

**Understanding Mid-Life and Older Age Mortality Declines: Evidence from Union Army
Veterans**

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ABSTRACT

Infectious disease earlier in life had a scarring effect on older age mortality among those reaching middle age around 1900, with the strongest negative effect on survival of having grown up in a large city. The effect of reduced early life and young adult infectious disease rates accounts for 13% of the 21 percentage point increase in survival rates among 50-64 year old males in the twentieth century. Reductions in mortality from infectious and other acute disease account for another 13% of the increase.

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1 Introduction

At the beginning of the twentieth century American men's life expectancy at age 55 was 17 years and at age 65 it was 11 years. By the century's end life expectancy at ages 55 and 65 had risen to 23 and 16 years, respectively. This increase in life expectancy was extremely slow at first, rising by only about half a year during the first three decades of the twentieth century. More recently, it has been more rapid. Between 1930 and 1960 life expectancy at these ages rose by a year and in the last four decades of the century rose by 4 years at age 55 and by 3 years at age 65.¹

Several factors may explain increases in life expectancy at older ages. Advances in medical care have reduced the deadliness of chronic conditions (Cutler et al. (1998), Velkova, Wolleswinkel-Van den Bosch, and Mackenbach (1997)). Life style changes such as the cessation of smoking or the switch to a low fat and lower sodium diet may account for some of the recent mortality improvements; however, the increase in smoking from the late 1900s through the first half of the twentieth century probably contributed to increased mortality rates. Occupational exposure to harmful dust and fumes has declined. Infectious disease rates have fallen. Those reaching middle age today were rarely exposed to infectious disease as children or young adults. There is extensive evidence that mid-life, young adult, and early childhood events matter to older age health and longevity (Doblehammer and Vaupel 2001; Gavrilov and Gavrilov 1999; Manton, Stallard, and Corder 1997; Costa 2000; Elo and Preston 1992; Barker 1992, 1994). But, there is also evidence that earlier life events have relatively little impact on older age mortality (Christensen et al. 1995; Kannisto, Christensen, and Vaupel 1997; Kannisto 1994). Selection may lead to only the healthiest surviving and mortality at older ages may therefore be lower than in a population not exposed to stress at young ages. Furthermore, current life events may be more important determinants of mortality than past life events.

¹See the Berkeley Mortality Database and Anderson (1999).

This paper examines a past population, veterans of the Union Army, to establish the role of infectious disease at early, young adult ages, and later ages and of occupation at young and mid-life ages on older age mortality. It also decomposes the twentieth century increase in older age survival rates into the portions due to reduced infectious disease rates and to occupational shifts from manual to white collar jobs and into the fractions attributable to early and young adult infectious disease rates and to the immediate effects of older age mortality reductions from infectious disease. The Union Army population was exposed to a wide variety of infectious diseases while in the army, faced very different disease experiences at young ages that depended upon the size of city of origin, faced high occupational risks, and had medical care that was ineffective at best. I can therefore examine the effect of untreated infectious disease on later mortality. Causes of death among Union Army veterans differ from those of modern populations because of the high death rates from infectious, diarrheal, and respiratory diseases that prevailed in the past. The findings therefore have implications for modeling the determinants of mortality in past populations. They also have implications for modern populations as well, because I can examine the determinants of waiting time until death from a specific cause.

The findings have implications for theories of aging. If variables related to infection are shown to have a large effect on later mortality, then the results suggest that senescent processes may be plastic and highly controllable. The findings therefore have implications for understanding future mortality trends, for assessing the age at which public health policies in countries undergoing an epidemiological transition should be targeted, and for evaluating the standard of living in the United States during industrialization. If infectious disease has a long reach, then the urbanization that accompanied industrialization may have been much more costly than suggested by comparisons of urban and rural mortality rates (Williamson 1990; Steckel and Floud 1997).

2 Empirical Framework

The paper models Union Army veterans' waiting time until death from all causes or from a specific cause by means of a Cox proportional hazard model. The hazard, $\lambda(t)$, or the rate at which spells are completed after duration t given that they last until at least t , is

$$\lambda(t) = \exp(x'\beta)\lambda_0(t) \quad (1)$$

$$\lambda(t) = \exp(x'_I\beta_I + x'_{C_y}\beta_{C_y} + x'_{C_o}\beta_{C_o} + x'_O\beta_O + x'_M\beta_M)\lambda_0(t) \quad (2)$$

where λ_0 is the baseline hazard and $\exp(x'\beta)$ is the relative hazard. The vector x consists of five basic types of variables: 1) direct indicators of episodes of infectious disease while in the army (x_I); 2) indicators of size of city of residence at enlistment in 1861-1864, a proxy for the severity of the disease environment during growing ages because most men who enlisted in a given size city grew up in that city size (x_{C_y}); 3) indicators of size of city of residence at older ages in 1900, a proxy for exposure to infectious disease at older ages (x_{C_o}); 4) indicators of occupation at enlistment and at older ages (x_O); and, 5) miscellaneous other control variables (x_M). Artisans, laborers, and farmers were more likely to suffer from respiratory symptoms than professionals or proprietors because of their greater exposure to harmful dust, fumes, and gases (Costa 2000). Antebellum cities of over 50,000 were particularly deadly. In addition to greater exposure to infectious disease, residents of larger cities may have had poorer housing and nutritional intakes, thus increasing their susceptibility to the severe disease environment. Although by 1900 the largest cities were less deadly than mid-size cities because of their earlier initiation of such sanitary reforms as water filtration (Preston and Haines 1991: 98), exposure to air-borne disease is likely to be greater in larger cities because of crowding. By 1995-1997 age-adjusted death rates were greater on average in rural than in urban areas, but this relationship varied by race, sex, and region (United States National Center for Health Statistics 2000).

The paper uses a competing risks framework, treating individuals who die from a cause of death other than that being examined as censored, and estimates the impact of infectious disease and occupation on waiting time until death from a specific cause. It examines all natural cause mortality, mortality from chronic disease, mortality from acute diseases, all heart disease, ischemic heart disease, myocarditis, respiratory illness, infectious illness, and stomach ailments.

