Intra-Urban Health Disparities: Survival in the Wards of 19th-Century American Cities

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Abstract

Survival rates were low in large 19th-century American cities. We ask whether this was attributable to a few "bad" wards or whether urban wards were uniformly bad. The paper employs two datasets. The Union Army database has been augmented with veterans who enlisted in and/or resided in Boston, Chicago, New York City (including Brooklyn), and Philadelphia. Additionally, the Historical Urban Ecology (HUE) database has been created containing ward-level data on health indicators, the expansion of public infrastructure, and socio-economic indicators. These data are used to construct a "Ward Development Index" which identifies "good" versus "bad" wards and is part of hazard ratio regressions. Preliminary results suggest there is little difference between good and bad wards in 1860. By 1900, however, the urban mortality penalty remains in bad wards and is much reduced in good wards. Understanding why this difference emerged is vital to understanding the urban mortality transition.

Most economic historians are aware that in the late nineteenth century urban areas were far less healthy places to live than rural areas, but far fewer are aware that urban health conditions varied more within America's largest cities than between them. Within the cities, measured death and reportable disease rates by wards—the cities' most common political division—demonstrated wide disparities between the "best" and "worst" wards with respect to a variety of indicators. For example, in 1890 the average gap in the infant death rate between wards of cities was 317 per 1,000, while the average gap between cities was 165 infant deaths per 1,000.

That the gap between cities is smaller is a result of averaging over the large disparities within the wards of each city. Available evidence indicates that these severe intra-urban disparities were reduced by 84 percent by 1950 and continued to decline during the balance of the twentieth century. Available evidence also indicates that disparities in environmental conditions across wards in eight large cities were also severe, with the worst wards in Baltimore in 1890 averaging 223 infant deaths per 1,000 and the best wards averaging 59 infant deaths per 1,000. The infant death rate demonstrates this same pattern prior to the formal reporting of mortality statistics, with the best average ward mortality rates in Boston at 98 deaths per 1,000 between 1837 and 1848, and the worst wards experiencing 265 deaths per 1,000. In this paper, we aim to measure the impact of environmental disparities (e.g., mortality, exposure to infectious disease, and public infrastructure) on the experience of Union Army veterans who at various times in their lives lived in at least one of six large cities. During the adult years of these veterans, urban areas grew dramatically and urban environmental and infrastructural improved markedly, but these improvements occurred to different degrees in different areas of each city. The improvement in urban environments occurred relatively quickly—84 percent of the difference in the infant mortality rate (IMR) between urban wards in 1900 was gone by 1950.[1]

1. The Urban Mortality Penalty and Transition

Many of the socioeconomic and environmental factors thought to affect the life span are spatially correlated; people living in the same area tended to be exposed to the same health factors. Numerous studies have found that morbidity and mortality levels are indeed highly associated with spatial characteristics; the urban mortality penalty of the late nineteenth-century

[1] This suggests that genetic or evolutionary theories are inadequate to explain the increases in health and longevity witnessed over the last century (Costa 2005). Thus, attention has been focused on the influence of early socioeconomic and epidemiological environments on later-life health and longevity (Fogel and Costa 1997).

United States is a prime example.[2] These spatial patterns are the result of a wide variety of mechanisms, and in this paper we focus on the causes of highly localized differences in morbidity and mortality at the ward level.

The mechanisms by which geographic variation in morbidity and mortality occurs are a subject of some dispute. If local differences in health outcomes are primarily due to differences in such things as cancer rates, injuries, or diseases of the circulatory and respiratory systems, geographic variation may be a result of socioeconomic factors and social risk factors, such as smoking prevalence and diet.[3] In this case, inherently geographic factors such as disease environments and local pollution are less relevant than behavioral factors. However, city size, geographic region, and the risk of water-borne diseases had large and important effects on mortality in the late nineteenth and early twentieth-century United States. These may have outweighed individual-level factors such as occupation and income.[4] Diseases such as malaria are strongly dependent on local environmental factors.[5] However, improvements in infectious disease environments and reductions in local pollutants as economic development occurs reduced the importance of purely geographic factors.

As a result of such changes, people began to live longer, healthier lives. There are several hypotheses about the causes of improvements in longevity and chronic conditions among the elderly. Some studies emphasize the reduction of malnutrition and the increased standard of living as factors that have driven this change.[6] There are studies arguing for the importance of personal health practices such as washing hands and boiling milk.[7] A number of

[2] See Cain and Hong (2009). Generally, rates of death and disease vary by region (Congdon 2007, Barford and Dorling 2007), type of environment (e.g., urban versus rural) (Preston and Haines 1991), city (Cutler and Miller 2005), and, of particular interest to this project, immediate surroundings (Altmayer *et al.* 2003, Hinman *et al.* 2006, Tiwari *et al.* 2006, Shah *et al.* 2006).
[3] Altmayer *et al.* (2003).
[4] Preston and Haines (1991), Cutler and Miller (2005).
[5] Hong (2007, 2011), Keating *et al.* (2004), Hakre *et al.* (2004), Humphreys (2001).
[6] Fogel (1994), McKeown (1976).
[7] Deaton and Paxson (2001), Ewbank and Preston (1990). Mokyr and Stein (1996).

studies have emphasized public health reforms such as water filtration, milk pasteurization, sanitation, and vaccination.[8] The role of medical care has also been considered as an important factor in mortality reduction, especially after the mid-twentieth century.[9] Fundamentally, all these factors have played a key role in reducing deaths from infectious diseases (e.g., typhoid, pneumonia, tuberculosis, and diarrhea) that were ubiquitous during the late nineteenth century and the early years of the twentieth century, including.[10] Numerous studies have provided convincing evidence highlighting the negative effects of exposure to many infectious diseases in utero, in infancy and in childhood on the rates of mortality, chronic disease prevalence, and disabilities in middle and older ages.[11] Studies based upon the Union Army sample that is used in this study have shown that infectious diseases in adolescence and early adulthood were associated with heart and respiratory problems after age 50 and that the local disease environment prior to enlistment affected health while in service.[12]

In addition to studies of exposure to infectious diseases in early life, there has been an explosive growth in research to find a significant link between mortality and morbidity after age 65 and health status earlier in the life cycle. Some studies focus on risk factors at midlife.[13] Still other studies have argued that a decline in number of offspring may be related to decreases in morbidity and mortality during early life and old age through more extensive investment in each of the fewer children.[14]

[8] Barker (1998), Fogel (2000, 2004a), Manton, Stallard, and Corder (1997), van Poppel and van der Heijden (1997).

[9] Cutler and Meara (2004), McDermott (1978).

[10] CDC (1999b).

[11] Barker (1992, 1998, 2003), Bengtsson and Lindstrom (2003), Blackwell, Hayward, and Crimmins (2001), Buck and Simpson (1982), Finch and Crimmins (2004), Jones (1956), Shaheen *et al.* (1994)

[12] Costa (2000) and Lee (2003), respectively.

[13] Reed *et al.* (1998) and Valkonen, Sihvonen and Lahelma (1997).

[14] Ali *et al.* (2001), Becker (1993), and Marmot (2004). Various life tables provide strong correlations between mortality rates at different ages (Coale and Demeny, 1983) and between infant mortality and longevity (National Central Bureau of Statistics, 1996).

The industrialization and urbanization that accompanied post-bellum economic growth were the major causes of socioeconomic disparities in health. As urban populations grew faster than accommodation could be built, rural and international migrants crowded together, and poor sanitary conditions increased exposure to infectious diseases.[15] Infant mortality rates and sanitary conditions varied significantly among wards of large cities.[16] Gretchen Condran and Rose Cheney report that the 37 wards in Philadelphia in 1895 had an average infant mortality rate of about 158 per 1,000, with a standard deviation of 70 per 1,000.[17] Using data on deaths by cause, Condran and Eileen Crimmins compare death rates across Philadelphia's wards for diarrheal diseases in 1880, 1910, 1920, and 1930, and for tuberculosis and pneumonia in 1880 and 1930.[18] Differences in material welfare by socioeconomic class increased or remained large during the nineteenth century.[19] And these inequalities in mortality rates were related to socioeconomic, residential, and racial status.[20]

It seems reasonable to assume there was a social gradient in health a century ago; they are found even in today's wealthiest countries. Income inequality may affect health through such unobservable psycho-social conditions such as hopelessness, social networks, relative hierarchy, and family conflict. Furthermore, those lower in the social hierarchy might be more exposed to violence, homicide, and a lack of social cohesion. In short, they may suffer higher levels of (perhaps even chronic levels of) stress. Workplace characteristics and types of jobs have been identified as factors in the onset of chronic diseases and disabilities.[21]

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[15] Mortality rates in urban areas, especially infant mortality rates, were much higher than those of rural areas (Shattuck 1850 and US Census Bureau 1896).

