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# A COHORT PERSPECTIVE OF U.S. ADULT MORTALITY

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# A COHORT PERSPECTIVE OF U.S. ADULT MORTALITY

**by**

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# Dedication

To my mom and dad and brother, whose love and support made this work possible.

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# A COHORT PERSPECTIVE OF U.S. ADULT MORTALITY

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This dissertation advances a cohort perspective to analyze trends in racial and educational disparities in U.S. adult mortality. The project is organized around three themes. First, I emphasize that recent temporal changes in U.S. adult mortality risk are rooted in cohort forces. Unfortunately, much of the mortality literature has failed to account for the fact that the sociohistorical conditions of U.S. cohorts have changed dramatically, and these changes have tremendous implications for population health and mortality trends. My work clearly shows the pitfalls of omitting these cohort effects from analyses of U.S. adult mortality risk. Second, I illustrate that because exposure to social and health conditions have changed over time, resources in adulthood are growing increasingly important in shaping U.S. adult mortality risk. In this regard, my findings also highlight growing disparities in U.S. mortality across race/ethnic gender groups. Third, I advance a cohort theory of U.S. mortality, drawing from both "fundamental cause" theory and a life course perspective of mortality but couching them in a cohort framework to highlight the importance of historical changes in U.S. social and health contexts in both childhood and adulthood.

This cohort perspective is then used to analyze three central topics in the U.S. mortality literature: the black-white crossover in older-adult mortality, the growing educational gap in U.S. adult mortality, and the origins and persistence of black-white inequalities in U.S. adult mortality. I estimate hierarchical age-period-cohort cross-classified random effects models using National Health Interview Survey-Linked Mortality Files between 1986 and 2006 to

simultaneously analyze age, period, and cohort patterns of U.S. adult mortality rates. I find (1) the black-white crossover is a cohort-specific phenomenon, (2) educational disparities in U.S. adult mortality rates are growing across birth cohorts, not time periods, and (3) racial disparities in U.S. adult mortality rates stem from cumulative racial stratification across both cohorts and the life course. Such findings have direct consequences for both mortality theories and policy recommendations. Only by considering the disparate sociohistorical conditions that U.S. cohorts have endured across their life courses can we fully understand and address current and future health disparities in the United States.

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# CHAPTER 1

# Introduction

## 1.1 Research Problem

This dissertation concerns temporal changes in U.S. adult mortality rates, and various ways to think about and measure these changes. I primarily focus on the intersection of the life course and cohort processes that drive trends and disparities in U.S. adult mortality rates. The themes are of special scientific interest in mortality research. Only recently have scholars and researchers begun to appreciate the degree to which cohorts' differing experiences fundamentally shape mortality risk across age. Indeed, compared to age and period effects, cohort effects have been given much less attention in the U.S. mortality literature. In this dissertation I show why this inattention is problematic for understanding processes driving mortality disparities and changes therein. As such, I call on scholars to integrate more cohort theories and measures in their analyses of U.S. mortality trends and disparities.

## 1.2 Measuring Mortality

Scholars have long noted the importance in accurately and correctly measuring population-level mortality. As early as the 1600s, written records were preserved in medieval parish registers in England, Sweden, and France. John Graunt, often regarded as the founding father of vital statistics, published his *Natural and Political Observations Made upon the Bills of Mortality* in England in 1662, the first document to demonstrate and record how mortality varies from year to year, by cause, by sex, and by region of residence (Graunt 1662). Since then,

governments, researchers, and medical practitioners have been concerned with estimating and tracking mortality patterns and trends. Indeed, accurately assessing temporal changes in mortality sheds light on the social processes and diseases causing death, and informs health policy and our understanding of the origins and persistence of health and mortality disparities.

Since the U.S. population is classified as being in the "age of delayed degenerative diseases" (Olshansky and Ault 1986), any temporal change in adult mortality patterns highlights changes to the underlying degenerative diseases affecting the population. As such, conceptualizing, measuring, and differentiating between age, period, and cohort effects of adult mortality are important for various demographic, social, and political reasons.

*Age*

Age contains the greatest variation in health and mortality rates. Biological processes associated with aging drive susceptibility in mortality risk across the life course and the age patterns of mortality exhibit incredibly robust and consistent patterns across time, nations, and mortality schedules. However, only minimal efforts have been made to understand how the aging process has changed, and continues to change, across time. Yet the life course can be rather variable across time periods, in that at any given time age effects on mortality risk reflect the distinct interaction between a given life course during a specific historical time period. Life course variation in mortality risk may also be a cohort phenomenon, in which the life course is shaped by the confluence of biological aging effects and a cohort's unique experience of history. That is, cohorts differ in their exposure time to risk factors on the one hand, and the benefits of medical inventions, public health measures, and improvements in nutrition on the other. Due to both period and cohort forces the association between aging processes and mortality risk may

very well vary across time. Consequently, it is distinguishing and capturing the other two components of temporal variation in mortality risk – period and cohort – that mortality researchers are generally most concerned with.

*Period*

Period effects of mortality are conceived of as variations in mortality at the time at which death is recorded, and which generally affect all age groups equally. These are best thought of as environmental, historical, and/or calamitous events that influence the risk of mortality for all members of a population (e.g., war, natural disasters, and epidemics of highly lethal infectious diseases). Advances in medical technologies and improvements in public health are often perceived to be and are measured as period effects, but reasons are developed in this dissertation to suggest otherwise (Cutler, Rosen and Vijan 2006; Cutler et al. 2010). In short, it is difficult to imagine that the diffusion, adoption, and application of health knowledge and technologies do not affect a population's health and mortality in a cohort fashion. This is because the cumulative effect of such knowledge and technologies reflect cohorts' disparate exposure times to them (Link 2008).

*Cohort*

Birth cohort (henceforth referred to as "cohort") effects stem from variations in mortality risk between groups of individuals born in the same year or set of years (e.g., persons born between 1905 and 1909 could be designated the five-year 1905 birth cohort). A cohort conception of mortality is important because it integrates historical changes in human society with a life course framework. That is, a cohort's members can carry the imprint or scar of

physical exposures to malnutrition, infectious diseases, and/or disabling processes as they age across the life course (Fogel 2005; Finch and Crimmins 2004; Case and Paxson 2010). Cohorts can also differ tremendously in their members' cumulative exposures to lifestyle risk factors, such as smoking (Wang and Preston 2009), as well as their cumulative exposure to health technologies, such as the percent of a cohort's members inoculated against the measles since 1963. Cohort differences in the cumulative time exposed to such physical threats or lifestyles on the one hand, and to improvements in health knowledge and technologies on the other, inherently shape cohorts' disparate mortality risks as they age across the life course.

Most studies of mortality trends simply document temporal fluctuations in mortality risk by using basic descriptive plots of age-specific or age-standardized death rates across time periods. Rarely have researchers explicitly attempted to disentangle period and cohort effects, and only infrequently do studies even theoretically distinguish them. National reports using vital statistics, for instance, most often are published using period-based life tables. Figure 1.1 below shows the age-specific probability of death ($q_x$) for the U.S. white men's populations at the age-range 64-99 for two time periods, 1990 and 2003. The time period 1990 is represented with a solid black line, while the time period 2003 is represented with a solid gray line. Ten-year birth cohorts are represented by colored square markers.

It is evident that the overall pattern of the age-specific $q_x$ between age 64 and 99 are significantly lower in the 2003 life table than in the 1990 life table. Factors behind this mortality reduction, however, cannot be known from simply looking at these plots. Evident in Figure 1.1, one cannot even discern whether the differences are period or cohort expressions. That is, if we compare 1990 and 2003 $q_x$ in the age range 79 to 85, we see at all ages the 2003 $q_x$ are lower than the 1990 $q_x$. However, we also note that at ages 79 and 80 in the 1990 life table we observe the

age-specific mortality risks of persons born in the cohort 1910-1919, whereas in the 2003 life table we observe the respective age-specific mortality risks of persons born in the cohort 1920-1929. Also, at ages 81 to 83 we are comparing cohorts 1900-1909 and 1920-1929, whereas at ages 84 and 85 we are comparing cohorts 1900-1909 and 1910-1919. Disentangling factors related to aging processes from temporal factors stemming from period and/or cohort phenomena are impossible using conventional linear estimates. And only until recently, analyses attempting to simultaneously assess age-period-cohort (APC) effects were limited by methodology and data availability (see Glenn 2005 for a thorough review of APC modeling using conventional techniques). Because of this, few advances in APC analytic modeling were made, and studies of U.S. mortality trends often produced results inconsistent with one another.

### 1.3 Methodological Advances in Age, Period, Cohort Modeling

Recently, great efforts have been undertaken to advance APC modeling techniques (see Smith 2008 and Yang 2011 for reviews of various new methods). Drawing heavily from the literature and methodology in biostatistics, numerous analytical methods have been developed to address the linear dependency between age, period, and cohort effects in models estimating mortality rates (Glenn 2005; Yang, Fu and Land 2004). In this dissertation I rely on recent advances in APC modeling made by Yang and Land (2006, 2008), a technique which employs a non-additive multilevel approach to identifying age, period, and cohort effects. As Fu (2008: 335) points out, it is "an undoubtful fact that the identity problem is *model specific rather than data specific* [sic]" (emphasis added). Fu further states that "confusion still exists and people may still be misled to a conclusion of pessimism that the APC data structure, rather than the proposed models, suffer the identifiability problem" (335). Furthermore, regarding HAPC

modeling specifically, Yang (2006: 45-46) clarifies, "the model identification problem induced by the linear dependency between age, period, and cohort – that is, Period = Age + Cohort – creates a major challenge for APC analysis from the point of *conventional linear models* (Mason and Smith 1985). The assumption of additivity, however, is only one approximation of the process of how social and demographic change occurs (Hobcraft, Menken and Preston 1982; Smith 2004). The hierarchical APC models introduced here are one family of *nonadditive models* that can be extremely useful for capturing the contextual effects of cohort membership and period events on a wide range of social demographic processes" (emphasis added). Both Fu and Yang are correct that the identification problem is not a data problem, but rather a modeling problem. As age, period, and cohort are modeled in the HAPC-CCREMs their effects are not linearly dependent. I use the HAPC-CCREM in my dissertation analyses of U.S. adult mortality because it is an appropriate tool with which to test my hypotheses. Greater details of the HAPC-CCREM as it applies to my chapter-specific hypotheses are provided in the chapters.

Overall, my dissertation advances a cohort perspective to explain why mortality disparities in the United States are changing or persisting. I draw from both fundamental cause theory and life course perspective, but couch these perspectives in a cohort framework in order to better understand trends in U.S. adult mortality. I employ the HAPC-CCREM to simultaneously estimate age, period, and cohort patterns of U.S. adult mortality rates, and provide evidence that significantly and forcefully suggests that recent trends and patterns are driven and shaped by cohort phenomena.

## 1.5 Data

*The NHIS-LMF and Data Structure(s)*

I use 19 survey waves of data from the National Health Interview Survey (NHIS) from years 1986 to 2004 that have been linked to vital statistics death records for the time January 1, 1986 to December 31, 2006. The resulting National Health Interview Survey-Linked Mortality Files (NHIS-LMF) provide information on over 1 million U.S. adults aged 25 and above whose subsequent survival status was followed for up to 21 years. These data contain over 100,000 identified deaths, permitting consistently stable estimates of rates of all-cause and some specific causes of U.S. adult mortality. Furthermore, diversity in self-identified race/ethnicity, nativity status, and gender permit detailed APC patterns of mortality for specific subgroups of the U.S. population. And lastly, the long follow-up period is of enough duration to estimate all three APC components in models of mortality rates, and collapsing the data into an aggregated APC cell structure generates multiple age-period-cohort patterns at every age-group. The cohort composition of the NHIS-LMF 1986-2006 across the distribution of age is provided in Figure 1.2. While the frequency of person-years across age were derived using single years of age, I indicate with vertical lines the five-year age-groups that are subsequently used to analyze age patterns of U.S. adult mortality rates in Chapters 2, 3, and 4.

As is evident in Figure 1.2, nearly all five-year age-groups (age-groups 30-34 to 80-84) comprise six different cohorts. Age-groups 25-29 and 85-89 are both composed of five cohorts, and age-group 90-94 contains four cohorts while only three cohorts comprise age-group 95-99. Taken together, the repeated cross-sectional design of the NHIS coupled with individual-level survival histories provide sufficient age and cohort overlap to estimate consistently reliable age, cohort, and period patterns of U.S. adult mortality between 1986 and 2006.

While specific alterations to the NHIS-LMF 1986-2006 will be discussed in each chapter, I will briefly introduce the general design of the aggregated data here. Each NHIS survey wave

was thoroughly cleaned and edited, and all waves were combined and matched to the computerized death records at the National Death Index (NDI). Only those respondents who have been properly identified are ultimately included in the data for analysis. Also, to focus on ages where mortality risk is high and death counts were sufficiently plentiful, and to limit the use of data where age is top coded, I restricted the NHIS-LMF to U.S.-born black and white respondents aged 25 to 84 at the time of the survey. These restrictions are applied to the data for all chapters. I then transformed the data structure into person-year format to follow the mortality status of each respondent at each year until December 31$^{st}$, 2006. Lastly, these individual survival histories were collapsed into aggregated subsamples of age-period-cohort blocks. The structure and age-ranges of these APC aggregated data differ across analyses and will be discussed in greater detail in each chapter. For now, I illustrate the general data structure in Figure 1.3, and discuss two hypothetical survival histories of NHIS-LMF respondents.

Here, I present two hypothetical cases of respondents aging across the person-period data, and the Lexis form of the APC cells composing the collapsed data structure. The hypothetical "green" respondent enters the data in the 1997 NHIS survey at age 52 and is right-censored from the data on December 31, 2006 at the age of 61. That is, because no death record is matched to the "green" respondent, she is assumed to be alive on December 31$^{st}$, 2006 and is therefore right-censored from the data at age 61. In the APC data structure she occupies the 1945-1949 birth cohort, three time periods (1995-1998; 1999-2002; and 2003-2006), and three age groups (50-54; 55-59; and 60-64). In all, she contributes five APC combinations to the data, five corresponding aggregated exposure times of life lived across these cells, and no event of death. Conversely, the "red" respondent enters the data in the 1986 NHIS survey at the age of 55 and dies in 1995 at the age of 64. In the APC structure he occupies the 1930-1934 birth cohort, three time periods

(1986-1990; 1991-1994; and 1995-1998), and two age groups (55-59 and 60-64), and contributes four APC combinations to the data. Furthermore, the "red" respondent contributes his own respective specific aggregated exposure times of life lived in these cells as well as one event of death.

Evidence of sufficient overlap of age and cohort is clearly apparent in Figure 1.3, in that for every age-period combination there are two, and sometimes three, birth cohorts represented. In Figure 1.3 there are a total of 39 unique APC combinations covering age ranges 50-54; 55-59; and 60-64. In subsequent chapter-specific analyses using the full age-ranges of the collapsed NHIS-LMF 1986-2006, there are anywhere from 126 to 168 APC cells. The reasons for the different cell counts will be explained in each chapter.

### 1.6 Outline of Dissertation

In all chapters of this dissertation I draw from existing theory to advance a cohort perspective of U.S. adult mortality risk. I then apply the HAPC-CCREM to the NHIS-LMF 1986-2006 data to illustrate the importance of cohort forces in driving changes in U.S. mortality rates. I do so for three important topics pertaining to U.S. adult mortality: (1) the U.S. black-white crossover in older adult mortality risk, (2) the association between educational attainment and U.S. adult mortality risk, and (3) the origins and persistence of racial disparities in U.S. adult mortality risk. I briefly introduce each topic below.

*Chapter 2*

In Chapter 2 of my dissertation I argue that the convergence and "crossover" of mortality rates of the U.S. black and white populations at the oldest-old ages (85+) reflect racial

differences in mortality selection across U.S. birth cohorts. As such, once I control for these cohort effects the average age-specific mortality rates of the U.S. black population should remain higher than the average age-specific mortality rates of the U.S. white population at every age group. Results from my HAPC-CCREM analyses of the NHIS-LMF 1986-2006 provide evidence strongly supporting this hypothesis. Indeed, when controlling for variation 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, consistent with existing research, results show that nearly all the recent temporal change in U.S. adult mortality risk was cohort driven (Yang 2008). 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 these different experiences are driving the convergence and crossover of mortality risk at older ages.

*Chapter 3*

In Chapter 3 of my dissertation I draw from the life course perspective, the theory of "technophysio evolution" (Fogel and Costa 1997), and the notion of a "cohort morbidity phenotype" (Finch and Crimmins 2004) to argue that the adult environment is growing more important in shaping U.S. adult mortality risk across birth cohorts. Because the adult environment is becoming more important, I further argue that human capital and resources associated with human capital are becoming increasingly important for navigating this adult environment and procuring greater knowledge of, access to, and use of health-related resources and technologies. As such, I hypothesize and find that education is growing more strongly associated with U.S. adult mortality risk across U.S. birth cohorts. Hierarchical cross-classified

random effects models are used to simultaneously measure age, period, and cohort patterns of mortality risk between 1986 and 2006 for non-Hispanic white and non-Hispanic black men and women with a less than high school education, a high school education, and a more than high school education, respectively. All-cause mortality risk and mortality risk from heart disease, lung cancer, and "unpreventable" cancers are examined. Findings reveal that temporal reductions to black and white male and female adult mortality rates were driven entirely by cohort changes in mortality. Findings also demonstrate that disparate cohort effects between education groups widened the education gap in all-cause mortality risk and mortality risk from heart disease and lung cancer across this time period. Educational disparities in mortality risk from unpreventable cancers, however, did not change. Consistent with fundamental cause theory, this research uncovers widening educational differences in adult mortality and supports the contention that a cohort perspective is most appropriate for understanding recent temporal changes in U.S. mortality risk.

*Chapter 4*

In Chapter 4, I analyze cohort changes to racial disparities in U.S. adult mortality rates, paying special attention to the intersection of race/ethnicity, socioeconomic resources, and historical changes in early-life conditions. Racial differences in U.S. adult mortality are often attributed to unequal distributions of socioeconomic resources between white and black populations. If socioeconomic resources are indeed "fundamental causes" of health and mortality disparities, then much of the black-white gap in adult mortality should be explained by disparities in white's and black's educational attainment and income during adulthood (Link and Phelan 1995, 1996). Thus far, however, empirical analyses of the mediating effects of

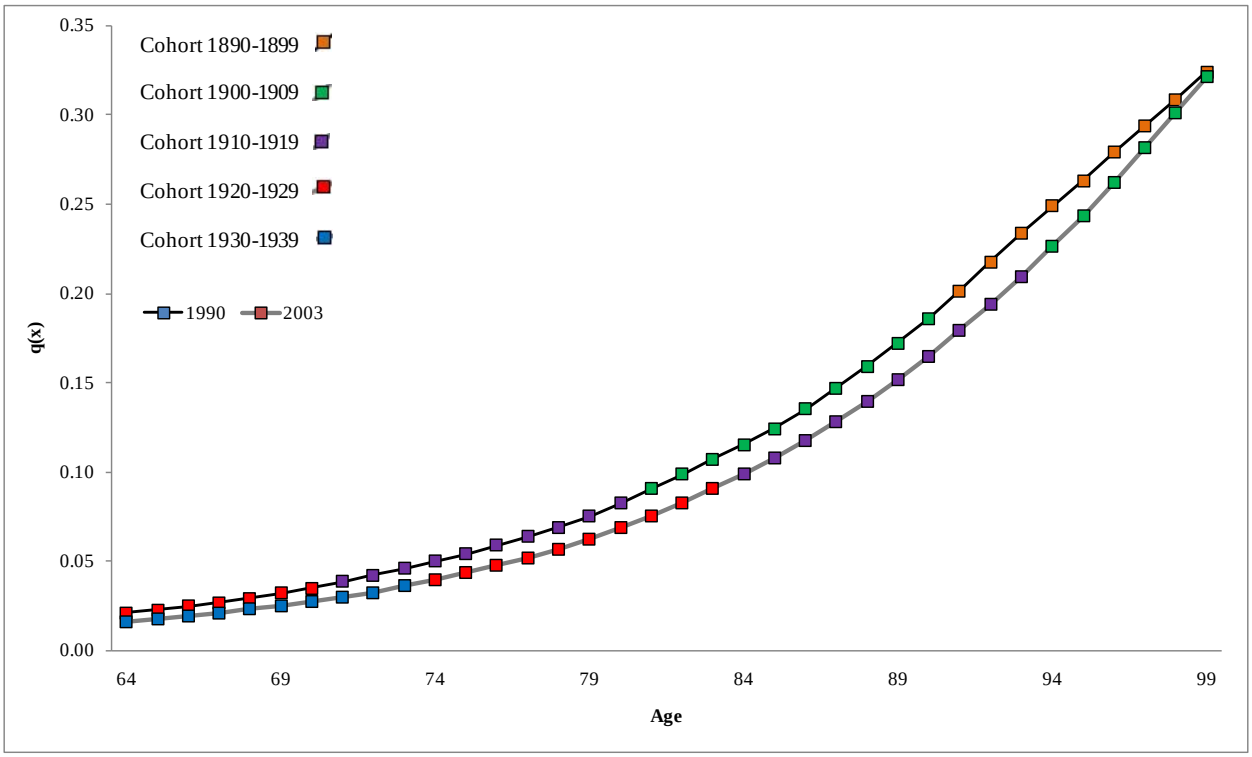
socioeconomic resources on U.S. racial disparities in mortality have provided inconclusive evidence. It also remains unresolved whether black and white populations derive significantly different health benefits from socioeconomic resources. Does the "color line" in U.S. mortality simply reflect unequal distributions of socioeconomic resources, or does it run much deeper (Du Bois 1903)? With regards to U.S. adult mortality risk, which is the more *fundamental* "fundamental social cause," race or socioeconomic status (SES)? Using a life course framework and the theory of technophysio evolution, I argue that racial differences in U.S. adult mortality stem from lifelong and historical processes of black disadvantage in both health and socioeconomic resources. While black-white disparities in adulthood SES are an important component of this disadvantage, the disadvantage is heavily rooted in cumulative life course and historical processes of racial stratification in the United States. Consequently, analyzing the black-white gap in adult mortality by looking only at racial inequalities in adult SES fails to capture many the long-term stratification processes driving black disadvantages in adult health and socioeconomic resources. In this chapter I shed light on some of these stratification processes by examining age, period, and cohort patterns of the U.S. black-white gap in adult mortality, and testing how cohort variation in early-life conditions and individual SES in adulthood affect these patterns. I first compare historical changes in U.S. black and white early-life conditions. I then employ Bayesian hierarchical age-period-cohort (HAPC) cross-classified random effects models (CCREM) to analyze age, period, and cohort patterns of black and white men's and women's U.S. adult mortality between 1986 and 2006. I then show that racial differences in cohort reductions in mortality are partly due to historical inequalities in U.S. black and white cohorts' childhood living conditions. Next, I investigate the effects that adulthood education, income, and poverty have on the changing (or persisting) black-white gap in U.S.

adult mortality. Results suggest that, in terms of adult mortality risk, black and white Americans derive significantly different benefits from socioeconomic resources in adulthood, and that these differences are changing across birth cohorts. Overall, these findings suggest that the black-white gap in U.S. adult mortality reflects more than just racial inequalities in the distribution of socioeconomic resources, and instead implicates processes consistent with cumulative disadvantage theory. The adult mortality risks for U.S. black men and women remained significantly and stagnantly higher than risks for U.S. white men and women between 1986 and 2006, and these differences stem from longstanding racial inequalities in childhood, racial inequalities in socioeconomic resources in adulthood, and racial inequalities in the ability to transfer these resources into lower mortality risk.

*Chapter 5*

I conclude my dissertation with a brief recap of the theory and findings in each chapter, as well as a discussion of the dissertation's limitations. I then allude to my future research agenda. Overall, results from analyses in my dissertation provide evidence strongly supporting my contention that cohort forces are driving recent U.S. adult mortality patterns and trends. In each chapter the shortcomings of a period perspective of mortality are articulated, exposed, and shown with regards to patterns and trends of disparities in U.S. adult mortality. While the NHIS-LMF 1986-2006 data are limited in a number of ways, results in this dissertation add to a growing literature suggesting the need to incorporate a life course and cohort perspective into empirical studies of U.S. mortality trends.

**Figure 1.1: U.S. White Men's Age-specific Probability of Death, 1990 and 2003.**




**Source: NVSS, http://www.cdc.gov/nchs/products/life_tables.htm**

**Figure 1.2: Cohort Composition across Age Range of NHIS-LMF 1986-2006.**

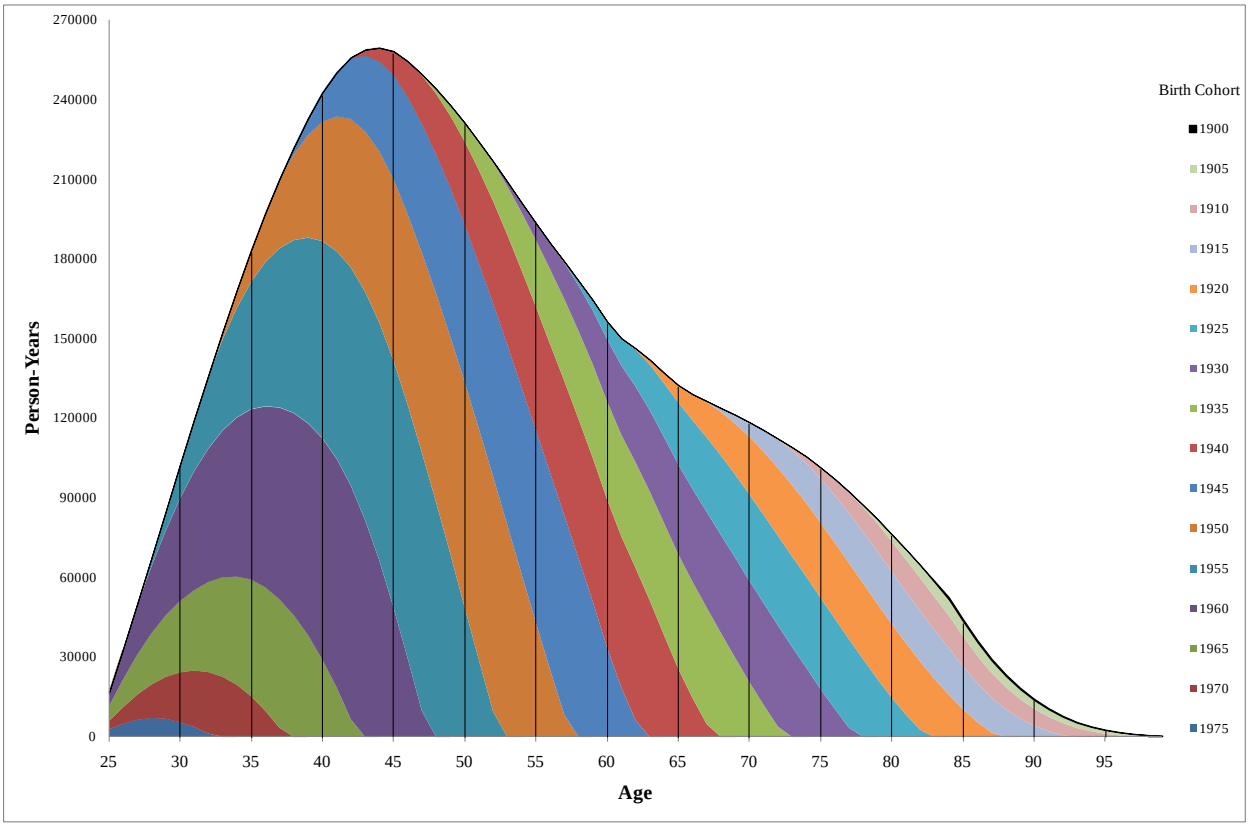
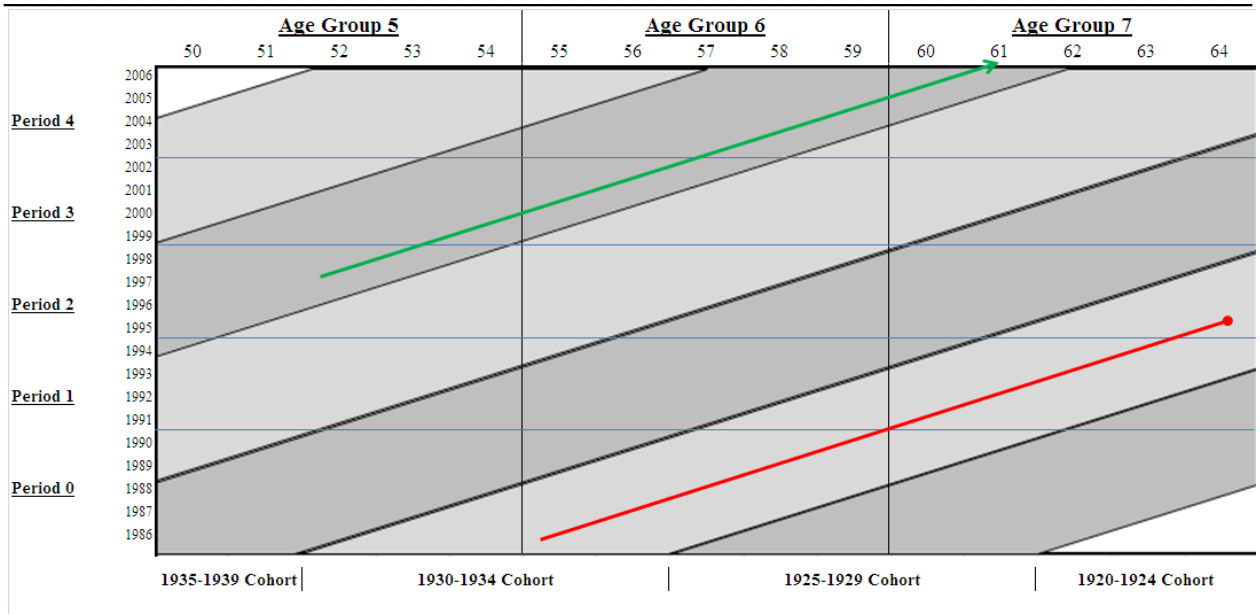


**Figure 1.3: Lexis Diagram of NHIS-LMF 1986-2006 APC Data Structure with Two Hypothetical NHIS Respondents' Survival Histories**




Note: The arrow on the green survival history represents the respondent being censored from the data on December 31st, 2006, while the circle on the red survival history represents a death.

# CHAPTER 2

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

### 2.1 Introduction

Analyzing old-age mortality risk of the U.S. population has proved difficult for some time. Sparse data in older-old (85+) age groups frequently preclude consistently 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 over 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 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 recently and repeatedly found to exist at around age 85 for both men and women, although the female crossover generally occurs at later ages (Kestenbaum 1992; Lynch, Brown and Harmsen 2003; Johnson 2000; Parnell and Owens 1999; Arias 2007).

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 (Manton and Stallard 1984; Vaupel, Manton and Stallard1979; Vaupel and Yashin 1985; Lynch et al. 2003; Nam 1995). Upon reaching older age groups the population that experienced higher mortality risk across the life course will be relatively composed of more robust members at these advanced ages, while 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 (Preston, Elo, Rosenwaike and Hill 1996; Preston, Elo and Stewart 1999; Preston and Elo 2006; Coale and Kisker 1986, 1990; Hussey and Elo 1997). This idea suggests that the mortality crossover is a product of age misreports, unmatched or uncounted deaths, and/or other data inaccuracies. Once data problems are accounted for, convergence of mortality risk is delayed to much older ages or eliminated altogether (Preston et al. 1996; Lynch et al. 2003).

While both explanations have been supported by existing research, arguments are developed below 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 black and white U.S. populations. As Ben-Shlomo and Kuh (2002) affirm, "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" (290). 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 make use of the 1986-2006 National Health Interview Survey Linked Mortality Files (NHIS-LMF) to analyze the mortality experiences of the older adult black and older adult white U.S. populations. The NHIS-LMF is a powerful data set that allows for understanding the extent to which cohort processes affect the mortality risk of the U.S. black and white male and female 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 dataset. 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 of black and white sex-specific 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 chapter supports this contention with regards 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.

## 2.2 Background

Mortality crossovers have been documented to occur in human populations in many countries and under many mortality schedules (Coale and Kisker 1986, 1990). Such crossovers have also been demonstrated to exist in nonhuman populations in similar ways (Nam 1995). In the United States, Arias (2007) documents 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) have 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 (Lynch et al. 2003; Eberstein, Nam and Heyman 2008).

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 (Manton and Stallard 1981; Vaupel et al 1979; Vaupel and Yashin 1985; Lynch et al. 2003). Eberstein et al.'s (2008)

recent findings most forcefully suggest the plausibility of the heterogeneity argument. By examining black and white old-age mortality rates by cause of death, the authors demonstrate that convergence and crossovers of mortality rates exist only for heart disease, cerebrovascular disease, influenza/pneumonia, and residual causes of death. For all other causes of death, 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 conclude 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 are drifting upwards in age (see Figure 2.1). Data from the early 1960s recorded male and female crossovers between white and nonwhite populations to have respectively occurred at ages 75 and 77 (Kestenbaum 1992). About ten years later the NCHS life table for 1969-1971 recorded the black-white crossovers to occur at ages 78 and 80 (due to space limitations women's crossovers are not shown in Figure 2.1). The most recent official estimates of U.S. mortality risk have found male and female crossovers between the white and black populations to occur, respectively, at ages 88 and 87 (Arias 2007).

The fact that the ages at which the crossover in mortality risk occurs are increasing suggests that cohort and/or period effects ought to 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 specifically argue that differences between black and white cohorts' cumulative exposures to changing health and mortality conditions over 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 over time.

### 2.2.1 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, 1978, and 1987) 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 to 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 (Cutler et al. 2006; Meara, Richards, and Cutler. 2008). For example, official U.S. life tables are most commonly offered in period format (as presented in Figure 2.1), projecting life expectancy of "synthetic" cohorts across their ages. While 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 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 between race/ethnicity, age, and mortality. Most life course research on the relationship between 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 race/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, but 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 differed in their lifetime exposures to risk factors such as years spent smoking. Further, race/ethnic differences in U.S. cohorts' exposures to health improvements and risk factors will invariably influence these race/ethnicities' cohorts' morbidity and mortality risks across their respective life courses.

These observations about cohort-specific life courses are 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) 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 race/ethnicity has played in shaping cohort-specific life course experiences of health and mortality in the United States is not fully understood. If life course risk factors of morbidity and mortality have indeed been changing across cohorts, it is likely that the changes have profoundly differed 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 (House et al. 1994; Blackwell, Hayward and Crimmins 2001; Beckett 2000; Ross and Wu 1995; Lynch 2003; Hayward et al. 2000). 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 and Phelan 1995; Link 2008; Pampel 2001). 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, race/ethnic cohort differences in education and other socioeconomic variables associated with risk factors (Lynch 2003), access and utilization of healthcare technologies (Glied and Lleras-Muney 2008), social support (Ross and Wu 1996), 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,

Stallard and Corder 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 (e.g., chronic obstructive pulmonary disease and lung cancer) (Jemal et al. 2005; CDC 1999; 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 (Manton et al. 1997; Eberstein et al. 2008). Thus, how different U.S. white and U.S. 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 – compositional changes in causes of death on one hand, and changes to race/ethnic-based differences in socioeconomic resources and living conditions 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; Easterlin 1997), smaller families, and improved nutrition (Fogel 2004, 2005; Fogel and Costa 1997) – are likely to be 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 these cohorts' elevated exposures to harsh early-life conditions, as well as disparate exposure to subsequent benefits of health-enhancing knowledge and technologies. Table 2.1 contains the general timing of a select number of important advances in nutrition and public health efforts (Jayachandran et al. 2010; Cutler and Miller 2005; Manton et al. 1997), 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; Hayward et al. 2000; Montez and Hayward 2010). Consistent with the points above, there are two themes to take away from Table 2.1. First, compared to subsequent birth cohorts, black 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. Secondly, 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 2005), malnutrition (Fogel 2005; Ben-Shlomo and Kuh 2004), and other childhood hardships (Montez and Hayward 2011) significantly raises 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 amongst 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, we should observe greater difference in older age heterogeneity between the race/ethnic groups, and thus a greater deal of black-white differences in mortality risk as well. This is because as 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, mortality research should revisit Riley's and Ryder's work on cohorts and move beyond one said "life course" to highlight cohorts' varying experiences in aging, health, and mortality. We can speak only limitedly of *a* life course in the same way we can speak limitedly of "life expectancy" calculated from period life tables. In both cases, we are favoring the cumulative sum over the heterogeneous parts. That is, we are speaking of an aggregated experience – a mortality schedule on one hand, and a single life course on the other – without acknowledging that the aggregation is composed of many different cohort experiences (Elwert and Winship 2010; Xie 2007). 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 American black and white cohorts that comprise those older age groups. In this chapter I account for disparate cohort effects of the black and white populations to see if the black-white crossover in U.S. mortality risk can be explained by cohort, not age, phenomena.

### 2.3 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 analysis proceeds 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 in order to determine the ages at which the sex-specific mortality crossovers occur in these data. Mortality risk was also estimated

from models using five-year age groupings. These five-year models were estimated in order to derive baseline comparisons for subsequent age-period-cohort (APC) analyses. I then make several adjustments to the single-year data in order to reduce the likelihood of poor data quality bias, especially amongst the older age groups. These adjustments are fourfold. One, NHIS respondents who relied on proxy reports were deleted from the sample. Two, 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. Three, respondents with missing values of educational attainment were deleted from the sample. And four, respondents over the age of 75 years at time of survey were 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 if the crossover stemmed from poor data quality bias. As a final step, I conduct APC analyses of adult mortality rates to account for disparate cohort and period effects in the age-specific mortality estimates. Specifically, I use hierarchical age-period-cohort (HAPC) cross-classified random effects models (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).

### 2.3.1 Data

I use 19 cross-sectional waves of the National Health Interview Survey (NHIS) 1986 through 2004, linked to the National Death Index (NDI) 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 31st, 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 (NCHS 2009). 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 live 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 their death because their self-reported age of 74 was recorded earlier in history. 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 utilizes data from all non-Hispanic black and all non-Hispanic 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 restricting the sample to non-Hispanic white and non-Hispanic 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 373,664 cases, a black female sample of 81,389 cases, and a white female sample of 413, 831 cases (see Table 2.2).

These race/ethnic-sex stratified samples were then transformed into person-period data to account for subsequent mortality risk from time of survey until December 31, 2006. Almost 20 percent of the black male NHIS sample was linked to a subsequent death across this time, versus about 17 percent of the white male NHIS sample. Almost 15 percent of the black female NHIS sample experienced a death before 2007, versus 14.69 percent of the white female sample. 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, in order to improve the quality of data amongst 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 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

[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.

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 2.3.

In my third analysis I estimate age-period-cohort (APC) models of black and white mortality risk in the NHIS-LMF 1986-2006. To do so, I collapsed the *Data A* samples into 137 cells of 5-year age-period-cohort blocks (due to 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 2.2. The data were collapsed into 5yr age X 5yr period X 5yr cohort cells for two primary reasons: (1) sparse mortality counts in the black men's and women's individual-level samples preclude stable APC modeling, and (2) to break the linear dependency between age, period, and cohort (Glenn 2005).

### 2.3.2 Methods

#### 2.3.2.1 *First Analysis: Age-specific Mortality Risk in Data A and Data C*

Using *Data A*, I use SAS 9.2 to fit fixed effects discrete-time hazard models to estimate age-only 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 complimentary-log-log transformation to make linear the binomial response mean as a generalized linear model (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 APC models. In these initial *Data C* analyses I use fixed effects log-linear models to estimate the 5-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.

#### 2.3.2.2 *Second Analysis: Age-specific Mortality Risk in Adjusted Data B*

Using the adjusted NHIS-LMF 1986-2006, or *Data B*, I re-estimate 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 we should likely see the age at which the

sex-specific crossovers occur to be higher in *Data B* than in *Data A* (Preston et al. 1996; Preston et al. 1999; Hill, Preston and Rosenwaike 2000; Preston and Elo 2006).

#### 2.3.2.3 *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 hierarchical age-period-cohort (HAPC) models for repeated cross-section survey data (Yang and Land 2006, 2008). These methods utilize a cross-classified random-effects model (CCREM) to embed each NHIS-LMF respondent within both a time period and birth cohort at a given age. Because the NHIS-LMF dataset follows individual mortality risk as respondents age across periods, each respondent can occupy several age-period-cohort combinations. Consequently, while 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, 1976). 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:

Level-1 within cell model: $$\ln(E[D_{ijk}]) = \alpha_{jk} + \beta_{jk} A_i + \ln(R_{ijk})$$

where $D_{ijk}$ denotes the counts of deaths of the $i$th age group for $i = 1, \ldots, n_{jk}$ age groups within the $j$th period for $j = 1, \ldots, J$ time period and the $k$th cohort for $k = 1, \ldots, K$ birth cohort; $A_i$ denotes the dummy five-year age groups $1,\ldots, n_{jk}$; $\alpha_{jk}$ is the intercept indicating the reference age

group (50-54) who was in period *j* and belong to cohort *k*; and ln($R_{ijk}$) is the natural log of the aggregated exposure time lived during the five-year age-period-cohort cell.

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

in which $\alpha_{jk}$ specifies that the fixed age effects vary from period to period and from cohort to cohort. $\pi_0$ is the expected mean at the reference age (50-54) averaged over all periods and cohorts; $t_{0j}$ is the overall 5-year period effect averaged over all five-year birth cohorts with variance $\sigma_{t0}$; and $c_{0k}$ is the overall 5-year cohort effect averaged over all five-year periods with variance $\sigma_{k0}$.

I combine the level-1 and level-2 models to estimate counts of deaths in each 5-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. Due to 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 (see Appendix). Second, I reran 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.

## 2.4 Results

Table 2.4 presents *Data C* estimates of fixed effects 5-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 insignificant at all commonly used α-levels. To observe the black-white mortality crossover in these 5-year collapsed NHIS-LMF 1986-2006 data, I plot the estimated logged mortality rates for each male and female race/ethnicity sample. In these 5-year data we see that for each sex the age-specific mortality risk of the two race/ethnic subpopulations converge and then crossover at around age 85 (see the right panel in Figure 2.2).

