004 is informative about the direction of age-specific bias in disability prevalence estimates and can be used to reduce such bias. The Medicare service use and cost data adjustments were applied not only in 2004, but to all prior waves of the survey to consistently adjust for bias in disability rate estimates over time (Manton, et al., 2006a). We computed the weights used for this analysis with the adjusted data.

## METHODS

The NLTCs makes 27 measures of impairment in ADLs (Katz, Ford, Moskowitz, Jackson, & Jaffe, 1963), IADLs (Lawton & Brody, 1969), physical performance measures (Nagi, 1976), and sensory function (vision). For each ADL and IADL, a battery of ancillary traits is assessed. To track changes in these measures, we used a multivariate procedure to identify the  $K$  disability dimensions represented by those 27 variables measured at six points in time (1982, 1984, 1989, 1994, 1999, 2004). This procedure reduces the effects of measurement error by using the correlations (redundancy) of a relatively large number of measures (dimensions) to define a smaller number of more reliable dimensions.

We did this by using grade of membership (GoM) procedures (Manton & Gu, 2007; Manton et al., 1993; Manton, Stallard, & Woodbury, 1991; Manton, Woodbury, & Tolley, 1994; Stallard, 2005), whereby convexly constrained scores (i.e., scores constrained to the range of 0–1 and to sum to 1 for each person) were estimated for each of the  $K$  dimensions (profiles or pure types) underlying the 27 measures. The longitudinal (for the six NLTCs) GoM model equations (i.e., the cross-temporal measurement models) are as follows:

$$\text{Prob}(x_{ij}(t) = l) = \sum_{k=1}^K (g_{ik}(t) \cdot \lambda_{kjl}(\cdot)), \quad (1)$$

where  $\sum_{k=1}^K g_{ik}(t) = 1$  and  $0 \leq g_{ik}(t) \leq 1$  at each measurement time  $t$  ( $t = 1, \dots, 6$ ),  $\sum_{l=1}^L \lambda_{kjl}(\cdot) = 1$  over all times of measurement, and  $x_{ij}(t)$  is the observed response vector.

Thus, a consistently (1982–2004) defined [by  $\lambda_{kjl}(\cdot)$ ] set of disability dimensions was estimated using data from all six NLTCs, within which the cross-time variation of the vector of  $K$  functional scores  $\hat{g}_i(t)$  for individuals was tracked (Abonyi, Babuska, & Szeifert, 2001). The number of dimensions,  $K$ , was determined by examining the difference in the likelihood

Table 2. Estimates of ( $\lambda_{kij} \times 100$ ) Describing Six Dimensions Identified From 27 Measures of the Ability to Perform Specific Activities in the 1982–2004 National Long-Term Care Survey Community Interviews

Characteristic	Subgroup	%	Percentage With Indicated Characteristics for Pure Type $k$ ( $\lambda_{kij}$ )					
			Active	Modest Impairment	Moderate Impairment	IADL	ADL	Frail
Needs help with (ADL)	Eating	10.2 (10.6)	0.0	0.0	0.0	0.0	0.0	82.2 (80.8)
	Getting in/out of bed	26.8 (27.6)	0.0	0.0	0.0	0.0	100.0	100.0
	Getting around inside	38.2 (40.3)	0.0	0.0	0.0	0.0	100.0	100.0
	Dressing	19.2 (20.8)	0.0	0.0	0.0	0.0	0.0	100.0
	Bathing	41.8 (43.9)	0.0	0.0	0.0	3.3 (0.0)	100.0	100.0
	Using toilet	24.9 (24.8)	0.0	0.0	0.0	0.0	94.3 (71.3)	100.0
Bedfast		0.7 (0.8)	0.0	0.0	0.0	0.0	0.0	5.1 (5.4)
No inside activity		1.4	0.0	0.0	0.0	0.0	0.0	9.6 (9.1)
Uses wheelchair		5.5 (6.2)	0.0	0.0	0.0	0.0	11.5 (16.0)	26.1 (22.5)
Needs help with (IADL)	Heavy housework	60.1 (67.6)	0.0	100.0	100.0	100.0	100.0	100.0
	Light housework	19.7 (21.7)	0.0	0.0	0.0	45.3 (42.7)	0.0	100.0
	Laundry	30.8 (35.5)	0.0	0.0	0.0	100.0	0.0	100.0
	Cooking	23.3 (25.9)	0.0	0.0	0.0	100.0	0.0	100.0
	Grocery shopping	42.1 (48.6)	0.0	0.0	0.0	100.0	100.0	100.0
	Getting about outside	48.7 (55.5)	0.0	0.0	0.0	100.0	100.0	100.0
	Traveling	43.7 (49.3)	0.0	0.0	0.0	100.0	100.0	85.2 (100.0)
	Managing money	20.8 (22.9)	0.0	0.0	0.0	100.0	0.0	100.0
	Taking medicine	20.4 (21.1)	0.0	0.0	0.0	100.0	0.0	100.0
	Telephoning	11.6 (14.6)	0.0	0.0	0.0	67.3 (89.8)	0.0	58.9 (75.8)
How much difficulty do you have Climbing 1 flight stairs	None	29.8 (22.1)	100.0	0.0	0.0	0.0	0.0	0.0
	Some	27.8 (29.2)	0.0	100.0	0.0	0.0	0.0	0.0
	Very difficult	24.3 (28.6)	0.0	0.0	100.0	100.0	45.6 (47.6)	7.1 (13.0)
	Cannot at all	18.1 (20.2)	0.0	0.0	0.0	0.0	54.4 (52.4)	81.1 (87.0)
Bending for socks	None	51.5 (46.3)	100.0	0.0	0.0	100.0	0.0	0.0
	Some	25.2 (27.0)	0.0	100.0	49.1 (46.6)	0.0	54.1 (51.0)	12.5 (10.3)
	Very difficult	14.1 (16.6)	0.0	0.0	50.9 (53.4)	0.0	45.9 (49.1)	22.5 (23.3)
	Cannot at all	9.2 (10.1)	0.0	0.0	0.0	0.0	0.0	65.0 (66.5)
Holding 10-lb. package	None	40.4 (33.8)	100.0	0.0	0.0	0.0	0.0	0.0
	Some	17.4 (17.7)	0.0	100.0	0.0	0.0	0.0	0.0
	Very difficult	13.1 (14.5)	0.0	0.0	74.7 (74.6)	69.9 (59.1)	0.0	0.0
	Cannot at all	29.1 (34.0)	0.0	0.0	25.0 (25.4)	30.1 (40.9)	100.0	100.0
Reaching over head	None	63.4 (58.8)	100.0	100.0	0.0	100.0	100.0	0.0
	Some	18.4 (20.1)	0.0	0.0	65.4 (62.6)	0.0	0.0	21.7 (22.0)
	Very difficult	10.7 (12.7)	0.0	0.0	28.4 (30.0)	0.0	0.0	30.7 (32.6)
	Cannot at all	7.5 (8.4)	0.0	0.0	6.2 (7.4)	0.0	0.0	47.6 (45.4)
Combing hair	None	76.1 (73.6)	100.0	100.0	0.0	100.0	100.0	0.0
	Some	13.6 (14.9)	0.0	0.0	82.7 (80.7)	0.0	0.0	28.1 (29.1)
	Very difficult	5.4 (6.3)	0.0	0.0	17.3 (19.3)	0.0	0.0	28.6 (29.1)
	Cannot at all	4.9 (5.2)	0.0	0.0	0.0	0.0	0.0	43.3 (41.8)
Washing hair	None	63.4 (58.7)	100.0	100.0	0.0	100.0	100.0	0.0
	Some	12.9 (13.9)	0.0	0.0	70.7 (67.5)	0.0	0.0	0.0
	Very difficult	7.5 (8.5)	0.0	0.0	29.3 (32.5)	0.0	0.0	11.8 (8.9)
	Cannot at all	16.1 (18.8)	0.0	0.0	0.0	0.0	0.0	88.2 (91.1)
Grasping small objects	None	70.6 (67.8)	100.0	100.0	0.0	100.0	100.0	19.0 (19.6)
	Some	18.2 (19.5)	0.0	0.0	76.5 (75.0)	0.0	0.0	27.8 (27.6)
	Very difficult	8.0 (9.3)	0.0	0.0	23.5 (25.0)	0.0	0.0	27.8 (28.9)
	Cannot at all	3.2 (3.4)	0.0	0.0	0.0	0.0	0.0	25.3 (23.9)
Can you see well enough to read a newspaper?	Yes	78.0 (75.7)	100.0	100.0	100.0	0.0	100.0	51.4 (49.2)

