This PDF is a selection from a published volume from the National Bureau of Economic Research

Volume Title: Health at Older Ages: The Causes and Consequences of Declining Disability among the Elderly

Volume Author/Editor: David M. Cutler and David A. Wise, editors

Volume Publisher: University of Chicago Press

Volume ISBN: 0-226-13231-5

Volume URL: http://www.nber.org/books/cutl08-1

Conference Date: October 8-11, 2004

Publication Date: January 2009

Chapter Title: The Health of Older Men in the Past

Chapter Author: Dora L. Costa

Chapter URL: http://www.nber.org/chapters/c11109

Chapter pages in book: (21 - 54)

1

The Health of Older Men in the Past

Dora L. Costa

1.1 Introduction

The population of the United States has been rapidly aging—slowly at first, more rapidly recently. In 1910, only 4 percent of the population was older than sixty-four. By 1940 the figure was 7 percent, and by 1990, 13 percent. By 2050 the figure is projected to rise to at least 20 percent (Costa 1998). If life expectancies increase faster than expected, the percentage of the population older than sixty-four in 2050 will be even greater. Vaupel (1991) has argued that children alive today may live ninety on average, or even one hundred years.

What are the consequences of mortality declines for older-age health? Gruenberg (1977) and Verbrugge (1984) have argued that rising longevity may increase both chronic disease and disability rates. Fries' (1980, 1989) theory of the aging process implies that the onset of chronic disease and disability rates can be postponed until the limit of life is reached, thus implying that both chronic disease and disability rates are decreasing. Manton (1982) has argued that even though declines in mortality may increase the prevalence of chronic disease, the rate of progression of chronic disease and therefore of disability may fall.

The empirical evidence available thus far shows that, on the whole, population aging has been accompanied by improvements in elderly chronic disease, disability, and functional limitation rates (for a review of recent

Dora L. Costa is a professor of economics at the University of California, Los Angeles, and a research associate of the National Bureau of Economic Research.

I thank the National Institute on Aging grants P30 AG12810 and R01 AG19805, and the Mary Woodard Lasker Charitable Trust and Michael E. DeBakey Foundation, NIH grants AG19637 and AG10120, the Robert Wood Johnson Foundation, and the Center for Advanced Study in the Behavioral Sciences.

trends see Freedman, Martin, and Schoeni [2002]). Costa (2000) finds that the average decline in chronic respiratory problems, valvular heart disease, arteriosclerosis, and joint and back problems was about 66 percent from the 1900s to the 1970s and 1980s, a decline of 0.7 percent per year. Costa and Steckel (1997) document that weight adjusted for height for older men has increased from dangerously low levels at the turn of the twentieth century. Costa (2002) finds that among men age sixty to seventy-four, functional limitations declined by 0.6 percent per year between 1910 and the 1990s. Improvements in functional limitation rates and also in disability rates accelerated since the 1980s. Functional limitation rates declined by 1.5 percent per year between 1992 and 1996 (Waidmann and Liu 2000) and by 1.3 to 1.6 percent per year between 1984 and 1993 or 1995 (Freedman and Martin 1998, 1999, 2000). Manton, Corder, and Stallard (1997) find that between 1982 and 1989 disability among older individuals declined by 1.1 percent per year and between 1989 and 1994 by 1.5 percent per year. In exactly comparable data for 1994 and 1999, disability declined by 2.1 percent per year (Manton and Gu 2001). Using different data sources and different measures of disability, Cutler and Richardson (1997) find declines of 0.5 to 1.0 percent per year between 1980 and 1990; Freedman and Martin (1998) observe declines of 0.9 to 2.3 percent per year between 1984 and 1993; and Crimmins, Saito, and Reynolds (1997) observe a disability decline of 0.9 percent per year between 1982 and 1993. Comparing the offspring of individuals in the Framingham study with the original cohort shows that among those age fifty-five to seventy, disability has fallen substantially between 1977 and 1994 (Allaire et al. 1999).

This improvement in health has not necessarily been continuous. Costa and Steckel (1997) report that chronic disease rates at older ages were higher for cohorts born between 1840 and 1849 than for those born between 1820 and 1829. Cycles may be present in more recent years as well. Although clinicians' reports document steady improvements in health since the 1970s (Waidmann, Bound, and Schoenbaum 1995), self-reported health declined during the 1970s and 1980s (Freedman and Martin 2000; Chirikos 1986; Colvez and Blanchet 1981; Crimmins 1990; Verbrugge 1984), suggesting either that health awareness has increased or that disability has fallen since the 1980s because of declines in the debilitating effects of chronic conditions. Nonetheless, the overall trend appears to be one of improving health.

This paper uses the records of Union Army veterans to document disease rates in the past and to examine why these disease rates were so high. It focuses on three broad disease states—heart conditions, musculoskeletal problems, and loss of cognitive functioning. Heart conditions and musculoskeletal problems represented the bulk of chronic conditions in the past (and still do today) and mental impairments today represent a large percentage of all medical expenditures. Understanding why disease rates were so high in the past will help us understand why the health and longevity of different cohorts has been rising in the United States and can inform our forecasts of morbidity and mortality trends.

Fogel and Costa (1997) argue that the recent increases in health and in longevity are too rapid to have been caused by genetic or evolutionary change, and that explanations should focus on changes in the physical environment. Many factors could account for changes in the physical environment, including the increased efficacy of medical care, lifestyle changes, rising incomes, and rising educational levels. My focus will be on the role of infectious disease and of occupational stress. My findings thus have implications for countries still undergoing an epidemiological transition, many of whom suffer from the same infectious diseases as the United States in the past and, like the United States in the past, have a high proportion of their population employed as manual laborers.

1.2 Environmental Conditions and Disease Etiology

I study three broad disease states—heart conditions, musculoskeletal problems, and loss of cognitive functioning.¹ Loss of cognitive functioning includes memory loss (which I also consider as a separate category), dementia, senility, depression, psychosis, and hallucinations, as well as other mental problems. The musculoskeletal problems that I consider are arthritis and back problems. The heart diseases that I examine are valvular heart disease, arteriosclerosis, and congestive heart failure. I also examine several symptoms and signs, namely, murmurs, tachycardia, bradycardia, irregular heartbeat, and a bounding pulse.

The diagnosed heart disease of the nineteenth and early twentieth century was primarily valvular, a disease involving any dysfunction or abnormality of the heart valves. In developed countries today ischemic heart disease (involving a weakened heart pump, either due to previous heart attacks or to current blockages of the coronary arteries) is the most common form of heart disease. However, it is unclear whether ischemic heart disease rates were low in the past or were often undiagnosed. Arteriosclerosis, a thickening and hardening of the artery walls, is commonly used as a term for atherosclerosis, in which fat, cholesterol, and other substances accumulate in the walls of arteries, forming plaques, which, when they rupture, cause heart attacks and stroke. However, arteriosclerosis can arise from other disease states such as diabetes mellitus or systemic or local inflammation. Congestive heart failure is a disorder in which the heart loses its ability to pump blood efficiently and which could arise from hypertension, coronary artery disease, or valvular heart disease. Arrythmias such as tachycardia (a

^{1.} Details about specific conditions, including common causes, are available from Medlineplus.

faster than normal heartbeat) and bradycardia (a slow heartbeat) can be life threatening and are more likely to occur in individuals with coronary heart disease or heart valve disorders. A bounding (strong and forceful) pulse can indicate high blood pressure or fluid overload, which in turn can be caused by congestive heart failure or aortic valve regurgitation, among other factors. An irregular heartbeat may indicate cardiac rhythm disorders or various other conditions, such as an overactive thyroid, metabolic or respiratory disorders, or even simply stress or anxiety.

Infectious disease plays a role in all three of the disease states that I examine. Rheumatic fever and infective endocarditis are the most common infections causing valvular heart disease. Group A streptococcal bacteria can cause both scarlet fever and acute rheumatic fever (which may follow scarlet fever). Rheumatic heart disease—the acute involvement of the heart (carditis)—results from rheumatic fever, but in about half of all cases will develop in the absence of any history of acute rheumatic fever, having been initiated by streptococcal infection (Benedek 1993). Those with a history of rheumatic fever are at greater risk of infective endocarditis, an infection and inflammation of the inner membrane of heart tissue. The end result of rheumatic fever or rheumatic heart disease is damage to the heart valves, primarily the mitral and aortic valve.

Both scarlet fever and rheumatic fever were common childhood illness in the United States in the nineteenth and early twentieth century, and rheumatic fever was the second most common cause of death among children age ten to nineteen as late as 1940 (Wolff 1948). Death rates from rheumatic and scarlet fever were very high at the beginning of the twentieth century, fluctuating considerably from year to year, but then falling beginning in the mid-1910s (see figs. 1.1 and 1.2). However, death rates from rheumatic fever remained high among children throughout the 1910s and 1920s. Salicylic acid was shown to be effective in the treatment of rheumatic fever in 1876 and was replaced by the better-tasting aspirin, first manufactured in 1889 (Vane 2000). From 1940 onward, as the link between streptococcal infections (treated with sulfa drugs and later with penicillin) and rheumatic fever became well established, deaths from rheumatic fever fell steadily for all age groups. Collins (1946) shows that the case rate of scarlet fever did not change from circa 1900 to 1945, but that case fatality rates fell sharply with the introduction of sulfa drugs in the late 1930s. Chapin (1926) hypothesized that earlier declines may have been due to decreases in the virulence of scarlet fever, perhaps because the adoption of effective isolation practices eliminated the more severe strains.

Special surveys and military examinations provide information about the incidence, history, and prevalence of scarlet fever, rheumatic fever, rheumatic heart disease, and valvular heart disease. Survey data from 1928–1931 reveal that 12 percent of those age twenty to twenty-four had ever had scarlet fever, most commonly prior to age fifteen (see table 1.1).

