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ABSTRACT

Beginning around 1880, public health issues and engineering advances spurred the installation of city water and sewer systems. As part of this growth, many cities chose to use lead service pipes to connect residences to city water systems. This choice had negative consequences for child mortality, although the consequences were often hard to observe amid the overall falling death rates. This paper uses national data from the public use sample of the 1900 Census of Population and data on city use of lead pipes in 1897 to estimate the effect of lead pipes on child mortality. In 1900, 29 percent of the married women in the United States who had given birth to at least one child and were age forty-five or younger lived in locations where lead service pipes were used to deliver water. Because the effect of lead pipes depended on the acidity and hardness of the water, much of the negative effect was concentrated on the densely populated eastern seaboard. In the full sample, women who lived on the eastern seaboard in cities with lead pipes experienced increased child mortality of 9.3 percent relative to the sample average. These estimates suggest that the number of child deaths attributable to the use of lead pipes numbered in the tens of thousands. Many surviving children may have experienced substantial IQ impairment as a result of lead exposure. The tragedy is that lead problems were avoidable, particularly once data became available on the toxicity of lead. These findings have implications for current policy and events.

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1. Introduction

The period 1880 to 1920 was a period of falling mortality in the United States for infants, children and adults. Much of this reduction has been attributed to installation of water and sewer systems in cities (Haines 2001; Ferrie and Troesken 2005). It is widely accepted that much of the improvement in child mortality in particular was the result of better nutrition and improved public health services that protected infants and young children from infectious diseases, particularly deaths from infantile diarrhea. Of studies that look at child mortality and mortality transitions in the United States, Preston and Haines (1991) is probably the best known and most important. In their analysis of census data from the turn of the century, Preston and Haines emphasize the importance of infectious diseases.

Against that backdrop is a lesser known story. Many of the new water systems employed lead service pipes which ran from iron street mains to the customer's home. Lead was considered a superior engineering material for this purpose because of its malleability and durability. Unfortunately, such expedience would cause substantial increases in child mortality rates from the use of lead pipes. The cause of death is traceable to high levels lead exposure in utero and to a lesser extent after birth.

This paper builds on Troesken's (2003) case study of the lead problem in Massachusetts. We show that the lead problem was national. In 1900, 29 percent of the married women in the United States who had given birth to at least one child and were ages forty-five or younger lived in locations where lead service pipes were used to deliver water. Because the effect of lead pipes depended on the acidity and hardness of the water, much of the negative effect was concentrated on the densely populated eastern seaboard.

To estimate the effect of lead pipes on child mortality, we use data from the public use sample of the 1900 Census of Population. In the 1900 census, in addition to the standard demographic information, women were asked to report the number of children ever born and the number of children surviving, excluding stillbirths. We combine these data with data on city use of lead pipes in 1897 and mortality rates from tuberculosis in 1900 to estimate the effect of lead pipes on child mortality. In the baseline regression, we estimate the effects of lead water delivery systems on child mortality controlling for the mother's characteristics, the father's occupation, average state temperature, precipitation and drought, city size, and region. The

negative effects of lead delivery systems were not widely known at this time, so the use of lead was predominantly a function of city size. To address possible issues of endogeneity, we also present instrumental variables estimates.

In the full sample, we find that in 1900 women who lived on the eastern seaboard in cities with lead pipes experienced increased child mortality of 9.3 percent relative to the sample average. This is an increase in child mortality of roughly 13 deaths per 1,000 children. In comparison, in 2000 the white infant mortality from all causes was 5.7 deaths per 1,000. Not only did the number of child deaths attributable to lead pipes number in the tens of thousands, but the number of deaths was more than twice the current infant mortality rate. Further, we find that the lead service pipes account for 47 percent of the so called urban penalty – the increased child mortality from living in a town of more than 2,500 people. Many surviving children may have experienced IQ and behavioral impairments as a result of water-related lead exposure, although we cannot demonstrate this.

Our estimates are biased downward by four factors. First, we do not observe mothers' residence at the time they had their children. Because the nineteenth and early twentieth century was an era of rapid urbanization, some fraction of women we observe in cities with lead service pipes may have had their children prior to the installation of the water system or in locations where lead was uncommon. Second, by 1900 some cities that did not use lead pipes in 1897 may have begun to use lead pipes later on, and vice versa. Third, some cities are identified as having mixed lead-non-lead delivery systems. We code these cities as if they had only lead service pipes. Fourth, we only observe the excess mortality due to lead. To the extent that some children in cities without lead pipes were killed by lead from prior exposure in cities with lead pipes, the actual effect would be larger than the estimated effect.

Because lead-related child mortality is clustered in heavily urbanized areas, particularly the Northeast, the results here have implications for our understanding of the urban fertility transition and regional variation in that transition. Previous studies of fertility change have been predicated on the assumption that transition was voluntary in the sense that women were choosing to engage in a variety of behaviors that had the result of reducing the birth rate. Children were no longer as valuable, women had more opportunities outside the home, and financial markets had produced alternatives to children as a means of ensuring support in ones

old age. If, however, this behavior was in part involuntary, because lead made it more difficult to get pregnant, stay pregnant, and deliver a baby that would live to adulthood, this had implications for our understanding of the transition and the role that industrialization and financial markets played in the transition.

Other types of service pipes were available, as were methods to at least partially mitigate the negative effect of lead service pipes. Nevertheless, even in the face of mounting evidence, use of lead pipes continued. Today the issue of lead in water is resurfacing, in part because of changing water treatment techniques. These new water treatment technologies are better able to destroy newly emerging pathogens such as cryptosporidium but also tend to make water more lead solvent. Recent lead levels in water in some locations in the District of Columbia are as high as or higher than historical levels recorded at the turn of the century (Renner 2004; Troesken 2006). Because testing of water in homes is rare, the extent of the problem is unknown.

Section 2 discusses the use of lead pipes and the circumstances under which leaching of lead was most likely to occur. Section 3 presents evidence on the toxicity of lead, why its effects were difficult to detect, and contemporary strategies for mitigation of the problem. Section 4 examines child mortality in the 1900 and 1910 samples. Section 5 discusses identification of the lead effects and presents the main results. Section 6 addresses the effects on surviving children. Section 7 concludes.

2. Lead Pipes

Municipal water systems became common beginning around 1880. Lead was often used in the construction of water service lines that connected individual homes and apartment buildings to street mains. Relative to other materials such as plain or galvanized iron or steel or cement lined iron, lead had two features that made it attractive to the engineers who designed public water systems: it was both malleable and durable. Malleability reduced labor costs by making it easier to bend the service main around existing infrastructure and obstructions. As for durability, the life of the typical lead service pipe was thirty-five years. By contrast, plain iron or steel pipe lasted sixteen years; galvanized pipe twenty years; and cement lined pipe twenty-eight years. Based solely on engineering concerns, these characteristics made lead the ideal material for service lines. As one prominent trade journal wrote: “Lead is in many respects the most

satisfactory material to use for service pipes. Its pliability and its comparative freedom from corrosive action make it almost ideal from a mechanical standpoint."[1]

Despite its attractiveness from an engineering standpoint, lead did have drawbacks. First, lead had higher up front costs. This was offset in many locations by its lower cost to maintain, because replacing broken service mains often required digging up paved streets and working around other infrastructure such as gas and sewer mains.[2] Second, there were concerns that lead service pipes affected water quality. Most engineers, however, appear to have believed that such concerns were overblown.[3]

Despite these drawbacks, the managers of municipal water systems often chose to use lead service pipes. This can be seen in two independent samples of cities. In 1916, the New England Water-Works Association surveyed 304 cities and towns, largely in New England, and found that 31 percent of these cities used lead or lead-lined services.[4] A second and independent sample is more geographically diverse and includes 797 cities and towns observed in 1897 from all over the United States. Table 1, which breaks down lead use for the second sample by city size, suggests a strong positive correlation between lead use and city size. For cities with populations less than 8,000, 33 percent used lead service pipes. In contrast, for cities with populations between 30,000 and 300,000, 72 percent used lead pipes; and for the largest cities, those with populations greater than 300,000, all but one (94 percent) used lead service pipes (Troesken and Beeson 2003).

In 1900, the Massachusetts State Board of Health, an early leader in examining the lead problem, investigated the amount of lead contained in household tap water in twenty-two municipalities across the state. Health officials took several samples of water from household faucets in these cities after the water had passed through lead service pipes; measured the lead content of these samples; and reported their findings in the annual report of the board of health.

