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Joseph P. Ferrie
Werner Troesken

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ABSTRACT

Between 1850 and 1925, the crude death rate in Chicago fell by 60 percent, driven by reductions in infectious disease rates and infant and child mortality. What lessons might be drawn from the mortality transition in Chicago, and American cities more generally? What were the policies that had the greatest effect on infectious diseases and childhood mortality? Were there local policies that slowed the mortality transition? If the transition to low mortality in American cities was driven by forces largely outside the control of local governments (higher per capita incomes or increases in the amount and quality of calories available to urban dwellers from rising agricultural productivity), then expensive public health projects, such as the construction of public water and sewer systems, probably should have taken a back seat to broader national policies to promote overall economic growth. The introduction of pure water explains between 30 and 50 percent of Chicago's mortality decline, and that other interventions, such as the introduction of the diphtheria antitoxin and milk inspection had much smaller effects. These findings have important implications for current policy debates and economic development strategies.

Joseph P. Ferrie
Department of Economics
Northwestern University
Evanston, IL 60208-2600
and NBER
ferrie@northwestern.edu

Werner Troesken
Department of History
University of Pittsburgh
Pittsburgh, PA 15260
and NBER
troesken@pitt.edu

I. Introduction

Between 1850 and 1925, the crude death rate in the City of Chicago fell by 60 percent. This dramatic reduction in overall mortality was driven by reductions in infectious disease rates and infant and child mortality. Before 1880, more than half of all deaths in the city occurred among children less than 5 years old, and the leading causes of death were diarrheal diseases—such as typhoid fever, cholera infantum, and dysentery—and respiratory diseases—such as tuberculosis, influenza, bronchitis, and pneumonia. By 1925, less than a quarter of all deaths in the city occurred among children less than 5; deaths from typhoid had been largely eradicated; and respiratory diseases, while still common, were being eclipsed by heart disease and cancer as the city's leading causes of death. In short, as Chicago moved from a high mortality to a low mortality environment, chronic diseases and diseases of old age replaced infectious diseases and child mortality as the city's primary causes of death.[1]

Chicago is a paradigmatic case. In Boston, Philadelphia, New York, and other large American cities, the identical transitions in mortality took place over the same time period, while in smaller American cities, the some transitions took place only a decade or two later (Haines 2001). Furthermore, with the exception of AIDS and certain diseases related to new forms of industrial pollution, Chicago's disease environment in 1880 is very similar to the disease environment observed in the developing world today.

Hence, for development agencies and policymakers today, the questions raised by Chicago's experience are clear and salient. What lessons might be drawn from the mortality transition in Chicago, and American cities more generally? In particular, what were the policies implemented by

[1]See table 1 for data and sources.

urban governments that had the greatest effect on infectious diseases and childhood mortality? Alternatively, were there any local policies that slowed the mortality transition? It is also possible that the transition from high to low mortality in American cities was driven by forces largely outside the control of local governments, such as rising per capita incomes and increases in the amount and quality of calories available to urban dwellers as a result of rising agricultural productivity. If so, this would suggest that expensive public health projects, such as the construction of public water and sewer systems, probably should take a back seat to broader national policies designed to promote overall economic growth. In this context, cheaper modes of promoting public health such as vaccination programs and information campaigns aimed at changing household behavior would also become more attractive.

With these larger motivations in mind, we use this paper as a vehicle to identify and explain the sources of Chicago's mortality transition.[2] We give particular attention to the effects of the development of the city's public water and sewer systems. Although the urban mortality transition in the United States has been widely and thoroughly studied, there are two novel aspects to our efforts in this paper. First, we revive, reformulate and test, a long-forgotten theory in the annals of American public-health: the Mills-Reincke phenomenon, also known as the Hazen theorem. Once a widely-accepted proposition among public-health practitioners in the United States and Europe, the Mills-Reincke phenomenon maintained that improvements in public water supplies not only reduced deaths from waterborne diseases such as typhoid fever and diarrhea, but also reduced deaths from diseases and ailments not usually thought of as being water-related, such as, kidney and heart failure,

[2]In many ways, our analysis complements a recent study by Cutler and Miller (2004). Cutler and Miller focus on a large sample of American cities, largely for the period following 1900. Our analysis focuses more narrowly, but also more deeply, on a single city and over a longer period of time. For Chicago, the most important improvements in the city's water supply occurred before 1900, as did the largest improvements mortality and disease rates.

tuberculosis, pneumonia, and influenza (Sedgwick and McNutt 1910). Evidence presented below supports most aspects of the Mills-Reincke phenomenon, and suggests that the long-term benefits of public water purification on human mortality were enormous, accounting for 30 to 50 percent of the mortality reduction observed in Chicago between 1850 and 1925.

Second, we analyze a new data set based on the 1880 Census. From the Census, we have gathered data on a large sample of households in the City of Chicago and merged these data with maps of Chicago's public water and sewer lines. Because the 1880 Census asked detailed questions about health outcomes in the household over the previous year, we are able to explore how access to public water and sewer lines affected household health outcomes, after controlling for other household characteristics. This analysis will help reconcile recent studies in the development literature which appear to report conflicting findings regarding the efficacy of pure water on household health outcomes. In particular, this analysis will highlight the folly of equating pure water with piped water, as is sometimes done in contemporary development studies. Chicago's experience shows that distributing impure water through public water pipes can actually cause disease and death rates to rise because public water supplies in this case hasten the diffusion of disease-causing pathogens.

II. Chicago's Mortality Transition

An Overview

Table 1 provides a closer look at the elements comprising Chicago's mortality transition. As the table shows, during the 1870s, the death rate from all causes in Chicago averaged 212 deaths per 10,000 persons. In contrast to modern populations, heart and kidney disease accounted for a relatively small number of deaths; less than 3 percent of all deaths were attributable to heart and kidney disease. Along the same lines, cancer accounted for less than 2 percent of all deaths. Instead,

most deaths occurred among the very young and were caused by infectious diseases. Specifically, about one-third of all deaths occurred among infants (children aged 0 to 1), and nearly one-fifth were caused by respiratory diseases, such as tuberculosis, influenza, pneumonia, and bronchitis. Child mortality (children aged 1 to 4), which is an aggregate measure of infectious childhood diseases such as measles, whooping cough, and scarlet fever, represented just under a quarter of all deaths. Typhoid fever and diphtheria, two prominent infectious diseases, combined to cause 8 percent of all deaths.

By the second decade of the twentieth century, Chicago's mortality profile had changed dramatically. The death rate from all causes had fallen by 40 percent, and infant and childhood mortality had fallen sharply. While child and infant mortality still accounted for about one-quarter of all deaths, this represented a large improvement over the 1870s, when they accounted for more than half of all deaths. Respiratory diseases, although they represented a larger share of all deaths between 1915 and 1925 than they had in the 1870s, had fallen by about one third. Typhoid and diptheria had all but disappeared, with typhoid accounting for less than one-half of 1 percent of all deaths. With the decline in infectious diseases, diseases of old-age began to emerge as leading causes of death. Heart and kidney disease, for example, accounted for nearly one quarter of all deaths observed between 1915 and 1925, while cancer alone accounted for 8 percent of all deaths.

Isolating the Possible Causes of the Mortality Transition

What were the causes of this dramatic decline in mortality? One obvious starting point in answering this question is to perform a simple decomposition and identify which diseases and age-related disease categories experienced the largest declines over this period. When this is done, it is clear that much of the improvement in overall mortality occurred because of improvements taking place in the disease environment for young children. Infant mortality rates fell by 54 points and

child mortality rates fell by 39 points, which is more than the total point decline in overall mortality (87 points).[3] This suggests that policies that affected the very young, and childhood diseases more generally, were likely important factors in the city's mortality decline. Alternatively, if one examines specific diseases rather than age-specific death rates, one finds that changes in typhoid, diphtheria, and respiratory diseases explain a non-trivial portion of the mortality decline. In particular, typhoid rates fell by 6 points and account for about 7 percent of the decline in overall mortality; diphtheria rates fell by 9 points and account for 10 percent of the decline; scarlet fever rates fell by 7 points and account for 8.5 percent of the decline; and respiratory diseases fell by 9.5 points and account for 11 percent of the decline. This suggests that policies aimed at combating infectious diseases are likely candidates in the search for policies that most reduced mortality.

A more direct way of isolating the policies that had the biggest effect on the mortality decline is to plot death rates over time and search for obvious breaks in the trend line. If these breaks in trend can be easily correlated with particular policy changes, this would help narrow the set of potential candidates. To this end, figure 1 plots the total mortality rate in Chicago, defined here as deaths from all causes per ten thousand persons, from 1853 through 1925. The results are striking. Although death rates appear to be trending downward almost from the start of the time series, that trend is modest and highly variable. Indeed this graph understates the variability observed between 1853 and 1880, as three epidemic years, when death rates were as high as 600, are dropped from the time series so that trends can be more easily established. The first clear break in trend occurs around 1893. In that year, death rates begin a sharp downward trend, and never again even remotely

[3]Put another way, the decline in infant mortality accounts for 45 percent of the total decline in overall mortality decline, while the decline in child mortality accounts for 62 percent. The percentages sum to more than 100 percent because death rates among older-aged persons are rising over the same time period.

approach levels between 200 and 250 which were commonplace before 1893. By the late 1890s, death rates had fallen to around 150, where they stayed until 1917, when another sharp break in trend is observed and death rates fall to around 110.

What were the relevant policy changes observed around 1893 and 1917? As a means of identifying the possible candidates, we surveyed the *Annual Reports of the Chicago Department of Health*, which periodically would publish histories of important changes in the city's policies regarding public health. For the years around 1893, the health department identified three possibilities. The first possibility is the completion of the four-mile water intake crib and the permanent closure of shoreline water intakes. To understand the significance of these changes note that Chicago dumped all of its sewage along the shores of Lake Michigan or into the Chicago River, which at this point emptied into the lake. By moving the water intakes far from shore, the city water supply was much less likely to become contaminated by sewage, and by the diseases carried in sewage. The second possibility is an administrative change in the city's milk inspection regime. A milk inspection bureau was first created by the city council in 1892. But in 1893 jurisdiction over milk inspection was transferred from this independent and largely ineffective bureau to the city health department. There is evidence that the health department became more aggressive in terms of inspections and publicizing the results of their inspections. The third possibility is the introduction of a public health program to combat diphtheria in 1895. This program involved the distribution of the diphtheria anti-toxin, which when introduced early during the course of the disease had a large effect on the case fatality rate.

Milk Inspection

At first glance, the most promising candidate is milk inspection. One can make a strong case that milk inspection, of all three of the policies specified above, would have had the largest impact

on infant and child mortality rates. There are many historical studies linking impure milk to gastro-intestinal diseases among infants, and gastrointestinal deaths accounted for about half of all infant deaths during this period. Furthermore, the data presented in table 1 suggest that changes in infant mortality rate represent the largest component of the decline in overall mortality observed between 1870 and 1925. Beyond this, there is evidence that inspections by the health department had an effect on the behavior of milk suppliers and on the quality of milk sold. In this regard, milk vendors could, and often were, imposed fines for selling below-grade milk. During the late 1890s, the health department started publishing the names of milk vendors who sold below grade milk, and data presented by the health department suggest that this reduced, by a significant amount, the quantity of below-grade milk sold in Chicago. In particular, between 1894 and the first half of 1895, the proportion of milk sampled by the health department that was declared below grade varied between 20 and 50 percent. After the health department began publishing the names of milk vendors selling below-grade milk in May of 1895, the proportion of milk sold in the city that was below grade averaged between 5 and 10 percent.[4]

But the claim that milk inspection played a substantial role in reducing overall mortality in general, and mortality among very young children in particular, does not withstand close empirical scrutiny. Consider, first, what milk inspection did and did not involve. When the city health department classified milk as below-grade it did so on the basis of the amount fat and natural cream contained in the milk—if there was not enough fat and cream in the milk, it was declared below grade. Milk was not declared below grade on the basis of bacterial counts and it was bacteria that gave children diarrhea, not fat and cream or the absence thereof. Furthermore, we are aware of no

[4]The discussion of milk inspection in Chicago is based on various issues of the annual reports of the Chicago Health Department.

no scientific evidence indicating that low-fat milk is a less conducive venue for bacterial proliferation than high-fat milk. In short, all Chicago milk inspection told consumers was whether or not they were receiving whole milk or skim milk. Of course it is possible that milk vendors who sold skim milk also adopted other careless practices such as not properly cooling the milk they were selling. The correlation between selling skim milk and careless sanitary practices, however, would have had to have been very large for milk inspection to have reduced infant mortality rates by any sizeable amount.

