Recent Declines in Chronic Disability in the Elderly U.S. Population: Risk Factors and Future Dynamics

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active life expectancy, activities of daily living, human capital, rehabilitation, molecular medicine

Abstract

As U.S. life expectancy has increased, questions arise as to how the quality of health and functioning in the elderly population has changed. Data from the 1982–2004 National Long-Term Care Survey (NLTCS) suggested that chronic disability prevalence above age 65 declined at an increasing rate with a 2.2% per annum rate of decline from 1999 to 2004 (71). Inflation-adjusted per capita Medicare expenditure rates in nondisabled persons also declined, 0.9% per annum from 1982 to 2004, which suggests that declines in disability were driven by improving health—not by increases in per capita health expenditures. Declines in disability prevalence were found in other U.S. national health surveys. Analyses of U.S. Civil War veterans suggest recent disability declines were continuations of declines in both chronic disease and disability occurring over the past century due to improved nutrition, sanitation, and education. Concerns exist about whether disability declines will continue because of recent increases in obesity prevalence.

INTRODUCTION

Long-term care (LTC): services from either paid or family sources provided to support chronically disabled persons

We examine recent trends in chronic disability in the U.S. elderly population and the health dynamics that drive them. Scientists questioned, until recently, whether chronic disability in the elderly was so intrinsically linked to human senescence that its age-specific risk could not be readily modified. This idea implied that, as the U.S. population ages, the economic and social dependence of the elderly population would increase and demands for both acute health care and long-term care (LTC) would grow, fiscally stressing the Medicare, Medicaid, and Social Security systems.

In fact, a range of recent data indicates that, although the prevalence of disability was already declining in 1982, the prevalence of chronic disability in the U.S. elderly population was declining at an accelerating per annum rate; the period with the highest rate of decline being observed between 1999 and 2004. The effects of these declines are illustrated in **Figure 1**. The prevalence, adjusted for age, has declined from 26.5% to 19.0%, 1982 to 2004 (7.5 percentage points or a relative decline of 28.3%).

Notably, the decline (7.5%) in the chronic disability prevalence rate was sufficient to keep the number of disabled persons similar in 1982 (7.1 million) and 2004 (6.9 million), despite the growth of the U.S. elderly population by 34.6% (i.e., from 26.9 to 36.2 million persons). This stability suggests that the observed declines in chronic disability are important factors in determining the recent fiscal status of Medicare and Medicaid. Thus it is crucial to assess what current morbidity and aging health dynamics imply for future disability trends. A central factor in this assessment is to determine how changes in obesity prevalence may affect trends as the post–World War II (WWII) baby boom cohorts age.

To assess current and future disability trends we start by assessing various types of evidence (e.g., morbidity and cause-specific mortality trends in addition to disability changes) on how health improved, and chronic disability declined, from 1980 to 2005. We then evaluate how the current statuses of obesity and diabetes may interact with major circulatory diseases, and their risk factors, to determine future chronic disability trends. This evaluation is then extended to

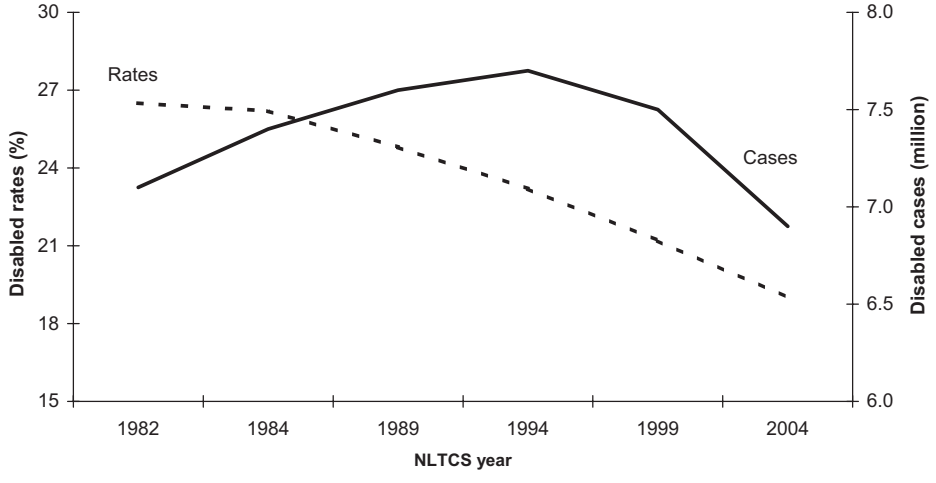

Figure 1

Disabled population and rates for NLTCS 1982–2004. Decline prevalence is illustrated (*left axis*, *dotted line*). The right axis (*solid line*) is the absolute number of elderly persons expected to be chronically disabled after adjusting rates for changes in the age distribution of the population.

consider other chronic diseases (e.g., cancer and arthritis) and younger age groups. Finally, we assess the future social, health care, and economic consequences of those disability trends.

BACKGROUND ON DISABILITY TRENDS AND HEALTH DYNAMICS

The existence of declines in chronic morbidity and disability rates in the U.S. elderly population has only recently been generally accepted by epidemiologists, geriatricians, and health policy makers. Specifically, after large declines in mortality at younger ages in the U.S. population in the first half of the twentieth century, the social and economic disruption of WWII and the Korean conflict led to increases in male cardiovascular disease (CVD) risks starting in 1954 in the U.S. and continued to 1968. U.S. females continued to manifest decreases in mortality over that period.

Increases in male CVD mortality from 1954 to 1968 led a number of authors to speculate on the adverse effects of industrialized society on public health, especially on the risks of aging-associated chronic degenerative diseases such as cancer and CVD and, by extension, related functional states such as chronic disability. This hypothesized relation was attributed to increased environmental hazards, increased social and economic stress, and the apparent failure of modern medicine to treat chronic degenerative diseases effectively (82). Omran (89) characterized health hazards associated with three stages of the epidemiological transition; the third phase was increases in chronic, degenerative, and manmade disease risks in modern industrial societies. McKinlay & McKinlay (83) focused on the apparent lack of efficacy of the clinical treatment of chronic diseases and the growth in the health effects of well-known behavioral risk factors such as cigarette smoking, unhealthy diet, and lack of physical exercise (5). Kramer (56) and Gruenberg (40) suggested that modern medicine had prolonged life, but most often in impaired health states. A prime example they gave was that surgical repairs of the myocardium allowed individuals with Down's syndrome to survive to reproductive ages. A pandemic of chronic degenerative and mental disorders was postulated (56).

