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

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Abstract Black–white differences in U.S. adult mortality have narrowed over the past five decades, but whether this narrowing unfolded on a period or cohort basis is unclear. 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 (APC) 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 men and women and white men and women. Recent cohort-based reductions in heart disease, stroke, lung cancer, female breast

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cancer, and other cancer mortality have been substantial and, save for breast cancer, have been 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 a 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.

Keywords Cause-specific mortality · Trends · United States · Disparities

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 age 75.1 in 2010, while life expectancy at birth among whites increased to age 78.9. The resulting 3.8-year disparity continues a long-term narrowing trend. Indeed, the black-white life expectancy disparity was wider than 7 years in 1993 and was estimated to be as wide as 14 or 15 years in 1900 (Arias 2010; Harper et al. 2007).

Although the trend indicates continued progress toward a closing of the black-white mortality disparity, these recent data also demonstrate that the United States fell well short of the 2010 goal of eliminating health disparities across different segments of the population (Healthy People 2011a, b).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 2011b). 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 estimates of life expectancy have decreased in some regions of the United States (Kindig and Cheng 2013) and among some segments of the U.S. population, particularly low-educated white women (Montez and Zajacova 2013; Olshansky et al. 2012). The latter point suggests that some closure of the black-white gap in U.S. life expectancy might reflect increasing mortality among some segments of the U.S. white population.

This article 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 by 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 periods that similarly affect all birth cohorts. However, evidence from recent studies suggests that substantial portions 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 et al. 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 article, then, is to more clearly illuminate 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, and/or within specific age groups and causes of death—is imperative for our understanding of the overall black-white disparity in mortality and for speculating whether the gap is likely to close further.

Literature Review

A number of 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 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, and accidents), even in the context of impressive age-adjusted mortality declines for most of them over this period (Keppel et al. 2010).

Other studies, however, have provided 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 by two years among men between 1993 and 2003. They identified more pronounced declines among blacks in mortality from 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 resulting from

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 these studies, however, 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 period, their mortality rates were significantly lower in comparison with previous birth cohorts passing through the same 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 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 difference 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, although 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 twentieth century.

Turning to race differences in cohort experiences, late nineteenth and early twentieth century black birth cohorts were exceptionally disadvantaged compared with white birth cohorts. In their comprehensive study of social change in America throughout the twentieth 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. . . . [T]heir 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. Although sanitation was poor, health knowledge was low compared with 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 % higher child mortality (ages <5) rates for blacks compared with whites at the time, concluding that race was "the single most important variable in predicting child mortality levels" (1991:94). They explained that "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 (Colen 2011; Crimmins et al. 2004; 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 risks (see Galobardes et al. (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 twentieth century were strongly predictive of mortality well before age 85. Warner and Hayward (2006) provided 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 the 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. 2004; Geronimus 1992; Jackson et al. 2011; Masters et al. 2012).

Although 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 twentieth 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 argued that whites' 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–World War II (WWII) 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 et al. 1997). And although 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 alluded 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 U.S. white mortality were shown to have been greater than cohort-based reductions in U.S. black mortality across the first half of the twentieth century (Masters 2012).

The post–WWII 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 Food Stamp Act of 1964) 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 colleagues (Almond and Chay 2006; Almond et al. 2007) showed that black infant mortality rates in the South declined significantly during the late 1960s. Other evidence suggests that all black Americans—but especially black women—experienced reduced mortality during the late 1960s (Kaplan et al. 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 Americans' 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–WWII 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 (Almond et al. 2007; Caldwell 1993; Easterlin 1996). Preston and Haines (1991:207) summarized the importance of such factors, paraphrasing 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." Enhanced access to mainstream social and economic opportunities and medical care for blacks—that is, a weaker American color line—may be particularly reflected in sizable reductions in chronic disease mortality for black cohorts growing up in post–WWII America: 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 1986). Black individuals born in cohorts late enough in history to spend their infancy,

childhood, and early adulthood in post–WWII America should have especially benefitted from these changes. Consequently, we hypothesize the following:

> *Hypothesis 1:* Adult mortality rates over the past 50 years fell significantly faster among black Americans born after WWII than for black Americans born before WWII.

Consistent with this hypothesis, the faster cohort-based reductions for blacks growing up in post–WWII 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: namely, heart disease, stroke, and cancers. Thus, we hypothesize the following:

> *Hypothesis 2:* Black-white differences in mortality rates from heart disease, stroke, and cancer have significantly narrowed across post–WWII 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, thus reflecting continued systemic social, economic, and health care discrimination and disadvantages that affect the black population (Colen 2011; Hummer and Chinn 2011; Williams and Jackson 2005; Williams et al. 2010). 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, although likely rapid among post–WWII 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 and 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 effective 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 recent reductions among blacks in particular, exhibit strong period-based variation. Although much of the variation in chronic disease mortality is likely associated with cohort-based factors, we believe that period-based variation is most prominent among causes of death affecting younger age groups. Consequently, we hypothesize the following:

> *Hypothesis 3:* Significant period-based reductions in black and white mortality rates occurred in recent decades for deaths resulting from infectious diseases,

homicide and legal intervention, and accidents. These reductions are likely more pronounced among blacks than among 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, midyear population (July 1) 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.[1]

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 U.S. "white" and "non-white" populations for years 1959 to 1968 and for the U.S. "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 2011), 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 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 U.S. blacks and whites for all-causes of death as well as 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

[1] Midyear population estimates for years 1959–1969 were obtained from the U.S. Census Bureau Population Estimates, 1900–1979 files (http://www.census.gov/popest/archives/pre-1980/PE-11.html). Estimates for years 1970–1989 were obtained from the U.S. Centers for Disease Control CDC Wonder, Census Population, 1970–2000 files (http://wonder.cdc.gov/wonder/help/Census1970-2000.html). Finally, estimates for years 1990–2009 were obtained from the U.S. Centers for Disease Control CDC Wonder, Bridged-Race Population Estimates (Vintage 2009) files (http://wonder.cdc.gov/bridged-race-population.html). ICPSR Study No. 20680 contains Multiple Cause of Death Files for years 1959–1967; ICPSR Study No. 3905 contains Multiple Cause of Death Files for years 1968–1973; ICPSR Study No. 3906 contains Multiple Cause of Death Files for years 1974–1978; and ICPSR Study No. 4640 contains Multiple Cause of Death Files for years 2000–2002. The ICPSR makes other individual Multiple Cause of Death Files separately available up to year 2005. Multiple Cause of Death Files for years 2006 through 2009 were obtained from the National Vital Statistics System Data, which are available online (http://www.cdc.gov/nchs/data_access/Vitalstatsonline.htm).

1959–1967, we classified cause of death in accordance with the seventh 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).[2]

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, 1999; Preston and Elo 2006). As such, we limit the age range to younger than 75 years to safeguard against these age-sensitive biases.[3] 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). 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 periods and five-year age groups; they range from birth cohort 1885–1895 to 1990–2000. Because of 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 1990 and 1994 only.[4] The data structure for the analysis is visible in Table 1, where age groups are depicted as rows, period 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.

The data structure fits the assumptions of APC analysis in that birth cohorts are linearly dependent on 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' (2012) Stata module *ie_rate*, which provides flexible extensions of Yang et al.'s (2004) intrinsic estimator (IE).

[2] To assess the validity of mortality rate estimates from our final data set, we compared race- and sex-specific five-year age-specific mortality rates for years 1968 to 1992 with corresponding five-year estimates made available in 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 in the BMD, save for estimates of black men's and women's mortality rates for 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.

[3] Age-period-cohort analyses were performed on an older sample composed of age groups 60–64, 65–69, 70–74, 75–79, and 80–84.

[4] We omitted 1963 from the 1960–1964 period because only one-quarter of deaths were officially recorded in 1963.