Nevertheless, for the sake of argument, assume for the moment that milk inspection had involved bacterial counts and that milk designated as below-grade was so designated because it contained high levels of bacteria. On a purely intuitive level, it is not clear that there would have been a strong correlation between the quality of milk sold and mortality rates among very young children. Because the harmful bacteria in milk proliferate rapidly, even high-grade milk—defined for the moment as milk with relatively low bacteria counts when sold—would have experienced rapid growth in the number of bacteria if the milk was not properly stored and cooled. Infants and children drinking improperly-stored high-grade milk would have experienced as much gastrointestinal distress as those who consumed low grade milk immediately after purchase, or low-grade milk that was properly stored. In addition, it was relatively easy for parents to protect their children against the diarrheal diseases resulting from impure milk. Breast-feeding infants and boiling the milk consumed by pre-school children were extremely effective preventive measures (Condran and Preston 1994; Ewbank and Preston 1990; Woodbury 1926).

Furthermore, it is difficult to find a strong correlation between child mortality and Chicago's milk inspection program. Consider the correlation between child mortality and the quality of milk (measured in terms of fat and cream content) across city wards. There was much variation in

mortality among children less than four years old across wards in Chicago. For example, in 1894, the child mortality rate in the sixteenth ward was 10.9 deaths per ten thousand, 5 times greater than child mortality in the twenty-forth ward (2.1 deaths per ten thousand). In the same year, variation in milk quality appears to have been much smaller: in the city ward with the worst milk supply, 56 percent of all milk sampled was deemed below-grade (N = 108), while in the city ward with the best milk supply, 35 percent of the sample was deemed below-grade (N = 238). Plotting the relationship child mortality rates across wards in 1894 against the proportion of all milk that failed inspection across wards in the same year, the correlation between milk quality and child mortality appears weak. See figure 2. More formally, if one uses these ward level data to regress child mortality rates against milk quality, a weak correlation emerges in the regression models with no controls. But as one adds control for population density (as proxied crudely by population levels), proportion of the population that was foreign-born, overall healthfulness of the city ward (as proxied by the death rate for all persons five years and older in 1894), and the quality of the ward's water supply (as proxied by the death rate from typhoid fever in 1893, a one year lag), the correlation between milk quality and child mortality vanishes. (Results available upon request.)

Another question about the demographic effects of milk inspection arises when one considers another milk-related measure passed by the Chicago City Council in 1908. In that year, the city mandated that all milk that did not come from a farm or vendor that had been certified as disease-free had to be pasteurized before being sold to consumers. This strikes us as a policy change that would have had a much larger effect on the bacterial content of milk than inspections based on the fat and cream content of milk. Yet if one examines infant and child mortality rates before and after this change, there is no evidence of large changes in mortality rates. For the three years preceding this change, infant (child) mortality rates averaged 31.1 (14.6) deaths per ten thousand,

while for the three years following this change, infant (child) mortality rates average 29.6 (13.9). These are hardly large reductions, and whatever reductions we do are observe, they were probably the result of pre-existing trends in mortality. This can be seen in greater detail in the figures 3 and 4, which are discussed below.

Two final pieces of evidence against milk inspection as an important source of improved mortality are the strong downward trends in infant and child mortality preceding the introduction of milk inspections around 1893. These trends are shown in figures 3 and 4. It is clear from both figures that infant and child mortality were trending down sharply in the years before effective milk inspection in 1893. Along with observed mortality rates, trend lines for the pre- and post-1893 periods are also shown. Death rates after 1893 do not appear to differ all that much, if at all, from the rates predicted by the pre-1893 trend lines. As for changes in trend, the downward trends in mortality appear to have slowed after the commencement of milk inspection in 1893. Formal regressions confirm visual inspection. Regressing infant mortality (the death rate for children under one year of age) rates against a time trend and dummy variable equal to one for all years following 1893, yields the following result:

Infant mortality rate = 1964.8**Constant* - 1.01**Year* - 11.1**Post-1893dummy*
t-statistic (8.58) (8.28) (2.72)

Performing the identical regression for child mortality rates (the death rate for children one to four years of age) yields the same result:

Child mortality rate = 1283.4**Constant* - .659**Year* - 10.4**Post-1893dummy*
t-statistic (6.86) (6.63) (3.10)

If one attributes the entire post-1893 reduction in infant and child mortality rates to milk inspection, these results suggest that milk inspection reduced infant (child) mortality rates by 11 (10) deaths per ten thousand. This, in turn, would suggest that milk inspection accounted for about 20

(25) percent of the reduction in infant (child) mortality. Although a program that reduces childhood mortality rates by 20 to 25 percent is significant, it is important to remember that the estimating procedure employed here is quite crude and conflates the effects of all policy changes occurring around 1893 with the effects of water filtration. In this way, the regression results overstate the share of the mortality reduction attributable to milk inspection. As will be made clear below, other policy changes in 1893 likely had a much larger effect on childhood mortality, but before turning to that evidence it was worth posing the following question: how could inspecting milk for fat and cream content possibly have reduced infant mortality rates by 20 to 25 percent? Reduced fat content might have undermined children's long-term growth and development and left them more vulnerable to infectious diseases at later life stages, but there is no scientific evidence to suggest that reducing milk's fat content would have increased infant and childhood diarrhea rates, the primary killer of infants and very young children.

The Diphtheria Antitoxin

With the development of the germ theory of disease during the late nineteenth century, doctors and medical researchers were able to identify and develop a variety of vaccines and antitoxins to prevent and cure infectious diseases. One of the most important of these was the development of the antitoxin for diphtheria. Introduced early on in the treatment of the disease, the diphtheria antitoxin had powerful curative effects, cutting the case fatality rate by roughly 40 percent for the most virulent strains of the disease (Hammonds 1999). In 1895, the City of Chicago launched a campaign to inform local doctors and parents of the antitoxin and to distribute the antitoxin to local hospitals and doctors. After documenting the effects of the antitoxin, the health department celebrated, claiming that the department had demonstrated "the great change in mortality wrought by the antitoxin." Figure 5 plots the death rate from diphtheria, measured as

deaths from diphtheria per ten thousand persons, from 1867 through 1925. The vertical line indicates the year 1895. Although diphtheria rates were starting to trend down before the health departments campaign in 1895, this was probably driven by the early adoption of the antitoxin by a handful of doctors and hospitals in the city. Overall, the trends in the death rate seem to suggest that the introduction of antitoxin, which was hastened by the policies of the city health department, had a large and lasting effect on the diphtheria death rate in Chicago.[5]

If one makes the extreme assumption that the entire decline in diphtheria death rates observed between 1870 and 1925 was the result of the health department's antitoxin campaign, this would suggest that 10 percent of the decline in overall mortality in Chicago is attributable to the city's diphtheria antitoxin campaign. While this estimate suggests the effects of the antitoxin campaign were non-trivial, it is an upper-bound estimate. Also, this still leaves a large proportion of the post-1893 reduction in mortality unexplained. In particular, the introduction of the diphtheria antitoxin might account for the decline diphtheria, but it cannot account for the large mortality reductions observed in other diseases, including typhoid, tuberculosis, and infant deaths from diarrhea.

Water Purification

Based on the data presented in table 1, water purification appears to be an improbable candidate for ushering in Chicago's mortality transition for two reasons. First, improvements in the milk supply would have had a much larger effect on infant death rates than purifying water for the simple reason that infants who were not breast fed were fed cow's milk. Infants were consuming little water and so it seems unlikely that improvements in the quality of water could have greatly

[5]The history diphtheria prevention in Chicago is based various issues of the annual reports of the Chicago Health Department.

affected infant health. We know much of the decline in overall human mortality in Chicago was driven by reductions in infant mortality. Second, while it is true that water purification measures in Chicago had large effects on the city's death rate from typhoid fever, typhoid fever represented a small portion of all deaths, even before water filtration. As shown in table 1, between 1870 and 1879, deaths from typhoid fever accounted for less than 3 percent of all deaths in the city, and its near eradication by 1925 explains less than 10 percent of the decline in overall mortality observed during this period.

Typhoid's middling performance as a killer is puzzling when one considers the lengths cities went to in trying to eliminate the disease. Consider the truly herculean efforts made by the City of Chicago to ensure that its water was free from bacterial pollution in the form of raw sewage. In 1893, the city installed a Four-Mile Water Intake Crib, an effort that required tunneling four miles out into Lake Michigan. In 1900, it opened the Chicago drainage canal, an investment that required reversing the flow of an entire river that otherwise would have carried raw sewage into Lake Michigan. After completion of the drainage canal, Chicago's sewage was carried downstream to the Mississippi River and St. Louis, Missouri. These were mammoth undertakings.[6] Nor were Chicago's efforts in this regard unique. New York City and Los Angeles built huge systems of aqueducts to gain access to water supplies that were pure. In general, spending on the construction of water and sewer systems by local governments far exceeded spending in all other areas during the late nineteenth century. For example, in 1885, American cities spent 3.5 times more on water and sewer systems than they spent on streets, and 8 times more than they spent on fire protection (Melosi 2001, p. 110). Why did local governments spend so much to eliminate what appears to have

[6]We base this discussion on the history of Chicago's water supply presented by the Chicago Bureau of Public Efficiency (1917), hereafter cited as CBPE (1917).

been a relatively pedestrian cause of death?

Whatever motivated cities to invest the capital necessary to promote water purity, the effects on typhoid were dramatic. Figure 6 plots typhoid rates in Chicago from 1853 through 1925. There are four vertical lines, each corresponding to technological improvements that would have promoted water purity. In 1893 and 1900, there were the installation of the aforementioned four-mile intake and the opening of the Chicago drainage canal. In 1913, the city began chlorinating part of its water supply and in 1917 it installed the second and final component in its chlorination system—treating water with chlorine destroyed almost all unwanted bacteria. Before 1893, typhoid rates averaged 7.3 deaths per ten thousand, and death rates were often as high as 10 to 15. After 1893, death rates never rose above 5, and shortly after the opening of the drainage canal in 1900, rates never rose above 2.5. The installation and extension of chlorination drove down typhoid rates still further until rates were hovering around 0 by the early 1920s (CBPE 1917).

It is interesting that the death rate from all causes except typhoid also appears to have responded to these water purification measures. This can be seen in figure 7, which plots the non-typhoid death from 1853 through 1925. Although the non-typhoid death rate is trending down before 1893, there appears to have been a discrete break in this trend with the installation of the four-mile intake and another break after the completion of the city's chlorination system in 1917. Perhaps the break non-typhoid deaths immediately after 1893 was driven by milk inspection and the introduction of the diphtheria antitoxin. If so, the post-1893 reduction in non-typhoid mortality should vanish once we remove diphtheria and infant mortality from non-typhoid death rates. Yet when this is done very similar patterns emerge. As figure 8 shows, there appears to have been regime shifts in the non-typhoid-diphtheria-infant death rate immediately after 1893 and again after the installation of the chlorination system in 1917, just as in figure 7.

This phenomenon was not unique to Chicago. During the late nineteenth and early twentieth century, public-health officials throughout the United States and Europe began noticing and documenting the identical patterns: death rates from a wide-variety of non-waterborne ailments, including pneumonia, tuberculosis, bronchitis, heart disease, and kidney disease seemed to improve following the installation of water purification and filtration systems (Sedgwick and MacNutt 1910). Perhaps the correlation was spurious, driven by some policy or behavioral change that was correlated with water filtration but somehow hidden to observers at the time. The problem, however, is that one cannot find any evidence in the historical record of what this hidden policy change might have been. What was it in Chicago? It clearly was not the diphtheria antitoxin or the inspection of milk for fat content. Yet it must have been something big to have affected such a broad class illnesses and diseases. Another possibility is that water filtration induced, or was at least associated with changes in personal behavior. Such behavioral changes conceivably could have affected a broad class of infectious diseases. But then one must ask, why was behavior changing at the exact moment water filtration and chlorination were initiated? Did water filtration induce behavioral changes in a multitude of cities and places? It seems a remote contingency.

III. The Mills-Reincke Phenomenon

The Discovery That Good Water Has Diffuse Health Effects

The first observers to identify the negative correlation between non-typhoid death rates and water purification were Hiram F. Mills and J.J. Reincke. Mills and Reincke worked independently and had had no contact prior to their discoveries. During the late 1880s and early 1890s, Mills worked as the chief engineer of the water company in Lawrence, Massachusetts and served as an official at the Massachusetts State Board of Health. Reincke worked as a public-health official in Hamburg, Germany around the same period. Mills documented changes in the non-typhoid death

rate following water filtration in both Lowell and Lawrence, Massachusetts around 1893. Reincke documented similar changes taking place in Hamburg, also around 1893 (Sedgwick and McNutt 1910; and Hazen 1907).

The observations of Mills and Reincke were refined and extended in two papers, one by Allen Hazen and the other by William T. Sedgwick, a professor of biology at the Massachusetts Institute of Technology. Hazen and Sedgewick explored the Mills-Reincke phenomenon in cities outside Lawrence, Lowell, and Hamburg. These cities included: Zurich, Switzerland; Albany, New York; Binghamton, New York; Watertown, New York; Newark, New Jersey; Jersey City, New Jersey; and Manchester, New Hampshire. Although Hazen and Sedgewick found that the size of the Mills-Reincke phenomenon varied from place to place, they concluded that, on average, for every one death from typhoid fever prevented by water filtration there would have been two to four deaths prevented from some other cause. Subsequent researchers documented the same phenomenon in Cincinnati, Boston, Baltimore, Pittsburgh, American military bases, and countless other cities and small towns (e.g., Fuller 1912; Friedrich 1912; McGee 1920). There were, of course, sceptics of the Mills-Reincke phenomenon but these sceptics seemed not so much to question the existence of the phenomenon, but its variability across time and space (e.g., Fink 1917). Why was it that in some cities, Mills-Reincke effects appeared very large—in New York, for example, for every one death prevented from typhoid perhaps ten or more deaths from other diseases might have been prevented—while in other cities, such as Philadelphia, the effects seemed much smaller, if there were any effects at all? Hazen and Sedgwick offered no explanations of such variability in their seminal papers on the topic.

