Preventing the White Death: Tuberculosis Dispensaries*

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Abstract

Tuberculosis (TB) is a leading cause of death worldwide and while treatable by antibiotics since the 1940s, drug resistant strains have started to emerge. This paper estimates the effects of the establishment of a pre-antibiotic public health institution, known as the TB dispensary designed to prevent the spread of the disease. Our difference-in-differences estimation reveals that the rollout of the dispensaries in cities in Denmark led to a 16 percent decline in the TB mortality rate, but no significant impacts on other diseases when performing placebo regressions. We next take advantage of the dispensaries explicit targeting on TB to setup a triple-differences model which exploits other diseases as controls and obtain a very similar magnitude of the effect. As for the mechanism, the evidence highlights the dispensaries' preventive actions, such as facilitating a local diffusion of (hygiene) knowledge about the disease. At an estimated cost of 76 dollars per saved life-year, this particular public health institution was extraordinarily cost effective. In addition, we find small positive spillover effects of the dispensaries on productivity, as measured by annual income per tax payer at the city level, digitized from historical tax-assessment records.

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1 Introduction

Differences in mortality across space and time are "phenomena worthy of serious attention by economists" (Cutler et al., 2006, p.7). The present research is motivated by this observation and focuses on the role of dissemination of knowledge about diseases and isolation as ways of reducing mortality. In particular, we study a pre-antibiotic, local health institution—known as a tuberculosis (TB) dispensary—with the primary purpose of preventing the spread of TB and provide empirical tests on whether the dispensaries in Denmark actually were successful in reducing TB mortality and how they otherwise influenced society at large.[1]

According to the World Health Organization (2015), TB is a major global health problem and ranks alongside HIV as a leading cause of death. As of 2014, there were 9.6 million new TB incidences, and even if it has been treatable by antibiotics since the 1940s, drug resistant strains now exist. It is therefore unsurprising that eliminating TB mortality is part of the UN's third sustainable development goal. While presently, TB is mainly confined to developing countries, before the advent of modern medicine, European countries and the US, however, also suffered from high rates of TB incidences and deaths.[2] Moreover, there was considerable variation between these areas with some countries being able to substantially reduce TB in the pre-antibiotic era. Daniels (1949, p.1066), for example, observes that the TB death rates fell in many European countries from above 200 per 100,000 in 1885 to below 100 in 1935. He points out that "the most striking fall was in Denmark; the rate there was one of the highest recorded in Europe in 1885, with a mortality of nearly 300, and in 1935 it was below 50", which is where our quasi-experiment unfolds. Schelde Møller (1950) ascribed this decline to the policies pursued in Denmark, which were instigated by the *National Association for the Fight Against Tuberculosis*. Among other public health measures, the National association established TB dispensaries locally which were rolled out across time and space differentially. To our knowledge, this paper is the first to quantitatively evaluate the implications of establishing this particular

[1] Tuberculosis was also known as the "the White Death" as used in the title. Other names include "the great white plague", "the robber of young", "The Captain of all these men of Death", Frith (2014). "The White Death" is also the title of Dormandy's 1999 book on tuberculosis.

We view infectious diseases, in our case TB, as "quintessential manifestations of the principle of an externality, itself a central concept in economics" (Gersovitz and Hammer, 2004, p.1).

[2] The TB mortality rate in the US fell from above 200 in 1900 to circa 60 per 100,000 in 1935 (Cutler and Meara, 2004).

TB institution, which more broadly allow us to consider the effectiveness of information and isolation as policy instruments to reduce mortality in a society.

The role of the dispensaries was to prevent the spread of the disease. Doctors would refer TB infected patients to the dispensaries, which would provide help, support and examination of the infected individual and his or her family. They also attempted to ascertain how the patient contracted the disease and whether others had contracted the disease because of contact with the patient. The dispensaries themselves were little more than a room used for linen, towels, disinfectants, and spittoons (Schelde Møller, 1950). All doctors should be able to refer patients to the dispensary, which was either led by a specialized doctor or nurse.[3] Writing in the 1940s, Holm (1946a, p.1429) summarizes the role of the TB dispensaries: "The known cases of tuberculosis, especially the particularly infectious, are under the observation of the tuberculosis dispensary, partly through frequent examinations of the patients in the dispensary and partly by visits of nurses to homes. The patients are given instruction to prevent the spread of infection. The tuberculosis dispensary provides the patient with a glass for sputum, sometimes bed clothing and laundry service. Occasionally, if required, the dispensary procures better dwellings for the patients."

Our analysis estimates the impact of the TB dispensaries on TB mortality for Danish cities covering the period 1890-1939. The first strategy employs difference-in-differences estimation in which the impact of TB dispensaries is estimated using the rollout over time and across cities. The effect on TB mortality is negative and statistically significant, while performing a number of placebo tests reveal no impact on scarlet fever, pneumonia, accidents and suicides, diphtheria, cancer, polio, and influenza. Yet, the analysis indicate that overall and child mortality were negatively affected, albeit these effects are substantially smaller compared to the direct effect on TB as expected. Our second strategy follows a triple-differences setup which exploits that the dispensaries were explicitly targeted at *only* preventing TB and assigns the aforementioned diseases (i.e., causes of death) as controls.[4] This experiment compares the development of TB mortality to the control diseases before and after the introduction of a dispensary in a given city.

[3]See also Høy-Nielsen (2012) for an excellent description of the development of TB in the first half of the 20th century, based on the working of a TB dispensary in Ribe county.

[4]The triple-differences model is also known as the difference-in-difference-in-differences model, see Wooldridge (2010, chapter 6).

Reassuringly both strategies result in very similar estimates, which suggest that the dispensaries reduced the TB mortality rate with around 16 percent.

For both strategies, we perform a number of robustness tests: For example, we have checked for differences in pretrends by estimating event-studies which compute the average effect of having a dispensary in the 10 years before and after implementation. These estimates show that the impact of the dispensaries are systematically negative only after their introduction and no effects in the years before. These common pretreatment trends, therefore, provide indirect support for our main identifying assumptions. Furthermore, random placebo tests, which randomly reassigns the intervention dates and then reestimate the models, show that our baseline estimates are in the very left tale of the resulting distributions, and along the same lines, estimating models which allow effects from the opening of dispensaries in nearby cities suggests a very limited role for spillover effects. Along with a battery of other robustness checks, we also show that the results are *not* driven by, e.g., pre-intervention population size, pre-intervention income, or pre-intervention inequality.

We also provide evidence on the exact mechanism by which the dispensaries reduced TB mortality, which, according to their policies, could happen in two ways. First, as also described above, the TB doctor at the dispensary would inform the infected individual about how to avoid spreading TB, and nurses would make home visits to assure that such guidelines were followed. We refer to this particular mechanism as the dissemination of (hygiene) knowledge/information about the disease. Second, the dispensaries would sometime direct infected individuals to either TB hospitals or sanatoria, which effectively means that they were isolated from the non-infected population (i.e., an isolation mechanism). In order to evaluate the relative importance of these two mechanisms, we collected an annual dataset with the number of patients in TB hospitals and sanatoria, and since we also know the former addresses of these institutions, it is possible to test what happens to the number of institutionalized TB patients when the dispensaries are being rolled out in their local attachment areas. We find a negative effect on the number of sanatoria patients, suggesting that the knowledge-about-disease mechanism is the most important one.[5]

[5]If the effect was only about isolation, one would expect to see an increase in the number of isolated patients; see also our discussion in the theoretical framework section. Also note that we demonstrate that our baseline findings are not con-founded by the spread of these two other types of TB institutions, and we do no find any evidence suggesting that they by themselves reduced TB mortality in Denmark, which stands in contrast to Hollingsworth (2014), who show that sanatoria in North Carolina reduced TB mortality among the white

Finally, we estimate the economic impacts of the dispensaries. First, we have collected data on the cost of the intervention and find that the cost of saving a life year is around 76 dollars, which compared to previous studies that evaluate the cost effectiveness of other types of health interventions (e.g., Cutler and Miller, 2005; Bailey and Goodman-Bacon, 2015), makes the dispensaries extraordinarily cost effective. Second, we collected and digitized annual tax income data at the city level from historical tax-assessment records and find small positive spillover effects of the dispensaries on productivity (as measured by income per tax payer). More concretely, we show the introduction of a dispensary increased annual income per tax payer growth by about 0.4 percentage points. This final finding supports recent research suggesting that eradicating TB is important for human capital accumulation and other economic outcomes; see, e.g., Hansen (2013), and Bütikofer and Salvanes (2015).[6] Also, circumstantial evidence suggests that TB had important economic consequences in the US: the US Bureau of Labor (1912) provides a back-of-the-envelope calculation of the loss for wage earners in terms of earning and finds that the losses amount to roughly 214 million US dollars in 1910 (about 0.6 percent of GNP).[7]

Using data for England and Wales, McKeown (1976) noted that 80 percent of the reduction in TB mortality happened before there was any effective medical treatments and generally argued that the declines in mortality of diseases such as TB, dysentery, cholera, etc. were unrelated to any public-health measures. This view has gained support by Fogel (1994; 1997), however, other prominent scholars, such as Preston (1975;[8] 1996), Szreter (1988), and Cutler et al. (2006), highlight the important role of various public-health interventions, which were set in motion by the germ theory of disease in the 1880s.[9] While recent research has shown that sanitation and clean-water supply were important drivers of the initial mortality decline in the

population, using an IV approach for identification. Adda (2016) shows that policies reducing interpersonal contacts reduces the spread of virues. Yet, they are found not to be cost-effective.

[6]See Weil (2014) for a comprehensive overview of the literature that studies the effect of health improvements on productivity.

[7]Evidence from present day Ecuador suggests that people getting the drug resistant strain are likely to earn less than 100 dollars per month due to disability (Rouzier et al., 2010).

[8]In Preston's seminal work of 1975, he argues that the upward movement of what has later been named the Preston curve (i.e. the life expectancy-income relationship) is due to public health investments and health technological progress.

[9]See also Deaton (2006)'s summary of the discussion between McKeown and Fogel on the one hand and Preston, Szreter (1988), Cutler and Miller on the other.

US (Cutler and Miller, 2005; Alsan and Goldin; 2015; Ferrie and Troesken; 2008), it seems as if relatively little is known about how (and by how much) public health in the form of distribution of information about diseases has contributed to the historical mortality decline, which is one of the main contributions of our paper.

Studying the impact of TB dispensaries in Denmark prior to the onset of modern medicine provides the advantage that any estimated effect cannot be confounded by provision of effective medicine. Moreover, as also noted by Williamson (1910), such TB dispensaries spread across Europe (and the US) in the start of the 20th century, and so in this way, this type of institution was not unique to Denmark.[10] The Danish case also provides the advantage that data are available on a large number of diseases and the opening dates of the dispensaries, in a setting in which national institutions cannot confound the results.

There are several reasons as to why studying the impact of TB dispensaries is interesting. First, they present an example in which public health institutions may have mattered prior to the advent of modern medicine contrary to what has been argued by McKeown (1976), Fogel (1994), and others. While we focus on the role of TB dispensaries, it is important to notice that we do not rule out that improved nutrition as emphasized by McKeown and Fogel could also have been important. Second, the dispensaries represent a clear illustration of a government response to the germ theory, which also has policy relevance today in a developing-country context, as they suggest a way of combating an externality producing infectious disease, especially because drug resistant TB now exists.[11] Related to this, there is even substantial concern about drug resistant infectious diseases and their future consequences in developed nations as recently pointed out by O'Grada (2015).

The rest of the paper is organized as follows. Section 2 provides historical background on the TB dispensaries and discusses other aspects of the Danish policy. Section 3 outlines a theoretical framework. Section 4 describes the data along with the results of balancing tests. Section

[10]The first dispensary was founded in Edinburgh, Scotland in 1887; France got its first dispensary in 1901; Germany in 1904, see Williamson (1910). However, it has been noted that Denmark had a high number of TB institutions in per capita terms (Lawrence, 2006, p. 340).

[11]Building on Gersovitz and Hammer (2004, p.3), we note that an infection externality arises, if when choosing their level of preventive effort, people do not fully take into account the costs to others who will become infected as a consequence of their being infectious. The dispensaries would arguably reduce the private cost of taking proper preventive measures as patients and relatives were instructed on how to avoid contagion so as to choose levels of preventive effort, which are closer to the social optimum.

5 explains the empirical strategies. Section 6 provides results, including robustness analyses. Section 7 provides evidence on the mechanism. Section 8 evaluates the economic consequences of the dispensaries. Finally, Section 9 concludes.

2 Historical background

This section explains the historical background and TB-targeted health policies used in Denmark before the advent of antibiotics and vaccination. We first describe the medical situation prior to modern medicine and the historical development of TB mortality in Danish cities over time. Second, we describe the intervention of interest: the rollout of the TB dispensaries. Third, we describe other policy measures taken against TB.

2.1 TB in Denmark, 1890-1950

TB is caused by bacteria of the Mycobacterium tuberculosis complex as discovered by Robert Koch in 1882. The most common type of TB occurs in the lungs—pulmonary TB—but TB can also affect other organs. Transmission of TB is by inhalation of infectious droplet nuclei containing viable bacilli, known as aerosol spread. Mycobacteria-laden droplet nuclei are formed when a patient with active pulmonary TB coughs and can remain suspended in the air for several hours. Sneezing or singing may also expel bacilli. The most frequent symptom is a non-remitting cough, which occurs in 95 percent of the cases. Many cases also include fever, nightsweat and weight loss. The TB bacteria has existed for a long time with the most conservative estimates being that it is 6,000 years old, and it has caused more deaths than any other disease during the last 200 years (Hemskerk et al., 2015).

In the Danish case, TB mortality rates were high in the late 19th century and early 20th century. Importantly for our study, the first antibiotics effectively treating TB, streptomycin, was not invented until the 1940s. Moreover, the vaccine against TB, the Bacillus Calmette-Guerin (BCG) vaccine, was not applied systematically until the 1940s for the whole country, and only at the remote island of Bornholm from 1936-40 prior to systematic use across the country (Holm, 1946b).[12] In fact, Holm mentions that BCG was carried out as an experiment of limited extent

[12]We perform robustness checks in which we exclude Bornholm as well as the period 1936-39 in our baseline

as it was only given to a few newborn children.[13] It should also be noted that the evidence on the effectiveness of the vaccine in stopping transmission is highly variable. According to Hemskerk et al. (2015) "BCG is the most widely used vaccine in the world but measures of effectiveness have varied widely, between 0 and 80 percent. Studies have however, consistently shown a protective effect against the most severe forms of childhood TB, including TB meningitis." This is corroborated by Nelson and Wells (2004), who note that meta-studies suggest that the protective effect is about 50 percent against pulmonary TB. Moreover, protection is stronger for e.g. TB meningitis, which children are more likely to develop (see below).[14]

Figure 1 shows the development of the TB mortality rate for the Danish city population from 1890 to 1950.[15] There is clearly an overall decline in the mortality of the disease, as also observed by Daniels (1949). Yet, there is a spike around World War I as well as a plateau around World War II. We note that the general pattern of decline is not unique to TB, but also holds for, e.g., pneumonia; see Online Appendix Figure A5. This suggests that there are common causes behind the decline in mortality, such as improved nutrition, wars and, for the late 1940s, modern medicine, which indicates the importance of controlling for time as well as other fixed effects in our empirical specifications.

Figure 1: TB mortality in the Danish cities, 1890-1950

Data also show that TB mortality and morbidity are declining simultaneously during this period. For the years 1921 to 1949, Schelde Møller (1950) provides data on mortality as well as

sample.

[13]Holm (1946b) mentions that the first BCG strain was received by the Danish Serum Institute in 1927. The strain was used very cautiosly, which was good since it led to complication in vaccinated children. He notes that 16 children were vaccinated in 1927 in Copenhagen, 7 in 1928, and 22 in 1929. As the strain used initially was "too potent" and in the 30s a new strain was used. Holm also gives data from 1936-1941 for the central TB dispensary in Copenhagen. In 1936, the number vaccinated was 82 and in 1939 when we end our sample it was less than 500. The annual reports from the TB dispensaries only mention vaccination for Frederiksberg from 1936. As mentioned below the program on the small island of Bornholm was much larger. As also noted below, we have checked whether these places drive our results, and they do not.

[14]Dormandy (1999, p. 347-349) reviews some of the early evidence on BCG. He notes that evidence from Gothenburg in Sweden indicated that BCG vaccinated children "developed primary tuberculosis four to ten times less often and pulmonary tuberculosis Stage three two to three times less often." (p. 348). In Britain, tests on 56,000 starting in 1949 children showed a pulmonary rate that was lower for the vaccinated children and no vaccinated child developed TB meningitis or military TB. Brimnes (2008) also reviews the evidence and covers the period after 1949. He cites studies that reported a 36 protection rate in Georgia and Alabama in the 1950s. He also cites the the Chingleput trial carried out from the late 1960s in India, which showed no protective effect.

[15]Online Appendix Figure A1 depicts a similar path for pulmonary TB mortality.

morbidity on pulmonary TB for the whole of Denmark, and as revealed by Online Appendix Figure A12, pulmonary TB mortality and morbidity per 1,000 people follow similar downward trends with a correlation of 0.86.

Online Appendix Figure A13 shows the average age distribution of TB mortality for the five year period 1921-25. It shows a wave like pattern with high mortality among infants and young children, with mortality declining to reach a low among the 5 to 15 years old, where mortality starts increasing to reach a peak in the early 20s with declining mortality thereafter. This pattern is consistent with the medical literature which has found that very young children with an immature immune system are at high risk of developing TB when exposed to the infectious bacteria, whereas older children are the least likely, with the risk rising as they meet adulthood (Marais et al., 2005). Furthermore, children are more likely to develop TB outside the lungs, such as military TB and TB meningitis (Nelson and Wells, 2004; Smith et al., 1997).

2.2 TB dispensaries

The National Association for the Fight Against Tuberculosis was established in 1901 and was originally focused on treatment, isolation and patient care. However, in 1906, the secretary of the association went on a field trip to Germany to study the system of dispensaries that was being established there. Following this, the first TB dispensary was opened in Copenhagen in 1908 in a five room apartment, which was funded by a private donor for 10,000 Danish kroner, corresponding to about 645,000 Danish kroner in present value (around 95,000 USD). The dispensaries spread to other cities in Denmark, and they would often be led by specialized doctors, though some were led by nurses. Figure 2 shows the rollout of the TB dispensaries in a series of maps for different time periods. In the period from 1908-1915, dispensaries were established in Copenhagen, Aarhus, and Odense (three of the largest cities), as well as in the smaller cities Vejle and Slagelse. In the period, 1916-1927 a few extra dispensaries were added, but as revealed by Figure 2, it was only from 1928 onwards that dispensaries started covering the whole country. The initial diffusion pattern is to some extent likely to reflect amendments to the TB law of 1905: the first amendment in 1912 states that private institutions focusing on the prevention of TB are eligible for state subsidies, and a second revision in 1919 implied that the dispensaries effectively became public institutions, although they remained organized

by the National Association until 1928. Due to the work of medical director *Johs. Frandsen* the goal hereafter became to have a national wide network of dispensaries, led by specialized TB doctors, such that each county in Denmark should have one main dispensary with branches placed in other cities within the same county (Simonsen, 1947). This process was completed around 1944.[16]

Figure 2: Spread of TB dispensaries across Danish cities

The dispensaries required only a room and the list of items, stated in the introduction, which includes, e.g., linen, towels, and disinfectants. Getting nurses with expertise in TB was initially a problem. This was solved by the National Association by offering specialized courses. From 1918, a cooperation between the association for nurses outside of Copenhagen and the National Association helped alleviate this problem. The lack of properly trained nurses have been suggested as a fundamental reason for the slow initial spread of dispensaries (Permin, 1912; Schelde Møller, 1950).

Before the 1940s, the dispensaries had five different activities that helped prevent the spread of TB. First, local doctors would be responsible for new notifications to the dispensaries. Second, the dispensaries would perform consultations with patients and instruct them on how to avoid transmitting the disease further. Third, the dispensary would supervise a number of homes of TB patients. Fourth, nurses employed at the TB dispensaries would make home visits. Finally, they would direct patients to other TB institutions.

By 1927, 24 percent of the population had access to TB dispensaries, whereas by 1939, 67 percent had access (Medical Reports, 1927; 1939). In 1927, the dispensaries had 3000 new patients and visited 15,000 homes (Medical Report, 1927). In 1939, 33,431 new individuals were referred to the dispensaries, 5,812 homes were supervised, 180,250 consultations were carried out, and nurses undertook 35,288 home visits (Medical Report, 1939). As mentioned, the dispensaries would also refer (some) patients to TB hospitals and sanatoria. For example, in 1935, the aggregate number was 969 patients, which then increases steadily the following years, however, relative to the number of consultations, the number was actually decreasing these years (Medical Reports, 1935-1946). For the 1940s, we also have some information on how

[16]In some cases, the dispensaries were organized at the municipality level.

much personnel the dispensaries were using: in 1943, for example, there were 89 nurses and 60 medical doctors employed at 71 dispensaries.[17]

From the 1940s, vaccination became common as mentioned above. The dispensaries performed vaccinations with statistics being reported systematically from 1943. Thus to avoid confounding our results with any impact of the BCG, we use the period until 1939 as our baseline sample in the empirical analysis below, though results are robust to including additional years.

This subsection concludes by providing graphical evidence on the impact of the dispensaries by graphing average city-demeaned TB mortality rates 10 years before and after the introduction of a TB dispensary in Figure 3.[18] While the TB mortality rate exhibits a downward trend, we see a clear discontinuity around the introduction of a dispensary. This is also illustrated by the red line which shows the linear prediction of the TB mortality rate before and after the dispensary, formed by regressing the average city-demeaned TB rate on a constant, a time trend, an indicator equal to one after the introduction of the dispensary, and the time trend interacted with the indicator. The coefficient on the indicator is -0.145 and is significant at the five percent level (standard error = 0.055), while the coefficient on the trend interacted with the indicator is small and insignificant (coefficient = 0.009; standard error = 0.007). We view this pattern as prima facie evidence that the opening of the dispensaries mattered for the development of TB mortality.

Figure 3: Average city-demeaned TB rate before and after TB dispensary

2.3 Sanatoria and hospitals

A number of TB sanatoria and TB hospitals were also founded during this time period. As with the dispensaries, the introduction of the sanatoria were inspired by German policies. Sanatoria were established around the country from the beginning of the 20th century and were often placed in the vicinity of a larger city, whereas the TB hospitals were placed in a city. The basic idea behind the sanatoria was that patients were given the best conditions for self-healing by

[17] As we note below, a method for diagnosing active TB was available from 1882. Some dispensaries also had x-ray equipment, but this was not common in smaller areas prior to the 1940s; Holm (1946a).

[18] We subtract city means to account for city specific fixed effects. City demeaning the TB rate causes the averages in the graph to appear negative, as most dispensaries were introduced in the 1930s and by this time TB had already fallen substantially relative to 1890; see Figure 1 and Figure 2.

getting fresh air and a balance between physical and mental rest, and work therapy on the other hand (Schelde Møller, 1950). The sanatoria took care of the stronger patients, whereas weak patients were sent to TB hospitals. This practice lasted until the 1940s.

Porter (1999) argues that sanatoria only provided a holiday for their inmates, and that there is little evidence that they mattered for the decline in TB mortality. The medical report for Denmark for 1903 contains circumstantial evidence that backs up this assessment with the chief medical officer of the medical district of Ringsted on Zealand, who had never observed any impact of a stay at a sanatorium. It is further observed in the same report that the sanatoria picks the stronger patients and leave the weaker patients to stay in hospitals in the cities. The medical reports also show that relatively few patients die at a sanatoria. Others such as Winge (1952) emphasize that sanatoria and TB hospitals both provided treatment and isolation and Bureau of Labor (1912) and Hollingsworth (2014) have a more positive assessments of the sanatoria. Our empirical analysis find little evidence that the TB hospitals and sanatoria reduced TB mortality by themselves and demonstrates that possible interactions between TB hospitals, sanatoria, and the dispensaries do not influence our baseline findings. In addition, we find that the dispensaries reduced the number of patients admitted at the sanatoria.

2.4 Information campaigns across the country

Active efforts to prevent the spread of TB by country wide information campaigns were also in place. Signs with "Do not spit on the pavement" printed on them were produced and distributed across cities, though they did not diffuse as much as the National Association had hoped for (Schelde Møller, 1950). From 1918, a poster with a similar message was sent to churches around the country. This type of information was distributed widely across the country, and we trust that time fixed effects will capture these in the empirical analysis. We also note that, in so far as the campaigns were effective, they would also be likely to impact the spread of other airborne, infectious diseases such as pneumonia and scarlet fever as transmission is similar. As mentioned, we control for time fixed effects in all models, and we note that we can control for a tighter set of fixed effects when we use a triple-differences set-up.

3 Theoretical framework

This section outlines a SIR model (Kermack and McKendrick, 1927) which has been modified to fit a pre-antibiotic population and disease environment, as described in the previous background section. We use this theoretical setup to clarify and illustrate how we think the introduction of the dispensaries influenced the development of TB mortality. In our modified model, the total population (P) consists of two sub populations, which we are interested in following: the non-TB infected population (S) and the TB infected population (I). Only individuals with active TB are in I, which implies that latent TB individuals are grouped in S as they are not contagious at this stage. Individuals in S are at risk of being infected and developing active TB and hereby becoming part of I.[19] This risk is denoted by the infection rate, which is endogenous (see below). The other way for individuals to exit S is by dying from other causes than TB. For simplicity, we assume that there is no co-mortality between TB and the other death causes. Individuals in I can either remain infected, die from TB, or recover, so that they again becomes part of the non-TB infected population and, therefore, at risk of contracting TB later, since no immunity against TB is granted after recovery. The chance of recovery is related positively to income (see below). Finally, we assume that all individuals are born into the non-TB infected population. This simplification is not crucial in any way. Note that we do not incorporate any kind of immunization (treatment) instruments into the model, as our empirical analysis investigates the effect of the dispensaries on TB mortality in an environment without these "treatment" possibilities.

These assumptions imply that the non-TB infected population develops according to:

$$S_{t+1} = S_t + B_t - I_t^{New} - D_t^{other} + R_t, \tag{1}$$

where $B_t = bS_t$ is the total number of births, $I_t^{New} = \lambda_t^I S_t$ denotes new TB infections (λ_t^I is the endogenous infection rate), $D_t^{other} = \lambda^D S_t$ is the total number of deaths due to other causes than TB, and $R_t = \lambda_t^R I_t$ is the total number of individuals recovering a TB infection (λ^R is the

[19] Our model simplifies a little here as in reality a TB infection might develop from latent to active without the individual being exposed to active TB (again). However, incorporating such a possibility does not change the theoretical predictions of the model (results are available upon request).

recovery rate). The TB infection rate is determined by:

$$\lambda_t^I = \frac{\beta}{y_t^\gamma}\left(\frac{I_t}{S_t}\right)^\alpha, \tag{2}$$

where y_t is income per capita and $0 < \alpha, \gamma < 1$. The first principal idea in (2) is that the number of infected individuals per non-infected population increases the risk that a random individual in S comes into contact with a TB infected individual.[20] The second idea is that given the risk of getting into contact with an infected individual, the disease-specific infectivity constant (β), discounted by the level of income (y_t^γ),[21] determines the probability of actually contracting TB. The infectivity constant is a function of:

$$0 \leq \beta = (\sigma - \theta)(1 - \delta) \leq 1, \tag{3}$$

where σ denotes the contagiousness of TB, θ reflects the degree of awareness in terms of avoiding contracting/transmitting the TB infection ($\sigma \geq \theta$), and $0 \leq \delta \leq 1$ is the share of infected individuals that are isolated. We think of θ and δ as the two parameters in the model possibly influenced by the dispensaries. As already indicated, the recovery rate is assumed to be a function of income:

$$0 \leq \lambda_t^R = \bar{\lambda}^R - \frac{\Psi}{y_t} \leq 1, \tag{4}$$

where $0 \leq \bar{\lambda}^R \leq 1$ is the maximum recovery rate and $\Psi > 0$ is a calibration parameter. Income per capita is assumed to grow at a constant rate:

$$y_{t+1} = (1 + g_y)y_t, \tag{5}$$

where g_y is the growth rate. We see that according to (2) and (4), income growth feeds into

[20] This is a slight modification compared to the standard SIR model, which simply assumes $\lambda_t^I = \beta I_t$.

