Water Quality, Morbidity, and Mortality in London, 1906-1926*

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Abstract

I examine the effects of chlorination on typhoid fever morbidity and mortality rates in London during the early decades of the twentieth century using a newly constructed panel data set at the borough-by-quarter-of-year level. A difference-in-differences identification strategy takes advantage of variation in the sources of water supply and the introduction of chlorination across parts of London in 1916. I find that chlorination accounts for 16 to 29 percent of the decline in the typhoid mortality rate during the sample period, with larger effects in the fourth quarter of the year when contaminated river water entered the water supply due to flooding.

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1 Introduction

The provision of sanitation infrastructure is an important means by which public health policies can improve health outcomes of the population. Protection of source water to create clean, safe drinking water remains a challenge in the developing world. In 2012, 748 million people lacked access to a source of improved drinking water, nearly a quarter (173 million) of whom rely on untreated surface water (WHO and UNICEF, 2014). Moreover, an estimated 502,000 diarrhea deaths were caused by inadequate drinking water (Prüss-Ustün *et al.*, 2014). During the twentieth century, the treatment of drinking water with chlorination and filtration all but eradicated waterborne diseases such as cholera, dysentery, and typhoid fever from the developed world.

This paper examines the effects of chlorination and water quality on typhoid fever morbidity and mortality rates in London, England, during the early decades of the twentieth century. Policymakers should be concerned with the effects of chlorination on typhoid fever morbidity and mortality given that the debilitating effects of typhoid fever among survivors can persist into adulthood and the eradication of typhoid has been shown to increase educational attainment (Beach *et al.*, 2014). I construct a new panel data set of water quality, morbidity and mortality measures at the borough-by-quarter-of-year level from London, England, between 1906 and 1926. Detailed historical data on the results of bacteriological tests and variation across the city in exposure to the chlorination of the water supply provide indicators of water quality. In addition, I obtain the number of reported cases of typhoid fever and deaths from typhoid at the borough-by-quarter-of-year level as measures for the prevalence of waterborne diseases. I focus on typhoid fever since the typhoid bacillus (*Salmonella Typhi*) is highly susceptible to chlorination (Banks, 1949), and it is as least as susceptible as the *E. coli* bacteria under most conditions (Ratnayaka *et al.*, 2009, p. 430).

This study makes three contributions to the existing economics and economic history literatures. First, I draw on quarterly data to help explain the considerable seasonal variation in water quality and waterborne disease rates, which is completely obscured in previous re-

search that relies on city-year panel data (Cutler and Miller, 2005; Alsan and Goldin, 2015). Similar to Kesztenbaum and Rosenthal (2014), the focus on a single city can uncover significant differences in water quality and mortality within a city that has been ignored by most existing studies. Second, I collect evidence on direct measures of water quality instead of inferring quality improvements from declining typhoid mortality rates or discrete changes in water supply or sanitation infrastructure across cities. Previous work has typically used typhoid fever mortality rates as a proxy for water quality and has thus been unable to examine the effects of water quality on population health (Beach *et al.*, 2014, 2015).[1] Third, in contrast to the exclusive focus of past studies on mortality rates as an outcome of interest, I also examine effects on morbidity to determine whether exposure to a contaminated water supply had potential scarring effects on survivors.

I use a difference-in-differences identification strategy which takes advantage of variation in the sources of water supply across London, and a policy intervention that introduced chlorination into the water supply in 1916. Boroughs that received water from the polluted Thames, Lee, and New River benefited from the chlorination of the water, whereas boroughs that received water from uncontaminated, deep chalk wells or continued to receive untreated filtered water were not affected. The chlorination of London's water supply has not received much attention from researchers despite representing one of the first instances of a large metropolitan area chlorinating its water supply. Luckin (1986) has suggested that the adoption of chlorination by the Metropolitan Water Board in 1916 may have brought about the final eradication of typhoid fever in London, but the effects of chlorination on mortality rates for waterborne diseases in London has not been tested empirically. I provide additional evidence of the effects of water quality on typhoid fever morbidity and mortality using the percentage of positive tests for the *B. coli* bacteria as a proxy for water quality, and a fixed effects regression strategy that controls for time-invariant borough-specific factors, year and

[1] A recent exception is Antman (2015) who uses the introduction of tea consumption in England during the 18th century as an exogenous change in water quality, and finds that parishes with lower initial water quality experienced larger declines in total mortality and mortality from waterborne diseases following years with large volumes of tea imports.

quarter-of-year fixed effects, as well as borough-specific linear time trends.

The main results from estimating the difference-in-differences model indicate that the chlorination of the London water supply had a substantial effect in reducing typhoid fever mortality. I find that chlorination reduced the typhoid mortality rate by 0.78 to 1.40 deaths per 100,000 persons, which accounts for 16 to 29 percent of the decline in typhoid mortality rate between 1906 and 1926. The effects are largest for chlorination exposure during the fourth quarter of the year – October to December – when flooding increased the risk of raw contaminated river water entering the water supply. During the winter months, the New River water supply was chlorinated to counteract the risk of contaminated water, whereas river flooding interfered with the chlorination of the Thames River supply. The effects of chlorination during the fourth quarter of the year account for 32 to 53 percent of the decline in typhoid mortality during the sample period. Turning now to the the fixed effects regression estimates, the results indicate that a one-standard-deviation decrease in water quality, proxied by the percentage of positive tests for *B. coli* bacteria, is associated with a 0.047 standard-deviation increase in typhoid mortality and morbidity per 100,000 persons. By 1905, typhoid fever had declined to much lower levels than during the nineteenth century, and public health officials had become less concerned about the connection between typhoid and a contaminated water supply (Luckin, 1986, p. 134), but the results suggest that unpurified water was still responsible for a non-trivial share of typhoid mortality.

1.1 Related Literature

This study contributes to the empirical literature on the effects of improved sanitation infrastructure and the historical debate over the causes of the mortality decline in England and Wales during the late-nineteenth and early-twentieth centuries. Mortality rates in England and Wales persisted at high levels throughout the nineteenth century, due to the urban penalty associated with living in crowded and unsanitary urban environments, and a growing share of the population living in cities, before declining in the final quarter of the

century. A prominent debate over the causes of the mortality decline followed the publication of McKeown (1976), who emphasized the role of rising real incomes and argued that improved nutrition strengthened the ability of the body's immune system to resist infection.[2] However, Floud *et al.* (2011) point out that the causality could run in the opposite direction, because infection can drastically limit the body's ability to digest nutrients consumed. Szreter (1988) challenged the McKeown thesis and argued that preventative public health measures implemented by municipal governments, as well as improvements in the quality of the water supply and sewage infrastructure were the decisive factors driving the mortality decline in the absence of coordinated policy from the central government. In more recent work, Kesztenbaum and Rosenthal (2014) find that increases in income and the diffusion of sewer connections both contributed to the decline in mortality and gains in life expectancy across Paris neighborhoods between 1885 and 1913. Another significant cause of mortality was airborne pollution as Hanlon (2015) finds higher mortality for respiratory and cardiovascular conditions in districts exposed to more industrial pollutants across England in the latter-half of the nineteenth century. The results in this paper provide support for the view that public health measures and improved municipal sanitation were among the factors responsible for the decline in mortality during the late-nineteenth and early-twentieth centuries.

Previous literature has exploited the differential timing of water purification and sanitation interventions across municipalities in the United States during the late-nineteenth and early-twentieth centuries to identify effects on mortality rates. Cutler and Miller (2005) examine the adoption of clean water technologies across U.S. cities and find that the joint implementation of filtration and chlorination accounted for 43 percent of the reduction in total mortality, 74 percent of the reduction in infant mortality and 62 percent of the reduction in child mortality between 1900 and 1936. Ferrie and Troesken (2008) focus on the city of Chicago and find that 30 to 50 percent of the reduction in total mortality can be attributed to water purification. In a study of municipalities across Massachusetts, Alsan and Goldin

[2]See Cutler *et al.* (2006) for a review of the determinants of mortality that considers both historical and contemporary evidence. See Harris (2004) for a review of the debate over the McKeown thesis.

(2015) find that the combined effects of clean water sources and effective sewerage systems accounted for 44 percent of the total change in the infant log mortality rate between 1880 and 1915. Similar evidence from late-nineteenth century England has been used to show effects of public health expenditures on mortality rates from waterborne diseases. Chapman (2014) finds that local government expenditures on water supply and sewerage between 1871 and 1890 accounted for 23 to 48 percent of the decline in mortality rates from cholera and diarrhea.[3] This study adds direct measures of water quality and an analysis of the seasonal variation in the effects of chlorination on typhoid fever morbidity and mortality to the existing literature.

Past studies have found mixed evidence on the effects of changes in ownership of waterworks on mortality rates from waterborne diseases. Troesken (1999) shows that private water companies in the U.S. were more likely than public waterworks to utilize filtration technology between 1880 and 1920, but does not find statistically significant evidence that municipalization reduced typhoid mortality rates. However, Troesken (2002) finds that public ownership reduced typhoid mortality rates for blacks by 90 percent. Beach *et al.* (2015) show that the municipalization of waterworks in England and Wales between 1869 and 1910 reduced waterborne disease rates by 12 to 18 percent. In contrast, Galiani *et al.* (2005) find that the privatization of water services in Argentina during the 1990s was associated with an 8 percent reduction in child mortality. Their result suggests that poor quality municipal governance in a developing country setting, in comparison to England and the U.S. during the nineteenth century, is less conducive to municipal ownership of the water supply. The mechanism through which the changes in ownership of waterworks affect mortality rates is not always clear from these studies. Contemporaries argued that publicly owned companies had stronger incentives than private water companies to invest in unprofitable but socially beneficial water filtration systems. However, the municipalization of water companies does not seem to have been motivated by the public health externalities associated with sanita-

[3] Cain and Rotella (2001) also find evidence that expenditures on sanitation infrastructure were associated with declines in mortality using evidence from U.S. cities between 1902 and 1929.

tion systems. Troesken and Geddes (2003) emphasize the view that public acquisition was driven by the absence of a credible commitment mechanism on the part of municipalities to prevent the expropriation of value from private waterworks. They argue that municipalization was efficient because it reduced the costs of enforcing contracts. This study finds that the effects of chlorination are comparable to, and perhaps even larger, than the effects of municipalization.

The development literature has found heterogeneous effects of improvements in water quality on self-reported health by maternal education (Jalan and Ravallion, 2003) and family income (Kumar and Vollmer, 2011), with poor households and less educated mothers being less likely to benefit from sanitation improvements. Larger effects are often found on child than on adult mortality. Recent work has provided evidence from randomized experiments, including Kremer *et al.* (2011) who show that investment in rural spring protection reduces fecal contamination by 66 percent, while diarrhea among children falls by 25 percent.[4] Devoto *et al.* (2011) document improvements in self reported well-being among households treated with a private water connection. Quasi-experimental evidence has been presented by other studies such as Zhang (2011) who finds that introducing village-level access to water in rural China reduced illness among adults by 11 percent and increased their weight-for-height ratio. This paper adds evidence of the effectiveness of chlorination from nineteenth-century London that is potentially relevant to large metropolitan areas in developing countries today. The results suggest that water and sanitation infrastructure improvements may be cost-effective interventions in developing countries today, given the low cost of chlorination and the continued prevalence of waterborne diseases such as diarrhea among the leading causes of infant and child mortality.

The remainder of the paper proceeds as follows. Section 2 provides historical background on the relationship between typhoid fever and contaminated river water, in addition to background on the development of water supply and purification technologies, both generally

[4]See Ahuja *et al.* (2010) for a review of this literature.

and with emphasis on the case of London; Section 3 describes the data sources and the procedure involved with constructing a borough-by-quarter-of-year panel data set; Section 4 lays out the difference-in-differences estimation strategy; Section 5 describes the main results; Section 6 studies the heterogeneity in the effect of chlorination by season, as well as by baseline levels of typhoid mortality and water quality; Section 7 performs robustness checks; and Section 8 concludes.