In the left panel of Figure 2.2 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* 5-year results, and all estimates are significant at the .001 α-level. For men, 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 Figure 2.2 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 re-estimated the single-year discrete-time hazard models of mortality risk on the adjusted *Data B* NHIS-LMF 1986-2006. Results from these data-adjusted analyses are graphically depicted in Figure 2.3. Here we see the single-year age estimates of black and white men's and women's log mortality risk plotted across age.

Despite improving our confidence in the samples' estimates of older age mortality risk, we see that the black-white crossover in each sex persists in *Data B.* That is, the black and white male NHIS-LMF 1986-2006 samples restricted to self-reporting respondents age 25-75 at time of survey still generate a black-white mortality crossover at age 84. 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. This largely stems from relative black-white differences in the changes to cohort composition of the older age groups. This is apparent in Table 2.5 below, in which we see that the age range 75-89 in *Data B* is relatively composed of more recent cohorts than is the same age range in *Data A.* However, relative to the black subsample, the adjusted *Data B* make the white subsample more composed of older cohorts. Because the white sample retains a greater degree of older cohort representation at the older ages, the reduction in mortality risk between the full sample and the adjusted sample is not as big as the reduction in the black sample. The ultimate result is to drive the age at which the crossover occurs downward.

Next, Table 2.6 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/ethnic-sex populations (Yang 2008). These findings are best depicted in Figure 2.4, in which we see a great deal of cohort variation in adult mortality risk for all four race/ethnicity-sex subpopulations (the period effects are insignificant and thus are not shown).

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 makeup 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 and Phelan 1995; Link 2008). 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 Figure 2.5, the patterns of estimated age effects on mortality for the non-Hispanic white and non-Hispanic black sex samples have significantly and remarkably changed from Figures 2.2 and 2.3.

After controlling for disparate cohort and period effects, the age effects on mortality rates in both the male and female black samples remains 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 cohort are plotted for each black and white, male and female model, we see patterns of age-specific log mortality patterns similar to those seen in Figure 2.2. 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 Figure 2.6, 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.

Specifically, Figure 2.6 displays the ratio of fitted age-specific black mortality rates to fitted age-specific white mortality rates, age-groups 60-64 to 90-94, by birth cohort. Due to insignificant effects for the age group 95-99, results at these ages are not included in the graph. In Figure 2.6 we can see for the male sample that no crossover exists for any cohort born after the 1910-1914 time period. That is, for all U.S. cohorts born in 1915 or later, black male fitted mortality rates between 1986 and 2006 were 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 we also see that the fitted black male mortality rates for the 1915 and 1920 cohorts are higher than the fitted white male mortality rates from the same birth cohorts. In short, while we observe black-white mortality crossovers 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.

## 2.5 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; Ryder 1965; Riley 1973, 1978, 1987). The question over 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. And further complicating these processes is the unequal ways that these cohort processes are conditioned by both gender and race.

In this study I showed 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 utilize data with these structures to assure they are accounting for both life course and temporal shifts in heterogeneity. Second, while 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, but 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 time of 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. Nevertheless, 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 (Mason and Cope 1987; Lynch 2003). 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 due 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* 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 to adult mortality risk in the United States, there are some limitations. First, the time period 1986-2006 is a small window to simultaneously analyze the forces of age, period, and cohort on U.S. adult mortality risk. Secondly, 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. While 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 (Ryder 1965; Riley 1987; Ben-Shlomo and Kuh 2002). Only by building a cohort perspective into life course analyses of mortality risk can we fully consider such contextual effects.

**Table 2.1. 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 | Chlorinization of Water | Vitamin D Fortification |
| | | 1st Public Health School | Cod Liver Oil | Vitamin B6 | Vitamin B3 Fortification |
| | | Malaria Control | Irridation of Milk | Food Relief Programs | Modern Sewage Treatment |
| | | | Iodization of Salt | Rural Sanitation | Rural Sanitation |
| | | | Maternal & Infancy Act | Sulfa Drugs | Penicillin |
| 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.

**Table 2.2. 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-'99) 2.40 | (1995-'99) 2.38 | (1995-'99) 2.41 | (1995-'99) 2.38 |
| Mean Cohort | (1945-'49) 10.43 | (1940-'44) 9.89 | (1945-'49) 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 | 9897.63 | 63,368.30 | 14,069.86 | 66,619.60 |
| n | 137 | 137 | 137 | 137 |

**Table 2.3. 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 |

**Table 2.4. GLM Fixed Effects Age Only Models 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 |
| | (.183) | (.141) | (.293) | (.222) |
| 30-34 | -1.326 | -1.541 | -1.635 | -1.789 |
| | (.068) | (.049) | (.082) | (.070) |
| 35-39 | -1.159 | -1.253 | -1.266 | -1.278 |
| | (.045) | (.029) | (.050) | (.037) |
| 40-44 | -0.846 | -0.876 | -0.967 | -0.860 |
| | (.037) | (.021) | (.040) | (.027) |
| 45-49 | -0.527 | -0.453 | -0.459 | -0.428 |
| | (.035) | (.019) | (.036) | (.024) |
| 50-54 | Ref. | Ref. | Ref. | Ref. |
| 55-59 | 0.291 | 0.419 | 0.284 | 0.484 |
| | (.035) | (.018) | (.037) | (.022) |
| 60-64 | 0.683 | 0.919 | 0.735 | 0.989 |
| | (.036) | (.017) | (.037) | (.022) |
| 65-69 | 1.092 | 1.396 | 1.088 | 1.429 |
| | (.035) | (.017) | (.036) | (.021) |
| 70-74 | 1.495 | 1.866 | 1.431 | 1.896 |
| | (.035) | (.016) | (.036) | (.019) |
| 75-79 | 1.781 | 2.297 | 1.840 | 2.355 |
| | (.039) | (.016) | (.036) | (.019) |
| 80-84 | 2.178 | 2.745 | 2.184 | 2.849 |
| | (.047) | (.017) | (.040) | (.019) |
| 85-89 | 2.504 | 3.250 | 2.544 | 3.362 |
| | (.086) | (.024) | (.055) | (.021) |
| 90-94 | 2.838 | 3.615 | 2.871 | 3.877 |
| | (.254) | (.070) | (.121) | (.034) |
| 95-99 | *2.373 | 3.671 | 2.884 | 4.132 |
| | (1.458) | (.437) | (.557) | (.139) |
| **Intercept** | -4.561 | -5.268 | -4.987 | -5.757 |
| **Model Fit** | | | | |
| -2LL | 1134.8 | 1783.4 | 1011.7 | 1500.3 |
| $\chi^2$/df | 2.63 | 6.41 | 1.66 | 4.33 |
| N | 137 | 137 | 137 | 137 |

* Not Significant

**Table 2. 5: Cohort Composition of Age Groups 75-79, 80-84, and 85-89 by Race and Data, NHIS-LMF 1986-2006**

| | Ages 75-79 | | | Ages 80-84 | | | Ages 85-89 | | |
|---|---|---|---|---|---|---|---|---|---|
| | Data A | Data B | % Change | Data A | Data B | % Change | Data A | Data B | % Change |
| *Black Men* | | | | | | | | | |
| 1900-1904 | --- | --- | --- | --- | --- | --- | 3.78 | 0.00 | **-100** |
| 1905-1909 | --- | --- | --- | 8.53 | 0.00 | **-100** | 17.55 | 0.00 | **-100** |
| 1910-1914 | 8.92 | 5.32 | **-40.36** | 19.15 | 8.53 | **-55.46** | 30.08 | 20.67 | **-31.30** |
| 1915-1919 | 20.66 | 19.30 | **-6.58** | 29.65 | 31.06 | **4.76** | 39.59 | 61.46 | **55.24** |
| 1920-1924 | 31.91 | 33.62 | **5.36** | 35.55 | 49.75 | **39.94** | 9.01 | 17.87 | **98.38** |
| 1925-1929 | 32.44 | 35.92 | **10.73** | 6.35 | 10.66 | **67.87** | --- | --- | --- |
| 1930-1934 | 5.22 | 5.84 | **11.88** | --- | --- | --- | --- | --- | --- |
| *White Men* | | | | | | | | | |
| 1900-1904 | --- | --- | --- | --- | --- | --- | 2.85 | 0.00 | **-100** |
| 1905-1909 | --- | --- | --- | 7.36 | 0.00 | **-100** | 15.36 | 0.00 | **-100** |
| 1910-1914 | 8.68 | 5.49 | **-36.75** | 19.14 | 8.82 | **-53.92** | 30.81 | 18.95 | **-38.51** |
| 1915-1919 | 20.79 | 19.93 | **-4.14** | 30.16 | 32.48 | **7.69** | 41.38 | 63.06 | **52.40** |
| 1920-1924 | 31.90 | 32.96 | **3.32** | 36.63 | 49.04 | **33.88** | 9.60 | 17.99 | **87.40** |
| 1925-1929 | 32.65 | 35.90 | **9.95** | 6.06 | 9.65 | **59.24** | --- | --- | --- |
| 1930-1934 | 5.21 | 5.71 | **9.60** | --- | --- | --- | --- | --- | --- |

Note: Numbers in cells are percents.

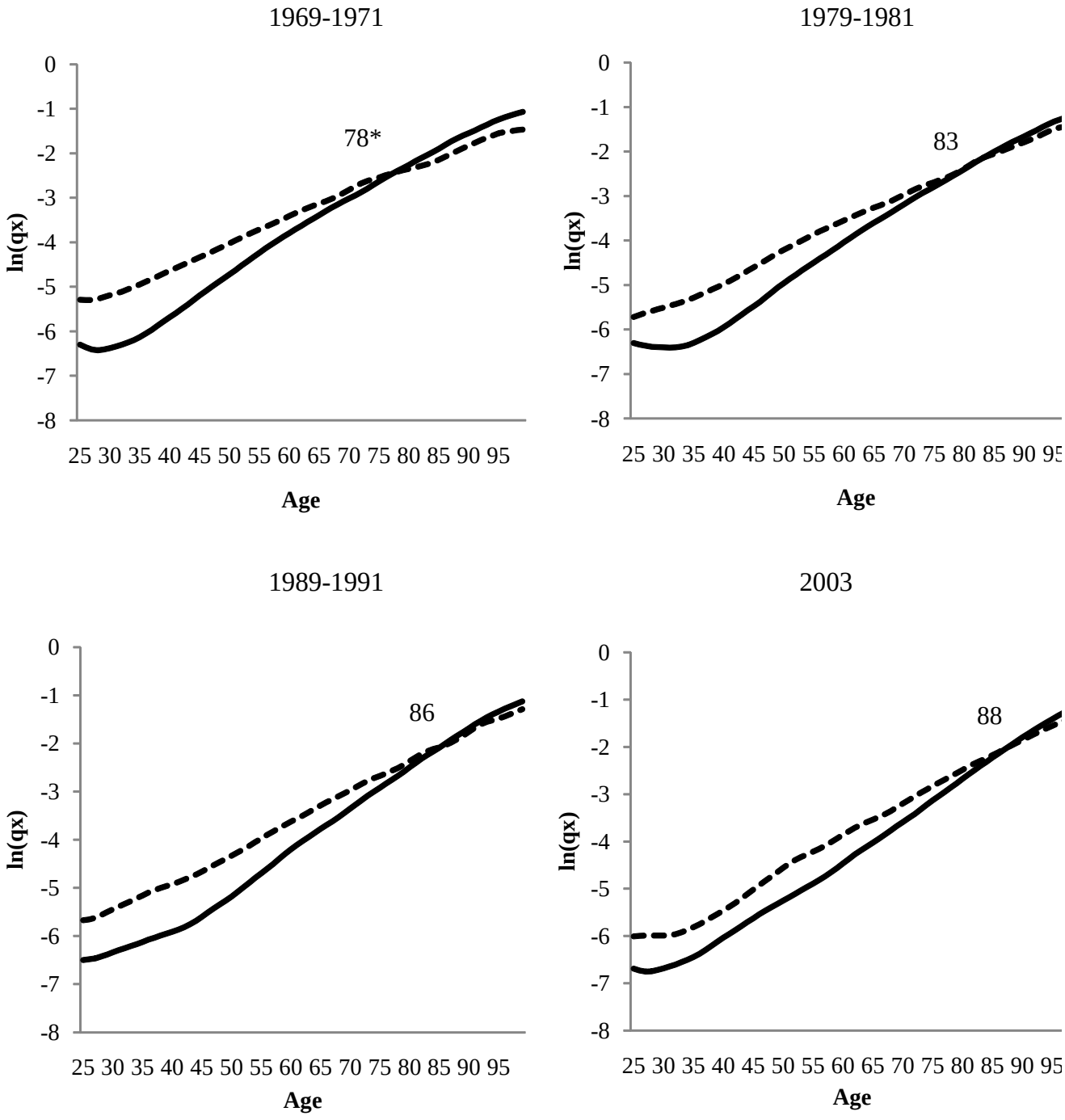
**Table 2.6. HAPC-CCREMs of U.S. non-Hispanic Black and non-Hispanic White Men's and Women's Adult All-cause Mortality Rates, 1986-2006**

| | Men | | Women | |
|---|---|---|---|---|
| ***Fixed Effects*** | **Black** | **White** | **Black** | **White** |
| **Age** | | | | |
| 25-29 | -.826 | -.934 | -1.760 | -1.495 |
| | (.195) | (.151) | (.308) | (.232) |
| 30-34 | -.867 | -1.018 | -1.256 | -.1374 |
| | (.088) | (.066) | (.113) | (.089) |
| 35-39 | -.777 | -.858 | -.995 | -.962 |
| | (.063) | (.044) | (.078) | (.056) |
| 40-44 | -.591 | -.614 | -.785 | -.646 |
| | (.049) | (.032) | (.059) | (.041) |
| 45-49 | -.426 | -.333 | -.412 | -.291 |
| | (.040) | (.023) | (.044) | (.029) |
| 50-54 | Ref. | Ref. | Ref. | Ref. |
| 55-59 | .254 | .297 | .188 | .297 |
| | (.042) | (.022) | (.045) | (.027) |
| 60-64 | .523 | .595 | .487 | .586 |
| | (.053) | (.029) | (.059) | (.035) |
| 65-69 | .843 | .848 | .767 | .802 |
| | (.062) | (.036) | (.073) | (.042) |
| 70-74 | 1.167 | 1.109 | 1.056 | 1.088 |
| | (.069) | (.043) | (.085) | (.048) |
| 75-79 | 1.400 | 1.353 | 1.412 | 1.382 |
| | (.078) | (.049) | (.090) | (.054) |
| 80-84 | 1.724 | 1.639 | 1.711 | 1.736 |
| | (.089) | (.056) | (.112) | (.061) |
| 85-89 | 2.036 | 1.988 | 2.040 | 2.110 |
| | (.123) | (.066) | (.131) | (.068) |
| 90-94 | 2.365 | 2.222 | 2.322 | 2.493 |
| | (.277) | (.099) | (.182) | (.080) |
| 95-99 | *1.911 | 2.175 | 2.259 | 2.642 |
| | (1.468) | (.446) | (.582) | (.162) |
| ***Random Effects*** | | | | |
| **Cohort** | | | | |
| 1970-1974 | -.595 | -1.119 | -.812 | -.822 |
| | (.185) | (.241) | (.265) | (.252) |
| 1965-1969 | -.686 | -.957 | -.461 | -.877 |
| | (.138) | (.204) | (.190) | (.198) |
| 1960-1964 | -.513 | -.819 | -.513 | -.721 |
| | (.121) | (.198) | (.173) | (.186) |
| 1955-1959 | -.265 | -.628 | -.205 | -.715 |
| | (.117) | (.195) | (.165) | (.183) |

| *Table 2.6 cont.* | Men | | Women | |
|---|---|---|---|---|
| | **Black** | **White** | **Black** | **White** |
| **Cohort (cont.)** | | | | |
| 1950-1954 | -.060 | -.485 | -.184 | -.624 |
| | (.115) | (.194) | (.161) | (.182) |
| 1945-1949 | -.119 | -.421 | -.169 | -.447 |
| | (.114) | (.193) | (.158) | (.180) |
| 1940-1944 | .037 | -.247 | .054 | -.253 |
| | (.113) | (.193) | (.156) | (.180) |
| 1935-1939 | .103 | -.038 | .142 | -.005 |
| | (.113) | (.193) | (.155) | (.179) |
| 1930-1934 | .227 | .174 | .190 | .178 |
| | (.114) | (.193) | (.156) | (.179) |
| 1925-1929 | .119 | .400 | .247 | .383 |
| | (.115) | (.193) | (.159) | (.180) |
| 1920-1924 | .379 | .572 | .316 | .518 |
| | (.117) | (.194) | (.162) | (.181) |
| 1915-1919 | .397 | .768 | .331 | .673 |
| | (.124) | (.195) | (.169) | (.182) |
| 1910-1914 | .411 | .919 | .368 | .815 |
| | (.142) | (.178) | (.179) | (.184) |
| 1905-1909 | .404 | 1.023 | .483 | .912 |
| | (.197) | (.205) | (.209) | (.188) |
| 1900-1904 | .052 | .858 | .214 | .984 |
| | (.372) | (.360) | (.443) | (.251) |
| **Period** | | | | |
| 2005-2009 | .008 | .224 | .103 | .282 |
| | (.013) | (.080) | (.049) | (.110) |
| 2000-2004 | -.007 | .101 | .034 | .174 |
| | (.012) | (.079) | (.044) | (.109) |
| 1995-1999 | -.005 | -.004 | .006 | .013 |
| | (.012) | (.079) | (.043) | (.109) |
| 1990-1994 | .005 | -.096 | -.055 | -.154 |
| | (.013) | (.079) | (.048) | (.110) |
| 1985-1989 | -.001 | -.225 | -.088 | -.314 |
| | (.014) | (.085) | (.066) | (.115) |
| **Intercept** | -4.488 | -4.910 | -4.867 | -5.367 |
| **Covariance Parameters** | | | | |
| Cohort | .154 | .542 | .320 | .470 |
| | (.075) | (.219) | --- | (.190) |
| Period | 1.98E-4 | .031 | .007 | .059 |
| | (.001) | (.023) | (.009) | (.043) |
| **Model Fit** | | | | |
| *-2LPL* | 123.00 | -65.51 | 86.44 | -57.41 |
| $\chi^2$/df | 1.38 | 1.00 | 1.07 | .99 |
| N | 137 | 137 | 137 | 137 |

Note: Numbers in parantheses are standard errors * Not Significant

**Figure 2.1: Logged Age-specific Adult Mortality Risk of U.S. Black and White Male Populations at Four Time Periods: 1969-1971; 1979-1981; 1989-1991; 2003.**




* Age at black-white mortality crossover.

Data Source: U.S. Decennial Life Tables, National Center for Health Statistics (NCHS)
http://www.cdc.gov/nchs/products/life_tables.htm

**Figure 2.2: Logged Single-year Age-specific Adult Mortality Risks and Logged Five-year Age-specific Mortality Rates of U.S. Black and White Men and Women, NHIS-LMF 1986-2006.**

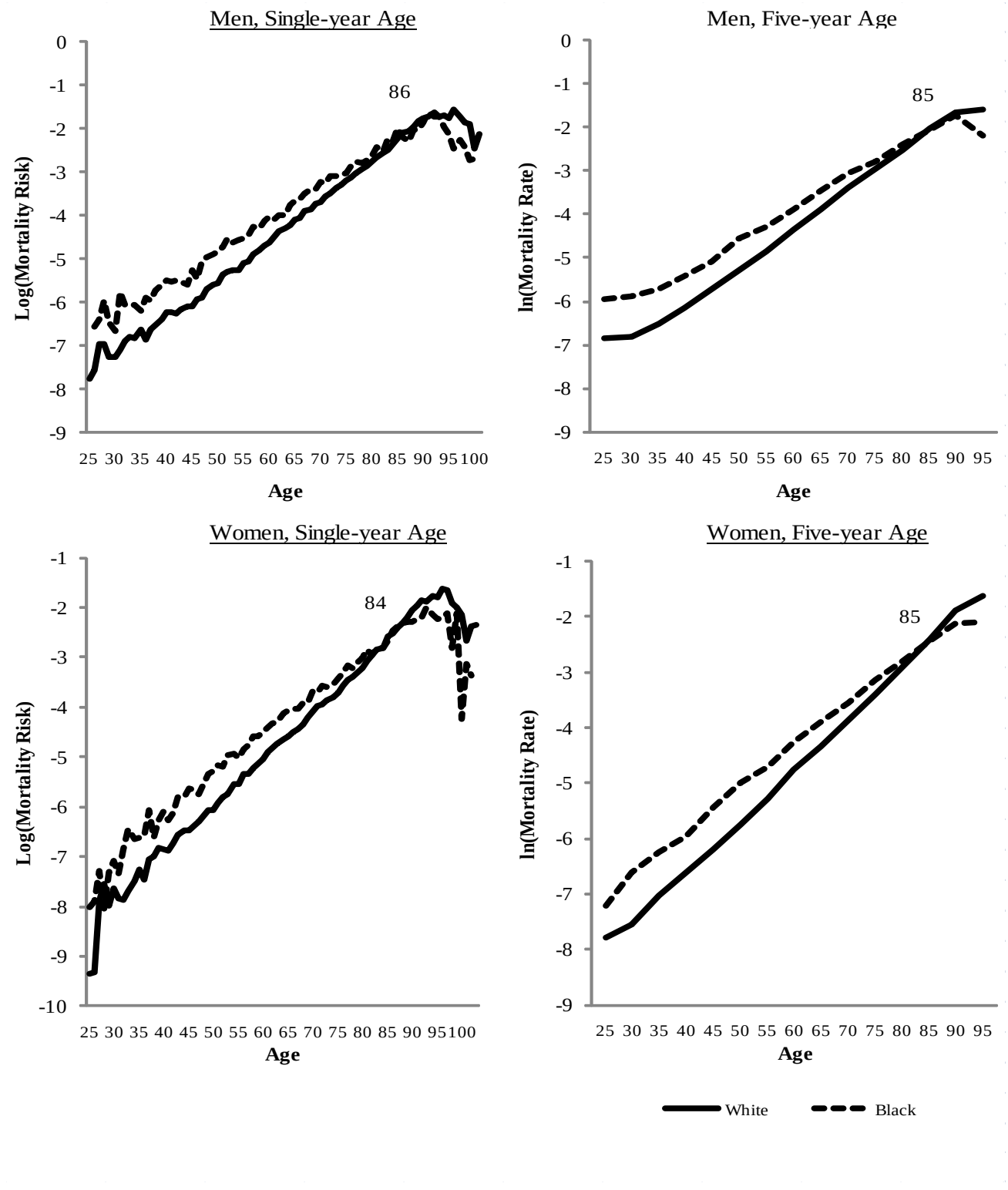


**Figure 2.3: Logged Single-year Age-specific Mortality Risks of U.S. Black & White Men & Women, NHIS-LMF 1986-2006: Self-Reports, Reported Education, & Calculated Ages 25-75 at Survey.**

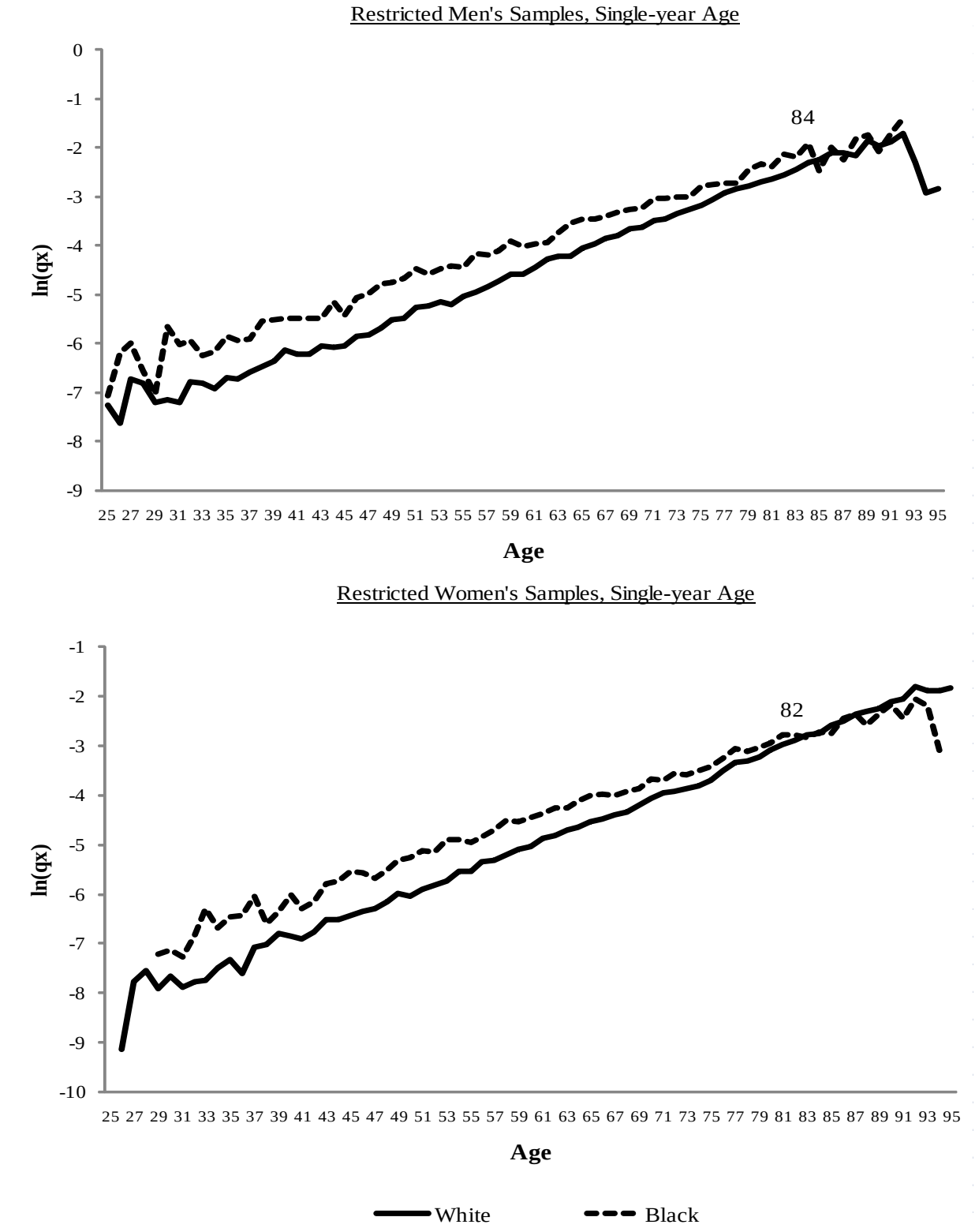


**Figure 2.4: HAPC-CCREM Estimates of Random Cohort Effects of U.S. Black & White Men's & Women's Mortality Rates, NHIS-LMF 1986-2006.**

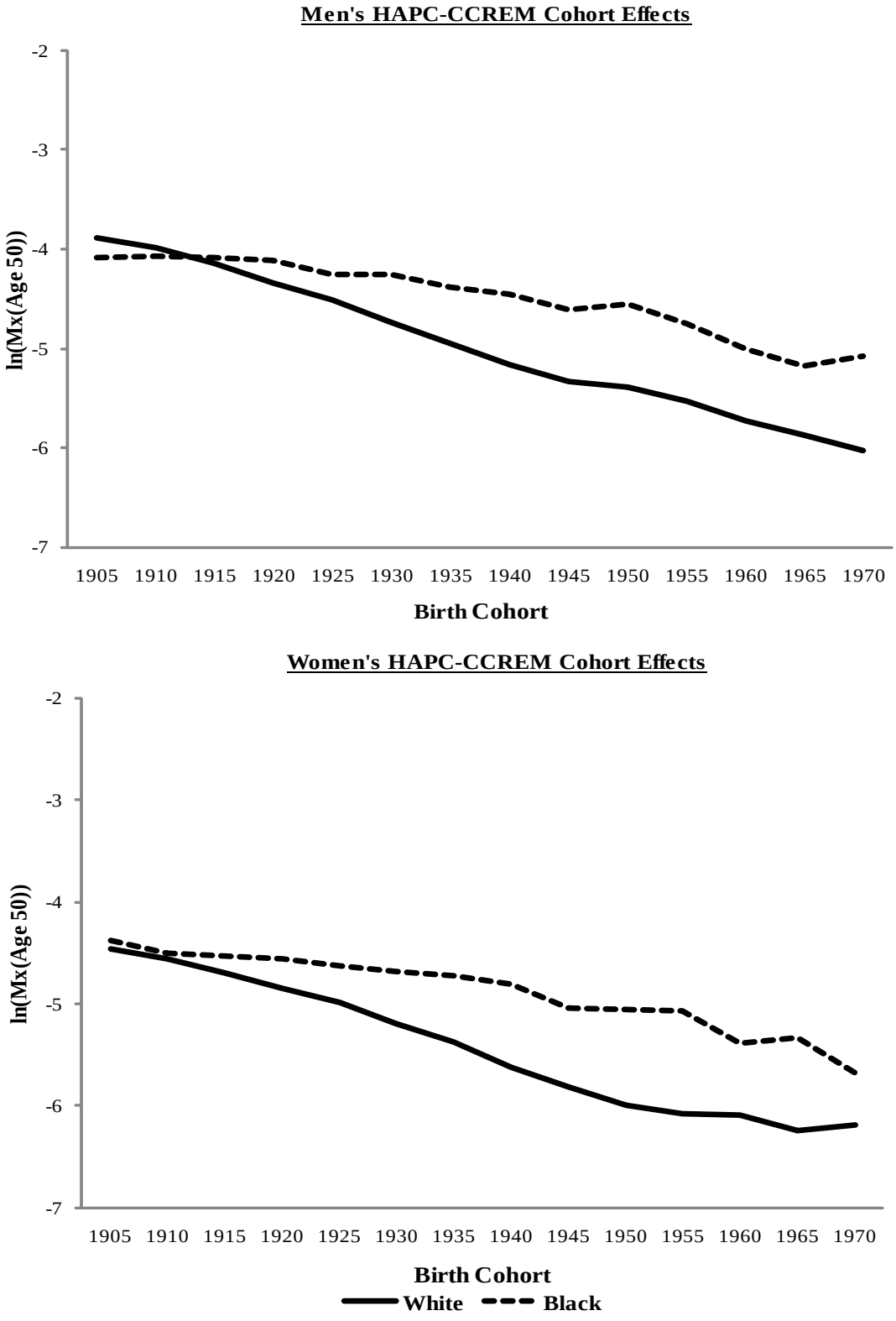


**Figure 2.5: HAPC-CCREM Estimates of Fixed Age Effects of U.S. Black & White Men's and Women's Mortality Rates, NHIS-LMF 1986-2006.**

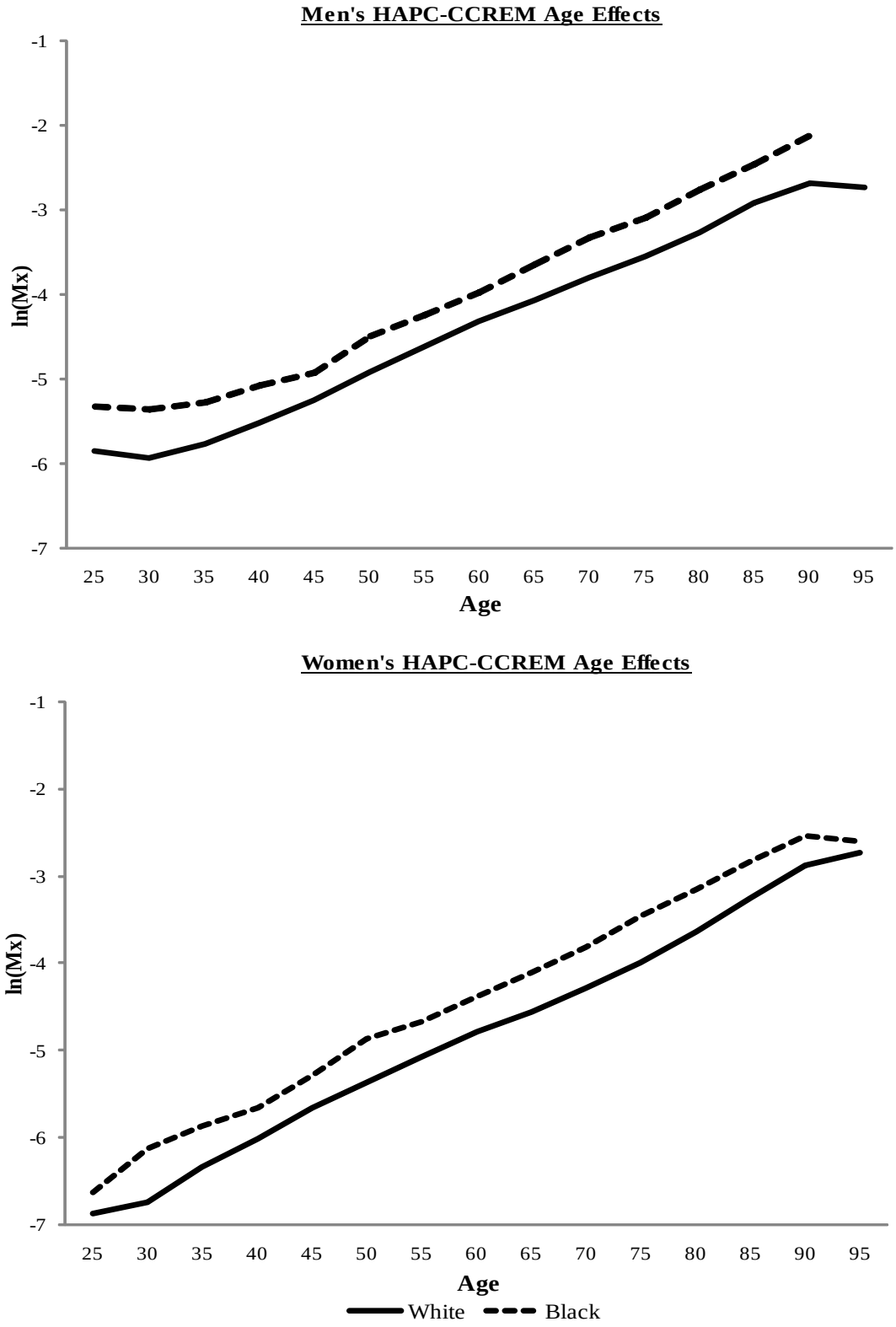
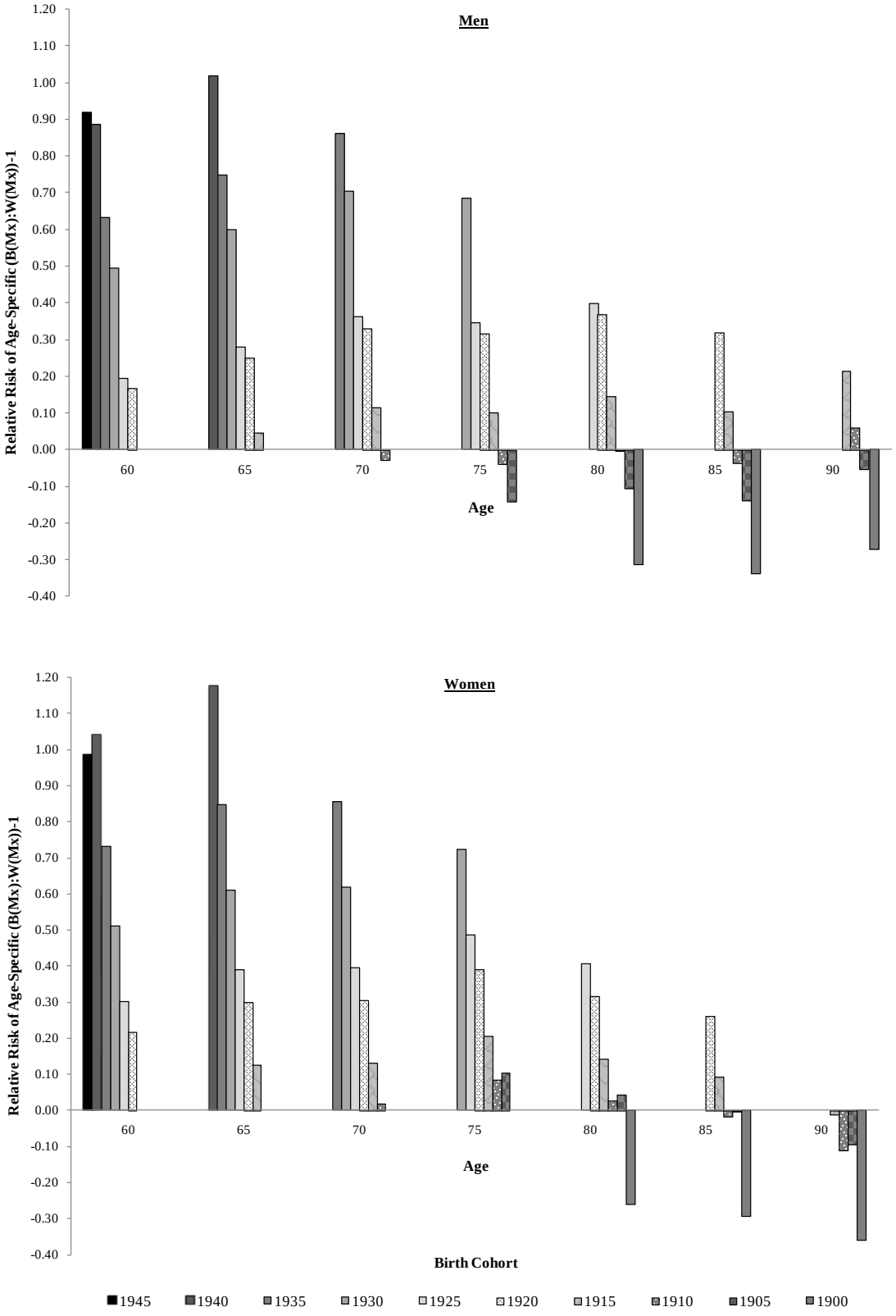


**Figure 2.6: Ratios of U.S. Black and White Men's and Women's Older-age Mortality Rates in the NHIS-LMF 1986-2006, by Birth Cohort.**



# CHAPTER 3

## Educational Differences in U.S. Adult Mortality: A Cohort Perspective

### 3.1 Introduction

Early in the twentieth century, tremendous achievements were made to improve health and reduce early death in the United States. Across the latter half of the century, steady gains in overall life expectancy were made by reducing mortality risk of degenerative diseases for middle and older adult age groups (Crimmins 1981; Yang 2008; Cutler et al. 2010). Research has largely attributed more recent mortality declines to temporal changes in a handful of specific causes of death, chief among these being heart disease, lung cancer, and other cancers (Jemal et al. 2005; Guyer et al. 2000; Cooper et al. 2000). While these achievements are to be celebrated, understanding the factors behind the increases in life expectancy remains limited. One problem is that many studies of U.S. mortality have employed a period framework to understand and analyze trends. In these cases, age-specific mortality risk at one time period is compared to age-specific mortality risk in a past time period, with little attention given to the cohort composition of the populations at those respective times. As a result of this approach, improvements in health outcomes and survival may be misattributed to the health inputs *during* that period (Healthy People 2010; Meara, Richards and Cutler 2008).

In this chapter I address this shortcoming by analyzing age, period, and cohort changes in the ways that educational attainment affected U.S. adult mortality risk between 1986 and 2006. To do so, I first bring together three perspectives of mortality risk: Link and Phelan's "fundamental cause" theory (1995), the life course perspective (Ben-Shlomo and Kuh 2002;

Montez and Hayward 2011), and Riley's "principle of cohort differences in aging" (1987). I frame recent changes in educational disparities of U.S. adult mortality using these perspectives, and I develop hypotheses regarding age, period, and cohort based differences in the education-mortality relationship. I further hypothesize that educational disparities in mortality, and cohort trends in these disparities, are stronger for causes of death that are under greater degree of human control, such as heart disease and lung cancer. I further hypothesize that cohort changes in the education-mortality relationship are stronger among U.S. non-Hispanic whites (henceforth "whites") than among U.S. non-Hispanic blacks (hence force "blacks"). I then employ new data and methods to simultaneously examine how age, period, and cohort (APC) effects on recent U.S. adult mortality risk differ by educational attainment. I do this separately for black and white women and men, as well as for major underlying causes of death. My findings overwhelmingly support the contention that cohort differences are responsible for growing educational disparities in adult mortality risk, and that cohort changes in the education-mortality relationship are more pronounced for whites than for blacks. I conclude by advocating for the use of a cohort perspective of mortality change over the more commonly used period perspective (Fogel 2004, 2005; Finch and Crimmins 2004; Ryder 1965; Riley 1987).

## 3.2 Theoretical Background and Hypotheses

### 3.2.1 *Educational Attainment and U.S. Adult Mortality Risk*

The association between educational attainment and mortality is widely studied in the social and health sciences. In the United States, the education-mortality relationship was first comprehensively documented with national-level data by Kitagawa and Hauser (1973). Their findings demonstrated that educational differences in mortality were substantial, were much

wider at younger than at older ages, and were somewhat larger among women than men. Since then, many studies have documented the education-mortality association and attempted to explain the ways by which education affects mortality risk (see Hummer and Lariscy [2011] for a recent review). In general, it has been repeatedly shown that individuals with relatively low levels of education have a significantly higher annual risk of mortality than those with more education. While it is widely acknowledged that a sizable portion of the education-mortality association is mediated by economic resources, social-psychological resources and health behaviors are also important mediators of this relationship (Denney et al. 2010; Lynch 2006; Mirowsky and Ross 2003; Ross and Wu 1995). Education has also been argued to directly affect mortality risk via the knowledge and use of available health technologies (Glied and Lleras-Muney 2008; Phelan, Link and Therhanifar 2010). Despite continued declines in both all-cause and cause-specific death rates, as well as aggregate increases in educational attainment, education disparities in U.S. mortality risk have widened across time (Lauderdale 2001; Pappas, et al. 1993; Meara et al. 2008; Montez et al. 2011). Widening educational disparities in mortality indicates fundamental socioeconomic stratification of a treasured resource – life itself – that must be better understood if efforts to close such a disparity are to succeed.

