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

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Abstract

We examine the impact of the free supply of diphtheria antitoxin, the first effective medical treatment for an infectious disease, on the historical health transition in Massachusetts. Using newly collected municipality-level data on the distribution of antitoxin and information from over 1.5 million death certificates from 1880 to 1914, we find that the rapid availability of antitoxin treatment significantly increased life expectancy at young ages. Our findings suggest that medicine, combined with an effective public health policy, played a more important role in improving life expectancy in the early 20th century than previously thought.

Keywords: Child mortality, life expectancy; medical technology; public health policy.
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1 Introduction

Life expectancy has increased 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 one born in the 1880s. The health transition in the United States began in the late 19th century, driven by improvements in nutrition and public health measures that reduced the prevalence and mortality of infectious diseases, the leading causes of death at the time (Cutler et al. 2006; Costa 2015). While there is an ongoing debate about the relative importance of various contributing factors, such as better living conditions and various public health programs, these advancements together likely played a key role in the early rise of life expectancy.[1] It is also widely claimed that medical treatments 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; Jayachandran et al. 2010; Catillon et al. 2018).

This paper studies the relationship between medical innovations, public health policy, the decline in infectious diseases, and improvements in life expectancy *before* the introduction of sulfonamides ("sulfa" drugs") in the late 1930s. We focus on the first widely-used medical treatment against an infectious disease: the diphtheria antitoxin. German physiologist, Emil von Behring invented the antitoxin serum in 1890, 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). We find that the free distribution policy of the antitoxin serum in the state of Massachusetts led to a decline in diphtheria mortality, increased life expectancy, and reduced school absenteeism, suggesting that medicine, in combination with an effective public health policy, played a more important role in the rise of life expectancy in the early 20th century than previously thought.

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 demographers question the effective use of the diphtheria antitoxin in the early 20th century

[1] Specifically, the early 20th-century decline in mortality from infectious diseases is attributed to different key factors: clean water technology and sanitation (e.g., Cutler and Miller 2005; Alsan and Goldin 2019), improved nutrition (e.g., Fogel 1994, 2004; McKeown 1976), better living conditions (e.g., Ager et al. 2024), public health programs (e.g., Moehling and Thomasson 2014; Egedesø et al. 2020), and environmental changes (e.g., Barreca et al. 2016; Beach and Hanlon 2018; Hanlon et al. 2021; Hanlon 2022). However, the relative importance of these factors remains controversial; some researchers even doubt their significance in explaining the observed mortality decline (e.g., Anderson et al. 2019, 2022; Clay et al. 2020).

(Preston and Haines 1991; Condran 2008; Thomasson 2018). Although the clinical effectiveness of the antitoxin serum was well-documented, its quantitative impact on population health remains controversial and its socioeconomic implications are even unknown.

Our goal is to examine whether the innovation of the diphtheria antitoxin serum, combined with an effective public health policy, contributed to gains in life expectancy before the "era of big medicine".[2] To do so, our empirical analysis utilizes newly collected municipality-level data on the diffusion of antitoxin and over 1.5 million death certificates in Massachusetts dating from 1880 to 1914. Using Massachusetts as a case study to evaluate the population health effects of the diphtheria antitoxin has several advantages. First, Massachusetts' State Board of Health (SBH) implemented a policy that provided the antitoxin to *all residents* free of charge, thereby reducing the possible income gradient in its adoption. This free-supply policy has subsequently been regarded as a milestone in the state's public health history. Second, the SBH kept a record of the number of antitoxin bottles distributed to each municipality from the beginning of the serum production in Massachusetts in 1895 to 1914. Compared to existing studies that evaluate the effects of medical innovations in the 1930s and 1940s, we can measure the uptake of antitoxin at the turn of the 20th century at the local level.[3] Finally, historical vital statistics from Massachusetts have been well-documented and are considered reliable.

We use individual death certificates to calculate infant and child mortality rates, as well as life tables from which we derive life expectancy for over 300 municipalities. This data allows us to examine whether the rapid and free diffusion of the diphtheria antitoxin led to a substantial increase in life expectancy and a decrease in infant and child mortality at the municipality level. One further advantage of our study is that we can evaluate the short- and long-term consequences of exposure to antitoxin during childhood. By combining the antitoxin roll-out with the complete-count U.S. census records, we can test whether exposure to antitoxin during childhood had any immediate impact on school attendance. In addition, with the latest links between censuses, we follow a sample of children residing in Massachusetts and examine whether antitoxin coverage during childhood affected labor market outcomes in their adulthood.

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

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

[3]In contrast, those studies are unable to directly measure the local diffusion of the technologies and need to rely on indirect measures (e.g., Acemoglu and Johnson 2007; Jayachandran et al. 2010; Alsan et al. 2021; Bhalotra et al. 2023).

strategy exploits that Massachusetts' public health policy led to a relatively rapid diffusion of antitoxin. 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 were historically more widely affected by diphtheria. Differential diphtheria mortality rates across municipalities before the introduction of the antitoxin treatment and the free-distribution policy 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 economic effects of significant health improvements (e.g., Acemoglu and Johnson 2007; Ager et al. 2018; Bütikofer and Salvanes 2020).

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 free-antitoxin policy; 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: (i) the inclusion of the pre-antitoxin mortality environment; (ii) controlling for other public health interventions (public water works 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. We also show that the "antitoxin effect" does not simply capture affected municipalities expanding their healthcare sector (by increasing the supply of doctors and nurses per capita).

Overall, our results suggest that the availability of the diphtheria antitoxin in Massachusetts improved the mortality environment, in particular for children. Our counterfactual analysis suggests that without antitoxin, combined with a free-distribution policy, diphtheria would have claimed approximately 1,800 additional lives, and life expectancy at age 1 would have been nearly two years lower by the end of our sample period. The evidence suggests that the diphtheria antitoxin increased life expectancy primarily by saving children, 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 conclude our empirical analysis by evaluating 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. First, we 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 by a more effective containment of diphtheria. To identify the effects, we ask whether children attended school for more months if they were exposed to the antitoxin treatment in their municipality. Our results suggest that this was the case and that the reduction in school absenteeism is quantitatively sizable. However, the fewer days absent from school for antitoxin-exposed children did not translate into any sizable effects on years of schooling and adult labor market outcomes.

Did the free distribution of antitoxin in Massachusetts primarily reduce consumer costs or also cost-effectively save lives? We found that both were the case. Compared with the cheapest private alternative, public production of antitoxin in Massachusetts saved consumers nearly $50 million (in 2023 USD) from 1895 to 1914. An important feature of this public health policy was that the treatment was freely available to even the poorest Massachusetts residents who otherwise would not have had the financial means to afford treatment with the serum. In light of this, we show that the cost per saved life was very cheap and ranged between $1,050 - $4,600 in 2023 USD, comparable to the cost of preventing death from tuberculosis through information provision (Egedesø et al. 2020). Hence, our study is an important illustration of how the combination of medicine and effective public health policy saved lives, made possible by the fact that the serum was never patented in the United States.

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. (2024) 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 two decades of the 20th century when infectious diseases started their long-term decline. We find that the free distribution of diphtheria antitoxin saved numerous children's lives, resulting in significant gains in life expectancy.

Our result—that the free distribution of antitoxin mostly reduced child mortality rates in late 19th- and early 20th-century—adds to the ongoing debate about the drivers of mor-

tality decline during the early stages of the historical health transition. While much of the existing literature has focused on interventions primarily affecting infants, the factors behind declines in childhood mortality have received less attention (see, e.g., Karbownik and Wray 2025). Our study highlights that the diphtheria antitoxin was more effective than previously recognized (Preston and Haines 1991; Condran 2008; Thomasson 2018), demonstrating that medical innovations, combined with progressive public health policies, contributed to the decline in infectious disease rates and improvements in childhood health much earlier than often assumed.

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 sizable (close to zero) 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 production and distribution in Massachusetts. Finally, we discuss the need for an appropriate 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.[4] 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. Left untreated, diphtheria can obstruct the airways and cause suffocation.[5] 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.[6]

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). In Germany, the country where the antitoxin was invented, diphtheria mortality rates were at similar levels (1.06 per 1,000 people) during this period. 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, vital records show that diphtheria accounted for up to 10% of all deaths during the epidemic years of 1863 and 1876-77, fluctuating around 4-5% in other years prior to the introduction of antitoxin. It was recognized by the state's vital registration authorities as a major cause of death, claiming more lives than other important childhood diseases, such as typhoid, measles, and scarlet fever (Appendix Figure A.1). 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

[4]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).

[5]The profession faced problems with the correct diagnosis in the early stages of a case of diphtheria or when only mild symptoms appeared. In this case, diphtheria resembled other forms of infectious diseases (e.g., throat infections). Hammonds (1999, p.9) writes: *"Until the 1890s diphtheria was a difficult disease to diagnose and to treat, and no cure was available."* In the early 1890s, the bacteriological diagnosis improved the detection of diphtheria.

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

1 accounted for only 0.4% of the deaths that year. For the U.S. death-registration area in 1910-14, the age distribution of diphtheria deaths is similar, and diphtheria was somewhat more deadly for boys under the age of 5, whereas girls between the ages of 5 and 9 were at slightly higher risk (Crum 1917). Moreover, the vital 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.

Figure 1 illustrates the diphtheria and croup (an upper respiratory infection that doctors often used colloquially for diphtheria infections at the time) mortality rate in Massachusetts from 1858 to 1914. The figure highlights the previously mentioned epidemics, with mortality peaking at nearly 2 deaths per 1,000 people. The epidemic year in 1876 alone resulted in approximately 3,300 deaths.[7] The dashed lines show trends for the periods 1858-1894 (pre-antitoxin period) and 1895-1914 (post-antitoxin period), revealing a trend break.[8] While diphtheria mortality averaged 0.9 deaths per 1,000 people in the pre-antitoxin period, it fell to around 0.2 deaths per 1,000 people in 1910. Although the disease was not as deadly as other infectious diseases, such as tuberculosis (Appendix Figure A.1), the skewed mortality-age profile suggests that a reduction in diphtheria deaths would have had a significant impact on life expectancy at younger ages. 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.

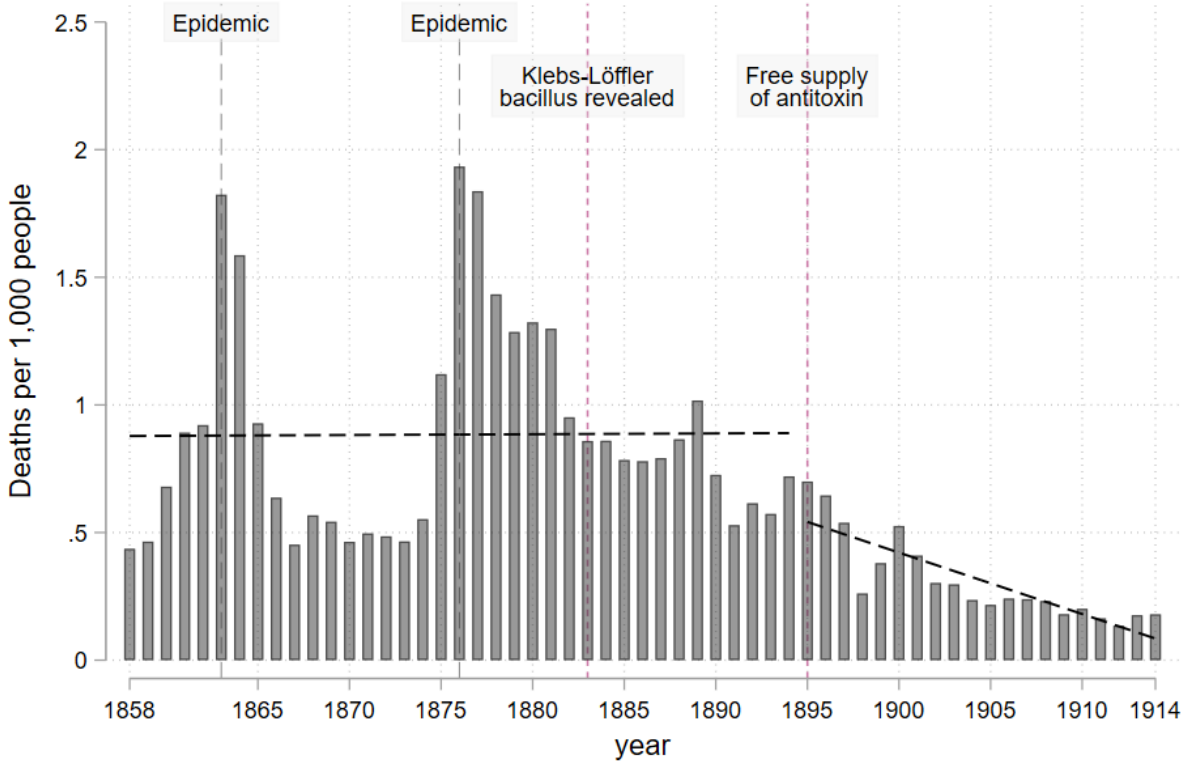
2.2 Antitoxin and its distribution in Massachusetts

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. 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

[7] A similar graph presented in the 1902 SBH report suggested that post-epidemic mortality rates were likely influenced by the epidemics themselves, complicating any conclusions about the aggregate mortality pattern prior to the free-distribution policy in 1895.

[8] On the other hand, if the pre-antitoxin period starts in 1880, as in our municipality regression analysis, there is no clear aggregate trend break. However, these aggregate trends mask the fact that some locations experienced larger declines in diphtheria mortality, but only in the post-antitoxin period, which we document in Section 5.

Figure 1: Death rate from diphtheria and croup



Notes: *The figure illustrates the trend in deaths from diphtheria and croup per 1,000 people in the State of Massachusetts from 1858 to 1914, based on data from the Massachusetts vital statistics (various years). Diphtheria was first recorded as a separate cause of death in 1858. The dashed lines represent the average death rates before and after the introduction of antitoxin in 1895. The years 1863 and 1876 are highlighted as epidemic years, as defined by SBH (1901).*

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 occurred after Pierre Paul Émile Roux, a French physician, bacteriologist, and immunologist, reported on the effectiveness of antitoxin in hospitals throughout Europe at the International Congress of Hygiene and Demography in Budapest in 1894. While Behring "initiated" patent applications in Germany, the United Kingdom, and the United States, the German patent law protected the manufacturing process, but not the product itself. Hence, only the trademark "Behring's Diphtherie-Heilmittel" was protected (Liebenau 1987; Hess 2008). This meant that anyone could copy the serum (as a product) and there was no U.S. patent restricting the production of the serum.[9] It was immediately manufactured for large-scale public health distribu-

[9]Behring applied for a U.S. patent for producing diphtheria antitoxin in horses in 1895, although

tion without the risk of patent infringement. Public health departments used large stables equipped with inoculated horses to produce vats of extracted blood and serum. In the United States, 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. In the first years, the diphtheria antitoxin was produced in the laboratory rooms at the Bussey Institution at Harvard University and then relocated to the laboratory of the SBH at Forest Hills (MA). Production of the serum increased rather quickly from 1,724 bottles in 1895 to 53,389 in 1900. The units in each bottle (i.e., the strength) were 1,000 units from 1895 to 1898 and 1,500 units from 1899 to 1914. In 1901, each patient would be given on average 5 bottles (Massachusetts State Board of Health 1902).[10] Over the next 14 years, production more than doubled, and in 1914 the SBH distributed 118,561 bottles of antitoxin.

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. We digitized these reports and used 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, making 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 antitoxin treatment of diphtheria 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 antitoxin in the 1890s. According to Crum (1917), the pre-antitoxin diphtheria mortality rates (considering 1889-

Roux had developed the method first. Nevertheless, the patent was granted in 1898, albeit without significant financial benefit to Behring, as Hoechst never enforced it due to fierce resistance from the medical profession and American pharmaceutical companies, such as Parke, Davis & Co. and the H. K. Mulford company, who became the first commercial producer of diphtheria antitoxin in the United States (Hess 2008; Beauchamp 2012; Kaufmann 2017).

[10]In 1901, the quantity of treatment given to patients varied from less than 1,000 units to more than 20,000 units (equivalent to 0.67-13 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.

93) 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 the antitoxin period. These results demonstrated the enthusiasm of medical contemporaries at the time regarding the breakthrough in treating diphtheria infections with the serum. Despite this, diphtheria mortality rates remained high in many countries at the beginning of the 20th century. The main issue was not about the serum's power—it remained practically unchanged for six months and thereafter only diminished very slowly (Massachusetts State Board of Health 1906, p.536, 541), but there were issues about unequal access, the inefficient deployment of the antitoxin serum, and the stage of the disease at which patients received the antitoxin treatment.[11]

These issues appear to play less of a role in Massachusetts. First, the free antitoxin policy addressed this issue of unequal access. Second, the instant supply of antitoxin was not a concern. According to the Massachusetts 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."* Moreover, public health officials were well aware that the antitoxin had to be given at an early stage to ensure the most effective treatment of diphtheria. The statistics published by the SBH suggest that antitoxin was used in Massachusetts efficiently whenever the diagnosis was made early. This is evidenced by the numerous documented cases where antitoxin was given to sick children in the first two days. While it was undoubtedly important to start the antitoxin treatment as early as possible, the fatality rates were still lower for cases where the serum treatment was not begun until after six days than in the period before antitoxin treatment was available, suggesting that the lethality of the disease was still reduced by the treatment to some extent. Overall, 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 (1895-1901). The SBH estimated that the antitoxin treatment saved 10,697 lives in these seven years (Massachusetts State Board of Health 1902, p.487).

Finally, in terms of evaluating the effectiveness of antitoxin, 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

[11]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. Contemporaneous studies also mentioned the issues 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 (Massachusetts State Board of Health 1902, p.486).

of the antitoxin in the decline of diphtheria. 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 combined with a proper identification strategy 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 young ages. Before outlining our identification strategy, we describe 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) "*The Annual Death Registers and Certificates*"; (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, 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 (also referred to as *"diseases"*), 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 per 1,000 people.[12] In addition, we use statistics on the following diseases in our analysis: bronchitis, digestive diseases (diarrhea, cholera, dysentery), tuberculosis, pneumonia, scarlet fever, whooping cough, typhoid, whooping cough, measles, apoplexy (i.e., sudden death), and accidental deaths.

The calculation of annual age-specific mortality rates at the municipality level is based on

[12]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.

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).[13] Our annual age-specific mortality rates are calculated as death counts over the population of that age group. Appendix Section A.1 provides further details on how we use and tabulate information from the death certificates.[14]

Using age-specific mortality rates, we construct municipal life tables for each year in our sample. For these calculations, we assume that deaths are evenly distributed across calendar years for all ages, except during the first year of life, where an infant death is assumed to correspond to one-third of a life year lived. The life table is truncated at age 79, with a maximum life expectancy set at 79.5 years. From these life tables, we calculate life expectancy at all ages, with a primary focus on life expectancy at age 1 as our main measure of population health. Appendix A.2 provides a detailed explanation of the life table construction, including assumptions, limitations, and alternative approaches to the single-year life table

We collected and digitized data on the supply of antitoxin (source iii) for each municipality (SBH, various years). If a municipality was not listed in a given count-year, it is assumed they did not receive any antitoxin directly from the SBH. However, according to the SBH, redistribution of antitoxin from listed to non-listed municipalities cannot be entirely ruled out, but for our analysis, we assume that non-listed municipalities did not receive any antitoxin bottles. Municipalities never listed during the years 1895 to 1914 are dropped from the baseline sample (results remain robust when including them as zeros).[15] From SBH (various years), we also collected annual data on the number of reported infections (cases) for diphtheria. Coverage expanded from 68 municipalities in 1891 to approximately 300 municipalities by the end of the sample period. As the SBH noted, the case data likely suffer

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

[14]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.

[15]The bottle-count year in the SBH reports does not align perfectly with the calendar year. For instance, SBH (1899) reports the supply for the period from March 1895 to March 1896. To address this, we assign the count year to the calendar year with the greatest overlap in terms of months.

from under-reporting and should be interpreted with caution. Nevertheless, we use this data to calculate case fatality and prevalence rates.

The population of Massachusetts was enumerated every fifth year from 1880 to 1915 through state and federal censuses. We obtain total population counts by municipality and census year from the vital statistics (source i), which reprinted the census results, and use log-linear interpolation to estimate population figures for the intervening years. The Massachusetts State Census, conducted every 10 years starting in 1855, provides detailed population statistics and other municipal characteristics, including the number of dwellings, rooms per dwelling, population density, and the share of foreign-born individuals. These data, digitized by Haines (2022), are utilized for different state census years (source iv).

We also use the complete count U.S. Census records (source iv) to measure population size by age groups, which we use to construct life tables (the 1890 records were lost). The aggregation of the census data at the municipality level is based on geo-referenced crosswalks 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 log-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 (source v). 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,[16] 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). The data sources and variable definitions are further explained in Appendix A.3 and A.4.

Adjusting for municipality boundary changes between 1876 and 1914 yields a sample of 339 municipalities, 57 of which never received any antitoxin from the SBH. Thus, our baseline

[16]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).

sample includes 282 municipalities. Using complete-count U.S. census data, which relies on geo-referenced crosswalks from Berkes et al. (2021), excludes two additional municipalities (Cohasset and Norwell).[17]

Appendix Table A.1 presents summary statistics for the main variables, separately for the pre-antitoxin period (1880-1894) and the post-antitoxin period (1895-1914). On average, diphtheria mortality declined in our sample from 0.87 in the pre-antitoxin period to 0.32 in the period when antitoxin became available. Child mortality (age 1-4) also declined substantially from 23 to 14 per 1,000 one to four-year-old children over the same period, while average life expectancy at age 1 increased from 49 to 54.

4 Estimation strategy

We start the empirical analysis by estimating the relationship between the local adoption of antitoxin and different mortality measures, as outlined by the following equation:

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

where y_{mt} is some measure of mortality (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 age 1, but we also report estimates for infant and child mortality as well as other diseases, infections, and fertility. The municipality-specific antitoxin adoption is given by $antitoxin_{mt}$, which is antitoxin bottles per 1,000 people supplied to a municipality m in year t by the SBH.[18] Since the SBH started its production and free supply of antitoxin in the Spring of 1895, this variable is by construction zero for all municipalities before this year. Municipality and year fixed effects are denoted by μ_m and μ_t, respectively. In the baseline specification, the year fixed effects are interacted with county fixed effects to account for unobserved county-specific trends. We will also show results using a less restrictive specification that only accounts for municipality and year fixed effects. The vector $\mathbf{X}'_{mt}$ contains various pre-antitoxin municipality characteristics interacted with year fixed effects and time-varying controls, such as the opening of hospitals. These controls are *only* included

[17] Consistent boundaries from 1876 are applied due to the construction of the denominator data for infant and child mortality (Appendix Section A.5).

[18] To address very high per capita bottle values, we replace values above the 95th percentile with the 95th percentile value (i.e., "winsorized" values), which increases the strength of the first stage. These high values are primarily due to antitoxin supplies allocated to hospitals. When we manually account for the number of bottles supplied to each hospital, the first-stage estimates for bottles per capita remain equally strong even without winsorizing (Appendix Figure A.2).

in the robustness analysis and explained in more detail below. The regression is weighted by the municipality population size in 1895, hence, estimates reflect changes for the average person in Massachusetts.[19] The error term is ε_{mt} and standard errors are, in the baseline, clustered at the municipality level.

