RACE AND OLDER AGE-MORTALITY: EVIDENCE FROM UNION ARMY VETERANS

by

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Abstract

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Black Union Army veterans faced an odds of dying at older ages that was 1.3 times higher than that of white Union Army veterans and black men did not achieve the older age mortality rates of white Union Army veterans until the 1970s. Deaths from infectious disease accounted for half of the black-white mortality gap. Blacks faced an urban mortality penalty that accounted for roughly 15 percent of the racial mortality differential. Excess mortality was concentrated in the 1841-50 cohort and was not evident in the South. Later black cohorts may have suffered more from a deterioration in living condition in the North in the post-bellum period.

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1 Introduction

The first cohort of African-Americans to reach age 65 in the twentieth century faced extremely high older age mortality rates. In the death registration states in 1900 black men age 55 to 64 and 65 to 74 had mortality rates that were one and a half times those of white men. Black men's mortality rates did not consistently fall below white men's mortality rates in 1900 until 1975 (see various issues of *Vital Statistics of the United States*).[1]

Why were black older age mortality rates at the beginning of the twentieth century so high relative to those of whites? This paper uses the records of the Union Army to document the older age mortality experience of the first white and black cohorts who reached middle and older ages in the twentieth century and to examine the role of infectious disease in explaining black-white mortality differentials. In the United States, African-Americans experienced a later epidemiological transition than whites. The start of the sustained decline in black child mortality rates lagged the decline in white child mortality rates by 20 to 30 years (Haines 2003).

The Union Army records enable me to examine the mortality experience of blacks who lived both in large cities (where the epidemiological transition first began) and in rural areas and both in the South and in the North. With few exceptions (e.g. Preston and Haines 1991), most studies of black and white mortality differentials have focused on larger cities or northern states because smaller cities and rural areas and southern states often did not collect mortality statistics. The data also enable me to examine the mortality experience of different cohorts. Cohorts differed in their early life experiences – compared to the 1820s the 1830s and even more so the 1840s were a time of disease epidemics in both the north and the south and of poor harvests (Kiple and King 1981: 127, 147-157). Cohorts also differed in their later life disease exposure. Conditions under freedom may have worsened. Contemporary observers reported a high incidence of tuberculosis,

[1]Because the death registration states were more urban than the rest of the country, mortality rates in 1900 may be over-estimated. However, this finding is also observed in other data such as the Union Army records used in this research.

pneumonia, typhoid, syphilis, malaria, pellagra, rickets, whooping cough, and hookworm among blacks in the postbellum era (Kiple and King 1981: 189: Holmes 1937: 39).

The implications of this paper extend beyond the Civil War cohort because the health of one generation can have a long reach. Lower health leads to lower productivity and output and therefore reduces investments in children's health and education. Poor child health in turn leads to poor health at older ages, as seen in studies of the long-term effects of place of birth, disease exposure, and season of birth on older age mortality (Preston, Hill, and Drevenstedt 1998; Almond 2003; Doblhammer and Vaupel 2001; Costa and Lahey 2005). The poor health of the black Civil War cohort may therefore have been transmitted to later generations.

2 Data

By the end of the Civil War, 186,017 black men had entered the US Colored Troops. Twenty-six percent of them came from the free states, 22 percent from the border states and the District of Columbia, and 50 percent from the Confederacy. Three-quarters of all Colored Troops were former slaves. The fraction of age-eligible black men who served was 78 percent in the free northern states, 34 percent in the border states, and 11 percent in the Confederacy (Metzer 1981).[2] In the Confederacy there were active recruitment efforts whenever an area was liberated by the Union Army and thus states that were occupied before the end of the war provided the most men.[3] In the random sample of black soldiers used in this paper, 91 percent were volunteers (a fraction similar to that for whites), 7 percent were substitutes, and 2 percent were draftees.

The black sample used in this paper is based upon the military service and pension records of

[2]This was a much higher fraction than that serving in the North where only 41 percent of all age eligible cohorts served (with much higher rates among those younger than 25 in 1861) (calculated from Fogel 1993).

[3]It was not just slaves who were mistreated who joined the Union Army. Among the first 10 slave narratives that recounted own or a family member's voluntary service in the Union Army (out of the first 12 narratives there are two recounting capture in the Union Army while a servant in the Confederate Army), 6 mentioned that the master was good, 3 did not mention the master, and 1 recounted escape to the Union Army after severe mistreatment. See the database, *American Slavery: A Composite Autobiography*.

5,673 black Union Army soldiers in 51 randomly chosen infantry companies.[4] The military service records provide information on events such as wartime deaths, injury and illness, antebellum occupation, age at enlistment, and place of enlistment. Place of enlistment was not as good a proxy for place of residence for southern enlistees as it was for northern enlistees because there were fewer recruiting centers in the South and these would draw from the neighboring countryside for enlistees. The pension records document postbellum mortality, residence, occupation, and writing ability of the enlistees.

Comparisons are drawn with a random sample of white Union Army soldiers, which is based upon the military service of almost 35,000 men and is described extensively elsewhere (e.g. Costa 2000; Costa 2002; Costa 2003).[5] Two different subsets of the Union Army samples are used. Both restrict the data to men on the pension rolls because these are the only source of information on subsequent mortality. One subset consists of everyone alive and on the pension rolls at age 60. This is used to examine cohort effects and provides data on 10,872 white native-born men and 894 black native-born men. The second subset is of everyone alive and on the pension rolls in 1900 and allows me to control for socioeconomic status and for residence circa 1900. This subset provides data on 9605 native-born white veterans and 954 native-born black veterans. The sample representativeness of the black Union Army data is discussed in detail in the Data Appendix.[6]

Entry on the pension rolls was relatively non-discriminatory by the standards of the time. Both black and white veterans were eligible for a pension for war-related injuries. Beginning in 1890 pensions were paid for any disability, regardless of its relation to the war, and the Pension Bureau began to consider age 65 or older as a disability in its own right unless men were "unusually vigorous." By 1907 old age was officially recognized by Congress as a disability. Among all men

[4]The records of all men in the 51 companies were collected. The sample represents roughly 2.7 percent of all blacks serving. The data were collected by a team of researchers led by Robert Fogel. Once the data are completed and cleaned, they will be available from the Center for Population Economics at the University of Chicago, http://www.cpe.uchicago.edu.

[5]The white Union Army sample is available at http://www.cpe.uchicago.edu.

[6]See Fogel (2001) for a discussion of the sample representativeness of the white Union Army data.

who identified themselves as Union veterans in the 1910 census, 86 percent of the white veterans and 79 percent of the black veterans were found in the pension records.[7]

Cause of death information is available for 44 percent of black veterans and 48 percent of white veterans. It is more likely to be available if there was a surviving spouse and is more common for later cohorts. Availability in the black sample does not depend upon region of residence, but it does depend upon urbanization in 1900. Cause of death is not known for 62 percent of black men who in 1900 lived in smaller cities whereas for black men living in one of the 100 largest cities cause of death is not known in 38 percent of the cases. Among black men with some cause of death information, 20 percent of those in the south had a vague or unclassifiable cause of death whereas only 10 percent of those in the north did. Among both whites and blacks vague and unclassifiable causes of death were also more common when there was no surviving spouse and are more common for earlier cohorts.