[21] We discount the infectivity constant with income as being well-nourished, for example, provides some immunity against contracting a TB infection. In addition, evidence suggests that reinfection (i.e., latent TB evolves into active TB) is less likely if people are well-nourished. As an alternative, we could have modelled the possibility of reinfection explicitly such as:

$$\lambda_t^I = \frac{1}{y_t^\gamma}\left(\beta\left(\frac{I_t}{S_t}\right)^\alpha + \lambda^{re}\right),$$

where λ^{re} is the reinfection rate, but this does not changes the quantitative results of the model.

the model through the infection rate as well as the recovery rate. The (active) TB infected population is given by:

$$I_{t+1} = I_t + I_t^{New} - D_t^{TB} - R_t, \tag{6}$$

where $D_t^{TB} = \lambda^{TB} I_t$ is the total number of TB deaths and λ^{TB} is the TB death rate among the TB infected population. Finally, we note that the TB mortality rate (out of the total population) is given by:

$$M_t = \frac{D_t^{TB}}{P_t} = \lambda^{TB} \frac{I_t}{S_t + I_t}. \tag{7}$$

Here we see that the two components of the TB mortality rate are λ^{TB} and the incidence rate, $I_t/(S_t + I_t)$, and since we think of λ^{TB} as being constant, any negative effect on the TB mortality rate due to the dispensaries (in this model), works through lowering the incidence rate.

Combining equations (1)-(7) yields a system of three first-order difference equations which characterises how the populations and income evolve over time. This system is reported in the Online Appendix, along with a phase diagram sketching the solution in Online Appendix Figure A14. We also show that if $g_y = 0$, $\alpha < 1$, along with a couple of other plausible parameter restrictions, the model convergences to a constant ratio of TB infected individuals to non-TB infected individual (i.e., $s \equiv I_t/S_t$), implying a constant TB incidence rate, $s/(1+s)$, and TB mortality rate, $\lambda^{TB} s/(1+s)$. If $g_y > 0$ these rates eventually converge toward zero.

This model is now used to evaluate quantitatively about the adjustment process of TB mortality after the introduction of the dispensaries. Specifically, we simulate the development of the TB mortality rate after the introduction of the dispensaries (as measured by an increment increase in θ) and compare it to a baseline simulation, which then corresponds to a counterfactual path without any changes in θ. This type of comparison is equivalent to our (empirical) event-studies, reported in section 6.3. The Online Appendix presents a detailed description of the parameter-value calibration of the model, but basically our strategy was to match first moments in the 1908-data in population size, the TB mortality rate, and the crude death and birth rates. Figure 4 depicts the simulated differences between the path of the TB mortality rate with dispensaries and the counterfactual. Importantly, we see that only after some four years, the effect has fully materialized. In this specific simulation, we also find that the TB mortality rate is 26 percent lower after five years, while the crude death rate has only decreased by around 1.5

percent, suggesting that we should find substantially smaller effects on the crude death rate compared to the TB mortality rate.

Finally, the model in generel informs us that whereas a decrease in the TB mortality rate can either be due to an increase in the isolation factor or awareness, the isolation (population) rate, $q_t \equiv Q_t/P_t = \delta I_t/(S_t + I_t)$, would increase if the effect is driven solely by isolation,[22] while it decreases if the effect works through increased awareness.[23] Using these theoretical predictions, our empirical analysis later provides evidence that the decline in TB should more likely be interpreted along the lines of increased awareness (i.e., information) or a combination of increased awareness and isolation, but not only isolation.

Figure 4: Simulation of TB mortality rate

4 Data and balancing tests

This section presents an overview of the various datasets collected for the empirical analyses. Along with detailed accounts of the data sources, descriptions and definitions of all the variables are reported in Online Appendix Table A1.

Data on the timing of the TB dispensaries are collected and digitized from the annual publication "Medicinalberetning for Kongeriget Denmark" published by Sundhedsstyrelsen,[24] for the years 1908–1946. We also obtain the date of commissioning of TB hospitals, sanatoria, and waterworks, from the Medical Reports.[25]

Most of the dispensaries in Denmark were placed in cities, so in order to evaluate their ability to reduce mortality, we digitized an annual city-level dataset, containing information on eight different causes of death (see next section), the crude death rate, child mortality, the crude birth rate, and population size for 87 cities (i.e., all market towns/cities in Denmark) over the period 1890–1950. The data are collected from the annual publications "Dødsårsagerne i

[22]This is under the assumption that the incidence elasticity with respect to δ is less than one. Nevertheless, it is clear that as $\delta \to 1$ this is *not* the case, since the incidence rate then becomes zero.

[23]For simplicity, we here assume that the number of isolated individuals is a fraction of all the infected individuals, and these two groups are similar in other observable aspects (e.g., the chance of recovery).

[24]Sundhedsstyrelsen, the National Health Service of Denmark, replaced det kgl. Sundhedskollegium in 1909, which published the reports before.

[25]We refer to the "Medicinalberetning for Kongeriget Danmark" as the "Medical Report".

Kongeriget Danmarks Byer" published by Sundhedsstyrelsen, which contains data from 1890 to 1919. By 1920, rural districts are added and the publication therefore changed its name to "Dødsårsagerne i Kongeriget Danmark", and from 1921 more cities are included due to the fact that certain areas previously belonging to Germany became part of Denmark after World War I.[26] From 1901 these statistics become more detailed, and from this year onwards, we are able to compile a panel with all eight causes of death (i.e., diseases), whereas from 1890 to 1900 only TB is available.

There are many reasons to believe that these historical mortality data are generally of high quality and this seems to be particularly true for TB. Diagnosing TB became easier due to a number of innovations, e.g. by "discovery of the acid-fast nature of the bacillus by Ehrlich in 1882, discovery of X rays by Roentgen in 1895, development of the tuberculin skin test by Von Pirquet and Mantoux in 1907-1908" (CDC).[27] The aforementioned discovery by Ehrlich allowed diagnosing active, rather than latent, TB by examining the sputum of a patient.[28] According to Holm (1946a), diagnosing TB in Denmark included "a tuberculin test, roentgenography, and examination of sputum or gastric lavage for the presence of tubercle bacilli" and, therefore, the medical innovations were applied for diagnosis. In addition, disease registration on pre-printed forms had been in place in the cities since 1856 (Johansen, 2002),[29] and on the quality of Danish (historical) medical statistics, Lindhardt (1938, p.28) observes that Danish and foreign investigators regard as being in the very front rank "as regards the conformity of the figures with the actual facts." She also highlights that pulmonary tuberculosis is easy to diagnose at death and that Denmark had a large number of well-trained physicians. She examined death certificates for 1924-35 for patients who died of pulmonary TB and found that less than one percent did not have the cause of death affirmed by a physician. She concludes that there is little scope of many deaths of pulmonary TB being wrongly labelled. This evidence all in all suggests that the quality of the vital statistics in Denmark seems to be very high of historical standards.

[26] We refer to the "Dødsårsagerne i Kongeriget Danmarks byer" and the "Dødsårsagerne i Kongeriget Danmark" as the "Cause of Death Statistics".

[27] See: `http://www.cdc.gov/mmwr/preview/mmwrhtml/00000222.htm`.

[28] Ehrlich famously self-diagnosed that he had TB in 1887, see Sakula (1982).

[29] Johansen (2002, p. 180) mentions that the TB mortality statistics for the 1890s are believed to underreport TB. This is an additional reason for running some regressions from 1901 only.

In the baseline, we stop the analysis in 1939, which is not crucial for the results, but we do so for two reasons. First, 1940-45 mark the years of World War II in Denmark, and we do not want to confound our results with this large shock. Second, the 1940s also mark the advent of modern medicine in which antibiotics for the treatment of TB became available. Also, the BCG vaccine became common over this period of time. Descriptive statistics for the mortality rates for TB and the seven other causes of death as well as death and fertility rates are given in Table 1, and Online Appendix Table A2 describes changes in TB mortality over time, along with the number of dispensaries over time. The aggregate development of mortality, child mortality, live births, and the other causes of deaths are depicted in the Online Appendix Figures A2-A11.

Our second dataset, which we also collected and digitized, contains annual information on the number of patients admitted to all TB hospitals and sanatoria in Denmark (Medical Reports, various years). There were about 45 TB hospitals and 39 sanatoria (on and off) throughout this time period (1903-1939). Using their former addresses, we calculate the annual number of patients in each county using GIS software, and our empirical analysis then estimates how these county numbers change when the dispensaries are being rolled out. While for the TB hospitals it is natural to use the number of dispensaries within the county as the attachment network, since the institutional setup was such patients could only be admitted to a TB hospital within their county of residence, this assignment rule is less straight forward for the sanatoria, as they could be used by patients (with a state subsidy) from across the country. Nevertheless, in order to make the results comparable, we use the same assignment rule for both institutions, which also can be supported by the presumption that people are more likely to opt for a sanatorium closer to their homes.

Finally, we digitized income per tax payer at the city level annually from 1904 to 1939, using historical tax-assessment records. These data are taken from "Statistiske Meddelelser", published by Statistics Denmark in the relevant years. These unique historical income data can be used to assess possible productivity spillover effects from the dispensaries, which, besides our cost-benefit analysis, allows for a broader evaluation of the total economic impacts. In addition, the robustness analysis for our mortality findings, exploits initial variation in income (interacted with year fixed effects), along with within city income inequality, as controls.[30]

[30]The within-city income inequality data are also obtained from Statistiske Meddelelser. Whereas the income

Table 1: Summary statistics

We conclude this section by commenting on a number of balancing tests reported in Online Appendix Table A3 in which we compare the population, TB rate, death rate, child mortality rate, tax income per capita, and taxpayers per capita (as a proxy for labor force participation). Comparing the initial five year mean of the variables between the TB dispensary adopters versus non-adopters, we observe no significant differences, although the mean population size of the adopters appears larger, driven by the few large cities in Denmark.[31] Splitting the sample between the 17 pre-1930 adopters and the 21 post-1930 adopters, we only observe significant differences between the means of income per capita and taxpayers per capita, with pre-1930 adopters having higher means. In the robustness analysis, we show that our results are robust to controlling for initial income. Finally, Online Appendix Figure A15 depicts the relationship between the initial TB rate and the year of establishment of a TB dispensary for the unbalanced and balanced panel. The relationships are negative, but far from being statistically significant.

5 Empirical strategies

The first strategy in our empirical analysis is based on difference-in-differences estimation that compares the mortality rate of different diseases before and after the introduction of a TB dispensary in a given city:

$$M_{c,t}^{d\in D} = \beta_d Dispensary_{c,t} + \alpha_c + \gamma_t + \mathbf{X}'_{c,t}\theta + \varepsilon_{c,t}^{d}, \tag{8}$$

where $M_{c,t}^{d\in D}$ indicates the mortality rate in city c at year t, $Dispensary_{c,t}$ is an indicator equal to one after the introduction of a TB dispensary in city c, the α_c's are city fixed effects, the γ_t's are year fixed effects, and $\mathbf{X}_{c,t}$ is a vector of controls included in the robustness analysis. The error term, $\varepsilon_{c,t}^{d}$, is allowed to cluster at the city level and to be heteroscedastic. We estimate equation (8) for the following set of diseases ($d \in D$): TB, cancer, influenza, pneumonia, accidents and

data start annually in 1904, the first year with annual inequality data is 1918.

[31] The initial five year means are defined as the mean of the years 1890-94 for the population, TB rate, and death rate, the mean of the years 1901-05 for the child mortality rate, and the mean of the years 1904-08 for the income per capita, and taxpayers per capita. For cities included in the dataset later, we use the mean of the first five year available.

suicides (including homicides from 1931), scarlet fever, diphtheria, and polio. Since the principal purpose of a dispensary was to prevent the spread of TB, we expect that $\hat{\beta}_{TB} < 0$, while there should be no (or smaller) effects on the remaining diseases (see also discussion below).

The second strategy exploits the explicit targeting of the dispensaries on preventing TB to estimate a triple-differences model which takes on the following form:

$$M_{d,c,t} = \beta \; Dispensary_{c,t} \times Prevent_d + \phi_{c,t} + \lambda_{d,t} + \mu_{d,c} + \varepsilon_{d,c,t}, \tag{9}$$

where the disease data have been stacked, so that $M_{c,t,d}$ is the mortality rate of disease d in city c at year t, $Dispensary_{c,t}$ is the same indicator as above but is now interacted with $Prevent_d$, indicating whether disease d was prevented (i.e., treated) by the dispensary, which, we assume, was only the case of TB. The most important advantage of the the triple-differences model is that it allows us to non-parametrically control for city-by-year fixed effects ($\phi_{c,t}$), disease-by-year fixed effects ($\lambda_{d,t}$), and disease-by-city fixed effects ($\mu_{d,c}$).[32] Thus, the experiment, which we now set up in the data, compares the development of TB mortality to non-treated diseases before and after the introduction of a dispensary in *one given* city, and we avoid comparing the development of TB mortality of larger to smaller cities, for example. The error term is given by $\varepsilon_{d,c,t}$ and cluster robustly at the city level.

We first note that the β's in equations (8) and (9) give the effects on the outcomes of being offered the possibility of treatment, which is known as an intention to treat (ITT) effect, and imply that we do *not* need to worry about the fact that, at the individual level, uptake is most likely endogenous. The main assumption of identifying the ITT effect in the difference-in-differences strategy is that cities with a dispensary would have changed similarly to other cities, if not for the introduction of the dispensary, while the identifying assumption in the triple-differences strategy is that the diseases (in a given city) would have changed similarly in the absence of a dispensary. An indirect test of these assumptions is to study whether the trends in the outcomes prior to treatment are parallel, which we do, along with studying the

[32] We note that the interaction fixed effects ($\phi_{c,t}$, $\lambda_{d,t}$, $\mu_{d,c}$) implicitly control for city fixed effects, year fixed effects, and disease fixed effects.

subsequent dynamics of the shock, by estimating the following event studies:

$$M_{c,t}^{TB} = \sum_{j \in T} \beta_j \times Dispensary_{c,t}^{\tau+j} + \alpha_c + \gamma_t + \varepsilon_{c,t}, \tag{10}$$

$$M_{c,t,d} = \sum_{j \in T} \beta_j \times Dispensary_{c,t}^{\tau+j} \times Prevent_d + \phi_{c,t} + \lambda_{d,t} + \mu_{d,c} + \varepsilon_{c,t,d}, \tag{11}$$

where $T = \{-10, \ldots, -2, 0, \ldots, 10\}$, and $Dispensary_{c,t}^{\tau+j}$ is an indicator equal to one when $t = \tau + j$, where τ is the year a dispensary was established in the city c, except for $Dispensary_{c,t}^{\tau-10}$ and $Dispensary_{c,t}^{\tau+10}$ that take on the value one given $t \leq \tau - 10$ and $t \geq \tau + 10$, respectively. The remaining variables are as defined above. The estimated coefficients $\hat{\beta}_j$ trace out the dynamic effects of the introduction of a TB dispensary relative to the omitted base year just before the intervention (i.e., $t = \tau - 1$). For the common pretrends assumption to hold, we should find that $\forall j < 0$ $\hat{\beta}_j \approx 0$. For example, systematically positively estimated coefficients prior to $t = \tau$ could indicate that a TB dispensary was introduced due to an unusual high level of TB mortality, while negative estimates could suggest that the rollout of the TB dispensaries is spuriously capturing a secular trend in TB mortality.

As the dispensaries were targeting TB, we expect that the effect of on the other diseases would be insignificant. However, this is ex-ante not necessarily the case due to competing risk and co-mortality. In the triple-differences strategy, competing risk would give rise to a downward bias, whereas co-mortality would give rise to a bias in the opposite direction. While the placebo-regression results in equation (8), in fact, suggest that there was no average effect of the dispensaries on the mortality of the other diseases we are considering, additional checks are also not supportive of these two possibilities.

Along with a range of other robustness checks, we also perform random-placebo tests that randomly reassigns the intervention year and reestimate equations (8) and (9). By repeating this procedure 2,499 times, we obtain distributions of the estimated coefficients which then can be compared to the estimates from using the true intervention years.

6 Results

We begin the analysis by presenting estimates of equation (8), which exploits year and city variation for the purpose of identification, corresponding to a standard difference-in-differences (DiD) model. These estimates are presented in subsection 6.1. Subsection 6.2 reports the results from the triple-differences (DiDiD) model of equation (9), which adds the control diseases to the setup. The findings from the event studies in equations (10) and (11) are reported in subsection 6.3. Finally, our robustness analyses are unfolded in subsection 6.4.

6.1 Difference-in-differences model

Table 2 reports the baseline results from estimating equation (8). We find negative estimates, which are statistically significant at the one percent level in all the specifications. Specifically, the estimate in column (1) suggests that an opening of a TB dispensary reduced TB mortality per 1,000 by 0.22. Given that the average TB mortality rate was 1.31, this corresponds to a reduction of about 16 percent. This is also the magnitude obtained when using a log transformation in column (2). To get a sense of this magnitude, TB mortality per 1,000 fell by 1.28 from 1907 to 1939 in the Danish city population, corresponding to a decline of 76 percent, which implies that the dispensaries explains about 20 percent of this development.

The baseline estimate remains stable both in magnitude and statistical significance when adding the one-year lagged TB mortality rate in (column 3) or up to five one-year lagged TB mortality rates (column 4). Finally, column (5) considers only pulmonary TB mortality as this particular form of TB is where we expect to see an influence from the preventive actions of the dispensaries.[33] We find a negative and statistically significant coefficient at the one percent level. Using the observed average of pulmonary TB, the magnitude of the estimate suggests that an opening of a dispensary reduces the pulmonary TB mortality rate by around 21 percent, which, as expected, is larger compared to the effect on all TB forms as reported in column (1).

Table 2: Effect of TB dispensaries using city by year data on TB

[33]Pulmonary TB accounts for the bulk of the variation in the overall TB mortality rate, that is, the ratio of pulmonary TB to total TB is 77 percent in the sample from 1901 to 1939.

Placebo outcomes Table 3 reports the results when using mortality rates of cancer, influenza, pneumonia, accidents and suicides, scarlet fever, diphtheria, and polio as placebo disease outcomes in equation (8). As it is only possible to obtain data on the placebo outcomes for the period 1901-1939, column (1) starts by showing that the effect on TB mortality remains negative, significant, and of the same magnitude for this restricted time period. Columns (2)–(8) next report the estimates for the placebo outcomes, and we see that the estimated coefficients are mostly negative, but small in numerical magnitude and always statistically insignificant. For example, for pneumonia in columns (4), which arguably is the disease most similar to TB, the estimated coefficient is not a precise zero, but it is about 65 percent smaller in numerical magnitude compared to the baseline, which means that an opening of a dispensary reduced the pneumonia mortality rate by 5.9 percent, albeit this magnitude remains insignificant. The coefficients on the remaining placebo diseases are even smaller in numerical magnitude. This pattern suggests that, in the end, the dispensaries mainly fulfilled their stated purpose of preventing and reducing TB and indicates that our baseline estimate is not picking up a general trend in mortality. It also seems to suggest that competing risk and co-mortality are not so important issues for these diseases, although one could argue that competing risk only materializes some years after the introduction of a dispensary and, as the DiD estimator is measuring the average effect for all the post-treatment years, this could result in insignificant DiD estimates. For this reason, we also estimate event studies for all the placebo diseases in order to check if this is indeed the case (see section 6.3).

Table 3: Placebo outcome regressions

Child mortality, overall mortality, and fertility Table 4 investigates whether the dispensaries had any impacts on child mortality, overall mortality, and fertility. The estimates, reported in columns (1) and (2), reveal that the rollout of the dispensaries reduced the child mortality rate by 10 percent, supporting the argument in Bütikofer and Salvanes (2015) that reducing TB benefited young children as well. This finding is also not surprising in the light of the age distribution of TB mortality for Denmark in, e.g., 1921-25 (see Online Appendix Figure A13), which shows that TB mortality for children below the age of five is as high as for adults of prime age.

Next, we investigate whether there is any measurable impact on overall mortality (i.e., the crude death rate). Columns (3) and (4) report negative coefficients which are significant at the 10 percent level. We see that dispensaries reduced the crude death rate by about three percent, which is substantially smaller compared to the effect on TB, which is very much in line with the simulation results from the modified SIR model in section 3 and therefore expected. These findings also suggest that the dispensaries reduced the disease death ratio for TB, which is also confirmed in Online Appendix Table A4, where the disease death ratio for TB is the outcome of interest.[34]

Previous research have argued that mortality changes might influence fertility patterns due to more women surviving to birth giving ages or replacement behavior (e.g., Acemoglu and Johnson, 2007; Ager et al., 2016). Columns (5) and (6) show positive, but insignificant estimates for the crude birth rate. One interpretation of these findings is that the effect coming from more women surviving to birth giving ages dominates possible replacement behavior, which would go in the opposite direction, since more children now survive.

Table 4: Effect of TB dispensaries using city by year data on other outcomes

6.2 Triple-differences model

Table 5 reports the findings of estimating equation (9), which is the triple-differences model, using all the aforementioned control diseases. This type of model allows us to include additional fixed effects as compared to the previous DiD model. For example, city-by-year fixed effects account for all the variation which occurs between the different cities over time, such as processes of convergence or divergence in, e.g., income, income inequality, or mortality; local political or institutional changes; migration; and pollution (see, e.g., Hanlon, 2015). Moreover, controlling for disease-by-city effects allows the basic mortality environment to be systematically different across the cities.

Column (1) shows an estimate which is quite similar to our baseline DiD estimate, that is, $\hat{\beta} = 0.19$ with standard error $= 0.07$, so evaluated at the average, we find that an opening of a dispensary reduces the TB mortality rate by 18 percent. Columns (2) and (3) demonstrate that

[34]The disease death ratio for TB is defined as the number of TB deaths divided by the total number of deaths.

this quantification is robust to the inclusion of one/five lagged dependent variables. Overall, we conclude that these two different ways (i.e., DiD and DiDiD) of estimating the effect of the dispensaries on TB mortality yield to the same conclusion, both in terms of sign and magnitude.

Table 5: Effect of TB dispensaries using city by year by disease data

6.3 Event-study analysis

This subsection reports evidence indirectly supporting the identifying assumption that the TB mortality rate would have continued its pre-treatment path in the absence of a TB dispensary by showing that there were no systematic trends prior to its introduction. Moreover, the subsequent dynamics, which is also revealed in these models, show that the TB dispensaries had a relatively fast permanent level effect on the TB mortality rate.

For convenience, instead of reporting the results in regression tables, we plot the estimated β_j's from the event studies of the DiD and DiDiD models, along with their 95 percent confidence intervals in Figure 5.[35] In both models, the estimated coefficients in the years preceding the TB dispensaries fluctuate non-systematically around zero, which supports the common pre-trend assumption. After the introduction, however, we observe a permanent downward shift in the level of the TB mortality rate. The estimated coefficients of equation (10) become significantly negative one year after the introduction of the TB dispensary and tend to become larger in absolute terms and more precisely estimated after more years have passed since introduction. Although the confidence interval of the coefficients estimated from equation (11) are wider, we observe a similar pattern, and the majority of the coefficients are significant at the 10 percent level especially some years after establishment.

The estimated coefficient patterns from these event studies are very much are line with our theoretical simulation, depicted in Figure 4, that is, our theory supports our empirical findings that the full effect of a dispensary materializes relative fast after its introduction. In addition, this is in line with estimates on time to death for the pre-chemotherapy era. Goodman and Fuller (2015) report that the median time to death of untreated TB is 2.5 years. Tiemersma et al. (2011) estimate that the average time to death is 3 years. The time to death from notification

[35]The regression tables are, however, also shown in Online Appendix Table A5.

for Denmark of pulmonary TB for the period 1925-34 was on average 14.3 months for males and 12.9 for females as reported by Lindhardt (1938), who analyzed around 40,000 notifications for this period. Given that TB takes time to develop, this is in line with the average suggested by Tiemersma et al. (2011). It should, however, be noted that nearly 30 percent die within 6 months of notification, and the medical literature suggests that some people develop TB within 1 to 3 years (Flynn and Chan, 2001) and death can happen after a few weeks as pointed out by Nagelkerke (2012). Moreover, there is substantial variance in how fast different age groups die. There is a significant number of infant (below 1 year) and child TB deaths and these groups die faster. The same is true for TB deaths in the group above age 65, who according to Lindhart (1937) died 4 months after notification on average.

Online Appendix Figures A16-A22 show the results from estimating event studies for all the placebo diseases in the DiD setup (i.e., equation 10). In general, we find no evidence of pre-treatment trends in any of these observables, and there seems to be no effects even after 10 years or more after the introduction of a dispensary, indicating that there is not competing risk or co-mortality between TB and our set of placebo diseases (even in the longer run).

Figure 5: Event-study estimates of impact on TB before and after TB dispensary

6.4 Robustness

We have carried out a number of robustness checks based on both the DiD model and the DiDiD model. First, we perform a random placebo test where we randomly shuffle the year of establishment of the TB dispensaries and reestimate the DiD and DiDiD models to exclude the possibility that we are capturing a spurious relationship. Second, we study explicitly if there are any spillover effects coming from the opening of a dispensary in a neighboring city. Third, we turn to other public health policies targeted at TB in the form of sanatoria and TB hospitals. Fourth, as poverty and inequality are possible determinants of TB mortality, we control for the pre-intervention income and a pre-intervention city-Gini coefficient. Fifth, we address treatment heterogeneity with respect to the initial TB rate, income and inequality. Finally, we discuss a series of additional robustness checks reported in the Online Appendix.

Random placebo test We have investigated possible misspecifications of the models caused by spurious relationships between the rollout of the dispensaries and the decline in TB by performing random placebo tests. Specifically, we randomly shuffle the year of commissioning of the dispensaries 2,499 times and obtain the placebo coefficients from re-estimating the DiD and the DiDiD models. Figure 6 shows the distribution of the placebo coefficients along with the original coefficients marked by the vertical dotted lines. The distributions resemble a Gaussian curve centered at zero with the actual coefficients positioned to the far left. The area to the left of the actual coefficients under the distributions are 0.0072 and 0.0056 for the DiD and DiDiD models respectively. This demonstrates that the true years of commissioning of the TB dispensaries are necessary to produce the results and further suggests that our results are not spurious.

Figure 6: The distribution of coefficients from random placebo test

Neighboring spillover effects It is quite possible that introducing a dispensary in one city has a negative spillover effect to another neighboring city's TB mortality rate, if there is some (market/population) integration between the cities. While this might be an interesting finding in itself, this would change the interpretation of our baseline estimates, such that they should be interpreted as relative effects, since the *Stable Unit Treatment Value Assumption (SUTVA)* would be violated in this case.

