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Its Waning, 1910-1930**

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ABSTRACT

The Phenomenon of Summer Diarrhea and Its Waning, 1910-1930

During the first two decades of the 20th century, diarrheal deaths among American infants and children surged every summer. Although we still do not know what pathogen (or pathogens) caused this phenomenon, the consensus view is that it was eventually controlled through public health efforts at the municipal level. Using data from 26 major American cities for the period 1910-1930, we document the phenomenon of summer diarrhea and explore its dissipation. We find that water filtration is associated with a 15-17 percent reduction in diarrheal mortality among children under the age of two during the non-summer months, but does not seem to have had an effect on diarrheal mortality during the summer. In general, we find little evidence to suggest that public health interventions undertaken at the municipal level contributed to the dissipation of summer diarrhea. Our results are relevant for many parts of the developing world today, where climate change is expected to affect the length and intensity of seasons as well as the incidence of diarrheal diseases.

JEL Classification: I10, I18, N3, Q54

Keywords: diarrheal mortality, infant mortality, public health

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1. INTRODUCTION

Summer diarrhea (also known as “cholera infantum” or the “disease of the season”) was first described in the medical literature by Benjamin Rush (1794). According to Rush (1794, p. 160), it would sometimes begin “with a diarrhoea, which can last for several days without any other symptom...but it more frequently comes on with a violent vomiting and purging, and a high fever.” He argued that it was likely caused by the “discharge of bile which generally introduces the disease...and exacerbations of the fever which accompanies it...” (Rush 1794, p. 163).

By the turn of the 20th century, physicians had gained a better understanding of how gastronomical diseases are contracted but still had not discovered why thousands of American infants and children were dying of diarrhea every summer. In fact, to this day, we can only speculate as to precisely what pathogen (or pathogens) caused the phenomenon (Meckel 1990; Thompson and Keeling 2012).

Economists and historians have argued that summer diarrhea was controlled through the combined effects of public health interventions (Meckel 1990; Fishback et al. 2011). Specifically, they point to municipal-level efforts during the early 1900s to purify water and milk supplies. Because breastfeeding was typically discontinued at an early age (Wolf 2003), and because there is anecdotal evidence that children were at the greatest risk of dying from diarrhea in their “second summer” (Condie 1858, p. 224; Moss 1903; Southworth 1904), focusing on these efforts is not without justification.¹

¹ See also Reedy (2007, p. 42), who asserted that summer diarrhea was controlled through “handwashing and safer food-handling procedures at home and in the marketplace” along with “better built and maintained sewage treatment plants” that “helped keep infants and children from playing in and drinking contaminated water.” Graham-Smith (1929) attributed the dissipation of summer diarrhea among infants in England to the introduction of the automobile, which had the effect of reducing horse manure in the streets.

In this study, we begin by documenting the phenomenon of summer diarrhea and its correlates using newly transcribed mortality data from 26 major American cities for the period 1910-1930. These data, which were originally published by the U.S. Bureau of the Census, include monthly counts of diarrheal deaths among children under the age of two. We show that 21,000 children under the age of two died of diarrhea in 1910, and 14,000 of these deaths occurred in the months of June-September. By the end of the period under study, the phenomenon of summer diarrhea had largely dissipated. Fewer than 4,000 children under the age of two died of diarrhea in 1930, and fewer than 1,500 of these deaths occurred in the summer.²

In the second half of the paper, we use data from these same 26 cities to estimate whether public health efforts at the municipal level contributed to the waning of summer diarrhea during the period 1910-1930.³ We find that the construction of a water filtration plant is associated with a 15-17 percent reduction in diarrheal mortality during non-summer months, consistent with the hypothesis that transmission occurred through contaminated water. By contrast, filtration had little effect on diarrheal mortality during the hottest 4 months of the year (June-September), which suggests that transmission was most likely through contaminated food or person-to-person contact, although efforts to purify the milk supply do not appear to have affected diarrheal mortality during the summer either. In fact, we find little evidence that public health interventions can explain the waning of summer diarrhea. We conclude that the phenomenon of summer diarrhea was serious and widespread, but, contrary to the consensus view, its dissipation

² In 1910, the ratio of diarrheal deaths to the population of children under the age of two was .031 (21,101/678,292 = .031). By 1920, this ratio had dropped to .015; by 1930, it had dropped to .005.

³ We focus on the period 1910-1930 because monthly-level mortality data are not available from the U.S. Bureau of the Census prior to 1910 or after 1930.

was not due to municipal-level efforts to purify the milk or water supplies or treat sewage. It is possible that improvements in the refrigeration chain, better nutrition, or some combination of these and other factors may have contributed to the dramatic reduction in diarrheal deaths among American infants and children during the summer months.

The results described above contribute to a literature comprised entirely of descriptive case studies (Cheney 1984; Condran 1988; Condran and Lentzner 2004). They also have implications for developing countries today, where climate change is expected to increase the risk of diarrheal diseases (Gray 2011; Friedrich 2013; Dhimal et al. 2016).⁴ In many parts of the world, diarrheal diseases still exhibit seasonality, although the intensity of this seasonality varies with region and climate (Patel et al. 2013; Platts-Mills et al. 2015; Xu et al. 2015; Baker 2016). Understanding the determinants of summer diarrhea and its eventual waning could help identify effective intervention strategies as the length and intensity of seasons change across the globe (Kiger 2017; Santer et al. 2018; National Oceanic and Atmospheric Administration 2019).⁵

In addition to providing the first econometric analysis on the relationship between public health interventions and diarrhea seasonality, we explore heterogeneous effects by race. Beyond the work of Troesken (2001, 2002), very little is known about how public health efforts at the turn of the 20th century affected minorities. We show that the reduction in diarrheal mortality among black children was far less dramatic than that experienced by their white counterparts. Even towards the end of the 1920s, black diarrheal mortality exhibited strong seasonality; the diarrheal mortality rate among white children still peaked during the summer months but at less

⁴ In tropical climates (e.g., Southeast Asian), climate change is expected to intensify already marked seasonal variation in rainfall (Christensen et al. 2007; Asian Development Bank 2009; Lyon and Camargo 2009), which could, in turn, exacerbate the incidence of diarrhea.

⁵ An emerging literature has identified existing technologies that can be used to adapt to climate change (Hsiang and Narita 2012; Auffhammer and Schlenker 2014; Klein et al. 2014; Barreca et al. 2016).

than half the rate experienced by black children. Although water filtration led to a reduction in non-summer diarrheal mortality among white children, it and the other public health interventions under study were essentially unrelated to diarrheal mortality among black children.

The remainder of the paper is organized as follows. We begin with an overview of diarrheal disease and a discussion of the previous literature. In Section 3, we document the summer diarrhea phenomenon using data on the 26 most populous cities in the United States as of 1910; in Section 4, we describe our empirical strategy, report our principal estimates, and consider various robustness checks; in Section 5, we explore heterogeneous effects by race. Section 6 concludes.

2. BACKGROUND

Today, diarrheal disease is the second-leading cause of death among children under the age of 5 (Liu et al. 2015). Between 500,000 and 800,000 children under the age of 5 die of diarrhea every year, most of whom are born to mothers in developing countries (Liu et al. 2015; Kovacs et al. 2015). A wide variety of bacteria, parasites and viruses cause diarrhea and other symptoms of gastroenteritis (Hodges and Gill. 2010). Infection is usually through contaminated food or water, or person-to-person contact (Pawlowski et al. 2009). In temperate climates, bacterial infections are more common during the summer (Ramos-Alvarez and Sabin 1958; Fletcher et al. 2013), while viral gastroenteritis is more common during the winter (Pawlowski et al. 2009). In tropical climates, the incidence of diarrhea appears to peak during the rainy season (Zhang et al. 2010; Phung et al. 2015; Xu et al. 2015; Kulinkina et al. 2016; Muluken et al. 2017).

Over 70 percent of total deaths from diarrhea occur among children under of the age of two (Walker et al. 2013). Susceptibility is highest at 6-11 months, presumably because exclusive

breastfeeding protects against infection and crawling brings children into contact with human and/or animal feces (Walker et al. 2013; Mduduzi et al. 2015). Death is typically caused by dehydration and loss of electrolytes (King et al. 2003), although malnutrition is often a contributing factor (Baqui and Ahmed 2006).

2.1. Previous Literature

In the decades leading up to its dissipation, summer diarrhea received a great deal of attention from physicians, who described its symptoms, noted that its victims were often born in crowded tenement housing districts, and proposed various causes.⁶ For instance, one school of thought held that exposure to summer heat was directly responsible for the annual wave of diarrheal deaths among infants and children (Miller 1879; Schereschewsky 1913), while another held that overfeeding was the cause (Burg 1902; Brennemann 1908; Tilden 1909). Even among physicians who believed that summer diarrhea was caused by bacteria, there were several competing theories as to the mode of transmission.⁷

Since its dissipation, only a handful of studies have examined the phenomenon of summer diarrhea. Cheney (1984) focused on the experience of Philadelphia during the period 1869-1921, while Condran (1988) focused on New York City during the period 1870-1919. These authors noted that infant mortality spiked every summer through the early 1900s, due principally to diarrheal diseases. By the second decade of the 20th century, the phenomenon of

⁶ See, for instance, Miller (1879), Burg (1902), Kiefer (1902), McKee (1902), Moss (1903), Southworth (1904), Ostheimer (1905), Snyder (1906), Brennemann (1908), Murphy (1908), Tilden (1909), Stoner (1912), and Youmans and Youmans (1922).

⁷ Zahorsky (1913, 1918) described the various theories of transmission. Hewitt (1910) and Youmans and Youmans (1922) argued that houseflies were the principal vector of transmission, a possibility that cannot be dismissed out of hand (Levine and Levine 1991; Förster et al. 2007).

summer diarrhea had begun to wane in both Philadelphia and New York City, which Cheney (1984) and Condran (1988) attributed to the purification of municipal milk supplies.

Certainly, many contemporary physicians and public health experts were convinced that purifying the milk supply was key to reducing diarrheal mortality during the summer.⁸ The refrigeration chain was still missing important links during this period (Rees 2013), and bacteria such as *E. coli* and *Shigella* grow rapidly in warm, moist summer conditions (Winfield and Groisman 2003; Noor 2013; Girma 2015). It is, however, difficult to rule out the possibility that other public health measures contributed to the observed reduction. For instance, Philadelphia began the process of delivering filtered water to its residents before 1906 and began treating it with chlorine in 1910 (Anderson et al. 2018), both of which could have reduced diarrheal mortality among infants and children during the summer months; New York City began delivering clean water from the Croton Reservoir to its residents in 1907 and began treating it with chlorine in 1911 (Anderson et al. 2018).

Condran and Lentzner (2004) used data from Chicago, New Orleans, and New York for the period 1870-1917 to document the phenomenon of summer diarrhea and its waning. They found that excess mortality during the summer months fell gradually in these cities after the turn of the 20th century, but noted that identifying the cause of this phenomenon is made exceedingly difficult by the large number of public health interventions that were undertaken at the municipal level. These authors concluded that better sanitation and efforts to purify the milk and water supplies likely contributed to the decline in summer diarrhea but wrote that no single factor is “sufficient to understand either the poor life chances of infants in nineteenth-century cities or the improvements in those chances...” (Condran and Lentzner 2004, p. 352).

⁸ See, for instance, Kiefer (1902), McKee (1902), Park and Holt (1904), Snyder (1906), M’Mechan (1907), Stoner (1912), and the United States Children’s Bureau (1914).

3. SUMMER DIARRHEA, 1910-1930

Our focus throughout this study is on diarrheal deaths among children under the age of two in major American cities, defined as the 26 most populous cities as of 1910.⁹ City-level counts of diarrheal deaths are available by month for the period 1910-1930 from *Mortality Statistics*, which was published annually by the U.S. Census Bureau.¹⁰ These counts include deaths due to cholera infantum, colitis, enteritis, enterocolitis, gastroenteritis, summer complaint, and other similar causes (United States Bureau of the Census 1910).

In 1910, there were 21,101 diarrheal deaths among children under the age of two in the 26 most populous American cities (Figure 1), accounting for 30 percent of total mortality in this age group.¹¹ Two-thirds of these deaths occurred in the months of June-September. Between 1910 and 1930, diarrheal mortality fell by 83 percent, to 3,513. The reduction in diarrheal deaths during the months of June-September was even more pronounced: only 1,482 children under the age of two died from diarrhea in the summer of 1930, a reduction of almost 90 percent as compared to the summer of 1910.¹²

Figure 2 shows annual diarrheal deaths among children under the age of two per 100,000 population.¹³ In 1910, there were 124 diarrheal deaths among children under the age of two per

⁹ These 26 cities are listed in Appendix Table 1.

