Is the "Long Arm of Childhood" Growing Shorter? Race, Socioeconomic Status, and Changes in U.S. Adult Mortality*

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ABSTRACT

The early-life conditions of U.S. birth cohorts have markedly improved over the past 100 years. At the same time, education in adulthood has grown increasingly important in securing good health and lowering mortality risk. Consequently, at the population-level the "long arm of childhood" is growing shorter with respect to U.S. adult mortality risk. Further, these changes are associated with cohort forces that substantially differ for the U.S. white and black populations. We use the NHIS-LMF 1986-2006 and U.S. census data to illustrate how racial differences in cohort patterns of U.S. adult mortality are tied to racial inequalities in both early-life conditions and disparate health returns to educational attainment. These long-term, cumulative, cohort processes sustain significant and substantive racial disparities in socioeconomic gradients of U.S. mortality.

INTRODUCTION

Racial disparities in U.S. mortality rates are substantively large, occur at all ages, and have persisted across time (Hummer 1996; Hummer and Chinn 2011; Davey Smith et al. 1998; Sloan et al. 2010). These patterns provide perhaps the starkest indicator of the enduring racial inequalities in contemporary America. At the same time, socioeconomic disparities in U.S. health and mortality are large and growing as well (Cutler et al. 2008; Masters, Hummer, and Powers forthcoming; Meara, Richards, and Cutler 2008; Montez et al. 2011). While racial and socioeconomic disparities in U.S. mortality are often analyzed together, evidence linking the two is mixed (Crimmins, Hayward, and Seeman 2004). Further, no comprehensive theory exists to explain their association or their persistence over time. We argue that education is growing increasingly important in garnering health and securing longevity in a rapidly changing world, but believe this change is occurring differently in the U.S. black and white populations. We further argue that racial and socioeconomic disparities in U.S. health and mortality are intricately linked through cohort processes. Drawing from fundamental cause theory and the notion of a "cohort morbidity phenotype," we employ a life course and cohort perspective to examine the intersection between race, socioeconomic status (SES), and adult mortality risk in the United States (Link and Phelan 1995; Finch and Crimmins 2004).

Our results suggest that education is becoming an increasingly bifurcated health resource reflecting long-term cumulative processes that are rooted in U.S. racial inequalities. On the one hand, evidence suggests that the "long arm of childhood" may be *longer* for blacks than for whites. That is, deleterious childhood effects take a greater aggregated toll on U.S. black cohorts' adult mortality risk than on U.S. white cohorts' adult mortality risk. On the other hand, mounting evidence also suggests that black Americans' socioeconomic and health attainment is often

hindered by discrimination, segregation, elevated stress, and other disadvantages faced during adulthood. These childhood and adulthood forces are not mutually exclusive, and our results are consistent with both bodies of evidence. In short, differences in the ability of white and black Americans to transfer socioeconomic resources into better health and longer lives stem from racial inequalities in both childhood and adulthood, and these inequalities are imbedded in cohorts' life course histories (Hayward et al. 2000; Masters et al. forthcoming).

Although black-white differences in life expectancy at birth have slightly narrowed in recent decades (Harper et al. 2007), a longer-term assessment of U.S. mortality shows that the relative gap in black-white men's mortality did not close in any significant way across the twentieth century (Sloan et al. 2010). No theory or body of empirical evidence satisfyingly explains the persistence of such a gap. Part of the problem is methodological. Relying on life expectancy to gauge the U.S. racial divide in life chances masks important information that might provide important clues about the reasons for the gap. Particularly important is the fact that 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, for example, 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?

Another limitation of monitoring trends in the race gap via life expectancy is that it cannot tell us if changes are predominantly period or cohort phenomena. Have changes in the U.S. black-white gap in life expectancy been a result of changing racial differences in health inputs during that time period, such as increased use of statins, antihypertensive drugs, antiretroviral AIDS medication, and/or other pharmacological and medical advances? (Chang

and Lauderdale 2009; Macinko and Elo 2009; Anderson, Green and Payne 2009)? Or, did the recent narrowing of the gap reflect changing cohort composition of the black and white populations across that time period? Or, both of these processes? In fact, the vast majority of the most recent narrowing of the black-white gap can be attributed to the drop in black mortality from high rates of homicide and HIV/AIDS-related deaths during the late 1980s and 1990s. Thus, rather than reflecting sustained improvement in relative black-white survival, the recent narrowing of the racial gap in U.S. life expectancy reflects recent age-specific period phenomena. How then do we explain the bigger picture, the persistence of the overall gap?

Most often, researchers point to the racial stratification of socioeconomic resources to explain racial inequalities in U.S. health and mortality (Crimmins et al. 2004; Williams et al. 2010). However, socioeconomic inequality is more often controlled in studies of black-white differences in health and mortality rather than seen as an important consequence of racism in its own right (Hummer 1996). 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, or merely controlling for socioeconomic resources, 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 apparent that researchers must employ a historical and life course perspective to more fully understand the origins and persistence of both socioeconomic and health inequalities between U.S. populations (Blackwell, Hayward and Crimmins 2001; Colen 2011; Crimmins et al. 2004; Ferraro, Farmer and Wybraniec 1997; Hayward and Montez 2011; Sharkey and Elwert 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; Hummer 1996; Shuey and Wilson 2008; Williams and Jackson 2005). These cumulative processes begin in childhood or even earlier, and produce 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 the present study, we 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. We advance a cohort perspective to explain the persistence of disparities in mortality, and the role childhood and adult socioeconomic resources play in shaping these disparities. The cohort framework integrates fundamental cause theory (Link and Phelan 1995), cumulative disadvantage theory, and the life course perspective (Ben-Shlomo and Kuh 2002; Montez and Hayward 2011). The 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 our hypotheses are cohort changes in 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 socioeconomic attainment and adult mortality risk. Five hypotheses guide our analysis.:

H1: Across the twentieth century, prevalences of harsh childhood living conditions were 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.

We 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. We also assess if cohort variation in childhood living conditions is associated with cohort changes to U.S. adult mortality risk:

H3a: Cohort-level measures of deleterious childhood conditions are significantly associated with U.S. black and white men's and women's adult mortality risk.
H3b: These associations are partly mediated by adult SES, but remain significantly associated with adult mortality even after accounting for measures of adult SES.

If evidence supports Hypotheses 1-3, this pattern is consistent with the idea 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 blacks than for whites. In fact, we make no predictions about possible racial differences in the substantive effects of early-life conditions on later-life mortality. However, if harsh early-life conditions are significantly associated with elevated 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. Cohort reductions in U.S.

adult mortality, thus, 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, and this may differ by race (Masters et al. forthcoming). This difference potentially stems 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 variance in adult mortality accounted for by 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. Second, black men and women are more likely than white men and women to face structural discrimination in schooling, housing, employment and other essential social and economic dimensions across the life course (Hummer 1996; Hummer and Chinn 2011; Satcher et al. 2005; Charles and Hurst 2002; Ondrich et al. 2003; Gordon et al. 2000; Williams and Collins 1995; Williams et al. 2010). These barriers may damper blacks' attempts to transfer socioeconomic resources into better health and lower mortality risk. Third, 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, consequently, increased adult mortality (Geronimus et al. 2010; Khansari et al. 2009; Simanek et al. 2008; Scharoun-Lee et al. 2009). Thus, our last hypothesis is:

H5: The association between U.S. adult mortality and adult socioeconomic resources is stronger in the U.S. white population than in the U.S. black population.