Morbidity compression: the concept that the number of years lived without disability is increasing faster than total life expectancy

Many of these arguments were linked to a model of the biological determinism of changes in health and function with age where, in the latter part of the twentieth century, further large increases in life expectancy would have to occur at later ages and be due to progress against chronic degenerative diseases and, eventually, even against the aging process itself. Many academic demographers (e.g., 7) and federal actuaries (86) accepted this biological determinism and assumed, even in official federal projections of the Social Security entitlement population (e.g., in 1977), that the biological limits to human life expectancy had been, or would shortly be, reached in the United States.

Fortunately, in 1969 the increase in male CVD mortality rates stopped and declines in male CVD mortality began. It became clear, by 1980, that the conclusions about health trends based on mortality trend data from the 1950s and 1960s needed to be scrutinized on many levels, i.e., for specific, interrelated chronic diseases, such as CVD, cerebrovascular disease, diabetes, and cancer, and as the conclusions related such chronic diseases to human senescence, the loss of physical function, and the emergence of frailty at late ages (33). Theorists who recognized this need for new models included Fries (34, 35), with his model of morbidity compression, which still assumed age trajectories of mortality were biologically fixed even though morbidity and disability trajectories could be altered, and Manton (64, 65), who in a model of "dynamic equilibrium" argued against strict biological determinism when applied to any health outcome, i.e., that making health changes required modifying a stochastic dynamic equilibrium with positive age-related correlations of improvements in morbidity, disability, and mortality. Analysts of all types initially worked

with significant limitations on nationally representative longitudinal data (18).

The initial evidence on population health (e.g., 1950–1980) came primarily from national total and cause-specific mortality analyses at late ages (78). The evidence for progress against CVD mortality became overwhelming by 1980 so that Senate action was undertaken in 1982 and 1983 to raise SSA's normal retirement age from 65 to 67 starting in 2000, to be completed by 2022. The adjustment was designed to be conservative owing to the lack of reliable longitudinal data in 1980 empirically linking disabling processes and morbid events for a lengthy time period before an individual's death (18).

A national health survey [the National Long Term Care Survey (NLTCS)] focusing on health status and disability of elderly Medicare enrollees was started in 1982 by the Health Care Finance Administration [now the Centers for Medicare and Medicaid Studies (CMS)] and the Office of the Assistant Secretary for Policy Evaluation (ASPE) at the Department of Health and Human Services. Analyses of the 1982, 1984, and 1989 NLTCS suggested that chronic disability prevalence rates in the U.S. elderly population had declined (66). Associated with that data, other clinical and longitudinal epidemiological studies suggested that greater plasticity existed in the human aging process at later ages than had been expected (60), indicating that prior age rates of decline in specific physiological functions had been overestimated [e.g., by a factor of two in some cases (55)] because many early studies had not controlled for increases with age in the prevalence of chronic degenerative diseases in the study population. Consequently, occult chronic disease confounded estimates of the age rate of loss of organ function with the more diffuse effects of human senescence and chronic disease. For example, the effects of occult chronic diseases were so large for immunosenescence that special study protocols for case selection [e.g., the "Senieur" protocol (44)] were instituted.

Because of the 1982–1989 NLTCS trends, the National Academy of Sciences (NAS) convened a panel in 1993 to examine the consistency of population evidence for declines in chronic disability in the U.S. elderly population. The panel concluded that the NLTCS evidence was consistent with a decline but that conclusions should wait until the 1994 NLTCS (32) was done. Analyses of the 1994 NLTCS showed that the disability decline had not only continued but accelerated (67). Other analyses (97) suggested that disability declines would be fiscally important for Medicare, Medicaid, and Social Security if they continued at the observed rate (1.5% per annum). Declines continued and were found in both 1999 (68) and 2004 (71) with evidence of an acceleration of the decline rate of disability prevalence from 1982 to 2004. The rate of decline averaged over the 22-year period was 1.5%. It accelerated to 1.8% per annum from 1994 to 1999 and to 2.2% per annum 1999 to 2004. In an independent analysis using longitudinal sample weights, Stallard (99) estimated for 1984 to 1999 a faster rate of disability decline of 1.6% per annum using disability defined based on more stringent Health Insurance Portability and Accountability Act (HIPAA) criterion. Other studies examined the quality of the U.S. health survey data on disability declines (31) and the consistency of evidence on disability declines across several national health surveys (29). The evidence across surveys supported the NLTCS observation of disability declines at later ages. Stallard's study (99) of declines in the community disabled population defined using HIPAA standards indicated that improvement was occurring even for severely disabled persons.

Analyses also suggest that large declines were evident for cognitive, as well as physical, impairments. Severe cognitive impairment in the NLTCS dropped 42% (nonage standardized) from 1982 to 1999 (70, 74). Similar declines (41%) were observed 1993 to 1998 in the HRS (Health Retirement Survey) (28). The cognitive declines in the NLTCS were substantiated (70, 74) using Medicare service

Longitudinal: persons are sampled at specific ages and followed and reinterviewed over time

National Long Term Care Survey (NLTCS): a national survey of Medicare-enrolled persons aged 65+ with detailed questions on disability and LTC use

CMS: Centers for Medicare and Medicaid Studies

HIPAA: Health Insurance Portability and Accountability Act

use data linked to the NLTCS, where ICD (International Classification of Diseases)-9 diagnoses were provided (i.e., post 1990). For the 1994 and 1999 NLTCS, we can determine if one, or more, of four ICD-9 dementia diagnoses (ICD-9 codes 290, 310, 797, and 331.0) were reported in Medicare service use records. These four categories differentiate between dementias due to Alzheimer's disease vs. dementias where circulatory disease (e.g., stroke) is the underlying mechanism. Alzheimer's disease had temporally stable rates. Severe cognitive impairment due to stroke and other types of circulatory diseases showed significant declines (74).

It is important to study disability declines for population groups such as men vs. women and for blacks vs whites. Health disparities in terms of chronic disabilities still remain, although recent disability declines for blacks were faster than for whites. Education is one factor underlying changes in those disparities; declines in low levels of education for blacks are larger than for whites with the proportion of the black population with 9–12 years of education increasing much faster (68). Geographic differences were also found in the 1999 and 2004 NLTCS with disability declines more rapid in Southern regions possibly because of the effects of weather on outside mobility and other activities. Males and females also showed differences with a higher prevalence of chronic disability and nursing home use by females, but disabled females had greater longevity than did disabled males.

