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SCARRING AND MORTALITY SELECTION AMONG CIVIL WAR POWS:
A LONG-TERM MORTALITY, MORBIDITY AND SOCIOECONOMIC FOLLOW-UP

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ABSTRACT

Debilitating events could leave either frailer or more robust survivors, depending on the extent of scarring and mortality selection. The majority of empirical analyses find frailer survivors. I find heterogeneous effects. Among severely stressed former Union Army POWs, which effect dominates 35 years after the end of the Civil War depends on age at imprisonment. Among survivors to 1900, those younger than 30 at imprisonment faced higher older age mortality and morbidity and worse socioeconomic outcomes than non-POW and other POW controls whereas those older than 30 at imprisonment faced a lower older age death risk than the controls.

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The first total war, the American Civil War, provides a unique opportunity to examine the long-run effects of acute malnutrition and the stresses associated with imprisonment on later life socioeconomic outcomes and older age mortality and morbidity. At Andersonville, the most notorious of the POW camps, roughly one-third of POWs died within 7 months (Rhodes 1904: 404) and forty percent of the men who passed through Andersonville died there.[1] Diarists accounts describe men weighing 90-95 pounds, down from almost 180, when they left Andersonville (Basile 1981; Ransom 1963). Walt Whitman wrote of the returning prisoners, "Can these be *men* – these little, livid brown, ash-streaked monkey-looking dwarfs? Are they not really mummied, dwindling corpses?"

This paper examines the effect of imprisonment in Confederate POW camps on the mortality, morbidity, occupational attainment, and property ownership of Union Army veterans 35 years after the end of the war. Conditions in Confederate POW camps deteriorated sharply when prisoner exchanges stopped, leading to severe over-crowding and reduced rations. Union Army non-POWs and POWs imprisoned when conditions were better provide control groups for POWs imprisoned when conditions were at their worst.

Starvation and the diseases and stresses of imprisonment could leave either permanently scarred survivors or very resilient survivors through positive selection effects. Models of mortality selection posit that when mortality at younger ages is high, frailer individuals die, leaving a more robust population that survives longer. These models have been used to explain the deceleration of the age pattern of mortality at older ages (Horiuchi and Wilmoth 1998) and the black-white mortality crossover (Manton and Stallard 1981; cf. Preston et al. 1996). Empirical analyses, however, predominately show positive associations between debilitating events and the morbidity and mortality of adults more than 20 years after the event (e.g. Horiuchi 1983; Finch and Crimmins 2004;

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[1]See United States War Department (1880- 1901), Series II, Vol. VIII, pp. 615, 781. In a longitudinal random sample roughly 38 percent of the 554 men held at Andersonville died there. In the National Park Services cross-sectional data base 40 percent of the listed men died at Andersonville (see Costa and Kahn 2007).

Barker 1992, 1994; Almond and Mazumder 2005; cf. Casselli and Capocaccia 1989; Kannisto et al. 1997). Studies of POWs are no exception, with the caveat that the biological processes through which insults at young adult ages affect older age mortality are different from the processes through which insults at ages when organs are still developing affect older age mortality. Dent et al. (1989) found that Australian WWII POWs held by the Japanese faced excess mortality rates compared to non-POW combatants more than 38 years after the war, even though the the two groups had similar degrees of medical morbidity (Goulston et al. 1985; Tennant, Goulston, and Dent 1986). Page and Brass (2001) and Page and Ostfeld (1994) report that among US POWs acute malnutrition was correlated with higher risks of death from ischemic heart disease, particularly after age 75. In contrast, studies of Holocaust survivors find no difference in the mortality of camp survivors compared with other European-born Jews both 20-41 years and 40-50 years following World War II (Williams et al. 1993; Collins et al. 2004), but the samples in these studies are small.

I find both positive and negative older age mortality effects of the Civil War POW experience. Among men younger than age 65 in 1900, POWs imprisoned when conditions were at their worst were more likely than non-POWs to die of any cause and to die of heart disease and stroke. They also exhibited more cardiovascular signs and symptoms and valvular heart disease. They were also more likely to be laborers and were less likely to own property. But among men older than 64 in 1900, those imprisoned when conditions were at their worst were less likely to die than their counterparts who were not POWs or who were POWs during better times. These POWs were older than age 30 at captivity and faced a higher mortality rate in captivity than younger men, suggesting that those older men who survived POW camps may have been more robust.

This study has several advantages over previous work on the long-run effects of starvation and the stresses of imprisonment. Twenty-five percent of the Union Army was above 30 years of age at enlistment[2], enabling me to examine how catastrophic stress affects different age groups. Because

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[2]Estimated from a random sample of Union Army soldiers.

records on the Union Army cohorts are in the public domain, I am able to examine a broader array of outcomes than many other studies. Finally, because the data are based on administrative records, including detailed medical examinations at older ages, I do not have to depend on self-reports.

1   Confederate POW camps

An estimated 211,411 Union soldiers were captured during the Civil War. 16,668 were never imprisoned because they were paroled on the field, but of the remaining 194,743 men, 30,218 died while in captivity (Rhodes 1904: 507). Thus, 7 percent of all U.S. soldiers were ever imprisoned compared to figures of 0.8 percent for World War II and 0.1 percent for Korea.[3] Until mid-1863 many POWs were exchanged immediately. Prisoner exchanges stopped as the two sides argued over the terms, particularly the treatment of black soldiers, who could be re-enslaved, and their white officers, who could be executed as leaders of a slave insurrection. Men were exchanged again in December of 1864 and early in 1865. The mean number of days spent in prison until death or release for men who were captured prior to mid-1863 was 20 whereas it was 92 for men who were captured after mid-1863.