*H5a. Specifically, the education and income gradients in adult mortality are **relatively larger** in the U.S. white population than in the U.S. black population.*

*H5b.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.*

We test our hypotheses in several steps. First, five indicators are used to compare U.S. black and white birth cohorts' childhood living conditions during the twentieth century (Hypotheses 1 and 2). Next, we examine age, period, and cohort patterns of black-white differences in U.S. adult mortality rates during the time period 1986 through 2006 to verify previous findings of significant and substantive racial differences in cohort effects on U.S. adult mortality (Hypothesis 4) (Masters forthcoming; Masters, Hummer, and Powers forthcoming; Hummer, Masters, and Finch forthcoming). We then incorporate cohort-based 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, we analyze how educational attainment, income, and poverty affect the age and cohort patterns of U.S. adult mortality risk. We do this separately for educational attainment and for income and poverty, and then together in a single model (Hypothesis 5). To conclude, we examine the effects of adult socioeconomic resources on the age and cohort patterns of U.S. black and white men's and women's adult mortality beyond the effects of cohort-level early-life conditions (further testing Hypotheses 3 and 5).

Findings provide evidence indicating: (1) U.S. black adult mortality rates are falling across cohorts at slower rates than U.S. white adult mortality rates, (2) variation in cohorts' childhood conditions is significantly associated with cohort reductions in black and white mortality, and (3) 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 all five hypotheses, as well as past researchers' contentions that racial inequalities in U.S. adult mortality reflect cumulative stratification processes across the life course (Hummer 1996; Warner and Hayward 2006; Hayward et al. 2000; Crimmins et al. 2004; Williams and Sternthal 2010). However, our findings extend this idea by demonstrating that racial stratification processes are inherently cohort phenomena. Cohorts' unique experiences of history shape disparate life course trajectories of health and socioeconomic attainment, and these cohort forces differ substantively by race (Ben-Shlomo and Kuh 2002; Hayward et al. 2000; Masters forthcoming; Masters et al. forthcoming; Riley 1987; Williams et al. 2010).

BACKGROUND

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

The age standardized death rate for the U.S. black population is comparable to white rates a quarter century ago (Williams and Jackson 2005). Empirical evidence has suggested a strong intersection of race/ethnicity and socioeconomic resources in explaining such disparities (Crimmins et al. 2004; Crimmins and Saito 2001; Hummer 1996; Hayward et al. 2000; Rogers 1992; Rogers, Hummer and Nam 2000; Satcher et al. 2005; Williams and Collins 1995; Williams and Jackson 2005). Studies vary, however, in the degree to which the racial stratification of socioeconomic resources accounts for race gaps in health and mortality (Crimmins et al. 2004; Rogers 1992; Williams et al. 2010; Williams and Jackson 2005; Jemal et al. 2005; Huie et al. 2003). The lack of consensus reflects the multitude of health measures (e.g., disease prevalence, disease incidence, functional limitation, disability, and mortality) and the various ways of conceiving and measuring SES.

In the broadest sense, SES 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, for example, represents social and cultural 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; Rogers 1992). For other measures of SES and other health outcomes, material resources explain very little of the gap (Crimmins et al. 2004; Kahn and Fazio 2005; Shuey and Wilson 2008;

Hummer 1996). These seemingly inconclusive findings raise questions 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 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 stratification processes 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 health and successful aging (Geronimus et al. 2010; McClaughlin et al. 2010; Ferraro and Kelley-Moore 2003).

Beyond Adult SES: Childhood Conditions

"Social chains of risk" 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 can affect one's own cognitive abilities, trajectory of schooling, employment opportunities, and/or other pathways of material and social attainment (Warner and Hayward 2006; Montez and Hayward 2010). Early-life conditions can also 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. 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 importance of cohorts' disparate lifetime exposures to infectious diseases, malnutrition, and bouts of inflammation on subsequent health and mortality risk (Blackwell, Hayward, and Crimmins 2001; Finch and Crimmins 2004; Costa 2000; Fogel 2004, 2005). For example, Finch and Crimmins (2004) found that Swedish birth cohorts that were the first to experience lowered infant and childhood mortality were also the first to experience subsequent declines in older age mortality. Reductions in exposure to inflammation and infectious in early life may directly have led to decreases in subsequent chronic disease morbidity and mortality later in life. 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, 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," they also differ in their susceptibility to later-life mortality risk.

Fogel (2004, 2005), Costa (2002), and Fogel and Costa (1997) report similar results. Their theory of "technophysio evolution" implies strong and lasting synergistic effects of changes in health endowments at birth, improved diet, and disparate exposures to risk factors across cohorts. If verified, technophysio evolution has profound implications for assessing mortality trends. For instance, 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." This perspective has received growing empirical support (Manton et al. 1997; Masters forthcoming; Masters et al. forthcoming; Yang 2008). Its themes were echoed by Manton et al. (1997) who cited 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 that were advanced 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.

The "Remarkable" Century and the "Color Line"

In 1900, over 30 percent of deaths in the United States occurred between birth and the age five. Characteristic of the first stage of the epidemiologic transition, the three leading causes

of deaths were all infectious diseases – pneumonia, tuberculosis, and diarrheal diseases – which together accounted for about one third of all deaths in the United States (Omran 1971; CDC 1999). During this time, the U.S. population had only minimal knowledge of or access to proper nutrition, 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 harsh as well, regardless of geographical location or if one lived in an urban or rural setting (Easterlin 1997; Ewbank 1987). Historically high fertility rates resulted in crowded dwellings and high rates of exposure to myriad infectious and parasitic diseases was 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.

Efforts were well underway, however, to understand, control, and prevent outbreaks of infectious and parasitic diseases and improve nutrition. 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 nutrition, public health, and 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 across the twentieth century (Cutler et al. ...; Jayachandran et al. 2010; Jones 1944; Mason et al. 1997). As a result, between 1900 and 1940 overall mortality levels fell faster in the United States than at any other time in history, with women and children experiencing the greatest reductions in mortality risk. By 2000, the age-adjusted death rate in the United States had been reduced by 56 percent when compared to 1900, and childhood deaths accounted 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 "remarkable," not only because of the unparalleled reductions in mortality but also because every commonly used indicator of standard of living improved, with the most marked improvements seen in the lower classes. Indeed, while economic growth and urbanization had progressed since the 1700s, 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). Since then, in the United States both prevalence and disparities in poor early-life conditions have decreased (Fogel 2005). As bouts with infection, malnutrition, and inflammation early in life have become less frequent, the "insults" they imprint on the "cohort morbidity phenotypes" of successive birth cohorts have become less important in determining the risk of older adult morbidity and mortality risk. As a result, improvements in adult conditions, rather than early life conditions, are becoming increasingly important across cohorts in shaping adult mortality risk (Masters et al. forthcoming).