Such observations are consistent with data on positive CVD risk factor trends in the NHANES, even within obese and diabetic groups (38, 45) and with continuing improvement in the medical management of circulatory disease and diabetes mellitus type II (e.g., 27). Cutler et al. (15) in analyses of the 1982–1999 NLTCS found that improved medical management of circulatory disease was responsible for much of the decline in disability prevalence. Furthermore, as chronic disability prevalence declined, the per-annum inflation-adjusted Medicare costs for nondisabled persons declined 0.9% per annum from 1982 to 1999 (75). Thus health had also improved in the nondisabled U.S. elderly population from 1982 to 2004.

LONG-TERM CHRONIC DISABILITY AND DISEASE TRENDS

Fogel & Costa (24) analyzed long-term declines in chronic disease and disability by examining changes in the prevalence of chronic diseases and disability in the Gould sample of Union Army Civil War recruits (13) and in Union Army Civil War veterans who applied for federal pensions in 1900 and 1910 (12, 13). The prevalence of chronic morbidity and disability in these two groups was compared, in one case, to more recent data on modern military recruits (13) and, in the second case, to general white male populations as assessed in the National Health Interview Surveys and in the National Health and Nutrition Examination Surveys in the 1980s and 1990s (12).

Fogel & Costa established from those data (especially the Civil War veterans) that there was a slow, long-term decline in chronic morbidity and disability averaging 6% per decade. These declines were related to improvements in food and water quality, sanitation, hygiene, and education, elements they referred to as "techno-physiological evolution" (24). These factors reduced infectious disease risks and chronic disease related to infectious disease insults and allowed body mass index (BMI) and physical fitness to increase slowly over time with positive economic and health benefits (23). It is in the context of declines in morbidity and disability over the past 100 years that the recent acceleration of chronic disability declines in the past 22 years of the NLTCS occurred (71).

CHANGES IN DISABILITY PREVALENCE

Changes in chronic disability prevalence can be assessed a number of ways. The first

Table 1 Estimates of the chronically disabled percent of the U.S. population aged 65+ [from Manton et al. (71)]

	1982	1984	1989	1994	1999	2004/5
Nondisabled	73.5	73.8	75.2	76.8	78.8	81.0
IADL only	5.7	6.0	4.5	4.4	3.3	2.4
1–2 ADLs	6.8	6.9	6.6	6.1	6.3	5.6
3–4 ADLs	2.9	3.0	3.7	3.4	3.7	3.8
5–6 ADLs	3.5	3.3	3.1	2.9	3.0	3.2
Institution[1]	7.5	7.0	6.9	6.3	4.9	4.0
Per annum % declines		0.6	1.1	1.3	1.8	2.2

[1]Institution refers primarily to nursing home beds, i.e., beds in residential facilities where nursing or medical care is available on a 24-hour basis.

method is to define a disability threshold for specific physical activities in the U.S. elderly population. One way to determine a disability threshold is to use measures of the ability to perform specific types of activities of daily living (ADLs) (50, 51) or instrumental activities of daily living (IADLs) (61). Such measures, in addition to being used often in geriatrics and gerontology, are also commonly used for federal and private actuarial and tax purposes to define a threshold for the need for LTC (99). In addition to ADLs and IADLs, other measures, such as the ability to perform simple physical tasks, are sometimes used (87). Also important in the definition of disability is the setting of a time criterion, e.g., how long the ADL or IADL impairment has lasted, or is expected to last (90 or more days).

Using these measures we examined changes in the distribution of chronic disability categories for elderly persons in the NLTCS in **Table 1**.

The per annum rate of disability decline increased from the background rate of 0.6% found 1900 to 1980 by Fogel & Costa to the 1.5% per annum rate of change found 1982 to 2004. Whereas declines from 1982 to 1994 were concentrated among IADLs, which can often be compensated for using assistive devices and changes in the built environment, recent declines were observed in both lightly and heavily impaired (including institutional) groups (71, 99). Changes in physical frailty with high levels of impairment will likely require physical rehabilitation and medical care innovation (e.g., new technologies for joint replacement; use of stem cells for growth of replacement ligaments and tendons) (33, 71).

Important in the overall decline of chronic disability in **Table 1** are reductions in the U.S. elderly institutional population, especially the large post-1994 declines. This institutional population reduction is due to three factors. First is the emergence of assisted living facilities (ALFs) as a residential LTC alternative to nursing homes. The second is the passage of the Balanced Budget Act in 1997 (and as refined in 1999), which mandated a prospective payment system for skilled nursing facilities, reimbursed by Medicare, which explicitly provided for rehabilitative care. Third is the growing popularity of home- and community-based waivers whose use was facilitated by the 1999 Olmstead court decision. The effects of the emergence of ALFs is illustrated in **Table 2**.

The ALF population in 1994 was of negligible size. By 1999 there were 810,000 persons in ALFs, and by 2004, 1.24 million. ALFs often provide a continuum of care. Thus, both chronically disabled community residents, and persons in the equivalent of nursing home beds, are found in ALFs along with nondisabled, socially independent

Activities of daily living (ADLs): Activities (e.g., inside mobility; bathing; dressing) necessary to be performed to live independently

IADLs: instrumental activities of daily living

ALF: assisted living facility

Table 2 Percentage of ALF and non-ALF use by disability groups. From 1982 to 2004 NLTCS tabulations

		1994	1999	2004
ALF	Nondisabled		41.30	39.06
	Comm disabled		43.53	32.63
	Institution		15.17	28.31
	Total		2.30	3.42
Non-ALF	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

persons. Although the proportion of non-ALF persons in institutions declined (from 6.0 to 3.3%) from 1994 to 2004, the proportion of ALF residents in nursing beds increased from 15.2% (123, 120 cases) to 28.3% (350,920 cases) between 1999 and 2004. Much of that shift was from disabled community residence to institutional beds, i.e., the intensity of care in ALFs increased from 1999 to 2004.

