New Perspectives on the Compression of Morbidity and Mortality

THE DYNAMICS OF MORBIDITY AND MORTALITY are central concerns in actuarial practice, having major implications for life, health, pension, and long-term care (LTC) insurers; for Medicare, Medicaid, and Social Security programs; for public policy planners; and for career and retirement planning among the general public.

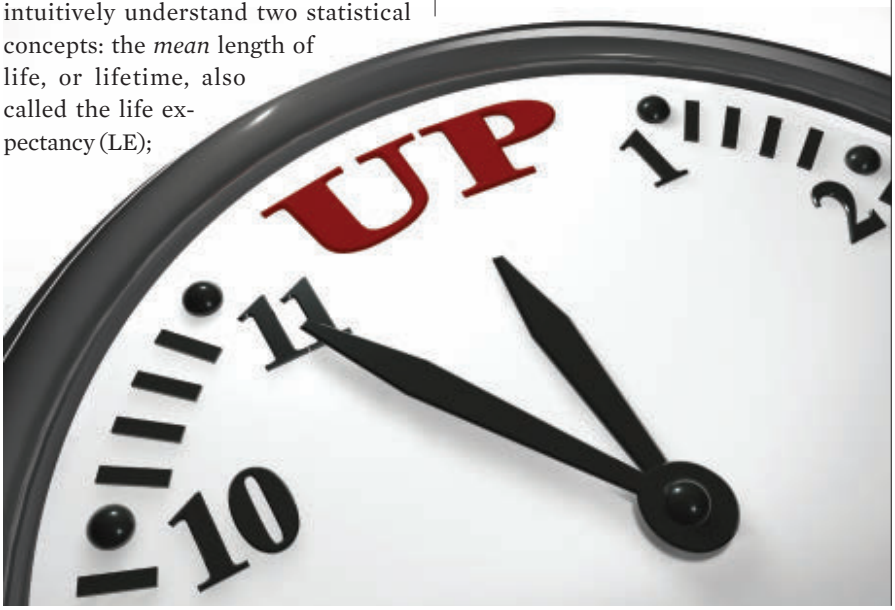
In his classic paper "Aging, Natural Death, and the Compression of Morbidity," published in 1980 in the *New England Journal of Medicine*, James Fries identified two aspects of those dynamics—the compression of morbidity and the compression of mortality—as critically important to a proper understanding of the past, which, in turn, was essential in developing valid forecasts of the future.

Despite the central importance of the two types of compression to Fries' thesis, his presentation of those concepts wasn't sufficiently rigorous to avoid a fundamental error that has continued uncorrected to the present time. His use of the word *rectangularization* where others have used *compression of mortality* underscores the lack of consistent terminology.

To continue, the reader needs to intuitively understand two statistical concepts: the *mean* length of life, or lifetime, also called the life expectancy (LE); and the *standard deviation* of lifetime.

- For any group of individuals, the *mean lifetime* is defined as the arithmetic average value of the individual lifetimes.
- For the same group of individuals, the *standard deviation of lifetime* measures the dispersion of the individual lifetimes around the mean lifetime. For technical reasons, the standard deviation is defined as the square root of the average sums of squares of deviations of the individual lifetimes from the mean lifetime.

The important intuitive concept is that the dispersion of the individual lifetimes is proportional to the standard deviation of lifetime. When the standard deviation declines, the dispersion declines, and vice versa. Both are typically measured in years.



Compression of Mortality

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

The survival curves for males and females in the top panels both display the well-known property of rectangularization—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.

The remaining LE at each age in each calendar year is computed as the area under the corresponding survival curve. By convention, this calculation assumes the existence of a hypothetical group exposed to the mortality conditions in effect during the selected calendar year. For males in 1900, the LE at birth (age 0) was 46 years, which increased to 74 years in 2000; for females, the corresponding values were 49 years and 79 years.

Ryan Edwards and Shripad Tuljapurkar noted in 2005 that the rectangularization effects starting at age 0 were greatly reduced when recomputed starting at age 10, which they argued was a better anchor point for studying divergences in mortality in developed countries. Starting at age 10 could also be justified using the ontogenesis model proposed by Daniel Levitis in 2011, in which declines in mortality between conception and maturity were tied to genetic and developmental malfunctions. Starting at age 10 is actually necessary to implement Fries' preferred method for quantifying the degree of rectangularization, as indicated below.