However, while reductions in infectious diseases, enhancements in nutrition, advances in medical and public health technologies improved childhood and maternal health, these advances were not equally shared across the population. WEB Du Bois presciently warned, "the problem of the Twentieth Century is the problem of the color line," and his prediction was especially astute regarding the country's racial inequalities in health and mortality (1903: *The Forethought*). 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 part of the century when the greatest advances in public health significantly benefitted the United States population. When reductions in overall U.S. mortality rapidly 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. This can be seen both in all-cause mortality as well as in specific causes of death. For instance, a substantial drop in mortality from respiratory tuberculosis occurred between 1910 and 1920, yet 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 to mortality from tuberculosis, as well as pregnancy/maternal causes, have been recorded as well (Jayachandran et al. 2010). Further, 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, while significant childhood 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 significant advances in public health and improvements in survival across the twentieth century, the relative size of these racial disparities remained unchanged for forty years.

To directly test Hypothesis 1 and Hypothesis 2 we draw from previous literature and present five cohort measures indicating the large and persistent racial differences in early-life conditions across twentieth century America. Existing literature and all five measures strongly support our 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 we 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 1), despite rapid reductions in these rates across the first half of the century.

[Figure 1 About Here]

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; CITE more) – then we presume that deleterious conditions in the first year of life were much more severe, much more widespread, or both, 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 no significant racial differences in the rates of change. Consequently, 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).

[Figure 2 About Here]

Third, Figure 2 compares regional and household circumstances into which U.S. black and white cohorts were born. Here again, we note large racial differences in multiple measures of childhood conditions. For each measure – the percent of a cohort born in South, percent born on farm, percent born into a large household, and prevalence of widowhood between ages 30-39 – we see that U.S. black cohorts endured higher prevalence, and began reducing prevalence much later, than U.S. white cohorts. We should note that Preston and Haines (1991) found significant protective effects on infant survival for U.S. cohorts growing up in rural areas and on farms.

However, their analyses focused on U.S. infant mortality the late 1800s, 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 in 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 was about equal (Cutler and Miller 2005). 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 mindful of the rapid changes in the childhood living conditions that occurred during these times. While Preston and Haines (1991) and Warner and Hayward (2006) found a negative association between childhood in a farming household and adult mortality risk, their results may be driven largely by their reference category and the fact that their data are representative of earlier cohorts (i.e., the 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. Again, consistent with Hypothesis 2, we see in Figure 2 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 in the 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 considerably different for U.S. black and white populations, differences which were driven in part by the rural and farming lifestyles of southern blacks, as well as the discrimination directed at both 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. Similarly, one-third of white women received some prenatal care, while the proportion among black women was only 12 percent" (Ewbank 1987: page #). Hospital segregation policies also played a part in these disparities (Almond, Chay and Greenstone 2006), and the differences were especially pronounced in the south. Between 1915 and 1940 substantial changes were made to delivery practices in the United States, and the advances resulted in 56 percent of white births taking place in hospitals by 1940 but 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 1918 Children's Bureau study of child care practices in a rural Mississippi found that rural mothers weaned their infants at too early of ages for proper nutrition. 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). Overall, the U.S. Children's Bureau conducted a series of studies in eight U.S. cities and documented that early weaning had a "devastating effect" on U.S. childhood health during the first decades of the twentieth century (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 probable 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 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 the bottom panel of Figure 2, which shows 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 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, we draw three implications for recent trends in U.S. black and white adult mortality. One, early-life 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 compose an increasing 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, we 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; Masters et al. forthcoming).

CURRENT AIM

We analyze racial differences in the association between socioeconomic resources and U.S. adult mortality by integrating a cohort perspective of mortality change. We do so in four steps. First, we estimate age, period, and cohort patterns of the black-white gap in adult mortality between 1986 and 2006. We 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, we 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. We first estimate APC models separately by educational attainment and by income level and then again with income mediating the effects of educational attainment. Finally, we 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 test our Hypotheses 3, 4 and 5. In general, we revisit the age-old question, "How much of the black-white gap in U.S. adult mortality risk is explained by SES?" We argue that this, in fact, is the wrong question to ask; in the United States the black-white gap in health and mortality is inextricably linked with SES. In today's rapidly changing world, education is growing increasingly associated with survival in the United States. However, it also is an increasingly bifurcated resource. Racial inequalities in both early-lfe and adult conditions make it more difficult for the U.S. black population to transfer education into good health and long life, relative to the U.S. white population. As such, we 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. Furthermore, cohort changes to these gradients are stronger in the U.S. white population than in the U.S. black population. In short, the U.S. white population derives greater – and increasingly greater – health benefits from education and income than does the U.S. black population.

ANALYTICAL STRATEGY

Data

We use 19 waves of the National Health Interview Surveys (NHIS), 1986 through 2004, linked to follow-up mortality information for each cross-section through December 31st, 2006 (NCHS 2010), and linked again to cohort-level indicators of early life conditions. The NHIS linked data were 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.

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 ample, and to limit the use of data where age is top coded, we restricted the NHIS-LMF to U.S.-born non-Hispanic black and white respondents aged 25 to 84 at time of survey and who were 25-99 years of age during the follow-up period. We also dropped from the sample any respondent with missing values of educational attainment, income level, poverty status, and

employment status. Lastly, to ensure we are 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. Grouped variables of birth cohort are composed of 15 five-year blocks ranging from 1900-1904 to 1975-1979. 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 age is coded in fourteen 5-year blocks ranging from 30-34 up to 95-99. Combining the blocks together, the sex- and race-specific samples for our adult mortality analyses are each composed of 157 unique APC cells, collectively accounting for 9,385,015 unique survival histories. Table 1 displays descriptive statistics for the individual-level data.

[Table 1 About Here]

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, we use three categories of education and income to guard against extrapolated linear effects. Educational attainment is measured as 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 the white and black populations, 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 U.S. cohorts born since mid-century attaining a high school degree or higher. The aggregate change in educational attainment is thought to be a primary factor in temporal reductions of U.S. adult mortality (Yang 2008; Lynch 2003). Here, we stratify 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 and cohort effects on mortality. We also use three categories of income to capture variation in the association between household income and adult mortality. Income is measured as earning at or below the poverty line, earning above poverty line but less than $45,000; and earning more than $45,000. This coding allows us to test where in the income gradient black-white differences in mortality are most pronounced. Black-white differences in U.S. income across cohorts reveal a significantly greater proportion of the black male and female samples reporting incomes below the poverty threshold (not shown). These distributions of education and income are consistent with other national estimates (Devas-Walt, Proctor and Smith 2010).

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 we 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 Figure 2). 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 non-white) and sex were obtained from the National Vital Statistics System's (NVSS) "Hist290" historical data document (Figure 1) (http://www.cdc.gov/nchs/nvss/mortality/hist290.htm).

Methods

We 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 U.S.-born respondents aged 30-84 at time of survey, and aged 30-99 at any time between 1986 and December 31st, 2006.

To test Hypothesis 3, we estimated the association between U.S. adult mortality and cohort-level early life conditions by modeling mortality on each early-life condition separately. To account for race and gender differences all models were stratified by race and sex. We then estimated a series of nested models that regressed rates of mortality on the full set of early-life measures. Lastly, we estimated the association between cohort-level early-life conditions while including individual-level adult SES as mediators of the association.