The strength of the relationship between education and mortality implies that education is a "fundamental social cause" of health and mortality risk (Link and Phelan 1995, 1996, 2002; Link 2008; Phelan et al. 2010). This is because education shapes individual-level "resources like knowledge, money, power, prestige, and social connections that strongly influence people's ability to avoid risks and to minimize the consequences of disease once it occurs" (Link and Phelan 1996: 472). Additionally, education influences the broad contexts in which individuals live and work, as well as the social networks individuals belongs to. These social contexts can

also shape risk factors (e.g., poor housing, second-hand smoke) and/or the knowledge and lifestyles individuals are exposed to (Phelan et al. 2010). A central tenet of fundamental cause theory is the staying power of education. That is, despite changes in our understanding of disease, health behaviors, and treatment of disease and disability, the association between education and mortality persists. This is because the well-educated continue to be the most likely to have access to, and to take advantage of, new knowledge, practices, and/or technologies that are related to morbidity and mortality risk (Link and Phelan 1995; Link 2008; Phelan et al. 2004; Glied and Lleras-Muney 2008). This implies that educational differences in mortality risk might grow wider during periods of rapid development of health technologies, especially for causes of death associated with such technologies (Chang and Lauderdale 2009). In general, though, fundamental cause theory is largely silent with regard to the patterns by which the education-mortality association differs by age and may change across birth cohorts. This chapter adds to this literature by detailing education's effects on mortality risk during a period of rapid development of health technologies (Glied and Lleras-Muney 2008; Chang and Lauderdale 2009; Lucas et al. 2006), rising socioeconomic inequality (Campbell et al. 2005; Pettit and Ewert 2009), and decreasing mortality risk (Xu et al. 2010).

### 3.2.2 *Educational Attainment and Mortality Risk: Age Differences*

Before turning to period and cohort differences in the education-mortality association, I briefly consider age differences in this relationship. There is continued debate concerning the manner by which education's effect on mortality changes with age. Some research has found that educational differences in mortality risk converge at the oldest adult ages, which supports an age-as-leveler perspective (Beckett 2000). Others, however, argue that the effects of education

on health and mortality increase with age. Lynch (2003), for example, found that the effect of education on self-rated health strengthens with age, and that this pattern has intensified across cohorts (see also Mirowsky and Ross 2008; Lauderdale 2001). Such findings support a cumulative advantage perspective.

Most existing literature has omitted cohort effects in age-related analyses of education and mortality. This is problematic because no single "life course" exists; rather, each cohort experiences a distinct life course, shaped by the confluence of age effects and each cohort's unique experience of history (Riley 1978, 1987; Riley and Riley, Jr. 1986; Ryder 1965). Indeed, Riley (1987), in advancing her "principle of cohort differences in aging," persuasively argued that people in different cohorts age in unique ways due to the disparate sociohistorical conditions in which their life courses unfold. It follows, then, that the way by which age conditions the education-mortality association may be changing across cohorts and, thus, analyses that omit cohort effects in the examination of the education-mortality relationship are biased (Ben-Shlomo and Kuh 2002). While not the main focus of this chapter, I hypothesize that:

*H1: educational differences in U.S. adult mortality are characteristic of all age groups once period and cohort effects are accounted for.*

### 3.2.3 *Educational Attainment and Period-Based Trends in Adult Mortality Risk*

The literature on U.S. mortality trends has generally concluded that the effect of socioeconomic status, education included, was quite strong at the beginning of the twentieth century, but that the relationship weakened by the middle of the century as the population underwent the epidemiologic transition (Warren and Hernandez 2007; Lynch 2003; Manton et al. 1997). This is evidenced by the fact that the leading emerging degenerative diseases in the mid-

twentieth century United States – heart disease, cancers, and other undiagnosed and/or at the time untreatable diseases – affected all socioeconomic subpopulations similarly. In fact, a positive association between education and coronary heart disease existed for U.S. males during the 1940s and 1950s, due in large part to high levels of smoking and meat consumption among high status men (Manton et al. 1997).

Only after greater knowledge of risk factors was gained, and the development of medical technologies to prevent and treat degenerative diseases progressed, did researchers begin to note a protective educational effect on U.S. mortality risk due to heart disease, stroke, and some cancers. Recognizing this, a host of studies in the late 1980s and early 1990s compared socioeconomic mortality differentials in the mid-1980s back to those of Kitagawa and Hauser (1973), who utilized data from 1960. The later analyses showed that educational differences in adult mortality widened between 1960 and the mid-1980s (Feldman et al. 1989; Pappas et al. 1993), particularly among men (Preston and Elo 1995).

More recent evidence suggests that the education-mortality relationship continued to widen during the 1990s and into the early 2000s. Cutler et al. (2010), for example, found that the education gap in mortality risk widened modestly for men but especially so for women during the 1990s (see also Jemal et al. 2008 and Meara et al. 2008). Most recently Montez et al. (2011) showed that the educational gap in adult mortality widened among U.S. adults aged 45-84 between 1986 and 2006, with both women and men at the highest educational levels exhibiting sharp mortality declines over this period, while women at the lowest educational level actually exhibited a mortality risk increase. In all, these papers document a continuation of the trend of widening educational differences in U.S. adult mortality that began at least as early as the mid

twentieth century, despite major policy initiatives designed to the contrary (e.g., Healthy People 2000 and Healthy People 2010). Thus, in general, I expect to find that:

*H2: the educational gap in U.S. adult mortality risk widened between 1986 and 2006.*

### 3.2.4 *Educational Attainment and Cohort-Based Trends in Adult Mortality Risk*

A major limitation of existing literature on U.S. mortality trends is its overwhelming adherence to a period perspective. The practice is puzzling because cohort perspectives have been central to sociological theories of social change for some time (Ryder 1965). Indeed, Smith (2008: 289) states that sociologists are "mad for cohorts" and the role cohort replacement plays in driving social change. Yet, despite this tradition, a cohort perspective is largely absent from recent sociological and epidemiological analyses of U.S. mortality disparities. In most studies of mortality trends, as well as official reports of U.S. death statistics (Xu et al. 2010), population-level mortality is perceived to be, measured as, and presented as a period phenomenon.

There are very important reasons, though, to consider a cohort perspective of mortality change in addition to the more commonly measured period perspective. On empirical grounds, Yang (2008) used newly developed age-period-cohort (APC) methods to analyze U.S. vital statistics data from 1960 to 1999, and showed that temporal reductions in U.S. adult mortality rates across this time were almost exclusively attributable to cohort, not period, effects. On theoretical grounds, I point to the growing literature on life course effects on health and mortality risk. While the idea that early-life conditions influence subsequent health and later-life mortality risk is not new, life course studies of mortality risk have surged in recent years. Considered to be a "long arm of childhood" (Hayward and Gorman 2004), harsh conditions in early life have been shown to have both direct and indirect effects on adult self-rated health (Kestila et al. 2006),

morbidity (Blackwell et al. 2001; O'Rand and Hamil-Luker 2007; Freedman et al. 2008), and mortality risk (Hayward and Gorman 2004). In one set of studies, socioeconomic conditions during childhood (e.g., family size and parental education) are shown to influence susceptibility to childhood disease (Case and Paxson 2010) and/or significantly affect trajectories of socioeconomic attainment, which, in turn affects adult mortality risk (Montez and Hayward 2011). In another set of studies, a poor uterine environment (Barker 2007), childhood malnourishment (Fogel 2005), and/or early bouts with infections and inflammation are shown to increase physical susceptibility to later life disease and mortality by scarring vital organs (Finch and Crimmins 2004; Crimmins and Finch 2006), stunting growth (Case and Paxson 2010; Fogel 2004), and/or contributing to an immune risk phenotype (Simanek et al. 2008). While researchers continue to investigate the pathways by which childhood conditions affect later-life mortality, evidence overwhelmingly suggests an association (see Montez and Hayward [2011] for a thorough review).

Yet, the extent to which the association between early-life conditions and later-life mortality has changed across U.S. birth cohorts is unknown. Consistent with Riley's (1987) "principle of cohort differences in aging," researchers are recognizing the need to embed the life course in the sociohistorical contexts in which it unfolds (Ben-Shlomo and Kuh 2002; Montez and Hayward 2011). This suggests that the life course is, in fact, a cohort-specific phenomenon and can vary over time. Indeed, Finch and Crimmins (2004) propose that enduring effects of cohorts' disparate exposures to early-life conditions produce distinct "cohort morbidity phenotypes." If cohorts vary in their exposures to early-life conditions then we should also expect cohort-specific variation in susceptibilities to later-life mortality risk. This perspective has

profound implications for understanding and analyzing how the education-mortality association has changed over time in the United States. I illustrate this via the following four points.

First, early-life conditions have clearly improved across U.S. birth cohorts. Early in the twentieth century major efforts were undertaken to reduce infectious diseases (Centers for Disease Control 1999), improve nutrition (Fogel 2004; Manton et al. 1997), and expand public health services (Cutler and Miller 2005; Easterlin 1997). Mortality rates fell faster across the first third of the twentieth century than at any other time in U.S. history, and both maternal and childhood health dramatically improved (Cutler and Miller 2005; Fogel 2004; Case and Paxson 2010; Warren and Hernandez 2007). Living conditions changed in substantial ways too, as urbanization, compulsory schooling (Glied and Lleras-Muney 2008), reductions in fertility, and expansion of consumer goods reshaped families, increased household safety, and improved general standards of living (Easterlin 1997; Fogel 2004). In Figure 3.1 I present cohort-level changes in the percent of U.S. infants born into large families (defined as having six or more members), the percent of infants born into farming households, the percent of infants born in southern states, and sex- and race/ethnic-specific infant mortality rates. While limited, these measures serve as proxies of the drastic improvements in living and health conditions that unfolded across U.S. birth cohorts during the twentieth century.[2] I present these cohort changes by race to illustrate the significant role race has played in shaping cohort changes in U.S. early-

[2] In APC analyses of U.S. adult mortality rates between 1986 and 2006 (not shown), these measures of early-life conditions, as well as cohorts' smoking patterns, were found to account for nearly all variance in cohort-level mortality. In a reduced model, smoking alone accounted for large amounts of cohort variance in mortality, but only for cohorts born after the 1930s. Cohorts born between 1900 and 1935 were not substantively affected by cohorts' smoking prevalence. Also, individual-level educational attainment accounted for only a small reduction in cohort variance in adult mortality between 1986 and 2006. These findings illustrate two key points. One, while indicators of the adult environment (i.e., education and smoking) explain some variance in cohort mortality risk, substantial cohort variance was not explained. Two, the variance explained by mechanisms associated with the adult environment was largely confined to cohorts born in the latter half of the twentieth century. Thus, the effects of both early-life conditions and adult conditions on U.S. adult mortality appear to be associated with cohorts.

life conditions. In every measure, black (or non-white) cohorts endured both higher levels of disadvantage and slower rates of improvement across time. These racial differences affect how we must think about the role of cohort changes to the education-mortality relationship.

As both the prevalence and severity of harsh early-life conditions were reduced across cohorts, and as greater proportions of cohorts survived into adulthood, the relative degree to which early-life conditions shaped cohorts' later-life mortality has lessened. Thus, my second point is that, in absolute terms, the association between early-life conditions and later-life mortality has diminished across cohorts. This is not to argue that the individual-level *strength* of the association between-early life conditions and later-life mortality risk has waned. Rather, I am making the simple point that childhood conditions at the cohort level are necessarily accounting for less variance in cohorts' overall adult mortality risk. While stemming from cohort improvements in early-life conditions, this also partly reflects disparate rates of cohort survival into adulthood. Indeed, relative to the experience of the 1900 U.S. female birth cohort, the 1960 female birth cohort enjoyed 26.0 percent greater survival to age 25 (Human Mortality Database 2010). Thus, as childhood mortality accounted for a diminishing portion of overall mortality, and as early-life conditions improved across cohorts, the relative degree to which early-life conditions affected later-life mortality changed across cohorts.

Third, as a result of reduced impacts of harsh infant and childhood conditions, mortality in adulthood comprises a greater proportion of overall U.S. mortality, and the adult environment grows relatively more important in shaping later-life mortality risk. Thus, on the one hand, personal behaviors such as exercise, diet, alcohol use, and cigarette smoking grew more important in shaping adult mortality risk (Rogers and Hackenberg 1987). On the other hand, knowledge of, access to, and use of preventative and curative health technologies such as

physical examinations, statins, beta-blockers, chemotherapy, and corrective surgeries grew increasingly important as well (Cutler et al. 2010).

Fourth, because the adult environment is growing relatively more important in shaping cohorts' adult mortality risk, the role of personal resources used to navigate this environment, collect and understand health-related information, procure helpful technologies, and take advantage of new health-related ideas should be growing increasingly important as well. Consistent with fundamental cause theory, I argue that education is becoming an ever greater resource that is used to "socially shape" access to new health knowledge and/or beneficial technologies as well as contexts that promote healthy behavior (Link 2008). And because the process of educational attainment and access to resources unfolds in a life course fashion and differentially so across cohorts, education is becoming more strongly associated with U.S. adult mortality risk across cohorts. Thus, based on both empirical and theoretical grounds, I hypothesize that:

*H3: the educational gap in U.S. adult mortality is widening across birth cohorts rather than across time periods.*

### 3.2.5 *Education and U.S. Adult Mortality Trends: Differences by Gender, Race, and Cause of Death*

Because U.S. women and men experience significantly different adult mortality risk, I stratify all of my analyses by sex. My hypotheses pertaining to age, period, and cohort differences in mortality equally apply to both men and women, but gender differences in cohort patterns of educational attainment, lifestyle risk factors, and gender-stratified economic opportunities, among other factors, should be kept in mind. For instance, cohort differences in

men's and women's smoking patterns will likely influence disparate patterns of heart disease and lung cancer mortality risk (Preston and Wang 2006; Pampel 2003).

U.S. health and mortality patterns also differ between blacks and whites in substantial and persistent ways (Williams and Sternthal 2010; Hummer and Chinn 2011). While differences in life expectancy between the U.S. white and black populations have modestly narrowed in recent years (Harper et al. 2007), black-white gaps in chronic disease (Hayward et al. 2000) and mortality risk (Hummer and Chinn 2011) remain distressingly large. Evidence suggests that the racial gaps in adult mortality reflect cumulative life course processes of health and socioeconomic disadvantages suffered by the black population (Hayward et al. 2000; Shuey and Willson 2008; Williams and Jackson 2005; Williams et al. 2010). Racial stratification of resources across the life course and across cohorts, combined with the persisting racial gap in U.S. health and mortality risk, suggests that cohort changes to the education-mortality relationship probably differs by race. On the one hand, black cohorts have, on average, endured higher prevalence and greater severity of early-life disadvantages than white cohorts (Figure 3.1) (Hayward et al. 2000). This implies that the extent to which adult mortality risk is influenced by "the long arm of childhood" is greater among blacks than among whites (Hayward and Gorman 2004). As such, the ability for black Americans to capitalize on education to improve adult health and reduce mortality risk may be hindered by persistent deleterious childhood effects. On the other hand, irrespective of education level, racial discrimination in employment, earnings, health care, housing, and other aspects of social life make the adult environment harsher for blacks (Williams and Jackson 2005; Tehranifar et al. 2009). As such, while I hypothesize that education is a resource growing increasingly important in shaping U.S. cohort mortality experiences, I further hypothesize that race conditions this effect. Thus:

*H4: cohort changes to educational disparities in adult all-cause mortality are stronger in the white population than in the black population.*

Lastly, cohort changes to the education-mortality relationship should be strong for causes of death that are significantly associated with risk factors such as smoking (Preston and Wang 2006; Link 2008) or diseases that are somewhat preventable or treatable with medical knowledge and technologies (Glied and Lleras-Mundes 2008; Phelan et al. 2010; Chang and Lauderdale 2009). This is because educational attainment, as argued above, has become an increasingly important resource in shaping access to healthy lifestyles and health information and care. Consistent with this, I should see large and growing educational disparities across cohorts for causes of death such as heart disease and lung cancer, while on the other hand, I expect significantly smaller educational gradients in mortality from cancers that are less preventable and/or are difficult to treat. I therefore further test fundamental cause theory by analyzing age, period, and cohort patterns of adult mortality risk for heart disease, lung cancer, and "unpreventable" cancers between 1986 and 2006 (Phelan et al. 2004).[3] Due to significantly smaller counts of cause-specific deaths among blacks, this cause-specific hypothesis is tested using only the white female and male subsamples.

*H5: educational differences in heart disease and lung cancer mortality are larger and widening across cohorts more so than educational differences in mortality from "unpreventable" cancers.*

### 3.3 Data

[3] A death was classified as an "unpreventable" cancer by using the NCHS's 113 Selected Causes of Death and Phelan et al.'s (2004) Appendix A. Deaths due to any cancer with a "preventability rating" lower than 4.0 were classified as "unpreventable cancers" and coded as 1, and all other deaths or censored cases were coded as 0.

I use data from nineteen National Health Interview Surveys (NHIS), 1986 through 2004, linked to follow-up mortality information for each cross-section through December 31st, 2006 (NCHS 2010). This linked data set was concatenated and made publicly available by the National Center for Health Statistics (NCHS) and the Integrated Health Interview Series (IHIS) project at the Minnesota Population Center (IHIS 2011). The NHIS uses a multistage probabilistic sampling design, and respondents of the NHIS are matched to the computerized mortality records of the National Death Index using a 14-item identification scheme (NCHS 2010). Respondents of the NHIS not eligible for matches to death records are dropped from the final sample; I use NCHS derived analytical weights to make results from this data set representative of the noninstitutionalized white and black adult population. The resulting 1986-2006 National Health Interview Survey-Linked Mortality Files (NHIS-LMF) are a unique combination of repeated cross-sectional surveys coupled with longitudinal annual records of individual respondents' survival status. These data have several advantages for studying trends in educational differences in mortality risk across cohorts and time. First, collapsing the repeated cross-sections of the NHIS with the individual-level longitudinal mortality histories breaks the linear dependency of age, period, and cohort effects in our analyses of mortality. Second, because links between the NHIS surveys and mortality follow-up range from 1986 to 2006, there is sufficient overlap between age, period, and cohort to estimate stable and reliable effects of all three variables. Further, unlike U.S. vital statistics data that rely on death certificate reports, NHIS respondents self-report their age, race, and educational attainment.

In order to ensure enough time for individuals to complete all measured levels of educational attainment, to focus on ages where mortality risk is high and death counts were most plentiful, and to limit the use of data where age is top coded, I restricted the NHIS-LMF to U.S.-

born black and white respondents aged 25 to 84 at time of survey who were 25-99 years of age during the follow-up period.[4] Limiting the data in this way trimmed my starting analytic sample sizes to 368,356 white males; 407,371 white females; 54,236 black males; and 78,280 black females. After annual exposure times to death were calculated, the resulting person-period datasets consisted of 4,505,955 white male person-years; 5,103,764 white female person-years; 637,699 black male person-years; and 962,276 black female person-years.

These person-year samples were then collapsed into aggregated subsamples of age-period-cohort blocks.[5] The coding of birth cohort results in 15 five-year blocks ranging from 1900-1904 to 1970-1974. The coding for period is made up of five blocks ranging from 1986-1990 to 2003-2006; the earliest period block spans five years because it contained the fewest number of deaths, while the remaining four periods spanned four years each. And the coding of age is in fifteen 5-year blocks ranging from 25-29 up to 95-99. Combining the blocks together, the sex- and race-specific samples for all-cause mortality analyses were each composed of 168 unique APC blocks. After limiting the cause-specific analyses to person-periods aged 40 and above and cohorts up through 1960-64 (due to the limited number of cause-specific deaths among younger adults born in recent cohorts), the sex-specific data for the cause of death

[4] Individuals aged beyond 99 years were censored from the sample because cell sizes and counts of death for the five-year age range 100-104 are very small.

[5] I attempted to analyze educational differences in U.S. adult mortality using HAPC discrete-time survival models. These models were estimated using the individual-level person-period data with a binomial family (1/0) and a complimentary-log-log link function, but convergence was achieved only for analyses of all-education/all-cause mortality of white men and women. To estimate the effects of education, and to analyze these effects by race and cause of death, I had to aggregate the data.

analyses each contained 132 unique APC blocks.[6] Table 3.1 displays descriptive statistics for the individual-level data, the person-period data, and the sex- and race-specific APC blocks.

I use three categories of educational attainment: less than high school (<HS), high school or equivalent (HS), and greater than high school (>HS). These categories each contain large numbers of individuals and a sufficient number of deaths across the age range of the study. Moreover, these categories have been shown to capture much of the differentiation in U.S. mortality risk by educational attainment (Montez et al. 2011). The educational composition of the U.S. population changed substantially across the twentieth century, with cohorts born early in the century being disproportionately composed of persons with a less than high school education. Conversely, cohorts born in the middle of the century experienced improved educational achievement, with the majority of cohorts born mid-century attaining a high school degree or higher. The aggregated change in educational attainment is thought to be a primary factor in the temporal decline of U.S. adult mortality (Yang 2008; Lynch 2003). Here, I stratify my analyses by educational attainment to allow for education-specific estimates of APC patterns of mortality. Educational differences in mortality were tested by estimating and contrasting education-specific confidence intervals of age, period, and cohort effects on mortality.

### 3.4 Analytic Methods

I use hierarchical age-period-cohort (HAPC) models for repeated cross-sectional survey data for analyses of all-cause white men's and women's adult mortality rates. For cause-specific

[6] Aggregated data did not always amount to 168 cells for all-cause mortality or to 132 cells for cause-specific mortality. The sample of black men with a HS education amounted to only 160 APC cells, and the sample of black men with a >HS education amounted to only 164 APC cells. Black women with a >HS education contained only 167 APC cells. Also, I limited the models of lung cancer mortality to ages 25-29 to 90-94 due to small cell sizes at the oldest age group. As a result of this, the white men's and women's samples used to analyze lung cancer mortality, irrespective of educational attainment, all contained 126 APC cells.

analyses and for black men's and women's mortality, Hierarchical Bayesian Models are estimated under a Gibbs Sampling approach (Yang and Land 2006; Gelman 2006; Lynch 2007). These methods utilize a cross-classified random effects model (CCREM) to embed each person-year observation within a shared time period and birth cohort at their given five-year age group. Goodness-of-fit statistics (see Table 3.2) from fixed effects models of APC analyses generally verify that all three effects should be included in the models for all race-sex subsamples. However, the more conservative BIC criterion, which penalizes models for numbers of parameters and sample size, indicates that period effects do not substantively improve model fit for black men and women.

Because the NHIS-LMF 1986-2006 data follows individual survival status as each respondent ages across periods, each respondent can occupy several APC combinations. Also, each five-year age block comprises multiple combinations of time periods and birth cohorts. Thus, while collinearity between the age, period, and cohort effects is high, these data do not suffer the identification problem induced by an absolute linear dependency between the three effects (Mason et al. 1973; Glenn 2005). The HAPC-CCREM modeling is an appropriate APC methodological tool, and has been shown to be more efficient than a fixed effects approach when data, such as the NHIS-LMF, are unbalanced (Yang and Land 2008). The HAPC-CCREM estimates fixed effects of the five-year age groups and random effects of the four-year period and five-year cohort groups, and is structured in the following way:

Level-1 within cell model:

$$\log\left[\mathrm{E}(D_{ijk})\right] = \alpha_{jk} + \beta_{jk} A_i + \log(R_{ijk}), \qquad (1)$$

where $D_{ijk}$ stands for the counts of deaths of the $i$th age group (for $i$ = 1, …, $n_{jk}$ age groups) within the $j$th period (for $j$ = 1, …, $J$ time periods) and the $k$th cohort (for $k$ = 1, …, $K$ birth

cohorts); $A_i$ denotes a dummy variable corresponding to each of the five-year age groups 1,…, $n_{jk}$; $\alpha_{jk}$ is the intercept indicating the reference age group (65-69) who was in period $j$ and belong to cohort $k$; and $\log(R_{ijk})$ is the natural log of the aggregated exposure time lived during each age-period-cohort cell.

Level-2 between cell random intercept model:

$$\alpha_{jk} = \pi_0 + t_{0j} + c_{0k}, \qquad (2)$$

in which $\alpha_{jk}$ specifies that the age effects vary from period to period and from cohort to cohort; $\pi_0$ is the expected mean at the reference age (65-69) averaged over all periods and cohorts; $t_{0j}$ is the overall four-year period effect averaged over all five-year birth cohorts with variance $\sigma^2_{t0}$; and $c_{0k}$ is the overall five-year cohort effect averaged over all four-year periods with variance $\sigma^2_{k0}$.

I combine the level-1 and level-2 models to estimate the expected log counts of deaths in each APC cell assuming that deaths follow a Poisson distribution. The aggregated exposure time lived within the cells is used as an offset to the model in order to generate results in the form of APC-specific log mortality rates. Data for black men and women, and subsamples of specific causes of death, contained small counts of death in some APC cells and required more robust modeling techniques, in which case I estimated Hierarchical Bayesian Models using a Gibbs sampling approach for all analyses of cause-specific mortality and for analyses of black men's and women's all-cause mortality (Gelman et al. 2006; Lynch 2007). I assumed noninformative prior distributions for all model parameters (Gelman 2006; Lynch 2003). Models were robust to alternate prior distributions.[7]

[7] To facilitate estimation, hyperpriors on variances of black men's and women's random period and cohort effects were set to narrower limits than (.001, .001).

## 3.5 Results

Figure 3.2 presents graphed estimates of fixed effects age coefficients and random effects period and cohort coefficients from analyses of all-education/all-cause mortality rates of U.S. adult white and black men and women between 1986 and 2006. Figure 3.3 presents graphed estimates of APC patterns of white men's and women's mortality rates from heart disease, lung cancer, and "unpreventable"cancers. I first present these estimates in Figures 3.2 and 3.3 to introduce the general APC patterns of U.S. adult mortality risk between 1986 and 2006. I then proceed to test our five hypotheses focusing on education-mortality trends.

### 3.5.1 *Trends in All-education/All-cause and All-education/Cause-specific Mortality*

The pattern of results from our HAPC-CCREM analyses of all-cause adult mortality rates between 1986 and 2006 for white and black men and women are consistent with findings from Yang (2008), who used vital statistics data for 1960-1999. As presented in Figure 3.2 and Figure 3.3, age effects on mortality follow the usual log-linear pattern, with slight tapering at the oldest-old age groups (85+). This is the case for all sex-race subsamples for all-cause mortality and also for most causes of death, with the exception of lung-cancer mortality. The distinct age pattern of lung-cancer mortality risk has age effects rising much more steeply than other specific causes of death, but these taper off around age 65 and plateau thereafter.

Also consistent with Yang (2008), our HAPC-CCREM estimates of all-cause and cause-specific mortality suggest that temporal changes in U.S. mortality risk across 1986 and 2006 were largely driven by cohort reductions in mortality rates. Little variation is found in estimated period effects of mortality, irrespective of race, sex, or cause of death, though I do find a slight increase in white men's and women's all-cause mortality across periods. Conversely, estimates

of cohort effects illustrate significant and persistent cohort declines in men's and women's all-cause mortality rates, and in mortality rates from heart disease. Consistent with previous findings, cohort patterns of lung cancer mortality closely follow cohort patterns of adult smoking (Wang and Preston 2009). Lastly, no cohort variation is visible for unpreventable cancers. These descriptive results support the idea that cohort processes were driving temporal reductions in U.S. mortality risk between 1986 and 2006. At the same time, note that cohort reductions in black men's and women's mortality between 1986 and 2006 were less steep than cohort reductions in white men's and women's mortality. I now turn to our examination of how these age, period, and cohort patterns differed by educational attainment.

### 3.5.2 *Educational Differences in Trends in U.S. Men's and Women's All-cause Mortality Risk*

Tables 3.1-3.4 present estimates of fixed effects age coefficients and random effects period and cohort coefficients from education-stratified HAPC-CCREM analyses of 1986-2006 all-cause adult mortality for black and white men, and black and white women, respectively. Taken together, the education-stratified models reveal tremendous educational variation in the size of both age and cohort effects, but very little variation across period effects. Our subsequent discussion of these models relies on the graphed results in Figures 3.4 through 3.6.

To test my first hypothesis, I present estimated age effects of men's and women's all-cause mortality rates, stratified by educational attainment, in Figure 3.4. For white men and women, the educational gap in age-specific mortality rates is preserved across all ages. That is, the difference in mortality between the <HS education group and the >HS education group is significant at all age-groups except 95-99, at which point large standard errors make the differences between the log mortality rates insignificant (tests of significance not shown). At no

age group, however, do the point-estimates of all-cause logged mortality rates for the <HS population converge with the point-estimates of all-cause logged mortality rates for the >HS population. This evidence supports our first hypothesis. For black men and women, however, the educational gap in age-specific mortality becomes insignificant in older adulthood. The estimated age effects of <HS all-cause mortality converges with the estimated age effects of >HS all-cause mortality for both black men and women; this evidence does not support our first hypothesis. These mixed results suggest the possible need for the age-as-leveler and cumulative disadvantage theories to incorporate race differences in the way education affects mortality risk across age. Indeed, race differences in educational returns, race differences in mortality selection, and race differences in life course processes of health and mortality can each affect how the education-mortality association changes with age (Hayward et al. 2000; Lynch et al. 2003).

I next turn to Hypotheses 2, 3, and 4 in which I contrast the estimated period and cohort effects of all-cause mortality rates between 1986 and 2006 for black and white men and women in the United States. In reviewing the variance components of random cohort and period effects from models of black and white men's and women's all-cause mortality between 1986 and 2006 (Appendix Tables 1-4), I draw three conclusions. In general, (1) cohort variance in mortality is found to be significantly and sizably larger than period variance, (2) across all models, cohort variance is found to be largest in the >HS education groups, and (3) race differences are evident in both of these patterns. Overall, then, this evidence supports Hypotheses 2, 3, and 4.[8]

To best display the results of these hypotheses tests, I refer to Figure 5, which presents education-specific estimates of log rates of mortality across cohorts for black and white men and women. Figure 6 is also presented, which illustrates the relatively negligible period variation in

[8] Significant tests of random effects cohort and period coefficients were conducted following Yang, Frenk, and Land's (2009) four-step guide to assessing significance in CCREMs.

U.S. adult mortality. For white men, black men, and white women, we see strong evidence that educational differences in U.S. adult mortality grew across birth cohorts between 1986 and 2006. We observe significant and substantive declines in all-cause mortality for white men and women in the >HS education groups, yet much smaller reductions in mortality risk for the <HS groups. Among white women we also note an increase in the HS education group's mortality across recent cohorts. For black men, we see evidence of a small and largely stable educational gap in mortality, though a modest widening of this educational gap is occurring across cohorts. Small cell sizes preclude estimates of early cohort mortality patterns in the >HS education group for black men, but a steady cohort decline in mortality is evident from the 1915-1919 birth cohort through more recent cohorts. Because no cohort decline in black men's mortality is observed in the <HS education group, the education gap in black men's mortality steadily widens across cohorts. Collectively, these findings provide evidence that supports both Hypotheses 2 and 3, in that educational differences in adult mortality grew during the period 1986 to 2006, and that the educational gap grew across cohorts, not periods.

Findings for black women's period and cohort trends exhibit several patterns that differ from the findings for the other race-sex groups. First, consistent with Hypothesis 4 and with findings for black men's mortality, I find evidence that the education gap in black women's mortality is significantly smaller than the respective education gap in white men's and women's adult mortality. Second, and also consistent with Hypothesis 4, I find that cohort changes to the educational gap are more modest among black women and men than among white women and men. In fact, for black women, I find no evidence of cohort changes in the education-mortality relationship. Conversely, I find growing educational disparities in black women's adult mortality across periods. Beyond age and cohort effects, black women with <HS education exhibited rising

mortality rates across the 1986-2006 time period, whereas black women with higher levels of education exhibited no significant temporal change in mortality. The period effect could reflect a number of processes across this time period that disproportionately affected the mortality risk of low-educated black women. Changing labor market opportunities, for example, have produced absolute and relative loss of earnings for low-educated black women (Pettit and Ewert 2009). Also, the obesity epidemic has been shown to be both a cohort and period phenomenon that has also disproportionately affected the less educated black female population (Reither, Hauser and Yang 2009; Odgen et al. 2006).

Despite the differing findings for black women, our general findings show that, first, reductions in U.S. adult all-cause mortality between 1986 and 2006 were driven overwhelmingly by cohort processes. Second, these reductions in mortality risk were significantly conditioned by educational attainment. And third, the way educational differences in mortality changed across cohorts was conditioned by race and gender. As such, I find evidence supporting our second, third, and fourth hypotheses. Indeed, evidence overwhelmingly suggests that the education gap in U.S. adult mortality risk grew substantially across this period, supporting Hypothesis 2. Further, the growth in educational disparities in white men's, black men's, and white women's mortality is a cohort trend, not a period trend, supporting Hypothesis 3. And lastly, cohort changes to educational disparities in mortality differ significantly by race, supporting Hypothesis 4.

### 3.5.3 *Trends in Educational Differences in Cause-Specific Mortality*

I next review results that test our fifth hypothesis, in which I contend that cohort changes to educational disparities in mortality are stronger for heart disease and lung cancer mortality

than they are for unpreventable cancers. Figure 3.7 presents education-specific estimates of mortality from heart disease, lung cancer, and unpreventable cancer across age and cohorts for white men. Figure 3.8 presents these findings for white women.

Educational differences in men's heart disease and lung cancer mortality rates across age are similar to the age patterns of educational differences in all-cause mortality. In both cases, the education gap in mortality is both large and observed at all age groups. Contrasted with these findings are the estimated age patterns of educational disparities in white men's mortality rates from unpreventable cancers. In this case we observe a much smaller educational gap, which also is estimated to exist only among middle-aged adults. In short, the educational gap in white men's mortality from unpreventable cancers is small and becomes insignificant relatively early in the life course. Results from APC analyses of white women's heart disease and lung cancer mortality are similar to men's, in that the educational gaps in age patterns of mortality are larger for heart disease and lung cancer mortality than for mortality from unpreventable cancers. However, whereas estimated educational differences in heart disease and lung cancer mortality were observed at all ages for white men, educational differences in mortality from these respective causes of death converge at older ages for white women.

Regarding cohort changes in cause-specific mortality, our results provide evidence that strongly supports Hypothesis 5. In white men's and women's heart disease mortality, we observe a significant and substantial difference between those with a >HS education and those with a <HS education. Changes in heart disease mortality across education groups widened the educational gap by a significant amount for both men and women. For men, we observe a stall or increase across cohorts for the <HS group's heart disease mortality, whereas we observe

continued cohort reductions in the >HS group's heart disease mortality. For women, we observe a stall in heart disease mortality for the most recent cohorts with a <HS education.

Educational disparities in men's and women's lung cancer mortality across cohorts follow a striking resemblance to cohort and educational patterns of smoking (Preston and Wang 2006; Wang and Preston 2009; Pampel 2003). Also, like educational disparities in heart disease mortality, men's and women's lung cancer mortality for those with a <HS education exhibits very little cohort variation, with lung cancer mortality rates essentially remaining the same across cohorts born in the 1930s through the 1950s. On the other hand, men and women with >HS education exhibit substantive reductions in lung cancer mortality across cohorts born after 1930.

Contrasted with the large and growing educational gaps in heart disease and lung cancer mortality in the U.S. white male and female populations, are the cohort patterns of unpreventable cancer mortality. For both men and women, we observe much smaller and far more stable educational disparities in mortality. Indeed, estimates of both cohort and period variance are not significant, and Figures 3.7 and 3.8 reveal no temporal change in educational differences in men's and women's unpreventable cancer mortality.

Overall, our APC estimates of U.S. white men's and women's mortality rates between 1986 and 2006 from heart disease, lung cancer, and unpreventable cancer strongly support Hypothesis 5. That is, educational disparities in heart disease and lung cancer mortality are significantly larger than educational disparities in unpreventable cancer mortality. Moreover, educational disparities in heart disease and lung cancer mortality grew significantly wider across birth cohorts between 1986 and 2006. Contrasted with these patterns, I found no evidence of temporal changes to educational disparities in unpreventable cancer mortality.

### 3.6 Discussion

In 1965, Norman Ryder advanced the cohort as a unit of analysis for the study of social change. In appealing to fellow researchers, Ryder (1965: 845) stressed the cohort's "distinctive composition and character [that] reflect[s] the circumstances of its unique origination and history." He urged sociologists to focus on a cohort's unique "flow of person-years" across history to best understand the effects that diffusing social norms and changing environments have on society. Matilda White Riley (1973, 1978, 1987) advanced this line of thinking with her "principle of cohort differences in aging," urging social researchers to recognize that cohorts age in distinct ways. Unfortunately, this cohort perspective has been underutilized in studies of U.S. mortality trends. Some life course researchers, however, have recently pushed the field in this direction, and have empirically demonstrated that cohorts have disparate "morbidity phenotypes" as a product of exposures to early-life conditions (Crimmins and Finch 2006; Finch and Crimmins 2004; Fogel and Costa 1997). However, these life course and cohort processes are not limited to childhood environments, but rather are likely extended into adult environments as well. As childhood conditions improve, cohorts' varying exposures to subsequent advances in health-enhancing and/or health-protecting knowledge, practices, and technology produce disparate health and mortality outcomes across the life course.

In this chapter, I argued that education has played an increasingly important role in these cohort processes. As the United States underwent the epidemiologic transition during the early- and mid-twentieth century, the disease patterns and causes of death shifted from infectious and communicable diseases that largely affected infants and children, to chronic and degenerative diseases that overwhelmingly affected the aged (Omran 2005 [1971]; Olshansky and Ault 1986). Research has shown that beyond the immediate effects of these changing disease patterns in

childhood, the transition has slowly uncovered cohort effects on chronic disease susceptibility at older ages as well (Costa 2002; Finch and Crimmins 2004; Fogel 2004, 2005; Fogel and Costa 1997; Manton et al. 1997). Thus, cohorts born in the later stages or after the epidemiologic transition have likely endured fewer and less harsh insults as they have aged, thus being increasingly composed of a more robust cohort morbidity phenotype. As the effects of the early-life environment on later-life mortality lessened across U.S. cohorts, advantages and disadvantages during adulthood have increased in importance.

These results provide strong evidence consistent with Link and Phelan's (1995) fundamental cause theory, but do so with special attention given to the cohort-based effects of education. Specifically, my investigation yielded the following five findings about adult mortality trends and disparities. First, I found empirical evidence consistent with previous findings that recent temporal changes to U.S. adult all-cause and cause-specific mortality risk were driven overwhelmingly by cohort processes (Yang 2008). Second, consistent with past research (e.g., Meara et al. 2008; Montez et al. 2011), I found that educational disparities in U.S. adult mortality have grown over the past two decades. Third, I demonstrated that these changes in educational disparities in adult mortality operated on a cohort, rather than period, basis. That is, I showed that cohorts with <HS education did not experience the same reductions in all-cause mortality risk between 1986 and 2006 as those cohorts with >HS education. Fourth, I further showed that cohort changes to the U.S. education-mortality relationship are conditioned by gender and race. And fifth, consistent with fundamental cause theory, I found that cohort changes to the education-mortality relationship are especially strong for deaths from heart disease and lung cancer. In contrast, I found a smaller and more stable educational gradient in mortality from "unpreventable" cancers.

My analyses are limited in several ways. First, the NHIS-LMF 1986-2006 captures a rather recent and short period of time. While the lack of demonstrated period effects could be influenced by this data structure, I am encouraged by the similarities between my overall trend results and Yang's (2008) trend results from a different data set from 1960 to 1999. Second, due to small cell sizes for certain combinations of education levels and specific causes of death, I was forced to measure educational attainment with only three levels. It would be useful, for example, to separate the "greater than high school" education group into a "some college" group and a "bachelors degree or higher" group. Future analyses of all-cause mortality should be able to do so because more recent older birth cohorts are characterized by higher and higher levels of education. Third, analyses in this chapter tested important foundational hypotheses of mortality change, but did not focus on the specific mechanisms responsible for the cohort effects I uncovered. Future work in this area should aim to append cohort and period based measures of social change onto data sets like the NHIS-LMF to better understand such mechanisms. Fourth, the education-mortality relationship for black women in these data exhibit a pattern that is inconsistent with those found in the white men's, white women's, and black men's samples. The unique patterns of black women's cohort and period patterns of mortality change beg for further research.

Despite these limitations, the present study provides clear and strong findings regarding the nature of temporal changes to educational differences in U.S. adult mortality risk. Consistent with both a cohort perspective of mortality change and fundamental cause theory, I found that cohort processes are driving temporal changes in adult mortality risk and that educational differences in adult mortality risk are growing across birth cohorts. While the cohort patterns driving the growing educational divide in mortality differ to some degree by gender, race, and

cause of death, the overall change is directly counter to the major U.S. goal of eliminating disparities in health across socioeconomic groups (Healthy People 2010).

These important findings emphasize the need for researchers to integrate a sociological understanding of cohort change into the analysis of individual- and group-level mortality risk changes. As Ben-Shlomo and Kuh advise health researchers, "changing individuals must be studied in a changing world" (2002: 290). Understanding sociohistorical contexts are central to understanding temporal changes in mortality risk, and a cohort perspective rooted in rich sociological theory is necessary for understanding these contexts. Next steps should be to build off the current results to analyze the temporal changes in educational differences in U.S. adult mortality at the individual-level, build in both individual- and cohort-level mechanisms of change, and consider greater variations in societal contexts of historical changes in mortality risk. I conclude by emphasizing the need for researchers to integrate a cohort perspective into questions of historical shifts in adult mortality risk and to recognize the increasing importance that education is playing in shaping those risks.