2 Historical Background

An important milestone in the emergence of the public health reform movement in England was the publication of Edwin Chadwick's *Report on the sanitary condition of the labouring population of Great Britain* in 1842, which presented an argument, radical for the time, that poor health was the cause of poverty (Chadwick, 1965). Chadwick's report called for sanitary reforms such as improvements in the public water supply and waste removal, which along with preventative measures against cholera epidemics in the 1840s and 1850s, focused on the removal of the sources from which diseases were thought to arise, particularly dirt, rotting organic matter, and overcrowded living quarters (Worboys, 2000, pp. 35-7). Chadwick was a supporter of the "miasmatic" theory of disease, popularized in the 1840s and 1850s, which postulated that putrefying organic matter in the air was the source of infectious diseases (Melosi, 2000).[5] During the late-1850s and early-1860s, following John Snow's investigation of the 1854 cholera epidemic that identified a waterborne source of the disease outbreak, a consensus emerged that the transmission of diseases such as cholera, typhoid fever, and diarrhea was water-borne, and that drinking water directly from the river was harmful to one's health. By the early 1880s, a general agreement emerged among water analysts that bacterial organisms were the cause of water-borne diseases.

[5] In the context of waterborne transmission, supporters of the miasmatic theory suggested that polluted rivers propagated diseases, not through the consumption of water, but via vapors rising from the water surface (Luckin, 1986, p. 108).

2.1 Typhoid fever and the contaminated water supply

Typhoid fever is a disease with no vector that spreads by fecal or oral transmission, typically via infected water, milk, or food.[6] The source of the typhoid bacillus (*Salmonella typhi*) can only be found in an infected person, either an existing sufferer or an immune carrier (Luckin, 1984, p. 104).[7] Waterborne typhoid has a long incubation period of 18 to 21 days, while food-borne typhoid can strike within 24 hours and incubate over a period of 7 to 9 days (Hardy, 1993, p. 156). Symptoms of typhoid fever included feeling tired and weak, a loss of appetite, and running a high and sustained fever (Luckin, 1986, p. 118). Diarrhea soon follows, leading to abdominal hemorrhage as the most common cause-of-death (Luckin, 1984, p. 104). The case fatality rate ranged between 10 and 20 percent, while the recovery process lasted up to 4 months. Prior to 1940, treatment was limited to bed rest and a healthy diet (Troesken, 1999, p. 928).

Typhoid fever mortality rates fluctuated considerably throughout the nineteenth century and into the early-twentieth century. Typhoid mortality increased as sanitary conditions in London deteriorated in the early nineteenth century when cesspools and the widespread use of water closets contaminated the Thames River. Water quality improved during the 1850s as the London water companies adopted filtration technology and moved intakes away from sewage outlets in the lower Thames. Although typhoid fever was not clearly distinguished from cases of typhus through the middle of the nineteenth century, it is suspected that typhoid mortality peaked in 1870.[8] Typhoid mortality rates in London declined substantially between 1875 and 1885 following improvements in water supply and drainage. In particular, constant water supply was extended to poorer districts, technological change in water filtration methods improved water quality, and innovations in the flushing mechanisms of water

[6] See Whipple (1908) for the state of knowledge about typhoid fever at the turn of the twentieth century.

[7] Typhoid patients typically gain immunity from the disease, with the exception of 5 percent of cases that experience a recurrence (Hardy, 1993, p. 155). The typhoid bacillus was first discovered by Karl Joseph Eberth in 1880.

[8] The *Annual Reports of the Registrar General* only reported separate data series for the two diseases starting in 1869 (Luckin, 1984).

closets improved domestic sanitation. After a period of stagnation, mortality rates continued to decline after 1900 due to greater emphasis on the immediate hospitalization of patients in isolation facilities, and improved knowledge about the disease transmission process (Hardy, 1993, pp. 153, 157-1).

The connection between typhoid fever and a contaminated water supply was debated throughout the nineteenth century. Until the 1850s, water chemists believed in the self-purifying power of rivers and argued that the dilution of contaminated organic matters would lead to their destruction by oxidation (Goubert, 1989). From the 1860s onward, public health authorities became more aware that filtration and chemicals could be used to treat the water supply and sewage waste, but the water companies denied any connection between poorly treated water and the spread of disease (Luckin, 1986, p. 39). The failure to discover the typhoid bacillus in the contaminated London waters, and the failure to confirm the nature of typhoid by inoculating an animal contributed to the disagreement over the causal agent of the disease (Hamlin, 1990, p. 279). By the 1870s, Medical Officers of Health (MOH) dismissed the possibility of a connection between typhoid fever mortality and a contaminated water supply, following improvements in water storage and filtration technology, but striking differentials in district-level typhoid mortality remained during the 1890s and public health officials again suspected that typhoid was transmitted by the water supply. In 1893, Shirley Foster Murphy found that typhoid mortality rates in London were more strongly correlated with the source of the water supply than with the level of poverty in a district. The MOH of St. George Hanover Square recognized that a typhoid fever outbreak during 1894 followed the delivery of unfiltered water from the Thames River during periods of flood (Luckin, 1986, p. 125-31).[9] Despite these incidents, London continued to rely on the imperfect filtration method to purify its water supply.

[9] A similar incident occurred in Paris at the end of the nineteenth century. Typically, the city's water supply originated from the Vanne and Dhuys Rivers, but when water authorities introduced water from the Seine River to the system, they observed an increase in the incidence of typhoid fever (Goubert, 1989, p. 49).

2.2 Chlorination

Initial scholarly discussions about chlorination preceded the formation of knowledge about the causes of waterborne diseases. The earliest recorded proposal for using chlorine to "destroy the foulness of the fluid" was made in Philadelphia in 1835, and published in *Human Health*, but the taste and smell of the water was believed to be "disagreeable" to most consumers (Baker, 1948, p. 326). Scientists concerned about the taste of chlorinated water did not realize that only a small dose of chlorine was needed to properly treat the water. Between 1840 and 1900, 15 patents were taken out in England for the disinfection and purification of water by adding substances such as a "hypochlorite" or "chlorine-permanganates," but these early patents did not demonstrate an understanding of how water purification would work. Such knowledge was demonstrated by the first American patent for the chlorination of water which was issued in 1888 to Albert Leeds, who proposed to treat water with chlorine gas generated through electrolysis and the use of a hydrochloric acid. After initial experiments by U.S. waterworks during the 1890s, the use of chlorination expanded in 1908 following its adoption on a small scape at Bubbly Creek in Chicago, and on a large scale at the Boonton Reservoir waterworks in Jersey City (Baker, 1948, p. 328-41). Adoption of chlorine continued over the subsequent decades, and by 1925, over 75 percent of municipal waterworks in the U.S. chlorinated their water supplies, a number that increased to 85 percent (4,590 out of 5,372 waterworks) by 1941.

The adoption of large-scale chlorination in England occurred around the same time as in the U.S, but its expansion was much slower. The benefits of chlorination were not appreciated by water analysts such as Percy Frankland who argued up until the early-1890s that filtration could almost guarantee the safety of the water supply (Hamlin, 1990, p. 277). Chlorine was first used to treat a public water supply in England following a typhoid outbreak in 1897 at Maidstone, where bleaching powder was applied as an emergency measure (Thresh *et al.*, 1943, p. 632). Following an outbreak of typhoid at Lincoln in December 1904 and lasting until 1911, an alkaline solution of sodium hypochlorite (Chloros) was added to the water supply

prior to slow sand filtration, which represented the first instance of the continuous treatment of a municipal water supply in England.[10] The first cases of installing a chlorine plant to provide routine treatment occurred at Reading in 1910 (Thresh *et al.*, 1943, p. 632) and at the Cheltenham waterworks in 1911. (Baker, 1948, p. 342). One of the officials involved with the chlorination at Lincoln, A.C. Houston, would later become the Metropolitan Water Examiner and lead experiments in 1916 that introduced chlorination to the London water supply.[11]

Chlorination would likely have been implemented in England earlier, and may have spread more rapidly, had it not been for the opposition of public health officials and the skepticism of consumers. In 1904, William Boby delivered chlorinating apparatus to the Christchurch waterworks, but the local Medical Officer of Health would not allow the use of chlorine treatment, despite knowledge that the water was contaminated. Boby attributed the decision by the MOH to a lack of education about the most recent advances in water science (Baker, 1948, p. 336). Many London consumers objected to drinking chlorinated water – which they referred to as "doped" water – due to its unpleasant taste and a belief that chlorination was a shortcut to the purification of the water supply (Metropolitan Water Board, 1922, p. 17).

2.3 The chlorination of the London water supply

London's water supply originated from multiple sources, generating useful variation in water quality across the metropolis. Throughout the nineteenth century, eight private companies supplied the city with water from the River Thames, the River Lea, the man-made New River canal, and deep chalk wells in the Kent district. Figure 1 shows the location of the chlorination plant that treated water supplied from the Thames River, after passing through

[10] The mechanism used to chlorinate the water was very crude, as the Chloros was simply stored in stone jars and added to the water supply at a constant rate (Metropolitan Water Board, 1953, p. 254).

[11] Houston also learned about the effectiveness of chlorination from its adoption in the United States, in particular, the treatment of the Bubbly Creek water with chlorine of lime at Chicago in 1908 (Metropolitan Water Board, 1916). Houston chose to chlorinate the water supply in London using bleaching powder, which had more available chlorine per unit of weight than Chloros, due to the large volume of water that entered the water supply – over 75 million gallons per day (Metropolitan Water Board, 1953, p. 255).

the Staines storage reservoirs and on its way down the Staines Aqueduct, before it entered the Sunbury, Kempton Park, Grand Junction and West Middlesex waterworks. The initial chlorination experiments began at Staines on May 1, 1916 and treatment expanded gradually over the next year. The intakes for water supplied to the Chelsea, Lambeth, and Southwark and Vauxhall waterworks were located further east on the Thames River. Chlorination was not applied at these waterworks due to a lack of storage reservoir capacity.

Since the chlorine was applied prior to filtration, the effectiveness of the treatment varied across London due to differences in the storage capacity at each of the waterworks. Water could still become polluted post-chlorination since it was held in open reservoirs exposed to surface runoff, airborne pollutants and bird droppings (Metropolitan Water Board, 1917, p. 27). Waterworks with larger storage capacity, such as West Middlesex and Kempton Park, which supplied the New River district, faced a greater risk of deterioration in water quality post-chlorination due bacterial growths that could develop with a longer period of storage. Prior to the introduction of chlorination, these districts would have had a less polluted water supply in comparison to waterworks with limited storage capacity, such as the Grand Junction and East London's Sunbury works, as the larger storage capacity reduced the need to send raw, unfiltered river water into the supply during periods of peak demand and flooding. The Director of Water Examination, A.C. Houston, recognized that "chlorinated water would filter best if not stored too long subsequent to chlorination," but no steps were taken the remedy the deficiencies of waterworks with large storage reservoirs (Metropolitan Water Board, 1918, p. 9). Thus, chlorination was less likely to be effective in areas of London that previously had better water quality, which would work against finding a result.

Water quality and the implementation of chlorine treatment also varied seasonally. Water quality deteriorated during the winter months due to increased rates of filtration and pollution from water runoff following heavy rainfall and flooding.[12] Chlorination was also

[12]Filtration rates increased in winter months to keep up with higher demand as the risk of burst pipes during periods of extreme cold meant that households kept their taps running (Metropolitan Water Board,

less effective in cold weather, because the chemical process required a longer contact period, but the Metropolitan Water Board was not willing to increase the dosage of chlorine due to concerns about the unpleasant taste of the water. The New River district received a large proportion of its water from wells, which was believed to be of higher purity than river water, and therefore did not chlorinate its water supply year around, relying instead on filtration technology. However, the quality of the water supply deteriorated rapidly during the winter months, as limited storage capacity and filtration rates double those found at other London waterworks could not prevent contaminated flood water from entering the system. In November 1919, the Metropolitan Water Board began the chlorinate the water supply of the New River district. Figure 2 shows the location where chlorine was applied to water supplied by the man-made New River canal, as well as the River Lee, which provided water to the East London district and was not chlorinated. Although the treatment of the New River water supply was mostly limited to the winter months from November to January, water supplied from the Thames River was not chlorinated consistently during the winter months as flooding made the water too turbid for filtration and waterworks resorted to using stored water that had not been chlorinated (Metropolitan Water Board, 1918, p. 5). Given the higher risk of contaminated river water entering the municipal water supply during the winter months, we may expect larger effects of chlorination at the beginning and end of the year.