3 Mortality of Black and White Veterans

The survival probabilities of black Union Army veterans were low relative to those of white veterans. Adjusting for age differences, after 20 years, only 40 percent of black veterans age 50-74 in 1900 were still alive compared to over 60 percent of native-born white veterans in the same age group (see Figure 1).[8] A comparison with men observed in the first National Health and Nutrition Examination Survey and followed until 1992 shows that, although the improvement in mortality rates has been greater among blacks than among whites, even in the 1970s black men only had achieved the survival probabilities of white Union Army soldiers. When black veterans are

[7]Soldiers who survived the war were less likely to have a pension if they were deserters (deserters who never returned to fight were ineligible), if they had never been injured in the war, if they had never been promoted, and if they were from a regiment that saw little fighting. Black soldiers were also less likely to have a pension if they had been born in the Confederacy, if they were free men at enlistment, and if they were dark-skinned.

[8]Figure 1 probably underestimates mortality improvements for both races for the population as a whole and particularly so for blacks because, as discussed in the Data Appendix, veterans may have had a more favorable mortality experience compared to the population as a whole.

compared with white veterans born abroad, they most closely resemble the Irish (Costa 2004b).

Cause of death information provides some clues on the sources of the large racial mortality differentials observed among Union Army veterans. Black death rates from genito-urinary disease and from the combined category of bronchitis, pneumonia, and influenza were particularly high relative to those of whites (see Table 1)[9] The category of genito-urinary disease probably includes deaths due to tertiary syphilis.[10] Syphilis rates were higher among the black population than the white population – during World War I, 12.63 per 1000 of white soldiers were diagnosed with syphilis compared to 64.99 per thousand of black soldiers (cited in Holmes 1937: 69). Black death rates from heart and cerebrovascular disease were lower than those of whites, but the etiology may have been different. The experience of the Metropolitan Life Insurance Company from 1911 to 1930 led one researcher to conclude that syphilis accounted for a third of all cardiac conditions among blacks and that it could explain both premature deaths from cerebral hemorrhage and arteriosclerosis (cited in Holmes 1937: 113). Other infectious diseases that have cardiac involvement include rheumatic fever, measles, and typhoid.

The causes of death shown in Table 1 show that disease experience varied both by race and by cohort. Whereas deaths from bronchitis, pneumonia, and influenza and from infectious disease fell for later white cohorts, they remained constant for black cohorts. Whereas deaths from heart and cerebrovascular disease rose for white cohorts, they remained constant for black cohorts, suggesting that black veterans were not yet experiencing the epidemiological transition with its shift from infectious to degenerative diseases. The proportion of deaths attributable to genito-urinary disease rose for both blacks and whites, but rose more for blacks.

Differences by cohort are seen not just in cause of death data but also in mortality data. Older

[9]Compared to men age 65-74 in the death registration states in 1914, veterans had lower infectious disease rates and higher heart disease rates. Black veterans also had higher death rates from influenza and pneumonia. (See Series HIST290, National Center for Health Statistics, http://www.nchs.gov.)

[10]Neurosyphilis can have urological manifestations; glomerulonephritis (a type of kidney disease) is a sequelae of tertiary syphilis; and, syphilis may increase the risk of prostate cancer (Hayes et al. 2000; Ertruk, Sheinfeld, and Davis 1989).

age mortality rates for later black cohorts increased whereas those for whites remained constant, leading to racial mortality differentials that were much smaller for earlier than for later cohorts (see Figure 4). Among men first observed at age 60 black life expectancy was lower for the cohort born in 1831-40 relative to the 1821-30 cohort and even lower for the cohort born in 1841-50.[11]

The Union Army data suggest that later cohorts, both white and black, faced worse early life conditions than early cohorts. Among white Union Army soldiers mean heights of men age 23-49 declined by 1.5cm between the 1821-30 cohort and the 1841-50 cohort. Among black Union Army soldiers the mean decline across cohorts was 2.1cm and declines are observed in all regions of the country.[12] However, despite a decline in heights for later cohorts of both races, only later black cohorts suffered elevated older age mortality rates. Unlike later white cohorts, later black cohorts did not experience an epidemiological transition and black health may have deteriorated under freedom as southern food production declined (Rose 1989; Ransom and Sutch 1977) and as rural blacks who may have lacked immunities to disease common in urban areas migrated to large cities.

Examining mortality rates by city size provides suggestive evidence on the role of infectious disease both early and later in life. A large city of enlistment was deadly for both whites and blacks, but especially so for blacks. Figure 2 shows that among men who enlisted in cities of 50,000 or

[11]This same cohort pattern is observed in mortality rates inferred from the decennial censuses (Costa 2004b), implying that the pattern does not arise from selection into the military. The decennial census data show that the 1851-60 cohort fared even worse than the 1841-50 cohort.

[12]The height declines observed in the Union Army data contrast with the findings of Steckel (2004), who finds that among young slaves listed in all of the available slave "manifests" (shipping documents used in inter-regional trade) there was no decline in height, a result consistent with that of Margo and Steckel (1982) using a smaller sample of manifests. Komlos (1992) finds a decline in heights for later cohorts of free blacks in Maryland similar to that observed among Union Army soldiers, but among Georgia convicts he finds a decline in the 1850s and 1880s (Komlos 1997). Bodenhorn (1999) finds a height decline for free Virginia blacks. Among Union Army soldiers measured by the Sanitary Commission, Costa (2004a) finds that black-white differences in vital capacity (a measure of lung capacity associated with mortality from respiratory disease and stroke) and in waist-hip ratio (a predictor of stroke and ischemic heart disease) were more pronounced among the younger cohorts. Differences between the military records and the manifests remain a puzzle. While differences may partially arise from the representation of different regions, a height decline is observed even among Union Army soldiers from the Deep South. Steckel (2004) looks at children up until age 16. Because growth occurred until age 23 or 25, blacks may have suffered stress as young adults that reduced heights. Steckel (2004) finds that the price of cotten fell relative to that of pork, the most expensive item in the slave diet.

more in 1860 there was a substantial urban mortality penalty at late ages for white veterans. The magnitude of the urban penalty for whites was equivalent to that of being a black slave. Although sample sizes are small, free blacks who enlisted in cities with a population of 50,000 or more suffered a tremendous older age mortality penalty. After 20 years less than one-quarter were still alive whereas almost of half of former slaves were still alive. These large cities were places such as Philadelphia where during the 1832 cholera epidemic the case rate was twice as high among blacks as among whites (Ewbank 1987). In contrast, there was no such urban penalty for black slaves, perhaps because place of enlistment was not as good an indicator of place of residence or birth for slaves as it was for free men.

