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Changes in the Average Age of
Death and the Variability in the
Age of Death for White and non-
White Men and Women, 1900-
2002

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LABOR AND POPULATION

Twentieth Century U.S. Racial Inequalities in Mortality:
Changes in the Average Age of Death and the Variability in the Age of Death
for White and non-White Men and Women, 1900-2002

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Abstract

This paper develops a new theoretical and empirical approach to studying historical trends in social inequalities in mortality. I describe how *within* group inequality in mortality (e.g. IQR_0) can be used to identify the timing and pace of epidemiological transitions, and I relate differences *between* population groups in IQR_0 to theory on social inequalities in mortality. Using data on mortality for U.S. white and non-white men and women over the period 1900-2002 and decomposing the patterns of change in IQR_0 , I find that mortality declines have been later, slower, and have involved more irregular age-composition among non-whites than whites. As a result, racial differences in IQR_0 have dynamically diverged and converged while there has been a century of convergence in e_0 . These findings show that IQR_0 provides information about mortality differences that are concealed by e_0 and they provide insight into the theoretical trends in mortality associated with economic development, diffusion of innovations, institutional changes, and social stratification.

INTRODUCTION

At the turn of the century, after nearly two hundred years in which death occurred at a young and highly variable age, European data shows that the average age of death and the variability in the age of death changed dramatically (Wilmoth and Horiuchi 1999; Robine 2001). 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). It is not clear whether this shift towards much later mortality and a much greater concentration of mortality at older ages occurred similarly for different population groups living within the same country, since few studies have simultaneously considered both questions of social inequalities in health and epidemiological transitions in health and mortality. In this study, I examine these between-group differences in within-group variability in survival by age for white and non-white men and women in the U.S.

A wealth of literature on social inequalities in health describes the differences in *average* levels of mortality between population groups such as those defined by race and social class (for example see: Williams and Collins 1995; Mackenbach and Kunst 1997). These studies describe how a range of factors --social, economic, 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. Within this research, the study of variability has emerged only recently, and it has focused on variability in resources (i.e. the relationship between income inequality and life expectancy) (Lynch et al. 2004; for a review see: Wilkinson and Pickett 2006), not variability in health.

To date, only one previous study has examined whether the patterns of variability in the age of death have been similar for different social groups in the U.S. at different periods of time (Edwards and Tuljapurkar 2005). Though this is an important first step in exploring between group differences in within-group variability, it provides only initial insights into historical patterns in inequality. The study focused on mortality patterns after 1950; thus it misses the dramatic changes in infant and child mortality that occurred at the turn of the century which were integral to the second epidemiological transition.

This study integrates the methodological approaches from demography and gerontology with theoretical and conceptual frameworks from medical sociology and social epidemiology to consider differences in mortality dynamics over the last century for white and non-white U.S. men and women. The consideration of between-group differences in within-group variability in survival by age raises new questions that are relevant to understanding the dynamics and determinants of social inequalities in health. For example, have changes in life expectancy and changes in the age-distribution of mortality occurred similarly for subpopulations living in the same nation (e.g. different racial groups)? Has there been a similar composition of epidemiological transitions by age and cause across populations and subpopulations? And has the pace of transitions been similar for subnational populations exposed to different social, economic, geographical and political conditions?

Theorizing Dynamics and Determinants of Variability

The theoretical frameworks used to understand inequalities in *average* levels of health offer an initial entrée for considering potential differences between population groups in the dynamics and determinants of *variability* in health. In specific, Smith (1983) suggests that long-term social inequalities in health involve one of three patterns: *converging* inequalities over time, *diverging* inequalities, or *stable* inequalities. I relate these patterns of inequality to theory on modernization and development, social stratification, diffusion, and human ecology in order to develop a theoretical framework that specifically addresses differences in the variability in survival by age.

Discussions about the relationship between technological development and improvements in health and longevity (for example: Omran 1971; McKeown 1976; Fogel 2000) provide insight into why the distribution of death might change over time. In this sense, the distribution of death provides information about the extent to which a population is able to control environmental shocks and their health consequences (such as famine and infectious disease which were tampered, respectively, through the first and second epidemiological transitions). Modernization and development, particularly the globalization of economic structures and norms which occurred over the later half of the twentieth century, could then be associated with increasing similarity in determinants of health and longevity between population groups (e.g. between industrialized and industrializing nations). This process would lead to *converging* inequalities in health for socially stratified population groups.

By incorporating modernization and development perspectives with social stratification, the potential for *stable* inequalities in health emerges. For example, Link and colleagues (1998)

describe how the distribution of resources and technological innovations are socially stratified in a pattern analogous to the social stratification of health and longevity. They focus on innovations associated with reductions in chronic disease mortality (i.e. the pattern of midlife and late adult mortality that comprises the third epidemiological transition), such as smoking cessation and cancer screening. According to this framework, persistent stratification has been a “fundamental cause” for persistent social group differences in health and longevity. The persistent stratification of resources and innovations leads to *stable* inequalities in health for socially stratified population groups.

Finally, a life course and development perspective on historical trends in health and longevity encourages one to consider differences in health within a cohort as it ages, and intergenerationally as cohorts replace one another. 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 (Carey and Judge 2001). I apply, to population groups, theory about accumulated disadvantages and advantages that has been developed with respect to individuals (Ross and Wu 1996; Beckett 2000). Thus, it is possible to hypothesize how resource accumulation can shape the health of population groups as they age and give birth to new cohorts.

Increasing differences in health between population groups would be expected when generations of accumulated inheritance, that is important for health, is passed from one generation to the next. This inheritance might not only involve social and economic resources (ranging from behavioral norms about prevention to wealth), but also biological precursors to longevity and successful aging. Thus, social inequalities in health would *diverge* because of

social group differences in the intergenerational accumulation of health-related norms and resources.

Measures of Variability

A measure of variability or heterogeneity, such as the interquartile range (*IQR*) of the distribution of deaths in the life table, is a particularly useful and appropriate indicator for questions such as those raised here about the differences in the timing, pace, and composition of epidemiological transitions in population groups. As a population ages changes in the age distribution of mortality provide information about the pace of an epidemiological transition and the changes in the causes of mortality associated with an epidemiological transition (Robine 2001). A widening age-distribution of mortality indicates when the beginning of changes in the age-composition (and by extension, epidemiological or cause-specific composition) of mortality occurs. In contrast, an age distribution that becomes narrower indicates the completion of either a forward or a reverse transition.

For example, changes in the age-distribution of mortality indicated by the *IQR* were used by Wilmoth and Horiuchi (1999) to describe the first, second and third epidemiological transitions in Sweden. They showed how, at the turn of the century, the distribution of mortality compressed dramatically (e.g. the *IQR* dropped). Their decomposition of changes in *IQR* into the age-specific mortality revealed that the compression of mortality was due to reductions in childhood and infant mortality that have associated in other studies with cause-specific declines in infectious disease (Wilmoth and Horiuchi 1999). Examination of between-group differences in the *IQR* and the decomposition of the *IQR* into its age-specific mortality components,

therefore, provides information about the age-composition of epidemiological transitions (Wilmoth and Horiuchi 1999).

A number of measures of variability or heterogeneity other than the *IQR* have been used in studies of mortality. Measures such as Keyfitz's *H-index*, entropy, the gini coefficient and the inter-quartile range (among numerous others) have been used to explore differences in life expectancy in various populations and subpopulations (i.e. subpopulations defined by sex), as well as differences in the process of rectangularization and the compression or expansion of mortality (Keyfitz and Golini 1975; Nusselder and Mackenback 1999). In their review of these indicators, Wilmoth and Horiuchi (1999), show that the various measures of variability are highly inter-correlated. They advocate using the *IQR* because it is relatively easy to calculate and interpret.

