Re-Examining the Contribution of Public Health Efforts to the Decline in Urban Mortality

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Abstract. Using data on 25 major American cities for the period 1900-1940, we explore the effects of municipal-level public health efforts that were viewed as critical in the fight against food- and water-borne diseases. In addition to studying interventions such as treating sewage and setting bacteriological standards for milk, which have received little attention, we provide new evidence on the effects of water filtration and chlorination, extending the work of previous scholars. Although water filtration is associated with an 11-12 percent reduction in infant mortality, none of the other interventions under study appear to have contributed to the observed mortality declines.

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1. INTRODUCTION

Since the mid-19th century, mortality rates in the Western world have plummeted and life expectancy has risen dramatically. Sometimes referred to as the mortality transition, this development is widely recognized as one of the most significant in the history of human welfare (Fogel 2004). Two features characterize the mortality transition. First, it was driven by reductions in infectious diseases and diseases of infancy and childhood (Omran 2005; Costa 2015). Second, it was concentrated in urban areas: at the turn of the 20th century, major cities were unsanitary havens of pestilence; by 1940, urban mortality rates were comparable to those of rural areas (Haines 2001; Cain and Hong 2009).

Traditionally, economists have attributed the mortality transition to rising incomes, better nutrition and the onset of modern economic growth (McKeown and Record 1962; McKeown 1976; Fogel 1997, 2004). More recent reviews of the literature emphasize the role of public health efforts, especially those aimed at purifying the water supply. For instance, Cutler et al. (2006) argue that public health efforts drove the dramatic reductions in food- and water-borne diseases at the turn of the 20th century. Similarly, Costa (2015) argues that clean-water technologies such as filtration and chlorination were "the biggest contributor[s] to the decline in infant mortality" (p. 559), but acknowledges that the effects of other public health interventions "remain understudied" (p. 546).

Using data on 25 major American cities for the years 1900-1940, the current study revisits the causes of the urban mortality decline at the turn of the 20th century. Specifically, we conduct a statistical horse race that attempts to distinguish the effects of ambitious, often extraordinarily expensive (Costa 2015, p. 554), public health interventions aimed at controlling mortality from food- and water-borne diseases. Following previous researchers (Troesken 2004; Cutler and Miller 2005; Beach et al. 2016; Knutsson 2018), we explore the extent to which filtering and chlorinating drinking water contributed to the decline in typhoid mortality observed during the period under study and, more generally, to the observed declines in total and infant mortality. In addition, we explore the effects of several other municipal-level efforts that were, at the time, viewed as critical in the fight against typhoid and other food- and water-borne diseases (Meckel 1990; Levitt et al. 2007; Melosi 2008) but have not received nearly as much attention from modern-day researchers. These interventions include: projects designed to deliver clean water from further afield such as aqueducts and water cribs; the treatment of sewage before its discharge into lakes, rivers and streams; requirements that milk sold within city limits meet strict bacteriological standards; and requirements that milk come from tuberculin-tested cows. Because the urban mortality transition was

characterized by substantial reductions in infant and childhood mortality (Omran 2005), and because exclusive breastfeeding was not the norm during the period under study (Wolf 2001, 2003), improvements in milk quality seem a particularly promising avenue to explore.

Consistent with the results of Troesken (2004), Cutler and Miller (2005) and Beach et al. (2016), we find that filtering the municipal water supply sharply reduced typhoid mortality. Water filtration is also associated with a (statistically insignificant) 1-2 percent decrease in total mortality and an 11-12 percent decrease in infant mortality. These latter estimates, however, are considerably smaller than those found by previous researchers, including Cutler and Miller (2005), the authors of the most influential study in this literature. Finally, we find little evidence that other municipal-level interventions such as chlorinating the water supply, building sewage treatment plants, and setting bacteriological standards for milk were effective.

The remainder of the paper is organized as follows. We begin with an overview of the mortality transition and public health efforts to control food- and water-borne diseases. In Section 3, we describe our data and empirical strategy; in Section 4, we report our principal estimates; and in Section 5 we explore their robustness and consider extensions to our baseline regression model. In Section 6, we directly compare our water filtration estimates to those of Cutler and Miller (2005) and document why they are so different. Using their original data and specification, we find that the estimated effect of filtration on infant mortality shrinks by more than two-thirds when a series of transcription errors are corrected. Section 7 concludes.

2. BACKGROUND

Americans experienced an unprecedented improvement in health and longevity in the late 19th and early 20th centuries (Haines 2001; Riley 2005; Troesken 2015). During the period 1850-1950, life expectancy at birth among whites increased by 75 percent from 39.5 to 69 years; among non-whites, life expectancy more than doubled from 23 to 60.8 years (Troesken 2015, p. 10).

This improvement in health and longevity was accompanied by radical changes in the U.S. disease profile (Jones et al. 2012). Before 1900, the leading causes of death were food- and water-borne diseases (e.g., diarrhea, enteritis and typhoid) and respiratory diseases (e.g., influenza and pulmonary TB). By 1940, deaths from these diseases were on the wane; chronic conditions such as heart disease and cancer had become the leading causes of death (Jones et al. 2012).

Ferrie and Troesken (2008) use data from Chicago to illustrate mortality trends in major U.S. cities. Between 1850 and 1925, the Chicago mortality rate fell by 60 percent, driven by the near

eradication of food- and water-borne diseases (which disproportionately affected infants and children) and reductions in mortality from respiratory diseases. As Chicago transitioned from a high- to low-mortality environment, so-called "modern diseases" replaced food-borne, water-borne and respiratory diseases as the primary causes of death.[1] Boston, Philadelphia, New York and other large American cities experienced their mortality/epidemiological transitions over the same period, while similar transitions occurred roughly one-decade later in smaller American cities (Haines 2001; Ferrie and Troesken 2008).

2.1. Efforts to supply clean water

Many experts believe that, after 1900, the mortality transition in Chicago and other U.S. cities was primarily driven by a series of public health interventions aimed at reducing food- and water-borne illnesses (Cutler et al. 2006; Ferrie and Troesken 2008).[2] Yet, it is far from clear which interventions were most effective, in part because the same city would often implement several of them within a few years of each other, making it difficult to isolate the effect of any single intervention, especially when researchers take a case-study approach.[3] Below, we describe the various water-related public health efforts undertaken by U.S. cities during the period under study:

Water filtration. Chicago did not begin filtering its water supply until 1947 (Baylis 1949), but filtration plants were built by most other major U.S. cities during the period under study. Of the 25 cities in our sample, all but 8 had built filtration plants by 1940 (Table 1). Originally developed to reduce discoloration and turbidity, water filtration gained support as the field of bacteriology advanced and city governments came under increasing political pressure to protect their citizens

[1] The phrase "modern diseases" suggests that other countries have experienced (or will experience) the equivalent of the U.S. epidemiological transition (Jones and Greene 2013). This, of course, is an oversimplification. Perhaps because economic development has been uneven, the epidemiological/mortality transition has not been universal (Frenk et al. 1989).

[2] There is an argument to be made that these same public health interventions reduced the within-city (i.e., across neighborhood) variation in mortality (Costa and Kahn 2015).

[3] For instance, Chicago built 5 separate tunnels under Lake Michigan during the period 1867-1892 in an effort to draw water from beyond its heavily polluted shoreline (Reynolds 1894); in 1871, Chicago effectively reversed the current of the Chicago River, which had previously carried sewage into Lake Michigan (Ferrie and Troesken 2008); in 1900, it completed the Sanitary and Ship Canal guaranteeing that, even in the worst weather, the river flowed away from the lake (Hering and Fuller. 1907; Ferrie and Troesken 2008); the North Shore Channel, designed to flush the sewage down the Sanitary and Ship Canal, was completed in 1910 (Gustaitis 2013); in 1912, the city began treating its water with chlorine before delivery to homes and businesses but the process of chlorination was not completed until 1917 (Jennings 1923); and, in 1908, the city passed an ordinance requiring that milk sold within its limits meet a strict bacteriological standard and come from tuberculin-tested cows. As described below, this ordinance had the effect of encouraging pasteurization.

from infectious diseases (McCarthy 1987; Melosi 2008, pp. 90-94).[4] Public health experts, armed with studies showing dramatic decreases in typhoid mortality after filtration systems were put in place (Clark 1907; Johnson 1907; Sedgwick and MacNutt. 1910; McLaughlin 1912; Johnson 1913), convinced skeptical policymakers that filtering water was a sound investment (Foss-Mollan 2001, pp. 80-116; Melosi 2008, p. 94).

Chlorination. In 1908, Jersey City famously became the first municipality in the United States to disinfect its water supply by continuously treating it with chlorine (Allen 1918). The process of chlorination was simple and inexpensive: water was added to bleaching powder (calcium hypochlorite) to make a thick paste, which was then mixed with the water supply before delivery (Hooker 1913).[5] In part because it was so inexpensive, chlorination quickly gained popularity (Hill 1911). By 1928, every city in our sample was chlorinating its water supply (Table 1), although issues with taste and odor discouraged many smaller communities from adopting the technology.[6]

Clean water projects. Chicago drew its water from Lake Michigan, but the city also dumped its sewage, which carried disease-causing pathogens, directly into the lake (or into the Chicago River, which flowed into the lake). In 1867, Chicago constructed a two-mile tunnel under Lake Michigan, enabling it to extend its water crib well beyond its heavily polluted shoreline; in 1892, a four-mile tunnel was constructed (Reynolds 1894; Sells 2017).[7] Several other American cities went to even greater lengths to deliver clean water, building aqueducts and tunnels that connected distant reservoirs with their water distribution systems (Table 2). For instance, in 1914 San

[4] The simplest method of water filtration (i.e., slow water filtration) consisted of "a body of sand of sufficient thickness, underlaid or supported by a few inches of course and fine gravel or broken stone, with a proper number of pipe underdrains to collect the water that passes through the sand…" (Clark 1907, p. 765). Mechanical filtration plants, which used coagulants and smaller filter beds, proved less expensive to build and maintain than slow water filtration plants and eventually supplanted the latter (Johnson 1914a, 1914b).

[5] Between 8 and 16 pounds of bleaching powder per million gallons of water were typically used (Hooker 1913, p. 65). Within a decade of it being used to disinfect the Jersey City water supply, bleaching powder (also known as "chloride of lime") had been supplanted by liquid chlorine, which was less expensive and did not have "the disagreeable odor and corrosive influences of chloride of lime" (Bowles 1919, p. 22).

[6] According to Melosi (2008, p. 139), only one-third of all U.S. waterworks used chlorine because of "taste and odor problems" as late as 1939.

[7] Water cribs are structures designed to collect water from near the bottom of a lake, which is then carried to an onshore pumping station. After the completion of these projects, typhoid deaths fell precipitously, as did deaths from respiratory diseases such as pneumonia and TB, leading Ferrie and Troesken (2008, p. 15) to conclude that water purification efforts reduced mortality "from diseases that other[wise] would not have been classified as waterborne or even water-related..." In fact, Ferrie and Troesken (2008, p. 15) conclude that 35-56 percent of the total mortality decline in Chicago is attributable to water purification efforts.

Francisco began constructing a system of dams, conduits, hydroelectric plants and aqueducts designed to deliver clean water from the Hetch Hetchy Reservoir to the Bay Area, a distance of over 150 miles (Hanson 2005).[8]

Sewage treatment plants. At the turn of the 20th century, dilution (i.e., discharge into lakes, rivers, streams, or the ocean) was the standard method of sewage disposal. Experts argued that the "natural power of self-purification" would render waste water clean and, ultimately, drinkable (Whipple 1914, p. 638).[9] Evolving attitudes, coupled with new technologies and regulatory efforts, prompted the construction of sewage treatment plants across the country (Tarr et al. 1984).[10] By the end of 1940, 15 of the 25 cities in our sample were treating their sewage (Table 3).[11]

Previous studies in this literature have focused on estimating the effects of building and extending sewer systems (i.e., providing sewerage) as distinct from treating sewage (i.e., using chemical or biological processes to remove contaminants from waste water) before its discharge into lakes, rivers and streams. For instance, using data from Paris for the period 1880–1914, Kesztenbaum and Rosenthal (2017) show that the provision of sewerage to a neighborhood added several years to the life expectancy of its residents. Using data on 60 municipalities in Massachusetts for the period 1880-1920, Alsan and Goldin (2019) examine the effects of clean water and access to a regional sewerage system on infant and child mortality. Instead of filtration or chlorination, Massachusetts opted to provide clean water through a system of "impounding reservoirs in which

[8] During the period 1900-1940, Boston, Jersey City, New York City, Newark and Providence also built aqueducts to transport clean water from reservoirs located in protected watersheds. According to Melosi (2008, p. 57), "distant sources of supply received great attention because they offered large and dependable quantities of water, but also because they provided alternatives to polluted or infected sources in the local area." Smaller cities, however, were "hard-pressed to seek distant sources" and were therefore more likely to build filtration plants (Melosi 2008, p. 57).

[9] Despite this argument, several cities (including Chicago and Detroit) went to great lengths to ensure that their sewage was disposed of downstream from the system intake (Hering and Fuller 1907; Cain 1977; Detroit Water and Sewerage Department 2002).

[10] During the period under study, the use of chemical precipitation (which involved the addition of coagulants to separate sludge from water) waned and more efficient methods of treating sewage, including the activated sludge process, were gradually adopted (Ardern and Lockett 1914; Reynolds 1933). Providence, Rhode Island was the first city in our sample to treat its sewage. The Providence plant, built in 1901, used chemical precipitation (Nixon 1995). None of the other cities in our sample adopted this technology and the Providence plant converted to using an activated sludge process in the mid-1930s.

[11] Philadelphia and San Francisco are not counted among these 15 cities because they were treating less than 25 percent of their effluent by the end of the period under study (Mohlman 1940). We code our sewage treatment indicator as equal to 0 for cities that were treating less than 25 percent of their effluent. If we apply a higher threshold (e.g., a city must have been treating at least 50 percent of their effluent), our results are qualitatively similar. Appendix Figure 1 illustrates the rollout of the water-related interventions during the period 1900-1940 for our sample of cities.

spring floodwaters were stored" (Alsan and Goldin 2019, p. 595). These authors find that the interaction of clean water and sewerage accounted for approximately one-third of the observed decline in child mortality (i.e., mortality among children under the age of 5) from 1880 to 1920 and nearly one-half of the observed decline in infant mortality.[12] By the early 1900s, every major U.S. city had installed sewers (Cain and Rotella 2001; Melosi 2008; Hoagland 2018), precluding us from using the same regressors as were used by Alsan and Goldin (2019).[13]

2.2. Efforts to supply clean milk

Diarrheal diseases were important contributors to infant mortality in the early 1900s and experts at the time were convinced that cleaning up the milk supply was key to combatting them (Brosco 1999). Yet, aside from Olmstead and Rhode (2004a) and Komisarow (2017), modern-day researchers have not paid a great deal of attention to milk-related public health efforts undertaken during this period.[14] Below, we describe two milk-related municipal-level public health interventions, the effects of which have not been explored sufficiently by previous researchers:

Bacteriological standards for milk. Boston was the first U.S. city to require that milk sold within its limits meet a bacteriological standard (specifically, a maximum of 500,000 bacteria per cubic centimeter). Adopted by the Boston Board of Health in 1905, the rule was initially "the subject of scoffing", but ultimately served as a model for cities across the United States (Rosenau 1908, p. 434).

Between 1900 and 1940, all but one of the 25 cities in our sample adopted a bacteriological standard for milk (Table 4).[15] Although such standards were not always enforced with vigor

[12] Cutler and Miller (2005) control for sewage treatment plants and the chlorination of sewage but only three out of 13 cities in their sample period constructed sewage treatment plants (Baltimore in 1911, Cleveland in 1922 and Milwaukee in 1925) and only one city chlorinated its sewage (Cleveland in 1922).

[13] Baltimore was the last major U.S. city to construct a sewer system. The first house was connected to the Baltimore system in 1911 but the project was not completed until 1915 (Hoagland 2018).

[14] Olmstead and Rhode (2004a) attribute the decline in bovine tuberculosis to a U.S. federal program that began in 1917 and was designed to eradicate the disease. See Olmstead and Rhode (2004b) for an excellent description of efforts to control bovine TB that preceded the federal program. Using data from 40 U.S. cities, Komisarow (2017) explores the effect of dairy farm inspections covering a "broad range of areas and practices, including the monitoring of animal health, farmworkers' sanitation practices, and farm worker health and hygiene" (p. 129) on mortality. She finds that the introduction of dairy farm inspections reduced mortality from diarrhea and enteritis among one-year-olds by 14 percent, but finds no effect among infants nor among children ages 2-4. In a robustness check, Alsan and Goldin (2015, p. 47) include the proportion of dairies in the county that did not contain "objectionable features" as a control.

[15] Komisarow (2017, p. 131) asserts that only 5 major U.S. cities adopted a bacteriological standard for milk after 1901 (Kansas City, Missouri in 1902, Cleveland in 1906, Milwaukee in 1906, Omaha in 1906 and St. Paul in 1907). This

(Olmstead and Rhode 2004a), many cities went to great lengths to ensure that they were adhered to by dairy farmers and milk vendors. For instance, Milwaukee employed 6 full-time inspectors to ensure that its milk supply was clean (Milwaukee Common Council 1918); Baltimore hired 6 new dairy-farm inspectors, two bacteriologists and two lab assistants shortly after its 1913 milk ordinance was passed (Carey 1913).

Ordinances requiring milk meet a bacteriological standard were (and are) sometimes referred to by historians and public health experts as "pasteurization ordinances" (Harding 1917, p. 57; Troesken 2015, pp. 33-34; Swinford 2016, p. 254; Komisarow 2017, p. 131) because they were difficult to meet without resorting to pasteurization and, in fact, had the effect of encouraging all but a handful of producers to pasteurize their milk (Meckel 1990, pp. 88-89).[16] Suggestive evidence of their effectiveness comes from the fact that diarrheal deaths in the summer, when bacteria counts in milk were highest, declined markedly between 1910 and 1930 (Cheney 1984; Condran 1988; Anderson et al. 2019b). Nonetheless, this was a period during which "many municipalities were also making significant strides in improving their water supply, sewerage, and refuse removal systems" (Meckel 1990, p. 89), and disentangling the effects of these various public-health interventions requires a more careful, comprehensive empirical approach than heretofore undertaken.

TB testing of dairy cows. Approximately 10 percent of dairy cows in the United States were infected with bovine TB in 1917, the year in which federal efforts to control the disease were begun (Olmstead and Rhode 2004a). Bovine TB can be transmitted to humans through the consumption of raw milk and represented a particularly serious threat to the health of children and infants because they had lower resistance to the disease (Olmstead and Rhode 2004a). Minneapolis

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assertion, which is incorrect, appears to be based on a misinterpretation of the "Collection of Milk" dates reported in Table 114 of Parker (1917, p. 371).

[16] By the mid-1920s, the majority of American cities with a population greater than 100,000 reported that more than 90 percent of their milk supply was pasteurized (Ayers 1932). Many ordinances explicitly exempted pasteurized milk from having to meet the bacteriological standard or allowed higher levels of bacteria in raw milk that was to be pasteurized before being sold. Cities that required milk sold within their limits to meet a bacteriological standard but exempted pasteurized milk include: Chicago, Cincinnati, Indianapolis, Milwaukee, Minneapolis, New York, Philadelphia, Providence and St Louis. During the period 1900-1940, only three cities in our sample (Detroit, Chicago and San Francisco), required that all milk sold within their limits be pasteurized. Detroit passed its pasteurization ordinance in 1915 without first requiring that milk meet a bacteriological standard and that it come from tuberculin-tested cows (Kiefer 1911; Clement and Warber 1918). One year later, the Chicago commissioner of health, worried about an outbreak of Polio, ordered that all milk sold in the city be pasteurized (Czaplicki 2007). This emergency measure, which was never rescinded, was viewed as necessary despite an ordinance requiring that unpasteurized milk sold within city limits meet strict a strict bacteriological standard and that it come from tuberculin-tested cows. The San Francisco pasteurization ordinance came into effect in 1939 (Skelly 1944). Boston, New York and Pittsburg banned the sale of raw milk between 1940 and 1946.

was the first city in our sample to require that milk sold within its limits come from tuberculin-tested cows but the ordinance was not popular among dairy farmers and, at least initially, was not well enforced (Keyes 1901; Baker 1910). Despite often bitter opposition from dairy farmers (Olmstead and Rhode 2007), 22 of the 25 cities in our sample required that milk come from tuberculin-tested cows by 1940.[17]

3. DATA AND EMPIRICAL FRAMEWORK

Our focus is on major U.S. cities, defined as having a population greater than 150,000 in 1900. This criterion is met by 24 cities.[18] In addition to these 24 cities, we included data from Memphis, Tennessee in our analyses. Although its population was less than 150,000 in 1900, Memphis was among the 13 cities examined by Cutler and Miller (2005). All of our principal results, discussed below, are robust to excluding Memphis. Figure 1 maps the 25 cities in our sample.