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[1] Information and quotations in this paragraph come from the *Engineering News*, September 28, 1916, pp. 594-96 (hereafter cited as EN); and from the Committee on Service Pipes (1917), p. 328 (hereafter cited as CSP). The editors of the *Engineering News* were not alone in suggesting that lead, even with concerns about safety, was the best material for service lines. A survey of the superintendents of forty-one municipal water companies found that about half (20) preferred lead service lines to all other types of lines. This survey was conducted in 1884 by water industry expert from New London, Connecticut. The results were reported in the *Journal of the New England Water Works Association*, September, 1917, pp. 346-47.

[2] In bigger cities, the cost of lead pipes was a smaller share of real estate values.

[3] EN, p. 595

[4] CSP, pp. 326-30; and EN, p. 594.

Officials also reported data about the chemical composition and qualities of the local water supply, including how hard the water was and how acidic it was.

The data derived from this investigation suggest four important observations. First, by today's standards, lead levels in the sampled municipalities were extraordinarily high. Across the twenty-two towns in the study, the average amount of lead in water in normal use was twenty-one times the EPA limit, and the average amount after standing overnight was fifty-eight times the EPA limit.[5] The largest value of a household in any town in ordinary use was 343 times the EPA limit and the largest value when left standing overnight was 762 times the EPA limit.

Second, the use of lead pipes increased the amount of lead contained in household tap water. Although there were plenty of possible sources of lead in tap water, two experiments suggest that lead dissolved from the interior lining of service pipes was the primary source of lead in drinking water. In the first experiment, eighteen samples of water from the Lowell, Boulevard well were passed through iron and lead service lines. In ordinary use, the water that traveled through the lead service line had three times the lead level of the water that traveled through the iron service pipe. When the water was allowed to stand overnight, the water that traveled through the lead service line had eight times the lead level of the water that traveled through the iron service pipe.[6] In an independent experiment conducted by two New York health officials during the 1930s, very similar findings were obtained.

Third, there were significant interaction effects. The amount of lead added to drinking water by lead service lines depended on at least two characteristics of the water supply: how hard and acidic it was. In towns with soft water or with water containing high levels of acid, large amounts of lead dissolved into the water supply if lead pipes were employed. In towns with hard or less acidic water, relatively small amounts of lead dissolved into the water.

The implications of the investigation were particularly severe for cities that used lead pipes and were located along on the eastern seaboard, because these cities had water that was generally both acidic and soft. Figure 1 shows the incidence of acid rain. While the data in the figure is from 1999, industrialization of the eastern corridor was well underway during the nineteenth century. The eastern corridor, particularly the Northeast, almost certainly experienced acid rain

[5] The current EPA standards allow water to contain 0.0015 parts of lead per 100,000 units of water.
[6] All reports of the Massachusetts State Board of Health are cited as MSBH. MSBH 1900, pp. 491-97

around the turn of the century. As early as the 1880s, scientists in England were discussing the possibility that acid rain might increase the tendency of water to take up dangerous amounts of lead. For example, Gilbert Kirker (1890) wrote a short article in the *British Medical Journal* arguing that "the products of combustion, which in that part of the country" were "poured into the atmosphere in great quantity and variety" gave the "moorland water supplies" their "abnormal and constant plumbosolvent action." Although Kirker did not refer to it as such, he was talking about acid rain, which is now recognized as a contributor to water-lead levels.[7]

Further, many of the lakes and other water sources were already marginally acidic prior to industrialization.[8] Figure 2 shows that the eastern corridor had surface water sources that were not able to resist acidification. According to the Environmental Protection Agency (2003) "Whether surface waters can resist acidification depends on the ability of the water and watershed soil to neutralize the acid deposition it receives. The best measure of this ability is acid neutralizing capacity (ANC), determined by the amount of dissolved compounds that will counteract acidity. ANC depends largely on the surrounding watershed's physical characteristics such as geology, soils and size. Surface water with an ANC of 200 micro equivalents per liter (μeq/L) is normal; ANC less than 50 micro equivalents per liter is considered highly sensitive to acidification. As illustrated in this map, surface waters in Florida, the Northeast, the Mid-Atlantic, Upper Midwest and parts of the West are highly sensitive to acidification (with surface water ANC of less than zero)."[9] ANC appears to be quite persistent over time, particularly in New England and the Ridge/Blue Ridge regions.[10] Figure 3 shows the average hardness of groundwater in the United States. The eastern corridor also had water that was soft (i.e., very low on the hardness scale). Hardness is an attribute of the soil and is extremely persistent over time.[11] Thus, we would expect the negative effects of lead pipes to have been more severe on the eastern seaboard than in other parts of the United States.

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[7] Alfred H. Allen (1882), a chemist, reported experimental results consistent with this argument.

[8] "Lakes inferred to have been measurably acidified by atmospheric deposition were already marginally acidic, typically with pH less than 6, before anthropogenic atmospheric pollution began more than 100 years ago. Therefore, full recovery of acidic lakes will not yield neutral pH." P. xii

[9] EPA (2006).

[10] EPA (2003), p. x.

[11] According to the United States Geological Survey (2006), "Water hardness is based on major-ion chemistry concentrations. Major-ion chemistry in ground water is relatively stable and generally does not change over time. Although the map [a map similar to the one in figure 3] illustrates data from 1975, these data have been found to be accurate and useful in current assessments."

Fourth, the amount lead taken from the interior walls of lead service pipes varied inversely with the age of the pipe. Holding the corrosiveness of water supplies constant, less lead was dissolved from old pipes than from new.[12] We will return to this issue later.

3. Lead Poisoning

To foreshadow later findings, the empirical work below suggests that the use of lead service pipes had a substantial effect on child mortality rates. For such findings to be believable, three questions need to be addressed. First, was the amount of lead contained in drinking water in some states sufficiently large to kill an infant or small child? Second, a large increase in death rates suggests that in at least some places, lead poisoning via water was killing a substantial fraction of all infants and young children. How could such an enormous factor in infant mortality have gone undetected? Third, how difficult was it for private homeowners to prevent water-related lead exposure? For example, today health officials recommend flushing pipes before using tap water for drinking or cooking to remove any residual lead from plumbing fixtures. Were such practices known in 1900? Would they have had significant effects?

Was There Enough Lead in Tap Water to Kill an Infant?

Lead exposure is especially serious because children absorb more lead than adults, and because the developing nervous systems of children are more susceptible to the effects of lead than are the systems of adults (Needleman and Belinger 1991; and Nebraska Epidemiology Report, 2001). At the turn of the century, infants and young children were exposed to lead through four major pathways: in utero through maternal water consumption or contemporaneous environmental exposure, in utero from leaching of lead from maternal bones, after birth through breast milk, and after birth through the addition of water to cow's milk.

Studies conducted during the early twentieth century suggest that high levels of maternal lead exposure can have enormous effects on the health outcomes of the very young. In one British study, women who were working in the lead industry experienced miscarriages and stillbirths at rates three times greater than those of women working in non-lead industries. A French study

[12] For example, Quam and Klein (1936 p. 779, particularly tables I and II.), using water samples with consistent chemical properties, compare the amount lead that was taken off old lead pipes and new pipes. They find that substantially more lead was introduced into water stored in new pipes than in old pipes. By the same token, studies by the Massachusetts State Board of Health conducted during the 1890s and early 1900s found that the most serious cases of lead poisoning tended to occur with newer lead pipes as opposed to old pipes. Empirically this could be useful, if we knew the distribution of the age of lead water lines within each city. Unfortunately, we do not know when cities began using lead lines or the fraction of households with lead service lines at any given point in time.

found that out of one thousand pregnancies among women working with lead, 608 culminated in premature delivery. According to a study published in 1908, "in certain Hungarian villages, where pottery glazing has been a home industry for generations, children born of lead poisoned parents are not only subject to convulsions, but, if they live, often have abnormally large, square heads, and this condition is associated with a lowered mentality."[13]

The strongest modern-day evidence that lead exposure, even in very small amounts, can result in fetal and infant death is presented by Wolpaw Reyes (2003). Using state-level data from the U.S., Wolpaw Reyes shows that the phase-out of leaded gasoline reduced infant mortality rates by 3 to 4 percent. Competing studies of the effects of lead on infant health are plagued by concerns about small samples, unobserved heterogeneity, and reverse causality whereby pre-existing poor health might determine the amount of lead exposure. Wolpaw Reyes, however, is able to control for most relevant confounding variables and uses variation in the consumption of leaded gasoline—a truly exogenous variable—to isolate the effects of moderate levels of lead exposure.

