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WATER PURIFICATION EFFORTS AND THE BLACK-WHITE INFANT MORTALITY GAP, 1906-1938

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ABSTRACT

According to Troesken (2004), efforts to purify municipal water supplies at the turn of the 20th century dramatically improved the relative health of blacks. There is, however, little empirical evidence to support the Troesken hypothesis. Using city-level data published by the U.S. Bureau of the Census for the period 1906-1938, we explore the relationship between water purification efforts and the black-white infant mortality gap. Our results suggest that, while water filtration was effective across the board, adding chlorine to the water supply reduced mortality only among black infants. Specifically, chlorination is associated with an 11 percent reduction in black infant mortality and a 13 percent reduction in the black-white infant mortality gap. We also find that chlorination led to a substantial reduction in the black-white diarrhea mortality gap among children under the age of 2, although this estimate is measured with less precision.

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1. INTRODUCTION

In the United States, black infants are more than twice as likely than their white counterparts to die within the first year of life (Aron 2013; Riddell et al. 2017). Although the black-white infant mortality gap has existed since at least the turn of the 20th century, when reliable mortality data by race first became available, its causes are still being researched and debated today (Ewbank 1987; Elder et al. 2016; Speights et al. 2017).

This study is the first to explore whether water purification efforts (i.e., the building of water filtration plants and adding chlorine to the water supply) at the municipal level can explain the evolution of the black-white infant mortality gap during the first decades of the 20th century, a period when urban mortality rates—especially infant mortality rates—were in steep decline (Cutler and Miller 2005; Anderson et al. forthcoming). Previous studies provide evidence that the construction of filtration plants led to substantial reductions in infant mortality (Cutler and Miller 2005; Anderson et. al. forthcoming), but these studies do not provide estimates by race.

In *Water, Race, and Disease*, Troesken (2004, p. *xv*) famously hypothesizes that, because urban blacks and whites lived in close proximity to each other ("almost side by side") at the turn of the 20th century and drew upon the same sources of drinking water, efforts to clean the water supply led to an improvement in the relative health of blacks.[1] The negative and statistically significant association between filtration and typhoid mortality among blacks living in major American cities documented by Troesken (2002) supports this hypothesis, but typhoid deaths were only a small proportion of total deaths during the first decades of the 20th century and never amounted to more than half of mortality from diarrhea/enteritis (Anderson et al. forthcoming),

[1] Specifically, Troesken (2004, p. 2004) writes that because "there was limited discrimination in the provision of public water and sewer lines...blacks benefited more than whites, in terms of disease reduction, from investments in water and sewer lines and water purification systems" (Troesken 2004, p. 204). For reviews of *Water, Race, and Disease*, see Kiple (2005), Harper (2007) and Humphreys (2007).

the second-leading cause of infant mortality (Wegman 2001). Whether, and the extent to which, water purification efforts contributed to the evolution of the black-white infant mortality gap is still an open question.

Using infant mortality data on 19 major American cities for the period 1906-1938, we find mixed evidence in support of the Troesken hypothesis. Specifically, water filtration is associated with infant mortality reductions of comparable magnitude among both blacks and whites. By contrast, there is no evidence that chlorination reduced white infant mortality; among blacks, however, chlorination is associated with an 11 percent reduction in infant mortality. Adding chlorine to the water supply is also associated with a 13 percent reduction in the black-white infant mortality gap and a 23 percent decrease in the black-white diarrhea/enteritis mortality gap among children under the age of 2, although it should be noted that this latter estimate is measured with less precision.

2. Background and Previous Studies

Contemporary public health experts recognized that the infant mortality rate was significantly higher among blacks than whites (Billings 1883; Du Bois 1899; Miller 1906). The reasons for this discrepancy, however, were fiercely 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 (1906) listed 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).

Previous studies provide strong evidence that water filtration efforts undertaken at the turn of the 20th century led to substantial reductions in infant mortality (Cutler and Miller 2005; Anderson et al. forthcoming). For instance, using data on 25 major American cities for the period 1900-1940, Anderson et al. (forthcoming) find that filtration is associated with an 11-12 percent reduction in infant mortality.[2] Although Cutler and Miller (2005) and Anderson et al. (forthcoming) do not find evidence that adding chlorine to the water improved the health of infants, neither of these studies provide estimates by race.

In fact, with only a few exceptions, historical studies on the relationship between water quality and mortality do not provide estimates by race. Among these exceptions is Troesken (2002), who draws upon data from 33 U.S. cities at the turn of the 20th century to estimate the relationship between water filtration and typhoid mortality.[3] Typhoid mortality is commonly used by researchers in this field as a proxy for water quality and to track diarrheal deaths (Cutler and Miller 2005; Ferrie and Troesken 2008; Clay et al. 2014). Troesken (2002) finds that water filtration was associated with a 53 percent reduction in the black typhoid mortality rate. By

[2] Using data on 13 major American cities for the period 1900-1936, Cutler and Miller (2005) find that the building of a water filtration plant is associated with a 35 percent reduction in infant mortality. However, Anderson et al. (forthcoming) show that, when a series of transcription errors are corrected, Cutler and Miller's (2005) infant mortality estimate is reduced by two-thirds. Studies on the relationship between early water purification efforts and infant/child mortality outside the United States include Knutsson (2017) and Ogasawara et al. (2018).

[3] The majority of these cities contributed 15 years of data to Troesken's analysis (1906-1920). Three cities (Charleston, SC; Charlotte, NC and Pittsburgh, PA) contributed 31 years of data (1890-1920).

contrast, filtration is associated with a (statistically insignificant) 16 percent reduction in the white typhoid mortality rate. Troesken (2002, p. 750) interprets this pattern of results as evidence "against the hypothesis that cities systematically denied African Americans access to public water systems." Relatedly, Troesken (2001) finds 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.[4]

3. DATA

Infant mortality counts by race at the city-year level for the period 1906-1938 come from *Mortality Statistics* and *Vital Statistics of the United States*, both of which were published annually by the U.S. Census Bureau. These data sources have been used by previous researchers interested in exploring the causes of the mortality transition (Cutler and Miller 2005; Anderson et al. 2019a, 2019b; Anderson et al. forthcoming), during which infant mortality rates fell precipitously (Cutler and Miller 2005; Anderson et al. forthcoming).[5] Our focus is on 19 of the most populous American cities as of 1910, which are listed in Online Appendix Table 1.[6]

[4] Specifically, Troesken (2001) finds that public ownership significantly reduced waterborne-disease mortality (defined as the sum of deaths due to cholera, diarrhea, dysentery and typhoid) among African Americans, but had a much smaller effect among whites. Collins and Thomasson (2004) examine the correlates of the black-white infant mortality gap in the United States. During the period 1920-1945, these authors find that a large portion of this gap can be explained by factors such as income, education and location. After 1945, "observable characteristics lost much of their explanatory power…" (Collins and Thomasson 2004, p. 772).