Several problems exist in disability prevalence models. First, they only crudely reflect the burden of disability on a population in terms of the numbers (not intensity) of persons affected. They are dependent on the specific disability measures made in a survey and the disability thresholds assumed (29). Fortunately the per annum rate of decline in disability prevalence is robust to the wording and selection of ADLs and IADLs in U.S. health surveys (29, 99). Disability declines were confirmed in the National Health Intervention Survey (NHIS) and the 1984 and 1994 Supplement on Aging (SOA) (e.g., 31, 63, 96), the Current Medicare Beneficiary Survey (107), the Health and Retirement Survey (29), and the 1984, 1990, 1991, and 1993 Survey of Income and Program Participation (30). Declines in disability prevalence were robust to different features of the surveys (e.g., survey type; list vs. household sample) and coverage [i.e., whether only of community residents or of the entire elderly population (31)].

METHODOLOGICAL SOLUTIONS TO DESCRIBING THE POPULATION BURDEN OF DISABILITY

Frailty Indices

Simple prevalence measures do not well describe features of individual disability dynamics and risk factors. One methodological fix is to use a frailty index, which is a sum of a number (~30) of measures of minor disability traits. The intent is, instead of identifying persons as disabled, to identify the level of frailty manifest by individuals. The frailty concept is intended to approximate better the measures of the age-related physical acquisition of frailty as clinically defined (e.g., 38, 84). Results from analyses of the 1982–1999 NLTCS showed that frailty measures well described the age rate of acquisition of frailty and the dependence of mortality on frailty levels (57, 58).

Multivariate Disability Indices

Alternately, one may apply multivariate procedures to a wide battery of disability measures (e.g., ADLs, IADLs, physical performance items, sensory deficits). Because disability items in surveys are generally discretely coded, an assumption of multivariate normality is generaly not appropriate. Instead one may use a multivariate procedure known

Physical performance: measures of the degree of difficulty in performing common physical tasks such as climbing stairs

Active life expectancy (ALE): years expected to be lived after age X without chronic disability

LE: life expectancy

as grade of membership (GoM) analysis (81). GoM identifies K disability profiles with scores on each profile indicating the degree to which the individual manifests the traits characterizing a profile. Seven disability types were identified in analyses of the 1982–2004 NLTCS (72). Individual scores on those profiles well predicted Medicare costs and mortality (69, 75). Other indices have been constructed (e.g., QALYS, DALYs) using unidimensional scaling of the level of health impairment based on either expert panel or interview evaluations assuming the subjective utility of perfect health is scored 1.0 and death is 0.0 (95).

ALE

The construction of utility weights for specific health states, whether based on a subjectively or objectively determined score, does not directly reflect the effects of duration in a specific functional state. Disability dynamics can be described using ALE measures (92). The simplest ALE measure combines life table parameters and sample survey–derived disability prevalence measures (102). We calculated ALE using disability measures from Fogel & Costa's Civil War Veterans studies for 1935–1980 and disability measures from the 1982–1999 NLTCS. In **Figure 2** ALE and LE are presented for ages 65–100 for 1935 and 1999 and projections for 2080.

At age 65 LE increased from 11.9 years in 1935 to 17.7 years in 1999 (+5.8 years), whereas ALE increased from 8.8 to 13.9 years (+5.1 years). This is an increase in the ALE to LE ratio from 73.9% to 78.5%, suggesting morbidity compression (34). Disability initially declined more slowly than mortality, i.e., 0.6% per annum 1935 to 1980. Consequently more morbidity compression occurred from 1980 to 1999 (i.e., from 72.8% to 78.5%). The declines in the ratio at ages 85+ were relatively larger and faster, i.e., the ALE/LE ratio of 23.3% in 1935 increased to 46.9% in 1999. The rate of morbidity compression for persons 85+ is greater from 1982 to 1999. Thus, the rate of morbidity compression is changing not only over time but also with age

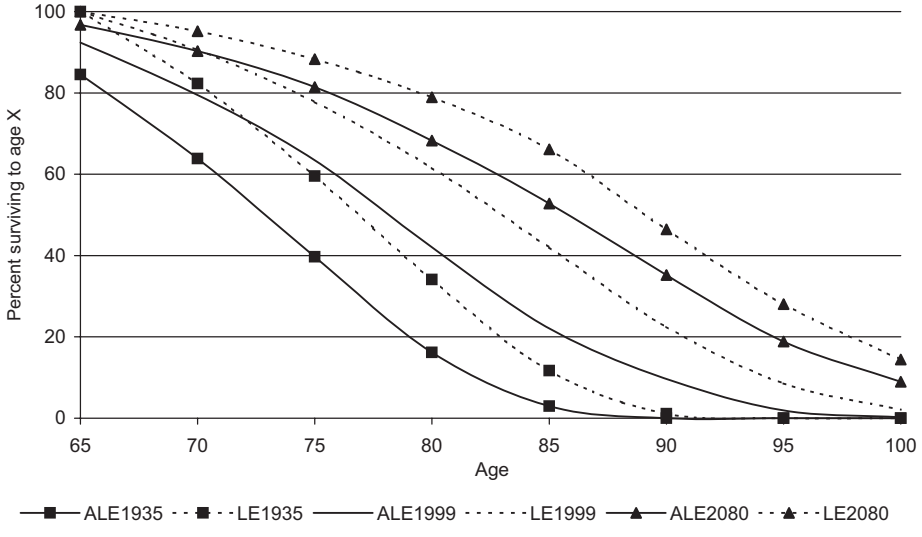

Figure 2

Survival curves with ([1]LE), and without ([2]ALE), disability for 1935, 1999, and 2080 (Source: 72). [1]LE 1935 is the curve describing the probability, starting at age 65, of surviving to a given age, starting in a given year (i.e., including disabled persons). [2]ALE 1935 is the curve describing the probability, starting at age 65, of surviving to a given age, in a given year (e.g., 1935) without chronic disability.

(72). Continuation of the 1982 to 1999 trends to 2080 would have important consequences for Medicare and Medicaid (see **Figure 2**).

Dynamic Equilibrium

The positive correlation of LE and ALE in the United States is indicative of a dynamic equilibrium (64, 65) for which evidence exists in the United States and in other countries. The dynamic equilibrium model is a generalization of the compression of morbidity hypothesis. It suggests that the correlation of the progression of morbidity and mortality processes requires, if adult mortality declines, that there be corresponding (but not one to one) reductions of the progression rate of morbidity and disablement processes.

In New Zealand morbidity compression, expansion of morbidity and dynamic equilibrium models were tested in large population surveys done in 1981 and 1996 (36). Institutional use declined, but moderate community disability increased, suggesting that the prevalence of LTC service needs increased but that the per capita intensity of service use decreased. Thus, the dynamic equilibrium model was argued to best fit this data, and the investigators suggested that such a model could imply slower growth of LTC needs even if the prevalence of disability increased.

