Medical Technology and Life Expectancy: Evidence from the Antitoxin Treatment of Diphtheria*

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Abstract

We study the impact of the first effective medical treatment for an infectious disease—diphtheria antitoxin—on the historical health transition in the United States. Using an instrumental variable for local antitoxin adoption rates and information from approximately 1.6 million death certificates from 1880 to 1914, we find that the rapid diffusion of antitoxin led to a substantial decline in diphtheria mortality rates and increased life expectancy at birth. Exposure to antitoxin also significantly reduced school absenteeism. Overall, our results suggest that medicine played a more important role in increasing life expectancy in the early 20th century than previously thought.

Keywords: Life expectancy; medical technology; antitoxin.
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1 Introduction

Life expectancy at birth has risen dramatically in the United States since the late 19th century. A child born in the 21st century can expect to live nearly 30 years longer than a child born in the 1880s. The U.S. health transition began in the late 19th century, when improved nutrition and public health measures reduced the prevalence and mortality of infectious diseases, which were the main causes of death at the time (Cutler et al. 2006; Costa 2015). These health improvements significantly increased life expectancy before medical innovations such as antibiotics and better medical care continued to increase life expectancy beginning in the late 1930s (e.g., Jayachandran and Lleras-Muney 2009; Jayachandran et al. 2010).[1] It is widely believed that medical innovations did not play a major role in the decline of infectious diseases and improvements in life expectancy before the late 1930s (Cutler 2005; Acemoglu and Johnson 2007; Catillon et al. 2018).

This paper studies the relationship between medical innovations, the decline in infectious diseases, and improvements in life expectancy *before* the onset of "the era of big medicine" in the 1930s.[2] We focus on the first widely-used medical treatment against an infectious disease: the diphtheria antitoxin. In 1890, German physiologist, Emil von Behring invented the antitoxin serum, and, in the late fall of 1894, the production of an antitoxin serum to treat diphtheria patients began in the United States (Preston and Haines 1991; Hammonds 1999).[3] Known as "the strangling angel of children", diphtheria was one of the deadliest infectious diseases for children at that time. In 1900 it contributed to about 2 percent of the crude death rate in the United States. Between 1900 and 1920, the annual diphtheria mortality rate fell significantly (Crum 1917). Contemporary observers have attributed this decline in death rates to the success of treating diphtheria patients with the antitoxin serum, but the historical demographic literature questions the effective use of the diphtheria antitoxin in the early 20th century (Preston and Haines 1991; Condran 2008; Thomasson 2018). Hence, even if the effectiveness of the antitoxin serum was clinically documented, its quantitative impact on population health is ex-ante unknown.

[1] There is an ongoing debate about the factors that led to the mortality decline from infectious diseases in the early 20th century (e.g., Anderson et al. 2019, 2022; Clay et al. 2020). These include clean water technology and sanitation (e.g., Cutler and Miller 2005; Alsan and Goldin 2019), nutrition (e.g., Fogel 1994, 2004; McKeown 1976), living conditions (e.g., Ager et al. 2024), public-health programs (e.g., Moehling and Thomasson 2014; Egedesø et al. 2020), and environmental factors (e.g., Barreca et al. 2016; Beach and Hanlon 2018; Hanlon et al. 2021; Hanlon 2022).

[2] Cutler et al. (2006) refer to the 1930s, with the introduction of sulfonamides ("sulfa" drugs), as the onset of the "era of big medicine".

[3] The New York City Department of Health produced the first diphtheria antitoxin outside of Europe in the late fall of 1894 (Liebenau 1987; Hammonds 1999).

Our goal is to evaluate whether the introduction of the antitoxin serum made a significant contribution to the historical health transition in the United States. To do so, we leverage newly collected municipality data on the diffusion of the diphtheria antitoxin, plus approximately 1.6 million death certificates in Massachusetts dating from 1880 to 1914, to study whether the treatment of diphtheria with the antitoxin serum contributed to life expectancy gains before the era of big medicine. Using Massachusetts as a case study to evaluate the population health effects of the diphtheria antitoxin has several advantages. First, Massachusetts provided the antitoxin to *all residents* free of charge, thereby reducing the possibility of an income gradient in its adoption. This free-supply policy has subsequently been regarded as a milestone in the public health history of the State. Second, the State Board of Health (henceforth the SBH) kept a record of the number of antitoxin bottles distributed to each municipality from the beginning of the campaign in 1895 to 1914. Compared to existing studies that evaluate the effects of medical innovations in the 1930s and 1940s, in our study, we can measure the uptake of new medical technology at the local level.[4] Third, historical vital statistics from Massachusetts have been well-documented and are considered reliable.

We use individual death certificates to calculate life tables, from which we obtain measures of life expectancy for over 250 municipalities in Massachusetts for each year from 1880 to 1914. This data allows us to examine whether the rapid diffusion of the diphtheria antitoxin led to a substantial increase in life expectancy at the municipality level, and a decrease in age-specific mortality rates, such as infant and child mortality. One further advantage of our study is that we can evaluate the short- and long-term consequences of exposure to the antitoxin at the individual level. By combining the antitoxin roll-out with the complete-count U.S. census records, we can test whether exposure to the antitoxin during childhood had any impact on school attendance. Furthermore, by following a linked sample of boys and girls, who lived in Massachusetts during their childhood, to adulthood, we can analyze whether antitoxin exposure during childhood affected labor market outcomes later in life.

The main challenge when estimating the effect of the antitoxin treatment on population health is reverse causality (the demand for medical treatment is higher during epidemics) and omitted-variable bias. In order to circumvent this identification problem, our empirical strategy exploits that the free distribution of the diphtheria antitoxin led to a relatively rapid diffusion of this medical innovation in Massachusetts (Figure 1). We leverage this sharp increase in the adoption rates and the fact that some municipalities stood to benefit more from the antitoxin serum in terms of potential mortality reductions, as these locations

[4]Previous larger-area studies on the effects of the 1930s and 1940s medical innovations are not able to measure the local diffusion of the technologies directly and rely on indirect measures (e.g., Acemoglu and Johnson 2007; Jayachandran et al. 2010; Alsan et al. 2021; Bhalotra et al. 2023).

were historically more widely affected by diphtheria (Figure 2). The differential diphtheria mortality rates across municipalities before the introduction of the antitoxin treatment and the rapid diffusion of this medical innovation allow us to construct an instrumental variable for the observed antitoxin adoption rates at the municipal level. In the reduced form, our strategy corresponds to an intensity of treatment design, which has been applied in previous work that has studied the effect of significant health improvements (e.g., Acemoglu and Johnson 2007; Ager et al. 2018; Bütikofer and Salvanes 2020). We also show estimates based on a slightly modified identification strategy, which in essence is more directly related to a classical shift-share instrument, where the aggregate supply of antitoxin bottles is distributed according to the pre-antitoxin municipality diphtheria mortality shares.

Importantly, we also conduct two falsification exercises showing that: (i) our treatment measure is *not* predictable of changes in diphtheria mortality rates and life expectancy prior to the availability of antitoxin; and (ii) that municipalities with a substantial uptake in the antitoxin treatment after 1895 were *not* already on a different path of the health transition in the 1880s. Our results are also robust to the following: (i) the inclusion of the pre-antitoxin mortality environment; (ii) other public health interventions (the provision of clean water and hospitals) and the pre-antitoxin distribution of doctors per capita; (iii) accounting for children's age structure; (iv) different functional forms; and (v) excluding (or controlling for the distance to) Boston (see Sections 5.3 and 5.4 for details). We also show that our "antitoxin effect" does not simply capture affected municipalities expanding their healthcare sector (by increasing the number of doctors and nurses per capita). Overall, our results suggest that the availability of the diphtheria antitoxin improved the mortality environment of municipalities in Massachusetts.

Our main findings are that the roll-out of the antitoxin serum contributed to a substantial decline in diphtheria mortality rates and improved life expectancy in early 20th-century Massachusetts. In our baseline specification, we find that antitoxin can explain 27 percent of the observed increase in life expectancy from 1894 to 1914. We provide evidence that child mortality was reduced and that antitoxin can explain 17 percent of the observed decline over this period. Thus, our result suggests that the adoption of the diphtheria antitoxin increased life expectancy at birth because it lowered child mortality rates, which is consistent with the pre-antitoxin age profile of diphtheria mortality. There is no strong evidence that our effects are gender specific or that the roll-out of antitoxin significantly changed fertility behavior. We also find robust second-order effects on stroke mortality and small detectable effects on other infectious diseases. Given the health complications of diphtheria (e.g., secondary pneumonia and myocarditis), these findings are not surprising, but their effect sizes are ex-ante unknown. This shows that a "general equilibrium" analysis like ours, which takes into

account phenomena such as co-mortality and competing risk, is important when assessing the effects of new medical innovations on population health. In other words, although it is clinically documented that antitoxin was an effective medicine, it is ex-ante unclear how much it mattered for the historical mortality transition. Our insights suggest that antitoxin played a substantial role in improving population health before the era of big medicine.

In the final part of our empirical analysis, we evaluate the short- and long-term effects of the antitoxin treatment at the individual level. Our empirical strategy is based on an intention-to-treat framework since we do not have information on whether an individual received the treatment. Since the antitoxin serum was the most effective for children under 10 years of age, we focus on their exposure to the antitoxin. We first consider whether the antitoxin treatment affected school attendance. Absenteeism from school due to sickness in the late 19th century was common, and it is expected that the antitoxin treatment not only prevented deaths from diphtheria but also reduced other childhood illnesses.[5] To identify the effects, we ask whether children attended school for more months if they were exposed to the antitoxin treatment in their municipality at ages 0-9.[6] Our results, based on approximately 350,000 children, suggest that this was the case. Children exposed to the antitoxin were almost 5 percent (relative to the sample mean) less likely to attend school for three months or fewer—a sizable effect. However, the fewer days absent from school for antitoxin-exposed children did not translate into any detectable long-term effects on years of schooling and adult labor market outcomes.

Our paper contributes to a revived debate on whether medical advances played a major role in the health transition before WWII. Conventional wisdom holds that technological progress in medicine and better medical care are not the key drivers of the decline in mortality rates and the gains in life expectancy during this period (Cutler 2005; Acemoglu and Johnson 2007; Catillon et al. 2018). However, this notion has been challenged by recent studies. Hollingsworth et al. (2022) find that a large-scale hospital modernization program in North Carolina that began in the late 1920s significantly reduced infant mortality. Other studies show that the wide availability of sulfa drugs in the late 1930s and the mass production of penicillin in the mid-1940s contributed to the decline in the rates of infectious diseases and maternal mortality, plus increased life expectancy at birth (Thomasson and Treber 2008; Jayachandran et al. 2010; Alsan et al. 2021). However, none of these papers evaluate whether medical advances contributed to the historical health transition already in the first

[5]This could be because the antitoxin treatment directly improved the health of treated individuals, or that it contributed to a more effective containment of diphtheria and reduced the spread of the bacteria more generally.

[6]We can test whether antitoxin exposure reduced absenteeism from school because the 1900 US Census recorded how many months a child of school age attended school during the year.

two decades of the 20th century when infectious diseases started their long-term decline. We show that the diphtheria antitoxin was an effective treatment for diphtheria infections and resulted in substantial gains in life expectancy at birth.

Our finding, that the free distribution of the diphtheria antitoxin substantially reduced child mortality rates in late 19th and early 20th century Massachusetts, relates to the ongoing debate about the causes of the mortality decline during the early phase of the historical health transition. The reductions in child mortality attributable to antitoxin—17 percent from 1894 to 1914—is a quantitatively sizable effect, alongside effect sizes of studies focusing on safe water, sewerage, and other public health efforts during this period in the US (e.g., Beach et al. 2016; Alsan and Goldin 2019; Clay et al. 2020). Our study demonstrates that the diphtheria antitoxin was far more effective than previously assumed (Preston and Haines 1991; Condran 2008; Thomasson 2018), and that medical technological progress contributed to the decline in the rate of infectious diseases much earlier than is generally believed.

Finally, our individual-level results speak to a large literature on the short- and long-term consequences of improvements in the mortality environment of children. Our short-term results show some parallels to development studies that evaluate the effect of health campaigns on school absenteeism (e.g., Miguel and Kremer 2004). In terms of examining the long-term consequences of medical innovations in a historical context, the closest to our study are the papers by Jayachandran and Lleras-Muney (2009), who show that declines in maternal mortality rates in Sri Lanka in the mid-20th century translated into improvements in adult female life expectancy and human-capital skills, and Bhalotra and Venkataramani (2015), who find that the introduction of sulfa drugs in the US in 1937 stimulated human capital accumulation and the economic mobility of affected children as adults. Economists also assessed the long-term effects of public health initiatives and find generally positive impacts on education and labor market outcomes of the affected cohorts (e.g., Bleakley 2007; Bütikofer and Salvanes 2020; Atwood 2022). In contrast to these studies, we find no detectable long-term effects of the antitoxin treatment.

2 Background

In this section, we first provide a brief introduction to diphtheria. We then focus on the development of the antitoxin serum and its distribution throughout Massachusetts. Finally, we discuss the need for a proper identification strategy to evaluate the results of contemporaneous studies that highlighted the (non-)effectiveness of the antitoxin serum.

2.1 A brief introduction to diphtheria

Diphtheria is a contagious bacterial infection that mainly affects the upper respiratory tract, but it can also spread to other areas of the body. The bacterium that causes diphtheria—*Corynebacterium diphtheriae*—produces a toxin that can cause severe damage to the body's tissues and organs. Transmission occurs from person to person via respiratory droplets from coughing or sneezing, as well as via contaminated food products. Symptoms include general weakness and a swollen neck.[7] Left untreated, diphtheria can obstruct the airways and cause suffocation. Other complications include secondary pneumonia, myocarditis (inflammation of the heart muscle) and neuritis (nerve inflammation). These complications can be life-threatening (causing strokes and heart attacks) and may lead to long-term health problems, such as paralysis and dysphagia. If the initial infection is treated immediately, these complications and sequential diseases can be avoided.[8]

Diphtheria emerged as a notable cause of death in the U.S. during the second half of the 19th century (e.g., Preston and Haines 1991). It was one of the most deadly infectious diseases, along with influenza, pneumonia, tuberculosis, and diarrhea, and accounted for about 2 percent of the crude death rate in the U.S. in 1900. The cumulative number of diphtheria deaths in the 10 largest U.S. cities during the pre-antitoxin years 1889-93 was approximately 40,000, which at the time was equivalent to the complete annihilation of the population of a medium-sized city like Brockton, MA. The annual death rate from diphtheria in these cities was nearly 1.2 deaths per 1,000 people. Boston was close to this average, with a mortality rate of 1.18 deaths per 1,000 people (Crum 1917).[9] By the end of the 19th century, diphtheria was still epidemic in rural areas but endemic in the urban areas on the East Coast (Hammonds 1999).

In Massachusetts, the vital records indicate that diphtheria accounted for up to 10% of all deaths during the peak years in the 1870s. Diphtheria, known as "the strangling angel of children", was mostly a childhood disease. In Massachusetts in 1890, approximately 85% of all diphtheria deaths affected those under the age of 10. Diphtheria accounted for 18% of the deaths in children aged 1 to 10, while diphtheria under the age of 1 accounted for only 0.4% of the deaths that year. The age distribution of diphtheria deaths is similar in the U.S. death-registration area. Diphtheria was somewhat more deadly for boys under the

[7]The *Corynebacterium diphtheriae* bacterium had been discovered by Edwin Klebs in 1883 and was related to the disease by Friedrich Löffler in 1884. It was later known as the Klebs-Löffler bacillus (Barksdale 1970).

[8]For further details, see also the descriptions by the *Centers for Disease Control and Prevention* (CDC) and Hadfield et al. (2000).

[9]In Germany, the country where the antitoxin was invented, diphtheria mortality rates were at similar levels (106 per 100,000 people) during this period.

age of 5, whereas girls between the ages of 5 and 9 were at slightly higher risk (Crum 1917). The records from Massachusetts also show that people died from diphtheria throughout the calendar year, but that death rates were generally higher between late fall and spring, making the disease similar to others, such as pneumonia and influenza, in terms of seasonality.

The number of deaths caused by diphtheria (and croup - an infection of the upper airway) in Massachusetts, despite significant annual variations, started to steadily decline from around 3,200 a year in the mid-1870s to less than 700 in 1914 (see Figure A.3).[10] In the 1910s, diphtheria mortality accounted for less than 1% of all deaths. Although the disease was not as deadly as other infectious diseases, such as tuberculosis, the skewed mortality-age profile suggests that a reduction in diphtheria deaths would have had a significant impact on life expectancy at birth. In the following two subsections, we discuss whether the introduction of the antitoxin serum played an important role in the decline of diphtheria mortality rates at the turn of the 20th century.

2.2 Antitoxin and its distribution in Massachusetts

In 1901, Emil von Behring received the first Nobel Prize in Medicine for his work on serum therapy, particularly for discovering an antitoxin treatment for diphtheria. Together with Shibasaburo Kitasato, he developed the antitoxin serum in Germany in 1890—less than a decade after identifying the Klebs-Löffler bacillus as the cause of diphtheria. The antitoxin serum was the first effective drug to treat an infectious disease.[11] It was produced by injecting a horse with many small doses of the toxin until a high concentration of the antitoxin built up in the horse's blood, producing the so-called "antiserum". Doctors then used this serum as a therapy for treating diphtheria patients. The success of the antitoxin treatment contributed to the success of bacteriology within medicine and improved the public image of doctors (Preston and Haines 1991; Rothstein 1992; Condran 2008).

The widespread diffusion of the diphtheria antitoxin took place after its effectiveness had been demonstrated at the International Congress of Hygiene and Demography in Budapest in 1894. In the U.S., the production of antitoxin started in New York City in the late fall of 1894, followed by Philadelphia and Boston (Liebenau 1987; Hammonds 1999). The SBH in Massachusetts began preparing for production and started distributing antitoxin *free of charge* throughout the state in March 1895, using its production facilities in Boston. Accord-

[10]Diphtheria was separately classified as a cause of death in the vital registration reports in Massachusetts starting in 1858.