**Table 3.1: Means of non-Hispanic White and Black Male and Female IHIS-LMF 1986-2006 Samples**

| | Men | | | | Women | | | |
|---|---|---|---|---|---|---|---|---|
| | Black | | White | | Black | | White | |
| | Mean | s | Mean | s | Mean | s | Mean | s |
| **Person-level Sample**[1] | | | | | | | | |
| Age | 46.49 | 14.9 | 47.88 | 15.2 | 46.66 | 15.3 | 49.06 | 16.0 |
| Year | 1994.26 | 5.4 | 1994.08 | 5.3 | 1994.17 | 5.4 | 1994.01 | 5.3 |
| Birth Year | 1947.77 | 15.8 | 1946.21 | 15.9 | 1947.51 | 16.1 | 1944.95 | 16.7 |
| % Less than High School | 29.52 | 45.6 | 15.50 | 36.2 | 28.15 | 45 | 15..15 | 35.9 |
| % High School Graduate | 37.86 | 48.5 | 35.13 | 47.7 | 37.12 | 48.3 | 39.79 | 49.0 |
| % Greater than High School | 32.61 | 46.9 | 49.37 | 50.0 | 34.73 | 47.6 | 45.05 | 49.8 |
| % Deceased | 19.98 | 40.0 | 16.64 | 37.2 | 14.93 | 35.6 | 14.34 | 35.1 |
| N | 54,236 | | 368,356 | | 78,280 | | 407,371 | |
| **Person-period Sample**[2] | | | | | | | | |
| Age | 49.70 | 13.2 | 52.21 | 15.0 | 50.82 | 13.0 | 54.00 | 15.8 |
| Year | 1999.46 | 4.9 | 1999.42 | 5.3 | 1999.54 | 4.5 | 1999.43 | 5.2 |
| Birth Year | 1949.76 | 13.4 | 1947.21 | 15.1 | 1948.72 | 13.2 | 1945.43 | 16.0 |
| % Less than High School | 26.43 | 41.5 | 14.29 | 35.8 | 26.16 | 38.5 | 14.39 | 35.4 |
| % High School Graduate | 39.09 | 45.9 | 35.56 | 49.0 | 38.02 | 42.5 | 40.61 | 49.5 |
| % Greater than High School | 34.48 | 44.7 | 50.15 | 51.2 | 35.82 | 42.0 | 45.00 | 50.1 |
| % Deceased | 1.48 | 11.3 | 1.29 | 11.6 | 1.09 | 9.08 | 1.10 | 10.5 |
| N | 637,699 | | 4,505,955 | | 962,276 | | 5,103,764 | |
| **168 APC-Education Cells**[3] | | | | | | | | |
| *< High School Sample* | | | | | | | | |
| 5-year Age Block [4] | 6.09 | 3.1 | 6.44 | 3.3 | 6.38 | 3.3 | 7.19 | 3.4 |
| 4-year Period Block [5] | 2.49 | 1.3 | 2.48 | 1.3 | 2.53 | 1.3 | 2.47 | 1.3 |
| 5-Year Cohort Block [6] | 8.82 | 3.2 | 8.47 | 3.4 | 8.57 | 3.3 | 7.70 | 3.4 |
| Cell Count Deceased | 61.59 | 58.6 | 219.19 | 243.1 | 67.13 | 70.1 | 242.25 | 284.4 |
| Cell Count N | 2132.30 | 1102.4 | 7505.02 | 3862.6 | 3025.10 | 1581.5 | 8357.18 | 4342.1 |
| *High School Sample* | | | | | | | | |
| 5-year Age Block | 4.02 | 2.6 | 4.92 | 2.9 | 4.39 | 2.7 | 5.59 | 3.1 |
| 4-year Period Block | 2.73 | 1.2 | 2.67 | 1.3 | 2.71 | 1.2 | 2.64 | 1.3 |
| 5-Year Cohort Block | 11.10 | 2.5 | 10.14 | 2.9 | 10.71 | 2.7 | 9.46 | 3.1 |
| Cell Count Deceased | 32.00 | 27.6 | 184.64 | 202.2 | 33.64 | 30.7 | 211.84 | 280.9 |
| Cell Count N | 4329.34 | 2963.1 | 23492.76 | 16051.2 | 5919.2 | 3992.9 | 27194.0 | 16781.3 |
| *> High School Sample* | | | | | | | | |
| 5-year Age Block | 3.94 | 2.4 | 4.73 | 2.7 | 3.98 | 2.5 | 4.65 | 2.9 |
| 4-year Period Block | 2.82 | 1.2 | 2.77 | 1.2 | 2.86 | 1.2 | 2.80 | 1.2 |
| 5-Year Cohort Block | 11.25 | 2.4 | 10.42 | 2.7 | 11.25 | 2.5 | 10.53 | 2.9 |
| Cell Count Deceased | 22.76 | 21.5 | 170.69 | 187.1 | 21.60 | 21.2 | 107.13 | 131.9 |
| Cell Count N | 3823.89 | 2687.0 | 35835.29 | 25354.7 | 5970.35 | 4287.7 | 36756.33 | 26942.9 |

1 Aged 25-84 at time of survey
2 Aged 25-99
3 Aged 25-99
4 Ranges from 0 to 14
5 Ranges from 0 to 4
6 Ranges from 1 to 16

**Table 3.2: Goodness-of-Fit Statistics for APC Fixed Effects Models**

| | non-Hispanic White Men | | | | non-Hispanic White Women | | | |
|---|---|---|---|---|---|---|---|---|
| | A | AP | AC | APC | A | AP | AC | APC |
| Deviance | 1929.3 | 1786.5 | 1494.6 | 1355.2 | 1701.0 | 1647.0 | 1587.3 | 1301.5 |
| AIC | 1959.3 | 1824.5 | 1554.6 | 1423.2 | 1731.0 | 1685.0 | 1647.3 | 1369.5 |
| BIC | 2006.1 | 1883.9 | 1648.3 | 1529.5 | 1777.9 | 1744.4 | 1741.0 | 1475.7 |
| df | 15 | 19 | 30 | 34 | 15 | 19 | 30 | 34 |
| | **non-Hispanic Black Men** | | | | **non-Hispanic Black Women** | | | |
| | A | AP | AC | APC | A | AP | AC | APC |
| Deviance | 1275.7 | 1225.9 | 1140.4 | 1122.3 | 1200.4 | 1194.9 | 1143.9 | 1103.9 |
| AIC | 1305.7 | 1263.9 | 1200.4 | 1190.3 | 1230.4 | 1232.9 | 1203.9 | 1171.9 |
| BIC | 1352.6 | 1323.3 | 1294.1 | 1296.5 | 1277.3 | 1292.2 | 1297.6 | 1278.2 |
| df | 15 | 19 | 30 | 34 | 15 | 19 | 30 | 34 |

Note: AIC is the Akaike Information Criterion and is estimated to be Deviance+2(df), and BIC refers to the Bayesian Information Criterion and is estimated to be Deviance+2((1/2)ln(N))(df)

**Table 3.3: HAPC-CCREM Estimates of White Male All-Cause Mortality, NHIS-LMF 1986-2006, by Educational Attainment**

| | <HS | | HS | | >HS | |
|---|---|---|---|---|---|---|
| | *B* | SE | *B* | SE | *B* | SE |
| **Fixed Effects** | | | | | | |
| **Age** | | | | | | |
| 25-29 | -2.290 | 0.337 | -2.285 | 0.235 | -2.054 | 0.248 |
| 30-34 | -2.268 | 0.155 | -2.139 | 0.127 | -2.104 | 0.140 |
| 35-39 | -2.140 | 0.118 | -1.923 | 0.102 | -1.825 | 0.109 |
| 40-44 | -1.677 | 0.096 | -1.610 | 0.085 | -1.702 | 0.091 |
| 45-49 | -1.370 | 0.080 | -1.218 | 0.071 | -1.344 | 0.075 |
| 50-54 | -0.997 | 0.063 | -0.880 | 0.058 | -0.925 | 0.061 |
| 55-59 | -0.517 | 0.045 | -0.595 | 0.044 | -0.630 | 0.048 |
| 60-64 | -0.246 | 0.030 | -0.261 | 0.031 | -0.273 | 0.035 |
| 65-69 | Ref | | Ref | | Ref | |
| 70-74 | 0.276 | 0.024 | 0.341 | 0.029 | 0.248 | 0.034 |
| 75-79 | 0.553 | 0.031 | 0.586 | 0.041 | 0.574 | 0.047 |
| 80-84 | 0.827 | 0.042 | 0.931 | 0.055 | 0.943 | 0.063 |
| 85-89 | 1.201 | 0.054 | 1.266 | 0.075 | 1.348 | 0.087 |
| 90-95 | 1.397 | 0.086 | 1.603 | 0.164 | 1.628 | 0.186 |
| 95-100 | 1.378 | 0.320 | 1.540 | 1.230 | 1.696 | 0.993 |
| Intercept | -3.520 | 0.099 | -4.037 | 0.071 | -4.383 | 0.075 |
| **Random Effects** | | | | | | |
| **Cohort** | | | | | | |
| 1900-1904 | 0.246 | 0.212 | 0.101 | 0.489 | 0.054 | 0.645 |
| 1905-1909 | 0.469 | 0.112 | 0.774 | 0.218 | 0.949 | 0.256 |
| 1910-1914 | 0.439 | 0.101 | 0.723 | 0.163 | 0.940 | 0.205 |
| 1915-1919 | 0.323 | 0.094 | 0.644 | 0.151 | 0.779 | 0.192 |
| 1920-1924 | 0.239 | 0.090 | 0.430 | 0.145 | 0.630 | 0.185 |
| 1925-1929 | 0.144 | 0.087 | 0.326 | 0.141 | 0.472 | 0.181 |
| 1930-1934 | -0.030 | 0.086 | 0.125 | 0.138 | 0.291 | 0.178 |
| 1935-1939 | -0.123 | 0.086 | -0.010 | 0.136 | 0.062 | 0.176 |
| 1940-1944 | -0.263 | 0.089 | -0.120 | 0.136 | -0.152 | 0.176 |
| 1945-1949 | -0.168 | 0.093 | -0.224 | 0.138 | -0.269 | 0.176 |
| 1950-1954 | -0.140 | 0.100 | -0.258 | 0.140 | -0.323 | 0.179 |
| 1955-1959 | -0.268 | 0.106 | -0.407 | 0.144 | -0.405 | 0.182 |
| 1960-1964 | -0.352 | 0.117 | -0.561 | 0.151 | -0.662 | 0.188 |
| 1965-1969 | -0.132 | 0.151 | -0.640 | 0.166 | -0.641 | 0.200 |
| 1970-1974 | -0.337 | 0.235 | -0.537 | 0.219 | -0.823 | 0.239 |
| 1975-1979 | -0.046 | 0.290 | -0.366 | 0.394 | -0.902 | 0.436 |

**Table 3.3 Continued**

| | | | | | | |
|---|---|---|---|---|---|---|
| **Period** | | | | | | |
| 1986-1990 | -0.125 | 0.056 | -0.203 | 0.076 | -0.141 | 0.075 |
| 1991-1994 | -0.084 | 0.051 | -0.037 | 0.067 | -0.091 | 0.062 |
| 1995-1998 | 0.005 | 0.050 | -0.009 | 0.065 | 0.005 | 0.059 |
| 1999-2002 | 0.074 | 0.050 | 0.090 | 0.065 | 0.066 | 0.059 |
| 2003-2006 | 0.129 | 0.052 | 0.159 | 0.068 | 0.161 | 0.062 |
| **Covariance Parameters** | | | | | | |
| Cohort | 0.089 | 0.067 | 0.253 | 0.137 | 0.434 | 0.224 |
| Period | 0.012 | 0.010 | 0.020 | 0.017 | 0.016 | 0.014 |
| Deviance | 126.72 | | 131.34 | | 186.06 | |
| N | 168 | | 168 | | 168 | |

**Table 3.4: HAPC-CCREM Estimates of Black Male All-Cause Mortality, NHIS-LMF 1986-2006, by Educational Attainment**

| | <HS | | HS | | >HS | |
|---|---|---|---|---|---|---|
| | *B* | SE | *B* | SE | *B* | SE |
| **Fixed Effects** | | | | | | |
| **Age** | | | | | | |
| 25-29 | -2.560 | 0.378 | -2.167 | 0.258 | -2.534 | 0.381 |
| 30-34 | -2.039 | 0.188 | -2.201 | 0.189 | -2.332 | 0.249 |
| 35-39 | -1.828 | 0.147 | -2.036 | 0.160 | -1.944 | 0.205 |
| 40-44 | -1.505 | 0.121 | -1.857 | 0.140 | -1.690 | 0.178 |
| 45-49 | -1.316 | 0.107 | -1.473 | 0.123 | -1.378 | 0.159 |
| 50-54 | -0.930 | 0.089 | -1.048 | 0.108 | -1.010 | 0.142 |
| 55-59 | -0.657 | 0.075 | -0.744 | 0.096 | -0.680 | 0.125 |
| 60-64 | -0.346 | 0.060 | -0.414 | 0.085 | -0.439 | 0.112 |
| 65-69 | Ref | | Ref | | Ref | |
| 70-74 | 0.281 | 0.052 | 0.439 | 0.080 | 0.372 | 0.105 |
| 75-79 | 0.541 | 0.057 | 0.755 | 0.093 | 0.754 | 0.122 |
| 80-84 | 0.923 | 0.064 | 0.984 | 0.110 | 1.143 | 0.141 |
| 85-89 | 1.164 | 0.076 | 1.556 | 0.135 | 1.522 | 0.172 |
| 90-95 | 1.453 | 0.101 | 1.777 | 0.219 | 2.279 | 0.245 |
| 95-100 | 1.077 | 0.210 | 1.193 | 0.810 | 2.173 | 0.656 |
| Intercept | -3.258 | 0.084 | -3.548 | 0.100 | -3.785 | 0.150 |
| **Random Effects** | | | | | | |
| **Cohort** | | | | | | |
| 1900-1904 | 0.003 | 0.115 | --- | --- | -0.004 | 0.348 |
| 1905-1909 | 0.033 | 0.084 | 0.162 | 0.175 | 0.068 | 0.221 |
| 1910-1914 | 0.066 | 0.074 | 0.166 | 0.140 | 0.082 | 0.188 |
| 1915-1919 | 0.044 | 0.067 | 0.034 | 0.121 | 0.213 | 0.167 |
| 1920-1924 | 0.062 | 0.063 | 0.075 | 0.109 | 0.244 | 0.153 |
| 1925-1929 | -0.012 | 0.062 | -0.051 | 0.104 | 0.105 | 0.146 |
| 1930-1934 | 0.031 | 0.063 | -0.004 | 0.100 | 0.085 | 0.139 |
| 1935-1939 | -0.052 | 0.067 | 0.060 | 0.099 | 0.055 | 0.138 |
| 1940-1944 | 0.032 | 0.072 | 0.020 | 0.102 | 0.025 | 0.136 |
| 1945-1949 | -0.074 | 0.081 | 0.042 | 0.105 | 0.012 | 0.136 |
| 1950-1954 | 0.016 | 0.088 | 0.150 | 0.107 | -0.017 | 0.140 |
| 1955-1959 | 0.028 | 0.096 | -0.034 | 0.117 | -0.055 | 0.148 |
| 1960-1964 | -0.107 | 0.109 | -0.071 | 0.128 | -0.133 | 0.168 |
| 1965-1969 | -0.048 | 0.131 | -0.275 | 0.165 | -0.367 | 0.215 |
| 1970-1974 | -0.011 | 0.149 | -0.248 | 0.211 | 0.010 | 0.241 |
| 1975-1979 | -0.030 | 0.160 | -0.075 | 0.239 | -0.281 | 0.353 |

**Table 3.4 Continued**

| | | | | | | |
|---|---|---|---|---|---|---|
| **Period** | | | | | | |
| 1986-1990 | -0.017 | 0.039 | 0.021 | 0.053 | 0.000 | 0.070 |
| 1991-1994 | 0.014 | 0.034 | -0.005 | 0.046 | 0.058 | 0.066 |
| 1995-1998 | 0.012 | 0.032 | 0.028 | 0.043 | -0.028 | 0.057 |
| 1999-2002 | 0.007 | 0.032 | -0.014 | 0.042 | -0.040 | 0.057 |
| 2003-2006 | -0.014 | 0.034 | -0.031 | 0.045 | 0.016 | 0.057 |
| **Covariance Parameters** | | | | | | |
| Cohort | 0.050 | 0.025 | 0.071 | 0.041 | 0.139 | 0.076 |
| Period | 0.003 | 0.005 | 0.005 | 0.064 | 0.009 | 0.022 |
| Deviance | 871.80 | | 757.81 | | 713.36 | |
| N | 168 | | 168 | | 168 | |

**Table 3.5: HAPC-CCREM Estimates of White Female All-Cause Mortality, NHIS-LMF 1986-2006, by Educational Attainment**

| | <HS | | HS | | >HS | |
|---|---|---|---|---|---|---|
| | *B* | SE | *B* | SE | *B* | SE |
| **Fixed Effects** | | | | | | |
| **Age** | | | | | | |
| 25-29 | -2.204 | 0.462 | -2.893 | 0.343 | -2.605 | 0.364 |
| 30-34 | -2.444 | 0.223 | -2.710 | 0.151 | -2.010 | 0.219 |
| 35-39 | -1.999 | 0.150 | -1.974 | 0.105 | -1.842 | 0.175 |
| 40-44 | -1.540 | 0.120 | -1.773 | 0.087 | -1.445 | 0.142 |
| 45-49 | -1.352 | 0.101 | -1.237 | 0.072 | -1.162 | 0.118 |
| 50-54 | -1.008 | 0.075 | -0.845 | 0.058 | -0.815 | 0.095 |
| 55-59 | -0.460 | 0.052 | -0.616 | 0.045 | -0.578 | 0.074 |
| 60-64 | -0.189 | 0.036 | -0.277 | 0.031 | -0.234 | 0.054 |
| 65-69 | Ref | | Ref | | Ref | |
| 70-74 | 0.244 | 0.028 | 0.326 | 0.027 | 0.327 | 0.048 |
| 75-79 | 0.498 | 0.035 | 0.650 | 0.037 | 0.666 | 0.061 |
| 80-84 | 0.850 | 0.044 | 1.056 | 0.048 | 1.032 | 0.077 |
| 85-89 | 1.204 | 0.054 | 1.460 | 0.062 | 1.477 | 0.093 |
| 90-95 | 1.571 | 0.068 | 1.915 | 0.091 | 1.894 | 0.112 |
| 95-100 | 1.670 | 0.129 | 2.224 | 0.294 | 2.079 | 0.142 |
| Intercept | -3.976 | 0.079 | -4.457 | 0.073 | -4.927 | 0.201 |
| **Random Effects** | | | | | | |
| **Cohort** | | | | | | |
| 1900-1904 | 0.440 | 0.169 | 0.166 | 0.344 | 1.109 | 0.249 |
| 1905-1909 | 0.465 | 0.109 | 0.556 | 0.151 | 1.031 | 0.223 |
| 1910-1914 | 0.387 | 0.102 | 0.548 | 0.128 | 0.862 | 0.211 |
| 1915-1919 | 0.298 | 0.097 | 0.436 | 0.120 | 0.760 | 0.202 |
| 1920-1924 | 0.190 | 0.093 | 0.317 | 0.114 | 0.581 | 0.194 |
| 1925-1929 | 0.102 | 0.091 | 0.196 | 0.110 | 0.461 | 0.187 |
| 1930-1934 | -0.029 | 0.091 | 0.036 | 0.108 | 0.260 | 0.182 |
| 1935-1939 | -0.086 | 0.092 | -0.106 | 0.107 | 0.070 | 0.180 |
| 1940-1944 | -0.162 | 0.095 | -0.230 | 0.108 | -0.205 | 0.181 |
| 1945-1949 | -0.161 | 0.101 | -0.353 | 0.110 | -0.320 | 0.184 |
| 1950-1954 | -0.315 | 0.113 | -0.501 | 0.114 | -0.470 | 0.190 |
| 1955-1959 | -0.389 | 0.122 | -0.463 | 0.120 | -0.696 | 0.200 |
| 1960-1964 | -0.338 | 0.136 | -0.349 | 0.128 | -0.743 | 0.214 |
| 1965-1969 | -0.132 | 0.181 | -0.307 | 0.152 | -1.008 | 0.252 |
| 1970-1974 | -0.250 | 0.263 | 0.166 | 0.230 | -0.929 | 0.316 |
| 1975-1979 | -0.019 | 0.306 | -0.112 | 0.367 | -0.987 | 0.537 |

**Table 3.5 Continued**

| | | | | | | |
|---|---|---|---|---|---|---|
| **Period** | | | | | | |
| 1986-1990 | -0.282 | 0.108 | -0.160 | 0.082 | -0.315 | 0.138 |
| 1991-1994 | -0.161 | 0.104 | -0.122 | 0.074 | -0.090 | 0.131 |
| 1995-1998 | -0.004 | 0.104 | -0.040 | 0.072 | 0.038 | 0.129 |
| 1999-2002 | 0.179 | 0.104 | 0.123 | 0.072 | 0.161 | 0.129 |
| 2003-2006 | 0.270 | 0.105 | 0.199 | 0.074 | 0.263 | 0.131 |
| **Covariance Parameters** | | | | | | |
| Cohort | 0.097 | 0.070 | 0.148 | 0.075 | 0.676 | 0.355 |
| Period | 0.053 | 0.039 | 0.025 | 0.019 | 0.097 | 0.130 |
| Deviance | 152.08 | | 155.01 | | 1016.94 | |
| N | 168 | | 168 | | 168 | |

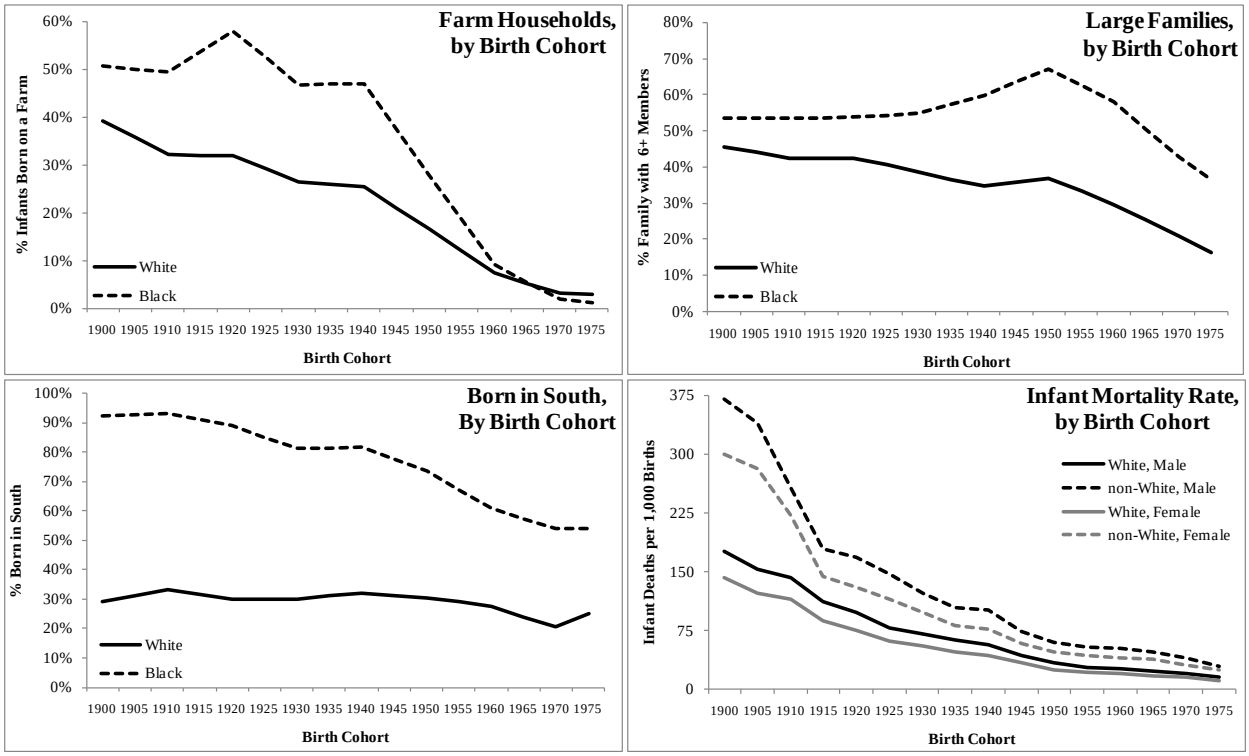
**Table 3.6: HAPC-CCREM Estimates of Black Female All-Cause Mortality, NHIS-LMF 1986-2006, by Educational Attainment**

| | <HS | | HS | | >HS | |
|---|---|---|---|---|---|---|
| | *B* | SE | *B* | SE | *B* | SE |
| **Fixed Effects** | | | | | | |
| **Age** | | | | | | |
| 25-29 | -2.752 | 0.439 | -3.147 | 0.385 | -3.377 | 0.453 |
| 30-34 | -2.498 | 0.237 | -2.435 | 0.196 | -2.722 | 0.234 |
| 35-39 | -1.933 | 0.161 | -2.076 | 0.156 | -2.412 | 0.189 |
| 40-44 | -1.705 | 0.138 | -1.732 | 0.134 | -2.054 | 0.165 |
| 45-49 | -1.134 | 0.111 | -1.466 | 0.121 | -1.589 | 0.145 |
| 50-54 | -0.794 | 0.095 | -0.967 | 0.105 | -1.196 | 0.132 |
| 55-59 | -0.541 | 0.080 | -0.628 | 0.092 | -0.957 | 0.124 |
| 60-64 | -0.226 | 0.066 | -0.287 | 0.080 | -0.448 | 0.108 |
| 65-69 | Ref | | Ref | | Ref | |
| 70-74 | 0.335 | 0.057 | 0.269 | 0.078 | 0.337 | 0.106 |
| 75-79 | 0.629 | 0.063 | 0.756 | 0.085 | 0.736 | 0.116 |
| 80-84 | 0.979 | 0.072 | 1.023 | 0.100 | 1.151 | 0.129 |
| 85-89 | 1.320 | 0.083 | 1.500 | 0.116 | 1.611 | 0.143 |
| 90-95 | 1.577 | 0.101 | 1.925 | 0.154 | 1.974 | 0.181 |
| 95-100 | 1.517 | 0.151 | 1.437 | 0.387 | --- | --- |
| Intercept | -3.798 | 0.138 | -4.017 | 0.098 | -4.091 | 0.117 |
| **Random Effects** | | | | | | |
| **Cohort** | | | | | | |
| 1900-1904 | 0.095 | 0.128 | 0.065 | 0.195 | 0.106 | 0.201 |
| 1905-1909 | 0.088 | 0.097 | 0.154 | 0.147 | 0.144 | 0.147 |
| 1910-1914 | -0.007 | 0.086 | 0.042 | 0.117 | 0.097 | 0.129 |
| 1915-1919 | 0.014 | 0.078 | 0.029 | 0.105 | -0.025 | 0.117 |
| 1920-1924 | 0.015 | 0.073 | 0.069 | 0.096 | -0.020 | 0.110 |
| 1925-1929 | 0.013 | 0.071 | 0.084 | 0.094 | -0.043 | 0.108 |
| 1930-1934 | -0.046 | 0.073 | 0.074 | 0.092 | -0.025 | 0.107 |
| 1935-1939 | 0.009 | 0.076 | -0.033 | 0.094 | 0.029 | 0.109 |
| 1940-1944 | -0.002 | 0.080 | 0.017 | 0.094 | 0.039 | 0.110 |
| 1945-1949 | -0.038 | 0.088 | -0.076 | 0.098 | 0.014 | 0.111 |
| 1950-1954 | 0.015 | 0.096 | -0.001 | 0.103 | 0.006 | 0.115 |
| 1955-1959 | 0.093 | 0.103 | -0.070 | 0.112 | 0.021 | 0.123 |
| 1960-1964 | -0.118 | 0.122 | -0.093 | 0.127 | -0.162 | 0.143 |
| 1965-1969 | -0.017 | 0.141 | -0.133 | 0.155 | -0.005 | 0.165 |
| 1970-1974 | -0.096 | 0.172 | -0.077 | 0.196 | -0.166 | 0.208 |
| 1975-1979 | -0.030 | 0.176 | 0.015 | 0.221 | -0.044 | 0.232 |

**Table 3.6 Continued**

| | | | | | | |
|---|---|---|---|---|---|---|
| **Period** | | | | | | |
| 1986-1990 | -0.202 | 0.123 | -0.022 | 0.052 | 0.013 | 0.053 |
| 1991-1994 | -0.019 | 0.120 | 0.027 | 0.046 | 0.001 | 0.046 |
| 1995-1998 | 0.071 | 0.120 | -0.009 | 0.041 | -0.016 | 0.043 |
| 1999-2002 | 0.150 | 0.121 | 0.026 | 0.041 | 0.003 | 0.041 |
| 2003-2006 | 0.108 | 0.123 | -0.018 | 0.041 | -0.001 | 0.041 |
| **Covariance Parameters** | | | | | | |
| Cohort | 0.031 | 0.020 | 0.054 | 0.029 | 0.058 | 0.031 |
| Period | 0.052 | 0.115 | 0.005 | 0.009 | 0.004 | 0.008 |
| Deviance | 883.81 | | 830.28 | | 722.35 | |
| N | 168 | | 168 | | 168 | |

**Figure 3.1: U.S. Birth Cohorts' Prevalence of Being Born into Farming Households, Large Families, and a Southern State, and Infant Mortality Rates.**




Sources: Birth cohorts born into farming households, large family size, and southern states estimated from Integrated Public Use Microdata (IPUMS), and infant mortality rates estimated from National Vital Statistics System's Historical Mortality.

**Figure 3.2: Age, Period, and Cohort HAPC-CCREM Estimated Mortality Rates, Black and White Men and Women, NHIS-LMF 1986-2006**

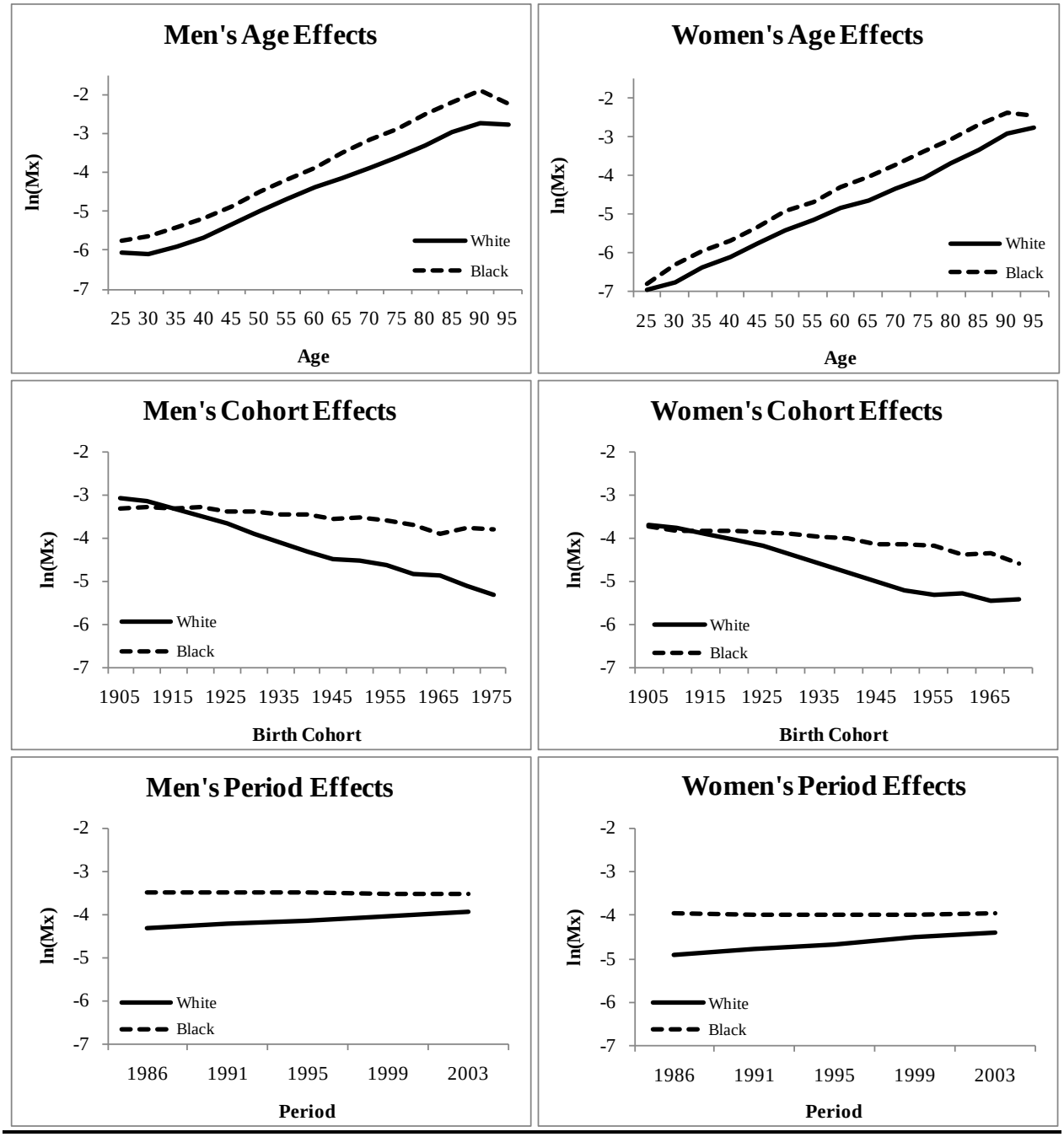


**Figure 3.3: Age, Period, and Cohort HAPC-CCREM Estimated Logged Mortality Rates from Heart Disease (HD), Lung Cancer (LC), and Unpreventable Cancers (UPC), non-Hispanic White Men and Women, NHIS-LMF 1986-2006**

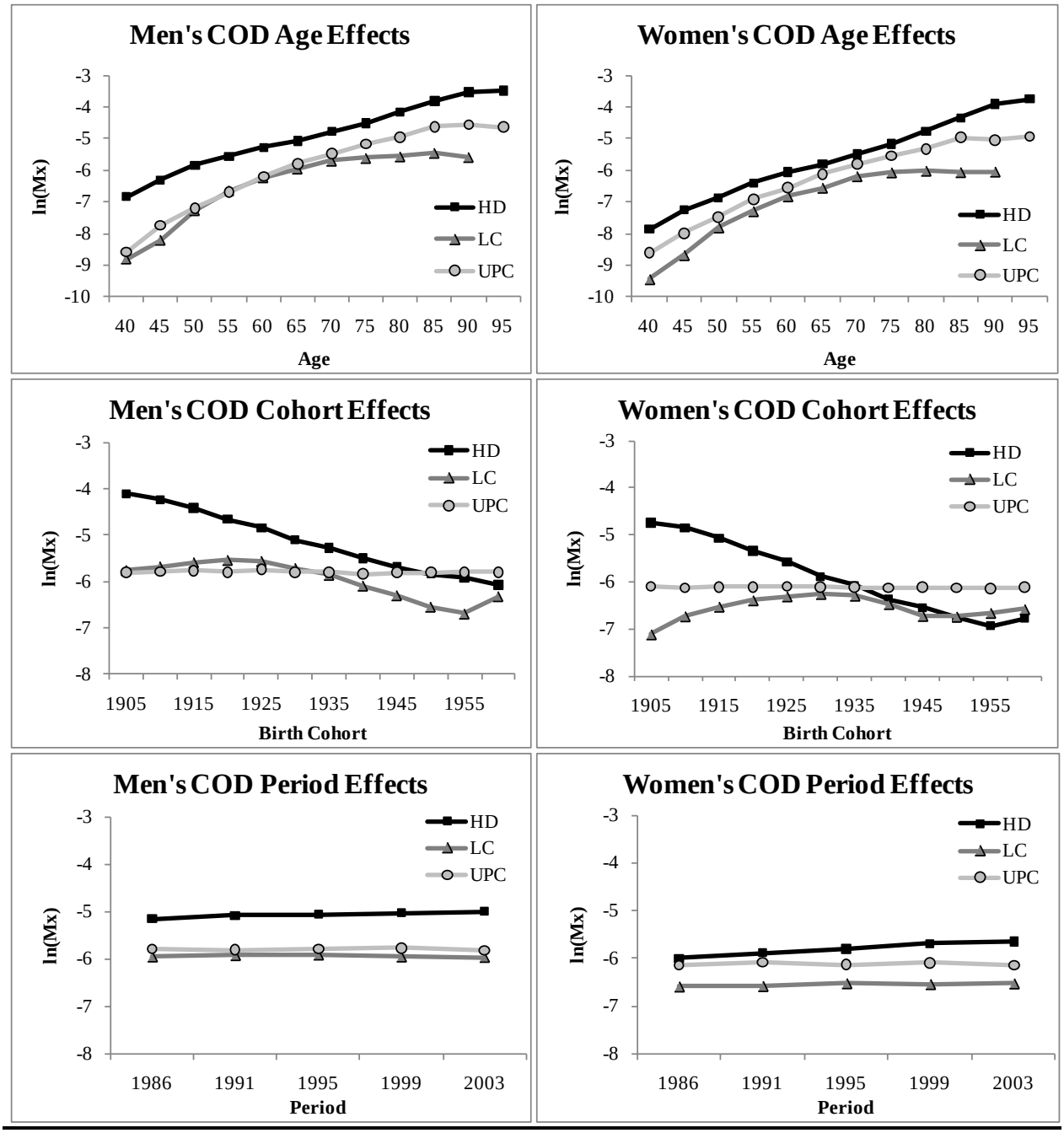


**Figure 3.4: Educational Differences in HAPC-CCREM Estimated Logged Mortality Rates across Age, Black and White Men and Women, NHIS-LMF 1986-2006**

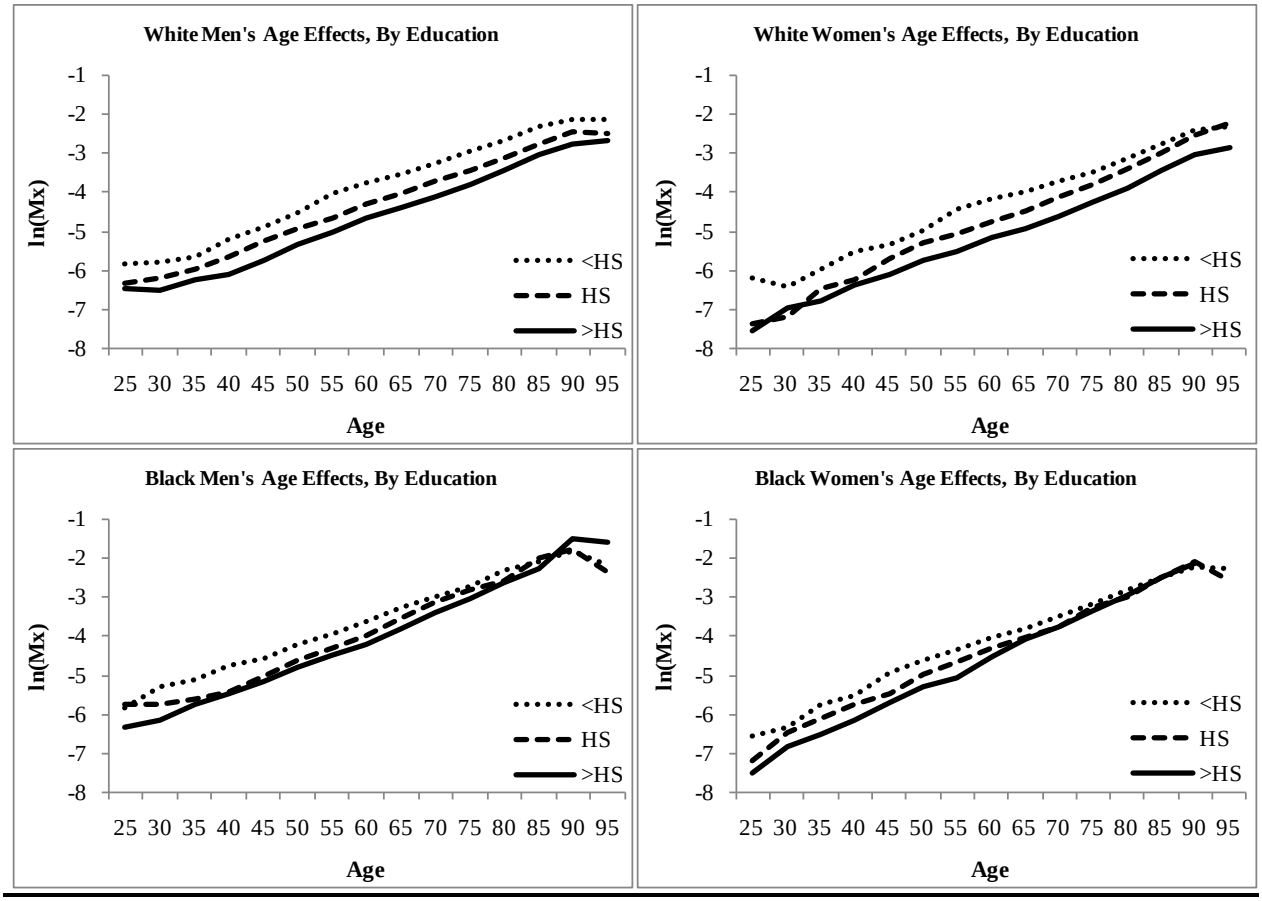


**Figure 3.5: Educational Differences in HAPC-CCREM Estimated Logged Mortality Rates across Cohorts, Black and White Men and Women, NHIS-LMF 1986-2006**

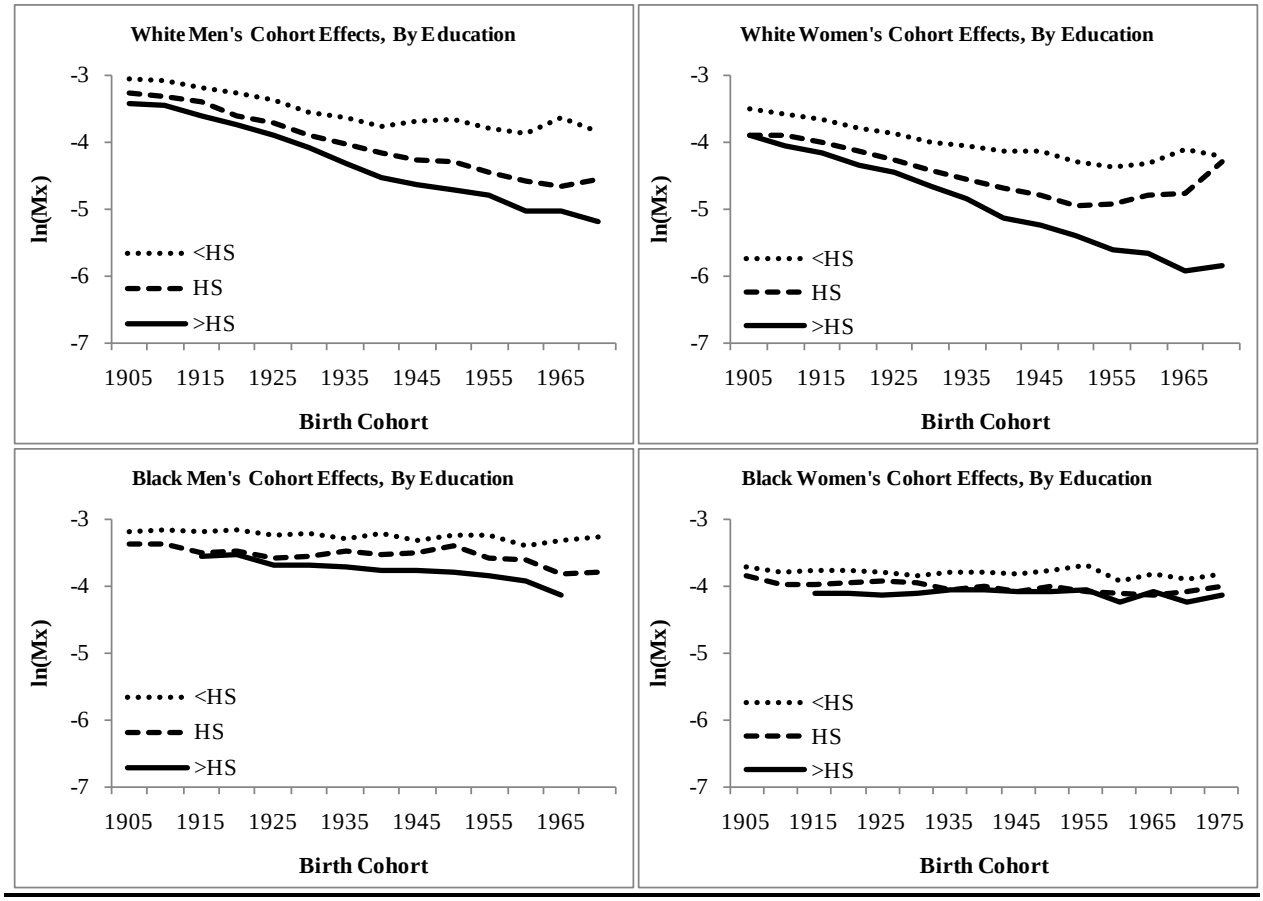


**Figure 3.6: Educational Differences in HAPC-CCREM Estimated Logged Mortality Rates across Periods, Black and White Men and Women, NHIS-LMF 1986-2006**