Recently, Edwards and Tuljapurkar (2005) have developed an alternative interquartile range that excludes mortality changes in infancy and childhood. This measure, which I will refer to as *IQR10* (note, they refer to the measure as S10) calculates the interquartile range of the distribution of mortality after age 10 rather than after age 0, or *IQR₀*. I use *IQR₀*, the measure advocated by Wilmoth and Horiuchi (1999), so that I can observe whether there were differences between whites and non-whites in the changes in infant and child mortality. These changes in mortality before age 10 have been associated in other literature with the second epidemiological transition (Arriaga 1984; Pollard 1988; Armelagos et al. 2005). In the discussion, I address the differences in the findings when *IQR10* is used instead of *IQR₀*.

I apply findings from previous studies on epidemiological transitions and changes in infectious disease mortality, maternal mortality, chronic disease mortality, and the extension of

later life to guide my examination of the differences in the timing, pace and age-composition of changes in mortality among white and non-white men and women in the U.S. In light of these findings, I decompose variability into age-specific mortality patterns that first allow me to focus on the timing and pace of changes in mortality in: infancy and childhood, midlife, and later life. I also conduct an analysis that focuses on mortality that might be associated with childbearing in midlife.

Previous studies have not explicitly addressed the benefits of using a measure of variability, such as IQR_{θ} , to study epidemiological transitions versus a measure of central tendency, such as the e_{θ} . Nor has previous literature related the within and between group differences in these demographic indicators to the historical changes in social inequalities in health. Thus the theoretical grounding for the use of these demographic indicators in studies of social inequality in health has been poorly developed. I use the temporal changes in the IQR_{θ} between 1900 and 2002, and the decomposition of these changes for whites and non-whites, to examine whether social inequalities in mortality in the U.S. have followed a theoretical pattern of converging, stable, or diverging inequalities in mortality.

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. The age-specific mortality data for every year between 1900-2002 is used to estimate life expectancy at age 'x', e_x , and the interquartile range of the age-distribution of deaths from age 'x' to the end of the life table, IQR_x . Then, differential changes in IQR_x over time are decomposed into age-specific mortality changes. This is conducted for each of the four population groups (i.e. white women, non-white women, white men and non-white men).

Then, I calculate two different ratios that allow me to examine the temporal pattern of racial differences in mortality among men and women (i.e. four ratios in total). These include the ratio of non-white versus white e_0 (i.e. (e_0 for non-whites) / (e_0 for whites)) and the ratio of non-white versus white IQR_0 (i.e. (IQR_0 for non-whites) / (IQR_0 for whites)). The changes in these ratios over time for men and for women are depicted in Figure 3.

Finally, I estimate 15-year changes in the age distribution of deaths for each of the four population groups using data from the beginning and end of seven different 15-year periods between 1900 and 2002¹. The decomposition of these 15-year changes in the IQR_0 is calculated using a method developed by Wilmoth and Horiuchi (1999). As noted above, the life table IQR_0 at time t is defined as the difference between the 25th and the 75th percentiles of the life table distribution of deaths such that $IQR(t) = x_{75}(t) - x_{25}(t)$. A change in the 'p'th percentile of the life table distribution of deaths between time 't₁' and 't₂' can be equated with changes in the force of mortality as follows:

¹ Note that the change between 1990 and 2002 is a twelve year change, and all equations are modified to accommodate this difference from the rest of the data.

$$\begin{aligned}\Delta x_p(t_1, t_2) &= x_p(t_2) - x_p(t_1) \\ &= \int_{t_1}^{t_2} \int_0^\infty -\frac{\partial}{\partial t} \mu(x, t) \cdot I_p(x, t) dx dt,\end{aligned}\quad (1)$$

where

$$I_p(x, t) = \begin{cases} 1 & \text{if } x < x_p(t) \\ 0 & \text{else} \end{cases}, \quad (2)$$

and where

$$\rho(x) = -\frac{\frac{\partial}{\partial t} \mu(x, t)}{\mu(x, t)} = -\frac{\partial}{\partial t} \ln(\mu(x, t)) \quad , \quad (3)$$

which is the relative rate of the mortality decline. Using these relationships, the change in the IQR_0 from between time ‘ t_1 ’ to ‘ t_2 ’ can be written as follows:

$$\begin{aligned}\Delta IQR_0(t_1, t_2) &= \Delta x_{75}(t_1, t_2) - \Delta x_{25}(t_1, t_2) \\ &= \int_{t_1}^{t_2} \int_0^\infty -\frac{\partial}{\partial t} \mu(x, t) \cdot \left[\frac{I_{75}(x, t)}{\mu(x_{75}(t), t)} - \frac{I_{25}(x, t)}{\mu(x_{25}(t), t)} \right] dx dt.\end{aligned}\quad (4)$$

This last relationship allows me to decompose the changes in IQR_0 from one time period to the next into changes in mortality reductions (or increases) in specific age groups. I have modified the Wilmoth and Horiuchi method to accommodate for the age-grouped data used in this study (ranging from single year to ten-year age groups). The numerical methods for calculating the integral in Equation 4 are described in Appendix A.

Figures 4 and 5 depict the age-composition of changes in the IQR_0 for men and women by race. Some age-groups from the original dataset are combined for ease of presentation and interpretation: infant and child mortality is age-group 0 and age-group 1-4 years; adolescent and young adult mortality is age-groups 15-24, 25-34, and 35-44 years; midlife is age-groups 45-54

and 55-64 years; and late adulthood is age-groups 65-74, 75-84, and 85 and older. Tabular results of the IQR_0 for the eight years used in the decomposition and age-specific mortality changes in IQR_0 for each population group are reported in Appendix B.

Table 1 highlights the contribution of mortality changes in the childbearing years to the total changes in the IQR_0 in each of the seven periods. Note that the percent contribution depicted in Table 1 is slightly smaller than the equivalent figure for adolescence and early adulthood depicted in Figure 5. Both percentages are calculated from the decomposition findings in Appendix B; however, Table 1 focuses explicitly on the ages most likely to contribute to maternal mortality (i.e. ages 15-34 rather than ages 15-44).

FINDINGS

What are the historical trends in IQR_0 and e_0 in the U.S. over the last century?

The trends in the average expected survival and the variability in survival by age are depicted for white and non-white men and women in Figures 1 and 2. Life expectancy increases roughly linearly from 1900 through 1950 for both men (Figure 1) and women (Figure 2), with the non-white to white gap in e_0 diminishing by nearly one half. Similarly, over the same period, there is an overall pattern of decline in the variability of the life table distribution of ages of death (IQR_0) for whites and non-whites. Following 1950, there is stagnation in the pace of changes in e_0 and IQR_0 . Two stepped increases in e_0 are particularly marked for non-white men and women; they occur over the period 1955 to 1970 and then 1975-1980 to 1995.

[Figure 1 & Figure 2 about here]

As a result of differences by race in the timing of declines in the IQR_0 (shown in Figures 1 and 2 and discussed in greater detail below), the trends in the IQR_0 first diverge and then

converge when comparing whites with non-whites. Reduction in IQR_0 occurs first for whites over the period 1900 to 1915. In this period, high variability in the age of death (IQR_0 about 60) persists for non-whites; thus, there is white to non-white divergence in age variability between 1900 and 1915. After 1915, convergence proceeds (with marked convergence between 1930 and 1950) as IQR_0 declines at a faster pace for non-whites than it does for whites.

The changes in the average age and variability of the age of death involve a crossover that differs by race and sex. At the turn of the century, the IQR_0 is greater than (or equal to) e_0 in every group. By the end of the century, IQR_0 is substantially smaller than e_0 . The trends in e_0 and IQR_0 crossover in about 1900 for white women, 1905-1915 for white men, 1920-1925 for non-white women, and 1925 for non-white men. At the beginning of the century, the e_0 is 50% to 75% of IQR_0 . By the end of the century, the e_0 is 3 to 4 times larger than IQR_0 . The changes in IQR_0 reflect dramatic rectangularization of the survival curves in all groups.

