Long-Term Trends in Adult Mortality for U.S. Blacks and Whites: An Examination of Period- and Cohort-Based Changes

Ryan K. Masters
Robert A. Hummer
Daniel A. Powers
Audrey Beck
Shih-Fan Lin
Brian Karl Finch

December 2013
Long-Term Trends in Adult Mortality for U.S. Blacks and Whites: An Examination of Period- and Cohort-Based Changes

Ryan K. Masters, University of Colorado at Boulder
Robert A. Hummer, University of Texas at Austin
Daniel A. Powers, University of Texas at Austin
Audrey Beck, San Diego State University
Shih-Fan Lin, San Diego State University
Brian Karl Finch, University of Southern California

1 This research was supported by grant 1 R01MD00425 from the National Institute for Minority and Health Disparities (principal investigator Brian K. Finch), the Robert Wood Johnson Foundation Health and Society Scholars Program, the MacArthur Foundation Network on an Aging Society (Jack Rowe, Director), and infrastructure grant 5 R24HD042849 from the Eunice Kennedy Shriver National Institute of Child Health and Human Development to the Population Research Center at the University of Texas at Austin. We thank the National Center for Health Statistics for making the data that we use here publicly available and the reviewers and Editor for their very helpful comments and suggestions.
Abstract

Black-white differences in U.S. adult mortality narrowed over the past five decades, but it’s unclear whether this narrowing unfolded on a period or cohort basis. The distinction has important implications for understanding the socioeconomic, public health, lifestyle, and medical mechanisms responsible for this narrowing. We use data from 1959 to 2009 and age-period-cohort models to examine period- and cohort-based changes in adult mortality for U.S. blacks and whites. We do so for all-cause mortality among persons aged 15-74, as well as for several underlying causes of death more pertinent for specific age groups. We find clear patterns of cohort-based reductions in mortality for both black and white men and women. Recent cohort-based reductions in heart disease, stroke, lung cancer, female breast cancer, and other cancer mortality have been substantial, and, save for breast cancer, especially pronounced for blacks. Period-based changes have also occurred and are especially pronounced for some causes of death. Period-based reductions in blacks’ and whites’ heart disease and stroke mortality are particularly impressive, as are recent period-based reductions in young men’s and women’s mortality from infectious diseases and homicide. These recent period changes are more pronounced among blacks. The substantial cohort-based trends in chronic disease mortality and recent period-based reductions for some causes of death suggest continuing slow closure of the black-white mortality gap. However, we also uncover troubling signs of recent cohort-based increases in heart disease mortality for both blacks and whites.
Introduction

The most recent annual report on U.S. mortality patterns and trends from the National Center for Health Statistics (NCHS) demonstrated the narrowest disparity in life expectancy at birth between blacks and whites ever reported (Murphy et al. 2013). Specifically, this report highlighted that life expectancy at birth among blacks increased to 75.1 years in 2010 while life expectancy at birth among whites increased to 78.9. The resulting 3.8-year disparity continues a long term narrowing trend. Indeed, the black-white life expectancy disparity was wider than seven years in 1993 and was estimated to be as wide as 14 or 15 years in 1900 (Arias, 2010; Harper et al. 2007).

While the trend indicates continued progress toward closing of the black-white mortality disparity, these recent data also demonstrate that the country fell well short of the 2010 goal of eliminating health disparities across different segments of the population (Healthy People 2010, 2011). Thus, the recent report on black and white life expectancy, showing decreasing mortality rates and the smallest racial disparity in U.S. history, continues to leave much room for improvement and does not bode well for achieving the Healthy People 2020 goal to “achieve health equity, eliminate disparities, and improve the health of all groups” (Healthy People 2020, 2011). Moreover, there is no guarantee that the black-white life expectancy disparity will continue to decrease; for example, there was a sizable increase in the black-white life expectancy gap between 1983 and 1993 (Harper et al. 2007; Kochanek et al. 1994). Also, recent findings reveal that life expectancy is decreasing in some regions of the United States (Kindig and Chen 2013) and among some segments of the U.S. population, particularly low-educated white men and women (Montez and Zajacova 2013; Olshansky et al. 2012). The latter point suggests that some closure of the black-white gap in US life expectancy partially reflects the relative and/or absolute declining longevity among some segments of the US white population.
This paper examines trends in U.S. black and white men’s and women’s adult mortality as they unfolded between 1959 and 2009, with a focus on examining both period-based and cohort-based trends. A vast majority of studies examining U.S. mortality change, including those focused on black-white disparities (e.g., Harper et al. 2007, 2012; Levine et al. 2001, 2010; Orsi et al. 2010; Satcher et al. 2005), do so using a strictly period-based approach. In other words, rate changes are examined on a year-to-year basis, which is a narrow definition of demographic time change. As a result, changes in those year-to-year rates are often thought to be prominently or solely influenced by factors operating within specific time periods that similarly affect all birth cohorts. However, evidence from recent studies suggests that substantial amounts of U.S. adult mortality rate changes over the past several decades reflect cohort-based variation in mortality risk (Masters 2012; Masters et al. 2012; Preston and Wang 2006; Reither, Yang, and Olshansky 2011; Yang 2008). Understanding whether mortality changes are operating on a period basis, a cohort basis, or both, has profound implications for both theories of mortality change and policies designed to lower mortality rates and close disparities between population subgroups. The major contribution of this paper, then, is to more clearly understand adult mortality trends for blacks and whites over the past 50 years, with specific attention given to how the changes are unfolding across the different dimensions of demographic time. A more specific understanding of black and white trends in adult mortality—that is, among specific birth cohorts, within specific periods of time, and/or within specific age groups and causes of death—is imperative toward our understanding of the overall black-white disparity in mortality and in speculating whether or not the gap is likely to further close.

**Literature Review**
A number of recent studies have focused on trends in black-white adult mortality disparities. Using similarly structured data sets on middle-aged American males in 1900-1914 and 1992-2006, Sloan et al. (2010) showed that the relative black-white mortality disparity in this age group was largely stagnant between these two periods, in spite of sizable absolute rate declines for each group. Levine et al. (2001) analyzed age-adjusted mortality for blacks and whites from 1933 to 1999 and found there had been no sustained decrease in relative black-white mortality differences since 1945. Later trend studies of U.S. mortality rates between 1960 and 2000 also demonstrated very little change in relative black-white mortality differences (Elo and Drevenstedt, 2004; Satcher et al. 2005). Likewise, a recent analysis of U.S. vital statistics data from 1990 to 2006 found that relative black-white disparities in mortality did not significantly decline for the five leading causes of death (heart disease, cancer, stroke, chronic lower respiratory disease, accidents), even in the context of impressive age-adjusted mortality declines for most of them over this time period (Keppel et al. 2010).

Other studies, however, provide evidence that the overall black-white mortality disparity, as measured by absolute differences, is narrowing. Harper et al. (2007) documented a narrowing of the black-white life expectancy disparity by one year among women and two years among men between 1993 and 2003. They identified more pronounced declines among blacks in mortality due to homicide, HIV, unintentional injuries, and heart disease (for women only) as the major contributors to the declining gap in life expectancy (see also Orsi et al. 2010). DeLancey et al. (2008) also identified more rapid decreases in smoking-related cancers among black males since 1990 as contributing to the decreasing overall gap. A follow-up study by Harper et al. (2012) documented a continued decline in the black-white life expectancy disparity (2003-2008) that was largely driven by more substantial declines in heart disease and HIV mortality for
blacks, and increases in mortality for white males due to unintentional injuries. Similarly, Macinko and Elo (2009) reported a narrowing black-white disparity in working-aged mortality since the late 1980s that was especially pronounced among men. All of the above studies, though, focused on period-based changes in mortality rather than examining both period-based and cohort-based changes.

