Demography (2012) 49:773–796
DOI 10.1007/s13524-012-0107-y

Uncrossing the U.S. Black-White Mortality Crossover: The Role of Cohort Forces in Life Course Mortality Risk

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Published online: 23 June 2012

Abstract In this article, I examine the black-white crossover in U.S. adult all-cause mortality, emphasizing how cohort effects condition age-specific estimates of mortality risk. I employ hierarchical age-period-cohort methods on the National Health Interview Survey-Linked Mortality Files between 1986 and 2006 to show that the black-white mortality crossover can be uncrossed by factoring out period and cohort effects of mortality risk. That is, when controlling for variations in cohort and period patterns of U.S. adult mortality, the estimated age effects of non-Hispanic black and non-Hispanic white U.S. adult mortality risk do not cross at any age. This is the case for both men and women. Further, results show that nearly all the recent temporal change in U.S. adult mortality risk was cohort driven. The findings support the contention that the non-Hispanic black and non-Hispanic white U.S. adult populations experienced disparate cohort patterns of mortality risk and that these different experiences are driving the convergence and crossover of mortality risk at older ages.

Keywords Mortality · Race · United States · Age-period-cohort · Crossover

Introduction

Analyzing mortality risk of the U.S. older adult population has proved difficult for some time. Sparse data in older-old (85+) age groups frequently preclude reliable estimates of mortality risk, and surprisingly low estimates are often found for subpopulations with comparatively high mortality risk during early and middle life. Such findings are central to an ongoing debate as to the existence of a "crossover" in the mortality risk of the U.S. non-Hispanic black and non-Hispanic white populations. A mortality crossover occurs when the higher age-specific mortality risk of one subpopulation (e.g., non-Hispanic black, henceforth referred to as "black") converges

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with and then becomes lower than the age-specific mortality risk of another subpopulation (e.g., non-Hispanic white, henceforth referred to as "white"). In the United States, a black-white mortality crossover has been repeatedly found to exist at around age 85 for both men and women (Arias 2006; Johnson 2000; Kestenbaum 1992; Lynch et al. 2003; Parnell and Owens 1999).

Two principal hypotheses have been advanced to explain crossover phenomena. The first emphasizes the combined effects of population heterogeneity in susceptibility to mortality (often times referred to as "frailty") within subgroups and selective mortality across the life course between these subgroups. That is, the two subpopulations' compositions of frail members differ across ages because one subpopulation is subjected to higher age-specific mortality risk across the life course (Lynch et al. 2003; Manton and Stallard 1981; Manton et al. 1984; Nam 1995; Vaupel et al. 1979; Vaupel and Yashin 1985). Upon reaching older age groups, the population that experienced higher mortality risk across the life course will be composed of relatively more robust members at these advanced ages, and the population that experienced lower mortality risk across the life course will have retained more frail members at advanced ages. The second hypothesis emphasizes how poor data quality biases estimates of older-age mortality risk (Coale and Kisker 1986; Preston and Elo 2006; Preston et al. 1996; Preston et al. 1999). This idea suggests that the mortality crossover is a product of age misreports, unmatched or uncounted deaths, and/or other data inaccuracies. After data problems are accounted for, convergence of mortality risk is delayed to much older ages or eliminated altogether (Lynch et al. 2003; Preston et al. 1996).

Although both explanations have been supported by existing research, arguments are developed herein that point to the need to consider *cohort* effects to better understand the processes driving mortality crossovers. This idea extends the heterogeneity argument considering that disparate cohort processes affect the life course mortality risks of the U.S. black and white populations. As Ben-Shlomo and Kuh (2002:290) affirmed, "The individual life course is embedded in the sociohistorical and biocultural context" of populations and thus "changing individuals must be studied in a changing world." The contexts of black and white America differed tremendously across the twentieth century, and these differences shaped life course patterns of these populations' mortality risks. Consequently, the enduring effects of different black and white sociohistorical contexts should have significant influences on older age mortality risk of these populations.

I use the 1986–2006 National Health Interview Survey Linked Mortality Files (NHIS-LMF) to analyze the mortality experiences of the U.S. older adult black and white populations. These analyses are conducted in several steps. First, both single-year and five-year age-specific estimates of black and white male and female mortality risk are calculated, using the entire NHIS-LMF 1986–2006 data set. Second, the single-year estimates are recalculated after adjusting the samples to improve data quality at advanced ages. Finally, I employ recently developed hierarchical age-period-cohort (HAPC) cross-classified random-effects models (CCREM) on the five-year data to simultaneously estimate effects of age, period, and cohort processes on black and white adult mortality rates in the United States between 1986 and 2006.

Employing these methods allows me to factor out the possible confounding effects of both period and cohort processes, preserving unadulterated average age effects on black and white men's and women's adult mortality rates (Yang and Land

2006). I discuss these specific adjustments and their implications for examining the U.S. black-white mortality crossover, as well as advancing a cohort-specific life course perspective for studying old-age mortality (Riley 1987). Indeed, consistent with Riley's (1973) notion of a "sociology of age," I argue that only by accounting for changing sociohistorical contexts across cohorts can the life course framework truly advance our understanding of age-specific mortality risk. This article supports this contention with regard to old-age mortality risk in the United States by illustrating that the black-white U.S. mortality crossover is likely due to disparate cohort patterns of U.S. black and white mortality across the twentieth century.

Background

Mortality crossovers have been documented to occur in human populations in many countries and under many mortality schedules (Coale and Kisker 1986). Such crossovers have also been demonstrated to exist in nonhuman populations in similar ways (Nam 1995). In the United States, Arias (2006) documented black-white crossovers in the probability of death at age 88 for men and age 87 for women in 2003, which are consistent with a long literature on the black-white mortality crossover in the United States. Why such crossovers are repeatedly found in estimates of old-age mortality risk from many data sources is still debated. Preston and Elo (2006) definitively stated that mortality crossovers are artifacts of poor data quality. Other researchers, however, continue to investigate the effects that heterogeneity and mortality selection have on the aggregated estimates of mortality rates (Eberstein et al. 2008; Lynch et al. 2003).

Heterogeneity-based explanations of the U.S. black-white crossover in age-specific mortality rates highlight the changing composition of populations across the life course. These explanations point to the fact that aggregate rates of mortality do not reflect actual risks of death for all members of a population at a specific age. Rather, aggregate rates are average assessments of mortality risk, reflecting the contributions of many frail and robust subpopulations at that age during a specific period of time. The degree of frailty or robustness of these subpopulations, in turn, is determined by the history of morbidity and mortality risk endured by their members across their life courses. Thus, proponents of the heterogeneity explanation of mortality crossovers place a great deal of emphasis on disparate mortality selection across the life course to explain old-age mortality risk, and thus old-age mortality crossovers (Lynch et al. 2003; Vaupel et al 1979; Vaupel and Yashin 1985). Eberstein et al.'s (2008) recent findings most forcefully suggested the plausibility of the heterogeneity argument. By examining black and white old-age mortality rates by cause of death, the authors demonstrated that convergence and crossovers of mortality rates operate primarily through heart disease–related mortality. Yet, for some causes of death, such as diabetes and malignant neoplasms, no black-white crossover in mortality rates was found. Because any data bias(es) would have to vary "by cause of death in a peculiar manner," the authors concluded that "the central mechanism for the patterns seems to be heterogeneity in frailty" (2008:226).