Finally, it is important to note that the 1918 Influenza Epidemic produced radical changes in the age distribution of survival in all subnational population groups. Differences between whites and non-whites in the age distribution of mortality were eliminated and life expectancy dropped precipitously. Consideration of the implications of the 1918 Influenza Epidemic for subnational differences in survival is beyond the scope of this project; therefore, these data are not included in subsequent analyses.

Does e_0 provide the same information as IQR_0 about differences in mortality patterns for men and women and for whites and non-whites?

The ratio of IQR_0 for non-whites versus whites and the ratio of e_0 for non-whites versus whites are contrasted in Figure 3. These ratios show how the relative differences in mortality

have changed over time, where a ratio of one indicates equality. As has been noted earlier in the discussion of Figures 1 and 2, the overall pattern of racial differences in e_0 involves *convergence*, and a pattern in which e_0 for non-whites is consistently *less* than the e_0 for whites. Over the last century, e_0 for non-whites versus whites has become increasingly more similar. At the turn of the century, e_0 for non-whites was about 65 to 70 percent of e_0 for white women and men, respectively. By the end of the century, the ratios were near one, with non-white e_0 about 95 to 97 percent of e_0 for white men and women, respectively.

[Figure 3 about here]

In contrast, IQR_0 has exhibited a dynamic trend that involves both *divergence and convergence*, and a trend in which the IQR_0 for non-whites is consistently *greater* than the IQR_0 for whites. First, there is an overall pattern of divergence in the first three decades of the century, as the differences between whites and non-whites become larger. Then, over the later part of the century the differences between whites and non-whites converge, with the ratio of IQR_0 for non-whites versus whites returning to its original value of about 1.2 at the turn of the century. At the points closest to equality, in 1900 and in 1990, IQR_0 for non-whites is about 20 percent larger than IQR_0 for whites. At the points most divergent from equality, in about 1945, IQR_0 for non-whites is 50 to 70 percent larger than IQR_0 for white men and women, respectively.

It is notable that the diverging and converging temporal trends in the ratio of IQR_0 are more irregular than the temporal trend of convergence for the ratio of e_0 . In addition, it is also noteworthy that there are gender differences in the extent of divergence and later convergence in IQR_0 between 1925 and 1955. The ratio of IQR_0 for women involves more divergence than it does for men as differences between non-white and white women continue to grow larger after

about 1930. This is the time point when the ratio of non-white-to-white IQR_0 peaks for men. For women, IQR_0 doesn't peak until about 1940. The pattern for men remains relatively stable and at its maximum over the period 1920 through 1940. These gender differences in the non-white-to-white ratio of IQR_0 are not observed for the non-white-to-white ratio of e_0 . The role of childbearing related gender differences in mortality patterns during this time period are considered in greater detail below.

What are the predominant patterns of historical change in IQR over the last century?

The mortality changes that produce periods of narrowing, stable and even a widening distribution of mortality over the last century are better understood by decomposing 15-year changes in IQR_0 into age-specific changes in mortality. Trends for white and non-white men and women are plotted in Figures 4 and 5.

Changes in the distribution of mortality in each 15-year period involve the combination of two trends: 1) *decreases* in age-specific mortality in some age-groups that *reduce* the width of the IQR_0 and 2) *increases* in age-specific mortality in other age-groups that *increase* the width of the IQR_0 . The positive and negative age-specific mortality contributions to the changes in the IQR_0 are plotted in Figures 4 and 5. For example the reduction of the IQR_0 between 1900 and 1915 among white men is determined by a large reduction in mortality in the ages 0-4 (of about -19 years) and modest reductions in the ages 5-14 and 15-44 (of about -4 years). The declines in mortality at young ages dramatically compensate for the very minimal increases in mortality in older ages (i.e. there was only a small increase of 0.5 years for the ages 45 years and older.) As a result, the overall change in IQR_0 for this period (1900-1915) involves a compression of the distribution of mortality towards older years (by about -22 years, see Table 1).

[Figure 4 and Figure 5 about here]

Figures 4 and 5 reveal that, in nearly every period, the relative balance of both compression (i.e. negative change in IQR_0) and expansion (i.e. positive change in IQR_0) for white and non-white men and women has produced, overall, a compression of mortality to older ages. Only in the period 1960 to 1975 is the expansion of mortality in the older ages equal to or greater than compression in the younger ages. Among white women in particular, increases in mortality between 1960 and 1975 were greater than the reductions in mortality such that there is a total 2.6 year expansion in mortality in this period (Table 1).

The compression of mortality associated with declines in infant and child mortality is large over the first half of the century. Between 1960 and 1975, there is stabilization and even expansion of mortality as increases in mortality in later life balance or even exceed reductions in early life. Then for the last three decades, there are small reductions in IQR_0 , primarily determined by reductions in midlife mortality.

There is evidence for rectangularization among women via the declining mortality expansion in ages 75 and older at the end of the century. Among white women, in particular, IQR_0 expansion attributable to ages 75 and older diminishes from a peak of 3.2 years in the period 1960-1975 to 0.1 years in the period 1990-2002. Similarly in non-white women, expansion of mortality in those 75 years and older slows considerably after the peak expansion in 1960-1975 and then expands again only slightly in the period 1990-2002 (Appendix B, Tables 3 and 5).

Are there differences between men and women in the change in IQR_0 attributable to changes in mortality during childbearing years?

The comparison of the trends in mortality for men and women observable in Figures 3, 4 and 5 (described above) reveals that, throughout the first half of the century, the compression of mortality attributable to mortality declines in midlife is consistently larger for women, and especially white women. There is a difference in the timing of the compression of mortality for whites and non-whites, such that Figure 3 shows that there is a widening, or divergence of mortality patterns for women during the 1920s, 1930s and 1940s. The decomposition of changes in IQR_0 depicted in Figures 4 and 5 also show that declines for white women attributable to declines in the age group 15-44 years (i.e. the diagonally striped shading) are especially marked in the periods 1915-1930 and 1930-1945. In contrast, similar declines in the age group 15-44 years for non-white women do not occur until the periods 1930-1945 and 1945-1960.

Table 1 compares the age-specific contribution of changes in the distribution of mortality for men and women during the childbearing years (i.e. ages 15-34 years). This is a slightly smaller age grouping than that displayed in Figures 4 and 5 which may more accurately differentiate sex differences in mortality associated with childbearing. For both white and non-whites in the first half of the century (with the exception of the period 1915-1930 among whites²), reduction in mortality during the childbearing years contributes 1.5 to 3 times as much to the overall compression of mortality among women as it does to overall compression of mortality among men in these three 15-year periods.

[Table 1 about here]

² It is noteworthy that mortality in the ages 15-34 years contributes about as much –and in fact slightly more-- to the compression of mortality among white men as it does among white women, i.e. 35% and 31% for white men and women respectively (Table 1).

For example between 1930 to 1945, mortality decline in the ages 15-34 years contributes to 36% of the overall reduction in the IQR_0 among white women, while it only contributes to 25% to the overall reduction in IQR_0 among men (Table 1). As a result, these declines in mortality contribute nearly 1.5 times as much to the compression of mortality among women as they do among men. In the same period among non-white women, mortality declines in the ages 15-34 years contribute to 100% of the overall reduction in the IQR_0 , while they contribute to only 54% of the overall reduction in the IQR_0 among men (Table 1). As a result, the compression of mortality which can be accounted for by declines in mortality during women's childbearing years account for nearly 2 times as much of the compression of mortality in the period for women as they do for men.

Are there differences between whites and non-whites in the timing and age composition of changes in IQR_0 ?

