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# SOCIOECONOMIC STATUS IN CHILDHOOD AND HEALTH AFTER AGE 70: A NEW LONGITUDINAL ANALYSIS FOR THE U.S., 1895-2005

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## ABSTRACT

The link between circumstances faced by individuals early in life (including those encountered in utero) and later life outcomes has been of increasing interest since the work of Barker in the 1970s on birth weight and adult disease. We provide such a life course perspective for the U.S. by following 45,000 U.S.-born males from the household where they resided before age 5 until their death and analyzing the link between the characteristics of their childhood environment – particularly, its socioeconomic status – and their longevity and specific cause of death. Individuals living before age 5 in lower SES households (measured by father's occupation and family home ownership) die younger and are more likely to die from heart disease than those living in higher SES households. The pathways potentially generating these effects are discussed.

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# *"Socioeconomic Status in Childhood and Health After Age 70: A New Longitudinal Analysis for the U.S., 1895-2005"*


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**Abstract**

The link between circumstances faced by individuals early in life (including those encountered *in utero*) and later life outcomes has been of increasing interest since the work of Barker in the 1970s on birth weight and adult disease. We provide such a life course perspective for the U.S. by following 45,000 U.S.-born males from the household where they resided before age 5 until their death and analyzing the link between the characteristics of their childhood environment – particularly, its socioeconomic status – and their longevity and specific cause of death. Individuals living before age 5 in lower SES households (measured by father's occupation and family home ownership) die younger and are more likely to die from heart disease than those living in higher SES households. The pathways potentially generating these effects are discussed.


## Introduction

Considerable attention has been devoted by epidemiologists and biologists to the links between early-life circumstances and later-life outcomes (Gluckman and Hanson, 2006). Both longevity and several specific mortality risks have been tied to conditions experienced very early in life. This literature has lacked two important components, however: large, nationally representative datasets that follow individuals over the course of an entire lifetime and an understanding of the economic and social context within which these early circumstances are encountered.

We attempt to address both of these shortcomings by providing the first examination of the link between economic circumstances faced in childhood and the health and longevity of individuals at older ages with a large dataset containing a rich set of early-life covariates. We do this by following more than 45,000 U.S.-born males from the 1900 U.S. Census of Population until their appearance in the Social Security Death Index and in state death records. This allows us to assess the link between family characteristics experienced by individuals when they were age 5 and under and both longevity and specific causes of death. Many of these characteristics (e.g. how many months the household head was unemployed, whether the family rented or owned its home) could never be accurately included (because of recall bias) in surveys that collect information retrospectively from individuals who have already achieved adulthood.

Though we lack the detailed information on later behaviors (e.g. smoking, obesity, substance abuse), we broaden the focus of this life course research by adding information on the economic characteristics of an individual's family. By doing so, we offer a new perspective on the economy-wide costs of poverty and economic insecurity: these circumstances can have lasting effects on lifetime health and productivity when they are experienced in childhood.

## The Developmental Origins Hypothesis

The observation that later life outcomes can reflect early circumstances has a long pedigree in the health sciences. Writing in 1829, Villermé asserted that "the circumstances which accompany poverty delay the age at which complete stature is reached and stunt adult height." (Villermé, 1829; quoted in Smith and Ebrahim, 2001) The link between early nutrition (including nutrition *in utero*) and later health was initially examined in the context of the "experiment" generated by the Dutch famine of 1944-45 (Stein *et al.*, 1975). The "fetal origins of adult disease" hypothesis (Barker, 1992) offered a specific mechanism linking insults suffered *in utero* (such as poor maternal nutrition or exposure to infectious disease) to chronic health impairments much later in life, such as coronary heart disease (CHD), type 2 diabetes, and osteoperosis. Long-lasting effects on later health of insults suffered in childhood can also be identified (Eriksson *et al.*, 2003; Singhal *et al.*, 2002). As a result of the recognition that circumstances both before birth and through early childhood can influence later health outcomes, some have suggested abandoning the term "fetal origins of adult disease" in favor of "developmental origins of health and disability," which encompasses the entire sequence from prenatal through neonatal and infancy through early childhood (Gluckman and Hanson, 2006).

The epidemiological evidence on the "developmental origins" hypothesis for specific chronic conditions is substantial (Godfrey, 2006). The link between low birthweight and CHD in adulthood initially identified by Osmond *et al.* (1993) and Barker (1998) in data from the U.K. has now been found in a variety of other populations (Frankel *et al.*, 1996; Rich-Edwards *et al.*, 1997). The focus in recent years has shifted from low birthweight itself to more comprehensive measures of fetal growth, such as head circumference and shortness or thinness (Barker *et al.*, 1993; Martyn *et al.*, 1996; Forsen *et al.*, 1997; Barker, 1998; Eriksson *et al.*, 1999, 2001). At the same time, links between growth after birth and CHD have now been established (Osmond *et al.*, 1993; Barker, 1998; Eriksson *et al.*, 2001). A wide range of other later-life complications are now recognized as linked to growth *in utero* and in childhood: type 2 diabetes (Lithell *et al.,* 1996), stroke (Martyn *et al.*, 1996), hypertension (Huxley *et al.*, 2000), muskuloskeletal health (Harvey and Cooper, 2004), respiratory health (Barker *et al.*, 1991), cognitive function (Gale *et al.*, 2003 and 2004), and mental health (Susser *et al.*, 1996; Sacker *et al.*, 1995).

Research on such linkages over the life course has been impeded in the U.S. by the combination of the lack of a centralized system of vital registration and high rates of interstate migration.[1] This has made it difficult to follow individuals over an entire lifetime. A notable exception is the recent Adverse Childhood Experiences (ACE) Study which is following more than 17,000 individuals and collecting information on current health, past behaviors, and adverse conditions experienced in childhood (e.g. psychological, physical, and sexual abuse, and household dysfunction) (Felitti *et al.*, 1998). The ACE Study examines only the most severe childhood experiences, and provides no information on how more common but less severe experiences – such as chronic or temporary household economic distress – are related to later outcomes.

The ACE Study finds that experiencing six or more adverse circumstances in childhood resulted in a statistically significant hazard ratio of 1.54, meaning that death was more than 50 percent more likely at every age for individuals who had experienced at least six adverse childhood experiences (Brown *et al.*, 2009). They find a clear dose-response relationship between adverse childhood experiences and the risk of engaging in behaviors (smoking, drug use, over-eating, unsafe sex) that contribute to the leading causes of death (Felitti *et al.*, 1998), and they find these early life conditions are associated with specific causes of death such as chronic obstructive pulmonary disease (Anda *et al.*, 2008), ischaemic heart disease (Dong *et al.*, 2004), autoimmune disease (Dube *et al.*, 2009), and liver disease (Dong *et al.*, 2003). One characteristic common to all of these analyses is the absence of information on the economic circumstances of the households in which individuals lived their early lives: if the economic stress experienced by a household was not sufficiently great to

[1] The U.S. National Center for Health Statistics (NCHS) receives electronic copies of the death records generated by each state and has compiled them into an annual Mortality Detail File since 1962. As a result of their agreements with the states, these NCHS files do not contain any identifying information that would make it possible to link decedents to their early life households. Since 1979, NCHS has provided a per-case service to search the files it possesses that do contain identifiers – the National Death Index (NDI). If the subject is know to be deceased, NCHS charges $5.00 for each matched record, so a sample of the size employed here would cost close to $250,000 to generate. In addition, the NDI only begins in 1979; the state death records that will be used here begin as early as the late 1950s.

generate psychological, physical, or sexual abuse or severe dysfunction then its influence will not be observed.

The modern study that is closest in methodology to the present study is Hayward and Gorman (2004) who use retrospective information on the early life household for individuals in the National Longitudinal Survey's Mature Men Cohort. For the U.S., we are aware of no other study linking early-life household-level economic circumstances to longevity. Their sample is both considerably smaller (4,562 adult men compared to nearly 45,000) and more subject to recall bias (they use self-reported early life circumstances – father's occupation and educational attainment, the absence of one or more parents, the mother's labor force status, and size of the place in which the family resided) than that used here, as early life conditions are reported by respondents themselves when they entered the survey after age 40.[2] Despite their relatively small sample size and retrospectively-reported early-life conditions, they find strong links between father's occupation, family structure, and mother's labor force status, though they are unable to assess the early-life correlates of specific cause of death or to include objective measure's of the family's circumstances or of the community's characteristics.

Economists and economic historians can offer a unique perspective on these issues, for two reasons. The first is that testing the "developmental origins of health and disability" is extremely time-consuming and expensive if done prospectively: large samples must be identified at birth if not earlier, and then followed for an entire lifetime to assess how very early life circumstances affect health through the oldest ages. Economic historians have recently conducted large-scale record linkage projects (Ferrie, 2005) that provide a tool that can be used to generate retrospective data to

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[2] Kauhanen *et al.* (2006) demonstrate the pitfalls of relying on retrospectively recalled measures of early life experience rather than objective measures actually generated in early life. The latter are much stronger predictors of longevity than the former when both are available.

address these questions at substantially lower cost than prospective studies. This can be done by identifying individuals in sources that reveal their health in the late twentieth century and early twenty-first century and locating those individuals in sources (e.g. manuscript schedules of the U.S. population censuses) that reveal their early-life circumstances.

The second contribution that economic historians can make to this literature is their detailed understanding of many of the household-level and community-level circumstances faced by individuals early in the century who are at risk to be linked to modern sources that document their later-life outcomes. Recent research on late-nineteenth and early-twentieth century families and communities – work on intrahousehold resource allocation decisions (Moehling, 2005; Logan, 2007), the urban mortality environment generally (Haines, 2001), the sanitation infrastructure of cities (Troesken, 2006; Ferrie and Troesken, 2008), the public health infrastructure and insurance markets (Thomasson, 2002; Thomasson and Treber, 2008; Almond *et al.,* 2010), and the impact of Depression-era spending on health and economic activity at the local level (Fishback *et al.,* 2007; Fishback *et al.,* 2005) – provides detailed context seldom available in modern studies of the link between early-life conditions and later-life outcomes.

An additional benefit of conducting this research through the lens of economic history is that it provides a long-run perspective on developments that have until now been discussed only within the narrow time-frame of their occurrence. For example, the epidemiological environment in the early twentieth century U.S. has been examined as a problem for early twentieth century Americans, particularly those living in cities. But if that environment and how it changed over time have an impact on individuals that is manifested only decades later, an important part of the story of those developments is being missed.

