

## Temporal Changes in Self-Rated Health: APC Models of Racial Disparities

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**Abstract:** This paper describes and attempts to explain temporal patterns in racial health disparities using appropriate models that simultaneously consider the unique effects of age, period, and cohort. First, we employ cross-classified random effects models of demographic age-period-cohort (APC) models in order to understand the patterns of self-rated health disparities over time. Second, we use decomposition models to understand how compositional shifts across cohorts with respect to socioeconomic status, marital status, and a host of other individual factors explain the disparities. Third, we examine the extent to which cohort-level measures of relative cohort size and economic and health conditions at birth explain the racial disparity in health. Cohort disparities increase through the 1935 cohort for women, falling thereafter; disparities for men exhibit a similar pattern but begin their overall decline with cohorts born earlier in the century. Differences in socioeconomic composition consistently contribute to disparities across cohorts; notably, disparities in marital status emerge as an increasingly important predictor across cohorts for women whereas disparities in employment emerge as increasingly salient across cohorts of men. Finally, our cohort characteristic models suggest that cohort economic conditions (percent large family, farm or Southern birth) reduce both male and female disparities in health. Poor macro economic conditions around the time of the great depression inflated disparities for those cohorts while more favorable conditions following World War II suppressed disparities; relative cohort size had no impact on cohort disparities in health.

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## **Introduction**

Health disparities are an intractable, but not inevitable feature of the American stratification system. Sub-population groups that bear the brunt of poverty, marginalized labor, discrimination, wage gaps, and segregation also bear the brunt of poor health (Williams and Collins 1995). This facet of American life has led to an increased focus on health disparities; in fact, Healthy People 2020 (HP2020) put forth a number of lofty goals for the health of our nation. While many gains were made towards the goals of increasing quality and years of healthy life, the United States fell well short of meeting another goal: to eliminate health disparities across subgroups of the population (DHHS 2011). One of the primary reasons why the United States did not reach this goal is most certainly our lack of understanding of the social processes that generate health disparities and how these disparities persist amidst overall declining morbidity and mortality rates (Hummer and Chinn 2011; Williams and Sternthal 2010). It is clear, for example, that genetic differences between population groups do not offer the answer to the question of persistent disparities (LaVeist 1994; Cooper 1986; Frank 2007). Consequently, investigations into possible social, economic, and structural explanations are necessary in order to better understand the origins and causes of contemporary health disparities.

One of the most disconcerting findings with regards to health disparities is the persistent Black-White gaps in U.S. health, morbidity, and mortality. Despite declining morbidity and mortality over time, researchers continue to find significant racial gaps persisting for a host of outcomes, including self-rated health, chronic conditions, disability, overall life expectancy, and age- and cause-specific mortality (Geronimus et al. 2001; Hayward et al. 2000; Hummer and Chinn 2011; Williams and Sternthal 2010). Such disparities are very well documented on a year-to-year, or cross-sectional basis and have also increasingly been tied to life course processes that unfold by age (Geronimus et al. 2010; Walsemann et al. 2008). Yet rarely are all temporal considerations of racial health disparities accounted for. That is, while many studies have documented how black-white health disparities change both across period time (e.g., Harper et al. 2007; Macinko and Elo 2009) and by age across the life-course (e.g., Geronimus et al. 2006; Jackson et al. 2011), research has tended to focus either on one or the other, with very little research considering both period time and life course processes simultaneously (Colen 2011). Moreover, very little attention in this literature has been given to possible cohort-based differences in health change; that is, are black-white gaps in health structured by the cohorts in which individuals are born, in addition to the effects of period time and age? With this limitation in mind, the intent of this paper is to describe black-white health disparities in the United States using modeling techniques that simultaneously estimate the independent effects of age, period, and cohort. We employ cross-classified random effects models (e.g. Yang and Land, 2006) to estimate temporal patterns of U.S. racial disparities in self-rated health. We then use decomposition methods to analyze how cohorts' compositional shifts in socioeconomic status, marital status, and a host of other individual factors explain the observed disparities. Finally, we consider potentially salient

cohort characteristics to understand their role in explaining historical disparities. First, we describe each temporal component (APC) in order to describe the unique contribution of each to population health disparities.

## **Background**

Much extant research on population health disparities has focused on the aging process as it relates to health outcomes (e.g., Manton and Gu 2001). Indeed, age is an indicator of the biological aging process which brings about internal physiological change due to an accumulation of exposure to pathogens, genetic manifestation of disease, and the biological breakdown of the human body (Yang and Land 2006). On average, increasing age is associated with health declines at the individual level and an increase in mortality rates at the population level. Further, while some research has recognized a closing black-white mortality gap among older adults (Jackson et al. 2011; Wing et al. 1985), other research suggests a growing racial disability gap with age (Freedman and Martin 1998; Schoeni et al. 1997). This focus on aging is obvious and appropriate.

Moreover, the persistence of health disparities over time suggests that social forces are operating that replicate themselves as individuals die and as other individuals are born into a social system. Hence, *period effects*—temporal social contexts that affect all age groups simultaneously, and *cohort effects*—unique conditions attributed to individuals within defined birth-year groupings--both play crucial roles in driving the dynamic patterns of health over time. Period and cohort in particular may be especially salient to understanding racial health disparities given that race represents differential access to social resources and differential risk-exposure (Colen 2011; Williams and Sternthal 2010). Unfortunately, these two temporal dimensions are often ignored in health disparities research, with some attention being paid to period effects through the analysis of national surveys over time. Previous research using the 1982-2007 IHIS suggests that period trends in poor/fair self-rated health declined during the 1980s, increased during the early 1990s, only to flatten out after a sharp decline in 1997 simultaneous with a redesign in the survey (Saloman et al. 2009). Blacks have experienced steeper declines in poor/fair self-rated health than Non-Hispanic whites, resulting in a narrowing disparity (Saloman et al. 2009). Only when the separate effects of age, period, and cohort are estimated simultaneously can we begin to uncover the sources of health disparity trends. Before elaborating on the potential contribution of age-period-cohort (APC) analyses to understanding the sources of changing health disparities over time, we now turn to a brief discussion on period and cohort effects and how they may have independent impacts on population health.

Period effects represent changes over time that affect the entire population simultaneously, but perhaps not equally. That is, while a period effect may be simultaneous for all members of a given population, they may affect sub-population groups (e.g., racial groups) quite differently. For example, it is possible that a large year-to-year increase in unemployment may affect those with less than a high school education more so than college graduates. Other possible period effects include

historical events such as wars, labor market characteristics, economic fluctuations, infectious disease outbreaks, diffusions of medical technology, and the development of new medical treatments and procedures (Yang 2006).

On the other hand, individuals born during similar periods and who enter into pre-existing social systems and experience similar formative environments can be defined as a birth cohort. Cohort effects—defined in this analysis as *birth cohorts*—represent variation between groups of individuals who are born in different years and experience different formative environments (Yang and Land 2008). While birth cohorts move through life together and experience similar historical and social events, “cohorts that experience different historical and social conditions differ in their exposure to socioeconomic, behavioral, and environmental risk factors. Cohort effects represent the effects of these factors that embark at the moment of exposure early in life and act persistently over time to produce health and mortality risk differences in specific cohorts” (Yang 2006, p. 2). But a major limitation in the health disparities literature is that few studies include cohort effects, while many more are focused on age and/or period effects.

However, not *all* members of the same birth cohort experience the same social processes and experiences. This is why the United States is wrought with persistent health disparities even within very new (and young) birth cohorts, such as the persistent racial gaps in low birth weight and infant mortality (Powers et al. 2006; Powers and Song 2009). In fact, with each successive birth cohort, there exists the opportunities for two sources of change. One, the cohort itself is made up of a unique set of individuals with social-, cultural-, and economic- capital who will generally inherit levels bestowed upon them by the previous generation but can also learn from the previous generation and create social change themselves (Mason and Fienberg 1985). Two, each successive cohort is confronted with a social environment that is partially resistant to change and will bestow *the same* types of risk and rewards upon population sub-groups, but these risk/reward regimes may also change over time (Mason and Fienberg 1985). Thus, studies of birth cohorts allow researchers to account for both the dynamic change in the composition of individuals in population sub-groups (such as racial groupings) and allows us to account for social change and the implications of social change on the health of each new generation. While dynamic change is obviously a part of historical process, the relatively conservative process of population replacement, replication, and socialization implies that social forces will continue to play themselves out even among constantly changing cohorts of individuals (Ryder 1965). Cohort differences in health and mortality have also been attributed to birth cohorts’ disparate lifetime exposures to pathogens, improvements in nutrition, and advances in health knowledge and medical technologies (Finch and Crimmins 2004; Fogel 2005; Manton, Stallard, and Corder 1997; Masters forthcoming; Yang 2008). That is, on the one hand, U.S. cohorts born in the first half of the twentieth century were exposed to higher rates of infectious diseases during childhood than were subsequent birth cohorts. On the other hand, during their adult years these same cohorts were less exposed to major advances in pharmacological, surgical, and other medical technologies. As such, the cohort represents a

unique socio-demographic concept which suggests that while dynamic change is possible, social forces play out differently on each successive birth cohort. That is, patterns may reflect persistent social forces that cause health disparities, but may also present a unique opportunity to study how cohorts change over time and ultimately affect the health of racial minorities.

Certainly, health disparity researchers have focused a great deal on changes over time, but have largely focused on changes over *interview years* from repeated cross-sectional data as the only source that contributes to the temporal dynamics of health. Subsequently, as demographers and sociologists have demonstrated, it is *crucial* to distinguish between age, period, and cohort when considering dynamic social change over time (Masters forthcoming; Ryder 1965; Yang 2008). The health of populations is no exception. Each temporal dimension has a potentially dynamic and unique effect on population health and assessing the impact of one temporal dimension while controlling for other dimensions is an *essential* part of any analysis that investigates health change over time and reflects these changes accurately.

In spite of the conceptual relevance of age-period-cohort effects for studying health trends, this framework has not been widely used due to the statistical difficulty of simultaneously disentangling the effects of age, period, and cohort (Glenn 2005). However, through the use of repeated cross-sectional surveys and by grouping individuals into multiple-year birth cohorts, we are able to overcome the limitations of conventional APC analyses. Therefore, it is our intention to directly study both period and cohort effects on black and white adult health change in the United States, while simultaneously accounting for age, to best describe changing patterns of black-white health disparities.

#### *Explanations of Racial Disparities in Health*

Although race has both ethnic and structural components, race truly is best captured as “an important marker of differential access to societal resources and rewards, and health status is no exception” (Williams 2005, p. 53; also see LaViest 1994). It is our intention to consider race in this fashion, while recognizing the mediating pathways through which race is generally thought to operate to influence health. We turn briefly to a discussion of some of these mediating factors (socio-demographic factors and health-behavior profiles), before returning to a consideration of the social determinants of population health and health disparities.