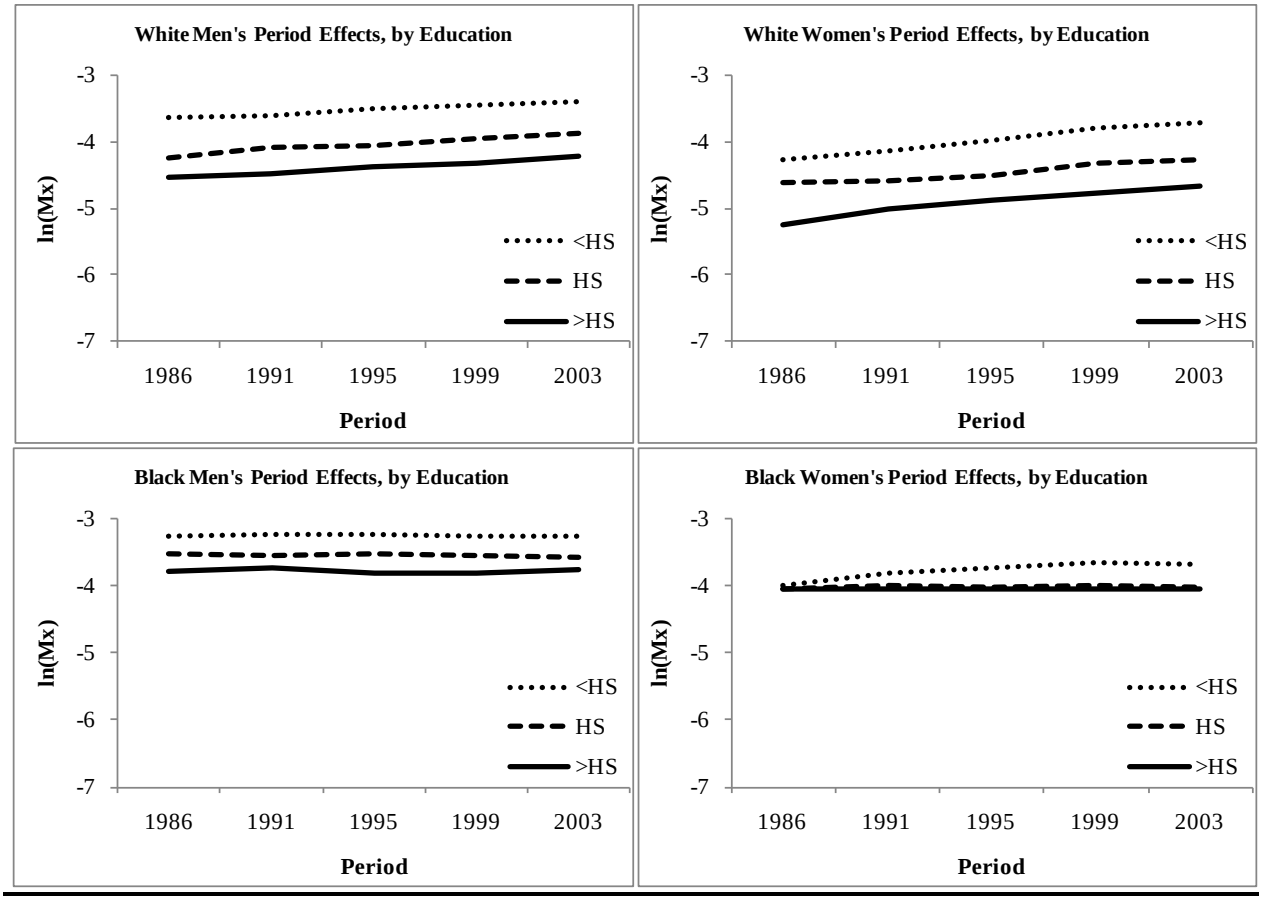


**Figure 3.7: Education-specific Cohort HAPC-CCREM Estimated Mortality Rates from Heart Disease, Lung Cancer, and Unpreventable Cancers for White Men, NHIS-LMF 1986-2006**

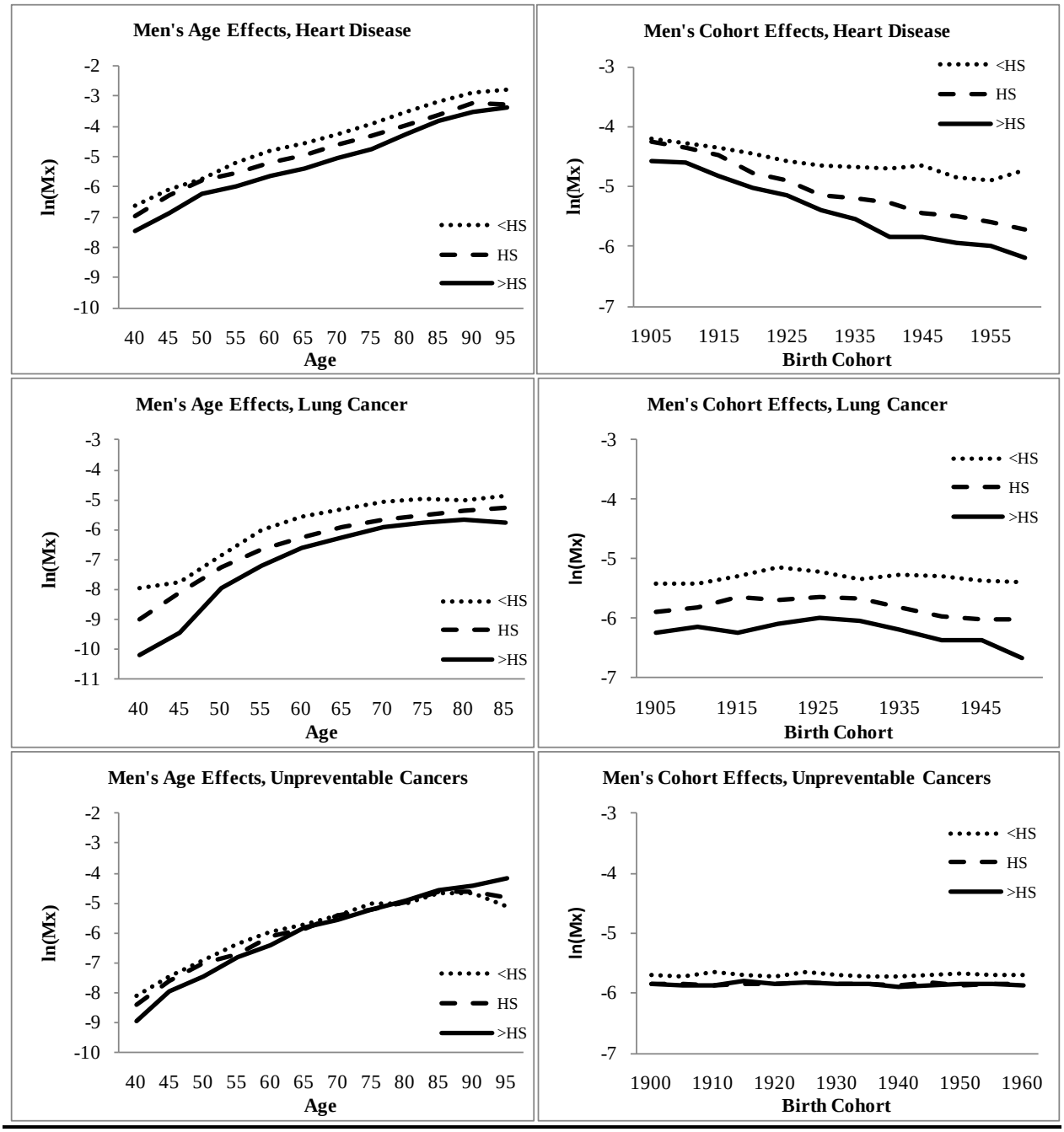
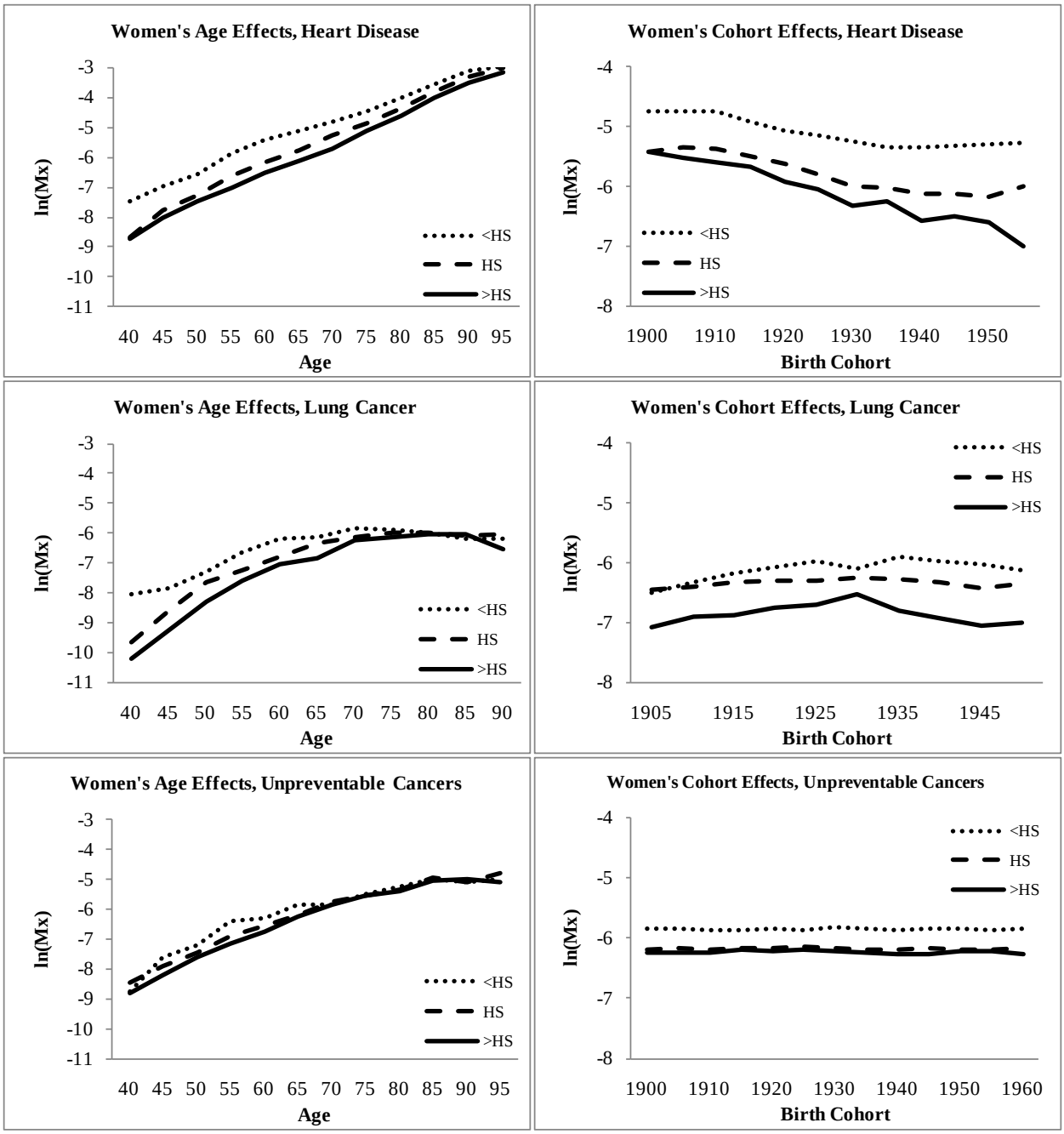


**Figure 3.8: Education-specific Cohort HAPC-CCREM Estimated Mortality Rates from Heart Disease, Lung Cancer, and Unpreventable Cancers for White Women, NHIS-LMF 1986-2006**



# CHAPTER 4

## The Color Line, Socioeconomic Resources, and Cohort Trends in U.S. Black and White Adult Mortality

### 4.1 Introduction and Hypotheses

Racial disparities in U.S. mortality rates are found to be substantively large, to occur at nearly all ages across the life course, and to have persisted across time (Hummer 1996; Hummer and Chinn 2011; Davey Smith et al. 1998; Sloan et al. 2010). Black-white differences in U.S. mortality rates are an important area of study, as they provide some of the best measures of the enduring racial inequalities in contemporary America. As such, racial disparities in health, disability, and mortality remain atop the list of health priorities in the nation (Healthy People 2020). While findings indicate that black-white differences in life expectancy at birth have slightly narrowed in recent decades (Harper et al. 2007), the gap remains distressingly large and many questions remain unanswered. In fact, a longer-term assessment of U.S. mortality shows that the relative gap in black and white men's mortality did not close in any significant way across the twentieth century (Sloan et al. 2010). Furthermore, there are severe limitations to using life expectancy to analyze the disparate mortality experiences of black and white Americans. For one, changes in life expectancy do not tell us where in the life course mortality risk has changed. Did the recent narrowing of the black-white mortality gap occur at all ages? Or has the recent narrowing stemmed from improvements in black Americans' survival across a particular age-range? Might the narrowing of the black-white gap in life expectancy reflect a relative worsening of white mortality across a particular age-range? These questions are difficult to answer when life expectancy is the sole measure used to gauge U.S. black and white mortality

trends. Second, measures of life expectancy do not tell us if changes are predominantly period or cohort phenomena. Have changes in the black-white gap in life expectancy resulted from changes in racial differences in health inputs during that time period, such as increased use of statins, antihypertensive drugs, antiretroviral AIDS medication, and other medication? (Chang and Lauderdale 2009; Macinko and Elo 2009; Anderson, Green and Payne 2009)? On the other hand, did the recent narrowing of the black-white gap in life expectancy simply reflect changing cohort composition of the black and white populations across that time period? Or, did the closing of the black-white gap in life expectancy reflect both of these processes? If the latter is the case, research and policy might need to consider how health inputs during a given time period can produce complex and disparate health benefits depending on a population's age and/or cohort composition (Fogel 2005). In fact, with regards to the narrowing of the black-white gap in life expectancy, the vast majority of the recent change can be attributed to the drop in black mortality from high rates of gang-related homicide and HIV/AIDS-related deaths during the late 1980s and 1990s (Figure 4.1). Thus, rather than reflecting sustained improvement in black survival relative to white survival, the recent narrowing of the racial gap in U.S. life expectancy has reflected age-specific period phenomena.

This point illustrates another problem with using life expectancy to assess the disparate mortality experiences of black and white Americans. What mechanisms of mortality are changing in the black and white populations that lead to corresponding changes to the black-white gap in life expectancy? Stratifying analyses by cause of death can touch upon some underlying factors, but we're mostly still left wondering about the processes and mechanisms behind such changes (Macinko and Elo 2009).

Most often, researchers point to racial inequalities in socioeconomic resources as the primary factors behind racial inequalities in U.S. health and mortality (Crimmins, Hayward, and Seeman 2004). However, this too has its limitations because "most of these studies fail to consider the sources of socioeconomic differences between blacks and whites" and "as a consequence, socioeconomic inequality is often controlled for in studies of black-white differences in health and mortality without being considered as an important consequence of racism in its own right" (Hummer 1996: 111). Indeed, as Hayward et al. (2000: 926) point out, "the greater prevalence of health problems among middle-aged blacks is the outcome of a *long-term and cumulative process* of health disadvantage over the life cycle" (emphasis added). Analyzing period-based changes in life expectancy cannot tap these long-term and cumulative processes, which lie at the heart of the black-white gap in U.S. mortality (Harris et al. 2006). It is becoming apparent that researchers must employ a historical and life course perspective to fully understand the origins and persistence of both socioeconomic and health inequalities between U.S. populations (Blackwell, Hayward and Crimmins 2001; Crimmins et al. 2004; Ferraro, Farmer and Wybraniec 1997; Hayward and Montez 2011; Shuey and Wilson 2008; Warner and Hayward 2006). Behind these cumulative processes are the unequal distribution and utilization of economic, material, and social resources across age and time between the U.S. black and white populations (Geronimus et al. 2010; Hayward et al. 2000; Shuey and Wilson 2008). These cumulative processes can begin in childhood or even earlier in life, and produce various lasting effects on the health and socioeconomic attainment of black and white populations (Barker 2007; Case and Paxson 2010; Montez and Hayward 2011; Fogel 2004; Palloni 2006; Warner and Hayward 2006).

In this chapter I compare age, period, and cohort patterns of U.S. black and white adult mortality risk, and analyze how childhood conditions and adult SES affect the racial gap in U.S. adult mortality. I advance a cohort perspective to explain why racial disparities in U.S. adult mortality are changing (or rather persisting), and the role socioeconomic resources play in these disparities. I draw from fundamental cause theory (Link and Phelan 1995), cumulative disadvantage theory, and the life course perspective (Ben-Shlomo and Kuh 2002; Montez and Hayward 2011), but couch these approaches in a cohort framework in order to better understand trends in the U.S. black-white mortality gap. This theoretical framework pulls heavily from Fogel and Costa's (1997) theory of "technophysio evolution" and Finch and Crimmins's (2004) notion of a "cohort morbidity phenotype." Central to my hypotheses are cohort changes to black and white early-life conditions across the twentieth century, and the implications these changes have for black-white differences in life course stratification of SES attainment and adult mortality risk. My five hypotheses are as follows:

*H1: Across the twentieth century, prevalence of harsh living conditions during childhood was higher in U.S. black cohorts than in U.S white cohorts.*

*H2: Improvements in childhood conditions occurred earlier in the twentieth century for U.S. white birth cohorts than for U.S. black birth cohorts.*

I aim to demonstrate that U.S. black cohorts' cumulative exposure time to harsh living conditions has been much greater than the cumulative exposure time endured by U.S. white cohorts. I further test if variation in childhood living conditions is associated with cohort changes to U.S. adult mortality risk:

*H3: Cohort-level measures of deleterious childhood conditions are significantly associated with U.S. black and white men's and women's adult mortality risk between*

*1986 and 2006.*

*a. These associations are partly mediated by adult SES, but remain significantly associated with adult mortality even after accounting for adult SES.*

If evidence supports Hypotheses 1-3, then I argue that the "long arm of childhood" is significantly stronger in shaping U.S. black cohorts' adult mortality than in shaping U.S. white cohorts' adult mortality (Hayward and Gorman 2004). This is not to argue that the individual effects of childhood conditions on adult mortality are stronger for black men and women than for white men and women. In fact, I make no predictions about possible racial differences in the substantive effects of early-life conditions on later-life mortality. But if harsh early-life conditions are significantly associated with adult mortality, and black cohorts endured greater exposure time to these early-life conditions, then the role these conditions play in shaping adult life chances and trajectories of attainment, adult health, and ultimately adult death are relatively bigger in the U.S. black population than in the U.S. white population. As a result of these historical changes to life course processes, and the racial differences in these changes, cohort reductions in U.S. adult mortality should be smaller in the U.S. black population than in the U.S. white population:

*H4: Cohort reductions in U.S. adult mortality between 1986 and 2006 were greater in the white population than in the black population.*

Also, as a result of changes in early-life conditions, changes in disease and cause-of-death patterns (i.e., the Epidemiologic Transition [Omran 1971; Olshansky and Ault 1986]), and improvements in health technologies, the adult environment has become more important over time in shaping adult mortality risk in the United States. Thus, individual resources that can be used to protect or enhance health in this adult environment are growing more important across

cohorts (results in Chapter 3 provide evidence supporting this contention). Furthermore, the power of individual resources in shaping U.S. adult mortality risk is hypothesized to differ by race. This racial difference in the association between socioeconomic resources and adult mortality can stem from a number of factors. First, changes in early-life conditions unfolded across U.S. black and white cohorts in different ways (Hypotheses 1 and 2). Thus, the degree to which deleterious childhood conditions still affect black and white cohorts' mortality risk should differ by race. That is, the variance in adult mortality associated with early-life conditions should be greater in the black population than in the white population, leaving less variance to be accounted for by adult socioeconomic resources (this is essentially Hypothesis 3 restated). Two, black men and women are more likely to face structural discrimination in schooling, housing, employment and other essential social and economic dimensions across the life course (Hummer 1996; Satcher et al. 2005; Charles and Hurst 2002; Ondrich et al. 2003; Gordon et al. 2000; Williams and Collins 1995). Therefore, black cohorts have been more likely to encounter barriers to their attempts to transfer socioeconomic resources into better health and lower mortality risk. That is, even if U.S. black men and women acquire the same level of socioeconomic resources in adulthood as white men and women, persistent discrimination hampers their ability to use these resources to their full potential to protect or enhance their health and survival (Kaufman, Cooper, and McGee 1997). And three, U.S. blacks are more likely than whites to endure chronic exposure to stressful situations, such as interpersonal discrimination, poor living conditions, and multiple caregiving roles, which have been shown to result in an allostatic load on the body's systems and increase its inflammatory processes (Geronimus et al. 2007). These, in turn, have been tied to heightened risk of immune, cardiovascular, obesity, and metabolic impairments and increased adult mortality (Geronimus et

al. 2011; Khansari et al. 2009; Simanek et al. 2008; Scharoun-Lee et al. 2009). Indeed, as early as age 30 black women have been shown to have significantly higher allostatic load scores than white men or women, which result in "an accelerated biological aging [in black women] of approximately 7.5 years compared with white women of the same chronological age" (Geronimus et al. 2007; Geronimus et al. 2010: 21, 29). For any and all of these reasons, the association between socioeconomic resources and U.S. adult mortality is conditioned by race in the United States. Thus, my last hypothesis is:

*H5: The association between adult mortality and adult socioeconomic resources is stronger in the U.S. white men's and women's populations than in the U.S. black men's and women's populations.*

a. *Specifically, the education and income gradients in adult mortality are larger in the U.S. white population than in the U.S. black population.*
b. *The education and income gradients in adult mortality are growing larger across birth cohorts in the U.S. white population than in the U.S. black population.*

Taken together, I aim to assess the degree to which black-white differences in U.S. adult mortality stem from more than just racial inequalities in the distribution of adult socioeconomic resources in the United States. How much of the black-white gap in mortality stems from lifelong, cumulative processes that begin in childhood? How much of the effect of adult socioeconomic resources on U.S. adult mortality stems from differential returns to education and income between the U.S. white and U.S. black populations?

I address these questions and test my hypotheses in several steps. First, I compare five indicators of childhood living conditions for U.S. black and white birth cohorts during the twentieth century (Hypotheses 1 and 2). Next, I examine age, period, and cohort patterns of

black-white differences in U.S. adult mortality rates during the time period 1986 through 2006 to demonstrate the significant and substantive racial differences in cohort effects on adult mortality (Hypothesis 4). I then incorporate measures of childhood conditions into the models to examine the extent to which cohort differences in black and white mortality are associated with disparate cohort changes to black and white men's and women's early-life conditions (Hypothesis 3). Next, I analyze how educational attainment, income, and poverty affect the age and cohort patterns of U.S. black and white men's and women's adult mortality. I do this separately for educational attainment and for income and poverty, and then together in a single model (Hypothesis 5). To conclude, I examine the effects of adult socioeconomic resources on the age and cohort patterns of black and white men's and women's adult mortality while controlling for cohort-level early-life conditions (further test of Hypotheses 3 and 5).

Findings provide evidence indicating: (1) U.S. black men's and women's adult mortality rates are falling across cohorts at slower rates than U.S. white men's and women's adult mortality rates, (2) variation in cohorts' childhood conditions is significantly associated with cohort reductions in U.S. black and white adult mortality, and (3) the effects of adult education, income, and poverty on U.S. adult mortality are significantly different for black and white men and women. In general, the educational and income gradients in U.S. adult mortality are greater in the white population than in the black population. Furthermore, these gradients are growing across U.S. white cohorts at significantly faster rates than across U.S. black cohorts. Taken together, the evidence supports past researchers' contentions that racial inequalities in adult mortality risk reflect cumulative stratification processes across the life course (Hummer 1996; Warner and Hayward 2006; Hayward et al. 2000; Crimmins et al. 2004). But the findings add to this literature by demonstrating the need to situate the life course within the contexts of cohorts'

unique experiences of history. Indeed, these stratification processes are inherently cohort phenomena, and they highlight the significant role race has played in shaping life course trajectories of health and socioeconomic attainment across U.S. history (Riley 1987; Ben-Shlomo and Kuh 2002; Hayward et al. 2000; Williams et al. 2010). In short, racial inequalities in U.S. adult mortality reflect more than unequal distributions in socioeconomic resources in adulthood. They reflect longstanding racial inequalities in both health and socioeconomic resources that stretch across both the life course and birth cohorts.

## 4.2 Background

### 4.2.1 *Socioeconomic Resources and the Black-White Gap in U.S. Mortality*

Racial disparities in health and mortality are widely studied from many disciplinary perspectives, yet they remain a pressing problem for U.S. health policy (Healthy People 2020). The age standardized death rate for the U.S. black population is comparable to the rates for whites a quarter century ago, and U.S. blacks have higher rates of disability, higher prevalence of extended periods of chronic illnesses, lower healthy life expectancies, and poorer self-rated health than U.S. whites (Clark 1997; Crimmins and Saito 2001; Kelly-Moore and Ferraro 2004; Hayward et al. 2000; Shuey and Wilson 2008; Williams and Collins 1995; Williams and Jackson 2005). It is the unfortunate case that for a long while racial disparities in health were explained by theories driven by beliefs in genetic differences and which sought to classify separate races of the human species along phenotypical lines. While these theories have been largely relegated to the fringe of contemporary scientific literature, it must be said that racial differences in various measures of U.S health and mortality reflect the social significance of race in America (Hummer 1996; Williams and Collins 1995). Indeed, in the United States, as elsewhere, race is a social

construct that has real and physical ramifications in the form of oppression, violence, denied opportunities, premature aging, and social and material inequalities (Williams et al. 2010; Geronimus et al. 2010). As such, racial categories in the United States reflect "both historical and contemporary social inequality and any attempt to understand racial disparities in health needs to consider the extent to which race is associated with SES" (Williams et al. 2010: 74).

Empirical evidence has suggested a strong intersection of race/ethnicity and socioeconomic resources in explaining health and mortality disparities (Crimmins, Hayward and Seeman 2004; Crimmins and Saito 2001; Hummer 1996; Hummer, Rogers and Eberstein 1998; Hayward et al. 2000; Rogers 1992; Rogers, Hummer and Nam 2000; Satcher et al. 2005; Williams and Collins 1995; Williams and Jackson 2005). Socioeconomic differences in health, disease prevalence, disability, and mortality have been documented in widely different times, countries, and populations (Dow and Rehkopf 2010). People with lower levels of education and with poorer financial resources have been found to be more likely to suffer from both infectious and chronic diseases, to be both physically and cognitively impaired, and to have significantly lower life expectancies than people with higher levels of socioeconomic resources (Preston and Taubman 1994; Williams 1990; Crimmins et al. 2004). And there is no doubt that the U.S. black population is disadvantaged relative to the U.S. white population in terms of educational attainment, income earnings, employment, wealth accumulation, and a number of other indicators of socioeconomic status (Williams and Collins 1995).

Yet there is no consensus on the degree to which racial disparities in the distribution of SES affect black-white health inequalities in the United States (Crimmins et al. 2004; Rogers 1992; Williams and Jackson 2005; Jemal et al. 2005). This is due in large part to the multitude of health measures (e.g., disease prevalence, disease incidence, functional limitation, disability, and

mortality, to name a few) and the various ways we can conceive of and measure socioeconomic status. In the broadest sense, socioeconomic status encapsulates long-term exposures to knowledge, opportunities, and material resources, and thus measures of SES attempt to reflect "the lifetime accumulation or experience of some types of capital" (Crimmins et al. 2004: 313). Education, thus, does well to represent social capital early in life and serves as a proxy for subsequent attainment of human capital across the life course, whereas income represents current or recent accumulation of material resources. For some measures of health, controlling for certain indicators of SES greatly reduces the black-white gaps in health and mortality. Huie et al. (2003), for instance, found that wealth, income, and education explain upwards of 50 percent of the black-white gap in mortality risk between ages 51 and 61. For other measures of SES and other measures of health, however, material resources explain very little of the racial differences in U.S. health (Crimmins et al. 2004; Kahn and Fazio 2005; Shuey and Wilson 2008; Hummer 1996). These seemingly inconclusive findings raise questions that are central to the ways by which black and white Americans are able to transfer their education, income, and other socioeconomic resources into good health and disability-free long lives. Indeed, Crimmins et al. (2004: 315) advise, "explaining the role of SES in racial and ethnic differences in health thus requires examining the relationship between race/ethnicity and lifetime SES as well as the link between SES and the potential mechanisms through which it works." In this sense, a life course perspective coupled with both cumulative disadvantage theory and fundamental cause theory presents a good framework to theorize and analyze the persistent gaps in black-white mortality (Kelley-Moore and Ferraro 2004; Link and Phelan 1995, 1996). Only by taking a "long view" of the stratification process can we uncover the origins and persistence of inequalities in childhood conditions (Palloni 2006; Warner and Hayward 2006), socioeconomic and status attainment

(Williams and Collins 1995), and stress processes that are detrimental to healthy and successful aging (Geronimus et al. 2010; McClaughlin et al. 2010; Ferraro and Kelley-Moore 2003).

### 4.2.2 *Beyond Adult SES: Childhood Conditions*

A life course perspective is particularly useful for analyzing racial differences in U.S. adult mortality risk. It forces us to look beyond contemporaneous and possibly static associations between adult SES and racial differences in adult mortality, and look instead to factors sustaining long-term stratification processes behind racial differences in health and socioeconomic resources (Hummer 1996). On the one hand, "social chains of risk" can originate in early life and are fueled by disadvantaged trajectories of subsequent socioeconomic attainment (Kuh et al. 2003; Palloni 2006). Household material resources during childhood, parental employment, and fractured and/or disrupted living conditions during childhood can negatively affect one's own cognitive abilities, trajectory of schooling, employment opportunities, and/or other pathways of material and social attainment (Warner and Hayward 2006). On the other hand, early-life conditions can operate through physical pathways, directly leaving indelible marks of disease susceptibility later in life. For instance, acute and/or chronic childhood infections and other sources of inflammatory processes have been shown to influence later susceptibility to certain chronic diseases (Barker 2007; Crimmins and Finch 2006; Gluckman et al. 2008; Simanek et al. 2008). Also, stunting from malnutrition and other interruptions in physical development of regulatory processes in the body have been found to be associated with a number of adult health outcomes (Case and Paxson 2010; Fogel 2004; Blackwell, Hayward, and Crimmins 2001). Furthermore, these physical pathways can originate prior to birth, reflecting poor uterine

environments stemming from a number of factors related to maternal health (Barker 1997; 2007; Fogel 2004).

These life course processes are not static, but instead reflect the unique historical circumstances of the time (Riley 1987). In short, they are cohort-specific phenomena. The changes in both the endowment of "health capital" across birth cohorts, and the depreciation of health resulting from poor childhood conditions each affect life course mortality risk of cohorts in different ways (Fogel 2004; Crimmins and Finch 2006). Indeed, evidence increasingly points to the important effect that cohorts' disparate lifetime exposures to infectious diseases, malnutrition, and bouts of inflammation have on subsequent health and mortality risk (Blackwell, Hayward, and Crimmins 2001; Finch and Crimmins 2004; Costa 2000; Fogel 2004, 2005). Analyzing mortality experiences of Swedish birth cohorts between 1751 and 1940, Finch and Crimmins (2004) find that those cohorts that were the first to experience lowered infant and childhood mortality were also the first to experience subsequent declines in older age mortality. It is believed, as such, that reductions in exposure to inflammation and infectious in early life directly led to decreases in subsequent chronic disease morbidity and mortality later in life. Finch and Crimmins (2004: 1739) thus argue that "improved childhood health and survival along with reduced chronic infections and inflammation…help to explain the widespread recent declines in old-age mortality." The authors posit that the aggregated insults of these early infections essentially scar a cohort, and that this scarring persists across the cohorts' life courses. Indeed, in their own words, these "enduring effects of early environment, even if conditions improved at later periods, could be designated as a 'cohort morbidity phenotype'" (2004: 1737). As cohorts differ in the magnitude of their "morbidity phenotype," it follows that they also differ in their susceptibility to later-life mortality risk.

Fogel (2004, 2005), Costa (2002), and Fogel and Costa (1997) have found similar results with various data sources, although their work has generally emphasized the synergism between improved nutrition, intergeneration transmissions of health endowments at birth, improved health-enhancing technologies, and reductions in early-life hardships across cohorts. Their theory of "technophysio evolution" also implies strong cohort effects in terms of both changes to health endowments at birth, improved diet, and disparate exposures to health risk across the life course. Their theory has profound implications for understanding sources of changes to adult mortality risk across cohorts. Arguing on behalf of a cohort perspective, Fogel (2005: S163) states, "not all improvements in the outcome of exposure to health risks between, say 1970 and 1990 are due to health interventions during that period. It could also reflect the improved physiologies experienced by later birth cohorts that are due to improved technologies in food production, public health practices, personal hygiene, diets, and medical interventions put into place decades before 1970, and hence cannot be attributed exclusively, perhaps even primarily, to health inputs between 1970 and 1990." Manton et al. (1997) made similar arguments when demonstrating cohort effects on both survival and functional capacity of the U.S. popualtion across age. They cite a number of improvements to diet (e.g., vitamin D supplementation during the 1920s, increases in vitamin $B_6$ fortified foods across the 1940s and 1950s, commercial food processing and increases in food regulation after the 1950s) and medical knowledge and practices (e.g., Jones Criteria for identifying and treating rheumatic fever) that were made across time in the United States. The combined effects of these health-enhancing developments on reducing chronic disease and mortality risk were largely related to cohorts' varying exposure times to their benefits.

Thus, like Finch and Crimmins, Fogel and colleagues also argue that reductions in disparities in childhood disease and mortality led to subsequent reductions in degenerative disease-related mortality risk at older ages as well. Citing work using Union Army data from the Early Indicators Project, Fogel (2005) shows that significant delays in the onset of chronic diseases across the twentieth century are linked to reductions of exposure to poor health early in life. And Costa (2000) estimates that as much as 10-25 percent of the decline in specific older aged chronic disease in the United States between 1900-1910 and 1971-1980 was due to decreases to specific infectious diseases during childhood and early life.

### 4.2.3 *The "Remarkable" Century*

To stress the magnitude of U.S. cohort changes in early-life conditions, I quickly review the dramatic increases in public health, nutrition, health, and survival across the twentieth century. In 1900, over 30 percent of deaths in the United States occurred in the childhood age range of 0-5 years. Characteristic of the first stage of the epidemiologic transition, the three leading causes of deaths were all infectious diseases (i.e., pneumonia, tuberculosis, and diarrheal diseases), which together accounted for about one third of all deaths in the United States (Omran 1971). Under these conditions, the U.S. population had only minimal knowledge of or access to proper nutrition (Fogel 2004), air-borne and water-borne infectious diseases were rampant (Cutler and Miller 2005; Colgrove 2002), and occupational hazards were extremely high (Rosner and Markowitz 1978; 1987). Household conditions were relatively harsh as well, regardless of geographical location or if one lived in an urban or rural setting (Easterlin 1997). Historically high fertility rates resulted in crowded dwellings and high rates of exposure to myriad infectious and parasitic diseases were common (Preston and Haines 1991). Furthermore, medical

knowledge and preventative and curative medical technologies were relatively off limits to the vast majority of the U.S. population.

Nevertheless, efforts were well underway to understand, control, and prevent outbreaks of infectious and parasitic diseases. Building on the foundation of recent discoveries in microbiology (e.g., advances in Germ Theory by Pasteur [1870s-1880s], Lister [1867], and Koch [1877]) as well advances in public health and nutrition, infectious and parasitic diseases were increasingly targeted and controlled. Indeed, already in 1900, 40 of the 45 states in the country had established a health department, and county-level health departments soon followed (CDC 1999). Subsequent advances in pharmaceuticals, such as the use of sulfa drugs in the 1930s and the discovery and application of penicillin in the 1940s, also helped to dramatically change the health environment of Americans (Jayachandran et al. 2010; Jones 1944). As a result of a number of changes, between 1900 and 1940 the overall levels of mortality fell faster in the United States than at any other time in history, with women and children experiencing the greatest reductions in mortality risk. It is not the point of this chapter to review these changes or engage in the debate about the causes of such changes (see Cutler et al. 2006; Easterlin 1997; and Fogel 2004 for more thorough reviews). However, it is my intent to highlight the remarkable shift in living conditions, reductions in infant and childhood diseases, and the epidemiologic shift that unfolded across cohorts born in twentieth century America. Indeed, by 2000, the age-adjusted death rate in the United States had been reduced by 56 percent when compared to 1900, and childhood deaths now account for only about one percent of all deaths in the United States (NCHS 2010; Guyer et al. 2000). Due to such unprecedented achievements, Fogel (2004) deemed the twentieth century to be “remarkable,” not only because of the historically unparalleled reductions in mortality but also because every commonly used indicator of standard

of living improved across this time. Furthermore, Fogel emphasizes that the most marked improvements were seen in the lower classes. While economic growth and urbanization had progressed in what would become the United States since pre-Revolutionary times, it was not until these material gains were coupled with, on the one hand, improvements in nutrition and, on the other hand, reductions in infectious disease via public health did the U.S. population significantly and sustainably reduce mortality (Fogel 2004; Preston 1980). Fogel emphasizes the synergistic relationship between nutrition and exposures to infectious disease to mark the "technophysio evolution" of human populations over the past three hundred years.

Fogel (2005) shows that both the overall prevalence and the disparities in poor early life conditions in the United States have decreased substantially. Consequently, as bouts with infection, malnutrition, and inflammation early in life have been greatly reduced across the twentieth century, the "insults" they imprint on the "cohort morbidity phenotypes" of successive birth cohorts are becoming less and less important in determining the risk of older adult morbidity and mortality risk. It follows, then, that improvements in adult conditions, rather than early life conditions, are becoming increasingly important across cohorts in shaping adult mortality risk (see Chapter 3 of this dissertation for more theory and evidence of this shift).

#### 4.2.4 *The Color Line*

While reductions in infectious diseases, enhancements in nutrition, and other improvements to childhood and maternal health dramatically increased survival in the United States across the twentieth century, these advances were not equally shared across the population. WEB Du Bois presciently warned that "the problem of the Twentieth Century is the problem of the color line" (1903: *The Forethought*). Du Bois was largely reflecting on what it

was to be black in post-emancipation America amidst continued and widespread institutionalized racism, but his statement was especially astute regarding the country's racial inequalities in health and mortality. Indeed, some of the greatest manifestations of racism and racial inequality in the United States are black-white disparities in health, disability, and length of life. Across the twentieth century, black Americans endured harsher living conditions, shorter lives, and greater disability than their white counterparts. These hardships reflected limited access to economic opportunities, hospital segregation policies (Almond, Chay, and Greenstone 2006), unequal living and working conditions, and the violent legacy of codified white supremacy (Massey and Denton 1993; Allen and Farley 1986). This was particularly the case during the first quarter of the century, at the time when the greatest advances in public health significantly benefitted the United States population. When the "remarkable" reductions in U.S. mortality unfolded across the first four decades of the twentieth century, black infant, child, and adult mortality rates remained significantly and stagnantly higher than white mortality rates at these respective ages. In fact, the black-white gaps in childhood and adult mortality remained stubbornly constant as these cohorts experienced the rapid increases in survival (Ewbank 1987). This can be seen both in all-cause mortality as well as for specific causes of death. While a substantial drop in mortality due to respiratory tuberculosis occurred between 1910 and 1920, the age-standardized mortality rates for whites dropped by 39 percent while the respective rates for blacks dropped only 26 percent (Ewbank 1987). Racial differences in subsequent reductions in mortality from tuberculosis and pregnancy/maternal causes are evident in Figure 4.2 (taken from Figure 6 of Jayachandran et al. 2010).

These disparate trends in U.S. black and white mortality came on the heels of the 1880s and 1890s, a time during which no significant changes occurred in black childhood mortality, but

significant mortality reductions occurred in the white population (Ewbank 1987). Thus, racial disparities in childhood survival grew wider across the decades leading up to the twentieth century, and, despite widespread advances in public health and improvements in survival across the twentieth century, the relative size of these racial disparities remained unchanged for forty years. My first and second hypotheses are thus:

*H1: Across the twentieth century, prevalence of harsh childhood conditions was higher in U.S. black cohorts than in U.S white cohorts.*

*H2: Reductions in harsh childhood conditions occurred earlier in U.S. white birth cohorts than in U.S. black cohorts.*

I draw from previous literature and also present five cohort measures to indicate the large and persistent racial differences in early-life conditions across twentieth century America. Existing literature and all five measures strongly support my first hypothesis that black cohorts born in the twentieth century endured higher prevalence of harsh early-life living conditions. First, to assess disparities during infancy I use data from the unpublished tables of the National Vital Statistics System's "Historical Mortality Data" to estimate white and non-white infant mortality rates (IMR). Results show extremely large racial differences in infant survival (Figure 4.3), despite rapid reductions in these rates across the first half of the century.