White veterans who in 1900 lived in one of the 100 largest cities fared no worse than whites who lived in smaller cities (see Figure 3), but black veterans faced an urban mortality penalty by size of city of residence in 1900. After 20 years 30 percent of black veterans who lived in large city in 1900 were still alive compared to 40 percent of veterans who lived in a smaller city. As cities instituted sanitary reforms, the urban mortality penalty for whites began to disappear, but delays in extending services to black areas of town meant that only after 1900 did improved sanitation and water supplies lower black mortality rates in cities (Haines 2003; Troesken 2004). Migration, particularly to large cities, may have led to a tuberculosis and respiratory epidemic among the black population. The black population may have been particularly vulnerable because of lack of prior exposure. Kiple and King (1981: 142) point out in that only in the third generation exposed to tuberculosis is classic pulmonary tuberculosis observed. In addition, racial differences in syphilis rates were probably bigger in larger cities because black men in metropolitan areas were more likely to live apart from their families than men in non-metropolitan areas.[13]

[13] A higher prevalence rate of syphilis and other sexually transmitted diseases in large cities is consistent with the high rates of childlessness observed among ever married black women. In 1910 among ever married black women age 40-49 living in metropolitan areas 18 percent were childless compared to 8 percent of those living in non-metropolitan areas. The comparable figures for white women of the same age were 12 and 9 percent, respectively (estimated from the integrated public use micro census samples).

4 Econometric Framework

I use the data sample of everyone alive and on the pension rolls by 1900 to analyze the penalty for being black and for being black in an urban area. I examine waiting time until death using hazard models, in which the estimated hazard $\lambda(t)$ is

$$\lambda(t) = \exp(B'\beta_B + C'\beta_C + (B' \times C')\beta_{BC} + X'\beta_X)\lambda_0(t) \quad (1)$$

where B is a vector indicating that the individual is black, C is vector of control variables indicating city size, $B \times C$ is the interaction between black and city size, X is a vector of control variables for wartime experience, socioeconomic status, and demographic characteristics, and $\lambda_0(t)$ is the baseline hazard which I assume to Gompertz, $\lambda_0(t) = \exp(\gamma t)$. I examine both cities at enlistment and city of residence circa 1900. I define a large city at enlistment as one whose population was 50,000 or more in 1860 (the top 13 cities). In previous work I found that among white veterans those who enlisted in one of these cities had higher older age mortality even controlling for later residence, but that there were no older age mortality effects to enlisting in smaller cities (Costa 2003). I define a large city of residence in 1900 as one of the top 100 cities in the United States (a population of 38,307 or more). Because the sample of black veterans is not yet linked to the 1900 census, place of residence information is often not precise, but the top 100 cities are easily identified. This classification will most likely underestimate the urban penalty. Relative to cities with populations below 5,000 and to rural areas, cities with populations of 5,000 to 25,000 had higher mortality rates in 1900 (Haines 2001).

For men for whom cause of death is known, I also estimate independent competing risk models for specific causes of death, treating other causes of death as censored. These provide some clues on why there was a penalty for being black and on why mortality differed by city size. Competing risk models also enable me to examine waiting time until death from all diseases other than infec-

tious and thus to deduce the effect of eliminating deaths from infectious disease on the penalty for being black.

The primary causes of death studied are the combined categories of 1) heart and cerebrovascular disease; 2) infectious and parasitic disease and bronchitis, pneumonia, and influenza; and, 3) genito-urinary disease (including all mention of the prostate and of kidney problems). I can further subdivide heart and cerebrovascular into its two separate components. Although heart disease is often not classified by type, in some cases I can identify valvular and ischemic heart diseases to investigate the etiology of heart disease.[14] The three categories were picked because of their importance in the Union Army data, but they were also important causes of death for the population as a whole. DuBois (1899: 159) wrote that in Philadelphia in 1884-90, "The Negroes exceed the white death rate largely in consumption, pneumonia, diseases of the urinary system, heart disease and dropsy, and in still-births; they exceed moderately in diarrheal diseases, diseases of the nervous system, malarial and typhoid fevers."

I use the data on everyone on the pension rolls by age 60 to examine how mortality rates differed by cohort. I test for cohort effects by looking within the black sample and using hazard models of the form

$$\lambda(t) = \exp(Y'\beta_Y + X'\beta_X)\lambda_0(t) \tag{2}$$

where Y is a vector indicating birth cohort, X is vector of control variables, and $\lambda_0(t)$ is the baseline hazard which I assume to Gompertz, $\lambda_0(t) = \exp(\gamma t)$. The hazard ratios that I report indicate whether a one unit change in an independent variable gives an increase or decrease in the odds of an event. Cause of death information and competing risk models provide additional evidence on why mortality experience differed by cohort.

[14] Valvular heart disease is defined as all deaths which mention valvular heart disease or rheumatism. Ischemic heart disease is defined as all mentions of atherosclerosis, coronary heart disease, heart failure, or myocardial insufficiency.

The control variables that I create are dummies indicating occupation at enlistment (farmer, artisan, laborer, professional or proprietor, and unknown), dummies indicating occupation circa 1900 (farmer, artisan, laborer, professional or proprietor, and unknown), a dummy indicating whether or not the veteran could write, dummies indicating region of residence circa 1900 (east, midwest or west, border, and south), dummies indicating whether the soldier ever was a POW and whether the soldier ever had specific medical conditions while in the army (tuberculosis, stomach, rheumatic fever, respiratory, diarrhea, typhoid, malaria, fever, and wounded), size of city of enlistment or residence in 1900, and age or cohort dummies.

5 Results

Table 2 shows that controlling for wartime experience, socioeconomic status, and later and earlier size of city of residence, the penalty for being black was an odds of dying 1.3 times higher than that of whites. In large cities in 1900, the penalty for being black was even higher. The odds of death were 1.3 times higher for black veterans who in 1900 lived in a large city than for black veterans who did not live in a large city whereas for whites there was no mortality effect of living in a large city in 1900.[15] The urban mortality penalty in the top 100 cities explains roughly 15 percent of racial differences in older age mortality rates.[16]

Odds of death and city size were not linearly related (not shown). There was no urban penalty for either whites or blacks of living in one of the top 13 cities in the country in 1900, perhaps because the largest cities were the first ones to install a modern sewage and water infrastructure

[15] Both free blacks and ex-slaves faced similarly sized urban penalties to living in one of the top 100 cities in 1900 relative to whites (not shown). The penalty for being black was especially large in the border states (not shown). When the white and black samples were restricted to men living within a particular region, the hazard ratio on the black dummy variable was 1.538 ($\hat{\sigma}$=0.153) for men living in the border states, 1.366 ($\hat{\sigma}$=0.144) for men living in the south, and 1.190 ($\hat{\sigma}$=0.087) for men living in the former free states. The racial mortality differential between the border states and the former free states was statistically significantly different at the 10 percent level.