While estimating equation (1) controls for time-invariant differences across municipalities and possible 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. This strategy exploits the fact that the SBH's policy made antitoxin freely available since production began in Massachusetts in 1895 and that some municipalities would benefit more from this policy than others because they were systematically more affected by diphtheria before antitoxin became available. 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, which allows us to estimate the following first-stage equation:

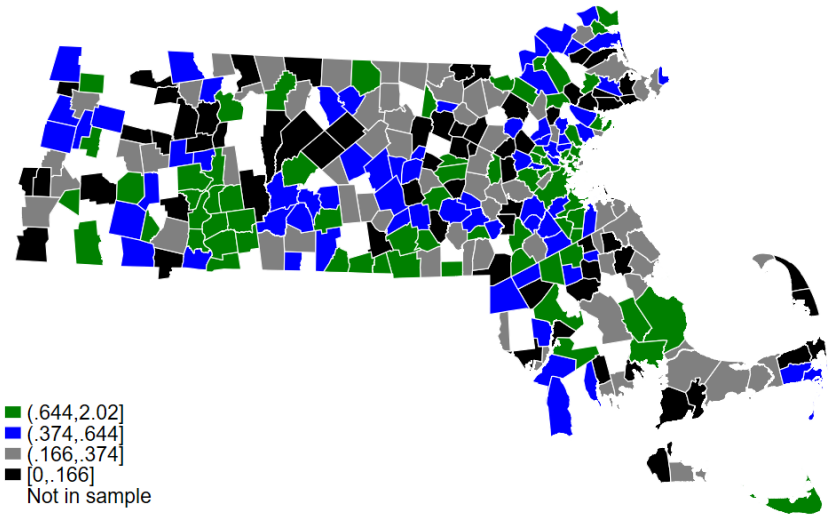
$$antitoxin_{mt} = \gamma treatment_m \times I_t \times (t - 1894) + \mu_m + \mu_{ct} + \mathbf{X}'_{mt}\Omega + \epsilon_{mt}, \tag{2}$$

where $treatment_m$ is the average pre-antitoxin (1888-94) diphtheria mortality rate, which is our cross-sectional measure of treatment intensity, I_t is a post-1895 indicator, and $(t-1894)$ is a linear trend. If fatality rates were similar across municipalities before the introduction of the antitoxin—a reasonable assumption given the lack of medical treatment options—$treatment_m$ reflects pre-antitoxin differences in disease prevalence across locations. Figure 2 depicts the spatial distribution of $treatment_m$, and illustrates the coverage of the baseline sample of municipalities. The remaining variables are defined as in equation (1). Both $antitoxin_{mt}$ and I_t are zero for all municipalities before the policy in 1895. The estimated coefficient of interest, $\hat{\gamma}$, quantifies how differences in the pre-antitoxin diphtheria mortality rates (or prevalence) translate into differences in the adoption of the antitoxin. The linear-trend specification is motivated by the gradual adoption of antitoxin in Massachusetts. If

[19] One 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. Nonetheless, we are also going to report unweighted estimates.

$\hat{\gamma} > 0$, this would imply that municipalities more affected by diphtheria before 1895 had a higher uptake of antitoxin when the technology became freely available for adoption.

Figure 2: Spatial variation in pre-antitoxin (1888-1894) diphtheria mortality rates by municipality



Notes: *This figure shows the spatial variation in treatment intensity measured as the average pre-antitoxin (1888-94) diphtheria mortality rate for the baseline sample of municipalities.*

The validity of this empirical design relies on the assumption that municipalities with different levels of treatment intensity followed similar trends in outcomes prior to the introduction of the free-distribution policy (i.e., the parallel pre-trends assumption). To assess this, we first present descriptive event studies, where we divide municipalities into quartiles based on treatment intensity and depict the average development of the outcomes for each group. Second, we provide more formal evidence by estimating reduced-form event-study coefficients using the following specification

$$y_{mt} = \sum_{k=1880-82}^{T} \alpha_k treatment_m \times I_t^k + \mu_m + \mu_t + \tilde{\varepsilon}_{mt}, \tag{3}$$

where $treatment_m$ is interacted with 3-year pooled fixed effects ($\sum_{k=1880-82}^{T} I_t^k$), with 1892-94 as the omitted baseline 3-year bin. The final pooled years in the sample are 1913-14. Pooling years increases statistical precision while using a single baseline year introduces greater uncertainty, which is particularly problematic for outcomes measured noisily at the annual level in smaller municipalities. Nevertheless, we also report estimates from an annual event-study model and an alternative approach where coefficients are restricted to follow a

spline function in the Appendix.[20] In the baseline reduced-form event studies, we also report confidence bands allowing for linear smoothness according to Rambachan and Roth (2023). Third, we also exploit the sharp cut-off date in 1895 to conduct falsification exercises in which we show that the instrument cannot explain changes in diphtheria mortality and life expectancy in the pre-antitoxin period. As discussed in Appendix Section A.6, our instrument closely parallels an alternative formulation in which the aggregate supply of bottles is allocated based on pre-antitoxin municipality diphtheria mortality shares. This shift-share IV design features one shifter and one share, aligning naturally with the identification framework proposed by Goldsmith-Pinkham et al. (2020). Our DiD setup naturally focuses on identification through the shares, and we implement their recommended falsification tests within our empirical framework.[21]

5 Results

5.1 Descriptive characteristics

In this subsection, we first examine how municipalities with different treatment intensities differed in pre-antitoxin observable municipality-level characteristics. We then present descriptive event studies, which not only provide insights into whether we can support the main identifying assumption of parallel trends but also illustrate the development of key outcome variables throughout the sample period.

Table 1 presents weighted least squares estimates from regressing treatment intensity on various pre-antitoxin municipality characteristics. Several observations are worth noting. First, municipalities with higher pre-antitoxin diphtheria mortality rates also experienced elevated mortality from other infectious diseases. This pattern is in part driven by urban characteristics, such as population density and persons per dwelling. Higher treatment municipalities also had a higher number of doctors relative to their population. Including county fixed effects does not alter these key insights (not shown in the table).

While our empirical design allows these pre-antitoxin level differences to be present, such imbalances increase the risk of our estimates capturing correlated shocks (e.g., other health interventions, such as improvements in water quality or the establishment of hospitals). One approach to address this would be to control for these imbalances directly (e.g., with year fixed effects). However, doing so could obscure potential mechanisms, particularly those re-

[20] Alsan and Goldin (2019) use a 2-event-year pooling event-study model. See also Miller (2023) for an excellent discussion of different approaches within the event-study framework.

[21] In our setup, computing the so-called Rotemberg weights is not meaningful since there is only one share, and it is clear that our identification derives entirely from the diphtheria mortality share.

Table 1: Treatment balance

DV: *treatment*	(1)	(2)	(3)	(4)	(5)	(6)	(7)
infec. rate, 88-94	0.124***	0.124***	0.100***	0.099***	0.068***	0.046	0.050*
	(0.038)	(0.038)	(0.022)	(0.022)	(0.026)	(0.030)	(0.028)
apoplexy rate, 88-94		-0.017	-0.200**	-0.199**	-0.226***	-0.106	-0.117
		(0.151)	(0.084)	(0.085)	(0.081)	(0.086)	(0.082)
doctors pp. in 95			0.216***	0.199***	0.167***	0.204***	0.201***
			(0.055)	(0.053)	(0.048)	(0.048)	(0.046)
dist Boston				-0.001	0.000	0.000	0.000
				(0.001)	(0.001)	(0.001)	(0.001)
persons pr 1,000 sqm in 95					0.022**	0.018**	0.018**
					(0.009)	(0.008)	(0.008)
persons pr. dwelling in 95						-0.035	-0.031
						(0.035)	(0.035)
persons pr. room in 95						1.287***	1.456***
						(0.482)	(0.530)
fb share in 95							-0.352
							(0.489)
N	282	282	280	280	280	280	280

Notes: *This table shows how different municipality characteristics are related to "treatment" intensity. "infect. rate" is the sum of mortality rates of eight infectious diseases (see full list in the data appendix); "apoplexy rate" is the mortality rate of the apoplexy (sudden death). Both averaged over the years 1888 to 1894. "doctors pp. in 95" is the number of doctors per 1,000 people in 1895; "dist Boston" is the aerial distance to Boston; "pers. pr 1,000 sqm" is measured as the number of people per 1,000 square miles in 1895; "pers. pr. dwelling in 95" is the number of people per dwelling in 1895; "pers. pr. room in 95" is the average number of persons per room in 1895; "fb share" is the share of foreign-born individuals in 1895. All regressions are weighted with the municipality population size in 1895. Robust standard errors are clustered in parenthesis. ***,**,* significant at, respectively, the 10, 5, and 1 percent level.*

lated to competing risks and co-mortality dynamics between diphtheria and other infectious diseases. Thus, our baseline specification excludes these controls, but in the robustness analysis, we examine the sensitivity of our 2SLS estimates to their inclusion, alongside controls for the roll-out of public waterworks, the establishment of different types of hospitals, and population shares.

We next present descriptive event studies, illustrating the development of key outcomes for municipalities grouped by treatment quartiles. Specifically, we display the population-weighted averages of the diphtheria mortality rate, life expectancy at age 1, and antitoxin bottles per 1,000 inhabitants for each group. Figure 3 is based on three-year moving averages

to smooth annual fluctuations and highlight broader trends. Panel (a) shows the development of the diphtheria mortality rate over time. In Panel (b), we adjust for initial differences by subtracting the 1894 diphtheria mortality rate from each group, bringing the analysis closer to the formal event-study framework. While the figure reveals a steady decline in diphtheria deaths throughout the sample period, convergence across groups primarily occurred during the post-antitoxin era. Panels (c) and (d) present the development of life expectancy at age 1, following the same structure. As with the diphtheria mortality rate, convergence across municipality groups mainly occurred after 1895. From 1880 to 1894, the gap in life expectancy between the most and least affected groups remained relatively stable at approximately 7-8 years but narrowed to around 2.5 years by 1914.[22]

Finally, Panel (e) illustrates the supply of antitoxin per 1,000 people, showing that municipalities with higher diphtheria incidence prior to the introduction of antitoxin consistently demanded more antitoxin per capita during the antitoxin period. Altogether, these descriptive event studies suggest that municipalities with the potential to benefit more from the free supply of antitoxin also adopted more of it, and these municipalities experienced larger declines in diphtheria mortality rates and larger increases in life expectancy.

5.2 Antitoxin and population health

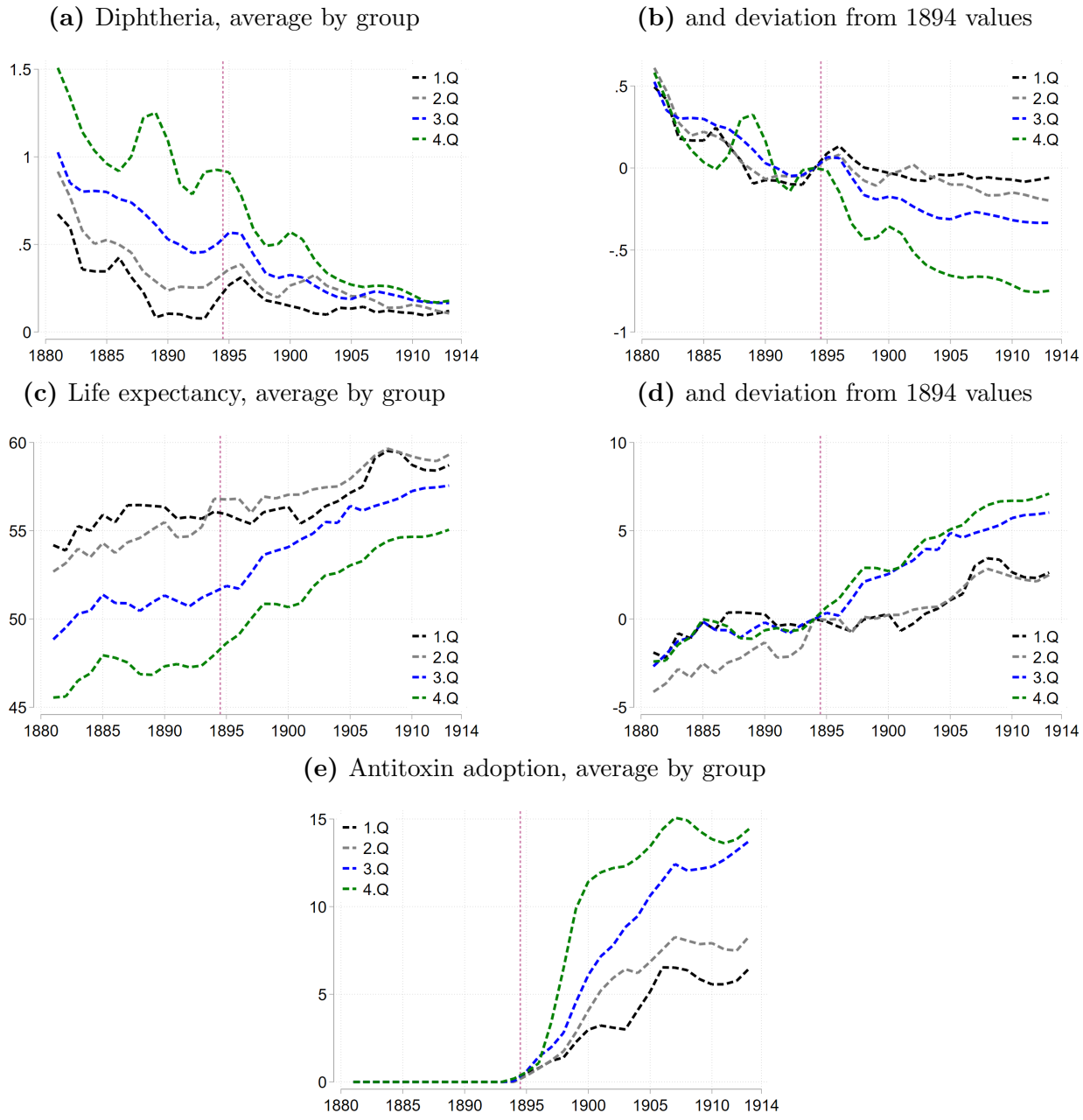
Before presenting the baseline estimates of the antitoxin's impact on health outcomes, we first provide formal evidence supporting the key identifying assumption of parallel trends. This is done by estimating reduced-form event studies and conducting direct falsification tests on our instrument.

First, Figure 4 shows the results from reduced-form event studies for the two main outcomes, as specified in equation (3). Both for diphtheria mortality (Panel a) and life expectancy at age 1 (Panel b), municipalities with different treatment intensities displayed similar trends before the policy. Following its implementation, municipalities with higher treatment intensities experienced relatively greater declines in diphtheria mortality and increases in life expectancy compared to those with lower treatment intensities. The estimated coefficients for the event year 1913-14 indicate that a one standard deviation increase in treatment reduces diphtheria mortality by 0.37 deaths per 1,000 people (-0.83 *0.44) and raises life expectancy by 1.68 years (3.82*0.44).

Overall, we find similar patterns when estimating annual event studies, but the post-antitoxin coefficients are estimated with less precision when using only a single baseline

[22] A similar pattern emerges if we first aggregate municipalities into these four groups then calculate life tables for each group. This approach addresses the challenges of constructing life tables for small populations (Appendix Figure A.3).

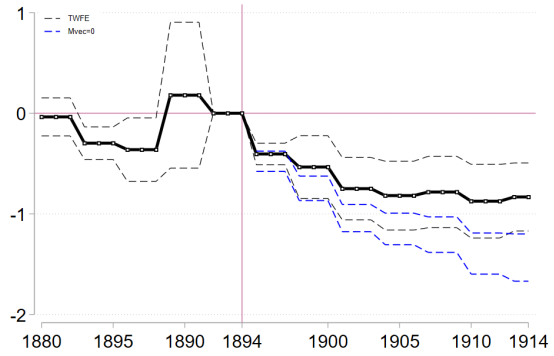
Figure 3: Descriptive event studies



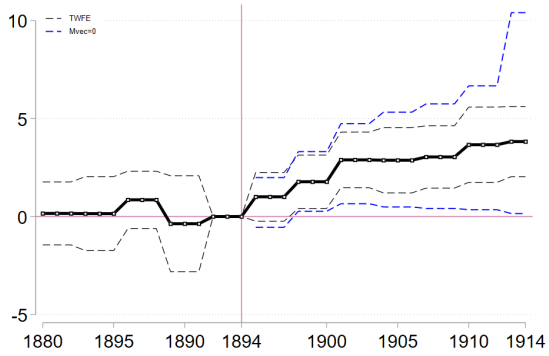
Notes: *The figure shows the development of the average diphtheria mortality rate per 1,000 people (Panel a and b) and life expectancy at age 1 (Panel c and d) and antitoxin bottles per 1,000 people (Panel e) by quartiles of treatment intensity. Panel a/c/e shows this development by group and Panel B/D in addition subtracts the 1894 moving average 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 (1888-94) diphtheria mortality rate. The vertical line indicates the first year when antitoxin became freely available for adoption.*

Figure 4: Reduced-form event-study estimates

(a) Diphtheria



(b) Life expectancy



Notes: *This figure shows reduced-form event study estimates for the diphtheria mortality rate, in Panel (a), and life expectancy at age 1 in Panel (b). We use a three-year pooling model, except for 1913 and 1914, which is two years. The omitted three years are 1892-1894. The regressions control for municipality and year fixed effects and are weighted by the 1895 municipality population size. The dashed lines are 95 percent confidence bands based on standard errors clustered at the municipality level. The blue dashed lines report confidence bands, allowing for linear smoothness (i.e., linear pre-trends) according to Rambachan and Roth (2023).*

comparison year (1894). Precision improves when the reference period is expanded to two or three years (Appendix Figure A.4). The results remain the same when using a spline function instead of pooling event-years (Appendix Figure A.5), when using life expectancy from the abridged life table, or when splitting the analysis by gender (Appendix Figure A.6).[23] Furthermore, we do not observe systematic pre-antitoxin trends for most other outcomes, including the diphtheria death ratio, infant mortality, child mortality rate, the crude death rate, accidents, birth rates, among others (Appendix Figures A.7 and A.8).

Second, we implement a more direct test to determine whether the instrument captures pre-antitoxin trends in the two outcomes. Specifically, we conduct a placebo test in which the policy would have been implemented in the early 1880s. If municipalities with substantial antitoxin uptake after 1895 were already on a different trajectory of health transition during the 1880s, this falsification exercise would reveal significant effects. Reassuringly, and in line with the previous evidence, this is not the case. Across all specifications using incorrect too-early start dates (assuming antitoxin distribution began in 1880 through 1885) and restricting the analysis to the pre-antitoxin period, we obtain small and statistically insignificant 2SLS

[23]Note, we do not observe a similar spike in the Rambachan and Roth (2023) upper confidence band as reported in Panel (b) of Figure 4 in the additional event studies for life expectancy.

estimates for both diphtheria mortality and life expectancy at age 1 (Appendix Figure A.9).[24]

We now turn to the baseline estimates presented in Table 2. Our analysis begins by estimating equation (1) using OLS (Panel a). The two primary outcomes are the diphtheria mortality rate (column 1) and life expectancy at age 1 (columns 2-4). For diphtheria mortality, the point estimates are statistically insignificant and close to zero. For life expectancy, the point estimates are even negative, with varying levels of statistical significance. Overall, the OLS estimates provide no evidence that the policy-driven antitoxin treatment improved population health and, in some cases, suggest the possibility of adverse effects.

A very different result is shown by using 2SLS as the estimation method (Panel b). For both diphtheria mortality and life expectancy, the β_{2SLS} estimates have the "expected" signs and are statistically significant at the 5-percent level with a strong first-stage relationship.[25] Specifically, the first-stage estimate is positive, highly statistically significant, and robust to the inclusion of controls examined in the subsequent robustness analysis (Appendix Table A.2).[26] The final three rows of Table 2 report that the Kleibergen-Paap F-statistic is well above 10, and confidence intervals are presented using the methods of Anderson and Rubin (1949) and Lee et al. (2022). Relative to the pre-antitoxin levels, the estimate in column 1 suggests that one additional bottle per 1,000 people reduced the diphtheria mortality rate by 11 percent. Since both diphtheria mortality and antitoxin bottles are scaled per 1,000 people, the estimates imply that it took approximately 10 bottles (1/0.099) to avert one death from diphtheria. Data from the SBH (1901-1904) indicate that, on average, each patient received about 5 bottles of antitoxin, suggesting that every second patient treated with antitoxin had their life saved.[27] This aligns with historical clinical evidence, which indicates that antitoxin halved the fatality rate (SBH, 1896), and our own case fatality data for Massachusetts (Appendix Figure A.11). 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 percent. The corresponding reduced-form estimates are reported in Panel (c) of Table 2.

Since gender is recorded on death certificates, we calculate life expectancy separately

[24]The sample period includes 1880 to 1896. Similar results are observed when restricting the sample to the pre-antitoxin period (up to 1894), though the first stage becomes stronger when additional years for antitoxin diffusion are included.

[25]The linear functional form is supported when plotting the relationship between the outcomes and antitoxin instrumented (Appendix Figure A.10)

[26]Using a decomposition exercise following Gelbach (2016), we find that population density and dwelling size (all interacted with a linear trend) are the key factors driving the decrease in the first-stage coefficient when robustness controls are added (Appendix Table A.3)

[27]According to the SBH (1902), the median number was ca. 2 bottles as some patients received many bottles. Using this number instead suggests that every fifth treated patient was saved.

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.	-0.006	-0.054**	-0.073***	-0.042
	(0.008)	(0.024)	(0.023)	(0.027)
Panel b: 2SLS estimates				
antitoxin p.c.	-0.099***	0.505***	0.473***	0.529***
	(0.018)	(0.161)	(0.164)	(0.168)
Panel c: Reduced-form estimates				
treat x I x yr	-0.044***	0.176***	0.165***	0.184***
	(0.005)	(0.045)	(0.047)	(0.046)
Mean pre-y	0.871	49.35	49.65	48.90
$N \times T$	9870	9800	9799	9799
N	282	280	280	280
KP-F-Stat	36.18	36.03	36.01	36.03
AR 95 CI	[-0.15:-0.07]	[0.23:0.89]	[0.19:0.86]	[0.25:0.94]
tf 95 CI	[-0.14:-0.06]	[0.15:0.86]	[0.11:0.83]	[0.16:0.90]

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 age 1 (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. KP-F-Stat is Kleibergen-Paap F statistic, AR 95 CI is the Anderson-Rubin 95% confidence intervals (Anderson and Rubin 1949) and tf 95% confidence intervals (Lee et al. 2022).*

for men and women. In columns 3-4 of Table 2, we investigate if antitoxin treatment had gender-specific effects. Although the point estimate is larger for men, the difference is not substantial enough to draw definitive conclusions about potential gender differences in the

effects of the antitoxin.

