MEDICAL TECHNOLOGY AND MORTALITY TRANSITION: THE DIPHTHERIA ANTITOXIN AND CHILDHOOD MORTALITY IN THE UNITED STATES, 1880-1910

by

Sadiq Salimi Rad

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ABSTRACT

Diphtheria was a deadly infectious disease in the late 19th and early 20th centuries, particularly among children. In 1895, an antitoxin was developed that could effectively treat the disease. This was the first and only infectious disease in the United States at the time with a scientifically-based treatment. To gauge the impact of access to the antitoxin on child mortality, I leverage large and stable differences in physicians per capita rates across 38 U.S. cities. Physicians were the primary distributors of the antitoxin at the time. For every percentage point increase in the rate of physicians per capita prior to the antitoxin's availability, there is a corresponding one percent reduction in child mortality. These findings suggest that the introduction of the antitoxin played an important role in saving children's lives and had a significant impact on the course of medical technology and child health in the United States.

INTRODUCTION

> "Mortality is one of the most important measures of social inequality because it indicates a group's success in providing members with the most highly prized of all attributes, **life** itself."

Samuel H. Preston and Michael R. Haines, *Child Mortality in Late Nineteen-Century America*, 1991.

At the turn of the twentieth century, diphtheria was the second leading cause of death among children. It accounted for 11 percent of the deaths in the 1- to 4-year-old age group and 14 percent in the 5- to 14-year-old age group (Preston and Haines, Table 1.1). While cases of diphtheria are exceedingly rare in the United States today, recent outbreaks have occurred in places such as Yemen, Bangladesh, and India (Das et al. 2016; Badell et al. 2021; Polonsky et al. 2021). Between 1892 and 1893, German scientist Emil Behring introduced diphtheria antitoxin, an effective treatment for diphtheria confirmed by clinical studies. By 1895, this antitoxin was widely available in the United States, and some major Canadian cities. Emil Behring received the Nobel Prize in Medicine in 1901 for the discovery of the diphtheria antitoxin.[1] Diphtheria was the first and only infectious disease that had an effective treatment available at the time. In fact, diphtheria has been called as "the *paradigmatic* disease of the so-called bacteriological revolution and the symbol of the triumph of scientific medicine in the

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[1] The Nobel Prize in Physiology or Medicine 1901 was awarded to Emil Adolf von Behring "for his work on serum therapy, especially its application against diphtheria, by which he has opened a new road in the domain of medical science and thereby placed in the hands of the physician a victorious weapon against illness and deaths" (The Nobel Prize website).

control of infectious disease" (Hammonds, 1999). The diphtheria mortality rate declined dramatically from 1880 to 1910, the time frame being examined.[2] The child mortality rate also declined by about 40 percent, going from 900 to 560 per 100,000 people.

The reduction in mortality, referred to as the mortality transition, was driven primarily by reductions in infant mortality and infectious disease (Costa 2015). Scholars traditionally relate this decline to better living conditions, the onset of economic growth, herd immunity due to natural selection, reduced virulence, and improved nutrition (Smith 2003; Daniel 2006; Kunitz 2007, pp. 196-197; Lönnroth et al. 2009; Mercer 2014, pp. 127-129). Basic public health measures, such as isolating patients in sanatoriums, were also proposed as an explanation for the reduced mortality rates (Wilson 1990; Fairchild and Oppenheimer 1998). But the effectiveness of such public health efforts has recently been called into question (Anderson et al. 2019). The effectiveness of a new medicine, specifically the diphtheria antitoxin, which was advertised and distributed by public health institutions, in contributing to the declining mortality trend (i.e., transition) at the turn of the twentieth century, remains an open question. This study, drawing on an original dataset, tries to address this question: did the diffusion of a novel medical technology, such as a vaccine or drug, help save children? In addressing this question, the current study relates to a growing literature on the relationship between technology and health.

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[2] See Figure 1. Mortality trends from 1880 to 1910 for cities in the sample.

Although the diphtheria antitoxin is remarkable with respect to its pioneering capacities in affecting health outcomes, it has not been studied in a systematic fashion. This is due in part to the scarcity of reliable health outcome data prior to 1900. Another explanation is that there is no differential timing in adoption of the antitoxin by different cities or states (i.e., availability in different medical markets).[3] This hurdle makes it difficult for a researcher to estimate the causal effect of an intervention when treatment is not staggered (i.e., all units are treated at one period and remain treated) (Athey and Imben 2022). Thus, for a variety of reasons, existing research on disease control and the mortality transition provide little evidence on the effects of the antitoxin campaign. This study uses data from primary sources (from 1880-1889) that complements previously available data (from 1900-1910) to explore the contribution of the antitoxin to the decline in child mortality in 38 cities in the United States. The strong link between the availability of the antitoxin to patients and physicians per capita (PPC) in each city motivates the main aspect of my analysis. The identifying variation comes from the historical, long-standing, and institutional idiosyncrasies that led to wide differences in PPC rates across cities. These differences are highly correlated to the certification laws for physicians in the 19th century. The validity of my identification strategy is based on the fact that PPC rates are persistent over the 30 years leading to 1900, after accounting for population dynamics over the period.

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[3] Almost all cities adopted diphtheria antitoxin in 1895. See Appendix Table A1 for the timing of the antitoxin adoption.

In the context of diphtheria antitoxin, the choice of a "dose-response" empirical strategy is a natural one. I estimate the reduced-form effect of the antitoxin in a difference-in-difference framework that compares child mortality rates before and after the antitoxin introduction in 1895 (first difference) between higher- and lower-PPC cities (second difference). The intensity of treatment is measured by the PPC rates. This empirical strategy eliminates the need for comparisons between cities that adopted the antitoxin at different points in time (earlier versus later), and differed in their pre-antitoxin mortality trends (Goodman-Bacon 2018b). The antitoxin is considered a one-time shock to all cities.[4] To ensure the validity of the analysis, a balancing test shows that the physicians per capita rates are uncorrelated with levels and trends of city demographics in the years before the antitoxin's introduction. Additionally, there was no major contemporaneous health program in 1895 or in the years before or immediately following 1895. The results from an event-study specification (Jacobson et al. 1993) show directly that mortality rates in higher- and lower-PPC cities did not trend differently for 15 years prior to the antitoxin.

The paper is the first to show in an empirically rigorous fashion that the antitoxin's introduction achieved its primary goal of saving children's lives. Higher-PPC cities experienced a decrease in both child and infant mortality rates relative to lower-

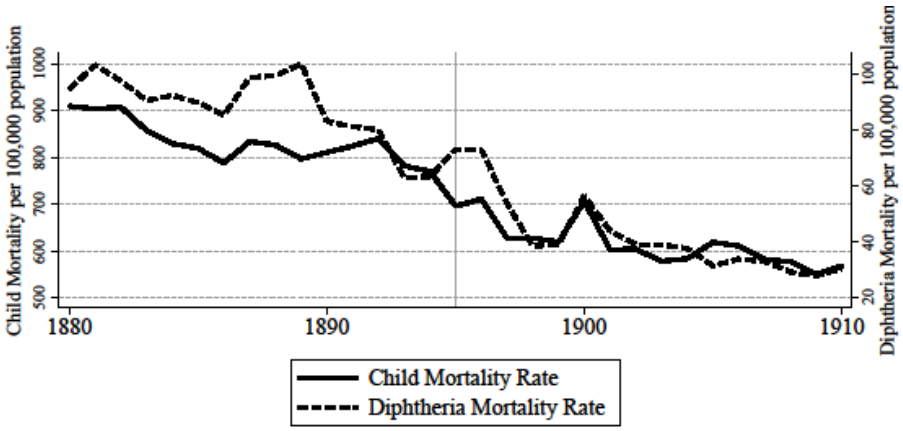
[4] Out-of-sample information on the introduction and availability of the antitoxin are collected from historical newspaper archives for majority of cities in the sample (list is provided in Appendix Table A1). For example, in February of 1895 the New York Times dedicated a column on page 3 to the introduction of the antitoxin by the city's health department and informed physicians that they could request the medicine free of charge.

PPC cities following the antitoxin's introduction, with each percentage point difference in the PPC rate resulting in a 1% decrease in child mortality rate. Diphtheria mortality rates showed a decrease as well, although with some lag. These effects were persistent throughout the sample period, up to 1910. An alternative specification, which compares child (under 5 years old) with nonchild mortality before and after the introduction of the antitoxin identifies the impact of the antitoxin on child mortality in a direction that supports main specification results.

The remainder of the paper is organized as follows. In chapter 2, I begin by providing background information on diphtheria bacteriology and the way the antitoxin treats the disease. An early study, which is possibly the first rigorous statistical analysis of a contagious disease, on diphtheria is discussed.[5] This chapter also provides some institutional and historical information on the importance of physicians in this context. In chapter 3, I describe the data sources. In chapter 4, I describe my empirical framework. Chapter 5 reports my results. Chapter 6 concludes.

[5] This is an important paper because it sheds light on how scholars dealt with causal relationships a century ago when modern econometric tools of causal inference had yet to be developed.

Figure 1. Child and Diphtheria Mortality Rates, 1880-1910



Sources: For 1880-1899, based on annual data from municipal and state public health reports: HathiTrust Digital Library, Google Books, or the archives at the National Library of Medicine in Bethesda, Maryland. For 1900-1910, based on Mortality Statistics, published annually by the U.S. Census Bureau.

BACKGROUND

Diphtheria—The Disease

Diphtheria is a *toxemia*. The *toxin* can cause damage to the kidneys, heart, and nerves. Diphtheria is defined as an acute infectious disease caused by *Bacillus Corynebacterium diphtheriae*.[6] Clinical manifestations occur one to seven days after infection with one or more strains of the bacillus.[7] The disease's signature feature is the buildup of a thick, gray membrane of dead cells in the nose and throat, which can obstruct breathing.[8] With their relatively small airways, children are particularly vulnerable to these potentially fatal breathing obstructions. Referred to as "childhood's deadly scourge" or the "plague among children," it was not uncommon for families to lose several children during a single diphtheria epidemic (Hammonds 1999; Klass 2021). Diphtheria mortality was often recorded incorrectly as a cause of death because of

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[6] Klebs and Loeffler receive credit for identifying *Bacillus Diptherae* in 1883 (Holt LE, 1913).

[7] The bacillus usually enters the body through one of three avenues: the upper respiratory track, mainly via droplets or discharges from infected persons; the secretions from the nose and throat of convalescent cases; or from the healthy throat of individuals who acquired the bacilli from being in contact with others having virulent germs.

[8] The disease is characterized by a local inflammatory lesion, usually in the larynx, called a psedo-membrance, and a toxic reaction involving primarily the heart and peripheral nerves. Death can result from respiratory obstruction by the membrane or from effects of the toxin on the heart, nervous system, or other organs. Although diphtheria toxin plays a part in the formation of the membrane its systematic effects following absorption are by far the most important.

imperfect diagnosis (for example, it was complicated by pneumonia in some cases) and incomplete recordings of death.[9]

Diphtheria—The Treatment

The antitoxin has the ability to neutralize the toxin produced by diphtheria. Emil Behring and his colleagues sought to create an artificial immunity against the poison produced by the diphtheria bacillus. They had increasing success with producing the antitoxin between 1891 and 1894.[10] The first antitoxin in the United States was produced in the laboratory of the New York City Board of Health and was available for use in treating cases of diphtheria in January of 1895 (New York Times, 1895). Physicians were encouraged to use it.

W.E.B Du Bois' Death Conundrum— Availability or Access

William Edward Burghardt (W.E.B.) Du Bois, an African American sociologist and civil rights activist, was born and raised in Great Barrington, Massachusetts. Racial prejudices were muted in his hometown and he received support from his teachers who recognized his brilliance (Du Bois, 1994). He would become the first African American

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[9] Its symptoms are very similar to other respiratory diseases, such as croup and whooping cough.

[10] Scientists grow diphtheria-causing bacteria in the laboratory and harvest its toxin. Next, they inject horses with the diphtheria toxin. As an immune response, the animal's blood produces diphtheria antitoxin. Scientists collect blood from the horses and separate out the antitoxin rich serum. Then, the antitoxin serum is purified for use as a medicine for people (National Library of Medicine—NIH). See the online source: https://www.nlm.nih.gov/exhibition/fromdnatobeer/exhibition-interactive/illustrations/diphtheria-alternative.html

to earn a Ph.D. at Harvard University and his distinguished career as a sociologist, social critic, and activist continued to the mid-20th century (Lewis, 2000). In 1899, however, he was a grieving father. On May 24, 1899, Burghardt Du Bois, son of W.E.B. and his wife Nina, died of diphtheria in Atlanta, Georgia (Du Bois, 1994).[11] Burghardt was two years old. Was his death a matter of *availability* of the antitoxin in Atlanta or *access* to it?

Nina Du Bois would never forgive her husband for leaving Philadelphia for Atlanta, where he had a postdoctoral fellowship. By the time of Burghardt Du Bois' death in 1899, it was established that the antitoxin treatment would dramatically reduce the likelihood of death from diphtheria when given within a few days of the onset of illness (Holt LE, 1913). Was Nina Du Bois' conjecture right? Would Burghardt have survived in Philadelphia? Karp and Gearing (2015) explore this hypothesis by investigating the availability of the antitoxin treatment to an African American child in Atlanta. They highlight race discrimination in access to medical services in the southern United States. They conclude:

> "Our hypothesis on undertaking this review was that an inability or unwillingness to provide antitoxin to a Negro child in the southern United States underlay Burghardt's death from diphtheria, and it was so. We also assumed that were Du Bois to have remained in Boston or Berlin where he had studied or in Philadelphia where he worked, Burghardt would have received diphtheria antitoxin. The antitoxin was available across the color line in Boston and to all in Berlin. The outcome of staying in Philadelphia is less clear and is beyond our abilities to determine."[12]

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[11] Atlanta, Georgia, where, at that time, the majority of African Americans were living a serf-like existence as land laborers or sharecroppers (Du Bois, 1994).

[12] They legitimately point to the segregated health services of the time. While racial analysis of the effectiveness of medicines is out of scope of this study, it is worth mentioning that the success of any medicine is highly hinged on the existence of a universal health system. The lack of a universal health system at the turn of twentieth

The two-year-old Du Bois' death story drives this study's use of physicians' services availability in each city as a measure of the antitoxin dosage intensity.

Physicians and Antitoxin—Anecdotal Evidence from the American Pediatric Society

The introduction of the antitoxin as the orthodox therapy in the treatment of diphtheria points to the role played by physicians in gaining the acceptance of the antitoxin by the public. However, physicians were split over the antitoxin's efficacy through 1895. In addition, the debate over the antitoxin explicitly addressed the professional authority of physicians because the antitoxin was introduced under the auspices of the health department (Hammonds 1999). The discussion of the antitoxin's efficacy extended to the national arena when it was the main topic of the annual meeting of the prestigious Association of American Physicians in 1895.[13] While physicians in the meeting were divided on the topic, William Welch summarized that there was ample experimental evidence in the support of diphtheria antitoxin and that it was the duty of physicians to use it.[14]

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century could explain the generally accepted opinion that anti-diphtheria efforts, though based on science, were unsuccessful and the disease remained unchecked. However, physicians played the role of universal health systems to some extent.