Finally, one simple way to illustrate how dangerous water lead could be to the developing fetus and child is to look at the history of abortion. During the late 1800s and early 1900s, a handful of commercial enterprises began manufacturing and marketing abortion pills made of diachylon (a lead-based plaster) in both England and the United States.[14] One popular brand of pill was described in the medical literature only as Dr. ___'s Famous Female Pills. The instructions directed women to take two pills four times a day over a two week period. An article in the *British Medical Journal* indicated that each pill contained .0005 grains of lead, implying that if a woman took the recommended daily dose of eight pills, she would have ingested .004 grains of lead per day (Troesken 2006). The efficacy of products like Dr. ____'s Famous Female Pills were idiosyncratic, affecting some individuals much more than others.[15] The medical literature of the day indicates that more than a few women overdosed and poisoned themselves, sometimes fatally. In other cases, not enough lead was ingested and the subsequent child was born small and unhealthy. However, most women apparently were able to ingest

[13] These and other studies are summarized in a report by United States Bureau of Labor Statistics (1919).
[14] Women were probably not aware that the active ingredient in the pills was lead, since this predates the pure food and drug movement.
[15] Also, it is not clear how carefully the suppliers of such pills calibrated the recommended dose.

diachylon pills with few outward signs of lead poisoning and simultaneously terminate their pregnancies (Troesken 2006).

Using the data on how much lead was contained in the recommended daily dose of these pills and comparing that level to the amount of lead contained in Massachusetts tap water, Troesken (2006) constructs a measure referred to as the *abortifacient equivalent* (*AE*). The *AE* is the amount of water an individual needed to consume in order to have been exposed to the same amount of lead as was contained in the recommended daily dose of Dr. ____'s Famous Female Pills. Data from Massachusetts in 1900 indicate that, in the typical Massachusetts town, a housewife needed to drink around 80 ounces of tap water per day to reach the *AE*, assuming she regularly flushed her pipes before drinking tap water. If, however, the housewife did not practice flushing and regularly consumed water allowed to stand in pipes for several hours, she need only have consumed 30 to 40 ounces of tap water per day. Furthermore, this is a situation where measures of central tendency can be deceiving. In places like Attleborough, Lowell, and Middleborough, housewives need only have consumed 1 to 10 ounces of tap water daily to reach the *AE*. Similarly, in Lawrence, Newton, and Weymouth, housewives need only have consumed 6 to 28 ounces of water daily to have reached the abortifacient equivalent (Troesken 2006).[16]

Data on the effect of adult exposure to lead is also suggestive, since children were typically more severely affected. Using a sample of more than two thousand Union-Army veterans, Troesken and Beeson (2003) compare the health outcomes of veterans living in towns with lead water lines to those of veterans living in towns with non-lead mains. They find evidence that the use of lead water lines increased the rate of nephropathy (kidney failure) among adult males.[17] Additional evidence of the adverse health effects of lead water lines can be found in two well-documented episodes of water-related lead poisoning. These two episodes involve Milton–a small town outside of Boston—and Lowell—a large industrial town in northeastern Massachusetts. These case studies demonstrate that, in a fairly short time, lead service lines could introduce enough lead into drinking water to cause death and insanity among adults (Troesken 2006).

[16] If she consumed the water as tea, which was common during the period, the required consumption would be less, because boiling concentrated the lead.

[17] Nephropathy is observed in adults when blood levels reach between 40 and 100 micrograms per deciliter (μg Pb/dl). Similar blood lead levels in children cause encephalopathy (brain-swelling).See Perazella (1996) and Xintaras (1992).

How Did All This Lead Poisoning Go Undetected?

Regarding the second question – How did all of this lead poisoning go unnoticed? – it is useful to consider the diagnostic challenge lead poisoning represented. Because lead affected multiple body systems, and because the symptoms of lead poisoning varied greatly from person-to-person according to the individual's size and general ability to withstand lead exposure, it was difficult for doctors in the nineteenth and early-twentieth century to diagnose lead poisoning on an individual basis. Absent some external or circumstantial condition to suggest lead poisoning – e.g., the patient worked as a painter, or lived close to a lead refinery – doctors appear to have been reluctant to ascribe the periodic case of paralysis, colic, constipation, kidney or liver trouble, and the like, to anything other than arthritis, diet, family history of disease, or excessive drinking.

For children, diagnosing lead poisoning accurately was even more problematic. In a recent social history of lead poisoning in America, Warren (2000) describes cases of childhood lead poisoning during the early twentieth century that were misdiagnosed as feeble-mindedness, summertime colic, appendicitis, polio, and convulsions and paralysis of unknown origin.[18] Warren, however, argues that the most common misdiagnosis for lead poisoning was tubercular meningitis: The early stages of both diseases produce appetite loss, headache, vomiting, and acute constipation. Gradually, neurological symptoms arise: photophobia, rigidity of the neck, lethargy, and paralysis of the limbs or facial nerves. The most serious cases end in convulsions, coma, and death. In summarizing a study of 105 tubercular meningitis cases treated in the Boston Children's Hospital between 1910 and 1913, Warren strongly suggests that more than half of these cases were, in fact, the result of lead poisoning and not tubercular meningitis. This study found that tubercle bacilli were identified in less than 25 percent of the cases, and that in over 60 percent of the cases, there was no pathological evidence of tubercular meningitis.

In terms of promoting a more accurate understanding of the effects of lead water lines, it is perhaps unfortunate that there were not more epidemic-like outbreaks of lead poisoning. If there had been, the dangers of lead pipes would have been clear and compelling. And municipalities might have been forced to abandon lead pipes for pipes that were less desirable from an

[18] Warren (2000), pp. 34-35.

engineering standpoint, but were much safer from a public health standpoint. Instead, lead water pipes quietly killed thousands of children every year.

Private Prevention of Lead Poisoning

For the results presented below to be credible, a third question must be addressed as well – Did homeowners know about the risk of lead in the water and if so were there ways to avoid lead exposure? It is not clear that many consumers were aware of the dangers of water-related lead poisoning. In turn-of-the-century America, most people were far more concerned about organic pollutants in tap water than they were about inorganic pollutants such as lead.

In 1900, public officials in some locations recommended that families with lead service pipes flush their plumbing systems by running taps for two minutes before using water for drinking and cooking. As late 1936, however, the *American Journal of Public Health* was publishing articles exhorting homeowners to flush their pipes before drinking tap water in order to minimize water-related lead exposure.[19] This suggests that at least some portion of the population was unaware of this strategy.

Flushing pipes in this way did eliminate some of the excess lead in tap water, but it did not eliminate all of it. Consider this excerpt from the *Milton News* (February 15, 1902), quoting the Massachusetts State Board of Health: "If the water is allowed to run freely, the quantity of lead taken up will be less than when the water is allowed to stand in the pipe for considerable time; but even when allowed to run freely it may contain a sufficiently large quantity of lead to produce serious injury."[20] The only way to have been sure of the lead content of one's tap water was to have it tested. If a homeowner did have their lead tested, a costly process, and found lead levels to be high, the homeowner then had to locate a source of drinking water that had lower lead levels. In the absence of compelling evidence on lead toxicity, few households were likely to take such steps.

4. Child Mortality in the 1900 and 1910 Censuses

In this section, we present the data and establish basic stylized facts about child mortality in 1900 and 1910. In the next section, we discuss identification of the effects of lead and present the main results.

[19] Quam and Klein (1936).
[20] *Milton News* February 15, 1902, p. 4

To investigate child mortality, we use the IPUMS 1-200 sample of the 1900 Census of Population. The 1900 census asked women to report the number of children ever born and the number surviving. The enumerator's instructions with respect to children ever born are as follows: "This question applies only to women, and its object is to get the number of children each woman has had, and whether the children are not living on the census day. Stillborn children are not to be counted."[21] The instructions for children surviving are: "Enter in column 12 the figure showing the number of these children living on the census day. Whether the children are living in your district or elsewhere makes no difference. If the woman has had no children, or if they are all dead, write. "0."" Like Costa and Kahn (2003), we restrict attention to women ages 45 or younger who were married less than 15 years and for whom number of children ever born is greater than zero and is less than or equal to the duration of the marriage.