The introduction of chlorination in London in 1916 was motivated by war-time cost savings, and not by the promise of improved protection against waterborne diseases.[13] The existing water purification technology of filtration was very costly since it required significant expenditures of coal to pump water into storage reservoirs. Chlorination represented an alternative purification method that reduced expenditures on coal by £3,150 per year

1919, p. 2).

[13]Other early examples of chlorination treatment can also be found at wartime. The British military used chlorine to treat all water supplies in the field during World War I (Thresh *et al.*, 1943, p. 633), while Philippe Bunau-Verilla provided chlorinated water to French troops at the Battle of Verdun in 1916 (Baker, 1948, p. 343).

(£826,800 in 2014).[14] Moreover, the Director of Water Examination, A.C. Houston, viewed it as a substitute for storage when he described that the "object is to avoid pumping charges and to render river water as pure after chlorination as if it had been stored in the Staines reservoir" (Metropolitan Water Board, 1918, p. 12). That chlorination could provide a more effective standard of purification than relying on filtration alone was not emphasized. The use of chlorination was also promoted as a means to postpone, and eventually abandon, plans for costly construction of expanded storage reservoirs at the New River waterworks (Metropolitan Water Board, 1922, p. 17). Thus, the initial timing of chlorination was arguably unrelated to current rates of waterborne diseases. Later, in Section 7, I provide evidence that chlorination was not implemented as an immediate response to high rates of typhoid mortality in specific boroughs of London.

At the time chlorination was first introduced in England, water experts knew very little about the mechanism of how the process of chlorination purified the water supply. The knowledge of workers at the Lincoln waterworks in 1905 was limited to a basic understanding that adding very small doses of chlorine to the water supply eliminated bacteria (Metropolitan Water Board, 1953, pp. 254-5). By the 1920s, water officials understood in general terms what factors determined the effectiveness of chlorine in sterilizing water. Officials were aware that exposure to a sufficient concentration of chlorine for an adequate contact time was needed to destroy bacteria, that water should be "reasonably clear and bright," and that impurities suspended in the water impeded chlorination by absorbing free chlorine and protecting bacteria (Thresh *et al.*, 1943, pp. 636-7).[15] The method of chlorination used in London was known as "pre-chlorination," given that the treatment was applied prior to filtration, and was chosen as a cost saving measure, despite knowledge that steril-

[14]This estimate is based on a war-time cost of coal of 13 shillings to pump one million gallons and an average daily supply of 75 million gallons of water (Metropolitan Water Board, 1916, p. 19)

[15]Current World Health Organization guidelines for chlorination of fecally polluted source water recommend a maximum turbidity of 1 nephelometric turbidity unit (NTU), a pH level less than 8.0, a minimum contact time of 30 minutes, and at least 0.5 mg/L of free chlorine during the entire contact period (WHO, 2011).

ization of water after filtration was more effective.[16] Houston experimented with the latter approach, but abandoned the efforts after objecting to the taste of the water with the presence of chlorine (Metropolitan Water Board, 1953, p. 256). Houston's trial suggests that it was within the realm of possibility for London to have developed more effective chlorination procedures during the 1920s had the Metropolitan Water Board gone further in investigating the benefits of super-chlorination.[17]

3 Data Sources

Borough-level data on cause-specific deaths, notifications of infectious disease cases, and births from 1906 to 1926 are obtained from the Quarterly Returns of the Registrar General (General Register Office, 1906-1926).[18] Figure 3 shows the boundaries of the twenty nine Metropolitan Boroughs that comprise the County of London and represent the geographic units of analysis. In addition to total mortality, deaths from typhoid (enteric) fever, measles, scarlet fever, whooping cough, and diphtheria are reported consistently throughout the sample period. Although these causes of death are not disaggregated by age group, the mortality data also contain deaths of infants (under 1 year of age), and deaths of individuals under 2 years of age from diarrhea and enteritis, as separate categories that are useful for examining the effects of water quality on early-life health.[19] The number of notified cases of infectious disease provide a measure of morbidity, and these are reported separately for cases of typhoid fever, scarlet fever, and diphtheria.

[16] Pre-chlorination of raw water that contains organic matter is now discouraged due to the formation of disinfection byproducts (DBP) that are possible human carcinogens (Ratnayaka *et al.*, 2009, p. 432).

[17] The super-chlorination method involved adding a dose of chlorine in excess of what was needed for disinfection, then dechlorinating it. The MWB successfully adopted the treatment in London after 1939 (Metropolitan Water Board, 1953, pp. 259-61).

[18] The Registrar General produced the Weekly (and Quarterly) Report of Births and Deaths in London starting in 1837. Following the passage of the Infectious Disease Notification Act on 1889, Medical Officers of Health produced weekly infectious disease notification reports, which include cases of infectious disease by registration district. Reporting was mandatory for general practitioners but not for hospitals.

[19] Smallpox is another category that is reported, but there are no deaths due to smallpox in most quarters-of-the-year. The reported deaths are limited to individuals in the civilian population, exclude non-residents of the County of London, and are adjusted to include deaths of London residents occurring in neighboring districts. Births are assigned to boroughs based on the place of registration.

The main specifications use typhoid fever as the primary measure of waterborne diseases. Mortality and morbidity rates for typhoid fever are computed as the number of deaths or reported cases in a borough-quarter per 100,000 persons. The quarterly mortality rates are scaled by a factor of four to be expressed in annual terms. Annual population figures are reported in the Quarterly Returns and are linearly interpolated from census-year data. Infant mortality (ages 0 to 1) and deaths due to diarrhea (ages 0 to 2) are expressed as rates per 1,000 live births.

The *Monthly Reports of the Water Examiner* contain statistics for bacteriological tests that measure the quality of the London water supply (Metropolitan Water Board, 1906-1921). These results provide an initial indication of how water quality varied across districts in London and over time. The primary measure of interest is the percentage of water samples that tested positive for the *B. coli* bacteria, a harmless bacteria which survived longer than most pathogens and was used as an indicator for the presence of bacteria causing waterborne diseases (Thresh *et al.*, 1943, p. 415). Given that the typhoid bacillus was more easily killed than the *B. coli* bacteria, it was assumed that the typhoid bacillus was absent from the water if a negative test result for the presence *B. coli* was obtained (Metropolitan Water Board, 1922, p. 16).[20] A second reported measure is the number of microbes contained in a sample of water, but given that the vast majority are harmless, microbe counts do not provide a very meaningful measure of water quality. Tests for water quality were conducted after filtration of the water supply at the waterworks of the eight private water companies that supplied water to "Water London." Figure 4 shows the district boundaries of the eight companies in 1903, immediately prior to the municipalization of the water supply by the London County Council. I use these boundaries to determine the areas of London exposed to the chlorine treatment and the water quality measures, and assume they stay constant throughout the sample period. In cases where multiple test locations were available for a district, such as

[20]The principle of *B. coli* test is similar to a standard test used today for routine monitoring of a water source in which the absence of *E. coli* bacteria in a 100 mL sample signals the absence of pathogenic bacteria (Ratnayaka *et al.*, 2009, p. 433).

the Grand Junction district which had waterworks at Hampton and Kew, the average test result was used. Monthly values were averaged over the quarter of year, ignoring months with missing values, to obtain a district-by-quarter panel.

The empirical strategy relies on knowing the precise timing of chlorination and the areas of London supplied with the treated water. The *Annual Reports of the Water Examiner*, published by the Metropolitan Water Board, indicate the number of days in an administrative year – which ran from April 1st to March 31th of the following year – that water from the Thames River that was treated with chlorine as it passed through the Staines Aqueduct.[21] I collect data on chlorination from its implementation in June 1916 through the end of 1926. For each district I compute the fraction of days during a quarter of year that it received chlorinated water as a proxy for exposure to the treatment.[22] For the New River water supply, the report indicates the exact dates during which chlorine was applied. The treatment variable is constructed as a continuous measure of exposure to chlorination proxied by the fraction of days in a quarter that a water district received chlorinated water. In the main specifications, I compute the quarterly exposure to the chlorine treatment using the more precise information on timing for the New River supply. I also show the robustness of the results to assigning chlorination exposure based on the number of days of treatment during an administrative year for all sources of supply.

The empirical strategy requires mapping measures of water quality reported monthly at different locations throughout London, in addition to the timing and geographic variation in exposure to chlorination, to mortality and morbidity rates reported quarterly at the borough-level. The bacteriological tests that provide a measure of water quality were conducted at the waterworks for each of the water districts. Similarly, information on the timing of chlorination was obtained for each water district. In order to assign water quality and

[21] The duration of treatment at the West Middlesex works differed from the rest of the Thames (Staines) supply in some years.

[22] In some years, the reports only indicate the fraction of the water supply that was treated during the administrative year. I show that the results are not sensitive to using either measure to define the exposure to the chlorine treatment.

chlorination exposure to each Metropolitan Borough, I geo-referenced a 1903 map showing the water supply boundaries of the Metropolitan Water Companies (Figure 4) and computed the fraction of area in a borough that overlapped with each water district. Monthly data on water quality by location was averaged to obtained quarterly averages by water district. Then, each borough was assigned the average water quality and chlorination exposure across water districts, weighted by the fraction of overlapping area.

4 Empirical Strategy

Before turning to the chlorination of the London water supply, I examine whether measures of water quality are associated with typhoid fever mortality and morbidity rates. In particular, I estimate the following equation:

$$(TMR)_{iyq} = \alpha + \beta \cdot (Water\ contamination)_{iyq} + \delta_i + \gamma_y + \mu_q + \delta_i \times t + \epsilon_{iyq} \qquad (1)$$

where i denotes borough, y denotes year, and q denotes quarter. $(TMR)_{iyq}$ is the mortality (morbidity) rate for typhoid fever per 100,000 persons, and $(Water\ contamination)_{iyq}$ is a continuous variable that measures the fraction of samples that test positive for the *B. coli* bacteria in borough i during year y and quarter q. I control for borough (δ_i), year (γ_y), and quarter (μ_q) fixed effects, in addition to borough-specific linear time trends ($\delta_i \times t$). Standard errors are clustered by borough.

The main empirical strategy exploits the plausibly exogenous timing of the introduction of chlorination and the differential exposure across the County of London. As shown in Figure 5, the chlorination of the water supply was restricted to boroughs north of the Thames River. Within the treatment area, exposure varied considerably from less than half of the water supply chlorinated in the eastern boroughs to over eighty percent of the water supply chlorinated in the western boroughs.

I use a difference-in-differences (D-in-D) identification strategy to estimate the effects of

exposure to chlorination on typhoid morbidity and mortality rates. Specifically, I estimate:

$$(TMR)_{iyq} = \alpha + \beta \cdot (Chlorination)_{iyq} + \delta_i + \gamma_y + \mu_q + \delta_i \times t + \epsilon_{iyq} \tag{2}$$

where $(Chlorination)_{iyq}$ is a continuous variable that measures the fraction of time that borough i was exposed to the chlorine treatment during year y and quarter q. The D-in-D coefficient of interest is β, which estimates the effect of the chlorination treatment on the typhoid mortality or morbidity rate $(TMR)_{iyq}$. The remainder of the specification is identical to Equation 1.