¹⁰ This data source has been used by other authors interested in the effects of public health interventions on mortality at the turn of the 20th century (Cutler and Miller 2005, 2018; Beach et al. 2016; Anderson et al. 2018, 2019, forthcoming; Alsan and Goldin forthcoming). Cause of death was obtained from the death certificate and coded using the *International Classification of Diseases*. When more than one medical condition was listed on the death certificate, cause of death was based on a standardized algorithm (Armstrong et al. 1999).

¹¹ Appendix Figure 1 shows annual diarrheal deaths as a percentage of total deaths among children under the age of two. Appendix Figure 2 shows summer diarrheal deaths as a percentage of total diarrheal deaths.

¹² In 1930, 42.2 percent of diarrheal deaths among children under the age of two occurred in the months of June-September, accounting for only 5 percent of total mortality among children in this age group.

¹³ City populations for 1910, 1920, and 1930 are from *Mortality Statistics*. Population was linearly interpolated for intercensal years.

100,000 population; by 1930, the diarrhea mortality rate had fallen to 14, a reduction of 89 percent. The summer diarrheal mortality rate fell from 82 to 6 over the same period, a reduction of 93 percent.

Figure 3 shows diarrheal deaths by month among children under the age of two per 100,000 population. It is clear from this figure that seasonality waned considerably during the period under study. For instance, the diarrhea mortality rate was 3.8 in January of 1910 but reached as high as 29.1 in July of that same year, a peak-to-trough ratio of 7.7. The peak-to-trough ratio had fallen to 3.5 by 1920, and it had fallen to 2.6 by 1930.¹⁴

3.1. Geography, Temperature, and Summer Diarrhea

Rush (1794, p. 159) wrote that summer diarrhea went by the name of “the April and May disease” in Charlestown “from making its first appearance in those two months...” He went on to observe that it “seldom appears in Philadelphia till the middle of June, or the beginning of July...” (p. 159). Other physicians agreed with Rush, asserting that the phenomenon began earlier, and lasted longer, in the South (King 1837; Copeland 1855; Condie 1858; Smith 1905).

The top panel of Figure 4 shows diarrheal deaths per 100,000 population by month for the first half of the period under study (i.e., 1910-1920) in northern versus southern cities using the 40th parallel north as the cutoff.¹⁵ Consistent with an observation first made by Copeland (1855), the diarrheal mortality rate was lower in southern, as compared to northern, cities.¹⁶

¹⁴ In September of 1930, diarrheal deaths among children under the age of two peaked at 2.0 per 100,000 population in the 26 cities that compose our sample; in December of 1930, there were only 0.8 diarrheal deaths per 100,000 population.

¹⁵ The cities in our sample below the 40th parallel north are Baltimore, Cincinnati, Indianapolis, Kansas City, Los Angeles, Louisville, New Orleans, Philadelphia, San Francisco, St. Louis, and Washington DC.

¹⁶ Copeland (1855, p. 385) observed: “In our Southern cities it is even less rife than in, Philadelphia, Baltimore, and New York, which proves that it is not dependent on heat alone, which some have supposed.” His observation was

Figure 4, however, provides little evidence that the phenomenon of summer diarrhea began earlier, or lasted longer, in cities located below the 40th parallel north, although mortality tended to peak in July as opposed to August.

The bottom panel of Figure 4 shows diarrheal deaths per 100,000 population by month in northern versus southern cities for the second half of the period under study (i.e., 1921-1930). During this later period, the monthly diarrheal mortality rate was, on average, slightly lower in northern cities, but again there is no evidence that the phenomenon of summer diarrhea began earlier or lasted longer in southern cities. However, it should be noted that only one of the cities in our sample (New Orleans, Louisiana) was located in the Deep South. In Figure 5, we show the monthly diarrheal mortality rate for New Orleans. During the period 1910-1920, the diarrheal mortality rate for children under the age of two in New Orleans began to climb in April and peaked in May; it peaked in June during the period 1921-1930.

We explore the relationship between temperature and diarrheal mortality in Figure 6. As discussed above, bacteria such as *E. coli* and *Shigella* grow rapidly in the heat of summer (Winfield and Groisman 2003; Noor 2013; Girma 2015), while viral infections that cause diarrhea typically peak in winter months (Pawlowski et al. 2009). It is not clear, however, whether such seasonality is due to temperature, humidity, changes in behavior, or host susceptibility (Ahmed et al. 2013).¹⁷

repeated by Rogers (1869) and Maxwell (1887). For instance, Maxwell (1887, p. 538), who practiced medicine in Ocala, Florida, wrote that the town had:

...about 2500 inhabitants and...it is entirely exempt from cholera infantum. During the last six summers I have resided and practiced medicine there, and have not seen or heard of a case of that disease.

¹⁷ According to a recent review, noroviruses “are the most common cause of sporadic cases and outbreaks of gastroenteritis across all age groups worldwide” (Ahmed et al. 2013, p. 1). Rotavirus infections are another important cause of gastroenteritis among children under the age of 5 (Patel et al. 2013). Hospitalizations and outpatient visits for rotavirus and norovirus infections typically peak in the winter months (D’Souza, Hall and

Figure 6 shows diarrheal deaths among children under the age of two per 100,000 population for the following city-month combinations: those with an average temperature less than 60° F, those with an average temperature between 60-70° F, and those with an average temperature equal to or greater than 70° F. Several patterns are evident in Figure 6.¹⁸ First, throughout the period under study, the diarrheal mortality rate was highest when the average temperature was at or above 70° F and lowest when the average temperature was below 60° F.¹⁹ Second, diarrheal mortality rates exhibited steady declines across all three of the temperature bands. Lastly, these declines were most pronounced at higher temperatures. For example, diarrheal deaths at or above 70° F fell by 91 percent, from 21.5 per 100,000 population in 1910 to 2.0 in 1930. By contrast, the diarrheal mortality rate at average temperatures less than 60° F fell by 77 percent, from 4.4 in 1910 to 1.0 in 1930.

In the next section, we explore whether the waning of summer diarrhea, documented in Figures 1-6, was related to public health interventions undertaken at the municipal level. A description of these interventions can be found in Anderson et al. (2018), who were interested in

Becker 2008; Gastañaduy et al. 2013; Patel et al. 2013). Parasitic infections, which can also cause diarrhea, are more common in the summer months (Amin 2002).

¹⁸ Data on temperature come from the nClimDiv data set at the National Climatic Data Center (NCDC) of the National Oceanic and Atmospheric Administration (NOAA), which is available at: <ftp://ftp.ncdc.noaa.gov/pub/data/cirs/climdiv/>. Monthly temperature variables are measured at the climate division level (there are 344 climate divisions covering the entire continental United States). We mapped each city in our sample to its corresponding climate division using ArcGIS. Please see the following document for more details on the construction of the nClimDiv data set: <ftp://ftp.ncdc.noaa.gov/pub/data/cirs/climdiv/divisional-readme.txt>.

¹⁹ This pattern is consistent with studies showing a strong, positive relationship between temperature and infant mortality in developing countries (Banerjee and Bhowmick 2016; Geruso and Spears 2018). See Barreca et al. (2016) for evidence that the positive relationship between temperature and mortality weakened during the 20th century. Barreca et al. (2016, p. 156) concluded that air conditioning was:

...a central determinant in the reduction of the mortality risk associated with high temperatures during the twentieth century. Specifically, the diffusion of residential air conditioning after 1960 is related to a statistically significant and economically meaningful reduction in the temperature-mortality relationship at high temperatures.

whether public health interventions could explain the declines in total and infant mortality over the first decades of the 20th century.²⁰ Anderson et al. (2018) found that filtration was associated with a (statistically insignificant) 15 percent reduction in diarrheal deaths among children under the age of two, an estimate that falls well short of explaining the dramatic reduction in diarrheal mortality during the early 1900s. Because these authors used annual mortality data, they could not examine the determinants of seasonality.

4. PUBLIC HEALTH EFFORTS AND THE WANING OF SUMMER DIARRHEA

Economists and historians have long asserted that public health efforts undertaken at the municipal level caused the waning of summer diarrhea (Cheney 1984; Condran 1988; Meckel 1990; Fishback et al. 2011). For instance, Cheney (1984) and Condran (1988) argued that efforts to purify milk supplies caused its waning, while Meckel (1990) and Fishback et al. (2011) pointed to the combined effects of purifying milk and water supplies.²¹

²⁰ Using data on 25 major American cities for the period 1900-1940, Anderson et al. (2018) explored the effects of filtration and chlorination on infant and total mortality. In addition, they explored the effects of sewage treatment, projects designed to deliver clean water from further afield such as aqueducts and cribs, and municipal efforts to clean up milk supplies. Contrary to the consensus view, Anderson et al. (2018) found that none of the interventions under study contributed substantially to the observed declines in mortality.

²¹ Fishback et al. (2011, p. 140) noted that as “pasteurized milk became more common and cities filtered public water supplies, the rates of typhoid and diarrhea no longer varied much by season or in response to temperature.” Meckel (1990, p. 89) was considerably more cautious. He wrote:

It is extremely difficult to assess with any certainty the effect that milk regulation and especially commercial pasteurization had on the urban infant death rate. Infant mortality is causally complex, and its reduction is usually tied to an amalgam of changes in the social and material environment. The implementation of milk regulations and the introduction of pasteurization came at a time when many municipalities were also making significant strides in improving their water supply, sewerage, and refuse removal systems...Nevertheless, infant mortality, and especially mortality from diarrheal diseases did decline concurrent with the implementation of effective milk regulations and the adoption of commercial pasteurization in America's larger cities. As one demographer has recently demonstrated, summer mortality among infants, which declined slowly between 1890 and 1910, dropped rapidly in the second decade of the century and by 1921 was all but negligible.

The remainder of this study is devoted to exploring the relationship between public health interventions at the municipal level and the phenomenon of summer diarrhea. We begin our exploration of this relationship by estimating the following regression:

$$(1) \quad \ln(Diarrhea_{ct}) = \beta_0 + \beta_1 Filtration_{ct} + \beta_2 Filtration_{ct} \times Summer_t + \beta_3 Chlorination_{ct} + \beta_4 Chlorination_{ct} \times Summer_t + \mathbf{X}_{ct}\boldsymbol{\beta}_5 + \nu_c + w_t + \Theta_c \cdot t + \varepsilon_{ct},$$

where *Diarrhea* is equal to diarrheal deaths per 100,000 population among children under the age of two in city *c* and month *t*, where $t = 1 \dots 252$.²² *Filtration* is an indicator for whether a water filtration plant was in operation and *Chlorination* is an indicator for whether the water supply was chemically treated.²³ These indicators are interacted with *Summer*, which is equal to 1 for the months of June-September and equal to zero for the non-summer months.

Demographic controls, based on information from the 1910, 1920, and 1930 Censuses (and linearly interpolated for intercensal months), are represented by the vector \mathbf{X}_{ct} and are listed in Table 1, along with descriptive statistics and definitions. City-level characteristics include the natural log of population and percentages of the population by gender, race, foreign-born status, and age group. City and month-by-year fixed effects are represented by the terms ν_c and w_t , respectively. The city fixed effects control for determinants of diarrheal mortality that were constant over time, and the month-by-year fixed effects control for common shocks. City-

²² Our data cover the period 1910-1930 ($12 \times 21 = 252$).

²³ Water filtration technology was originally developed to reduce discoloration and turbidity, but gained support as the study of bacteriology advanced and city governments came under increasing pressure to protect their citizens from infectious diseases (McCarthy 1987; Melosi 2008). Unlike water filtration, the chlorination process was simple and inexpensive: water was added to calcium hypochlorite, which was then mixed with the water supply before delivery (Hooker 1913). See Melosi (2008) and Anderson et al. (2018) for more information on the history and technical aspects of these two interventions.

specific linear time trends, represented by the term $\Theta_c \cdot t$, account for the possibility that diarrheal mortality evolved at different rates in the 26 cities that compose our sample. All regressions are weighted by city populations and standard errors are corrected for clustering at the city level (Bertrand et al. 2004).

Our primary interest is in the parameters β_1 through β_4 . β_1 represents the effect of filtration on diarrheal mortality in the non-summer months, while $\beta_1 + \beta_2$ represents the effect of filtration in the months of June-September; likewise, β_3 represents the effect of chlorination on diarrheal mortality in the non-summer months, while $\beta_3 + \beta_4$ represents the effect of chlorination in the months of June-September. During the period 1910-1930, 8 cities adopted filtration technology, and 24 cities began treating their water with chlorine (Appendix Table 1).