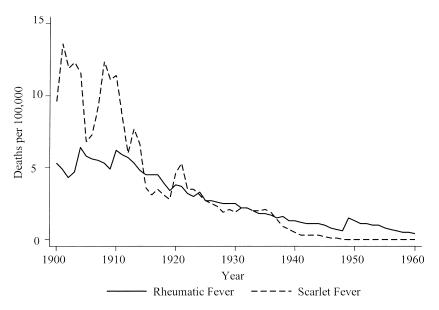


Fig. 1.1 Deaths per 100,000 from rheumatic fever and scarlet fever, 1900–1960 *Note:* Deaths are for death registrations states only. From various issues of *Vital Statistics of the United States.*

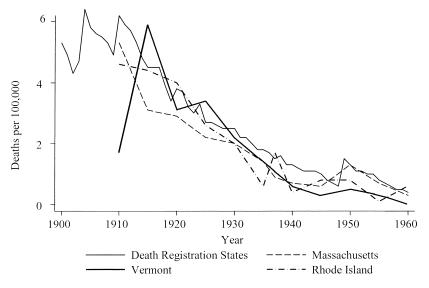


Fig. 1.2 Deaths from rheumatic fever, death registration states and selected states *Note:* From various issues of *Vital Statistics of the United States.*

	Ever had, living	Ever had, living and dead	Annual incidence in survey year
Rheumatic fever, 1935–1936			
Age 20–24	6.9	8.5	0.3
Age 15–19	5.3	6.0	0.4
Under age 15	3.2	3.4	5.6
Scarlet fever, 1928–1931			
Age 20–24	118.7		
Age 15–19	129.7		
Scarlet fever, 1935–1936			
Age 15–19	112.0		2.1
Under age 15	78.0		10.8
Heart disease, 1935–1936			
Age 20–24			0.8
Age 15–19			0.8
Under age 15			0.9

Table 1.1Incidence and history of rheumatic fever, scarlet fever, and heart disease
among white children (rates per 1000), 1928–1931 and 1935–1936

Notes: The rates for ever had for 1928–1931 are based upon a survey of 9,000 families as reported in Collins (1938) and those for 1935–1936 are based upon mother's answers to the survey question of the 1936 Communicable Disease Study as reported in Collins (1946, 1947) and Collins and Councell (1943). Past histories of rheumatic fever are for the children of the native-born. The living and dead category includes children who died of rheumatic fever and who, if they had survived, would have been age 20–24 in 1936. Annual incidence rates in the survey year are based both the 1936 Communicable Disease Study and upon the 1935–1936 National Health Survey as reported in Collins (1946, 1947). Annual incidence rates for heart disease are based upon the National Health Survey as reported in Collins (1946).

Table 1.1 also shows that less than 1 percent of those age twenty to twentyfour in 1935–1936 reported ever having had rheumatic fever. However, physical examinations of Connecticut seventh graders in 1934 and 1940 suggest much higher rates of rheumatic heart disease—4 percent in industrial cities, 3 percent in semi-industrial cities, and 1 percent in rural towns (Paul and Deutsch 1941). Among the young men examined for military service, 3 percent had valvular heart disease during World War I and 2 percent had either valvular heart disease or rheumatic heart disease during World War II (see table 1.2).

Other infectious diseases that affect cardiac functioning include latestage syphilis, measles, and typhoid fever. Studies of typhoid and measles patients in developing countries reveal electrocardiogram abnormalities (Khosla 1981; Olowu and Taiwo 1990). Measles and diphtheria can lead to myocarditis. Tertiary (late-stage) syphilis can result in aortitis and aneurisms. Infections that have been implicated in atherosclerosis include helicobacter pylori, a bacterium that causes gastritis and stomach ailments; chlamdyia pneumonia, a bacterium that causes acute upper and lower respiratory infections; Coxsackie B4 virus, generally causing symptoms no

	Acute rheumatic fever	Rheumatic heart disease	Valvular heart disease
WWI			32.1
WWII			
19,923 men prior to May 31, 1941		2.5	18.2
Nov. 1940–Sept. 1941			
All races	0.1	3.5	12.6
Whites	0.1	3.8	12.7
Blacks	0.0	1.5	11.7
Apr. 1942–Dec. 1943		3.6	12.9
All races		3.6	12.9
Whites		4.0	12.6
Blacks		1.2	15.0

Table 1.2 Rheumatic heart disease and valvular heart disease rates per 1,000 men examined for military service, World War I and World War II

Sources: Love and Davenport (1920); U. S. Selective Service System (1941, 1943, 1944).

Note: Valvular diseases of the heart include aortic insufficiency, aortic stenosis, mitral insufficiency, mitral stenosis, combined aortic and mitral lesions, pulmonic lesions, tricuspid lesions, and unclassified valvular lesions. This category excludes unspecified murmurs. Adding unspecified murmurs would increase the rate per 1,000 from 12.6 to 24.9 for Nov. 1940–Sept. 1941.

more serious than a common cold or sore throat; and some herpes viridae (see reviews by Lindholt et al. 1999; Valtonen 1991; Wong, Gallagher, and Ward 1999). The evidence linking chlamdyia pneumonia to atherosclerosis is the strongest; however, helicobacter pylori infection may be especially harmful when folate absorption is reduced, either because of decreased consumption of ascorbic acid or of folates (Markle 1997).

Estimates of prevalence rates in the first half of the twentieth century exist for some of the commonly recognized infections. Among those older than sixty-four interviewed in health surveys conducted in 1928–1931, 11 percent reported having had typhoid fever at some point in their lives and 8 percent reported having had diphtheria (Collins 1936, 1937). These numbers are probably underestimates. Tests in three cities in the 1920s showed that nearly 60 percent of adults had acquired immunity to diphtheria prior to any artificial immunization (cited in Collins 1937). Venereal disease was also widespread. At least 3 percent of men examined for military service during World War I had venereal diseases (Love and Davenport 1920) and reexaminations yielded higher prevalence rates, showing that 5 percent of men entering the army had syphilis and 23 percent had gonorrhea (Brandt 1987). The more careful examinations done by the military services during World War II included serological tests, and found that 4 percent of men had syphilis (U.S. Selective Service System 1943).

The writings of settlers allow us to trace the history of what has been

described as "the United States disease of the late nineteenth century"malaria (Innes 1993). Often confused with typhoid fever, even in the early twentieth century, and sometimes even confused with yellow fever and influenza, two malarial strains were common in the United States. Vivax malaria, introduced to local mosquitoes by European colonists in the early seventeenth century, spread into the upper Mississippi Valley as far north as Canada. The more malignant falciparum malaria, introduced by African slaves in the late seventeenth century, became the dominant strain in southern states such as South Carolina or Georgia that were south of the thirty-fifth parallel (Innes 1993; Humphreys 2001). Settlement of the Midwest caused an initial upsurge in malaria, followed by stability in the 1850s, and then another upsurge as the Civil War brought men into endemic regions and returned them to their local communities as carriers to start epidemics in areas that had been free of malaria for many years. By the 1880s and 1890s malaria in the upper Mississippi Valley began to recede as drainage efforts reduced mosquito breeding sites, the installation of screens on homes reduced transmission to human hosts, a growing livestock population diverted mosquitoes from human to animal hosts, and as transportation moved from water to rail (Ackernecht 1945; Humphreys 2001). Malaria then became solely a problem of the South, where it remained a problem in rural areas even into the 1940s. By World War II, blood tests of men examined for military service revealed that zero percent of the U.S. population was infected (U.S. Selective Service System 1943).

In modern populations lasting cardiac damage due to malaria is rare, but in some cases falciparum malaria can lead to myocarditis and persistent tachycardia (Charles and Bertrand 1982). Excessive doses of quinine, widely used in the nineteenth century to alleviate recurrent attacks of vivax malaria (but which only cures falciparum), can lead to fatal cardiotoxicity and produces cardiac symptoms such as tachycardia and bradycardia, particularly in patients with renal insufficiency or hypokaliemia (Charles and Bertrand 1982).

Both infectious disease and mechanical work stress play a role in musculoskeletal conditions. The joint inflammation of arthritis can result from a bacterial or viral infection, an autoimmune disease (rheumatoid arthritis), mechanical injury to a joint, or mechanical wear and tear that breaks down the joint cartilege (osteoarthritis). Musculoskeletal symptoms are common with many infections, including malaria, syphilis, rheumatic fever, influenza, variola, vaccinia, gonorrhea, mumps, and tuberculosis. With some injuries and diseases, the inflammation may never go away or may cause permanent damage.

Disease and injury can also cause brain damage. Common causes of memory loss include stroke, head trauma, epileptic seizures, brain infections such as herpes encephalitis, and Alzheimer's disease. Less common causes include cerebral malaria, which often results in damage to the subcortical white matter and the fronto-temporal areas of the neocortex, leading to depression, poor memory, personality change, and irritability and violence (Varney et al. 1997). Typhoid fever is accompanied by delirium, hallucinations, fluctuating moods, confusion, attention deficit disorder, and encephalitis (in rare cases) Ali et al. 1997). Encephalitis is also a rare complication of measles (Bergen 1998). Tuberculosis can involve the central nervous system in the form of subacute meningitis or intracerebral granulomas and result in focal epilepsy (Bergen 1998). Tyas et al. (2001) found that individuals who had received vaccinations for either influenza, tetanus, polio, or diphtheria faced less risk of Alzheimer's disease. Another cause of long-term memory loss is exposure to poisonous fumes (Tyas et al. 2001; Kishi et al. 1993). In addition, most studies find that individuals with fewer years of schooling are at greater risk of Alzheimer's disease (e.g., Tyas et al. 2001) and are less able to recover cognitive functioning after a stroke.

Some of the risk factors for memory loss were greater among Americans of the nineteenth and early twentieth centuries than among Americans today. Risk of death from violence fell from 1900 to 1960, regardless of whether homicide deaths are counted, suggesting that the risk of injury in both the workplace and the home has declined. Mortality from stroke in the past was also high. Ten percent of Union Army veterans age fifty to sixty-four in 1900 were dead of stroke by 1917. In contrast, a seventeenyear follow-up of Americans of the same age alive in the early 1970s showed no stroke deaths (Costa 2003). Stroke survival in 1900 was rare, but those who did survive were much more disabled than stroke survivors today. Having had a stroke increased the probability of paralysis by 0.7 among Union Army veterans age sixty to seventy-four in 1910, but among Americans in the late 1980s and early 1990s this probability increased by only 0.1 (Costa 2002).