**Period and Cohort Changes in U.S. Adult Mortality by Race**

Should we expect U.S. adult mortality trends to be driven by period-based changes, cohort-based changes, or both? Empirically, Yang (2008) demonstrated that changes in U.S. adult all-cause and major chronic disease mortality between 1960 and 1999 unfolded almost exclusively on a cohort, rather than a period, basis; this pattern was consistent for men and women. That is, as more recent birth cohorts of adults passed through the 1960 to 1999 time period, their mortality rates were significantly lower in comparison to previous birth cohorts passing through the same time period, net of both age- and period-based mortality variation. In contrast, net of cohort- and age-based variation, Yang found only very modest reductions in period-based mortality rates across the 1960-1999 time periods.

Theoretically, individuals live their lives in particular birth cohorts that are structured by unique opportunities, constraints, and normative contexts (Carlson 2008; Crimmins and Finch 2006; Easterlin 1980; Pampel 2005; Riley 1987; Ryder 1965). The differential rates by which population subgroups die during adulthood should, at least in part, reflect the different sociohistorical, lifestyle, and biological contexts in which cohorts have lived their lives. In this regard, researchers have emphasized the importance of cohorts’ disparate cumulative exposures to health-enhancing and health-jeopardizing living conditions, diseases, behavioral risk factors,
and medical advances across their respective life courses. Preston and Wang (2006), for example, showed that fluctuation in the U.S. sex differential in adult mortality between 1948 and 2003 was highly sensitive to cohort-based cigarette smoking patterns of men and women. Susser (1982: 35) demonstrated that between 1900 and 1977, “successive generations [in England and Wales] had carried their own risk of peptic ulcer mortality through life,” and Frost (1940: 96) concluded that deaths from tuberculosis during old age were “the residuals of higher [tuberculosis] rates in earlier life.” More recently, Masters et al. (2012) examined education-specific period- and cohort- trends in U.S. adult mortality between 1986 and 2006,. They found substantial declines in adult mortality for recent cohorts of highly educated men and women, while mortality rates among less educated adults in more recent birth cohorts were similar to those of their earlier cohort counterparts. As a result, there are now larger educational gradients in U.S. adult mortality among recent birth cohorts relative to cohorts born in the early portion of the 20th century.

Turning to race differences in cohort experiences, late 19th and early 20th century black birth cohorts were exceptionally disadvantaged compared to white birth cohorts. In their comprehensive study of social change in America throughout the 20th century, Fischer and Hout (2006: 55-56) wrote that, “African-origin Americans began the twentieth century locked in rural isolation, hemmed in by legal discrimination in the South, and held back by the legacies of slavery… their poverty was deeper, their lack of education and industrial skills more glaring, and the prejudice and discrimination they faced far more severe than whites.” Writing at the time, Du Bois (1899) documented enormous race differences in health in his classic study of Philadelphia’s Seventh Ward, giving particular attention to the deplorable housing, sanitation, and health care contexts in which urban blacks tended to live. Later, Preston and Haines (1991)
comprehensively documented the context of child health in American society around 1900. While sanitation was poor, health knowledge was low compared to today’s standards, and medical care was rudimentary for all Americans at the time, such conditions were especially harsh among blacks. Statistically, Preston and Haines (1991) estimated 56 percent higher child mortality (ages <5) rates for blacks compared to whites at the time, concluding that race was, “the single most important variable in predicting child mortality levels” (94). They explained, “race was a caste-like status in 1900, and the degraded social and economic circumstances of blacks, who had virtually no chance of entering the mainstream of American life, is undoubtedly reflected in their exceptionally high mortality” (Preston and Haines 1991: 210).

With a few notable exceptions, what many contemporary analysts of adult mortality overlook is that the racially specific contexts of black and white cohorts’ infancy, childhood, and adolescence play out in the life course health of black and white adults for many decades afterward (Collins 2011; Crimmins, Hayward, and Seeman 2005; Geronimus et al. 2006; Hayward and Gorman 2004; Hayward et al. 2000; Masters 2012). A growing body of research suggests that childhood socioeconomic circumstances are significantly associated with later life mortality risk (see Galobardes, Lynch and Davey Smith [2004, 2008]; Godfrey and Hanson [2009]; and Montez and Hayward [2011] for systematic reviews). Focusing exclusively on African Americans, Preston et al. (1998) showed that relatively advantaged childhood conditions (e.g., farm background, literate parents, two parent households) in 1900-1910 were strongly predictive of survival up through age 85. The corollary, of course, is that exceptionally poor early life conditions for African Americans in the early 20th century were strongly predictive of mortality (well) before age 85. Warner and Hayward (2006) provide additional evidence of such long-term life course effects by showing that the recent black-white gap in male adult mortality
is partially accounted for by racially disparate childhood socioeconomic and health conditions that characterized the early 1900s. This body of work makes it clear that cohort-specific early life conditions likely exhibit important associations with the mortality prospects of American adults for many decades into the future (Montez and Hayward 2011). Moreover, scholars are growing increasingly attentive to the fact that black and white cohorts endured significantly different sociohistorical conditions, which, in turn, have shaped significantly different life course health trajectories for these groups (Colen 2011; Crimmins et al. 2005; Geronimus 1992; Jackson et al. 2011; Masters et al. 2012).

While black birth cohorts of the 1920s, 1930s, and early 1940s were born into somewhat more favorable social, economic, and health contexts than their counterparts born in the first two decades of the 20th century, gains among white birth cohorts of the late 1920s to mid-1940s—the “lucky few” (Carlson 2008)—were especially substantial along a number of dimensions. In part, Carlson argues, white gains among this set of birth cohorts were due to their small demographic size relative to other birth cohorts, the sustained economic boom of the post-war era as these cohorts came of adult age, and their dominant position in the American racial hierarchy. Furthermore, major public health efforts across these decades dramatically reduced infections and improved nutrition, which, in turn, lowered maternal, infant, and child mortality (Cutler and Miller 2005; Floud et al. 2011; Manton, Stallard, and Corder 1997). And while black birth cohorts of the 1920s, 1930s, and early 1940s benefited in some ways from the same demographic, economic, and public health forces, including access to better employment prospects during the Great Migration (Jaynes 2012), they also “grew up just a little too early to enjoy ‘lucky’ changes still in their future” (Carlson 2008: 164). By this, Carlson alludes to the institutionalized and legislated racism that prohibited the black population from fully benefiting
from the general social, economic, and health-related advances being made at the time. Such disparate conditions early in life, set in motion long-term life course processes that likely affected the subsequent mortality risk of black and white members of these cohorts in starkly different ways. Indeed, cohort-based reductions in US white mortality have been shown to be greater than cohort-based reductions in US black mortality across the first half of the twentieth century (Masters 2012).

The post-World War II era in the United States finally resulted in the slow dismantling of legal forms of segregation, the eventual passage of civil rights legislation, and the enactment of Medicaid, Medicare, and other Great Society legislation such as the 1964 Food Stamp Act that helped to usher in greater legal, social, and health care equality between black and white Americans. The significance of these changes on the immediate health of black Americans has been shown to be quite dramatic. For instance, Almond and Chay (2006) showed that black infant mortality rates in the South significantly dropped during the late 1960s. Other evidence suggests that all black Americans – but especially black women – experienced reduced mortality during the late-1960s (Kaplan, Ranjit, and Burgard 2008). The effect of such momentous changes in the legal, social, and health environment in the United States likely resulted in a “period shock” on black American’s adult mortality beginning in the mid-1960s.

But more than just short-term effects, an improved legal environment, increased educational and economic opportunities, and enhanced access to medical care and technologies are likely to have significant long-term health implications for black cohorts growing up in post-World War II America. Indeed, the greatly expanded social capacity for health that developed across the early part of the twentieth century in the United States finally began to be extended to the black population (Abramowitz 1995; Caldwell 1993; Easterlin 1999). The importance of
such factors is summarized by Preston and Haines (1991), who paraphrase Winslow’s point that “in assigning responsibility for rapid health progress, the possibility of widespread social organization to combat disease could almost be placed alongside the discovery of the germ theory in importance” (207). Enhanced access to mainstream social and economic opportunities and medical care for blacks—i.e., a weaker American color line—may be particularly reflected in sizable reductions in chronic disease mortality for black cohorts growing up in post-World War II America. This is because the long-term benefits of higher levels of education, decent wages, and access to basic and essential medical care take hold and allow individuals to better prevent and manage the chronic diseases that have dominated the structure of American mortality patterns since the epidemiologic transition (Olshansky and Ault 1984). Black cohorts born late enough in history to spend their infancy, childhood, and early adulthoods in post-World War II America should have especially benefitted from these changes. Consequently, we hypothesize that:

**H1:** Adult mortality rates over the past 50 years fell significantly faster among black Americans born after World War II than for black Americans born before World War II.