Note: Coefficients in parentheses are from 1982 to 1994 analysis if different from current analysis. ADL = activity of daily living; IADL = instrumental ADL.

function used to estimate (1) (approximately distributed as  $1/2 \chi^2$ ) as  $K$  is successively increased by 1. As in previous analyses of NLTCs disability measures, an analysis of community residents (assigning the relatively homogeneous institutional residents to a seventh disability profile where  $g_{i7} = 1$ ; all other  $g_{ik} = 0$ ) found the optimum value of  $K$  to be 6.

Erosheva, Fienberg, and Joutard (2007) derived a model that they described as a latent class GoM model. Their estimation was by Bayesian techniques; ours was by a frequency approach. As they stated, ours is a continuous mixture rather than a discrete mixture model. Because class membership in our model is permitted to be partial, it is more parsimonious.



Table 3. Person's Age in Each Survey Year Based on Birth Year

Cohort Birth Year	Survey Year											
	1982		1984		1989		1994		1999		2004	
	Age	N <sup>a</sup>	Age	N <sup>a</sup>	Age	N <sup>a</sup>	Age	N <sup>a</sup>	Age	N <sup>a</sup>	Age	N <sup>a</sup>
1893–1897	85–89	1,697	87–91	1,208	92–96	539	97–101	381	102–106	144	107–111	443
1898–1902	80–84	2,783	82–86	2,153	87–91	1,213	92–96	658	97–101	401	102–106	274
1903–1907	75–79	4,037	77–81	3,389	82–86	2,342	87–91	1,211	92–96	704	97–101	762
1908–1912	70–74	5,108	72–76	4,512	77–81	3,516	82–86	2,303	87–91	1,267	92–96	1,128
1913–1917	65–69	6,001	67–71	5,591	72–76	2,925	77–81	3,536	82–86	2,466	87–91	1,346
1918–1922	60–64		62–66		67–71	5,645	72–76	5,386	77–81	5,430	82–86	3,623
1923–1927	55–59		57–61		62–66		67–71	3,941	72–76	2,958	77–81	3,019
1928–1932	50–54		52–56		57–61		62–66		67–71	4,272	72–76	3,069
1933–1937	45–49		47–51		52–56		57–61		62–66		67–71	4,626

Notes: The age ranges marked in bold below the horizontal mark are not yet eligible for the surveys. The step mark traces the 67–71 cohort through four survey waves.

<sup>a</sup>At start of observation.

Instead of the 9 or 10 profiles Erosheva and colleagues selected to describe NLTCs disability, we have usually found only 6 to be necessary (e.g., Berkman, Singer & Manton, 1989).

To ensure that the meaning of institutional status remained constant over time, we determined it from two variables. One is residence type, where a person can be a resident in a group dwelling. The second variable describes the disability state of the person. Thus, a resident of an assisted living facility (ALF; which emerged in large numbers in 1999) can be (a) in an independent living situation and not disabled, (b) in an independent living situation and disabled, (c) in a nursing home bed (i.e., with health care available on a 24-hr basis). In Table 1 we present the distribution of the population in these different states in 1994, 1999, and 2004.

The proportion of people in ALFs in nursing home beds grew dramatically from 1994 to 1999 (i.e., after the introduction of the Balanced Budget Act of 1997). It grew another 50% from 1999 to 2004 (from 0.81 million to 1.24 million cases). The proportion of ALF cases in nursing home beds increased from 1999 to 2004, suggesting that the intensity of care in ALFs was increasing. Thus, the definition of the

(nursing home bed) institutional population defined for Type 7, by using two traits, was adjusted to reflect the rapid growth of ALFs from 1994 to 2004.

The substance of the six community disability dimensions is described by comparing values of the  $\lambda_{kjl}$  relative to the cross-NLTCs average probability for each of the 27 disability measures (see, e.g., Berkman et al., 1989). In Table 2, estimates of  $\lambda_{kjl}$  are presented for each of six disability profiles, with a short characterization of each presented as column headings. In Table 2, many of the  $\lambda$  coefficients estimated from the 1982 to 2004 data exactly match the results of the GoM analyses of the 1982 to 1994 data presented in Manton and Land (2000). In Table 2 we present coefficients (in parentheses) for only those  $\lambda$  estimates in Manton and Land that are not identical to the 1982 to 2004 results. There are very few substantial differences, emphasizing the temporal stability of the GoM  $\lambda$  estimates in addition to their dimensional stability. This stability is consistent with large-scale simulation studies of the ability of GoM to accurately extract parametric structures from “noisy” high-dimensional categorical data (Kovtun, Akushevich, Manton, & Tolley, 2007). This is likely due to the simple structure constraints ( $\lambda_{kjl}$  set to convex space boundaries) imposed—especially on the binary ADL and IADL variables.