[11]The smallpox vaccination, for example, had been used for immunization purposes since the beginning of the 18th century (e.g., Ager et al. 2018), but it could not be used as a treatment once an individual was infected by the disease.

ing to the Massachussets State Board of Health (1902, p.491), *"the serum has been distributed throughout the state wherever it has been called for, to local health boards, contagious diseases hospitals and to physicians in private practice, the latter being usually supplied through the local boards of health."*

The SBH kept records of the number of antitoxin bottles distributed to municipalities in Massachusetts and published these numbers in their annual reports from 1895 to 1914. The diphtheria antitoxin was produced at the laboratory of the SBH at Forest Hills (MA).[12] Production of the serum increased rather quickly from 1,724 bottles in 1895 to 53,389 in 1900. Over the next 14 years, production more than doubled and the SBH distributed 118,561 bottles of antitoxin in 1914, which corresponded to close to 14 bottles per 1,000 people.[13] We digitized these reports and use the statistics in our empirical analysis below. Our analysis ends in 1914 because the SBH stopped publishing data on the distribution of antitoxin after that. At this time, advances were also being made in the development of a diphtheria vaccine, and eventually, a diphtheria toxoid was developed in the 1920s, allowing mass immunization against diphtheria possible. This toxoid, along with some refinements, is still in use today (e.g., Plotkin 2014). Hence, we are estimating the effect of the antitoxin treatment during a time when medical immunization against diphtheria was not yet available.

2.3 The effectiveness of the antitoxin

How effective was the treatment of diphtheria with the antitoxin at the beginning of the 20th century? Contemporary publications report various diphtheria mortality statistics for selected areas throughout the world, both before and after the development of the antitoxin in the 1890s. According to Crum (1917), the pre-antitoxin (typically 1889-93) diphtheria mortality rates varied from 18.8 (Ireland) to 411.9 (Serbia) deaths per 100,000 people, while during the antitoxin period (typically 1910-14), the rate per 100,000 people varied from 40.1 (Serbia) to 6.8 (Chile). Several countries, including the U.S., experienced a large decline in diphtheria mortality rates during this period. In Massachusetts, the fatality rate of diphtheria fell from 28.3 percent (1891-94) in the pre-antitoxin era to 13.1 percent in the antitoxin era

[12] In the first years, the production of the serum was carried out at the laboratory rooms in the Bussey Institution at Harvard University.

[13] According to the SBH annual reports (various years), each bottle contained 15-20 cubic centimeters of serum in 1895. The strength of the serum gradually increased over time, which resulted in a decrease in the amount of serum per bottle (each containing 1,500-2,000 units). The quantity of treatment given to patients varied from less than 1,000 units to more than 20,000 units (equivalent to 0.5-13 bottles per treatment), however, 54% of patients received less than 5,000 units. Small doses were sometimes given to the family members of infected patients (particularly siblings), as the antitoxin provided short-lived immunization against the disease.

(1895-1901). The SBH estimated that the antitoxin treatment saved 10,697 lives in these seven years (Massachussets State Board of Health 1902, p.487). These results demonstrated the enthusiasm of medical contemporaries at the time regarding the breakthrough in treating diphtheria infections with the serum. Yet, diphtheria mortality rates remained high at the beginning of the 20th century, possibly due to unequal access, the inefficient deployment of the antitoxin serum, and the stage of the disease at which patients received the antitoxin treatment as the fatality of diphtheria increased if the serum treatment was substantially delayed (e.g., from 6.6% on the first two days of illness to 17.8% on the sixth day of illness or later (Massachussets State Board of Health 1902, p.486)).[14]

Looking at the total number of deaths caused by diphtheria (and croup) in Massachusetts from 1858 to 1914, we cannot (visually) detect a clear trend break in the time series after the free distribution of the antitoxin in 1895 (see Figure A.3). However, we also observe that the gradual decline in the number of diphtheria deaths since the mid-1870s was not caused by changes in the population at risk, as the mortality rate for diphtheria followed a similar trend. This means that simply comparing the mortality rate for diphtheria before and after the introduction of the antitoxin in 1895, as has been done in previous studies (e.g., Crum 1917), may not accurately reflect the importance of the antitoxin in the decline of diphtheria. Hence, without proper data at the local or individual level, and random variations in the way the antitoxin was administered, it remains a challenge to causally identify the effectiveness of the antitoxin treatment.

Our detailed municipality-level data allow us to evaluate whether the free and widespread distribution of the antitoxin in Massachusetts after 1895 contributed to historical health transition by reducing the mortality rate from diphtheria and increasing life expectancy at birth. Before outlining our identification strategy, we describes the data used in this study.

3 Data

Our empirical analysis draws on five main data sources: (i) the "*The Annual Report on Birth, Marriages, and Deaths in Massachusetts*" from 1880 to 1914; (ii) annual death registers and certificates from 1880 to 1914; (iii) "*The Annual Report of the State Board of Health of Massachusetts* from 1895 to 1914; (iv) the complete-count U.S. Census records (1880,

[14]Thomasson (2018) mentions that the antitoxin treatment did not readily diffuse, citing a 1907 State Board of Health report from Indiana. Similarly, Preston and Haines (1991) argues that the still high diphtheria mortality rates at the start of the 20th century indicate limited effectiveness in the deployment of the antitoxin. Anecdotal evidence further suggests unequal access to antitoxin at the turn of the 20th century. Illustrative examples include the story about how science "conquered" diphtheria here or the history of diphtheria in Canada here.

1900 and 1910) from IPUMS (Ruggles et al. 2021) and municipality-level statistics from Massachusetts' State Censuses (1880, 1885, 1895, 1905, and 1915) from Haines (2022); and (v) newly publicly available crosswalks of linked individuals across Censuses from the Census Tree Project (Price et al. 2021; Buckles et al. 2023). Except for (iv) and (v), we collected, digitized, and cleaned the data.

The history of Massachusetts' vital records (source i) is well documented and the death registration system (starting in 1842) is generally considered reliable and of high quality. By 1900, only around one percent of all deaths were unregistered. The decline in unknown causes of death towards the end of the 19th century further reveals that data on the causes of death increased substantially in accuracy (Gutman 1956). We digitized the annual mortality statistics by including causes of death (referred to as *"diseases"* hereafter), as well as the number of live births for each municipality from 1880 to 1914. Our main disease variable is the number of deaths from diphtheria.[15] In addition, we use statistics on the following diseases in our analysis: bronchitis, digestive diseases (diarrhea, cholera, and typhoid), tuberculosis of the lungs (TB), pneumonia, scarlet fever, whooping cough, and strokes (apoplexy and cerebral hemorrhage), plus accidental deaths.

The calculation of annual age-specific mortality rates at the municipality level is based on individual death certificates (source ii). These records have been digitized and are provided by *FamilySearch.org* as part of the collection "Massachusetts Deaths, 1841-1914". For the sample period 1880 to 1914, the records include 1,633,553 deaths in total. We derive infant mortality rates by dividing the number of infant deaths by birth counts. Mortality rates for children aged 1-4 are obtained by dividing the death counts by the corresponding population of this age group (which is imputed based on births and cumulative deaths for each age cohort).[16] Our annual age-specific mortality rates (up to the age of 100 years) are calculated as death counts over the population of that age group. The Appendix A.1 provides further details on how we use and tabulate information from the death certificates.[17]

[15]From 1880 to 1901, these deaths are reported in the category "diphtheria and croup", while from 1902 to 1912 they are reported separately as "diphtheria" and "diphtheritic croup", and for 1913 and 1914 they are reported as only one category "diphtheria" (but this also contains deaths from croup). Therefore, we have constructed our diphtheria variable such that it includes deaths from diphtheria and (diphtheritic) croup in all years.

[16]We use the same approach as Alsan and Goldin (2019). For each age from 1 to 4, we impute the population stock by subtracting the cumulative deaths for an age cohort from the births for the age cohort. Note, this approach assumes that there is no in- or out-migration of young children.

[17]In the raw digitized death certificates, there is a number of death records with a missing age after the year 1905. We assign these age-missing deaths into age-specific deaths in proportion to the age distribution for all death records with non-missing ages in the same year, sex, and municipality. In addition, we only assign age-missing deaths with a documented spouse to ages above 15 years.

Using the age-specific mortality rates, we can construct municipality-specific life tables for every year in our sample. For this calculation, we assume deaths to be equally distributed across the calendar years for all ages, except for the first year of life, where it is assumed that an infant death corresponds to one-third of a life year lived. We close the table at age 100 by calculating the life years lived as one divided by the mortality rate at age 100. From the life tables, we can compute life expectancy at all ages, but we focus on life expectancy at birth as our main measure of population health. We further use the age-specific mortality rates as outcomes in the regression analysis.

We also collected and digitized data on the supply of antitoxin (source iii) to each municipality from the annual reports of the SBH from 1895 (the start of the antitoxin campaign) to 1914 (the last year when this information was published). These reports contain information on the number of bottles supplied to each municipality (see also Section 2.2). If municipalities were not listed, they did not receive any antitoxin directly from the SBH.[18] From the SBH reports, we also collected annual data (1891-1914) on the number of infections (cases) for the following diseases: diphtheria, scarlet fever, typhoid fever, measles, and smallpox. The coverage increased from 68 municipalities in 1891 to around 300 municipalities at end of our sample period. Since the SHB mentions that their case data likely suffer from under-reporting, these findings should be interpreted with caution.

Municipality population data (1880-1915) are based on the Massachusetts State Census records and complete count U.S. census records from IPUMS (source iv). Population is interpolated linearly between the census years to construct annual mortality rates. The State Census of Massachusetts was taken every 10 years starting in 1855 and contains detailed population statistics (included in Haines (2022)), as well as information about manufacturing, agriculture, and commercial activity which we digitized (not included in Haines (2022)) at the town level. Haines (2022) also collected a special tabulation of the 1880 U.S. Census for Massachusetts with detailed population statistics at the municipality level, which is also included in our empirical analysis. From Haines (2022), we also obtain a number of other municipality characteristics, such as the number of dwellings, rooms within dwellings, population density, and the share of foreign-born individuals.

We also use the complete count U.S. census records to measure population size by age groups, which we use to construct life tables (the 1890 records were lost in a fire). The aggregation of the census data at the municipality level is based on geo-referenced crosswalks

[18]However, the possibility of redistribution of antitoxin from listed to non-listed municipalities cannot be excluded according to the SBH. As a baseline, we assume that non-listed municipalities *did not* receive any antitoxin bottles. We also replace extremely large per capita values with the 95 percentile value ("winsorized" values). Our conclusions are generally robust to not making this adjustment, however, it increases the precision of our estimates.

of individuals from Berkes et al. (2021), which contain the geographic coordinates for every census-designated location. For every individual listed in the census, the crosswalks contain the historical individual-level identifier (HISTID) provided by IPUMS together with the geo-referenced location of the individual. The crosswalks are merged with the complete-count census records by HISTID to construct different municipality-level characteristics, such as population by age. We linearly interpolate population by age between the census years.

Finally, we construct a linked sample (1900-1940) of individuals based on the newly publicly available crosswalks from the Census Tree Project (https://www.censustree.org/). The Census Tree (CT) contains publicly available crosswalks between decennial censuses based on the 1850-1940 complete count U.S. census records provided by IPUMS. Compared to existing publicly available crosswalk files (the Census Linking Project (CLP) and the IPUMS Multigenerational Longitudinal Panel project (MLP)) that also contain links of individuals between historical U.S. Census records,[19] the CT archives a substantially higher match rate (over 70% for men and over 60% for women) including systematic links for women in non-adjacent censuses (e.g., 1900-1940). The quality of the CT links is high and were independently verified (Buckles et al. 2023).

4 Estimation strategy

In this section, we outline our estimation strategy. Our baseline sample period contains annual observations at the municipality level from 1880 to 1914. The sample ends in 1914 because of data availability on the supply of antitoxin. We start the empirical analysis by estimating the relationship between the local adoption of the antitoxin and population health, as outlined by the following equation:

$$y_{mt} = \beta \, antitoxin_{mt} + \mu_m + \mu_{ct} + \mathbf{X}'_{mt}\Gamma + \varepsilon_{mt}, \tag{1}$$

where y_{mt} is some measure of population health (e.g., life expectancy, mortality rates by disease or age) in municipality m at year t. Our main focus is on the diphtheria mortality rate and life expectancy at birth, but later we also report estimates for infant and child mortality as well as other diseases, infections, and fertility. The municipality-specific adoption of the antitoxin is given by $antitoxin_{mt}$, which is antitoxin bottles per 1,000 people supplied to a municipality m in year t by the SBH. Since the SBH started its supply of antitoxin in

[19] For more details about the existing crosswalks, see https://censuslinkingproject.org/about/ for the CLP (contains only links for males–the match rate ranges between 20-30%) and https://usa.ipums.org/usa/mlp/mlp.shtml for the MLP (contains links for males and females but only for adjacent census years–the match rate is 55% for males and 42% for women).

1895, this variable is by construction zero for all municipalities beforehand. Municipality and county-by-year fixed effects are denoted by μ_c and μ_{ct}, respectively. The county-by-year fixed effects flexibly account for county-specific trends in the outcome variable. The vector $\mathbf{X}'_{mt}$ contains various pre-antitoxin municipality characteristics interacted with year fixed effects. The set of controls includes the pre-antitoxin mortality rates from other infectious respiratory and waterborne diseases and strokes, distance to Boston, measures of population density and overcrowding, and the foreign-born share (all measured in 1880 and interacted with a full set of year-fixed effects). The regression is weighted by the municipality population size in 1895, hence, estimates reflect changes for the average person in Massachusetts.[20] The error term is ε_{mt} and standard errors are clustered at the municipality level. Summary statistics are reported in Appendix Table A.1.

While estimating equation (1) controls for time-invariant differences across municipalities and time-varying differences across counties, the least-squares estimate of β is likely biased due to reverse causality and omitted variables. For example, the demand for antitoxin in a municipality is likely higher during a diphtheria outbreak. If this bias is sufficiently strong, it might even seem as if the antitoxin treatment *reduced* population health when estimating β with least squares.

We address this identification problem by using a two-stage least squares (2SLS) method. Our estimation strategy exploits the fact that antitoxin became suddenly and freely available in 1895 and that some municipalities stood to benefit more from this development than others, as they were systematically more severely affected by diphtheria prior to the availability of the antitoxin.[21] A similar empirical strategy is applied in studies such as Acemoglu and Johnson (2007), Bleakley (2007), and Bütikofer and Salvanes (2020). Compared to these studies, specific data on the local uptake of the medical innovation is available in our setting (i.e., the number of antitoxin bottles distributed), which allows us to estimate the following first-stage equation:

$$antitoxin_{mt} = \gamma treatment_m \times I_t \times (t - 1894) + \mu_m + \mu_{ct} + \mathbf{X}'_{mt}\Omega + \epsilon_{mt}, \tag{2}$$

where $treatment_m$ is average pre-antitoxin (1891-94) diphtheria mortality rate,[22] which is

[20]The other reason why our regressions are weighted by population size is that we calculate annual life tables for all municipalities, including those with smaller populations. It is evident that life expectancy will be measured with error, in particular for smaller areas. Regressions weighted by population place less emphasis on these observations.

[21]For example, we observe in our mortality data that diphtheria was more widespread in urban municipalities, which is to be expected given that the disease is airborne. In rare cases, milk products can also serve as a source for transmission.

[22]The direct effect of $treatment_m$ is absorbed by municipality fixed effects. We document the

our cross-sectional measure of treatment intensity, I_t is a post-1895 indicator, and $(t-1894)$ is a linear trend. The remaining variables are defined as in an estimating equation (1). Both $antitoxin_{mt}$ and I_t are zero for all municipalities prior to the uptake of the antitoxin campaign in 1895. The estimated coefficient of interest, $\hat{\gamma}$, quantifies how differences in the pre-antitoxin diphtheria mortality rates translate into differences in the adoption speed of the antitoxin. The linear-trend specification is motivated by the gradual adoption of antitoxin in Massachusetts (see Figure 1).[23] If $\hat{\gamma} > 0$, this would imply that municipalities more affected by diphtheria prior to 1895 have a higher adoption speed of the antitoxin treatment when the technology became available. The sharp cut-off date in 1895 is also used to conduct falsification exercises in which we show that our instrument measure cannot explain changes in diphtheria mortality and life expectancy in the *pre-antitoxin* period.

We also estimate a model in which the mortality outcome is by year, municipality, and the cause of death. In this "stacked model", the main right-hand-side variable in equations (1) and (2) is interacted with an indicator for diphtheria—the disease expected to be most influenced by the supply of antitoxin. The disease dimension brings an additional source of variation to the empirical model, which allows us to control for interaction fixed effects (municipality-by-year, cause-of-death-by-year, and cause-of-death-by-municipality) and, thus, reduces omitted variable concerns further. The key identification assumption for this stacked specification is common trends in the *difference* in the mortality rate of diphtheria, and other diseases across municipalities with different treatment intensities, if the distribution of the antitoxin treatment did not happen.

Finally, we obtain the same conclusions using an alternative formulation of the instrument in which the aggregate supply of bottles is distributed according to pre-antitoxin municipality diphtheria mortality shares (i.e., a shift-share type of instrument). Appendix Section A.2 provides a detailed explanation of this alternative instrument and its relationship to that used in equation (2).

5 Results

5.1 Descriptive evidence

First, we present descriptive evidence on the evolution of diphtheria mortality rates, the evolution of life expectancy at birth, and antitoxin adoption rates in Massachusetts by treat-

spatial distribution of treatment intensity in Figure 2, which also gives an impression of the distribution of municipalities included in the baseline sample.

[23] As a robustness check, we also deploy a concave trend-break ("logged year"), which matters for the magnitudes of the counterfactual calculations, but not for the magnitude of the 2SLS estimates.

ment intensity (above/below median) for the years 1880 to 1914. Panel A of Figure 3 shows the development of the diphtheria mortality rate as a three-year moving average for municipalities grouped by *treatment intensity*. In Panel B, we additionally subtract the 1894 diphtheria mortality rates. While the figure shows a steady decline in diphtheria deaths from 0.9 to 1.4 per 1,000 people in 1880 to around 0.25 deaths per 1,000 people at the end of the sample, convergence in the rate across groups mainly occurred during the post-antitoxin era. This pattern suggests that municipalities with higher pre-antitoxin diphtheria mortality rates benefited more from the free supply of antitoxin starting in 1895.