[13] See abstract of papers here at National Library of Medicine: https://collections.nlm.nih.gov/catalog/nlm:nlmuid-101160970-bk

[14] Public health authorities by the time perceived the importance of medical community support in success of the antitoxin. So, they actively attended medical meetings for campaigning the new remedy. For example, in April 1895, Hermann Biggs, director of the bacteriological laboratories of the New York Department of Health, was the first

By the summer of 1896, many physicians across the United States had gained nearly one year of experience using the antitoxin to treat diphtheria. In May of that year, the American Pediatric Society formed a committee to investigate the antitoxin's use in private practice cases. The committee gathered data from 613 physicians in 114 cities and towns across 15 states, the District of Columbia, and Canada. They examined 3,384 cases and concluded that the antitoxin was being widely used. These are the only descriptive data available on the first-stage outcome not available elsewhere: the number of physicians who used the antitoxin.

The committee expressed its surprise of the prevalence of the antitoxin use and the increasing number of physicians adopting the treatment explicitly:

> "The first surprise of the Committee was in learning how very widely the serum treatment had been employed, especially in the Eastern and mid-Western states. With more time, the number of cases collected might easily have been doubled and perhaps trebled; but enough reports have come in to enable one to see what opinion was held on the 1st of May, 1896, by American physicians who have used this remedy (1896, p.1)."[15]

The report was favorably in support of the use of the antitoxin and was accepted by the members of the American Pediatric Society:

> "… It is certain that antitoxin has passed the stage of experiment; that it has secured a firm foot-hold as a therapeutic agent; that it is a powerful remedy in 'controlling diphtheria', and is 'here to stay'.(Report of the American Pediatric, 1896, p.532 , emphasis added)."

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person who presented his paper "Some Experiences in the Production and Use of diphtheria Antitoxine" at the meeting of the New York Academy of Medicine.

[15] "The Report of the American Pediatric Society's Collective Investigation into the Use of Antitoxin in the Treatment of Diphtheria in Private Practice" (1896).

The introduction of the antitoxin and the delayed, divided response of physicians in adopting the remedy is reflected in diphtheria mortality statistics published by New York Department of Health.16 Figure 2 shows the trend of deaths from diphtheria from 7 years before to 8 years after the antitoxin introduction in 1895. Compared to 1894, the number of deaths decreased in 1895 by about 1000 deaths while the number of cases stayed almost the same. This could be due to the antitoxin shock and the early adopters of the remedy. It was only 3 years after the introduction of the antitoxin, in 1898, that the number of deaths met its new natural rate—about 1,000 deaths per year. Taking rapid population growth at the time into account, this reduction is striking. This delayed response in the number of deaths and cases could be due to two important factors:17 physicians' full commitment and diphtheria epidemiology. In some cases, patients given the antitoxin developed a lasting immunity against diphtheria. Also, it was a common practice that the antitoxin was given to healthy people as an immunizing agent to protect them against future possible infections.[18] Therefore, the antitoxin contributed indirectly to the reduction of deaths from diphtheria through reducing the transmission coefficient in

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[16] The City's health department was a pioneer in the introduction and production of the antitoxin, and campaigning against diphtheria in the United States. Evelynn Hammonds gives a detailed account of health department efforts for controlling diphtheria from 1880 to 1930 in her seminal book 'Childhood's Deadly Scourge' (1999).

[17] This lagged effect also shows up in event-study estimates (Figure 6).

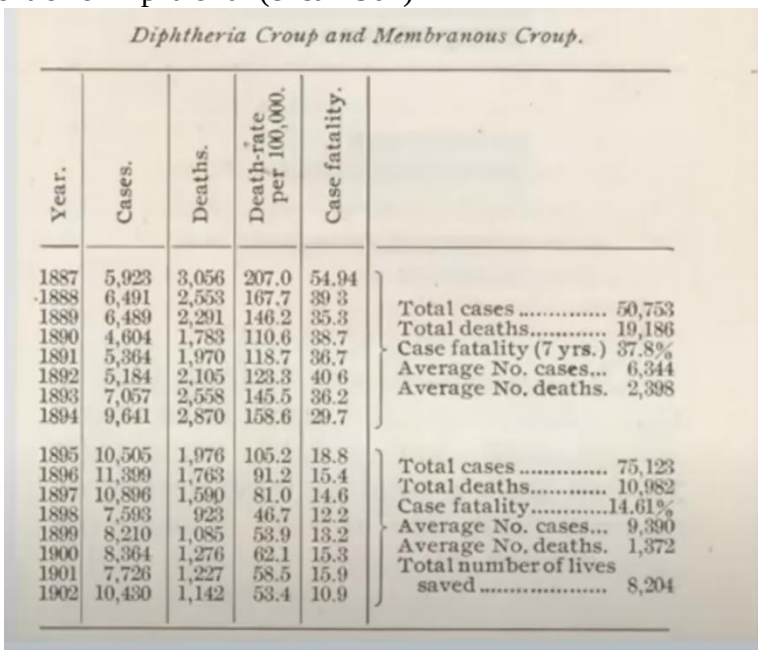
[18] It continued to be an advisable practice until the 1910s, when the first diphtheria vaccine was introduced. The vaccine was a mixture of toxin-antitoxin accompanied by Schick test. The test determined if the person has already developed immunity against diphtheria.

the Standard Inflammatory Response (SIR) model of disease diffusion.[19] That is, the antitoxin made diphtheria less contagious.

Nonetheless the debate on the efficacy of the diphtheria antitoxin lasted over twenty years after the introduction of the diphtheria antitoxin. Matthias Nicoll, from New York state department of health, wrote in 1915:

> "The death rate among actual cases has been immensely lowered by the use of antitoxin. It is the duty of the state to place an adequate supply at the disposal of every practicing physician, to do everything possible to encourage its use and to provide for its administration to those who are unable to pay for the services of a private physician. This is all that can

Figure 2. New York City Department of Health Report on the Control of Diphtheria (circa 1902)



Diphtheria Croup and Membranous Croup.

Year.	Cases.	Deaths.	Death-rate per 100,000.	Case fatality.	
1887	5,923	3,056	207.0	54.94	
1888	6,491	2,553	167.7	39 3	
1889	6,489	2,291	146.2	35.3	Total cases 50,753
1890	4,604	1,783	110.6	38.7	Total deaths..... 19,186
1891	5,364	1,970	118.7	36.7	Case fatality (7 yrs.) 37.8%
1892	5,184	2,105	123.3	40 6	Average No. cases... 6,344
1893	7,057	2,558	145.5	36.2	Average No. deaths. 2,398
1894	9,641	2,870	158.6	29.7	
1895	10,505	1,976	105.2	18.8	Total cases 75,123
1896	11,399	1,763	91.2	15.4	Total deaths..... 10,982
1897	10,896	1,590	81.0	14.6	Case fatality.....14.61%
1898	7,593	923	46.7	12.2	Average No. cases... 9,390
1899	8,210	1,085	53.9	13.2	Average No. deaths. 1,372
1900	8,364	1,276	62.1	15.3	Total number of lives
1901	7,726	1,227	58.5	15.9	saved 8,204
1902	10,430	1,142	53.4	10.9	

[19] The SIR model was developed by Kermack and McKendrick (1927, 1932, 1933). The transmission coefficient, β, is equal to the probability that an individual with active diphtheria will transmit his or her infection. See Anderson et al. (2019) footnote 28 for a discussion of the SIR model in the context of TB control. See Adda (2016) for an application of the SIR model to high-frequency data on viral diseases in France.

> reasonably be expected from health officials. The further lowering of the death rate rests absolutely in the hands of practicing physicians."20

A Statistical Study of Diphtheria

Frederick S. Crum (1917) used official reports from 15 major American cities that adopted the antitoxin in 1895 to analyze the effect of the antitoxin on deaths from diphtheria. This is one of the first data-based statistically rigorous studies that explored a medicine's efficacy. His study is based on comparing the difference in the number of deaths from diphtheria before and after the introduction of the antitoxin in 15 cities. "CHART 2" of Crum's paper summarizes his results (Figure 3 presents the chart below). While by this time there were few physicians who would be bold enough to openly proclaim opposition to the use of the antitoxin (such an expression of opinion would be a bad advertisement in any enlightened community) (Crum, 1917), he was motivated to provide some statistical evidence for the efficacy of the antitoxin. To account for potential confounding factors, he iterates the comparison by age, gender, race, season, climate, level of ground water, et cetera. He acknowledges that "what the underlying factors are which contribute to this result and what their relative importance is still a more or less baffling mystery." The importance of the paper is twofold. First, it highlights the interaction of health authorities and physicians and their role in success of the antitoxin as the primary channel of distribution at the time. Second, the author tries to

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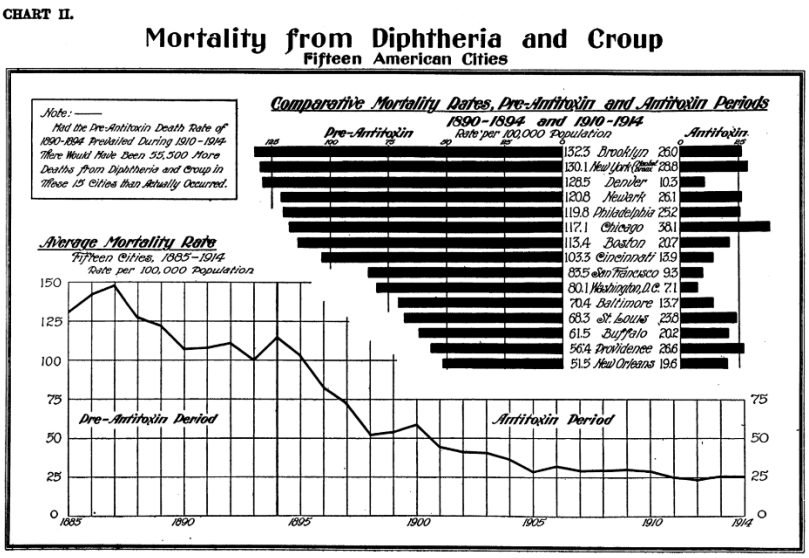
[20] Matthias Nicoll, Jr., M.D., Health News, November 1915, N. Y. State Department of Health.

make some causal statements while being cautious about the shortcomings of his study in controlling for confounding factors. In conclusion he says:

> "Finally, however, it must be admitted that many of the possibly very important conditions favorable to the development and spread of diphtheria are not well understood, …the possible variations in the virulence of the diphtheria germ under varying conditions of 'time' and 'place' are among the factors which require more extensive research than has thus far been possible (emphasis added)."

Figure 3. A Statistical Study of Diphtheria (1917), p. 449, The American Journal of Public Health, Volume VIII, No 5



DATA

I draw on multiple archival sources to assemble a dataset on health outcomes. I use the publicly available sources to compile physicians per capita rates and potential confounders.[21]

[21] Two potential policy-related confounding factors are the existence of health committees and a division of child hygiene in each city in 1895. The report on the Social Statistics of Cities, published in 1895 (this report is based on the 1890 Census, which is published by Census Office of the Department of the Interior), states on page 14 that "out of 292 cities reported regarding their several boards of health, 14 reported 'no board' and 2 reported the function that the functions of a health board were performed by the county medical society, while the remaining have regular boards." Thus, by 1895, health committees were prevalent in the United States, especially in the major and middle-sized cities included in my sample. One aspect of research on pre-1900 health topics is the fact that many health policies have yet to be activated. For instance, table 2 of Moehling and Thompson (2012) shows that first divisions of child hygiene in the United States were initiated after 1914-15. So, neither health committees nor divisions of child hygiene are binding factors in my analysis because all cities had the former while no city had the latter in 1895. The pre-1895 bed capacity of hospitals would also not be a determining factor in my analysis, largely because there were few cities in the United States with hospitals that accepted diphtheria patients at the time (Hammond, 1999). Bacteriological laboratories, mainly run by health departments that offered diphtheria test kits to physicians, were also not a major player for two reasons. First, there was a conflict of interest between physicians and authorities in the use of lab tests for the diagnosis of the disease. Physicians were not open to replacing their own clinical diagnosis with lab tests because it threatened their occupational status as the ultimate healer agent. Also, physicians legitimately doubted the validity of tests. Second, it was well established that the antitoxin treatment would dramatically reduce the likelihood of death from the disease when given within a few days of the onset of illness (Holt LE, 1913), while the process of taking a sample and sending it to a laboratory took considerable time. So, physicians were inclined (and even were encouraged, as reflected and advised in the report of American Pediatric Society) to use the antitoxin based on their diagnosis.

Physicians Per Capita

I used the decennial census of 1900[22] to calculate physicians per capita (PPC) rates by dividing the number of private physicians and surgeons by the population of each city. Since this produces very small numbers that are all less than one, I multiplied the PPC variable by 10000 to avoid problems of having a regressor with values less than one in my regression. The resulting continuous variable maintains the ordinality of the original PPC variable within a range from 18 to 45.

As mentioned above, 1900 PPC rates are post-treatment measures of the intensity of the treatment in 1895. Considering the lack of data from the 1890 Census,[23] 1900 is the closest year to the treatment year. PPC rates from 1870, the earliest census year that comprehensively includes almost all cities in the sample, produces the same result as 1900 PPC rates as the measure of treatment, suggesting the persistence of PPC well before and after the introduction of the antitoxin in 1895.[24]

My identification strategy assumes that the relative number of physicians evolved exogenous to the introduction of the antitoxin. Visual evidence in the form of a univariate regression line in Figure 4, which is provided in the next section, shows that variation in PPC rates came from long-standing institutional features of cities. More evidence on this

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[22] 1900 census occupational classification system, recorded in the IPUMS variable OCC (049:Physicians and Surgeons), is used. Population data come from the decennial censuses and are linearly imputed for intercensal years.

[23] As mentioned before, most of the 1890 population schedules were badly damaged by a fire in the Commerce Department building in January 1921.

[24] Event-study figure that uses 1870 instead of 1900 PPC rates shows the same pattern. The figure is presented in Appendix Figure A3.

is presented in the following sections, including a balancing test and instrumenting 1870 rates for 1900 rates.

Health Outcome—Mortality

This paper focuses on mortality as the main health outcome. It is an extreme health outcome but unambiguous indicator of poor health and access. It is also well measured. The city-level mortality data comes from a wide range of sources. For the pre-1900 period, mortality counts are from annual municipal and state public health records.[25]These records were obtained through interlibrary loan, the HathiTrust Digital Library, Google Books, or the archives at the National Library of Medicine in Bethesda, Maryland. For instance, mortality data for Philadelphia, Pennsylvania, was obtained from the HathiTrust Digital Library (1880-1887, 1890, 1892), the National Library of Medicine Archives (1888-1889, 1894-1896, 1899), and Google Books (1891, 1893, 1897-1898).[26] For the period 1900-1910, mortality counts come from *Mortality Statistics*, which was published annually by the U.S. Census Bureau beginning in 1900.[27]

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[25] I am grateful to my advisor Mark Anderson for graciously providing me with the pre-1900 mortality data. I am also thankful to Michael Mckelligott for collecting the data.
[26] Appendix Table A2 lists the sources from which the pre-1900 mortality data were obtained.
[27] Mortality data from *Mortality Statistics* have been used by other researchers interested in the effectiveness of public health interventions. For instance, see Cutler and Miller (2005), Hoehn-Velasco (2018), Anderson et al. (2019), and Hoehn-Velasco and Wrigley-Field (2022).