Individual socio-economic status (SES), given its strong linkages with health (Link and Phelan, 1995) is the most compelling source of variation between racial/ethnic groups that might account for health disparities (House and Williams, 2000). Although some research suggests that disparities in health and mortality are completely accounted for by individual socio-economic status (Rogers, 1992), most research indicates otherwise—that is, that racial/ethnic disparities in health are substantially reduced net of SES but not completely eliminated (Crimmins, Hayward and Seeman 2004; House and Williams, 2000; Hummer and Chinn 2011). On the other hand, the determinants of racial/ethnic differences in SES remain the subject of a large body of social demographic research, much of which suggests that a significant portion of these differences are due

to residential context and job availability and quality (Huffman and Cohen, 2004). Another contributor to these persistent disparities has been differential rates in risky health behaviors; however, health behaviors have not been able to fully account for residual disparities (Finch, Frank, and Hummer, 2000; Lantz et al. 2001). Among other possibilities for black-white health disparities are discrimination (Gee and Ford 2011; LaVeist 2000; Williams, Neighbors, and Jackson, 2003), discrimination-related life course stress (Geronimus et al. 2010; Sternthal et al. 2011), differential incarceration (Schnittker et al. 2011), and racial residential segregation (House 2002; Williams and Collins, 2001). In fact, one recent study (Do et al. 2008) confirms the growing supposition that race is a proxy for exposure to differential social conditions and revealed that residential context may account for 15–76% of the African-American/White disparities in self-rated health that were previously unaccounted for by individual-level control variables.

#### *Cohort Characteristics Affecting Health.*

*Economic and Health Conditions at Birth.* Extant literature suggests that poor economic conditions early in life negatively impact health, cognitive functioning, and survival at older ages (Case et al. 2005; Van den Berg, Lindeboom, and Portrait, 2006; Van den Berg, Doblhammer, and Christensen, 2009; Doblhammer, Van den Berg, and Fritze, 2011). This may also include early exposure to infectious diseases which can lead to chronic inflammation which in turn influences health, including the risk of cardiovascular disease, and mortality (Finch and Crimmins, 2004; Crimmins and Finch, 2006; McDade et al. 2010). With respect to macro economic conditions, it is hypothesized that periods of recession, can act as a household stressor, reducing available resources, medical care and nutrition and increasing exposure to diseases. On the other hand, boom periods may ensure optimal infant (or fetal) nutrition, health and development. This early health then, in turn, is expected to have long-term implications for survival. Evidence suggests that these pathways may operate through epigenetic changes during critical periods of development; Barker (1997) argues that fetal undernutrition can lead to cell division deficiencies that increase the risk of cardiovascular disease. Related to household stressors, Miller and Chen (2010) find that being raised in harsh family environment is associated with a greater pro-inflammatory phenotype over time. Doblhammer and colleagues (2011) argue that the pro-inflammatory phenotype can create an allostatic toll resulting, long-term, in a higher risk of chronic diseases. As they are exogenous at the individual level, business cycle effects at birth on later health and mortality avoid the simultaneity bias of individual level socio-economic conditions and health and are argued to be causal in nature (Van den Berg, Lindeboom, and Portrait, 2006). With respect to disparities, we expect that economic conditions will exacerbate inequalities in light of both evidence suggesting that black employment is more sensitive to these downturns (Gilroy 1974) and because of inequities in the safety net of wealth accumulation.

*Relative Cohort Size.* Members of larger cohorts are expected to experience persistent disadvantages stemming, in part, from the consequences of increased economic competition once the cohort enters the labor market (e.g. higher

unemployment and lower wages) (Easterlin, 1978, 1987). O'Brien and colleagues also argue that large cohorts put extreme demands on the school system and community resources (O'Brien, Stockard and Isaacson, 1999). Beyond the stressors of economic competition, a natural extension is that larger cohorts also stress the health care system, having consequences for the quality and quantity of medical care available. Research indicates that cohort size is associated with a number of indicators of disadvantage, from homicide and property crime rates to individual earnings (O'Brien, 1989; O'Brien, Stockard and Isaacson, 1999; Welch, 1979). Although, we expect that the economic competition and scarce resources that arise from relative cohort size will be more detrimental among blacks, it is also possible that if blacks and whites are not frequently in competition over the same types of jobs relative cohort size will not explain any of the cohort disparity.

*Segregation.* Although not previously explored as a birth cohort factor, segregation and relatedly, discrimination has been found to be associated with poor birth outcomes (e.g. low birthweight), which is in turn associated with poor health such as cardiovascular disease (Bell et al. 2006; Grady, 2006; Mustillo et al. 2004). We also anticipate that segregation may act as a proxy for the disparity in living and housing conditions. Racial residential segregation can be a primary cause of racial disparity in socioeconomic status (SES is higher in better residential area), education opportunities (e.g. better public school in metropolitan area), employment opportunities (e.g. blacks live in the area where high paying entry-level jobs are not available), and even health behaviors (e.g. more swimming pools and playing fields in the economically advantageous residential area) (Williams and Collins, 2001). This may also include early exposure to infectious diseases which can lead to chronic inflammation which in turn influences health, including the risk of cardiovascular disease, and mortality (Finch and Crimmins, 2004; Crimmins and Finch, 2006; McDade et al. 2010).

Thus, beyond describing black-white health change across temporal dimensions, this paper also examines some of these major factors thought to account for black-white health disparities, particularly as those disparities are changing across periods and/or cohorts, as well as by age. While no study can possibly take into account the full range of factors that influence black-white health disparities, we focus here on some of the most important sociodemographic mediators thought to be responsible for the continued disparities.

## **Data and Measures**

### **Sample and Measures**

We utilize the Integrated Health Interview Series (IHIS) available through the Minnesota Population Center (Minnesota Population Center and State Health Access Data Assistance Center, 2010). This series is comprised of harmonized annual data, beginning in 1969, from the U.S. National Health Interview Survey (National Center for Health Statistics, NCHS, 2006). The harmonization of the IHIS data to conform to differential measurement and response categories over multiple NHIS years is described in detail on the IHIS website ([www.ihis.us](http://www.ihis.us)). However, when significant changes occurred, we

highlight measurement differences in the description of each of our covariates below. We use the harmonized data from 1972, which is when the question on self-rated health was first asked, up through 2009, the latest survey year currently available.

The NHIS is a cross-sectional household survey, covering the noninstitutionalized civilian population residing in the United States at the time of the interview. Given our primary interest in racial disparities, we restrict our sample to Non-Hispanic Whites and Blacks<sup>1</sup> who are not missing information on the dependent variable of self-rated health. We also restrict our analysis to those aged 25 and older; because most individuals 25 and above are beyond the age of typical college graduation, we can be more confident that their education level at interview is reflective of their ultimate attainment. Given smaller sample sizes for earlier cohorts, we exclude individuals born before 1893 (what we define as the beginning of the 1895 five-year cohort band). To ensure consistency with later waves of data, we excluded observations that rely on proxy reports between 1972 and 1996. With these restrictions, our final sample includes 1,304,201 Non-Hispanic Whites and 208,896 Blacks.

Our dependent variable is drawn from a measure of self-rated health available from 1972 continuously through 2009. Respondents who refuse to answer the question or respond “unknown” make up less than one percent of the sample in each wave and are dropped from the analysis. Beginning with the 1982 survey, both the question wording and the response categories changed. Before 1982, respondents were asked: “Compared to other persons [person’s] age, would you say that his health is excellent, good, fair or poor?” Thereafter, respondents were asked “Would you say that [person’s] health in general is excellent, very good, good, fair or poor?” To alleviate the comparability issue that arises due to differences in responses categories, we create a dichotomous measure of whether the respondent rated their health as fair or poor as compared to good, very good or excellent. There does not appear to be evidence of a discontinuity in the trend line in 1982; this suggests that this measurement issue does not unduly influence our results.

We chose this particular health measure for several reasons: (1) studies show that a poor or fair self-rating of health has strong predictive value for subsequent mortality above and beyond clinical assessments (Benyamini and Idler, 1999; Idler and Benyamini, 1997); (2) other studies have found relationships between poor SRH and subsequent functional decline and disability (Idler and Angel, 1990); (3) although evidence that SRH is associated with specific disease outcomes is limited (Ferraro, Farmer, and Wybraniec, 1997; Menec, Chipperfield and Perry, 1999), other studies suggests that SRH is associated with current morbidity (Ferraro and Farmer, 1999); and (4) SRH is often more sensitive to change in response to external factors than are physiologic parameters. In short, these studies find that SRH is a well-validated indicator of adult health and

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<sup>1</sup> Prior to 1976, Hispanic ethnicity was not asked; in these cases, individuals were coded according to their stated racial category. As an approximation of the severity of miscategorization, in the first few years the question is available Hispanics make up, on average, less than 5% and less than 3% of the White and Black racial categories, respectively.



mortality risk, above and beyond physician assessments, and that a single measure on a survey can help to capture a great deal of information about one's health status. Further, given large subsequent mortality risk differences between those who rate themselves in fair/poor health compared to good/very good/excellent health (Benjamins et al. 2004), there is solid empirical precedence for dichotomizing this variable. Further, other work has demonstrated that fair/poor self-ratings of health have similar mortality risks for blacks and whites and for both men and women within each of these racial classifications (McGee, Liao, Cao, and Cooper, 1999). This finding indicates that SRH is a valid measure of future mortality risk for our populations of interest, suggesting that our use of this measure for analyses of health disparities between blacks and whites is valid. Across all years of the data we use, 14.4% of men responded that they were in fair or poor health whereas 16.3% of women did so. There is also considerable diversity in health reports by race. For men, only 13.4% of Whites, but 22.0% of Blacks indicated they were in fair or poor health. Likewise, 14.7% of White women compared to 25.9% of Black women responded that they were in fair or poor health.

[Table 1 About Here]

We use a continuous measure of age as well as an age squared term. Age was top-coded at ninety-nine years old until 1996 and eighty-five years old beginning in 1997; we top-coded age in all waves to eighty-five<sup>2</sup>. The mean age of our sample is approximately 48 for men and 49 for women; Blacks are, on average, approximately one to two years younger (46.9 and 47.2, for men and women, respectively). Period, defined as survey year, is measured as a series of dummy variables with 1972—the first available year of self-rated health data—as the omitted category. We group individuals into 5-year cohort bands based on their year of birth to break the linear dependence between age, period and cohort. We define these cohorts by their midpoint; for example, the “1950 cohort,” which is used as the reference category, includes individuals born in 1948, 1949, 1950, 1951, and 1952. The average respondent comes from the 1940 cohort (born in 1938-1942).