Table 2 presents the summary statistics for mothers. The 1900 sample includes 24,483 women and 70,544 children ever born nationwide. The first three rows present the sample averages for the fraction of a women's children who have died, the number of children that died, and the number of children ever born. It is important to note that the numbers in Table 2 may understate the true child mortality rate, since we are only sampling women who were alive at the time of the census. In particular, women who died in childbirth are not sampled, and many of those babies may not have survived. The women in the sample were predominantly white, literate, rural, did not own their own home and did not report that they were in the workforce. The average age in both years was about 29. The average marital duration was 8 years. Figure 4 shows the distribution for the women in our sample of the number of children reported to have died. In 1900, 69 percent of women had had no children die.

One significant issue when considering mortality is how to combine the experiences of women and children of different ages. Following Trussell and Preston (1982), we create a mortality index. Our index is at the level of the individual mother and compares her actual child mortality to her expected child mortality for her martial duration. The latter is calculated using the Coale and Demeny West model level 13. The index has the advantage of being simple to interpret, since the mean mortality experience is 1.[22]

[21] 1900 enumerators instructions are from http://www.ipums.umn.edu/usa/voliii/inst1900.html#136.

[22] The index could in principle have problems, because of difference in national mortality declines. According to Preston and Haines (1992), "Because the national mortality decline was relatively slow in the years preceding 1900

The 1900 child mortality rates to age 5 implied by the mortality index – 0.175 – is somewhat higher than, for example, white infant mortality rates reported by Haines (2005). In 1895 and 1904, the white infant mortality rates were 0.111 and 0.097. The difference arises for at least three reasons. First, we are examining child and not just infant mortality. Second, 10 percent of our sample was nonwhite and nonwhites, who were predominantly black, had higher infant mortality rates than whites. Third, our sample is all children born to mothers up to age 45, thus some children were born during periods of higher infant mortality. For instance, the white infant mortality rates in 1880 and 1890 were 0.215 and 0.151.

Urban-rural differences in infant and child mortality during this period have been widely discussed, and we observe these in our sample as well (Preston and Haines (1991))[23]. Table 3 summarizes the urban and rural averages for children ever born, children that die, and the maternal mortality index for the 1900 census. In 1900 the mortality index for rural women was statistically significantly lower than the mortality index for urban women.

The foregoing comparison of urban-rural mortality rates does not, however, control for the mother's characteristics or variation in state climates. It is important to control for variation in climate across states, because this is known to affect both adult and child mortality. We use three state variables – average temperature, average precipitation, and months of extreme drought. Generally mortality is thought to be declining in temperature and increasing in precipitation. Months of extreme drought captures the disease environment in very dry areas.[24]

To better understand child mortality, we turn to regression analysis. One issue we have not yet addressed is the most appropriate econometric approach. Figure 4 suggests that either negative binomial estimation on the raw count data might be appropriate. This would imply a Tobit approach might be appropriate for the mortality index, because actual child mortality in 69 percent of the cases was 0, causing the index to be zero. The data is, however, not really censored in the classical sense. We use ordinary least squares regressions primarily for convenience of interpretation. Our results are remarkably robust to the choice of estimation technique.

… the biases of intergroup comparisons should be minor.", p. 90. In our subsample, the mean mortality experience was slightly lower than for 1900 as a whole 0.909, so we recalibrated it so the mean was 1.

[23] Urban is defined as cities of 2,500 residents or more.

[24] The variables were constructed by aggregating the National Climatic Data Center county-level data available on Price Fishback's website: http://www.u.arizona.edu/~fishback/.

In column 1 of Table 4, we regress the maternal mortality index on maternal characteristics and husband's occupation. We include eight dummy variables for husband's occupation and ten dummy variables for the mother's birthplace. The omitted variables are farmer and native born. In column 2, we add the three measures of state climate to the variables in column 1. In column 3, we add a dummy variable for urban to the variables in column 2. Urban is defined by the census as a town of 2,500 or more. The relatively small increases in fit as measured by R-squared from the addition of the state climate and urban variables suggest that maternal characteristics and husband's occupation explain most of the variation in the mortality index. All of the maternal characteristics, with the exception of the birthplace dummies, are significant at the five percent level or better. The mortality index was lower for women with fewer children, who had been in longer marriages, were white, could read, were in the middle of the age distribution, were not working, and owned their own home. In columns 1 and 2, all of the eight (unreported) coefficients on husbands' occupational categories implied significantly greater mortality than which was farmer. Once we control for whether the location was urban, the effects are smaller and not always significant. The urban penalty in 1900 was a 12.1 percent increase in the mortality index relative to the sample average.[25]

5. The Effect of Lead Pipes on Child Mortality

In this section, we begin by discussing a number of issues related to the identification of the effects of lead and then present the main results.

Cross Sectional Identification

In principle, there are two possible ways to identify the effect of lead pipes on infant mortality – using cross sectional data or time series data. After describing our approach to the cross sectional identification, we will discuss two types of time series identification.

To examine the effect of lead pipes, we first determined, for every city identified by name in the census sample, whether a city had lead service pipes or not. Data on this comes from The Manual of American Waterworks, 1897.[26] All but two of the cities were listed in the manual, as were many other cities not specifically identified in the census sample. For 21 of the 170 cities identified in the census sample, the listing in the manual did not specify the type of service pipes.

[25] All increased mortality risks are relative to the sample average of 1. The actual increase relative to unaffected women will be slightly higher.

[26] There were both earlier and later versions of the manual. The earlier version (1892) was incomplete, and the later version (1915) did not list the type of service pipes.

Table 5 shows for the census regions, their population share and the share of women in our sample living in cities that are listed as having lead service pipes. Overall, 22 percent of the women in our sample in 1900 lived in cities known to have lead pipes.

One issue is that a few cities not specifically identified in the census sample had lead pipes. In the fourth column of Table 5, we impute whether a city had lead pipes based on the city population for cities not listed by name in the census sample. The imputed values are based on Table 1. This expands the percentage of women in our sample who lived in cities with lead pipes to 29 percent in 1900.

There are at least two sources of bias in the coding of lead. First, if a city adopted or ceased using lead service pipes after 1897, the city will be miscoded and the effect of lead will be biased downward. Second, 32 of the 170 cities in 1900 were listed as having a mix of lead and some other type of service pipes. We classify these together with the 72 cities that only listed lead as known lead cities.[27] Depending on the mix of lead and other service pipes, this will tend to bias down any effect of lead.

If we were to find a positive coefficient on lead in a regression of child mortality, the question is whether the coefficient on lead pipes is capturing just the effect of lead pipes or some other features of the city, such as a poor disease environment. There are two possible approaches to resolving this problem. One is to use city size together with state climate variables to approximate the city level disease environment. The advantage of controlling for city disease environments with a quadratic of population and average state-level temperature and precipitation is that it allows us to use all of the data. The disadvantage is that these variables may or many not provide a good fit. One lingering concern is that cities with lead pipes east of the Mississippi are still somehow different from both similar sized cities without lead pipes east of the Mississippi and similar sized cities with and without lead pipes west of the Mississippi. In the second approach, rather than proxy for city-level disease environment, we use the death rate from tuberculosis per 100,000 for the city in 1900.[28] We use the tuberculosis rate, because it captures the overall disease environment. For example, tuberculosis is highly correlated with the city death rate; yet it is not a leading cause of death for young children. This limits the sample to

[27] They were classified together, because we already have 10 lead-region combinations. There were not enough observations for mixed lead cities (32) to identify an additional 5 mixed lead combinations.

[28] Ideally these measures of the disease environment would be for an earlier time period, but the first reliable data for registration cities begin in 1900.

cities explicitly identified in the census for which we were able to obtain data on the city disease environment.

In both cases, we exploit regional differences in the impact of lead on child mortality to identify the effect. As we discussed, the toxicity of lead pipes depended heavily on the properties of the water running through them. For example, we would expect lead pipes to have a more severe impact east of the Mississippi than west of the Mississippi, because of the acidity and hardness of the water. Consider four cities, each with populations of 100,000. Let one city be in the East and have lead pipes, one city be in the East and not have lead pipes, one city be in the West and have lead pipes, and one city be in the West has not have lead pipes. If mortality was a function of city size, we would expect, all else equal that women in the four cities would have similar mortality experiences. If mortality was a function of lead, however, the women in the East with lead pipes should have elevated mortality. Given variation in city sizes, locations, and use of lead pipes, we can separate the city size and lead hypotheses.

