



California Center for Population Research
University of California - Los Angeles

Health, Wartime, Stress, and Unit Cohesion: Evidence from Union Army Veterans

Dora L. Costa
Matthew E. Kahn

CCPR-023-08

December 2008
Latest Revised: September 2008

California Center for Population Research
On-Line Working Paper Series

HEALTH, WARTIME STRESS, AND UNIT COHESION: EVIDENCE FROM UNION ARMY
VETERANS

by

Dora L. Costa
UCLA and NBER
costa@econ.ucla.edu

Matthew E. Kahn
UCLA and NBER
mkahn@ioe.ucla.edu

September 4, 2008

We both gratefully acknowledge the support of NIH grants R01 AG19637 and R01 AG027960. Dora Costa also gratefully acknowledges the support of NIH grant P01 AG10120. We thank Louis Cain, Chulhee Lee, Louis Nguyen, Nevin Scrimshaw, Avron Spiro, and seminar participants at RAND, the University of California, Berkeley, UCLA, USC, the NBER Aging Summer Institute, and the conference “Economic History: Retrospect and Prospect” for comments.

Health, Wartime Stress, and Unit Cohesion: Evidence from Union Army Veterans

Abstract

JEL Classification: I12, Z13

We find that veterans of the Union Army who faced greater wartime stress (as measured by higher battlefield mortality rates) experienced higher mortality rates at older ages, but that men who were from more cohesive companies were statistically significantly less likely to be affected by wartime stress. Our results hold for overall mortality, mortality from ischemic heart disease and stroke, and new diagnoses of arteriosclerosis. Our findings represent one of the first long-run health follow-ups of the interaction between stress and social networks in a human population in which both stress and social networks are arguably exogenous.

Dora L. Costa
UCLA Department of Economics
9272 Bunche Hall
Los Angeles, CA 90095-1477
and NBER
costa@econ.ucla.edu

Matthew E. Kahn
UCLA Institute of the Environment
La Kretz Hall, Suite 300
Los Angeles, CA 90095-1496
and NBER
mkahn@ioe.ucla.edu

1 Introduction

Economic and epidemiological research has linked social networks to health. People who report themselves to be socially isolated, both in the number and quality of their personal relationships, face a higher mortality risk from all causes. In particular, they face a greater risk of death from several infectious, neoplastic, and cardiovascular diseases (e.g. Seeman 1996; Cohen et al. 1997; Caspi et al. 2006; Kroenke et al. 2006; Reynolds and Kaplan 1990; Hawkey et al. 2006; Lett et al. 2007). Social networks affect health through both biological and social pathways. Friends provide physical, cognitive, and economic assistance, health information, and the peer pressure needed to re-enforce good health habits (Aizer and Currie 2004; Miguel and Kremer 2007; Christakis and Fowler 2007; Rao, Mobius, and Rosenblat 2007; Gresenz, Rogowski, and Escarce 2007). Social networks may positively affect cellular immune response (Thomas et al. 1985; Cohen et al. 1992) and neuroendocrine functioning (Seeman et al. 1994); feelings of social isolation may even be linked to alterations in the activity of genes that drive inflammation, the first response of the immune system (Cole et al. 2007).

Many studies have investigated how social networks mediate the effects of stress (e.g. Lett et al. 2007; Bolger and Amaril 2007; House, Landis, and Umberson 1988). Stress is associated with several chronic diseases, particularly cardiovascular disease. Experimentally induced stress leads to atherosclerosis and hypertension in primates and mice (Henry 1977). Job stress leads to greater risk of cardiovascular disease (Marmot and Wilkinson 1999) and working in low-control jobs raises mortality risk (Amick et al. 2002). Vietnam veterans with post-traumatic stress syndrome face higher overall and cardiovascular mortality (Boscarino 2006b) and a higher prevalence of cardiovascular disorders, including myocardial infarctions (Boscarino 1997). Subramanian, Elwert, and Christakis (2008) present suggestive evidence that the well-known effect of widowhood on older age mortality is modified by the neighborhood concentration of widowed individuals. Perceptions of social support are associated with better adjustment to stressful events (e.g. Lett

et al. 2007; House, Landis, and Umberson 1988), even though actual social support often is not correlated with better adjustment (e.g. Bolger and Amaril 2007).

Social networks could either mitigate or accentuate the effects of stress. They could mitigate the effects of stress through beneficial effects on psychological and physical well-being. But, they could accentuate the effects of stress if the initial trauma involves the death of friends or family (e.g. the well-established effect of death of a spouse on the mortality of a survivor) or if well-intentioned support efforts remind individuals of the initial trauma or of their lack of coping ability.

This paper examines whether social networks mitigate or accentuate the effects of wartime stress on older age mortality and morbidity using a unique longitudinal database of veterans of the Union Army in the American Civil War, 1861-5. We study how the interaction between unit cohesiveness and combat mortality affected older age all-cause mortality, mortality by cause, and morbidity. In most studies (with the exception of animal studies, e.g. Thaker et al. 2006; Capitanio et al. 1998; Levine and Mody 2003; Lyons, Ha, and Levine 1995, Cohen et al. 1992), social networks are not exogenous and individuals choose their social networks. Thus those who are socially isolated may be socially isolated because they are in poor health. In our Civil War setting, cohesiveness is arguably exogenous (and varied considerably across companies) because of the way companies were formed and because companies were rarely replenished. In addition, combat mortality varied across units because it depended on where a unit was in a battle.

The Civil War provides a unique opportunity to examine how social networks influence the long-term effects of stress. The Civil War was unique. During the Vietnam War individuals were rotated in and out of units. During World War II units were replenished with new men and wounded men who had recuperated were sent to new units. A researcher would therefore need to collect data not just on individuals and their initial units but also on all units the individual served in. In addition, privacy concerns might make obtaining records difficult. A difficulty in studying World I is that many of these records were destroyed in a fire. Only the Civil War therefore enables

us to examine at low cost the interaction between unit cohesion and wartime stress on older age mortality.

2 Effects of Wartime Trauma

Among many causes, stress can result from war, natural disaster, divorce, lack of control on the job, or even disrupted sleep patterns. The brain responds to stress by cognitively assessing the threat potential and then orchestrating a physiological and behavioral response in which stress hormones are released.

This release of stress hormones may trigger several psychiatric disorders, including post-traumatic stress syndrome (PTSD). After the initial trauma, a person will re-experience it in the form of recurrent memories, dreams, feelings that the event is recurring, and psychological and physical distress when reminded of the trauma. It is the recurrent nature of the trauma that may contribute to higher mortality. PTSD in particular has been identified as the intervening variable linking trauma and subsequent mortality. For example, Boscarino (2006a) finds that among Vietnam veterans combat exposure was not associated with mortality once he controlled for PTSD. Allostatic load (the cumulative wear and tear that results from repeated efforts to adapt to stressors over time), as measured by a composite index of biological risk factors, is particularly elevated in women with PTSD (Glover, Stuber, and Poland 2006). Data on World War II and Korean War POWs suggest that PTSD symptoms follow a pattern of immediate onset and gradual decline, followed by increasing symptom levels at older ages (Port, Engdahl, and Frazier 2001). A forty year follow-up of World War II soldiers who had seen fierce fighting found that 18 percent currently had PTSD (Sutker, Allain, and Winstead 1993). Additional psychiatric disorders that can follow trauma include depression and anxiety disorders. However, among Vietnam veterans depression was not consistently associated with mortality once other factors were controlled for (Boscarino

2008).

How individuals respond to the initial trauma will depend on their personality type, financial resources, and social network. Social support may have a “direct” effect on psychological and physical well-being that is independent of stress levels (Andrews, Tennant, Hewson, and Vaillant 1978; Solomon, Mikulincer, and Hobfoll 1986). Alternatively or in addition to the direct effect, social support may have a “stress-buffering” effect, that is it aids stress resistance under high-stress conditions but has little effect under low-stress conditions (Hobfoll and Walfisch 1984; Wilcox 1981).

During wartime men in a more cohesive company may code social support as physical safety (Shay 2002: 210) and be less likely to develop PTSD. Capt. Frank Hollinger of the 19th USCT wrote, “I have always found comforting in battle the companionship of a friend, one in whom you had confidence, one you felt assured would stand by you until the last” (quoted by Hess 1997: 117). Alternatively, friends might provide emotional consolation. They could provide exoneration for killing, the promise that they would not be forgotten, and decrease men’s fear of death. After World War II, Audie Murphy (1949: 158) wrote of his experiences, “At this moment the grave seems merely an open door that divides us from our comrades.”

Quantitative evidence on social networks and PTSD comes from the Yom Kippur War where the unexpected attack led men to fight in different tank crews. PTSD rates immediately after the war were higher among men who did not end up with their usual tank crew in the chaos to get to the front (Gal 1986: 217; Belenky, Noy, and Solomon 1987). But a tank crew is more likely to lose either all or none of its men than an infantry company. Among the men of an infantry company, being in a more cohesive company might worsen the effects of stress if men lose those who are close to them.

Shell shock, combat fatigue, and post-traumatic stress (all names for the same phenomenon in different wars) were not recognized as disorders during the Civil War (see Hyams, Wignall, and

Roswell 1996 for a history of PTSD). Not flinching under enemy bullets was viewed as a test of manhood and those who failed courted contempt (McPherson 1997: 77-78). Nonetheless, Oliver Wendell Holmes could write in 1864, “I tell you that many a man has gone crazy since the end of this campaign began from the terrible pressure on mind & body” (quoted in McPherson 1997: 165). McPherson (1997: 166) describes how “after eighteen hours of continuous combat at the Bloody Angle of Spotsylvania, a Union lieutenant the next morning found enemy soldiers piled three or four deep in the trenches, mostly dead, but one Rebel sat up praying at the top of his voice and others were gibbering in insanity.”