Men who were captured after mid-1863 faced ever worsening conditions as the crowds of prisoners increased. In a random sample of soldiers, 4 percent of the men captured before July of 1863 died in captivity whereas 27 percent of those captured July 1863 or later died in captivity. In contrast, the total wartime mortality rate was 14 percent (Costa and Kahn 2007b). Although men could buy food and goods within POW camps and trade with the guards, men who had been imprisoned for long had nothing left to trade. POWs suffered from poor and meager rations, from contaminated water, from grounds covered with human excrement and with other filth, from a want of shoes, clothing, and blankets (having often been stripped of these by needy Confederate soldiers),

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[3]Estimated from the figures in U.S. Department of Veterans Affairs (2004) and from http://www.cwc.lsu.edu/cwc/other/stats/warcost.htm.

from a lack of shelter in the open stockades that constituted camps such as Andersonville and Millen, from the risk of being robbed and murdered by fellow prisoners, and from trigger-happy guards. Andersonville (also known as Camp Sumter and located in Georgia), with a maximum capacity of 10,000 men held at one point 32,899 men (Speer 1997: 332). As crowding increased, rations were stretched thin. According to Warren Lee Goss, "The first morning after our arrival [at Andersonville on May 1, 1864] about twenty pounds of bacon and a bushel of Indian meal was given to me to distribute among ninety men. We had no wood to cook with ..." (Goss 1866: 75). By June of 1864 corn bread and corn meal (a large fraction of which was ground cob) became the principal staples of the diet, and by August most prisoners were reported to be suffering from scurvy (Marvel 1994: 74, 179). Once pellagra became recognized as a public health problem in the United States, Niles (among others) argued that veterans' descriptions of "how the men had a supposed eczema; ... how their skins were rough and hard, and how their hands were sore and cracked; how their bowels were chronically loose ... [and how] the melancholy deepening into the different forms of dementia" were suggestive of pellagra but added that "[w]hether or not this was really pellagra will probably not be positively known" (Niles 1912: 26-27). The chief recorded causes of death were scurvy, diarrhea, and dysentery. Scorbutic ulcers became gangrenous.[4] Andersonville was emptied of men in September of 1864 when Sherman's army threatened and, although still used as a POW camp, never achieved the same levels of crowding.

Camp crowding was the single most important predictor of death as a POW (Costa and Kahn 2007b, 2009: Figure 2, p. 147). The individual characteristic that mattered the most was age. In a random sample of soldiers, 14 percent of those who became POWs before age 30 died whereas 21 percent of those who became POWs after age 30 died. In this same sample, 31 percent of those who became POWs before age 30 at Andersonville died compared to 54 percent of those who became POWs after age 30 at Andersonville.

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[4]Testimony from the trial of Captain Wirtz, reprinted in Ransom (1963).

2 POW Status and Older Age Outcomes

POWs suffered from starvation, disease, and psychological stress. Apriori, this earlier life experience could leave either a more robust older population through mortality selection or a frailer older population through scarring. Suppose that frailty in the population prior to entry in the POW camp is normally distributed with mean μ with frailer individuals below the mean. Assume that scarring shifts the distribution toward the left so that everyone is frailer and the new mean is μ'. If selection truncates the new distribution at frailty level a, mean frailty becomes $\mu' + \sigma\left[\frac{\phi((a-\mu')/\sigma)}{1-\Phi((a-\mu')/\sigma)}\right]$. Whether the average person becomes frailer or more robust depends both on the extent of scarring and mortality selection. Because I observe the effect of the POW experience on older age outcomes, I can only determine which effect, on average, dominates.

The scarring effects of POW status on older age outcomes could operate through several channels. Poor health when discharged from the army, by limiting men's ability to work and to climb the occupational ladder, would increase their chances of being laborers rather than farmers or artisans and decrease their wealth, their likelihood of owning property, and their odds of being married. Their lower socioeconomic status might then place them at greater risk of developing cardiovascular disease, either through greater exposure to disease, their worse nutrition, or stress. However, POW status in itself could also have an effect on later life heart disease.

The evidence linking starvation, disease, and psychological stress to heart disease is largely epidemiological. Although there is no clear biological mechanism linking nutritional deprivation to subsequent chronic heart disease, cardiac atrophy has been found in healthy adult volunteers subjected to a semi-starvation diet, in starved rats, in individuals on energy-restricted diets, and in patients suffering from cardiac cachexia. However, during the rehabilitation of healthy adult volunteers on a semi-starvation diet, heart size increased rapidly after the initial decline (Keys et al. 1950: 203-206).

Dietary deficiencies also have cardiac sequelae. Thiamine deficiency leads to peripheral va-

sodilation, potentially resulting in cardiac failure, and selenium deficiency has been associated with cardiomyopathy (see Webb, Kiess, and Chan-Yan 1986 for a review). Folates and vitamins B6 and B12 are required for the metabolism of homocysteine to methionine. Elevated homocysteine levels are a risk factor for coronary heart disease and ischemic stroke (see the review by Fairfield and Fletcher 2002). The reporting of such signs of vitamin deficiency as edema and night blindness while in WWII Japanese prison camps (where the diet consisted mainly of polished white rice) is correlated with cardiovascular morbidity and mortality (Page and Ostfeld 1994).

The weakened immune systems of the starved POWs placed them at greater risk of developing infectious disease and camp crowding increased their exposure to infectious disease. Some of infectious agents most commonly linked to heart disease include rheumatic fever (valvular heart disease), the coxsackie B virus (pericarditis and myocarditis), and chlamydia pneumoniae, helicobacter pylori, and dental infections (atherosclerosis via inflammation). Those who have had a prior attack of rheumatic fever (common in the Civil War armies) are highly susceptible to recurrences after future streptococcal infections. A study of former British Far East WWII POWs (Gill 1983) found a higher proportion of deaths mentioning rheumatic heart disease compared to the general population, but the number of cases was small.

Physiological changes associated with the dysregulation of the stress system are implicated in the development of a variety of illnesses, including hypertension and atherosclerosis. Former POWs are more likely to suffer from depression and PTSD than veterans who were not POWs (Page 1992) and POWs with PTSD had statistically significant increased risks of cardiovascular diseases including hypertension and chronic ischemic heart disease when compared to both non-POWs and POWs without PTSD (Kang et al. 2006).

Starvation is associated with a fall in heart rate, blood pressure, and blood volume and during refeeding there is a sudden reversal in these compensatory factors. Cardiac output needs to rise to handle increased salt, water and energy loads (see Webb, Kiess, and Chan-Yan 1986 for a review).

Congestive cardiac failure is a danger during refeeding of starved individuals. Refeeding syndrome has been noted in Japanese-held WWII POWs (Schnitker, Mattman, Bliss 1951), malnourished children (Edozien and Rahim-Kahn 1968), and patients suffering from anorexia nervosa (Casiero and Frishman 2006). The long-run effects of refeeding on chronic heart conditions are unknown.

