BECOMING OLDEST-OLD:
EVIDENCE FROM HISTORICAL U.S. DATA

Dora L. Costa
Joanna Lahey

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ABSTRACT

We argue that the environment determines life span, using historical data to show that such indicators of environmental insults in early childhood and young adulthood as quarter of birth, residence, occupation, wealth, and the incidence of specific infectious diseases affected older age mortality. Consistent with improvements in early life factors, we find that the effect of quarter of birth on older age mortality has diminished over the twentieth century and that the declining impact of quarter of birth explains 16 to 17 percent of the difference in ten year mortality rates of Americans age 60-79 in 1900 and in 1960-1980. We estimate that at least one-fifth of the increase between 1900 and 1999 in the probability of a 65 year old surviving to age 85 may be attributable to early life conditions. We also present suggestive evidence on the mortality trajectory of the oldest old in the first half of the twentieth century that implies that the shape of the mortality trajectory, though not its level, has remained constant.

Dora L. Costa
MIT
Department of Economics
E52-274C
50 Memorial Drive
Cambridge, MA 02142
and NBER
costa@mit.edu

Joanna Lahey
MIT
Department of Economics
E52
50 Memorial Drive
Cambridge, MA 02142
jlahey@mit.edu

The twentieth century has witnessed remarkable declines in older age mortality in Western Europe, Japan, and the United States (Robine and Vaupel 2002; Kannisto 1996; Vaupel 1997; Wilmoth et al. 2000). The first documented 111 year old died in 1932 and the first verified 122 year old died in 1997 (Vaupel 1997). Not only has the maximum age of death increased, but the number of people living beyond 100 has increased as well, calling into question whether the upper limit to the length of human life postulated by Fries (1980) is anywhere in sight and whether such an upper limit even exists (Vaupel and Jeune 1995; Wilmoth and Lundström 1996). In the United States life expectancy in 1900 at age 65 for both sexes combined was less than 12 years and only 13 percent of all 65 year olds could expect to reach age 85 and join the ranks of the oldest old. By century's end life expectancy at age 65 had risen to almost 17 years and 42 percent of all 65 year olds could expect to live until age 85. This increase in life expectancy was extremely slow at first, rising by only half a year during the first three decades of the twentieth century. Since 1930 the increase in life expectancy at age 65 has accelerated to 2 years between 1930 and 1960 and then to 3 years between 1960 and 1999.[1]

What accounts for the twentieth century increase in life expectancy and in the proportion of the older population who have become oldest-old? Scientists account for change by genetic evolution, postulating that mutations with bad effects at late ages have bad effects at young ages thus weeding out late-age failures (e.g. Charlesworth 2001). In contrast, demographers and economists have pointed to the environment. Robine (2003; 2001) argues that contrary to most scientists' assumptions, the environment, not genetics, determines life span with genetics accounting for individual variation in life spans. Empirically we observe that as individuals age mortality rates increase at a roughly constant rate until around age 109 and then plateau or even decline (Thatcher et al. 1998; Vaupel et al. 1998). Robine (2003) hypothesizes that while

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[1]See the Berkeley Mortality Database and the Human Mortality DataBase, http://www.demog.berkeley.edu. All life expectancies are period life expectancies.

the shape of the mortality trajectory may be a biological constant, its level, the rate of increase in mortality rates, and perhaps even the age at which the plateau is reached, is determined by the environment. Fogel and Costa (1997) and Fogel (1994) have argued that the unprecedented degree of control that humans have gained over their environment in the last 300 years has led to changes in human physiology that have lengthened life and improved health. Just over the last 100 years major environmental changes in the developed world include the virtual elimination of the infectious diseases that ravaged childhood, the dramatic expansion of the supply of calories and of micro-nutrients, the control of chronic disease conditions through new drugs and surgeries, increased knowledge (though not always application) of good health and dietary habits, and reduced exposure to physical hazards, including environmental pollutants and poisons, both in the home and in the workplace.

If the environment determines life span then such environmental insults as infectious disease, poor nutrition, low socio-economic status, or occupational hazards have a permanent scarring effect, leading to death at later ages by leaving survivors with permanent damage to their hearts, lungs, or other organs as in the case of rheumatic fever or tuberculosis. However, it is not clear a priori that environmental insults necessarily reduce longevity. Individuals who are more frequently exposed to disease may acquire full or partial immunities to such diseases as typhoid or influenza. If only the healthiest survive insults, then selection may lead to a negative relationship between insults and later mortality. Although a large body of research has found evidence that environmental insults, including those in utero and early childhood, affect older age health and longevity (Costa 2003b; Doblhammer and Vaupel 2001; Preston, Hill, and Drevenstedt 1998; Manton, Stallard, and Corder 1997; Costa 2000b; Elo and Preston 1992; Barker 1992, 1994; cf. Christensen et al. 1995; Kannisto, Christensen, and Vaupel 1997; Kannisto 1994), much of this research can be criticized on the grounds that an observed positive relationship between insults and later death may result from correlation between insults and an omitted variable such

as life-long poverty (Paneth and Susser 1995).

This paper reviews the literature on causes of lengthening life, emphasizing what scholars have learned from studying past populations, and uses historical data to assess the impact of environmental insults in early childhood and at later ages on the mortality of men and women in their sixties and seventies, focusing primarily on the effects of insults related to nutrition and infectious disease. Although we do not present new evidence on the roles of improvements in medical care, changes in health or dietary habits, and reduced exposure to environmental pollutants and poisons in the twentieth century mortality decline, we briefly discuss these factors. We present new evidence on the effects of immigration on historical United States mortality patterns. Manton and Vaupel (1995) suggest that because immigrants are healthier, recent increases in life expectancy in the United States could arise from immigration. All of our analyses emphasize the predictors of survival to become oldest-old, a rare achievement for most of our ancestors. Although the high mortality rates of historic populations do not allow us to investigate the predictors of survivorship among the oldest-old, we present some suggestive evidence on the trajectory of mortality among the oldest-old Union Army veterans.

The paper first reviews the types of historical data that are available. The next two sections discuss the impact of insults in early childhood and in later life, respectively, on older age mortality. The indicators of environmental stress that we focus on are quarter of birth, residence, socioeconomic status, and the incidence of specific infectious diseases. The fourth section examines the mortality experience of immigrants relative to the native-born in both the nineteenth and the twentieth century. The fifth section evaluates the likely magnitude of environmental insults on older age mortality. The sixth section discusses anthropometric indicators of environmental stress, because these have been widely used in the literature. The seventh section discusses alternative explanations for the mortality decline at older ages. The eighth section presents our evidence on the mortality trajectory of the oldest-old. We then summarize our results and conclude

with some thoughts on future trends in life expectancy at older ages.

1   Historical Data

Historical data are useful for establishing long-term mortality trends and for investigating the determinants of mortality under very different environmental conditions. Past populations were exposed to a wide variety of infectious diseases which varied greatly depending upon size of city of residence. They faced greater nutritional stress both because of their greater poverty and because their intake of vitamins and nutrients was determined by the local agricultural cycle. In addition, they faced high occupational risks and had medical care that was ineffective at best, thus allowing us to examine the effects of untreated infectious disease on later mortality.

Much of the initial work with historical data used aggregate data (e.g. Preston and Van de Walle 1978; Condran and Cheney 1982) and these are still valuable in understanding mortality trends at older ages (e.g. Wilmoth and Horiuchi 1999). However, a disadvantage of aggregate data is that it is very hard to identify the effects of environmental stress on mortality and even harder to distinguish the effects of different types of stresses. Demographers and economic historians have therefore turned to individual-level data.

Individual-level data has consisted of either cross-sectional data with retrospective mortality information or longitudinal data. Cross-sectional data with retrospective mortality information includes the 1900 and 1910 micro-census data with its questions on number of children ever born and number of children surviving used by Preston and Haines (1991) to study the predictors of child mortality. It also includes the 1850 and 1860 census mortality schedules linked to the 1850 and 1860 census population schedules used by Ferrie (2003).

Group level longitudinal data can be created from such individual-level data as the census micro samples. For example, Lleras-Muney (2002) used the 1960-1980 micro census

samples to examine the causal effect of education on 10 year mortality, creating cells based upon sex/cohort and state of birth and instrumenting for education with the compulsory school laws that were in effect between 1915-1939. A potential drawback to creating group level artificial cohorts is that for the variables of interest the cell sizes may be too small.

The individual-level longitudinal datasets that have been created have varied widely in the number of years of life that they cover. Steckel (1988) linked households across the 1850 and 1860 censuses to study the mortality of women and children. Preston, Hill, and Drevenstedt (1998) linked African-Americans who survived until age 85 to either their 1900 or 1910 household and used these censuses to draw a control sample. Perhaps the most ambitious linkage project of all has been the creation of the dataset, *The Aging of Union Army Veterans*, available for download from http://www.cpe.uchicago.edu. These data, collected by a team of researchers led by Robert Fogel, cover the life histories of men born between 1820 and 1850, and who reached age 65 between 1885 and 1915. The sample is based upon 35,570 white males who were mustered into the Union Army during 1861-1865. Socioeconomic and biomedical histories of the recruits from childhood to death were created by linking together information from different sources, including military records, pension records, reports from examining surgeons, and the 1850, 1860, 1900, and 1910 censuses.

The new results in this paper are based upon both group-level longitudinal data created from the public use micro census samples and upon individual-level longitudinal data from the Union Army data.[2]

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[2]The results presented here for the Union Army data differ from those in Costa (2003b) because a larger sample size is used and because the effects of additional variables such as quarter of birth and family income are examined.