**Previous Historical Research on the Link Between Early and Later Life**

Several studies using historical populations have previously examined the link between circumstances early in life and late life health. The study of the "Dutch Famine" by Stein *et al.* (1975) was among the first. Since then, two approaches have been followed: (1) the identification of changes to the economic, social, or epidemiological environment experienced by particular birth cohorts that can then be traced to later outcomes of cohorts that experienced those shocks; and (2) the linkage of individuals across the life course to allow a direct examination of the link between each household's circumstances and the outcomes for individuals who experienced those circumstances early in their lives. This study takes the second approach, but in important ways complements the first approach.

Following the approach of Stein *et al.* (1975) that examines the association between longevity and macro shocks, van den Berg *et al.* (2006) linked macroeconomic records and death records for the Netherlands from 1812 to 2000 to assess the effect on longevity of having been born at different points in the business cycle. They find that individuals born in recessions live several years fewer than those born in better economic conditions. Similarly, Banerjee *et al.* (2010) find that those born in affected regions during the 1863 to 1890 phylloxera outbreak in France (which destroyed much of the wine crop and led to wide-spread hardship) differed in their later outcomes from those not in affected regions, but the effect was manifested in physical stature (a height gap of 1.8 millimeters at age 20) rather than in longevity. Bengtsson and Lindström (2000) find that between 1760 and 1894 in Sweden, the disease environment encountered in the first year of life is strongly associated with mortality at much older ages (55 to 80), though there is no apparent link to the food prices faced by the child's family early in the child's life. Finch and Crimmins (2004) document a similar link between the early disease environment and later mortality in Sweden since 1751, emphasizing the effect of disease exposure and inflammation. Alter and Oris (2005) find that rural-to-urban migrants (who experienced a relatively benign disease environment early im life) had better later life mortality than those born in mid-nineteenth century Belgian cities. Finally, Almond (2006) finds that those cohorts *in utero* during the 1918-19 influenza pandemic had worse health than those born before or after the outbreak; these effects are observed as late as 1980, more than 60 years after the event.

Palme and Sandgren (2008) employ the individual-level approach similar to that adopted here. They examine a region in Sweden where the 1928-42 household incomes of individuals born in 1928 can be linked to death records. They find that higher household income in early life was associated with lower mortality, and that low incomes were also associated with a higher risk of death from cancer. For Finland, Kauhanen *et al.* (2006) linked both objective measures of early life conditions and measures derived from recollections later in life to mortality and found that the former but not the latter was a strong predictor of adult mortality. Campbell and Lee (2009) examine a population in China that can be followed from childhood to death and find pronounced effects on mortality at older ages from the loss of a mother early in life, short birth intervals in the family, and high maternal age. These are among the only studies of which we are aware that follow individuals over the entire life course to assess the association between circumstances specific to the individual's early-childhood household and their later outcomes.

The approach adopted here – linking individuals from their appearance in the 1900 U.S. Census of Population to their appearance in Social Security and state death records – is the first time this approach has been applied to the U.S., resulting in a large, nationally representative sample. In this respect, it brings the individually-linked-records approach from the community or regional scale to the national scale. By linking census records that were contemporaneous with the child's early life, it avoids the problem of poor recall later in life of early life conditions identified as a problem by

Kauhanen *et al.* (2006). Finally, it provides the opportunity to assess the link between longevity and a variety of measures of household economic stress experienced early in life, each of which captures a different dimension of economic deprivation. At the same time, by employing data for a wide variety of locations in the U.S. at the start of the twentieth century, it allows us to examine community-level characteristics and evaluate in the cross-section some of the time-series differences identified in the shocks-based approach.

## Data Sources

We use three sources of data in our analysis: (1) individual-level records of individuals enumerated in the 1900 U.S. Census of Population (from the IPUMS 5% 1900 sample; see Ruggles *et al.*, 2009); (2) individual-level records contained in the Social Security Administration's Death Master File (DMF) (Hill and Rosenwaike, 2001/2002); and (3) computerized death certificates from California, Illinois, Massachusetts, Missouri, and Ohio. These sources contain sufficient common identifying information that it is feasible to link large numbers of individuals across all three sources.

The choice of 1900 as the census year to use as the base sample was made on the basis of three considerations: (1) this was the only early twentieth century U.S. census to report the actual year and month of birth for each individual – information that makes linkage to the DMF and state death records much easier; (2) this census (along with 1910) also provides a unique measure of the health environment at the household level – mothers reported both the number of children born and the number surviving; and (3) the cohort of individuals age 5 and under in 1900 has now completely died off so there is no right-censoring issue in the survival analysis we undertake. The linkage was restricted to U.S.-born males. The nativity constraint assures that we are observing circumstances experienced in the U.S. rather than abroad. The gender constraint was imposed because the linkage to the DMF and state death records relies on surnames, which will frequently change for females when they marry.

The linkage from the 1900 5% IPUMS file to the DMF was done on the basis of 5 characteristics: given name, middle initial (if reported in both sources), surname (with a slight tolerance allowed for mis-reporting in the census), month of birth, and year of birth. The initial IPUMS sample contained 254,641 individuals who were U.S.-born, male, reported as a "child" of another household member, not in group quarters, and age 0-5 in 1900, with month of birth reported and full first name (rather than just an initial) reported. Of these, 72,987 were linked to the DMF with at least one link and 51,179 were linked with exactly one.[3]

The rate of linkage with at least one link to the DMF (72,987/254,641=29%) compares very favorably to the projected linkage rate based on survival to the first year of the DMF (1965), inclusion in the DMF, and accurately reporting date of birth in both sources. For example, based on the 1900-2000 IPUMS samples, 46% of U.S.-born males under age 5 in 1900 survived to 1965 (3.0/6.5 million). Hill and Rosenwaike (2001/2002) examined the completeness of the DMF by age and calendar year, finding that 86% of males age 5 and under in 1900 who survived to 1965 should have appeared in the DMF.[4] Finally, Hambright (1969, p. 417) finds that 27% of white males and

[3] The Social Security Administration's NUMIDENT file, from which the DMF is extracted, provides additional identifying information that could potentially be used to resolve some of the cases in which an individual from the 1900 census was linked to more than one individual in the DMF. For example, the NUMIDENT reports the individual's place of birth (state, county, minor civil division) and full names for both parents. Unfortunately, when the NUMIDENT was computerized in the 1970s, this information had already been removed from the files of individuals who had already begun receiving Social Security benefits, which will be all of the individuals who were age 5 and under in 1900. There are statistical techniques to overcome this sort of uncertainty in an outcome measure, but the simpler expedient of focusing only on those individuals linked from one 1900 census record to exactly one DMF record will be used here.

[4] The incompleteness of the DMF results from two factors: (1) only individuals who ever had a spell of "covered employment" (i.e. work subject to FICA withholding) over their lifetimes entered the Social Security system, which will exclude individuals who were exclusively farmers or domestic workers for their entire work lives and who retired before these workers were brought into the Social Security system in the late

57% of nonwhite males had a different year of birth reported on their state death certificate and their U.S. census record from earlier in the same year in which they died (1960). If we assume that the accuracy for age reporting in comparing the census and the DMF is 75%, that the probability of inclusion in the DMF conditional on survival to 1965 is 86%, and that the survival rate from 1900 to 1965 is 46%, then the predicted rate of matching between the 1900 census and the DMF is 30%, only slightly higher than the actual rate achieved.

Table 1 reports the characteristics of the linked sample (for only the 44,620 observations for which all of the characteristics to be used in the analysis below are reported) in the first four columns. The last column shows the marginal effect of each characteristic on the probability that a unique match was made from the 1900 census to the DMF. The statistical significance of many of the variables on the probability of linkage is not surprising: recall that linkage will be higher for those who survive to 1965, who enter the Social Security system before their retirement, and whose census and DMF information is accurately reported. Individuals whose fathers were in higher status occupations, whose parents were literate, and who had at least one U.S.-born parent were more likely to be linked. The magnitude of these effects relative to the probability of linkage with all characteristics reported (20% for the sample of individuals linked to exactly one DMF record), however, is generally quite small. For example, individuals whose parents rented rather than owned their homes were only 1.3 percentage points less likely to be linked than otherwise identical individuals. The analysis in the following section will be conditional on survival to age 70, so the impact of even these small differences will be minimized.

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1950s; and (2) individuals who died before they began collecting their Social Security benefits are less likely to enter the DMF in the first 15 years after 1965 when reporting of deaths by surviving family members was voluntary before reporting of deaths to Social Security by funeral directors was required.

The second linkage performed was between 1900 census records (again using U.S.-born males who were age 5 and under in 1900, reported as the child of another household member, not residing in group quarters, and reporting a valid month and year of birth and a full given name) and computerized death records from five states that reflect a cross-section of locations: California, Illinois, Massachusetts, Missouri, and Ohio.[5] These records report an individual's state of birth as well as name and date of birth, so this additional information was used in the linkage process. This resulted in 6,542 unique matches, and a distribution across the five states in the linked data (California 33.7%, Illinois 19.7%, Massachusetts 11.8%, Missouri 12.2%, and Ohio 22.6%) quite similar to the distribution of deaths 1972-2004 in the DMF across these five states among those born 1894-1900 (California 36.5%, Illinois 20.7%, Massachusetts 12.5%, Missouri 10.8%, and Ohio 19.5%). Principal cause of death was coded by each state using the International Statistical Classification of Diseases and Related Health Problems (ICD) which has undergone two revisions since 1978. It was necessary to standardize these codes across ICD versions 8, 9, and 10.[6] Causes were then grouped into 7 broad categories employed by the Centers for Disease Control and the National Center for Health Statistics: (1) heart disease, (2) cancer, (3) chronic lower respiratory disease, (4) cerebrovascular disease, (5) influenza and pneumonia, (6) accidents and injuries, and (7) other. Though a case can be made for including "accidents and injuries" as a cause of death possibly influenced by early life circumstances (if, for example, those circumstances lead to a higher

[5] These five states were chosen because (1) they are among the easiest states from which computerized death records can be obtained; (2) their records contain place of birth information as early as 1970 (facilitating the linkage process); and (3) they each contain a mix of urban and rural locations. It is unfortunate that no Southern states have yet been identified that meet even two of these criteria.

[6] The most significant complication in the recoding was the lack of a code for Alzheimer's disease in ICD-8 and ICD-9. This condition accounts for roughly 9% of deaths above age 65 by the year 2000. In order to keep the classification of causes by group consistent across the three ICD versions, Alzheimer's was placed in the "other" category.

likelihood of physical frailty or cognitive impairment at older ages which make an individual more susceptible to accidents and injuries), this category is included here instead as a falsification test: the link to early circumstances is more tenuous, so we expect the effect of early life circumstances to be more attenuated for this category than for any other.