Municipal-level mortality data come from *Mortality Statistics* and *Vital Statistics of the United States*, both of which were published annually by the U.S. Census Bureau.[19] From 1900 to 1940, annual mortality rates in major American cities fell from 1,840 to 1,164 per 100,000 population, or 37 percent (Figure 2). The reduction in the infant mortality rate was even more dramatic, falling from 383 to 80 per 100,000 population, or 79 percent (Figure 2).

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[17] For instance, due to opposition, the city of Cleveland did not initially require that dairy farmers comply with a tuberculin-testing ordinance that was passed in 1906 (Lane and Weld 1908). A Chicago tuberculin-testing ordinance, passed in 1908, faced so much resistance from dairy farmers that the Illinois legislature, under pressure from rural constituents, passed a law prohibiting any city or town in the state from requiring that dairy cattle be tested for TB (Wolf 2001, pp. 59-63; Czaplicki 2007). Many ordinances explicitly exempted pasteurized milk from having to come from tuberculin-tested cows. Cities that required milk sold within their limits to come from tuberculin-tested cows but exempted pasteurized milk include: Baltimore, Boston, Chicago, Cleveland, Indianapolis, Minneapolis, New York and Rochester. Appendix Figure 2 illustrates the roll-out of the milk-related interventions during the period 1900-1940 for our sample of cities.

[18] It proved to be extremely difficult to find intervention dates for cities with a population of less than 150,000. We were often unable to determine whether chlorine was ever added to the water supply during the period 1900-1940, whether a bacteriological standard was set for milk, or whether dairy cows were tested for TB.

[19] Specifically, mortality counts come from *Mortality Statistics* for the period 1900-1936 (U.S. Bureau of the Census 1906-1938) and from *Vital Statistics of the United States* for the period 1937-1940 (U.S. Bureau of the Census 1939-1941, 1943). Cause of death was determined by the *International List of Causes of Death* (Moriyama et al. 2011). When two or more causes of death were reported on the death certificate, a set of rules proposed by Jacques Bertillon, a French statistician and demographer, were used to determine the primary underlying cause of death. For instance, according to Bertillon, if one of the diseases was an "immediate and frequent complication of the other, the death should be classified under the heading of the primary disease" (Moriyama et al. 2011, p. 30). While Bertillon generally gave the highest priority to acts of violence, he also emphasized the importance of infectious diseases. Periodic revisions to the cause-of-death definitions are accounted for by the year fixed effects. Mortality rates are calculated using population data from the decennial censuses (and linearly imputed for intercensal years).

Typhoid deaths have been used as a proxy for water quality and to track diarrheal deaths (Cutler and Miller 2005; Ferrie and Troesken 2008; Clay et al. 2014), but it is worth noting that they represented only a small proportion of total deaths and never amounted to more than half of mortality from diarrhea/enteritis.[20] In 1900, there were 39 typhoid deaths per 100,000 population and 130 diarrhea/enteritis deaths per 100,000 population (Figure 3 and Appendix Figure 5); by the 1920s, the typhoid mortality rate was approaching zero (Figure 3) and the diarrhea/enteritis mortality rate was not far behind (Appendix Figure 5). Mortality from non-pulmonary TB also plunged during the period under study (Appendix Figure 6). In fact, by the mid-1930s non-pulmonary TB mortality among children under the age of 2 had essentially been eradicated and non-pulmonary TB morality in other age groups was approaching zero.[21]

We begin our exploration of which municipal-level public health efforts, if any, were responsible for the trends discussed above by estimating the following baseline regression:

$$(1) \qquad \ln(\textit{Mortality Rate}_{ct}) = \beta_0 + \boldsymbol{Z}_{ct}\boldsymbol{\beta_1} + \boldsymbol{X}_{ct}\boldsymbol{\beta_2} + v_c + w_t + \Theta_c \cdot t + \varepsilon_{ct},$$

where c indexes cities and t indexes years. Our primary interest is in the variables that compose the vector $\boldsymbol{Z}_{ct}$, which were constructed using information available from a wide variety of primary and secondary sources. Specifically, the vector $\boldsymbol{Z}_{ct}$ includes separate indicators for whether city c filtered its water supply in year t, whether it treated its water with chlorine and whether it had completed a clean water project.[22]

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[20] See Appendix Figures 3 and 4 for more information. Cutler and Miller (2005) assume a three-to-one ratio of diarrhea/enteritis deaths to typhoid deaths. Based on this assumption, they conclude that "reductions in all waterborne diseases account for about 8% of the reduction in total mortality…." (Cutler and Miller 2005, p. 14). In our data, the ratio of diarrhea/enteritis to typhoid deaths ranges from 3.3 (in 1900) to 15.6 (in 1920). During the period under study, diarrhea and enteritis deaths at the municipal level were reported in one category ("diarrhea and enteritis") by *Mortality Statistics* and *Vital Statistics of the United States*. Total mortality from diarrhea/enteritis is not available for the years 1939 or 1940. Enteritis is inflammation of the small intestine but can also include inflammation of the large intestine and stomach (Pietrangelo 2016).

[21] Our data do not distinguish between bovine TB mortality (caused by *M. bovis*), which was often transmitted through milk, and non-bovine TB mortality (caused by *M. tuberculosis*), which was much more common and typically spread through airborne particles (Anderson et al. 2019a). Price (1939) estimates that *M. bovis* accounted for 12 percent of total TB infections in the United States and there is evidence that approximately half of adult bovine TB sufferers exhibited pulmonary infections (Olmstead and Rhode 2004a). By contrast, only 10 percent of bovine TB infections were pulmonary among young children (Olmstead and Rhode 2004a).

[22] Tables 1 and 2 detail the water purification efforts among cities in our sample. The references used to construct Tables 1 and 2 are given in Appendix B.

The vector of controls, $\boldsymbol{X}_{ct}$, is composed of city characteristics taken from the 1900-1940 decennial censuses (and linearly interpolated for intercensal years). Specifically, it includes the percent of the population that was female, nonwhite, foreign born, under 15 years of age, 15-44 years of age and 45 years of age or older. The terms ν_c and ω_t represent city and year fixed effects, respectively. The city fixed effects control for city-level determinants of mortality that were constant over time, and the year fixed effects control for common shocks. In addition to these fixed effects, we include city-specific linear time trends ($\Theta_c \cdot t$) to account for the possibility that mortality rates evolved differently in cities that adopted certain technologies versus those that did not.

After estimating the baseline regression described above, we augment the vector $\boldsymbol{Z}_{ct}$ with an indicator for whether city c treated or diverted its sewage in year t. Finally, our fully specified model includes an indicator for whether city c required that milk sold within its limits meet a bacteriological standard and an indicator for whether it required that milk come from tuberculin-tested cows.[23] Descriptive statistics and variable definitions are reported in Table 5.

4. BASELINE RESULTS

We present estimates from the baseline model, which focuses on the relationship between total mortality and water purification efforts, in the first column of Table 6. Standard errors, corrected for clustering at the city level (Bertrand et al. 2004), are reported in parentheses; p-values calculated from the wild bootstrap procedure method suggested by Cameron et al. (2008) and Cameron and Miller (2015) are reported in curly brackets.

While the estimated coefficient of the filtration indicator is negative, it is small and not statistically significant at conventional levels. Taken at face value, it would suggest that filtering the municipal water supply reduced the total mortality rate by 2 log points, or 2.0 percent ($e^{-.02}$ -1 = -.020). By contrast, Cutler and Miller (2005), whose empirical strategy and data are closest to ours, find that filtration is associated with a 15 percent reduction in total mortality.[24] The estimated

[23] Detroit passed its pasteurization ordinance in 1915 without first requiring that milk meet a bacteriological standard and that it come from tuberculin-tested cows (Kiefer 1911; Clement and Warber 1918). The bacteriological standard and tuberculin-testing indicators are set equal to .67 for Detroit in 1915 (the ordinance same into effect on May 1) and set equal to 1 in subsequent years. When information on the exact date of implementation was available, the intervention indicators were set equal to the proportion of the year during which they were in effect. We experimented with including a mandatory pasteurization ordinance indicator on the right-hand side of our regressions. The results were qualitatively similar to those reported and discussed below.

[24] Cutler and Miller (2005) report that water filtration is associated with a reduction in total mortality of 16 log points ($e^{-.16} - 1 = -.148$). These authors estimate a regression model similar to equation (1) using data from 13 major American

coefficients of the chlorination and clean-water-project indicators are also small and statistically insignificant.

The remaining columns of Table 6 report estimates from regression models in which the sewage treatment/diversion and milk-related indicators are included on the right-hand side. The estimated effect of treating/diverting sewage is small, positive and never statistically significant at conventional levels. Likewise, the coefficients of the milk-related intervention indicators are small, consistently positive, but imprecisely estimated.

We turn our attention to infant mortality in Table 7. Across all specifications, there is strong evidence of a negative relationship between water filtration and infant mortality. For instance, filtration is association with a 12 percent reduction in the infant mortality rate if we do not include the sewage- and milk-related intervention indicators; with these indicators on the right-hand side of the regression model, it is associated with an 11 percent reduction. By contrast, Cutler and Miller (2005) find that filtration was associated with a 35 percent reduction in the infant mortality rate.[25]

To gauge the extent to which the overall infant mortality decline can be explained by filtration, we calculated a predicted infant mortality rate for every year *t* (and its 90 percent confidence interval) under the assumption that no filtration plants were constructed during the period 1900-1940. The results from this exercise are shown in Figure 4. Predicted infant mortality rates are from a regression model that controls for the variables listed in Table 5, city fixed effects, year fixed effects and city-specific linear trends. For the sake of comparison, Figure 4 also plots actual infant mortality rates. The actual and predicted mortality rates are not far apart throughout the period under study, suggesting that filtration on its own cannot explain the overall decline in infant mortality.[26]

Aside from filtration, there is little evidence that any other public health intervention contributed to the decline in infant mortality between 1900 and 1940. In fact, the estimated effects

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cities for the period 1900-1936. In Section 6, we directly compare our filtration estimates to those of Cutler and Miller (2005) and document why they are so different, at least with regard to infant and total mortality.

[25] Cutler and Miller (2005) report that water filtration is associated with a reduction in the infant mortality rate of 43 log points ($e^{-.43} - 1 = -.349$).

[26] For instance, the actual infant mortality rate among the 25 cities in our sample was 80 per 100,000 population in 1940. In the absence of filtration plants, we predict that it would have been 97 per 100,000 population. Even using the upper bound of the 90 percent confidence interval, we predict that the infant mortality rate would have fallen by 71 percent (from 383 to 110 per 100,000 population) had no filtration plants been built by the 25 cities in our sample during the period 1900-1940.

of treating/diverting sewage, setting a bacteriological standard for milk and requiring dairy cows to be tested for TB are consistently positive (but statistically insignificant), and chlorination is associated with a statistically significant 9-10 percent increase in the infant mortality rate.[27] Finally, the estimated effects of completing a clean water project, although negative, are not statistically significant at conventional levels.[28]

5. ROBUSTNESS CHECKS AND EXTENSIONS

In Table 8, we explore the robustness of the estimated relationship between filtration and infant mortality. We begin by controlling for the manufacturing wage in city *c* and year *t*, which can be thought of as measuring the purchasing power of urban workers. The estimated coefficient of the filtration indicator retains its magnitude and significance, suggesting that the effect of filtration cannot be explained by changes in the purchasing power of urban workers.[29]

Additional robustness checks include controlling for region-by-year fixed effects, not weighting by population, dropping New York City (the most populous city in the sample), dropping the years 1917-1920 to avoid potential confounding from the effects of the 1918 influenza pandemic and specifying the dependent variable in levels (as opposed to taking its log). The results of these robustness checks provide evidence that the estimated effect of water filtration on infant mortality is not an artifact of specification or sample choice. The positive (and admittedly counterintuitive) association between chlorination and infant mortality, however, appears to be quite sensitive to small changes in specification and sample composition. For instance, it shrinks and loses

[27] Cutler and Miller (2005) find a negative, but insignificant, relationship between chlorination and infant mortality, although the coefficient of the interaction between chlorination and filtration is positive. These authors ascribe this pattern of results to the fact that only a handful of cities in their sample adopted chlorination technology before building a filtration plant. We, however, observe 9 cities that added chlorine to their water supply before the construction of a filtration plant (Table 1).

[28] Interacting the filtration indicator with *Chlorination, Clean Water Project* or *Sewage Treatment/Diversion* produced coefficient estimates that were negative in sign but not statistically significant. Likewise, interacting the chlorination indicator with *Clean Water Project* or *Sewage Treatment/Diversion* produced negative but insignificant coefficient estimates.

[29] Data on the manufacturing wage come from the *Biennial Census of Manufactures*. The manufacturing wage is linearly imputed for missing years and deflated using price indices in Rees and Jacobs (1961) and the Bureau of Labor Statistics (1973). Rapid industrialization after 1900 led to substantial increases in the purchasing power of workers as measured by the manufacturing wage (Bry 1960; Rees and Jacobs 1961). With the stock market crash in October of 1929 and the onset of the Great Depression, the ranks of the unemployed swelled but real wages continued to increase (Margo 1993). Evidence that the typical American diet underwent profound changes during the period under study is provided by Bente and Gerrior (2002). Because the *Census of Manufactures* was discontinued after 1937, the column (1) regression is based on the years 1900-1937. If we do not control for the manufacturing wage and restrict our attention to the period 1900-1937, the coefficient of the filtration indicator is equal to -.101 and is statistically significant at the 5 percent level.

significance when we include region-by-year fixed effects, do not weight by population, or run the regression in levels as opposed to using the semi-log specification.

Next, we estimate a modified version of equation (1), in which the filtration indicator is decomposed into a series of mutually exclusive lags. The results, which are reported in Table 9, suggest that the effect of constructing a water filtration plant gradually became stronger over time. Specifically, filtration is associated with a (statistically insignificant) 6-7 percent decrease in the infant mortality in the year of implementation (*Year 0*); two years after implementation, filtration is associated with a 9-10 percent decrease; and, after 5 or more years, filtration is associated with a 16 percent decrease in the infant mortality rate.[30] Replacing the filtration indicator with a series of its leads and lags produces a similar pattern of results (Figure 5). Again, the effect of filtration increases after *Year 0* and there is no evidence that infant mortality rates began trending downwards prior to the adoption of filtration, suggesting the parallel trends assumption is satisfied.[31]

5.1. Estimates by cause of death

Municipal water purification efforts were often undertaken with the explicit goal of reducing (or even eradicating) typhoid infections (Foss-Mollan 2001; Melosi 2008) and, more often than not, public health officials could point to fewer typhoid deaths after these efforts came to fruition (Eddy 1913; Elms 1914; Jennings 1918; Morse and Hall 1919).[32] Nonetheless, it was widely recognized that typhoid mortality could be used as a gauge of overall water quality and that mortality from other water-borne diseases should, at least in theory, decline with the adoption of filtration and/or chlorination technology (Sedgwick and MacNutt. 1910; Hill 1911; Jordan 1916).

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[30] Even after a filtration plant came online, it could be some time before filtered water was delivered throughout the city. For instance, during its first 6 months of operation, the Cleveland filtration plant mixed its output with "raw" (i.e., unfiltered) water before delivery (Unknown Author 1918). The effort to provide filtered water to every neighborhood in Philadelphia took more than three years (McCarthy 1987).

[31] In Appendix Figure 7, we show a similar event study for filtration and total mortality. Consistent with the parallel trends assumption, there is little evidence of an association between filtration and total mortality prior to *Year 0*. Event studies for chlorination, clean water projects, sewage treatment/diversion, requiring that milk meet a bacteriological standard, and requiring that milk come from TB-tested cows are shown in Appendix Figures 8 and 9 (total mortality) and Appendix Figures 10 and 11 (infant mortality). Again, there is little evidence that mortality rates began trending downwards before implementation of these various interventions.

[32] Interestingly, there was much consternation and complaint when typhoid mortality did not fall immediately after the Washington D.C. filtration plant was put into operation (Horton 1906; Woodword 1907) and blame was placed on infected dairy products and other sources (Horton 1906; Sedgwick 1906).

In the first column of Table 10, we report estimates from a modified version of equation (1) where the dependent variable is the typhoid mortality rate per 100,000 population. Because there were zero typhoid deaths for 20 city-year combinations, we used the quartic root of the typhoid mortality rate instead of taking its natural log.[33] The marginal effects of the various interventions on the typhoid mortality rate are reported in square brackets beneath the wild cluster bootstrapped p-values. We find that the adoption of water filtration technology substantially reduced typhoid mortality. Specifically, filtration is associated with 4.6 fewer deaths from typhoid per 100,000 population, or a 36 percent reduction relative to the mean.[34] Decomposing the filtration indicator into a series of mutually exclusive lags produces evidence that the effect of filtration on typhoid mortality gradually became stronger over time (Appendix Table 2). Replacing the filtration indicator with a series of its leads and lags produces no evidence that typhoid mortality rates began trending downwards prior to the adoption of filtration (Figure 6). There is, however, little evidence that the other interventions under study were related to typhoid mortality.

In the remaining columns of Table 10, we turn our attention to diarrhea/enteritis and non-pulmonary TB mortality.[35] Although previous researchers have investigated the effects of filtration and chlorination on typhoid mortality (Troesken 2004; Cutler and Miller 2005; Beach et al. 2016),

[33] This method of dealing with zeros has been used by Thomas et al. (2006), Tarozzi et al. (2014), Ashraf et al. (2015) and Anderson et al. (2016), among others. Taking the natural log of the rate or taking the natural log of the rate plus 1 produced similar results.

[34] Cutler and Miller (2005) find that filtration is associated with a reduction in the typhoid mortality rate of 46 log points, or 37 percent ($e^{-.46} - 1 = -.369$). To gauge whether the typhoid mortality decline documented in Figure 3 can be explained by filtration, we calculated a predicted typhoid mortality rate for every year *t* (and its 90 percent confidence interval) under the assumption that no water filtration plants were built (Appendix Figure 12). Predicted typhoid mortality rates are from a regression model that controls for the variables listed in Table 5, city fixed effects, year fixed effects and city-specific linear trends. The actual typhoid mortality rate among the 25 cities in our sample was 0.44 per 100,000 population in 1940. In the absence of filtration plants, we predict that it would have been 8.3 per 100,000 population. Using the upper bound of the 90 percent confidence interval, we predict that the typhoid mortality rate would have fallen by 59 percent (from 39 to 16 per 100,000 population) had no filtration plants been built during the period 1900-1940. The estimated relationship between filtration and typhoid mortality is generally robust to specification and sample choice (Appendix Table 1). For instance, controlling for the manufacturing wage, filtration is associated with 4.9 fewer typhoid deaths per 100,000 population (or a 36 percent reduction relative to the mean).

[35] Specifically, we report estimates of equation (1) using the natural log of total diarrhea/enteritis deaths per 100,000 population as the dependent variable and the natural log of diarrhea/enteritis deaths among children under the age of two per 100,000 population. We also report estimates of equation (1) using the natural log of total non-pulmonary TB deaths per 100,000 population as the dependent variable and the quartic root of non-pulmonary TB deaths among children under the age of two per 100,000 population. Although half of adult bovine TB sufferers exhibited pulmonary infections, only 10 percent of bovine TB infections were pulmonary among young children (Olmstead and Rhode 2004a). Because there were zero non-pulmonary TB deaths among children under the age of two for 12 city-year combinations, we used the quartic root function instead of the natural log. Marginal effects are reported in square brackets below the wild cluster bootstrapped p-values.

this is the first study that uses historical data to examine the effects of these interventions on diarrheal mortality.[36] It is also the first study to test whether turn-of-the-20th century efforts to purify the milk supply at the municipal level led to reductions in mortality from non-pulmonary TB mortality.

As noted above, diarrheal deaths fell to near-zero levels during the period under study, at least in major American cities (Appendix Figure 5). The results reported in Table 10 provide evidence that filtration contributed, albeit modestly, to this phenomenon. Specifically, the building of a water filtration plant is associated with a statistically significant 14 percent reduction in the diarrhea/enteritis mortality rate.[37] By contrast, the estimated effects of the milk-related interventions are consistently positive and imprecise. In fact, aside from filtration, there is little evidence that any of the public health interventions under study contributed to the falling rates of diarrhea/enteritis mortality.[38] Similarly, the public health interventions under study seem to have been essentially unrelated to the non-pulmonary TB mortality rate.[39] Even municipal ordinances requiring that milk meet strict bacteriological standards and that dairy cows be tested for TB seem to have been

[36] Cutler and Miller (2005) have mortality data on diarrhea/enteritis mortality for one year, 1900. Based on these data, they assume a three-to-one ratio of diarrhea/enteritis deaths to typhoid deaths. Using data on 60 municipalities in Massachusetts for the period 1880-1920, Alsan and Goldin (2019) find that the interaction of sewerage and clean water is associated with a reduction in gastrointestinal mortality among children. Several studies in the economics literature use more recent data to examine the effect of water purification efforts on diarrheal mortality among children in the developing world. For instance, Bhalotra et al. (2017) estimate that a Mexican program that increased the share of the population receiving chlorinated water by 30 percentage points led to a substantial reduction in diarrheal mortality among children.