[16] The difference in the predicted number of years lived between blacks and whites in the sample was 2.0 years. Had there been no men in large cities in 1900 the difference between whites and blacks would have fallen to 1.7 years.

(and suggesting also that persistence of residence does not explain the effect of city of enlistment on older age mortality). For whites there was no urban penalty to living in the next 87 largest cities (the hazard ratio was 1.014, $\hat{\sigma}$=0.049), but for blacks there was a substantial penalty (the hazard ratio was 1.328, $\hat{\sigma}$=0.144).

Table 2 also shows that there was a permanent scarring effect of enlisting in a large city for whites (their odds of dying were 1.2 times greater than those of whites enlisting in a small city) whereas for blacks there was none. (Blacks enlisting in large cities had lower mortality rates than those enlisting in smaller cities, largely because this category is dominated by former slaves who enlisted in large southern cities but may not have lived there.) Hazard regressions that examine the effects of slave status reveal the same patterns seen in Figure 2 (not shown). Controlling for wartime experience, socioeconomic status, and later and earlier residence, both ex-slaves and freemen fared worse than whites. Although the hazard ratio on the interaction term between the dummy for a free black man and the dummy for enlisting in a large city was not statistically significantly different from 1, the magnitude of the hazard ratio implies that free blacks faced an additional urban penalty.[17]

Table 3, which examines the correlates of mortality by cause, shows that elevated black mortality rates both in large and small cities and in rural areas are due to infectious and parasitic disease and to bronchitis, influenza, and pneumonia. The odds of a black veteran dying from infectious disease were 1.5 times higher than those of a white veteran. When I estimate a competing risk model in which I treat deaths from infectious and parasitic disease and bronchitis, influenza, and pneumonia as censored I find that the odds of a black veteran dying from another cause of death were 1.1 times higher than those of a white veteran and that, based upon predicted median years lived beyond 1900, eliminating deaths from infectious disease would have reduced the black-white

[17] In this specification the hazard ratio on enlisting in a city of 50,000 or more was 1.17 ($\hat{\sigma}$ =0.065) and the interaction term between a black male being free and enlisting in a city of 50,000 or more was 1.16 ($\hat{\sigma}$ =-.242).

mortality differential by 50 percent.[18] Using data on all veterans who were on the pension rolls by age 60 yields similar results – eliminating infectious disease would have reduced the racial mortality penalty in median years lived beyond age 60 by 56 percent.[19]

Table 3 shows that blacks living in a large city in 1900 faced an odds of dying from infectious disease that was 1.3 times that of blacks living in a small city or of whites (though the difference was not statistically significant), an odds of dying of genito-urinary disease that was twice that of blacks living in a smaller city or of whites and an odds of dying from cerebrovascular and heart disease that was 1.5 times that of blacks living in a smaller city or of whites. Excess black deaths in cities from the combined category of cerebrovascular and heart disease were mainly due to cerebrovascular disease. In smaller cities and rural areas blacks were at lower risk of death from cerebrovascular disease than whites (the hazard ratio on the black dummy was 0.428 with a standard error of 0.146 and the hazard ratio on the interaction between black and the top 100 cities was 3.327 with a standard error of 1.354). Recall that because black men in metropolitan areas were more likely to live apart from their families than black men in non-metropolitan areas, syphilis and other sexually transmitted diseases may have been more prevalent in large cities, leading to higher death rates from genito-urinary disease and from cerebrovascular disease.[20] Blacks in both urban and rural areas suffered from a higher death rate from valvular heart disease (the hazard ratio on the black dummy was 2.770, with a standard error of 0.662), but there was no urban penalty for valvular heart disease. There is some suggestion of an urban penalty to ischemic disease, but the results are not statistically significant.

Differences in socioeconomic status, as proxied by occupation circa enlistment and 1900, could not explain racial mortality differentials. Among whites, men who were laborers circa 1900 had

[18]The difference in predicted median years lived would have fallen from 1.2 to 0.6.

[19]Predictions from a competing risk model in which infectious and parasitic disease and deaths from bronchitis and influenza are treated as censored implies that the predicted median number of years lived beyond age 60 would have fallen from 0.9 to 0.4 years.

[20]The connection between syphilis and later disease is apparent from army records. Among blacks, wartime syphlis predicted death from genito-urinary and from cerebrovascular disease.

higher mortality rates than men who were professionals and proprietors or farmers. Among blacks only men who were servants circa 1900 had a mortality advantage.

Hazard models show that black cohorts born 1841-52 were at greater risk of death from all causes, from genito-urinary disease and from infectious and parasitic disease, from bronchitis, influenza, and pneumonia than earlier black and white cohorts (see Table 4). The odds of death from all causes for a black veteran born 1841-52 were 1.4 times higher than those of black veterans born earlier and 1.5 times higher than those of whites. The odds of death from genito-urinary disease for a black veteran born 1841-52 were 1.7 times higher than those of a black veteran born before 1841 or those of a white veteran. The odds of death from infectious and parasitic disease for a black veteran born 1841-52 were 1.8 times higher than those of a black veteran born before 1841.[21] In contrast, white cohorts born 1841-52 faced a risk of death from infectious and parasitic disease that was 0.7 times lower than that faced by earlier white cohorts.

Later black cohorts also faced higher death rates from cerebrovascular and heart disease than earlier cohorts. Although the hazard ratio is not statistically significantly different from 1, its magnitude implies that the odds of death from cerebrovascular and heart disease were 1.3 times higher for the black cohorts born 1841-52 than for those born earlier. In contrast, white cohorts born 1841-52 faced a risk of death from cerebrovascular and heart disease that was 1.1 times higher than those of earlier white cohorts. Excess deaths among the later black cohorts are due not to cerebrovascular disease, but to heart disease, especially valvular heart disease. When the sample was restricted to blacks only, the odds of death from valvular heart disease for the cohort born 1841-52 were 2.147 times as high as for those born earlier. Unlike blacks, later white cohorts faced lower risk of death from valvular heart disease and a higher risk of death from cerebrovascular and ischemic heart disease compared to earlier white cohorts. The hazard ratios on the 1841-52 cohort dummy in the competing risk regressions on death from cerebrovascular, valvular, and ischemic

[21] Eliminating infectious diseases as a cause of death would not have affected the mortality gap between the 1841-52 cohort and earlier cohorts.

heart disease were 1.153 ($\hat{\sigma}$=0.099), 0.803 ($\hat{\sigma}$=0.093), and 1.226 ($\hat{\sigma}$=0.126), respectively.

