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Information and Disease Prevention: Tuberculosis Dispensaries*

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Abstract

Tuberculosis (TB) is a leading cause of death worldwide according to the WHO. This paper estimates the effect of TB dispensaries, designed to prevent the spread of the disease before the advent of modern medicine. Our difference-in-differences estimation reveals that the roll-out of the TB dispensaries across Danish cities led to a 16 percent decline in the TB mortality rate, but no significant impacts on other diseases when performing placebo regressions. We obtain very similar estimates from a triple-differences setup, warranting a causal interpretation of our findings. Overall, our conclusion suggests, contrary to McKeown (1976), that public policy played an important role for the decline in TB mortality. It also suggests that dispensaries are of policy relevance for developing countries today as a measure to counter the externalities created by TB and modern drug resistant strains.

Keywords: Tuberculosis mortality, public health, information, disease prevention, infection externality.

JEL codes: D62, H23, I15, I18, N34.

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1 Introduction

Differences in mortality across space and time are "phenomena worthy of serious attention by economists" (Cutler, Deaton and Lleras-Muney, 2006, p.7). The present research is motivated by this observation and focuses on the decline in mortality from infectious diseases, which arguably are of interest to economists since they are "quintessential manifestations of the principle of an externality, itself a central concept in economics" (Gersovitz and Hammer, 2004, p.1). In particular, we study a historical, local public health institution known as a dispensary, targeted at preventing the spread of tuberculosis (TB) and provide empirical tests on whether the spread of dispensaries reduced TB mortality.

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 cases, and even if TB 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.[1] Presently, TB cases are mainly found in developing countries, though this was not always the case. Before the advent of modern medicine, European countries also suffered with high rates of TB cases and deaths. Moreover, there was considerable variation within Europe with some countries being able to reduce TB in the pre-modern medicine 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".[2] Using data for England and Wales, McKeown (1976) noted, along similar lines, that 80 percent of the reduction in TB mortality happened before there was any effective treatment. He argued that the decline was unrelated to any public health measure. This view has more recently been supported by Fogel (1994, 1997), while other scholars who are more sceptical, highlight the role of public health measures, which were set in motion by the germ theory of disease in the 1880s (e.g., Preston,

[1] The overall goal is to "ensure healthy lives and promote well-being for all at all ages", see `http://www.un.org/sustainabledevelopment/health/`.

[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).

1996; Cutler, Deaton and Lleras-Muney, 2006).[3]

To our knowledge, this paper is the first to evaluate quantitatively the effect of introducing TB dispensaries on TB mortality. Our quasi-experiment unfolds in Denmark, which Daniels (1949) singles out as particularly successful in combating TB. This is backed up by Holm (1946a, p.1426), who notes that Denmark had the lowest TB mortality prior to World War II—34 per 100,000 population. Schelde Møller (1950) ascribed this to the policies pursued in Denmark, which were instigated by the *National Foundation for the Fight Against Tuberculosis*. Among other public health measures, the National Foundation established TB dispensaries locally which were rolled-out across time and space differentially.

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 (Schelde Møller, 1950, p.74). The dispensaries themselves were little more than a room used for linen, towels, disinfectants, and spittoons (Schelde Møller, 1950, p.76). All doctors should be able to refer patients to the dispensary, which was either led by a specialized doctor or nurse.[4] 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." As can be inferred from the description of the dispensaries, they were targeted at alleviating the externalities associated with TB infection. Building on Gersovitz and Hammer (2004, p.3),[5] we note that an infection externality arises, if when choosing

[3] A related study on the impact of public health interventions is Cutler and Miller (2005), who study the impact of water filtration and chlorination on mortality from waterborne diseases as well as overall mortality; see also Alsan and Goldin (2015).

[4] 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.

[5] They also provide a theoretical model for studying policy choices regarding treatment and prevention for how to address the externalities created by infectious diseases. For further discussion of the public economics of TB control, see Jacks (2001).

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 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.

Our analysis estimates the impact of the TB dispensaries on TB mortality for Danish cities covering the period 1890-1939. Our first strategy employs a difference-in-differences strategy in which the impact of TB dispensaries is estimated using the roll-out over time across cities. The effect on TB mortality is negative and statistically significant, while performing a number of placebo tests on other diseases (i.e., causes of deaths) reveal no impact on scarlet fever, pneumonia, accidents and suicides, diphtheria, cancer, polio, and influenza. Yet, the results indicate a negative impact on the overall mortality rate in the reduced form, which is corroborated by the fact that TB mortality and overall mortality are strongly and positively correlated.[6]

Our second empirical strategy follows a triple-differences setup which exploits that the dispensaries were targeted at TB and include in our panel the other diseases as controls.[7] This experiment compares the development of TB mortality to the control diseases before and after the introduction of TB dispensary in a given city, making it possible to relax the difference-in-differences assumption of conditionally random assignment of the roll-out. Reassuringly both strategies result in very similar estimates, which suggest that the TB dispensaries reduced the TB mortality rate with about 16 percent.

For both strategies we perform a number of robustness tests. For example, we start by checking for pretrends by estimating "flexible" models which compute the average effect of having TB dispensaries in the 10 years before and after implementation. These estimates show that the impact of TB dispensaries are systematically negative only after their introduction and no effects in the years before. This evidence rules out that the baseline estimates are contaminated by secular declines in TB mortality and reverse causality (i.e., adoption of TB dispensaries due to high TB mortality). We also show that the results are not driven by, e.g.,

[6]We also find that the TB dispensaries had a negative and significant impact on infant mortality.

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

pre-intervention population size, pre-intervention income, or pre-intervention inequality.

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 noted by Williamson (1910), TB dispensaries spread around Europe in the early 20th century,[8] and so in this way, dispensaries were not unique to Denmark. However, Denmark had a high number of TB institutions in per capita terms (Lawrence, 2006, p. 340). The Danish case also provides the advantage that data are available on a large number of diseases and the timing of TB 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 emphasize that the TB dispensaries may have mattered, 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 TB dispensaries have policy relevance today in a developing-country context, and suggest a way of combating an externality producing infectious disease, especially because drug resistant TB now exists. Finally, recent research suggests that eradicating TB is important for human capital accumulation and other economic outcomes; see, e.g., Hansen (2013), and Bütikofer and Salvanes (2015).[9] In fact, 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 213,540,000 dollars in 1910 (about 0.6 percent of GNP).[10]

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 explains the empirical strategy. Section 4 describes the data. Section 5 provides results. Section 6 contains

[8]The first dispensary was founded in Edinburgh, Scotland in 1887; France got its first dispensary in 1901; Germany in 1904, see Williamson (1910).

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

[10]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).

robustness analyses. Finally, Section 7 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 in TB mortality in Danish cities over time. Second, we describe the intervention of interest, the roll-out of TB dispensaries. Third, we describe other 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 (Hemskerk et al., 2015, chapter 1; Schelde Møller, 1950, p.11). The most common type of TB occurs in the lungs (pulmonary tuberculosis), 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 (Hemskerk et al., 2015, chapter 2). 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 (Hemskerk et al., 2015, chapter 3). The TB bacteria has existed for a long time with the most conservative estimates being that it is 6,000 years old, see Hemskerk et al. (2015, chapter 1) and it has caused more deaths than any other disease during the last 200 years.

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).[11] It should also be noted that the vaccine is largely ineffective in stopping transmission,

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

according to Hemskerk et al. (2015, p.53), who also observe that: "The only vaccine currently available for TB is the bacillus Calmette-Guerin (BCG) vaccine developed by serial passage of Mycobacterium bovis and introduced in 1921. 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." Other measures included bringing patients to sanatoria, which relied on self-healing; see more below.

Figure 1 shows the development of the TB mortality rate for the Danish city population from 1890 to 1950.[12] There is clearly an overall decline in mortality from the disease. 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 Figure A5 in the Appendix. 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

2.2 TB dispensaries

The National Foundation 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 foundation went on a field trip to Germany to study the system of dispensaries that was being established. Following this, the first TB dispensary was established in Copenhagen in 1908 in a five room apartment, which was funded by a private donor who put in 10,000 Danish kroner, corresponding to about 640,000 Danish kroner in present value.[13] 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 roll-out 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

sample, see also footnote 26 below.

[12]Figure A1 in the Appendix depicts a similar path for pulmonary TB mortality.

[13]The present value is as of 2014 and converted from the 1908 value using a consumer price index.

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.

Figure 2: Spread of TB dispensaries across Danish cities

The dispensaries were run by the National Foundation to begin with, but from 1928-29, the goal became to have a network of dispensaries led by specialized doctors and funded by a county or a municipality. This process was completed around 1944. 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 Foundation by offering specialized courses. From 1918, a cooperation between the association for nurses outside of Copenhagen and the National Foundation helped alleviate this problem. The lack of properly trained nurses have been suggested as a reason for the slow initial spread of dispensaries (Schelde Møller, 1950).

Before World War II, the TB 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 dispensary would supervise a number of homes of TB patients. Third, the dispensaries would perform consultations with patients. Fourth, nurses employed at the TB dispensaries would make home visits. Finally, they would direct patients to TB hospitals as well as other TB institutions. By 1927, 24 percent of the population had access to TB dispensaries, whereas by 1939, 67 percent had access (Medical Reports for 1927 and 1939). In 1927, the dispensaries had 3000 new patients and visited 15,000 homes (Medical Report for 1927). By 1939, 33,431 individuals had been referred to the dispensaries, 5,812 homes were supervised, 180,250 consultations were carried out, nurses undertook 35,288 home visits (Medical report for 1939). Finally, about 2,903 persons were directed to other TB institutions.[14] For the 1940s, we also have some information on how much personnel the dispensaries were using. In 1943, there were 89 nurses and 60 medical doctors employed at 71 dispensaries.[15]

From the 1940s, vaccination became common as mentioned above. The dispensaries performed vaccinations with statistics being reported from 1943. Thus to avoid confounding our results

[14]To get a sense of the magnitude of the increase in e.g home visits, we note that total population was 3.4 million in 1925 and 3.7 in 1940.

[15]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).

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 TB dispensaries. Specifically, we graph average city-demeaned TB death rates 10 years before and after the introduction of a TB dispensary in Figure 3.[16] While the TB death rates exhibit a downward trend, we see a clear discontinuity around the introduction of a TB dispensary. This is also illustrated by the red line which shows 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 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). This pattern is prima facie evidence that the opening of the TB 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

Prior to the establishment of the dispensaries, a number of TB sanatoria and TB hospitals were founded. 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 getting fresh air and a balance between physical and mental rest, and work therapy on the other hand (Schelde Møller, 1950, p.41). The sanatoria took care of the stronger patients, whereas weak patients were sent to TB hospitals. This practice lasted until the 1940s.