Persons exactly like the first disability profile are not chronically disabled and have no physical limitations (i.e., the  $\lambda_{kjl}$  in Table 2 indicates not needing help with any ADL or IADL and no difficulty with any of the eight physical activities).

The second and third disability profiles show little ADL or IADL disability but manifest modest amounts of physical impairment. The second profile has slightly less impairment than the third. The fourth profile manifests primarily IADL limitations, showing significant physical impairment only with climbing stairs and holding a 10-lb. package. Profiles 5 and 6 show significant ADL impairment in addition to difficulty with physical performance measures (Nagi, 1976), with the sixth profile being the most frail. The sixth profile is the community group most similar to institutional residents (seventh profile).

Though each of these profiles is substantively meaningful, and can be viewed as quantitatively defined by a profile of specific traits, the estimates of the mixing coefficients, the  $g_{ik}$ ,

Table 4. Life Expectancy of the National Long-Term Care Survey Cohort Aged 65 and Older in 1982

		Estimates From SSA Life Tables	
Age	GoM Model	Period	Cohort (Birth Year 1917)
Men			
65	15.01	14.23	14.91
67	13.77	13.21	13.74
72	10.94	10.80	11.02
77	8.50	8.48	8.55
82	6.51	6.34	6.37
87	5.00	4.58	4.62
Women			
65	18.85	18.48	18.90
67	17.41	17.16	17.02
72	14.01	14.02	13.99
77	10.92	10.85	10.83
82	8.21	7.98	8.05
87	6.01	5.71	5.76

Note: SSA = Social Security Administration; GoM = grade of membership.

Table 5.  $\theta$  Difference Between Disability Dynamic and  $\theta$  Only (Gompertz) Models by Cohort and Gender

		Women				Men			
		$\theta$ Difference Between				$\theta$ Difference Between			
		65–74	75–84	Two Cohorts	$t$	65–74	75–84	Two Cohorts	$t$
Disability dynamic model	$\theta$ (%)	6.55	7.55	1.00	7.7	6.24	7.01	0.77	4.3
Gompertz	$\theta$ (%)	9.33	10.37	1.04	8.0	7.73	8.59	0.86	4.8
( $\mu_0 e^{\theta t}$ )	(SE)	(0.08)	(0.10)	(0.13)		(0.08)	(0.16)	(0.18)	
$\theta$ Difference between two models (%)		–2.78	–2.82			–1.49	–1.58		
$t$		–24.6	–20.0			–13.3	–7.1		
$\theta$ Difference over the Gompertz model (%)		29.8	27.2			19.3	18.4		

are not probabilities of a case being exactly in one of the  $K$  groups. Because the  $g_{ik}$ s are bounded by 0 or 1, the disability space defined by the  $\lambda_{kjl}$  describes the boundaries of a convex space as opposed to, for example, a cluster analysis, where the  $\lambda_{kjl}$ s define the centroids of groups with cases being both within and external to the space bounded by the centroids. In cluster, or latent class, analyses (Lazarsfeld & Henry, 1968), the probabilities of being in a group are based on the distance of the case to the centroid in the J-variable measurement space, where the distances to the centroids on the measurements are normed relative to the dispersion (and correlation) of the variables (e.g., Mahalanobis  $D^2$  statistic). In GoM, the convexity constraints require the  $g_{ik}$  to reconstruct the observed traits of the case as a weighted sum of the profiles defining the convex space boundaries. By defining a “bounding” convex space, the  $\lambda_{kjl}$ s have greater stability because a simpler parametric structure (more  $\lambda_{kjl}$ s are 0 or 1) is defined and  $g_{ik}(t)$ s are strictly constrained to the interior of the convex space.

The  $g_{ik}(t)$ s, estimated by using maximum likelihood procedures, can be used in extended Fokker-Planck equations (Frank, 2005; Risken, 1996) to statistically estimate the disability dynamic and mortality parameters necessary for cohort ALE calculations (Manton et al., 1992). The Fokker-Planck equation describes changes with time of the population distribution of scores on the  $K$  disability profiles, where the distribution changes as a function of fixed (e.g., age and elapsed time) and stochastic (e.g., prior disability state and age- and disability-specific mortality) effects. Fuzzy set scores are frequently used as state variables in nonlinear stochastic process models in engineering studies of complex, nonlinear systems. The properties of the process in the original measurement space can be shown to be preserved by the ancillary process defined over the reduced dimension, filtered fuzzy state space (Gutierrez, 1994).

To estimate the parameters of the Fokker-Planck equation we use a generalization of the likelihood function in Manton and Stallard (1988) that describes changes over time in the  $g_{ik}(t)$  estimated using the GoM model, where changes in the distribution of the  $g_{ik}(t)$  are also caused by systematic mortality selection (i.e., the risk of loss of a given case due to mortality is a systematic function of the  $g_{ik}(t)$ , time, and age). The data on exact times to death are provided in the linked Medicare records.

In the simplest case (i.e., for a Gaussian Markov process), the likelihood function can be decomposed into three terms that can be independently estimated (i.e., the “initial” fuzzy state distribution, the auto-regressive process of the  $g_{ik}(t)$ , and

a quadratic mortality function). In the case of a process over the convex space of GoM disability scores, interaction terms must be added to the diffusion term (i.e., the process is no longer Markovian, but is semi-Markovian; Frank, 2005; Manton et al., 1992), with the diffusion process no longer Gaussian (i.e., it is “anomalous” with higher order moments and cumulants due to

Table 6. Quadratic Mortality Functions for Two Male and Female Cohorts Estimated From the 1982–2004 National Long-Term Care Survey

Cohort	PT 1	PT 2	PT 3	PT 4	PT 5	PT 6	PT 7
Aged 65–74 in 1982							
Men							
PT 1	0.05	0.06	0.08	0.08	0.07	0.10	0.09
PT 2		0.07	0.10	0.09	0.09	0.12	0.11
PT 3			0.14	0.13	0.12	0.16	0.15
PT 4				0.13	0.12	0.16	0.15
PT 5					0.11	0.15	0.14
PT 6						0.20	0.19
PT 7							0.17
Women							
PT 1	0.04	0.04	0.05	0.07	0.05	0.08	0.08
PT 2		0.04	0.05	0.07	0.05	0.08	0.08
PT 3			0.07	0.10	0.06	0.11	0.10
PT 4				0.14	0.09	0.16	0.15
PT 5					0.06	0.10	0.10
PT 6						0.18	0.17
PT 7							0.16
Aged 75–84 in 1982							
Men							
PT 1	0.05	0.07	0.08	0.08	0.07	0.10	0.09
PT 2		0.09	0.10	0.10	0.08	0.13	0.11
PT 3			0.11	0.12	0.10	0.14	0.13
PT 4				0.12	0.10	0.15	0.13
PT 5					0.08	0.13	0.11
PT 6						0.19	0.16
PT 7							0.14
Women							
PT 1	0.04	0.05	0.04	0.06	0.05	0.07	0.07
PT 2		0.06	0.05	0.07	0.07	0.09	0.08
PT 3			0.05	0.06	0.06	0.08	0.08
PT 4				0.08	0.08	0.10	0.10
PT 5					0.08	0.10	0.10
PT 6						0.13	0.13
PT 7							0.12

Note: PT = pure type.

