Preliminary Manuscript:

Understanding the social stratification and diffusion of health-related innovations using the 20th century pattern of converging, diverging and stable racial disparities in mortality in the U.S.

The changes in the expectation and variability of survival between the nineteenth and twentieth centuries describe a process of epidemiological transition from infectious to chronic disease that has been associated with improvements in nutrition, public health, social and economic development, and medical innovations (Omran 1971; McKeown 1976; Szreter 2004). A wealth of literature on social inequalities in health describes the differences mortality between population groups defined by race (for example see Kitagawa and Hauser 1973; Keith and Smith 1988; Preston et al 2003; and for review see Williams and Collins 1995). These studies describe how a range of factors --social, economic, cultural, geographical and political— demarcate groups of people who have different life opportunities, political rights, and different exposure to social norms and environmental hazards all of which are important for determining health and longevity.

As I will describe in greater detail below, this study focuses on the types of cleavages and respective access to resources that are indicated by racial differences in mortality. I consider differences in mortality between age-groups in which specific causes of mortality predominant and which can be used to reflect epidemiological transitions in the principal cause of mortality. This is not the first study to relate racial differences in social conditions to racial differences in epidemiological transition (Potter 1980). However, it is the first study to formally develop and

test hypotheses about the pattern of historical change in social disparities in survival in a way that can provide new insights about the social stratification and diffusion of innovations and resources through the process of epidemiological transition.

BACKGROUND

There are for potential patterns of between-group differences in population outcomes that may be reflected in the racial differences in mortality observed historically: mortality may be converging, diverging, stable or dynamically converging and then diverging. These trends can be related to theoretical literature substantiating different mechanisms through which health and survival becomes socially stratified. I describe each in turn.

Narrowing or converging inequalities over time: In the early twentieth century, economic development and the associated improvements in public infrastructure –particularly those important for the transmission of infectious disease, such as water and sanitation— as well as public education systems and the development of welfare state programs (such as social security) have offered the possibility of diminishing the inequalities in environmental exposures and resources that influence health and survival. The relevance of economic development to diminishing inequality in survival has been studied with respect to inequalities between countries (Preston 1976; Wilson 2001). At a within-country level, this process would lead to a hypothesis of *converging* racial inequalities in health over the last century.

Stable inequalities over time: The theory of social conditions as a 'fundamental cause' of disparities in health describes how new innovations and resources that are relevant to health are

consistently distributed through the population in a way that makes those at the 'top' of the social hierarchy advantaged with respect to health and longevity and those at the bottom disadvantaged (Link et al. 1998; Link and Phelan 2000). This approach derives from a larger body of research in which a stratified distribution of 'life chances', exposures, and 'life events' are linked with inequalities in health and survival observed in different historical period and contexts (Villerme 1840; Engels 1844[1987]; Virchow 1848; Antonovsky 1967). This is hypothesized to produce a *stable* inequality in the health and longevity between social groups.

Widening or diverging inequalities over time: Widening differences in health and survival between social groups, as well as nations, have been highlighted by researchers concerned about the trends in economic inequality over the later half of the twentieth century (Duleep 1989; Pappas 1993; Schalick et al 2000; McMichael et al 2004). These studies link divergence in health to theory about the role of deindustrialization, globalization and other aspects of technological and market change that they argue have disproportionately benefited individuals, groups and nations at the top of the social hierarchy. This is not the only theoretical arguments that supports widening disparities, though. A hypothesis of widening inequalities over time, also emerges from employing existing life course theory to populations—i.e. recognizing that mechanisms leading to widening of social inequalities over age (Ross and Wu 1996) can be applied to populations to hypothesize widening of social inequalities over historical time.

The health and aging of cohorts reflect the accumulated resources of individuals comprising the cohorts, as well as the resources (both biological and social) that they have inherited from previous cohorts—i.e. the cohort members' parents and grandparents (Carey and Judge 2001).

Just like how theory about accumulated advantages and disadvantages can be used to explain why there are widening disparities in health outcomes between social groups as the individuals comprising them age, it can also be applied to explain widening disparities in health observed between social groups over historical time. Like with the aging of individuals, the historical trends of subpopulations (comprising social groups) may entail 'positive feed back-loops' of disadvantages as new cohorts with (or without) inherited social and health resources enter and refresh the populations over historical time.