The trends in the positive and negative 15-year changes in the IQR_0 plotted in Figures 1 and 2 reveal that the age-composition and the timing of mortality changes are different for whites and non-whites. Over the first part of the twentieth century, reductions in the IQR_0 in the white population became exponentially smaller in size. At the turn of the century, 15-year change in the total IQR_0 was as much as -20 and -22 years (men and women respectively), with half as large of a change in the following 15 year period and nearly one-quarter as large of a change in the subsequent 15-year period (Table 1). This exponential decline in mortality produced a relatively rapid compression of the distribution of the mortality towards older ages.

In contrast, trends in the distribution of mortality in the non-white population have been more irregular, delayed, and less rapid. The greatest declines in the IQR_0 occur 15 years later

among non-whites than they do among whites (Table 1). At the turn of the century, the IQR_0 is large and changes in the IQR_0 among non-whites were small (and even positive among men) (Figures 5 and 6). It is not until the period 1915-1930 that the 15-year change among non-whites is at its peak, with a decline in IQR_0 of -10 and -14 years (men and women respectively, Table 1). This is an overall smaller total reduction in IQR_0 than occurred among whites in the period of peak change (i.e. the peak compression was $-9.9/-19.5 = 51\%$ smaller for non-white women than white women, Table 1) In addition, the pace of change following this period of peak change in IQR_0 is faster among whites than among non-whites.

There was a much faster rate of decline in the compression of mortality for whites than non-whites. In the period following the greatest amount of mortality compression among whites, there was less than half as much compression in the IQR_0 as there had been in the peak period (i.e. for white women $-9.4/-19.5 = 48\%$, see Table 1). Among non-whites, there was more stability in the compression of mortality from the peak period to the next. Reductions in IQR_0 in the 15-years following the period with the greatest mortality compression for non-whites remained relatively large –more than two-thirds of the size of the peak period (i.e. for non-white women, $-8.2/-14.2 = 70\%$).

As noted earlier, the compression of mortality in 1900-1915 among white men is primarily attributable to reductions in infant and child mortality. This is also the case among white women; 76% and 86% of the mortality change in this period for white women and white men is attributable to declines in mortality in the ages 0-4 years (Appendix B, Tables 2 & 3). In addition the predominant contribution of changes in infant and childhood mortality (about 80%

of ΔIQR_0) to the peak change in IQR_0 is also observed among non-whites, despite the 15-year delay in the compression of mortality.

Figures 5 and 6 reveal that while the compression of mortality is delayed in infancy and childhood among non-whites, the timing of age-specific expansion of mortality has irregular patterns of similarity and difference for whites and non-whites. There is similar timing of mortality expansion in late adulthood (i.e. 65+ years) for whites and non-whites. Among women, expansion in the ages 65+ years peaks in the period 1960-1975, and among men it peaks (or at least is at maximum) in the period 1990-2002.

In contrast, the timing of mortality expansion in midlife (i.e. 45-65 years) is different for whites and non-whites. The changes in IQR_0 , that are attributable to expansion of mortality in midlife, peak for non-whites in the period 1930-1945. For non-white women there is nearly a 5 year increase in the IQR_0 attributable to increases in mortality in midlife (which is compensated by the large declines in mortality at ages younger than 45 years). This type of expansion attributable to increases in midlife mortality is not clearly observed among whites over the twentieth century. In fact, maximum values of mortality compression in the age group 45-65 years do not have a clear pattern by sex or race (Figures 4 and 5). There is a maximum in the period 1990-2002 for white men, 1945-1960 for white women, and 1990-2002 for non-white women, and only very minimal reduction in 1990-2002 for non-white men (Appendix B).

Finally, in the period 1960-1975, there is mortality expansion among non-white men that is attributable to increases in mortality in late childhood or early adolescence that is not observed in any other population group.

DISCUSSION

This study of the average age of death and the variability in the age of death for two social groups in the U.S. provides theoretical and empirical insights on the determinants and dynamics of survival over the last century. First, the differences in timing, pace and composition of mortality changes for white and non-white men and women suggests that a modernization and development theoretical approach to population change, in which groups become more similar with increasing economic development, does not fit the data. Secondly, my analysis identifies a dynamic pattern of divergence and convergence in the indicator describing mortality variability (i.e. IQR_0) that is not observed in the measure of central tendency (i.e. e_0). This suggests that several empirical indicators may be necessary to fully understand population change.

The overall convergence in the magnitude of racial differences in indicators of the central tendency of mortality over the last century (i.e. average mortality rates and e_0) at first blush supports previous arguments for the overall salubrious effects of economic development and modernization across populations (McKeown 1976; Fogel 2000). So does the similar timing of reduction in mortality during childbearing years among both white and non-white women between 1930-1945. Consistent with theories about the role of technological development, this is a period when previous research has suggested innovations to reduce maternal mortality began to be fully realized (Berry 1977; United States Department of Health and Human Services (US DHHS) et al. 2004)³.

However, closer examination of the mortality overall trends using an indicator of the variability of survival reveals that the changes in IQR_0 among whites predominantly have been a

³ These innovations included the institutionalization of practice guidelines, increases in trained birth attendants, maternal mortality review committees, and medical technologies to improve aseptic conditions and blood transfusions.

combination of larger, earlier, and more rapid declines in mortality than those experienced by non-whites (see Figures 1 and 2). This is most dramatically observed via the 15-year lag in the reductions in infant and child mortality and the associated fifteen year lag in the second epidemiological transition. In addition, comparisons of the decomposition of IQR_0 change for whites and non-whites, reveals that non-whites experienced unique age-patterns of mortality change that were not a delayed or compressed equivalent of mortality patterns among whites. In particular, there is a large increase in midlife mortality among non-White men and women in 1940-1945 that offsets reduction of mortality in early life to an extent never observed among whites during the period (see Figures 4 and 5). Moreover, there are increases in mortality in adolescence and young adulthood between 1960-1975 for non-white men, perhaps due to increases in homicide among black men (Elo and Drevenstedt 2004), that are not observed among white men or white and non-white women.

Although these data do not support a theoretical framework in which economic development “rises all boats”, it is not clear which of the other two alternative theoretical frameworks, the stratified diffusion of technology or spatially stratified human ecology, the data support. There are differences in the trends of e_0 and IQR_0 in the U.S. for two subnational populations (whites and non-whites) who have experienced very different social, political, cultural, and even environmental exposures and resources. These differences could support a theoretical framework in which mortality change in different age-groups occurs at a different pace for whites and non-whites due to stratified diffusion of technology (where technology is comprised of both resources and knowledge), consistent with Link and Phelan’s “fundamental

cause” approach to social inequalities in health. At the same time the differences could also suggest that each population has experienced a different social ecology of mortality.

The fifteen year delay in child and infant mortality improvements in the non-white population may provide a lever for future analyses which seek to distinguish between, or even integrate, the “fundamental cause” approach and the social ecology approach to the differences in epidemiological transitions. If “public goods”, such as sanitation systems, clean water, and public health interventions leading to improvements in the milk supply, were some of the primary determinants of reductions in mortality at the turn of the century (Szreter 2004; Cutler and Miller 2005), then, in order to reflect the persistent stratification of technology described by Link and colleagues, there would need to be differences in the distribution and access to these supposedly “public goods” for whites and non-whites. There is some evidence that sanitation infrastructure important for mortality declines at the turn of the century did reach blacks later than whites (Troesken 2002). Troesken’s study, however, also reported substantial between-city variation that could be interpreted as support for the differential social ecology hypothesis.

Although the “fundamental cause” framework most clearly lends itself to an interpretation of stable inequalities, and in fact this is the shape of trends in inequalities that have been described by Link and Phelan (Link and Phelan 2002), I suggest that a pattern of persistently diverging and converging inequalities is also consistent with social stratification theory described in the “fundamental cause” approach. The stratified diffusion of technologies may not proceed smoothly from one innovation to the next. As a result, these new technological shocks (in which there are innovations in knowledge, industry, medical advancements, etc.) may

produce periods of divergence, convergence, and stability in the distribution of health within and between populations over time.