Table 7. 2-Year Transition Coefficients (D[24m]) Between Seven Profiles Evaluated at Age 75 for Two Male and Female Cohorts

Age 77	Profile for Age 75						
	PT 1	PT 2	PT 3	PT 4	PT 5	PT 6	PT 7
<b>Men</b>							
Aged 65–74 in 1982							
PT 1	0.93	0.02	0.01	0.01	0.01	0.01	0.02
PT 2	0.25	0.52	0.04	0.03	0.05	0.05	0.05
PT 3	0.19	0.09	0.47	0.06	0.07	0.07	0.06
PT 4	0.22	0.05	0.05	0.48	0.06	0.08	0.07
PT 5	0.15	0.07	0.05	0.04	0.57	0.06	0.07
PT 6	0.18	0.05	0.04	0.05	0.05	0.57	0.06
PT 7	0.18	0.03	0.02	0.02	0.03	0.02	0.70
Aged 75–84 in 1982							
PT 1	0.87	0.02	0.01	0.01	0.03	0.03	0.03
PT 2	0.18	0.50	0.05	0.05	0.10	0.08	0.04
PT 3	0.18	0.10	0.41	0.07	0.10	0.10	0.05
PT 4	0.17	0.07	0.06	0.46	0.07	0.10	0.07
PT 5	0.20	0.07	0.05	0.04	0.50	0.08	0.05
PT 6	0.16	0.05	0.03	0.04	0.06	0.58	0.07
PT 7	0.17	0.01	0.01	0.02	0.02	0.02	0.76
<b>Women</b>							
Aged 65–74 in 1982							
PT 1	0.90	0.02	0.01	0.01	0.02	0.01	0.03
PT 2	0.21	0.49	0.08	0.04	0.08	0.05	0.05
PT 3	0.18	0.09	0.47	0.04	0.10	0.07	0.05
PT 4	0.19	0.08	0.07	0.44	0.09	0.08	0.06
PT 5	0.17	0.07	0.07	0.04	0.54	0.07	0.04
PT 6	0.15	0.05	0.07	0.04	0.10	0.52	0.07
PT 7	0.16	0.02	0.02	0.01	0.02	0.02	0.75
Aged 75–84 in 1982							
PT 1	0.85	0.03	0.02	0.02	0.03	0.02	0.03
PT 2	0.38	0.21	0.08	0.06	0.12	0.06	0.09
PT 3	0.32	0.10	0.19	0.08	0.14	0.09	0.09
PT 4	0.32	0.10	0.09	0.16	0.13	0.09	0.11
PT 5	0.31	0.10	0.09	0.07	0.28	0.10	0.07
PT 6	0.28	0.08	0.08	0.07	0.15	0.27	0.08
PT 7	0.22	0.04	0.03	0.04	0.04	0.03	0.60

Note: PT = pure type.

the interaction terms representing historical traces of prior score distributions).

The dynamic equation for each of  $K$  disability dimensions, in a cohort tracked on a monthly basis, is

$$g_{ik}(m+1) = d_k(m)g_{ik}(m) + \varepsilon_{ik}(m), \quad (2a)$$

where the expected value of the normalized (see Manton et al., 1992) error  $\varepsilon_{ik}(m) = 0$  and where  $m$  is the age of the cohort at a specific time. This can be written for  $K$  simultaneous equations as

$$\tilde{g}_{i \cdot (m+1)} = D_m \tilde{g}_{i \cdot m} + \tilde{\varepsilon}_{i \cdot m}, \quad (2b)$$

where  $D$  is a  $K \times K$  convexly constrained (individual scores add to 1) disability score change matrix with finer time increments (e.g., monthly,  $m = 60$ ) between surveys. The expected value of  $\varepsilon_{ik \cdot m} \varepsilon_{ik \cdot m}^T$  is the  $K \times K$  diffusion matrix,  $\Sigma_m$ , reflecting the effects of stochastic shocks on the multidimensional disability process.

To reflect the impact of mortality on the trajectory of the disability scores, one estimates a generalized quadratic mortality function using monthly disability scores (estimable from Equation 2b) and the date of death for each individual (grouped by month) derived from Medicare vital statistics records. The hazard over the period  $m$  to  $m+1$  for a cohort is

$$\mu(m, m+1) = (\tilde{g}_{i \cdot m}^T Q_m \tilde{g}_{i \cdot m}) e^{\theta m}, \quad (3)$$

where the  $\tilde{g}_{i \cdot m}^T$  for a person at a given time is generated from Equation 2b. The exponential term  $e^{\theta m}$  in Equation 3 represents age-related (nonlinear) increases in mortality within a cohort not captured by the  $\tilde{g}_{i \cdot m}$  dynamics. The hazard matrix,  $Q_m$ , reflects the dependence of an individual's mortality risk on the seven disability scores evaluated at time  $m$  for an individual using Equation 2b. Because the first profile represents healthy persons with no disabilities, it can be interpreted as the origin of the  $K$ -dimensional disability space in Equations 2b and 3.

Statistical estimates of the parameters in Equations 2b and 3 (i.e.,  $Q$ ,  $D$ , and  $\Sigma$ ) were used to calculate disability state-specific life table parameters by successively (for each life table age interval) calculating the five equations that define a cohort life table whose parameters are conditioned on disability-specific mortality selection and disability dynamics (Manton et al., 1992, 1993). Life table functions and  $g_{ik}(m)$  distributions can be estimated for the total population—or for an individual exactly starting in a given disability state at a selected age.

## RESULTS

To estimate the cohort model and calculate disability-specific life tables, one must organize the NLTCs and Medicare data on a cohort basis; Table 3 illustrates this.