Figure 1 shows child deaths (i.e., deaths among children under the age of 5) per 100,000 population for the 38 cities in my sample.[28] In 1880, there were about 900 child deaths per 100,000 population. By 1910, the child mortality rate had fallen to 560. Figure 1 also shows diphtheria mortality rate from 1880 to 1910, which had fallen from about 100 to 30 per 100,000 population.

Out-of-Sample Information—Historical Newspaper Archives

I consulted historical newspaper archives to garner out-of-sample information on the year the antitoxin was introduced in different cities.[29] A list of the year of the antitoxin's introduction in different cities is presented in the Appendix Table A1. Two issues are associated with this list. First, the list is not exhaustive because not all cities had an active local newspaper, or some of the newspapers have not been digitized and archived yet. Second, among cities for which I found historical evidence of the use of the antitoxin, the majority adopted the remedy in 1895 (i.e., no staggered treatment). I do not use this information directly in my analysis, but it provides a reasonable foundation for my empirical strategy. I use 1895 as the treatment year in my analysis based on information gathered from these historical sources.

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[28] The sample comprises nearly all major and medium-sized cities of the period, selected based solely on the availability of at least 10 years of pretreatment data. This sample is the most balanced dataset that could be constructed with the available data sources. Only 8 municipalities, mostly small cities - Fall River, Lawrence, Lowell, Lynn, Somerville, Springfield, Worcester, and Cambridge - are missing data from 1891 to 1897. However, the majority of cities in the sample have the complete series (1880-1910).

[29] newspapers.com is the main source of investigation.

RESEARCH DESIGN: DIFFERENCE-IN-DIFFERENCE USING PHYSICIANS PER CAPITA

The coverage of the antitoxin was larger in cities with higher PPC rates. The research design exploits the historical differences in PPC rates using a difference-in-difference strategy that compares mortality rates before and after the antitoxin's introduction (first difference) between cities with higher and lower PPC rates (second difference). Categorizing cities by their base PPC rates in 1900, denoted by PPC_c^*, provides a fixed ranking by which to compare mortality and avoids using differences in the antitoxin timing and year-to-year changes in PPC rates as sources of identifying variation (Goodman-Bacon, 2021). If PPC_c^* is uncorrelated with other cross-city changes in mortality (excludability) and PPC_c^* predicts the the antitoxin dosage (relevance), this strategy eliminates bias from differential counterfactual trends.

Base PPC rates are plausibly excludable instruments because cross-city variation in relative number of physicians come from predetermined institutional factors unrelated to other city's characteristics when the antitoxin introduced first. The design's main identifying assumption—that in the absence of the antitoxin, mortality would have evolved similarly in higher- and lower-PPC cities—is likely to hold for two reasons. First, identifying variation in PPC rates were long-run, stable features of cities and did not emerge contemporaneously in the 1890s. That is, PPC_c^* is correlated with past PPC rates in 1870 and 1880. In univariate regressions of PPC_c^* on PPC rates in 1870 and 1880, slope coefficients are precisely estimated and not statistically distinguishable across years (Figure 4, and Appendix Figure A2). This would address the concerns that

cities made policy choices or physicians changed their behavior in anticipation of (or after) the antitoxin's introduction. Second, the long-run institutional variation in PPC rates is largely uncorrelated with cities demographic characteristics in years before 1895 (Table 1).

Physicians Per Capita—A Persistent Measure

To measure the availability of the antitoxin in different cities in 1895, I used physicians per capita (PPC) rates from 1900 due to technical considerations and data availability restrictions.[30] [31] One might criticize this as a post-treatment measure. Two concerns should be addressed here. First, the relative number of physicians in 1900 might not represent the relative number of physicians in 1895. The examination of the evolution of the PPC institution, however, suggests a smooth, linear, and persistent growth over the 25 years before 1895 and in the following years. Therefore, using PPC rates in 1900 did not significantly alter the control and treatment group structures compared to using PPC rates in 1895. This is demonstrated in Figure 4, which shows a linear and persistent relationship between 1870 and 1900 PPC rates. In a univariate regression of PPC rates in 1900 on PPC rates in 1870, the null hypothesis that the slope of the line is equal to 1

[30] Using the 1880 and 1870 PPC rates do not change the sign of the estimates (Appendix Figure A3).

[31] The primary sources used for compiling PPC rates are Census Occupational Codes from 1870 through 1900. Census data for 1890 is not available because the documents were badly damaged by a fire in 1921. The exact description of data sources is provided in data section.

could not be rejected at conventional levels. This indicates that the higher-PPC cities in 1870 maintained their status up to 1900 and vice versa.[32]

The second concern is the likely endogenous response of physicians to the antitoxin availability. Physicians were likely to move to medical markets where the demand for their diphtheria-related services was facilitated by higher availability of the antitoxin. If this is true, my estimates are positively biased. However, physicians were unlikely to move to cities where the antitoxin was more available. First of all, access to the antitoxin did not require physicians to move to larger cities, as health authorities were committed to supplying enough drugs for their market use.[33] In addition, strict certification rules and laws restricted the free movement of physicians between different cities. Finally, the historical literature on diphtheria suggests that physicians did not perceive the antitoxin as a breakthrough medicine that would incentivize them to move to high-availability regions. Therefore, it is unlikely that the availability of antitoxin affected the supply of physicians' services to diphtheria patients at an extensive margin.

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[32] Using PPC rates from 1880 Census also produces almost the same picture with a caveat. Population dynamics made the line slightly steeper. Plotting two lines in a graph, one could see the convergence of two lines and the similar dispersion pattern of data points. The figure plotting both lines is presented in Appendix Figure A2.

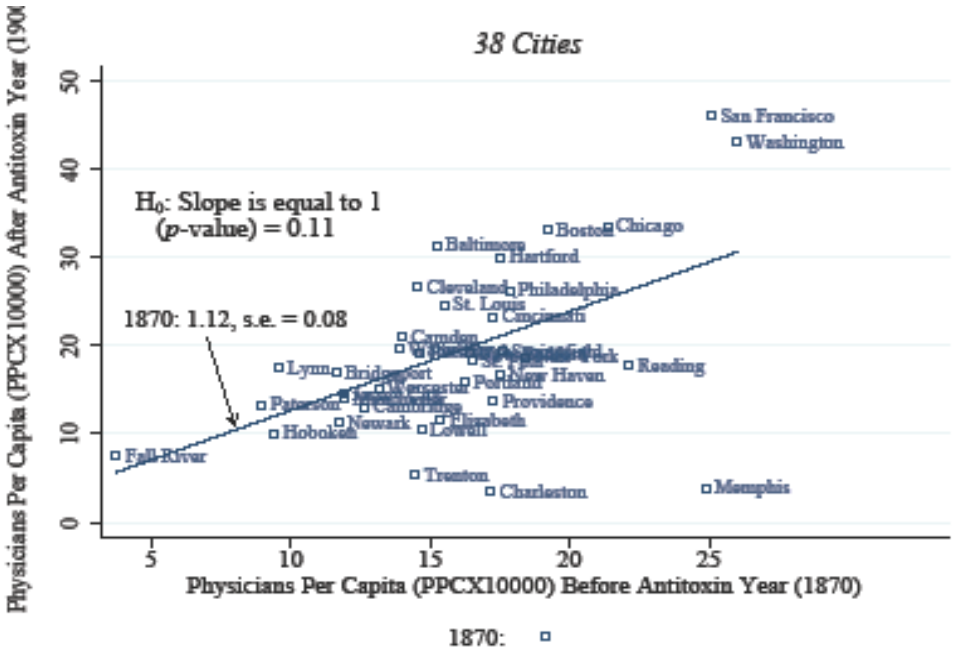
[33] The supporting evidence for this claim is the report of American Pediatric Society on the antitoxin's efficacy cited above. In 1896, just one year after the introduction (and well before 1900) the Society showed that the treatment was being abundantly used in 3,384 cases from 613 different physicians in 114 cities and towns in 15 states, the District of Columbia, and Canada. The report confirms that from the beginning access to antitoxin for physicians was not a binding constraint.

Physicians Per Capita—Evidence for Exogeneity

In order to verify the absence of correlation between PPC rates and cities' demographic characteristics in the years prior to 1895, I estimate the following equation for a range of city outcomes (y_{ct}):

$$y_{ct} = \alpha + \beta_0 PPC_c^* + \beta_1 PPC_c^* \times (y - 1894) + \epsilon_{ct}$$

Figure 4. Stability in Cross-City PPC Variation, 1870 through 1900



Notes: The figure presents a scatter plot and fitted value of the relationship between physicians per capita rates (PPC) in 1900 (y-axis) and PPC rates in 1870 (x-axis). The results show that the cross-city variation in PPC was very stable over time. Pre-antitoxin PPC rates strongly predict post-antitoxin PPC rates (including PPC rates in the year of the antitoxin, 1895), and the relationship itself does not change over time. Rates are multiplied by 10000. Sources: Based on 1870 and 1900 census occupational classification system, recorded in the IPUMS variable OCC (049:Physicians and Surgeons)

This is a test for balance in levels in the year 1894, the last year before the antitoxin's introduction, and in linear pre-antitoxin trends. The coefficient β_0 is the relationship between PPC_c^* and levels of each outcome variable in 1894 ($Ho: \beta_0 = 0$), and β_1 is the relationship between PPC_c^* and linear trends in each outcome variable ($Ho: \beta_1 = 0$). Table 1 provides the results of this test. The pre-antitoxin mean of each dependent variable is presented in column 1.

Panel C reveals that physicians' certification laws can largely explain the source of exogenous variation in PPC rates, with many significant estimates. Certification laws do not predict mortality rates, as shown in Table 4. However, there is a strong correlation between PPC rates and mortality rates, as documented in Figure 5. These observations suggest that PPC rates are measuring the effect of another factor on mortality rates, with access to diphtheria antitoxin being the potential candidate, given the research design.

There is no evidence of the relationship between base PPC rates and levels and trends of child, infant, and diphtheria mortality rates, suggesting that there is no reverse causality. In panel A, all estimated coefficients in column 2 and column 3 are imprecise and indistinguishable from zero. Panel B shows that PPC rates are also not significantly correlated with most socioeconomic variables that proxy quality and standard of life. These results support the claim that the variation in PPC rates was due to idiosyncratic city-level historical institutions that are not related to mortalities or other socioeconomic outcomes.

Table 1. Balancing Test: The Relationship Between Base PPC Rates and Pre-Antitoxin City Mortality Rates, Socioeconomic Variables, and Physicians Certification Laws in Levels and Trends

Dependent Variable	Pre-Antitoxin Mean	Level (PPC*)	Trend (PPC*×Year)
	(1)	(2)	(3)
	A. Demographic Outcomes 1880-1895		
Child Mortality	720	0.34	-3.21
		(0.38)	(3.22)
Infant Mortality	467.8	0.22	-0.81
		(0.23)	(1.97)
Diphtheria Mortality	73.6	0.03	-0.73
		(0.04)	(0.46)
	B. Socioeconomic Outcomes 1880-1895		
% Black	0.04	0.04	4.30
		(0.36)	(22.56)
% Female	0.50	0.01	-0.36
		(0.04)	(3.11)
% Foreign	0.29	0.06	-13.98
		(0.35)	(24.42)
% Under 18	0.35	0.17	-8.62
		(0.11)	(7.59)
% Literate	0.74	-0.06	18.56***
		(0.07)	(4.96)
	C. Physicians Certification laws (measured by state)		
Registration Law	0.65	-2.30	-248.67***
=1 if state had a registration law		(2.14)	(108.70)
Exam Board	0.90	-22.61***	-17.17
=1 if state had an examining board		(6.15)	(82.90)
Mandatory Exam	0.63	-22.60***	-240.24***
=1 if state mandated examination for medical practice		(7.91)	(81.85)
Mandatory Diploma	0.52	-21.35***	-225.05***
=1 if state required a diploma as a prerequisite for certification		(8.56)	(89.14)
Inferior Schools Exclusion	0.61	-15,85***	-36.35
=1 if state could exclude graduates of inferior schools from certification		(5.88)	(115.49)
Prelimenary Education Requirement	0.52	-15.84	-160.56
=1 if state medical boards could set prelimanary education requirements		(9.28)	(97.47)
Ethics Code Board	0.70	-17.89***	46.36
=1 if state medical board could refuse/revoke certifications for conduct issues		(5.62)	(101.25)

Notes: The table presents weighted estimates from the balancing test equation given in the text. Column 2 presents the relationship between PPC_c^* and levels of each variable in 1894 (β_0). Column 3 presents the relationship between PPC_c^* and linear trends in each variable (β_1). Sources: Information on licencing laws and educational requirements for physicians is from Hamowy (1979) and Baker (1984). Also see sources for Figure 6.

Table 1 does not include a crucial socioeconomic variable, which is income and earnings. In contrast to the positive relationship between income and health outcomes in urban areas during the 20th century, mortality rates were higher in urban areas compared to rural areas in the period under study (1880-1900), also known as the Gilded Age. This era was marked by rapid industrialization, real wage growth, unprecedented immigration to cities, and extreme wealth inequality, leading to poverty.[34] Other relevant healthcare variables such as hospital capacity and divisions of child hygiene are discussed in the data section.[35]

While base PPC rates do not predict primary mortality rates of cities (Table 1) before 1895, it is assumed that it can strongly predict the antitoxin use in different cities. The results presented in the following sections are based on this assumption.