[5] Anderson et al. (forthcoming) focus on city-level mortality data for the period 1900-1940. City-level infant mortality data by race are not available for the years 1900-1906 or 1939-1940.

[6] With the exceptions of Los Angeles, CA and Seattle, WA, these cities had populations greater than 150,000 in 1900 and reported mortality counts by race. Although Los Angeles and Seattle were smaller than the other cities used in the analysis, they were included because information on their public health interventions was available as were mortality counts by race.

Because information on live births at the municipal level is not available for this period, infant mortality rates (IMRs) are calculated per 100,000 of the race-specific population. Previous studies exploring the determinants of city-level mortality during this period calculate IMRs in a similar fashion (Cutler and Miller 2005; Clay et al. 2014; Komisarow 2017; Anderson et al. forthcoming).[7]

Figure 1 shows the black IMR, the white IMR and the black-white IMR ratio for the years 1906-1938.[8] Infant mortality rates fell dramatically among both blacks and whites during these years. At first, the black IMR fell faster than the white IMR, leading to a substantial reduction in the black-white IMR ratio. Specifically, between 1906 and 1915, the black-white IMR ratio fell from 1.91 to 1.34, or 29.8 percent. This trend reversed after 1915 and the black-white IMR ratio increased to 2.07 by 1938, or 54.5 percent.

Online Appendix Table 1 provides filtration and chlorination dates for each of the 19 cities that contributed data to the analysis. These dates correspond to those used by previous researchers and were obtained from a mix of primary and secondary sources.[9] During the period under study, 12 of the 19 cities that contributed data to the analysis constructed water filtration plants and all 19 began adding chlorine to their drinking water.[10]

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[7] *Mortality Statistics* began reporting live births for registration states in 1915 (Linder and Grove 1947, Table 44).

[8] City-level infant mortality counts by race (white versus nonwhite) are from *Mortality Statistics* for the period 1906-1936 (U.S. Bureau of the Census 1908-1938) and from *Vital Statistics of the United States* for the period 1937-1938 (U.S. Bureau of the Census 1939-1940). Because over 95 percent of the minority population in the United States was black during the first decades of the 20th century (Gibson and Jung 2002), we refer to "nonwhites" as "blacks." City-level populations by race come from the decennial census and intercensal population estimates were produced by linear interpolation.

[9] For the list of sources, see the appendices in Anderson et al. (2019b) and Anderson et al. (forthcoming). In Online Appendix Table 1, we report the years for which infant mortality counts were available for every city in the analysis.

[10] See the National Center for Environmental Health (1999), Melosi (2008) and Anderson et al. (forthcoming) for more information on the history and technical aspects of chlorination and filtration. Filtration technology was developed as a method of reducing the turbidity and improving the taste of drinking water. It became more popular as the study of bacteriology advanced and municipal governments came under pressure to protect their citizens from

4. EMPIRICAL STRATEGY

We begin our examination of whether water purification efforts contributed to the trends documented in Figure 1, by estimating the following regression by race:

$$(1) \qquad \ln(IMR_{ct}) = \beta_0 + \beta_1 Filtration_{ct} + \beta_2 Chlorination_{ct} + \boldsymbol{X}_{ct}\boldsymbol{\beta_3} + v_c + w_t + \Theta_c \cdot t + \varepsilon_{ct},$$

where c indexes cities and t indexes years. Our primary interest is in the indicators *Filtration* and *Chlorination*. *Filtration* is equal to 1 if city c was filtering its water supply in year t (and is equal to 0 otherwise); *Chlorination* is equal to 1 if city c was adding chlorine to its water supply in year t (and is equal to 0 otherwise).[11]

The vector of controls, $\boldsymbol{X}_{ct}$, is composed of city demographics (i.e., percent female, nonwhite, foreign born, under 15 years of age, 15-44 years of age and 45 years of age or older) taken from the 1900-1940 Censuses and linearly interpolated for intercensal years. In addition to these demographics, we control for other public health interventions that could have impacted IMRs. These interventions, which are described by Anderson et al. (forthcoming), include an indicator for whether a city completed a large-scale project to deliver clean water from further afield (e.g., aqueducts and water cribs), an indicator for whether sewage was treated before its discharge into rivers and lakes, and indicators for municipal-level efforts to provide clean milk.[12]

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water-borne diseases, especially typhoid. In 1908, Jersey City, New Jersey became the first city to routinely (i.e., continuously) add chlorine to its water supply in an effort to combat water-borne diseases. Because the chlorination process was simple and inexpensive, other major cities quickly followed suit. By 1925, every city in our sample was chlorinating its water supply.

[11] When the exact date was available, these indicators were set equal to the proportion of the year during which the water supply was filtered/chlorinated.

[12] Examples of clean water projects include the "Five Mile Crib," which began delivering water from Lake Erie to Cleveland, OH on April 6, 1904, and the building of the Wanaque Reservoir, which began delivering water to Newark, NJ on March 20, 1930.

Online Appendix Table 2 presents descriptive statistics and definitions of the variables contained in $\boldsymbol{X}_{ct}$.

Finally, city fixed effects (v_c) control for city-level determinants of mortality that were constant over time and the year fixed effects (w_t) control for common shocks such as changes in medical knowledge and technology.[13] City-specific linear time trends ($\Theta_c \cdot t$) account for the possibility that mortality rates evolved differently across the cities in the sample.