Dynamic progression of inequalities over time. I hypothesize that a combination of the above trends is likely to be observed as inequalities in populations are tracked over historical time. This entails a synthesis of the theoretical models described above, and a refinement of the "fundamental cause" theory. In specific, I point out that in order for there to be 'stable inequalities', as hypothesized by Link and colleagues and other researchers applying this model (Link et al. 1998; Link and Phelan 2000; Warren and Hernandez 2007), there would need to be a continuous balance between the diffusion of past innovations to the 'bottom' of the social hierarchy and the development of new innovations and adoption by the 'top' of the social hierarchy. In other words, if differences in survival are hypothesized to be constant, there should not be an initial period of widening differences as the innovation diffuses through the 'advantaged' group(s) and then converging of differences as the innovation subsequently diffuses through the 'disadvantaged' group(s).

I test which of these four possible patterns is most consistent with the patterns of mortality in the U.S. over the past 100 years.

DATA AND METHODS

The data come from the National Vital Statistics System for the US. Age-specific mortality rates from each year were accessed in tabular form from the National Center for Health Statistics in the U.S. Centers for Disease Control (CDC/NCHS 1960; CDC/NCHS 1970; CDC/NCHS 1980; CDC/NCHS 2000; United States Department of Health and Human Services (US DHHS) et al. 2004). The U.S. has consistently reported mortality rates from 1900-2002 by a white and 'non-white' racial classification for men and women in the following age groups: 0 years, 1-4 years, 5-14 years, 15-24 years, 25-34 years, 35-44 years, 45-54 years, 55-64 years, 65-74 years, 85+ years. These data are presented by cause-relevant age groups as described below.

Categorization of race. In these preliminary findings, I demarcate race by 'white' and 'non-white' classification. This is to reduce the complexity of consistently coding the changing racial categories over the last century. I recognize that the category 'non-white' has become increasingly heterogeneous through this period. Although, during the first half of the century this subpopulation was primarily composed of persons currently categorized as 'non-Hispanic black' by CDC, NCHS and Census, during the later half of the century the group included an increasing proportion of other racial and ethnic minorities (Rumbaut 1994). In my final analyses, I will conduct sensitivity analyses to consider the influence of these changes in the composition of the 'non-white' subpopulation.

Identification of cause-relevant age-groups. I present trends in racial disparities in mortality for age groups that are divided by the influence of cause-specific mortality in that specific age

group. The trends in mortality in specific age-groups, thus provide a marker of the relative differences of causes of mortality that have been associated with changes in life expectancy over the twentieth century. These include changes in the prevalence of infectious disease, accidental and violent mortality, chronic disease mortality, and mortality associated with senescence (Gage 1994; Bongaarts 2005; Cutler 2005). The use of changes in age-specific mortality (and the related changes in the age-distribution of mortality) have been applied by previous researchers to indicate the changes in the principal causes of mortality that reflect epidemiological transitions (Wilmoth and Horiuchi 1999; Salomon and Murray 2002; Robine 2001; Weden and Brown 2008).

Measurement of racial disparities in survival. In order to be transparent about the way in which mortality disparities are changing over time, I have selected to use the patterns of age-specific mortality that are divided by cause-relevant age-groups, rather than other indicators of population health (like life expectancy from age zero, life expectancy from age 15, or a measure of inequality in the age-distribution of survival). For example, trends in composite indicators, like life expectancy at age zero will be heavily influenced by the trends in mortality at age 0 due to the large skew in the distribution of mortality produced by mortality in early life. For each age group, I calculate the racial disparity in the age-specific mortality rate ratio (RR): (mortality age x| non-white)/ (mortality age-group x | white). These trends are presented for early life (age 0, age 1, and age 5-14); adolescence and young adulthood (age 15-24); midlife (age 25-34, age 35-44, age 45-54), and late adulthood (age 55-64, age 65-74, age 75-84, and age 85+).

PRELIMINARY FINDINGS AND DISCUSSION

In these preliminary analyses, I present findings for age-specific mortality rate ratios (RR) for men. These findings provide initial insight into the trends in racial disparities that will be examined in the final manuscript. In the final analyses, I will present the findings for both men and women at all ages within the cause-relevant age-groups noted above. Although the preliminary findings presented here provide a strong case for dynamic patterns of racial disparities over time, my subsequent, the comparisons by sex will provide further insight into the social diffusion of longevity promoting innovations—as well as longevity-compromising exposures—that have been relevant to particular causes of disease.