It is important to note that the age at which black-white mortality crossovers are found to occur in the United States population is drifting upward (see Fig. 1). Data from the early 1960s recorded male and female crossovers between white and nonwhite

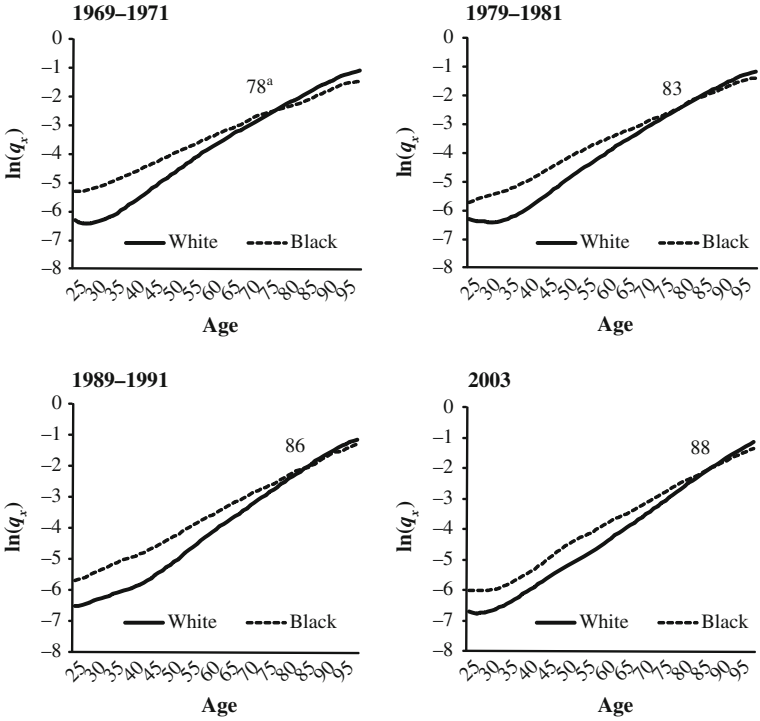

Fig. 1 Logged age-specific mortality risk of U.S. black and white male populations across time. Data are from the National Center for Health Statistics (NCHS) (http://www.cdc.gov/nchs/products/life_tables.htm). [a]Denotes the age at black-white crossover

populations to have occurred at ages 75 and 77, respectively (Kestenbaum 1992). About 10 years later, the NCHS life table for 1969–1971 recorded the black-white crossovers to occur at ages 78 and 80 for men and women, respectively. (Because of space limitations, women's crossovers are not shown in Fig. 1.) Recent official estimates of U.S. mortality risk have found male and female crossovers between the white and black populations to occur at ages 88 and 87, respectively (Arias 2006).

The fact that the ages at which the crossover in mortality risk occurs are increasing suggests that cohort and/or period effects should be considered when examining such phenomena. Cohort and period effects could capture secular mortality trends stemming from improving data quality at older ages, temporal changes to the effects of heterogeneity, or both. I argue that differences between black and white cohorts' cumulative exposures to changing health and mortality conditions during the twentieth century are the primary cause of the increasing age at which the black-white mortality crossover occurs. My argument extends the heterogeneity perspective to consider how mortality selection in the white and black populations is changing.

The Importance of Disparate Cohort Exposures

In 1965, Norman Ryder highlighted the role of intercohort differentiation in the study of social change. Ryder rightly emphasized that each cohort moves through history as "a flow of person-years," with a "distinct composition and character reflecting the

circumstances of its unique origination and history" (Ryder 1965:845). Matilda White Riley (1973) built on Ryder's work and pioneered a "sociology of age," which advanced a perspective of health and aging that embedded the life course in a cohort understanding of aging. For the most part, mortality research in the United States has moved away from these traditions. Compared with age and period effects, cohort effects have been given much less attention in studies of the variations in U.S. adult mortality risk. Even in analyses highlighting temporal changes in mortality risk, cohort effects have been largely omitted in favor of presenting changes in terms of (perceived) period phenomena (Meara et al. 2008). For example, official U.S. life tables are most commonly offered in period format, projecting life expectancy of "synthetic" cohorts across their ages. Although period changes might have influenced early temporal shifts in mortality risk across the epidemiologic transition, recent temporal patterns of U.S. adult mortality have been overwhelmingly driven by cohort phenomena (Yang 2008).

The omission of cohort effects from life course theories of health and mortality risk has limited the field's understanding of the relationships among race/ethnicity, age, and mortality. Most life course research on the relationship among race/ethnicity and health and mortality focuses solely on age effects. That is, the bulk of the literature is concerned only with the ways in which disease, disability, and mortality risk across age differ between racial/ethnic groups. Only minimal efforts have been made to understand how the aging process has changed—and continues to change—across cohorts (Lauderdale 2001; Montez and Hayward 2011). Yet, as Riley's work has emphasized, there is no single "life course" to speak of. Rather, each cohort experiences a distinct life course shaped by the confluence of biological aging effects and that cohort's unique experience of history. Observing that members of each cohort share a "common location" in history, Riley's sociology of age reminds researchers that "the life course is not fixed, but widely flexible" and that "cohorts can age in different ways" (1973:39, 43). Indeed, U.S. cohorts have differed in their exposure to the benefits of medical inventions, public health measures, and improvements in nutrition, as well as in their lifetime exposures to risk factors, such as years spent smoking. Further, racial/ethnic differences in U.S. cohorts' exposures to health improvements and risk factors will invariably influence morbidity and mortality risks for cohorts of these races/ethnicities across their respective life courses.

Recognizing cohort variation in life courses is central to understanding the black-white crossover in mortality. Chronic disease epidemiology has long noted the effect of early life exposures on later life susceptibility to disease and mortality, and social and health demographers have recently been paying closer attention to the influence of early life malnutrition (Fogel 2005; Kuh and Ben-Shlomo 2004) and inflammatory infection (Finch and Crimmins 2004) on subsequent age mortality risk. These effects, as well as the cumulative effect of cohort exposures to other risk factors (e.g., person-years spent smoking (Preston and Wang 2006)) and/or health-enhancing knowledge and technologies (e.g., proportion of cohort inoculated against infectious diseases) across the life course, are important at shaping disparate "cohort morbidity phenotypes," which affect cohorts' mortality risk across age (Finch and Crimmins 2004).

The role that race/ethnicity has played in shaping cohort-specific life course experiences of health and mortality in the United States is not fully understood. But if life course risk factors of morbidity and mortality have indeed been changing across

cohorts, those changes have likely differed profoundly for U.S black and white cohorts. This hypothesis largely stems from evidence in several areas of study. First, the life course literature has demonstrated that early life conditions, educational attainment, social support, and other resources that differ by race/ethnicity significantly and substantively condition mortality and morbidity risk across age (Blackwell et al. 2001; Hayward et al. 2000; Ross and Wu 1995). Second, research has shown that access to and use of new health-enhancing or health-protecting knowledge, practices, and/or technologies are to a large degree conditioned by social position and personal resources (Glied and Lleras-Muney 2008; Link 2008; Link and Phelan 1995; Pampel 2005). Indeed, the adoption of "new health-enhancing knowledge and technology" is highly variable, and thus "come to have effects on population health through a thick distribution of social, political, and economic circumstances" (Link 2008:370). Consequently, racial/ethnic cohort differences in education and other socioeconomic variables associated with risk factors, access and utilization of healthcare technologies, social support, and other health-related variables suggest race/ethnicity–based differences in cohort effects of mortality risk as well.

Also, U.S. cohorts experienced dramatic change in disease patterns and risk factors across the twentieth century, in turn affecting mortality risk across the life course (Manton et al. 1997; Yang 2008). This shift was important for both all-cause mortality risk and for specific causes of death, such as heart disease, stroke, and respiratory diseases (Jemal et al. 2005; Meara et al. 2008; Yang 2008). These changes are incredibly significant because the composition of deaths by specific causes directly affects age patterns of mortality risk, and also because the changes are likely to have differed by sex and race/ethnicity (Eberstein et al. 2008; Manton et al. 1997). Thus, how U.S. white and black cohorts lived through these compositional shifts in causes of deaths across the twentieth century should affect each cohort's age patterns of mortality risk, even at older ages.