Table 6 reports the results from controlling for the opening of a dispensary in a neighboring city, where neighbor is classified as being a city within 10km (columns 1 and 2), 30km (columns 3 and 4), or 50km (columns 5 and 6). We find that the both the DiD and the DiDiD point estimates are (if anything) numerically larger compared to their baseline values, and they are all significant at the five percent level or more. In addition, the neighbor estimates are all negative, but only statistically so in the DiDiD model within 30km (column 4), that is, there is only limited evidence of spillover effects from the opening of dispensaries in neighboring cities. We, therefore, conclude that the SUTVA condition is largely satisfied and if anything the baseline underestimates the total effect of the dispensaries.

Table 6: Effect of TB dispensaries controlling for neighboring dispensaries

Sanatoria and TB hospitals Table 7 investigates the robustness of our results to the commissioning of sanatoria and TB hospitals. There are around 80 of these institutions spread across the country during this time period, and most of them open quite early. As most sanatoria are placed at the countryside (outside cities), we attempt to capture their influence on TB mortality by the sum of the reciprocal distances from the city to all sanatoria located within the county of that city, and before the opening, we assume that the distance is infinite. Our imposed restriction that the sanatorium has to be located within the county of the city makes for an easier comparison with the results for the TB hospitals, but we obtain very similar estimates relaxing this restriction. We measure the impact of the TB hospitals by counting the number of TB hospitals in each county and assign this number to all the cities within that county. This choice is motived by institutional reasons in the sense that an infected individual would only be referred to a TB hospital within his county of residence (similar results are obtained using reciprocal distances within the county).

Columns (1)-(4) report the results for the sanatoria, while columns (5)-(8) report the results for the TB hospitals. In the DiDiD model, reported in the even-numbered columns, we assume that the sanatoria and TB hospitals only prevent TB. The following results are worthwhile noticing: First, we see that both the DiD and the DiDiD estimates of the dispensaries remain relatively stable in magnitude and statistical significance, if anything, the numerical magnitude increases somewhat (columns 7 and 8). Second, we cannot say that the commissioning of sanatoria had no effects on TB mortality; the point estimates are positive, but too imprecisely estimated to say anything with just some degree of certainty. Third, the estimate on the interaction between the rollout of dispensaries and sanatoria is very close to zero and highly insignificant in both models (columns 3 and 4). This could suggest that isolation is not the main mechanism by which the dispensaries reduce TB mortality. On the other hand, it could also simply reflect that infected individuals had the liberty of choosing the sanatorium across the country that they liked the most. If this choice is largely unrelated to the distance from where they lived, we cannot measure the local impact of the sanatoria and their interaction with the dispensaries. Fourth, and finally, we see very little impact of the TB hospitals and their interaction with the dispensaries on TB mortality. Since we can be more sure here that we should be able to measure their local impact (due to the institutional setup), the latter caveat

is less likely to apply here.

Table 7: Effect of TB dispensaries controlling for commissioning of TB hospitals and sanatoria

Controlling for income and inequality before the intervention TB mortality could to some degree be biased towards the citizens with limited means, who potentially suffered from malnutrition. This is a conceivable concern regarding the DiD analysis, if the income level of the cities affected the rollout of the dispensaries. For example, richer places may suffer less from the disease or could better attract nurses trained in treating TB. This is less of a concern for the DiDiD model, as we control for time-varying city fixed effects and time-varying disease fixed effects. Still, if income affected TB mortality more strongly as compared to other diseases, this would remain an issue.[36] In the DiDiD model, we, therefore, make the *very* conservative assumption that pre-intervention variation in income and inequality only potentially influences TB, which is necessary to make, as these interactions otherwise would be absorbed by the city-by-year fixed effects.

Columns (1) and (2) of Table 8 present estimates conditioning on the log tax income per capita (for the tax year 1904-05) interacted with a full set of time fixed effects, and in the DiDiD model also interacted with the prevent indicator.[37] In the tax year 1918-19, the income distribution of the citizens liable to pay income taxes became available, and it becomes possible to calculate a Gini coefficient for each city. Columns (3) and (4) include the Gini coefficient of 1918-1919 interacted with a full set of time fixed effects (and in the DiDiD model also interacted with the prevent indicator), and exclude cities which adopted a dispensary before 1920 to control for inequality within the cities.[38,39] Columns (5) and (6) control simultaneously for

[36]We note that this concern is being addressed somewhat by using similar infectious diseases, such as pneumonia and scarlet fever, as controls which are also likely to be affected by income (see Online Appendix Table A6).

[37]Denmark introduced income taxation in 1903 (Aidt and Jensen, 2009) for which reason taxable income is available from 1904 onwards. According to Philip (1955, p.57), the main principles remained the same throughout the period we study. There was an exemption of DKK 800 in Copenhagen and of DKK 700 in the market towns. The income tax was progressive and rates were initially very low (Seligman: 1908, p.83), but increased over time, see Philip (1955, p.56).

[38]For cities included in the dataset later than 1904 and 1918, we use the income per capita and the Gini coefficient in the year they are added to the data. None of the cities added to the dataset, had a TB dispensary initially. We cannot observe people with income below 800 DKK a year, as they were not liable to taxes

[39]We do not include the contemporary income per capita and Gini coefficient, because TB mortality and income or inequality are likely jointly determined.

pre-intervention income and inequality. As seen from the reported estimates, even when we control for these pre-intervention variation interactions, the results are similar to the baseline, although marginally larger numerically.

Table 8: Effect of TB dispensaries controlling for pre-intervention income and inequality

Treatment heterogeneity Table 9 explores possible treatment heterogeneity in the dimensions of initial TB mortality, population density, tax income per capita, and within-city income inequality. Columns (1) and (2) include an interaction term between the dispensary indicator and the initial (standardized) 1890 TB mortality rate in equation (8) and (9) respectively. Likewise, columns (3) and (4) include the interaction between (standardized) 1890 population density and the dispensary indicator. Columns (5) and (6) include an interaction term between the dispensary indicator and the (standardized) 1904 log tax income per capita. Finally, in columns (7) and (9), cities which adopted a dispensary before 1920 are excluded from the sample, and an interaction term between the dispensary indicator and the (standardized) 1918 Gini coefficient is included.[40]

Reassuringly, compared to the baseline estimates, the point estimate of the TB dispensary remains unchanged in all specifications. The interaction between the dispensary and the initial TB mortality rate enters negative and significant into both the DiD and the DiDiD models, implying that a one standard deviation increase in the initial TB mortality rate increases the effect of the dispensary by 82 percent in case of the DiD estimate. It also suggests that the TB dispensaries were less effective in areas with low initial TB mortality, which we use in Online Appendix A7 to propose a modified treatment measure (see below). We find small positive estimates on the initial population-density interaction. The interaction between the dispensary and the initial log tax income per capita enters positively and significantly into both models suggesting that cities with a higher initial income benefited less from a dispensary. This finding can be explained by the hypothesis that higher income leads to a better state of nutrition thereby decreasing the likelihood of dying from TB. Lastly, the interaction term with the initial

[40]For cities included in the dataset later than 1890, we use the TB mortality rate in the year they are added to the data.

Gini coefficient is small and insignificant, hence within-city income inequality does not seem to affect the treatment effect of the dispensaries. Thus, the effect does not vary with inequality within the city, but rather with income inequality between the cities.

Table 9: Treatment heterogeneity of TB dispensaries

Additional robustness checks Finally, we briefly mention our battery of additional robustness checks, reported in the Online Appendix. First, it is possible that the general information on avoiding TB affected other airborne infectious diseases due to better hygienic practices of not spitting on the street or coughing in public. While the placebo outcome regressions suggest that there are little spillover effects to other (airborne) diseases, one might (at the least ex-ante) believe that there are spillover effects on scarlet fever and pneumonia. According to Jayachandran et al. (2010), these two diseases are the infectious diseases which bear the most similarities with TB. We address this issue in two alternative ways. First we limit the control diseases to the most similar diseases; scarlet fever and pneumonia. If the dispensaries only affected TB through general information that potentially could affect similar infectious diseases, we should find a smaller effect using this subset of control diseases. Second, we limit the panel by excluding all *infectious* diseases, using only cancer, and accidents and suicides as controls, which are unlikely to be affected by the intervention. Online Appendix Table A6 shows DiDiD estimates that are very similar to the baseline, using these alternative subsets of control diseases.

Online Appendix A7 shows the basic DiD and DiDiD estimates, using two alternative treatment measures. First, instead of the baseline indicator, we use a treatment measure that takes on the value zero before the intervention and years since the adoption of the dispensary afterwards. We find negative and significant estimates, which, along the lines of our event studies, suggests that the effect is (weakly) increasing over time. Second, we have tried to exploit the five-year average TB mortality rate just prior to the intervention as a measure of treatment intensity, similar in spirit to the basic idea of Bleakley (2007), that is, cities with higher levels of pre-intervention TB stand to gain more from the opening of a dispensary. Also with this second measure, we obtain strong negative effects.

The BCG vaccine only became widespread in Denmark in the 1940s, but the remote island of Bornholm experimented with the vaccine from 1936. Online Appendix Table A8 demonstrates

that our results are robust to excluding the cities on Bornholm and limiting the panel to 1935.[41] Given that the BCG vaccine is less effective against pulmonary TB—the main component of TB mortality—it is not surprisingly that the results are largely unaffected. Moreover, limiting the panel to 1935 also excludes the possibility that our results are influenced by the rollout a universal home-visiting program for mothers and their infants starting in Denmark in 1937 (Wüst, 2012).

Online Appendix Tables A9 documents that our findings are generally robust to excluding Copenhagen (the capital and largest city), the five largest cities, and all cities that adopted a dispensary before 1920.[42] Online Appendix Table A10 further shows that the results are robust to controlling for the lag of log population, including the log of the initial population interacted with time fixed effects, weighting the estimation by the log of the initial population, and including a city-specific linear time trend, although it should be noted that in the baseline DiDiD settings, the linear trend is a special case of the non-parametric city specific trend, we control for by city-by-year fixed effects.

An additional concern could be that the effect from TB dispensary is confounded by other general public measures against infectious diseases at the time. To address this, we have controlled for the commissioning of waterworks in the cities, as clean water have been emphasized as an important public health intervention at this time, affecting mortality by improving sanitation and water supply (Cutler and Miller, 2005, Alsan and Goldin, 2015). TB is not a water-born disease, however, clean water could have general implication for overall mortality. As revealed by Online Appendix Table A11, this does not change our findings.[43]

The system of TB dispensaries in Denmark was organized around larger central TB dispensaries with branches in different cities (Holm, 1946a). While the two types of branches performed similar tasks, main dispensaries would often be led by specialized doctors, who also often worked at TB hospitals (Holm, 1946a). To analyze whether the main dispensaries had a

[41]The Medical Report for 1940 describes that special efforts were being made on Bornholm from 1936 onwards. 1159 people were vaccinated in this period.

[42]In 1890 and 1901 the five largest cities were by far Copenhagen, Frederiksberg, Aarhus, Odense, and Aalborg, with populations of 378,235, 76,231, 51,814, 40,138, and 31,457 as of 1901 respectively, with the sixth and seventh largest cities being Horsens and Randers with populations of 22,243 and 20,057.

[43]The first waterworks were commissioned in Odense in 1853 and by 1890 the five largest cities could all provide its citizens with clean water from waterworks. In 1890, 25 of the cities in sample had waterworks, by 1901, 36 cites had waterworks, and by 1939, 85 of the 87 cities had waterworks.

different effect than their branches, we split the TB dispensary indicators in equation (8) and (9) into an indicator equal to one after the introduction of a main dispensary in a given city and a similar indicator for the introduction of a branch dispensary. We present the results in Appendix Table A12. We cannot reject that the effects of the main and branch dispensaries are statistically equal and the magnitude of the estimated effects are similar to the baseline results.

Appendix Table A13 documents that the results are robust to reducing the sample to the 74 cities which constitutes a balanced panel, and extending the sample period from 1939 to 1946; the year after the last dispensary were established. The absolute magnitude of the coefficient on TB dispensary is somewhat reduced when extending the year to 1946, although it is still highly statistically significant.

Finally, we note that pasteurization has been argued to have negative impacts on Bovine TB (BTB), scarlet fever, and diphtheria. Since milk would contain bacteria from these diseases (Wilson, 1943; Jensen, 2002), pasteurization, however, would kill these and, therefore, prevent transmission. Moreover, as argued by Olmstead and Rhode (2004, p.768), BTB was mainly associated with non-pulmonary TB and had a different transmission. Evidence by Jensen et al. (1940) shows that in many towns, there was no BTB and the percentage was below 10 for all age groups. Above we found a strong effect on pulmonary TB, which suggests that our results are not explained by pasteurization and BTB.[44] Moreover, our placebo regressions revealed no impact on scarlet fever and diphtheria, which would also be impacted by pasteurization. We also note that results are robust to ending the sample in 1935 at which time BTB was still highly prevalent. Indeed, eradication of BTB only gathered momentum after 1935 as infected herds still counted 60-80 percent in 1937; see Groth-Petersen et al. (1959). We therefore conclude that our results are unlikely to be explained by BTB eradication and pasteurization.

7 Mechanisms

While the robustness analysis already showed that our baseline findings are not likely to be confounded by sanatoria and TB hospitals, and there generally seems to be little evidence

[44]The decline in non-pulmonary TB cannot alone explain the estimated effect of the TB dispensaries. Running the difference-in-difference regression on the non-pulmonary TB rate results in a small coefficient of -0.041 which is significant at the 10 percent level (standard error $= 0.021$).

suggesting any kind of interaction effect, this section exploits information on the number of patients admitted to these two institutions across counties over time to further consider the possible mechanisms.

Given the stated purpose and policies of the dispensaries, our baseline finding can be interpreted along the lines of increased awareness about how to avoid transmitting and contracting TB (i.e., information) and/or that the dispensaries directed infected individuals to sanatoria and TB hospitals (i.e., isolation). As also emphasized in the theoretical-framework section, data on the (institutionalized) isolation rate provides a possibility to investigate the relative importance of these two mechanisms as one would expect the isolation rate to increase if the baseline effect is only driven by isolation.[45]

Therefore, we now estimate the following model:

$$q_{i,t}^{v \in V} = \beta_v \; Dispensary_{c,t} + \alpha_i + \gamma_t + \varepsilon_{i,t}^{v}, \tag{12}$$

where $q_{i,t}^{v \in V}$ is the log number of admitted patients or the number of admitted patients per capita (i.e., isolation rate) in county i at year t. The patients can either be admitted to a sanatorium or a TB hospital ($v \in V$). Since our unit of analysis now is at higher level (i.e., county instead of city, but it remains yearly), $Dispensary_{c,t}$ counts the number of dispensaries in the county in a given year, α_i and γ_t are county and year fixed effects, and $\varepsilon_{i,t}^{v}$ is the error term. Note that the data section describes how we allocated the collected sanatoria- and TB hospitals-level patient data to the counties.

Table 10 reports the results from estimating equation (12), where columns (1) and (2) report the sanatoria results and columns (3) and (4) report the TB hospital results. We see that the number of sanatoria patients decrease when the local number of dispensaries increase: the isolation rate declines by 16 percent for one extra dispensary in the county (column 2), and column (1) simply shows that this effect is not driven by the denominator (i.e., county population size), but rather the number of admitted patients. This evidence indicates that the TB incidence rate is in fact decreasing and that our baseline estimate cannot only be driven by isolation, indirectly suggesting that the distribution of information on TB was important.

[45] This statement is generally not true, but it is in fact the case under plausible assumptions.

As seen from the two remaining columns of the table, we find no evidence of an effect on the number of patients in TB hospitals.

Table 10: Effect of TB dispensaries on patients in sanatoria and tuberculosis hospitals

8 Cost-benefits and spillover productivity effects

This section first evaluates the direct cost and benefits of the 38 dispensaries commissioned from 1908 to 1939, after which we study whether the dispensaries had any (indirect) productivity effects. The baseline DiD estimate from column (1) in Table 2 suggests that the dispensaries averted 6,363 death over the period 1908-1939 with a 95 percent confidence spanning from 5,290 to 7,435; see Table 11. Yet the number of saved lives is uninformative unless we obtain information on which age groups were saved. From 1921 the Cause of Death Statistics provides country-wide data on the TB mortality age distribution. We employ the age distribution of TB mortality of the five-year period 1921 to 1925 as the counter factual distribution of TB mortality without the prevention efforts of the dispensaries. By weighting the age-specific life expectancy in each year from 1908 to 1939 by the assumed age distribution of TB mortality, we get a measure of the life-years saved for the average averted TB death for each year.[46] The total life-years saved from 1908 to 1939 are hereby estimated to be 251,345 years (95 percent confidence interval; 209,111 to 293,579).

The annual reports of the National Foundation provide accounts of the expenditures of nine of the TB dispensaries in various years between 1912 to 1939.[47] Using the average value of the yearly per capita cost of the nine dispensaries in 2015 US dollar to value the total cost of the dispensaries that operated from 1908 to 1939 amounts to 19 million dollars. This suggests that the cost of saving a life amounted to 3,008 dollars and the cost of saving a life-year equalled 76 dollars (95 percent confidence intervals; 3,618 to 2,574 and 92 to 65 respectively).

With a cost per life-year saved as low as 76 dollars there is little doubt that the benefits of the

[46] The life expectancy in Denmark is obtained from the Human Mortality Database, see `http://www.mortality.org/`.

[47] We have expenses for the cities Bogense, Kerteminde, Odense, Randers, Silkeborg, Slagelse, Vejle, Viborg, and Aarhus see, Nationalforeningen til Tuberkulosens Bekæmpelse, (1914-1940).

dispensaries must outweigh the cost, but it is still interesting to get a sense of the magnitude of the benefits. The newest estimates on value of a life-year in Denmark today is just above 190,000 dollars.[48] However, it is well established both theoretically and empirically that the value of life should be viewed as a normal good in the sense that it increases with income (Hammitt and Robinson, 2011). Studies investigating the relationship between the value of life and income in Denmark are unavailable. We therefore turn to the study of the US by Costa and Kahn (2004). They estimate that the elasticity of the value of life with respect to the per capita gross national product is between 1.5 and 1.7 based on data from 1940 to 1980. To be conservative, we use the upper bound of this estimate and the growth rate of income per capita in Denmark from 1908 to 2007, which implies a value of a life-year in 1908 of 6,977 dollars.[49] Based on the estimate of the 1908 value of a life-year, the total benefit of the dispensaries amounts to 1,754 million dollars (95 percent confidence interval; 1,459 to 2,048). As shown in Table 11, this amounts to a social rate of return of 92 to 1 (95 percent confidence interval; 76:1 to 107:1).

Table 11: Cost-benefits of TB dispensaries

While the economic benefits of the dispensaries clearly outweigh their costs, it also possible that the reduction in the incidence and mortality of TB had indirect positive effects on average working productivity. Therefore, along the lines of previous studies on how health interventions influence economic productivity, human capital, etc. (e.g., Bleakley, 2007; Bütikofer and Salvanes 2015), we finally study whether the dispensaries affected working productivity. For this purpose, we digitized city level annual tax income (per tax payer) from 1904 to 1939. Table 12 presents the results from regressing log tax income per tax payer on the rollout of the dispensaries (using our baseline treatment measure and years since adoption), city and year fixed effects. While the baseline effect, reported in column (1), is only borderline significant, the point estimate suggests that an opening of a dispensary increases income by two percent. Column (2) exploits

[48] See the Danish Environmental Economic Council: www.dors.dk/files/media/rapporter/2016/M16/m16.pdf.

[49] The calculated value of a life year in 1908 is derived from the CES function $VOLY_{1908} = VOLY_{2015}\left(Y_{1908}/Y_{2015}\right)^{\epsilon}$, where ϵ is the income elasticity, $VOLY_t$ is the value of a life year, and Y_t is the income per capita, where the subscript t denotes the year. Income per capita data are from the Maddison Project, see `http://www.ggdc.net/maddison/maddison-project/home.htm`. Because of data limitations, we use the ratio $\left(Y_{1908}/Y_{2007}\right)^{\epsilon}$ to deflate the value of a life year in 2015, as the Maddison Project only provides income per capita data up until 2010, and according to Statistics Denmark GDP in Denmark in 2007 were at the highest level recorded so far. Income per capita in Denmark grew 606 percent from 1908 to 2007.

years since the dispensary opened as the treatment measure, implying that the estimated coefficient measures how the opening of a dispensary influenced the annual growth rate of income afterwards. Our estimate, which is now significant at the one percent level, reveals that a dispensary increases the annual income growth by 0.4 percentage points. The fact that only the latter estimate is significant reflects that the income effect takes some years to materialize and is generally increasing over time. We think that the most plausible interpretation of the income estimates is that workers have fewer sick days as TB declines.

Table 12: Productivity effects of TB dispensaries

9 Concluding remarks

This research has shown that the introduction of TB dispensaries reduced TB mortality. This holds both in a DiD setup using time and city variation as well as when deploying DiDiD estimation. In both models, we provide indirect support of the main identifying assumption by showing that trends are parallel prior to treatment and a series of robustness checks.

We show that the most likely interpretation of these findings is that dispensaries are spreading knowledge to people on how to avoid transmitting and contracting TB, and in this way our results grant an important role for public-health in this form for the observed decline in TB prior to the advent of modern medicine. Yet, we acknowledge that increased income and nutrition, as stressed by McKeown (1976), could have been important as well, and our research is not designed to answer the question as to whether the TB dispensaries had a stronger impact than improved nutrition. In addition, we note that other public-health institutions such as TB hospitals and sanatoria seem to have had little on the development of TB over this period. While our study does not as such aim to evaluate these other type of TB institutions, these results can be seen as suggestive that not all public-health institutions mattered equally.

We also showed that the dispensaries were very cost effective compared to other public-health interventions previously evaluated in the literature, and we take our analysis one step further (compared to these studies) by demonstrating positive spillover effects to working productivity. To our mind, the most plausible interpretation of these positive productivity effects is that when TB declines, workers have fewer sick days because of TB infection.

Our research also provides some reason for believing that TB dispensaries could be an important, relatively cheap public intervention for combating TB in developing countries in which the disease is still highly prevalent. This knowledge is important as drug resistant strains have come into existence, however, we also acknowledge that transplanting TB dispensaries to developing countries is not a trivial task. Finally, it could be argued that the TB dispensaries studied were specific to Denmark, but the institution was transplanted from Germany, and that it was present in many other European countries and the US For this reason, our results are also suggestive on what role they could have played elsewhere.

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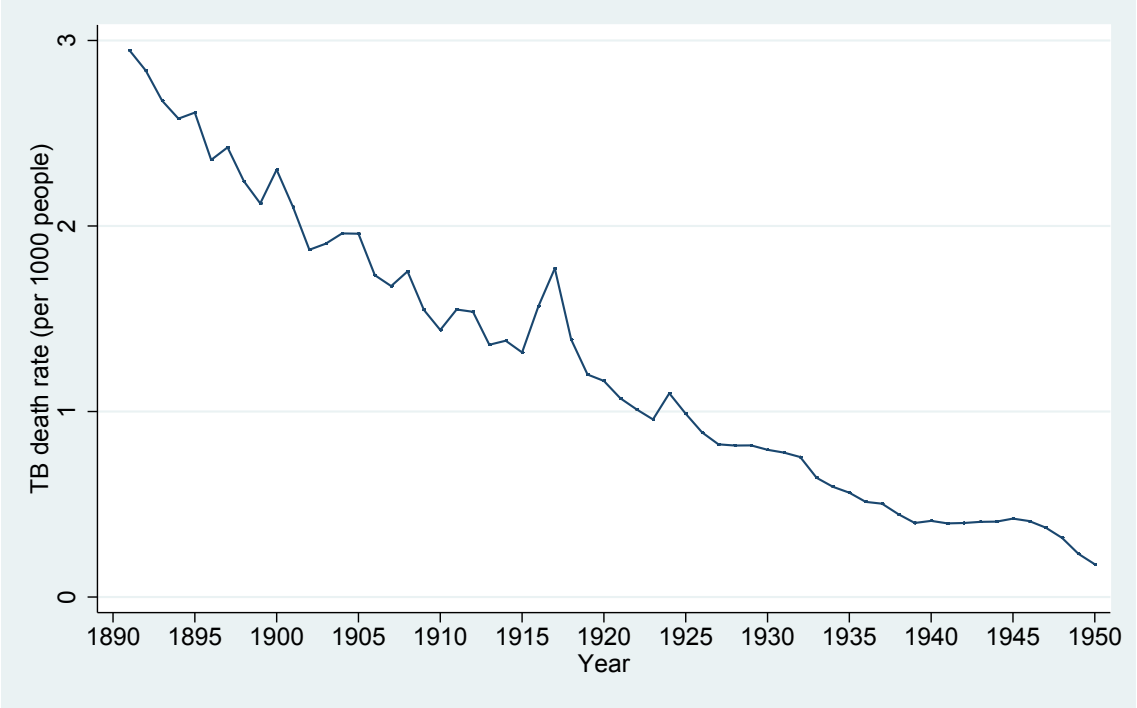
Sundhedsstyrelsen, Dødsaarsagerne i Kongeriget Danmarks byer, 1890-1919.

Sundhedsstyrelsen, Dødsaarsagerne i Kongeriget Danmark, 1920-1950.

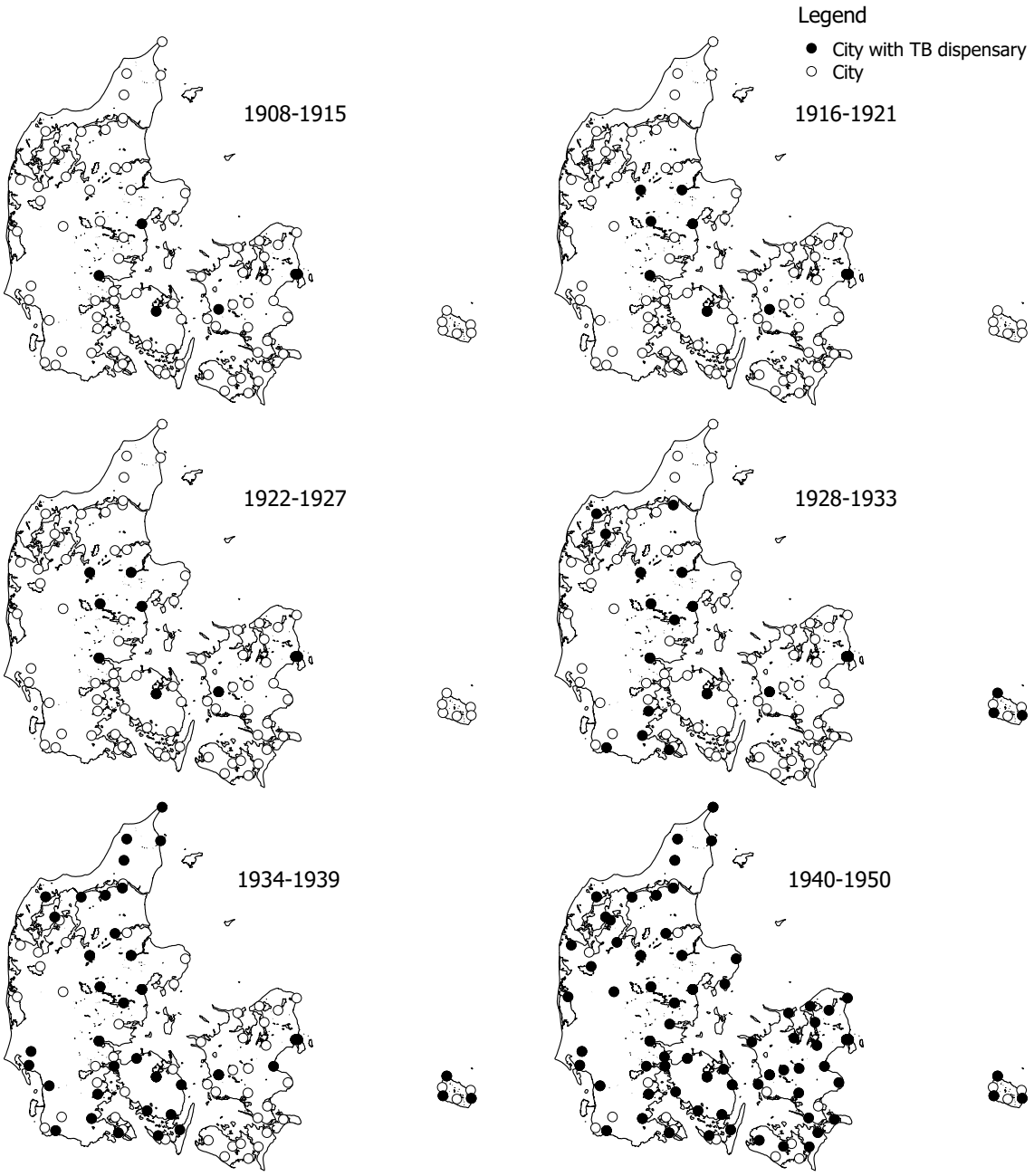
Sundhedsstyrelsen, Medicinalberetningen for Kongeriget Danmark, 1890-1950.