Porter (1999, pp.282-284) notes that sanatoria only provided a holiday for their inmates, and that there is no evidence that they mattered for the decline in TB mortality. The medical

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

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 also 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 and that they were, in general, established before the dispensaries. Our empirical analysis demonstrates that possible interactions between TB hospitals, sanatoria, and the dispensaries do not influence our baseline findings.

2.4 Information campaigns across the country

There were also an active effort to prevent the spread of TB by country wide information campaigns. 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 Foundation had hoped for (Schelde Møller, 1950, p.84). 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 Empirical strategy

The first step in the empirical analysis follows a 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:

$$M_{c,t}^{d\in D} = \beta_d Dispensary_{c,t} + \alpha_c + \gamma_t + \varepsilon_{c,t}^{d}, \tag{1}$$

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 $\varepsilon^d_{c,t}$ is the error term, which is allowed to cluster at the city level and to be heteroscedastic. We estimate equation (1) for the following set of diseases ($d \in D$): TB, cancer, influenza, pneumonia, accidents and 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.

The identifying assumption in estimating β_d is that the roll-out of the TB dispensaries was conditionally randomly assigned. While demonstrating that a dispensary primarily influences TB mortality provides suggestive evidence in this direction, our next approach exploits the fact that only TB was "treated" to set up a related experiment in the data. In particular, the second step of our analysis performs a triple-differences estimation:

$$M_{c,t,d} = \beta \; \mathit{Dispensary}_{c,t} \times \mathit{Prevent}_d + \phi_{c,t} + \lambda_{d,t} + \mu_{d,c} + \varepsilon_{c,t,d}, \tag{2}$$

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, $\mathit{Dispensary}_{c,t}$ is the same indicator as above but is now interacted with $\mathit{Prevent}_d$, indicating whether disease d was prevented (i.e., treated) by the dispensary, which, we assume, was only the case of TB. The $\phi_{c,t}$'s are city-by-year fixed effects, the $\lambda_{d,t}$'s are disease-by-year fixed effects, $\mu_{d,c}$ are disease-by-city fixed effects, and $\varepsilon_{c,t,d}$ is the error term. 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.

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 a given city, which makes it possible to relax the assumption of conditionally random assignment on the roll-out of the dispensaries, and we *avoid* comparing the development of TB mortality of, for example, larger cities to smaller cities.

In both estimation steps, we test for the common-pretrend assumption and study the subsequent dynamics by also reporting the results from estimating the following flexible models:

$$M^{TB}_{c,t} = \sum_{j \in T} \beta_j \times \mathit{Dispensary}^{\tau+j}_{c,t} + \alpha_c + \gamma_t + \varepsilon_{c,t}, \tag{3}$$

$$M_{c,t,d} = \sum_{j \in T} \beta_j \times Dispensary_{c,t}^{\tau+j} \times Prevent_d + \phi_{c,t} + \lambda_{d,t} + \mu_{d,c} + \varepsilon_{c,t,d}, \tag{4}$$

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 period 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 period just before the intervention. 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 in a city, while negative estimates could suggest that the roll-out of the TB dispensaries is spuriously capturing a secular trend in TB mortality.

4 Data

This section presents an overview of the data collected for the empirical analysis. Description and definitions of the variables used in the analysis are reported in Appendix Table A1, along with detailed accounts of the data sources. Data on the timing of the TB dispensaries are collected from the annual publication "Medicinalberetning for Kongeriget Denmark" published by Sundhedsstyrelsen,[17] from 1890 and onwards; we also obtain the date of commissioning of TB hospitals, sanatoria, and waterworks.[18] Disease statistics were digitized from the annual publications "Dødsårsagerne i Kongeriget Danmarks byer" published by Sundhedsstyrelsen which contains city-level data from 1890 to 1919. From 1920, rural districts are added and the publication 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.[19] From 1901 these statistics become more detailed, and from this year we are able to compile a disease panel. Our robustness analysis uses

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

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

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

pre-intervention income and inequality data, which are taken from "Statistiske Meddelelser" published by Statistics Denmark. In the end, we obtain an unbalanced panel of 87 cities observed over the period 1890-1950. In the baseline analysis, we cut off the data in 1939. This is, however, 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.

We further note that diagnosing the disease 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).[20] The aforementioned discovery by Ehrlich allowed diagnosing active, rather than latent, TB by examining the sputum of a patient.[21] 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.

We finally note that disease registration on pre-printed forms had been in place in the cities since 1856 (Johansen, 2002, p.131).[22] 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 Tables 1 and 2. The aggregate development of the other causes of deaths is depicted in the Appendix Figures A5-A11.

Table 1: Summary statistics

Table 2: Number of TB dispensaries and TB mortality over time

5 Results

We begin the analysis by presenting estimates of equation (1), which exploits year and city variation for the purpose of identification, corresponding to a standard difference-in-differences

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

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

[22] 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.

model. These estimates are presented in subsection 5.1. Subsection 5.2 reports the results from the triple-differences model of equation (2), which adds the control diseases to the setup. For both strategies, the results from estimating the flexible models are reported in subsection 5.3.

5.1 Difference-in-differences model

Table 3 reports the baseline results from estimating equation (1). 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). Moreover, as TB mortality per 1,000 reduced by 1.7 from 1907 to 1939 in the Danish city population, our baseline estimate suggests that the network of TB dispensaries explains about 20 percent of this development.

The baseline estimate remains stable both in magnitude and statistical significance when adding the lagged TB mortality rate in column (3).[23] The small magnitude on the lagged TB mortality rate indicates that the full effect a TB dispensary is attained just one year after the implementation. Next, column (4) considers only pulmonary TB mortality as this particular form of TB accounts for the bulk of the variation in the overall TB mortality rate. We find a negative and statistically significant coefficient at the one percent level, which is rather similar in magnitude compared to the baseline.

Table 3: Effect of TB dispensaries using city by year data on TB

Placebo models Table 4 reports the results when using mortality from cancer, influenza, pneumonia, accidents and suicides, scarlet fever, diphtheria, and polio as the outcomes in equation (1). As seen from the table, the estimated coefficients are mostly negative, but small in numerical magnitude and always statistically highly insignificant. For example, the estimated coefficient for pneumonia mortality, which arguably is the disease most similar to TB, is about 65 percent smaller in numerical magnitude compared to the baseline (i.e., $\hat{\beta} = -0.074$; standard

[23] As we have time series for nearly 50 years, we are not strongly concerned about Nickell bias. This is confirmed when using the alternative Arellano-Bond estimator, which gives a similar result [available upon request].

error = 0.059), and the coefficients on the remaining placebo diseases are even smaller. This pattern suggests that the TB dispensaries mainly served to reduce TB mortality and indicates that our baseline estimate is not simply picking up a general trend in mortality.

Table 4: Placebo panel regressions

Fertility, infant mortality, and overall mortality Table 5 investigates whether the TB dispensaries had an impact on fertility, infant mortality and overall mortality. Acemoglu and Johnson (2007) argue that the epidemiological transition mechanically led to higher fertility due to more women surviving to birth giving ages. Columns (1) and (2) show that while the estimated coefficients have the expected positive signs, the impact on the crude birth rate is statistically insignificant.

The estimates, reported in columns (3) and (4), reveal that the roll-out of the TB dispensaries reduced the infant mortality rate by about by 9 percent, supporting the argument in Bütikofer and Salvanes (2015) that eradicating TB benefited very young children as well.

Next, we investigate whether the TB dispensaries had an impact on overall mortality as measured by the crude death rate. Columns (5) and (6) report negative coefficients which are significant at the 10 percent level. Thus, while the effect on overall mortality is imprecisely estimated, it does have the expected sign. The semi-elasticity, reported in column (5), reveals that an introduction of a TB dispensary would reduce the overall mortality rate by circa three percent, which can be compared with the baseline semi-elasticity for the TB mortality rate equal to -0.16. These estimates suggest that the TB dispensaries reduced the disease death ratio for TB, which is also confirmed in Appendix Table A2, where the disease death ratio for TB is the outcome of interest.[24]

Columns (7) and (8) reveal a positive correlation between TB mortality and overall mortality, suggesting that, in fact, reducing TB mortality reduces overall mortality as also indicated by the previous estimates. This is also consistent with the pattern in the raw data: in the 1901, the TB mortality share out of total mortality was about 13 percent, while in 1939 the share was reduced to three percent, and throughout this period, the overall mortality rate fell with 34 percent.

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

Table 5: Effect of TB dispensaries using city by year data on other outcomes

5.2 Triple-differences model

Table 6 reports the findings of estimating equation (2), 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 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/divergence in, e.g., income, income inequality, or mortality; local political/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) reports the estimate only controlling for city, disease and year fixed effects. This yields a larger, in absolute value, coefficient than before. However, when adding city-by-year fixed effects, disease-by-year fixed effects, and disease-by-city fixed effects, which is the full triple-differences model, in column (2), we obtain an estimate which is quite similar to the baseline difference-in-differences estimate, that is, $\hat{\beta} = 0.19$ with standard error = 0.07. Thus, both strategies provide estimates that are very close to each other, both in terms of magnitude and statistical significance, supporting the assumption of conditional random assignment of the roll-out in the difference-in-differences estimation.

Next, columns (3) and (4) add a lagged dependent variable. The estimate does not change and the magnitude of the lagged TB rate in column (3) indicates that the full effect of a dispensary is attained within two years after the intervention. When controlling for *all* interactions of the fixed effects in column (4), the coefficients on the lagged TB rate deflates and turns statistically insignificant, indicating that the TB dispensary had a more or less instantaneous effect on TB mortality.