If we take these white and nonwhite IMRs as proxies for differential exposure to infectious disease, disparate access to good nutrition, and/or differences in maternal health – all of which have been demonstrated to be significantly associated with infant mortality (Fogel 2004) – then we can presume that deleterious conditions in the first year of life were much more

severe, much more widespread, or both more severe and widespread in U.S. black cohorts than in U.S. white cohorts.

Second, regarding childhood living conditions, Ewbank (1987) analyzed U.S. black and white childhood mortality rates between 1900 and 1940 and found that the probability of black death by age five "declined from 264 per 1,000 to 80 in 1940, a decline of 66 percent. During this same period the rate for whites continued the decline experienced between 1880 and 1900, dropping 67 percent from 161 in 1900 to 53 in 190" (108). Thus, despite substantial declines in black childhood mortality, the improvements across this time did not reduce the relative black-white inequalities in childhood mortality. In fact, U.S. black childhood mortality rates doggedly remained about 70 percent higher than white childhood mortality rates between 1910 and 1940 (Ewbank 1987).

Also, Figures 4.4 and 4.5 compare the regional and household conditions into which U.S. black and white cohorts were born. Here again, we note large racial differences in these factors. While Preston and Haines (1991) found significant protective effects on infant survival for U.S. cohorts growing up in rural areas and on farms, their analyses focused on U.S. infant mortality the late 1800s (i.e., prior to the rapid and transformative advances in public health and reductions in IMRs during the first decades of the twentieth century). And while Warner and Hayward (2006) also found a protective effect of being raised on a farm for U.S. black men's adult mortality, the reference category of their model was men born in large cities. It is possible that advances in nutrition, hygiene, prenatal care, and public health across the early 1900s altered the relationship between region, farming households, and child survival for white and black cohorts. Consistent with this idea, Ewbanks (1987) found evidence that black infant and child mortality between 1900 and 1930 was higher in New York State – where 88 percent of the black

population was concentrated in urban areas – than in North and South Carolina, whose populations were largely dispersed among rural farming households and small rural towns. Yet, through massive public health efforts the "urban penalty" in the United States was nearly eliminated by 1930, and by the late 1920s the black infant mortality rates in the three states were about equal (Cutler and Miller 2005). Furthermore, by 1940 the black infant mortality rate in New York was only 56, whereas it was 74 and 86 in North and South Carolina, respectively (Ewbanks 1987). Thus, we must be incredibly mindful of the rapid changes that were taking place during these times. While Preston and Haines (1991) and Warner and Hayward (2006) found a negative association between living in a farming household during childhood and adult mortality risk, their results may be driven largely by their reference category and the fact that their data are representative of older cohorts (late 1800s for Preston and Haines and 1906-1921 for Warner and Hayward). Especially important to consider is the racial component that surrounded and affected these changes. Consistent with Hypothesis 2, we see in Figures 4.4 and 4.5 that cohort changes in the prevalence of blacks being born on farms and being born in the South lagged behind cohort changes in the white population.

There are also remarkably large racial differences pertaining to various mechanisms by which childhood conditions directly affected U.S. childhood survival. Indeed, prenatal care, maternal and child nutrition, and household structure across the twentieth century were quite different for U.S. black and white populations, differences which were driven partly by the rural and farming lifestyles of southern blacks and the discrimination endured by both urban northern and southern blacks. Regarding prenatal care, the Children's Bureau study in rural Mississippi found that "79 percent of the white women were delivered by a physician but the proportion among blacks was only 8 percent [sic]. Similarly, one-third of white women received some

prenatal care, while the proportion among black women was only 12 percent" (Ewbank 1987: 123). Hospital segregation policies played a part in these disparities (Almond, Chay and Greenstone 2006), and the differences were especially pronounced in the south. While between 1915 and 1940 substantial changes were made to delivery practices in the United States, the advances resulted in 56 percent of white births taking place in hospitals by 1940 and only 22 percent for black births. And in the southern states the rates of hospitalized births were 36 percent in the white population and only 12 percent in the black population (Ewbank 1987).

Furthermore, regarding nutrition, "the Children's Bureau study of child care practices in a rural area of Mississipi in 1918, commented that 'the often-repeated criticism of the feeding customs of rural mothers that they feed their babies from the table at too early an age and delay weaning too long held true'" (Ewbank 1987: 118). Early weaning and the replacement of mother's milk with solid foods has been linked to poor child development, increased susceptibility to childhood infectious disease, and increased risk of morbidity and mortality (Claeson et al. 2003; Victora et al. 1987; WHO 2006). Large racial differences in infant weaning were found in early twentieth century America, with 60 percent of black infants and 35 percent of white infants receiving solid foods by their fourth month of life. These infant feeding patterns were also found in a similar study in North Carolina in 1916 (Bradley and Williamson 1918). The U.S. Children's Bureau conducted a series of studies in eight U.S. cities and documented a "devastating effect of mixed feeding (that is, a combination of breast milk and supplementary foods) and early weaning" (Ewbank 1987: 118). Racial differences in these patterns, and the fact that we note strong differences between blacks and whites born into farming and Southern families, strongly suggests substantial racial differences in infant, childhood, and developmental conditions for black and white cohorts born in these times. It is a likely case that poor, black,

farming mothers in the South during the early twentieth century were tasked with arduous duties of both home life and assisting on the farm. Time for infant care and breastfeeding, and maternal energy levels and health, were likely much lower for these mothers than for non-farming white mothers who, on average, worked out of the home less often and also had longer birth intervals. Consistent with this, the U.S. Children's Bureau further found that "father's income, employment of mothers, and shorter birth intervals accounted for much of the black-white differences in infant mortality rates in many of the areas studied" (Ewbank 1987: 118-119). Findings from Warner and Hayward (2006) also support this for black and white men's adult mortality. These findings also tie directly into Figures 4.6 and 4.7, which show black-white differences in the percent of cohorts born into large households (defined as having six or more members) and the percent of women aged 30-39 that were widowed across time periods.

Taken together, this evidence suggests harsh childhood living conditions were indeed much more prevalent and more persistent across U.S. black cohorts than across U.S. white cohorts. By coupling these findings with the growing evidence that tie early-life conditions to both direct and indirect effects on subsequent health outcomes and increased mortality risk, I draw three implications for recent trends in U.S. black and white adult mortality. One, early-conditions and U.S. adult mortality risk are becoming less associated across birth cohorts. That is, the total variance in U.S. adult mortality risk associated with conditions in childhood should be smaller in recent cohorts that endured fewer and less harsh early-life conditions. Because these recent cohorts undoubtedly comprise a greater proportion of the adult population, the aggregate association between childhood conditions and U.S. adult mortality risk should be getting weaker across birth cohorts. Second, we should see significant racial differences in these cohort changes to adult mortality because the historical changes in childhood conditions have

unfolded across U.S. black and white birth cohorts in remarkably different ways. And, third, because of the first two implications, we should find that socioeconomic resources in adulthood are becoming more strongly associated with U.S. adult mortality risk across cohorts, but that this change should be stronger in the U.S. white population than in the U.S. black population. Just as life expectancy in the U.S. black population is lagging some twenty years behind life expectancy in the U.S. white population, it is apparent that these cohort changes in the black population are lagging behind the cohorts changes in the white population as well.

Overall then, I presume that the long term effects of racial differences in early-life conditions, and the cohort changes to these conditions, are differentially affecting attainment of socioeconomic resources, adult health, and adult mortality risk of U.S. black and white populations (Gluckman et al. 2008; Warner and Hayward 2006; Hayward et al. 2000; Hayward and Gorman; Finch and Crimmins 2004; Case and Paxson 2010).

### 4.3 Current Aim

I revisit the black-white gap in U.S. adult mortality by integrating a cohort perspective of mortality change. I do so in four steps. First, I estimate age, period, and cohort patterns of the black-white gap in adult mortality between 1986 and 2006. I then estimate the amount of cohort variation in black and white men's and women's adult mortality associated with four cohort-level measures of childhood living conditions: the percent of a cohort born in the South, the percent of a cohort born on a farm, the percent of a cohort born into a large household, and a cohort's rate of infant mortality. Next, I estimate age, period, and cohort patterns of black and white men's and women's mortality across two adult-level measures of socioeconomic status: educational attainment and income level. I first estimate these APC patterns separately for

educational attainment and for income level and then again for educational attainment controlling for income level. Finally, I re-estimate the age, period and cohort patterns of black and white men's and women's mortality across adult-level socioeconomic status controlling for cohort-level measures of childhood conditions.

These analyses are performed to test my Hypotheses 3, 4 and 5. In general, I aim to assess the age-old question, "how much the black-white gap in U.S. adult mortality risk can be explained by measures of adult SES?" I argue that the educational and income gradients in U.S. black adult mortality are smaller than the respective gradients in U.S. white adult mortality, and thus explain only little of the racial gap in mortality. Furthermore, cohort changes to these gradients are stronger in the U.S. white population than in the U.S. black population.

### 4.4 Data and Measures

To analyze U.S. adult mortality I use data from nineteen National Health Interview Surveys (NHIS), 1986 through 2004, linked to follow-up mortality information for each cross-section through December 31$^{st}$, 2006 (NCHS 2010). This linked data set was concatenated and made publicly available by the National Center for Health Statistics (NCHS) and the Integrated Health Interview Series (IHIS) project at the Minnesota Population Center (Ruggles 2011). The NHIS uses a multistage probabilistic sampling design, and respondents of the NHIS are matched to the mortality records of the National Death Index using a 14-item identification scheme (NCHS 2010). Respondents of the NHIS not eligible for matches to death records are dropped from the final sample. The resulting 1986-2006 National Health Interview Survey-Linked Mortality Files (NHIS-LMF) are a unique combination of repeated cross-sectional surveys coupled with longitudinal annual records of individual respondents' survival status.

In order to ensure enough time for individuals to complete all measured levels of educational attainment, to focus on ages where mortality risk is high and death counts were sufficiently plentiful, and to limit the use of data where age is top coded, I restricted the NHIS-LMF to U.S.-born black and white respondents aged 25 to 84 at time of survey and who were 30-99 years of age during the follow-up period. I also dropped from the sample any respondent with missing values of educational attainment, income, poverty status, and employment status. Lastly, to ensure I am capturing early-life conditions in the United States, all respondents not born in the United States were dropped from the sample. Limiting the data in these ways resulted in starting analytic sample sizes of 306,287 white males; 335,573 white females; 42,423 black males; and 60,846 black females.

Each subsample was then transformed into person-year samples to create individual-level survival times, and these individual survival histories were further collapsed into aggregated subsamples of age-period-cohort blocks. The coding of birth cohort comprises 15 five-year blocks ranging from 1900-1904 to 1970-1974. The coding for period results in five distinct blocks ranging from 1986-1990 to 2003-2006; the earliest period block spans five years because it contained the fewest number of deaths, while the remaining four periods spanned four years each. And the coding of age is in fourteen 5-year blocks ranging from 30-34 up to 95-99. Combining the blocks together, the sex- and race-specific samples for my adult mortality analyses each comprise 157 unique APC blocks. Table 4.1 displays descriptive statistics for the individual-level data.

### 4.4.1 *Adult Socioeconomic Resources*

Some researchers have argued that the association between SES and adult mortality follows a linear functional form (Adler et al. 1994; Pappas et al. 1993), while others have argued that the relationship is predominantly driven by the concentration of mortality at the lower end of socioeconomic attainment (Finch 2003). Research has shown that a linear functional form of the education-mortality relationship inadequately captures all the effects of education on U.S. adult mortality (Montez et al. 2011). Thus, in the present studies, I use three categories of each adult SES measure to guard against extrapolations of effects across a linear functional form: less than high school (<HS), high school or equivalent (HS), and greater than high school (>HS). These categories have been shown to capture much of the differentiation in U.S. mortality risk by educational attainment (Montez et al. 2011).

The educational composition of the U.S. population changed substantially across the twentieth century, for both white and black populations, with cohorts born early in the century being disproportionately composed of persons with a less than high school education (see Figure 4.8). Conversely, cohorts born in the middle of the century experienced improved educational achievement, with the majority of U.S. cohorts born since mid-century attaining a high school degree or higher. The aggregated change in educational attainment is thought to be a primary factor in the temporal decline of U.S. adult mortality (Yang 2008; Lynch 2003). Here, I stratify my analyses by educational attainment to allow for education-specific estimates of APC patterns of mortality. Educational differences in mortality were tested by estimating and contrasting education-specific confidence intervals of age, period, and cohort effects on mortality. I also use three categories of income to capture variation in the association between household income and adult mortality: at or below the poverty line, above poverty line but earning less than $45,000; and earning more than $45,000. This coding allows me to test where in the gradient of income

black-white differences in mortality are most pronounced. Black-white differences in U.S. income across cohorts are evident in Figure 4.9, with a significantly greater proportion of the black male and female samples reporting incomes below the poverty threshold. These distributions of education and income are consistent with other national estimates (Devas-Walt, Proctor and Smith 2010).

### 4.4.2 *Early-life Indicators*

Estimates of cohort-level early-life conditions were obtained from multiple sources. Using the integrated public-use microdata (IPUMS) from U.S. Censuses 1900 through 1980 I estimated the percent of a cohort born in southern U.S. states, the percent of a cohort born on a farm, and the percent of a cohort born into a household with six or more members (i.e., measures presented in Figures 4.4-4.6). Because these data were collected every ten years, measures for the five-year birth cohorts falling between these data are linear averages of adjacent estimates. These estimates were calculated for the black and white samples separately. Cohort-specific infant mortality rates by race (white and nonwhite) and sex were obtained from the National Vital Statistics System's (NVSS) "Hist290" historical data document (http://www.cdc.gov/nchs/nvss/mortality/hist290.htm).

## 4.5 Analytic Methods

I estimated U.S. non-Hispanic black and non-Hispanic white men's and women's adult mortality rates using Bayesian Hierarchical Age-Period-Cohort (HAPC) cross-classified random effects models. All models are stratified by sex and race/ethnicity and limited to native-born U.S.

respondents aged 25-84 at time of survey, and aged 30-99 in the analyses. Models were estimated in the following succession:

1. black/white, male/female stratified APC;
2. black/white, male/female APC with cohort-level childhood conditions;
3. black/white, male/female APC with individual-level adult education varying across age and cohort;
4. black/white, male/female APC with individual-level adult income varying across age and cohort;
5. black/white, male/female APC with individual-level adult education varying across age and cohort and non-varying individual-level adult income;
6. black/white, male/female APC with individual-level adult education varying across age and cohort, non-varying individual-level adult income, and cohort-level childhood conditions.

These models were estimated separately by sex and race/ethnicity to establish the black-white differences in age, period, and cohort patterns of U.S. adult mortality between 1986 and 2006. To test Hypothesis 3, I estimated the association between U.S. adult mortality and cohort-level early life conditions by modeling mortality on each early-life condition separately. I then estimated a series of nested models that regress rates of mortality on the full set of early-life measures. Lastly, I estimated the association between cohort-level early-life conditions while including individual-level adult SES as mediators of the association.

To test Hypothesis 5, I estimated the association between U.S. adult mortality and age- and cohort-varying individual-level adult SES, measured as educational attainment and household income. These associations were modeled separately, and then I re-estimated the

association between educational attainment and U.S. adult mortality while including household income as a mediating effect.

The HAPC-CCREM estimates fixed effects of the five-year age groups and random effects of the four-year period and five-year cohort groups, and is structured in the following way:

Level-1 within cell model:

$$\log[E(D_{ijk})] = \alpha_{jk} + \beta_{jk} A_i + \log(R_{ijk})$$

for the early-life model:

$$\log[E(D_{ijk})] = \alpha_{jk} + \beta_{1jk} A_i + \beta_2 ELI + \log(R_{ijk})$$

for the education model:

$$\log[E(D_{ijk})] = \alpha_{1jk} + \beta_{1jk} Agths_{1i} + \beta_{2jk} Ahs_{2i} + \beta_{3jk} Alths_{3i} + \log(R_{ijk})$$

for the income model:

$$\log[E(D_{ijk})] = \alpha_{2jk} + \beta_{1jk} Apov_{1i} + \beta_{2jk} Amidinc_{2i} + \beta_{3jk} Ahighinc_{3i} + \log(R_{ijk})$$

for the education and income model:

$$\log[E(D_{ijk})] = \alpha_{1jk} + \beta_{1jk} Agths_{1i} + \beta_{2jk} Ahs_{2i} + \beta_{3jk} Alths_{3i} + \beta_4 pov + \beta_5 highinc + \log(R_{ijk})$$

for the early-life, education, and income model:

$$\log[E(D_{ijk})] = \alpha_{1jk} + \beta_{1jk} Agths_{1i} + \beta_{2jk} Ahs_{2i} + \beta_{3jk} Alths_{3i} + \beta_4 pov + \beta_5 highinc + \beta_6 ELI + \log(R_{ijk})$$

where $D_{ijk}$ stands for the counts of deaths of the $i$th age group (for $i$ = 1, …, $n_{jk}$ age groups) within the $j$th period (for $j$ = 1, …, $J$ time periods) and the $k$th cohort (for $k$ = 1, …, $K$ birth cohorts); $A_i$ denotes a dummy variable corresponding to each five-year age groups 1,…, $n_{jk}$; *ELI* represents early-life indicators corresponding to each variable 1,…,$n$; $Agths_{1i}$ denotes a dummy variable corresponding to each of the five-year age groups 1,…, $n_{1jk}$ for >HS educational

attainment; $Ahs_{2i}$ denotes a dummy variable corresponding to each of the five-year age groups 1,…, $n_{2jk}$ for HS educational attainment; $Althss_{3i}$ denotes a dummy variable corresponding to each of the five-year age groups 1,…, $n_{3jk}$ for <HS educational attainment; $Apov_{1i}$ denotes a dummy variable corresponding to each of the five-year age groups 1,…, $n_{1jk}$ for poverty status; $Amidinc_{2i}$ denotes a dummy variable corresponding to each of the five-year age groups 1,…, $n_{2jk}$ for income greater than poverty status but less than $45,000; $Ahighinc_{3i}$ denotes a dummy variable corresponding to each of the five-year age groups 1,…, $n_{3jk}$ for income>$45,000; *pov* denotes a time-, age-, and cohort-invariant fixed effect of poverty status; *highinc* denotes a time-, age-, and cohort-invariant fixed effect of income>$45,000; $\alpha_{jk}$ is the intercept indicating the reference age group (65-69) who was in period *j* and belong to cohort *k*; and log($R_{ijk}$) is the natural log of the aggregated exposure time lived during each age-period-cohort cell.

Level-2 between cell random intercept model:

$$\alpha_{jk} = \pi_0 + t_{0j} + c_{0k},$$

for the education model and income model:

$$\alpha_{1jk} = \pi_{10} + t_{0j} + c_{10k} + c_{20k} + c_{30k}$$

in which $\alpha_{jk}$ specifies that the fixed age effects vary from period to period and from cohort to cohort; $\pi_0$ and $\pi_{10}$ the expected mean at the reference age (65-69; and 65-69 for >HS education or 65-69 for mid-income) averaged over all periods and cohorts; $t_{0j}$ is the overall four-year period effect averaged over all five-year birth cohorts with variance $\sigma^2_{t0}$; and $c_{10k}$ and $c_{20k}$ and $c_{30k}$ are the education (or income)-specific five-year cohort effect averaged over all four-year periods with variances $\sigma^2_{k10,}\sigma^2_{k30,}\sigma^2_{k30}$.

I combine the level-1 and level-2 models to estimate the expected log counts of deaths in each APC cell assuming that deaths follow a Poisson distribution. The aggregated exposure time lived within the cells is used as an offset to the model in order to generate results in the form of APC-specific log mortality rates. Hierarchical Bayesian Models using a Gibbs sampling approach were used for all analyses (Gelman et al. 2006; Lynch 2007). I assumed noninformative prior distributions for all model parameters (Gelman 2006; Lynch 2003).

**4.6 Results**

Table 4.2 and Table 4.3 show estimated cohort and period variance components of U.S. black and white women's and men's adult mortality rates between 1986 and 2006, respectively.

For both men and women, results from the "APC" model indicate that the U.S. white population experienced significantly more cohort variation in adult mortality between 1986 and 2006 than the U.S. black population. Further evidence of these racial differences in cohort adult mortality trends is shown in Figure 4.10, which plots Bayesian estimates of individual random cohort effects of U.S. black and white men's and women's adult mortality trends.

This evidence strongly supports Hypothesis 4, in that we see significantly greater cohort reductions in U.S. adult mortality rates for the white men's and women's populations than the black men's and women's populations.

In reviewing Tables 4.2 and 4.3, we find evidence that much of this cohort variation, irrespective of race or gender, is associated with the prevalence of U.S. birth cohorts being born in the South. Cohort variance components for U.S. white and black men's and women's adult mortality have all been significantly reduced by accounting for percent of cohort born in the South, and the individual fitted standardized coefficients of "% Born in South" are all significant

at the .001 α-level. Results show that a one standard deviation increase in the percent of a cohort being born in the South is associated with over a .3 standard deviation increase in U.S. adult mortality between 1986 and 2006. This finding holds for white and black men's and women's mortality, with little variation in the substantive effect on adult mortality, save for a slightly smaller association with black men's mortality.

For U.S. white men and women, and for U.S. black women, this cohort-level association between percent born in the South and adult mortality risk is entirely mediated by cohorts' household living conditions and level of infant mortality. The association between U.S. white and black women's cohort prevalence of being born in the South is accounted for by the cohort prevalence of being born on a farm, which in turn is partly mediated by cohort variation in infant mortality rates. For U.S. white men, the association between the percent of a cohort born in the South and adult mortality is mediated by the percent born on a farm, the percent born into large households, and cohorts' levels of infant mortality. For U.S. black men, the association between the percent of a cohort born in the South and later-life mortality is not accounted for by variation in either of the household measures or infant mortality rate. The persisting association between the percent of a cohort born in the South and black men's adult mortality possibly suggests that growing up in the South present threats to black males' health and survival that reach beyond childhood, and/or hinders their attainment trajectories. Indeed, evidence from subsequent models that incorporate individual-level covariates suggests that the cohort-level effect of black men being born in the South is significantly reduced by variation in individual-level educational attainment and income (see models "Early-Education" and "Early-Education-Income").

Table 4.4 and Table 4.5 present estimates of random cohort and period variance components, and estimates of individual-level adult socioeconomic resources and cohort-level

childhood living conditions on U.S. adult men's and women's mortality across a series of HAPC-CCREMs. Men's results are presented in Table 4.4, and estimates for women are found in Table 4.5.

### 4.6.1 *Black-White Differences in the Education-Mortality Association*

#### 4.6.1.1 *Men*

Results from the "Education" model in Table 4.4 provide evidence that strongly supports both parts a) and b) of my fifth hypothesis. Regarding part a), which proposed that the educational gradient in U.S. adult mortality between 1986 and 2006 was larger for white men than black men, I find that at age 65 U.S. white men with a <HS education have a log mortality rate 1.062 higher than white men with a >HS education. The respective difference for U.S. black men is only .661. In more practical terms, we can see the racial difference in the effect of educational attainment on U.S. men's adult log mortality rate in Figure 4.11.

For U.S. men with a <HS education, we observe no significant difference in white and black men's adult mortality (top left panel of Figure 4.11). Yet, when we compare the fitted log mortality rates of U.S. black and white men with a >HS education we note a significant racial difference in men's adult mortality at all age groups. Findings therefore suggest that U.S. white men derive a significantly greater protective effect on their mortality from educational achievement than do U.S. black men.

Results from the men's sample provide evidence supporting part b) of my fifth hypothesis as well. Education-specific cohort covariance parameters indicate greater cohort variation in U.S. white men's mortality rates at higher levels of educational attainment than at lower levels of educational attainment, and the differences in these variations are significantly

greater than those in U.S. black men's mortality. These differences are most clearly shown in the bottom panels of Figure 4.11, wherein the education-specific log mortality rates at age 65 of U.S. black and white men are plotted across birth cohorts. For black men, we observe no significant cohort changes to the adult mortality risk of the <HS and >HS populations. As a result, the educational gradient in U.S. black men's adult mortality rates remained unchanged across the time period 1986 to 2006. For U.S white men, we observe reductions across birth cohorts 1900 and 1940 in adult mortality rates for both the <HS and >HS populations. Thereafter, however, we see no cohort reductions for the <HS white men's population but continued cohort reductions in the >HS white men's population, thereby increasing the educational gradient in U.S. white men's adult mortality.

#### 4.6.1.2 *Women*

Results presented in Table 4.5 show educational gradients in U.S. white and black women's adult mortality, and cohort changes therein, that are largely consistent with the patterns observed in U.S. men's adult mortality. One gender difference is worth additional attention. The racial difference in the educational gradient in U.S. women's adult mortality is significantly larger than the racial difference in the educational gradient in U.S. men's mortality. U.S. white women with a <HS education have an estimated log mortality rate 1.269 higher than U.S. white women with a >HS education. For U.S. black women, the log mortality difference between <HS and >HS is only .275, which is insignificant at all commonly used α-levels. The implications of this racial difference in the educational gradient of U.S. women's adult mortality are depicted in Figure 4.12.

Similar to the finding for U.S. men, no significant racial difference is found in adult mortality rates for U.S. women with a <HS education (top left panel of Figure 4.12). However, when we look at the effect of high educational attainment on U.S. black and white women's adult mortality, we see a significant racial difference, and this black-white gap grows with age. Thus, like the results found in the men's samples, I find evidence supporting part a) of my fifth hypothesis. The effect of educational attainment on U.S. adult women's mortality between 1986 and 2006 is found to have been significantly and substantively stronger for white women than for black women. Evidence supporting part b) of hypothesis 5 is especially strong in women's cohort patterns of U.S. adult mortality trends. Like cohort changes to U.S. black men's educational gradient in adult mortality, no evidence is found to suggest cohort changes to the educational gradient in U.S. black women's adult mortality between 1986 and 2006. Conversely, cohort changes to U.S. white women's educational gradient are significant and growing quite rapidly. U.S. white women with a <HS education are simply not keeping up with the pace of cohort reductions in adult mortality experienced by white women with a >HS education. The result, therefore, is a large and growing education-gap in U.S. white women's adult mortality.

### 4.6.2 *Black-White Differences in the Income-Mortality Association*

#### 4.6.2.1 *Women*

Results from the "Income" model in Table 4.5 are consistent with the effects of education on U.S. women's adult mortality rates in Table 4.5. The income gradient in U.S. women's adult mortality is significantly conditioned by race, and the differences are most evident at the lower end of the income distribution. However, it must be pointed out that the coefficients presented in Table 4.4 and Table 4.5 represent the effects of income level on mortality at age 65. To best

illustrate racial differences in the associations between poverty status and high income with U.S. women's adult mortality, I have plotted the effects across age in Figure 4.13.

Results show that income-mortality relationship for U.S. white women increases with age, whereas the income gradient in U.S. black women's mortality is both smaller than the gradient in the white female population and depreciates with age. Also, we see that no significant racial difference in U.S. women's mortality exists in the population with incomes below the poverty threshold. Conversely, a significant racial difference in U.S. women's mortality is evident for women making over $45,000, and this racial gap in mortality grows with age. All of these findings provide further support for part a) of Hypothesis 5. Results from black and white women's "Income" models also provide evidence supporting part b) of Hypothesis 5. The income gradient in U.S. black women's mortality between 1986 and 2006 remained quite stable across birth cohorts. U.S. white women's income gradient in adult mortality, on the other hand, grew significantly and sustainably across birth cohorts.

#### 4.6.2.2 *Men*

U.S. men's results from the "Income" model in Table 4.4 are consistent with the patterns observed in U.S. women's income-mortality association. The effect of an income below the poverty line on U.S. white men's mortality at age 65 is 1.562, which is significantly and sizably larger than the .479 effect for U.S. black men. Also, as seen in Figure 4.14, the income gradient in black men's mortality is much smaller than the gradient in white men's mortality at all ages. Furthermore, as is the case for U.S. black women, the income gradient in black men's mortality diminishes with age and becomes insignificant at the oldest age-groups. In contrast to these patterns, the income gradient in U.S. white men's adult mortality increases across age.

This is also the case when we compare the black-white gap in U.S. men's mortality at different income levels. For U.S. men earning incomes below the poverty line, white men's mortality rates are estimated to be higher than mortality rates in the black men's population, though these differences are insignificant at all commonly used α-levels. Among men with incomes greater than $45,000, the black-white gap is significant and there is some evidence indicating that the racial gap grows larger with age.

Results from the "Income" models reveal no significant cohort changes to the income gradient in U.S. black men's mortality rates. This is most clearly shown in the bottom left panel of Figure 4.14. Regarding the income gradient in U.S. white men's adult mortality, results from the "Income" model depict interesting and unique changes to adult mortality between 1986 and 2006. While the income gradient in white men's adult mortality is, on average, the largest among the four race/sex-populations, results provide no evidence suggesting any cohort changes in the income-gap of U.S. white men's mortality risk. The income gap is significantly large across all cohorts, but the mortality reductions in the population earning income below the poverty line kept pace with the reductions in mortality rates for the U.S. white men's population earning more than $45,000. In each population, cohort changes in the U.S. white men's mortality has stalled across birth cohorts 1945-1949 to 1970-1974. This stalling in cohort changes to the mortality risk of men with a >$45,000 income has resulted in a mortality difference between U.S. white men earning high incomes and U.S. white men earning mid-level incomes that is insignificant in more recent cohorts.

In general, evidence supports both part a) and part b) of my fifth hypothesis. The educational and income gradients in the U.S. white men's and women's populations are significantly larger than the respective gradients in the U.S. black populations, and the cohort

growth of these gradients are larger in the white population than in the black population. As such, evidence suggests that the U.S. white population derives significantly more health benefits from adulthood SES than the U.S. black population. This implies that the black-white gap in adult mortality reflects more than racial inequalities in the distribution of these socioeconomic resources. Indeed, among those black and white men and women with the same socioeconomic resources, whether it be education or income, the mortality benefit of such SES attainment was greater in the U.S. white population than in the U.S. black population. To further support this pattern, I next re-estimate the "Education" and "Education-Income" models while controlling for the effects of cohorts' early-life conditions on later-life mortality rates.

### 4.6.3 *Adult SES and Early-life Indicators*

#### 4.6.3.1 *Men*

Accounting for cohorts' variation in early-life conditions significantly changes the educational gradient in both the U.S. white and black men's adult mortality rates between 1986 and 2006. For white men (Table 4.4), all education-specific variance components are significantly reduced, and the individual-level effect of a <HS education at age 65-69 has been reduced from 1.062 to .863. Thus, I find evidence that a significant proportion of the education-mortality relationship in U.S. white men is explained by the possibly disparate early-life conditions for the <HS and >HS white male populations. Also, surprisingly, the previously insignificant difference (.294) between the HS and >HS white men's populations is now a significant .544 difference after controlling for cohort variation in U.S. white men's cohorts' early-life conditions.

We find similar effects in the U.S. black men's sample. The .661 difference between the log mortality rates of the <HS and the >HS population has been reduced to .622 when accounting for variation in the percent of U.S. black cohorts born in the South. Also consistent with findings in the white male sample, the H.S.->H.S. difference in black men's mortality significantly increased from .452 in the "Education" model to .597 in the "Edu-Early" model.

Accounting for the mediating effects of income of U.S. adult men's mortality further reduces education's effect on black men's adult mortality, and also changes the substantive effects of early-life conditions of adult mortality (see "Early-Edu-Inc" model results in Table 4.4). The difference in logged rates of mortality between U.S. white men with a <HS education and U.S. white men with a >HS education has been reduced from 1.062 in the "Education" only model to .863 in the "Early-Edu" model to .728 in the "Early-Edu-Income" model. The mortality difference between HS and >HS groups in the U.S. white male population is entirely explained by variation in income. The effect of cohort variation in early-life conditions on U.S. white men's adult mortality was reduced by accounting for adult education and income, suggesting a possible mediating effect of significantly different socioeconomic attainment for cohorts with various early-life conditions. Supportive of this idea is the fact that accounting for cohort variation in U.S. white men's early-life conditions accounts for more of the mortality difference between <HS and >HS white men's mortality rates than the mediating effects of individual-level income (i.e., the <HS to >HS effect of 1.062 is reduced to .913 when accounting for individual-level income, whereas it's reduced to .863 when accounting for cohort-level early-life conditions).

#### 4.6.3.2 *Women*

Accounting for early-life conditions in the adult SES-mortality relationship in the U.S. white and black women's populations produce results that are inconsistent with those found in the men's models (Table 4.5).

First, far more than in the men's models, the effects of early-life conditions on U.S. women's adult mortality remain largely unchanged after accounting for adult educational attainment. For white men, the effects of % Born on Farm, % Born in Large Family, and Infant Mortality Rate were reduced from .219; .278; and .278 to .163; .230; and .267, respectively. For U.S. white women, the significant effects of % Born on Farm and Infant Mortality Rate on adult mortality were reduced from .381 and .268 to .375 and .250, respectively, when accounting for adult education. Furthermore, educational attainment reduced the effect of % Born in the South on U.S. black men's adult mortality from .316 to .269. For U.S. black women, accounting for individual-level education reduced the effects of % Born on a Farm and Infant Mortality Rate from .284 and .214 to .242 and .211, respectively. Overall then, findings suggest that the association between early-life conditions and adult mortality risk in the U.S. women's population is not explained by subsequent educational differences in adulthood. On the other hand, in the U.S. men's population, a significant proportion of the effects of early-life conditions on adult mortality is accounted for by educational attainment in adulthood.

This difference is also apparent in the persisting effects of educational attainment on U.S. women's adult mortality. When controlling for both cohort variation in early-life conditions and also accounting for income's mediating effects of educational attainment on adult mortality, we see that the effect of <HS and HS on U.S. white women's mortality are insignificantly changed from 1.269 to 1.273 and from .553 to .526, respectively. For U.S. black women, I find peculiarly changing results pertaining to the effects of educational attainment on adult mortality. As

previously stated, the educational gradient in U.S. black women's mortality in the NHIS-LMF 1986-2006 was estimated to be insignificant (<HS effect of .275 and HS effect of .018). When controlling for early-life conditions, however, the educational gradient actually becomes larger and significant. In the "Early-Education" model presented in Table 4.5, we see that the <HS effect is now a significant .649 and the HS effect is a significant .523. Thus, disparate variation in cohort early-life conditions mask the effects of educational attainment on U.S. black women's adult mortality. This could stem from a number of processes related to the cohort and education composition of black women represented in the NHIS-LMF, but future research is necessary before drawing any substantive conclusions.

**4.7 Discussion**

Racial differences in U.S. health and mortality persisted across the twentieth century, despite major advances to improve health and extend life at all ages. Several lines of research have made the case that the enduring racial inequalities of past generations are still affecting the present-day black-white gap in adult mortality (Fogel 2005; Warner and Hayward 2006). Indeed, as Crimmins et al. (2004: 316) point out, "current prevalence of health problems is affected by a cohort's entire history of rates of disease onset, duration of conditions, and rates of survival." Thus, studies taking a "long view" have begun in earnest to link early childhood health, development, and subsequent attainment of socioeconomic resources to illustrate the long-term stratification processes driving racial and SES differences in health across the life course (Palloni 2006; Montez and Hayward 2010; Warner and Hayward 2006; Shuey and Wilson 2008).

In this chapter I added to this literature by showing that these stratification processes are inherently cohort-specific phenomena. Consistent with Finch and Crimmins's (2004) notion of a

"cohort morbidity phenotype," my findings indicate that the enduring effects of childhood conditions on adult mortality risk vary significantly across U.S. birth cohorts. These cohort processes were significantly conditioned by race, thus helping to perpetuate racial differences in both health and socioeconomic attainment across the life course.

In particular, I find evidence consistent with all five of my hypotheses, which can be summarized as follows. One, across the twentieth century U.S. black cohorts endured higher prevalence of harsh early-life conditions than U.S. white cohorts. Two, cohort measures of harsh living conditions during childhood were found to be positively and significantly associated with U.S. adult mortality, irrespective of race or gender, and even after controlling for the mediating effects of education and income in adulthood. Three, reductions in prevalence of harsh early-life conditions occurred significantly earlier in time for U.S. white birth cohorts than U.S. black birth cohorts. As such, the cumulative exposure time to deleterious childhood conditions has been and remains significantly higher in U.S. black cohorts than in U.S. white cohorts. Four, cohort reductions in U.S. adult mortality between 1986 and 2006 were significantly greater in the white population than the black population. Five, the educational and income gradients in U.S. adult mortality were found to be significantly stronger in the white population than in the black population. And six, the educational and income gradients in U.S. white mortality are growing significantly wider across cohorts, whereas the gradients are constant across U.S. black cohorts. Taken together, the evidence is consistent with my theoretical framing that variation in cohort changes in early-life conditions affect the long-term stratification of U.S. racial differences in childhood health, subsequent resource attainment, subsequent health, and ultimately mortality risk in adulthood.

The analyses in this chapter are not without limitation. First, mortality selection is incredibly high in the oldest U.S. birth cohorts, and this is especially the case in the NHIS-LMF data. A healthy participant effect is most likely strong in the NHIS, and links to death records at the NDI are difficult to make in the U.S. black male and female populations at the oldest ages (Preston, Elo, Rosenwaike and Hill 1996). I am comforted by the fact, however, that the cohort patterns in the results are not concentrated in a select few birth cohorts, but rather are observed across all the data. Also, because I am not using a continuous measure of birth cohorts, the cohort effects cannot be extrapolated or driven by outliers or faulty data. Second, I unfortunately have only cohort-level measures of early-life conditions. Thus, I cannot actually link respondents' own living conditions in childhood to their subsequent attainment of education and income, nor to their health and mortality risk in adulthood. Third, I do not have early-life conditions for the education-specific subsamples. That is, measures of cohort early-life conditions are shared across education and income levels. It would be helpful to have the early-life conditions for each level of educational attainment and income to better link the ways that early-life conditions produce disparate paths of subsequent socioeconomic attainment for U.S. black and white cohorts.

While the results of these analyses should be interpreted within the context of these limitations, the findings add to a growing literature linking early-life conditions and racial differences in disparate trajectories of socioeconomic attainment, adult health, and adult mortality in the United States.