Understanding both of these trends—changes in racial differences in socioeconomic resources and living conditions on one hand, and compositional changes in causes of death on the other—in terms of shifting cohorts' exposure times across the life course is essential to analyzing the black-white crossover in mortality risk. This idea is briefly specified via the following points. First, regardless of race/ethnicity, cohorts born in the earliest years of the twentieth century—prior to the advent of vaccines, penicillin, and sulfa drugs (Jayachandran et al. 2010); public health campaigns (Cutler and Miller 2005), smaller families; and improved nutrition (Fogel 2005; Fogel and Costa 1997)—are likely composed of relatively larger proportions of robust members at the oldest-old age groups than are subsequent cohorts born in the mid-twentieth century. This is due to the earlier cohorts' elevated exposures to harsh early life conditions, as well as disparate exposure to subsequent health-enhancing knowledge and technologies. Table 1 contains the general timing of a select number of important advances in nutrition and public health efforts, as well as proportions of black and white cohorts born into select living conditions associated with elevated disease and mortality risk (Blackwell et al. 2001; Ewbank 1987; Hayward et al. 2000; Montez and Hayward 2011). These measures of household conditions were estimated using 1900–1940 Integrated Public Use Microdata Series (IPUMS) of U.S. Census data (Ruggles et al. 2010). Consistent with the points presented earlier, two themes can be taken away from Table 1. First, compared with subsequent birth cohorts, black

Table 1 Timing of select advances in nutrition, public health, and medical technologies as well as U.S. black and white cohort household characteristics

	1900s	1910s	1920s	1930s	1940s
		Dietary Guidelines	Refrigeration	Chlorination of Water	Vitamin D Fortification
		First Public Health School	Cod Liver Oil	Vitamin B6	Vitamin B3 Fortification
		Malaria Control	Irradiation of Milk	Food Relief Programs	Modern Sewage Treatment
			Iodization of Salt	Sulfa Drugs	Penicillin
			Sheppard-Towner Maternity and Infancy Protection Act	Rural Sanitation	Rural Sanitation
Black, 6+ Family Members	53.54	53.63	53.75	55.02	59.80
White, 6+ Family Members	45.58	42.36	42.55	38.44	34.80
Black, Farm	50.60	49.36	58.03	46.76	47.03
White, Farm	39.27	32.12	32.06	26.59	25.51
Black, South	89.55	90.41	85.67	79.24	77.75
White, South	27.94	30.08	28.48	28.25	29.25
Black, Rented	80.85	78.95	82.31	81.95	83.23
White, Rented	60.51	63.69	64.20	65.68	69.38

Note: Household statistics are percentage of infants born into said condition and are calculated using IPUMS data, 1900–1940.

and white cohorts born at the turn of the twentieth century endured the difficulties of their childhoods without the benefits of widespread public health campaigns (1920s–1940s), nutritional knowledge (1920s–1940s), penicillin (1942), or other knowledge and technologies to improve or protect health. Second, the black-white differences in harsh household conditions during childhood are significantly smaller in these early cohorts than are the differences in cohorts born later in the century.

These differences are important because a great deal of evidence suggests that early life bouts with infection and inflammation (Finch and Crimmins 2004), malnutrition (Ben-Shlomo and Kuh 2002; Fogel 2005), and other childhood hardships (Montez and Hayward 2011) significantly raise mortality risk later in life. At the most advanced ages, we should presume that only the most robust members of birth cohorts born in the early twentieth century, irrespective of race/ethnicity, could survive to these advanced ages after having endured a lifetime full of exposure to such hardships. In short, mortality selection should be the greatest among these early cohorts. Thus, at the oldest-old age groups, I hypothesize that the heterogeneity of the black and white populations in the earliest birth cohorts are quite similar. For cohorts born later, one should observe greater difference in older age heterogeneity between the racial/ethnic groups, and thus greater black-white differences in mortality risk as well. This is because although these latter cohorts benefitted from improved early life conditions, the importance of early and mid-adulthood became relatively more

important in shaping older-age mortality risk. As a result of increased variation in socioeconomic status, risk factors, and access to and use of health provisions, greater heterogeneity is preserved into older ages.

In lieu of these trends, researchers should revisit Riley's and Ryder's work on cohorts, move beyond one said "life course," and highlight cohorts' varying experiences in aging, health, and mortality. When evaluating the black-white crossover in age-specific mortality risk in the United States, we must consider the possibility that risk of death reflects more than the biological process of aging. It also reflects the disparate life course experiences of black and white cohorts that compose those older age groups. In this article, I account for disparate cohort effects of the black and white populations to see whether the black-white crossover in U.S. mortality risk can be explained by cohort phenomena.

Analytic Strategy

I exploit the unique design of the National Health Interview Survey-Linked Mortality Files (NHIS-LMF) 1986–2006 to compare estimates of age-specific mortality risk of the black and white male and female populations in the United States. The analyses proceed across three steps in the following way. First, I compare the single-year age-specific mortality risks for black and white male and female respondents in the NHIS-LMF to determine the ages at which the sex-specific mortality crossovers occur in these data. Mortality risk is also estimated from models by using five-year age groupings. These five-year models are estimated to derive baseline comparisons for subsequent age-period-cohort analyses. I then make several adjustments to the single-year data to reduce the likelihood of poor data quality bias, especially amongst the older age groups. These adjustments are fourfold: (1) NHIS respondents who relied on proxy reports were deleted from the sample; (2) respondents' reported ages at time of survey were replaced with their calculated ages at time of survey to increase accuracy of age estimates at both the time of survey and the time of censoring or death; (3) respondents with missing values of educational attainment were deleted from the sample; and (4) respondents over the age of 75 at the time of the survey are deleted from the sample. Models estimating single-year age-specific mortality risk for the black and white male and female samples were then rerun on these restricted samples to determine whether the crossover stemmed from poor data quality bias. As a final step, I conduct age-period-cohort analyses of adult mortality rates to account for disparate cohort and period effects in the age-specific mortality estimates. Specifically, I use HAPC-CCREM, using five-year age, five-year period, and five-year cohort groupings to estimate age-specific mortality rates for the black and white male and female samples while controlling for both period and cohort effects (Yang and Land 2006, 2008).

Data

I use 19 cross-sectional waves of the National Health Interview Survey (NHIS) 1986–2004, linked to the National Death Index via the Multiple Cause of Death (MCD) file, through the end of 2006 (NCHS 2010). The resulting National Health Interview

Survey-Linked Mortality Files (NHIS-LMF) 1986–2006 data are a unique combination of repeated cross-sectional survey waves coupled with longitudinal follow-up of individual respondents' yearly mortality status through December 31, 2006. The NHIS uses a multistage probabilistic sampling design, and respondents of the NHIS are matched to the MCD mortality files, using a 14-item identification scheme. Respondents not eligible or not reliably matched are dropped from the analyses, and the use of analytical weights makes results from the NHIS-LMF representative of the noninstitutionalized U.S. adult population, aged 25 to 99.

The NHIS-LMF data have several unique advantages for studying the U.S. black-white mortality crossover. First, ages are self-reported by respondents at the time of the NHIS survey. Therefore, unlike death certificates used in U.S. official estimates of mortality risk, the ages at time of death in the NHIS-LMF data are not reported by next of kin or some other impersonal source. Also, because respondents in the NHIS-LMF can be tracked for up to 21 years of subsequent mortality risk, the oldest-old (aged 85+) cases in the NHIS-LMF 1986–2006 are captured at younger ages at the time of their survey, increasing confidence in age reports for the oldest-old cases. For instance, a respondent aged 74 years at time of the 1987 NHIS survey might be matched to a death record in 2003, meaning that this respondent was about age 91 at time of death. I can have great confidence that this person was indeed 91 at the time of death because their self-reported age of 74 was recorded earlier. As such, I have greater confidence in the ages of both the survivors and those who reportedly died in the NHIS-LMF than I have in the official vital statistics.