Estimates of β_1 through β_4 are reported in the first column of Table 2. Filtration is associated with a 16 percent reduction in the diarrheal mortality rate during the non-summer months ($e^{-.176} - 1 = .161$), which is consistent with the hypothesis that transmission in the non-summer months occurred through contaminated water. By contrast, we find little evidence that filtration was effective in the months of June-September: the estimate of β_2 is positive and almost entirely offsets the estimate of β_1 .²⁴ Neither the estimate of β_3 nor the estimate of β_4 is statistically distinguishable from zero.

In the second column of Table 2, we introduce two additional municipal-level public health interventions and their interactions with the summer indicator. The first, *Clean Water Project*, is equal to 1 if a new aqueduct or underground tunnel was built to deliver clean water (and is equal to zero otherwise).²⁵ The second, *Sewage Treated*, is an indicator for whether the

²⁴ The sum of these two estimates, -.016, is not statistically significant at conventional levels.

²⁵ Identification comes from water projects undertaken by 4 cities during the period under study (Buffalo, Los Angeles, Providence, and Newark). See Appendix Table 2 for more information.

city treated its sewage before discharging it into local waterways.²⁶ With their inclusion as controls, water filtration is associated with a 17 percent reduction in the diarrheal mortality rate during the non-summer months. However, the estimated coefficients of *Clean Water Project*, *Sewage Treated*, and their interactions are not statistically significant at conventional levels.

In the third and final column of Table 2, we introduce an indicator for whether city *c* required milk sold within its limits to meet a strict bacteriological standard. This indicator also appears on the right-hand side of the estimating equation interacted with *Summer*. During the period 1910-1930, 15 cities passed ordinances requiring that milk sold within their limits meet a bacteriological standard (see Appendix Table 4). Because such ordinances were difficult to meet without resorting to pasteurization (Meckel 1990, pp. 88-89), they are often referred to as “pasteurization ordinances” (Harding 1917, p. 57; Swinford 2016, p. 254; Komisarow 2017, p. 131). For instance, New York passed an ordinance, effective on January 1, 1912, requiring that “certified” raw milk have less than 30,000 bacteria per cubic centimeter, and that “inspected” raw milk have less than 60,000 bacteria per cubic centimeter.²⁷

²⁶ Identification comes from sewage treatment plants built by 8 cities during the period under study (Baltimore, Chicago, Cleveland, Indianapolis, Jersey City, Milwaukee, Newark, and Rochester). Following Anderson et al. (2018), we code our sewage treatment indicator as equal to zero for cities that were treating less than 25 percent of their effluent (Mohlman 1940). See Appendix Table 3 for more information. With the exception of Anderson et al. (2018), previous studies have focused on estimating the effects of providing sewerage (i.e., the building and extending of sewer systems) as distinct from treating sewage (i.e., using chemical or biological processes to remove contaminants from waste water) before its discharge. See, for instance, Kesztenbaum and Rosenthal (2017) and Alsan and Goldin (forthcoming).

²⁷ Pasteurized “selected milk” was required to have less than 50,000 bacteria per cubic centimeter (New York 1912; New York Department of Health 1913). Other cities 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. During the period 1910-1930, only two cities in our sample (Detroit and Chicago) 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).

Anderson et al. (2018) found little evidence that setting a bacteriological standard for milk reduced infant mortality. Our results are consistent with theirs. Specifically, the estimated coefficient of the bacteriological standard indicator is actually positive and statistically significant at the .10 level. By contrast, the estimated coefficient of the interaction between *Bacteriological Standard* and the summer indicator is negative and larger in absolute magnitude, but it is not sufficiently precise to reject the null.²⁸

4.1. Robustness Checks

Rapid industrialization during the period under study led to substantial increases in purchasing power (Bry 1960; Rees and Jacobs 1961). In the first column of Table 3, we explore whether the filtration estimates described above are robust to controlling for the real manufacturing wage, which can be thought of as measuring the purchasing power of urban workers.²⁹ The estimated coefficient of the filtration indicator retains its magnitude and significance (specifically, water filtration is associated with a 14 percent reduction in diarrheal mortality during the non-summer months), suggesting that the negative relationship between filtration and diarrheal mortality in the non-summer months cannot be explained by changes in the purchasing power of urban workers.

In addition to including the manufacturing wage on the right-hand side of equation (1), we experiment with controlling for region-by-year fixed effects, not weighting by population,

²⁸ In Appendix Table 5, we replace the *Summer* indicator with a variable equal to 1 if the average temperature in city c during month t was equal to or greater than 70° F. The results from this exercise are qualitatively similar to those reported in Table 2.

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).

dropping the most populous city (i.e., New York City) from the sample, dropping the years 1917-1920 to avoid potential confounding from the effects of the 1918 Spanish flu pandemic, and specifying the dependent variable in levels. The results of these robustness checks, which are shown in the remaining columns of Table 3, suggest that the negative relationship between water filtration and diarrheal mortality in the non-summer months is not an artifact of specification or sample choice.

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 are reported in the first two columns of Table 4. They suggest that the effect of water filtration on diarrheal deaths occurring during non-summer months was immediate. Specifically, filtration is associated with a 15 percent decrease in diarrheal mortality in the year of implementation (*Year 0*); after 5 or more years, it is associated with a 19 percent decrease. Consistent with the results discussed above, there is little evidence that filtration affected diarrheal deaths in the months of June-September.

In the last two columns of Table 4, we replace the filtration indicator with a series of its leads and lags. The lagged effects are similar to those described above. In *Year 0*, filtration is associated with a 15 percent decrease in non-summer diarrheal mortality; after 5 or more years, filtration is associated with a 20 percent decrease in non-summer diarrheal mortality. Again, there is no evidence that filtration had an impact on diarrheal deaths in the months of June-September. Consistent with the parallel-trends assumption, the coefficient estimates on the leads of the filtration indicator are not statistically distinguishable from zero. Figures 7 and 8 reproduce the results reported in columns (3) and (4) of Table 4, respectively.³⁰

³⁰ There is reason to believe that the effect of opening a water filtration plant might, at least initially, be muted. For instance, the Cleveland filtration plant mixed its output with “raw” (i.e., unfiltered) water before delivery during its first 6 months of operation (Unknown Author 1918). The effort to provide filtered water to every neighborhood in Philadelphia took more than seven years (Cutler and Miller 2018). Cutler and Miller (2018) argued that the

5. RACE AND SUMMER DIARRHEA

Public health experts at the turn of the 20th century recognized that black infants were more likely to die from diarrheal diseases, including “cholera infantum,” than their white counterparts (Billings 1883; Du Bois 1899; Miller 1906). The reasons for this discrepancy, however, were fervently debated. For instance, in an infamous treatise published by the American Economic Association, Hoffman (1896) argued that “excessive infant mortality among the colored population is largely the result of individual neglect, as well as in part due to inherited organic weakness...” (p. 69). Hoffman (1896) went on to recommend that philanthropic efforts not be directed toward reducing the black infant mortality rate for fear that such efforts would make blacks “even more dependent on the white race at the present time than...previous to emancipation” (p. 329). Other observers argued that the discrepancy was due to “matters of condition” as opposed to “racial traits and tendencies” (Miller 1906, p. 90). Citing Du Bois (1899), Miller went on to list the conditions that explained why black children in Philadelphia were not as healthy as white children. Among them were a lack of education, the fact that “66 per cent of Philadelphia Negro woman work”, and bad water (Miller 1906, p. 90).

With only a few exceptions, historical studies on the provision of clean water and mortality have not provided estimates by race. Among these exceptions, Troesken (2001) found that switching from privately to publicly owned water companies narrowed the black-white waterborne-disease mortality gap in 14 North Carolina towns during the period 1889-1908.³¹

Philadelphia water filtration indicator should “turn on” in 1909, the year in which the largest filtration facility (Torresdale) began providing the majority of Philadelphia residents with clean drinking water. The pre-treatment trends in Figures 7 and 8 looked similar when we experimented with turning on the Philadelphia filtration indicator in 1909 as opposed to 1906.

³¹ Troesken’s (2001) outcome of interest was the sum of mortality rates for the following waterborne diseases: cholera, diarrhea, dysentery, and typhoid.

Analyzing a sample of 33 U.S. cities at the turn of the 20th century, Troesken (2002) found that municipal water filtration was associated with a more than 50 percent reduction in typhoid mortality among blacks. However, it is worth noting that typhoid deaths represented only a small proportion of total mortality and never amounted to more than half of mortality from diarrhea/enteritis. In 1914, among the black population in the United States, there were 35.9 typhoid deaths per 100,000 population and 109 diarrhea/enteritis deaths per 100,000 population; in 1920, the black typhoid and diarrheal mortality rates were 18.8 and 68.1, respectively; in 1930, the black typhoid and diarrheal mortality rates were 13.7 and 43.7, respectively.³²

Figure 9 shows annual diarrheal mortality rates among children under the age of two by race for the years 1910-1930.³³ During this period, diarrheal mortality data were available by race for 19 of the 26 cities under study.³⁴ The black diarrheal mortality rate in these cities was clearly on a downward trend, but it was always considerably higher than the corresponding white rate. For instance, 131.9 black children under the age of two died of diarrhea per 100,000 population in 1910, while 88.0 white children died of diarrhea that same year. By 1930, the last year for which we have mortality counts at the monthly level, 30.4 black children under the age of two died of diarrhea per 100,000 population as compared to 10.2 white children.

³² These rates are based on the overall “registration area” in the United States, come from *Mortality Statistics*, and were first published in 1914. The black diarrheal mortality rate among children under the age of two in 1914, 1920, and 1930 was 87.8, 54.2, and 33.7, respectively.

³³ Municipal-level mortality counts by race (white versus nonwhite) are available from *Mortality Statistics*. Although not strictly correct, we refer to “nonwhites” as “blacks.” During this period, over 95 percent of the minority population in the United States was black (Gibson and Jung 2002). Population estimates by race for the cities in our sample and the years 1910, 1920, and 1930 are also available from *Mortality Statistics*. Population was linearly interpolated to produce diarrheal mortality rates by race at the city-month-year level.

³⁴ Diarrheal mortality data by race are unavailable for Buffalo, Jersey City, Milwaukee, Minneapolis, Providence, Rochester, and St. Paul for the entire period under study. Diarrheal mortality data by race are unavailable for 1911 regardless of the city under study. Appendix Table 6 lists the years of data available for each of the cities used in the analysis.

Figure 10 shows black versus white diarrheal deaths per 100,000 population by month for the period 1910-1930. During the non-summer months of the first decade under study (1910-1920), black children were more likely to die of diarrhea than their white counterparts, but the difference in rates was not substantial.³⁵ In the summer months (June-September), diarrheal deaths among black children were much more prevalent than among white children. For instance, the black diarrheal mortality rate peaked at 12.3 per 100,000 population in 1919, while the white diarrheal mortality rate peaked at 6.9 that same year.

During the second decade under study (1921-1930), there was marked decline in diarrheal mortality among white children, which took place throughout the year regardless of the season. There was also a decline in black diarrheal mortality, but it was not nearly as dramatic. Moreover, even towards the end of the period under study, black diarrheal mortality clearly exhibited seasonality.

In Table 5, we explore the relationship between public health interventions and summer diarrhea by race. Specifically, we estimate equation (1) for white and black children separately. Given the results reported in Tables 2 and 3, our focus will be on the parameters β_1 and β_2 , where β_1 represents the effect of water filtration on diarrheal mortality in the non-summer months and $\beta_1 + \beta_2$ represents its effect in the months of June-September.

Water filtration is negatively related to non-summer diarrheal mortality among white children. Specifically, filtration led to a 9.4 percent reduction in the white diarrheal mortality rate during the non-summer months ($e^{-.099} - 1 = -.094$), although the estimate of β_2 more than offsets the estimate of β_1 . Among black children, neither the estimate of β_1 nor the estimate of β_2

³⁵ Appendix Figure 3 shows that the black-white diarrheal mortality gap clearly spiked during the summer months, sometimes exceeding 5 deaths per 100,000 population. In the non-summer months, however, the annual mean difference between black and white diarrheal mortality was usually 1 to 2 deaths per 100,000 population (Appendix Figure 4).

is statistically distinguishable from zero. Moreover, both of these estimates are actually positive. Consistent with the results first reported in Table 2, we find little evidence that the other public health interventions under study reduced diarrheal mortality among children, black or white.