Each row in Table 3 represents a 5-year birth cohort with the age of persons in the cohort at each survey. These 5-year cohorts can be further aggregated to provide sufficient data for a cohort to estimate the dynamics and mortality coefficients for the disability–mortality process for which we calculated life tables. We use the 5-year grouping in Table 3 for convenience in presentation. One can see that, by 2004, the  $N$  for the group aged 80 to 84 in 1982 was getting small ( $N = 274$  for persons aged 102–106), so we grouped two 5-year cohorts for our analyses to have better parameter estimates for advanced ages.

Using the average experience of all NLTCs cohorts born 1917 or earlier, followed for 24 years, we produced the LE estimates in Table 4 using the life table model with disability dynamics and an age-dependent specific hazard function. Those estimates were close to the Social Security cohort LE estimated for persons born in 1917 (Bell & Miller, 2002). Men aged 65 in the NLTCs cohort model had an LE of 15.01 years compared to 14.91 years for the Social Security life tables. The Social Security period life table estimate for 1982, as expected, was lower (i.e., 14.23 years). Thus, the NLTCs sample and linked Medicare mortality data reproduced well official Social Security Administration survival estimates for both men and women.

To calculate LE for a cohort, one needs estimates of the age-dependence parameter,  $\theta$ , and of the disability-dependent cohort mortality function,  $Q$ . We estimated these parameters (see Table 5) by using two different model specifications. First, in Table 5 we estimated the value of  $\theta$  with no individual

Table 8. Life Tables for Male and Female Cohorts in 1982 With Age- and Profile-Specific Mean Disability Scores

			Average $g_{ik}(m)$						
Age	Lx	Ex	PT1	PT2	PT3	PT4	PT5	PT6	INST
Men									
65–74									
75	100000	9.54	0.871	0.034	0.015	0.017	0.022	0.025	0.016
80	70597	7.47	0.805	0.043	0.020	0.021	0.030	0.033	0.049
85	42033	5.88	0.765	0.047	0.022	0.026	0.042	0.042	0.056
90	20050	4.77	0.734	0.046	0.023	0.031	0.055	0.049	0.063
95	7562	4.07	0.815	0.045	0.012	0.025	0.041	0.029	0.033
100	2391	3.30	0.884	0.038	0.005	0.016	0.022	0.019	0.015
75–84									
75	100000	8.80	0.864	0.029	0.014	0.019	0.023	0.026	0.026
80	69042	6.61	0.785	0.039	0.019	0.025	0.03	0.039	0.063
85	38337	4.94	0.686	0.048	0.023	0.041	0.046	0.051	0.104
90	15625	3.80	0.669	0.048	0.025	0.032	0.05	0.054	0.121
95	4442	3.08	0.709	0.050	0.019	0.035	0.05	0.036	0.102
100	924	2.33	0.789	0.016	0.008	0.022	0.089	0.023	0.053
Women									
65–74									
75	100000	12.29	0.810	0.041	0.031	0.017	0.040	0.022	0.039
80	79909	9.73	0.724	0.053	0.035	0.024	0.057	0.035	0.070
85	56352	7.74	0.653	0.054	0.037	0.029	0.073	0.050	0.104
90	32907	6.51	0.599	0.053	0.036	0.040	0.093	0.054	0.126
95	16215	5.90	0.692	0.053	0.031	0.032	0.088	0.028	0.076
100	7670	4.99	0.797	0.050	0.024	0.020	0.066	0.010	0.033
75–84									
75	100000	11.59	0.829	0.035	0.025	0.020	0.044	0.023	0.024
80	81351	8.65	0.690	0.045	0.041	0.034	0.064	0.041	0.085
85	56739	6.27	0.540	0.055	0.043	0.047	0.085	0.060	0.169
90	30302	4.61	0.461	0.056	0.041	0.047	0.083	0.075	0.237
95	11367	3.54	0.462	0.051	0.036	0.043	0.080	0.078	0.250
100	2832	2.99	0.537	0.033	0.028	0.046	0.076	0.076	0.205

Note: Lx = number living to age x; Ex = expected life remaining at age x; PT = pure type; INST = Institutional.

variation in the  $g_{ik}(m)$  represented. This was equivalent to estimating a simple Gompertz function. Second, we estimated a more complex model in which we generated the dynamics of disability by using the transition equations in Equation 2b (i.e., the representation of disability as a constant [Gompertz proportionality] factor was replaced by estimation of a disability dynamic hazard matrix where the matrix of risk factor values at each age for each individual was generated by a K-dimensional disability dynamic model).

The addition of the disability dynamics (the set of K  $g_{ik}(m)$  estimated on a monthly basis) in the hazard function reduced the unexplained variation in mortality due to age ( $\theta$ ) for women aged 65 to 74 (in 1982) by 2.78 (29.8%,  $t = 24.6$ ) and for women aged 75 to 84 by 2.82 (27.2%,  $t = 20.0$ ). For men, the effects of disability on the age trajectory of cohort mortality were smaller (1.49 and 1.50) but still highly significant ( $ts = 13.3$  and 7.1). Comparisons can be made across cohorts for men and women both before and after adjusting for disability dynamics. The  $\theta$  differences for both male and female cohorts were similar in size (1.0 and 1.04 for women; 0.77 and 0.86 for men) whether disability dynamics were adjusted or not and were both highly significant. This suggests the effects of

disability on mortality independent of age are robust (i.e., reductions in  $\theta$  are large because much of the variation due to age is accounted for by the dynamics of disability).

To describe disability-dependent mortality, it is also necessary to examine the age-independent relation of the  $g_{ik}(m)$  to mortality (i.e., the matrix of risk coefficients  $Q$  driven by disability dynamics). These are in Table 6.

For nondisabled men and women (i.e., Type 1, persons with 1 on the first nondisabled profile), the mortality coefficients were the same for both cohorts (to the number of decimal places presented), although the cohort-specific age-dependence parameter,  $\theta$  (in Table 5), with disability dynamics was modestly higher for women (0.0655 and 0.0755 for women compared to 0.0624 and 0.0701 for men), indicating that female mortality increases faster with age in both cohorts. For disabled women (i.e., those with nonzero scores on Profiles 2 to 7), mortality, in both cohorts, was generally lower than for men.