Table 1 Black women's five-year all-cause mortality rates by five-year time periods, 1955–1959 to 2005–2009

Age	1955–1959	1960–1964	1965–1969	1970–1974	1975–1979	1980–1984	1985–1989	1990–1994	1995–1999	2000–2004	2005–2009
15–19	82.8	81.4	*83.8*	80.8	57.8	47.1	48.5	52.9	48.7	40.9	37.5
20–24	136.8	131.9	135.0	*136.9*	103.3	83.2	82.8	87.3	76.6	69.7	63.4
25–29	186.1	205.5	193.4	181.8	*137.6*	117.2	122.6	130.0	110.3	96.2	86.2
30–34	289.7	304.5	301.3	255.1	182.5	*160.7*	182.7	189.9	162.7	136.8	120.8
35–39	**418.2**	457.9	454.8	393.8	285.6	233.8	*245.8*	267.3	245.1	210.2	179.6
40–44	627.1	**660.5**	656.1	589.4	436.6	364.0	353.8	*362.6*	354.9	330.3	284.2
45–49	893.5	897.1	**924.0**	841.2	658.4	565.1	522.1	519.7	*499.6*	488.0	441.5
50–54	1,357.2	1,390.3	1,252.7	**1,156.0**	966.7	856.2	820.8	759.0	706.4	*689.1*	652.5
55–59	1,958.3	1,870.1	1,738.1	1,523.2	**1,339.7**	1,278.6	1,202.0	1,142.4	1,065.1	988.5	*890.7*
60–64	2,653.3	3,164.3	2,682.6	2,232.4	1,850.7	**1,833.2**	1,827.1	1,666.8	1,590.5	1,450.1	1,288.4
65–69	3,245.0	3,322.4	3,477.1	3,000.6	2,525.7	2,416.1	**2,490.8**	2,409.8	2,233.0	2,102.3	1,848.7
70–74	4,415.1	4,592.3	4,430.7	4,773.1	3,821.2	3,600.0	3,524.6	**3,399.3**	3,442.9	3,041.3	2,621.7

Notes: Bold figures represent the 1920–1930 birth cohort. Italicized figures represent the 1950–1960 birth cohort.

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 follows:

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

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 β_i^A and β_j^P, respectively. β_k^C denotes the kth 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 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 τ parameters in the APC model are multiplicative effects whose product is 1 over the levels of each factor. Under this normalization, the constant term τ_0 is the scaled grand mean of all five-year age-specific mortality rates (see Yang et al. (2004, 2008) and Powers (2013) for descriptions of the underlying vector geometry and discussions of the estimation techniques). Models estimating all-cause mortality include all age groups 15–19 to 70–74. The models estimating chronic disease mortality 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

Table S4 in Online Resource 1 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 groups, five-year periods, and 10-year cohorts 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 Fig. 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; they are not to be interpreted as representative of the actual mortality rates experienced by a specific cohort or in a specific period.

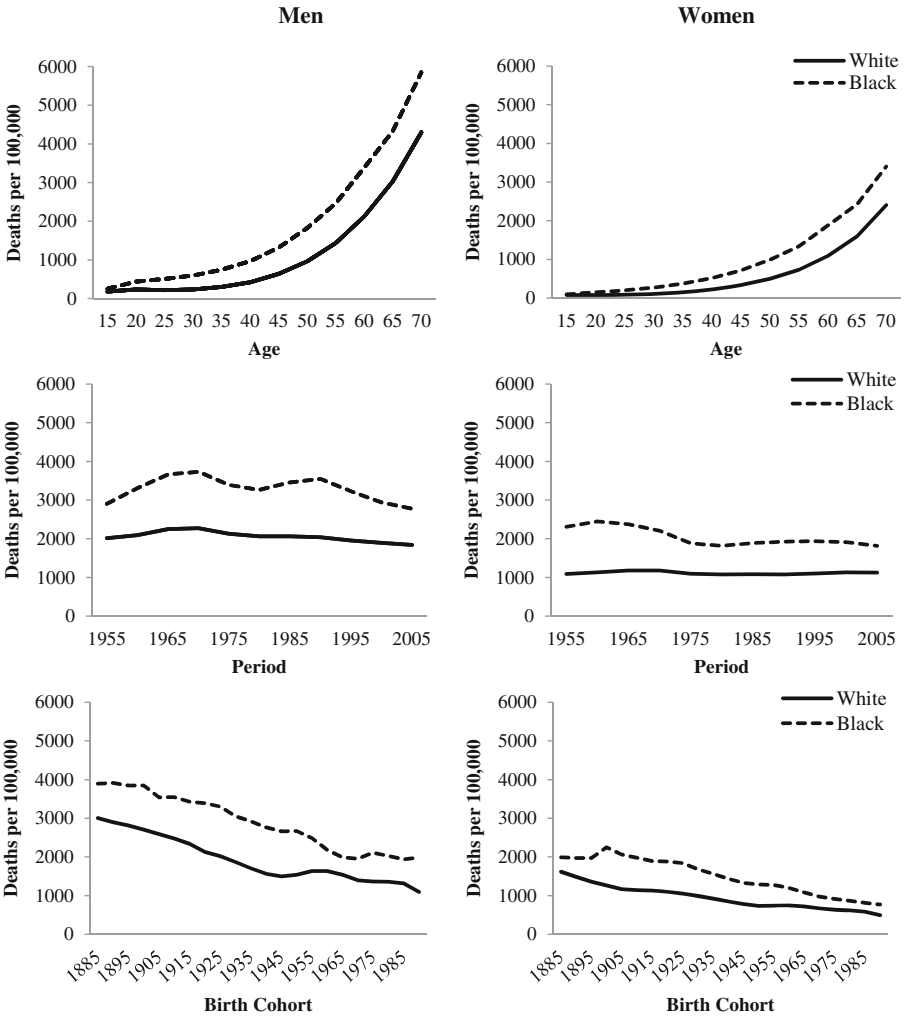


Fig. 1 Age, period, cohort patterns in U.S. adult all-cause mortality rates, 1959–2009. Age patterns are estimated at cohort 1920–1930 and period 1975–1979. Period patterns are estimated at age 60–64 and cohort 1920–1930. Cohort patterns are estimated at age 60–64 and period 1975–1979

When comparing the period- and cohort-based trends illustrated in Fig. 1, we see significantly greater reductions in black and white men's and women's adult mortality rates across cohorts than across 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 Fig. 1 is evidence consistent with previous work showing that reductions in adult mortality rates between 1959 and 2009 for cohorts born before the middle of the twentieth century were greater among U.S. whites than among U.S. blacks (Masters 2012). These differences between black and white cohorts' mortality are clearly seen in Fig. 2, which graphically depicts the differences as relative rate ratios. Holding constant age-based (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 twentieth 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 twentieth century. Cohort-based race differences in women's mortality grew significantly greater at the turn of the century, thereafter steadily remaining high across subsequent cohorts born before the middle of the twentieth century before declining among post–WWII cohorts.

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 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: period-based changes subsequently increased black men's mortality across the 1980–1984 to 1990–1994 periods before resuming their decline during the late 1990s and 2000s.

Results depicted in Figs. 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 WWII. 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 Fig. 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 (i.e., 15–19 to 30–34) black and white men, the details of which will be discussed later herein. Evidence supporting our first hypothesis is also found in the relatively fast

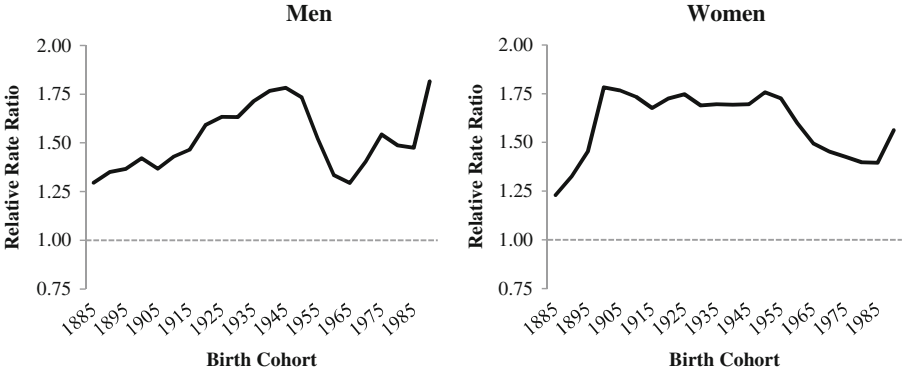


Fig. 2 Relative rate ratios between black and white birth cohorts' adult all-cause mortality rates, 1959–2009. Estimated at age 60–64 and period 1975–1979

cohort-based reductions in black women's mortality for cohorts born after WWII. 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.