[37] We assess the robustness of the relationship between filtration and diarrhea/enteritis mortality in Appendix Table 3 and we explore lagged effects in Appendix Table 4. In Appendix Figure 13, we gauge the extent to which the observed diarrhea/enteritis mortality decline during the period 1900-1940 can be explained by water filtration. Specifically, we plot actual diarrhea/enteritis mortality rates and the predicted diarrhea/enteritis mortality rate for every year *t* (and its 90 percent confidence interval) under the assumption that no water filtration plants were built. The actual and predicted rates are never far apart throughout the period under study, suggesting that filtration did not contribute substantially to the decline in diarrhea/enteritis mortality. The estimated effect of filtration on the diarrhea/enteritis mortality rate loses significance when we drop the years 1917-1920 or specify the dependent variable in levels. Finally, there is no evidence that diarrhea/enteritis mortality rates began trending downwards prior to the adoption of filtration (Appendix Figure 14).

[38] The completion of a clean water project is associated with a 15 percent reduction in the total diarrhea/enteritis mortality rate. It is also associated with a 15 percent reduction in the diarrhea/enteritis mortality rate among children under the age of two. Neither of these estimates, however, are statistically significant at conventional levels.

[39] Non-pulmonary TB mortality counts by age are unavailable at the municipal level for the years 1937-1940.

ineffective at reducing the non-pulmonary TB mortality rate, a result that perhaps can be attributed to lack of enforcement. [40]

6. COMPARING OUR ESTIMATES TO THOSE OF CUTLER AND MILLER

Using data from 13 major American cities for the period 1900-1936, Cutler and Miller (2005), hereafter C&M, investigate the effects of water filtration and chlorination on mortality. C&M's empirical strategy is similar to ours and their mortality data also come from *Mortality Statistics*. Their cities, listed in Appendix Table 5, were similar to ours in terms of population and demographic characteristics (Appendix Table 6).

C&M find that water filtration is associated with a reduction in total mortality of 16 log points (= -15 percent) and a reduction in infant mortality of 43 log points (= -35 percent). C&M's estimate of the effect of filtration on typhoid mortality is almost exactly equal to the estimate we report in the first column of Table 10. However, C&M's infant and total mortality estimates are considerably larger. In this section, we explore why.

We begin by reproducing C&M's estimates of the effects of filtration and chlorination on total mortality using their specification and original data. These estimates are reported in column (1) of Table 11.[41] While C&M's specification is similar to ours, they control for 5 lags of the total mortality rate.[42] Because the inaugural issue of *Mortality Statistics* was published in 1900, including these lags effectively restricts their analysis to the period 1905-1936. In Appendix Table 5, we compare their specification and sample to ours.[43]

In column (2) of Table 11, we correct their standard errors for clustering at the municipal level (Bertrand et al. 2004). With this correction, the filtration coefficient remains significant at the 5

[40] In general, enforcement of milk ordinances was strictest in the largest cities. Fuchs and Frank (1939) surveyed municipal health departments across the country regarding their milk purification efforts in 1936. Cities with a population greater than 500,000 employed an average of 27.6 full-time milk inspectors; cities with a population between 100,000 and 500,000 employed an average of 3.7 full-time milk inspectors. When we restricted our attention to cities with a population 200,000 or greater in 1900, the estimated effects of these ordinances on the non-pulmonary TB mortality were still small and insignificant.

[41] We are grateful to Cutler and Miller for graciously providing us with their data and do-file. These estimates correspond to those reported by C&M in the second column of their Table 5.

[42] Baltagi (2013) shows that including lags of the outcome as controls is problematic with fixed effects, which necessitates a generalized methods of moments approach.

[43] In Appendix Table 7, we list and describe the controls used by C&M.

percent level, but the coefficient of the interaction between chlorination and filtration is no longer statistically significant. C&M interpret the positive and significant coefficient of the interaction between chlorination and filtration as evidence that these were "substitute technologies" (p. 11).

In the third column of Table 11, we correct a minor transcription error. Memphis did not provide mortality data to the U.S. Census Bureau in 1916, so C&M assign Memphis a total mortality rate of zero for this year. Because they use a semi-log specification, the 1916 Memphis observation is dropped from their regression, but the 5 lags of the 1916 Memphis total mortality rate are incorrectly coded as zeros. Dropping these 5 observations with missing lagged mortality rates reduces the estimated effect of filtration from -16 log points to -13 log points.

For the years 1900-1917, C&M rely on total mortality rates published in *Mortality Statistics*.[44] By contrast, for the post-1917 period, C&M calculate total mortality rates using mortality counts from *Mortality Statistics* and U.S. Bureau of the Census population estimates (linearly imputing population for intercensal years). In the fourth column of Table 11, we use this latter method to consistently calculate total mortality rates for the entire period under study, which reduces the estimated impact of filtration from -13 log points to -8 log points.[45]

In the fifth column of Table 11, we correct C&M's chlorination and filtration dates.[46] These corrections reduce the filtration estimate further, to -4.3 log points, which is less than a third the size of C&M's original estimate. In the last column of Table 11, we switch from C&M's specification to ours but restrict the sample to their years (i.e., 1905-1936) and cities. Filtration is associated with a

[44] For the years 1900-1909, the differences between C&M's total mortality rates and ours are trivial. For these years, C&M use revised mortality rates published in the 1909 *Mortality Statistics* volume (see Table IV in U.S. Bureau of the Census (1909)). On the other hand, for the years 1910-1917, C&M's total mortality rates and ours are often substantially different. C&M's total mortality rates for the period 1910-1917 can be found in Table III in U.S. Bureau of the Census (1910, 1911, 1913, 1914, 1915, 1916, 1917).

[45] Population estimates at the city level for 1910 can be found in Table I of U.S. Bureau of the Census (1910). Population estimates for 1920 can be found in Table IB of U.S. Bureau of the Census (1920). We also corrected two minor mortality count transcriptions errors made by C&M for Baltimore in 1929 and New Orleans in 1930.

[46] The majority of these corrections were trivial. For instance, according to C&M, Baltimore began filtering its water supply in 1914, but the Montebello Water Filtration Plant did not begin operations until September 13, 1915 (Hendrickson 2012). However, the chlorination dates provided by C&M for Memphis, Milwaukee and St. Louis are off by at least 5 years. Memphis began chlorinating its water supply in 1920, not after 1936 (Typhoid in the Large Cities 1921, 1922); Milwaukee began chlorinating its water supply in 1910, not 1915 (Jennings 1918); and St. Louis began chlorinating its water supply in 1913, not 1919 (Wall 1920). Chicago began experimenting with chlorinating its water supply in 1912, but full chlorination was not achieved until 1917 (Jennings 1923). We chose to use 1912 as the start date for Chicago chlorination but using 1917 does not appreciably change our results or those of C&M. Likewise, the process of delivering filtered water to all the neighborhoods of Philadelphia began before 1906 and was not completed until 1909 (Philadelphia 1909). We chose to use 1906 as the start date for Philadelphia filtration but using 1909 does not appreciably change our results or those of C&M.

statistically insignificant reduction in total mortality of 4.2 log points, which is still considerably larger than the estimate we report in the last column of Table 6, suggesting that the effect of filtration on total mortality in the 13 cities analyzed by C&M may have been larger than its effect in our expanded sample of 25 cities.[47]

We turn our attention to C&M's infant mortality estimates in Table 12. Again, we begin by reproducing C&M's estimates of the effects of chlorination and filtration on infant mortality using their original data.[48] In the second column of Table 12, we show that the estimated relationship between filtration and infant mortality is still significant at the one percent level after correcting their standard errors for clustering at the municipal level. In the third column, we drop the 5 Memphis observations with missing lagged total mortality, which reduces the estimated effect of filtration on infant mortality from -43 to -37 log points. In the fourth column, we correct a series of transcription errors in C&M's infant mortality counts (79 of 410 infant mortality counts were incorrectly transcribed). These errors are detailed in Appendix Table 8.[49] Correcting them reduces the estimated effect of filtration on infant mortality by almost two-thirds, from -37 to -13 log points. Correcting C&M's chlorination and filtration dates reduces the filtration estimate still further, to a statistically insignificant -3.8 log points. Finally, when we switch from C&M's specification to ours (but restrict the sample to their years and cities), filtration is associated with a reduction in infant mortality of 9.6 log points, which is similar in magnitude to our estimates reported in Table 7.[50]

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[47] The estimated effect of filtration on total mortality reported in the last column of Table 14 is very similar in magnitude to an estimate produced by Catillon, Cutler and Getzen (2018) using data for the period 1905-1936 from the same 13 cities (see the first column of their Table 2). Catillon, Cutler and Getzen (2018) used C&M's specification, but omitted the city-specific linear trends and the following three controls listed in Appendix Table 7: ln(*Poplation*), *Filtration w/in 5 Years* and *Chlorination w/in 5 Years*.

[48] These estimates correspond to those reported by C&M in the third column of their Table 5.

[49] In 9 out of the 13 cities in their sample, C&M make systematic transcription errors for the years 1910-1917. In these city-year combinations, infant mortality counts are reported with decimal values. In Appendix Table 8, we list the correct counts from *Mortality Statistics* alongside the incorrect values recorded by C&M. The differences between their recorded values and the correct counts are not large for some cities, such as Chicago and St. Louis. For other cities, however, the differences are substantial. For instance, the values recorded by C&M for Memphis and New Orleans are roughly 50 percent smaller than the correct counts from *Mortality Statistics*. In all cases, the values recorded by C&M are less than the true values. In addition to the systematic errors for the period 1910-1917, C&M make several other transcription mistakes, some of which can easily be explained. For example, for Pittsburgh in 1901, C&M appear to have incorrectly recorded the overall mortality count (6,578 deaths) instead of the infant mortality count (1,580 deaths). For Pittsburgh in 1904, they appear to have incorrectly recorded 1,771 infant deaths as "771" infant deaths.

[50] A final difference between C&M's specification and ours is that they divide infant mortality by infant population rather than overall population when calculating the infant mortality rate. If we do this, the filtration estimate reported in column (6) in Table 12 becomes smaller in magnitude and loses precision (*Filtration* estimate = -.066, with a standard error = .045).

7. CONCLUSION

The mortality transition is of obvious importance to policymakers and economic historians, but its causes are not well understood. Prominent economists such as Fogle and McKeown argued that rising incomes and better nutrition were the primary drivers of the morality transition (McKeown and Record 1962; McKeown 1976; Fogel 1997, 2004). More recently, however, economists have emphasized the role of municipal-level public health interventions. For instance, Costa (2015) argues that filtration and chlorination were the primary drivers of the reduction in infant mortality; citing Cutler and Miller (2005), Cutler et al. (2006) argue that public health interventions such filtration and chlorination were responsible for the dramatic reductions in food- and water-borne diseases at the turn of the 20th century.

In this study, we reevaluate the impact of public health interventions on the urban mortality decline in the United States at the turn of the 20th century. Specifically, using data on 25 major American cities for the years 1900-1940, we attempt to distinguish the effects of municipal-level efforts to purify water and milk supplies. Previous researchers, including Cutler and Miller (2005), Troesken (2004), Beach et al. (2016) and Knutsson (2018) focus on estimating the contributions of filtering and chlorinating drinking water to the urban mortality decline. In addition to these interventions, we examine the treatment of sewage before discharging it into lakes, rivers and streams, the construction of large-scale projects designed to deliver clean water from further afield, requirements that milk sold within city limits meet strict bacteriological standards, and requirements that milk come from tuberculin-tested cows.

Consistent with the results of turn-of-the-20th-century studies (Clark 1907; Johnson 1907; Sedgwick and MacNutt. 1910; Johnson 1913; McLaughlin 1912), as well as more recent research (Troesken 2004; Cutler and Miller 2005; Beach et al. 2016), we find that filtering the municipal water supply sharply reduced typhoid mortality. The building of a water filtration plant is also associated with an 11-12 percent reduction in infant mortality and a 14 percent reduction in the diarrhea/enteritis mortality rate. Although these latter estimates are statistically significant, they are not nearly large enough to explain the overall declines in infant and diarrheal mortality observed during the period 1900-1940. Finally, we find little evidence that the other public interventions, including chlorination, setting bacteriological standards for milk, and treating sewage were effective, although it should be noted that we do not have information on morbidity (as opposed to mortality) and all of these interventions could have led to a better quality of life.

Leaning on the results of Cuter and Miller (2005), scholars such as Cutler et al. (2006) and Costa (2015) argue that municipal public health efforts were important drivers of the mortality transition at the turn of the 20th century. Because the study by Cutler and Miller (2005) is so influential, it is important to document why our filtration estimates are different from theirs. Using their original data and specification, we find that the estimated effect of filtration on total mortality shrinks by half, from -16 log points to -8 log points, when we correct a handful of transcription errors and use U.S. Bureau of the Census population estimates to consistently calculate the total mortality rate for the entire period under study, 1900-1936. Correcting a series of transcription errors in their infant mortality counts (79 of 410 infant mortality counts were incorrectly transcribed) reduces the estimated effect of filtration on infant mortality by two-thirds, from -43 log points to -13 log points.

If public health interventions such as chlorination and filtration were not the primary drivers of the reduction in urban mortality between 1900 and 1940, what were? Two recent studies provide evidence that public health efforts to combat tuberculosis (the second-leading cause of death at the turn of the 20th century) were ineffective (Anderson et al. 2019a; Clay et al. 2019), but it is possible that better living conditions and improved nutrition contributed substantially to the mortality transition. It is also possible that advancements in medical techniques and new technologies such as refrigeration played important roles. These are, however, educated guesses. We hope that readers will embrace the implicit challenge inherent in our results and redouble their efforts to discover the true causes of the urban mortality transition.

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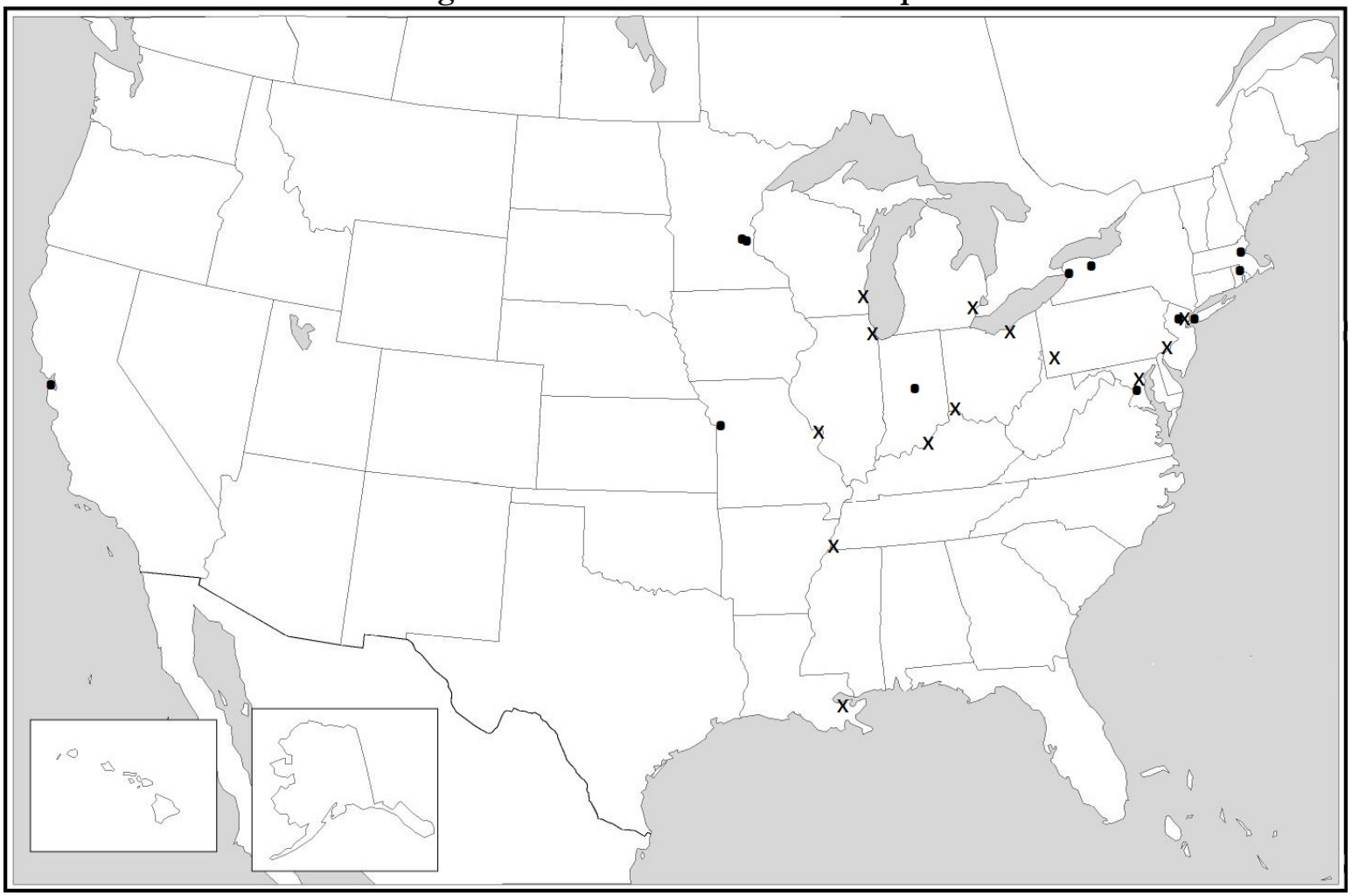
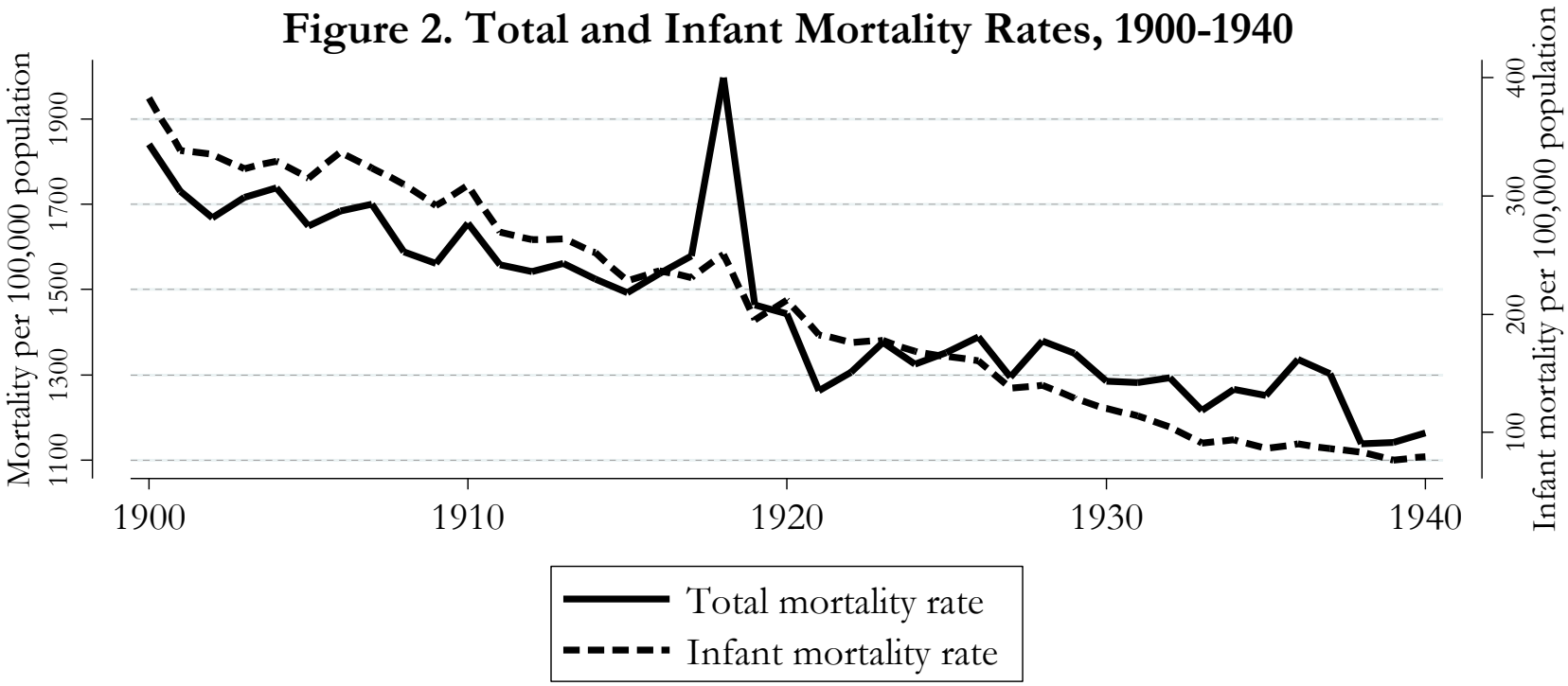
Figure 1. Locations of Cities in Sample



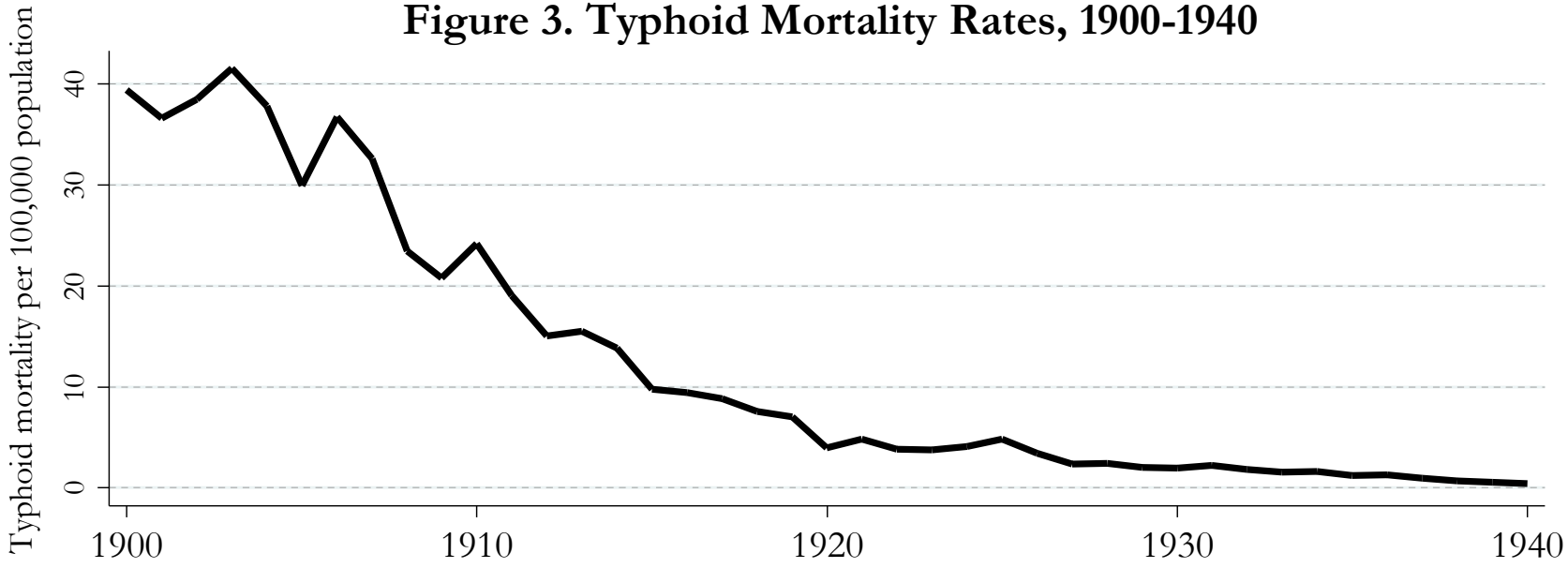
Notes: The 13 cities also examined by Cutler and Miller (2005) are denoted with an "X."