We also include a number of indicators of socioeconomic status and household structure as well as a control for geographic residence. A series of dichotomous indicators capture marital status, including: *married*, *never-married* and *other* (widowed, separated, or divorced). Overall, 76.5% of men and 64.5% of women are married at the time of the survey; 11.1% and 9.0% of men and women are never-married, respectively and 12.0% and 26.1% of men and women are divorced, widowed or separated. Marital status differs markedly by race; Blacks are less apt to be married (only 60.8% of men and 41.3% of women), and more likely to be never-married, separated, divorced or widowed. For example, 7.2% of White women are never-married compared to 20.0% of Black women and 24.1% of White women are divorced, separated or widowed relative to 37.7% of Black women. A continuous measure of household size was also included; on average family

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<sup>2</sup> We also estimated age-restricted models to age 84 which showed a similar pattern of results. Models without a standardized top code yielded slightly more modest age disparities among men, but all other results were similar.

size is approximately 3 individuals. Regional indicators control for residence in the West, South, Northeast, and North Central/Midwest (West=reference). Blacks are more likely to reside in the South and less likely to reside in the West relative to Whites. An individual's highest level of education is captured by: less than a high school degree, high school degree, some college, and college degree or higher. Approximately 57.5% of men and 62.9% of women reported having a high school degree or less whereas 23.5% of men and 17.4% of women reported having a college degree. Although the proportion with only some college experience is similar by race, there is greater inequity at the tails of the educational distribution. Blacks are less likely to have a college degree and more likely to have less than a high school degree. Respondents are asked whether they were part of the labor force during the preceding one to two weeks (before 1997 the reference period is two weeks, thereafter it is one week). From this question we created indicators of employment: employed (includes "has a job but is not currently working"), unemployed but looking, and unemployed, not looking for work. We also consider anyone sixty-five and older who is not employed or looking for work retired. Compared to men, women are less likely to be working and more apt to be out of the labor force (unemployed and not looking for work). White and Black women have similar employment profiles, with Black women slightly more likely to be employed. In contrast, there are large racial differences in employment among men. Black men are less likely to be employed (67.3%) relative to White men (74.7%), and more likely to be unemployed and not looking for work. We also include a measure of family income adjusted for household size and Consumer Price Index<sup>3</sup>. We include indicators of missingness for marital status and income, which are missing for 0.3% and 11-12% of respondents, respectively. Racial disparities in income are apparent despite our truncated income distribution; Black men have family incomes that are, on average, approximately \$2,500 less than White men whereas Black women have family incomes that are on average \$4,000 less than White women. We examine six measures of cohort characteristics (See Appendix Chart 1): relative cohort size, health and economic conditions at birth. Following O'Brien and colleagues (1999), we measure relative cohort size by the percent of those 15 to 64 who are 15-24 when the cohort entered the labor market (age 15 to 19). This captures the relative size of the cohort to the overall labor force pool as well as accounting for crowding or competition for jobs from those slightly older. This measure is limited in that we cannot capture the extent to which blacks and whites may not be competing for the same jobs or the same pool of resources within any given cohort size (i.e. for school funding, hospitals, etc). Following Doblhammer, van den Berg, and Fritze (2011) and a broader literature on measures of aggregate economic conditions, we capture economic conditions at birth using the natural logarithm of the cyclical

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<sup>3</sup> As only interval level family income is available for the range of years we utilize, we first recode each category to its midpoint. Unfortunately, the top code for family income changed over time. Until 1981, the highest possible income category was "\$25,000 or more"; whereas in later waves the top code was \$50,000 or more (through 1996), \$75,000 or more (through 2006), and \$100,000 thereafter. We allowed top codes to vary over time; results with a consistent top code (\$25,000) yielded similar results. We then adjust income for economies of scale by dividing family income by the square root of household size. We then rescale for inflation dividing income by  $(CPI_{\text{year of survey}}/100)$ , with 1982-1984 as the reference point (Bureau of Labor Statistics, 2010).

component of the real GDP per capita. To obtain these estimates we apply the Hodrick-Prescott filter with smoothing value of 500 to time series data derived from historical GDP estimates (Maddison, 2010). Using IPUMS decennial census data (Minnesota Population Center) from 1900 through 1980, we create race-specific measures of the % of children under a year old whose parents report they were born in the South, the percent living in large families (6 or greater members), and the percent living on a farm. We linearly interpolate measures between censuses. We also used historical infant mortality rate tables from the National Vital Statistics System (Lindner and Grove, 1943; Grove and Hetzel, 1968; CDC/National Center for Health Statistics, 2012) to construct our cohort measures of infant mortality rates. Before 1933, rates include only those states that had death registrations, thereafter all states are included. Rates specific to the black population are not available before 1960, available inconsistently during the 1960s, and consistently after 1968. Although there is not a sizable discrepancy between rates in 1959 when all non-Whites were aggregated and 1960 when Blacks were listed separately, the infant mortality rates that we use to approximate black IMRs before 1960 are biased by the inclusion of other non-White infants. For each cohort characteristic, we standardized the measure to allow comparisons across measures.

## **Analytic Methods**

### The Identification Problem

Limitations to the conventional strategies using an APC framework are widely known. The difficulty in estimating separate age, period, and cohort effects simultaneously is owed to the model identification problem. That is, there is an exact linear dependence between age, period, and cohort:  $\text{Period} = \text{Age} + \text{Cohort}$ . This implies that infinite possible solutions exist for regression coefficient estimates (Glenn 2005).

Most extant APC analyses use aggregate population level data and have attempted to solve the identification problem in a variety of ways. As identified by Yang and Land (2008), the most common strategies include: (1) placing at least one additional identifying constraint on the parameters to be estimated (e.g., constraining the cohort effects to be equal and constant across time) (Mason et al. 1973; Fienberg and Mason 1978, 1985), (2) assuming that the cohort or period effects are proportional to certain measured variables (Fienberg and Mason 1985; Heckman and Robb 1985; O'Brien 2000) and (3) transforming at least one of the age, period, or cohort variables so that the relationship is no longer linear (Mason et al. 1973; Fienberg and Mason 1985). The well recognized limitations to these strategies are the sensitivity of model estimates to the often arbitrary choice of the identifying constraints and the possible inappropriateness of assuming a non-linear relationship of either age, period, or cohort for particular health outcomes. In the absence of strong a priori theory that supports specific constraint specifications or a non-linear relationship, an analytical strategy that does not impose either of these restrictions is preferred.

To break the linear dependence between age, period, and cohort, we group survey respondents across multiple years according to a five-year birth cohort (e.g., 1938-1942 as the 1940 birth cohort). For example, a 60 year old in the 1940 birth cohort may have been interviewed in any year (i.e. period) between 1998 and 2002. With this coding scheme we assume that there is little to no variation within these 5-year cohort groupings, which may lead to slightly biased period and aging effects. However, it should be noted that the aggregation of individuals into virtually any birth cohort makes the same type of assumptions and risks potential bias given the potential variability within any given cohort aggregation.

*Cross-Classified Hierarchical Models for Binary Outcomes*

We first examine U.S. gender-specific health patterns across time, adjusted for individual-level characteristics, to determine if there are cohort and period effects and if these effects change across time. In addition to a series of socio-demographic factors, we also adjust for age and age squared in order to account for age effects; models are separately specified by gender. Because the data has a multilevel structure (individuals nested within periods and cohorts), we estimate cross-classified hierarchical models in order to account for the possible non-independence of individuals within cohorts/periods who may share similar attributes or experiences unique to their cohorts or periods of the surveys. While standard regression models (e.g., OLS) may underestimate the standard errors, leading to inflated t-statistics, the mixed-effects model approach accounts for this possible non-independence between survey respondents and will adjust for the standard errors appropriately. A cross-classified model is appropriate because individuals are not strictly nested within periods or vice-versa. For example, individuals within specific cohorts may or may not be nested within the same periods.

Relying on the notation and description from Yang & Land (2006), our race-specific models of self-rated health (SRH) will be specified as follows:

$$SRH_{ijk} = \beta_{0jk} + \beta_1 age_{ijk} + \beta_2 x_{ijk} + e_{ijk}, \quad e_{ijk} \sim N(0, \sigma^2) \quad \text{Level 1} \quad \text{Equation 1}$$

$$\beta_{0jk} = \gamma_0 + u_{0j} + v_{0k}, \quad u_{0j} \sim N(0, \tau_u), \quad v_{0k} \sim N(0, \tau_v) \quad \text{Level 2} \quad \text{Equation 2}$$

for  $i=1, 2, \dots, n_{jk}$  individuals within cohort  $j$  and period  $k$ ;

$j = 1, \dots, J$  birth cohorts;

$k = 1, \dots, K$  time periods (survey years);

where, within each birth cohort  $j$  and survey year  $k$ , respondent  $i$ 's self-rated health is modeled as a function of his age, race,

and a vector,  $X$ , of individual-level characteristics (e.g., income, labor force status, education).  $\beta_1 \dots \beta_v$  are the level 1

fixed effects and  $e_{ijk}$  is the random individual effect that is assumed to be normally distributed with mean 0 and a within-cell

variance  $\sigma^2$ . At level 2,  $\beta_{jk}$  is the intercept, which in this model, represents the mean fair/poor self-rated health of individuals who belong to birth cohort j and surveyed in year k.  $\gamma_0$  is the model intercept, or grand-mean health level of all individuals;  $u_j$  is the residual random effect of cohort j (i.e., the contribution of cohort j averaged over all periods on  $\beta_{jk}$ ) and is assumed to be normally distributed with mean 0 and a within-cell variance  $\tau_u$ ; and  $v_k$  is the residual random effect of period k (i.e., the contribution of period k averaged over all cohorts) and is assumed to be normally distributed with mean 0 and a within-cell variance  $\tau_v$ . As in all hierarchical mixed-models, we can partition the variance in health outcomes into components at the individual and higher level factors. In this case, the proportion of health variation due to cohort effects can

be calculated as:  $\frac{\tau_u}{\sigma^2 + \tau_u + \tau_v}$ . Similarly, the proportion of variance due to period effects is:  $\frac{\tau_v}{\sigma^2 + \tau_u + \tau_v}$ . If the model does not adjust for individual-level covariates, these proportions would reflect the gross variances due to either period or cohort effects. These measures can be used to estimate the possible overall relative contributions of individual, period, and cohort factors in explaining the variation of health across time. When the estimated model includes individual-level covariates (such as Equation 3.1), these proportions would reflect the proportion of variances due to either period or cohort effects, net of individual-level characteristics. To aid interpretation of these models, we generate the predicted probabilities of fair/poor health from recovered random cohort and period effects which have been adjusted by the covariates held at their mean (more information is also available upon request).

#### Decomposition Models for a Nonlinear Outcome

Based on a fully adjusted regression model which simultaneously controls for individual-level covariates in addition to age and period, we decompose these disparities by cohort to determine the relative contribution of each covariate to the observed health disparities<sup>4</sup>. We now briefly outline this decomposition approach, in which self-rated health (fair/poor versus other) between Blacks and Whites in a single cohort (1950) is compared. While decomposition methods for linear models are straightforward and widely used, only recently have methodological advances extended these approaches to nonlinear models (Fairlie 1999; Fairlie 2005). We now briefly outline this decomposition approach, in which hypertension (hypertensive versus other) between blacks and whites in a single cohort (1950) is compared. A nonlinear decomposition of the regression model is as follows, as adapted from Fairlie (1999):

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<sup>4</sup> These cohort-specific models decompose the unadjusted predicted probability gap in fair/poor health; to more closely approximate the CCREM models, we have included controls for period and age. The unadjusted cohort disparities from the decomposition models closely resemble the disparities from a CCREM intercept-only model (not shown).

$$\bar{Y}_{W50} - \bar{Y}_{B50} = \left[ \sum_{i=1}^{N^{W50}} \frac{F(X_i^{W50})\beta^{W50}}{N^{W50}} - \sum_{i=1}^{N^{B50}} \frac{F(X_i^{B50})\beta^{W50}}{N^{B50}} \right] + \left[ \sum_{i=1}^{N^{B50}} \frac{F(X_i^{B50})\beta^{B50}}{N^{B50}} - \sum_{i=1}^{N^{B50}} \frac{F(X_i^{B50})\beta^{W50}}{N^{B50}} \right] \quad \text{Equation 3}$$

where  $\bar{Y}_{W50}$  is the mean probability of hypertension of whites in 1950 and  $\bar{Y}_{B50}$  is the mean probability of hypertension for

blacks in the 1950 cohort. The first term,  $\left[ \sum_{i=1}^{N^{W50}} \frac{F(X_i^{W50})\beta^{W50}}{N^{W50}} - \sum_{i=1}^{N^{B50}} \frac{F(X_i^{B50})\beta^{W50}}{N^{B50}} \right]$ , represents the contribution of

compositional changes to the overall change in the probability of reporting poor health between whites and blacks. That is, it is the difference in rates of poor/fair health had blacks faced the same returns to risk —measured by individual-level covariates such as marital status, education, and income—as Whites in the 1950 cohort. The second term,

$\left[ \sum_{i=1}^{N^{B50}} \frac{F(X_i^{B50})\beta^{B50}}{N^{B50}} - \sum_{i=1}^{N^{B50}} \frac{F(X_i^{B50})\beta^{W50}}{N^{B50}} \right]$ , is the portion of the difference due to changes in the effects of the coefficients

for the measured covariates. Specifically, it assesses the contribution to changes in rates of poor/fair health that would have occurred if black returns to risk equaled those of whites in the 1950 cohort and if group characteristics were held fixed at the white levels. Given that the second term often includes the influence of any unmeasured covariates, we focus on the influence of compositional factors in explaining the racial health gap. This decomposition strategy is repeated and summarized across the range of our birth cohorts.