The availability of quarterly data also allows me to examine the seasonal variation in the effects of chlorination on typhoid rates. During the nineteenth century, the incidence of typhoid fever was highest during the summer and fall months of August to October (Luckin, 1984, p. 104), but after the turn-of-the-century the seasonal pattern of the disease changed, as the highest mortality rates shifted to the winter months of November and December (Hardy, 1993, p. 181). Much of the increase in incidence during the winter was attributed to river flooding, because existing filtration technology could not handle faster rates of water flow and more unfiltered water flowed through the system. Figure 8 shows that the new seasonal pattern continued to hold during the sample period from 1906 to 1925, with the highest typhoid mortality rates observed during the fourth quarter of the year (October to December), at 3.5 deaths per 100,000 persons compared with only 1.8 during the second quarter, while the positive tests for *B. coli* bacteria are highest between December and February.[23] The seasonal pattern for the number of microbes differs with the largest measurements taken during the summer months from May to September. The high values in the summer reflect algae blooms unrelated to waterborne diseases and thus it is not surprising that the microbe counts do not correlate with typhoid mortality or morbidity. To examine the seasonality in

[23]The pattern is similar for typhoid morbidity rates with a high of 2.0 deaths per 100,000 persons during the fourth quarter compared with a low of 1.2 during the second quarter.

the effects of chlorination, I estimate:

$$(TMR)_{iyq} = \alpha + \sum_{j=1}^{4} \beta_j \cdot (Chlorination \times q_j)_{iyq} + \delta_i + \gamma_y + \mu_q + \delta_i \times t + \epsilon_{iyq} \tag{3}$$

where β_j is the coefficient on the interaction of $(Chlorination)_{iyq}$ with an indicator for quarter-of-year j. Given the presence of more contaminated river water during the winter, I expect larger effects of chlorination during the 1st and 4th quarter of the year.

5 Results

I begin by examining the association between typhoid fever and water quality using borough-by-quarter-level panel data from London. Panel A of Table 2 presents the results from estimating Equation 1 in which I regress the typhoid morbidity and mortality rates on water quality, proxied by the percentage of water samples that tested positive for *B. coli* bacteria. In columns (1) and (2), the dependent variable is the annualized mortality rate for typhoid fever per 100,000 persons, while columns (3) and (4) replace the dependent variable with morbidity rate. In my preferred specifications, found in columns (2) and (4), I control for borough-specific time trends, in addition to year, month, and borough fixed effects.

I find that a one-standard-deviation increase in the fraction of positive tests for *B. coli* bacteria (3.8 percent) – in other words, a decrease in water quality – is associated with a 0.047 standard deviation (0.18 deaths) increase in typhoid mortality per 100,000 persons. A one-standard-deviation change in water quality is also associated with a 0.047 (0.84 reported cases) standard-deviation increase in typhoid morbidity per 100,000 persons. The magnitudes of these effects correspond to about one-quarter to one-third of the effects of increases in sanitary expenditure on the mortality rate for cholera, diarrhea and dysentery found by Chapman (2014) for England between 1872 and 1900.[24] It is not surprising that the effects

[24]Here I compare the 0.047 standard deviation effect of water quality to the 0.15 to 0.20 standard deviation effect of sanitary expenditures found by Chapman (2014).

of water contamination are smaller, given that the changes in the percentage of positive tests for *B. coli* bacteria likely represent very small changes in water quality.

The correlation between typhoid fever and water quality can be seen in Figure 6, which shows borough-level averages of the typhoid morbidity and mortality rates, calculated over the sample period from 1906 to 1926. Figure 6 also shows the percentage of positive tests for *B. coli* bacteria and the number of microbes. There is substantial geographic variation in all measures across the boroughs of London. For example, the typhoid mortality rate ranges from a low of 1.8 to a high of 6.3 deaths per 100,000 persons, while the share of positive tests for *B. coli* bacteria range from 1.4 to 6.3 percent. For both measures, the unhealthy conditions are located in the poorer boroughs in East London, while the healthy conditions are generally found in the wealthier boroughs of West London.

I now turn to the main difference-in-differences regression results. I estimate Equation 2, which compares boroughs exposed to varying rates of chlorination to boroughs supplied with non-chlorinated water, before and after the introduction of the treatment in 1916. The results are presented in Table 3 which follows the same structure as Table 2. In Panel A, the treatment of interest is a continuous variable measuring the fraction of the water supply treated with chlorine, which varies at the borough-by-quarter level. Moving from a borough with no exposure to chlorination to a borough with full exposure to the treatment reduced the typhoid mortality rate by 1.40 deaths per 100,000 persons.[25] At the average level of exposure to chlorination among treated boroughs (55.7 percent), chlorination reduced the typhoid mortality rate by 0.78 deaths per 100,000 persons.[26] Chlorination is also associated with a reduction in the typhoid mortality rate by 0.44 reported cases per 100,000 persons, but this effect is not statistically significant.

[25] A one standard deviation increase in exposure to chlorination (30 percent) is associated with a 0.11 standard deviation (0.42 deaths per 100,000 persons) reduction in typhoid fever mortality.

[26] The average exposure of boroughs that received at least some chlorination is calculated from the initiation of the treatment in 1916q2 through the end of 1926. Figure 9 plots the coefficients on chlorination exposure interacted with year of treatment dummies (1916 to 1926) from a regression with the typhoid fever mortality rate as the dependent variable. Given that the coefficients are only statistically significant at the 5-percent level in 1919 and 1923, and of similar magnitude for the two years, the plot does not conclusively show whether the effect of chlorination increased or decreased over time.

A comparison of the estimated effect of chlorination to the overall decline in typhoid mortality during the sample period can put the magnitude of the effect into perspective. Figure 7a plots the time trend in the typhoid mortality rate per 100,000 persons from 1906 to 1926, averaged over the London boroughs. During the sample period, the typhoid mortality rate in London declined from 5.51 to 0.61 deaths per 100,000 persons, or 89 percent. The effect of full exposure to chlorination accounts for 28.6 percent (1.40/4.90) of the decline in typhoid mortality during the sample period, while the average exposure to chlorination (55.7 percent) accounts for 15.9 percent (0.78/4.90) of the decline. The magnitudes of these chlorination effects are comparable to the 26 percent (25%/96%) joint effect of chlorination and filtration on typhoid fever mortality found by Cutler and Miller (2005), with the caveat that I do not observe differences in filtration rates.[27] Whereas the results in Cutler and Miller (2005) appear to be driven by improvements in filtration technology, the results in this paper potentially point to a greater independent role of chlorination in reducing typhoid mortality rates. The chlorination effect is economically meaningful, and similar in magnitude to the 12 to 18 percent effect of the municipalization of waterworks found by Beach *et al.* (2015) for England during the second half of the nineteenth century.

6 Heterogeneity

While the overall effect of chlorination appears to be large, it may obscure significant differences in the effects by season. The descriptive evidence of seasonality in typhoid fever mortality and measures of water quality motivates the estimation of Equation 3, in which the chlorination treatment variable is interacted with quarter-of-year dummy variables. The results are shown in Panel B of Tables 2 and 3. In both cases, I find large and statistically significant effects of water quality during the fourth quarter of the year (October to

[27] The 26 percent joint effect on typhoid fever mortality is not reported in the text by Cutler and Miller (2005), and is found in column (1) of Table 5 in their paper. In a future revision to this paper, I will be able to estimate joint effects of chlorination and filtration by incorporating information on the quality of filtration such as the capacity of storage reservoirs and the number of acres of filter beds cleaned.

December) on typhoid fever mortality, even larger than the overall effects reported above. In particular, in the preferred specification of column (2), a one-standard-deviation increase in the percentage of positive tests for *B. coli* bacteria is associated with a 0.13 standard-deviation increase in typhoid fever mortality (0.49 deaths). Full exposure to chlorination reduced typhoid fever mortality by 2.61 deaths per 100,000 persons, while the average exposure to chlorination during the fourth quarter (60 percent) is associated with a reduction in the typhoid mortality rate by 1.57 deaths, which account for 53.3 percent (2.61/4.90) and 32 percent (1.57/4.90), respectively, of the decline in typhoid fever mortality during the sample period. These results suggest that chlorination had its largest impact during the winter, when the increased the water flow associated with river flooding forced water officials to supply Londoners with unfiltered river water from the Thames River. On the other hand, the quality of the water supplied from the New River deteriorated even more, but chlorine treatment was introduced during the winter months, potentially averting a higher rate of mortality due to typhoid fever.

The estimates in the main specification do not allow the effect of chlorination to vary with baseline typhoid mortality rates. In Panel A of Table 4 the variable of interest is an interaction between exposure to chlorination and the baseline typhoid mortality rate. In column (1), data from 1906 is used to calculate baseline mortality and data from 1907 to 1926 is used in the estimation sample. Moving from columns (2) to (5), an additional year of data is used in each subsequent column to calculate baseline mortality and excluded from the estimation sample. In column (3), the coefficient on the interaction term implies that at the mean chlorination exposure (55.7 percent), boroughs with an average baseline typhoid mortality rate of 4.86 deaths per 100,000 persons would experience a reduction in typhoid mortality by 0.57 deaths (or 11.6 percent of the sample period mortality decline) relative to boroughs with no typhoid mortality and no exposure to chlorination. In Panel B, I include the overall effect of chlorination in addition to the interaction term and find that the latter is not statistically significant. Thus, there is no evidence that the effect of chlorination is

larger in boroughs that had higher levels of typhoid mortality. This result is consistent with anecdotal evidence that the incidence of typhoid fever was equally common among the poor and the aristocracy (Hardy, 1993, p. 151).

The effect of chlorination may also depend upon the initial level of water quality in a borough. In Panel A of Table 5, the interaction of exposure to chlorination and the baseline level of water quality – proxied by the percentage of positive tests for *B. coli* bacteria – is not statistically significant. However, when the independent effect of chlorination is included as a regressor in addition to the interaction term, as shown in Panel B, I find that the effect of chlorination varied significantly with baseline water quality.[28] The estimates from column (3) imply that chlorination at the average treatment exposure in a borough with average baseline water quality (5.24 percent positive tests for *B. coli*) reduced the typhoid mortality rate by 0.09 deaths per 100,000 persons. The positive coefficient on the interaction term indicates that chlorination was less effective in boroughs with poorer water quality.

7 Robustness checks

A potential threat to identification is that chlorination of the London water supply was implemented in a way that prioritized specific boroughs in response to high rates of typhoid mortality. If this was the case, then the estimates would capture the process of mean reversion following high mortality rates prior to the intervention. I address this concern by including lagged dependent variables in the regression. Figure 10 plots the coefficient on exposure to chlorination from separate regressions which include between 1 to 20 lags of the typhoid mortality rate. The magnitude of the chlorination effect is very stable across the specifications, ranging from 1.19 to 1.43 deaths per 100,000 persons. This finding suggests that the main result cannot be explained by the introduction of chlorination that targeted boroughs with high typhoid mortality in prior years. Moreover, chlorination was introduced

[28] The linear combination of exposure to chlorination and the interaction term is statistically significant in all specifications.

as a war-time cost saving measure that eliminated costly expenditures on coal needed to pump water into storage reservoirs, indicating that the health of the population was not a primary concern behind the implementation decision.

Table 6 presents results from a series of placebo regressions in which I examine the effects of chlorination on non-typhoid mortality and morbidity rates. The results are mixed as chlorination is associated with lower rates of diphtheria mortality and scarlet fever morbidity. These results could indicate the presence of the Mills-Reincke phenomenon, in which water quality is correlated with rates of non-waterborne diseases (Ferrie and Troesken, 2008). Given that typhoid fever has a low case fatality rate, typhoid victims who survived the illness may have experienced a scarring effect that left them vulnerable to other infectious diseases. Chlorination is also associated with higher rates of measles mortality and overall infant mortality, indicating that the intervention may not have been implemented randomly and may be correlated with other factors that affect the rates of non-typhoid mortality. On the other hand, the effect of *B. coli* tests on non-typhoid infectious diseases is essentially zero in all specifications with the exception of tuberculosis rates which increase as water quality deteriorates. This suggests that that the percentage of positive tests for the *B. coli* bacteria represents a good proxy for water quality and does not pick up the effect of an unobserved factor.