An important shortcoming of the linkage process is that it captures only those individuals whose were enumerated in the 1900 census, who had their age in the census and on their death certificate accurately recorded, and – in the case of the state death records – who died in one of the five states from which death records were obtained. All of these introduce the possibility of bias. We have identified three potential biases and provide sensitivity analyses in two Appendix tables that address all three.

In linking African Americans age 85+ who died in the first two weeks of January 1985 back to the 1900 U.S. Census manuscripts, Preston *et al.* (1996, 1998) and Hill *et al.* (2000) found substantial (+20%) disagreement for blacks between age reported in the 1900 or 1910 census and in 1985 death certificates. Hambright (1969) finds the mis-reporting rate to be twice as high for blacks as for whites even when the census and the death certificate pertain to the same year (1960). Age mis-reporting, to the extent that it differs by race, will result in a sample that has too few blacks. Similarly, if households lower in socioeconomic status are less likely to report ages correctly, either in the census or in death records, the linked sample will be biased toward high-SES households.

One solution for the bias against blacks and low-SES households would be to take a sample of individuals from death records and attempt to locate them in the census manuscripts, which is essentially the procedure employed by Preston *et al.* For the anti-black bias, a simpler expedient is used here – conducting alternative analyses that are limited to whites. For the anti-low-SES bias, assessing the bias by looking at linkage rates by SES at death will not help if the poor linkage of low-

SES households results from their exclusion from the census or their mis-reporting of information in the census or if their SES has changed substantially since early life. Instead, we have provided alternative analyses in which the share of households with low SES in 1900 (households in which the father was a laborer) has been arbitrarily increased by 20%, and the shares of the other SES groups have been diminished correspondingly to yield a weighted sample with the same number of observations as the original sample.[7]

A third source of bias pertains only to the 1900 census records linked forward to the state death records. Since we have death records from only five states, the linked population will be a mix of (1) people who both were born and died in these states; and (2) people who were born elsewhere and later migrated into one of these five states. Compared to group (1), group (2) will mechanically have greater longevity since migrants are likely to have been at risk to migrate longer than non-migrants. This issue is further complicated by differential rates of migration by race and socioeconomic status, and the possibility that migration selects for those those with better economic prospects and those in better health. This may be a particular problem in the present case as the period following 1900 saw a substantial migration of blacks out of the South, many of whom ended up in some of the states we are using (Ohio, Illinois, Missouri). Some recent evidence suggests that the selectivity by economic prospects and health for blacks might not be particularly large. Eichenlaub *et al.* (2010) find that blacks who left the South in the Great Migration fared no better

[7] This assumes that the link between covariates and outcomes among the low-SES households that *were* matched is the same as that among the low-SES households that *were not* matched. If even among low-SES households, some characteristics are associated with both the probability of linkage and mortality later in life, the sample will remain biased. If the true relationship between SES and longevity is positive, weighting will bias the case against finding such a relationship: in reality, more individuals born into low-SES households will have died before 1965 than individuals born into high-SES households, so the weighted sample will contain too many low-SES households that have been selected for longer life spans (on the basis of their survival to 1965) than in the population of individuals born into low-SES households.

than intra-South movers or non-movers, and Black *et al.* (2010) find that age-specific mortality rates did not differ when blacks who migrated out of the South are compared to blacks who remained in the South, even after taking account of the endogeneity of the migration decision.

Differences between blacks and whites in the ages at which migration occurs could still generate longevity differences, since migration will in general select for individuals who are older. This selectivity will differ according to when in the life course migration is most likely to occur. Black *et al.* (2009, Table 2, Panel B) show that among blacks, the fraction residing in their state of birth has risen steadily since 1950, but for whites this fraction has steadily fallen over the same period. These opposing trends reflect the end of the Great Migration for blacks (which coincided with a search for work and generally occurred when these individuals were younger) and the start of substantial Sun Belt migration for whites (which coincided with retirement and generally occurred when these individuals were older). As a result, within our five states there could be too many long-lived whites and too few long-lived blacks relative to what we would observe if we had death records for all fifty states. To account for this possibility, two approaches are adopted in the analysis of cause of death: (1) an analysis that focuses exclusively on whites; and (2) an analysis that focuses exclusively on individuals who were born in the state where they died.

**Socioeconomic Status, Survival After Age 70, and Specific Cause of Death**

The large sample of individuals we have linked from the 1900 U.S. Census to the DMF and to the death records of five large states allows us to include such economic influences on later health and to do so with a sample that is nearly ten times larger than that of Hayward and Gorman (2004) and better able than theirs to assess links between SES in childhood and specific cause of death. Two health outcomes will be examined here: longevity (years survived after age 70) and specific cause of death. Several measures of the economic circumstances faced by families around 1900 will be used:

1. Father's occupation, measured either by an occupational prestige score on a continuous scale from 0 to 100 (Hoge *et al.*, 1964) or by three categorical variables ("white collar," "farmer," and "skilled/semiskilled") where the excluded category is "laborer"

2. Months unemployed for the household head in the 12 month preceding the 1900 census

3. Home ownership, measured by the household's response to the question on whether the house was rented or owned and if it was owned whether it was mortgaged or owned free and clear

4. An interaction between whether the home was rented and the number of months the household head was unemployed in order to capture the link between particularly precarious economic circumstances and subsequent health.

Each of these early-life household SES measures captures a different dimension of the household's economic circumstances, so we anticipate they will have different associations with the later life health of the child. The household head's occupation is a relatively stable measure of the flow of resources into the household: it likely reflects the resources that are available to be divided between consumption and investment on a regular basis. Fathers in lower-income occupations (laborers) will have children who are chronically poorer fed and housed and may be less able to make investments in the child's human capital (health or education) that other fathers. A strong link between this measure of SES and longevity could indicate the importance of chronic economic stress throughout early life (including *in utero*).

The household head's months of unemployment in the 12 months prior to the census is an indicator of episodic rather than chronic economic stress for the household. It represents interruptions to the household's income flow. If the household has a stock of resources that it can draw down to compensate for short-run income fluctuations, these spells of unemployment need not diminish the household's current consumption and impair the child's long-run health. To account for this possibility, the household's home ownership status is included as well – renters are less likely to have substantial savings and collateral against which to borrow, so their ability to even out fluctuations in income is limited. An interaction between months of unemployment and whether the family rented rather than owned its dwelling is included to allow for the possibility that unemployment's association with longevity is stronger when the family is asset-poor. There are several margins at which the household can economize when income declines. Families that own their home do not need to worry about making rental payments, so they have greater flexibility; families that rent instead may economize on expenditures such as food (a possibility explored below), which can have deleterious long-term consequences for its children's health. The precariousness of the economic circumstances of families that rent their homes might also be associated with more subtle effects (also described below) that cause changes in behavior across a lifetime.

The analysis also includes a set of county-level variables to account for the possibility that household-level characteristics merely reflect circumstances experienced across the county. For example, the fraction of the county's dwellings that are rented or mortgaged rather than owned outright is included, so the link between longevity and the household's rental status is net of the influence of the prevalence of rental dwellings in the larger community (where the latter may reflect a higher-density housing stock or generally low levels of household income). The fraction of the county's children employed in manufacturing is included to account for the possibility that as they become older, the children observed under age 5 in 1900 may face different opportunities to trade off schooling for paid employment. The anticipated link between this variable and longevity, however, is ambiguous: children who have more opportunity to work may receive less education if their work reduces their school time, but if the income they bring to the household increases their leverage in the intra-household bargaining process, they may garner a larger share of household resources in exchange for their diminished human capital (Moehling, 2005; Logan, 2007). The county-wide fraction of males and females employed in manufacturing is included as well to account for the possibility that episodic economic stress may be more severely felt in places with a greater sensitivity to business cycle fluctuations (e.g. counties more reliant on manufacturing for employment). Finally, the average number of persons per dwelling is included as well to allow crowding elsewhere in the county to be associated with an individual's outcomes even if his own family is not particularly space-constrained.

The hazard analysis in Table 2 begins with the simplest specification in Columns 1 and 2: survival as a function of early life SES (measured by the household head's months of unemployment and either the father's occupational prestige or the father's specific occupation group), along with a set of control variables.[8] Hazard ratios above one indicate that the covariate is associated with a reduced survival time and values below one indicate that it is associated with an increased survival time.[9] When occupational prestige is used as the SES indicator in Column 1, a positive and statistically significant SES-longevity association appears: a child born into a household one standard deviation above the mean SES (48: manager) faces a mortality risk after age 70 that is 10 percentage

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[8] The survival analysis assumes a Weibull hazard. The null hypothesis that this is true cannot be rejected for any of the eight specifications in Table 2. We also experimented with the Gompetrz, exponential, log-logistic, and gamma distributions for duration dependence and found no change in the qualitative findings described below. The analysis also assumes the presence of a gamma-distributed frailty parameter, which acts much like a random effect in the context of an ordinary least squares regression – it can account for some forms of unobserved individual heterogeneity. We were unable to account for family effects because only 5% of the observations linked to the SSDI were from families that had another member in the linked sample, and because the parameters of interest (e.g. father's occupation) do not vary at the household level and would be absorbed into any family fixed effect. In order to ensure that all individuals are "at risk" for the same number of years, the analysis here is limited to deaths at age 70½ and older.

[9] Note that, like Palme and Sandgren (2008), we are reluctant to conclude that the relationships we detect are causal. Like them, we cannot rule out genetic inheritance or the social transmission of habits, and are aware of the possibility of omitted variables. Almond, Chay, and Lee (2005) show that the omission of genetic factors can substantially bias cross-sectional estimates of the impact on later-life outcomes of at least one very early-life condition (exposure to programs to prevent low birth weight).

points lower than a child born into a household one standard deviation below the mean SES (22: janitor). When occupational categories are used instead of prestige (Column 2), the highest SES occupations (white collar workers and farmers) have children whose mortality risk is 11-18 percentage points less after age 70 than the lowest SES occupations (skilled/semiskilled and laborers). There is no discernable link between longevity and the household head's months of unemployment.[10]

The balance of Table 2 includes a variety of additional household characteristics in order to see whether the positive SES-longevity link remains after other influences on longevity are removed. Columns 3 and 4 add home ownership and its interaction with the household head's unemployment. Individuals whose parents rented rather than owner their homes had lower survival rates after age 70: children of renters had a mortality risk 8-11 percent greater than that of children of owners. This relationship is statistically significant in each specification where it appears. An interaction between home rental and unemployment does not, however, put the individual at additional risk over that associated with home rental itself.