After estimating the baseline regression described above for whites and blacks separately, we replace the IMR with the black-white infant mortality gap, measured as $\ln\left(\frac{Black\ IMR}{White\ IMR}\right)$, to estimate the following regression[14]:

$$(2) \qquad \ln\left(\frac{Black\ IMR_{ct}}{White\ IMR_{ct}}\right) = \alpha_0 + \alpha_1 Filtration_{ct} + \alpha_2 Chlorination_{ct} + \boldsymbol{X}_{ct}\boldsymbol{\alpha_3} + v_c + w_t + \Theta_c \cdot t + \varepsilon_{ct}.$$

Our interest is in the estimates of α_1 and α_2. Negative and statistically significant estimates of α_1 and α_2 would lend support to Troesken's (2004) hypothesis.

5. RESULTS

In columns (1) and (2) of Table 1, we report estimates on the relationship between infant mortality and water purification efforts by race. Standard errors are corrected for clustering at

[13] For instance, during the period under study there was "great progress in understanding fluid and electrolyte therapy," which likely contributed to the sharp decline in infant mortality from diarrheal diseases (Wegman 2001, p. 404S).

[14] Similarly constructed outcomes have been used by previous researchers interested in exploring the determinants of other types of black-white gaps (LaFree et al. 2010; Quillian et al. 2017; Myers et al. 2018).

the city level (Bertrand et al. 2004).[15] Filtering drinking water is associated with a 12 percent reduction ($e^{-0.123} - 1 = -0.116$) in the white infant mortality rate, and a 19 percent reduction ($e^{-0.205} - 1 = -0.185$) in the black infant mortality rate. While the effect for blacks is nearly 60 percent larger than for whites, we cannot formally reject the hypothesis that the estimated coefficients are equal. By contrast, adding chlorine to the water supply appears to have only been effective at reducing the black IMR. The estimated coefficient on *Chlorination* is small, positive and statistically indistinguishable from zero for white infants; for black infants, chlorination is associated with an 11 percent reduction in the IMR, and this estimate is statistically significant at the 5 percent level.[16]

The third column of Table 1 shows the associations between water purification efforts and $\ln\left(\frac{Black\ IMR}{White\ IMR}\right)$, our measure of the black-white infant mortality gap. Chlorination is associated with a 13 percent decrease in the black-white IMR ratio, while the association between filtration and the black-white IMR ratio is statistically indistinguishable from zero.[17]

[15] P-values calculated from the wild cluster bootstrap procedure method suggested by Cameron et al. (2008) and Cameron and Miller (2015) are reported in curly brackets.

[16] In the online appendix, we present these results graphically using event studies, replacing *Filtration* and *Chlorination* with sets of mutually exclusive leads and lags. Importantly, Online Appendix Figures 1 and 2 show no evidence that IMRs began trending downwards prior to the adoption of filtration, suggesting the parallel trends assumption is satisfied. Online Appendix Figures 3 and 4 show chlorination-related results that are also consistent with parallel pre-treatment trends.

[17] In Online Appendix Table 3, we show the coefficient estimates on the other public health interventions contained in $\boldsymbol{X}_{ct}$. In general, there is little evidence that clean water projects or efforts to clean the municipal milk supply reduced mortality among either white or black infants. However, sewage treatment is associated with a 13 percent reduction in the white IMR. Anderson et al. (forthcoming) find no evidence that treatment of a city's effluent contributed to the observed IMR decline in their sample of 25 cities for the period 1900-1940. Using data on municipalities in Massachusetts for the period 1880-1920, Alsan and Goldin (2019) study the effects of clean water and access to a regional sewerage system on infant and child mortality. They find that the interaction of clean water and sewerage explained roughly one-third of the observed decline in child mortality and nearly one-half of the observed decline infant mortality.

6. WATER PURIFICATION AND THE DIARRHEA MORTALITY GAP

Diarrheal diseases were the second-leading cause of death among infants living in the United States at the turn of the 20th century, just behind pneumonia/influenza (Wegman 2001, p. 404S). Moreover, black infants and children were much more likely to die from diarrheal diseases, including "cholera infantum," than their white counterparts (Billings 1883; Du Bois 1899; Miller 1906).[18] Although myriad factors likely contributed to the black-white diarrheal mortality gap, our focus is on water purification efforts at the municipal level. Specifically, we estimate the slightly modified version of equation (1) by race:

$$(3) \qquad \ln(Diarrhea_{ct}) = \beta_0 + \beta_1 Filtration_{ct} + \beta_2 Chlorination_{ct} + \boldsymbol{X_{ct}\beta_3} + v_c + w_t + \Theta_c \cdot t + \varepsilon_{ct},$$

where the IMR is replaced by diarrhea/enteritis deaths among children under the age of two per 100,000 of the race-specific population.[19] We also estimate a modified version of equation (2):

$$(4) \qquad \ln\left(\frac{Black\ Diarrhea_{ct}}{White\ Diarrhea_{ct}}\right) = \alpha_0 + \alpha_1 Filtration_{ct} + \alpha_2 Chlorination_{ct} + \boldsymbol{X_{ct}\alpha_3} + v_c + w_t + \Theta_c \cdot t + \varepsilon_{ct},$$

[18] The label "cholera infantum" was often applied to particularly virulent episodes of diarrhea that struck infants and children in the summer months (Anderson et al. 2019b).

[19] City-level counts of diarrhea/enteritis deaths among children under the age of two by race (i.e., white versus nonwhite) are from *Mortality Statistics* for the period 1906-1936 (U.S. Bureau of the Census 1908-1938) and from *Vital Statistics of the United States* for the year 1937 (U.S. Bureau of the Census 1939). 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 Online Appendix Table 1, we report the years for which diarrhea/enteritis mortality counts were available for every city in our sample. City-level counts of diarrhea/enteritis deaths are not available for 1938.

where the black-white infant mortality gap is replaced by the black-white diarrhea mortality gap, measured as $\ln\left(\frac{Black\ Diarrhea}{White\ Diarrha}\right)$.[20]

Estimates of equation (3) are reported in the first two columns of Table 2. These estimates provide evidence that the relationship between chlorination and the black-white infant mortality gap documented in Table 2 is, at least in part, driven by the impact of chlorination on diarrheal diseases. Specifically, we find that chlorination is associated with a 17 percent reduction in the diarrhea/enteritis mortality rate among black children under the age of two. The estimated effect of chlorination on diarrhea mortality among white children, while negative, is small in magnitude and nowhere near statistically significant. There is little evidence that filtration led to reductions in diarrhea/enteritis mortality among either white or black children.