Higher socioeconomic status has historically enabled men to buy lesscrowded housing, cleaner food and water, warmer clothes and shelter, more and better food, and less work away from home for pregnant women or mothers with small children. However, the evidence on the impact of income on mortality (for which I have much more extensive information than health) in historical United States populations is mixed. Wealth conveyed no systematic advantage for the survival of women and children in households matched in the 1850 and 1860 censuses (Steckel 1988). Preston and Haines (1991) used the question on the number of children ever born and the number of children still living in the 1900 census to report that place of residence and race were the most important correlates of child survival in the late nineteenth century, much more important than fathers' occupation. In contrast, early researchers emphasized the importance of social class. Rochester (1923) reported that within the high disease environment of U.S. cities there was a steep gradient between infant mortality and family income. Chapin (1924) reported that in Providence, Rhode Island in

1865 the annual crude death rate for taxpayers was eleven per thousand, while the corresponding rate for nontaxpayers was twenty-five per thousand. Recent research has also emphasized the importance of social class to mortality in past populations. Costa and Lahey (2005) found that among Union Army veterans observed at ages sixty to seventy-four, those of higher lifelong socioeconomic status were favored in survival. Ferrie (2003) linked households in 1850 and 1860 to one-year mortality rates as reported in the 1850 and 1860 censuses of mortality, and found a wealth gradient in rural areas of the United States. Those with greater personal property wealth were less likely to die from any cause and were less likely to die from consumption, a disease associated with crowding and poor housing. They were not more likely to die of cholera, a disease spread through contaminated water supplies, at a time when individuals did not know how to protect themselves.

The sample that I study—Union Army veterans—faced many unnatural situations during the course of their military service. The total number of deaths in the Civil War equaled the total number killed in almost all other U.S. wars combined, and more than one out of every five white men participating died, over half of them from disease (Vinovskis 1990). Union Army veterans who lived to 1910 had seen, on average, 13 percent of the men within their companies die during the war. In one company over half of the men died during the war. In addition, 4 percent of these survivors had personally experienced head injuries and had also experienced very high disease incidence. Twenty-nine percent of them had had diarrhea, 14 percent had had respiratory ailments, 12 percent had had rheumatic fever or experienced rheumatic athropathies, 6 percent had had measles, and another 6 percent had had typhoid (see table 1.3). Although according to wartime records only 4 percent of men had had malaria, records of the examining surgeons suggest that by 1910 7 percent had had malaria and 8 percent another fever (see table 1.3). Infectious diseases acquired in the army may have been more severe than those acquired in civilian life if repeated rapid passage increased the virulence of infectious agents. Contemporary observers noted that measles among Civil War troops was a much severer infection than that witnessed in the civilian population, often followed by such complications as chronic bronchitis, pneumonia, pleurisy, chronic diarrhea, and general debility (Cliff, Haggett, and Smallman-Raynor 1993).

Men who had been prisoners of war, as was true for 9 percent of those who survived to 1910, may have been at particularly high risk. Studies of World War II and Korean War POWs suggest that some of the sequelae of acute malnutrition include higher risks of death from ischemic heart disease, particularly after age seventy-five (Page and Brass 2001; Page and Ostfeld 1994), greater prevalence rates for duodenal ulcer and strongyloidiasis (Goulston et al. 1985), for neurological disorders (Gibberd and

	Rate/1,000
During War	
Diarrhea	292.7
Malaria	35.4
Respiratory	141.5
Measles	63.4
Tuberculosis	16.9
Typhoid	60.8
Rheumatic fever or rheumatic athropathies	115.0
Stomach ailments	15.7
Syphilis	13.6
Ever noted by examining surgeon	
Malaria	69.0
Any fevers, including scarlet fever, but excluding those due to	
malaria, typhoid, measles, rubella, mumps, and meningitis	75.5

Table 1.3History of infectious disease (rates per 1000), Union Army veterans age
60–79 in 1910

Simmonds 1980), and for psychiatric problems, particularly psychoneurosis (Beebe 1975).

Thus far I have stressed the positive relationship between early life conditions and chronic disease at older ages, but the relationship could be negative as well. If genetic susceptibility to death from infectious disease or other insults at early 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 morbidity rate of such a cohort may be lower relative to a cohort in which more genetically frail individuals survive. Additionally, cohorts who survive infectious disease may acquire partial or complete immunity and therefore may have lower mortality rates. Lee (2003) finds that under the extreme disease conditions of Union Army camp life, growing up in a large city (an extremely unhealthy locale circa 1860) relative to an isolated rural area had a beneficial mortality effect, because men from isolated areas lacked immunities to the diseases that ravaged Union Army camps. However, Costa (2003) and Costa and Lahey (2003) find that among soldiers who survived until 1900 or 1910, those who had grown up in a large city faced a shorter old age.

1.2.1 Data

The data used in this paper are drawn from the military records of the Union Army and from the Union Army pension program.² This pension program was the most widespread form of assistance to the elderly prior to

2. The data are available from http://www.cpe.uchicago.edu/ and were collected by a team led by Robert Fogel.

Social Security, covering 90 percent of all veterans by 1910 and benefiting an estimated 25 percent of the population older than sixty-four, whether as a couple consisting of the former soldier and his wife, the single or widowed veteran, or the widows of veterans (Costa 1998).

The Union Army pension program began in 1862, when Congress established the basic system of pension laws, known as the General Law pension system, to provide pensions to both regular and volunteer recruits who were severely disabled as a direct result of military service (see Costa 1998) for a history of the Union Army pension program). The Union Army pension program became a universal disability and old-age pension program for veterans with the passage of the Act of June 27, 1890, which specified that any disability entitled 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 instructed the examining surgeons in 1890 to grant a minimum pension to all men at least sixty-five years of age unless they were unusually vigorous. Veterans, however, had every incentive to undergo a complete examination, because those with a severe chronic condition, particularly if it could be traced to wartime experience, were eligible for larger pensions. The surgeons rated the severity of specific conditions using detailed guidelines provided by the Pension Bureau.

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 and 1910 censuses, which provide occupational information. The sample was drawn as a cluster sample of companies (roughly 100 men) and includes all enlisted men within a company. All men were linked to military records, which provide information on stress at young adult ages, such as prisoner of war status, whether the soldier was ever discharged for disability, and such illnesses as measles, diarrhea, tuberculosis, typhoid, rheumatism, acute respiratory infections (e.g., pneumonia, bronchitis), malaria, and war injuries. About 2 percent of these men were not yet collecting a pension in 1910, either because their applications had been rejected or because they had not yet applied for a pension. A surgeon's exam is available for 93 percent of all men who had a pension in 1910. Men for whom a surgeon's exam is missing tended to be men who entered at a late age and received a pension on the basis of age. I restrict the sample to men with a surgeon's record because I am interested in analyzing the effect of a specific condition as noted by the examining surgeon on later health outcomes. When I redefine my health outcome variables by assuming that men without a surgeon's exam had no chronic conditions, the conclusions I draw from my regression analyses about the importance of infectious disease, socioeconomic factors, and other variables remain unchanged.

Men who entered the Union Army were probably healthier than the

population as a whole. An examination of men who were rejected for military service suggests that mean height for the population was about 0.18 inches less than the mean of the recruits. Once men entered the service, rural farmers, who were the better-nourished segment of society, were more likely to die, because they lacked immunities to such common camp diseases as measles and typhoid (Lee 1997). However, men who survived the war (regardless of occupation) were only 0.02 inches shorter than all recruits at enlistment, suggesting that the war itself induced minimal survivorship selection on the basis of height and hence on early net nutritional status. Increased exposure to disease probably left men in worse health than when they entered the army, but by age fifty even men who had grown up in rural areas and had not served had probably been exposed to as many infectious diseases as veterans because of increased migration. Although little is known about the experience of Union Army veterans from the time they left the service until they appear on the pension rolls, several tests indicate that this sample is representative of the general population before the war in terms of wealth and circa 1900 in terms of mortality experience.³

I use the descriptions of the examining surgeons for heart, musculoskeletal, neurological, and infectious disease disorders. For heart disease the physician described pulse rate and heartbeat characteristics; whether a murmur was present and its timing, type, and location, and which valves were involved; whether there was enlargement, oedema, cyanosis, dyspnoea, or arteriosclerosis. I define valvular heart disease as any mention of either mitral or aortic valve murmurs. I define congestive heart failure as the contemporaneous mention of edema, cyanosis, and dyspne.⁴ Note that arteriosclerosis refers to peripheral arteriosclerosis (symptomatic not just of atherosclerosis but also of diabetes or inflammation). Descriptions of rheumatism include where the rheumatism was located and whether pain, tenderness, swelling, or crepitation was associated with the joint. Descriptions of nervous system disorders include descriptions of balance, aphasia, paralysis, reflexes, neuralgia, vertigo, headaches, seizures, memory loss, and any indications of loss of mental power, including mental illness. Descriptions of infectious disease include both diseases suffered while in the army and also diseases suffered while out of the army, including current ill-

3. Among all adult males age twenty and over in the households to which recruits were linked in the 1860 census, mean wealth was similar to that found in a random sample, suggesting that military service was not very selective of men of lower socioeconomic status. In fact, 95 percent of the sample consisted of volunteers. Cohort life expectancies of veterans who reached age sixty between 1901 and 1910 resemble the cohort life expectancies found in genealogies, and the distribution of deaths from specific causes for all veterans who died between 1905 and 1915 does not differ significantly from the distribution of expected number of deaths from those causes in the death registration states in 1910 (Costa 1998).