Of the remaining covariates in Table 2, the most interesting are the child mortality rate in the family and the county-wide variables. A child born into a household with a child mortality rate ½ standard deviation below the mean (1%) had a mortality risk 2.8 percentage points lower than that of a child born into a household with a child mortality rate ½ standard deviation above the mean (14%). The family's child mortality rate may be capturing the association between the community mortality rate and longevity, but it may also be a function of a general frailty in the children born to

[10] To assess whether the link between longevity and a father's unemployment differed by the age at which the individual experienced that early life circumstance, analyses like those in Table 2 were done separately for individuals who were under age 1 in 1900. The association between longevity and unemployment was unchanged.

particular mothers – frailty which manifests itself in extreme cases in early death and in less extreme cases within the same household in earlier death even after achieving age 70.[11] Of the county-wide variables, the only statistically significant results are the negative link to risk for the fraction of the county's children employed in manufacturing (risk rises by 2.5 percentage points going from ½ standard deviation above the mean to ½ standard deviation below the mean, suggesting that greater availability of employment opportunities for children was protective with respect to longevity, perhaps through the intra-household resource allocation channel described above) and the positive link with fraction of dwellings that were not owned outright.

The appendix contains a version of Table 2 that is modified in two respects: in Column 1 of Table A1 , the analysis is limited to whites only, and in Column 2 of Table A1, the analysis is both limited to whites and uses a weight for low-SES households (laborers) that is 20% higher than in the actual linked data. These analyses are designed to allow for the possibility that the results in Table 2 are being driven by the exclusion of blacks whose ages were poorly reported or by the exclusion of low-SES households that might have escaped census enumeration altogether. Neither change alters the basic findings in Table 2: lower risk for children born into high-prestige occupation households and home-owning households. The magnitudes of these associations persist as well as their statistical significance.

Despite the inclusion of a broad set of additional controls, the association between early-life SES and longevity shown in Columns 1 and 2 persists: its magnitude is reduced only slightly and its

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[11] For a subset of 10 states in the U.S. Death Registration Area as of 1900 (Connecticut, Indiana, Maine, Massachusetts, Michigan, New Hampshire, New Jersey, New York, Rhode Island, and Vermont) and the District of Columbia, we are in the process of adding city-level detail on mortality, as well as rural mortality.

statistical significance remains high. This suggests that SES is not simply a reflection of other household characteristics. But are the coefficients in Table 2 large or small?

One way to evaluate their magnitude is to compare them to the associations estimated in Hayward and Gorman (2004) who have a similar methodology. They find that the hazard ratio for the sons of farmers, compared to the sons of unskilled laborers, was 0.83, compared to 0.82 in Table 2. Their hazard ratios for white collar workers ranges from 0.64 to 0.85, compared to 0.89 in Table 2. Though they do not have information on home ownership status, it is possible to get a sense of the magnitude of its association with longevity in Table 2 by comparing it to some of the well-known health risks Hayward and Gorman do include: children of renters in Table 2 faced a mortality risk roughly 10% greater than did children of owners, while Hayward and Gorman report that individuals who were smokers after age 45 had a mortality risk 25% higher than non-smokers. The link between growing up in a rented home and longevity, then, is nearly half the size of the link between smoking and longevity much later in life.

Brown *et al.* (2009, p. 393) report that individuals who suffered six or more of the adverse childhood experiences they examine (which include violence and substance abuse in the household, loss of a parent, and sexual, physical, and emotional abuse) had a hazard for mortality at or below age 75 that was 74% above that of individuals who experienced none of these conditions. Though their figures are not directly comparable to those in Table 2 as they pertain to an analysis of mortality before age 75 rather than after age 70, this again provides a rough guide to the magnitude of the association between early life and longevity uncovered here: the growing up with a farmer father was associated with a reduced risk of 18.3 percent, compared to a reduction of 43 percent in the ACE for individuals who experience none of the adverse childhood experiences rather than six of them.

Table 3 turns from longevity to the specific causes of death recorded for individuals in their state death certificates.[12] It is necessary to account for the fact that these causes of death are competing risks. A proportional hazards model of the sub-hazard of each competing risk is used (Fine and Gray, 1999); this makes calculation of the cumulative incidence function straightforward. The standard Cox proportional hazards model (in which each competing risk is treated as a censoring event) produced similar results. The table reports the sub-hazard ratio for each covariate, together with the standard error and significance level for the coefficient underlying the ratio; values above one again indicate that the covariate reduces survival time and values below one indicate that it increases survival time.

The strongest association between cause of death and SES is in heart disease: children born into households that were renters were 11.7% more likely to die from heart disease at each age after age 70 than children born into homes that were owned free and clear. Those living as children in households that were renters or owners with a mortgage were less likely to die from influenza or pneumonia than those living in homes owned free and clear. In order to account for the possibility that the precariousness of a household's income was a greater problem when it rented rather than owned its home outright, an interaction between the household head's unemployment and whether the home was rented was added. For heart disease, the interaction was statistically insignificant and its associated hazard rate was below one. If we nonetheless take the coefficients for heart disease at face value (including both the direct unemployment and rental status terms and their interaction), we can predict the corresponding incidence of heart disease at each age for an individual: (1) living in a rented home with 6 months of household head unemployment; (2) living in a rented home with no

[12] In order to ensure that all individuals are "at risk" for the same number of years, the analysis here is limited to deaths at age 77½ and older.

household head unemployment; (3) living in a home owned free and clear with 6 months of household head unemployment; and (4) living in a home owned free and clear and no household head unemployment is shown in Figure 2 which presents the cumulative incidence function (CIF) for each of these combinations.

Among the other covariates, the most substantial link between cause of death and early-life conditions comes from the absence of a father, which raises the probability of death from chronic lower respiratory disease by a factor of three. This finding is particularly interesting as it is the early life conditions in our analysis most similar to two of the adverse childhood experiences in the ACE Study. Chronic lower respiratory disease was also elevated among those born in the west but depressed among those whose parents were both born outside the U.S. Living in a household that had experienced greater child mortality was associated with a lower risk at each age of dying from influenza or pneumonia. The only characteristics that predicted differential rates of death from cancer were residence under age 5 in an urban place (higher risk), larger family size (lower risk), and residence in a county where more children were employed in manufacturing (lower risk).

The only previous study to examine the link between cause-specific mortality and household circumstances early in life at the individual level, Palme and Sandgren (2008), found that family income early in life was strongly protective for death from cancer. The present study found no such link. There is strong evidence from the "developmental origins" literature that the link between early life conditions and heart disease is relatively strong, while no such strong link has been established for cancer.[13] Rural Sweden is also a substantially different environment from the early twentieth century U.S., particularly rapidly growing urban places, so perhaps such a difference is to be

[13] The only exception is an association between early life weight and breast cancer (Victora *et al.*, 2008).

expected. Finally, Palme and Sandgren follow their subjects only to age 75, while the competing risks analysis of cause of death conducted here uses individuals who had already survived to age 77½. The discrepancy between their findings and ours nonetheless merits further examination.

In order to account for the possibility described above that the results in Table 3 are driven by peculiarities of the sample (particularly, the absence of blacks whose ages were often poorly reported, the absence of those in low-SES households who may have been missed by the census altogether, and the presence of interstate migrants whose behavior might be very different from that of non-migrants), three strategies are now followed: (1) a separate analysis for whites only; (2) an analysis with low-SES households weight by an additional 20% over their representation in the original linked sample; and (3) a separate analysis for non-migrants only (those who died in the same state where they were born). These appear in the second of the two Appendix tables, Table A2. Only the last of these changes the results – it reduces the coefficient on "Home Rented" below statistical significance (from a *p*-value=0.026 to a *p*-value=0.176), which may reflect the substantial reduction in the sample size, which goes from 6,503 to 3,767.

**Pathways and Explanations**

Several mechanisms can account for the observed positive SES-health gradient and the association between low SES and heart disease. The simplest is that the SES we observe in 1900 is a proxy for the SES experienced when the individual was *in utero*. There are widely-accepted explanations for how deprivation during crucial periods of fetal development can lead to specific health risks later in life. But the analysis of longevity in Table 2 was unchanged when the sample was separated into those under age 1 (for whom the father's 1900 occupation and unemployment and the family's housing situation would provide the best measure of *in utero* stress) and others. The link between these early life characteristics and later health even when "early life" includes time after the extreme plasticity of the *in utero* experience has ended suggests that another pathway may be in operation.

An alternative mechanism is that measured SES in 1900 is not so much a proxy for recent SES as it is a proxy for the SES individuals will experience over the rest of their lives. Parents with poor job prospects and few assets transmit those disadvantages to the next generation through inadequate investments in the health and schooling of their children, who in turn experience lives of continual economic disadvantage. The cumulative effect of this disadvantage, as well as the behaviors that accompany it (substance abuse, obesity, high-risk sexual activity), then leads to premature death. This hypothesis cannot be ruled out at this point, as it requires additional information on the economic circumstances faced by these individuals later in life. When we possess such information (e.g. adult income), we can then see whether poor health outcomes persist even for those who improve upon the economic circumstances they faced in early childhood.[14] Such a mechanism, however, seems inconsistent with high rates of intergenerational economic mobility. Ferrie (2005, p. 208, Figure 1) shows that mobility across generations, at least when measured by self-reported occupation, remained as high for the cohort of sons born 1880-1895 and observed in 1920 as it had been for other cohorts born in the nineteenth century when rates of intergenerational mobility exceeded those in Britain at the same time or later seen in the late twentieth century U.S.[15]

The third potential pathway focuses instead on a direct link between early-life experience at age 5 and under and long-term health. The relationship between early-life SES and later-life health is seen in father's occupation and the household's home ownership, but not in the father's

[14] We are in the process of obtaining such information from the Social Security Administration.

[15] Xie and Killewald (forthcoming) have questioned these mobility estimates. See their comment, and the authors' response.

unemployment experience. This suggests that is chronic rather than episodic economic stress at ages 5 and under that is damaging to later life health.[16] Families that rented rather than owned their homes in the late nineteenth century spent a larger fraction of total household income on food (43%) than families that owned their homes (38%).[17] Though in actual expenditures renters spent only $1.60 less on food each year per child than owners, the higher share of food in their family budget left them more vulnerable to even short spells of reduced income. This could result in direct negative consequences for children's development among renters if the margin that must be reduced when income falls was more likely to be food than was the case among owners.

