

Compression of Morbidity and Mortality: New Perspectives¹

Eric Stallard, ASA, MAAA, FCA²

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² Research Professor and Associate Director of the Center for Population Health and Aging, Duke Population Research Institute and Social Science Research Institute, Duke University, Durham, NC 27708 USA

ABSTRACT

Compression of morbidity is a reduction over time in the total lifetime days of chronic disability, reflecting a balance between (1) morbidity incidence rates and (2) case-continuance rates—generated by case-fatality and case-recovery rates. Chronic disability includes limitations in activities of daily living and cognitive impairment, which can be covered by long-term care insurance.

Morbidity improvement can lead to a compression of morbidity if the reductions in age-specific prevalence rates are sufficiently large to overcome the increases in lifetime disability due to concurrent mortality improvements and progressively higher disability prevalence rates with increasing age.

Compression of mortality is a reduction over time in the variance of age at death. Such reductions are generally accompanied by increases in the mean age at death; otherwise, for the variances to decrease, the death rates above the mean age at death would need to increase, and this has rarely been the case.

Mortality improvement is a reduction over time in the age-specific death rates and a corresponding increase in the cumulative survival probabilities and age-specific residual life expectancies. Mortality improvement does not necessarily imply concurrent compression of mortality.

This paper reviews these concepts, describes how they are related, shows how they apply to changes in mortality over the past century and to changes in morbidity over the past 30 years, and discusses their implications for future changes in the United States.

INTRODUCTION

Compression of morbidity (Fries 1980) is a reduction over time in the total lifetime days of chronic disability, reflecting a balance between morbidity incidence rates and case-continuance rates, the latter of which are generated by case-fatality and case-recovery rates. In this paper, chronic disability is defined to include limitations in activities of daily living (ADLs) and cognitive impairment (CI). These two risks are the most important risks with respect to the potential loss of independence in the elderly population; these are the only risks covered by tax-qualified long-term care insurance (Internal Revenue Service 1997). Other definitions of chronic disability may include diagnosed diseases. However, such diseases, without concurrent ADL and CI limitations, may occur earlier in the disablement process (Verbrugge and Jette 1994), and may exhibit more complex patterns of temporal changes than those exhibited by the patterns of changes in ADL and CI limitations.

Thus, our definition of morbidity compression focuses on the reduction in lifetime ADL and/or CI disability days. Our definition is relevant because there were large declines in the United States from 1984–2004 at age 65+ (the observation period and age range for the National Long Term Care Survey, or NLTCs) in both types of lifetime disability days, for males and females. Our analyses of the NLTCs using this definition indicated that the relative rates of decline in ADL expectancies were similar and very substantial for both males and females (17.5 percent and 19.0 percent, respectively); the relative rates of decline in CI expectancies were even larger (27.7 percent and 36.1 percent, respectively). Also of note, the absolute levels of ADL and CI expectancies were 1.9–2.2 times larger for females.

Compression of mortality is a reduction over time in the variance, or variability, of age-at-death, leading to progressively more “rectangular” survival functions (Olshansky, Carnes and Desesquelles 2001).

Each of the two types of compression is complex, making it difficult to assess the implications of change in one measure for change in the other. Mortality compression is generally accompanied by increases in the mean age-at-death (also called life expectancy, or LE). Otherwise, death rates at older ages would need to increase for the variances to decrease. Mortality compression causes a rectangularization of the lifetime survival function.

There were large rectangularization effects over the entire 20th century at age 0+, but recently these effects were much less, as were longer-term effects recomputed for age 10+ (Edwards and Tuljapurkar 2005). Theoretical lower limits of the variances of ages at death suggest the potential for future rectangularization effects is limited (Tuljapurkar and Edwards 2011), with the result that future changes will mostly comprise approximately parallel shifts of survival functions at age 65+. Average ages at death (LEs) should continue to increase in the United States. As of 2007, international LE rankings at birth (males 25th, females 27th) and age 65 (males 18th, females 24th) indicate large potentials for LE gains, without any effective biological constraints (NCHS 2011).

Most importantly, mortality compression is not necessary for morbidity compression. Mortality improvement, with static morbidity rates, would lead to increased morbidity (termed a survival increment; e.g., for ADL and/or CI disability over the period 1984–2004, the survival increments were 0.44 year for males and 0.24 year for females). Actual mortality improvement, without mortality compression, has been counterbalanced by an even greater reduction in morbidity (termed a morbidity decrement; e.g., for ADL and/or CI disability over the period 1984–2004, the morbidity decrements were 0.83 year for males and 1.21 years for females).

The remainder of this paper reviews these concepts, describes how they are related, shows how they apply to changes in mortality over the past century and to changes in morbidity over the past 30 years, and discusses their implications for future changes in the United States.

DATA

The primary data source for the mortality analysis was the Social Security Administration's database on sex-specific life tables described in Actuarial Study No. 120 (Bell and Miller 2005).

The primary data source for the morbidity analysis was the National Long Term Care Survey (Manton, Stallard and Corder 1998; Manton, Gu and Lamb 2006). The NLTCs was designed to measure disability and use of long-term care (LTC) among a representative sample of the U.S. elderly (age 65+) population at multiple points in time from 1982 to 2004. The cumulative sample size (n) over all six survey years (waves) was 49,258 distinct people.

The six survey years were 1982, 1984, 1989, 1994, 1999 and 2004. Each wave consisted of a telephone screener interview followed by an in-person detailed interview for those respondents who met various disability screening criteria (designated as "screen-ins"). In-person screening visits were also conducted for those respondents who could not be contacted by telephone, followed by detailed interviews for those who screened-in. The number of people who completed the screener interviews defined the cross-sectional sample size for each survey year.

Each survey year, the cross-sectional sample size was in the range 16,000–21,000, with approximately 6,000–7,500 detailed in-person interviews for people who met various disability screening criteria. Detailed interviews were conducted for both community and institutional residents at all survey years except for 1982, when the fact of institutionalization was noted without further information being collected. The institutional detailed interview was a shortened, modified form of the community detailed interview with sample sizes in the range 970–1,770 for the period 1984–2004.

Disability included basic and instrumental ADL (abbreviated as ADL and IADL, respectively) impairments whose duration had lasted or were expected to last three or more months, cognitive impairment, and institutionalization in a nursing home or similar LTC facility. During the later waves of the NLTCs, the options for residing in an assisted living facility (ALF) expanded substantially. Approximately half of the ALF residents in 2004 were classified as institutionalized using the standard temporally consistent NLTCs protocol for making this determination.