Figure 2. Total and Infant Mortality Rates, 1900-1940



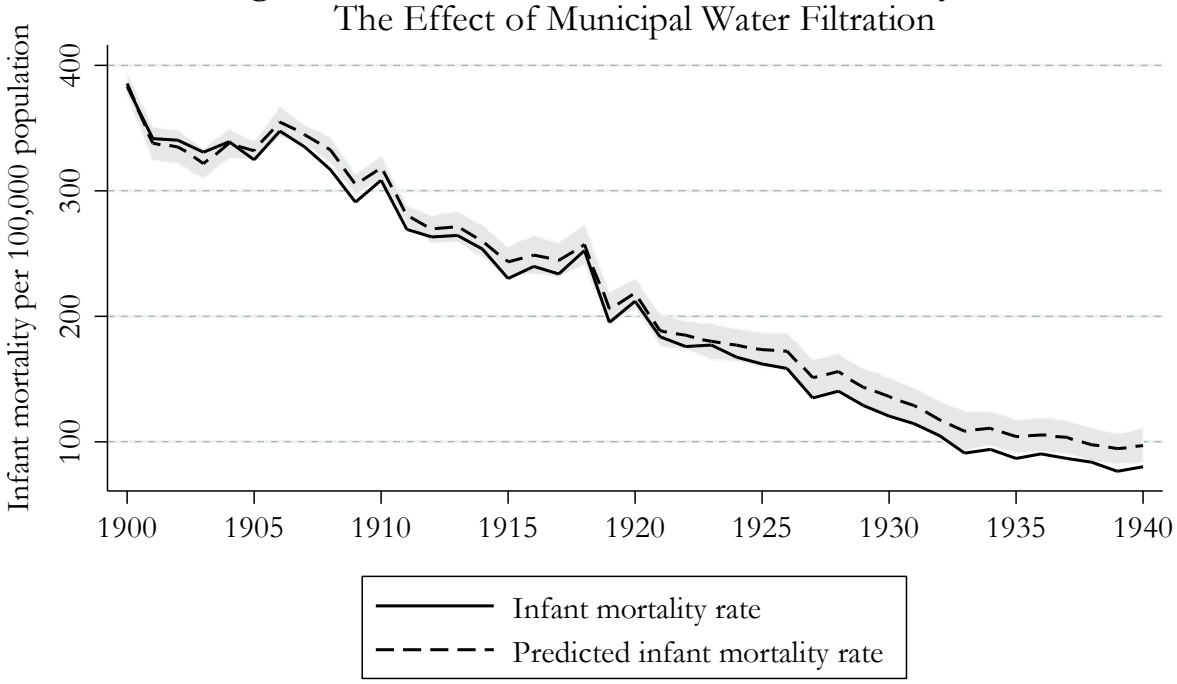
Notes: Based on annual data from *Mortality Statistics* and *Vital Statistics of the United States* for the period 1900-1940, published by the U.S. Census Bureau.

Figure 3. Typhoid Mortality Rates, 1900-1940



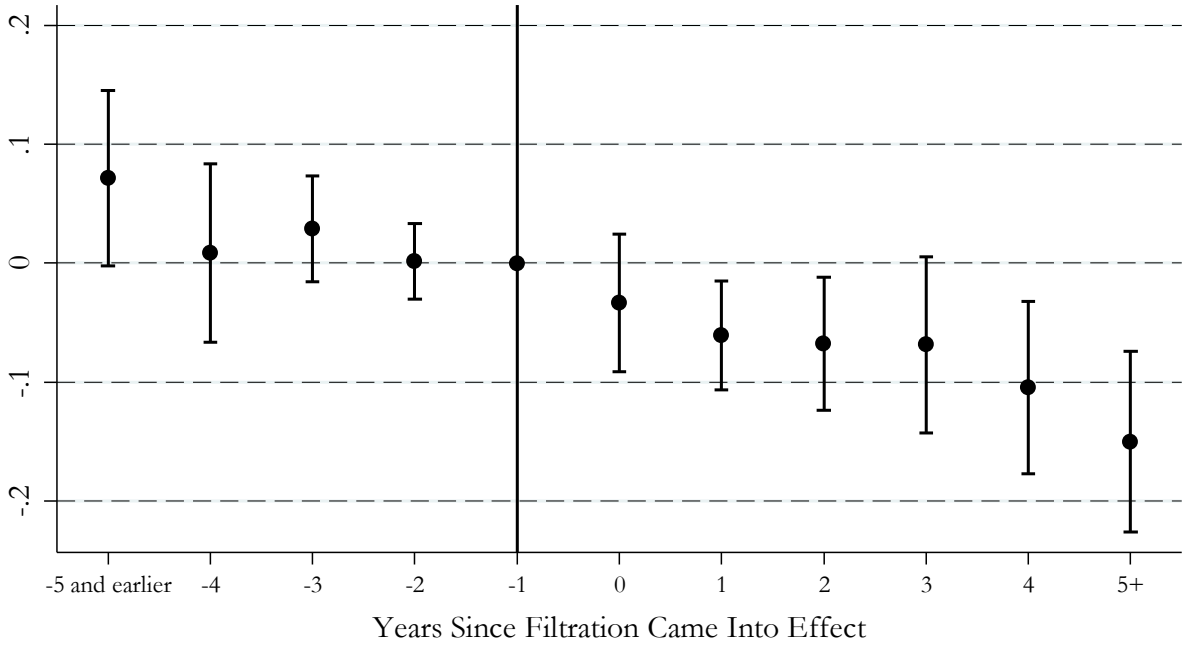
Notes: Based on annual data from *Mortality Statistics* and *Vital Statistics of the United States* for the period 1900-1940, published by the U.S. Census Bureau.

Figure 4. Actual vs. Predicted Infant Mortality Rates
The Effect of Municipal Water Filtration



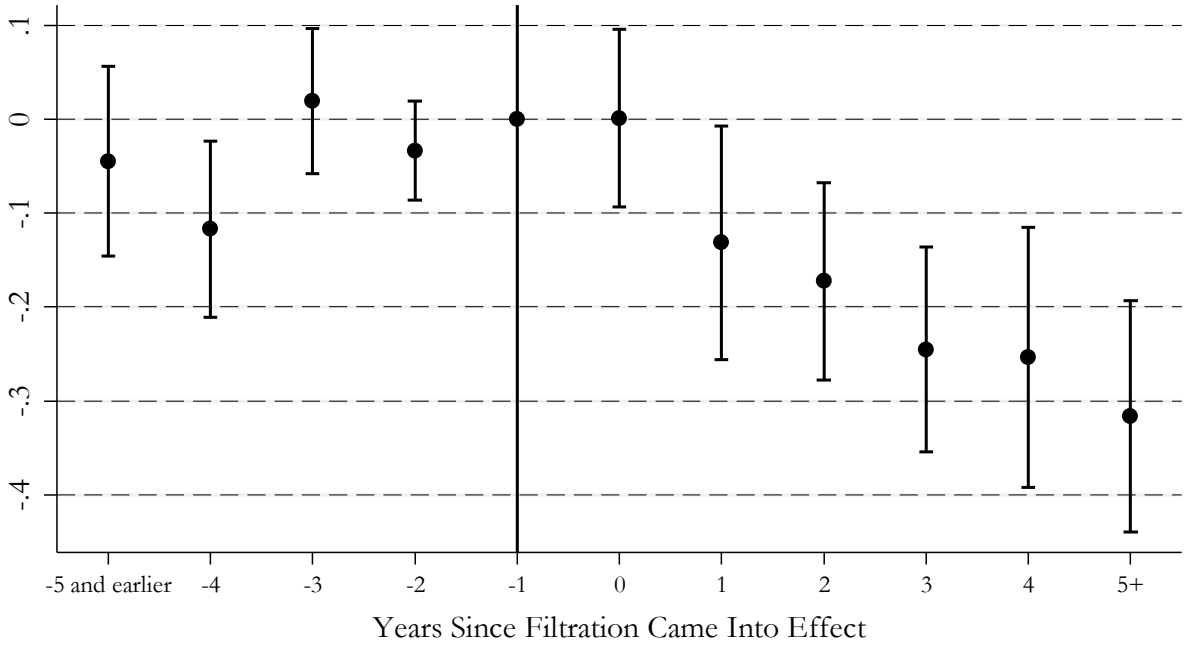
Notes: Based on annual data from *Mortality Statistics* and *Vital Statistics of the United States* for the period 1900-1940, published by the U.S. Census Bureau. Predicted infant mortality rates are calculated under the assumption that municipalities did not filter their water supply. Shaded area represents 90% confidence region around infant mortality rates.

Figure 5. Pre- and Post-Filtration Trends in Infant Mortality



Notes: OLS coefficient estimates (and their 90% confidence intervals) are reported, where the omitted category is 1 year before treatment. The dependent variable is equal to the natural log of the number of infant deaths per 100,000 population in city *c* and year *t*. Controls include the demographic characteristics and remaining public health interventions listed in Table 5, city fixed effects, year fixed effects, and city-specific linear trends. Regressions are weighted by city population. Standard errors are corrected for clustering at the city level.

Figure 6. Pre- and Post-Filtration Trends in Typhoid Mortality



Notes: OLS coefficient estimates (and their 90% confidence intervals) are reported, where the omitted category is 1 year before treatment. The dependent variable is equal to the quartic root of the number of typhoid deaths per 100,000 population in city *c* and year *t*. Controls include the demographic characteristics and remaining public health interventions listed in Table 5, city fixed effects, year fixed effects, and city-specific linear trends. Regressions are weighted by city population. Standard errors are corrected for clustering at the city level.

Table 1. Municipal Water Purification, 1900-1940

City and State	Water Filtration Plant[a]	Water Treated with Chlorine[b]
Baltimore, Maryland	1915	1911
Boston, Massachusetts	…	1928
Buffalo, New York	1926	1914
Chicago, Illinois	…	1912
Cincinnati, Ohio	1907	1918
Cleveland, Ohio	1918	1911
Detroit, Michigan	1923	1913
Indianapolis, Indiana	1904	1909
Jersey City, New Jersey	…	1908
Kansas City, Missouri	1928	1911
Louisville, Kentucky	1909	1913
Memphis, Tennessee	…	1920
Milwaukee, Wisconsin	1939	1910
Minneapolis, Minnesota	1913	1910
Newark, New Jersey	…	1921
New Orleans, Louisiana	1909	1915
New York, New York	…	1911
Philadelphia, Pennsylvania	1906	1910
Pittsburgh, Pennsylvania	1908	1910
Providence, Rhode Island	1904	1917
Rochester, New York	…	1925
San Francisco, California	…	1922
St. Louis, Missouri	1915	1913
St. Paul, Minnesota	1923	1920
Washington, D.C.	1905	1923

[a] **Philadelphia, PA**: Filtration began before 1906, but not all parts of the city received filtered water until February, 1909. **Pittsburgh, PA**: By October 1908, the water supply of peninsular Pittsburgh was being filtered. In 1909 and 1914, the Southside and the Northside, respectively, began receiving filtered water.

[b] **Chicago, IL**: Chlorination began in 1912, but full chlorination was not achieved until 1917. **Milwaukee, WI**: Water was chlorinated from June, 1910-December, 1910; February, 1912-March 1912; April, 1912 onwards. **Newark, NJ**: Chlorine was used in rare, emergency-only cases beginning in 1913; continuous use started in 1921. **Philadelphia, PA**: Water was chlorinated from December, 1910-April, 1911; December, 1911-February, 1913; November, 1913 onwards. **Pittsburgh, PA**: Water was chlorinated from January, 1910-March, 1910; November, 1910-April, 1911; August, 1911 onwards.

Table 2. Clean Water Projects, 1900-1940

City and State	Clean Water Project	Description
Boston, Massachusetts	1904	Water was conveyed by the Wachusett/Weston Aqueduct to the Weston Reservoir. Water was first delivered to metropolitan Boston on December 29, 1904.
Buffalo, New York	1913	Water intake, located on Lake Erie's Emerald Channel, was completed on May 12, 1913.
Cleveland, Ohio	1904	Cleveland built the first tunnel (the "Five Mile Crib") to draw water from Lake Erie. It went into operation on April 6, 1904.
Jersey City, New Jersey	1904	The Boonton Reservoir began delivering water to Jersey City on May 23, 1904.
Newark, New Jersey	1930	The Wanaque Reservoir began delivering water to Newark on March 20, 1930.
New York, New York	1907	The New Croton Dam was completed on January 1, 1907 and began delivering water to New York City on November 6, 1907. The Catskills Aqueduct began delivering water to the Bronx on December 27, 1915. By January 22, 1917, all other boroughs were receiving water.
Providence, Rhode Island	1926	The Scituate Reservoir began delivering water to Providence on September 30, 1926.
San Francisco, California	1934	The Hetch Hetchy Reservoir began delivering water to San Francisco on October 28, 1934.

Table 3. Sewage Treatment/Diversion, 1900-1940

City and State	Sewage Treatment Plant/Sewage Diversion	Description
Baltimore, Maryland	1911	Operation of the sewage treatment plant was begun "in the latter part of 1911" (Wagenhals et al. 1925).
Buffalo, New York	1938	The sewage treatment plant was completed in June, 1938.
Chicago, Illinois	1907	In 1907, the last sewer outfalls emptying into Lake Michigan were shut off.
Cleveland, Ohio	1922	The first sewage treatment plant was opened in 1922. By 1928, two additional plants were in operation.
Detroit, Michigan	1912	The Detroit River Interceptor was built in 1912. It intercepted sewage and discharged it below the intake for drinking water. Detroit began treating its sewage in February, 1940.
Indianapolis, Indiana	1925	The sewage treatment plant began operations in May, 1925.
Jersey City, New Jersey	1924	The sewage treatment plant was built in 1924 and upgraded in 1937.
Milwaukee, Wisconsin	1925	The sewage treatment plant began operations in June, 1925.
Minneapolis, Minnesota	1938	The sewage treatment plant began operations in June, 1938. It served both Minneapolis and St. Paul.
Newark, New Jersey	1924	The sewage treatment plant began operations in 1924.
New York, New York	1937	The Wards Island sewage treatment plant began operations in October, 1937.
Providence, Rhode Island	1901	The Providence sewage treatment plant, built in 1901, used chemical precipitation. It converted to using an activated sludge process in the mid-1930s.
Rochester, New York	1917	The sewage treatment plant began operations in March, 1917
St. Paul, Minnesota	1938	The sewage treatment plant began operations in June, 1938. It served both Minneapolis and St. Paul
Washington, D.C.	1938	The sewage treatment plant began operations in 1938.

Table 4. Municipal Milk Purification, 1900-1940

City and State	Bacteriological Standard[a]	Tuberculin Testing of Cows[b]
Baltimore, Maryland	1913	1917
Boston, Massachusetts	1905	1924
Buffalo, New York	1918	…
Chicago, Illinois	1909	1909
Cincinnati, Ohio	1914	1907
Cleveland, Ohio	1906	1906
Detroit, Michigan	1915	1915
Indianapolis, Indiana	1916	1916
Jersey City, New Jersey	1915	1915
Kansas City, Missouri	1910	1910
Louisville, Kentucky	1932	1932
Memphis, Tennessee	1917	1910
Milwaukee, Wisconsin	1908	1908
Minneapolis, Minnesota	1907	1895
Newark, New Jersey	1913	1913
New Orleans, Louisiana	1923	1923
New York, New York	1912	1914
Philadelphia, Pennsylvania	1915	1930
Pittsburgh, Pennsylvania	1910	…
Providence, Rhode Island	1915	…
Rochester, New York	1907	1922
San Francisco, California	1909	1909
St. Louis, Missouri	1923	1928
St. Paul, Minnesota	1907	1907
Washington, D.C.	…	1925

[a] **Baltimore, MD**: On October 15, 1912, Baltimore passed an ordinance setting a bacteriological standard, but the first milk inspectors did not start working until January 1, 1913. **Boston, MA**: On March 1, 1905, the Boston Board of Health (in conjunction with the State Board of Health) set a bacteriological standard for health inspectors to follow. On January 8, 1913, Boston passed an ordinance that required licensing of milk producers and set a bacteriological standard. **Buffalo, NY**: The Buffalo Health Commissioner conducted bacteriological tests of milk as early as 1907, but standards were not set by law until October 9, 1918. **Chicago, IL:** The Chicago milk ordinance that came into effect on January 1, 1909 was nullified by the Illinois legislature on June 12, 1911. A new ordinance, passed on August 14, 1912, required that non-pasteurized milk come from tuberculin-tested cows and meet a bacteriological standard. On July 22, 1916, the Chicago Commissioner of Health required that all milk be pasteurized. **Detroit, MI:** An ordinance required that all milk sold in Detroit be pasteurized as of May 1, 1915. **Philadelphia, PA**: As of October 15, 1909, dairy farmers were required to have a license. Although inspections were conducted under rules set by the Board of Health, a bacteriological standard was not enforced until July 1, 1915.

[b] **St. Louis, MO**: Ordinance for tuberculin testing of raw milk was set on March 21, 1928. Ordinance for tuberculin testing of all milk, including pasteurized milk, was set on March 21, 1930.

Table 5. Descriptive Statistics

	Mean (SD)	Description
Filtration	.428 (.493)	= 1 if city had a water filtration plant, = 0 otherwise
Chlorination	.625 (.480)	= 1 if city chemically treated water supply, = 0 otherwise
Clean Water Project	.236 (.423)	= 1 if city had completed a clean water project, = 0 otherwise
Sewage Treatment/Diversion	.248 (.430)	= 1 if city had a sewage treatment plant or diverted sewage away from drinking water supply, = 0 otherwise
Bacteriological Standard	.616 (.484)	= 1 if city set bacteriological standard for milk supply, = 0 otherwise
TB Test	.515 (.497)	= 1 if city required tuberculin testing of cows, = 0 otherwise
% Female	.503 (.016)	Percent of city population that was female
% Nonwhite	.090 (.100)	Percent of city population that was nonwhite
% Foreign	.193 (.109)	Percent of city population that was foreign born
% Under 15	.255 (.032)	Percent of city population that was under 15 years of age
% 15 to 44	.529 (.025)	Percent of city population that was 15 to 44 years of age
% 45 and Older	.216 (.038)	Percent of city population that was 45 years of age or older
N	1,024	

Notes: Unweighted means with standard deviations in parentheses. When information on the exact date of implementation was available, the water- and milk-related interventions took on a value between 0 and 1 the year in which they went into effect.