In the third column of Table 2, we report the estimated effects of water purification efforts on the black-white diarrhea/enteritis mortality ratio. Chlorination is associated with a 23 percent reduction in the black-white diarrhea mortality gap, but this estimate is not statistically significant at conventional levels (p-value = .123).[21] The estimates reported in Table 2 provide no evidence that water filtration contributed to the narrowing of the black-white diarrhea mortality gap during this period.[22]

[20] $Black\ Diarrhea_{ct}$ is equal to the number of diarrhea/enteritis deaths among black children under the age of two per 100,000 black population and $White\ Diarrhea_{ct}$ is equal to the number of diarrhea/enteritis deaths among white children under the age of two per 100,000 white population. Because the black diarrhea/enteritis mortality count is equal to 0 for five observations, we add 1 to $Black\ Diarrhea_{ct}$ before taking its natural log. The estimates presented below are similar if we simply drop the zeros from the sample or use the quartic root function, rather than take the natural log. The quartic root function has been used by Thomas et al. (2006), Tarozzi et al. (2014) and Ashraf et al. (2015), among others, to deal with zeros.

[21] The p-value from the wild cluster bootstrap procedure is actually slightly smaller, equal to .105.

[22] In Online Appendix Table 4, we show the coefficient estimates on the other public health interventions that compose the vector $\boldsymbol{X}_{ct}$. In general, there is little evidence that clean water projects, sewage treatment, or efforts to clean the municipal milk supply reduced diarrhea/enteritis mortality among white or black children.

7. CONCLUSION

Our estimates provide evidence that filtering municipal water supplies reduced black and white IMRs in this sample of cities for the period 1906-1938.[23] By contrast, and consistent with Troesken's hypothesis, adding chlorine to the water supply appears to have been effective only among black infants. Specifically, chlorination is associated with an 11 percent reduction in the black IMR and a 13 percent reduction of the black-white IMR ratio, our measure of the black-white infant mortality gap.

Chlorination appears to have narrowed the black-white infant mortality gap, at least in part, through its effect on diarrheal diseases. We find that chlorination was associated with a 17 percent reduction in diarrhea/enteritis mortality among black children under the age of two for the period 1906-1937. Although the estimated effect of chlorination on diarrhea mortality among white children is also negative, it is small and statistically insignificant at conventional levels. We find little evidence to suggest that filtration led to reductions in diarrhea/enteritis mortality among either white or black children under the age of two.

In *Water, Race, and Disease*, Troesken (2004) observes that, at the turn of the 20th century, urban blacks and whites lived in close proximity to each other and drew upon the same sources of drinking water. According to Troesken (2004), when municipalities purified the water supply, this had the effect of improving the health of blacks relative to whites. The negative association between adding chlorine to municipal water supplies and the black-white infant mortality gap lends support to his often-cited and well-known argument.

[23] It is worth noting that the size of the estimated filtration effects presented above are similar to those reported by Anderson et al. (forthcoming). As noted above, these authors find that water filtration is associated with an 11-12 percent reduction in infant mortality. Because Anderson et al. (forthcoming) do not explore effects by race, they are able to draw upon on a longer panel of data (i.e., 1900-1940 vs. 1906-1938) for a larger set of cities (i.e., 25 cities vs. 19 cities).

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Figure 1. Infant Mortality Rates by Race, 1906-1938