To test Hypothesis 5, we estimated the association between U.S. adult mortality and age- and cohort-varying individual-level adult SES, measured by individual educational attainment and household income. These associations were modeled separately, and we then 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: $$\log[E(D_{ijk})] = \alpha_{jk} + \beta 1_{jk} A_i + BX + \log(R_{ijk}) \quad (1)$$

where D_{ijk} stands for the counts of deaths of the ith age group (for $i = 1, \ldots, n_{jk}$ age groups) within the jth period (for $j = 1, \ldots, J$ time periods) and the kth cohort (for $k = 1, \ldots, K$ birth cohorts); A_i denotes a dummy variable corresponding to each five-year age groups $1,\ldots, n_{jk}$; BX indicates a vector of model-specific covariates and their corresponding effects; α_{jk} is the intercept indicating the reference age group (65-69) during period j and belonging to cohort k; and $\log(R_{ijk})$ is the natural log of the aggregated exposure time lived during each age-period-cohort cell.

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

for the education model and income models: $$\alpha_{1jk} = \pi_{10} + t_{0j} + c_{10k} + c_{20k} + c_{30k} \quad (3)$$

in which α_{jk} specifies that the fixed age effects vary from period to period and from cohort to cohort; π_0 and π_{10} are the respective expected mean at the reference age 65-69 in APC models with no covariates (1), and 65-69 for the >HS population or 65-69 for the mid-income population in APC models with covariates, averaged over all periods and cohorts; t_{0j} is the overall four-year period effect averaged over all five-year birth cohorts with variance σ^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 $\sigma^2_{c10,}\sigma^2_{c30,}\sigma^2_{c30}$.

We 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. The software programs R and WinBUGS were used to estimate Hierarchical Bayesian Models using Gibbs sampling for all analyses (Gelman et al. 2006; Lynch 2007; Yang 2006). Analytical scripts are available upon request.

RESULTS

Early-life Conditions and U.S. Adult Mortality

Table 2 presents estimated cohort and period variance components of U.S. black and white women's and men's adult mortality rates between 1986 and 2006.

[Table 2 About Here]

For both men and women, results from Model 1 indicate that the U.S. white population experienced significantly greater 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 revealed in Bayesian estimates of individual random cohort effects of U.S. black and white men's and women's adult mortality trends (results not shown, but consistent with Masters et al. forthcoming). This evidence strongly supports Hypothesis 4, in that we observe 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 Table 2, we find evidence that much of this cohort variation, irrespective of race or gender, is associated with the proportion of birth cohorts born in Southern states. 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 mortality rates 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 male and female cohorts, and for U.S. black female cohorts, this association is mediated by cohorts' household living conditions and level of infant mortality. The association between "% born in South" and mortality rates for U.S. black and white women is accounted for by the cohort proportion being born on a farm, which in turn is partly mediated by cohort variation in infant mortality rates. Similarly, 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, however, 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 variation in cohort 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 being born 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 associated with variation in individual-level educational attainment and income (see models "Early-Education" and "Early-Education-Income in Table 3"). Overall, this evidence supports part a) of Hypothesis 3, in that we see strong associations between indicators of cohorts' early-life conditions and subsequent risk of adult mortality. Part b) of Hypothesis 3 also receives strong empirical support in the findings of Models 4 and 5 in Tables 3 and 4, in which the associations between U.S. cohorts' early-life conditions and adult mortality remain significant even after controlling for levels of education and income in adulthood.

Socioeconomic Resources and U.S. Adult Mortality

Table 3 and Table 4 present estimates of random cohort and period variance components, estimates of cohort-level childhood living conditions, and estimates of individual-level adult socioeconomic resources and on U.S. adult men's and women's mortality across a nested series of HAPC-CCREMs. Men's results are presented in Table 3, and estimates for women are presented in Table 4.

[Table 3 About Here]

Black-White Differences in the Education-Mortality Association

Results from Model 1 in Table 3 provide evidence that strongly supports both parts a) and b) of our fifth hypothesis. Regarding part 5a), which proposed that the educational gradient in U.S. adult mortality between 1986 and 2006 was larger for white men than black men, we 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, a significant and substantively large difference. 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 across age in Figure 3.

[Figure 3 About Here]

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 3). 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. This evidence supports the contention that U.S. white men derive a significantly greater protective effect from educational achievement than do U.S. black men.

Results from the men's sample provide evidence supporting part b) of our 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 respectively found in U.S. black men's mortality. These differences are most clearly shown in the bottom panels of Figure 3, in which 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 only very small cohort changes in 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 did not significantly change 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 only minimal cohort reductions in 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. (Similar results are

found in Model 2, which uses income as a measure of SES, but due to space limitations these results are not discussed here. An appendix can be found online)

[Table 4 About Here]

Results presented in Table 4 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 not significant at any 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.

[Figure 4 About Here]

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). 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, we find evidence supporting part a) of our 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 in the educational gradient in U.S. black women's adult mortality between 1986 and 2006. Conversely, cohort changes in 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. Results from Model 3 further show that while income mediates the effects of educational attainment on U.S. adult mortality rates, irrespective of race or gender, significant and large education effects persist.

In general, evidence supports both part a) and part b) of our 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 growth of these gradients across cohorts is 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 SES attainment was greater in the U.S. white population than in the U.S. black population. To further support this pattern, we re-estimated the Models 1 and 3 while controlling for the effects of cohorts' early-life conditions on later-life mortality rates.

Childhood Conditions, Adult Socioeconomic Resources, and Adult Mortality

Models 4 and 5 in Tables 3 and 4 present estimates of the associations between early-life conditions, adulthood education and income, and U.S. adult mortality rates. For men, accounting for cohorts' variation in early-life conditions significantly changes the educational gradient in both the U.S. white and black population's adult mortality rates. For white men, 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 1.152 in Model 1 to .699 in Model4. Thus, we find evidence that a significant proportion of the education-mortality relationship in U.S. white men is associated with the possibly disparate early-life conditions experienced by the <HS and >HS white male populations. We find similar effects in the U.S. black men's sample. The .710 difference between the log mortality rates of the <HS and the >HS population in Model 2 has been reduced to .478 when accounting for variation in the percent of U.S. black cohorts born in the South in Model 4. The narrowing of these educational gradients can be seen in Figure 5.

[Figure 5 About Here]

Model 5 further tests mediating effects of income on the education-mortality relationship for U.S. men beyond the effects of cohorts' early-life conditions. The .699 difference in logged mortality between U.S. white men with a <HS education and those with a >HS education in Model 4 has been reduced to .641 in Model 5. A similary small reduction in the mortality difference between HS and >HS groups is seen (.345 to .304). In the U.S. black male population, mediating effects of income are also found. The .478 difference in logged mortality between the <HS and >HS populations in Model 4 are significantly reduced to .178 in Model 5, whereas the non significant .178 effects in Model 4 are not significantly changed to .259. Thus, we find only weak evidence that the education-mortality relationship in the U.S. adult male population is primarily explained by variation in income. Indeed, we find strong evidence that cohort variation

in U.S. men's early-life conditions accounts for more of the mortality difference between <HS and >HS men's mortality rates than the mediating effects of individual-level income. For white men the <HS to >HS effect of 1.152 in Model 1 is reduced to .913 when accounting for individual-level income in Model 2, whereas it's reduced to .699 when accounting for cohort-level early-life conditions in Model 4. That futher accounting for income reduces the .699 difference to .641 suggests only minimal further mediating effects of income.