Physicians Per Capita— Correlation with Changes in Mortality Rates

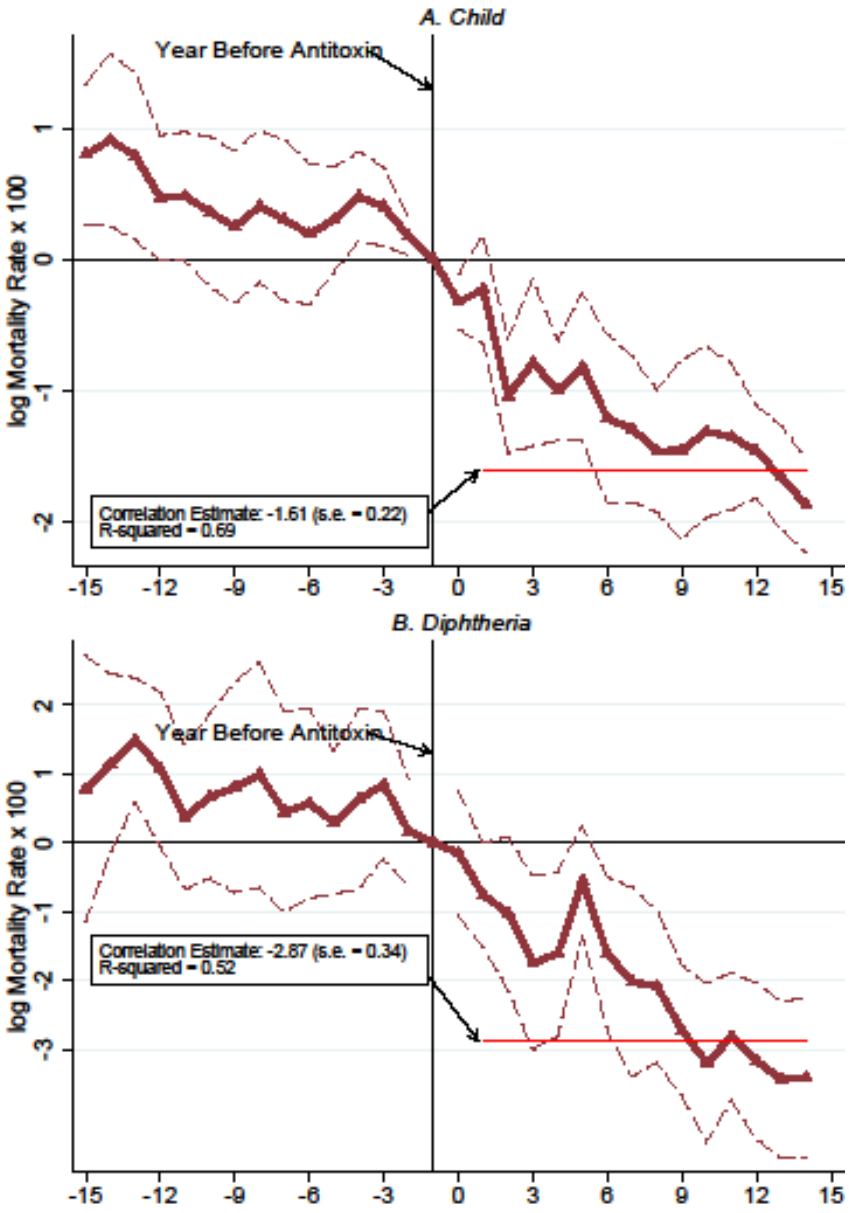
Figure 4 shows how dispersed the distribution of PPC rates is in my sample of cities. This provides clear evidence of substantial differences in access to physicians' services, which correlates with the disparity in mortality rates.

To gauge the statistical significance of these associations, I estimate fixed effects models that relate changes in mortality rates to changes in physicians per capita rates at the city level. By comparing differences across cities in changes over time, the analysis controls for all permanent city-wide factors that are correlated with mortality rates

[34] I test the wealth hypothesis directly in the results section.
[35] See footnote 23.

(Almond et al. 2006). For example, the association in metropolitan areas is identified from differences in changes between New York City and Chicago. Figure 5 shows the event-study specification of this fixed effects model. Fixed effects regression results for child and diphtheria mortality rates are presented in boxes. While these results should not be given a causal interpretation, they suggest that increased access to the antitoxin was a factor in decreasing mortality rates.

Figure 5. Fixed Effects Correlation Between Physicians Per Capita Rates and Mortality Rates



Notes: All specifications include city fixed effects. The explanatory variable is base physicians per capita rates which interacted with event year dummies for 15 years before and after the introduction of the antitoxin in 1895. The coefficient on the post-1895 dummy, which is one for years after 1895 in the regression, is presented as the correlation estimate in the box. All regressions are weighted by city population. Sources: See sources for Figure 6.

Event-Study Specification

To estimate the relationship between the diffusion of the diphtheria antitoxin and child mortality, I consider an event-study specification. Pre- and post-treatment are defined by dummy variables that measure the time relative to the antitoxin's introduction in 1895, and treatment/control groups are dictated by the continuous variable of base PPC rates (PPC_c^*):

$$Ln(MR_{ct}) = \alpha_0 + v_c + \lambda_t + \alpha_{rt} + \boldsymbol{X'_c}\beta + PPC_c^*[\sum_{y=-15}^{-2} \pi_y 1(t - 1895 = y) + \sum_{y=0}^{15} \theta_y 1(t - 1895 = y)] + \epsilon_{ct}$$

where $Ln(MR_{ct})$ is the natural log of the child mortality rate in city c and year $t = 1880 \ldots 1910$.[36] City fixed effects, v_c, control for any time-invariant, city-specific determinants of mortality or rigid institutional differences across cities. year fixed effects, λ_t, account for common shocks to cities. The term α_{rt} controls for region-by-year fixed effects. My preferred specification of X'_c includes two measures of population density and two measures of wealth (mean personal properties and mean personal real estates).[37] The event-year dummies, y, are equal to 1 when the year of observation is $y = -15, \ldots, 0, \ldots, 15$ years from 1895, the year of the antitoxin's introduction. In my specification, there is no control for the antitoxin timing group because the take-up of the

[36] The results shown below remain quantitatively similar without natural log transformation of the dependent variable.

[37] One measure determines if an urban area has a population density of at least 1000 persons per square (URBAREA). The other categorize cities based on the size of place (SIZEPL)

antitoxin was quick and universal. The lack of the antitoxin timing groups force identification to come only from comparisons across PPC_c^*.[38] I cluster standard errors at the city level.

I omit $y = -1$ from equation (1), which normalizes the estimates of π_y and θ_y to 0 in that event year. π_y and θ_y map out the relationship between log mortality and base PPC rates through the 15 years before and after the antitoxin's introduction. The π_y are falsification tests. They trace out the relationship between mortality rates and base PPC rates over the 15 years before the antitoxin was introduced. Over the 15 years leading to 1895, The π_y are statistically indistinguishable from zero, suggesting the common trends assumption hold. The θ_y are the measures of the effects of an additional percentage point of base PPC rates on mortality rates over time. Considering the delayed acceptance by physicians and lagged epidemiological impacts of the antitoxin, each θ_y is equivalent to a distinct experiment in which the antitoxin dose differs by $PPC_c^* \times (y^* - 1894), y* = 1895, \ldots, 1910$. After full commitment of physicians, which is about 3 years after the introduction, the pattern of θ_y for remaining years should be similar (i.e., these years had the same experiment). Thus, this specification identifies heterogeneity in the antitoxin effects over time.[39]

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[38] Even if I were able to determine the differential timing of the antitoxin adoption, and there were enough variations on that dimension for a timing-only estimator to identify the effect of the antitoxin, I speculate that the timing of the antitoxin's introduction in each city was not random.

[39] θ_y identify heterogenous effects by amount of exposure in different years.

In my preferred specification, I do not include unit-specific linear time trends. A limitation of the city-specific linear time trends specification is that any differential trends are only allowed to vary linearly, which is a very restrictive assumption in the context of highly discontinuous changes of the mortality transition era. In addition, while the event-study figures clearly show the time-varying treatment effects, unit-specific time trends cannot distinguish between such effects and preexisting trends (Lee and Solon 2011).[40] To address this, I follow Bhuller et al. (2013) and Goodman-Bacon (2018a) by estimating a linear pretrend interacted with PPC rates for only the antitoxin timing group on data through event time -1, extrapolate through all years, and subtract the fitted trend from all data points.[41]

To determine when the antitoxin matters the most in the post-treatment period, I conduct a series of trend-break exercises on the event-study parameters to identify the structural break points (see Figure 6). Drawing from the literature on structural breaks, I select the point that yields the best fitting model for changes in the outcome (Card, Mas, and Rothstein 2008; Goodman-Bacon 2021). I fit a pretend that goes through 0 at time -2,

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[40] I also estimate city-specific linear time trend models. The event-study figures using this specification are presented in Appendix Figures A4 for the child outcome. Event-study and DD estimates (in boxes) using three different specifications are presented in Appendix Figures A5, and A6 for infant and diphtheria outcomes.

[41] I partial out a single pre-trend variable that equals $PPC_c^* \times (c - 1895)$ because there is only one antitoxin timing group. This pre-trend adjustment strategy does not change the trend-break point estimates which will be explained below. Because this detrending strategy produces more precise results, I prefer them to results from unit-specific time trend models.

a phase-in trend that starts somewhere in an interval between -1 to 13,[42] and a post-trend that starts at 14. I select and report the coefficients for the break point that maximizes the joint significance F-statistics on the spline terms. This exercise consistently identifies event-year 1 for the child outcome as the trend-break point.[43] Figure 6 shows event-study and trend-break estimates for both adjusted and unadjusted data.

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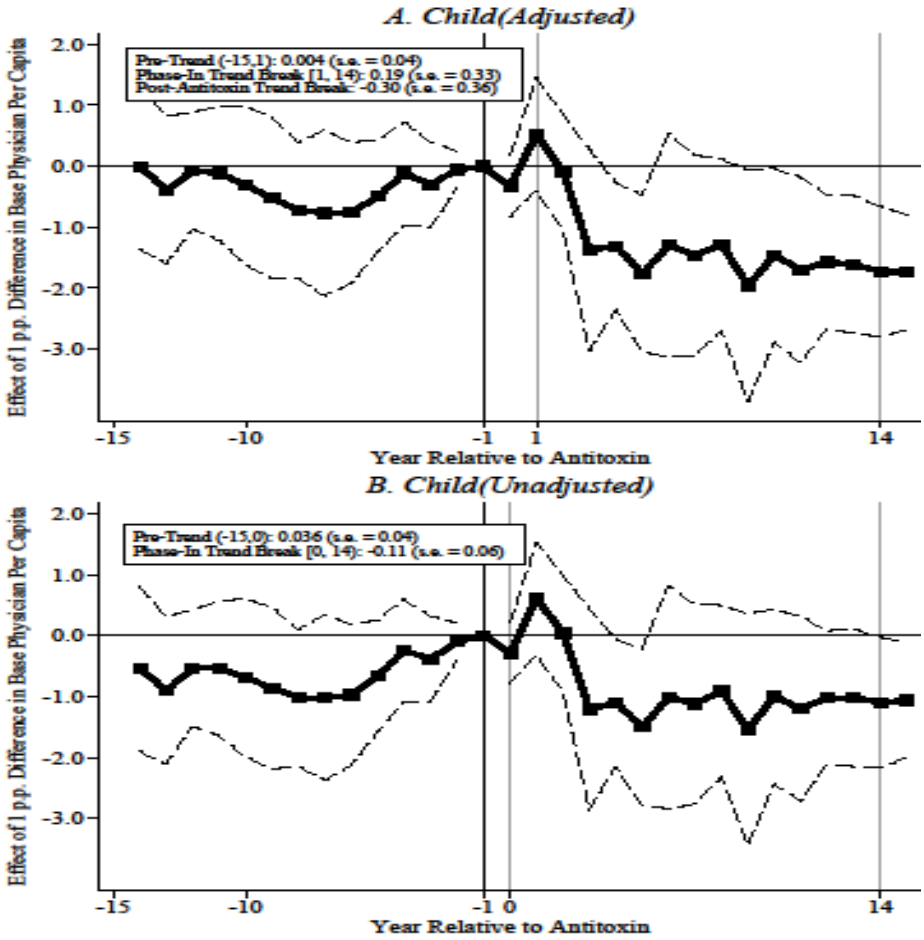
[42] The program loops through different points in [-2,14] for a trend-break, and compares *F*-statistics. The choice of event-year 14 as the starting point of post-trend is arbitrary. By imposing zero at 14, I give enough time after treatment for any lagged break point. Event-year -2 also allows for any arbitrary break just before 1895 which is not related to the antitoxin's introduction. All trend-break points, which are reported as the best fitting structural breaks, occurred in or after event-year 0 and before event year 5. A plot of the corresponding trend-break *F*-statistics are presented in Appendix Figure A10.

[43] The estimated trend-break point for infant outcome is event year 0, and for diphtheria outcome is event year 4. I also run this exercise for typhoid outcome, which is not treated with the antitoxin. The estimated trend-break point for typhoid is year 13. This supports that the PPC design identifies the impact on diphtheria-related mortalities. Trend-break results along with event-study figures for other outcomes are presented in Appendix Figures A7, A8, and A9.

Figure 6. Physicians Per Capita and Child Mortality: Event-study Estimates of Antitoxin's Effect on Child Mortality (coefficients X 100)



Notes: The dependent variable is the log of the child mortality rate. The sample includes 38 cities' mortality series from 1880 to 1910. The figure plots the estimated coefficients on interactions between base physicains per capita (PPC) rates in 1900 and event-time dummies for 15 years before and after 1895. The event-time -1 is omitted. The specification includes year and city fixed effects. The panel A estimates adjust for a linear trend interacted with base PPC for the only antitoxin year, 1895, for event-times prior to -1. Estimates are weighted by city population. Standard errors are clustered at the city level to allow for arbitrary serial correlation within cities. The trend break points come from maximizing the F-statistic on the three terms that use different breakpoints from -2 through 14. A plot of these F-statistics is presented in Appendix Figure A10. Sources: Mortality counts from HathiTrust Digital Library, Google Books, or the archives at the National Library of Medicine in Bethesda, Maryland, and Mortality Statistics. PPC rates from 1900 census occupational classification system, recorded in the IPUMS variable OCC (049:Physicians and Surgeons).

RESULTS

The event-study estimates in Figure 6 show that access to the antitoxin is strongly associated with reduction in child mortality. The trend-break points in panel A, adjusted for a pre-trend (detrended), produces year 1 as the structural break point. Panel B is presented to show that the detrending strategy does not change the estimated trend-break point radically— year 0 is identified as the break point here. The trend-break phase-in shape, starting in the year (or a year from) of the antitoxin's introduction, supports the PPC-based research design. The point estimates do not show an upward or downward trend before the introduction of the antitoxin in 1895—a key test of the design.

Intention-to-Treat Effect of the Diphtheria Antitoxin on Child Mortality

The primary mechanism through which the diphtheria antitoxin should affect mortality is by increasing the utilization of physician's services. Figure 6 presents event-study estimates of the antitoxin's intention-to-treat (ITT) effect on the log mortality rate for children aged under 5. In the 15 years leading to the antitoxin's introduction, estimates are small and indistinguishable from zero. The flat pre-trend strongly supports the PPC-based research design by ruling out differential trends. The estimates show a clear downward trend 2 years after the antitoxin's introduction, persisting until 15 years after 1895. The lagged response in child mortality may be due to the delayed acceptance of the antitoxin by physicians and the slow emergence of its epidemiological impacts on

mortality rates. However, the spike in the year 1896 is likely caused by other factors unrelated to diphtheria.[44]

Table 2 presents grouped event-study estimates in panel A.[45] The estimates trace out the evolution of the antitoxin's effect in each column for a different specification. Although pre-antitoxin estimates are imprecise and similar in magnitude, post-antitoxin estimates outline an increasing negative impact structure vividly. Panel B presents single post-treatment DD estimates. Child mortality fell by about one percent (-1.01, s.e. = 0.46; Table 2, panel B, column 2) for each percentage point difference in base PPC rates. Table 2 shows that the treatment effect of the antitoxin on child mortality is robust to a number of different specifications. Estimates in column 1 are from the simplest specification: controlling for PPC_c^* variable and comparing the slope coefficients between mortality and PPC_c^* across event times. This specification also shows a significant effect of the antitoxin, matching the estimated effect from the full specification (-1.10, s.e. = 0.50; Table 2, panel B, column 1). Additionally, the event-study full specification (column 2) estimates are presented in Figure 6.[46] To consider alternative explanations for changes in mortality rates, I make adjustments to my model in columns 3 and 4 by incorporating

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[44] The event-study figure for diphtheria outcome does not document any jump in mortality for 1896. See Appendix Figure A6.