3   Data

The data come from two different sources – a sample of Union Army soldiers (the Fogel sample), some of whom were taken prisoners, and a random selection of men who were at Andersonville. The Fogel sample comes from a full sample of all men within 303 companies.[5] Complete military records are available for these men and these provide information on wartime service and on demographic and socioeconomic characteristics at enlistment.[6] These data contain 3,175 cases of captivity with known dates of capture and of release or death for 3,040 men.[7] I know which prison a man entered and on what date, whether he survived or whether he died. These records are not limited to men who were at Andersonville.

The National Park Service's Andersonville database contains 35,323 men and was drawn from such disparate sources as the lists of the dead and published state muster rolls.[8] While the sample does not cover the entire population of Andersonville (and probably never can given the lack of complete records), it comes close. An estimated 45,000 men passed through Andersonville (United

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[5]The data are available at http://www.cpe.uchicago.edu and were collected by a team of researchers led by Robert Fogel. The sample of 35,570 represents roughly 1.3 percent of all whites mustered into the Union Army and 8 percent of all regiments that comprised the Union Army. Ninety-one percent of the sample consists of volunteers, with the remainder evenly divided between draftees and substitutes. The data are based upon a 100 percent sample of all enlisted men in 331 randomly chosen companies. The sample is limited to 303 companies because complete data are not yet available on all 331 companies.

[6]Linkage to the 1860 census reveals that the sample is representative of the Northern population of military age in terms of 1860 real estate and personal property wealth and in terms of literacy rates.

[7]Because of the system of prisoner exchange (and the hope that it would be revived), the South had an incentive to record information on men who were captured.

[8]A searchable version of the database is available on-line as part of the Soldiers and Sailors system at http://www.itd.nps.gov/cwss.

States War Department (1880-1901), Series II, Vol. VIII, p. 789). The data provide capture and death dates (but are not always complete) and also information on the soldier's name, rank, regiment, and company. I collected a random sample of roughly 1,000 men who lived to 1900 (as ascertained from their pension records) from this database.

The men in both samples are linked to their military service records, their pension records (including the detailed reports of the examining surgeons), and the 1860, 1880, and 1900 censuses. Linkage procedures for the 1880 census differ across samples. The Andersonville sample used information on residence and on family members from the pension records, whereas the linkage for Fogel sample was done purely on name and state (or country) and year of birth. The Andersonville sample is also linked to the 1870 census, as is a small subsample of the sample of Union Army soldiers (i.e. those soldiers enlisting in DC, Delaware,Iowa, Kansas, Maine, Minnesota, Missouri, Ohio, and West Virginia).[9] The military service records provide detailed information on men's wartime illnesses, wounds, and captivity. The pension and surgeons' records provide information on date of death after the war, cause of death, and chronic conditions after the war. These records also contain information on wartime illnesses (for POWs either before or after captivity). The censuses provide socioeconomic and demographic information, including on wealth (in 1860 and 1870), occupation, and marital status.

I restrict both samples to men who survived to 1900 and who were on the pension rolls by 1900.[10] I also restrict the samples to men who have information on date of death and, among former POWs, men who have complete information on capture dates. This leaves 11,025 non-POWs, 889 POWs from the Fogel sample, and 893 POWs from the Andersonville sample. I lose 65 men in the Fogel sample (4 of them POWs) in the regressions because of missing information on age in 1900. Information on cause of death is available for 47 percent of the Fogel sample and for

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[9]See Costa and Kahn (2007a) for details on the 1880 sample and Lee (2005) for details on the 1870 sample.

[10]Because information on cause of death comes from the pension records, men are not at risk to die until they are on the pension rolls.

50 percent of the Andersonville sample. Cause of death is more commonly available when there was a surviving spouse. Causes of death is often vague, e.g. "heart disease." I therefore examine the combined category of heart disease, as well as the more specific categories of stroke, valvular heart disease and pericarditis, and the combined category of angina, atherosclerosis, arteriosclerosis, coronary occlusion, coronary thrombosis, myocarditis, and endocarditis. Fleming (1997) and Finlayson (1985) argue that mentions of angina, atherosclerosis, coronary occupation, and coronary thrombosis may represent ischemic heart disease and I will refer to this combined category as ischemic heart disease though it may not necessarily be equivalent to ischemic heart disease today.

The detailed records of the examining surgeons provide information on various chronic conditions, symptoms, and signs. Patient accounts, physical examination, and diagnostic information from surgeons' exams were used to determine the existence of medical conditions based on diagnostic criteria. These criteria, determined by actively practicing physicians knowledgeable in medical history, were derived from modern medical knowledge while recognizing the limitations of 19th and early 20th century medicine. The primary limitation is that examining surgeons were constrained by what they could detect through sight, touch, feel, and smell. For example, the records describe heart murmurs and their location. Valvular heart disease is diagnosed from a murmur in the aortic or mitral valve as noted in the records. The examining surgeons diagnosed arteriosclerosis by feeling whether the arteries had hardened. Arteriosclerosis therefore refers to peripheral arteriosclerosis and could be either atherosclerosis, an associated disease (such as diabetes), or local inflammation. The examining surgeons also noted whether the pulse was irregular or bounding and the presence of arrythmia, tachycardia, or bradycardia. The examining surgeons were unable to detect any of the conditions that required modern diagnostic equipment, such as hypertension.[11] However, the team of current physicians who reviewed the data reported that if in

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[11]Shell shock, combat fatigue, and post-traumatic stress (all names for the same phenomenon in different wars) were not recognized as disorders either during or after the Civil War (see Hyams, Wignall, and Roswell 1996 for a history of PTSD). The records therefore provide little information on psychiatric disorders.

the field, with no diagnostic equipment, they could not do any better. (For a detailed discussion of potential biases in the surgeons' exams see Costa 2000, 2002). Surgeons records are available for over 95 percent of the men in my sample. These records are less likely to be found both for the severely wounded and for men who applied for a pension on the basis of age rather than a chronic condition.

I examined men's time spent in captivity in six month intervals and classified men either as being POWs for a lengthy time during the period of no prisoner exchanges (non-exchange POWs), that is captured July 1863 to July 1864, or as being POWs earlier or later, that is captured prior to July 1863 or captured July 1864 or later (exchange period POWs). These measures reflect both time spent in captivity and conditions at the time of capture. Although men captured in July 1864 might not be exchanged until December, they faced only about a month of Andersonville at its worst (and some came in with money or goods) and were less likely to develop scurvy, a sign of severe malnutrition.