**Table 4.1: Means of non-Hispanic White and Black, Male and Female NHIS-LMF 1986-2006 Samples**

| | Women | | | | Men | | | |
|---|---|---|---|---|---|---|---|---|
| | Black | | White | | Black | | White | |
| | Mean | St. Dev. | Mean | St. Dev. | Mean | St. Dev. | Mean | St. Dev. |
| **Person-level Sample** | | | | | | | | |
| Age | 45.99 | 15.1 | 48.08 | 15.7 | 46.18 | 14.8 | 47.16 | 15.0 |
| Year | 1993.97 | 5.3 | 1993.77 | 5.2 | 1994.11 | 5.4 | 1993.85 | 5.2 |
| Birth Year | 1947.98 | 16.0 | 1945.69 | 16.5 | 1947.93 | 15.7 | 1946.69 | 15.7 |
| % Deceased | 14.78 | 35.5 | 13.87 | 34.6 | 20.23 | 40.2 | 16.28 | 36.9 |
| *Cohort Childhood Indicators* | | | | | | | | |
| Born in South | 72.97 | 11.0 | 29.44 | 2.7 | 73.08 | 10.9 | 29.40 | 2.7 |
| Born on Farm | 29.82 | 18.0 | 19.09 | 9.4 | 30.10 | 18.0 | 18.67 | 9.2 |
| Born into Large Household | 58.06 | 6.5 | 34.91 | 5.6 | 58.13 | 6.4 | 34.67 | 5.5 |
| Infant Mortality Rate | 68.88 | 43.0 | 40.80 | 25.4 | 86.48 | 50.6 | 50.48 | 29.24 |
| *Adult Socioeconomic Indicators* | | | | | | | | |
| <HS | 27.31 | 44.6 | 14.48 | 35.2 | 28.99 | 45.4 | 14.96 | 35.7 |
| HS | 36.84 | 48.2 | 39.11 | 48.8 | 37.14 | 48.3 | 34.53 | 47.5 |
| >HS | 35.86 | 48.0 | 46.41 | 49.9 | 33.86 | 47.3 | 50.52 | 50.0 |
| >$45,000 Income | 17.94 | 38.4 | 37.35 | 48.4 | 24.96 | 43.3 | 42.09 | 49.4 |
| Mid-Income | 53.41 | 49.9 | 55.30 | 49.7 | 57.91 | 49.4 | 53.10 | 49.9 |
| In Poverty | 28.65 | 45.2 | 7.35 | 26.1 | 17.13 | 37.7 | 4.81 | 21.4 |
| N | 60,846 | | 335,573 | | 42,423 | | 306,287 | |

**Table 4.2: HAPC-CCREM Results of Men's Adult Mortality with Cohort-level Childhood Covariates**

| | APC | | South | | Household | | Infant Mortality[2] | |
|---|---|---|---|---|---|---|---|---|
| | White | Black | White | Black | White | Black | White | Black |
| *Covariance Parameter* | | | | | | | | |
| Cohort | .637 | .088 | .410 | .002 | .052 | .003 | .016 | .008 |
| Period | .078 | .003 | .041 | .002 | .051 | .002 | .048 | .003 |
| *Childhood Conditions* [1] | | | | | | | | |
| % Born in South | | | .365 *** | .316 *** | -.206 | .300 *** | -.059 | .316 * |
| % Born on Farm | | | | | .554 *** | .032 | .219 *** | .036 |
| % Born in Large Family | | | | | .294 | .013 | .278 *** | .023 |
| Infant Mortality Rate | | | | | | | .278 *** | .022 |
| Intercept | -4.152 | -3.529 | -4.194 | -3.513 | -4.068 | -3.538 | -3.991 | -3.551 |
| Deviance | 1230.2 | 992.9 | 1230.3 | 982.3 | 1228.7 | 982.9 | 1228.6 | 982.1 |

1 All Measures Centered on Grand Mean and Standardized by Standard Deviation

2 In a reduced HAPC model of black men's mortality, % born on farm and IMR were significant at the .001 α-level.

* p<.05 ** p<.01 *** p<.001 using 1-tailed tests

**Table 4.3: HAPC-CCREM Results of Women's Adult Mortality with Cohort-level Childhood Covariates**

| | APC | | South | | Household | | Infant Mortality | |
|---|---|---|---|---|---|---|---|---|
| | White | Black | White | Black | White | Black | White | Black |
| *Covariance Parameter* | | | | | | | | |
| Cohort | .671 | .140 | .369 | .005 | .041 | .009 | .023 | .005 |
| Period | .180 | .013 | .082 | .022 | .127 | .039 | .095 | .045 |
| *Childhood Conditions* [1] | | | | | | | | |
| % Born in South | | | .364 *** | .358 *** | -.120 * | .463 ** | -.068 | .034 |
| % Born on Farm | | | | | .674 *** | -.043 | .381 *** | .284 ** |
| % Born in Large Family | | | | | .100 | -.022 | .139 | .028 |
| Infant Mortality Rate | | | | | | | .268 *** | .214 *** |
| Intercept | -4.675 | -4.055 | -4.570 | -4.049 | -4.519 | -4.044 | -4.613 | -4.064 |
| Deviance | 1213.1 | 1001.6 | 1213.5 | 991.9 | 1211.2 | 989.3 | 1211.1 | 985.1 |

1 All Measures Centered on Grand Mean and Standardized by Standard Deviation

* p<.05 ** p<.01 *** p<.001 using 1-tailed tests

**Table 4.4: HAPC-CCREM Results of Men's Adult Mortality with Cohort-level Childhood Covariates and Adult SES**

| | Education | | Income | | Edu-Inc[3] | | Edu-Early | | Early-Edu-Inc | |
|---|---|---|---|---|---|---|---|---|---|---|
| | White | Black | White | Black | White | Black | White | Black | White | Black |
| *Covariance Parameter* | | | | | | | | | | |
| Cohort | | | | | | | | | | |
| <HS Cohort | .107 | .019 | | | .161 | .005 | .012 | .003 | .019 | .002 |
| HS Cohort | .300 | .034 | | | .373 | .181 | .034 | .042 | .087 | .020 |
| >HS Cohort | .375 | .071 | | | .302 | .028 | .028 | .046 | .038 | .016 |
| Poverty Cohort | | | .344 | .042 | | | | | | |
| "Middle" Income Cohort | | | .560 | .020 | | | | | | |
| >$45K Income Cohort | | | .346 | .074 | | | | | | |
| Period | .026 | .002 | .126 | .002 | .037 | .002 | .084 | .004 | .104 | .005 |
| *Childhood Conditions* [1] | | | | | | | | | | |
| % Born in South | | | | | | | | .269 *** | | .237 *** |
| % Born on Farm | | | | | | | .163 * | | .214 *** | |
| % Born in Large Family | | | | | | | .230 *** | | .108 *** | |
| Infant Mortality Rate | | | | | | | .267 *** | | .212 *** | |
| *Adult Socioeconomic Resources* [2] | | | | | | | | | | |
| <HS | 1.062 *** | .661 ** | | | .913 * | .612 ** | .863 *** | .622 * | .728 ** | .516 ** |
| HS | .294 | .452 ** | | | .173 | .121 | .544 * | .597 *** | .135 | .411 |
| <= Poverty Line | | | 1.562 ** | .479 * | .382 *** | .322 *** | | | .383 *** | .317 *** |
| >$45,000 Income | | | -.177 | -.350 | -.382 *** | -.440 *** | | | -.384 *** | -.439 *** |
| Intercept | -4.490 | -4.076 | -4.671 | -3.726 | -4.182 | -3.805 | -4.334 | -4.099 | -4.157 | -3.883 |
| Deviance | 3060.5 | 2180.5 | 2882.6 | 2155.7 | 6993.3 | 4540.9 | 3037.0 | 2165.2 | 6966.1 | 4525.9 |

1 All Measures Centered on Grand Mean and Standardized by Standard Deviation

2 Reference is *>HS* and *Poverty<Income<$45,000* at age-group *65-69*

3 Income included only as level-1 covariate

* p<.05 ** p<.01 *** p<.001 using 1-tailed tests

**Table 4.5: HAPC-CCREM Results of Women's Adult Mortality with Cohort-level Childhood Covariates and Adult SES**

| | Education | | Income | | Edu-Inc[3] | | Early-Edu | | Early-Edu-Inc | |
|---|---|---|---|---|---|---|---|---|---|---|
| | White | Black | White | Black | White | Black | White | Black | White | Black |
| *Covariance Parameter* | | | | | | | | | | |
| Cohort | | | | | | | | | | |
| <HS Cohort | .028 | .002 | | | .072 | .012 | .023 | .005 | .022 | .004 |
| HS Cohort | .360 | .023 | | | .293 | .187 | .043 | .017 | .166 | .022 |
| >HS Cohort | .489 | .049 | | | .233 | .052 | .037 | .027 | .083 | .015 |
| Poverty Cohort | | | .045 | .036 | | | | | | |
| "Middle" Income Cohort | | | .369 | .078 | | | | | | |
| >$45K Income Cohort | | | .534 | .107 | | | | | | |
| Period | .093 | .020 | .066 | .014 | .069 | .022 | .206 | .084 | .384 | .072 |
| *Childhood Conditions* [1] | | | | | | | | | | |
| % Born in South | | | | | | | -.034 | .110 | .077 | .087 |
| % Born on Farm | | | | | | | .375 *** | .242 ** | .424 *** | .208 |
| % Born in Large Family | | | | | | | .062 | .019 | -.109 ** | .007 |
| Infant Mortality Rate | | | | | | | .250 *** | .211 *** | .132 *** | .170 ** |
| *Adult Socioeconomic Resources* [2] | | | | | | | | | | |
| <HS | 1.269 * | .275 | | | 1.092 * | .410 | 1.206 *** | .649 ** | 1.273 *** | .318 |
| HS | .553 | .018 | | | .281 *** | .176 | .662 ** | .523 ** | .526 *** | .243 |
| <= Poverty Line | | | 1.220 *** | .237 | .313 *** | .280 *** | | | .314 *** | .281 *** |
| >$45,000 Income | | | -.540 | -.448 | -.277 *** | -.328 *** | | | -.275 *** | -.322 *** |
| Intercept | -5.018 | -3.993 | -4.872 | -4.282 | -4.865 | -4.322 | -5.005 | -4.524 | -4.844 | -4.308 |
| Deviance | 2975.3 | 2266.0 | 2841.8 | 2145.5 | 7013.9 | 4661.1 | 2945.5 | 2233.8 | 6961.1 | 4635.5 |

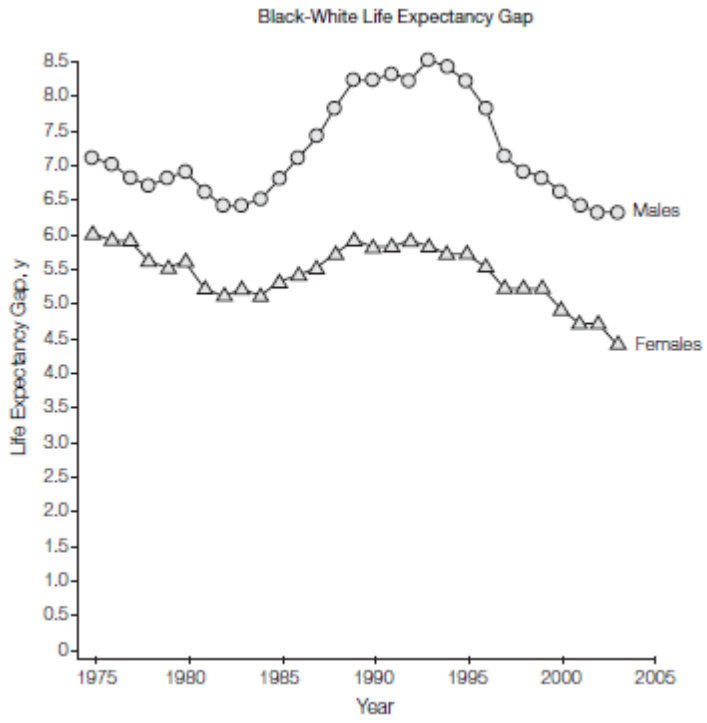
1 All Measures Centered on Grand Mean and Standardized by Standard Deviation

2 Reference is *>HS* and *Poverty<Income<$45,000* at age-group *65-69*

3 Income included only as level-1 covariate

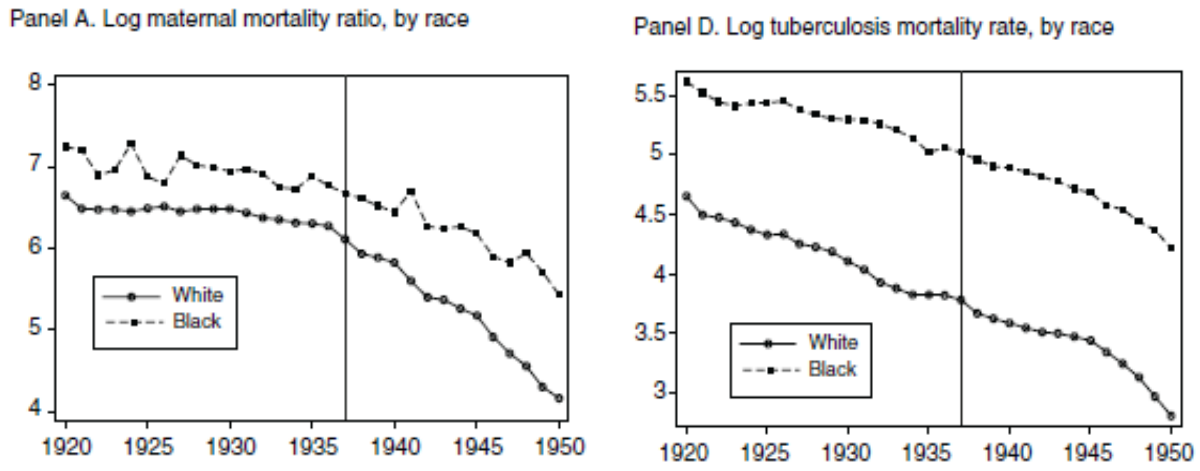
* p<.05 ** p<.01 *** p<.001 using 1-tailed tests

**Figure 4.1. U.S. Black-White Life Expectancy Gap, 1975-2005**



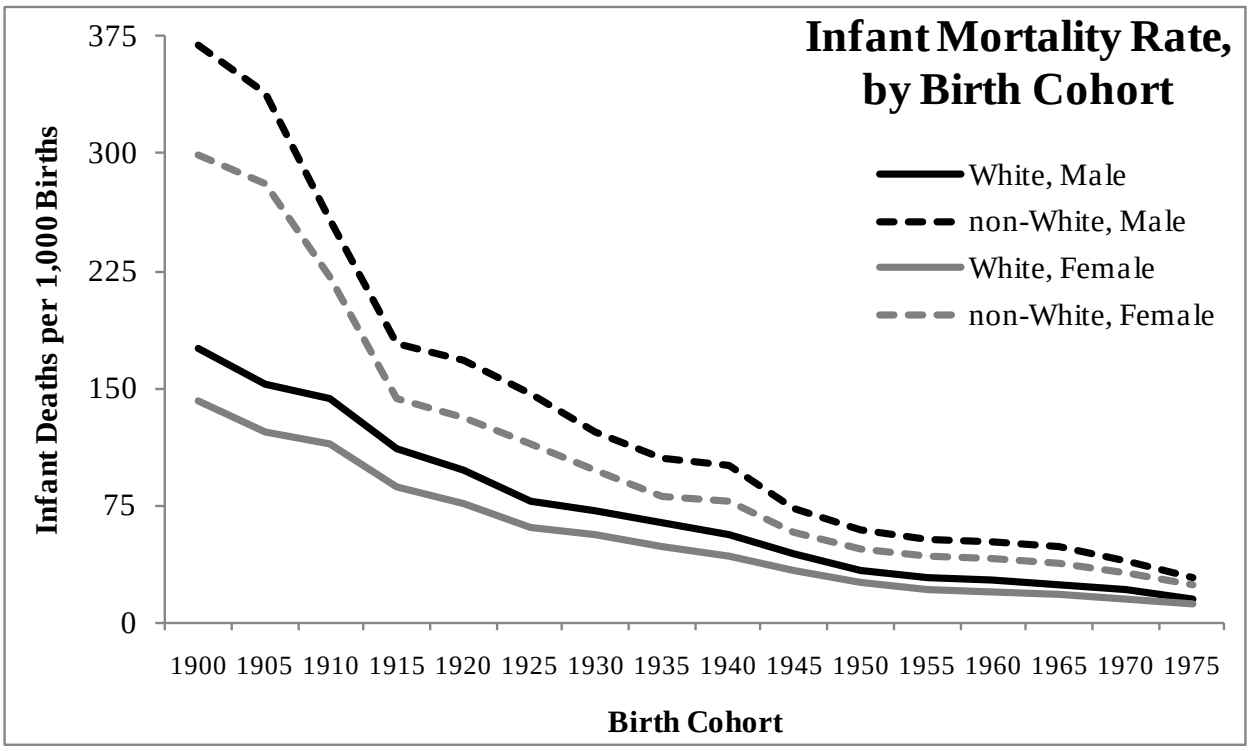

Source: Harper et al. 2007, Figure 1

**Figure 4.2. U.S. Black-White Gaps in Maternal Mortality and Tuberculosis Mortality, 1920-1950**



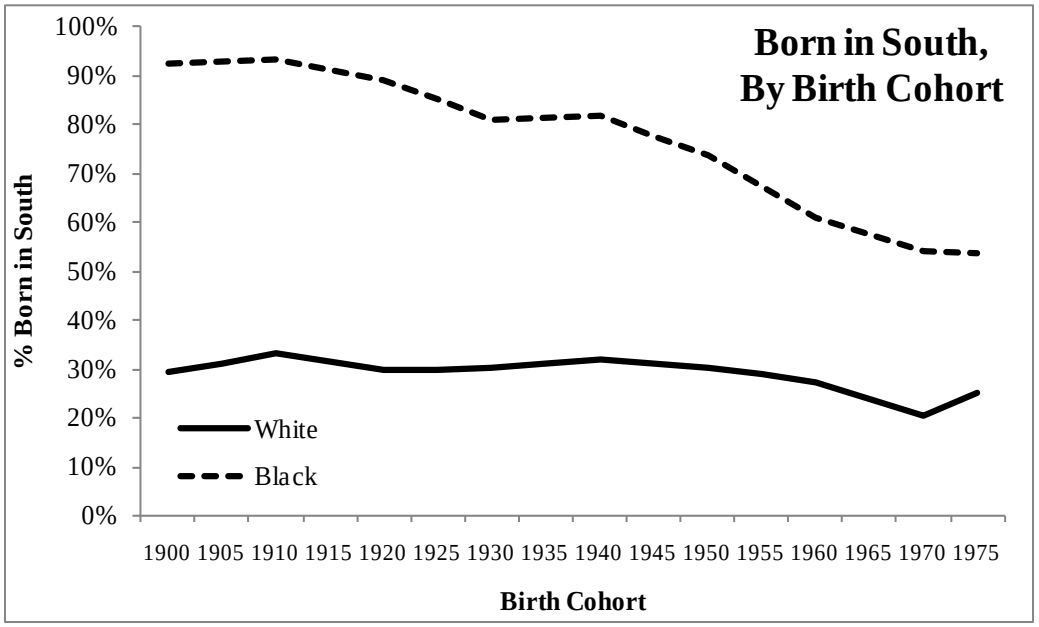

Source: Jayachandran et al. 2010, Figure 6

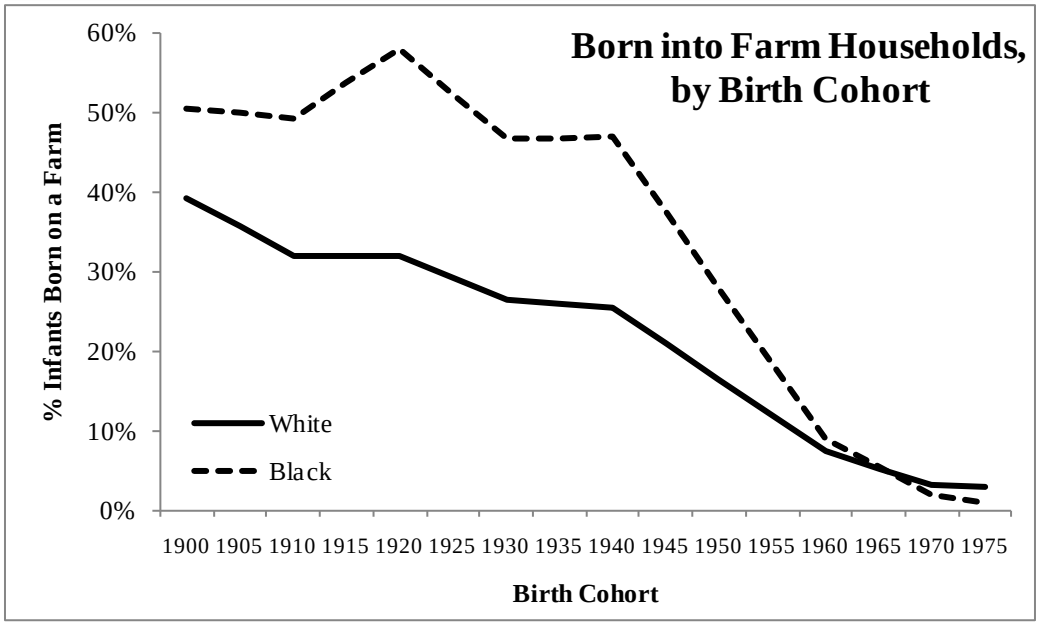
**Figure 4.3. U.S. White and Non-White Infant Mortality Rates by Birth Cohort.**



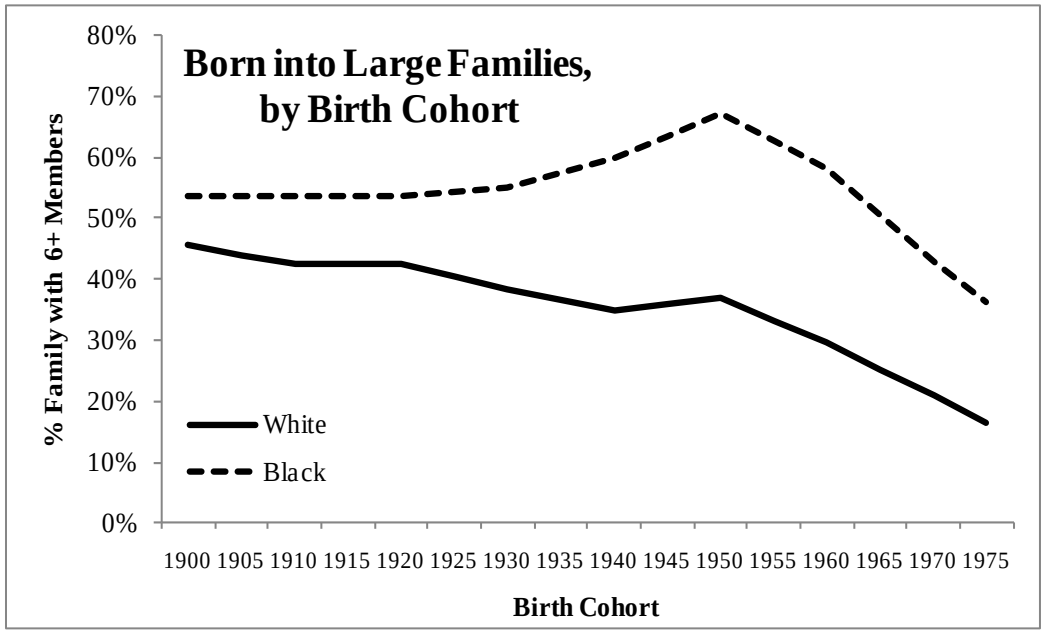

Source: National Vital Statistics System's Historical Mortality, HIST209.

**Figures 4.4 & 4.5. U.S. Black and White Prevalence of Infants born in Southern States and on a Farm**



**Figures 4.6 & 4.7. U.S. Black and White Prevalence of Infants born in Large Household and Prevalence of Widowed Women Aged 30-39 by Period.**



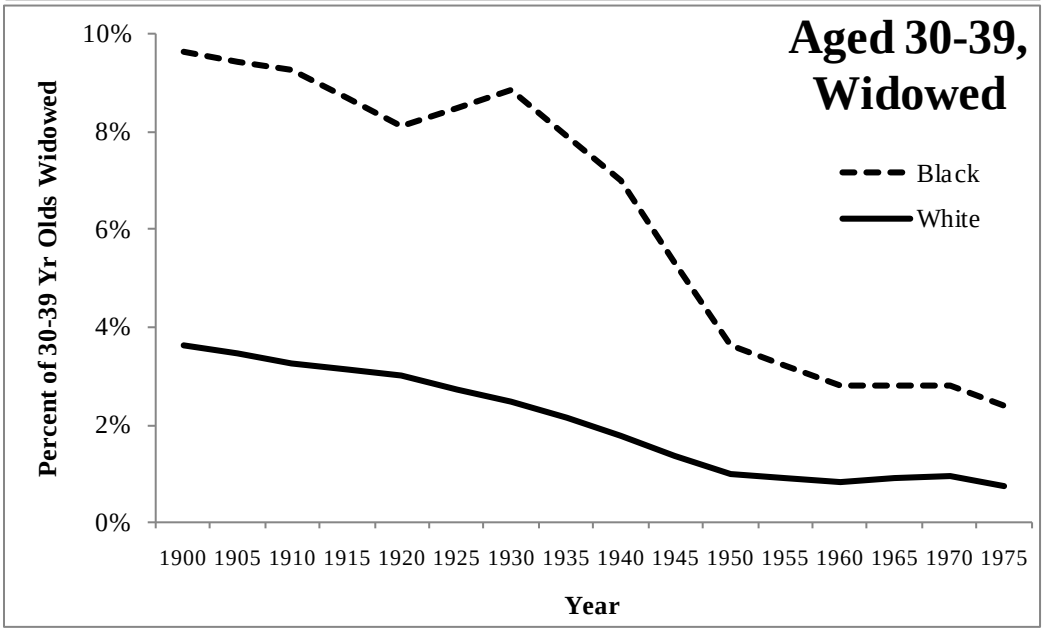
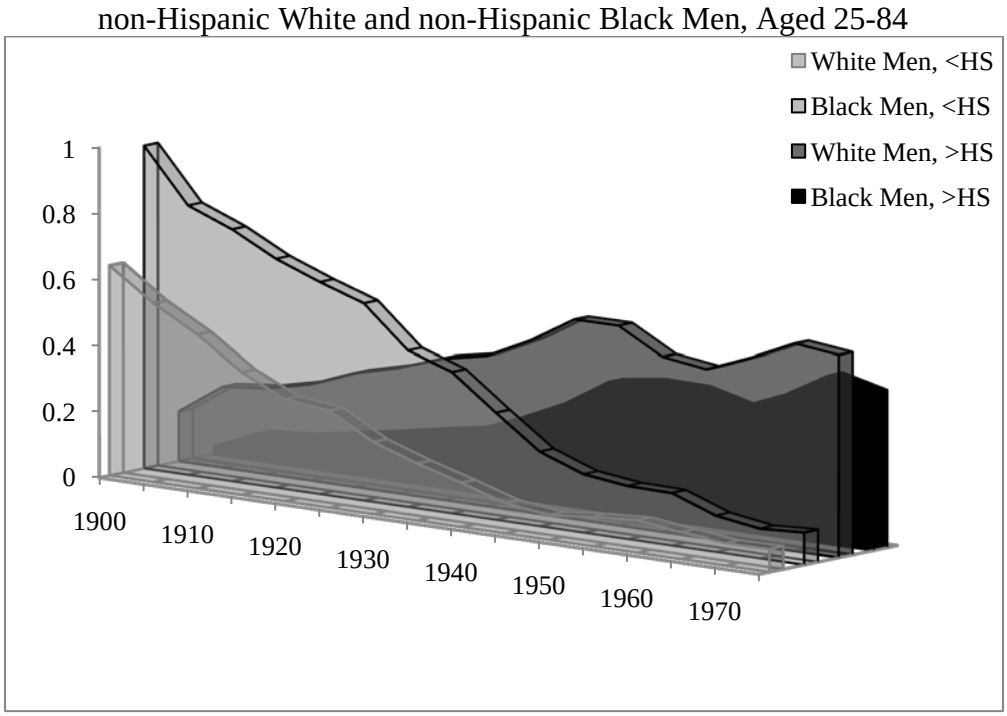



**Figure 4.8. Distributions of Educational Attainment across Cohort, NHIS-LMF 1986-2004.**



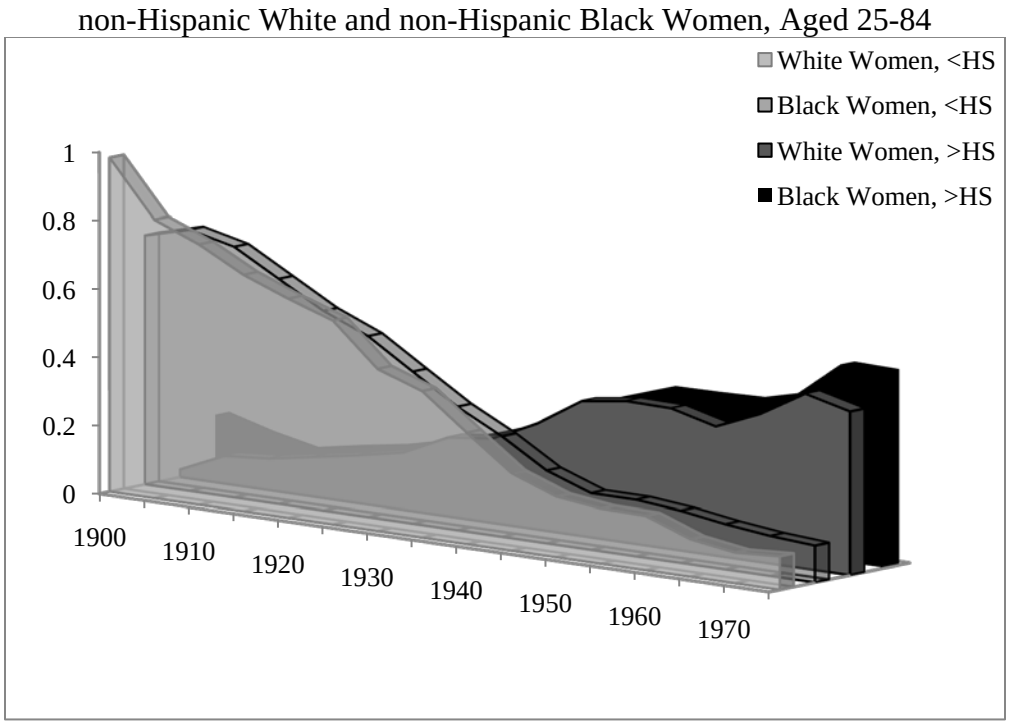
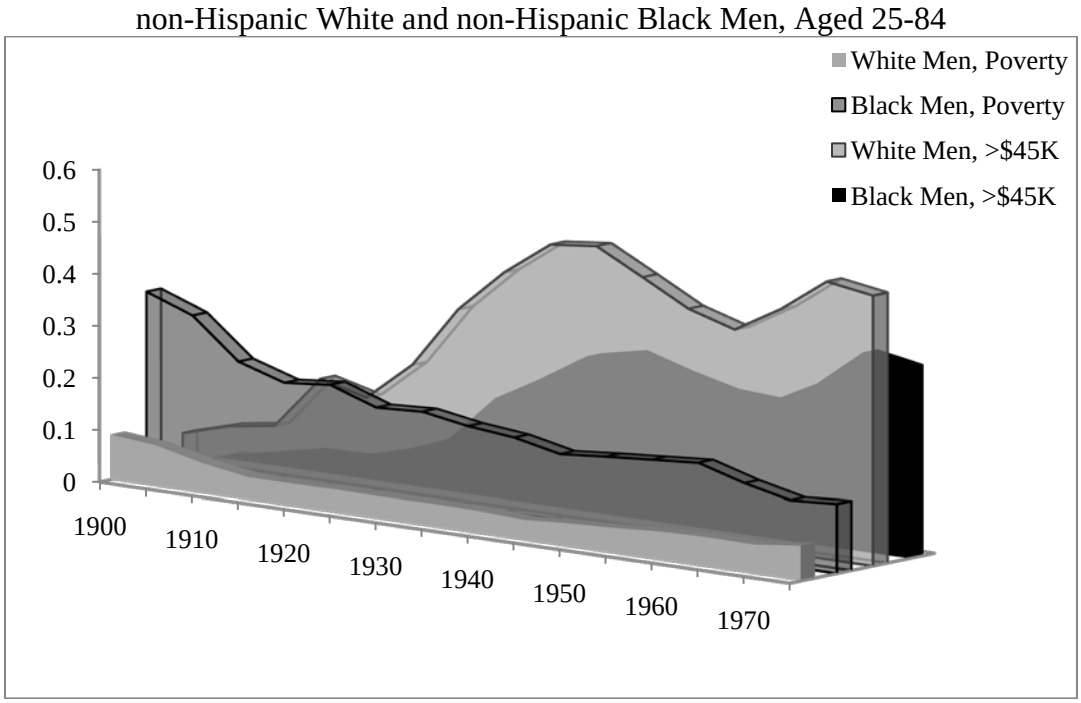



**Figure 4.9. Distributions of Income across Cohort, NHIS-LMF 1986-2004.**



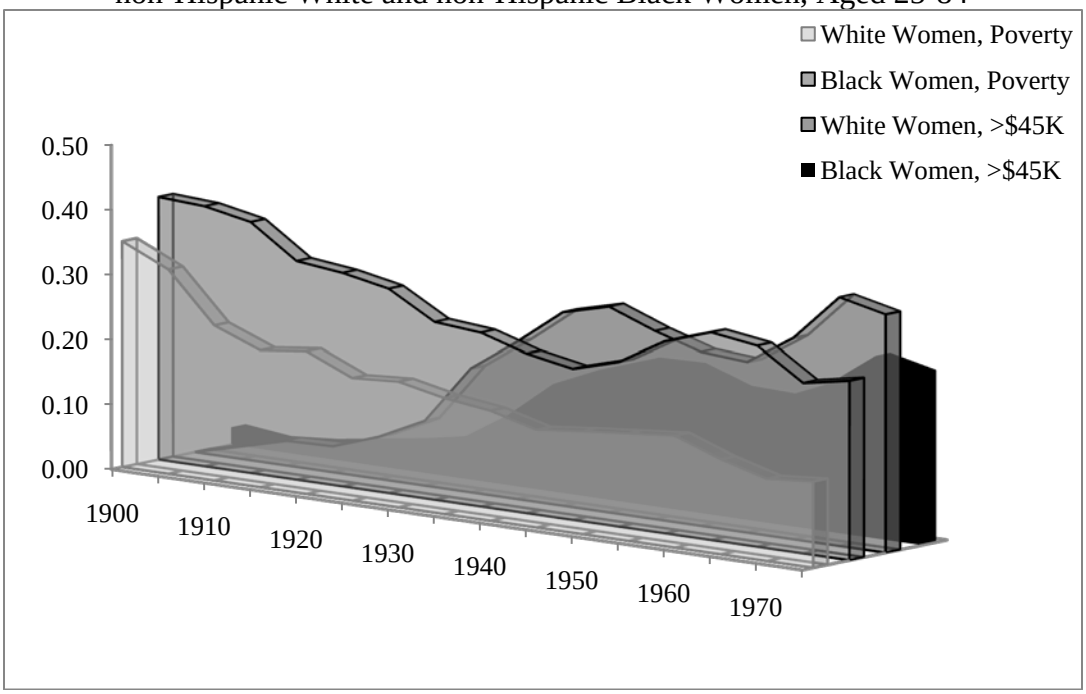



**Figure 4.10. U.S. Black and White Men's and Women's Fitted Adult Logged Mortality Rates Across Birth Cohorts, NHIS-LMF 1986-2006.**

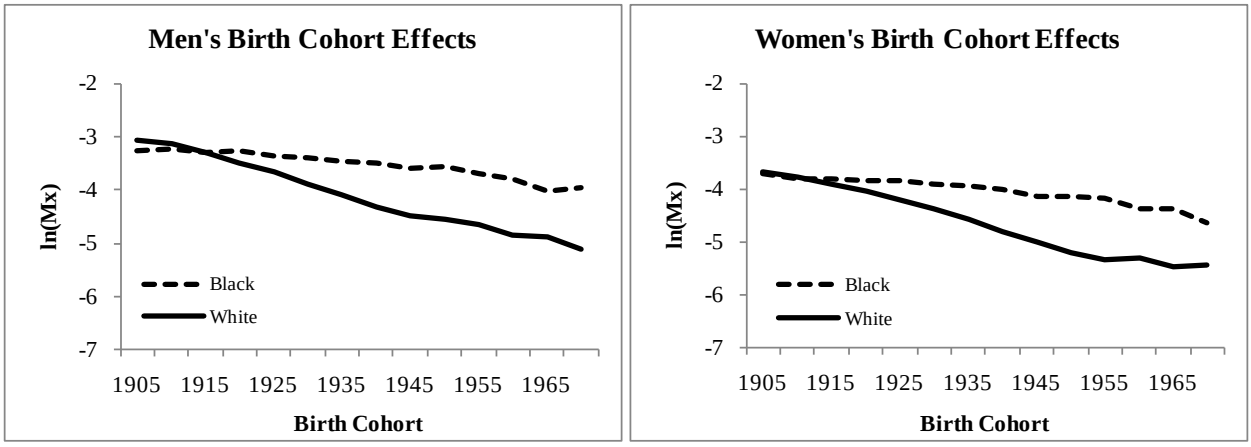


**Figure 4.11. Racial Differences in Education's Effect on U.S. Black and White Men's Adult Mortality, Age and Cohort, NHIS-LMF 1986-2006.**

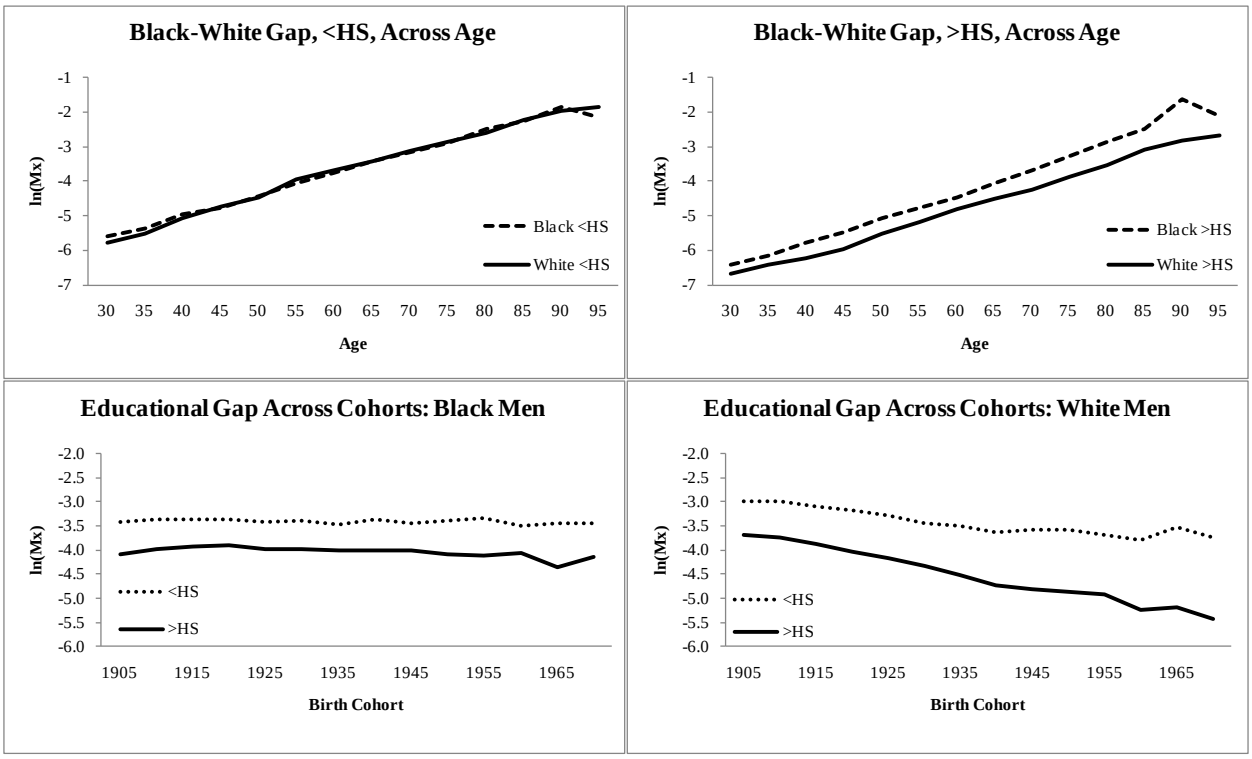


**Figure 4.12. Racial Differences in Education's Effect on U.S. Black and White Women's Adult Mortality, Age and Cohort, NHIS-LMF 1986-2006.**

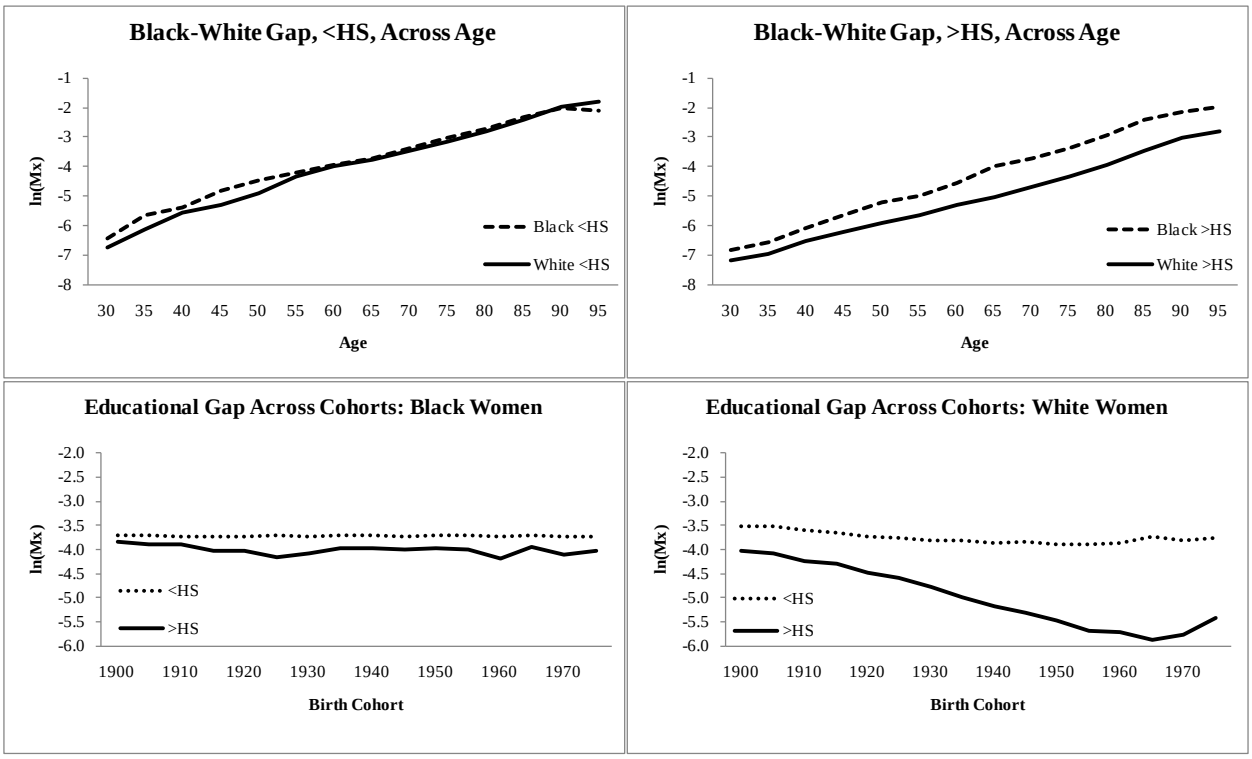


**Figure 4.13. Racial Differences in Income's Effect on U.S. Black and White Women's Adult Mortality, Age and Cohort, NHIS-LMF 1986-2006**