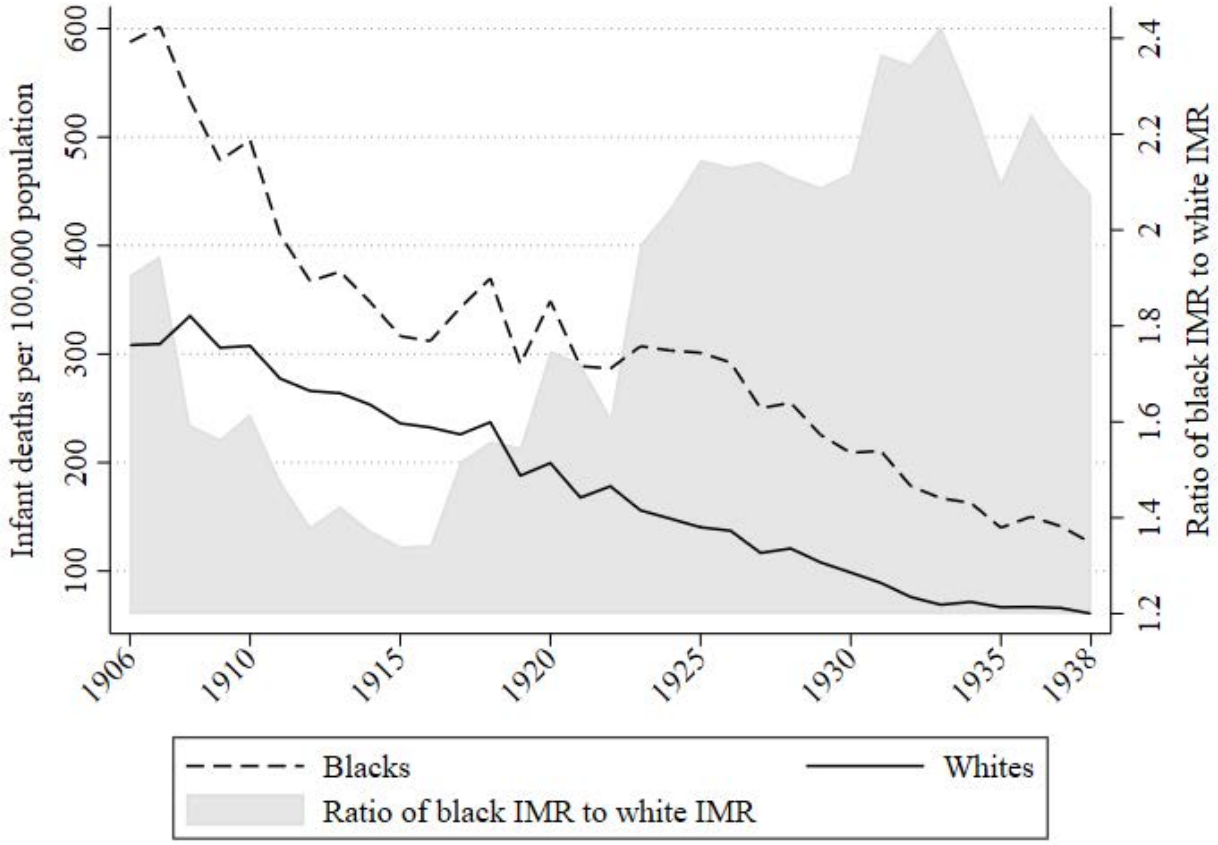


Table 1. Water Purification Efforts and Infant Mortality by Race, 1906-1938

	(1)	(2)	(3)
	ln(*White IMR*)	ln(*Black IMR*)	$\ln\left(\frac{Black\ IMR}{White\ IMR}\right)$
Filtration	-.123*	-.205***	-.106
	(.064)	(.059)	(.106)
	{.155}	{.060}	{.458}
Chlorination	.023	-.119**	-.138**
	(.049)	(.045)	(.061)
	{.666}	{.023}	{.045}
N	506	506	506
R^2	.976	.891	.719

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

Notes: Based on annual data from *Mortality Statistics* and *Vital Statistics of the United States* for the period 1906-1938, published by the U.S. Bureau of the Census. Each column reports estimates from a separate OLS regression. In columns (1)-(2), the dependent variable is equal to the natural log of the number of race-specific infant deaths per 100,000 of the relevant population in city *c* and year *t*, and the regressions are weighted by the relevant city population. In column (3), the dependent variable is equal to the natural log of the ratio of *Black IMR* to *White IMR*, and the regression is weighted by city population. Controls include the city-level characteristics listed in Online Appendix Table 2, city fixed effects, year fixed effects and city-specific linear time trends. 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 2. Water Purification Efforts and Diarrheal Mortality Among Children Under the Age of Two by Race, 1906-1937

	(1)	(2)	(3)
	ln(*White Diarrhea*)	ln(*Black Diarrhea*)	$\ln\left(\frac{Black\ Diarrhea}{White\ Diarrhea}\right)$
Filtration	-.157	.096	.294
	(.133)	(.096)	(.177)
	{.342}	{.331}	{.190}
Chlorination	-.014	-.190**	-.256
	(.125)	(.082)	(.158)
	{.930}	{.012}	{.105}
N	446	446	446
R^2	.970	.881	.613

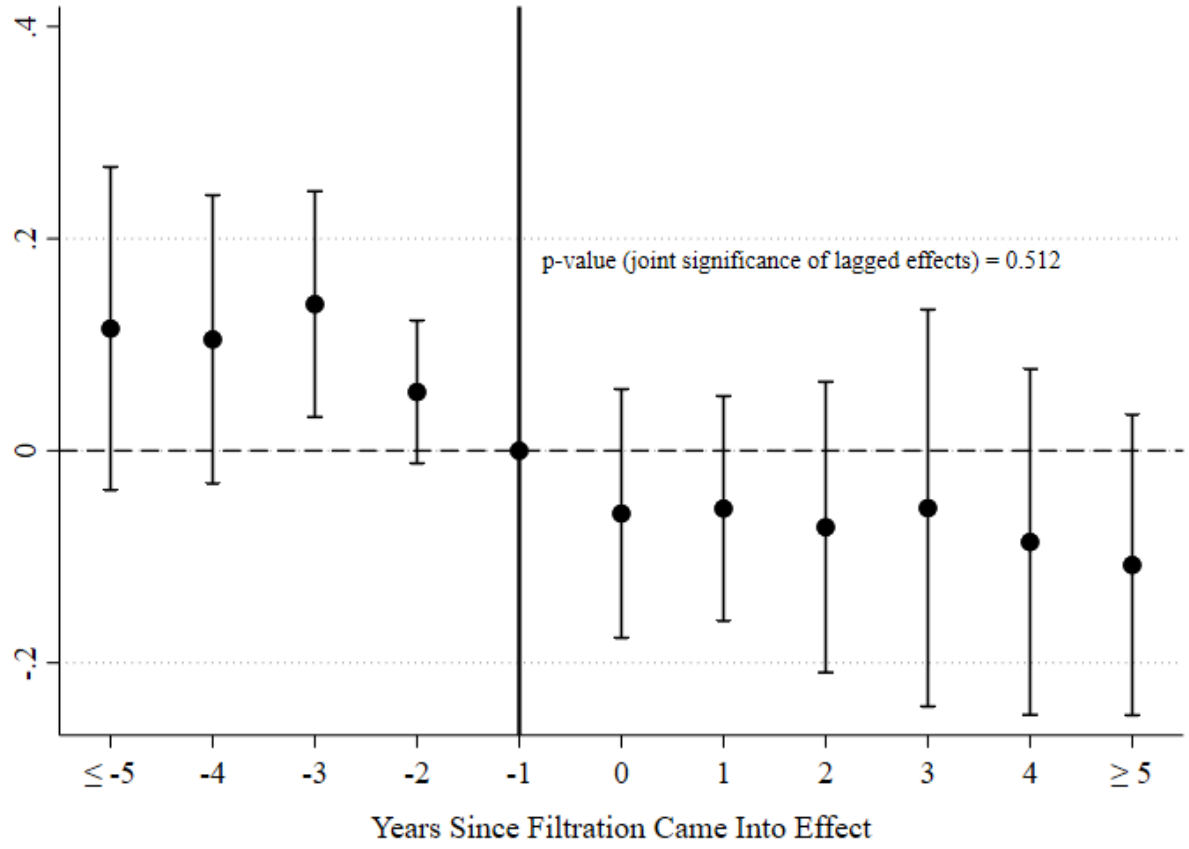
*Statistically significant at 10% level; ** at 5% level; *** at 1% level.