Table 1 shows that the prevalence of scurvy as mentioned in either wartime records or the later surgeons' reports was higher among men who were captured July 1863 to July 1864 than among men who were captured earlier, later, or were never captured. In the Fogel sample wartime records mention scurvy for 11 percent of all non-POWs, 14 percent of POWs captured before July 1863, 16 percent of POWs captured after June 1864 and 23 percent for POWs captured between July 1863 and July 1864. The reports of the examining surgeons mention scurvy for 1 percent of non-POWs, 2-5 percent of POWs captured before July 1863 or after June 1864, and 8 percent of POWs captured between July 1863 and July 1864. The Andersonville sample also yields differential scurvy rates by time of capture, but with higher prevalence rates. (Within the Fogel sample the roughly 100 men who were at Andersonville at its worst had somewhat higher prevalence rates for wartime scurvy mentions than the men in the Andersonville sample, but slightly lower surgeons' certificates mentions of scurvy. The results are not shown.)

This analysis combines the Fogel and the Andersonville samples to obtain a larger working sample. Compared to the POWs in the Fogel sample, the men in the Andersonville sample were more likely to have been prisoners in the non-exchange period, were more likely to have enlisted in 1863 or later than in 1861 or 1862, were younger, and were more likely to have been laborers at enlistment. Compared to exchange period POWs, non-exchange period POWs were more likely to be laborers at enlistment and less likely to be 1864 enlistees.

Although among survivors to 1900 men who were POWs in any time period were more likely than non-POWs to be on the pension rolls before 1890, former POW status by itself did not entitle men to a pension. In 1900, former non-exchange period POWs received an average pension of $11.80 per month whereas non-POWs received one of $11.20 per month. Because controlling for pension amount did not affect the coefficients on POW status, pension amount is not included in the control variables.

Compared to non-POWs, POWs were more likely to be volunteers, to have enlisted earlier (1862), to be slightly better off, and to be from companies that experienced higher death rates, as might be expected from men who were captured in the field. (However, having been recently wounded did not predict POW camp survival, perhaps because those who made it to a POW camp were those less severely wounded.) The results of a probit in which the dependent variable is POW status and the independent variables are individual economic and demographic characteristics and the number of men in the company who were ever wounded or who ever died shows that these two company characteristics were the main predictors of POW status (Costa and Kahn 2007b). POWs are not necessarily a random sample of soldiers on unobservables either. If a subset of men "fight to the death" I would never see them in a POW camp. Thus, I am less likely to sample the most ideological men.

The veterans used in the analysis were all alive and on the pension rolls in 1900. Because the data are drawn from administrative records and the only source of death information is from the

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pension rolls, the sample cannot be used to investigate morbidity and mortality immediately after the war. Those on the pension rolls immediately after the war were the most disabled (and men imprisoned at Andersonville were more likely to be on the rolls than other ex-POWs). Studies of mortality are possible only once the Union Army pension program becomes a universal old-age and disability program.[12]

4   Empirical Strategy

I examine the effect of POW status on mortality subsequent to 1900, among men both younger than age 65 and those older than 64, by running Cox hazard models of the form

$$h(t) = h_0(t)\exp(\beta_e(\text{Exchange POW}) + \beta_{ne}(\text{Non-exchange POW}) + \beta_s\text{SES} \qquad (1)$$

where $h_0(t)$ is the baseline hazard. Differences in the specifications are in the socioeconomic variables used. The initial specification includes only those SES variables describing socioeconomic status prior to or at enlistment, i.e. dummy variables equal to one if the veteran was laborer at enlistment, if the veteran enlisted in a city of 50,000 or more, if the veteran had personal property wealth in 1860, if the veteran was found in the 1860 census, and enlistment year dummies (1862, 1863, 1864-5, with 1861 as the omitted dummy).[13] Later specifications control for subsequent socioeconomic status (dummy variables equal to one if the veteran was a laborer in 1870, if the veteran owned property in 1870, if the veteran was a laborer in 1880, if the veteran owned property in 1900, and if the veteran was married tin 1900) to determine if the effects of POW status are mediated through socioeconomic status.

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[12]World War II POWs held by the Japanese experienced higher mortality rates for at least 8 years after the war whereas those held by the Germans did not. Excess mortality was due to tuberculosis and to trauma, including suicide, with the highest mortality rates among the youngest men (Cohn and Cooper 1954; Nefzger 1970; Keehn 1980).

[13]Additional socioeconomic variables were investigated, including whether the veteran was a private (privates faced higher mortality rates in POW camps than non-privates), but none of these were statistically significant and did not affect the other coefficients.

I examine the effects of POW status on mortality from all heart disease and from valvular heart disease and pericarditis, stroke, and from the combined category of ischemic heart disease, myocarditis, and endocarditis using a competing risks hazard model for both age groups of men. Other causes of death are assumed to be independent.

I also examine the effects of POW status on cardiovascular morbidity in 1900 using a series of probit models of the form

$$\text{Morbidity} = \beta_0 + \beta_e(\text{Exchange POW}) + \beta_{ne}(\text{Non-exchange POW}) + \beta_s\text{SES} + u \qquad (2)$$

where the morbidity measures are arteriosclerosis, irregular heart beat, presence of murmurs, and valvular heart disease. The SES variables describing socioeconomic status prior to or at enlistment are the same ones used in Equation 1.

I examine the effect of POW status on socioeconomic status of men alive in 1900 by running probit regressions of the form,

$$\text{SES} = \beta_0 + \beta_e(\text{Exchange POW}) + \beta_{ne}(\text{Non-exchange POW}) + \beta_s(\text{Past SES}) + u \qquad (3)$$

where the dependent SES measures are the same ones used in Equation 1.

5 POW Status and Mortality

5.1 Results

The left panel of Figure 1 shows the Kaplan-Meier hazard rates for men less than 65 years of age in 1900 for no exchange period POWs, POWs captured either early or late, and non-POWs. The right panel, for men older than 64 in 1900, shows that compared to non-POWs and POWs captured either early or late, POWs captured during the no exchange period were longer-lived.

Figure 2 shows the hazard rates for death from any type of cardiovascular disease by age group in 1900 among men with information on cause of death. Regardless of age group, no exchange period POWs were more likely to die of heart disease than non-POWs and POWs captured either early or late.