Consistent with this hypothesis, the faster cohort-based reductions for blacks growing up in post-World War II America should be most prominent for causes of death related to chronic diseases. Indeed, the slow closure of the black-white mortality gap across cohorts should be largely driven by more recent black cohorts’ significantly decreasing mortality rates from the major chronic conditions of adult mortality: heart disease, stroke, and cancers. Thus, we hypothesize:
H2: Black-white differences in mortality rates from heart disease, stroke, and cancer have significantly narrowed across post-World War II birth cohorts.

That said, there remain very wide black-white differences among recent birth cohorts in early life health (e.g., low birth weight), childhood poverty, and educational opportunities and attainment, reflecting continued systemic social, economic, and health care discrimination and disadvantages that affect the African American population (Colen 2011; Hummer and Chinn 2011; Williams et al. 2010; Williams and Braboy Jackson 2005). These patterns suggest that even very recent birth cohorts of blacks face health and mortality disadvantages relative to their white peers. Thus, historical mortality gains made by black Americans, while likely rapid among post-World War II cohorts and likely narrowing the black-white mortality gap in chronic diseases, have not been large enough to fully close the black-white gap in mortality.

At the same time, the best work that has decomposed recent black-white mortality trends has found that the causes of death responsible for the recent narrowing in the race mortality gap are related to policy interventions aimed at curtailing external threats (such as homicide and accidents) and improved access/quality of specific medical care and technological innovations (such as HIV/AIDS) (Macinko and Elo 2009). Such factors are largely concentrated among younger age groups and may be quite responsive to period effects (e.g., development of highly-active antiretroviral therapy to fight HIV/AIDS, improved emergency response times to save accident victims, and more intense policing in high crime areas to reduce homicides) rather than longer-term life course processes that are specific to particular birth cohorts. Thus, there is also reason to believe that some mortality changes for blacks and whites, and particularly recent reductions among blacks, exhibit strong period-based variation. While much of the variation in
chronic disease mortality is likely associated with cohort-based factors, we believe period-based variation is most prominent among causes of death affecting younger age groups. Consequently, we hypothesize:

H3: Significant period-based reductions in black and white mortality rates occurred in recent decades for deaths due to infectious diseases, homicide and legal intervention, and accidents. These reductions are likely more pronounced among blacks than whites.

Data and Methods

Data

Our data source is official U.S. mortality records: death certificate based counts of death in the numerator and Census based counts or estimates in the denominator. Denominator estimates of the age-specific, mid-year population (July 1st) for blacks and whites were used to approximate person-years lived in each calendar year from 1959 through 2009 and were obtained from three official data sources. Yearly counts of death by five-year age group were obtained from annual National Center for Health Statistics (NCHS) Multiple Cause of Death Files made publicly available by the Interuniversity Consortium for Political and Social Research (ICPSR 2012).²

We focus our analysis on all blacks and all whites, regardless of Hispanic ethnicity, and exclude other race groups because of their smaller size over the long time frame being considered. The data used in our analyses are available only for the US “white” and “non-white” populations for years 1959 to 1968 and for the US “white” and “black” populations for years 1969 through 2009. Because Hispanic death counts were unavailable in some states between
1969 and the late 1980s, and because it is well known that Hispanic deaths are undercounted in Vital Statistics data (Arias 2011), we focus our comparison on blacks and whites. It is important to note that Hispanics comprised an increasing share of the black and (especially) the white populations between 1959 and 2009. However, because Hispanic adult mortality rates are quite similar to those of whites (Arias 2010), the omission of ethnicity in our rate tabulations should have little impact on mortality trends among all whites and all blacks (regardless of Hispanic ethnicity) over the time period under consideration. This is especially true for the older birth cohorts that contribute the disproportionate amount of deaths in these data.

We estimated five-year age-specific mortality rates for US blacks and whites for all-causes of death and deaths classified as being caused by heart disease, stroke, lung cancer, breast cancer (for women), all other cancers, homicide and legal intervention, accidents, infectious diseases, and residual causes. For deaths occurring in years 1959-1967, we classified cause of death in accordance with the 7th Revision of the International Classification of Diseases (ICD-7); for deaths occurring in years 1968-1978 we classified cause of death in accordance with the ICD-8; for deaths occurring in years 1979-1998 we classified cause of death in accordance with the ICD-9; and for deaths occurring in years 1999-2009 we classified cause of death in accordance with the ICD-10. Unlike some causes of death for which comparability across ICD revisions is difficult (e.g., septicemia, influenza and pneumonia, Alzheimer’s disease), the causes of death we investigate have been found to be quite stable across these versions of the ICD (Anderson et al. 2001; Klebba and Scott 1980).³

Methods
Data were arranged in 12 five-year age groupings \((A)\) ranging from 15-19 to 70-74 and mortality rates were estimated across 11 five-year periods \((P)\) spanning 1955-1959 to 2005-2009. Age was capped at 74 years for two primary reasons. First, research has shown that age misreports among the older black population significantly biases estimates of old-age mortality (Preston et al. 1996; Preston and Elo 2006). As such, we limit the age range to < 75 years to safeguard against these age-sensitive biases.\(^4\) Second, evidence suggests that black-white differences in adult mortality risk are greatest in middle and late-middle ages and smallest at older ages (Hummer and Chinn 2011; Stewart 2006). We therefore focus our analyses on relatively early deaths because we are interested in period- and cohort-based trends in mortality rates where the largest race differences exist.

Ten-year birth cohorts \((C)\) were computed as direct linear combinations of the five-year time periods and five-year age groups, and range from birth cohort 1885-1895 to 1990-2000. Due to the unbalanced design of the data, the 1955-1959 period is composed only of the 1959 wave and the 1990-2000 cohort contains persons born between years 1990 and 1994 only.\(^5\) The data structure for the analysis is visible in Table 1, where age groups are depicted as rows, period time is depicted in the columns, and birth cohorts are represented in the diagonals. The values presented in Table 1 are estimates of U.S. black women’s five-year age-specific mortality rates.

**Table 1 about Here**

The data structure fits the assumptions of APC analysis in that birth cohorts are linearly dependent on time period and age group: \(C = P - A\). To simultaneously estimate the age, period, and cohort changes in U.S. black and white men’s and women’s adult mortality rates between 1959 and 2009 we used Powers’s (2012) Stata module `ie_rate`, which provides flexible extensions of Yang et al.’s (2004) intrinsic estimator (IE). Logged counts of deaths within each
APC cell are assumed to follow a Poisson distribution and offsetting the logged aggregated exposure time lived across each cell estimates a rate model, specified as:

$$\log E(r_{ij}) = \log E\left(\frac{d_{ij}}{n_{ij}}\right) = \beta_0 + \beta_i^A + \beta_j^P + \beta_k^C, \quad (1)$$

where $\log E(r_{ij})$ is the logarithm of the expected mortality rate based on $d_{ij}$ deaths and exposure $n_{ij}$ pertaining to cell $ij$ of the cross-tabulated data in Table 1. Effects associated with age interval $i$ (for $i = 1, \ldots, I$ age groups) and with period $j$ (for $j = 1, \ldots, J$ periods) are captured by $\beta_i^A$ and $\beta_j^P$, respectively. $\beta_k^C$ denotes the $k$th diagonal of birth cohort effect (for $k = 1, \ldots, I + J - 1$ birth cohorts), where the index $k = I - i + j$. In these data, $I = 12$ and $J = 11$ for $N = 132$ age by period cells occupied by 22 birth cohorts.