[45] In an event-study specification, I combine event-time dummies into seven bins {[-15,-11], [-10,-7], [-6, -2], [0], [1,5], [6, 10], [11, 15]}, and a difference-in-difference specification which estimates one post-treatment effect for years [1, 15].

[46] My analysis includes 38 cities, which is enough for avoiding the bias from standard error estimation using a small number of clusters (Bertrand et al. 2004; Cameron et al. 2008). As suggested by Cameron et al., a row in panel B shows two-sided p-values from 1,000 draws of a wild cluster bootstrap percentile t procedure. The standard error clustering and bootstrap procedure are the same across all specifications.

additional factors. While I cannot directly measure living standards, I account for unobserved variables that might influence the quality of life. To achieve this, I use two different proxies for population density and include region-by-year fixed effects. As a result, the estimate experiences a small decrease (-0.92, s.e. = 0.51; table 2, panel B, column 3). Furthermore, when I introduce measures of wealth into my preferred specification, the DD estimate only experiences a slight reduction but remains statistically significant (-0.88, s.e. = 0.52; table 2, panel B, column 4). In the last column, I present the results (both event-study and DD estimates) for infant mortality.[47] There is no significant result for infant, which is aligned with the fact that diphtheria primarily affected children aged between 1 and 5 years. The lack of significant findings for infants aligns with the fact that diphtheria primarily affected children between the ages of 1 and 5 years. Infant mortality includes neonatal deaths (i.e., deaths occurring during the first month after birth) that are less likely to be related to diphtheria.

Robustness Checks

Table 3 presents a series of robustness checks to validate the findings of the main specification event-study and DD estimates in Table 2. Column 1 represents the main specification estimates, serving as a benchmark for comparison (table 2, column 1). In

[47] The negative effect reported for infant mortality would probably driven by post-neonatal mortality (i.e., deaths occurring 1-12 month after birth). In the 1890s there is no evidence of any fundamental change in the access to midwifery services or newborn hygiene practices. So, while neonatal deaths comprised a significant fraction of the death under 1, it is unlikely that neonatal deaths accounted for change in overall infant mortality. Appendix Figure A1 depicts the infant mortality trend.

column 2, the adjustment for the pre-trend exercise is omitted, resulting in a DD estimate that is approximately one-third of the magnitude of the main specification, but still suggesting a negative effect of the antitoxin on child mortality. While these estimates are no longer statistically significant, the event-study estimates remain similar to those in the original specification. As a result, I cannot reject the equality of event-study estimates in the first two columns. The results are robust to weighting by population. Column 3 presents unweighted estimates.[48] Column 4 presents two-stage least squares estimates. The continuous variable constructed using 1870 PPC rates is instrumented for PPC_c^*. The IV result (-1.52, s.e. = 0.65) supports the PPC-based research design. Considering the significant segregation in access to white physicians and the reduction in total mortality driven by white mortality, column 4 provides a result for a different measure of access, i.e., white physicians per capita. This measure produces a more significant and larger estimate (-1.21, s.e. = 0.47).

Given that PPC is a persistent measure, likely unaffected by the introduction of the antitoxin, interpolated PPC for 1895 (column 5) produces estimates that are of the same size and significance as PPC for 1900. Consistent with the findings in previous

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[48] A Hausman test cannot reject the null hypothesis that weighted and unweighted estimates are equal for either grouped event-study estimates (p-value = .26) or the DD estimate (p-value = .75; Deaton 1997). This test is to detect unmodeled heterogeneity or other forms of misspecification, which lead the two estimators to disagree (DuMouchel et al. 1983; Solon et al. 2015). I fail to reject the null of equality. However, I present more precise unweighted estimate for DD model and less precise weighted estimates for event-study model. The reason for these divergent preferences is the fact that p-value for event-study model is less convincing. I choose the more conservative estimates: weighted event-study estimates (Figure 6). DD estimates are similar in magnitude barring the unweighted one is more precise.

columns, the results in column 5 further support the relationship between PPC and the effectiveness of the antitoxin in reducing child mortality.

Table 4 presents the DD estimates examining the association between physicians' certification laws and child mortality, aiming to investigate whether physicians' supply legislations had any impact on mortality rates. The five most binding laws with physicians per capita variable (from Table 1, panel C) are given in Table 5. The most restrictive laws are physician registration law and examination board. There is no evidence of a negative effect of these laws on mortality; instead, they seem to be associated with an increase in mortality rates. As a result, it is unlikely that the negative effect captured by the PPC variable is due to these laws.

Table 2. Reduced-Form Estimates: The Relationship between Base PPC Rates and Log Child Mortality by Specification (coefficients X 100)

	(1)	(2)	(3)	(4)	(5)
	A. Grouped Event-Study Estimates				
Pre-Antitoxin	Child Mortality				Infant Mortality
(Years -15 to -11)× PPC*	-0.27	-0.13	0.19	0.14	0.20
	(0.49)	(0.54)	(0.60)	(0.60)	(0.66)
(Years -10 to -7)× PPC*	-0.73	-0.57	-0.27	-0.31	-0.01
	(0.60)	(0.63)	(0.56)	(0.56)	(0.54)
(Years -6 to -2)× PPC*	-0.41	-0.34	-0.28	-0.28	-0.06
	(0.35)	(0.35)	(0.31)	(0.31)	(0.30)
Post-Antitoxin					
(Year 0)× PPC*	-0.31	-0.33	-0.25	-0.25	0.00
	(0.25)	(0.25)	(0.37)	(0.37)	(0.30)
(Years 1 to 5)× PPC*	-0.94**	-0.80**	-0.51*	-0.50*	-0.68**
	(0.41)	(0.40)	(0.27)	(0.27)	(0.22)
(Years 6 to 10)× PPC*	-1.71**	-1.49*	-1.23**	-1.23**	-1.15**
	(0.80)	(0.81)	(0.49)	(0.50)	(0.40)
(Years 11 to 15)× PPC*	-1.89***	-1.66**	-1.44**	-1.41**	-1.24**
	(0.55)	(0.55)	(0.50)	(0.51)	(0.43)
R^2	0.80	0.88	0.93	0.93	0.91
Joint Significance of Lags (*p* -value)	0.00	0.00	0.05	0.07	0.01

Table 2. CONTINUED

	B. Difference-in-Differences Estimates				
Post-Antitoxin×PPC*	-1.10**	-1.01**	-0.92*	-0.88*	-0.43
	(0.50)	(0.46)	(0.51)	(0.52)	(0.50)
Bootstrap *p* -value	(0.100)	(0.075)	(0.10)	(0.080)	(0.060)
R^2	0.80	0.88	0.93	0.93	0.85
Observations	1,060	1,060	1,012	1,012	1,017
Covariates					
	High-PPC FE, time-to-Antitoxin dummies	City FE, year FE	(2), region-by-year FE, two measures of population density	(3), two measures of wealth	(4), (preferred specification)
Child Mortality Rate in *t**-*1*			784.7		

Notes: The outcome variable is the log of the child mortality rate adjusted for the pretrend (detrended). Panel A tabulates estimated coefficients on the interaction between groups of time-to-Antitoxin dummies (1{t-1895 ∈ [a,b]}) and the continuous variable of base PPC rates. The estimates represent the effect in log points of a one percentage point difference in base PPC rates (multiplied by 100). The estimates are normalized to zero in the year before the antitoxin's introduction. Standard errors, clustered by city, are in parentheses. The model includes city and year fixed effects. The row labeled "Joint Significance of Lags" presents the p-value from an F-test of the joint significance of post-antitoxin coefficients. A Hausman test does not reject the null hypothesis that weighted and unweighted estimates are equal for either the grouped event-study model (p-value .26) or the DD model (p-value .75: Deaton 1997; Solon et al. 2015). In panel B, the p-values from 1000 draws of a wild-cluster bootstrap percentile t procedure are in parentheses (Cameron et al. 2008). * Statistically significant at 10% level; ** at 5% level; *** at 1% level. Sources: See sources for Figure 6.

Table 3. Robustness Check: The Relationship between Base PPC Rates and Log Child Mortality by Specification (coefficients X 100)

	(1)	(2)	(3)	(4)	(5)	(6)
	A. Grouped Event-Study Estimates					
Pre-Antitoxin	Child Mortality					
(Years -15 to -11)× PPC*	0.14	-0.34	0.59	0.84	-0.14	0.28
	(0.60)	(0.60)	(1.73)	(0.62)	(0.57)	(0.69)
(Years -10 to -7)× PPC*	-0.31	-0.63	0.42	0.34	-0.64	-0.19
	(0.56)	(0.56)	(1.06)	(0.79)	(0.66)	(0.64)
(Years -6 to -2)× PPC*	-0.28	-0.41	-0.14	0.16	-0.37	-0.25
	(0.31)	(0.31)	(0.58)	(0.36)	(0.36)	(0.37)
Post-Antitoxin						
(Year 0)× PPC*	-0.25	-0.21	-0.05	-0.63	-0.35	-0.31
	(0.37)	(0.37)	(0.48)	(0.42)	(0.27)	(0.42)
(Years 1 to 5)× PPC*	-0.50*	-0.32	-0.35	-0.87*	-0.92**	-0.51*
	(0.27)	(0.27)	(0.68)	(0.56)	(0.43)	(0.30)
(Years 6 to 10)× PPC*	-1.23**	-0.84*	-1.47	-1.16	-1.77**	-1.18**
	(0.50)	(0.50)	(1.08)	(1.17)	(0.81)	(0.59)
(Years 11 to 15)× PPC*	-1.41**	-081	-1.10	-1.41*	-1.95***	-1.4**
	(0.51)	(0.51)	(1.41)	(0.88)	(0.51)	(0.60)
R^2	0.93	0.87	0.94	0.88	0.89	0.92
Joint Significance of Lags (*p* -value)	0.07	0.41	0.43	0.01	0.00	0.15

Table 3. CONTINUED

	B. Difference-in-Differences Estimates					
Post-Antitoxin×PPC*	-0.88*	-0.24	-0.96**	-1.52**	-1.21**	-0.95'
	(0.52)	(0.51)	(0.37)	(0.65)	(0.47)	(0.61)
Bootstrap *p* -value	(0.080)	(0.490)	(0.007)	(0.070)	(0.004)	(0.060)
R^2	0.93	0.87	0.86	0.88	0.89	0.93
Observations	1,012	1,012	1,012	1,012	1,012	1,012
Covariates	(4) in Tbale 2, (preferred specification)	(1), not detrended (unadjusted)	(1), unweighted	(1), IV using 1870 PPC rates	(1), measured by white physicians per capita	(1), measured by interpolated 1895 PPC
Log Mortality Rate in *t**-1*			784.7			

Notes: The outcome variable is the log of the child mortality rate adjusted for the pretrend (detrended). Panel A tabulates estimated coefficients on the interaction between groups of time-to-Antitoxin dummies (1{t-1895 ∈ [a,b]}) and the continuous variable of base PPC rates. The estimates represent the effect in log points of a one percentage point difference in base PPC rates (multiplied by 100). The estimates are normalized to zero in the year before the antitoxin's introduction. Standard errors, clustered by city, are in parentheses. The model includes city and year fixed effects. The row labeled "Joint Significance of Lags" presents the p-value from an F-test of the joint significance of post-antitoxin coefficients. A Hausman test does not reject the null hypothesis that weighted and unweighted estimates are equal for either the grouped event-study model (p-value .26) or the DD model (p-value .75: Deaton 1997; Solon et al. 2015). In panel B, the p-values from 1000 draws of a wild-cluster bootstrap percentile t procedure are in parentheses (Cameron et al. 2008). * Statistically significant at 10% level; ** at 5% level; *** at 1% level. Sources: See sources for Figure 6.

Table 4. The Relationship between Physicians Certification Laws and Log Child Mortality (coefficients X 100)

	(1)	(2)	(3)	(4)	(5)
	Difference-in-Differences Estimates				
	Child Mortality				
Post-Antitoxin× Certification law*	21.1***	21.7***	-7.2	-1.84	-11.5
	(4.23)	(4.0)	(7.14)	(9.53)	(7.13)
R^2	0.85	0.90	0.87	0.89	0.89
Observations	1,012	1,012	1,012	1,012	1,012
Covariates	(4) in Tbale 2 (preferred specification), interacted with registration laws	(1), interacted with examination board law	(1), interacted with mandated examination law	(1), interacted with mandatory diploma	(1), interacted with inferior school exclusion
Log Mortality Rate in *t*-1*	784.7				

Notes:The outcome variable is the log of the child mortality rate adjusted for the pretrend (detrended). Each column shows estimated coefficient on the interaction between post-antitoxin dummy and the dummy variable of a physicians certification law. The estimates are multiplied by 100. Standard errors, clustered by city, are in parentheses. * Statistically significant at 10% level; ** at 5% level; *** at 1% level. Sources: See sources for Figure 6.

CONCLUSION

The introduction of the diphtheria antitoxin marked a groundbreaking advancement in the history of medical technologies. According to laboratory trials, it had significant potential for curing the disease. The production of this antitoxin involved processes that aligned with the principles of modern medicine development. Nonetheless, historical literature has portrayed the diphtheria antitoxin as somewhat unsuccessful in controlling the disease. While acknowledging the novelty of the treatment, the literature has raised doubts about its overall effectiveness. One of the major challenges to disentangle the causal effect of this intervention on mortality lies in the complexities of historical context, where many other important socioeconomic factors are confounding. Up until recently, the lack of mortality data before 1900 further hindered a systematic analysis on this topic.

Using newly transcribed data, I revisit the topic. My analysis hinges on the premise that the access to the remedy is the key in identifying the effectiveness of the antitoxin. To measure the penetration of the antitoxin in various cities, I utilize the physicians per capita (PPC), as physicians served as the main channel for distributing the antitoxin. Deploying PPC rates ensures that my econometric model captures the differences in mortality rates attributable to differences in access to the antitoxin through physicians. In the sampled cities, physicians per capita rates widely vary, which leads to differences in the availability and accessibility of the antitoxin. Considering that the introduction of the antitoxin occurred during a period of mortality transition, it becomes crucial to carefully account for secular trends. To address this, I incorporate detrending in

my analysis. This entails estimating a pre-treatment trend and extrapolating it into the future, effectively eliminating the need to assume linear time trends. Upon conducting this exercise, I find that the introduction of the antitoxin contributes to the decline in mortality rates for children aged 1 to 5 years. The sensitivity analysis shows promising results.

The results suggest that the antitoxin was important in reducing child mortality due to diphtheria in the US, underscoring the value of this novel medical technology. However, when examining a snapshot of mortality data specifically for children under 15 in 1900, it becomes apparent that an analysis utilizing this age group's mortality data would likely yield even more substantial estimates of the antitoxin's effect.