What types of cities use lead pipes? In other words, is the adoption of lead pipes truly exogenous? There are two sources of evidence on this. The first is the evidence from Table 1, which includes a large number of cities. The use of lead pipes is clearly increasing in city size. The second is the evidence from the smaller sample of cities that were i) identified by name in the census, ii) listed in The Manual of American Waterworks, 1897 and the type of service pipes was specifically identified. Table 6 presents logit regressions of lead pipes on city characteristics. In the first column, we include just the log of city size. The coefficient is positive and significant. In the second column, we add state temperature and precipitation. Neither is significant, and the coefficient on log of city size remains positive and significant. In the third regression, we include whether the city is located east or west of the Mississippi. The coefficient on this dummy variable is not significant, but the coefficient on monthly precipitation is now negative and significant. The reason for this is unclear. Finally, having adopted lead pipes prior to 1897 is uncorrelated with 1900 tuberculosis rate. Thus, it would appear that conditional on city size and precipitation, the adoption of lead pipes by cities was approximately random. We believe that adoption of lead pipes can be treated as exogenous. For comparison purposes, we will present instrumental variables results.

Time Series Identification

An alternative approach to identifying the effects of lead would be to examine mortality rates in cities that had recently adopted or abandoned lead pipes. Although the sample of cities that adopted pipes is large, time series data are not available on infant and child mortality prior to 1900. Even if such data were available, the data would not include maternal characteristics. The evidence we discussed earlier on lead poisoning in Milton and Lowell suggests that the introduction of lead pipes probably did cause infant and child mortality to spike, since it caused adult mortality to do so. Further, using detailed data collected by the Massachusetts Department of Health, Troesken (2003) shows that cross sectional infant death rates for children ages 0-1 and ages 1-2 were strongly increasing in the percentage of water mains that were newly installed, but only in cities using lead service pipes. This occurred because oxidization tended to reduce the amount of lead leaching into the water over time. Thus, the available evidence suggests that lead pipes were causally associated with death. Unfortunately, we do not have information on when water mains or service pipes were installed, so we cannot control for this directly.

One could in principle also examine the small number of cases where lead pipes were abandoned, which is to say cities literally dug up their lead pipes (Troesken 2006). Lead-pipe abandonment, however, does not offer as clean an experiment as one might think for two reasons. First, since lead solder was commonly used when joining non-lead service pipes, it is possible that replacing lead service pipes could increase lead exposure. This is particularly likely if the lead service pipes had been in use for some time, and so had oxidized. Second, recall the four pathways through which infants and young children were exposed to lead: i) maternal water consumption in utero, ii) maternal bone leaching, ii) maternal water consumption and bone leaching via breast milk and iv) dilution of cows milk or transmission via cows milk if the cow is exposed to lead. Even if lead exposure through maternal water consumption is reduced to zero, maternal bone leaching will continue. Thus, death rates might not fall sharply in the short term.

Results

Table 7 summarizes the (known) lead and no lead averages for children ever born, children that die, and the mortality index for the 1900. As expected, the difference between the mortality in known lead and no lead areas is statistically significant in 1900.

Table 8 reports the regression that control for mother's characteristics, father's occupation, two measure of a state's climate, the natural log of city size, a dummy variable for a city size of

zero, and in columns 2-4, measures of whether a city had lead pipes or not. The unreported coefficients on the mother's characteristics are extremely similar to the coefficients reported in Table 4.

In column 1, where we control for region but not lead, the coefficient on the natural log of city population is statistically significant. In column 2, where we introduce five census regions and five lead variables, the coefficients on some regions and lead variables are significant and others are not. The important thing to note is the basic pattern. The coefficients for lead on census regions east of the Mississippi are positive or negative and insignificantly different than the Northeast and the coefficients on census regions west of the Mississippi are negative and significantly different than the Northeast. This pattern is consistent with the eastern states generally having relatively soft water (0-60), high levels of acid rain, and, to varying degrees, water sources with low acid neutralization capabilities. We use this information to aggregate the census regions into two superregions, one east of the Mississippi and one west of the Mississippi.

In column 3, we include three dummy variables that indicate superregion and lead. The natural log of city size remains statistically significant and positive, but the coefficient is substantially smaller than in column 1. As for lead dummies, the omitted variable is East-No Lead. Relative to East-No Lead, East-Lead has an increased mortality risk of 9.3 percent. The difference between East-No Lead and East-Lead is statistically significant. West-Lead is not significantly different from East-No Lead, which supports our earlier findings regarding the effect of lead in the West. Finally, West-No Lead is positive and statistically significantly different from East-No Lead. At 10.2 percent, the increased mortality risk from being in West-No Lead that is roughly the same size as the increased mortality risk from being in East-Lead. We suspect this reflects greater isolation in more rural parts of these regions. Indeed, as we demonstrate below, once the sample is restricted to towns with populations greater than 2,500, this otherwise odd result vanishes.

In columns 4 and 5, we exclude cities with populations of zero and cities with populations of less than 2,500. Excluding cities with population zero cuts the sample size roughly in half. The effects in column 4 are, however, very close to what we found in column 3. That is, the coefficients on East-Lead and West-No Lead are positive, significant, and of similar magnitude. Again, the coefficient on the natural log of city size remains statistically significant and positive.

In column 5 when we exclude cities with populations of less than 2,500, the coefficient on West-No Lead becomes insignificant, which is consistent with our hypothesis that it reflects the greater isolation in small towns west of the Mississippi. The coefficient on East-Lead, however, remains significant. The increased mortality risk – 9.6 percent – is similar to what we found in column 3. The coefficient on the natural log of city size remains marginally statistically significant and positive.

Overall, in 1900 the mortality index for women who lived east of the Mississippi and had lead pipes was 9.3 percent higher than the sample average. In 1900, women in cities known to have lead pipes East of the Mississippi were 19 percent of the United States population. If we include women who we impute had lead, this increases to 24 percent of the United States population. This represents roughly 44,000 child fatalities due to lead.[29]

We know that any effects of lead that we find are biased downward, because all cities for which we do not have information on lead pipes are coded as zeros. To address this issue, in Table 9, we limit our sample to cities for which we observe the type of service pipe. This reduces the sample size to less than on-quarter the size of the sample in columns 1-3 of Table 8. Because of the small sample size and the fact that a very large share of the observations are for the East-Lead, we reduce the region-lead variables from four to two. One is East-Lead and the other (omitted) variable includes West-Lead, West-No Lead, and East-No Lead.

In column 1 of Table 9, the coefficient on East-Lead is positive, significant, and of similar magnitude to the coefficients in Table 8. The coefficient on the natural log of city size is not significant, however, probably because of the diminished variation in city size. In column 2, we control for the tuberculosis rate in 1900. This diminishes the sample size slightly relative to column 1. To the extent that the tuberculosis rate captures the contemporaneous, and possibly the retrospective, health environment, we would expect the coefficient on the natural log of city size not to be significant, since we were using city size as a proxy for the health environment. City size is not statistically significant.

One remaining concern is that the use of lead pipes is endogenous. In columns 3 and 4 of Table 9, we present the results of instrumental variable estimates. As we found in Tables 1 and 6, the most natural instrument for lead is the natural log of city size. City size is a strong

[29] 70,000 children x 1-200 sample x 0.24 x 0.013 increase in mortality (as opposed to the mortality index) = 44,000.

instrument; the only strong instrument in the set of possible instruments that we explored.[30] The validity of the results depends on whether one believes the exclusion restriction. The coefficient on the natural log of city size is not significant in columns 1 and 2, which is reassuring, but not necessarily compelling. It is quite plausible, however, that state temperature, precipitation, and drought either alone or together with the city-level death rate from tuberculosis capture all the relevant dimensions of the city disease environment for this sub-sample of larger cities. The coefficients on lead in the second stage are statistically significant and large. The women in eastern cities with lead pipes have mortality indices that are 31.5-35.8 percent higher than the sample average.

The 1910 Census also asked women about children ever born and children surviving. If we follow the 1900 cohort of women forward by adjusting the age and marriage duration, we find in unreported regressions similar effects of lead if we restrict attention to cities for which we know in whether they had lead service pipes or not in 1897 (e.g. the cities in Table 9). If we add the younger women, however, the effects are not always significant.[31] This probably reflects the gradual oxidization of lead service pipes, most of which would have been put in place prior to 1900. Oxidization would have diminished the amount of lead being leached over time. Thus, younger women would have received lower lead exposure than their older counterparts.

