

# Black-White Mortality Crossover: New Evidence from Social Security Mortality Records\*

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## Abstract

The Black-White mortality crossover is well-studied demographic paradox. Black Americans experience higher age-specific mortality rates than White Americans throughout most of the life course, but this puzzlingly reverses at advanced ages. The leading explanation for the Black-White mortality crossover centers around selective mortality over the life course. Black Americans who survived higher age-specific mortality risk throughout their life course are highly selected on robustness, and have lower mortality than White Americans in late life. However, skeptics argue the Black-White mortality crossover is simply a data artifact from age misreporting or related data quality issues. We use large-scale linked administrative data ( $N = 2.3$  million) to document the Black-White mortality crossover for cohorts born in the early 20<sup>th</sup> century. We find evidence the crossover is not a data artifact and cannot be uncrossed using sociodemographic characteristics alone.

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# 1 Introduction

The Black-White mortality crossover is a long-standing demographic paradox. The crossover occurs when non-Hispanic Black Americans experience higher age-specific mortality rates than Non-Hispanic Whites Americans until very late in life. At advanced ages, the age-specific mortality rates first converge and then cross over, with Black mortality being lower than White mortality. The crossover has been repeatedly documented in the United States (Sautter et al., 2012; Dupre, Franzese and Parrado, 2006; Masters, 2012; Lynch, Brown and Harmsen, 2003; Hummer, 1996). However, there is little consensus on the explanation for the Black-White mortality crossover: critics have questioned these findings, suggesting that the apparent crossover is simply an artifact of sparse or poor-quality mortality data at the most advanced ages (Preston and Elo, 2006; Lynch, Brown and Harmsen, 2003; Preston et al., 1996; Preston, Elo and Preston, 1999). Others have theorized that the crossover is the product of selective mortality over the life course (Vaupel, Manton and Stallard, 1979; Vaupel and Yashin, 1985; Wrigley-Field, 2014, 2020).

Understanding the Black-White mortality crossover is important for several reasons. First, the mortality crossover has implications for our understanding of inequality at the most advanced ages. Is there really a narrowing of mortality conditions for Black and White Americans among the oldest old? Or is the crossover just a data artifact or a ruse of heterogeneity in susceptibility to mortality? Second, the Black-White mortality crossover is a useful empirical example for developing theoretical frameworks of mortality selection. Finally, insights gained from studying the Black-White mortality crossover can be applied to related research areas, such as mortality compression and deceleration of mortality rates at advanced ages (Lynch, Brown and Harmsen, 2003).

In this study, we use linked administrative mortality data from the CenSoc-DMF (N = 2.3 million) to investigate the Black-White mortality crossover. The unprecedented size of the CenSoc-DMF dataset, along with its rich array of covariates, allows us to empirically assess two of the main explanations for the Black-White crossover. We find a mortality crossover for the male birth cohorts of 1890–1905 at age 85 and a crossover for the male birth cohorts of 1906–1915 at age 90. Our analysis is restricted to men, as surname changes for some women

30 during marriage make linking women between the 1940 Census and the DMF mortality  
31 records infeasible. The quality of our mortality data, paired with a sensitivity analysis,  
32 allows us to rule out that our observed crossovers are simply an artifact of age misreports  
33 or exaggerations. We then stratify for observed heterogeneity to test whether the crossover  
34 can be uncrossed using sociodemographic characteristics, finding that the crossover persists  
35 across all subgroups. We conclude that unobserved heterogeneity may still be responsible, or  
36 there are indeed as-yet unknown protective factors that influence race differentials at older  
37 ages in ways that are different than at younger ages.

## 38 2 Background

### 39 2.1 Past Studies on the Black-White Mortality Crossover

40 Since its original discovery by Sibley (1930), the Black-White mortality crossover has been  
41 repeatedly documented in the United States (Manton, Poss and Wing, 1979; Berkman,  
42 Singer and Manton, 1989; Lynch, Brown and Harmsen, 2003; Dupre, Franzese and Parrado,  
43 2006; Sautter et al., 2012; Kestenbaum, 1992; Masters, 2012). The Black-White mortality  
44 crossover has also served as a motivating example for a growing body of methodological  
45 work on theoretical models of mortality selection (Vaupel and Yashin, 1985; Vaupel, Manton  
46 and Stallard, 1979; Wrigley-Field, 2014, 2020). More recently, a handful of empirical studies  
47 have investigated the contribution of covariates such as socioeconomic status or religious  
48 attendances to the Black-White mortality crossover (Dupre, Franzese and Parrado, 2006;  
49 Sautter et al., 2012; Yao and Robert, 2011; Berkman, Singer and Manton, 1989).

50 Table 1 presents several of the major empirical studies documenting the Black-White  
51 crossover. Across studies, the “age of crossover”—the age at which Black age-specific mor-  
52 tality rates first become lower than White age-specific mortality rates—occurs between the  
53 ages of 74 and 90, generally centered around 85. However, the age at crossover has been  
54 trending upwards over the course of the 20<sup>th</sup> century (Masters, 2012). In the 1960s, the  
55 crossover observed at age 75 for men and age 77 for women (Kestenbaum, 1992). In the  
56 1970s, the age at crossover was observed at ages of 78 for men and 80 for women (Masters,

57 2012). More recently, the crossover has been observed at ages 88 for men and 87 for women  
58 in U.S. lifetables from 2003 (Arias, 2006). This upward trend in the timing of the age of  
59 crossover suggests that differential cohort experiences are an important consideration for any  
60 study of the Black-White crossover.

Data Source	Age of Crossover	Covariates	Age Verification	Citation
Tennessee Vital Statistics	74			Sibley (1930)
Evans County Study	85 (f); 80 (m)			Wing et al. (1985)
Medicare Enrollment	88 (f); 86 (m)			Kestenbaum (1992)
U.S. Death Certificates	90 (f); 85 (m)		✓	Preston et al. (1996)
Medicare Enrollment	85–86			Parnell and Owens (1999)
Survey on Asset and Health Dynamics Among the Oldest Old	81			Johnson (2000)
Berkeley Mortality Database	79–87		✓	Lynch, Brown and Harmsen (2003)
Medicare Enrollment	80–85			Arias (2006)
Established Populations for Epidemiologic Studies of the Elderly	83 (f); 79 (m)	Religious Attendance		Dupre, Franzese and Parrado (2006)
Americans' Changing Lives study	80	Education, Income, Neighborhood Socioeconomic Disadvantage Index		Yao and Robert (2011)
National Health Interview Survey-Linked Mortality Files	85			Masters (2012)
Established Populations for Epidemiologic Studies of the Elderly	83 (f); 79 (m)			Sautter et al. (2012)
NCHS Multiple Cause-of-Death public-use files	87	Education, Income		Fenelon (2013)
National Longitudinal Mortality Study	85			Şahin and Heiland (2017)

Table 1: Past studies of the Black-White mortality crossover.