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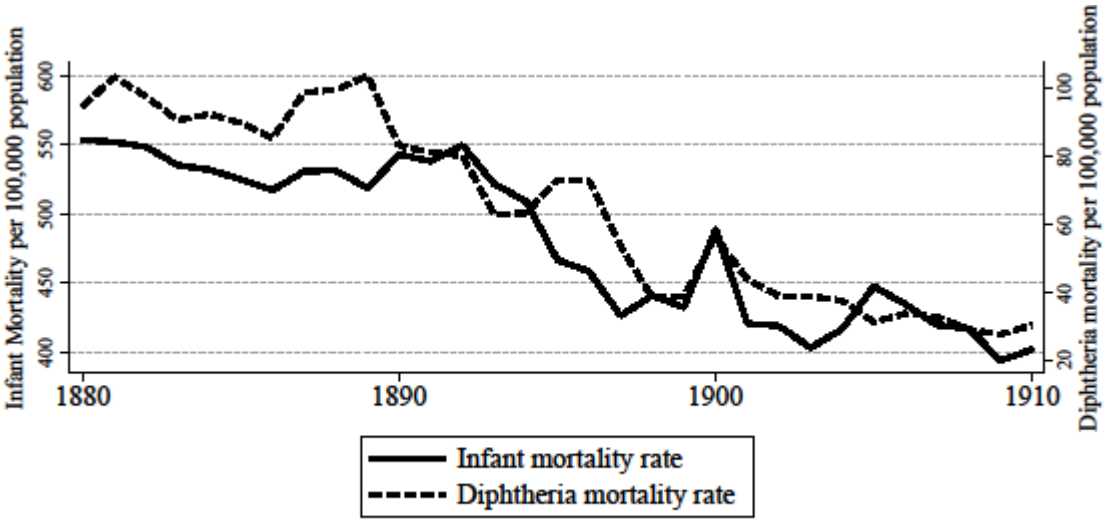
Athey and Imbens (2022) (Jayachandran, Lleras-Muney and Smith, 2010) (Preston and Haines, 1991) (Das et al., 2016) (Badell et al., 2021) (Polonsky et al., 2021) (Costa, 2015) (Smith, 2003) (Daniel, 2006) (Kunitz et al., 2007) (L¨onnroth et al., 2009) (Mercer, 2014) (?) (Fairchild and Oppenheimer, 1998) (Anderson et al., 2019) (Atwood, 2022) (Hammonds, 1999) (Goodman-Bacon, 2018b) (Jacobson, LaLonde and Sullivan, 1993) (Almond, Chay and Greenstone, 2006) (Klass, 2021) (LE, 1913) (Du Bois, 1994) (Du Bois, 2000) (Karp and Gearing, 2015) (Holt et al., 1896) (Crum, 1917) (Kermack and McKendrick, 1927) (Kermack and McKendrick, 1932) (Kermack and McKendrick, 1933) (Adda, 2016) (Cutler and Miller, 2005) (Hoehn-Velasco, 2018) (Hoehn-Velasco and Wrigley-Field, 2022) (Moehling and Thomasson, 2012) (Lee and Solon, 2011) (Bhuller et al., 2013) (Goodman-Bacon, 2018a) (Card, Mas and Rothstein, 2007) (Bertrand, Duflo and Mullainathan, 2004) (Cameron, Gelbach and Miller, 2008) (DuMouchel and Duncan, 1983) (Solon, Haider and Wooldridge, 2015)

APPENDICES

APPENDIX A

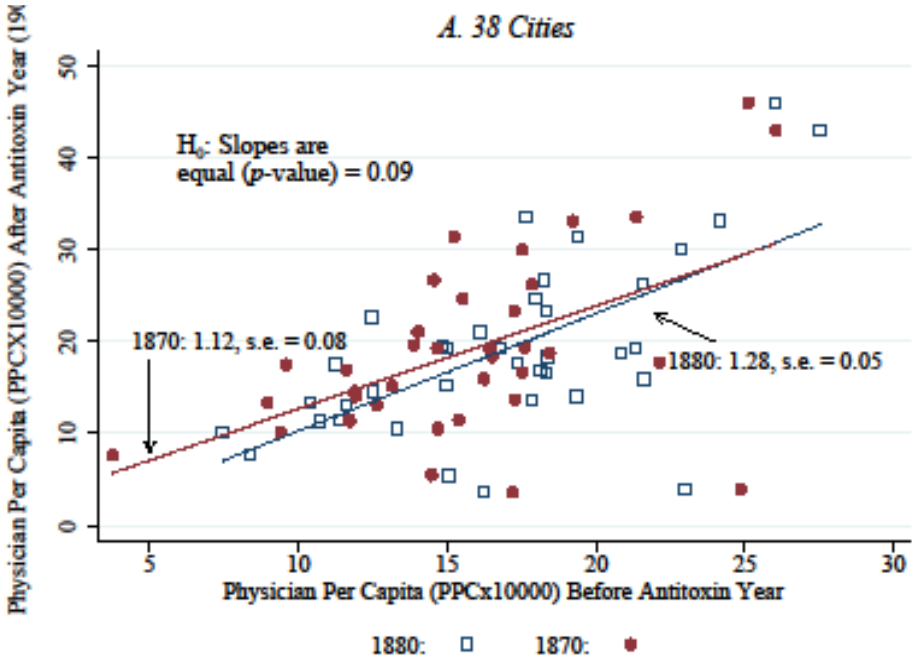
FIGURES AND TABLES REFERENCED

Appendix Figure A1. Infant and Diphtheria Mortality Rates, 1880-1910



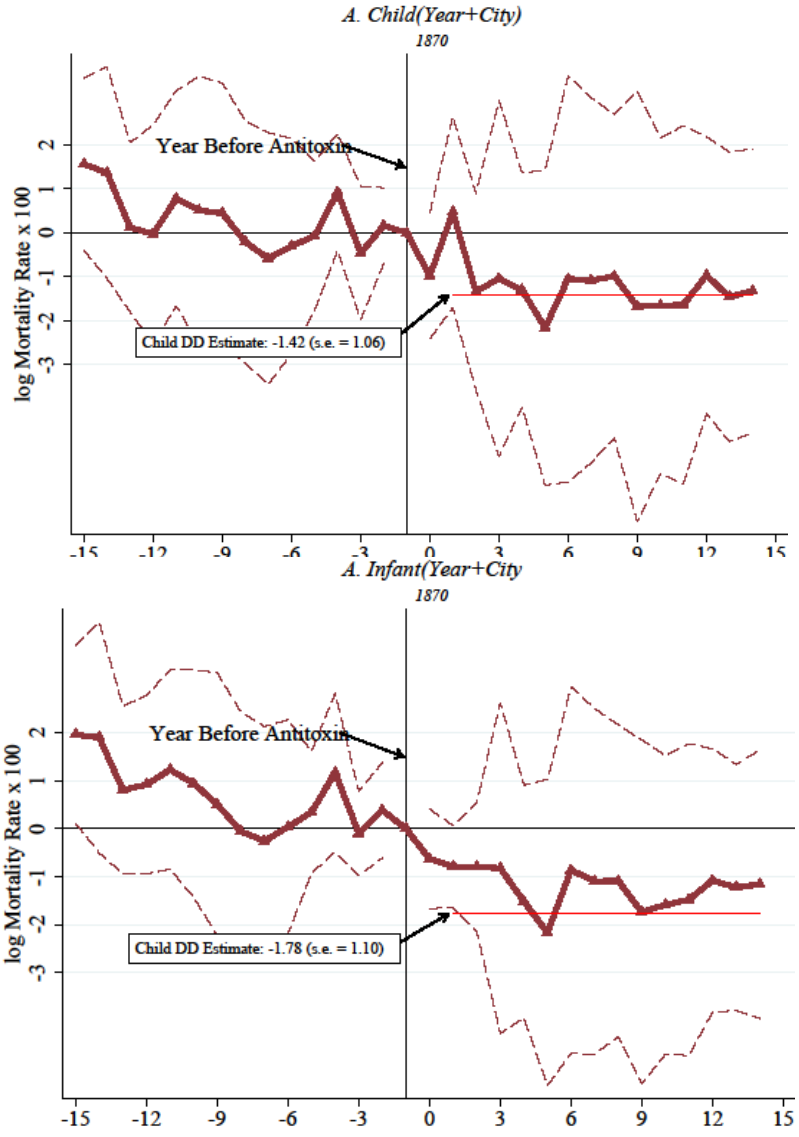
Sources: For 1880-1899, based on annual data from municipal and state public health reports: HathiTrust Digital Library, Google Books, or the archives at the National Library of Medicine in Bethesda, Maryland. For 1900-1910, based on Mortality Statistics, published annually by the U.S. Census Bureau.

Appendix Figure A2. Stability in Cross-City PPC Variation, 1870 and 1880 through 1900



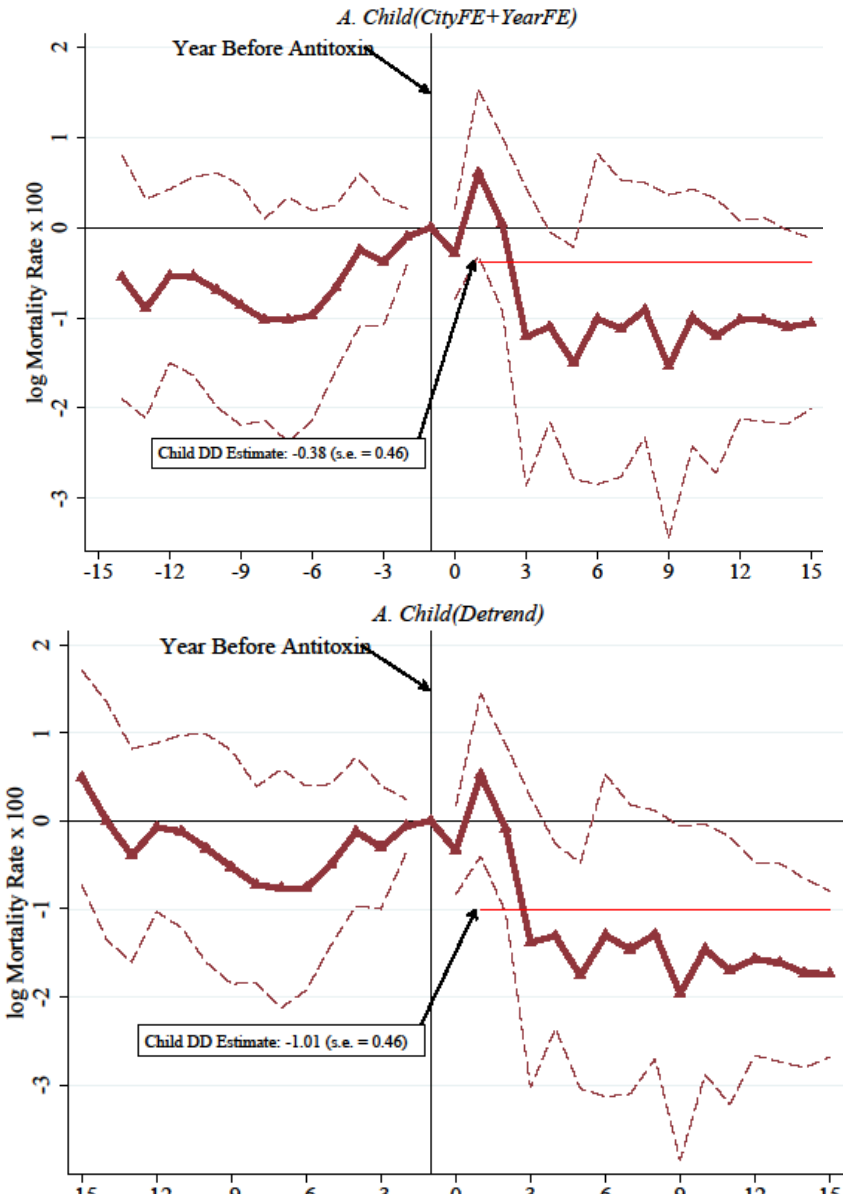
Notes: The figure presents scatter plots and fitted values of the relationship between physicians per capita rates (PPC) in 1900 (y-axis) and PPC rates in 1870 and 1880 (x-axis). The results show that the cross-city variation in PPC rates was remarkably stable over time. Pre-antitoxin PPC rates strongly predict post-antitoxin PPC rates (including PPC rates in the year of the introduction of antitoxin, 1895), and the relationship itself does not change over time.The p-value from a test that the slopes are equal is presented. Rates are multiplied by 10000. Sources: Based on 1870, 1880, and 1900 census occupational classification system, recorded in the IPUMS variable OCC (049:Physicians and Surgeons)

Appendix Figure A3. Event-Study Estimates of The Effetct of Antitoxin on Mortality: Using 1870 Physician Per Capita Rates (coefficients X 100)



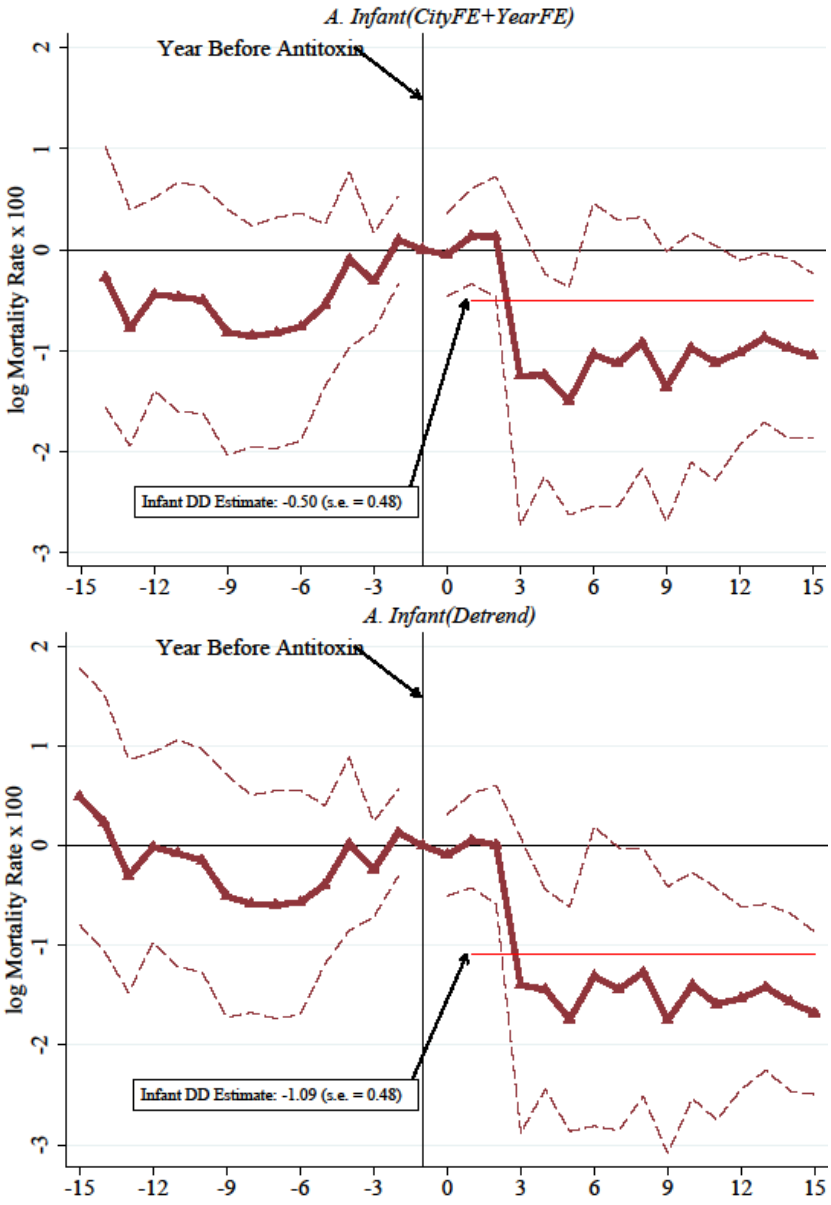
Notes: The dependent variable is the log of mortality rate. The figure plots the estimated coefficients on interactions between base physicians per capita (PPC) rates in 1870 and event-time dummies for 15 years before and after 1895. The event-time -1 is omitted. The specification includes year and city fixed effects. Estimates are weighted by city population. Standard errors are clustered at the city level. Sources: See sources for Figure 6.