Table 6. The Effects of Water Quality, Sewage Treatment/Diversion, and Clean Milk on Total Mortality

	(1)	(2)	(3)	(4)	(5)
			Total Mortality		
Water					
Filtration	-.020	…	…	-.019	-.011
	(.016)			(.016)	(.016)
	{.236}			{.271}	{.504}
Chlorination	.015	…	…	.012	.009
	(.014)			(.013)	(.013)
	{.412}			{.443}	{.547}
Clean Water Project	-.017	…	…	-.014	-.011
	(.025)			(.024)	(.023)
	{.620}			{.687}	{.710}
Sewage					
Sewage Treatment/Diversion	…	.025	…	.022	.018
		(.025)		(.024)	(.022)
		{.596}		{.579}	{.563}
Milk					
Bacteriological Standard	…	…	.016	…	.015
			(.017)		(.018)
			{.419}		{.507}
TB Test	…	…	.019	…	.015
			(.014)		(.013)
			{.200}		{.276}
Mean of total mortality rate	1,462	1,462	1,462	1,462	1,462
N	1,024	1,024	1,024	1,024	1,024
R^2	.930	.930	.931	.931	.931

*Statistically significant at 10% level; ** at 5% level; *** at 1% level.

Notes: Based on annual data from *Mortality Statistics* for the period 1900-1940, published by the U.S. Census Bureau. Each column represents the results from a separate OLS regression. The dependent variable is equal to the natural log of the number of deaths per 100,000 population in city *c* and year *t*. Controls include the demographic characteristics listed in Table 5, city fixed effects, year fixed effects and city-specific linear trends. Regressions are weighted by city population. Standard errors, corrected for clustering at the city level, are in parentheses. P-values from wild cluster bootstrap procedure are in curly brackets and are based on 1,000 replications.

Table 7. The Effects of Water Quality, Sewage Treatment/Diversion and Clean Milk on Infant Mortality

	(1)	(2)	(3)	(4)	(5)
	Infant Mortality (*Under Age 1*)				
Water					
Filtration	-.131***	…	…	-.128***	-.111**
	(.045)			(.044)	(.041)
	{.023}			{.022}	{.028}
Chlorination	.093**	…	…	.088**	.082**
	(.044)			(.038)	(.039)
	{.095}			{.079}	{.113}
Clean Water Project	-.069	…	…	-.061	-.055
	(.066)			(.061)	(.058)
	{.425}			{.463}	{.480}
Sewage					
Sewage Treatment/Diversion	…	.067	…	.048	.040
		(.067)		(.056)	(.049)
		{.794}		{.790}	{.727}
Milk					
Bacteriological Standard	…	…	.040	…	.024
			(.036)		(.037)
			{.354}		{.631}
TB Test	…	…	.061	…	.040
			(.042)		(.031)
			{.282}		{.328}
Mean of infant mortality rate	206.4	206.4	206.4	206.4	206.4
N	1,024	1,024	1,024	1,024	1,024
R^2	.981	.979	.980	.981	.981

*Statistically significant at 10% level; ** at 5% level; *** at 1% level.

Notes: Based on annual data from *Mortality Statistics* for the period 1900-1940, published by the U.S. Census Bureau. Each column represents the results from a separate OLS regression. The dependent variable is equal to the natural log of the number of infant deaths per 100,000 population in city *c* and year *t*. Controls include the demographic characteristics listed in Table 5, city fixed effects, year fixed effects and city-specific linear trends. Regressions are weighted by city population. Standard errors, corrected for clustering at the city level, are in parentheses. P-values from wild cluster bootstrap procedure are in curly brackets and are based on 1,000 replications.

Table 8. Robustness Checks: The Effects of Water Quality, Sewage Treatment/Diversion, and Clean Milk on Infant Mortality

	(1)	(2)	(3)	(4)	(5)	(6)
	Control for wages	Control for region-by-year fixed effects	Unweighted	Drop New York City	Drop years 1917-1920	Dependent variable in levels
Water						
Filtration	-.098**	-.097**	-.133***	-.116***	-.094**	-21.6*
	(.039)	(.043)	(.037)	(.035)	(.043)	(11.0)
	{.046}	{.080}	{.004}	{.009}	{.067}	{.099}
Chlorination	.072*	.029	.050	.094**	.078*	12.5
	(.035)	(.033)	(.044)	(.039)	(.039)	(9.19)
	{.114}	{.427}	{.340}	{.100}	{.115}	{.258}
Clean Water Project	-.059	-.104**	.037	.014	-.036	-21.3*
	(.041)	(.039)	(.053)	(.087)	(.054)	(11.1)
	{.262}	{.048}	{.646}	{.922}	{.612}	{.305}
Sewage						
Sewage Treatment/Diversion	.080*	.053	.011	.073	.037	7.27
	(.044)	(.037)	(.038)	(.047)	(.049)	(11.3)
	{.337}	{.540}	{.848}	{.463}	{.745}	{.766}
Milk						
Bacteriological Standard	.025	.056	.070*	.030	.007	1.38
	(.037)	(.037)	(.036)	(.045)	(.035)	(8.55)
	{.584}	{.249}	{.104}	{.606}	{.863}	{.903}
TB Test	.038	.010	.006	.056*	.047	7.16
	(.028)	(.031)	(.039)	(.032)	(.029)	(7.40)
	{.255}	{.791}	{.923}	{.182}	{.191}	{.434}
Mean of infant mortality rate	216.4	206.4	206.4	206.0	204.6	206.4
N	949	1,024	1,024	983	924	1,024
R^2	.980	.987	.965	.975	.983	.972

*Statistically significant at 10% level; ** at 5% level; *** at 1% level.

Notes: Based on annual data from *Mortality Statistics* for the period 1900-1940, published by the U.S. Census Bureau. Each column represents the results from a separate OLS regression. In columns (1)-(5), the dependent variable is equal to the natural log of the number of infant deaths per 100,000 population in city *c a*nd year *t*. In column (6), the dependent variable is equal to the number of infant deaths per 100,000 population in city c and year *t*. Controls include the demographic characteristics listed in Table 5, city fixed effects, year fixed effects and city-specific linear trends. In columns (1)-(2) and (4)-(6), regressions are weighted by city population. Standard errors, corrected for clustering at the city level, are in parentheses. P-values from wild cluster bootstrap procedure are in curly brackets and are based on 1,000 replications.

Table 9. Infant Mortality and Lags of *Filtration*

	(1)	(2)	(3)
		Infant Mortality	
Year 0	-.063	-.067	-.070
	(.043)	(.043)	(.044)
	{.213}	{.181}	{.182}
1 Year After Filtration	-.088**	-.093**	-.096**
	(.036)	(.036)	(.037)
	{.026}	{.023}	{.025}
2 Years After Filtration	-.092**	-.098***	-.101***
	(.035)	(.035)	(.036)
	{.019}	{.016}	{.016}
3+ Years After Filtration	-.147***	…	…
	(.043)		
	{.004}		
3 Years After Filtration	…	-.099**	-.102**
		(.042)	(.042)
		{.021}	{.016}
4 Years After Filtration	…	-.133***	-.137***
		(.045)	(.046)
		{.014}	{.015}
5+ Years After Filtration	…	-.169***	…
		(.049)	
		{.008}	
5 Years After Filtration	…	…	-.142***
			(.043)
			{.006}
6 Years After Filtration	…	…	-.168***
			(.035)
			{.001}
7+ Years After Filtration	…	…	-.181***
			(.061)
			{.016}
Mean of infant mortality rate	206.4	206.4	206.4
N	1,024	1,024	1,024
R^2	.982	.982	.982

*Statistically significant at 10% level; ** at 5% level; *** at 1% level.

Notes: Based on annual data from *Mortality Statistics* for the period 1900-1940, published by the U.S. Census Bureau. Each column represents the results from a separate OLS regression. The dependent variable is equal to the natural log of the number of infant deaths per 100,000 population in city *c* and year *t*. Controls include the demographic characteristics and remaining public health interventions listed in Table 5, city fixed effects, year fixed effects and city-specific linear trends. Regressions are weighted by city population. Standard errors, corrected for clustering at the city level, are in parentheses. P-values from wild cluster bootstrap procedure are in curly brackets and are based on 1,000 replications.

Table 10. The Effects of Water Quality, Sewage Treatment/Diversion and Clean Milk on Mortality by Cause

	(1)	(2)	(3)	(4)	(5)
	Typhoid Mortality	*Diarrhea/ Enteritis Mortality*	*Diarrhea/ Enteritis Mortality (Under Age 2)*	*Non-Pulmonary TB Mortality*	*Non-Pulmonary TB Mortality (Under Age 2)*
Water					
Filtration	-.170**	-.154*	-.158	.038	.024
	(.070)	(.085)	(.099)	(.057)	(.028)
	{.025}	{.116}	{.167}	{.535}	{.426}
	[-4.56]				[.272]
Chlorination	.005	.089	.107	.008	-.007
	(.040)	(.079)	(.096)	(.054)	(.033)
	{.913}	{.389}	{.428}	{.914}	{.837}
	[.127]				[-.079]
Clean Water Project	.054	-.158	-.159	.073	.035
	(.075)	(.147)	(.198)	(.045)	(.028)
	{.555}	{.455}	{.538}	{.131}	{.180}
	[1.46]				[.395]
Sewage					
Sewage Treatment/Diversion	-.025	.190	.196	-.067	-.026
	(.057)	(.120)	(.148)	(.042)	(.027)
	{.703}	{.423}	{.477}	{.171}	{.409}
	[-.683]				[-.296]
Milk					
Bacteriological Standard	-.052	.028	.048	.028	.008
	(.037)	(.055)	(.066)	(.042)	(.027)
	{.213}	{.607}	{.458}	{.573}	{.755}
	[-1.41]				[.088]
TB Test	.040	.160*	.159	-.002	.004
	(.063)	(.078)	(.097)	(.041)	(.030)
	{.642}	{.122}	{.265}	{.961}	{.904}
	[1.07]				[.044]
Years	1900-1940	1900-1938	1900-1940	1900-1940	1900-1936
Mean of specified mortality rate	12.6	64.9	51.5	17.3	3.98
N	1,024	974	1,024	1,024	924
R^2	.939	.971	.964	.937	.843

*Statistically significant at 10% level; ** at 5% level; *** at 1% level.

Notes: Based on annual data from *Mortality Statistics*, published by the U.S. Census Bureau. Each column represents the results from a separate OLS regression. The dependent variable is equal to the natural log (or, in the case of typhoid and non-pulmonary TB for children under the age of 2, the quartic root) of the number of specified deaths per 100,000 population in city *c* and year *t*. Controls include the demographic characteristics listed in Table 5, city fixed effects, year fixed effects and city-specific linear trends. Regressions are weighted by city population. Standard errors, corrected for clustering at the city level, are in parentheses. P-values from wild cluster bootstrap procedure are in curly brackets and are based on 1,000 replications. Marginal effects are in square brackets.

Table 11. Comparing our Total Mortality Estimates to those of Cutler and Miller (2005)

	(1)	(2)	(3)	(4)	(5)	(6)
	Replicating C&M's original estimates	Column (1) + clustered standard errors	Column (2) + Memphis, TN correction	Column (3) + consistent population estimates used to calculate mortality rates	Column (4) + corrected filtration and chlorination dates	Our specification, limited to C&M's city-years
Filtration	-.162***	-.162**	-.134**	-.081**	-.043	-.042
	(.036)	(.064)	(.053)	(.028)	(.034)	(.027)
		{.059}	{.019}	{.034}	{.293}	{.157}
Chlorination	-.017	-.017	-.010	-.039	-.049*	-.019
	(.025)	(.034)	(.024)	(.026)	(.026)	(.029)
		{.621}	{.671}	{.215}	{.096}	{.511}
*Filtration*Chlorination*	.047**	.047	.032	.054**	.043	.031
	(.022)	(.031)	(.025)	(.024)	(.025)	(.037)
		{.154}	{.215}	{.071}	{.127}	{.459}
Years	1905-1936	1905-1936	1905-1936	1905-1936	1905-1936	1905-1936
Mean of total mortality rate	1,504	1,504	1,498	1,494	1,494	1,491
N	415	415	410	410	410	415
R^2	.957	.957	.963	.970	.969	.957

*Statistically significant at 10% level; ** at 5% level; *** at 1% level.

Notes: Based on annual data from *Mortality Statistics* for the period 1905-1936, published by the U.S. Census Bureau. Each column represents the results from a separate OLS regression. The dependent variable is equal to the natural log of the number of deaths per 100,000 population in city *c* and year *t*. Controls for the Cutler and Miller (2005) regressions shown in columns (1)-(5) include those listed in Appendix Table 7, city fixed effects, year fixed effects and city-specific linear trends. In column (6), controls include the demographic characteristics and remaining public health interventions listed in Table 5, city fixed effects, year fixed effects and city-specific linear trends. In columns (1)-(5), the regressions are unweighted. In column (6), the regression is weighted by city population. In column (1), Huber-White corrected standard errors are in parentheses. In columns (2)-(6), standard errors, corrected for clustering at the city level, are in parentheses. P-values from wild cluster bootstrap procedure are in curly brackets and are based on 1,000 replications.

Table 12. Comparing our Infant Mortality Estimates to those of Cutler and Miller (2005)

	(1)	(2)	(3)	(4)	(5)	(6)
	Replicating C&M's original estimates	Column (1) + clustered standard errors	Column (2) + Memphis, TN correction	Column (3) + corrected infant mortality counts	Column (4) + corrected filtration and chlorination dates	Our specification, limited to C&M's city-years
Filtration	-.429***	-.429***	-.373***	-.125*	-.038	-.096**
	(.090)	(.138)	(.111)	(.068)	(.071)	(.044)
		{.060}	{.012}	{.167}	{.661}	{.039}
Chlorination	-.085	-.085	-.072	.017	.035	.083
	(.083)	(.104)	(.104)	(.038)	(.059)	(.050)
		{.433}	{.509}	{.676}	{.712}	{.221}
*Filtration*Chlorination*	.056	.056	.034	.052	-.007	-.036
	(.074)	(.083)	(.077)	(.048)	(.039)	(.060)
		{.551}	{.714}	{.358}	{.843}	{.645}
Years	1905-1936	1905-1936	1905-1936	1905-1936	1905-1936	1905-1936
Mean of infant mortality rate	11,076	11,076	11,057	11,626	11,626	214.3
N	415	415	410	410	410	415
R^2	.828	.828	.835	.955	.955	.983

*Statistically significant at 10% level; ** at 5% level; *** at 1% level.

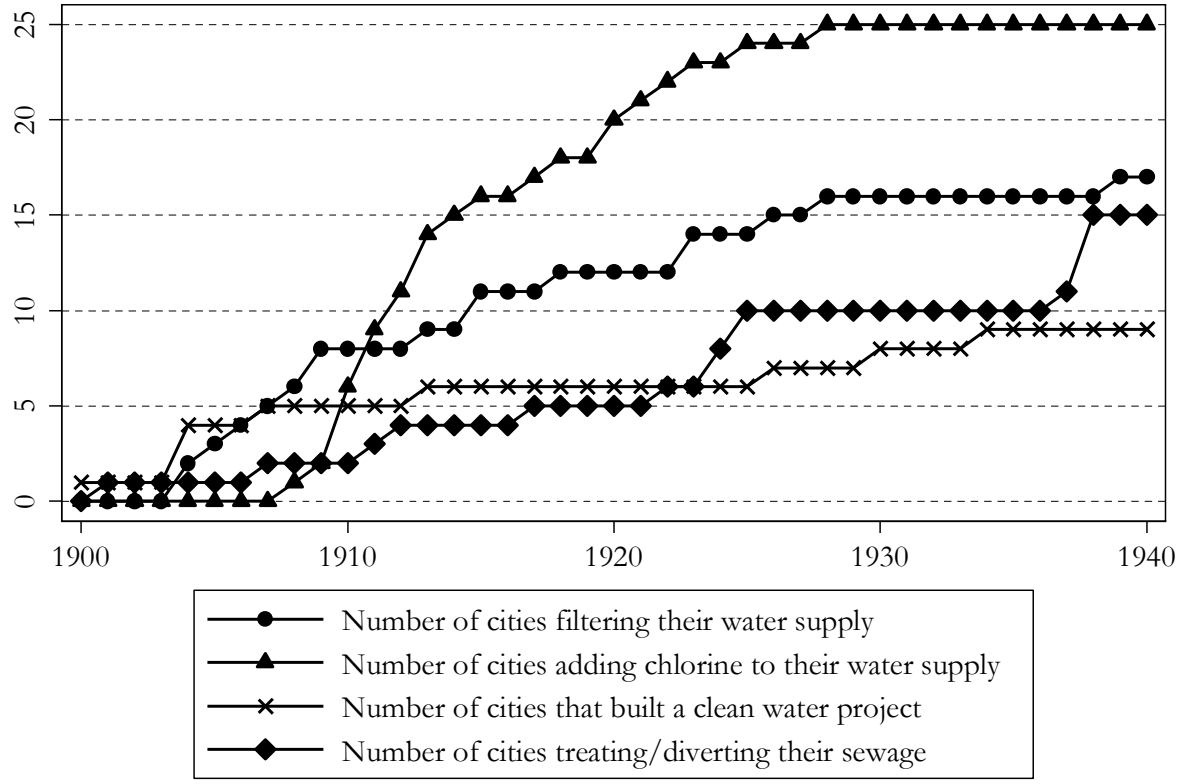
Notes: Based on annual data from *Mortality Statistics* for the period 1905-1936, published by the U.S. Census Bureau. Each column represents the results from a separate OLS regression. In columns (1)-(5), the dependent variable is equal to the natural log of the number of infant deaths per 100,000 infant population in city c and year *t*. In column (6), the dependent variable is equal to the natural log of the number of infant deaths per 100,000 population in city *c* and year *t*. Controls for the Cutler and Miller (2005) regressions shown in columns (1)-(5) include those listed in Appendix Table 7, city fixed effects, year fixed effects and city-specific linear trends. In column (6), controls include the demographic characteristics and remaining public health interventions listed in Table 5, city fixed effects, year fixed effects and city-specific linear trends. In columns (1)-(5), the regressions are unweighted. In column (6), the regression is weighted by city population. In column (1), Huber-White corrected standard errors are in parentheses. In columns (2)-(6), standard errors, corrected for clustering at the city level, are in parentheses. P-values from wild cluster bootstrap procedure are in curly brackets and are based on 1,000 replications.