Early Life: In Figure 1, I depict the ratio of mortality in early life (ages 0, 1-4, and 5-14), and I depict the differences in age-specific mortality rates for non-whites and whites that reflect the differences in the pace of declines in mortality (for a representative early-life age group: age 5-14). The pattern of racial disparities entails a *convergence in mortality* over time. From an initial disparity of about two times greater mortality among non-whites than whites (mortality rate ratio, RR is between 2 and 2.75 over 1900-1905), mortality differences diminish and then stabilize for children (RR between 1.25 to 2.0 over the period 1920-1999). These changes in mortality before age 10 have been associated in other literature with the second epidemiological transition from high infectious disease and infant mortality to dramatic declines in both (Arriaga 1984; Pollard 1988; Armelagos et al. 2005). The findings presented here demonstrate that these declines occurred first among whites and then non-whites in a manner that produced *convergence* to a *stable,* relatively low level of racial disparity[1].

[1] It is noteworthy that for infants, the racial disparities may actually demonstrate a pattern of convergence and then divergence. In age 0, the RR drops to about 1.5 in 1920 and then climbs to about 2.0 by 1999). In fact, recent trends in racial disparities for infant mortality show a widening of disparities from 1950 through the end of the century (Singh 1995). These trends may reflect the social diffusion of new medical technologies available in neonatal

Adolescence and Young Adulthood: Figure 2 also shows an overarching pattern of *convergence*. The mortality rate ratio is high and relatively constant (with a slight increase) for adolescents age 15-24 over the period 1900 through 1940 (RR= 2 in 1900 and 2.5 in 1940). The rate ratio then declines to a much lower (though fluctuating) level of racial disparity by the end of the century (RR=1.5 in 1960, 1.25 in 1980, and 1.30 in 2000). It is noteworthy that although racial inequalities are observed to have dropped during the later half of the twentieth century, there are period up-ticks in disparities during the 1970s and 1990s. These disjunctures in the pattern may be attributed to increases in homicide among black men and racial disparities in HIV/AIDS described in other research (i.e. see Elo and Drevenstedt 2004). I expect that analysis of trends in racial disparities for women may offer further insights into the changing diffusion of exposures and resources influencing mortality. For example, this may include the decline in maternal mortality at the turn of the century and its relationship, at least in part, to antibiotics and changes in medical practice (CDC 1999).

Midlife: Figure 3 shows a pattern of initially relatively low disparity among adults in midlife (RR is about 1.5 between 1900 through 1920) that increases (most dramatically among the youngest adults) to a level of 2 to 3 times greater mortality among non-whites than whites between 1920 and 1940. The diminishes to a rate ratio of 1.5 to 2.5 over the period 1940 to 1960. There is a second peak in mortality disparities over the next twenty years and then mortality disparities diminish to a level equal to that at the beginning of the century (RR=1.5).

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intensive care units or other social, behavioral or economic factors potentially related to widening differences in maternal health.

The pattern of *diverging and then converging* disparities during the first half of the century reflects a continued decline in mortality among white men that is not observed among non-white men until 1940. The rapid decline of mortality among non-whites observed in the following twenty years then produces the convergence in rate ratios between 1940 and 1960. This pattern of divergence and then convergence is consistent with delayed diffusion of longevity promoting resources among non-whites, such as the improvements in cardiac care or may even be related to reductions in early life exposures to infectious disease that have been related to later life chronic conditions (Fogel and Costa 1997). As observed here, these trends in a delayed reduction of mortality among non-whites would be expected in the twenty years following the public health advances in sanitation and food processing that historical analysis suggests also were delayed in researching nonwhites (Troesken 2002). In contrast, the peak at the end of the century for adults parallels that experienced by young adults, and similarly reflects similar increases in non-white mortality that are consistent with the rise in homicide and HIV/AIDS discussed earlier.

Late adulthood: Figure 4 shows a pattern of stable and low levels of racial disparity, with relative *advantages* to non-whites among the oldest members of each population (i.e. those age 75-84 and 85+). The overall pattern in late adulthood largely reflects a stable absence of inequality (among the oldest members) or stable, slight disadvantage among those who have recently entered late adulthood that has changed little over the last century and mortality rate ratios just below one for adults age 75-84, and age 85+) that is consistent with previous findings on the mortality crossover observed among the oldest old (for example see Lynch 2003)[2]. The trends in the mortality rate by race for adults age 65 that is also depicted in Figure 4 show that

[2] The timing of the crossover, the age where the crossover occurs, and whether it exists at all is an active area of research entailing considerations of data quality and methodology (Preston et al. 1996; Preston et al. 2003; Lynch 2003).

although their have been declines in mortality in late adulthood that increased in pace after 1970, these declines have been observed similarly among whites and nonwhites. The pattern of *stable disparities* observed among older adults is consistent with the absence of innovations in knowledge or technology that can be used to substantially reduce mortality. If we are indeed headed towards a new epidemiological transition in which cancer is reduced as a cause of disease and there is a slowing of senescence (Horiuchi 1997; Robine 2001), it is likely that these innovations designed to address mortality among the oldest old (for example innovations in behavior or technology that leads to reductions in cognitive decline and physical frailty) will also entail patterns of social stratification and diffusion that have been observed with respect previous epidemiological transitions.