Notes: Based on annual data from *Mortality Statistics* and *Vital Statistics of the United States* for the period 1906-1937, published by the U.S. Bureau of the Census. Each column represents the results from a separate OLS regression. In columns (1)-(2), the dependent variable is equal to the natural log of the number of race-specific diarrhea/enteritis deaths among children under the age of two per 100,000 of the relevant population in city *c* and year *t*, and the regressions are weighted by the relevant city population. In column (3), the dependent variable is equal to the natural log of the ratio of *Black Diarrhea* to *White Diarrhea*, and the regression is weighted by city population. Controls include the city-level characteristics listed in Online Appendix Table 2, city fixed effects, year fixed effects and city-specific linear time trends. 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

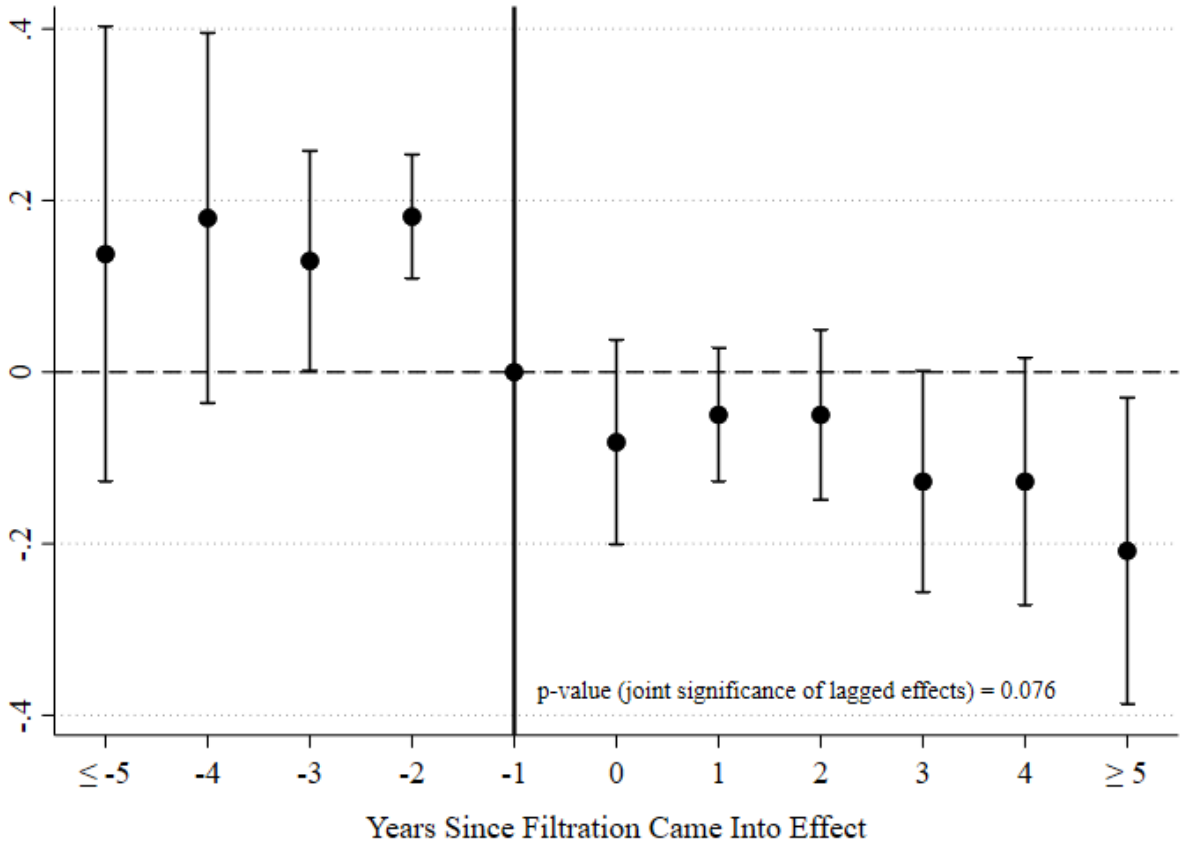
For Online Publication

Online Appendix Figure 1. Pre- and Post-Filtration Trends in White Infant Mortality



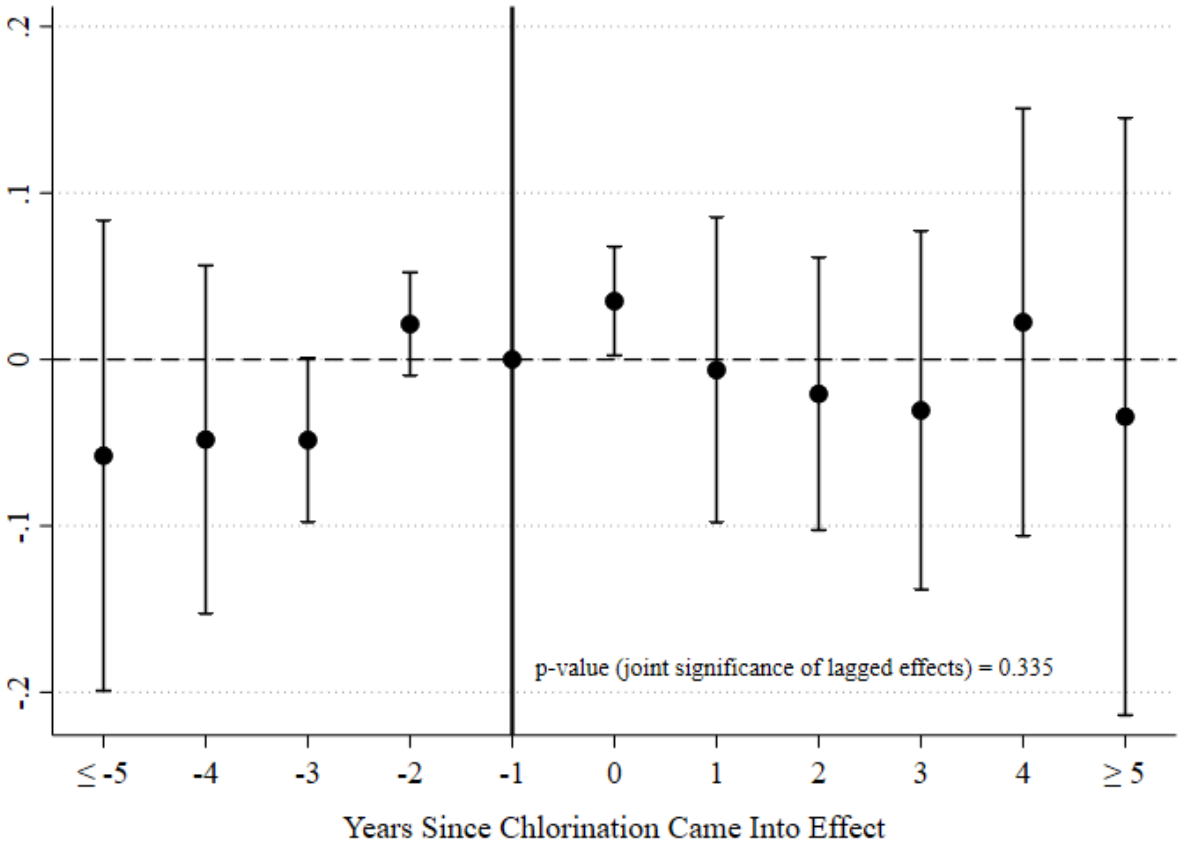
Notes: OLS coefficient estimates (and their 90% confidence intervals) are reported, where the omitted category is 1 year before treatment. The dependent variable is equal to the natural log of the number of white infant deaths per 100,000 of the white population in city *c* and year *t*, and the regressions is weighted by the white city population. Controls include the city-level characteristics listed in Online Appendix Table 2, city fixed effects, year fixed effects and city-specific linear time trends. Standard errors are corrected for clustering at the city level.