[16] Citizens' Association of New York (1866), Duffy (1968), Leavitt (1996), Rosner (1995), US Census Bureau (1896) .

[17] Condran and Cheney (1982).

[18] Condran and Crimmins (1980).

[19] Williams (1976).

[20] See Preston and Haines (1991).

[21] Vaananen *et al.* (2003).

Social networks enable individuals to find employment through job referrals.[22] Individuals might also simply prefer to live near people of the same ethnicity because of preferences, language issues, or even the availability of ethnic consumer goods in the neighborhood. Social networks could be formed along ethnic lines or by men from the same military unit during the Civil War. Dora Costa and Matthew Kahn showed that men were statistically significantly more likely to migrate to states where other men in their companies lived.[23] If the social network influences the choice of location, the expected quality of a veteran's environment will depend on the size and the distribution of the veteran's social network. The social network and its evolution over time then become candidate instruments for the sequence of environments faced by a veteran. For each veteran, two types of social networks will be constructed. The first is based on the distribution (over locations) at a particular point in time of people born in the same location as a veteran. The idea is that people born in close proximity are more likely to know each other and hence be part of the same social network. The second type of social network is based on the distribution (over locations) of soldiers who served in the same company as a veteran. The motivation here is that people who served together share common experiences and are thus more likely to form a friendship network that persists over time and space.

Individuals will choose where to live on the basis of several desired characteristics, such as distance to the central business district or to their place of work, availability of transport, the presence of friends or other social networks, rents, and the presence of various amenities ranging from specific types of stores to parks to ecological characteristics. Characteristics other than ecological characteristics can be used as the basis for an initial selection equation, provided that they are uncorrelated with health. We can thus allow for essential heterogeneity

[22] Montgomery (1991), Carrington *et al.* (1996), Munshi (2003).
[23] Costa and Kahn (2006).

in health types provided that unhealthy wards have offsetting characteristics (e.g., low land prices) valued by some individuals.

Numerous studies emphasize the introduction of modern methods of sanitation and other public health programs. Cleaning up the water supply, improving the distribution of basic nutrients, draining swamps and otherwise disrupting vectors of disease, improving waste disposal, and vaccinations can be achieved quickly and cheaply. Many studies have investigated the link between historical public health interventions and declines in mortality rates and infectious disease deaths at the turn of the twentieth century. The introduction of water filtration technology and urban sanitation infrastructures played a key role in the decline of waterborne diseases, particularly typhoid.[24] However, early water-piping systems in the nineteenth century were not very effective in preventing water-related infectious diseases.[25] While many cities had built water systems by the late nineteenth century, few of them had simultaneously constructed sewer systems to remove wastewater. Chicago in the 1850s was the first American city to install a comprehensive sewer system. Most cities simply allowed waste to be dumped into the streets to find its way to a water course, perhaps the one that supplied the city's water. This situation was worsened by the widespread adoption of water-carriage technologies such as the water closet. Wastewater carriage could cause health hazards such as contamination of the subsoil through leakage, pollution of waterways with threats to drinking-water supplies, and the generation of disease-bearing sewer gas. In addition, the failure of upstream cities to dispose of their sewage effectively negatively affected water supplies downstream.[26]

[24] Blake (1956), Cain and Rotella (2001), Cutler and Miller (2005), Ferrie and Troesken (2008), McCarthy (1987), Troesken (2004).
[25] Cutler and Miller (2005).
[26] Cain (1978), Cutler and Miller (2005), Tarr *et al.* (1984).

2, Historical Urban Ecological (HUE) data

The Historical Urban Ecological (HUE) data set contains ward-level tabular and spatial data from 1830 to 1930 for the cities of Baltimore, Boston, Brooklyn, Chicago, Cincinnati, New York (Manhattan), and Philadelphia. This new resource provides new opportunities for analyzing and visualizing changing socioeconomic conditions and health environments in the largest US cities during the urban mortality transition. It contains tabular data related to disease, mortality, and population at the ward level as well as detailed geographic information system (GIS) reconstructions of the historical ward boundaries and street network for each study city. In general, previous literature has been stymied by a lack of data on conditions within cities. Exceptions include Craddock's (2000) examination of typhoid in San Francisco that showed typhoid rates were highest in the immigrant Chinatown neighborhood and Condran and Cheney (1982) who used the gradual rollout of water filtration by neighborhood to examine the effect of filtration on reducing typhoid fever.[27]

The tabular data primarily consist of health and mortality statistics—death and diseases at the ward level—published in annual reports from either the local department of health or in the city annual reports.[28] The statistics published by each city generally expanded over time. Early reports tend to be terse, collecting only key vital statistics or statistics idiosyncratic to the city.[29] Published statistics expanded widely during the latter half of the 19th century, responding in part to the desire of cities to track and prevent disease outbreaks. As methods of medical diagnosis improved, cities published more data on cause-specific case and mortality rates. City health departments were in close contact with each other, which resulted in the rapid dissemination of advances in medicine and the equally rapid introduction of new statistics into

[27] Craddock (2000), and Condran and Cheney (1982).
[28] A full list of the sources used is infeasible here, but is available on the CPE website.
[29] For example, the earliest data for Boston lists only the number of polling stations by ward in 1800, although by 1810 they began publishing population by ward.

annual health reports.[30] Regarding frequency, although many statistics are available at the monthly or even weekly level, this release includes only data at the yearly level. Quarterly, monthly, and weekly data are planned for a future release. The annual reports of the municipal institutions often included a variety of additional useful data, which are also included in the tabular data. Table 1 shows the earliest available ward-level data for several broad categories.

[Table 1 about here]

The HUE database also contributes to a growing body of historical GIS (HGIS) data.[31] The core of the HUE GIS data is composed of reconstructed historical street centerline shapefiles for each of the sample cities contemporary to 1930. The streets paths within our study cities were generally static from their inception until the installation of the interstate highway system and the emergence of urban renewal projects, which followed the study period. The street reconstruction process drew from a variety of historical resources including early aerial surveys, topographic surveys by the US Geological Survey, and fire insurance maps by several authors. The data were tested against surviving landmark control points and met a five-meter accuracy tolerance. In other words, the HUE GIS data can place a historical feature to within five meters of its original location. Figure 1 shows the use of a historical fire insurance map to accurately reconstruct streets in the Corlear's Hook district of New York City. The street

[30] For example, the 1874 New York Report of the Board of Health includes a discussion (p.26-27) on whether a recent cholera infection in New Orleans was Asiatic cholera or its more benign cousin cholera-morbus. A doctor was dispatched to determine the nature of this strain, and in his subsequent report he describes conferring with physicians in Cincinnati, OH, and Nashville and Murfreesboro, TN, on its pathology and how it should be appropriately classified (p.415-418).

[31] Similar projects have created GIS resources covering a variety of historical periods and geographic scales. National HGIS projects in the United States such as the National Historical GIS Project (https://www.nhgis.org) and the Atlas of Historical County Boundaries (http://publications.newberry.org/ahcbp/index.html) as well as a number of international efforts (Gregory *et al.* 2002, De Moor and Wiedemann 2001), enable researchers to explore census and other aggregated statistical data spatially across time. The HUE data set builds on previous work conducted at the within-city level, enabling visualization and analysis across longer time intervals. These projects include the Urban Transition Historical GIS Project (Logan *et al*. 2011) which developed enumeration district shapefiles for 39 US cities for use with the Minnesota Population Center's IPUMS (Integrated Public Use Microdata Series) 1880 Census. Related studies have also been undertaken in Boston (http://dca.lib.tufts.edu/features/bostonstreets/index.html), Newport, KY and Alexandra, VA (DeBats and Lethbridge, 2005) and Montreal (Gilliland and Olson, 2003; Gilliland *et al.*, 2011).

files were then used as a guide to reconstruct the ward boundaries for each ward system employed from 1830 through 1930.[32] The ward boundary histories, spanning a century of vigorous urban development, will allow researchers to perform multi-dimensional analyses over longer intervals and finer spatial scales than previously possible.