In summary, I argue that the disparate trends for whites and non-whites reflect different patterns of changes in mortality that are consistent with the hypothesis of a *dynamic progression* in diffusion and social stratification of longevity-promoting resources. The timing and progression of these trends, as well as the presence and absence of racial disparities during different time periods are also consistent with the types of innovations in longevity-promoting knowledge, policies, technology, and social and economic developments that have been described with respect to the epidemiological transition from infectious to chronic disease. (Omran 1971; McKeown 1976; Szreter 2004). The final analyses to be developed for this study, will refine and further contextualize how the patterns of racial disparity in mortality over the 20th century elucidates overarching dynamics of social stratification observed in a population over time.

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Figure 1. Racial disparities in mortality among U.S. males in early life, 1900-2000

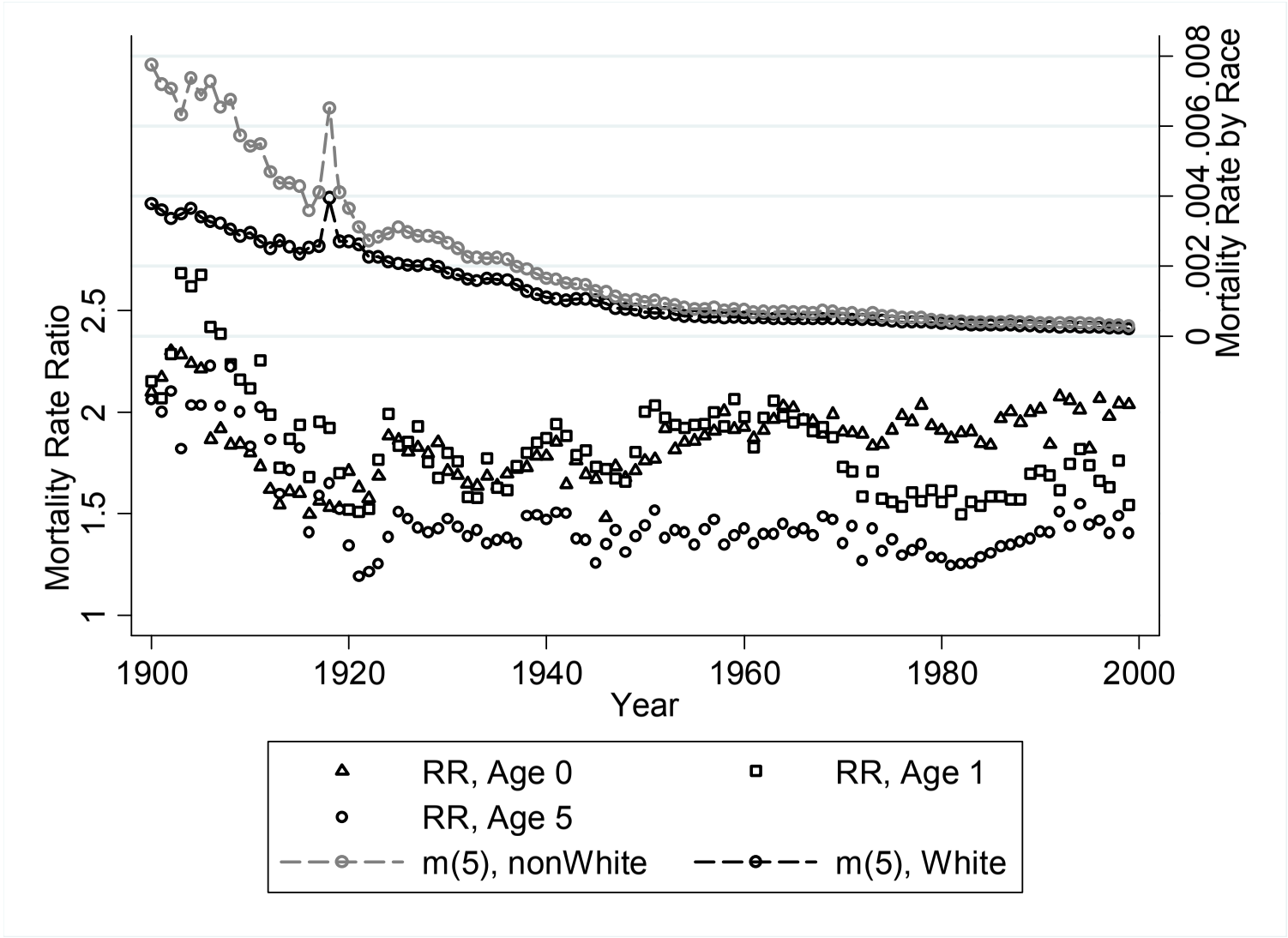

Figure 2. Racial disparities in mortality among U.S. adolescent and young adult males, 1900-2000

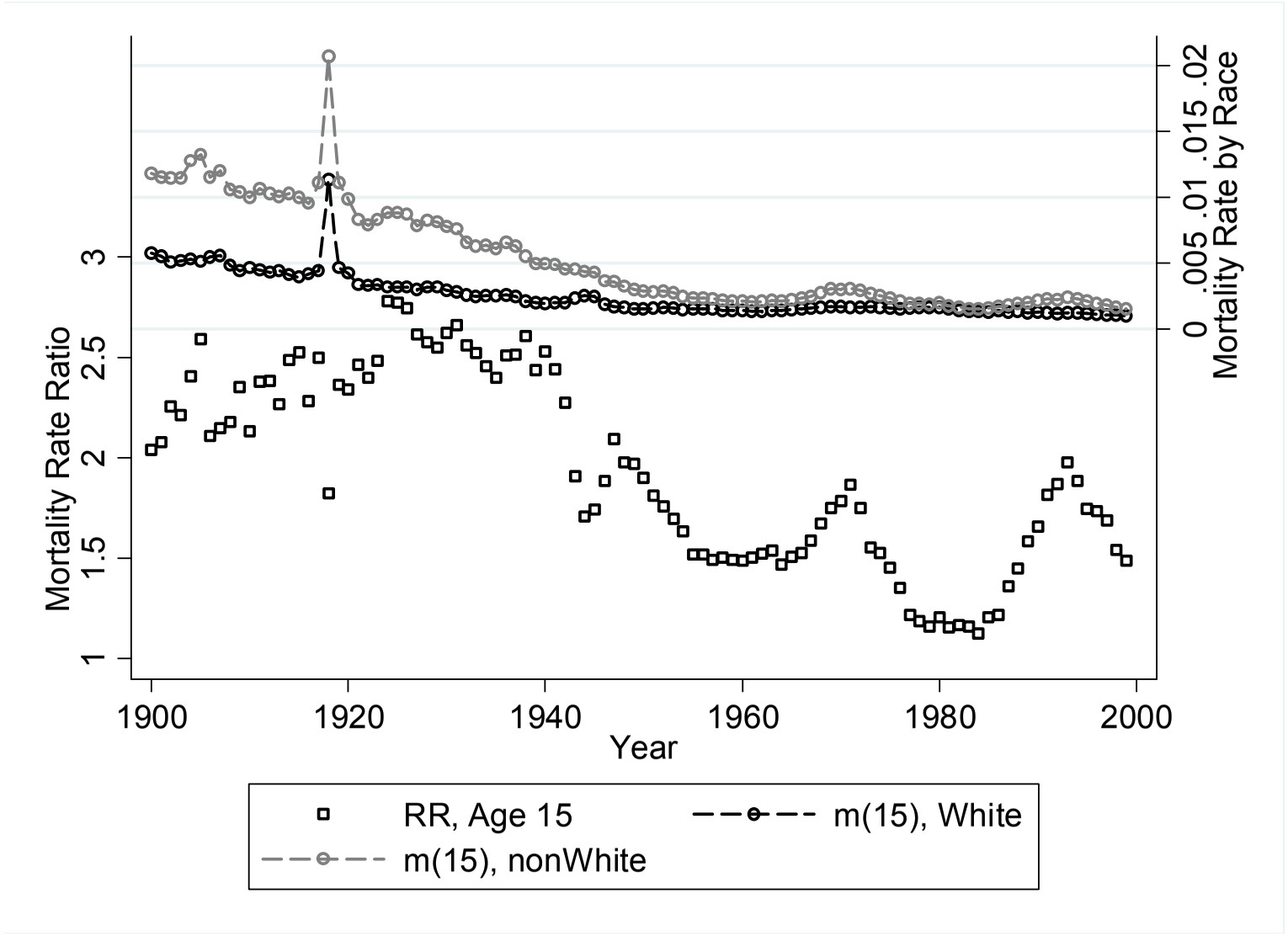

Figure 3. Racial disparities in mortality among U.S. adult males in midlife, 1900-2000