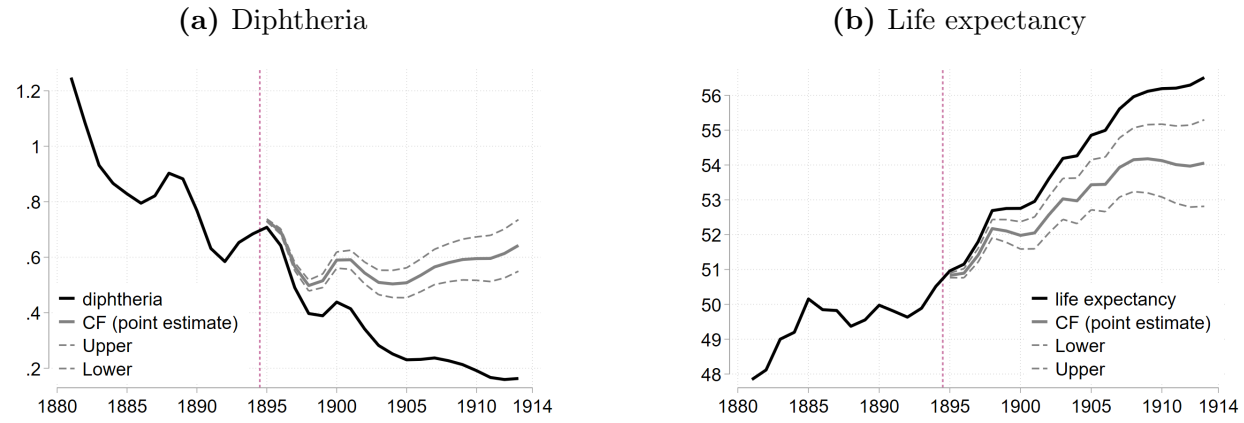
In Figure 5, we use the reduced-form estimates from Table 2 to conduct counterfactual experiments that simulate annual delays in the implementation of the policy. For instance, in 1895, we assume a one-year delay, while by the end of the sample window, the delay extends to 20 years (i.e., no free distribution policy during the sample period). The counterfactual outcome for a given year is calculated as the observed outcome minus the predicted change. The counterfactual, reported in Panel (a), reveals that had the policy not been implemented by the end of the sample period, diphtheria would have caused 0.48 more deaths per 1,000 people ($0.64 - 0.16$). This corresponds to saving around 1,800 lives, using the population size of Massachusetts in 1914. Panel (b) shows that life expectancy at age 1 would have been almost two years lower in this case. The exact counterfactual dynamics are shaped by the linear trend-break assumption. Had we instead, for example, assumed a concave year trend-break (i.e., logged year since policy), the number of deaths averted and life years saved would increase earlier in the post-antitoxin period but we would obtain relatively similar conclusions by the end of the sample (Appendix Figure A.12).

Next, we use the diphtheria counterfactual to assess whether the estimated impact on life expectancy is plausible. Specifically, we examine whether the decline in diphtheria deaths can explain the antitoxin-driven rise in life expectancy, as documented in Panel (b) of Figure 5. To do so, we calculate the number of diphtheria deaths averted and incorporate these figures into a life table for the entire state. We assume that the averted deaths are evenly distributed across the age range of 1-5 years. This analysis suggests that the counterfactual reduction in diphtheria deaths could account for an increase in life expectancy of 1.4 years. This estimate is about 0.6 years smaller than the direct effect of antitoxin on life expectancy but when considering statistical uncertainty, we cannot rule out the possibility that the entire direct estimated effect on life expectancy can be driven by reductions in diphtheria mortality (Appendix Figure A.13). Finally, the SBH (1901) estimated the number of deaths averted by antitoxin by comparing aggregate fatality rates before and after the policy's implementation up to 1901. We incorporated these historical estimates into the life table for Massachusets. According to this evaluation, antitoxin increased life expectancy by approximately two years, which is an even more optimistic view compared to our estimates (Appendix Figure A.13).

5.3 Robustness

In this subsection, we present robustness checks for our baseline model. First, our findings remain robust when controlling for all pre-antitoxin variables (reported in Table 1) interacted with year fixed effects. Additionally, we account for time-varying population shares, the roll-

Figure 5: Counterfactual when delaying the policy



Notes: *This figure uses the baseline reduced-form estimates (and 95 percent confidence bands) to calculate the counterfactual (CF) development for the diphtheria mortality rate (Panel a) and life expectancy at age 1 (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.*

out of public waterworks, the establishment of new general hospitals, and infectious disease hospitals. Importantly, these adjustments do not alter the core insights of our analysis (rows 2-5 in Appendix Figure A.14).[28]

Second, we test the robustness of our results using alternative specifications. These include transforming the treatment variable to its logarithmic form, using log years since the policy, omitting weights, excluding Boston, incorporating multiple lags of the dependent variable to account for potential persistence in the outcome, replacing county-year fixed effects with year fixed effects, controlling for initial population size quartiles interacted with year fixed effects, and including never listed antitoxin bottle's municipalities as zeros instead of dropping them as we do in the baseline specification. None of these adjustments change the overall findings (rows 6-13 in Appendix Figure A.14).

Third, we show that our results are robust to using an alternative estimation approach using a shift-share IV design, in which we allocate the aggregate number of bottles based on local pre-antitoxin mortality shares. However, such an IV would in our setup rely on a single shifter and a single share, and identification would be based on the exogeneity of the shares as outlined by Goldsmith-Pinkham et al. (2020). In our continuous DiD strategy, we inherently conceptualize identification within an exposure design framework as suggested by Goldsmith-Pinkham et al. (2020), which draws on the same sources of variation (Appendix

[28]For both outcomes, the part of the main effects that can be explained by the co-variates is not significant according to the Gelbach (2016) decomposition exercise (Appendix Table A.4).

Section A.6). Nonetheless, when we apply the shift-share IV design, the resulting 2SLS estimates are nearly identical to our baseline results (row 14 in Appendix Figure A.14).

Moreover, we present several robustness checks that are specific to life expectancy. First, we obtain similar estimates when life expectancy at age 1 is derived from an abridged life table. Unlike single-year age intervals, the abridged life table uses five-year age groups, except for the first year of life. This approach also allows us to incorporate population counts from Haines (2022). Second, we obtain similar estimates using life expectancy at birth, which is expected since this measure also captures lives saved beyond the first year of life. Third, our mortality data—whether from vital statistics or death certificates—are based on the place of occurrence, which can introduce bias, for example, in areas with hospitals that may appear as mortality hotspots. This is a common issue in historical U.S. mortality studies. However, for death certificates up to 1905, we retrieved place-of-residence information and recalculated life expectancy at age 1. This measure yields the same insights (rows 15-17 in Panel b of Appendix Figure A.14.

Finally, our baseline specification clusters standard errors at the municipality level. One could also cluster at the county level, however, with only 14 counties, it is necessary to account for the small number of clusters. To address this, we use wild-cluster bootstrapping. This adjustment does not affect our baseline insights (Appendix Figure A.15).

5.4 Other vital 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 (infant and child) population health (e.g., Alsan and Goldin 2019). 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 (Appendix Section A.1).

For the infant mortality rate (columns 1 to 3), the average effect of antitoxin is only statistically significant for females and all three estimates are relatively small in magnitude when compared to the pre-antitoxin mean in the outcomes. In contrast, for the child mortality rate (columns 4 to 6), the point estimate is negative, highly statistically significant, and similar in magnitude for both genders. The percentage decline associated with one additional bottle of antitoxin per 1,000 people, relative to the pre-antitoxin mean, is close to 5 percent. Using single-year age-specific mortality rates from age 0 to 10, we further document that the effects are concentrated at ages 2-9 (see Appendix Table A.5). These findings highlight that the availability of antitoxin free of charge played a role in improving child health in

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.	-2.849	-3.332*	-2.219	-1.177***	-1.243***	-1.166***
	(1.842)	(1.707)	(2.106)	(0.371)	(0.382)	(0.432)
Mean pre-y	153.2	140.7	166.4	23.21	22.58	24.04
$N \times T$	9870	9870	9870	9870	9870	9870
N	282	282	282	282	282	282
KP-F-Stat	36.14	36.14	36.14	36.14	36.14	36.14
AR 95 CI	[-6.90:0.49]	[-7.21:-0.24]	[-6.69:1.76]	[-2.08:-0.56]	[-2.17:-0.61]	[-2.21:-0.45]
tf 95 CI	[-6.88:1.18]	[-7.06:0.4]	[-6.82:2.39]	[-1.99:-0.37]	[-2.08:-0.41]	[-2.11:-0.22]

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 births 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) are clustered at the municipality level. ***, **, and * indicate significance at the 1, 5, and 10 percent level. KP-F-Stat is Kleibergen-Paap F statistic, AR 95 CI is the Anderson-Rubin 95% confidence intervals (Anderson and Rubin 1949) and tf 95% confidence intervals (Lee et al. 2022).*

Massachusetts during the early 20th century.

Next, we examine how antitoxin influenced other mortality and vital outcomes, as summarized in Table 4. First, we show that antitoxin reduced the mortality burden of diphtheria by lowering the diphtheria mortality ratio (i.e., diphtheria deaths per 1,000 total deaths) in column 1. Second, we find negative but statistically insignificant effects on the case-fatality rate (diphtheria deaths per 1,000 cases). This result is not entirely unexpected, as antitoxin would theoretically reduce fatality rates across all municipalities. This pattern is also reflected in the aggregate trends for the case-fatality rate (Appendix Figure A.11). However, since the disease was more prevalent in some areas, these aggregate reductions would imply that communities with higher prevalence experienced larger reductions in diphtheria mortality rates, making this finding consistent with the negative effects on the diphtheria mortality rate. In addition, column 3 shows that antitoxin reduced the prevalence rate (i.e., diphtheria per 1,000 people), which is consistent with that antitoxin was also used for short-run immunization purposes.[29] Third, we observe second-order effects on mortality

[29]The case data, available only from 1891, is relatively unbalanced and is often measured with error. Thus, our results based on case data should be interpreted with caution.

from apoplexy, a proxy for heart- and stroke related conditions, and other infectious diseases (columns 4 and 5). These effects are relatively small in magnitude compared to pre-antitoxin means, supporting their classification as second-order effects. Declines in diphtheria mortality potentially reducing mortality from other diseases is not surprising, given patterns of co-mortality. Fourth, we find no effects on deaths from accidents, which is reassuring, as it is unlikely that diphtheria would influence accidental deaths (column 6). Finally, we observe small and statistically insignificant negative effects on the crude birth rate (column 7).

Table 4: Other vital outcomes

	(1) diph ratio	(2) case-fatality	(3) prevalence	(4) apoplexy	(5) infec rate	(6) accidents	(7) birth rate
antitoxin p.c.	-4.103***	-10.316	-0.116**	-0.048***	-0.152***	0.032	-0.446
	(0.880)	(7.424)	(0.056)	(0.011)	(0.047)	(0.040)	(0.296)
AR 95 CI	[-6.45:-2.71]	[-29.78:1.81]	[-0.25:-0.02]	[-0.07:-0.03]	[-0.26:-0.07]	[-0.05:0.12]	[-1.10:0.09]
tf 95 CI	[-6.03:-2.18]	[-26.30:5.66]	[-0.24:0.00]	[-0.07:-0.02]	[-0.26:-0.05]	[-0.06:0.12]	[-1.09:0.2]
Mean pre-y	42.97	402.6	1.794	0.531	7.564	0.720	26.46
$N \times T$	9861	3677	4851	9870	9870	9870	9870
N	282	268	279	282	282	282	282
KP-F-Stat	36.12	24.67	31.20	36.14	36.14	36.14	36.14

Notes: *This table reports effects on the diphtheria death ratio (col. 1), the case-fatality rate (col. 2), the prevalence rate (col. 3), other causes of deaths (col. 4-6), and the crude birth rate (col. 7) using the baseline annual linear trend-break model as outlined in equation (2). The sample includes the years 1880 to 1914, except for the fatality and prevalence rates (from 1891 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 are clustered at the municipality level. ***, **, and * indicate significance at the 1, 5, and 10 percent level. KP-F-Stat is Kleibergen-Paap F statistic, AR 95 CI is the Anderson-Rubin 95% confidence intervals (Anderson and Rubin 1949) and tf 95% confidence intervals (Lee et al. 2022).*

In addition, we conducted a complementary analysis to examine the effects on individual mortality rates. Specifically, we "stacked" the cause-of-death mortality, creating a panel dataset where the dimensions are municipality-year-disease. This approach allows us to include municipality-by-year, disease-by-year-by-county, and municipality-by-disease fixed effects. These additional fixed effects further reduce concerns that omitted variables might drive our estimates. We implement this extended set of controls by interacting the main right-hand-side variables in equations (1) and (2) with an indicator for diphtheria, based on our hypothesis that antitoxin treatment should have a direct effect on this disease. While the baseline model resembles a difference-in-differences framework, the stacked model aligns more closely with a triple-differences design. We test this approach using different sets of

control diseases (e.g., all diseases, only childhood diseases, etc.). Regardless of the control group used, we consistently find negative and significant effects in the mortality specification (Appendix Table A.6).

Does the observed "antitoxin effect" merely reflect the broader expansion of the healthcare sector during this period? Municipalities heavily affected by diphtheria may have requested additional doctors, meaning that the increase in antitoxin bottles per 1,000 people could simply indicate a greater availability of medical professionals in these areas. While our robustness analysis includes controls for pre-antitoxin doctors per capita interacted with year fixed effects, we address this issue more explicitly by examining the number of doctors, nurses, and pharmacists per 1,000 people at the municipality level. Using data from the full-count U.S. censuses, we treat these healthcare workforce measures as outcomes in our regression framework. We apply the baseline annual 2SLS specification, interpolating occupation rates between census years to fill gaps in the data. To account for potential lagged effects, we also analyze the data by age groups, as changes in the healthcare workforce may take time to materialize. Across all specifications, the effects are quantitatively small and statistically insignificant (Appendix Table A.7). Thus, while the healthcare sector undoubtedly played a role in the mortality transition by facilitating the diffusion of antitoxin (Appendix Table A.2), our findings suggest that the health benefits from antitoxin treatment were not simply a byproduct of affected municipalities expanding their healthcare workforce.

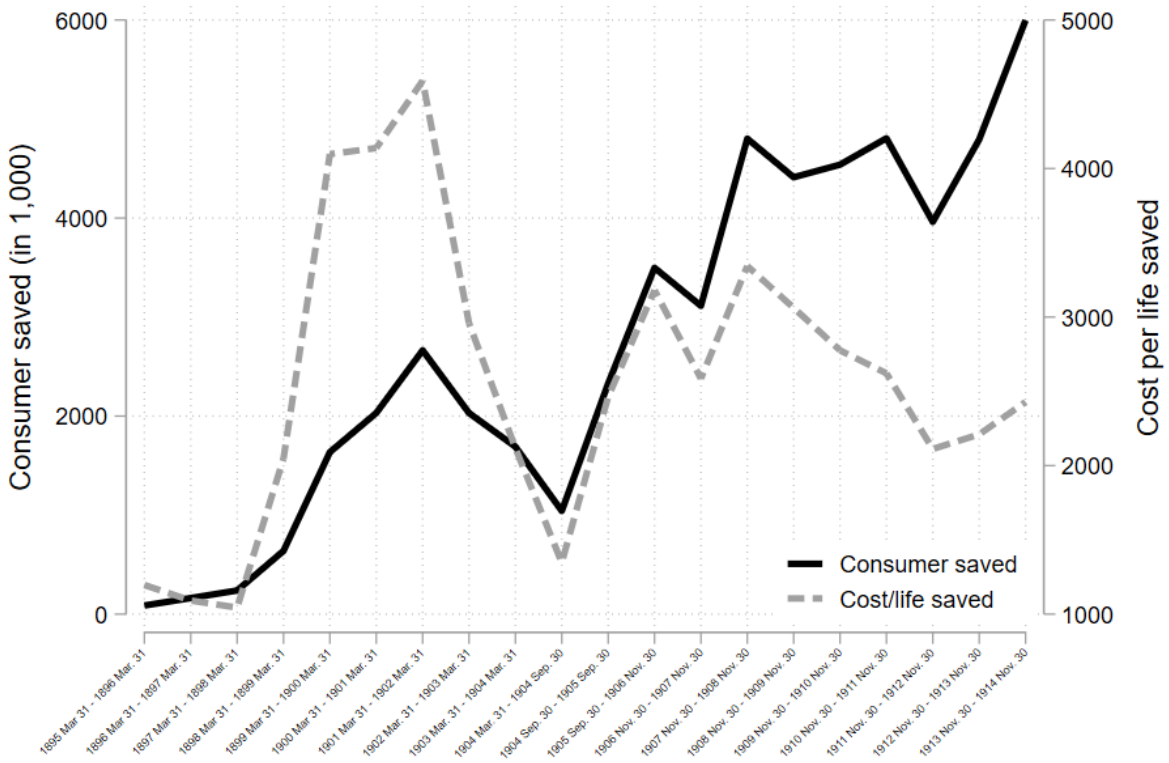
5.5 The economics of policy

In this subsection, we discuss the counterfactual scenario to determine whether the free-distribution policy primarily reduced consumer costs or genuinely saved lives. We also analyze the cost-effectiveness of the intervention and compare the policy to other historical public health strategies in terms of costs per life saved.

We begin by considering the appropriate counterfactual. Since consumers could purchase antitoxin on the private market, it is important to ask: Did the policy (in conjunction with the medical technology) save lives, or did it primarily reduce costs for consumers? The SBH (1902) evaluated the effects of antitoxin during the first six years of the policy, offering insights into this question. The report estimated production cost of \$0.15 per bottle, compared to the cheapest private alternative at \$1.50 per bottle. Clearly, the policy reduced costs for consumers. Multiplying this cost difference by the number of bottles supplied between 1895 and 1914 suggests total savings of nearly \$50 million in 2023 USD. The solid black curve in Figure 6 depicts the annual savings in 2023 dollars.[30]

[30]These numbers represent the savings experienced by consumers who received the antitoxin for free, compared to the increased state tax revenues required to fund its public production. For those

Figure 6: Savings and cost per life saved



Notes: *This figure shows the annual savings for consumers in Massachusetts, in 2023 USD, resulting from the free distribution policy compared to the private market alternative (black solid line, left y-axis). It also reports the cost per life saved in 2023 dollars (gray dashed curve, right y-axis). The x-axis represents the SBH bottle production periods: For instance, the year 1895 corresponds to the SBH production period from March 31, 1895, to March 31, 1896. Notice, the calendar year 1904 reflects only six months of production, from March 31, 1904, to September 30, 1904, while the calendar year 1905 spans 14 months, from September 30, 1905, to November 30, 1906. All other production periods cover 12 months. See Appendix Table A.8* for additional details.

But did the policy save lives? The same report suggests that it did. The SBH conducted a survey in 1901, asking those responsible for distributing the drug (doctors, druggists, local boards of health, etc.) whether patients would have chosen the private alternative. The general response was that a large proportion of the treated patients, in some instances up to 90-95 percent, would have not chosen the private alternative (Massachusetts State Board of Health 1902, p.489). The SBH concluded that a significant proportion of treated patients, many of them from immigrant and poor households, would have been unable to access antitoxin without the policy. Thus, according to this survey, the historical demand for

who actually used the antitoxin, the savings would have been even greater, whereas non-consumers bore only the burden of higher taxes.

antitoxin was highly elastic, and we argue that the relevant counterfactual is a scenario in which the free-distribution policy was not implemented and most lives would not have been saved, even though the treatment possibility existed. Our findings should therefore generally be interpreted within the context of the interaction between medical treatment and public health policy.

Next, we evaluate the cost-effectiveness of the policy. We apply the $1.50 per bottle production costs for the years 1895 to 1903, where 1902 and 1903 have been adjusted using the consumer price index. From 1904 onward, total expenditures for antitoxin production are available in the annual reports.[31] We use the baseline counterfactual, reported in Figure 5, to calculate the production costs per saved life for each year. Appendix Table A.8 shows that the cost per life saved varied from year to year ($137-$601 converted to 2023 USD). Importantly, even the maximum cost of $601 per life saved is much cheaper compared to any other studied historical public health intervention.

However, this quantification overlooks additional costs patients likely incurred for physician services or hospital care when receiving the treatment. To estimate these costs, we first approximate the number of patients treated with antitoxin by dividing the total number of bottles supplied by five, our best estimate for the average bottles per patient (SBH, 1902). Assuming each treatment cost $5 in 1901 dollars, we can calculate the total cost of saving lives with antitoxin treatment.[32] Figure 6 shows that the cost ranges from $1,050 to $4,600 in 2023 USD (averaging $2,570), which is comparable to the cost of averting a tuberculosis death through information provision during the first half of the 20th century in Denmark (Egedesø et al. 2020), for example.

Since diphtheria primarily affected children, while tuberculosis deaths were more concentrated among older individuals, it is more appropriate to compare the two interventions in terms of cost per life-year saved. Assuming each life saved occurred at age one, the implied cost per life year saved is approximately $50 in 2023 USD, based on a pre-period life expectancy at age one of ca. 50 years—making it more cost-effective than tuberculosis information provision, which was estimated at around $76-$155 in 2015 USD (Egedesø et al. 2020). On the other hand, if the antitoxin treatment cost in hospitals and at doctors was $10 instead of $5, the cost per life year saved would rise to around $93, bringing the two interventions closer in cost-effectiveness. However, this would still be cheaper than the cost of saving life-years with clean water, which is estimated to be around $500 in 2003 USD (Cutler and Miller 2005). Compared to Hollingsworth et al. (2024), who estimate the cost

[31]However, as these figures include vaccine costs, they overestimate antitoxin production costs.

[32]For example, the fees of vaccination ranged between $1-$5 USD in 1892 according to the March-July issue of The Chicago Medical Recorder (1892, p.419).

of saving a life through a hospital modernization program in North Carolina (1927-1942) at approximately $17,000 in 2017 USD, saving lives through the free distribution of antitoxin was significantly more cost-effective. This is not entirely surprising, as the antitoxin policy was more targeted and leveraged an already established health infrastructure.

6 Effects on School Attendance and Adult Outcomes

The free antitoxin distribution policy in Massachusetts reduced consumer costs and cost-effectively saved lives. We have shown that the availability of the antitoxin serum substantially reduced diphtheria mortality rates and increased life expectancy at the municipality level. In the final part of the empirical analysis, we leverage the richness of the complete count U.S. Census data to examine the consequences of exposure to antitoxin at the individual level. Because our results show that the antitoxin treatment for diphtheria was most effective for children below age 10, we focus on young people in this section. In particular, we examine whether the availability of antitoxin treatment had any short-term effects on school attendance and long-term consequences for exposed children in terms of educational attainment and adult labor market outcomes.

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 counted as attending), in the 1900 Census, 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 1900, pupils in Massachusetts were required to stay eight months in school, and the compulsory school age was between age seven and fourteen. Besides compulsory schooling laws, there were also child labor law regulations in place that children under fourteen should not be employed in a factory, workshop, or mercantile establishment.[33] Overall, Massachusetts had a strong school attendance mandate at the turn of the 20th century. This is also reflected in the report of the U.S. Bureau of Education (1891, p.486), which mentions that in Massachusetts *"the compulsory law works well and is generally obeyed [and] the employment law is quite thoroughly enforced"*. Historians of education concluded that truancy was already a minor problem in Massachusetts in the 1890s (Lazerson 1971).

[33]Massachusetts introduced compulsory schooling in 1852, and since then the compulsory attendance law had been modified several times. Since 1894, pupils were required to stay eight months in school and in 1898 the compulsory age limits were further revised. At the time of our school attendance analysis in 1900, the average length of schooling was nine months (Lingwall 2014; Massachusetts State Board of Education 1901).

In fact, one of the main reasons for preventing a child from attending school was diphtheria (and other contiguous diseases such as smallpox and scarlet fever). The 1898 school law in Massachusetts required two weeks to have elapsed since recovery from diphtheria before return to school is permissible (Massachusetts State Board of Education 1899, p.579). Hence, diphtheria was a well-known factor that contributed to school absenteeism at that time, and we use the detailed information from the Census in 1900 to test whether exposure to antitoxin increased the likelihood of children regularly attending school.

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:

$$y_{ibm} = \beta Exposure_{bm} + \mu_b + \mu_m + \Gamma X_{im} + \epsilon_{ibm}, \tag{4}$$

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) at least for eight 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] 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.

[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 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.

Table 5: 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 ≧ 8 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	522,287	431,553	361,632	301,980	361,632	301,980
R-squared	0.143	0.164	0.029	0.040	0.042	0.053
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 (Y)	0.652	0.659	0.108	0.108	0.776	0.777

Notes: *This table reports how antitoxin exposure affected school attendance in 1900. The dependent variable is a dummy of whether a child between ages 5-15 attended school at all (columns 1-2); for no more than three months (columns 3-4); and for at least eight 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. All regressions include fixed effects for municipality and year of birth. Columns 2, 4, and 6 include a set of individual controls. 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 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 eight 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.9).[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: for a child living in a municipality with average antitoxin exposure (1.8 bottles per 1,000 people), we find that antitoxin exposure reduced

[35]Note, results remain qualitatively unchanged if we restrict the sample to include only children aged 5-15 who were born in Massachusetts or instrument the actual antitoxin exposure with the treatment intensity based on our 2SLS approach in equation (4).

the likelihood of attending school for three or fewer months by 8.3 percent and increased the likelihood of attending school for at least eight months by almost 1.4 percent relative to the sample mean.