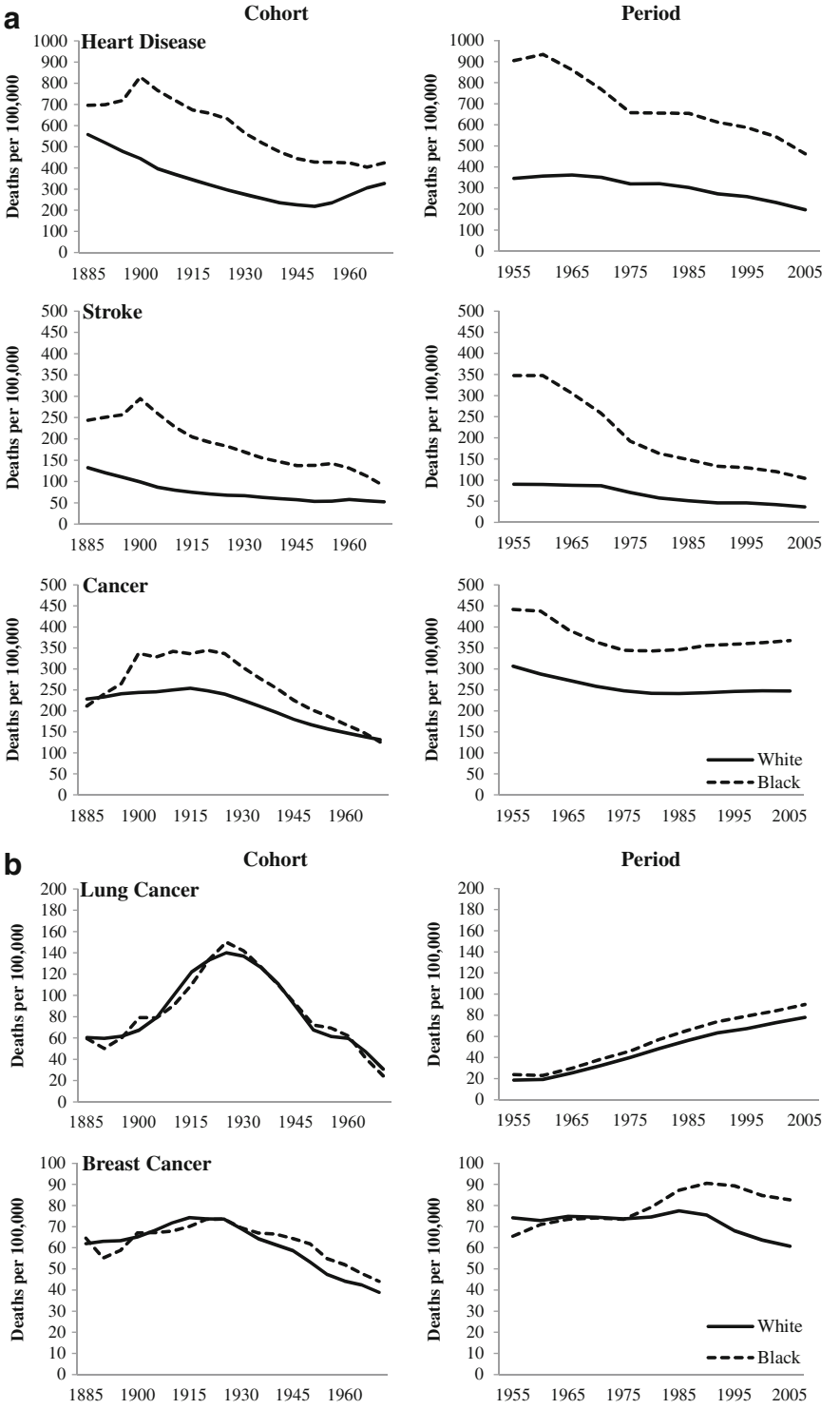
Trends in Major Chronic Disease Mortality

Figure 3a and b present period- and cohort-based changes in black and white women's mortality rates from major chronic diseases, and Fig. 4a and b present 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 driven by cohort-based trends in heart disease, stroke, and non-lung/non-breast cancers for black and white men and women, and by lung cancer mortality for black and white men.

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 (Fig. 3a for women and Fig. 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

Fig. 3 Period and cohort trends in U.S. women's chronic disease mortality rates, 1959–2009. Cohort patterns ▶ are estimated at age 60–64 and period 1975–1979. Period patterns are estimated at age 60–64 and cohort 1915–1925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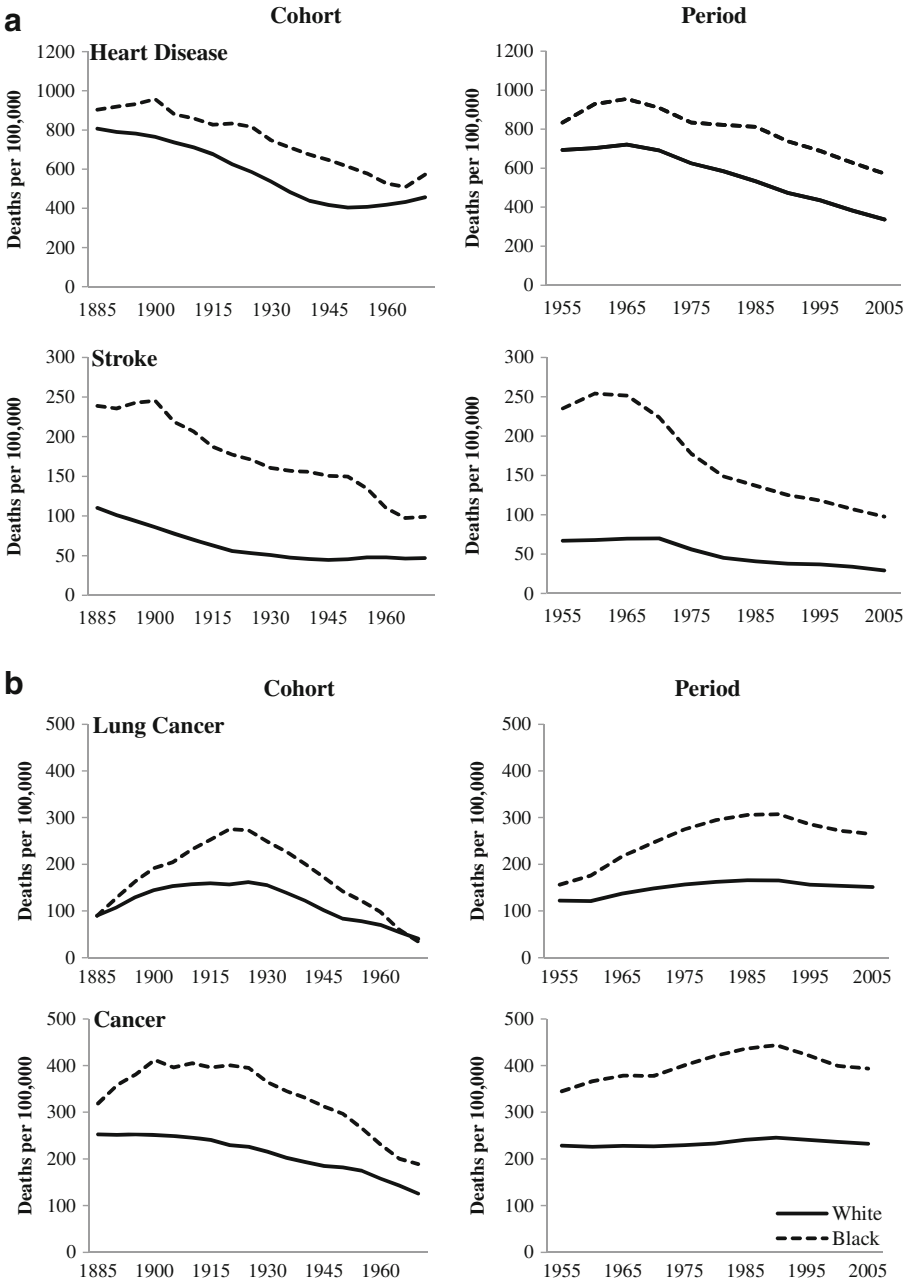

Fig. 4 Period and cohort trends in U.S. men's chronic disease mortality rates, 1959–2009. Cohort patterns are estimated at age 55–59 and period 1975–1979. Period patterns are estimated at age 55–59 and cohort 1915–1925

Fig. 3a, cohort-based racial differences in women's other cancer mortality were quite large and stagnant for cohorts born between 1900 and 1930. However, across cohorts

1930–1940 to 1965–1975, Fig. 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, Fig. 4b reveals significant recent cohort-based reductions among all men. 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 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 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 periods. The general trend of increasing period-based changes largely reflects compositional age changes across periods, in which the birth cohorts with higher rates of smoking (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 (Tehranifar 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. In addition, similar to period-based trends in men's other cancer mortality, we find 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 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 (Fig. 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. Very little period-based variation in breast cancer mortality existed 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, which began in 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 et al. 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 that 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 U.S. Black and White Cause-Specific Mortality at Younger Ages

Period- and cohort-based trends in U.S. black and white young women's mortality from homicides, accidents, and infectious diseases are presented in Fig. 5, and trends among U.S. black and white young men are presented in Fig. 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 women.