Historical accounts are relatively silent on whether Civil War trauma left a lasting effect. Most diaries cease with the end of the war. Dean (1997) argued for a lasting effect of wartime trauma but based his finding on a biased sample of Civil War veterans who were in soldiers homes. Drawing from the same database as our study, Pizarro, Silver, and Prause (2006) found that the fraction of the company who died during the war affected the probability of cardiac and gastrointestinal disease among the survivors, but not older age mortality. However, because half of all Civil War deaths were from disease, they may be measuring the effects of wartime illness on later disease outcomes.¹ They may not find an effect of company wartime mortality on older age mortality because of the way they constructed their sample.²

We observe men during the war and then from about 1900 onwards, when roughly 90 percent of all white veterans were on the pension rolls and therefore enter our dataset (Costa 1998: 198). Short of insanity that led to commitment to an asylum (of which there are very few cases), senile

¹Most of their cardiac disease category consists of heart disease that resulted from rheumatic fever (Costa 2000) and is therefore unlikely to be linked to wartime trauma. Pizarro, Silver, and Prause (2006) did not control for specific wartime illnesses, even though these data are available.

²They restricted the sample to men who lived until 1890 or later but did not restrict the sample to men who were on the pension rolls by 1890. Because all date of death information comes from the pension rolls, men are not at risk to die until they are on the pension rolls. Analysis time therefore needs to begin at the time men enter the pension rolls. In addition, Pizarro, Silver, and Prause (2006) may not find an effect of the fraction of the company who died on older age mortality if because of soldiers’ changing companies, the fraction of the company who died becomes a poor indicator of a veteran’s wartime experience.

dementia, or the aftermaths of stroke, we cannot observe psychiatric disorders. (Because PTSD was accepted into the diagnostic literature only in 1980, we never observe who had PTSD.) We also cannot observe any health effects of trauma until men entered the pension rolls. However, we would expect stronger effects of psychiatric disorders on mortality from natural causes and on cardiovascular health at older ages because cardiovascular problems increase with age.

3 Empirical Framework

We will begin by examining the effect of wartime stress on older age mortality. We will investigate the use of different measures of battlefield stress such as fraction of the company dying of wounds, number of the company dying of wounds, number of the regiment killed in action, maximum number of men in a regiment killed in a single engagement and logarithms of these quantities to account for potential non-linearities. These measures will reflect differences in the strength of ties between men (presumably stronger in a company than in a regiment because companies were more of a local neighborhood), in the nature of the trauma (a single, big traumatic event versus a repetition of the trauma), and in localized stress levels. Although the regiment was sent into battle as a unit, companies could have different battle experiences because their locations on the battlefield differed.

Using the year 1900 as our baseline period (when the majority of veterans were between ages 55-64), we estimate a Gompertz hazard model of time until death in years

$$h(t) = \lambda \exp(\gamma t) \tag{1}$$

$$\lambda = \exp(\beta_s s + \beta_c c + \beta_x x) \tag{2}$$

where s is a measure of stress and c is a measure of company cohesion.³ If $\beta_s > 0$ (or if the hazard

³We obtain similar coefficients using a Cox proportional hazards model but prefer the Gompertz because we can

ratio $\exp(\beta_s) > 1$) then stress increases older age mortality. The vector of control variables, x , includes age in 1900, and measures of wartime experience and socioeconomic status.

If stress affects the mortality experience of only those veterans who develop psychiatric disorders, then our estimate of β_s captures both the probability of developing psychiatric disorders (an unobservable) and the effects of psychiatric disorders on older age mortality. Because men could avoid wartime stress by straggling (remaining in the rear, an unobservable) we may underestimate the effect of our stress measure on older age mortality because it no longer proxies for men's actual war-time experience. Men could also avoid wartime stress by deserting. Many of these men are lost to follow-up because they never returned to their units and were ineligible for the pension which provides a record of their death. If deserters were inherently at greater risk to develop psychiatric disorders then the effect of stress will be underestimated.

Once we have established which measure of stress best predicts older age mortality, our primary specification becomes the Gompertz hazard model of time until death in years

$$h(t) = \lambda \exp(\gamma t) \tag{3}$$

$$\lambda = \exp(\beta_s s + \beta_c c + \beta_{sc}(s \times c) + \beta_x x) \tag{4}$$

where the only difference with our previous specification is the inclusion of the interaction term between company cohesion and wartime stress. We are thus allowing for a heterogeneous treatment effect; that is, while $\beta_s > 0$, we are allowing for $\beta_{sc} < 0$ (or a hazard ratio that is less than one).

One of the challenges of quantification is the definition of company cohesion. Most studies of unit cohesion use either answers to questionnaires or information on how long the unit was together (e.g. Solomon, Mikulincer, and Hobfoll 1987). We rely on our past work for a revealed preference approach to creating an index of cohesion (Costa and Kahn 2003a,b). There is a large literature (summarized in Costa and Kahn 2003a) showing that people in more diverse communities are less

predict survivor proportions from the model output.

willing to join organizations, volunteer, pay taxes for public goods, and vote than people in more homogenous communities. A soldier’s unit, the men he lives and fights with, has always been his community. A World War I German soldier wrote, “The company is the only truly existent community. This community allows neither time nor rest for a personal life. It forces us into its circle for life is at stake” (Shils and Janowitz 1948). During the Civil War, this unit consisted of the roughly 100 men in a soldier’s company.

Roughly ten percent of all Union Army soldiers deserted. They were more likely to desert if they were from more diverse companies, controlling for individual characteristics (including time of enlistment), ideology, and morale (including recent company deaths) (Costa and Kahn 2003b). We therefore call a company cohesive if it was less diverse in ethnicity, occupation, and age. As we will discuss in more detail later, we construct an index of company cohesion based on the coefficients on company heterogeneity in a desertion regression. Our index weights thus give the effect of company heterogeneity on desertion (and hence arguably cohesion) controlling for other factors, including commitment to the cause and breakdown in combat (as proxied by recent company deaths).

We investigate why stress and social networks might affect health using several strategies. First, we examine whether stress and social networks affected socioeconomic status at older ages and investigate how our results change if we control for socioeconomic status and health at older ages. Second, assuming that causes of death are independent, we estimate a competing risks model of mortality by cause. Finally, we estimate probit equations to examine what chronic condition veterans who survived to 1915 developed between 1900 and 1915. That is, we estimate

$$\Pr(C = 1) = \Phi(\beta_s s + \beta_c c + \beta_{sc}(s \times c) + \beta_x x) \quad (5)$$

where C is an indicator variable for a specific chronic condition, s is our measure of stress, c is our measure of company cohesion, and x is our vector of control variables. If wartime stress increases

the probability of developing a chronic condition, then $\beta_s > 0$. If company cohesion mitigates the effects of wartime stress, then $\beta_{sc} < 0$.

4 Data

Our dataset is based on a sample of roughly 35,000 white men in 303 Union Army infantry companies collected under the auspices of Robert Fogel and available at the website of the University of Chicago's Center for Population Economics (<http://www.cpe.uchicago.edu>).⁴ These soldiers were linked to the 1850, 1860, 1880, 1900, and 1910 census and to pension records to create a longitudinal dataset.

The Union Army pension program began in 1862 to provide assistance to soldiers wounded during the war. In 1890 the program was expanded and any disability entitled a veteran to a pension, doubling the number of veterans on the rolls overnight. Old age was considered a disability in practice and then became a disability by law in 1907.

Detailed medical records are available for veterans because any veteran who applied for a pension or who wished for a pension increase was examined by a board of surgeons. Ninety-three percent of all men on the pension rolls had an exam. Those who applied on the basis of age were less likely to have an exam and in the analysis are assumed not to have any chronic conditions.

The examining surgeons could note a chronic condition, a symptom, or a sign through sight, touch, feel, and smell. Cardiovascular conditions illustrate how their examinations can be used. We diagnose valvular heart disease from a murmur in the aortic or mitral valve noted in the exam. We diagnose congestive heart failure as concurrent edema, cyanosis, and dyspnea. 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

⁴The full sample contains 39,000 men in 331 companies but the full sample was not available at the time of analysis. Data on almost 6,000 men in 56 companies of black troops, also available at this website, were not used because relatively few black troops saw intensive action.

disease (such as diabetes), or local inflammation. The examining surgeons also noted whether the pulse was irregular or bounding and the presence of arrhythmia, tachycardia, or bradycardia. The examining surgeons were unable to detect any of the conditions that required modern diagnostic equipment, such as hypertension. However, the team of physicians who reviewed the data reported that if in 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).

We restrict our sample to men who were alive and at least age 50 in 1900 and who were on the pension rolls. This leaves us with a sample size of 12,119 men. We also restrict the sample to men for whom we have complete enlistment and discharge information and men who did not change companies. Men who changed companies were most commonly those promoted to officer or the original volunteers who enlisted for 90 days and then re-enlisted in another company when their term was up. These two sample restrictions reduce the sample size to 7,721 men.

We constructed several variables. Our measures of battlefield stress are both on the company level based on the full sample of 35,000 men and on the regiment level based on a database compiled from Frederick Dyer's *A Compendium of the War of the Rebellion* and from William Fox's *Regimental Losses in the American Civil War, 1861-1865*.⁵ These enable us to examine the fraction of the company dying of wounds (including deaths from septicimia resulting from wounds sustained in action), the fraction of the company that died of wounds, the number in the company that died of wounds, the number in the regiment killed in action, the maximum number killed in a single engagement, and logarithms of the above. As seen in Figure 1, during the war some men were in companies with high death rates from wounds, while others were in companies with no deaths from wounds.

We treat our measures of stress as exogenous, that is we assume that the characteristics of the company did not determine whether it was placed in a more dangerous place on the battlefield.

⁵This regiment level database is available from <http://www.cpe.uchicago.edu>.

The unit that went into battle was the regiment and regiments contained both homogeneous and diverse companies. The order of battle was often determined by when a specific regiment arrived and if it had had time to rest after its long march.

