Whose mortality decelerates?

Multi-stage mortality selection and the poverty puzzle

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ABSTRACT:

A pervasive demographic result is that mortality decelerates: it rises more slowly at very old ages than at younger ones. We extend deceleration analysis by incorporating new dimensions of social stratification, estimating deceleration in U.S. subpopulations defined by baseline health and poverty status, as well as race and sex. Using U.S. Medicare data, we follow 28 million Americans—nearly the entire elderly population—from 1993 to 2002, estimating nearly non-parametric mortality hazards. Our results create a puzzle. The traditional explanation of deceleration is mortality selection: populations become increasingly robust as their frailest members die. Conventional interpretations posit that populations with higher mortality should decelerate at younger ages since they are subject to greater selective pressure. We find the expected pattern along lines of race and sex: higher-mortality African-Americans and men decelerate earlier than white Americans and women. For health and poverty, however, the pattern reverses: it is the non-sick and, especially, the non-poor whose mortality decelerates substantially earlier and more sharply than their higher-mortality counterparts. To explain this contradiction with the conventional view, we extend mortality selection theory. Drawing on the demographic literature suggesting mutual causation between poverty and ill health, our multi-stage mortality selection model suggests that the non-poor population may be more heavily selected because the frail tend to become poor. The dynamism of this substantively plausible model, however, comes at a price. Since people enter the poor population from the non-poor, the poor's frailty composition changes with selection on the non-poor population. We show that this relationship between populations makes deceleration order essentially unpredictable, and discuss implications for using deceleration patterns to understand health inequalities.

Most demographic models posit that human mortality increases exponentially as the population ages, assuming, for example, the Gompertz Law of Mortality. Yet many populations' mortality rises more slowly at very advanced ages than the exponential increase it displays throughout most of adulthood. Such mortality deceleration has been documented in human populations (Olshansky 1998; Vaupel 1997; Horiuchi and Wilmoth 1998; Lynch and Brown 2001; Lynch, Brown, and Harmsen 2003); animal and insect studies, such as as fruit flies (Carey et al 1992; Fukui, Xiu and Curtsinger 1993) and nematode worms (Vaupel 1997); and even automobiles (Vaupel 1997, Vaupel and Owen 1998). Demographers have studied mortality deceleration for a variety of reasons. Early work was primarily interested in accurate descriptions of population-level mortality in old age (see Olshansky 1998 for a review). Subsequent work studied mortality deceleration to test the Gompertz Law of Mortality (Rosenberg et al 1973), inform the debate on natural limits to life expectancy (e.g., Vaupel 1997), study social inequality (Lynch, Brown and Harmsen 2003), and move from population-level to individual-level models of mortality (e.g., Vaupel and Gampe 2009).

The dominant explanation for mortality deceleration in the literature is *mortality selection*: over a cohort's lifespan, those most prone to dying do so early, leaving a remaining population with a correspondingly diminished average hazard of death. In the language of mortality selection, the *frailest* die, leaving a more robust population at old ages.

One way of empirically testing the selection explanation relies on comparing the deceleration order of high- and low-mortality groups. Traditional mortality selection theory implies that high-mortality populations should decelerate at younger ages than their lower-mortality counterparts because high mortality intensifies selective pressure. Indeed, a major theoretical result in the mortality selection literature is that (given appropriate assumptions) even

when two populations have the same shape of their initial distribution of heterogeneity in mortality risk, the one with greater absolute mortality will be more heavily selected for robustness (Vaupel and Yashin 1985; Vaupel, Manton and Stallard 1979; Kannisto 1992). This result has been used, for example, to explain the black-white mortality crossover in the United States: even when the two populations are assumed to have equal baseline frailty distributions, African-Americans' higher mortality throughout most of the lifecourse can make them so much more selected than white Americans that at the oldest ages, their average mortality is lower than whites' (Manton and Stallard 1981; Nam 1995; Lynch, Brown and Harmsen 2003; Dupre 2006; Elwert and Wrigley-Field, unpublished).

This paper draws on a more complicated model of mortality selection to explain new empirical results that contradict this conventional exposition. Using U.S. Medicare data on nearly the entire U.S. elderly population, we estimate deceleration among populations defined by key dimensions of stratification: sex, race, health, and poverty status. These results create a puzzle for the conventional selection theory. We find the expected pattern of early deceleration among high-mortality groups along lines of race and sex: higher-mortality African-Americans and men decelerate earlier than white Americans and women. For health and poverty, however, the pattern reverses: it is the lower-mortality non-sick, and especially, the non-poor whose mortality decelerates substantially earlier than their higher-mortality counterparts.

To explain this contradiction with the conventional view, we draw attention beyond mortality to a broader range of dynamic processes acting on heterogeneous populations. Our *multi-stage mortality selection* hypothesis suggests that frailer individuals are more likely, not only to die, but also to become poor. As a result, high-mortality (poor) groups are not only selected for robustness, but also potentially replenished by frail additions, whereas low-mortality

(non-poor) groups are powerfully selected for robustness. Given proper circumstances, low-mortality groups may then decelerate earlier than high-mortality groups, a fact that might account for the early deceleration we find among the non-poor. In that sense, our results offer evidence for deep inequalities within U.S. subpopulations, with disadvantage cutting across social and health domains.

The multi-stage model's flexibility also creates a dilemma: the model can equally well accommodate *early* or *late* deceleration among the poor—rendering deceleration order uninformative as a test of the selection hypothesis against individual-level alternatives (i.e., against the theory that aging processes really decelerate at the individual level). We conclude that this dilemma suggests the need for a different strategy to test substantively realistic selection models against individual-level alternatives than analyzing aggregate mortality patterns. However, our empirical and theoretical results suggest that *if* mortality deceleration is explained by selection—that is, by changes in population composition rather than changes in individuals' mortality gradients—then changes in poverty status, not only mortality itself, must be changing the distribution of mortality risk across U.S. subpopulations.

Previous Results on the Age of Deceleration

Gompertz himself knew (1825) that the Gompertz Law of Mortality—the claim that mortality continually accelerates—does not hold at the oldest ages. Olshansky (1998) documents a long history of awareness of mortality deceleration, variously timed between ages 75 and 90, beginning with Gompertz and Makeham in the nineteenth century and extending to Strehler and Mildvan's (1960) classic study. More recent analyses often find older ages of deceleration onset than those early studies, due in part to the relatively recent adoption of deceleration measures

based on derivatives of the mortality hazard itself, rather than its logarithm. For example, in a comparison of methods, Rau et al (2009) variously date deceleration in English and Welsh female populations at 65, 92, or 103, depending on the deceleration measure adopted. In a study of super-centenarians using the International Database on Longevity, Vaupel and Gampe (2009) find that by age 110, the annual probability of death has leveled into a constant value at fifty percent, although that unique dataset, since it focuses only on the very oldest verified ages, does not allow the onset of deceleration to be timed.

A small number of important studies have studied deceleration for variously defined specific groups within national populations. Horiuchi and Wilmoth (1998) compare deceleration timing across cohorts and period in Sweden (from 1861-1990) and Japan (from 1951-1990), and across causes of death in Japan. In both countries, they find that the age at deceleration has risen as senescent mortality fell. Using multiple measures of deceleration on Human Mortality Database data, Rau et al (2009) confirm the finding of a rising age at deceleration between 1950 and 2004 for female populations in Sweden and Japan, as well as the United States, England and Wales, Poland, and the former East German areas.

In the United States, Lynch and Brown (2001) compare deceleration between white men and white women from 1968-1992, and Lynch, Brown and Harmsen (2003) compare blacks and whites between 1970 and 1992. Like Horiuchi and Wilmoth, they find that for white men and women, the age at deceleration has risen over time as mortality fell. White women in their (2001) study decelerate later than white men, but their age at deceleration rises more slowly; they decelerate at age 95 in 1968 and 96 by 1992, while white men's age jumped from 93 to 95 in the same period. Estimating a parametric model on a sample aged 20-100, Lynch et al's (2003) racial comparison finds that African-American age at deceleration varies significantly depending

on whether the data are adjusted for possible sources of age misreporting: in 1970 it is 96 according to the raw data and 104 when adjusted, and by 1992 it has fallen to 92 or 101, respectively. Meanwhile, the aggregate white subpopulation's age at deceleration stays nearly constant at 95. Since our study examines U.S. populations beginning in 1993, Lynch et al's results form a historical as well as a theoretical background for this paper.

Previous work on group-specific deceleration thus highlights two themes critical to this study. First, previous research compares mortality deceleration across groups defined by dimensions of social stratification (i.e., race and sex in Lynch et al) or physiological states (i.e., disease susceptibility in Horiuchi and Wilmoth). Second, previous work predicts that, all else equal, lower mortality should delay deceleration (as in the various studies' finding that deceleration ages rise across period).

Mortality Deceleration, Mortality Selection, and Senescence

Two explanations compete to explain mortality deceleration: mortality selection and decelerating senescence (aging). The difference between these hypotheses is vividly illustrated by Vaupel's (1997; Vaupel and Owen 1998) analysis of automobiles' 'mortality' deceleration. Selection occurs because deficient cars 'die' earlier, leaving behind cars that are more robust—hence, mortality decelerates. Decelerating senescence occurs because older cars, presumably driven by older drivers, are driven less often and with greater care. Thus, the processes that wear and tear a vehicle slow down, also resulting in decelerating mortality. The difference between selection and senescence is the difference between mortality deceleration explanations based at the population level or the individual level.

This paper examines these hypotheses by comparing the order in which subpopulations' mortality begins to decelerate and exploring the conditions under which that ordering is compatible with the mortality selection explanation. Here, we explain the logic of this method and why previous demographers have adopted this approach.

If we could measure comprehensively the underlying causes of mortality and how individuals acquire them, we could directly examine whether mortality decelerates because the particular people who are susceptible to those causes die out (selection), or because people in general become less susceptible at very old ages (senescence). But despite recent strides in identifying key physiological components of aging (e.g., allostatic loads, inflammation (Yang and Lee 2010), accumulated stress (Geronimus et al 2006)), the aging process in general remains mysterious. A small number of studies have made important contributions by modeling the aging process and unobserved heterogeneity together, even though knowledge of mortality risks of course remains partial. For example, Manton et al (1994, 1995) model mortality as a function of fixed, unobserved individual frailty and observed physiological states that are themselves modeled as stochastic functions of individuals' physiological history. But making these models analytically tractable requires assuming homogeneity within key parts of the analysis—so, for example, in the studies by Manton and colleagues, individuals' mortality risk conditional on their physiological state is heterogeneous, but the risk of entering or exiting any physiological state, given the individual's past states, is homogeneous. Yet as will become clear, the question raised in the present study is precisely whether mortality risk is plausibly homogeneous within and across groups whose boundaries seem, at first glance, to have little to do with mortality.

Absent comprehensive measures of individual risk factors of mortality, demographers are left trying to adjudicate between the hypotheses using the mortality patterns themselves. The

fundamental problem is that for any particular aggregate mortality pattern, selection and senescence can never be fully separated from one another empirically. The senescence hypothesis holds that individuals' mortality risk eventually decelerates, if they live long enough. Without the ability to decompose mortality risk into specific traits, however, we can only measure individuals' mortality risk at a given age with the coarsely dichotomous measure of whether or not they survived it. As a result, measures of mortality risk across age always concern the mortality of groups. Since any group whose mortality risk is internally heterogeneous—that is, any group identifiable with real data—presumably experiences some degree of mortality selection, we are left with the problem of whether to attribute the resulting patterns to senescence or selection.