[Figure 1 about here]

The HUE GIS data also provide a framework for the construction and analysis of surviving data at multiple levels of spatial granularity: inter-urban, intra-urban (at the ward level), block-, and even address-level. This analysis utilizes the GIS data to locate, or geocode, the historical residential addresses of the Union Army Veterans following the Civil War. We linked the veterans to the tabular ecological data by joining the geocoded residential locations to their corresponding ward. Figure 2 shows the location of all the addresses at which a member of Union Army Veterans Sample ever resided in our study cities over a layer containing the 1900 ward boundaries. Each point represents the residential location of a veteran at some point in their lives. Future research will be able to incorporate additional characteristics with possible health consequences including the location of factories, historical transit lines, and access to water and sewer pipes, all of which could influence the local health environment.

The HUE dataset also provides a versatile framework for future researchers to reconstruct and analyze additional layers of historical data. Many administrative boundaries, including districts for police, fire departments, parishes, precincts, and enumeration districts, follow the paths of historical streets. They also allow more accurate geo-referencing of historical maps, which facilitates the extraction of data from maps. The Center for Population Economics expects that all of the tabular and GIS data will be publicly available within a year.

[Figure 2 about here]

[32] Shapefiles for each city are available for download from the Center for Population Economics website (http://www.cpe.uchicago.edu)

3. Living in Good and Bad-Quality Wards and Longevity

We next turn to investigate how individuals' longevity was affected by their residence at various points during their lifetimes, using the Union Army veterans' lifetime records. In general, we categorize their residences in terms of population (i.e. urban and rural) and classify it as having been in a good and bad-quality ward according to a ward development index if it ws in one of the four large cities we are studying. To better understand that index, we first take an overview of intra-urban disparities in health and mortality, then look closely into conditions in our four sample cities.

3.1 Data

The original Union Army veterans' sample consists of 39,517 veterans who are linked to various historical records such as military service records, carded medical records, regimental history, surgeon's certificates, pension records, and US federal census records. However, those living in large cities were under-represented in the original sample. We lacked sufficient information with which we could examine the effect of urban ecological conditions on later life (health and longevity) conditions, i.e. intra-urban disparities. The main reason why this is true is that urban veterans died too young to be in the pension records, which provide the details later in the lives of these veterans. To rectify the situation, the Center has recently collected additional urban veterans who were enlisted in six large cities: Baltimore, Boston, Chicago, Cincinnati, New York City (Brooklyn), and Philadelphia. So far the records for 10,558 new urban veterans have been completed from four cities: Boston (1,692), Chicago (1,611), New York City (4,287), and Philadelphia (2,968). Work on Baltimore and Cincinnati continues. The present study is based on both original samples and new urban samples from the four completed cities.

3.2 Variables

The key variable in this section is the type of residence at three points of lifetime: at birth, in 1860, and in 1900. Because veterans were born around 1840, their residence in 1860 and 1900 represents life conditions at age 20 and 60, respectively, on average. For each of the three residences, we obtained information on the state, county, town or city, and ward (if the residence was in one of the four cities) from various sources such as military records, 1860 and 1900 census records, and pension records.

We classified birthplace into three groups: US urban areas, US rural areas, and foreign countries, where urban areas are defined as one of the top 100 cities in 1860 (whose populations were more than 9,550). Information at the ward level is available if veterans lived in the four large cities in 1860 and 1900, and we can examine within-city disparities for those large cities in both years. In particular, we classify residence in 1860 and 1900 into four groups: good and bad-quality wards (as measured by the ward development index discussed below) , urban areas other than the four cities (as defined above), and rural areas.

Our aim is to estimate how veterans' longevity was affected by their type of residence at these three points in their lives. Consequently, those whose year of birth or death is unknown are dropped from the regression analysis. In addition to the type of residence, we consider various measures of lifetime experiences and conditions as determinants of longevity. We control for wartime experiences using enlistment year (a measure of the length of wartime exposure), initial rank (socioeconomic status in early life), prisoner-of-war experience (wartime stress), and number of wartime diseases and wounds (wartime health). Socioeconomic status in early life is measured by total household wealth and occupation found in the 1860 census records. Later-life socioeconomic status is measured by literacy, occupation, marital status, home ownership and position in household found in the 1900 census records.

3.3 Ward Development Index

As noted, we obtained information on wards in the four large cities (Boston, Chicago, New York City, and Philadelphia) for 1860 and 1900. To examine intra-urban health disparities, those wards are classified into good or bad-quality wards according to a ward development index. This index, which is a similar concept to the UN human development index used to rank countries by level of human development, is intended to measure the overall quality of ward ecological condition or ward-level living standards.

Following the formula of the UN human development index, we consider three aspects of ward quality: life expectancy, education, and income. To measure ward-level life expectancy, we searched for a crude mortality rate from each city's annual health reports. We employ the adult literacy rate and school attendance rate to quantify the educational level, which are calculated from 1860 and 1900 IPUMS. Also using the IUPMS dataset, we calculated a ward-level average occupational income score as a proxy of the income level.

To estimate the ward development index, we first transformed each variable into a unit-free index between 0 and 1 for 1860 and 1900, using the following equation.

$$X_{it} = \frac{X_{it} - \min\ X_{it}}{X_{it} - \max\ X_{it}}$$

i: crude morality rate, adult literacy rate, school attendance rate, occupational income score

t: 1860 and 1900

where min(X_{it}) and max(X_{it}) are the minimum and maximum value of each variable X_i within all the available wards in 1860 or 1900. Then the ward development index is estimated by calculating a weighted average of four variables, as follows.

$$WDI_t = X_{cdr,t} + \tfrac{2}{3}X_{lit,t} + \tfrac{1}{3}X_{sch,t} + X_{income,t} \div 3, \text{ where t = 1860 or 1900}$$

That is, the education component is two-thirds contributed by the adult literacy rate (*lit*) and one-third by the school attendance rate (*sch*). Life expectancy, education and income are uniformly weighted.

There were 68 wards in the four large cities in 1860. The 1860 ward development index ranged between 0.35 and 0.94; its mean and standard deviation are 0.72 and 0.12, respectively. There were 147 wards in those cities in 1900. The index is between 0.30 and 0.91; its mean and standard deviation are 0.65 and 0.12, respectively. These statistics say that there was a substantial disparity in living standards within cities throughout the nineteenth century.

Figure 3 shows the distribution of good (denoted by light gray) and bad-quality wards (denoted by dark gray) in 1860 and 1900 in terms of the ward development index estimated above. We use each year's median value of the index as the cut-off point. Three features are found from the maps. First, as the increase in the number of wards suggest, the city boundaries expanded between 1860 and 1900, especially in Boston and Chicago. Second, good and bad-quality wards were somehow equally distributed across the four large cities. Third, a frequent change from good to bad-quality wards between 1860 and 1900 or vice versa is observed.

[Figure 3 about here]

3.4 Effect of Lifetime Residence on Longevity

We estimate the effect of lifetime residence on longevity in two ways. Our first approach is to use veterans who survived the Civil War and to analyze whether their age at death was affected by type of places where they lived before the war, i.e. birthplace and residence in 1860. The second approach is to use veterans who survived up to 1900 and to examine whether the likelihood of living longer depended on the quality of the later residence as well, i.e. that in 1900.