4. Although more restrictive definitions are possible (e.g., in the case of congestive heart failure, including cardiomegaly as a criterion and excluding coexisting respiratory infection and asthma), the results are robust to minor variations in definitions.

nesses. I use these descriptions of infectious disease as explanatory variables, classifying them into malaria and into descriptions of any fever, including scarlet fever.

With the exception of my infectious disease category, the symptoms, signs, and conditions that I examine did not require any diagnostic equipment that was unavailable to late nineteenth-century physicians. Because nineteenth-century definitions were looser and because nineteenth-century physicians were more accustomed to direct observation, this may bias past prevalence rates upward relative to recent prevalence rates. However, after 1907, when men received a pension on the basis of age, prevalence rates in the Union Army sample are likely to be understated. Prevalence rates may also be underestimated in the Union Army sample because men may have developed a condition between the time of the last exam and 1910 (see Costa 2000 and 2002 for a discussion of potential biases).

The disease reports from soldiers' military records tend to be terse, oneor two-word descriptions. I classify rheumatic fever and rheumatic athropathies as one category because the common description *rheumatism* could refer to either rheumatic fever, an underlying chronic condition, traumatic arthritis, or any viral infection accompanied by arthritic symptoms. The reports submitted by camp doctors to the surgeon general distinguish between acute and chronic rheumatism, and these suggest that 40 percent of all cases of rheumatism were acute and were mainly caused by rheumatic fever. However, some of the chronic cases may have been prolonged acute rheumatic fever (Bollet 1991).

Health Trends at Older Ages

Union Army veterans were already disabled by chronic conditions by age fifty. Among those on the pension rolls and with a surgeon's exam by 1895 (when program eligibility had already been broadened), 16 percent of men age fifty to fifty-nine had valvular heart disease and 44 percent of men in that age group had joint problems. As Union Army veterans aged, the burden of disability rose. By ages sixty-five to seventy-four, 29 percent had valvular heart disease, 10 percent had congestive heart failure, 13 percent had arteriosclerosis, 59 percent had joint problems, 51 percent had back problems, and 4 percent had memory loss (see table 1.4).

Compared to white men examined in the National Health and Nutrition Examination Surveys (NHANES), Union Army veterans aged prematurely by ten to twenty years. By ages sixty-five to seventy-four, Union Army veterans had the congestive heart failure rates of seventy-five to eighty-four-year-old men in the 1988–1994 NHANES III. Their rates of valvular heart disease at ages fifty to fifty-nine were already more than twice those of sixty-five to seventy-four-year olds in the 1976–1980 NHANES II (see fig. 1.3). At ages fifty-five to sixty-four, Union Army veterans looked like seventy-five to eighty-four-year-old men in 1988–1994 in

Ta	ble	1.4

Prevalence rates (%) among Union Army veterans by year and age

				Year (Age))		
	1895 (50–59)	1900 (55–64)	1905 (60–69)	1910 (65–74)	1915 (70–79)	1920 (75–84)	1925 (80–89)
Heart							
Murmur	26.99	35.34	42.42	44.77	44.24	44.48	51.48
Valvular	15.49	21.76	27.16	28.93	28.64	28.66	33.27
Congestive heart failure	0.59	3.82	7.99	10.18	10.20	10.05	11.61
Arteriosclerosis	0.95	2.12	6.57	10.79	12.79	14.93	28.44
Tachycardia	20.11	24.71	29.96	31.83	31.26	31.49	32.87
Bradychardia	2.58	3.67	5.00	5.70	6.17	5.98	7.38
Bounding pulse	8.77	12.19	16.49	17.35	17.17	16.77	15.75
Irregular heart rate	31.18	40.65	48.52	51.01	51.33	51.39	57.87
Musculoskeletal							
Joint problems	43.56	51.64	57.77	59.06	58.72	58.26	59.94
Back problems	36.00	43.57	49.58	50.91	50.62	50.65	49.80
Cognitive							
All mental problems	6.54	8.16	10.30	11.31	11.19	12.89	20.18
Memory loss	1.38	2.35	3.39	3.54	3.76	4.63	6.20

Note: Estimated for all men who were on the pension rolls in 1895 and with a surgeon's exam by 1895.

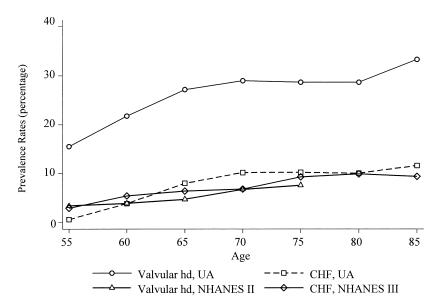


Fig. 1.3 Heart disease rates by age, Union Army veterans and white men in NHANES II and III

Note: Valvular heart disease rates in NHANES II are physician-reported. Congestive heart failure rates in NHANES III are self-reported. Disease rates in the Union Army are physician-reported. All NHANES rates are weighted using examination or interview weights. Ages are centered at the marks around a ten year age interval.

terms of joint problems (see fig. 1.4). At age fifty to fifty-nine they looked like sixty-five to seventy-four-year-old men in 1976–1980 in terms of back problems (see fig. 1.5).

Variation in chronic disease prevalence rates within the Union Army sample by infectious disease rates and by occupational class was even greater than variation in prevalence rates across the Union Army and more recent samples. Figures 1.6–1.8 show that men who were professionals and proprietors circa 1900 had the lowest rates of valvular heart disease, joint problems, and back problems at all ages than all other occupational groups. In the case of joint and back problems, at no age did professionals and proprietors ever resemble those in manual labor in terms of chronic conditions. Figures 1.9–1.11 show that those men who had had rheumatic fever during the war faced much higher prevalence rates of valvular heart disease, joint problems, and back problems at all ages than those who had not had rheumatic fever. A sixty-year-old man who had had rheumatic fever had a slightly higher prevalence rate of valvular heart disease than a seventy-year-old man who had not had rheumatic fever. A sixty-five-yearold man who had had rheumatic fever had a slightly higher prevalence rate than an eighty-five-year-old who had not had rheumatic fever. In the case of joint and back problems, at no age did those who had not had rheumatic fever have prevalence rates as high as those who had had rheumatic fever.

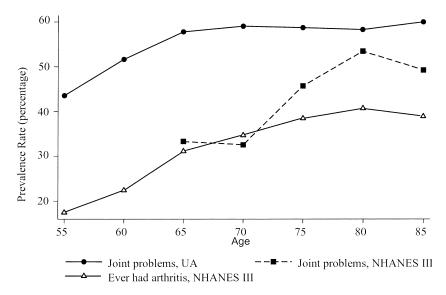


Fig. 1.4 Joint problems by age, Union Army veterans and white men in NHANES III

Note: Joint problems are physician-reported. "Ever had arthritis" is self-reported. Disease rates in the Union Army are physician-reported. All NHANES figures are weighted using examination or interview weights. Ages are centered at the marks around a ten year age interval.

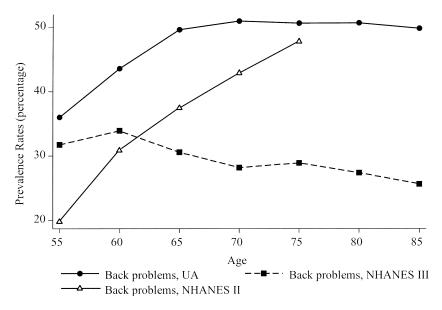


Fig. 1.5 Back problems by age, Union Army veterans and White Men in NHANES II and NHANES III

Note: Back problems in NHANES II are physician-reported. Back problems in NHANES III are self-reported. Disease rates in the Union Army are physician-reported. All NHANES figures are weighted using examination or interview weights. Ages are centered at the marks around a ten year age interval.

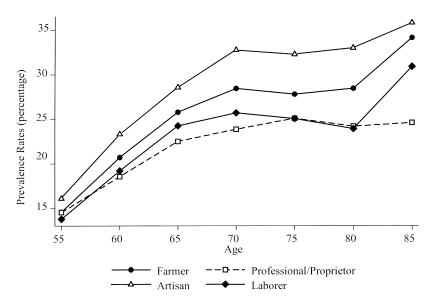


Fig. 1.6 Valvular heart disease prevalence rates by age and by occupational class circa 1900

Note: Ages are centered at the marks around a ten year interval.

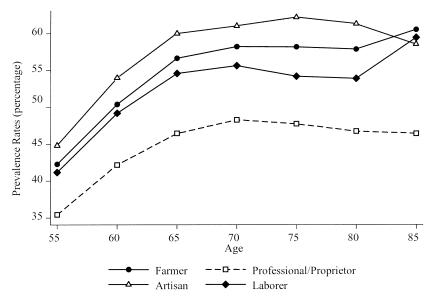


Fig. 1.7 Joint problems prevalence rates by age and by occupational class circa 1900

Note: Ages are centered at the marks around a ten year age interval.

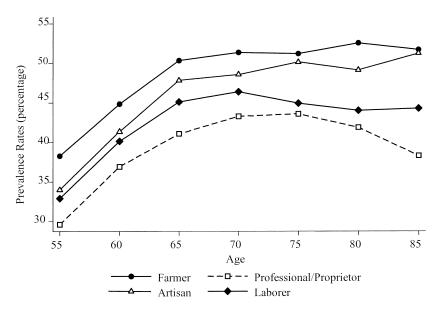


Fig. 1.8 Back problems prevalence rates by age and by occupational class circa 1900

Note: Ages are centered at the marks around a ten year age interval.

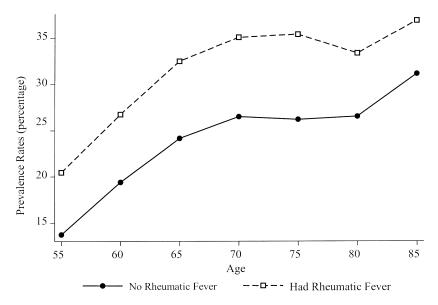


Fig. 1.9 Valvular heart disease prevalence rates by age and whether had rheumatic fever during the Civil War

Note: Ages are centered at the marks around a ten year age interval.