2   Insults in Early Childhood

There is a large body of literature stressing the connection between intrauterine and infant growth and premature morbidity and mortality at ages above 50. The diseases that are most closely linked to anthropological markers of maternal and intrauterine deprivation are hypertension, coronary heart disease, cerebral hemorrhage, and type II diabetes (Barker 1992, 1996; Eriksson et al. 2000). Much of the evidence comes from studies carried out by Barker and his collegues in Britain, but there is also evidence from Sweden, Finland, and India (Hyppönen et al. 2001; Leon et al. 1998; Forsén et al. 1999; Stein et al. 1996). These studies have been criticized for not adequately adjusting for such confounding factors as middle-age variations in nutrition and life style and for reflecting the impact of random error (Huxley, Neil, and Collins 2002; Paneth and Susser 1995; Scrimshaw 1997). Other studies have found no evidence that intrauterine growth retardation affects later mortality. Christensen et al. (1995) compare twins and singletons and find no mortality differentials except among women age 60-84, among whom twin mortality was higher. Almond, Chay, and Lee (2002) find no evidence that the lighter birth weight twin suffers increased mortality after one year. But, twinning may well be a different process from intrauterine growth retardation. Kannisto, Christensen, and Vaupel (1997) find no evidence of increased mortality in later life for cohorts born during famine compared to cohorts born 5 years before and 5 years after the famine, but variation across cohorts may not be enough to identify the effects of famine if nutritional stress in childhood and of the mother also has an effect on mortality. Steckel (1989) documents that although American slave children experienced such severe deprivation that by four and half years of age they were only at the 0.2 height centile, below the level of the poorest populations of developing countries, they experienced remarkable catch-up growth when they reached field hand age and large quantities of meat were introduced into their diets. By adulthood slaves were only slightly shorter than whites and their mortality

rates at ages 20-24 were comparable to those of whites, suggesting to us that while slaves'

early childhood did leave a scarring effect, rapid improvements in conditions during adolescence

minimized the scar's impact.

Some of the strongest evidence for the impact of early life environmental factors on

older age mortality comes from studies of the effect of month of birth (Doblhammer and Vaupel

2001; Doblhammer 1999; Gavrilov and Gavrilov 1999). Doblhammer and Vaupel (2001) find that

in Austria and Denmark fifty year olds born in the fourth quarter live longer than those born in the

second quarter whereas in Austalia the pattern among the native-born is shifted by half a year but

among the British-born it is similar to that observed in Austria and Denmark. Kanjanapipatkul

(2002) finds that Union Army veterans born in the second quarter experienced shorter life-spans

than Union Army veterans born in the fourth quarter. (In contrast, Huntington (1938) found that

in a sample of genealogies individuals born in February or March lived longer than those born in

July or August.) Doblhammer and Vaupel (2001) reject the hypotheses that season of birth is an

indicator of social status or of age and hypothesize that month of birth has an effect on older age

mortality through birth weight, pointing out that in Austria during the crisis years of 1916-1922

children born in the fall had higher birth weights than those born in other seasons (Ward 1993:

22).

Additional evidence for the impact of early life environmental factors on older age

mortality comes from historical studies of the impact of place of birth. Preston, Hill, and

Drevenstedt (1998) find that among African-Americans farm background was a strong predictor

of survival to age 85. Costa (2003) finds that enlisting in a large city (a proxy for growing up in

a large city) had a large negative effect on survival among Union Army veterans who lived until

age 50 even controlling for size of current city of residence. There is, however, also evidence that

under the extreme disease conditions of Union Army camp life growing up in a large city 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 (Lee 2003).

In the United States both quarter and region of birth predict ten year mortality rates of 60-79 year-olds in 1960-1980 (see Table 1). Controlling for age group, sex, and cohort, the ten year mortality rate of men and women born in the Midwest and West was lower by 0.01 and 0.05, respectively, than that of their counterparts born in the Northeast. The ten year mortality rate of men and women born in the second quarter was higher by 0.03 than that of their counterparts born in the fourth quarter, an 8 percent increase in the mean ten year mortality rate of 0.36, and the effect was strongly statistically significant.[3]

Figures 1 and 2 illustrate the importance of season and of place of birth on the survival probabilities of Union Army veterans who were age 60-74 in 1900. Adjusting for age, veterans born in the second quarter had lower survival probabilities than those born during the second quarter. Those born during the first and third quarters also had higher survival probabilities than those born during the second quarter (not shown). Veterans who enlisted in a city whose population was above 50,000 in 1860 (the largest 16 cities in the country), faced an elevated mortality risk relative to those who enlisted in smaller cities. Even though food was plentiful and may have been more varied than in rural areas, rising population density and poor sanitation made the largest cities in the country particularly lethal. Life expectancy at birth in the largest Northern cities was only 24. Because city of enlistment was a good indicator of city of birth (Costa 2003b), city of enlistment provides a good indication of environmental stress both at birth and in early childhood.

Data on birth weight from Johns Hopkins Hospital provide suggestive evidence that those born during the spring (March-May) fared worse than those born at other times of the year (see Figure 3). Controlling for prematurity, the sex and race of the child, cohort, and mother's

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[3]We did not find differences in the effect of quarter of birth by region.

demographic characteristics, babies born in the spring weighed 81 grams less than those born in the summer. For full-term births the difference was 73 grams controlling for gestational age. When season of birth was defined by quarter, then babies born during the second quarter (April-June) were significantly more likely to be born prematurely than babies born in other quarters, controlling for sex and race of the child, cohort, and mother's demographic characteristics (Costa 2003a). Low birth weights and high prematurity rates in the spring may be related to maternal vitamin levels. A study of a rural North Carolina mill town begun in 1939 found that in the spring vitamin levels were at their lowest point (Beardsley 1989: 204).

Although season of birth and therefore arguably birth weight has a statistically significant effect on old age mortality, it is not clear that in the United States declines in older age mortality can be directly linked to improvements in characteristics at birth. In the United States, birth weights have remained roughly constant over the twentieth century (Costa 2003a, 1998) and even the population of Philadelphia Almshouse in the mid-nineteenth century had high birth weights by modern standards (Goldin and Margo 1989).[4] Birth weights in the past may have compared favorably with those today because many low birth weight babies who were stillborn in the past would have survived with modern obstretical knowledge and a modern disease environment. Including stillbirths in average birth weight implies that average birth weight increased slightly over the twentieth century (Costa 2003a). The constancy in live birth weights over the twentieth century suggests either that better measures of the intrauterine environment are needed or that explanations for improvements in health at older ages may need to focus on changes in the disease environment, changes that affected all age groups but that were particularly important in the first five years of life.

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[4]European birth weights were low compared to those of the white United States population (Ward 1993), suggesting that improvements in the intrauterine environment may explain a greater proportion of the European increase in life expectancy at older ages.

3   Insults in Later Life

Infectious diseases that were common in the past included typhoid, rheumatic fever, respiratory infections such as bronchitis and pneumonia, malaria, measles, tuberculosis, and gastrointestinal ailments, including cholera and diarrhea. These were not just diseases of childhood and their recurrence might have compounded any initial scarring effects. The medical and epidemiological literature provides many examples of the possible links between these infectious diseases and chronic conditions (see Costa 2000 for more details). Rheumatic fever can damage heart valves. Measles and typhoid fever affect cardiac functioning. Infections that have been implicated in atherosclerosis include helicobacter pylori, a bacterium that causes gastritis and stomach ulcers, and chlamydia pneumonia, a bacterium that causes acute upper and lower respiratory tract infections. Individuals at risk of later susceptibility to pneumonia, chronic obstructive lung disease, and tuberculosis include those who have had prior upper respiratory tract infections and tuberculosis.

Costa (2000,2003) examined the morbidity and mortality experience at older ages of Union Army veterans who had experienced the common infectious diseases while in the army camps. She found that acute respiratory infections while in the army increased the probability of chronic respiratory problems and arrhythmias at older ages; measles, the probability of chronic respiratory problems and of valvular heart disease; typhoid fever, of valvular heart disease and irregular pulse; tuberculosis, of chronic respiratory problems; rheumatic fever or post-rheumatic arthopathies of valvular heart disease, arrhythmias, congestive heart failure, joint problems, and back problems; diarrhea, of arteriosclerosis; and malaria, of joint problems. But, none of these army camp infectious diseases were statistically significant predictors of all-cause mortality. Some conditions did predict mortality by cause. For example, respiratory infection predicted death from respiratory disease, diarrhea predicted death from gastrointestinal and stomach ailments,

and rheumatic fever predicted death from myocarditis. Men who had been prisoners of war and therefore had probably suffered gastrointestinal ailments during their imprisonment, were more likely to die from stomach ailments. Because a large fraction of deaths among Union Army veterans were from infectious, respiratory, and diarrheal disease, 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 have been more important.

Occupational hazards may also have scarring effects on older age mortality and morbidity. Lung diseases and respiratory symptoms resulting from occupational exposure to dust, fumes, or gases include asthma, chronic bronchitis, chronic air flow limitation, and turberculosis. Workers in mining, steel foundries, tool grinding, glassmaking, metal casting, and stone polishing are particularly prone to occupational lung disease. Farmers are also affected because they inhale organic dust from moldy plant materials and from animal waste, hair, and feathers. Occupational stress is also an important determinant of musculoskeletal capacity. Among Union Army veterans, those in a manual occupation (including farming), disproportionately suffered from arthritis and from back problems at older ages. Veterans who had worked in occupations where they were exposed to dust and fumes were more likely to suffer respiratory ailments at older ages (Costa 2000). Although laborers were significantly more likely to die of heart disease than farmers, occupation had no effect on the all cause mortality of Union Army veterans (Costa 2003b). Note that in data on more recent populations, occupational differences in all-cause mortality are more pronounced. The most common causes of death may now be those where socioeconomic status has the largest impact. Alternatively, the relationship between socioeconomic status and mortality may have changed. Both United States and British studies from the 1930s and 1940s found a positive correlation between high socioeconomic status and risk of coronary heart disease in men, but studies from 1940 to 1960 found a negative correlation (Manton et al. 1997).

Evidence for the impact of income on mortality in historical United States populations

is mixed. Wealth conveyed no systematic advantage for survival of women and children in households matched in the 1850 and 1860 censuses (Steckel 1998). Preston and Haines (1991) used the questions on number of children ever born and 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 father's occupation. Preston and Ewbank (1990) find that infant mortality differentials by social class increased from 1900 to 1930, perhaps because the germ theory of disease first spread to the more educated classes. However, even before knowledge of the germ theory of disease, income enabled individuals to buy lower room congestion, housing in an area of a city with better sanitation, better food, and less work away from home for the pregnant mother. Within U.S. cities there was a steep gradient between infant mortality and family income (e.g. see Rochester 1923). Chapin (1924) reported that in Providence, Rhode Island in 1865 the annual crude death rate for taxpayers was 11 per thousand, while the corresponding rate for non-taxpayers was 25 per thousand. 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 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 because they did not know how to protect themselves from cholera.