## Results

Given the length and level of detail in each regression model, we do not report the results of all regressions, but rather summarize the results in a series of graphical displays<sup>5</sup>. Given the structure of the data (i.e. the oldest cohorts are not observed at young ages and, likewise, the youngest cohorts are not observed at the oldest ages) as well as concerns with differential mortality, we use caution in interpreting patterns in the cohort tails as well as patterns for older individuals.

Figures 1, 2, and 3 present overall trends in self-rated health in terms of predicted probabilities of fair/poor health by age, period and cohort, respectively. Each figure shows predicted probabilities of poor/fair health for both U.S. adult men and women after adjustment for the other age-period-cohort components and after including covariates. Figure 1 illustrates the predicted probabilities for fair/poor health by age for men and women. Before including covariates (dotted black and gray lines), the predicted probabilities of poor/fair health for both men and women increase steadily with age; gender differences are minimal. After controlling for covariates (solid black and gray lines), the age probabilities show a curvilinear pattern with less pronounced increases at older ages and higher probabilities for women relative to men after age 35<sup>6</sup>. In Figure 2, the

<sup>5</sup> These regression results are available from the authors upon request.

<sup>6</sup> We considered various functional forms for the age variable, including: age; age + age<sup>2</sup> + age<sup>3</sup>; and age fixed effects. We found that age + age<sup>2</sup> to be the most parsimonious and robust to various categorizations of period; our results were very similar regardless of the age transformation.

predicted probabilities of fair/poor health by period are shown. Controlling for age, there is an extremely modest decline across periods for both women and men. With our set of demographic and socioeconomic indicators in the model, period trends appear much flatter for women and there is greater divergence between men and women across survey year.

After accounting for age, cohort trends are consistent with extant literature on pre-Depression cohorts (Idler, 1993); the predicted probability of fair/poor health initially increases across cohorts for women, peaking around the 1915/1920 cohorts before beginning a modest decline. For men, a similar peak is shown for the 1915/1920 cohorts, but after a modest decline for the cohorts thereafter, fair/poor self-rated health again increases to its highest points for the 1970 and 1975 cohorts. Similar to the age and period results, women have a higher predicted probability of fair/poor health relative to men across all cohorts. Accounting for demographic and socioeconomic covariates illustrates the importance of such factors in driving cohort trends in fair/poor health as the probabilities for men and women become strikingly lower across all cohorts and similar to one another through the 1930 cohort. Cohort trends by gender diverge after 1930, with cohorts of women showing a modest decline in fair/poor self-rated health and cohorts of men showing a steeper decline. For the overlapping cohorts in the respective studies, our results are consistent with the general upward cohort trend observed by Yang and Lee (2009) with different data and methods.

[FIGURES 1-3 ABOUT HERE]

#### Racial Disparities in Self-Rated Health

Figures 4, 5, and 6 show the differences in predicted probabilities of reporting poor/fair health between White and Black men and women. We refer to these differences in probability estimates as graphs of the Black/White disparity. Figure 4 illustrates curvilinear age disparities that are greater at all ages for women; these disparities remain, but decline less precipitously at older ages after accounting for socio-demographic and socioeconomic factors. For example, the racial disparity in poor/fair self-rated health for women more than doubles from age 30 to 60, but the curvilinear pattern suggests that the disparities for women in their 40s are rather similar for women in their 70s. Figure 5 illustrates racial disparities by period. Although disparities are larger for women than men, the inequity in self-rated health follows a similar, overall flat pattern across periods for both sexes.

[FIGURES 4-6 ABOUT HERE]

Finally, figure 6 illustrates disparities by cohort. Turning first to the disparities in which we do not account for possible explanatory covariates, our results show that the disparity is higher for women than men for most of the cohorts, increasing to its largest points from the 1925 through the 1945 cohort. Before including covariates, the disparity declines across cohorts beginning after the earliest surviving cohorts (e.g. 1895-1910). After including covariates, the differences between male and female patterns narrows for the earliest cohorts but is otherwise similar. Foreshadowing our decomposition

results, the gaps become substantially more modest after accounting for social, demographic and economic differences between Blacks and Whites. The disparities are substantially reduced for cohorts born before 1945, similar through the 1960 cohort, and slightly inflated thereafter. The sharp decline in the racial disparity for women, and more modest decline for men coincide with cohorts that entered the labor market after the civil and women's rights movements.

To summarize, after accounting for demographic and social differences, the cohort racial disparities in SRH for women increased slightly for those born in the beginning through the mid- 20<sup>th</sup> century and declines after the 1945 cohort have brought disparities to their lowest levels have only recently returned the disparity to those seen among early 20<sup>th</sup> century cohorts. On the other hand, the racial disparities for men, despite a relatively flat pattern across earliest available 20<sup>th</sup> century cohorts, show uniform declines from the 1915 cohort, which bodes well for future health and mortality disparities. Our covariate adjusted models suggest that at least part of these dramatic disparities observed over successive cohorts born in the first half of the 20<sup>th</sup> century can be accounted for by differences in social and economic characteristics. However, the question remains as to which characteristics are the most important for particular cohorts; we utilize decomposition methods to uncover these contributions.

#### Explaining Racial Cohort Disparities in Health

Using decomposition techniques, we examine how the various factors included in our models contribute to explaining the unadjusted racial health gap in successive birth cohorts (dashed lines in Figure 6), with a particular focus on the part of the gap due to differences in composition. The importance of composition relative to the "effects" (a component which also includes the influence of unmeasured covariates) varies across cohorts and by gender. Compositional differences explain approximately seventy to eighty percent of the total gap for men fairly consistently across cohorts. In contrast, compositional differences explain much less of the cohort-specific gaps for women. For most cohorts, compositional differences account for forty to fifty percent of the total female gap in fair/poor health; however, more of the gap is explained by composition for the oldest and youngest cohorts. This suggests that unmeasured factors (e.g. residential segregation or risky health behaviors) and weak returns/discrimination are particularly important in understanding inequality for the female cohorts for which the disparity is greatest. The overall lower explanatory power of compositional differences for women suggests that lower returns to various factors (e.g. education or marriage) may be more important in understanding the racial gaps of women than men, and that additional, unmeasured, factors may be important in understanding female disparities in health.

Figures 7 and 8 concern the part of the gap due to composition and illustrate what proportion is due to each of the factors considered: education, employment, income, marriage, geographic region and other factors. In standardizing the total compositional contributions across cohorts it is possible to see how compositional differences, such as the lower average



educational attainment of Blacks, have changed across cohorts in terms of relative importance for understanding the racial disparity gap. For men (Figure 7), differences between Blacks and Whites in education levels contribute consistently to explaining compositional differences in fair/poor health. Mean education differences are likewise important for women (Figure 8), contributing approximately fifty percent for cohorts entering the labor market around WWII. Regional distributions, particularly racial differences in Southern residence, decline in importance across cohorts for both men and women.

[FIGURES 7-8 ABOUT HERE]

For men, differences in income decline in importance, whereas differences in employment, driven mostly by differences in those not in the labor force, are increasingly important in understanding more recent cohort disparities. This is consistent with dramatic increases in joblessness among young Black men beginning in the 1970s related to trends of increasing incarceration and stricter child support enforcement (Holzer, Offner, and Sorenson, 2005; Small and Newman, 2001). For women, mean differences in income consistently contribute to the gap for women across the range of cohorts. Also in contrast to men, differences in employment initially favor Black women for cohorts before 1950. For example, for the 1920 cohort, the gap would actually be larger if Black women were distributed in the labor market in a similar way to White women. However, consistent with other research finding a racial crossover in labor force participation (Browne, 1997), this appears to change in more recent cohorts of women, such that employment differences now explain a small portion of the fair/poor gap. Finally, for both men and women, racial differences in marriage patterns contribute to the fair/poor gap in health, starting, most noticeably, with the 1925 cohort. For men, differences in marriage remain an extremely small factor in understanding health disparities whereas these differences are much more important in understanding gaps in health for more recent cohorts of women. These patterns are consistent with changes in marriage over time, including more rapid increases in age at first marriage for Blacks relative to Whites, greater increases in being never-married for Blacks (and particularly for women) and higher rates of remarriage for Whites (Bennett et al. 1989).

[FIGURES 9-10 ABOUT HERE]

Finally, we introduced cohort characteristics to our CCREM models to understand how disparities would change after accounting for relative cohort size, health and economic conditions at birth. Looking first at how the cohort characteristics impact the disparities for men, Figure 9 indicates that each of the characteristics we introduce reduce the disparity. The measure of relative cohort size and the infant mortality rate show similar reductions in the disparity with the most dramatic reductions seen for cohorts prior to 1930. The pattern for economic conditions at birth results in similar reductions with two important exceptions. The disparities are reduced for cohorts born around 1935, suggesting that part of their elevated probability is a function of the extremely negative economic conditions at birth that may have impacted blacks

more so than whites. Likewise, the post-WW II boom served to reduce the disparity below what it would have been otherwise as once we control for these conditions the disparity is elevated for these cohorts. Our final results will also detail more information on each of these cohort estimates, including information on the reduction of cohort variation with the introduction of each measure. Although percent Southern born shows similar reductions in the disparities to the other characteristics mentioned after the 1930 cohort, but results in the largest declines in the disparity of all of the characteristics considered for the earlier cohorts. The percent of the cohort born to large families results in modest disparity reductions up until the 1950 cohorts when it results in a dramatic decline in disparities; this coincides historically with an increase in the percent with large families among blacks and continued decline among whites (See Appendix Chart 1). Including the percent of the group's cohort born on the farm dramatically reduces the disparity through the 1960 cohort, commiserate with the higher, but converging, percentages of blacks relative to whites born on a farm across cohorts. The characteristics also result in reductions in cohort disparities for women. We find that, similar to men, relative cohort size and the infant mortality rate result in similar reductions; these reductions are smaller than those for economic conditions at birth, southern birth, economic conditions at birth and farm born. We also see the same dramatic decline in the self-rated health disparity with the introduction of family size for the post-1950 cohorts.