Since exposure to the antitoxin treatment increased the time children spent in school, we can test whether this had any implications for educational attainment and labor market outcomes of 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 Table 5 using the linked sample and obtain very similar results (Appendix Table A.10).

Using the linked sample, we can test whether exposure to the antitoxin treatment during childhood had any detectable long-run effects. Table 6 reports the estimates for educational attainment (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-collar occupation (column 4), the occupational income score (column 5), and wages (column 6).[36] The estimating equation is (4) and the estimation method is least squares.[37] Although school absenteeism rates for exposed children fell considerably in 1900, we only find a quantitatively negligible positive effect (statistically significant at the 10-percent level) on educational attainment (column 1). This result is driven by men (Appendix Table A.11) and those who moved out of Massachusets (Appendix Table A.12). Overall, being exposed as a child in a municipality with average antitoxin exposure (2.4 bottles per 1,000 people) increased educational attainment by 0.2 percent or seven days—a negligible effect.

A similar picture emerges when considering the adult labor market outcomes of exposed children in columns (2)-(6). Regarding occupational choices (columns 2-4), the estimated coefficient on antitoxin exposure is always statistically insignificant and very small in size (precise null effects). As for the occupational income score and wages (columns 5-6), the estimated coefficient on antitoxin exposure is positive and statistically significant (at least at the 10-percent level) and is again driven by men and those who moved out of their place of residence in 1900 (Appendix Tables A.11 and A.12); but quantitatively the estimates are very close to zero.[38] Moreover, we also find no clear pattern that exposed children worked as

[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 instrumenting the actual antitoxin exposure with the treatment intensity based on our 2SLS approach in equation (4).

[38]Appendix Table A.14 shows that the quantitatively small positive effect on wages does not

Table 6: The Long-run Effects of Antitoxin Treatment

	(1) years in school	(2) low-skill occ	(3) blue-collar occ	(4) white-collar occ	(5) ln(occscore)	(6) ln(wages)
Exposure	0.008*	0.0001	-0.0006	0.0000	0.0011**	0.0018*
	(0.005)	(0.0004)	(0.0005)	(0.0005)	(0.0005)	(0.0010)
Observations	237,775	246,859	246,859	246,859	165,375	134,820
R-squared	0.172	0.087	0.102	0.096	0.101	0.115
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
Mean (Y)	10.09	0.226	0.123	0.308	3.276	7.135

Notes: *This table reports how antitoxin exposure during childhood affected labor market outcomes as adults. The sample includes 5 to 15-year-old children who lived in Massachusetts in 1900 linked to 1940 using the crosswalks from the Census Tree Project. The following outcomes in 1940 are used as dependent variables: educational attainment (column 1), a dummy of 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. All regressions control for municipality and year of birth fixed effects and a set of individual controls (see page 37 for details). 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.*

adults in better occupations or had a higher occupational income score when looking at these outcomes at the ages of 25-35 (linked sample 1900 to 1920) or at the ages of 35-45 (linked sample 1900-1930).[39] The results are reported in Appendix Table A.13. We also obtain qualitatively similar results when using the Census Linking Project (CLP) crosswalks.[40]

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. Our near-zero long-term results could imply, that conditional on surviving into adulthood, the antitoxin treatment made it possible for children who grew up in a high diphtheria mortality environment to end

depend on the functional form and that children exposed to antitoxin were also not more likely to report a business income (in the 1940 Census enumerators only asked whether a person (age 14+) had a non-wage/salary income over $50.)

[39]There is no information on individual wages in the historical Censuses before 1940.

[40]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.

up in similar occupations as adults as children who grew up in a low diphtheria mortality environment—a catching-up effect of being less sick. On the other hand, the antitoxin treatment also allowed "weaker" children to survive and those could perform worse in the long run and pull down educational attainment and labor market outcomes—a negative effect of having more "weaker" children reach adulthood. Apart from the fact that exposed individuals are not more likely to work in low-skilled/low-paid occupations, we show in Appendix Table A.14 that exposure to antitoxin treatment did not result in i) a lower likelihood of working at all, ii) being more likely employed on public relief (New Deal) projects, iii) being more likely unemployed, or iv) having a higher likelihood of being unable to work as adults. This suggests that our near-zero long-term results are unlikely to be driven by a compositional change in the population that reaches adulthood. One alternative interpretation of the results is that the lower school absenteeism rates due to the availability of the antitoxin treatment might not be large enough to generate significant increases in educational attainment and better adult labor market outcomes.[41]

7 Concluding remarks

This paper contributes to the debate on whether medical advances played a 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 Board of Health implemented a policy that provided municipalities with diphtheria antitoxin free of charge for medical use. We found that providing the antitoxin serum free of charge contributed to a significant reduction in the diphtheria mortality rate and increased life expectancy. Our baseline estimate suggests that without the policy, the disease would have claimed an additional 1,800 lives, and life expectancy at age 1 would have been nearly two years lower in 1914. In comparison, Jayachandran et al. (2010) estimated that sulfa drugs increased life expectancy by 0.3-0.7 years over a 6-year period (1937 to 1943), while we find that antitoxin increased life expectancy by 2 years over a 20-year period, making the two interventions comparable to each other in terms of average annual effects.

[41]Based on the linked sample 1900-1940 for Table 6, we regressed educational attainment and wages on a dummy whether a child aged 5-15 in Massachusetts attended school regularly (8 months or more) in 1900 and fixed effects for birth year and municipality and find that regular attendance in 1900 significantly increased years of schooling (by 0.22 or two percent relative to the sample mean) and log wages in 1940 by 3.3 percent suggesting that regular school attendance in 1900 had some likely impact on completed education and adult labor market outcomes.

More generally, we showed that antitoxin mainly increased life expectancy by reducing child mortality rates, while much of the existing literature has focused on interventions primarily affecting infants, the factors behind declines in childhood mortality have received less attention (see, e.g., Karbownik and Wray 2025). Our study suggests that medical innovation in combination with an effective public health policy is one such factor. In fact, the historical evidence indicates that the demand for antitoxin was highly elastic, and without the free-distribution policy, most lives would not have been saved, even though the treatment was available on the private market. Thus, our findings should be understood in the context of the interaction between medical treatments and public health policies. Notably, the public health officials in Massachusetts were able to implement the policy because the serum was never patented in the U.S., highlighting the trade-off between patent protection in the medical market and the possibility of saving lives from a public health perspective. Overall, our research suggests that the combination of medical innovation and public health policy played a more significant role in increasing life expectancy in the early 20th century than previously acknowledged.

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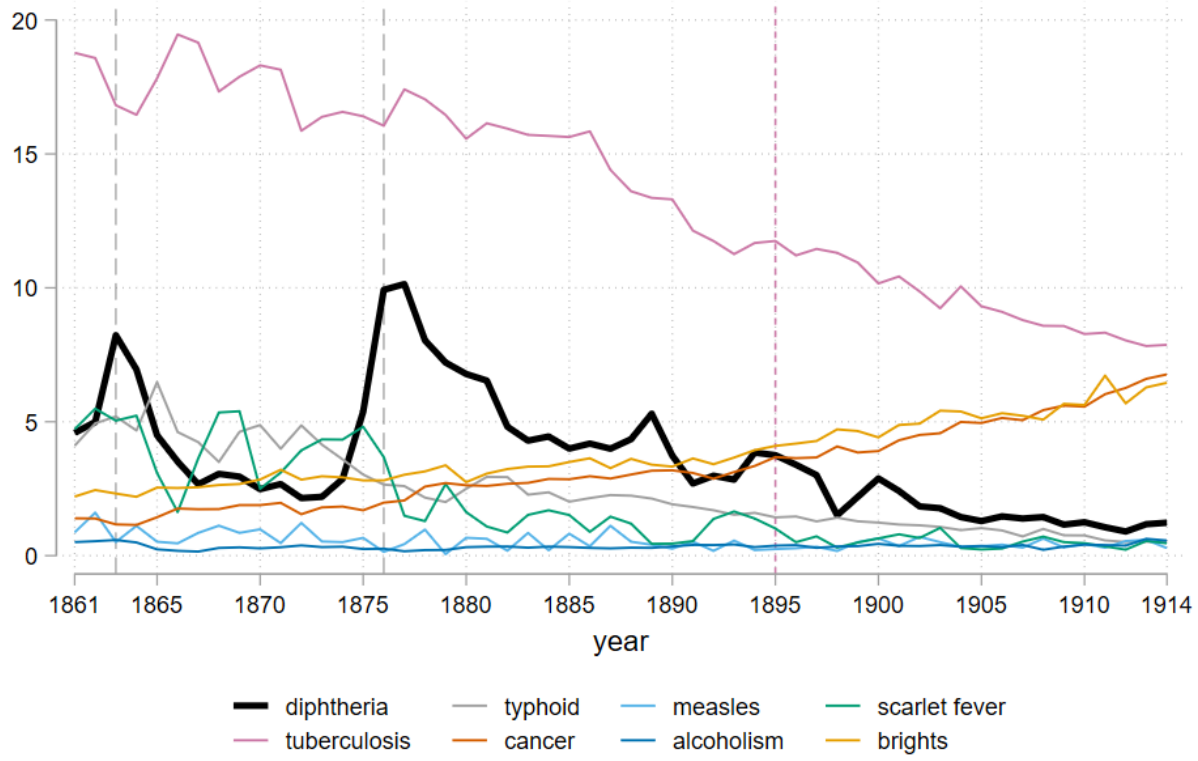
A Supplemental Online Appendix (not for publication)

Additional Figures

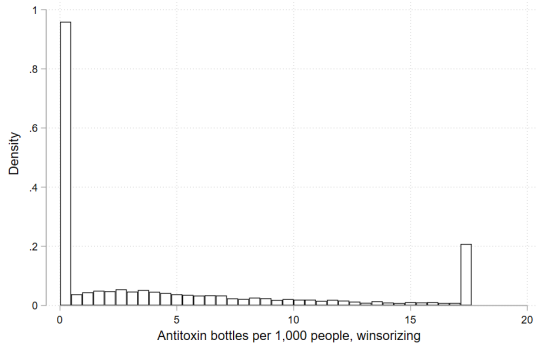
Figure A.1: Cause-specific death ratios



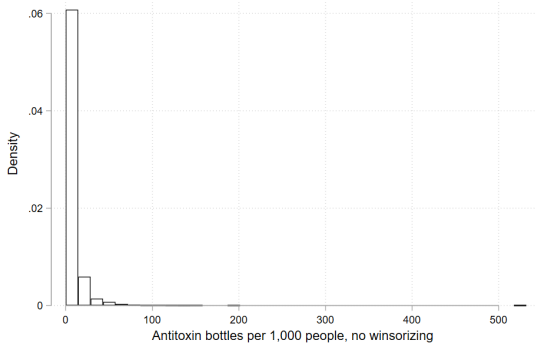
Notes: *This figure illustrates the trends in various major causes of death, as categorized in "the Annual Report on Births, Marriages, and Deaths in Massachusetts" (1914), shown as a percentage of the total number of deaths in the state. The bold black curve represents the percentage of deaths caused by diphtheria, which, for instance, approaches 10 percent in 1875.*

Figure A.2: Bottles distribution and winsorizing

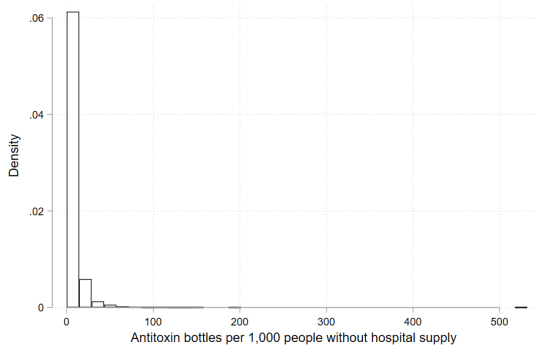
(a) Distribution when winsorizing



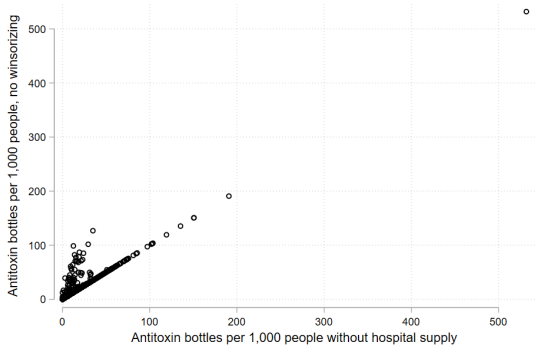
(b) Distribution without winsorizing



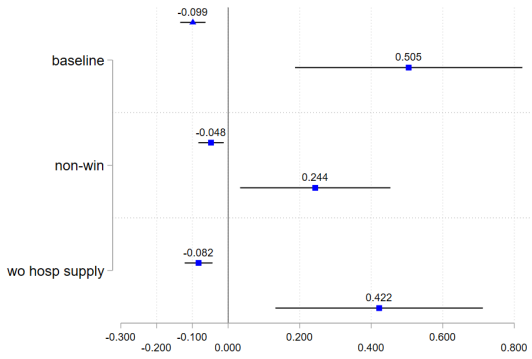
(c) No winsor and without hospital supply



(d) Relationship between (b) and (c)



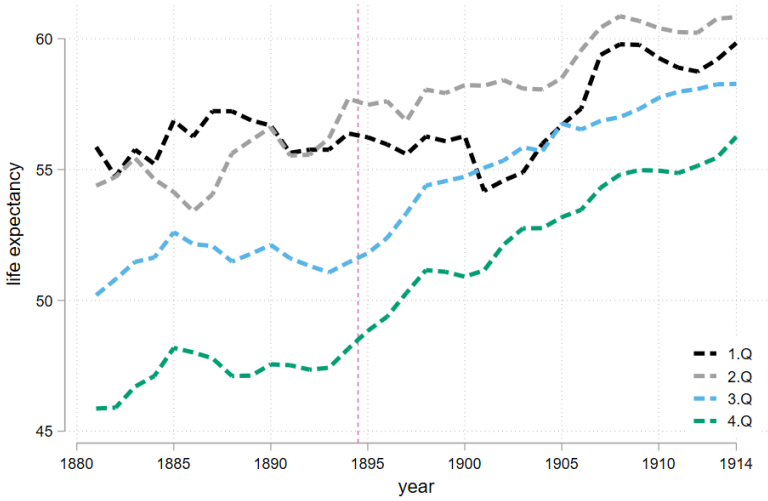
(e) Robustness of baseline estimates



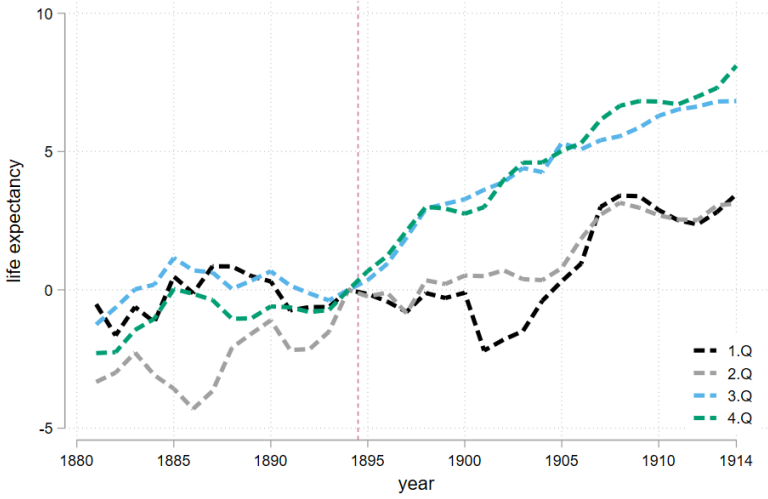
Notes: *This figure illustrates the distribution of antitoxin bottles per 1,000 people from 1895 onward. Panel (a) shows the distribution with winsorization applied (i.e., baseline), while Panel (b) presents the distribution without winsorization. Panel (c) displays the unwinsorized distribution, excluding antitoxin bottles supplied to hospitals. Panel (d) compares the distributions shown in Panels (b) and (c). Finally, Panel (e) reports the 2SLS estimates for diphtheria and life expectancy at age 1 using these three measures: the winsorized baseline, the unwinsorized, and the unwinsorized excluding hospital-supplied bottles, as the endogenous explanatory variables. The KP-F-Stats are for diphtheria 35.53 (baseline); 7.35; 30.25, respectively, and for life expectancy 33.69 (baseline); 6.35; 30.03, respectively.*

Figure A.3: Development of life expectancy at age 1 by treatment intensity

(a) Average by group



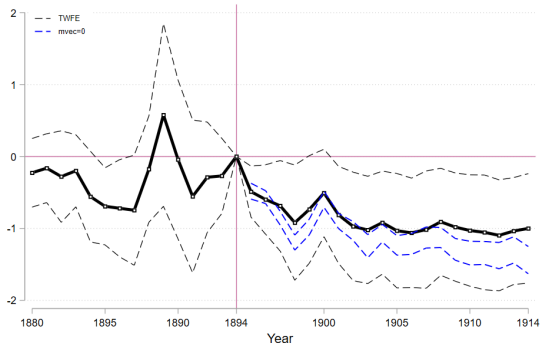
(b) And deviation from 1894 values



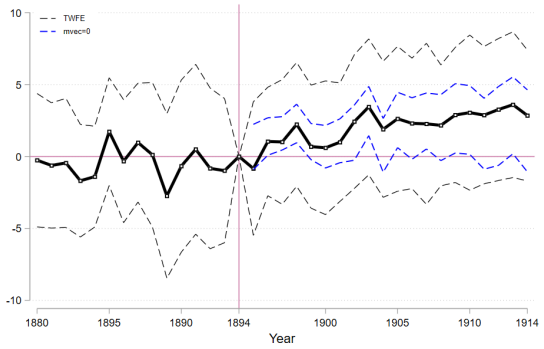
Notes: *This figure shows the development of life expectancy at age 1 by groups according to quartiles of treatment intensity. In particular, we have collapsed the baseline sample of municipalities into four regions (according to their level of treatment) and then for each region calculated life expectancy at age 1 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.4: Annual event-study estimates

(a) Diphtheria, 1894 omitted

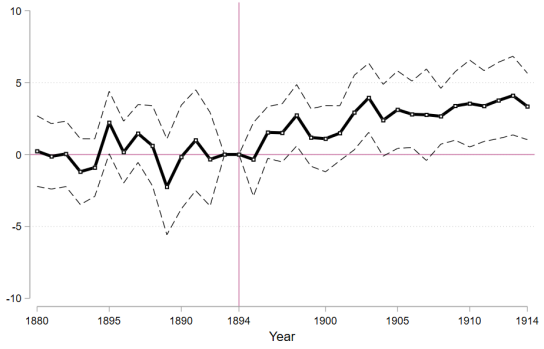


(b) Life expectancy at age 1, 1894 omitted

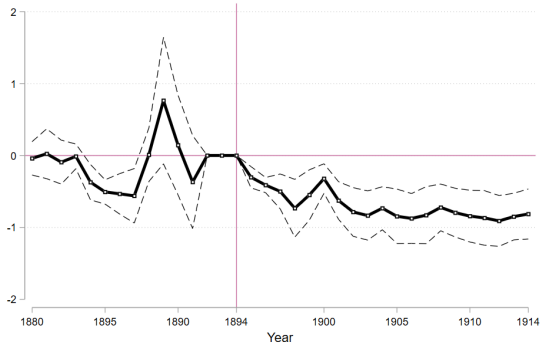


(c) Diphtheria, 1893 & 1894 omitted

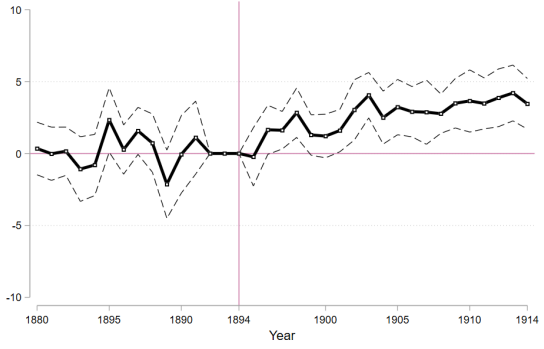
(d) Life expectancy at age 1, 1893 & 1894 omitted



(e) Diphtheria, 1892-1894 omitted



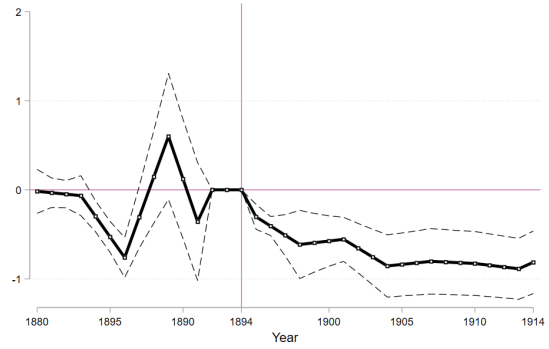
(f) Life expectancy at age 1, 1892-1894 omitted



Notes: *This figure shows reduced-form event study estimates for the diphtheria mortality rate, in Panels (a), (c) and (e), and life expectancy at age 1 in Panel (b), (d), (f). The event dummies are annual, contrast to the baseline, which uses a three-year pooling. The omitted years are 1894 (in Panels a and b), 1893 and 1894 (in Panels c and d), and 1892-1893 (in Panels e and f). All regressions control for municipality and year fixed effects and weighted by the 1895 municipality population size. The dashed curves are 95 percent confidence bands based standard errors clustered at the municipality level. The blue dashed curves (in Panels a snd b) are confidence bands, allowing for linear smoothness (i.e., linear pre-trends) according to Rambachan and Roth (2023).*

Figure A.5: Spline event-study estimates

(a) Diphtheria

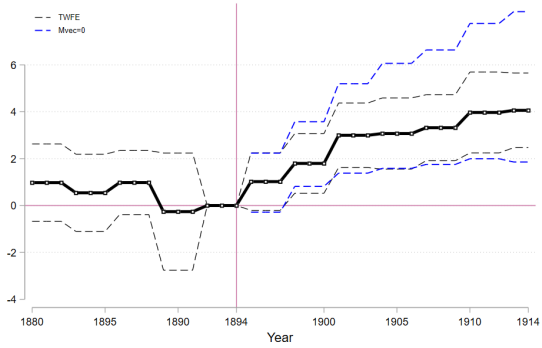


(b) Life expectancy

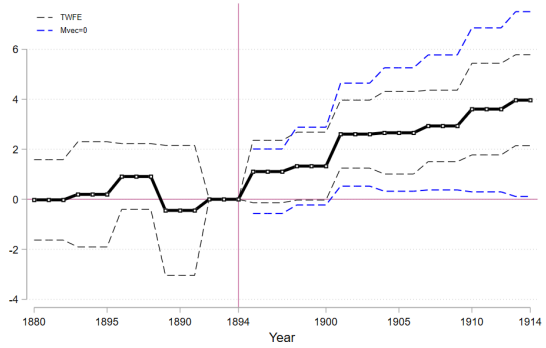
Notes: *This figure shows reduced-form event-study estimates for the diphtheria mortality rate, in Panel (a), and life expectancy at age 1 in Panel (b). We use a three-year spline model. The regressions control for municipality and year fixed effects and weighted by the 1895 municipality population size. The dashed lines are 95 percent confidence bands based standard errors clustered at the municipality level.*