Because I carry out three separate analyses of black and white male and female mortality risk on the NHIS-LMF 1986–2006, I use three separate samples. The first analysis uses data from all black and white male and female respondents in the NHIS between 1986 and 2004 who were eligibly included in the NHIS-LMF between their survey date and December 31, 2006 (NCHS 2010). After I restricted the sample to white and black male and female respondents aged 25 to 84 at time of survey, the data contained 926,236 cases. This sample was then stratified by race/ethnicity and sex to generate a black male sample of 57,352 cases, a white male sample of 373,665 cases, a black female sample of 81,389 cases, and a white female sample of 413,831 cases (Table 2).

These race/ethnicity–sex stratified samples were then transformed into person-period data to account for subsequent mortality risk from time of survey until December 31, 2006. This first set of stratified person-period samples will be collectively referred to as "Data A." The Data A samples were also adjusted in two ways, producing data structures—"Data B" and "Data C"—which are described below.

In my second analysis, I wish to account for the possibility of poor data biasing mortality estimates at the oldest age groups. Thus, to improve the quality of data among the older-aged respondents in the NHIS-LMF 1986–2006, I made several adjustments to the sample. To improve confidence in age reports, I refined the NHIS-LMF data by computing new ages at time of survey based on respondent-reported month and year of birth, and the year and quarter-year of interview. I also omitted from this second sample any respondent with missing values on their birth year, birth month, or educational attainment.[1] Finally, to decrease age misreports among

[1] Preliminary analyses of older-age mortality in the NHIS-LMF found that respondents with missing educational attainment had implausibly low risk of mortality at older ages.

Table 2 Means of non-Hispanic white and black male and female NHIS-LMF 1986–2006 samples

	Black Male	White Male	Black Female	White Female
Data A, Survey Sample				
Mean age	46.33	47.88	46.54	49.13
Mean survey year	1994.11	1993.80	1993.98	1993.73
Mean birth year	1947.32	1945.46	1946.98	1944.14
% Deceased	19.62	17.00	14.88	14.69
n	57,352	373,665	81,389	413,831
Data A, Person-Period Sample				
Mean age	51.25	52.78	52.01	54.43
Mean current year	1998.52	1998.47	1998.54	1998.47
Mean birth year	1947.27	1945.69	1946.54	1944.05
Mean duration of follow-up	13.43	13.80	13.81	13.97
% Deceased	1.64	1.36	1.19	1.15
n	684,014	4,663,419	1,015,466	5,287,613
Data C, Collapsed Sample				
Mean age	(45–49) 4.55	(50–54) 5.08	(45–49) 4.78	(50–54) 5.45
Mean period	(1995–1999) 2.40	(1995–1999) 2.38	(1995–1999) 2.41	(1995–1999) 2.38
Mean cohort	(1945–1949) 10.43	(1940–1944) 9.89	(1945–1949) 10.21	(1940–1944) 9.52
Mean exposure time	9,340.13	60,018.05	13,317.47	63,163.61
Mean count of deaths	95.20	500.49	98.38	463.88
Mean cell count	9,897.63	63,368.30	14,069.86	66,619.60
n	137	137	137	137

respondents in the older age groups, I restricted the sample to respondents aged 25 to 75 at time of survey who were the sole provider of information in the NHIS. Respondents who relied on proxies to report their age were deleted from the sample. Because mortality status in the NHIS-LMF 1986–2006 can be followed for as many as 21 years, mortality risk in this restricted sample can still be reliably analyzed into the early 90s. Sample sizes and means of the race/ethnicity–sex stratified restricted NHIS-LMF 1986–2006 samples, henceforth referred to as "Data B," are displayed in Table 3.

In my third analysis, I estimate age-period-cohort models of black and white adult mortality rates in the NHIS-LMF 1986–2006. To do so, I collapsed the Data A samples into 137 cells of five-year age-period-cohort blocks. (Because of sparse cell counts, the cells associated with the five-year birth cohort 1975–1979 were omitted.) These collapsed data, which will henceforth be referred to as "Data C," can be seen in the bottom panel of Table 2. The data were collapsed into five-year age by five-year period by five-year cohort cells for two primary reasons: (1) sparse mortality counts in the black men's and women's individual-level samples preclude stable age-period-cohort modeling, and (2) the aggregated data structure breaks the linear dependency between age, period, and cohort (Glenn 2005).

Table 3 Means of non-Hispanic white and black male and female NHIS-LMF 1986–2006 samples, restricted by age, educational attainment, and proxy status

	Black Male	White Male	Black Female	White Female
Data B, Survey Sample				
Mean age	46.10	47.47	45.60	47.47
Mean survey year	1995.22	1994.90	1994.40	1994.08
Mean birth year	1949.13	1947.42	1948.80	1946.61
% Deceased	17.26	14.39	12.80	11.10
n	40,394	253,468	66,549	332,273
Data B, Person-Period Sample				
Mean age	50.84	52.32	51.11	53.03
Mean current year	1999.03	1998.94	1998.76	1998.68
Mean birth year	1948.18	1946.62	1947.65	1945.65
Mean duration of follow-up	12.65	13.09	13.64	13.94
% Deceased	1.57	1.25	1.05	0.88
n	442,557	2,918,831	808,479	4,190,391

Methods

First Analysis: Age-Specific Mortality Risk in Data A and Data C

Using Data A and SAS 9.2, I estimate fixed-effects discrete-time hazard models to derive age effects of mortality risk for the black and white male and female populations in the United States between 1986 and 2006. These models assume a binomial distribution for the occurrence of mortality at a single age, with a complementary log-log transformation to make linear the binomial response (Powers and Xie 2000). Estimates of the log coefficients are plotted against each other to observe the single-year age at which the sex-specific black-white mortality crossovers occur in these data. Also, because I wish to incorporate both period and cohort effects in subsequent analyses of black and white male and female U.S. adult mortality risk between 1986 and 2006, I also estimate a five-year age-only model using the collapsed Data C. Results from these analyses provide baseline comparisons with results from the subsequent age-period-cohort models. In these initial Data C analyses, I use fixed effects log-linear models to estimate the five-year age-only effects of mortality rates for the black and white male and female populations in the United States between 1986 and 2006. These models assume a Poisson distribution for counts of deaths in each five-year age cell. Offsetting the natural log of the aggregated exposure time lived by all members in the respective cell results in a model for mortality rates. Fifteen five-year age group effects are computed for each race/ethnicity–sex subpopulation, and the log coefficients are plotted against each other to illustrate the occurrence of a black-white crossover in mortality risk for both the male and female samples.

Second Analysis: Age-Specific Mortality Risk in Adjusted Data B

Using the adjusted NHIS-LMF 1986–2006 (Data B), I reestimate the single-year fixed-effects discrete-time hazard models performed on Data A. That is, I employ the binomial distribution with a complimentary log-log link function to measure the event of a death (0/1) at a given age. As a result, I can determine the differences in fixed-effects age estimates of mortality risk for the black and white male and female NHIS-LMF 1986–2006 samples that were due to the data adjustments made between Data A and Data B. If the black-white mortality crossover in the NHIS-LMF 1986–2006 is at all driven by data quality bias(es) associated with age, missing educational attainment, or proxy reporting status, then the ages at which the sex-specific crossovers occur are likely to be higher in Data B than in Data A (Hill et al. 2000; Preston et al. 1996; Preston et al. 1999; Preston and Elo 2006).