Table 2 presents average age at death and average year of birth by type of places (defined in section 3.2). In terms of the average age at death, it is clearly observed that those who were born or lived in rural areas in 1860 and 1900 lived longer than those who were born or lived in urban areas or were born in foreign countries. This is true whether one looks at those who survived the war or those who survived up to 1900. Looking into intra-urban disparities, there is little difference in longevity between good and bad-quality wards in 1860. However, it is found that veterans who resided in good-quality wards in 1900 lived longer than those in bad-quality wards. Considering the average year of birth, it looks like that veterans in good-quality wards of large cities in 1900 lived longer than those in small cities.

[Table 2 about here]

To estimate the statistical significance of a hierarchy in the urban mortality penalty by type of place found in Table 3, we use a Cox proportional hazard model that specifies the hazard for veteran *i* as $\lambda_{it}\ t\ = \lambda_{0t}\ t\ + \exp(X_i\beta)$, where $\lambda_{0t}\ t$ is the hazard in a group at time *t*. We use two subsamples: one group is veterans who survived the Civil War and the other group is those who survived up to 1900. Thus time is measured as years since 1865 for the former and years since 1900 for the latter. X_i is an $n \times p$ matrix that includes *p* components, which are listed in section 3.2, associated with each of *n* veterans. The β's are the estimated log hazard ratios, the multiplier by which the hazard is increased or decreased with a unit change in the variable X as compared to the reference group. The estimated hazard ratio is based on a year-by-year evaluation of the data.

Table 3 reports the results for veterans who survived the Civil War, where the key variables are the specified type of area at birth and in 1860. Models (1) and (2) estimate the effect of birthplace and residence in 1860, separately; models (3)-(6) combine both effects, extending controls such as wartime experiences and socioeconomic status in 1860. In particular, model (6) uses a strict specification by including state-of-birth and 1860-state fixed effects to control for local characteristics.

[Table 3 about here]

The results have two main features. First, the estimated hazard ratios suggest that those who were born or spent their early years in US rural areas lived significantly longer. The benefit is much more substantial when they lived in rural areas in 1860. As a rough rule of thumb, a 0.1 difference in a hazard ratio is equal to approximately 1.2 years of life. Thus in terms of the hazard ratio in model (6), the rural veterans are estimated to have lived 2.6 more years after 1865 than veterans from good-wards in the four large cities. Second, the hazard ratio increases from rural areas to small cities, bad wards, and good wards. However, the disparity between good and bad wards is statistically insignificant.

Using veterans who survived up to 1900, Table 4 estimates the hazard ratios after 1900 by the type of area in which the veteran lived in 1900, as well as by those in early life. The main finding in Table 4 is that there exists a significant difference in hazard ratios between good and bad wards in 1900 even under the strict controls of the three types of fixed effects in model (6). On the basis of the hazard ratio in model (6), it is suggested that those from bad-quality wards in the four large cities had about 2.3-years shorter lives after 1900 than those from good-quality wards, on average. In fact, the hazard ratio of good-ward veterans in 1900 is statistically indistinguishable from those of small cities or rural areas. On the other hand, the lack of any disparity between good and bad wards in 1860 remains. This implies that the mortality penalty for urban veterans who lived under poor ecological conditions (i.e. bad-quality wards) was more substantial in the late nineteenth century than it was in the mid-nineteenth century.

[Table 4 about here]

4. Intra-Urban Disparities

We finally investigate the disparity more closely by looking into the four large cities: Boston, Chicago, New York City and Philadelphia. The aim of this section is to reveal what aspects of urban conditions caused the mortality penalty.

4.1 Longevity by Ward Development Index

Between the original Union Army veterans' sample and the new additional urban sample, we found 610 veterans who lived in the four cities in 1860 and 683 veterans who lived there in 1900. Figure 4 shows the relationship between these veterans' age at death and the ward development index for 1860 and 1900; the figures in upper panel are scatter plots of within-ward average age at death against the index, while those in the lower panel display wards aggregated each year into 10 groups representing each decile of a rank ordering of the index. In general, the 1860 index seems to be irrelevant to urban veterans' longevity; the positive relationship between the 1900 index and longevity looks substantial, suggesting that the difference in longevity between best and worst-quality ward groups was about 3 years. The figures well support the Cox regression results in the previous section.

[Figure 4 about here]

In Table 5, we run two types of regressions (Cox proportional-hazard model and OLS) for two sub-samples (war survivors and 1900 survivors) to investigate the significance and magnitude of the intra-urban disparities. The dependent variable for the OLS regressions is age at death; instead of dummies of good or bad wards, we use the ward development index as a control variable.

The key result is that the quality of wards measured by the ward development index is highly related with the longevity of veterans who lived in the four large cities. But this is found only for 1900, not for 1860, which is consistent with the findings in Tables 3 and 4. The effect of ward quality in 1900 is significant across the specifications. For 147 wards in 1900, the ward development index ranges between 0.30 and 0.91. Thus, the coefficient in model (5) and panel C suggests that, for veterans who survived to 1900, there was about 2 years difference in longevity between those who lived in the best and worst wards in 1900.

[Table 5 about here]

4.2 Various Aspects of Urban Ecological Conditions in 1900

On the basis of the results in Table 5, we concentrate on the intra-urban disparities in 1900. So far we have used ward development index as a measure of ward quality, this section considers various aspects of urban conditions. First, the three components of the index (life expectancy, education and income) will be examined respectively. This will provide evidence that shows which component was more significant in affecting urban population's health and mortality. Second, we also employ various ward-level ecological and socioeconomic variables, replacing the ward development index. Sanitary conditions are measured by the percentage of streets with water pipes, and we include the ward's population density. Geographical features are calculated with within-ward mean and variance of elevation; child survival rate and child mortality rate will reflect the level of epidemiological and disease environment. Finally, to measure ward-level socioeconomic status, we use the percentages of homeowners and married adults, and the male labor force participation rate. Those variables are obtained from Historical Urban Ecology Dataset discussed in section 2.

Table 6 reports the estimated coefficients of the above variables. We use three different regression models: Cox proportional hazard regression in model (1), OLS in model (2), and logit regression in models (3) and (4). In the logit regressions, the dependent variables are the dummies that indicate whether they survived up to 1910 or 1920; the reported coefficient is the marginal effect.

[Table 6 about here]

The results are summarized as follow. First, each individual component of the ward development index has a significant impact on longevity after 1900, but none are strongly correlated with longevity. This implies that urban conditions depended on various factors, not just health, but education and economic status as well. Second, population density and sanitary conditions are found to be significant but only in the logit regressions; a higher population density in 1900 lowered longevity among urban veterans. Third, the disease environment measured by the child survival rate and child mortality rate is estimated to have a very substantial role in determining veterans' longevity. Finally, socioeconomic status such as the percentages of homeowners and married adults and the male labor force participation rate does not have a significant impact on longevity.

5. Summary and Conclusion

This is a preliminary (and incomplete, for that matter) look at what a large amount of new data can tell us about the urban mortality penalty that was present in the nineteenth century and disappeared in the twentieth. The focus here is on four cities, but it will eventually be six. We have only begun to scratch the surface of the data that is (or will become part of) HUE. Nevertheless, some things stand out even at this stage.

The ward development index created to identify good versus bad wards performs well. in this paper. We have used the median value to divide wards into good and bad, but there are clearly many other ways to approach that, and, in future work, we will be examining the sensitivity of the index in several ways. We plan to experiment with the individual series that comprise the index as well as alternative definitions of good and bad (e.g., top vs. bottom 40% of wards).

There is no statistical difference with respect to longevity in 1860 between good and bad wards, but there is in 1900. Between 1860 and 1900, the bad wards remain bad, but the good wards change to resemble healthier places. This is consistent with the pioneering work of Ted Meeker and others who argued that the big change began in the 1880s.[33] We will try to refine our statistical approach to see if we can better date the transition. We find some evidence that such things as urban density and sanitation matter in the logit regressions, but not in the other specifications. There is clear evidence that the improvement in the disease environment is important.

The evidence points toward the acceptance of the germ theory as being of crucial importance. Knowledge of the transmission of disease led to improvements that benefited those with the knowledge more than others. The three main explanations for the decline of the urban mortality penalty (economic improvement, sanitary improvement, and declining density) all find support in this work. With time and the inclusion of additional data, we hope to be able to say more. However, given that the three explanations are interrelated, it is less a question of picking one than understanding the mechanism. A focus on the germ theory is a step in that direction.