Online Appendix Figure 2. Pre- and Post-Filtration Trends in Black Infant Mortality



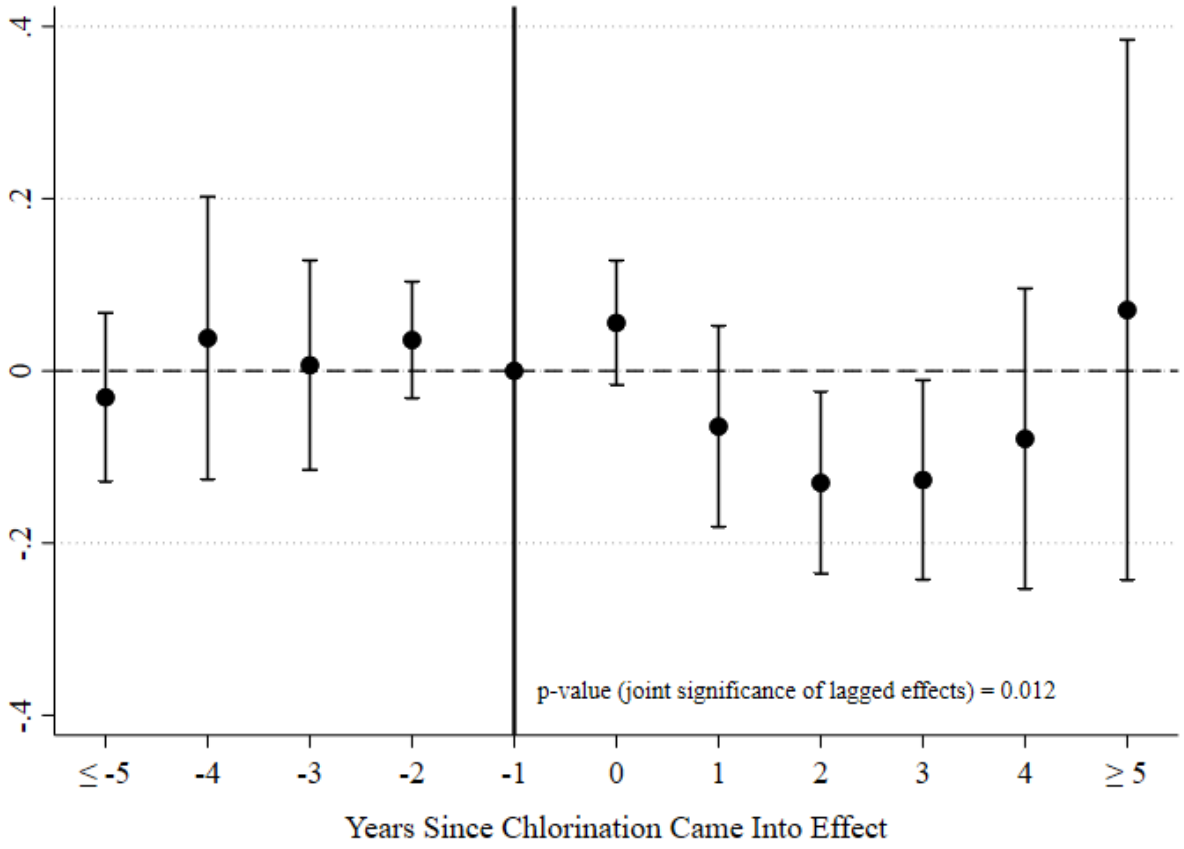
Notes: OLS coefficient estimates (and their 90% confidence intervals) are reported, where the omitted category is 1 year before treatment. The dependent variable is equal to the natural log of the number of black infant deaths per 100,000 of the black population in city *c* and year *t*, and the regressions is weighted by the black city population. Controls include the city-level characteristics listed in Online Appendix Table 2, city fixed effects, year fixed effects and city-specific linear time trends. Standard errors are corrected for clustering at the city level.

Online Appendix Figure 3. Pre- and Post-Chlorination Trends in White 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 white infant deaths per 100,000 of the white population in city *c* and year *t*, and the regressions is weighted by the white city population. Controls include the city-level characteristics listed in Online Appendix Table 2, city fixed effects, year fixed effects and city-specific linear time trends. Standard errors are corrected for clustering at the city level.

Online Appendix Figure 4. Pre- and Post-Chlorination Trends in Black 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 black infant deaths per 100,000 of the black population in city *c* and year *t*, and the regressions is weighted by the black city population. Controls include the city-level characteristics listed in Online Appendix Table 2, city fixed effects, year fixed effects and city-specific linear time trends. Standard errors are corrected for clustering at the city level.