Third Analysis: Age-Period-Cohort Analysis of Mortality Rates in Data C

To incorporate period and cohort effects into analyses of the U.S. black-white mortality crossovers, I employ recently developed HAPC models for repeated cross-section survey data (Yang and Land 2006, 2008). These methods use a CCREM to embed each NHIS-LMF respondent within both a time period and birth cohort at a given age. Because the NHIS-LMF data set follows individual mortality risk as respondents age across periods, each respondent can occupy several age-period-cohort combinations. Consequently, although collinearity between the three effects is very high, these data do not suffer the "indentification problem" induced by an absolute linear dependency among age, period, and cohort (Glenn 2005; Mason et al. 1973). Further, the HAPC-CCREM model is an appropriate methodological tool to measure the three processes simultaneously, and has been shown to be more efficient than a fixed-effects approach when data, such as the NHIS-LMF 1986–2006, are unbalanced (Yang and Land 2008). The HAPC-CCREM model estimates fixed effects of the five-year age groups and random effects of the five-year period and five-year cohort groups, and is structured in the following way:

$$\text{Level 1 within-cell model:} \quad \log\left[\mathrm{E}(D_{ijk})\right] = \alpha_{jk} + \sum_l \beta_l A_{ijkl} + \log(R_{ijk}),$$

where D_{ijk} is the count of deaths occurring in the ith age group within the jth period and the kth cohort. A_{ijkl} denotes a set of dummy variables corresponding to the five-year age groups with fixed effects β_l; α_{jk} is a random intercept indicating the log mortality rate in the reference age group (50–54) in period j and belonging to cohort k; and log (R_{ijk}) is the natural log of the aggregated exposure time lived during each age-period-cohort cell.

$$\text{Level 2 between-cell random intercept model:} \quad \alpha_{jk} = \pi_0 + t_{0j} + c_{0k},$$

in which α_{jk} specifies that the fixed age effects vary from period to period and from cohort to cohort; π_0 is the log mortality rate at the reference age (50–54) averaged over all periods and cohorts; t_{0j} is the overall five-year period effect averaged over all five-year birth cohorts with variance σ^2_{t0}; and c_{0k} is the overall five-year cohort effect averaged over all five-year periods with variance σ^2_{c0}.

I combine the Level 1 and Level 2 models to estimate counts of deaths in each five-year age-period-cohort cell using SAS 9.2's PROC GLIMMIX, assuming a Poisson distribution for counts of deaths and an offset for the logarithm of the aggregated person-years lived across each cell to generate age-period-cohort–specific mortality rates. Because of collinearity and small cell sizes in some age-period-cohort combinations, HAPC-CCREM models did not converge for the black female sample. I dealt with this in two ways. First, I carried out a sensitivity analysis in R using alternative estimation algorithms applicable to this class of problems, including maximum marginal likelihood (using both Laplacian and Gaussian-Quadrature methods) and hierarchical Bayesian models estimated using Markov Chain Monte Carlo (MCMC) under a Gibbs sampling approach. Second, I reestimated the HAPC-CCREM in SAS using a constrained cohort covariance value of .32. This value for the constrained cohort covariance parameter was estimated from three chains of the hierarchical MCMC Bayesian model after 10,000 simulations. Also, multiple values of the constrained parameter were tested, and model results were contrasted with results from corresponding fixed-effects models to guide my final selection of the constrained value.

Results

Table 4 presents Data C estimates of fixed effects five-year age coefficients from age-only analyses of mortality risk for non-Hispanic black and non-Hispanic white male and female samples. (Tabulated results of age-only estimates from Data A and Data B are not shown, but are illustrated in subsequent figures.) For each race/ethnicity–sex sample, nearly all age-group effects are significant at the .001 α level. For black males, the age group 95–99 is not significant at all commonly used α levels. To observe the black-white mortality crossover in these five-year collapsed NHIS-LMF 1986–2006 data, I plot the estimated logged mortality rates for each male and female race/ethnicity sample. In these five-year data, one can see that for each sex, the age-specific mortality risk of the two racial/ethnic subpopulations converge and then crossover at around age 85 (see the right panel in Fig. 2).

In the left panel of Fig. 2 are graphed the logged single-year age estimates of black and white men's and women's mortality risk from the NHIS-LMF 1986–2006 Data A discrete-time hazard models. These single-year fixed-effects estimates of age coefficients from Data A are consistent with the patterns observed in the Data C five-year results, and all estimates are significant at the .001 α level. For men, the mortality risk in the black sample between 1986 and 2006 was higher than the mortality risk of the white sample at every age until about 86. At this point, the black mortality risk converged with and then became lower than the mortality risk of the white sample. Similarly, for women in the NHIS-LMF 1986–2006, the black-white mortality crossover is observed to take place in the single-year age-only Data A sample at around age 84.

The extent to which the crossovers observed in Fig. 2 are products of poor data bias(es) or differences in heterogeneity between the black and white samples is unknown. To address the former concern, I reestimated the single-year discrete-time hazard models of mortality risk on the adjusted Data B NHIS-LMF 1986–

Table 4 Fixed-effects generalized linear models of age effects of non-Hispanic black and non-Hispanic white men's and women's mortality risk, 1986–2006

	Men		Women	
Fixed Effects	Black	White	Black	White
Age				
25–29	−1.329	−1.564	−2.220	−2.003
	(0.183)	(0.141)	(0.293)	(0.222)
30–34	−1.326	−1.541	−1.635	−1.789
	(0.068)	(0.049)	(0.082)	(0.070)
35–39	−1.159	−1.253	−1.266	−1.278
	(0.045)	(0.029)	(0.050)	(0.037)
40–44	−0.846	−0.876	−0.967	−0.860
	(0.037)	(0.021)	(0.040)	(0.027)
45–49	−0.527	−0.453	−0.459	−0.428
	(0.035)	(0.019)	(0.036)	(0.024)
50–54 (ref.)	—	—	—	—
55–59	0.291	0.419	0.284	0.484
	(0.035)	(0.018)	(0.037)	(0.022)
60–64	0.683	0.919	0.735	0.989
	(0.036)	(0.017)	(0.037)	(0.022)
65–69	1.092	1.396	1.088	1.429
	(0.035)	(0.017)	(0.036)	(0.021)
70–74	1.495	1.866	1.431	1.896
	(0.035)	(0.016)	(0.036)	(0.019)
75–79	1.781	2.297	1.840	2.355
	(0.039)	(0.016)	(0.036)	(0.019)
80–84	2.178	2.745	2.184	2.849
	(0.047)	(0.017)	(0.040)	(0.019)
85–89	2.504	3.250	2.544	3.362
	(0.086)	(0.024)	(0.055)	(0.021)
90–94	2.838	3.615	2.871	3.877
	(0.254)	(0.070)	(0.121)	(0.034)
95–99	2.373[a]	3.671	2.884	4.132
	(1.458)	(0.437)	(0.557)	(0.139)
Intercept	−4.561	−5.268	−4.987	−5.757
Model Fit				
−2 Log-Likelihood	1,134.8	1,783.4	1,011.7	1,500.3
χ^2/df	2.63	6.41	1.66	4.33
N	137	137	137	137

Note: Numbers in parentheses are standard errors.

[a]Not significant.