Panels C and D of Figure 3 present the evolution of life expectancy, following the same structure as in Panels A and B. Population-weighted average life expectancy increased from 40 years at the beginning of the 1880s to 47 years in the 1910s (not reported). As with the diphtheria mortality rate, we only see convergence across the two municipality groups *after* the introduction of the antitoxin treatment in 1895—the gap in life expectancy remained constant at about six years from 1880 to 1894 and reduced to approximately two years by 1914.[24] We obtain a similar pattern in Appendix Figure A.4, where we first collapse municipalities into the two groups of treatment intensity and then calculate the life tables for each group, which avoids the problem of calculating life tables for small populations.

Figure 1 displays the supply of antitoxin per 1,000 people in Massachusetts since 1895. The population-weighted average supply was close to 14 bottles per 1,000 people at the end of our sample period (not reported), and we also observe that municipalities more affected by diphtheria prior to the antitoxin treatment (the above-median sample) adopted more antitoxin in per capita terms throughout the antitoxin period. Overall, when considering the evolution of diphtheria and life expectancy at birth altogether, we conclude that municipalities with the potential to benefit more from antitoxin also adopted more of it. These municipalities experienced larger declines in diphtheria mortality rates and larger increases in life expectancy at birth. We exploit these features more systemically in our regression analysis in the next subsections.

5.2 Determinants of the antitoxin diffusion

We begin the regression analysis by studying how the adoption of antitoxin is related to different pre-antitoxin municipality characteristics. This exercise is based on different versions of the estimating equation (2), which we later use as the corresponding first stage in the

[24]Note, while we do not use the level of life expectancy (but only changes) in our empirical analysis, we nonetheless obtain reasonable estimates. For example, we find that the population-weighted average life expectancy in Massachusetts in 1890 was 41.4 years and 44.2 in 1901, while is quite close to the official contemporary statistics (Glover 1921).

2SLS framework. The estimates are reported in Table 1. All regressions include municipality and county-by-year fixed effects and are weighted by population size in 1895. The baseline estimate, reported in column 1, implies that 10 years into the use of the antitoxin serum, the adoption rate is close to three bottles per 1,000 people in a municipality with an average treatment intensity. If the treatment intensity is increased by one standard deviation, this number would be approximately 3.3 bottles per 1,000 people. This point estimate is statistically significant at the 1% level.

Column 2 of Table 1 shows that this pattern of adoption is robust to controlling for the mortality rate from other respiratory infectious and waterborne diseases (i.e., bronchitis, tuberculosis, scarlet fever, pneumonia, whooping cough, measles, digestive, and typhoid) and the stroke mortality rate. These measures are constructed in a similar way as "treatment" by averaging over the pre-antitoxin years 1889 to 1894 (measured in logarithmic units). This result suggests that our "treatment" measure is not simply capturing the general mortality environment of a municipality, which, at that time, was mainly driven by infectious diseases. The remaining columns control for medical doctors per 1,000 people, distance to Boston, population density (persons per 1,000 square miles), crowding (persons per dwelling and per room), and the foreign-born share (all measured in 1895, interacted with the indicator and a linear trend). The rate of antitoxin adoption was higher in places with more medical doctors and in more densely and overcrowded municipalities where infectious diseases could spread more easily (see e.g., Ager et al. 2024).[25] While the magnitude of the treatment effect reduces to approximately 1.4 bottle per 1,000 people (after 10 years) in the most conservative specification (column 7), the point estimate remains statistically significant at the 1% level. Finally, Appendix Table A.2 reports the results of a pre-antitoxin balance test, in which we observe that treatment is significantly correlated with the mortality rate of other infectious diseases and the number of physicians per capita.

5.3 Antitoxin and population health

Before we incorporate the insight from Table 1 (that some municipalities had more to benefit from with the adoption of antitoxin), into our 2SLS framework, we conduct two falsification exercises. First, in Appendix Table A.3, we show results from a placebo experiment using false start dates for the antitoxin distribution in Massachusetts. The idea is to consider what would have happened if the free distribution of antitoxin after 1895 would have instead occurred at the beginning of the 1880s. If municipalities with a substantial uptake in

[25]Since we later demonstrate that antitoxin reduced mortality and increased life expectancy, the positive coefficient on pre-antitoxin doctors indicates that the supply of doctors contributed positively to the health transition by facilitating the diffusion of antitoxin.

antitoxin after 1895 were already on a different path of the health transition in the 1880s, our placebo exercise would detect significant effects. Reassuringly, and consistent with the descriptive evidence, this is not the case. In all specifications with incorrect start dates (assuming the antitoxin distribution begins in 1880, ..., 1885), and restricting the sample to include only the pre-antitoxin period, we obtain small and insignificant 2SLS estimates for the diphtheria mortality rate and life expectancy at birth.[26] Second, consistent with the descriptive evidence reported in Figure 3, Appendix Figure A.8 reports reduced-form event study estimates for life expectancy and diphtheria (Panel A and B), which also suggests that our instrument is not picking pre-existing trends in the outcomes. Overall, it seems plausible that post-antitoxin improvements in our health outcomes are, in fact, related to the free distribution of the antitoxin treatment.

Table 2 presents our main results on how the adoption of antitoxin influenced population health. Our empirical investigation of this relationship begins by estimating (1) with least squares (Panel A), using annual observations from 1880 to 1914. Municipalities are weighted by population size in 1895. The two main outcomes are the diphtheria mortality rate (column 1) and life expectancy at birth (columns 2-4). We see that the point estimates, β_{OLS}, for diphtheria mortality and life expectancy are statistically insignificant and close to zero. Overall, when estimating the relationship simply by least squares, we cannot reject the hypothesis that the adoption of the antitoxin treatment did not improve population health.

A different picture emerges when using 2SLS as the method of estimation (Panel B). For both outcomes, the point estimate of β_{2SLS} has the expected sign and is statistically significant at the 5-percent level with a strong first-stage relationship (the Kleibergen-Paap F-statistic is well over 10). Relative to pre-antitoxin average life expectancy, the 2SLS estimate, reported in column 2, indicates that one more bottle of antitoxin per 1,000 people increases life expectancy by 1.2 percent. The corresponding reduced-form estimates are reported in Panel C of Table 2. Moreover, we present results based on a more classical shift-share approach in which the aggregate number of bottles are distributed according to pre-antitoxin diphtheria mortality shares. The estimates using this alternative approach are reported in Appendix Table A.4 and are similar to the 2SLS estimates presented in Table 2.

Based on the statistical evidence from contemporaneous publications (see Section 2.3), the antitoxin treatment could have gender-specific effects. Because gender is stated on the death certificate, we can calculate life expectancy separately for men and women. Columns 3 and 4 of Table 2 reveal that there are no differential effects of the antitoxin treatment on

[26]The sample period includes the years 1880 to 1896. Similar results are obtained if the sample period is restricted to the pre-antitoxin period (up to 1894), but the strength of the first stage is increased when including more years for the diffusion of the antitoxin.

life expectancy at birth by gender.[27]

In Figure 4, we use the reduced-form estimate of Table 2 to conduct counterfactual experiments with annual delays in the free distribution of antitoxin, which allows us to interpret the magnitude of our estimates. For example, in 1895, we assume a delay of one year, while at the end of our sample window, the delay is assumed to be 20 years (i.e., no distribution of antitoxin within our sample period). The counterfactual (in a given year) is the observed outcome minus the predicted change. In terms of life expectancy (Panel A), we observe a 1.4 year difference at the end of the sample for average treatment intensity. In comparison, from 1895 to 1914, life expectancy in Massachusetts increased by almost 5 years, implying that the availability of antitoxin can potentially explain up to 27 percent of the observed increase in life expectancy over this period. Panel B demonstrates that antitoxin can explain almost half of the decline in the observed diphtheria mortality rate. The no-antitoxin scenario is the most extreme counterfactual, and if we instead assume a 5-10-year delay, these numbers become somewhat smaller. However, this still indicates that antitoxin played an important role in the historical mortality transition in Massachusetts at the beginning of the 20th century.[28]

5.4 Robustness

Next, we present a number of robustness checks based on the baseline annual linear trend-break model as outlined in equation (2). We use these findings to think about the sensitivity of our baseline counterfactual calculations. The first exercises are outlined in Appendix Table A.5. First, we show in columns 1 and 6 that our results remain robust when controlling for various municipality characteristics associated with urbanity and the mortality environment. The additional control variables included are those robustly correlated with the adoption of the antitoxin treatment from Table 1: mortality rates from other infectious respiratory and waterborne diseases, doctors per 1,000 people, distance to Boston, population density and people per dwelling (all measured prior to antitoxin availability and interacted with a full set of year fixed effects). Second, we add time-varying controls for the population shares of children (ages 0, 1 to 4, 5 to 9, 10 to 14). This may be important because diphtheria was mostly a childhood disease, although, by definition, life expectancy takes into account

[27]The data availability of death certificates used to calculate life expectancy in columns 2-4 reduces the sample of municipalities by 14.

[28]We get a similar counterfactual quantification combining the first stage and 2SLS estimates and use these annual delays in terms of the supply of antitoxin bottles. However, it is more straightforward to use the reduced-form estimates when expressing the statistical uncertainty around the different counterfactual scenarios, as we do in Figure 4.

variations in age structures. As seen in Columns 2 and 7, this does not change our baseline conclusion. Third, we have obtained adoption dates for the introduction of public water works and the roll-out of hospitals from the SBH annual reports and control for these in columns 3 and 8. Adding those potential confounders does not affect our findings (point estimates change very little). Fourth, we obtain similar insights using alternative functional forms: in columns 4 and 9, the 2SLS estimates are based on a level-log model, while in column 10 we use a log-log model. The latter functional form is more suitable for life expectancy at birth as the outcome, which has only a few zeros. This is not the case for the diphtheria mortality rate, which in some years could be zero in smaller places. Hence, we report a reduced-form Poisson estimate in column 5. This model takes into account that the outcome variable is highly skewed. For all these modified specifications, we reach the same conclusion, that the free supply of antitoxin improved population health.

Appendix Table A.6 presents further robustness checks. Columns 1 and 5 report estimates similar to the baseline when using treatment intensity logged, which implies that fewer municipalities are included in the sample (i.e., municipalities with zero pre-diphtheria mortality rates). Unweighted 2SLS estimates are reported in columns 2 and 6. In columns 3 and 7, we scale the number of antitoxin bottles by the number of children aged 0 to 10 (the most affected group) instead of the entire population and, in columns 4 and 8, Boston is excluded from the sample. Again, we see that our baseline conclusion is robust to these modifications.[29] Finally, we address the issue that life expectancy, in many instances, is derived from municipalities with relatively few deaths during a particular year and age. In column 9, the outcome is life expectancy at birth, but the denominators in the life tables are based on five-year population age groups from the State census (Haines 2022) instead of single-year age groups from the Federal census. In column 10, we calculate life expectancy at birth for five-calendar years instead of single calendar years. In both specifications, we obtain point estimates relatively similar to our baseline specification.[30]

In Figure A.9, we summarize the robustness exercise in terms of our counterfactual calculations. To this, we add a specification, where the trend is concave in years since 1894 (i.e., logged year) instead of linear as in the baseline. While this reduced-form estimate

[29]Appendix Table A.7 demonstrates that our findings are robust to the inclusion of lagged outcomes to capture persistence in mortality and life expectancy and also, potentially, mean-reverting dynamics (it should be noted that in this specification, the inclusion of lagged dependent variables produces biased and inconsistent estimates). The results are also robust if we instead include the initial (1880) outcome variable interacted by year fixed effects (not reported).

[30]Moreover, in Appendix Figure A.4, we collapse municipalities into two regions according to their treatment intensity (below and above median) and calculate life expectancy for each region, which should also avoid the problem of small areas in life tables. This figure also provides reduced-form evidence of antitoxin increasing life expectancy.

is statistically significant at the one percent level, this turns out to be the most conservative counterfactual, since here we multiply the reduced-form estimate with a log-linear trend instead of a linear trend (thus, we obtain the same 2SLS estimates in the two types of specifications). The main takeaway from our robustness analysis, as visualized in Figure A.9, is that antitoxin can explain at the very least 6 percent of the increase in life expectancy at the end of our sample window (see blue dashed line in Panel A).

5.5 Other mortality and health-care sector outcomes

Since diphtheria mainly affected children, Table 3 presents the impact of the antitoxin treatment on infant and child mortality rates (total and by gender). These variables are often used in the literature as important markers of (child) population health (e.g., Alsan and Goldin 2019). Moreover, this exercise also serves as an additional robustness check for the data used in the denominator. For diphtheria mortality (and other diseases, reported below), we use population counts from the State census, which are reported in the vital statistics or available in (Haines 2022). For the life tables, we use population counts by single-year age groups from the Federal census. Since deaths during the first year of life often occur within the first couple of months, it is common to use the number of live births to represent the population at risk when constructing the infant mortality *rate*. We follow this approach using the birth counts from individual birth records. The child mortality rate spans the ages 1 to 4, and we use the individual death and birth records to construct the number of people in that age group (see Appendix Section A.1 for details).

For the infant mortality rate (columns 1 to 3), the average antitoxin effect is negative, but far from being significant and for boys the point estimate is even positive but statistically insignificant.[31] For the child mortality rate (columns 4 to 6), the point estimate is negative, highly statistically significant, and quantitatively similar for both genders. The percent declines are the largest for the child mortality rate (relative to the pre-antitoxin mean). In particular, the estimate in column 4 suggests that a one-bottle increase in antitoxin per 1,000 people reduces the child mortality rate by about 3 percent (measured relative to the pre-antitoxin mean). In Appendix Figure A.10, we use the reduced-form estimate of column 4 to construct a counterfactual for the child mortality rate similar to Figure 4. We observe that antitoxin can explain up to 17 percent of the decline in the child mortality rate at the end of the sample period.

In Appendix Table A.8, we also consider age-specific mortality rates (ages 0 to 10) directly from the life tables as outcomes. Across all ages, the point estimate on antitoxin per 1,000

[31]Note, the results and relatively large standard errors are not driven by outliers.

people is negative and, in eight out of 11 cases, statistically significant at conventional levels. Evaluated relative to the relevant pre-antitoxin mean mortality rate, the negative effects are most pronounced at ages 1 to 4, which is consistent with the historic age profile for diphtheria mortality (e.g., Crum 1917) and the evidence reported in Table 3. For example, the estimated coefficient at age 4 implies that a one-bottle increase in antitoxin leads to a 4 percent decrease in this age-specific mortality rate.[32]

Next, we assess whether the availability of the antitoxin treatment reduces the mortality burden from diphtheria. We use the diphtheria mortality ratio (i.e., the number of diphtheria deaths per 1,000 deaths) to address this question. Column 1 of Table 4 reports the result. The point estimate is negative and statistically significant at the 1-percent level indicating that the effect of the diphtheria antitoxin was first-order and reduced the mortality burden of diphtheria. In the remainder of Table 4, we investigate whether the antitoxin treatment reduced the fatality rate of diphtheria, had second-order effects on other causes of death, and changed fertility behavior. Column 2 provides suggestive evidence that the antitoxin treatment reduced fatality (diphtheria death per 1,000 cases of diphtheria), however, the estimate is not statistically significant at any conventional levels. In columns 3 to 5, we consider other diseases as outcomes (deaths from strokes, other infectious diseases, and accidents). In all specifications, we control for the pre-antitoxin mortality rates of the outcome interacted with year fixed effects. The antitoxin effect on strokes reported in column 3 is negative and statistically significant at the 5-percent level, while the effect on other infectious diseases (i.e., bronchitis, tuberculosis, scarlet fever, pneumonia, whooping cough, measles, typhoid, and digestive illnesses) reported in column 4 is small in magnitude (relative to the pre-antitoxin mean), but significant at the 10-percent level. We find no effects on deaths from accidents, which is to be expected (column 5).

In general, second-order mortality effects are not that surprising given the health complications of diphtheria and secondary infections. This also means that assessing the effect of antitoxin on life expectancy using data on the mortality burden of diphtheria in back-of-the-envelope calculation would likely underestimate the true effect, since these simple calculations misses possible spillover-effects to other diseases. There is also some evidence that the antitoxin treatment reduced the crude birth rate (column 6), but the effect size is small in magnitude and statistically insignificant.

[32]The outcomes in Appendix Table A.8 are so-called q-type mortality rates, which are being used in the calculation of the life tables. The relationship between the m-type mortality rate and the q-type is given by: $q_x = m_x/(1+(1-a_x)m_x)$, where m_x is the (m-type) age-specific mortality rate and a_x is the expected number of months an individual at a given age lives within the calendar year. For age 0, we use $a_0 = 1/3$; for all other ages reported in Appendix Table A.8, we set a_x equal to one-half, which is also our approach when setting up the life tables.

Appendix Table A.9 provides a complementary analysis to study the effects on individual mortality rates (i.e., "diseases"). We "stacked" the cause-of-death mortality rates (columns 1-5) and cases rates (column 6), so the panel dimension becomes municipality-year-disease. In this specification, we can control for municipality-by-year, disease-by-year-by-county, and municipality-by-disease fixed effects. Accounting for these additional set of fixed effects further mitigates the concern that our estimates might be driven by omitted variables. We can add this additional set of controls since we interact the main right-hand-side variables in equations (1) and (2) with an indicator for diphtheria (since our working hypothesis is that the antitoxin treatment should have a first-order effect on this disease). While the baseline model can be interpreted along the lines of a difference-in-differences estimation, the stacked model is more similar to a triple-differences estimation. In the first column, all 13 causes of death are included as controls, while in columns 2 to 5, we vary the included control causes (e.g., column 3 only includes childhood diseases as controls).[33] In all five specifications, the point estimates are between -0.11 and -0.06, which is close to our baseline estimates. The final column shows the effect of the antitoxin treatment on the number of diphtheria infections relative to the number of infections of scarlet fever, typhoid fever, measles, and smallpox. The estimated coefficient is negative but not statistically significant at conventional levels. Taken together, these results reveal that municipalities with higher rates of antitoxin adoption experienced larger declines in their diphtheria mortality rate relative to other diseases.