However justified, the pattern of rectangularization was altered substantially, as shown in the middle panels of Figure 1. For males in 1900, the LE at age 10 was 50 years, which increased to 65 years in

FIGURE 1: Life Table Survival Functions Starting at Ages 0, 10, and 65, U.S. Males and Females by Calendar Year 1900–2000 (SSA Data)

FIGURE 1A: U.S. Males, Starting at Age 0

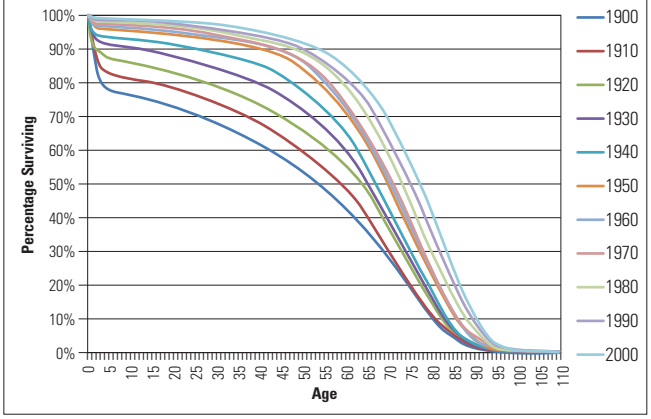


FIGURE 1B: U.S. Females, Starting at Age 0

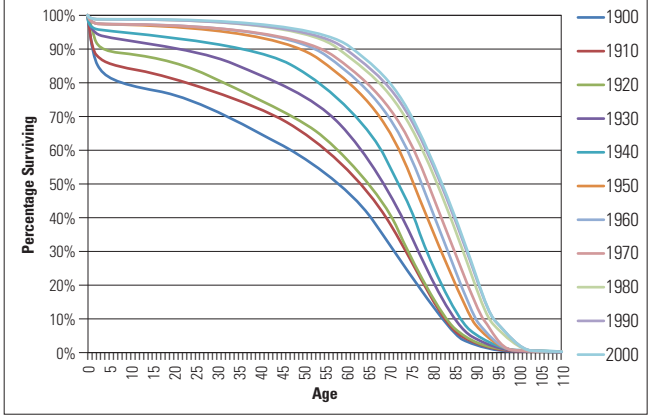


FIGURE 1C: U.S. Males, Starting at Age 10

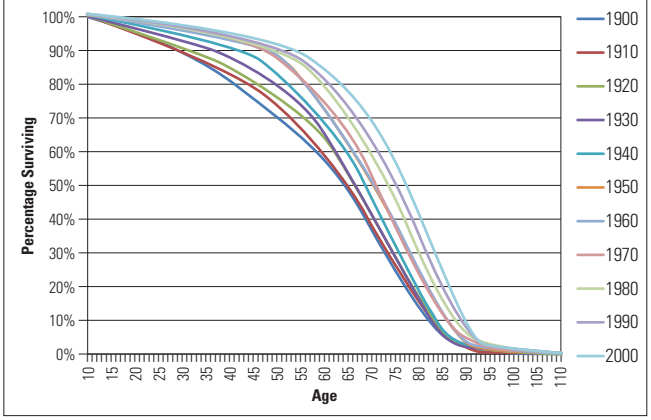


FIGURE 1D: U.S. Females, Starting at Age 10

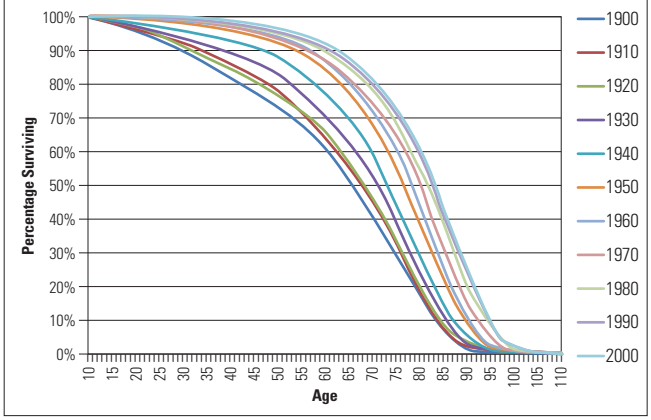


FIGURE 1E: U.S. Males, Starting at Age 65

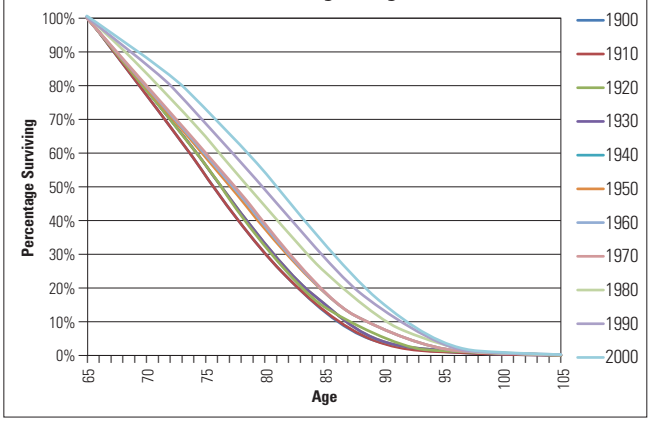
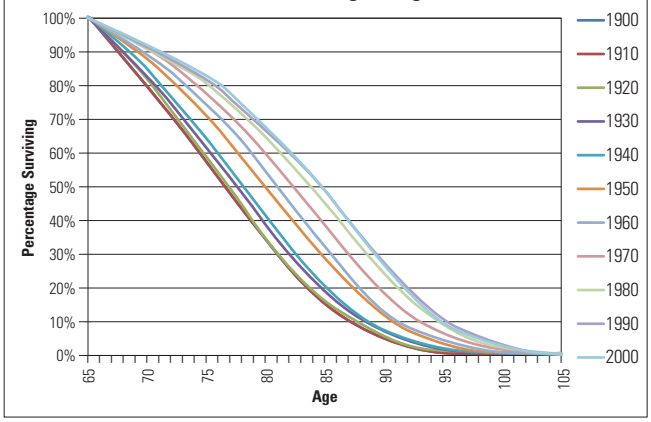


FIGURE 1F: U.S. Females, Starting at Age 65



SOURCE: AUTHOR

2000; for females, the corresponding values were 51 and 70 years. For both sexes, the cross-temporal movements between the adjacent curves were less variable than in the top panels.

The bottom panels of Figure 1 display the patterns of rectangularization starting at age 65. For males in 1900, the LE at age 65 was 11.3 years, which increased to 15.9 years in 2000; for females, the corresponding values were 12.0 and 19.0 years. For both sexes, the changes between the adjacent curves were slower during than after the period 1900–1940.

Whereas the changes in LEs shown in Figure 1 are well known, the rectangularization effects are not. They need to be quantified to gain greater insight into the nature of the compression of mortality. Our approach to quantification used the standard deviations of the hypothetical

individual lifetimes assumed to generate each survival curve. This was consistent with use of the LE as the mean of the same hypothetical distribution of individual lifetimes. Moreover, the standard deviation of lifetime at or above age 10 was Fries' preferred statistic for quantifying the degree of rectangularization at any point in time. Fries predicted in 1984 that this statistic would decline if life expectancy at age 10 continued to increase, which, in fact, it did.