Appendix Figure A4. Event-Study Estimates of The Effetct of Antitoxin on Child Mortality by Specification (coefficients X 100)



Notes: The dependent variable is the log of the child mortality rate. The figure plots the estimated coefficients on interactions between base physicians per capita (PPC) rates in 1900 and event-time dummies for 15 years before and after 1895. The event-time -1 is omitted. The first specification includes year and city fixed effects. The second specification adds linear city-specific time trends. The last specification uses detrended data (adjusting for a linear trend interacted with base PPC rates for event-times prior to -1. Estimates are weighted by city population. Standard errors are clustered at the city level. Sources: See sources for Figure 6.

Appendix Figure A5. Event-Study Estimates of The Effetct of Antitoxin on Infant Mortality by Specification (coefficients X 100)



Notes: The dependent variable is the log of Infant mortality rate. The figure plots the estimated coefficients on interactions between base physicians per capita (PPC) rate in 1900 and event-time dummies for 15 years before and after 1895. The event-time -1 is omitted. The first specification includes year and city fixed effects. The second specification adds linear city-specific time trends. The last specification uses detrended data (adjusting for a linear trend interacted with base PPC rates for event-times prior to -1. Estimates are weighted by city population. Standard errors are clustered at the city level. Sources: See sources for Figure 6.

Appendix Figure A6. Event-Study Estimates of The Effetct of Antitoxin on Diphtheria Mortality By Specification (coefficients X 100)

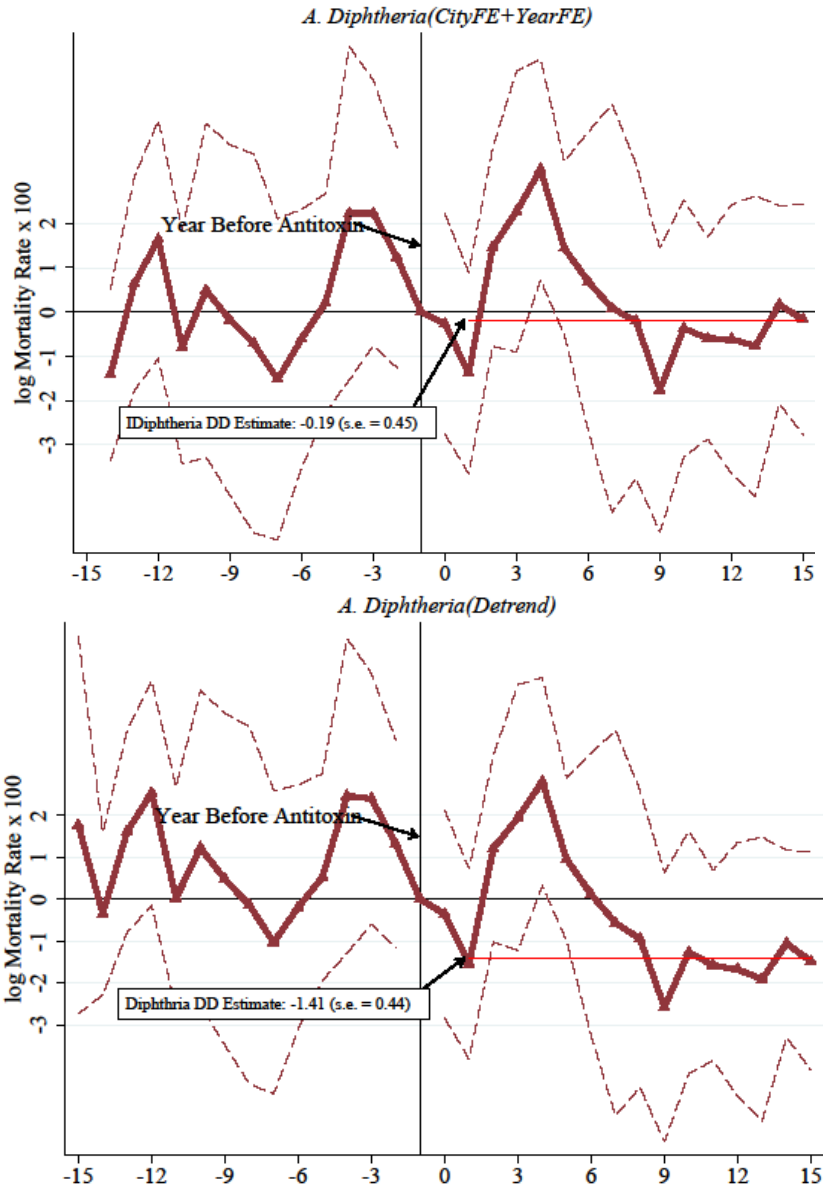
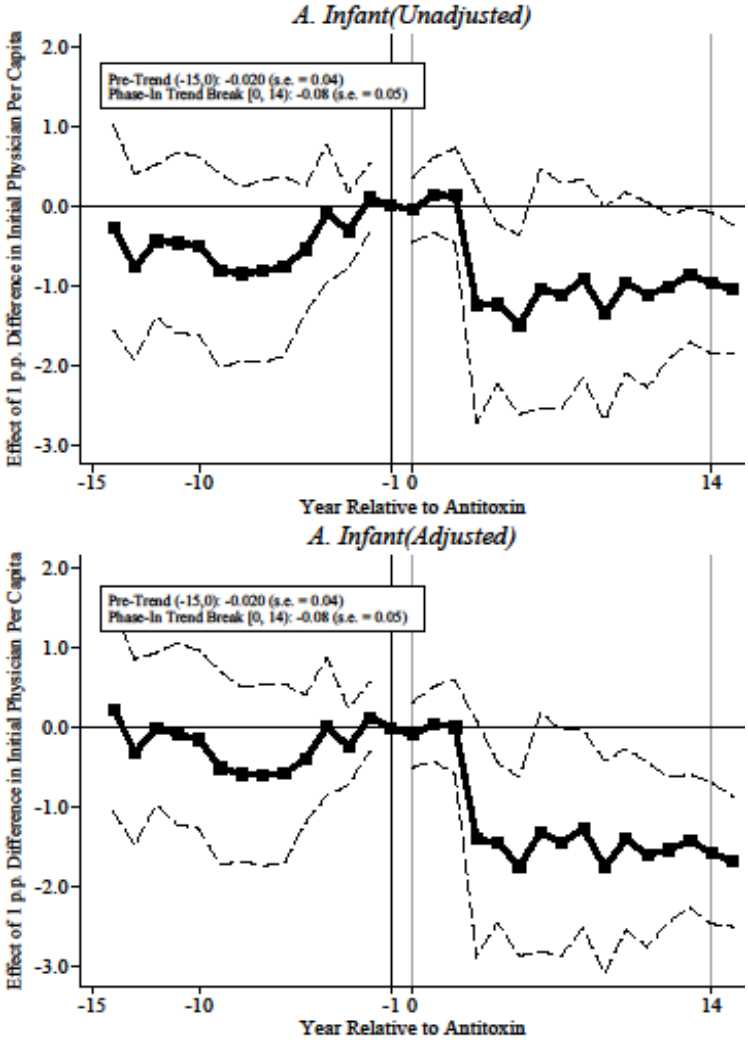


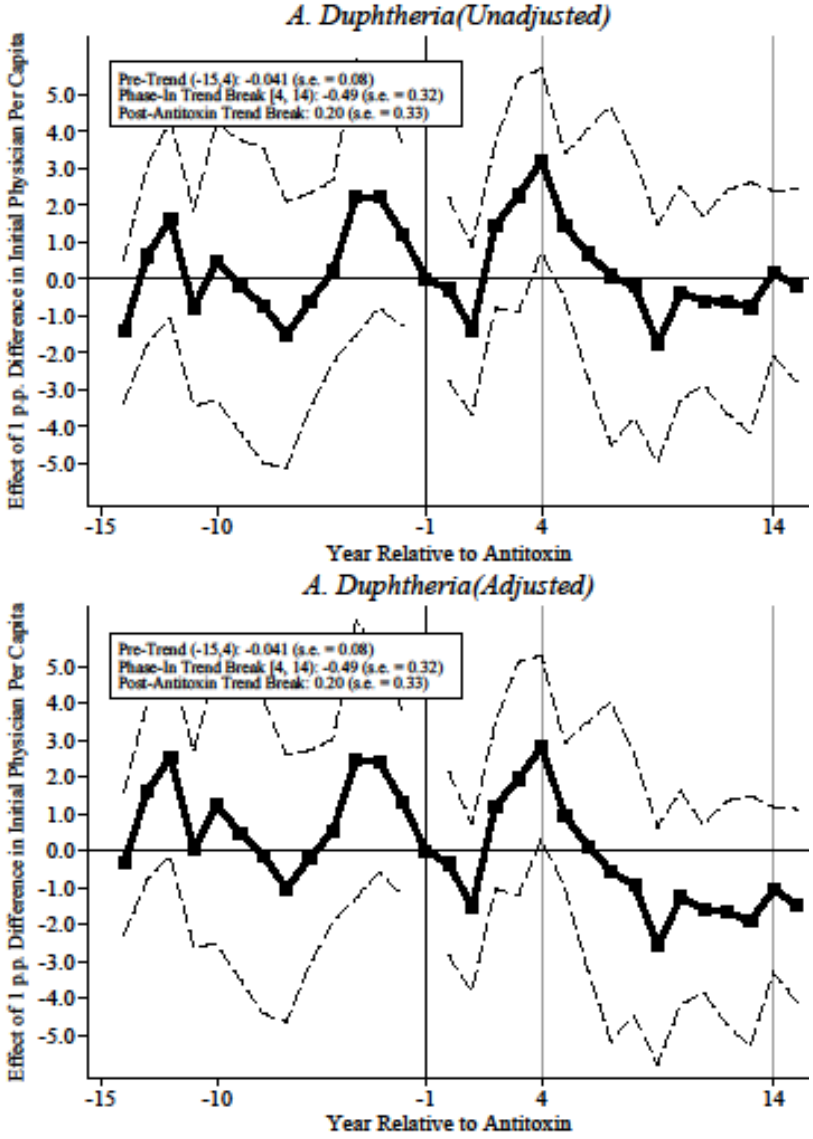
Figure 1 Notes: The dependent variable is the log of diphtheria mortality rate. The figure plots the estimated coefficients on interactions between base physicians per capita (PPC) rates in 1900 and event-time dummies for 15 years before and after 1895. The event-time -1 is omitted. The first specification includes year and city fixed effects. The second specification adds linear city-specific time trends. The last specification uses detrended data (adjusting for a linear trend interacted with base PPC rates for event-times prior to -1. Estimates are weighted by city population. Standard errors are clustered at the city level. Sources: See sources for Figure 6.

Appendix Figure A7. Physicians Per Capita and Infant Mortality: Event-study Estimates of Antitoxin's Effect on Infant Mortality (coefficients X 100)



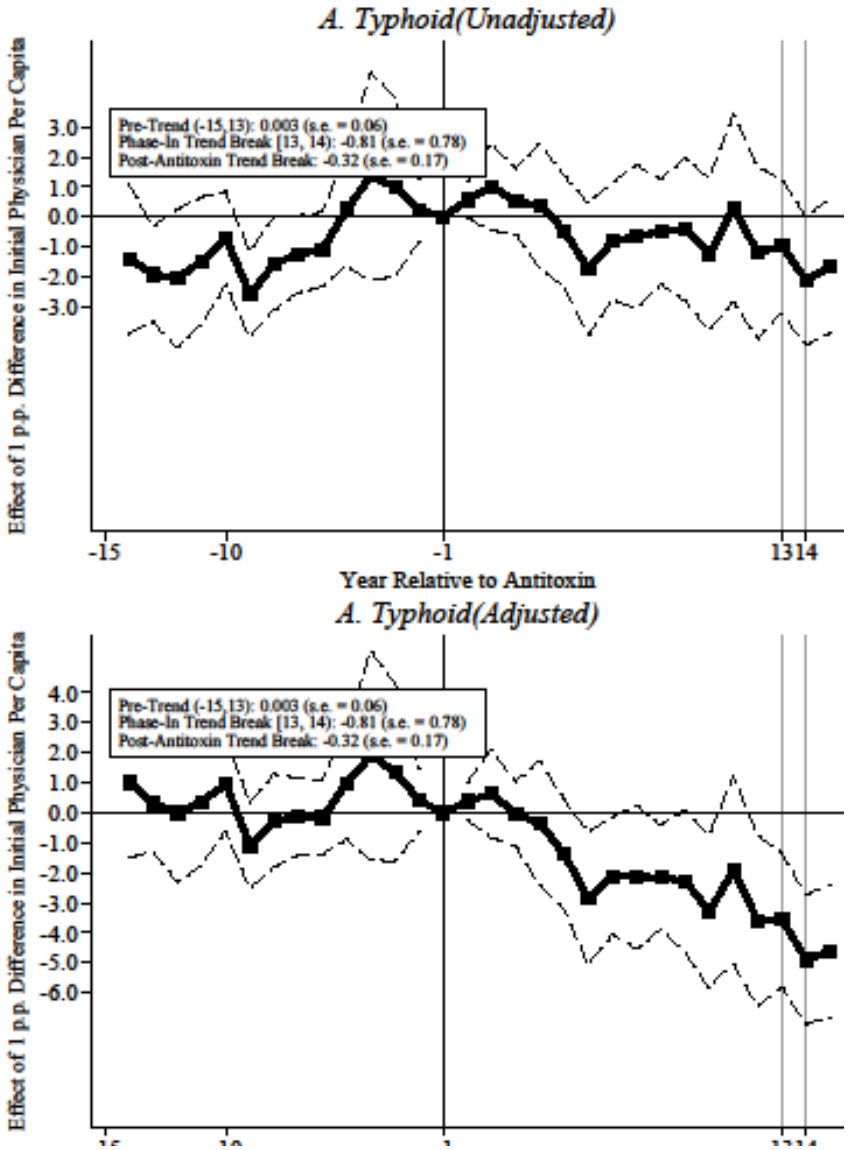
Notes: The dependent variable is the log of infant mortality rate. The figure plots the estimated coefficients on interactions between base physicians per capita (PPC) rates in 1900 and event-time dummies for 15 years before and after 1895. The event-time -1 is omitted. The specification includes year and city fixed effects. In the second panel, estimates adjust for a linear trend interacted with base PPC for the only antitoxin year, 1895, for event-times prior to -1. Estimates are weighted by city population. Standard errors are clustered at the city level to allow for arbitrary serial correlation within cities. The trend break points come from maximizing the F-statistic on the three terms that use different breakpoints from -2 through 14. A plot of these F-statistics is presented in Appendix Figure A10.Sources: Mortality counts from HathiTrust Digital Library, Google Books, or the archives at the National Library of Medicine in Bethesda, Maryland, and Mortality Statistics. PPC rates from 1900 census occupational classification system, recorded in the IPUMS variable OCC (049:Physicians and Surgeons).