One remaining issue is whether the "antitoxin effect" simply captures the expansion of the healthcare sector at that time. Municipalities severely affected by diphtheria could have requested more doctors, such that the increased number of antitoxin bottles per 1,000 people simply reflects affected municipalities having more medical doctors available. To check whether this was the case, we obtain the number of doctors, nurses, and pharmacists per 1,000 people at the municipality level from the full-count U.S. censuses. We then apply the baseline annual 2SLS specification, which means that we had to interpolate the occupation rates between the census years. Table 5 summarizes the results for doctors (Panel A), nurses (Panel B), and pharmacists (Panel C). In all specifications, the effects are quantitatively small and statistically insignificant. Although the healthcare sector contributed to the mortality transition by facilitating the diffusion of antitoxin (see Table 1), our results suggest that the health benefits from the antitoxin treatment did not simply reflect more affected municipalities expanding their healthcare sector.

[33]Including, for example, stroke deaths as a control disease, in column 1, can be problematic, since in Table 4 we demonstrate that these were affected by the antitoxin treatment, which is why the subsequent columns include different sets of control diseases.

6 Effects on School Attendance and Adult Outcomes

Thus far, we have documented that the availability of the antitoxin serum substantially reduced diphtheria mortality rates and increased life expectancy at birth. Our results also reveal that the antitoxin treatment for diphtheria was most effective for children below age 10. In this section, we examine whether exposure to antitoxin treatment affected school attendance in the short term and had implications on the educational attainment and labor market outcomes of exposed children as adults.

We start our analysis by combining a measure of antitoxin exposure during childhood with individual data on school attendance from the U.S. Census in 1900. While other historical US Censuses (1850-1930) only contained a question about school attendance (as little as one day of school during the previous (census) year counted as attending), in 1900, enumerators also asked how many months a person of school age attended school during the census year (June 1, 1899 to May 31, 1900). In Massachusetts, the average length of schooling in each year during the 1890s was nine months (Massachussets State Board of Education 1901, p.101).

Since we do not have information about the antitoxin treatment at the individual level, we cannot distinguish whether the use of the antitoxin directly affected the sickness of treated individuals or whether a more efficient containment of diphtheria reduced the spread of the bacteria more generally. Instead, our estimation approach utilizes the annual variation in antitoxin treatment across municipalities at the time when children were 0-9 years old. This allows us to test whether young children with potential access to the antitoxin treatment were less sick and could therefore attend school for more months during the year. Our sample for the short-term analysis includes all 5 to 15-year-old white children who lived in Massachusetts in 1900.

The econometric model of this subsection is described by the following equation:

$$y_{ibm} = \beta Exposure_{bm} + \mu_b + \mu_m + \Gamma X_{im} + \epsilon_{ibm}, \tag{3}$$

where y_{ibm} is a dummy variable if child i born in year b living in municipality m attends school (i) at all; (ii) for three months or less; and (iii) for at least for nine months. The variable of interest, $Exposure_{bm}$, captures the average exposure to the antitoxin treatment over the first nine years of life. For example, a child born in 1890 living in municipality m in 1900 is assigned the average number of antitoxin bottles per 1,000 people supplied to this municipality over the years 1890-99 (note there was no antitoxin available before 1895).[34]

[34]The assumption is that children received the antitoxin treatment in the municipality in which they were listed in the 1900 Census. If a child was younger than nine years in 1900, we only assign

All specifications include fixed effects for municipality (μ_m) and birth year (μ_b). The set of controls, X_{im}, includes dummies for gender, place of birth, rural, year of immigration, and a set of parental controls including dummies for mother's and father's birthplace, their year of immigration, age of the mother and father, whether the mother and father were literate, whether the father or mother was absent at the time of the census, and whether the father worked in a white-/blue-collar skilled occupation. We cluster standard errors at the municipality level.

Table 6 summarizes the results. For each outcome, we report two specifications. The first specification only controls for municipality and birth year fixed effects, while the second specification also includes the set of individual and parental controls. Exposure to the antitoxin treatment during childhood did not increase school attendance along the extensive margin (columns 1-2), however, given that a child attended, they stayed in school for more months. In particular, we show that more exposed children were less likely to stay in school for three months or less (columns 3-4), and, instead, they attended school for at least nine months in 1900 (columns 5-6). The estimated coefficients are statistically significant at the 1-percent level. There are no noticeable differences by gender and family background (see Appendix Table A.10).[35] We regard this as suggestive evidence that access to the antitoxin treatment reduced sickness in class and thus children could attend school more regularly. The results are also quantitatively sizable: evaluated at the sample mean, antitoxin exposure reduced the likelihood of attending school for three or fewer months by almost 5 percent and increased the likelihood of attending school for at least nine months by almost 1 percent.

Since exposure to the antitoxin treatment increased the time children spent in school, it would be interesting to know if this had any implications for educational attainment and labor market outcomes of the affected children as adults. To answer this question, we use the CT crosswalks for the years 1900 to 1940 to follow 5- to 15-year-old boys and girls living in Massachusetts in 1900 into adulthood (these individuals were aged 45 to 55 in 1940). The CT obtains higher match rates than existing linking methods without substantially increasing false positives (Price et al. 2021), and it is also reassuring that we can replicate the results of Table 6 using the linked sample (see Appendix Table A.11).

Using the linked sample, we can test whether exposure to the antitoxin treatment during childhood had any detectable long-run effects. Table 7 reports the estimates for years of schooling (column 1) and the following labor market outcomes: whether the person worked in a low-skilled occupation (column 2), in a blue-collar skilled occupation (column 3), in a white-

the average exposure up to their current age in 1900. For the long-term analysis, we use the average exposure over the first nine years of life.

[35]Note, results remain qualitatively unchanged if we restrict the sample to include only children aged 5-15 who were born in Massachusetts.

collar occupation (column 4), the occupational income score (column 5), and wages (column 6).[36] The estimating equation is (3) and the estimation method is least squares.[37] Although school absenteeism rates for exposed children decreased in 1900, we find no evidence that these children went for more years to school and had better labor market outcomes as adults. The estimated effect of exposure to antitoxin during childhood on the years of schooling in column (1) is statistically insignificant and quantitatively close to zero. A similar picture emerges when considering the adult labor market outcomes of exposed children in columns (2)-(6). The estimated coefficient on antitoxin exposure is always statistically insignificant and very small in size (precise null effects). This is also the case when looking at individuals at the ages of 25-35 (linked sample 1900 to 1920) or at the ages of 35-45 (linked sample 1900-1930). The results are reported in Appendix Table A.12. We also obtain similar results when using the crosswalks of linked men from the Census Linking Project (CLP).[38]

Our near-zero (and statistically insignificant) long-term results could imply, that conditional on surviving into adulthood, the antitoxin treatment enabled children who grew up in municipalities with high rates of diphtheria to end up in similar occupations as adults as children from municipalities with lower rates of diphtheria. Alternatively, the effect of reduced school absenteeism might not be large enough to generate significant increases in the years of schooling and better labor market outcomes of the exposed boys as adults. Overall, our results suggest that while exposure to the antitoxin treatment during childhood likely reduced absenteeism from school, the long-term effects on educational attainment and labor market outcomes a few decades later appear negligible.

7 Concluding remarks

This paper contributes to the debate on whether medical advances played an important role in the early phase of the health transition in the United States. We examined the health effects of the diphtheria antitoxin, which was developed to combat diphtheria—a leading cause of death in children in the early 20th century. Our focus was on Massachusetts, whose historical vital statistics are reliable and well-documented. In 1895, the Massachusetts State

[36]We use IPUMS variable OCC1950 to define white-collar jobs (codes 0-490, excluding farmers and farm managers), blue-collar skilled occupations (codes 500-595), and low-skilled occupations (codes 600-970).

[37]We obtain similar results when replacing the actual antitoxin exposure with the predicted distribution of antitoxin based on our 2SLS approach in equation (3).

[38]The CLP crosswalks contain only men over time and produce a lower match rate than the Census Tree Project. See https://censuslinkingproject.org/ and Abramitzky et al. (2021) for more details on the linking method and the corresponding match rates of the CLP links.

Board of Health began providing municipalities with diphtheria antitoxin free of charge for medical use. Using over 1.5 million death certificates and municipality data on the adoption of the antitoxin treatment for the years 1880 to 1914, we find that the provision of antitoxin serum substantially reduced diphtheria mortality rates and increased life expectancy at birth. Our baseline estimate indicates that antitoxin can explain 27 percent of the increase in life expectancy at birth and 17 percent of the decrease in child mortality. Thus, the observed increase in life expectancy was primarily driven by the decrease in child mortality. Since infant and child mortality accounted for the largest proportion of the total mortality burden at that time, the diffusion of the antitoxin treatment—by preventing mortality due to a major childhood disease—led to noticeable improvements in health before "the era of big medicine". This suggests that medical innovations may have played a much larger role in increasing life expectancy in the early 20th century than previously thought.

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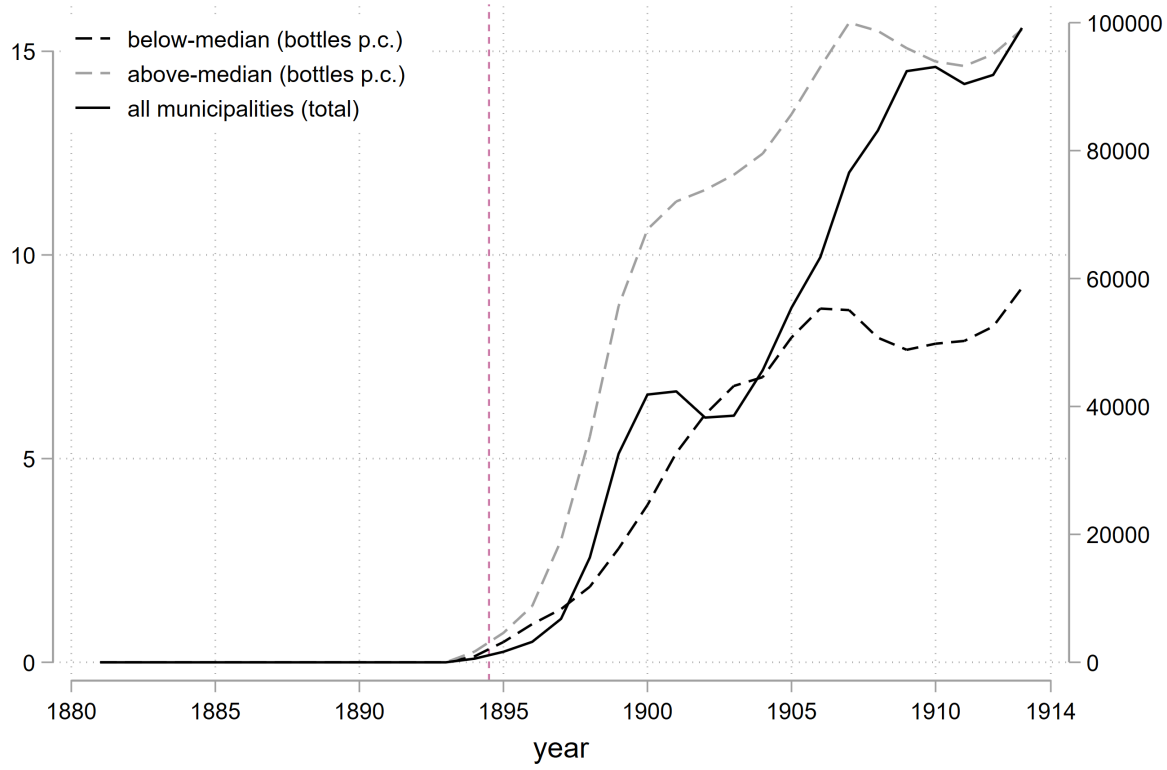
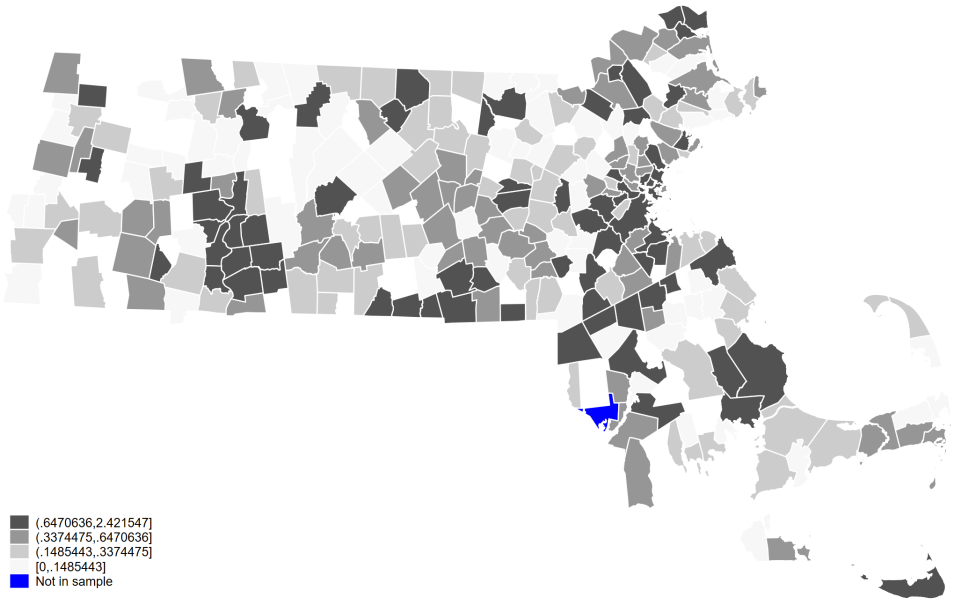
Figure 1: Antitoxin adoption rates



Notes: *The figure shows the development of the average number of antitoxin bottles per 1,000 people by treatment intensity (1. y-axis) and the total amount of bottles to all municipalities (2. y-axis). We report three-year moving averages and use the 1895 municipality population size as weights. Treatment intensity is measured as the average pre-antitoxin (1891-94) diphtheria mortality rate in logarithmic units. The vertical line indicates the first year when antitoxin became freely available for adoption.*

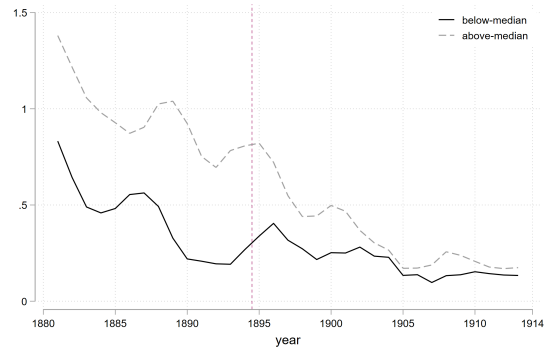
Figure 2: Spatial variation in pre-antitoxin (1889-1894) diphtheria mortality rates



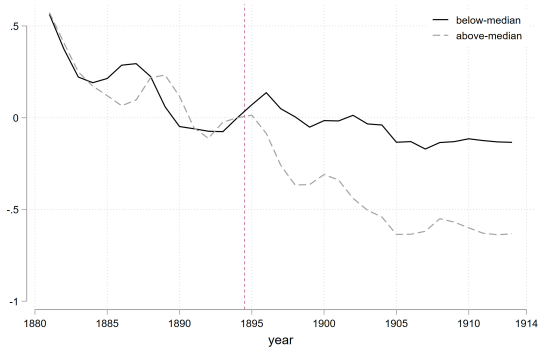
Notes: *This figure shows the spatial variation in diphtheria mortality rates (i.e., treatment intensity) as defined in Equation (2).*

Figure 3: Diphtheria mortality rates and life expectancy at birth

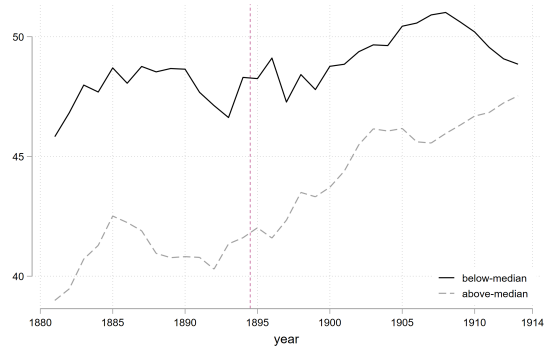
(a) Diphtheria, average by group



(b) and deviation from 1894 values



(c) Life expectancy, average by group

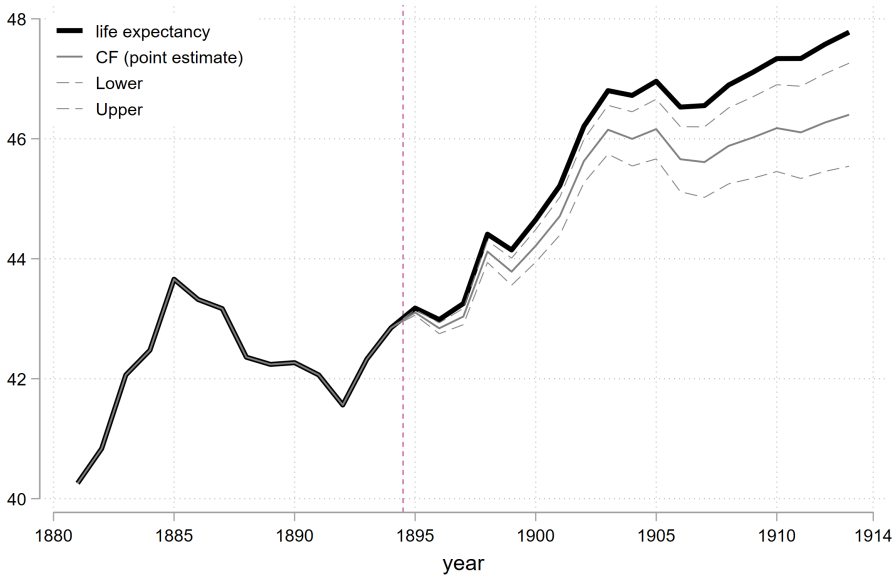


(d) and deviation from 1894 values

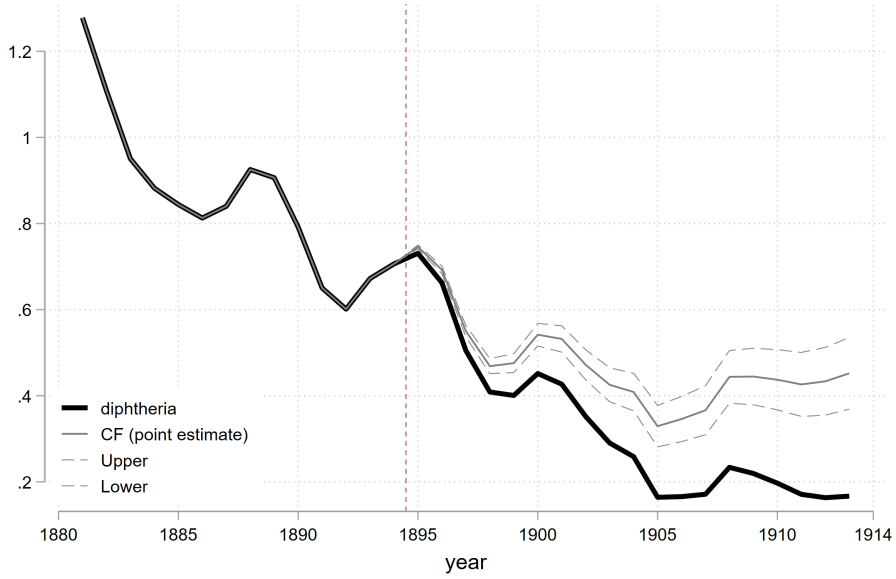
Notes: *The figure shows the development of the average diphtheria mortality rate per 1,000 people (Panel A and B) and life expectancy at birth (Panel C and D) by treatment intensity (below/above median). Panel A/C shows this development by group and Panel B/D in addition subtracts the 1894 value for each group. We report three-year moving averages and use the 1895 municipality population size as weight. Treatment intensity is measured as the average pre-antitoxin (1889-94) diphtheria mortality rate. The vertical line indicates the first year when antitoxin became freely available for adoption.*