In this paper, we use ADL and CI disability measures designed to be maximally compatible with the 1996 federal Health Insurance Portability and Accountability Act (HIPAA) requirements for tax-qualified long-term care insurance and services (Internal Revenue Service 1997); for details, see Stallard and Yashin (2014).

METHODS

We use life table survival functions directly to visualize the rectangularization process, and to compute the life expectancies and standard deviations of length of life at the corresponding calendar years. All calculations were based on the "period" life tables, which reflect the mortality conditions for all ages for which people are alive in each given calendar year, not the alternative "cohort" life tables, which reflect the actual survival for a population born in a given calendar year and followed, or projected, over time to extinction.

Hence, we define the life expectancy at age x in year y as follows:

$$e_{x,y} = \int_0^{\infty} {}_t p_{x,y} dt$$

where

$${}_t p_{x,y} = l_{x+t,y} / l_{x,y}$$

and

$$l_{x+t,y} = \text{probability of survival from birth to age } x+t \text{ in year } y.$$

The life expectancy value provides a summarization of the age-specific mortality probabilities in a given population at a given time. Our measures of the compression of mortality are based on the changes in the standard deviation of age at death in each associated life table, based on the method in Bowers et al. (1986, 63). The ratio of the standard deviation to the mean forms another measure, the coefficient of variation, which was recommended by Gavrilova et al. (2012) to supplement comparisons based on the standard deviation when the means are very different.

The period life table is readily extended to define the disabled life expectancy (DLE) at age x in year y using Sullivan's (1971) method, as follows:

$$e_{Dx,y} = \int_0^{\infty} {}_t p_{x,y} \pi_{x+t,y} dt$$

where

$${}_t p_{x,y} = l_{x+t,y} / l_{x,y}$$

and

$$\pi_{x+t,y} = \text{disability prevalence at age } x+t, \text{ in year } y.$$

The disabled life expectancy value provides a summarization of the age-specific disability prevalence rates in a given population at a given time.

The change from year y_0 to year $y > y_0$ in disabled life expectancy at age x can be decomposed into two components: (1) a survival increment (SI) that reflects the increase in DLE which would have occurred had the disability prevalence rates remained constant; and (2) a morbidity decrement (MD) that reflects the decrease in DLE which would have occurred had the survival function remained constant. Hence,

$$\begin{aligned} e_{Dx,y} - e_{Dx,y_0} &= \int_0^{\infty} ({}_t p_{x,y} \pi_{x+t,y} - {}_t p_{x,y_0} \pi_{x+t,y_0}) dt \\ &= \int_0^{\infty} ({}_t p_{x,y} - {}_t p_{x,y_0}) \pi_{x+t,y_0} dt \quad \text{(Survival Increment)} \\ &\quad - \int_0^{\infty} {}_t p_{x,y} (\pi_{x+t,y_0} - \pi_{x+t,y}) dt \quad \text{-- (Morbidity Decrement).} \end{aligned}$$

The following comments are relevant:

1. For the DLE to decline from y_0 to y , the morbidity decrement must be larger than the survival increment.

2. If the morbidity decrement is positive but smaller than the survival increment, the DLE will increase despite the fact that morbidity has improved.

The first and second cases are the two possible forms of morbidity improvement. The first case is of particular interest to LTC insurance actuaries because it is the only case where the total lifetime days of chronic disability at and beyond age x declines. It constitutes a special form of morbidity improvement called morbidity compression (Fries 1980, 1983, 1989, 2005). The second case is a form of morbidity improvement that occurs without morbidity compression. Indeed, for this case, the total lifetime days of chronic disability at and beyond age x would increase, thereby generating a morbidity expansion. There is a third possibility, that of a negative morbidity decrement—termed morbidity deterioration—which, in combination with mortality improvement, would also generate a morbidity expansion, but this possibility is not considered further in this paper.

A common alternative form of summarization of the age-specific morbidity or disability rates for a given calendar year is based on age standardization. We define the age-standardized disability rate in year y as a function of the age-specific disability prevalence rates applied to some arbitrary standard vector of age-specific population counts, as follows:

$$\text{ASDR}_y(\{N_x\}) = \frac{\sum_{x=65}^{\omega} N_x \cdot \pi_{x,y}}{\sum_{x=65}^{\omega} N_x}$$

where

N_x = Standard (mid-year) population at age x

and

$\pi_{x,y}$ = Disability prevalence rate at age x in year y .

Age standardization is used by demographers to make cross-temporal comparisons, i.e., temporal differences in the population age structure are controlled by using a constant age structure in all comparisons.

RESULTS

Compression of Mortality

We begin by visualizing the rectangularization process, using the Social Security Administration (SSA) life tables for the period 1900–2000 shown in figures 1 and 2 for males and females, respectively.

The survival curves for males and females both display the well-known property of rectangularization, whereby the survival function values at each age initially appear to move upward while the ages at which the largest declines in the function occur appear to move progressively to the right. As noted above, the life expectancy in each calendar year is the area under the corresponding survival curve. For males in 1900, the LE was 46.0 years, which increased to 74.0 years in 2000 (table 1); for females, the corresponding values were 48.6 years and 79.4 years, respectively (table 2).

Figure 1: Life Table Survival Functions, U.S. Males by Calendar Year 1900-2000 (SSA Data)

