Published in final edited form as:
Demography. 2012 August ; 49(3): 819–840. doi:10.1007/s13524-012-0104-1.

The Significance of Education for Mortality Compression in the United States*

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Abstract

Recent studies of old-age mortality trends assess whether longevity improvements over time are linked to increasing compression of mortality at advanced ages. The historical backdrop of these studies is the long-term improvements in a population's socioeconomic resources that fueled longevity gains. We extend this line of inquiry by examining whether socioeconomic differences in longevity *within* a population are accompanied by old-age mortality compression. Specifically, we document educational differences in longevity and mortality compression for older men and women in the United States. Drawing on the fundamental cause of disease framework, we hypothesize that both longevity and compression increase with higher levels of education and that women with the highest levels of education will exhibit the greatest degree of longevity and compression. Results based on the Health and Retirement Study and the National Health Interview Survey Linked Mortality File confirm a strong educational gradient in both longevity and mortality compression. We also find that mortality is more compressed within educational groups among women than men. The results suggest that educational attainment in the United States maximizes life chances by delaying the biological aging process.

A growing number of old-age mortality studies examine national-level trends in mortality compression to evaluate changes in the distribution of deaths at very old ages that accompany changes in longevity. A driving question has been whether improvements in longevity have been accompanied by increasing compression of old-age mortality or whether improvements in longevity have led to a "shifting mortality scenario" where longevity improves but the distribution of old-age deaths remains unchanged. *Within a population*, an enormous body of evidence documents that longevity increases with greater educational attainment, yet few studies have examined whether education is also associated with mortality compression. This leaves a number of important questions unresolved. Does more education confer a *maximization* of life chances through both greater longevity *and* compression of old-age mortality, or does more education simply shift the distribution of old-age deaths upwards? If compression is occurring at higher levels of education, to what extent do those with less education differ from those with more education? Given women's

*This research was supported by a research grant from the Eunice Kennedy Shriver National Institute of Child Health and Human Development (1 R01-HD053696, PI Robert A. Hummer) and by infrastructure (5 R24 HD042849) and training (5 T32 HD007081) grants awarded to the Population Research Center at the University of Texas at Austin by the Eunice Kennedy Shriver National Institute of Health and Child Development. The authors would like to extend their thanks to the four anonymous reviewers and members of education-health-mortality working group at the University of Texas at Austin for their helpful suggestions and comments.

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mortality advantage relative to men, do women experience greater mortality compression with at higher levels of education than men?

We evaluate these questions for the United States civilian non-institutionalized population aged 50 years and older. Our analysis is based on two major indicators drawn from recent analyses of mortality compression (Cheung and Robine 2006, 2007; Cheung, et al. 2005; Cheung, Robine, and Caselli 2008; Kannisto 2001) that allow us to characterize the distribution of deaths at older ages for major education-gender groups: the modal age of death (M), an indicator of the typical length of life, and the compression of mortality above the modal age of death (SD(M+)). We hypothesize that both the length of life and compression increase with higher levels of education. Our hypothesis is based on a wealth of evidence documenting education's profound influence on mortality through adult socioeconomic achievement processes, access to and use of health care, stronger social support, greater information seeking, and less risky health behaviors (Elo 2009; Hummer and Lariscy 2011). We extend this hypothesis and argue that at higher levels of education within a population, deaths are increasingly redistributed from younger to older ages such that mortality is compressed into a smaller and later portion of life. Because older American women have a shallower educational gradient in mortality than men (Montez, et al. 2009; Zajacova and Hummer 2009), we expect that the educational gradient in the modal age of death will be smaller for women compared to men. However, we expect highly educated women to exhibit the greatest degree of mortality compression due to the fact that women live longer than men.

Educational Differences In Longevity and Compression

As a fundamental cause of mortality, education shapes a person's exposure to multiple health risks and is linked to a malleable set of material and non-material resources that allow individuals to maximize their potential for a long and healthy life over time and in multiple socio-environmental and socio-epidemiologic contexts (Link 2008; Link and Phelan 1995). This is manifested in a number of ways. First, persons with more education have lower levels of acute and chronic disease, physiological impairment, and psychological distress relative to the less educated (Mirowsky and Ross 2003). Education also is inversely linked to a number of deleterious health behaviors including smoking, excessive alcohol consumption, and physical inactivity (Pampel, Krueger, and Denney 2010; Ross and Mirowsky 2003). Additionally, persons with more education and/or income are among the first to adopt and reap the greatest benefits from life-saving medical technologies (Chang and Lauderdale 2009; Glied and Lleras-Muney 2009; Link and Phelan 1995; Phelan, et al. 2004).

Moreover, research on the “under the skin” processes responsible for the link between socioeconomic status and health highlights the important role played by differential exposure to chronic and acute stressors. The socioeconomically disadvantaged are exposed to higher levels of stress over the life course than the socioeconomically advantaged (Hatch and Dorenwald 2007; Thoits 2010; Turner and Avison 2003). This is important because research consistently shows that prolonged exposure to stress takes a toll on multiple physiological systems linked to cardiovascular, immunological, inflammatory, and metabolic processes (Adler and Stewart 2010; McEwen 1998; McEwen and Stellar 1993; Seeman, et al. 2010; Seeman, et al. 2008). The accumulation of health insults over the life course essentially results in premature aging or “weathering” among socioeconomically disadvantaged populations (Crimmins, Kim, Seeman 2009; Crimmins and Seeman 2004; Geronimus, et al. 2006). Evidence for socioeconomic differences in the biological aging process is implied by studies reporting lower levels of age-specific biological risk (Crimmins, et al. 2009; Geronimus, et al. 2006) and a later onset of chronic diseases

(Crimmins, Hayward, and Seeman 2004; Hayward, et al. 2000) among socioeconomically advantaged groups. Socioeconomic differentials in the biological aging process ultimately are manifested in differential longevity. By extension, because persons with higher levels of education essentially postpone mortality until later ages, we should observe mortality compression accompanying longevity at higher levels of education.

Most research on mortality compression examines variability within or between nations over time under the common, but tacit, assumption that cross-national differentials reflect socio-environmental and technological disparities intimately linked to socioeconomic development. As the epidemiologic transition took hold, technological and socioeconomic advances allowed humans to become increasingly adept at controlling their environment, and this improved the robustness of populations and, ultimately, increased longevity (Fogel 2004; Fogel and Costa 1997). In the United States, these changes ultimately caused the survival curve to appear increasingly rectangular over the course of the 20th century as deaths were redistributed from younger to older ages and morbidity and mortality were compressed into a smaller and later portion of life (Fries 1980, 1983).

Fogel and Costa (1997) referred to this symbiosis of technology, socio-environmental conditions, and biological processes as technophysio evolution to distinguish this uniquely *socio-biological* form of human evolution from evolutionary processes based in the human genome. Undoubtedly, the diffusion of mass education within populations played a prominent role in the technophysio evolution as well as other processes linked to longevity extension, because the individual and institutional factors linked to the spread of education increased the social capacity for population health (Easterlin 1998; Hayward, Crimmins, and Zhang 2006; Hidajat, Hayward and Saito 2007). Through education, individuals acquire the skills and resources necessary to gain a greater degree of control over their environment and maximize their potential for a longer, healthier life via the adoption of healthier lifestyles and reduced exposure to a wide range of socioenvironmental health risks. Moreover, this increased ability of individuals to control their lives was reinforced by the healthcare system's expanded capability to meet the health needs of populations (Hidajat, et al., 2007). In time, the combined effects of these individual and institutional factors proved instrumental in allowing humans to extend the length of their lives.