Future analyses which associate the temporal and spatial variability in mortality with the temporal and spatial distribution of technology could provide promising new insights into the emergence of social inequalities in mortality. In addition, consideration of differences in cause-specific composition of mortality changes may help to illuminate whether social stratification has lead to completely unique patterns of epidemiological transition.

There are data limitations which require that some of my conclusions about racial differences in the heterogeneity of survival be made cautiously. First there is a data quality issue determined by the reporting of social group categories. By considering the mortality experiences of white and non-white men and women, I seek to capture differences in resources and social conditions associated with the history of racism in the U.S. and the implications of race-based residential isolation and racial homogamy in marriage and family formation. These groups offer an admittedly porous and crude categorization of racial status that is necessitated by my desire to study historical trends in U.S. mortality from the second epidemiological transition forward.

Persistent heterogeneity in one group may be determined by historically heterogeneous group membership, typically arising from changes in immigration. I am most concerned about the implications of immigration for the white to non-white trends observed during the later half of the twentieth century. This is a period when increases in immigration by Hispanics and Asians (Rumbaut 1994) may have broadened the extent of heterogeneity in mortality (because expected age of death in these two non-white subgroups is lower than it is for blacks). However as mentioned earlier, the immigration trends potentially leading to heterogeneity in the non-white

category are not concurrent with my major findings about the declines in infant and child mortality. These findings are observed during the first three decades when the composition of the non-white racial group is predominantly black (analysis available from author upon request). In fact, if composition bias influenced my major findings about the delay of the second epidemiological transition, they would arise from differences in the racial/ethnic composition of the 'white' category due to the changing waves of German, Irish, Jewish, and Italian immigration (Pedraza 1995).

Secondly, there are issues of data quality and coverage. Between 1900 and 1933, the death registration area expanded from 10 states to coverage of the entire continental U.S. (Grove and Hetzel 1968). Previous analysis of racial differences in mortality excluded data prior to 1930 due to the poor representation of southern states (Preston et al 2003). This was an important consideration because most of the blacks lived in the south during the early twentieth century (Warren 1997). The previous analyses also developed methods to address the undercount of blacks in the registration systems and census (Preston et al. 2003). Nevertheless, these corrections in childhood and infancy were minimal relative to corrections in older age-groups. Since I find that the most dramatic differences in IQR_0 involved changes in childhood and infancy, I am less concerned about the issues of data quality raised in these previous analyses than data coverage.

Coverage is a greater concern because the major declines observed in non-white mortality occurred in 1930-1945, when data coverage in the continental U.S. became complete. Incomplete mortality coverage in the south prior to the 1930s would be a problem if mortality was lower in the south and declined quicker than it did in the north. Although it is the case that prior to 1900

mortality among blacks in the south was lower than blacks the north (typically associated with northern urbanicity, segregation and urban squalor (Ewbank 1987)), there was also often great within-city variation in the black-white differences in mortality during this period (Warren 1997). The temporal variations in mortality and their reduction across white and non-white populations, albeit at different rates, between 1900-1950 might provide as much support for the role of 20th century technologies in reducing infant, child and maternal mortality as they do support for the potential role of bias in the findings. In fact, the regional differences in mortality, their association with urban-rural mortality patterns, and the timing of the Great Migration of blacks from the rural south to urban areas in the north all suggest that the findings observed here are not data artifacts but complicated patterns of migration, social stratification and diffusion of technology.

CONCLUSION

In this study, I have examined a century of racial differences in the average level and distribution of mortality for men and for women in the U.S. By comparing the changes in heterogeneity from one period to the next, I have been able to identify differences in the timing of mortality compression and mortality expansion at various ages for white and non-white men and women. These trends have provided insight into the theoretical relationships between economic development, institutional change, stratification, and social group differences in mortality. They have also suggested that there are differences in the cause-specific patterns of mortality in these two population groups that have been associated with the changes in infectious

and chronic disease mortality during the second and third epidemiological transitions. Future research might further examine the role of mortality in intergenerational cycles of social stratification, or it may seek to better understand how institutionalized racism shapes more or less healthy social environments in the U.S.

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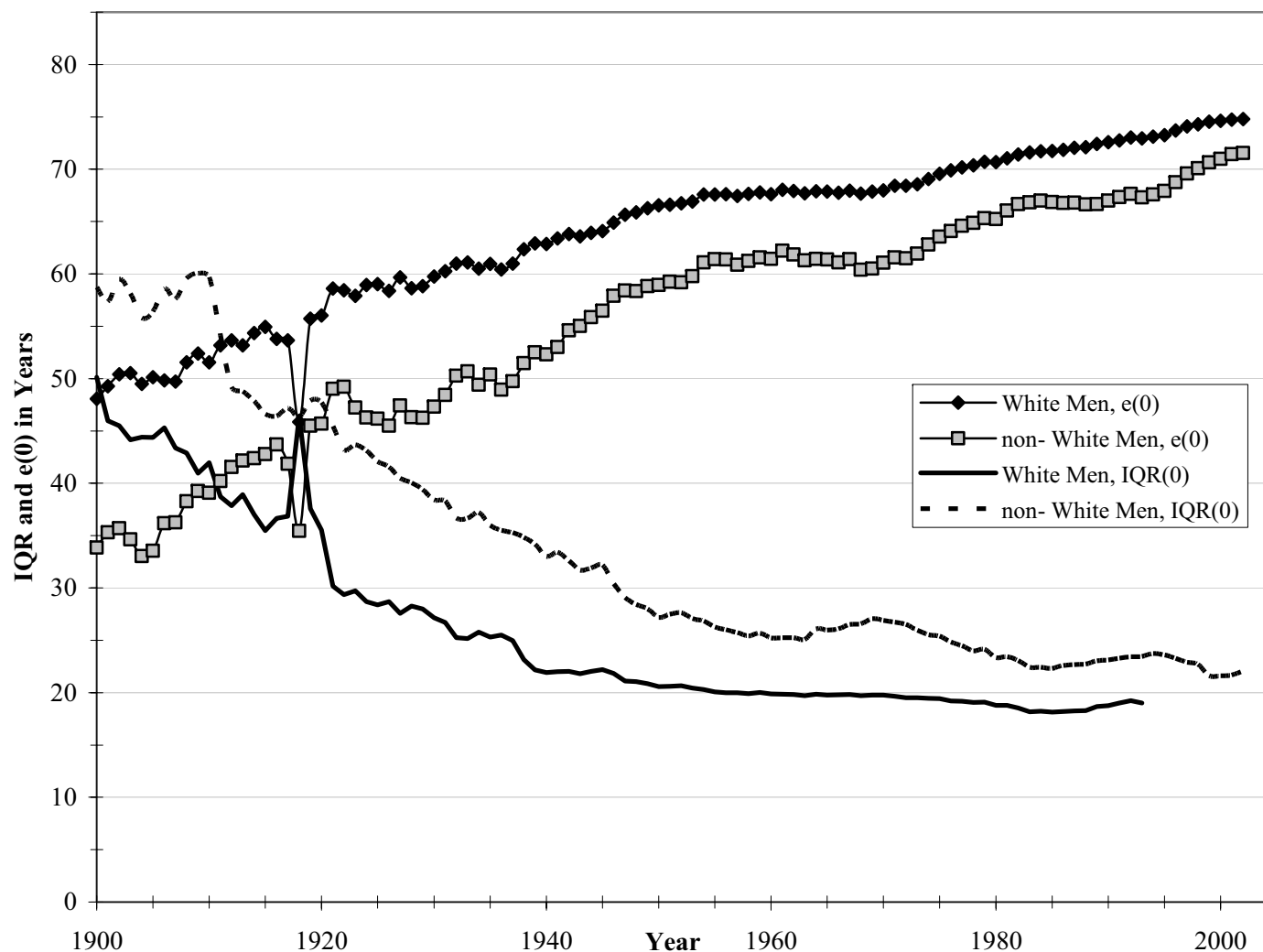
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Table 1. Total Change in the Life Table Interquartile Range (ΔIQR_0), ΔIQR_0 Attributable to Mortality Change in Childbearing Ages (15-34 years), U.S. White & non-White Men & Women, 1900-2002