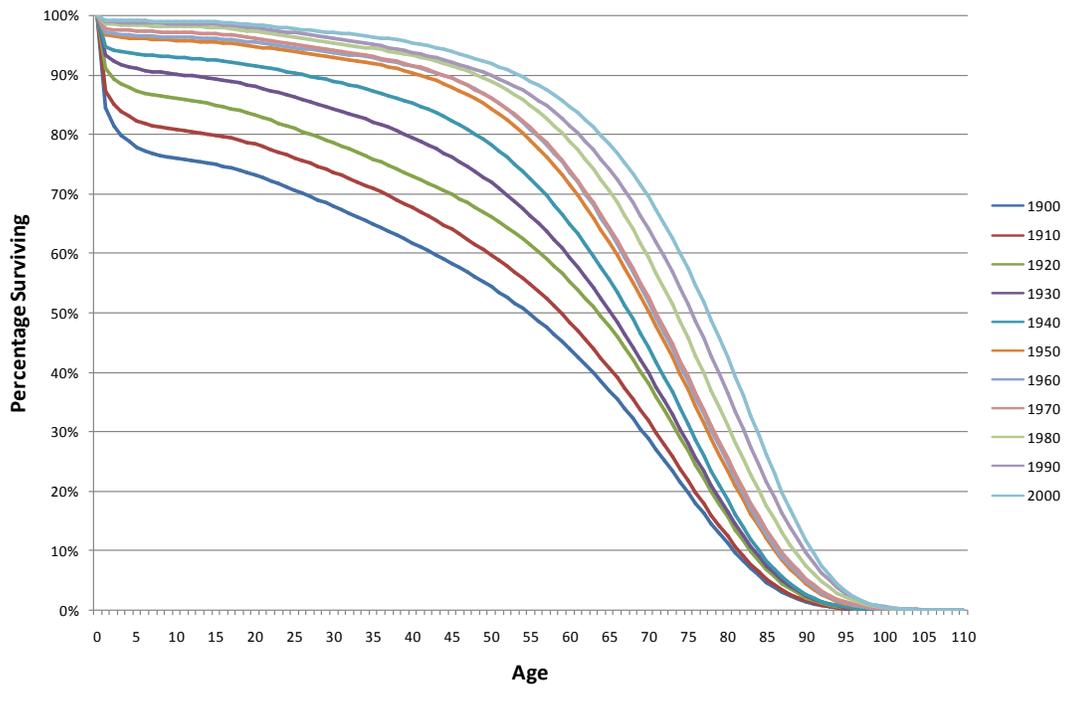
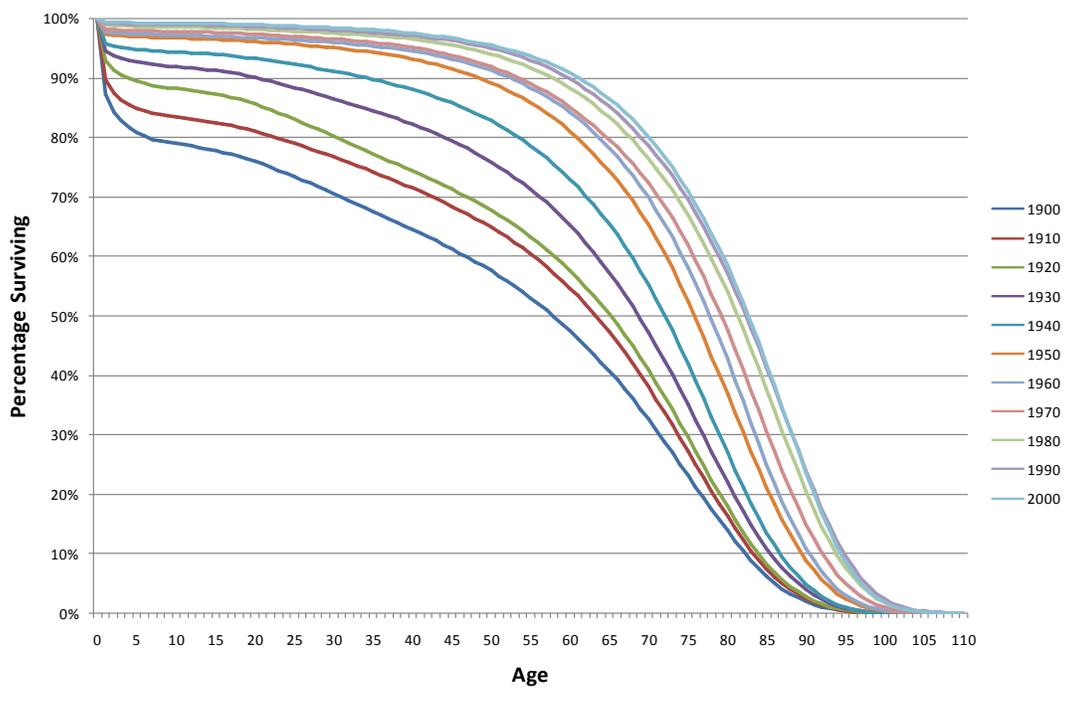


Figure 2: Life Table Survival Functions, U.S. Females by Calendar Year 1900-2000 (SSA Data)



Edwards and Tuljapurkar (2005) noted that the rectangularization effects at age 0+ were much less when recomputed for age 10+, which they argued is a better anchor point for studying divergences in mortality in developed countries.

The altered pattern of rectangularization can be seen clearly in figures 3 and 4. For males in 1900, the LE at age 10 was 50.0 years, which increased to 64.8 years in 2000 (table 1); for females, the corresponding values were 51.1 years and 70.0 years, respectively (table 2).

For both sexes, the movement between the adjacent curves appears to be more evenly spaced than in the previous figures.

Given our focus on morbidity compression at older ages, it will be informative to review the patterns of rectangularization at ages 65 and above. These are shown in figures 5 and 6. For males in 1900, the LE at age 65 was 11.3 years, which increased to 15.9 years in 2000 (table 1); for females, the corresponding values were 12.0 years and 19.0 years, respectively (table 2).

For both sexes, the movement between the adjacent curves appears to be very much slower for the period 1900–1940 than in the previous two figures, with more rapid changes thereafter.

Quantification of the extent of rectangularization of each survival curve was based on the standard deviation of the ages of death for the ages underlying each curve. These statistics are presented in tables 1 and 2 for males and females, respectively, along with the corresponding life expectancies and coefficients of variation.