6. CONCLUSION

Although progress has been made over the last few decades, diarrheal disease is still the second-leading cause of death among children under the age of 5 (Liu et al. 2015), and it continues to exhibit pronounced seasonality in many parts of the developing world (Zhang et al. 2010; Phung et al. 2015; Xu et al. 2015; Kulinkina et al. 2016; Muluken et al. 2017). Because this seasonality is expected to intensify with climate change (Christensen et al. 2007; Asian Development Bank 2009; Lyon and Camargo 2009), understanding its causes is taking on a new urgency.

In the United States at the turn of the 20th century, tens of thousands of American children would die from diarrhea-related disease every summer. One of the principal contributions of this study is to simply document the scale of this phenomenon. Using newly transcribed diarrheal mortality data at the monthly level for the 26 most populous American cities as of 1910, we find that summer diarrhea was a major contributor to child mortality. For instance, in 1910 there were over 21,000 diarrheal deaths among children under the age of two in these cities, accounting for 30 percent of total mortality in this age group; two-thirds of diarrheal deaths among these children occurred in the months of June-September.

The phenomenon of summer diarrhea had largely dissipated by 1930. In that year, only 3,513 children under the age of two died from diarrhea in the 26 cities under study, and only

1,482 of these deaths occurred in the summer months. The precise cause of summer diarrhea was never isolated and the memory of its toll eventually receded.

Economists and historians generally believe that the dissipation of summer diarrhea was due to public health efforts undertaken at the municipal level (Cheney 1984; Condran 1987; Meckel 1990; Fishback et al. 2011). Evidence for this belief, however, is anecdotal or based on a handful of case studies (Cheney 1984; Condran 1987; Condran and Lentzner. 2004). In addition to documenting the phenomenon of summer diarrhea, we explore whether its waning over the period 1910-1930 was, in fact, related to public health interventions undertaken at the municipal level. We find that the building of a water filtration plant is associated with a 15-17 percent reduction in diarrheal mortality during non-summer months. By contrast, there is no evidence that water filtration led to a reduction in diarrheal mortality during the months of June-September, nor is there evidence that other municipal-level public health efforts (including sewage treatment plants and setting strict bacteriological standards for milk) resulted in the dissipation of summer diarrhea. This pattern of results is consistent with those of Anderson et al. (forthcoming) and Anderson et al. (2018), who concluded that public health efforts at the municipal level were not important drivers of the urban mortality transition.

Finally, because the U.S. Bureau of the Census published diarrheal mortality counts by race, we are able to document that the decline in diarrheal mortality among black children was much less dramatic than that experienced by their white counterparts. Even at the end of the period under study, black diarrheal mortality still exhibited strong seasonality and generally peaked at more than double the white rate. While we show that the adoption of water filtration technology reduced non-summer diarrheal mortality among white children, it seems to have had no effect on diarrheal mortality among black children, regardless of the season.

Our results suggest that public health interventions at the municipal level did not contribute to the dissipation of summer diarrhea in the United States, nor did they contribute to the pronounced decline in diarrheal mortality among black children. Perhaps other, more difficult-to-measure factors, such as nutrition, improvements in medical care, the adoption of more hygienic practices, or better living conditions were responsible for the waning of summer diarrhea.

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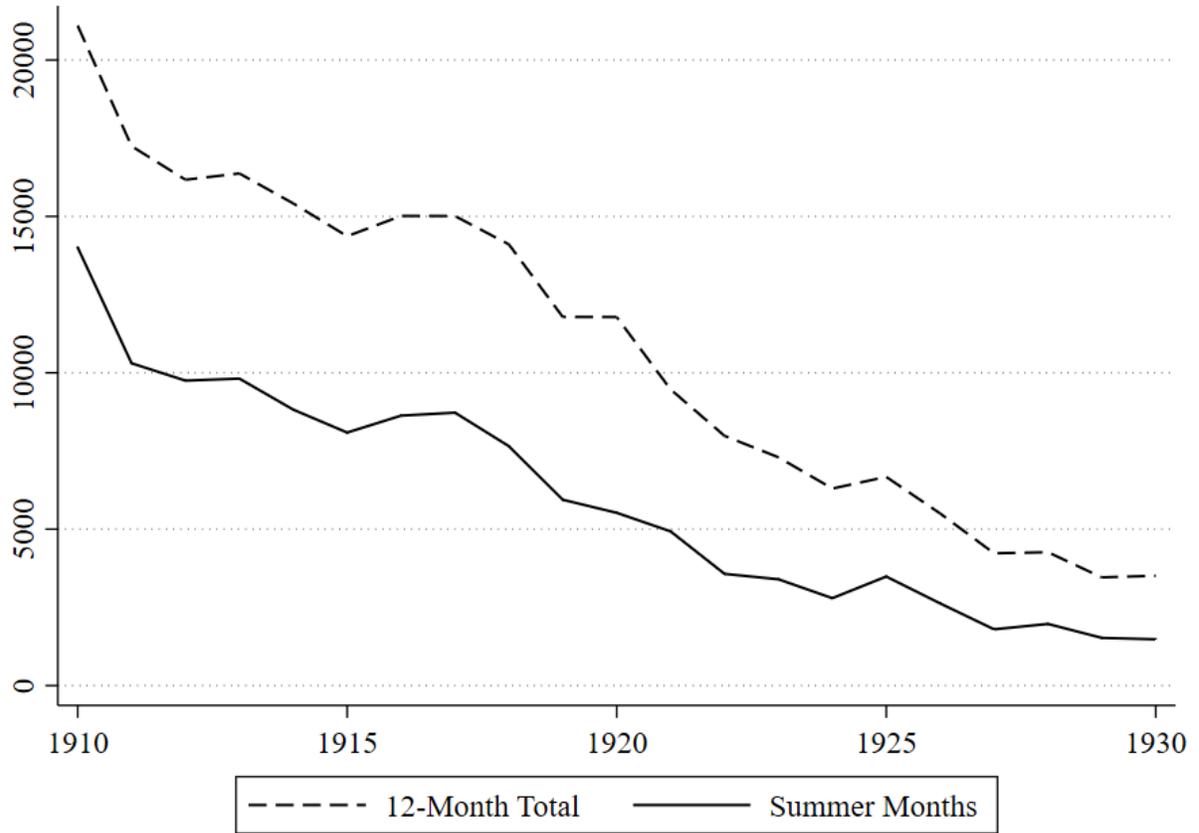
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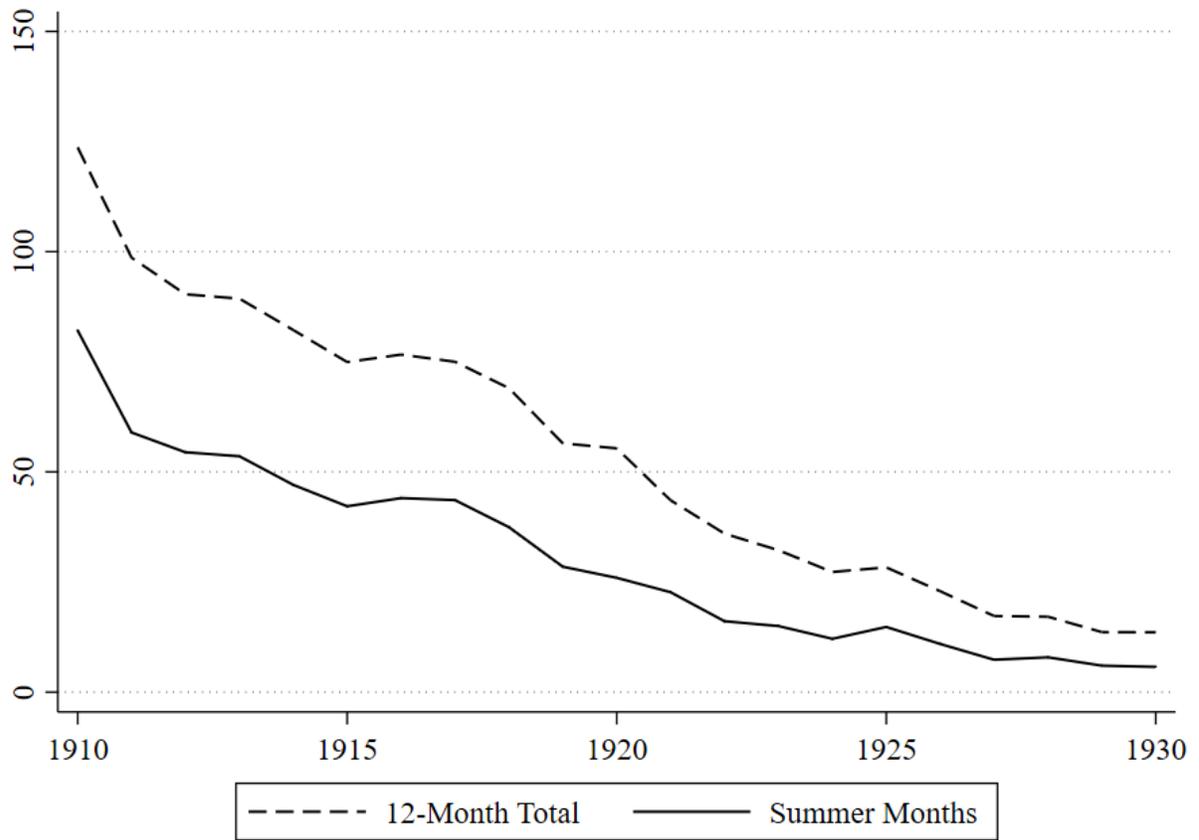
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Figure 1. Diarrheal Mortality Among Children Under the Age of Two



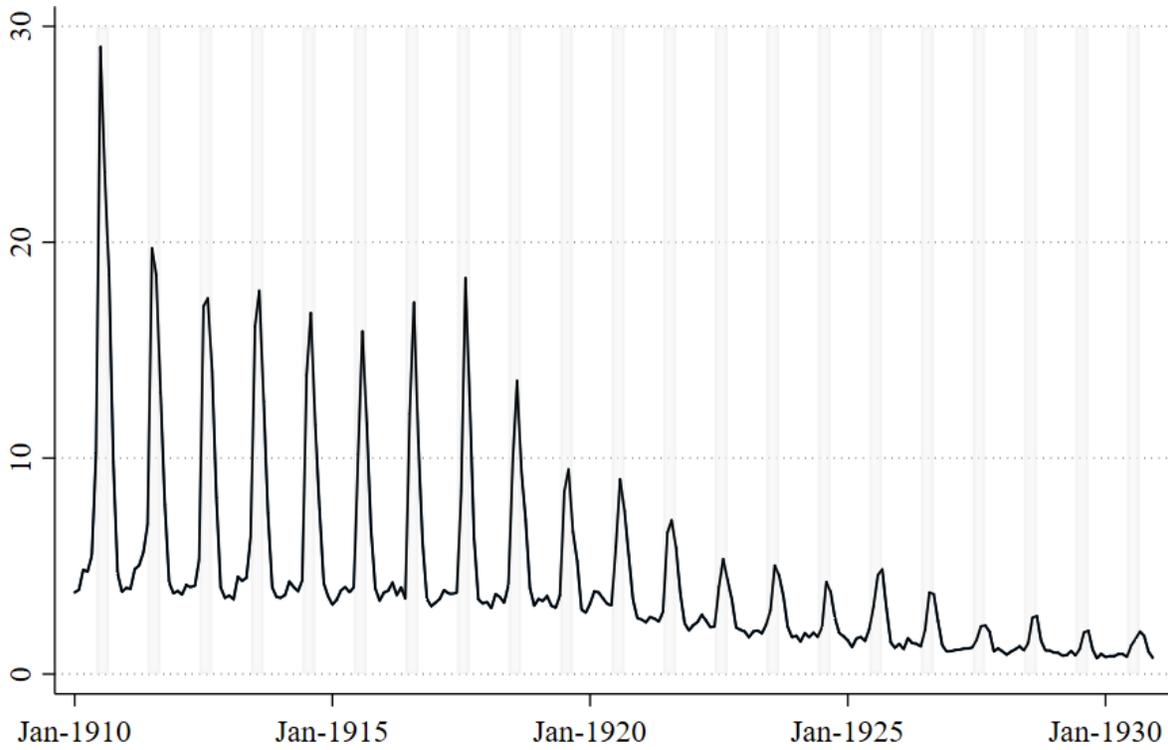
Notes: Based on data from *Mortality Statistics* for the 26 cities under study, published by the U.S. Census Bureau. The summer months are defined as June through September.