Figures 4 and 5 illustrate the impact of personal property wealth in 1860 and of occupation at enlistment on the age adjusted survival probabilities of Union Army veterans age 60-74 in 1900. Those of higher socioeconomic status were favored in survival. These measures of socioeconomic status will be strongly correlated with the socioeconomic status of the household veterans grew up in as children (if they had not yet formed their own households then wealth is parental family wealth) and will also be correlated with socioeconomic status later in life.

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Although we cannot adjust for wealth later in the life-cycle, when we adjust for occupational class at older ages we find that occupation at enlistment still has an effect on older age mortality.

The graphs of survival probabilities over time of Union Army veterans show that the effects of such environmental factors as quarter of birth, city of enlistment, wealth, and occupation diminish with age. They also suggest that at very late ages, survival probabilities no longer fall as steeply as at younger ages.

4   Immigration

Recent increases in life expectancy in the United States may arise from increased migration. Manton and Vaupel (1995) suggest that immigrants are healthier than the people who did not choose to immigrate, and probably have healthier children as a result. Nineteenth century migration, however, may have been harmful to health. European soldiers were long aware that strange "climates" could have fatal effects (Curtin 1989). Large cities, and particularly their immigrant wards, were the sources of many epidemics. Sánchez (2003) used the longitudinal residential information in the Union Army records to trace the effects of internal migration on life expectancy. He found that migrants were more likely to die of infectious disease than non-migrants and that although mortality rates were higher among migrants to urban areas than among migrants to rural areas, even migrants to rural areas faced an elevated mortality risk compared to non-migrants.

Table 2 shows that the impact of foreign-birth on the ten year mortality rate of white 60-79 year olds differs by cohort. There was no statistically significant difference in mortality rates at older ages between immigrants and the native-born among cohorts born 1830-1859. But, among cohorts born 1870-1899, the foreign-born faced a higher mortality risk at older ages and among cohorts born 1900-1929 the foreign born faced a lower mortality risk at older ages than the

native-born. When we examined birth place of origin we found that for the 1830-59 and 1870-99 cohorts the Irish faced higher mortality risk than the Germans or the English and that this risk was much higher for the earlier Irish cohort (which had greater life-long poverty and which was born during the Potato Famine) than the later one. For the 1900-29 cohort those born in Mexico and, to a lesser extent, those born in Mediterranean countries faced a lower mortality risk perhaps because immigration from these countries was temporary and those in need of family support returned home.

5 Accounting for Mortality Improvements

Regression analysis allows us to assess the relative importance of different types of environmental stress over the life cycle among Union Army veterans age 60-74 in 1900. We model the probability of survival to age 85 by means of a probit equation,

$$Prob(I = 1) = Prob(\epsilon < X'\beta) = \Phi(X'\beta) \tag{1}$$

where $I = 1$ if the veteran survived until age 85, $\Phi()$ is a standard normal cumulative distribution function, and X is a vector of control variables. The effect of a unit change in one of the independent variables on the probability of having a given chronic condition is given by the partial derivative of the probit function P with respect to that independent variable. We model the number of years lived after 1900 by means of a hazard model, in which our estimated hazard $\lambda(t)$ is

$$\lambda(t) = \exp(X'\beta)\lambda_0(t) \, , \tag{2}$$

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where X is the vector of control variables and $\lambda_O(t)$ is the baseline hazard which we assume to be Gompertz, $\lambda_O(t) = \exp(\gamma t)$.[5] The hazard ratios that we report indicate whether a one unit change in an independent variable gives an increase/decrease in the odds of an event. For the sample of men for whom cause of death is known, we estimate separate independent competing risk models for five specific causes of death, treating other causes of death as censored. The five specific causes of death that we examine are death from heart disease (excluding stroke), death from stroke, death from infectious diseases (including tuberculosis), death from both chronic and acute respiratory ailments such as pneumonia, influenza, and acute bronchitis, and death from stomach ailments such as gastritis/duodentitis and ulcers. Our estimated hazard, $\lambda_i(t)$, for one of our five models (i), is

$$\lambda_i(t_i) = \exp(X'\beta)\lambda_{i0}(t), \tag{3}$$

where X is the vector of control variables and $\lambda_{iO}(t)$ is the baseline hazard which we assume to be Gompertz.

The primary control variables used in the analysis are season of birth (four quarters); size of city of enlistment (50,000 and over, 25,000-50,000, 2500-25,000, and less than 2500); a dummy equal to one if the household's total personal property wealth in 1860 was greater than \$250; occupation at enlistment and occupation circa 1900, using last known occupation for the retired (farmer, professional, manager or proprietor, clerical service, craft, operative, service, and laborer); prisoner of war status; and infectious disease experienced while in the service (diar-

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[5]A Gompertz model is favored over an exponential, Weibull, or log-logistic model by the Aikaike information criterion, but using these other models yields very similar conclusions. Using the Gompertz model, but not the other models, we find evidence for individual level heterogeneity suggesting that after a while individual hazards begin to fall. However, the estimated heterogeneity parameter is quantitatively small and the parameter estimates remain very similar, perhaps because relatively few individuals survive to ages where individual hazards begin to fall. (As we discuss later, the hazard does not fall until after age 99, when only 1 percent of the sample was alive.) For simplicity we therefore present the Gompertz models.

rhea, respiratory, measles, tuberculosis, rheumatism/rheumatic fever, malaria, typhoid, stomach ailments, and syphilis). Additional control variables include age in 1900, indicators of missing information, a dummy indicating whether the veteran was ever wounded in the war, size of city of residence in 1900, and marital status in 1900.

Being born in the second relative to the fourth quarter decreased the probability of survival to age 85 by 0.04, a decline of 23 percent relative to the predicted sample survival probability of 0.17.[6] Enlisting in a large rather than a small city had a larger effect on the probability of survival to age 85, lowering the probability of survival to age 85 by 0.06. Relative to men who were farmers at enlistment, men who were laborers had a survival probability that was lower by 0.05. Occupation circa 1900 and wartime service had no effect on the probability of surviving until age 85. Although enlisting in a large city had the largest effects on mortality, relatively few men enlisted in large city. If no one had enlisted in a large city the predicted probability of survival would have been 0.18. In contrast if no one had been born in the second quarter the predicted probability of survival would have been 0.19. If everyone had been a farmer at enlistment the predicted probability of survival would have been 0.19 and if everyone had been a professional the predicted probability would have been 0.21.

The all-cause mortality hazard model also emphasizes the importance of size of city of enlistment. Enlisting in a large city increased the odds of dying by 1.24. Being born in both the second and third quarter relative to the fourth quarter increased the odds of dying by 1.10. Non-farmers faced an elevated mortality risk. Although being an operative at enlistment rather than a farmer increased the odds of dying by 2.02, a large standard error means that the effect

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[6]The probability of survival in this sample this sample is higher than that from a 1900 life table for the death registration states. In the 1900 life table men age 65 (the mean age in the Union Army sample) had only a 13 percent chance of surviving to age 85 and a life expectancy of 11.4 years. In contrast life expectancy among Union Army veterans was 12.6. Mortality rates life table for the death registration states probably overstate the national average because the death registration states were disproportionately the urban, Northeastern states in which mortality was higher.

was not statistically significant. Those with high personal property wealth faced a lower odds of death. Using the hazard model specification, the largest effects on mortality come from high personal property wealth. If everyone had had high personal property wealth then the predicted median number of years lived after 1900 would have been 12.8, rather than 12.3. If everyone had been a farmer, the predicted median number of years would have been 12.6 and if everyone had been a professional the predicted median number of years would have been 12.4. If no one had been born in the second quarter the predicted median number of years lived would have been 12.5 and if no one had enlisted in a large city the predicted median number of years lived would have been 12.4.

Examining mortality by cause of death allows us to evaluate the pathways by which our measures of environmental stress affected mortality. Tables 4 and 5 show the impact of variables that had an effect on mortality from one of the five specific causes. Men born in the third relative to the fourth quarter were more likely to die of heart disease. Men born in either of the first three quarters were more likely to die of stroke than men born in the fourth quarter, with the highest probability of death for those born in the second quarter. Quarter of birth did not have a statistically significant impact on death from infectious, respiratory, or stomach ailments. A manual non-farming occupation at enlistment relative to being a farmer or a professional increased the probability of death from heart disease or stroke, but not from infectious, respiratory, or stomach ailments. Rheumatic fever while in the service increased the probability of death of death from heart disease, tuberculosis while in the service increased the probability of death from infectious disease, respiratory ailments while in the service increased the probability of death from respiratory disease, and diarrhea and measles while in the service and having been a prisoner of war increased the probability of death from stomach ailments. Size of city of enlistment had no effect on mortality by cause. None of the explanatory variables had any effect on deaths from violence.

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A comparison of the effect of quarter of birth on the ten year mortality rates of 60-79 year old Union Army veterans and Americans in 1960-1980 shows that the effect of quarter of birth has lessened (see Table 6). Being born in either the second or third rather than the fourth quarter increased Union Army veterans' probability of dying by 0.04, a 9 percent increase in the mean ten year mortality rate of 0.45. In contrast, in 1960-1980 being born in the second rather than the fourth quarter increased the mean ten year mortality rate of 0.36 by 0.03, an 8 percent increase, and being born in the third rather than the fourth quarter increased the ten year mortality rate by 0.01, a 4 percent increase. The declining impact of being born in the summer, a time when the disease burden from water-borne diseases was especially high, suggests improvements in either the intrauterine or the early infancy environment.[7]

The declining impact of season of birth accounts for roughly 16 to 17 percent of the 0.087 difference in the ten year mortality rates of Union Army veterans and of Americans in 1960-1980. Differences in the seasonality of births in the two samples are negligible.[8] Assuming that ten year mortality rates in the Union Army sample and the recent sample can be written as linear functions of quarter of birth using the coefficients in Table 6, the difference in ten year mortality rates due to changes in the coefficients on birth weights can be written as $q1_{UA}(\beta_{q1,UA} - \beta_{q1,R}) + q2_{UA}(\beta_{q2,UA} - \beta_{q2,R}) + q3_{UA}(\beta_{q3,UA} - \beta_{q3,R})$ using the recent function and as $q1_R(\beta_{q1,UA} - \beta_{q1,R}) + q2_R(\beta_{q2,UA} - \beta_{q2,R}) + q3_R(\beta_{q3,UA} - \beta_{q3,R})$ using the Union Army function, where $q1$, $q2$, and $q3$ indicate the first, second, and third quarters and where the subscripts UA and R indicate the Union Army sample and the recent sample, respectively. These respective sums yield 0.014 and 0.015, which represents 16 and 17 percent of the difference in ten year mortality rates. The

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[7]Kanjanapipatkul (2002) finds that in recent United States data the seasonal pattern differs from that observed at Johns Hopkins and that since the 1960s seasonal fluctuations in birth weight have diminished.