Appendix

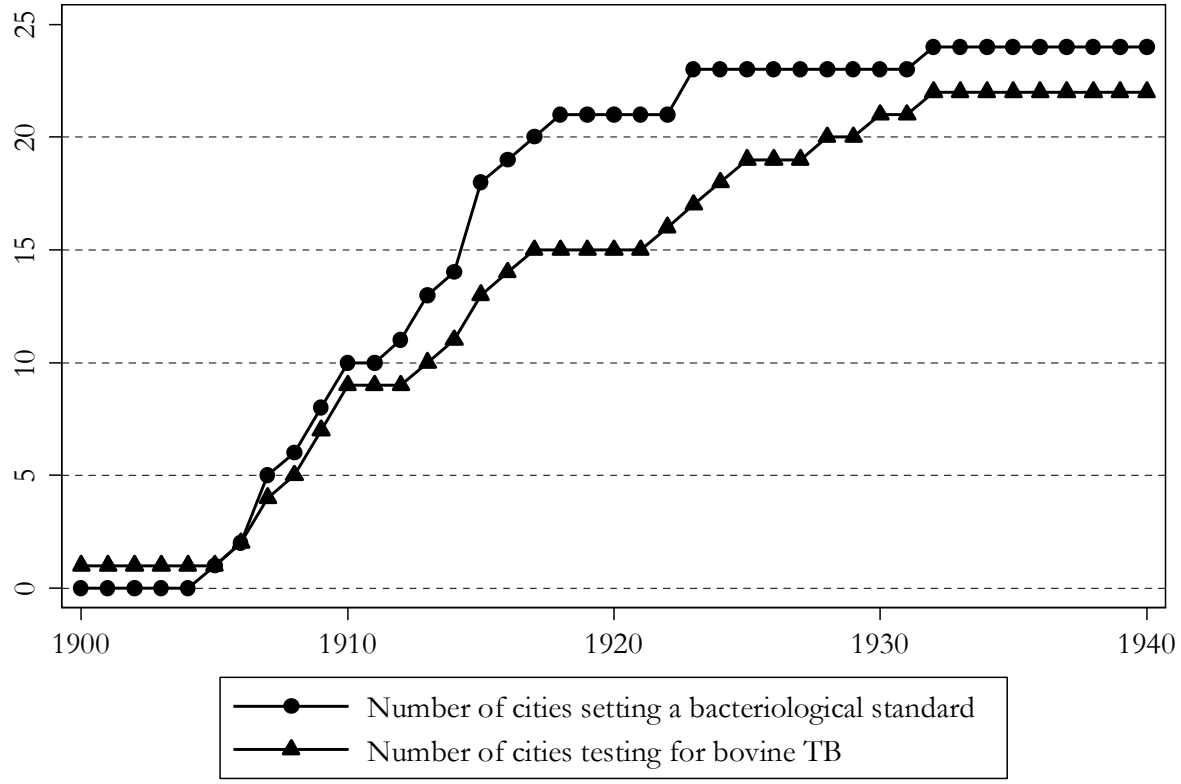
For Online Publication

Appendix A

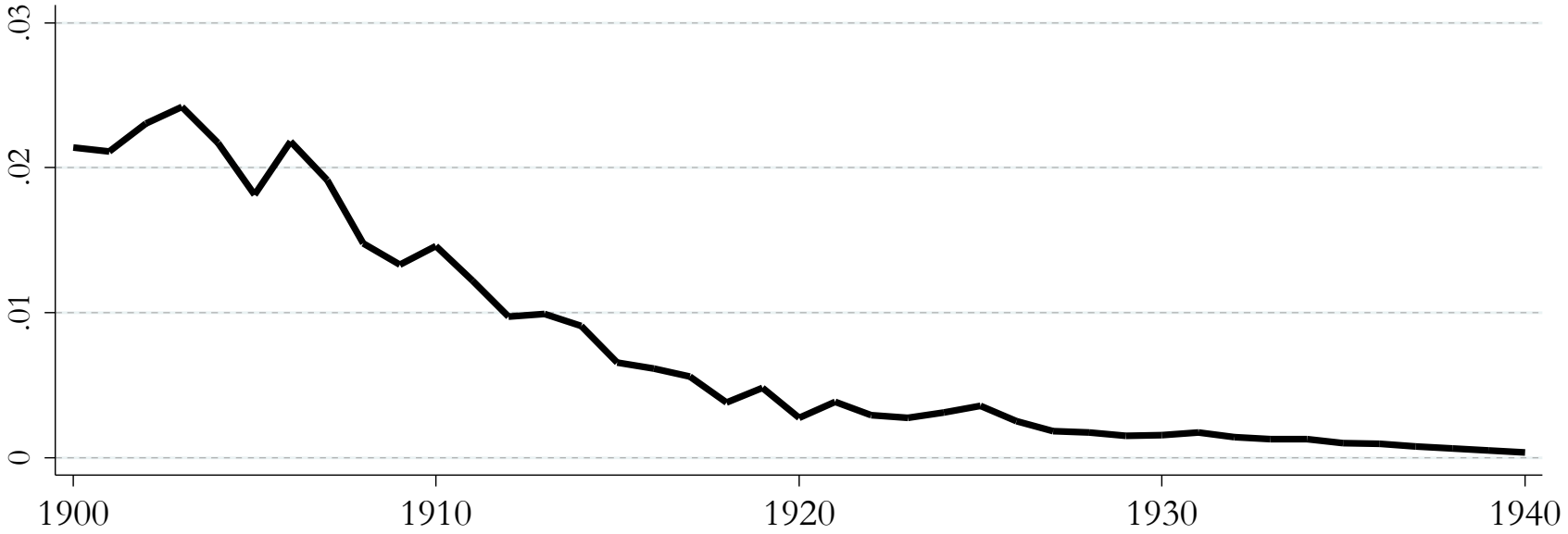
Appendix Figure 1. Municipal Water-Related Interventions Over Time



Appendix Figure 2. Municipal Milk-Related Interventions Over Time

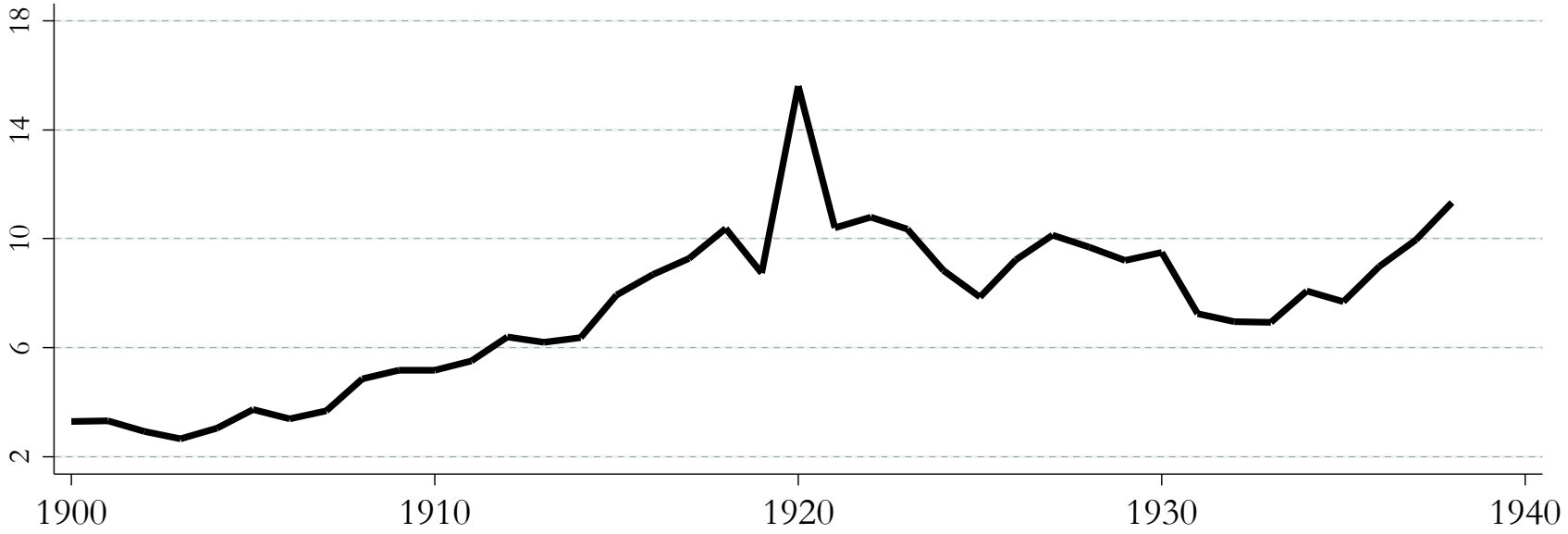


Appendix Figure 3. Percent of Total Mortality Due to Typhoid, 1900-1940



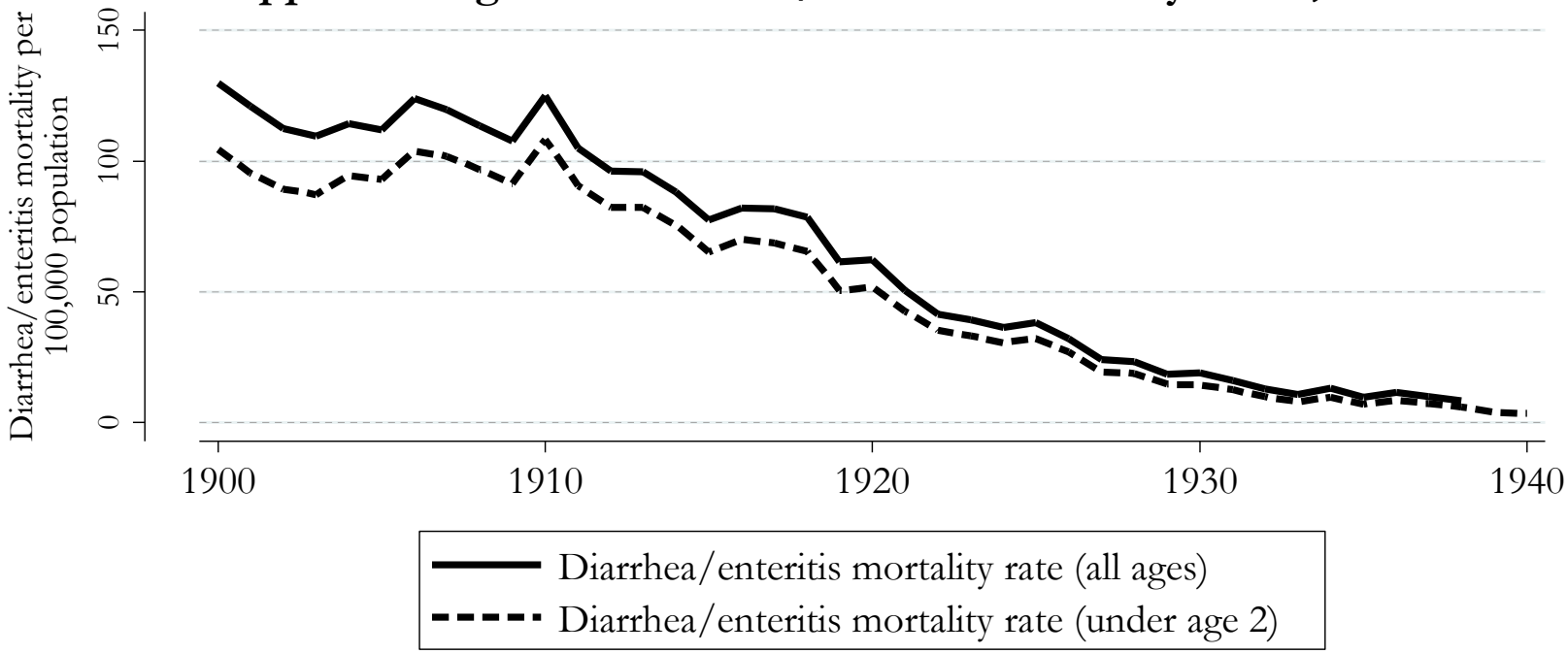
Notes: Based on annual data from *Mortality Statistics* and *Vital Statistics of the United States* for the period 1900-1940, published by the U.S. Census Bureau.

Appendix Figure 4. Ratio of Diarrhea/Enteritis to Typhoid Mortality, 1900-1938



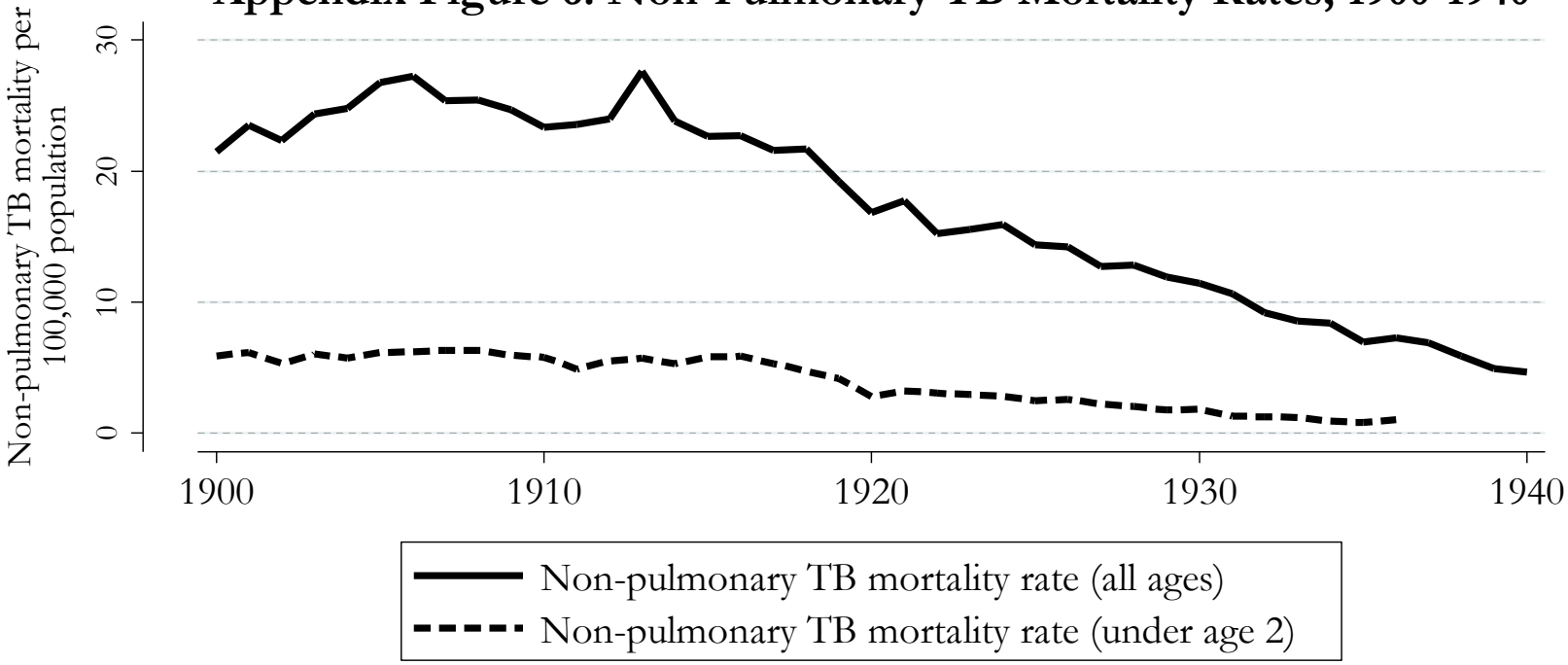
Notes: Based on annual data from *Mortality Statistics* and *Vital Statistics of the United States* for the period 1900-1938, published by the U.S. Census Bureau.

Appendix Figure 5. Diarrhea/Enteritis Mortality Rates, 1900-1940



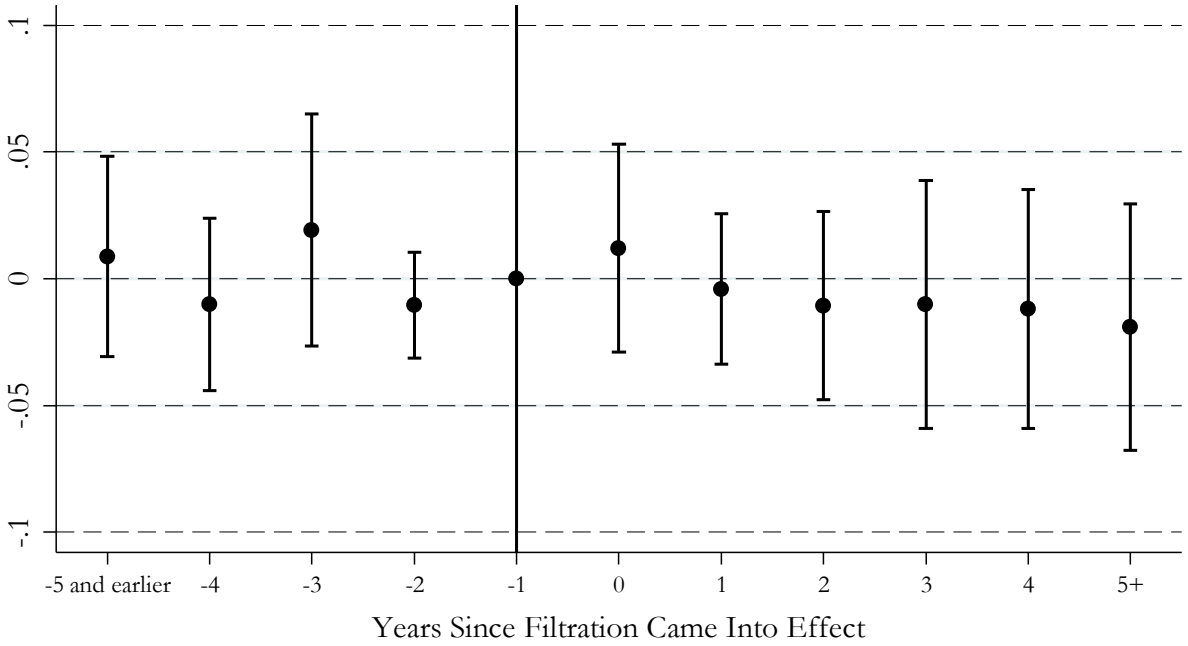
Notes: Based on annual data from *Mortality Statistics* and *Vital Statistics of the United States* for the period 1900-1940, published by the U.S. Census Bureau.

Appendix Figure 6. Non-Pulmonary TB Mortality Rates, 1900-1940



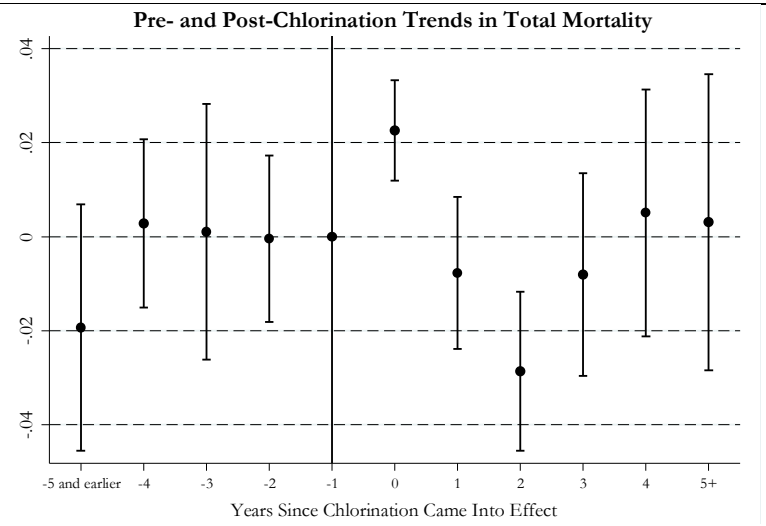
Notes: Based on annual data from *Mortality Statistics* and *Vital Statistics of the United States* for the period 1900-1940, published by the U.S. Census Bureau.

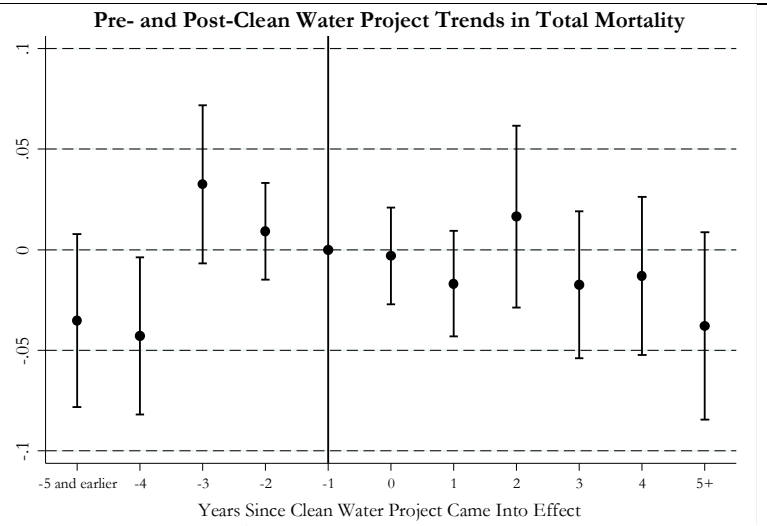
Appendix Figure 7. Pre- and Post-Filtration Trends in Total Mortality

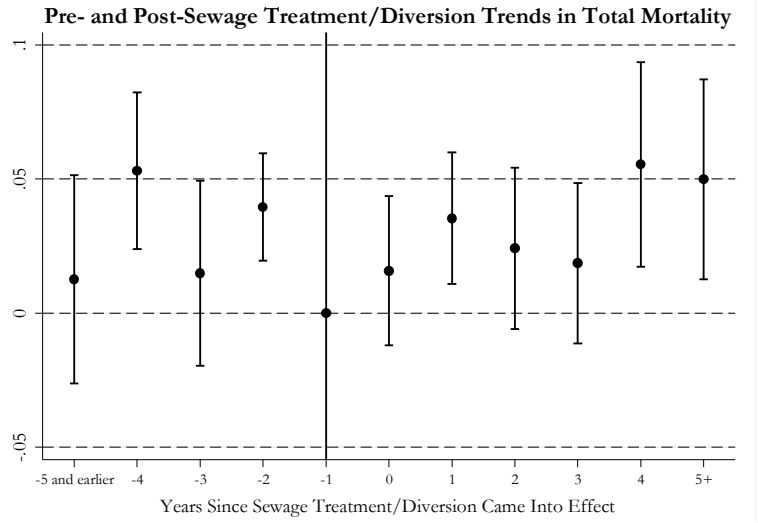


Notes: OLS coefficient estimates (and their 90% confidence intervals) are reported, where the omitted category is 1 year before treatment. The dependent variable is equal to the natural log of the number of deaths per 100,000 population in city *c* and year *t*. Controls include the demographic characteristics and remaining public health interventions listed in Table 5, city fixed effects, year fixed effects, and city-specific linear trends. Regressions are weighted by city population. Standard errors are corrected for clustering at the city level.