To complete the description of the linked (simultaneously estimated) disability dynamic mortality processes, we need estimates of the dynamic matrix,  $D$  (Equation 2b), to produce disability-dependent cohort life tables. In Table 7 are male and female 2-year (24-month) disability dynamic estimates (matrix



Table 9. Life Expectancy by Cohorts for Different Profiles

Cohort	Age	General	PT 1	PT 2	PT 3	PT 4	PT 5	PT 6	PT 7
<b>Men</b>									
65–74									
	65	14.76	17.01	13.80	8.74	9.32	10.28	6.65	7.46
	70	11.97	14.23	11.37	6.99	7.48	8.31	5.24	5.92
	75	9.56	11.75	9.25	5.52	5.93	6.63	4.08	4.63
	80	7.55	9.58	7.43	4.31	4.65	5.22	3.14	3.59
	85	6.02	7.71	5.89	3.33	3.60	4.07	2.40	2.75
	90	4.97	6.12	4.61	2.54	2.76	3.13	1.82	2.09
	95	4.28	4.80	3.57	1.93	2.10	2.39	1.37	1.58
	100	3.41	3.73	2.74	1.45	1.58	1.81	1.02	1.18
75–84									
	75	8.80	10.72	7.67	6.31	6.04	8.01	4.27	5.28
	80	6.61	8.53	5.96	4.84	4.62	6.24	3.21	4.01
	85	4.94	6.67	4.56	3.66	3.49	4.79	2.38	3.01
	90	3.80	5.14	3.44	2.73	2.59	3.62	1.75	2.22
	95	3.08	3.90	2.56	2.01	1.91	2.70	1.27	1.63
	100	2.33	2.91	1.88	1.47	1.39	1.99	0.92	1.18
<b>Women</b>									
65–74									
	65	18.68	22.63	21.63	16.05	10.63	17.87	8.97	9.85
	70	15.28	19.27	18.35	13.29	8.54	14.93	7.13	7.87
	75	12.27	16.20	15.36	10.86	6.77	12.30	5.59	6.21
	80	9.71	13.43	12.68	8.74	5.29	9.99	4.32	4.83
	85	7.72	10.97	10.32	6.93	4.08	7.99	3.30	3.70
	90	6.54	8.84	8.28	5.42	3.11	6.31	2.49	2.81
	95	6.07	7.02	6.55	4.19	2.34	4.91	1.87	2.11
	100	5.20	5.51	5.11	3.19	1.75	3.77	1.39	1.57
75–84									
	75	11.59	15.17	11.77	12.94	9.53	9.72	7.04	7.42
	80	8.65	12.29	9.32	10.34	7.42	7.58	5.35	5.67
	85	6.27	9.77	7.24	8.10	5.66	5.80	4.00	4.25
	90	4.61	7.62	5.52	6.23	4.25	4.35	2.94	3.13
	95	3.54	5.83	4.13	4.70	3.13	3.21	2.13	2.27
	100	2.99	4.38	3.04	3.48	2.27	2.33	1.52	1.63

Note: PT = pure type.

D) for two 10-year cohort groups, one aged 65 to 74 and the other aged 75 to 84, in 1982. The D coefficients for the seven equations were evaluated (for numerical convenience) as 2-year disability transition (changes in the K scores) rates from age 75 to 77. The lower diagonal elements of the matrices represent an average increase in a healthier state (i.e., state with a lower index than the diagonal) at age 77; upper diagonal elements represent loss of function by age 77.

The elements in the upper and lower triangular components of the transition matrix can each be summed within cohorts to get the net quantitative disability changes for these seven disability profile scores. For the 65- to 74-year-old male cohort, the average score of nondisabled men (Type 1) on the frail profile (Type 6) increased 0.01 in 2 years, whereas the average score of frail men (Type 6) on the nondisabled profile (Type 1) increased 0.18. For the 75- to 84-year-old nondisabled male cohort, the score for the frail (Type 6) profile increased 0.03, whereas the score of frail men on the nondisabled profile increased 0.16. For nondisabled women in the age 75 to 84 cohort, the average score on the frail profile increased 0.02, whereas the “frail women” score on the nondisabled profile

increased 0.28. The imbalance favoring transitions to less disabled states is, in part, due to the high mortality in the frail states among persons who do not rapidly improve their disability—especially at advanced ages (see Table 6 hazard coefficients).

The diagonal values indicate the stability of each person's state-specific score for ages 75 to 77 (e.g., the nondisabled profile score for men aged 65–74 in 1982 was 0.93 over 2 years). This can be compared to the much less stable 0.57 for frail men. For the 75- to 84-year-old male cohort, the score on the frail profile remained 0.58. Nondisabled men maintained an average score of 0.87 over 2 years (i.e., the older cohort had a smaller [6.5%] chance of retaining good functional capacity).

For women, the score on the nondisabled profile was 0.90 for the cohort aged 65 to 74 and 0.52 for frail women. For nondisabled women aged 75 to 84, the score was 0.85 over 2 years, whereas the score for frail women was 0.27. Persistence in intermediate disability states was roughly the same for men and women in the 65 to 74 cohort. Men in the age 75 to 84 cohort had a higher persistence. Said differently, female disability dynamics in the age 75 to 84 cohort were greater than those for either the male cohort or for the age 65 to 74 female cohort. In the transition score estimates (the average proportion of a specific disability state retained by an individual—not the proportion of individuals in a homogenous state), there is a greater chance of increasing the score on nondisabled states and a lower persistence of residence in institutions, net of mortality.

With the disability dynamics ( $D$ ) and mortality ( $\theta$  and  $Q$ ) parameter estimates we calculated the cohort-specific life tables for men and women in Table 8.

In Table 8 we present gender- and cohort-specific life tables, starting at age 75 (in Table 9 the youngest cohort is first observed at age 75 when the oldest cohorts are age 65) with the average  $g_{ik}(m)$  for each of the  $K$  profiles. For men the score on Type 1 was always higher for the youngest cohort. For women the Type 1 score at age 75 was higher for the older cohort and then, at all later ages, was higher for the younger cohort. This is due to the decline in institutional use by women in the younger cohort. For Type 6 and Type 7 the youngest cohort had lower  $g_{ik}(m)$  scores (less severe disability) than the older cohort—for men and for all women but those aged 75.

In Table 9 we present the LE for persons in each of the  $K$  pure types. This differs from Table 8 in that, instead of presenting the average  $g_{ik}(m)$  at a specific age, we present the integral of the survival curve in a given pure type  $K$  ( $g_{ik}(m) = 1$ ) for persons in that disability state for all ages after age  $X$ . Overall LE after age 75 was roughly three quarters of a year greater for both the younger male and female cohorts. Most of the differences between cohorts result from a longer LE for the younger cohort in Type 1 (e.g., 1.03 years for men and women).