The more cohesive companies were the ones that were the most homogeneous in birthplace, occupation, and age (Costa and Kahn 2003b). We therefore constructed an index of company cohesion using the hazard ratios from a hazard model predicting time until desertion to weigh company birthplace, occupation, and age diversity.⁶ That is, our index for company j , I_j is

$$I_j = \alpha_B B + \alpha_O O + \alpha_A A \quad (6)$$

where B is birthplace fragmentation, O is occupational fragmentation, A is the coefficient of variation of age multiplied by 100, and the α s are the hazard ratios (details are available in the Data Appendix). We then labeled as “highly cohesive” a company that was below the median on our index.⁷ We also run specifications using the individual components of this index to examine which of our diversity measures is the best predictor of older age mortality. However, we lose information by using the individual components of our index of cohesion rather than our index.

Why was there diversity in companies? All regiments were formed locally. Costa and Kahn (2008: 57-73) examined Civil War diaries and letters to identify six sources of diversity within companies. The volunteer infantry regiments consisted of 10 companies, each containing roughly 100 men, commanded by a captain and two lieutenants, who were often volunteer officers drawn from state militias, men of political significance, or other prominent men in the community. At the beginning of the war, men would enlist with one or several friends but rarely with fifty. Once companies were full, they would take no more men, and friends would need to find another company

⁶Our hazard model of time until desertion controls for potential confounders such as year of enlistment, company mortality rates, ideology in county of enlistment, and own characteristics such as ethnicity, socioeconomic status, and volunteer status.

⁷Although company composition might change with desertions and deaths, we obtain similar results when we use company heterogeneity at the end rather than the beginning of the war.

or regiment. Men's eagerness to get to the front led them to pick regiments thought to be departing soon. And they quickly left regiments that were late in departing, even enlisting in the regiment of another state. Later in the war, when the new recruits were not so eager, men might enlist in a distant town to receive a large bounty, adding to company diversity. Although a company was generally not replenished with new men when disease, military casualties, and desertions whittled down its numbers, some states added new recruits to existing regiments and regiments whose members' three year terms were up were reconstituted with veterans and new men. Finally, the need to travel to recruiting stations increased company diversity. Farmers and farmers' sons had to travel to town to enlist. Small towns could not raise an entire company, so their men would enlist elsewhere and do so only with a few friends. Commissioned officers were responsible for finding their own men and often had to scour the entire state to fill their regiments. James Garfield, who later became president, traveled throughout Ohio holding revival-style meetings.

Statements in soldiers' diaries and letters indicate that they were thrown together with strangers. Amos Stearns, who enlisted with five of his friends, lamented "Life in the army was very different from life at home. In one place we could choose our companions and those we wished to associate with, but in the army how different" (Kent 1976: 214-5). One soldier wrote home, "We have a remarkable civil and Religious company. . . . i think it is a providencial circumstance that I enlisted in this company for I hear that there is desperate wickedness in very regiments i came so near enlisting in" (letter of David Close, Nov. 4, 1862, 126th Ohio Volunteer Infantry, Company D (<http://www.frontierfamilies.net/family/DCletters.htm>)).

Our control variables are age in 1900, measures of individual wartime stress (whether the soldier was wounded and how severely, POW status, and illnesses experienced), rank, if the soldier was in a support position, household personal property wealth in 1860, occupation at enlistment, country of birth, if volunteer, population of city of enlistment, if deserter, if illiterate, and fixed effects for the state served. In addition, we examine if our findings change when we control for

occupation in 1900, marital status in 1900, home ownership in 1900, and BMI circa 1900.

Table 1 illustrates how the characteristics of men change from the starting sample of 35,000 soldiers to the war survivors and the pensioners in 1900. Men who survived the war were more likely to be deserters, non-POWs, non-farmers, support and officers, rich, and short. A slight wound rather no wound or a severe wound increased men's chances of surviving. Men were more likely to have survived the war if they were not ill during the war, particularly from measles, typhoid, a respiratory condition, and smallpox. Men in the more cohesive companies were less likely to survive because they were also the men who were less likely to desert. Although ten percent of soldiers deserted, roughly 13 percent of the surviving soldiers had ever deserted.

Compared to the war survivors, men who were alive and on the pension rolls in 1900 were the non-deserters (deserters were not eligible for a pension), men who enlisted in smaller cities, farmers, the native-born and the German-born (all groups with lower post-war mortality rates), and support and officers. Men who were wounded in the war were more likely to be on the pension rolls, as were men who were ill during the war, particularly from cardiovascular causes, smallpox, typhoid, malaria, fever, gastric causes, sunstroke, rheumatic fever, measles, and diarrhea. Even controlling for all other factors, company cohesion was not a statistically significant predictor of being alive and on the pension rolls in 1900.

We cannot observe what happened to men after the war ended and before they entered the pension rolls. Until men entered the pension rolls they were not at risk of dying (because the pension is our only source of information on date of death) and only after 1890, when pensions become widely available to all veterans, is the sample representative of the veteran population. We begin our analysis in 1900 because linkage to the census provides us with information on socioeconomic variables. If the most traumatized men died before 1900 we may underestimate the effects of wartime stress on older age mortality. When we searched for all men known to have survived the war in the 1880 census, we did not find that either wartime stress or company cohesion

or the interaction term between wartime stress and company cohesion were statistically significant predictors of being found in the 1880 census and therefore arguably of mortality.

5 Results

We test three hypotheses. The first is that wartime stress increases mortality at older ages. The second is that company cohesion mitigates the effect of wartime stress on older age mortality. Company cohesion could buffer the effects of stress if men in more cohesive companies are less likely to develop psychiatric disorders or it could accentuate the effects of stress if men lose those who are very close to them. The third hypothesis is that company cohesion reduces the effects of wartime stress on the probability of developing cardiovascular disease.

5.1 Wartime Stress and Mortality

Survival probabilities were slightly higher among men in companies with low death rates from wounds than among men in companies in the top death rate decile (see Figure 2). Table 2 shows that the fraction of the company dying of wounds has a statistically significant effect on older age mortality controlling for many individual characteristics, company cohesion, and state of regiment fixed effects. The fraction of the company dying of wounds has a larger impact on older age mortality than other measures of wartime stress and the linear form of the specification illustrates this best. The number of men in the company dying of wounds has a stronger effect than the number killed in the regiment. The fraction of men in the company dying of illness also has an effect on older age mortality but the effect is not as strong as the fraction killed; when both are entered simultaneously in the regression, statistical significance on both coefficients disappears. However, the coefficients are jointly statistically significant. We prefer to use the fraction of men dying of wounds rather than the total fraction dying in the war because the fraction of men dying

of illnesses might be a proxy for unobserved individual illness. We find some evidence that the maximum number of men killed in a single battle has an effect on older age mortality, but the effect is non-linear and not as strong as the fraction of the company dying of wounds. When we included both the logarithm of the maximum number of men killed in a single battle and the total number of men killed in the regiment, statistical significance on both wartime stress measures disappeared but the coefficients were jointly statistically significant. We found no evidence that the fraction of the company dying of wounds might proxy for unobserved wartime own probability of being wounded. When we included whether or not a veteran claimed a wound on his pension application (presumably anyone who was wounded had every incentive to claim this on the pension), the coefficient on the fraction of the company wounded remained roughly similar at 2.119 ($\hat{\sigma} = 0.779$).

We investigated whether there were any interaction effects between personal characteristics and wartime stress. When we controlled for age at enlistment we found that men younger than 17 faced an odds of dying 1.286 ($\hat{\sigma} = 0.176$) greater than men age 17-40.⁸ But we found no evidence of any interaction effects between age and wartime stress. We also found no interaction effects between whether a soldier was wounded in the war and wartime stress. We find some suggestive evidence that the Irish and the British were more adversely affected by wartime stress than the native-born and the Germans less so.

We tested whether our estimates of the impact of wartime stress depended on the timing of enlistment and days served. Because companies that were formed earlier fought in the war longer, our measures of wartime stress are greater for companies organized earlier. When we included dummy variables indicating year of enlistment in our specification, we found that the hazard ratio on the fraction of the company dying of wounds fell from 2.139 to 1.647 ($\hat{\sigma} = 0.655$), statistically indistinguishable from one. The hazard ratios on the year of enlistment dummies were individually statistically indistinguishable from one. However, the hazard ratios on the fraction of the company

⁸When we controlled for ten year birth cohorts, the hazard ratio on age less than 17 becomes 1.237 ($\hat{\sigma} = 0.161$), still statistically significant at the 10 percent level.

dying of wounds and the dummy variables indicating year of enlistment were jointly statistically significantly different from one ($\chi^2(2) = 4.91$). Similarly, when we controlled for days served, we found that the hazard ratio on the fraction of the company dying of wounds fell from 2.139 to 1.900 ($\hat{\sigma} = 0.766$) and that the hazard ratio on days served was statistically significantly different from one. But the hazard ratio on the fraction of the company dying of wounds and the hazard ratio on days served were jointly statistically significantly different from one ($\chi^2(2) = 5.69$). We found no evidence of interaction effects between days served and wartime stress.

5.2 Wartime Stress, Cohesion, and Later Outcomes

As seen in Figure 3, when company death rates from wounds were above the median, men who were in cohesive companies had higher older age survival probabilities than men who were in companies where cohesion was low. However, when company death rates from wounds were below the median, company cohesion did not affect survival probabilities, suggesting that social support aids stress resistance in high-stress situations but has no effect in low-stress conditions.