The non-typhoid death rates that were the most responsive to improvements in water purity, at least according to Hazen and Sedwick, were the following: infantile gastroenteritis or diarrhea;

tuberculosis; pneumonia; influenza; bronchitis; heart disease; and kidney disease. There was also evidence that malaria rates seemed to respond to changes in water quality, although malaria rates were so low in cooler climates this had no discernible effect on changes in overall mortality in places like Chicago. Why did water purification have an effect on these diseases, which with the exception of gastroenteritis, were clearly non-waterborne? Some commentators during the 1910s believed that it was possible that the pathogens causing tuberculosis and pneumonia could possibly have been transferred through water (e.g., Fink 1917). They cited animal experiments showing that rats and guinea pigs injected with water tainted by the wastes of tuberculosis patients later developed the disease. Brown, Petroff, and Heise (1916) also discovered that tubercle bacilli survived in human waste and that when dumped in a river the tubercle bacilli could be found alive 3 miles downstream. At this stage of our research, however, we are not aware of any modern scientific evidence showing that humans can become infected with tuberculosis or pneumonia by drinking the water polluted by such pathogens.

In the sections that follow we identify and explain three possible channels through which the Mills-Reincke phenomenon could have functioned. The first channel flows from the difficulties of diagnosing typhoid, which during its early stages, closely resembled respiratory diseases such as pneumonia, bronchitis, and tuberculosis and was also commonly mistaken for malaria. Because of this, the more deaths there were from typhoid, the more deaths there were from the misdiagnosed cases of pneumonia, tuberculosis, malaria, and the like. The second channel builds on the observation that typhoid was an especially virulent disease that left a person vulnerable to secondary infections even if he or she survived its direct effects. Throughout the nineteenth and early twentieth century, it was common for an individual who survived the immediate effects of typhoid to succumb to pneumonia, tuberculosis, heart failure, kidney failure, meningitis, or some other

ailment within a year or two of recovering from typhoid. Because of these sequella, typhoid epidemics often had lingering effects, raising death rates from respiratory diseases, other infectious diseases, and heart and kidney failure years after the epidemic had subsided. The third channel through which the Mills-Reincke phenomenon might have worked focuses on its effects on the diarrheal death rates of infants and very young children.

Diagnosing Typhoid

People usually contracted typhoid by drinking water contaminated by the fecal wastes of typhoid sufferers. Other modes of transmissions included flies, shellfish from polluted lakes and rivers, milk, and direct person-to-person infections. Once they enter the body, typhoid bacilli had a one to three week incubation period. During incubation, an infected individual experienced mild fatigue, loss of appetite, and minor muscle aches. After incubation, the victim experienced more severe symptoms: chills, coated tongue, nose bleeds, coughing, insomnia, nausea, and diarrhea. At its early stages, typhoid's symptoms often resembled those of respiratory diseases and pneumonia was often present. In nearly all cases, typhoid victims experienced severe fever. Body temperatures could reach as high as 105º Fahrenheit. A week or so after incubation, rose-colored spots sometimes appeared on the patient's abdomen. For much of the nineteenth century, these rose-colored rashes were the only symptom doctors could use to identify typhoid definitively, but unfortunately in terms of promoting accurate diagnoses, these rashes appeared in only 5 to 20 percent of all cases (Curschmann 1901; Kiple 1993; Whipple 1908).

Three weeks after incubation, the disease was at its worst. The patient was delirious, emaciated, and often had blood-tinged stools. One in five typhoid victims experienced a gastrointestinal hemorrhage. Internal hemorrhaging resulted when typhoid perforated the intestinal wall, and frequently continued on to attack the kidneys and liver. The risk of pulmonary

complications, such as pneumonia and tuberculosis, was high at this time. Perhaps as many as two-thirds of the deaths associated with typhoid fever were not due directly to the effects of typhoid, but to tuberculosis, pneumonia, and other complications such as kidney and liver damage. The high fever associated with typhoid was so severe that about one-half of all victims experienced neuropsychiatric disorders at the peak of the disease. These disorders included encyphalopathy (brain-swelling), nervous tremors and other Parkinson-like symptoms, abnormal behavior, babbling speech, confusion, and visual hallucinations. If, however, the patient survived all of this, the fever began to fall off and a long period of recovery set in. It could take as long as four to eight months to fully recover. Surprisingly, given the severity of typhoid's symptoms, more than 90 percent of its victims survived (Curschmann 1901; Kiple 1993; Whipple 1908).

Because typhoid affected so many bodily systems and had such a broad and generic symptomology, it was easily mistaken for other diseases. Although the rise of the germ theory of disease made it possible to diagnose typhoid with greater accuracy, mistakes in diagnosis persisted well into the twentieth century. Consider, for example, the problems doctors had distinguishing between typhoid and malaria. For much of the nineteenth century, doctors often lumped together deaths associated with high fevers and diarrhea into one category referred to as "typho-malarial fever." The discoveries of scientists like William Budd (1873) and others made it clear that, while diseases like typhoid and malaria shared certain symptoms, they were in fact distinct diseases caused by two entirely different pathogens. They were not spread by the same mysterious poison floating in the air. Moreover, over the course the late nineteenth century, doctors were able to distinguish typhoid from malaria in at least some cases because of the distinctive abdominal rash that sometimes developed. By the turn of the twentieth century, doctors were able, at least in theory, to positively identify typhoid through the Widal test, a laboratory analysis of the blood or stools of ill patients.

Such diagnostic tools were predicated on the understanding that typhoid was caused by a unique bacterium (Blackburn 1898).

Nonetheless, throughout the nineteenth and early twentieth century, most deaths attributed to malaria should, in fact, have been attributed to typhoid fever. Mistaken diagnoses occurred when blood tests were not available, perhaps because the patient had already perished. In these situations, doctors had to rely on more primitive, and less accurate, diagnostic techniques. If one explores how malaria rates in Chicago responded to water purification measures, the tendency to confuse typhoid with malaria is amply demonstrated. Figure 9 plots the death rate from malaria in Chicago from 1884 through 1914. Before the water purification measures in 1893, malaria death rates in Chicago hovered between 1.2 and 1.6; after 1893, these rates plummeted, falling to less .4 within a few years. The City of Chicago implemented no measures designed to combat malaria during this period, and the pattern observed in Chicago is repeated over and over again in other American cities: installing water filters and chlorination induced immediate and large reductions in malaria rates (Troesken 2004, pp. 171-78). Unless one believes that water filtration killed lots of mosquitos, there is no explanation for this pervasive and widespread pattern other than the tendency to confuse typhoid with malaria.

Nineteenth and early twentieth century physicians also had great difficulty distinguishing typhoid from pneumonia and tuberculosis. Hobart Emery Hare, a physician writing during the early 1900s, described the case of a patient who entered the hospital with a severe cough and labored breathing, bloody sputum, a high pulse rate, along with a 102 degree fever. Because these were all classic symptoms of pneumonia, "a diagnosis of croupuos pneumonia was made (Hare and Beardsley 1909, p. 54)." Not until the tenth day of the patient's illness did symptoms more suggestive of typhoid begin to appear: rose-colored abdominal spots; blood-tinged stools; and

persistent diarrhea. Hare went to explain that:

> The difficulty in diagnosticating [sic] these cases lies in the distinctly local manifestations and the fact that in some patients the fever may be quite high, delirium of an active form may be marked, and every symptom pointing to intestinal typhoid lesions may be absent. The question naturally arises as to the frequency with which this form of enteric fever occurs, but statistics concerning it are difficult to collect, since in many instances the condition is never recognized, or is recognized very late, and is not by any means always reported.

And again later in his treatise on typhoid, Hare (p. 105) wrote:

> When the pneumonia ushers in the attack of typhoid fever the symptoms of pneumonitis so mask those produced by the bacillus typhosus that a diagnosis of pneumonia is often made and maintained until some characteristic sign of typhoid fever, such as hemorrhage from the bowel or the appearance of specific eruption, reveals the true nature of the illness. Sometimes the diagnosis is not made except at autopsy.

Although the extant historical record contains no direct evidence on the frequency with which deaths from typhoid were mistakenly ascribed to pneumonia and other respiratory diseases, it is possible to construct indirect estimates. By exploring the correlation between typhoid death rates and death rates from respiratory diseases over time, one can draw inferences about the frequency of missed diagnoses.

Typhoid Fever

The fact that typhoid killed only 5 to 10 percent of its victims might lead one to wonder just how significant this disease could have been for human health and longevity. But typhoid's low case fatality rate understates the disease's true impact, because when typhoid did not kill you quickly and directly, it killed you slowly and indirectly. The evidence presented below shows that typhoid survivors faced an elevated risk of dying from heart failure, respiratory problems, neurological problems, and liver and kidney failure in the years following their recovery. If one considers the severity of typhoid's symptoms, which ranged from hallucinations to acute renal failure, it is not surprising that exposure to typhoid undermined a person's long-term health prospects even if they

survived the immediate effects of the disease.

Typhoid affected nearly all bodily systems, and it left many of them permanently damaged. As for the cardiovascular system, Khosla (1981) reports that 12 percent of typhoid patients studied during the mid-twentieth century experienced some sort of cardiac event while fighting off the disease. Using a sample of nearly 30,000 Union Army recruits, Costa (2000) shows that, holding everything else constant, those recruits who fell victim to typhoid during the Civil War, had elevated rates of heart disease and heart troubles later in life. In particular, recruits who fell victim to typhoid had about a 25 percent higher risk of heart trouble later in life than those who had not experienced typhoid. As for typhoid's impact on respiratory function, probably the country's foremost expert on typhoid at the turn of the century, Whipple (1908, p. 6) wrote: "It is said that not over a third of the deaths from typhoid fever are due directly to the effects of the disease, i.e., to the effects of the typho-toxin. Two-thirds of the deaths are due to the numerous complications, among which tuberculosis and pneumonia are prominent." Elsewhere, Whipple (1908, p. 3) explained that the risk of contracting pulmonary complications such as tuberculosis and pneumonia was especially high when typhoid was at its peak, around the second or third week of the illness. There is systematic evidence to support such beliefs.

In a recent paper with the apt title, "The Liver in Typhoid Fever: Always Affected, Not Just a Complication", Morgenstern and Hayes (1991) review the evidence documenting typhoid's effect on the liver. According to Morgenstern and Hayes, 90 percent of all typhoid cases had some degree of liver involvement, ranging from biochemical abnormalities to overt hepatitis—about 5 percent of all typhoid victims developed hepatitis. Kidney problems were also common, ranging from swelling and high protein levels in the urine, to full-blown nephritis. Studies of current populations suggest that at least 10 to 15 percent of typhoid victims would have experienced a significant abnormality in

kidney function (Khosla and Lochan 1992; Abu et al. 1975). In one study performed during the 1990s, 79 percent of all typhoid victims experienced symptoms consistent with urinary tract infections (Mathal et al. 1995). Studies from the early twentieth century found that acute kidney failure followed in between 1 and 3 percent of all typhoid cases, and that of these, 30 to 60 percent of the patients eventually died from kidney failure (Hare and Beardsely 1909, pp. 115-18). Today, complications with the kidney and liver can be managed through dialysis and drug treatment, limiting the long-term damage to these vital organs. However, among historical populations, typhoid was allowed to run its course and it seems likely that the disease would have left the kidneys and liver irreparably compromised for a large portion of the population that survived the short term effects of the disease.

Studies of modern-day populations suggest that between 35 to 85 percent of all typhoid victims would have experienced some sort of neurological complications. These complications would have included severe confusion (30 to 70 percent of all cases), some type of swelling or inflamation of the brain or spinal chord (5 to 10 percent of all cases), and acute schizophrenia or psychosis (1 to 2 percent of all cases).[7] Historical authors made frequent reference to the restlessness, insomnia, melancholy, and hysteria associated with typhoid fever, and presented evidence that typhoid sometimes induced permanent insanity. Autopsies of people who have been killed by typhoid reveal diffuse damage to neurons, softening of the brain's vascular system, the formation of abscesses on the brain, and discharges of pus in the brain and meninges. Even for those who survived typhoid, though, all of these complications left scares and lesions. In turn of the century America, several studies found evidence that meningitis was a complicating factor in about one of every ten typhoid deaths (Hare and Beardsely 1909, pp. 158-73).

[7]See Ali et al. (1997) for the neuropsychiatric pathologies associated with typhoid fever.