Nevertheless, there is a real claim at the heart of the selection hypothesis that, alongside auxiliary assumptions, has been used to formulate testable predictions about mortality deceleration. The mortality selection hypothesis, as we formulate it, claims that mortality deceleration at age *a* results from *which* individuals died before reaching *a*, and therefore, that had a different group of individuals died before age *a* than actually did, deceleration need not have occurred. The chief premise underlying this claim is that the variation in individuals' mortality risk must be stably ordered at key ages. This means that individuals who are relatively higher or lower in mortality risk at age *a*, when deceleration occurs, must also have been so at the earlier ages during which much of the cohort's mortality occurred. Thus, had the ones higher in mortality risk not died, they would still have higher mortality risk at old ages, and therefore would raise the population's mortality, preventing deceleration. Or, had more of the ones high in mortality risk died, they would not later be in the population, and their absence would produce deceleration in the aggregate mortality.

This stable ordering condition is fairly minimal, but is not obviously met in all cases. A heart attack at age forty might kill you, but it might also save your life if it gets you to change your chain-smoking ways (this example was suggested to us by Erik Olin Wright); the same might be said of the negative pleiotropies that were once the staple of proposals that lifespans have an inherent natural limit. But we argue that this condition is the minimal theoretical commitment of a mortality selection explanation of deceleration. Indeed, the model proposed in this study extends this condition into a stronger claim, arguing that if selection is fully to explain observed deceleration, then disadvantage must persist across not only age, but also domain—so that individuals at comparatively high risk of dying are also at comparatively high risk of social disadvantages, such as poverty. Like the more conventional condition requiring stable ordering of mortality risk across age, this is a substantive claim about inequality that might turn out empirically to be true or false.

This claim about the relationship between individuals' mortality risk over age has been combined with more specific assumptions to test mortality selection in two ways (for an explicit discussion of the logic underlying the adoption of the selection explanation as a null hypothesis, see Horiuchi and Wilmoth [1998]). One tradition, associated with the founders of mortality selection theory (e.g., Vaupel et al [1979], Vaupel and Yashin [1985]), assumes that the differences between individuals' mortality risk are proportional over age. This tradition operationalizes frailty as a trait fixed in individuals, raising or lowering their mortality in relation to the average of their birth cohort by a constant factor across age. By also assuming a flexible Gamma parameterization of a cohort's frailty distribution at birth, studies in this tradition can estimate the parameters of that distribution. Early studies then endeavored to directly assess the plausibility of the resulting frailty distribution; for example, Vaupel and Carey (1993; see also

Vaupel 1997) use this method to argue that selection alone does not plausibly explain the deceleration observed in Mediterranean fruit fly populations. The same parametric approach to modeling frailty has been widely applied to human populations (e.g., Manton and Vaupel [1985], Vaupel [1988], Yashin and Iachine [1997], Vaupel and Gampe [2009]). In contrast with the medfly studies, however, studies of human mortality generally do not explicitly assess the plausibility of the parameters derived from assuming Gamma-distributed frailty—perhaps because the extremely broad range of biological and social factors potentially comprising human 'frailty' makes this untenable. For an important strand of research, Gamma-distributed fixed frailty is now a premise rather than an object of study.

The second tradition testing the selection explanation relaxes this first tradition's assumptions about the functional form of mortality-risk heterogeneity *within* a population, replacing them with assumptions about the comparability of mortality-risk heterogeneity *across* (sub-)populations. This tradition, of which the present study is a part, compares the *order* of populations' deceleration, rather than fitting the total mortality curve to a parametric frailty form; it requires that the signs, but not the magnitudes, of differences between individuals' mortality risk be relatively stable over age. The logic of this approach uses the variation in population-level mortality experiences to approximate an answer to the question: What would have happened to *this* population if a different subset of its membership had died—would it have decelerated earlier, later, or not at all? In this tradition are the previously discussed studies by Lynch and Brown (2001), Lynch et al (2003), and Horiuchi and Wilmoth (1997). Their consistent finding that the age at deceleration rises over time as mortality falls illustrates a general prediction of mortality selection theory, which forms the key theoretical backdrop to this

study: all else equal, higher-mortality populations should decelerate at younger ages than lower-mortality populations, since the former are more heavily selected.

Whether or not they commit themselves to a functional form for the distribution of individual-level frailty, all previous studies that we are aware of assume a functional form (such as a logistic or arctangent curve) for population-level mortality. Lynch et al (2003), for example, adopt a relatively flexible parametric (arctangent) mortality function to mitigate possible distortions arising from a small sample at the oldest African-American ages. This correction, however, is purchased at the price of assumptions about the functional form of population-level mortality over age, even though ascertaining the correct form of this function is a major purpose of studies that attempt to document mortality deceleration.

These parametric assumptions, moreover, are particularly problematic when the purpose of a study is to compare deceleration patterns across populations. Parameter estimation will draw more heavily from younger ages in populations whose age structure is young than in populations with a larger composition of the oldest ages. In the U.S. context, then, it is precisely among the smaller African-American subpopulations that globally parametric curves run the greatest danger of understating the divergence of oldest-age hazards from their earlier trajectory by incorrectly interpreting the divergence as noise. Clearly, methods that are more conservative in identifying deceleration for some populations than others are problematic for studies whose purpose is to compare the age at deceleration.

In this analysis, therefore, we estimate nearly non-parametric population-level hazards to ask: What would need to be true for mortality selection to fully explain the deceleration order of U.S. subpopulations defined by major dimensions of stratification? What does this tell us about mortality selection and, ultimately, about unequal mortality experiences in the United States?

Data

We analyze a large, longitudinal dataset derived from Medicare Claims Databases that follows 28.7 million elderly Americans from 1993 to 2002 for a total of over 200 million person years. Medicare databases capture 96 percent of Americans above age 65 in 1993 (Kestenbaum 2000). Since this dataset combines information from several different Medicare files, the files were matched using unique individual-level identifiers, with the record linkage rate exceeding 99 percent. The major benefits of these data are their near-population coverage, their accuracy, their precision, and their inclusion of covariates representing important dimensions of heterogeneity in mortality risk.

Crucial to any analysis of mortality deceleration is the accuracy of reported ages in the data, since overestimation of the oldest ages may have marred some previous efforts to measure deceleration in the United States (Elo and Preston 1994; Preston et al 1996; Hill et al 1997; Kannisto et al 1994; but see also Hill et al 2000). Vital statistics in this study's dataset are drawn from the Social Security Administration (SSA)'s Master Beneficiary Record file. Prior evaluations (Sohn et al 2006) suggest that these Master Beneficiary Record data surpass all other national mortality datasets—even the SSA Death Master File—in matching the National Death Index. In a comparison of an earlier period of Medicare data to Census and death certificate data, Kestenbaum (2002) also finds Medicare to be the most accurate. The accuracy of age reporting stems from the SSA's rigorous monitoring to prevent fraudulent benefit claims. This monitoring became more stringent in 1965, when ages were required to be verified at the time of Medicare enrollment. Preston et al (1996) therefore suggest that the ages in SSA data are particularly accurate among cohorts born after around 1900, since the post-1965 enrollment procedures

preclude some potential sources of bias that Preston and his colleagues identified in death certificates, where ages are assessed only at the time of death. The oldest cohorts in this study's dataset were born in 1896; those contributing to the oldest age, 96, were born in 1896-1905, which means the majority were born after Preston's 1900 cutoff. In addition, we exclude ages 65-66 to mitigate against possible selection bias at the earliest Medicare-eligible ages, since some people may wait to enroll in Medicare if they do not urgently need the health coverage.

Beyond birth and death dates, the data include respondents' sex, race, geographic region, poverty, and health. These covariates are measured only at baseline. Sex and race information come from the Medicare Vital Status file; the race variable is drawn from the SSA's Master Beneficiary Record and updated from self-reported race from applications for replacement Social Security cards. The racial comparison is limited to African-Americans and white Americans because previous research has supported the accuracy of those classifications (Lauderdale and Goldberg 1996; Arday et al 2000; Elwert and Christakis 2006). The regions are Census regions, which divide the U.S. into ten areas. Baseline poverty status is measured as joint eligibility for Medicaid and Medicare in 1992 (following Clark and Hulbert 1998; see also http://www.cms.hhs.gov/dualeligibles).

The baseline health measure is constructed to summarize detailed health information into a standardized unidimensional measure. The Medicare Provider and Analysis Review file provides in-patient hospitalization records for 1992, from which are extracted physician-provided information about chronic illnesses. These are summarized into Charlson Comorbidity Scores (Charlson et al 1987), a weighted count of serious chronic conditions, widely used in medical research (see, e.g., Valderas et al 2009) and considered a reliable predictor of succumbing to further health stresses. Work on the Cumulative Index (Yashin et al 2007, Kulminski et al 2007)

also suggests that morbidity measures summarizing the number of distinct health detriments, as these Charlson scores do, are excellent measures of aging and health deterioration processes. To ensure sufficient sample size for all covariate combinations while maintaining important health distinctions, we use a dichotomous measure comparing those with Charlson scores of two or more (two chronic conditions or one very serious one) to those with scores of zero (no chronic conditions).

Methods

Non-parametric hazard estimation

We calculate nearly non-parametric yearly mortality hazards using poisson regression on a high-dimensional contingency table. Poisson regression estimates death rates by estimating an expected count of deaths for each combination of covariates and adjusting for exposure (by entering it into the equation with a log-linear coefficient constrained to one, generating an expected count divided by exposure, i.e., a rate); our data permit us to adjust for exposure measured to the day. The rates are calculated independently for each sex and race according to the following equation:

$$\mu = \exp[\alpha + \sum_{a=65}^{96} \boldsymbol{A}\beta_{1,a} + P\beta_2 + \sum_{a=65}^{96} P * \boldsymbol{A}\beta_{3,a} + S\beta_4 + \sum_{a=65}^{96} S * \boldsymbol{A}\beta_{5,a} + P * S\beta_6 + C\beta_7 + \sum_{r=1}^{10} \boldsymbol{R}\beta_{8,r}]$$

In this model, **A** is a vector of indicator variables for the thirty ages included in the study. We use indicator variables so that the estimated hazard is not constrained to any particular functional form over age. The *p* and *s* terms represent poverty and sickness, respectively. Each is entered as

a main effect and also interacted with each age indicator, so that the estimated effects of poverty and sickness also vary freely over age. Poverty and sickness are also interacted with one another so that the main effect of poverty can differ between those with and without chronic illnesses, and vice-versa. Finally, we adjust for yearly cohort *c* and for geographic region, the latter represented in the vector of indicator variables **R**. Models with more flexible parameterization of cohort, including models with separate indicators for each cohort, differed only negligibly from those with the log-linear cohort term we report here. This may reflect the relatively small number of cohorts observed in these data, nine yearly cohorts for each age; one might expect a more complicated cohort pattern over a longer stretch of time. We estimate two versions of this model for each race and sex: one omitting all terms containing *s*, *p*, or **R**, which measures age-specific hazards adjusted only for cohort, and a full model including poverty, sickness, and region.