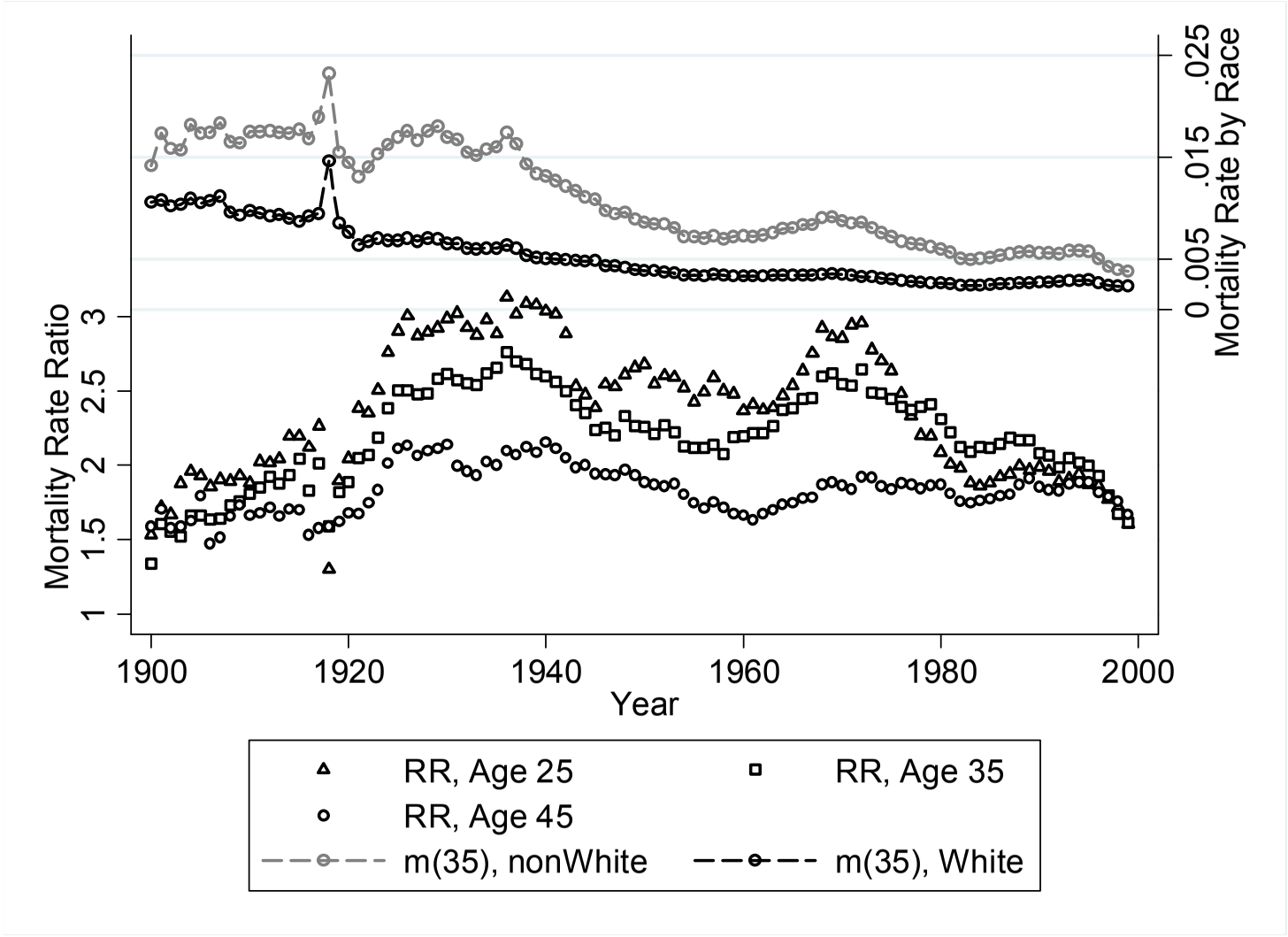

Figure 4. Racial disparities in mortality among U.S. males in late adulthood, 1900-2000