[8]The fractions born in the first, second, third, and fourth quarters were 0.261, 0.252, 0.241, and 0.245, respectively, in the Union Army sample. The respective fractions for Americans in 1960-1980 were 0.266, 0.232, 0.257, and 0.245.

difference in ten year mortality rates due to changes in season of birth is virtually zero.

Rising wealth, an improving disease environment, declining occupational risks, and a more favorable intrauterine environment may explain at least one-fifth of the increase between 1900 and 1999 in the proportion of the population living to age 85 and of the increase in life expectancy at age 65. Between 1900 and 1999, the probability that a 65 year old man (the mean age in the Union Army sample) would survive to age 85 rose from 13 to 35 percent and life expectancy at age 65 rose from 11.4 to 13.5 years.[9] Had all men in the Union Army sample had a very favorable experience, here proxied as the same mortality experience as men who were farmers at enlistment, who grew up in a small city, who were born in the fourth quarter, and who had high personal property wealth, their predicted probability of living until age 85 would have been 0.22 and their predicted median number of years lived would have been 13.5 years. By modern standards this is not a very favorable experience, but over three-quarters of Union Army veterans fared much worse in terms of mortality. The increase of 0.05 in the predicted probability of living to age 85 and the increase of 1.2 years in predicted median number of years lived represents roughly one-fifth of the observed increase in the period life tables. Much of the increase in survival probabilities, however, cannot be explained by our regression models, perhaps because many risks are not observed, but also because, as noted for quarter of birth, the relationship between risk factors and mortality has changed.

6   Anthropometric Measures

Anthropometric measures provide useful proxies for environmental stress. Heights have been widely used as proxies for the environmental stress faced by past populations because they

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[9]See the Berkeley Mortality Database and the Human Mortality DataBase, http://www.demog.berkeley.edu. All life expectancies are period life expectancies.

were commonly recorded by the military for identification purposes. Additional anthropometric measures that have been used include Body Mass Index (BMI) and measures of central body fat. Poor net nutritional status during the growing years (including the fetal stage) leads to a shorter population and poor current net nutritional status to a lighter population. Low birth weight for gestational age babies not only grow up to be shorter, but they may also grow up to have greater abdominal fat deposits, controlling for body mass (Paz et al. 1993; Lagerström et al. 1994).

Height, weight, and fat patterning are predictors of subsequent morbidity and mortality. Costa (1993) and Costa and Steckel (1997) found that the relationship between height and BMI and subsequent mortality was similar among a sample of Union Army veterans and among modern Norwegians males. All-cause mortality first declines with height to reach a minimum at heights close to 185cm and then starts to rise.[10] All-cause mortality risk first declines at low weights as BMI increases, stays relatively flat over BMI levels from the low to high twenties, then starts to rise again, but less steeply than at very low BMIs. In recent data low BMIs are associated with respiratory disease and high BMIs with heart disease. Height appears to be inversely related to heart and respiratory disease and positively related to the hormonal cancers (Barker 1992). It is also related to stroke (McCarron et al. 2001). Abdominal fat distribution is associated with antecedents of cardiovascular disease such as hypertension, non-insulin dependent diabetes, high plasma concentrations of atherogenic lipids, and low concentrations of high density lipoprotein cholesterol (Ohlson et al. 1985; Blair et al. 1984; Folsom et al. 1989). A 23-year follow-up study of WWII soldiers measured prior to discharge in 1946-47 shows that a standard deviation increase in waist-hip ratio above the mean increased mortality risk from ischemic and cerebrovascular heart disease by up to 1.24 times and that this relationship was linear (Terry et al. 1992). Yao et al.

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[10]However, in later work Costa (2003) did not find a relationship between height and older age mortality among Union Army veterans. Costa (1993) showed that the relationship between height and mortality among Norwegian males was very sensitive to sample size.

(1991) find that measures of central body are linearly related to cardiovascular disease, but, like BMI, they have a U-shaped relation with all cause mortality. Costa (forthcoming) found that in a sample of Civil War soldiers measured by the United States Sanitary Commission, waist-hip ratio at young adult ages was an excellent predictor of older age mortality from ischemic heart disease or stroke and that the relationship was U-shaped.

In the sample of Union Army veterans used in this paper, height at enlistment was not a predictor of older age mortality. BMI in 1900 was a U-shaped predictor of both all-cause and respiratory disease mortality, but did not significantly affect death from the other specific causes, suggesting that BMI at older ages may be reflecting current underlying disease conditions, particularly respiratory ones. Controlling for all other early and later life socioeconomic and demographic characteristics, men with a BMI of 24 circa 1900 were the most likely to survive to age 85.

Past populations were shorter, lighter, and, controlling for BMI, had more abdominal fat (Fogel and Costa 1997; Costa and Steckel 1997; Costa forthcoming). How did changes in frame size affect mortality trends? Costa (1993) calculated that increases in BMI from 1900 to 1986 explain roughly 20 percent of the total decline in mortality above age 50. Costa (forthcoming) estimated that changes in frame size (height, BMI, and waist-hip ratio) accounted for almost half of the mortality declines among 56-72 year old white men between 1914 and 1988. The impact of changes in frame size on mortality may have been even larger in other countries. Changes in French height and weight jointly explain about 90 percent of the decline in French mortality rates between 1785 and 1870 and about half of the decline in the twentieth century, with much of the mortality improvement prior to 1870 due to increases in height and much of it after 1870 due to increases in weight (Fogel and Costa 1997). These calculations reinforce the importance of environmental stress (though improvements in BMI could arise from better medical control of chronic conditions), but suggest that the childhood factors proxied by height have become less

important over time.

7   Other Factors

We estimated that at least one-fifth of the increase between 1900 and 1999 in the probability of a 65 year old surviving to age 85 to become oldest-old may be attributable to improvements in early life conditions. What might account for the remaining four-fifths? Potential explanations include improvements in medical care and knowledge, a move away from salt as a food preservative with the rise of refrigeration, and declines in environmental pollution and poison, among other factors, and these are the explanations that we review below in greater detail. (Other explanations such as smoking may explain the recent decline in death rates, but probably initially increased death rates during the twentieth century.)

Inadequate medical care, such as lack of treatment for heart attacks, can have immediate effects on mortality. Suggestive evidence for the impact of medical care on mortality comes from cross-country mortality comparisons. Velkova, Volleswinkel-Van den Bosch, and Mackenbach (1997) report that temporary life expectancy from birth to age 75 (the average number of years a cohort of newly born infants would live between birth and age 75) was higher by 1.25 to 6.29 years for men and 1.09 to 3.44 years for women in Western compared to Central and Eastern Europe. Excluding neonatal deaths, mortality from conditions amenable to medical intervention accounted for 11 to 50 percent of the difference in temporary life expectancy among men and 24 to 59 percent of the difference among women. Gjonça, Brockman, and Maier (2000) report that among those older than 79 mortality in Western Germany was decreasing since the 1970s whereas in East Germany mortality did not decline until the late 1980s and the decline accelerated sharply after German re-unification when the Western health care and pension system was installed in East Germany and when purchases of fresh fruits and vegetables increased. Bailey and Garber

(1997) point out that the United States devotes more resources to curing lung cancer, breast cancer, and diabetes than the United Kingdom and has the more favorable outcomes for breast and lung cancer whereas the United Kingdom has the most favorable outcomes for diabetes, a condition controlled by patient behavior not by capital intensive equipment.

Evidence for the effects of medical care on mortality also come from clinical studies of specific conditions. Cutler, McClellan, and Newhouse (1999) document the substantial change in heart attack treatment from the mid-1970s to the mid-1990s and, based upon their review of clinical studies, conclude that changes in medical treatment used in the management of acute myocardial infarction account for approximately 55 percent of the reduction in mortality that has occurred in acute myocardial infarction cases between 1975 and 1995, a time period when heart attack mortality fell by almost one-third. Their analysis of Medicare claims data from 1984 to 1991 confirms that mortality rates following a heart attack have fallen sharply (by 2 percentage points during the initial hospital stay and by 5 percentage points after one year) and have fallen more for heart attack patients than for the general population of the same age, suggesting that improved medical treatment is the most likely explanation.

The shift from salting and smoking to preserve foods to refrigeration has been pointed to as an explanation for the decline in stroke mortality.[11] Thus in Belgium where only 21 percent of households had a refrigerator in 1960 compared to 96 percent in the United States (Lebergott 1993: 111), researchers documented a 50 percent decline in mean 24-hour sodium excretion in subjects studied between 1966 and 1979 (e.g. Joossens, Kesteloot, and Amery 1979). But, a comparison of age-adjusted international stroke mortality trends (see de Looper and Bhatia 1998) reveals no clear pattern by refrigerator diffusion. In the United States 44 percent of homes had a refrigerator in 1940, a level achieved in France only 20 years later (Lebergott 1993: 111, 113), but

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[11]It has also been pointed to as an explanation for declines in stomach cancer because of the interaction between salt and helicobactor pylori (Manton, Stallard, and Corder 1997).

declines in stroke mortality for men and women were of a similar order of magnitude (around 70 percent) in the two countries between 1950-54 and 1992. Cummins (1983) found that although salt purchases in England and Wales declined between 1958 and 1978, coincident with declines in stroke mortality, the picture is confused by an increase in meals eaten outside the home and by the consumption of more processed foods. Other scholars have questioned whether there even is a relationship between the sodium contents of food and salt use and hypertension (e.g. Harlan et al. 1984). Lawlor et al. (2002) used data on British autopsies to infer that although both cerebral infarct mortality and cerebral hemorrhage mortality have fallen since 1932, the different patterns suggest that the aetiological factors differ. Estimated cerebral infarct mortality first rose and then declined sharply after the 1960s, following a pattern similar to that of coronary heart disease mortality. In contrast, estimated cerebral hemorrhage mortality declined steadily. They hypothesize that whereas smoking and cholesterol may be important risk factors for coronary heart disease and cerebral infarct, early life risk factors may be more important for cerebral hemorrhage.[12]

Rising urbanization and industrialization, signalled by the heavy black clouds that hung over many early twentieth century cities (Stradling 1999), may have initially contributed to an upsurge in the United States in respiratory disease and mortality, which then declined with anti-smoke and anti-pollution efforts after World War II. Studies based upon both geographic and temporal variation in air pollution find substantial effects of pollution on mortality and health. Ransom and Pope (1995) measured the external health costs of air pollution by using variation in the operation of a steel mill in a mountain valley in Central Utah and by using a nearby valley as a control. They reported that elevated pollution levels were associated with significant declines in childrens' lung function and that deaths in Utah county when the steel mill was operating were

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[12]Birth weight is strongly inversely associated with cerebral hemorrhage but not with cerebral infarct (Hyppönen et al. 2001; Eriksson et al. 2000).