Estimating derivatives

We estimate the first derivative of the hazard at age *a* as the average difference between the (smoothed) hazard at *a* and the hazards of the immediately adjacent age in each direction, and the second derivative as the average difference in first derivatives between *a* and the adjacent ages. Before estimating the derivatives, we smooth the hazards using a running-mean smoother with a smoothing window of only a single observation on each side.

In estimating the derivatives, we lose information at the endpoints of the age interval. This is necessitated by a dilemma for calculating derivatives of a hazard whose estimated relationship to age is not parametrically constrained. The dilemma in brief is that estimating the derivatives requires some degree of both smoothing and extrapolation, yet each of these can induce

distortions near the endpoints of the age interval. Our procedure avoids artifactual deceleration while nevertheless allowing the derivatives to be estimated, albeit by sacrificing the endpoints.

Estimating the derivatives requires smoothing because small fluctuations in the hazard can produce exaggerated distortions in the derivatives. This is particularly true for small fluctuations representing tempo effects, because they will in general produce a negative correlation between successive ages' random deviations from their expected hazards. For example, if a smaller than expected proportion of those on the cusp of death die at age 89, we would expect a larger than average number of deaths at age 90. This correlation would tend to exaggerate the slope at age 89. Moreover, estimating the derivatives requires interpolation, because derivatives are defined only on continuous functions. This becomes problematic only at the endpoints of the age interval. Here, the analyst faces a choice between taking the differential on only one side, or extrapolating the data to an additional age. Yet taking a differential on only one side will necessarily distort estimates of a changing derivative—for example, if the hazard is accelerating, taking the differential only from the left at the oldest age will underestimate the derivative, potentially introducing artifactual deceleration—and extrapolation is arbitrary when the purpose of the study is to ascertain an unknown shape for the hazard's derivatives. Meanwhile, a parallel problem arises with smoothers, since an accelerating hazard at age *a*, smoothed only from ages younger than *a*, will be underestimated unless the smoothing function explicitly assumes acceleration. The distortion created in the hazards will generally be very small, but may be substantial in the derivatives. (In an unreported analysis, we verified this empirically by artificially limiting the age interval, and found that in each new interval, the derivatives were distorted at the endpoints but not elsewhere.) Thus, to estimate derivatives at the endpoints of the age interval, an analyst must choose between assuming acceleration, which potentially masks

real deceleration, or estimating derivatives at the oldest ages only from the preceding ones, which potentially induces false deceleration.

Given this dilemma, we choose not to estimate derivatives at the endpoints so that we can impose an equal smoothing window and equal differentiation window on all points included in the deceleration analyses. We do this by using a running mean smoother with a smoothing window of a single observation on each side, then dropping the endpoints before calculating the derivatives as described above. We also drop the endpoints after each derivative calculation. Thus, we estimate unsmoothed hazards on ages 65-96; smoothed hazards on aged 66-95; first derivatives on ages 67-94; and second derivatives on ages 68-93.

This strategy, which imposes minimal and local patterns on the data, differs from the more globally parametric hazard estimation common to previous deceleration studies in two respects. First, and importantly, since we smooth on a collapsed form of the dataset in which the each age is represented by a single observation, the smoothing does not draw more heavily from early ages in some populations than others, regardless of each population's age distribution. Second, the smoothing does not impose the assumption that the mortality hazard has a single maximum – an assumption undergirding most attempts to isolate a single origin age for mortality deceleration. Indeed, some of the subpopulations' hazards appear to have several local maxima that are stable over reasonable smoothing and seem to represent real fluctuations in the hazard curves. Other local maxima may, however, more reasonably be considered noise. For this reason, we analyze derivatives by inspection as well as by calculating their global maxima.

Measuring deceleration

Mortality deceleration is measured against the conventional Null model of monotonically increasing acceleration in the age-specific hazard of death, embodied in the Gompertz law of mortality. Table 1 defines three plausible measures of deceleration by first, second, and third derivatives of the hazard of death with respect to age. (1) *Relative deceleration* occurs if mortality increases and accelerates, but accelerates more slowly as the population ages. With relative deceleration, the first derivative (slope) and the second derivative (acceleration) are positive, but the third derivative (jerk) is negative. (2) *Absolute deceleration* occurs if mortality increases, but increases more slowly as the population ages. Absolute deceleration is measured by a positive slope and negative acceleration. (3) Finally, *mortality decline* occurs if the hazard of death at older ages is lower than it is at younger ages—the slope itself has turned negative.

Clearly, among these three measures, relative deceleration represents the most sensitive. Rau et al (2009) argue that relative deceleration best captures demographers' intuition for when mortality deviates from the increasingly accelerating mortality of the Gompertz law with its positive three derivatives. Empirically, however, relative deceleration is potentially problematic precisely because it is so sensitive: third derivatives may fluctuate more wildly in response to sampling variation, tempo effects, and data sparseness than lower-order derivatives. For this reason, we focus on absolute deceleration as a more conservative measure that is more robust to temporary aberrations from true underlying trends, as do Lynch and Brown (2001) and Lynch et al (2003). Where sample size allows—in the largest groups defined by race and sex only—we additionally investigate relative deceleration.

We note that there is no reason *a priori* to assume—*pace* Gompertz—that mortality should decelerate only once. If individuals' mortality risk continually accelerates but populations

continually are selected, the interaction of those two dynamics might well result in alternating periods of acceleration and deceleration—reflecting not the tempo effects built into stochastic processes with fine-grained time intervals, but the 'real' pattern of mortality. Likewise, since the real processes of individual aging are largely unknown, if senescence does decelerate, it might just as well re-accelerate. To avoid the "false" detection of temporary deceleration, we measure the onset of absolute deceleration by the age of maximum first derivative of the smoothed hazards during the observation window (i.e., immediately preceding negative acceleration), and we measure the onset of relative deceleration by the age of maximum second derivative (i.e., immediately preceding decreasing acceleration). The latter measure, which by definition requires the third derivative to be zero and immediately thereafter negative, allows us to estimate relative deceleration without estimating the third derivative at the cost of data loss at the endpoints. Visual inspection of all derivatives augments these rules.

Results

Sample descriptives

Table 2 shows descriptive statistics for the sample at baseline and select mortality data. Over all, all groups defined by sex, race, poverty, and sickness are sufficiently populated to permit essentially non-parametric analysis. The smallest group—poor, sick black men—is 17,316 individuals. Of the 28,658,175 people alive at baseline, 3,278,355 are white women, 693,583 are black women, 1,808,486 are white men, and 276,829 are black men. We note that within each sex, black Americans are twice as likely as white Americans to be poor at baseline (36 percent for black women and 18 percent for black men vs. 10 percent for white women and 5 percent for white men). By age 90, half of black women are poor. By contrast, blacks do not appear greatly

more likely to be sick than whites (8 percent for black women and 9 percent for black men vs. 6 percent for white women and 8 percent for white men). These poverty and sickness patterns—with blacks far more likely than whites to be poor, but only somewhat more likely to be sick—are qualitatively the same for each age group. Over the course of the nine years of follow-up, 12,614,287 individuals, or 47 percent of the sample, die. Nevertheless, all groups remain well-populated; the average time in the data is 6.8 years, and ranges from 6.3 years for black men to 7.0 years for white women.

We compare age- and cohort-specific morality for various groups in the sample to known levels from vital statistics, collapsing our data into ten-year age intervals to produce mortality estimates on populations equivalent to those captured in U.S. Center for Disease Control (CDC) estimates from 1993. Since the CDC life tables do not differentiate ages above 85, we focus on validating our mortality estimates in the 75-85 range. For each group defined by sex, race, and age interval in this year, our results are within a percentage point of the CDC estimates. These results therefore show excellent agreement with known mortality data, fostering confidence in the quality of our data.

Race and sex comparison

Figure 1 shows the unsmoothed and smoothed mortality hazards, their slopes (first derivatives), and their accelerations (second derivatives) by sex and race, from which Table 3 gleans each group's age at absolute deceleration (maximum first derivative) and relative deceleration (maximum second derivative). In the first two panels of Figure 1—those showing the hazards and first derivatives—the onset of absolute deceleration is marked with a cross symbol, and the lines are black for subpopulations that decelerate absolutely and grey for those

that do not. The third panel, showing the second derivatives, employs the same symbols and colors to mark the onset of *relative* deceleration.

We find evidence of absolute deceleration in three of the four populations. The first derivative peaks at age 87 for black men, 92 for white men, and 91 for black women. By contrast, for white women, the maximum slope occurs at age 94—the maximum age included in the first derivative measure. In other words, we find no evidence of absolute deceleration before age 95 for white women.

This qualitative ordering of the populations is the same when we turn to the age at maximum second derivative, though as expected, the ages are younger. Here, we do find a maximum before the oldest age (now 93) for all populations: for black men, at age 85; for white men and black women, 88; and for white women, at age 90. The gap between the other populations' ages at onset of relative deceleration is two to four years before their onset of absolute deceleration. This suggests the possibility that white women decelerate absolutely shortly after our data are truncated. However, visual examination of white women's second derivative suggests that their apparent relative deceleration may instead reflect our data happening to truncate during the valley of an oscillation in this derivative. By contrast, the other three populations' derivatives show a precipitous decline.

These results conform to the general prediction of mortality selection theory: it is the sex and race that have higher mortality for most of their lifespans—each sex's black population has higher mortality until their early 80s in these data (Elwert & Wrigley-Field, unpublished)—who decelerate first.

Race, sex, baseline health and poverty comparison

Next, we investigate subgroups defined by the combinations of sex, race, poverty, and baseline chronic disease. Since these subgroups are much smaller than the previously considered sex and race groupings, and second derivatives are correspondingly more fickle, we now focus solely on absolute deceleration. Figures 2 and 3 show each subpopulations' hazards and slopes, respectively, and Table 4 gives the age at maximum slope (onset of absolute deceleration) for each subpopulation. As before, the age at onset of absolute deceleration is marked with a cross, and black lines are reserved for subpopulations that decelerate, grey for those that do not. Analyzing these slopes, we find the opposite pattern as when we considered only race and sex: within each population, it is the lower-mortality subpopulations, if any, that decelerate.

The earliest deceleration is among non-sick, non-poor black men, who reach their maximum slope at age 86, while black men who are either sick or poor do not appear to decelerate. Non-poor black women and white men reach their maximum slope in their early 90s regardless of sickness status. By contrast, none of the four subpopulations of white women appear to decelerate before the end of follow-up: their highest slope is at age 94, the highest age at which we can reliably estimate it. In summary, the poor groups, in general, do not appear to decelerate (excepting sick, poor black women), and within the non-poor groups, deceleration order follows that in groups defined only by sex and race. Comparing the columns of Table 4 illustrates the pattern: within each race and sex—with the exception of white women, who do not decelerate in these data—whether a group decelerates is largely predicted by whether it is poor. Moreover, the pattern is in the opposite direction that what we might have expected on the basis of selection theory: although the poor presumably are more selected by mortality, it is the non-poor whose mortality decelerates.