[33] Meeker (1971 and 1974), Condran and Crimmins (1978), and Haines (2001).

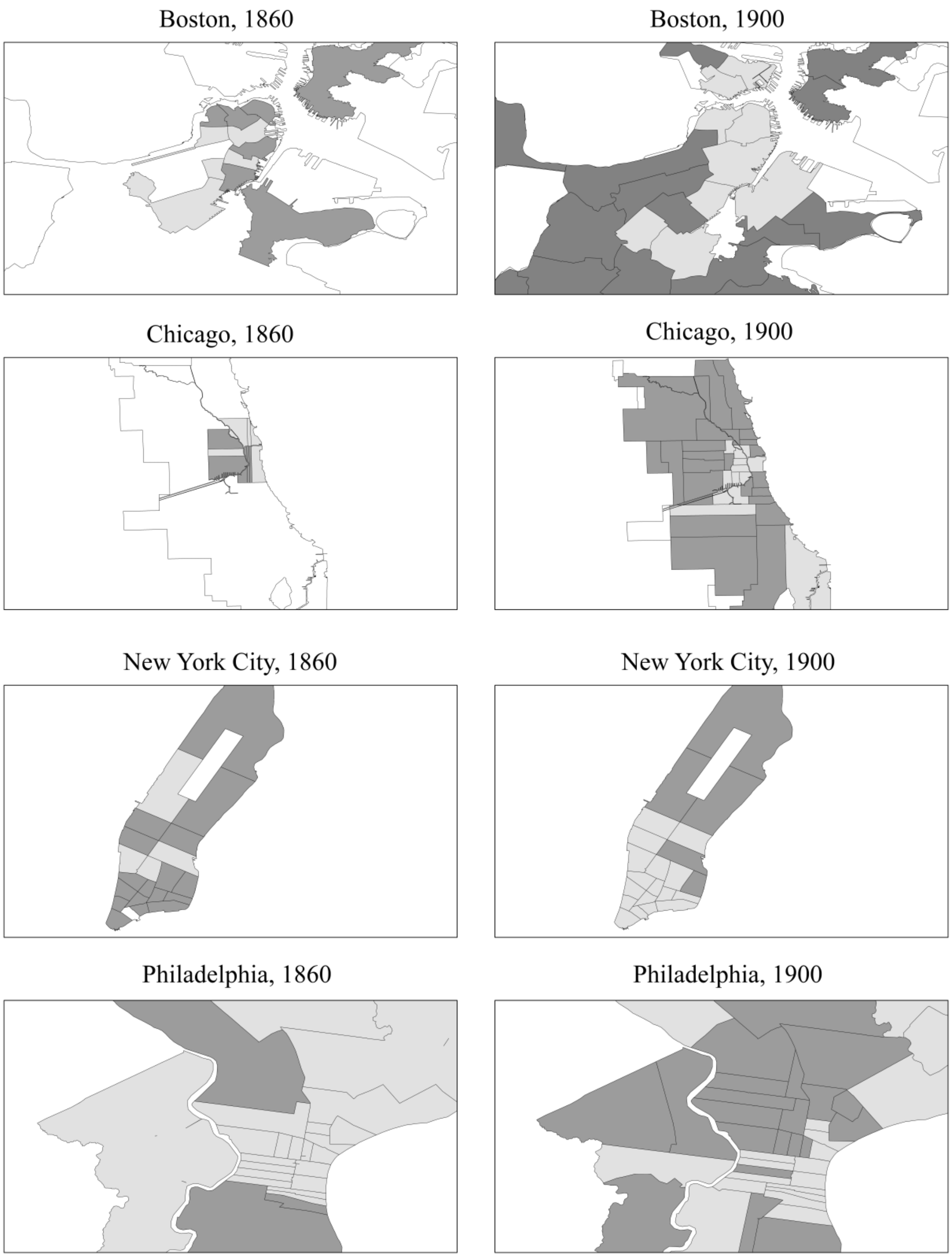
Figure 1a: Contemporary New York City Aerial Photo and Contemporary Streets

Figure 1b:1924 New York City Aerial Photo and Contemporary Streets

Figure 1c: 1924 New York City Aerial Photo with HUE Historical Street Reconstruction

Figure 2: All Union Army Veteran Residential Locations Overlaid Upon 1900 Ward Boundaries

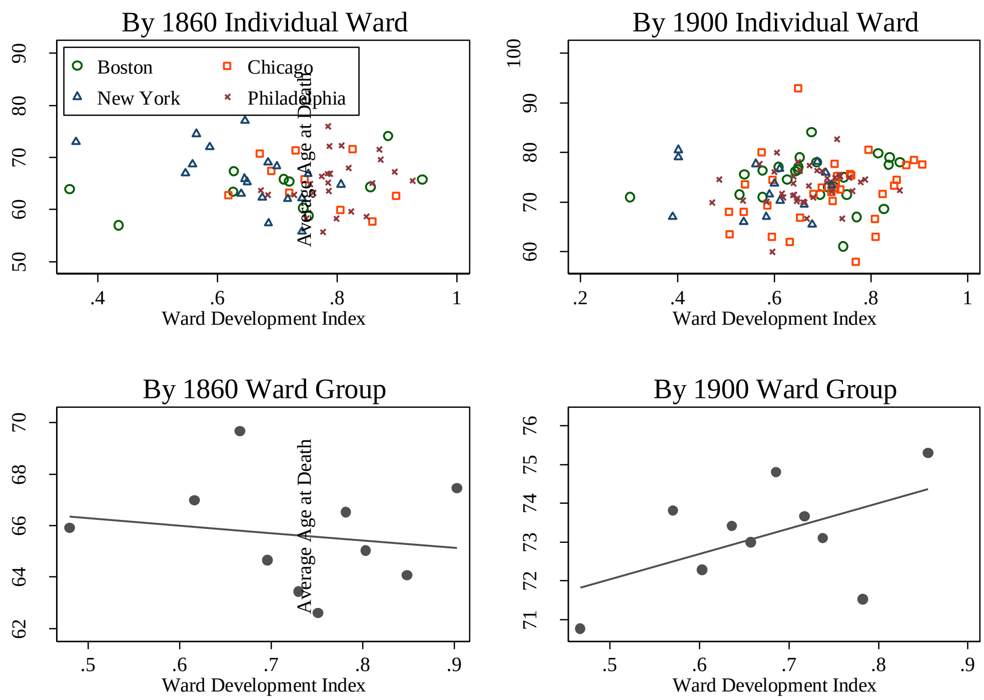
Figure 3. Good and Bad-Quality Wards of Boston, Chicago, Philadelphia and New York City, 1860 and 1900



Source: Historical Urban Ecology Database, Center for Population Economics

Note: Dark gray denotes bad-quality wards. The cut-off ward development index for bad wards is 0.74 for 1860 and 0.65 for 1900.

Figure 4. Age at Death by Ward Development Index in 1860 and 1900



Note: For the figures in lower panel, we clustered wards into 10 groups according to each year's ward quality index.

Table 1: Earliest Year Ward-Level Data is Available, by City and Category

	Crime	Disease (deaths from and cases of)	Municipal (tax, elections)	Property	Vital Statistics
Baltimore	-	1881	1888	1890	1879
Boston	1900	1877	1800	1810	1849
Brooklyn	1892	1867	1855	1838	1870
Chicago	1875	1866	1860	1866	1866
Cincinnati	1875	1874	-	1870	1867
Manhattan	-	1874	1854	1830	1865
Philadelphia	1863	1861	1877	1875	1860

Notes: Years in columns refer to date statistic first appears in city annual reports. Crime refers to statistics related to criminality (e.g. homicides); diseases include cases of and deaths from specific diseases; municipal records include results from municipal elections and tax statistics; property refers to values and amount of personal property; vital statistics are births and deaths.