POWs captured during the non-exchange period and younger than age 65 in 1900 were 1.1 times likelier to die than non-POWs, controlling only for characteristics at enlistment and personal property wealth in 1860 (see Table 2). POWs captured during the exchange period faced the same odds of death as non-POWs. Controlling for laborer status in 1880 and property ownership and marital status in 1900 does not change the results. Neither does controlling for wartime illnesses and wounds and mentions of scurvy in the surgeons' exams.[14] The coefficient on an interaction between POW status and wartime rheumatic fever was not statistically significant (results not shown). Controlling for arteriosclerosis, valvular heart disease, heart murmurs, and irregular heart rate in 1900 did not affect the coefficients on POW status (results not shown), suggesting that the effects of POW status on mortality do not operate through conditions already observed in 1900.

The last six regressions in Table 2 show that the excess mortality of POWs captured during the non-exchange period and younger than 65 in 1900 comes from heart disease. POWs captured during the non-exchange period were 1.2 times as likely to die of heart disease as non-POWs, even controlling for laborer status in 1880, property ownership and marital status in 1900, and wartime illnesses and wounds. POWs captured during the non-exchange period were 9 times more likely to die of valvular heart disease (the caveat is that the number of deaths known to be from valvular heart disease was small), 1.3 times more likely to die of stroke, and 1.8 times more likely to die of the combined category of ischemic, myocarditis, and endocarditis than non-POWs. These cause of death effects do not arise from the sample being restricted to men with a cause of death: the fourth regression yields similar results as the first three regressions on the entire sample.

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[14]Even with no controls for type of POW, a mention of scurvy was not a statistically significant predictor of death.

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Among men older than 64, POWs captured during the non-exchange period were 0.9 times as likely to die as non-POWs (see Table 3). However, as control variables are added, the effect becomes statistically insignificant. The excess of non-POWs deaths comes partially from the combined category of ischemic, myocarditis, and endocarditis and also from pneumonia and influenza (not shown), however, none of the effects were statistically significant. Non-exchange POWs were more likely than POWs to die of other heart conditions, but the effects are statistically significant only for valvular heart disease (where the number of cases is small). There is evidence of unobserved heterogeneity (frailty) among men older than 64, but not among men younger than 65. Using a gompertz specification and assuming the unobserved heterogeneity, θ, has a gamma distribution yielded a $\chi^2(01)$ of 0.10 and 19.27 for the test of significance of θ for men less than age 65 and greater than age 64, respectively. However, explicitly controlling for this heterogeneity did not affect the hazard ratios on POW status.

Tests of the proportional hazard assumption in the regressions in Tables 2 and 3 showed that it was not met for the first three regressions. (It was met for all other regressions, except for all heart conditions with controls for later socioeconomic status. Testing the proportional hazard models on the basis of the Schoenfeld residuals for men younger than 65 yielded $\chi^2(11) =$16.64, 12.26, 29.86, 34.50, 11.74, 12.11, 7.80, respectively for each of the last 7 specifications.) However, the alternate strategies of stratifying and restricting the sample to a 15 year observation period yielded very similar results and satisfied the proportional hazards assumption. When the samples were restricted to a 15 year observation period, the hazard ratio on no exchange period POW became 1.182 ($\hat{\sigma}^2$=0.067) for men younger than 65 and 0.817 ($\hat{\sigma}^2$=0.079) for men older than 64. (Testing the proportional hazard models on the basis of the Schoenfeld residuals after regressions in which the controls were enlistment characteristics and 1860 personal property wealth yielded a $\chi^2(11)$ of 13.93 for men less than age 65 and one of 13.75 for men older than 64.) When the entire sample period was used, the proportional hazards assumption failed to hold for POWs who

were exchanged, for men who enlisted in a large city, and for men found in the 1860 census. I therefore re-ran the specifications, omitting 1860 personal property wealth from the specification, and stratifying on city size and whether the POW was an exchange period POW. I found that the hazard rate on non-exchange period POW was 1.088 ($\hat{\sigma}^2$=0.043) for men younger than age 65 and testing the Schoenfeld residuals yielded a $\chi^2(7)$ of 11.17.

5.2 Explanations

Potential explanations for the high survival rates of ex-POWs older than 64 in 1900 include mortality selection in the POW camp and mortality selection when men were in their fifties and early sixties. There is suggestive evidence that high POW camp mortality rates among older men killed the frail, perhaps those with latent chronic conditions while in the army, leaving a more robust population. Among non-POWs who survived to 1900, 11 men had brothers (as determined from the 1850 and 1860 censuses) who had been POWs. Average year of death for the 7 men with a brother who had survived the worst POW period was 1920 compared to 1910 for the 4 men without a survivor brother. Although the difference is statistically significant, the small number of men makes the evidence only suggestive.

There is no evidence that high mortality rates when men were in their fifties and early sixties left a more robust older population. When I took the sample of men younger than 65 in 1900 and restricted the sample to those alive in 1 910 and examined their mortality experience after 1910, I found that POWs during the non-exchange period were still 1.1 times less likely to die than non-POWs (results not shown).

Comparing non-exchange POWs with non-POWs of all ages in 1900 reveals a non-linear relationship between age at captivity and older age mortality. I restricted the sample to non-exchange POWs and non-POWs and created a set of five dummy variables indicating whether the veteran had been captured prior to age 20, at age 20-24, 25-29, 30-34, and age 35 or older. Controlling for

age in 1900, size of enlistment city, laborer status at enlistment, native-born, and year of enlistment dummies, I found that the hazard ratios on the captivity dummies were 0.981 ($\hat{\sigma}^2$=0.080), 1.109 ($\hat{\sigma}^2$=0.057), 1.083 ($\hat{\sigma}^2$=0.075), 0.894 ($\hat{\sigma}^2$=0.091), and 0.807 ($\hat{\sigma}^2$=0.128). Within the Fogel sample, the death rate for men at Andersonville was 35 percent for men younger than 20, 30 percent for men in their twenties, and 54 percent for men age 30 or older.

An explanation for the poor survival rate of former non-exchange period POWs younger than age 65 at captivity is scarring. Scarring implies that ex-POWs who faced a lower mortality risk should do better. Costa and Kahn (2007b) found that those with kin at Andersonville, as proxied by the number of men in the company with the same last name, were more likely to survive. Among the men who survived to 1900 and who had been imprisoned when conditions were at their worst, those who had had kin in the camp (as proxied by at least one man with the same last name in the company), faced an odds of death of 0.809 ($\hat{\sigma}^2$=0.074) compared to men with no kin, controlling for age in 1900 (full Cox hazard results not shown). Having had kin in the camp was not a statistically significant predictor of older age mortality among men who had been in POW camps when conditions were better.