Figure A.6: Event-study estimates, additional life expectancy outcomes

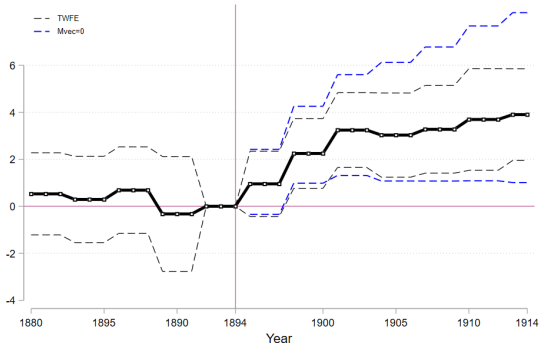
(a) Abridged life table



(b) Female



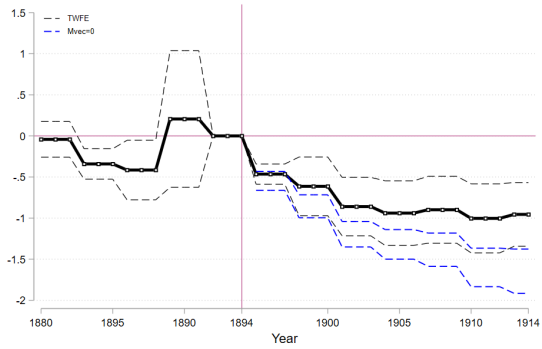
(c) Male



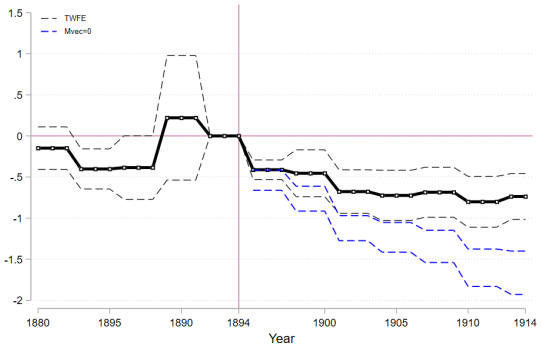
Notes: *This figure shows reduced-form event-study estimates for additional life expectancy outcomes. In Panel (a), the outcome is life expectancy at age 1 derived from the abridged life table; in Panel (b) the outcome is female life expectancy at age 1; in Panel (c) the outcome is male life expectancy at age 1. In all regressions, we use a three-year pooling model, except for 1913 and 1914, which is two years. The omitted three years are 1892-1894. The regressions control for municipality and year fixed effects and weighted by the 1895 municipality population size. The dashed curves are 95 percent confidence bands based standard errors clustered at the municipality level. The blue dashed curves are confidence bands, allowing for linear smoothness (i.e., linear pre-trends) according to Rambachan and Roth (2023).*

Figure A.7: Event-study estimates (relative to pre-antitoxin outcome), additional mortality outcomes I

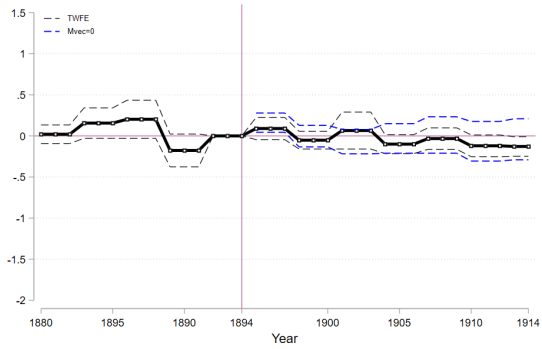
(a) Diphtheria mortality rate



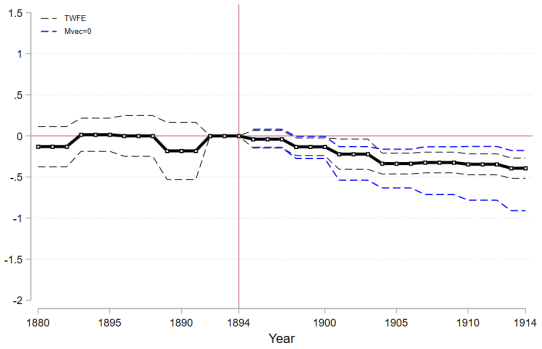
(b) Diphtheria death ratio



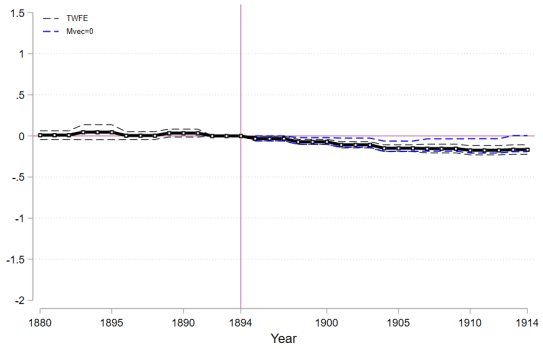
(c) Infant mortality rate



(d) Child mortality rate

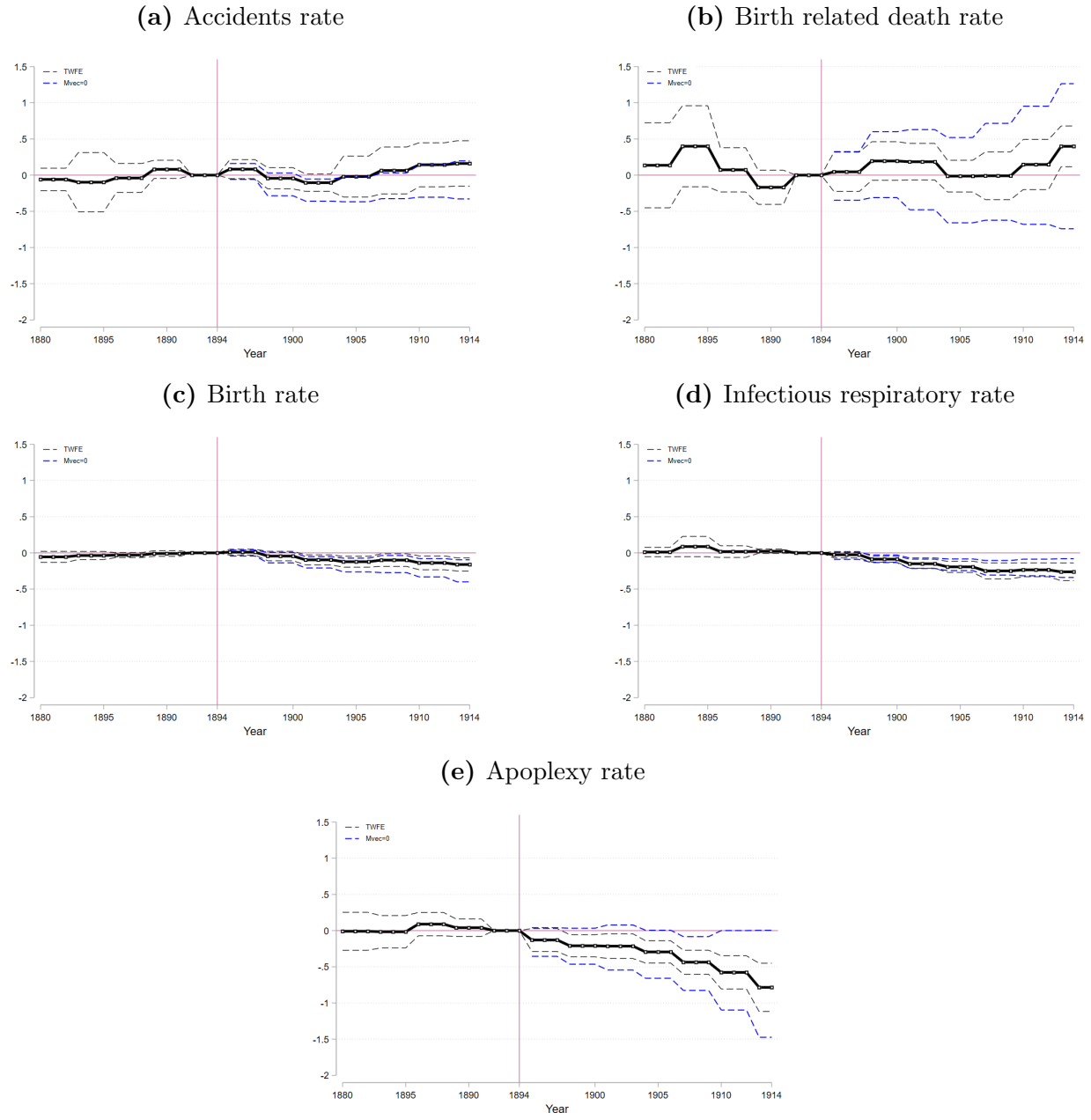


(e) Crude death rate



Notes: *This figure shows reduced-form event-study estimates for additional mortality outcomes. All estimates have been rescaled by the size of the pre-antitoxin outcome (i.e., the average from 1880 to 1894). Thus, one can interpret the estimates as percent changes. For comparison, Panel (a) shows the percent reductions in the diphtheria mortality rate. The different outcomes are described in the sub-figure title. In all regressions, we use a three-year pooling model, except for 1913 and 1914, which is two years. The omitted three years are 1892-1894. The regressions control for municipality and year fixed effects and weighted by the 1895 municipality population size. The dashed curves are 95 percent confidence bands based standard errors clustered at the municipality level.*

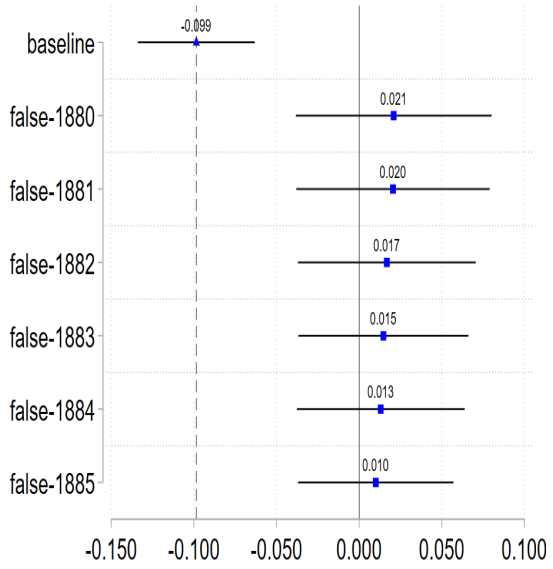
Figure A.8: Event-study estimates (relative to pre-antitoxin outcome), additional mortality outcomes II



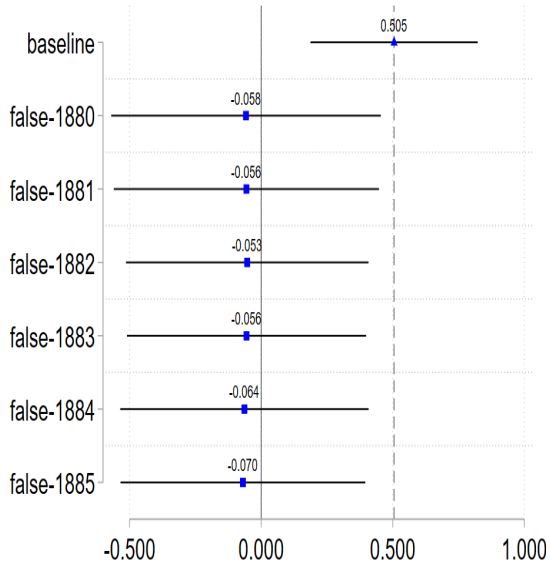
Notes: *This figure shows reduced-form event-study estimates for additional mortality outcomes. All estimates have been rescaled by the size of the pre-antitoxin outcome (i.e., the average from 1880 to 1894). Thus, one can interpret the estimates as percent changes. For comparison, Panel (a) shows the percent reductions in the diphtheria mortality rate. The different outcomes are described in the sub-figure title. In all regressions, we use a three-year pooling model, except for 1913 and 1914, which is two years. The omitted three years are 1892-1894. The regressions control for municipality and year fixed effects and weighted by the 1895 municipality population size. The black dashed curves are 95 percent confidence bands based standard errors clustered at the municipality level. The blue dashed curves are confidence bands, allowing for linear smoothness (i.e., linear pre-trends) according to Rambachan and Roth (2023).*

Figure A.9: False antitoxin start dates

(a) Diphtheria



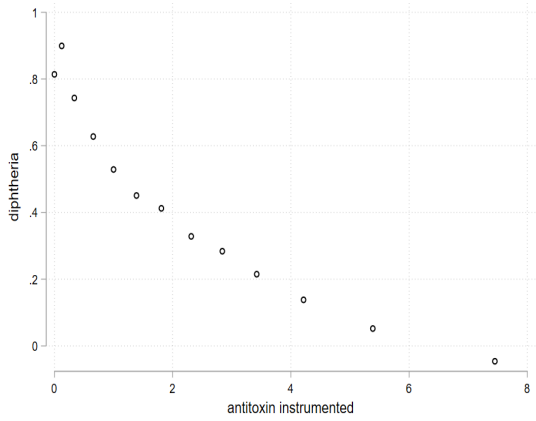
(b) Life expectancy at age 1



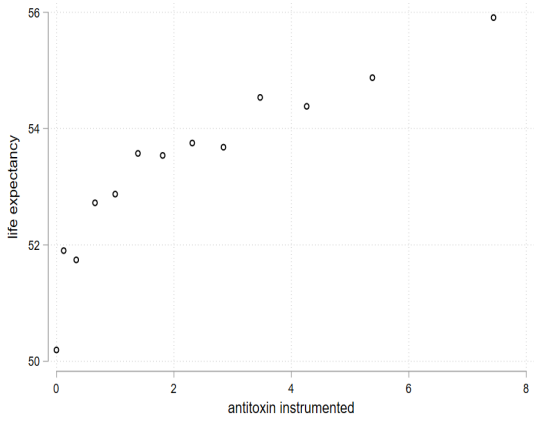
Notes: *This figure reports 2SLS estimates when assuming false start dates for the free distribution of antitoxin. In Panel (a), the outcome is the diphtheria mortality rate, and in Panel (b), the outcome is life expectancy at age 1. Both outcomes are measured for the years 1880 to 1896, and in 'false-1880', we assume that the distribution of antitoxin starts in 1880 and in 'false-1881' the start year is 1881, etc. For comparison, the baseline estimates are also reported as 'baseline'. All regressions are weighted by the municipality population size in 1895 and control for municipality and county-by-year fixed effects. Standard errors account for arbitrary heteroskedasticity and are clustered at the municipality level. The horizontal black solid lines are 95 percent confidence bands.*

Figure A.10: Binscatter plots

(a) Diphtheria

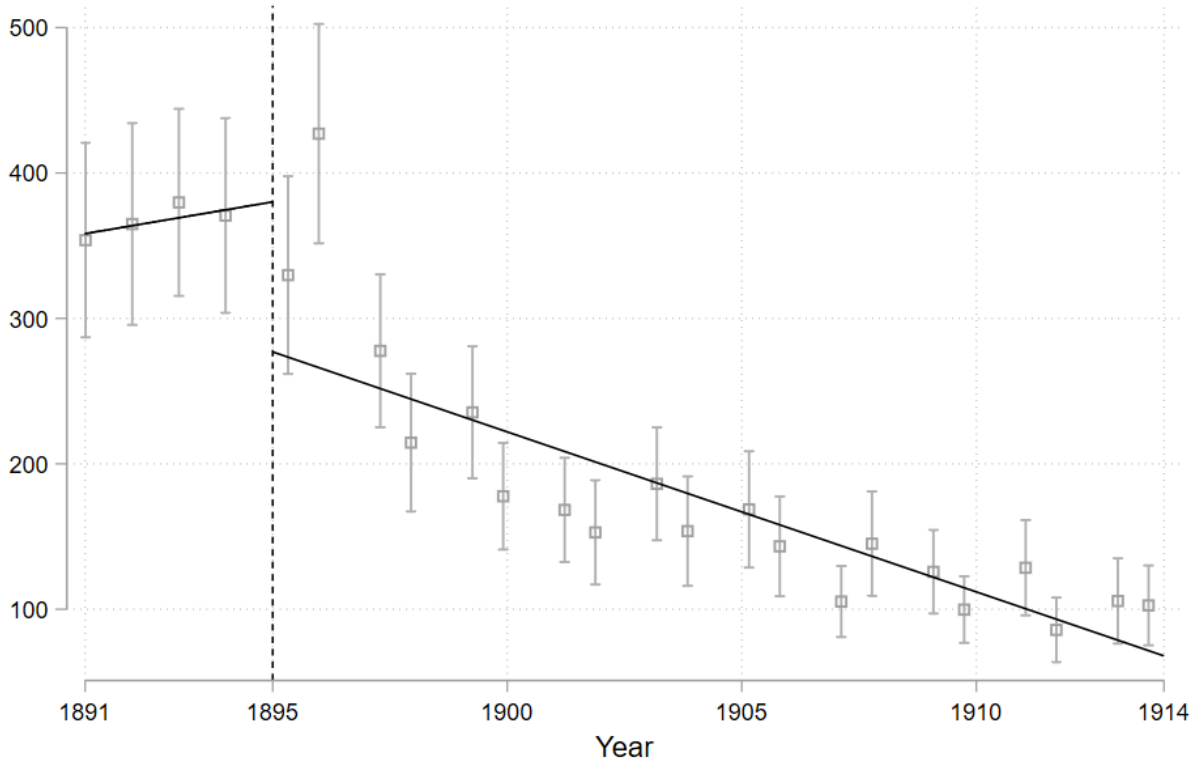


(b) Life expectancy at age 1



Notes: *This figure shows the relationship between the two outcomes and antitoxin instrumented in binned scatterplots, using the residualization procedure as described in Cattaneo et al. (2024) to control for the baseline fixed effects.*

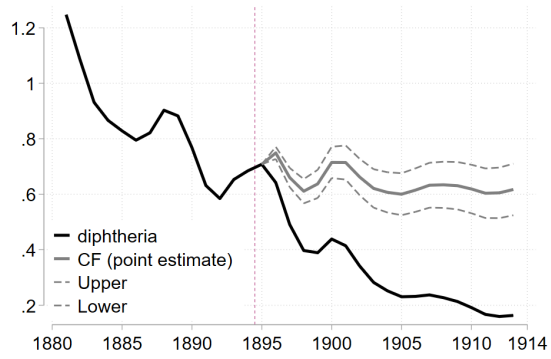
Figure A.11: Trends in the diphtheria case-fatality rate



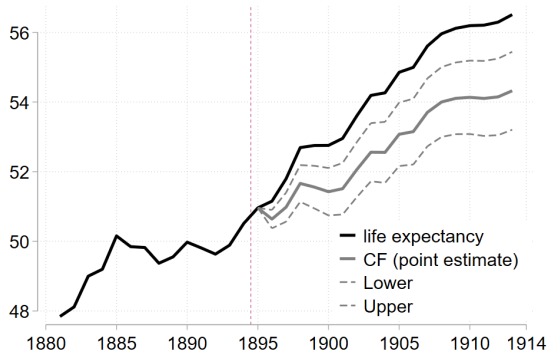
Notes: *This figure shows the development of the case-fatality rate, defined as the number of diphtheria deaths per 1,000 cases. Case data are available only from 1891, and the panel is unbalanced. Observations exceeding 1,000 have been capped at 1,000. The gray vertical lines represent 95% confidence intervals.*

Figure A.12: Robustness to counterfactual when delaying the policy

(a) Diphtheria

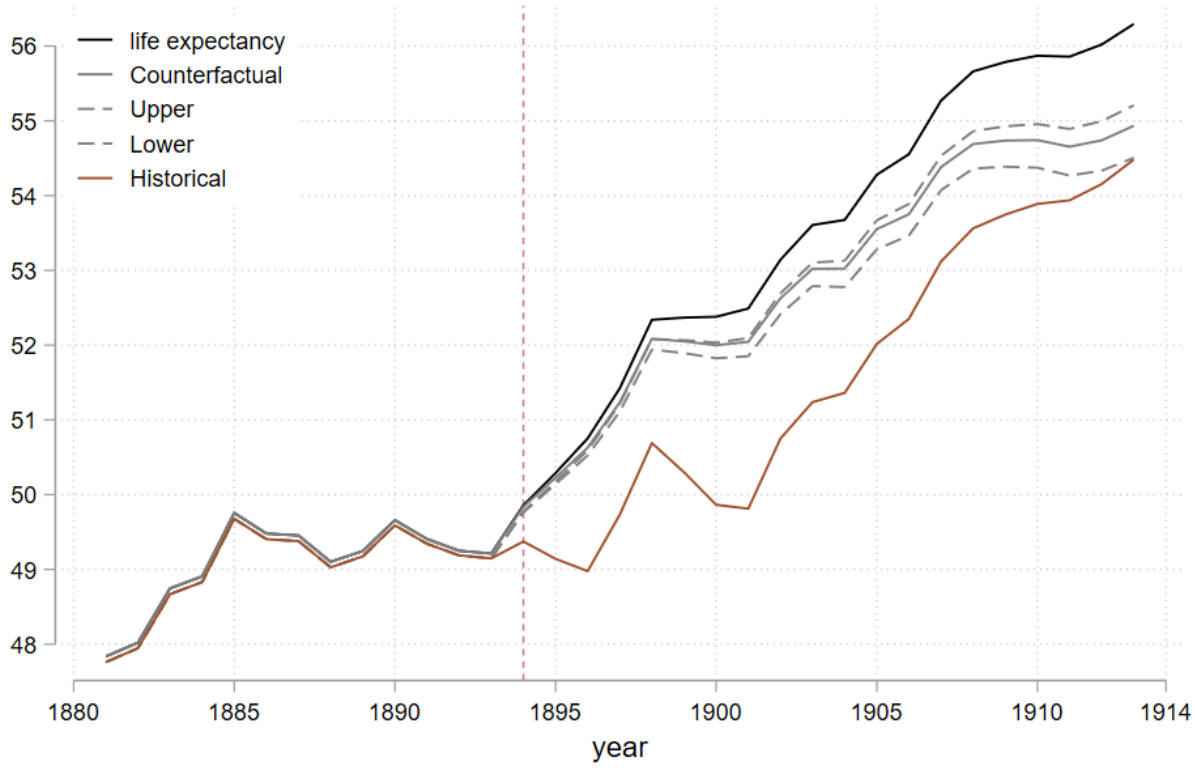


(b) Life expectancy



Notes: *This figure presents reduced-form estimates using logged year instead of linear year to calculate the counterfactual (CF) trends for the diphtheria mortality rate (Panel A) and life expectancy at age 1 (Panel B). The CF trends, based on annual delays in average treatment intensity, are shown as gray solid lines, with dashed lines representing 95% confidence intervals. The solid black lines depict the observed population-weighted averages of the outcomes. All curves are smoothed using three-year moving averages.*