Table 2. Average Age at Death and Year of Birth by Type of Places over Lifetime

	Veterans who Survived the Civil War			Veterans who Survived up to 1900		
	Average Age at Death	Average Year of Birth	Sample Size	Average Age at death	Average Year of Birth	Sample Size
Birthplace						
US Urban Areas	68.0	1838.9	2,052	74.5	1840.1	1,268
US Rural Areas	70.3	1837.9	16,710	76.0	1839.4	11,369
Foreign Countries	68.7	1834.6	5,343	76.1	1837.0	2,866
Residence in 1860						
Good Wards	65.5	1836.6	283	73.5	1839.2	149
Bad Wards	65.0	1834.9	327	73.9	1838.7	162
Urban Areas	67.1	1835.6	976	75.9	1838.3	527
Rural Areas	70.0	1837.4	22,519	75.9	1839.1	14,665
Residence in 1900						
Good Wards				74.4	1840.2	337
Bad Wards				73.1	1839.4	346
Urban Areas				74.6	1839.4	1,550
Rural Areas				76.2	1839.0	13,270

Note: We used an index of living standards to measure the quality of wards in four large cities including Boston, Chicago, New York City and Philadelphia. Wards in 1860 and 1900 were evenly divided into two quality group (i.e. good and bad wards) in terms of the index. US urban areas are defined as cities whose populations in 1860 were more than 10,000.

Table 3. Type of Residence in Early Life and Estimated Hazard Ratio among Civil War Survivors

	(1)	(2)	(3)	(4)	(5)	(6)
Wartime Experiences				X	X	X
SES Controls					X	X
Fixed Effects						X
Birthplace: Reference = US Urban Areas						
US Rural Areas	0.8645***		0.9125***	0.9273***	0.9291***	0.9552*
	(0.0201)		(0.0222)	(0.0226)	(0.0228)	(0.0244)
Foreign Countries	1.0083		1.0511*	1.0630**	1.0628**	0.9598
	(0.0264)		(0.0283)	(0.0287)	(0.0287)	(0.3153)
Residence c.1860: Reference = Good Wards						
Bad Wards		1.0013	0.9811	0.9773	0.9635	0.9751
		(0.0874)	(0.0859)	(0.0860)	(0.0845)	(0.0875)
Urban Areas		0.8224***	0.8235***	0.8379**	0.8295***	0.8881
		(0.0587)	(0.0591)	(0.0607)	(0.0599)	(0.0664)
Rural Areas		0.6996***	0.7308***	0.7544***	0.7469***	0.7820***
		(0.0440)	(0.0469)	(0.0490)	(0.0488)	(0.0533)

Note: We used 24,105 veterans whose places at birth and in 1860 are known and who survived the Civil War. The table reports estimated hazard ratio and robust standard error in parentheses. Single asterisk denotes statistical significance at the 90% level of confidence, double 95%, triple 99%. All the regressions control for the year of birth. Variables of wartime experiences include dummies of enlistment year, initial rank and POW, and number of wartime diseases and wounds. Socioeconomic controls include total household wealth and occupational dummies in 1860. In model (6), state-of-birth and 1860-state fixed effects are added. See the text and Table 2 for the classification of places.

Table 4. Type of Lifetime Residence and Estimated Hazard Ratio among Old Veterans

	(1)	(2)	(3)	(4)	(5)	(6)
Wartime Experiences				X	X	X
SES Controls					X	X
Fixed Effects						X
Birthplace: Reference = US Urban Areas						
US Rural Areas	0.9050***		0.9371**	0.9456*	0.9589	0.9871
	(0.0289)		(0.0305)	(0.0307)	(0.0313)	(0.0336)
Foreign Countries	0.9880		1.0054	1.0168	1.0233	1.0481
	(0.0350)		(0.0356)	(0.0359)	(0.0362)	(0.4280)
Residence c.1860: Reference = Good Wards						
Bad Wards	0.9547		0.9751	0.9698	0.9548	0.9549
	(0.1142)		(0.1188)	(0.1189)	(0.1167)	(0.1199)
Urban Areas	0.7756***		0.8064**	0.8161**	0.8057**	0.8634
	(0.0733)		(0.0791)	(0.0808)	(0.0792)	(0.0883)
Rural Areas	0.7840***		0.8550*	0.8742	0.8505*	0.8879
	(0.0671)		(0.0765)	(0.0790)	(0.0770)	(0.0838)
Residence c.1900: Reference = Good Wards						
Bad Wards		1.2712***	1.2518***	1.2361**	1.2194**	1.1906**
		(0.1109)	(0.1075)	(0.1055)	(0.1045)	(0.1030)
Urban Areas		1.0431	1.0731	1.0704	1.0632	1.0597
		(0.0708)	(0.0726)	(0.0718)	(0.0718)	(0.0738)
Rural Areas		0.9046	0.9457	0.9537	0.9811	1.0165
		(0.0570)	(0.0605)	(0.0604)	(0.0629)	(0.0665)

Note: We used 15,503 veterans whose places at birth, in 1860 and in 1900 are known and who survived up to 1900. The table reports estimated hazard ratio and robust standard error in parentheses. Single asterisk denotes statistical significance at the 90% level of confidence, double 95%, triple 99%. All the regressions control for the year of birth. Variables of wartime experiences include dummies of enlistment year, initial rank and POW, and number of wartime diseases and wounds. Socioeconomic controls include total household wealth in 1860, occupational dummies in 1860, dummies of literacy, occupation, marital status, home ownership and household head in 1900. In model (6), state-of-birth, 1860-state, and 1900-state fixed effects are added. See the text and Table 2 for the classification of places.

Table 5. Estimated Effect of Ward-Quality Disparities on Longevity

	(1)	(2)	(3)	(4)	(5)
Previous Residence		X	X	X	X
Wartime Experiences			X	X	X
SES Controls				X	X
City Fixed Effects					X
Panel A: Sample = War Survivors, Year of Residence = 1860, Estimation = Proportional-Hazards					
Ward Development Index	0.7987	1.0624	1.1670	1.2372	0.8818
	(0.2662)	(0.3861)	(0.4487)	(0.4813)	(0.3819)
Panel B: Sample = War Survivors, Year of Residence = 1860, Estimation = OLS					
Ward Development Index	4.3524	0.7600	0.2004	-0.5359	5.9389
	(5.0392)	(5.1840)	(5.3960)	(5.4268)	(6.1169)
Panel C: Sample = 1900 Survivors, Year of Residence = 1900, Estimation = Proportional-Hazards					
Ward Development Index	0.2478***	0.3138***	0.3036***	0.3271***	0.2535***
	(0.0940)	(0.1186)	(0.1177)	(0.1330)	(0.1158)
Panel D: Sample = 1900 Survivors, Year of Residence = 1900, Estimation = OLS					
Ward Development Index	12.4546***	10.7519***	10.7531***	9.8391***	11.0952***
	(3.0720)	(3.0833)	(3.0968)	(3.1973)	(3.3785)

Note: Panels A and B use 610 veterans who survived the Civil War and who lived in a ward at the four cities in 1860. Panels C and B use 683 veterans who survived up to 1900 and who lived in a ward at the four cities in 1900. Panels A and C report hazard ratio and robust standard error estimated by Cox proportional-hazards regressions; panels B and D are based on OLS regressions whose dependent variable is age at death. Single asterisk denotes statistical significance at the 90% level of confidence, double 95%, triple 99%. Previous residence is birth place for panels A and B; it is birth place and 1860 residence for panels C and B. Previous residence is controlled by place dummies as used in Tables 3 and 4. All the regressions control for the year of birth. Other control variables are the same with those used in Table 3 and 4.