Table 3 shows that using the fraction of the company dying of wounds, either in a linear or logarithmic form, and controlling for individual characteristics and state of regiment fixed effects, being in a cohesive company reduced the negative effects of stress on older age mortality. (Using other measures of wartime stress reveals a similar pattern.) Cohesion by itself did not affect older age mortality. An increase of 0.01 in the fraction of the company dying of wounds increased the odds of dying by 0.06 for men in an un-cohesive company and by 0.01 for men in a cohesive company. The mean of the predicted mortality probabilities for every individual would have been 50.5 percent if all men had been in an un-cohesive company and 49.9 percent if they had been in a cohesive company. The effects of stress and company cohesion remain roughly the same when we control for socioeconomic status in 1900, marital status in 1900, and health (as proxied by the Body Mass Index or BMI) in 1900. We did not find that wartime stress predicts these control

variables, suggesting that we are not uncovering the effects of wartime stress as mediated through socioeconomic status and health in 1900.

We investigated a quartile rather than a median split on company cohesion. Although we lost power, we found that the difference that mattered was that between the two top and two bottom quartiles.

We may overestimate the extent to which company cohesion mitigates wartime stress if men who sought out more cohesive companies were the men most likely to develop psychiatric disorders or if we are confounding the effects of cohesion with those of home community. However, we did not find that county of enlistment characteristics had any predictive power. Finding a company that was a good match was largely a matter of luck. Until the first battle, soldiers could not know if any of their comrades or officers were good soldiers. The volunteers were all civilians.

We estimated models of unobserved heterogeneity because some individuals might be more susceptible to stress than others. Assuming that unobserved heterogeneity can be modeled as having a gamma distribution, tests revealed evidence of heterogeneity. However, our basic results remained unchanged. The hazard ratios were 6.844 ($\hat{\sigma} = 3.298$) on the fraction of the company killed, 1.038 ($\hat{\sigma} = 0.388$) on the dummy variable for a cohesive company, and 0.154 ($\hat{\sigma} = 0.098$) on the interaction between company killed and the dummy variable for a cohesive company.

We also investigated whether the degree of company cohesiveness depended on the timing of enlistment. Although the weights used for our index of cohesion control for the timing of enlistment, we may not fully capture that early companies were the more cohesive companies. We therefore ran our specification including a dummy for early enlistment (enlistment in 1861) and the interaction of this dummy with our measure of company cohesion. The resulting hazard ratios on our dummy for enlistment and on the interaction term of this dummy with our measure of company cohesion were, respectively, 0.989 ($\hat{\sigma} = 0.084$) and 2.901 ($\hat{\sigma} = 3.365$), both statistically indistin-

guishable from one.⁹ The hazard ratios on the fraction of the company wounded and the interaction term between the fraction of the company wounded and our measure of company cohesion were, respectively, 4.858 ($\hat{\sigma} = 2.254$) and 0.167 ($\hat{\sigma} = 0.097$), both highly statistically significant from one.

We also investigated different specifications of the hazard. Again, our basic results remained unchanged. For example, when we estimated a Weibull model we obtained hazard ratios of 5.248 ($\hat{\sigma} = 2.082$) and 0.196 ($\hat{\sigma} = .101$) on the fraction of the company dying of wounds and on the interaction term between the fraction of the company killed and the dummy variable for a cohesive company.

Table 4 shows that the most statistically significant interaction effects on mortality are seen in mortality from ischemic heart disease and stroke. An increase of 0.01 in the company killed increases the odds of dying from ischemic heart disease and stroke by 0.50 for men in un-cohesive companies, but by only 0.02 for men in high cohesion companies. The effects on the odds of dying of a respiratory disease are even larger, but the standard errors are very large as well. No interaction effects between wartime stress and company cohesion were found on other causes of death.

When we examined the probability of men who lived until 1915 developing a heart condition between 1900 and 1915, we found that men in a more cohesive company were less likely to develop arteriosclerosis and bounding pulse than men in a less cohesive company (see Table 5). The predicted probability of developing heart disease is 0.138 for men in an un-cohesive company and 0.127 for men in a cohesive company. The predicted probability of developing bounding pulse is 0.081 for men in an un-cohesive company and 0.068 for men in a cohesive company. Bounding pulse is often associated with high blood pressure or fluid overload. Although we present results for arteriosclerosis and bounding pulse in the same table, they can be almost considered mutually exclusive physical findings because arteriosclerosis is accompanied by occlusive disease and hence

⁹Using enlistment prior to 1863 made little difference to our results.

decreased pulse on examination. We found that while 27 percent of men with arteriosclerosis in 1915 had ever had bounding pulse in an examination, 73 percent of them had ever had a weak pulse in an examination.

There were no differences in the rates of developing valvular heart disease, congestive heart failure, or other heart rate abnormalities by company cohesion. As a falsification exercise, we examined the effects of company cohesion and stress on the probability of developing a hernia between 1900 and 1915 among men who did have a hernia in 1900 (4430 observations). Because hernias result from unusual pressure on the abdomen such as that due to heavy lifting, obesity, or even aging, there should be no effects and we found none. The derivatives on the fraction of the company that was wounded and the interaction term between the fraction of the company that was wounded and company cohesion were -0.061 ($\hat{\sigma} = 0.126$) and -0.036 ($\hat{\sigma} = 0.159$), respectively.

Company cohesion mitigates the effects of stress largely through birthplace and age cohesion effects. When we ran a specification in which we included measures of whether a company was below the mean in birthplace, occupation, and age homogeneity and interacted these with our measures of wartime stress, we found that the coefficient on the fraction of the company dying of wounds was 1.968 ($\hat{\sigma} = 0.784$) and that the interaction terms on birthplace and age homogeneity were 0.789 ($\hat{\sigma} = 0.209$) and 0.707 ($\hat{\sigma} = 0.164$), respectively. In contrast, the interaction term on occupation homogeneity was 2.150 ($\hat{\sigma} = 0.423$).

6 Conclusion

We found that being in a more cohesive company reduced the negative, long-term consequences of wartime stress. The strongest effect of wartime stress on older age mortality and on the probability of developing specific conditions was observed for ischemic and stroke causes of death and the probability of developing arteriosclerosis and bounding pulse. Men in more cohesive companies

were less likely to develop cardiovascular disorders later in life when exposed to wartime stress than men in less cohesive companies. We suspect that men under stress who developed cardiovascular conditions later in life suffered from undiagnosed psychiatric conditions. Why might men who faced similar stress levels but were in more cohesive companies never develop psychiatric conditions? We can rule out a positive effect of peers on risk avoidance. Because men in more cohesive companies were less likely to desert, they also faced a higher risk of death. Having a social support network may have led men to reappraise battlefield threats or provided emotional consolation after the battle. Although our results are derived from a past population, it is one of the few human populations to provide us with measures of stress, of long-run outcomes, and of exogenous social networks.

Studies of the negative health effects of stress in recent populations have attracted a great deal of attention (e.g. Geronimus 1992, Marmot and Wilkinson 1999). Stress was by no means the most important predictor of older age mortality in past populations. For example, the negative impact of growing up in large city (where infectious diseases were common and nutritional status was poorer) was much greater than the effect of wartime stress (Costa and Lahey 2005). Although our results suggest that declines in psychological stress played at most a small role in long-run improvements in elderly health and longevity, stress may become a relatively more important factor in developed country populations as early life conditions have improved.

Data Appendix

Our index of cohesion is based on the regression described below (see Costa and Kahn 2003b for further details).

We use a time-varying independent competing risk hazard model to estimate days from entry into the company (muster-in) until the first case of desertion. We treat men as censored if they

died, were discharged, changed companies, became prisoners of war, or were missing in action. Our estimated hazard, $\lambda(t)$, is

$$\lambda(t_i) = \exp(x'_I\beta_I + x'_C\beta_C + x'_D\beta_D + x'_M\beta_M)\lambda(t) \quad (7)$$

where I indexes the individual variables, C indexes the community variables, D indexes the ideology variables, M indexes the morale variables (some of which are time-varying) and $\lambda(t)$ is the baseline hazard which we assume to be Weibull. The survival function thus takes the form, $\exp(-\lambda_j t_j^p)$ for subject j, where p is the duration dependence parameter and can be interpreted as representing whether men who were in the war longer became more or less committed soldiers. We cluster on companies.

Our independent variables are:

Socio-economic and Demographic Characteristics

1. **Occupation.** Dummy variables indicating whether at enlistment the recruit reported his occupation as farmer, artisan, professional or proprietor, or laborer. Farmers' sons who were not yet farmers in their own right would generally report themselves as farmers.
2. **Birth place** Dummy variables indicating whether at enlistment the recruit reported his birth place as the US, Germany, Ireland, Great Britain, or other.
3. **Age at enlistment.** Age at first enlistment.
4. **Height in inches.** Height in inches at first enlistment.
5. **Married in 1860.** This variable is inferred from family member order and age in the 1860 census. This variable was set equal to 0 if the recruit was not linked to the 1860 census.
6. **Log(total household personal property) in 1860.** This variable is the sum of personal

property wealth of everyone in the recruits' 1860 household. This variable is set equal to 0 if the recruit was not linked to the 1860 census.

7. **Missing census information.** A dummy equal to one if the recruit was not linked to the 1860 census. Linkage rates from the military service records to the 1860 census were 57 percent. The main characteristic that predicted linkage failure was foreign birth.
8. **Illiterate.** This variable is from the 1860 census and provides illiteracy information only for those age 20 and older.
9. **Missing illiteracy information.** A dummy equal to one if we do not know whether the recruits was illiterate, either because he was not linked to the 1860 census or because he was less than age 20 in 1860.
10. **Region effects.** Our region dummies are New England, Middle Atlantic, East North Central, West North Central, Border, and West.

Community Characteristics

1. **Birth place fragmentation.** We calculated, by company, the fraction of individuals born in the US in New England, in the Middle Atlantic, in the East North Central, in the West North Central, the Border states, the south, and the west and born abroad in Germany, Ireland, Canada, Great Britain, Scandinavia, northwestern Europe (France, Belgium, Luxembourg, the Netherlands), other areas of Europe, and other areas of the world. Our birthplace fragmentation index, f_i , is then

$$f_i = 1 - \sum_k s_{ki}^2,$$

where k represents the categories and where s_{ki} is the share of men of born in place k in company i .