Another explanation, unrelated to scarring, for the worse survival outcomes of non-exchange period POWs younger than 65 is that the characteristics that enabled men to survive captivity hurt their survival chances at older ages. One potential candidate for such a characteristic is height. The tall were less likely to survive POW camps because all men received the same size ration and the tall need more food (Costa and Kahn 2007b). In very large samples, the tall are less likely to die of cardiovascular disease (Waaler 1984). However, the camp survival advantage of being short was small (Costa and Kahn 2007b) and controlling for height in the regressions did not affect the coefficients on POW status (results not shown).

6 POW Status and Cardiovascular Morbidity

Among men who were younger than 65, having been a POW during the non-exchange period increased the probability of having murmurs and valvular disease in 1900 by 0.04 compared to non-POWs for each condition (see Table 4). Men who were POWs during the exchange period were not statistically different from non-POWs. There was no change in the probability of arteriosclerosis or irregular heart beat. However, controlling for later life socioeconomic status and wartime health conditions, both non-exchange and exchange period POWs were statistically significantly more likely to have an irregular heart beat (results not shown). The coefficients on exchange period and non-exchange period POW were 0.047 ($\hat{\sigma}$ =0.020) and 0.039 ($\hat{\sigma}$ =0.020). POW status did not predict any cardiovascular conditions among men older than 64.

7 POW Status and Socioeconomic Outcomes

Among men younger than 65 in 1900, POWs in 1870, regardless of when they were captured, were more likely to be laborers and less likely to have any personal property wealth, controlling for laborer status at enlistment and personal property wealth in 1860 (see Table 5). By 1880, POWs captured during the exchange period resemble non-POWs. Their laborer status is similar in 1880, as is property ownership in 1900. However, POWs captured during the no exchange period were more likely to be laborers in 1880 and were less likely to own property in 1900 than non-POWs. POW status had no effect on marital status in 1900 (or 1880, not shown).

POW status did not have a statistically significant effect on the occupational, home ownership, and marital status of men older than 64 (see Table 6). However, former POWs, regardless of when they were captured, were less likely to have personal property wealth in 1870. POWs captured when the exchange system had stopped were more likely to be laborers than non-POWs in both 1870 and 1880, but the effect is not statistically significant. The effects of POW status on property

ownership in 1900 are positive, but statistically insignificant.

8   Conclusion

Using data on Union Army soldiers, I found that imprisonment as a POW had very heterogeneous effects on older age outcomes, depending on age at captivity. Men above age 30 at captivity when conditions were at their worst were more likely to die in captivity but 35 years after the end of the war the survivors had a lower mortality rate than non-POWs or POWs imprisoned when conditions were better and the same morbidity rate and socioeconomic outcomes. Thirty-five years after the end of the war, men below age 30 at captivity had worse socioeconomic outcomes, were more likely to die of cardiovascular and cerebrovascular disease, and faced greater cardiovascular morbidity.

The mortality findings for men below age 30 at captivity are consistent with studies of Japanese held World War II POWs who faced different social, physical, disease, and medical environments but who also experienced severe malnutrition and even worse psychological stress, (Dent et al. 1989; Page and Brass 2001; Page and Ostfeld 1994), suggesting that there are certain common sequelae of starvation and the stress of imprisonment. The mortality findings for men above age 30 at captivity may differ from those of Japanese held World War II POWs because when conditions in Civil War camps were at their worst, mortality rates from disease and malnutrition were higher than in Japanese POW camps, potentially leading to greater mortality selection in Civil War camps. Mortality effects may need to be very high for selection effects to operate: the mortality rate for men above age 30 at Andersonville was 54 percent compared to one of 30 percent for men in their twenties.

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Table 1: Prevalence of Scurvy by Sample and POW Status

| | Wartime Mention | | Mentioned by Surgeons | |
	%	N	%	N
Fogel sample				
Non-POWs	10.6	11,025	1.3	10,503
All POWs	18.3	889	5.2	842
POWs captured before July 1863	13.5	230	1.8	222
POWs captured July 1863-June 1864	22.9	380	7.7	352
POWs captured after June 1864	16.1	279	4.9	268
Andersonville sample	21.7	893	19.5	878
captured July 1863-June1864	28.5	523	22.9	516
captured after June 1864	12.2	370	14.6	362

The sample consists of all veterans alive and on the pension rolls in 1900, with full captivity date information. Scurvy mentioned by the surgeons is noted post-war.

Table 2: Effect of POW Status on Overall and Heart Disease Mortality, Men Younger than 65 in 1900

	Entire Sample			Known Cause of Death						
	Overall Mortality			All Causes	Heart Disease			Val-vular	Stroke	Is-chemic
Dummy=1 if Non-POW										
POW, Captured Early or Late	0.991	1.016	1.017	1.031	1.041	1.042	1.020	4.970 ‡	1.058	1.244
	(0.040)	(0.041)	(0.042)	(0.058)	(0.088)	(0.090)	(0.088)	(1.870)	(0.161)	(0.238)
POW, No Exchange	1.089†	1.110‡	1.110†	1.137†	1.211†	1.191†	1.127	9.028‡	1.296*	1.845‡
	(0.043)	(0.044)	(0.047)	(0.062)	(0.098)	(0.099)	(0.099)	(2.995)	(0.182)	(0.310)
Pre- and Enlistment SES Controls	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y
Post-war SES Controls	N	Y	Y	N	N	Y	Y	N	N	N
Wartime Health and Scurvy Controls	N	N	Y	N	N	N	Y	N	N	N
Number of failures	9394	9394	9394	4879	2124	2124	2124	54	649	376
Observations	9394	9394	9394	4879	4879	4879	4879	4879	4879	4879

The sample consists of all veterans alive and on the pension rolls in 1900, with full information on dates of captivity and age in 1900. Regressions are Equation 2 in the text and are from a Cox regression. The table gives hazard ratios. Standard errors are in parentheses. The symbols ‡, †, and * indicate significance at the 1, 5, and 10 percent level, respectively.