Even in the absence of actual scarcity, the stress of simply living with the prospect of periods of scarcity may induce physiological responses in the developing child that lead to later life health difficulties. This is the mechanism advanced by the ACE Study: early-life stress induces a series of changes in brain chemistry that can lead later in life to the adoption of specific behaviors (substance abuse, unsafe sexual activity, physical inactivity) and psychological conditions (depression, negative affect) that are themselves important contributors to premature mortality and to elevated risk for specific health conditions. Evidence for this view in the ACE study comes from the reduction in the estimated association between early-childhood experiences and later health when later-life behaviors

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[16] An alternative explanation for why the link between longevity and unemployment is weak is that unemployment reported in the 1900 census did not reflect the household head's recent past unemployment experience. Davis's (2004) new business cycle chronology locates a severe recession from 1892 (peak) to 1897 (trough). Margo (2000, p. 242; see also Hatton and Williamson, 1991) notes that, unlike modern U.S. labor markets, the nineteenth century U.S. labor market had a weak relationship between personal characteristics and the probability of unemployment (resulting in an unemployment regime that others have characterized as an "industrial lottery"). Taken together, these facts suggest that many of the households reporting no unemployment in the 1900 census would have been unemployed for some time in the five years prior to the census.

[17] Calculated from the 1889/1890 Commissioner of Labor consumer expenditure survey (Haines, 2006).

are introduced as mediating influences. In the absence of information on specific later-life behaviors, we cannot say whether the SES-health gradient we observe is a direct or indirect relationship.

In separate analyses of SES and specific cause of death (not shown), where occupational categories were used in place of occupational prestige, the only substantial difference across the four categories was an elevated risk of cancer for children born to skilled and semi-skilled workers. The association between cancer deaths and growing up in a household with a skilled or semiskilled father remains unexplained at this point, though we are exploring two mechanisms: (1) fathers in these workplaces, more so than white collar, farmer, or general unskilled laborer fathers, introduce hazardous materials into their own homes, and the health of their children is compromised from early life as a result (Chiaradia *et al.*, 1997); (2) children of these father are more likely to follow them into similar occupations and experience environmental hazards in their own workplaces as adults. When the sample is expanded and we can look more closely at the specific industries in which skilled and semiskilled fathers were employed, we will be able to assess the first mechanism; when we have information on the later-life occupations of our linked individuals, we can also assess the second.

**Conclusions**

The relationship between childhood SES (measured either by father's occupation or home ownership) and health much later in life (measured either by longevity or risk for specific health conditions) persists even after the inclusion of a rich set of individual-level and household-level covariates. That such associations are observed at all after age 70 is an indication of the strength of this relationship: as the ACE Study notes,

> as individuals continue to age into the 7$^{th}$, 8$^{th}$, and 9$^{th}$ decades, it becomes more challenging to discern any influence of exposure to traumatic stress during childhood despite evidence suggesting that such events may become "hardwired" into an individual's biology. This is particularly true for mortality outcomes because most deaths occur among people in older age groups (Brown *et al.*, 2009, p. 394)

In future work, we can include at least some measures (education, income) of later-life SES that might mediate between early life and later health.

The link between SES early in life and health in later life demonstrates that, for the twentieth century U.S., some portion of the burden of economic insecurity is borne at a time quite distant from that when the insecurity is experienced. Some caution must be exercised in interpreting these findings, however, as there are at least four important shortcomings of the analysis conducted here. The first is the lack (at present) of information on circumstances at mid-life (e.g. occupation and home ownership or rental in 1930, income or educational attainment in 1940). As noted above, it is possible that the circumstances observed in childhood are merely an indicator of the individual's subsequent conditions, and that these conditions in turn drive the link to longevity and specific causes of death.[18] The second shortcoming is the lack of information on behaviors such as smoking and drinking.[19] The third is the lack (at present) of any information on females, for whom the association between early-life conditions and later-life health and longevity has been shown in other contexts to be very different from that of men (Catalano and Bruckner, 2006). Finally, the linked data used here has no information on morbidity – the only indicator of the quality of the additional years of longevity associated with higher early-life SES is the specific cause of an individual's death. When these linked records are joined to Social Security disability records, there will be some scope for further analysis of how many of the extra years of life associated with a high-SES childhood are lived free from the most debilitating conditions.

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[18] The elimination of the statistical significance of father's occupation as a predictor of mortality in Hayward and Gorman (2004) when own later-life occupation and education were used as well suggests that the same may happen here.

[19] Both the ACE study and Hayward and Gorman (2004) reveal how introduction of these covariates can reduce the magnitude of the association between early-life circumstances on longevity.

Despite these caveats, the present study has demonstrated the feasibility of following substantial numbers of individuals from early life to death, and shows substantial evidence of links between the circumstances experienced in life and both longevity and specific cause of death. The strongest links to longevity come from father's occupation and whether the family owned a home. The former may be associated with low levels of household resources over a span of years, and the latter may be associated with the family's lack of financial reserves and susceptibility to short-run fluctuations in income. Perhaps most surprisingly, an association between these circumstances and both longevity and specific cause of death can be observed 70 years after the early-life circumstances are experienced, despite the innumerable intervening life course events. This suggests both the strength of these links and the extent to which an analysis of household economic circumstances may be quite inadequate in the absence of a life-course perspective.

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Table 1. Descriptive Statistics for Variables in Survival Analysis and Marginal Effects on Matching.

| Variable | Mean | Standard Deviation | Minimum | Maximum | $\frac{\partial P(Matched)}{\partial X_j}$ |
|---|---|---|---|---|---|
| *Individual*: | | | | | |
| Residence in Northeast | 0.2665 | 0.4421 | 0.0000 | 1.0000 | |
| Residence in Midwest | 0.3968 | 0.4892 | 0.0000 | 1.0000 | -0.0049*** |
| Residence in South | 0.2874 | 0.4526 | 0.0000 | 1.0000 | -0.0555*** |
| Residence in West | 0.0493 | 0.2166 | 0.0000 | 1.0000 | -0.0221*** |
| Urban (Pop. > 2,500) | 0.3334 | 0.4714 | 0.0000 | 1.0000 | -0.0132*** |
| Non-White | | | | | |
| Born Jan-Mar | 0.2604 | 0.4388 | 0.0000 | 1.0000 | -0.0036 |
| Born Apr-Jun | 0.2350 | 0.4240 | 0.0000 | 1.0000 | -0.0082*** |
| Born Jul-Sep | 0.2595 | 0.4384 | 0.0000 | 1.0000 | 0.0022 |
| Born Oct-Dec | 0.2452 | 0.4302 | 0.0000 | 1.0000 | |
| | | | | | |
| *Family:* | 0.0686 | 0.2529 | 0.0000 | 1.0000 | -0.0451*** |
| Occup. Prestige of Hhld. Head | 35.4016 | 12.7508 | 0.0000 | 81.5000 | 0.0008*** |
| White Collar | 0.1415 | 0.3485 | 0.0000 | 1.0000 | |
| Farmer | 0.4083 | 0.4915 | 0.0000 | 1.0000 | |
| Skilled/Semiskilled | 0.2563 | 0.4366 | 0.0000 | 1.0000 | |
| Laborer | 0.1939 | 0.3954 | 0.0000 | 1.0000 | |
| Hhld. Head Mos. Unemployed | 0.5444 | 1.5908 | 0.0000 | 12.0000 | -0.0006 |
| Home Owned & Mortgaged | 0.1786 | 0.3830 | 0.0000 | 1.0000 | 0.0074*** |
| Home Rented | 0.5593 | 0.4965 | 0.0000 | 1.0000 | -0.0130*** |
| (Home Rented) x (Mos.Unemp.) | 0.3609 | 1.3002 | 0.0000 | 12.0000 | -0.0003 |
| Father Absent | 0.0213 | 0.1445 | 0.0000 | 1.0000 | 0.0311*** |
| Mother Absent | 0.0082 | 0.0903 | 0.0000 | 1.0000 | -0.0044 |
| Family Size | 3.7555 | 2.1076 | 1.0000 | 14.0000 | -0.0011*** |
| Family Child Mortality (0-100) | 8.3731 | 15.6404 | 0.0000 | 92.3077 | -0.0001*** |
| Father Foreign Born | 0.0734 | 0.2608 | 0.0000 | 1.0000 | -0.0011 |
| Mother Foreign Born | 0.0420 | 0.2006 | 0.0000 | 1.0000 | -0.0005 |
| Both Parents Foreign Born | 0.1561 | 0.3629 | 0.0000 | 1.0000 | -0.0439*** |
| Father Literate | 0.9046 | 0.2937 | 0.0000 | 1.0000 | 0.0316*** |
| Mother Literate | 0.9049 | 0.2934 | 0.0000 | 1.0000 | 0.0366*** |
| Mother in Labor Force | 0.0345 | 0.1825 | 0.0000 | 1.0000 | -0.0015 |
| | | | | | |
| *County:* | | | | | |
| Pct. Children in Mfg. | 0.6523 | 0.9753 | 0.0000 | 15.8358 | -0.0005 |
| Pct. Adult Males in Mfg. | 14.7935 | 13.2897 | 0.0000 | 74.1936 | 0.0003** |
| Pct. Adult Females in Mfg. | 3.5850 | 5.0691 | 0.0000 | 43.3329 | 0.0016*** |
| Avg. Persons/Dwelling | 5.5800 | 2.2889 | 3.1156 | 20.3944 | -0.0038*** |
| Pct. Homes Rented/Mortgaged | 63.7607 | 14.2318 | 3.9911 | 93.6131 | -0.0001 |

Note: Observations for descriptive statistics: 44,620. For the probit regression on matching from the 1900 U.S. Census to the Social Security Death Index, * p<0.10, ** p<0.05, *** p<0.01, Predicted Prob.=0.1984, L.R. $\chi^2$=3934.78, Prob. > $\chi^2$ = 0.0000, Pseudo-$R^2$=0.0171, Observations=227,789.