Table 6: Effect of TB dispensaries using city by year by disease data

5.3 Flexible model

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 any TB dispensary by showing that there were no systematic trends prior to its introduction. Moreover, the subsequent dynamics, which is also revealed in the flexible 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 flexible difference-in-differences and triple-differences models, along with their 95 percent confidence intervals in Figure 4.[25] 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 (3) become significantly negative and, although the confidence interval of the coefficients estimated from equation (4) are wider, we observe that the majority of the coefficients are significant at the 10 percent level. The patterns of the estimates are consistent with the effects being stronger after a couple of years have passed, which is in line with the fact that the median time to death of untreated TB is 2.5 years (Goodman and Fuller, 2015).[26]

In Appendix Table A4, we show an alternative test of the common-pretrend assumption, which includes intervention lead dummy variables. If a TB dispensary was introduced due to an unusual high level of TB mortality, the coefficients on the leads would come out positive and statistically significant, while negative estimates could suggest that the roll-out of the TB dispensaries is spuriously capturing a secular trend in TB mortality. Reassuringly, however, the leads never come out statistically significant (individually or jointly) in both the difference-in-differences and the triple-differences models. In fact, only the estimated coefficients on the intervention variable in the period of implementation are statistically significant and the magnitude is similar to the baseline estimate.

Figure 4: Flexible estimates of impact on TB before and after TB dispensary

[25] The regression tables are, however, also shown in Appendix Table A3.

[26] Nagelkerke (2012) agrees with Goodman and Fuller (2015), but notes that death can happen after a few weeks.

6 Robustness

We have carried out a number of robustness checks based on both the difference-in-differences model and the triple-differences model. First, we demonstrate that the result from the triple-differences model is robust to limiting the set of control diseases. Second, we turn to other public health policies targeted at TB in the form of sanatoria and TB hospitals. Third, we investigate whether the results are confounded by the introduction of the BCG vaccine. Fourth, we address that the roll-out of the TB dispensaries and TB mortality could be related to the population size. Fifth, as poverty and inequality are possible determinants of TB incidences and mortality, we control for the pre-intervention income and a pre-intervention city-Gini coefficient. Finally, we discuss additional robustness checks reported in the Appendix.

Subset of control diseases As discussed in Section 2.4, 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 spill-over effects on other (airborne) diseases, one could worry that there are spill-over 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 TB dispensaries only affected TB through general information that potentially could affect similar infectious diseases we should find a smaller effect. Secondly, 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. Table 7 presents the results of the limited control disease panels. Column (1) and (2) report the results of the triple-differences models estimated with the disease panel using scarlet fever and pneumonia as controls. When not controlling for all the interactions of the fixed effects in column (1), the magnitude of the coefficient on the TB dispensary is smaller in absolute value compared to the baseline estimate, but once we control for all possible fixed effects in column (2), the estimated coefficient corresponds to the baseline. Column (3) and (4) show the estimation with cancer and accidents and suicides as control diseases. In column (3), which does not include all fixed effects, the coefficient appears larger, but once we include all fixed effects in column (4) the results are very similar to the baseline.

Table 7: Effect of TB dispensaries using city by year by disease data with alternative disease panels

As the effect of the TB dispensaries estimated using similar infectious diseases as controls in column (2) is only slightly smaller than the estimated effect using non-infectious diseases as controls in column (4), it is unlikely that the impact of the TB dispensaries on TB mortality should be attributed to general information on hygienic practices of the time that potentially would affect similar diseases.

Sanatoria and TB hospitals Table 8 investigates the robustness of our results to the commissioning of TB hospitals and sanatoria. Column (1), the difference-in-differences model, augments the baseline specification with an indicator equal to one if a TB hospital was build in a given city over the considered time period. In the triple-differences model in column (2), the same indicator is included and interacted with the $Prevent_{TB}$ indicator defined as in equation (2). In columns (3) and (4), we include an interaction term between the TB hospital and TB dispensary indicators to test for possible complementarities, which Holm (1946a) argued were present. Both the coefficients on TB hospital and the interaction term enters insignificantly into the equation. Compared to the baseline results, the coefficients on the TB dispensary are about the same size and highly significant, except when we control for the interaction between TB hospital and TB dispensary in columns (3) and (4), where the coefficients on TB dispensary are only significant at the 10 percent level. We attribute this to the inclusion of extra insignificant variables, decreasing the precision of the estimate.

Table 8: Effect of TB dispensaries controlling for commissioning of TB hospitals and sanatoria

In columns (5) to (8) of Table 8, we proceed by controlling for the commissioning of sanatoria in a similar way, with the TB hospitals. Columns (5) and (6) include an indicator equal to one if a city is within 50 km of a sanatorium, and in the triple-differences model in column (6), the indicator is interacted with the $Prevent_{TB}$ indicator. Columns (7) and (8) likewise include the interaction between TB dispensary and sanatorium. We treat all cities within a 50 km radius of a sanatorium as it seems plausible that a sanatoria close to a city are more likely to treat patients from that city. The results are largely unchanged when we include the sanatorium indicator in the difference-in-differences model and the triple-differences model, respectively. Sanatoria show no significant effect in either model, supporting the hypothesis of Porter (1999) that they did not matter for the decline in TB mortality. We find no complementary effect of sanatorium and TB dispensary when we include the interaction, although we note that the coefficient on the TB dispensary in both the difference-in-differences model and the triple differences model of columns (7) and (8), respectively, increases in absolute magnitude. Finally, columns (9) and (10) include both the TB hospital and sanatorium indicators. Including these variables does not impact our main result.

Controlling for the BCG vaccine The BCG vaccine only became widespread in Denmark in the 1940s, but the remote island of Bornholm experimented with the vaccine from 1936. Hence, Appendix Table A5 shows that our results are robust to excluding the cities on Bornholm and limiting the panel to 1935.[27] Given that the BCG vaccine is ineffective against pulmonary TB, the main component of TB mortality, it is hardly surprising that the results are largely unaffected. Moreover, limiting the panel to 1935 also exludes the possibility that our results are influenced by the roll-out a universal home visiting program for mothers and their infants starting in Denmark in 1937 (Wüst, 2012).

Excluding large cities and early adopters Columns (1) and (2) of Table 9 report the results when excluding Copenhagen—the capital and largest city—in the difference-in-differences and the triple-differences settings. Columns (3) and (4) show the results when excluding the five largest cities, and columns (5) and (6) exclude all cities that adopted a TB dispensary before 1920.[28] The results are very similar to the baseline, although we find that the absolute magnitude of the coefficient rises slightly when excluding the five largest cities and the pre-1920 adopters

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

[28] 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.

of TB dispensaries. Appendix Table A6 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 triple-differences setting, the linear trend is a special case of the non-parametric city specific trend, we control for by city-by-year fixed effects.

Table 9: Effect of TB dispensaries excluding large cities and early adopters

Controlling for income and inequality before the intervention Next, we address that 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 difference-in-differences analysis, if the income level of the cities affected the roll-out of the TB 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 triple-differences model, as we control for time varying city fixed effects and time varying disease fixed effects. Yet, if income affected TB mortality more strongly as compared to other diseases, this would remain an issue. We note that this concern has already been addressed somewhat by using similar infectious diseases, such as pneumonia and scarlet fever, as controls which are also likely to be affected by income. However, to further address this, we present results controlling for pre-intervention income and income inequality. Columns (1) and (2) of Table 10 present estimates conditioning on the log of the total income of the citizens liable to pay income taxes for the tax year 1904-05 per capita interacted with time fixed effects.[29] 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 the cities. Columns (3) and (4) include the Gini coefficient of 1918-1919 interacted with time fixed effects and exclude cities which adopted a TB dispensary before 1920 to control for inequality within the cities.[30,31] When we control for pre-intervention income,

[29] 1904-1905 denotes tax years. Denmark introduced income taxation in 1903 (Aidt and Jensen, 2009) for which reason taxable income is available from 1904 onwards.

[30] 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. Non 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

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

reported in columns (1) and (2), and pre-intervention inequality, reported in columns (3) and (4), the results are similar to baseline, although marginally larger numerically.

Table 10: Effect of TB dispensaries controlling for pre-intervention income and inequality

Additional robustness checks We finally mention a number of additional robustness checks reported in the Appendix. First, 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 (Cutler and Miller, 2005, Alsan and Goldin, 2015). We note that TB is not a water-born disease, but clean water could have general implication for overall mortality. As revealed by Appendix Table A7, this does not change our findings.[32] Hence, the effect of the TB dispensaries primarily derives from the specific information and prevention targeted at TB.

Second, 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 worked at TB hospitals (Holm, 1946a). To analyze whether the main dispensaries had a different effect than their branches, we split the TB dispensary indicators in equation (1) and (2) 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 A8. 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.

Finally, Appendix Table A9 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. We note that the absolute magnitude of the coefficient on TB dispensary is reduced when extending the year to 1946, although it

[32]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.

is still highly statistically significant. The reduced coefficient is to be expected, as the result is confounded by the roll-out of the BCG vaccine, and the advent of streptomycin, the first effective drug against TB.

7 Concluding remarks

This research has shown that the introduction of TB dispensaries reduced TB mortality. This holds both in a difference-in-differences setup using time and city variation as well as when deploying triple-differences estimation. The baseline result is robust to large number of checks including controlling for initial population, pre-intervention income and inequality, different sample periods, and city specific linear trends.

Our results grant a role for public health institutions for the observed decline in TB mortality prior to the advent of modern medicine. Yet, it is important to 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. Yet, we also note that other public health institutions such as TB hospitals and sanatoria seem to have had little or no effect on the development of TB mortality over this period.

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.

While the TB dispensaries had an effect on mortality, we leave the question as to whether they also had economic effects unanswered. In future research, we therefore plan to investigate whether the TB dispensaries also had direct impacts on local incomes and education levels.