Table 3: Effect of POW Status on Overall and Heart Disease Mortality, Men Older than 65 in 1900

	Entire Sample			Known Cause of Death						
	Overall Mortality			All Causes	Heart Disease			Val-vular	Stroke	Is-chemic
Dummy=1 if Non-POW										
POW, Early or Late Capture	0.951	0.976	0.983	0.922	1.083	1.121	1.174	8.918 ‡	0.333*	1.095
	(0.072)	(0.075)	(0.076)	(0.107)	(0.195)	(0.204)	(0.216)	(5.413)	(0.195)	(0.475)
POW, No Exchange	0.841 †	0.871*	0.888	0.906	1.177	1.139	1.193	7.702 ‡	1.502	0.604
	(0.068)	(0.072)	(0.076)	(0.113)	(0.212)	(0.212)	(0.233)	(5.208)	(0.468)	(0.364)
Pre- and Enlistment SES Controls	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y
Post-war SES Controls	N	Y	Y	N	N	Y	Y	N	N	N
Wartime and Scurvy Health Controls	N	N	Y	N	N	N	Y	N	N	N
Number of failures	3344	3344	3344	1438	536	536	536	16	148	80
Observations	3344	3344	3344	1438	1438	1438	1438	1438	1438	1438

The sample consists of all veterans alive and on the pension rolls in 1900, with full information on dates of captivity and age in 1900. Regressions are Equation 2 in the text and are from a Cox regression. The table gives hazard ratios. Standard errors are in parentheses. The symbols ‡, †, and * indicate significance at the 1, 5, and 10 percent level, respectively.

Table 4: Effect of POW Status on Heart Disease Morbidity in 1900 by Age Group

	Younger than 65				Older than 64			
	Arteriosclerosis	Irregular Heart Beat	Valvular Heart Disease	Heart Murmur	Arteriosclerosis	Irregular Heart Beat	Valvular Heart Disease	Heart Murmur
Dummy=1 if Non-POW								
POW, Captured Early or Late	-0.005	0.042†	0.011	0.022	0.018	-0.001	0.054	0.048
	(0.005)	(0.020)	(0.017)	(0.019)	(0.018)	(0.038)	(0.034)	(0.037)
POW, No Exchange	0.005	0.030	0.041†	0.039†	-0.021	-0.001	0.056	0.013
	(0.006)	(0.020)	(0.017)	(0.019)	(0.013)	(0.041)	(0.037)	(0.039)
Pre- and Enlistment SES Controls	Y	Y	Y	Y	Y	Y	Y	Y
Pseudo R^2	0.019	0.006	0.002	0.003	0.022	0.001	0.003	0.003
Mean Dependent Variable	0.023	0.465	0.212	0.339	0.051	0.507	0.234	0.339
Observations	9394	9394	9394	9394	3344	3344	3344	3344

The sample consists of all veterans alive and on the pension rolls in 1900, with full information on dates of captivity and age in 1900. The regressions are Equation 3 in the text. The table gives derivates. Standard errors are in parentheses. The symbol † indicates significance at the 5 percent level.

Table 5: Effect of POW Status on SES Outcomes, Men Younger than 65 in 1900

|	Dependent Variable: Dummy=1 if				
	1870 Laborer	1870 Wealth Holder	1880 Laborer	1900 Home Owner	1900 Married Man
Dummy=1 if					
Non-POW					
POW and Captured					
Early or late	0.114‡	-0.892‡	-0.008	-0.008	-0.000
	(0.037)	(0.058)	(0.023)	(0.021)	(0.015)
No exchange period	0.149‡	-0.823‡	0.084‡	-0.042†	0.011
	(0.034)	(0.059)	(0.024)	(0.021)	(0.014)
Pre- and Enlistment					
SES Controls	Y	Y	Y	Y	Y
Pseudo R^2	0.064	0.354	0.039	0.022	0.01
Mean Dependent Variable	0.296	0.825	0.24	0.631	0.862
Observations	1352	1352	3339	7871	7871

The sample consists of all veterans alive and on the pension rolls in 1900, with full information on dates of captivity and age in 1900. Regressions are Equation 1 in the text and come from a probit. The derivatives are given in the table. Standard errors are in parentheses. The symbols † and ‡ indicate significance at the 5 and 1 percent level, respectively.

Table 6: Effect of POW Status on SES Outcomes, Men Older than 64 in 1900

	Dependent Variable: Dummy=1 if				
	1870 Laborer	1870 Wealth Holder	1880 Laborer	1900 Home Owner	1900 Married Man
Dummy=1 if					
Non-POW					
POW, Captured Early or Late	-0.010	-0.640‡	0.041	-0.013	0.003
	(0.053)	(0.073)	(0.046)	(0.041)	(0.035)
POW, No Exchange	0.089	-0.574‡	0.061	0.019	0.037
	(0.055)	(0.079)	(0.045)	(0.043)	(0.036)
Pre- and Enlistment					
SES Controls	Y	Y	Y	Y	Y
Pseudo R^2	0.211	0.474	0.083	0.042	0.035
Mean Dependent	0.183	0.905	0.206	0.617	0.756
Observations	410	410	1160	2709	2709