Figure 2. Diarrheal Mortality Among Children Under the Age of Two per 100,000 Population



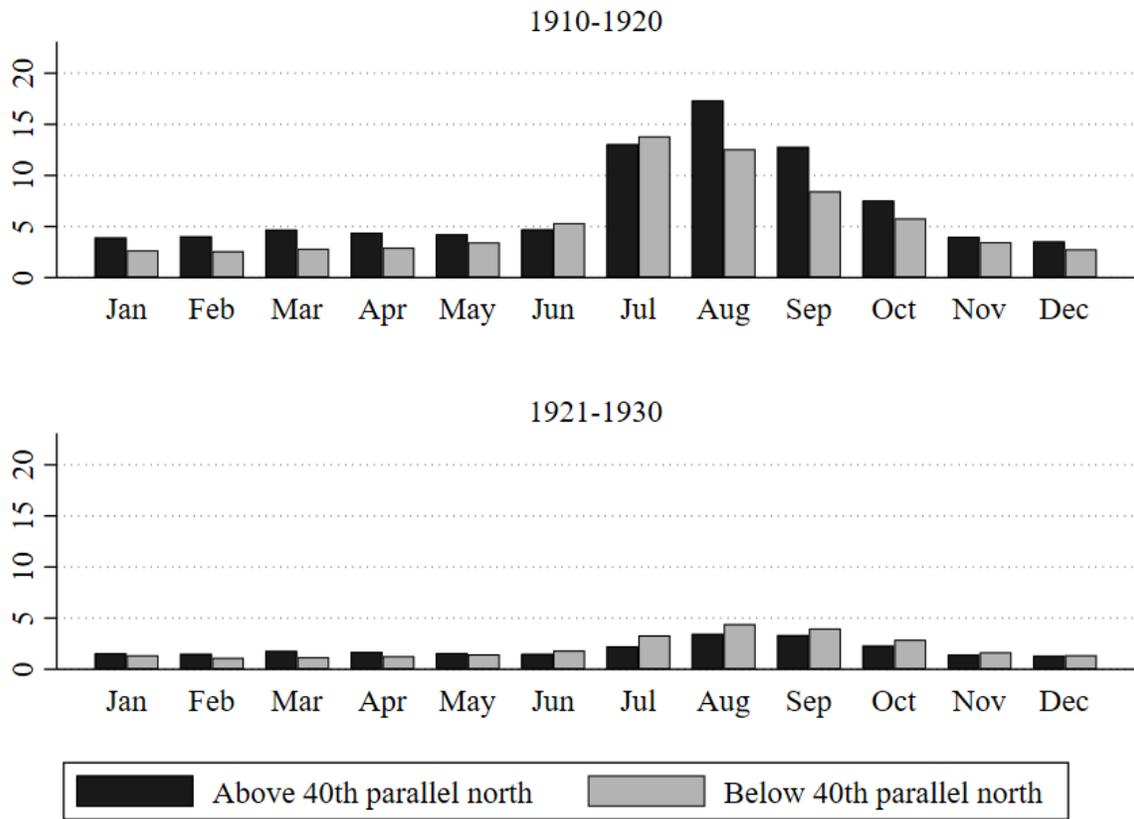
Notes: Based on data from *Mortality Statistics* for the 26 cities under study, published by the U.S. Census Bureau. The summer months are defined as June through September.

Figure 3. Monthly Diarrheal Mortality Among Children Under the Age of Two per 100,000 Population



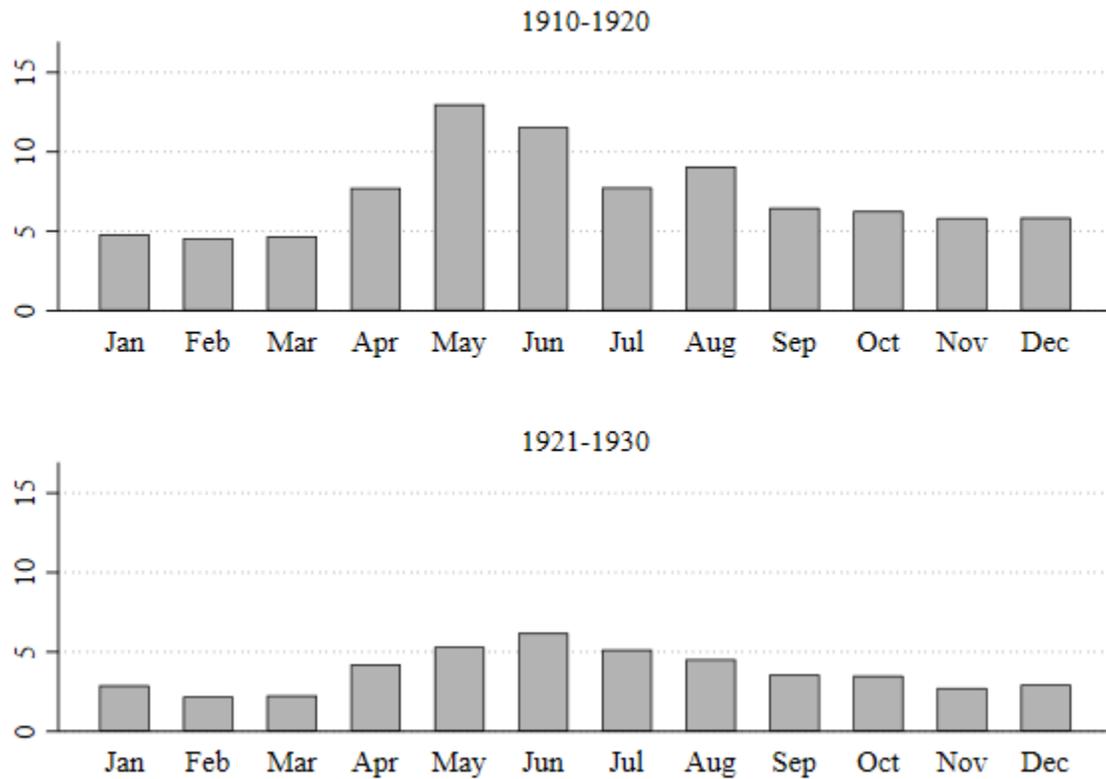
Notes: Based on data from *Mortality Statistics* for the 26 cities under study, published by the U.S. Census Bureau. The shaded vertical bars indicate the summer months (June-September).

Figure 4. Monthly Diarrheal Mortality Among Children Under the Age of Two per 100,000 Population, Above and Below 40th Parallel North



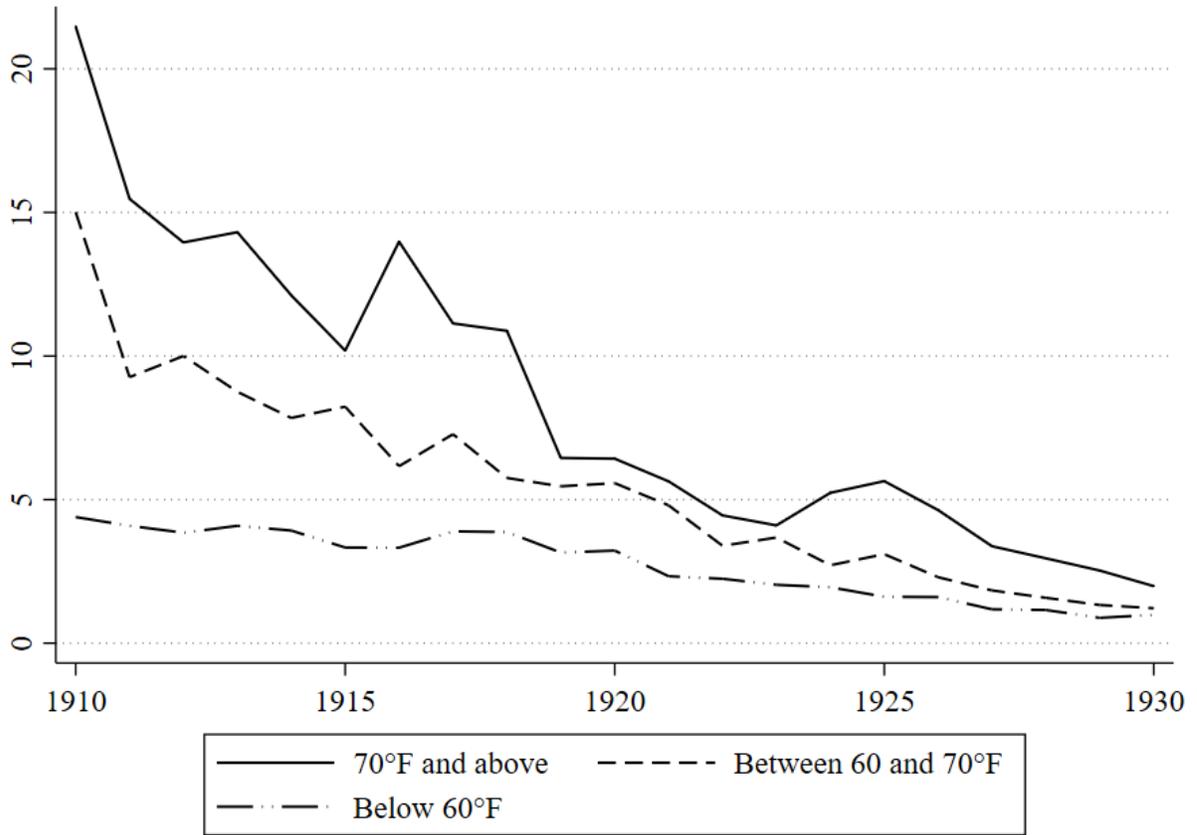
Notes: Based on data from *Mortality Statistics* for the 26 cities under study, published by the U.S. Census Bureau.

Figure 5. Monthly Diarrheal Mortality Among Children Under the Age of Two per 100,000 Population in New Orleans



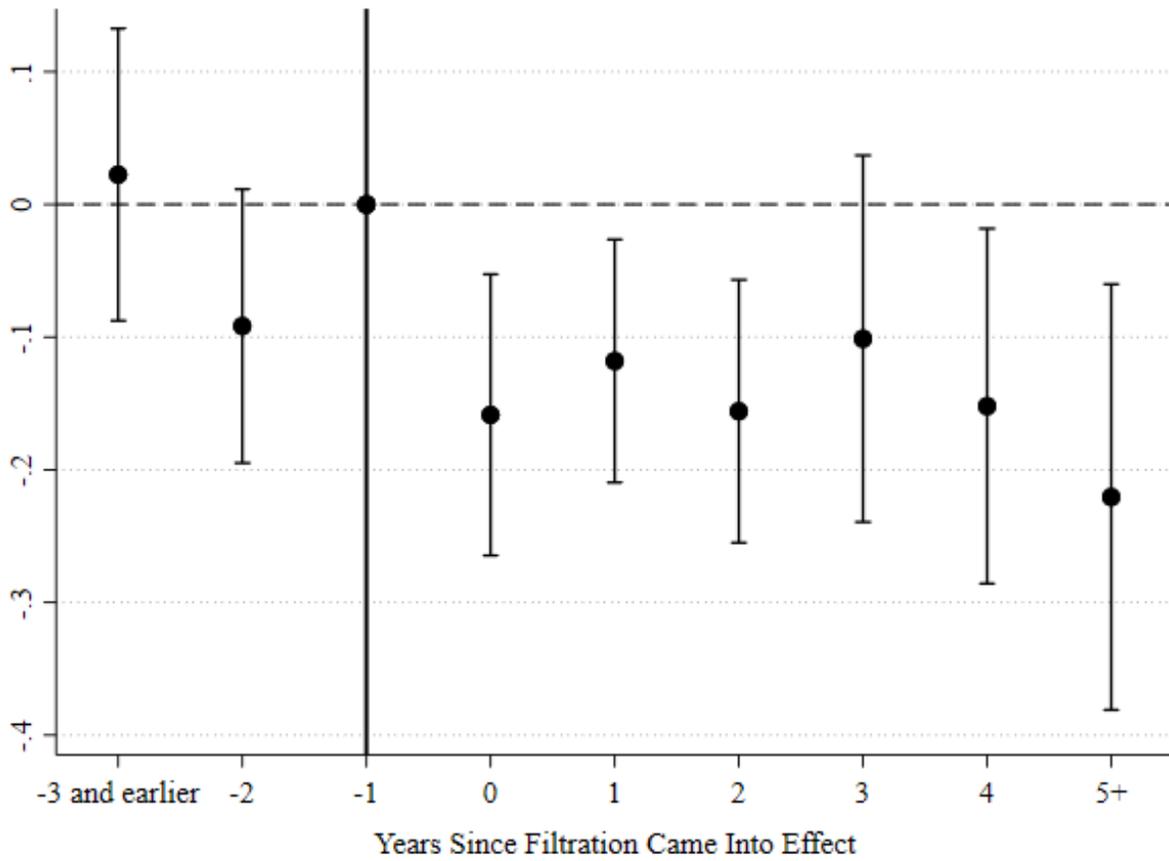
Notes: Based on data from *Mortality Statistics* for New Orleans, Louisiana, published by the U.S. Census Bureau.