Appendix Figure A8. Physicians Per Capita and Diphtheria Mortality: Event-study Estimates of Antitoxin's Effect on Diphtheria Mortality (coefficients X 100)



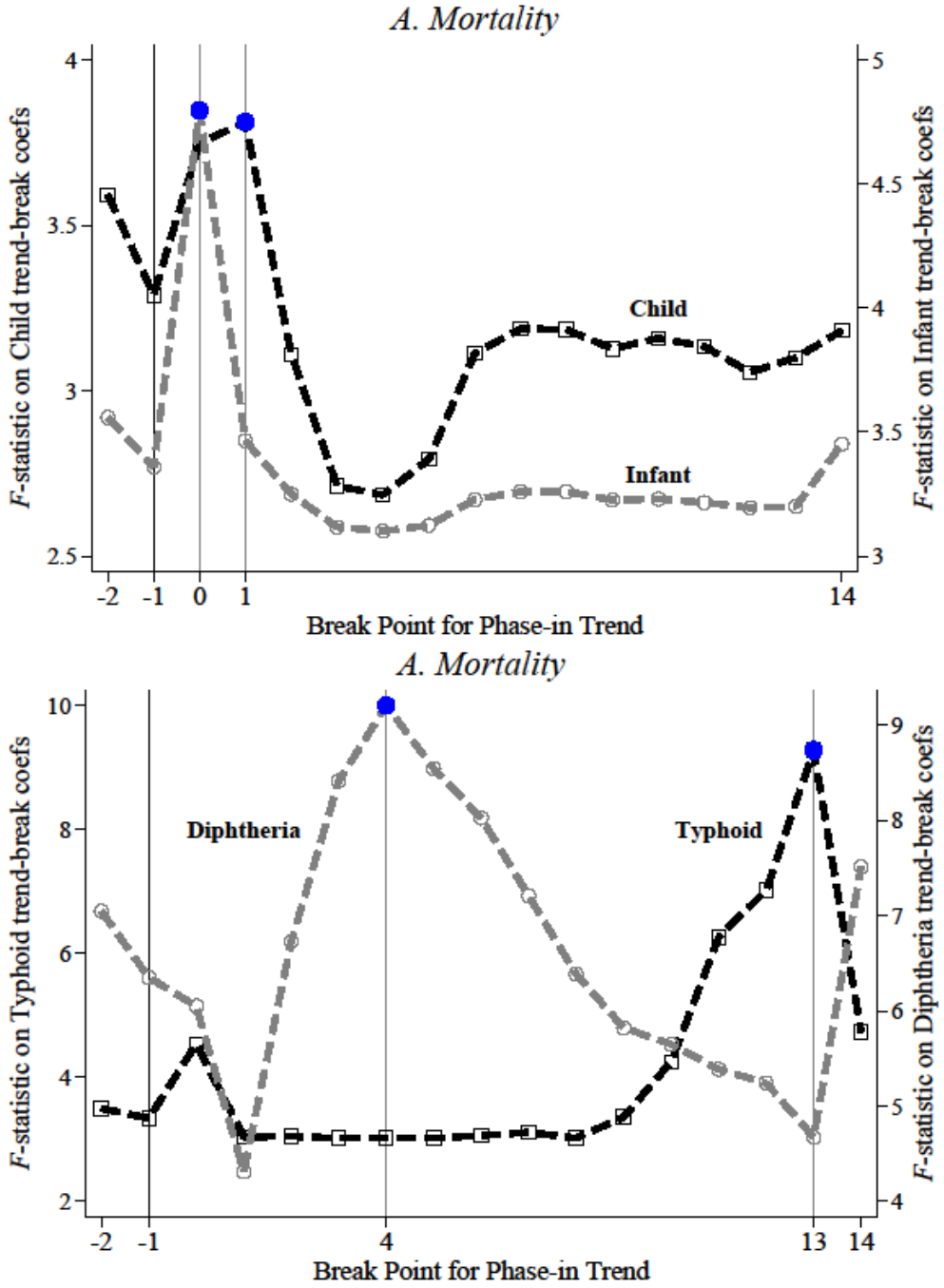
Notes: The dependent variable is the log of diphtheria mortality rate. The figure plots the estimated coefficients on interactions between base physicians per capita (PPC) rates in 1900 and event-time dummies for 15 years before and after 1895. The event-time -1 is omitted. The specification includes year and city fixed effects. In the second panel, estimates adjust for a linear trend interacted with Base PPC for the only antitoxin year, 1895, for event-times prior to -1. Estimates are weighted by city population. Standard errors are clustered at the city level to allow for arbitrary serial correlation within cities. The trend break points come from maximizing the F-statistic on the three terms that use different breakpoints from -2 through 14. A plot of these F-statistics is presented in Appendix Figure A10.Sources: See sources of Appendix Figure A7.

Appendix Figure A9. Physicians Per Capita and Typhoid Mortality: Event-study Estimates of Antitoxin's Effect on Typhoid Mortality (coefficients x 100)



Notes: The dependent variable is the log of typhoid mortality rate. The figure plots the estimated coefficients on interactions between base physicians per capita (PPC) rates in 1900 and event-time dummies for 15 years before and after 1895. The event-time -1 is omitted. The specification includes year and city fixed effects. In the second panel, estimates adjust for a linear trend interacted with Base PPC for the only antitoxin year, 1895, for event-times prior to -1. Estimates are weighted by city population. Standard errors are clustered at the city level to allow for arbitrary serial correlation within cities. The trend break points come from maximizing the F-statistic on the three terms that use different breakpoints from -2 through 14. A plot of these F-statistics is presented in Appendix Figure A10.Sources: See sources of Appendix Figure A7.

Appendix Figure A10. Trend-Break F-statistics, Mortality.



Notes: F-statistics are from the joint significance test of the event-year variable, its interaction with a dummy for event-years greater than or equal to x (where x is given by the x-axis in the figure), and its interaction with a dummy for event-years less than or equal to -2.

Appendix Table A1. Municipal Antitoxin Availability Years

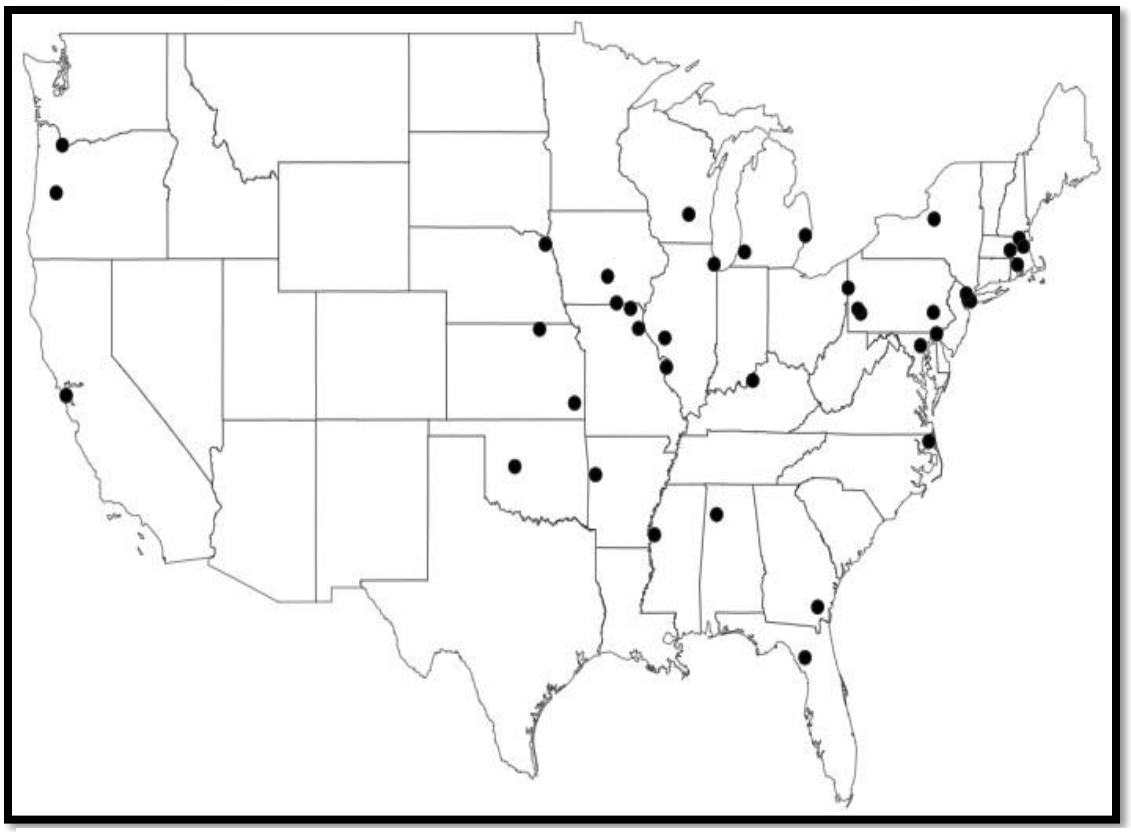
State	City	Antitoxin
CA	San Francisco	1896
CT	Hartford	1895
CT	New Haven	1895
DC	Washington	1895
GA	Atlanta	1895
IL	Chicago	1895
LA	New Orleans	1895
MA	Boston	1895
MD	Baltimore	1895
MN	St. Paul	1895
MO	St. Louis	1895
NE	Omaha	1895
NJ	Camden	1895
NJ	Newark	1895
NY	Buffalo	1895
NY	New York	1894
OH	Cleveland	1895
OH	Dayton	1895
OH	Toledo	1895
PA	Philadelphia	
PA	Scranton	1895
TN	Memphis	1895
VA	Richmond	1895
WI	Milwaukee	1895
OH	Cincinnati	1895
PA	Pittsburgh	1895
MI	Detroit	1895
NJ	Jersey city	
KY	Louisville	
RI	Providence	
IN	Indianapolis	

Sources: Newspaper archives and historical medical journals.

APPENDIX B

FIGURES AND TABLES NOT REFERENCED

Appendix Figure B2. Locations of Cities in Sample



Notes: Sample includes 38 cities in 18 states.

Appendix Figure B2. Diphtheria Mortality Trend for 4 Major Cities

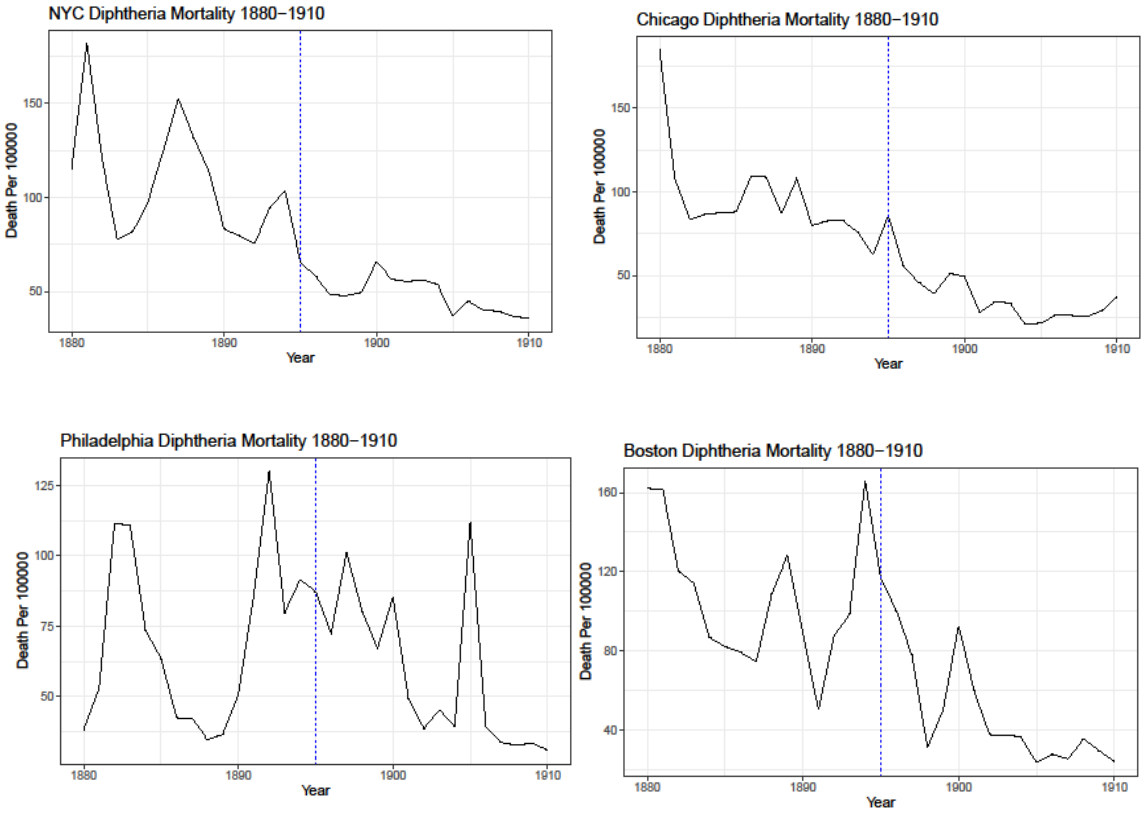


Figure 3 Appendix Table B1. Ranking of PPC Rates on 1870 and 1900

City, State	Physicians Per Capita Ranking in 1900	Physicians Per Capita Ranking in 1870
San Francisco, CA	1	2
Washington, DC	2	1
Chicago, IL	3	5
Boston, MA	4	6
Baltimore, MD	5	20
Hartford, CT	6	10
Cleveland, OH	7	23
Philadelphia, PA	8	8
St. Louis, MO	9	18
Cincinnati, OH	10	13
Somerville, MA	11	38 (missing)
Camden, NJ	12	25
Waterbury, CT	13	26
Springfield, IL	14	9
Pittsburgh, PA	15	22
New Bedford, MA	16	16
New York, NY	17	7
Lawrence, KS	18	37 (missing)
St. Paul, MN	19	15
Reading, PA	20	4
Lynn, MA	21	33
Bridgeport, CT	22	32
New Haven, CT	23	11
Portland, ME	24	17
Worcester, MA	25	27
Jersey City, NJ	26	30
Manchester, NH	27	29
Providence, RI	28	12
Paterson, NJ	29	35
Cambridge, MA	30	28
Elizabeth, NJ	31	19
Newark, NJ	32	31
Lowell, MA	33	21
Hoboken, NJ	34	34
Fall River, MA	35	36
Trenton, NJ	36	24
Memphis, TN	37	3
Charleston, SC	38	14