Figure 1: TB mortality in the Danish cities, 1890-1950



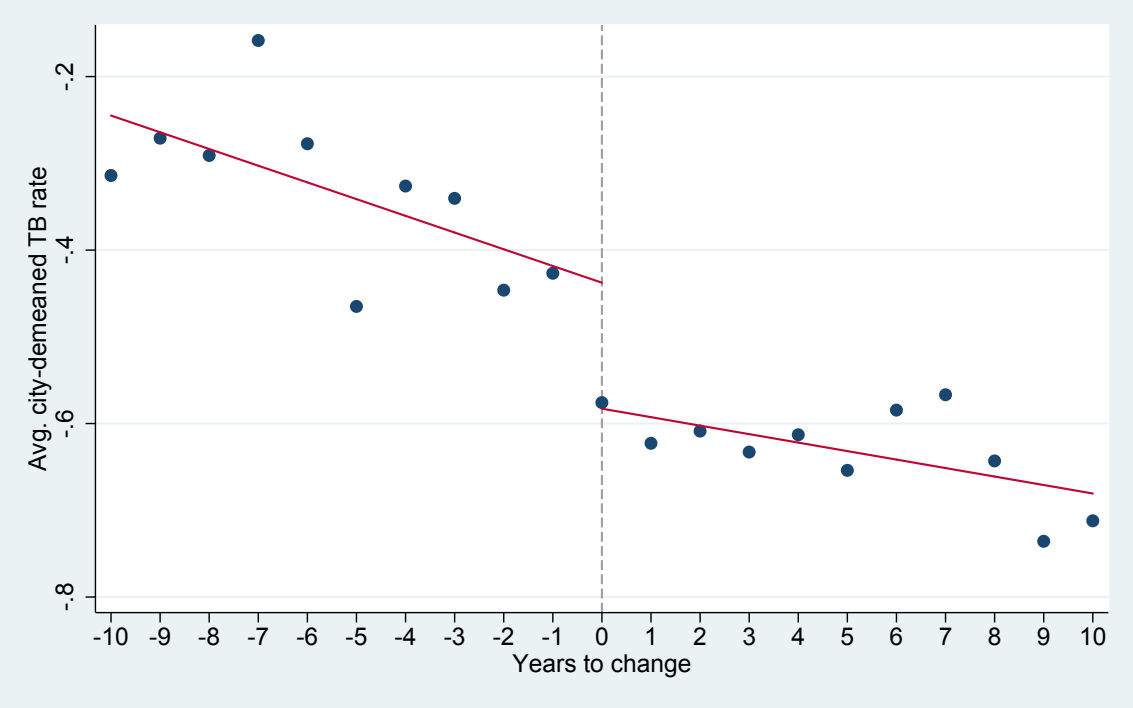
Notes: The graph show the national development of TB mortality in the Danish cities per 1,000 people. Source: the Cause of Death Statistics (1890-1950) and the authors own calculations.

Figure 2: Spread of TB dispensaries across Danish cities



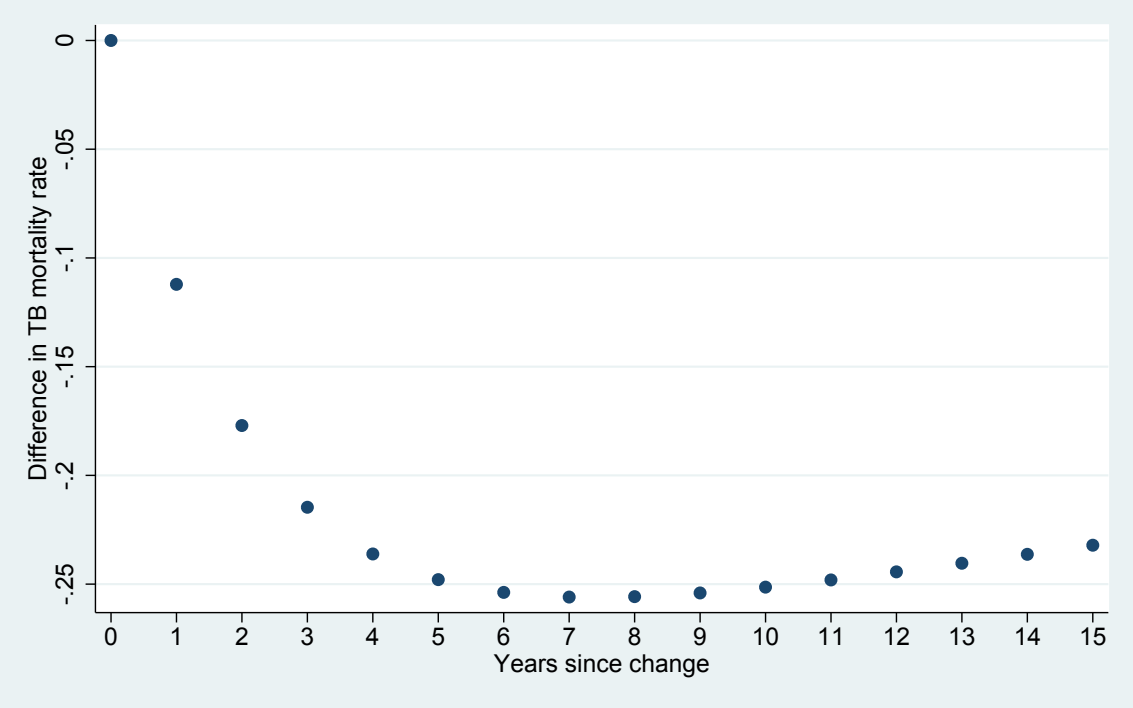
Notes: The maps show cities in the sample, and when TB dispensaries were established. Source: the Medical Reports (1890-1950).

Figure 3: Average city-demeaned TB rate before and after TB dispensary



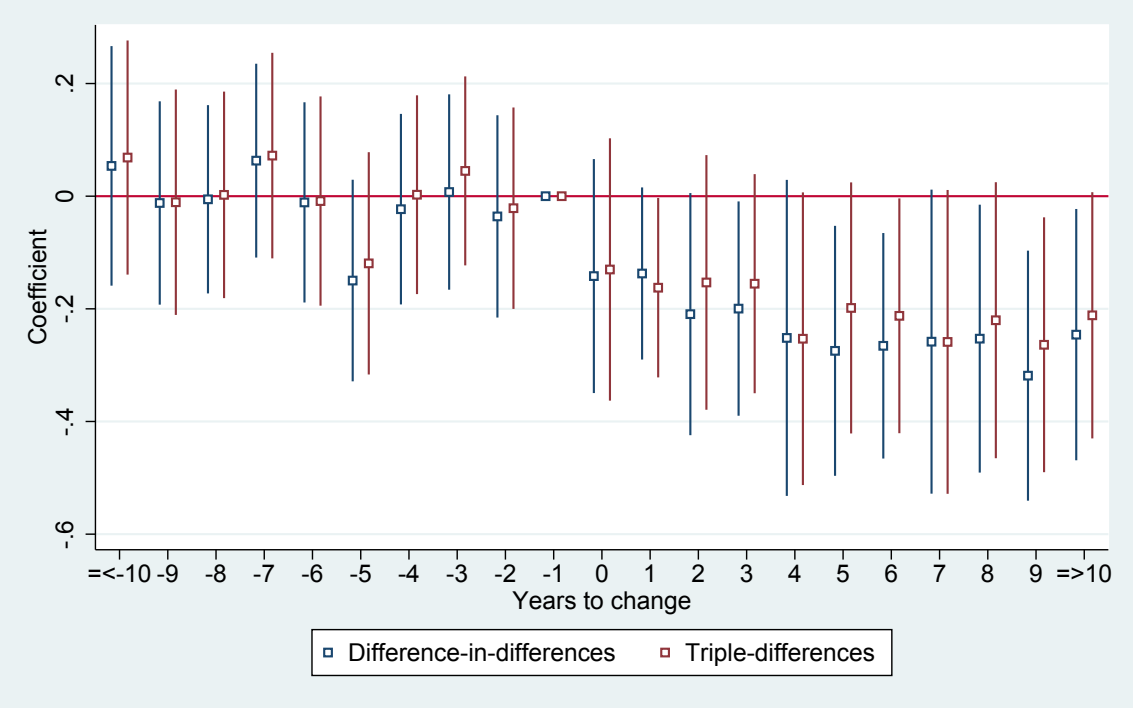
Notes: The graph shows the average city-demeaned TB rate before and after the introduction of a TB dispensary in a city, marked by the vertical dashed line, for the period 1890-1939. The red line is the linear prediction of the TB rate before and after the dispensary, formed by regressing the average city-demeaned TB rate on a constant, a time trend, an indicator equal to one after the introduction of the dispensary, and a second time trend interacted with the indicator. The coefficient on the indicator is -0.145 (standard error $= 0.055$), the coefficient on the trend interacted with the indicator is 0.009 (standard error $= 0.007$).

Figure 4: Simulation of TB mortality rate



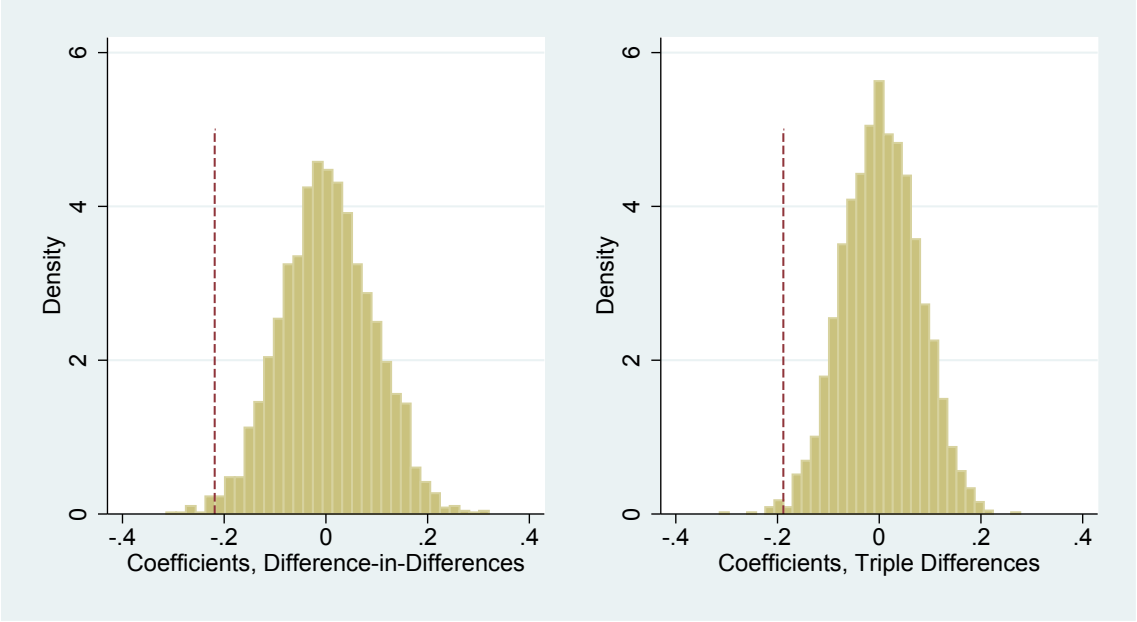
Notes: The graph shows the simulated differences bewteen the path of the TB mortality rate with dispensaries and the counterfactual in the SIR model.

Figure 5: Event study estimates of impact on TB before and after TB dispensary



Notes: The graph shows the $\hat{\beta}_j$ coefficients and their 95% confidence interval from estimating equation (3) and (4) with year $\tau - 1$ as baseline, the year before the intervention. The estimated coefficients are shown in Table A2 in the Appendix.

Figure 6: The distributions of coefficients from random placebo test



Notes: The left and right graph show the distribution of the coefficient on the TB dispensary after estimating the difference-in-differences model of equation (1), and the triple differences model of equation (2) and after reestimating the models after the commissioning date of the TB dispensaries are randomly reshuffled 2,499 times respectively. The stippled vertical lines show the placement of the coefficients from running the difference-in-differences and triple differences model with the true commissioning date. The area to the left of the stippled lines are 0.0072 and 0.0056 for the difference-in-differences, and the triple differences models respectively.

Table 1: Summary statistics

Variable	Period	No. Obs.	Mean	Std. Dev.	Min	Max
TB rate	1890–1939	3,981	1.305	1.036	0.000	7.123
Death rate	1890–1939	3,981	13.931	3.729	2.500	42.740
TB rate	1901–1939	3,165	1.033	0.804	0.000	6.667
Pulmonary rate	1901–1939	3,165	0.789	0.661	0.000	5.556
Birth rate	1901–1939	3,165	22.348	5.766	1.429	45.455
Child mortality rate	1901–1939	3,165	113.136	53.063	0.000	583.333
Death rate	1901–1939	3,165	12.978	2.912	2.500	33.333
Accident and suicide rate	1901–1939	3,165	0.472	0.396	0.000	3.077
Cancer rate	1901–1939	3,165	1.421	0.746	0.000	13.333
Diphtheria rate	1901–1939	3,165	0.081	0.202	0.000	2.210
Influenza rate	1901–1939	3,165	0.347	0.699	0.000	9.630
Pneumonia rate	1901–1939	3,165	1.250	0.755	0.000	6.667
Polio rate	1901–1939	3,165	0.004	0.034	0.000	0.625
Scarlet fever rate	1901–1939	3,165	0.026	0.123	0.000	3.268

Notes: This table report summary statistics for the main variables used in the regression analysis. See Table A1 in the Appendix for the definition of the variables.

Table 2: Effect of TB dispensaries using city by year data on TB

Dep. variable:	TB rate	log(TB rate)	TB rate	TB rate	Pulmonary rate
	(1)	(2)	(3)	(4)	(5)
TB dispensary$_{c,t}$	−0.2187***	−0.1587***	−0.1885***	−0.1539***	−0.1680***
	(0.0774)	(0.0533)	(0.0658)	(0.0539)	(0.0569)
TB rate$_{c,t-1}$			0.1489***	0.1190***	
			(0.0245)	(0.0234)	
TB rate$_{c,t-2}$				0.0417**	
				(0.0205)	
TB rate$_{c,t-3}$				0.1041***	
				(0.0248)	
TB rate$_{c,t-4}$				0.0554**	
				(0.0223)	
TB rate$_{c,t-5}$				0.0276	
				(0.0202)	
Avg. dep. var.	1.3055	0.1014	1.280	1.1708	0.7886
Long run effect			−0.2215***	−0.2360***	
City FE	Yes	Yes	Yes	Yes	Yes
Year FE	Yes	Yes	Yes	Yes	Yes
Time period	1890-1939	1890-1939	1891-1939	1895-1939	1901-1939
Observations	3,981	3,662	3,894	3,546	3,165
R-squared	0.5333	0.5801	0.5389	0.5208	0.3886
Cities	87	87	87	87	87

Notes: The table reports least squares estimates. In column (1), (3), and (4) the left-hand-side variable is the TB mortality per 1,000 people, in column (2) the log is taken of this variable, and in column (5) the left-hand-side variable is the pulmonary TB mortality per 1,000 people. All regressions include city and year fixed effects. TB dispensary$_{c,t}$ is an indicator variable equal to one after the introduction of a TB dispensary, and TB rate$_{c,t-i}$ is the i year lagged dependent variable. Long run effect is the steady-state value of the estimated model in column (3), and (4). Robust standard errors clustered at the city level are in parentheses.
*, **, and ***, determine significance levels of 10%, 5% og 1% respectively.

Table 3: Placebo outcome regressions

	Baseline	Placebo						
Dep. variable:	TB rate	Cancer rate	Influen-za rate	Pneumo-nia rate	Accident and suicide rate	Scarlet fever rate	Diphthe-ria rate	Polio rate
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
TB dispensary$_{c,t}$	−0.2089***	−0.0390	−0.0195	−0.0739	−0.0077	−0.0076	0.0083	−0.0032
	(0.0671)	(0.0516)	(0.0266)	(0.0580)	(0.0257)	(0.0064)	(0.0148)	(0.0025)
Avg. dep. var.	1.0335	1.4211	0.3471	1.2502	0.4715	0.0262	0.0813	0.0045
City FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Year FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Time period	1901-1939	1901-1939	1901-1939	1901-1939	1901-1939	1901-1939	1901-1939	1901-1939
Observations	3,165	3,165	3,165	3,165	3,165	3,165	3,165	3,165
R-squared	0.4394	0.1535	0.5957	0.1900	0.0970	0.0878	0.1100	0.0906
Cities	87	87	87	87	87	87	87	87

Notes: The table reports least squares estimates. In column (1) the left-hand-side variable is the TB mortality per 1,000 people, in column (2) it is the cancer mortality per 1,000 people, in column (3) it is the influenza mortality per 1,000 people, in column (4) it is the pneumonia mortality per 1,000 people, in column (5) it is the accidents and suicides deaths per 1,000 people, and from 1931 including hommicides, in column (6) it is the scarlet fever mortality per 1,000 people, in column (7) it is the diphtheria mortality per 1,000 people, and in column (8) it is the polio mortality per 1,000 people. All regressions include city and year fixed effects. TB dispensary$_{c,t}$ is an indicator variable equal to one after the introduction of a TB dispensary. Robust standard errors clustered at the city level are in parentheses.
*, **, and ***, determine significance levels of 10%, 5% og 1% respectively.

Table 4: Effect of TB dispensaries using city by year data on other outcomes

Dep. variable:	Child mortality rate	log(Child mortality rate)	Death rate	log(Death rate)	Birth rate	log(Birth rate)
	(1)	(2)	(3)	(4)	(5)	(6)
TB dispensary$_{c,t}$	-12.7606^{***}	-0.1058^{***}	-0.5143^{*}	-0.0377^{*}	0.2891	0.0276
	(4.0883)	(0.0391)	(0.2839)	(0.0213)	(0.4991)	(0.0256)
Avg. dep. var.	113.1356	4.6531	13.9311	2.6013	22.3478	3.0713
City FE	Yes	Yes	Yes	Yes	Yes	Yes
Year FE	Yes	Yes	Yes	Yes	Yes	Yes
Time period	1901-1939	1901-1939	1890-1939	1890-1939	1901-1939	1901-1939
Observations	3,165	3,089	3,981	3,981	3,165	3,165
R-squared	0.3020	0.2991	0.4852	0.4671	0.6712	0.6508
Cities	87	87	87	87	87	87

Notes: The table reports least squares estimates. In column in column (1) the left-hand-side variable is the mortality among 0 to 5 year old per 1,000 live births from 1901 to 1936 and from 1937 to 1939 the mortality among 0 to 4 year old per 1,000 live births, in column (2) the log is taken of this variable, in column (3) the left-hand-side variable is the mortality excluding still borns per 1,000 people, in column (4) the log is taken of this variable, in column (5) the left-hand-side variable is the number of births excluding still borns per 1,000 people, and in column (6) the log is taken of this variable. All regressions include city and year fixed effects. TB dispensary$_{c,t}$ is an indicator variable equal to one after the introduction of a TB dispensary. Robust standard errors clustered at the city level are in parentheses.
*, **, and ***, determine significance levels of 10%, 5% og 1% respectively.

Table 5: Effect of TB dispensaries using city by year by disease data

Dep. variable:	Disease		
	(1)	(2)	(3)
TB dispensary$_{c,t}$	−0.1885***	−0.1795***	−0.1885***
	(0.0653)	(0.0633)	(0.0626)
Disease$_{c,t-1}$		0.0224	0.0111
		(0.0167)	(0.0173)
Disease$_{c,t-2}$			0.0440***
			(0.0137)
Disease$_{c,t-3}$			0.0139
			(0.0142)
Disease$_{c,t-4}$			0.0132
			(0.0121)
Disease$_{c,t-5}$			−0.0076
			(0.0116)
Avg. TB rate	1.0335	1.0125	0.9231
Long run effect		−0.1837***	−0.2036***
City FE	Yes	Yes	Yes
Year FE	Yes	Yes	Yes
Disease FE	Yes	Yes	Yes
Disease FE × Year FE	Yes	Yes	Yes
Disease FE × City FE	Yes	Yes	Yes
City FE × Year FE	Yes	Yes	Yes
Time period	1901-1939	1902-1939	1906-1939
Observations	25,320	24,624	21,840
R-squared	0.6969	0.6973	0.7060
Cities	87	87	87

Notes: The table reports least squares estimates. In column (1) to (3) the left-hand-side variable is the stacked causes of death from TB, cancer, influenza, pneumonia, accidents and suicides (including homicides from 1931), scarlet fever, diphtheria, and polio per 1,000 people. All regressions include city, year, disease, disease-by-year, disease-by-city, and city-by-year fixed effects. TB dispensary$_{c,t}$ is an indicator variable equal to one after the introduction of a TB dispensary multiplied by an indicator equal to one if the disease on the left-hand-side is TB, and Disease$_{c,t-i}$ is the i year lagged dependent variable. Robust standard errors clustered at the city level are in parentheses.

*, **, and ***, determine significance levels of 10%, 5% og 1% respectively.

Table 6: Effect of TB dispensaries controlling for neighboring dispensaries

Dep. variable:	TB rate	Disease	TB rate	Disease	TB rate	Disease
	(1)	(2)	(3)	(4)	(5)	(6)
TB dispensary$_{c,t}$	−0.2220***	−0.1922***	−0.2574***	−0.2405***	−0.2448**	−0.2388***
	(0.0767)	(0.0652)	(0.0819)	(0.0685)	(0.0932)	(0.0764)
TB dispensary$_{c,t,10}$	−0.0461	−0.0452				
	(0.2210)	(0.1687)				
TB dispensary$_{c,t,30}$			−0.1193	−0.1577*		
			(0.0936)	(0.0816)		
TB dispensary$_{c,t,50}$					−0.0475	−0.0935
					(0.0778)	(0.0644)
Avg. TB rate	1.3055	1.0335	1.3055	1.0335	1.3055	1.0335
City FE	Yes	Yes	Yes	Yes	Yes	Yes
Year FE	Yes	Yes	Yes	Yes	Yes	Yes
Disease FE	No	Yes	No	Yes	No	Yes
Disease FE × Year FE	No	Yes	No	Yes	No	Yes
Disease FE × City FE	No	Yes	No	Yes	No	Yes
City FE × Year FE	No	Yes	No	Yes	No	Yes
Time period	1890-1939	1901-1939	1890-1939	1901-1939	1890-1939	1901-1939
Observations	3,981	25,320	3,981	25,320	3,981	25,320
R-squared	0.5333	0.6969	0.5340	0.6971	0.5334	0.6970
Cities	87	87	87	87	87	87

Notes: The table reports least squares estimates. In column (1), (3), and (5) the left-hand-side variable is the TB mortality per 1,000 people, and in column (2), (4), and (6) the left-hand-side variable is the stacked causes of death from TB, cancer, influenza, pneumonia, accidents and suicides (including homicides from 1931), scarlet fever, diphtheria, and polio per 1,000 people. All regressions include city and year fixed effects, and addtionally column (2), (4), and (6) include disease, disease-by-year, disease-by-city, and city-by-year fixed effects. TB dispensary$_{c,t}$ is an indicator variable equal to one after the introduction of a TB dispensary, and TB dispensary$_{c,t,r}$ is an indicator variable equal to one after the introduction of a TB dispensary in a city within a r kilometer radius onless TB dispensary$_{c,t} = 1$, multiplied by an indicator equal to one if the disease on the left-hand-side is TB respectively. Robust standard errors clustered at the city level are in parentheses.
*, **, and ***, determine significance levels of 10%, 5% og 1%

Table 7: Effect of TB dispensaries controlling for commissioning of TB hospitals and sanatoria

Dep. variable:	TB rate	Disease	TB rate	Disease	TB rate	Disease	TB rate	Disease
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
TB dispensary$_{c,t}$	−0.2206***	−0.1888***	−0.2125**	−0.1888**	−0.2234***	−0.1902***	−0.3014**	−0.2634***
	(0.0771)	(0.0653)	(0.0868)	(0.0729)	(0.0787)	(0.0656)	(0.1293)	(0.0957)
Sanatorium$_{c,t}$	0.2452	0.0971	0.2758	0.0971				
	(0.1753)	(0.1532)	(0.2310)	(0.1930)				
TB dispensary$_{c,t}$ × Sanatorium$_{c,t}$			−0.0633	0.0000				
			(0.1911)	(0.1284)				
TB hospital$_{c,t}$					0.0186	0.0147	0.0074	0.0034
					(0.0378)	(0.0365)	(0.0429)	(0.0428)
TB dispensary$_{c,t}$ × TB hospital$_{c,t}$							0.0499	0.0488
							(0.0565)	(0.0480)
City FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Year FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Disease FE	No	Yes	No	Yes	No	Yes	No	Yes
Disease FE × Year FE	No	Yes	No	Yes	No	Yes	No	Yes
Disease FE × City FE	No	Yes	No	Yes	No	Yes	No	Yes
City FE × Year FE	No	Yes	No	Yes	No	Yes	No	Yes
Time period	1890-1939	1901-1939	1890-1939	1901-1939	1890-1939	1901-1939	1890-1939	1901-1939
Observations	3,981	25,320	3,981	25,320	3,981	25,320	3,981	25,320
R-squared	0.5338	0.6969	0.5338	0.6969	0.5334	0.6969	0.5336	0.6969
Cities	87	87	87	87	87	87	87	87

Notes: The table reports least squares estimates. In column (1), (3), (5), and (7) the left-hand-side variable is the TB mortality per 1,000 people, in column (2), (4), (6), and (8) the left-hand-side variable is the stacked causes of death from TB, cancer, influenza, pneumonia, accidents and suicides (including homicides from 1931), scarlet fever, diphtheria, and polio per 1,000 people. All regressions include city and year fixed effects, and addtionally column (2), (4), (6), and (8) include disease, disease-by-year, disease-by-city, and city-by-year fixed effects. TB dispensary$_{c,t}$ is an indicator variable equal to one after the introduction of a TB dispensary, TB hospital$_{c,t}$ counts the number of TB hospitals within a county at time t, Sanatorium$_{c,t}$ is the sum of the reciprocal distance to every sanatorium within a county from city c, and TB dispensary$_{c,t}$ × TB hospital$_{c,t}$ and TB dispensary$_{c,t}$ × Sanatorium$_{c,t}$ are interactions of the aforementioned variables. All right hand side variables are multiplied by an indicator equal to one if the disease on the left-hand-side is TB. Robust standard errors clustered at the city level are in parentheses.
*, **, and ***, determine significance levels of 10%, 5% og 1% respectively.