## 61 2.2 Explanations for the Black-White Crossover

62 There are three prominent explanations for the Black-White mortality crossover. The evi-  
63 dence to date is not yet seen as conclusive, and population scholars are increasingly seeking  
64 explanations for the Black-White mortality crossover. These competing explanations are  
65 outlined below.

### 66 2.2.1 Data Artifact

67 One explanation for the Black-White crossover is that there is no crossover at all. Rather,  
68 differential age-misreporting or exaggeration, uncounted or unmatched deaths, and other  
69 inaccuracies can lead to a spurious crossover. According to this perspective, once these data  
70 errors are accounted for, the crossover disappears or is delayed until even more advanced  
71 ages (Preston and Elo, 2006; Lynch, Brown and Harmsen, 2003; Preston et al., 1996; Preston,  
72 Elo and Preston, 1999).

73 This perspective was most clearly advanced by Preston et al. (1996), who linked death  
74 certificates to both decennial census records (1900, 1910, and 1920) and the Social Security  
75 Death Master File (DMF). This linkage exercise demonstrated that misreporting was com-  
76 mon; over 50% of Black women decedents had disagreement between the ages of death on  
77 their death certificate and their Social Security record. Upon correcting for misreporting  
78 in these death rates for Black Americans, the crossover disappeared. As further evidence  
79 of age misreporting, Preston and Elo (2006) in a follow-up study demonstrated that the  
80 age-specific mortality rates for Black Americans above 85 were lower than the age-specific  
81 mortality rates in the lowest-mortality countries.

### 82 2.2.2 Age-As-A-Leveler

83 The “naive” theoretical explanation for the Black-White mortality crossover is that for the  
84 oldest-old, mortality conditions converge for Black and White Americans. According to this  
85 *age-as-a-leveler* hypothesis, older adults are increasingly separated from the unequal social  
86 institutions that contribute to racial health disparities, such as the education system, the  
87 labor market and the criminal justice system. The departure from these stressors of daily

88 living may cause mortality rates to converge in later life (Kim and Miech, 2009). Further,  
89 increased availability of a social safety net in later life, including Medicare and Social Security,  
90 and stronger kin and support networks, could cause age-specific mortality rates to converge  
91 in the oldest ages.

92 In this sense, old age acts as a “leveler” and causes a convergence in age-specific mortality  
93 rates; real racial disadvantage attenuates at the most advanced age. However, it is unclear  
94 why such attenuation of disadvantage at the most advanced ages would cause a crossover,  
95 rather than simply a convergence. Further, this hypothesis is at odds with a large body of  
96 research documenting racial inequality in the U.S. (Bryan L. Sykes and Michelle Maroto,  
97 2016; Alexander, 2010; Riddle and Sinclair, 2019; Perry and Morris, 2014).

### 98 **2.2.3 Heterogeneity in Frailty**

99 The most famous explanation for the mortality crossover comes from theoretical models of  
100 mortality selection. Mortality selection models begin with the premise that people vary  
101 systematically in mortality risk. In this frailty modeling tradition, as a cohort ages, it  
102 becomes increasingly composed of robust individuals. This mortality selection can occur  
103 unequally across population subgroups, and has been hypothesized to explain mortality  
104 crossovers, mortality deceleration, and mortality compression (Lynch, Brown and Harmsen,  
105 2003; Wrigley-Field, 2014).

106 In the case of the Black-White crossover, Black Americans who faced higher mortality  
107 risks in early and midlife will be composed of a greater proportion of robust individuals  
108 in later life, resulting in their age-specific mortality rates becoming lower than those of  
109 White Americans, who faced lower mortality risks earlier in their life course. In other  
110 words, the Black Americans who survive to the most advanced ages are more highly selected  
111 for robustness than their White counterparts, and will have lower mortality at advanced  
112 ages (Wrigley-Field, 2020; Vaupel and Yashin, 1985).

113 Skeptics of this heterogeneity in frailty explanation point out that poor health conditions  
114 in early life can “scar” survivors, leading to higher mortality in later life (Preston and Elo,  
115 2006). The limited number of empirical investigations have suggested that the dominant  
116 direction of mortality conditions at different points in the life course is positively: higher

117 mortality risk in early life is associated with higher mortality later in the life course (Finch  
118 and Crimmins, 2004; Janssen et al., 2004; Preston, 1970).

119 This study has two specific aims. First, we establish the mortality crossover as real,  
120 not a data artifact. Second, we provide empirical evidence that the crossover cannot be  
121 uncrossed using sociodemographic characteristics alone. The remainder of the paper proceeds  
122 as follows. In the Section 3, we describe the complete count census data and mortality records  
123 used in our analysis. We then describe our methods for mortality estimation in the absence  
124 of denominators in Section 4. In Section 5 and Section 6, we present and interpret our  
125 findings and discuss their implications for our understanding of mortality selection.

## 126 3 Data

127 This study uses complete count 1940 Census data, mortality records from the Social Security  
128 Death Master File (DMF), and record linkage techniques to construct a large-scale dataset  
129 with rich covariates and mortality outcomes. This dataset, termed the CenSoc-DMF (Gold-  
130 stein et al., 2021), links the complete count 1940 Census (Ruggles et al., 2020) to the DMF.  
131 The DMF is a collection of over 83 million death records reported to the Social Security  
132 Administration, with nearly complete mortality coverage between 1975–2005 (Alexander,  
133 2018; Hill, 2001). However, the DMF does not contain any socioeconomic or demographic  
134 variables. To obtain individual-level covariates, we link the DMF mortality records to 1940  
135 Census records. The resulting matched file includes only men, as surname changes due to  
136 marriage for some women make the systematic linkage of women infeasible.

137 We link individual records in the complete count 1940 Census to the DMF using first  
138 name, last name, and year of birth using the ABE exact match record linkage algorithm  
139 (Abramitzky, Boustan and Eriksson, 2012, 2014; Abramitzky and Boustan, 2017; Abramitzky  
140 et al., 2021). To reduce false matches, we restrict to matches where names are unique  
141 within and across datasets for a  $\pm 2$  year window. This approach prioritizes minimizing the  
142 number of false matches over maximizing the overall match rate; this minimizes the amount  
143 of systematic bias introduced by false matches (Ruggles, Fitch and Roberts, 2018).