We use Powers’s (2012) `ie_rate` Stata module to model the APC terms as centered effects:

$$E(r_{ij}) = \tau_0 \tau_i^A \tau_j^P \tau_k^C, \quad (2)$$

where $\prod_i \tau_i^A = \prod_j \tau_j^P = \prod_k \tau_k^C = 1$. The $\tau$ parameters in the APC model are multiplicative effects whose product is 1 over the levels of each factor. Under this normalization, the constant term $\tau_0$ is the scaled grand mean of all five-year age-specific mortality rates (see Yang, Fu, and Land [2004], Yang et al. [2008], and Powers [2013] for descriptions of the underlying vector geometry and discussions of the estimation techniques). The models estimating all-cause mortality include all age groups 15-19 to 70-74. The models estimating chronic disease mortality (and residual causes) are limited to age groups 35-39 to 70-74, and the models estimating mortality from external causes of death and infectious causes are limited to age groups 15-19 to 30-34. All scripts, data, and output are available upon request.
Results

Trends in All-cause Mortality

Online Table S2 contains estimates of five-year age, five-year period, and 10-year cohort coefficients on all-cause mortality rates for U.S. black and white men and women aged 15-74 between 1959 and 2009. Figure 1 presents graphed estimates of deaths per 100,000 persons by five-year age, five-year period, and 10-year cohort separately, holding constant the variation associated with the other two temporal dimensions. For example, the age patterns of men’s and women’s mortality presented in the top panels of Figure 1 are estimated at Cohort 1920-1930 and Period 1975-1979 so that the observed patterns entirely reflect age-based variation in mortality. Thus, the graphical depictions in all figures are used only to isolate and present the patterns of each temporal dimension, and are not to be interpreted as representative of the actual mortality rates experienced by a specific cohort or in a specific time period.

Figure 1 about Here

When comparing the period- and cohort-based trends illustrated in Figure 1 we see significantly greater reductions in black and white men’s and women’s adult mortality rates across cohorts than across time periods. This evidence is consistent with Yang’s (2008) findings, in that black and white men’s and women’s all-cause mortality trends over the past 50 years were more strongly associated with cohort-based changes than with period-based changes. Also apparent in Figure 1 is evidence consistent with previous work showing reductions in adult mortality rates between 1959 and 2009 for cohorts born before the middle of the 20th century were greater among US whites than among US blacks (Masters 2012). These differences between black and white cohorts’ mortality are clearly seen in Figure 2, which graphically
depicts the differences as relative rate ratios. Holding constant age- (60-64) and period-based (1975-1979) variation in mortality, the relative black-white difference in men’s adult mortality for cohorts born before the 20th century was about 1.25. The relative black-white difference in men’s mortality grew significantly larger across subsequent birth cohorts, peaking at about 1.75 for men born in the late 1930s and early 1940s. The black-white cohort-based difference in women’s adult mortality was also about 1.25 for cohorts born before the 20th century. Cohort-based race differences in women’s mortality grew significantly greater at the turn of the century and thereafter steadily remained high across subsequent cohorts born before the middle of the 20th century, before declining among post World War II cohorts.

Figure 2 about Here

Figure 1 also presents evidence consistent with previous work showing dramatic and substantive period-based reductions in black men’s and women’s adult mortality during the 1965-1969 to 1975-1979 time periods (Almond and Chay 2006; Kaplan et al. 2008). Reductions in black women’s mortality rates over that 15-year period are particularly impressive and rapidly closed black-white differences in women’s period-based mortality patterns across this time. Reductions in black men’s mortality rates, conversely, were short-lived, as period-based changes subsequently increased black men’s mortality across the 1980-1984 to 1990-1994 time periods, before resuming their decline during the late 1990s and 2000s.

Results depicted in Figures 1 and 2 also provide some evidence consistent with our first hypothesis. For example, cohort-based reductions in black men’s mortality rates were substantively large across those cohorts born after World War II. These rapid cohort-based reductions in black men’s mortality – coupled with stalling and then rising cohort-based changes in white men’s mortality for cohorts born in the 1950s and 1960s – resulted in rapid cohort-based
closure of the black-white gap in men’s mortality. This is most apparent in Figure 2, wherein we see cohort-based relative rate ratios between black and white men’s mortality dropping from 1.75 to 1.25 across the 1945 to 1965 birth cohorts. However, cohort-based increases for black men born during the 1970s and steady cohort-based reductions in white men’s mortality quickly reversed the cohort-based closure of the black-white mortality gap and increased differences. Behind these recent cohort-based trends are the mortality experiences of younger aged (i.e., 15-19 to 30-34) black and white men, details of which will be discussed below. Evidence supporting our first hypothesis is also found in the relatively fast cohort-based reductions in black women’s mortality for cohorts born after World War II. These reductions, coupled with slowing rates of cohort-based reductions in white women’s mortality, resulted in a cohort-based narrowing of the black-white gap in women’s mortality across cohorts 1955-1964 to 1985-1994.

**Figures 3a and 3b about Here**

*Trends in Major Chronic Disease Mortality*

Figures 3a and 3b present period- and cohort-based changes in black and white women’s mortality rates from major chronic diseases, and Figures 4a and 4b presents respective changes for black and white men. In all figures, save breast cancer mortality, we find evidence supporting our second hypothesis. Cohort-based reductions in black men’s and women’s all-cause mortality rates that spurred a cohort-based narrowing of black-white differences in U.S. adult all-cause mortality were largely driven by cohort-based reductions in deaths from chronic diseases. Indeed, the cohort-based closure of black-white differences in mortality is predominantly being driven by black and white men’s and women’s cohort-based trends in heart disease, stroke, and non-lung/non-breast cancers, and also for men, lung-cancer mortality.

**Figures 4a and 4b about Here**
Cohort-based reductions in U.S. adult mortality from heart disease and stroke between 1959 and 2009 were substantial for all population subgroups born between 1900 and the 1950s (Figure 3a for women and Figure 4a for men). Among more recent cohorts, however, we find evidence consistent with past findings of cohort-based stalling (for black women) and cohort-based increases (for black men and whites) in heart disease mortality rates (Reither et al. 2011; Yang 2008). A consistent pattern of cohort-based stalling in stroke-related mortality is also evident, although substantial reductions are seen in the most recent cohorts of black men and women. Furthermore, the cohort-based changes in heart disease and stroke mortality are coupled with impressive and sustained period-based reductions in heart disease mortality for white men and women. Large period-reductions in black men’s and women’s heart disease mortality occurred during the 1960s (especially for black women) and, following a stalling across the 1970s, further period-based reductions in heart disease mortality continued for the next three decades. These period-based reductions slightly narrowed the black-white period gap in heart disease mortality among women, but not among men. A narrowing period-based black-white gap in stroke mortality is also observed among both men and women.

Much of the black-white narrowing in U.S. adult mortality reflects impressive recent cohort-based reductions in black men’s and women’s mortality from non-lung/non-breast (“other”) cancers. Significant cohort-based reductions in other cancer mortality has also occurred in the white population, but the rates of cohort-based reductions among recent black cohorts outpaced those of their white counterparts and have greatly narrowed black-white cohort-based differences in other cancer mortality. As seen in Figure 3a, cohort-based racial differences in women’s other cancer mortality were quite large and stagnant for cohorts born between 1900 and 1930. But across cohorts 1930-1940 to 1965-1975, Figure 3a shows a rapid cohort-based
narrowing of black-white differences in other cancer mortality between 1959 and 2009. Also, we observe significant period-based reductions in women’s mortality from other cancers across the 1950s and 1960s, followed by period-based stalling among white women and small, but steady increases among black women. Regarding trends in black and white men’s non-lung (other) cancer mortality, we see significant recent cohort-based reductions among all men in Figure 4b. Between 1959 and 2009, white men experienced steady cohort-based reductions in mortality from other cancers; black men, following cohort-based increases and stalling across the 1885-1895 and 1925-1935 birth cohorts, experienced remarkable cohort-based decreases across recent cohorts. These trends among recent cohorts have significantly narrowed the black-white gap in men’s other cancer mortality. Conversely, period-based changes in men’s other cancer mortality have been more sobering. On the one hand, no significant period-based variation is seen in white men’s other cancer mortality. On the other hand, a steady period-based increase in black men’s mortality from other cancers is observed from the 1955-1959 period to the 1990-1994 period, significantly widening the black-white gap across these time periods. However, the small but significant reversal of period-based trends in black men’s other cancer mortality across the 1995-1999 through 2005-2009 time periods is a promising sign of improvement.