Table 8: Effect of TB dispensaries controlling for pre-intervention income and inequality

Dep. variable:	TB rate	Disease	TB rate	Disease	TB rate	Disease
	Baseline panel		Excluding pre 1920 adopters		Excluding pre 1920 adopters	
	(1)	(2)	(3)	(4)	(5)	(6)
TB dispensary$_{c,t}$	-0.2310^{***}	-0.2029^{***}	-0.2362^{**}	-0.2230^{***}	-0.2268^{**}	-0.2147^{**}
	(0.0757)	(0.0619)	(0.0992)	(0.0837)	(0.1088)	(0.0889)
Avg. TB rate	1.3055	1.0335	1.3091	1.0339	1.3091	1.0339
Pre-int. log(income) × Year Fe	Yes	Yes	No	No	Yes	Yes
Pre-int. Gini × Year Fe	No	No	Yes	Yes	Yes	Yes
City FE	Yes	Yes	Yes	Yes	Yes	Yes
Year FE	Yes	Yes	Yes	Yes	Yes	Yes
Disease FE	No	Yes	No	Yes	No	Yes
Disease FE × Year FE	No	Yes	No	Yes	No	Yes
Disease FE × City FE	No	Yes	No	Yes	No	Yes
City FE × Year FE	No	Yes	No	Yes	No	Yes
Time period	1890-1939	1901-1939	1890-1939	1901-1939	1890-1939	1901-1939
Observations	3,981	25,320	3,341	20,992	3,341	20,992
R-squared	0.5406	0.6978	0.5113	0.6919	0.5214	0.6931
Cities	87	87	68	68	68	68

Notes: The table reports least squares estimates. In column (1), (3), and (5) the left-hand-side variable is the TB mortality per 1,000 people, and in column (2), (4), and (6) the left-hand-side variable is the stacked causes of death from TB, cancer, influenza, pneumonia, accidents and suicides (including homicides from 1931), scarlet fever, diphtheria, and polio per 1,000 people. All regressions include city and year fixed effects, and addtionally column (2), (4), and (6) include disease, disease-by-year, disease-by-city, and city-by-year fixed effects. TB dispensary$_{c,t}$ is an indicator variable equal to one after the introduction of a TB dispensary. In column (1), (2), (5), and (6) the 1904-05 log income per capita interacted with year fixed effects is included, and in column (3), (4), (5), and (6) the 1918-19 income distribution based Gini coefficient interacted with year fixed effects is included. Additionally, the TB dispensary$_{c,t}$ indicator, the 1904-05 income, and the 1918-19 Gini coefficient are interacted with an indicator equal to one if the disease on the left-hand-side is TB. For cities included in the dataset later than 1904 and 1918, we use income per tax payer and Gini coefficients in the year they are added to the data. In column (3), (4), (5), and (6) cities which adopted a TB dispensary before 1920 are excluded from the sample. Robust standard errors clustered at the city level are in parentheses.
*, **, and ***, determine significance levels of 10%, 5% og 1% respectively.

Table 9: Treatment heterogeneity of TB dispensaries

Dep. variable:	TB rate	Disease	TB rate	Disease	TB rate	Disease	TB rate	Disease
	Baseline panel						Excluding pre 1920 adopters	
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
TB dispensary$_{c,t}$	−0.1814***	−0.1691***	−0.2355***	−0.2020***	−0.2174***	−0.1868***	−0.2567**	−0.2435***
	(0.0680)	(0.0606)	(0.0787)	(0.0636)	(0.0750)	(0.0638)	(0.0997)	(0.0842)
TB dispensary$_{c,t}$ × Initial TB rate$_c$	−0.1502**	−0.1037*						
	(0.0738)	(0.0572)						
TB dispensary$_{c,t}$ × Pre-int. pop. density$_c$			0.0418*	0.0622**				
			(0.0229)	(0.0285)				
TB dispensary$_{c,t}$ × Pre-int. log(income)$_c$					−0.0572	−0.0597		
					(0.0452)	(0.0424)		
TB dispensary$_{c,t}$ × Pre-int. Gini$_c$							0.0063	−0.0383
							(0.0541)	(0.0453)
Avg. TB rate	1.3055	1.0335	1.3055	1.0335	1.3055	1.0335	1.3091	1.0339
City FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Year FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Disease FE	No	Yes	No	Yes	No	Yes	No	Yes
Disease FE × Year FE	No	Yes	No	Yes	No	Yes	No	Yes
Disease FE × City FE	No	Yes	No	Yes	No	Yes	No	Yes
City FE × Year FE	No	Yes	No	Yes	No	Yes	No	Yes
Time period	1890-1939	1901-1939	1890-1939	1901-1939	1890-1939	1901-1939	1890-1939	1901-1939
Observations	3,981	25,320	3,981	25,320	3,981	25,320	3,341	20,992
R-squared	0.5342	0.6970	0.5335	0.6969	0.5336	0.6969	0.5053	0.6909
Cities	87	87	87	87	87	87	68	68

Notes: The table reports least squares estimates. In column (1), (3), and (5) the left-hand-side variable is the TB mortality per 1,000 people, and in column (2), (4), and (6) the left-hand-side variable is the stacked causes of death from TB, cancer, influenza, pneumonia, accidents and suicides (including homicides from 1931), scarlet fever, diphtheria, and polio per 1,000 people. All regressions include city and year fixed effects, and addtionally column (2), (4), and (6) include disease, disease-by-year, disease-by-city, and city-by-year fixed effects. TB dispensary$_{c,t}$ is an indicator variable equal to one after the introduction of a TB dispensary multiplied by an indicator equal to one if the disease on the left-hand-side is TB. Initial TB rate$_c$, Pre-int. log(income)$_c$, and Pre-int. Gini$_c$ are the 1890 TB mortality per 1,000 people, the 1904-05 log income per capita, and the 1918-19 income distribution based Gini coefficient respectively, where both are standardized to have a zero mean and standard deviation of one. For cities included in the dataset later than 1890, 1904, and 1918, we use the TB mortality rate, income per taxpayer, and Gini coefficients in the year they are added to the dataset. In column (5) and (6) cities which adopted a TB dispensary before 1920 are excluded from the sample. Robust standard errors clustered at the city level are in parentheses.

*, **, and ***, determine significance levels of 10%, 5% og 1% respectively.

Table 10: Effect of TB dispensaries on patients in sanatoria and tuberculosis hospitals

Dep. variable:	log(patients in sanatoria)	patients in sanatoria rate	log(patients in TB hospitals)	patients in TB hospitals rate
	(1)	(2)	(3)	(4)
TB dispensary$_{a,t}$	-0.1390^{**}	-1.8369^{**}	0.0105	0.4168
	(0.0613)	(0.7052)	(0.0964)	(0.2611)
Avg. dep. var.	5.6344	11.5084	4.6065	3.0551
County FE	Yes	Yes	Yes	Yes
Year FE	Yes	Yes	Yes	Yes
Time period	1901-1939	1901-1939	1908-1939	1908-1939
Observations	488	488	504	504
R-squared	0.8882	0.8772	0.8037	0.6352
Counties	18	18	17	17

Notes: The table reports least squares estimates at the county level. In column (1) the left-hand-side variable is the log of patients in santoria, in column (2) the left-hand-side variable is patients in sanatorie per 1,000 people, in column (3) the left-hand-side variable is the log of patients in TB hospitals, and in column (4) the left-hand-side variable is patients in TB hospitals per 1,000 people. The number of patients in any sanatoria are not available for the year 1928. All regressions include county, and year fixed effects. TB dispensary$_{a,t}$ is the number of TB dispensaries in county a at time t. Robust standard errors clustered at the county level are in parentheses.
*, **, and ***, determine significance levels of 10%, 5% og 1% respectively.

Table 11: Cost-benefits of TB dispensaries

	Point estimate	95% CI low	95% CI high
Deaths averted from 1908 to 1939	6,363	5,397	7,328
Life-years saved from 1908 to 1939	251,345	213,212	289,478
Cost of TB dispensaries from 1908 to 1939	19,140,432		
Cost per averted death	3,008	3,546	2,612
Cost per life-year saved	76	90	66
Benefits of TB dispensaries from 1908 to 1939	1,753,675,865	1,487,617,665	2,019,734,066
Social rate of return	92 : 1	78 : 1	106 : 1

Notes: The table reports cost and benefits of the TB dispensaries. Based on the baseline difference-in-differences estimate in Table 3, column (1) the number of death averted by TB dispensaries commissioned between 1908 and 1939 are predicted. The life years saved are calculated by weighting the age distribution of TB mortality from 1921 to 1925 by the age specific life expectancy of Denmark in each year from 1908 to 1939. The total costs of the TB dispensaries from 1908 to 1939 are estimted by the yearly per capita cost in 2015 US dollar implied by the financial accounts of nine TB dispensaries. The cost per averted death and the cost per life-year saved are estimated by divideing the deaths averted adn the life years saved by the total costs respectively. The benefits of the TB dispensaries are calculated by multipliying the 1908 value of a life year (VOLY) by the number of death averted by the TB dispensaries. The 1908 VOLY, is derived from the CES function $VOLY_{1908} = VOLY_{2015}(Y_{1908}/Y_{2007})^{\epsilon}$, where ϵ is the income elasticity assumed to be 1.7, and Y_t is the income per capita peaking in 2007 before the Great Recession. The 2015 VOLY is assummed to be 190,000 US dollars. The social rate of return is calculated by dividing the total benefits with the tocal costs.

Table 12: Effect of TB dispensaries on income

Dep. variable:	log(income pr. tax payer)	
	(1)	(2)
TB dispensary$_{c,t}$	0.0230	
	(0.0159)	
TB dispensary$_{c,t} \times (t + 1 - j_c)$		0.0040***
		(0.0015)
City FE	Yes	Yes
Year FE	Yes	Yes
Time period	1904-1939	1904-1939
Observations	2,940	2,940
R-squared	0.8384	0.8404
Cities	87	87

Notes: The table reports least squares estimates. In column (1) and (2) the left-hand-side variable is the log income per tax payer. All regressions include city, and year fixed effects. TB dispensary$_{c,t}$ is an indicator variable equal to one after the introduction of a TB dispensary, j_c is the year of the introduction of a dispensary, and t is the year. Robust standard errors clustered at the city level are in parentheses.
*, **, and ***, determine significance levels of 10%, 5% og 1% respectively.

Online Appendix:

Preventing the White Death: Tuberculosis Dispensaries

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January 2017

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Mortality by cause and age in Danish cities

This section outlines the aggregate city development of various cause-specific mortality rates (from 1901 to 1950), as well as aggregate TB incidence (from 1920 to 1950) and the age distribution of TB mortality in 1921-1925.

Figure A1: Pulmonary TB mortality in the Danish cities, 1901-1950

Figure A2: Mortality in the Danish cities, 1890-1950

Figure A3: Infant mortality in the Danish cities, 1901-1950

Figure A4: Live births in the Danish cities, 1901-1950

Figure A5: Pneumonia mortality in the Danish cities, 1901-1950

Figure A6: Scarlet fever mortality in the Danish cities, 1901-1950

Figure A7: Cancer mortality in the Danish cities, 1901-1950

Figure A8: Accident and suicide mortality in the Danish cities, 1901-1950

Figure A9: Polio mortality in the Danish cities, 1901-1950

Figure A10: Influenza mortality in the Danish cities, 1901-1950

Figure A11: Diphtheria mortality in the Danish cities, 1901-1950

Figure A12: Pulmonary TB morbidity and mortality in Denmark, 1921-1949

Figure A13: Age distribution of TB mortality, 1921-25

Theoretical framework

This section provides supporting information to the theoretical framework (i.e., the modified SIR model), discussed in the paper. A summary of the model's equations (as outlined in the paper) is given here:

$$S_{t+1} = S_t + B_t - I_t^{New} - D_t^{other} + R_t,\ S_0 \text{ given}$$

$$B_t = bS_t,$$

$$D_t^{other} = \lambda^D S_t,$$

$$I_t^{New} = \lambda_t^I S_t,$$

$$\lambda_t^I = \frac{\beta}{y_t^\gamma}\left(\frac{I_t}{S_t}\right)^\alpha,$$

$$(\sigma - \theta)(1 - \delta)$$

$$y_{t+1} = (1 + g_y)y_t,\ y_0 \text{ given},$$

$$R_t = \lambda_t^R I_t,$$

$$\lambda_t^R = \bar{\lambda}^R - \frac{\Psi}{y_t},$$

$$I_{t+1} = I_t + I_t^{New} - D_t^{TB} - R_t,\ I_0 \text{ given},$$

$$D_t^{TB} = \lambda^{TB} I_t.$$

Our next step is to solve the model. First, using these equations yields to the following system of three first-order difference equations which fully characterise how the populations and income evolve over time:

$$\begin{aligned} S_{t+1} &= \left(1 + b - \lambda^D\right) S_t - \frac{\beta}{y_t^\gamma}\left(\frac{I_t}{S_t}\right)^\alpha S_t + \left(\bar{\lambda}_t^R - \frac{\Psi}{y_t}\right) I_t,\ S_0 \text{ given}, \\ I_{t+1} &= \left(1 - \lambda^{TB} - \left(\bar{\lambda}^R - \frac{\Psi}{y_t}\right)\right) I_t + \frac{\beta}{y_t^\gamma}\left(\frac{I_t}{S_t}\right)^\alpha S_t,\ I_0 \text{ given}, \\ y_{t+1} &= (1 + g_y)y_t,\ y_0 \text{ given}. \end{aligned} \tag{1}$$

Next, assume that $g_y = 0$, $y_{t+1} = y_t = y$, and rewrite the system of the two remaining first order equations in terms of $s_t \equiv I_t/S_t$. We start by the difference equation for I_t:

$$\begin{aligned} I_{t+1} &= \left(1 - \lambda^{TB} - \left(\bar{\lambda}^R - \frac{\Psi}{y}\right)\right) I_t + \frac{\beta}{y^\gamma}\left(\frac{I_t}{S_t}\right)^\alpha S_t \Leftrightarrow \\ G_t^I &\equiv \frac{I_{t+1}}{I_t} = \left(1 - \lambda^{TB} - \left(\bar{\lambda}^R - \frac{\Psi}{y}\right)\right) + \frac{\beta}{y^\gamma}\left(\frac{S_t}{I_t}\right)^{1-\alpha} \Leftrightarrow \\ G_t^I &= 1 - \lambda^{TB} - \bar{\lambda}^R + \frac{\Psi}{y} + \frac{\beta}{y^\gamma} s_t^{\alpha-1}. \end{aligned} \tag{2}$$

And now the difference equation for S_t :

$$\begin{aligned} S_{t+1} &= \left(1 + b - \lambda^D\right) S_t - \frac{\beta}{y^\gamma}\left(\frac{I_t}{S_t}\right)^\alpha S_t + \left(\bar{\lambda}_t^R - \frac{\Psi}{y}\right) I_t \Leftrightarrow \\ G_t^S &\equiv \frac{S_{t+1}}{S_t} = \left(1 + b - \lambda^D - \frac{\beta}{y^\gamma}\left(\frac{I_t}{S_t}\right)^\alpha + \left(\bar{\lambda}_t^R - \frac{\Psi}{y}\right)\frac{I_t}{S_t}\right) \Leftrightarrow \\ G_t^S &= 1 + b - \lambda^D - \frac{\beta}{y^\gamma} s_t^\alpha + \left(\bar{\lambda}_t^R - \frac{\Psi}{y}\right) s_t. \end{aligned} \tag{3}$$

Equations (2) and (3) can now be depicted in a $G, s-$diagram. If these two curves intersect, S and I are growing at the same rates and s becomes constant. Next, equation (2) shows that if $\alpha < 1$ i) G_t^I is falling in s; ii) $G_t^I \to \infty$ when $s \to 0$; and iii) $G_t^I \to 1 - \lambda^{TB} - \bar{\lambda}^R$ as $s \to \infty$. From equation (3), we see that i) G_t^S is decreasing in s if $s < \tilde{s}$, where:

$$\tilde{s} \equiv \left(\frac{\alpha\beta}{y^\gamma\left(\bar{\lambda}_t^R - \frac{\Psi}{y}\right)}\right)^{\frac{1}{1-\alpha}}, \tag{4}$$

and increasing when $s > \tilde{s}$; ii) $G_t^S(0) = 1 + b - \lambda^D$; and iii) $G_t^I \to \infty$ when $s \to \infty$. Now, assuming that $1 - \lambda^{TB} - \bar{\lambda}^R + \frac{\Psi}{y} > 1 + b - \lambda^D - \frac{\beta}{y^\gamma}\tilde{s}^\alpha + \left(\bar{\lambda}_t^R - \frac{\Psi}{y}\right)\tilde{s}$, we know that (3) for sure intersects (2) from below, and we are guaranteed *one* global stable steady state as, in this case, if $s' > s^*$ then $G_t^S > G_t^I$ and S is growing more than I_t, implying that $s_t \equiv S_t/I_t$ falls until $s' = s^*$. Likewise if $s' < s^*$ then $G_t^S < G_t^I$ and S is growing less than I_t, implying that $s_t \equiv S_t/I_t$ increases until $s = s^*$. In other words, if $s^* > \tilde{s}$, s^* is a stable steady state. Figure A14 depicts (2) and (3) under the above-mentioned parameter restrictions, and it shows that s^* is in fact globally stable.

Figure A14: $G, s-$diagram

From the position of the two curves (2 and 3) in the Figure A14, we see how s^* changes when various parameters of the model are changed: $\frac{ds^*}{db} > 0$, $\frac{ds^*}{d\lambda^D} < 0$, $\frac{ds^*}{d\lambda^{TB}} < 0$, $\frac{ds^*}{d\lambda^R} < 0$, $\frac{ds^*}{dy} < 0$, $\frac{ds^*}{d\theta} < 0$, and $\frac{ds^*}{d\delta} < 0$. We note that although the TB incidence rate decreases when λ^{TB} increases (i.e., $ds^*/d\lambda^{TB} < 0$), the TB mortality rate would still increase. So if one finds empirically that the incidence rate and mortality rates go in opposite directions, this could be evidence of that λ^{TB} has changed.

For our simulation exercise, reported in Figure 4 of the paper, we use the following set of parameter values:

$$\begin{aligned} S_0 &= 14,000;\ I_0 = 293.95;\ b = 0.0281;\ \lambda^D = 0.013661;\ \lambda^{TB} = 0.08 \\ \alpha &= \gamma = 0.5;\ y_0 = 1;\ g_y = 0.015;\ \Psi = 0.1;\ \sigma = 0.05;\ \theta_1 = 0;\ \delta = 0 \end{aligned}$$

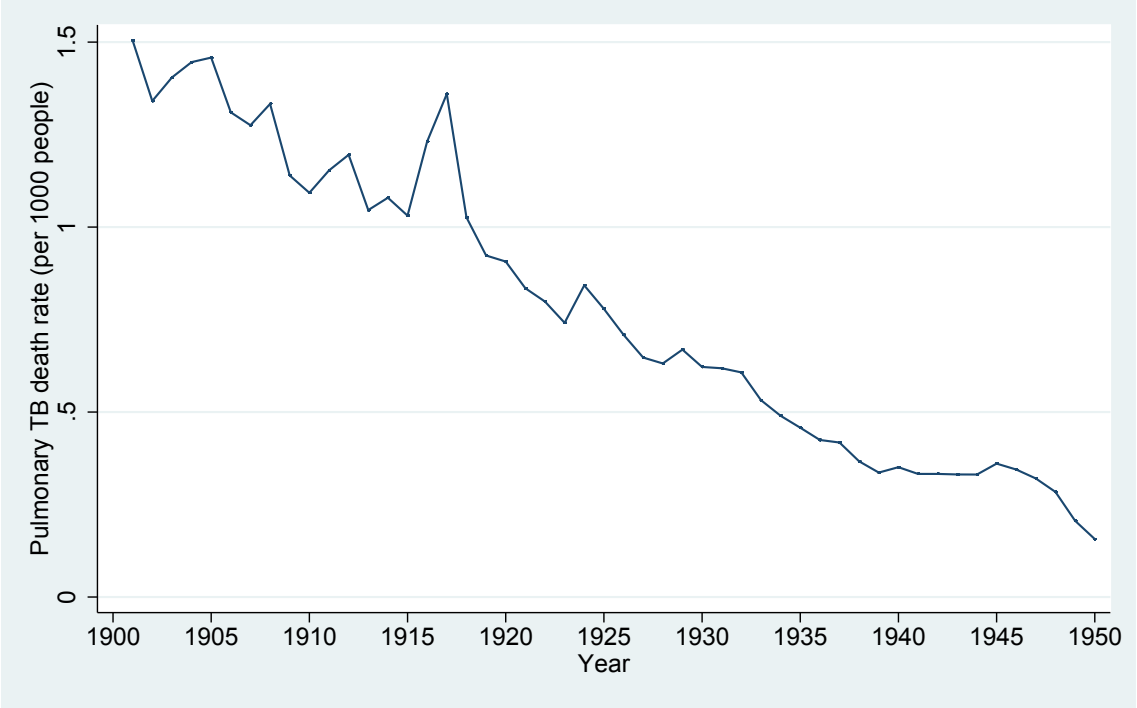
The values of S_0, I_0, b, λ^D are set as to roughly match first moments in our data in 1908 in terms of population size, TB death rate, crude birth rate, and crude death rate. Using Danish TB mortality data for men above the age of 20 at the turn of the 20th century, Heiberg (1902) finds that about 13 percent of all TB deaths occurs within the first year and λ^{TB} is accordingly set below this threshold value at 8 percent. The values α and γ are set so as to get at baseline path for the TB mortality rate which resembles the observed one. We set $y_0 = 1$ and $\Psi = 0.1$ so that we get an annual recovery rate in the interval $[0.4; 0.5]$. We could alternatively have set y_0 to match income per capita from our tax-records data and adjusted Ψ accordingly to get the same interval of recovery rates; of course, this yields to the same findings. The average annual growth rate in (tax) income per capita is the neighborhood of around 1.5 percent per year, so $g_y = 0.015$. Finally, in the baseline, we set $\sigma = 0.05$, $\theta_1 = 0$, and $\delta = 0$, such that the baseline infectivity rate of TB is 5 percent. These parameters setting constitute our baseline path for the TB mortality rate. When simulating the introduction of the dispensaries, θ_1 is increased from zero to $\theta_2 = 0.01$, which lowers the initial infectivity rate to 4 percent. We choose this magnitude in order to mimic the magnitude of the decline in TB mortality as we observe it in the data, however, we note that it still remains interesting to study the shape of the time path due to this parameter change, and see if it look at all as the empirical counterpart.

Data and balancing

Additional event-study figures

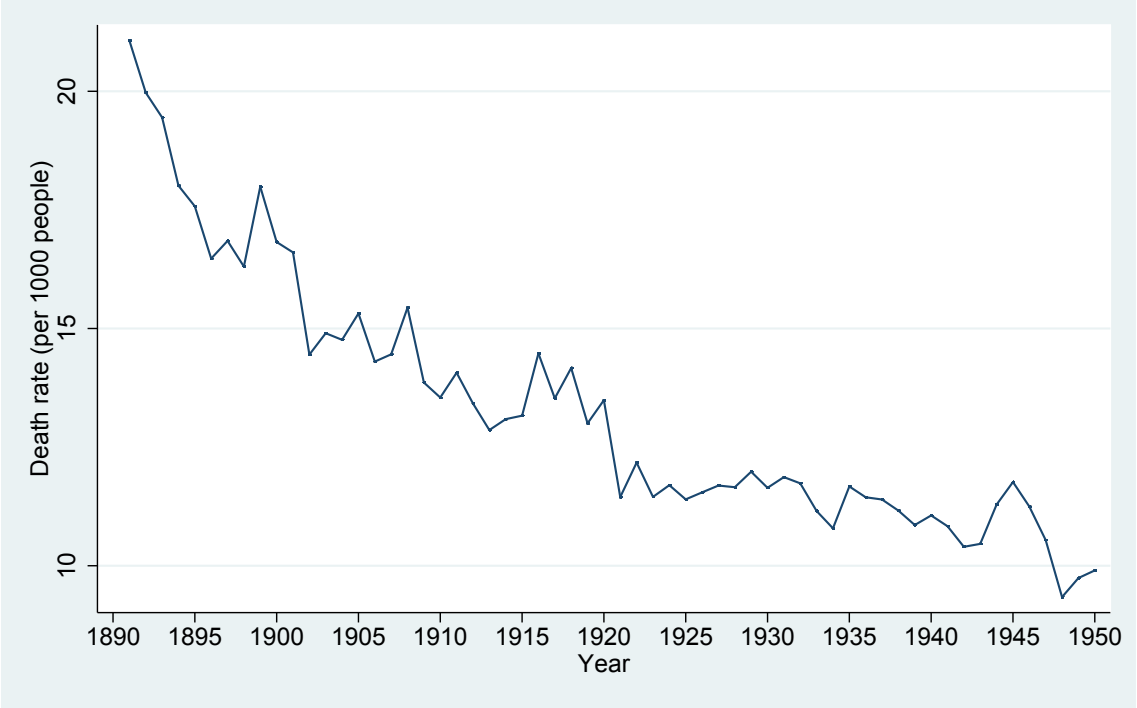
Additional robustness tests

Figure A1: Pulmonary TB mortality in the Danish cities, 1901-1950



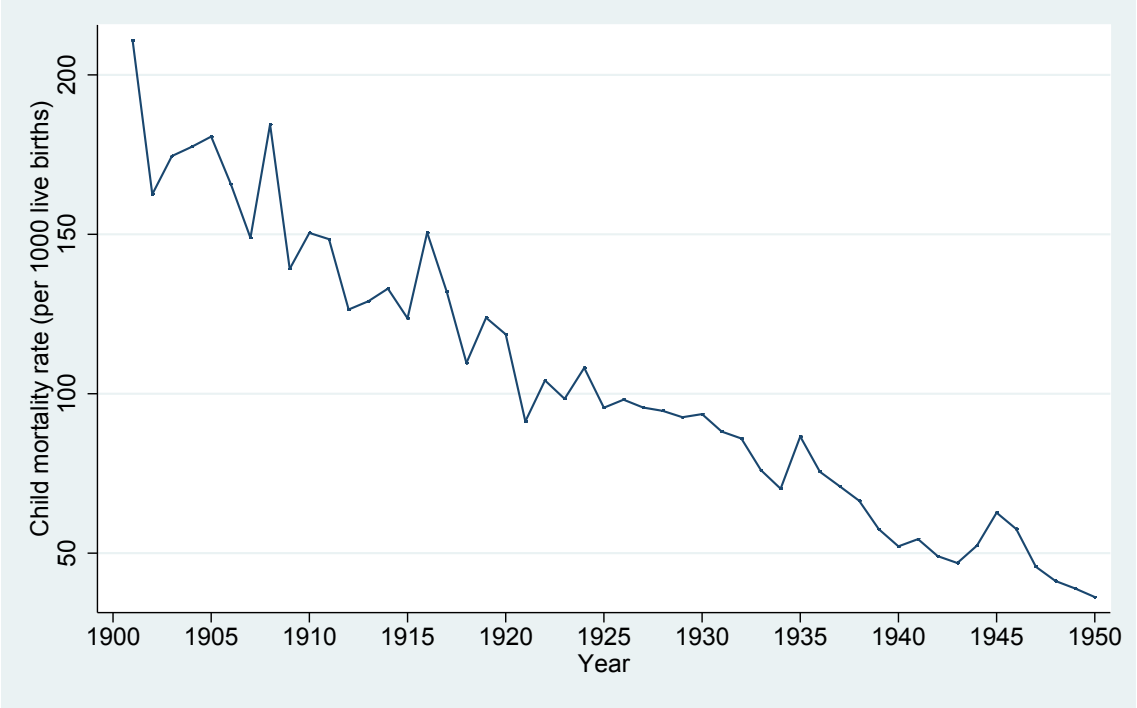
Notes: The graph show the national development of pulmonary TB mortality in the Danish cities per 1,000 people. Source: the Cause of Death Statistics (1901-1950) and the authors own calculations.