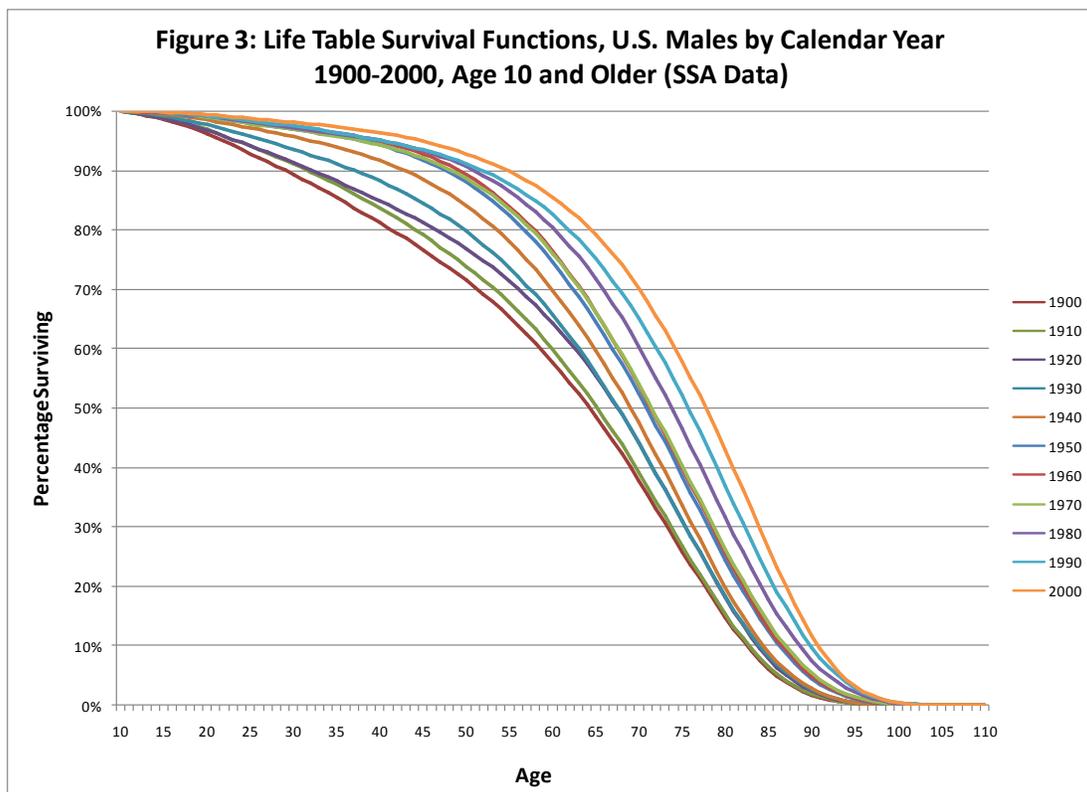


Figure 4: Life Table Survival Functions, U.S. Females by Calendar Year 1900-2000, Age 10 and Older (SSA Data)

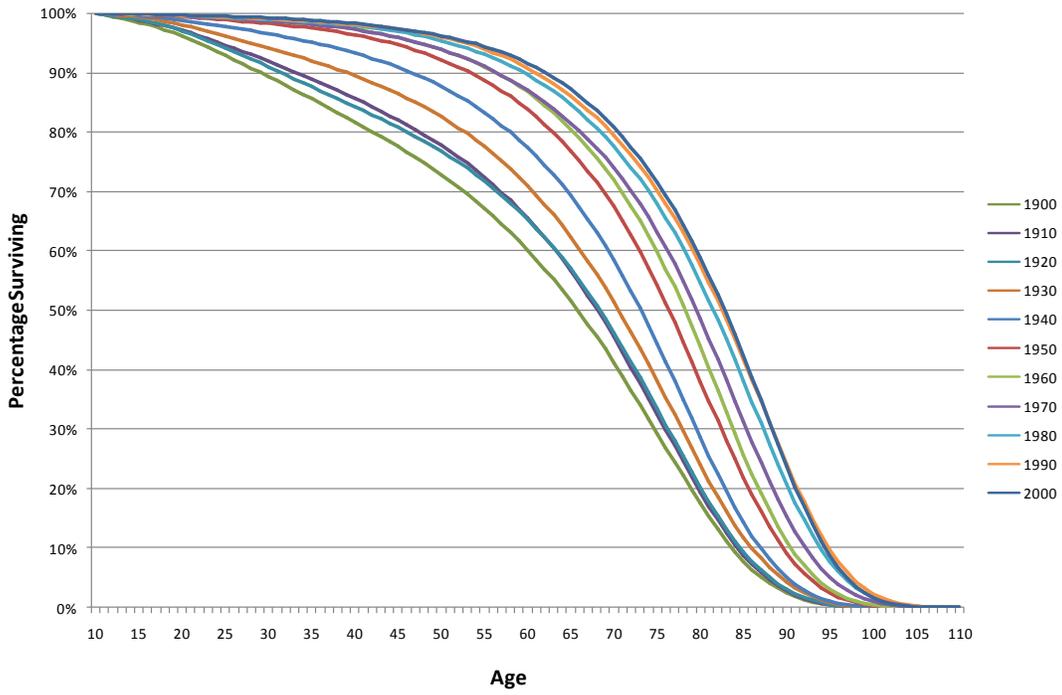
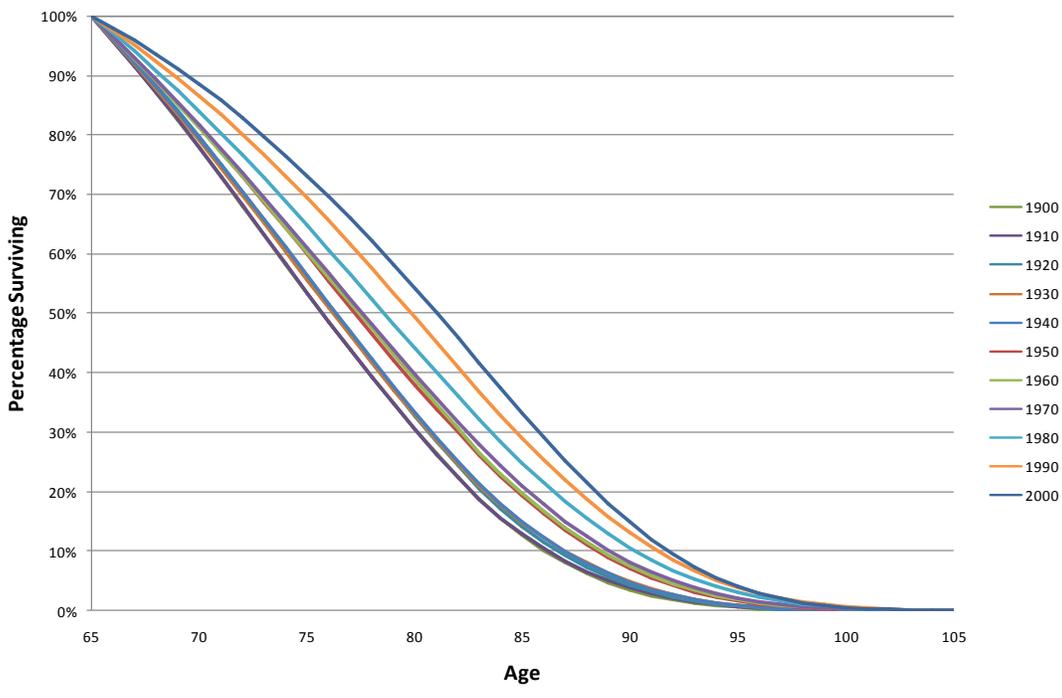
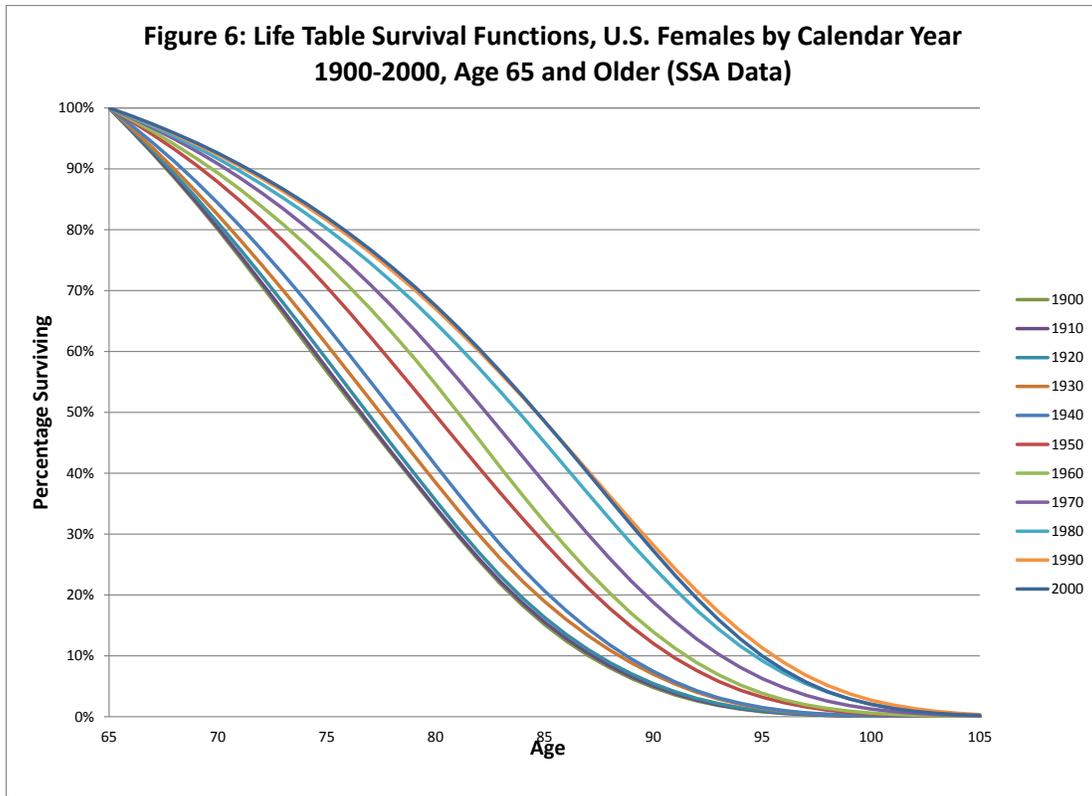