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.

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, 1980s, and 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 period are easily detectable in all subgroups and likely reflect decreasing deaths from automobile accidents following the 1968 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, although 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).

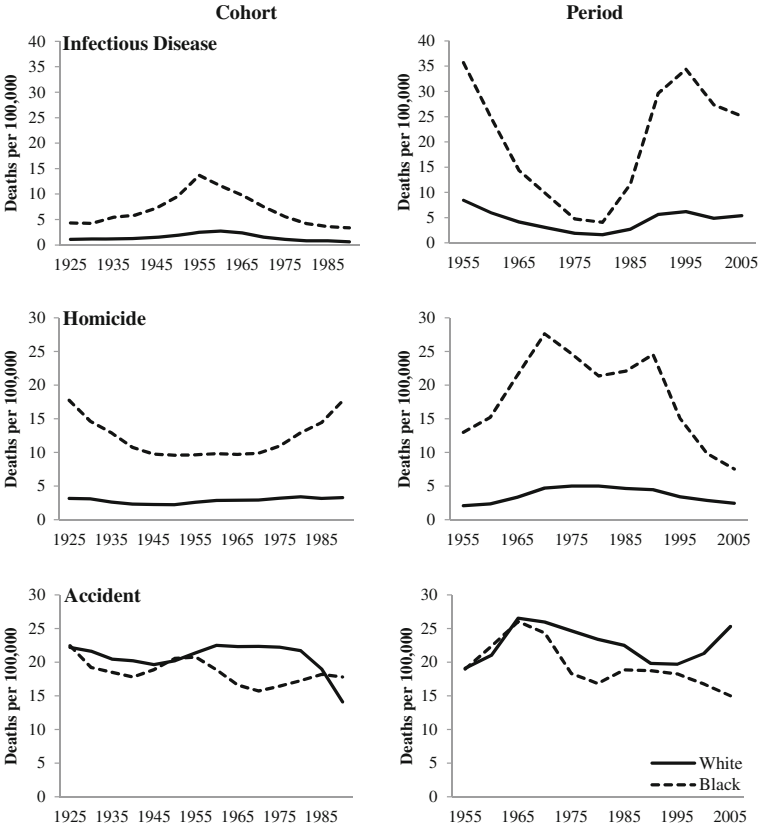

Fig. 5 Period and cohort trends in U.S. young women's mortality from infectious disease and external causes, 1959–2009. Infectious disease patterns are estimated at age 30–34, period 1985–1989, and cohort 1960–1970. Homicide patterns are estimated at age 20–24, period 2000–2004, and cohort 1960–1970. Accident patterns are estimated at age 20–24, period 1985–1989, and cohort 1960–1970

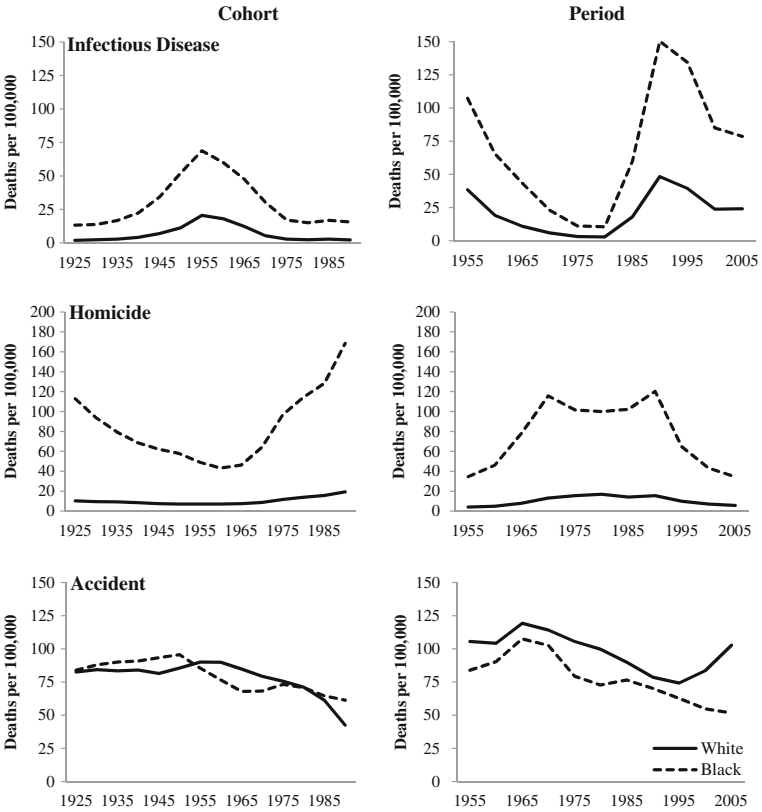

Fig. 6 Period and cohort trends in U.S. young men's mortality from infectious disease and external causes,1959–2009. Infectious disease patterns are estimated at age 30–34, period 1985–1989, and cohort 1960–1970. Homicide patterns are estimated at age 20–24, period 2000–2004, and cohort 1960–1970. Accident patterns are estimated at age 20–24, period 1985–1989, and cohort 1960–1970

Sensitivity Analyses

Serious concerns have been raised regarding whether the IE produces unbiased estimates of the parameters underlying APC trends (Fienberg 2013; Luo 2013). Recently, Luo (2013) used simulation exercises to show the IE was unable to retrieve "true [APC] effects in various circumstances" (p. 1947), and thus concluded that the IE "can yield biased and potentially misleading estimates" (p. 1962). We agree with Luo (2013) that any APC solution "provides just *one* possible solution from the infinite number of solutions" and "should not be regarded as *the true solution* or *the uniquely preferred solution* without theoretical justification" (p. 1965). That said, on both theoretical and empirical grounds, we defend the IE as the preferred solution to estimating APC variation in U.S. adult mortality rates in the National Vital Statistics System data. First, the circumstances under which Luo showed the IE's inability to retrieve true APC effects do not arise in the NVSS data. Second, we assessed the robustness of our results using several alternative APC estimation techniques and simulation exercises and found our results to be highly consistent using the different approaches.

To illustrate these points, we first descriptively, graphically, and statistically examined the presence of age-, period-, and cohort-based variation in U.S. adult mortality rates (Yang and Land 2013). Next, we used the results to simulate data with added error, 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; Luo 2013). Next, to assess sensitivity to alternative coding, we fit two models using an ANOVA (or centered effects) coding scheme—one using the last categories of the age, period, and cohort factors as reference, and another using the first factor levels as reference—to see whether the period- and cohort-based variation in mortality were sensitive to the choice of reference category (Powers 2012). Finally, we examined age-, period-, and cohort-based variation in U.S. adult mortality rates by fitting Markov chain Monte Carlo (MCMC) hierarchical age-period-cohort (HAPC) cross-classified random-effects models (CCREM) (Yang and Land 2013).

Results from all three sensitivity analyses are consistent with those presented in this article using the IE implemented in the *ie_rate* program (Powers 2012). A more thorough discussion of these sensitivity analyses and results is available in Online Resource 1.

Discussion

Multiple estimates show that each year, there are between 75,000 and 100,000 excess premature deaths for U.S. blacks compared with 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 WWII. 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 WWII 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 with young white men and women with respect to infectious diseases 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. However, such findings are consistent with results from previous cohort-based analyses of U.S. mortality (Reither et al. 2011; Yang 2008) 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. Although 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 2010), 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, although 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 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 et al. 2009), persisting and, by some measures, increasing segregation (Sharkey 2012; Sharkey and Elwert 2011), and other indicators of concentrated disadvantage for black Americans 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 allude only 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 (Preston et al. 1996, 1999; Preston and Elo 2006). However, the United States population is increasingly composed of the aged, and mortality is increasingly being compressed to occur among older 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. Although 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.