Figure 4: Counterfactual scenarios

(a) Life expectancy at birth



(b) Diphtheria mortality rate



Notes: *This figure uses the baseline reduced-form estimates (and 95 percent confidence bands) to calculate the counterfactual (CF) development for the life expectancy at birth (Panel A) and the diphtheria mortality rate (Panel B). The CF calculations are based on annual delays for average treatment intensity. These are the gray solid curves (and dashed ones indicate the 95-percent confidence bands. The solid black curves are the observed population weighted averages of the outcomes. All curves are three-year moving averages.*

Table 1: Antitoxin adoption by municipality characteristics

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
treatment x I x (t-1894)	0.320***	0.240***	0.236***	0.211***	0.151***	0.138***	0.142***	0.143***
	(0.054)	(0.055)	(0.056)	(0.055)	(0.051)	(0.049)	(0.049)	(0.050)
ln(infec. rate, 89-94) x I x (t-1894)		0.248***	0.251***	0.255***	0.354***	0.166**	0.055	0.057
		(0.075)	(0.076)	(0.072)	(0.072)	(0.072)	(0.070)	(0.086)
ln(stroke rate, 89-94) x I x (t-1894)			-0.003	-0.042	-0.043	-0.067*	0.018	0.018
			(0.041)	(0.044)	(0.040)	(0.037)	(0.042)	(0.043)
doctors pr. capita in 95 x I x (t-1894)				0.091***	0.074**	0.123***	0.136***	0.136***
				(0.033)	(0.029)	(0.034)	(0.037)	(0.037)
dist Boston x I x (t-1894)					-0.009***	-0.006***	-0.006***	-0.006***
					(0.002)	(0.002)	(0.002)	(0.002)
persons pr 1,000 sqm in 95 x I x (t-1894)						0.038***	0.028***	0.028***
						(0.009)	(0.009)	(0.009)
persons pr. dwelling in 95 x I x (t-1894)							0.072*	0.072*
							(0.038)	(0.040)
persons pr. room in 95 x I x (t-1894)							-0.131	-0.125
							(0.512)	(0.484)
fb share in 95 x I x (t-1894)								-0.013
								(0.386)
$N \times$ T	9,625	9,625	9,380	9,360	9,360	9,360	9,360	9,360

Notes: *This table reports how the number of antitoxin bottles per 1,000 people is related to baseline municipality characteristics. The variable of interest, "treatment", is the diphtheria mortality rate averaged over the pre-antitoxin years 1891 to 1894; "doctors pr. capita" is the number of doctors pr. 1,000 people in 1895; "dist Boston" is the aerial distance to Boston; "pop density in 95" is measured as the number of people per 1,000 square miles in 1895; "persons pr. dwelling in 95" is the number of people per dwelling in 1895; "persons pr. room in 95" is the average number of persons per in 1895; "fb share" is the share of foreign-born individuals in 1895. These municipality characteristics are interacted with a post-1895 dummy and a linear trend ($I \times (t - 1894)$). All regressions control for municipality and county-by-year fixed effects and are weighted by population size in 1895. The sample period is annually from 1880 to 1914. Standard errors (in parentheses) account for arbitrary heteroskedasticity and are clustered at the municipality level. ***, **, and * indicate significance at the 1, 5, and 10 percent level.*

Table 2: The effect of antitoxin on population health

	(1) diphteria all	(2) life exp all	(3) life exp female	(4) life exp male
Panel A: OLS estimates				
antitoxin p.c.	-0.007	-0.006	0.006	0.036
	(0.007)	(0.029)	(0.031)	(0.030)
Panel B: 2SLS estimates				
antitoxin p.c.	-0.107***	0.506***	0.698***	0.675***
	(0.023)	(0.170)	(0.197)	(0.196)
Panel C: Reduced-form estimates				
treat x I x yr	-0.024***	0.094***	0.101***	0.124***
	(0.004)	(0.031)	(0.031)	(0.034)
Mean pre-y	0.904	41.93	43.21	40.66
$N \times T$	9625	9135	9135	9135
N	275	261	261	261
F-Stat	35.67	33.83	33.83	33.83

Notes: *This table reports OLS estimates (Panel A), 2SLS estimates (Panel B), and reduced-form estimates (Panel C) of the relationship between the adoption of antitoxin per 1,000 people and the diphtheria mortality rate (column 1) and life expectancy at birth (columns 2-4). The sample includes annual observations at the municipality level from 1880 to 1914. All regressions are weighted with the municipality population size in 1895 and control for municipality and county-by-year fixed effects. "Mean pre-y" is the mean of the outcome measured over the relevant pre-antitoxin period. Standard errors (in parentheses) account for arbitrary heteroskedasticity and are clustered at the municipality level. ***, **, and * indicate significance at the 1, 5, and 10 percent level.*

Table 3: Effects on the infant and child mortality rates

	(1) infant rate	(2) infant rate female	(3) infant rate male	(4) child rate	(5) child rate female	(6) child rate male
antitoxin p.c.	-0.480	-1.221	0.237	-0.632***	-0.636***	-0.644***
	(1.074)	(1.057)	(1.163)	(0.206)	(0.216)	(0.220)
Mean pre-y	152.1	139.1	163.3	23.35	22.69	24.01
$N \times T$	9169	9169	9169	9169	9169	9169
N	262	262	262	262	262	262
F-Stat	33.96	33.96	33.96	33.96	33.96	33.96

Notes: *This table reports effects on infant and child mortality rates using the linear trend-break model as outlined in Equation (2). The infant mortality rate uses the number of live birth in the denominator to measure the population at risk, while it is the population aged 1 to 4 for the child mortality rate. The rates are expressed per 1,000 births or per 1,000 children aged 1 to 4. All regressions are weighted by the municipality population size in 1895 and control for municipality and county-by-year fixed effects. Standard errors (in parentheses) account for arbitrary heteroskedasticity and are clustered at the municipality level. ***, **, and * indicate significance at the 1, 5, and 10 percent level.*

Table 4: Other vital outcomes

	(1) diph ratio	(2) fatality	(3) stroke	(4) infec rate	(5) accidents	(6) birth rate
antitoxin p.c.	-3.546***	-2.442	-0.028*	-0.104*	0.011	-0.496
	(0.915)	(17.230)	(0.015)	(0.062)	(0.013)	(0.681)
Mean pre-y	42.90	449.5	0.545	7.756	0.738	27.09
$N \times T$	9616	2030	9625	9625	9625	9625
N	275	111	275	275	275	275
F-Stat	46.03	23.53	45.72	38.96	43.91	19.57

Notes: *This table reports effects on the diphtheria death ratio (column 1), the fatality rate (column 2), other causes of deaths (columns 3-5), and the crude birth rate (column 6) using the baseline annual linear trend-break model as outlined in equation (2). The sample includes the years 1880 to 1914. All variables are expressed per 1,000 people. All regressions are weighted by the municipality population size in 1895 and control for municipality and county by-year fixed effects. Each regression also controls for the pre-antitoxin outcome (in 1894) interacted with year fixed effects in order to capture possible convergence effects. Standard errors (in parentheses) account for arbitrary heteroskedasticity and are clustered at the municipality level. ***, **, and * indicate significance at the 1, 5, and 10 percent level.*

Table 5: Effects on the health-care sector

	(1)	(2)	(3)	(4)	(5)
age groups	all	20-29	30-39	40-49	50-59
Panel A: Doctors					
antitoxin p.c.	-0.029	-0.013	-0.010	-0.011	0.003
	(0.020)	(0.010)	(0.008)	(0.008)	(0.007)
Panel B: Nurses					
antitoxin p.c.	-0.018	-0.003	0.005	-0.009	-0.001
	(0.088)	(0.062)	(0.017)	(0.008)	(0.004)
Panel C: Pharmacists					
antitoxin p.c.	-0.004	-0.004	-0.001	0.002	-0.003
	(0.014)	(0.005)	(0.008)	(0.004)	(0.004)
$N \times T$	9590	9590	9590	9590	9590
N	274	274	274	274	274
F-Stat	35.54	35.54	35.54	35.54	35.54

Notes: *This table reports the impact of the antitoxin treatment on the number of doctors per 1,000 people (Panel A), the number of nurses per 1,000 people (Panel B), and the number of pharmacists per 1,000 people (Panel C). The top row indicates the corresponding age group (e.g., column 2 provides the number of doctors/nurses/pharmacists in the ages 20-29). The method of estimation is 2SLS using the baseline annual linear trend-break model as outlined in Equation (2). The sample includes the years 1880 to 1914. All regressions are weighted by the municipality population size in 1895 and control for municipality and county-by-year fixed effects. Standard errors (in parentheses) account for arbitrary heteroskedasticity and are clustered at the municipality level. ***, **, and * indicate significance at the 1, 5, and 10 percent level.*

Table 6: Antitoxin Treatment and School Attendance in 1900

	(1)	(2)	(3)	(4)	(5)	(6)
	==1 if attends at all		== 1 if attends ≦ 3 months		== 1 if attends ≧ 9 months	
Exposure	0.002	0.002	-0.005***	-0.005***	0.006***	0.006***
	(0.003)	(0.003)	(0.001)	(0.001)	(0.001)	(0.001)
Observations	512,670	423,268	354,712	296,002	354,712	296,002
R-squared	0.143	0.165	0.029	0.039	0.099	0.111
Municipality FE	YES	YES	YES	YES	YES	YES
Year of Birth FE	YES	YES	YES	YES	YES	YES
Ind. Controls	NO	YES	NO	YES	NO	YES
Mean pre-y	0.652	0.659	0.107	0.107	0.680	0.680

Notes: *This table reports how antitoxin exposure affected school attendance. The sample includes 5 to 15-year-old children in Massachusetts in 1900. The dependent variable is a dummy of whether a child attended school at all (columns 1-2); for no more than three months (columns 3-4); and for at least nine months (columns 5-6). The variable of interest, "Exposure", denotes the average number of antitoxin bottles per 1,000 people that a child during the first nine years was exposed to (see page 24 for details). All regressions control for municipality and year of birth fixed effects. Columns 2, 4, and 6 also include the following set of controls: dummies for gender, place of birth, rural, year of immigration, and a set of parental controls including dummies for mother's and father's birthplace, their year of immigration, age of the mother and father, mother's and father's literacy, whether the father or mother was absent at the time of the census, and whether the father worked in a white-/blue-collar skilled occupation. Standard errors (in parentheses) account for arbitrary heteroskedasticity and are clustered at the municipality level. ***, **, and * indicate significance at the 1, 5, and 10 percent level.*

Table 7: The Long-run Effects of Antitoxin Treatment

	(1) yrs in school	(2) low-skilled	(3) blue-collar skilled	(4) white-collar	(5) ln(occscore)	(6) ln(wages)
Exposure	0.008	0.0001	-0.0007	0.0001	0.0008	0.0018
	(0.005)	(0.0005)	(0.0005)	(0.0005)	(0.0005)	(0.0011)
Observations	232,410	241,359	241,359	241,359	161,679	131,740
R-squared	0.172	0.0873	0.1022	0.0963	0.1013	0.1159
Mean pre-y	10.09	0.225	0.123	0.309	3.276	7.136
Municipality FE	YES	YES	YES		YES	YES
Year of Birth FE	YES	YES	YES	YES	YES	YES
Ind. Controls	YES	YES	YES	YES	YES	YES

Notes: *This table reports how antitoxin exposure affected labor market outcomes of exposed children as adults. The sample includes 5 to 15-year-old children in Massachusetts in 1900 linked to 1940 using the crosswalks from the Census Tree Project. The following outcomes in 1940 are used as dependent variable: educational attainment (column 1), a dummy whether the individual works in a low-skilled (column 2), blue-collar skilled (column 3), or white-collar occupation (column 4), the ln occupational income score (column 5), and ln wages (column 6). The variable of interest, "Exposure", denotes the average number of antitoxin bottles per 1,000 people that a child during the first nine years was exposed to (see page 24 for details). All regressions control for municipality and year of birth fixed effects and the following set of controls: dummies for gender, place of birth, rural, year of immigration, and a set of parental controls including dummies for mother's and father's birthplace, their year of immigration, age of the mother and father, mother's and father's literacy, whether the father or mother was absent at the time of the census, and whether the father worked in a white-/blue-collar skilled occupation. Standard errors (in parentheses) account for arbitrary heteroskedasticity and are clustered at the municipality level. ***, **, and * indicate significance at the 1, 5, and 10 percent level.*

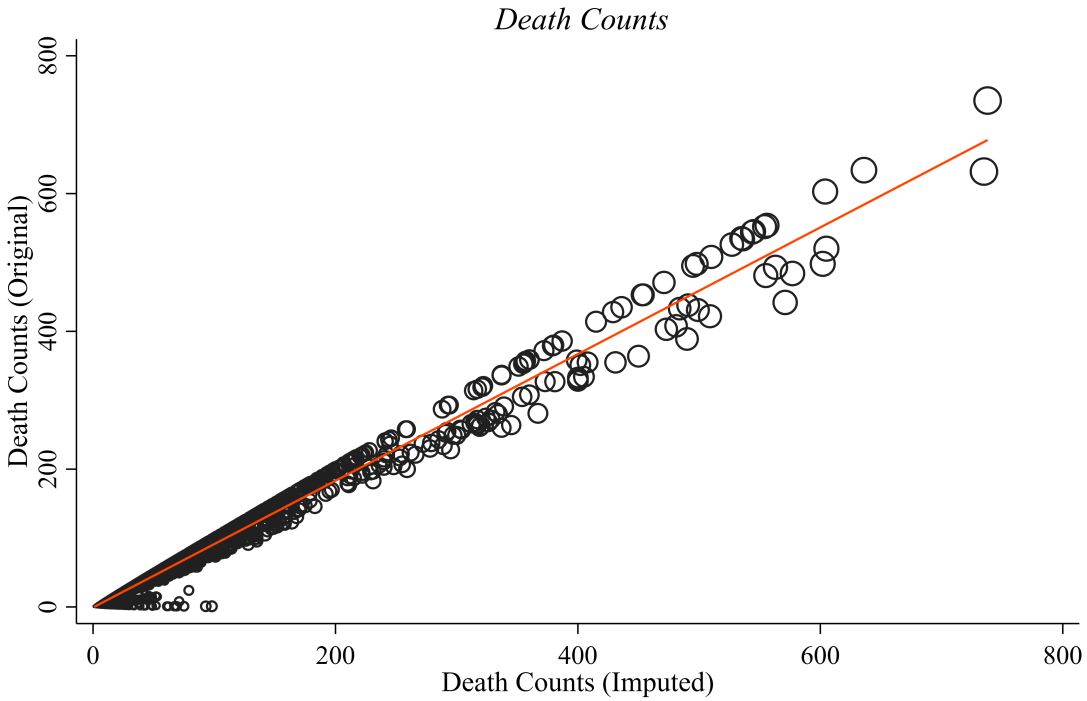
A Online Appendix

A.1 Individual death records and mortality rates by age

We aggregate individual death records at the annual municipality level for a given age to obtain age-specific death counts. We use them to compute the infant mortality rate, the child mortality rate, and life tables, where life expediencies (at various ages) can be extracted, although our regression analysis focuses on life expectancy at birth.

Individual death records have been digitized and are available as part of the data collection Massachusetts Deaths, 1841-1915, through FamilySearch.org. As the death records do not report decedents' ages and fail to digitize accurate ages, we distribute the number of such records across ages 0 to 100, based on the age distribution of other death records with known ages in the same municipality, year, and other demographic traits. More specifically, we separate all death records into groups, which are defined by a municipality, death year, death season (April-September as warm season, and October-March as cold season), the decedent's sex, the decedent's nativity (born in Massachusetts/other US States/foreign country/unknown), and the decedent's marital status (single/ever-married/unknown). Then, we distribute the number of age-missing death records across ages 0 to 100, based on the age distribution of all death records with known ages in the same group. The rich information on death records allows us to group decedents in such a disaggregated way and to make our imputation more accurately by using the age-distribution of similar decedents. We compare the death counts, which include or exclude the redistributed age-missing death records in Figure A.1.