## **Discussion**

Period, as defined by survey year, tells only one part of the temporal story of racial health disparities. Specifically, measuring survey year only confounds the story of both aging and cohort changes in disparities over time. In order to fully understand temporal change and the factors that contribute to these changes, researchers must begin to incorporate the full demographic toolkit of age, period, and cohort into their studies of temporal population health change (e.g., Yang 2008). Each of these temporal components has not only a unique theoretical meaning, but has highly divergent implications for studies of population health in general and studies of health disparities in particular.

Using a unique data set of repeated cross-sections of NHIS data, normalized over survey years by the IHIS project at the University of Minnesota, we were able to simultaneously explore the temporal patterns in self-rated health in addition to exploring the temporal patterns in SRH disparity by further decomposing the composition of cohorts to determine the individual-level factors that contributed to the observed dramatic changes in disparities over time.

Our results for aging exhibit the well-known pattern of declining health with increasing age but also show a curvilinear racial disparities pattern that is inconsistent with a weathering process (e.g., Geronimus et al. 2006; Walsemann et al. 2008). Second, our results for period indicate both broad downward trends in the probability of reporting fair/poor SRH over time as well as a negligible overall racial disparity and downward period disparity trend for Black males relative to White males; at

the same time, we found a more dramatic decline in period-based racial disparities for women. Third, our results indicate an uneven decrease in the probability of reporting fair/poor health across successive birth cohorts, but very different cohort disparity trends by gender. For men, a once increasing cohort-based racial disparity, flattened out for most of the cohorts born in the mid-1900's, before declining for more recent cohorts. Regardless, this interesting result may be noteworthy as time passes and the results of health ratings play themselves out in terms of actual morbidity and mortality risks. On the other hand, women experienced a long and significant trend of increasing disparities that has only recently found its way back to pre-1910 cohort levels.

Finally, our decomposition results also demonstrate some very interesting trends in terms of what factors are contributing to cohort disparities as well as which factors are gaining or losing in importance over time for health disparities. While the usual suspects of socio-economic factors (education, income, employment) continue to demonstrate their relevance for health disparities research, factors such as residence in the South begin to decline in importance in more recent birth cohorts for both men and women. Of particular and emerging importance however, marital status becomes a particularly salient contributor to observed health disparities among women in cohorts from 1925 onwards while employment status emerges in the 1910 cohort for men and becomes a major contributor to observed health disparities from 1950 onward. Although deleterious trends in marriage and employment may re-exacerbate inequalities as they continue to play out over the life course for younger cohorts, our extant results are heartening for the prospect of future cohort inequities. For both men and women, not only are recent disparities declining, but these disparities are disproportionately explained by differences in average socio-demographic characteristics (modifiable factors such as educational attainment or employment) rather than discrimination or returns to these endowments.

As we noted above, in order to break the linear dependence of age, period, and cohort, we utilized repeated cross-sections of survey data and grouped individuals into five-year cohort bands. Thus, we made the assumption of invariant year-to-year cohort effects within a given cohort. However, given the small changes observed in both age and period estimates, we don't feel that this aggregation introduces any more bias than other standard approaches. The major source of bias would be considering cohort effects without simultaneously considering period and age effects. In short, although we make assumptions of within-cohort invariance, this is necessary to identify our models and is no more hazardous of an assumption than other conventional approaches.

Our results must also be interpreted with caution due to other selection effects that occur at various levels that would exclude the most frail or socio-economically disadvantaged members of the U.S. population. Although our data are nationally representative, selective mortality excludes the least healthy from our estimates of racial health disparities. We have no way of knowing what these disparities would have looked like if mortality had not previously occurred to create a

more select sub-sample (Hayward et al. 2000). Clearly, with increasing age, mortality selection plays a greater role as well as when we examine birth cohorts from the early 20<sup>th</sup> century. We know, based on differential mortality rates, that Black and White adults do not die at the same rates and as such, our disparity estimates are biased by this non-random mortality. Further, the NHIS surveys the non-institutionalized population so selective migration into nursing homes and long-term care facilities further increases the potential for bias. If institutionalization of the frail and elderly occur at differential prevalence, then our estimates of disparity are potentially further biased. Again, these biases will be magnified by age and within the older birth cohorts. Finally, incarceration, which has known wide rates of racial divergence, may further contribute to bias in the estimates of racial health disparities. We do not introduce these potential sources of bias as unique or epidemic to our chosen APC approach—rather, they are endemic to all studies of population health that measure non-institutionalized populations with selective mortality. However, they are further magnified in our study by looking at especially old cohorts, and as such, patterns in the left- and right- tails of our distributions should be interpreted very cautiously.

In summary, it is clear that the concept of the cohort has important theoretical implications for population health research and has particular import for the study of health disparities. While aging and period have retained seemingly exclusive rights over population health approaches, the time for the theoretical and analytical cohort approach has arrived. In order to thoroughly and accurately study temporal changes in population health and health disparities, we need to carefully begin to incorporate the concept of the cohort into our studies. Future studies should pay careful attention to the potential biases of excluding any of these temporal factors and should move beyond the descriptive nature of this study. We briefly explored how changing cohort composition can lead to not only differential health disparities over time, but also can have differential explanatory power over time. However, this focus on cohort composition can and should be extended to larger macro-characteristics of cohorts in order to explain how temporal characteristics and social conditions and change can affect the health of the population as well as population health disparities. To summarize, future studies should explore cohort and period characteristics that may be responsible for observed temporal health disparities. In this way, the cohort approach to population health can begin to truly transform itself into a social study of the conditions leading to differential mortality by race and socio-economic status.

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Table 1. Descriptive Statistics of Self-Rated Health and Covariates, by Gender and Race

			White		Black	
	Men	Women	Men	Women	Men	Women
<b>Outcome</b>						
Fair/Poor Health (%)	15.5	16.2	14.6	14.5	22.3	26.5
<b>Covariates</b>						
Age	49.9	49.9	50.1	50.2	48.0	47.7
Period	1992.1	1989.6	1991.8	1989.2	1994.2	1992.2
Cohort	1942.2	1939.7	1941.7	1938.9	1946.2	1944.5
<i>Region of Residence</i>						
Northeast (%)	21.2	21.7	21.7	22.3	17.6	18.6
North Central/Mid-west (%)	26.3	26.8	27.3	27.9	19.4	19.7
South (%)	33.9	34.4	31.2	31.3	52.6	52.9
West (%)	18.6	17.1	19.8	18.5	10.4	8.8
<i>Marital Status</i>						
Married (%)	69.6	63.1	72.0	67.0	54.5	39.9
Nevermarried (%)	13.5	8.9	12.4	7.0	20.3	20.2
Divorced/Widowed/Separated (%)	16.9	28.0	15.6	26.0	25.2	39.9
Missing Marital Status (%)	0.4	0.4	0.4	0.3	0.9	1.1
Family Size	2.5	2.7	2.5	2.7	2.6	2.9
<i>Education Level</i>						
High School Degree or Less (%)	57.5	62.9	53.0	61.2	65.9	67.9
Some College (%)	20.9	20.2	20.9	20.2	21.0	20.3
College Degree or More (%)	24.5	17.6	26.1	18.6	13.1	11.8
<i>Employment Status</i>						
Employed (%)	68.4	49.9	68.9	49.1	64.6	54.2
Unemployed, Looking (%)	3.2	2.6	2.9	2.3	5.1	4.5
Unemployed, Not Looking (%)	10.8	27.6	10.0	27.9	16.2	25.9
Retired (%)	17.6	19.9	18.2	20.7	14.1	15.4
Missing Employment Status (%)	0.4	0.3	0.4	0.3	0.7	0.6
Family Income	15634.0	14045.1	16055.0	14636.8	12782.0	10531.7
Missing Family Income	9.8	11.5	9.4	11.1	12.5	14.0
<i>Cohort Characteristics</i>						
Relative Cohort Size at Labor Market Entry	13.2	13.2	13.2	13.2	13.2	13.2
Segregation at Birth	0.72	0.71	0.71	0.70	0.73	0.73
Economic Conditions at Birth	-0.03	-0.04	-0.03	-0.04	-0.03	-0.03
N	592141	920956	515975	788226	76166	155714

Fig. 1 Predicted Probability of Fair/Poor Health: Age Trends by Gender: Adjusted for Age, and Age and Covariates (Xi)

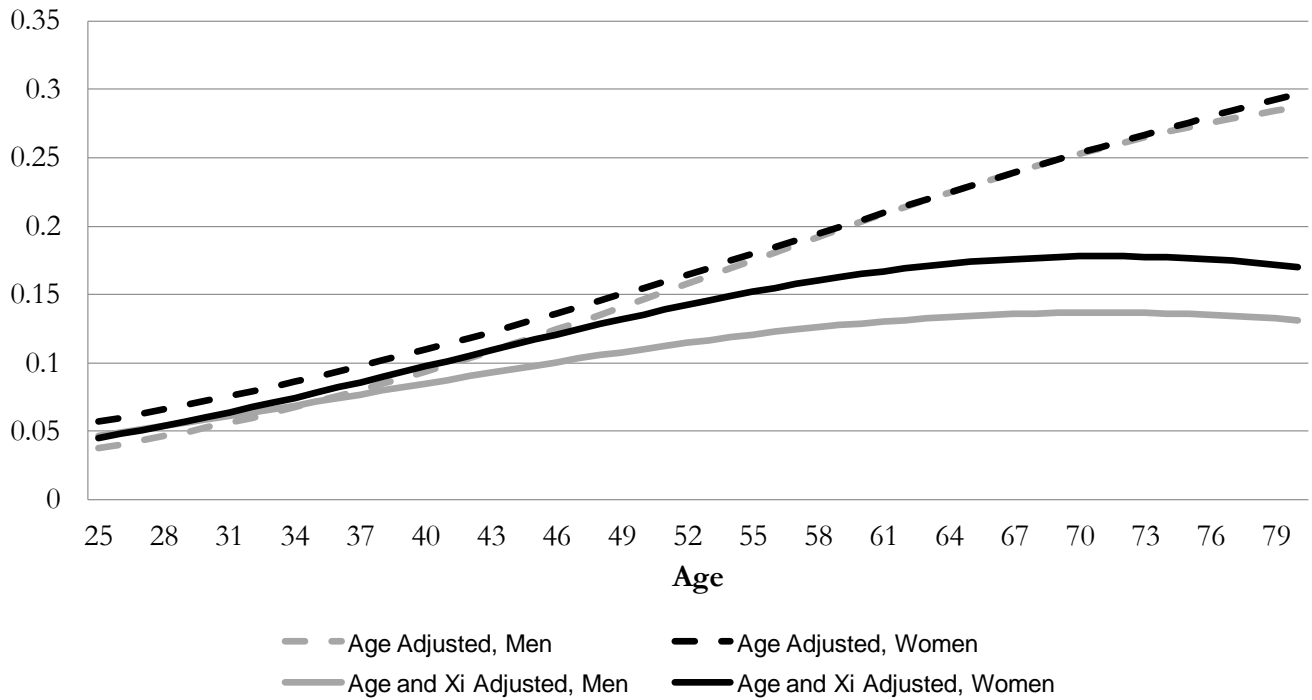


Fig. 2 Predicted Probability of Fair/Poor Health: Period Trends by Gender: Adjusted for Age, and Age and Covariates (Xi)