2. **Occupational fragmentation.** We calculated, by company, the fraction of individuals who were farmers, higher class professionals and proprietors, lower class professionals and proprietors, artisans, higher class laborers, lower class laborers, and unknown. Our occupational fragmentation index is then calculated similarly to our birthplace fragmentation index.
3. **Coefficient of variation for age.** We calculated, by company, the coefficient of variation for age at enlistment.
4. **Population in city of enlistment.** We obtained population in city of enlistment from *Union Army Recruits in White Regiments in the United States, 1861-1865 (ICPSR 9425)*, Robert W. Fogel, Stanley L. Engerman, Clayne Pope, and Larry Wimmer, Principal Investigators. Cities that could not be identified were assumed to be cities of population less than 2,500.

Ideology Variables

1. **Year of muster.** Dummy variables indicating the year that the soldier was first mustered in.
2. **Volunteer.** A dummy equal to one if the recruit was a volunteer instead of a draftee or a substitute.
3. **Percent of vote in 1860 Presidential election.** We obtained by county of enlistment the fraction of the vote cast for Lincoln and for other candidates from *Electoral Data for Counties in the United States: Presidential and Congressional Races, 1840-1972 (ICPSR 8611)*, Jerome M. Clubb, William H. Flanigan, and Nancy H. Zingale, Principal Investigators. Because we cannot attribute a county to each recruit, our categories are percent in county of enlistment voting for Lincoln, other candidate, and unknown.

Morale Variables

1. **Fraction in company dying.** We calculated, by company, the fraction dying overall and the fraction dying (among all men at risk to die) within all half years that each recruit served.

Our means present the fraction dying overall. Our regression results use the time-varying covariate, fraction of men at risk dying during all half years that each recruit served.

2. **Fraction of major Union victories.** This is a time-varying variable that indicates for each half year that the recruit was in the service the fraction of major Union victories to all major battles in that half-year. It takes the value 0 if there were no major battles.

Our cohesion index uses the coefficients on birth place fragmentation, occupation fragmentation, and the coefficients of variation for age. We tested if measures of county-level fragmentation for the male population of military age perform better than our company fragmentation measures. Higher birth place fragmentation in county of enlistment increased desertion rates, but the effect was not statistically significant. We found no effect at all of county-level occupational fragmentation.

We also investigated using alternative measures of birth place and occupational diversity such as percent of own nativity or occupation and concentration ratios. Concentration ratios for birth-place and occupation were collinear but individually a higher concentration ratio significantly decreased the probability of desertion. Measures such as percent of own nativity or occupation are not suited to the Union Army data because there was no dominant ethnic group. However, we found that laborers were more likely to desert if the proportion of laborers in the company was high.

References

- [1] Aizer, Anna and Janet Currie. 2004. "Networks or Neighborhoods? Correlations in the Use of Publicly-Funded Maternity Care in California." *Journal of Public Economics*. 88(12): 2573-85.
- [2] Amick III, Benjamin C, Peggy McDonough, Hong Chang, William H. Rogers, Carl F. Pieper, and Greg Duncan. 2002. "Relationship Between All-Cause Mortality and Cumulative Working Life Course Psychological and Physical Exposures in the United States Labor Market from 1968 to 1992." *Psychosomatic Medicine*. 64: 370-81.
- [3] Andrews, G, C Tennant, DM Hewson, and GE Valiant. 1978. "Life event stress, social support, coping style, and risk of psychological impairment." *Journal of Nervous and Mental Disease*. 166: 307-15.
- [4] Belenky, GL, S Noy, and Z Solomon. 1987. "Battle stress, morale, cohesion, combat effectiveness, heroism and psychiatric casualties: The Israeli experience." In G.L. Belenky (Ed.), *Contemporary Studies in Combat Psychiatry*. Westport, CT: Greenwood Press, Inc.
- [5] Bolger, Niall and David Amarel. 2007. "Effects of Social Support Visibility on Adjustment to Stress: Experimental Evidence." *Journal of Personality and Social Psychology*. 92(3): 458-75.
- [6] Bolger, Niall, Adam Zuckerman, and Ronald C. Kessler. 2000. "Invisible Support and Adjustment to Stress." *Journal of Personality and Social Psychology*. 79(6): 953-61.
- [7] Boscarino, JA. 2008. "Psychobiologic Predictors of Disease Mortality After Psychological Trauma: Implications for Research and Clinical Surveillance." *The Journal of Nervous and Mental Disease*. 196(2): 100-7.
- [8] Boscarino, JA. 2006a. "External-cause mortality after psychologic trauma: the effects of stress exposure and predisposition." *Comprehensive Psychiatry*. 47: 503-14.
- [9] Boscarino, JA. 2006b. "Posttraumatic stress disorder and mortality among U.S. Army veterans 30 years after military service." *Annals of Epidemiology*. 16(4): 248-56.
- [10] Boscarino, JA. 1997. "Diseases among men 20 years after exposure to severe stress: implications for clinical research and medical care." *Psychosomatic Medicine*. 59(6): 605-14.
- [11] Capitanio JP, SP Mendoza, NW Lerche, WA Mason. 1998. "Social stress results in altered glucocorticoid regulation and shorter survival in simian acquired immune deficiency syndrome." *Proceedings of the National Academy of Sciences, USA*. 95: 4714-19.
- [12] Caspi, A, H Harrington, TE Moffitt, BJ Milne, and R Poulton. 2006. "Socially isolated children 20 years later: risk of cardiovascular disease." 160(8): 805-11.

- [13] Christakis, Nicholas A. and James H. Fowler. 2007. "The Spread of Obesity in a Large Social Network over 32 Years." *The New England Journal of Medicine*. 357: 370-79.
- [14] Cohen S, WJ Doyle, DP Skoner, BS Rabin, JM Gwaltney. 1997. "Social Ties and Susceptibility to the Common Cold." *Journal of the American Medical Association*. 277: 1940-44.
- [15] Cohen S, JR Kaplan, J Cunnick et al. 1992. "Chronic social stress, affiliation and cellular immune response in nonhuman primates." *Psycholog Sci*. 4: 301-10.
- [16] Cole, Steve W., Louise C. Hawkey, Jesus M. Arevalo, Caroline Y. Sung, Robert M. Rose, and John T. Cacioppo. 2007. *Genome Biology*. 8(9): R189.1-R189.13. <http://genomebiology.com/2007/8/9/R189>
- [17] Costa, Dora L. 2002. "Changing Chronic Disease Rates and Long-term Declines in Functional Limitation Among Older Men." *Demography*. 39(1): 119-38.
- [18] Costa, Dora L. 2000. "Understanding the Twentieth Century Decline in Chronic Conditions Among Older Men." *Demography*. 37(1): 53-72.
- [19] Costa, Dora L. 1998. *The Evolution of Retirement: An American Economic History, 1880-1990*. Chicago: The University of Chicago Press.
- [20] Costa, Dora L. and Matthew E. Kahn. 2008. *Heroes and Cowards: The Social Face of War*. Princeton, NJ: Princeton University Press.
- [21] Costa, Dora L. and Matthew E. Kahn. 2003a. "Civic Engagement and Community Heterogeneity: An Economist's Perspective." *Perspectives on Politics*. 1(1).
- [22] Costa, Dora L. and Matthew E. Kahn. 2003b. "Cowards and Heroes: Group Loyalty in the American Civil War." *Quarterly Journal of Economics*. 118(2): 519-48.
- [23] Costa, Dora L. and Joanna N. Lahey. 2005. "Becoming Oldest-Old: Evidence from Historical US Data." *Genus*. 61(1): 125-61.
- [24] Dean Jr., Eric T. 1997. *Shook over Hell: Post-Traumatic Stress, Vietnam, and the Civil War*. Cambridge, MA: Harvard University Press.
- [25] Gal, Reuven. 1986. *A Portrait of the Israeli Soldier*. Westport, CT: Greenwood Press.
- [26] Geronimus, AT. 1992. "The weathering hypothesis and the health of African-American women and infants: evidence and speculations." *Ethnicity & Disease*. 2(3): 207-21.
- [27] Glover, Dorie A, Margaret Stuber, and Russell E. Poland. 2006. "Allostatic Load in Women With and Without PTSD Symptoms." *Psychiatry*. 69(3): 191-203.
- [28] Gresenz, Carole Roan, Jeannette Rogowski, and José J. Escarce. "Social Networks and Access to Health Care Among Mexican-Americans. NBER Working Paper 13460. October 2007.