Appendix Figure 8

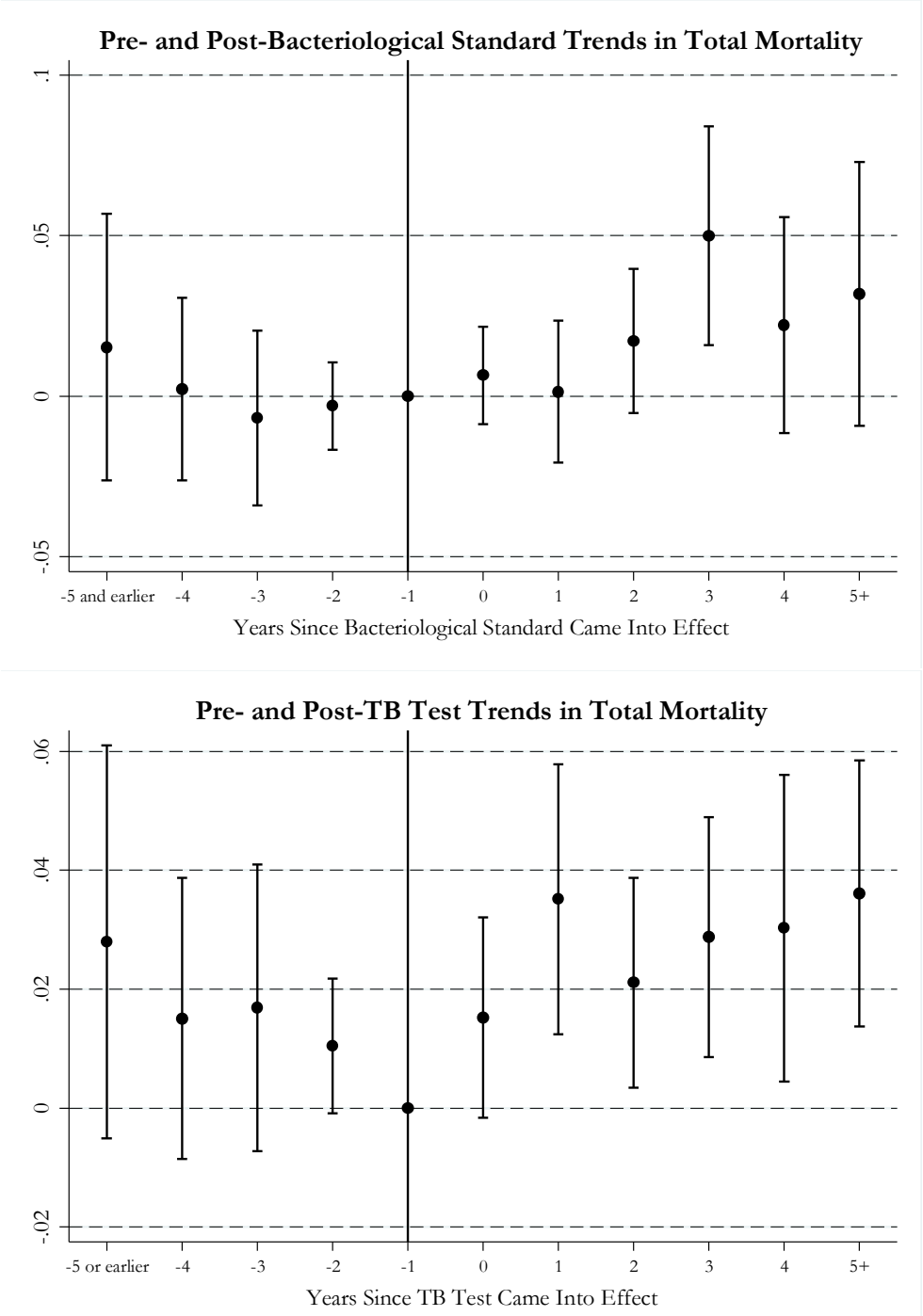






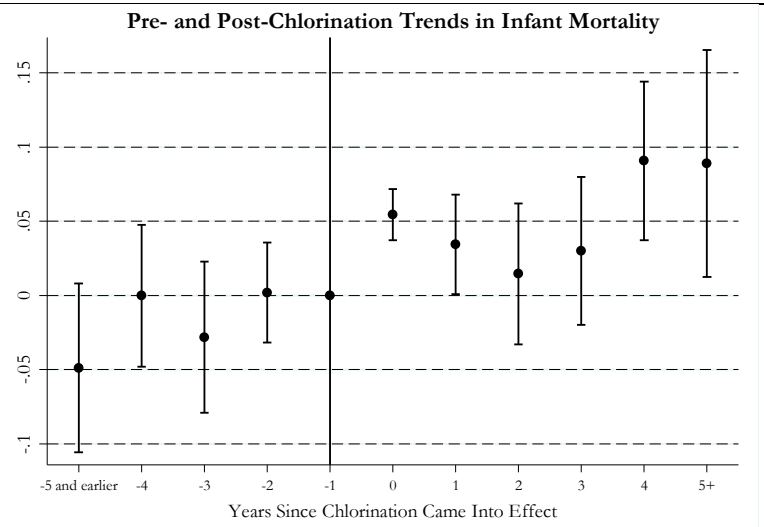
Notes: OLS coefficient estimates (and their 90% confidence intervals) are reported, where the omitted category is 1 year before treatment. The dependent variable is equal to the natural log of the number of deaths per 100,000 population in city *c* and year *t*. Controls include the demographic characteristics and remaining public health interventions listed in Table 5, city fixed effects, year fixed effects, and city-specific linear trends. Regressions are weighted by city population. Standard errors are corrected for clustering at the city level.

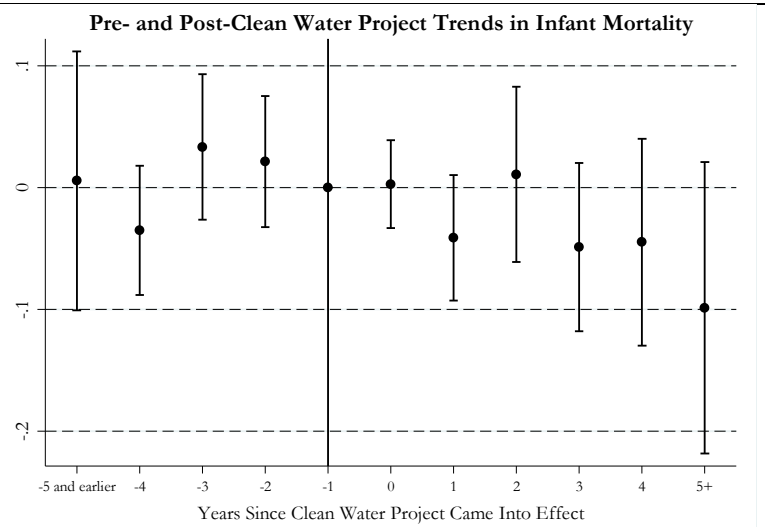
Appendix Figure 9

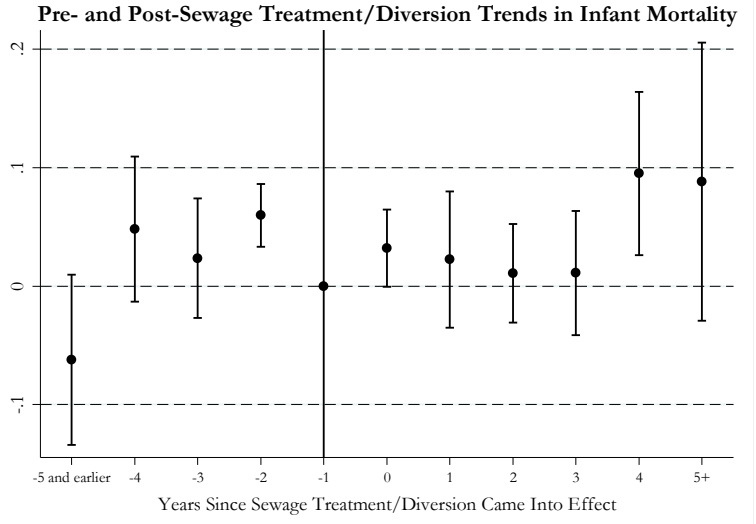


Notes: OLS coefficient estimates (and their 90% confidence intervals) are reported, where the omitted category is 1 year before treatment. The dependent variable is equal to the natural log of the number of deaths per 100,000 population in city *c* and year *t*. Controls include the demographic characteristics and remaining public health interventions listed in Table 5, city fixed effects, year fixed effects, and city-specific linear trends. Regressions are weighted by city population. Standard errors are corrected for clustering at the city level.

Appendix Figure 10

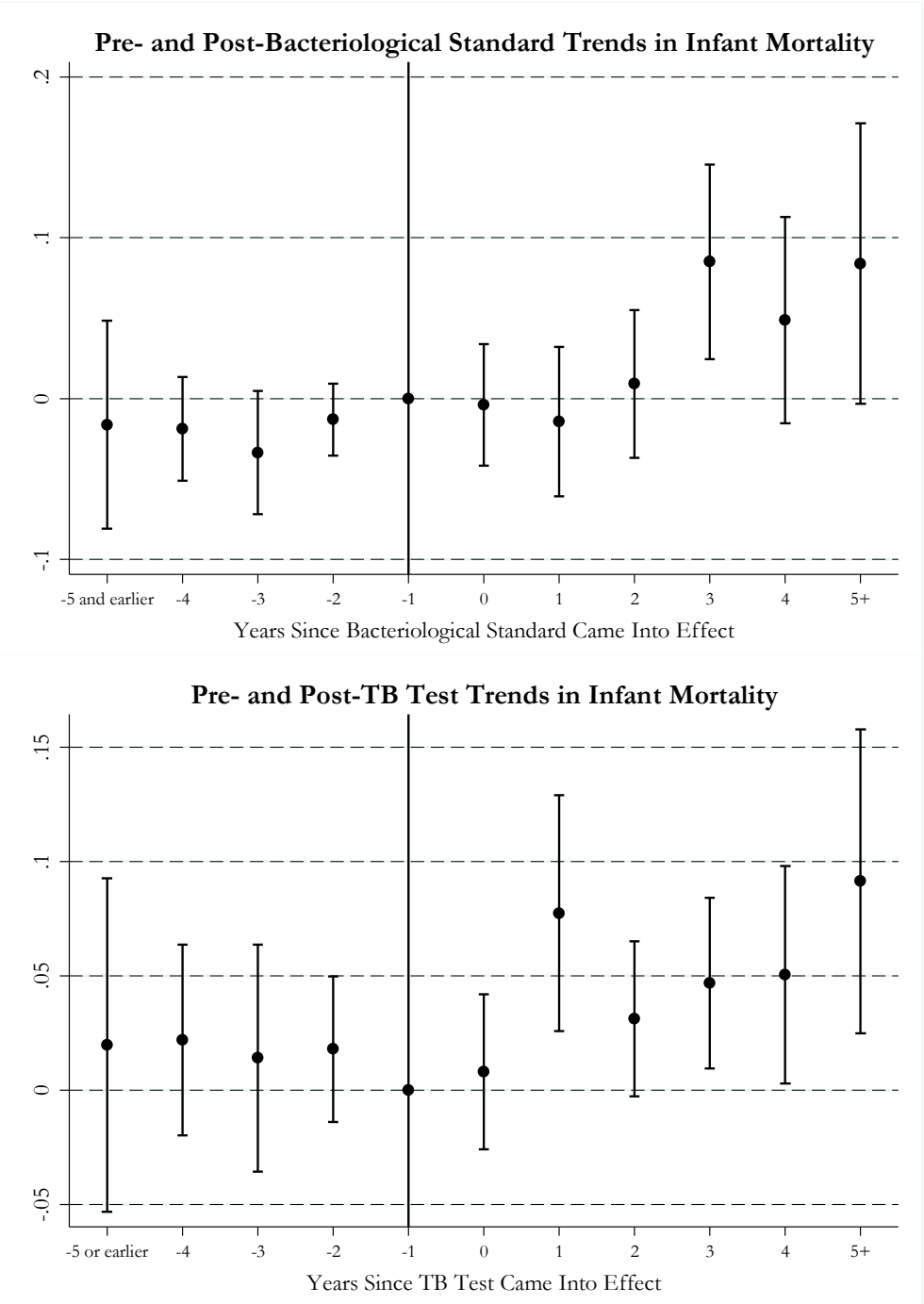






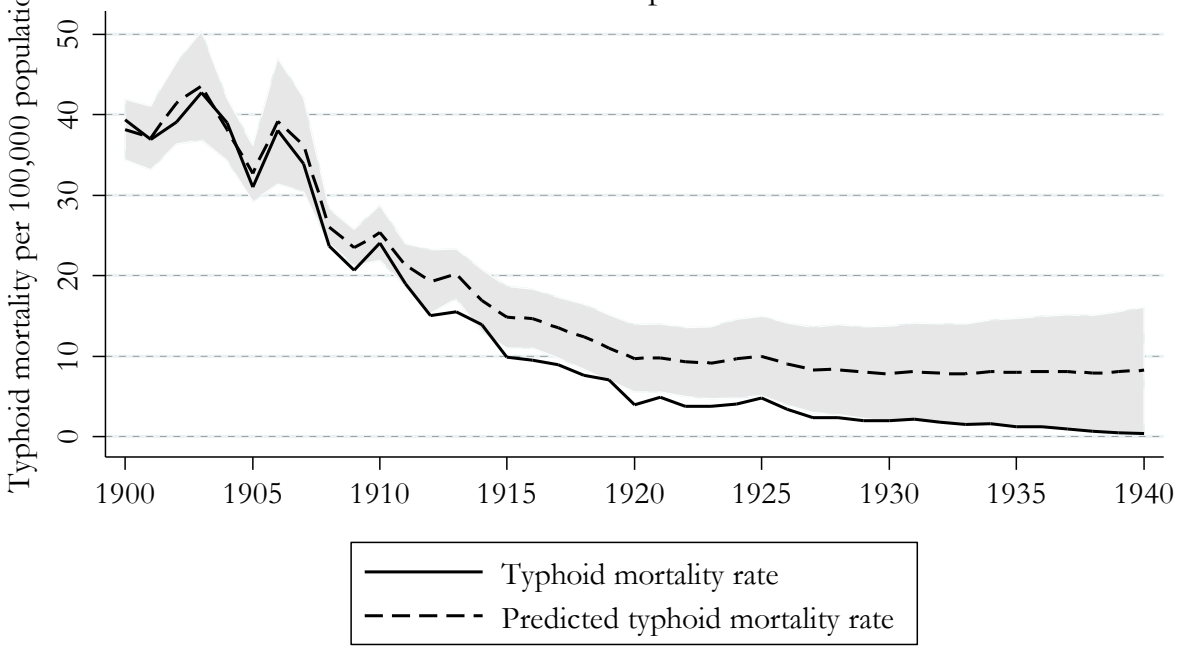
Notes: OLS coefficient estimates (and their 90% confidence intervals) are reported, where the omitted category is 1 year before treatment. The dependent variable is equal to the natural log of the number of infant deaths per 100,000 population in city *c* and year *t*. Controls include the demographic characteristics and remaining public health interventions listed in Table 5, city fixed effects, year fixed effects, and city-specific linear trends. Regressions are weighted by city population. Standard errors are corrected for clustering at the city level.

Appendix Figure 11



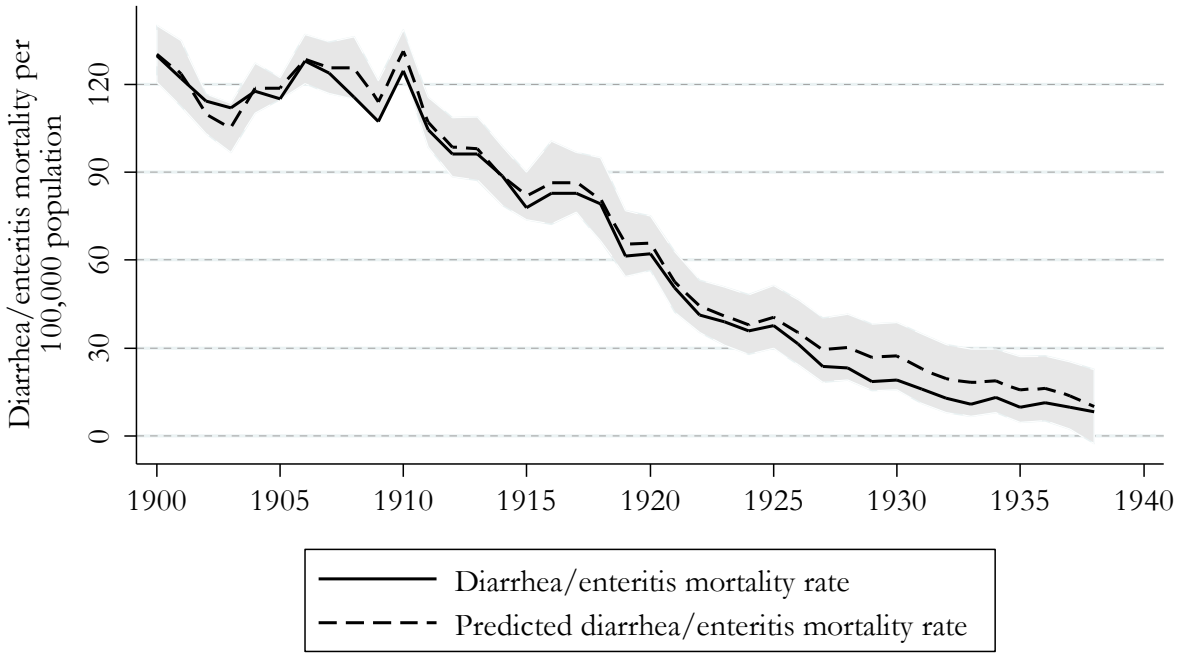
Notes: OLS coefficient estimates (and their 90% confidence intervals) are reported, where the omitted category is 1 year before treatment. The dependent variable is equal to the natural log of the number of infant deaths per 100,000 population in city *c* and year *t*. Controls include the demographic characteristics and remaining public health interventions listed in Table 5, city fixed effects, year fixed effects, and city-specific linear trends. Regressions are weighted by city population. Standard errors are corrected for clustering at the city level.

Appendix Figure 12. Actual vs. Predicted Typhoid Mortality Rates
The Effect of Municipal Water Filtration



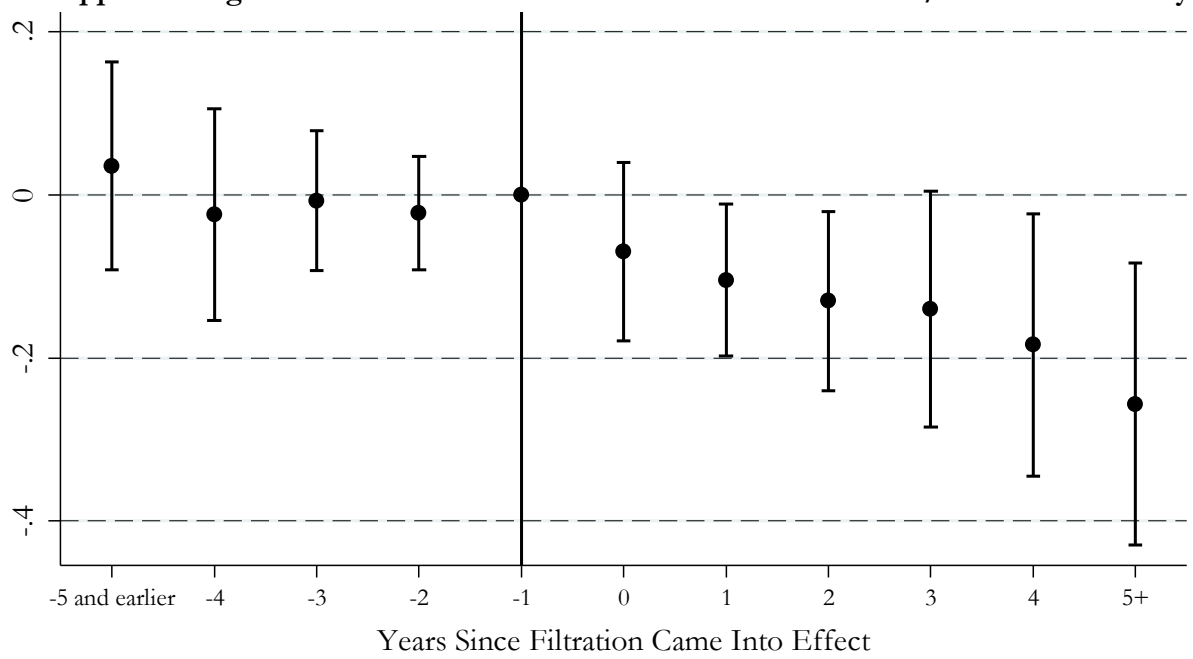
Notes: Based on annual data from *Mortality Statistics* and *Vital Statistics of the United States* for the period 1900-1940, published by the U.S. Census Bureau. Predicted typhoid mortality rates are calculated under the assumption that municipalities did not filter their water supply. Shaded area represents 90% confidence region around typhoid mortality rates.

Appendix Figure 13. Actual vs. Predicted Diarrhea/Enteritis Mortality Rates
The Effect of Municipal Water Filtration



Notes: Based on annual data from *Mortality Statistics* and *Vital Statistics of the United States* for the period 1900-1938, published by the U.S. Census Bureau. Predicted diarrhea/enteritis mortality rates are calculated under the assumption that municipalities did not filter their water supply. Shaded area represents 90% confidence region around diarrhea/enteritis mortality rates.