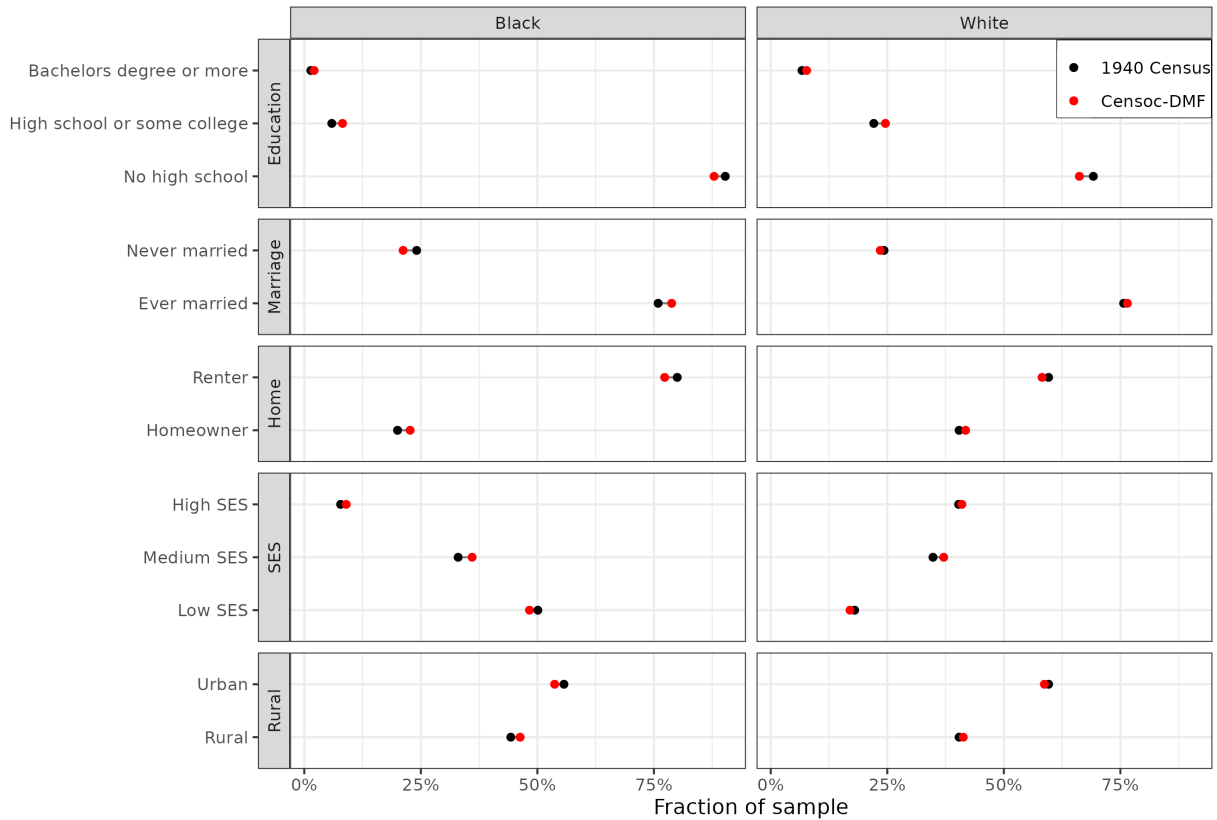


Figure 1: Each facet shows the composition of the CenSoc-DMF (Red) and the complete count 1940 Census (black) for a given covariate for Black and White matches. The matched sample has slightly higher socioeconomic status than the general population.

### 144 3.1 Representativeness of Matches

145 Our mortality-adjusted match rate is approximately 20% (Breen and Osborne, 2022). To  
 146 demonstrate that our matched sample is representative of the general population within  
 147 racial groups, we compare the composition of our matched sample to the general population  
 148 in the 1940 Census. Figure 1 shows our matched sample is broadly representative of the  
 149 general population within racial groups, except with slightly higher socioeconomic status.

### 150 3.2 Reliability of DMF

151 A key consideration for our study is the reliability of the DMF mortality records. The DMF  
 152 is extracted from the Social Security Numident, and contains over 75 million death records.  
 153 The death coverage between 1975–2005 is nearly complete, containing approximately 95%

154 coverage for deaths occurring after the age of 65 (Hill, 2001; Alexander, 2018). Death  
155 coverage rates drop after after 2005, and the DMF has substantial coverage gaps beginning  
156 in 2011 (Maynard, 2019). Our analysis is restricted to deaths occurring in our mortality  
157 observation window of 1975–2005.

158 The DMF does not explicitly include information on age of death. Rather, the DMF  
159 contains information on date of birth and date of death from which age of death can be  
160 imputed (Preston et al., 1996). Therefore, to assess the reliability of the imputed age of  
161 death, we need to investigate the quality of the reported date of birth and date of death.

162 Dates of death are directly reported to the Social Security Administration from a funeral  
163 director or a family member. These reports are generally made directly following a death,  
164 minimizing the likelihood of misreporting. The date of death in the DMF almost always  
165 exactly matches the date of death in the corresponding death certificates (Hill, Preston and  
166 Rosenwaike, 2000).

167 Information on date of birth is submitted personally by the decedent in conjunction with  
168 a benefit claim. The Social Security Administration closely tracks age to determine eligibility  
169 for benefits. Age verification is a required condition for entitlement to benefits, and stringent  
170 tests were put in place in 1965. The focal cohorts of this study would have become eligible  
171 for Social Security benefits after these age verification procedures were put in place.

172 To empirically assess the reliability of the date of birth information in the DMF, we look  
173 at heaping on year of birth. Heaping, a common indicator of data quality, is the systematic  
174 misstatement of ages or dates to round or terminal ages (e.g., end in “0” or “5.”) We find  
175 minimal date heaping on year of birth, as shown in Figure 2. However, there is slightly  
176 higher heaping for Black Americans than White Americans. To investigate whether this  
177 age heaping has any affect on our observed crossover, we conduct a sensitivity analysis by  
178 dropping years of birth that end in terminal ages and re-estimating the observed crossover.

179 The nature of our sample provides additional reassurance that the reported age of birth  
180 is accurate. For an individual to be successfully matched and included in our sample, their  
181 reported age in the 1940 Census must correspond to  $\pm 2$  years of their year of birth reported  
182 in the DMF. Therefore, mortality records where the year of birth is misreported by over two  
183 years will be excluded from our sample. This is similar to the validation approach taken