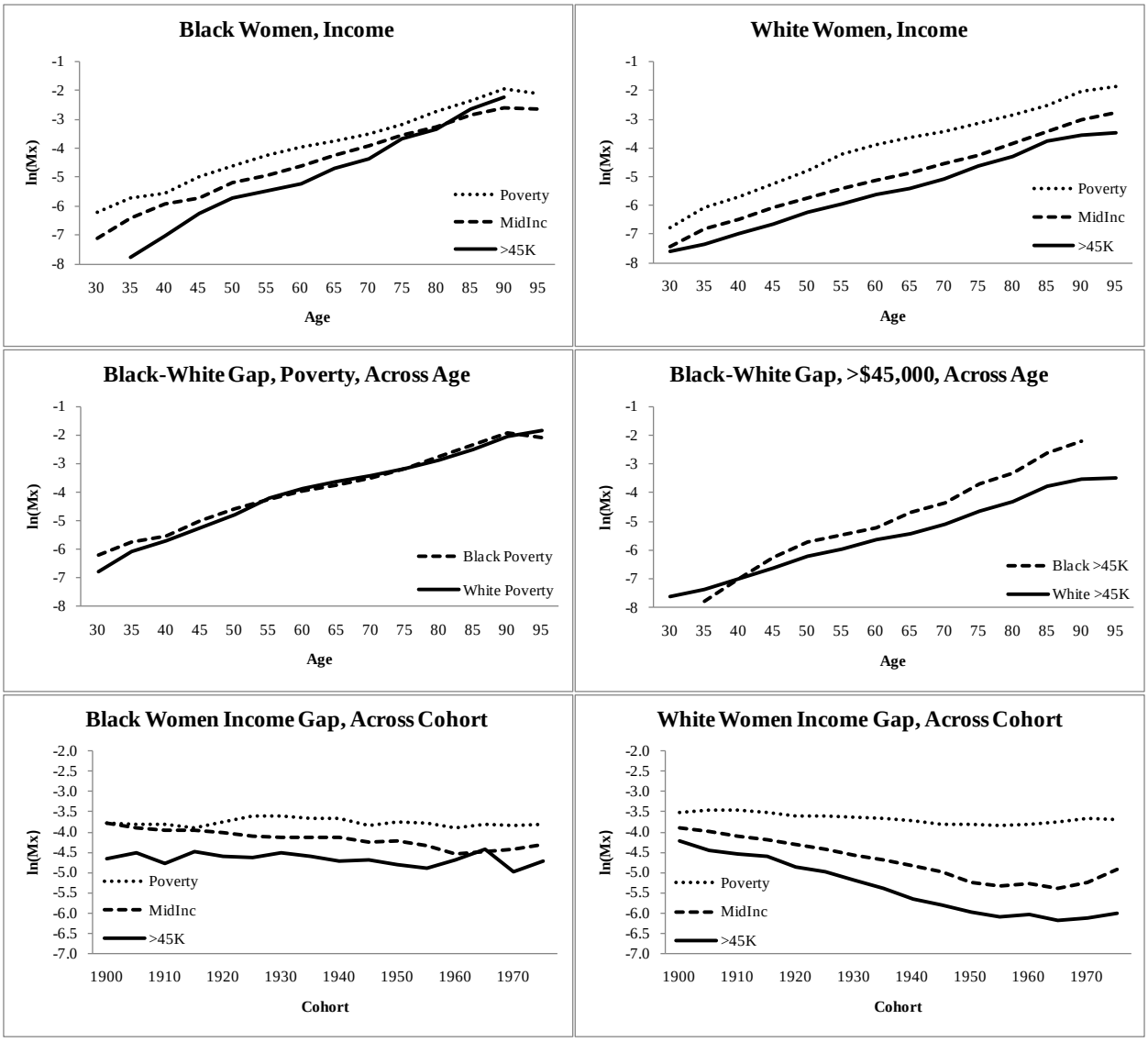
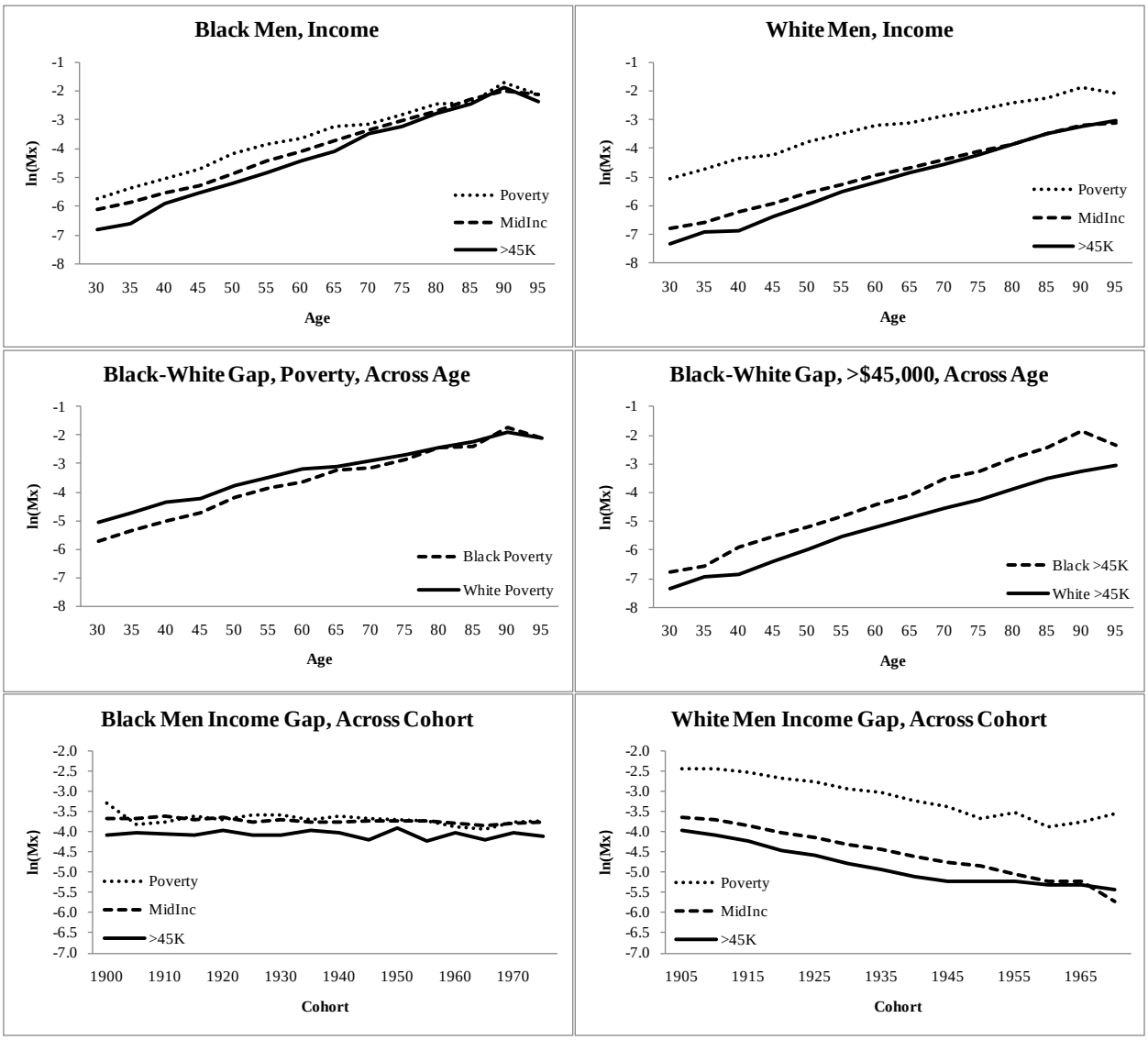


**Figure 4.14. Racial Differences in Income's Effect on U.S. Black and White Men's Adult Mortality, Age and Cohort, NHIS-LMF 1986-2006**



# CHAPTER 5

## Conclusions and Future Work

### 5.1 Review

Over the twentieth century, tremendous achievements were made to improve health and reduce early death in the United States. These achievements have continued into the twenty-first century as well, but understanding the factors behind the improved health and increased life expectancy remains limited. As I show in each chapter of my dissertation, the predominant approach to understanding mortality trends is limited in two important ways. First, a period framework is most often employed to analyze trends in health and mortality. That is, mortality risk is generally assessed at one time period and compared to a past time period, with little attention given to the cohort composition of the populations at those respective times. As a result, improvements in health outcomes or survival are often misattributed to the health inputs *in* that period. Second, the life course perspective is increasingly playing a greater role in our understanding of aging, health, and mortality. On the one hand, this practice is to be celebrated and extended. On the other hand, the life course perspective, when applied to health, is often times treated as a constant. That is, research often fails to recognize that "the life course is not fixed, but widely flexible" (Riley 1978: 39) and, thus, needs to be analyzed as being embedded in the sociohistorical context in which it unfolds (Ben-Shlomo and Kuh 2002).

This dissertation aimed to address these limitations and grew out of my interest in cohort differences in the ways educational attainment and other factors in adulthood affect U.S. adult mortality risk. In this research I combined tenets of the theory of "technophysio evolution" (Fogel and Costa 1997), the concept of a "cohort morbidity phenotype" (Finch and Crimmins

2005), and the "social shaping" of health (Link 2008) to highlight the ways by which educational attainment has grown increasingly associated with cohorts' health and mortality risk in the United States. As this research developed, it became increasingly clear to me that *exposure time* (to poor living conditions, infectious diseases, and poor nutrition in childhood; and stressors, health risk factors, and health-enhancing/protecting technologies in adulthood) needs to hold a central place in our analyses of adult and older-age health and mortality risk. That is, we need to conceive of cohort-specific life courses, being mindful of the disparate exposure times that different cohorts had to poor living conditions, acute and chronic infections, and advances in healthcare, nutrition, medical technologies and other significant factors that shape mortality risk across the life course. Consistent with this theoretical approach, evidence from my analyses support the following findings:

*Chapter 2*

Estimates of age-specific mortality rates reflect both age and cohort processes. Once I disentangle cohort, period, and age effects the fitted average age-specific mortality rates of U.S. black and white respondents do not cross at any age. Thus, the black-white crossover is a cohort-specific phenomenon.

*Chapter 3*

The adult environment is growing more important at shaping adult mortality, thus socioeconomic resources such as education are becoming more associated with adult mortality risk. This is inherently a cohort process, reflecting cumulative exposure time to early-life conditions and disparate "cohort morbidity phenotypes," exposure time to lifestyle risk factors (e.g., smoking, diet, sedentary lifestyle), and exposure time to

beneficial knowledge and technologies. As such, I found: (1) the educational gap in U.S. adult mortality risk is growing, (2) the educational gap is growing across cohorts, not periods, (3) The educational gap in U.S. adult mortality is growing more rapidly for the non-Hispanic white population than the non-Hispanic black population, and (4) the educational gap is growing more rapidly across cohorts for causes of death that are under greater degrees of human control (e.g., heart disease and lung cancer compared to "unpreventable" cancers).

*Chapter 4*

Racial disparities in U.S. adult mortality risk persisted across the time period 1986-2006, and these disparities result from more than the unequal distributions of socioeconomic resources in adulthood. One the one hand, U.S. black cohorts endured higher rates of exposure to deleterious childhood conditions, the effects which continue to shape adult mortality risk beyond individuals' socioeconomic resources in adulthood. On the other hand, the U.S. white population derives greater survival benefits from adult socioeconomic resources than the U.S. black population. Furthermore, the educational and income gradients in U.S. adult mortality are growing across U.S. white cohorts, while the gradients are stagnant across U.S. black cohorts.

## 5.2 Limitations

The analyses in this dissertation are not without limitations, some of which might severely alter or even drive the results. To start with, there are a number of concerns when relying on only one dataset (NHIS-LMF) and only one outcome (adult mortality) to infer

population-level patterns and trends. Regarding the dataset, the NHIS-LMF suffers from a number of selection issues. First, the data are representative only of the noninstitutionalized U.S. population. Respondents residing in assisted-living facilities, enlisted in the military, imprisoned, or otherwise institutionalized at the time of the NHIS survey are excluded from the sampling frame. If the likelihood of exclusion is associated with one's birth cohort, age, race/ethnicity, education level, or any other significant variable of interest, then the sampling selection could alter the association between said variable and mortality risk. Even more of a concern, if one's likelihood of death is associated with one's chance of inclusion in the NHIS-lMF, then bias is surely included in the data. That is, selecting on the outcome is even more problematic than selecting on covariates. I am most certain that the NHIS suffers from a "healthy participant effect, in that only those U.S. residents of adequate health to both live in a noninstitutionalized setting and answer the survey are included in the sample (Mendes 2007).

Secondly, the matching of the NHIS to the NDI increasingly becomes select across survey waves. That is, for survey waves 1986 through the early 1990s, the matching rate to the NDI is remarkably high. Beginning with the survey changes in 1997, however, the matching rate falls to as low as 85 percent (NCHS 2010). This increasingly select matching process also selects on the outcome, invariably prompting an association between period (i.e., survey wave) and mortality risk. Both of these selection effects most likely differ by educational attainment and race/ethnicity. Lastly, mortality selection in general is evident in the NHIS-LMF 1986-2006 data. That is, different mortality schedules across the life course, irrespective of their representation in the NHIS-LMF, is occurring in the U.S. population. Indeed, this cohort change is at the heart of my dissertation and I specifically theorized about the effects of racial differences in mortality selection in Chapter One. Nevertheless, I must concede that results pertaining to cohort patterns

of mortality in the NHIS-LMF inherently reflect the mortality risk of those cohort members that have survived long enough to be included in the NHIS. For example, what is it to model the mortality rates of the 1910 birth cohort from ages 72 to 96 for the U.S. black population when only a fraction of this cohort is alive at these ages? Furthermore, what is it to contrast racial differences in this cohort's mortality rates across age and time when the survival rate of the cohort significantly differs by race? Indeed, if, say, 25 percent of this cohort's white population is alive at age 75, but only 15 percent of this cohort's black population is alive at age 75, what are we to make of survivability from that age to 96? In other words, this is a classic prevalence/incidence problem.

To further illustrate this issue, I revisit the hypothesis I made in Chapter One. There, I argued that at the oldest-old ages only the most robust members of birth cohorts, irrespective of race, could have survived. Thus, the black-white crossover in mortality rates at these ages reflects cohort processes. Central to this argument is the frailty composition of these cohorts. To assess frailty of cohorts in the NHIS-LMF I used a crude measure as described by Oeppen (2011): frailty is simply the difference between the observed number of deaths in a given group and the expected number of deaths in that group as specified by a relevant mortality schedule. While Oeppen's groups referred to households, we can easily extend his logic to consider cohorts as distinct groups, and thus calculate cohort-specific frailties. To assess cohort-level frailty of white and black men in the NHIS-LMF 1986-2006 I used the 1985, 1995, and 2000 U.S. men's mortality rates as my reference mortality schedules (Human Mortality Database). While crude, the HMDB life tables provide reasonable benchmarks of age-specific mortality rates for the U.S. male population between the years 1986 and 2006. For time periods 1986 to 1989 the 1985 HMDB $M_x$ is applied, for periods 1990 to 1999 the 1995 HMDB $M_x$ is applied,

and for periods 2000 to 2006 the 2000 HMDB $M_x$ is applied. I calculated the expected number of deaths per five-year cohort in the white and black men's NHIS-LMF sample as a function of the period-specific $M_x$ provided in HMDB life table, the average age of the cohort cell per period, and the cumulative exposure time lived in each cell. To illustrate, here are data for the1930 birth cohort of U.S. white men in the NHIS-LMF at time period 1998:

| Obs | coh5yr | period | entry | n | expose | tdead | expect | frailty | avgage |
|---|---|---|---|---|---|---|---|---|---|
| 226 | 7 | 1998 | 0 | 16713 | 16547.0 | 332 | 435.682 | -0.23798 | 65.9966 |

To calculate the expected number of deaths (435.682), I applied the HMDB death rate of U.S. men aged 65-70 in 1995 (.02633; not shown in data) to the exposure time lived by the 1930 birth cohort in time period 1998 (16547.0). Because the observed number of deaths of this birth cohort at year 1998 was only 332, the estimated frailty of this birth cohort at this time is (332-435.682)/435.682 = -.238. Estimated mean levels of "robustness" for U.S. white and black men's birth cohorts in the NHIS-LMF 1986-2006 are displayed in Figure 5.1. The degree of robustness was derived by multiplying the level of frailty by -1.

In the top row we observe trend in the level of U.S. white and black men's cohort-level robustness. Because both samples are compared to the same HMBD benchmarks, there are large racial differences in the distributions. For white men, we observe high degrees of robustness in those cohorts making up the oldest-old age groups, which is consistent with mortality selection in the NHIS-LMF. Indeed, across subsequent cohorts the level of robustness drops into negative values and then rapidly increases across more recent birth cohorts. For black men, we also observe high degrees of robustness in the 1900 and 1905 cohorts. Indeed, the degree of

robustness in these birth cohorts is higher in the black sample than in the white sample. In subsequent birth cohorts, however, negative values of robustness (or strong indication of excess mortality) are estimated, indicating rapidly increasing frailty across U.S. black birth cohorts. These racial differences in U.S. birth cohort frailty are entirely consistent with my theoretical framing in Chapter One and with past research indicating that heterogeneity is increasing across U.S. black birth cohorts (Lynch et al. 2003). These patterns of cohort frailty, however, are confounded by at least two processes that these descriptive estimates fail to condition on: age composition and a "healthy participant effect" (Mendes 2007). First, the black and white cohort patterns are entirely consistent with mortality selection across the life course. For white men, the degree of robustness decreases across the life course as the population ages and becomes frailer. Some mortality selection is evident as the level of robustness upticks to high levels for cohorts whose members are in the oldest-old age groups (i.e., 1900 and 1905). For black men, we see the greatest difference in black-white mortality in cohorts whose members are in late-middle age-groups (i.e., 1935 to 1955), with mortality selection of cohorts' frailest members increasing the robustness of cohorts as we go back in time across birth cohorts. That is, from the 1940 birth cohort back to the 1900 birth cohort we observe a dramatic decrease in the frailty of the black male sample. Second, as seen in the bottom row of Figures 5.1 and 5.2, there is a sizable and significant difference in the degree of robustness between respondents who just entered the NHIS-LMF and respondents who have remained in the dataset after having been sampled in at an earlier time. That is, in the bottom row of Figure 5.1 we see that the degree of robustness of respondents who just entered the data is significantly higher than the degree of robustness of respondents who were already in the data. In short, the figures show a sizable sampling selection effect, irrespective of race.

To account for the confounding effects of sampling selection and age composition, I regress white and black men's frailty in the NHIS-LMF on the average age of cohort cells, a dummy variable indicating when the time period equals the survey year, and fixed effects of birth cohort. The average age variable is centered on age 52.1 in the black men's sample and age 53.2 in the white men's sample, and the reference category for the cohort measure is 1945-1949. Results of three OLS models, stratified by race, are presented in Table 5.1. In Model 1 I regress frailty only on age composition of cohort cells. A significant and sizable association is found in the white men's sample, but the association is not significant in the black men's sample. This finding is consistent with past research and my Chapter One theoretical framework. As cohorts of white men age across the life course they become frailer, whereas no such effect occurs in the black men's population because mortality selection is much more severe. In Model 2 I add a control for sampling selection and find strong and significant effects on frailty in both the white and black men's samples. Controlling for age composition, I find evidence suggesting that sampling selection is stronger in the black men's sample than the white men's sample (-.531 to -.239). This too is consistent with a greater degree of mortality selection in the black men's population than the white men's population. Lastly, in Model 3 I test for significant cohort trends in white and black men's frailty in the NHIS-LMF, controlling for age composition and sampling selection. For both the white and black male NHIS-LMF samples, I find significant evidence of cohort trends in frailty beyond age and sampling selection. To best illustrate the cohort trends in black and white men's robustness, negative values of the estimated cohort coefficients are plotted in Figure 5.2 below (i.e., I multiplied the coefficients by -1 to indicate robustness).

The cohort patterns of robustness in the black and white male samples of the NHIS-LMF are quite similar in the respect that I find strong evidence indicating that frailty is increasing

across U.S. birth cohorts in both the black and white male samples. This is what we'd expect once we control for aging processes and sampling selection. As mortality risk is reduced across time, a greater proportion of the population survives into adulthood, thereby reducing mortality selection and preserving a greater degree of heterogeneity across cohorts. These cohort changes are central to many issues in mortality research, a few of which were demonstrated in these chapters.

While I have just found evidence supporting the presence of mortality and sampling selection in the NHIS-LMF, I remain confident that the confounding effects of mortality on the APC patterns are minimal. This is because I am not extrapolating the cohort effects. I capture variation in cohort mortality by using dummy indicators of birth cohort, and I use cell weights to make respective weights indicative of representation in both the population and the sample. That is, the cell weights are a combination of individual-level proportional weights and cell-level frequency weights. As such, the weights combined with the dummy coding of a cohort isolate individual cohort effects to one cohort. Thus, while it may be the case that cohort effects at the tails (e.g., older cohorts 1900-1910 and more recent cohorts 1960-1975) are substantially affected by selection effects (mortality, healthy participant, educational attainment, etc.), the estimated cohort effects for birth cohorts in the middle of the sample range are not skewed by these cohorts' effects.

Lastly, another limitation of the current analyses is their omission of several mechanisms and confounders. Testing individual-, group, and cohort-level mechanisms is necessary for understanding changing health and mortality processes in the U.S. population. The measures I use to represent early-life conditions and adult socioeconomic resources are severely limited, and I fail to include any measures of lifestyle behaviors, familial or social support, intergenerational

processes of health and/or status transmission, disablement processes, and a number of other indicators that are repeatedly found to be associated with adult mortality risk in the U.S. population. Future research must address these shortcomings.

### 5.3 Agenda

My future work will primarily build off my dissertation, as I continue to examine the sociohistorical factors that shape health and mortality trends in U.S. populations. Expanding this work is incredibly important for illustrating how health-related inequalities in the United States persist, and figuring out strategies to eliminate them. While inequalities in health and mortality rightfully receive a great deal of attention in academic, policy, and media circles, I believe our current approaches are misguided by their failure to consider long-term cohort effects. As such, I will continue to advance a cohort framework for analyzing trends in health disparities, rooting the life course perspective in a richer understanding of the sociohistorical conditions that cohorts endure across time. Thus far, my dissertation has shown that educational attainment is growing increasingly important at shaping U.S. adult mortality risk across cohorts. That the education-mortality gradient is growing across cohorts partly reflects the fact that the adult environment is becoming more important in shaping the health profiles of U.S. populations. The life course literature on health and mortality has been overwhelmingly interested in establishing the links between early childhood health and older adult mortality risk. However, as childhood diseases have been reduced, the adult environment has grown relatively more important at shaping morbidity and mortality risk in later life. As a result, taking an accurate account of cohorts' disparate exposures to changes in both childhood and adult environments is essential for understanding current and future health and mortality trends. Being mindful of the enduring

effects of past and current racism in America is especially important in this regard. The prevalence, severity, and persistence of harsh early-life conditions are all higher for U.S. black cohorts than for U.S. white cohorts.

In terms of specific questions about life course and cohort changes to U.S. health and mortality, I am largely interested in two avenues of research. The first regards the changes to the availability, access, and use of health care technologies across ages and cohorts in the United States. Recent and rapid changes in medical knowledge, drugs, and procedures have significantly altered preventative, management, and restorative measures of health. I am interested in examining how these measures have disparately affected cohorts, as well as how life course effects condition the effectiveness of these later life interventions. Second, I am interested in cohort increases in obesity and changes to overall nutrition. Dramatic changes in diet and risk factors have been documented in the U.S. population, and research has begun to link obesity and/or nutritional deficiencies to the onset of "primary" and "secondary" frailty (i.e., frailty in the presence or absence of chronic illness) (Fried et al. 2004).

To continue my research I am submitting an application for access to the Medicare Beneficiary Survey (MCBS) Access to Care data 1991-2008, which will permit analyses of various prospective health measures in older age such as functional limitations and disabilities, physician diagnoses of conditions and disease, and old-age mortality. The MCBS dataset has several unique advantages for analyzing temporal change in aging processes, the prevalence and incidence of functional limitations, and the onset of frailty. The unique sample is generated from rotating longitudinal panels, spanning nearly twenty years of time and thus permits age-period-cohort analyses of old-age health and mortality patterns in the United States. Also, the sample includes both the non-institutionalized and institutionalized older-adult U.S. populations,

contains much information pertaining to health insurance, access to, and utilization of services, and is linked to the social security mortality files. The design, measures, and linked mortality files allow for rich analyses of the multiple pathways by which aging, senescence, and frailties determine old-age mortality risk. Further, I also plan to utilize the National Health and Nutrition Examination Surveys (NHANES) to integrate questions of health behaviors, social support, and additional health outcomes. Each dataset will enable me to further explore cohort changes to race/ethnic and educational differences in functional limitations, disease patterns, and access to and use of health technologies.

### 5.3.1 *Research Questions*

1. *"Successful Aging" versus Older-Age "Frailty" across Cohorts*. In my dissertation work I examine cohort differences in the relationship between educational attainment, race, and U.S. adult mortality risk. I would like to extend this line of work to incorporate broader health measures and examine disparate cohort effects in the ways educational attainment affects the onset of disease, functional limitations, disability, and other dimensions of "frailty" in older age Americans. Using the NHIS-LMF 1986-2006, the MCBS 1991-2007, and the GSS 1972-2008, I would like to examine cohort differences in the extent of "successful" aging in the United States (McLaughlin et al. 2010). I believe that the same theoretical framework for understanding how cohort processes affect change in U.S. adult mortality risk (i.e., a cohort-specific life course perspective) can be similarly applied to understanding how disease and disability are being compressed into increasingly older ages, and the inequalities in these processes.

2. *Health Care Use and "Recovery*." The MCBS contains rich data pertaining to older Americans' use of healthcare services, the onset and progression of disease, recovery from

diseases and/or disabilities, and cause-specific mortality. These data therefore permit multistate analyses of the multiple pathways by which (1) the onset of functional limitations, disability, and/or other dimensions of frailty occurs, (2) individuals respond to and manage their illness, disability, and/or other health condition, and thus individuals either (3) recover from their condition(s) or (4) die from either their condition(s) and/or other causes. I am interested in applying a cohort-specific life course perspective to examine how these health managing processes are shaped by the confluence of cohort experiences, socioeconomic resources such as education, and available health technologies of the time.

3. *Obesity, Education, and Onset of Disease in Adulthood*. The United States federal government has, for the first time, devoted more resources to fight obesity in the U.S. population than to fight tobacco use. While smoking has been and continues to be the leading burden of disease in the United States, obesity, combined with a sedentary lifestyle, is quickly emerging as the most significant lifestyle risk factors associated with the leading causes of death in this country. The obesity epidemic is certainly a period phenomenon, but there are important cohort factors to consider as well (Reither, Hauser, and Yang 2008). Not only is obesity a significant risk factor that health researchers must pay close attention to, but the *time spent* obese is an equally important factor for consideration. Indeed, the problem of America's obesity epidemic is not only that we're so heavy, but that we've been so heavy for so long. Modeling and forecasting life course processes for those cohorts that developed through childhood, adolescence, and early adulthood during the American obesity epidemic will be important for understanding the long-term implications of obesity in the U.S. population. In this case, then, I am not only interested in cohort processes as they relate to obesity and old age health, but I am interested in analyzing young adult health as well.

4. *Cohorts, Cumulative Exposure, and Biomarkers*. The growing discipline of biodemography has advanced the study of population health in multiple ways. Employing the life course perspective to analyze multiple dimensions of health is vital to understanding the physical and social factors that shape susceptibility to disease and early death. Environmental factors, stress, hormonal change, and other physiological factors such as inheritable susceptibilities to disease are necessary considerations for building an integrative field of biodemography. Important to this endeavor is paying close attention to cumulative exposure time to these factors, and recognizing the central role a cohort perspective plays in shaping these exposure times. I hope to investigate cohort variation in the bio- and social-shaping of disease processes, the mechanisms by which the life course is changing across cohorts, and the implications for current and future disparities in U.S. health and mortality.

**Table 5.1: OLS Regression Results of U.S. White and Black Men's Frailty, NHIS-LMF 1986-2006**

| | Model 1 | | | | Model 2 | | | | Model 3 | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Black | | White | | Black | | White | | Black | | White | |
| | b | SE | b | SE | b | SE | b | SE | b | SE | b | SE |
| Average Age | -.0010 | (.0018) | .0099 | (.0008) | -.0020 | (.0018) | .0095 | (.0009) | .0005 | (.0056) | .0151 | (.0023) |
| Recruit | | | | | -.5308 | (.0963) | -.2389 | (.0317) | -.5073 | (.0971) | -.2118 | (.0310) |
| Cohort 1900 | | | | | | | | | -.8617 | (.2377) | -.5030 | (.1102) |
| Cohort 1905 | | | | | | | | | -.7179 | (.2278) | -.3994 | (.0928) |
| Cohort 1910 | | | | | | | | | -.5669 | (.2117) | -.2778 | (.0901) |
| Cohort 1915 | | | | | | | | | -.4756 | (.1900) | -.1757 | (.0829) |
| Cohort 1920 | | | | | | | | | -.2234 | (.1744) | -.1475 | (.0732) |
| Cohort 1925 | | | | | | | | | -.2415 | (.1530) | -.0549 | (.0686) |
| Cohort 1930 | | | | | | | | | -.1076 | (.1403) | -.0641 | (.0611) |
| Cohort 1935 | | | | | | | | | -.0684 | (.1183) | .0024 | (.0582) |
| Cohort 1940 | | | | | | | | | .0347 | (.1050) | -.0123 | (.0485) |
| Cohort 1950 | | | | | | | | | .0703 | (.1144) | .0345 | (.0474) |
| Cohort 1955 | | | | | | | | | -.0417 | (.1120) | .0810 | (.0507) |
| Cohort 1960 | | | | | | | | | -.1286 | (.1612) | .0620 | (.0650) |
| Cohort 1965 | | | | | | | | | -.2736 | (.1749) | .1992 | (.0904) |
| Intercept | .5132 | | -.1668 | | .5582 | | -.1463 | | .6468 | | -.1588 | |
| df | 1 | | 1 | | 2 | | 2 | | 15 | | 15 | |
| $R^2$ | .0007 | | .3009 | | .0836 | | .3676 | | .1468 | | .4009 | |

Note: N of observations = 4,429,601 person-years; N of cells = 514

**Figure 5.1: Observed Cohort Robustness in White and Black Men's NHIS-LMF 1986-2006 Samples (Top Row), by Survey Entry Status (Bottom Row)**

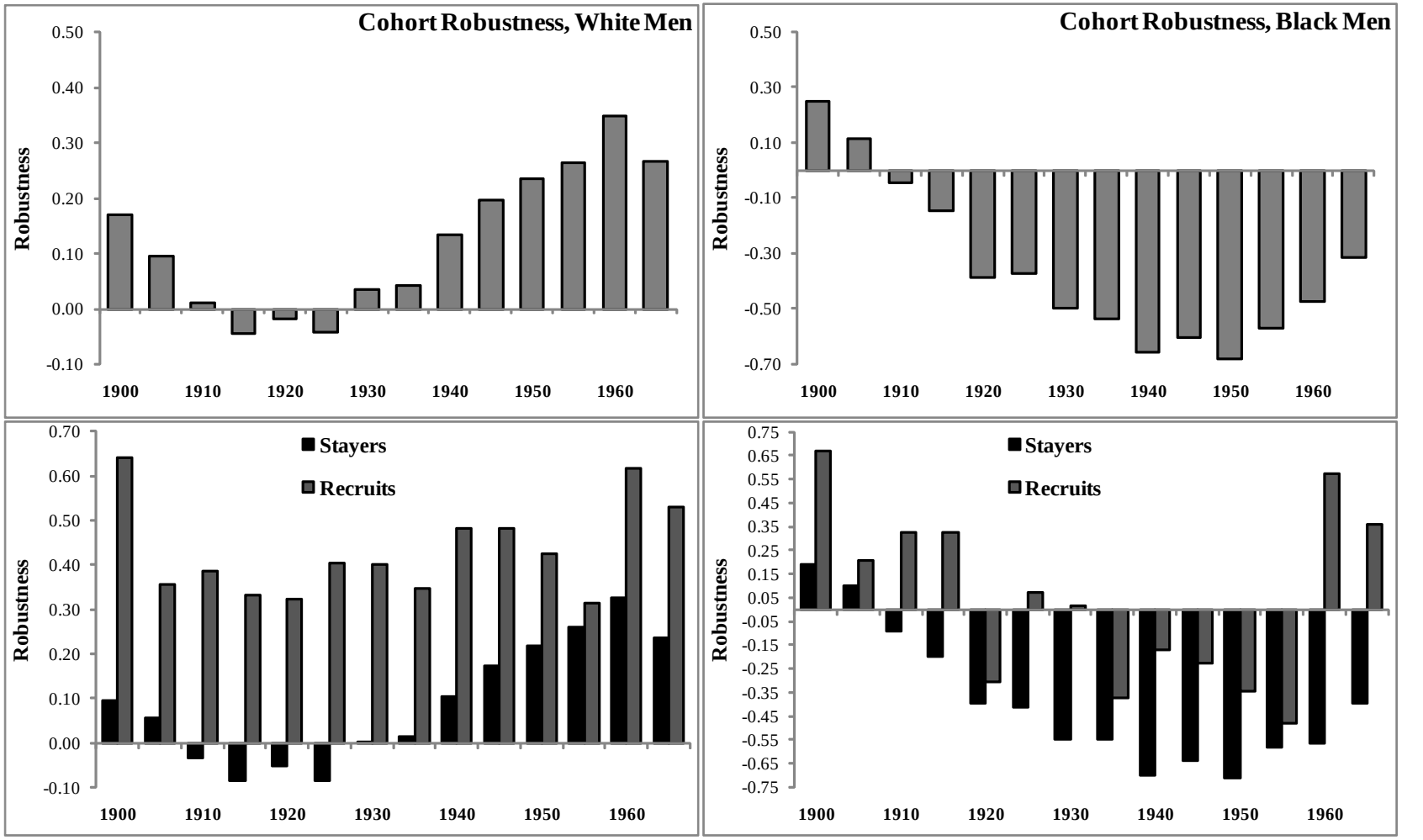
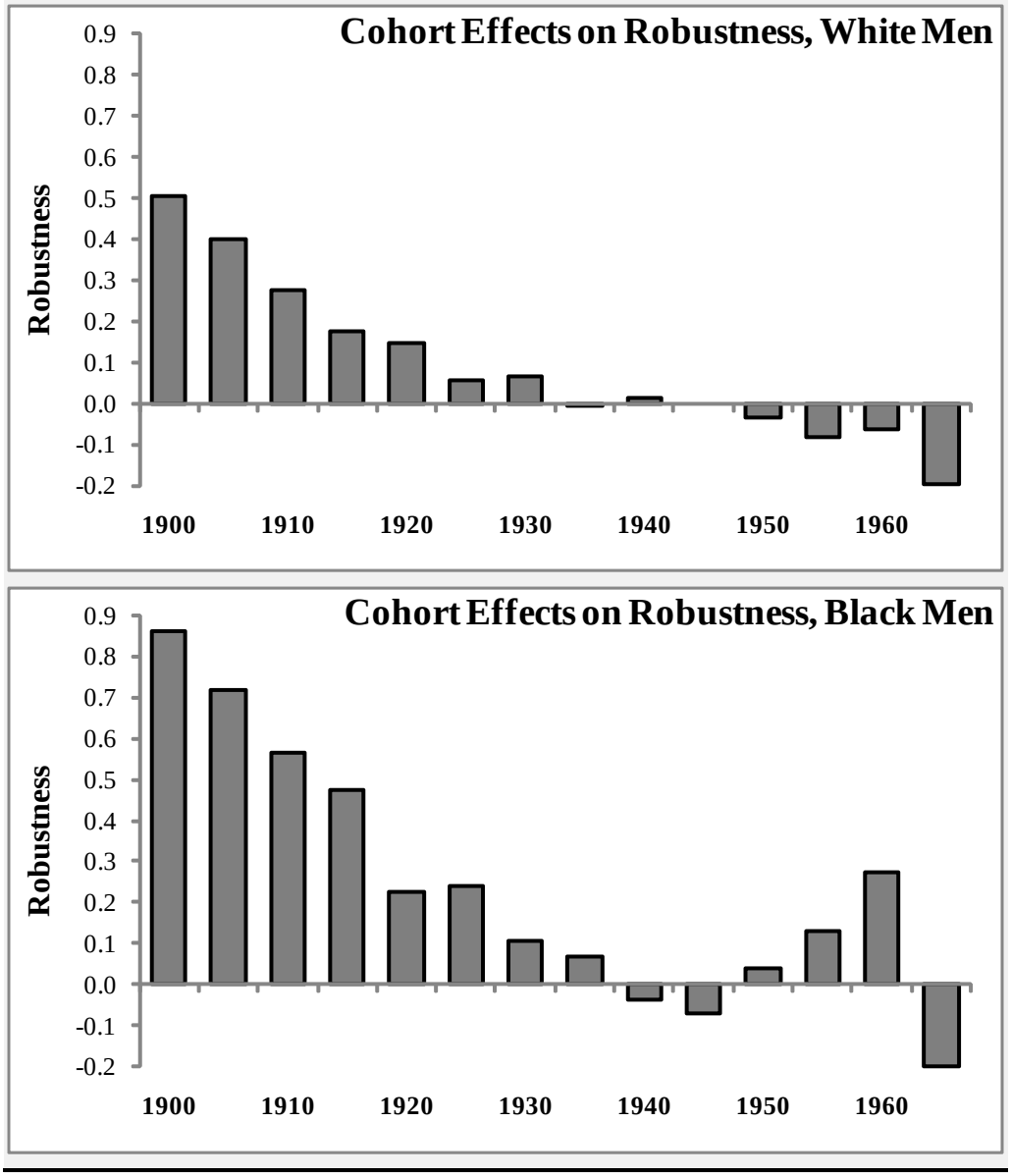


**Figure 5.2: White and Black Men's Cohort-level Robustness, NHIS-LMF 1986-2006, Conditioning on Age Composition and Survey Entry**



# Appendix

SAS 9.2 script estimating 5yr Age x 4yr Period x 5yr Cohort HAPC-CCREM model of U.S. black men's mortality by using SAS's PROC GLIMMIX:

```
title 'CCREM – Black Men' ;
proc glimmix data = apcbm maxopt = 150 ;
  class coh5yr per4yr ;
  model tdead = age25 age30 age35 age40 age45 age50 age55 age60
                age70 age75 age80 age85 age90 age95
              / dist = poisson
                link = log
                offset = lnexp
                solution ;
  random coh5yr / solution ;
  random per4yr / solution ;
  weight w ;
run ;
```

Program in R estimating 5yr Age x 5yr Period x 5yr Cohort HAPC-CCREM model of U.S. black men's mortality using alternative algorithms and MCMC under a Gibbs sampling approach. Packages *lme4*, *BRugs*, and *R2WinBUGS* used:

```
setwd("C:/Users/Ryan/Documents/EduMort/Crossover
Paper/Demography/R&R/R_men")
getwd()

dead <- read.table("nhbm_140data.txt", header = TRUE)

tdead <- dead$tdead
per5yr <-dead$per5yr
age5yr <-dead$age5yr
coh5yr <-dead$coh5yr
w <- dead$cellw
lnexp <- dead$lnexp
age25 <- as.numeric(dead$age5yr==0)
age30 <- as.numeric(dead$age5yr==1)
age35 <- as.numeric(dead$age5yr==2)
age40 <- as.numeric(dead$age5yr==3)
age45 <- as.numeric(dead$age5yr==4)
age50 <- as.numeric(dead$age5yr==5)
age55 <- as.numeric(dead$age5yr==6)
age60 <- as.numeric(dead$age5yr==7)
age65 <- as.numeric(dead$age5yr==8)
age70 <- as.numeric(dead$age5yr==9)
age75 <- as.numeric(dead$age5yr==10)
age80 <- as.numeric(dead$age5yr==11)
```

```
age85 <- as.numeric(dead$age5yr==12)
age90 <- as.numeric(dead$age5yr==13)
age95 <- as.numeric(dead$age5yr==14)


library(lme4)


# fit a Generalized Linear Mixed Model (Laplace default)
gm1 <- glmer(tdead ~ age25 + age30 + age35 + age40 + age45 + age55 +
age60 + age65 + age70 + age75 + age80 + age85 + age90 + age95 + (1 |
per5yr) + (1 | coh5yr) + offset(lnexp), family = poisson, weights=w)

# extract random effects
R <- ranef(gm1)
U <- R$per5yr
V <- R$coh5yr
U
V

library(BRugs)
library(R2WinBUGS)


M <- length(unique(per5yr))
perID <- unique(per5yr) + 1
# these are not used
c1 <- as.numeric(coh5yr==1)
c2 <- as.numeric(coh5yr==2)
c3 <- as.numeric(coh5yr==3)
c4 <- as.numeric(coh5yr==4)
c5 <- as.numeric(coh5yr==5)
c6 <- as.numeric(coh5yr==6)
c7 <- as.numeric(coh5yr==7)
c8 <- as.numeric(coh5yr==8)
c9 <- as.numeric(coh5yr==9)
c10 <- as.numeric(coh5yr==10)
c11 <- as.numeric(coh5yr==11)
c12 <- as.numeric(coh5yr==12)
c13 <- as.numeric(coh5yr==13)
c14 <- as.numeric(coh5yr==14)
c15 <- as.numeric(coh5yr==15)
c16 <- as.numeric(coh5yr==16)

p1 <- as.numeric(per5yr==0)
p2 <- as.numeric(per5yr==1)
p3 <- as.numeric(per5yr==2)
p4 <- as.numeric(per5yr==3)
p5 <- as.numeric(per5yr==4)


# use part of the b.mle vector for starting values to the MCMC
```

```
b.mle <- coef(glm(tdead ~ age25 + age30 + age35 + age40 + age45 +
age55 +
                        age60 + age65 + age70 + age75 + age80 + age85 +
                        age90 + age95 +
c1 + c2 + c3 + c4 + c5 + c6 + c8 + c9 + c10 + c11 + c12 + c13 + c14 +
c15 + c16 + p2 + p2 + p4 + p5 + offset(lnexp), weights=w,
family=poisson))


N <- length(tdead)
A <- array(c(age25, age30, age35, age40, age45, age55, age60, age65,
age70, age75, age80, age85, age90, age95), c(140,14))

perID <- per5yr + 1
cohID <- coh5yr

# data for MCMC
data <- list(N=N,
             M=M,
             Y=tdead,
          lnexp=lnexp,
          perID=perID,
          cohID=cohID,
          A=structure(.Data=A, .Dim=c(140,14))
          )

#MCMC model
APCmod<- function()
{
    # normal priors on random effects
    for ( k in 1 : M) {
     U[k] ~ dnorm(0, tauU)
     }
     for ( j in 1: 16) {
     V[j] ~ dnorm(0,tauV)
     }
     # likelihood Y~pois(mu)
            for ( i in 1 : N ) {
               Y[i] ~ dpois(mu[i])
                    log(mu[i]) <- a  + inprod(b[],A[i,]) + lnexp[i] +
U[perID[i]] + V[cohID[i]]
               }
     # normal priors on fixed effect (with big variances so as to make
them uninfomative)
                  a   ~ dnorm(0,1.0E-6)
              for(i in 1:14) {
                b[i] ~ dnorm(0,1.0E-6)
                }
     # uninformative priors on variance components
              tauU ~ dgamma(0.01, 0.01)
              tauV ~ dgamma(0.01, 0.01)
```

```
# transform precisions to variances
               sigma2U <- 1/tauU
               sigma2V <- 1/tauV
}
## some temporary filename:
filename <- file.path("C:/Users/Ryan/Documents/EduMort/Crossover
Paper/Demography/R&R", "APCmod.bug")

## write model file:
writeModel(APCmod, filename)

## and let's take a look:
file.show(filename)

inits1 <- function() {
list(a=b.mle[1], b=b.mle[2:15], tauU=1/.002, tauV=1/.3)
}

# run a test model for debugging under WinBUGS because debug option
does not yet work in OpenBUGS

# update inside of WinBUGS

bugs(data, inits=inits1, debug=TRUE ,
model.file="C:/Users/Ryan/Documents/EduMort/Crossover
Paper/Demography/R&R/APCmod.bug", n.chains=3, parameters = c("a","b",
"sigma2U", "sigma2V", "U", "V"), n.iter=10000, n.thin=1,
bugs.directory="c:/Program Files/WinBUGS14")
```

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