8 Conclusion

This paper has examined the effect of chlorination of the water supply on typhoid mortality and morbidity rates in London between 1906 and 1926. Previous literature has emphasized the role of improvements in water supply infrastructure and filtration technology in contributing to the decline in mortality during the second half of the nineteenth century. In comparison, the early decades of the twentieth century had relatively lower typhoid mortality, but chlorination still had large effects on the rate of typhoid mortality. Exposure to

chlorination accounted for 16 to 29 percent of the reduction in typhoid mortality during the sample period. The effect is largest and driven by exposure to chlorination during the winter months, a fact that has been missed by previous studies that have analyzed annual-level and cross-city data sets. The result suggests the need for policy to focus on interventions that purify water supplies during periods of weather-related deterioration in water quality.

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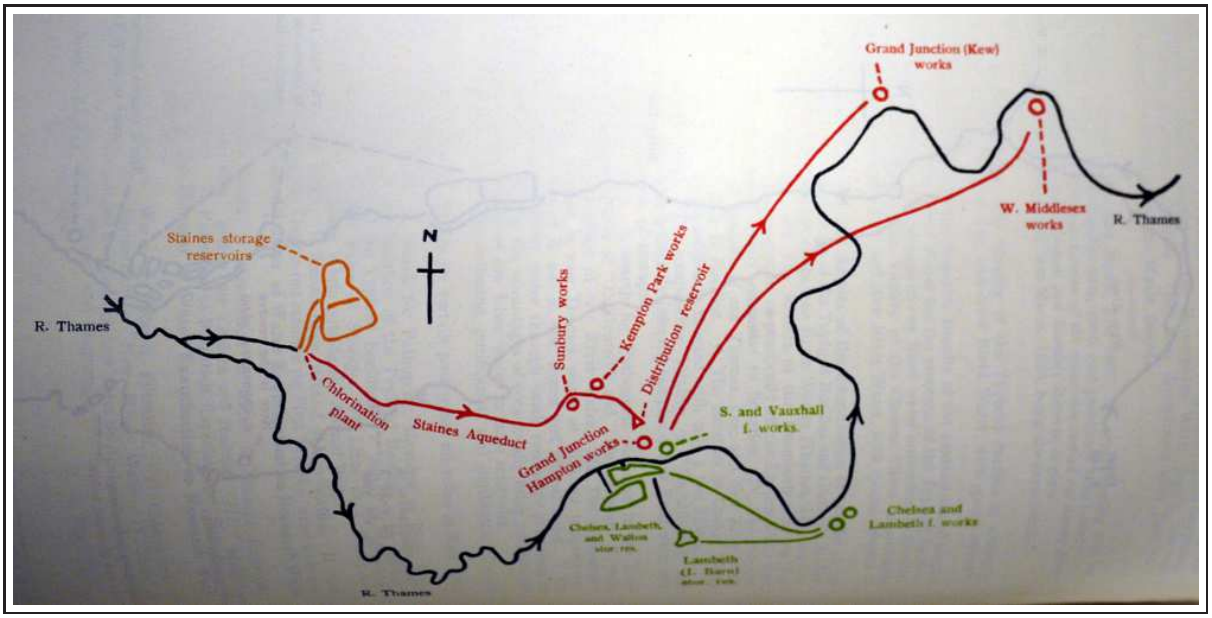
Figures

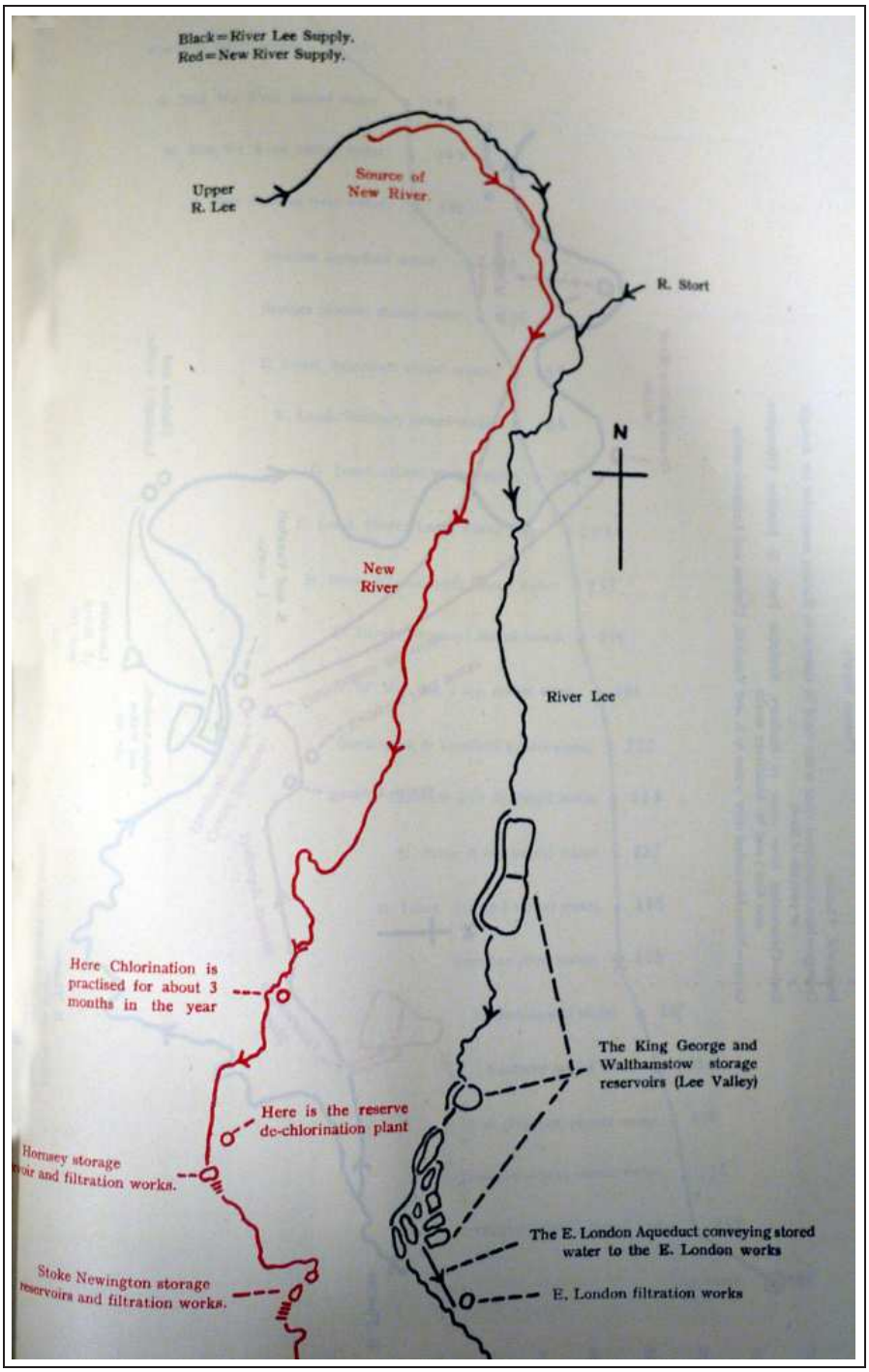
Figure 1: Chlorination of River Thames water supply



Notes: Figure 1 shows the River Thames (black line), the Staines reservoir that holds raw river water for periods of drought or flood (orange line), the Staines Aqueduct that transports chlorinated river water to the Sunbury, Kempton Park, Grand Junction (Hampton and Kew), and West Middlesex waterworks (red line), as well as the non-chlorinated river water that supplies the Southwark & Vauxhall, Chelsea, and Lambeth waterworks (green line).

Source: Metropolitan Water Board (1923), "Report on the results of the Chemical and Bacteriological Examination of the London Waters for the Twelve Months ended 31st March, 1923" ACC/2558/MW/W/01/004. London Metropolitan Archives.

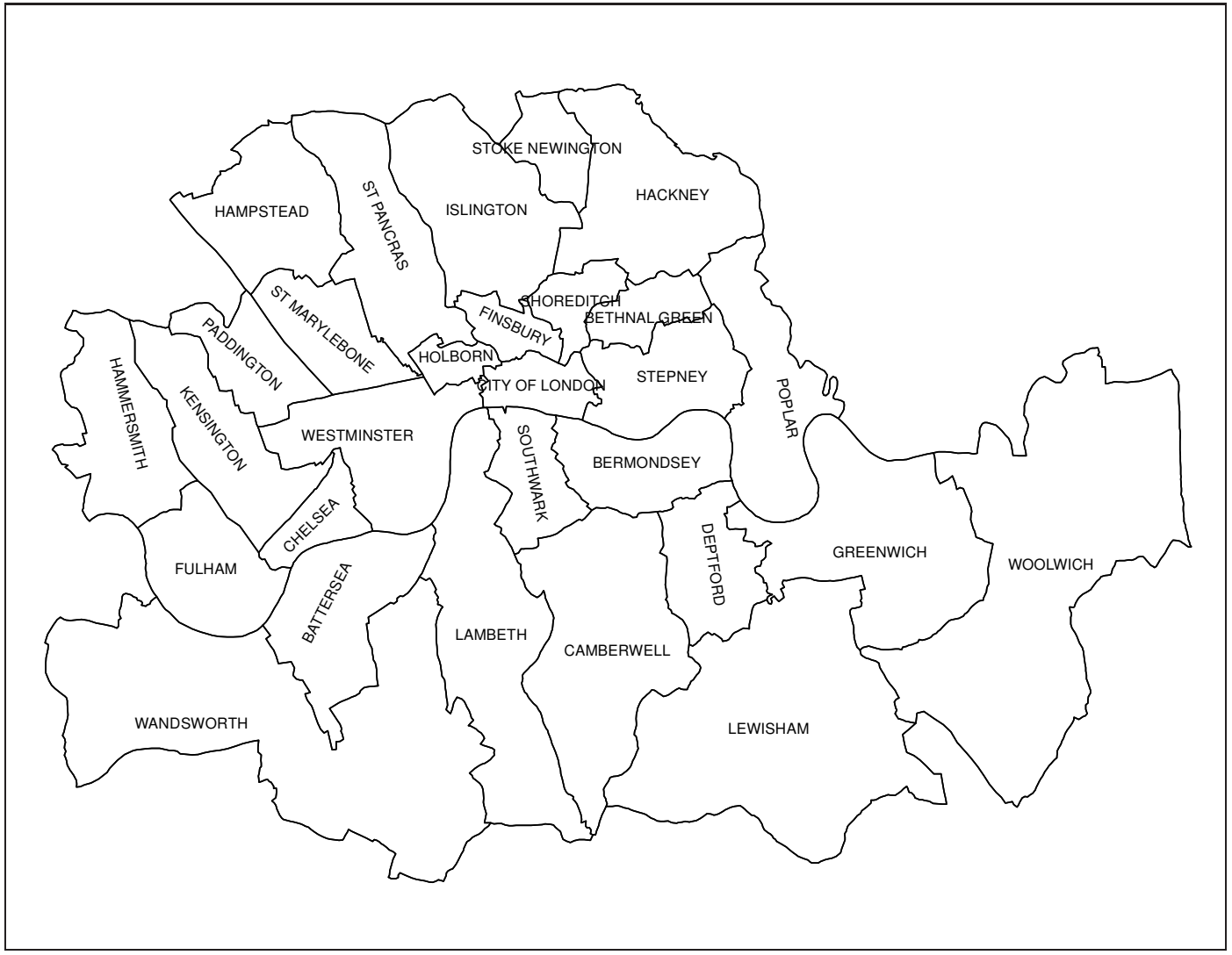
Figure 2: Chlorination of New River water supply



Notes: Figure 2 shows the non-chlorinated River Lee (black line) water supplied to the East London district and the chlorinated New River water supply (red line).