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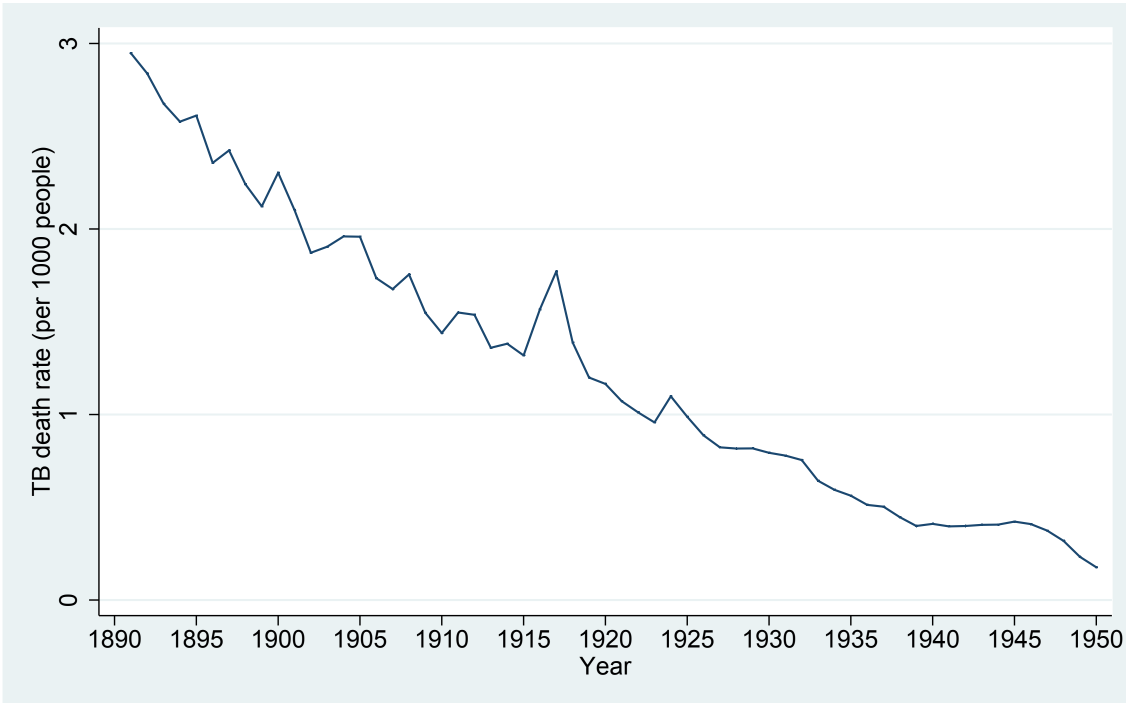
Appendix

This appendix contains Tables A1-A8 and Figures A1-A10 as referred to in the main text.

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

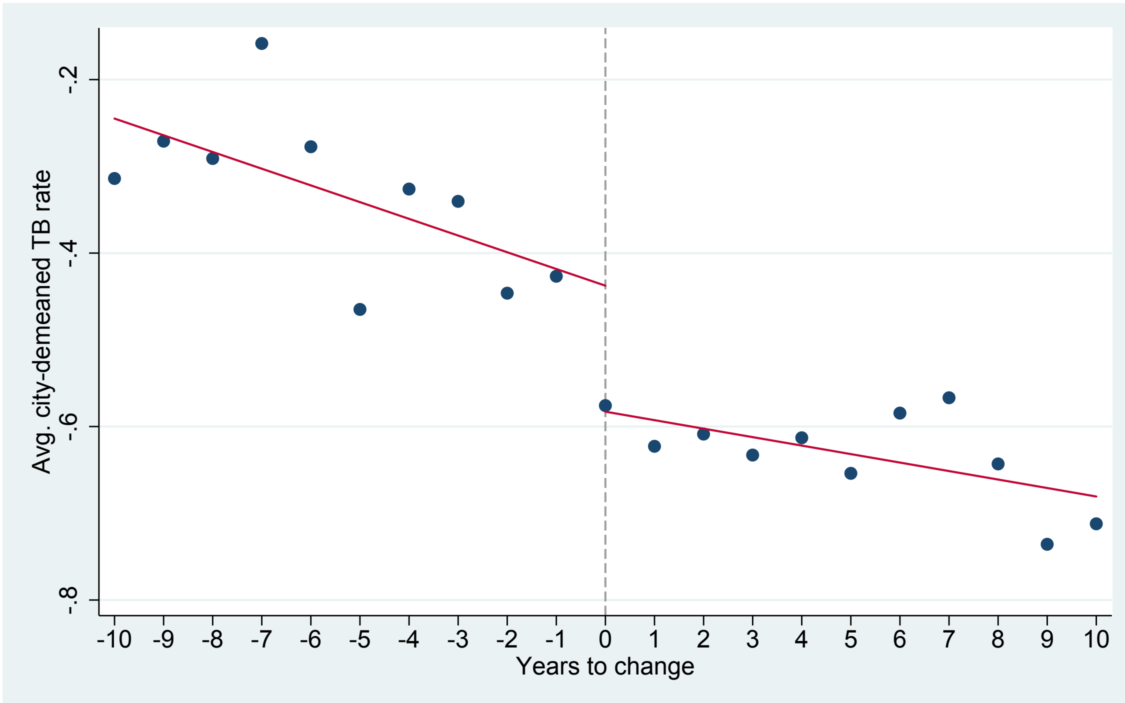


Notes: The data show the national development of TB mortality in the Danish cities per 1,000 people. Source: the Cause of Death Statistics (1890-1950) and the authors own calculations.

Figure 2: Spread of TB dispensaries across Danish cities

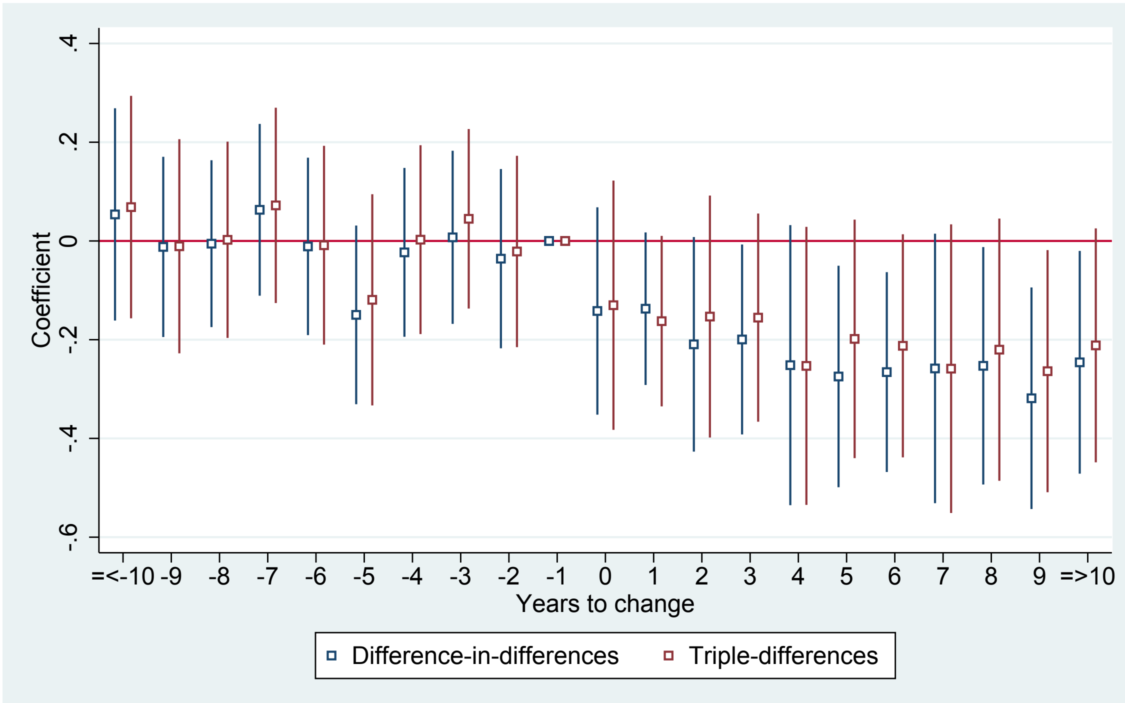
Notes: The maps show cities in the sample, and when TB dispensaries were established. Source: the Medical Reports (1890-1950).

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: Flexible 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.

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
Infant mortality rate	1901–1939	3,165	92.802	44.789	0.000	400.000
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
Flue 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: 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 3: Effect of TB dispensaries using city by year data on TB

Dep. variable:	TB rate	log(TB rate)	TB rate	Pulmonary rate
	(1)	(2)	(3)	(4)
TB dispensary$_{c,t}$	-0.2187^{***}	-0.1587^{***}	-0.1885^{***}	-0.1680^{***}
	(0.0783)	(0.0540)	(0.0666)	(0.0577)
TB rate$_{c,t-1}$			0.1489^{***}	
			(0.0248)	
Long run effect			-0.2215^{***}	
City FE	Yes	Yes	Yes	Yes
Year FE	Yes	Yes	Yes	Yes
Time period	1890-1939	1890-1939	1891-1939	1901-1939
Observations	3,981	3,662	3,894	3,165
R-squared	0.5333	0.5801	0.5389	0.3886
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, in column (2) the log is taken of this variable, and in column (4) 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$_{c,t-1}$ is a lagged dependent variable. Long run effect is the steady-state value of the estimated model in column (3). Robust standard errors clustered at the city level are in parentheses.
*, **, and ***, determine significance levels of 10%, 5% og 1% respectively.

Table 4: Placebo panel regressions

	Baseline	Placebo						
Dep. variable:	TB rate	Cancer rate	Flue rate	Pneumonia rate	Accident and suicide rate	Scarlet fever rate	Diphtheria 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
	(0.0680)	(0.0524)	(0.0269)	(0.0588)	(0.0261)	(0.0065)	(0.0150)	(0.0025)
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 5: Effect of TB dispensaries using city by year data on other outcomes

Dep. variable:	Birth rate	log(Birth rate)	Infant mortality rate	log(Infant mortality rate)	Death rate	log(Death rate)	Death rate	log(Death) rate)
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
TB dispensary$_{c,t}$	0.2891	0.0276	-9.3230^{**}	-0.0939^{**}	-0.5143^{*}	-0.0377^{*}		
	(0.5061)	(0.0260)	(3.6678)	(0.0420)	(0.2871)	(0.0216)		
TB rate$_{c,t}$							1.4617^{***}	
							(0.1020)	
log(TB rate$_{c,t}$)								0.1192^{***}
								(0.0095)
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	1890-1939	1890-1939	1890-1939	1890-1939
Observations	3,165	3,165	3,165	3,056	3,981	3,981	3,981	3,662
R-squared	0.6712	0.6508	0.2684	0.2687	0.4852	0.4671	0.5617	0.5584
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 number of births excluding still borns per 1,000 people, in column (2) the log is taken of this variable, in column (3) the left-hand-side variable is the mortality among 0 to 1 year old per 1,000 live births, in column (4) the log is taken of this variable, in column (5) and (7) the left-hand-side variable is the mortality excluding still borns per 1,000 people, in column (6) and (8) 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, TB rate$_{c,t}$ is the TB mortality per 1,000 people, and log(TB rate$_{c,t}$) is the log of this 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 using city by year by disease data