Though the long-term trend in most developed nations is toward increased mortality compression, substantial differentials in the variability of lifetimes exist across low mortality countries (Canudas-Romo 2008; Edwards and Tuljapurkar 2005; Smits and Monden 2009; Wilmoth and Horiuchi 1999). Indeed, prior research shows that mortality compression is not an inevitable consequence of increased longevity. Some nations (Canudas-Romo 2008; Edwards and Tuljapurkar 2005) and/or subpopulations within nations (Lynch and Brown 2001; Lynch, Brown, and Harmsen 2003) have gone through periods of mortality decompression. These periods, however, are usually short-lived. In some low mortality countries, the long-term trend toward greater compression has slowed considerably in recent years (Wilmoth and Horiuchi 1999) and begun to give way to a shifting mortality scenario where longevity continues to increase without an appreciable change in the distribution of deaths across the age-range (Bongaarts 2005; Bongaarts and Feeney 2002, 2003; Canudas-Romo 2008; Cheung and Robine 2007; Cheung, et al. 2008; Thatcher, et al. 2010). This suggests, therefore, that health inputs such as increased education potentially could shift the entire distribution of lifetimes upward without redistributing deaths from younger to older ages as levels of education increase. This scenario seems more feasible in describing population mortality changes over time to the extent that health inputs are population wide (e.g., population-wide changes in health care and health care access). However, *within* a population, the unequal distribution of resources allows the advantaged group to garner health advantages compared to less advantaged groups, and it is these resources that should

lead to the redistribution of deaths from younger to older ages among the advantaged group. Consequently, monitoring differences in variability in the age of death provides important insights into population health disparities (Engelman, Canudas-Romo, and Agree 2010).

Despite an extensive literature documenting an unequivocal link between socioeconomic status and mortality, only a handful of studies directly examine differentials in mortality compression across socioeconomically advantaged and disadvantaged sub-populations (Edwards and Tuljapurkar 2005; Go, et al. 1995; Lynch, et al. 2003; Shkolnikov, Andreev, and Begun 2003; Van Raalte, Kunst, and Mackenbach 2009). Three studies examined race-ethnic differentials in mortality compression in the United States, where these differentials are viewed as an indicator of socioeconomic inequality. Go and colleagues (1995) examined race-ethnic specific California vital statistics data for the years 1970, 1980, and 1990 respectively. They documented disparate trends across race-gender groups, and periods. However *within* each period, mortality was substantially more compressed among whites (e.g., the most socioeconomically advantaged population) than other racial-ethnic groups. Moreover, within race-ethnic groups and periods, mortality generally was more compressed for women than men. In an analysis of black and white deaths drawn from U.S. vital statistics data between 1970 and 1992, Lynch, et al. (2003) documented an overall trend toward increased compression among blacks and some evidence of decompression among whites. However, after adjusting for data quality, blacks displayed consistently higher levels of dispersion around the age at death relative to whites at any given point in time. In an analysis of U.S. vital statistics data from the late 1960s to the early 1990s, Edwards and Tuljapurkar (2005) found that blacks experienced considerably more variability in the age of death (e.g., less compression) than whites.

To our knowledge, only three studies have directly examined income and educational differences in mortality compression and only one of these focuses on the United States. In an analysis of Russian men ages 20 to 64 in 1979 and 1989 using the Gini Coefficient to measure variability around the average age at death across educational groups, Shkolnikov and colleagues (2003) found that higher levels of education were associated with a greater degree of mortality compression within each respective year. More recently, Edwards and Tuljapurkar (2005) examined socioeconomic differentials in mortality compression by constructing life tables among education and income groups using data from the U.S. National Longitudinal Mortality Study (NLMS) for the 1980s. In analyses not disaggregated by gender, Edwards and Tuljapurkar (2005), similar to Shkolnikov, et al. (2003), found that mortality was more compressed at higher levels of education and income than at lower levels of education and income. Finally, a recent study examined mortality compression across educational groups in eleven European countries (Van Raalte, et al. 2009). In all of the countries examined, the authors found that mortality was more compressed among women than men and among the highly educated relative to those with less education. Interestingly, the study also found that highly educated groups displayed similar levels of compression across countries, but educational gradients in both life expectancy and mortality compression within countries were more pronounced in Eastern Europe than in Western Europe. The presence of an East-West divide might imply that low levels of overall socioeconomic inequality coupled with a highly developed social welfare system reduces educational inequalities in both longevity and mortality compression. Consistent with the fundamental cause perspective, the shallower educational gradient in longevity and mortality compression in Western Europe likely is due to the availability and use of various institutional, social, economic, and behavioral resources that work to promote health and longevity.

Taken together, the evidence – albeit limited – clearly suggests that at any given point in time, old-age mortality is more compressed among the socioeconomically advantaged than

the socioeconomically disadvantaged. In accord with the notion that socioeconomic conditions are a fundamental cause of mortality, this implies that the biological processes governing the human lifespan are conditional on an array of socioenvironmental factors.

Measuring Longevity and Mortality Compression

Mortality patterns and differentials are most often measured using life expectancy at birth. However, recent research suggests that life expectancy can sometimes provide an incomplete picture of older-age mortality dynamics (Canudas-Romo 2010; Kannisto 2001). Life expectancy changes in response to mortality reductions at all ages, but is particularly sensitive to mortality reductions at younger ages (Canudas-Romo 2010; Cheung and Robine 2007; Kannisto 2001). For example, life expectancy increased precipitously in the early stages of the epidemiologic transition as a direct result of sharp reductions in infant and child mortality, but rose modestly in the later stages of the transition as mortality reductions at younger ages stagnated and reductions in older age mortality began to accelerate (Canudas-Romo 2010; Wilmoth 2000).

Moreover, life expectancy is a poor index of variability the average age of death (Engleman, et al. 2010). Longevity and variability are two conceptually distinct and equally important components of mortality measurement. However, as the mean of a skewed distribution, life expectancy essentially conflates longevity and variability in the distribution of lifetimes. In recognition of the fact that life expectancy provides an incomplete picture of older-age mortality dynamics, recent studies have begun to incorporate alternative measures of longevity coupled with explicit measures of variability (Canudas-Romo 2008, 2010; Cheung, et al. 2005; Cheung and Robine 2006, 2008; Kannisto 1996, 2001). Though a number of alternative measures have been proposed (see Cheung, et al. 2005 for an overview), the most prominent are the late-life modal age of death and the standard deviation above the modal age at death.

The modal age at death uniquely characterizes the typical lifespan of a given species conditional on current conditions (Kannisto 2001; Lexis 1878). To illustrate this idea, Lexis (1878) classified life table deaths (e.g., the d(x) series) into three groups based on their relationship to the biological aging process (see Figure 1).[1] The first group included deaths in infancy and early childhood. According to Lexis, these deaths are the result of exogenous factors and/or congenital abnormalities completely unrelated to the aging process. The second group contains “premature” deaths in late childhood up to middle adulthood. Lexis attributed deaths in this group to a combination of exogenous factors (i.e., violence, accidents, infectious agents, chance, etc.) and early-onset forms of disease typically encountered at older ages. At older ages, premature deaths give way to a third group of “normal” deaths distributed symmetrically around the late-life mode. The deaths in this group were considered normal because the vast majority were a direct result of the age-related deterioration of biological processes (e.g., senescence).[2] Kannisto (2001) built upon Lexis's ideas and introduced the standard deviation above the mode (SD(M+)) to measure variability in the age of death. Deaths at and above the mode primarily reflect senescent processes. Therefore, substantial differences in variability above the mode suggest that populations differ in the ability to maximize their potential for a long and healthy life.

In this paper, we incorporate the conceptual and methodological insights found in the mortality compression literature to a gain a more comprehensive understanding of

[1]Véron and Rohrbasser (2003) provide a detailed account of the approach taken by Lexis (1878).
[2]Lexis maintained that premature and normal deaths are etiologically distinct, but recognized that, in practice, it is difficult to distinguish between premature and normal deaths in the transitional region of the curve.

education-mortality disparities in the United States. Accordingly, we make several important contributions to the education-mortality literature. To our knowledge, this is the first study to examine educational differences in the modal age at death in the United States. Given that the mode primarily measures senescent deaths (Lexis 1878; Kannisto 2001; Canudas-Romo 2008), the analyses provide unique insights into the pivotal role played by education in shaping the biological aging process. This is important because several studies suggest that the mode is a better indicator of longevity than life expectancy in mortality regimes where longevity is a function primarily of mortality reductions at older ages (Canudas-Romo 2010; Cheung and Robine 2007; Kannisto 2001). Additionally, the only other study focusing on educational differences in mortality compression in the United States examined data from the 1980s, only compared persons without a high school education to high school graduates, and did not disaggregate by gender (Edwards and Tuljapurkar 2005). Thus, our analyses extend Edwards and Tuljapurkar (2005) in several important ways by examining data from multiple data sources through 2006, comparing three education groups, and evaluating gender-education differentials in longevity and mortality compression in the United States. Importantly, to our knowledge, our study is the first to examine gender-education differentials in mortality compression in the United States. Finally, studies on mortality compression very rarely evaluate differences across groups via formal tests of statistical significance (exceptions include Lynch and Brown [2001] and Lynch et al. [2003]), and our analyses are the first to examine whether there are statistically significant gender-education differentials in mortality compression using data from the United States. The dearth of research on educational differentials in mortality compression represents a significant gap in our understanding of how basic socio-demographic processes influence old age mortality.