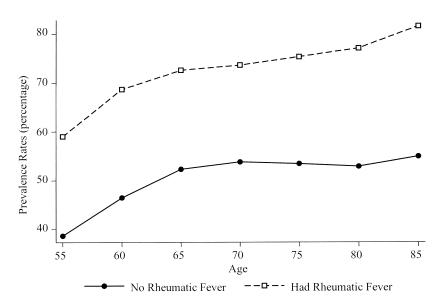


Fig. 1.10 Joint problems prevalence rates by age and whether had rheumatic fever during the Civil War

Note: Ages are centered at the marks around a ten year age interval.

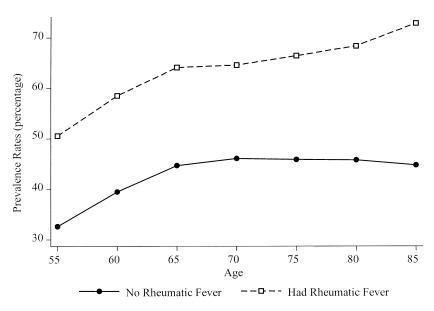


Fig. 1.11 Back problems prevalence rates by age and whether had rheumatic fever during the Civil War

Note: Ages are centered at the marks around a ten year age interval.

Results

I use probit regressions to determine what factors predicted the probability that a veteran would have one of three disease states in 1910. That is, I run regressions of the form

$$Pr(I = 1) = Pr(\varepsilon < X' \beta) = \Phi(X' \beta)$$

where I = 1 if a veteran had a specific chronic condition, sign, or symptom associated with one of the three disease states, $\Phi()$ is a standard normal cumulative distribution function, and **X** is a vector of control variables. The dependent variables are equal to 1 if the veteran has one of the following specific chronic conditions, signs, or symptoms: a heart murmur, valvular heart disease, arteriosclerosis, congestive heart failure, an irregular pulse, tachycardia, bradychardia, a bounding pulse, joint problems, back problems, any mental problems, and memory loss.

Rheumatic fever during wartime and an examining surgeon's record of either malaria or other infectious diseases increase the probability of heart disease, signs, or symptoms for all of our disease states among Union Army veterans age sixty to seventy-nine in 1910 (see tables 1.5 and 1.6). Having had rheumatic fever during the war increased the probability of valvular heart disease by 0.08 and the probability of both arteriosclerosis and congestive heart failure by 0.05. Having had malaria increased the

Table 1.5 Selected cardiovascular condition probits (marginal effects), Union Army veterans age 60–79 in 1910	lar condition pro	bits (margina)	l effects), Unio	n Army veteran	is age 60–79 in	1910		
	Murmur	nur	Valv	Valvular	Arteriosclerosis	clerosis	CHF	F
	$\frac{\partial P}{\partial x}$	Standard Error						
Dummy = 1 if occupation at enlistment Farmer								
Professional or proprietor	-0.062^{***}	0.023	-0.042^{*}	0.022	-0.027^{*}	0.015	-0.032^{**}	0.013
Artisan	-0.075^{***}	0.018	-0.043^{**}	0.017	-0.009	0.011	-0.017	0.010
Laborer	-0.039^{**}	0.016	-0.005	0.017	0.012	0.011	-0.010	0.011
Dummy = 1 if occupation circa 1900								
Farmer								
Professional or proprietor	-0.040^{**}	0.017	-0.032^{**}	0.016	-0.006	0.011	-0.017*	0.010
Artisan	0.031^{*}	0.019	0.034^{*}	0.019	-0.000	0.013	-0.009	0.011
Laborer	-0.000	0.016	-0.008	0.014	0.007	0.009	-0.008	0.009
Logarithm of personal wealth in 1860	-0.003	0.003	0.003	0.003	-0.000	0.002	-0.004^{**}	0.002
Dummy = 1 if writes	-0.010	0.026	-0.043*	0.023	-0.016	0.016	-0.012	0.017
Dummy = 1 if city size								
50,000+	0.007	0.017	-0.018	0.016	0.005	0.009	0.018^{*}	0.011
25,000-50,000	-0.018	0.033	-0.005	0.031	0.009	0.019	-0.000	0.020
2,500–25,000 < 2 500	0.011	0.018	-0.007	0.018	0.028^{***}	0.011	0.009	0.011
Dumy = 1 if in war had								
Diarrhea	0.023^{*}	0.014	-0.002	0.012	0.002	0.007	0.007	0.007
Malaria	-0.018	0.032	0.011	0.029	-0.010	0.017	0.003	0.018

(continued)

	Murmur	mur	Valvular	ular	Arteriosclerosis	clerosis	CHF	ц
	$\frac{\partial P}{\partial x}$	Standard Error						
Respiratory infection	0.035**	0.017	0.035**	0.016	0.002	0.011	0.023*	0.011
Measles	-0.034	0.023	-0.022	0.021	-0.008	0.014	-0.016	0.013
Tuberculosis	0.062	0.040	-0.016	0.035	0.011	0.027	0.022	0.027
Typhoid	0.011	0.027	0.023	0.024	0.000	0.014	0.012	0.015
Rheumatic fever	0.112^{***}	0.019	0.075^{***}	0.018	0.053^{***}	0.012	0.051^{***}	0.013
Stomach ailments	0.077	0.047	0.039	0.043	0.065^{**}	0.035	0.038	0.033
Syphilis	0.031	0.053	-0.027	0.043	0.039	0.034	-0.037	0.023
Dummy =1 if surgeons found evidence								
Malaria	0.161^{***}	0.023	0.137^{***}	0.022	0.042^{***}	0.017	0.058^{***}	0.020
Other infections	0.071^{***}	0.021	0.084^{***}	0.019	0.042^{***}	0.013	0.045^{***}	0.015
Proportion of company who died	0.046	0.080	0.034	0.091	-0.029	0.051	0.010	0.052
Dummy = 1 if POW	0.045**	0.022	0.057***	0.021	0.013	0.014	0.009	0.014
Pseudo \mathbb{R}^2	0.0	22	0.018	18	0.020	0	0.027	7
Observed P	0.436	36	0.285	85	0.103	3	0.095	5
Observations	7,349	49	7,349	49	7,349	6	7,349	6

(continued)

Table 1.5

war, dummies for quarter of birth, a dummy if occupation circa 1900 was unknown, and dummies if the individual was not linked to the 1900 or 1860 census.

*** Significant at the 1 percent level. ** Significant at the 5 percent level.

* Significant at the 10 percent level.

Table 1.6	Selected heart rate and pulse characteristic probits (marginal effects), Union Army veterans age 60–79 in 1910	pulse character	istic probits (m	arginal effects)	, Union Army	veterans age 60	79 in 1910		
		Irregular	ular	Tachycardia	ardia	Bradycardia	ardia	Bour	Bounding
		$\frac{\partial P}{\partial x}$	Standard Error						
Dummy = 1 if occ	Dummy = 1 if occupation at enlistment								
Farmer									
Professional or proprietor	proprietor	-0.044	0.028	0.022	0.027	-0.009	0.010	-0.041^{**}	0.017
Artisan	1	-0.081^{***}	0.022	0.006	0.019	-0.021^{***}	0.007	-0.014	0.015
Laborer		-0.025^{**}	0.021	-0.039^{***}	0.016	0.004	0.008	-0.017	0.014
Dummy = 1 if occupation circa 1900	upation circa 1900								
Farmer									
Professional or proprietor	roprietor	-0.047^{***}	0.019	-0.005	0.017	-0.005	0.008	0.037^{***}	0.013
Artisan		-0.005	0.022	0.010	0.019	0.007	0.009	0.004	0.015
Laborer		-0.022^{*}	0.017	0.016	0.015	0.000	0.007	0.010	0.012
Logarithm of perse	Logarithm of personal wealth in 1860	-0.012^{***}	0.003	-0.010^{***}	0.003	-0.002*	0.001	-0.004^{*}	0.002
Dummy = 1 if writes	es	-0.038	0.027	-0.017	0.023	-0.004	0.011	-0.014	0.019
Dummy = 1 if city	size								
50,000+		-0.017	0.018	-0.010	0.016	-0.001	0.007	0.008	0.012
25,000-50,000		-0.101^{***}	0.028	-0.099^{***}	0.032	-0.000	0.014	-0.001	0.024
2,500-25,000		-0.020	0.021	-0.003	0.017	0.000	0.007	0.017	0.015
< 2,500									
Dummy = 1 if in war had	ar had								
Diarrhea		0.029^{**}	0.013	0.010	0.012	0.004	0.006	0.011	0.009
Malaria		-0.000	0.036	-0.002	0.029	0.001	0.013	-0.026	0.019
Respiratory infection	ction	0.037**	0.017	0.036^{***}	0.015	0.005	0.008	0.025	0.013
									(continued)

	Irregular	ular	Tachycardia	cardia	Bradycardia	ardia	Bounding	ding
	$\frac{\partial P}{\partial x}$	Standard Error						
Measles	0.001	0.023	0.008	0.023	0.007	0.011	0.018	0.019
Tuberculosis	0.047	0.044	0.034	0.041	-0.015	0.016	0.007	0.034
Typhoid	0.028	0.024	0.019	0.024	-0.007	0.010	0.011	0.018
Rheumatic fever	0.101^{***}	0.018	0.070^{***}	0.018	0.016^{*}	0.009	0.049^{***}	0.016
Stomach ailments	0.104^{**}	0.049	0.060	0.044	-0.012	0.017	0.085^{**}	0.040
Syphilis	-0.029	0.050	0.052	0.045	-0.027	0.014	0.013	0.037
Dummy = 1 if surgeons found evidence								
Malaria	0.126^{**}	0.024	0.122^{***}	0.024	0.017	0.012	0.069^{***}	0.019
Other infections	0.088^{**}	0.021	0.079^{***}	0.020	0.030^{***}	0.012	0.037^{***}	0.016
Proportion of company who died	0.062	0.096	0.136^{*}	0.082	0.020	0.034	0.027	0.061
Dummy = 1 if POW	0.053^{**}	0.024	0.039^{*}	0.021	-0.004	0.008	0.007	0.017
Pseudo R^2	0.0	26	0.018	18	0.01	6	0.0	6
Observed P	0.4	0.492	0.302	02	0.054	4	0.165	55
Observations	7,3	49	7,34	49	7,34	6	7,34	6†

dummies for quarter of birth, a dummy if occupation circa 1900 was unknown, and dummies if the individual was not linked to the 1900 or 1860 census. *** Significant at the 1 percent level.