Dep. variable:	Disease			
	(1)	(2)	(3)	(4)
TB dispensary$_{c,t}$	-0.3010^{***}	-0.1885^{***}	-0.2187^{***}	-0.1795^{**}
	(0.0553)	(0.0708)	(0.0432)	(0.0686)
Disease$_{c,t-1}$			0.2158^{***}	0.0224
			(0.0224)	(0.0181)
Long run effect			-0.2788^{***}	-0.1837^{***}
City FE	Yes	Yes	Yes	Yes
Year FE	Yes	Yes	Yes	Yes
Disease FE	Yes	Yes	Yes	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	1901-1939	1901-1939	1902-1939	1902-1939
Observations	25,320	25,320	24,624	24,624
R-squared	0.5071	0.6969	0.5314	0.6973
Cities	87	87	87	87

Notes: The table reports least squares estimates. In column (1) to (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, year, and disease fixed effects, and addtionally column (2) and (4) include disease-by-year fixed effects, disease-by-city fixed effects, 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$_{c,t-1}$ is a 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 7: 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)	(3)	(4)
TB dispensary$_{c,t}$	-0.2262^{***}	-0.1681^{**}	-0.3554^{***}	-0.1855^{**}
	(0.0499)	(0.0843)	(0.0561)	(0.0871)
City FE	Yes	Yes	Yes	Yes
Year FE	Yes	Yes	Yes	Yes
Disease FE	Yes	Yes	Yes	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	1901-1939	1901-1939	1901-1939	1901-1939
Observations	9,495	9,495	9,495	9,495
R-squared	0.5072	0.7413	0.2999	0.6477
Cities	87	87	87	87

Notes: The table reports least squares estimates. In column (1) and (2) the left-hand-side variable is the stacked causes of death from TB, pneumonia, and scarlet fever per 1,000 people. In column (3) and (4) 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, and disease fixed effects, and addtionally column (2) and (4) include disease-by-year fixed effects, disease-by-city fixed effects, 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 8: Effect of TB dispensaries controlling for commissioning of TB hospitals and sanatoria

Dep. variable:	TB rate	Cause of death	TB rate	Cause of death	TB rate	Cause of death	TB rate	Cause of death	TB rate	Cause of death
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)
TB dispensary$_{c,t}$	−0.2226***	−0.1892***	−0.1961*	−0.1588*	−0.2181***	−0.1865***	−0.3127***	−0.3011***	−0.2220***	−0.1873***
	(0.0783)	(0.0702)	(0.1006)	(0.0841)	(0.0786)	(0.0705)	(0.1129)	(0.1140)	(0.0785)	(0.0698)
TB hospital$_{c,t}$	0.0316	0.0129	0.0501	0.0379					0.0318	0.0145
	(0.0903)	(0.0935)	(0.1011)	(0.1026)					(0.0904)	(0.0937)
TB dispensary$_{c,t}$ × TB hospital$_{c,t}$			−0.0719	−0.0847						
			(0.1290)	(0.1177)						
Sanatorium$_{c,t}$					−0.0142	0.0774	−0.0207	0.0696	−0.0145	0.0777
					(0.0945)	(0.1186)	(0.0963)	(0.1206)	(0.0945)	(0.1186)
TB dispensary$_{c,t}$ × Sanatorium$_{c,t}$							0.1114	0.1390		
							(0.1332)	(0.1319)		
City FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Year FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Disease FE	No	Yes	No	Yes	No	Yes	No	Yes	No	Yes
Disease FE × Year FE	No	Yes	No	Yes	No	Yes	No	Yes	No	Yes
Disease FE × City FE	No	Yes	No	Yes	No	Yes	No	Yes	No	Yes
City FE × Year FE	No	Yes	No	Yes	No	Yes	No	Yes	No	Yes
Time period	1890-1939	1901-1939	1890-1935	1901-1935	1890-1939	1901-1939	1890-1935	1901-1935	1890-1935	1901-1935
Observations	3,981	25,320	3,981	25,320	3,981	25,320	3,981	25,320	3,981	25,320
R-squared	0.5333	0.6969	0.5334	0.6969	0.5333	0.6969	0.5334	0.6969	0.5333	0.6969
Cities	87	87	87	87	87	87	87	87	87	87

Notes: The table reports least squares estimates. In column (1), (3), (5), (7), and (9) the left-hand-side variable is the TB mortality per 1,000 people, in column (2), (4), (6), (8), and (10) 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), (8), and (10) include disease fixed effects, disease-by-year fixed effects, disease-by-city fixed effects, 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$_{c,t}$ is an indicator variable equal to one after the introduction of a TB hospital in the city, Sanatorium$_{c,t}$ is an indicator variable equal to one after the introduction of a sanatorium within 50 km of the city, and TB dispensary$_{c,t}$ × TB hospital$_{c,t}$ and TB dispensary$_{c,t}$ × Sanatorium$_{c,t}$ are interactions of the aforementioned indicators. 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 9: 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^{***}
	(0.0806)	(0.0716)	(0.0878)	(0.0739)	(0.0935)	(0.0854)
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 fixed effects, disease-by-year fixed effects, disease-by-city fixed effects, 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.

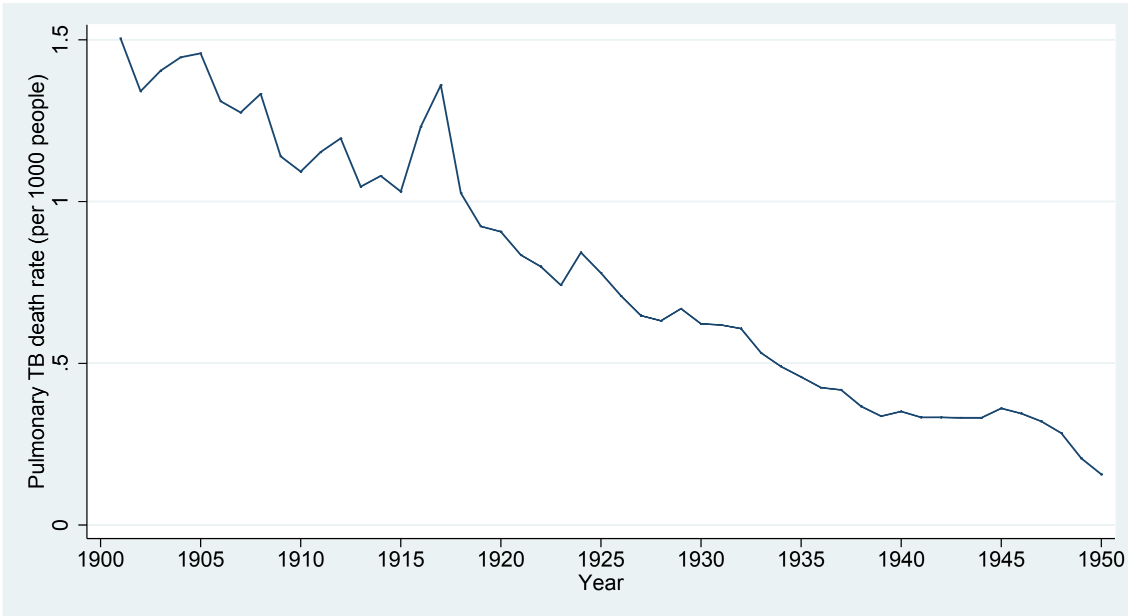
*, **, and ***, determine significance levels of 10%, 5% og 1% respectively.

Table 10: Effect of TB dispensaries controlling for pre-intervention income and inequality

Dep. variable:	TB rate	Disease	TB rate	Disease
	Baseline panel		Excluding pre 1920 adopters	
	(1)	(2)	(3)	(4)
TB dispensary$_{c,t}$	-0.2310^{***}	-0.2080^{***}	-0.2362^{**}	-0.2566^{***}
	(0.0765)	(0.0670)	(0.1002)	(0.0865)
Pre-int. log(income) × Year Fe	Yes	Yes	No	No
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
Pre-int. Gini × Year Fe	No	No	Yes	Yes
City FE × Year FE	No	No	No	No
Time period	1890-1939	1901-1939	1890-1939	1901-1939
Observations	3,981	25,320	3,341	20,992
R-squared	0.5406	0.6489	0.5113	0.6432
Cities	87	87	68	68

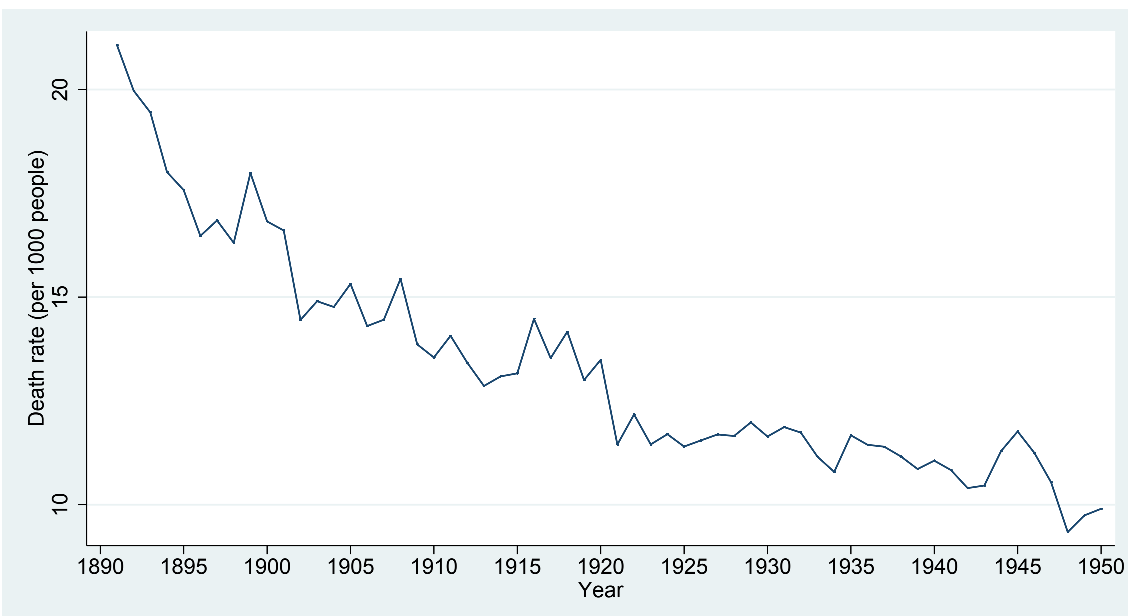
Notes: The table reports least squares estimates. In column (1) and (3) the left-hand-side variable is the TB mortality per 1,000 people, and in column (2) and (4) the left-hand-side variable is the stacked causes of death from TB, cancer, influenza, pneumonia, accidents and suicides (including homicides 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) include disease fixed effects, disease-by-year fixed effects, and disease-by-city 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 1904-05 income per capita interacted with year fixed effects is included, and in column (3) and (4) the 1918-19 income distribution based Gini coefficient interacted with year fixed effects is included. For cities included in the dataset later than 1904 and 1918, we use income per capita and Gini coefficients in the year they are added to the data. In column (3) and (4) 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.