Having found substantial increases in women's mortality indices associated with lead, one question is: How much of the urban penalty is actually attributable to lead and not to disease environments of bigger cities? We find that that lead accounts for 47 percent of the urban penalty.[32]

Comparison with Detailed Studies of Massachusetts

Apart from the instrumental variables estimates, the negative effects of lead we find are smaller in magnitude than the negative effects Troesken (2003) finds for Massachusetts. For example, he finds that towns with lead pipes had mortality rates for children 0-1 that were 39

[30] We examined a number of state characteristics such as the slope, depth of soil, and share of clay in the soil. We also examined the characteristics of the water system (Troesken 2003). The values of the F-tests in the first stage were well below 10.

[31] The instrumental variable regressions are significant for both cohorts.

[32] We column 3 in Table 4 with the identical regression to which you add a dummy variable for whether a city is East-Lead or not. The urban penalty falls from a 12.1 percent increase in the mortality index to a 6.4 percent increase in the mortality index.

percent higher than towns without lead pipes. Similarly, mortality rates for children 1-2 were 24 percent higher in towns with lead pipes than in towns without lead pipes.

The difference in the magnitudes can be at least partially explained differences in the data. First, because of the very detailed data maintained by the Massachusetts Department of Health, Troesken is able to link current death rates to the town's use of lead pipes. Because of the nature of the census data, we are linking retrospective death rates for women currently living in a particularly city with that city's use of lead pipes. Recall the substantial shift in the population of women living in towns with lead pipes between 1900 and 1910. Some of this represented literal migration and some of it represented the growth of towns to a size where they warranted a municipal water system. If 25 percent of the women in lead areas had had their children before the introduction of lead pipes in their town or in other localities without lead pipes, the magnitudes of our measured effects would rise by 33 percent to 12 or 13 percent.

Second, Troesken has detailed information on the age at which death occurs. Troesken shows large and statistically significant negative effects of lead for children 0-2, but positive and insignificant effects for children 2-4 and 10 and older. This suggests that some of the children who were killed by lead might have later been killed by other diseases. In contrast, this study examines retrospective death by all causes and does not include information on the ages of the children who died. Thus, we estimate the net effect of lead on the death rate, and not the gross effect as Troesken does. Finally, Troesken did not have information on mothers' characteristics or fathers' occupation. We control for these characteristics directly.

6. Conclusion

The results here indicate that the use of lead water pipes increased the mortality index by 9.3 percent in 1900 for women living in cities with lead pipes. This implies that the use of lead water pipes resulted in tens of thousands childhood deaths during the early twentieth century. Having shown this, it is important to draw a distinction between partial and general equilibrium effects. Clearly the partial effect was negative; many lives could have been saved by using service pipes composed of iron, cement, or steel. However, the general equilibrium effects are less clear. If avoiding the use of lead service pipes delayed the introduction and extension of urban water supplies, many more children and adults would have died from waterborne diseases

such as typhoid fever and infantile diarrhea and deaths from these causes likely would have exceeded deaths from lead poisoning. Alternatively, the upfront monetary costs of iron, cement, and steel service pipes were actually lower than those associated with lead. It is, therefore, possible that using pipes of other materials might have hastened the expansion of urban water systems and promoted the willingness of homeowners (who often had to pay for the service pipe) to connect to local water supplies. Having said this, Troesken (2003; 2006) explicitly tests this proposition and finds no evidence to support the idea using non-lead pipes hastened the expansion of urban water systems.

One area that we would like to explore in future research is the relationship between water lead and adverse outcomes in terms of educational attainment, literacy, and criminal behavior. In particular, it would be desirable to understand how water affected the children who survived in utero and during the early stages of development. It is well known that lead exposure reduces IQ and gives rise to behavioral problems in children and young adults. Moreover, it seems likely that if there were enough lead in drinking water to induce death among developing infants there likely would have been enough lead to inhibit the neurological development of the surviving children. We are pursuing two possible avenues in this direction. Although data on from intelligence tests during the early twentieth century are scarce and not especially reliable even when available, it might be possible to use census data on literacy and recent moves to explore the effects of lead on education. We are also searching for state-level education departments circa 1910 for evidence on test scores and performance across cities with high and low water-lead levels.

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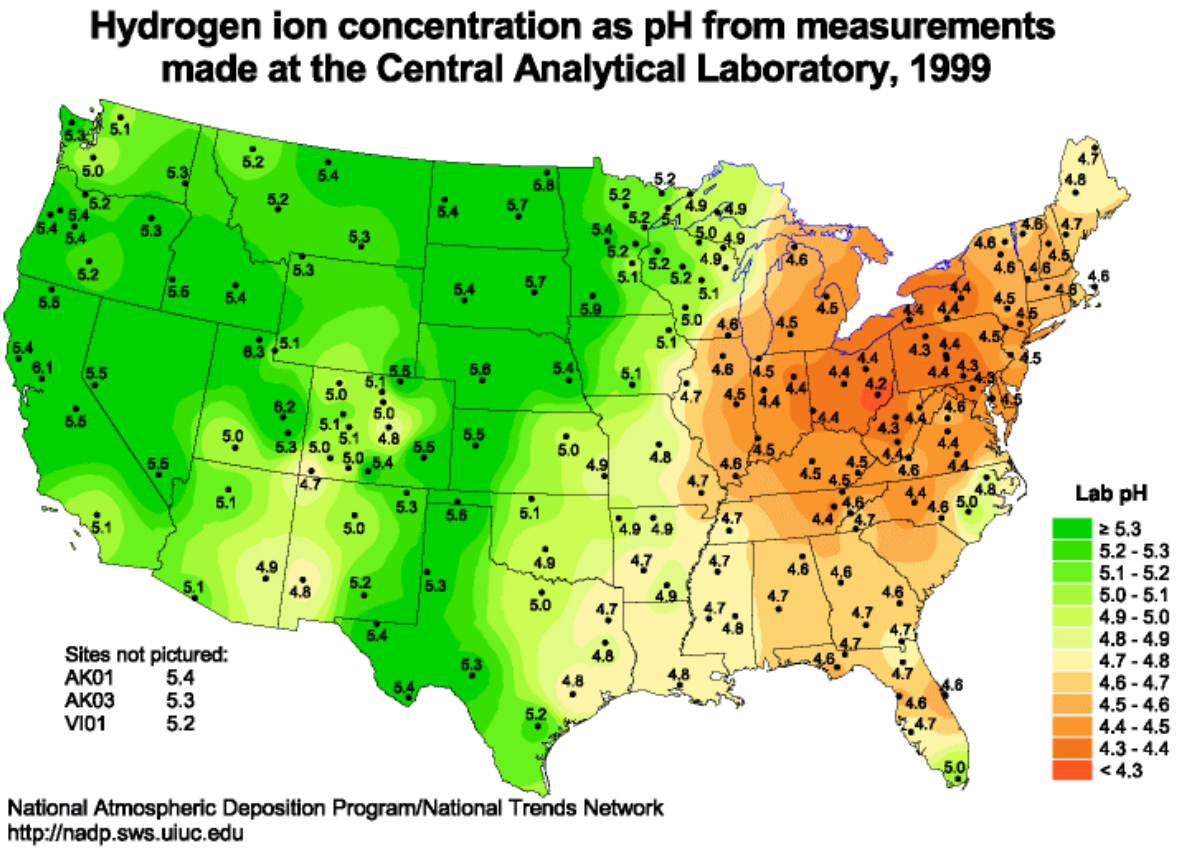
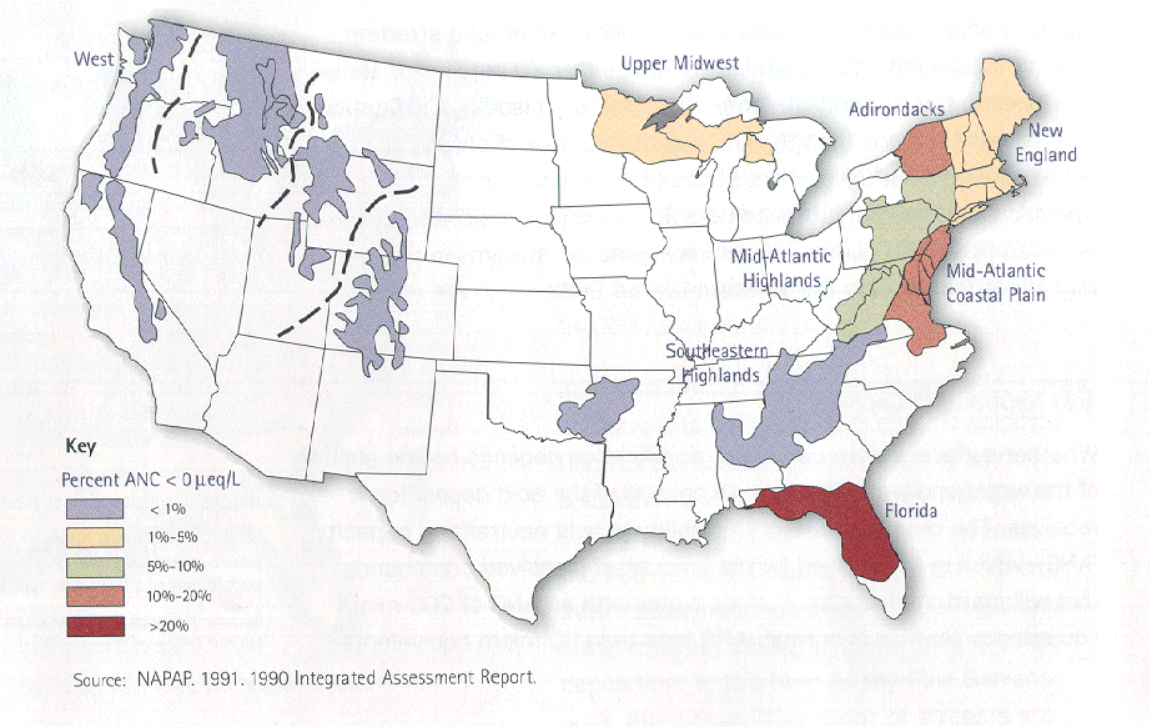
Figure 1: Acid Rain in the United States