Which apparent decelerations seem most meaningful, and which might be artifacts of truncating the data during a deceleration phase of a regular oscillation in the slopes? While we cannot answer this definitively, visual inspection suggests that we might be most skeptical of the apparent deceleration among sick black women and white men because the decline in slope among those groups is more nearly comparable to earlier periods of slope oscillation in those groups. On the other hand, the deceleration evident among the non-sick, non-poor—except among white women—is clearly distinct from earlier patterns in the slopes. For black men, it contrasts dramatically with the apparent sharp acceleration among the sick in the mid-90s ages. For white men, as well, the maximum slope of the non-poor, sick subpopulation at age 93 obscures a five-year period of nearly constant slope between ages 85-90 before a return to increasing slopes from ages 90-93. If patterns that deviate from constantly increasing acceleration are evidence of mortality selection, then we seem to have the strongest evidence of selection among the non-sick and, especially, non-poor subpopulations. This result is striking, since the conventional expectation that higher mortality populations are the most heavily selected would lead us to expect the reverse.

The slope analysis reveals one additional puzzle. All the sick subpopulations (except poor, sick black women) evince an apparently negative slope until age 69 or 70. These slopes rise sharply until the mid-70s, while the non-sick slopes rise much more slowly. We suggest that one possible explanation of these patterns is differential diagnosis. If chronic illnesses are under-diagnosed until the early 70s—several years after Medicare universalizes health care coverage at age 65—then those whose morbidity is particularly high might be disproportionately represented among the diagnosed. If those highly-morbid chronically ill have higher mortality than their relatively less morbid counterparts, then mortality will be exaggerated among the sick until

diagnoses catch up. An alternative explanation is that mortality selection among the sick has been strong enough by age 68 to induce a true hazard decline in all sick populations, which then reverses as new people become sick, diluting the robustness of the surviving sick subpopulations. However, we do not wish to assert this interpretation definitively as long as incomplete diagnosis remains a possibility.

Multi-Stage Mortality Selection

Traditional mortality selection theory predicts that, all else equal, higher-mortality populations should decelerate first, since they experience greater selective pressure, and thus, lose their frail members more quickly (e.g., Vaupel and Yashin 1985; Vaupel et al 1979). Our empirical results confirmed this prediction along lines of race and sex: higher-mortality African-Americans decelerate before whites, and men before women. When stratified by acquired conditions, namely poverty and chronic illness, however, these results produce an apparent puzzle. It is the lower-mortality populations, and in particular the non-poor, whose mortality decelerates earlier and farther.

Our proposed solution to this puzzle draws on a dynamic theory of group membership and mortality in which the path to death potentially consists of multiple transitions, first from membership in an advantaged group to membership in a disadvantaged group, and then to death. The conventional prediction that higher-mortality groups should decelerate first is predicated on mortality alone changing groups' frailty composition. This assumption is valid when groups are closed and frailty is assumed to be fixed (though see Elwert and Wrigley-Field, unpublished, for criticism of the latter assumption). When groups are entered by more means than birth and exited by more means than death, however, it makes sense to ask *who* enters (and exits) them. Our

argument is that the people who are likeliest to die may also be likeliest to become poor. If this is true, then transitions into poverty can tend to remove the non-poor population's frail members, just as mortality tends to remove all populations' frail. This is *multi-stage mortality selection*: some people start out neither sick nor poor and are 'selected into' illness or poverty before being 'selected into' death. Thus, we argue that a selection model applied to poverty-status groups on an analogy to fixed traits—one that assumes that frailty distributions change by mortality alone—predicts early deceleration among the poor, a prediction contradicted by our empirical results. By contrast, we will show that a model allowing movement into poverty to diminish the frailty composition of the non-poor group, and increase that of the poor group, makes our results sensible. Therefore, we argue, our results provide empirical support for the multi-stage model compared to what we call the fixed-trait selection alternative.

We will focus on poverty, since our empirical results were most dramatic when comparing the poor to the non-poor. For simplicity, we will also assume that, while some elderly people become poor, no one becomes non-poor at old ages. This assumption is unrealistic, since elderly Americans in fact commonly fluctuate in and out of poverty (Dodge 1995; Rank and Hirschl 1999), with as many as forty percent of the elderly poor exiting poverty within a year, although the majority of them remain "near-poor" (Jensen and McLaughlin 1997). We nevertheless assume lack of movement in this direction in order to highlight the sensitivity of deceleration order in a multi-stage selection model, even when the number of possible transitions is reduced to three: from non-poor to poor, non-poor to dead, and poor to dead. For the same reason, we assume that the risk of becoming poor is constant over age or is increasing at a constant rate (in one example to follow), although a non-monotonic function of poverty risk over age might be substantively plausible and would complicate this analysis.

Traditional Mortality Selection vs. Multi-Stage Mortality Selection

Earlier, we argued that the key assumption underpinning a mortality selection explanation of deceleration was that aging by and large is a monotone transformation of mortality risk, preserving the ordering of individuals' mortality risks: If one person has higher frailty than another person at one age, then she will also have higher frailty at the next age, should they both survive to it. This is a strong, though perhaps justified, assumption about both biology and social processes of health disadvantage. Multi-stage mortality selection adds an additional assumption about the associations of individuals' propensities: that mortality risk is positively associated with the risk of poverty as well. Therefore, when we refer to *frailty* in this discussion, we are referring to individuals' mortality risk, and we are assuming both that this risk is positively associated with their risk of becoming poor, and that the difference between the frailty of any two individuals has a constant sign (positive or negative) throughout their lives.

The hypothesis here is not merely that the poor are more likely to die than the non-poor, a fact that the conditions of poverty make unsurprising. Rather, it is that the people whose circumstances lead them to live their elderly years in poverty are people whose mortality risk would have been elevated compared to their peers, *even had they not become poor*. If traditional mortality selection theory assumes that some people are consistently disadvantaged in health across their life course, multi-stage mortality selection assumes that those people who are disadvantaged in health also tend to become disadvantaged economically. The multi-stage model therefore posits that, to an important degree, propensities for seemingly disparate kinds of disadvantage—in survival and in wealth—are clustered in the United States.

Previous work has compared mortality patterns across populations defined by acquired traits—including religion (Dupre 2006), poverty (Elwert & Wrigley-Field, unpublished), and health (Manton 1997)—in order to infer how those traits contribute to or mitigate against mortality selection. We contend that these comparisons can be problematic if they do not account for the correlations between individuals' risk of acquiring these traits and their risk of dying—that is, their frailty.

Recent demographic literature implies that such correlations may be pervasive, since it suggests that poverty in adult ages might stem from complex interactions between socioeconomic and health conditions earlier in life. In particular, demographers have increasingly directed attention at hypotheses that health early in life influences economic outcomes later, for example in Alberto Palloni's (2006) PAA presidential address (see also, e.g., Mulatu and Schooler 2002; Smith 2004; Case, Fertig, and Paxson 2005; Currie and Moretti 2005; Palloni et al 2009; but see also Warren 2009). It seems natural to imagine that early life health conditions whose effects are important enough to alter adult economic trajectories might also independently hasten death, whether or not this happens by means of the chronic medical conditions this study has employed. Indeed, the range of plausible candidates for mutual causes of poverty and mortality is very broad, and is in no way limited to 'biological' conditions; neighborhood effects are another example suggested by recent sociology (see Ellen, Mijanovich, and Dillman 2003 for a review; but see also Oakes 2003). To date, the hypotheses in the health selection literature have been difficult to prove empirically (Palloni, Milesi, and White 2010). To the extent that we need selection to explain mortality deceleration, and specifically that we need selection of the frail into poverty to account for this study's empirical results, these results constitute positive evidence for some of that literature's central concerns.

The multi-stage mortality selection model also draws on the observation by Lynch et al (2003) that the conventional prediction of earlier deceleration among higher-mortality groups can fail when other social processes than mortality have led the higher-mortality group to have more frail members at baseline. Here, we generalize that exception to a situation where the higher-mortality group continually gains new frail members. Finally, the multi-stage model draws on a small but important literature on dynamic selection models. Mohtashemi and Levins (1992) show that a race crossover can arise when blacks and whites have different rates of movement between subgroups that vary in mortality, even if the races have equal mortality within each subgroup. One assumption of their model is that the higher-mortality subgroup always experiences a greater total decrement rate than the lower-mortality subgroup. In this discussion, we will show that early deceleration among higher-mortality (poor) subgroups is possible whether or not that assumption holds. Our model also draws on the theoretical observation by Rogers (1992) that the effects of mortality selection on population composition can be mitigated when populations continually are entered as well as exited (his examples are unemployment and marital status). While Rogers frames this result as applying specifically to repeatable events, we show that this observation applies even to populations that are entered only once, as the poor subpopulation is assumed to be in our simplified model. The continued application of mortality selection models to groups defined by mutable traits like poverty or religion, without considering whether the acquisition of those traits varies with mortality risk, shows that demographers have not necessarily heeded Rogers's call. Our paper constitutes an empirical demonstration that, in order to generate accurate predictions, analyzing mortality selection across such groups may require explicit modeling of the dynamic movement of heterogeneous populations by several pathways, rather than mortality alone.

In what follows, we first make the multi-stage mortality selection model more explicit, showing that it can indeed accommodate earlier deceleration among non-poor subgroups. We then show, however, that the model is equally consistent with *late* deceleration among the non-poor, compared to the poor: movement between groups renders deceleration order sensitive to assumptions, even under the very restrictive conditions we outlined about the lack of movement from poor to non-poor and the stability of the poverty transition rates over age. We conclude by discussing the implications of this model sensitivity for attempts to understand who becomes poor, and who survives into the oldest ages. We argue that, since even the very simplified dynamic selection model discussed here can accommodate any deceleration order, the conventional demographic practice of comparing deceleration patterns across populations may not suffice to adjudicate between selection and senescence in populations that can be entered as well as exited. Yet since deceleration order *can* adjudicate between different selection models, our results provide conditional evidence for health selection.

Deceleration order in a multi-stage framework

From the perspective of the non-poor group, the generalization to a multi-stage selection model is straightforward because exiting to poverty can be treated just like exiting to death. The situation of the poor group, however, is more complex. First, in contrast to the non-poor group, whose members at any given age have all survived the risks of poverty and death for the same period of time, members of the poor group at any given age have not all experienced the same set of selective pressures, since they became poor at different times. Moreover, the relationship between the time spent in poverty and the selective pressures experienced is complex. For expositional simplicity, imagine that the poor are composed of just two groups—the

longstanding poor and the recent poor. The longstanding poor became poor early, suggesting that their initial ranks on average were among the frailest of their birth cohort; yet they also have long survived the elevated poverty risks associated with mortality, suggesting that they increasingly are whittled down to the robust. The recent poor, meanwhile, withstood the risks of poverty for longer and therefore are entering poverty from a non-poor group that is itself becoming increasingly robust through selection, but they have not yet been pared down by the additional mortality exerted by poverty. In this section, we illustrate how this complex set of selection dynamics can produce *either early or late deceleration* among the poor group compared to the non-poor.

How the non-poor can decelerate first—The fact that people become poor creates two new dynamics in the multi-stage model: a decrement dynamic for the non-poor group, and an increment dynamic for the poor. Either one alone, or the two together, can lead mortality to decelerate in the non-poor group before the poor.

The decrement dynamic is that frail individuals have multiple means of exiting the non-poor group. Even though mortality is higher among the poor, the total exit rate to poverty and death can be higher among the non-poor. This is the simplest extension of mortality selection's logic. Because this decrement dynamic increases selection in the non-poor group, it can lead that group's mortality to decelerate earlier.