Figure A2: Mortality in the Danish cities, 1890-1950



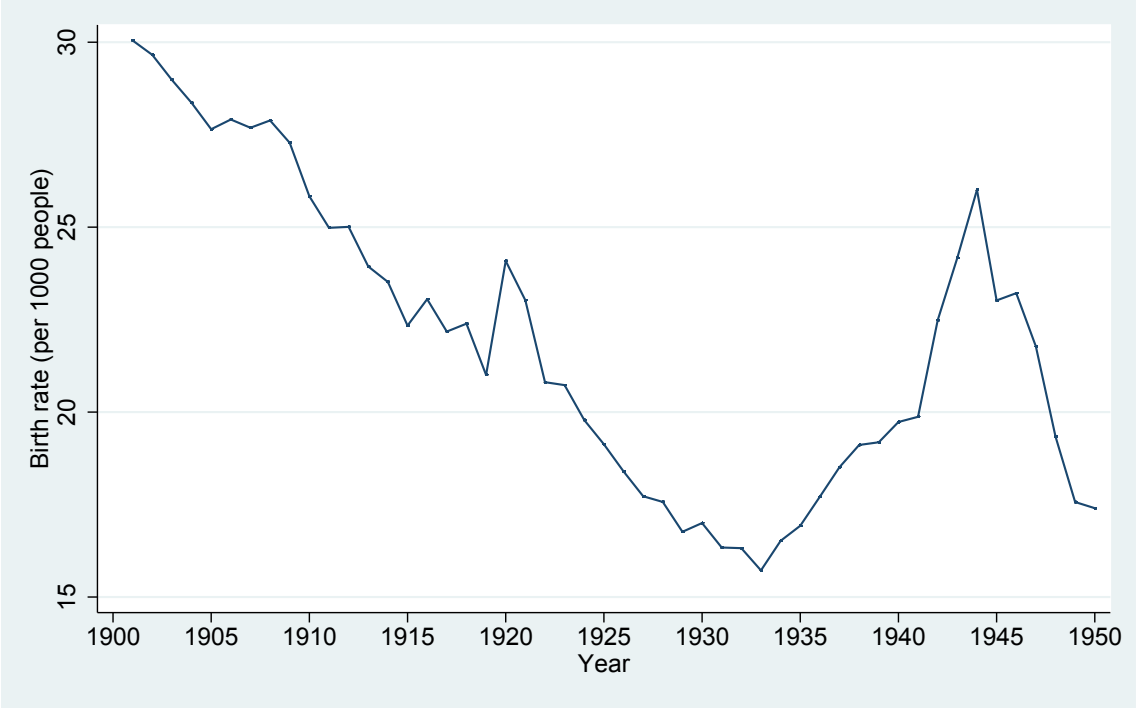
Notes: The graph show the national development of mortality excluding still borns in the Danish cities per 1,000 people. Source: the Cause of Death Statistics (1901-1950) and the authors own calculations.

Figure A3: Child mortality in the Danish cities, 1901-1950



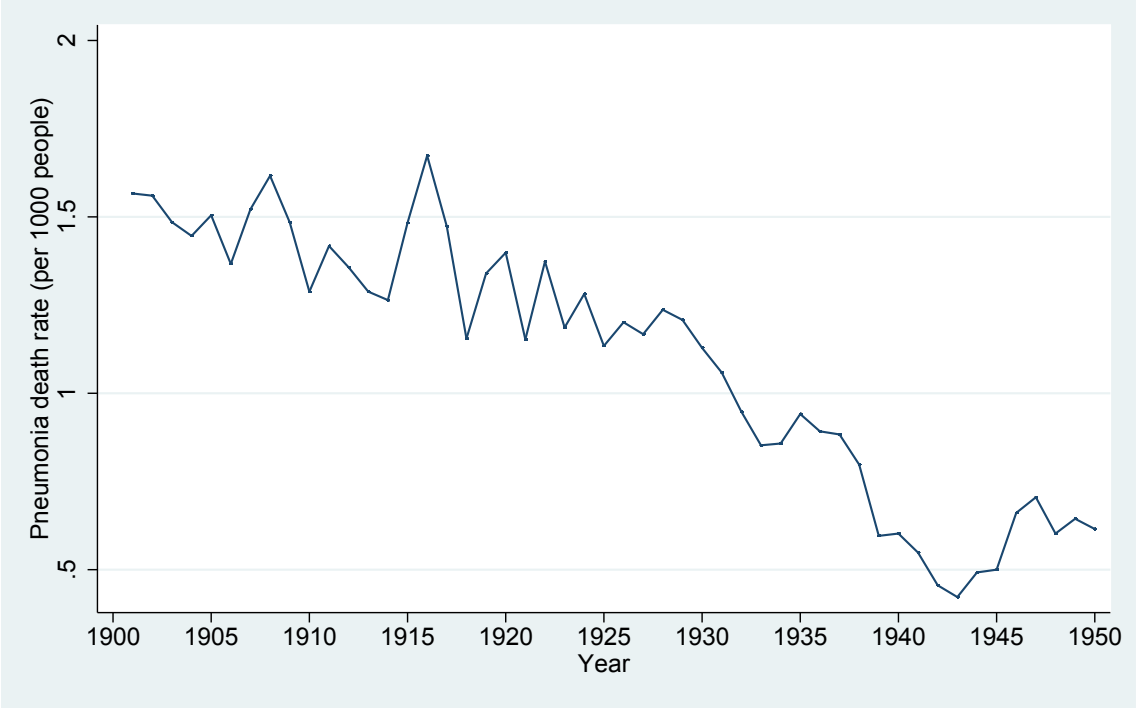
Notes: The graph show the national development of mortality among 0 to 5 year old in the Danish cities per 1,000 live births from 1901 to 1936 annd from 1937 to 1950 the national development of mortality among 0 to 4 year old in the Danish cities per 1,000 live births. Source: the Cause of Death Statistics (1901-1950) and the authors own calculations.

Figure A4: Live births in the Danish cities, 1901-1950



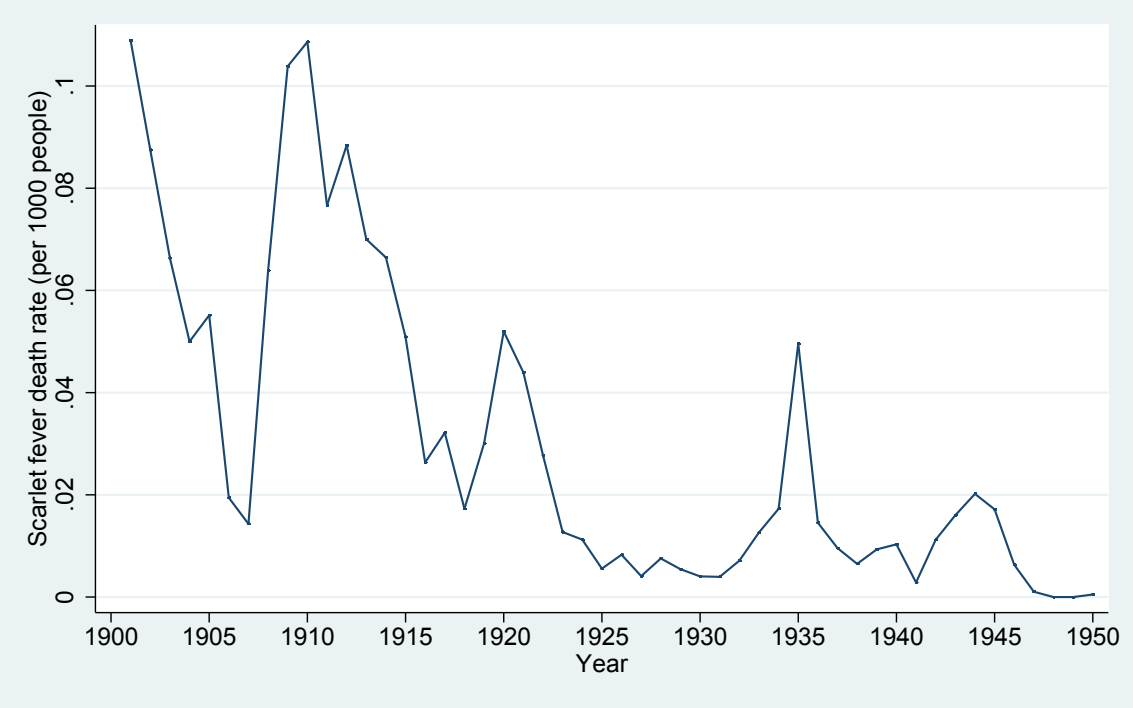
Notes: The graph show the national development of the birth rate in the Danish cities per 1,000 people. Source: the Cause of Death Statistics (1901-1950) and the authors own calculations.

Figure A5: Pneumonia mortality in the Danish cities, 1901-1950



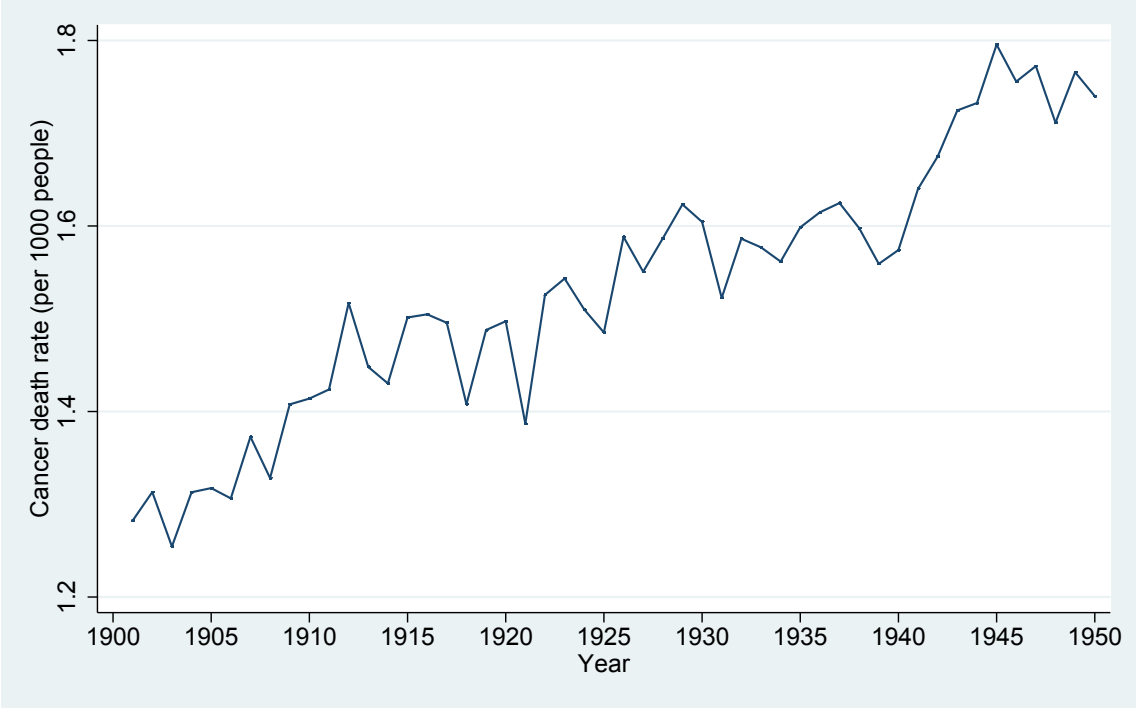
Notes: The graph show the national development of pneumonia mortality in the Danish cities per 1,000 people.
Source: the Cause of Death Statistics (1901-1950) and the authors own calculations.

Figure A6: Scarlet fever mortality in the Danish cities, 1901-1950



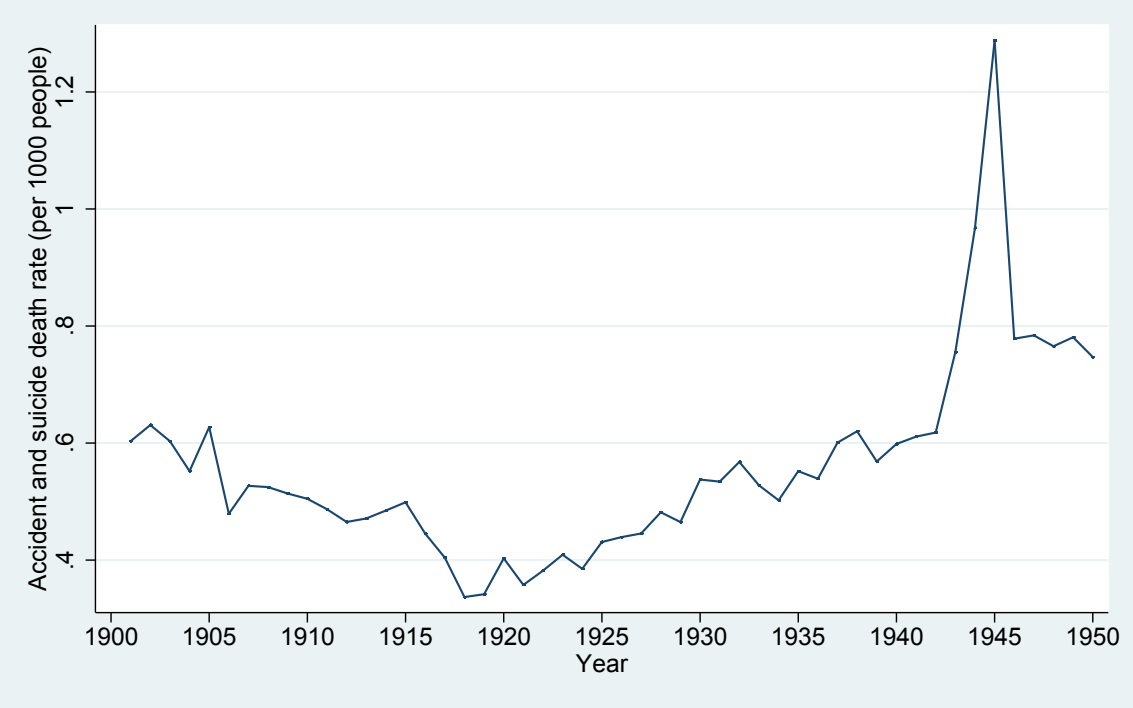
Notes: The graph show the national development of scarlet fever mortality in the Danish cities per 1,000 people.
Source: the Cause of Death Statistics (1901-1950) and the authors own calculations.

Figure A7: Cancer mortality in the Danish cities, 1901-1950



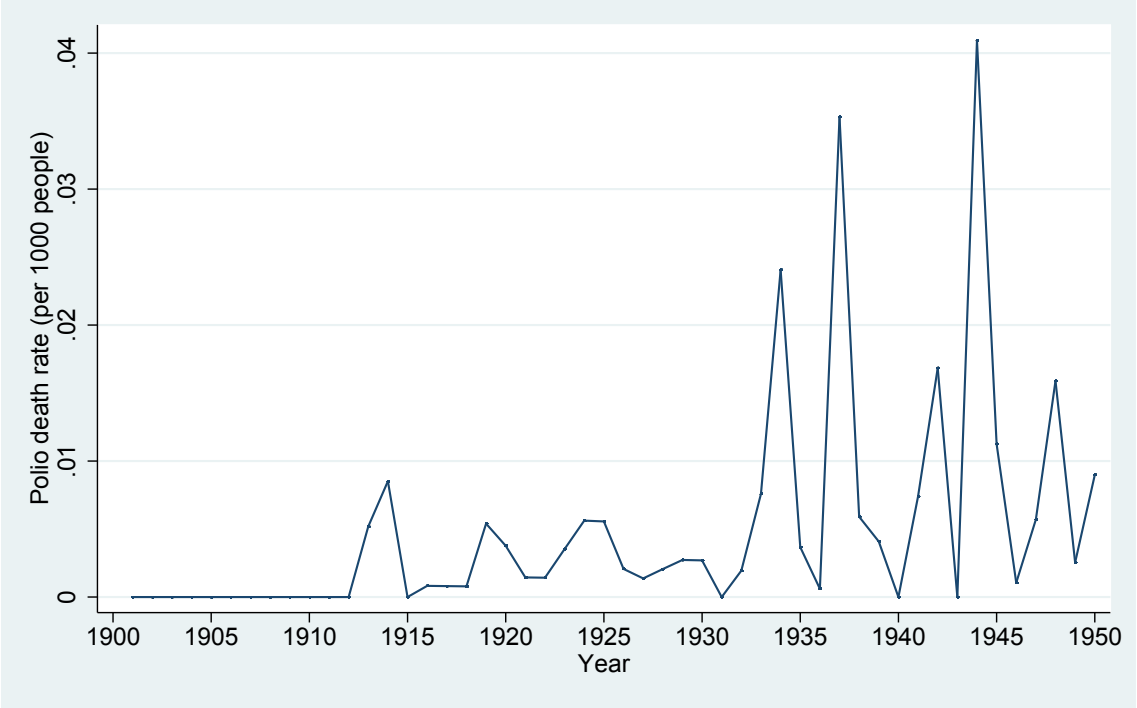
Notes: The graph show the national development of cancer mortality in the Danish cities per 1,000 people. Source: the Cause of Death Statistics (1901-1950) and the authors own calculations.

Figure A8: Accident and suicide mortality in the Danish cities, 1901-1950



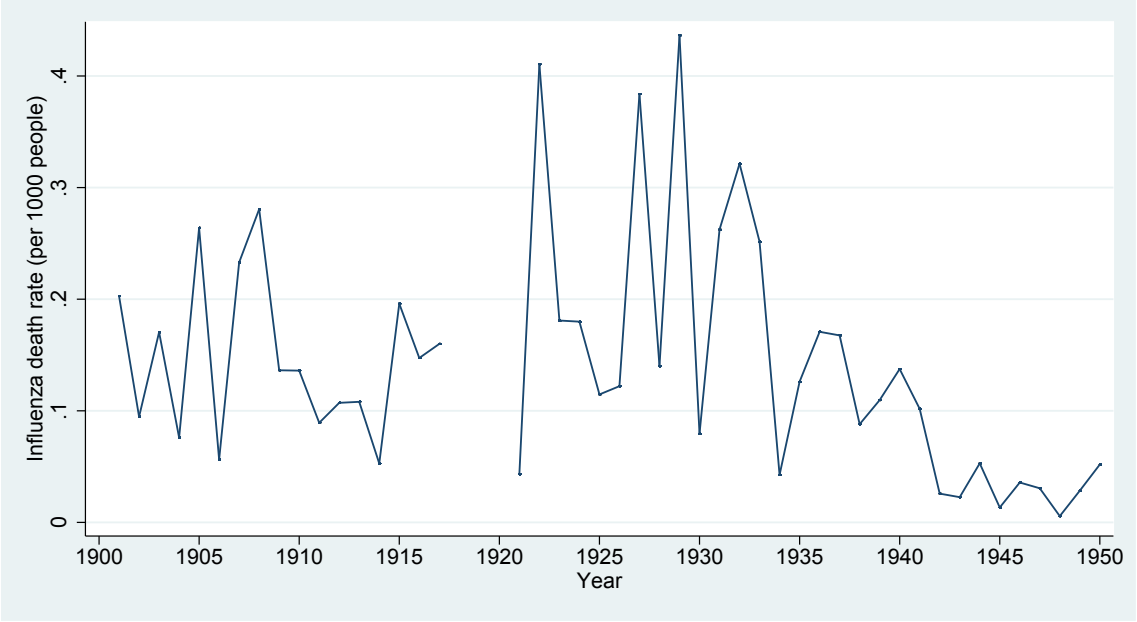
Notes: The graph show the national development of deaths from accidents and suicides in the Danish cities per 1,000 people. From 1931 the data also includes the number of deaths from homocides. Source: the Cause of Death Statistics (1901-1950) and the authors own calculations.

Figure A9: Polio mortality in the Danish cities, 1901-1950



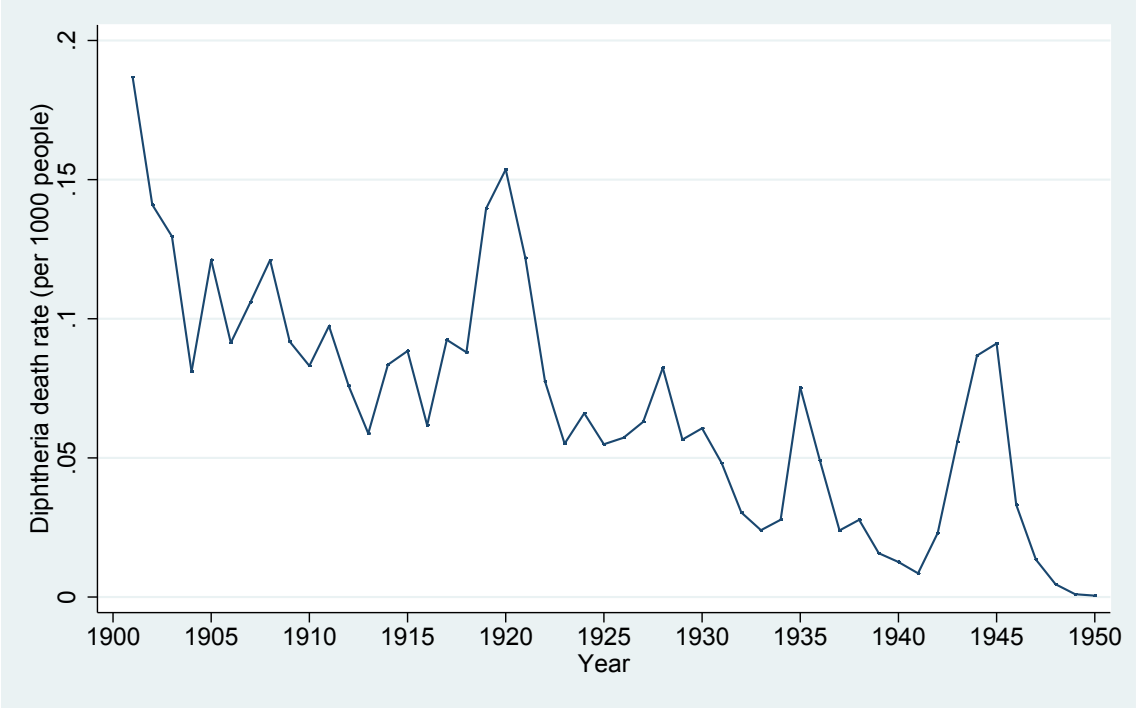
Notes: The graph show the national development of polio mortality in the Danish cities per 1,000 people. Source: the Cause of Death Statistics (1901-1950) and the authors own calculations.

Figure A10: Influenza mortality in the Danish cities, 1901-1950



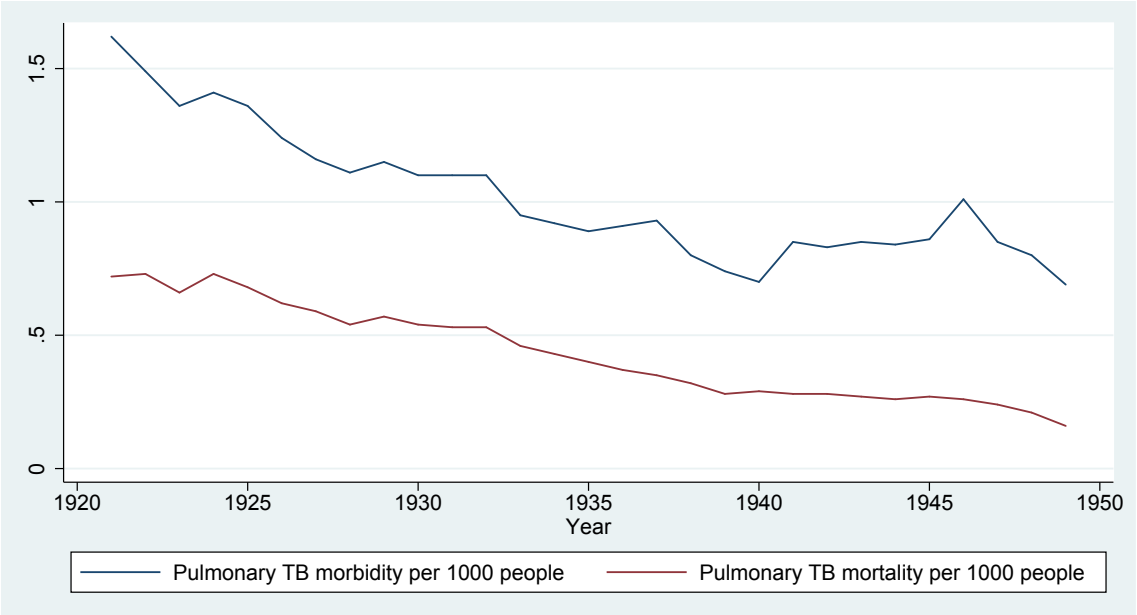
Notes: The graph show the national development of influenza mortality in the Danish cities per 1,000 people. The years 1918, 1919, and 1920 are not shown on graph because of high values due to the Spanish Flue. The influenza death rate were in the years 1918 to 1920, 3.27, 1.37, and 1.31 respectively. Source: the Cause of Death Statistics (1901-1950) and the authors own calculations.

Figure A11: Diphtheria mortality in the Danish cities, 1901-1950



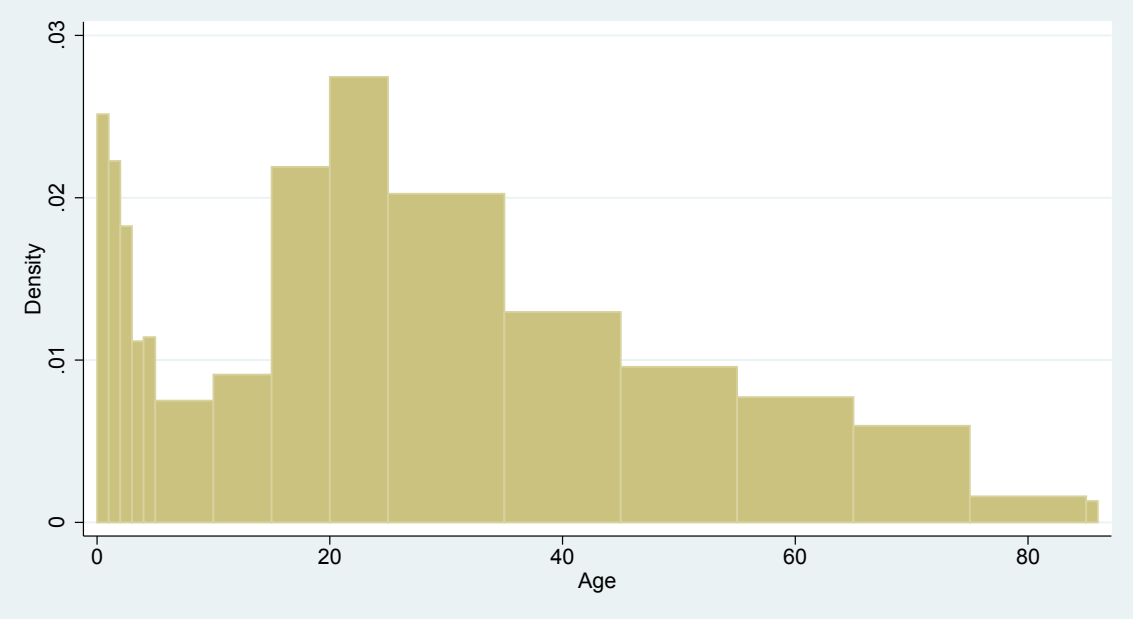
Notes: The graph show the national development of diphtheria mortality in the Danish cities per 1,000 people. Source: the Cause of Death Statistics (1901-1950) and the authors own calculations.