Figure 3b reveals that black and white women’s cohort-based trends in lung cancer mortality are nearly identical and strongly follow cohort trends in cigarette smoking (Preston and Wang 2006; Wang and Preston 2009). Further, we find evidence of significant increases in black-white mortality differences across time periods. The general trend of increasing period-based changes largely reflects compositional age changes across time periods, in which the birth cohorts with higher smoking rates (1915-1935) are aging into older age groups that are most susceptible to lung cancer mortality. The widening period-based black-white differences may
reflect disparate cancer treatments between blacks and whites, race differences in age of cancer
detection/diagnosis, or both (Terhanifar et al. 2009). Figure 4b shows those cohort-based
changes in black and white men’s lung cancer mortality between 1959 and 2009 also followed
cohort trends in smoking. Yet, unlike cohort trends among women we find significant race
differences among men, with much greater cohort variation in black men’s lung cancer mortality
than in white men’s lung cancer mortality. Black-white differences in men’s lung cancer
mortality between 1959 and 2009 grew significantly wider across birth cohorts born between
1885 and 1930. Thereafter, however, cohort-based reductions in black men’s lung cancer
mortality outpaced respective cohort-based reductions among white men, thereby rapidly
narrowing the black-white gap in lung cancer mortality across recent cohorts. Also, similar to
period-based trends in men’s other cancer mortality, we also uncover that black-white period-
based differences in men’s lung cancer mortality grew significantly wider across periods 1955-
1959 to 1985-1990. Some period-based narrowing of the black-white gap is occurring across
more recent time periods as black men’s period-based trends in lung cancer mortality are
decreasing.

Finally, the only case in which the racial disparity in chronic disease mortality is
widening across both periods and cohorts is for women’s breast cancer (Figure 3b). Between
1959 and 2009, both period- and cohort-based patterns in black women’s breast cancer mortality
trended worse than the respective patterns in white women’s breast cancer mortality. Cohort-
based reductions in breast cancer mortality for both black and white women are impressive
across cohorts born after the 1920s, but the rate of cohort-based reductions among black women
is slower than the rate among white women. Even more significantly affecting the black-white
gap in women’s breast cancer mortality are black-white disparities in recent period-based
changes. There was very little period-based variation in breast cancer mortality prior to the widespread use of screening technologies (e.g., mammography, breast MRI), surgeries, and considerable public health campaigns raising women’s awareness of breast cancer (e.g., Susan G. Komen for the Cure [1982]). Such factors likely spurred the rapid period-based reductions in white women’s breast cancer mortality rates since 1985 (Menashe et al. 2009). Conversely, we see significant period-based increases in black women’s breast cancer mortality across the late 1970s and into the early 1990s, and only very recently did breast cancer mortality rates for black women stabilize and begin to fall. Such differences possibly reflected the onset and spread of the U.S. obesity epidemic, which has disproportionately affected black women (Reither, Hauser, and Yang 2009). Indeed, obesity has been shown to be a leading risk factor in the onset of breast cancer (Brown and Simpson 2009) and some research suggests obesity significantly decreases breast cancer screening behavior (Cohen et al. 2008). The disparities also likely reflect differences in screening policies, access, and practice patterns between black and white women in the United States (Menashe et al. 2009). These large and growing racial disparities in both cohort- and period-based trends of a preventable cancer are of serious public health concern.

*Trends in US Black and White Cause-Specific Mortality at Younger Ages*

**Figure 5 about Here**

Period- and cohort-based trends in U.S. black and white young women’s mortality from homicides, accidents, and infectious diseases are presented in Figure 5, and trends among U.S. black and white young men are presented in Figure 6. Stark differences exist between men and women in levels of mortality from these causes of death. Indeed, the mortality rates among young men from these causes of death are about three to four times greater than among young
Cohort-based increases in infectious disease mortality were common for black and white men and women born between 1950 and 1965, reflecting these cohorts’ unique experiences during the rapid period-based increases in infectious disease mortality of the early 1980s and 1990s. Overall trends were largely driven by period-based changes, which overwhelmingly correspond to the onset and rapid outbreak of HIV/AIDS. The period-based increases were much more pronounced in the black population, and race differences in mortality rates were relatively greater among women than among men. Furthermore, recent period-based reductions in infectious disease mortality have been more rapid among men than among women, and more pronounced in the black population than in the white population.

**Figure 6 about Here**

Variation in black men’s and women’s homicide mortality is evident in cohorts living during periods of high rates of homicide in the 1960s, the 1980s, and the early 1990s. Rapid cohort-based increases in black men’s homicide mortality are especially striking among more recent birth cohorts, as homicide has become increasingly concentrated among younger, black male victims of firearms and drug-related crimes (Blumstein et al. 2000). However, period-based decreases in black homicide rates are very pronounced after the mid-1990s, which have generally been thought to reflect changes in drug markets, police responses to gun-carrying youths, rapid incarceration of young black men (Pettit 2012), and efforts to restrict access to firearms (Blumstein et al. 2000). The period-based changes rapidly narrowed the black-white gap in homicide mortality, which, together with the period-based changes in infectious disease mortality, strongly supports our third hypothesis.

Patterns in cohort-based variation in accident mortality are unsystematic, but period-
based reductions starting in the 1965-1970 time period are very detectable in all sub groups, and likely reflect decreasing deaths from automobile accidents following the 1968 seat belt legislation mandating that all new cars be equipped with driver and passenger seat belts. We also see steady and significant recent period-based reductions among black men and women, while there are large period-based increases in white men’s and women’s mortality, likely stemming from trends in accidental deaths related to both illicit and pharmaceutical drug-overdoses (Hall et al. 2008; Miech et al. 2012).

Discussion
Multiple estimates show that each year there are between 75,000 and 100,000 excess premature deaths for U.S. blacks compared to U.S. whites (Levine et al., 2001; Satcher et al., 2005; Williams and Jackson, 2005). Results from our analyses implicate various period- and cohort-based changes in black and white U.S. adult mortality patterns behind these disparities. Taken together, the results provide evidence that is both consistent with existing research and supportive of our three hypotheses. First, changes in U.S. black and white all-cause mortality between 1959 and 2009 exhibited more pronounced cohort trends than period trends. Second, cohort-based changes in all-cause mortality were more pronounced in the white population than in the black population for birth cohorts born before World War II. Third, black men and (especially) women experienced striking period-based reductions in all-cause mortality during the 1960s and 1970s. Fourth, supporting our first hypothesis, cohort-based reductions in black men’s and women’s all-cause mortality were faster among birth cohorts born after World War II than among cohorts born earlier in the twentieth century. Fifth, supporting our second hypothesis, black men and women made significant recent cohort-based gains on white men’s and women’s chronic disease mortality rates. And sixth, supporting our third hypothesis, young
black men and women also made significant recent period-based gains in comparison to young white men and women with respect to infectious disease and external causes of death.

The findings reported here illustrate the advantages of taking a nuanced approach to understanding mortality trends that is attentive to both period- and cohort-based influences on U.S. black and white mortality. Indeed, behind the slow, continued narrowing of black-white differences in life expectancy are disparate period- and cohort-based changes in age-specific mortality rates that vary by cause of death. Traditional approaches that have estimated mortality trends using period-based models have masked important variation in black-white mortality that has unfolded disparately across birth cohorts. For example, cohort-based stalling and increasing rates of heart disease mortality among recent cohorts of black and white men and women are a particular concern that goes largely unnoticed by period-based analyses of U.S. mortality trends. But such findings are consistent with results from previous cohort-based analyses of U.S. mortality (Yang 2008; Reither et al. 2011) and might alert us to the possible effects of the U.S. obesity epidemic and other long-term, cumulative, life course effects on adult mortality trends. Thus, accurately assessing the variation behind general trends in all-cause mortality is necessary for understanding successes (e.g., period-based reductions in heart disease and stroke mortality), revealing where improvements might continue (e.g., cohort-based reductions in lung cancer mortality), and alerting policy-makers to persisting or widening differences in mortality risks (e.g., stalling cohort-based variation in heart disease and widening cohort-based racial differences in breast cancer mortality).