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3.2 percent higher per day than when it was not. Chay and Greenstone (2003) used geographic variation in the effects of the 1981-1982 recession to find that pollution significantly affects infant deaths, particularly within one month and one day of birth, suggesting that fetal exposure may be harmful. They also find that the incidence of low birth weight babies (less than 2500 grams) falls with declines in pollution.

Another environmental hazard at the beginning of the twentieth century was lead poisoning, which by impairing the immune system, could have far reaching effects on older age mortality (e.g. see Fischbein et al. 1993). Troesken (2003) documents that in 1897 half of United States municipalities were using water from lead pipes and that lead levels in Massachusetts towns ranged from 3 to 411 times current Environmental Protection Agency guidelines. He finds that the use of lead pipes increased infant mortality rates by 25 to 50 percent, depending on the age of the lead pipes and on the corrosiveness of the water supply. Corrosiveness of the water supply was a problem particularly in New England. Troesken (2003) estimates that between 1910 and 1921, the decline in lead levels in New Hampshire water either from the gradual oxidization of lead pipes and their replacement or from declining industrial pollution cut infant deaths rates by up 16 percent. Given that roughly 28 percent of all births in the death registration areas were from New England, we calculate that if all of New England experienced New Hampshire's decline in lead levels then 12 percent of the decline in infant mortality between 1910 and 1921 is explained by reductions in lead levels.

8   The Mortality of the Oldest-Old

The evidence that we have presented suggests that the environment determines the level of mortality. But, by examining advanced ages can we say anything about the mortality trajectory? Is its shape the biological constant postulated by Robine (2003) in which mortality rates increase

and then plateau at very advanced ages? Figure 6 presents suggestive evidence from a sample of Union Army veterans who survived to age 85.[13] Mortality rates increase at a regular rate from age 85 to age 90, continue to increase at ages 90 to 95 and 95 to 98, but at lower rates, and then plateau. The oldest age of death is 105. The nonparametric estimate of the hazard (not shown) first rises and then declines after around age 98. Our evidence can only be suggestive – because 95 percent of the sample died before reaching age 95 and 99 percent of the sample died before reaching age 99 there are only 12 observations on the mortality plateau.

Very few environmental characteristics predict survival among the oldest-old. Men who were farmers circa 1900 lived longer than non-farmers and men who had not been wounded in the war lived longer than men who had. In contrast, when we did not restrict our sample to the oldest-old we found substantial effects on older age mortality of quarter of birth, residency in early childhood, occupation, wealth at young ages, and infectious disease experience when young. However, in our younger sample we did not find a statistically significant effect of war wounds and we found somewhat smaller farm versus non-farm occupational effects.

9   Conclusion

This paper has used longitudinal data on past populations who faced extreme variations in local environmental conditions to study the role of environmental stress throughout the life cycle on older-age mortality. We found that environmental insults in early childhood and during young adulthood reduced life expectancy. Among Union Army veterans age 60-74 in 1900, those born in either the second or third quarter relative to the fourth, those enlisting in a large city, and

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[13]The sample used differs from that used in our previous analysis. We use all Union Army veterans who survived to age 85 and who were on the pension rolls by age 85 regardless of their pension or survivorship status in 1900 or their age in 1900. We verified birth year using both the military service records and the pension records.

those of low socioeconomic status in their youth whether measured by occupation at enlistment or family wealth before the war did not live as long and were less likely to become the oldest-old. The infectious disease that soldiers had while in the army affected their later probability of death from a specific cause. Those who had had rheumatic fever as young men in the army were more likely to die of heart disease after age 60, those who had had tuberculosis were more likely to die of infectious disease, those who had had a respiratory disease were more likely to die of a respiratory disease, and those who had had diarrhea or had been prisoners of war were more likely to die of stomach ailments. Among Americans in their sixties and seventies in 1960-1980, mortality rates also varied by place of birth and mortality rates were higher for those born in the second and third quarters relative to the fourth quarter, but the magnitude of the effect of quarter of birth, particularly for the third quarter, was smaller in these more recent data than among Union Army veterans. We showed that in the first third of the twentieth century, the birth weights of children born in the spring were lower than those of children born in the summer, suggesting that quarter of birth may in part proxy for intrauterine growth retardation, though it could also reflect seasonal differences in the disease environment a child was born into. We calculated that 16 to 17 percent of the difference in older age ten year mortality rates between Union Army veterans and Americans in 1960-1980 was attributable to the declining impact of quarter of birth on mortality rates. We also estimated that at least one-fifth of the increase between 1900 and 1999 in the probability of a 65 year old surviving to age 85 may be attributable to early life conditions.

What explains improvements in early life conditions? The twentieth century witnessed the filtration and chlorination of water, the construction of integrated sewage systems and the adoption of sewage treatment, the cleaning of the milk supply and the eradication of bovine tuberculosis, widespread vaccination against childhood diseases, improved commercial food processing and the supplementation of foods with iodine and iron, the decline in exposure to environmental pollution and poisons both in the home and in the workplace, the decrease in

crowding that coincided with reductions in fertility and immigration and with rising incomes, and the availability of more varied and fresher food with the growing use of refrigeration in transportion, in stores, and in the home. The relative contributions of public health campaigns, technological change, and rising wealth to the improvement in early life conditions still remains an issue for future research.

Our findings have implications for future mortality trends. The cohort which will reach age 65 in 2020 was born in 1955 after the steep plunge in infant and child mortality and the historical record suggests that this cohort may be particularly long-lived compared to past cohorts. However, mortality rates may not fall as steeply for the cohorts born after 1955 as for earlier cohorts. The difference in early life conditions for cohorts born in 1955 instead of 1995 was not as stark as that for cohorts born in 1915 instead of 1955. For cohorts born after 1955 improvements in older age mortality rates will need to come from medical care or better health habits and unless the rate of innovation in medical increases the rate of reduction in future mortality improvements may not be as sharp as it has been in the recent past. Our suggestive evidence that the mortality trajectory of Union Army veterans reached a plateau at older ages does however provide grounds for optimism. As long as we can change the level of the plateau, investments in health at very old ages will not hit diminishing returns.

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Table 1: Effects of Region and Season of Birth on Ten Year Mortality Rates of 60-79 Year-Olds in the United States, 1960-1980

	Coef	Std Err
Dummy=1 if born in the		
Northeast		
Midwest	-0.014‡	0.006
South	-0.005	0.005
West	-0.053‡	0.005
Dummy=1 if born in		
First quarter	0.003	0.005
Second quarter	0.030‡	0.006
Third quarter	0.013†	0.006
Fourth quarter		
Observations	128	
R^2	0.984	

Source: Authors' calculations from the Integrated Public Use Census Samples. Each observation is an age group, sex, cohort, region, and quarter of birth cell. The sample was restricted to the native-born. The dependent variable is the 10 year mortality rate of each cell. The mean weighted 10 year mortality rate was 0.36. Additional controls include dummies for age 70 or older, female, and birth cohort (1900-1909 and 1910-1919, with 1890-1899 as the omitted category). The ordinary least squares regression is weighted by cell size. The symbols ‡ and † indicate that the coefficient is statistically significantly different from zero at the 1 and 5 percent level, respectively.

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Table 2: Effects of Foreign Birth on Ten Year Mortality Rates of White 60-79 Year Olds, by Cohort

	Census Year		
	1900-1920	1940-1990	
	Birth Year		
	1830-59	1870-99	1900-29
Coefficient on foreign-birth	0.011	0.032[‡]	-0.029[*]
Standard error	(0.009)	(0.007)	(0.015)
R^2	0.992	0.993	0.987
Observations	16	20	20
Mortality rate (weighted)	0.501	0.450	0.335

Source: Authors' calculations from the Integrated Public Use Census Samples. Each observation is an age group, sex, cohort, and native-born/foreign-born cell. The dependent variable is the 10 year mortality rate of each cell. Average weighted mortality rates were 0.50, 0.45, and 0.34 for the 1830-59, 1870-99, 1900-29 cohorts, respectively. Additional controls include dummies for age 70 or older, female, and birth cohort. The ordinary least squares regression is weighted by cell size. The symbols ‡ and ∗ indicate that the coefficient is statistically significantly different from zero at the 1 and 10 percent level, respectively.