The ratio of the standard deviation to the mean forms a dimensionless statistic—the coefficient of variation—that summarizes the relative variability of the individual lifetimes compared with the group-specific LE.

Figure 2 displays graphs of the three sets of statistics corresponding to the survival curves in Figure 1. The top panels provide the full set of details for the LEs discussed above. Of note, the LEs at age 10 were higher than at age 0 during 1900–1910 for both sexes, consistent with the very high infant and childhood mortality at that time. The LEs at age 10 increased by 5.6 years for males and 4.6 years for females between 1960 and 2000.

The middle panels display the standard deviations. The standard deviations of lifetime at age 10 and above reached a relative low in 1960 followed by variations within a narrow range thereafter, consistent with the curve-shifting to the right seen in Figure 1 but conflicting with Fries' prediction that they would decrease given that life expectancy at age 10 had increased.

Thus, the near constancy of the standard deviations of lifetime at or above age 10 in the period 1960-2000 indicates that there was no significant compression of mortality in this period. The differences between the standard deviations of lifetime at or above age 0 versus at or above age 10 were wholly attributable to changes in mortality below age 10, reflecting ontogenic rather than senescent effects.

In contrast, the standard deviations of lifetime at age 65 and above increased gradually over the entire set of life tables, indicating that there was increasing variability in the lifetimes in the elderly population. The patterns were similar for males and females.