Figure A12: Pulmonary TB morbidity and mortality in Denmark, 1921-1949



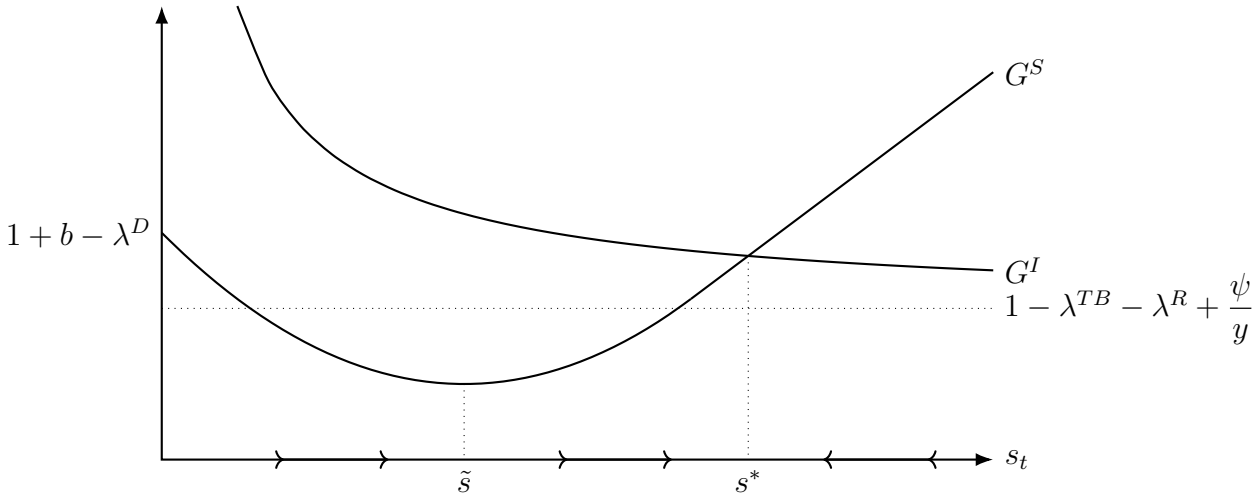
Notes: The graph show the national development of pulmonary TB morbidity as well as mortality in Denmark per 1,000 people. Source: Schelde Møller (1950, p.145).

Figure A13: Age distribution of TB mortality, 1921-25



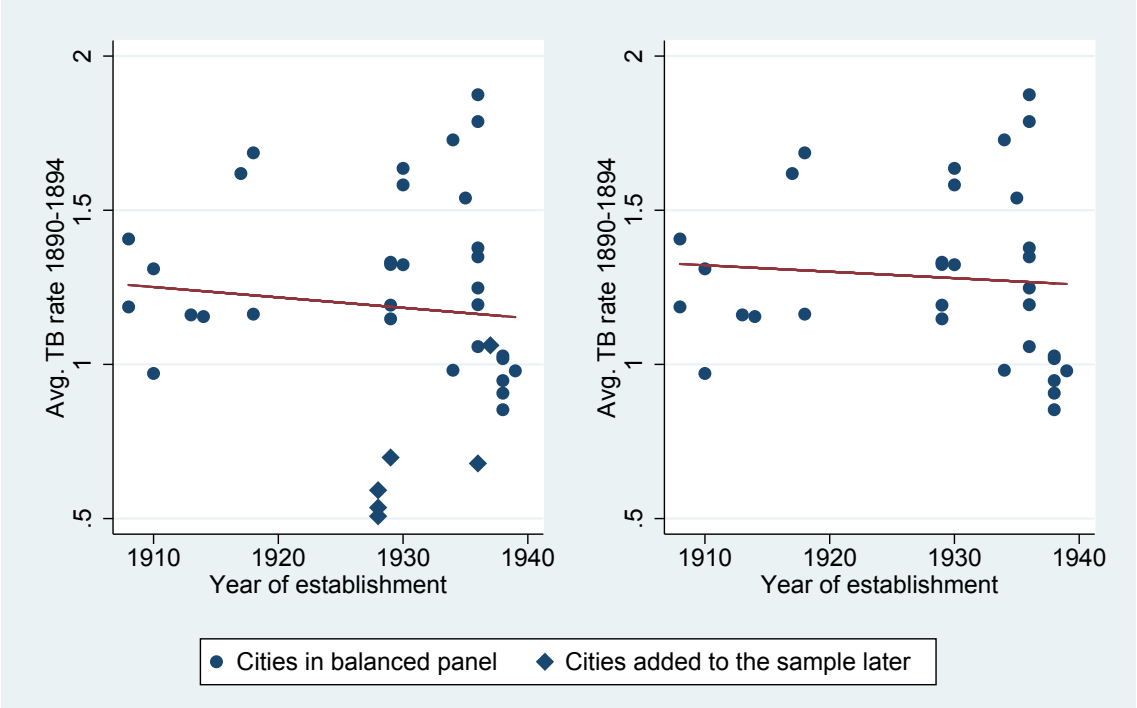
Notes: The graph shows the average age distribution of TB mortality for the five year period 1921-25.

Figure A14: $G, s-$diagram



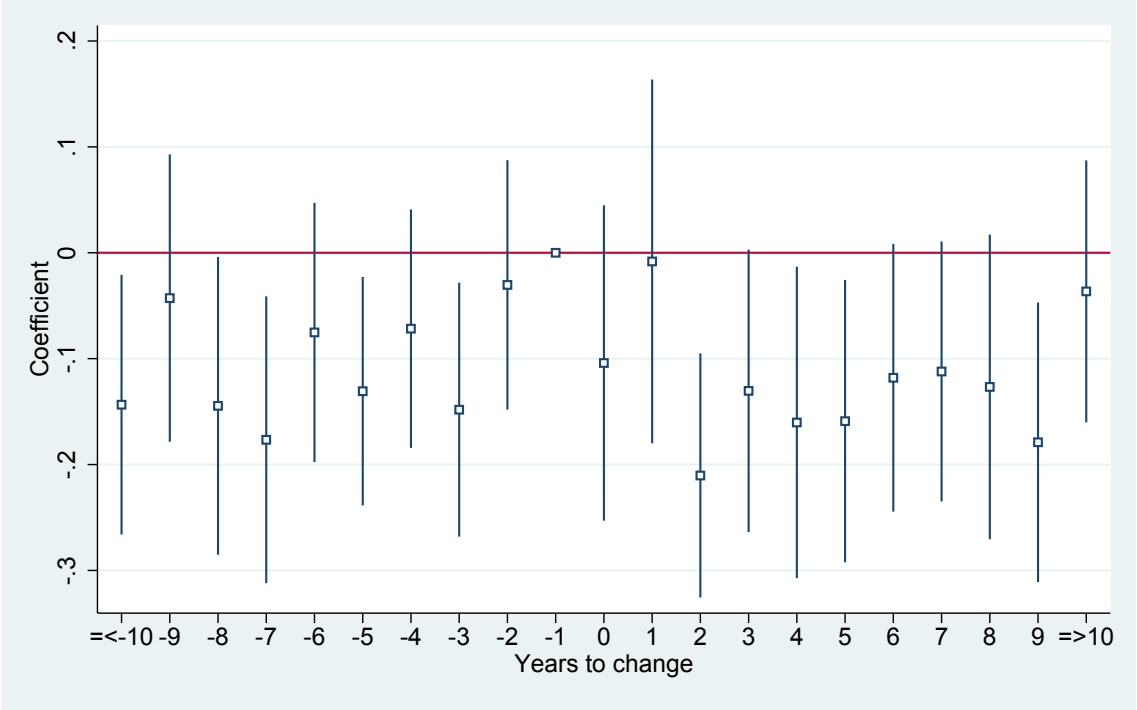
Notes: Depiction of equation (2) and (3).

Figure A15:



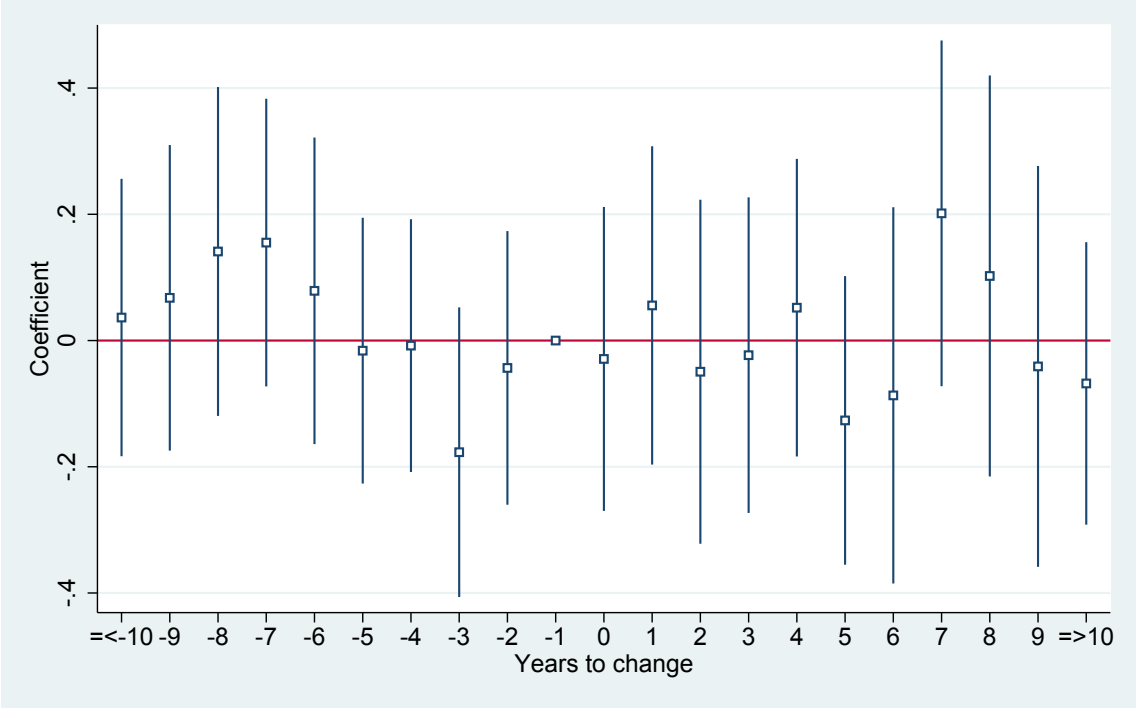
Notes: Relationship between the initial TB death rate and the year of establishment of a TB dispensary.

Figure A16: Event study estimates of impact on accidents and suicides before and after TB dispensary



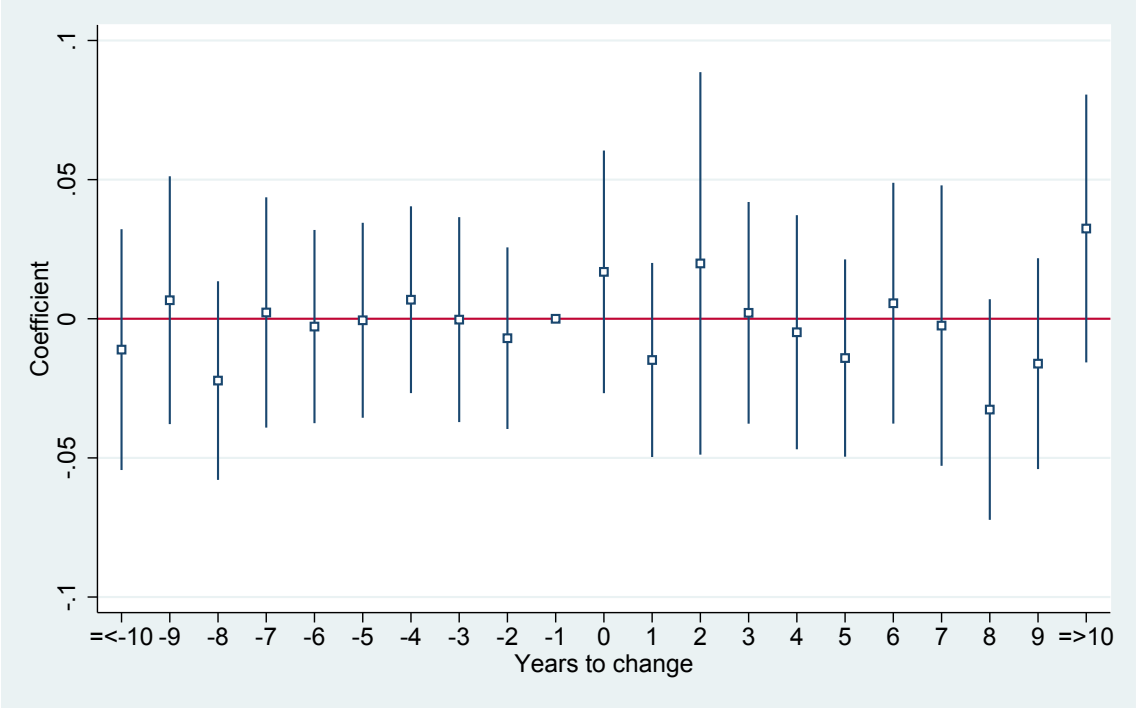
Notes: The graph shows the $\hat{\beta}_j$ coefficients and their 95% confidence interval from estimating equation (3) and (4) with the accidents and suicides (including homicides from 1931) mortality per 1,000 people as the dependent variable and year $\tau - 1$ as baseline, the year before the intervention. A table with the estimated coefficients are available upon request.

Figure A17: Event study estimates of impact on cancer before and after TB dispensary



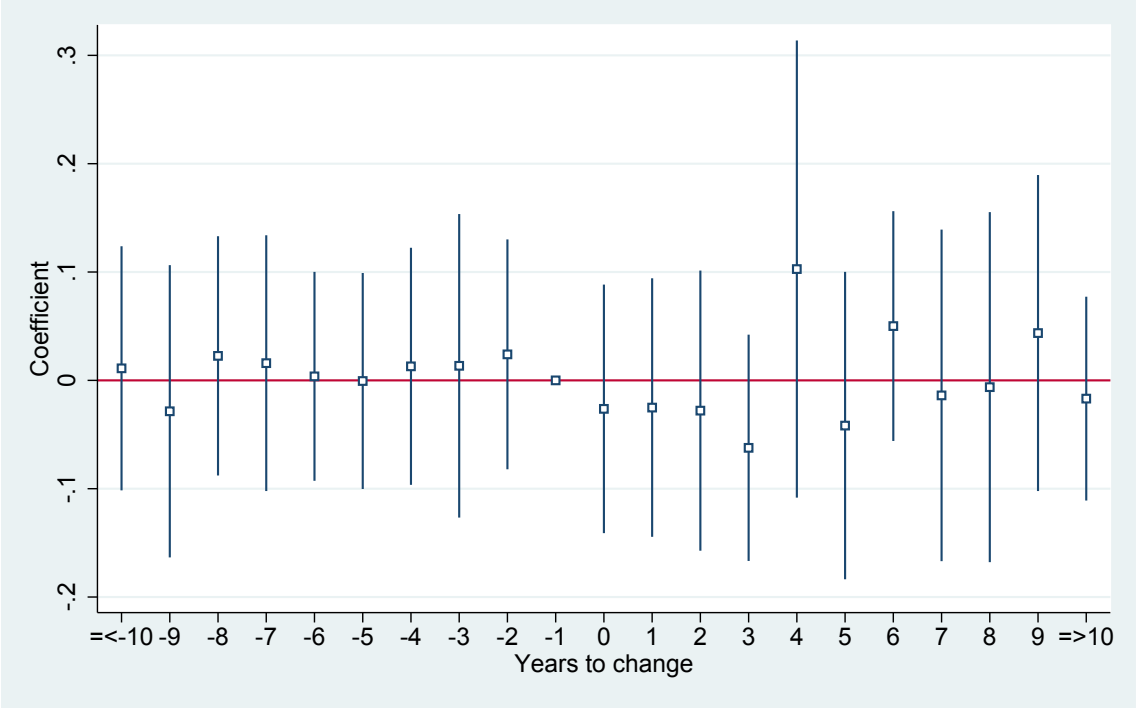
Notes: The graph shows the $\hat{\beta}_j$ coefficients and their 95% confidence interval from estimating equation (3) and (4) with the cancer mortality per 1,000 people as the dependent variable and year $\tau - 1$ as baseline, the year before the intervention. A table with the estimated coefficients are available upon request.

Figure A18: Event study estimates of impact on diphtheria before and after TB dispensary



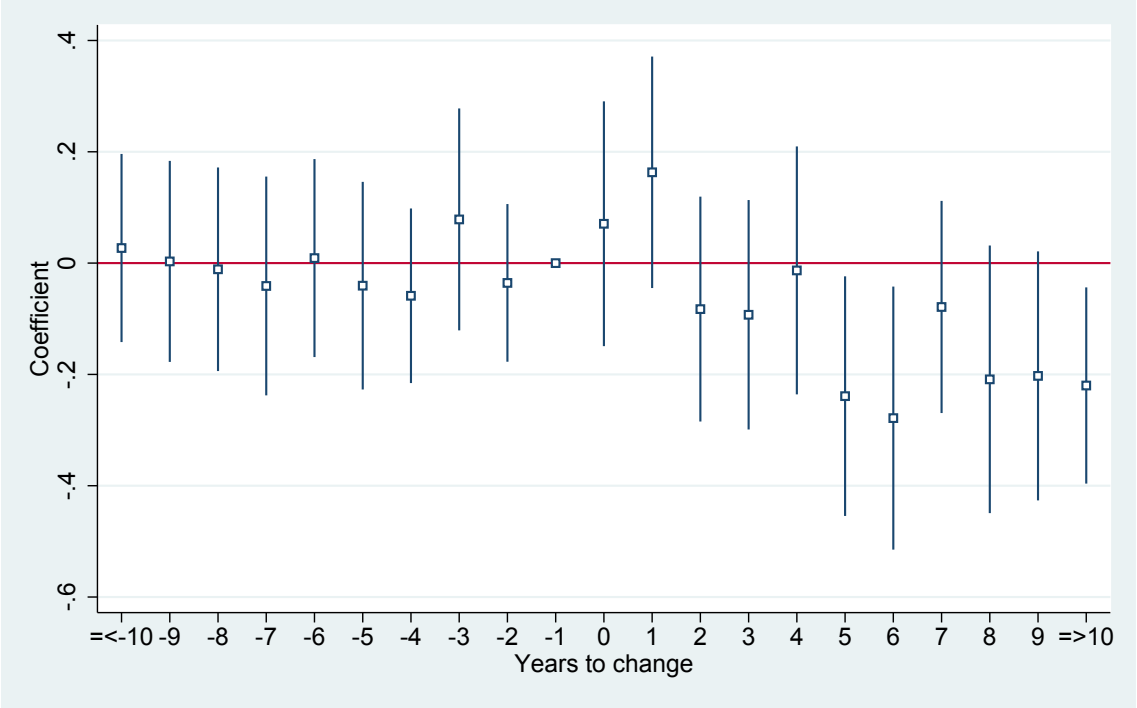
Notes: The graph shows the $\hat{\beta}_j$ coefficients and their 95% confidence interval from estimating equation (3) and (4) with the diphtheria mortality per 1,000 people as the dependent variable and year $\tau - 1$ as baseline, the year before the intervention. A table with the estimated coefficients are available upon request.

Figure A19: Event study estimates of impact on influenza before and after TB dispensary



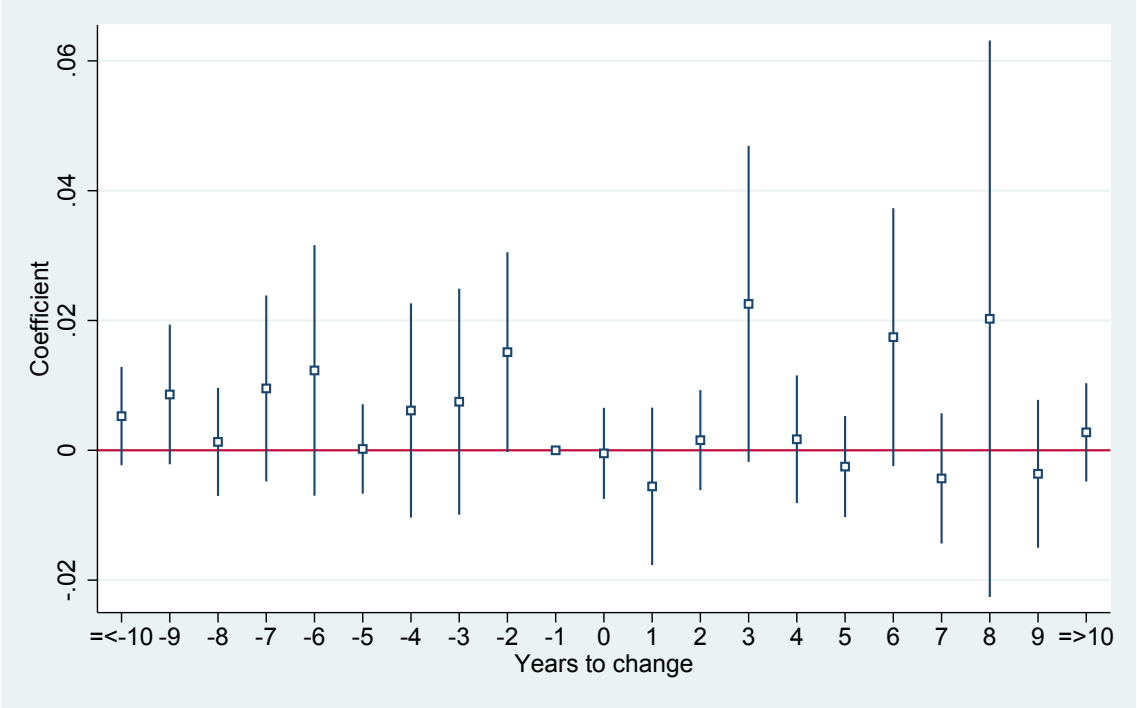
Notes: The graph shows the $\hat{\beta}_j$ coefficients and their 95% confidence interval from estimating equation (3) and (4) with the influenza mortality per 1,000 people as the dependent variable and year $\tau - 1$ as baseline, the year before the intervention. A table with the estimated coefficients are available upon request.

Figure A20: Event study estimates of impact on pneumonia before and after TB dispensary



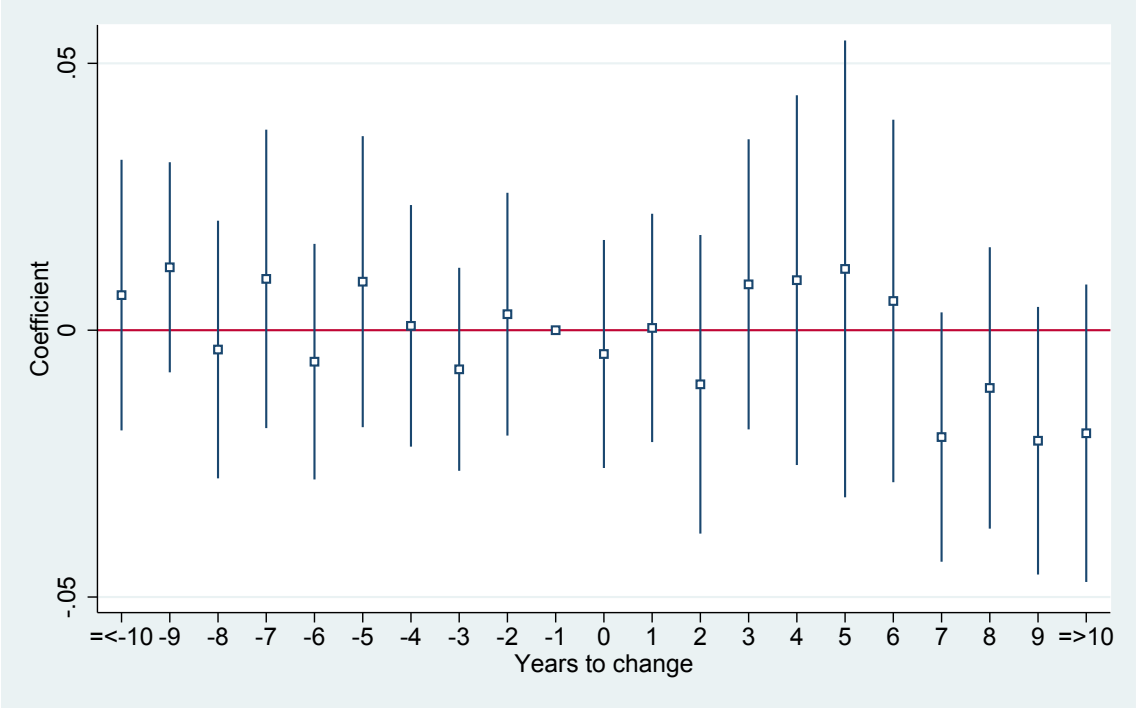
Notes: The graph shows the $\hat{\beta}_j$ coefficients and their 95% confidence interval from estimating equation (3) and (4) with the pneumonia mortality per 1,000 people as the dependent variable and year $\tau - 1$ as baseline, the year before the intervention. A table with the estimated coefficients are available upon request.

Figure A21: Event study estimates of impact on polio before and after TB dispensary



Notes: The graph shows the $\hat{\beta}_j$ coefficients and their 95% confidence interval from estimating equation (3) and (4) with the polio mortality per 1,000 people as the dependent variable and year $\tau - 1$ as baseline, the year before the intervention. A table with the estimated coefficients are available upon request.

Figure A22: Event study estimates of impact on scarlet fever before and after TB dispensary



Notes: The graph shows the $\hat{\beta}_j$ coefficients and their 95% confidence interval from estimating equation (3) and (4) with the scarlet fever mortality per 1,000 people as the dependent variable and year $\tau - 1$ as baseline, the year before the intervention. A table with the estimated coefficients are available upon request.