To study age- and disability-specific dynamics and age- and disability-specific mortality, we need to track risk factor and disability changes longitudinally in individuals. This can be done using a cohort-based stochastic process model to describe population changes in disability intensity as attenuated by systematic mortality selection (73, 79, 80). The effects of disability dynamics on mortality are presented in **Table 3** for two birth cohorts, one aged 65–74 and one aged 75–84, in 1982 (73).

In **Table 3** θ is a parameter in the Gompertz model that represents the age rate of increase (percent per year of age) for mortality rates for males and females in two birth cohorts. The Gompertz function is a mathematical expression, used frequently by actuaries and demographers, that describes how mortality rates increase with age (i.e., as θ% per year of age). In the disability dynamic model, θ is estimated conditionally on age changes in disability. A comparison of θ for the model with and without disability dynamics indicates that 30% of the effect of age on mortality (6.55/9.33) for females can be attributed to disability dynamics. For males the disability dynamic effect is smaller (20%). Mortality increases more slowly in the younger birth cohort (aged 65–74 in 1982). In addition to having smaller θ values, in estimates of disability transition matrices the younger cohort was more likely to stay at lower disability intensities, i.e., the younger cohort had both disability-specific mortality advantages and disability-dynamic advantages (73). These disability and mortality estimates of dynamics are based on Markov assumptions allowing disability dynamics and mortality

Table 3 Cohort differences of θ between a disability dynamic covariate model and a simple Gompertz mortality model. From Manton et al. (73)

		Female			Male		
		65–74	75–84	θ difference between 2 cohorts	65–74	75–84	θ difference between 2 cohorts
Disability dynamic model	θ (%)	6.55	7.55	1.00	6.24	7.01	0.77
Gompertz only	θ (%)	9.33	10.37	1.04	7.73	8.59	0.86
	(*SE*)	(0.08)	(0.10)	(0.13)	(0.08)	(0.16)	(0.18)
θ difference between two models (%)		2.78	−2.82		−1.49	−1.58	
t value		−24.6	−20.0		−13.3	−7.1	

to interact over time to describe better the changes in individual disability trajectories net of mortality selection. Estimates of dynamics, however, are only as good as the assumption of Markovity, i.e., that dynamics and mortality changes are independent given the disability scores describing the person's health state. To generalize the disability dynamic model one can modify health transitions to reflect health changes between NLTCS using daily Medicare service use records linked to the NLTCS. This is done by modeling the acquisition of medical conditions in a semi-Markov generalization of the model where trajectories incorporate interactions of disability-specific dynamic coefficients with medical conditions acquired at specific dates.

RISK FACTOR TRENDS AND FUTURE DISABILITY

Obesity Trends

Although the evidence for disability declines in the U.S. elderly population is persuasive, recently a controversy has arisen over whether improvement in the health and functioning of the U.S. elderly population can continue because of the growing prevalence of obesity in the U.S. population. Some researchers have suggested that future increases in obesity and disability may signal future declines in U.S. life expectancy (88) and greatly increased future Medicare costs (100).

Bleich et al. (6) examined the factors driving obesity changes in developed countries using macrodata from a variety of sources. They found that 82% of the increase in adult obesity was due to increased caloric consumption with reductions in energy expenditure playing a small role. They suggest that increases in caloric consumption are driven by technological innovations reducing the relative price of food. One solution to the problem would be to increase the relative price of food. This might be done by better regulating the food supply and enhancing its nutritional quality, e.g., by trans fat elimination or folate supplementation. Improving health may also require improved health care (e.g., better control of elevated cholesterol, blood pressure, and blood glucose levels) to reduce the morbid consequences of diet-related health problems, such as diabetes. These increased health care costs may partly compensate for lower food prices. Additionally, it is not clear that the current relative price advantage of food consumption over other goods will be maintained, e.g., increased use of corn and other grains for biofuels is currently driving up the cost of foods such as beef and milk and may have dire consequences in less-developed countries.

Generally, increases in the prevalence of obesity in the U.S. population are assessed in terms of BMI. Because obesity (BMI > 30.0) has been linked to diabetes mellitus type II and, consequently, to increased CVD and stroke risks and certain cancers (esophageal adenocarcinima, breast, pancreas), speculation suggests that disability prevalence in the elderly may begin to increase around 2012 because of increases in CVD morbidity and related physical and cognitive disability (59).

This speculation is consistent with recent increases in disability benefits paid by Social Security, i.e., from 3.8 million persons qualifying for benefits in 1983 to 7.6 million in 2002 (8). However, it is difficult to assess the direction of the linkage of obesity to work-related disability and loss of productivity. Analyses of the relation of obesity and disability to U.S. labor-force participation and productivity have used such surveys as the National Longitudinal Study of Youth and the Panel Study of Income Dynamics (9, 10, 104). Unfortunately, the results of these kinds of studies are not clear because of difficulties in sorting out causation, e.g., does obesity cause the work- and labor-force impairment or does inactivity due to disability result in increases in obesity (8)? Additionally, obesity itself has sometimes been identified as a type of disability for Social Security Program purposes (2, 8). Furthermore, the physiological heterogeneity of obesity (e.g., hypercellular obesity in childhood, hypertrophic obesity in

adulthood) is generally not well recognized in national health survey instruments (3).

CVD and Diabetes

Some epidemiological and demographic evidence suggests that dire predictions about the effects of obesity on chronic disability and Medicare expenses may not occur. One prediction is that the strength of the relation of obesity to mortality, above age 65, attenuates (91). Flegal et al. (21, 22) conducted analyses involving both corrections of methodological errors in earlier studies (e.g., (1)) and use of more recent survey (NHANES) data where the effects of medical innovations to reduce morbidity in certain risk factor states may strongly temper obesity-driven increases in chronic morbidity and mortality (14). Flegal's results suggested that the health effects of obesity, at least on mortality, had been overestimated in many studies.

For example, in Framingham (26) the age- and sex-adjusted incidence of CVD declined 20% faster from 1950 to 1995 in diabetics than in nondiabetics, although diabetics still have significantly higher CVD incidence. Fox et al. (26), analyzing 50-year results in the Framingham Heart Study, confirmed the decline (from 3.0 to 2.5) in the hazard of diabetes as a CVD risk factor, even though there was also a significant decline in CVD among nondiabetics. An increase in the attributable risk of diabetes for CVD (from 5.4% to 8.7%) was not significant after multivariate controls (for CVD risk factors, age, and sex).