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 consisten with those found in the men's models (Models 4 and 5 in Table 4). In Model 4, education remains significantly associated with adult mortality, but the substantive effects have been considerably reduced from estimates in Model 1. For U.S. white women, the <HS to >HS effect of 1.269 and the HS to >HS effect of .553 in Model 1 have been respectively reduced to significant estimates of .924 and .461. Accounting for early-life conditions in the U.S. black female population reduced the significant .664 difference in logged mortality rates between <HS and >HS to .426. The total substantive effect of these reductions is best seen by comparing the top-panel of Figure 5, which shows U.S. women's black-white differences in adult mortality across age for <HS and >HS after controlling for cohort variation in early-life conditions, and the middle-panel of Figure 3. The black-white gap in U.S. women's mortality at these respective education levels have been significantly reduced, with the black-white gap in women's mortality in the <HS population entirely explained by disparate variation in early-life conditions.

Lastly, findings suggest that the role of income in the black-white gap in U.S. women's mortality is quite different from its role in explaining the black-white gap in U.S. men's mortality. Accounting for income in Model 5 widens the educational differences in U.S. white

women's adult mortality, increasing the <HS effect of .924 in Model 4 to 1.229 in Model 5. Similarly, the .461 HS effect in Model 4 increases, not significantly, to .500 after accounting for adult income in Model 5. The effect of educational attainment on U.S. white women's adult mortality rates, after accounting for early-life conditions, is therefore negatively mediated by income. This, however, is not the case in the U.S. black women's population. The significant .426 effect of <HS on U.S. black women's mortality in Model 4 is reduced to a non significant .390 effect in Model 5, indicating that income significantly accounts for a large part of the educational gap in U.S. black women's adult mortality risk. The significant role of income in U.S. black women's adult mortality is further seen in Model 3, wherein the significant effect of education on U.S. black women's adult mortality is accounted for by the significant effect of income.

This finding is incredibly important when coupled with the finding that early-life conditions fail to explain the association between educational attainment and U.S. black women's adult mortality (see Model 1 to Model 4). In the U.S. white female population, a significant proportion of the education-mortality association is accounted for by cohorts' disparate early-life conditions, not by variation in income levels. In the U.S. black female population, the exact opposite is true. Variation in cohorts' early-life conditions do little to dampen the effects of adult educational attainment on mortality, yet variation in income level effectly explains away the significance of education's effect. Indeed, when comparing Model 3 to Model 5 we see that accounting for cohorts' early-life conditions does not significantly alter either the effects of education or income on U.S. black women's adult mortality, despite the early-life conditions being significantly and substantively associated with mortality.

DISCUSSION

Racial disparities in U.S. health and mortality persisted across the twentieth century, despite major advances to improve health and extend life at all ages, as well as recent policy initiatives to eliminate health disparities (Healthy People 2000, 2010, 2020). 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 in order to more fully 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 paper we add 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," our findings indicate that the enduring effects of childhood conditions on adult mortality risk vary significantly across U.S. birth cohorts. Further, in the United States these cohort processes are significantly conditioned by race, thus helping to perpetuate racial differences in both health and socioeconomic attainment across the life course.

In this line, we find evidence consistent with all five 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 and/or confounding 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. Further, accounting for cohort variation in childhood conditions narrowed the black-white differences in educational and income gradients in adult mortality risk. And six, cohort changes (i.e., growth) in the educational and income gradients in U.S. adult mortality are significantly greater in the white population than in the black population. Taken together, the evidence is consistent with our 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 paper 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). We are 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 we are not using a continuous measure of birth cohorts, the cohort effects cannot be extrapolated or driven by outliers or faulty data. Lastly, our estimates of cohort patterns of U.S. black and white mortality are very consistent with estimates of cohort patterns in official mortality rates (Hummer, Masters, and Finch forthcoming). Second, we unfortunately have only cohort-level measures of early-life conditions. Thus, we cannot link individual respondents' own living conditions in childhood to their subsequent attainment of education and income, nor to their health and mortality risk in adulthood. Third, we do not have early-life conditions for education-specific subsamples. That is, measures of cohorts' 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 our 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 1: Descriptive Statistics 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.
Age	46.0	15.1	48.1	15.7	46.2	14.8	47.2	15.0
Year	1994.0	5.3	1993.8	5.2	1994.1	5.4	1993.9	5.2
Birth Year	1948.0	16.0	1945.7	16.5	1947.9	15.7	1946.7	15.7
Deceased	14.8	35.5	13.9	34.6	20.2	40.2	16.3	36.9
*Cohort Childhood Indicators**								
Born in South	73.0	11.0	29.4	2.7	73.1	10.9	29.4	2.7
Born on Farm	29.8	18.0	19.1	9.4	30.1	18.0	18.7	9.2
Born into Large Household	58.1	6.5	34.9	5.6	58.1	6.4	34.7	5.5
Infant Mortality Rate	68.9	43.0	40.8	25.4	86.5	50.6	50.5	29.24
Adult Socioeconomic Indicators								
<HS	27.3	44.6	14.5	35.2	29.0	45.4	15.0	35.7
HS	36.8	48.2	39.1	48.8	37.1	48.3	34.5	47.5
>HS	35.9	48.0	46.4	49.9	33.9	47.3	50.5	50.0
Income >$45,000	17.9	38.4	37.4	48.4	25.0	43.3	42.1	49.4
Mid-Income	53.4	49.9	55.3	49.7	57.9	49.4	53.1	49.9
Income < Poverty Threshold	28.7	45.2	7.4	26.1	17.1	37.7	4.8	21.4
N	60,846		335,573		42,423		306,287	

Note: Estimates from person-level samples

* Estimated from IPUMS 1900-1980

Table 2: HAPC-CCREMs of U.S. Adult Mortality Rates with Cohort-level Childhood Covariates

Men	Model 1		Model 2		Model 3		Model 4	
	White	Black	White	Black	White	Black	White	Black
Covariance Parameter								
Cohort	.625	.130	.374	.046	.041	.030	.015	.037
Period	.065	.002	.040	.003	.036	.003	.043	.004
Childhood Conditions [1]								
% Born in South			.406***	.411***	-.150 *	.416***	-.014	.400
% Born on Farm					.486***	-.020	.123 *	.002
% Born in Large Family					.313***	.030	.251***	.046
Infant Mortality Rate							.367***	.025
Intercept	-3.966	-3.527	-4.126	-3.559	-4.032	-3.578	-4.062	-3.586
Deviance	1230.0	989.1	1230.0	979.7	1229.1	979.8	1228.9	979.8
APC Cells	157	157	157	157	157	157	157	157

Women	Model 1		Model 2		Model 3		Model 4	
	White	Black	White	Black	White	Black	White	Black
Covariance Parameter								
Cohort	.663	.231	.410	.072	.042	.038	.024	.035
Period	.105	.027	.096	.054	.088	.041	.106	.041
Childhood Conditions [1]								
% Born in South			.440***	.517***	-.151 *	.480***	-.033	.312 *
% Born on Farm					.651***	.009	.266 *	.068
% Born in Large Family					.159	.014	.148	.044
Infant Mortality Rate							.336***	.180 *
Intercept	-4.590	-4.050	-4.758	-4.052	-4.550	-4.130	-4.651	-4.127
Deviance	1213.0	994.1	1212.5	982.4	1211.0	983.3	1211.5	981.7
APC Cells	157	157	157	157	157	157	157	157

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

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

Table 3: HAPC-CCREM Results of Men's Adult Mortality with Cohort-level Childhood Covariates and Adult SES