For males, the terminal values in 2000 were 15.2 years at age 10 and above and 8.3 years at age 65 and above; for females, the corresponding values were 13.8 years and 8.7 years.

The coefficients of variation exhibited patterns consistent with the absence of mortality compression above age 10 for later calendar years. The coefficient of variation was recommended by Natalia Gavrilova to supplement comparisons based on the standard deviation when the LEs were very different, as they were in Figure 2.

Resolving a Fundamental Error

Fries proposed that the natural limit to the process of mortality compression would yield an LE of 85 years with a standard deviation of four years. So what was the fundamental error in his proposal?

Comparison of his four-year standard deviation with the 15.2- and 13.8-year results in Figure 2 for ages 10 and above indicates that a standard deviation of four years cannot plausibly be a limit point of that process.

OK, if not a *plausible* limit point, is it a *possible* limit point?

Absolutely not! And the reason has important implications for our understanding of future mortality.

Tuljapurkar and Edwards developed theoretical lower bounds to the standard deviations of lifetimes, which they used to argue in 2011 that the potential for future rectangularization effects was limited. In making this argument, they showed that the standard deviation of lifetimes was approximately $1/\beta$, where β is the exponential rate of increase in the Gompertz mortality function. Assuming, like Fries, that the mortality rate doubled every eight years, so that $\beta = 0.087$, they estimated a lower-bound standard deviation of 11.5 years, which though well below the standard deviations for lifetimes above age 10 in Figure 2, was 2.9 times larger than Fries' proposed four-year limit.

Moreover, the exact result for the Gompertz standard deviation had been derived by John Pollard and Emil Valkovics in 1992 as $1.28255/\beta$, which yielded, for the above assumptions, a limiting standard deviation of 14.7 years, clearly too high because it exceeded the 13.8-year value for females in Figure 2. An alternative benchmark with $\beta = 0.100$ yielded a limiting standard deviation of 12.8 years, comfortably below the 13.8-year female value yet 3.2 times as large as Fries' proposed four-year limit.

Pollard and Valkovics' refinement increased the lower bound of the standard deviation by 28 percent, further strengthening Tuljapurkar and Edwards' argument that future rectangularization of the mortality survival curve will be limited.

If so, then the primary mechanism for future mortality improvement will be the right-shifting of survival, and this may simplify some mortality forecasting models and eliminate others.

Compression of Morbidity

Long after the publication of his 1980 article, Fries came to realize that his formulation of the process of the compression of morbidity did not actually require a fixed LE upper limit of 85 years with a four-year standard deviation; both statistics could be changed and indeed could continue to change over time if one alternative condition were met. As he wrote in 2005, "For the Compression of Morbidity to occur, increases in the age of onset of chronic infirmity would have to be more rapid than increases in life expectancy."

This condition clearly refers to the average changes in the population;

FIGURE 2: Life Table Functions for Ages 0, 10, and 65, U.S. Males and Females by Calendar Year 1900–2000 (SSA Data)