Jagger et al. (46) suggested that improved management of stroke, cognitive impairment, arthritis, and visual impairment could all increase future disability-free LE on the basis of a 10-year follow-up study of 13,000 persons aged 65+ in the United Kingdom. The United States is advantaged in that CVD risk factor improvements and heart and stroke mortality declines continued despite increases in BMI (37, 38). Daviglus et al. (16) showed that improved CVD risk factor profiles in middle age were related to improved quality of life in elderly persons. Other recent analyses found that the obesity-related metabolic syndrome was not strongly predictive of CVD risks and renal dysfunction, suggesting that current BMI may not be the best predictor of health effects but instead perhaps whether BMI was raised earlier in life and stayed raised for a long (several decades) period of time (4).

In several recent large studies, the relation of obesity to disability was found to be complex. Snih et al. (98) followed 12,275 adults aged 65+ who were not initially disabled when interviewed between 1982 and 1993. During the follow-up period, both underweight (BMI < 18.5) and obese (BMI > 30.0) persons were more likely to be disabled. Mortality was lowest for persons with a BMI of 25.0 to 34.9, and disability-free LE was highest for BMIs of 25.0 to 29.9—consistent with Fogel's (23) long-term evidence showing that the current health optimal BMI was in the range of 26 to 27.

Arthritis

The complexity of the obesity-disability relation may be due, in part, to selected health-protective effects of obesity, especially at late age. Disability-protective effects of moderately elevated BMI might include such factors as reduced fracture risk. A NIH consensus conference suggested that a negative relation between osteoporosis and osteoarthritis may exist with an elevated risk of osteoarthritis in persons with high bone density. Thus, hormone replacement therapy and treatments for osteoporosis may have a partly counterbalancing impact on osteoarthritis (19). Other studies suggest a paradoxical effect of BMI on survival in rheumatoid arthritis mediated by comorbidity and levels of systemic inflammation (17).

Leveille et al. (62) found, in the NHANES, that although obesity increased over cohorts, that increase was not related to cohort increases in arthritis prevalence in either the young or the old "baby boom" cohorts. One explanation is that the older cohorts could

have a greater arthritis burden from being involved in occupations with greater physical demands (e.g., agricultural workers in periods when there was less farm mechanization; manufacturing jobs before occupational safety programs). Thus, the increase in obesity might be due, in part, to the declining physical (potentially debilitating) demands of certain jobs. A second explanation offered were changes in diagnostic criterion for osteoarthritis. The future burden of arthritis in baby boomers may also be mediated by such medical advances as improved medications (e.g., NSAIDS, including over the counter medications and new disease-modifying prescription drugs for rheumatoid arthritis) and innovations in knee and hip replacement.

Additionally, total and cause-specific mortality trends continue to show strong declines: Total LE increased 0.4 years from 2003 to 2004, cancer mortality now shows a decline, and CVD continues to decline. From 1990 to 2004 cancer mortality rates have declined more than 10%. These positive cause-specific and total mortality trends can be linked to a wide variety of survey (e.g., the NHANES) evidence suggesting that, except for obesity, major risk factor trends have been positive (e.g., reductions in hypertension, hypercholesteremia, and smoking) and that among both diabetics (27, 38) and the obese (37) there has been improved management of risk factors and chronic disease complications. For example, in one study the use of statins (109) significantly lowered the risk of CVD in diabetics. In another large study, improved clinical control of hypertension/hyperlipidemia and glycolated hemoglobin increased Qalys (95).

Childhood Obesity

Data on obesity increases in children in the United States are difficult to interpret because frank CVD morbidity usually occurs much later. Furthermore, the risk factor state in adults is often independent of BMI in childhood when adult BMI is controlled, suggesting imperfect tracking of BMI levels from childhood to adulthood. Recent analyses of longitudinal data, however from both the Bogulusa Heart Study and the Cardiovascular Risk in Young Finns Study suggest that risk factors in childhood, including BMI, are independently (of contemporaneous adult risk factor values) associated with intima-media thickness in adulthood. Chiolero et al. (11), in contrast, did not find strong evidence for longitudinal increases in blood pressure associated with increasing obesity in children. This caused the authors to speculate whether "favorable secular trends in other determinants of blood pressure (e.g., dietary factors, birth weight, etc.) may have attenuated the apparently limited impact of the epidemic of overweight on blood pressure in children." Ford et al. (25), after examining the effects of obesity in children aged 2–17, also concluded, "The obesity epidemic among U.S. youths appears not to have had serious effects on their metabolic health at least in terms of trends in various risk factors for cardiovascular disease" (p. 1543).

Overall the evidence on obesity risk factor relations over time in children seems consistent with the evidence on risk factor trends for U.S. adults, although data on biomarkers of specific vascular changes in childhood raise concern (49). Although the adverse health consequences of childhood obesity may now be muted by health care interventions, primary and secondary preventive steps are preferred and likely more cost effective. Several measures to reduce childhood obesity have been suggested in a recent NAS (2004) study, including increasing physical activity and improving the quality of diet in children.

Cancer

In addition to questions about the effects of obesity and diabetes on heart disease and stroke, investigators have observed relations of obesity, diabetes, and various types of cancer. Empirical relations of obesity to tumors have been noted for anatomic sites such as breast, colorectal, endometrium,

liver, esophageal, pancreatic, kidney, and gallbladder.

These relations are complex: Up to one third of such cancers occur in diabetics. The National Cancer Institute indicates obesity may be protective for premenopausal breast cancer but may be a risk factor for postmenopausal breast cancer (105, 106). This finding is consistent with physiological differences in the two types of tumors; premenopausal breast tumors are highly aggressive and related to family pedigree but are not heavily estrogen dependent, which contrasts with postmenopausal disease (77).

The relation of obesity and colorectal cancer may be mediated by diabetes through the elevation of serum glucose, insulin resistance, and select growth factors [e.g., insulin-like growth factors (47, 54)] to colorectal (and other) cancers in both European (British) and Asian (Korean and Japanese) populations. The relation exists even in Korean populations where the prevalence of obesity was relatively low.