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



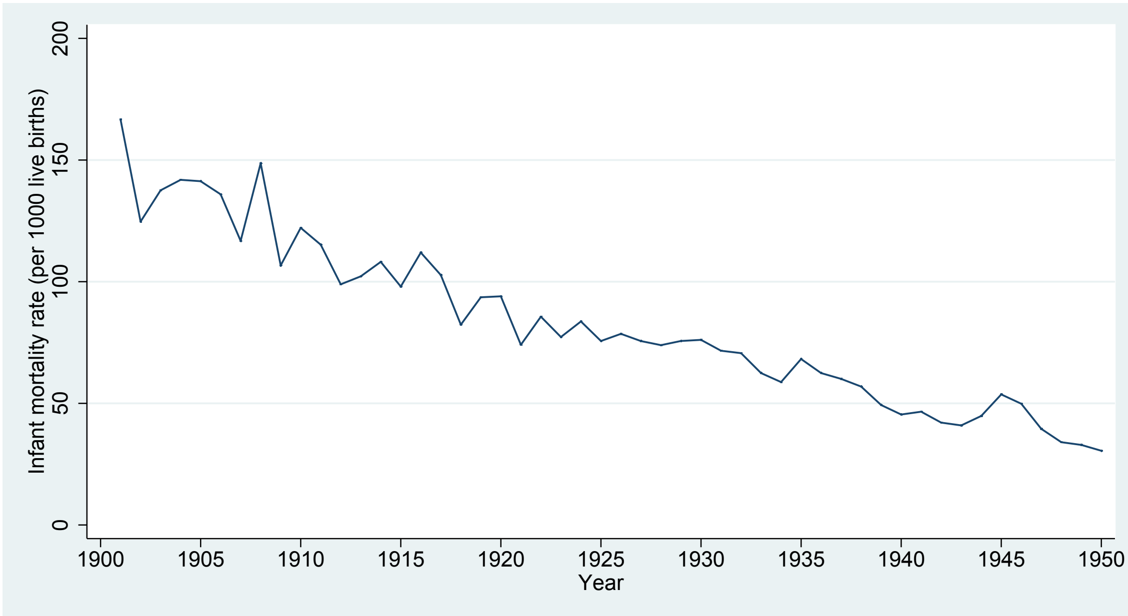
Notes: The data show the national development of pulmonary TB mortality in the Danish cities per 1,000 people. Source: the Cause of Death Statistics (1901-1950) and the authors own calculations.

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



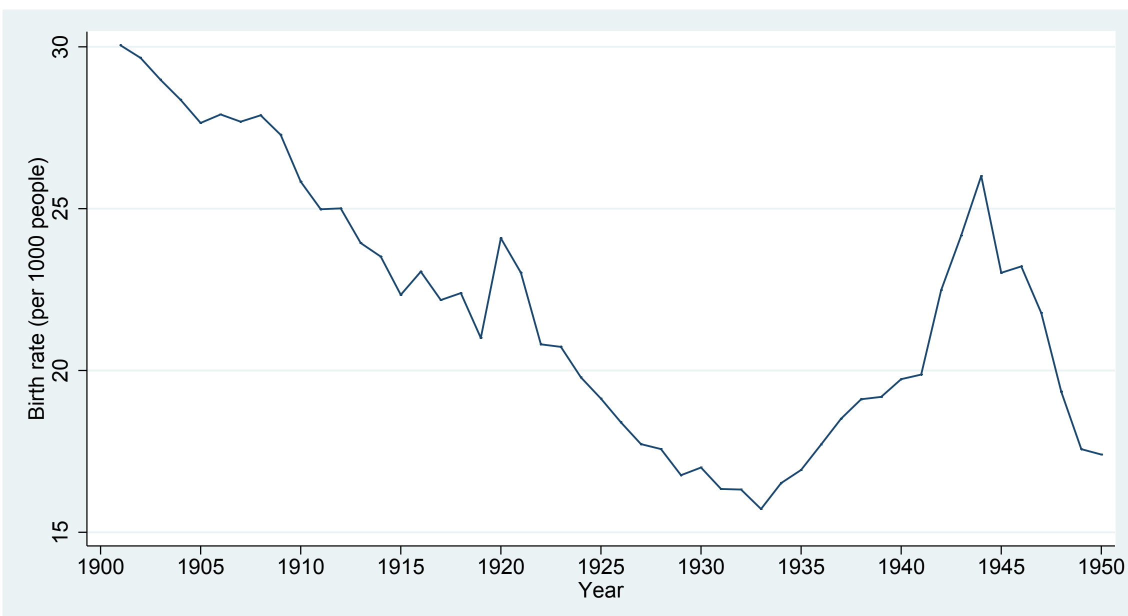
Notes: The data show the national development of mortality excluding still borns in the Danish cities per 1,000 people. Source: the Cause of Death Statistics (1901-1950) and the authors own calculations.

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



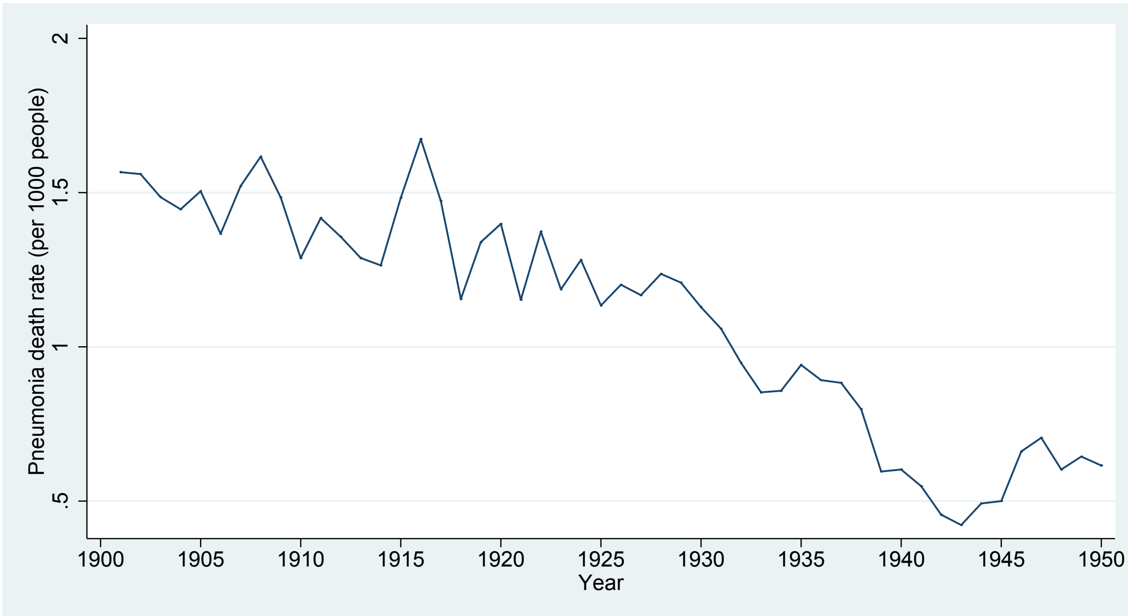
Notes: The data show the national development of mortality among 0 to 1 year old in the Danish cities per 1,000 live births. Source: the Cause of Death Statistics (1901-1950) and the authors own calculations.

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



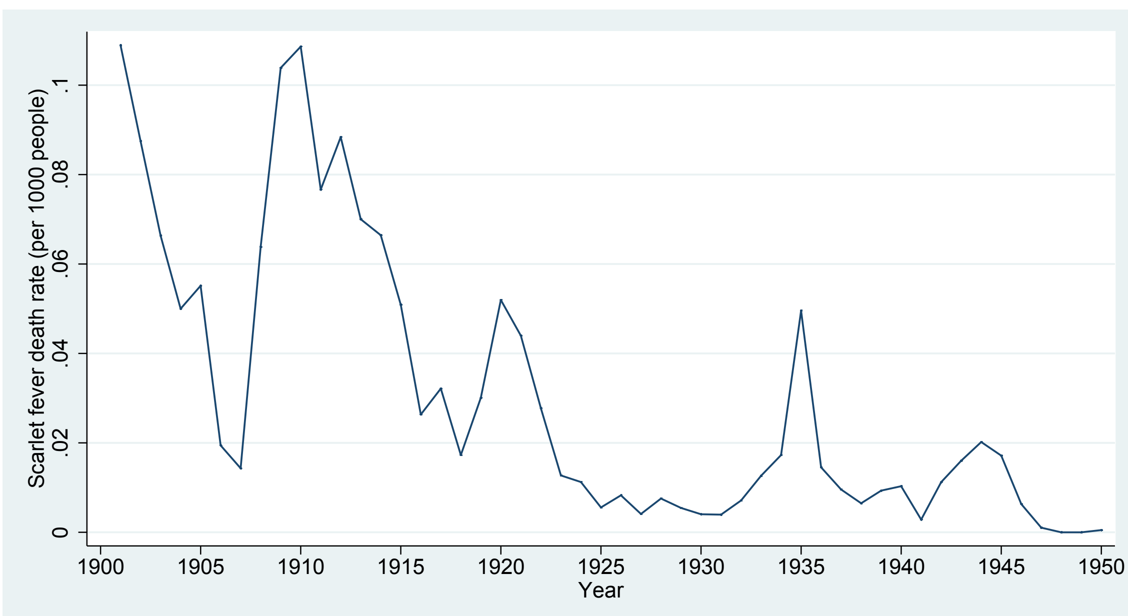
Notes: The data show the national development of the birth rate in the Danish cities per 1,000 people. Source: the Cause of Death Statistics (1901-1950) and the authors own calculations.