Table 2. Survival Analysis of Age at Death, U.S.-Born Males Age 70½+ (Hazard Ratios).

| | (1) | (2) | (3) | (4) | (5) | (6) | (7) | (8) |
|---|---|---|---|---|---|---|---|---|
| *Individual and Family Level:* | | | | | | | | |
| Father's Occ. Prestige (0-100) | 0.996*** | | 0.996*** | | 0.996*** | | 0.996*** | |
| | (0.001) | | (0.001) | | (0.001) | | (0.001) | |
| Father White Collar | | 0.890*** | | 0.904** | | 0.905** | | 0.892** |
| | | (0.047) | | (0.047) | | (0.047) | | (0.047) |
| Father Farmer | | 0.817*** | | 0.837*** | | 0.838*** | | 0.830*** |
| | | (0.037) | | (0.038) | | (0.038) | | (0.038) |
| Father Skilled/Semiskilled | | 0.984 | | 0.990 | | 0.990 | | 0.990 |
| | | (0.038) | | (0.038) | | (0.038) | | (0.038) |
| Father's Mos. Unemployed | 1.005 | 1.000 | 1.002 | 0.995 | 1.002 | 0.995 | 1.002 | 0.995 |
| | (0.008) | (0.008) | (0.014) | (0.014) | (0.014) | (0.014) | (0.014) | (0.014) |
| Family Child Mortality (0-100) | 1.002*** | 1.002*** | 1.002*** | 1.002*** | 1.002*** | 1.002*** | 1.002*** | 1.002*** |
| | (0.001) | (0.001) | (0.001) | (0.001) | (0.001) | (0.001) | (0.001) | (0.001) |
| Home Owned & Mortgaged | | | 0.987 | 0.992 | 0.987 | 0.992 | 0.980 | 0.985 |
| | | | (0.041) | (0.041) | (0.041) | (0.041) | (0.042) | (0.041) |
| Home Rented | | | 1.113*** | 1.102*** | 1.110*** | 1.101*** | 1.090*** | 1.081** |
| | | | (0.034) | (0.034) | (0.034) | (0.034) | (0.035) | (0.035) |
| (Home Rented) x (Fath. Mos. Unemp.) | | | 1.004 | 1.006 | 1.004 | 1.006 | 1.004 | 1.006 |
| | | | (0.016) | (0.016) | (0.016) | (0.016) | (0.016) | (0.016) |
| Family Size | | | | | 0.998 | 1.000 | 0.999 | 1.001 |
| | | | | | (0.006) | (0.006) | (0.006) | (0.006) |
| Father Literate | | | | | | | 0.969 | 0.965 |
| | | | | | | | (0.054) | (0.055) |
| Mother Literate | | | | | | | 0.927 | 0.923 |
| | | | | | | | (0.050) | (0.050) |
| Mother in Labor Force | | | | | | | 1.016 | 1.012 |
| | | | | | | | (0.079) | (0.079) |

(Continued)

Table 2. (Continued)

| | (1) | (2) | (3) | (4) | (5) | (6) | (7) | (8) |
|---|---|---|---|---|---|---|---|---|
| *County Level:* | | | | | | | | |
| Pct. Children in Mfg. | | | | | | | 0.964** | 0.964** |
| | | | | | | | (0.018) | (0.018) |
| Pct. Adult Males in Mfg. | | | | | | | 1.001 | 1.001 |
| | | | | | | | (0.002) | (0.002) |
| Pct. Adult Females in Mfg. | | | | | | | 0.997 | 0.998 |
| | | | | | | | (0.004) | (0.004) |
| Avg. Persons/Dwelling | | | | | | | 1.000 | 1.001 |
| | | | | | | | (0.004) | (0.004) |
| Pct. Homes Rented/Mortgaged | | | | | | | 1.003** | 1.003** |
| | | | | | | | (0.001) | (0.001) |
| ln(*p*) (Weibull Shape) | 3.423*** | 3.423*** | 3.421*** | 3.421*** | 3.421*** | 3.421*** | 3.419*** | 3.419*** |
| | (0.025) | (0.025) | (0.025) | (0.025) | (0.025) | (0.025) | (0.025) | (0.025) |
| ln(θ) (Gamma Variance) | 0.847*** | 0.847*** | 0.843*** | 0.843*** | 0.843*** | 0.843*** | 0.839*** | 0.839*** |
| | (0.042) | (0.043) | (0.042) | (0.043) | (0.043) | (0.043) | (0.042) | (0.042) |
| Controls | | | | | | | | |
| 1900 Region of Residence | X | X | X | X | X | X | X | X |
| 1900 Urban Residence | X | X | X | X | X | X | X | X |
| Race | X | X | X | X | X | X | X | X |
| Birth Quarter | | | | | X | X | X | X |
| Father Absent in 1900 | X | X | X | X | X | X | X | X |
| Mother Absent in 1900 | | | | | X | X | X | X |
| Father Foreign-Born | | | | | | | X | X |
| Mother Foreign-Born | | | | | | | X | X |
| Both Parents Foreign-Born | | | | | | | X | X |

(Continued)

Table 2. (Continued)

| | (1) | (2) | (3) | (4) | (5) | (6) | (7) | (8) |
|---|---|---|---|---|---|---|---|---|
| Likelihood Ratio $\chi^2$ | 95.405 | 105.846 | 115.976 | 123.216 | 122.044 | 128.687 | 197.667 | 206.562 |
| Prob. > $\chi^2$ | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 |
| Observations | 44,620 | 44,620 | 44,620 | 44,620 | 44,620 | 44,620 | 44,620 | 44,620 |
| Median Age at Death | 80.321 | 80.321 | 80.321 | 80.321 | 80.321 | 80.321 | 80.321 | 80.321 |

Note: Robust standard errors (clustered by county) of the coefficients underlying the hazard ratios in parentheses. Each model assumes a Cox proportional hazard, a Weibull duration dependence distribution (shape parameter *p*), and a gamma frailty distribution (mean one, variance $\theta$).

* p<0.10 ** p<0.05 *** p<0.01

Table 3. Cause-Specific Survival Analysis Accounting for Competing Risks U.S.-Born Males Age 77½+ (Sub-Hazard Ratios).

| Characteristic in 1900 Census Manuscripts | Cause of Death: Accidents | Heart Disease | Cancer | Chronic Lower Respiratory Disease | Cerebro-vascular Disease | Influenza and Pneumonia | Other |
|---|---|---|---|---|---|---|---|
| *Individual and Family Level:* | | | | | | | |
| Residence in Midwest | 1.016 | 1.053 | 1.005 | 1.211 | 1.020 | 0.778 | 0.846 |
| | (0.274) | (0.075) | (0.105) | (0.213) | (0.119) | (0.122) | (0.114) |
| Residence in South | 0.640 | 1.178* | 0.996 | 1.234 | 0.990 | 0.810 | 0.742 |
| | (0.252) | (0.117) | (0.164) | (0.352) | (0.175) | (0.206) | (0.160) |
| Residence in West | 0.763 | 1.042 | 1.016 | 1.676** | 1.072 | 0.742 | 0.847 |
| | (0.261) | (0.100) | (0.160) | (0.401) | (0.172) | (0.198) | (0.154) |
| Urban (Pop. > 2,500) | 0.901 | 0.889** | 1.179** | 0.981 | 0.963 | 1.137 | 1.226* |
| | (0.180) | (0.047) | (0.094) | (0.162) | (0.093) | (0.146) | (0.141) |
| Non-White | 1.732 | 0.913 | 0.785 | 0.949 | 0.560* | 1.394 | 1.551* |
| | (0.789) | (0.117) | (0.188) | (0.443) | (0.173) | (0.446) | (0.395) |
| Occup. Prestige of Hhld. Head | 1.000 | 0.999 | 1.002 | 0.996 | 1.000 | 0.993 | 1.004 |
| | (0.006) | (0.002) | (0.003) | (0.005) | (0.004) | (0.004) | (0.004) |
| Father Absent | 0.789 | 0.750 | 0.818 | 3.036** | 1.420 | 0.216* | 2.436** |
| | (0.681) | (0.144) | (0.257) | (1.623) | (0.486) | (0.171) | (1.050) |
| Mother Absent | 0.852 | 1.006 | 1.028 | 1.559 | 0.836 | 2.066 | 0.238 |
| | (0.925) | (0.248) | (0.451) | (0.905) | (0.374) | (1.240) | (0.245) |
| Hhld. Head Mos. Unemployed | 0.870 | 1.030 | 1.012 | 1.016 | 0.958 | 0.940 | 0.934 |
| | (0.091) | (0.019) | (0.031) | (0.057) | (0.037) | (0.057) | (0.052) |

(Continued)

Table 3. (Continued)

| Characteristic in 1900 Census Manuscripts | Cause of Death: Accidents | Heart Disease | Cancer | Chronic Lower Respiratory Disease | Cerebro-vascular Disease | Influenza and Pneumonia | Other |
|---|---|---|---|---|---|---|---|
| Home Owned & Mortgaged | 0.746 | 1.100 | 0.964 | 0.985 | 0.917 | 0.743* | 1.260* |
| | (0.167) | (0.066) | (0.092) | (0.190) | (0.097) | (0.121) | (0.173) |
| Home Rented | 0.736* | 1.117** | 0.915 | 1.079 | 0.857* | 0.756** | 1.223* |
| | (0.128) | (0.056) | (0.078) | (0.170) | (0.079) | (0.101) | (0.144) |
| (Home Rented) x (Mos.Unemp.) | 1.171 | 0.966 | 0.997 | 0.985 | 1.054 | 1.069 | 1.070 |
| | (0.138) | (0.023) | (0.035) | (0.068) | (0.050) | (0.078) | (0.068) |
| Born Jan-Mar | 0.717 | 1.084 | 1.034 | 0.866 | 0.967 | 0.891 | 1.009 |
| | (0.153) | (0.056) | (0.092) | (0.151) | (0.099) | (0.132) | (0.120) |
| Born Apr-Jun | 0.980 | 1.032 | 1.014 | 0.966 | 0.904 | 1.072 | 1.076 |
| | (0.200) | (0.057) | (0.088) | (0.162) | (0.105) | (0.169) | (0.132) |
| Born Jul-Sep | 1.248 | 0.931 | 1.119 | 0.937 | 1.197* | 0.841 | 0.966 |
| | (0.229) | (0.047) | (0.085) | (0.152) | (0.116) | (0.116) | (0.119) |
| Family Size | 0.930* | 1.005 | 0.966** | 0.971 | 1.012 | 1.049* | 1.042* |
| | (0.037) | (0.010) | (0.016) | (0.030) | (0.019) | (0.027) | (0.022) |
| Family Child Mortality (0-100) | 1.006 | 1.002 | 1.000 | 1.000 | 0.998 | 0.996** | 1.000 |
| | (0.005) | (0.001) | (0.003) | (0.004) | (0.002) | (0.002) | (0.003) |
| Father Foreign Born | 0.913 | 0.959 | 0.906 | 1.064 | 0.865 | 0.814 | 1.511*** |
| | (0.245) | (0.066) | (0.109) | (0.241) | (0.126) | (0.181) | (0.208) |
| Mother Foreign Born | 1.067 | 0.877 | 0.883 | 0.759 | 1.387** | 1.218 | 1.280 |
| | (0.375) | (0.086) | (0.135) | (0.233) | (0.200) | (0.278) | (0.258) |
| Both Parents Foreign Born | 1.296 | 0.996 | 0.999 | 0.584** | 1.000 | 1.352** | 0.944 |
| | (0.288) | (0.061) | (0.100) | (0.131) | (0.102) | (0.187) | (0.130) |