Table A1: Data explanations

Variable:	Explanation and source:
TB rate:	Number of death from any form of tuberculosis per 1,000 people. Source: Cause of Death Statistics (1890-1950).
Pulmonary rate:	Number of death from pulmonary tuberculosis per 1,000 people. Source: Cause of Death Statistics (1890-1950).
Birth rate:	Number of live births per 1,000 people (reference).
Death rate:	Number of death excluding still births per 1,000 people, as still births are only available from 1901. Source: Cause of Death Statistics (1890-1950).
Child mortality rate:	Number of deaths among 0 to 5 year old per 1,000 live births from 1901 to 1936 and number of deaths among 0 to 4 year old per 1,000 live births from 1937 to 1950. Source: Cause of Death Statistics (1901-1950).
Cancer rate:	Number of death from any form of cancer per 1,000 people. Source: Cause of Death Statistics (1890-1950).
Influenza rate:	Number of death from influenza per 1,000 people. Source: Cause of Death Statistics (1890-1950).
Pneumonia rate:	Number of death from any form of pneumonia per 1,000 people. Source: Cause of Death Statistics (1890-1950).
Accident and suicide rate:	Number of death from accidents and suicides, including homicides from 1931, per 1,000 people. Source: Cause of Death Statistics (1890-1950).
Scarlet fever rate:	Number of death from scarlet fever per 1,000 people. Source: Cause of Death Statistics (1890-1950).
Diphtheria rate:	Number of deaths from diphtheria per 1,000 people. Source: Cause of Death Statistics (1890-1950).
Polio rate:	Number of death from polio per 1,000 people. Source: Cause of Death Statistics (1890-1950).
Population:	Number of inhabitants. Source: Cause of Death Statistics (1890-1950).
TB dispensary:	The presence of either a main tuberculosis dispensary or a branch dispensary. Source: Medical Report (1890-1950).
Main dispensary:	The presence of a main tuberculosis dispensary. Source: Medical Report (1890-1950).
Branch dispensary:	The presence of a branch tuberculosis dispensary. Source: Medical Report (1890-1950).
TB hospital:	The presence of TB hospital. Source: Medical Report (1890-1950).
Sanatorium:	The presence of a sanatorium. Source: Medical Report (1890-1950).
Income:	Total income of the inhabitants liable to pay taxes per capita. Source: Statistiske meddelelser (1905-1940).
Gini:	The Gini coefficient for the inhabitants liable to pay taxes calculated using the lowest point in the intervals of the income distribution. Source: Statistiske meddelelser (1919, 1922).
Waterworks:	The presence of waterworks. Source: Medical Report (1890-1950).

Notes: This table describes the main variables used in the analysis.

Table A2: Number of TB dispensaries and TB mortality over time.

			TB rate			
Period	No. Cities	No. TB dispensaries	Mean	Std. dev.	Min	Max
1890–94	74	0	2.613	1.224	0.000	7.123
1895–99	75	0	2.139	1.063	0.000	5.556
1900–04	75	0	1.948	0.976	0.000	6.667
1905–09	75	2	1.546	0.894	0.000	5.549
1910–14	76	6	1.275	0.721	0.000	4.502
1915–19	77	9	1.120	0.765	0.000	5.490
1920–24	87	9	0.939	0.581	0.000	3.243
1925–29	87	17	0.733	0.507	0.000	3.077
1930–34	87	22	0.625	0.511	0.000	5.484
1935–39	87	38	0.453	0.410	0.000	3.636
1940–46	87	70	0.330	0.311	0.000	2.000
1890–1939	87	38	1.305	1.036	0.000	7.123
1890–1946	87	70	1.176	1.026	0.000	7.123

Notes: This table show the number of TB dispensaries over time along with the five-year average TB mortality rate. Source: the Cause of Death Statistics (1890-1950) and the Medical Reports (1890-1950).

Table A3: Balance tests

	A. Comparing adopters versus non-adopters of TB disp.					
		Adopters		Non-adopters		Mean-com-
	Period	Mean	Std. Dev.	Mean	Std. Dev.	parison test
No. observations		38		49		
Population	1890-94	17090.82	52605.14	3522.837	3318.552	[0.1209]
Population density	1890-94	1475.377	2673.166	986.4744	1601.942	[0.3229]
TB rate	1890-94	2.5454	0.8850	2.2928	0.9068	[0.1950]
Death rate	1890-94	18.1684	3.5012	18.3322	3.5023	[0.8293]
Child mortality rate	1901-05	152.5576	35.4638	143.4616	33.5975	[0.2283]
Income per capita	1904-08	363.0695	294.7243	335.8881	208.5662	[0.6311]
Taxpayers per capita	1904-08	0.1517	0.0789	0.1507	0.0807	[0.9566]
	B. Comparing pre-1931 adopters versus later adopters of TB disp.					
		Pre-1931 adopters		Post-1931 adopters		Mean-com-
	Period	Mean	Std. Dev.	Mean	Std. Dev.	parison test
No. observations		17		21		
Population	1890-94	31850.25	77176.83	5142.714	4408.774	[0.1733]
Population density	1890-94	2222.403	3837.729	870.6421	760.8533	[0.1708]
TB rate	1890-94	2.4524	1.0596	2.6207	0.7331	[0.5827]
Death rate	1890-94	17.5426	3.8763	18.6750	3.1711	[0.3392]
Child mortality rate	1901-05	149.4898	37.9308	155.0411	34.0769	[0.6418]
Income per capita	1904-08	485.6045	395.5262	263.8745	111.5728	[0.0377]
Taxpayers per capita	1904-08	0.1849	0.1060	0.1248	0.0286	[0.0357]

Notes: This table reports balance tests between adopters and non-adopters of TB dispensaries in the period 1890 to 1939 in part A of the table, and balance tests between adobters from 1890 to 1930 and adobters from 1931 and 1939 in part B of the table. The variables compared between are the mean of the 1890-1894 population, the population density as the mean of the 1890-1894 population divided by the 1890 acreage of the city in square kilometers, the mean of the 1890-1894 TB death rate, the mean of the 1890-94 death rate, the mean of the 1901-1905 child moratlity rate, the mean of the 1904-1908 income per capita, and the mean of the 1904-1908 taxpayers per capita. In brackets are shown p-values from a mean-comparision Welch's t-test.

Table A4: Effect of TB dispensaries on the TB death ratios and TB in level

Dep. variable:	TB ratio	log(TB ratio)	TB ratio	TB ratio	Pulmonary ratio	TB level	Disease level
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
TB dispensary$_{c,t}$	−9.3469**	−0.1159**	−9.0258**	−8.3966**	−8.7835**	−16.7565*	−11.3041***
	(4.5847)	(0.0498)	(4.1631)	(3.6222)	(3.7312)	(8.7024)	(4.2811)
TB ratio$_{c,t-1}$			0.0819***	0.0678***			
			(0.0251)	(0.0227)			
TB ratio$_{c,t-2}$				0.0429**			
				(0.0193)			
TB ratio$_{c,t-3}$				0.0697***			
				(0.0203)			
TB ratio$_{c,t-4}$				0.0484**			
				(0.0192)			
TB ratio$_{c,t-5}$				0.0335*			
				(0.0195)			
Avg. TB	89.2388	4.3983	88.1641	84.2148	59.3497	19.9618	18.0528
Long run effect			−9.8312**	−11.3839**			
City FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Year FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Disease FE	No	No	No	No	No	No	Yes
City FE × Year FE	No	No	No	No	No	No	Yes
Disease FE × Year FE	No	No	No	No	No	No	Yes
Disease FE × City FE	No	No	No	No	No	No	Yes
Time period	1890-1939	1890-1939	1891-1939	1895-1939	1901-1939	1890-1939	1901-1939
Observations	3,981	3,662	3,894	3,546	3,165	3,981	25,320
R-squared	0.4482	0.4852	0.4503	0.4522	0.3491	0.9388	0.9243
Cities	87	87	87	87	87	87	87

Notes: The table reports least squares estimates. In column (1), (3), and (4) the left-hand-side variable is the TB mortality per 1,000 death, in column (2) the log is taken of this variable, in column (5) the left-hand-side variable is the pulmonary TB mortality per 1,000 death, in column (6) the left-hand-side variable is TB deaths, and in column (7) the left-hand-side variable is the stacked causes of death from TB, cancer, influenza, pneumonia, accidents and suicides (including homicides from 1931), scarlet fever, diphtheria, and polio in levels. TB dispensary$_{c,t}$ is an indicator variable equal to one after the introduction of a TB dispensary multiplied by an indicator equal to one if the disease on the left-hand-side is TB, and TB rate$_{c,t-i}$ is the i year lagged dependent variable. Long run effect is the steady-state value of the estimated model in column (3), and (4). All regressions includes city and year fixed effects, and addtionally column (7) includes disease, disease-by-year, disease-by-city, and city-by-year fixed effects. Robust standard errors clustered at the city level are in parentheses.

*, **, and ***, determine significance levels of 10%, 5% og 1% respectively.

Table A5: Event studies of the impact of a TB dispensary

Dep. variable:	TB rate	Disease
	(1)	(2)
$\text{Dispensary}_{c,t-10}^{\tau-10}$	0.0538	0.0686
	(0.1070)	(0.1045)
$\text{Dispensary}_{c,t-9}^{\tau-9}$	−0.0121	−0.0107
	(0.0908)	(0.1006)
$\text{Dispensary}_{c,t-8}^{\tau-8}$	−0.0055	0.0023
	(0.0841)	(0.0922)
$\text{Dispensary}_{c,t-7}^{\tau-7}$	0.0631	0.0721
	(0.0865)	(0.0918)
$\text{Dispensary}_{c,t-6}^{\tau-6}$	−0.0110	−0.0086
	(0.0894)	(0.0934)
$\text{Dispensary}_{c,t-5}^{\tau-5}$	−0.1497*	−0.1192
	(0.0900)	(0.0992)
$\text{Dispensary}_{c,t-4}^{\tau-4}$	−0.0231	0.0026
	(0.0851)	(0.0888)
$\text{Dispensary}_{c,t-3}^{\tau-3}$	0.0074	0.0449
	(0.0872)	(0.0844)
$\text{Dispensary}_{c,t-2}^{\tau-2}$	−0.0359	−0.0213
	(0.0903)	(0.0899)
$\text{Dispensary}_{c,t}^{\tau}$	−0.1418	−0.1301
	(0.1045)	(0.1171)
$\text{Dispensary}_{c,t+1}^{\tau+1}$	−0.1372*	−0.1624**
	(0.0768)	(0.0801)
$\text{Dispensary}_{c,t+2}^{\tau+2}$	−0.2094*	−0.1531
	(0.1081)	(0.1137)
$\text{Dispensary}_{c,t+3}^{\tau+3}$	−0.1996**	−0.1553
	(0.0957)	(0.0978)
$\text{Dispensary}_{c,t+4}^{\tau+4}$	−0.2516*	−0.2531*
	(0.1411)	(0.1307)
$\text{Dispensary}_{c,t+5}^{\tau+5}$	−0.2745**	−0.1984*
	(0.1116)	(0.1121)
$\text{Dispensary}_{c,t+6}^{\tau+6}$	−0.2656***	−0.2124**
	(0.1007)	(0.1048)
$\text{Dispensary}_{c,t+7}^{\tau+7}$	−0.2582*	−0.2587*
	(0.1358)	(0.1356)
$\text{Dispensary}_{c,t+8}^{\tau+8}$	−0.2529**	−0.2202*
	(0.1196)	(0.1232)
$\text{Dispensary}_{c,t+9}^{\tau+9}$	−0.3186***	−0.2638**
	(0.1116)	(0.1138)
$\text{Dispensary}_{c,t+10}^{\tau+10}$	−0.2458**	−0.2115*
	(0.1122)	(0.1099)
City FE	Yes	Yes
Year FE	Yes	Yes
Disease FE	No	Yes
Disease FE × Year FE	No	Yes
Disease FE × City FE	No	Yes
City FE × Year FE	No	Yes
Time period	1890-1939	1901-1939
Observations	3,981	25,320
R-squared	0.5341	0.6970
Cities	87	87

Notes: The table reports least squares estimates. In column (1) the left-hand-side variable is the TB mortality per 1,000 people, and in column (2) the left-hand-side variable is the stacked causes of death from TB, cancer, influenza, pneumonia, accidents and suicides (including homicides from 1931), scarlet fever, diphtheria, and polio per 1,000 people. All regressions includes city and year fixed effects, and addtionally column (2) includes disease, disease-by-year, disease-by-city, and city-by-year fixed effects. TB $\text{dispensary}_{c,t+j}^{\tau+j}$ were $T = \{-10, \ldots, -2, 0, \ldots, 10\}$ is an indicator equal to one when $t = \tau + j$ where τ marks the period of introduction of a TB dispensary. All indicators are multiplied by an indicator equal to one if the disease on the left-hand-side is TB. Robust standard errors clustered at the city level are in parentheses.

*, **, and ***, determine significance levels of 10%, 5% og 1% respectively.

Table A6: Effect of TB dispensaries using city by year by disease data with alternative disease panels

Dep. variable:	Disease Non-treated diseases: Pneumonia, Scarlet fever	Disease Non-treated diseases: Cancer, Accidents and suicides
	(1)	(2)
TB dispensary$_{c,t}$	−0.1681**	−0.1855***
	(0.0679)	(0.0701)
Avg. TB rate	1.0335	1.0335
City FE	Yes	Yes
Year FE	Yes	Yes
Disease FE	Yes	Yes
Disease FE × Year FE	Yes	Yes
Disease FE × City FE	Yes	Yes
City FE × Year FE	Yes	Yes
Time period	1901-1939	1901-1939
Observations	9,495	9,495
R-squared	0.7413	0.6477
Cities	87	87

Notes: The table reports least squares estimates. In column (1) the left-hand-side variable is the stacked causes of death from TB, pneumonia, and scarlet fever per 1,000 people. In column (2) the left-hand-side variable is the stacked causes of death from TB, cancer, and accidents and suicides (including homicides from 1931) per 1,000 people. All regressions include city, year, disease, disease-by-year, disease-by-city, and city-by-year fixed effects. TB dispensary$_{c,t}$ is an indicator variable equal to one after the introduction of a TB dispensary multiplied by an indicator equal to one if the disease on the left-hand-side is TB. Robust standard errors clustered at the city level are in parentheses.
*, **, and ***, determine significance levels of 10%, 5% og 1% respectively.

Table A7: Effect of TB dispensaries; alternative treatment measures

Dep. variable:	TB rate	Disease	TB rate	Disease
	(1)	(2)	(3)	(4)
TB dispensary$_{c,t} \times (t + 1 - j_c)$	-0.0117^{**}	-0.0084^{*}		
	(0.0053)	(0.0046)		
TB dispensary$_{c,t} \times$ TB rate$_{c,j}$			-0.1729^{***}	-0.1606^{***}
			(0.0622)	(0.0528)
Avg. TB rate	1.3055	1.0335	1.3055	1.0335
City FE	Yes	Yes	Yes	Yes
Year FE	Yes	Yes	Yes	Yes
Disease FE	No	Yes	No	Yes
City FE $\times$ Year FE	No	Yes	No	Yes
Disease FE $\times$ Year FE	No	Yes	No	Yes
Disease FE $\times$ City FE	No	Yes	No	Yes
Time period	1890-1939	1901-1939	1890-1935	1901-1935
Observations	3,981	25,320	3,981	25,320
R-squared	0.5323	0.6967	0.5332	0.6969
Cities	87	87	87	87

Notes: The table reports least squares estimates. In column (1), and (3) the left-hand-side variable is the TB mortality per 1,000 people, and in column (2), and (4) the left-hand-side variable is the stacked causes of death from TB, cancer, influenza, pneumonia, accidents and suicides (including homicides from 1931), scarlet fever, diphtheria, and polio per 1,000 people. All regressions include city and year fixed effects, and addtionally column (2), and (4) includes disease, disease-by-year, disease-by-city, and city-by-year fixed effects. TB dispensary$_{c,t}$ is an indicator variable equal to one after the introduction of a TB dispensary multiplied by an indicator equal to one if the disease on the left-hand-side is TB, j_c is the year of the introduction of a dispensary, t is the year, and TB rate$_{c,j}$ is the average TB rate in five years before the introduction of a TB dispensary standardized to have a zero mean and standard deviation of one. Robust standard errors clustered at the city level are in parentheses. *, **, and ***, determine significance levels of 10%, 5% og 1% respectively.

Table A8: Effect of TB dispensaries controlling for early BCG trials

Dep. variable:	TB rate	Disease	TB rate	Disease
	Excluding Bornholm		Panel limited to 1935	
	(1)	(2)	(3)	(4)
TB dispensary$_{c,t}$	−0.2004**	−0.1758**	−0.2106**	−0.1866**
	(0.0838)	(0.0707)	(0.0921)	(0.0815)
Avg. TB rate	1.3066	1.3897	1.0302	1.1085
City FE	Yes	Yes	Yes	Yes
Year FE	Yes	Yes	Yes	Yes
Disease FE	No	Yes	No	Yes
City FE × Year FE	No	Yes	No	Yes
Disease FE × Year FE	No	Yes	No	Yes
Disease FE × City FE	No	Yes	No	Yes
Time period	1890-1939	1901-1939	1890-1935	1901-1935
Observations	3,681	23,448	3,633	22,536
R-squared	0.5541	0.7109	0.5104	0.6999
Cities	81	81	87	87

Notes: The table reports least squares estimates. In column (1), and (3) the left-hand-side variable is the TB mortality per 1,000 people, and in column (2), and (4) the left-hand-side variable is the stacked causes of death from TB, cancer, influenza, pneumonia, accidents and suicides (including homicides from 1931), scarlet fever, diphtheria, and polio per 1,000 people. All regressions include city and year fixed effects, and addtionally column (2), and (4) includes disease, disease-by-year, disease-by-city, and city-by-year fixed effects. TB dispensary$_{c,t}$ is an indicator variable equal to one after the introduction of a TB dispensary multiplied by an indicator equal to one if the disease on the left-hand-side is TB. In column (1) and (2) the six cities on the island of Bornholm are excluded from the sample, and in column (3), and (4) the years 1936 to 1939 are excluded. Robust standard errors clustered at the city level are in parentheses.
*, **, and ***, determine significance levels of 10%, 5% og 1% respectively.

Table A9: Effect of TB dispensaries excluding large cities and early adopters

Dep. variable:	TB rate	Disease	TB rate	Disease	TB rate	Disease
	Excluding Copenhagen		Excluding 5 largest cities		Excluding pre 1920 adopters	
	(1)	(2)	(3)	(4)	(5)	(6)
TB dispensary$_{c,t}$	-0.2304^{***}	-0.1994^{***}	-0.2669^{***}	-0.2269^{***}	-0.2588^{***}	-0.2316^{***}
	(0.0797)	(0.0661)	(0.0868)	(0.0681)	(0.0925)	(0.0788)
Avg. TB rate	1.3015	1.0300	1.2892	1.0196	1.3091	1.0339
City FE	Yes	Yes	Yes	Yes	Yes	Yes
Year FE	Yes	Yes	Yes	Yes	Yes	Yes
Disease FE	No	Yes	No	Yes	No	Yes
Disease FE × Year FE	No	Yes	No	Yes	No	Yes
Disease FE × City FE	No	Yes	No	Yes	No	Yes
City FE × Year FE	No	Yes	No	Yes	No	Yes
Time period	1890-1939	1901-1939	1890-1939	1901-1939	1890-1939	1901-1939
Observations	3,931	25,008	3,731	23,760	3,341	20,992
R-squared	0.5301	0.6943	0.5173	0.6850	0.5053	0.6909
Cities	86	86	82	82	68	68

Notes: The table reports least squares estimates. In column (1), (3), and (5) the left-hand-side variable is the TB mortality per 1,000 people, and in column (2), (4), and (6) the left-hand-side variable is the stacked causes of death from TB, cancer, influenza, pneumonia, accidents and suicides (including homicides from 1931), scarlet fever, diphtheria, and polio per 1,000 people. All regressions include city and year fixed effects, and addtionally column (2), (4), and (6) include disease, disease-by-year, disease-by-city, and city-by-year fixed effects. TB dispensary$_{c,t}$ is an indicator variable equal to one after the introduction of a TB dispensary multiplied by an indicator equal to one if the disease on the left-hand-side is TB. In column (1) and (2) the city Copenhagen is excluded from the sample, in column (3) and (4) the five largest cities as of 1890 and 1901 are excluded, and in column (5) and (6) cities which adopted a TB dispensary before 1920 are excluded. Robust standard errors clustered at the city level are in parentheses.
*, **, and ***, determine significance levels of 10%, 5% og 1% respectively.

Table A10: Effect of TB dispensaries controlling for lagged population, initial population, weighting by city sizes, and linear trend

Dep. variable:	TB rate	Disease	TB rate	Disease	TB rate	Disease	TB rate
	OLS				WLS		OLS
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
TB dispensary$_{c,t}$	−0.1825**	−0.1809***	−0.2337***	−0.2113***	−0.2074***	−0.1808***	−0.1240*
	(0.0760)	(0.0644)	(0.0807)	(0.0642)	(0.0759)	(0.0656)	(0.0731)
log(Population$_{c,t-1}$)	−0.6804***	−0.2389					
	(0.2304)	(0.2147)					
Avg. TB rate	1.2804	1.0125	1.3055	1.0335	1.3055	1.0335	1.3055
City FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Year FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Disease FE	No	Yes	No	Yes	No	Yes	No
City FE × Year FE	No	Yes	No	Yes	No	Yes	No
Disease FE × Year FE	No	Yes	No	Yes	No	Yes	No
Disease FE × City FE	No	Yes	No	Yes	No	Yes	No
Initial log(population) × Year FE	No	No	Yes	Yes	No	No	No
Linear trend × City FE	No	No	No	No	No	No	Yes
Time period	1891-1939	1902-1939	1890-1939	1901-1939	1890-1939	1901-1939	1890-1939
Observations	3,894	24,624	3,981	25,320	3,981	25,320	3,981
R-squared	0.5342	0.6972	0.5376	0.6976	0.5502	0.7125	0.5818
Cities	87	87	87	87	87	87	87

Notes: The table reports ordinary least squares estimates, except column (5) and (6) repporting least squares weighted on initial log population. In column (1), (3), (5), and (7) the left-hand-side variable is the TB mortality per 1,000 people, and in column (2), (4), and (6) the left-hand-side variable is the stacked causes of death from TB, cancer, influenza, pneumonia, accidents and suicides (including homicides from 1931), scarlet fever, diphtheria, and polio per 1,000 people. All regressions include city and year fixed effects, and addtionally column (2), (4), and (6) include disease, disease-by-year, disease-by-city, and city-by-year fixed effects. TB dispensary$_{c,t}$ is an indicator variable equal to one after the introduction of a TB dispensary, and log(Population$_{c,t-1}$) is the lagged log population, where both variables are multiplied by an indicator equal to one if the disease on the left-hand-side is TB. In column (3), and (4) the initial log population (the log population of the first year the city is included in the sample) interacted with year fixed effects and multiplied by an indicator equal to one if the disease on the left-hand-side is TB is included, and in column (7) a city specific linear time trend is included. Robust standard errors clustered at the city level are in parentheses.
*, **, and ***, determine significance levels of 10%, 5% og 1% respectively.

Table A11: Effect of TB dispensaries controlling for commissioning of waterworks

Dep. variable:	TB rate	Disease
	(1)	(2)
TB dispensary$_{c,t}$	−0.2331***	−0.1966***
	(0.0760)	(0.0641)
Waterworks$_{c,t}$	−0.0973	−0.0828
	(0.0902)	(0.0992)
Avg. TB rate	1.3055	1.0335
City FE	Yes	Yes
Year FE	Yes	Yes
Disease FE	No	Yes
City FE × Year FE	No	Yes
Disease FE × Year FE	No	Yes
Disease FE × City FE	No	Yes
Time period	1890-1939	1901-1939
Observations	3,981	25,320
R-squared	0.5339	0.6969
Cities	87	87

Notes: The table reports least squares estimates. In column (1) the left-hand-side variable is the TB mortality per 1,000 people, and in column (2) the left-hand-side variable is the stacked causes of death from TB, cancer, influenza, pneumonia, accidents and suicides (including homicides from 1931), scarlet fever, diphtheria, and polio per 1,000 people. All regressions include city and year fixed effects, and addtionally column (2) include disease, disease-by-year, disease-by-city, and city-by-year fixed effects. TB dispensary$_{c,t}$ is an indicator variable equal to one after the introduction of a TB dispensary, and Waterworks$_{c,t}$ are an indicator equal to one after the introduction of waterworks, where both are multiplied by an indicator equal to one if the disease on the left-hand-side is TB. Robust standard errors clustered at the city level are in parentheses.
*, **, and ***, determine significance levels of 10%, 5% og 1% respectively.

Table A12: Effect of main and branch TB dispensaries

Dep. variable:	TB rate	Disease
	(1)	(2)
Main dispensary$_{c,t}$	−0.2109**	−0.2007**
	(0.0921)	(0.0864)
Branch dispensary$_{c,t}$	−0.2316*	−0.1722**
	(0.1176)	(0.0836)
Avg. TB rate	1.3055	1.0335
Joint significance of main and branch dispensary	[0.0214]	[0.0188]
Test of equivalence between main and branch dispensary	[0.8821]	[0.7964]
City FE	Yes	Yes
Year FE	Yes	Yes
Disease FE	No	Yes
Disease FE × Year FE	No	Yes
Disease FE × City FE	No	Yes
City FE × Year FE	No	Yes
Time period	1890-1939	1901-1939
Observations	3,981	25,320
R-squared	0.5333	0.6969
Cities	87	87

Notes: The table reports least squares estimates. In column (1) the left-hand-side variable is the TB mortality per 1,000 people, and in column (2) the left-hand-side variable is the stacked causes of death from TB, cancer, influenza, pneumonia, accidents and suicides (including homicides from 1931), scarlet fever, diphtheria, and polio per 1,000 people. All regressions include city and year fixed effects, and addtionally column (2) include disease, disease-by-year, disease-by-city, and city-by-year fixed effects. Main dispensary$_{c,t}$ is an indicator variable equal to one after the introduction of a main TB dispensary, and Branch dispensary$_{c,t}$ is an indicator variable equal to one after the introduction of a branch TB dispensary, where both indicators are multiplied by an indicator equal to one if the disease on the left-hand-side is TB. Robust standard errors clustered at the city level are in parentheses, and p-values in brackets.
*, **, and ***, determine significance levels of 10%, 5% og 1% respectively.

Table A13: Effect of TB dispensaries in a balanced and extended panel

Dep. variable:	TB rate	Disease	TB rate	Disease
	Balanced panel		Panel extended to 1946	
	(1)	(2)	(3)	(4)
TB dispensary$_{c,t}$	-0.2435^{***}	-0.2132^{***}	-0.1894^{***}	-0.1433^{***}
	(0.0849)	(0.0728)	(0.0600)	(0.0492)
Avg. TB rate	1.3396	1.0516	1.1760	0.9199
City FE	Yes	Yes	Yes	Yes
Year FE	Yes	Yes	Yes	Yes
Disease FE	No	Yes	No	Yes
Disease FE × Year FE	No	Yes	No	Yes
Disease FE × City FE	No	Yes	No	Yes
City FE × Year FE	No	Yes	No	Yes
Time period	1890-1939	1901-1939	1890-1946	1901-1946
Observations	3,700	23,088	4,590	30,192
R-squared	0.5343	0.7079	0.5700	0.7008
Cities	74	74	87	87

Notes: The table reports least squares estimates. In column (1), and (3) the left-hand-side variable is the TB mortality per 1,000 people, and in column (2), and (4) the left-hand-side variable is the stacked causes of death from TB, cancer, influenza, pneumonia, accidents and suicides (including homicides from 1931), scarlet fever, diphtheria, and polio per 1,000 people. All regressions include city and year fixed effects, and addtionally column (2), and (4) includes disease, disease-by-year, disease-by-city, and city-by-year fixed effects. TB dispensary$_{c,t}$ is an indicator variable equal to one after the introduction of a TB dispensary multiplied by an indicator equal to one if the disease on the left-hand-side is TB. In column (1), and (2) the sample are the 74 cities that constitutes a balanced panel, and in column (3), and (4) the sample is extended to also include the years 1940 to 1946. Robust standard errors clustered at the city level are in parentheses.
*, **, and ***, determine significance levels of 10%, 5% og 1% respectively.