The linkage may be genetic in that certain tumor suppressor proteins (LKB1) are linked to enzymes that are targets for several drugs designed to treat type 2 diabetes (42). Thus, the observed relation may be due to genetic factors determining hormonal states, increasing susceptibility to both obesity and certain types of tumor growth rather than to obesity alone. The importance of determining the correct causal path is that recent increases in obesity prevalence are likely due to environmental, and not genetic, factors so that the recent obesity epidemic may produce a diluted relation of obesity to cancer risks.

The increase in the diagnosis of diabetes and recent major improvements in its treatment (49) may also partly modulate certain cancer risks through modification of insulin and insulin growth factor production using diabetes medications. For example the Quest study indicated diabetes control [measured by hemoglobin a1C (49)] improved by 44% from 2001 to 2006. Such a relation would also suggest that improvements in diabetes control by medication could reduce cancer mortality risks among diabetics [especially males (39)]. Indeed, certain diabetes treatment agents may even have value in treatments for multiple myeloma and pancreatic cancer (90).

Heterogeneity in Obesity

A factor that has not been well studied is the complex dynamics of the population distribution of BMI. Specifically the BMI categories used to define normal weight, over weight, and obesity are based on global World Health Organization standards. It is arguable whether those standards define optimal health in all ethnic groups in all countries regardless of level of development and over time. For example, owing to genetic differences, diabetes prevalence may rise faster in certain Asian countries (e.g., India) as development progresses.

Studies by Su (101) and Henderson (43) of the Union Army cohort and of men in NHANES-I showed that the mean BMI had increased 3 to 4 points in NHANES-I along with an increase in the variance of the BMI distribution; the BMI value associated with the lowest mortality risk in the NHANES-I was in the range of 26 to 28. One explanation for this increase is suggested by Costa's (13) analysis of the Gould sample of Civil War recruits where, although BMI had increased significantly, much of the gain was concentrated in lean body mass with demonstrable increases in fitness and strength. The plausibility of this argument is supported by the imperfect correlation of BMI to body fat found in athletes and persons with high levels of physical fitness.

Thus, one should perhaps be examining better physiologically defined and measured phenomena such as sarcopenic obesity driven by conversion of lean to fat body mass with age. This evidence seems consistent with the role of sarcopenia in the acquisition of frailty with age in Fried et al. (33). Sarcopenic obesity requires study of the role of specific inflammatory cytokines (e.g., C-reactive protein, IL-6, TNF-α) that may influence the

Human capital: the total number of person years expected to be lived capable of productive social and economic activities

relation of muscle loss to fat gain (e.g., 93, 94). Sarcopenic obesity interventions may involve the use of inflammatory process mediators, statins on hypercholesteremia, and multiple agents in hypertension. Literature in children suggests that the prenatal period, the period of adiposity rebound, and adolescence may reflect growth stages where the development of persistent obesity and comorbidities are quite different (52).

Improvements have occurred not only in the clinical management and treatment of risk factors and early disease in individuals but also in public health population interventions. For example, continuing declines in smoking may allow modest increases in BMI, offsetting part of their benefit. Yang et al. (108) showed that the implementation of folic acid supplementation of grain products in 1998 in the United States and Canada may have accelerated declines in stroke mortality. In the United States, the decline in stroke mortality between 1990 and 1997 was 0.3% per annum. The rate of decrease grew to 2.9% per annum between 1998 and 2002. In Canada the decline rate increased from 1% (1990 to 1997) to 5.4% (1998 to 2002). These declines were correlated with increases in average blood folate concentrations and reductions in homocysteine levels. In England and Wales, which do not supplement with folate, stroke mortality did not significantly change between 1990 and 2002. Because of the role of stroke in dementia there is reason to expect that these mortality declines will be reflected in future declines in dementia produced by circulatory diseases (46, 74). Thus, recent population and individual risk factor (14) and disease interventions have strongly modulated the obesity-risk-factor-disease-disability relations on which arguments about future disability trends have been based (100).

Currently the primary public health problem associated with obesity is that the distribution of such risk factor and disease management services is not universal in the U.S. population; education, income, and health insurance coverage determine how well obesity-related risk factors are controlled in specific populations (48, 53). This is an area that is of crucial public health and research significance as suggested by Ferrucci & Alley (20) and emphasized by the economic arguments in the next section. Consequently improvement in studying disability risk-factor mechanisms such as obesity, and the more complete dissemination of such health services, will be crucial to controlling the future population health consequences of obesity. However, broader dissemination of services to modify those mechanisms, in addition to treatment and secondary prevention innovations, may cause U.S. disability declines to continue for a long time into the future.

SOCIAL AND ECONOMIC IMPLICATIONS OF DISABILITY TRENDS

Just as changes in chronic disease risk factors have consequences for the rate of progression of disablement processes, changes in disability and functioning at late age have important consequences for social and economic processes (97). We have mentioned the potential effects on federal LTC service and health care programs, but the risk factors also have consequences more generally in that they determine to what age persons can expect to continue to contribute to the social and economic productivity of the United States.

Murphy & Topel (85) found that increases in life expectancy from 1970 to 2000, which tended to occur at later ages owing to reductions in CVD mortality, increased U.S. social wealth (both increases in GDP and human capital) by $95 trillion dollars. They also examined (85) the theoretical benefits of health quality improvement, which, despite not having good longitudinal data on health and functioning, suggested that a year gain in quality of health had a greater (perhaps double) economic effect than a year's gain in LE. Consequences of decreases in the prevalence of chronic disease and disability, and increases

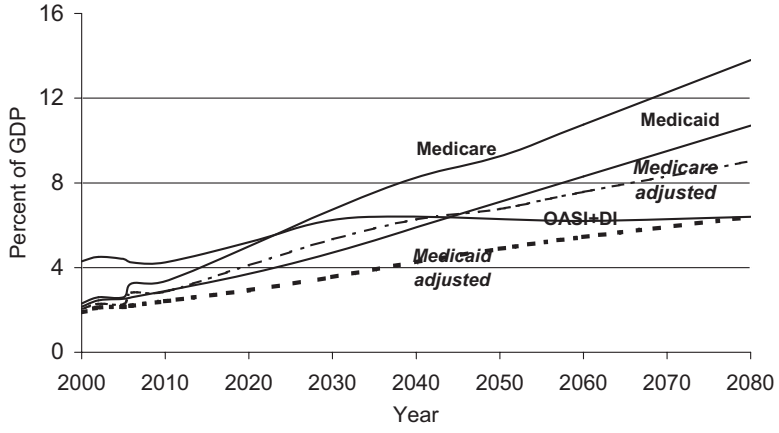

Figure 3

Projected (to 2080) Social Security, Medicare, and Medicaid GDP shares with, and without, adjustments to Medicare and Medicaid cost for disability declines and improved health (from Reference 76).

in human capital, can be understood from **Figure 3**.