(Continued)

Table 3. (Continued)

| Characteristic in 1900 Census Manuscripts | Cause of Death: Accidents | Heart Disease | Cancer | Chronic Lower Respiratory Disease | Cerebro-vascular Disease | Influenza and Pneumonia | Other |
|---|---|---|---|---|---|---|---|
| Father Literate | 1.564 | 0.884 | 0.838 | 1.518 | 1.119 | 0.957 | 1.530 |
| | (0.755) | (0.100) | (0.156) | (0.603) | (0.247) | (0.321) | (0.451) |
| Mother Literate | 1.121 | 1.207* | 0.955 | 0.673 | 0.733* | 1.479 | 0.922 |
| | (0.476) | (0.125) | (0.176) | (0.193) | (0.132) | (0.448) | (0.211) |
| *County Level:* | | | | | | | |
| Pct. Children in Mfg. | 0.954 | 1.039 | 0.850** | 1.041 | 1.095 | 0.916 | 0.954 |
| | (0.202) | (0.043) | (0.063) | (0.098) | (0.078) | (0.119) | (0.090) |
| Pct. Adult Males in Mfg. | 0.975* | 1.005 | 1.009* | 1.008 | 0.998 | 0.986* | 0.988* |
| | (0.013) | (0.003) | (0.005) | (0.009) | (0.005) | (0.008) | (0.006) |
| Pct. Adult Females in Mfg. | 1.041* | 0.994 | 1.004 | 1.017 | 0.988 | 1.018 | 1.015 |
| | (0.024) | (0.008) | (0.014) | (0.021) | (0.013) | (0.022) | (0.017) |
| Avg. Persons/Dwelling | 1.026 | 0.995 | 1.029 | 1.009 | 0.995 | 0.976 | 0.953 |
| | (0.051) | (0.012) | (0.022) | (0.047) | (0.024) | (0.043) | (0.029) |
| Pct. Homes Rented/Mortgaged | 1.004 | 1.001 | 0.998 | 0.988** | 0.999 | 1.008 | 1.002 |
| | (0.009) | (0.003) | (0.004) | (0.006) | (0.005) | (0.007) | (0.006) |
| Pseudo Log-Likelihood | -1762.847 | -23650.849 | -9186.638 | -2387.528 | -6395.202 | -3083.831 | -4739.541 |
| Pseudo Log-Likelihood $\chi^2$ | 37.359 | 43.205 | 40.691 | 37.371 | 43.533 | 49.101 | 53.073 |
| Predicted Probability | 0.089 | 0.025 | 0.044 | 0.088 | 0.023 | 0.006 | 0.002 |
| Observations | 6,503 | 6,503 | 6,503 | 6,503 | 6,503 | 6,503 | 6,503 |

Notes: Cause-specific sub-hazard ratios (Fine and Gray, 1999) are shown along with the robust standard errors that correspond to the coefficients underlying the sub-hazard ratios.
* p<0.10, ** p<0.05, *** p<0.01

Table A1. Survival Analysis of Age at Death, U.S.-Born Males Age 70½+ (Hazard Ratios).

| | White Only | White Only & Low SES Weighted +20% |
|---|---|---|
| *Individual Level:* | | |
| Father's Occ. Prestige (0-100) | 0.995*** | 0.995*** |
| | (0.001) | (0.001) |
| Father's Mos. Unemployed | 1.002 | 1.000 |
| | (0.014) | (0.014) |
| Family Child Mortality (0-100) | 1.002** | 1.002* |
| | (0.001) | (0.001) |
| Home Owned & Mortgaged | 0.980 | 0.980 |
| | (0.042) | (0.042) |
| Home Rented | 1.089** | 1.091** |
| | (0.036) | (0.036) |
| (Home Rented) x (Fath. Mos. Unemp.) | 1.002 | 1.003 |
| | (0.016) | (0.017) |
| Family Size | 1.001 | 1.001 |
| | (0.007) | (0.007) |
| Father Literate | 0.939 | 0.931 |
| | (0.063) | (0.063) |
| Mother Literate | 0.917 | 0.919 |
| | (0.058) | (0.058) |
| Mother in Labor Force | 0.962 | 0.984 |
| | (0.099) | (0.100) |
| *County Level:* | | |
| Pct. Children in Mfg. | 0.970* | 0.969* |
| | (0.018) | (0.019) |
| Pct. Adult Males in Mfg. | 1.001 | 1.002 |
| | (0.002) | (0.002) |
| Pct. Adult Females in Mfg. | 0.997 | 0.997 |
| | (0.004) | (0.004) |
| Avg. Persons/Dwelling | 1.000 | 1.000 |
| | (0.004) | (0.004) |
| Pct. Homes Rented/Mortgaged | 1.003** | 1.003** |
| | (0.001) | (0.001) |
| ln($p$) (Weibull Shape) | 3.408*** | 3.418*** |
| | (0.025) | (0.026) |
| ln(θ) (Gamma Variance) | 0.822*** | 0.838*** |
| | (0.043) | (0..043) |

(Continued)

Table A1 (Continued).

| | White Only | White Only & Low SES Weighted +20% |
|---|---|---|
| Controls | | |
| 1900 Region of Residence | X | X |
| 1900 Urban Residence | X | X |
| Race | X | X |
| Birth Quarter | X | X |
| Father Absent in 1900 | X | X |
| Mother Absent in 1900 | X | X |
| Father Foreign-Born | X | X |
| Mother Foreign-Born | X | X |
| Both Parents Foreign-Born | X | X |
| | | |
| Likelihood Ratio $\chi^2$ | 176.864 | 178.028 |
| Prob. > $\chi^2$ | 0.000 | 0.000 |
| Observations | 41,557 | 41,557 |
| Median Age at Death | 80.349 | 80.349 |

Note: Robust standard errors (clustered by county) of the coefficients underlying the hazard ratios in parentheses. Each model assumes a Cox proportional hazard, a Weibull duration dependence distribution (shape parameter *p*), and a gamma frailty distribution (mean one, variance θ).
* p<0.10 ** p<0.05 *** p<0.01

Table A2. Cause-Specific Survival Analysis Accounting for Competing Risks U.S.-Born Males Age 77½+, (Sub-Hazard Ratios).

| | Cause of Death | | | | | | |
|---|---|---|---|---|---|---|---|
| Characteristic in 1900 Census Manuscripts | Accidents | Heart Disease | Cancer | Chronic Lower Respiratory Disease | Cerebro-vascular Disease | Influenza and Pneumonia | Other |
| *a. White Only* | | | | | | | |
| Occup. Prestige of Hhld. Head | 1.002 | 0.998 | 1.002 | 0.998 | 1.000 | 0.994 | 1.005 |
| | (0.006) | (0.002) | (0.003) | (0.005) | (0.004) | (0.005) | (0.004) |
| Father Absent | 1.033 | 0.738 | 0.858 | 2.603* | 1.377 | 0.223* | 2.777** |
| | (0.969) | (0.149) | (0.277) | (1.352) | (0.488) | (0.181) | (1.309) |
| Mother Absent | 0.849 | 1.037 | 0.925 | 1.371 | 0.928 | 1.839 | 0.248 |
| | (0.939) | (0.258) | (0.407) | (0.787) | (0.422) | (1.253) | (0.255) |
| Hhld. Head Mos. Unemployed | 0.873 | 1.030 | 1.008 | 1.020 | 0.959 | 0.941 | 0.942 |
| | (0.091) | (0.019) | (0.032) | (0.056) | (0.038) | (0.058) | (0.051) |
| Home Owned & Mortgaged | 0.760 | 1.099 | 0.950 | 0.942 | 0.925 | 0.757* | 1.273* |
| | (0.171) | (0.066) | (0.091) | (0.184) | (0.098) | (0.124) | (0.179) |
| Home Rented | 0.741* | 1.112** | 0.919 | 1.055 | 0.871 | 0.768* | 1.239* |
| | (0.130) | (0.056) | (0.079) | (0.168) | (0.081) | (0.105) | (0.150) |
| (Home Rented) x (Mos.Unemp.) | 1.136 | 0.968 | 0.997 | 0.987 | 1.054 | 1.064 | 1.066 |
| | (0.135) | (0.023) | (0.035) | (0.067) | (0.051) | (0.080) | (0.067) |
| Family Size | 0.933* | 1.007 | 0.967** | 0.979 | 1.008 | 1.055** | 1.033 |
| | (0.038) | (0.010) | (0.016) | (0.030) | (0.019) | (0.027) | (0.022) |
| Family Child Mortality (0-100) | 1.006 | 1.002 | 1.000 | 0.999 | 0.997 | 0.996** | 1.000 |
| | (0.005) | (0.001) | (0.003) | (0.004) | (0.002) | (0.002) | (0.003) |
| Father Literate | 1.877 | 0.906 | 0.867 | 1.167 | 1.131 | 0.938 | 1.624 |
| | (1.132) | (0.113) | (0.174) | (0.428) | (0.268) | (0.352) | (0.560) |
| Mother Literate | 1.114 | 1.202 | 0.837 | 0.597* | 0.803 | 1.744 | 0.932 |
| | (0.515) | (0.137) | (0.150) | (0.165) | (0.157) | (0.613) | (0.235) |

(Continued)

Table A2 (Continued).