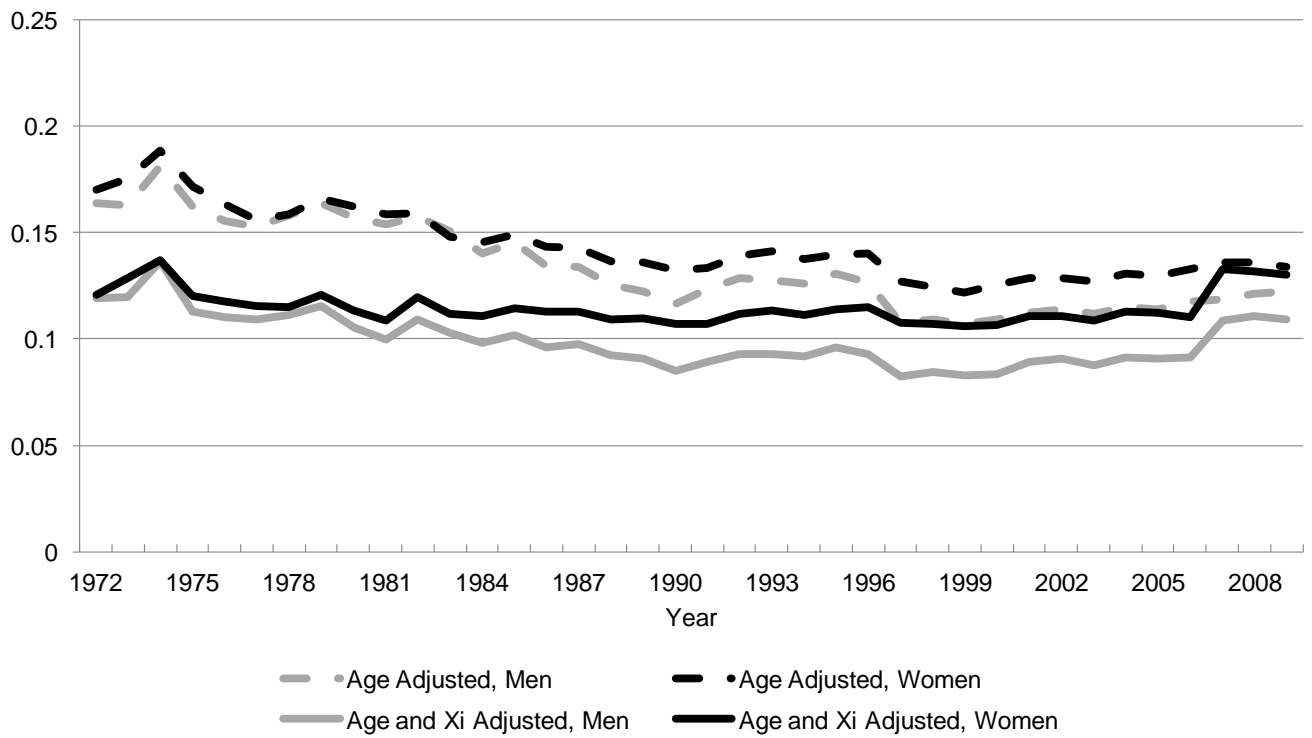


Fig. 3 Predicted Probability of Fair/Poor Health: Cohort Trends by Gender: Adjusted for Age, and Age and Covariates (Xi)

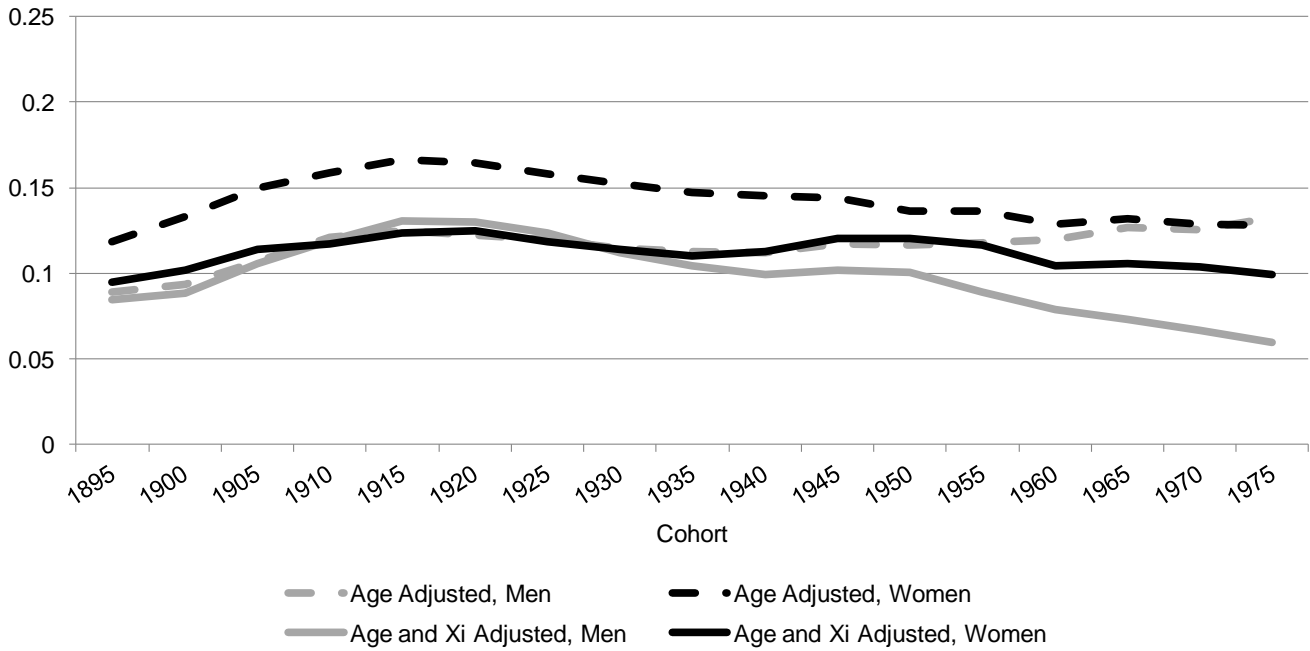


Fig. 4 Predicted Probability of Fair/Poor Health: Age Disparities by Gender: Adjusted for Age, and Age and Covariates (Xi)

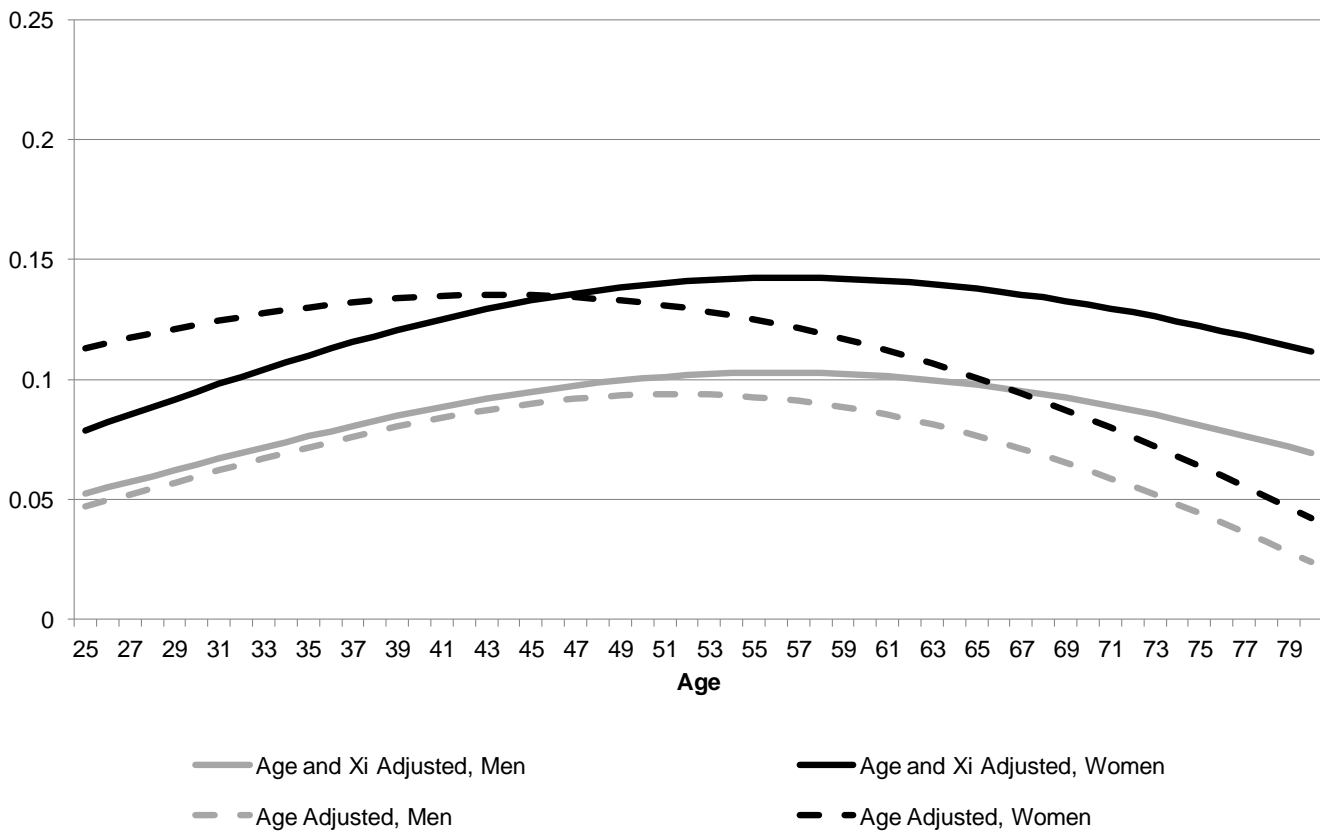


Fig. 5 Predicted Probability of Fair/Poor Health: Period Disparities by Gender: Adjusted for Age, and Age and Covariates (Xi)