Figure 5: Life Table Survival Functions, U.S. Males by Calendar Year 1900-2000, Age 65 and Older (SSA Data)





For both sexes, the standard deviations for age 10+ reached a relative low in 1960 followed by variations in a narrow range, consistent with the curve-shifting to the right seen in figures 3 and 4. In contrast, the standard deviations for age 65+ increased gradually over the entire set of life tables, indicating there was increasing variability in the ages at death in the elderly population. The coefficients of variation exhibit patterns consistent with the absence of compression above age 10 for the later years.

**Table 1
Means, Standard Deviations, and Coefficients of Variation of Ages at Death, by Calendar Year,
Starting At Birth, Age 10, and Age 65, U.S. Males (SSA Data)**

Year	Life Expectancy, at			Standard Deviation			Coefficient of Variation (%)		
	Age 0	Age 10	Age 65	Age 0+	Age 10+	Age 65+	Age 0+	Age 10+	Age 65+
1900	46.0	50.0	11.3	30.3	19.8	7.0	66.0	39.5	62.0
1910	49.7	51.2	11.4	29.1	19.1	7.1	58.5	37.3	62.2
1920	54.3	52.9	11.8	27.7	19.2	7.2	51.1	36.4	60.6
1930	57.8	54.0	11.8	25.3	18.0	7.3	43.7	33.3	61.7
1940	61.5	56.0	11.9	23.0	16.6	7.3	37.5	29.7	60.9
1950	65.6	58.4	12.8	20.5	15.8	7.7	31.2	27.1	60.3
1960	66.7	59.2	12.9	19.8	15.5	7.8	29.7	26.1	60.5
1970	67.2	59.1	13.1	19.3	16.0	7.9	28.8	27.0	60.4
1980	69.9	61.2	14.0	18.3	15.8	8.2	26.1	25.8	58.4
1990	71.8	62.8	15.1	17.8	15.9	8.3	24.8	25.3	55.2
2000	74.0	64.8	15.9	16.8	15.2	8.3	22.6	23.4	51.9

Source: Author's calculations based on SSA life tables.

Table 2
Means, Standard Deviations, and Coefficients of Variation of Ages at Death, by Calendar Year, Starting At Birth, Age 10, and Age 65, U.S. Females (SSA Data)

Year	Life Expectancy, at			Standard Deviation			Coefficient of Variation		
	Age 0	Age 10	Age 65	Age 0+	Age 10+	Age 65+	Age 0+	Age 10+	Age 65+
1900	48.6	51.1	12.0	30.1	20.2	7.3	62.0	39.4	60.4
1910	53.3	53.6	12.1	28.9	19.1	7.3	54.2	35.7	60.2
1920	56.1	53.4	12.3	27.1	19.6	7.3	48.3	36.8	59.4
1930	61.2	56.5	12.9	24.8	18.1	7.6	40.5	32.0	58.6
1940	65.7	59.6	13.4	22.4	16.4	7.6	34.1	27.5	56.3
1950	71.1	63.5	15.1	19.6	15.0	8.0	27.5	23.6	53.2
1960	73.3	65.4	15.9	18.7	14.3	8.0	25.5	21.9	50.7
1970	74.9	66.5	17.1	18.4	14.8	8.4	24.5	22.3	49.3
1980	77.5	68.7	18.4	17.1	14.4	8.8	22.0	21.0	48.0
1990	78.9	69.7	19.1	16.3	14.3	9.0	20.7	20.5	47.1
2000	79.4	70.0	19.0	15.4	13.8	8.7	19.4	19.7	46.1

Source: Author's calculations based on SSA life tables.