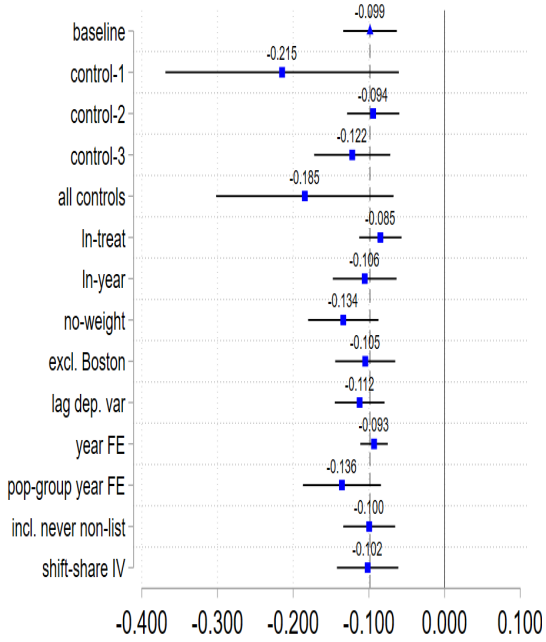
Figure A.13: Plausibility of magnitudes



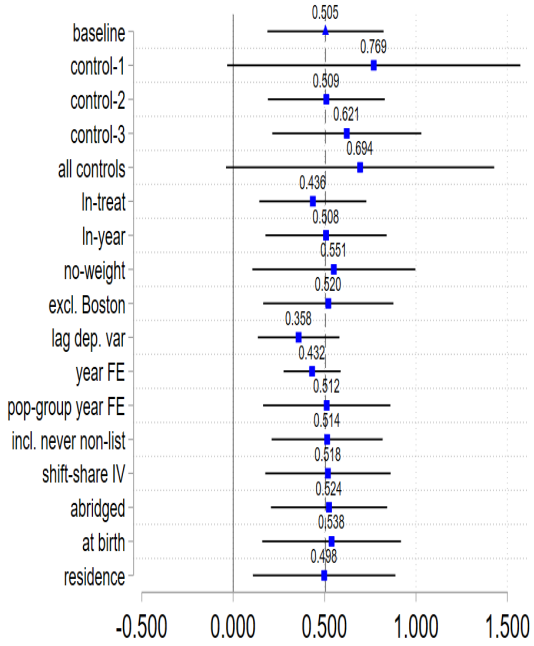
Notes: *This figure performs plausibility checks based on the counterfactual analysis for diphtheria shown in Panel (a) of Figure 5. We calculate the number of lives saved by comparing the observed diphtheria mortality rate to the counterfactual rate. We then assume these saved lives were evenly distributed across ages 1 to 5 and subtract these figures from the aggregate life table for Massachusetts. The historical counterfactual is based on the number of lives saved according to SBH report from 1901. This report provides the number of live saved due to antitoxin for the years 1895 and 1901. For the remaining years, we use the average of lives saved for the years 1899, 1900, and 1901.*

Figure A.14: Robustness checks

(a) Diphtheria



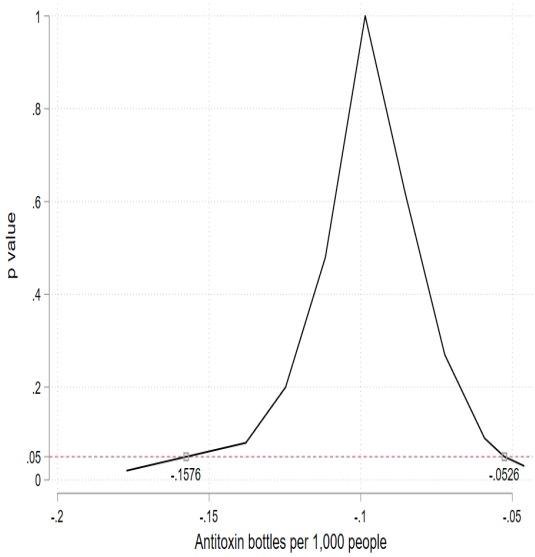
(b) Life expectancy at age 1



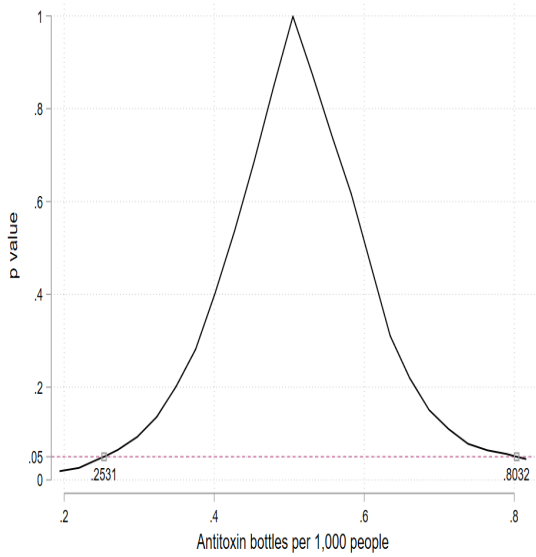
Notes: *This figure presents 2SLS estimates of the baseline model subjected to various robustness checks. The top row highlights the baseline estimates for the diphtheria mortality rate (Panel a) and life expectancy at age 1 (Panel b). The specification 'control-1' includes controls for all pre-antitoxin variables listed in Table 1, along with year fixed effects. 'Control-2' adds population shares by age groups (0, 1-4, 5-9, and 10-14), while 'control-3' incorporates controls for the roll-out of pubic water works, infectious disease hospitals, and general hospitals. The 'all controls' specification combines all these controls in one model. The 'ln-treat' specification uses logged treatment levels instead of levels in the baseline model. The 'ln-year' specification replaces the linear year variable with the natural logarithm of years since the policy was implemented. The 'no-weight' specification removes the weighting by running unweighted regressions. The 'excl. Boston' specification excludes the municipality of Boston. The 'lag. dep. var' specification includes up to five lags of the dependent variable. The 'year FE' specification replaces county-year fixed effects with year fixed effects, while the 'pop-group year FE' specification includes interactions between population size quartile groups from 1895 and year fixed effects. The 'incl. never non-list' specification includes never non-listed municipalities in terms of antitoxin bottles as zeros instead of dropping them from the sample. The 'shift-share IV' specification employs the shift-share instrument instead of the baseline IV. The 'abridged' and 'at birth' specifications provide estimates using life expectancy at age 1 from the abridged life table and life expectancy at birth, respectively. The 'residence' specification uses place of residence rather than place of occurrence (due to data limitations, the sample here ends in 1905). All regressions are weighted by the municipality population size in 1895 (except for the 'no-weight' specification) and include controls for municipality and county-by-year or year fixed effects. Standard errors are robust to heteroskedasticity and clustered at the municipality level. Horizontal lines represent 95% confidence intervals*

Figure A.15: Confidence intervals when clustering at the county level

(a) Diphtheria

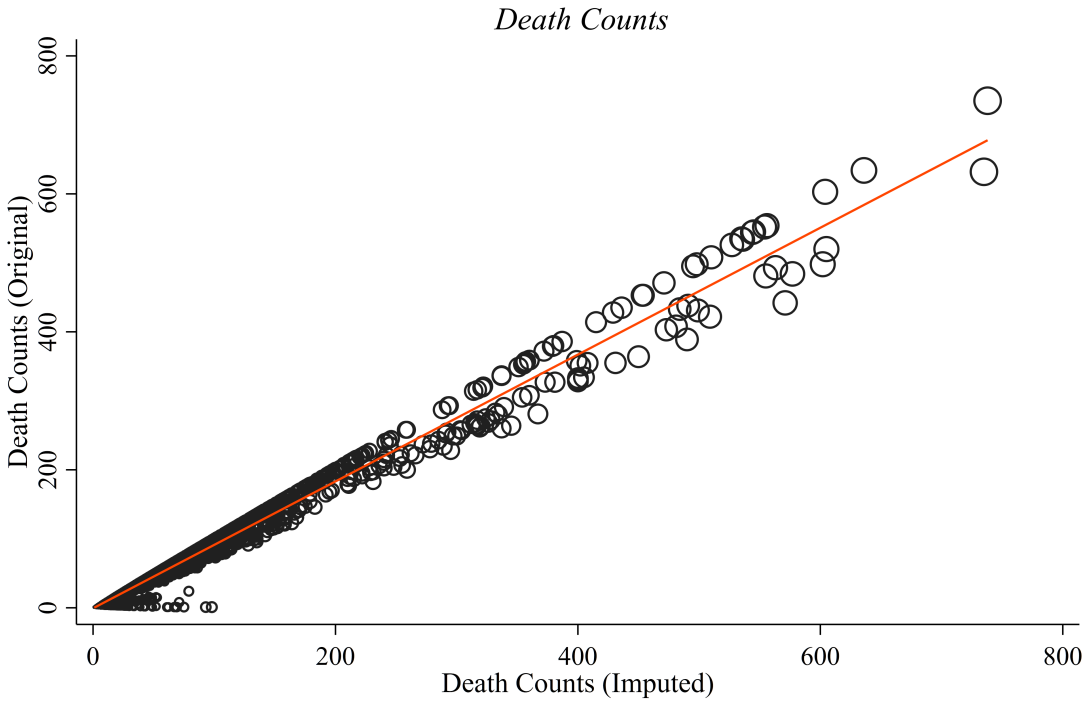


(b) Life expectancy at age 1



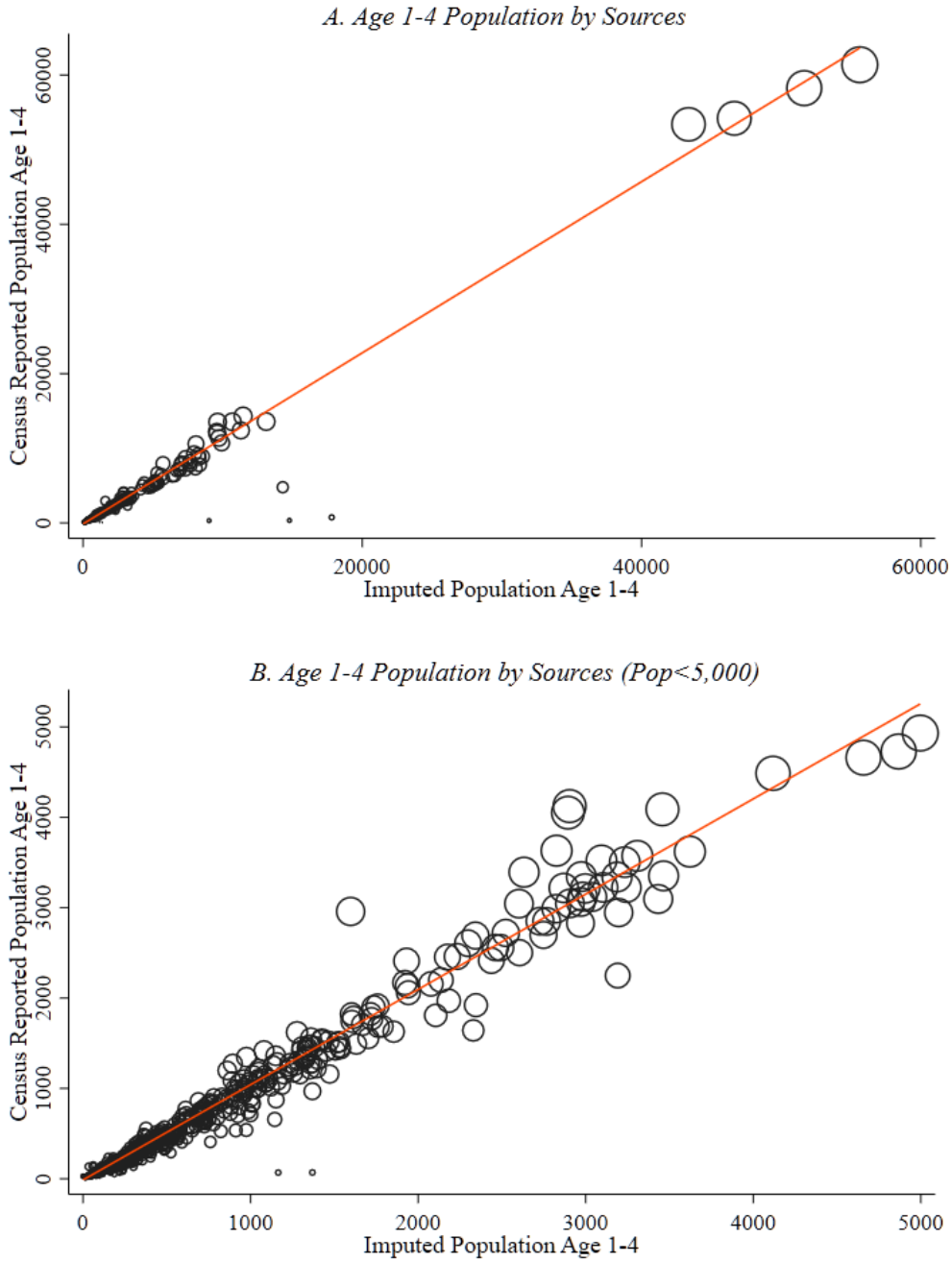
Notes: *This figure presents confidence intervals for the baseline model, with diphtheria mortality rate (Panel a) and life expectancy at age 1 (Panel b) as the outcomes, clustering standard errors at the county level. Given that Massachusetts has only 14 counties, the confidence intervals are calculated using wild-cluster bootstrapping with 100 replications.*

Figure A.16: 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.*

Figure A.17: 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*

Additional Tables

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

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)
	Pre-antitoxin period: 1880-1894					*Post-antitoxin period: 1895-1914*				
	N	mean	p25	p50	p75	N	mean	p25	p50	p75
diphteria rate	282	0.871	0.640	0.841	1.103	282	0.317	0.225	0.329	0.420
life expectancy at age 1	280	49.35	43.95	49.93	52.45	280	54.32	50.65	54.04	57.34
life expectancy at age 1 (abridged)	282	49.27	43.95	50.29	52.39	282	53.99	50.08	53.66	57.17
child mortality rate	282	23.21	17.91	23.45	29.40	282	14.32	10.65	13.87	18.80
infant mortality rate	282	153.2	130.5	159.9	165.3	282	146.1	124.9	145.0	152.0
population	282	113,350	6,143	27,773	77,148	282	171,512	9,895	44,115	105,042
antitoxin p.c.	282	0	0	0	0	282	9.481	5.991	9.421	13.78
treatment	282	0.735	0.477	0.685	1.155	282	0.735	0.477	0.685	1.155

Notes: *This table reports summary statistics for selected key variables averaged over the pre-antitoxin period (1880 to 1894) in columns (1)-(5) and the post-antitoxin periods (1895 to 1914) in columns (6)-(10), using the 1890 municipality population size as weight.*

Table A.2: Antitoxin adoption by municipality characteristics

	(1)	(2)	(3)	(4)	(5)	(6)
treatment × I × (t-1894)	0.348***	0.283***	0.262***	0.153***	0.162***	0.172***
	(0.058)	(0.058)	(0.056)	(0.049)	(0.046)	(0.046)
infect. rate × I × (t-1894)		0.025**	0.031***	-0.021	-0.022	-0.022
		(0.010)	(0.011)	(0.015)	(0.015)	(0.015)
apoplexy rate × I × (t-1894)		-0.089	-0.120**	-0.005	0.026	0.023
		(0.058)	(0.059)	(0.060)	(0.059)	(0.060)
doctors pc. × I × (t-1894)			0.098***	0.106***	0.092***	0.096***
			(0.032)	(0.032)	(0.030)	(0.030)
dist. Boston × I × (t-1894)				-0.004***	-0.003**	-0.003**
				(0.001)	(0.001)	(0.001)
pop. density × I × (t-1894)				0.028***	0.020**	0.019**
				(0.007)	(0.009)	(0.009)
dwel. size × I × (t-1894)				0.066**	0.024	0.026
				(0.034)	(0.030)	(0.030)
room size × I × (t-1894)				0.155	0.404	0.415
				(0.387)	(0.361)	(0.348)
immigrant × I × (t-1894)				0.022	0.269	0.254
				(0.359)	(0.362)	(0.370)
water intervention					-0.501*	-0.405
					(0.289)	(0.281)
infectious hospital					2.059**	2.088**
					(0.915)	(0.909)
general hospital					0.692**	0.669*
					(0.332)	(0.344)
age composition	no	no	no	no	no	yes
N	9870	9870	9800	9800	9800	9800

Notes: *This table reports the correlation between the number of antitoxin bottles per 1,000 people and selected municipality characteristics. The key variable, "treatment", is the average diphtheria mortality rate over 1889–1894. "Infect. rate" is the total mortality rates for eight infectious diseases (see data appendix), and "apoplexy rate" is the apoplexy (sudden death) mortality rate, both averaged over 1889–1894. "doctors pc." is doctors per 1,000 people in 1895; "dist. Boston" is the aerial distance to Boston; "pop. density" is population per 1,000 sq. miles in 1895; "dwel. size" and "room size" are the number of people per dwelling and per room in 1895, respectively; and "immigrant" is the share of foreign-born individuals in 1895. These pre-antitoxin period characteristics are interacted with a post-1895 dummy (I) and a linear trend (t-1894). The vector of age composition includes percentage of population at age 0, 1-4, 5-9, and 10-14. All regressions control for municipality and county-by-year fixed effects, and are weighted by the 1895 population. Standard errors (reported in parentheses) are robust and clustered at the municipality level. ***, **, and * indicate significance at 1%, 5%, and 10% levels.*

Table A.3: Decomposing the change of antitoxin adoption by treatment into municipality covariates

	(1) Base	(2) Model-1	(3) Explained	(4) Model-2	(5) Explained
treatment × I × (t-1894)	0.348	0.153	0.195	0.172	0.176
	(0.058)	(0.049)	(0.044)	(0.046)	(0.044)
Municipality Covariates:					
infect. rate × I × (t-1894)	No	Yes	-0.040	Yes	-0.042
			(0.031)		(0.030)
apoplexy rate × I × (t-1894)	No	Yes	0.001	Yes	-0.004
			(0.011)		(0.011)
doctors pc. × I × (t-1894)	No	Yes	0.002	Yes	0.002
			(0.016)		(0.014)
dist. Boston × I × (t-1894)	No	Yes	0.016	Yes	0.012
			(0.013)		(0.010)
pop. density × I × (t-1894)	No	Yes	0.083	Yes	0.057
			(0.030)		(0.028)
dwel. size × I × (t-1894)	No	Yes	0.112	Yes	0.045
			(0.064)		(0.051)
room size × I × (t-1894)	No	Yes	0.019	Yes	0.052
			(0.047)		(0.042)
immigrant × I × (t-1894)	No	Yes	0.003	Yes	0.029
			(0.040)		(0.042)
water intervention	No	No		Yes	0.004
					(0.003)
infectious hospital	No	No		Yes	0.029
					(0.024)
general hospital	No	No		Yes	0.005
					(0.005)
age composition	No	No		Yes	-0.012
					(0.006)

Notes: *This table decomposes the changes in the coefficients of the "treatment", which is interacted with a post-1895 dummy and a linear time trend (t-1984), as reported in Table A.2. Column (1) reports the coefficient on the treatment variable in a base model without any municipality-level covariates (Table A.2, column 1). Column (2) shows the coefficient on the treatment variable in the model including pre-antitoxin municipality covariates, interacted with a post-1895 dummy and a linear time trend (Table A.2, column 4). Column (3) decomposes the coefficient change between column (1) and column (2) into municipality covariates, based on Gelbach (2016). Column (4) provides the coefficient on the treatment variable in a full model controlling for additional municipality covariates, including the presence of a city sanitation system, infectious disease hospitals, general hospitals, and the age composition of the population (Table A.2, column 6). Column (5) decomposes the coefficient change between the base model (Column 1) and the full model (Column 4). Standard errors are clustered at the municipality level and are reported in parentheses.*

Table A.4: Decomposing the change of estimated effect of antitoxin distribution on public health into municipality covariates

	Diphteria Mortality Rate			Life Expectancy at Age 1		
	Base	Full	Explained	Base	Full	Explained
	(1)	(2)	(3)	(4)	(5)	(6)
antitoxin p.c.	-0.099	-0.185	0.086	0.505	0.694	-0.189
	(0.018)	(0.059)	(0.046)	(0.161)	(0.372)	(0.266)
Municipality Covariates:						
infect. rate × Year FE	No	Yes	-0.021	No	Yes	0.216
			(0.017)			(0.113)
apoplexy rate × Year FE	No	Yes	-0.002	No	Yes	0.070
			(0.006)			(0.041)
doctors pc. × Year FE	No	Yes	0.001	No	Yes	-0.012
			(0.009)			(0.071)
dist. Boston × Year FE	No	Yes	0.006	No	Yes	-0.038
			(0.006)			(0.039)
pop. density × Year FE	No	Yes	0.027	No	Yes	-0.163
			(0.020)			(0.102)
dwel. size × Year FE	No	Yes	0.032	No	Yes	-0.109
			(0.030)			(0.197)
room size × Year FE	No	Yes	0.024	No	Yes	-0.217
			(0.030)			(0.203)
immigrant × Year FE	No	Yes	0.015	No	Yes	0.065
			(0.023)			(0.125)
water intervention	No	Yes	0.002	No	Yes	-0.002
			(0.002)			(0.013)
infectious hospital	No	Yes	0.010	No	Yes	-0.038
			(0.008)			(0.042)
general hospital	No	Yes	0.003	No	Yes	0.012
			(0.003)			(0.014)
age composition	No	Yes	-0.011	No	Yes	0.027
			(0.006)			(0.029)

Notes: *This table decomposes the change in the causal effect of antitoxin distribution on public health into a list of municipality covariates, based on the approach by Gelbach (2016). The causal effects are estimated using the instrumental variable approach, as described in the main text. The estimates are visualized in Figure A.14. Column (1) reports the 2SLS estimate of the effect of antitoxin distribution on the diphtheria mortality rate in a model that excludes municipality covariates (see also Figure A.14, panel (a), baseline). Column (2) reports the estimated causal effect in a full specification that includes a comprehensive list of municipality covariates specified in Table A.2, column (6) (see also Figure A.14, panel (a), all controls). Column (3) decomposes the difference in estimates between columns (1) and (2) into all municipality covariates. Similarly, columns (4)-(6) decompose the change in estimated causal effects of antitoxin diffusion on life expectancy at age 1. Standard errors are reported in parentheses and are clustered at the municipality level.*

Table A.5: 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.	-3.123*	-1.497	-1.208***	-0.855***	-0.747***	-0.583***	-0.389***	-0.276**	-0.161*	-0.238**	-0.065
	(1.731)	(0.969)	(0.315)	(0.229)	(0.203)	(0.162)	(0.142)	(0.121)	(0.097)	(0.108)	(0.092)
Mean pre-y	180.3	46.16	19.37	13.14	9.944	7.798	6.294	5.242	4.420	3.742	3.338
$N \times T$	9800	9800	9800	9800	9800	9799	9800	9800	9800	9800	9800
N	280	280	280	280	280	280	280	280	280	280	280
KP-F Stat.	35.99	35.99	35.99	35.99	35.99	35.98	35.99	35.99	35.99	35.99	35.99
AR 95 CI	[-6.93:0.15]	[-3.77:0.19]	[-1.97:-0.69]	[-1.41:-0.47]	[-1.23:-0.39]	[-0.98:-0.31]	[-0.72:-0.14]	[-0.56:-0.07]	[-0.39:0.01]	[-0.50:-0.05]	[-0.27:0.11]
tf 95 CI	[-6.91:0.66]	[-3.61:0.62]	[-1.90:-0.52]	[-1.36:-0.35]	[-1.19:-0.30]	[-0.94:-0.23]	[-.70: -0.08]	[-0.54:-0.01]	[-0.37:0.05]	[-0.47:0.00]	[-0.27:0.13]

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. KP-F-Stat is Kleibergen-Paap F statistic, AR 95 CI is the Anderson-Rubin 95% confidence intervals (Anderson and Rubin 1949) and tf 95% confidence intervals (Lee et al. 2022).*

Table A.6: Stacked mortality model

	(1) mortality	(2) mortality	(3) mortality	(4) mortality	(5) mortality
antitoxin p.c. x I	-0.101***	-0.159**	-0.108***	-0.077***	-0.079***
	(0.028)	(0.066)	(0.029)	(0.024)	(0.024)
Controls	all	exogenous	childhood	declining	waterborne
$N \times T$	95862	14748	29496	44244	22122
N	282	282	282	282	282
KP-F Stat.	16.84	16.84	16.84	16.84	16.84