FIGURE 2A: Life Expectancy at Ages 0, 10, and 65, U.S. Males

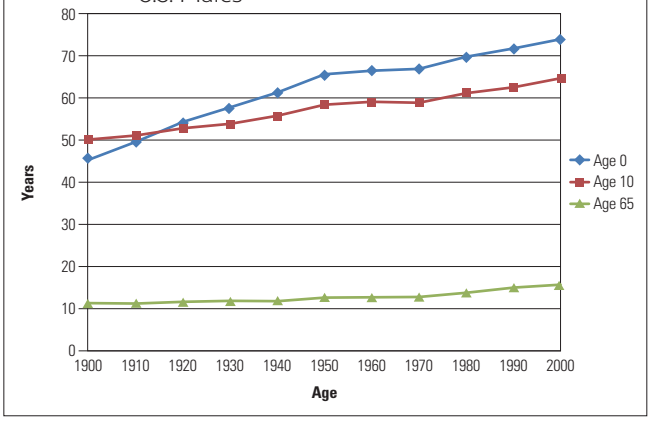


FIGURE 2B: Life Expectancy at Ages 0, 10, and 65, U.S. Females

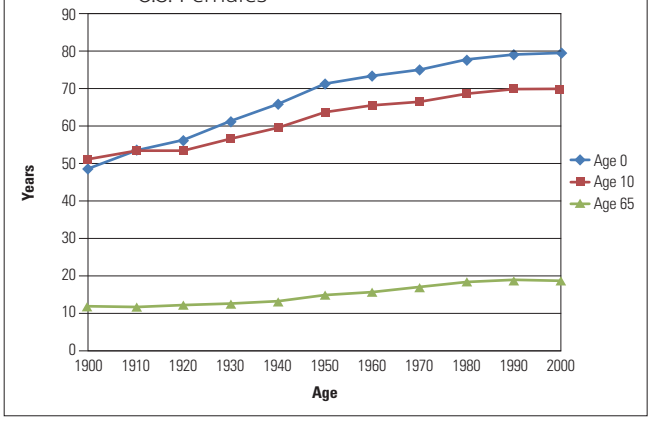


FIGURE 2C: Standard Deviation of Lifetime Above Ages 0, 10, and 65, U.S. Males

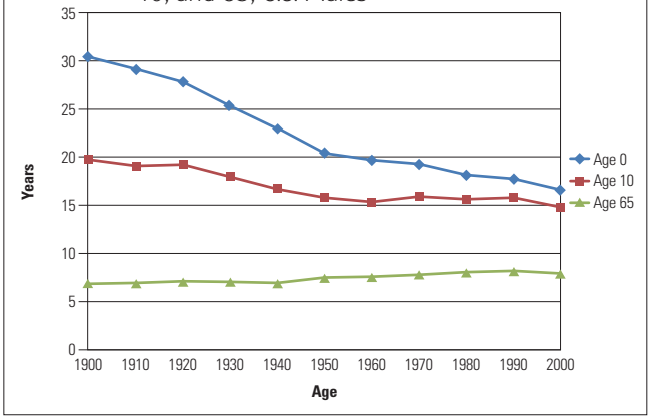


FIGURE 2D: Standard Deviation of Lifetime Above Ages 0, 10, and 65, U.S. Females

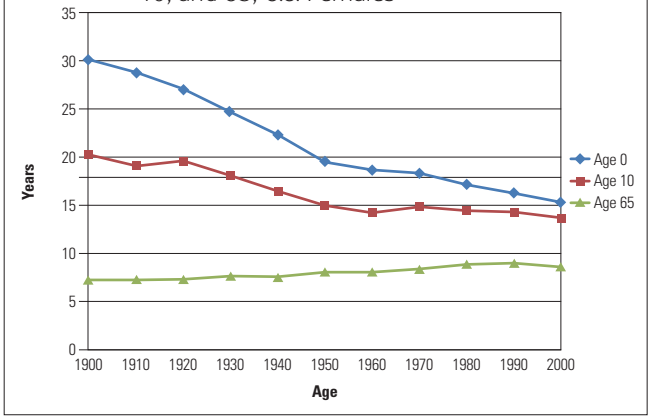


FIGURE 2E: Coefficient of Variation of Lifetime Above Ages 0,10, and 65, U.S. Males

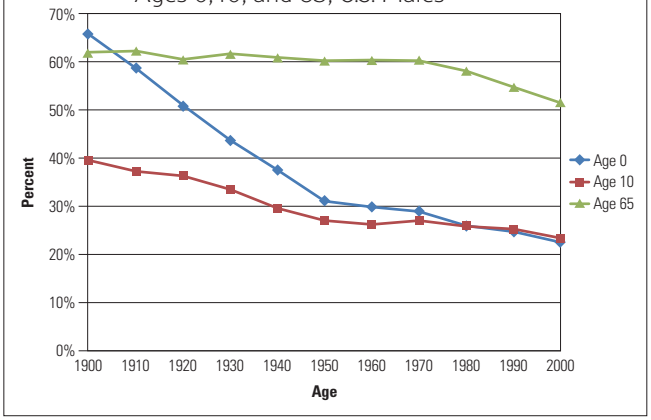
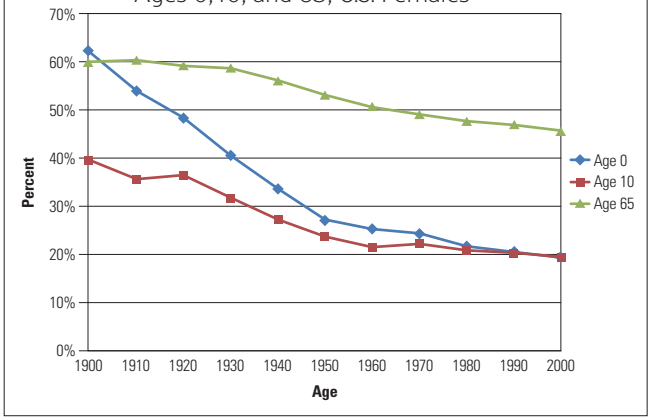