Appendix Figure 14. Pre- and Post-Filtration Trends in Diarrhea/Enteritis Mortality



Notes: OLS coefficient estimates (and their 90% confidence intervals) are reported, where the omitted category is 1 year before treatment. The dependent variable is equal to the natural log of the number of diarrhea/enteritis deaths per 100,000 population in city *c* and year *t*. Controls include the demographic characteristics and remaining public health interventions listed in Table 5, city fixed effects, year fixed effects, and city-specific linear trends. Regressions are weighted by city population. Standard errors are corrected for clustering at the city level.

Appendix Table 1. Robustness Checks: The Effects of Water Quality, Sewage Treatment/Diversion, and Clean Milk on Typhoid Mortality

	(1)	(2)	(3)	(4)	(5)	(6)
	Control for wages	Control for region-by-year fixed effects	Unweighted	Drop New York City	Drop years 1917-1920	Dependent variable in levels
Water						
Filtration	-.172**	-.188**	-.205***	-.168**	-.183**	-9.70*
	(.072)	(.079)	(.070)	(.070)	(.076)	(4.88)
	{.037}	{.014}	{.002}	{.044}	{.020}	{.033}
	[-4.88]	[-5.05]	[-5.48]	[-4.58]	[-5.08]	
Chlorination	.010	.019	.006	-.012	-.002	-.802
	(.038)	(.049)	(.041)	(.042)	(.041)	(2.03)
	{.808}	{.732}	{.897}	{.808}	{.965}	{.707}
	[.271]	[.514]	[.155]	[-.318]	[-.062]	
Clean Water Project	.043	.068	-.046	-.048	.049	5.11
	(.074)	(.099)	(.069)	(.113)	(.074)	(5.08)
	{.627}	{.560}	{.565}	{.750}	{.587}	{.437}
	[1.22]	[1.83]	[-1.23]	[-1.32]	[1.35]	
Sewage						
Sewage Treatment/Diversion	-.013	-.050	-.060	-.042	-.014	1.11
	(.062)	(.052)	(.054)	(.064)	(.055)	(1.83)
	{.850}	{.394}	{.332}	{.577}	{.826}	{.543}
	[-.374]	[-1.33]	[-1.62]	[-1.14]	[-.400]	
Milk						
Bacteriological Standard	-.052	-.069*	-.047	-.061	-.056	-3.68
	(.036)	(.039)	(.045)	(.048)	(.039)	(2.72)
	{.178}	{.099}	{.352}	{.311}	{.212}	{.232}
	[-1.47]	[-1.84]	[-1.25]	[-1.65]	[-1.54]	
TB Test	.029	.094*	.036	.045	.045	2.06
	(.060)	(.052)	(.066)	(.072)	(.069)	(2.53)
	{.702}	{.246}	{.629}	{.671}	{.631}	{.499}
	[.818]	[2.53]	[.970]	[1.23]	[1.26]	
Mean of typhoid mortality rate	13.6	12.6	12.6	12.9	13.3	12.6
N	949	1,024	1,024	983	924	1,024
R^2	.938	.948	.911	.929	.943	.818

*Statistically significant at 10% level; ** at 5% level; *** at 1% level.

Notes: Based on annual data from *Mortality Statistics* for the period 1900-1940, published by the U.S. Census Bureau. Each column represents the results from a separate OLS regression. In columns (1)-(5), the dependent variable is equal to the quartic root of the number of typhoid deaths per 100,000 population in city *c* and year *t*. In column (6), the dependent variable is equal to the number of typhoid deaths per 100,000 population in city c and year *t*. Controls include the demographic characteristics listed in Table 5, city fixed effects, year fixed effects and city-specific linear trends. In columns (1)-(2) and (4)-(6), regressions are weighted by city population. Standard errors, corrected for clustering at the city level, are in parentheses. P-values from wild cluster bootstrap procedure are in curly brackets and are based on 1,000 replications. Marginal effects are in square brackets.

Appendix Table 2. Typhoid Mortality and Lags of *Filtration*

	(1)	(2)	(3)
	Typhoid Mortality		
Year 0	.042	.036	.033
	(.078)	(.078)	(.078)
	{.744}	{.783}	{.806}
	[1.13]	[.964]	[.891]
1 Year After Filtration	-.089	-.096	-.099
	(.088)	(.088)	(.087)
	{.370}	{.330}	{.309}
	[-2.39]	[-2.57]	[-2.65]
2 Years After Filtration	-.130	-.139*	-.142*
	(.076)	(.076)	(.076)
	{.153}	{.127}	{.120}
	[-3.48]	[-3.71]	[-3.81]
3+ Years After Filtration	-257***	…	…
	(.067)		
	{.002}		
	[-6.88]		
3 Years After Filtration	…	-.211***	-.214***
		(.067)	(.068)
		{.004}	{.004}
		[-5.64]	[-5.75]
4 Years After Filtration	…	-.219***	-.224***
		(.075)	(.076)
		{.035}	{.035}
		[-5.87]	[-5.99]
5+ Years After Filtration	…	-.286***	…
		(.072)	
		{.003}	
		[-7.66]	
5 Years After Filtration	…	…	-.270***
			(.066)
			{.007}
			[-7.24]
6 Years After Filtration	…	…	-.267***
			(.070)
			{.006}
			[-7.16]
7+ Years After Filtration	…	…	-.300***
			(.081)
			{.011}
			[-8.04]
Mean of typhoid mortality rate	12.6	12.6	12.6
N	1,024	1,024	1,024
R^2	.941	.941	.942

*Statistically significant at 10% level; ** at 5% level; *** at 1% level.

Notes: Based on annual data from *Mortality Statistics* for the period 1900-1940, published by the U.S. Census Bureau. Each column represents the results from a separate OLS regression. The dependent variable is equal to the quartic root of the number of typhoid deaths per 100,000 population in city *c* and year *t*. Controls include the demographic characteristics and remaining public health interventions listed in Table 5, city fixed effects, year fixed effects and city-specific linear trends. Regressions are weighted by city population. Standard errors, corrected for clustering at the city level, are in parentheses. P-values from wild cluster bootstrap procedure are in curly brackets and are based on 1,000 replications. Marginal effects are in square brackets.

Appendix Table 3. Robustness Checks: The Effects of Water Quality, Sewage Treatment/Diversion, and Clean Milk on Diarrhea/Enteritis Mortality

	(1)	(2)	(3)	(4)	(5)	(6)
	Control for wages	Control for region-by-year fixed effects	Unweighted	Drop New York City	Drop years 1917-1920	Dependent variable in levels
Water						
Filtration	-.139*	-.145	-.209***	-.171**	-.147	-6.93
	(.076)	(.087)	(.058)	(.065)	(.089)	(9.35)
	{.090}	{.152}	{.004}	{.022}	{.150}	{.521}
Chlorination	.072	-.010	.034	.149*	.095	6.56
	(.066)	(.066)	(.076)	(.072)	(.086)	(5.88)
	{.381}	{.911}	{.716}	{.175}	{.381}	{.391}
Clean Water Project	-.128	-.234**	.142	.135	-.122	-22.5*
	(.118)	(.112)	(.140)	(.186)	(.135)	(11.0)
	{.404}	{.147}	{.420}	{.568}	{.503}	{.309}
Sewage						
Sewage Treatment/Diversion	.206*	.203**	.141	.259**	.172	13.7
	(.117)	(.090)	(.092)	(.098)	(.112)	(10.8)
	{.400}	{.195}	{.181}	{.118}	{.430}	{.579}
Milk						
Bacteriological Standard	.038	.094	.100	.040	.009	-1.75
	(.051)	(.069)	(.066)	(.071)	(.057)	(4.61)
	{.448}	{.241}	{.238}	{.564}	{.888}	{.714}
TB Test	.154**	.106**	.102	.217**	.177**	10.7*
	(.073)	(.046)	(.079)	(.079)	(.073)	(5.78)
	{.116}	{.033}	{.309}	{.062}	{.075}	{.159}
Mean of diarrhea/enteritis mortality rate	66.4	64.9	64.9	64.6	64.2	64.9
N	949	974	974	935	874	974
R^2	.972	.979	.957	.968	.974	.939

*Statistically significant at 10% level; ** at 5% level; *** at 1% level.

Notes: Based on annual data from *Mortality Statistics* for the period 1900-1938, published by the U.S. Census Bureau. Each column represents the results from a separate OLS regression. In columns (1)-(5), the dependent variable is equal to the natural log of the number of diarrhea/enteritis deaths per 100,000 population in city *c* and year *t*. In column (6), the dependent variable is equal to the number of diarrhea/enteritis deaths per 100,000 population in city *c* and year *t*. Controls include the demographic characteristics listed in Table 5, city fixed effects, year fixed effects and city-specific linear trends. In columns (1)-(2) and (4)-(6), regressions are weighted by city population. Standard errors, corrected for clustering at the city level, are in parentheses. P-values from wild cluster bootstrap procedure are in curly brackets and are based on 1,000 replications.

Appendix Table 4. Diarrhea/Enteritis Mortality and Lags of *Filtration*

	(1)	(2)	(3)
	Diarrhea/Enteritis Mortality		
Year 0	-.066	-.074	-.078
	(.076)	(.076)	(.078)
	{.450}	{.409}	{.391}
1 Year After Filtration	-.099	-.108*	-.113*
	(.062)	(.062)	(.064)
	{.111}	{.089}	{.088}
2 Years After Filtration	-.122*	-.134**	-.138**
	(.063)	(.063)	(.063)
	{.067}	{.053}	{.040}
3+ Years After Filtration	-.213**	…	…
	(.099)		
	{.047}		
3 Years After Filtration	…	-.143	-.148
		(.089)	(.088)
		{.107}	{.089}
4 Years After Filtration	…	-.185*	-.191*
		(.106)	(.106)
		{.126}	{.116}
5+ Years After Filtration	…	-.251**	…
		(.108)	
		{.040}	
5 Years After Filtration	…	…	-.209*
			(.104)
			{.073}
6 Years After Filtration	…	…	-.253**
			(.100)
			{.027}
7+ Years After Filtration	…	…	-.270**
			(.124)
			{.066}
Mean of dependent variable	64.9	64.9	64.9
N	974	974	974
R^2	.971	.971	.971

*Statistically significant at 10% level; ** at 5% level; *** at 1% level.

Notes: Based on annual data from *Mortality Statistics* for the period 1900-1938, published by the U.S. Census Bureau. Each column represents the results from a separate OLS regression. The dependent variable is equal to the natural log of the number of diarrhea/enteritis deaths per 100,000 population in city *c* and year *t*. Controls include the demographic characteristics and remaining public health interventions listed in Table 5, city fixed effects, year fixed effects and city-specific linear trends. Regressions are weighted by city population. Standard errors, corrected for clustering at the city level, are in parentheses. P-values from wild cluster bootstrap procedure are in curly brackets and are based on 1,000 replications.

Appendix Table 5. Comparing our Preferred Specification to that of Cutler and Miller (2005)

	C&M Specification	Our Specification
Set of controls	See Appendix Table 7	See Table 5
Model choice	OLS	OLS
Weighting	Unweighted	Weighted by city population
Standard errors	Huber-White	Clustered at the city level
N	415	1,024
Years	1905-1936	1900-1940
Number of cities	13	25
	City in C&M sample	City in our sample
Baltimore, MD	yes	yes
Boston, MA	…	yes
Buffalo, NY	…	yes
Chicago, IL	yes	yes
Cincinnati, OH	yes	yes
Cleveland, OH	yes	yes
Detroit, MI	yes	yes
Indianapolis, IN	…	yes
Jersey City, NJ	yes	yes
Kansas City, MO	…	yes
Louisville, KY	yes	yes
Memphis, TN	yes	yes
Milwaukee, WI	yes	yes
Minneapolis, MN	…	yes
Newark, NJ	…	yes
New Orleans, LA	yes	yes
New York, NY	…	yes
Philadelphia, PA	yes	yes
Pittsburgh, PA	yes	yes
Providence, RI	…	yes
Rochester, NY	…	yes
San Francisco, CA	…	yes
St. Louis, MO	yes	yes
St. Paul, MN	…	yes
Washington, D.C.	…	yes

Appendix Table 6. Comparing City Characteristics of our Sample to that of Cutler and Miller (2005)

	Mean (SD)	
	C&M Sample	Our Sample
Population	800,805	806,454
	(724,467)	(1,174,634)
% Female	.502	.503
	(.015)	(.016)
% Nonwhite	.116	.090
	(.107)	(.100)
% Foreign	.175	.193
	(.100)	(.109)
% Under 15	.261	.255
	(.025)	(.032)
% 15 to 44	.531	.529
	(.022)	(.025)
% 45 and Older	.208	.216
	(.032)	(.038)
N	415	1,024

Notes: Unweighted means with standard deviations in parentheses.

Appendix Table 7. List of Controls in Cutler and Miller (2005)

	Description
Filtration	= 1 if city had a water filtration plant, = 0 otherwise
Chlorination	= 1 if city chemically treated water supply, = 0 otherwise
Filtration w/in 5 Years	= 1 if city began filtering water supply within 5 years, = 0 otherwise
Chlorination w/in 5 Years	= 1 if city began chemically treating water supply within 5 years, = 0 otherwise
Sewage Treatment [a]	= 1 if city had a sewage treatment plant, = 0 otherwise
Sewage Chlorination[b]	= 1 if city chemically treated its sewage, = 0 otherwise
Lake Michigan Outfalls	= 1 for Chicago after Lake Michigan sewer outfalls were shut off, = 0 otherwise
Cleveland Intake Tunnel	= 1 for Cleveland after intake tunnel was built to draw water from Lake Erie, = 0 otherwise
$\ln(Mortality)_{t-1}$	One-year lag of natural log of city mortality rate
$\ln(Mortality)_{t-2}$	Two-year lag of natural log of city mortality rate
$\ln(Mortality)_{t-3}$	Three-year lag of natural log of city mortality rate
$\ln(Mortality)_{t-4}$	Four-year lag of natural log of city mortality rate
$\ln(Mortality)_{t-5}$	Five-year lag of natural log of city mortality rate
ln(*Population*)	Natural log of city population
% Female	Percent of city population that was female
% Black	Percent of city population that was black
% Other Nonwhite	Percent of city population that was a nonwhite race other than black
% Foreign	Percent of city population that was foreign born
% Under 1	Percent of city population that was under 1 years of age
% 1 to 4	Percent of city population that was 1 to 4 years of age
% 5 to 9	Percent of city population that was 5 to 9 years of age
% 10 to 14	Percent of city population that was 10 to 14 years of age
% 15 to 19	Percent of city population that was 15 to 19 years of age
% 20 to 24	Percent of city population that was 20 to 24 years of age
% 25 to 34	Percent of city population that was 25 to 34 years of age
% 35 to 44	Percent of city population that was 35 to 44 years of age
% 45 to 64	Percent of city population that was 45 to 64 years of age
% 65 and Older	Percent of city population that was 65 years of age or older

[a] Three cities in C&M's sample period constructed sewage treatment plants (Baltimore in 1911, Cleveland in 1922 and Milwaukee in 1925).

[b] One city in C&M's sample period chlorinated its sewage (Cleveland in 1922).

Appendix Table 8. Differences in Recorded Infant Mortality Counts between Cutler and Miller (2005) and the U.S. Census Bureau's *Mortality Statistics*

City	Year	C&M's recorded infant mortality count[a]	Correct infant mortality count from *Mortality Statistics*[b]	Reason for difference (when known)
Baltimore, MD	1910	1417.07	2146	
	1911	1295.99	1960	
	1912	1384.03	2022	
	1913	1343	2011	
	1914	1312.43	1949	
	1915	1093.48	1626	
	1916	1158.78	1770	
	1917	1183.75	1780	
Chicago, IL	1910	6595.52	6844	
	1911	6017.86	6252	
	1912	6394.31	6678	
	1913	6649.87	6939	
	1914	6571.52	6878	
	1915	5942.99	6219	
	1916	6566.35	6910	
	1917	6246.72	6664	
	1931	766	2992	To calculate, one needs to add white infant mortality (=2,617) and nonwhite infant mortality (=375). It appears as if C&M incorrectly added mortality for one-year-olds, rather than infants, for whites (=391) and nonwhite infant mortality, which gives their recorded total of 766.
Cincinnati, OH	1910	793.435	917	
	1911	630.712	721	
	1912	693.419	805	
	1913	706.664	801	
	1914	637.264	750	
	1915	524.823	619	
	1916	623.426	736	
	1917	563.757	688	
Cleveland, OH	1924	2366	1386	To calculate, one needs to add white infant mortality (=1,219) and nonwhite infant mortality (=167). It appears as if C&M incorrectly added overall nonwhite mortality (=1,147) and white infant mortality (=1,219), which gives their recorded total of 2,366.
Detroit, MI	1920	2734	2885	C&M incorrectly recorded infant mortality for whites only, which was 2,734. Nonwhite infant mortality was 151.

Jersey City, NJ				No mistakes for Jersey City
Louisville, KY	1910	328.389	503	
	1911	298.928	441	
	1912	46.4965	448	
	1913	322.27	486	
	1914	338.518	496	
	1915	250.146	379	
	1916	283.827	418	
	1917	271.929	397	
Memphis, TN	1910	173.2	345	
	1911	175.645	348	
	1912	185.973	373	
	1913	157.88	319	
	1914	158.831	317	
	1915	118.738	228	
	1916	0	...	Data for Memphis, TN are not reported in 1916.
	1917	158.43	311	
Milwaukee, WI	1926	865	856	C&M incorrectly transposed the "5" and "6"
New Orleans, LA	1910	571.931	1061	
	1911	595.471	1071	
	1912	416.903	774	
	1913	500.74	934	
	1914	477.419	883	
	1915	492.79	927	
	1916	404.008	757	
	1917	446.364	866	
Philadelphia, PA	1910	4557.6	5334	
	1911	4093.3	4769	
	1912	3659.92	4201	
	1913	3925.69	4618	
	1914	4170.24	4870	
	1915	3634.78	4233	
	1916	3669.4	4252	
	1917	3921.49	4637	
Pittsburgh, PA	1901	6578	1580	C&M incorrectly recorded the overall mortality count instead of the infant mortality count.
	1904	771	1771	C&M incorrectly entered "1771" as "771"
	1910	2024.02	2259	
	1911	1648.01	1812	
	1912	1648.71	1811	

	1913	1754.65	1957
	1914	1672.1	1868
	1915	1670.73	1765
	1916	1688.55	1893
	1917	1744.14	1983
	1924	1530	1440
St. Louis, MO	1910	1452.74	1689
	1911	1345.61	1573
	1912	1263.1	1467
	1913	1246.83	1478
	1914	1278.93	1508
	1915	1014.65	1181
	1916	1061.51	1264
	1917	1012.88	1252

[a] In the Cutler and Miller data set, the variable "mort0_1" represents the infant mortality count. Their dependent variable of interest, the natural log of the infant mortality rate, can be recreated with the following STATA command:

gen lninfmrt = ln((mort0_1*100000)/age0_1), where "age0_1" is the city infant population.

[b] The infant mortality counts for each year listed above can be found in the following *Mortality Statistics* tables:

Year	Location
1901	*Mortality Statistics 1900 to 1904*, Table 2, pp. 180-197; or Table 8, pp. 270-311
1904	*Mortality Statistics 1900 to 1904*, Table 2, pp. 654-671; or Table 8, pp. 744-785
1910	*Mortality Statistics 1910*, Table 3, pp. 204-251; or Table 9, pp. 455-501; or Table 11, pp. 533-574
1911	*Mortality Statistics 1911*, Table 1, pp. 150-173; or Table 2, pp. 174-257; or Table 7, pp. 466-512; or Table 9, pp. 537-567
1912	*Mortality Statistics 1912*, Table 1, pp. 28-49; or Table 6, pp. 255-301; or Table 8, pp. 335-377
1913	*Mortality Statistics 1913*, Table 1, pp. 222-243; or Table 6, pp. 486-539; or Table 8, pp. 577-625
1914	*Mortality Statistics 1914*, Table 3, pp. 192-219; or Table 4, pp. 220-303; or Table 9, pp. 567-621; or Table 11, pp. 660-709
1915	*Mortality Statistics 1915*, Table 3, pp. 184-211; or Table 4, pp. 212-297; or Table 9, pp. 553-607; or Table 11, pp. 645-694
1916	*Mortality Statistics 1916*, Table 3, pp. 150-175; or Table 9, pp. 406-449; or Table 11, pp. 483-525
1917	*Mortality Statistics 1917*, Table 3, pp. 172-197; or Table 9, pp. 441-487; or Table 11, pp. 523-568
1920	*Mortality Statistics 1920*, Table 3, pp. 140-174; or Table 9, pp. 479-539; or Table 11, pp. 586-646
1924	*Mortality Statistics 1924*, Table 9, pp. 358-390
1926	*Mortality Statistics 1926*, Table 9, pp. 285-317
1931	*Mortality Statistics 1931*, Table 9, pp. 396-440

Appendix B

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