Compression of Morbidity

The available information for our assessment of the compression of morbidity derives from the National Long Term Care Survey, covering the 20-year period 1984–2004, a period selected because it was the longest possible period that could be considered from this data source. Tables 3 and 4 present the age-specific and total prevalence rates for 1984 and 2004 for males and females, respectively, meeting either the HIPAA ADL or CI disability triggers.

The overall decline for males was 25.9 percent, but this increased to 39.5–39.7 percent with age standardization, which provided substantially more accurate summarizations of the age-specific relative changes. Alternatively, the combined ADL/CI prevalence rates declined 2.50 percent per year, or 25 percent per decade. The standard errors are shown along with the *t*-statistics, which were highly statistically significant.

The overall decline for females was 20.0 percent, but this increased to 34.8–35.5 percent with age standardization. The *t*-statistics were also highly statistically significant for these trends.

Tables 5 and 6 present the corresponding life expectancies and disabled life expectancies and the decompositions of the DLE changes into the survival increments and morbidity decrements, as described above.

The DLEs for males were 1.64 years in 1984 and 1.26 years in 2004. The corresponding DLEs for females were 3.26 and 2.29 years, respectively. The declines in DLEs were 0.39 year for males and 0.97 year for females, which represented, respectively, 23.5 percent of the 1984 DLE for males and 29.8 percent of the 1984 DLE for females. The *t*-statistics indicated that the reductions in DLE were highly statistically significant for both sexes, as were the component survival increments and morbidity decrements. It is these reductions in DLE that constituted the compression of morbidity which we were seeking to quantify.

Figures 7 and 8 display the joint relative survival functions for males in 1984 and 2004 under the Sullivan (1971) method for calculating DLE, based on the static hierarchy of no-disability, CI only, ADL impairment only, and combined ADL and CI impairments. Corresponding results for females are displayed in figures 9 and 10. For both sexes, there was a visually apparent reduction in the amount of disability between 1984 and 2004. In addition, the figures, especially figure 9, clearly display much higher levels of disability for females.

Table 3
Percent of Population Meeting Either HIPAA Trigger, United States 1984 and 2004, Males, Age 65 and Above, by Age and Totaled Over Age, with Two Modes of Age Standardization

Age	1984	2004	Change	% Change	Annual Rate of Decline; 20 yr.
65-69	4.0	2.5	-1.5	-37.7	2.34%
70-74	7.8	4.4	-3.45	-44.1	2.87%
75-79	11.2	7.6	-3.69	-32.8	1.97%
80-84	18.3	11.0	-7.30	-39.9	2.51%
85-89	30.5	17.4	-13.09	-42.9	2.76%
90-94	47.6	28.3	-19.26	-40.5	2.56%
95+	64.9	38.5	-26.44	-40.7	2.58%
Total	10.1	7.5	-2.61	-25.9	1.49%
1984 ASDR	10.1	6.1	-3.98	-39.5	2.48%
2004 ASDR	12.4	7.5	-4.92	-39.7	2.50%
Standard Error					
Total	0.34	0.34	0.48		
1984 ASDR	0.34	0.30	0.45		
2004 ASDR	0.41	0.34	0.53		
<i>t</i> -statistic					
Total	30.01	21.91	-5.45		
1984 ASDR	30.01	20.48	-8.87		
2004 ASDR	30.15	21.91	-9.21		

NOTE: ASDR denotes age-standardized disability rate; the 1984 ASDR and 2004 ASDR results were age-standardized, respectively, to the 1984 and 2004 NLTCs weighted male population. The HIPAA triggers are based on 2+ ADL Impairments or 3+ errors on the Short Portable Mental Status Questionnaire.

Source: Author's calculations based on the 1984 and 2004 NLTCs.

Table 4
Percent of Population Meeting Either HIPAA Trigger, United States 1984 and 2004, Females, Age 65 and Above, by Age and Totaled Over Age, with Two Modes of Age Standardization

Age	1984	2004	Change	% Change	Annual Rate of Decline; 20 yr.
65-69	4.5	3.1	-1.4	-31.4	1.87%
70-74	7.6	4.4	-3.20	-42.1	2.69%
75-79	13.2	8.1	-5.10	-38.6	2.41%
80-84	24.4	14.7	-9.69	-39.7	2.50%
85-89	40.4	28.3	-12.10	-30.0	1.76%
90-94	61.0	38.8	-22.14	-36.3	2.23%
95+	76.1	63.8	-12.23	-16.1	0.87%
Total	15.0	12.0	-3.00	-20.0	1.11%
1984 ASDR	15.0	9.7	-5.32	-35.5	2.17%
2004 ASDR	18.4	12.0	-6.40	-34.8	2.12%
Standard Error					
Total	0.31	0.34	0.46		
1984 ASDR	0.31	0.30	0.43		
2004 ASDR	0.36	0.34	0.50		
<i>t</i> -statistic					
Total	49.03	35.07	-6.55		
1984 ASDR	49.03	32.51	-12.48		
2004 ASDR	51.11	35.07	-12.92		

NOTE: ASDR denotes age-standardized disability rate; the 1984 ASDR and 2004 ASDR results were age-standardized, respectively, to the 1984 and 2004 NLTCs weighted female population. The HIPAA triggers are based on 2+ ADL Impairments or 3+ errors on the Short Portable Mental Status Questionnaire.

Source: Author's calculations based on the 1984 and 2004 NLTCs.

Table 5
Components of Change in Male Life Expectancy and HIPAA ADL/CI
Expectancy (in Years at Age 65), United States 1984 and 2004

At Age 65	Year			Survival Increment	Morbidity Decrement
	1984	2004	Change		
Life Expectancy	14.41	16.67	2.26	2.26	—
HIPAA ADL/CI Expectancy	1.64	1.26	-0.39	0.44	0.83
Standard Error	0.05	0.06	0.08	0.02	0.09
<i>t</i> -statistic	30.54	21.97	-4.93	25.75	9.25

Source: Author's calculations based on the 1984 and 2004 NLTCs.