A further comparison can be made with the life table calculations by, for the same age, starting the population in a given type (e.g., Type 1) and seeing how the LE and disability distribution evolves after that age given the interaction over time of age-specific disability dynamics diffusion and systematic mortality selection. This reflects a disability state decomposition of the stochastic interactions of cohort disability and mortality dynamics. This is shown in Table 10 for both

Table 10. Life Tables (Starting in PT1 at Age 75) for Two Male and Female Cohorts

Age	Lx	Ex	PT1	PT2	PT3	PT4	PT5	PT6	INST
<b>Men</b>									
65–74 in 1982									
75	100000	9.89	1.000	0.000	0.000	0.000	0.000	0.000	0.000
80	73080	7.58	0.849	0.033	0.015	0.016	0.023	0.025	0.038
85	44239	5.91	0.776	0.045	0.021	0.025	0.040	0.040	0.053
90	21222	4.77	0.736	0.045	0.023	0.031	0.055	0.048	0.062
95	8016	4.07	0.815	0.045	0.012	0.025	0.041	0.029	0.033
100	2536	3.30	0.884	0.038	0.005	0.016	0.022	0.019	0.015
75–84 in 1982									
75	100000	9.04	1.000	0.000	0.000	0.000	0.000	0.000	0.000
80	71095	6.67	0.816	0.034	0.017	0.022	0.026	0.034	0.051
85	39901	4.95	0.692	0.047	0.023	0.040	0.045	0.051	0.102
90	16305	3.80	0.670	0.048	0.025	0.032	0.050	0.054	0.121
95	4638	3.08	0.709	0.050	0.019	0.035	0.050	0.036	0.102
100	965	2.33	0.789	0.016	0.008	0.022	0.089	0.023	0.053
<b>Women</b>									
65–74 in 1982									
75	100000	12.73	1.000	0.000	0.000	0.000	0.000	0.000	0.000
80	82322	9.89	0.792	0.041	0.026	0.019	0.043	0.027	0.051
85	58998	7.79	0.672	0.051	0.035	0.028	0.069	0.047	0.098
90	34691	6.53	0.604	0.052	0.035	0.040	0.092	0.053	0.124
95	17136	5.91	0.694	0.052	0.031	0.032	0.088	0.028	0.076
100	8113	4.99	0.798	0.049	0.024	0.019	0.066	0.010	0.033
75–84 in 1982									
75	100000	11.83	1.000	0.000	0.000	0.000	0.000	0.000	0.000
80	82877	8.72	0.732	0.041	0.036	0.030	0.056	0.035	0.071
85	58330	6.29	0.549	0.055	0.043	0.046	0.084	0.059	0.164
90	31254	4.61	0.462	0.056	0.041	0.047	0.083	0.075	0.235
95	11735	3.54	0.462	0.051	0.036	0.043	0.080	0.078	0.249
100	2925	2.99	0.537	0.033	0.028	0.046	0.076	0.076	0.205

Note: Lx = number living to age x; Ex = expected life remaining at age x; PT = pure type; INST = institutional.

younger and older cohorts, where the life table populations are started at age 75 in Type 1 (and, in Table 11, in Type 7). The cohort comparisons show that, for men and women who were initially nondisabled, the younger cohort had higher LE and a greater likelihood of staying nondisabled.

For persons starting in Type 7, we found a different pattern in Table 11 for men, with the older cohort having a higher proportion found in institutions after age 85. The older male cohort had a longer LE at age 75, but the younger cohort had longer LE at older ages.

In Table 10, the fraction of the younger surviving male cohort that was still in Type 1 after 15 years (age 90) was 0.736 and increased to 0.884 at age 100—perhaps reflecting the age dependence of disability-specific mortality. For the older cohort, the corresponding numbers were both lower (i.e., 0.670 and 0.789). The same pattern occurred for women, but the nondisabled scores were even lower (0.604 and 0.462) after 15 years (at age 90), rising to 0.798 and 0.537 at age 100. For women, the older cohort had a longer LE at age 75 for those starting in Type 7 and a greater likelihood of remaining in Type 7. This likely reflects changes in the use of nursing homes at later ages due to factors such as the 1997 Balanced Budget Act. As for men, the younger cohort had longer LE past age 80.

To visualize the relative age-specific cohort changes in ALE, disabled LE, and total LE, we present in Figures 1, 2, and 3 changes in the proportion of life after age *X* expected to be lived in three specific groups of disability states.

In Figure 1 (scores are summed) we show how the proportion of LE at a specific age that is expected to be ALE (here defined as the sum of the Type 1, 2, and 3 disability scores) changes for pairs of male and female cohorts. For both genders, the proportion of LE that is ALE is higher for the younger cohort. Also noticeable is the improvement in the proportion of ALE at ages 85 and older. This implies that there is (a) more morbidity compression in the younger cohort with (b) a higher rate of morbidity compression occurring at advanced ages in the younger cohort. Thus, this model does not require assuming the proportionality of either age- or cohort-specific mortality rates. This is a very important result in evaluating the continuation of chronic disability declines over the long run.

In Figure 2 we present the age-specific proportion of LE expected to be lived in intermediate disability states (Types 4 and 5). For both younger male and female cohorts, this declines after age 85. There is a greater tendency to enter extreme disability states in the younger cohorts. This implies that the dynamics of disability are changing in a fundamental way, with a greater ability for rehabilitation of functional loss in persons

Table 11. Life Tables (Starting in PT7 at Age 75) for Two Male and Female Cohorts in 1982

Age	Lx	Ex	PT1	PT2	PT3	PT4	PT5	PT6	INST
<b>Men</b>									
65–74									
75	100000	6.19	0.000	0.000	0.000	0.000	0.000	0.000	1.000
80	47394	5.66	0.336	0.050	0.027	0.035	0.042	0.047	0.463
85	21760	4.68	0.461	0.055	0.031	0.036	0.058	0.068	0.291
90	8334	3.65	0.492	0.051	0.031	0.041	0.073	0.082	0.229
95	2265	2.80	0.481	0.054	0.032	0.045	0.082	0.083	0.222
100	382	2.13	0.479	0.057	0.030	0.044	0.084	0.082	0.226
75–84									
75	100000	6.70	0.000	0.000	0.000	0.000	0.000	0.000	1.000
80	52995	5.64	0.385	0.041	0.022	0.030	0.034	0.056	0.431
85	25049	4.36	0.468	0.052	0.027	0.049	0.053	0.078	0.273
90	8909	3.21	0.450	0.053	0.030	0.046	0.062	0.096	0.263
95	1964	2.35	0.413	0.061	0.027	0.056	0.073	0.094	0.275
100	219	1.63	0.385	0.031	0.020	0.052	0.093	0.106	0.313
<b>Women</b>									
65–74									
75	100000	8.43	0.000	0.000	0.000	0.000	0.000	0.000	1.000
80	59201	7.72	0.302	0.048	0.036	0.026	0.049	0.044	0.495
85	35726	6.26	0.422	0.052	0.039	0.034	0.078	0.068	0.307
90	18657	4.83	0.393	0.049	0.037	0.045	0.095	0.083	0.298
95	7402	3.73	0.367	0.048	0.037	0.048	0.102	0.089	0.309
100	2079	2.86	0.367	0.048	0.037	0.048	0.102	0.089	0.309
75–84									
75	100000	9.58	0.000	0.000	0.000	0.000	0.000	0.000	1.000
80	68749	7.89	0.366	0.046	0.045	0.039	0.072	0.056	0.377
85	44377	5.87	0.386	0.055	0.046	0.051	0.092	0.075	0.295
90	22689	4.17	0.306	0.053	0.044	0.053	0.092	0.102	0.350
95	7708	2.90	0.233	0.046	0.038	0.047	0.084	0.122	0.431
100	1384	2.02	0.178	0.028	0.032	0.056	0.087	0.145	0.476