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



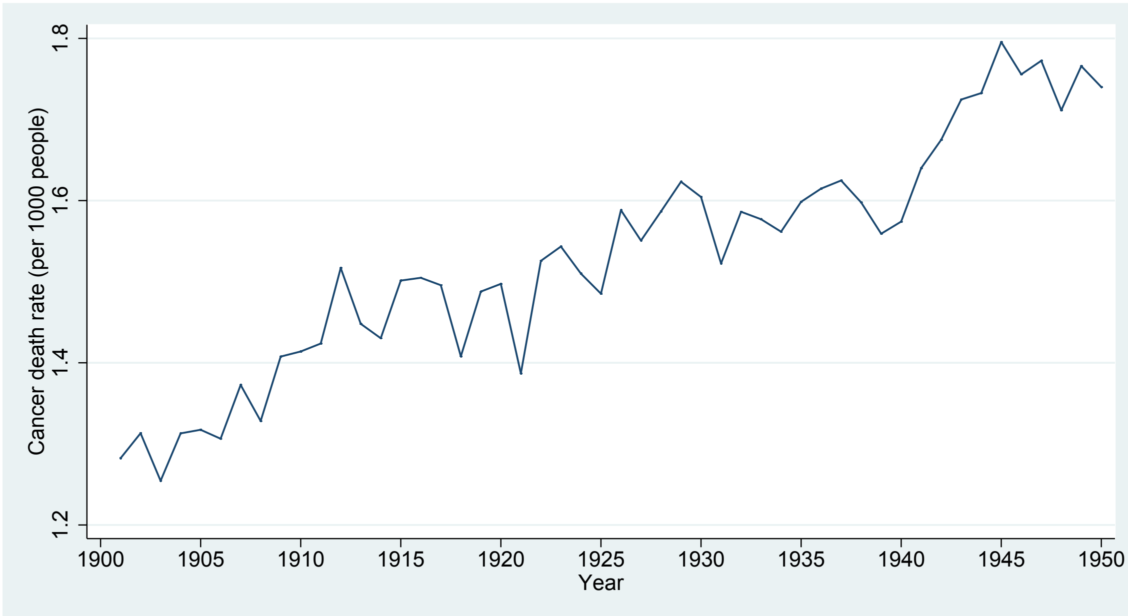
Notes: The data show the national development of pneumonia mortality in the Danish cities per 1,000 people. Source: the Cause of Death Statistics (1901-1950) and the authors own calculations.

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



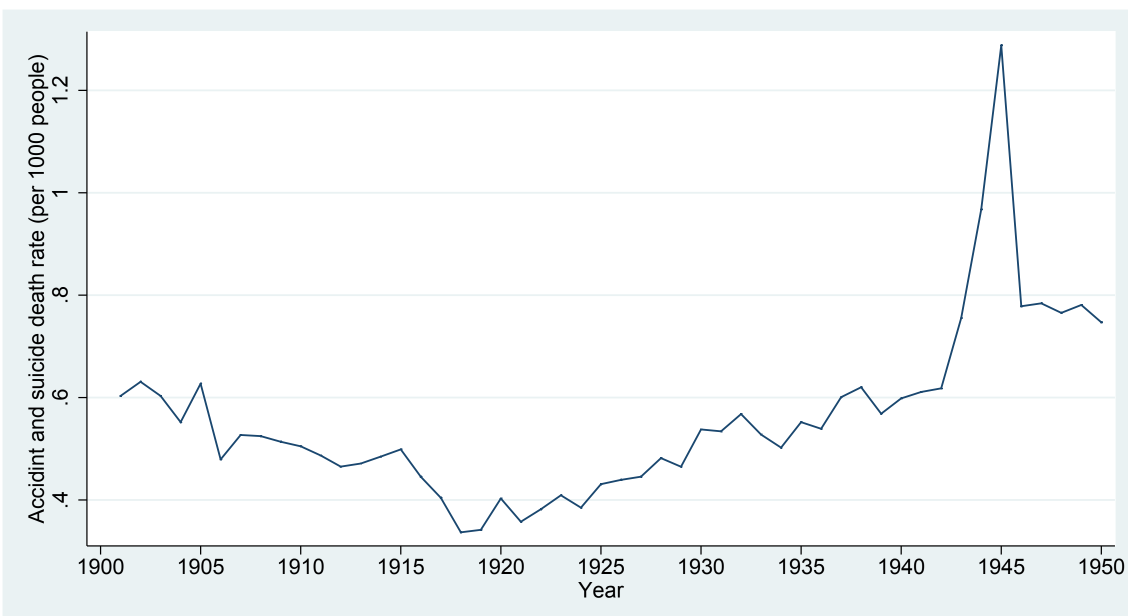
Notes: The data show the national development of scarlet fever mortality in the Danish cities per 1,000 people. Source: the Cause of Death Statistics (1901-1950) and the authors own calculations.

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



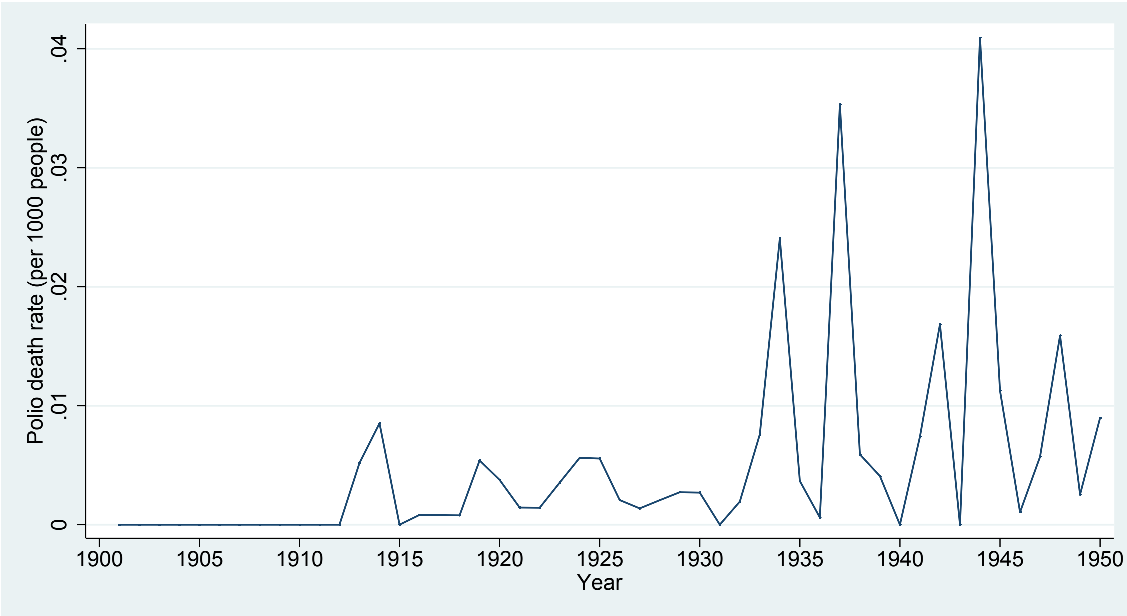
Notes: The data show the national development of cancer mortality in the Danish cities per 1,000 people. Source: the Cause of Death Statistics (1901-1950) and the authors own calculations.

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



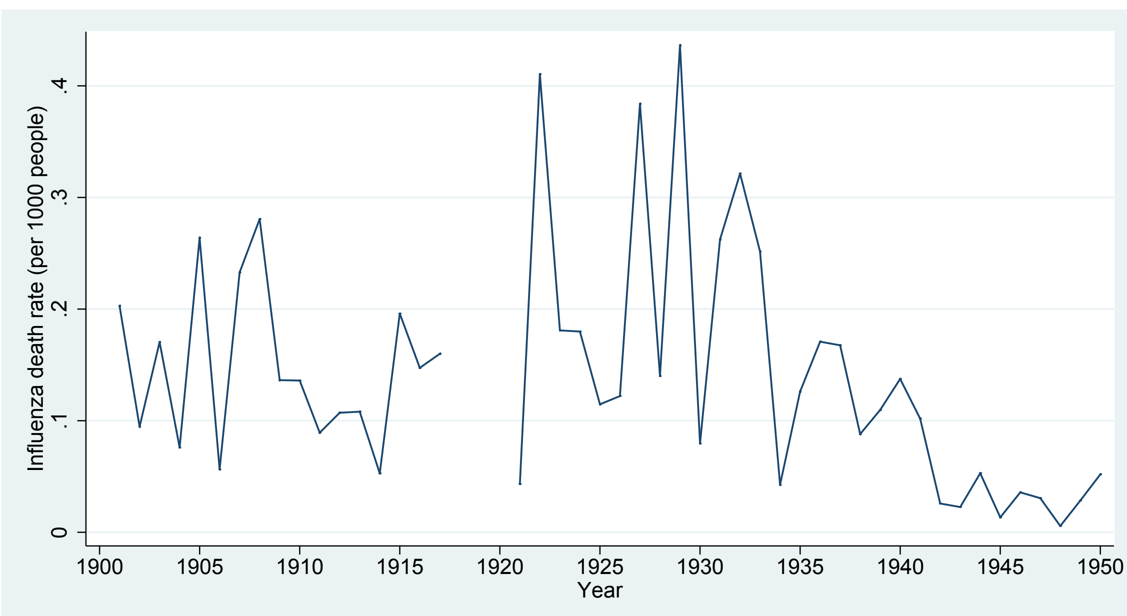
Notes: The data show the national development of deaths from accidients and suicids in the Danish cities per 1,000 people. From 1931 the data also includes the number of deaths from homocides. Source: the Cause of Death Statistics (1901-1950) and the authors own calculations.