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In a recent *Demography* article, Luo (2013) raised serious concerns about using the intrinsic estimator (IE) to fit age-period-cohort (APC) models. We fully acknowledge the major issues with the IE in assessing APC variations in social and demographic phenomena, and are particularly sensitive to the charges regarding the IE's ability to estimate unbiased and consistent APC effects. We think that Luo's (2013) paper critical of the IE will be an important advance in APC methodology, especially because it reminds readers that there will never be "an all-purpose solution to the APC conundrum" (to quote from Norval Glenn 2005: 20 in a Sage Publication on this topic). The majority of invited commentators on Luo's paper in the same *Demography* issue agree with this position (e.g., see Fienberg 2013; Held and Riebler 2013). Indeed, in affirming Glenn's point, Luo demonstrates that the IE, under certain conditions, is unable to retrieve the "true" APC effects in simulated data, and concludes the paper by strongly cautioning against the use of the IE as a general solution to identification problems.

Overall, we agree with many of Luo's points in her assessment of the IE. Yet for the purposes of our paper we believe the IE remains the most appropriate method with which to analyze the NVSS data. Our position on this matter is straightforward: First, to assess the IE's ability to estimate unbiased and consistent APC effects, Luo depicts data circumstances that are, on the one hand, highly unlikely to transpire in real-world data and that, on the other hand, are of no substantive concern to the data we analyze. Second, results from models we fit using alternative APC techniques are entirely consistent with the results estimated from the IE in our paper. And third, our investigation of period- and cohort-based trends in US adult mortality is entirely in line with Luo's suggestion to use "APC models that are informed by social theories" (1965).

To the third point, we hope the reader is satisfied with our paper's sections documenting trends in US adult mortality and our theoretical motivations for separating period- and cohort-based trends in our analyses. To address the first two points, we proceed across five steps in this appendix: (1) we replicate the exercises performed by Luo (2013) on three simulated datasets, (2) we next show why APC models should not have been fitted to the simulated data used in Luo's exercises, and also demonstrate that the NVSS data do not adhere to the specific conditions characterizing Luo's simulated data, (3) to assess the robustness of our findings, we simulate data from estimated APC coefficients from IE APC models in our paper and refit IE models to those simulated data, (4) to assess sensitivity to alternative coding, we fit two models using an ANOVA (or centered effects) coding scheme, one using the last categories of the age, period, and cohort factors as reference, and another using the first factor levels as reference, to see if the period- and cohort-based variation in mortality were sensitive to the choice of reference category (Powers 2012), and (5) to further assess robustness of our findings, we estimate APC variation in US adult mortality by fitting Markov Chain Monte Carlo (MCMC) Hierarchical Age-Period-Cohort (HAPC) Cross-classified Random Effects Models (CCREM) (Yang and Land 2013).

Replicate Luo's Simulations

We first create the exact same data structures from Equations (18), (19), and (20) in Luo's simulation exercises (2013: 1954). We perform these replications to show that the analytical approach we use to test the robustness of our results is the very same approach used by Luo to assess the IE in her paper. We also replicate these data to demonstrate the unique characteristics of Luo's data and to show these characteristics are not found in the NVSS data we analyze. In short, we want to assure the reader that we are subjecting our results to the same critical assessment used by Luo in her evaluation of the IE. We then use simulation techniques to arrive at Luo's results.

Here is a replication of Luo's Table 3 (1954), with her "Bias" column having been replaced with values from our APC simulations (indicated by the title, "Replicate")

Table S1. Replicating Simulated Data Sets from Luo's (2013) Data Generating Processes

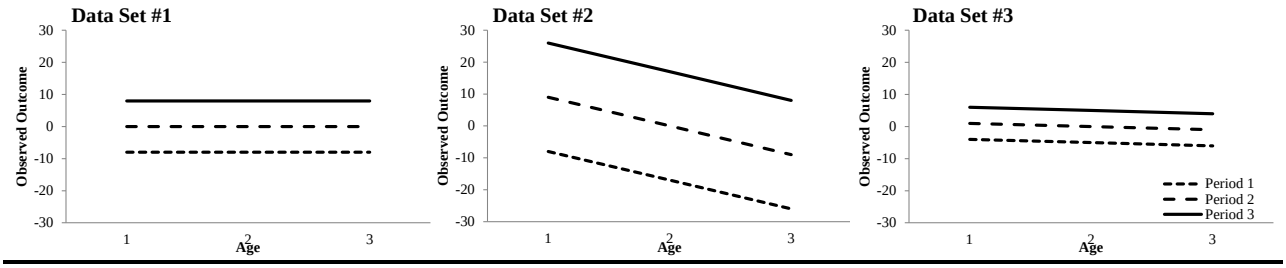
		Data 1			Data 2			Data 3		
		"Truth"	Luo	Replicate	"Truth"	Luo	Replicate	"Truth"	Luo	Replicate
Age	1	-1	-0.997	-1.007	-1	5.747	5.743	-3	0.249	0.243
	2	0	-0.002	-0.006	0	0.002	-0.006	0	0.000	-0.006
	3	1	0.999	1.014	1	-5.749	-5.736	3	-0.249	-0.236
Period	1	-7	-6.999	-6.987	-7	-13.750	-13.737	-1	-4.250	-4.237
	2	0	-0.002	-0.020	0	0.002	-0.020	0	-0.002	-0.020
	3	7	7.002	7.007	7	13.748	13.757	1	4.252	4.257
Cohort	1	-2	-2.001	-1.995	-20	-6.497	-6.495	-8	-1.500	-1.495
	2	-1	-0.998	-0.981	-10	-3.253	-3.231	-4	-0.750	-0.731
	3	0	-0.001	0.009	0	0.002	0.009	0	0.000	0.009
	4	1	1.004	1.010	10	3.250	3.260	4	0.750	0.760
	5	2	1.996	1.956	20	6.498	6.456	8	1.500	1.456

The APC effects we estimate are compared with Luo's estimates, as well as with the "true" effects in the data generating processes she used to simulate these data. In each case, we demonstrate that we are able to replicate the exercises performed by Luo. Thus, to begin, we confirm Luo's findings that the IE is indeed unable to retrieve the "true" age, period, and cohort effects when the method is applied to these simulated data.

Unique Data Conditions

Next, we contend that the circumstances that Luo created to show the IE's inability to retrieve the "true" age, period, and cohort effects are highly unlikely to transpire in real-world applications, such as the temporal trends observed in US adult mortality rates between 1959 and 2009. As seen in the figures below, in all simulated datasets Luo set the functional forms of all APC effects on the outcome Y to be exactly linear.

Figure S1. Period-based Variation in Predicted Age-specific Y Values in Luo's (2013) Three Data Sets.



Consequently, if one predicts the outcome Y for each data set from the "true" age, period, and cohort effects, one sees that Luo has fabricated worlds in which the age effects are completely parallel to one another. Yet no empirical example has been provided nor has any theoretical case been made for the simultaneous or singular existence of perfectly linear age, period, and cohort effects in a demographic or social outcome. As a result of assuming and thus assigning linear APC effects, the only temporal trends observable in these data are across time periods, wherein the age trajectories of Y are simply "shifted" across periods, and remain parallel to one another. Thus, no cohort trends are detected such that a full APC model should never have been fitted (Yang 2008; Yang and Land 2013). Indeed, as the outcomes in these data do not exhibit any cohort-based variation, goodness of model fit statistics (e.g., BIC) suggest that that

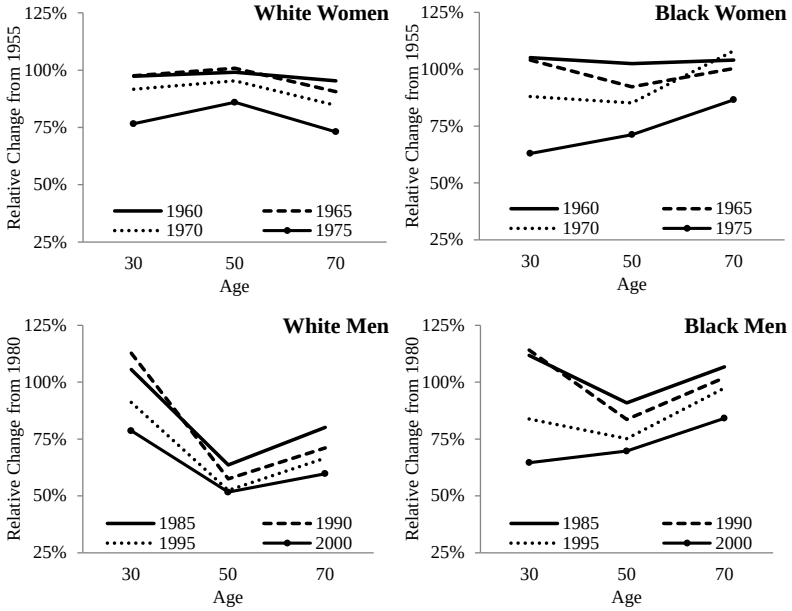
fitting a simpler age-period model would be preferred to fitting a full APC model for all data sets used in Luo's simulations.