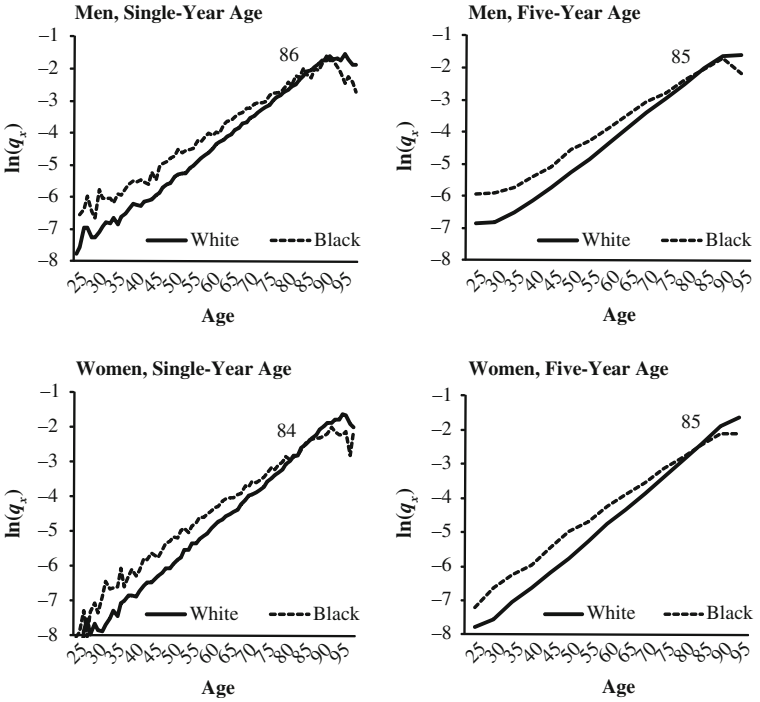

Fig. 2 Logged single-year age-specific adult mortality risks and logged five-year age-specific mortality rates of U.S. black and white males and females, NHIS-LMF 1986–2006

2006. Results from these data-adjusted analyses are graphically depicted in the left panel of Fig. 3. Here, the single-year age estimates of black and white men's and women's log mortality risk are plotted across age.

Despite improving our confidence in the estimates of older-age mortality risk, Data B show that the black-white crossover in each sex persists. That is, the black and white male NHIS-LMF 1986–2006 samples restricted to self-reporting respondents age 25–75 at the time of the survey still generate a black-white mortality crossover at age 85. Similarly, the same adjusted black and white female samples generate a black-white mortality crossover at age 82. Thus, rather than pushing back the age at which the crossover occurs, adjustments made to improve the reliability of the data have drawn the black-white mortality crossover downward in age for both sexes.

Next, Table 5 presents results from the HAPC-CCREM analyses of mortality rates for black and white male and female samples. The fixed age effects are presented in the top frame of the table, and Bayesian solutions for the estimated random components of the models are presented in the bottom frame of the table. Consistent with previous studies, I find very little period variation in U.S. mortality rates from 1986 to 2006, but significant and substantive cohort variation in U.S. mortality rates is found for all race/ethnicity–sex populations (Yang 2008). These findings are best depicted in the right panel of Fig. 4, which shows a great deal of cohort variation in adult mortality risk for all four race/ethnicity-sex subpopulations. The nonsignificant period effects are also presented in the left panel of Fig. 4.

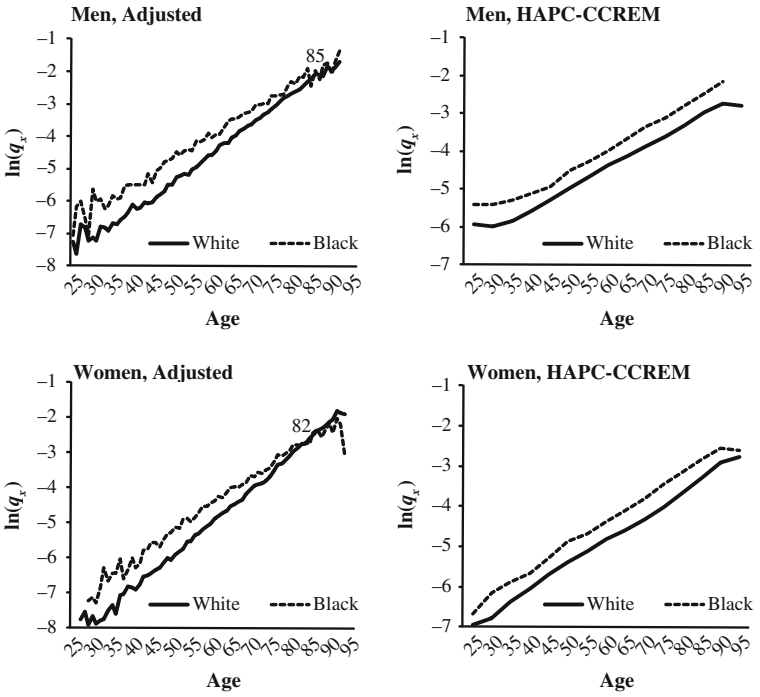

Fig. 3 Fitted logged mortality risks of U.S. black and white male and female adjusted samples of NHIS-LMF 1986–2006, and fitted logged mortality rates from HAPC-CCREMs

More importantly, the findings suggest a great deal of disparate cohort patterns in mortality risk between non-Hispanic black and non-Hispanic white populations. Black-white differences in estimated cohort effects for those birth cohorts that make up the oldest-old age groups (i.e., 1900 to 1925) are much smaller than black-white differences in cohort effects for birth cohorts from the mid-twentieth century. Also, consistent with the fundamental cause theory, cohort reductions in the white male and female populations' mortality rates between 1986 and 2006 were significantly greater than respective cohort reductions in the black male and female populations (Link 2008; Link and Phelan 1995). Accounting for these disparate cohort effects profoundly impacts the estimates of the age effects of mortality rates for the sex-specific black and white samples. As seen in the right panel of Fig. 3, the patterns of estimated age effects on mortality for the white and black male and female samples have significantly and remarkably changed from Fig. 2.

After disparate cohort and period effects are controlled for, the estimated age effects on mortality rates in both the male and female black samples remain significantly higher than the age effects on mortality rates in both the male and female white samples at all ages. That is, by accounting for cohort and period variation in U.S. adult mortality rates between 1986 and 2006, I am able to uncross the U.S. black-white crossover in fitted age effects on mortality rates. In effect, the black-white mortality crossover in U.S. mortality risk reflects disparate cohort effects between the non-Hispanic black and non-Hispanic white populations. This is not to say that the black-white crossover in age-specific mortality rates has been entirely uncrossed. In fact, when the combined estimated effects of age, period, and

Table 5 HAPC-CCREMs of U.S. non-Hispanic black and non-Hispanic white men's and women's adult all-cause mortality rates, 1986–2006

	Men		Women	
	Black	White	Black	White
Fixed Effects				
Age				
25–29	−0.826	−0.934	−1.760	−1.495
	(0.195)	(0.151)	(0.308)	(0.232)
30–34	−0.867	−1.018	−1.256	−0.1374
	(0.088)	(0.066)	(0.113)	(0.089)
35–39	−0.777	−0.858	−0.995	−0.962
	(0.063)	(0.044)	(0.078)	(0.056)
40–44	−0.591	−0.614	−0.785	−0.646
	(0.049)	(0.032)	(0.059)	(0.041)
45–49	−0.426	−0.333	−0.412	−0.291
	(0.040)	(0.023)	(0.044)	(.029)
50–54 (ref.)	—	—	—	—
55–59	0.254	0.297	0.188	0.297
	(0.042)	(0.022)	(0.045)	(0.027)
60–64	0.523	0.595	0.487	0.586
	(0.053)	(0.029)	(0.059)	(0.035)
65–69	0.843	0.848	0.767	0.802
	(0.062)	(0.036)	(0.073)	(0.042)
70–74	1.167	1.109	1.056	1.088
	(0.069)	(0.043)	(0.085)	(0.048)
75–79	1.400	1.353	1.412	1.382
	(0.078)	(0.049)	(0.090)	(0.054)
80–84	1.724	1.639	1.711	1.736
	(0.089)	(0.056)	(0.112)	(0.061)
85–89	2.036	1.988	2.040	2.110
	(0.123)	(0.066)	(0.131)	(0.068)
90–94	2.365	2.222	2.322	2.493
	(0.277)	(0.099)	(0.182)	(0.080)
95–99	1.911[a]	2.175	2.259	2.642
	(1.468)	(0.446)	(0.582)	(0.162)
Random Effects				
Cohort				
1970–1974	−0.595	−1.119	−0.812	−0.822
	(0.185)	(0.241)	(0.265)	(0.252)
1965–1969	−0.686	−0.957	−0.461	−0.877
	(0.138)	(0.204)	(0.190)	(0.198)
1960–1964	−0.513	−0.819	−0.513	−0.721
	(0.121)	(0.198)	(0.173)	(0.186)