Online Appendix Table 1. Municipal Water Purification Dates and Years of Data Availability

City and state	Year city began filtering water supply	Year city began chlorinating water supply	Years covered for infant mortality counts by race	Years covered for diarrhea/enteritis mortality counts by race
Baltimore, MD	1915	1911	1906-1938	1906-1910, 1912-1937
Boston, MA	…	1928	1908-1922, 1937-1938	1908-1909, 1913-1915, 1918, 1937
Chicago, IL	…	1912	1908-1938	1908-1910, 1912-1937
Cincinnati, OH	1907	1918	1908-1938	1908-1910, 1912-1937
Cleveland, OH	1918	1911	1920-1938	1920-1937
Detroit, MI	1923	1913	1920-1938	1920-1937
Indianapolis, IN	1904	1909	1908-1938	1908-1910, 1912-1937
Kansas City, MO	1928	1911	1906-1938	1906-1910, 1912-1937
Los Angeles, CA	…	1925	1910-1938	1914, 1915, 1918-1937
Louisville, KY	1909	1913	1906-1938	1906-1910, 1912-1937
New Orleans, LA	1909	1915	1906-1938	1906-1910, 1912-1937
New York, NY	…	1911	1908-1938	1908-1910, 1912-1937
Newark, NJ	…	1921	1920-1922, 1930-1938	1930-1937
Philadelphia, PA	1906	1910	1908-1938	1908-1910, 1912-1937
Pittsburgh, PA	1908	1910	1908-1938	1908-1910, 1912-1937
St. Louis, MO	1915	1913	1908-1938	1908-1910, 1912-1937
San Francisco, CA	…	1922	1909-1922, 1930-1938	1909, 1913-1915, 1918, 1930-1937
Seattle, WA	…	1911	1920-1922, 1937-1938	1919, 1937
Washington, D.C.	1905	1923	1906-1938	1906-1910, 1912-1937

Online Appendix Table 2. Descriptive Statistics

	Mean (SD)	Description
Filtration	.608 (.487)	= 1 if city had a water filtration plant, = 0 otherwise
Chlorination	.764 (.420)	= 1 if city chemically treated water supply, = 0 otherwise
Clean Water Project	.218 (.412)	= 1 if city had completed a clean water project, = 0 otherwise
Sewage Treatment/Diversion	.236 (.424)	= 1 if city had a sewage treatment plant or diverted sewage away from drinking water supply, = 0 otherwise
Bacteriological Standard	.734 (.439)	= 1 if city set bacteriological standard for milk supply, = 0 otherwise
TB Test	.623 (.482)	= 1 if city required tuberculin testing of cows, = 0 otherwise
ln(Population)	13.5 (.848)	Natural log of the city population
% Female	.504 (.017)	Percent of city population that was female
% Nonwhite	.111 (.075)	Percent of city population that was nonwhite
% Foreign	.163 (.106)	Percent of city population that was foreign born
% Under 15	.240 (.031)	Percent of city population that was under 15 years of age
% 15 to 44	.529 (.021)	Percent of city population that was 15 to 44 years of age
% 45 and Older	.230 (.035)	Percent of city population that was 45 years of age or older
N	506	

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.

Online Appendix Table 3. Water Purification Efforts and Infant Mortality by Race, 1906-1938

	(1)	(2)	(3)
	ln(*White IMR*)	ln(*Black IMR*)	$\ln\left(\frac{Black\ IMR}{White\ IMR}\right)$
Filtration	-.123*	-.205***	-.106
	(.064)	(.059)	(.106)
	{.155}	{.060}	{.458}
Chlorination	.023	-.119**	-.138**
	(.049)	(.045)	(.061)
	{.666}	{.023}	{.045}
Clean Water Project	.091	-.486	-.584
	(.124)	(.431)	(.509)
	{.594}	{.926}	{.959}
Sewage Treatment/Diversion	-.143**	-.050	.143**
	(.038)	(.071)	(.064)
	{.000}	{.453}	{.016}
Bacteriological Standard	.020	-.084	-.110
	(.043)	(.082)	(.078)
	{.688}	{.449}	{.351}
TB Test	.025	-.040	-.047
	(.030)	(.044)	(.059)
	{.381}	{.368}	{.352}
N	506	506	506
R^2	.976	.891	.719

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

Notes: Based on annual data from *Mortality Statistics* and *Vital Statistics of the United States* for the period 1906-1938, published by the U.S. Bureau of the Census. Each column reports estimates from a separate OLS regression. In columns (1)-(2), the dependent variable is equal to the natural log of the number of race-specific infant deaths per 100,000 of the relevant population in city *c* and year *t*, and the regressions are weighted by the relevant city population. In column (3), the dependent variable is equal to the natural log of the ratio of *Black IMR* to *White IMR*, and the regression is weighted by city population. Controls include the city-level characteristics listed in Online Appendix Table 2, city fixed effects, year fixed effects and city-specific linear time trends. 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.

Online Appendix Table 4. Water Purification Efforts and Diarrheal Mortality Among Children Under the Age of Two by Race, 1906-1937

	(1)	(2)	(3)
	ln(*White Diarrhea*)	ln(*Black Diarrhea*)	$\ln\left(\frac{Black\ Diarrhea}{White\ Diarrhea}\right)$
Filtration	-.157	.096	.294
	(.133)	(.096)	(.177)
	{.342}	{.331}	{.190}
Chlorination	-.014	-.190**	-.256
	(.125)	(.082)	(.158)
	{.930}	{.012}	{.105}
Clean Water Project	.597***	-.113	-.916***
	(.127)	(.173)	(.250)
	{.534}	{.646}	{.632}
Sewage Treatment/Diversion	.025	.148	.081
	(.154)	(.220)	(.154)
	{.901}	{.627}	{.621}
Bacteriological Standard	-.039	.020	.075
	(.079)	(.155)	(.097)
	{.699}	{.917}	{.536}
TB Test	.059	.054	-.009
	(.092)	(.088)	(.094)
	{.545}	{.615}	{.922}
N	446	446	446
R^2	.970	.881	.613

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

Notes: Based on annual data from *Mortality Statistics* for the period 1906-1937, published by the U.S. Census Bureau. Each column represents the results from a separate OLS regression. In columns (1)-(2), the dependent variable is equal to the natural log of the number of race-specific diarrhea/enteritis deaths among children under the age of two per 100,000 of the relevant population in city *c* and year *t*, and the regressions are weighted by the relevant city population. In column (3), the dependent variable is equal to the natural log of the ratio of *Black Diarrhea* to *White Diarrhea*, and the regression is weighted by city population. Controls include the city-level characteristics listed in Online Appendix Table 2, city fixed effects, year fixed effects and city-specific linear time trends. 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.