The increment dynamic is that frail individuals can enter the poor group. Even if frail individuals exit the poor group (to death) more quickly than the non-poor, the addition of new frail members can offset this depletion, leading the poor group's average frailty to decline more slowly than the non-poor's. In that case, the number of people becoming poor at each age must be small enough that it does not lead the non-poor group to have a greater decrement rate, yet

large enough to overcome the poor's greater decrement rate, precluding mortality from making the poor group overwhelmingly robust. This is possible if the non-poor group is substantially larger than the poor (as, indeed, is the case for all of our populations except the oldest black women). The same set of frail people then can represent a small decrement from the non-poor, changing its frailty composition slightly, and a large increment into the poor, changing its frailty composition by quite a bit. The poor group would then include a small, extremely robust subgroup of longtime poor and a larger group of frailer recent poor. Whether or not the frail have a higher rate of exit among the non-poor than the poor, increments that increase the frailty composition of the poor group will lead its mortality to decelerate later than it would have. The continual entry of new frail could prevent or delay deceleration in the poor group, regardless of whether the non-poor group is losing its frail members quickly or slowly relative to its own size.

The increment dynamic is inherently more complicated than the decrement dynamic, since the changes in one group (the poor) reflect processes unfolding, not on it, but on another group (the non-poor) whose composition and size are also changing. Next we show that the consequences of this dependence between groups can be perverse.

How the poor can decelerate first—Paradoxically, the addition of the frailest non-poor can make the poor group less, not more, frail. This possibility arises out of the interaction of the two dynamics just discussed. Those who become poor later may be more robust than those who do earlier—even than those who do earlier and subsequently survive—and this can happen in two ways. First, if the risk of poverty increases with age at the individual level, then selection into poverty reaches deeper into the non-poor group, with a continually more robust subgroup of the non-poor becoming at risk for poverty. Second, however, even if the risk of poverty is constant

over age in individuals, those who enter poverty later may be more robust than those who did earlier. Since the non-poor group experiences its own compositional changes as it ages (due to the selective pressure of its two decrements), the newly poor are drawn from an increasingly robust group at risk. The starkest case of this is that the non-poor group simply runs out of its frailest members. Even if the size of the subgroup becoming poor then shrinks, its frailty will be lower than those who already are poor. The effect on the frailty composition of the poor group will depend on the details of those parameters in comparison with the preexisting poor group's size and average frailty.

In this latter case, if the newly poor are so robust that they lower the average frailty of the poor group as a whole (potentially precipitating mortality deceleration), it is because even the frailest non-poor are less frail than the average poor. Yet the *rate* of frailty decline may be faster among the poor, and may effect a deceleration in that group even if the non-poor group has not decelerated. Thus, it is possible in principle for intense selection of the *non-poor* group to cause deceleration in the *poor* group, even when the non-poor group has not itself decelerated.

To illustrate how the changing composition of the non-poor group and increasing individual-level poverty risk can change the composition of successive poverty cohorts, we offer the example of a single trait: ever-acquired wealth. Imagine that becoming poor in old age is a function of the total wealth one has acquired; in this model, individuals become poor by outliving their wealth. The first to become poor, then, will be those whose wealth was minimal. Imagine, too, that ever-acquired wealth is negatively correlated with mortality. Then these early poor are frail; that is, their low wealth stems from conditions that also raise their mortality, such as their lifetime employment, neighborhood, illnesses earlier in life, etc. As time passes, however, people whose total wealth was greater nevertheless run it out and become poor. These

new poor are worse off than their contemporaries who are avoiding poverty altogether. But they are better off than those who became poor before them: their ever-accumulated wealth is greater. The non-poor group is certainly richer than the poor, but the poor group nevertheless may be increasing sharply in ever-accumulated wealth, even though its current wealth is minimal. To the extent that lifetime total wealth is a stand-in for survival advantages, this is a scenario in which the poor group's mortality might decelerate because of its new membership.

Deceleration order across populations—Given the flexibility of the multi-stage selection model in predicting earlier deceleration of either group, it is striking that our empirical results so consistently find deceleration first among the non-poor. It seems particularly surprising that black women and white men have such similar deceleration patterns. These are the populations whose lifetime mortality is at roughly similar levels before the black-white crossover in their mid-80s, but the populations with the most divergent poverty experiences: black women are the most likely, and white men the least likely, to be poor. This is a puzzle for the multi-stage model, and we suggest that it should raise the specter of senescence-based explanations for selection. To explain early deceleration among the non-poor and non-sick, these explanations apparently would need to have the form that individuals' senescence can decelerate, but that the conditions of poverty and sickness somehow delay or preclude this.

The relationship between poverty and sickness also merits further thought. This is particularly so because we might expect that there are broadly two classes of ways to become poor in old age. One is to simply run out one's wealth, as in the example provided above. The other is to have a health crisis, or to have a spouse with a health crisis. These multiple means of becoming poor suggest that poverty or its lack may indicate quite different economic

backgrounds for the sick compared to the non-sick. A more realistic model of becoming poor or sick would consider the heterogeneity in causes of these conditions. This study suggests, however, that even if one could explicate a more realistic selection model, it may not be possible to generate predictions about mortality deceleration in the subgroups defined by these acquired traits.

Conclusion

This paper presents new evidence of mortality deceleration in the United States and provides a first investigation into mortality deceleration in groups defined by poverty and health. We draw on high-quality longitudinal data representing nearly the entire U.S. elderly population to estimate hazards that are essentially non-parametric. We find that among groups defined by race and sex, deceleration occurs first in groups whose mortality is highest: black men decelerate first, and white women last, if at all. When we further divide the population by poverty and sickness, this ordering across race and sex is maintained among the non-poor groups. Across poverty and sickness, however, we find a surprising result. It is among the non-sick and, especially, the non-poor that mortality decelerates.

This paper has therefore presented and offered a tentative solution to a demographic puzzle: Why does the mortality of relatively advantaged non-poor populations decelerate before that of the poor—even though mortality more heavily selects the poor? Our analysis showed that one way to account for this new result is to posit that the same people who are predisposed to die relatively early are also predisposed to become poor.

Behind this multi-stage mortality selection model lies a broadly applicable methodological point: when comparing old-age populations that can be entered as well as exited, we should

consider how the totality of exits and entries from those populations selects their membership and potentially distorts what is being compared. The argument of this paper suggests that this point applies equally to studies of mortality selection itself (for example, studies of deceleration or of mortality crossovers) as to other kinds of cross-population comparisons. As demographic datasets increasingly allow old-age mortality to be investigated alongside such mutable traits as socio-economic status, health conditions, and social statuses (such as religion or marital status), these results suggest that it may become increasingly important for demographers to model how mortality risk might covary with the propensity to move in and out of these statuses. The assumption from fixed-trait selection models that mortality alone alters the frailty distribution of a subpopulation may generate fundamentally misleading predictions.

However, the significance of the multi-stage selection hypothesis is more than methodological. In the multi-stage framework, selection is not just a technical nuisance to be controlled away, but a substantively meaningful reflection of the intensity and the consistency—across age and domain—of disadvantage within populations. To the extent that the multi-stage hypothesis can better account for our results than conventional (fixed-trait) mortality selection, then, this paper offers evidence for inequality within U.S. subpopulations that is deep, lasting, and enacted in multiple contexts of social life.

Our analysis of multi-stage mortality selection has cast doubt on the link between selection and predictable mortality patterns for groups that can be entered, a link that was supposed to be the best way to anchor those patterns to the competition between selection and senescence hypotheses. We have suggested that this link can be broken in both directions. On one hand, we have shown theoretically that selection patterns that have in common the key fact of associated risks for poverty and death nevertheless can produce opposite deceleration order. Conversely, our

empirical results have also suggested that very different selection patterns (such as the differential selection of black women and white men into poverty) nevertheless can produce surprisingly similar patterns of deceleration. These facts make deceleration order essentially unpredictable from a multi-stage selection model without detailed information about the heterogeneous risks of moving in and out of each population. Taken together, these results raise a question as to whether mortality deceleration, or any aggregate mortality patterns, really can adjudicate between realistic selection models and senescence models.

An alternative might be to begin to open the black box by directly examining the transitions in and out of populations. Direct investigation of how important mortality risks *become* distributed across subpopulations, by selective mortality and selective entry into disadvantaging social categories, can begin to tell us how biological and social inequalities interact to produce divergent population-level mortality. Such investigation may also provide a more substantively-grounded basis for choosing among complex selection models and provisionally bounding their parameters, possibly allowing population-level mortality patterns to be leveraged in a new way to answer the core questions that mortality deceleration studies seek to address: What forms of biological and social inequality within human populations are so consistent and so intense that the survival of only the most advantaged alters the whole population's mortality curve? And does the aging process ever really slow down?

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TABLES

TABLE 1. Four possible states of the hazard, defined by its derivatives.

	Mortality hazard	Mortality slope	Acceleration	Third derivative
Gompertz mortality	Increasing	Increasing	Increasing	Positive
Relative deceleration	Increasing	Increasing	Decreasing, but positive	Negative
Absolute deceleration	Increasing	Decreasing, but positive	Negative	
Mortality decline	Decreasing	Negative		

TABLE 2. Descriptive statistics

Sample size

	Women	Men	TOTAL
White	15,796,197	10,562,056	26,358,253
Black	1,427,226	872,696	2,299,922
TOTAL	17,223,423	11,434,752	**28,658,175**

Person-years

	Women	Men	TOTAL
White	113,000,000	71,500,000	
Black	9,990,616	5,590,898	
			200,081,514

Avg, follow-up	Women	Men
White	7	6.6
Black	6.9	6.3

6.8 years overall average follow-up (s.d. = 2.90)

Observed mortality: Yearly hazard (unsmoothed) at selected ages

		70	75	80	85	90	95
Men	Black	0.054	0.073	0.103	0.146	0.200	0.255
	White	0.046	0.067	0.103	0.160	0.240	0.328
Women	Black	0.024	0.036	0.055	0.087	0.131	0.187
	White	0.019	0.031	0.051	0.090	0.157	0.257

(TABLE 2 continued.)