The paper determines whether there is a scarring effect of earlier infectious disease and how much of the difference in survival probabilities between Union Army veterans and a modern population is accounted for by reductions in infectious disease rates and how much by occupational shifts. It also calculates the portion of the difference attributable to immediate reductions in infectious disease rates and their mortality sequelae and the portion attributable to early life infectious disease rates. It begins by estimating hazard models for all cause mortality, A , for mortality from chronic conditions (all deaths other than infectious, respiratory, or diarrheal diseases), H , for mortality from infectious and parasitic disease, F , and for mortality from specific causes (all heart, ischemic, myocarditis, respiratory illness, infectious, stomach), P . The survivor functions, or the probabilities of not dying from cause J at least until t if other causes of death are precluded, $S_J(t)$, $J = A, H, F, P$, are

$$S_J(t) = \exp[\ln S_{J,0}(t) \exp(x'_I \beta_{J,I} + x'_{C_y} \beta_{J,C_y} + x'_{C_o} \beta_{J,C_o} + x'_O \beta_{J,O} + x'_M \beta_{J,M})] \quad (3)$$

where $S_{J,0}(t)$ is the baseline survivor function. Assuming no infectious disease in the army ($x_I = 0$) and no exposure to infectious disease at young and older ages (no elevated mortality risk in large cities, $x_{C_y} = 0$ and $x_{C_o} = 0$), the survival functions become

$$S_{IC_y C_o J}(t) = \exp[\ln S_{J,0}(t) \exp(x'_O \beta_{J,O} + x'_M \beta_{J,M})]. \quad (4)$$

Assuming elimination of infectious disease in the army and of exposure to disease at young ages

only (no elevated mortality risk in large cities of enlistment, $x_I = 0$ and $x_{C_y} = 0$), the survival functions are

$$S_{C_yIJ}(t) = \exp[\ln S_{J,0}(t) \exp(x'_{C_o}\beta_{J,C_o} + x'_O\beta_{J,O} + x'_M\beta_{J,M})]. \quad (5)$$

Finally, if the occupational distribution had been the same as that prevailing today ($x_O = x_{TO}$), the survivor functions become

$$S_{OJ}(t) = \exp[\ln S_{J,0}(t) \exp(x'_I\beta_{J,I} + x'_{C_y}\beta_{J,C_y} + x'_{C_o}\beta_{J,C_o} + x'_O\beta_{J,TO} + x'_M\beta_{J,M})]. \quad (6)$$

The survivor function will underestimate the effect of reduced job hazards on older age mortality because reduced hazard within occupation is not measured.

The paper presents two different calculations decomposing the increase in survival rates into the portion due to reduced infectious disease rates and the portion due to occupational shifts. The paper first uses the all cause mortality equation to estimate the increase in survivor probabilities assuming no infectious disease ($x_I = 0$, $x_{C_y} = 0$, and $x_{C_o} = 0$) and assuming today's occupational distribution ($x_O = x_{TO}$),

$$S_{IC_yC_oA}(t) - S_A(t) \quad (7)$$

$$S_{OA}(t) - S_A(t). \quad (8)$$

Secondly, the paper estimates the estimate the increase in survivor probabilities for both the chronic and acute disease specifications assuming no infectious disease at young ages ($x_I = 0$ and $x_{C_y} = 0$) and today's occupational distribution. The paper also estimates the effect of immediate reductions in infectious disease rates and in acute disease mortality, that is eliminating

infectious, respiratory, and parasitic disease as a cause of death, as

$$S_H(t) - S_A(t) \tag{9}$$

multiplied by the probability of dying from an acute disease. The additional gain from eliminating infectious disease at young ages is

$$[S_{IC_yH}(t) - S_H(t)]. \tag{10}$$

Because many individuals in the sample undoubtedly suffered from infectious disease either before or after their army experience (though perhaps less severe forms of these infections) and because individuals in small cities were exposed to infectious disease as well, this estimate will understate the gain from eliminating early life and young adult infectious disease among Union Army veterans. Dividing this estimate by the observed difference in survivorship rates between Union Army veterans and a modern population yields a rough estimate of the gain in survivorship rates due to reductions in infectious disease rates. Unobserved disease episodes among Union Army veterans will lead me to underestimate this gain whereas unobserved disease episodes among men in modern populations and unobserved changes in the scarring effects of infectious disease will lead me to overestimate this gain.

Decomposing the increase in survivor rates into the portion due to eliminating early life and young adult infectious disease assumes that there is a gain at older ages to eliminating infectious disease at younger ages. But, cohorts who survive infectious diseases may acquire partial or complete immunity from such diseases as measles, typhoid, and malaria and therefore may have lower mortality rates. If genetic susceptibility to death from infectious disease or other insults at young ages is positively correlated with genetic susceptibility to develop chronic disease at older ages, then, because fewer genetically “frail” individuals survive to old age, the mortality

rate of such a cohort may be lower relative to a cohort in which more genetically frail individuals survive. Stress in early life may, however, lead not to selection of the fittest but to permanent scarring.

The medical literature provides many examples of the scarring effects of infectious disease.² Infectious diseases that can affect cardiac functioning include rheumatic heart disease, late stage syphilis, measles, and typhoid fever. Infections that have been implicated in atherosclerosis include include helicobacter pylori, a bacterium that causes gastritis and stomach ulcers, and chlamydia pneumonia, a bacterium that causes acute upper and lower respiratory infections. Individuals at risk of later susceptibility both to pneumonia and to chronic obstructive lung disease, include those who have had prior upper respiratory infections (including those accompanying falciparum malaria) and on the job exposure to dust and fumes. Correlates of later gastritis and ulcers include infection with helicobacter pylori and having been a prisoner of war status, perhaps because many suffered gastrointestinal problems during their imprisonment.

3 Data

The data used in this paper are drawn from a unique longitudinal dataset based upon the records of the Union Army pension program.³ This pension program was the most widespread form of assistance to the elderly prior to Social Security, covering 85% of all Union Army veterans by 1900 and 90% by 1910 (Costa 1998:160). The program began in 1862 to provide pensions to both regular and volunteer recruits who were severely disabled as a direct result of military

²See National Bureau of Economic Research Working Paper No. 8000 for an in-depth discussion.