Figure A.1: Imputed and Original Death Counts



Notes: *This figure presents scatterplots of imputed death counts, which include redistributed death cases with missing-age (x-axis) and the death counts excluding redistributed death cases (y-axis). Each scatterplot represents a year-municipality observation between 1895 and 1915. Size of scatterplots represent the imputed death counts, and the fitted line comes from a bivariate regression weighted by the imputed death counts.*

The individual micro data, from the Federal censuses (IPUMS) for the years 1880, 1900, and 1910, allow us to calculate population sizes for single-year age groups by municipality. We linear interpolate between the census years and combine them with the imputed municipal annual death counts by age in order to calculate age-specific mortality rates for the ages 0, 1, .., 100 for each calendar year between 1880 and 1914, which are used in the construction of the life tables.[39] The construction of the life tables is explained in the main text.

In order to assess the sensitivity of the use of population data from the Federal census, we construct other measures of age-specific mortality rates using alternative denominator data sources.[40] In particular, we calculate the infant mortality rate at the annual municipality level by combining the death counts at age 0 and the birth counts aggregated from individual

[39]We extrapolate the population data for the years 1911 to 1914.

[40]In the calculation of the cause-specific mortality rates, we use total population data from the State census, which are available every fifth year, but they are not available by single-year age groups in all the State census years.

birth records, which are also digitized and available through FamilySearch.org. Specifically, the infant mortality rate is defined as follows:

$$IMR_{mt} = \frac{Deaths^{0}_{mt}}{Births_{mt}} \tag{A.1}$$

where $Deaths^{0}_{mt}$ is the death counts for age 0 in municipality m and year t, and $Births_{mt}$ is the birth counts in the same municipality and year. As an additional check of robustness, we also use birth counts from the tabulated vital statistics (same source as cause- of-death data).

We calculate the mortality rate of children aged 1 to 4 at the annual municipality level as follows:

$$CMR_{mt} = \frac{Deaths^{1-4}_{mt}}{Pop^{1-4}_{mt}} \tag{A.2}$$

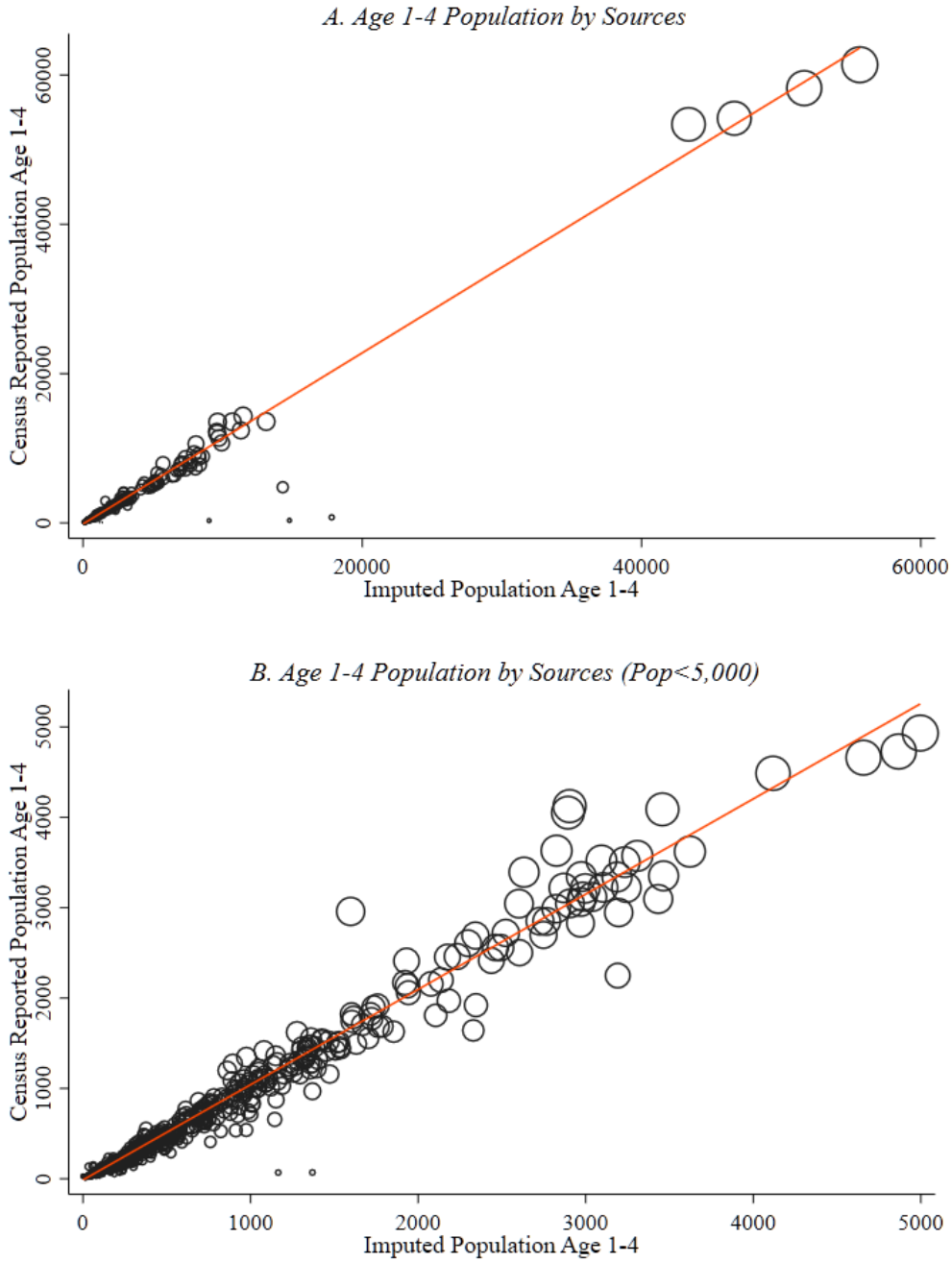
where $Deaths^{1-4}_{mt}$ is the deaths aged 1 to 4 in municipality m and year t, and Pop^{1-4}_{mt} is population aged 1 to 4 in the same municipality and year. Instead of using the Federal interpolated population data, we impute the annual population aged 1 to 4 based on cumulative births and deaths of corresponding cohorts in prior years. This approach is also used by Alsan and Goldin (2019) and Eriksson et al. (2020), Specifically, the population is imputed as follows:

$$Pop^{1-4}_{mt} = \sum_{a=1}^{a=4} \left[Births_{m,t-a} - \textstyle\sum_{k=1}^{k=a} Deaths^{a-k}_{m,t-k} \right] \tag{A.3}$$

where $Births_{m,t-a}$ is is the number of births in municipality m and year $(t-a)$, and $Deaths^{a-k}_{m,t-k}$ is the number of deaths aged $(a-k)$ in municipality m and year $(t-k)$. In fact, the term $Births_{m,t-a}$ is the total number of children born a years ago (standing in year t), and the term $\sum_{k=1}^{k=a} Deaths^{a-k}_{m,t-k}$ is the cumulative deaths for children aged a between their birth year and the year t. We implicitly assume that the migration of children is negligible and all population changes were accounted by deaths.

We compare our imputed population with the census-reported population in years when Federal or State censuses were available. Figure A.2 below shows the imputed population fit well the census-reported population at the municipality level in census years.

Figure A.2: Imputed and Census Reported Population Age 1-4



Notes: *This figures present scatterplots of imputed population aged 1 to 4 (x-axis) and census-reported population aged 1 to 4 (y-axis) in the census years of 1895, 1900, 1905, 1910, and 1915. Census-reported populations come from Federal censuses 1900 and 1910; and Massachusetts State census in 1895, 1905, and 1915. Each scatter-plot represents an observation of municipality and census year. Size of scatterplots represents the population size, and the fitted line is from a regression weighted by imputed population size*

A.2 Relation to Shift-share Instrument

In this Appendix subsection, we show that our baseline instrumental variable, reported in Equation (1), is closely related to an alternative instrument, where the aggregate number of bottles is distributed according to municipality specific diphtheria shares (a "shift-share" type of instrument). For convenience, we repeat the structure of our baseline instrument here:

$$IV_{mt}^{base} = treatment_m \times I_t \times (t - 1894), \tag{A.4}$$

where treatment intensity is defined as the diphtheria mortality rate averaged across the pre-antitoxin years 1889 to 94:

$$treatment_m = \ln \bar{d}_m^{pre}. \tag{A.5}$$

A shift-share type of instrumental variable can be defined defined as:

$$IV_{mt}^{alt} = B_t \frac{D_m^{pre}}{D_{MA}^{pre}} \frac{1}{P_m^{94}}, \tag{A.6}$$

where B_t is the total number of antitoxin bottles supplied to the municipalities in our sample, D_i^{pre} is the total number of diphtheria deaths from 1889 to 1894 in municipality m, D^{MA} is the total number of diphtheria deaths in our sample of municipalities during the same pre-antitoxin years (i.e., D_m^{pre}/D_{MA}^{pre} is the share of diphtheria deaths in municipality m), and P_m^{94} is the municipality population size in 1894. Accordingly, IV_{mt}^{base} is the predicted number of antitoxin bottles per capita, where the aggregate number of bottles supplied by the SBH each year is distributed according to the pre-antitoxin mortality share and the scaling population size is fixed to the pre-antitoxin year of 1894. Alternatively, we could have let the population vary by year, but this assumption would be less conservative (as population size itself is influenced by the use of the antitoxin) and the connection to our baseline instrument would be less obvious. Let us provide a simple example of how the prediction works. If Boston had, say, 20% of all diphtheria deaths prior to the antitoxin treatment, the municipality is allocated 20% of all bottles in each year and then the predicted number of total bottles available to Boston is scaled by its pre-antitoxin population size.

In the following, we show how the two instruments relate to each other. Assume that the baseline treatment takes this slight alternative form:

$$treatment_m = \frac{D_m^{pre}}{P_m^{94}}, \tag{A.7}$$

where instead of taking the average mortality rates over multiple years, we sum all pre-antitoxin diphtheria deaths and scale with the population size of 1894. The interpretation of this ratio remains relatively close to a (mortality) rate, and using the formulation in Equation (A.7) as treatment intensity for the baseline instrument provides very similar results (available upon request). Next, we substitute this into Equation (A.4) and rearrange:

$$\begin{aligned} IV_{mt}^{base} &= \frac{D_m^{pre}}{P_m^{94}} \times I_t \times (t - 1894) \Leftrightarrow \\ D_m^{pre} &= \frac{P_m^{94} IV_{mt}^{base}}{I_t \times (t - 1894)}, \end{aligned} \tag{A.8}$$

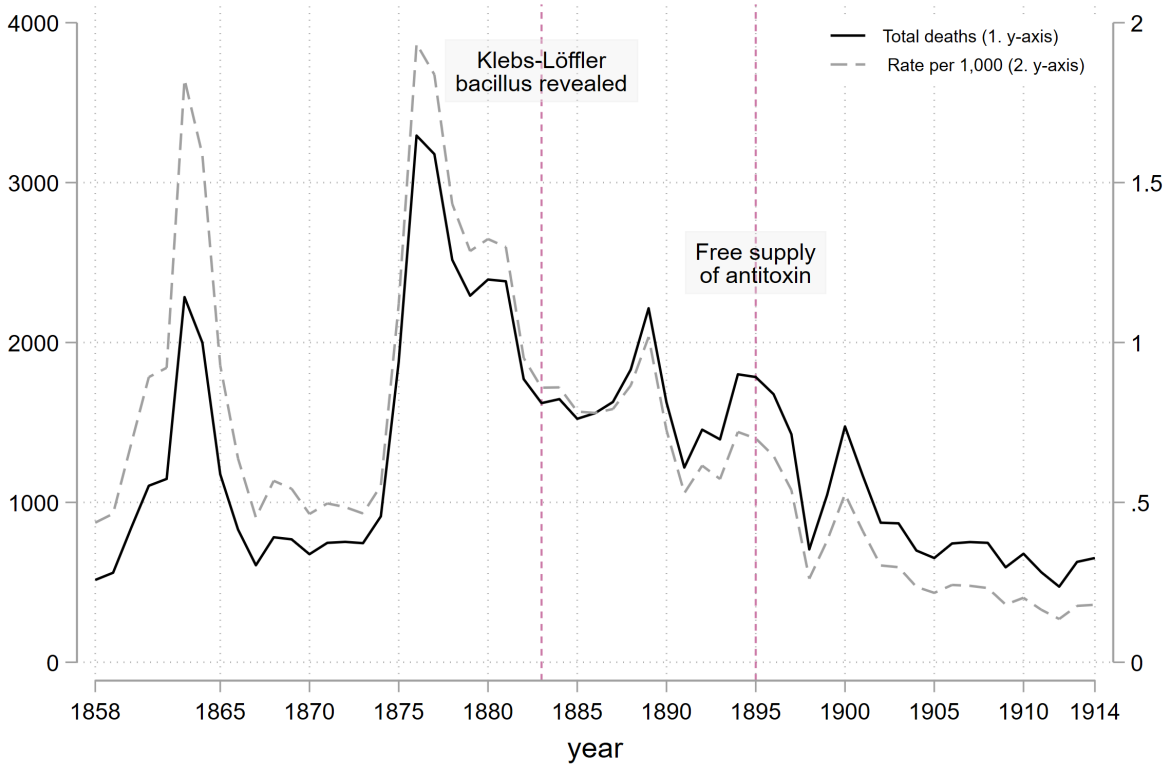
which we combine with Equation (A.7) to give:

$$\begin{aligned} IV_{mt}^{alt} &= B_t \frac{\frac{P_m^{94} IV_{mt}^{base}}{I_t \times (t-1894)}}{D^{MA}} \frac{1}{P_m^{94}} \Leftrightarrow \\ IV_{mt}^{alt} &= \frac{B_t}{D^{MA} \times \tau} IV_{mt}^{base}, \end{aligned} \tag{A.9}$$

where we, in the last line, have omitted the indicator (I_t) for simplicity, since this only reflects the fact that B_t is per definition zero before 1895, and τ is accordingly defined as the linear trend $\tau \equiv (t - 1894)$ for $t > 1894$. From this last expression, we observe that the difference between the instruments is the scaling factor $(B_t / \left(D^{MA}\tau\right))$, which is possibly time-varying, but unrelated to municipality specific conditions. Therefore, whether we use one or the other instrument should not be important in terms of obtaining consistent estimates. The 2SLS estimates for diphtheria and life expectancy using the alternative shift-share type of instrument are reported in Table A.4.

A.3 Additional results

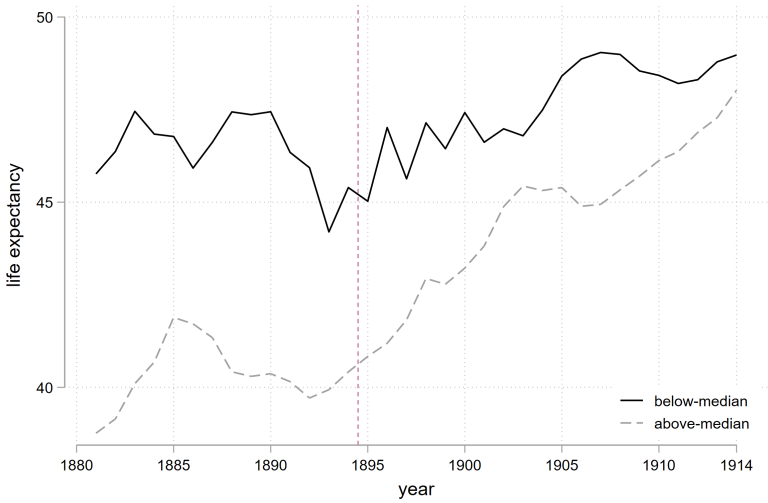
Figure A.3: Total number of diphtheria and croup deaths



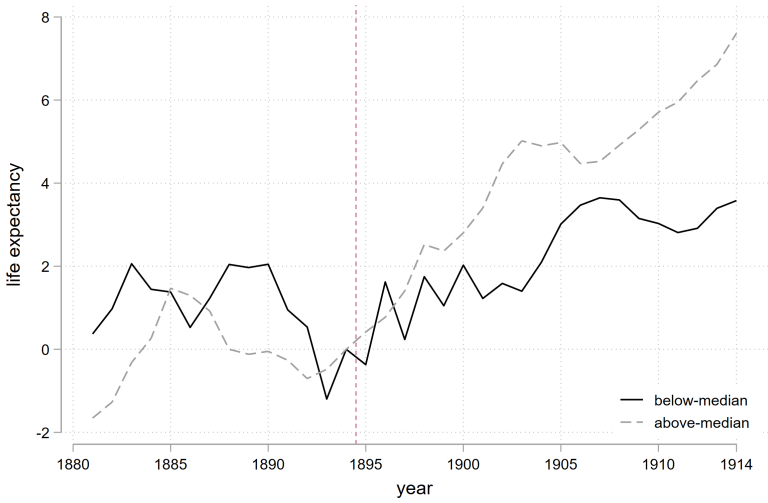
Notes: *The figure shows the development of the total number of deaths due to diphtheria and croup (black solid line) and the rate per 1,000 people (gray dashed line) from 1858 to 1914 for the State of Massachusetts. The data have been obtained from the vital statistics of Massachusetts (various years). 1858 marks the first year where diphtheria was recorded as a separate cause of death in the vital statistics.*