FIGURE 2F: Coefficient of Variation of Lifetime Above Ages 0,10, and 65, U.S. Females



SOURCE: AUTHOR

individuals would continue to exhibit variability in their attained lifetimes and ages of onset of morbidity.

To support his theory, Fries relied, in part, on results derived from the National Long Term Care Survey, which are updated in this section using disability data for the 20-year period 1984–2004, with disability defined by simulating the HIPAA ADL (1996 Health Insurance Portability and Accountability Act activities of daily living) and CI (cognitive impairment) triggers used by long-term care insurers.

Table 1 presents the LEs, disabled life expectancies (DLEs), and decompositions of the DLE changes into the survival increments and morbidity

TABLE 1: Components of Change in Life Expectancy and HIPAA ADL/CI Expectancy (in Years at Age 65), United States, 1984 and 2004

At Age 65	1984	2004	Change	Survival Increment	Morbidity Decrement
Male					
Life Expectancy	14.41	16.67	2.26	2.26	—
HIPAA ADL/CI Expectancy	1.64	1.26	–0.38	0.44	0.82
Female					
Life Expectancy	18.66	19.50	0.84	0.84	—
HIPAA ADL/CI Expectancy	3.26	2.29	–0.97	0.24	1.21

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

decrements. In these calculations, the DLEs are components of the LEs; they are not intended to be added to the LEs. They can be subtracted from the LEs, and if so, the results represent the values of the disability-free life expectancies (DFLEs).

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. The declines in DLEs were 0.38 years for males and 0.97 years for females, which represented, respectively, 23 percent of the 1984 DLE for males and 30 percent of the 1984 DLE for females.

The reductions in DLE constitute our quantification of the compression of morbidity. These reductions were statistically highly significant for both sexes, as were the component survival increments and morbidity decrements. These latter components quantify the interactions between mortality improvement and morbidity compression.

To illustrate, had morbidity remained static—with fixed age-specific prevalence rates—for females during 1984–2004, the DLE would have increased by 0.24 years, from 3.26 to 3.50 years, rather than decreasing to 2.29 years. The 1.21-year difference between the 3.50- and 2.29-year values was reported in the table as the morbidity decrement. Similarly, for males the morbidity decrement was 0.82 years, which more than offset the 0.44-year survival increment.

This offsetting property allows us to restate Fries' 2005 condition with greater precision: For the compression of morbidity to occur, the morbidity decrement must exceed the survival increment.

Figure 3 displays the joint relative survival functions for males and females in 1984 and 2004 based on a disability hierarchy with four levels: no disability, CI only, ADL impairment only, and combined ADL and CI impairments. The disability hierarchy was used to partition the area under each survival curve into four regions—one for each disability level—with the size (area) of each region equal to the corresponding DLE or DFLE. For both sexes, there was a visible reduction in the lifetime years of disability between 1984 and 2004, consistent with the changes shown in Table 1. In addition, the figure clearly displayed much higher levels of disability for females, also consistent with the differences shown in Table 1.

Uncoupling Morbidity and Mortality

The results demonstrate that there was a very substantial and highly statistically significant compression of morbidity for both males and females in the U.S. over the period 1984–2004, based on the assumption that the term "morbidity" could be operationalized using the HIPAA ADL and CI triggering criteria.

It's true that analyses using different definitions of morbidity or focusing on specific subpopulations could lead to different results. For example, Eileen Crimmins and Hirám Beltrán-Sánchez reported in 2011 that the compression of morbidity reversed during 1998–2008 in a study in which morbidity was defined in terms of loss of mobility functioning among the non-institutionalized population. Still, the choice of HIPAA-based measures combined with a data source—the National Long Term Care Survey—that covers all subgroups of the elderly population was more appropriate for the issues in this article.

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.