	Calculated 15-year Change in Years						
	1900-15	1915-30	1930-45	1945-60	1960-75	1975-90	1990-2002
<i>White</i>							
<i>Men:</i> Total ΔIQR_0 , attributable to all ages	-22.0	-9.6	-5.1	-3.6	0.5	0.3	1.6
(1) Subtotal ΔIQR_0 , attributable to ages 15-34	-1.6	-3.4	-1.3	-1.6	0.1	-0.1	-0.3
(2) % ΔIQR_0 , attributable to ages 15-34	7%	35%	25%	44%	20%	-33%	-19%
<i>Women:</i> Total ΔIQR_0 , attributable to all ages	-19.5	-9.4	-7.5	-3	2.6	0.1	-0.5
(1) Subtotal ΔIQR_0 , attributable to ages 15-34	-3.7	-2.9	-2.7	-0.8	-0.1	-0.1	0
(2) % ΔIQR_0 , attributable to ages 15-34	19%	31%	36%	27%	-4%	-100%	0%
<i>Relative Difference: (% $\Delta IQR_{0\text{Women}}$)/ (% $\Delta IQR_{0\text{Men}}$)</i>	2.5	0.9	1.4	0.6	-0.2	3.0	0.0
<i>Non-White</i>							
<i>Men:</i> Total ΔIQR_0 , attributable to all ages	0.5	-14.2	-8.2	-8.5	0.4	-1.9	-1.7
(1) Subtotal ΔIQR_0 , attributable to ages 15-34	0.2	0.2	-4.4	-4.3	0.5	-0.7	-1.3
(2) % ΔIQR_0 , attributable to ages 15-34	40%	-1%	54%	51%	125%	37%	76%
<i>Women:</i> Total ΔIQR_0 , attributable to all ages	-3.8	-9.9	-6.9	-7	0.4	-2.1	-1.2
(1) Subtotal ΔIQR_0 , attributable to ages 15-34	0	-0.4	-6.9	-3.7	-0.7	-0.2	-0.3
(2) % ΔIQR_0 , attributable to ages 15-34	0%	4%	100%	53%	-175%	10%	25%
<i>Relative Difference: (% $\Delta IQR_{0\text{Women}}$)/ (% $\Delta IQR_{0\text{Men}}$)</i>	0.0	-2.9	1.9	1.0	-1.4	0.3	0.3

Figure 1 Historical Trends in Life Expectancy (e_0) & Interquartile Range of Life Table Distribution of Deaths (IQR_0) for U.S. non-White & White Men, 1900-2002*



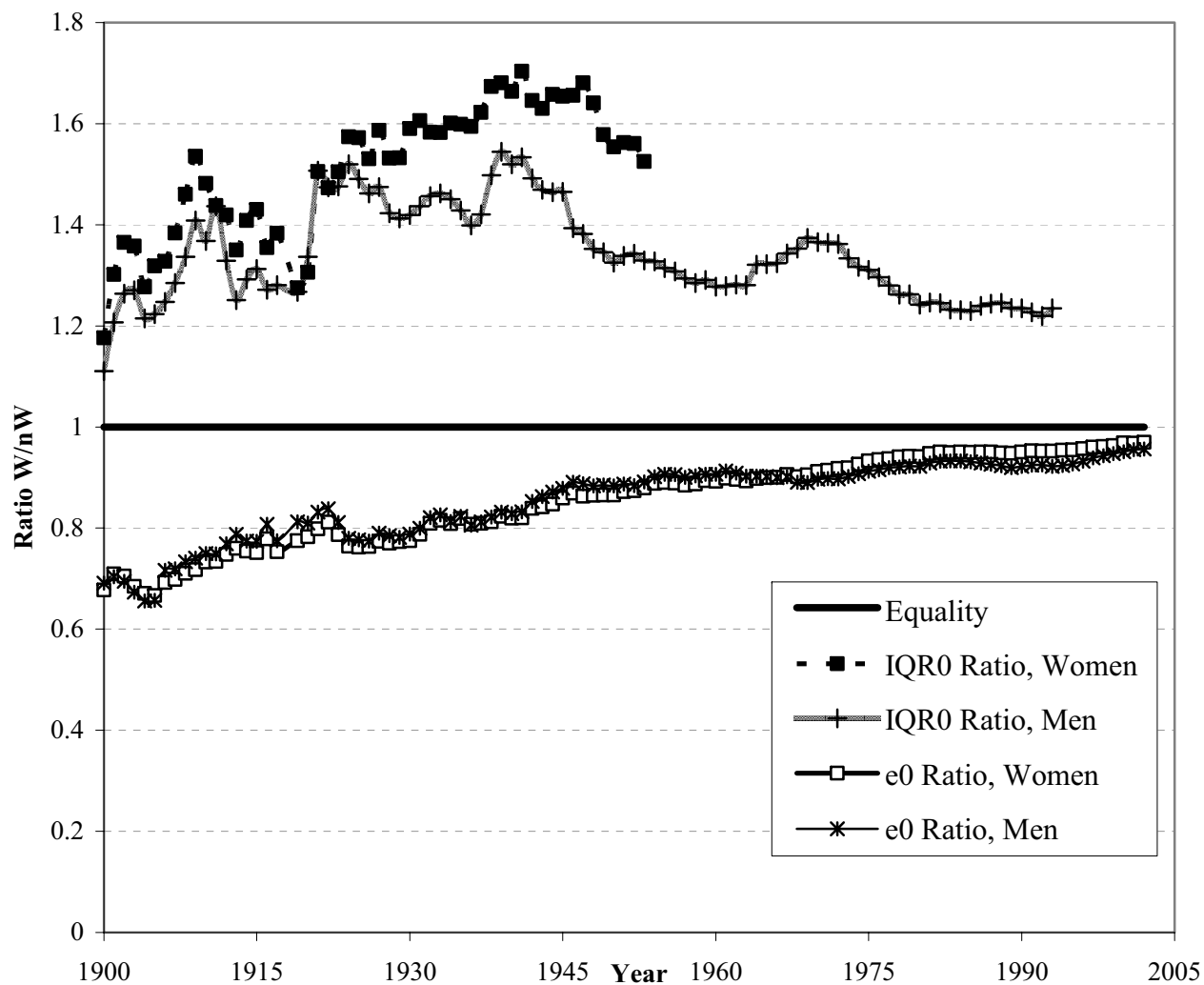
*Note: IQR_0 is not depicted for the years after 1993 for white men because the upper quartile lies above the last age-group of mortality (85+ years).