Table 3: Probability Model of Surviving to Age 85 and Hazard Model of Years to Death, Union Army Veterans Age 60-74 in 1900

	Probit Model		Hazard Model	
	$\frac{\partial P}{\partial x}$	Std Err	Haz Rat	Std Err
Dummy=1 if born				
First quarter	-0.012	0.016	1.062	0.048
Second quarter	-0.036†	0.016	1.105†	0.051
Third quarter	-0.014	0.017	1.095†	0.051
Fourth quarter				
Dummy=1 if enlisted in city with population of				
50,000+	-0.055*	0.027	1.241‡	0.102
25,000-50,000	-0.028	0.032	1.063	0.096
2,500-25,000	-0.016	0.013	1.056	0.036
Less than 2,500				
Dummy=1 if personal property wealth in				
1860 household above $250	0.023	0.016	0.925*	0.039
Dummy=1 if occupation at enlistment				
Farmer				
Professional	0.022	0.038	1.024	0.097
Manager/Proprietor	-0.100*	0.044	1.183	0.211
Clerical/Service	-0.008*	0.053	1.073	0.157
Craft	-0.034	0.018	1.130†	0.057
Service			1.118*	0.076
Operative	-0.050†	0.022	2.022	0.836
Laborer	-0.014	0.021	1.074	0.062
Dummy=1 if occupation circa 1900				
Farmer				
Professional	-0.001	0.034	1.079	0.098
Manager/Proprietor	0.008	0.026	0.976	0.067
Clerical/Service	0.001	0.034	0.956	0.087
Craft	-0.003	0.020	1.009	0.054
Operative	-0.024	0.024	1.208‡	0.082
Service	-0.009	0.048	1.144	0.146
Laborer	-0.001	0.019	1.066	0.055
Dummy=1 if was POW	-0.020	0.019	1.046	0.055
Dummy=1 if in service had				
Diarrhea	-0.002	0.012	1.017	0.034
Respiratory	-0.020	0.016	1.065	0.047
Measles	0.016	0.027	0.903	0.063
Tuberculosis	0.022	0.043	0.900	0.096
Rheumatism/rheumatic fever	-0.022	0.016	1.033	0.045
Observations	4,641		4,647	
Pseudo R^2	0.030			
γ			0.090‡	0.002
$\chi^2(42)$ for				
Significance of all coefficients			481.770	0.000

The dependent variable for the probit equation is whether the individual lived until age 85, an age achieved by 17 percent of the sample. The dependent variable in the hazard model is years lived, where average years lived are 12.6 years. The symbols *, †, and ‡ indicate that in the probit the coefficient is significantly different from 0 and that in the hazard model the coefficient is significantly different from 1, at the 10, 5, and 1 percent level, respectively. Additional covariates include age in 1900 dummies, indicators of missing occupational information, dummies for whether the veteran was ever wounded in the war or ever had malaria, typhoid, stomach problems, or syphilis, dummies for size of city of residence in 1900, marital status in 1900, a dummy indicating if quarter of birth was unknown, and dummies indicating linkage to the 1860 and 1900 census.

Table 4: Independent Competing Risk Model of Years until Death from Heart Disease (Excluding Stroke) and from Stroke, Union Army Veterans Age 60-74 in 1900

	Heart Disease		Stroke	
	Haz Rat	Std Err	Haz Rat	Std Err
Dummy=1 if born				
First quarter	0.964	0.121	1.583†	0.336
Second quarter	1.141	0.139	1.701‡	0.362
Third quarter	1.251*	0.156	1.596†	0.347
Fourth quarter				
Dummy=1 if occupation circa 1900				
Farmer				
Professional	1.187	0.274	1.174	0.495
Manager/Proprietor	1.146	0.217	1.510	0.430
Clerical/Service	1.442*	0.294	1.172	0.407
Craft	1.319†	0.183	1.429	0.325
Operative	1.324*	0.225	1.658*	0.440
Service	1.472	0.407	1.921	0.788
Laborer	1.392‡	0.185	1.051	0.245
Dummy=1 if in service had				
Rheumatism/rheumatic fever	1.249†	0.133	0.778	0.161
Observations	2,178		2,178	
γ	0.081‡	0.005	0.106‡	0.008
$\chi^2(42)$ for				
Significance of all coefficients	84.760	0.000	62.780	0.021

The dependent variables are years lived until death from the specific causes. The symbols $*$, $\dagger$, and $\ddagger$ indicate that the coefficient is significantly different from 1 at the 10, 5, and 1 percent level, respectively. Additional covariates include dummies for size of city of enlistment, a dummy for personal property wealth in 1860, dummies for occupation at enlistment, a dummy if the veteran was a POW, dummies if the veteran had in service diarrhea, respiratory conditions, measles, or tuberculopsis, age in 1900 dummies, indicators of missing occupational information, dummies for whether the veteran was ever wounded in the war or ever had malaria, typhoid, stomach problems, or syphilis, dummies for size of city of residence in 1900, marital status in 1900, a dummy indicating if quarter of birth was unknown, and dummies indicating linkage to the 1860 and 1900 census.

Table 5: Effects of Illness in Service on Years until Death from Selected Acute Conditions (Independent Competing Risks Model), Union Army Veterans Age 60-74 in 1900

|	Infectious		Respiratory		Stomach	
	Haz Rat	Std Err	Haz Rat	Std Err	Haz Rat	Std Err
Dummy=1 if was POW	1.170	0.476	1.027	0.231	1.592‡	0.393
Dummy=1 if in service had						
Diarrhea	0.828	0.235	0.906	0.130	1.562‡	0.276
Respiratory	0.967	0.344	1.982‡	0.320	1.389	0.307
Measles	0.741	0.541	0.864	0.285	1.782*	0.579
Tuberculosis	4.113‡	1.870	0.988	0.326	0.240	0.243
Rheumatism/rheumatic fever	0.830	0.303	0.855	0.164	1.070	0.243
Observations	2,178		2,178		2,178	
γ	-0.027	0.020	0.092‡	0.008	0.037†	0.012
$\chi^2(42)$ for						
Significance of all coefficients	38.750	0.614	73.490	0.002	45.570	0.326

The dependent variables are years lived until death from the specific causes. The category respiratory includes chronic respiratory conditions, pneumonia, influenza, and acute bronchitis. The category stomach includes gastritis/duondentis and ulcers. The category infectious includes tuberculosis. The symbols *, †, and ‡ indicate that the coefficient is significantly different from 1 at the 10, 5, and 1 percent level, respectively. Additional covariates include dummies for quarter of birth, dummies for size of city of enlistment, a dummy for personal property wealth in 1860, dummies for occupation at enlistment, dummies for occupation circa 1900, age dummies, indicators of missing occupational information, dummies for whether the veteran was ever wounded in the war or ever had malaria, typhoid, stomach problems, or syphilis, dummies for size of city of residence in 1900, marital status in 1900, a dummy indicating if quarter of birth was unknown, and dummies indicating linkage to the 1860 and 1900 census.

Table 6: Comparison of the Effects of Quarter of Birth on Ten Year Mortality Rates of 60-79 Year Olds, Union Army Veterans, 1900, and Native-Born Americans, 1960-1980

| | UA Veterans | | 1960-1980 | |
	Coef	Std Err	Coef	Std Err
Dummy=1 if born in				
First quarter	0.020	0.022	0.003	0.005
Second quarter	0.042*	0.023	0.030‡	0.006
Third quarter	0.041*	0.023	0.013†	0.006
Fourth quarter				
Observations	4927		128	
Pseudo R^2/R^2	0.036		0.984	

The Union Army regression results are from a probit equation on individual level data in which the dependent variable is whether the veteran died within 10 years of being observed alive in 1900. The control variables are the full set of control variables used in Table 3. The 1960-1980 results are from the weighted ordinary least squares equation using group-level data given in Table 1. Ten year mortality rates in the Union Army sample are 0.445 and in 1960-1980 are 0.358. The symbols ‡ and † indicate that the coefficient is statistically significantly different from zero at the 1 and 5 percent level, respectively.