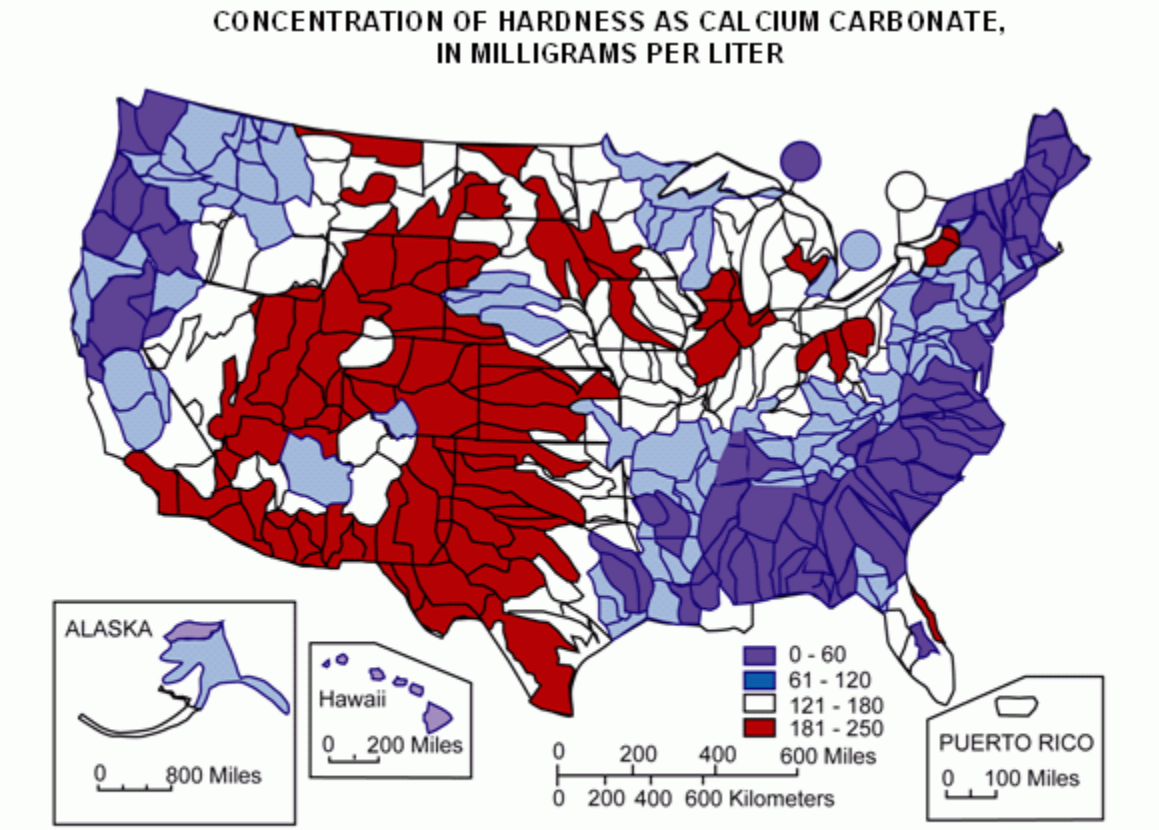
Notes: Image from http://www.epa.gov/castnet/images/ph_map.gif

Figure 2: Percentage of Acidic Surface Waters in Surveyed Regions



Notes: Image from http://www.epa.gov/airmarkets/cmap/images/fig14x.gif

Figure 3: Water Hardness in the United States



Notes: Image from http://ga.water.usgs.gov/edu/graphics/HardnessMap.gif

Figure 4: Distribution of Mothers by the Number of Children Who Have Died, 1900

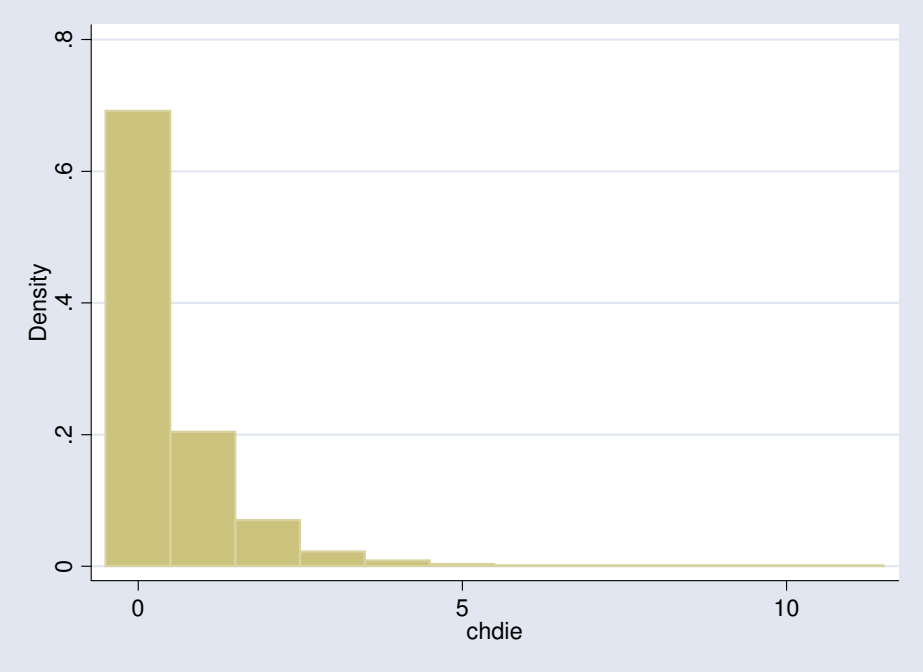


Table 1: Use of Lead Service Pipes by City Size

		Cities using		
City size in 1900	no. of cities	only lead	lead & other	no lead
Population > 300,000	16	8 (50%)	7 (44%)	1 (16%)
30,000 < Pop < 300,000	107	55 (51%)	22 (21%)	30 (28%)
8,000 < Pop < 30,000	156	46 (29%)	36 (23%)	74 (47%)
Population < 8,000	518	100 (19%)	72 (14%)	346 (67%)
All towns and cities	797	209 (26%)	137 (17%)	451 (57%)

Notes: From Troesken and Beeson (2003).

Table 2: Summary Statistics for 1900

	1900
Children die	0.469
	(0.866)
Children ever born	2.881
	(1.759)
White	0.896
Literate	0.896
Urban	0.400
Own home	0.386
Working	0.035
Age	29.093
	(5.614)
Duration marriage	7.858
	(3.579)
Observations	24,483

Notes: These averages are computed at the level of the mother. Standard deviations are in parentheses. Standard deviations are not reported for dummy variables.

Table 3: Urban-Rural Averages in 1900

	1900	1900
	Rural	Urban
Children born	4.09	3.72***
	(1.98)	(1.91)
Children die	0.71	0.79***
	(1.07)	(0.19)
Mortality Index	0.937	1.107***
	(1.376)	1.552
Observations	44,252	26,292

Notes: These averages are computed at the level of the child. Standard deviations are in parentheses. *, **, and *** denote statistical significance at the 10, 5, and 1 percent levels.