Figure 6. Diarrheal Mortality Among Children Under the Age of Two per 100,000 Population by Average Temperature



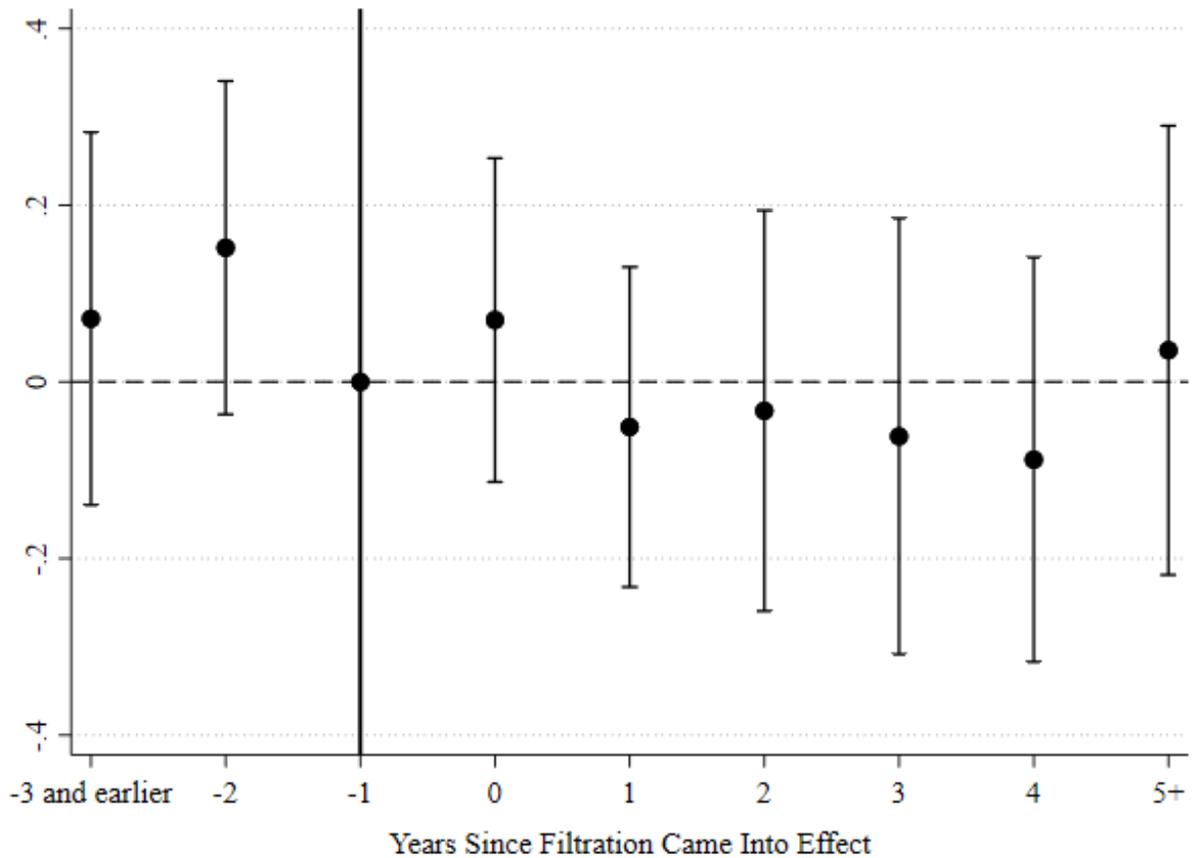
Notes: Based on data from *Mortality Statistics* for the 26 cities under study, published by the U.S. Census Bureau.

Figure 7. Pre- and Post-Filtration Trends in Diarrheal Mortality During Non-Summer Months



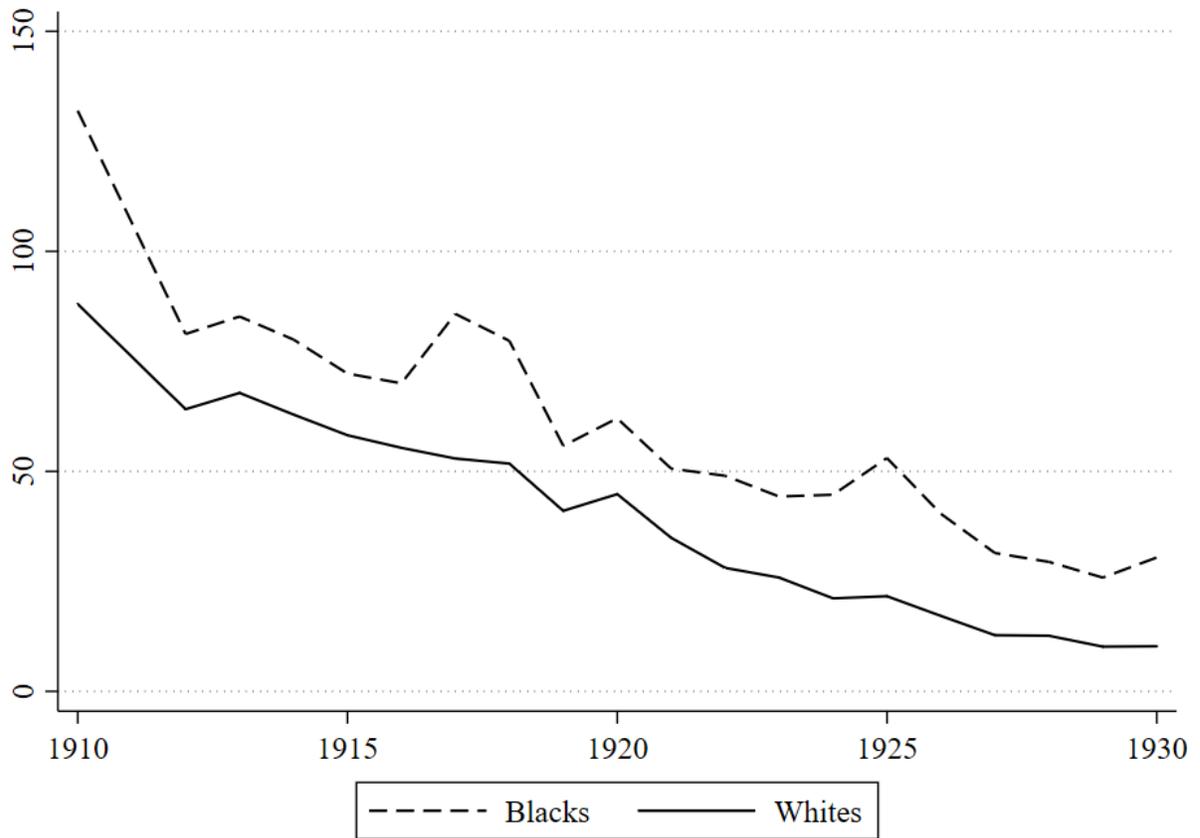
Notes: OLS coefficient estimates (and their 90% confidence intervals) are reported, where the omitted category is one year before treatment. The dependent variable is equal to the natural log of the number of diarrheal deaths among children under the age of two per 100,000 population in city c and month t . Controls include the city characteristics listed in Table 1, city fixed effects, month-by-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 8. Pre- and Post-Filtration Trends in Diarrheal Mortality During Summer Months



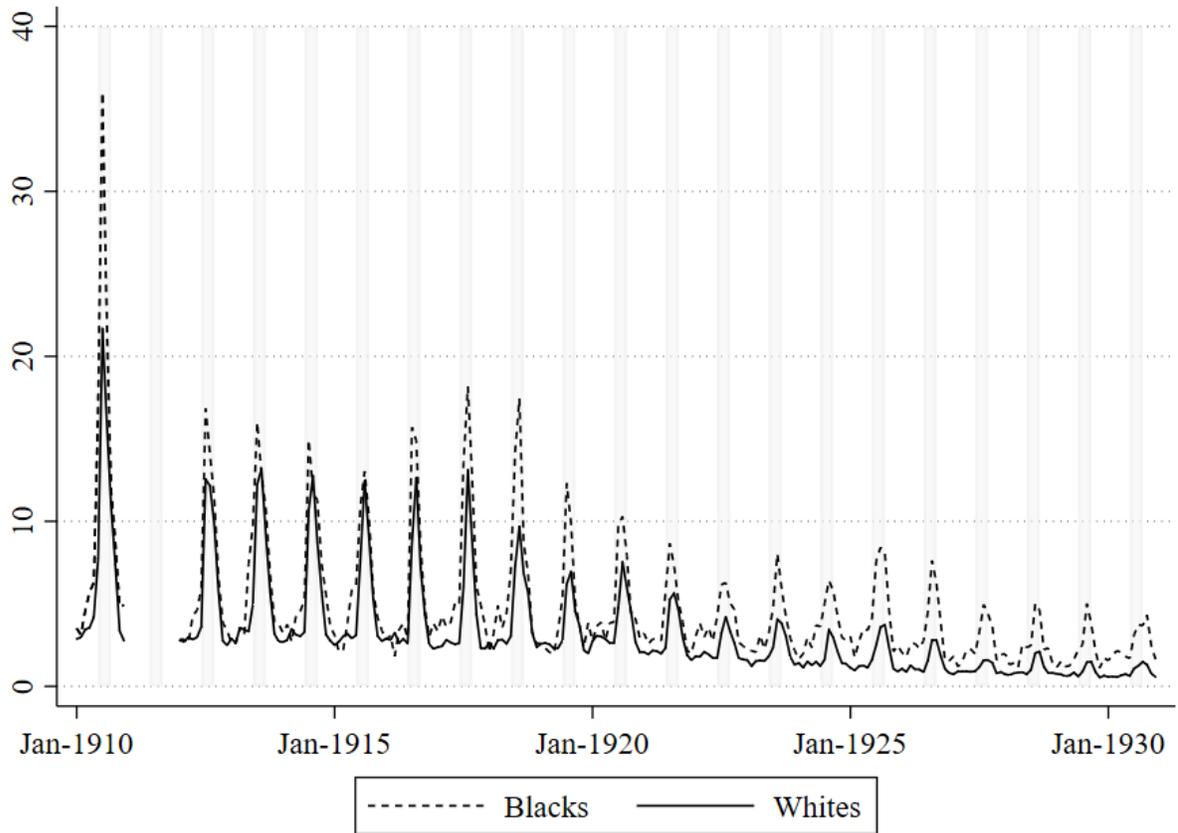
Notes: OLS coefficient estimates (and their 90% confidence intervals) are reported, where the omitted category is one year before treatment. The dependent variable is equal to the natural log of the number of diarrheal deaths among children under the age of two per 100,000 population in city c and month t . Controls include the city characteristics listed in Table 1, city fixed effects, month-by-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 9. Diarrheal Mortality Among Children Under the Age of Two per 100,000 Population by Race



Notes: Based on data from *Mortality Statistics* for the 19 cities under study, published by the U.S. Census Bureau. Diarrheal mortality counts by race are unavailable for 1911.

Figure 10. Monthly Diarrheal Mortality Among Children Under the Age of Two per 100,000 Population by Race



Notes: Based on data from *Mortality Statistics* for the 19 cities under study, published by the U.S. Census Bureau. The shaded vertical bars indicate the summer months (June-September). Diarrheal mortality counts by race are unavailable for 1911.

Table 1. Descriptive Statistics

	Mean (SD)	Description
<i>Diarrhea</i>	4.08 (5.16)	Monthly diarrheal mortality per 100,000 population among children under the age of two
<i>ln(Population)</i>	13.2 (.762)	Natural log of the city population
<i>Percent Female</i>	.500 (.018)	Percent of the city population that was female
<i>Percent Nonwhite</i>	.072 (.072)	Percent of the city population that was nonwhite
<i>Percent Foreign</i>	.212 (.096)	Percent of the city population that was foreign born
<i>Percent Age < 15</i>	.253 (.031)	Percent of the city population that was less than 15 years of age
<i>Percent Age 15-44</i>	.529 (.025)	Percent of the city population that was 15-44 years of age
<i>Percent Age 45+</i>	.218 (.030)	Percent of the city population that was 45 years of age or older

N = 6,552

Notes: Unweighted means with standard deviations in parentheses.