Table 6. Estimates Effect of Ward Ecological and Socioeconomic Conditions in 1900 on Longevity

Key Control Variable	Summary Statistics		Proportional-Hazards Model	OLS	Logit	
	Mean	S.D.			Death Year ≥ 1910	Death Year ≥ 1920
			(1)	(2)	(3)	(4)
Panel A: Ward Development Index and Its Components						
WDI	0.71	0.10	0.2535***	11.0952***	0.4783**	0.6296***
			(0.1158)	(3.3785)	(0.2152)	(0.1999)
Occupational Income Score	0.47	0.17	0.5212***	5.4298***	0.9986**	1.3962***
			(0.1215)	(1.8465)	(0.5032)	(0.5308)
Literacy Rate	0.89	0.11	0.4766*	5.9784**	1.4803*	1.0236
			(0.1910)	(2.8240)	(0.7976)	(1.0058)
School-Enrollment Rate	0.63	0.12	0.8858	1.8940	0.8759	0.6423
			(0.3177)	(2.6743)	(0.7424)	(0.8200)
Crude Death Rate	0.84	0.14	0.5347*	4.4417*	0.5173	2.9533**
			(0.1778)	(2.4228)	(0.6859)	(1.1949)
Panel B: Sanitary and Geographical Condition						
Population Density	59.19	59.96	1.0009	-0.0085	-0.0029*	-0.0010
			(0.0009)	(0.0065)	(0.0017)	(0.0018)
Water Pipe	10.87	25.23	1.0019	-0.0137	-0.0060	-0.0023
			(0.0020)	(0.0151)	(0.0040)	(0.0042)
Mean Elevation	63.90	73.62	1.0006	0.0048	-0.0045	-0.0043
			(0.0024)	(0.0193)	(0.0055)	(0.0064)
Variance of Elevation	95.63	193.28	1.0001	-0.0004	-0.0004	-0.0004
			(0.0002)	(0.0015)	(0.0004)	(0.0005)
Panel C: Disease Environment						
Child Survival Rate	0.85	0.02	0.0013***	50.8661***	5.5176	12.1002***
			(0.0026)	(15.2511)	(4.2101)	(4.5264)
Est. Child Mortality Rate	60.10	29.28	1.0019	-0.0141	-0.0022	-0.0092**
			(0.0016)	(0.0100)	(0.0029)	(0.0040)
Panel D: Socioeconomic Status						
Ratio of Homeowners	0.22	0.09	1.6199	-1.4484	-0.7798	0.4613
			(0.8560)	(4.0629)	(1.0808)	(1.0523)
Ratio of Married Adults	0.35	0.04	0.8671	5.0964	1.6116	2.9324
			(1.0227)	(8.7742)	(2.3317)	(2.6304)
Male Labor Force Participation Rate	0.92	0.04	2.1472	-3.3052	0.9560	1.0565
			(2.6047)	(8.1965)	(2.2148)	(2.0130)

Note: We use 683 veterans who lived in one of the four large cities (Boston, Chicago, New York City, Philadelphia) in 1900. The variables listed in the first column measure various aspects of ward living standards. Each variable is used as a key variable in the regression, respectively. We only report the coefficient of the variable and its robust standard error. The coefficient in logit regression is that of marginal effect. We uses the same specification with that of model (5) in Table 5. Single asterisk denotes statistical significance at the 90% level of confidence, double 95%, triple 99%.

Bibliography

Ali M, Emch M, Tofail F, Baqui AH. 2001. Implications of health care provision on acute lower respiratory infection mortality in Bangladeshi children. *Social Science & Medicine* 52(2): 267-277.

Altmayer CA, Hutchison BG, Torrance-Rynard VL, Hurley J, Birch S, Eyles JD. 2003. Geographic disparity in premature mortality in Ontario, 1992-1996. *International Journal of Health Geographics* 2(7).

Barford A, Dorling D. 2007. The shape of the global causes of death. *International Journal of Health Geographics* 6(48).

Barker DJP. 1992. *Fetal and infant origins of adult disease.* London: British Medical Journal Publishing Group.

Barker DJP. 1998. *Mothers, babies, and health in later life*. Edinburgh: Churchill Livingstone.

Barker DJP. 2003. *The best start in life*. London: Century.

Becker GS. 1993. *Human Capital*. Chicago: University of Chicago Press.

Bell FC. 1997. Social Security area population projections: 1997. In *Social Security Administration Actuarial Study No. 112*. Washington: Social Security Administration, Office of the Chief Actuary.

Bengtsson T, Lindstrom M. 2003. Airborne infectious diseases during infancy and mortality in later life in southern Sweden, 1766-1894. *International Journal of Epidemiology* 32: 286-294.

Blackwell DL, Hayward MD, Crimmins EM. 2001. Does childhood health affect chronic morbidity in later life? *Social Science & Medicine* 52(8): 1269-1284.

Blake NM. 1956. *Water for the cities: A history of the urban water supply problem in the United States.* Syracuse University Press.

Buck C, Simpson H. 1982. Infant diarrhea and subsequent mortality from heart disease and cancer. *Journal of Epidemiology and Community Health* 36: 27-30.

Cain, LP. 1978. *Sanitation Strategy for a Lakefront Metropolis: The Case of Chicago.* Northern Illinois University Press.

Cain LP, Hong, SC. 2009. Survival in 19th century cities: The larger the city, the smaller your chances, *Explorations in Economic History* 46(4): 450-463,

Cain LP, Rotella EJ. 2001. Death and spending: Did urban mortality shocks lead to municipal expenditure increases? *Annales de Démographie Historique*, Pt. 1: 139-154.

Cain, LP, Rotella, EJ. 2008. Epidemics, Demonstration Effects, and Municipal Investment in Sanitation Capital. In Joshua Rosenbloom, editor, *Quantitative Economic History: The Good of Counting.* London and New York: Routledge.

Carrington W, Detragiache E, Vishwanath T. 1996. Migration with endogenous moving costs. *American Economic Review* 86: 909-930.

CDC. 1999b. CDC on infectious diseases in the United States, 1900-99. *Population and Development Review* 25(3): 635-640.

Citizens' Association of New York. 1866. *Report of the council of hygiene and public health of the Citizens' Association of New York upon the sanitary condition of the city*. New York: D. Appleton.

Coale A, Demeny P. 1983. *Regional model life tables and stable populations*. New York: Academic Press.

Condran GA, Cheney RA. 1982. Mortality trends in Philadelphia: Age- and cause-specific death rates, 1870-1930. *Demography* 19(1): 97-123.

Condran GA, Crimmins EM. 1978. Public Health Measures and Mortality in U.S. Cities in the Late 19th Century, *Human Ecology*: 6(1) 27-54.

Condran GA, Crimmins EM. 1980. Mortality differentials between rural and urban areas of states in the northeastern United States 1890-1900. *Journal of Historical Geography* 6(2): 179-202.

Congdon P. 2007. A model for spatial variations in life expectancy; Mortality in Chinese regions in 2000. *International Journal of Health Geographics* 6(16).

Costa DL. 2000. Understanding the twentieth-century decline in chronic conditions among older men. *Demography* 37(1): 53-72.

Costa DL. 2005. Causes of improving health and longevity at older ages: A review of the explanations. *Genus* 61: 21–38.

Costa DL, Kahn ME. 2006. Forging a New Identity: The Costs and Benefits of Diversity in Civil War Combat Units for Black Slaves and Freemen. *Journal of Economic History.*

Craddock, Susan. 2000. *City of Plagues: Disease, Poverty, and Deviance in San Francisco*. Minneapolis: University of Minnesota,

Cutler DM, Meara E. 2004. Changes in the age distribution of mortality over the twentieth century. In *Perspectives on the economics of aging*, ed. DA Wise. Chicago: The University of Chicago Press.

Cutler DM, Miller G. 2005. The role of public health improvements in health advances: The twentieth century United States. *Demography* 42(1): 1-22.

Deaton A, Paxson C. 2001. Mortality, education, income, and inequality among American cohorts. In *Themes in the economics of aging*, ed. DA Wise. Chicago: University of Chicago Press.

DeBats DA, Lethbridge M. 2005. GIS and the City: Nineteenth-Century Residential Patterns. *Historical Geography:* 33 78-98.

De Moor M, Wiedemann T. 2001. Reconstructing Territorial Units and Hierarchies: A Belgian Example. *History and Computing:* 13(1) 71- 97.

Duffy J. 1968. A history of public health in New York City, 1625-1866. New York: Russell Sage Foundation.