	Model 1		Model 2		Model 3[3]		Model 4		Model 5	
	White	Black	White	Black	White	Black	White	Black	White	Black
Covariance Parameter										
<HS Cohort	.116	.017			.161	.002	.013	.003	.017	.004
HS Cohort	.309	.028			.373	.051	.003	.027	.032	.037
>HS Cohort	.404	.157			.302	.049	.014	.035	.031	.025
Poverty Cohort			.249	.044						
"Middle" Income Cohort			.510	.037						
>$45K Income Cohort			.316	.124						
Period	.025	.002	.033	.002	.037	.002	.041	.005	.115	.007
Childhood Conditions [1]										
% Born in South								.284 ***		.261 ***
% Born on Farm							.144 **		.203 ***	
% Born in Large Family							.217 ***		.120 **	
Infant Mortality Rate							.299 ***		.245 ***	
Adult Socioeconomic Resources [2]										
<HS	1.152 **	.710			.913 **	.514 **	.699 **	.478 **	.641 **	.331 *
HS	.283	.377			.173	.364 **	.345 *	.178	.304 *	.259
<= Poverty Line			.876 *	.573 *	.382 ***	.320 ***			.381 ***	.320 ***
>$45,000 Income			-.273	.141	-.382 ***	-.441 ***			-.383 ***	-.442 ***
Intercept	-4.490	-3.842	-4.179	-4.207	-4.182	-3.823	-4.296	-3.819	-4.184	-3.839
Deviance	3057.5	2181.0	2888.0	2155.0	6993.0	4540.0	3040.0	2164.2	6964.9	4524.6
APC Cells	471	458	471	458	1393	1354	471	458	1393	1354

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

2 References are *>HS* at *65-69* and *Poverty<Income<$45,000* at *65-69*

3 Income included only as level-1 covariate

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

Standard Errors Available Upon Request

Table 4: HAPC-CCREM Results of Women's Adult Mortality with Cohort-level Childhood Covariates and Adult SES

	Model 1		Model 2		Model 3[3]		Model 4		Model 5	
	White	Black	White	Black	White	Black	White	Black	White	Black
Covariance Parameter										
<HS Cohort	.028	.002			.072	.003	.009	.004	.015	.004
HS Cohort	.360	.023			.293	.081	.025	.041	.188	.028
>HS Cohort	.489	.145			.233	.113	.037	.060	.089	.053
Poverty Cohort			.045	.031						
"Middle" Income Cohort			.369	.177						
>$45K Income Cohort			.534	.110						
Period	.093	.015	.066	.017	.069	.013	.145	.071	.283	.073
Childhood Conditions [1]										
% Born in South										
% Born on Farm							.311 ***	.290 ***	.384 ***	.261 ***
% Born in Large Family							.077	.034	-.020	.023
Infant Mortality Rate							.237 ***	.221 ***	.180 **	.194 **
Adult Socioeconomic Resources [2]										
<HS	1.269 **	.664 *			1.092 *	.378	.924 **	.426 **	1.229 **	.390
HS	.553 *	.384			.281 ***	.407	.461 **	.274	.500 **	.221
<= Poverty Line			1.220 ***	.581 *	.313 ***	.277 ***			.313 ***	.278 ***
>$45,000 Income			-.540	-.406	-.277 ***	-.322 ***			-.276 ***	-.319 ***
Intercept	-5.018	-4.074	-4.872	-4.323	-4.865	-4.339	-4.996	-4.462	-4.912	-4.158
Deviance	2975.3	2265.0	2841.8	2141.0	7013.9	4665.0	2951.0	2233.3	6959.5	4636.9
APC Cells	471	467	471	467	1392	1313	471	467	1392	1313

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

2 References are *>HS* at *65-69* and *Poverty<Income<$45,000* at *65-69*

3 Income included only as level-1 covariate

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

Standard Errors Available Upon Request

Figure 1. U.S. White and Non-White Cohorts' Infant Mortality Rates, 1900-1975

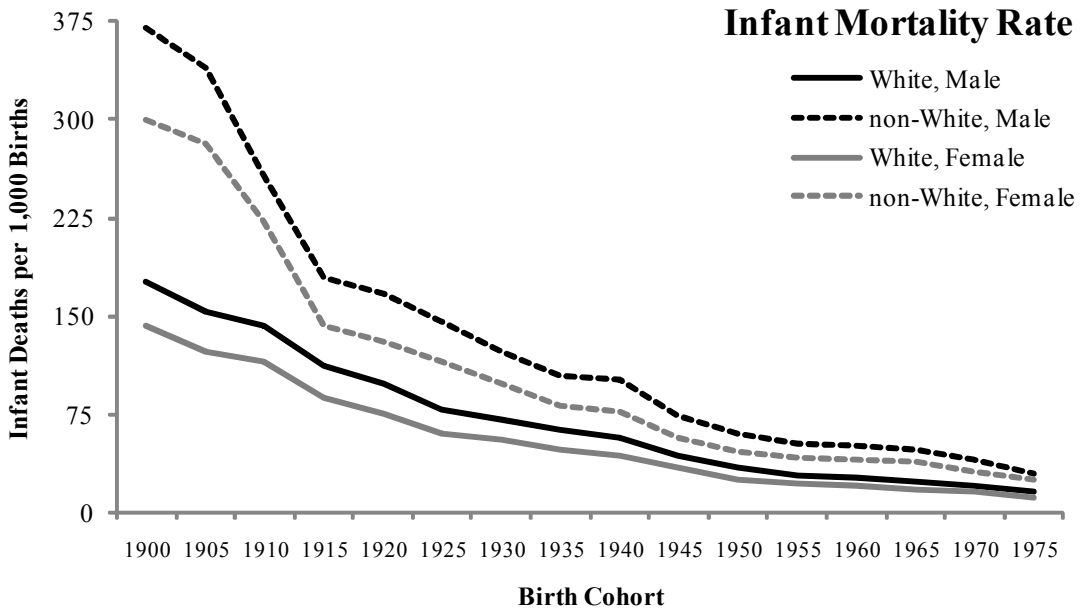


Figure 2. Select Childhood Conditions of U.S. Black and White Birth Cohorts, 1900-1975