Methods

Data

We draw on two nationally representative data sources. The first is the Health and Retirement Study (HRS) for the years 1992-2006 linked to the National Death Index (NDI). The HRS is a longitudinal, household survey representative of the U.S. civilian, non-institutionalized population ages 51 and above and their spouses (HRS 2008). The second data set is the public- use National Health Interview Survey Linked Mortality Files (NHIS-LMF) which links the 1986-2000 NHIS surveys to the 1986-2002 NDI. The NHIS is a cross sectional, household survey representative of the U.S. civilian, non-institutionalized adult population ages 18 and over conducted annually by the U.S. National Center for Health Statistics (NCHS). The NDI is a collection of state death records maintained by NCHS. Survey data from the HRS and NHIS are probabilistically linked to records in the NDI (for additional information on the NHIS-LMF see Lochner, et al. [2008]; for additional information on the HRS see Servais [2008]).

We imposed several restrictions on the HRS and NHIS-LMF data (see Table A1 in the Appendix for an overview). First, all of the analyses were restricted to native-born non-Hispanic white respondents ages 50 and older with complete information on education and valid sample weights. We imposed this restriction to improve data quality and to reduce population heterogeneity[3]. In the NHIS-LMF, we restrict our analyses to the 1989-1996 NHIS survey years, because information on nativity was not collected prior to 1989 and because the top-coding of age at 85+ years in the 1997 survey and beyond makes the data set less useful for the analysis of old age mortality patterns. We also limit our analyses in the NHIS-LMF to respondents ages 50 to 89 at interview because preliminary analyses

[3]We also ran analyses that included all race-ethnic groups (available on request). Although our substantive conclusions remain unchanged, in the models including all race-ethnic groups, the modal ages of death were 0.09 - 0.81 years lower and the standard deviations above the mode were 0.08 - 0.34 higher than those presented in Table 3.

(available on request) suggested that the quality of the NDI matches in the NHIS-LMF declines among respondents aged 90 and above; this is true particularly for females. These conclusions are consistent with the results of a recent report by NCHS comparing mortality estimates from the NHIS-LMF and the U.S. Vital Statistics data (Ingram, Lochner, and Cox 2008).

Table 1 compares estimates for e_{65} from three nationally representative data sets - the HRS, NHIS-LMF, and the National Longitudinal Mortality Survey (NLMS; Lin, Rogot, Johnson, Sorlie, and Arias 2003) - with estimates from U.S. vital statistics data. The results indicate that estimates for life expectancy at age 65 *(e_{65})* generated from the HRS, NHIS-LMF, and NLMS are around one year higher than those based on the U.S. vital statistics data and that these differences are statistically significant ($p \leq 0.05$). This is consistent with the fact that the three survey-based data sets exclude institutionalized respondents. Moreover, the results in Table 1 also indicate equivalent levels of divergence between the survey estimates and vital statistics data. Notably, for women, the HRS and NHIS-LMF estimates actually are closer to the vital statistics estimates than the NLMS; thus, we use the HRS and NHIS-LMF rather than the NLMS. The HRS and NHIS-LMF data files are also much more current than the NLMS. We utilize the HRS and NHIS-LMF instead of U.S. vital statistics data because education is often misreported on U.S. death certificates (Rostron 2010). The final analytic sample sizes are 20,909 in the HRS and 158,322 in the NHIS-LMF, respectively. The HRS contains 5,935 deaths (n = 2,942 for men, n = 2,993 for women). The NHIS-LMF contains 44,330 deaths (n = 22,207 for men, n = 22,123 for women). Table 2 shows the number of deaths and person-years of exposure by education and gender in the HRS and NHIS-LMF.

Measures

The dependent variable is all-cause mortality. HRS deaths were identified via the Tracker file and/or the NDI. If either file indicated that a respondent was deceased, we coded the person as dead. In the NHIS-LMF, deaths were identified solely from the probabilistic linkage to the NDI. Although relying on the NDI to identify decedents in the NHIS-LMF results in slightly lower mortality rates than should be the case, the shape of the distribution of deaths in the NHIS-LMF is not appreciably altered. Exposure to the risk of death is measured in calendar years and deaths are assumed to occur in the middle of the year. Persons surviving an interval were exposed to the risk of death for one full year, while persons who died in an interval were exposed to the risk of death for only half a year.

The independent variables in our analyses are age, gender, and education. Age is our only time-varying variable and is measured on January 1st of each year. Exact age in each year was obtained by subtracting the interview date from the self-reported date of birth. Persons missing month of birth were assigned to June (HRS: n = 32, NHIS-LMF: n = 28). Missing year of birth in the NHIS-LMF (n=28) was imputed by subtracting self-reported age from the interview year. In the HRS, decedents missing information on date of death in the Tracker file who did not have an NDI death record were assigned the median date of death in the interview interval in which they died (n = 209). In order to approximate a central death rate, we defined exact age on January 1st of each calendar year as age x – 0.5 to age x + 0.49. Gender was dichotomized. Education in the HRS and NHIS-LMF is self-reported and recorded in years of completed formal schooling. In preliminary analyses, we examined the functional form of the relationship between education (in years) and the risk of death by gender. The results (available on request) indicated that disaggregating the 13+ years of education category into categories for 13-15 and 16+ years marginally improved the fit for men, but not women. Thus, we trichotomized educational attainment into the following categories: 0-11 years, 12 years, and 13+ years, which largely is consistent with the best fitting form recently identified by Montez, Hummer, and Hayward (2011). In the United

States, these education categories roughly correspond to less than a high school education, high school graduate, and some college education or higher.

Educational attainment is the most commonly used indicator of socioeconomic status (SES) in studies of adult mortality (Elo 2009; Hummer and Lariscy 2011), and there are several reasons why we employ education as our measure of SES. First, educational attainment is a key determinant of a person's position in the stratification system because it is usually fixed in early adulthood and is a strong predictor of other dimensions of SES such as income and occupation. Second, educational attainment is also less prone to issues of health endogeneity than are income and occupation. Third, education also has the added advantage of being a proxy for human, social, and cultural capital. These are important determinants of health from the fundamental cause perspective, but difficult to measure in population-based research. Finally, missing data is much less of an issue with educational attainment in comparison to income and occupation.

Methodology

Our first task was to derive a set of gender-education specific life tables using a multivariate life table approach (Teachman and Hayward 1993). To do this, we reformatted the data into person-year files with each interval corresponding to a calendar year (i.e., January 1st to December 31st) and estimated a set of gender-education specific Gompertz models that regressed the risk of death on age on January 1st.[4] To minimize the amount of structure we imposed on the associations between age, gender, and education, we estimated six gender-education specific models. The models are as follows:

$$\ln \mathrm{m_m}(x) = \beta_{\mathrm{m}0} + \beta_{m1}\mathrm{AGE}_x \quad (1)$$

$$\ln m_{\mathrm{w}}(x) = \beta_{\mathrm{w}0} + \beta_{\mathrm{w}1}\mathrm{AGE}_x \quad (2)$$

where,

$$m(x) = \lim_{n\to 0}\frac{P(x, x+n)}{n}.$$

The first model is for men and the second model is for women. The models regress exact age at January 1st in a given year on the risk of death. The models were estimated separately for each of the educational groups. The parameter estimates from the gender-education specific Gompertz models were used to solve the regression equations and calculate age-specific mortality rates for each education-gender group (Teachman and Hayward 1993). The rates are analogous to exponentially smoothed occurrence-exposure rates. The life tables begin at exact age 50 and are closed at age 100+. The HRS and NHIS-LMF were weighted to represent the U.S. non-institutionalized, civilian population and the weights were normalized so that the sum of the sample weights equals the number of observations. The models also account for the complexity of the HRS and NHIS-LMF sampling designs.