What were the net effects of all these complications and sequella on the future health prospects of typhoid survivors, and more importantly, what implications does this have for the Mills-Reincke phenomenon? A study conducted by Louis I. Dublin, a statistician for the Metropolitan Life Insurance Company, helps answer these questions. Dublin (1915) followed 1,574 typhoid survivors over a three year period using life-insurance records and published his results in the *American Journal of Public Health*. Comparing the mortality rates of typhoid survivors to the mortality rates of similarly-aged persons who had never suffered from typhoid, he found that during the first year after recovery, typhoid survivors were, on average, 3 times more likely to have died than those who had never been exposed to typhoid, and that in the second year after recovery, typhoid survivors were 2 times more likely to have died than non-typhoid survivors. By the third year after recovery, however, typhoid survivors did not face an elevated risk of mortality. The two biggest killers of the typhoid survivors were tuberculosis (39 percent of all deaths) and heart failure (23 percent). Other prominent killers included kidney failure (8 percent) and pneumonia (7 percent).

Because typhoid had such large and general effects on human health, it seems plausible that public health measures aimed at eradicating this disease would have affected mortality from a much broader class of diseases and illnesses, including respiratory diseases, heart failure, and kidney failure. Testing for such a phenomena would be a straightforward exercise. Suppose that one compiled a time series of death rates from these various causes and regressed the current death from tuberculosis, pneumonia, or the like, against lagged values of the typhoid rate. The discussion above suggests that lagged typhoid rates would have raised death rates from these other diseases, holding everything else constant.

Water Purity and Infantile Diarrhea

The waterborne disease that killed the most people, of any age, was diarrhea. Diarrheal

deaths occurred primarily among children less than two years of age, and diarrheal illness was probably the leading cause of death for the very young. For example, a study of infant mortality in Baltimore, Maryland, conducted in 1915 found that 43 percent of all infant deaths were the result of diarrhea (Troesken 2004, pp. 27-8; Woodbury 1926). Exactly how many of these diarrheal deaths were the result of impure water is unclear. At the time, many public health experts believed that impure milk was a major cause of infantile diarrhea. The basis for this belief was that breast-fed infants had significantly lower mortality rates from diarrhea than milk-fed infants. On the other hand, some of the infantile deaths attributed to diarrhea were almost certainly typhoid fever, but because of the difficulties of diagnosing typhoid at the time, particularly among the very young, these deaths were mistakenly attributed to diarrhea. Moreover, typhoid was sometimes transmitted through impure milk, and the correlation between breast-feeding and low infant mortality might partly reflect the fact that children were more likely to have been exposed to typhoid through cow's milk than through breast's milk. Lastly, the correlation between breast-feeding and infant health is not limited to diarrhea. Infants who were breast feed also had significantly lower non-diarrheal death rates than infants who were milk fed (Troesken 2004, pp. 23-30).

Today, there is a voluminous literature on the determinants of infant mortality. This literature suggests that among both modern-day and historical populations the introduction of public water and sewer lines has reduced infant deaths from diarrheal diseases, but that the efficacy of these interventions has varied across time and space. For example, exploiting a sample of nearly ten thousand women from urban Brazil during the 1970s, Merrick (1985) finds that access to piped water explained about 20 percent of the variation in infant deaths. Merrick, however, finds that familial characteristics such as maternal literacy had a larger effect. Using household level data from multiple countries in Africa, Asia, and the Americas during the 1980s, Esrey (1996) finds that access

to clean water and sanitary sewers resulted in significantly lower infant deaths and increased height and body weight for infants, adding perhaps as much as one to two kilograms to an infant's weight. Studying German cities between 1890 and 1910, Brown (1989) finds that about 40 percent of the reduction in childhood mortality that occurred over this period can be attributed to improvements in public water and sewer systems.

Although literature cited above indicates that there would have been strong a correlation between water quality and infant health outcomes in Chicago, it does not explain why that correlation should have existed. Indeed, one wonders how bad water ever made it to the mouths of infants. In nineteenth-century Chicago, children under one or two years of age would have consumed little water, and instead consumed mostly breast's milk or cow's milk. Furthermore, even if very small children regularly consumed water, those amounts would have been small and easily purified through boiling or the parents could have purchased bottled water. Put another way, it was easy for parents to adopt low-cost and highly effective health practices to prevent bad water from affecting their infants and young children. One way to resolve this puzzle is to appeal to the slow diffusion of the germ theory disease: most nineteenth-century parents did not know about the benefits boiling water and breast-feeding (Ewbank and Preston 1990). We think this answer is incomplete and obscures the relationship between pure water and infant mortality.

We maintain that a sizeable fraction of all the infant deaths were really typhoid, but given the virulence of typhoid, the infants perished before the disease could have ever been diagnosed. We further maintain that it was not easy or low-cost, in any sense, for parents to prevent typhoid-contaminated water or feces from reaching the mouths and stomachs of their children. What do we adduce to support such claims? As shown below, the extraordinary resilience of the typhoid bacillus allowed it to survive and proliferate despite the best efforts of even the most vigilant and health-

conscious parents.

Typhoid bacteria could live in ice, frozen solid for several months. Though sunlight eventually killed them, they could withstand heat up to 160º Fahrenheit. Dried on inanimate objects–clothes, knives, forks, and so on–they survived for several months. In dust, sand, and dirt, the bacilli lived for several weeks. There were cases where individuals were infected with typhoid after consuming fruits and vegetables that were merely sprayed with typhoid-contaminated water. Even washing tainted glasses or silverware with alcohol did not always kill typhoid. Now consider that in 1910, the typical American household consumed 20-40 gallons of water per day. Although only a fraction of this water was used for cooking and drinking, the resiliency of typhoid meant that families would have needed to boil all of their water, whatever its use, to have been completely safe from the disease. In this context, imagine how difficult it would have been to maintain clean and safe plates and eating utensils for young children. A bottle or plate washed with soapy tap water might have remained safe if a heavy layer of soap remained; but rinsing with contaminated tap water only would have reintroduced typhoid bacteria onto these objects (Troesken 2004, p. 51-2).

A typhoid epidemic that occurred in Plymouth, Pennsylvania at the turn of the twentieth century serves to illustrate of the resiliency of typhoid bacteria, as well as their ability to proliferate rapidly in even fairly hostile environments. Plymouth was located in a mountainous area in eastern Pennsylvania and drew its water from a small lake located in the mountains north of the city. There was one home on the shore of the lake, and other than this home, the lake was completely isolated from other potential sources of pollution. One winter, an adult male from this home traveled to New York City, and unbeknownst to him, contracted typhoid. Upon his return in early February, the family disposed of one of his evacuations on the frozen and snow-covered ground on the shores of the lake. Soon thereafter he was diagnosed with typhoid, and his family was ordered by local

health officials to dispose of all subsequent waste only after disinfecting it with acid and by throwing it in the woods far away from the lake. According to all available accounts, the family followed the requests of local health officials. Almost two months later, the temperature warmed, the ground thawed, and there were heavy rains. A small pile of contaminated feces, frozen solid for nearly two months, was then washed into the lake and ultimately resulted in one of the worst typhoid epidemics in American history (Whipple 1908, pp. 24-7).

Skeptical observers might argue that, even in the absence of public water filtration, parents could have secured pure water at low cost by adopting the one of the following three practices not yet considered: they could have tested their water for bacterial contamination, and only in those cases where bacteria counts where recorded, need they have been extra vigilant in terms of boiling their water and washing their hands; they could have purchased bottled water; and they could have installed household filters on their taps. Several factors, however, limited the efficacy of such practices. First, it was costly for individual households to test their water for contamination. It required a scientist trained in bacteriology to identify intestinal bacteria in water. Second, bottled water was much more expensive than tap water. In 1920, tap water from public systems sold at .015 to .025 cents per gallon, while bottled water sold for 8 to 10 cents per gallon, or in other words, three to seven hundred times the price of tap water (Troesken 2004, pp. 51-3).

Third, while household filters were effective in removing large mineral deposits from water and in providing an element of water softening, they were not effective in eliminating bacteria from water, and in some cases, could actually cause bacteria to proliferate. Most household filters attached to kitchen faucets and contained a porous stone. When the homeowner turned on the faucet, water flowed through the pores of the stone. In theory, this process was supposed to filter out objectionable material, including bacteria. Unfortunately, the pores of even the most finely-

pored stones were many times larger than even the largest bacteria, and most bacteria went through the filter untouched. Furthermore, those bacteria that did not get through often lodged themselves on the interior walls of the stone, where they stayed untouched by any efforts to clean the stone with a brush. These tiny, interior pores were an ideal breeding ground for bacteria, and once inside, bacteria proliferated. Consider the description of an experiment conducted by the Florida Board of Health (Troesken 2004, 52-3):

> During the past two years a number of determinations, to ascertain the merits of the household filter, have been made. A popular type of filter was tested. Jacksonville tap water with an initial bacterial count of 35 per cubic centimeter was filtered through one of the porous stone filters; filtered water had a count of 950 in one instance and over a million in another.

IV. The Mills-Reincke Phenomenon in Chicago: Econometric Evidence

Estimating Strategies

In this section, we use simple econometric techniques to estimate the size and sources of the Mills-Reincke phenomenon in Chicago. We begin by estimating variants of the following regression equation:

$$(1) \qquad y_t = \$_0 + \$_1 x_{t-k} + \$_2 y_{t-1} + \$_3 Year + \,,_t,$$

where y_t is the death rate from a disease, or class of diseases, other than typhoid fever in year t in the City of Chicago; x_t is the death rate from typhoid fever in year t-k (k = 0,1,2); y_{t-1} is the dependent variable lagged one year; *Year* is a time trend; and , is an error term. As for the structure of the error term, we estimate Newey-West standard errors that allow for autocorrelation up to three periods. This model is estimated with city-level data that extends from 1853 through 1925. The data come from the *Annual Reports of the Chicago Department of Health.* Here, as in the rest of the paper, death rates are in terms of deaths per ten thousand persons.

With this rudimentary model we can address a number questions about the Mills-Reincke

phenomenon in Chicago. In particular, the discussion above suggests that an epidemic of typhoid fever in year t would have resulted in higher death rates from pneumonia and tuberculosis in year t+1 because those typhoid victims who had survived the epidemic would have been compromised and vulnerable to secondary infections. Similarly, because typhoid adversely affected kidney and heart function, unusually high typhoid rates in year t would imply higher deaths from kidney and heart failure in year t+1. By letting *y* equal the death rate from all causes other than typhoid, we are able to estimate the size of the Mills Reincke phenomenon in Chicago, and by estimating how the relationship between non-typhoid deaths and typhoid changes over time we are able to better isolate the sources of the Mills-Reincke phenomenon. For example, if the Mills-Reincke phenomenon was driven by the fact that doctors in Chicago consistently diagnosed typhoid as pneumonia, one would expect that the phenomenon would have been much larger between 1860 and 1880 than it was between, say, 1900 and 1920, largely because the ability to accurately diagnose typhoid improved dramatically between 1880 and 1900 with the development of Widal tests (Blackburn 1898).

In the second part of our estimation, we ask if the Mills-Reincke phenomenon was unique to typhoid or if other diseases shared similar characteristics. Specifically, we estimate the model above using data on infectious diseases such as scarlet fever and diphtheria, and ask whether unusually large outbreaks of scarlet fever and diphtheria were associated with higher death rates from non-scarlet-fever or non-diphtheria causes. In as much as the advocates of the Mills-Reincke phenomenon used this construct to justify massive expenditures on public water and sewer systems, one would like to know if typhoid were a truly extraordinary disease in terms of generating deaths from secondary causes. If other infectious diseases generated a similar phenomenon, the case for singling-out typhoid for eradication would be undermined. Also, estimating a Mills-Reincke relationship for non-typhoid diseases like scarlet fever and diphtheria will provide a check regarding

the reliability of our model: if all diseases exhibit a strong Mills-Reincke effect, this would suggest that our estimates for typhoid are picking up some unobservable shock, unrelated to the secondary effects of typhoid.

A concern with the estimation strategies discussed thus far is that they rely on city-level time series data for Chicago alone. Because there exists only a single time series, it is much harder to control for shocks and events than it would be with time series data on multiple units of analysis. It would, therefore, be desirable to have some sort of panel-data estimation. To this end, we have assembled time series data on typhoid death rates for all thirty-four of Chicago's wards during the late-nineteenth century. With the panel data in hand, we are able to estimate the size of the Mills-Reincke phenomenon in Chicago after controlling for mortality shocks common to all city wards. In the panel-data analysis, we estimate variants on the following equation:

$$(2) \qquad Z_{it} = \alpha + \beta + \gamma T_{it} + \varepsilon_{it},$$

where, Z_{it} is the death rate from all causes except typhoid fever in ward i (i = 1-34) in year t (t = 1891-1894, 1896-1900); α is a vector ward dummies; β is a vector of year dummies; T_{it} is the death from typhoid fever in ward i in year t; and ε_{it} is a random error term.