** Significant at the 5 percent level.

* Significant at the 10 percent level.

(continued)

Table 1.6

probability of valvular heart disease by 0.14 and having had another infection increased this probability by 0.08.

Other infectious disease conditions and proxies for infectious disease conditions affected selected heart conditions. Respiratory infections while in the army were statistically significant predictors of murmurs, valvular heart disease, congestive heart failure, irregular heartbeat, and tachycardia. Stomach ailments while in the army increased the probability of arteriosclerosis. Men who had faced the severe malnutrition and harsh disease environment of POW camps were more likely to have valvular heart disease and to exhibit murmurs, irregular pulse, and tachycardia.

Rheumatic fever and malaria were also strong predictors of joint and back problems (see table 1.7). Rheumatic fever while in the army increased the probability of joint problems by 0.17 and of back problems by 0.15. Malaria noted by an examining surgeon increased the probability of joint problems by 0.10 and of back problems by 0.12. Other infections increased the probability of back problems by 0.05.

Wartime typhoid fever, tuberculosis, malaria, or other infectious diseases as noted by the examining surgeons predicted mental problems (see table 1.8). Those who had had typhoid fever saw their probability of all mental problems increase by 0.03, and those who had had tuberculosis saw their probability of memory loss increase by 0.03. Malaria increased the probability of all mental problems by 0.04 and of memory loss by 0.02. Other infections increased the probability of mental problems by 0.06 and of memory loss by 0.02.

Other predictors of all mental problems and of cognitive functioning included having had a head wound during the war, having had a stroke at older ages, having epilepsy, and, in the case of memory loss, wartime stress as measured by the percentage of the company who died during the war. A head wound during the war increased the probability of all mental problems by 0.09 and of memory loss by 0.06. Having had a stroke increased the probability of all mental problems by 0.45 and of memory loss by 0.28. Having epilepsy increased the probability of all mental problems by 0.42 and of memory loss by 0.22. An increase of a standard deviation (0.086) in the percentage of the company that died increased the probability of memory loss by 0.006.

I find that socioeconomic factors were important predictors of all of the three disease categories that I study. Controlling for occupation circa 1900, men who were professionals and proprietors at enlistment were significantly less likely to have valvular heart disease, arteriosclerosis, congestive heart failure, and back problems compared to men who were farmers or laborers. Controlling for occupation at enlistment, men who were professionals and proprietors circa 1900 were significantly less likely to have valvular heart disease, congestive heart failure, and joint or back problems than men who were farmers or laborers circa 1900. Men with

	Joint Pr	oblems	Back Pr	oblems
	$\frac{\partial P}{\partial x}$	Standard Error	$\frac{\partial P}{\partial x}$	Standard Error
	0.7	-	0,7	
Dummy = 1 if occupation at enlistment Farmer				
	-0.033	0.026	-0.064**	0.027
Professional or proprietor Artisan	-0.033 -0.002	0.020	-0.004 -0.048**	0.027
Laborer	_0.002 0.035*	0.017	0.002	0.020
	0.035*	0.019	0.002	0.020
Dummy = 1 if occupation circa 1900 Farmer				
	-0.094***	0.019	-0.067***	0.017
Professional or proprietor				
Artisan Laborer	$0.018 \\ -0.005$	0.020 0.016	$-0.006 \\ -0.001$	0.020 0.017
Logarithm of personal wealth in 1860	-0.001	0.003	0.006**	0.003
Dummy = 1 if writes	0.027	0.025	0.025	0.025
Dummy = 1 if city size	0.011	0.015	0.004	0.016
50,000+	0.011	0.015	-0.004	0.016
25,000-50,000	0.007	0.028	-0.010	0.033
2,500-25,000	0.013*	0.017	-0.006	0.018
< 2,500				
Dummy = 1 if in war had				
Diarrhea	-0.009	0.012	0.013	0.014
Malaria	0.024	0.030	0.012	0.033
Respiratory infection	-0.036^{**}	0.018	-0.031*	0.017
Measles	-0.023	0.022	0.001	0.024
Tuberculosis	-0.004	0.042	-0.023	0.043
Typhoid	-0.027	0.026	-0.011	0.024
Rheumatic fever	0.170***	0.017	0.147***	0.017
Stomach ailments	-0.008	0.047	0.054	0.041
Syphilis	0.038	0.050	-0.001	0.048
Dummy = 1 if surgeons found evidence				
Malaria	0.099***	0.023	0.121***	0.023
Other infections	0.005	0.021	0.050***	0.021
Proportion of company who died	-0.121*	0.074	-0.081	0.082
Dummy = 1 if POW	0.011	0.019	-0.009	0.023
Pseudo R^2	0.0	24	0.0	20
Observed P	0.6	15	0.5	18
Observations	7,3	49	7,3	49

Table 1.7 Musculoskeletal characteristic probits (marginal effects), Union Army veterans age 60–79 in 1910

Note: Robust standard errors, clustering on the company. Additional control variables include a dummy equal to one if the veteran was ever wounded in the war, dummies for quarter of birth, a dummy if occupation circa 1900 was unknown, and dummies if the individual was not linked to the 1900 or 1860 census.

*** Significant at the 1 percent level.

** Significant at the 5 percent level.

* Significant at the 10 percent level.

	All mental	problems	Memo	ry loss
	$\frac{\partial P}{\partial P}$	Standard	$\frac{\partial P}{\partial P}$	Standard
	∂x	Error	∂x	Error
Dummy = 1 if occupation at enlistment				
Farmer				
Professional or proprietor	0.019	0.017	0.008	0.009
Artisan	0.014	0.013	0.009	0.007
Laborer	0.002	0.011	-0.004	0.004
Dummy = 1 if occupation circa 1900				
Farmer				
Professional or proprietor	0.026**	0.012	-0.001	0.005
Artisan	0.017	0.013	-0.003	0.005
Laborer	0.007	0.010	0.005	0.005
Logarithm of personal wealth in 1860	-0.002	0.002	-0.002^{**}	0.001
Dummy = 1 if writes	-0.060^{***}	0.018	-0.015*	0.010
Dummy = 1 if city size				
50,000+	0.016	0.010	0.005	0.004
25,000-50,000	0.018	0.022	-0.003	0.008
2,500-25,000	0.004	0.011	0.002	0.005
< 2,500				
Dummy = 1 if in war had				
Diarrhea	0.018**	0.009	-0.004	0.004
Malaria	-0.018	0.017	0.003	0.009
Respiratory infection	-0.014*	0.011	0.001	0.005
Measles	-0.002	0.016	-0.001	0.006
Tuberculosis	0.016	0.031	0.027*	0.020
Typhoid	0.029*	0.018	-0.004	0.006
Rheumatic fever	-0.007	0.011	-0.001	0.005
Stomach ailments	0.045	0.034	0.017	0.020
Syphilis	0.044	0.035	0.014	0.018
Dummy = 1 if surgeons found evidence				
Malaria	0.044***	0.017	0.016**	0.009
Other infections	0.060***	0.017	0.016***	0.007
Dummy = 1 if head wound in war	0.086***	0.023	0.064***	0.017
Proportion of company who died	0.050	0.049	0.070***	0.019
Dummy = 1 if POW	0.008	0.014	-0.002	0.005
Dummy = 1 if had stroke	0.464***	0.031	0.277***	0.029
Dummy = 1 if epilepsy	0.419***	0.057	0.217***	0.042
Pseudo R^2	0.1	36	0.2	49
Observed P	0.1	17	0.0	39
Observations	7,3	49	7,3	49

Cognitive characteristic probits (marginal effects), Union Army veterans age 60–79 in 1910

Note: Robust standard errors, clustering on the company. Additional control variables include a dummy equal to one if the veteran was ever wounded in the war, dummies for quarter of birth, a dummy if occupation circa 1900 was unknown, and dummies if the individual was not linked to the 1900 or 1860 census.

*** Significant at the 1 percent level.

Table 1.8

** Significant at the 5 percent level.

* Significant at the 10 percent level.

higher personal property wealth in 1860 were significantly less likely to have developed congestive heart failure and arrythmias by 1910 and to have experienced memory loss. The illiterate were more likely to have mental problems or memory loss by 1910.

Predicting Declines in Chronic Conditions

How much of the difference in chronic disease rates between Union Army veterans and men in the late twentieth century is explained by infectious disease prevalence and socioeconomic conditions? In this section, I consider the case of valvular heart disease and of arthritis, both of which exhibited remarkable declines in prevalence rates. Among white men age sixty to seventy-nine in the 1988–1994 NHANES, the prevalence of arthritis was 38.4 percent compared to a predicted prevalence of 61.5 among Union Army veterans. Among white men age sixty to seventy-four (older age groups were not examined) in the 1976–1980 NHANES, the prevalence of valvular heart disease was 5.4 percent, compared to a predicted prevalence of 28.5 among Union Army veterans.