Observed mortality: Yearly hazard (unsmoothed) at age 85

		Non-sick	Sick		
		Non-poor	Poor	Non-poor	Poor
Men	Black	0.097	0.127	0.223	0.252
	White	0.115	0.187	0.252	0.346
Women	Black	0.053	0.070	0.150	0.166
	White	0.058	0.101	0.153	0.194

TABLE 3. Age at onset of mortality deceleration

	Men		**Women**	
	Absolute deceleration	Relative deceleration	Absolute deceleration	Relative deceleration
Black	87	85	91	88
White	92	88	none	90

TA BLE 4. Age at onset of mortality deceleration: Absolute deceleration

		Non-Poor	**Poor**
Black Men	**Non-Sick**	86	None
	Sick	None	None
White Men	**Non-Sick**	92	None
	Sick	93	None
Black Women	**Non-Sick**	91	None
	Sick	91	91
White Women	**Non-Sick**	None	None
	Sick	None	None

FIGURES

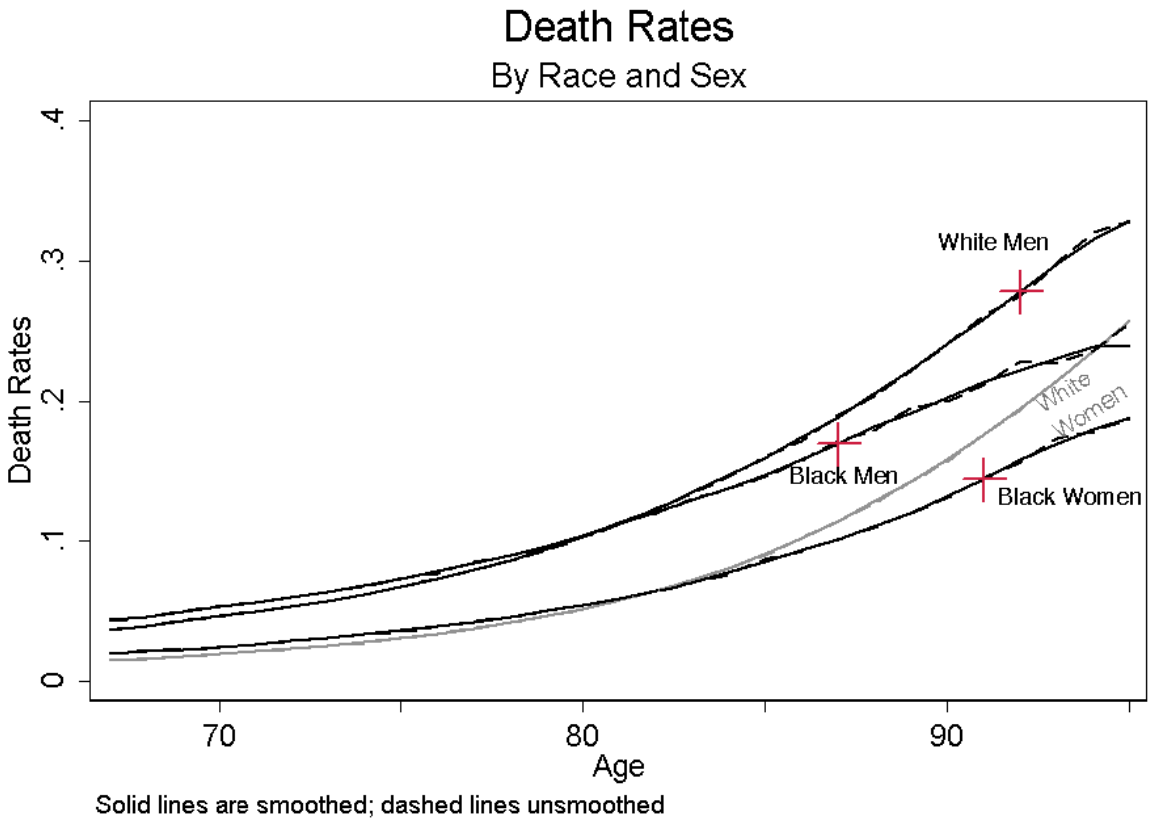
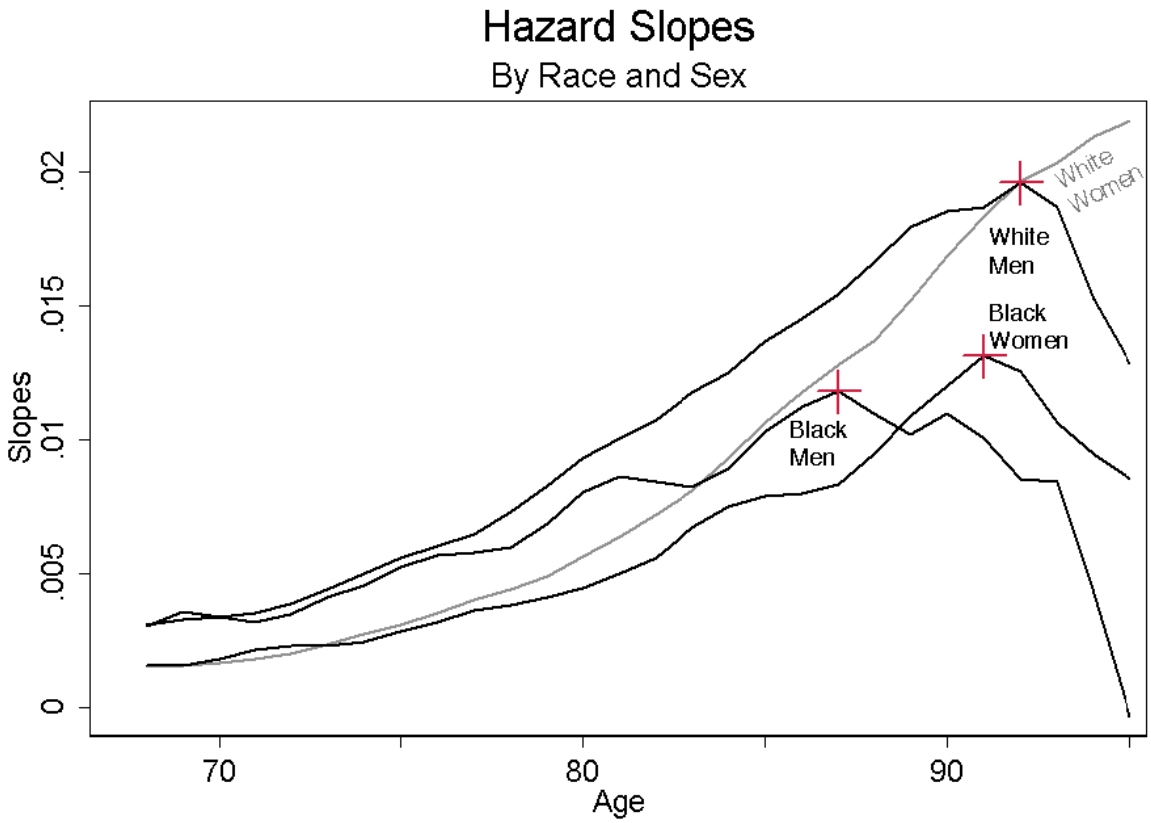


Figure 1: Panel A



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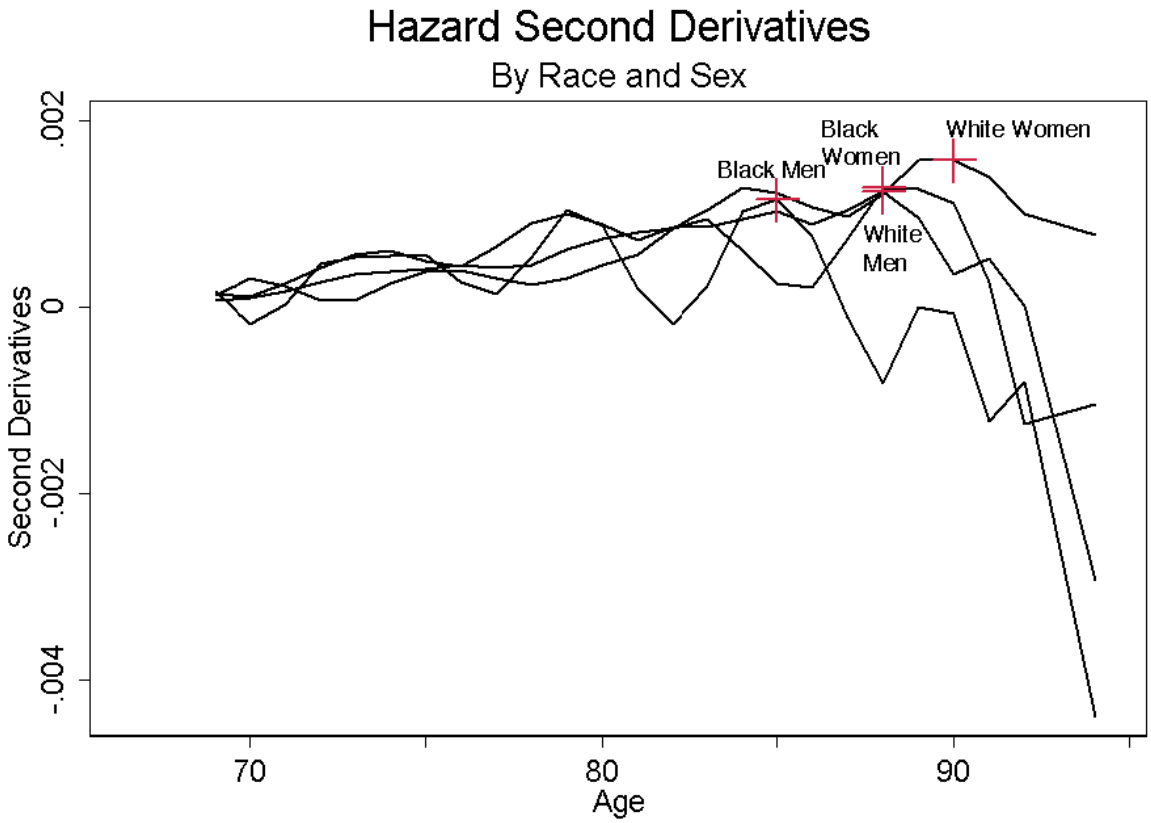

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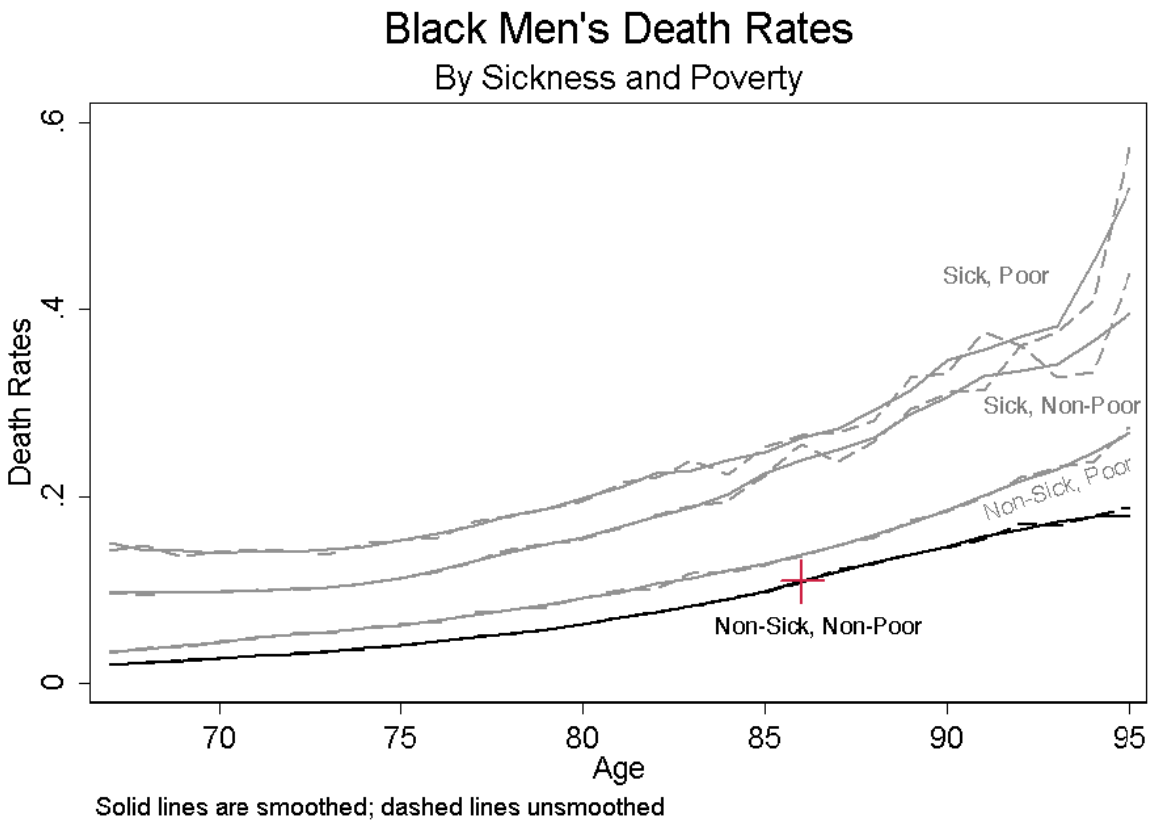