Table 6
Components of Change in Female Life Expectancy and HIPAA ADL/CI
Expectancy (in Years at Age 65), United States 1984 and 2004

At Age 65	Year			Survival Increment	Morbidity Decrement
	1984	2004	Change		
Life Expectancy	18.66	19.50	0.84	0.84	—
HIPAA ADL/CI Expectancy	3.26	2.29	-0.97	0.24	1.21
Standard Error	0.06	0.06	0.09	0.01	0.09
<i>t</i> -statistic	51.54	35.28	-10.70	47.01	12.87

Source: Author's calculations based on the 1984 and 2004 NLTCs.

DISCUSSION

Our results demonstrated a very substantial and highly statistically significant compression of morbidity for both males and females in the United States over the period 1984–2004, based on our assumption that the term morbidity could be operationalized using the HIPAA ADL and CI triggering criteria. We acknowledge that analyses using different definitions of morbidity or focusing on specific subpopulations could lead to different results. For example, Crimmins and Beltran-Sanchez (2011) reported an expansion of morbidity from 1998–2008 in a study where morbidity was defined in terms of loss of mobility functioning among the noninstitutionalized population.

We believe our choice of HIPAA-based measures of morbidity combined with a data source (i.e., the NLTCs) that covers all subgroups of the elderly population was more appropriate for addressing the issues in this paper.

Figure 7: Joint Relative Survival at Ages 65+, Meets Either HIPAA Trigger, United States, 1984, Males

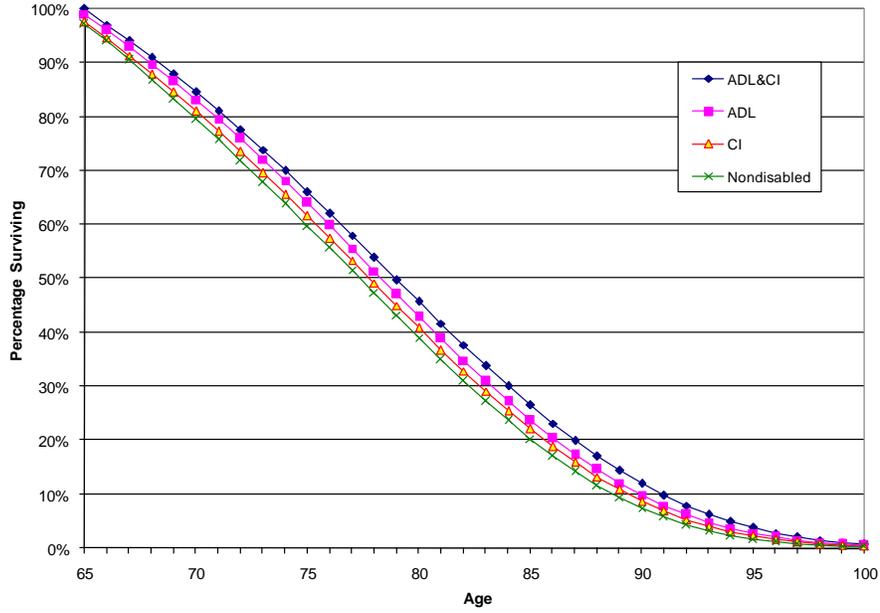


Figure 8: Joint Relative Survival at Ages 65+, Meets Either HIPAA Trigger, United States, 2004, Males

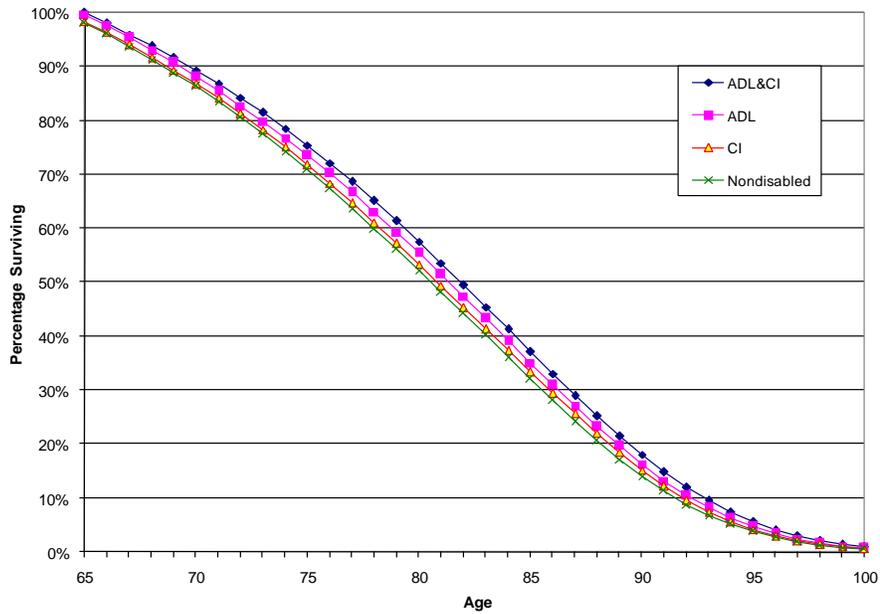


Figure 9: Joint Relative Survival at Ages 65+, Meets Either HIPAA Trigger, United States, 1984, Females