[4]We also estimated a series of Logit models to examine the sensitivity of our results to the model specification. The results from the Logit and Gompertz models were virtually identical and did not alter our substantive conclusions. For example, depending on the dataset and gender, the Logit model produced modes that were 0.15 - 0.36 years higher and standard deviations above the mode that were 0.07 – 0.18 years lower than those shown in Table 3.

A microsimulation approach was used to construct our life tables. We evaluated possible mortality for each member of a life table cohort, e.g., in this study 700,000 women at age 50 with less than a high school education, by comparing a random number from a uniform distribution for the transition rates for the 50-51 age interval. For each person surviving to age 51, we generate a new random number from the uniform distribution to compare with the transition probabilities for the age 51-52 interval. This process is repeated one year at a time for each person until his or her death. Once this process is repeated for all members of the cohort, we have the simulated life times for the cohort from which the life table functions can be easily calculated by averaging over the individual life times. For example, life expectancy is computed by the average number of years lived for the simulated cohort. In this study, we used a 700,000-person cohort as opposed to a standard life table cohort of 100,000 in order to obtain stable estimates for the modal age at death and the standard deviation above the modal age at death. For more information on the microsimulation approach we employ, refer to Cai, et al. (2010).

After constructing the life tables, we measured mortality compression following the mathematical approach outlined by Kannisto (2001) based on the exact modal age of death (M) instead of life expectancy as the measure of longevity. M is obtained via interpolation and calculated as (Kannisto, 2001, p. 163):

$$\mathrm{M}=x+\frac{d(x)-d(x-1)}{[d(x)-d(x-1)]+[d(x)-d(x+1)]}, \quad (3)$$

where x is the age corresponding to the largest value in the life table decrement function, $d(x)$ is the number of life table deaths at x, $d(x-1)$ is $d(x)$ at x - 1, and $d(x+1)$ is $d(x)$ at x + 1.

According to Kannisto (2001), the mode – unlike life expectancy – is not subject to bias when the age range being examined is truncated (see Robine [2001] for an empirical illustration of the problems posed by truncation). Using the mode is an important advantage in our analyses because our life tables begin at exact age 50. Although not discussed in great detail, we also present the number of life table deaths at M (e.g., d(M)). Following Kannisto (2001), we measure compression via the standard deviation above the modal age of death (e.g., SD(M+)). Assuming that deaths are distributed uniformly within a given age interval, the formula for the standard deviation of individual lifetimes above the modal age of death is

$$SD(M+)=\sqrt{\frac{\sum_{i=1}^{n}(x_i-\bar{x})^2}{n}}, \quad (4)$$

where the numerator represents the sum of the squared positive deviations from the modal age of death and the denominator represents the number of age intervals above the mode (Cheung, et al., 2005, p. 254). Smaller values for SD(M+) indicate greater levels of mortality compression. Importantly, since the survival curve takes on an increasingly rectangular appearance as life expectancy – or in our case the modal age at death – increases alongside decreased variability in the modal age at death, evaluating the results for M and SD(M+) in conjunction with one another also allows us to assess the degree to which the survival curve is more or less rectangular between educational groups. Similar to Cheung, et al. (2005), we follow Eakin and Witten's (1995) suggestion to normalize age and the probability of survival. [5] For age, this was accomplished simply by dividing each exact age x by the exact modal age at death (M). Normalization was carried out for the probability of

survival by dividing l_x by l_0 (e.g., 700,000), which rescaled the probability of survival to range from zero to one.

Finally, an important aspect of this paper is our use of an innovative rescaling bootstrap approach to obtain standard errors for the compression parameters (for details, see Cai et al. 2010). This procedure generates repeated estimates of the life table functions and compression parameters by randomly drawing a series of bootstrap samples (n = 300) from our analytic sample. From these bootstrap samples, we generate distributions of the compression parameters which allow us to estimate sampling variability. We then combined this information with the original estimates to construct confidence intervals and conduct significance tests across gender-education groups.

Results

Figures 2-5 clearly demonstrate that education fundamentally alters the shape of the d_x and l_x curves. For women in the HRS, education is associated with a positive shift in the distribution of lifetimes as the number of deaths at the mode increases and the amount of variability around the mode decreases. For example, in Figure 2, there are significantly fewer survivors at the mode among women with 0-11 years of education (d(M) = 23,585, 95% CI = 22,410 - 24,761) than there are among women with 13+ years of education (d(M) = 28,833, 95% CI = 27,173 -30,494). This is because low educated women are much more likely to die before the modal age at death in the "premature" region of the d_x curve. Moreover, this is also why the survival curves for women in the HRS become increasingly rectangular at higher levels of education (see Figure 4). As shown Figures 3 and 5, the same general patterns hold for men. However, the educational gradients in longevity and mortality compression are shallower for men than women. For example, despite the fact that the differences are statistically significant, relatively few life table deaths at the mode separate the least (d(M) = 23,021, 95% CI = 21,673 – 24,369) and most (d(M) = 24,845, 95% CI = 23,453 – 26,237) educated men in the HRS (Figure 3). Consequently, Figure 5 indicates that the survival curves for men in the HRS are much less rectangular at each educational level relative to those for women (Figure 4). These patterns emerge because deaths are dispersed more widely at all levels of education among men relative to women; this is particularly the case at higher levels of education. A large proportion of men in every educational group die prior to the mode, which leaves fewer survivors at the modal age of death. Finally, as we discuss in greater detail below, one of the most interesting results drawn from Figures 2 – 5 is that mortality patterns for the least educated women share striking similarities to the mortality patterns for the most educated men.

In sum, higher levels of education are associated with a strong positive shift in d_x and the modal age of death, considerably less variability around the mode, and thus an increasingly rectangular survival curve. These differences in the distribution of deaths and survivorship are particularly pronounced among women. Highly educated women clearly have the highest modal age of death and the least variability around the mode compared to all other gender-education groups. The results are consistent with our hypotheses that education is associated with greater longevity and mortality compression, and that mortality compression is more pronounced for women than men.

Table 3 contains the values for the modal ages of death (M), number of life table deaths at the mode (d(M)), and the standard deviation above the modal age at death (SD(M+)). We

[5]Eakin and Witten (1995) recommend this to better facilitate interpretations over time and between different populations. Given that the current analyses are cross-sectional, this is technically not necessary. We do it nonetheless in the event that other researchers would like to compare their results to those presented herein. The results are interpreted the same whether or not this is performed.

also show the 95% confidence intervals for these indicators. The left side of Table 3 displays the results from the HRS and the right side displays the results for the NHIS-LMF. Although a small number of differences in statistical significance exist, the results from the HRS and NHIS-LMF are strikingly similar. This is notable particularly given the differences in data quality and the years covered by each respective dataset. Given the similarities in the results across data sources, we focus our discussion on the HRS because it approximates the U.S. Vital Statistics data more closely and covers a more recent period than the NHIS-LMF.

The results for the modal age at death show that women with 11 or fewer years of education in the HRS (M = 85.28, 95% CI = 83.85-86.71) have a mode over five years lower than women with 13 or more years of education (M = 90.24, 95% CI = 89.32-91.16). Likewise, the mode is about five years lower for the least educated men compared to the most educated men (M = 80.84, 95% CI = 78.95-82.73 vs. M = 85.92, 95% CI = 84.63-87.20). The confidence intervals demonstrate that the mode is significantly lower for the least educated women compared to women with a high school or college education. Among men in the HRS, the mode for those with 0-11 years of education differs significantly from men with 13 or more years of education; in the NHIS-LMF the difference between men with 0-11 years of education and men with 12 years of education is also statistically significant.