Table S2: Goodness-of-Fit Statistics for Luo Simulated Data

	Data 1			
	A	AP	AC	APC
Log Likelihood	-33071.6	-14181.2	-14181.3	-14180.5
BIC	66170.9	28408.4	28427.1	28434.7
df	3	5	7	9
	Data 2			
	A	AP	AC	APC
Log Likelihood	-40527.7	-14202.8	-14181.3	-14202.8
BIC	81083.1	28451.5	28483.0	28460.8
df	3	5	7	9
	Data 3			
	A	AP	AC	APC
Log Likelihood	-28538.0	-14199.1	-14212.3	-14229.1
BIC	57103.7	28444.2	28470.7	28513.4
df	3	5	7	9

Finally, and more to the matter at hand, the circumstances in the simulated data in Luo's exercises (2013) bear no resemblance to the data we use for our analyses. Exploratory analyses of the functional form of age effects on all-cause or cause-specific mortality risk in the United States and the functional form of temporal variation in U.S. mortality rates (i.e., period and/or cohort effects) reveal non-linear patterns. In no instance do we find evidence suggesting linear age patterns or linear period- or cohort-based trends in US adult mortality rates. Below are plots of percent changes in US age-specific mortality rates across time periods to illustrate that temporal change in age trajectories of US adult mortality rates are indeed not parallel to one another.

Figure S2. Period-based Variation in Age Trajectories in U.S. Men's and Women's All-cause Mortality.



Note: % denotes change in age-specific mortality in period relative to mortality in 1955-1959 period for women, and % change relative to the 1980-1984 period for men.

We pick three ages to mimic the three age category data structure used by Luo, and pick two different ranges of time periods (earlier for women, later for men) to showcase the fact that age-variation in US black and white mortality was changing across the entire range of data (e.g., in an early period of the data [1955-1975] we see race differences in women's mortality, and in a later period of the data [1980-2000] we also see race differences in men's mortality).

Finally, Table S3 include model fit statistics (BIC) that indicate that fitting full APC models are preferred to fitting AP or AC models to the data analyzed in our paper (the BIC is calculated for models fit to all-cause mortality, but BIC for models analyzing cause-specific mortality indicate the APC models are preferred to AP or AC).

Table S3: Goodness-of-Fit Statistics for U.S. Mortality Rate Models

	Black Men				**Black Women**			
	A	AP	AC	APC	A	AP	AC	APC
Log Likelihood	206908.2	32975.2	44066.5	26458.0	176056.2	15224.0	17628.0	7593.8
BIC	-413746.1	-65816.1	-87928.4	-52647.4	-35042.1	-30313.7	-35051.4	-14919.0
df	11	21	32	42	11	21	32	42
	White Men				**White Women**			
	A	AP	AC	APC	A	AP	AC	APC
Log Likelihood	1404904.0	124259.1	65015.2	41767.7	404142.6	31201.1	24997.7	13170.8
BIC	-2809738.0	-248383.9	-129825.6	-83266.7	-808214.8	-62267.9	-49790.7	-26073.0
df	11	21	32	42	11	21	32	42

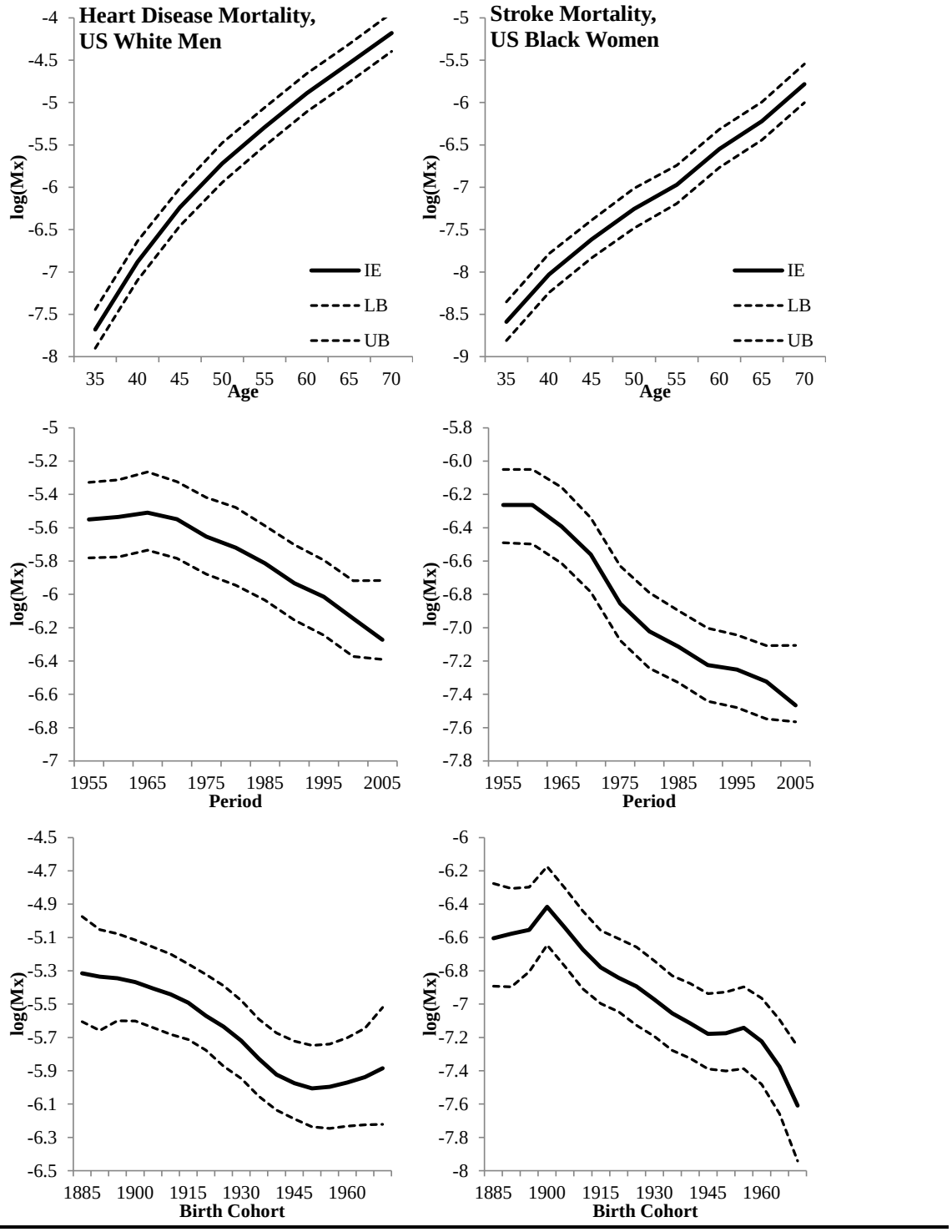
Note: BIC is the Bayesian Information Criterion and is estimated to be -2*log(L) + df*log(N)

Sensitivity Analysis 1: Simulation Diagnostics

We then apply the same simulation approach used by Luo (2013) to the estimated effects we reported in our original manuscript. If simulation techniques are an effective way to determine whether or not the IE can fit "true" effects in simulated data sets, then simulation techniques should be equally effective at confirming whether or not estimated results from real data are valid and consistent (Gelman and Hill 2007). Thus, we use the estimated coefficients from IE models presented in our original manuscript to simulate data with additional variation in the outcomes (i.e., with an added error drawn from a normal distribution with a mean of 0 and a variance of 2). We then refit the IE estimator models to the simulated data to determine how well the IE can retrieve the original effects in the presence of the simulated error. If the IE models are as tenuous as Luo portrays them to be, then the added error in the simulated data will pose too difficult a challenge for the IE to retrieve the original effects – as it appeared to be in the three datasets above.

Figure S3, for example, are results showing mean estimates of APC effects from IE models fitted to 1,000 simulated data sets for US black women's stroke mortality and US white men's heart disease mortality.