Why did later black cohorts suffer elevated mortality rates at older ages? Although both later white and black cohorts suffered poorer early life conditions, only black cohorts were affected and controlling for height as a measure of early life conditions had no effect on estimated cohort effects. Height was not even a statistically significant predictor of mortality. Later cohorts were slightly more likely to migrate to large cities, but the difference in migration rates was small (0.24 vs 0.21) and cohort effects persist even controlling for size of city of residence circa 1900.[22] In addition, all cause mortality rates were greater for later black cohorts regardless of size of city of residence.[23] Interestingly, the deterioration in mortality rates while observed in the New England and the Middle Atlantic, the border states, and the Midwest, is not evident in the Jim Crow South. Later black cohorts in the North may have suffered a greater deterioration in living conditions because the migration of freedmen from the South to the North worsened race relations and led to competition in the labor market (DuBois 1899). European immigrants provided additional labor market competition. Sacerdote (2005) argues that one of the reasons for the rapid convergence in the economic and educational outcomes of former slaves and freemen was because whites began treating free blacks and former slaves with the same contempt. The poorer early life conditions of later black cohorts could only increase their vulnerability.[24]

[22]Looking at deaths from all causes within the black sample, a specification which controls for size of city of residence and for an interaction term between size of city of residence and cohort yields a hazard ratio on the 1841-52 cohort of 1.295 ($\hat{\sigma} = 0.113$).

[23]The interaction term between a large city and a cohort dummy yielded a coefficient that was small and insignificant in the black sample. However, the excess infectious and parasitic disease mortality of the 1841-52 black cohort was predominately an urban phenomenon.

[24]Supporting evidence is found in an examination of survivorship rates across decennial censuses for those age 20-49 and those age 50-79. These show that black-white mortality differentials increased sharply in the early 1900s for all age groups. However, these regressions also show that a mortality penalty for black cohorts born close to the end of slavery was not just an old-age phenomenon. Thus interactions between early life and late life conditions may have been important.

6 Conclusion

At older ages, black Union Army veterans faced an odds of dying that was 1.3 times higher than that of whites. They were more likely than white veterans to die of infectious and parasitic disease. Had it been possible to eliminate deaths from infectious disease, the racial mortality differential in years lived at older ages would have fallen by 50-56 percent. This is probably an underestimate of the effect of eliminating infectious disease. Excess blacks' deaths from genito-urinary disease were related to syphilis and excess deaths from heart disease were related to rheumatic fever. In large cities, blacks faced an urban mortality penalty that accounted for roughly 15 percent of the mortality differential between blacks and whites.

The mortality penalty to being black varied widely by cohort. The 1821-30 cohort did not pay a penalty for being black, but the 1841-50 black cohort faced an odds of dying that 1.4 times higher than that of earlier black cohorts and had higher mortality rates from all causes, from infectious and parasitic disease, and from degenerative diseases with an infectious disease origin such as genito-urinary disease and valvular heart disease. In contrast, older age mortality rates were the same across different white cohorts. The deterioration in black mortality rates is not observed in the South, suggesting that later black cohorts may have suffered from a deterioration in living conditions for blacks in the North.

It was not until the 1970s that older black men achieved the mortality rates of white Union Army veterans. Why did change take so long? This paper has emphasized the role of infectious disease and of the deterioration in black mortality rates in explaining the black-white mortality gap. Because blacks and the poor could ill afford self-protection measures or treatment, declines in infectious disease rates among blacks had to await public health investments in sanitation and water filtration (Troesken 2004; Costa and Kahn 2003), public health campaigns such as those against hookworm (Bleakley 2002), and improvements in the black-white wage gap.

Data Appendix

The black sample is representative of the US Colored Troops in terms of geography and slave status and therefore was more northern than the population as a whole. It contains men who, circa 1900, were of slightly higher occupational status and more urban than the postbellum black population of the same age. Twenty-two percent of black veterans in the sample lived in one of the top 100 cities in the United States circa 1900. Among non-veterans in the same age group in 1910 the comparable figure was 10 percent. Among black veterans who were on the pension rolls by 1900, 41 percent of those reporting a last occupation were farmers and 11 percent were professionals, proprietors, or artisans. The comparable figures for all blacks in the same age group reporting an occupation in 1900 were 49 and 8 percent, respectively.[25]

The ten year mortality rates of both white and black veterans are within the expected range for the population as a whole. Census survivorship imply that the 10 year mortality rate for men age 50-59 was 0.386 for blacks and 0.232 for whites. In the more urban (and hence higher mortality) death registration states the ten year mortality rate was 0.382 for blacks and 0.223 for whites. The mortality rate for Union Army veterans age 50-59 was 0.346 for blacks and 0.215 for whites. The ten year mortality rate for white men in a sample of rural genealogies was 0.166 (Kunze 1979).

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Table 1: Causes of Death by Race and Cohort

	White			Black	
	b.1821	b.1831	b.1841	b.1831	b.1841
Heart disease	23.49	29.76	33.70	30.50	30.24
Cerebrovascular disease	6.76	10.99	13.14	7.80	7.80
Bronchitis, pneumonia, and influenza	10.32	9.75	8.89	13.48	14.63
Infectious and parasitic	11.03	6.38	4.28	4.96	4.88
Genito-urinary	7.30	11.25	12.36	12.06	15.12
Chronic respiratory	1.96	2.18	1.22	2.13	1.46
Violence	4.63	2.38	2.90	0.71	1.95
Diabetes	1.07	0.83	1.10	0.00	1.46
Cancer	3.56	4.56	6.16	2.13	2.44
Stomach	4.45	4.51	2.90	2.84	1.46
Paralysis	5.87	3.21	3.02	2.84	3.41
Other	19.57	14.20	10.32	20.57	15.12

The samples were restricted to men who were alive and on the pension rolls at age 60. No figures are given for the 1821 black cohort because the sample size is too small.

Table 2: Effect of Race and Size of City of Enlistment and of 1900 Residence on Time Until Death From All Causes

	Hazard Ratio	Hazard Ratio	Hazard Ratio
Dummy=1 if black	1.336‡	1.282‡	1.314‡
	(0.064)	(0.066)	(0.069)
Dummy=1 if enlisted in large city	1.083	1.085*	1.167‡
	(0.084)	(0.054)	(0.065)
Black × enlisted in large city			0.724‡
			(0.088)
Dummy=1 if lived in one of 100 largest cities in 1900	1.073*	1.030	1.020
	(0.041)	(0.044)	(0.044)
Black × 100 largest city		1.231†	1.276‡
		(0.112)	(0.118)
γ	0.084‡	0.084‡	0.084‡
	(0.001)	(0.001)	(0.001)
Log-Likelihood	-10508.176	-10505.617	-10501.933

9720 observations. All regressions are Gompertz hazard models. The sample consists of men who were age 50-74 and on the pension rolls in 1900. Additional control variables include dummies indicating occupation at enlistment (farmer, artisan, laborer, and professional or proprietor), dummies indicating occupation circa 1900 (farmer, artisan, laborer, and professional or proprietor), a dummy indicating whether the veteran could write, dummies indicating region of residence circa 1900 (east, midwest or west, border, and south), dummies indicating whether the soldier ever was a POW and whether the soldier ever had specific medical conditions while in service (tuberculosis, stomach, rheumatic fever, respiratory, diarrhea, typhoid, malaria, fever, and wound), age dummies, and dummies indicating missing occupation or size of city of residence or enlistment. The symbols ‡, †, and $*$ indicate that the coefficient is significantly different from 0 at the 1, 5, and 10 percent level respectively.