Note also that in both the HRS and NHIS-LMF, the modal age at death is higher for women than men for all education groups. However, the mode for women with the lowest education does not differ statistically from the mode for men with the highest education. Scanning across all of the education-gender groups, the mode ranges from a low of 80.84 years for men with 0-11 years of education to 90.94 years for women with 13 or more years of education – a difference of about 10 years.

The results in Table 3 also quantify mortality compression. The level of compression significantly differs between those with 0-11 and 13 years of education for both men (SD(M+) = 8.19, 95% CI = 7.32-9.05 vs. SD(M+) = 6.58, 95% CI = 5.98-7.17) and women (SD(M+) = 6.95, 95% CI = 6.31-7.58 vs. SD(M+) = 4.77, 95% CI = 4.34-5.20). The confidence intervals for SD(M+) overlap slightly for those with 12 years of education and 13 or more years of education in the HRS. As such, the differences between these two groups are not statistically significant ($p \geq 0.05$). However, upon further examination, we did find evidence for marginal significance ($p \leq 0.10$). In the HRS, mortality is significantly more compressed among women with 12 years of education compared to women with 0-11 years of education. Mortality compression is not significantly different between men with 12 and 0-11 years of education in the HRS, but it is significantly more compressed between men with 12 (SD(M+) = 7.36, 95% CI = 6.81-7.91) and 0-11 (SD(M+) = 8.69, 95% CI = 7.98-9.40) years of education in the NHIS-LMF. With the larger number of deaths in the NHIS-LMF, the two highest education groups differ significantly in their degree of compression above the mode.

As was the case with the modal age at death, important gender differentials in compression are also evident. As hypothesized, deaths above the mode generally are more compressed for women than men ($p \leq 0.05$) within each respective educational group. The only exception to this pattern is found among the lowest educated groups in the HRS, where levels of compression between men and women are not significantly different. However, as with the modal age at death, gains in compression among women appear to plateau at higher levels of education. In the HRS, these differences are statistically significant for persons with 12 and 13 or more years of education, but not for those with 0-11 years of education. In the NHIS-LMF, however, statistically significant gender differentials in compression are present when comparing all levels of education.

Conclusion

Numerous studies have examined national-level trends in old-age mortality compression, but considerably less attention has been paid to socioeconomic differentials in compression. Indeed, despite a vast literature linking education and mortality (see Hummer and Lariscy 2011 for a review), we are aware of only three studies (e.g., Edwards and Tuljapurkar 2005; Shkolnikov, et al. 2003; Van Raalte, et al. 2009) that examine educational differentials in mortality compression. Unlike the present paper, most prior studies do not systematically address gender-education differentials in compression and, to our knowledge, no prior studies of educational differences in compression conduct formal tests for statistical significance. Overall, our results support the hypothesis that mortality compression increases with education. The modal age at death is higher, mortality is more compressed, and – as a direct result of these two factors – the survival curve is more rectangular among the highly educated relative to the less educated. Given that differences in the modal age at death and compression above the mode primarily capture senescent deaths, the results imply that highly educated populations are able to use their considerable resources to maximize their life chances under current conditions and, ultimately, delay the biological aging process.

The results also point to important gender differences in the association between education and mortality compression. Surprisingly, gender differentials in mortality compression are often overlooked, even in national-level studies (Lynch and Brown 2001). Consistent with our hypothesis, we found that mortality is more compressed among women than men within educational groups. In light of the fact that women also have significantly higher modal ages at death, the compression results suggest that the survival curve is more rectangular for women than men at any given level of education. Men display relatively linear gains in the modal age at death and compression above the modal age at death with education, but gains among women appear to plateau at higher levels of education. This could signal that highly educated women in the United States are encountering some resistance against future gains in longevity and mortality compression because they are reaping the maximum benefits from their education under current conditions. Alternatively, this could also suggest that highly educated women currently are in a transitional phase in which they are moving from an era of mortality compression to an era of shifting mortality. However, given that our data are cross-sectional, it is difficult to know if this is actually the case. Future research should explore this possibility. Furthermore, the similarities between the least educated women and most educated men are remarkable. The modal age of death and standard deviation above the modal age at death are virtually identical between these two groups. The d(x) curves for the groups also share many similarities. The reasons for this likely are due to a complex set of biological, socio-environmental, and socio-behavioral factors. In particular, this may be due, at least in part, to education and gender differences in smoking histories among these cohorts (Denney, et al. 2010; Preston and Wang 2006). Future research should explore the role played by cohort smoking patterns.

Although these findings advance our understanding of the social distribution of mortality compression, the analyses have several limitations. First, beginning around age 80, our death rates (m_x) are consistently lower than the rates found in the U.S. Vital Statistics. This problem is amplified somewhat in the NHIS-LMF due to its sole reliance on probabilistic linkage to the NDI, which fails to capture some deaths due to missing information on the criteria used for matching (see Ingram et al. [2008] for additional information). Another important factor contributing to lower deaths rates at the older ages in the HRS and NHIS-LMF is that the HRS and NHIS-LMF sampling frames exclude institutionalized populations at baseline. Second, there are relatively few deaths at the oldest ages, particularly in the HRS. This is one of the primary reasons we chose to supplement the HRS data with the NHIS-LMF. Third, as previously stated, our trichotomized measure of education is not

definitive. We examined a few alternative functional forms of the relationship between education (in years) and the risk of death for males and females and found that a three-category specification of education provided the best overall fit among these cohorts. This is fairly consistent with the best fitting form indentified in a recent analysis by Montez, et al. (forthcoming). Nonetheless, alternative specifications should be explored in greater detail, especially when new birth cohorts enter old age with higher and higher levels of education.

Prior studies demonstrate that education has a profound effect on adult mortality that is evident even at different stages of economic development and the institutionalization of health care systems (Elo 2009). Here, we have shown that education is also associated with mortality compression, and in the case of women, points to the maximization of life chances among the highly educated in the present historical era. This pattern raises a number of questions with regard to historical as well as future trends in the association between education and mortality compression. For example, would we find a similar educational gradient in compression in earlier periods even though the modal ages of death were lower? If socially advantaged persons are better able to maximize their health subject to historical conditions as the fundamental causes of disease perspective maintains (Link and Phelan 1995), then this may well be the case. Under this scenario, longevity in the population as a whole would change significantly over time while educational inequality in compression would persist and perhaps even grow. Will we see future increases in compression as modal ages are expected to rise and education increases? Our study focuses on a single period of time and we have no way to gauge what levels of mortality compression are possible with improved mortality and greater levels of educational attainment. Given the case of Japan and several other nations, however, where modal ages of death are rising in a highly educated population but compression has remained relatively constant, we might well see a shifting mortality scenario whereby improvements in the modal ages of death among highly educated persons are accompanied by a concomitant shift in the distribution of lifetimes about the mode.

Our findings also highlight the enormity of the gender gap in compression. Education is clearly important for men in terms of increased modal ages of death and compression. However, the levels of compression among the most highly educated men do not significantly differ from the level of compression experienced by women in the lowest education group. The degree to which highly educated men might ultimately close the gender gap in compression is unclear. Given the substantial advantages that the highly educated have relative to those with less education, monitoring mortality compression among highly educated men and women may provide a glimpse into the mortality dynamics of the population as a whole in the future.

Appendix

Appendix Table A1
Characteristics of the Analytic Samples in the HRS and NHIS-LMF

	HRS	NHIS-LMF
Respondents		
Age range at interview	50 - 100+	50 - 89
Age range at end of follow-up	50 - 100+	50 - 100+
Race-Ethnicity	Non-Hispanic white	Non-Hispanic white
Nativity	U.S. born	U.S. born

	HRS	NHIS-LMF
Survey Data		
Interview years	1992 - 2006	1989 - 2001
Design	Longitudinal; Civilian non-institutionalized population	Cross-Sectional; Civilian non-institutionalized population
Mortality Data		
Follow-up years	1992 - 2006	1989 - 2002
Follow-up source	Probabilistic match to the NDI; HRS Tracker files	Probabilistic match to the NDI

Notes: HRS = Health and Retirement Study; NHIS-LMF = National Health Interview Survey Linked Mortality Files; NDI = National Death Index; Refer to the methods section for additional information.