Figure S3. Mean APC Effects Estimated from 1,000 Simulations of IE Fitted to Simulated Data for US Black Women's Stroke Mortality and US White Men's Heart Disease Mortality.



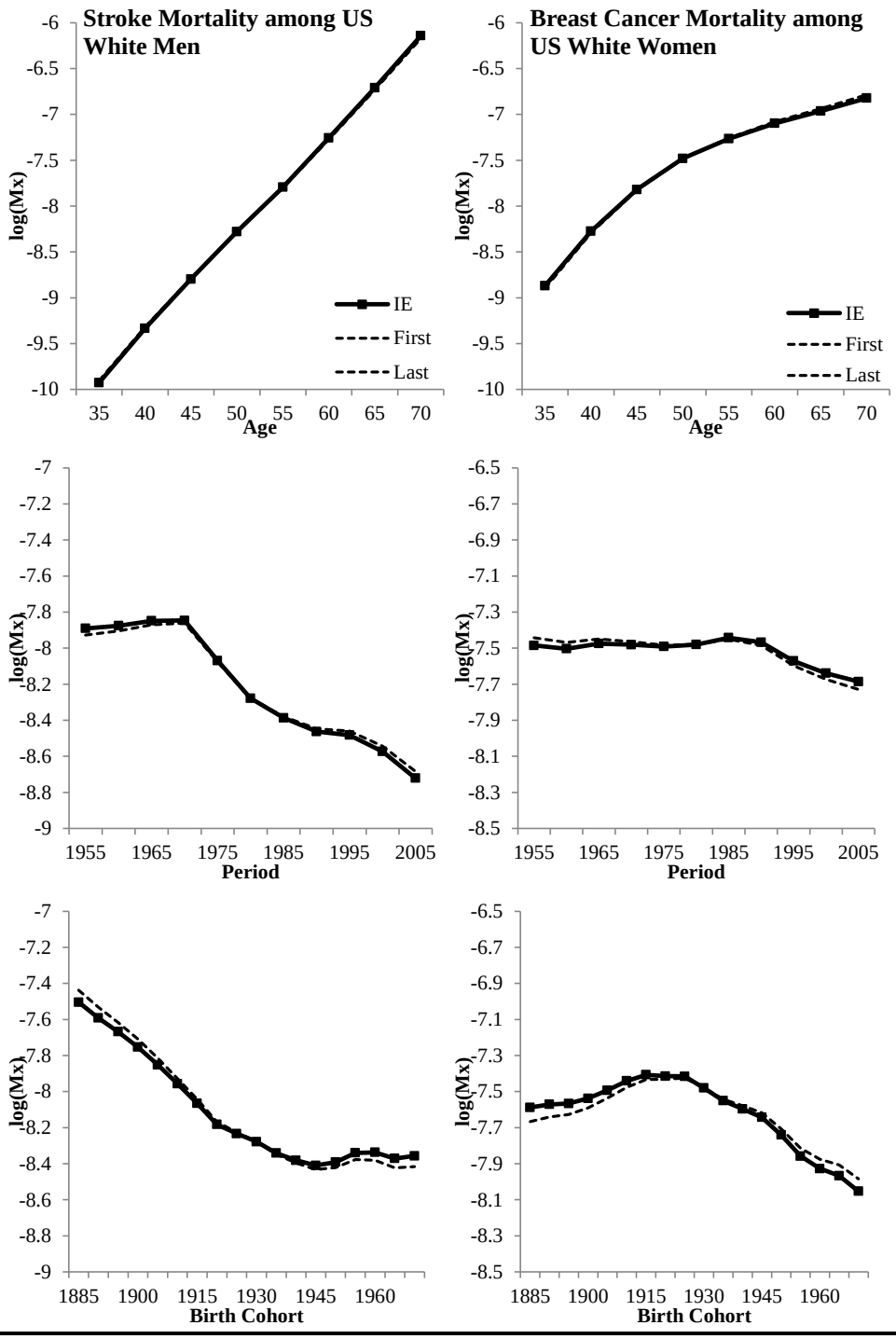
Taken together, we show: (1) Luo's criticisms of the IE apply to a very limited set of conditions (i.e., linear functional forms of all APC effects), which likely do not exist in real-life data structures, (2) our data are not subject to these conditions, and (3) using the very methods Luo used to advance her critique of the IE,

we show that the IE is able to estimate APC coefficients from simulated data that are consistent with and not significantly different from the effects estimated from the models used in our paper.

Sensitivity Analysis 2: Alternative Reference Category

Next, we recognize that the IE has been subject to additional criticism from researchers familiar with APC modeling techniques, beyond the criticisms raised by Luo. Specifically, on the one hand, Powers (2012) introduced a Stata module (ie_rate) and showed that Yang, Fu, and Land's (2004) intrinsic estimator fits within a longstanding statistical tradition of estimation techniques relating to ill-conditioned models, including ridge regression and principal components regression. In a 2013 paper, Powers uses these modules to analyze age-, period-, and cohort-based variation in US black and white infant mortality rates over the past several decades. Elsewhere, Powers has also shown that the IE produces different age, period, and cohort effects depending on the choice of reference category. That is, the amount of age-, period-, and cohort-based variation in the outcome estimated by the IE is often influenced by the choice of the age, period, and cohort reference categories. This issue is similar to the one raised by Held and Riebler (2013) in their commentary on Luo (2013), and is an issue that has been recognized in other APC applications (e.g., Tu, et al. [2012]). In contrast to Yang et al.'s (2008) apc_ie program, which permits only one APC normalization, Powers's (2012) ie_rate module allows the researcher to entertain numerous possible parameterizations of the APC effects, and thus gauge the sensitivity of APC results to alternative coding schemes. To address the severity of this concern in our estimates, we used Powers's ie_rate Stata module to fit APC IE models with two different parameterizations of the reference categories: the "first" category level of the age, period, and cohort factors set as the reference and the "last" category level of the age, period, and cohort factors set as the reference, while retaining the sum-to-zero constraint on each of the APC factors (Yang et al.'s ([2008] apc_ie program uses the last level of each APC factor in its sum-to-zero ANOVA-type coding). Thus, we are able to see the discrepancies in estimated age-, period-, and cohort-based variation in US adult mortality between the alternative coding schemes. For all models, we find an exact correspondence between results from models fitted using apc_ie and results from models fitted using ie_rate when the last factor level of each APC factor is used as the referent. More importantly, we find very little variation in the estimated APC effects from models fitted using the first categories as references and models fitted using the last categories as references. In Figure S4 we present age-, period-, and cohort-based variation in estimated log mortality rates for US white men from stroke and for US white women from breast cancer.

Figure S4. APC Effects Estimated from Alternatively-coded IE Models Fitted to US White Men's Stroke Mortality and US White Women's Breast Cancer Mortality.



Results show the APC effects estimated from IE models fitted to these data do not vary significantly when changing the age, period, and cohort reference categories.

Sensitivity Analysis 3: Markov Chain Monte Carlo Hierarchical Cross-classified Random Effects Models

Finally, we compare the results from our APC IE models with results estimated using a Markov Chain Monte Carlo (MCMC) approach to fit Hierarchical Age-Period-Cohort (HAPC) Cross-classified Random Effects Models (CCREM). These results are entirely consistent with our IE-based results for all-cause mortality, across all causes of death, and for all subgroups of the population. Below, we present age-, period-, and cohort-based variation in estimated log mortality rates for US black women from infectious diseases and for US black men from heart disease.

Figure S5. APC Effects Estimated from Fitting Models using HAPC-CCREM and IE for US Black Women's Infectious Disease Mortality and US Black Men's Heart Disease Mortality.