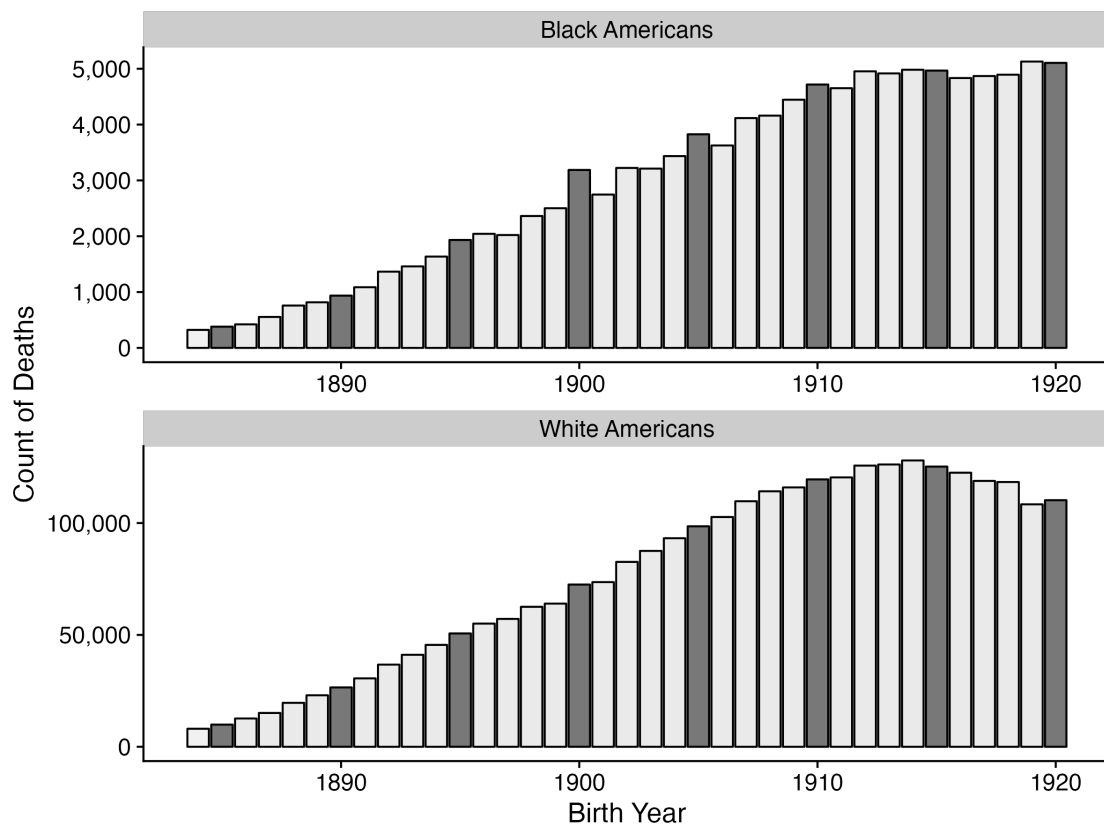


Figure 2: Highlighted years (dark grey) show very slight amounts of age-heaping on the terminal digits “0” or “5,” suggesting the DMF has minimal misreporting of year of birth.

184 by Hill, Preston and Rosenwaike (2000); Preston et al. (1996), and gives an additional level  
185 of reassurance that the reported birth year is accurate.

## 186 4 Methods

### 187 4.1 Estimating Mortality Rates

188 The CenSoc-DMF dataset only includes deaths for the left and right (“doubly”) truncated  
189 window of 1975 to 2005. Further, the CenSoc-DMF does not include any measure of sur-  
190 vivorship, as we have no way of determining whether an individual observed in the 1940  
191 Census died outside our observation window window or was not successfully matched to  
192 their death record. The absence of any measure of a denominator precludes conventional  
193 occurrence-exposure methods for estimating mortality rates (Alexander, 2018).

194 To overcome this, we use two different methods to estimate mortality rates in the absence  
195 of denominators. First, for the earlier cohorts of 1890–1905, we use the reverse survival  
196 method to estimate mortality rates. This approach assumes that all persons in the cohort  
197 have died by the end of our mortality observation window in 2005. Specifically, we estimate  
198 the total number of survivors at a given age by summing up all the deaths occurring above  
199 that age, and then estimating the age-specific mortality rates using the age-specific ratios of  
200 deaths to survivors. This method is only appropriate for the cohorts born before 1905, for  
201 which only a few survivors to age 100 will die after 2005.

202 Second, for the later-born cohorts of 1906-1915, those that we cannot assume are extinct  
203 by 2005, we assume the distribution of deaths within a cohort follows a Gompertz distri-  
204 bution and use maximum likelihood estimation methods to estimate the parameters of this  
205 distribution (Goldstein et al., 2023; Gompertz, 1825). Specifically, the hazard of dying at  
206 age  $x$  is:

$$h_0(x) = ae^{bx} \tag{1}$$

207 where  $a$  is a background level of mortality at age  $x$ ,  $b$  is the rate of mortality increase with  
208 age, and  $h(x)$  describes the hazard schedule. This approach allows us to estimate age-specific  
209 mortality rates for both ages where we did and did not observe deaths.

## 210 4.2 Stratifying on Observed Dimensions of Heterogeneity

211 The classical mortality selection model used to explain the crossover is unidimensional. That  
212 is, all heterogeneity in susceptibility to mortality is captured in a single parameter (“frailty”).  
213 A growing body of empirical research on the Black-White mortality crossover has used  
214 individual-level covariates to study the observed dimensions of heterogeneity that constitute  
215 frailty. Borrowing logic from unidimensional mortality selection model, these studies inves-  
216 tigated how controlling for some piece of frailty changes the age of crossover (Sautter et al.,  
217 2012; Dupre, Franzese and Parrado, 2006). Yet theoretical advances have demonstrated  
218 that the unidimensional mortality selection model is not an appropriate starting point for  
219 empirical work. When there is both observed and unobserved heterogeneity, stratifying on  
220 observed heterogeneity can cause the age at crossover to either move up or down (Wrigley-  
221 Field, 2020). In other words, the age at crossover will always change when some factor  
222 related to both race and mortality is controlled for.

223 One important exception occurs when an observed dimension of heterogeneity constitutes  
224 a large portion of the overall heterogeneity. In this setting, if the crossover is caused by  
225 heterogeneity in frailty, stratifying on a covariate that represents over 50% of total frailty  
226 will uncross the crossover (Wrigley-Field, 2020). For empirical researchers, this implies that  
227 combining many covariates into a single risk measure is a promising strategy for examining  
228 the role of observed heterogeneity in explaining the crossover.

229 To investigate the role that observed heterogeneity plays on the mortality crossover, we  
230 use socioeconomic covariates available in the 1940 Census. First, we investigate the crossover  
231 in six distinct subgroups: individuals with high education (more than 8 years), individuals  
232 with low education (less than 8 years), individuals with high income (above the median  
233 income), individuals with low income (below the median income), homeowners, and renters.  
234 On each subgroup, we estimate age-specific mortality rates using the reverse survival method.  
235 We then combine these covariates into a single risk score and investigate the crossover in  
236 subgroups defined by risk. Together, these analyses allows us to investigate whether the  
237 crossover still persists when we stratify on major pieces of frailty.

## 238 5 Results

239 We first analyze the Black-White mortality crossover using both the reverse survival method  
240 and our parametric Gompertz approach. Next, we present results on observed mortality  
241 selection. Finally, we examine whether our observed heterogeneity can help explain the  
242 Black-White mortality crossover.

### 243 5.1 Black-White Mortality Crossover

244 We first examine the Black-White crossover for the pooled birth cohorts of 1890-1905. [Fig-](#)  
245 [ure 3a](#) shows a clear mortality crossover at age 86, consistent with past findings. For this  
246 analysis, we estimated age-specific mortality rates using the reverse survival method. Be-  
247 cause our mortality data showed very slight heaping on year of birth, as a sensitivity analysis,  
248 we recalculate our age-specific mortality rates excluding birth years with potential age heap-  
249 ing: 1890, 1895, 1900, and 1905. [Figure 3b](#) shows that the crossover persists, suggesting that  
250 low quality mortality data is not responsible for the crossover.