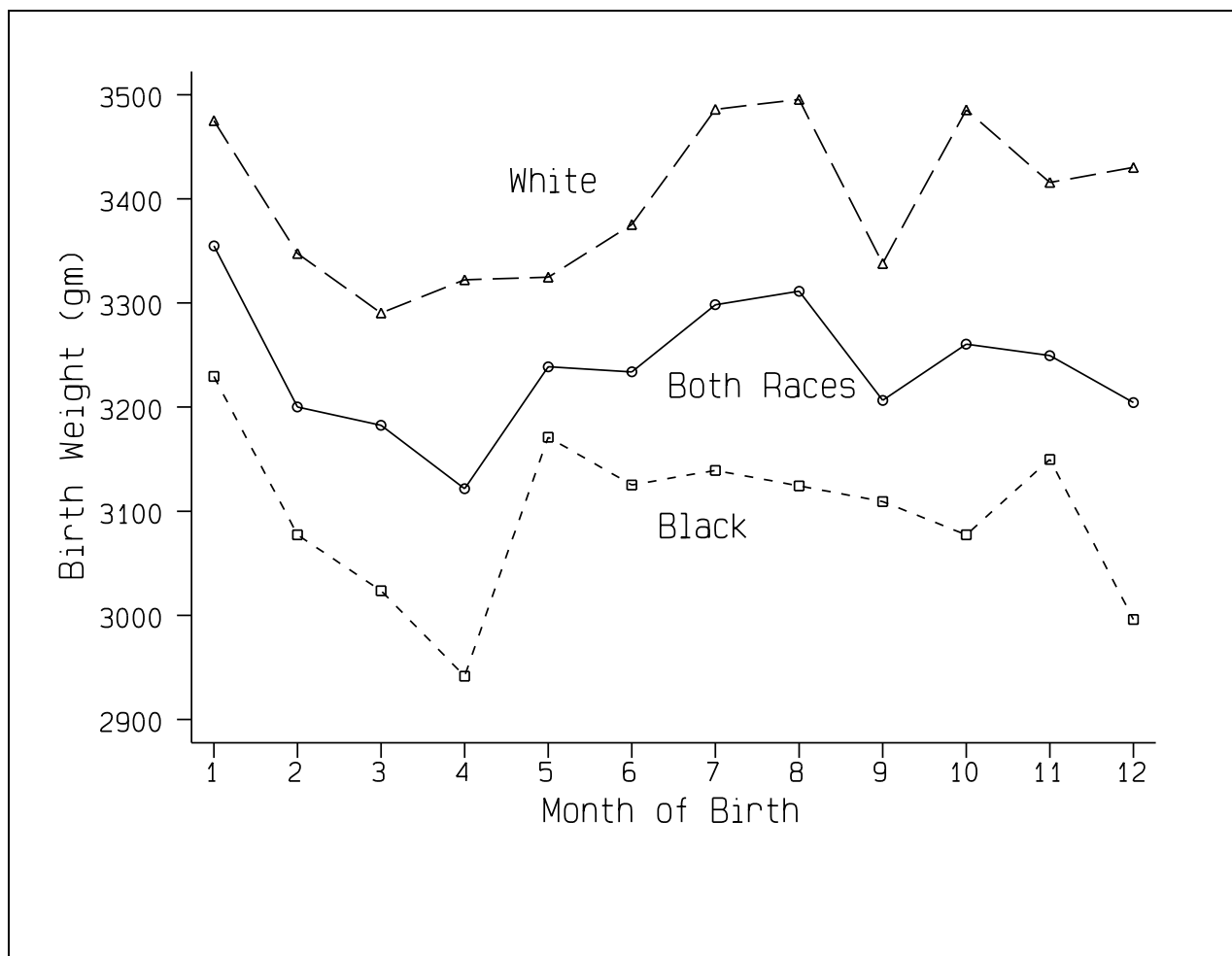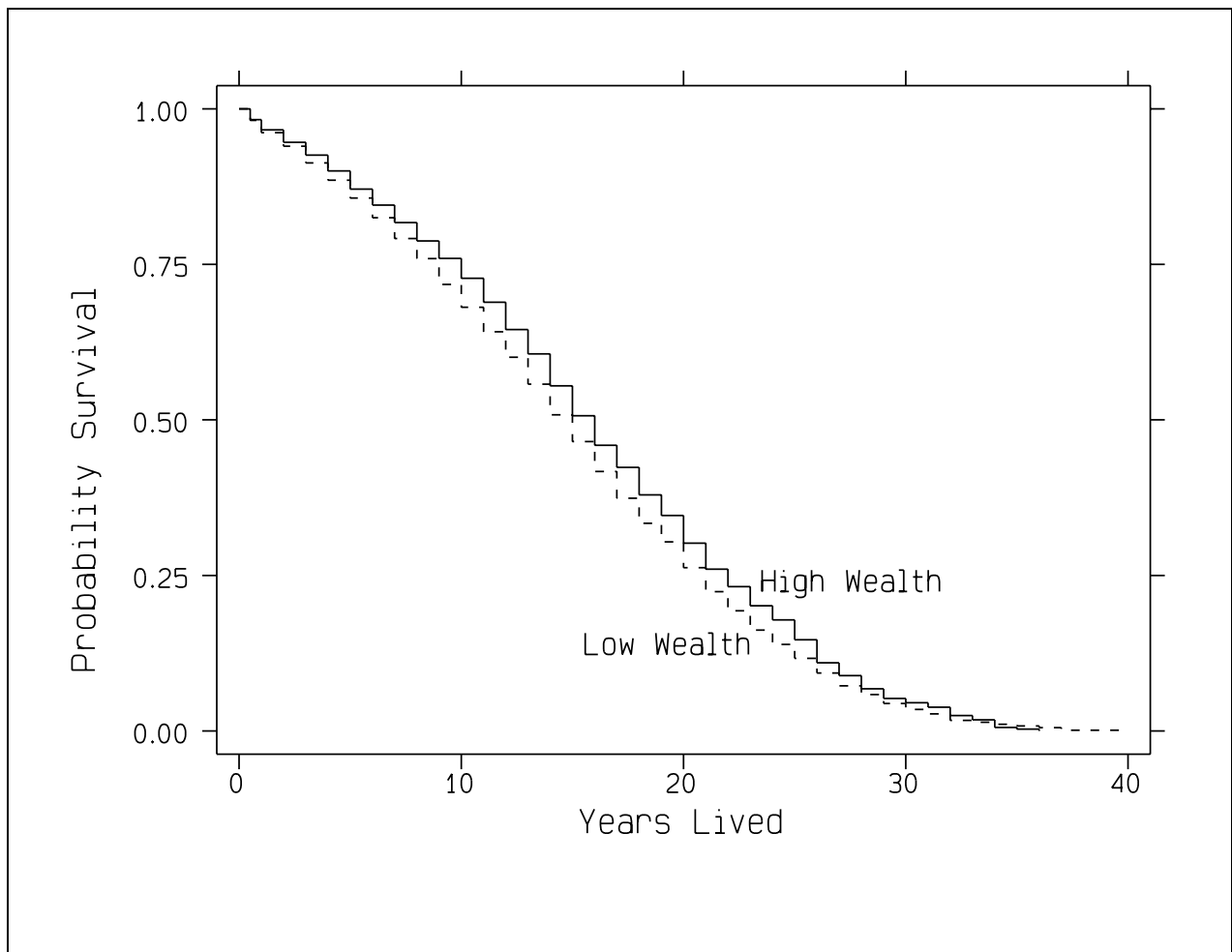Figure 1: Survival Probabilities Over Time by Quarter of Birth, Union Army Veterans Age 60-74 in 1900
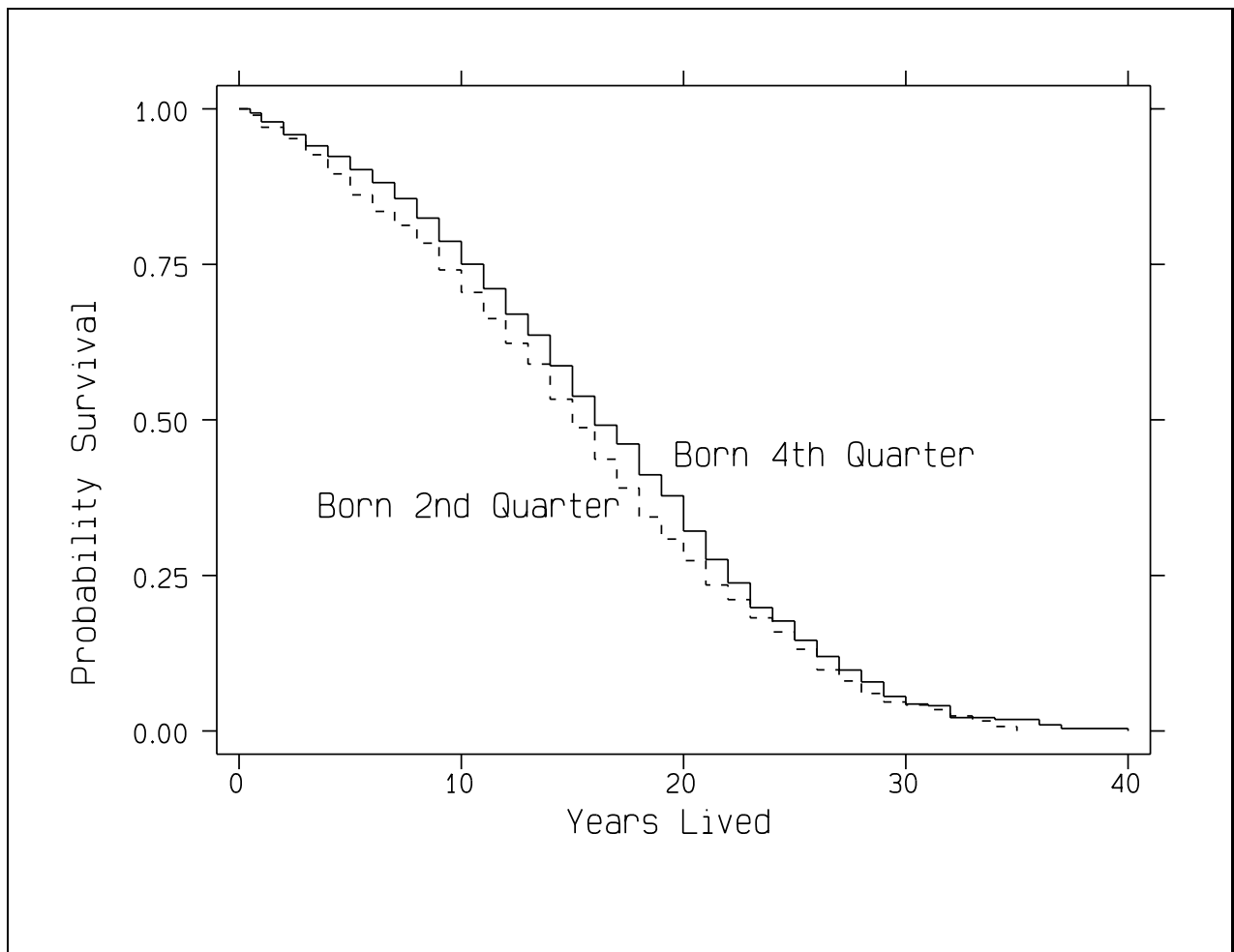
Source: Authors' calculations. Survival probabilities are adjusted for age. The sample was restricted to native-born veterans who were on the pension rolls by 1900.

Figure 2: Survival Probabilities Over Time by Size of City of Enlistment, Union Army Veterans Age 60-74 in 1900
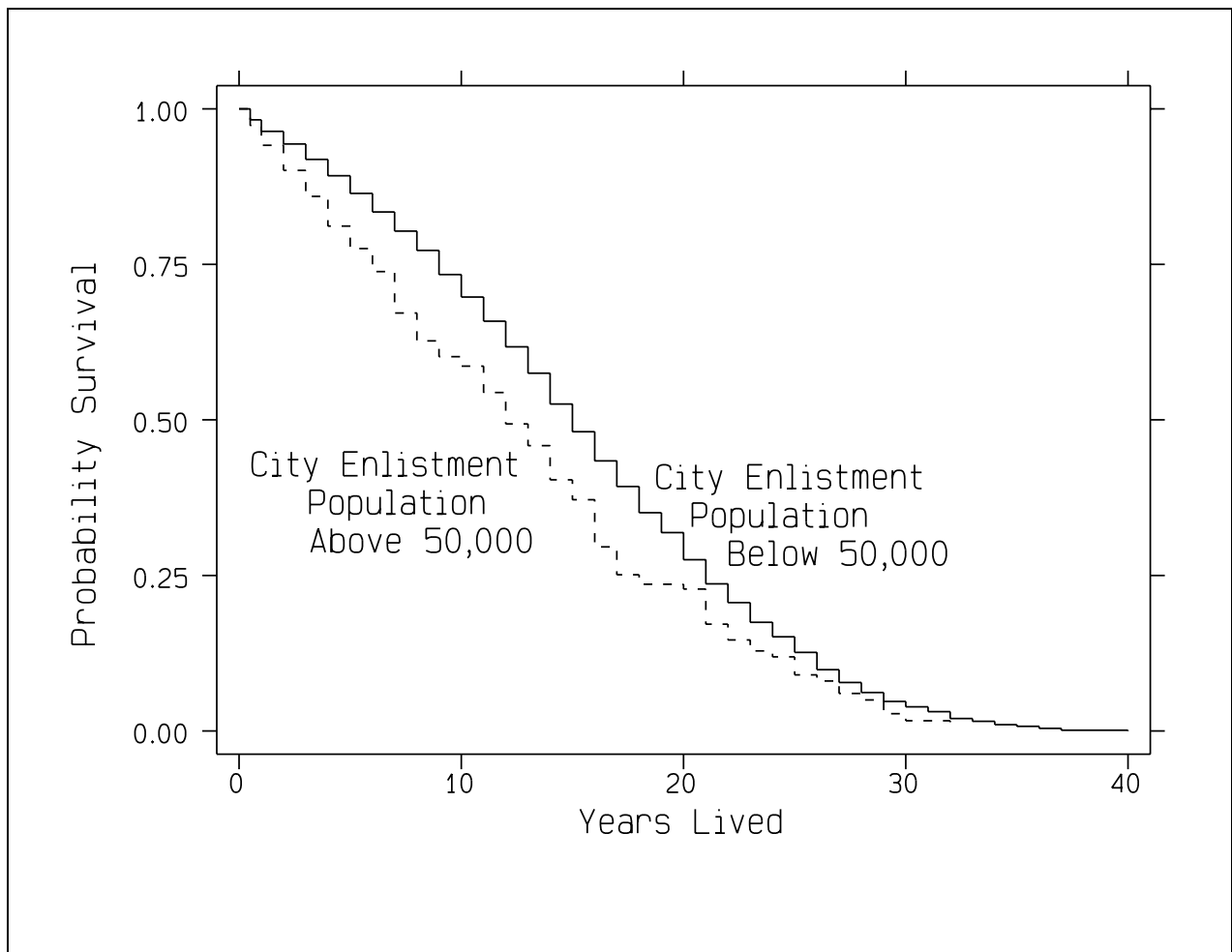
Source: Authors' calculations. Survival probabilities are adjusted for age. The sample was restricted to native-born veterans who were on the pension rolls by 1900.