³The data are *Aging of Veterans of the Union Army: Military, Pension, and Medical Records, 1820-1940 (ICPSR 6837)*, *Aging of Veterans of the Union Army: Surgeons' Certificates, 1860-1940 (ICPSR 2877)*, *Aging of Veterans of the Union Army: United States Federal Census Records, 1850, 1860, 1900, 1910 (ICPSR 6836)*. See also <http://www.cpe.uchicago.edu/> for further information.

service (see Costa (1998:197-212) for a history of the Union Army pension program). It became a universal disability and old-age pension program for veterans in 1890, with any disability entitling the veteran to a pension. Even though old age was not recognized by statute law as sufficient cause to qualify for a pension until 1907, the Pension Bureau granted the minimum pension to those age 65 unless they were “unusually vigorous.” The records contain complete medical examinations conducted by a board of three examining surgeons. The surgeons rated the severity of specific conditions using detailed guidelines provided by the Pension Bureau. Several tests indicate that the population is representative of the general population circa 1900 in terms of mortality experience (Costa 1998: 197-212), suggesting that veterans’ older age mortality was determined more by environmental factors common to the population as a whole than by wartime experience.

Copious records were generated by the Union Army pension program. Pension applications included detailed medical examinations both for men whose pension application or bid for a pension increase was rejected and for men whose applications were accepted. These records have been linked to the 1900 census and to military service records. The 1900 census provides occupational and residential information and the military service records information on stress at young adult ages such as prisoner of war status and such illnesses as measles, diarrhea, tuberculosis, typhoid, rheumatism, acute respiratory infections (e.g. pneumonia, bronchitis, influenza), malaria, and war injuries.

The sample is restricted to native-born men linked to the 1900 census, aged 50 to 64 in 1900, and on the pension rolls by 1900. A surgeon’s exam detailing medical conditions is available for 93% of men. The sample is restricted to the 48% of men whose pension records provide information on cause of death. For the most part, these were men with a surviving spouse and men who lived longer. Health conditions in 1900 as noted by the examining surgeons do not predict whether information on cause of death is missing. If men in my sample are on the

whole healthier, I am likely to underestimate the impact of infectious disease and occupation on mortality. However, including men with unknown causes of death in the sample and treating them as censored yields similar results. Total sample size for men with known cause of death is 2,907. In the analysis all deaths beyond a 25 year observation period are censored. Shorter follow-up periods yield similar results but do not provide enough observations for some causes of death such as myocarditis. Beyond this 25 year observation period variables cease to have predictive power. Those who survived beyond 25 years were on average older than 83, suggesting that in this sample living to oldest-old ages is not predictable.

Causes of death were coded as heart disease, respiratory illness (chronic respiratory and pneumonia, influenza, and bronchitis), stomach ailments (gastritis/duodenitis and ulcers), infectious (including tuberculosis but excluding pneumonia, influenza, and bronchitis), cancer, diarrheal, and genito-urinary. Heart disease was subdivided into cerebrovascular, valvular, ischemic (atherosclerosis, arteriosclerosis, coronary occlusion, and coronary thrombosis), and myocarditis.⁴ Acute disease includes all infectious, respiratory, and diarrheal disease. Men with vague and unclassifiable causes of death tended to be those without a surviving spouse. Conditions noted by the examining surgeons in 1900 predicted mortality by cause: valvular heart disease in 1900 predicted death from valvular heart disease but not from ischemic heart disease; stroke, angina, and varicose veins predicted cerebrovascular deaths; stroke predicted death from ischemic heart disease; irregular pulse predicted death from myocarditis; bronchitis and emphysema predicted death from respiratory causes; gastritis weakly predicted death from stomach ailments; and, prostate problems predicted death from genito-urinary causes. A tumor in 1900 was not a good predictor of death from cancer, perhaps because the examining surgeons could

⁴See Fleming (1997) and Finlayson (1985) for a history of diagnosis of ischemic heart disease. Chronic and acute respiratory illnesses are classified together because of the difficulty in distinguishing between the two in cause of death information.

only diagnose the tumors that they could visually observe.

Table 1 shows major causes of death among Union Army veterans over a 25 year period and, over a 17 year period, among Union Army veterans and white men in the first National Health and Nutrition Examination Survey, a random sample of the population. This table shows that heart disease is now largely ischemic, whereas in the past, because valvular heart disease in 1900 was a strong predictor of death from unidentifiable heart disease, up to 21% of all deaths may have been from valvular heart. Table 1 also illustrates the high levels of infectious, respiratory, and diarrheal disease in the past relative to today. This suggests that it may be difficult to detect the effects of early life and young adult events on all cause mortality because such current events as recent exposure to infectious and parasitic disease may be more important.

4 Results

Recall that I model the relationship between such early life and young adult events as infectious disease while in the army and cause specific morbidity at mid and late ages by means of a competing risks proportional hazards model,

$$\lambda(t) = \exp(x'_I\beta_I + x'_{C_y}\beta_{C_y} + x'_{C_o}\beta_{C_o} + x'_O\beta_O + x'_M\beta_M)\lambda_0(t) \quad (11)$$

where λ_0 is the baseline hazard and where the estimated exponentiated coefficients have the interpretation of hazard ratios for a one unit change in x . Deaths from causes other than those examined are treated as censored. The vector x_M consists of age in 1900 and dummy variables indicating whether the veteran was ever wounded in the war or was ever a prisoner of war. The vector x_{C_y} consists of dummy variables for size of city at enlistment (over 50,000, between 25,000 and 50,000, 2,500 to 25,000, less than 2,500) and the vector x_{C_o} consists of dummy variables for size of city of residence in 1900 (one of the top 10 largest cities in the country, one of next 11th

to 36th largest cities, and a smaller city or not a city). Because city of birth is known for only a fraction of the sample, I use size of enlistment city as a proxy. Among those recruits with both pieces of information, 71% of those enlisting in a city of more than 50,000 in 1860 were born in the same-sized 1860 city and 77% of those enlisting in a city of less than 2,500 were born in the same-sized 1860 city. The vector x_O consists of dummy variables indicating occupation at enlistment (professional or proprietor, farmer, artisan, laborer, or unknown) and occupation circa 1900 (professional or proprietor, farmer, artisan, or laborer). Those out of the labor force in 1900 are assigned their pre-retirement occupation as recorded in the pension records. Finally, the vector x_I consists of dummy variables specifying whether the veteran while in the army ever was hospitalized for or was absent because of diarrhea or cholera, respiratory infections (bronchitis, pneumonia, and influenza), measles, typhoid, malaria, tuberculosis, rheumatic fever or rheumatic arthropathies, such stomach ailments as gastritis and ulcers, and syphilis. Fifty percent of the sample had no recorded cases of these illnesses. The mean number of different types of illnesses was 0.75 and the maximum was 5. While there are many cases of a statistically significant positive association between illnesses controlling for enlistment characteristics, there are no significant negative associations.