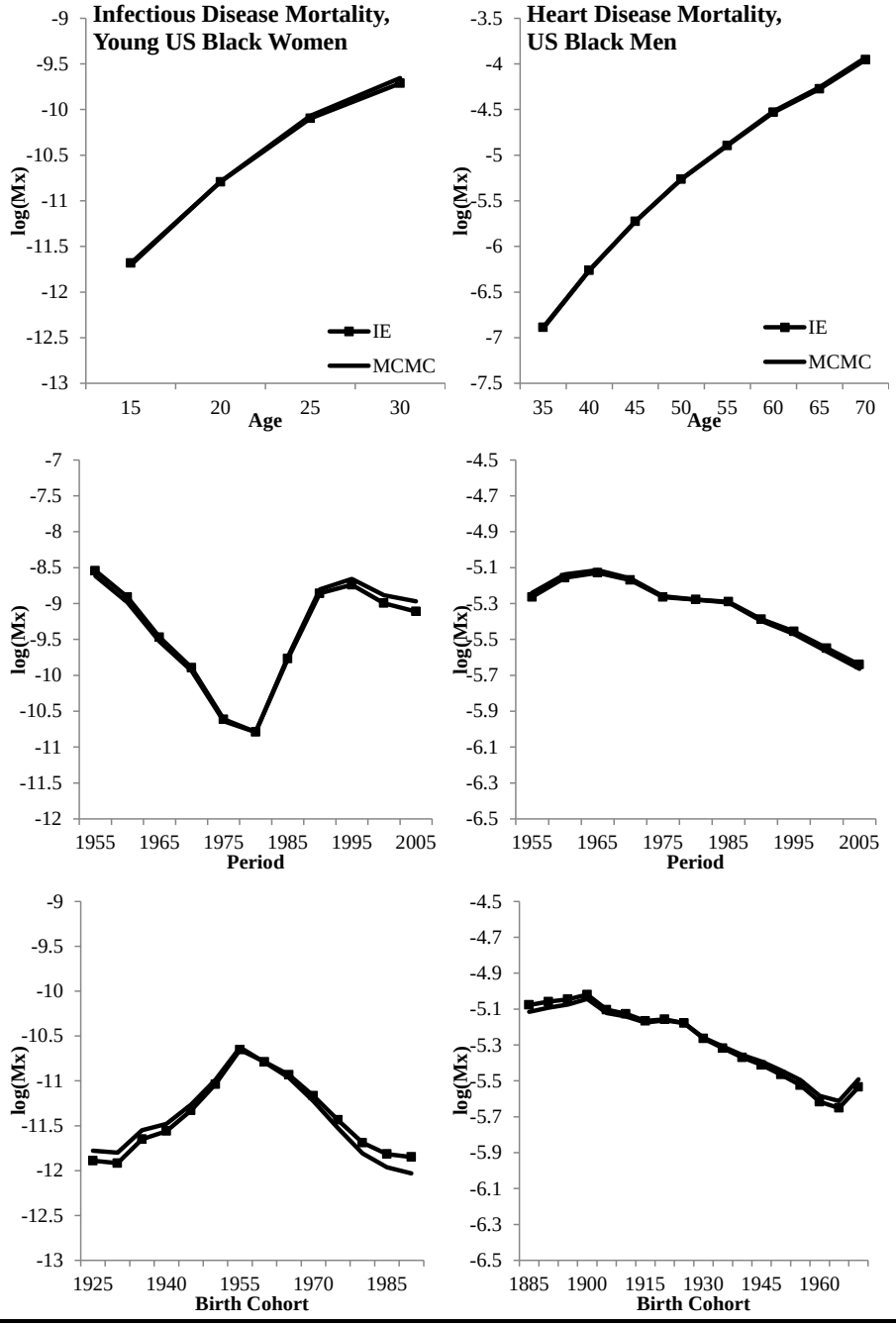


Table S4: IE Estimates of Age, Period, and Cohort Effects on U.S. Adult Women's & Men's All-Cause Mortality Rates, 1959-2009.

	Black Women		White Women		Black Men		White Men	
	b	z	*b*	z	*b*	z	*b*	z
Age								
15-19	-1.84	-370.8	-1.42	-632.7	-1.58	-519.6	-1.30	-923.1
20-24	-1.38	-351.3	-1.37	-650.1	-1.03	-429.5	-1.04	-851.8
25-29	-1.10	-311.8	-1.31	-633.4	-0.90	-381.9	-1.11	-866.0
30-34	-0.80	-260.2	-1.09	-586.3	-0.73	-330.8	-1.02	-828.1
35-39	-0.48	-179.5	-0.75	-473.8	-0.50	-250.5	-0.78	-708.0
40-44	-0.15	-64.0	-0.35	-263.9	-0.24	-131.7	-0.44	-463.8
45-49	0.17	83.4	0.07	62.7	0.07	39.7	-0.03	-43.0
50-54	0.50	264.3	0.47	486.7	0.39	257.6	0.38	543.4
55-59	0.80	451.9	0.85	1000.9	0.69	481.2	0.78	1254.9
60-64	1.14	680.4	1.25	1625.9	1.01	726.0	1.17	2067.2
65-69	1.40	849.6	1.63	2253.4	1.26	909.5	1.53	2827.2
70-74	1.73	1051.8	2.04	2904.6	1.56	1117.9	1.88	3569.9
Period								
1955-1959	0.13	31.1	-0.02	-12.0	-0.12	-34.2	-0.02	-13.4
1960-1964	0.19	80.6	0.01	13.1	0.01	4.3	0.02	24.5
1965-1969	0.15	77.8	0.06	63.9	0.11	67.2	0.09	136.9
1970-1974	0.08	41.2	0.06	62.7	0.13	79.7	0.10	154.0
1975-1979	-0.08	-40.6	-0.02	-12.3	0.03	22.1	0.04	60.6
1980-1984	-0.11	-60.1	-0.04	-42.7	0.00	-3.0	0.00	7.6
1985-1989	-0.08	-40.9	-0.03	-34.3	0.05	36.1	0.01	8.7
1990-1994	-0.06	-30.5	-0.03	-40.2	0.08	55.5	-0.01	-9.7
1995-1999	-0.05	-27.0	-0.01	-14.2	-0.02	-10.9	-0.05	-73.6
2000-2004	-0.06	-34.8	0.01	14.9	-0.11	-73.9	-0.08	-121.2
2005-2009	-0.12	-60.6	0.01	12.9	-0.17	-108.2	-0.11	-158.1
Cohort								
1885-1894	0.34	32.2	0.59	167.3	0.33	35.2	0.49	168.9
1890-1899	0.32	66.2	0.50	285.5	0.34	77.1	0.45	319.6
1895-1904	0.33	89.1	0.41	298.2	0.32	100.0	0.42	390.3
1900-1909	0.46	148.0	0.34	270.3	0.32	116.8	0.38	396.4
1905-1914	0.37	129.6	0.26	224.0	0.24	94.8	0.34	382.2
1910-1919	0.33	123.6	0.24	217.3	0.24	103.9	0.29	352.3
1915-1924	0.28	111.7	0.23	214.3	0.20	93.8	0.23	288.4
1920-1929	0.28	114.9	0.19	185.5	0.19	94.8	0.14	176.0
1925-1934	0.26	109.7	0.16	152.7	0.17	84.4	0.09	109.4
1930-1939	0.17	71.7	0.10	96.5	0.09	46.1	0.01	14.0
1935-1944	0.09	39.6	0.02	21.7	0.05	24.1	-0.08	-97.8

Table S4 Continued

Cohort								
1940-1949	0.01	3.3	-0.06	-47.1	-0.01	-5.2	-0.17	-180.6
1945-1954	-0.07	-26.0	-0.14	-102.6	-0.05	-23.9	-0.21	-217.8
1950-1950	-0.10	-35.6	-0.21	-136.0	-0.05	-23.0	-0.18	-177.3
1955-1964	-0.11	-36.2	-0.20	-116.8	-0.12	-53.4	-0.12	-109.1
1960-1969	-0.17	-50.5	-0.18	-96.4	-0.25	-101.6	-0.12	-97.7
1965-1974	-0.28	-67.6	-0.22	-94.8	-0.34	-118.6	-0.18	-122.1
1970-1979	-0.38	-75.5	-0.29	-103.0	-0.36	-107.4	-0.28	-154.0
1975-1984	-0.45	-71.6	-0.34	-97.3	-0.28	-74.6	-0.30	-138.9
1980-1989	-0.49	-66.2	-0.37	-92.6	-0.32	-73.5	-0.31	-126.1
1985-1994	-0.56	-56.9	-0.44	-88.8	-0.37	-65.8	-0.34	-115.9
1990-1999	-0.62	-35.4	-0.60	-72.3	-0.34	-35.4	-0.53	-97.7
Intercept	-5.32		-5.94		-4.63		-5.2	
Log Likelihood	7593.8		13170.8		26458.0		41767.7	
BIC	-14919		-26073		-52647		-83267	
df	42		42		42		42	
N	522,247,989		3,666,814,918		460,667,686		3,574,541,743	
Deaths	3,173,102		16,132,557		4,584,013		26,449,147	