Table 5 (continued)

	Men		Women	
	Black	White	Black	White
1955–1959	−0.265	−0.628	−0.205	−0.715
	(0.117)	(0.195)	(0.165)	(0.183)
1950–1954	−0.060	−0.485	−0.184	−0.624
	(0.115)	(0.194)	(0.161)	(0.182)
1945–1949	−0.119	−0.421	−0.169	−0.447
	(0.114)	(0.193)	(0.158)	(0.180)
1940–1944	0.037	−0.247	0.054	−0.253
	(0.113)	(0.193)	(0.156)	(0.180)
1935–1939	0.103	−0.038	0.142	−0.005
	(0.113)	(0.193)	(0.155)	(0.179)
1930–1934	0.227	0.174	0.190	0.178
	(0.114)	(0.193)	(0.156)	(0.179)
1925–1929	0.119	0.400	0.247	0.383
	(0.115)	(0.193)	(0.159)	(0.180)
1920–1924	0.379	0.572	0.316	0.518
	(0.117)	(0.194)	(0.162)	(0.181)
1915–1919	0.397	0.768	0.331	0.673
	(0.124)	(0.195)	(0.169)	(0.182)
1910–1914	0.411	0.919	0.368	0.815
	(0.142)	(0.178)	(0.179)	(0.184)
1905–1909	0.404	1.023	0.483	0.912
	(0.197)	(0.205)	(0.209)	(0.188)
1900–1904	0.052	0.858	0.214	0.984
	(0.372)	(0.360)	(0.443)	(0.251)
Period				
2005–2009	0.008	0.224	0.103	0.282
	(0.013)	(0.080)	(0.049)	(0.110)
2000–2004	−0.007	0.101	0.034	0.174
	(0.012)	(0.079)	(0.044)	(0.109)
1995–1999	−0.005	−0.004	0.006	0.013
	(0.012)	(0.079)	(0.043)	(0.109)
1990–1994	0.005	−0.096	−0.055	−0.154
	(0.013)	(0.079)	(0.048)	(0.110)
1985–1989	−0.001	−0.225	−0.088	−0.314
	(0.014)	(0.085)	(0.066)	(0.115)
Intercept	−4.488	−4.910	−4.867	−5.367
Covariance Parameters				
Cohort	0.154	0.542	0.320	0.470
	(0.075)	(0.219)	—	(0.190)

Table 5 (continued)

	Men		Women	
	Black	White	Black	White
Period	1.98E-4	0.031	0.007	0.059
	(0.001)	(0.023)	(0.009)	(0.043)
Model Fit				
−2LPL	123.00	−65.51	86.44	−57.41
χ^2 / *df*	1.38	1.00	1.07	0.99
N	137	137	137	137

Note: Numbers in parentheses are standard errors.

[a]Not significant.

cohort are plotted for each black and white, male and female model, the patterns of age-specific log mortality rates are similar to those seen in the right panel of Fig. 2. However, what the findings suggest is that the convergence and crossover of non-Hispanic black and non-Hispanic white mortality risk in the United States is chiefly a product of disparate cohort-specific age effects between the two populations. Indeed, as illustrated in Fig. 5, the black-white mortality crossover for both the male and female samples occurs only for those respondents born in the 1900, 1905, and 1910 birth cohorts.

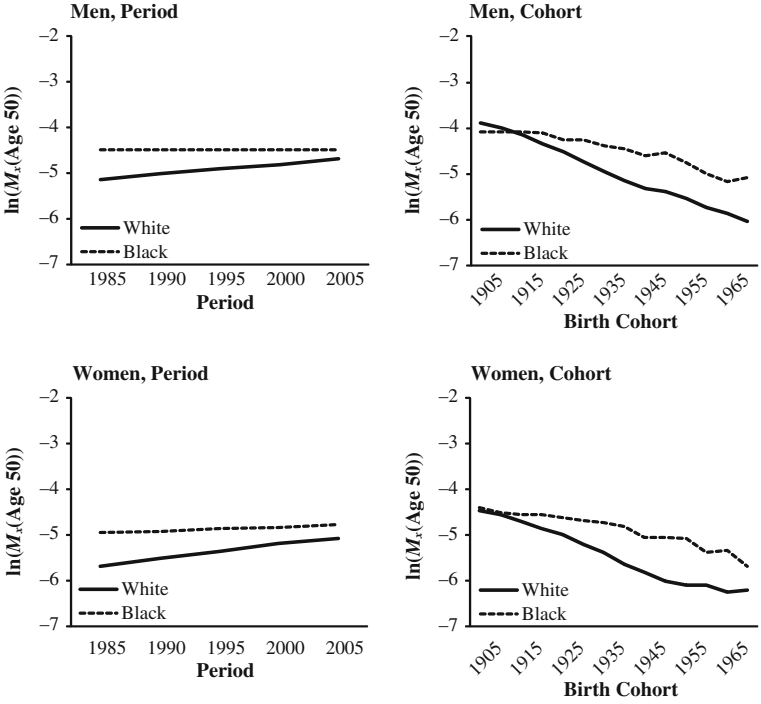

Fig. 4 HAPC-CCREM estimates of random period and cohort effects of U.S. black and white male and female mortality rates, NHIS-LMF 1986–2006