I use the regressions for Union Army veterans to calculate lower-bound estimates of the declines due to differences in the disease environment and to changes in the occupational distribution. Thus I assume that I could decrease rheumatic fever rates from 11.5 percent to only 0.7 percent, equal to the percentage age twenty to twenty-four who in 1935-1936 reported ever having rheumatic fever (see table 1.1). I also assume that I could decrease malaria rates from 6.9 to 0.3 percent, equal to the percentage of individuals in the 1928-1931 health surveys who reported having had malaria in the past twelve months (Collins 1944). Because, as previously noted, the case fatality of scarlet fever declined sharply, I further assume that the coefficient on fever becomes zero, but that the other coefficients remain unchanged. Finally, I increase the proportion of white collar workers in our sample and decrease the proportion of laborers and of farmers by assuming that the occupational distribution of Union Army veterans at enlistment was the same as that of men in 1940 and that the occupational distribution of Union Army veterans circa 1900 was the same as that of men in 1980. Table 1.9 presents the results of my prediction exercise for Union Army veterans.

Table 1.9 shows that decreases in the prevalence of the specific infectious diseases that I examine and changes in the occupational distribution would decrease the prevalence of valvular heart disease from 28.5 to 23.6 percent and of arthritis from 61.5 to 55.9 percent. Given that the prevalence of valvular heart disease fell by roughly 22.5 percentage points and that the prevalence of arthritis fell by 23.1 percentage points, I can explain roughly 22 percent of the decline in valvular heart disease and 24 percent of the decline in arthritis. The effects of disease declines and of occupational shifts are of roughly similar magnitudes.

	Valvular heart disease (%)	Arthritis (%)
Predicted, based upon actual values	28.5	61.5
If decline in rheumatic fever from 11.5% to 0.7%	27.7	59.6
If decline in malaria from 6.9% to 0.3%	27.5	60.7
If the coefficient on fever becomes 0	27.8	61.5
Predicted, all of above three effects	25.9	58.7
Shift in occupational distribution	26.1	58.8
Infectious disease changes and occupational distribution	23.6	55.9

Table 1.9 Predicted prevalence of valvular heart disease and arthritis among Union Army veterans age 60–79 in 1910

Note: Based upon the regressions shown in tables 1.5 and 1.7. See the text for details.

1.3 Conclusion

Past populations who lived at a time when medical care was ineffective at best provide us a unique opportunity to study the effects of untreated infectious disease and physical injuries on chronic disease. I have shown that in the past, occupation was an important determinant of valvular heart disease, congestive heart failure, and joint and back problems, suggesting that higher socioeconomic status protected against mechanical wear and tear and that it purchased less crowding and therefore less infectious disease, such as rheumatic fever. Infectious diseases such as rheumatic fever and malaria predicted various heart and musculoskeletal conditions. Prisoner of War (POW) status, perhaps a proxy for both nutritional deprivation and infectious disease, predicted various heart conditions, including valvular heart disease. In addition, respiratory disease predicted various heart conditions, and typhoid, tuberculosis, and malaria predicted mental problems. Stroke was a particularly important predictor of mental problems and of memory loss, illustrating the value of stroke therapies. Additional predictors of memory loss included illiteracy, head wounds, and the percentage of a veteran's company who had died during the war, suggesting that wartime trauma also played a role.

My findings suggest that the high probability of physical injury on the job and in the home in the past, the high rates of infectious disease, and incomes that were too low to purchase uncrowded housing all explain why chronic disease rates were historically so high among men. Declining disease rates and the shift from blue-collar to white-collar jobs explain at least 22 percent of the decline in valvular heart disease since 1900 and 24 percent of the decline? I am probably underestimating the portion of the decline due to infectious disease because I cannot observe childhood illnesses. Additional potential explanations include the mechanization of blue-collar jobs and reductions in work injuries within jobs, innovations in

medical care, and the diffusion of accurate medical knowledge to individuals, and improvements in the consumed food supply.

Why did infectious disease rates, which played such an important role in the decline in chronic disease rates, fall? Early work emphasized advances in medical technology, rising incomes and living standards, public health reforms, improved personal hygiene, and natural factors such as the declining virulence of pathogens (United Nations 1953 and 1973). McKewon (1976), arguing by a process of elimination, upset this consensus view and claimed that because mortality declines began prior to any changes in medical technology or in public health reforms, the primary explanation had to be improved nutrition. Fogel (1997) argued for the importance not of nutrition per se, but rather of net nutrition—that is, the difference between food intake and the demand made on that intake by disease, climate, and work. Those with parasitic diseases suffer depletion of iron supplies despite their consumption of an otherwise healthy diet. Recurrent sufferers from gastrointestinal diseases cannot digest all of the ingested nutrients.

Recent work on public health reforms that has utilized microdata or city-level data has emphasized the efficacy of these reforms. While there may have already been a declining trend in such water-borne diseases as typhoid or diarrhea, cities' sanitary reforms led to substantial declines in death rates and disproportionately benefited the poor because they had neither the knowledge nor the income to protect themselves in a highdisease environment (Cain and Rotella 2001; Troesken 2004; Costa and Kahn 2006). In addition to large investments in sewage, clean water, and a clean milk supply, cities also invested in disease reporting and quarantining systems and, in conjunction with private philanthropists, in well-baby and -child care (including vaccination), and in campaigns against specific diseases such as syphilis. While doctors may not have been able to prescribe penicillin or antibiotics, they did learn how to quarantine patients to prevent diseases from quickly spreading and hence also from becoming particularly virulent as they jumped from patient to patient. Public health efforts were not limited to large cities. There were various health campaigns in rural areas, the most notable that against hookworm in the rural South, a campaign that also disproportionately benefited the poor (Bleakley 2007). Foods were fortified with iron. There is clearly much more work to be done quantifying the contributions of various health campaigns and innovations in care. Only when this quantification is complete will we be able to determine with certainty why infectious disease rates fell.

References

Ackerknecht, E. H. 1945. Malaria in the upper Mississippi Valley, 1760–1900. Baltimore, MD: The Johns Hopkins Press.

- Ali, G. S., M. A. Rashid, P. A. Kamli, Shah, and G. Q. Allagaband. 1997. Spectrum of neuropsychiatric complications in 791 cases of typhoid fever. Tropical Medicine and International Health 2 (4): 314-8.
- Allaire, S. H., M. P. LaValley, S. R. Evans, G. T. O'Connor, M. Kelly-Hayes, R. F. Meenan, D. Levy, D. T. Felson. 1999. Evidence for decline in disability and improved health among persons aged 55 to 70 Years: The Framingham Heart Study. American Journal of Public Health 89 (11): 1678-83.
- Beebe, G. W. 1975. Follow-up studies of World War II and Korean War prisoners. II. Morbidity, disability, and maladjustments. American Journal of Epidemiology 101 (5): 400-422.
- Benedek, Thomas G. 1993. Rheumatic fever and rheumatic heart disease. In The Cambridge world history of human disease, ed. Kenneth F. Kiple, 970-77. Cambridge: Cambridge University Press.
- Bergen, D. C. 1998. Preventable neurological diseases worldwide. Neuroepidemiology 17 (2): 67–73.
- Bleakley, Hoyt. 2007. Disease and development: Evidence from hookworm eradication in the American South. Quarterly Journal of Economics 122, 1:73-117.
- Bollet, Alfred Jay. 1991. Rheumatic diseases among Civil War troops. Arthritis and Rheumatism 34 (9): 1197–1203.
- Brandt, Allan M. 1987. No magic bullet: A social history of venereal disease in the United States since 1880. New York: Oxford University Press.
- Cain, Louis P., and Elyce J. Rotella. 2001. Death and spending: Urban mortality and municipal expenditure on sanitation. Annales De Demographie Historique 1:139-54.
- Chapin, Charles V. 1924. Deaths among taxpayers and non-taxpayers, income tax, Providence, 1865. American Journal of Public Health 14:647-51.

. 1926. Changes in type of contagious disease with special reference to smallpox and scarlet fever. Journal of Preventive Medicine 1 (September): 1–29.

- Charles, D., and E. Bertrand. 1982. Coeur et paludisme. Medicine tropicale: Revue du corps de Sante Colonial 42 (4): 405–9.
- Chirikos, T. N. 1986. Accounting for the historical rise in work-disability prevalence. The Milbank Quarterly 64:271–301.
- Cliff, Andrew, Peter Haggett, and Matthew Smallman-Raynor. 1993. Measles: An historical geography of a major human viral disease from global expansion to local retreat, 1840-1990. Oxford, UK: Blackwell Reference.
- Collins, Selwyn D. 1936. History and frequency of typhoid fever immunizations and cases in 9,000 families. Based on nation-wide periodic canvasses, 1928-31. United States Public Health Service. Public Health Reports 51 (28): 897-926.

. 1937. History and frequency of diptheria immunizations and cases in 9,000 families. Based on nation-wide periodic canvasses, 1928-31. United States Public Health Service. Public Health Reports 51 (51): 1736-73.

. 1938. History and frequency of clinical scarlet fever cases and of injections for artificial immunization among 9,000 families. Based on nation-wide periodic canvasses, 1928-31. United States Public Health Service. Public Health Reports 53 (11): 409-27.

-. 1944. The incidence of illness and the volume of medical services among 9,000 canvassed families. Washington, DC: Federal Security Agency, United States Public Health Service.

. 1946. Diptheria incidence and trends in relation to artificial immunization, with some comparative data for scarlet fever. United States Public Health Service. Public Health Reports 61 (7): 203-40.

-. 1947. The incidence of rheumatic fever as recorded in general morbidity surveys of families. United States Public Health Service. Public Health Reports. Supplement. No. 198.