In this regard, there are legitimate reasons to caution that we might not expect continued closure of black-white differences in U.S. mortality rates in the coming decades. While the United States has done well to compress disease, disability, and mortality to occur at increasingly
older ages, new evidence shows a potential reversal of this trend (Crimmins and Beltran-Sanchez 2011), and race remains an extremely important factor in shaping life chances for health and longevity. Indeed, race – as a social construct deployed as a tool for discriminating against and oppressing large segments of the population (Williams et al. 2010) – remains a fundamental social cause of disease and mortality in America (Link and Phelan 1995; Phelan et al. 2010). There are strikingly strong race differences, for example, in access to and use of curative and protective health care technologies (Williams et al. 2010), which is consistent with Fundamental Cause Theory’s (Link and Phelan 1995) assertion that those who are socially advantaged are most likely to benefit from advances in health-related knowledge and technologies. Consequently, continued innovation and implementation of pharmacological and medical technologies to prevent, manage, and/or cure diseases will likely affect black and white chronic disease mortality in significantly different ways. For example, while procedures to identify and treat many cancers have greatly improved in the United States, evidence shows clear black-white differences in cancer survival even after identifying and treating the disease (Tehranifar et al. 2009). This evidence is consistent with findings presented here that showed significant widening of the black-white gap in cancer mortality across recent time periods. Thus, we must be mindful of the stark racial inequalities in access and use of health care in the United States, which, in turn, may strongly condition the health returns of new treatments for black and white Americans (Frisbie et al. 2004). We must also recognize that racial inequalities affect both period-based changes in mortality risk and also influence long term, cumulative processes that shape cohort-based changes in mortality risk. Recent period-based changes in the United States, such as the mass imprisonment of black men (Lyons and Pettit 2011; Pettit 2012; Wildeman and Muller 2012), persisting educational and income inequality (Pettit and Ewert 2009; Cataldi, Laird, and
KewalRamani 2009), persisting and increasing segregation (Sharkey 2012; Sharkey and Elwert 2011), and other indicators of concentrated disadvantage across recent years will likely affect cohort-based trends in U.S. black and white adult mortality for many years to come.

The analyses here are not without limitation. First, the results are entirely descriptive. We only allude to possible period- and cohort-based processes behind the patterns of U.S. black and white adult mortality trends and no examinations of mechanisms are performed. Second, we omit the oldest age groups from our analyses because data quality issues have been shown to bias estimates of mortality patterns of the elderly population. However, the United States population is increasingly composed of the aged and mortality is increasingly being compressed to occur among higher age groups. Future mortality analyses considering both period- and cohort-based factors among the older aged population are needed. Third, mortality is the only health outcome we analyze. Behind the black-white differences in life expectancy are not only disparate mortality rates between black and white Americans, but also disparate prevalence and incidence of chronic diseases, functional limitations, and disabilities that generate very different health profiles for the black and white populations. Fourth, we necessarily limit our analysis to blacks and whites because our 50-year time horizon precludes the identification of ethnicity, but we recognize that future analyses should consider other minority populations when data allow.

These limitations aside, the findings here illustrate advantages in analyzing U.S. mortality patterns that are attentive to both period- and cohort-based changes, especially as they relate to the disparate mortality experiences of the U.S. black and white populations. While the twentieth century witnessed radical transformations in living standards and longevity for U.S. whites and (especially) blacks, race-based differences in health and longevity remain large and race remains a predominant social factor in American life. Researchers and policy-makers concerned with
reducing racial inequalities in health and longevity in the United States need to understand how
period and cohort contexts shape disparate life course health trajectories of black and white
populations. Thus, national commitments to improving health and longevity must be attentive to
conditions and policies that affect both immediate and long-term health and survival advances
for all subgroups of the population.
Endnotes

1. We empirically tested whether accounting for cohort variation in adult mortality improves model fit by estimating age-specific mortality rates for U.S. black and white men and women across a series of models. Specifically, we estimated a baseline Age model that includes only five-year age-specific mortality rates, an Age-Period model that controls for five-year time periods 1955-1959 through 2005-2009, an Age-Cohort model that accounts for ten-year birth cohorts 1885-1895 through 1990-2000, and an Age-Period-Cohort (APC) model that includes all three temporal dimensions. Estimated Likelihood Ratio tests and Bayesian Information Criteria (BIC) presented in online Table S1 indicate that the APC model provides the best fitting estimates of U.S. black and white men’s and women’s mortality rates between 1959 and 2009. Further impetus to more stringently test this hypothesis is also found in Figure S1, which contrasts relative period-based changes in age-specific mortality rates for U.S. black and white men and women between 1959 and 2009. The figure depicts percent changes in mortality rates for black and white men and women for ages 30-34, 50-54, and 70-74 across the time period 1959-2009. If cohort-based changes in mortality were not occurring, we would observe parallel rates of mortality change for the age groups across time (Glenn 2005). In contrast, Figure S1 shows significantly different patterns in period-based mortality change between the age groups, suggesting cohort variations in adult mortality over the past 50 years.


3. To assess the validity of mortality rate estimates from our final dataset, race- and sex-specific five-year age-specific mortality rates for years 1968 to 1992 were compared to corresponding five-year estimates made available at the Berkeley Mortality Database (BMD) (http://demog.berkeley.edu/~bmd/states.html). Our yearly estimates between ages 15-19 and 70-74 are nearly identical to those made available at BMD, save for estimates of black men’s and women’s mortality rates for years 1970-1974. Across these years, our estimates are more stable than the BMD estimates. All tables and figures comparing our estimates of U.S. black and white men’s and women’s five-year age-specific mortality rates between 1968 and 1992 with BMD’s respective estimates are available upon request.

4. Age-Period-Cohort analyses were performed on an older aged sample composed of age groups
60-64; 65-69; 70-74; 75-79; and 80-84.

5. Year 1963 was omitted from the 1960-1964 period due to the fact that only $\frac{1}{4}$ of deaths were officially recorded in 1963.

6. We tested the robustness of the results by conducting several sensitivity analyses. First, we used the results to simulate data and then reanalyzed the age-, period-, and cohort-based variation in men’s and women’s logged mortality rates using the IE models (Gelman and Hill 2007). Next, we changed the reference categories for the age, period, and cohort groups to see if the period- and cohort-based variation in mortality were sensitive to the selection of reference category. Finally, we examined age-, period-, and cohort-based variation in US mortality rates by fitting Markov Chain Monte Carlo (MCMC) Hierarchical Age-Period-Cohort (HAPC) Cross-classified Random Effects Models (CCREM). Results from all three sensitivity analyses are consistent with those presented in this paper using the IE from the `ie_rate` program (Powers 2013). Results from these sensitivity analyses are available upon request.
References