251 For the cohorts of 1905-1915, which are not extinct by the end of our mortality observation  
252 window in 2005, we fit a parametric Gompertz model to calculate age-specific mortality  
253 rates ([Goldstein et al., 2023](#)). We perform maximum likelihood estimation for the Black and  
254 White groups separately. [Figure 4](#) shows a mortality crossover at age 90, slightly higher than  
255 our observed age at crossover for the cohorts of 1890–1905. A higher age at crossover for  
256 later birth cohorts is consistent with past studies ([Masters, 2012](#)).

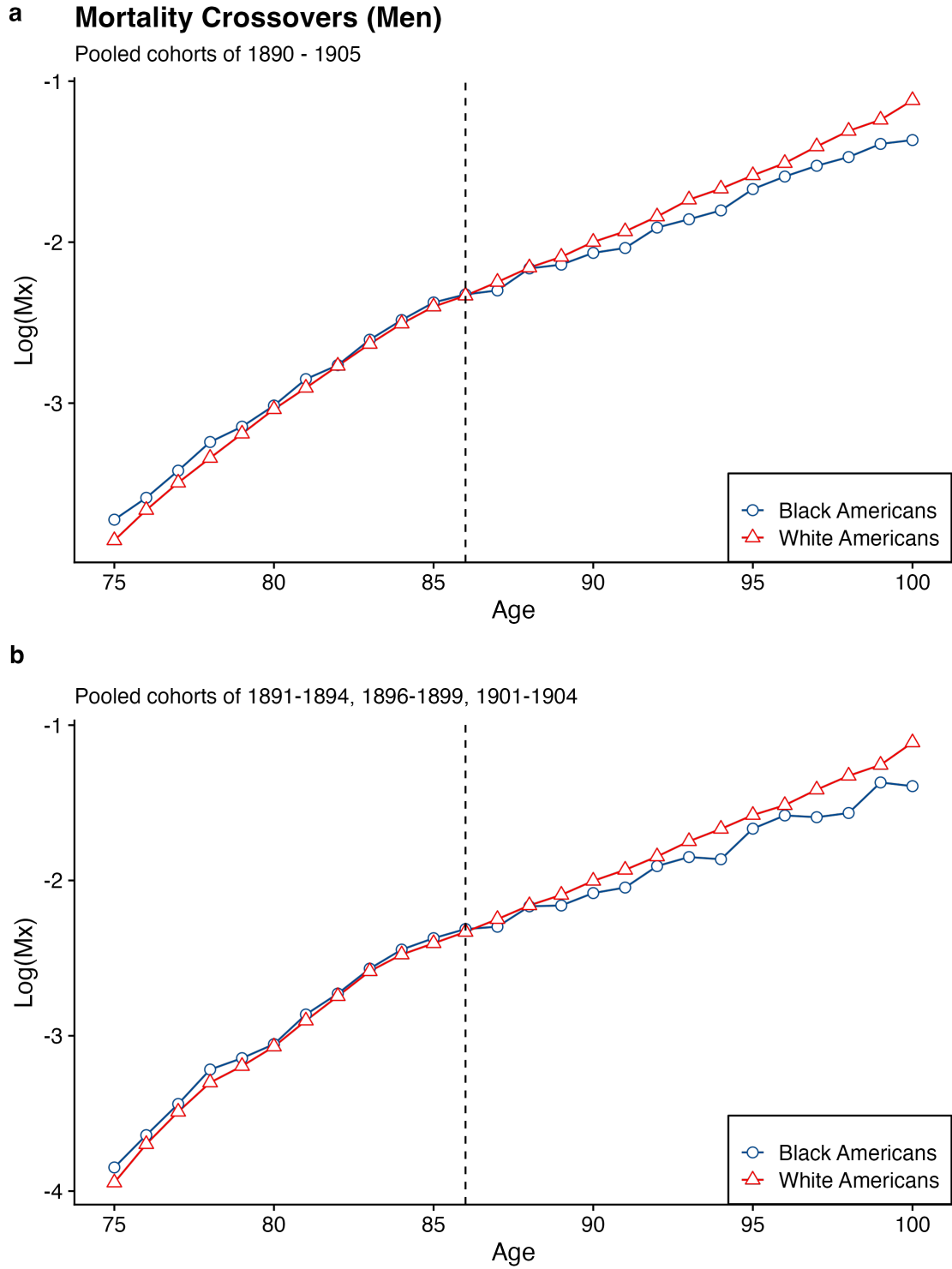


Figure 3: Panel (a) shows the Black-White mortality crossover for the cohorts of 1890-1905. Panel (b) shows the mortality crossover dropping the cohorts of 1890, 1895, 1900, and 1905, where we observed slight but detectable age heaping. The mortality rates were estimated using the reverse survival method.

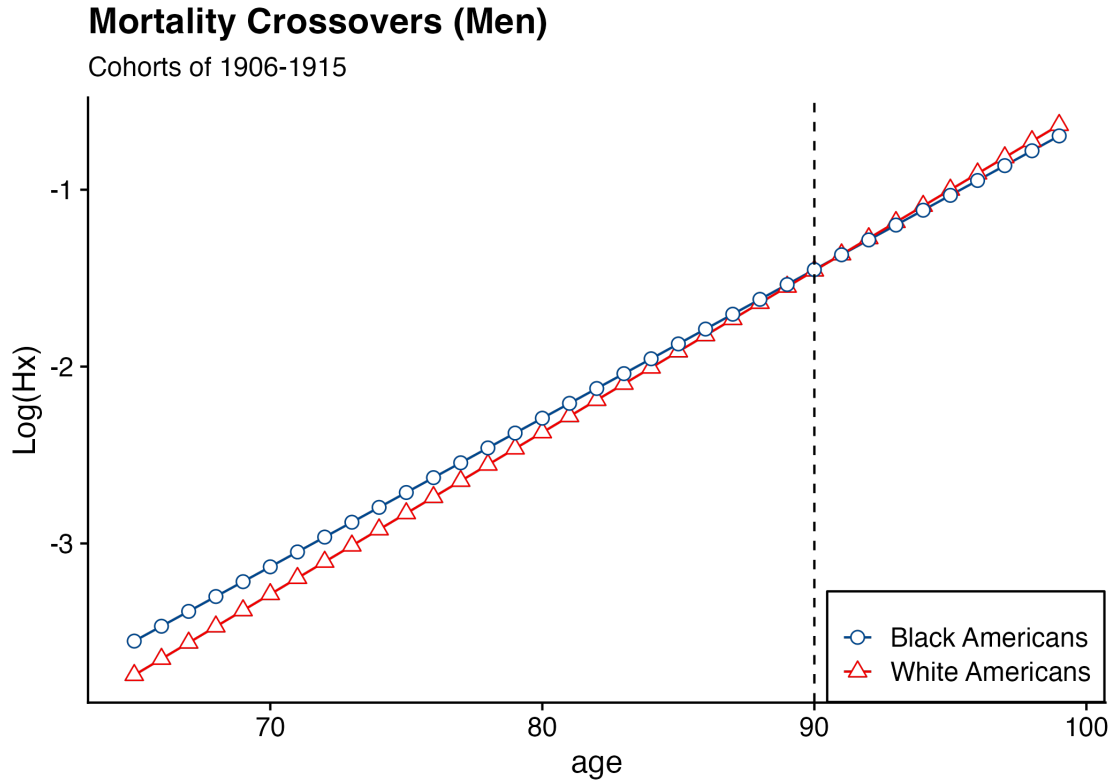


Figure 4: Black White mortality crossovers for cohorts of 1905-1915.