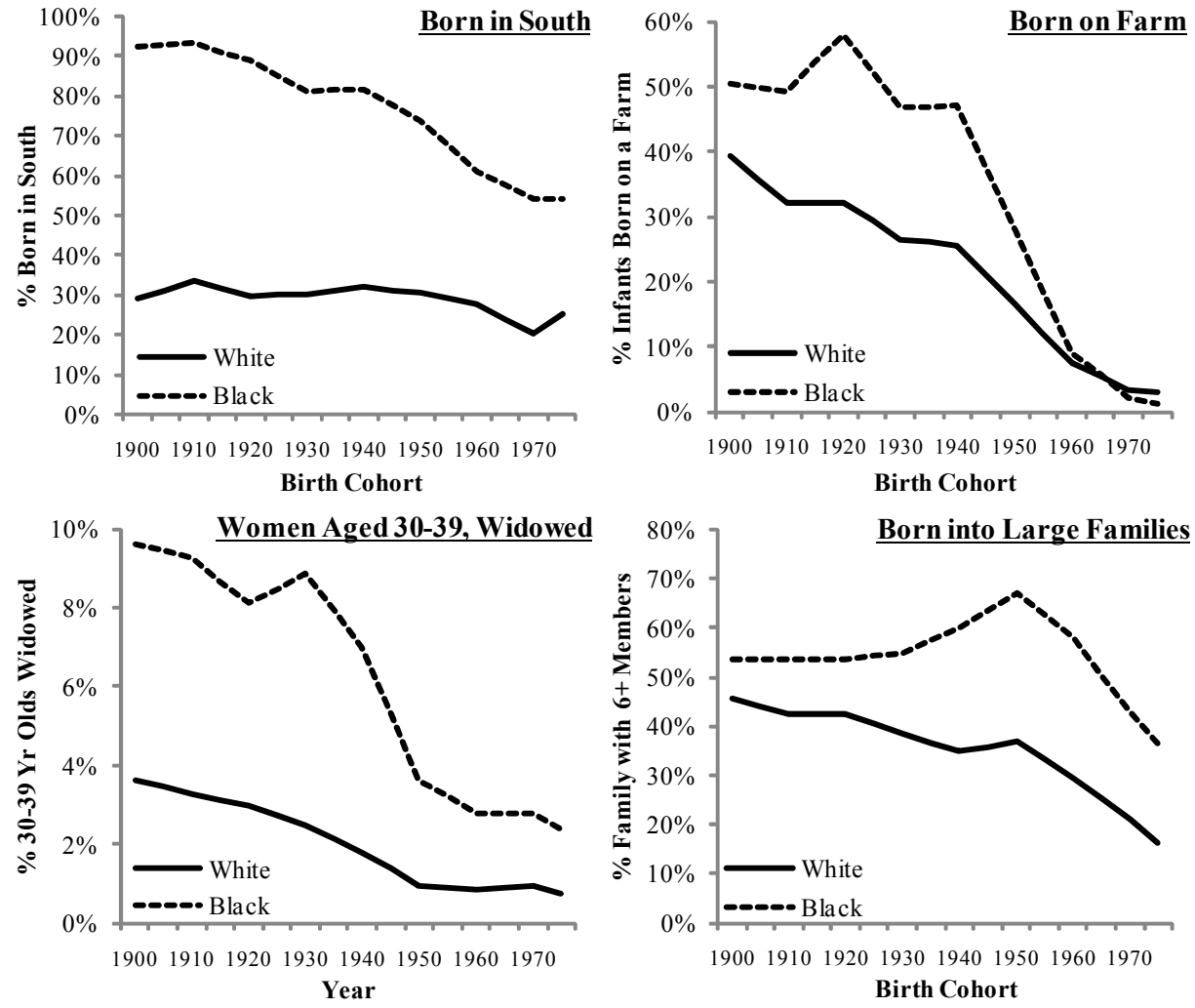


Figure 3. Effects of Education on U.S. Black and White Men's Adult Mortality, 1986-2006

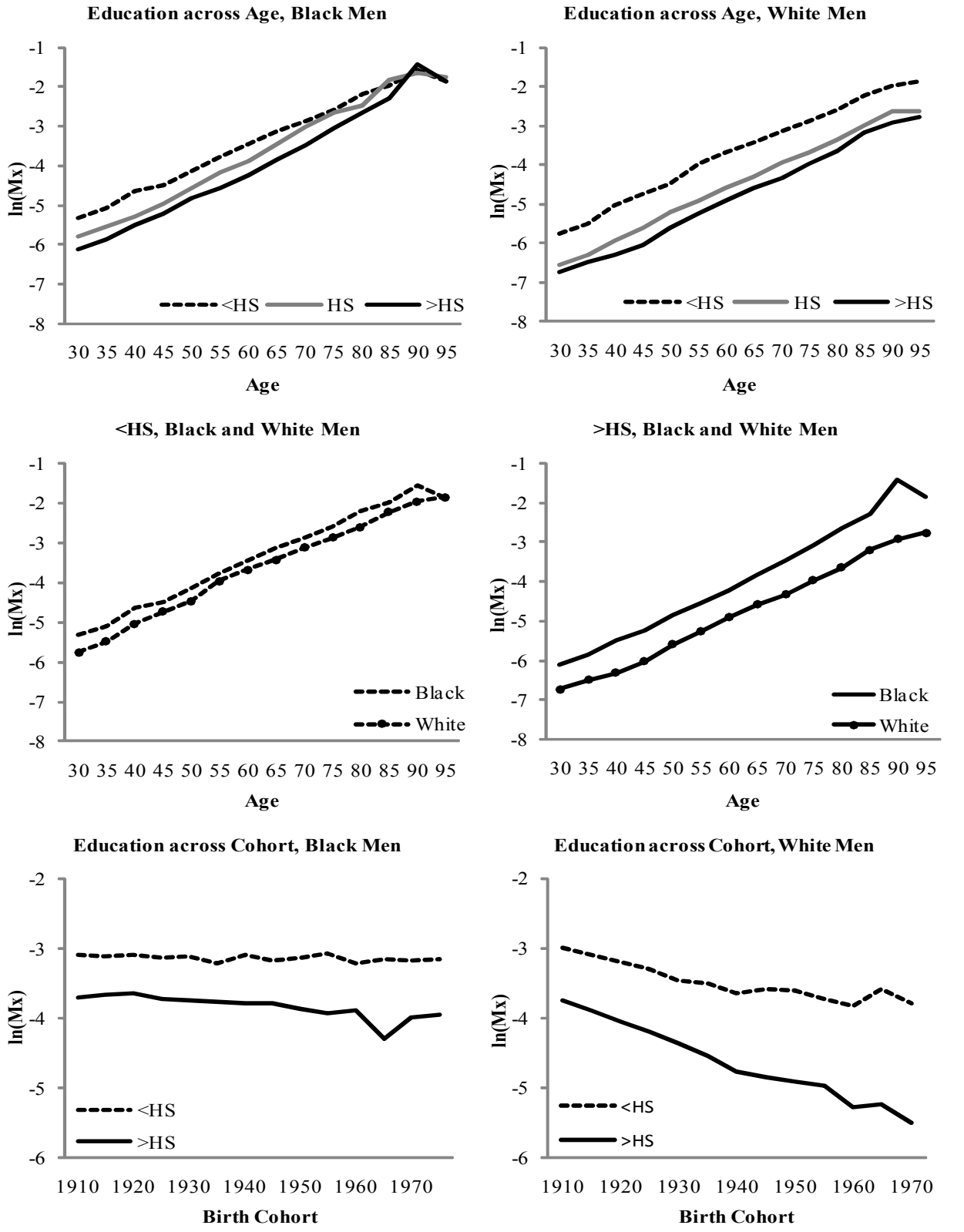


Figure 4. Effects of Education on U.S. Black and White Women's Adult Mortality, 1986-2006