Results of the City-Level Time-Series Analysis

Table 2 reports the results of city-level time-series regressions where the non-typhoid death rate is regressed the typhoid death rate. A lag of the dependent variable is included as a regressor in all specifications and controls unobservable health events unrelated to typhoid and its sequella. Also four dummy variables for epidemic years, such as the influenza epidemic in 1918, are included in all of the specifications; the results do not change dramatically if these dummies are excluded. The models are estimated over three periods: 1853-1925; before 1880; and after 1867. We explore the effects before 1880 to see if the Mills-Reincke effects were larger during earlier periods, which would

be suggestive of rampant mistaken typhoid diagnoses on the part of Chicago doctors. We restrict the sample to years after 1867 to make the results comparable to later regressions using other non-typhoid diseases. (Data from other causes of death are often unavailable before 1867.)

The results for the full sample support the idea that there was some sort of Mills-Reincke effect at work in Chicago. If one looks at the coefficient on contemporaneous typhoid rates, for every one additional death from typhoid fever there were 9.9 deaths from other causes for the entire period (regression 1); 15.8 deaths in the pre-1880 period (regression 3); and 4.4 deaths in the post-1867 period (regression 5). The relatively large effects for the pre-1880 period are consistent with the hypothesis that the Mills-Reincke phenomenon was related, in part, to doctors mistakenly attributing deaths from typhoid to other causes, as we would expect such mistakes to have been much more common before 1880 than after. If one looks at the coefficient on the typhoid rate lagged by one year, the estimated Mills-Reincke effects do not appear to have been as large as for the contemporaneous rate, but they are still substantial. For every one additional death from typhoid fever in the previous year, there were 4.3 deaths from other causes for the entire period (regression 2); 7.3 deaths in the pre-1880 period (regression 4); and 4.0 deaths in the post-1867 period (regression 6). These results are suggestive that typhoid had large secondary effects, leaving its survivors vulnerable to subsequent infections from pneumonia, tuberculosis, or heart and kidney failure.

Table 3 reports the results of regressions in which we explore typhoid's effects on the death rate from other specific diseases. These results suggest that for every one death reported from typhoid fever there was one additional death among infants and perhaps among children aged one to four. There is no evidence that typhoid affected deaths from heart failure in Chicago, but there is a significant relationship between the contemporaneous typhoid death rate and deaths from kidney

failure. This result suggests that for one additional death from typhoid there were .8 additional deaths from kidney failure. The results are consistent with the qualitative evidence presented in section III about the difficulties in distinguishing typhoid from respiratory diseases and about the likelihood of typhoid leaving persons vulnerable to secondary infections from tuberculosis and pneumonia. For every one additional death from typhoid fever, there were 1 to 1.5 additional deaths from tuberculosis and pneumonia.

Table 4 explores the possibility that infectious diseases other than typhoid also exhibited Mills-Reincke effects. If we find that Mills-Reincke effects were not limited to typhoid, but were quite general, this would undermine the case that typhoid had some unique and diffuse effect on overall human mortality. We have gathered data on all disease specific death rates available for the City of Chicago and report results for all of these diseases. The results in table 4 suggest that typhoid was a special kind of killer. The death rate from pneumonia is not correlated with the death rate from non-pneumonia causes; the death rate from bronchitis is not correlated with the death rate from non-bronchitis causes; the death rate from whooping cough is not correlated with the death rate from non-whooping cough causes; and the death rate from diphtheria is not correlated with the death rate from non-diphtheria causes. For two diseases—influenza and scarlet fever—there is evidence of a reverse Mills-Reincke effect: influenza and scarlet fever appear to have been killing off the weakest and most vulnerable parts of the population so that high death rates from these diseases actually reduced death rates from other causes.

There are two diseases, however, that exhibit similar patterns to typhoid: measles and tuberculosis. Although the coefficient on measles in statistically insignificant, the point estimate suggests that for one additional death from measles there were two additional deaths from non-measles causes. The coefficient on tuberculosis is significant and suggests that for every one

additional death from tuberculosis there were two to three additional deaths from non-tuberculosis causes. These coefficients are about one-half the size of those estimated for typhoid fever. Furthermore, the coefficient on typhoid was robust to using the lagged rather than contemporaneous death rate. This is not true of tuberculosis. As table 5 shows, when the lagged tuberculosis death rate is used, the coefficient falls below zero and is statistically insignificant. The result for measles is stronger though its implied effects are still much smaller those estimated for typhoid. As shown in table 1, during the late nineteenth century, the death rate from typhoid fever exceeded the death rate from measles by a factor of 3.4. Furthermore, when we regress the death rate from all causes except typhoid and measles, against the death rate for measles and the death rate for typhoid fever, the estimated secondary effects of typhoid appear far larger than those for measles. As table 5 shows, the estimated coefficient on typhoid is from 33 to 100 percent larger than the estimated coefficient on measles.

In the final part of our empirical analysis of the Mills-Reincke phenomenon, we ask whether the estimated effects of typhoid on non-typhoid death rates withstands a panel data analysis. Our estimating strategy was described above in equation (2). The results presented in table 6 suggest that, for the most part, they do. The first two regressions highlight the effects of adding time dummies and controlling for unobserved health shocks. The effect appears quite small, with the coefficient on contemporaneous typhoid rates falling by .22 points (4 percent) after adding time dummies to the regression. Compare regressions (1) and (2). Using the lagged typhoid rate rather than the contemporaneous typhoid rate does not dramatically alter the findings. Overall the estimates from regressions (1) through (3) are consistent with the time series analysis, and imply that for every one additional death from typhoid there were four to five additional deaths from non-typhoid causes. However, if we add a lag of the non-typhoid death rate, the estimated Mills-Reincke

effect falls to 2.3 deaths when contemporaneous typhoid rates are used, and to zero when lagged typhoid rates are used. This last finding is the only piece of evidence we can find that is inconsistent with the Mills-Reincke phenomenon.

Implications for Typhoid's Role in Chicago's Mortality Transition

In table 7, we calculate the proportion of all deaths ultimately attributable to typhoid fever based on the estimates presented above. The largest estimate of Mills-Reincke effect is 15 but usually our estimates fall between 4 and 7. To be clear, these estimates suggest that for every one additional death from typhoid fever there four to seven deaths from other causes that occurred either because there were cases of typhoid being mistakenly diagnosed as pneumonia, diarrhea, or tuberculosis, or because typhoid left its many survivors vulnerable to secondary infections and kidney failure. Assuming a Mills-Reincke factor of 7, about 27 percent of all deaths during the 1870s (.204 + .062) would have been attributable to typhoid fever, its sequella, or the under- diagnoses of typhoid fever. In this scenario, eradicating typhoid would have reduced the overall death rate by 49 points which represents about 56 percent of the total decline in mortality observed in Chicago. Assuming a Mills-Reincke factor of 4, about 15 percent of all deaths during the 1870s would have been attributable to typhoid fever, its sequella, or the under- diagnoses of typhoid fever. In this scenario, eradicating typhoid would have reduced the overall death rate by 30 points which represents about 35 percent of the total decline in mortality observed in Chicago. To the extent that the purification of Chicago's water supply was largely responsible for the eradication of typhoid in the city, this suggests that pure water played a significant role in promoting Chicago's transition from high to low mortality.

V. Mortality, Waters, and Sewers in 1880: Household-Level Evidence

Although Chicago's mortality transition was influenced by the city's water system in the last decades of the nineteenth century, substantial positive effects are concentrated in the years after 1893. Mortality from typhoid and its sequella remained high even in the 1880s when a significant fraction of the city already had access to piped city water. The 1880 federal census provides a unique opportunity to assess the impact of water and sewer access at the household level, allowing us to investigate the effects of these major infrastructure projects before the city's mortality transition. In addition, this analysis of the effects of access to city water before public purification measures will help make a broader methodological criticism of a literature in demography and development economics which uses piped water as a proxy of pure water.

Beginning in 1850, as part of the regular decennial census, information on mortality in the twelve months preceding the census was acquired from each household enumerated in the population schedules. At each household they visited, census marshals asked whether any deaths had occurred in the last year. Responses (including the decedent's name, age, sex, race, marital status, occupation, birthplace, and cause and month of death) were included on a "Mortality Schedule" separate from the "Population Schedule." In 1870 and 1880, the census marshals identified each death with the household number in the population schedule that reported the death. In 1880, the population schedule for the first time included the exact street address of each household. Together with water and sewer maps for Chicago in 1880, the combination of the mortality and population schedules allows us to create a dataset of decedents and survivors with a wealth of information on each individual's demographic and socioeconomic characteristics, and whether they lived in a dwelling with access to the city's water and sewer systems.

We have collected data for, and performed analysis on 12 of the city's enumeration districts

(each of roughly 2,500 persons) shown in figure 10. The districts were chosen to represent diverse economic and geographic traits. For example, the Loop (district 2) was primarily commercial, the lakefront (districts 13 and 23) was affluent, and the neighborhoods along the South Branch of the river (8, 9, 10, 49, 50, 51) had more unskilled workers, blacks, immigrants, and children. Several characteristics of the enumeration districts are shown in table 8. The sample contains 29,665 individuals (299 decedents). Unfortunately, there is a great deal of overlap between the water and sewer systems in these areas: only 1,248 individuals resided on streets where one was present but not the other (table 9). It will thus be difficult to disentangle their separate effects until additional enumeration districts are added. Though cause of death was recorded, the number of decedents presently included was too small to allow any use to be made of this information.

The effects of water and sewer access are analyzed in tables 10 and 11. With controls only for age and access to the water and sewer systems, widespread benefits of public infrastructure are not immediately apparent. Mortality is lower with water or sewer access only for those over age 45, and perhaps for children age 1 to 4 for the sewer system. For infants, the effect of either water or sewer access is substantial and perverse: access actually raised mortality by more than 50 per thousand from a predicted value of 80 per thousand. When both water and sewer access are entered simultaneously, young children experience the most striking effects: sewer access lowers mortality and water access raises it. When additional controls for individual and dwelling characteristics are included in table 11, this latter result remains with the sewer and water effects almost exactly balancing. Separate analyses (not shown) with the full set of controls used in table 11 but water and sewer access entered in separate regressions duplicated the finding for infants: either water or sewer access was associated with higher mortality, while either was associated with lower mortality for adults over age 45.

The finding that access to public water lines in 1880 produced higher mortality is surprising and significant. One possible explanation is that the city chose which districts to serve based on the disease rates in that district—the districts with the highest disease rates got served first. This possibility, however, is ruled out by the logic that guided the system's construction. Water had to flow from a fixed source (the lake), and sewers had to flow to fixed destinations (the lake and the river). Neighborhoods were then connected in a system radiating out from these points. A more likely possibility is that the city's water before 1893 was impure (contemporaries noted that small fish would sometimes emerge from the tap), particularly in relation to other water sources (e.g., wells and bottled water). In any case, these results call into question any close correspondence between access to water or sewers and universally lower mortality before 1893. Moreover, these results highlight the danger of equating pure water with piped water, as is often done in the development literature (e.g., Lee et al. 1997 and Merrick 1985). The results here suggest that this is a questionable practice. Piped water alone does not reduce mortality; the water must also be filtered and purified to prevent disease.

VI. Conclusions

The central findings of this paper are twofold. First, pure water had diffuse health benefits. It not only reduced the death rate from diseases that were directly waterborne such as typhoid and diarrhea. It also reduced the death rate from diseases that other would not have been classified as waterborne or even water-related, such as influenza, pneumonia, heart disease, and tuberculosis. Water purification had diffuse health benefits because typhoid fever, a disease spread mainly through impure water, was a special kind of killer. Typhoid did not kill most of its victims quickly and directly—the disease itself had a case fatality rate of only 5 to 10 percent. Rather most people survived the bout(s) with typhoid, but only by the skin of their teeth. The typical typhoid survivor

was so weakened and compromised by the disease that he or she would later succumb to some other infectious disease like tuberculosis, or die of kidney or heart failure. As a result, when the City of Chicago began taking steps to assure that its water supply was free from disease, death rates from all sorts of non-waterborne diseases fell along with typhoid.

The paper's second central finding flows directly from the first. Because water purification had diffuse health effects, Chicago's mortality transition was driven primarily by improvements in the city's water supply, particularly those efforts to move water intakes far from sewage outflows. The empirical estimates suggest that between 35 and 56 percent of the mortality decline that occurred in Chicago between 1870 and 1925 was related to water purification and the associated eradication of typhoid fever. Relatedly, in the final analytical section of the paper, we showed that one should not equate pure water with piped water as is commonly done in the relevant economic and demographic literatures. Before the City of Chicago began separating sewage from water intakes in 1893, access to the city water did not improve health outcomes. On the contrary, it appears that access to piped-city water during this period was associated with higher mortality.