Figure 3: Birth Weight and Month of Birth by Race of Live Births Attended by Johns Hopkins Physicians, 1897-1935
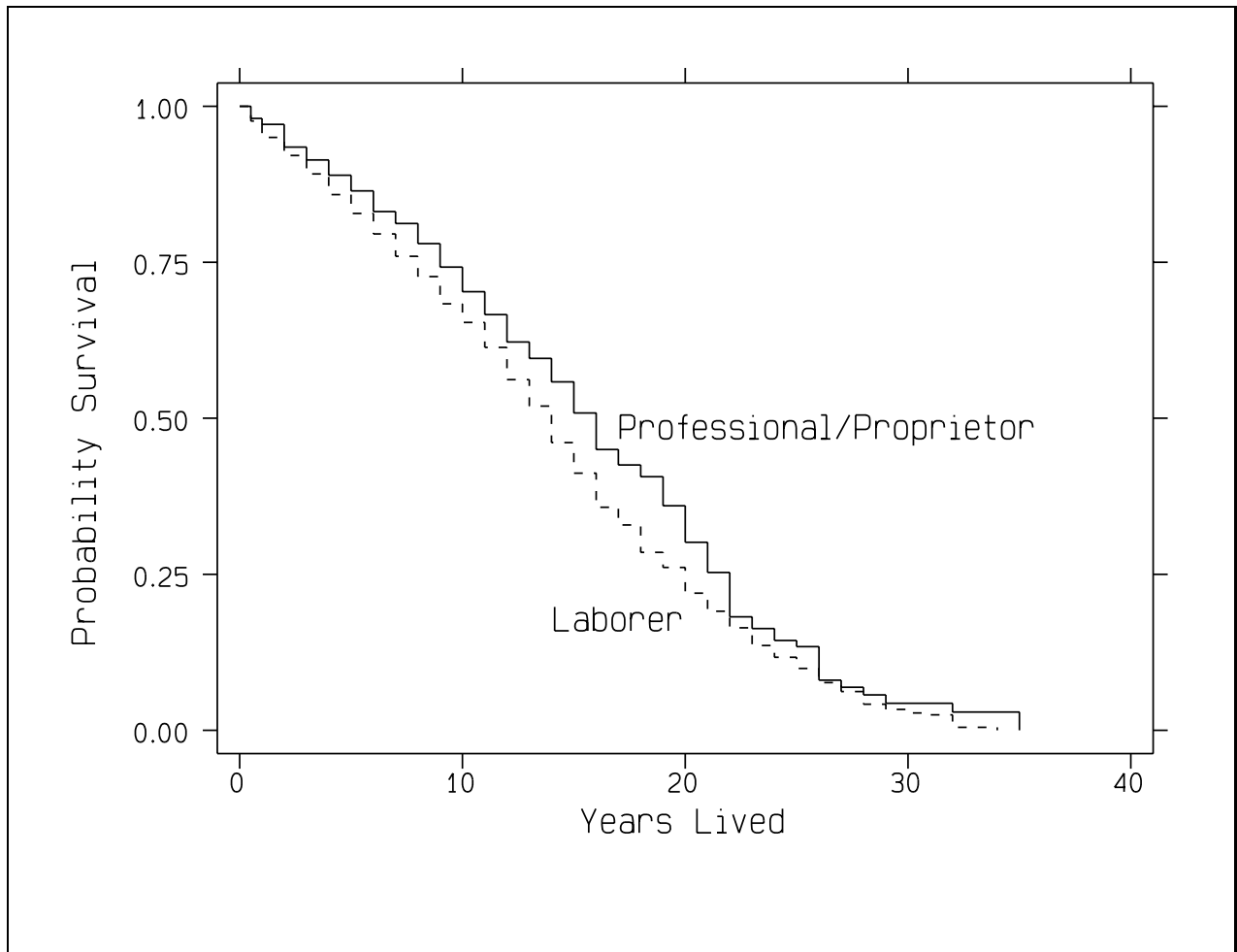
Source: Authors' calculations. See Costa (2003) for data details.

Figure 4: Survival Probabilities Over Time by Personal Property Wealth in 1860 Family, Union Army Veterans Age 60-74 in 1900


Source: Authors' calculations. High Wealth is defined as holdings of $250 or more. Survival probabilities are adjusted for age. The sample was restricted to native-born veterans who were on the pension rolls by 1900.

Figure 5: Survival Probabilities Over Time by Occupation at Enlistment, Union Army Veterans Age 60-74 in 1900

Source: Authors' calculations. Survival probabilities are adjusted for age. The sample was restricted to native-born veterans who were on the pension rolls by 1900.

Figure 6: Probability of Death for Union Army Veterans Age 85 and Older
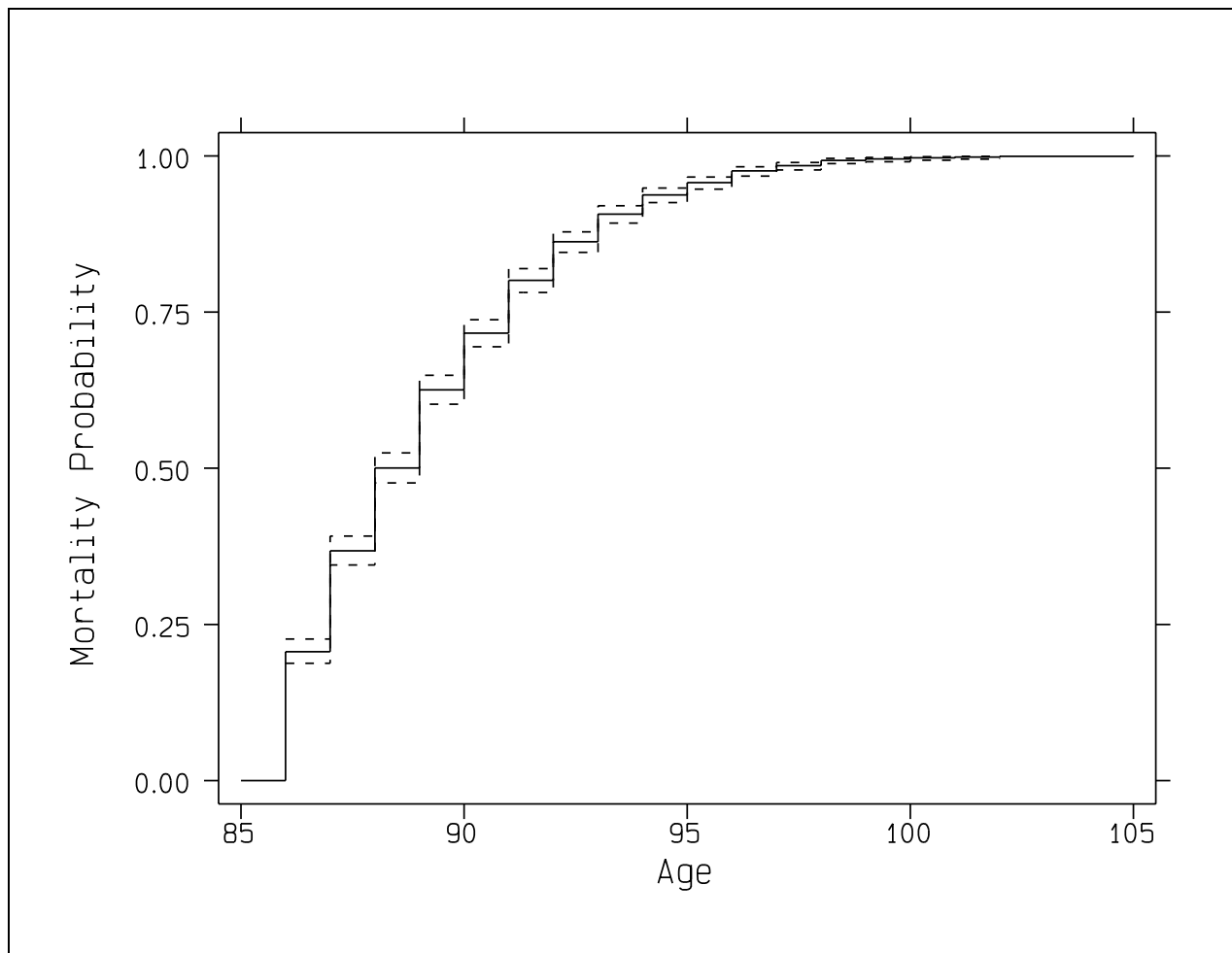
Source: Authors' calculations. The sample consists of 2036 Union Army veterans who survived to age 85 and were on the pension rolls by age 85. All ages were verified using both the pension records and the military records. The dashed lines indicate 95 percent confidence intervals.