Table 2 presents the results for all cause mortality and for mortality from all chronic disease and from all acute (or, more precisely, infectious, respiratory, and parasitic) disease. Tables 3 and 4 present the results for mortality from respiratory illnesses, stomach ailments, infectious disease, all heart ailments except cerebrovascular, ischemic heart disease, and myocarditis. Examining whether specific infectious diseases have an effect on mortality by cause may reveal scarring effects not observed in the all-cause regressions. Except for deaths from infectious disease in Table 4, I cannot reject the hypothesis that all coefficients in the regressions are jointly statistically significantly different from 1. Using the scaled Schoenfeld residuals and Grambsch and Therneau's (1994) global test, I also cannot reject the hypothesis that the proportional

hazards assumption is met. These tables do not present results for deaths from cerebrovascular, valvular heart disease, cancer, and genito-urinary causes. None of the explanatory variables were significant predictors of death from stroke, suggesting that an unobservable such as salt intake may be the culprit. Laborers were at greater risk relative to professionals and proprietors of dying from valvular heart disease, but none of the wartime disease variables were predictors. Hepatitis infection during the war was the only significant predictor of cancer deaths, perhaps because deaths from stomach cancer were a significant proportion of all recognized cancer deaths. Only a large city of enlistment predicted deaths from genito-urinary causes. None of the variables predicted violent or accidental deaths.

Tables 2, 3, and 4 illustrate the importance of distinguishing mortality by cause of death, particularly in investigating the impact of infectious disease while in the army. Stomach ailments while in the army significantly predicted mortality from all causes, all chronic disease, all heart disease other than cerebrovascular, and ischemic heart disease. Stomach ailments also significantly predicted mortality stomach causes at the 5% level of significance when other infectious wartime disease covariates were excluded. Diarrhea was a significant predictor of mortality from stomach ailments. It was also the only illness that significantly provided protection against mortality, in this case from respiratory illness, though the possible pathway is not clear.⁵ Respiratory infection significantly predicted mortality from respiratory illnesses and predicted death from acute illnesses at the 5% level when other wartime disease covariates were excluded. Malaria was a significant predictor of mortality from respiratory illnesses at the 5% level when other wartime disease covariates were excluded. Rheumatic fever significantly predicted mortality from all heart disease other than cerebrovascular at the 10% level of significance and from myocarditis at the 5% level. Syphilis was a significant predictor of death from all heart disease

⁵Seven percent of the sample suffered both from diarrhea and from respiratory infections while in the army. The two conditions are significantly, positively associated controlling for enlistment characteristics.

and from myocarditis. Measles was a significant predictor of death from myocarditis. Syphilis was a significant predictor of death from infectious disease at the 5% level of significance when other wartime disease covariates were excluded. Typhoid was not a significant predictor of death from any cause. Neither was tuberculosis, but the size of the coefficient in the regressions of death from acute disease and from infectious disease was substantial. Interactions between wartime illnesses had no predictive power.

Infectious diseases that were significant predictors of cause specific mortality also significantly predicted the probability of having a related disease state in 1900 and in 1910 (see Costa 2000). Malaria and respiratory infections while in the army significantly predicted respiratory problems at older ages. Diarrhea had a protective effect for “dullness of chest.” Rheumatic fever significantly predicted tachycardia, irregular pulse, congestive heart failure, murmurs, and arteriosclerosis. Syphilis was associated with arteriosclerosis and tachycardia. Stomach ailments significantly predicted tachycardia. Not all predictors of disease states in 1900 or in 1910 were predictors of death from related causes. Whereas measles was a significant predictor of respiratory distress later in life and typhoid of irregular pulse and valvular heart disease, neither measles nor typhoid predicted death from related causes.

Tables 2, 3 and 4 show that having probably grown up in a large city had a lasting effect on mortality even controlling for later residence. Men who enlisted in a city of 50,000 or more faced a significantly greater risk of death from all cause mortality, from all chronic disease, from all respiratory, infectious, and parasitic disease, from respiratory illness, from stomach ailments, from all heart disease except for cerebrovascular, and from ischemic heart disease. They also faced a greater risk of mortality from infectious disease, though not significantly so. All of the city of enlistment dummies were jointly significantly different from 1 at the 1% level in the all cause and all chronic regressions and at the 5% level in the respiratory regressions. I cannot ascertain whether men who enlisted in a larger city faced a greater risk of death from myocarditis because

of collinearities between large city of enlistment and large city of residence in 1900. Large city of residence in 1900 was a significant predictor of death from myocarditis, from respiratory disease, and from all respiratory, infectious, and parasitic diseases.⁶

The other measure of environmental stress with significant predictive power was prisoner of war status. Former prisoners of war faced substantially greater risk of death from stomach ailments, though not from other causes.

Men who worked as laborers and artisans faced an elevated risk of death relative to professionals and proprietors and to farmers. Laborers were significantly more likely than professionals and proprietors to die of stomach ailments, all heart disease, and myocarditis. Artisans were significantly more likely to die of all heart disease relative to professionals and proprietors. Farmers were significantly less likely than professionals and proprietors to die of ischemic heart disease and of all chronic disease. Both artisans and laborers were more likely to die of respiratory disease (and from all acute illnesses), though not significantly so. The dummy variables indicating occupation circa 1900 were jointly significantly different from 1 at the 1% level in the all cause, all chronic, all heart, and myocarditis regressions.