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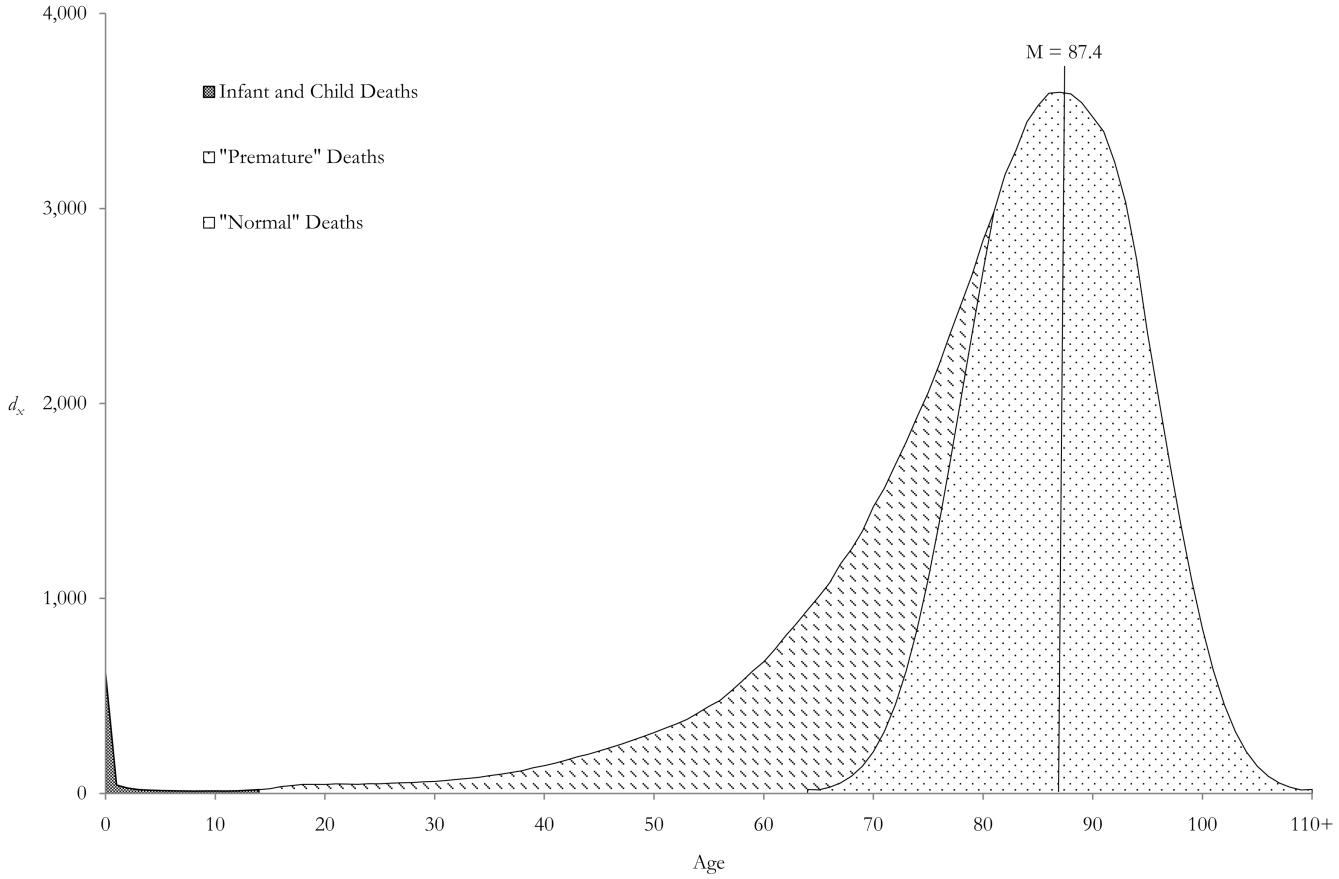

Figure 1.
A Hypothetical Illustration of Lexis's Classification of Life Table Deaths for Women in the United States, 2002-2006
Source: Human Mortality Database. The figure contains a five-year average d_x over 2002-2006 period.

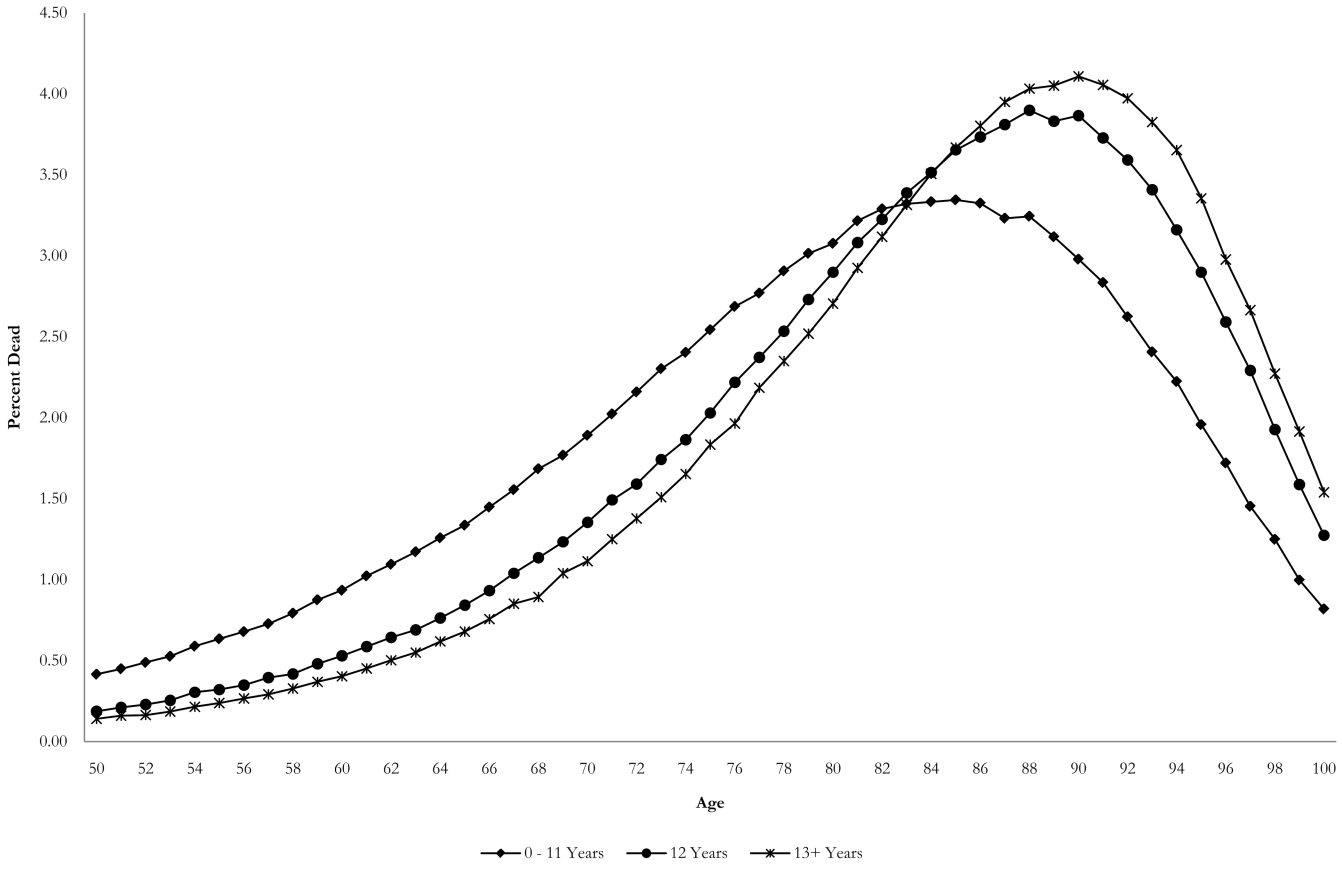

Figure 2.
Age-Specific Percentage of Life Table Deaths from Simulation Models by Education, HRS Women