These results jointly imply that very substantial amounts of morbidity compression occurred during a period in which there was little if any mortality compression. This finding indicates that the two processes are not closely tied: Morbidity compression does not require concurrent mortality compression. □

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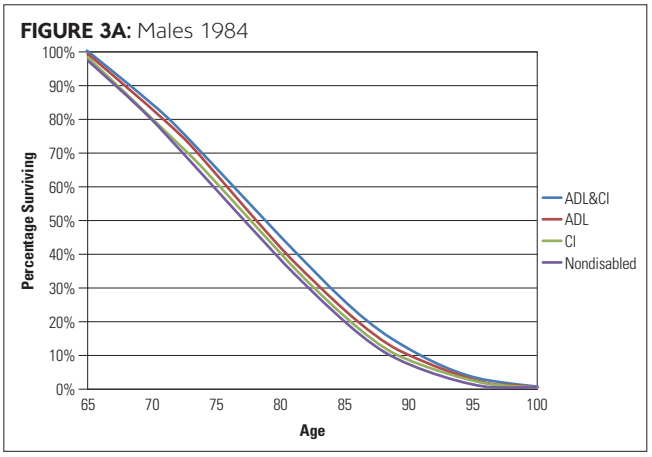
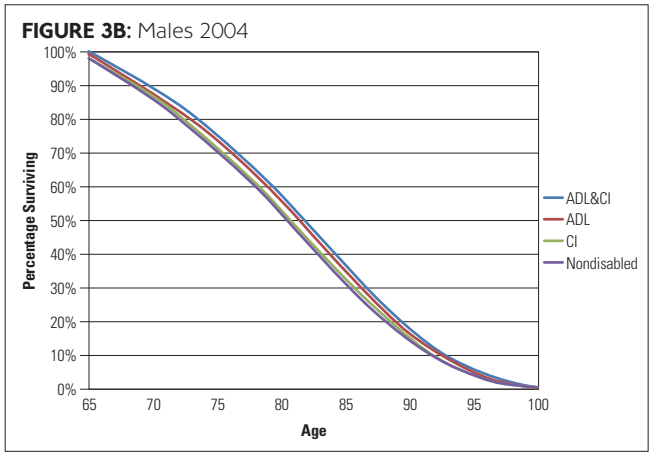
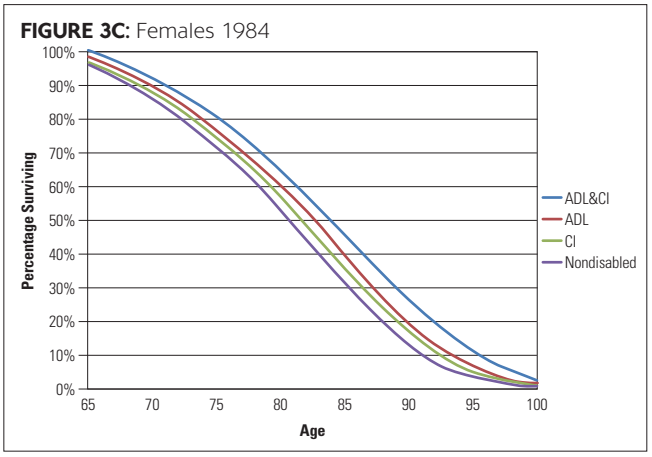
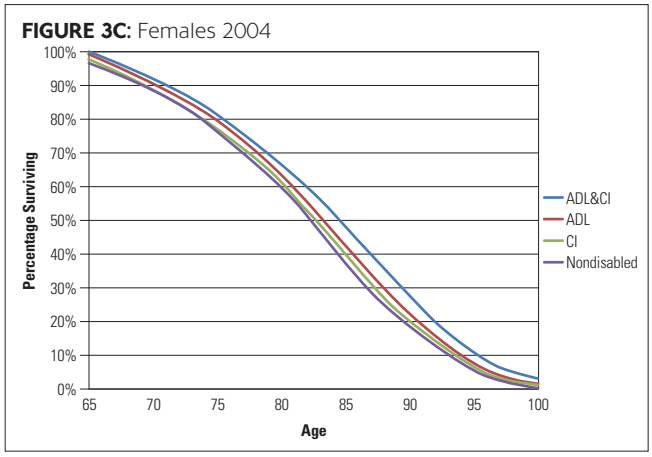
FIGURE 3: Joint Relative Survival at Ages 65+, Meets Either HIPAA Trigger, U.S. Males and Females, 1984 and 2004









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