## 257 5.2 Observed Mortality Selection

258 To investigate mortality selection, we track how the characteristics of survivors change as  
 259 a cohort ages and members of the cohort die off. We focus on how the composition of the  
 260 cohorts of 1909–1911 changes with respect to employment, educational attainment, socioe-  
 261 conomic status score, wage and salary income, homeownership status, and residing in the  
 262 south. We interpret an increase in a dimension of socioeconomic status as a cohort ages to  
 263 be evidence of selective mortality: more frail individuals are dying off at earlier ages.

264 As shown in Figure 5, we do observe selective mortality, which is more pronounced for  
 265 White Americans than Black Americans. For instance, members of the cohort of 1909–1911  
 266 who survived to age 65 have approximately 10 years of education, while members of the  
 267 cohort who survived to age 90 have approximately 10.6 years of education. The difference is  
 268 more slight for the cohort of Black Americans: survivors at age 65 had 6.6 years of education,  
 269 and survivors at age 90 had 6.7 years of education. Across all of the covariates tested, we



270 find that the the surviving members of a cohort becoming more increasingly advantaged as  
 271 the cohort ages.

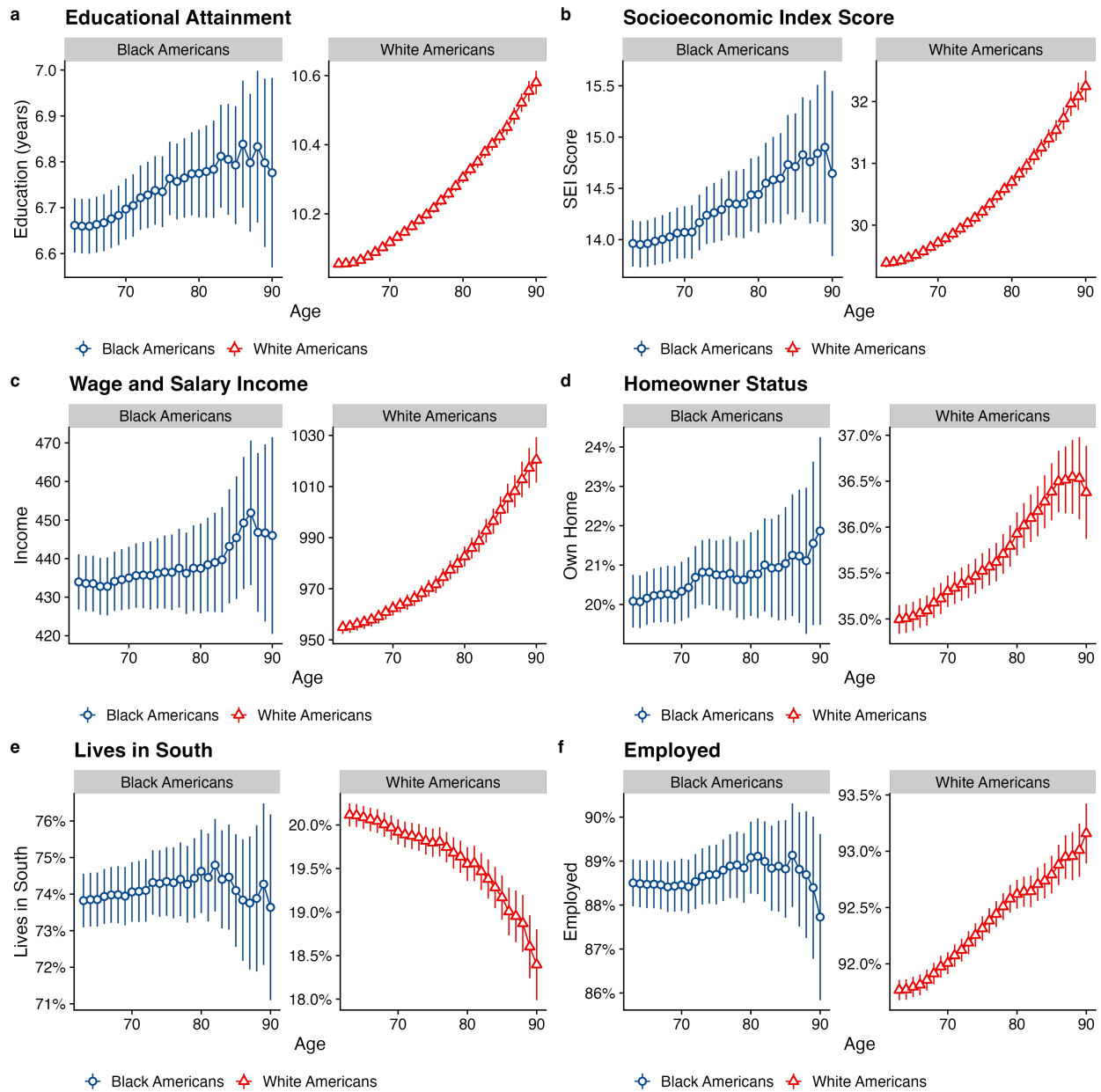


Figure 5: Changing composition of the survivors. We see only modest evidence of selection. Error bands show 95% uncertainty intervals.