In **Figure 3** we examine the projected long-term trajectory of increases in Medicare and Medicaid spending assessed in terms of the proportion of GDP consumed by each program. In addition, we present the proportion of GDP projected to be consumed by Social Security.

Figure 3 shows that Social Security, to 2080, represents only 6.4%–6.8% of GDP. Faster growth as a proportion of GDP is projected, however, for both Medicare and Medicaid. The growth of Medicaid is of particular interest because much of Medicaid spending is for LTC of disabled and institutionalized persons, driven largely by increases in the U.S. elderly population. If disability prevalence does not change further by 2080, 24.5% of GDP could be expended on Medicare and Medicaid combined. The alternate (dotted lines) trajectories reflect the proportion of GDP consumed by Medicare and Medicaid assuming a continuation of the 1.5% per annum decline in disability prevalence. The 1.5% decline would reduce the proportion of GDP spent on Medicare and Medicaid to ~14%. If the 1.5% per annum decline is not preserved or, if as certain researchers suggest, disability increases were to begin in 2012–2015, the consequences for the U.S. economy would be extremely serious.

Significant increases in health spending can be economically justified if that investment results in increased health care productivity measured in terms of improved health and functioning. Hall & Jones (41) suggest that health spending could rise to 30% of GDP by 2050 owing to increases in the value of improved health as U.S. society becomes more affluent, i.e., that the marginal elasticity of health care consumption is higher than for most nonhealth care consumer goods (23). Furthermore, the labor force is aging faster than the population owing to increased demands for education (103). Thus preservation of human capital at later ages will become increasingly important in promoting future U.S. economic growth (76). This becomes a critical issue for future biomedical research in that, by 2040, the prevalence of chronic disability below age 85 will have progressed to levels such that, to preserve the 1.5% per annum rate of decline, improvements in health and functioning will have to occur at increasingly older ages—ages (e.g., 95+) where interventions in chronic morbidity and disability processes have been less well studied. A better scientific understanding of the relation of obesity, sarcopenic obesity, inflammatory

processes, and disability will thus be required (20).

CONCLUSIONS

Substantial epidemiological, clinical, and demographic evidence suggests that there have been long-term improvements in health and functioning in the U.S. elderly population and that the rate of improvement has recently increased. Evidence shows that recent decreases in disability rates are increasingly due to biomedical research and its translation into clinical innovations, controlling risk factor levels and early disease and affecting the rate of progression of major chronic diseases such as CVD and, most recently, cancer.

Some economists and public health experts have expressed concern that increases in obesity, especially in younger adults, may reduce and eventually reverse the rate of disability improvement. Evidence against this suggests that public health and clinical innovations have already strongly attenuated the historical linkage of obesity and CVD morbidity. A question of importance is thus precisely how, and to what degree, this linkage has been broken. Areas where substantial data indicate that the linkage was most attenuated are among obesity, diabetes mellitus type II, and CVD.

The age-related linkages of obesity, inflammation, sarcopenia, and diabetes with disability and longevity are complex and currently not fully understood. Furthermore, no current national longitudinal U.S. survey contains the detailed obesity measures to assess these linkages satisfactorily. Equally obvious is that, despite medical progress, the lack of a socio-economically and ethically equitable distribution of existing clinical and public health services effective against CVD and its risk factors means there is still considerable additional progress that can be made against such diseases with existing biomedical interventions. Indeed, a study of the counterfactual case (e.g., comprising the obesity, diabetes, CVD risk factor relation of 30–40 years ago compared with the findings by Flegal and others) suggests that without the progress made against stroke and CVD risk factors, in the intervening 40 years the health burden of chronic disability, and health expenditures, that would manifest because of observed obesity prevalence increases would have been considerable. Thus the story that is currently being written may be one of a disability epidemic that has been largely avoided to date by advances in the biomedical management of the CVD consequences of obesity.

There still exist many areas where additional significant progress in reducing disability can be made with improvements in the social and economically distributive mechanisms of health care with a need for additional biomedical research on the management of specific types of obesity and basic metabolic processes. For improvements to continue to 2080, interventions must be made at increasingly late ages, taking advantage of the move from palliative, or possibly curative, health care to reliance on molecular medicine with a health system focused increasingly on underlying physiological processes (e.g., cytokine dynamics) and functional regeneration, rather than on treating discrete disease entities (i.e., regaining physiological reserves of major organ systems).

SUMMARY POINTS

1. Significant declines in the prevalence of chronic disability have occurred between 1982 and 2005; the annual rate of decline is accelerating.
2. Concurrent with declines in chronic disability prevalence have been declines in inflation-adjusted per capita Medicare expenditures for nondisabled persons.

3. Slow but continuous declines in chronic disability prevalence have been observed since the beginning of the twentieth century.
4. Declines in chronic disability have been observed concurrently with increases in U.S. life expectancy.
5. CVD risk factor trends in the United States, except for BMI, have tended to be positive in the past 40 years as assessed in the NHANES.
6. The prevalence of several major chronic diseases has tended to increase with increases in diabetes mellitus type II explained in part by improvement in population screening.
7. Within certain groups (e.g., obese, diabetics), other risk factors and certain morbid outcomes are being better managed.
8. Starting in 1994 the risk of entering, and over time, the absolute size of, the U.S. elderly nursing home population has declined.

UNRESOLVED ISSUES

1. What will be the future effect on chronic disability of obesity in the elderly?
2. What is the relation of obesity at early and middle ages to late-age disability and mortality?
3. What is the potential for reductions in disability at ages 95+?
4. With the growing ability to make more precise diagnoses, how will the average functional consequences of the increased prevalence of specific disease diagnoses change?
5. What are the implications of recent changes of the lethality of diseases such as AIDS and cancer for the future prevalence of chronic disability?
6. What will be the future balance of public health innovations, and new clinical treatments, in controlling the prevalence of chronic disability?
7. How should disability be assessed for determining the growth of human capital in the U.S. population aged 65 and above?
8. What are major controllable risk factors for chronic disability in the elderly?

DISCLOSURE STATEMENT

The author is not aware of any biases that might be perceived as affecting the objectivity of this review.

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