- [29] Hawkey, LC, CM Masi, JD Berry, JT Cacioppo. 2006. "Loneliness is a unique predictor of age-related differences in systolic blood pressure." *Psychology and Aging*. 21(1): 152-64.
- [30] Hess, Earl J. 1997. *The Union Soldier in Battle: Enduring the Ordeal of Combat*. Lawrence, KS: The University of Kansas Press.
- [31] Hobfoll, SE and S Walfisch. 1984. "Coping with a threat to life: A longitudinal study of self concept, social support, and psychological distress." *American Journal of Community Psychology*. 12: 87-100.
- [32] House, James S., Karl R. Landis, and Debra Umberson. 1988. "Social Relationships and Health." *Science*. 241(4865): 540-5.
- [33] Hyams, Kenneth C., F. Stephen Wignall, and Robert Roswell. 1996. "War Syndromes and their Evaluation: From the U.S. Civil War to the Persian Gulf War." *Annals of Internal Medicine*. 125(5): 398-405.
- [34] Kent, Arthur A. (Ed.) 1976. *Three Years with Company K: Sergt. Austin C. Stearns Company K 13th Mass. Infantry (Deceased)*. Cranberry, NJ: Associated University Presses.
- [35] Kroenke CH, LD Kubzansky, ES Schernhammer, MD Holmes, and I Kawachi. "Social networks, social support, and survival after breast cancer diagnosis." *Journal of Clinical Oncology*. 24(7): 1105-11.
- [36] Lett, HS, JA Blumenthal, MA Babyak, DJ Catellier, RM Carney, LF Berkman, MM Burg, P Mitchell, AS Jaffe, and N Schneiderman. 2007. "Social support and prognosis in patients at increased psychological risk recovering from myocardial infarction." *Health Psychology*. 26(4): 418-27.
- [37] Levine S and T Mody. 2003. "The long-term psychobiological consequences of intermittent postnatal separation in the squirrel monkey." *Neurosci Biobehav Rev*. 27: 83-9.
- [38] Lyons, DM, CM Ha, and S Levine. 1995. "Social effects and circadian rhythms in squirrel monkey pituitary-adrenal activity." *Horm Behav*. 29: 177-90.
- [39] Marmot, Michael and Richard G. Wilkinson (Eds.). 1999. *Social Determinants of Health*. Oxford: Oxford University Press.
- [40] Pizarro J, RC Silver, and J Prause. 2006. "Physical and mental health costs of traumatic war experiences among Civil War veterans." *Archives of General Psychiatry*. 63(2): 193-200.
- [41] McPherson, James M. 1997. *For Cause and Comrades: Why Men Fought in the Civil War*. New York and Oxford: Oxford University Press.
- [42] Port, CL, B Engdahl, and P Frazier. 2001. "A longitudinal and retrospective study of PTSD among older prisoners of war." *The American Journal of Psychiatry*. 158(9): 1474-9.

- [43] Miguel, Edward and Michael Kremer. 2007. "The Illusion of Sustainability." *Quarterly Journal of Economics*. 122: 1007-65.
- [44] Murphy, Audie. 1949. *To Hell and Back*. New York: Holt.
- [45] Rao, Neel, Markus Mobius, and Tanya Rosenblat. 2007. "Social Networks and Vaccination Decisions." Unpublished Ms. Harvard University.
- [46] Reynolds P and GA Kaplan. 1990. "Social connections and risk for cancer: prospective evidence from the Alameda County Study." *Behavioral Medicine*. 16(3): 101-10.
- [47] Seeman, Teresa E. 1996. "Social Ties and Health: The Benefits of Social Integration." *Annals of Epidemiology*. 6: 442-51.
- [48] Shay, Jonathan. 2002. *Odysseus in America: Combat Trauma and the Trials of Homecoming*. New York and London: Scribner.
- [49] Shils, Edward A. and Morris Janowitz. 1948. "Cohesion and Disintegration in the Wehrmacht in World War II." *Public Opinion Quarterly*. 12(2): 280-315.
- [50] Solomon, Zahava, Mario Mikulincer, and Stevan E. Hobfoll. 1987. "Objective Versus Subjective Measurement of Stress and Social Support: Combat-Related Reactions." *Journal of Consulting and Clinical Psychology*. 55(4): 577-83.
- [51] Solomon, Zahava, Mario Mikulincer, and Stevan E. Hobfoll. 1986. "Effects of Social Support and Battle Intensity on Loneliness and Breakdown During Combat." *Journal of Personality and Psychology*. 51(6): 1269-76.
- [52] Subramanian SV, F Elwert, and N Christakis. 2008. "Widowhood and mortality among the elderly: The modifying role of neighborhood concentration of widowed individuals." *Social Science and Medicine*. 66(4): 873-84.
- [53] Sutker, PB, AN Allain, Jr., and DK Winstead. 1993. "Psychopathology and psychiatric diagnoses of World War II Pacific theater prisoner of war survivors and combat veterans." *American Journal of Psychiatry*. 150(2): 240-5.
- [54] Thaker, PH, LY Han, AA Kamat, JM Arevalo, R Takahashi, C Lu, NB Jennings, G Armaiz-Pena, JA Bankson, M Ravoori, et al. 2006. "Chronic stress promotes tumor growth and angiogenesis in a mouse model of ovarian carcinoma." *Nat Med*. 12: 939-44.
- [55] Thomas, PD, JM Goodwin, JS Goodwin. 1985. "Effect of social support on stress-related changes in cholesterol level, uric acid level, and immune function in an elderly sample." *American Journal of Psychiatry*. 142: 735-7.
- [56] Wilcox, BL. 1981. "Social support, life stress, and psychological adjustment: A test of the buffering hypothesis." *American Journal of Community Psychology*. 9: 371-87.

Table 1: Characteristics of Soldiers, War Survivors, and Pensioners in 1900

	Soldiers		War Survivors		Pensioners in 1900	
	Mean	Std Dev	Mean	Std Dev	Mean	Std Dev
Age at enlistment	25.751	7.603	25.761	7.588	24.045	6.228
Log(population) in enlistment city	8.622	1.884	8.659	1.896	8.219	1.513
Dummy=1 if at enlistment						
Farmer						
Professional or proprietor	0.075	0.264	0.079	0.270	0.059	0.235
Artisan	0.200	0.400	0.205	0.404	0.173	0.378
Laborer	0.212	0.409	0.220	0.415	0.160	0.366
Unknown	0.007	0.085	0.008	0.087	0.007	0.084
Log(household personal property wealth) in 1860	2.676	4.902	2.718	4.886	2.987	4.792
Dummy=1 if						
US-born						
British	0.039	0.193	0.041	0.197	0.027	0.161
Irish	0.087	0.282	0.089	0.285	0.036	0.186
German	0.074	0.262	0.076	0.266	0.056	0.231
Other	0.054	0.225	0.056	0.230	0.041	0.199
Height in inches	67.599	2.621	67.557	2.614	67.726	2.568
Dummy=1 if POW who						
captured early in war	0.035	0.184	0.034	0.181	0.035	0.184
captured late in war	0.048	0.214	0.040	0.196	0.045	0.207
Dummy=1 if deserter	0.103	0.304	0.130	0.337	0.046	0.209
Dummy=1 if						
Private						
Support	0.026	0.158	0.027	0.163	0.031	0.173
Commissioned or						
Non-commissioned officer	0.171	0.376	0.174	0.379	0.210	0.407
Dummy=1 if						
Slight wound	0.032	0.175	0.034	0.180	0.043	0.204
Severe wound	0.259	0.438	0.251	0.434	0.321	0.467
Ill during war	0.644	0.479	0.633	0.482	0.732	0.443
Dummy=1 if company cohesive	0.487	0.500	0.482	0.500	0.492	0.500
Fraction company died of wounds	0.038	0.037	0.036	0.036	0.035	0.037

34,941 soldiers, 30,801 war survivors, and 11,921 pensioners. Pensioners are restricted to men on the pension rolls in 1900 and with known date of death. We created an index of company cohesion based on diversity within a company in birthplace, occupation, and age (see the text for details).

Table 2: Effects of Different Stress Measures on Older Age Mortality

	Haz.	Haz.	Haz.	Haz.	Haz.	Haz.	Haz.	Haz.	Haz.
	Rat.	Rat.	Rat.	Rat.	Rat.	Rat.	Rat.	Rat.	Rat.
Fraction company dying	1.530 [†] (0.972)								
Fraction company dying of wounds		2.139 [†] (0.788)		1.747 (0.636)					
Log(fraction company dying of wounds)			1.010 [†] (0.005)						
Fraction company dying of illness			1.310 [†] (0.181)	1.239 (0.172)					
Number in company dying of wounds					1.005 [†] (0.002)			1.003 (0.003)	
Number in regiment killed							1.001* (0.001)	1.001 (0.001)	
Maximum number killed in regiment in single battle									1.000 (0.001)
Log(maximum number killed in regiment in single battle)									1.010 [†] (0.004)
Test of joint significance, $\chi^2(2)$				5.32 (0.070)				5.47 (0.065)	

Hazard ratios are from a Gompertz model of years until death. Restricted to men on the pension rolls in 1900, with known date of death, who did not change companies, and with both muster-in and discharge information. Additional control variables are age in 1900, dummy variables indicating whether the veteran had been wounded slightly in the war, whether he had ever been wounded severely, whether he had been a POW early in the war, and whether he had been a POW late in the war, occupation at enlistment dummy variables (professional or proprietor, artisan, laborer, and unknown, with farmer as the omitted category), country of birth dummy variables (Britain, Ireland, Germany, and other foreign country, with US as the omitted category), dummy variables indicating whether the veteran had been in a support position or had been a commissioned or non-commissioned officer, a dummy variable indicating if the veteran had deserted, the logarithm of household personal property wealth in 1860, a dummy variable indicating that the veteran was illiterate, dummies indicating that information on wealth and on literacy was missing, a dummy variable indicating that the veteran had been a volunteer, wartime disease dummies (typhoid, smallpox, respiratory, rheumatic fever, measles, diarrhea, insanity, malaria, fever, syphilis, gonorrhea, hepatitis, and cardiovascular), and state of regiment fixed effects. Clustered standard errors. The symbols †, ‡, and * indicate that the hazard ratio differs from 1 at the 1, 5, and 10 percent level of statistical significance. 7,494 observations.