51

- Collins, Selwyn D., and Clara Councell. 1943. Extent of immunization and case histories for diptheria, smallpox, scarlet fever, and typhoid fever in 200,000 surveyed families in 28 large cities. United States Public Health Service. *Public Health Reports* 58 (30): 1121–51.
- Colvez, A., and M. Blanchet. 1981. Disability trends in the United States population 1966–76: Analysis of reported causes. *American Journal of Public Health* 71 (5): 464–71.
- Costa, Dora L. 1998. The evolution of retirement: An American economic history, 1880–1990. Chicago: University of Chicago Press.
- ——. 2000. Understanding the twentieth-century decline in chronic conditions among older men. *Demography* 37 (1): 53–72.
- ——. 2002. Changing chronic disease rates and long-term declines in functional limitation among older men. *Demography* 39 (1): 119–38.
- ------. 2003. Understanding mid-life and older age mortality declines: Evidence from Union Army veterans. *Journal of Econometrics* 112 (1): 175–92.
- Costa, Dora L., and Matthew E. Kahn. 2006. Public health and mortality: What can we learn from the past? In *Public policy and the income distribution*, Alan J. Auerbach, David Card, and John M. Quigley, ed. New York: Russell Sage Foundation.
- Costa, Dora L., and Johanna N. Lahey. 2005. Becoming oldest-old: Evidence from historical U.S. data. *Genus* 61 (1): 125–26.
- Costa, Dora L., and Richard H. Steckel. 1997. Long-term trends in health, welfare, and economic growth in the United States. In *Health and welfare during industrialization*, ed. R. Floud and R. H. Steckel, 47–89. Chicago: University of Chicago Press.
- Crimmins, E. M. 1990. Are Americans healthier as well as longer-Lived? *Journal of Insurance Medicine* 22:89–92.
- Crimmins, E. M., Y. Saito, and S. L. Reynolds. 1997. Further evidence on trends in the prevalence and disability and incidence of disability among older americans from two sources: The LSOA and the NHIS. *Journals of Gerontology, Series B, Pyschological Sciences and Social Sciences* 52 (2): S59–S71.
- Ferrie, Joseph P. 2003. The rich and the dead: Socioeconomic status and mortality in the United States, 1850–1860. In *Health and labor force participation over the life cycle: Evidence from the past*, ed. D. L. Costa, 11–50. Chicago: The University of Chicago Press.
- Fogel, Robert W. 1997. Secular trends in nutrition and mortality. In *Handbook of population and family economics* Vol. 1A, ed. Mark R. Rosenzweig and Oded Stark, 434–81. Amsterdam: Elsevier.
- Fogel, Robert W., and Dora L. Costa. 1997. A theory of technophysio evolution, with some implications for forecasting population, health care costs, and pension costs. *Demography*, 34 (1): 49–66.
- Freedman, Victoria A., and Linda G. Martin. 1998. Understanding trends in functional limitations among older Americans. *American Journal of Public Health* 88 (10): 1457–62.
- ——. 2000. The contribution of chronic conditions to aggregate changes in oldage functioning. *American Journal of Public Health* 90 (11): 1755–60.
- Freedman, Victoria A., Linda G. Martin, and Robert F. Schoeni. 2002. Recent trends in disability and functioning among older adults in the United States: A systematic review. *Journal of the American Medical Association* 288 (24): 3137–46.
- Fries, J. F. 1980. Aging, natural death, and the compression of morbidity. New England Journal of Medicine 303:130–36.
- Fries, J. F. 1989. The compression of morbidity: Near or far? *Milbank Quarterly* 67 (2): 208–32.
- Gibberd, F. B., and J. P. Simmonds. 1980. Neurological disease in ex-far-east prisoners of war. *Lancet* 2 (8186): 135–37.

- Goulston, K. J., O. F. Dent, P. H. Chapuis, G. Chapman, C. I. Smith, A. D. Tait, and C. C. Tenant. 1985. Gastrointestinal morbidity among World War II prisoners of war: 40 years on. *Medical Journal of Australia* 143 (1): 6–10.
- Gruenberg, E. M. 1977. The failures of success. *Milbank Memorial Fund Quarterly* (Winter): 3–24.
- Humphreys, Margaret. 2001. *Malaria: Poverty, race, and public health in the United States.* Baltimore, M. D. and London: The Johns Hopkins University Press.
- Innes, Frank C. 1993. The geography of human disease: North America. In *The Cambridge world history of human disease*, ed. Kenneth F. Kiple, 519–34. Cambridge: Cambridge University Press.
- Khosla, S. N. 1981. The heart in enteric (typhoid) fever. *Journal of Tropical Medicine and Hygiene* 84 (3): 125–31.
- Kishi, R., R. Doi, Y. Fukuchi, H. Satoh, T. Satoh, A. Ono, F. Moriwaka, K. Tashiro, and N. Takahata. 1993. Subjective symptoms and neurobehavioral performances of ex-mercury miners at an average of 18 years after the cessation of chronic exposure to mercury vapor. Mercury Workers Study Group. *Environmental Research* 62 (2): 289–302.
- Lee, Chulhee. 1997. Socioeconomic background, diesase, and mortality among Union recruits: Implications for economic and demographic history. *Explorations in Economic History* 34:27–55.
- Lindholt, J. S., H. Fasting, E. W. Henneberg, and L. Ostergaard. 1999. A review of chlamydia pneumoniae and atherosclerosis. *European Journal of Vascular and Endovascular Surgery* 17 (4): 283–89.
- Love, Albert G., and Charles B. Davenport. 1920. *Defects found in drafted men. Statistical information compiled from the draft records showing the physical condition of the men registered and examined in pursuance of the requirements of the Selective-Service Act.* Washington, D.C.: Government Printing Office.
- Manton, Kenneth G. 1982. Changing concepts of morbidity and mortality in the elderly population. *Milbank Memorial Fund Quarterly* 60 (2): 183–244.
- Markle, H. V. 1997. Coronary artery disease associated with helicobacter pylori infection is at least partially due to inadequate folate status. *Medical Hypotheses* 49 (4): 289–92.
- Medlineplus. n.d. Medical Encyclopedia. United States National Library of Medicine and United States National Institutes of Health. http://www.nlm.nih.gov/ medlineplus/
- McKewon, Thomas. 1976. The modern rise of population. London: Edward Arnold.
- Olowu, A. O., and O. Taiwo. 1990. Electrocardiographic changes after recovery from measles. *Tropical Doctor* 20 (3): 123–26.
- Page, W. F., and A. M. Ostfeld. 1994. Malnutrition and subsequent ischemic heart disease in former prisoners of war of World War II and Korean conflict. *Journal* of Clinical Epidemiology 47 (12): 1437–41.
- Page, W. F., and Brass, L. M. 2001. Long-term heart disease and stroke mortality among former American prisoners of war of World War II and the Korean conflict: Results of a 50-year follow-up. *Military Medicine* 166 (9): 803–8.
- Paul, John R., and Joyce V. Deutsch. 1941. Rheumatic fever in Connecticut: A general survey. In *Rheumatic fever in Connecticut*, 1–33. Hartford, CT: Connecticut State Department of Health.
- Preston, Samuel H., and Michael R. Haines. 1991. *Fatal years: Child mortality in late nineteenth-century America.* Princeton: Princeton University Press.
- Rochester, Anna. 1923. Infant mortality: Results of a field study in Baltimore, MD. Based on births in one year. U.S. Department of Labor, Children's Bureau. Bureau Publication no. 119. Washington, D.C.: Government Printing Office.
- Steckel, Richard H. 1998. The health and mortality of women and children, 1850–60. *Journal of Economic History* 48 (2): 333–45.

Troesken, Werner. 2004. Water, race, and disease. Cambridge, MA: MIT Press.

Tyas, Suzanne L., Jure Manfreda, Laurel L. Strain, and Patrick R. Montgomery. 2001. Risk factors for Alzheimer's Disease: A population-based longitudinal study in Manitoba, Canada. *International Journal of Epidemiology* 30 (3): 590–97.

United Nations. 1953. The determinants and consequences of population trends. *Population Studies*, no. 17. New York: United Nations.

———. 1973. The determinants and consequences of population trends. *Population Studies*, no. 50. New York: United Nations.

United States Selective Service System. 1941. Analysis of reports of physical examination: Summary of data from 19,923 reports of physical examination. Medical Statistics Bulletin no. 1. Washington DC: U.S. Selective Service System.

——. 1943. Causes of rejection and incidence of defects: Local board examinations of selective service registrants in peacetime. An analysis of reports of physical examination from 21 selected states. Medical Statistics Bulletin no. 2. Washington DC: U.S. Selective Service System.

—. 1944. Physical examinations of selective registrants during wartime: An analysis of reports for the continental United States and each state. April 1942–December 1943. Medical Statistics Bulletin no. 3. Washington DC: U.S. Selective Service System.

- Valtonen, V. V. 1991. Infection as a risk factor for infarction and atherosclerosis. *Annals of Medicine* 23 (5): 539–43.
- Vane, Sir John. 2000. Aspirin and other anti-inflammatory drugs. *Thorax* 55 (Suppl 2): S3–S9.
- Varney, N. R., R. J. Roberts, J. A. Springer, S. K. Connell, and P. S. Wood. 1997. Neuropsychiatric sequelae of cerebral malaria in Vietnam veterans. *The Journal* of Nervous and Mental Disease 185 (11): 695–703.
- Vaupel, James H. 1991. The impact of population aging on health and health care costs: Uncertainties and new evidence about life expectancy. Odense, Denmark: Odense University, Center for Health and Social Policy.
- Verbrugge, L. M. 1984. Longer life but worsening health? Trends in health and mortality of middle aged and older persons. *Milbank Quarterly* 62:475–519.
- Vinovskis, Maris. 1990. Have social historians lost the Civil War? Some preliminary demographic speculations. In *Toward a history of the American Civil War*, ed. Maris Vinovskis, 1–30. Cambridge: Cambridge University Press.
- Waidmann, T., J. Bound, and M. Schoenbaum. 1995. The illusion of failure: Trends in self-reported health of the US elderly. *Milbank Quarterly* 73 (2): 253–87.
- Waidmann, T. A., and K. Liu. 2000. Disability trends among elderly persons and disability trends for the future. *The Journals of Gerontology, Series B, Psychological and Social Sciences* 55 (5): S298–307.
- Wolff, George. 1948. *Childhood mortality from rheumatic fever and heart disease*. Federal Security Agency. Social Security Administration. Children's Bureau Pub. 322. Washington D.C.: Government Printing Office.
- Wong, Y. K., P. J. Gallagher, and M. E. Ward. 1999. Chlamydia pneumoniae and atherosclerosis. *Heart* 81 (3): 232–38.