Note: Lx = number living to age x; Ex = expected life remaining at age x; PT = pure type; INST = institutional.

with many chronic diseases. This dynamic is operating more vigorously at later ages, suggesting increased capacity for reducing prevalence within the age ranges currently well monitored. The older male cohort does not exhibit this behavior.

In Figure 3 we examine how much of LE at a given age is spent in highly impaired states (Types 6 and 7). This declines dramatically for the younger female cohort.

These statistics can be used to determine if morbidity compression is manifesting itself across cohorts—and across age within cohorts. We examine these statistics in Table 12.

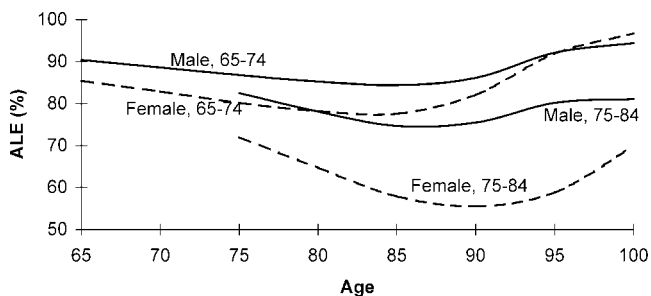


Figure 1. Percent active life expectancy (ALE) age distributions by cohort group.

Morbidity compression is occurring across cohorts for males and females at both ages 75 and 85. Morbidity compression is evidenced by a ratio less than 1.0, is occurring across cohorts for both male and females, and is occurring more rapidly at later ages. The faster decline in morbidity compression at late ages is also surprising, given that other authors argued that most disability declines were at lower levels of impairment (IADLs). The model can detect these nonlinear changes because it can monitor the interaction of disability-specific mortality and nonlinear (convexly constrained) disability dynamics.

## DISCUSSION

ALE measures based on the Sullivan (1971) method are useful in examining how health and function change in relation to LE in a population. In Manton and colleagues (2006b), we illustrated this for the period 1935 to 1999 with projections made to 2080. To examine disability trajectories for individuals in specific birth cohorts, however, requires different computations. In those calculations, birth cohorts are defined and disability transitions are estimated simultaneously with age-dependent mortality functions for individuals. With a cohort model, it is possible to determine which biological and socioeconomic mechanisms may be involved in forming trajectories by relating the disability score distribution in cohorts to other covariates for individuals (e.g., biomarkers,

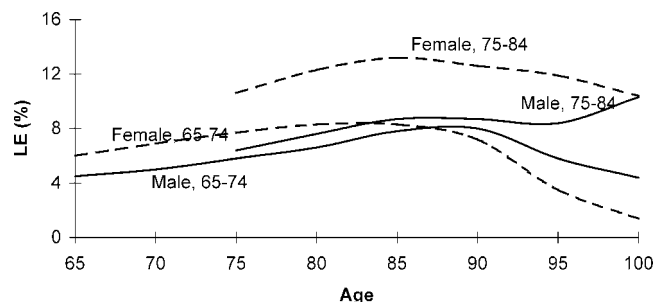


Figure 2. Percent life expectancy (LE) distributions for Pure Types 4 and 5 by cohort group.

education, and income; Manton & Gu, 2007). It is then possible to discuss which factors covary with disability over time. This also provides the necessary elements for examining the behavior of the more general dynamic equilibrium models of health and function (Manton, 1989).

We found several consistent findings in our cohort analyses. First, it is clear, for both men and women, that the younger cohort has greater longevity and can expect a greater proportion of the life span to be spent in a nondisabled state. Second, the longevity cohort difference is on the order of 0.7 to 1.0 years—even when the effects of cohort differences in disability dynamics are taken into account. Third, the shape of the within-cohort hazard function differs between cohorts. Thus, there appears to be a particular dynamic equilibrium across these two cohorts where ALE increases faster than morbidity dependence, with the rate of improvement declining (faster) at higher levels of disability and at later ages. Though the 24 years of observation is limited in making cohort comparisons over broad elderly age groups, there is sufficient information to suggest strong cohort effects being manifest in this model of dynamic equilibrium. Roughly 10 years of additional cohort follow-up would be necessary to evaluate arguments that increased obesity prevalence in World War II baby boom cohorts could reverse the disability declines of the past 24 years. Such cohort studies will be crucial to understanding what the aging of baby boom cohorts will do to the long-term fiscal stability of the Medicare and Medicaid systems due to cohort changes in health and function (Manton, Lamb, et al., 2007).

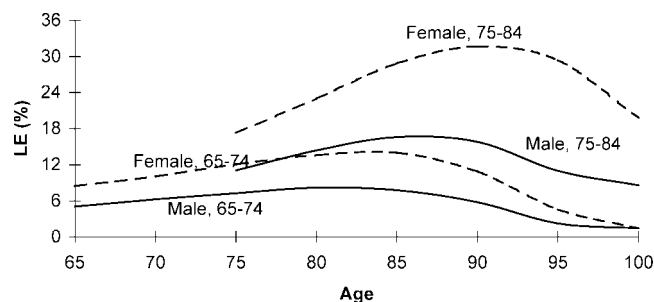


Figure 3. Percent life expectancy (LE) distributions for Pure Types 6 and 7 by cohort group.

Table 12. Percentage of Life Expectancy That Is Active Life Expectancy at Ages 75 and 85 by Cohort and Gender

Age	Men	Women
75		
Cohort aged 65–74	81.2	72.7
Cohort aged 75–84	76.6	63.4
85		
Cohort aged 65–74	78.7	71.0
Cohort aged 75–84	67.8	48.6

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