Table 3: Effects of Company Cohesion and Stress on Older Age Mortality

	(1)	(2)	(3)	(4)	(5)
	Haz.	Haz.	Haz.	Haz.	Haz.
	Rat.	Rat.	Rat.	Rat.	Rat.
Log(fraction company dying of wounds)		1.020 [‡] (0.007)			
Fraction company dying of wounds	6.347 [‡] (2.809)		6.770 [‡] (3.031)	8.397 [‡] (4.096)	7.848 [‡] (3.846)
Dummy=1 if cohesive	1.035 (0.036)	0.890 [†] (0.046)	1.044 (0.037)	1.047 (0.038)	1.043 (0.037)
Fraction company dying of wounds × Dummy=1 if cohesive	0.161 [‡] (0.094)	0.982* (0.009)	0.156 [‡] (0.091)	0.126 [‡] (0.074)	0.133 [‡] (0.079)
Includes 1900 SES, marriage	N	N	Y	Y	Y
Restricted to known BMI	N	N	N	Y	Y
Includes BMI	N	N	N	N	Y

Hazard ratios are from a Gompertz model of years until death. Restricted to men on the pension rolls in 1900, with known date of death, who did not change companies, and with both muster-in and discharge information. Additional control variables are age in 1900, dummy variables indicating whether the veteran had been wounded slightly in the war, whether he had ever been wounded severely, whether he had been a POW early in the war, and whether he had been a POW late in the war, occupation at enlistment dummy variables (professional or proprietor, artisan, laborer, and unknown, with farmer as the omitted category), country of birth dummy variables (Britain, Ireland, Germany, and other foreign country, with US as the omitted category), dummy variables indicating whether the veteran had been in a support position or had been a commissioned or non-commissioned officer, a dummy variable indicating if the veteran had deserted, the logarithm of household personal property wealth in 1860, a dummy variable indicating that the veteran was illiterate, dummies indicating that information on wealth and on literacy was missing, a dummy variable indicating that the veteran had been a volunteer, wartime disease dummies (typhoid, smallpox, respiratory, rheumatic fever, measles, diarrhea, insanity, malaria, fever, syphilis, gonorrhea, hepatitis, and cardiovascular), and state of regiment fixed effects. Clustered standard errors. The symbols †, ‡, and * indicate that the hazard ratio differs from 1 at the 1, 5, and 10 percent level of statistical significance. Specifications 1, 2, and 3 contain 7,494 observations. Specifications 4 and 5 contain 6,845 observations.

Table 4: Effects of Company Cohesion and Stress on Different Causes of Death at Older Ages

	All known, excl. violence Haz. Rat.	Ischemic and stroke Haz. Rat.	Other cardiovascular Haz. Rat.	Respiratory Haz. Rat.
Fraction dying wounds	15.859 [‡] (9.635)	50.364 [‡] (71.395)	2.338 (3.133)	132.819* (362.328)
Dummy=1 if cohesive	1.051 (0.052)	1.120 (0.113)	1.040 (0.106)	1.323 (0.202)
Fraction dying wounds × Dummy=1 if cohesive	0.161 [†] (0.125)	0.041* (0.075)	0.684 (1.201)	0.007* (0.022)

Hazard ratios are from a Gompertz model of years until death by cause. Competing causes of death are assumed to be independent. Restricted to men on the pension rolls in 1900, with known date of death, who did not change companies, and with both muster-in and discharge information. Additional control variables are age in 1900, dummy variables indicating whether the veteran had been wounded slightly in the war, whether he had ever been wounded severely, whether he had been a POW early in the war, and whether he had been a POW late in the war, occupation at enlistment dummy variables (professional or proprietor, artisan, laborer, and unknown, with farmer as the omitted category), country of birth dummy variables (Britain, Ireland, Germany, and other foreign country, with US as the omitted category), dummy variables indicating whether the veteran had been in a support position or had been a commissioned or non-commissioned officer, a dummy variable indicating if the veteran had deserted, the logarithm of household personal property wealth in 1860, a dummy variable indicating that the veteran was illiterate, dummies indicating that information on wealth and on literacy was missing, a dummy variable indicating that the veteran had been a volunteer, wartime disease dummies (typhoid, smallpox, respiratory, rheumatic fever, measles, diarrhea, insanity, malaria, fever, syphilis, gonorrhea, hepatitis, and cardiovascular), and state of regiment fixed effects. Clustered standard errors. The symbols [‡], [†], and * indicate that the hazard ratio differs from 1 at the 1, 5, and 10 percent level of statistical significance. 3,650 observations.

Table 5: Effects of Company Cohesion and Stress on Probability of Developing Arteriosclerosis and Bounding Pulse

	Arteriosclerosis		Bounding Pulse	
	$\frac{\partial P}{\partial x}$	$\frac{\partial P}{\partial x}$	$\frac{\partial P}{\partial x}$	$\frac{\partial P}{\partial x}$
Fraction company wounded	0.227	0.484 [‡]	0.101	0.393 [‡]
	(0.164)	(0.221)	(0.116)	(0.132)
Dummy=1 if cohesive	-0.009	0.016	-0.011	0.010
	(0.013)	(0.016)	(0.009)	(0.012)
Fraction company wounded × Dummy=1 if cohesive		-0.554*		-0.579 [‡]
		(0.275)		(0.214)
Pseudo R^2	0.040	0.041	0.069	0.074

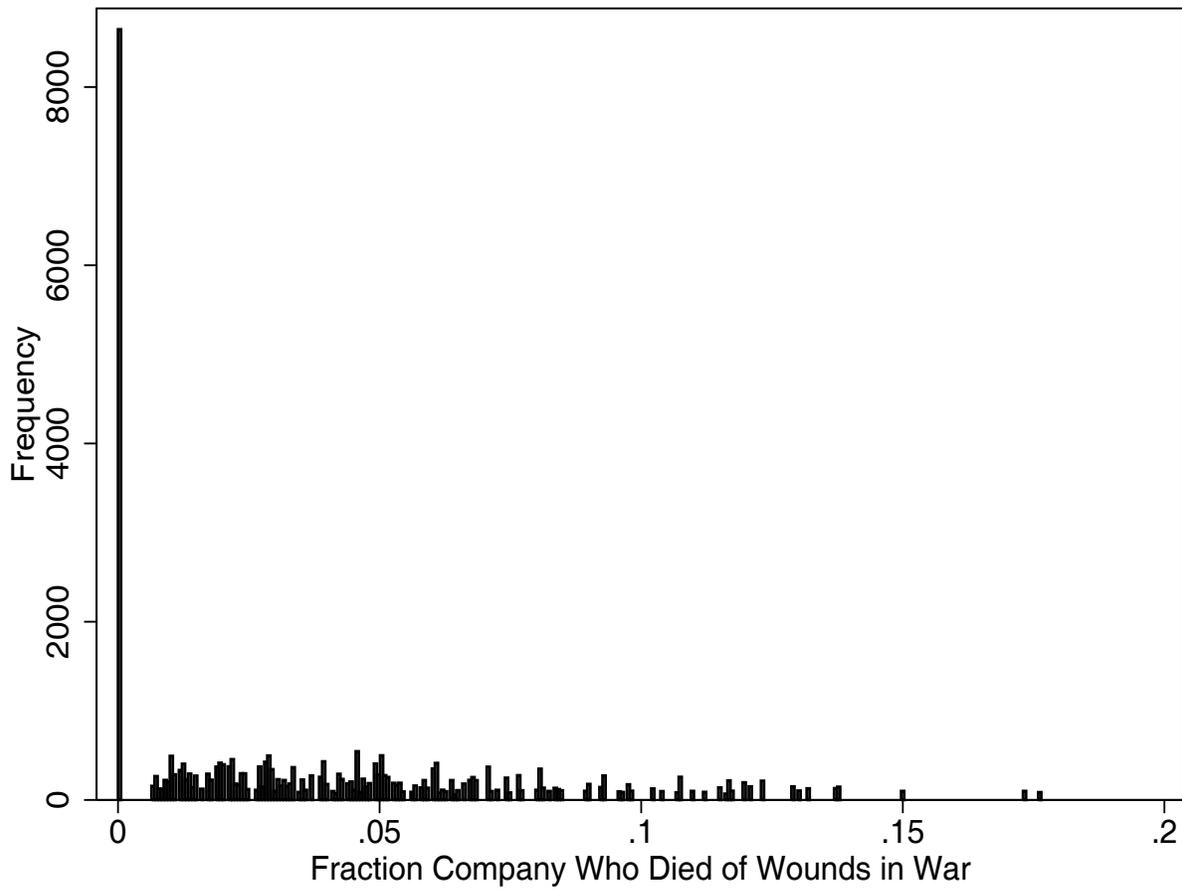
Results are from a probit model. The dependent variables in the first two regressions are equal to one if the veteran developed arteriosclerosis between 1900 and 1915. The dependent variables in the last two regressions are equal to one if the veteran developed bounding pulse between 1900 and 1915. The samples are restricted to veterans alive in 1915, age 50-64 in 1900, and who did not have arteriosclerosis or bounding pulse, respectively, in 1900. The samples are also restricted to men on the pension rolls in 1900, with known date of death, who did not change companies, and with both muster-in and discharge information. Additional control variables are age in 1900, dummy variables indicating whether the veteran had been wounded slightly in the war, whether he had ever been wounded severely, whether he had been a POW early in the war, and whether he had been a POW late in the war, occupation at enlistment dummy variables (professional or proprietor, artisan, laborer, and unknown, with farmer as the omitted category), country of birth dummy variables (Britain, Ireland, Germany, and other foreign country, with US as the omitted category), dummy variables indicating whether the veteran had been in a support position or had been a commissioned or non-commissioned officer, a dummy variable indicating if the veteran had deserted, the logarithm of household personal property wealth in 1860, a dummy variable indicating that the veteran was illiterate, dummies indicating that information on wealth and on literacy was missing, a dummy variable indicating that the veteran had been a volunteer, wartime disease dummies (typhoid, smallpox, respiratory, rheumatic fever, measles, diarrhea, insanity, malaria, fever, syphilis, gonorrhea, hepatitis, and cardiovascular), quarter of birth dummies (including one for missing), and state of regiment fixed effects. Clustered standard errors. The symbols ‡, †, and * indicate that the hazard ratio differs from 1 at the 1, 5, and 10 percent level of statistical significance. 2,821 observations in the arteriosclerosis regression and 2,592 observations in the bounding pulse regression.