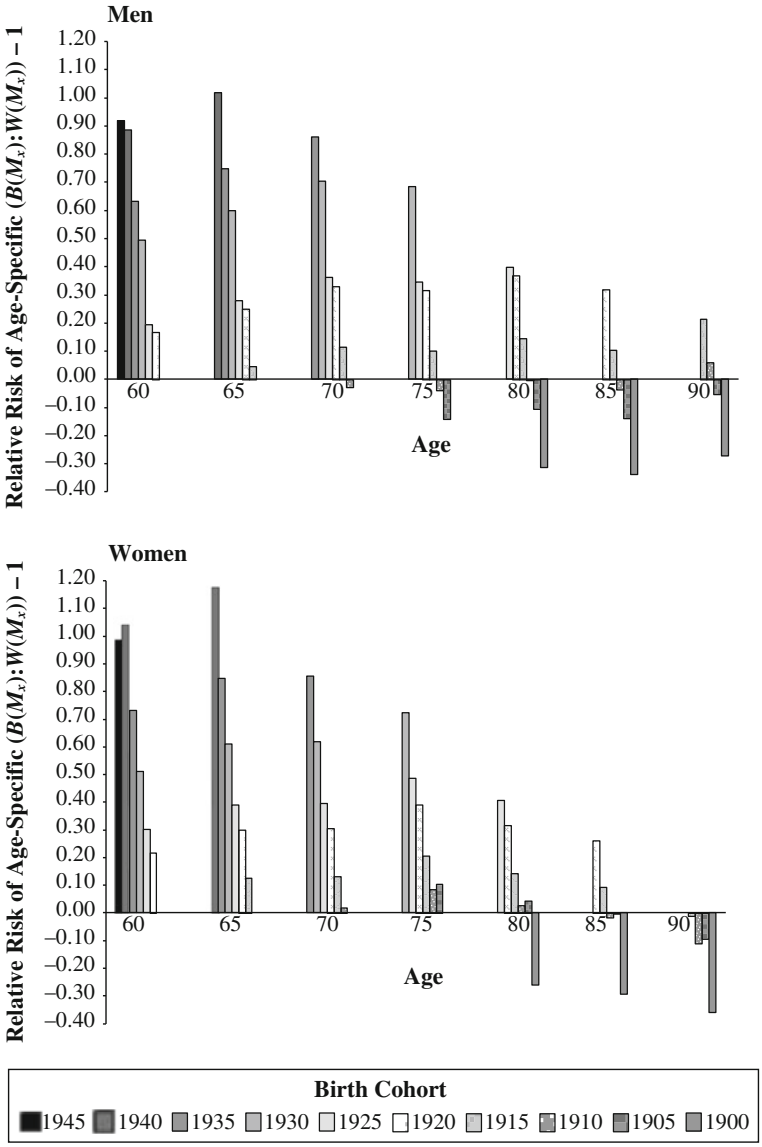

Fig. 5 Ratios of U.S. black and white men's and women's older-age mortality rates in the NHIS-LMF 1986–2006, by birth cohort

Specifically, Fig. 5 displays the ratio of fitted age-specific black mortality rates to fitted age-specific white mortality rates across age-groups 60–64 to 90–94 by birth cohort. Because of nonsignificant effects for the age group 95–99, results at these ages are not included. Figure 5 shows that for the male sample, no crossover exists for any cohort born after the 1910–1914 period. That is, for all U.S. cohorts born in 1915 or later, black male fitted mortality rates between 1986 and 2006 are higher than white male fitted mortality rates at every age group. For instance, for the 1900 and 1905 cohorts, black male mortality rates are lower than white male mortality rates at

ages 80, 85, and 90. However, at these respective ages, the black male fitted mortality rates for the 1915 and 1920 cohorts are higher than the white male fitted mortality rates from the same birth cohorts. In short, although black-white mortality crossovers are observed at several older age groups, these crossovers are cohort-specific phenomena. Thus, the observed black-white crossover in fitted age-specific estimates of mortality risk is driven entirely by cohort differences in black and white male mortality risk in the 1900, 1905, and 1910 birth cohorts. This is also largely the case with the female sample. However, unlike the male sample, the 1915 birth cohort in the female sample also experiences a black-white crossover in the fitted estimates of mortality rates at age group 90–94.

Discussion

The evidence for cohort patterns of black and white, male and female mortality risk support the heterogeneity explanation of the black-white mortality crossover. However, the results add an important finding to consider. Researchers analyzing life course processes of health and mortality must recognize that changes to population composition and processes of aging occur within a cohort-based sociohistorical context (Ben-Shlomo and Kuh 2002; Montez and Hayward 2011; Riley 1973, 1978, 1987; Ryder 1965). The question concerning the existence and timing of a mortality crossover between the black and white U.S. populations will continue to attract attention from demographers and other health researchers. Increasingly important in this regard are questions pertaining to population composition and heterogeneity across both age and cohorts. Mortality risk at a given age in a given calendar year reflects cohorts' life course experiences and "cohort morbidity phenotypes," and researchers should unpack and explain these relevant cohort forces when comparing age-specific mortality risks of different populations (Finch and Crimmins 2004). That is, increases in health knowledge and/or advances in health-enhancing technologies unfold across cohorts in disparate ways. Further complicating these processes is the unequal ways that these cohort processes are conditioned by both gender and race.

In this article, I show that considering these disparate cohort forces is necessary for better understanding the black-white mortality crossover in the United States population. Specifically, I contribute three key findings. First, linked survey-mortality data such as the NHIS-LMF 1986–2006 provide a unique chance to simultaneously analyze age, period, and cohort effects of mortality patterns and trends. In attempts to analyze changing population composition, researchers should increasingly use data with these structures to assure that they are accounting for both life course and temporal shifts in heterogeneity. Second, although data quality issues remain a problem in survey-based data, efforts to adjust the samples to rectify any bias failed to account for the black-white mortality crossover in the NHIS-LMF 1986–2006. This is consistent with some cases of past research, yet my efforts to improve data quality shifted the age at the mortality crossover downward (Lynch et al. 2003; Preston et al. 1996). This largely reflects the fact that restricting the sample to respondents aged 25–75 at the time of the survey changed the black and white samples' respective composition of early birth cohorts in different ways. For both the black and white samples, the age restriction in Data B reduced the composition of

earlier birth cohorts at older ages and increased the composition of later birth cohorts, but the change was greater in the black sample. Thus, the age restriction in Data B biased the data to reflect later cohorts' mortality risks, but did so differently for the black samples than for the white samples. The ultimate result from these race/ethnicity differences in these changes was to drive the age at which the crossover occurs downward. That these substantial adjustments made little difference in the relationship between U.S. black and white older-adult mortality risks suggests that quality bias is relatively minimal in these data. Lastly, I found evidence supporting my hypothesis that the observed U.S. black-white crossovers in men's and women's mortality risk in the NHIS-LMF 1986–2006 is overwhelmingly attributable to disparate cohort effects of mortality. This finding is consistent with evidence that the crossover age is increasing, and is a further indication that the black population is becoming more heterogeneous across cohorts (Lynch et al. 2003). The finding is also consistent with work that has emphasized the "*interdependence* [italics in the original] of aging and social change" within a cohort perspective (Riley 1987:2).

Although this study has provided additional insight and consideration into black-white differences in older-adult mortality, the mortality crossover, and frameworks for understanding temporal changes in U.S. adult mortality risk, there are some limitations. First, the time period 1986–2006 is a small window in which to simultaneously analyze the forces of age, period, and cohort on U.S. adult mortality risk. Second, there is a great deal of selection into the NHIS. Differences between the black and white samples in rates of institutionalization, healthy participant effects, and use of proxy reporting can affect the heterogeneity of the older-age black and white male and female NHIS-LMF samples. Lastly, the age-period-cohort analyses were carried out on aggregated data, precluding investigations of individual-level controls, mediators, or two-way effects. Despite these limitations, this study has demonstrated the important role played by cohort patterns of mortality in explaining black-white differences in U.S. old-age mortality risk. The mortality crossover does indeed exist, but only for specific cohorts born early in the twentieth century. Although black-white differences in life course exposures generate differences in heterogeneity of mortality risk across age, we must recognize that these life course processes are inherently embedded in sociohistorical contexts (Ben-Shlomo and Kuh 2002; Riley 1987; Ryder 1965). Only by building a cohort perspective into life course analyses of mortality risk can we fully consider such contextual effects.

Acknowledgments Dr. Masters is currently a Robert Wood Johnson Foundation Health & Society Scholar at Columbia University. This research was completed when Dr. Masters was supported by a grant (1 R01-HD053696, PI Robert A. Hummer) from the Eunice Kennedy Shriver National Institute of Child Health Research and Human Development and by an NICHD infrastructure grant (5 R24 HD042849) awarded to the Population Research Center (PRC) at the University of Texas at Austin. This is a revised article based on a presentation at the 2010 annual meeting of the American Sociological Association, August 14–17, Atlanta, GA. The author thanks Bob Hummer, Mark Hayward, Dan Powers, and the anonymous reviewers for very helpful comments and advice on this draft.

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