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 12 control diseases (typhoid, tuberculosis, pneumonia, scarlet fever, measles, whooping cough, bronchitis, accidents, childbirth, meningitis, apoplexy, and digestive diseases). Column 2 only includes "exogenous" causes as controls (accidents). 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). 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.7: 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.	-0.021	-0.009	-0.011	-0.010	0.003
	(0.022)	(0.010)	(0.008)	(0.010)	(0.008)
Mean pre-y	1.634	0.273	0.484	0.355	0.280
AR 95 CI	[-0.07:0.02]	[-0.03:0.01]	[-0.03:0.01]	[-0.03:0.01]	[-0.01:.02]
tf 95 CI	[-0.07:0.03]	[-0.03:0.01]	[-0.03:0.01]	[-0.03:0.01]	[-0.02:0.02]
Panel B: Nurses					
antitoxin p.c.	0.056	0.064	0.009	-0.010	0.001
	(0.107)	(0.079)	(0.018)	(0.008)	(0.004)
Mean pre-y	0.100	0.0467	0.0323	0.00995	0.00501
AR 95 CI	[-0.15:0.28]	[-0.08:0.23]	[-0.03:0.04]	[-0.03:0.01]	[-0.01:0.01]
tf 95 CI	[-0.18:0.29]	[-0.11:0.24]	[-0.03:0.05]	[-0.03:0.01]	[-0.01:0.01]
Panel C: Pharmacists					
antitoxin p.c.	-0.009	-0.001	-0.007	0.003	-0.007
	(0.014)	(0.007)	(0.007)	(0.005)	(0.005)
Mean pre-y	0.742	0.222	0.240	0.137	0.0784
AR 95 CI	[-0.04:0.02]	[-0.01:0.01]	[-0.02:0.01]	[-0.01:0.01]	[-0.02:0.00]
tf 95 CI	[-0.04:0.02]	[-0.02:0.01]	[-0.02:0.01]	[-0.01:0.01]	[-0.02:0.00]
$N \times T$	9800	9800	9800	9800	9800
N	280	280	280	280	280
KP-F-Stat	35.99	35.99	35.99	35.99	35.99

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. KP-F-Stat is Kleibergen-Paap F statistic, AR 95 CI is the Anderson-Rubin 95% confidence intervals (Anderson and Rubin 1949) and tf 95% confidence intervals (Lee et al. 2022).*

Table A.8: Cost effectiveness analysis

(1) Year	(2) SBH Period (start-end)	(3) Account expenses ($)	(4) Bottles (Q)	(5) Cost pr bottle ($)	(6) Market price pr. bottle ($)	(7) Saved consum ($)	(8) Patients treated (Q)	(9) Total cost ($)	(10) Cost pr. life saved (prd only) ($)	(11) in 2023 ($)	(12) Cost pr. life saved (total) ($)	(13) in 2023 ($)
1895	1895 Mar. 31 - 1896 Mar. 31	-	1,724	0.15	1.50	2,326.47	344.80	1,983.53	4.18	156.51	31.95	1,196.15
1896	1896 Mar. 31 - 1897 Mar. 31	-	3,219	0.15	1.50	4,343.92	643.80	3,703.58	3.81	142.83	29.16	1,091.60
1897	1897 Mar. 31 - 1898 Mar. 31	-	4,668	0.15	1.50	6,299.29	933.60	5,370.71	3.60	136.61	27.55	1,044.11
1898	1898 Mar. 31 - 1899 Mar. 31	-	12,491	0.15	1.50	16,856.13	2,498.20	14,371.37	7.07	267.85	54.02	2,047.13
1899	1899 Mar. 31 - 1900 Mar. 31	-	31,997	0.15	1.50	43,178.75	6,399.40	36,813.75	14.14	536.09	108.11	4,097.27
1900	1900 Mar. 31 - 1901 Mar. 31	-	40,211	0.15	1.50	54,263.23	8,042.20	46,264.27	14.46	541.44	110.55	4,138.12
1901	1901 Mar. 31 - 1902 Mar. 31	-	53,389	0.15	1.50	72,046.45	10,677.80	61,426.05	16.24	600.70	124.16	4,591.10
1902	1902 Mar. 31 - 1903 Mar. 31	-	40,211	0.15	1.54	55,596.48	8,042.20	46,413.00	10.82	395.36	80.98	2,958.71
1903	1903 Mar. 31 - 1904 Mar. 31	-	33,475	0.16	1.57	47,337.57	6,695.00	38,755.69	8.08	288.55	59.28	2,117.71
1904	1904 Mar. 31 - 1904 Sep. 30	5,858.28	22,255	0.26	1.59	29,533.73	4,451.00	28,113.28	7.95	280.78	38.16	1,347.42
1905	1904 Sep. 30 - 1905 Sep. 30	9,301.52	47,387	0.20	1.57	65,184.56	9,477.40	56,688.52	11.31	404.17	68.96	2,463.24
1906	1905 Sep. 30 - 1906 Nov. 30	13,067.39	70,424	0.19	1.61	100,095.50	14,084.80	83,491.39	14.24	497.85	91.03	3,180.92
1907	1906 Nov. 30 - 1907 Nov. 30	14,491.00	64,087	0.23	1.68	93,094.85	12,817.40	78,578.00	14.25	476.82	77.30	2,585.56
1908	1907 Nov. 30 - 1908 Nov. 30	14,999.98	94,645	0.16	1.64	140,571.29	18,929.00	109,644.98	13.38	457.37	97.87	3,343.23
1909	1908 Nov. 30 - 1909 Nov. 30	18,783.25	90,131	0.21	1.63	127,707.31	18,026.20	108,914.25	15.28	528.07	88.63	3,062.01
1910	1909 Nov. 30 - 1910 Nov. 30	19,992.31	92,623	0.22	1.70	137,205.08	18,524.60	112,615.31	14.89	492.75	83.90	2,775.63
1911	1910 Nov. 30 - 1911 Nov. 30	18,544.14	96,522	0.19	1.70	145,270.53	19,304.40	115,066.14	12.76	422.44	79.23	2,621.24
1912	1911 Nov. 30 - 1912 Nov. 30	19,968.68	82,085	0.24	1.73	122,217.87	16,417.00	102,053.68	12.75	413.28	65.16	2,112.14
1913	1912 Nov. 30 - 1913 Nov. 30	20,377.20	96,891	0.21	1.77	151,027.03	19,378.20	117,268.20	12.09	384.01	69.63	2,209.92
1914	1913 Nov. 30 - 1914 Nov. 30	20,968.85	118,561	0.18	1.79	190,736.82	23,712.20	139,529.85	11.60	364.98	77.24	2,428.66

Notes: *Column (1) shows the calendar year, while column (2) lists the SBH production period associated with it. Column (3) reports the expenditure for antitoxin production based on SBH accounts, available only from 1904 onward. Column (4) provides the total number of bottles produced. Column (5) shows the cost per bottle: $0.15 for 1901, adjusted for inflation in 1902-1903, and calculated by dividing columns (3) and (4) from 1904 onward. Column (6) lists market prices per bottle, based on SBH (1901) and adjusted for inflation starting in 1902. Column (7) estimates total consumer savings under the free distribution policy, calculated as the difference between columns (6) and (5), multiplied by column (4). Column (8) reports the number of patients treated, assuming five bottles per patient as per SBH (1901). Column (9) calculates total costs, combining production costs and an assumed additional $5 per patient. Column (10) gives the cost per averted diphtheria death, considering only production costs and using a baseline counterfactual. Column (11) adjusts this figure to 2023 dollar values. Columns (12) and (13) provide the corresponding 2023 values based on total costs instead of production costs alone.*

Table A.9: Replication of Table 5 by Gender and Parental Background

	(1)	(2)	(3)	(4)	(5)	(6)
PANEL A	== 1 if attends $\leqq$ 3 months					
Sample	Boys	Girls	Parents FB	Parents US	Father Low Skill	Father High Skill
Exposure	-0.005***	-0.005***	-0.005***	-0.006***	-0.005***	-0.005***
	(0.001)	(0.001)	(0.001)	(0.002)	(0.001)	(0.001)
Observations	151,378	150,555	137,091	110,803	120,930	181,017
R-squared	0.043	0.042	0.047	0.041	0.051	0.038
Mean(Y)	0.107	0.109	0.114	0.100	0.111	0.105
PANEL B	== 1 if attends $\geqq$ 8 months					
Sample	Boys	Girls	Parents FB	Parents US	Father Low Skill	Father High Skill
Exposure	0.006***	0.006***	0.006***	0.006***	0.005***	0.006***
	(0.001)	(0.001)	(0.001)	(0.002)	(0.001)	(0.001)
Observations	151,378	150,555	137,091	110,803	120,930	181,017
R-squared	0.057	0.055	0.060	0.057	0.066	0.052
Pre-Y mean	0.780	0.773	0.769	0.785	0.770	0.781
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 in 1900 by gender (columns 1-2), parental birthplace (columns 3-4), and the father's socioeconomic status (columns 5-6). The dependent variable is a dummy of whether a child aged 5-15 attended school for no more than three months in Panel A and for at least eight 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. All regressions include fixed effects for municipality and year of birth and a set of individual controls (except excluding the gender dummies columns 1-2) as outlined on page 37. 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: Replication of Table 5 based on the linked sample

	(1)	(2)	(3)	(4)	(5)	(6)
	==1 if attends at all		== 1 if attends $\leqq$ 3 months		== 1 if attends $\geqq$ 8 months	
Exposure	0.003	0.003	-0.005***	-0.005***	0.007***	0.007***
	(0.003)	(0.003)	(0.001)	(0.001)	(0.001)	(0.001)
Observations	246,889	246,859	178,170	178,132	178,170	178,132
R-squared	0.145	0.158	0.033	0.040	0.046	0.053
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	0.682	0.682	0.104	0.104	0.784	0.784

Notes: *This table replicates 5 using the linked sample (1900 to 1940). The sample includes 5 to 15-year-old children who lived in Massachusetts in 1900 and 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. The controls are the same as in Table 5. 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: Replication of Table 6 by Gender

PANEL A	(1) years in school	(2) low-skill occ	(3) blue-collar occ	(4) white-collar occ	(5) ln(occscore)	(6) ln(wages)
			MEN			
Exposure	0.014**	0.0006	-0.0009	0.0003	0.0016***	0.0021*
	(0.006)	(0.0006)	(0.0008)	(0.0006)	(0.0005)	(0.0012)
Observations	138,788	144,184	144,184	144,184	134,236	108,871
R-squared	0.165	0.046	0.025	0.071	0.039	0.059
Mean(Y)	9.953	0.307	0.206	0.398	3.326	7.236
PANEL B	years in school	low-skill occ	blue-collar occ	white-collar occ	ln(occscore)	ln(wages)
			WOMEN			
Exposure	-0.002	-0.0007	0.0000	-0.0005	-0.0020*	0.0013
	(0.005)	(0.0005)	(0.0001)	(0.0007)	(0.0012)	(0.0025)
Observations	98,932	102,621	102,621	102,621	31,067	25,874
R-squared	0.192	0.034	0.009	0.029	0.050	0.094
Mean(Y)	10.29	0.112	0.007	0.182	3.060	6.715
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 during childhood affected labor market outcomes as adults by gender. The sample includes 5 to 15-year-old children who lived in Massachusetts in 1900 and can be linked to 1940 using the crosswalks from the Census Tree Project. The following outcomes in 1940 are used as dependent variables: educational attainment (column 1), a dummy of 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. All regressions control for municipality and year of birth fixed effects and a set of individual controls (see page 37 for details). 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: Replication of Table 6 by Mover Status

PANEL A	(1) years in school	(2) low-skill occ	(3) blue-collar occ	(4) white-collar occ	(5) ln(occscore)	(6) ln(wages)
			STAYER			
Exposure	-0.002	0.0005	-0.0001	-0.0005	0.0009	-0.0009
	(0.005)	(0.0007)	(0.0005)	(0.0008)	(0.0008)	(0.0015)
Observations	122,894	128,710	128,710	128,710	84,777	69,957
R-squared	0.180	0.106	0.115	0.079	0.112	0.135
Mean(Y)	9.765	0.250	0.120	0.278	3.253	7.049
PANEL B	years in school	low-skill occ	blue-collar occ	white-collar occ	ln(occscore)	ln(wages)
			MOVER (OUT OF STATE)			
Exposure	0.019***	-0.0008	-0.0018**	0.0019*	0.0021*	0.0061**
	(0.006)	(0.0007)	(0.0009)	(0.0010)	(0.0012)	(0.0027)
Observations	57,122	58,151	58,151	58,151	41,094	32,530
R-squared	0.229	0.090	0.097	0.151	0.101	0.121
Mean(Y)	10.58	0.189	0.129	0.369	3.310	7.270
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 during childhood affected labor market outcomes as adults by mover status. The sample includes 5 to 15-year-old children who lived in Massachusetts in 1900 linked to 1940 using the crosswalks from the Census Tree Project. The following outcomes in 1940 are used as dependent variables: educational attainment (column 1), a dummy of 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. All regressions control for municipality and year of birth fixed effects and a set of individual controls (see page 37 for details). 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.13: The Long-run Effects of Antitoxin Treatment – Different Periods

	(1) low-skilled	(2) blue-collar skilled	(3) white-collar	(4) ln(occscore)
Panel A: Sample 1900-1920				
Exposure	-0.0024***	0.0006	0.0024***	0.0008
	(0.0007)	(0.0004)	(0.0005)	(0.0005)
Observations	251,017	251,017	251,017	166,396
R-squared	0.114	0.154	0.067	0.106
Mean(Y)	0.235	0.135	0.284	3.229
Panel B: Sample 1900-1930				
Exposure	0.0002	-0.0003	0.0003	-0.0002
	(0.0004)	(0.0003)	(0.0006)	(0.0005)
Observations	267,003	267,003	267,003	183,978
R-squared	0.097	0.121	0.105	0.119
Mean(Y)	0.218	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 during childhood affected labor market outcomes as adults based on a sample 5 to 15-year-old children who lived in Massachusetts in 1900 and can be linked to 1920 (Panel A) and 1930 (Panel B) using the crosswalks from the Census Tree Project. 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. All regressions control for municipality and year of birth fixed effects and a set of individual controls (see page 37 for details). 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.14: The Long-run Effects of Antitoxin on Employment Status

	(1) wages	(2) bus. income over 50 USD	(3) works at all	(4) works in PWA projects	(5) without work	(6) unable to work
Exposure	3.2776**	-0.0002	-0.0003	-0.0009***	-0.0001	0.0001
	(1.4417)	(0.0007)	(0.0005)	(0.0002)	(0.0004)	(0.0001)
Observations	134,807	230,727	246,859	246,859	163,566	246,859
R-squared	0.126	0.036	0.350	0.022	0.010	0.007
TOWN FE	YES	YES	YES	YES	YES	YES
YOB FE	YES	YES	YES	YES	YES	YES
CONTROLS	YES	YES	YES	YES	YES	YES
Mean(Y)	1655	0.253	0.620	0.039	0.064	0.026

Notes: *This table reports how antitoxin exposure during childhood affected the employment status as adults. The sample includes 5 to 15-year-old children who lived in Massachusetts in 1900 and can be linked to 1940 using the crosswalks from the Census Tree Project. The following outcomes in 1940 are used as dependent variables: wages (column 1), a dummy of whether the individual has a business income over 50 USD (column 2), works at all (column 3), was employed on public emergency work projects (column 4), was unemployed (column 5), or unable to work (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. All regressions control for municipality and year of birth fixed effects and a set of individual controls (see page 37 for details). 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.1 Individual death records and mortality rates by age

More than 17 million individual death records from Massachusetts between 1880 and 1914 have been digitized and published by *FamilySearch.org*, one of the world's leading genealogy platforms. These records provide detailed geographic and demographic information, enabling the aggregation of individual data into death counts at age-year-municipality level. However, processing these raw death records involves two major technical challenges.

The first challenge arises from a significant number of records with missing age information, caused by incomplete details in the original records or transcription errors. Before 1906, the proportion of records with missing ages was below 2%, but it rose to an average of 26.8% between 1906 and 1914. To address this issue, we redistribute the counts of these records across ages 0 to 100, based on the age distribution of records with non-missing ages. This redistribution is performed within each group defined by municipality, death year, death season (April-September or October-March), decedent's sex (male, female, or unknown), nativity (born in Massachusetts, other U.S. states, foreign countries, or unknown), and marital status (never married, ever married, or unknown).

Figure A.16 compares the original death counts (excluding records with missing ages) to the death counts incorporating the redistributed missing-age records for the 1906-1914 period. The results demonstrate a strong positive correlation, indicating that records with missing ages do not significantly distort the overall mortality distribution. Our baseline analysis relies on these imputed death counts.

The second challenge involves standardizing geographic information for both the place of death and the decedent's residence. The original records lacked consistent place names, necessitating the cleaning of 12,229 unique transcribed death locations across 1,765,574 death records. These locations were then assigned to 319 municipalities with standardized boundaries. As a result, we identified a municipality of death for 99.59% of all records (98.95% within Massachusetts and 0.64% outside Massachusetts but registered in the state).

To account for the possibility that many individuals died away from their usual residence, often in hospitals, we also standardize residence information where available. Unfortunately, residence data was only available for deaths before 1906. For this period, we cleaned 9,344 unique non-standardized residence places and assigned them to the same standardized municipalities. Among 1,227,487 death records from 1880 to 1905, we successfully identified residence municipalities for 1,225,708 records (1,205,558 within Massachusetts and 20,150 outside the state). Furthermore, among the 1,201,174 deaths with identified death and residence municipalities within Massachusetts, 95.4% (1,145,497 cases) occurred in the same municipality. This evidence suggests that deviations between death and residence locations were not a significant issue during this period. For our baseline analysis, we rely on death locations, which are more comprehensively recorded after 1906.

Using the death counts and population data, we then calculate age-specific mortality rates, which serve as the basis for constructing life tables and estimating life expectancy. Population data by age are primarily derived from the full-count microdata of the federal census for the years 1880, 1900, and 1910 (Ruggles et al. 2017). For these census years, we aggregate individuals by single-year ages from 0 to 79 and by municipality, then log-linearly interpolate the population for non-census years between 1880 and 1910. For the years 1911 to 1914, we extrapolate population estimates.

In addition to general age-specific mortality rates, we place particular emphasis on infant mortality (age under 1) and child mortality (ages 1-4), given that young children were the most vulnerable to diphtheria infections. To measure mortality rates in these early age groups more accurately, we utilize birth counts aggregated from individual birth records, which are also digitized and made publicly available by *FamilySearch.org*. Similar to the death records, birth records also contain detailed geographic information, enabling us to calculate birth counts at the municipality-year level. Specifically, we define the infant mortality rate as follows:

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

where $Deaths^{0}_{mt}$ denotes the number of deaths at age 0 in municipality m and year t, and $Births_{mt}$ represents the number of births in the same municipality and year.

Next, we calculate the child mortality rate (ages 1-4) in municipality m and year t as follows:

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

where $Deaths^{1-4}_{mt}$ is the number of deaths among children aged 1-4 in municipality m and year t, and Pop^{1-4}_{mt} represents the population of children aged 1-4 in the same municipality and year. Rather than relying on interpolated population data, we impute the annual population of children aged 1-4 based on cumulative births and deaths for the corresponding cohorts in prior years, assuming that migration among children in this age group is negligible. This approach is also employed by Alsan and Goldin (2019) and Eriksson et al. (2020). Specifically, the population of children aged 1-4 is imputed as follows:

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

Here, $Births_{m,t-a}$ denotes the number of births in municipality m during year $(t-a)$, while $Deaths^{a-k}_{m,t-k}$ represents the number of deaths at age $(a-k)$ in municipality m during year $(t-k)$. For clarity, consider the cohort of children born two years prior ($a = 2$) as an example. In this case, $Births_{m,t-2}$ refers to the total number of children born in municipality m two years earlier (relative to year t), while the sum of $Deaths^{2-1}_{m,t-1}$ and $Deaths^{2-2}_{m,t-2}$ corresponds to the cumulative number of these children who died over the past two years.

Finally, We compare our imputed population of children aged 1 to 4 with the population directly reported by federal or state censuses in years when were available. Figure A.17 shows the imputed population fit well the census-reported population at the municipality level in census years.

A.2 Construction of life tables and life expectancy

This appendix explains the setup of the period life tables used to derive the various life expectancy measures. These tables estimate the number of years a hypothetical person or cohort can expect to live at a given age, based on the prevailing age-specific mortality rates in calendar year t. Our baseline life expectancy measure is derived from the single-year age life table, which we will explain first. However, we also present life expectancy derived from the abridged life table (i.e., using broader age groups instead of single-year intervals), which will be explained afterward.

A.2.1 The single-year age life table

In the baseline measure of life expectancy, the death counts from the individual-level death certificates are combined with population data by age from the federal censuses (Ruggles et al. 2021). We use data for the years 1880, 1900, and 1910, and log-linearly interpolate for the intervening years (and extrapolate from 1911 to 1914). This allows us to compute m-type mortality rates for each municipality and year:

$$m_{xmt} = \frac{\theta_{xmt}}{P_{xmt}}, \tag{A.4}$$

where x denotes age, m indicates municipality, and t represents the calendar year. Since we calculate mortality rates for each municipality and year, the population count for some ages can be zero, particularly in smaller municipalities and at older ages. When this occurs, it leads to missing values in Eq. (A.4), and the summation of life years will stop when computing the life table. To mitigate this issue, we stop the life-table calculations at age 80 (further details below). Additionally, if the population count at age x is zero and the number of deaths is also zero, we assume $m_{xmt} = 0$.[42]

In the next step, we calculate the q-type mortality rates as:

$$q_{xmt} = \frac{m_{xmt}}{1 + (1 - a_{xmt})m_{xmt}}, \tag{A.5}$$

where a_{xmt} is the average age of death at each age x. For $x > 1$, this is set to 0.5 (i.e., for those who die, say, at age 5, we assume that they lived for half a year). For the first year of life, this is set to 1/3 (i.e., those who die as infant—before turning 1—lived on average 4 months), as deaths during the first year of life are typically not equally distributed across the calendar year, but rather concentrated around the first months of life. These weights (a_{xmt}) are assumed not to vary across municipalities and time.

In principle, we could "close" the life table by assuming that everyone dies in the age interval 80-100 (i.e., $q_{80-100mt} = 1$) and calculate:

$$a_{81-100mt} = \frac{1}{m_{81-100mt}}. \tag{A.6}$$

[42] This is one additional reason as to why we construct abridged life tables, where such occurrences are less likely.

This would be a standard way of closing a life table. The problem with this approach, in our context, is that in some municipalities for some years $m_{80-100mt} = 0$ (or it could be a very small number), which would make life-years lived (per person) in the final age group go to infinity (or become a very high number). Thus, as already indicated, we stop the life table at age 79 and if $a_{79mt} = 0.5$ (as we assume), the number of life-years lived (per person) in the final age is always 0.5 and maximum life expectancy at birth is 79.5. We obtain very similar results if we close the life tables in the more standard way, following the approach outlined in Eq. (A.6). In addition, when we aggregate to four areas in Massachusetts by quartiles of treatment, we continue the life-table calculation up to age 99 and close by assuming that life-years lived per person at $x = 100$ is equal to 0.5, as here we do not have the "small-area problem".

Now, we are ready to set up the life table. The number of deaths during age x is:

$$d_{xmt} = q_{xmt} l_{xmt}, \tag{A.7}$$

where l_{xmt} is the number of people alive in the beginning of age x. We set $l_{0mt} = 1$. This means that the size of the hypothetical cohort is normalized to 1 for all years and all municipalities, which is just a normalization and any other number could have been used without influencing the calculation of life expectancy. The number of life/person-years lived between age x and age $x + 1$ is:

$$\begin{aligned} L_{xmt} &= a_{xmt} l_{xmt} + (1 - a_{xmt}) l_{x+1mt},\ x \leq 78, \\ L_{xmt} &= a_{xmt} l_{xmt},\ x = 79, \end{aligned} \tag{A.8}$$

where we see that the maximum age is 79 and all people are assumed to die in that age.