The sample consists of all veterans alive and on the pension rolls in 1900, with full information on dates of captivity and age in 1900. Regressions are Equation 1 in the text and come from a probit. The derivatives are given in the table. Standard errors are in parentheses. The symbols † and ‡ indicate significance at the 5 and 1 percent level, respectively.
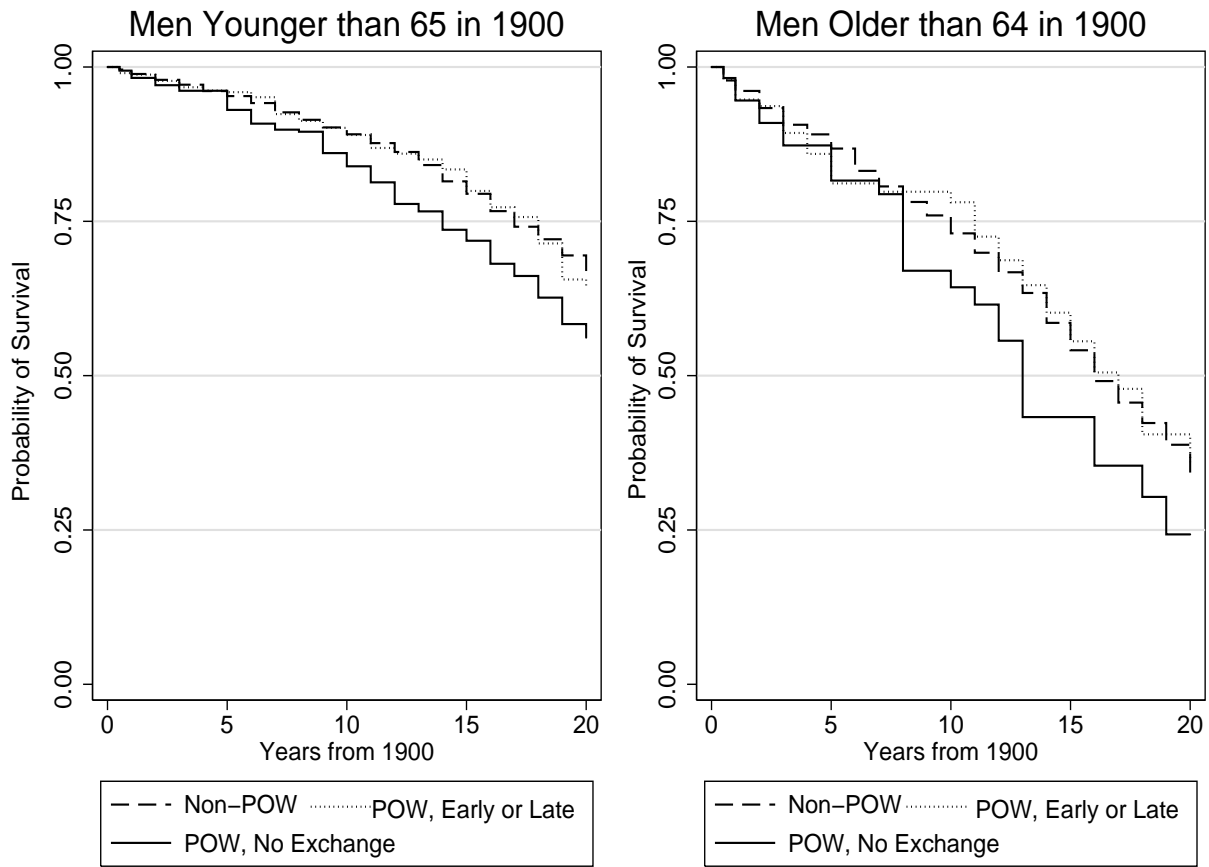
Figure 1: Survival Rates, by Age Group in 1900 and Former POW Status
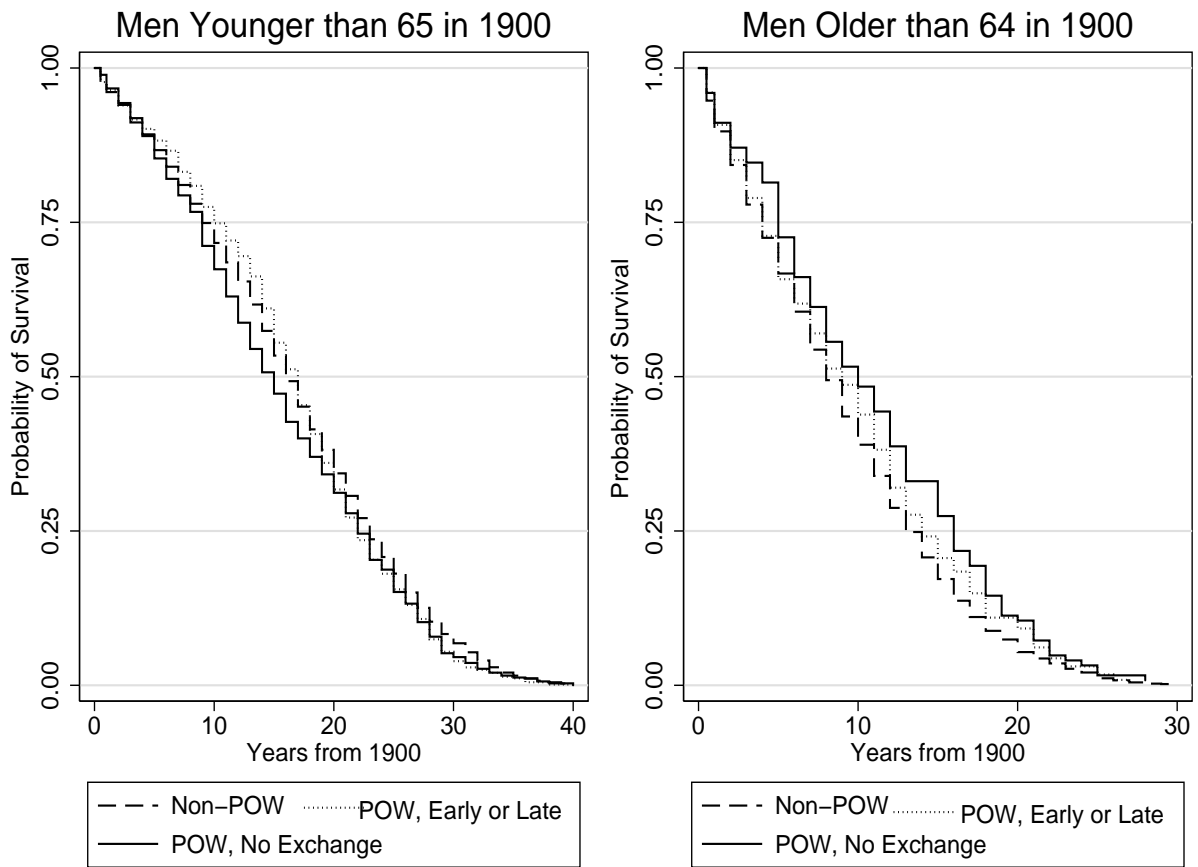
The figures give Kaplan-Meier survival curves. The sample consists of all veterans alive and on the pension rolls in 1900, with full information on dates of captivity and age in 1900.

Figure 2: Cardiovascular Disease Survival Rates, by Age Group in 1900 and Former POW Status

The figures give Kaplan-Meier survival curves for men with known causes of death. Deaths from other causes are treated as censored and are assumed to be in independent. The sample consists of all veterans alive and on the pension rolls in 1900, with full information on dates of captivity and age in 1900.