Figure A.4: Development of Life Expectancy at Birth by Treatment Intensity

(a) Average by group

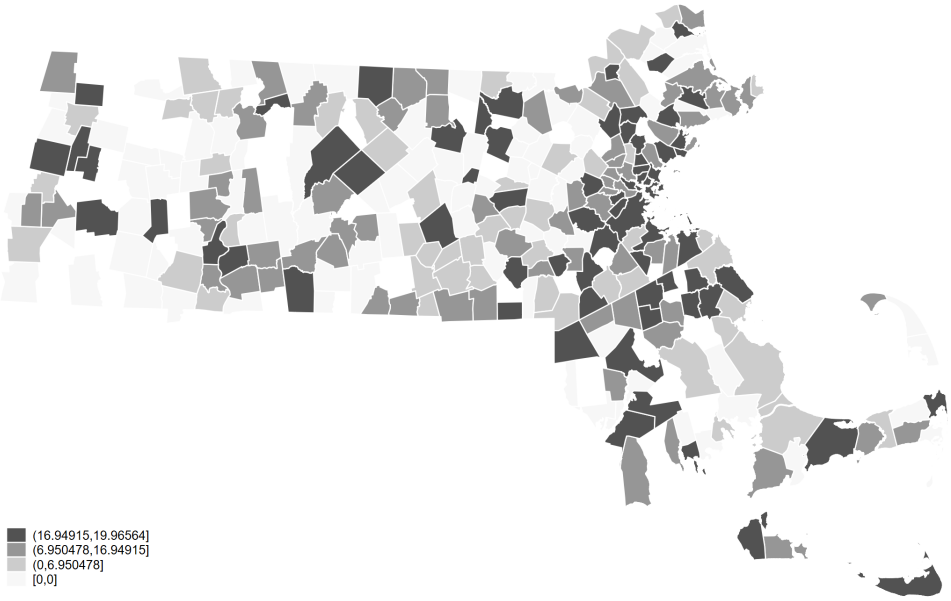


(b) And deviation from 1894 values



Notes: *This figure shows the development of life expectancy for municipalities with above- and below-median treatment intensity. In particular, we have collapsed the baseline sample of municipalities into two regions (according to their treatment intensity) and then for each region (i.e., high-low treatment intensity regions) calculated life expectancy at birth for each year. This avoids the problem of small populations when deriving the life tables and calculating life expectancy. Panel A shows the three-year moving average by group, while Panel B additionally take the deviation from 1894 values for each group.*

Figure A.5: Spatial Variation in Antitoxin Supply in 1914



Notes: *This figures show the spatial variation in the supply of antitoxin bottles per 1,000 people in 1914. Blue colored municipalities are not included in the baseline sample.*

Figure A.6: Spatial Variation in Doctors per 1,000 People in 1895

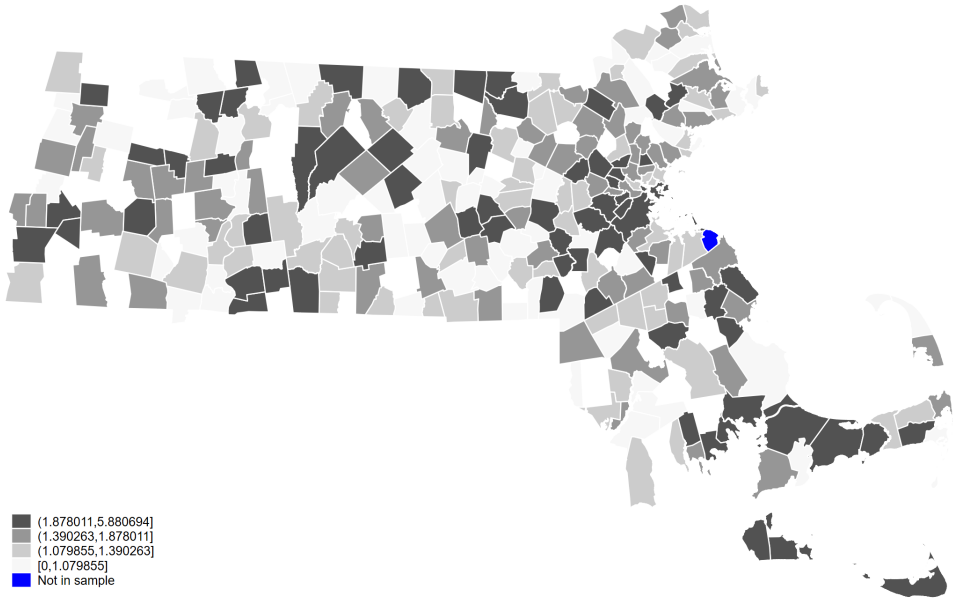
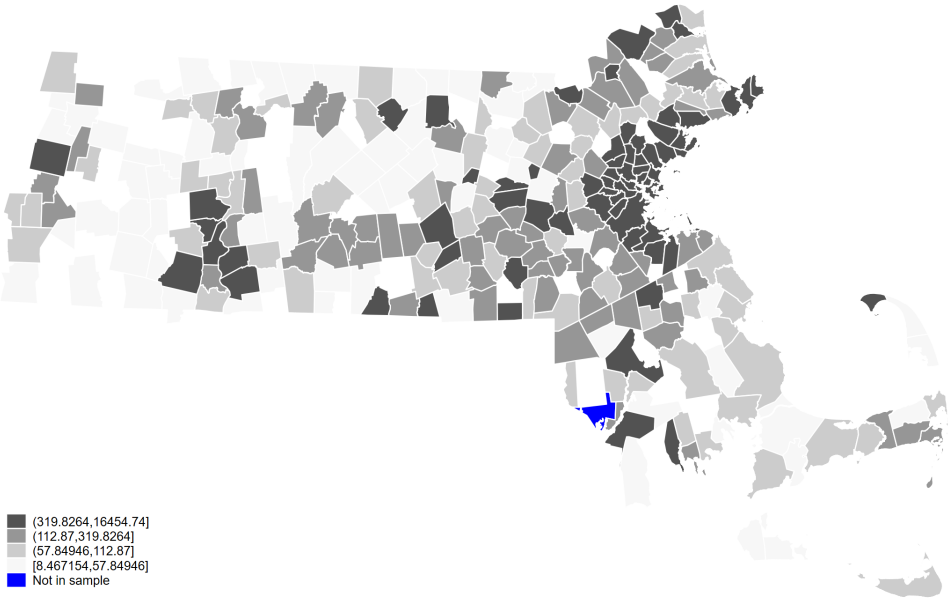


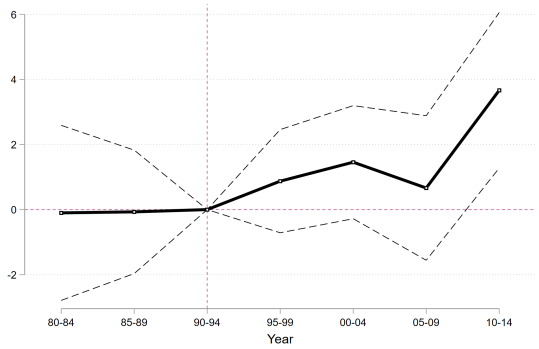
Figure A.7: Spatial Variation in Population Density in 1895



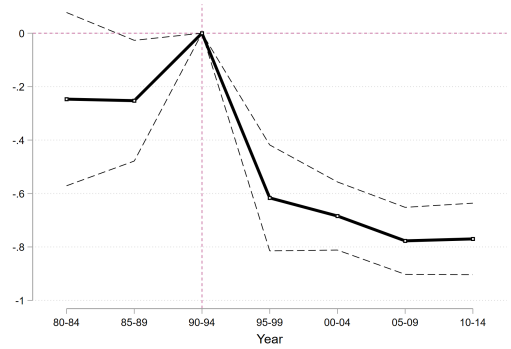
Notes: *These figures show the spatial variation doctors per 1,000 people in 1895 and the number of people per square mile in 1895 (population density), which are two important predictors of the diffusion speed of antitoxin. Blue colored municipalities are not included in the baseline sample.*

Figure A.8: Event study estimates for life expectancy and child mortality

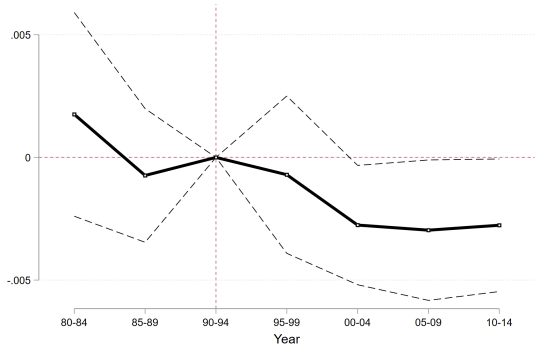
(a) Life expectancy



(b) Diphtheria mortality rate



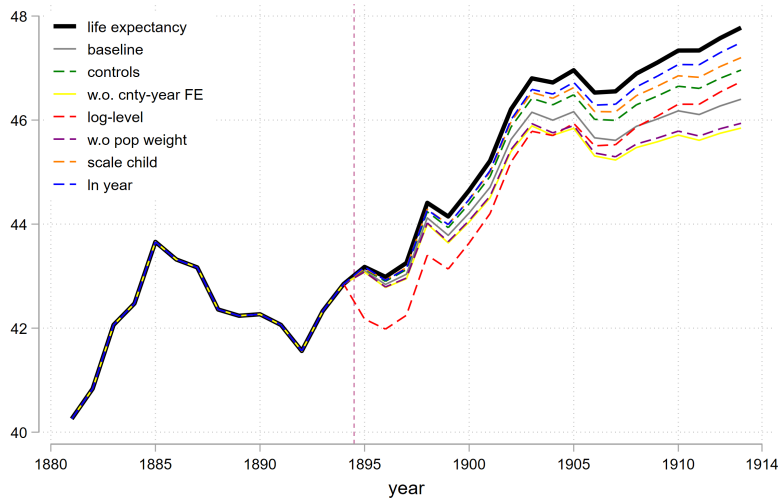
(c) Child mortality rate



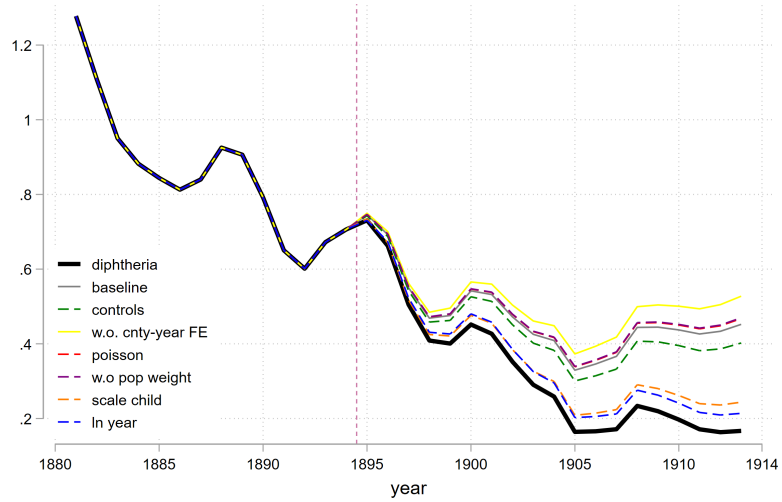
Notes: *This figure reports reduced-form event study estimates, where the outcome (life expectancy in Panel A, Diphtheria mortality rate in Panel B, and the child mortality rate in Panel C) has been regressed on 5-year binned event-dummies interacted with treatment, while controlling for municipality and county-year fixed effects.*

Figure A.9: Robustness of counterfactual scenarios

(a) Life expectancy at birth

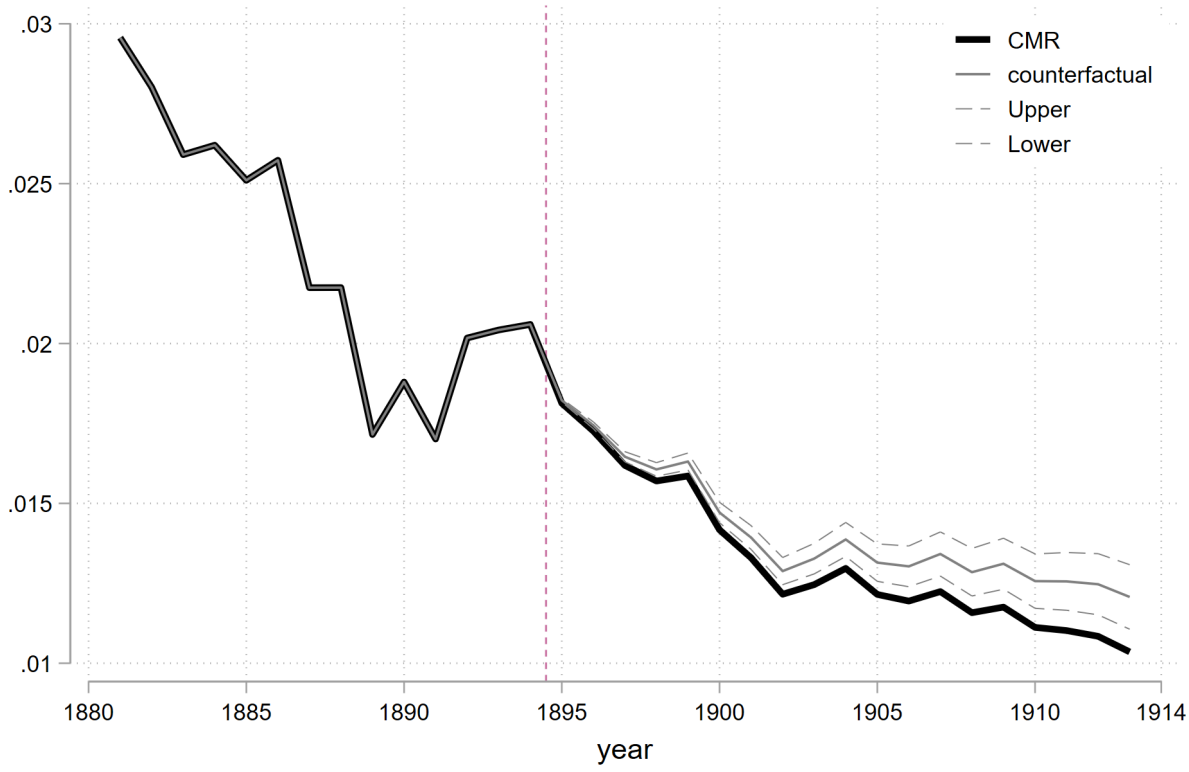


(b) Diphtheria mortality rate



Notes: *This figure uses reduced-form estimates from the different robustness exercises (2SLS estimates reported in Appendix Tables A.5 and A.6) to calculate the counterfactual (CF) developments for life expectancy at birth (Panel A) and the diphtheria mortality rate (Panel B). The gray curve is the baseline model. The green dashed curve controls for all the additional control variables. The yellow curve replace county-year fixed effects with year fixed effects. The red dashed curve use the log-level specification for life expectancy and the Poisson model for the diphtheria mortality rate. The purple curve use non-weighted regressions. The orange dashed curve use treatment intensity that is scaled with the number of children instead of total population. The blue dashed line models the trend to be concave instead of linear. The CF calculations are based on annual delays for average treatment intensity 95-percent confidence are not reported. The solid black curves are the observed population weighted averages of the two outcomes. All curves are three-year moving averages.*

Figure A.10: Counterfactual scenario for child mortality



Notes: *This figure use reduced-form estimates (and 95 percent confidence bands) to calculate the counterfactual (CF) development for the child mortality rate (gray solid curves and dashed ones indicate the 95-percent confidence bands). The CF calculations are based on annual delays for average treatment intensity The solid black curve is the observed population weighted averages of the child mortality rate. All curves are three-year moving averages.*

Table A.1: Summary Statistics by Pre- and Post-antitoxin Periods

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)
	period 1					period 2				
	N	mean	p25	p50	p75	N	mean	p25	p50	p75
diphteria rate	275	0.893	0.656	0.846	1.099	275	0.316	0.234	0.319	0.405
life expectancy	261	42.10	36.01	42.30	46.29	261	45.96	42.04	45.42	50.04
child mortality rate	262	23.35	18.03	24.01	30.80	262	13.04	9.573	12.04	17.74
infant mortality rate	262	152.1	126.8	156.7	168.0	262	132.2	111.4	129.8	137.7
population	275	117,164	6,874	32,270	58,291	275	140,531	9,561	51,428	102,698
antitoxin p.c.	275	0	0	0	0	275	10.40	6.227	10.01	15.10
treatment	275	0.732	0.443	0.647	1.220	275	0.732	0.443	0.647	1.220

Notes: *This table reports summary statistics for selected key variables averaged over the pre-antitoxin period (1880 to 1894) and the post-antitoxin periods (1895 to 1914), using the 1890 municipality population size as weight.*

Table A.2: Treatment Balance

	(1) treatment	(2) treatment	(3) treatment	(4) treatment	(5) treatment	(6) treatment	(7) treatment
ln(infec. rate, 89-94)	0.613***	0.619***	0.607***	0.634***	0.611***	0.619***	0.527***
	(0.076)	(0.171)	(0.161)	(0.146)	(0.148)	(0.163)	(0.153)
ln(stroke rate, 89-94)		-0.079	-0.122*	-0.120*	-0.123*	-0.093	-0.070
		(0.066)	(0.069)	(0.069)	(0.069)	(0.079)	(0.077)
doctors pr. capita in 95			0.092*	0.082*	0.088*	0.128**	0.114**
			(0.051)	(0.049)	(0.049)	(0.054)	(0.053)
dist Boston				-0.004*	-0.003	-0.004	-0.004
				(0.002)	(0.002)	(0.002)	(0.002)
persons pr 1,000 sqm in 95					0.005	0.002	0.003
					(0.009)	(0.008)	(0.008)
persons pr. dwelling in 95						-0.049	-0.056
						(0.034)	(0.036)
persons pr. room in 95						0.944	0.592
						(0.603)	(0.614)
fb share in 95							0.776
							(0.617)
N	275	268	267	267	267	267	267

Notes: *This table shows how different municipality characteristics are related to "treatment" intensity (i.e., the outcome in this table), which is used in combination with the sudden availability of antitoxin in 1895 as our 2SLS strategy. The explanatory variables are explained in Table 1. All regressions control for county fixed effects and are weighted with the 1890 municipality population size in 1890. Robust standard errors clustered in parenthesis. ***,**,* significant at, respectively, the 10, 5, and 1 percent level.*