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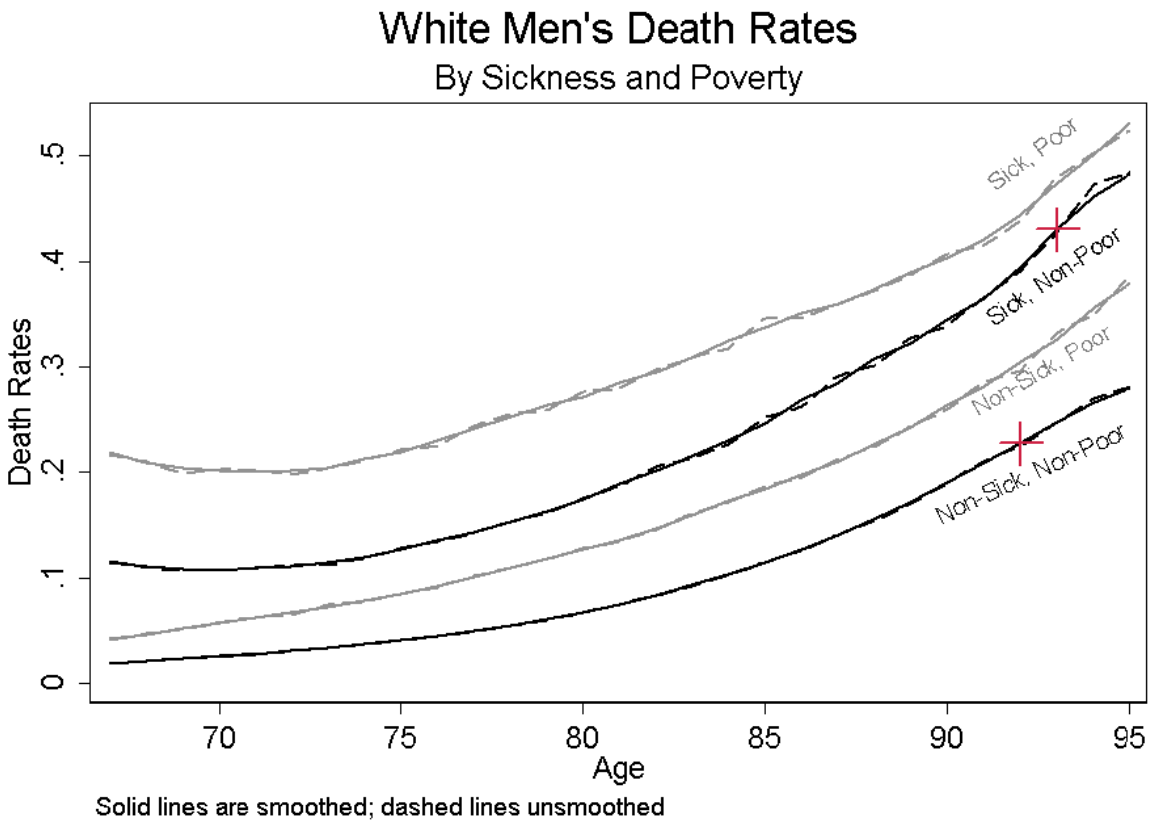

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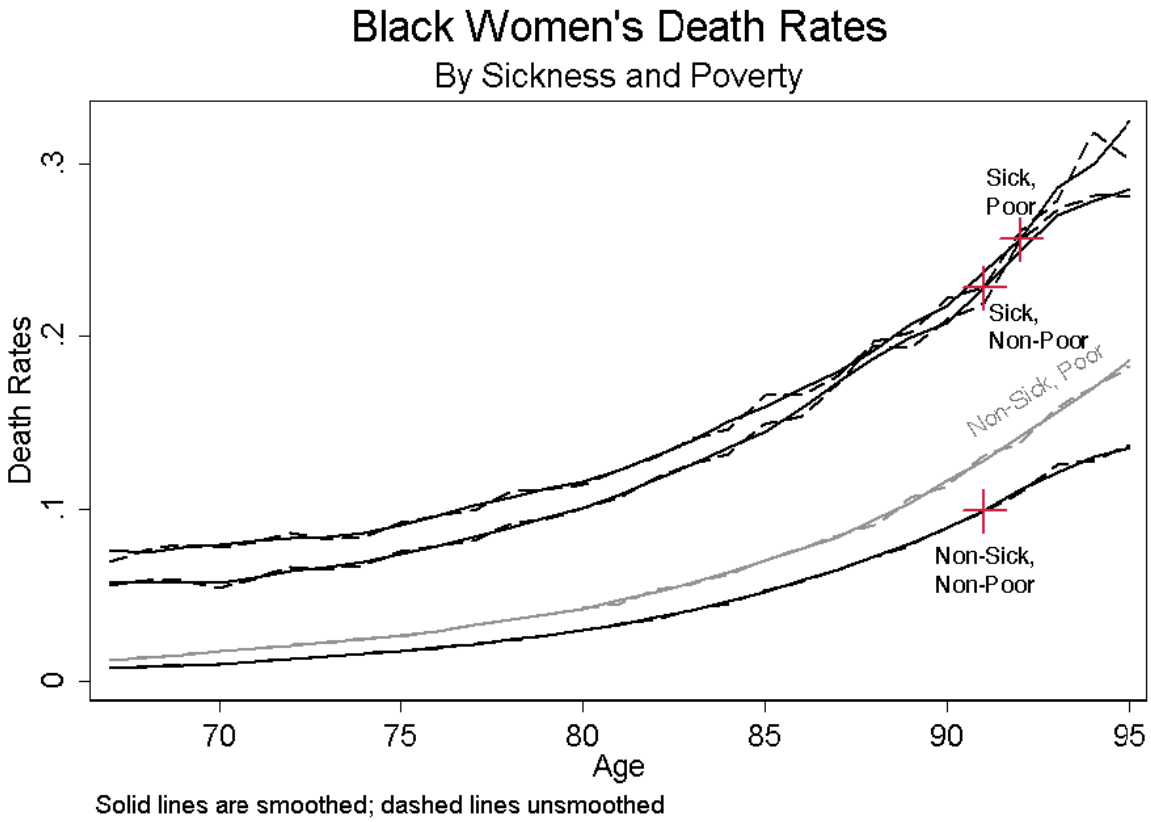

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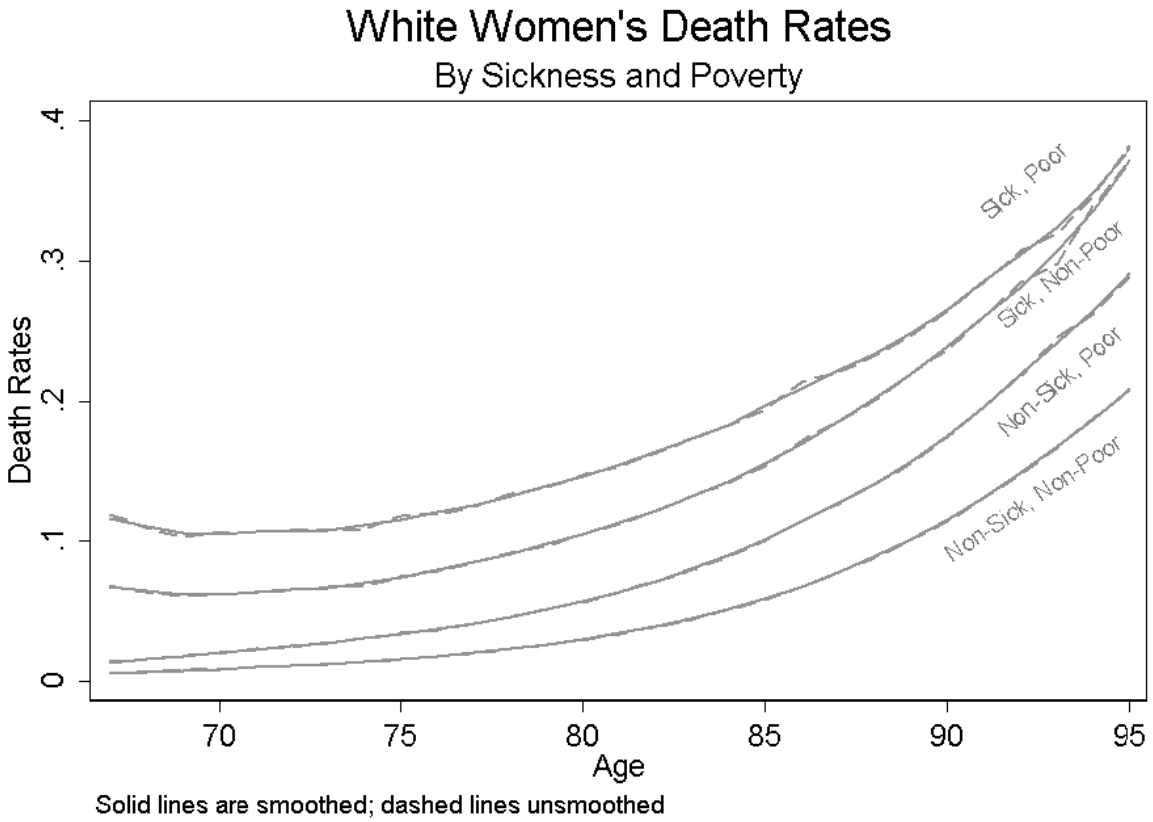