Next, the total number of person-years lived after age x can be computed as:

$$T_{xmt} = \sum_{u=x}^{79} L_{xmt}. \tag{A.9}$$

Finally, life expectancy at different age can be calculated as:

$$e_{xmt} = \frac{T_{xmt}}{l_{xmt}}. \tag{A.10}$$

Life expectancy at birth and age 1 are thus calculated as:

$$\begin{aligned} e_{0mt} &= \frac{T_{0mt}}{l_{0mt}} \\ e_{1mt} &= \frac{T_{1mt}}{l_{1mt}}. \end{aligned} \tag{A.11}$$

Thus, for life expectancy at age 1, we sum all the person-years lived from age 1 to the final age-group and scale with the number of people alive at age 1.

A.2.2 The abridged life table

In order to reduce the problem of missing mortality rates when using one-year age groups, we also compute life expectancies from an abridged life table, which uses wider age groups instead of single-year ages. This has the additional advantage that we can use population counts from Haines (2022), which is available for the years (1880, 1885, 1895, 1905, 1915). We only use these population counts up until the census year of 1905 as the tabulated version of the 1915-Census only contains age groups that cannot be harmonized with the previous tabulated state census years. We make log-linear interpolation in-between census years and extrapolate from 1906 to 1914.

We consider the following age groups 0, 1-4, 5-9, .., 75-79. We stop the life table at age 75-79. In part due the difficulties mentioned above, but also because we do have population data above age 80 in a consistent manner from the tabulated state censuses. In this case, this means that life expectancy at birth can maximum take the value 77.5, because everyone dies off (by assumption) in the last age interval, and we assume that deaths are evenly distributed within this age-group, so life years lived in this age interval is on average 2.5

The m-type mortality rates are given by:

$$\begin{aligned} {}_1m_{0mt} &= \frac{{}_1\theta_{0mt}}{{}_1P_{0mt}} \\ {}_4m_{1mt} &= \frac{{}_4\theta_{1mt}}{{}_4P_{1mt}} \\ {}_5m_{xmt} &= \frac{{}_5\theta_{xmt}}{{}_5P_{xmt}}, \end{aligned} \tag{A.12}$$

where the first year of life is treated as a single-year age group (${}_1m_{0mt}$), ages 1 to 4 as a 4-year age group (${}_4m_{1mt}$), and the remaining ages as 5-year groups. This is transformed into q-type mortality rates as:

$$\begin{aligned} {}_1q_{0mt} &= \frac{{}_1m_{0mt}}{1 + (1 - a_{0mt}){}_1m_{0mt}} \\ {}_4q_{1mt} &= \frac{4m_{xmt}}{1 + 4(1 - a_{1mt}){}_4m_{1mt}} \\ {}_5q_{xmt} &= \frac{5m_{xmt}}{1 + 5(1 - a_{xmt}){}_5m_{xmt}} \end{aligned} \tag{A.13}$$

where ${}_1a_{0mt} = 1/3$ and the remaining $a's$ are set to $1/2$.

The number of deaths are now indicated by ${}_1d_{0mt}$, ${}_4d_{1mt}$ and ${}_5d_{xmt}$ and the number of

person-years lived between two ages are:

$$
\begin{aligned}
{}_1L_{0mt} &= a_{0mt}l_{0mt} + (1 - a_{0mt})l_{4mt} \\
{}_4L_{1mt} &= 4\left(a_{xmt}l_{1mt} + (1 - a_{xmt})l_{4mt}\right) \\
{}_5L_{xmt} &= 5\left(a_{xmt}l_{xmt} + (1 - a_{xmt})l_{x+5mt}\right),\ x \leq 70 \\
{}_5L_{xmt} &= 5\left(a_{xmt}l_{xmt}\right).\ x = 75
\end{aligned} \tag{A.14}
$$

Life expectancies at birth and age 1 can thus be calculated as:

$$
\begin{aligned}
e_{0mt} &= \frac{{}_1L_{0mt} +{}_4 L_{1mt} + \sum_{i=x}^{75} {}_5L_{imt}}{l_{0mt}} \\
e_{1-4mt} &= \frac{{}_4L_{1mt} + \sum_{i=x}^{75} {}_5L_{imt}}{l_{1mt}}
\end{aligned} \tag{A.15}
$$

A.2.3 Limitations

There are several limitations in how we calculate life expectancy for all municipalities in Massachusetts each year. First, we rely on two different data sources to estimate age-specific mortality rates: death certificates and federal/state census data. However, the federal/state population is only measured every 10 or 5 years, which introduces considerable uncertainty in the interpolated population estimates by age, especially for smaller areas. When the population for a specific age group in a given area is estimated to be zero but deaths are recorded in that category, the age-specific mortality rate is set to missing. If this occurs at relatively early ages, it can bias life expectancy estimates downward, making them artificially short.

Another problem is related to small populations tend to have more variability in mortality rates, making estimates less stable and more susceptible to random fluctuations. In particular, in some calendar years, there might be very few deaths, while in other years many (relative to population size). The few death years will make life expectancy too high and vise versa.

As discussed in previous subsections, we address these limitations in several ways. First, we truncate the life table at age 79, assuming that individuals who reach this age live an additional half year, capping life expectancy at 79.5 years. Second, we observe similar results when using life expectancy from the abridged life table, which is less susceptible to these issues. Third, we group Massachusetts into four larger areas based on treatment intensity, where the smallest population is approximately 100,000 people. This aggregation shows similar descriptive patterns to those observed using the municipality-level data.

A.3 Main data sources:

Annual report of Birth, Marriages and Deaths, (i)

- Publication title: "*39th to the 73th Report(s) to the Legislature of Massachusetts related to the Registrar and Return of Birth, Marriages, and Deaths in the Commonwealth*"
  - Period: annually from 1880 to 1914 (publications years 1881-1915)
  - Variables (by municipality and year):
    1. Causes/diseases: bronchitis, lung tuberculosis, scarlet fever, pneumonia, whopping cough, measles, typhoid, digestive (diarrhea cholera dysentery), apoplexy, and accidents.
    2. Live births
    3. Population counts, reprinted from State and Federal censuses.
  - Reference in text: Vital Statics Report (year) or source (i)
  - Publisher: BOSTON: Rand, Avery, & Co., Printers to the Commonwealth, 117 Franklin Street.

Individual Death Records, (ii)

- Publication title: "Massachusetts Deaths, 1841-1915, 1921-1924."
  - Period: annually from 1880 to 1914 (publications years 1841-1915 and 1921-1924)
  - Variables (by municipality, death year, and death age):
    1. aggregated death counts
    2. infant mortality rate
    3. child mortality rate (age 1-4)
  - Reference in text: individual death records (certificates)
  - Publisher: Images. FamilySearch. http://FamilySearch.org. Citing Secretary of State. State Archives, Boston.

Individual Birth Records, (iii)

- Publication title: "Massachusetts Births, 1841-1915."
  - Period: annually from 1880 to 1914 (publications years 1841-1915)
  - Variables (by municipality and year):
    1. aggregated birth counts and estimated population at age 1-4
    2. infant mortality rate
    3. child mortality rate (age 1-4)
  - Reference in text: individual birth records (certificates)
  - Publisher: Images. FamilySearch. http://FamilySearch.org. Citing Secretary of State. State Archives, Boston.

Annual report of the State Board of Health of Massachusetts, (iv)

- Publication title: "*27th to 46th Annual Report(s) of the State Board of Health of Massachusetts*"
  - Period: annually from 1895 to 1914 (publications years 1896-1915)
  - Variables (by municipality and year):
    1. Supply of antitoxin bottles .
    2. Case counts for diseases: diphtheria, typhoid, measles, smallpox.
    3. Water from public works (year of introduction).
  - Reference in text: SBH (year) or source iii
  - Publisher: Boston: Wright & Potter Printing Co., State Printers, 32 Derne Street.

Full-count Federal Censuses, (i)

- Publication titles: "IPUMS Ancestry Full Count Data: Version 4.0"
  - Period: census years of 1880, 1900, 1910 (publication years are 1850, 1860, 1870, 1880, 1900, 1910 ,l920, 1930, 1940, 1950)
  - Variables: (by municipality and census year)
    1. total population in census years
    2. age-specific population and population for certain age groups (including age 0, 1-4, 5-10, 11-14)
  - Reference in text: full-count censuses
  - Publisher: Steven Ruggles, Matt A. Nelson, Matthew Sobek, Catherine A. Fitch, Ronald Goeken, J. David Hacker, Evan Roberts, and J. Robert Warren. Minneapolis, MN: IPUMS, 2024. https://doi.org/10.18128/D014.V4.0

Massachusetts State Censuses, (vi)

- Publication titles: "Massachusetts State Censuses, 1855-1915"
  - Period: 1885, 1895, 1905, and 1915 (publication years: every ten years between 1855 and 1915)
  - Variables: (by municipality and census year)
    1. total population in census years (also used to interpolate annual total population)
    2. population for certain age groups (age 0, 1-4, 5-10, and 11-14)
  - Reference in text: Massachusetts state censuses
  - Publisher: Haines, Michael R. Massachusetts State Censuses, 1855-1915. Inter-university Consortium for Political and Social Research [distributor], 2022-03-09. https://doi.org/10.3886/ICPSR38179.v1

- Original Reference:
  1885: Massachusetts. Bureau of statistics of labor. The census of Massachusetts: 1885. Prepared under the direction of Carroll D. Wright, chief of the Bureau of statistics of labor...Boston, Wright & Potter printing company, state printers, 1887-1888. 3 v. in 4;
  1895: Massachusetts. Bureau of statistics of labor. Census of the Commonwealth of Massachusetts, 1895. Prepared under the direction of Horace G. Wadlin, chief of the Bureau of statistics of labor... Boston, Wright & Potter printing co., state printers, 1896-1900. 7 v.
  1905: Massachusetts. Bureau of statistics of labor. Census of the Commonwealth of Massachusetts, 1905. Prepared under the direction of the chief of the Bureau of statistics of labor... Boston, Wright & Potter printing co., state printers, 1908-1910. 4 v.
  1915: Massachusetts. Bureau of statistics. The decennial census, 1915. Taken under the direction of Charles F. Gettemy, Director of the Bureau of statistics. Boston, Wright & Potter printing co., state printers, 1918. Ix, 749 p.

Linked individuals between Censuses, (vii)

- Publication titles: "The Census Tree, 1900-1940"
  - Period: 1900 and 1940 Census
  - Variables: (at linked individual level)
    1. school absence rate
    2. years of schooling, observed in the 1940 Census
    3. labor market outcomes (occupational categories as white-collar, blue-collar unskilled, or skilled; occupational income score, and wage), observed in the 1940 Census.
  - Reference in text: Census Tree Project/Links
  - Publisher: Price, Joseph, Buckles, Kasey, Haws, Adrian, and Wilbert, Haley. The Census Tree, 1900-1940. Ann Arbor, MI: Inter-university Consortium for Political and Social Research [distributor], 2023-08-11. https://doi.org/10.3886/E193262V1

A.4 Variable definitions and remarks:

"**Population**" ≡ municipality population size

- The baseline population measure is obtained from Federal and State Census enumerations.
- These counts are available every fifth year from 1880 to 1915 by municipality and reprinted in Source (i) from where we digitized them.
- We apply log-linear interpolation in-between census years.
- Population size in 1895 from this sources is used as weight in the weighted regressions.
- We also derive population-by-age counts from the complete count U.S. census records (1880, 1900, 1910) from Jonas Helgertz and Sobek (2023) and apply log-linear interpolation in-between these Federal census years. This source is used in the single-year life table construction.
- We also use population-by-age-group counts from Haines (2022). These are available, for our purpose, 1880, 1885, 1895, and 1905. We apply log-linear interpolation in-between these years. This source is used in the abridged life-table construction and for the age composition variables.
- "**diphtheria all**" ≡ diphtheria mortality rate (for both sexed), defined as diphtheria (and croup) deaths per 1,000 people.
  - Nominator: death counts obtained from Source (i).
  - Denominator: baseline population variable.
  - Variation: municipality-by-year from 1880 to 1914.
  - In the main text used in: Table 2 and Figures 3, 4, 5.
  - *Note: all cause-specific mortality rates have been derived like this and from these two sources.*
- "**treatment**" ≡ average diphtheria mortality rate from 1888 to 1894.
- "**life exp all**" ≡ life expectancy at age 1 for both sexes.
  - In the baseline, derived from the single-year life tables.
  - This life-table type uses data from Source (ii) and the Federal Census population data (Source iv).
  - Variation: municipality-by-year from 1880 to 1914.
  - In the main text used in: Table 2 and Figures 3, 4, 5.
  - We also construct this variable by sex ("life exp female" & "life exp male")
  - We also construct life expectancy at birth from this source. See estimates in Appendix Figure A.14 ("at birth").

  - We also construct life expectancy at age 1 using place of residence instead of place of occurrence, but only up to 1905, due to data availability. See estimates in Appendix Figure A.14 ("residence").
  - We also derived life expectancy at age 1 using the abridged table, which uses population counts by age-group data from the State Censuses (Haines 2022). See estimates in Appendix Figure A.14 ("abridged").
  - See further details in Appendix A.2.

- "**antitoxin p.c.**" ≡ number of antitoxin bottles per 1,000 people
  - Nominator: bottle counts are obtained from Source (iii).
  - Denominator: baseline population variable.
  - A municipality not listed in a given report is assumed to received zero bottles that year.
  - Municipalities never listed with bottles during the sample period 1895 to 1914 are dropped from the sample.
  - Distribution years do not match perfectly with the calender year. We assign the distribution year to the calender year with the most overlaps in terms of months. See Appendix Table A.8 for the exact distribution years.
  - In the baseline, antitoxin p.c. has been winsorized, using cuts(0 95) with "winsor2" in STATA.
  - Appendix Figure A.2 reports the distribution of antitoxin p.c. with and without winsorizing, and it documents that that winsorizing improves the first-stage fit due to large values of antitoxin to hospitals.
  - Variation: municipality-by-year from 1880 to 1914. (by definition zero before 1895).

- "**infant rate**" ≡ infant mortality rate, defined as death below the age of 1 per 1,000 live births
  - Nominator: death counts by age obtained from Source ii.
  - Denominator: Live-birth counts obtained from vital statistics.
  - We also construct this variable by sex ("infant rate female" & "infant rate male").
  - Variation: municipality-by-year from 1880 to 1914.
  - In the main text used in: Table 3.
  - See further details in Appendix A.1.

- "**child rate**" ≡ child mortality rate, defined as death between ages 1 and 4 per 1,000 children of this age-group
  - Nominator: death counts by age obtained from Source ii.

  - Denominator: we use death and birth from vital statistics to calculate the size of the age group 1-4.
  - We also construct this variable by sex ("child rate female" & "child rate male").
  - Variation: municipality-by-year from 1880 to 1914.
  - In the main text used in: Table 3.
  - See further details in Appendix A.1.

- "**infec. rate, 88-94**" ≡ average mortality rate (per 1,000 people) from 1889 to 1894 for infectious diseases.
  - Diseases included: bronchitis, lung tuberculosis, scarlet fever, pneumonia, whopping cough, measles, typhoid, and digestive.
  - Variation: municipality.
  - In the main text used in: Table 1.

- "**apoplexy rate, 88-94**" ≡ average mortality rate from apoplexy per 1,000 people from 1889 to 1894.
  - Apoplexy is a historical cause of death and refers to sudden death, which is only the measure of heart/stroke related death during this time period at the municipality level.
  - Variation: municipality.

- "**doctor pr. capita in 95**" ≡ medical doctors per 1,000 people in 1895.
  - Nominator: medical-doctor counts obtained from the Federal Censuses (Source iv) for the census years 1880, 1900, and 1910.
  - Denominator: baseline population variable.
  - Variation: municipality.

- "**dist Boston** is aerial distance to Boston.
  - Calculated using "geodist" in STATA based on longitude and latitudes for each municipality.

- "**person pr 1,000 sqm in 95**" ≡ number of people in 1895 per 1,000 square miles
  - Nominator: baseline population variable, measured in 1895.
  - Denominator: area of municipality obtained in 1915 (Haines 2022).
  - Variation: municipality.

- "**person pr dwelling in 95**" ≡ number of people per dwelling houses in 1895
  - Nominator: baseline population variable, measured in 1895.

  - Denominator: total number of dwelling houses in 1895 (Haines 2022).
  - Variation: municipality.

- "**person pr room in 95**" ≡ number of people per number of room in dwelling houses in 1895
  - Nominator: baseline population variable, measured in 1895.
  - Denominator: total number of rooms in dwelling houses in 1895 Haines (2022).
  - Variation: municipality.

- "**fb share in 95**" ≡ number of foreign born people per population in 1895
  - Nominator: baseline population variable, measured in 1895.
  - Denominator: total number of foreign born from State Census in 1895 Haines (2022).
  - Variation: municipality.

- "**diph ratio**" ≡ the number of diphtheria (and croup) death per 1,000 deaths
  - Nominator and denominator: both death counts are obtained from Source (i).

- "**case-fatality**" ≡ the number of diphtheria (and croup) death per 1,000 cases of diphtheria
  - Nominator: baseline population variable, measured in 1895.
  - Denominator: case counts are obtained from Source (iii).
  - Due to case data availability, this variable is only available from 1891 onward.
  - Municipalities with values greater than 1,000 is truncated at 1,000.
  - Variation: municipality-by-year from 1891 to 1914.

- "**apoplexy**" ≡ mortality rate from apoplexy per 1,000 people.
  - Apoplexy is a historical cause of death and refers to sudden death, which is only measure of heart related death during this time period.
  - Variation: municipality-by-year from 1880 to 1914.

- "**infec rate**" ≡ mortality rate from infectious diseases per 1,000 people
  - Diseases included: bronchitis, lung tuberculosis, scarlet fever, pneumonia, whopping cough, measles, typhoid, and digestive.
  - Variation: municipality-by-year from 1880 to 1914.

- "**accidents**" ≡ mortality rate from accidents per 1,000 people.
  - Nominator: death counts from Source (i).

  - Denominator: baseline population variable.
  - Variation: municipality-by-year from 1880 to 1914.

- "**birth rate**" ≡ crude birth rate, defined as the number of live birth per 1,000 people.
  - Nominator: birth counts from Source (i).
  - Denominator: baseline population variable.
  - Variation: municipality-by-year from 1880 to 1914.

- "**Doctors**", "**Nurses**", & "**Pharmacists**" ≡ as the number of people in the given occupation per 1,000 people.
  - Nominator: occupation counts from the Federal Census (Source iv).
  - Denominator: baseline population variable.
  - The rate variables have interpolated in-between census years.
  - This is also calculated, where the occupation counts have divided into the age-groups (20-29, 30-39, 40-49, and 50-59).
  - Variation: municipality-by-year from 1880 to 1914.

- "**Water intervention**" ≡ roll-out variable, based on the establishment date of public water works.
  - Records the establishment date of public water works.
  - Source: SBH (1930).
  - Variation: municipality-by-year from 1880 to 1914.

- "**Infectious hospital**" & "**General hospital**" ≡ roll-out variable, based on the establishment date of an infectious disease or a general hospitals.
  - Records the establishment date of the hospital by type (infectious or general hospital).
  - Source: Benevolent Institutions, 1910. Department of Commerce, Bureau of the Census.
  - Variation: municipality-by-year from 1880 to 1914.

- "**Age composition**" ≡ population shares for the age groups 0, 1-4, 5-9, and 10-14.
  - Population share are obtained using data from Haines (2022).
  - Variation: municipality-by-year from 1880 to 1914.

A.5 Historically Consistent Municipality Boundaries

During our primary sample period (1880-1914), the county boundaries in Massachusetts underwent several changes due to the separation or annexation of municipalities (towns). To construct a balanced and comparable panel, we adjusted the year-by-year town boundaries as follows:

(1) If municipality A was separated from municipality B and incorporated between 1876 and 1914, we combined the two municipalities and aggregated them into municipality B. We extend the adjustment period back to 1876 to account for lagged births and deaths between 1876 and 1880, which are used to estimate child mortality rates for ages 1-4 in 1880.

(2) If municipality A was incorporated before 1876 but annexed into municipality B between 1876 and 1914, we also combined the two municipalities and aggregated them into municipality B.

Below is a complete, chronologically ordered list of municipality boundary changes that occurred during the sample period:

- 1876: Merrimac was separated from Amesbury and incorporated as an independent town.
- 1878: North Adams was separated from Adams and incorporated as an independent town; Hampden was separated from Wilbraham and incorporated as an independent town.
- 1880: Cottage City was separated from Edgartown and incorporated as an independent town. It was later renamed Oak Bluffs in 1907.
- 1881: Wellesley was separated from Needham and established by the Massachusetts legislature.
- 1884: Bourne was separated from Sandwich and incorporated as an independent town.
- 1885: Millis was separated from Dedham and incorporated as an independent town.
- 1886: Hopedale was separated from Milford and incorporated as an independent town.
- 1888: Avon was separated from Stoughton and incorporated as an independent town.
- 1897: Bradford was annexed to the city of Haverhill; Westwood (then referred to as "West Dedham") was separated from Dedham and incorporated as an independent town.
- 1905: Plainville was separated from Wrentham and incorporated as an independent town.
- 1912: Hyde Park was annexed into the city of Boston.

A.6 Relation to shift-share like instrument

In this appendix, 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" like instrument). For convenience, we repeat the structure of our baseline instrument here:

$$IV_{mt}^{base} = treatment_m \times I_t \times (t - 1894), \tag{A.16}$$

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

$$treatment_m = d_m^{pre}. \tag{A.17}$$

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

$$IV_{mt}^{alt} = B_t \frac{D_m^{pre}}{D_{MA}^{pre}} \frac{1}{P_m^{94}}, \tag{A.18}$$

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:

$$treatment_m = \frac{D_m^{pre}}{P_m^{94}}, \tag{A.19}$$

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.19) as treatment intensity for the baseline instrument provides very similar

results (available upon request). Next, we substitute this into Equation (A.16) and rearrange:

$$IV_{mt}^{base} = \frac{D_m^{pre}}{P_m^{94}} \times I_t \times (t - 1894) \Leftrightarrow$$
$$D_m^{pre} = \frac{P_m^{94} IV_{mt}^{base}}{I_t \times (t - 1894)}, \tag{A.20}$$

which we combine with Equation (A.19) to give:

$$IV_{mt}^{alt} = B_t \frac{\frac{P_m^{94} IV_{mt}^{base}}{I_t \times (t-1894)}}{D^{MA}} \frac{1}{P_m^{94}} \Leftrightarrow$$
$$IV_{mt}^{alt} = \frac{B_t}{D^{MA} \times \tau} IV_{mt}^{base}, \tag{A.21}$$

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 Appendix Figure A.14 as the specification "shift-share IV".

A.7 Determinants of the antitoxin diffusion

In this appendix section, we provide a more detailed description of on 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). The estimates are reported in Table A.2. 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 antitoxin (in 1904), the adoption rate is 1.49 bottles per 1,000 people in a municipality with average treatment intensity ($0.35 * 10 * 0.43$) and the range goes from 0 to 7.01 bottles when moving from zero to maximum treatment ($0.35 * 10 * 2.01$). This is our baseline first-stage specification, which is used in Table 2

In the remaining columns, we add the different pre-antitoxin characteristics, also interacted with the indicator and year trend. In column 6, where all controls are included at the same time, we see that the most robust determinants of the adoption of antitoxin is the instrument, along with the number of doctors per capita, distance to Boston, population density, and the opening of new infectious disease hospitals, in particular.

Using a decomposition exercise following Gelbach (2016), we find that population density, dwelling size, and doctors per capita (all interacted with a linear trend) are the key factors driving the decrease in the first-stage coefficient when robustness controls are added (Appendix Table A.3)