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Table 1

	1870-1879		1915-1925 (excluding 1917)	
	Death rate	Share of all deaths	Death rate	Share of all deaths
Deaths by cause:				
All causes	211.8	1.000	125.3	1.000
Typhoid fever	6.2	.029	.2	.002
Measles	1.8	.009	.6	.005
Scarlet Fever	7.8	.037	.5	.004
Whooping Cough	2.8	.013	.5	.004
Diphtheria	11.2	.053	2.2	.018
Respiratory diseases	36.7	.173	27.2	.217
Heart disease	5.1	.024	18.3	.146
Kidney disease	1.6	.008	9.1	.071
Cancer	1.4	.014	9.6	.078
Deaths by age-group:				
Infant mortality (< 1)	74.0	.349	18.3	.162
Child mortality (1-4)	48.1	.227	8.9	.073

Table 2

	1855-1925		Before 1880		After 1867	
	(1)	(2)	(3)	(4)	(5)	(6)
Non-typhoid$_{t-1}$	.019	-.026	-.067	-.131*	.380*	.202*
	(.090)	(.055)	(.136)	(.090)	(.088)	(.137)
Typhoid$_{t}$	9.90*	. . .	15.8*	. . .	4.37*	. . .
	(4.39)		(6.42)		(.905)	
Typhoid$_{t-1}$	1.65*	4.34*	. . .	7.30*	. . .	4.00*
	(.435)	(1.12)		(2.15)		(1.58)
Year	-1.11*	-1.31*	-4.00*	-1.78*	-.569*	-.945*
	(.527)	(.295)	(2.07)	(.653)	(.197)	(.301)
F-statistic	59.8†	48.8†	3.43*	7.20*	299.8†	154.4†
N	76	76	28	28	58	58

Table 3

	Infant (0-1)		Child (1-4)		Heart		Kidney		Respiratory	
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)
$Depvar_{t-1}$	.594*	.515*	.147	.143	.674*	.670*	.147	.143	.519*	.519*
	(.115)	(.137)	(.217)	(.194)	(.147)	(.145)	(.217)	(.194)	(.181)	(.193)
$Typhoid_t$	1.65*	. . .	-.334	. . .	.003	. . .	-.334	. . .	1.16*	. . .
	(.435)		(.796)		(.034)		(.796)		(.276)	
$Typhoid_{t-1}$	. . .	1.09*	. . .	1.01*	. . .	-.043	. . .	1.01*	. . .	.714
		(.498)		(.405)		(.043)		(.405)		(.632)
Year	-.227*	-.409*	-.847	-.644	.095*	.089*	-.847	-.644	.036	-.027*
	(.142)	(.184)	(.250)	(.178)	(.038)	(.039)	(.250)	(.178)	(.076)	(.103)
F-statistic	215.3†	52.1	76.9	26.6	73.5†	27.5	40.7	31.9	93.8†	93.1
N	58	57	58	57	58	57	58	57	58	57

Table 4

	Dependent variable: death rate from all causes except *Y*, where *Y* equals							
	Pneumonia	Influenza	Bronchitis	Whooping Cough	Scarlet Fever	Diphtheria	Tuberculosis	Measles
Y_t	.539	-.886	.326	.105	-.840*	.042	2.04*	2.52
	(.594)	(.734)	(1.13)	(3.82)	(.443)	(.388)	(1.15)	(2.76)
NoY_{t-1}	.535*	.487*	.463*	.458*	.466*	.514*	.436*	.407*
	(.120)	(.118)	(.100)	(.121)	(.121)	(.125)	(.102)	(.092)
Year	-1.09*	-1.05*	-1.04*	-1.12	-1.18*	-.889*	-.575*	-1.11*
	(.311)	(.332)	(.333)	(.209)	(.343)	(.402)	(.251)	(.342)
F-statistic	140.5	105.3	177.3	214.1	207.0	187.4	159.4	244.4
N	58	58	58	58	58	58	58	58

Table 5

	Tuberculosis		Measles	
	NoTb	*NoTbTy*	*NoMeasTy*	*NoMeasTy*
$Allother_{t-1}$	.457*	.371*	.233*	.144
	(.119)	(.088)	(.088)	(.146)
$Typhoid_{t}$	. . .	3.93*	4.33*	. . .
		(.731)	(1.08)	
$Typhoid_{t-1}$	. . .	. . .	. . .	4.17*
				(1.47)
Tb_{t}	. . .	1.19*	. . .	. . .
		(.577)		
Tb_{t-1}	-.074	. . .	. . .	. . .
	(.577)			
$Measles_{t}$	. . .	. . .	2.21*	. . .
			(1.70)	
$Measles_{t-1}$	. . .	. . .	. . .	3.51*
				(3.04)
Year	-1.04*	-.219	-.513*	-.823*
	(.271)	(.234)	(.188)	(.222)
F-statistic	190.8	284.1	278.6	215.3
N	58	58	58	58

Table 6

	Death rate from all causes, except typhoid				
	(1)	(2)	(3)	(4)	(5)
Non-typhoid$_{t-1}$	. . .	. . .	. . .	.884*	.946*
				(.050)	(.049)
Typhoid$_{t}$	5.22*	5.00*	. . .	2.32*	. . .
	(.405)	(.697)		(.909)	
Typhoid$_{t-1}$	. . .	. . .	3.90*	. . .	-.015
			(.697)		(.487)
Ward dummies	yes	yes	yes	yes	yes
Year dummies	no	yes	yes	yes	yes
Adjusted-R2	.462	.497	.497	.765	.759
N	306	306	306	272	272

Table 7

	1870-79	Additional typhoid deaths, 1870-79	1915-25	Change	Attributable to typhoid
Death rate from all causes	211.8		125.3	86.5	
Death rate from typhoid	6.2	6.2 (.029)	.2	6.0	.069
Mills-Reincke factor, 1870-1879					
15		93 (.439)	.8	92.2	1.066
9		55.8 (.263)	.8	55.0	0.636
7		43.4 (.205)	.8	42.6	0.492
4		24.8 (.117)	.8	24.0	0.277
3		18.6 (.089)	.8	17.8	0.206

Note that for the 1915-1925 period, the year of the influenza epidemic is excluded and that we assume a Mills-Reincke factor of 4, which seems quite generous given that by this time the ability to diagnose typhoid would have been far superior than it had been during the late nineteenth century.

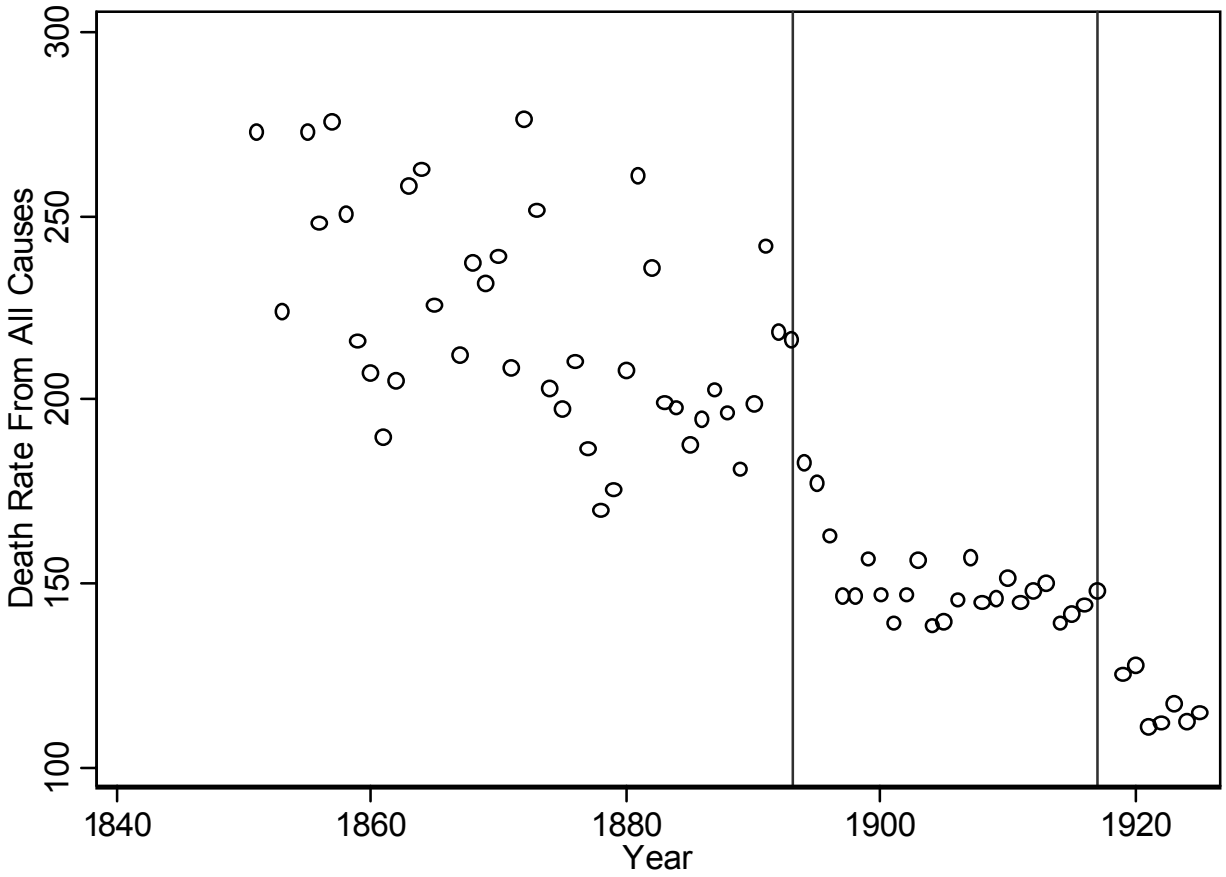
Death Rate From All Causes
300
250
200
150
100
1840
1860
1880
1900
1920
Year

Figure 1

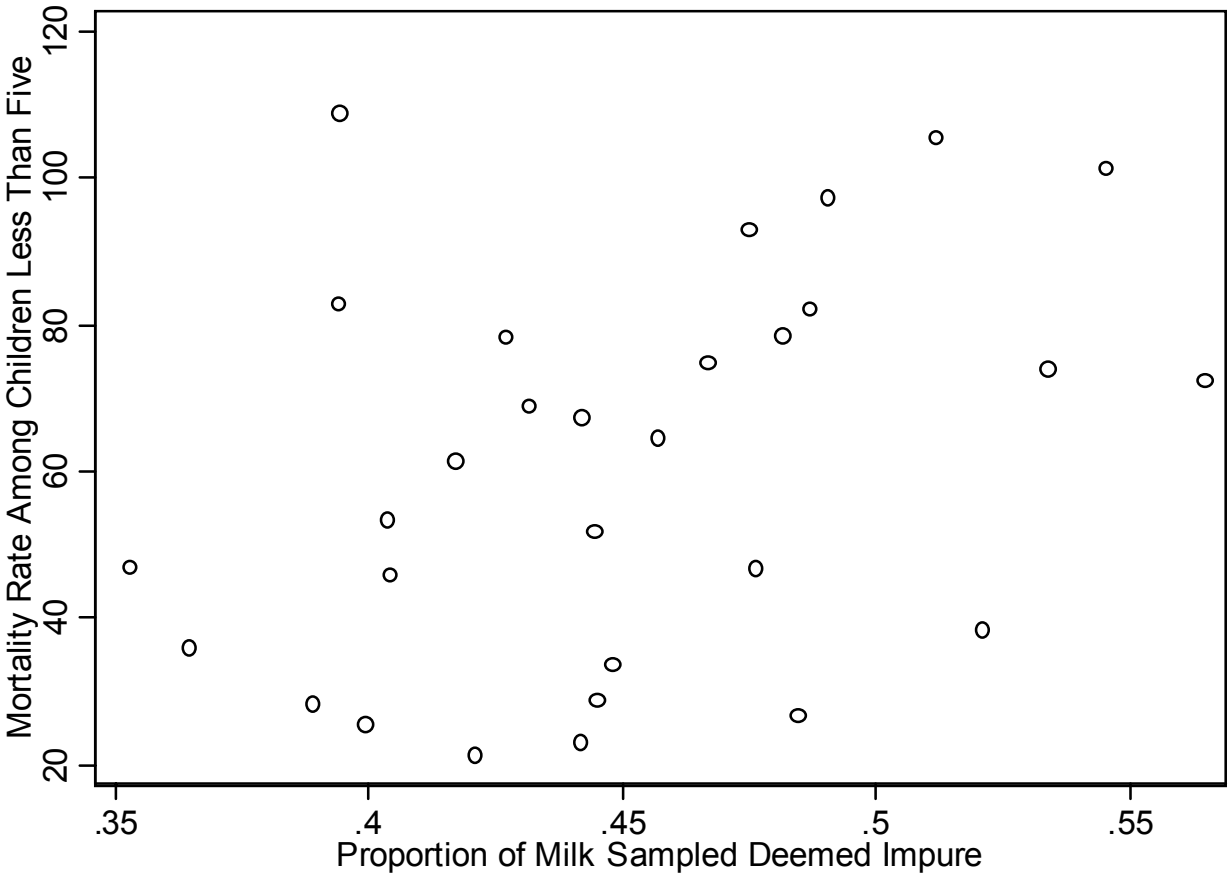
Mortality Rate Among Children Less Than Five
20
40
60
80
100
120
.35
.4
.45
.5
.55
Proportion of Milk Sampled Deemed Impure