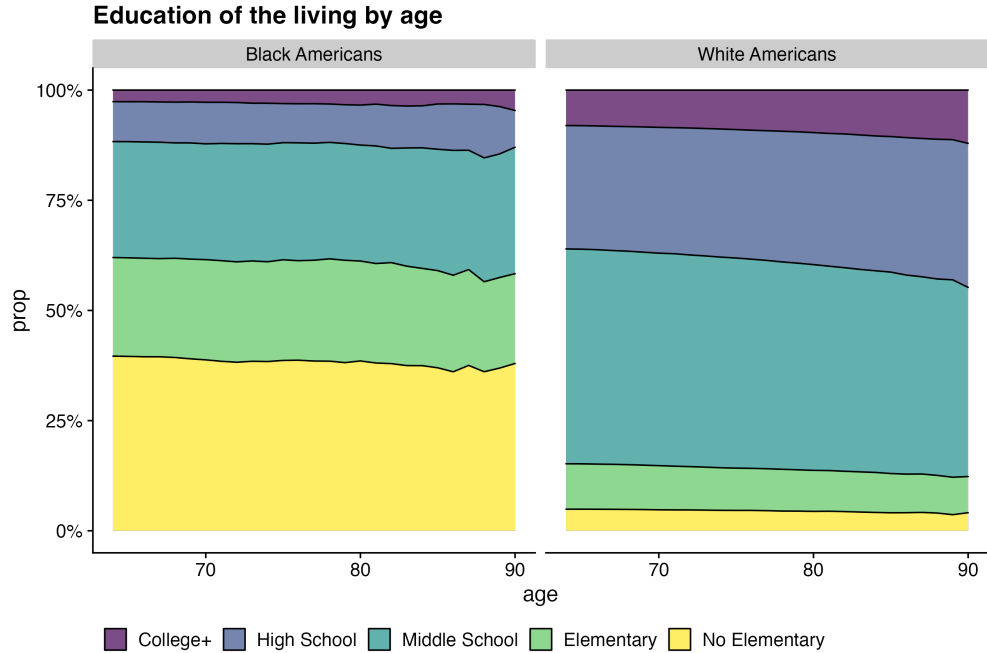


Figure 6: Changing educational composition of the survivors.

272 **5.3 The Surprising Non-Effect of Observed Heterogeneity on the**  
 273 **Mortality Crossover**

274 Next, we investigate the effect of observed heterogeneity on the mortality crossover. We split  
 275 our 1890-1905 birth cohort sample into different population subgroups defined by education,  
 276 homeownership, and wage and salary income. [Figure 7](#) shows the result of this analysis:  
 277 the crossover persists across all subgroups.

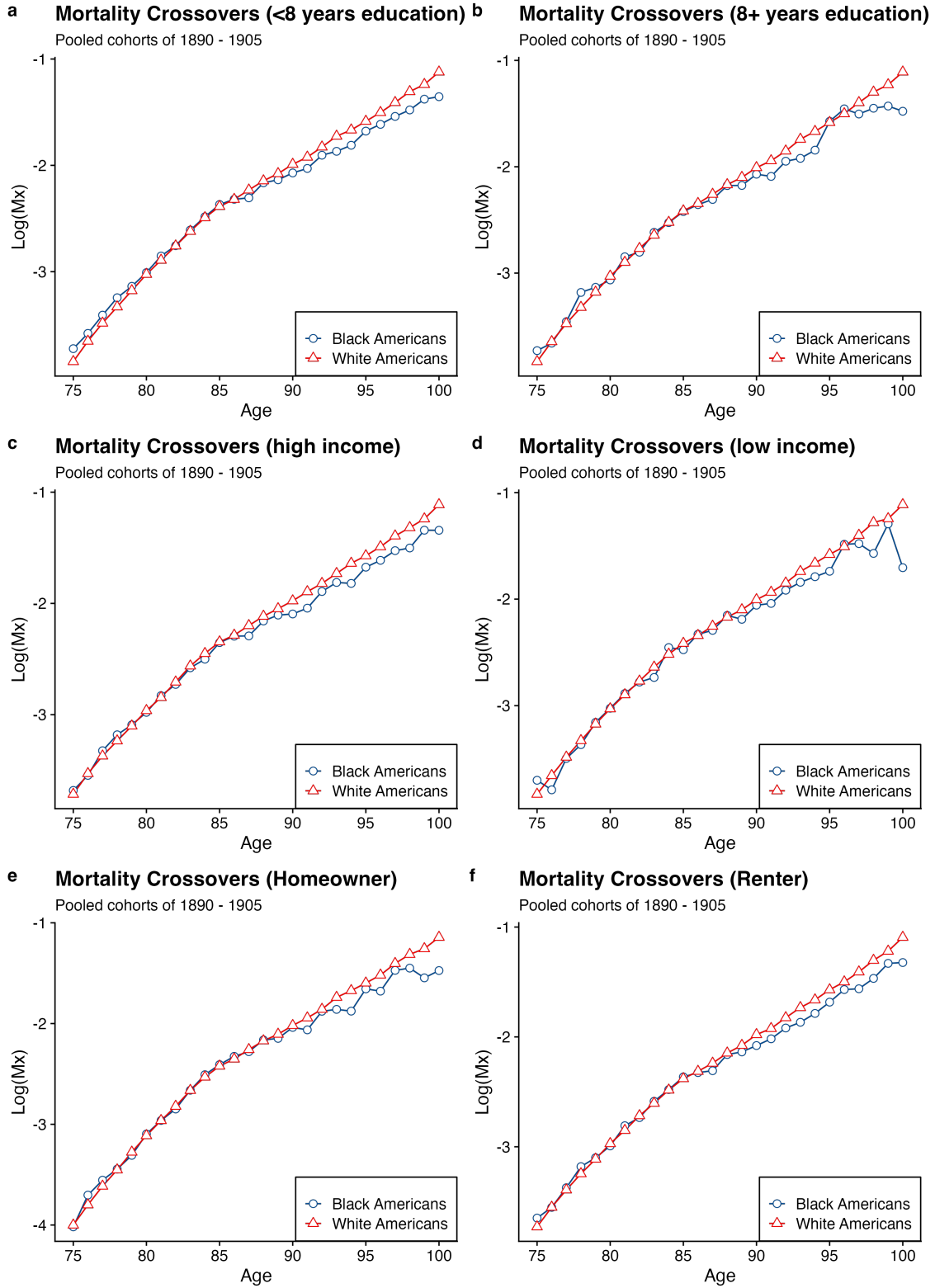


Figure 7: Black White mortality crossover for different subgroups defined by socioeconomic status.

278 Next, we follow the advice outlined in [Wrigley-Field \(2020\)](#) and investigate the mortality  
279 crossover stratified by risk scores. To construct the risk score, we aggregate together the  
280 following covariates into a single score: education, wage and salary income, socioeconomic  
281 index, marital status, employment, living in the South, and owning a home. The motivation  
282 for constructing this risk score is to capture as much of the heterogeneity in frailty as possible  
283 in one score. To estimate the risk score, we fit linear regressions of the form:

$$\text{death\_age}_i = \text{educ}_i + \text{income}_i + \text{homeowner}_i + \text{marital\_status}_i + \text{southern}_i + \epsilon_i. \quad (2)$$

284 [Figure 8a](#) presents the age-specific mortality rates for Black and White men within risk  
285 group. We see the crossover persists in all different risk groups. [Figure 8b](#) plots the difference  
286 in log hazards, again finding a clear crossover for all three groups.

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<sup>0</sup>We first fit this model on our analytic sample, those born between 1890-1905. To avoid potentially over-fitting, we also fit the model on the out-of-sample cohort of 1906. Results from both models provided highly comparable predictions of risk score.