On the whole occupational shifts have a smaller impact on the predicted 25 year survival probability, $S(25)$, than reductions in infectious disease rates. Table 5 illustrates the effect on the predicted survival probability of setting all diseases while in the army equal to zero, setting all large city dummies equal to zero (arguably reducing infectious disease rates), and setting the occupational distribution in 1900 to that prevailing in 1970 and the occupational distribution at enlistment to that prevailing in 1920. Except for death from respiratory and infectious disease, occupational shifts have a smaller impact on the relative survival probability even though the

⁶Large city of residence in 1900 does not become a significant predictor of death from other causes when size of city of enlistment is omitted nor is the interaction between large city of enlistment and large city in 1900 a significant of death from respiratory heart disease.

impact of infectious disease on the survival probability is 1 likely to be understated because of unobserved disease episodes.

I also ran specifications that included Body Mass Index (BMI) circa 1900, a measure of current net nutritional status, and adult height measured at enlistment, a measure of net nutritional status during the growing years. BMI was strong predictor of all cause mortality, all infectious, respiratory, and parasitic mortality, mortality from infectious disease, and mortality from respiratory disease. However, BMI may well reflect an underlying condition such as tuberculosis and is strongly correlated with the prevalence of chronic respiratory problems circa 1900. Height did not predict older age mortality, in contrast to findings obtained in Costa (1993). Sample size may be an issue. Using a sample of 309,554 men from modern Norway, Costa (1993) found that in contrast to results from the full sample in random subsamples of 10,000 the relationship between height and mortality became very sensitive to sample restrictions and a smooth curve did not always result.

5 Explaining Rising Survivorship

What is the role of reduced infectious disease rates and of occupational shifts from manual to white collar work in explaining the long-run increase in survivorship at older ages? The Union Army data can provide valuable clues on the scarring effects of infectious disease because it is possible to observe the long-run effects of growing up in a large city and of infectious disease at young adult ages. The difference in 17 year survival rates between Union Army veterans and white men in NHANES was 21 percentage points. Table 6 presents two different calculations. The first calculation uses the all cause mortality regression in Table 2 and sets the wartime disease dummies to 0 and all city size variables to small, yielding an increase in the predicted survival rate ($S_A(17)$) in the Union Army sample of 3 percentage points and therefore explaining

16% of the difference in survival rates between the Union Army sample and NHANES. A similar calculation setting the occupational distribution in 1900 to that prevailing in 1970 and the occupational distribution at enlistment to that prevailing in 1920 implies that occupational shifts explain none of the difference in survival rates between the Union Army sample and NHANES because farmers faced favorable survival rates and their numbers declined. The second calculation uses the regressions for chronic and acute disease mortality in Table 2 to obtain the probability of not dying from chronic disease, $S_H(17)$ and the probability of not dying from infectious, respiratory, and parasitic causes, $S_F(17)$. Eliminating infectious disease early in life and at adult ages (as proxied by wartime disease dummies and by city size at enlistment) would raise each survival rate by 3 percentage points. Occupational shifts play a negligible role. Eliminating infectious, respiratory, and parasitic diseases as a cause of death would raise survival rates by 15% and, because 18% of men died of these causes, would therefore explain 13% of the increase in survival rates between the Union Army sample and NHANES.⁷ Eliminating early life infectious disease leads to a further 3 percentage point increase in survival rates and explains 12% of the increase in survival rates. The total reduction due to the elimination of infectious, respiratory, and parasitic disease is 5 percentage points or 26% of the difference in survival rates between the Union Army sample and NHANES. The remaining 74% of the decline may be explained by such unobservables as the reduced use of salt and smoke as preservatives with the rise of refrigeration, improved dietary and health habits, and the increased efficacy of medical care.

⁷This is a slight overestimate because slightly less than 2% of men in NHANES died from infectious disease.

6 Implications

This paper has shown that early life events can lead to permanent scarring. Episodes of infectious disease while in the army and growing up in a large city, a proxy for exposure to infectious disease, had a negative impact on the 25 year survival rate of Union Army veterans aged 50 to 64 in 1900. The effect of specific diseases depended upon the cause of death that was being investigated, implying that studies of past populations need to examine mortality by cause because of the high prevalence of respiratory, infectious, and diarrheal deaths in past populations. Predictors of death from various types of heart disease among Union Army veterans included stomach ailments, rheumatic fever, syphilis, and measles while in the army. Of particular note was the relationship between stomach ailments and death from ischemic heart disease, suggesting that such infections as helicobacter pylori may have played a role. Predictors of waiting time until death from respiratory disease included prior respiratory infections and malaria, which is frequently accompanied by respiratory infections in non-immune individuals. Predictors of waiting time until death from stomach ailments were prior stomach ailments and diarrhea. Tuberculosis while in the army predicted death from infectious disease at older ages, suggesting that the nodular scars left by tuberculosis in the apices of one or both lungs could be the seeds for later active tuberculosis many years later.

The results suggest that the costs of the urbanization that accompanied industrialization prior to the widespread acceptance of the germ theory of disease and the accompanying institution of sanitary reforms were high. Men who probably grew up in a large city faced much higher mortality rates at older ages from all causes and from both chronic and acute disease controlling for size of city of residence at older ages. Men who lived in a large city in 1900 faced a higher mortality rate from all acute (infectious, respiratory, and parasitic) causes of death.

Reduced infectious disease rates and reduced mortality from acute disease explained

up to 26% of the increase in 17 year survival rates between the Union Army sample and men first observed in the National Health and Nutrition Examination Survey in 1971-1975. Period effects accounted for up to 13% of the increase in 17 year survival rates and probably depended upon public health reforms, the increased efficacy of medical care, particularly antibiotics, and improved overall health making infectious disease less deadly. Early life and young adult events also explained 13% of the rise in survival rates – an underestimate because of unobserved infections early in life. Occupational shifts from manual to white collar jobs were relatively unimportant in accounting for any of the increase in survival rates. Occupational shifts were far more important in explaining declines in disability because of their role in such debilitating chronic conditions as back problems and arthritis (Costa 2000).