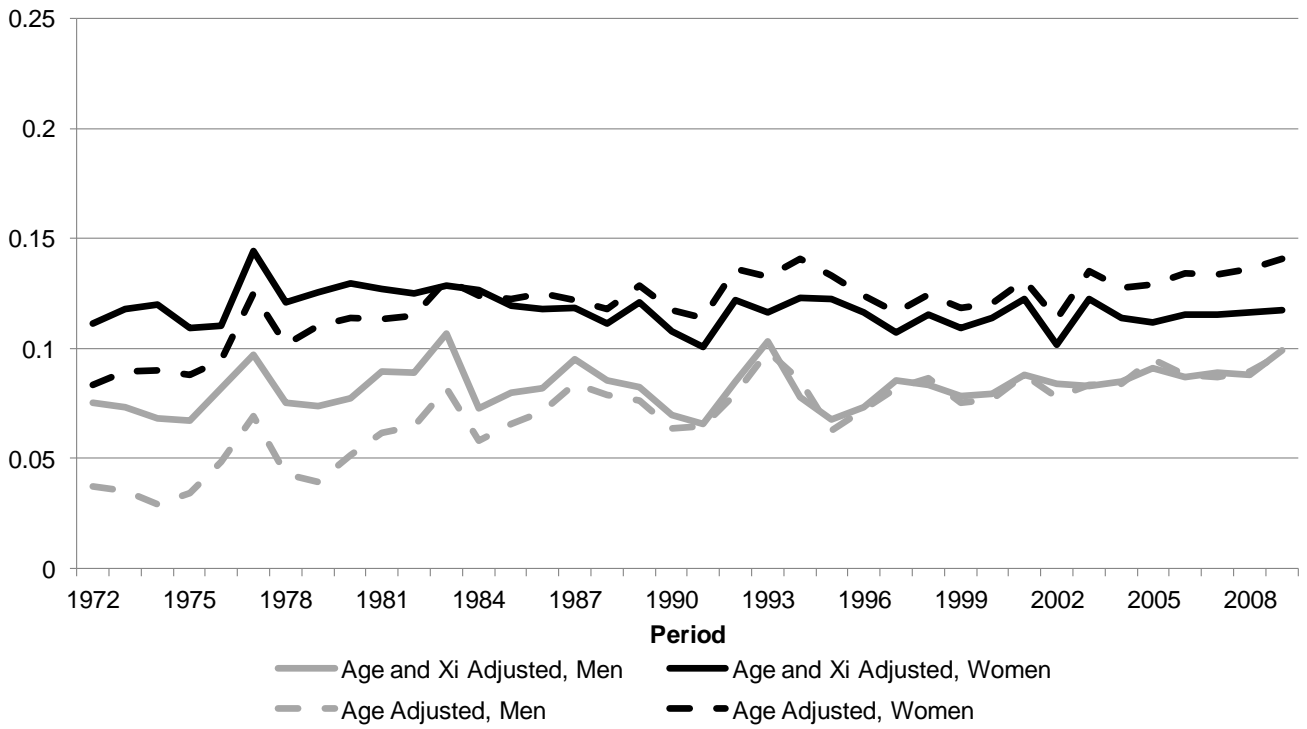


Fig. 6 Predicted Probability of Fair/Poor Health: Cohort Disparities by Gender: Adjusted for Age, and Age and Covariates (Xi)

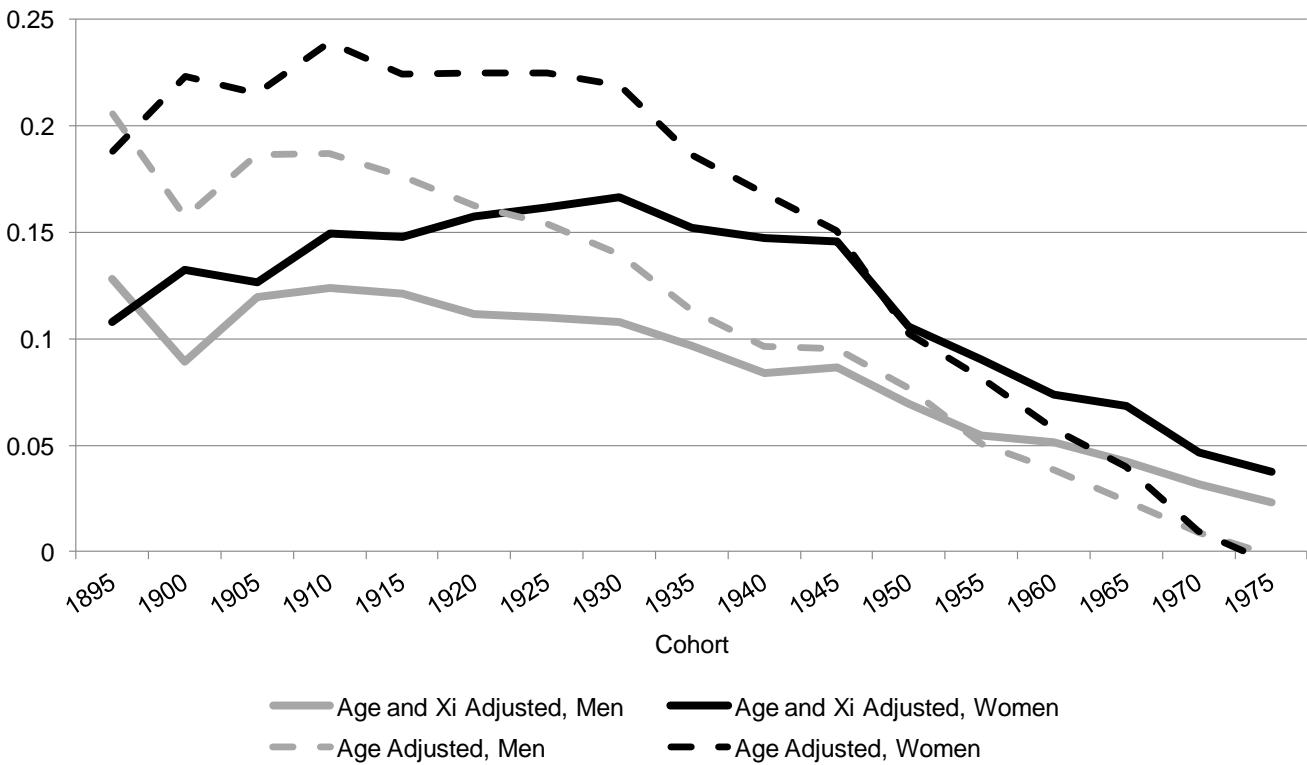


Fig 7. Contributions of Covariates (%) to Disparity Explained by Endowments, Men

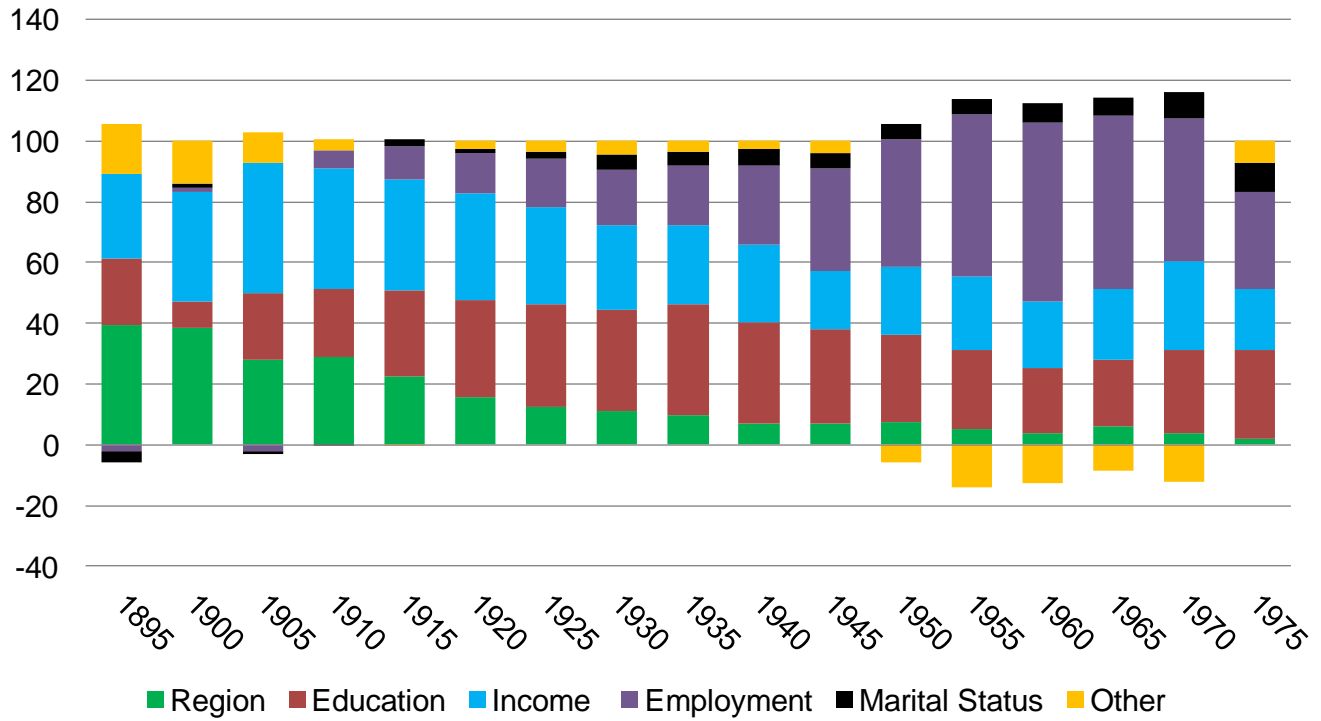


Fig 8. Contributions of Covariates (%) to Disparity Explained by Endowments, Women