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



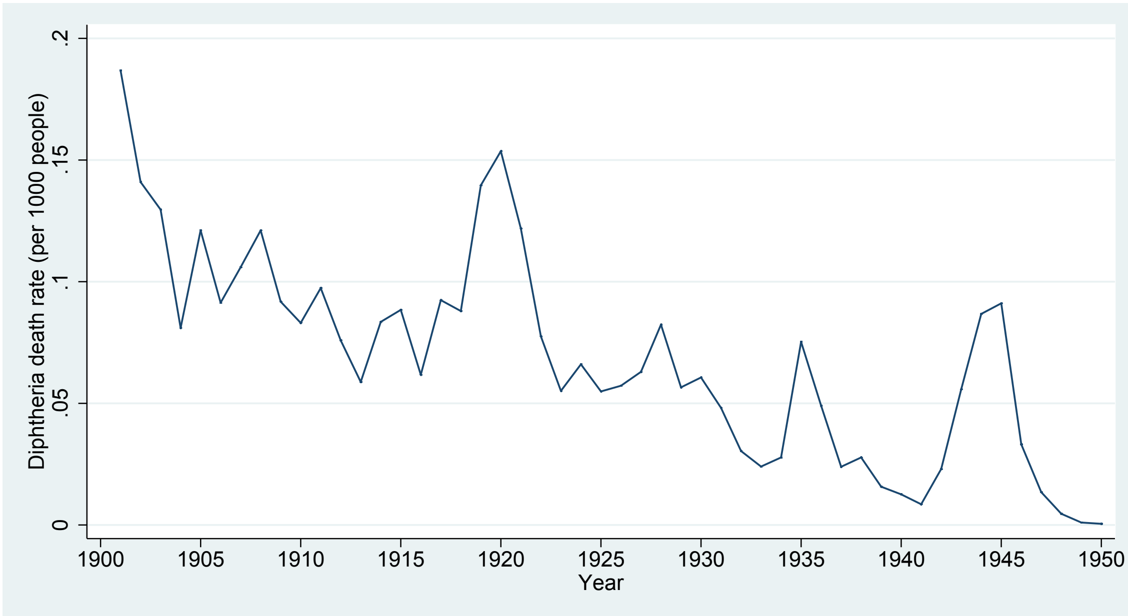
Notes: The data show the national development of polio mortality in the Danish cities per 1,000 people. Source: the Cause of Death Statistics (1901-1950) and the authors own calculations.

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



Notes: The data 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 data 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.

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).
Infant mortality rate:	Number of deaths among 0 to 1 year old per 1,000 live births. 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).
Flue 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, 1914, 1918, 1922).
Gini:	The Gini coefficient for the inhabitants liable to pay taxes calculated using the lowest point in the intervals of the income distribution from. 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: Effect of TB dispensaries on the TB death ratio

Dep. variable:	TB ratio	log(TB ratio)	TB ratio	Pulmonary ratio
	(1)	(2)	(3)	(4)
TB dispensary$_{c,t}$	-9.3469^{**}	-0.1159^{**}	-9.0258^{**}	-8.7835^{**}
	(4.6363)	(0.0504)	(4.2110)	(3.7842)
TB ratio$_{c,t-1}$			0.0819^{***}	
			(0.0254)	
Long run effect			-9.8312^{**}	
City FE	Yes	Yes	Yes	Yes
Year FE	Yes	Yes	Yes	Yes
Time period	1890-1939	1890-1939	1891-1939	1901-1939
Observations	3,981	3,662	3,894	3,165
R-squared	0.4482	0.4852	0.4503	0.3491
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 death, in column (2) the log is taken of this variable, and in column (4) the left-hand-side variable is the pulmonary TB mortality per 1,000 death. 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 ratio$_{c,t-1}$ is a lagged dependent variable. Long run effect is the steady-state value of the estimated model in column (3). Robust standard errors clustered at the city level are in parentheses.
*, **, and ***, determine significance levels of 10%, 5% og 1% respectively.

Table A3: Flexible estimates of impact on TB before and after TB dispensary

Dep. variable:	TB rate	Disease
	(1)	(2)
$\text{Dispensary}_{c,t-10}^{\tau-10}$	0.0538	0.0686
	(0.1082)	(0.1133)
$\text{Dispensary}_{c,t-9}^{\tau-9}$	−0.0121	−0.0107
	(0.0918)	(0.1091)
$\text{Dispensary}_{c,t-8}^{\tau-8}$	−0.0055	0.0023
	(0.0850)	(0.1000)
$\text{Dispensary}_{c,t-7}^{\tau-7}$	0.0631	0.0721
	(0.0875)	(0.0995)
$\text{Dispensary}_{c,t-6}^{\tau-6}$	−0.0110	−0.0086
	(0.0904)	(0.1013)
$\text{Dispensary}_{c,t-5}^{\tau-5}$	−0.1497	−0.1192
	(0.0910)	(0.1076)
$\text{Dispensary}_{c,t-4}^{\tau-4}$	−0.0231	0.0026
	(0.0860)	(0.0962)
$\text{Dispensary}_{c,t-3}^{\tau-3}$	0.0074	0.0449
	(0.0882)	(0.0915)
$\text{Dispensary}_{c,t-2}^{\tau-2}$	−0.0359	−0.0213
	(0.0913)	(0.0975)
$\text{Dispensary}_{c,t}^{\tau}$	−0.1418	−0.1301
	(0.1056)	(0.1270)
$\text{Dispensary}_{c,t+1}^{\tau+1}$	−0.1372*	−0.1624*
	(0.0777)	(0.0869)
$\text{Dispensary}_{c,t+2}^{\tau+2}$	−0.2094*	−0.1531
	(0.1093)	(0.1233)
$\text{Dispensary}_{c,t+3}^{\tau+3}$	−0.1996**	−0.1553
	(0.0968)	(0.1061)
$\text{Dispensary}_{c,t+4}^{\tau+4}$	−0.2516*	−0.2531*
	(0.1427)	(0.1417)
$\text{Dispensary}_{c,t+5}^{\tau+5}$	−0.2745**	−0.1984
	(0.1129)	(0.1215)
$\text{Dispensary}_{c,t+6}^{\tau+6}$	−0.2656**	−0.2124*
	(0.1018)	(0.1137)
$\text{Dispensary}_{c,t+7}^{\tau+7}$	−0.2582*	−0.2587*
	(0.1373)	(0.1471)
$\text{Dispensary}_{c,t+8}^{\tau+8}$	−0.2529**	−0.2202
	(0.1210)	(0.1336)
$\text{Dispensary}_{c,t+9}^{\tau+9}$	−0.3186***	−0.2638**
	(0.1129)	(0.1234)
$\text{Dispensary}_{c,t+10}^{\tau+10}$	−0.2458**	−0.2115*
	(0.1135)	(0.1192)
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 fixed effects, disease-by-year fixed effects, disease-by-city fixed effects, 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 A4: Alternative test for common pretrend

Dep. variable:	TB rate	Disease
	(1)	(2)
TB dispensary$_{c,t}$	−0.2128**	−0.1769*
	(0.0869)	(0.0948)
TB dispensary$_{c,t+1}$	0.0362	0.0236
	(0.0910)	(0.0973)
TB dispensary$_{c,t+2}$	−0.0427	−0.0648
	(0.0660)	(0.0748)
TB dispensary$_{c,t+3}$	0.0322	0.0442
	(0.0902)	(0.0944)
TB dispensary$_{c,t+4}$	−0.0409	−0.0204
	(0.0707)	(0.0749)
Joint significance of leads	[0.9391]	[0.9283]
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	0.5333	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) includes disease fixed effects, disease-by-year fixed effects, disease-by-city fixed effects, and city-by-year fixed effects. TB dispensary$_{c,t+j}$ 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, where j specifies leads of this variable. Robust standard errors clustered at the city level are in parentheses.

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

Table A5: 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^{**}
	(0.0847)	(0.0766)	(0.0932)	(0.0885)
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 fixed effects, disease-by-year fixed effects, disease-by-city fixed effects, 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 A6: 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	Disease
	OLS				WLS		OLS	
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
TB dispensary$_{c,t}$	-0.1825^{**}	-0.2005^{***}	-0.2337^{***}	-0.2056^{***}	-0.2074^{***}	-0.1803^{**}	-0.1240^{*}	-0.1869^{***}
	(0.0769)	(0.0673)	(0.0816)	(0.0657)	(0.0767)	(0.0711)	(0.0740)	(0.0661)
log(Population$_{c,t-1}$)	-0.6804^{***}	-0.1202^{**}						
	(0.2331)	(0.0481)						
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
Initial log(population) × Year FE	No	No	Yes	Yes	No	No	No	No
City FE × Year FE	No	No	No	No	No	Yes	No	No
Linear trend × City FE	No	No	No	No	No	No	Yes	Yes
Time period	1891-1939	1902-1939	1890-1939	1901-1939	1890-1939	1901-1939	1890-1939	1901-1939
Observations	3,894	24,624	3,981	25,320	3,981	25,320	3,981	25,320
R-squared	0.5342	0.6490	0.5376	0.6487	0.5502	0.7135	0.5818	0.6515
Cities	87	87	87	87	87	87	87	87

Notes: The table reports ordinary least squares estimates, except column (5) and (6) repporting least squres 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), (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 fixed effects, disease-by-year fixed effects, and disease-by-city fixed effects, and column (6) also includes 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 log(Population$_{c,t-1}$) is the lagged log population. In column (3), and (4) the initial log population interacted with year fixed effects is included, and in column (7), and (8) 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 A7: Effect of TB dispensaries controlling for commissioning of waterworks

Dep. variable:	TB rate	Disease
	(1)	(2)
TB dispensary$_{c,t}$	-0.2331^{***}	-0.2097^{***}
	(0.0768)	(0.0676)
Waterworks$_{c,t}$	-0.0973	-0.0080
	(0.0913)	(0.0229)
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	No
Time period	1890-1939	1901-1939
Observations	3,981	25,320
R-squared	0.5339	0.6481
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 fixed effects, disease-by-year fixed effects, and disease-by-city 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 Waterworks$_{c,t}$ are an indicator equal to one after the introduction of waterworks. 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 main and branch TB dispensaries

Dep. variable:	TB rate	Disease
	(1)	(2)
Main dispensary$_{c,t}$	-0.2109^{**}	-0.2007^{**}
	(0.0931)	(0.0936)
Branch dispensary$_{c,t}$	-0.2316^{*}	-0.1722^{*}
	(0.1189)	(0.0907)
Joint significance of main and branch dispensary	[0.0232]	[0.0333]
Test of equivalence between main and branch dispensary	[0.8834]	[0.8119]
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 fixed effects, disease-by-year fixed effects, disease-by-city fixed effects, 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 ***, determine significance levels of 10%, 5% og 1% respectively.

Table A9: Effect of TB dispensaries in a balanced and extended panel

Dep. variable:	TB rate	Disease	TB rate	Disease
	Balanced panel OLS		Panel extended to 1946	
	(1)	(2)	(3)	(4)
TB dispensary$_{c,t}$	-0.2435^{***}	-0.2132^{***}	-0.1894^{***}	-0.1433^{***}
	(0.0858)	(0.0788)	(0.0606)	(0.0532)
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 fixed effects, disease-by-year fixed effects, disease-by-city fixed effects, 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.