The findings imply that our analyses of the costs and benefits of public health programs, particularly in developing countries, should account not only for the period effect of reduced infectious disease rates, but also for lifetime cohort effects. They also imply that our forecasts of future trends in mortality rates need to account for reductions in infectious disease rates. In the United States those who reached age 55 in 1960 were born when 14% of children died before age 1 and those who reached that age in 1995 when almost 6% of children died before age 1. Those cohorts who will reach age 55 in 2025 were born when only 2% of children died before age 1. Although the United States has already seen most of the benefits of reductions in infectious disease rates, many developing countries are still undergoing an epidemiological transition and these countries may have much larger elderly populations than expected.

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Table 1: Distribution of Causes of Death, Union Army Veterans Alive in 1900 and White Men Alive in the First National Health and Nutrition Examination Survey in 1971-1975

	25 Year Follow-up UA	17 Year Follow-up UA	Follow-up NHANES
Proportion surviving			
Entire sample	18.69	43.65	64.94
Proportion of deceased			
With cause of death	48.11	50.27	100.00
Causes of death (% all known causes)			
All Heart	41.32	38.37	51.70
Cerebrovascular	11.57	10.06	0.00
Ischemic	5.84	4.42	41.73
Myocarditis	2.27	0.93	0.00
Valvular	5.37	5.06	1.05
Other and unknown	16.27	17.90	8.92
Cancer	6.10	5.85	33.86
Respiratory	10.69	10.85	0.00
Bronchitis, pneumonia, influenza	8.57	8.42	0.00
Infectious	4.29	5.35	1.57
Tuberculosis	2.74	3.57	0.00
Stomach	4.24	5.06	3.67
Diarrhea	1.81	2.00	0.00
Genito-urinary	12.35	12.62	0.52
Violence and accidents	3.00	3.14	3.15
Other and unclassifiable	16.20	16.76	5.53

UA= Union Army sample, NHANES= National Health and Nutrition Examination Survey. Both samples exclude the foreign-born. The UA sample is restricted to men linked to the 1900 census. Causes of death in the UA sample are for 2,709 observations. NHANES contains 1,101 observations. The follow-up survey used was *National Health and Nutrition Examination Survey: Epidemiological Follow-Up Study, 1992 (ICPSR 6861)*. Because the number of deaths in the NHANES sample is relatively small, there no deaths for some causes. Other and unclassifiable in the UA sample includes such conditions as diabetes, paralysis, etc.

Table 2: Predictors from Cox Proportional Hazard Competing Risk Model for All Cause Disease Mortality, All Chronic Disease Mortality, and All Infectious, Respiratory, and Parasitic Mortality

	Mean	All Cause		All Chronic		All Infectious, Respiratory,Parasitic	
		Hazard Ratio	Std Err	Hazard Ratio	Std Err	Hazard Ratio	Std Err
Age in 1900	57.787	1.108 [†]	0.009	1.106 [†]	0.010	1.117 [†]	0.020
Dummy=1 if population in enlistment city							
≥ 50,000	0.049	1.561 [†]	0.180	1.524 [†]	0.199	1.702 [†]	0.417
25,000–50,000	0.029	1.089	0.159	1.188	0.186	0.672	0.282
2,500–25,000	0.287	1.063	0.062	1.097	0.071	0.926	0.126
< 2,500	0.635						
Dummy=1 if occupation circa 1900							
Farmer	0.436	0.834	0.062	0.775 [‡]	0.064	1.161	0.207
Professional/proprietor	0.196						
Artisan	0.148	1.117	0.098	1.070	0.103	1.364	0.284
Laborer	0.220	1.124	0.089	1.087	0.094	1.320	0.250
Dummy=1 if occupation at enlistment							
Farmer	0.416	1.214	0.168	1.250	0.194	1.101	0.336
Professional/proprietor	0.038						
Artisan	0.077	1.251	0.195	1.372	0.238	0.844	0.303
Laborer	0.083	0.988	0.156	1.040	0.183	1.320	0.250
Unknown	0.386	1.203	0.164	1.242	0.190	1.081	0.325
Dummy=1 if in war							
Wounded	0.339	0.936	0.051	0.937	0.056	0.927	0.115
Prisoner of war	0.101	1.077	0.088	1.145	0.102	0.800	0.166
Dummy=1 if in war had							
Diarrhea	0.315	1.045	0.058	1.105	0.068	0.824	0.108
Malaria	0.039	0.930	0.127	0.796	0.131	1.487	0.375
Respiratory infection	0.141	0.996	0.073	0.918	0.081	1.348	0.216
Measles	0.082	0.918	0.089	0.972	0.103	0.708	0.170
Tuberculosis	0.022	0.816	0.152	0.632	0.149	1.505	0.464
Typhoid	0.066	0.938	0.097	0.879	0.104	1.200	0.255
Rheumatic fever/arthropathies	0.127	1.101	0.083	1.074	0.092	1.208	0.199
Stomach ailments	0.015	1.559 [†]	0.292	1.740 [†]	0.342	1.016	0.516
Syphilis	0.012	1.116	0.256	1.105	0.284	1.187	0.609
Dummy=1 if in 1900 lived in							
One of 10 largest cities	0.042	1.121	0.142	1.021	0.148	1.627	0.427
11th to 36th largest city	0.071	1.048	0.103	1.017	0.112	1.190	0.264
Small city/non-city	0.887						
$\chi^2(24)$ for							
Log likelihood ratio		220.76		192.47		66.98	
Proportional hazards assumption		28.15		25.44		24.05	

2,709 observations. The symbols †, and ‡ indicate that the coefficient is significantly different from 1 at the 5% and 1% level, respectively. The log-likelihood ratio test is for equality of all coefficients to 1. The sample consists of Union Army veterans on the pension rolls by 1900, age 50-64 in 1900, linked to the 1900 census, and with cause of death information. The follow-up period is 25 years. Deaths during the 1918 influenza pandemic were excluded. Excluding these deaths did not materially change the results. Deaths from violence and accidents were treated as censored. The category chronic excludes all respiratory deaths, tuberculosis, diarrheal, and other parasitic and infectious diseases. These excluded deaths are in the category infectious, respiratory, and parasitic. The proportional hazards assumption is tested by testing for a non-zero slope in the generalized linear regression of the scaled Schoenfeld residuals on a function of time.