Figure 2

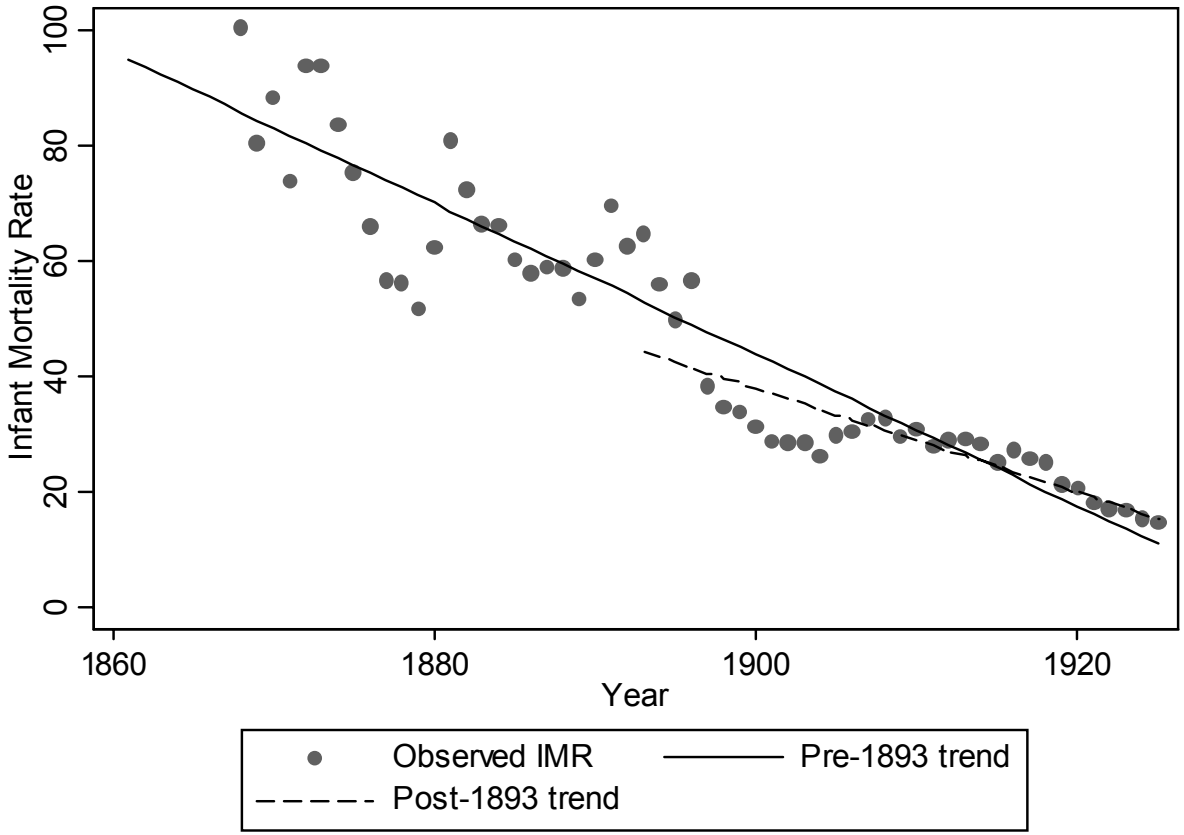
Infant Mortality Rate
0
20
40
60
80
100
1860
1880
1900
1920
Year
Observed IMR
Pre-1893 trend
Post-1893 trend

Figure 3

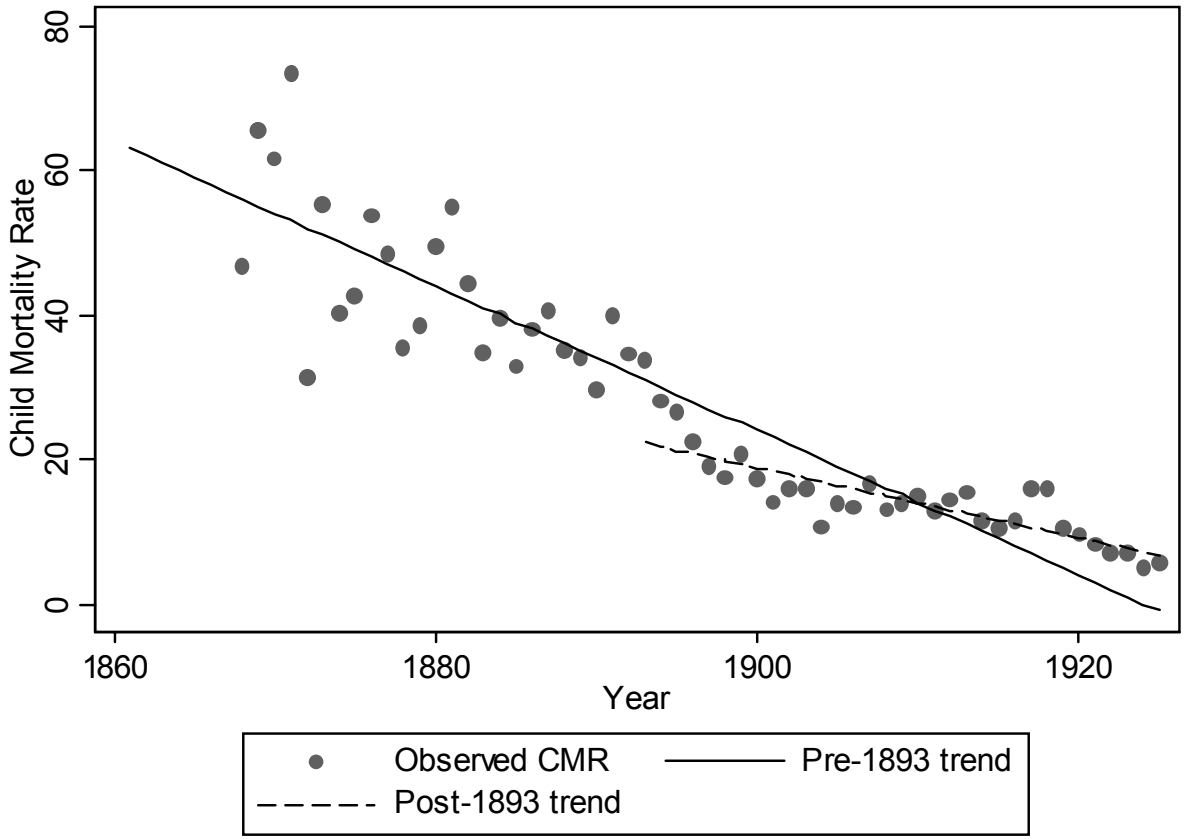
Child Mortality Rate
0
20
40
60
80
1860
1880
1900
1920
Year
Observed CMR
Pre-1893 trend
Post-1893 trend

Figure 4


Death Rate From Diphtheria
0
10
20
30
1860
1880
1900
1920
Year

Figure 5

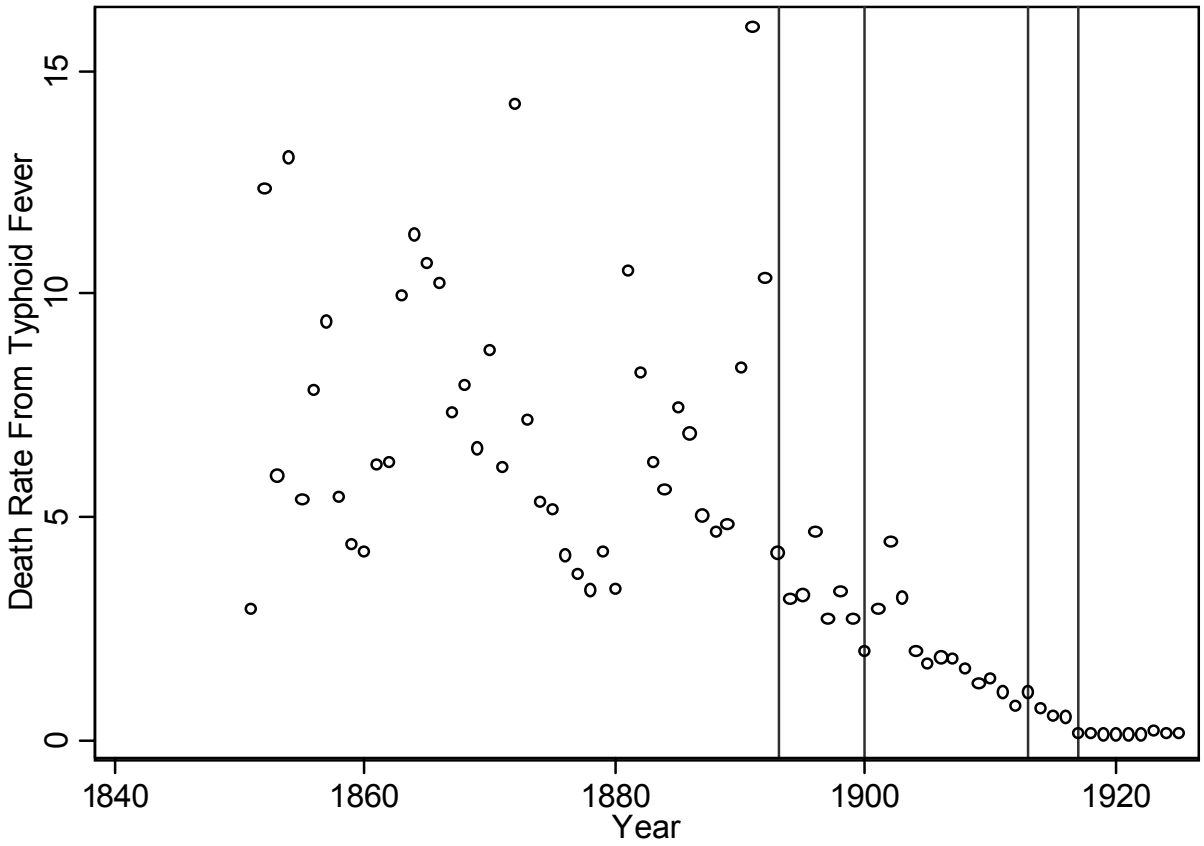
Death Rate From Typhoid Fever
0
5
10
15
1840
1860
1880
1900
1920
Year

Figure 6

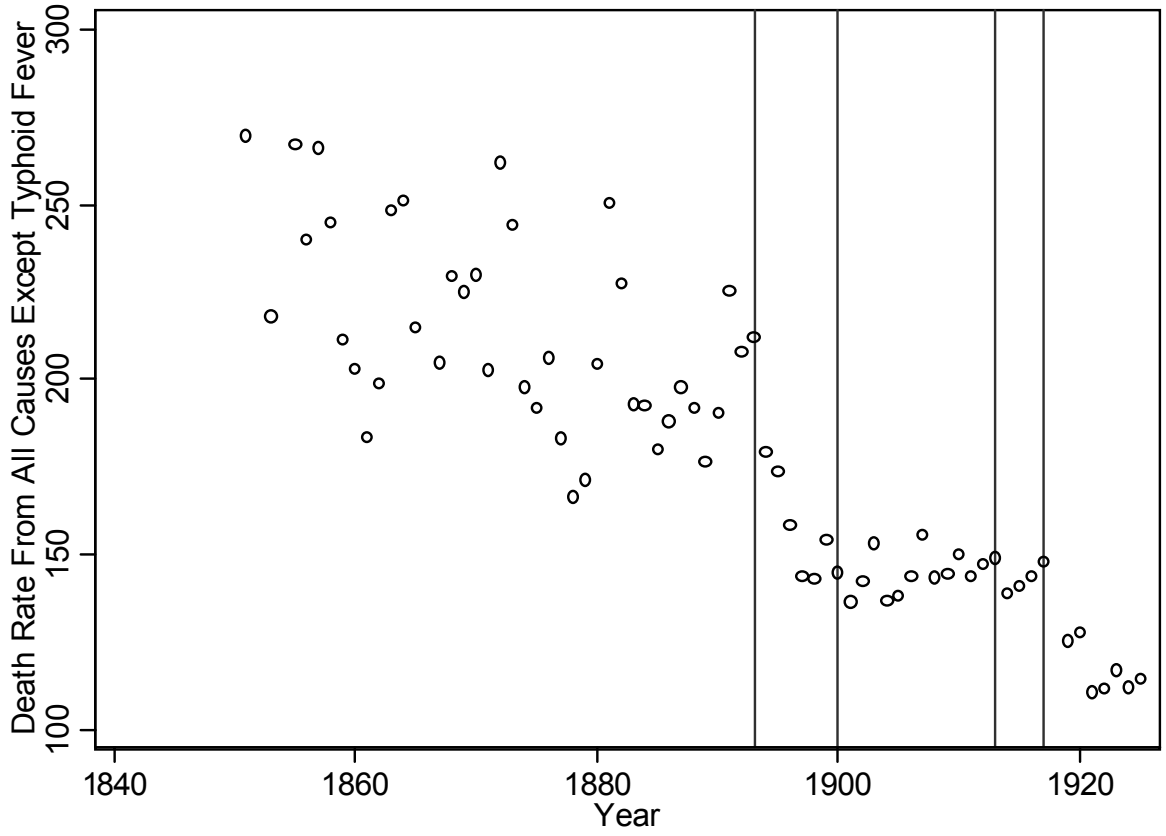
Death Rate From All Causes Except Typhoid Fever
100
150
200
250
300
1840
1860
1880
1900
1920
Year