### Table 1. Black Women's Five-year All-cause Mortality Rates by Five-year Time Periods, 1955-1959 to 2005-2009.

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>15-19</td>
<td>82.8</td>
<td>81.4</td>
<td>83.8</td>
<td>80.8</td>
<td>57.8</td>
<td>47.1</td>
<td>48.5</td>
<td>52.9</td>
<td>48.7</td>
<td>40.9</td>
<td>37.5</td>
</tr>
<tr>
<td>20-24</td>
<td>136.8</td>
<td>131.9</td>
<td>135.0</td>
<td>136.9</td>
<td>103.3</td>
<td>83.2</td>
<td>82.8</td>
<td>87.3</td>
<td>76.6</td>
<td>69.7</td>
<td>63.4</td>
</tr>
<tr>
<td>25-29</td>
<td>186.1</td>
<td>205.5</td>
<td>193.4</td>
<td>181.8</td>
<td>137.6</td>
<td>117.2</td>
<td>122.6</td>
<td>130.0</td>
<td>110.3</td>
<td>96.2</td>
<td>86.2</td>
</tr>
<tr>
<td>30-34</td>
<td>289.7</td>
<td>304.5</td>
<td>301.3</td>
<td>255.1</td>
<td>182.5</td>
<td>160.7</td>
<td>182.7</td>
<td>189.9</td>
<td>162.7</td>
<td>136.8</td>
<td>120.8</td>
</tr>
<tr>
<td>35-39</td>
<td>418.2</td>
<td>457.9</td>
<td>454.8</td>
<td>393.8</td>
<td>285.6</td>
<td>233.8</td>
<td>245.8</td>
<td>267.3</td>
<td>245.1</td>
<td>210.2</td>
<td>179.6</td>
</tr>
<tr>
<td>40-44</td>
<td>627.1</td>
<td>660.5</td>
<td>656.1</td>
<td>589.4</td>
<td>436.6</td>
<td>364.0</td>
<td>353.8</td>
<td>362.6</td>
<td>354.9</td>
<td>330.3</td>
<td>284.2</td>
</tr>
<tr>
<td>45-49</td>
<td>893.5</td>
<td>897.1</td>
<td>924.0</td>
<td>841.2</td>
<td>658.4</td>
<td>565.1</td>
<td>522.1</td>
<td>519.7</td>
<td>499.6</td>
<td>488.0</td>
<td>441.5</td>
</tr>
<tr>
<td>50-54</td>
<td>1357.2</td>
<td>1390.3</td>
<td>1252.7</td>
<td>1156.0</td>
<td>966.7</td>
<td>856.2</td>
<td>820.8</td>
<td>759.0</td>
<td>706.4</td>
<td>689.1</td>
<td>652.5</td>
</tr>
<tr>
<td>55-59</td>
<td>1598.3</td>
<td>1870.1</td>
<td>1738.1</td>
<td>1523.2</td>
<td>1339.7</td>
<td>1278.6</td>
<td>1202.0</td>
<td>1142.4</td>
<td>1065.1</td>
<td>988.5</td>
<td>890.7</td>
</tr>
<tr>
<td>60-64</td>
<td>2653.3</td>
<td>3164.3</td>
<td>2682.6</td>
<td>2232.4</td>
<td>1850.7</td>
<td>1833.2</td>
<td>1827.1</td>
<td>1666.8</td>
<td>1590.5</td>
<td>1450.1</td>
<td>1288.4</td>
</tr>
<tr>
<td>65-69</td>
<td>3245.0</td>
<td>3322.4</td>
<td>3477.1</td>
<td>3000.6</td>
<td>2525.7</td>
<td>2416.1</td>
<td>2490.8</td>
<td>2409.8</td>
<td>2233.0</td>
<td>2102.3</td>
<td>1848.7</td>
</tr>
<tr>
<td>70-74</td>
<td>4415.1</td>
<td>4592.3</td>
<td>4430.7</td>
<td>4773.1</td>
<td>3821.2</td>
<td>3600.0</td>
<td>3524.6</td>
<td>3399.3</td>
<td>3442.9</td>
<td>3041.3</td>
<td>2621.7</td>
</tr>
</tbody>
</table>

1920-1930 Birth Cohort | 1950-1960 Birth Cohort
Figure 1. Age, Period, Cohort Patterns in U.S. Adult All-Cause Mortality Rates, 1959-2009.

Age Patterns estimated at Cohort 1920-1930 and Period 1975-1979
Period Patterns estimated at Age 60-64 and Cohort 1920-1930
Cohort Patterns estimated at Age 60-64 and Period 1975-1979
Figure 2. Relative Rate Ratios between Black and White Birth Cohort's Adult All-Cause Mortality Rates, 1959-2009.

<table>
<thead>
<tr>
<th>Birth Cohort</th>
<th>Relative Rate Ratio</th>
<th>Birth Cohort</th>
<th>Relative Rate Ratio</th>
</tr>
</thead>
<tbody>
<tr>
<td>1885-1895</td>
<td>0.75</td>
<td>1885-1895</td>
<td>0.75</td>
</tr>
<tr>
<td>1895-1905</td>
<td>1.00</td>
<td>1905-1915</td>
<td>1.00</td>
</tr>
<tr>
<td>1915-1925</td>
<td>1.25</td>
<td>1925-1935</td>
<td>1.25</td>
</tr>
<tr>
<td>1925-1935</td>
<td>1.50</td>
<td>1935-1945</td>
<td>1.50</td>
</tr>
<tr>
<td>1935-1945</td>
<td>1.75</td>
<td>1945-1955</td>
<td>1.75</td>
</tr>
<tr>
<td>1945-1955</td>
<td>2.00</td>
<td>1955-1965</td>
<td>2.00</td>
</tr>
</tbody>
</table>

Note: Estimated at Age 60-64 and Period 1975-1979
Figure 3a. Period and Cohort Trends in U.S. Women's Chronic Disease Mortality, 1959-2009.

Cohort Patterns estimated at Age 60-64 and Period 1975-1979
Period Patterns estimated at Age 60-64 and Cohort 1915-1925
Figure 3b. Period and Cohort Trends in U.S. Women's Chronic Disease Mortality, 1959-2009.

Cohort Patterns estimated at Age 60-64 and Period 1975-1979
Period Patterns estimated at Age 60-64 and Cohort 1915-1925
Figure 4a. Period and Cohort Trends in U.S. Men's Chronic Disease Mortality, 1959-2009.

Cohort Patterns estimated at Age 55-59 and Period 1975-1979
Period Patterns estimated at Age 55-59 and Cohort 1915-1925
Figure 4b. Period and Cohort Trends in U.S. Men's Chronic Disease Mortality, 1959-2009.

Cohort Patterns estimated at Age 55-59 and Period 1975-1979
Period Patterns estimated at Age 55-59 and Cohort 1915-1925
Figure 5. Period and Cohort Trends in U.S. Young Women's Mortality from Infectious Disease and External Causes, 1959-2009.

Infectious Disease Patterns estimated at Age 30-34, Period 1985-1989, and Cohort 1960-1970
Figure 6. Period and Cohort Trends in U.S. Young Men's Mortality from Infectious Disease and External Causes, 1959-2009.

Infectious Disease Patterns estimated at Age 30-34, Period 1985-1989, and Cohort 1960-1970
## Supplementary Material

Table S1: Goodness-of-Fit Statistics for U.S. Mortality Rate Models

<table>
<thead>
<tr>
<th></th>
<th>Black Men</th>
<th></th>
<th>Black Women</th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Log Likelihood</td>
<td>BIC</td>
<td>df</td>
<td>Log Likelihood</td>
<td>BIC</td>
<td>df</td>
</tr>
<tr>
<td></td>
<td>A</td>
<td>AP</td>
<td>AC</td>
<td>APC</td>
<td>A</td>
<td>AP</td>
</tr>
<tr>
<td>Black Men</td>
<td>206908.2</td>
<td>32975.2</td>
<td>44066.5</td>
<td>26458.0</td>
<td>176056.2</td>
<td>15224.0</td>
</tr>
<tr>
<td>BIC</td>
<td>-413746.1</td>
<td>-65816.1</td>
<td>-87928.4</td>
<td>-52647.4</td>
<td>-35042.1</td>
<td>-30313.7</td>
</tr>
<tr>
<td>df</td>
<td>11</td>
<td>21</td>
<td>32</td>
<td>42</td>
<td>11</td>
<td>21</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th></th>
<th>White Men</th>
<th></th>
<th>Black Women</th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Log Likelihood</td>
<td>BIC</td>
<td>df</td>
<td>Log Likelihood</td>
<td>BIC</td>
<td>df</td>
</tr>
<tr>
<td></td>
<td>A</td>
<td>AP</td>
<td>AC</td>
<td>APC</td>
<td>A</td>
<td>AP</td>
</tr>
<tr>
<td>White Men</td>
<td>1404904.0</td>
<td>124259.1</td>
<td>65015.2</td>
<td>41767.7</td>
<td>404142.6</td>
<td>31201.1</td>
</tr>
<tr>
<td>BIC</td>
<td>-2809738.0</td>
<td>-248383.9</td>
<td>-129825.6</td>
<td>-83266.7</td>
<td>-808214.8</td>
<td>-62267.9</td>
</tr>
<tr>
<td>df</td>
<td>11</td>
<td>21</td>
<td>32</td>
<td>42</td>
<td>11</td>
<td>21</td>
</tr>
</tbody>
</table>

Note: BIC is the Bayesian Information Criterion and is estimated to be Deviance +2((1/2)ln(N))(df)
Figure S1. Period Trends in U.S. Men's and Women's Mortality, 1959-2009, by Age Group.