Table A.3: False Free-antitoxin Start Dates

	(1) dipht	(2) dipht	(3) dipht	(4) dipht	(5) dipht	(6) dipht	(7) life	(8) life	(9) life	(10) life	(11) life	(12) life
antitoxin p.c.	0.015						0.055					
	(0.028)						(0.330)					
antitoxin p.c.		0.015						0.054				
		(0.028)						(0.324)				
antitoxin p.c.			0.011						0.063			
			(0.027)						(0.301)			
antitoxin p.c.				0.008						0.075		
				(0.028)						(0.301)		
antitoxin p.c.					0.004						0.085	
					(0.031)						(0.312)	
antitoxin p.c.						0.001						0.085
						(0.031)						(0.306)
Start	1880	1881	1882	1883	1884	1885	1880	1881	1882	1883	1884	1885
Sample	1880-96	1880-96	1880-96	1880-96	1880-96	1880-96	1880-96	1880-96	1880-96	1880-96	1880-96	1880-96
$N \times T$	4675	4675	4675	4675	4675	4675	4437	4437	4437	4437	4437	4437
N	275	275	275	275	275	275	261	261	261	261	261	261
F-Stat	34.49	25.93	22.44	15.89	11.40	9.653	37.40	28.27	23.49	16.95	12.48	10.77

Notes: *This table reports the results from assuming false start dates for the free distribution of antitoxin in the annual model. In columns 1-6 the outcome is the diphtheria mortality rate and in columns 7-12 the outcome is life expectancy at birth. In columns 1 and 7, we assume that antitoxin was being distributed freely from 1880 onward, in columns 2 and 8, we assume the starting date was the year 1881, etc. The sample includes the years 1880 to 1896. We assume an annual linear trend-break model for the diffusion of antitoxin as outlined in Equation (2). All regressions are weighted by the municipality population size in 1895 and control for municipality and county-by-year fixed effects. Standard errors (in parentheses) account for arbitrary heteroskedasticity and are clustered at the municipality level. ***, **, and * indicate significance at the 1, 5, and 10 percent level.*

Table A.4: Alternative Instrument - Shift-share Instrument

	(1) diph rate	(2) diph rate	(3) life exp	(4) life exp
antitoxin p.c.	-0.110***	-0.122***	0.489***	0.690***
	(0.027)	(0.021)	(0.176)	(0.241)
Weight	yes	no	yes	no
$N \times T$	9135	9135	9135	9135
N	261	261	261	261
F-Stat	31.52	52.45	31.52	52.45

Notes: *This table reports the effects on diphtheria mortality and life expectancy from an alternative instrument that is derived by using the pre-antitoxin (1889 to 94) diphtheria mortality shares to allocate the aggregate number of antitoxin bottles supplied by the SBH to each municipality each year. This predicted number of antitoxin bottles is then scaled by the 1894 municipality population size. See Appendix Section A.2 for more details. In columns 1 and 3, regression are weighted by the municipality population size in 1895. All regressions control for municipality and county-by-year fixed effects. Standard errors (in parentheses) account for arbitrary heteroskedasticity and are clustered at the municipality level. ***, **, and * indicate significance at the 1, 5, and 10 percent level.*

Table A.5: Robustness Checks I

	(1) diph	(2) diph	(3) diph	(4) diph	(5) diph	(6) life	(7) life	(8) life	(9) life	(10) ln(life)
antitoxin p.c.	-0.212***	-0.102***	-0.111***			0.710*	0.487***	0.507***		
	(0.079)	(0.022)	(0.024)			(0.412)	(0.167)	(0.176)		
share 0		0.169*					-0.428			
		(0.094)					(0.763)			
share 1-4		-0.007					-0.516			
		(0.042)					(0.342)			
share 5-9		-0.016					0.309			
		(0.037)					(0.342)			
share 10-14		0.002					0.435			
		(0.031)					(0.296)			
clean water			-0.231***					0.421		
			(0.085)					(0.588)		
hospitals			0.106					-0.786		
			(0.110)					(0.919)		
ln antitoxin p.c.				-1.287***					5.168**	0.131**
				(0.371)					(2.275)	(0.055)
Add controls	yes	yes	no	no	no	yes	yes	no	no	no
Spec.	level-level	level-level	level-level	level-log	r-f	level-level	level-level	level-level	level-log	log-log
estimator	2SLS	2SLS	2SLS	2SLS	Poisson	2SLS	2SLS	2SLS	2SLS	2SLS
$N \times T$	9590	9625	9625	7946	9554	9135	9135	9135	7533	7532
N	274	275	275	275	275	261	261	261	261	261
F-Stat	8.080	39.91	34.91	19.67		8.356	38.79	33.60	20.45	20.45

Notes: *This table reports various robustness checks based on the baseline annual linear trend-break model as outlined in Equation (2). The sample includes the years 1880 to 1914. The outcomes are the diphtheria mortality rates (columns 1-4) and life expectancy at birth (columns 5-8). Columns 1 and 5 include the following municipality-level controls: pre-antitoxin respiratory, waterborne, and stroke mortality rates, doctors pr. capita, distance to Boston, population density, person pr. dwelling, person pr. room, and the foreign-born share (all measured either in 1895 or before and interacted with a full set of year fixed effects). The results presented in columns 2 and 6 account for the roll-out of clean water and hospitals. Results in columns 3 and 7 are based on a level-log model, whereas column 8 reports results using a log-log functional form. The reduced-form coefficient in column 4 is estimated by Poisson. All regressions are weighted by the municipality population size in 1895 and control for municipality and county-by-year fixed effects. Standard errors (in parentheses) account for arbitrary heteroskedasticity and are clustered at the municipality level. ***, **, and * indicate significance at the 1, 5, and 10 percent level.*

Table A.6: Robustness Checks II

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)
	diph rate	diph rate	diph rate	diph rate	life exp	life exp	life exp	life exp	life exp 5-year age	life exp 5-year calendar
antitoxin p.c.	-0.084***	-0.121***		-0.122***	0.506***	0.718***		0.722***	0.697***	0.549**
	(0.016)	(0.018)		(0.018)	(0.170)	(0.239)		(0.242)	(0.260)	(0.234)
antitoxin per child			-0.035***				0.263***			
			(0.007)				(0.087)			
Treatment	log	baseline	children	children	log	baseline	children	children	baseline	baseline
Weight	baseline	no	baseline	baseline	baseline	no	baseline	baseline	baseline	baseline
Sample	baseline	baseline	baseline	excl Boston	baseline	baseline	baseline	excl Boston	baseline	baseline
$N \times T$	8295	9625	9640	9590	9135	9150	9170	9115	9185	9150
N	237	275	276	274	261	262	262	261	263	262
F-Stat	46.10	58.64	38.57	57.33	33.83	56.83	38.07	55.92	56.92	56.83

Notes: *This table reports various robustness checks based on the baseline annual linear trend-break model as outlined in Equation (2). The sample includes the years 1880 to 1914. The outcomes are the diphtheria mortality rates (columns 1-4) and life expectancy at birth (columns 5-10). Columns 1 and 5 use our treatment measure logged. In Columns 2 and 6, the regressions are unweighted. Columns 3 and 7 scale treatment and antitoxin with the number of children (ages 0-10) instead of the total population. In Columns 4 and 8, Boston is excluded from the sample. In Column 9, life expectancy at birth is calculated on the basis of five-year age groups instead of single-year age groups, whereas in column 10 life expectancy at birth is based on five calendar years (1880-85, 1890-94, 1895-89, 1900-04, 1905-09, 1910-14). All regressions control for municipality and county-by-year fixed effects. Standard errors (in parentheses) account for arbitrary heteroskedasticity and are clustered at the municipality level. ***, **, and * indicate significance at the 1, 5, and 10 percent level.*

Table A.7: Controlling for Lagged Mortality Outcomes

	(1) diph	(2) diph	(3) diph	(4) diph	(5) life	(6) life	(7) life	(8) life
antitoxin p.c.	-0.090***	-0.098***	-0.102***	-0.113***	0.473***	0.442***	0.412***	0.408***
	(0.017)	(0.019)	(0.020)	(0.019)	(0.149)	(0.138)	(0.133)	(0.140)
Lags	1	1-2	1-3	1-5	1	1-2	1-3	1-5
$N \times T$	9350	9075	8800	8250	8874	8613	8352	7830
N	275	275	275	275	261	261	261	261
F-Stat	38.02	38.88	40.27	40.68	33.86	34.00	34.11	34.27

Notes: *This table shows 2SLS estimates for the baseline annual linear trend-break model as outlined in Equation (2) when controlling for lagged dependent variables. The number of lags included is reported at the bottom of the table. The outcome variables are the diphtheria mortality rate (Columns 1-4) and life expectancy at birth (Columns 5-8). All regressions control for municipality and county-by-year fixed effects. Standard errors (in parentheses) account for arbitrary heteroskedasticity and are clustered at the municipality level. ***, **, and * indicate significance at the 1, 5, and 10 percent level.*

Table A.8: Effects on Age-specific Mortality Rates

	(1) age 0	(2) age 1	(3) age 2	(4) age 3	(5) age 4	(6) age 5	(7) age 6	(8) age 7	(9) age 8	(10) age 9	(11) age 10
antitoxin p.c.	-2.058	-0.946	-0.784***	-0.551***	-0.567***	-0.423***	-0.282**	-0.031	-0.099	-0.200*	-0.005
	(1.484)	(0.893)	(0.271)	(0.184)	(0.172)	(0.143)	(0.116)	(0.087)	(0.087)	(0.114)	(0.077)
Mean pre-y	189.1	55.35	24.83	17.17	13.14	10.29	8.114	6.958	5.612	4.725	3.968
$N \times T$	9130	9131	9128	9129	9131	9131	9135	9132	9133	9133	9134
N	261	261	261	261	261	261	261	261	261	261	261
F-Stat	33.80	33.76	33.72	33.74	33.76	33.75	33.83	33.77	33.80	33.80	33.81

Notes: *This table reports the effects on q-type age-specific mortality rates (ages 0 to 10) using the linear trend-break model as outlined in Equation (2). The outcomes are expressed per 1,000 individuals of the relevant age group. The sample includes the years 1880 to 1914. All regressions are weighted by the municipality population size in 1895 and control for municipality and county-by-year fixed effects. Standard errors (in parentheses) account for arbitrary heteroskedasticity and are clustered at the municipality level. ***, **, and * indicate significance at the 1, 5, and 10 percent level.*

Table A.9: Stacked Model

	(1) mortality	(2) mortality	(3) mortality	(4) mortality	(5) mortality	(6) cases
antitoxin p.c. x I	-0.082***	-0.110***	-0.089***	-0.067***	-0.057***	-0.048
	(0.019)	(0.030)	(0.020)	(0.016)	(0.015)	(0.062)
Controls	all	exogenous	childhood	declining	waterborne	cases
$N \times T \times D$	100,366	21,507	28,676	43,014	21,507	16,090
N	275	275	275	275	275	250
F-Stat	16.73	16.73	16.73	16.73	16.73	13.84

Notes: *This table reports 2SLS estimated from a stacked model that resembles the baseline model, but the panel is now three-dimensional (municipality-year-disease). We interact the main RHS variables in Equations (1) and (2) with an indicator for diphtheria. Column 1 includes all 13 control diseases (typhoid, tuberculosis, pneumonia, scarlet fever, measles, whooping cough, bronchitis, accidents, suicides, childbirth, meningitis, strokes, and digestive diseases). Column 2 only includes "exogenous" causes as controls (accidents and suicides). Column 3 only includes childhood diseases as controls (scarlet fever, whooping cough, measles). Column 4 only includes diseases where we also observe secular declines during the pre-antitoxin period as controls (typhoid, tuberculosis, scarlet fever, meningitis, and digestive diseases). Column 5 only includes waterborne diseases as controls (typhoid and digestive diseases). In column 6, the outcome in the infection rate (or cases per 1,000 people), where the controls are the infection rates of scarlet fever, typhoid, measles, and smallpox. All regressions are weighted by the municipality population size in 1895 and control for municipality-by-year, disease-by-year-by-county, and municipality-by-disease fixed effects. Standard errors (in parentheses) account for arbitrary heteroskedasticity and are clustered at the municipality level. ***, **, and * indicate significance at the 1, 5, and 10 percent level.*

Table A.10: Antitoxin Treatment and School Attendance: Robustness

	(1)	(2)	(3)	(4)	(5)	(6)
PANEL A			== 1 if attends $\leqq$ 3 months			
Sample	Boys	Girls	FB Parents	US Parents	Dad Skill (Low)	Dad Skill (High)
Exposure	-0.005***	-0.005***	-0.005***	-0.006***	-0.005***	-0.005***
	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)
Observations	148,440	147,514	135,019	108,050	118,095	177,873
R-squared	0.043	0.042	0.047	0.041	0.051	0.038
Mean(Y)	0.106	0.108	0.114	0.0994	0.111	0.105
PANEL B			== 1 if attends $\geqq$ 9 months			
Sample	Boys	Girls	FB Parents	US Parents	Dad Skill (Low)	Dad Skill (High)
Exposure	0.006***	0.006***	0.005***	0.005***	0.005***	0.006***
	(0.001)	(0.001)	(0.002)	(0.001)	(0.002)	(0.001)
Observations	148,440	147,514	135,019	108,050	118,095	177,873
R-squared	0.118	0.108	0.096	0.146	0.120	0.112
Pre-Y mean	0.685	0.676	0.685	0.674	0.674	0.685
Municipality FE	YES	YES	YES	YES	YES	YES
Year of Birth FE	YES	YES	YES	YES	YES	YES
Ind. Controls	YES	YES	YES	YES	YES	YES

Notes: *This table reports how antitoxin exposure affected school attendance. The sample includes 5 to 15-year-old children in Massachusetts in 1900. The dependent variable is a dummy of whether a child attended school for no more than three months in Panel A and for at least nine months in Panel B. The variable of interest, "Exposure", denotes the average number of antitoxin bottles per 1,000 people that a child during the first nine years was exposed to (see page 22 for details). All regressions control for municipality and year of birth fixed effects and include the following set of controls: dummies for gender (except columns 1-2), place of birth, rural, year of immigration, and a set of parental controls including dummies for mother's and father's birthplace, their year of immigration, age of the mother and father, mother's and father's literacy, whether the father or mother was absent at the time of the census, and whether the father worked in a white-/blue-collar skilled occupation. Standard errors (in parentheses) account for arbitrary heteroskedasticity and are clustered at the municipality level. ***, **, and * indicate significance at the 1, 5, and 10 percent level.*

Table A.11: Antitoxin Treatment and School Attendance in 1900 – Linked Sample

	(1)	(2)	(3)	(4)	(5)	(6)
	==1 if attends at all		== 1 if attends ≦ 3 months		== 1 if attends ≧ 9 months	
Exposure	0.003	0.003	-0.005***	-0.005***	0.006***	0.006***
	(0.003)	(0.003)	(0.001)	(0.001)	(0.001)	(0.001)
Observations	241,391	241,359	174,258	174,218	174,258	174,218
R-squared	0.164	0.176	0.035	0.041	0.111	0.119
Municipality FE	YES	YES	YES	YES	YES	YES
Year of Birth FE	YES	YES	YES	YES	YES	YES
Ind. Controls	NO	YES	NO	YES	NO	YES
Mean pre-y	0.722	0.722	0.103	0.103	0.685	0.685

Notes: *This table replicates Table 6 using the linked sample (1900 to 1940). The sample includes 5 to 15-year-old children in Massachusetts in 1900 that can be linked to 1940 using the crosswalks from the Census Tree Project. The dependent variable is a dummy of whether a child attended school at all (columns 1-2); for no more than three months (columns 3-4); and for at least nine months (columns 5-6). The variable of interest, "Exposure", denotes the average number of antitoxin bottles per 1,000 people that a child during the first nine years was exposed to (see page 24 for details). All regressions control for municipality and year of birth fixed effects. Columns 2, 4, and 6 also include the following set of controls: dummies for gender, place of birth, rural, year of immigration, and a set of parental controls including dummies for mother's and father's birthplace, their year of immigration, age of the mother and father, mother's and father's literacy, whether the father or mother was absent at the time of the census, and whether the father worked in a white-/blue-collar skilled occupation. Standard errors (in parentheses) account for arbitrary heteroskedasticity and are clustered at the municipality level. ***, **, and * indicate significance at the 1, 5, and 10 percent level.*

Table A.12: The Long-run Effects of Antitoxin Treatment

	(1) low-skilled	(2) blue-collar skilled	(3) white-collar	(4) ln(occscore)
Panel A: Sample 1900-1920				
Exposure	-0.0027***	0.0003	0.0029***	0.0005
	(0.0007)	(0.0005)	(0.0005)	(0.0007)
Observations	245,301	245,301	245,301	162,487
R-squared	0.1131	0.1537	0.0672	0.1064
Mean(Y)	0.234	0.135	0.284	3.229
Panel B: Sample 1900-1930				
Exposure	0.0003	-0.0004	0.0004	-0.0004
	(0.0005)	(0.0004)	(0.0006)	(0.0005)
Observations	261,097	261,097	261,097	179,878
R-squared	0.0968	0.1206	0.1053	0.1197
Mean(Y)	0.217	0.135	0.325	3.290
Municipality FE	YES	YES	YES	
Year of Birth FE	YES	YES	YES	YES
Ind. Controls	YES	YES	YES	YES

Notes: *This table reports how antitoxin exposure affected labor market outcomes of exposed children as adults. The sample includes 5 to 15-year-old children in Massachusetts in 1900 linked to 1920 (Panel A) and 1930 (Panel B). The dependent variable is a dummy of whether the person works in the terminal year in a low-skilled (column 1), blue-collar skilled (column 2), or white-collar occupation (column 3), and the ln occupational income score (column 4). The variable of interest, "Exposure", denotes the average number of antitoxin bottles per 1,000 people that a child during the first nine years was exposed to (see page 24 for details). All regressions control for municipality and year of birth fixed effects and the following set of controls: dummies for gender, place of birth, rural, year of immigration, and a set of parental controls including dummies for mother's and father's birthplace, their year of immigration, age of the mother and father, mother's and father's literacy, whether the father or mother was absent at the time of the census, and whether the father worked in a white-/blue-collar skilled occupation. Standard errors (in parentheses) account for arbitrary heteroskedasticity and are clustered at the municipality level. ***, **, and * indicate significance at the 1, 5, and 10 percent level.*