Table 6: Competing Risk Hazard Model for Desertion

	Desertion	
	Hazard Ratio	Std Err
Dummy=1 if occupation		
Farmer		
Artisan	1.435 [‡]	0.093
Professional/proprietor	1.359 [‡]	0.105
Laborer	1.572 [‡]	0.121
Dummy=1 if born in		
US		
Germany	0.884	0.146
Ireland	1.310 [‡]	0.103
Great Britain	1.396 [‡]	0.148
Other	1.245 [†]	0.120
Age at enlistment	0.985 [‡]	0.003
Dummy=1 if married	1.382 [‡]	0.128
Log(total household personal property), 1860	0.950 [‡]	0.017
Dummy=1 if illiterate	1.601 [‡]	0.243
Company-level measures		
Birth place fragmentation	1.405	0.496
Occupational fragmentation	3.428 [†]	1.682
Coefficient of variation for age × 100	1.032 [*]	0.017
Log(population) city enlistment	1.058 [†]	0.028
Dummy=1 if mustered in		
1861		
1862	1.632 [‡]	0.200
1863	2.338 [‡]	0.437
1864	1.472 [‡]	0.196
1865	2.628 [‡]	0.437
Dummy=1 if volunteer	0.749 [†]	0.100
Percent in county of enlistment voting for		
Lincoln	0.995 [†]	0.003
Percent in company dying (time-varying)	1.036 [‡]	0.011
Fraction Union victories (time-varying)	0.610 [‡]	0.075
Duration dependence parameter	0.682	0.027
χ^{33} for significance		
of all coefficients	784.32	

Days until desertion are measured from first mustering in. Standard errors are clustered on the company. The symbols *, †, and ‡ indicate that the coefficient is significantly different from 1 at the 10, 5, and 1 percent level, respectively. Significance of all coefficients is for equality of all coefficients to 1. Men who died, became POWs, were discharged, were missing in action, or changed companies before first desertion are treated as censored. Covariates include height in inches and dummy variables indicating missing information for occupation, the 1860 census, literacy, and county voting. Included region fixed effects are for Middle Atlantic, East North Central, West North Central, Border, and West (New England is the omitted category). Source: Costa and Kahn (2003b).

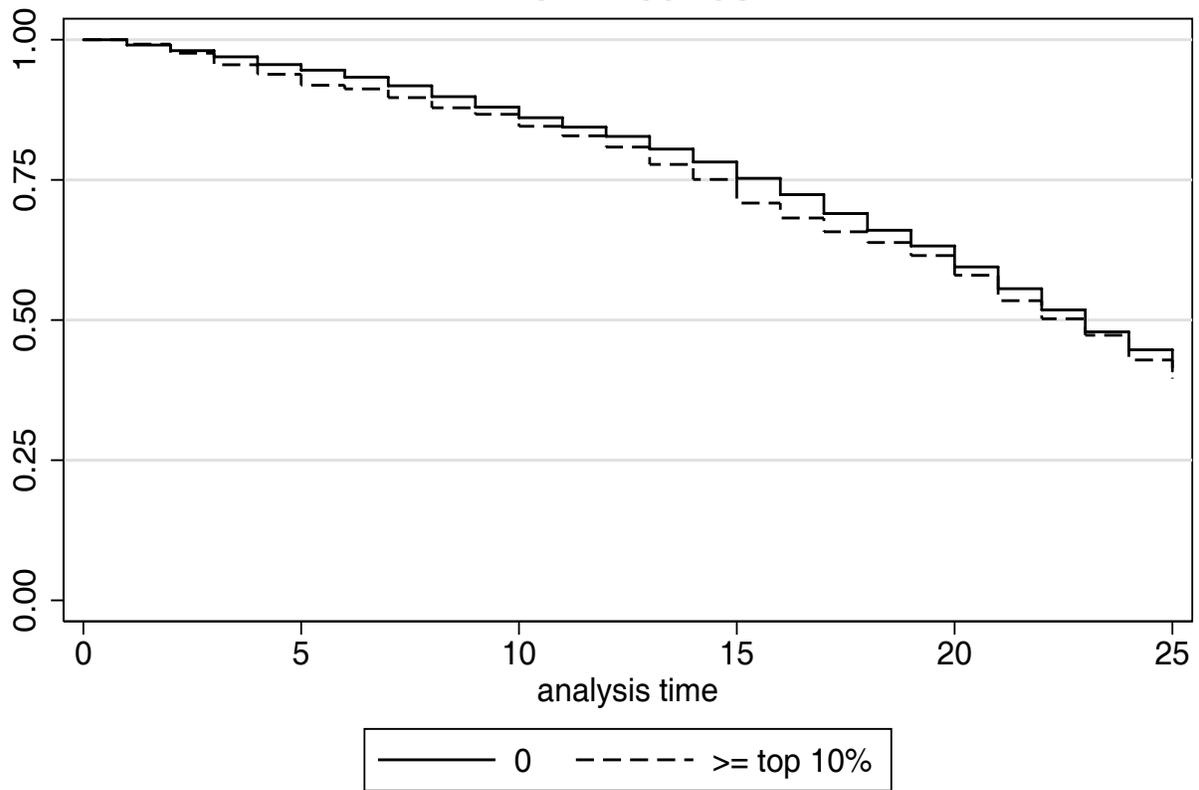
Figure 1: Wartime Frequency Distribution of Company Mortality from Wounds



Estimated from the full wartime sample of 35,000 men. The frequency gives the numbers of soldiers whose companies had the specified mortality.

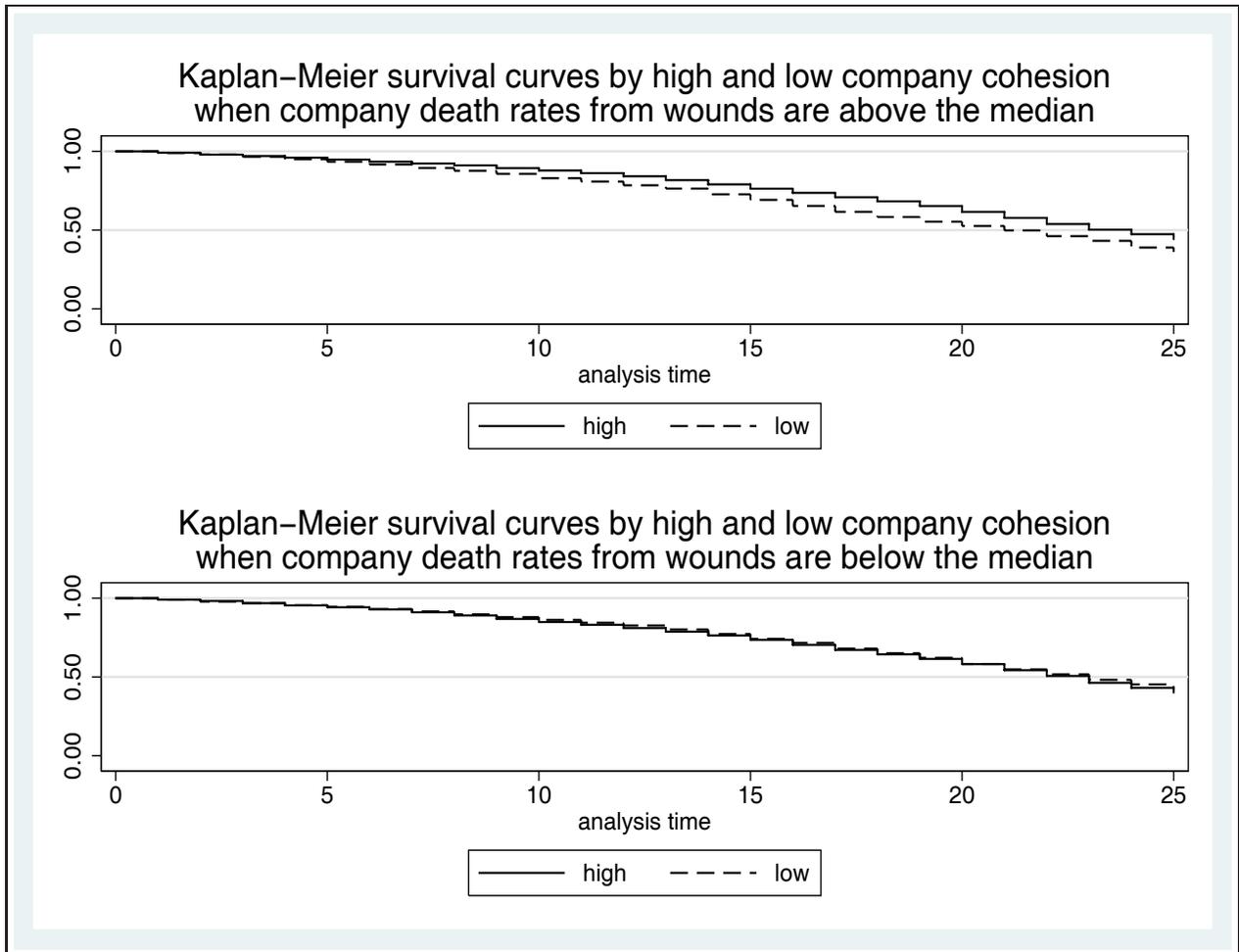
Figure 2: Survival Rates, by Company Death Rates from Wounds

Kaplan–Meier survival curves by high and low company death rates from wounds



Restricted to men on the pension rolls in 1900, with known date of death, who did not change companies, and with both muster-in and discharge information. Analysis time is time in years. The survival curve is adjusted for age. The top tenth percentile for company death rates from wounds was 8.9 percent.

Figure 3: Survival Rates, by Low and High Company Cohesion and by Company Death Rates from Wounds



Restricted to men on the pension rolls in 1900, with known date of death, who did not change companies, and with both muster-in and discharge information. Analysis time is time in years. Survival curves are adjusted for age. The median company death rate from wounds was 2.7 percent.