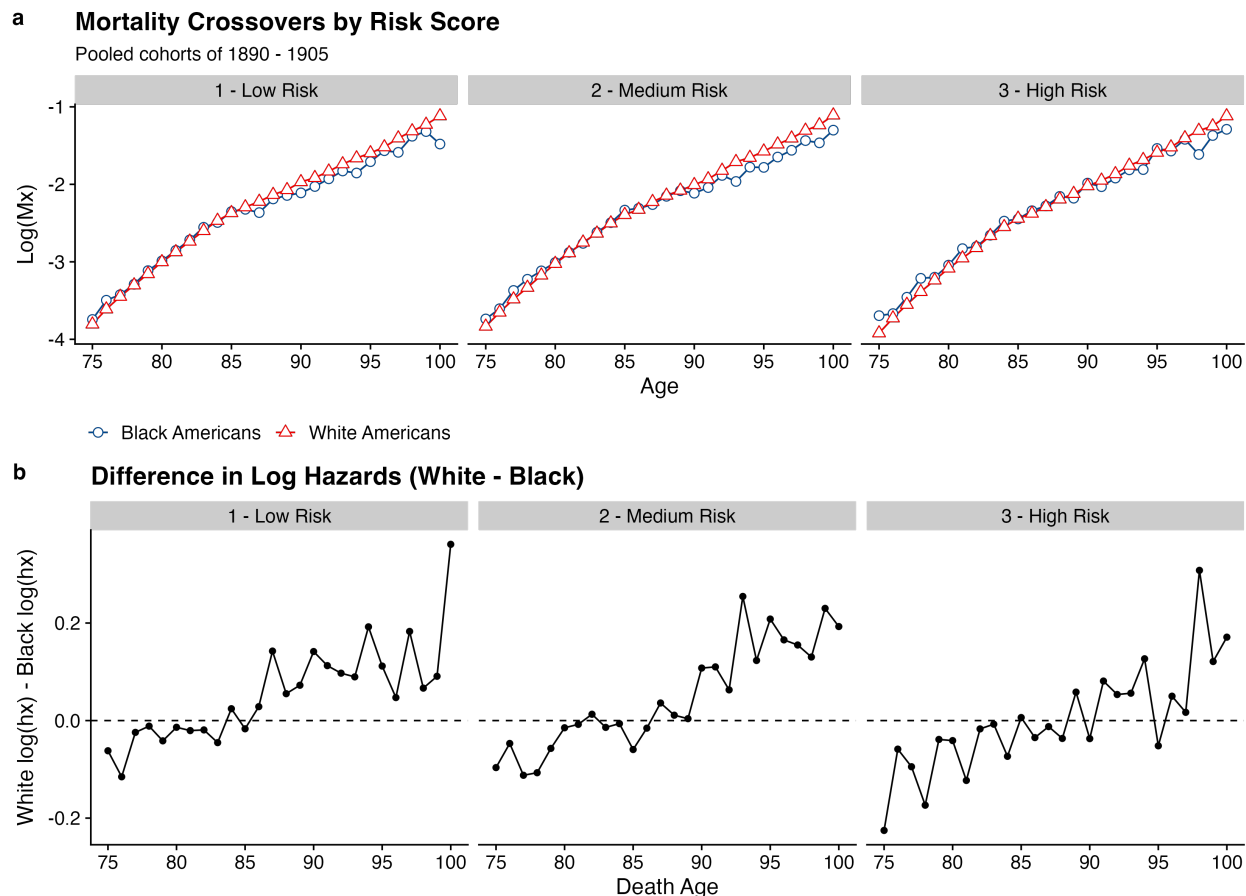


Figure 8: Black White mortality crossover by risk score.

## 6 Discussion

This study uses mortality records from the Death Master File (DMF) linked to the 1940 Census to investigate the Black-White mortality crossover. We find a clear mortality crossover at age 85 for men in the birth cohorts of 1890–1905 using reverse survival methods to estimate age-specific mortality rates. Using a Gompertz parametric maximum likelihood approach, we find a mortality crossover at age 90 for the birth cohorts of 1906–1915. Given the reliability of the DMF mortality data, we interpret this as evidence that the Black-White mortality crossover is not simply an artifact of sparse data or age misreporting: the crossover persists even when we restrict the sample to the highest-quality mortality data.

Using individual-level characteristics from the 1940 Census, we investigate observable mortality selection. We find clear evidence of selective mortality: as a cohorts ages, the

298 survivors have increasingly higher educational attainment, rates of homeownership, rates of  
299 employment (in 1940), and wage and salary income. However, the observable selection is  
300 relatively modest, and is more pronounced for White Americans than Black Americans. The  
301 lack of observable mortality selection for Black Americans is perhaps attributable to the  
302 weaker correlation between covariates, such as education or income, and mortality risk for  
303 Black Americans (Card and Krueger, 1992).

304 Our investigation of the Black-White mortality crossover for subgroups defined by so-  
305 cioeconomic characteristics indicated a clear crossover in every subgroup. Additionally, the  
306 crossover persisted when we stratified on a risk score that aggregated many mortality covari-  
307 ates. This suggests that stratifying on observed socioeconomic dimensions of heterogeneity  
308 does not explain the crossover. There are two potential explanations for this finding. First,  
309 it is possible that sociodemographic characteristics alone simply do not capture enough of  
310 the heterogeneity in frailty to really uncross the crossover. Second, it is possible that the  
311 crossover is not driven by heterogeneity in frailty at all; rather, there is actually some true  
312 narrowing of inequality at the most advanced ages.

313 Taken together, our results suggest that the mortality crossover is real and not an artifact  
314 of measurement or data errors. Our data allows us to study the mortality experience of real  
315 cohorts, not the synthetic period measures commonly used to study the crossover. However,  
316 our study cannot make definitive about the theoretical explanations for the crossover. While  
317 our study found that stratifying on observed dimensions of frailty such as educational at-  
318 tainment or homeownership does not explain the crossover, it is possible that we are simply  
319 not capturing enough of the heterogeneity in frailty to uncross the crossover.

320 There are several limitations and avenues for future research. First, we only observe  
321 mortality window of 1975–2005, so our analyses are restricted to birth cohorts that would  
322 be experiencing a crossover in our mortality observation window. Second, it is possible  
323 that the sociodemographic characteristics we observe only constitute a very small piece  
324 of frailty and therefore have limited utility for explaining the crossover. Future research  
325 could test whether covariates that capture more of the heterogeneity in frailty, such as  
326 biomarkers, anthropometric measures (weight, height), and direct measurement of subjective  
327 and objective health on the crossover. It is important to acknowledge that the present study

328 benefits from an exceptionally large sample size, making it potentially challenging for other  
329 studies to achieve comparable levels of precision. Third, while we find little evidence of age  
330 misstatement or exaggeration, it is possible there remain undetected age misreports in the  
331 DMF. Finally, this analysis is limited in scope to men. Broadening this study to include  
332 women is necessary to make complete claims about health and longevity disparities in the  
333 most advanced ages.

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