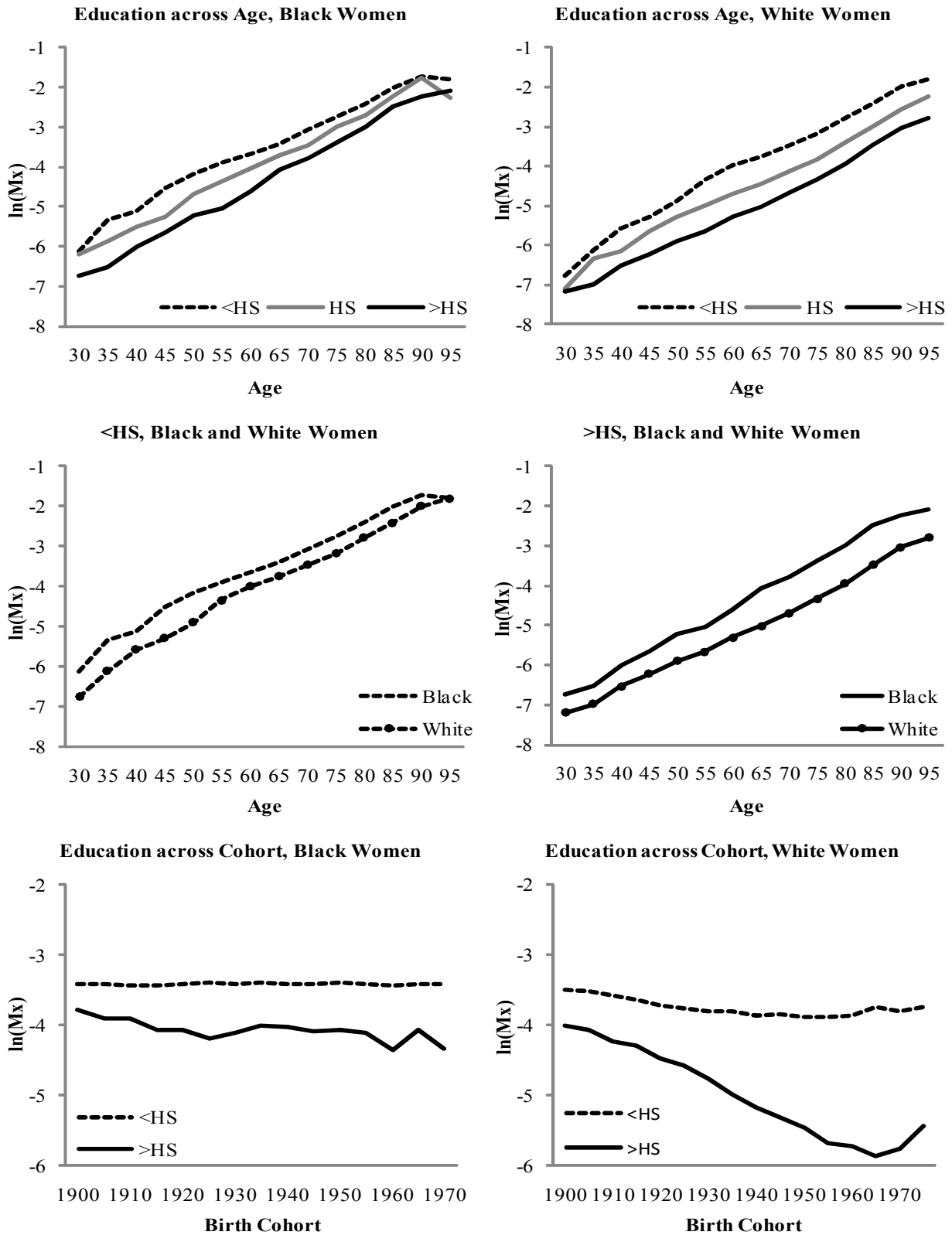
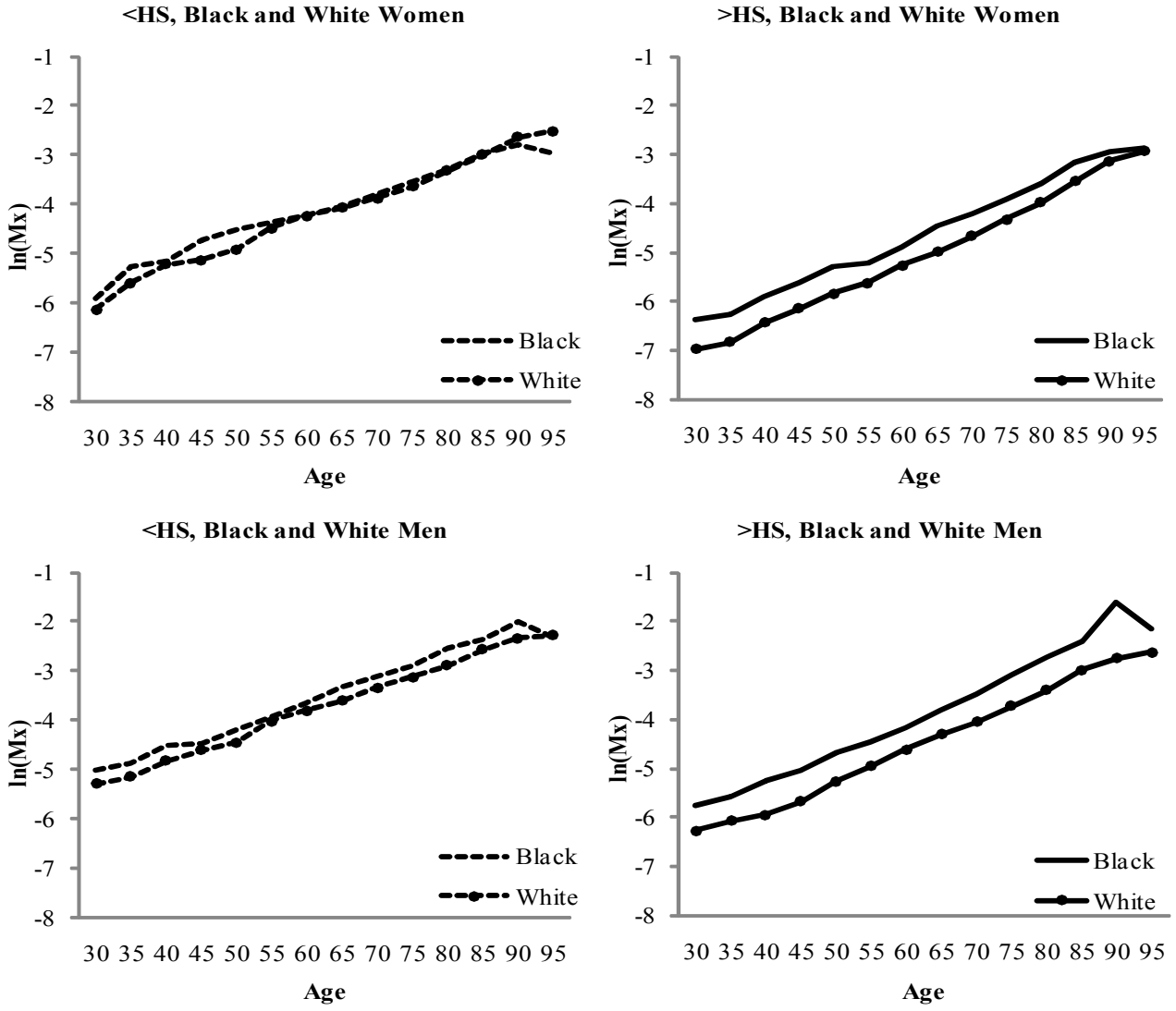


Figure 5. Effects of Education on U.S. Black and White Women's & Men's Adult Mortality, 1986-2006, Controlling for Effects of Cohort-level Variation in Early-life Conditions.



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