Table 3: Effect of Race and Large City Residence in 1900 on Time Until Death From Specific Causes

	Infectious/ Parasitic/ Bronchitis Hazard Ratio	Genito-urinary Hazard Ratio	Cerebro-vascular and Heart Hazard Ratio
Dummy=1 if black	1.489‡	1.024	0.937
	(0.0253)	(0.022)	(0.121)
Dummy=1 if lived in one of 100 largest cities in 1900	1.023	0.901	1.095
	(0.148)	(0.140)	(0.087)
Black × 100 largest city	1.307	1.967†	1.528†
	(0.366)	(0.624)	(0.294)
γ	0.063‡	0.086‡	0.096‡
	(0.004)	(0.005)	(0.002)
Log-Likelihood	-2412.713	-1849.735	-3553.942

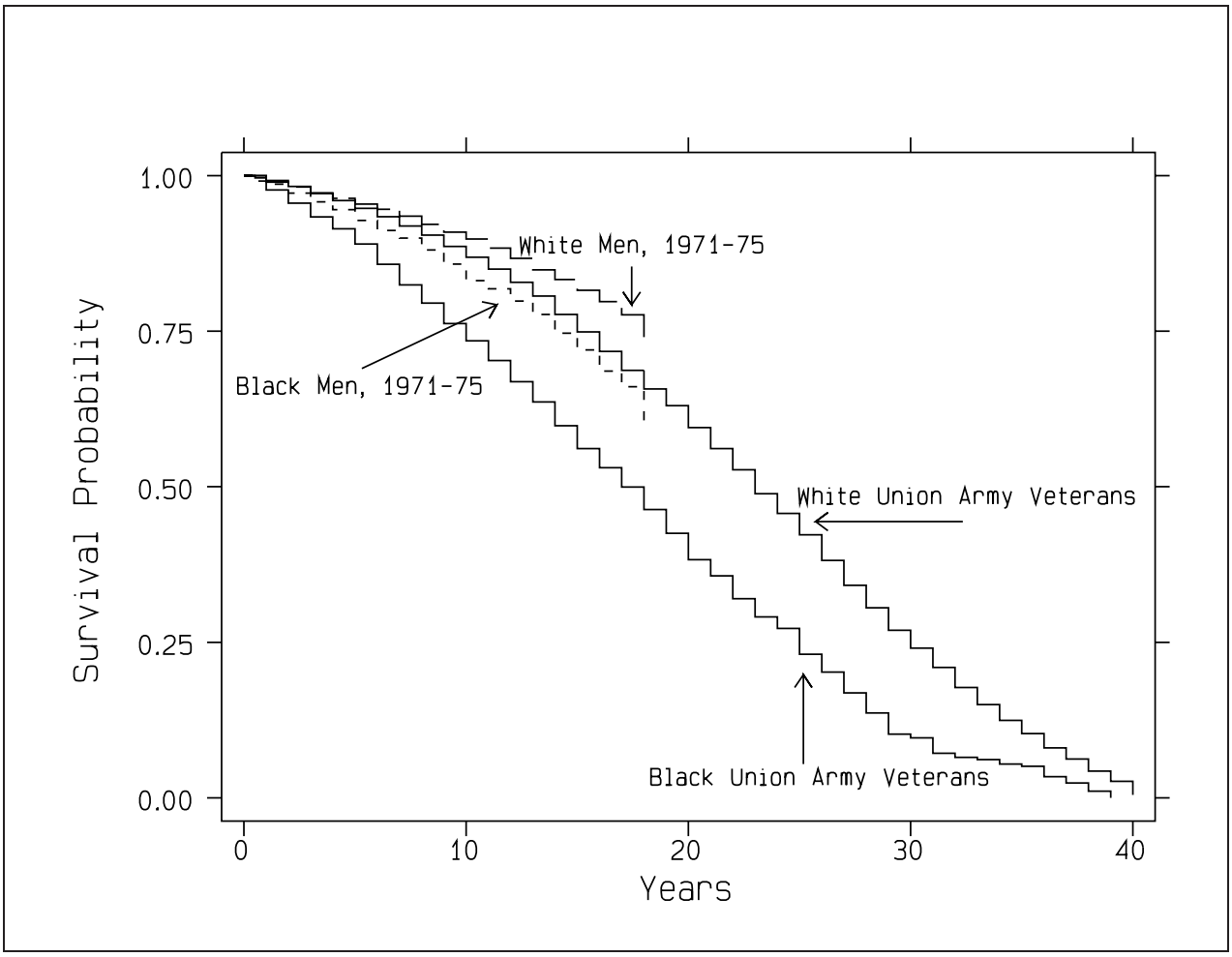
5031 observations. All regressions are Gompertz hazard models. The sample consists of who were age 50-74 and on the pension rolls in 1900. Additional control variables include dummies indicating occupation at enlistment (farmer, artisan, laborer, and professional or proprietor), dummies indicating occupation circa 1900 (farmer, artisan, laborer, and professional or proprietor), a dummy indicating whether the veteran could write, dummies indicating region of residence circa 1900 (east, midwest or west, border, and south), dummies indicating whether the soldier ever was a POW and whether the soldier ever had specific medical conditions while in service (tuberculosis, stomach, rheumatic fever, respiratory, diarrhea, typhoid, malaria, fever, and wound), age dummies, and dummies indicating missing occupation or size of city of residence or enlistment. The symbols ‡, †, and $*$ indicate that the coefficient is significantly different from 0 at the 1, 5, and 10 percent level respectively.