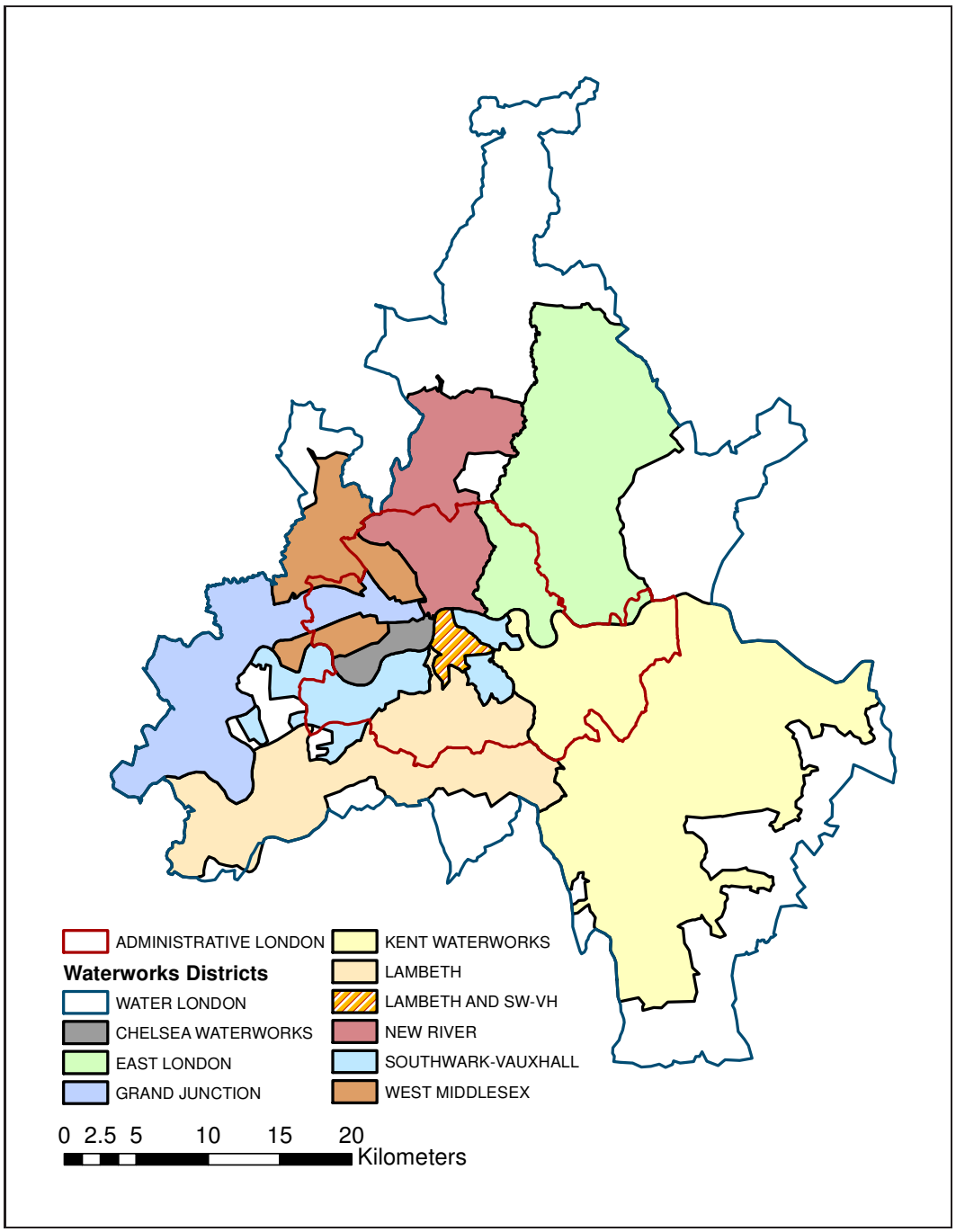
Source: Metropolitan Water Board (1923), "Report on the results of the Chemical and Bacteriological Examination of the London Waters for the Twelve Months ended 31st March, 1923" ACC/2558/MW/W/01/004. London Metropolitan Archives.

Figure 3: Metropolitan Borough boundaries in the County of London



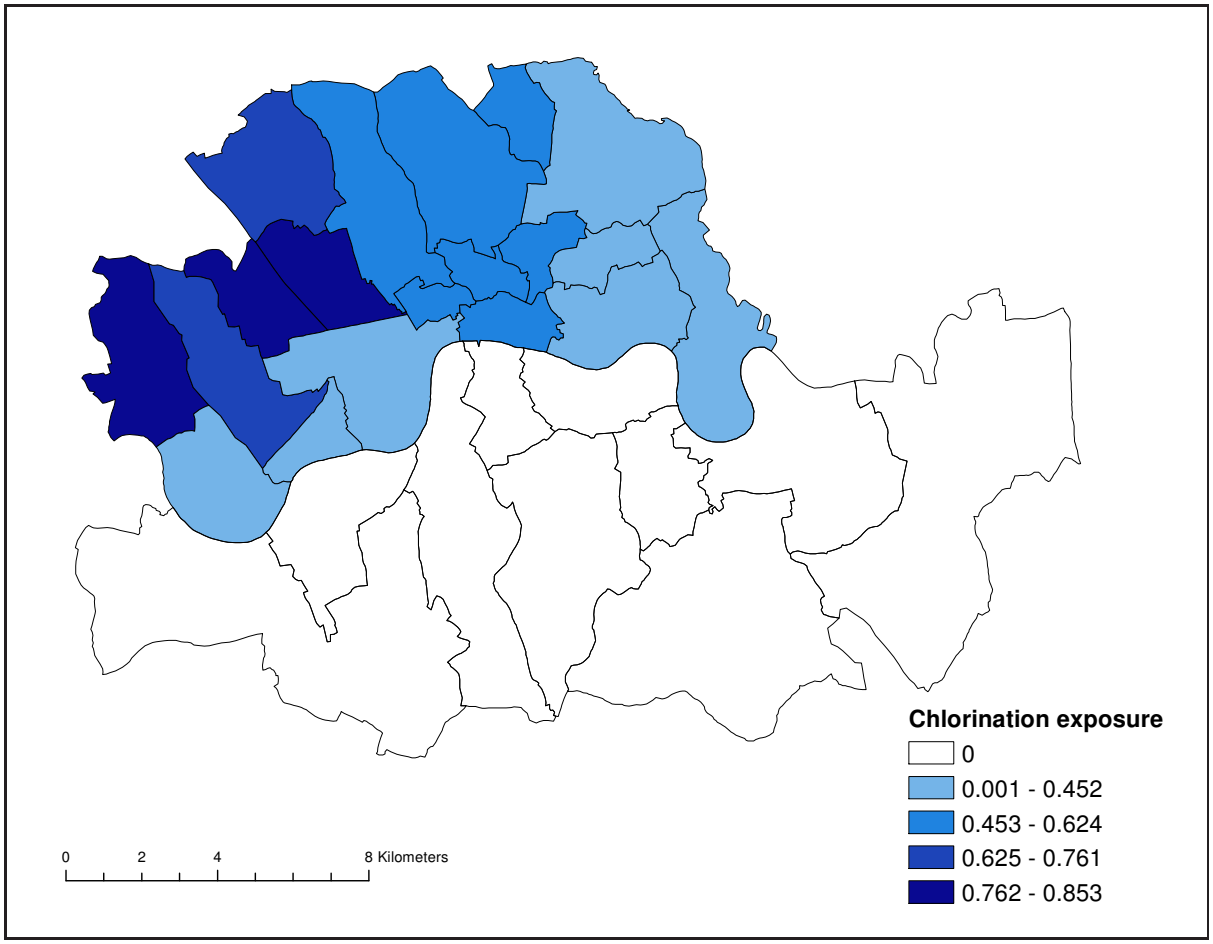
Notes: Figure 3 shows the boundaries for the County of London and the Metropolitan Boroughs. Source: Great Britain Historical GIS Project (1911).

Figure 4: Water London and the Metropolitan Water Companies (1903)



Notes: Figure 4 illustrates the boundary of "Water London" in blue, the administrative boundary of the County of London in red, and the water supply boundaries of the Metropolitan Water Companies in 1903. Source: Metropolitan Water Board (1903).

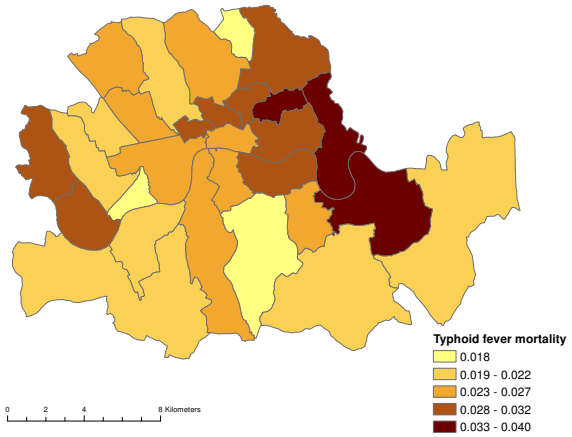
Figure 5: Exposure to chlorination, 1916-1926



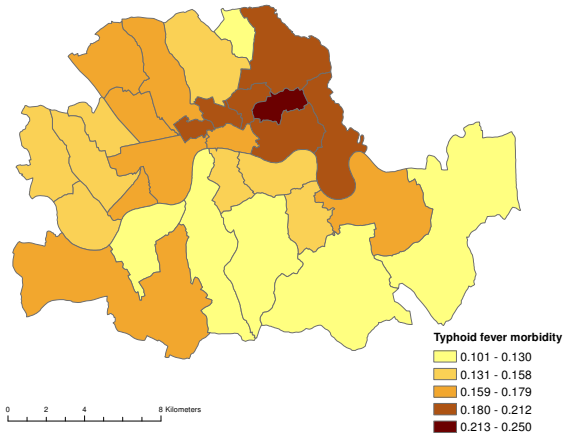
Notes: Figure 5 depicts the exposure of each London borough to chlorination treatment between June 1916 and December 1926. The exposure measure is an estimate of the fraction of the water supply that is chlorinated.

Figure 6: Geographic variation in typhoid fever and water quality, 1906-1926

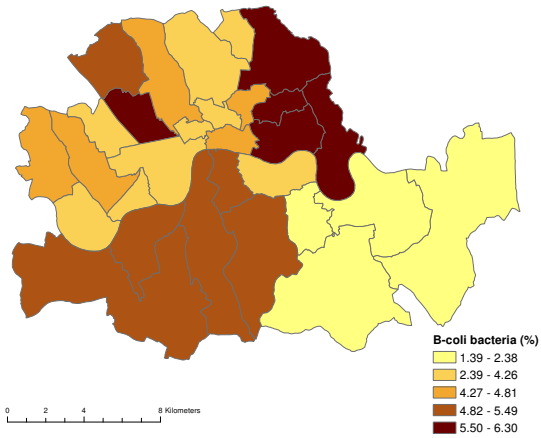
(a) Typhoid mortality rate



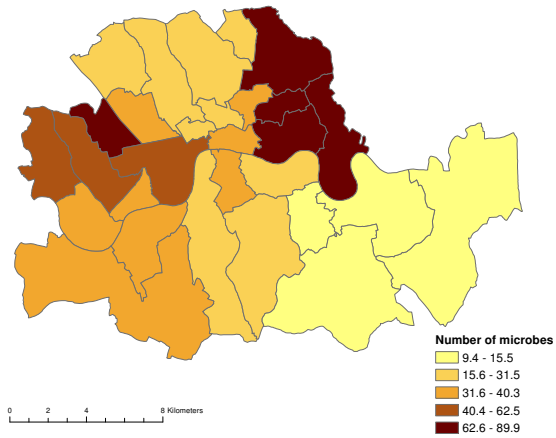
(b) Typhoid morbidity rate



(c) Positive tests for *B. coli* bacteria (%)



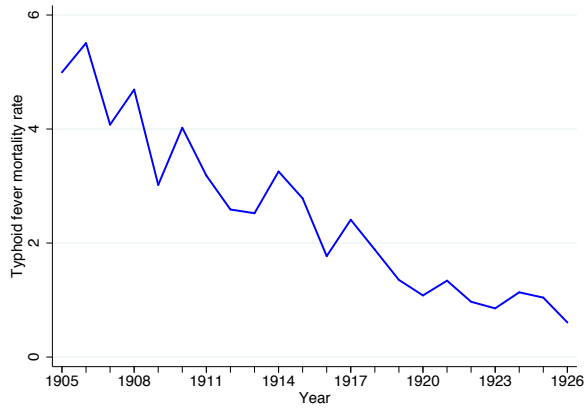
(d) Number of microbes



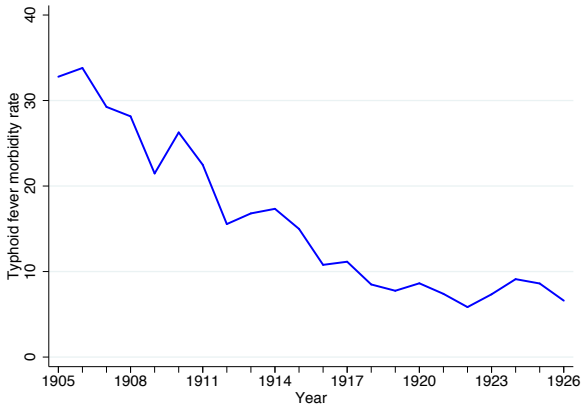
Notes: Figure 6 shows borough-level averages for typhoid morbidity and mortality rate, the percentage of positive tests for *B. coli* bacteria, and the number of microbes, calculated over the sample period of 1906 to 1926. Values are assigned to one of five categories, with darker colors indicating less healthy conditions. Sources: Great Britain Historical GIS Project (1911) for the GIS boundary files, *Quarterly Returns* for the mortality and morbidity data, and *Monthly Report of the Metropolitan Water Examiner* for the water quality data.