| Characteristic in 1900 Census Manuscripts | Cause of Death: Accidents | Heart Disease | Cancer | Chronic Lower Respiratory Disease | Cerebro-vascular Disease | Influenza and Pneumonia | Other |
|---|---|---|---|---|---|---|---|
| Pseudo Log-Likelihood | -1706.427 | -22949.326 | -8970.181 | -2319.972 | -6274.463 | -2973.264 | -4566.223 |
| Pseudo Log-Likelihood $\chi^2$ | 36.579 | 40.610 | 39.415 | 35.071 | 37.063 | 50.731 | 47.855 |
| Probability | 0.082 | 0.034 | 0.045 | 0.110 | 0.074 | 0.003 | 0.006 |
| Observations | 6,333 | 6,333 | 6,333 | 6,333 | 6,333 | 6,333 | 6,333 |
| | | | | | | | |
| *b. Non-Migrants Only* | | | | | | | |
| Occup. Prestige of Hhld. Head | 0.995 | 0.997 | 1.002 | 1.003 | 1.002 | 0.993 | 1.006 |
| | (0.008) | (0.002) | (0.004) | (0.007) | (0.005) | (0.006) | (0.006) |
| Father Absent | 3.047 | 0.740 | 0.706 | 3.554* | 1.037 | 0.165 | 6.753*** |
| | (4.050) | (0.184) | (0.299) | (2.620) | (0.526) | (0.185) | (4.896) |
| Mother Absent | 0.000*** | 0.972 | 1.086 | 1.394 | 0.955 | 2.060 | 0.000*** |
| | (0.000) | (0.299) | (0.571) | (1.014) | (0.505) | (1.711) | (0.000) |
| Hhld. Head Mos. Unemployed | 0.828 | 1.035 | 0.974 | 1.095 | 0.953 | 0.860 | 0.895 |
| | (0.150) | (0.024) | (0.050) | (0.068) | (0.047) | (0.088) | (0.078) |
| Home Owned & Mortgaged | 0.837 | 1.103 | 1.009 | 1.101 | 0.842 | 0.739 | 1.189 |
| | (0.240) | (0.086) | (0.128) | (0.284) | (0.118) | (0.177) | (0.227) |
| Home Rented | 0.663* | 1.093 | 0.957 | 1.131 | 0.804* | 0.881 | 1.231 |
| | (0.153) | (0.071) | (0.114) | (0.237) | (0.098) | (0.170) | (0.206) |
| (Home Rented) x (Mos.Unemp.) | 1.235 | 0.961 | 1.001 | 0.952 | 1.086 | 1.167 | 1.124 |
| | (0.239) | (0.029) | (0.051) | (0.074) | (0.063) | (0.139) | (0.107) |
| Family Size | 0.948 | 1.028** | 0.941*** | 1.006 | 1.014 | 1.003 | 1.017 |
| | (0.046) | (0.012) | (0.020) | (0.038) | (0.024) | (0.037) | (0.030) |

(Continued)

Table A2 (Continued).

| Characteristic in 1900 Census Manuscripts | Accidents | Heart Disease | Cancer | Chronic Lower Respiratory Disease | Cerebro-vascular Disease | Influenza and Pneumonia | Other |
|---|---|---|---|---|---|---|---|
| | Cause of Death | | | | | | |
| Family Child Mortality (0-100) | 1.009 | 1.002 | 1.003 | 0.997 | 0.994** | 0.993 | 0.999 |
| | (0.006) | (0.002) | (0.003) | (0.006) | (0.003) | (0.004) | (0.005) |
| Father Literate | 4.005 | 0.867 | 0.794 | 1.268 | 1.070 | 0.731 | 3.435** |
| | (4.440) | (0.139) | (0.211) | (0.669) | (0.338) | (0.356) | (1.955) |
| Mother Literate | 1.041 | 1.156 | 1.041 | 0.562 | 0.777 | 1.537 | 0.664 |
| | (0.655) | (0.172) | (0.279) | (0.222) | (0.202) | (0.657) | (0.191) |
| Pseudo Log-Likelihood | -948.161 | -13089.105 | -4947.927 | -1256.729 | -3491.889 | -1503.086 | -2432.147 |
| Pseudo Log-Likelihood $\chi^2$ | 7437.379 | 35.299 | 1198.682 | 1089.829 | 969.954 | 122.408 | 6558.265 |
| Probability | 0.000 | 0.132 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 |
| Observations | 3,767 | 3,767 | 3,767 | 3,767 | 3,767 | 3,767 | 3,767 |
| | | | | | | | |
| *c. Low SES Weighted* +20% | | | | | | | |
| Occup. Prestige of Hhld. Head | 0.999 | 0.999 | 1.003 | 0.996 | 1.000 | 0.993 | 1.004 |
| | (0.006) | (0.002) | (0.003) | (0.005) | (0.004) | (0.004) | (0.004) |
| Father Absent | 0.819 | 0.743 | 0.827 | 3.273** | 1.445 | 0.210* | 2.317* |
| | (0.706) | (0.147) | (0.270) | (1.789) | (0.505) | (0.167) | (1.025) |
| Mother Absent | 0.807 | 1.050 | 1.003 | 1.579 | 0.811 | 2.099 | 0.215 |
| | (0.883) | (0.270) | (0.445) | (1.036) | (0.400) | (1.281) | (0.222) |
| Hhld. Head Mos. Unemployed | 0.867 | 1.031 | 1.016 | 1.014 | 0.956 | 0.939 | 0.932 |
| | (0.101) | (0.020) | (0.031) | (0.057) | (0.038) | (0.057) | (0.053) |
| Home Owned & Mortgaged | 0.750 | 1.092 | 0.984 | 0.995 | 0.909 | 0.737* | 1.262* |
| | (0.175) | (0.068) | (0.096) | (0.199) | (0.098) | (0.121) | (0.176) |

(Continued)

Table A2 (Continued).

| Characteristic in 1900 Census Manuscripts | Cause of Death: Accidents | Heart Disease | Cancer | Chronic Lower Respiratory Disease | Cerebro-vascular Disease | Influenza and Pneumonia | Other |
|---|---|---|---|---|---|---|---|
| Home Rented | 0.735* | 1.118** | 0.923 | 1.096 | 0.853* | 0.754** | 1.211 |
| | (0.131) | (0.057) | (0.081) | (0.178) | (0.081) | (0.103) | (0.148) |
| (Home Rented) x (Mos.Unemp.) | 1.176 | 0.963 | 0.993 | 0.981 | 1.057 | 1.078 | 1.075 |
| | (0.150) | (0.023) | (0.035) | (0.067) | (0.052) | (0.079) | (0.070) |
| Family Size | 0.929* | 1.007 | 0.965** | 0.975 | 1.011 | 1.041 | 1.043** |
| | (0.038) | (0.010) | (0.017) | (0.031) | (0.019) | (0.028) | (0.022) |
| Family Child Mortality (0-100) | 1.006 | 1.001 | 1.001 | 1.000 | 0.997 | 0.996** | 1.000 |
| | (0.005) | (0.001) | (0.003) | (0.004) | (0.002) | (0.002) | (0.003) |
| Father Literate | 1.662 | 0.876 | 0.820 | 1.656 | 1.144 | 0.930 | 1.500 |
| | (0.799) | (0.102) | (0.156) | (0.680) | (0.257) | (0.319) | (0.449) |
| Mother Literate | 1.078 | 1.205* | 0.967 | 0.709 | 0.737 | 1.468 | 0.872 |
| | (0.470) | (0.129) | (0.183) | (0.210) | (0.138) | (0.457) | (0.210) |
| Pseudo Log-Likelihood | -1693.057 | -22773.543 | -8797.377 | -2311.021 | -6153.353 | -2978.877 | -4586.717 |
| Pseudo Log-Likelihood $\chi^2$ | 37.640 | 41.001 | 36.433 | 34.816 | 41.940 | 46.614 | 51.205 |
| Probability | 0.084 | 0.041 | 0.106 | 0.144 | 0.033 | 0.011 | 0.003 |
| Observations | 6,284 | 6,284 | 6,284 | 6,284 | 6,284 | 6,284 | 6,284 |

Notes: Cause-specific sub-hazard ratios (Fine and Gray, 1999) are shown along with the robust standard errors that correspond to the coefficients underlying the sub-hazard ratios. Each model includes controls for 1900 Region of Residence, 1900 Urban Residence, Race, Birth Quarter, Father Foreign-Born, Mother Foreign-Born, Both Parents Foreign-Born, Pct. Children in Mfg, Pct. Adult Males in Mfg, Pct. Adult Females in Mfg., Avg. Persons/Dwelling, and Pct. Homes Rented/Mortgaged.
* p<0.10, ** p<0.05, *** p<0.01

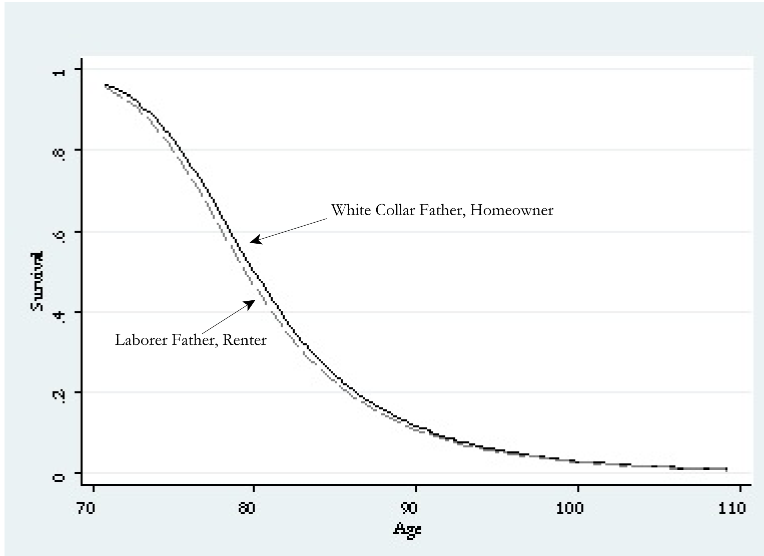


**Figure 1**. Predicted Survival After Age 70 By Father's Occupation and Home Ownership Status, Using Coefficients in Table 2, Column 4.

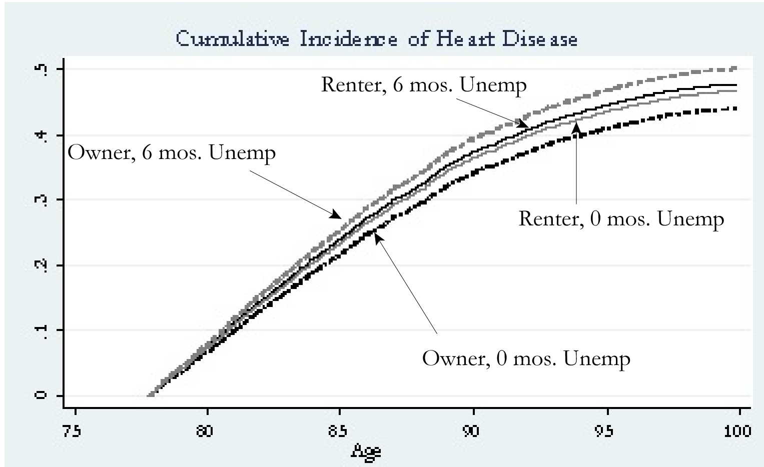


**Figure 2**. Cumulative Incidence Function for Heart Disease by Home Ownership Status and Months of Household Head Unemployment.