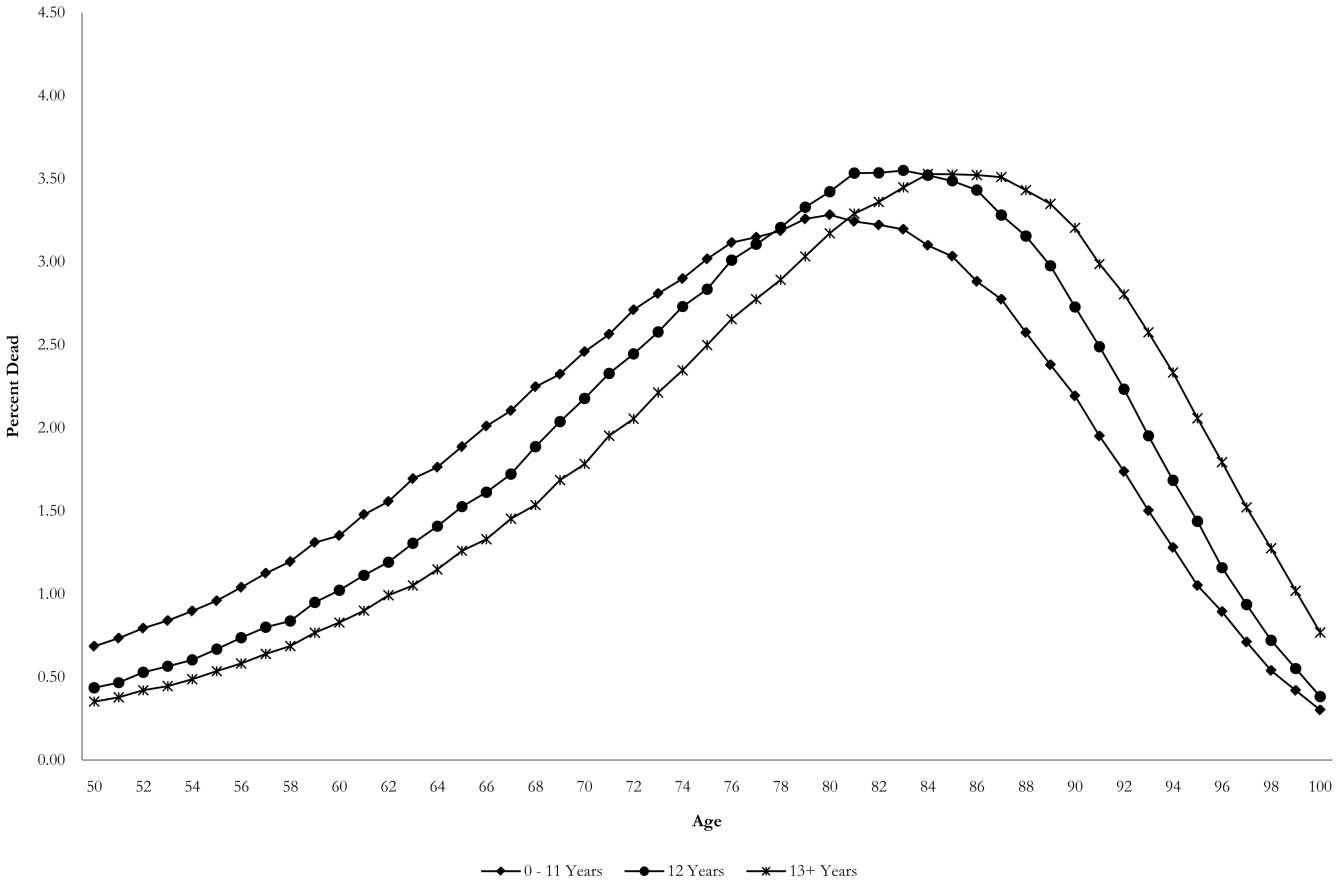

Figure 3.
Age-Specific Percentage of Life Table Deaths from Simulation Models by Education, HRS Men

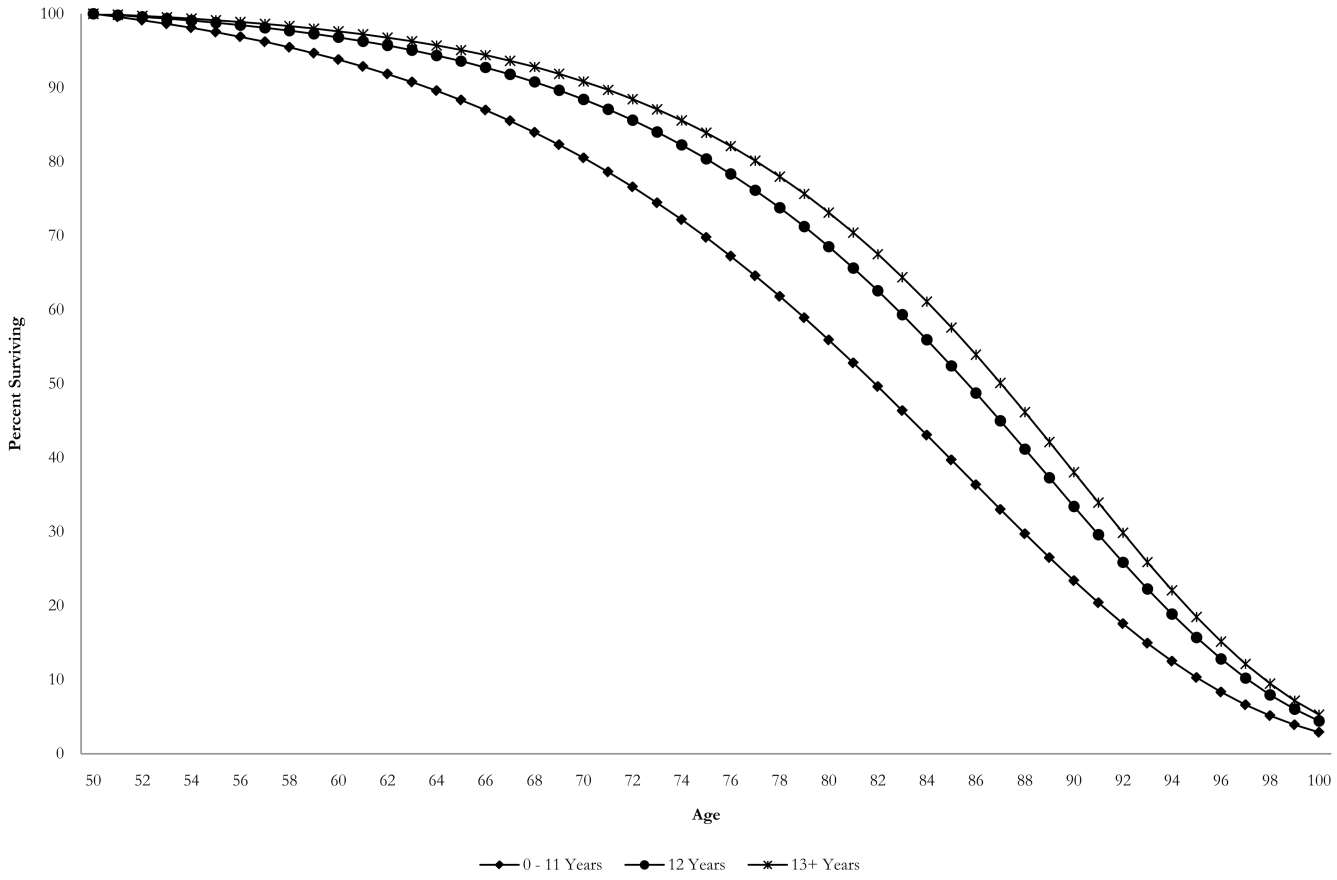

Figure 4.
Age-Specific Percentage of Life Table Survivors from Simulation Models by Education, HRS Women