Table 2. Public Health Interventions and Summer Diarrhea, 1910-1930

	(1)	(2)	(3)
	ln(<i>Diarrhea</i>)		
<i>Filtration</i>	-.176*** (.056)	-.186*** (.047)	-.161*** (.047)
<i>Filtration</i> × <i>Summer</i>	.160** (.077)	.214** (.100)	.190* (.099)
<i>Chlorination</i>	-.021 (.067)	-.017 (.059)	-.029 (.062)
<i>Chlorination</i> × <i>Summer</i>	.078 (.100)	.069 (.081)	.081 (.081)
<i>Clean Water Project</i>102 (.132)	.120 (.131)
<i>Clean Water Project</i> × <i>Summer</i>106 (.097)	.116 (.094)
<i>Sewage Treated</i>038 (.077)	.034 (.074)
<i>Sewage Treated</i> × <i>Summer</i>004 (.109)	.017 (.109)
<i>Bacteriological Standard</i>111* (.060)
<i>Bacteriological Standard</i> × <i>Summer</i>	-.135 (.085)
N	6,552	6,552	6,552
R ²	.856	.857	.858

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

Notes: Based on annual data from *Mortality Statistics* for the period 1910-1930, 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 diarrheal deaths among children under the age of two per 100,000 population in city *c* and month *t*. Controls include the city characteristics listed in Table 1, city fixed effects, month-by-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.

Table 3. Robustness Checks: Water Purification Efforts and Summer Diarrhea

	(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
<i>Filtration</i>	-.149*** (.043)	-.192*** (.047)	-.106* (.053)	-.154*** (.044)	-.149*** (.049)	-1.08*** (.351)
<i>Filtration</i> × <i>Summer</i>	.191* (.099)	.179* (.092)	.140 (.095)	.209** (.099)	-.159* (.087)	1.17 (.699)
<i>Chlorination</i>	-.033 (.060)	-.048 (.046)	-.026 (.052)	.007 (.057)	-.037 (.063)	-.466 (.430)
<i>Chlorination</i> × <i>Summer</i>	.079 (.080)	.080 (.077)	.076 (.090)	.027 (.090)	.096 (.083)	1.52* (.792)
N	6,529	6,552	6,552	6,300	5,304	6,552
R ²	.859	.865	.787	.836	.865	.802

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

Notes: Based on annual data from *Mortality Statistics* for the period 1910-1930, 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 diarrheal deaths among children under the age of two per 100,000 population in city c and year t . In column (6), the dependent variable is equal to the number of diarrheal deaths among children under the age of two per 100,000 population in city c and year t . Controls include the city characteristics listed in Table 1, the other policy interventions (*Clean Water Project*, *Sewage Treated*, and *Bacteriological Standard*) and their interaction with *Summer*, city fixed effects, month-by-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.

Table 4. Diarrheal Mortality and Leads and Lags of Water Filtration

	(1)	(2)	(3)	(4)
	$\ln(\text{Diarrhea})$		$\ln(\text{Diarrhea})$	
	Non-Summer Months	Summer Months	Non-Summer Months	Summer Months
<i>3 or More Years Prior to Filtration</i>022 (.065)	.071 (.123)
<i>2 Years Prior to Filtration</i>	-.092 (.060)	.149 (.110)
<i>1 Year Prior to Filtration</i>
<i>Year 0</i>	-.168** (.065)	.045 (.093)	-.159** (.062)	.070 (.107)
<i>1 Year After Filtration</i>	-.124** (.059)	-.075 (.093)	-.118** (.054)	-.051 (.106)
<i>2 Years After Filtration</i>	-.161** (.067)	-.052 (.124)	-.156** (.058)	-.033 (.133)
<i>3 Years After Filtration</i>	-.103 (.075)	-.081 (.140)	-.101 (.081)	-.062 (.144)
<i>4 Years After Filtration</i>	-.152* (.076)	-.105 (.128)	-.152* (.078)	-.088 (.134)
<i>5 or More Years After Filtration</i>	-.216** (.097)	.022 (.148)	-.221** (.094)	.036 (.149)
N	6,552	6,552	6,552	6,552
R ²	.859	.859	.860	.860

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

Notes: Based on annual data from *Mortality Statistics* for the period 1910-1930, published by the U.S. Census Bureau. Columns (1)-(2) represent the results from an OLS regression where the omitted category is all years prior to filtration. Columns (3)-(4) represent results from an OLS regression where the omitted category is 1 year prior to filtration. The even-numbered columns represent results based on the sum of the coefficient estimate on the indicated lead (or lag) and the coefficient estimate on the interaction between the indicated lead (or lag) and *Summer*. The dependent variable is equal to the natural log of the number of diarrheal deaths among children under the age of two per 100,000 population in city *c* and month *t*. Controls include the city characteristics listed in Table 1, the other policy interventions (*Clean Water Project*, *Sewage Treated*, and *Bacteriological Standard*) and their interaction with *Summer*, city fixed effects, month-by-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.

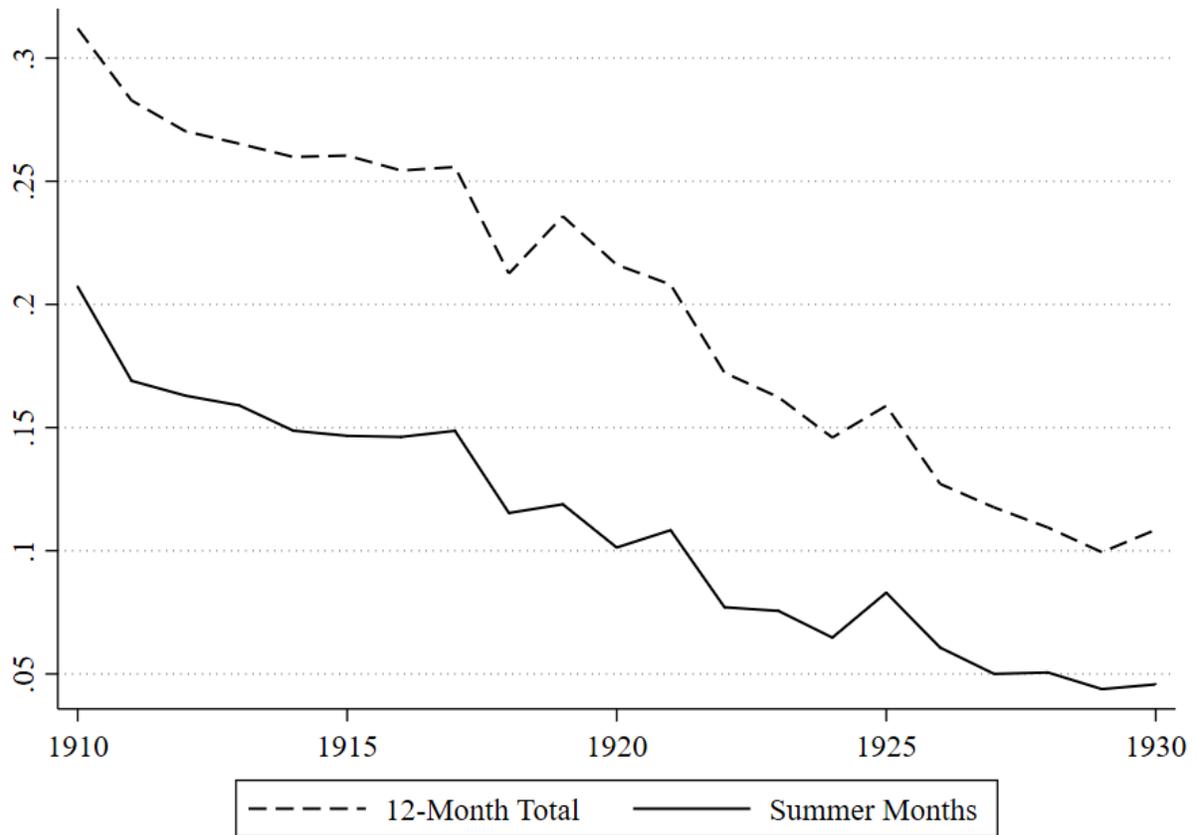
Table 5. Public Health Interventions and Summer Diarrhea by Race

	(1)	(2)
	$\ln(\text{Diarrhea} - \text{Whites})$	$\ln(\text{Diarrhea} - \text{Blacks})$
<i>Filtration</i>	-.099* (.056)	.108 (.124)
<i>Filtration</i> × <i>Summer</i>	.208* (.111)	.165 (.153)
<i>Chlorination</i>	-.088 (.081)	-.086 (.068)
<i>Chlorination</i> × <i>Summer</i>	.196** (.082)	-.022 (.160)
<i>Clean Water Project</i>	.085 (.116)	1.74*** (.220)
<i>Clean Water Project</i> × <i>Summer</i>	.081 (.108)	-.151 (.183)
<i>Sewage Treated</i>	.059 (.075)	-.184 (.223)
<i>Sewage Treated</i> × <i>Summer</i>	-.091 (.126)	-.006 (.160)
<i>Bacteriological Standard</i>	-.003 (.047)	-.136 (.111)
<i>Bacteriological Standard</i> × <i>Summer</i>	.002 (.085)	-.068 (.183)
N	3,456	3,456
R ²	.887	.529

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

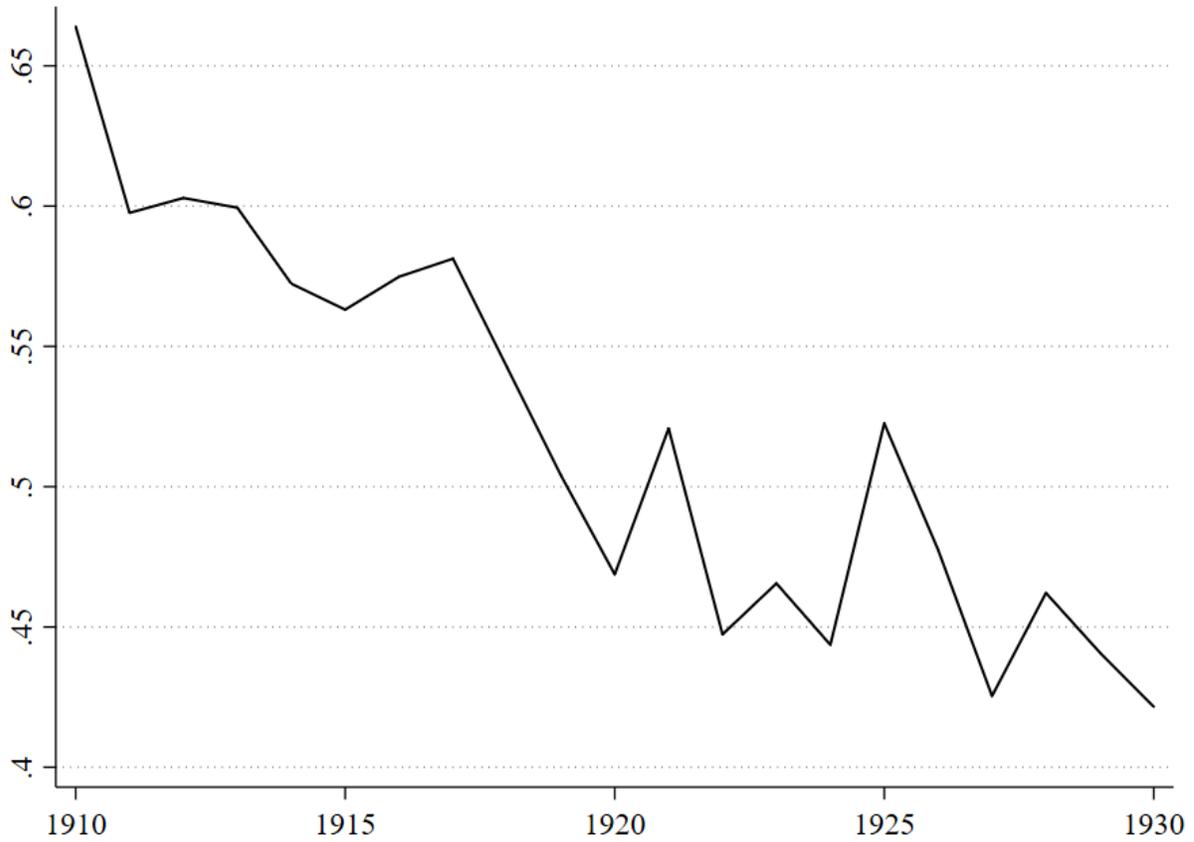
Notes: Based on annual data from *Mortality Statistics* for the period 1910-1930, published by the U.S. Census Bureau. Each column represents the results from a separate OLS regression. The dependent variable is equal to the natural log of the number of diarrheal deaths among children under the age of two per 100,000 of the relevant population in city c and month t . Controls include the city characteristics listed in Table 1, city fixed effects, month-by-year fixed effects, and city-specific linear trends. Regressions are weighted by the relevant city population. Standard errors, corrected for clustering at the city level, are in parentheses.