Figure 2: Panel D

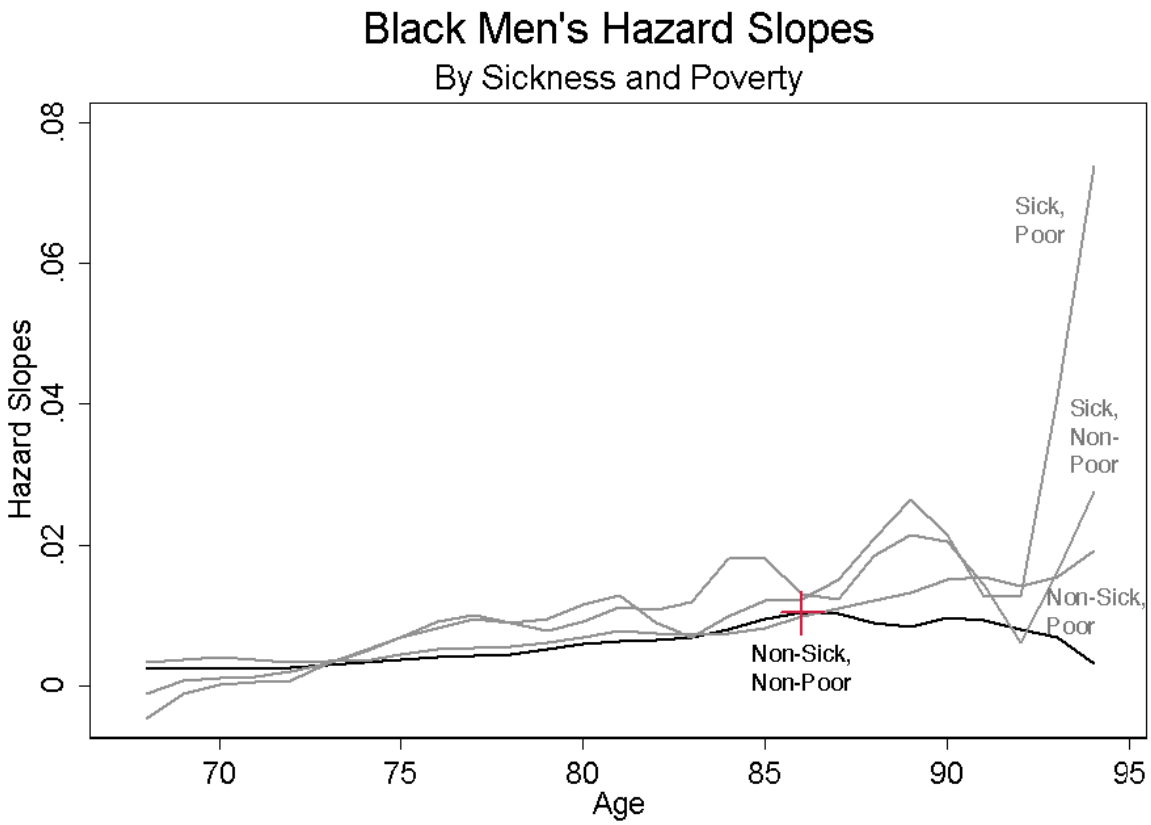
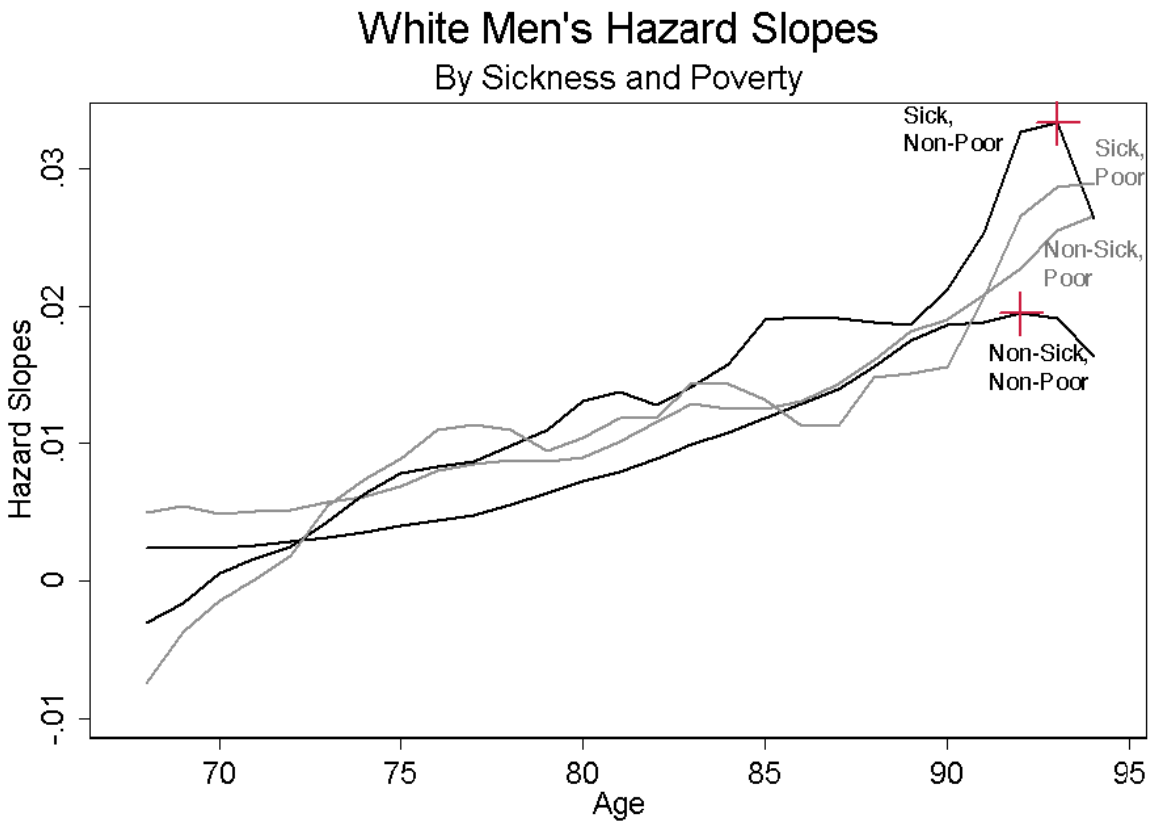


Figure 3: Panel A



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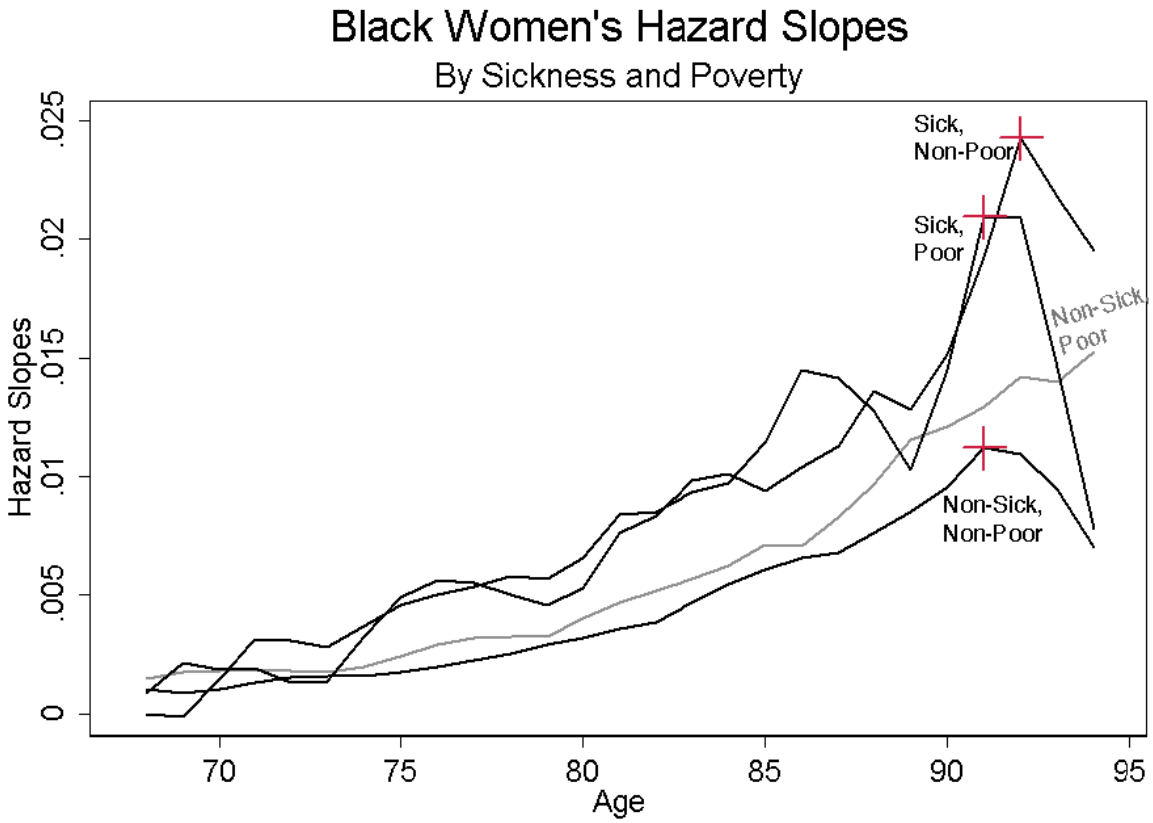

Figure 3: Panel C

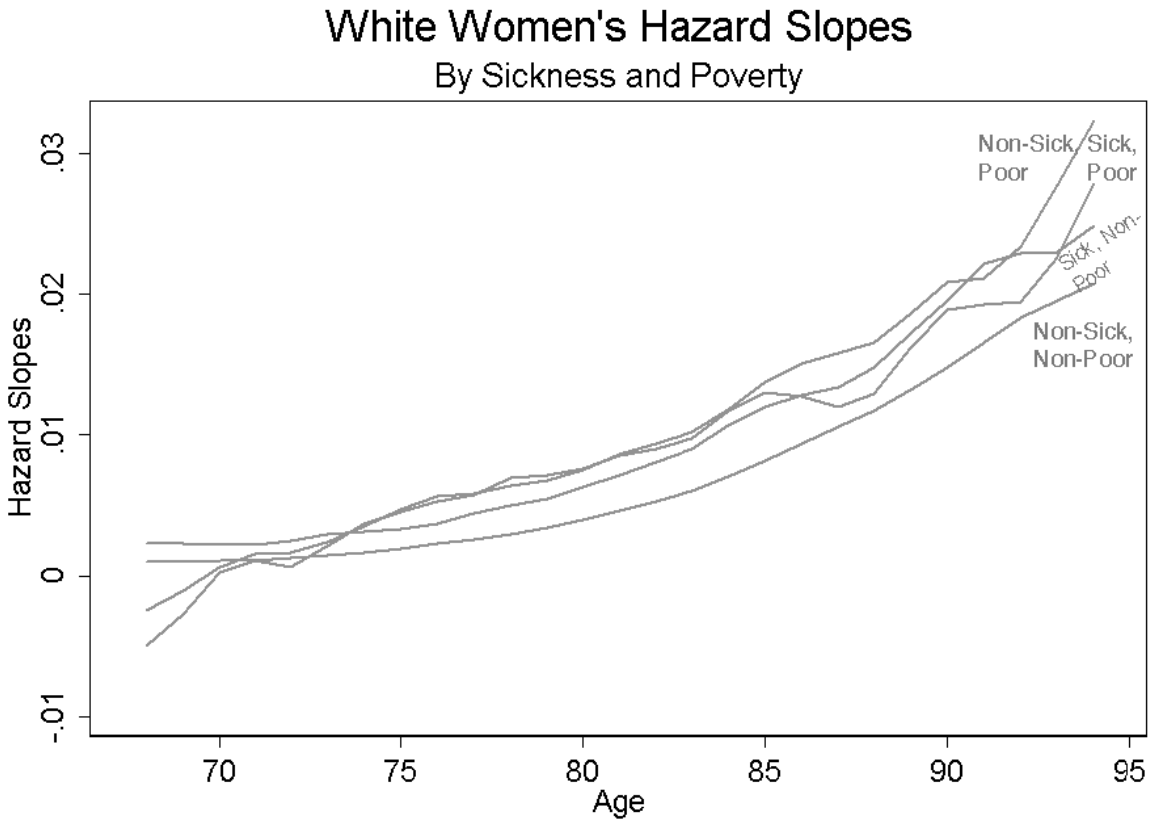

Figure 3: Panel D