Table 4: Effect of Race and Cohort on Time Until Death From All Causes and from Specific Causes

	All Causes Hazard Ratio	Infectious/ Parasitic/ Bronchitis Hazard Ratio	Genito-Urinary Hazard Ratio	Cerebro-vascular and Heart Hazard Ratio
Dummy=1 if black	1.111*	1.170	0.985	0.833
	(0.065)	(0.219)	(0.243)	(0.114)
Dummy=1 if born before 1841				
Dummy=1 if born 1841-52	0.982	0.717‡	1.083	1.110†
	(0.021)	(0.054)	(0.093)	(0.049)
Black $\times$ b.1841-52	1.384‡	1.780†	1.694*	1.290
	(0.107)	(0.444)	(0.524)	(0.231)
γ	0.087‡	0.067‡	0.090‡	0.093‡
	(0.001)	(0.004)	(0.005)	(0.002)
Log-Likelihood	-10330.419	-2496.661	-1830.873	-3854.649

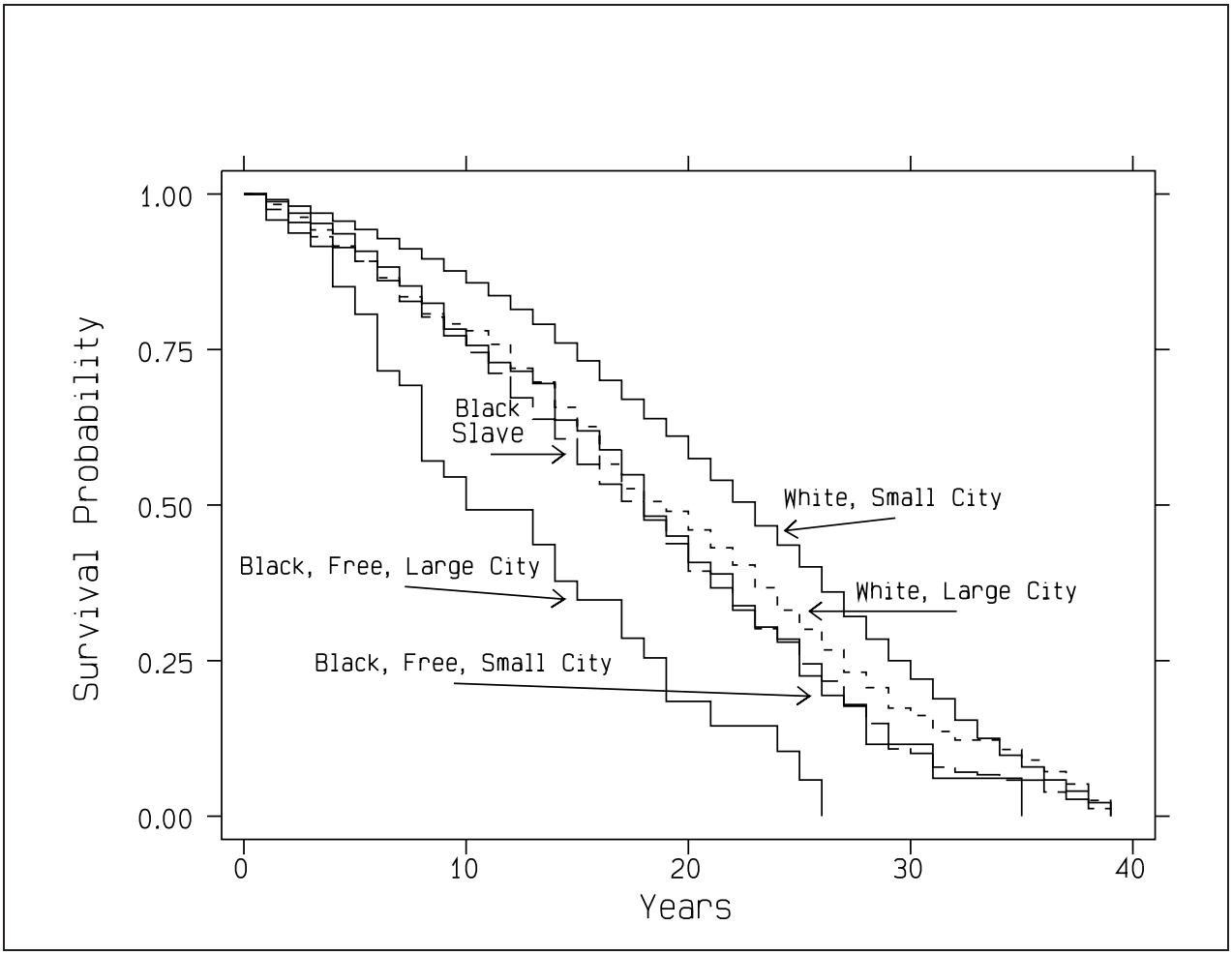
10,485 observations in the all cause mortality regression and 5,305 observations in the cause specific mortality regressions. The sample consists of men who were alive and on the pensions rolls at age 60. All regressions are Gompertz hazard models. Other causes of death are censored and assume independent competing risks. Additional control variables include a dummy equal to one if the soldier could write, dummies indicating occupation at enlistment, and dummmies indicating whether the soldier was ever a POW and whether the soldiers ever had specific medical conditions while in the service. The symbols ‡, †, and $*$ indicate that the coefficient is significantly different from 0 at the 1, 5, and 10 percent level respectively.

Figure 1: Survival Probabilities by Race, Union Army Veterans and Men in NHANES I, Age 50-74



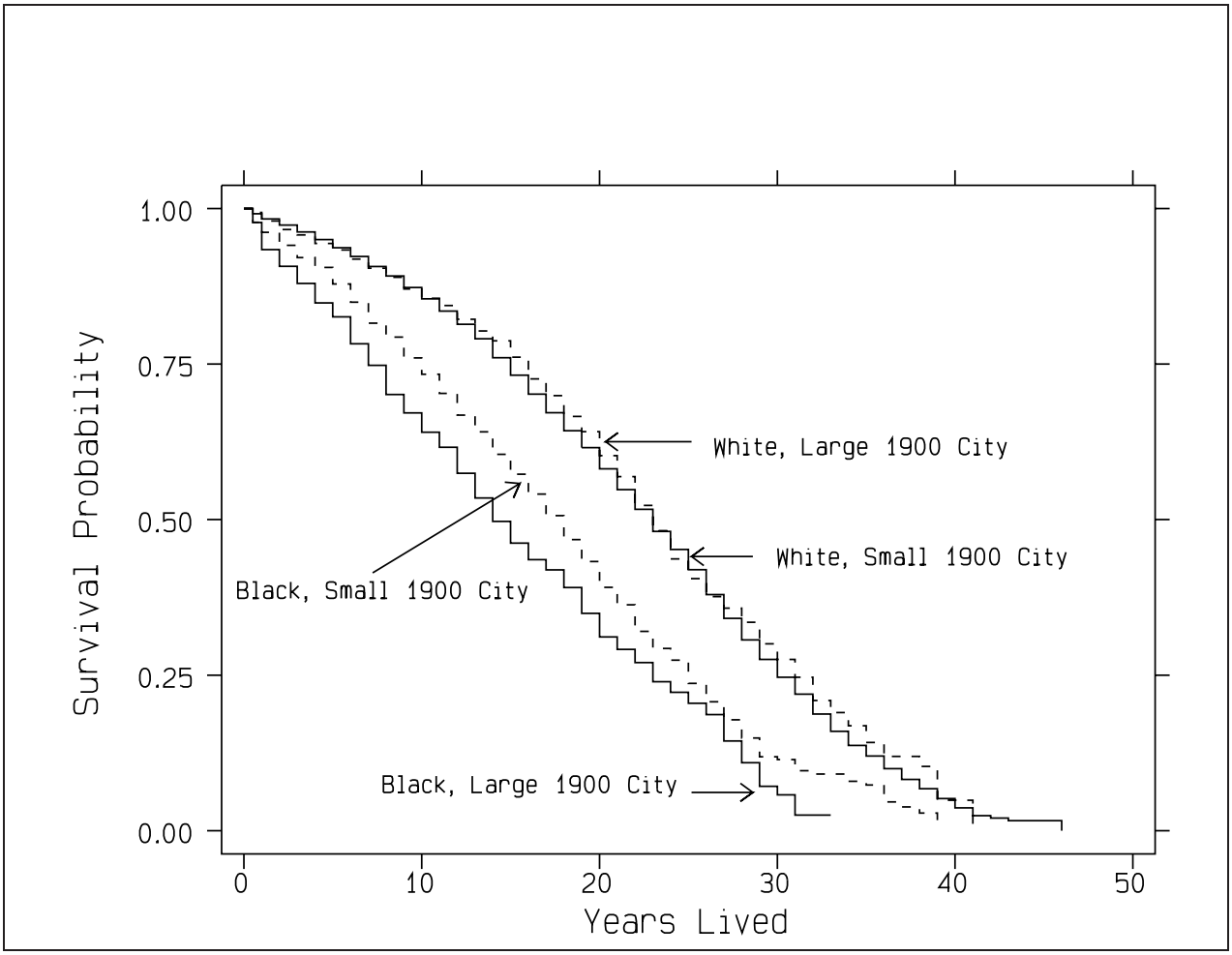
The Union Army sample is restricted to the native-born, those alive in 1900, and those on the pension rolls by 1900. All men are aged 50-74 at the time of observation. Survival curves are adjusted for age. 984 black Union Army veterans. 9,605 white Union Army veterans.