Ewbank DC, Preston SH. 1990. Personal health behaviour and the decline in infant and child mortality: The United States, 1900-1930. In *What we know about health transition: The cultural, social and behavioural determinants of health*, ed. J Caldwell et al., 116-149. Canberra: Australian National University Printing Service.

Ezzati M, Lopez AD, Rodgers A, Vander Hoorn S, Murray CJL. 2002. Selected major risk factors and global and regional burden of disease. *Lancet* 360(9343): 1347-1360.

Farr W. 1843. *Regulations; and a Statistical Nosology: Comprising the Causes of Death, Classified and Alphabetically Arranged, with Notes and Observations.* London: W. Clowes and Sons.

Ferrie JP, Troesken W. 2008. Water and Chicago's mortality transition, 1850–1925, "Explorations in Economic History, 45(1) 1-16.

Finch CE, Crimmins EM. 2004. Inflammatory exposure and historical changes in human life-spans. *Science* 305(5691): 1736-1739.

Fogel RW. 1994. Economic growth, population theory, and physiology: The bearing of long-term processes on the making of economic policy. *American Economic Review* 84: 369-395.

Fogel RW. 2000. *The fourth Great Awakening and the future of egalitarianism*. Chicago: University of Chicago Press.

Fogel RW. 2004a. *The escape from hunger and premature death, 1700-2100: Europe, America, and the third world.* New York: Cambridge University Press.

Fogel RW, Costa DL. 1997. A theory of technophysio evolution, with some implications for forecasting population, health care costs, and pension costs. *Demography* 34: 49–66.

Gilliland JA, Olson SH, Gauvreau D. 2011. Did Segregation Increase as the City Expanded?: The Case of Montreal, 1881-1901. *Social Science History*: 35(4) 465-503.

Gilliland JA, Olson SH. 2003. Montréal, L'avenir Du Passé." *GÉOinfo*, January-February 5-7.

Gregory IN, Bennett C, Gilham VL, Southall HR. 2002. The Great Britain Historical GIS Project: From Maps to Changing Human Geography. *The Cartographic Journal:* 39(1) 37-49.

Haines, M. 2001. The Urban Mortality Transition in the United States: 1800-1940. *Annales de Demographie Historique* 2001:33-64.

Hakre S, Masuoka P, Vanzie E, Roberts DR. 2004. Spatial correlations of mapped malaria rates with environmental factors in Belize, Central America. *International Journal of Health Geographics* 3(6).

Hinman SE, Blackburn JK, Curtis A. 2006. Spatial and temporal structure of typhoid outbreaks in Washinton, D.C., 1906-1909: Evaluating local clustering with the G_i^* statistic. *International Journal of Health Geographics* 5(13).

Hong, SC. 2007. The Burden of Early Exposure to Malaria in the United States, 1850–1860: Malnutrition and Immune Disorders, *The Journal of Economic History*: 67(04), 1001-1035,

Hong, SC. 2011. Malaria and Economic Productivity: A Longitudinal Analysis of the American Case. The Journal of Economic History 71(3): 654-671.

Humphreys M. 2001. *Malaria: Poverty, race, and public health in the United States*. Baltimore: Johns Hopkins University Press.

Jones H. 1956. A special consideration of the aging process, disease, and life expectancy. *Advances in Biological and Medical Physics* 4: 281-337.

Leavitt JW. 1996. *The healthiest city: Milwaukee and the politics of health reform.* Madison: University of Wisconsin Press.

Lee C. 2003. Prior exposure to disease and later health and mortality. In *Health and Labor Force Participation over the Life Cycle*, ed. DL Costa, 51–87. Chicago and London: The University of Chicago Press.

Logan JR, Jindrich J, Shin H, Zhang W. 2011. Mapping America in 1880: The Urban Transition Historical GIS Project. *Historical Methods: A Journal of Quantitative and Interdisciplinary History*: 44(1) 49-60.

Manton KG, Corder LS, Stallard E. 1997. Chronic disability trends in elderly United States populations: 1982-1994. *Proceedings of the National Academy of Sciences USA* 94(6): 2593-2598.

Marmot M. 2004. *The status syndrome: How social standing affects our health and longevity*. New York: Times Books/Henry Holt.

McCarthy MP. 1987. *Typhoid and the Politics of Public Health in Nineteenth-Century Philadelphia.* Philadelphia: American Philosophical Society.

McDermott W. 1978. Medicine: The public good and one's own. *Perspectives in Biology and Medicine* 21(2): 167-187.

McKeown T. 1976. *The modern rise of population*. New York: Academic Press.

Meeker, E. 1971. The improving health of the United States, 1850–1915. *Explorations in Economic History*: 9(1), 353-373.

Meeker, E. 1974. The Social Rate of Return on Investment in Public Health, 1880-1910. *The Journal of Economic History*: 34(3) 392-421.

Mokyr, J., Stein, R. 1996. Science, Health, and Household Technology: The Effect of the Pasteur Revolution on Consumer Demand. In Timothy F. Bresnahan and Robert J. Gordon, editors, *The Economics of New Goods*. Chicago: University of Chicago Press..

Montgomery J. 1991. Social networks and labor-market outcomes: Toward an economic analysis. *American Economic Review* 81: 1408-1418.

Munshi K. 2003. Networks in the modern economy: Mexican migrants in the US labor market. *Quarterly Journal of Economics* 118(2): 549-599.

National Central Bureau of Statistics. 1996. *Population statistics, infant mortality, 1891-1995, life tables 1891-1995*. Stockholm, Sweden: SCB.

Preston SH, Haines M. 1991. *Fatal Years: Child Mortality in Late Nineteenth Century America*. Princeton: Princeton University Press.

Reed DM, Foley DJ, White LR, Heimovitz H, Burchfiel CM, Masaki K. 1998. Predictors of health aging in men with high life expectancies. *American Journal of Public Health* 88(10): 1463-1468.

Rosner D. ed. 1995. *Hives of sickness: Public health & epidemics in New York City*. New Brunswick: Rutgers University Press for City Museum of New York.

Shah AM, Whitman S, Silva A. 2006. Variations in the health conditions of 6 Chicago community areas: A case for local-level data. *American Journal of Public Health* 96(8): 1485-1491.

Shaheen SO, Barker DJP, Shiell AW, Crocker FJ, Wield GA, Holgate ST. 1994. The relationship between pneumonia in early childhood and impaired lung function in late adult life. *American Journal of Respiratory and Critical Care Medicine* 149(3): 616-619.

Shattuck L. 1850. *Report of a general plan for the promotion of public and personal health, devised, prepared and recommended by the commissioners appointed under a resolve of the legislature of Massachusetts, relating to a sanitary survey of the state.* Boston: Dutton & Wentworth.

Tarr JA, McCurley JI, McMichael FC, Yosie T. 1984. Water and wastes: A retrospective assessment of wastewater technology in the United States, 1800-1932. *Technology and Culture* 25(2): 226-263.

Tiwari N, Adhikari CMS, Tewari A, Kandpal V. 2006. Investigation of geo-spatial hotspots for the occurrence of tuberculosis in Almora district, India, using GIS and spatial scan statistic. *International Journal of Health Geographics* 5(33).

Troesken W. 2004. *Water, race and disease*. Cambridge: MIT Press.

US Census Bureau. 1896. *Report on vital and social statistics in the United States at the eleventh census, 1890: Part II - vital statistics: Cities of 100,000 population and upward.* Washington: Government Printing Office.

Vaananen A, Toppinen-Tanner S, Kalimo R, Vahtera J, Peiro JM. 2003. Job characteristics, physical and psychological symptoms, and social support as antecedents of sickness absence among men and women in the private industrial sector. *Social Science & Medicine* 57(5): 807-824.

Valkonen T, Sihvonen A-P, Lahelma E. 1997. Health expectancy by level of education in Finland. *Social Science and Medicine* 44(6): 801-808.

van Poppel F, van der Heijden C. 1997. The effects of water supply on infant and childhood mortality: A review of historical evidence. *Health Transition Review: The Cultural, Social, and Behavioural Determinants of Health* 7: 113-148.

Williams JG. 1976. American prices and urban inequality since 1820. *Journal of Economic History* 36(2): 303-333.