Figure 2. Historical Trends in Life Expectancy (e_0) & Interquartile Range of Life Table Distribution of Deaths (IQR_0) for U.S. non-White & White Women, 1900-2002



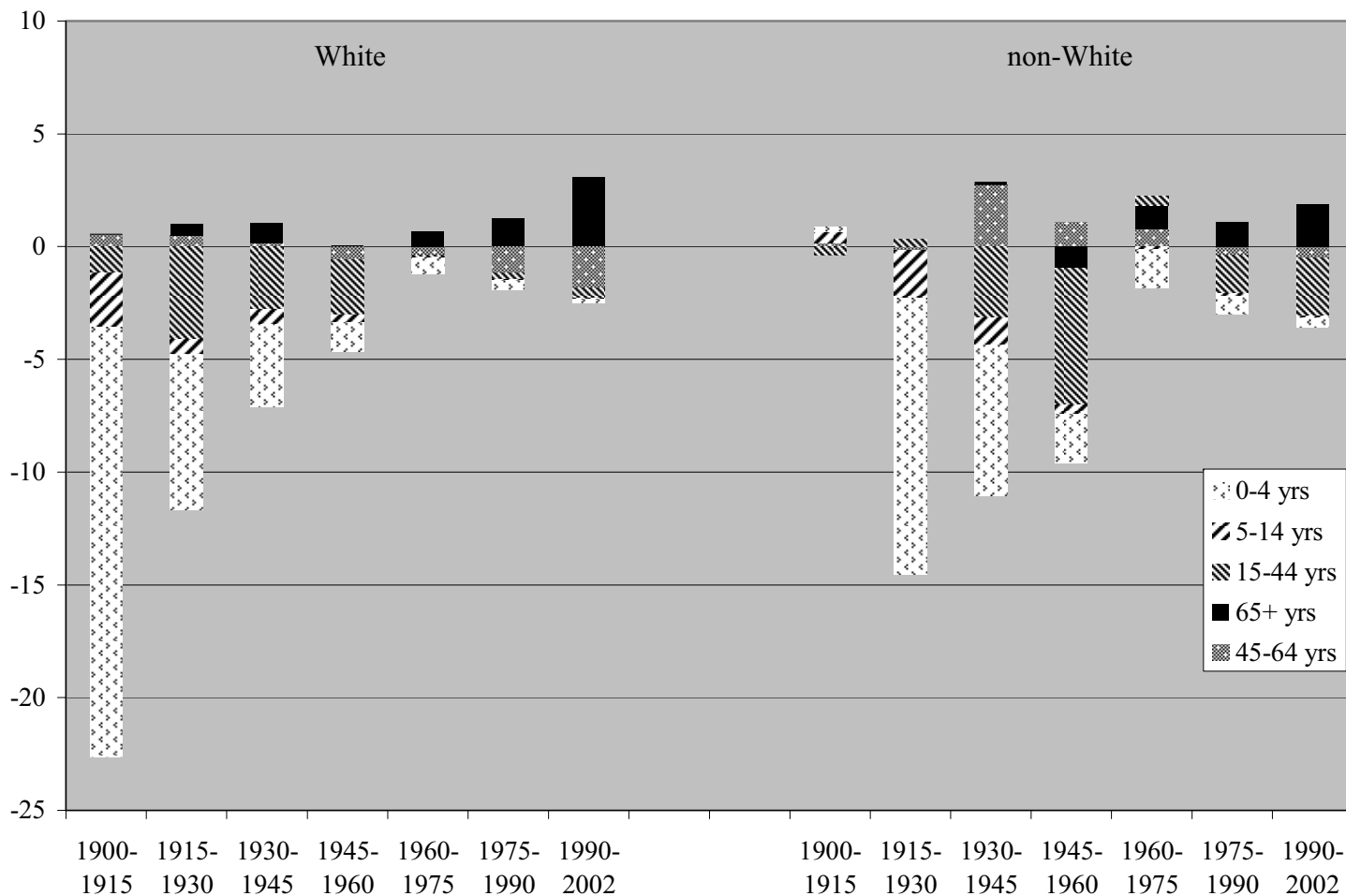
*Note: IQR_0 are not depicted for the years after 1953 for white women and after 1971 for non-white women because the upper quartile lies above the last age-group of mortality (85+ years).

Figure 3: Non-White versus White Ratio of IQR_0 and e_0 for Men and Women, U.S. 1900-2002*



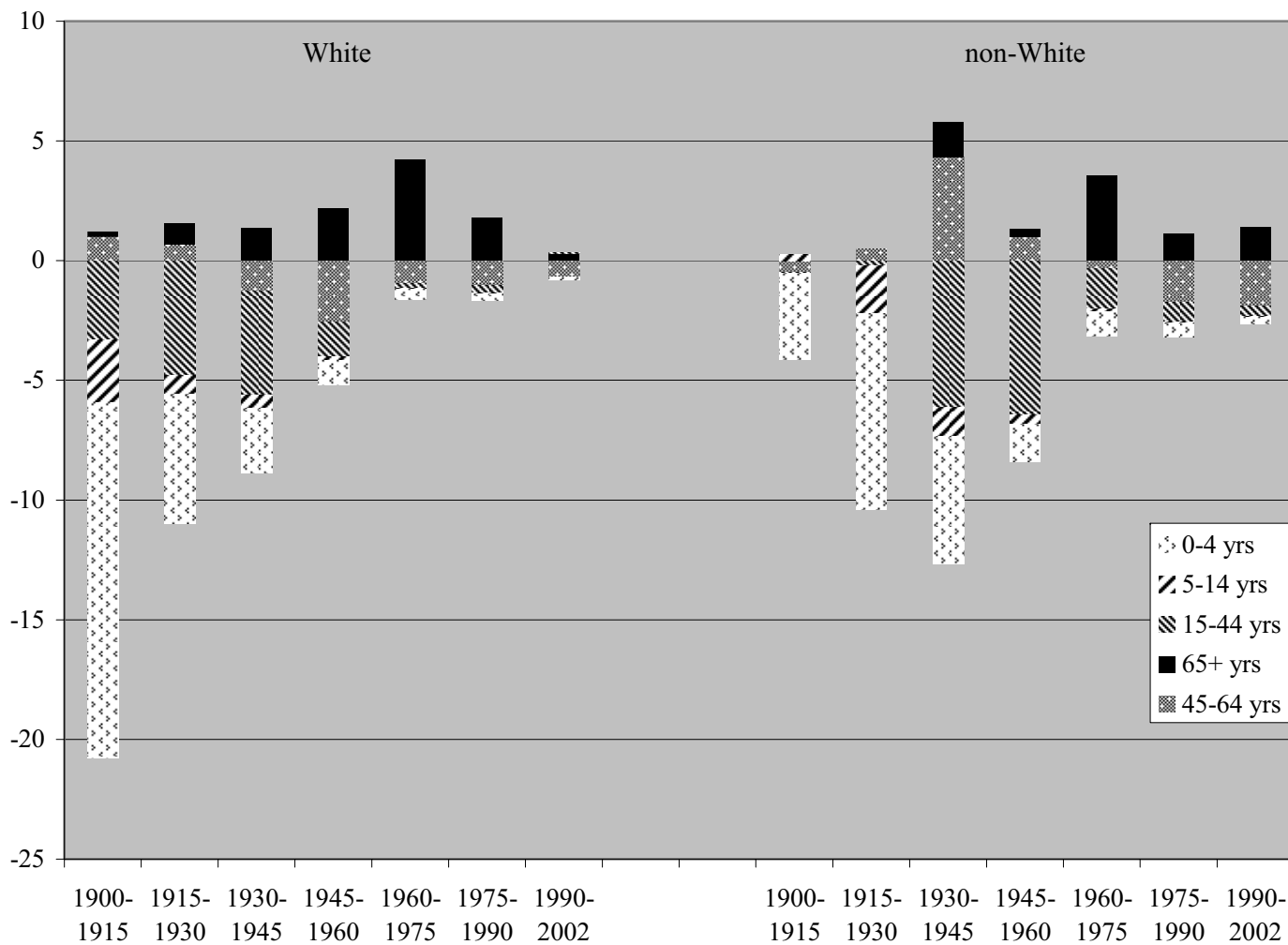
*Note: Figure excludes data for the 1918 Influenza Epidemic. The ratio of $(IQR_0 \text{ non-white}) / (IQR_0 \text{ white})$ are not depicted for the years after 1953 for women and after 1993 for men because the upper quartile for whites lies above the last age-group of mortality (85+ years).

Figure 4. Decomposition of 15-year Changes in IQR_0 into Age-specific Mortality Changes, U.S. White & non-White Men 1900-2002*



*Note: The Wilmoth and Horiuchi (1999) method allows me to decompose changes in IQR_0 when the upper quartile lies in the open-ended mortality age-group; this involves the periods after and including 1993 for white men (See Appendix A for details).