Figure 2: Survival Probabilities by Race and Size City of Enlistment, Union Army Veterans



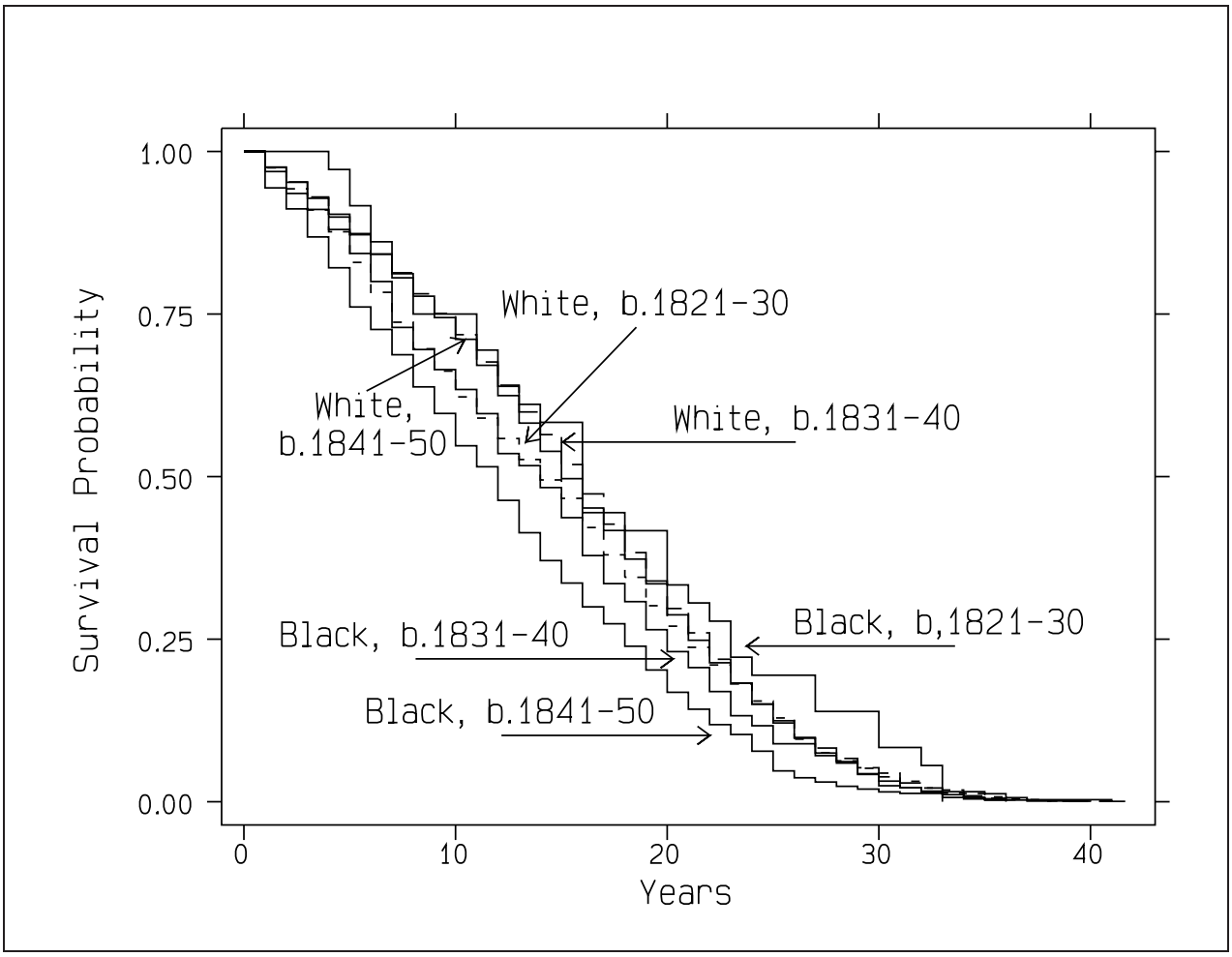
The Union Army sample is restricted to the native-born, to those alive in 1900, and to those on the pension rolls by 1900. All men are aged 50-74 at the time of observation. Survival curves are adjusted for age. City size is city at enlistment. A large city is defined as one with 50,000 or more people. 894 blacks and 9,605 whites.

Figure 3: Survival Probabilities by Race and Size City of 1900 Residence, Union Army Veterans



The Union Army sample is restricted to the native-born, to those alive in 1900, and to those on the pension rolls by 1900. All men are aged 50-74 at the time of observation. Survival curves are adjusted for age. A large city is one of the top 100 cities in 1900 and a small city is either a rural area or a city that was not one of the top 100 cities in 1900. 894 blacks and 9,605 whites.

Figure 4: Survival Probabilities by Race and Cohort, Union Army Veterans



The Union Army sample is restricted to the native-born and those observed alive and on the pension rolls by age 60. Years is years until death from age 60. 825 black Union Army veterans. 9,750 white Union Army veterans.