Table 3: Predictors from Cox Proportional Hazard Competing Risk Model of Mortality from Respiratory, Stomach, and Infectious Disease

	Mean	Respiratory		Stomach		Infectious	
		Hazard Ratio	Std Err	Hazard Ratio	Std Err	Hazard Ratio	Std Err
Age in 1900	57.787	1.115 [†]	0.025	1.146 [‡]	0.040	1.082 [†]	0.039
Dummy=1 if population in enlistment city							
≥ 50,000	0.049	2.019 [‡]	0.580	2.484	1.231	1.361	0.684
25,000–50,000	0.029	0.352	0.253	1.765	0.948	1.203	0.733
2,500–25,000	0.287	1.032	0.172	1.333	0.338	0.679	0.197
< 2,500	0.635						
Dummy=1 if occupation circa 1900							
Farmer	0.436	1.085	0.238	1.029	0.351	0.994	0.343
Professional/proprietor	0.196						
Artisan	0.148	1.393	0.356	1.219	0.501	1.191	0.466
Laborer	0.220	1.295	0.301	1.464	0.524	1.152	0.418
Dummy=1 if occupation at enlistment							
Farmer	0.416	0.858	0.292	1.224	0.679	3.920	4.048
Professional/proprietor	0.038						
Artisan	0.077	0.500	0.217	1.002	0.658	5.290	5.634
Laborer	0.083	0.614	0.252	0.767	0.509	2.709	3.000
Unknown	0.386	0.800	0.268	0.872	0.484	4.030	4.134
Dummy=1 if in war							
Wounded	0.339	0.949	0.147	0.704	0.176	0.796	0.201
Prisoner of war	0.101	0.897	0.225	1.863 [†]	0.559	0.573	0.267
Dummy=1 if in war had							
Diarrhea	0.315	0.647 [‡]	0.112	1.680 [†]	0.389	0.990	0.252
Malaria	0.039	1.771	0.531	1.225	0.646	1.022	0.614
Respiratory infection	0.141	1.725 [‡]	0.331	0.961	0.307	0.735	0.269
Measles	0.082	0.918	0.208	1.289	0.468	0.906	0.392
Tuberculosis	0.022	1.090	0.470			2.976	1.478
Typhoid	0.066	1.327	0.342	0.671	0.317	0.740	0.387
Rheumatic fever/athropathies	0.127	0.930	0.211	1.203	0.373	1.268	0.406
Stomach ailments	0.015	1.321	0.777	2.915	1.746	0.916	0.927
Syphilis	0.012	0.916	0.662			2.390	1.771
Dummy=1 if in 1900 lived in							
One of 10 largest cities	0.042	1.743	0.547	0.921	0.569	1.706	0.852
11th to 36th largest city	0.071	1.187	0.327	1.414	0.544	1.244	0.540
Small city/non-city	0.887						
$\chi^2(24), \chi^2(22)$ for							
Log likelihood ratio		57.97		35.71		23.52	
Proportional hazards assumption		21.45		19.09		19.11	

2,907 observations. The symbols [†] and [‡] indicate that the coefficient is significantly different from 1 at the 5% and 1% level, respectively. The log-likelihood ratio test is for equality of all coefficients to 1. The sample consists of Union Army veterans on the pension rolls by 1900, age 50-64 in 1900, linked to the 1900 census, and with cause of death information. The follow-up period is 25 years. Deaths during the 1918 influenza pandemic were excluded. Excluding these deaths did not materially change the results. Deaths from violence and accidents were treated as censored. The category respiratory includes chronic respiratory conditions, pneumonia, influenza, and acute bronchitis. The category stomach includes gastritis/duodentis and ulcers. The category infectious includes tuberculosis. The proportional hazards assumption is tested by testing for a non-zero slope in the generalized linear regression of the scaled Schoenfeld residuals on a function of time.

Table 4: Predictors from Cox Proportional Hazard Competing Risk Model of Mortality from All Heart Disease Except Cerebrovascular, Ischemic Heart Disease, and Myocarditis

	Mean	All Heart		Ischemic		Myocarditis	
		Hazard Ratio	Std Err	Hazard Ratio	Std Err	Hazard Ratio	Std Err
Age in 1900	57.787	1.108 [†]	0.015	1.168 [‡]	0.037	1.106	0.056
Dummy=1 if population in enlistment city							
≥ 50,000	0.049	1.558 [†]	0.306	1.941	0.782	0.000	0.000
25,000–50,000	0.029	1.086	0.275	2.431 [†]	1.007	1.168	0.896
2,500–25,000	0.287	1.153	0.113	1.164	0.260	0.952	0.350
< 2,500	0.635						
Dummy=1 if occupation circa 1900							
Farmer	0.436	0.844	0.110	0.501 [‡]	0.137	0.514	0.273
Professional/proprietor	0.196						
Artisan	0.148	1.246	0.185	0.939	0.282	1.745	0.955
Laborer	0.220	1.307 [†]	0.175	0.773	0.220	2.303	1.074
Dummy=1 if occupation at enlistment							
Farmer	0.416	1.383	0.345	0.859	0.383	1.083	0.870
Professional/proprietor	0.038						
Artisan	0.077	1.450	0.401	1.272	0.624	0.338	0.356
Laborer	0.083	1.081	0.306	0.536	0.311	0.867	0.783
Unknown	0.386	1.424	0.350	1.132	0.484	0.809	0.645
Dummy=1 if in war							
Wounded	0.339	0.915	0.085	0.948	0.195	0.908	0.306
Prisoner of war	0.101	1.173	0.159	0.645	0.242	1.514	0.694
Dummy=1 if in war had							
Diarrhea	0.315	1.122	0.106	1.052	0.227	0.827	0.293
Malaria	0.039	0.783	0.197	0.774	0.465	0.433	0.456
Respiratory infection	0.141	0.823	0.114	0.851	0.267	1.254	0.522
Measles	0.082	0.936	0.153	0.871	0.348	2.304 [†]	0.959
Tuberculosis	0.022	1.159	0.325	0.797	0.586		
Typhoid	0.066	0.903	0.161	0.783	0.337	1.127	0.701
Rheumatic fever/arthropathies	0.127	1.218	0.150	1.372	0.367	2.132 [†]	0.819
Stomach ailments	0.015	2.109 [‡]	0.578	3.341 [†]	1.778	2.609	2.710
Syphilis	0.012	1.962 [†]	0.593	2.464	1.351	7.530 [†]	5.887
Dummy=1 if in 1900 lived in							
Top 10 largest cities	0.042	1.071	0.235	0.521	0.314	2.843 [†]	1.815
11th to 36th largest city	0.071	0.997	0.170	1.150	0.394	1.869	0.942
Small city/non-city	0.887						
$\chi^2(24)/\chi^2(23)$ for							
Log likelihood ratio		104.36		61.53		41.12	
Proportional hazards assumption		22.45		27.32		28.56	