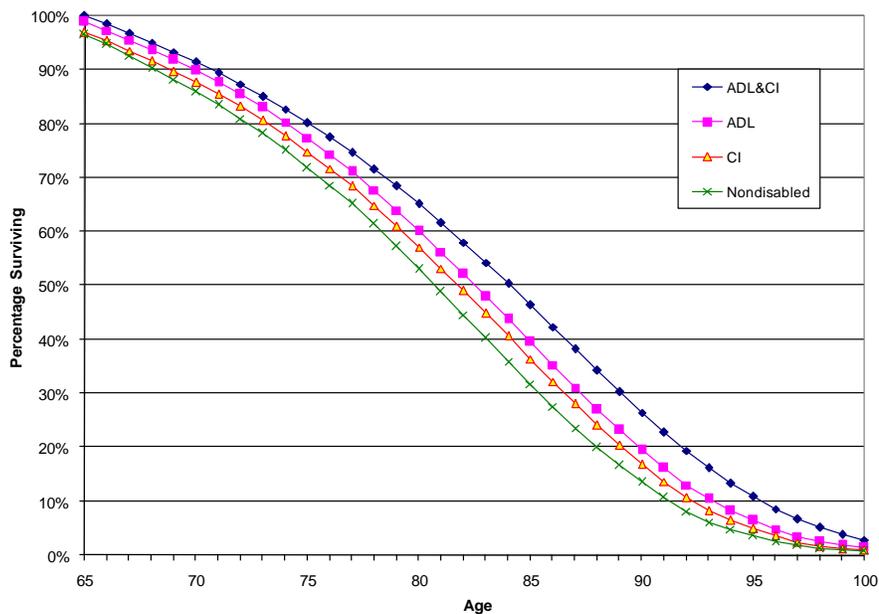
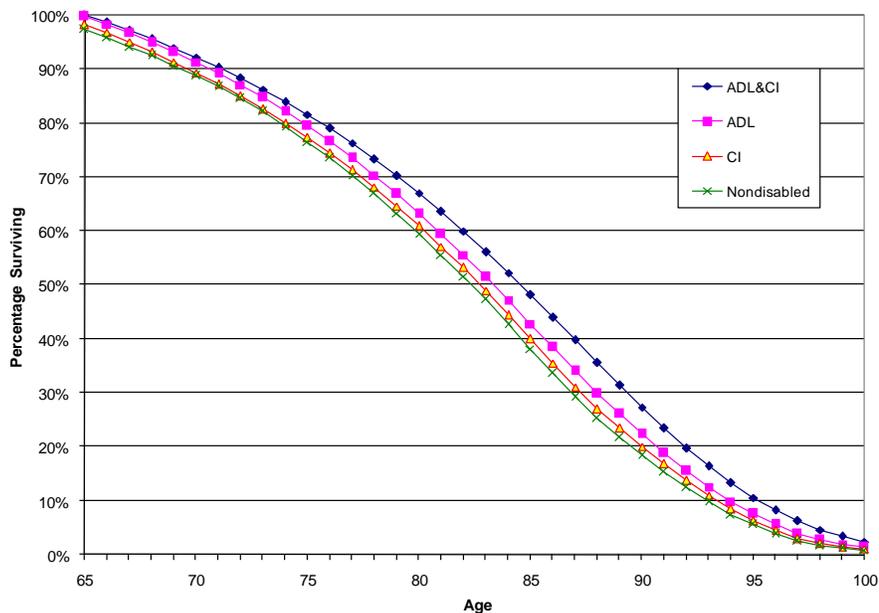


Figure 10: Joint Relative Survival at Ages 65+, Meets Either HIPAA Trigger, United States, 2004, Females



Over a much longer period, 1960–2000, there was a substantial slowdown in the degree of mortality compression, which was initially manifest as a rectangularization of the survival curve for all ages beyond birth. Analysis of the changes in the conditional survival curves for all ages

beyond age 10 or age 65 revealed that the process of mortality compression had mostly run its course by the latter half of the 20th century.

Combining these results, we see that a very substantial amount of morbidity compression occurred during a period in which there was little, if any, mortality compression. This finding indicated the two processes were not closely tied: morbidity compression did not require concurrent mortality compression.

Our final comments address the lower limits for the variances of the ages at death and their implications for future changes in mortality.

Tuljapurkar and Edwards (2011, 506) developed theoretical lower limits for the variances of ages at death which implied that the potential for future rectangularization effects is limited. They showed that, for the Gompertz mortality model with $\mu(x) = \mu_0 e^{\beta x}$, it follows that the variance of age at death is approximately $\sigma^2 \approx 1/\beta^2$. Assuming $\beta = 0.087$, they estimated that $\sigma = 11.5$ years, a value which could form a lower bound for the standard deviations of age at death above age 10 (e.g., tables 1 and 2).

The exact result for the Gompertz variance was previously given by Pollard and Valkovics (1992, equation 22) as $\sigma^2 = 1.644934/\beta^2$, which yields, for the above assumptions, $\sigma = 14.7$ years, a value too high to be a lower bound because it exceeds four of the last five standard deviation values above age 10 for females (table 2). Given the equivalent inverse relationship, $\sigma = 1.282550/\beta$, it follows that a lower σ value requires a higher β value. For example, $\beta = 0.10$ yields $\sigma = 12.8$ years while $\beta = 0.1282550$ yields $\sigma = 10.0$ years. These particular β values were selected to be near or above the upper limits of β values for human populations over the past century (e.g., see Zheng, Yang and Land 2011).

In each case, use of Pollard and Valkovics' (1992) refinement increases the lower bound σ value by 28 percent, which further strengthens the argument based on Tuljapurkar and Edwards (2011) that future rectangularization of the mortality survival curve will be limited. This suggests that right-shifting of survival will be the primary mechanism for future mortality improvement, and this may simplify some mortality forecasting models.

Pollard and Valkovics' (1992) refinement also indicates that the lower bound σ value is highly likely to be above 10 years, and is probably closer to 12.8 years. This is important because it indicates that the morbidity compression mechanism originally described by Fries (1980) in which the ideal average lifespan would be 85 years with a standard deviation of about four years is highly implausible. Fries (1989) increased the ideal standard deviation to seven to eight years, which, though close to the values for age 65 and above in tables 1 and 2, was still substantially below the 10.0–12.8 year lower bounds for ages 10 and above. Fries eventually realized the compression of morbidity did not actually require a fixed ideal average lifespan of 85 years with a four-, seven- or eight-year standard deviation; both statistics could be changed and indeed could continue to change over time if one alternative condition were met (Fries 2005): "Increases in the age of onset of chronic infirmity would have to be more rapid than increases in life expectancy." This condition was fully consistent with the formulation in this paper under which the compression of morbidity occurred only when the morbidity decrement exceeded the survival increment.

This poses three related questions: Will morbidity compression continue indefinitely? Will it reach a stable lower limit? Or will it reverse direction and become a morbidity expansion? Fries, Bruce and Chakravarty (2011) observed that the morbidity compression seen over the past 30 years was achieved without a coherent health-promotion strategy in place. They argued that continued morbidity compression was not inevitable, but it could be made to continue into the foreseeable

future using a four-part health-promotion strategy consisting of primordial prevention (risk-factor elimination), primary prevention (risk-factor reduction), secondary prevention (disease specific) and tertiary prevention (morbidity treatment/reduction). Moreover, these same efforts would likely lead to further reductions in mortality beyond those that would have occurred in their absence.

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