Figure 7: Time trends in typhoid fever mortality and morbidity, and water quality

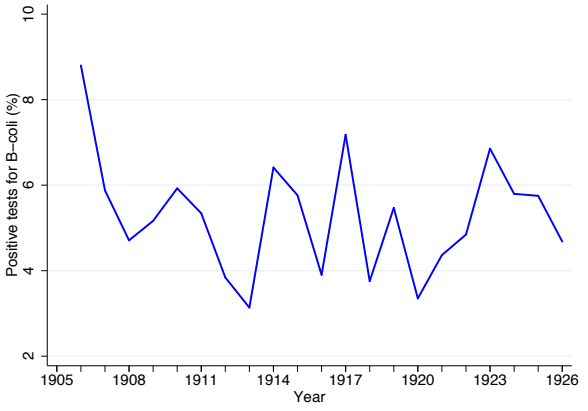
(a) Typhoid mortality rate



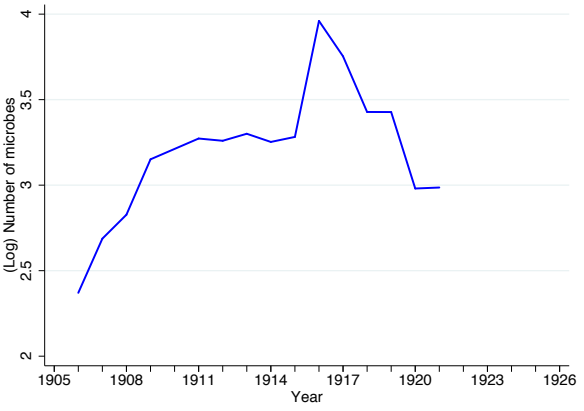
(b) Typhoid morbidity rate



(c) Positive tests for *B. coli* bacteria (%)



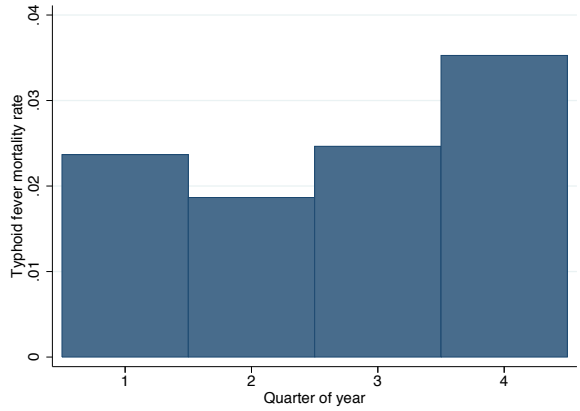
(d) (Log) Number of microbes



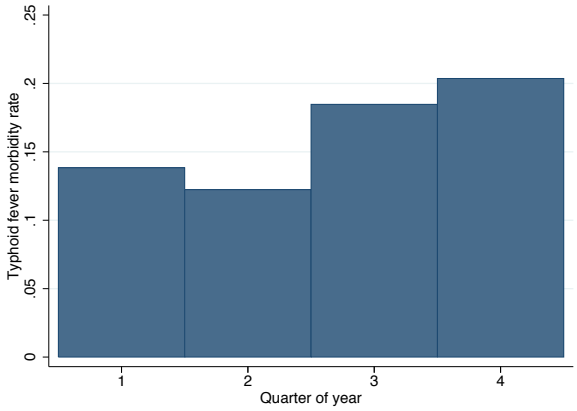
Notes: Figure 7 shows the time trend for typhoid morbidity and mortality rate, the percentage of positive tests for *B. coli* bacteria, and the number of microbes, over the sample period of 1906 to 1926. Sources: *Quarterly Returns* for the mortality and morbidity data, and *Monthly Report of the Metropolitan Water Examiner* for the water quality data.

Figure 8: Seasonality in typhoid fever mortality and morbidity, and water quality

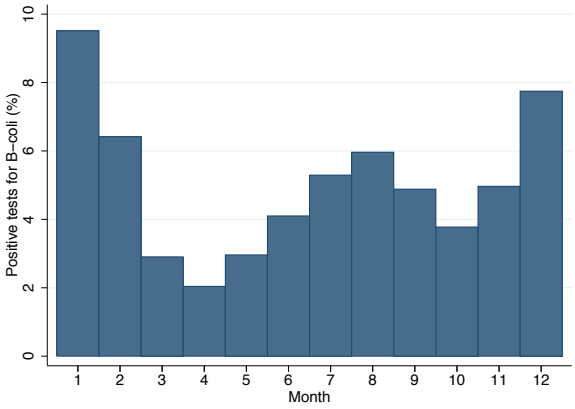
(a) Typhoid mortality rate



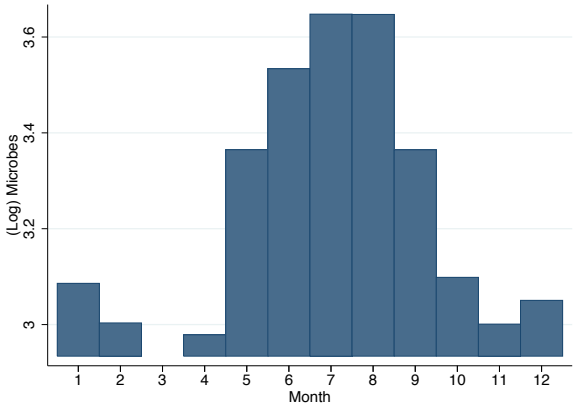
(b) Typhoid morbidity rate



(c) Positive tests for *B. coli* bacteria (%)

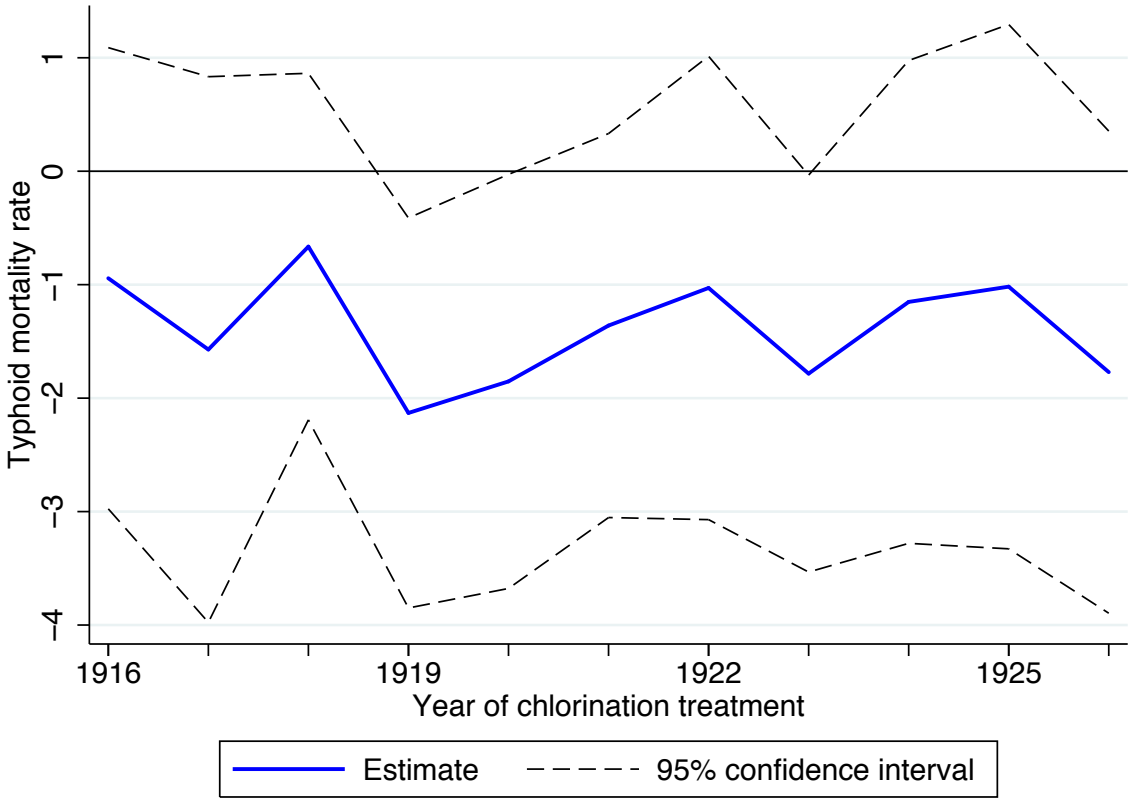


(d) (Log) Number of microbes



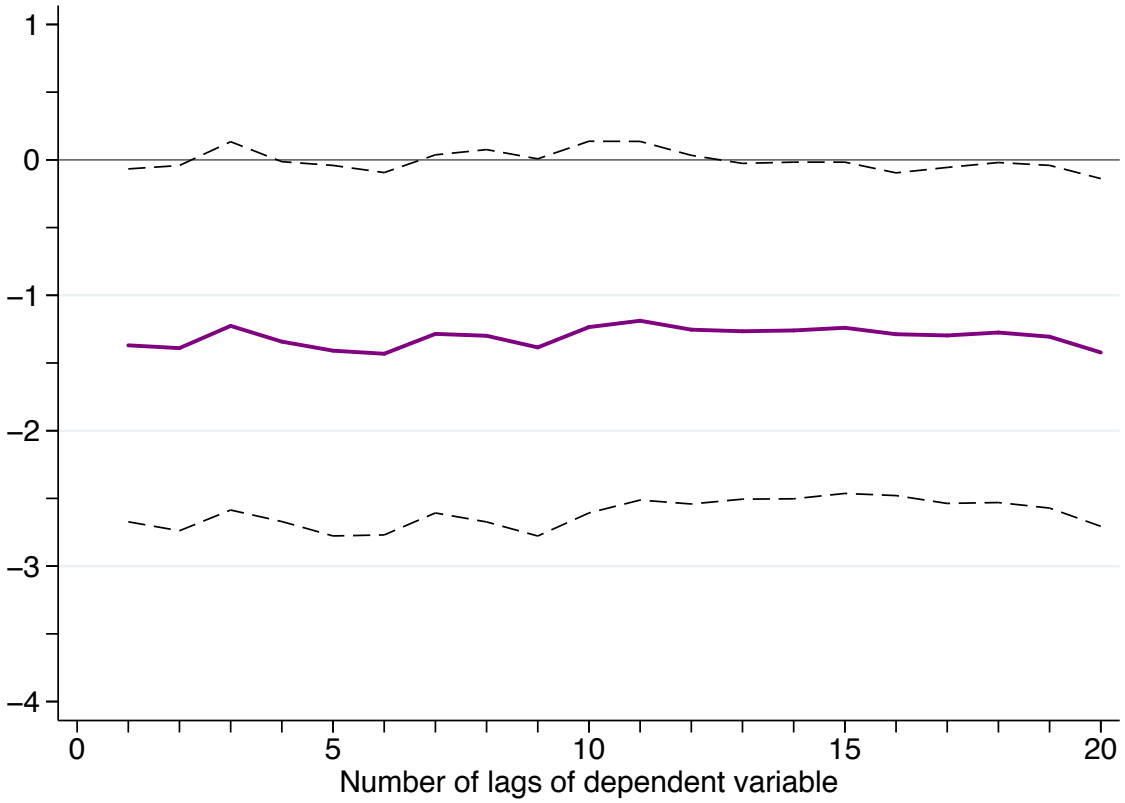
Notes: Figure 8 shows the quarterly seasonality in typhoid morbidity and mortality rate, as well as the monthly seasonality in the percentage of positive tests for *B. coli* bacteria, and the number of microbes, over the sample period of 1906 to 1926. Sources: *Quarterly Returns* for the mortality and morbidity data, and *Monthly Report of the Metropolitan Water Examiner* for the water quality data.