2,709 observations. The symbols † and ‡ indicate that the coefficient is significantly different from 1 at the 5% and 1% level, respectively. The log-likelihood ratio test is for equality of all coefficients to 1. The sample consists of Union Army veterans on the pension rolls by 1900, age 50-64 in 1900, and with cause of death information. The follow-up period is 25 years. Deaths during the 1918 influenza pandemic were excluded. Excluding these deaths did not materially change the results. Deaths from violence and accidents were treated as censored. The category all heart excludes cerebrovascular. The proportional hazards assumption is tested by testing for a non-zero slope in the generalized linear regression of the scaled Schoenfeld residuals on a function of time.

Table 5: Effect of Infectious Disease Reductions and Occupational Shifts on the Predicted Survival Rate of 60 Year Old Men

	Predicted at Character- istics (A)	No Wartime Infections (B)	No Wartime Infections and Small City (C)	Shift in Occupational Distribution (D)
All cause	0.241 (0.142)	0.250 (0.142)	0.259 (0.142)	0.231 (0.111)
Respiratory	0.861 (0.091)	0.860 (0.091)	0.868 (0.080)	0.878 (0.093)
Stomach	0.943 (0.025)	0.954 (0.025)	0.961 (0.025)	0.944 (0.025)
Infectious	0.951 (0.012)	0.951 (0.012)	0.950 (0.012)	0.956 (0.012)
All heart except cerebrovascular	0.646 (0.069)	0.662 (0.095)	0.674 (0.095)	0.641 (0.067)
Ischemic	0.891 (0.089)	0.900 (0.089)	0.904 (0.089)	0.905 (0.086)
Myocarditis	0.953 (0.021)	0.965 (0.021)	0.969 (0.021)	0.951 (0.020)

Column A gives the predicted survival probability assuming actual wartime infectious disease, residence city, and occupational class characteristics. Column B gives the predicted survival probability setting all diseases while in the army equal to zero. Column C gives the predicted survival probability setting all diseases while in the army equal to zero and setting all large city dummies equal to zero. Column D gives the predicted survival probability setting the occupational distribution in 1900 to that prevailing in 1970 and the occupational distribution at enlistment to that prevailing in 1920. Standard errors in parentheses. Survival probabilities are probabilities of survival until death from a specific cause. Deaths from other causes are censored. All survival probabilities are predicted setting whether a veteran was ever wounded in the war or was ever a prisoner of war equal to 0. Age is set equal to 60.

Table 6: Decomposition of the Difference in the 17 Year Survival Rate between NHANES and the Union Army Sample

	% Point Increase in UA Survival Rate (A)	% of Sample at Risk (B)	% Difference UA and NHANES Accounted by (A) (C)
Difference NHANES and UA survival rates=21.3% points			
1) Change in all cause mortality, $\Delta S_A(17)$			
Total infectious=0, $S_{IC_yCoA}(17) - S_A(17)$	3.3	100.00	15.5
Occupational shift, $S_{OA}(17) - S_A(17)$	-0.2	100.00	-
2) Examining both chronic and acute disease			
i) Change in chronic disease mortality, $\Delta S_H(17)$			
Total early life infectious=0, $S_{C_yIH}(17) - S_H(17)$	2.8	81.8	10.8
Occupational shift	-1.0	81.8	-
Change in acute disease mortality, $\Delta S_F(17)$			
Total early life infectious=0, $S_{C_yIF}(17) - S_F(17)$	3.3	18.2	2.8
Occupational shift, $S_{OF}(17) - S_F(17)$	0.3	18.2	0.3
ii) Increase due to eliminating immediate acute mortality as a cause of death,			
$S_H(17) - S_A(17)$	14.6	18.2	12.5
iii) Increase due to early life infectious if all deaths from chronic disease,			
$S_{C_yIH}(17) - S_H(17)$	2.8	100.00	13.2
iv) Total due to infectious ($14.6 \times .182 + 2.8$)	5.5	100.00	25.8

UA=Union Army, NHANES=National Health and Examination Survey. Column A gives the percentage point increase in survival rates in the Union Army sample setting different combination of wartime infectious disease equal to zero, city size dummies equal to zero, and occupational classes at enlistment and at older ages equal to the 1920 and 1970 distributions, respectively. In calculation 1) the increase in the UA survival rate was predicted based upon the all cause mortality regression in Table 2. In calculation 2) this increase was predicted based upon the chronic and acute disease regressions in Table 2. Calculation 2) contains 4 steps. The first step examines the impact on changes in both chronic and acute disease mortality of setting wartime infectious disease equal to zero and large city dummies equal to zero and of occupational shifts. The second step examines the impact of eliminating acute disease as a cause of death. The third step examines the effect of setting wartime infectious disease equal to zero and large cities dummies equal to zero if all deaths are from chronic disease. The fourth step calculates the total percentage point increase in survival rates attributable to eliminating infectious disease. Column B gives the percentage of the sample at risk. This percentage varies depending upon cause of death. Column C gives the percentage of the difference in survival rates between the Union Army sample and NHANES accounted for by Column A.