Note: % denotes age-specific mortality in period relative to mortality in 1955-1959 period.

<table>
<thead>
<tr>
<th></th>
<th>Black Women</th>
<th>White Women</th>
<th>Black Men</th>
<th>White Men</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>$B$</td>
<td>$t$</td>
<td>$B$</td>
<td>$t$</td>
</tr>
<tr>
<td><strong>Age</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>15-19</td>
<td>-1.84</td>
<td>-370.8</td>
<td>-1.42</td>
<td>-632.7</td>
</tr>
<tr>
<td>20-24</td>
<td>-1.38</td>
<td>-351.3</td>
<td>-1.67</td>
<td>-650.1</td>
</tr>
<tr>
<td>25-29</td>
<td>-1.10</td>
<td>-311.8</td>
<td>-1.31</td>
<td>-633.4</td>
</tr>
<tr>
<td>30-34</td>
<td>-0.80</td>
<td>-260.2</td>
<td>-1.09</td>
<td>-586.3</td>
</tr>
<tr>
<td>35-39</td>
<td>-0.48</td>
<td>-179.5</td>
<td>-0.75</td>
<td>-473.8</td>
</tr>
<tr>
<td>40-44</td>
<td>-0.15</td>
<td>-64.0</td>
<td>-0.35</td>
<td>-263.9</td>
</tr>
<tr>
<td>45-49</td>
<td>0.17</td>
<td>83.4</td>
<td>0.07</td>
<td>62.7</td>
</tr>
<tr>
<td>50-54</td>
<td>0.50</td>
<td>264.3</td>
<td>0.47</td>
<td>486.7</td>
</tr>
<tr>
<td>55-59</td>
<td>0.80</td>
<td>451.9</td>
<td>0.85</td>
<td>1000.9</td>
</tr>
<tr>
<td>60-64</td>
<td>1.14</td>
<td>680.4</td>
<td>1.25</td>
<td>1625.9</td>
</tr>
<tr>
<td>65-69</td>
<td>1.40</td>
<td>849.6</td>
<td>1.63</td>
<td>2253.4</td>
</tr>
<tr>
<td>70-74</td>
<td>1.73</td>
<td>1051.8</td>
<td>2.04</td>
<td>2904.6</td>
</tr>
<tr>
<td><strong>Period</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1955-1959</td>
<td>0.13</td>
<td>31.1</td>
<td>-0.02</td>
<td>-12.0</td>
</tr>
<tr>
<td>1960-1964</td>
<td>0.19</td>
<td>80.6</td>
<td>0.01</td>
<td>13.1</td>
</tr>
<tr>
<td>1965-1969</td>
<td>0.15</td>
<td>77.8</td>
<td>0.06</td>
<td>63.9</td>
</tr>
<tr>
<td>1970-1974</td>
<td>0.08</td>
<td>41.2</td>
<td>0.06</td>
<td>62.7</td>
</tr>
<tr>
<td>1975-1979</td>
<td>-0.08</td>
<td>-40.6</td>
<td>-0.02</td>
<td>-12.3</td>
</tr>
<tr>
<td>1980-1984</td>
<td>-0.11</td>
<td>-60.1</td>
<td>-0.04</td>
<td>-42.7</td>
</tr>
<tr>
<td>1985-1989</td>
<td>-0.08</td>
<td>-40.9</td>
<td>-0.03</td>
<td>-34.3</td>
</tr>
<tr>
<td>1990-1994</td>
<td>-0.06</td>
<td>-30.5</td>
<td>-0.03</td>
<td>-40.2</td>
</tr>
<tr>
<td>1995-1999</td>
<td>-0.05</td>
<td>-27.0</td>
<td>-0.01</td>
<td>-14.2</td>
</tr>
<tr>
<td>2000-2004</td>
<td>-0.06</td>
<td>-34.8</td>
<td>0.01</td>
<td>14.9</td>
</tr>
<tr>
<td>2005-2009</td>
<td>-0.12</td>
<td>-60.6</td>
<td>0.01</td>
<td>12.9</td>
</tr>
<tr>
<td><strong>Cohort</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1885-1894</td>
<td>0.34</td>
<td>32.2</td>
<td>0.59</td>
<td>167.3</td>
</tr>
<tr>
<td>1890-1899</td>
<td>0.32</td>
<td>66.2</td>
<td>0.50</td>
<td>285.5</td>
</tr>
<tr>
<td>1895-1904</td>
<td>0.33</td>
<td>89.1</td>
<td>0.41</td>
<td>298.2</td>
</tr>
<tr>
<td>1900-1909</td>
<td>0.46</td>
<td>148.0</td>
<td>0.34</td>
<td>270.3</td>
</tr>
<tr>
<td>1905-1914</td>
<td>0.37</td>
<td>129.6</td>
<td>0.26</td>
<td>224.0</td>
</tr>
<tr>
<td>1910-1919</td>
<td>0.33</td>
<td>123.6</td>
<td>0.24</td>
<td>217.3</td>
</tr>
<tr>
<td>1915-1924</td>
<td>0.28</td>
<td>111.7</td>
<td>0.23</td>
<td>214.3</td>
</tr>
<tr>
<td>1920-1929</td>
<td>0.28</td>
<td>114.9</td>
<td>0.19</td>
<td>185.5</td>
</tr>
<tr>
<td>1925-1934</td>
<td>0.26</td>
<td>109.7</td>
<td>0.16</td>
<td>152.7</td>
</tr>
<tr>
<td>1930-1939</td>
<td>0.17</td>
<td>71.7</td>
<td>0.10</td>
<td>96.5</td>
</tr>
<tr>
<td>1935-1944</td>
<td>0.09</td>
<td>39.6</td>
<td>0.02</td>
<td>21.7</td>
</tr>
<tr>
<td>Cohort</td>
<td>Log Likelihood</td>
<td>BIC</td>
<td>df</td>
<td>N</td>
</tr>
<tr>
<td>--------------</td>
<td>----------------</td>
<td>------</td>
<td>------</td>
<td>--------------</td>
</tr>
<tr>
<td>1940-1949</td>
<td>7593.8</td>
<td>13170.8</td>
<td>26458.0</td>
<td>41767.7</td>
</tr>
<tr>
<td>1945-1954</td>
<td>-14919</td>
<td>-26073</td>
<td>-52647</td>
<td>-83267</td>
</tr>
<tr>
<td>1950-1950</td>
<td>-522,247,989</td>
<td>3,666,814,918</td>
<td>460,667,686</td>
<td>3,574,541,743</td>
</tr>
<tr>
<td>1955-1964</td>
<td>-512,722,705.7</td>
<td>3,660,413,918</td>
<td>460,667,686</td>
<td>3,574,541,743</td>
</tr>
<tr>
<td>1965-1974</td>
<td>-512,722,705.7</td>
<td>3,660,413,918</td>
<td>460,667,686</td>
<td>3,574,541,743</td>
</tr>
<tr>
<td>1970-1979</td>
<td>-512,722,705.7</td>
<td>3,660,413,918</td>
<td>460,667,686</td>
<td>3,574,541,743</td>
</tr>
<tr>
<td>1975-1984</td>
<td>-512,722,705.7</td>
<td>3,660,413,918</td>
<td>460,667,686</td>
<td>3,574,541,743</td>
</tr>
<tr>
<td>1990-1999</td>
<td>-512,722,705.7</td>
<td>3,660,413,918</td>
<td>460,667,686</td>
<td>3,574,541,743</td>
</tr>
<tr>
<td>Intercept</td>
<td>-5.32</td>
<td>-5.94</td>
<td>-4.63</td>
<td>-5.2</td>
</tr>
</tbody>
</table>