Figure 9: Effects of chlorination on typhoid fever mortality by year of treatment



Notes: Figure 9 plots the coefficients on the fractional exposure to the chlorination treatment interacted with year dummies from a regression with the annualized mortality rate (per 100,000 persons) in a borough-quarter as the dependent variable. The regression controls for year, quarter and borough fixed effects, as well as borough-specific linear time trends.

Figure 10: Effects of chlorination: Robustness to lagged dependent variable



Notes: Figure 10 plots the coefficients on the fractional exposure to the chlorination treatment from regressions with varying numbers of lags of the dependent variable. In each case, the dependent variable is the annualized mortality rate (per 100,000 persons) in a borough-quarter as the dependent variable. The regression controls for year, quarter and borough fixed effects, as well as borough-specific linear time trends.

Tables

Table 1: Summary Statistics

		No-chlorine		Chlorine	
	(1) All	(2) Pre	(3) Post	(4) Pre	(5) Post
Typhoid fever mortality rate	2.450	3.214	1.327	3.821	1.351
	(3.917)	(3.487)	(2.007)	(5.169)	(2.853)
Log infant mortality rate	4.440	4.637	4.267	4.605	4.275
	(0.447)	(0.282)	(0.376)	(0.382)	(0.504)
Log age [0,2] diarrhea mortality rate	2.437	2.473	2.123	2.602	2.433
	(0.917)	(1.070)	(0.824)	(0.965)	(0.774)
Typhoid fever morbidity rate	15.501	20.163	7.229	24.108	9.310
	(17.642)	(14.481)	(7.233)	(22.255)	(12.956)
Chlorination	0.187	0.000	0.000	0.000	0.557
	(0.300)	(0.000)	(0.000)	(0.000)	(0.249)
B-coli (%)	4.427	4.850	2.732	4.682	4.864
	(3.755)	(5.342)	(2.131)	(3.727)	(3.207)
Log number of microbes	3.467	3.040	3.137	3.584	3.830
	(0.896)	(0.797)	(0.674)	(0.988)	(0.677)
Observations	2436	410	430	779	817

Notes: Table 1 displays means and standard deviations for observations of variables used in estimation. An observation is a borough-quarter-of-year. The *Pre* period runs from 1906q1 to 1916q2 and the *Post* period runs from 1916q1 to 1926q4.

Table 2: Effects of *B. coli* bacteria on typhoid morbidity and mortality

	Mortality		Morbidity	
	(1)	(2)	(3)	(4)
	Panel A: Main effects of B-coli bacteria			
B-coli (%)	0.044**	0.049**	0.259***	0.223***
	(0.018)	(0.020)	(0.072)	(0.071)
Adj. R^2	0.145	0.152	0.265	0.290
	Panel B: Effects of B-coli bacteria by quarter of year			
B-coli (%) × Q1	−0.006	−0.001	−0.018	−0.033
	(0.024)	(0.024)	(0.085)	(0.076)
B-coli (%) × Q2	0.001	0.012	0.033	0.005
	(0.064)	(0.066)	(0.294)	(0.292)
B-coli (%) × Q3	0.081*	0.093*	0.392**	0.391**
	(0.043)	(0.047)	(0.166)	(0.170)
B-coli (%) × Q4	0.133***	0.128***	0.811***	0.698***
	(0.041)	(0.042)	(0.191)	(0.196)
Adj. R^2	0.147	0.154	0.269	0.293
Borough-trends	N	Y	N	Y
Mean of Y	2.450	2.450	15.501	15.501

Notes: N = 2436. In Columns (1) and (2) the dependent variable is the annualized typhoid fever mortality rate (per 100,000 persons) in a given borough and quarter-of-year. Columns (3) to (4) replace the dependent variable with the annualized typhoid morbidity rate (per 100,000 persons) in a borough-quarter. In Panel A, the variable *B-coli (%)* measures the fraction of laboratory samples that tested positive for the B-coli bacteria in different tests. In Panel B, *B-coli (%)* is interacted with quarter-of-year dummies. All specifications include year, month and borough fixed effects. Standard errors are clustered by borough.

Table 3: Effects of chlorination on typhoid morbidity and mortality

	Mortality		Morbidity	
	(1)	(2)	(3)	(4)
	Panel A: Main effects of chlorination			
Chlorination	-0.852^{*}	-1.397^{**}	-0.397	-0.444
	(0.468)	(0.642)	(2.817)	(2.862)
Adj. R^2	0.145	0.152	0.263	0.288
	Panel B: Effects of chlorination by quarter of year			
Chlorination × Q1	-0.740	-0.927	0.661	1.660
	(0.509)	(0.612)	(2.407)	(2.761)
Chlorination × Q2	0.777	0.493	6.441^{**}	7.019^{**}
	(0.584)	(0.778)	(2.821)	(3.240)
Chlorination × Q3	-0.069	-0.362	-1.014	-0.635
	(0.662)	(0.900)	(3.789)	(3.928)
Chlorination × Q4	-2.396^{***}	-2.609^{***}	-4.985	-4.133
	(0.660)	(0.763)	(3.351)	(3.364)
Adj. R^2	0.152	0.159	0.267	0.292
Borough-trends	N	Y	N	Y
Mean of Y	2.450	2.450	15.501	15.501

Notes: N = 2436. In Columns (1) and (2) the dependent variable is the annualized typhoid fever mortality rate (per 100,000 persons) in a given borough and quarter-of-year. Columns (3) to (4) replace the dependent variable with the annualized typhoid morbidity rate (per 100,000 persons) in a borough-quarter. In Panel A, the variable *Chlorination* is an estimate of the fraction of the water supply in a borough that received the chlorination treatment during the quarter. In Panel B, *Chlorination* is interacted with quarter-of-year dummies. All specifications include year, month and borough fixed effects. Standard errors are clustered by borough.

Table 4: Effects of chlorination by baseline typhoid mortality rate

	Number of years averaged for baseline typhoid mortality				
	(1)	(2)	(3)	(4)	(5)
Panel A: Interaction of chlorination exposure and baseline typhoid mortality					
Chlorination × Baseline mortality	−0.130	−0.184	−0.210*	−0.205	−0.264*
	(0.082)	(0.120)	(0.118)	(0.125)	(0.133)
Adj. R^2	0.133	0.125	0.097	0.099	0.081
Panel B: Chlorination exposure and interaction with baseline typhoid mortality					
Chlorination	−1.589*	−1.813*	−0.698	−1.371	−0.222
	(0.841)	(0.999)	(1.020)	(1.269)	(1.140)
Chlorination × Baseline mortality	0.062	0.137	−0.085	0.072	−0.220
	(0.133)	(0.207)	(0.225)	(0.305)	(0.273)
Observation	2320	2204	2088	1972	1856
Adj. R^2	0.134	0.126	0.096	0.099	0.081
Mean of Y	2.318	2.217	2.048	1.982	1.831

Notes: In each specification the dependent variable is the annualized typhoid fever mortality rate (per 100,000 persons) in a given borough and quarter-of-year. In column (1) data from 1906 is used to calculate the baseline typhoid mortality rate and data from 1907 to 1926 is used for estimation. In each subsequent column, an additional year is used to calculate baseline mortality and is excluded from the estimation sample. Thus, in Column (5) the baseline period is 1906 to 1910 and the estimation period is 1911 to 1926. All specifications include year, month and borough fixed effects, as well as borough-specific time trends. Standard errors are clustered by borough.

Table 5: Effects of chlorination by baseline level of water quality

	Number of years averaged for baseline water quality				
	(1)	(2)	(3)	(4)	(5)
Panel A: Interaction of chlorination exposure and baseline water quality					
Chlorination × Water quality	−0.169	−0.235	−0.182	−0.179	−0.208
	(0.140)	(0.141)	(0.134)	(0.132)	(0.134)
Adj. R^2	0.133	0.125	0.096	0.099	0.081
Panel B: Chlorination exposure and interaction with baseline water quality					
Chlorination	−2.398***	−4.007***	−4.526**	−4.101***	−3.520**
	(0.852)	(1.415)	(1.811)	(1.448)	(1.627)
Chlorination × Water quality	0.260	0.663*	0.836*	0.761**	0.566
	(0.171)	(0.335)	(0.420)	(0.341)	(0.361)
Observation	2320	2204	2088	1972	1856
Adj. R^2	0.134	0.126	0.097	0.100	0.081
Mean of Y	2.318	2.217	2.048	1.982	1.831

Notes: In each specification the dependent variable is the annualized typhoid fever mortality rate (per 100,000 persons) in a given borough and quarter-of-year. In column (1) data from 1906 is used to calculate baseline water quality and data from 1907 to 1926 is used for estimation. In each subsequent column, an additional year is used to calculate baseline water quality and is excluded from the estimation sample. Thus, in Column (5) the baseline period is 1906 to 1910 and the estimation period is 1911 to 1926. All specifications include year, month and borough fixed effects, as well as borough-specific time trends. Standard errors are clustered by borough.

Table 6: Effects of water quality on other diseases

	Mortality				Log Mortality		Morbidity	
	Measles	Scarlet fever	Diphtheria	Tuberculosis	Infant	Diarrhea [0,2]	Scarlet fever	Diphtheria
				Panel A: Effects of chlorination on other diseases				
Chlorination	0.298^{***}	-0.015	-0.044^{***}	-0.017	0.150^{***}	0.127	-0.947^{***}	-0.230
	(0.080)	(0.010)	(0.015)	(0.072)	(0.048)	(0.127)	(0.292)	(0.176)
Mean of Y	0.310	0.053	0.148	1.376	4.440	2.437	3.386	2.168
Adj. R^2	0.205	0.322	0.244	0.485	0.531	0.571	0.529	0.476
Observations	2436	2436	2436	1856	2433	2304	2436	2436
				Panel B: Effects of B-coli bacteria				
B-coli (%)	-0.000	0.000	0.000	0.006^{***}	-0.001	-0.001	-0.011	-0.007
	(0.002)	(0.000)	(0.001)	(0.002)	(0.001)	(0.004)	(0.010)	(0.005)
Mean of Y	0.310	0.053	0.148	1.376	4.440	2.437	3.386	2.168
Adj. R^2	0.201	0.321	0.243	0.488	0.529	0.571	0.528	0.476
Observations	2436	2436	2436	1856	2433	2304	2436	2436

Notes: In Columns (1) to (4) the dependent variables are annualized mortality rates (per 100,000 people) in a given borough and quarter-of-year for non-waterborne diseases. Columns (5) and (6) replace the dependent variable with the log infant mortality rate (per 100,000 live births) in a borough-quarter, and the age 0 to 2 mortality rate for diarrhea, respectively. In columns (7) and (8) the dependent variables are morbidity rates (per 1000 people) In Panel A, the variable *Chlorination* is an estimate of the fraction of the water supply in a borough that received the chlorination treatment during the quarter. In Panel B, *B-coli (%)* measures the fraction of (typical) laboratory samples that tested positive for the B-coli bacteria in 10 c.c. of water. All specifications include borough-specific linear time trends as well as year, month and borough fixed effects. Standard errors are clustered by borough.