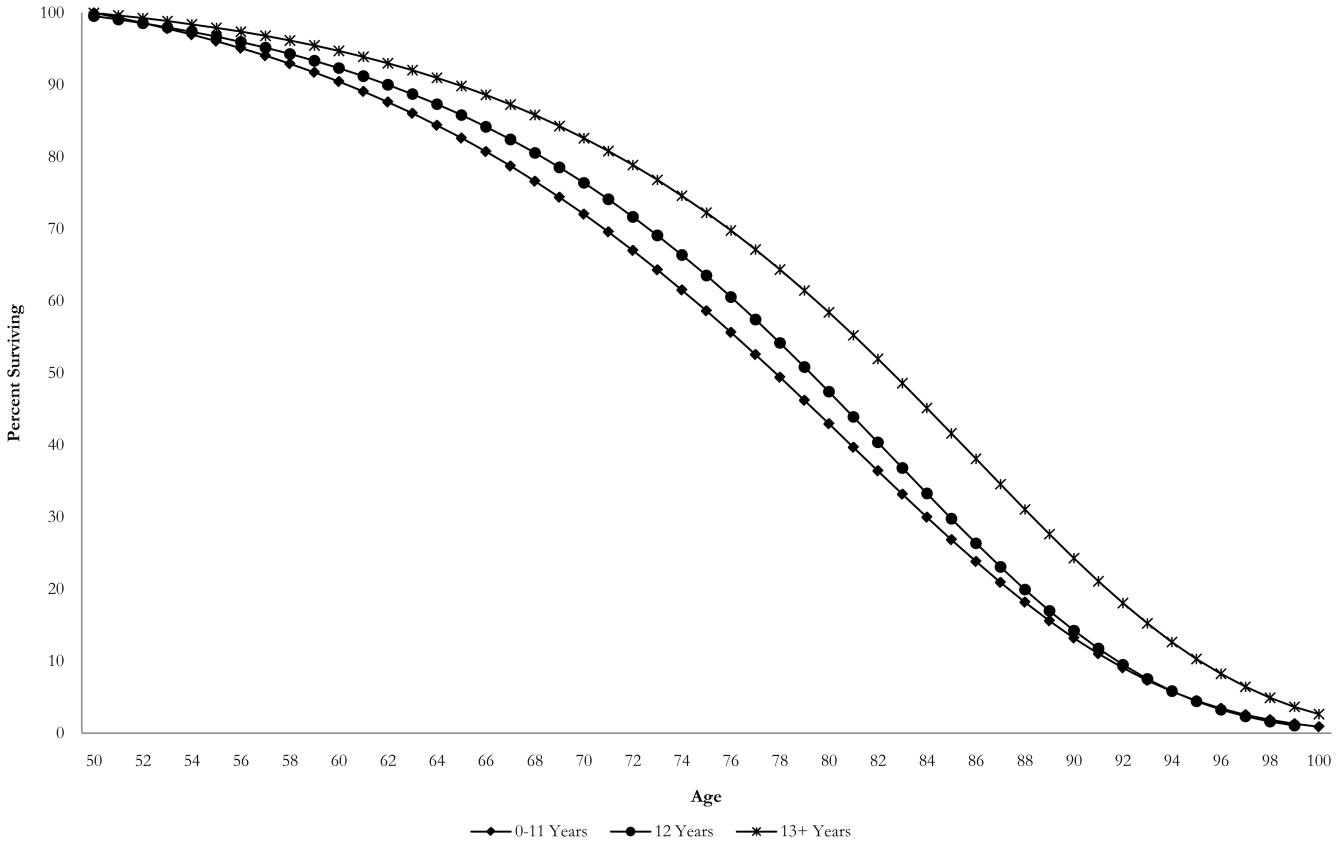

Figure 5.
Age-Specific Percentage of Life Table Survivors from Simulation Models by Education, HRS Men

Table 1

Comparative Life Expectancy at Age 65 by Gender

	NLMS[a] (1979-1989)	US[b] (1985)	NHIS-LMF[d] (1989-2002)	US[c] (1995)	HRS[e] (1992-2006)	US[c] (1999)
Women						
	20.20	18.71	20.08	19.17	20.17	19.19
	(20.08 - 20.32)	(18.65 - 18.77)	(19.98 - 20.18)	(19.11 - 19.23)	(19.90 - 20.44)	(19.13 - 19.25)
Men						
	15.50	14.58	16.55	15.63	16.94	16.05
	(15.38 - 15.62)	(14.53 - 14.63)	(16.45 - 16.65)	(15.57 - 15.69)	(16.66 - 17.21)	(15.99 - 16.11)

Notes: The 95% confidence intervals are calculated using the method outlined in Chiang (1960).

[a] *Source*: National Longitudinal Mortality Study (NLMS, 1979-1989); Lin et al. (2003, Table 2, p. 242). The estimates are for whites. The standard errors presented in the original text were used to construct the confidence intervals.

[b] *Source*: National Center for Health Statistics (1989). The estimates are for whites in 1985.

[c] *Source*: Human Mortality Database. The estimates include all race-ethnic groups in 1995 and 1999.

[d] *Source*: National Health Interview Survey Linked Mortality Files (NHIS-LMF, NHIS: 1989-1996, NDI: 1989-2002). The analyses are for all race-ethnic groups and were restricted to men and women ages 50+ with valid sample weights.

[e] *Source*: Health and Retirement Survey (HRS, 1992-2006). The analyses are for all race-ethnic groups and were restricted to men and women ages 50+ with valid sample weights.

Table 2

Number of Deaths and Person Years of Exposure to the Risk of Death in the HRS and NHIS-LMF by Gender and Education

	HRS		NHIS-LMF	
	Deaths	**Exposure**	**Deaths**	**Exposure**
Women				
0-11 Years	1,153	26,183.5	8,968	219,156.0
12 Years	1,037	48,389.5	8,371	371,855.5
13+ Years	803	42,939.5	4,784	241,619.0
Men				
0-11 Years	1,065	20,940.5	8,845	170,226.5
12 Years	925	30,229.5	7,262	234,815.0
13+ Years	952	40,419.0	6,100	255,031.0
Total	5,935	209,101.5	44,330	1,492,703.0

*Note*s: The numbers are not weighted. The estimates are for non-Hispanic whites.

Sources: Health and Retirement Study (HRS, 1992-2006) and National Health Interview Survey Linked Mortality Files (NHIS-LMF, 1989-2002).

Table 3

Education and Gender Differentials in the Modal Age of Death (M) and the Standard Deviation Above the Modal Age of Death (SD(M+)), and the Number of Life Table Deaths at the Mode (d(M)) in the HRS and NHIS-LMF

	HRS			NHIS-LMF		
	0-11 Years	**12 Years**	**13+ Years**	**0-11 Years**	**12 Years**	**13+ Years**
Women						
M	85.28	88.94	90.24	85.65	88.38	89.95
	(83.85-86.71)	(87.79-90.09)	(89.32-91.16)	(84.34-86.97)	(87.28-89.49)	(88.98-90.92)
SD(M+)	6.95	5.36	4.77	6.97	5.69	5.03
	(6.31-7.58)	(4.84-5.89)	(4.34-5.20)	(6.33-7.61)	(5.16-6.22)	(4.56-5.49)
d(M)	23,585	27,175	28,833	22,515	25,400	26,032
	(22,410 - 24,761)	(25,413 - 28,937)	(27,173 - 30,494)	(21,910 - 23,121)	(24,878 - 25,923)	(25,368 - 26,695)
Men						
M	80.84	83.51	85.92	79.59	82.95	85.34
	(78.95-82.73)	(82.13-84.88)	(84.63-87.2)	(78-81.18)	(81.76-84.15)	(84.34-86.34)
SD(M+)	8.19	7.14	6.58	8.69	7.36	6.54
	(7.32-9.05)	(6.46-7.83)	(5.98-7.17)	(7.98-9.40)	(6.81-7.91)	(6.09-6.99)
d(M)	23,021	24,970	24,845	22,174	24,503	26,083
	(21,673 - 24,369)	(23,330 - 26,609)	(23,453 - 26,237)	(21,706 - 22,641)	(23,983 - 25,023)	(25,396 - 26,771)

Notes: Estimates are for non-Hispanic whites. 95% confidence intervals are in parentheses. M = Modal age at death; SD(M+) = Standard deviation above the modal age at death; d(M) = Life table deaths at the modal age of death.

Sources: HRS (1992-2006) and NHIS-LMF (1989-2002)