Figure 5. Decomposition of 15-year Changes in IQR_0 into Age-specific Mortality Changes, U.S. White & non-White Women 1900-2002*



*Note: The Wilmoth and Horiuchi (1999) method allows me to decompose changes in IQR_0 when the upper quartile lies in the open-ended mortality age-group; this involves the periods after and including 1953 for white women and 1971 for non-white women (See Appendix A for details).

Appendix A. Numerical Methods for Decomposing Temporal Changes in IQR_0

The calculation of the change in the IQR_0 described in Equations 1-4 is conducted using the numerical methods developed by Wilmoth and Horiuchi (See Appendix B of Wilmoth and Horiuchi 1999). These methods involve dividing the integral in Equation 5 into $N=20$ sub-intervals in which the force of mortality at the midpoint of the n^{th} sub-interval (if $n=1, 2, \dots, N$) is:

$$\mu^n(x) = \mu(x, t_1 + \frac{2n-1}{2N} \Delta t) = \mu(x, t_1) \cdot \exp \left(- \left(\frac{2n-1}{2N} \Delta t \cdot \rho(x) \right) \right). \quad (5)$$

The division of the integral into 20 sub-intervals for each 15-year period in this study allows me to estimate mortality changes when the upper quartile is in the open-ended age group.

I make a small adjustment to Wilmoth and Horiuchi's numerical methods for calculating the integral in Equation 5 in order to address the 15-year periods and the age-grouped mortality rates for mortality in the ages 1-4 years, 5-15 years, ..., and 85+ years. For example, the relative rate of mortality decline between t_1 and t_2 is calculated as

$$\rho(x) = -\frac{1}{\Delta t} \ln \left(\frac{\mu(x, t_2)}{\mu(x, t_1)} \right), \text{ where } \Delta t = 15. \quad (6)$$

Thus, similarly, the change in the IQR_0 can be obtained as follows:

$$\Delta IQR_0(t_1, t_2) \approx \sum_{n=1}^N \left\{ \frac{\Delta t}{N} \sum_{x=0}^{85+} \rho(x) \cdot \mu^n(x, t) \cdot \left[\frac{I_{75}^n}{\mu^n(x_{75})} - \frac{I_{25}^n}{\mu^n(x_{25})} \right] dx \right\}, \quad (7)$$

where dx varies according to the size of the age-group x (i.e. for age 0, $dx=1$; for age 1-4, $dx=4$; and for age 5-14, 15-24, ..., 75-84, $dx=10$).

Appendix B. Tables Detailing Decomposition of 15-Year Changes in the Interquartile Range (ΔIQR_0), Amount & % ΔIQR_0 Attributable to Mortality Change

Table 2. White U.S. Men by Age, 1900-2002

Age in years	Calculated 15-year Change in Years						
	1900-15	1915-30	1930-45	1945-60	1960-75	1975-90	1990-2002
Total ΔIQR_0 ,							
all ages	-22.0	-10.6	-6.1	-4.6	-0.5	-0.7	0.6
0	-11.3	-5.0	-2.4	-1.1	-0.6	-0.3	-0.1
1-4	-7.7	-1.9	-1.2	-0.2	-0.1	-0.1	0.0
5-14	-2.4	-0.7	-0.7	-0.3	-0.1	-0.1	0.0
15-24	-1.5	-1.2	-0.4	-0.7	0.1	-0.2	-0.1
25-34	-0.1	-2.2	-0.9	-0.9	0.0	0.1	-0.2
35-44	0.3	-0.7	-1.4	-1.0	-0.2	-0.1	-0.1
45-54	0.3	0.3	0.0	-0.9	-0.8	-1.3	-0.2
55-64	0.2	0.3	0.2	0.3	0.4	0.1	-1.6
65-74	0.0	0.5	0.9	0.1	0.7	1.2	1.1
75-84	0.0	0.0	0.0	0.0	0.0	0.0	2.0
85+	0.0	0.0	0.0	0.0	0.0	0.0	0.0

Table 3. White U.S. Women by Age, 1900-2002

Age in years	Calculated 15-year Change in Years						
	1900-15	1915-30	1930-45	1945-60	1960-75	1975-90	1990-2002
Total ΔIQR_0 ,							
all ages	-19.5	-9.4	-7.5	-3.0	2.6	0.1	-0.5
0	-8.5	-3.7	-1.7	-0.8	-0.4	-0.2	-0.1
1-4	-6.4	-1.7	-1.0	-0.2	-0.1	0.0	0.0
5-14	-2.6	-0.8	-0.6	-0.2	0.0	0.0	0.0
15-24	-2.4	-1.2	-1.2	-0.3	0.0	0.0	0.0
25-34	-1.3	-1.7	-1.5	-0.5	-0.1	-0.1	0.0
35-44	0.4	-1.9	-1.6	-0.7	-0.1	-0.2	0.1
45-54	0.5	0.3	-1.9	-1.1	-0.2	-0.5	-0.1
55-64	0.5	0.4	0.7	-1.4	-0.7	-0.5	-0.6
65-74	0.2	0.9	1.3	1.2	1.1	0.3	0.2
75-84	0.0	0.0	0.0	1.0	3.2	1.4	0.1
85+	0.0	0.0	0.0	0.0	0.0	0.0	0.0

Appendix B. (Cont'd) Decomposition of 15-Year Changes in ΔIQR_0 **Table 4. non-White U.S. Men by Age, 1900-2002**

Age in years	Calculated 15-year Change in Years						
	1900-15	1915-30	1930-45	1945-60	1960-75	1975-90	1990-2002
Total ΔIQR_0 ,							
all ages	0.5	-14.2	-8.2	-8.5	0.4	-1.9	-1.7
0	-0.1	-7.8	-4.3	-1.7	-1.4	-0.7	-0.3
1-4	0.3	-4.5	-2.3	-0.4	-0.3	-0.1	-0.1
5-14	0.5	-2.1	-1.2	-0.4	-0.1	-0.1	-0.1
15-24	0.3	0.0	-3.1	-1.7	0.2	-0.1	-0.4
25-34	-0.1	0.2	-1.3	-2.6	0.3	-0.6	-0.9
35-44	-0.6	0.2	1.2	-1.7	-0.1	-1.0	-1.3
45-54	0.2	-0.6	1.1	0.9	0.2	-1.2	-1.4
55-64	0.0	0.4	1.6	0.1	0.6	0.9	0.9
65-74	0.0	0.0	0.1	-1.0	1.0	1.1	1.6
75-84	0.0	0.0	0.0	0.0	0.0	0.0	0.3
85+	0.0	0.0	0.0	0.0	0.0	0.0	0.0

Table 5. non-White U.S. Women by Age, 1900-2002

Age in years	Calculated 15-year Change in Years						
	1900-15	1915-30	1930-45	1945-60	1960-75	1975-90	1990-2002
Total ΔIQR_0 ,							
all ages	-3.8	-9.9	-6.9	-7.0	0.4	-2.1	-1.2
0	-2.6	-4.6	-3.4	-1.2	-0.9	-0.5	-0.2
1-4	-1.0	-3.6	-1.9	-0.4	-0.2	-0.1	0.0
5-14	0.3	-2.0	-1.2	-0.4	-0.1	0.0	0.0
15-24	0.1	-0.6	-4.0	-1.7	-0.1	-0.1	-0.1
25-34	-0.1	0.2	-2.9	-2.0	-0.6	-0.1	-0.2
35-44	-0.1	0.2	0.7	-2.7	-1.0	-0.6	-0.2
45-54	-0.4	0.1	1.6	0.3	-2.0	-1.2	-0.4
55-64	-0.1	0.5	2.7	0.7	1.7	-0.5	-1.5
65-74	0.0	0.0	1.5	0.4	1.6	1.2	0.7
75-84	0.0	0.0	0.0	0.0	1.9	-0.1	0.7
85+	0.0	0.0	0.0	0.0	0.0	0.0	0.0