**Appendix Figure 1. Ratio of Diarrheal Mortality to Total Mortality
Among Children Under the Age of Two**



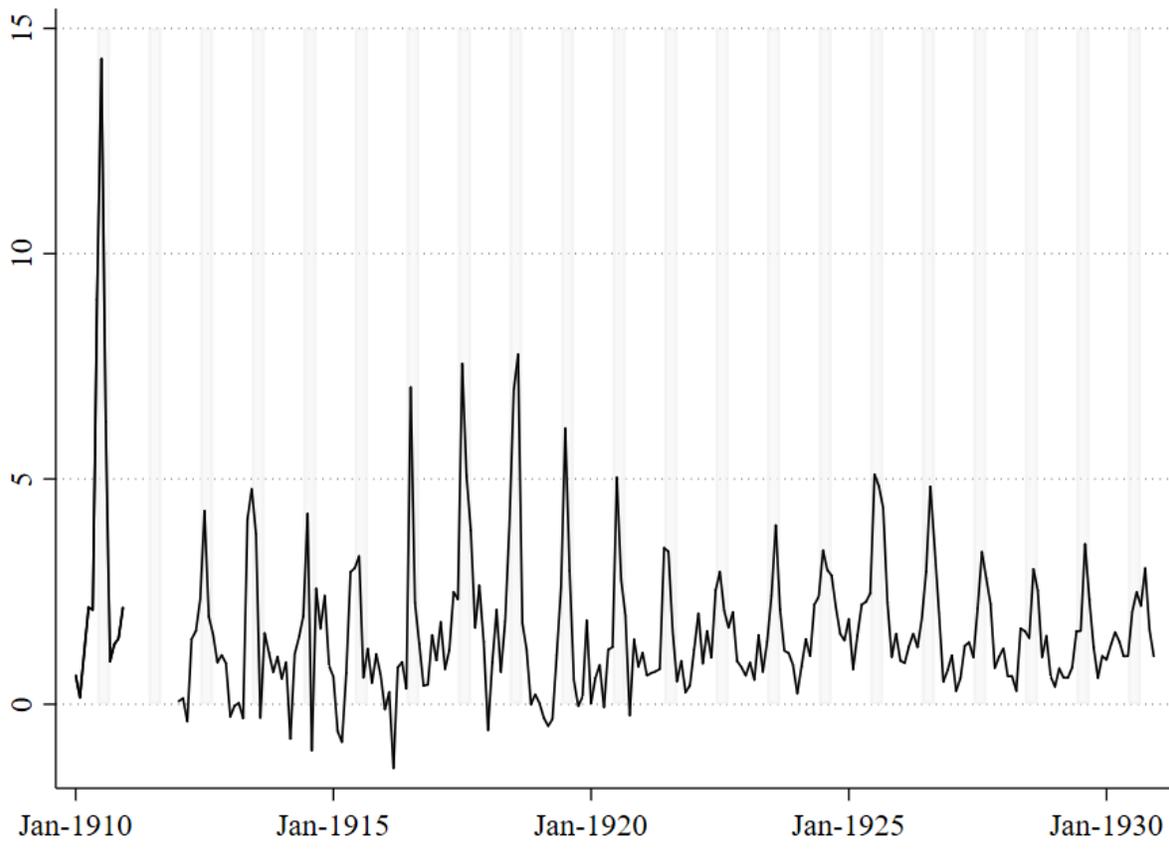
Notes: Based on data from *Mortality Statistics* for the 26 cities under study, published by the U.S. Census Bureau. The summer months are defined as June through September.

Appendix Figure 2. Ratio of Summer Diarrheal Mortality to Total Diarrheal Mortality Among Children Under the Age of Two



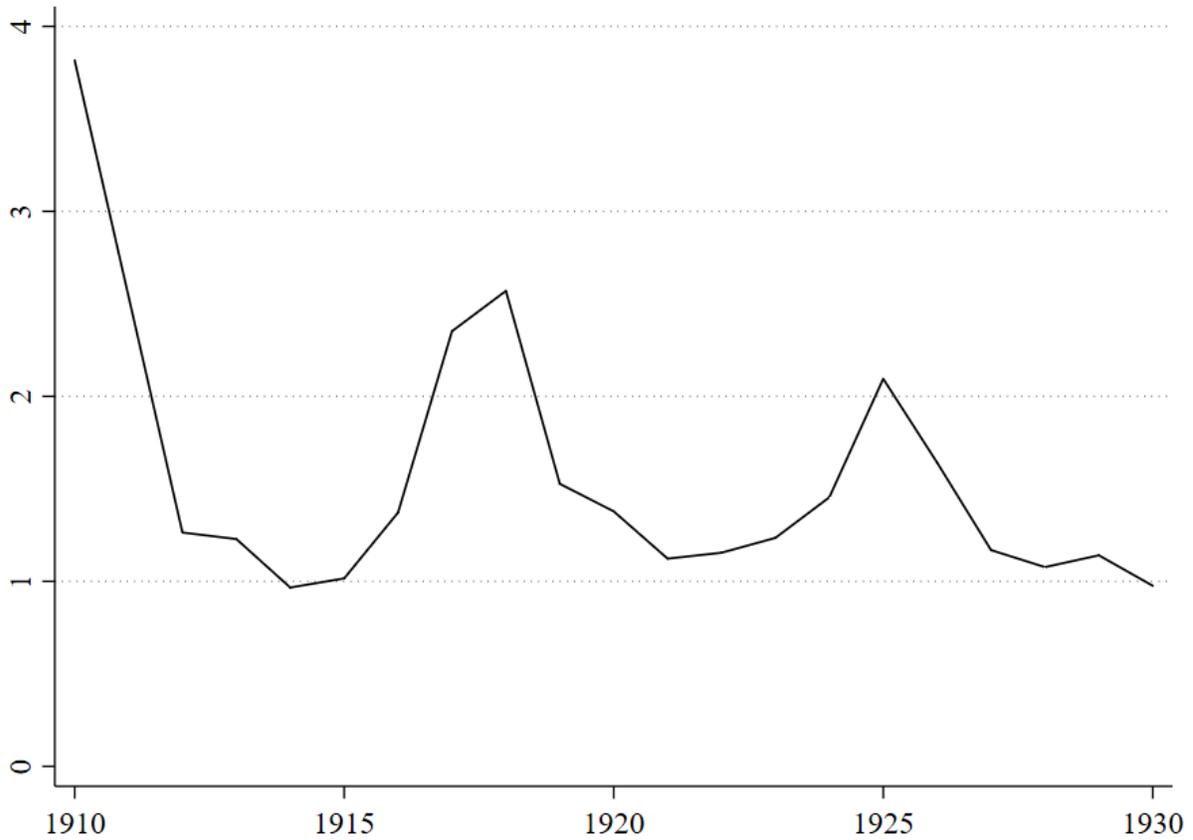
Notes: Based on data from *Mortality Statistics* for the 26 cities under study, published by the U.S. Census Bureau. The summer months are defined as June through September.

Appendix Figure 3. Monthly Black-White Diarrheal Mortality Gap Among Children Under the Age of Two per 100,000 Population



Notes: Based on data from *Mortality Statistics* for the 19 cities under study, published by the U.S. Census Bureau. The shaded vertical bars indicate the summer months (June-September). Diarrheal mortality counts by race are unavailable for 1911.

Appendix Figure 4. Black-White Diarrheal Mortality Gap Among Children Under the Age of Two per 100,000 Population During Non-Summer Months



Notes: Based on data from *Mortality Statistics* for the 19 cities under study, published by the U.S. Census Bureau. Diarrheal mortality counts by race are unavailable for 1911.

Appendix Table 1. Municipal Water Purification, 1900-1930

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
Los Angeles, California	...	1925
Louisville, Kentucky	1909	1913
Milwaukee, Wisconsin	...	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
Seattle, Washington	...	1911
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.

Notes: Identification of the *Filtration* indicator comes from the cities that began filtering their water supply during the period 1910-1930. Identification of the *Chlorination* indicator comes from the cities that began chlorinating their water supply during the period 1910-1930.

Appendix Table 2. Clean Water Projects, 1900-1930

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.
Los Angeles, California	1913	Los Angeles began receiving water from Owens Valley on November 5, 1913.
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.

Notes: Identification of the *Clean Water Project* indicator comes from the cities that undertook clean water project during the period 1910-1930.

Appendix Table 3. Sewage Treatment/Diversion, 1900-1930

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).
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.
Newark, New Jersey	1924	The sewage treatment plant began operations in 1924.
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

Notes: Identification of the *Sewage Treated* indicator comes from the cities that began treating their sewage during the period 1910-1930.

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Appendix Table 4. Bacteriological Standards for Milk, 1900-1930

City and State	Bacteriological Standard ^a
Baltimore, Maryland	1913
Boston, Massachusetts	1905
Buffalo, New York	1918
Chicago, Illinois	1909
Cincinnati, Ohio	1914
Cleveland, Ohio	1906
Detroit, Michigan	1915
Indianapolis, Indiana	1916
Jersey City, New Jersey	1915
Kansas City, Missouri	1910
Los Angeles, California	1905
Milwaukee, Wisconsin	1908
Minneapolis, Minnesota	1907
Newark, New Jersey	1913
New Orleans, Louisiana	1923
New York, New York	1912
Philadelphia, Pennsylvania	1915
Pittsburgh, Pennsylvania	1910
Providence, Rhode Island	1915
Rochester, New York	1907
San Francisco, California	1909
Seattle, Washington	1910
St. Louis, Missouri	1923
St. Paul, Minnesota	1907

^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.

Notes: Identification of the *Bacteriological Standard* indicator comes from the cities that began requiring that milk sold within their limits meet a bacteriological standard during the period 1910-1930.

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**Appendix Table 5. Public Health Interventions and Summer Diarrhea,
1910-1930**

	(1)	(2)	(3)
	ln(<i>Diarrhea</i>)		
<i>Filtration</i>	-.137** (.058)	-.131*** (.056)	-.113*** (.054)
<i>Filtration</i> × 70°F and Above	.209** (.091)	.234** (.098)	.236** (.100)
<i>Chlorination</i>	-.005 (.051)	-.002 (.046)	-.014 (.047)
<i>Chlorination</i> × 70°F and Above	.056 (.075)	.033 (.073)	.071 (.073)
<i>Clean Water Project</i>123 (.139)	.148 (.136)
<i>Clean Water Project</i> × 70°F and Above096 (.085)	.117 (.095)
<i>Sewage Treated</i>044 (.066)	.044 (.061)
<i>Sewage Treated</i> × 70°F and Above	...	-.007 (.140)	.013 (.145)
<i>Bacteriological Standard</i>077 (.049)
<i>Bacteriological Standard</i> × 70°F and Above	-.064 (.088)
N	6,552	6,552	6,552
R ²	.857	.858	.858

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

Notes: Based on annual data from *Mortality Statistics* for the period 1910-1930, 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 diarrheal deaths among children under the age of two per 100,000 population in city *c* and month *t*. Controls include the city characteristics listed in Table 1, city fixed effects, month-by-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.

**Appendix Table 6. Cities and Years Covered
in Summer Diarrhea Analysis by Race**

City and State	Years Covered
Baltimore, Maryland	1910, 1912-1930
Boston, Massachusetts	1913-1915, 1918
Chicago, Illinois	1910, 1912-1930
Cincinnati, Ohio	1910, 1912-1930
Cleveland, Ohio	1920-1930
Detroit, Michigan	1920-1930
Indianapolis, Indiana	1910, 1912-1930
Kansas City, Missouri	1910, 1912-1930
Los Angeles, California	1914, 1915, 1918-1930
Louisville, Kentucky	1910, 1912-1930
New Orleans, Louisiana	1910, 1912-1930
New York, New York	1910, 1912-1930
Newark, New Jersey	1930
Philadelphia, Pennsylvania	1910, 1912-1930
Pittsburgh, Pennsylvania	1910, 1912-1930
San Francisco, California	1913-1915, 1918, 1930
Seattle, Washington	1919
St. Louis, Missouri	1910, 1912-1930
Washington, D.C.	1910, 1912-1930