Table 4: Mortality and Urban Locations

	1	2	3
	Mortality Index	Mortality Index	Mortality Index
Children born	0.176***	0.175***	0.177***
	0.014	0.015	0.015
Duration of marriage	-0.042***	-0.042***	-0.042***
	0.003	0.003	0.002
White	-0.338***	-0.313***	-0.310***
	0.047	0.048	0.047
Read	-0.116***	-0.108***	-0.118***
	0.031	0.025	0.021
Age	-0.111***	-0.109***	-0.111***
	0.008	0.008	0.009
Age squared	0.0019***	0.0018***	0.0018***
	0.0001	0.0001	0.0001
Working	0.232**	0.211**	0.208**
	0.103	0.094	0.094
Own home	-0.076**	-0.070**	-0.056**
	0.032	0.030	0.023
State temperature		-0.002	-0.0006
		0.003	0.0023
State precipitation		0.020*	0.017*
		0.011	0.010
State drought		-0.0031***	-0.0027***
		0.0007	0.0007
Urban			0.120***
			0.038
Birthplace FE	Y	Y	Y
Husband occ FE	Y	Y	Y
Observations	70,544	70,544	70,544
R-squared	0.0720	0.0724	0.0733

Notes: All regressions are run with robust standard errors clustered by city. Standard errors are reported below point estimates. *, **, and *** denote statistical significance at the 10, 5, and 1 percent levels. The mother's birth location fixed effects are as follows: birth location = 1 if born in United States (omitted), birth location = 2 if born in Canada or the Atlantic Islands, birth location = 3 if born in Northern Europe, birth location = 4 if born in the United Kingdom, birth location = 5 if born in Ireland, birth location = 6 if born in Germany, birth location = 7 if born in Poland or the Russian Empire, birth location = 8 if born in Italy, birth location = 9 if born in Southern Europe excluding Italy, birth location = 10 if born in Central or Eastern Europe excluding Poland and Germany, birth location = 0 for all other locations. For more details, see http://www.ipums.umn.edu/usa/pethnicity/bplgb.html. The husband's occupation fixed effects are as follows: occupation = 1 if occupation is professional/technical, occupation = 2 if occupation is farmer (omitted), occupation = 3 if occupation is manager, official, proprietor, occupation = 4 if occupation is clerical and kindred, occupation = 5 if occupation is sales worker, occupation = 6 if occupation is craftsman, occupation = 7 if occupation = operative, occupation = 8 if occupation is service worker or farm laborer, occupation = 9 if occupation is a non occupational response or blank. For more details, see http://www.ipums.umn.edu/usa/pwork/occ1950b.html

Table 5: Lead Exposure by Region of Women in Sample in 1900

Region	Pop Share	Share with known lead	Share total pop with lead	Share with known/ imputed lead	Share total pop with lead
1900					
New England & Mid Atlantic	0.27	0.41	0.11	0.50	0.14
East North Central	0.22	0.29	0.06	0.34	0.07
West North Central	0.13	0.13	0.02	0.19	0.02
South Atlantic & East South Central	0.23	0.06	0.01	0.12	0.03
West South Central & Mountain & Pacific	0.15	0.10	0.01	0.17	0.02
Total	*1.00*		*0.22*		*0.29*

Table 6: Determinants of Use of Lead Pipes (Marginal effects reported)

	1	2	3	4
	Lead	Lead	Lead	Lead
Ln(citypop)	0.112*** 0.040	0.112*** 0.042	0.120*** 0.043	0.147*** 0.052
State temperature		-0.0003 0.0083	0.0029 0.0082	0.0076 0.0109
State precipitation		-0.096 0.076	-0.156* 0.088	-0.193* 0.109
West of Mississippi			-0.179 0.168	-0.216 0.213
Tuberculosis 1900				0.0006 0.0007
Observations	147	147	147	118
R-squared	0.0465	0.0606	0.0664	0.1001

Notes: *, **, and *** denote statistical significance at the 10, 5, and 1 percent levels. Only observations in which the cities are listed by name in the census and are specifically identified as having lead service pipes or not having lead service pipes are used.

Table 7: No Lead and Known Lead Averages in 1900

	1900	1900
	No Lead	Known Lead
Children born	4.01	3.73***
	(1.98)	(1.91)
Children die	0.72	0.84***
	(1.09)	(1.23)
Mortality Index	0.960	1.153***
	(1.28)	(1.05)
Observations	55,903	14,641

Notes: These averages are computed at the level of the child. Standard deviations are in parentheses. In this table and all tables that follow, *, **, and *** denote statistical significance at the 10, 5, and 1 percent levels.

Table 8: Mortality Index and Lead Service Pipes

	1	2	3	4	5
	Mortality Index	Mortality Index	Mortality Index	MI Citypop>0	MI Urban
East North Central	-0.063*** 0.018	-0.045*** 0.011			
West North Central	-0.015 0.044	0.029* 0.017			
South Atlantic & East SC	-0.104*** 0.032	-0.096*** 0.029			
West SC & Mtn. & Pacific	-0.000 0.043	0.034 0.026			
Lead		0.077 0.053			
Lead-ENC		-0.030 0.045			
Lead-WNC		-0.221*** 0.053			
Lead-SAESC		0.146 0.135			
Lead-WSCMP		-0.185** 0.089			
East-Lead			0.093** 0.045	0.107** 0.043	0.095*** 0.046
West-No Lead			0.102*** 0.016	0.140*** 0.034	0.072 0.057
West-Lead			-0.041 0.059	-0.016 0.055	-0.017 0.059
Ln(City Pop)	0.023*** 0.003	0.017** 0.007	0.017** 0.006	0.016** 0.006	0.015* 0.009
Zero Pop	0.026 0.016	0.010 0.021	0.006 0.019		
State temperature	0.0013 0.0010	0.0009 0.0010	-0.0021 0.0015	-0.004** 0.002	-0.006** 0.002
State precipitation	0.041*** 0.012	0.046*** 0.010	0.051*** 0.009	0.066*** 0.021	0.077** 0.031
State drought	-0.0014* 0.0008	-0.0017** 0.0008	-0.0025*** 0.0007	-0.0025* 0.0015	-0.002 0.002
Maternal characteristics	Y	Y	Y	Y	Y
Birthplace FE	Y	Y	Y	Y	Y
Husb. occ FE	Y	Y	Y	Y	Y
Observations	70,403	70,403	70,403	32,846	26,009
R-Squared	0.0745	0.0751	0.0747	0.0856	0.0956

Notes: *, **, and *** denote statistical significance at the 10, 5, and 1 percent levels. All regressions are run with robust standard errors clustered by city. Standard errors are reported below point estimates. The omitted region in columns 1 and 2 is New England/Mid Atlantic. The omitted region-lead variable in columns 3-5 is East-No Lead. ENC is East North Central; WNC is West North Central; SAESC is South Atlantic and East South Central; WSCMP is West South Central, Mountain, and Pacific.

Table 9: Mortality Index for Cities Where the Type of Service Pipe is Known

	1	2	3	4
	Mortality Index	Mortality Index	IV	IV
East-Lead	0.106**	0.096*	0.314**	0.357***
	0.050	0.058	0.126	0.134
Ln(City Pop)	0.016	0.024		
	0.011	0.015		
State temperature	-0.0055	-0.0060	-0.0014	-0.0007
	0.0041	0.0040	0.0053	0.0055
State precipitation	0.041	0.049	-0.014	-0.022
	0.040	0.042	0.053	0.053
State drought	-0.0059**	-0.0072***	-0.0087***	-0.010***
	0.0023	0.0023	0.0029	0.003
Tuberculosis		-0.0006		-0.0004
		0.0004		0.0004
Maternal characteristics	Y	Y	Y	Y
Birthplace FE	Y	Y	Y	Y
Husband occ FE	Y	Y	Y	Y
Observations	16,735	15,803	16,735	15,803
R-Squared	0.1000	0.1002		

Notes: *, **, and *** denote statistical significance at the 10, 5, and 1 percent levels. All regressions are run with robust standard errors clustered by city. Standard errors are reported below point estimates. Birthplace FE were reduced to a dummy variable for whether or not the woman was born in the United States or not. Husband's occupation FE were reduced by creating a single category for occupational categories 3-8. The F-test for the first stage is 12.87 (column 3) and 10.24 (column 4)