Figure 7


Death Rate Except Typhoid, Infant, Diphtheria
80
100
120
140
160
1860
1880
1900
1920
Year

Figure 8

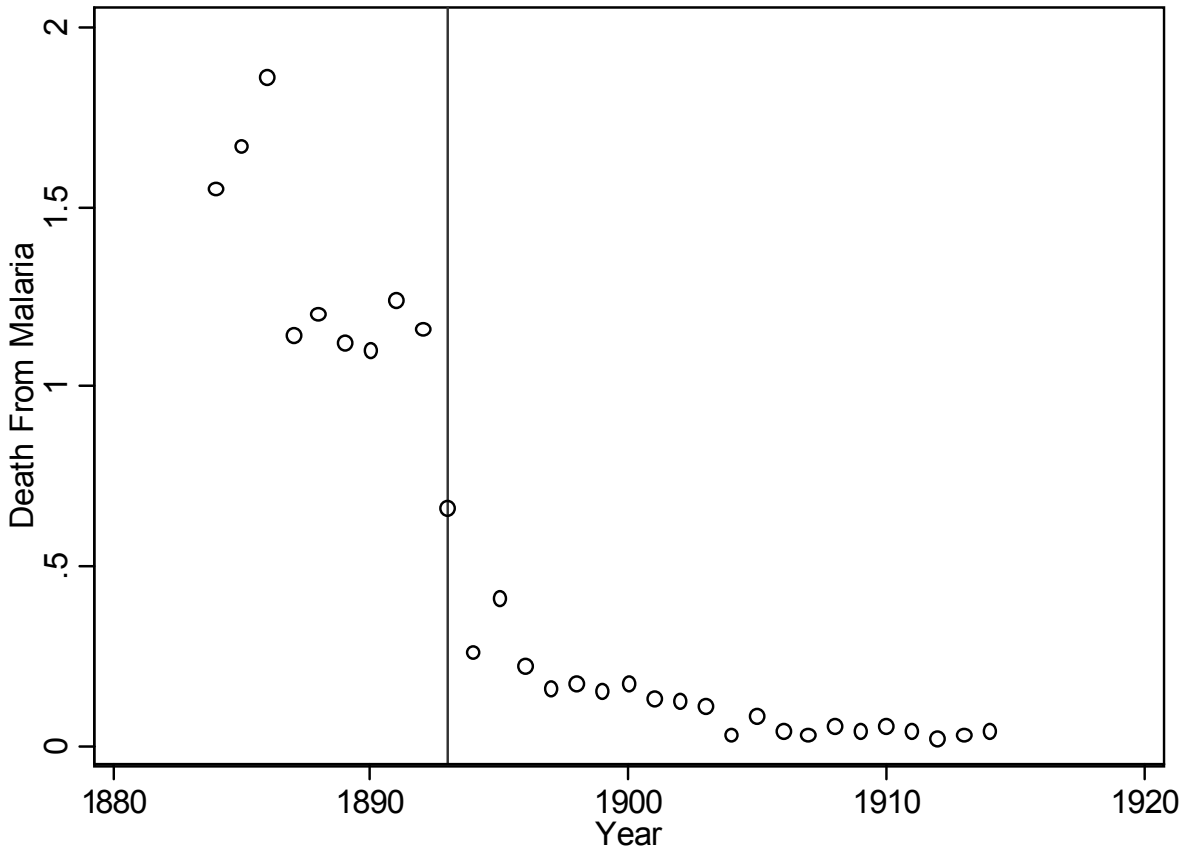

Figure 9

Figure 10. Enumeration Districts Used in 1880 Mortality Analysis

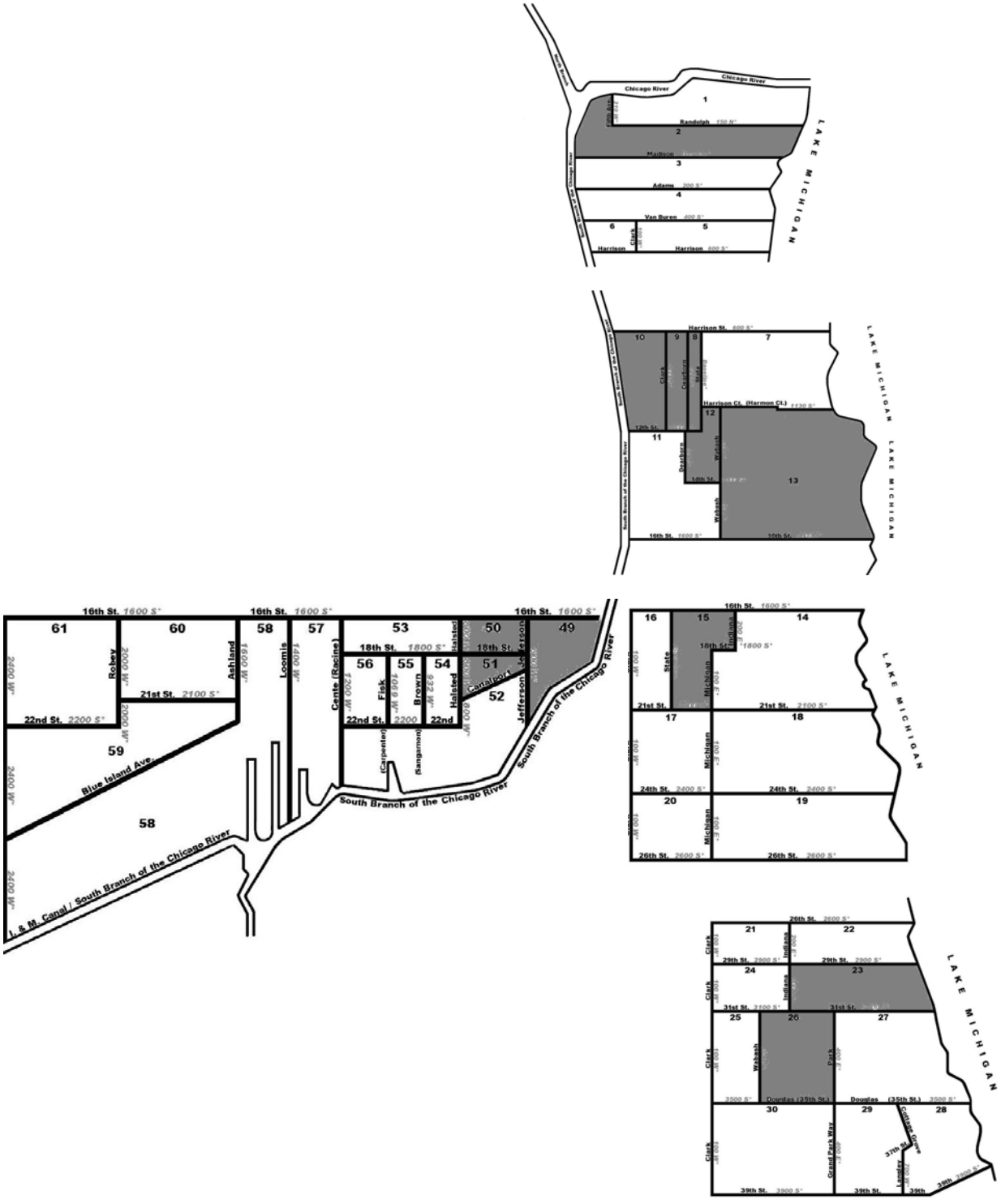

Table 8. Characteristics of Chicago & Enumeration Districts Used

	White	Foreign Born	Laborer (Male 20+)	Under Age 20
City	98.7%	41.2%	15.9%	45.5%
Dist 2	94.5	38.0	1.8	25.7
Dists 8, 9, & 10	74.0	35.0	15.3	43.8
Dists 12, 13, & 15	95.5	29.2	7.4	35.5
Dists 23 & 26	98.5	24.7	1.7	42.6
Dists 49, 50, & 51	100.0	48.7	32.0	51.6

Table 9. Sewer Access by Water Access

	Water	
Sewer	N	Y
N	8,033	347
Y	901	20,384

Table 10. Marginal Effects of Water and Sewer Access on Mortality

	(1) Infants	(2) Age 1-4	(3) Age 5-19	(4) Age 20-44	(5) Age 45+
Water	0.0561	-0.0019	-0.0010	0.0000	-0.0076
	(2.80)***	(0.31)	(0.60)	(0.01)	(2.16)**
Age		-0.0176	-0.0003	0.0001	0.0007
		(6.14)***	(1.51)	(1.48)	(4.81)***
Dummies for Enum. Districts	Y	Y	Y	Y	Y
Observations	822	2950	8689	13269	3935
Predicted Probability	0.0788	0.0266	0.0054	0.0027	0.0103

Absolute value of z statistics in parentheses
* significant at 10%; ** significant at 5%; *** significant at 1%

	(1) Infants	(2) Age 1-4	(3) Age 5-19	(4) Age 20-44	(5) Age 45+
Sewer	0.0547	-0.0111	-0.0013	-0.0003	-0.0070
	(2.72)***	(1.83)*	(0.77)	(0.30)	(1.95)*
Age		-0.0173	-0.0003	0.0001	0.0007
		(6.12)***	(1.49)	(1.46)	(4.83)***
Dummies for Enum. Districts	Y	Y	Y	Y	Y
Observations	822	2951	8699	13286	3944
Predicted Probability	0.0790	0.0262	0.0054	0.0027	0.0104

Absolute value of z statistics in parentheses
* significant at 10%; ** significant at 5%; *** significant at 1%

	(1) Infants	(2) Age 1-4	(3) Age 5-19	(4) Age 20-44	(5) Age 45+
Sewer	0.0186	-0.0621	-0.0024	-0.0021	-0.0003
	(0.33)	(3.52)***	(0.56)	(0.73)	(0.04)
Water	0.0408	0.0364	0.0010	0.0014	-0.0073
	(0.76)	(3.03)***	(0.27)	(0.66)	(0.88)
Age		-0.0167	-0.0003	0.0001	0.0007
		(6.05)***	(1.48)	(1.46)	(4.81)***
Dummies for Enum. Districts	Y	Y	Y	Y	Y
Observations	822	2950	8689	13269	3935
Predicted Probability	0.0788	0.0254	0.0054	0.0027	0.0103

Absolute value of z statistics in parentheses
* significant at 10%; ** significant at 5%; *** significant at 1%

Table 11. Marginal Effects of Water and Sewer Access on Mortality

	(1) Infants	(2) Age 1-4	(3) Age 5-19	(4) Age 20-44	(5) Age 45+
Male	0.0075	0.0071	-0.0005	-0.0008	0.0033
	(0.42)	(1.41)	(0.42)	(1.39)	(1.48)
White	-0.1072	0.0090	-0.0017	-0.0004	-0.0258
	(2.10)**	(0.63)	(0.39)	(0.27)	(2.46)**
Born In State	0.0475	0.0039	-0.0004	0.0021	0.1322
	(1.05)	(0.35)	(0.22)	(1.59)	(4.89)***
White Col Hhld Head	0.0412	-0.0091	-0.0028	0.0013	-0.0026
	(1.45)	(1.34)	(1.72)*	(1.69)*	(1.02)
Blue Col Hhld Head	0.0200	-0.0100	-0.0029	-0.0007	-0.0042
	(0.88)	(1.78)*	(2.02)**	(0.99)	(1.81)*
Family Size	0.0023	0.0005	0.0001	0.0003	0.0003
	(0.69)	(0.53)	(0.41)	(3.40)***	(0.83)
Dwelling Size	-0.0070	-0.0000	-0.0005	0.0003	0.0004
	(1.65)*	(0.03)	(1.27)	(2.41)**	(0.74)
Sewer	0.0105	-0.0353	-0.0016	-0.0015	0.0038
	(0.17)	(2.36)**	(0.47)	(0.61)	(0.77)
Water	0.0386	0.0324	0.0026	0.0011	-0.0124
	(0.66)	(2.90)***	(0.87)	(0.67)	(1.63)
Age		-0.0152	-0.0003	0.0001	0.0006
		(5.94)***	(1.70)*	(2.19)**	(5.57)***
Born in U.S.		0.0167	0.0008	-0.0008	-0.0054
		(1.19)	(0.34)	(0.97)	(1.90)*
Married			0.0201	-0.0012	0.0022
			(1.99)**	(1.63)	(0.61)
Widowed				-0.0011	-0.0016
				(0.97)	(0.38)
Predicted Probability	0.0666	0.0211	0.0042	0.0015	0.0059
Observations	797	2778	8241	13153	3904

Absolute value of z statistics in parentheses

* significant at 10%; ** significant at 5%; *** significant at 1%