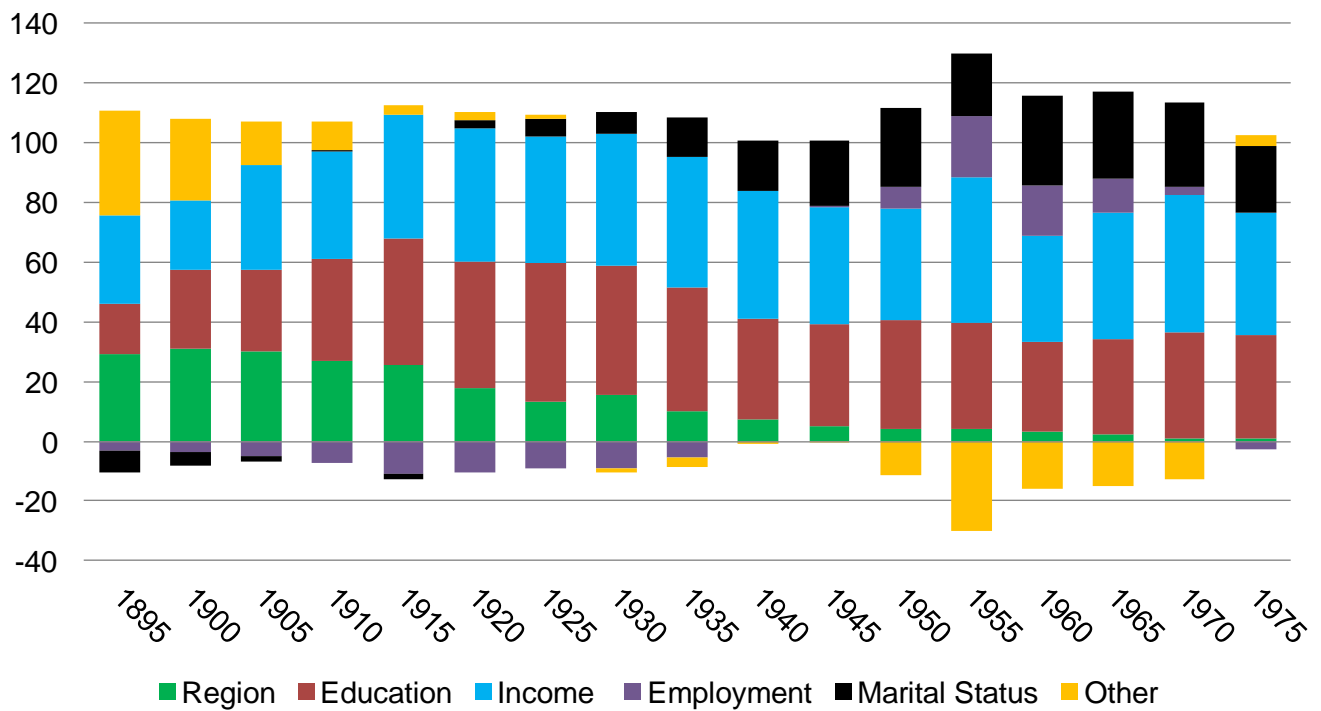
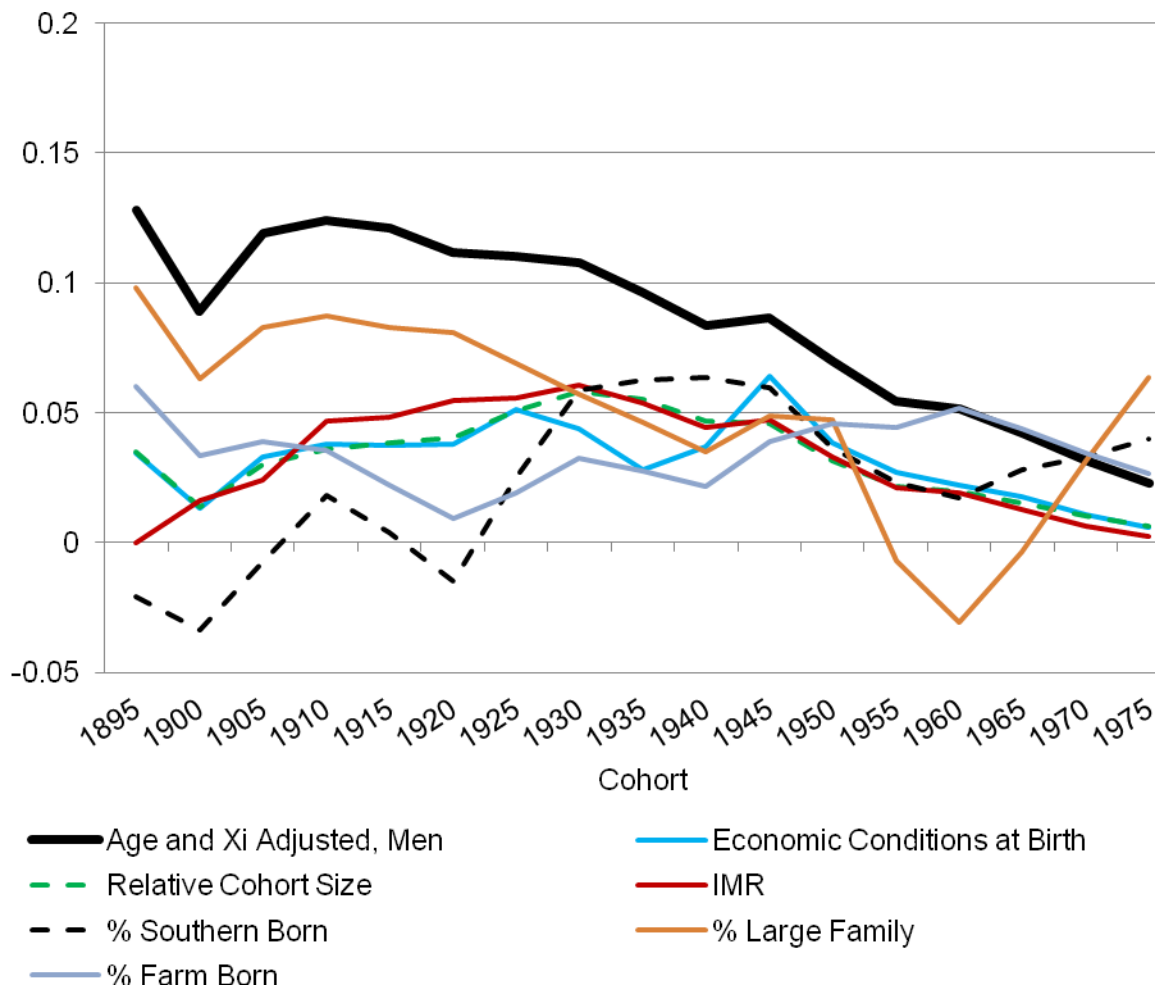


Fig 9. Predicted Probability of Fair/Poor Health: Cohort Disparities with Explanatory Cohort Characteristics Included, Men



Appendix Chart 1: Descriptive Trends in Cohort Characteristics

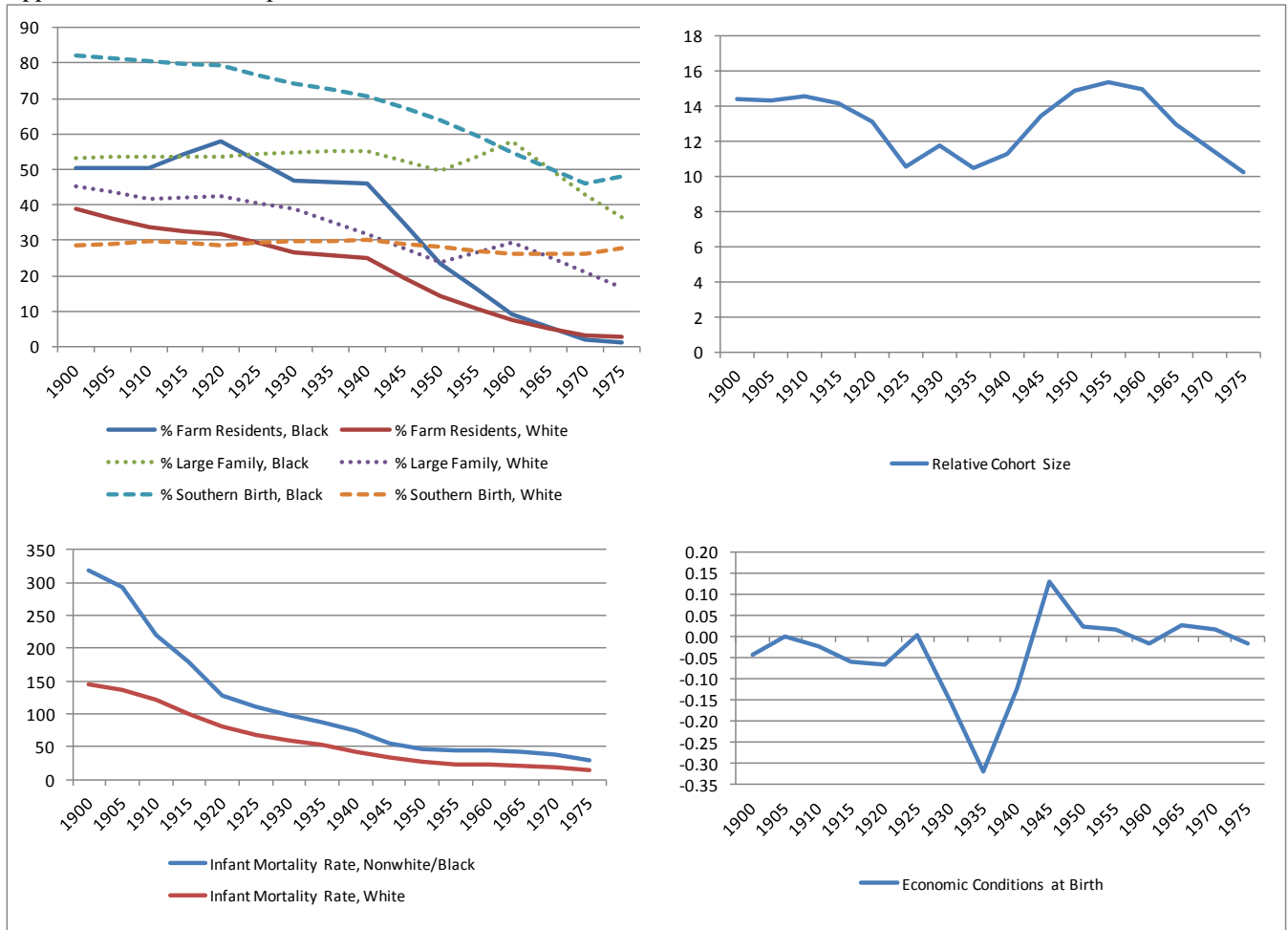
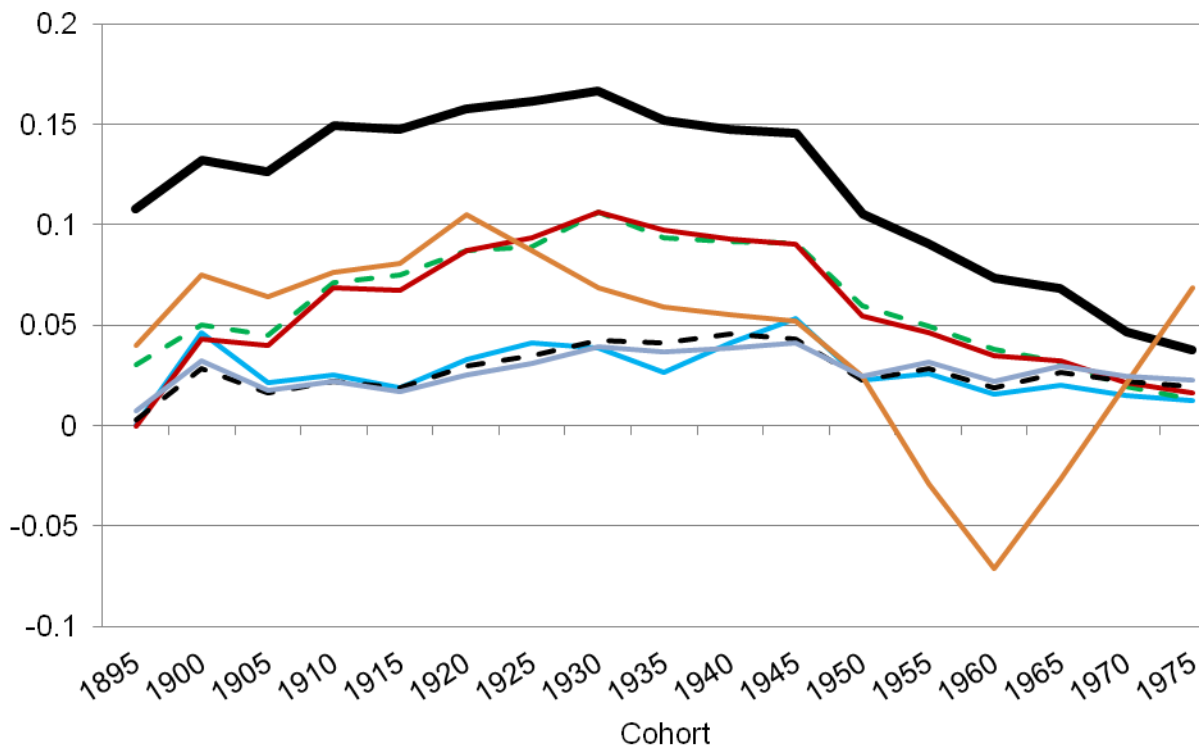


Fig 10. Predicted Probability of Fair/Poor Health: Cohort Disparities with Explanatory Cohort Characteristics Included, Women



- Age and Xi Adjusted, Women
- Relative Cohort Size
- % Southern Born
- % Farm Born
- Economic Conditions at Birth
- IMR
- % Large Family